中华医学会第十次妇科肿瘤学
中青年医师学术会议

论文汇编

2025 年 9 月 成都

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论文发言

OR_001

Artificial Intelligence for Predictive Biomarker in Gynecological Oncology: A Scoping Review of Technical Challenges and Clinical Translation

Wu Jingyuan Peng Wu*
Department of Obstetrics and Gynecology, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, Hubei, P. R. China.

Immunotherapy has reshaped the therapeutic landscape of gynecological oncology, offering superior outcomes over chemotherapy in both monotherapy and combination treatment. However, the identification of patients most likely to benefit from immune checkpoint inhibitors (ICIs) remains suboptimal, owing to the limited predictive accuracy of current biomarkers such as PD-L1 expression, microsatellite instability-high (MSI-H) status, and tumor mutational burden (TMB). These markers fail to fully capture the multidimensional complexity of tumor–immune interactions. Static tissue-level features, including radiomics, pathomics, and microenvironmental signatures, provide constrained snapshots. Molecular correlates, such as gene expression profiles, T-cell receptor repertoires (TCR), and stromal characteristics, are further complicated by inter-patient heterogeneity and intra-tumoral spatiotemporal dynamics. Emerging liquid biopsy modalities, including circulating tumor DNA and microbiome signatures, enable longitudinal monitoring but demand rigorous multimodal validation. Artificial intelligence (AI)-enabled integration of heterogeneous data types offers a transformative solution: the development of 'meta-biomarkers' that reconcile clinical relevance, technical reproducibility, and cost-efficiency. This Review critically examines current strategies for multimodal biomarker discovery, with an emphasis on data fusion, feature engineering, and prospective validation. Large-scale, multi-institutional trials across the biomarker lifecycle are urgently needed to realize AI-driven precision immunotherapy.

OR_002

148 例中国子宫肉瘤患者临床病理特征及预后分析

黄婷 张果 王建六
北京大学人民医院

背景 子宫肉瘤是一种罕见但高度异质性的恶性肿瘤，不同病理类型预后差异显著，且诊断困难，缺乏特异性标志物，治疗选择有限。本研究旨在分析子宫肉瘤的临床病理特征、预后因素及潜在治疗靶点。
方法 回顾性分析 1996-2025 年北京大学人民医院治疗的 148 例子宫肉瘤患者的临床资料、病理类型、免疫组化特征、生存结局及复发模式，并利用生物信息学方法探索不同亚型的分子特征。
结果 148 例子宫肉瘤患者年龄分布在 49±12.8 岁，最常见病理类型为子宫平滑肌肉瘤（57/148，38.5%），其次为低级别子宫内膜间质肉瘤（44/148，29.7%）。子宫肉瘤患者 5 年复发率 50.5%（46/91），复发的中位时间为 19.3（2.4-214.5 月）个月，转移位置以盆腔和肺最为常见；患者 5 年和 10 年的总生存率分别为 80.2%（73/91）和 84.0（42/50），其中 LGESS 5 年生存率 90.3%（28/31），显著优于高级别子宫内膜间质肉瘤 83.3%（5/6）、未分化肉瘤 50.0%（1/2）和平滑肌肉瘤 61.8%（21/34）。多因素分析显示，子宫肉瘤病理类型、分期、核分裂相和凝固性坏死与复发密切相关；病理类型、分期和凝固性坏死是患者总生存期和无进展生存期的共同独立危险因素。72.7%的子宫平滑肌肉瘤表达 ER，57.1%的子宫平滑肌肉瘤患者表达 PR，且子宫平滑肌肉瘤 ER 和 PR 表达均与 PFS 显著相关，ER 高表达患者的总生存期优于 ER 表达阴性者，而 PR 高表达患者的总生存期略优于 PR 阴性者，但无统计学差异。转录组学数据提示高级别子宫内膜间质肉瘤呈现免疫抑制微环境（CSF1R/CSF3R 高表达），而平滑肌肉瘤以细胞周期基因激活和同源重组缺陷为特征。

结论 子宫肉瘤的预后高度依赖病理亚型和分期，子宫肉瘤高危患者应加强肺部监测。分子特征分析提示免疫治疗（CSF1R 抑制剂+PD-1）和靶向治疗（PARP/CHEK 抑制剂）可能改善高级别间质肉瘤和平滑肌肉瘤的疗效，需进一步临床验证。

OR_003

The Role of Central Nervous System Tumor Methylation Classifier in the Classification of Ovarian Primitive Neuroectodermal Tumor

Tianyu Zhang[1] Jie Yang[1] Leiming Wang[2] Xiaohua Shi[3] Jiaxin Yang[1*]
1.Department of Obstetrics and Gynecology, Peking Union Medical College Hospital
2.Department of Pathology, Xuanwu Hospital, Capital Medical University
3.Department of Pathology, Peking Union Medical College Hospital

Background and Aim Ovarian neuroectodermal tumors (NETs) are rare malignancies with poorly defined diagnostic criteria and heterogeneous clinical behavior, posing significant challenges in clinical treatment. We aimed to assess the utility of DNA methylation in the diagnostic classification and prognostic stratification of ovarian NETs.
Methods This retrospective study included 15 patients diagnosed with ovarian NETs at Peking Union Medical College Hospital between 2010 and 2024. Paraffin-embedded tumor tissues underwent clinicopathologic review, DNA methylation microarray assay, EWSR1 fluorescence in situ hybridization, and immunohistochemistry (SALL4, GFAP, Nestin, p53, SOX2, SOX11, and LIN28A).
Results The median age at diagnosis of ovarian NETs was 19 years (range 9–73 years). Morphologic diagnosis included 3 medulloblastoma, 1 neuroblastoma, 3 embryonal tumors with multilayered rosettes (ETMR), 3 ependymoma, 1 high-grade glioma, 1 gliosarcoma, 1 low-grade neuronal-glial tumor and 2 tumors cannot be specified. 73.3% (11/15) presented with teratoma background. All tumors exhibited lacked EWSR1 gene rearrangement. Methylation classification was consistent with morphologic diagnosis in 30% patients (5/15). A novel ETMR, non-C19MC-altered type ovarian tumor were identified in 3 patients. The median follow-up period was 14.9 months (range 2.1-216.4 months), during which 60.0% of patients experienced recurrence or disease progression and the mortality rate was 33.3%. Patients with ETMR non-C19MC-altered subtype and unmatched tumors exhibited extremely poor outcomes, with 80% (4/5) mortality within 12 months.
Conclusions DNA methylation profiling resolves diagnostic ambiguity in ovarian neuroectodermal tumors, stratifying tumors into prognostically distinct subgroups. Leveraging the central nervous system (CNS) tumor methylation classifier to diagnose peripheral neuroectodermal tumors offers critical clinical insights for these rare malignancies, enabling precise molecular subtyping, prognostication, and alignment with CNS-targeted therapeutic strategies.

OR_004

Management and prognosis of ultra high-risk gestational trophoblastic neoplasia patients: a long-term retrospective study

Meng Mao
The First Affiliated Hospital of Zhengzhou University

Background The prognosis for patients with ultra high-risk gestational trophoblastic neoplasia (GTN) is significantly worse, and there is currently no consensus regarding the optimal treatment strategies for this specific patient population.
Objectives This study aims to investigate the clinical characteristics, treatment responses, outcomes, and prognostic risk factors in ultra high-risk GTN patients with a FIGO score of 13 or higher.

Design: Retrospective study.
Methods Medical records of 36 GTN patients with FIGO score ≥ 13, treated at the first affiliated hospital of Zhengzhou University, China, from January 2015 to June 2024, were retrospectively reviewed. Chi-square tests, univariate analysis, and Kaplan-Meier survival analysis were employed for data analysis.
Results Etoposide, methotrexate, actinomycin D, cyclophosphamide, and vincristine (EMA-CO) was the most commonly used chemotherapy regimen (17/36, 47.2%). Twenty-one patients responded well to the initial chemotherapy regimen and achieved complete remission. One patient was switched to salvage chemotherapy due to resistance to the initial regimen and subsequently achieved complete remission (CR). Four patients experienced relapse; of these, three attained CR. The median follow-up time was 46 months (range 12-85 months). The CR rate was 69.4% (25/36). A total of 20 patients (55.6%) received adjuvant treatments, including surgery and radiotherapy. Stage Ⅳ disease, liver metastases, uncommon distant metastatic sites, and ≥3 metastatic sites were significant predictors of mortality.
Conclusions In our study, the overall CR rate for ultra high-risk GTN patients with FIGO score≥ 13 was 69.4%. Five patients (45.5%) experienced early mortality. All patients with brain metastases who received chemotherapy in conjunction with whole-brain or stereotactic radiotherapy achieved CR. Immune checkpoint inhibitors demonstrated potential efficacy in treating chemotherapy-resistant GTN.

OR_005

基于免疫特征的妇科透明细胞癌分子分型及临床意义

胡潇潇[1,2] 龙潇冉[1,2] 臧婧羽[1,2] 庄光磊[1,2] 殷霞[1,2*]
1.上海交通大学医学院附属仁济医院妇产科 2.上海市妇科肿瘤重点实验室

目的 探究卵巢透明细胞癌（ovarian clear cell carcinoma, OCCC）和子宫内膜透明细胞癌（endometrial clear cell carcinoma, ECCC）的免疫微环境差异，评估免疫表型与临床预后的相关性，为个体化肿瘤免疫治疗提供理论依据。
方法 建立多中心临床研究队列（4 个中心，117 例 ECCC，102 例 OCCC），整合单细胞测序（n = 6）、空间转录组（n = 2）及多重免疫荧光（n = 186）分析妇科透明细胞癌的免疫特征。根据 IHC 定量分析肿瘤核心和侵袭性边缘的 CD3+和 CD8+T 细胞分布，进行 Immunoscore 评分及划分免疫表型（浸润型/排除型/沙漠型），结合临床资料分析不同免疫表型的临床预后。
结果 多重免疫荧光分析提示 ECCC 的 CD4+、CD8+T 淋巴细胞及 CD79a+B 淋巴细胞丰度显著高于 OCCC（P < 0.001），而 OCCC 侵袭性边缘 CD14+髓系细胞更富集（*P* = 0.003）。空间分析表明 ECCC 免疫细胞与肿瘤细胞的平均最近邻距更短（*P* = 0.012），提示更紧密的免疫浸润。单细胞测序提示两者免疫细胞组成相似，但 ECCC 的淋巴细胞占比更高（22.2% vs 12.9%），OCCC 中性粒细胞比例显著高于 ECCC（46.0% vs 4.6%）。免疫组化分析进一步证实 OCCC 的 Immunoscore 阴性比例（71.9% vs 55.6%, *P* < 0.001）和免疫沙漠型比例（70.2% vs 42.4%, *P* < 0.001）均显著高于 ECCC。生存分析显示免疫沙漠型透明细胞癌的无进展生存期和总生存期比免疫浸润型更短，而免疫排除型表现出中等疾病进展风险。
结论 卵巢和子宫内膜透明细胞癌在肿瘤免疫特征和空间分布模式方面存在显著差异，ECCC 的免疫浸润更为丰富、紧密，以淋系来源为主。不同的免疫表型与患者的预后密切相关，针对不同类型透明细胞癌患者的免疫表型进行个体化治疗，有望提高治疗效果。

OR_006

女性生殖系统肝样腺癌临床病理特征及全基因组测序分析

夷恬进 徐炼 周圣涛*
四川大学华西第二医院

背景 女性生殖系统肝样腺癌（Hepatoid Carcinoma, HC）是一种罕见且高度恶性的肿瘤，因缺乏对其分子发病机制的认知，临床治疗手段有限，患者预后极差。本研究旨在系统性描绘其临床病理特征与基因组全景，以揭示其核心发病机制并寻找潜在的治疗靶点。
方法 本研究回顾性纳入 2010 年 1 月至 2024 年 6 月期间在华西第二医院妇科诊治的 HC 患者的临床病理资料，通过免疫组化（IHC）明确病理诊断，对所有样本进行全基因组测序（WGS）。运用生物信息学方法分析了肿瘤异质性、全基因组复制（WGD）、肿瘤突变负荷（TMB）等，并对原发灶（T vs N）、转移灶（M vs N）及两者之间（M vs T）的体细胞突变进行三组比较。
结果 共纳入 13 例样本（包括原发灶 5 例、转移灶 3 例及正常组织 5 例），分别来自 5 例 HC 患者（卵巢 2 例、输卵管 1 例、宫颈 1 例和阴道 1 例）。临床上，75%（3/4）患者为 FIGO III-IV 期（1 例患者因高龄无法行全面分期手术除外）和血清 AFP 水平急剧升高（中位数：2594 ng/ml，范围：298-10237.8 ng/ml）。IHC 证实了肿瘤的肝样分化（AFP/Glypican-3/HepPar-1 弥漫性强阳性）及高度增殖活性（p53 突变型表达 100%，Ki-67 指数 70-80%）。所有患者均接受了手术治疗，并辅以含铂类药物的化疗方案，部分患者联用了贝伐珠单抗（2 例）或放疗（1 例，阴道 HC）。基因组层面，我们发现：（1）60%（3/5）病例发生了 WGD，并伴有显著的高倍性特征（Ploidy > 3.0），表明染色体倍增可能驱动该肿瘤演进的机制之一。（2）所有肿瘤样本均表现出超高肿瘤突变负荷（TMB-High）表型（672-1582 Mut/Mb），提示免疫检查点抑制剂可能是一种有效的治疗策略。（3）体细胞突变的三组比较初步描绘了肿瘤进展的路径。其中，MUC16 突变在全部三组比较（T vs N、M vs N 和 M vs T）中均存在，贯穿于肿瘤从发生到转移的整个生命周期；RNF213 突变在原发灶和转移灶（T vs N 和 M vs N ）中共有；ZFHX4、DNAH2、QRICH2、FBXL18 突变只在转移灶（M vs N 和 M vs T）中出现。
结论 本研究首次初步揭示了女性生殖系统 HC 的基因组全景，WGD、高 TMB 是其标志性分子事件，以 MUC16、RNF213 等基因为代表的突变特征进一步揭示其发病机制，为开发靶向治疗策略提供了初步依据。

OR_007

妇科恶性肿瘤术后淋巴囊肿并发感染的高危因素分析

邓子晗 衣欢*
福建省妇幼保健院

目的 本研究评估妇科恶性肿瘤术后淋巴囊肿并发感染的临床特点及相关危险因素，探讨其预防和治疗策略，旨在为妇科恶性肿瘤术后淋巴囊肿的妇女提供更好的预后。
方法 本研究为回顾性队列研究，系统性分析福建省妇幼保健院妇科 2016 年 1 月至 2023 年 12 月收治的 276 例妇科恶性肿瘤淋巴结清扫术后并发淋巴囊肿患者的临床资料。依据术后淋巴囊肿是否并发感染将患者分为感染组（n=97）与非感染组（n=179）。比较两组患者的基线资料（如年龄、BMI、合并症）、手术相关指标（手术时长、术中出血量、淋巴结清扫范围、淋巴管处理方式）、生化检查结果（包括血糖、白蛋白水平）、淋巴囊肿特征（大小、位置、出现时间、引流量及性状）以及血压情况、是否放化疗等危险因素。
结果 通过单因素和 Logistic 回归分析显示，感染组患者较非感染组具有显著升高的感染风险，相关因素包括：手术持续时间延长、淋巴囊肿体积更大且存在时间更长、术中淋巴管断端采用钝性分离 (vs 锐性分离)、术后引流管微生物培养阳性以及引流区域感觉异常，以上差异均有统计学意义（P＜0.05）。

这些发现强调了优化手术技术（如控制手术时长、优先选择锐性分离淋巴管）及加强术后引流管理（包括密切监测引流液性状、警惕神经感觉异常）对于降低淋巴囊肿感染风险的重要性。

结论 妇科恶性肿瘤术后淋巴囊肿并发感染受多种因素影响，早期识别高危因素并采取针对性预防措施有助于降低感染发生率，改善患者预后。

OR_008

卵巢恶性生殖细胞肿瘤循环肿瘤 DNA 的检测及应用

张馨月 杨洁 杨佳欣*
中国医学科学院北京协和医院

研究目的 分层诊疗是卵巢恶性生殖细胞肿瘤（MOGCT）目前重点研究的方向，循环肿瘤 DNA（ctDNA）检测是微小残留病灶（MRD）的主要检测方法之一。为系统了解 MOGCT 患者在不同临床阶段的 ctDNA 状态，分析 ctDNA 与预后之间的关系，寻找更精准的肿瘤标志物和分层依据，开展本项研究。

材料与方法 研究纳入自 2023 年 1 月至 2024 年 3 月在中国医学科学院北京协和医院初次就诊的卵巢恶性生殖细胞肿瘤患者。在手术当日留取患者肿瘤组织样本。在患者手术前、化疗结束时、化疗结束 3 个月、6 个月、12 个月留取外周血。采用基于肿瘤突变信息的个体化 ctDNA 检测方法进行样本检测。使用 R 4.2.2 进行统计分析。

结果 本研究最终纳入 14 例患者进行 ctDNA 检测和分析，中位诊断年龄 14.5 岁，其中 7 例卵黄囊瘤，2 例无性细胞瘤，1 例未成熟畸胎瘤伴 PNET，4 例混合性生殖细胞肿瘤。4 例患者 FIGO 分期 I 期，2 例为 II 期，6 例为 III 期，2 例为 IV 期。对肿瘤组织的全外显子测序（WES）数据进行分析，5 例患者存在 I/II 级的体细胞突变，2 例患者存在 4/5 级胚系突变，6 例患者观测到 i(12p)，2 例患者观测到 12 号短臂染色体拷贝数扩增。患者基线 ctDNA 阳性率 92.9%，在治疗结束时，ctDNA 阳性率明显下降，13 例患者中仅 3 例（23%）患者为阳性。通过 Kaplan-Meier 生存分析来评估不同时间点 ctDNA 状态与患者预后的相关性。在化疗后 3 个月，ctDNA 阳性组患者无进展生存期（PFS）与阴性组相比显著缩短（log rank P=0.044，HR=10.22，95%CI 0.824-1413.551）。化疗结束后任一时间点 ctDNA 阳性的患者 PFS 较持续阴性组缩短，中位 PFS 分别为 3.1 个月与未达到（log rank P=0.15，HR=4.549，95%CI 0.472-43.842）。此外，ctDNA 在疾病治疗和随访期间对肿瘤情况有监测作用。本研究纳入的一例患者术前非特异性的 NSE、CA125 升高，手术和化疗后肿瘤标志物一直正常，但通过检测 ctDNA 升高发现了肿瘤复发，并且得到了影像学证实，说明对于缺乏特异性好、灵敏度高的血清肿瘤标志物的患者，ctDNA 是一个较好的无创监测指标。

结论 本研究首次实现了 MOGCT 患者的 ctDNA 检测，证实了基于肿瘤突变信息的个体化 ctDNA 检测方法的可行性。ctDNA 状态和肿瘤预后相关，治疗后 ctDNA 阳性患者肿瘤复发风险高。在疾病治疗和随访期间 ctDNA 的动态变化可以辅助疗效评估并为发现复发提供依据。

OR_009

妇科恶性肿瘤患者术后肺栓塞的临床特征分析与临床防治策略

张灿
四川省肿瘤医院

目的 探讨妇科恶性肿瘤患者术后发生肺栓塞（pulmonary embolism ，PE）的临床相关因素与防治策略。

方法 收集 2019 年 1 月 1 日至 2024 年 10 月 31 日期间于四川省肿瘤医院妇瘤科行手术治疗的妇科恶性肿瘤患者的临床资料，将发生 PE 的 17 例患者作为 PE 组，同时计算机随机抽取同期非 PE 的妇科恶性肿瘤手术患者 182 例作为对照组。回顾性分析妇科恶性肿瘤并发 PE 的危险因素。
结果 17 例 PE 患者平均年龄为 59.7 岁。PE 发生的中位时间为术后 4 天。17 例 PE 患者手术方式均为开腹,中位手术时长为 3.9 小时。临床症状主要表现为气紧、胸闷，无症状低氧血症 1 例。17 例 PE 患者均经 CT 肺动脉血管造影（computed tomography pulmonary angiography，CTPA）显示肺动脉及其分支充盈缺损确诊，其中双侧 PE 9 例，单侧 PE 8 例。约 58.8%（10/17）合并有双下肢静脉血栓。17 例 PE 患者经依诺肝素钠抗凝治疗后均好转。单因素分析显示，妇科恶性肿瘤患者术后发生 PE 与 D-二聚体、血红蛋白、白细胞计数、年龄、开腹手术、术中输血、合并基础疾病（高血压、糖尿病）明显相关（P<0.05）。进一步行多因素分析显示，D-二聚体、血红蛋白、白细胞计数是影响患者 PE 的独立危险因素（P<0.05）。
结论 妇科恶性肿瘤患者术后 PE 不仅严重影响患者健康甚至危及生命，也会影响患者术后恢复、增加住院时长，故临床医生应引起高度重视。对于具有高龄肥胖、合并高血压和糖尿病、伴有大量腹水和巨大盆腔包块、手术时长超过 3 小时、术中大血管损伤等，术后检验结果示 D-二聚体升高、血红蛋白降低、白细胞计数升高的妇科恶性肿瘤患者，应重视预防措施，且一旦患者术后出现胸闷、气紧、无症状低氧血症等症状时，立即进行 PE 相关检查及处理，以降低相关风险，改善患者预后。

OR_010

耐药妊娠滋养细胞肿瘤患者的预后危险因素

毛铭懿 蒋芳 向阳*
北京协和医院

研究目的 约 30%的妊娠滋养细胞肿瘤（gestational trophoblastic neoplasia，GTN）患者在一线化疗后出现耐药，耐药是威胁 GTN 患者生存的重要因素。本研究旨在探究耐药妊娠滋养细胞肿瘤患者的预后危险因素。
材料与方法 本研究为回顾性队列研究，纳入了 2002 年 1 月至 2019 年 6 月在北京协和医院接受多药联合挽救化疗、FIGO 评分≥5 分的耐药 GTN 患者。为评估 FIGO 评分系统在此类人群中的预后预测价值，我们对比了不同 FIGO 评分分数段初治与耐药患者的治疗疗效与预后差异。同时，回顾了耐药患者治疗时的各项临床参数，利用单因素及多因素 logistic 回归分析评估耐药患者再次耐药的危险因素，并构建了针对耐药患者的新预后评分系统。
结果 研究共纳入了 458 例初治和 253 例耐药 GTN 患者。通过对比初治及耐药患者的治疗反应，我们发现 FIGO 评分≥9 分的耐药患者，再次耐药及疾病进展的风险显著高于同分数段的初治患者（表 1）。耐药患者的 FIGO 评分与再次耐药风险显著正相关。通过受试者工作特征曲线分析，我们确定了以 10 分为切点值的新分组，将耐药患者分为了 2 组，FIGO 评分≥10 分的患者治疗疗效及预后显著劣于 FIGO 评分＜10 分的患者。此外，logistic 回归分析显示化疗开始间隔≥12 个月、化疗前 β-hCG 水平≥500 IU/L、多次耐药史、既往失败化疗疗程数≥7 次为耐药患者再次耐药的独立危险因素。基于此，我们构建了一个新型的简化评分系统，用于预测耐药患者的后续耐药风险（表 2）。
结论 耐药 GTN 患者具有独特的预后特征，本研究提出了将 FIGO 评分≥10 分作为新的切点值，并建立了新型评分体系，以更好地识别再次耐药高风险人群。

OR_011

多学科诊疗模式在卵巢癌肝及肝周转移手术中的实践分析

郭环[1,2] 王永鹏[1*] 金山[1] 陈丹[1] 张悦[1]
1.辽宁省肿瘤医院/大连理工大学附属肿瘤医院 2.辽宁中医药大学

目的 探讨多学科诊疗模式指导下卵巢癌肝及肝周转移灶的发病特点及预后。
方法 回顾性分析 2021 年 10 月至 2024 年 6 月在辽宁省肿瘤医院妇科同一 MDT 团队术前精准评估及术中联合手术治疗的 70 例原发卵巢癌伴有肝及肝周转移的患者队列，收集临床资料，包括年龄、ECOG 评分、CA125、HE4、初始治疗方式、肿瘤细胞残留、病理类型、FIGO 分期、术后化疗、靶向治疗、基因检测、维持治疗以及随访结果，比较不同因素对预后的影响。分析肝脏及其周围区域转移病灶的位置及占比情况。同时对影响卵巢癌患者无进展生存时间的相关因素进行单因素分析以及多因素分析，并以此探讨多学科诊疗模式指导下卵巢癌肝及肝周转移灶切除达到 RD0 后对患者无进展生存期的影响。
结果 70 例患者中位年龄 59 岁。PDS 组中 RD0：45 例，RD1：9 例，IDS 组中 RD0：14 例、RD1：2 例。卵巢癌肝周转移部位中：膈肌转移 55 例（78.57%），其次小网膜 23 例（32.86%），肝左叶间裂 22 例（31.43%），肝肾隐窝 11 例（15.71%），肝正中叶间裂 8 例（11.43%），肝脏腹膜 6 例（8.57%），肝实质 4 例（5.71%）。单因素分析初始治疗方式及肿瘤细胞残留均与卵巢癌患者无进展生存期显著相关，具有统计学意义（$P<0.05$），多因素 Cox 分析结果显示，初始细胞减灭术且达到 RD0 与卵巢癌患者无进展生存期显著相关（$P<0.05$），是影响卵巢癌患者无进展生存期（PFS）的独立保护因素。本组患者的 Kaplan-Meier 生存分析结果，1 年 PFS 率为 82%，2 年 PFS 率为 61%。
结论 卵巢癌肝及肝周转移病灶主要位于膈肌表面或肝周间隙。针对卵巢癌肝及肝周的转移特点，通过 MDT 团队术前精准评估手术难度及风险制定手术策略，尽最大努力选择初次细胞减灭术作为治疗方式，术中联合精准切除病灶，提高 RD0 切除率，有效延缓病情进展，为患者带来更优的生存获益。

OR_012

Web of Science 核心合集 2005—2025 年间妇科恶性肿瘤生育力保存研究的文献计量与可视化分析

李馨舒[1,2,3] 哈春芳[2*]
1.宁夏医科大学临床医学院 2.宁夏医科大学总医院 3.宁夏医科大学生育力保持重点实验室

研究目的 通过对妇科恶性肿瘤生育力保存领域研究进行分析，探讨近二十年该领城研究热点及发展趋势，为今后我国妇科恶性肿瘤生育力保存的研究提供参考。
材料与方法 使用 Web of Science 核心数据库检索 2005 年 1 月 1 日-2025 年 8 月 1 日发表的有关妇科恶性肿瘤生育力保存的相关文献；使用 VOSviewer 1.6.17、CiteSpace V6.4.R1 软件对文献进行可视化分析，探讨该领域研究现状及热点。
结果 年度产出呈“指数-平台”曲线：2005-2007 年年均不足 20 篇；2008 年起显著上升，2021 年达 116 篇峰值；2022-2025 年稳定在 67-95 篇之间，提示研究进入“质量深耕”阶段。4864 位作者中，Scambia, Giovanni（24 篇）产量最高；Raspagliesi, Francesco 以 106 条合作连线成为网络“桥梁”。271 位高频作者形成 17 个聚类，88.6%处于同一网络，表明合作紧密。68 个国家参与研究，美国 254 篇居首，中国 184 篇次之，但总链接强度（33 vs 114）落后，提示我国跨国合作不足。意大利、英国、法国依托 ESGO 网络形成高密度欧洲聚类。机构方面，得克萨斯大学系统（48 篇）与巴黎公立医院集团（中心性 0.19）分别为产量与枢纽双冠军；复旦大学 32 篇排第五，但中心性仅 0.02，显示国际话语权仍待提升。223 种期刊刊文，《Gynecologic Oncology》（144 篇）与《Int J Gynecol Cancer》（124 篇）领跑；IF 最高的《Fertility and Sterility》（7.0）兼顾生殖医学与肿瘤学，是跨学科交流平台。共现网络显示，“cervical cancer”（308 次）与“fertility preservation”（284 次）构成最大簇；“endometrial

cancer”“adenocarcinoma”因高中心性成为知识桥梁。突现词演变呈现三阶段：2005-2013 年：“pelvic lymphadenectomy”“vaginal radical trachelectomy”提示手术路径创新；2014-2018 年：“fertility”“young patients”聚焦适应证拓展；2019-2025 年：“oral progestin”“reproductive outcome”标志研究重心向药物保育与妊娠结局迁移。

结论 近二十年妇科恶性肿瘤生育力保存的研究越来越受到学者们关注，全球妇科恶性肿瘤生育力保存领域已完成“技术验证—循证整合”的第一次跃迁。我国需在高质量真实世界数据、跨学科合作及支付政策上同步发力，让更多年轻肿瘤患者“既活得长，也生得好”。

OR_013

Epitranscriptomic mechanisms and implications of RNA m5C modification in gynecologic cancer

Zhonghao Mao[1,2,3] Yan Tian[1,2,3] Lisha Wu[2,3] Yu Zhang[1,2,3*]
1.Department of Gynecology, Xiangya Hospital, Central South University
2.Gynecological Oncology Research and Engineering Center of Hunan Province
3.National Clinical Research Center for Geriatric Disorders, Xiangya Hospital, Central South University

Gynecologic cancer is an extremely complex disease characterized by abnormal cell growth due to genetic and environmental factors. With the rise of the field of epigenetic transcriptomics, 5-methylcytidine (m5C) modification has been identified as one of the most common chemical modifications occurring in various RNA types. The writers, erasers, and readers of m5C modification regulate cancer initiation, progression, and therapeutic responses, such as the proliferation, metastasis, angiogenesis, metabolic reprogramming, immune escape, and therapeutic resistance of tumour cells, by regulating RNA stability, translation, nuclear export, and splicing processes. In this review, we elucidate the biological process of m5C modification, summarize the abnormal expression of RNA-modifying proteins (RMPs) in gynecologic cancer, explore their functional effects on malignant hallmarks of cancer and molecular mechanisms, and prospect the potential clinical application value of m5C.

OR_014

HPV Integration Initiates a Carcinogenic Positive Feedback Loop via Recognition of Stripe Transcription Factors

Canhui Cao[1] Peng Wu[2*]
1.Tongji Hospital of Tongji Medical College of Huazhong University of Science and Technology
2.Union Hospital of Tongji Medical College of Huazhong University of Science and Technology

Introduction Integration of high-risk human papillomavirus (hrHPV) genomes markedly increases during the transition from precancerous lesions to invasive carcinoma, representing a pivotal event in HPV-associated tumorigenesis. However, the molecular mechanisms by which hrHPV integration drives malignant transformation—and the therapeutic vulnerabilities it creates—remain largely undefined.

Methods We employed cervical epithelial organoids and genetically engineered mouse models with HPV-KLF5 integration to model hrHPV-driven malignant progression. A comprehensive multi-omics approach—including high-throughput viral integration detection (HIVID), high-resolution chromosome conformation capture (Hi-C), ATAC-seq, ChIP-seq, single-cell and spatial RNA sequencing—was used to characterize the chromatin and transcriptional landscapes associated with HPV integration. Clinical samples and animal models were further used to validate the role of stripe transcription factors (STFs) in mediating oncogenic reprogramming.

Results Our models successfully recapitulated hrHPV integration-driven carcinogenesis. Multi-omics analyses revealed that HPV integration enhances chromatin accessibility and promotes the formation of oncogenic super-enhancer–promoter loops via recognition of STFs. Single-cell RNA-seq showed a marked upregulation of STF expression and activity in HPV-positive cervical and head and neck cancer trajectories. Spatial transcriptomics confirmed that STF activation occurs in regions of hrHPV integration and is suppressible by STF inhibition, underscoring its functional role in lesion progression. **Conclusions** Our findings identify a carcinogenic positive feedback loop initiated by hrHPV integration through STF recognition. Targeting STFs may represent a promising therapeutic strategy for HPV-driven malignancies.

OR_015

STR 分子分型技术辅助妊娠滋养细胞肿瘤成因诊断的应用

陈婷婷 朱国华 陈金娟 周先荣 鹿欣*
复旦大学附属妇产科医院

目的 妊娠滋养细胞肿瘤是一组来源于胎盘滋养细胞疾病的罕见恶性肿瘤，包括绒毛膜癌，以及更为罕见的胎盘部位滋养细胞肿瘤和上皮样滋养细胞肿瘤，上述肿瘤分为妊娠相关和非妊娠相关两大类，其中妊娠相关肿瘤可继发于水泡状胎块，也可以继发于流产宫外孕或足月产后。临床上此类病例经常缺乏病理学证据，推断其成因尤为困难，而肿瘤成因的确定，对于此类肿瘤的评分和治疗方案的制定，随访和预后的分析具有重要意义。此类肿瘤的病理组织学诊断困难，通过病理组织学诊断更无法判断其来源。在样本可及的情况下，STR 分子分型技术是确定其成因的金标准，本研究利用该技术对本院若干绒毛膜癌病例做出来源鉴定，为临床提供治疗依据。

材料与方法 本研究中的样本来自本中心 2021-2025 年绒癌 FFPE 标本（共 50 例）。以患者颊粘膜 DNA 为对照组，绒癌或前次妊娠物 FFPE 标本的 DNA 为实验组（镜检富集显微切割病灶，抽取核酸），使用 STR-MR21D 试剂盒对肿瘤样本和前次妊娠物/水泡状胎块的 DNA 行多重荧光 PCR 和 3500DX 毛细管电泳，对峰图进行分析。

结果 50 例样本中，12 例绒癌样本的 STR 峰图与患者颊粘膜峰图拷贝数一致，确认为非妊娠相关绒癌。38 例绒癌样本 STR 峰图可见父系来源峰，确认为妊娠相关绒癌。在这些妊娠相关绒癌中，22 例由于绒癌样本有母体组织微量污染，仅能确认为妊娠相关；15 例样本，可以通过绒癌成分的精准切割或与前次妊娠物样本的峰图比对，进一步精准确认妊娠来源，其中 9 例来源于纯和双雄二倍体水泡状胎块，7 例来源于杂合双雄二倍体水泡状胎块，未见足月产后或其它流产物来源。

结论 由上述绒癌的 STR 分型结论可见，非妊娠相关绒癌的占比并不低（占比 24%），且其中占比 1/4（3 例/12 例）为具有妊娠史的育龄期妇女，此类病例在缺乏分子病理诊断的情况下，极易被当作妊娠绒癌进行临床处理，而非妊娠绒癌的恶性程度往往高于妊娠绒癌，且对传统妊娠绒癌化疗方案不敏感，误诊将造成不良的临床结局。 妊娠相关绒癌占大多数（占比 76%），在样本理想的情况下，可以精准区分为杂合双雄或纯和双雄二倍体水泡状胎块来源，由于 STR 技术的敏感性，水泡状胎块的 FFPE 样本可以溯源 5-7 年以上，根据现有研究，杂合双雄二倍体来源或前次水泡胎时间久远可能具有更大的潜在风险。STR 分子分型的精准诊断，为临床对绒癌进行精细化风险评分和诊断以及患者的预后和随访，提供重要依据。

OR_016

Precise treatment of chemotherapy-induced premature ovarian failure: A co-transplantation system for restoring dysfunctional ovaries

Jiachen Wu Lingjuan Wang Kezhen Li*
Department of Gynecological Oncology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

Objective This study aimed to investigate the therapeutic potential, safety, and mechanisms of a newly constructed co-transplantation system involving bone marrow mesenchymal stem cells (BMSCs) and multi-biofunctional graphene oxide-enhanced poly-L-lactic acid (GO/PLLA) nanofiber scaffolds for the restoration of impaired ovarian function.
Methods GO/PLLA nanofiber scaffolds with varying concentrations of GO nanosheets were co-cultured with BMSCs to select the most cytocompatible scaffold for constructing the co-transplantation system. Female C57BL/6J mice were divided into five groups to assess the impact of the co-transplantation system on ovarian damage repair, short- and long-term health of offspring, and safety. Bulk RNA sequencing and single-cell RNA sequencing were employed to investigate the potential mechanisms of the co-transplantation system in promoting ovarian damage repair.
Results The co-transplantation system involving BMSCs and 1.0wt% GO/PLLA nanofiber scaffolds showed superior cell proliferation ability and viability in vitro. In vivo experiments demonstrated that the co-transplantation system effectively restored ovarian function, improved oocyte quality and natural fertility in mice with premature ovarian failure, without adverse effects on offspring health. Differential gene expression analysis revealed changes in the activities of chemokines, cytokines, immune response, and metabolic pathways in ovarian tissue.
Conclusion Research applications of the co-transplantation system have confirmed its potential for restoring ovarian function and improving fertility, while further enhancing the understanding of the in vivo differentiation process of BMSCs.

OR_017

Single-cell Landscape of the Tumor Immune Microenvironment in Human Gynecologic Malignancies

Junfen Xu*
Department of Gynecologic Oncology, Women's Hospital, Zhejiang University School of Medicine, Hangzhou, Zhejiang, China

Objective The study aims to systematically characterize the tumor immune microenvironment (TIME) of the three most prevalent gynecological malignancies—tubo-ovarian cancer, endometrial cancer, and cervical cancer—an area that has been underexplored and limits clinical translation, particularly for immunotherapy development.
Materials and Methods We integrated publicly available single-cell RNA sequencing (scRNA-seq) datasets comprising 111 tumor and non-malignant tissue samples. A total of 272,389 $CD45^+$ immune cells were extracted for comprehensive analysis. These cells were categorized into various immune subsets, and their distribution, functional states, and clinical relevance were assessed. Selected findings were further validated through multi-color immunohistochemistry.
Results The analysis revealed eleven subsets of monocytes/macrophages, six $CD4^+$ T cell subsets, eight $CD8^+$ T cell subsets, and five B cell subsets. A pro-angiogenic macrophage population associated with NF-κB signaling was enriched in tumors with poor clinical outcomes. In contrast, an interferon-primed macrophage subset that recruits T cells via CXCL9/10/11 was linked to favorable survival.

CD8⁺ T cells demonstrated diverse roles in tubo-ovarian cancer: exhausted T cells (Tex) contributed to immune dysfunction and poorer prognosis, while tissue-resident memory T cells (Trm) in early-stage tumors were associated with effective immune surveillance. Additionally, co-stimulatory and co-inhibitory receptor interactions were mapped, and B cell subsets were found to have distinct prognostic implications.
Conclusion This integrative single-cell study offers a detailed landscape of immune cell heterogeneity and function within the TIME of major gynecologic cancers. The findings provide novel insights into immune regulation and potential biomarkers, offering valuable guidance for improving and personalizing immunotherapeutic strategies in these malignancies.

OR_018

负载纳米马达驱动型细胞外囊泡的水凝胶在治疗化疗诱导卵巢损伤中的应用研究

张欣怡
武汉华中科技大学同济医学院附属同济医院

目的 化疗药物在治疗恶性肿瘤的同时会损伤卵巢组织，导致早发性卵巢功能不全（POI）。本研究构建了负载纳米马达驱动型细胞外囊泡（LEV）的水凝胶系统（H-LEV），用于治疗化疗引起的卵巢损伤，并探索其机制。
方法 首先，通过胆固醇-L-精氨酸膜锚定技术将 L-精氨酸修饰至 EV 表面，形成 LEV，并利用透射电镜、蛋白印迹（WB）和纳米流式等技术对其进行表征。通过检测活性氧（ROS）水平、一氧化氮（NO）产量等验证 LEV 的体外功能和运动能力。随后，合成温度响应性水凝胶，并通过扫描电镜表征其性能。用水凝胶负载 LEV 后，通过 Transwell 培养、ROS 检测和细胞凋亡实验验证 H-LEV 的释放动力学和对 POI 细胞模型的修复能力。在体内实验中，向顺铂诱导的 POI 模型小鼠卵巢囊内原位注射 H-LEV，通过小动物成像仪观察释放动力学，并通过卵泡计数和激素水平检测评估治疗效果和安全性。最后，通过转录组测序和生信分析探究 H-LEV 的治疗机制，并利用 WB 和免疫组化染色验证相关通路。
结果 本研究成功构建的 LEV 能有效降低 POI 细胞模型中的 ROS 水平、一氧化氮合酶（NOS）活性和细胞凋亡比例，同时通过产生 NO 增强运动能力。水凝胶在 37℃下迅速成胶，具有多孔结构和良好机械性能。H-LEV 能持续 21 天稳定释放 LEV，显著改善 POI 细胞模型的氧化应激和凋亡水平。体内实验显示，H-LEV 在卵巢周围富集并缓慢释放，显著提高 POI 小鼠的卵泡数量、性激素水平和生育力，且安全性良好。转录组分析表明，H-LEV 治疗后卵巢中氧化应激相关通路和 HIF-1 信号通路基因上调。验证实验证实，H-LEV 通过激活 Nrf2 通路上调抗氧化酶（SOD2、GSS、GPX1）表达，减少细胞凋亡；同时激活 MEK/ERK/HIF-1/VEGF 通路，促进微血管生成，两者协同改善了卵巢微环境。
结论 本研究成功构建了 H-LEV 系统，其负载的 LEV 能响应和降低 ROS 水平，并产生 NO 驱动自身运动。H-LEV 提高了 LEV 在体内的储留时间，显著改善化疗损伤小鼠的卵巢功能和生育力，其机制涉及 Nrf2 通路和 MEK/ERK/HIF-1/VEGF 通路的协同作用。

OR_019

Evaluation of Individualized 3D Brachytherapy Strategies Based on HR-CTV Maximum Cross-Sectional Area: A Single-Center Retrospective Study

Chengdian He
重庆大学附属肿瘤医院

Objective To explore the influence of the maximum cross-sectional area (MCSA) on the individualized selection of three-dimensional brachytherapy (3D-BT) techniques for cervical cancer, by comparing the correlation between different structural parameters of the high-risk clinical target volume (HR-CTV) and 3D-BT treatment techniques. This study aims to identify the MCSA threshold that guides clinical decision-making.
Methods This retrospective study included 30 cervical cancer patients (160 treatment plans) receiving radical radiotherapy from September 2024 to January 2025. Patients were divided into three groups based on the brachytherapy technique: T+2O+IS BT (47 plans), T+2O BT (56 plans), and T+IS BT (57 plans). Spearman rank correlation and unpaired t-tests assessed the relationship between HR-CTV parameters (MCSA, volume, maximum lateral diameter (MLD), maximum anterior-posterior diameter (MAPD), and maximum cranio-caudal diameter (MCCD)) and treatment techniques. A Two-Dimensional Grid Search combined with Chi-Squared Test determined the MCSA threshold.Additionally, a simulation was performed to remove interstitial needles from the T+2O+IS BT group, comparing dose differences between T+2O+IS-Without needles BT and T+2O+IS BT.
Results MCSA showed the strongest correlation with the T+2O+IS BT group ($r=0.74$, $P<0.0001$). When MCSA ≥ 18 cm², the T+2O+IS BT group had significantly lower bladder D2cc doses compared to the T+2O BT group (341.72 ± 41.63 vs. 393.41 ± 48.45 cGy, $P<0.0001$). Similar reductions were observed for rectum D2cc (315.63 ± 54.40 vs. 342.38 ± 57.14 cGy, $P<0.0001$) and sigmoid colon D2cc (207.31 ± 68.04 vs. 236.20 ± 78.73 cGy, $P<0.0001$). No significant dose differences were found for OARs when MCSA < 18 cm² ($P>0.05$).
Conclusion MCSA is a key parameter for selecting 3D-BT techniques. When MCSA ≥ 18 cm², T+2O+IS BT is recommended for better target coverage and reduced OAR doses. When MCSA < 18 cm², T+2O BT is sufficient without significant OAR risk increase.

OR_020

基于不同分期探究外阴癌淋巴结转移相关因素分析

张冰心
山东大学齐鲁医院

研究目的 淋巴结转移（LNM）是外阴癌独立预后因素。研究证实，LNM 患者 5 年生存率显著降低，淋巴结状态是手术分期核心指标。目前淋巴结转移相关因素探究数据有限，且局限于总人群，缺乏基于不同分期的因素探究。
据 NCCN 指南，T1a 患者建议行单纯外阴部分切除，不推荐行淋巴结清扫，筛选危险因素，具备淋巴结转移高危因素患者，可推荐行前哨淋巴结切除术（SLNB），以避免漏诊；T1b、肿瘤≤4cm 的 T2 患者，建议行淋巴结切除/SLNB，但目前两种淋巴结评估方式无选择标准，且淋巴结切除术相关并发症多，可筛选出此人群中危险因素，推荐高危患者行淋巴结切除，低危患者患者行 SLNB；T2 大病灶（＞4cm）、T3 患者，建议行腹股沟-股淋巴结切除术评估淋巴结状态后行同步放化疗，目前有研究探究 SLNB 可行性，筛选出低危患者建议行 SLNB，以避免非转移淋巴结的过度切除。

材料与方法 本研究是一项基于全国多中心的回顾性研究，数据来源于齐鲁医院、辽宁省肿瘤医院、郑州大学第一附属医院等七家中心，共纳入 1328 例外阴癌患者。采用单、多因素 COX 回归分析，基于不同分期分析以下临床病理因素：年龄、吸烟、肿瘤大小、肿瘤位置、肿瘤病理分级、HPV 状态、免疫指标（P16、Ki-67 等）。排除影像学、术中探查提示淋巴结转移、未行淋巴结清扫及非早期患者。
结果 在临床 T3 分期前的外阴癌患者单变量分析中，吸烟（$p = 0.001$），肿瘤＞4cm（$p < 0.001$），病理分级（中、低分化）（$p < 0.001$），P16 阳性（$p < 0.001$）和淋巴结转移显著相关。
结论 T1b、肿瘤≤4cm 的 T2 患者中，肿瘤＞4cm、肿瘤低分化、P16 阳性的淋巴结转移风险高，此部分患者建议选择腹股沟-股淋巴结切除术；T2 大病灶（＞4cm）、T3 患者中，吸烟、阴蒂位置肿瘤、肿瘤低分化的淋巴结转移风险高，不建议此部分患者行 SLNB，应行腹股沟-股淋巴结切除术后同步放化疗。

OR_021

Exploring molecular heterogeneity and therapeutic targets of metastatic/recurrent cervical cancer based on deep analysis of large panel genes

Yukai Zhu* Lei Wang Xingtao Long Dongling Zou
Chongqing University Cancer Hospital

Purpose Over 50% patients of advanced cervical cancer experience disease recurrence. Due to genomic evolution, metastatic/recurrent tumors may harbor new driving mutations or resistance mechanisms, and its molecular characteristics need to be systematically analyzed. This study combined multi-dimensional data (genomic, demographic, and therapeutic) to elucidate heterogeneity in treatment responses and validate predictive biomarkers in metastatic/recurrent cervical cancer management.
Methods A prospective whole-exome sequencing (WES) analysis was performed on metastatic/recurrent cervical cancer specimens using BGI's NGS platform. Immunohistochemical detection of PD-L1 was performed on tumor tissue. Clinical data, including histology, stage at diagnosis, treatment history, and survival status were obtained from medical records.
Results A total of 302 patients with metastatic/recurrent cervical cancer (squamous, 209; adenocarcinoma, 31; gastric type, 35; neuroendocrine, 11; and other, 17) underwent next-generation sequencing genetic testing. The most prevalent genomic alterations in PIK3CA (39%), KMT2C (26%), KMT2D (21%), MUC16 (19%),FBXW7(15%),TP53(15%) and EP300(13%). Squamous cell carcinoma (SCC) showed significantly higher mutation rates in PIK3CA (45%), KMT2C (29%), and MUC16 (22%) than adenocarcinoma (AC). The difference in KMT2C mutation frequency was particularly notable (29% in SCC vs 6% in AC; p=0.014).Conversely, adenocarcinoma showed elevated mutation rates in KRAS and TP53 relative to squamous cell carcinoma (KRAS: 19% vs 4%, p<0.001; TP53: 35% vs 5%, p<0.001) .CDKN2A (15.9% vs. 0%, p=0.003) and SMARCA4 (13.6% vs. 3.1%, p=0.024) mutations were significantly enriched in non-HPV-associated adenocarcinomas compared to HPV-associated cases, suggesting distinct molecular mechanisms between these subtypes. Among patients with high tumor mutational burden (TMB >10 mutations/Mb), which accounted for 15.9% of cases, two were also identified as microsatellite instability-high (MSI-H).
Germline mutation analysis in patients with cervical cancer identified 11 carriers, including two with hereditary cancer syndromes (Lynch syndrome/MSH2 and HRD-related BARD1), featuring predominantly pathogenic variants (splicing/frameshift/nonsense) in HRD pathway genes (ATM/PALB2/RAD51B/BARD1/FANCA). PD-L1 was positive (CPS≥1) in 89.8% of 49 cases (5 CPS＜1，28 CPS 1-10, 16 CPS≥10). Squamous cell carcinoma has the highest positivity rate，96.1% (25/26), HPV-related adenocarcinoma was 83.3%（5/6）, Non-HPV-related adenocarcinoma was 82.4%%（14/17）.

Among 77 TMB-H patients, 20 received immunotherapy，the DCR was 66.7% (1 CR, 5 PR, 6 SD). In contrast, among 207 TMB-L patients, 26 received immunotherapy, with a DCR of 38% (2 CR, 5 PR, 3 SD) .
Genomic analysis identified cervical cancer's high-frequency mutations predominantly enriched in nucleoplasm, chromatin remodeling, and DNA binding pathways. Subtype-specific profiling revealed squamous cell carcinomas and gastric-type adenocarcinomas shared nuclear transcriptional regulation pathways, while adenocarcinomas uniquely activated EGFR/PI3K-AKT signaling. KEGG pathway analysis demonstrated conserved signal transduction mechanisms with squamous cell carcinomas exhibiting distinct chromosome regulation patterns.
Conclusions Our comprehensive genomic profiling of 302 metastatic/recurrent cervical cancer patients revealed significant molecular heterogeneity across histological subtypes, with distinct mutation patterns and pathway activations. PIK3CA alterations were prevalent across all subtypes, reinforcing its role as a key oncogenic driver. Squamous cell carcinomas exhibited unique mutation profiles in TP53, KRAS, and KMT2C compared to adenocarcinomas, while non-HPV-associated adenocarcinomas showed enrichment in CDKN2A and SMARCA4 mutations, suggesting divergent tumorigenic mechanisms. Additionally, a subset of patients harbored germline pathogenic variants in HRD-related genes, including two cases of hereditary cancer syndromes, highlighting the potential clinical relevance of genetic counseling and testing.
Immunotherapy response analysis indicated a trend toward improved disease control rates (DCR) in TMB-high (TMB-H) patients (50% vs. 39%). The high PD-L1 positivity indicating the effectiveness of immunotherapy for cervical cancer. Pathway analysis underscored the importance of chromatin remodeling and nuclear transcriptional regulation in squamous and gastric-type adenocarcinomas, while adenocarcinomas uniquely relied on EGFR/PI3K-AKT signaling.
These findings emphasize the need for histology-specific therapeutic strategies and biomarker-driven approaches, including PI3K inhibitors, HRD-targeted agents, and immunotherapy, to optimize precision treatment in advanced cervical cancer. Further prospective studies with larger cohorts are warranted to validate these observations and refine patient stratification.

OR_022

Genome-wide and Multi-omics Mapping of HPV Integration Signatures in Cervical Pre-Invasive and Invasive disease

Junpeng Fan Tao Zhang Ding Ma Chaoyang Sun Xiaoyuan Huang*
Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology

Cervical cancer, caused by the high-risk HPV, remains a significant global public health concern. HPV integration is a critical step in the development of cervical carcinogenesis. HPV capture sequencing on a large population (n=12,719) was constructed and multi-omics evidence from a cervical cancer cohort (n=96) was used for validation. The landscape of high-risk HPV infections and integration for a population of 12,719 cases was constructed. Characterizing HPV integration signatures at various stages of cervical lesions, we identified two HPV integration signatures (chromosome 21: 8,000,000–9,000,000 associated integration model (Chr21-INT MOD) and JAK/STAT-associated integration model (JAK/STAT-INT MOD)) that appeared to progress with the escalation of the lesion grade. Multi-omics analysis revealed that these two HPV-integrated signatures displayed similar expression profiles in the HPV genome (upregulation of E1/E2/E4/E5) and host genome (downregulation of the JAK/STAT pathway). Additionally, these signatures correlated with an immunosuppressive microenvironment. The findings provide insights into HPV integration, contributing to its potential application in the risk stratification and precision treatment of cervical lesions.

OR_023

Preliminary Study on Prediction of Immune Microenvironment and Prognosis of Cervical Cancer Patients Based on MRI Radiomics before Treatment

zaiyu Li ya Zhang zheng Li*
The Third Afhlated Hospital of Kunming Medical University,Yunnan Cancer Hospital, Peking University Cancer Hospital Yunnan

Objective (s) Patients with early-stage cervical cancer generally have a favorable prognosis, whereas those with recurrent metastasis face a significantly poorer outlook. Immune checkpoint inhibitors (ICIs) are now standard first- and second-line therapies for recurrent metastatic cervical cancer, relying on effector cells in the immune microenvironment, particularly CD8+ T cells and CD68+ macrophages. However, a non-invasive method to assess the immune microenvironment before treatment, which could predict patient response to immunotherapy, remains unavailable. Recent studies suggest that pre-treatment MRI-based imaging histology might predict the immune microenvironment's status and patient prognosis. This study analyzed clinicopathological data and MRI multisequence imaging of cervical cancer patients pre-treatment, constructing a radiomics model to predict CD8+ T cell and CD68+ macrophage expression in tissue specimens. This model aims to support the development of a non-invasive tool for predicting cervical cancer prognosis and immunotherapy responsiveness.

Methods

1. This study retrospectively included patients diagnosed with cervical cancer who underwent radical treatment at our hospital from January 1 to December 31, 2016. We collected their clinicopathological, follow-up, and pre-treatment MRI data after applying inclusion and exclusion criteria. Immunohistochemical staining was conducted on the complete paraffin-embedded pathology sections to score the expression of CD8+ T lymphocytes and CD68+ macrophages. The correlation between these expression levels and clinicopathological factors was analyzed using the chi-square test. Additionally, the relationship between expression levels and patient prognosis was examined using COX regression analysis and Kaplan-Meier curves.

2. The radiomics model construction involved collecting T2WI, DWI, and DCE-MRI phase imaging data from patients' MRI scans taken within two weeks before treatment. These data, saved in DICOM format, were imported into 3D Slicer 5.6.1 software. Radiologists delineated the tumor region's region of interest (ROI) and extracted imaging histology features. Features from each sequence's ROIs were extracted and normalized using the FAE (0.6.0) software in Python (3.7.6) via arithmetic mean. Subsequently, features underwent PCC downscaling and further selection using RFE.

2.1 Construction of Radiomics Model for Predicting CD8+ T Cell and CD68+ Macrophage Expression: CD8+ T cells and CD68+ macrophages were categorized into low and medium-high expression groups based on immunohistochemistry results. Patients were split into training and test sets in a 7:3 ratio. A support vector machine (SVM) classifier was employed to optimize model hyperparameters via ten-fold cross-validation. The imaging histology model was developed to examine its correlation with CD8+ T cell and CD68+ macrophage expression in pathology sections. Model efficacy was assessed using the area under the curve (AUC), sensitivity, specificity, accuracy, positive predictive value, and negative predictive value.

2.2 Construction of Radiomics Prognosis Model: A ten-fold cross-validation approach was employed to develop the radiomics prognosis model using a logistic regression classifier. Given the limited sample size, the dataset was not split into separate training and test sets; instead, the entire dataset was used for model construction and internal validation through cross-validation. Model performance was evaluated using metrics such as AUC, sensitivity, specificity, accuracy, positive predictive value, and negative predictive value.

Results

1. A total of 144 cervical cancer patients meeting the nadir criteria were included in the study. Correlation analysis revealed a significant association between CD8+ T cell expression levels and lymphovascular mesenchymal infiltration, as well as overall and progression-free survival. Patients with elevated CD8+ T cell expression exhibited significantly longer progression-free and overall survival.

2. Three single-sequence models and one multiple-sequence fusion model were developed based on CD8+ T cell expression levels. The T2WI sequence model achieved a test set AUC of 0.694 (0.5267-0.8602), demonstrating high specificity (0.8261) but low sensitivity (0.4000), negative predictive value (0.6129), positive predictive value (0.6667), and accuracy (0.6279), indicating limited diagnostic efficacy. The DWI model showed an improved test set AUC of 0.728 (0.5706-0.8855), with increased sensitivity, accuracy, and positive predictive value compared to the T2WI model, albeit with reduced specificity and negative predictive value. The DCE-MRI model had a test set AUC of 0.6652 (0.4995-0.8310), with lower sensitivity, specificity, negative predictive value, and positive predictive value, but a higher negative predictive value than the other models. The fusion model, combining T2WI, DWI, and DCE-MRI sequences, achieved a test set AUC of 0.8022 (0.6549-0.9495) and significantly higher specificity (0.9565) than individual sequences. This imaging histology model, based on 2,553 features from preoperative MRI sequences, more accurately reflects CD8+ T cell infiltration in tumor tissue sections. In the radiomics models based on CD68+ macrophage expression, the T2WI sequence model achieved a test set AUC of 0.6190 (95% CI: 0.4388-0.8043), demonstrating high sensitivity (0.75), a positive predictive value of 0.75, and the highest accuracy (0.6744) among the models, albeit with lower specificity (0.5333) and negative predictive value (0.5333). The DWI sequence model exhibited a test set AUC of 0.650 (95% CI: 0.4826-0.8174), surpassing the T2WI model in overall AUC but falling short in sensitivity, specificity, accuracy, and predictive values. The DCE-MRI sequence model showed a test set AUC of 0.554 (95% CI: 0.3764-0.7323), indicating poor diagnostic performance. The combined model of all three sequences yielded a test set AUC of 0.672 (95% CI: 0.5151-0.8373), with enhanced sensitivity, accuracy, and positive predictive value, though specificity and negative predictive value remained low. Overall, the model effectively predicted CD68+ macrophage infiltration.

3. Four radiomics models were developed based on patients' 5-year overall survival. The T2WI model demonstrated a validation set AUC of 0.860 (95% CI: 0.7241-0.9952), with high specificity (0.9618), accuracy (0.9236), and negative predictive value (0.9545), but lower sensitivity (0.5385) and positive predictive value (0.5833). The DWI sequence model had a validation set AUC of 0.754 (95% CI: 0.5696-0.9383), also showing high specificity, accuracy, and negative predictive value, yet lower sensitivity and positive predictive value. Similarly, the DCE-MRI sequence model exhibited a test set AUC of 0.754 (95% CI: 0.5696-0.9383), with comparable results to the DWI model. The combined model of all three sequences achieved a validation set AUC of 0.903 (95% CI: 0.7955-1.0000), with enhanced specificity, accuracy, and negative predictive value, and significantly improved sensitivity and negative predictive value compared to the other models. This suggests the combined model is highly effective in predicting 5-year cervical cancer survival, although it requires external validation.

Conclusion(s)

1. The radiomics model utilizing 2,553 features derived from T2-weighted imaging (T2WI), diffusion-weighted imaging (DWI), and dynamic contrast-enhanced (DCE) sequences of pre-treatment MRI more accurately represents CD8+ T cell infiltration in tumor tissue sections.

2. The pre-treatment MRI histology model effectively predicted CD68+ macrophage infiltration.

3. Utilizing T2WI, DWI, and DCE-MRI sequences, this model may also predict the 5-year overall survival of cervical cancer patients, potentially due to its capacity to forecast CD8+ T-cell infiltration in the immune microenvironment. However, external validation and further evaluation are necessary.

OR_024

CS Ratio is an immune-related prognostic biomarker for cervical cancer

Peiqin Shi Pengpeng Qu*
TIANJIN CENTRAL HOSPITAL OF GYNECOLOGY OBSTETRICS

Background The tumor microenvironment (TME) plays a crucial role in cancer progression, but its complex structure and significant variability among patients present considerable challenges for research. Recent studies have demonstrated that macrophage polarization states, defined by the expression levels of CXCL9 and SPP1 (CS Ratio), are more prognostically relevant than traditional M1/M2 markers. The CS polarization state reflects a highly coordinated network of pro-tumor and anti-tumor variables, offering a simplified yet effective immune response indicator for the complex TME. The CS Ratio has been shown to correlate with the abundance of anti-tumor immune cells, the gene expression programs of tumor-infiltrating cells, and responses to immunotherapy.
Cervical cancer, one of the most common gynecological malignancies, still faces limited therapeutic options. CXCL9, a member of the CXC chemokine family, plays a critical role in immune regulation, inflammation, tumor growth, angiogenesis, and metastasis. Similarly, SPP1, a cytokine, influences immune-related pathways by regulating molecules such as interferon-γ and interleukin-12. However, no studies have systematically investigated the role of the CS Ratio in cervical cancer or its relationship with immunotherapy characteristics. Research in this area could provide critical insights into the role and clinical potential of the CS Ratio in cervical cancer and related tumors.
Methods The expression ratio of CXCL9 to SPP1 was analyzed in cervical cancer patients using data from the Gene Expression Omnibus (GEO) database, which revealed significant differences. Data for cervical cancer patients were obtained from The Cancer Genome Atlas (TCGA) database. The optimal cutoff value for the CS Ratio was determined using the maxstat package in R, and Kaplan-Meier (KM) survival curves were constructed. Patients were categorized into High and Low groups based on the median CS Ratio. Immune scores were analyzed, and immune cell infiltration was assessed using CIBERSORT. Differences in the CS Ratio were evaluated across patients with varying pathological T stages and FIGO stages. Additionally, receiver operating characteristic (ROC) analysis was performed using the pROC package in R to calculate the area under the curve (AUC). Univariate and multivariate Cox regression analyses were performed to evaluate the potential of the CS Ratio as an independent prognostic factor in cervical cancer. A Cox regression-based nomogram integrating four key features was subsequently developed for the TCGA-CESC cohort. Nomogram performance was assessed using calibration curves and ROC analysis.
Results The CS Ratio was significantly lower in cervical cancer patients compared to normal controls ($P < 0.05$). KM survival curves indicated that patients in the CS High group exhibited better prognoses. Immune score analysis revealed significantly higher immune scores ($P < 0.05$) and lower tumor purity ($P < 0.05$)in the CS High group compared to the Low group. CIBERSORT analysis revealed significantly higher proportions of CD8+ T cells ($P < 0.05$) and M1 macrophages ($P < 0.05$), and a significantly lower proportion of M2 macrophages ($P < 0.05$), in the CS High group compared to the Low group. The CS Ratio significantly decreased with advancing FIGO stage ($P < 0.05$). Both univariate ($P < 0.05$) and multivariate Cox regression analyses ($P < 0.05$) confirmed the CS Ratio as an independent prognostic factor. ROC analysis demonstrated that the CS Ratio had higher AUC values for predicting 1-year (AUC=0.69), 3-year (AUC=0.66), and 5-year OS (AUC=0.68) than CXCL9 or SPP1 alone. The Cox regression-based nomogram integrating four key features demonstrated predictive capability for 1-, 3-, and 5-year OS in CESC patients (Concordance Index = 0.751; 95% CI: 0.678–0.824; p = 1.50Í10-11). Significant survival differences were observed between the high-risk and low-risk groups based on the nomogram score. ROC analysis yielded high AUC values for survival prediction: 0.85 (95% CI: 0.94-0.75) at 1-year, 0.74 (95% CI:0.84-0.64) at 3-year, and 0.72 (95% CI:0.84-0.61) at 5-year.

Conclusion The CS Ratio may serve as a more effective prognostic biomarker for cervical cancer patients.

OR_025

宫颈癌中等分割在线自适应放疗的疗效及安全性分析：一项前瞻性单臂 I 期临床研究

曾政 晏俊芳 胡克 张福泉
中国医学科学院北京协和医院

研究目的 开展宫颈癌中等分割在线自适应放疗（oART）的前瞻性 I 期临床研究，评估患者初步疗效及急性毒副反应。
材料与方法 前瞻性入组病理确诊的宫颈癌患者 30 例，所有受试者接受每天 oART，临床靶区（CTV）处方剂量为 43.35Gy/17 次，阳性淋巴结则采用同步推量，剂量为 54.40Gy/17 次。CTV 分开勾画并根据不同解剖结构进行 5-10 mm 外放。CT/MRI 引导的三维近距离放疗，处方剂量 30Gy/5 次，同步顺铂 40mg/m2 周疗，至少 3 周期。肿瘤疗效和毒副反应分别通过 RECIST 1.1 和 CTCAE 5.0 标准评价分级，通过欧洲癌症研究与治疗组织生命质量核心量表（EORTC-QLQ-C30）和宫颈癌特异性生命质量量表（EORTC-QLQ-CX24）报道患者不同治疗时间点的生活质量。
研究结果 30 例患者完成中等分割 oART 联合近距离治疗，外照射中位时间为 22 天（范围：22-27 天），总治疗中位时间为 43 天（范围：38-55 天）。外照射完成时肿瘤平均退缩率为 83.27%，3 个月的临床完全缓解率为 100%。中位随访 13.0 个月，1 年的总生存率率和无病生存率分别为 100%和 93.2%。急性毒性反应中，3 级胃肠道毒性发生率为 10%，无 3 级及以上泌尿系毒性。生活质量方面，中等分割 oART 期间在功能领域得分显著降低，在症状领域得分显著升高，在近距离治疗期间开始改善，治疗后 1 个月和 4 个月逐步恢复基线水平甚至更高。
研究结论 宫颈癌中等分割放疗联合近距离治疗具有缩短治疗时间的优势，且疗效和毒性反应可接受。然而，仍需长期随访和大样本多中心研究进一步验证其应用前景。

OR_026

CIN3 外切缘阳性与进一步切子宫后的 HPV 延迟清除有关

任叶丹 许君芬*
浙江大学医学院附属妇产科医院

研究目的 宫颈上皮内瘤变 3 级（CIN3）的患者行宫颈锥切术后，锥切组织切缘阳性常提示宫颈仍有残留病灶或 HPV 持续感染的风险增加。本研究旨在探讨宫颈锥切术后 CIN3 切缘阳性并行子宫切除术的患者术后 HPV 清除情况，并评估 HPV 清除延迟的影响因素。
材料与方法 本研究回顾性纳入 2014–2024 年间因宫颈上皮内瘤变行宫颈锥切术，术后诊断 CIN3 并因 CIN3 切缘阳性进一步行子宫切除术或残余宫颈切除术的 288 例患者。所有患者在子宫切除术或残余宫颈切除术后的 3–12 个月接受了术后的第一次 HPV 检测。按术后首次 HPV 检测结果分组，构建多因素 Logistic 回归模型分析术后第一次复查 HPV 仍为阳性的潜在危险因素，并辅以亚组分析与 Kaplan–Meier 清除曲线评估术后 HPV 转变动态。
结果 术后首次随访中，17.4%的患者 HPV 持续阳性。单因素分析显示，持续 HPV 阳性与锥切组织外切缘 CIN3 阳性、年龄较大及孕次较少显著相关（均 $p<0.05$）。多因素分析结果则指出，锥切组织外切缘 CIN3 受累是持续 HPV 感染的唯一独立预测因素（OR 4.47，95% CI 1.30–15.54，$p=0.016$）。

这一不良影响在以下人群中更为明显：年龄≥50 岁、感染非 HPV 16/18/45 型、绝经后、未保留卵巢以及接受微创手术的患者（均 p<0.05）。多数患者在术后两年内实现 HPV 清除。
结论 外侧切缘阳性显著关联术后 HPV 清除延迟，提示对高风险患者应加强个体化术后随访与管理，以降低 HPV 持续感染引起病变的风险。

OR_027

ZBTB5 enhances the resistance of cervical cancer to paclitaxel by regulating BCL6

Yue He Yunfan Wang Zhenyou Liu Chen Ji Yang Liu Yan Wang Yumei Wu*
1.Beijing Obstetrics and Gynecology Hospital, Capital Medical University. Beijing Maternal and Child Health Care Hospital

PURPOSE Preliminary clarification of the mechanism of paclitaxel (PTX) resistance in cervical squamous cell carcinoma with ZBTB5.
METHOD Using cell function experiments, such as plate imaging time, 3-(4, 5-Dimethylthiazol-2-yl)-2, 5-diphenyltetrazolium bromide (MTT) cell proliferation, and FACS-based single staining to detect apoptosis performance. Then, the downstream mechanism of ZBTB5-mediated resistance to paclitaxel was examined using Ingenuity Pathway Analysis (IPA) bioinformatics analysis and the qPCR analysis was used to validate its downstream genes. ChIP assay and Dual-Luciferase tests were performed to verify the mechanism of ZBTB5 acting on downstream factors.
RESULTS (1) Overexpression of ZBTB5 culture was obtained in PTX, and immunofluorescence showed that overexpression of ZBTB5 enhanced cervical squamous cell carcinoma resistance to PTX by functioning in the cell nucleus. (2) ZBTB5 knockdown inhibited cell cloning and proliferation and enhanced apoptosis of cervical squamous carcinoma cells in PTX and reduced resistance to PTX in SiHa cells compared with the non-knockdown group (p<0.05). (3) Through whole-genome microarray and IPA bioinformatics analysis, there were 609 differential genes compared with NC group after the deletion of ZBTB5. Based on IPA analysis, the classical signal pathway showed that the significantly activated signal pathway was HGF signal pathway, of which five differential genes, integrin, PXN, FLG-γ, PI3K and MEKK are important factors in this pathway. Analysis of upstream regulatory factors showed that let-7 was predicted to be strongly activated, and there were 13 uniformly activated genes in this gene, TGFB1 was predicted to be strongly inhibited, and there were 48 uniformly suppressed genes in this gene, BCL6 was among them. The change of differential gene expression level based on IPA analysis showed that apoptosis of fiber last cell lines was most significantly activated, and PML and SERPINB 3/4 were key genes. Based on IPA analysis, the regulation network ranking first in the regulation effect shows that the data set may affect the gene function of ZBTB5 due to the activation of the regulator aplasia or hydroplasia by the differential gene AREG and other genes such as MMP14. (4) A ChIP assay was performed after overexpression of ZBTB5, which suggested that the BCL6 gene is located 395 bp from the TSS region and may be regulated by ZBTB5, which may be the promoter of BCL6. This result was consistent with that of the IPA bioinformatics analysis. (5) Dual-Luciferase test show that ZBTB5 can serve as an activation transcription factor for the BCL6 gene.
CONCLUSION ZBTB5 is involved in PTX resistance in cervical squamous cell carcinoma by regulating BCL6. It was hypothesized that ZBTB5 may regulate BCL6 as an activation transcription factor to affect tumor cell proliferation and participate in PTX resistance in cervical squamous cell carcinoma.

OR_028

Effect of laparoscopic para-aortic and pelvic lymphadenectomy for patients with FIGO stage IIb-IIIC cervical cancer before CCRT：A single-center retrospective study

Qin Zhang
Chongqing university cancer hospital

Objectives The first aim of this study was to assess the efficacy of laparoscopic para-aortic and pelvic lymphadenectomy prior to concurrent chemoradiation therapy(CCRT) in patients with cervical cancer FIGO stages IIb-IIIC. The second aim was to to evaluate the main influencing factors to predict the patient's prognosis.
Methods In this retrospective study, 396 patients who completed their therapy at Chongqing university cancer hospital between April 2014 and April 2020 were included. 97 patients who underwent laparoscopic para-aortic and pelvic lymphadenectomy before CCRT were defined as the surgical staging group. 299 patients who underwent CCRT were defined as the clinical staging group. Kaplan-Meier and log-rank methods were used to establish and compare progression free survival (PFS) and overall survival (OS) curves of two groups, respectively. A Cox proportional hazards model was used to assess factors affecting the patient's prognosis.
Results There were no statistical differences in baseline characteristics between the 2 groups. The average number of lymph nodes (LNs) removed in surgical staging group was 35.4±13.2. The five-year PFS and OS rates of the surgical staging group were slightly lower than those of the clinical staging group (67.0% vs. 73.6% and 76.3% vs. 79.9%, respectively; $p > 0.05$). For patients with LNs metastases, the five-year PFS and OS rates of the surgical staging group were lower than those of the clinical staging group (50.9% vs. 64.0% and 64.2% vs. 68.3%, respectively; $p > 0.05$). For patients with negative LNs, the five-year PFS and OS rates were similar in surgical staging group and the clinical staging group (86.4% vs. 84.8% and 90.9% vs. 90.6%, respectively; $p > 0.05$). Univariate analysis indicates that histology types including adenocarcinoma and adenosquamous carcinoma, pelvic wall involved, higher squamous cell carcinoma (SCC) antigen , positive LNs were found to be poor prognosticators for PFS and OS($p<0.05$). Multivariate analysis identified that Adenocarcinoma($p = 0.015$, HR: 2.665 [1.211, 5.865]), Pelvic wall involved($p = 0.003$, HR: 1.826 [1.222, 2.730]), and positive LN ($p = 0.000$, HR: 2.956 [1.889, 4.627]) were independent risk factors for the PFS. Adenocarcinoma($p =0.046$, HR: 2.375 [1.017, 5.549]), Adenosquamous($p = 0.047$, HR: 3.653 [1.014, 13.157]), pelvic wall involved($p = 0.006$, HR: 1.861 [1.192, 2.906]), and positive LNs ($p = 0.000$, HR: 2.963 [1.798, 4.849]) were independent predictors of the OS.
Conclusions In patients with stage IIb-IIIC cervical cancer, laparoscopic para-aortic and pelvic lymphadenectomy before CCRT can't improve the patient's PFS and OS. Tumor histology, pelvic wall involved, and para-aortic and pelvic lymph nodes (LNs) metastasis are the main factors that affect the prognosis.

OR_029

宫颈癌前哨淋巴结不同转移对预后的影响：一项多中心真实世界回顾性研究

吴忱[1] 侯弘毅[2] 吕雪琰[1] 李斌[1*]
1.中国医学科学院肿瘤医院 2.北京大学人民医院

研究目的 回顾性分析 IA2-IIA1 期宫颈癌前哨淋巴结不同转移对患者预后的影响。
材料与方法 研究对象为 2015 年 11 月-2020 年 11 月中国医学科学院肿瘤医院和北京大学人民医院妇科收治的 91 例需手术治疗的早期子宫颈癌（IA2-IIA1，2014FIGO 分期）患者，术前影像评估均无淋巴结转移。术中分别于宫颈注射纳米炭或吲哚菁绿进行前哨淋巴结(SLN)示踪并切除,术中将切除的 SLN 行快速冰冻病理检测,无论 SLN 冰冻病理结果如何,均进行系统性盆腔淋巴结切除术(PLND)。所有手术切除的淋巴结均接受常规病理学检查和免疫组织化学染色,病理结果阴性的 SLN,按照简易超分期方法切片,分别行 H&E 染色和 AE1/AE3 免疫组化染色。SLN 转移分为大体转移(MA; 转移灶> 2mm);微小转移(MM; 转移灶 0.2~2mm);孤立肿瘤细胞转移(ITC; 转移灶< 0.2mm)。回顾性收集患者的临床资料和肿瘤病理特征，并随访其生存状态及复发情况。利用 Kaplan-Meier 曲线和 Log-Rank 检验进行分析，通过 Kernel 函数评估前哨淋巴结转移风险因素，多因素 Cox 比例风险回归分析前哨淋巴结不同转移状态与 PFS 和 OS 之间的关系。
结果
（1）91 例患者中，术中前哨淋巴结单侧显影 34 例（37.4%），双侧显影 57 例（62.6%），显影成功率 100%，每例患者 SLN 中位数 4（2，6）个，切除的盆腔淋巴结中位数 30（24，38）个。SLN 活检阴性预测值 100%，准确性为 100.0%。
（2）前哨淋巴结术后病理超分期结果，阴性 77 例（84.6%），孤立肿瘤细胞 1 例（1.1%），微小转移 5 例（5.5%），大体转移 8 例（8.8%）。常规病理学染色合并病理超分期检测淋巴结转移的检出率为 15.4%(14/91)。宫颈癌浸润深度大于 8mm,LVSI(+),肿瘤大于 4cm 是前哨淋巴结转移的风险因素。
（3）中位随访时间 85 个月，复发 10 例（11%），死亡 7 例（7.7%）。复发和死亡的多因素分析显示只与 SLN MM 和 MAC 有关，与 ITC 无关，与其他临床病理因素无关。
结论 早期宫颈癌患者 SLN 微转移和宏转移与复发和死亡均显著相关，5 例微转移均为病理超分期发现，因此应重视 SLN 超分期，以发现微转移，密切随诊。

OR_030

建立基于多实例学习的宫颈癌预后预测模型

刘妍淇[1,2] 王宏伟[3] 杨雯[4*] 刘慧敏[2]
1.解放军医学院 2.中国人民解放军总医院第一医学中心
3.中国人民解放军总医院第四医学中心病理科 4.中国人民解放军总医院第七医学中心妇产科

背景 宫颈癌是全球女性癌症相关死亡的主要原因之一，其预后受肿瘤异质性、微环境及治疗等多因素影响。传统组织病理学评估对预后的预测存在局限，亟需更精准的量化工具。随着人工智能的不断发展，机器学习与全切片数字图像（WSI）的结合为建立预后预测模型提供了新视角。然而，WSI 分辨率高、区域异质性强，缺乏像素级标注等问题制约了模型效能。多实例学习（MIL）通过弱监督方式采用“包”级别训练方式，直接处理整张切片与患者标签的关系，为上述挑战提供了解决方案。
目的 本研究旨在开发一种基于 MIL 的病理组学预后预测模型，整合宫颈癌 WSI 的高通量特征，构建无进展生存期（PFS）和总生存期（OS）预测工具。

材料与方法 回顾性纳入解放军总医院第一医学中心 2017 年到 2023 年病理结果为宫颈癌、可以获得完整临床-病理资料的患者，经过整合与筛选，共纳入 382 例。收集其病理图像以及临床信息。随机抽取 80%的数据作为训练集，剩下的 20%作为测试集。首先使用 CLAM 算法对病理图像进行处理，将患者的 WSI 切割成小的的图像块，大小为 224x224 像素，接着用 pretrained resnet18 提取出每个图像块的高通量特征，将其聚合成包特征级表示。采用 TransMIL 的基本架构聚合形态学信息和编码空间信息，通过显式注入整个 WSI 的全局特征。计算整个 WSI 的全局特征的公式如下所示：
Gintra=AGG(x1,x2,…,xn)其中 AGG 为聚合函数
通过以上步骤获得患者级别的嵌入表征后通过 NLLSurvLoss 分类头进行预测。然后使用多因素 COX 回归对临床特征进行筛选，将显著预测因子与多实例学习结果进行融合生成 Nomogram（列线图），进行生存风险评分。使用 C-index、校准曲线、K-M 曲线对模型进行性能评估。
结果 通过五折交叉检验，模型的平均 C-index 达到了 0.71。校准曲线显示出模型拟合较好，布里尔分数约为 0.15。同时根据列线图评分，我们将患者分为高风险和低风险组，两组的 Kaplan - Meier 生存概率曲线显示出统计学差异（P<0.0001）。
结论 本研究通过弱监督方式提取 WSI 中的关键预后信息，构建了基于 MIL 的宫颈癌病理组学预后模型。在个体化治疗中为临床决策提供更有力支持。

OR_031

Targeting Cervical Small Cell Neuroendocrine Carcinoma: A High-Throughput Drug Screening Approach Using Organoids

Zhen Zheng Yang Xiang*
Department of Obstetrics and Gynecology, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences & Peking Union Medical College, National Clinical Research Center for Obstetric & Gynecologic Diseases

Background Small cell neuroendocrine carcinoma of the cervix (SCNEC) is a rare and highly aggressive subtype of cervical cancer characterized by rapid progression and dismal prognosis. The scarcity of effective treatment options and limited availability of clinical trial data have hindered therapeutic advancements. Patient-derived organoids (PDOs) offer a promising alternative to conventional cell lines and xenograft models by preserving the genetic and histological features of the original tumor. This study aimed to establish NECC organoid models and conduct high-throughput drug screening to identify candidate therapies for precision treatment.
Methods PDOs were generated from freshly resected tumor tissues of patients with cervical neuroendocrine carcinoma. Immunohistochemical staining was performed to compare the expression of neuroendocrine markers between organoids and their parental tumors. A high-throughput drug screen was then conducted using a library of 315 FDA-approved anti-cancer compounds. In addition, PDO-derived xenografts (PDO-X) were established in immunodeficient mice to evaluate tumorigenicity and translational potential in vivo.
Results Organoid cultures were successfully established from 11 NECC patient samples, achieving a 100% establishment rate. These included PDOs derived from treatment-naïve SCNEC, post-neoadjuvant chemotherapy SCNEC, and mixed neuroendocrine carcinoma subtypes. The PDOs recapitulated the histopathological and genetic characteristics of their corresponding primary tumors. Among the compounds screened, Exatecan Mesylate exhibited the most potent anti-tumor activity, achieving up to 90% growth inhibition across models. In vivo studies demonstrated a tumor formation rate of 40% in nude mice and 100% in NOG mice, confirming the robust tumorigenic capacity of NECC PDOs.
Conclusion This study presents a robust platform for modeling cervical neuroendocrine carcinoma using PDO technology. The established organoids faithfully retained the molecular features of primary tumors and demonstrated significant utility in both in vitro and in vivo settings. High-throughput screening identified Exatecan Mesylate as a promising therapeutic candidate, showing potent efficacy

in NECC organoids. The successful engraftment in immunocompromised mice further supports the translational relevance of this model for future mechanistic and therapeutic investigations.

OR_032

TNFA 在宫颈癌前病变演变中的作用及微生物驱动机制研究

胡杏媛 庄绪翠 罗弯弯 孙朝阳*
华中科技大学同济医学院附属同济医院

研究目的 宫颈癌的发生发展是一个多阶段、渐进性的过程，从正常宫颈组织逐步进展为低级别鳞状上皮内病变（LSIL）、高级别鳞状上皮内病变（HSIL），最终发展为浸润性宫颈癌。尽管高危型人乳头瘤病毒（HPV）感染是宫颈癌的主要致病因素，但宿主免疫微环境的动态变化在疾病进展中的作用尚未完全阐明。近年来，单细胞转录组测序（scRNA-seq）技术的发展为解析肿瘤微环境的异质性提供了新的视角。本研究通过整合宫颈正常组织、LSIL、HSIL 及宫颈癌的单细胞转录组数据，系统分析了肿瘤相关功能模块的动态变化，重点关注 TNFA 信号通路的演变规律，并探讨其潜在的微生物驱动机制，以期为宫颈癌的早期预警和靶向干预提供新的理论依据。
材料与方法 本研究整合了宫颈正常组织、LSIL、HSIL 及宫颈癌 68 个单细胞转录组样本，系统解析宫颈病变进程中细胞亚群的异质性特征，揭示 TNFA 信号通路在宫颈癌前病变恶性演变中的作用机制。基于差异表达基因对肿瘤细胞亚群进行功能富集分析，并采用非负矩阵分解（NMF）识别关键功能模块。通过追踪 TNFA 功能模块的动态变化，结合细胞因子芯片检测和微生物组关联分析，深入探讨 TNFA 模块与宫颈局部微生物群落之间的潜在调控关系。
结果 本研究通过非负矩阵分解（NMF）方法鉴定出 TNFA 信号通路相关功能模块，该模块在肿瘤细胞中呈现疾病进展的增加趋势。研究发现，TNFA 模块的异常活化与宫颈微生态紊乱密切相关，微生物 α 多样性指数显著降低。机制研究证实，特定微生物的代谢产物可通过激活 IKK/NF-κB 信号通路，促进巨噬细胞等免疫细胞持续分泌 TNFA。
结论 本研究确立了 TNFA 功能模块作为宫颈病变恶性进展分子标志物的临床价值，揭示了"微生物群-免疫系统-肿瘤发生"的三者互作机制，为开发基于免疫微环境调控的早期干预策略提供了重要的理论依据。

OR_033

68Ga-THP-NB1 PET/CT 在晚期/复发性宫颈癌患者中 PDL1 表达的显像效果

宋楠 郑虹*
北京大学肿瘤医院

研究目的 程序性死亡受体 1(programmed death receptor 1，PD1)和程序性死亡配体 1(programmed death ligand 1，PD-L1)抑制剂在宫颈癌治疗中起到重要作用，晚期宫颈癌治疗多是化疗联合免疫治疗，需要探索评估免疫治疗疗效的有效方式。新型 68 Ga 标记的 THP-NB1 PET/CT 可评估 PDL1 的表达，在晚期宫颈癌患者中评估 68 Ga-THP-NB1 PET/CT 的表达情况。
材料与方法 采用位点特异性放射性标记法制备 68 Ga-THP-NB1，首次人体临床转化试验。在初次诊断晚期宫颈癌患者中评价了安全性、放射剂量测定、示踪剂摄取与免疫组织化学染色情况。
结果 已入组 5 例晚期宫颈鳞癌患者，按 2018 年 FIGO 分期诊断 3 例为ⅢIc(r)期，2 例为ⅣB 期。5 例患者宫颈病灶活检进行 PDL1 免疫组化检测，4 例 CPS≥1，其中最高 80，最低 2，1 例患者 CPS＜1。平均 68 Ga-THP-NB1 注射剂量为 2.92mCi， 摄取主要见于肾脏和脾脏，肝脏、膀胱、结肠、垂体、唾

液腺、心肌也有较低摄取。辐射剂量在可靠范围内。4 例免疫组化 PDL1(+)的患者中，CPS 最高表达及最低表达患者 68 Ga-THP-NB1 的肿瘤摄取（SUVmax）分别为 8.6 和 5.2，1 例 PDL1(-)68 Ga-THP-NB1 的肿瘤摄取（SUVmax）7.2。

结论 68Ga-THP-NB1 PET/CT 在宫颈癌患者应用有良好的安全性，在 PDL1 阳性病灶中显示出较高 SUV 值，该示踪剂可以检测肿瘤中的 PD-L1 表达，它可能使预测 PD-1/PDL1 免疫治疗联合化疗的反应成为可能。需要在大量病例中进行确认。

OR_034

HMGA2 调控 DNA 损伤修复通路介导宫颈癌放疗抵抗的机制研究

李艳红 孙阳*
福建省肿瘤医院

研究目的 宫颈癌是最常见的妇科恶性肿瘤。宫颈癌的放疗抵抗性是临床治疗上亟待解决的问题。HMGA2（High Mobility Group AT-Hook 2）是一种结构性转录因子，它参与了包括宫颈癌在内的多种恶性肿瘤的生物学过程。然而，其在宫颈癌中对放射敏感性的影响及其机制仍然未知。

材料与方法 本研究通过临床样本转录组测序和生信分析，筛选出 HMGA2 作为宫颈癌放射抵抗的预测因子。采用一定放疗剂量下的 HMGA2 敲减和过表达宫颈癌细胞株的克隆形成实验初步验证。此后，通过转录组测序结合生信学分析，筛选出 DNA 损伤修复通路：同源重组(Homologous recombination，HR）及非同源末端链接（Non-homologous end joining，NHEJ）作为 HMGA2 增加宫颈癌细胞放疗抗性的可能影响通路，并应用 wb 实验及体内实验进行验证。

结果 与宫颈癌放疗敏感组织相比，宫颈癌放疗抵抗组织中 HMGA2 表达水平升高，且 HMGA2 水平升高与宫颈癌不良预后相关。此外，HMGA2 敲减后宫颈癌细胞放疗敏感性增加。进一步通过 wb 实验论证，HR 及 NHEJ 通路可能是 HMGA2 增加宫颈癌放疗抗性的作用机制。最后，通过体内实验验证了这些结果。

结论 这些发现揭示了 HMGA2 可能是克服宫颈癌放射抵抗的一个有吸引力的治疗靶点。

OR_035

替雷利珠单抗联合新辅助化疗治疗局部晚期宫颈癌的有效性及安全性研究

张文博 赵路阳 李立安 孟元光*
解放军总医院第七医学中心妇产医学部

研究目的 回顾性评估新辅助化疗联合程序性死亡受体-1（PD-1）抑制剂替雷利珠单抗免疫治疗方案在局部晚期宫颈癌（LACC）患者中的疗效及安全性。

方法 研究纳入 2022 年 10 月至 2024 年 8 月解放军总医院第一医学中心收治、接受新辅助化疗联合替雷利珠单抗免疫的 52 例 LACC 癌患者进行回顾性分析，临床分期参照 FIGO 2018 年修订的标准为 IB3、IIA2 及 IIB~IVA 期，患者接受新辅助化疗联合替雷利珠单抗免疫治疗，每 3 周给药一次，化疗方案为紫杉醇+铂类静滴，治疗后临床评估无法手术的患者接受同步放化疗，评估可手术的患者行根治性手术，术后患者接受 NCCN 指南推荐的抗肿瘤治疗。

结果 52 例患者接受新辅助化疗联合免疫治疗后部分缓解 29 例（55.8%），病情稳定 8 例（15.4%），病情进展 4 例（7.7%），客观缓解率 77%（40/52），疾病控制率 92.3%（48/52），新辅助治疗后 38 例（73.1%）患者接受手术治疗，13 例（25%）接受同步放化疗，1 例（1.9%）患者因免疫性心肌炎建议同步放化疗，其因个人因素未行，现中药治疗中，手术患者中病理完全缓解 8 例（21.1%，8/38），

28 例（73.7%）病理降期，随访截止 2025 年 4 月，无进展生存率 92.3%，总生存率 98.1%，新辅助治疗过程中 14 例治疗相关不良事件发生率为 84.6%（44/52），30 例（57.7%）患者出现 1-2 级不良反应，14 例（26.9%）患者出现 3-4 级不良反应，14 例（26.9%）出现免疫相关不良事件，所有 irAEs 经内分泌科、心内科、呼吸科等相关科室会诊及对症处理后均得到控制，不良反应经对症治疗后可耐受或缓解，未出现治疗相关的死亡，患者耐受性良好，手术患者中 11 例（28.9%）出现术后并发症，主要包括肺部感染、泌尿系感染、乳糜瘘、低蛋白血症、腹泻、肝功能异常、发热、切口愈合不良、憋气等，10 例（26.3%）患者输血。

结论 新辅助化疗联合替雷利珠单抗免疫治疗对于 LACC 患者具有较好的抗肿瘤活性和可接受的安全性。

OR_036

对巨块型宫颈癌发生宫旁浸润的影响因素及其治疗决策的探索

杨静
南京医科大学第一附属医院

目的 由于肿瘤局部控制不佳及远处转移，巨块型宫颈癌（bulky cervical cancer，BCC）的治疗面临巨大的挑战。本研究分析了影响宫旁浸润（parametrial invasion，PMI）发生的因素，以及接受不同治疗后的生存结局，并分析影响其结局的危险因素，旨在为 BCC 的治疗选择提供证据。

方法 本回顾性研究纳入了 2010 年 1 月至 2023 年 6 月于本中心就诊的 462 例 BBC 患者的临床病理资料。使用 Logistic 回归分析影响 PMI 发生的因素，使用 Kaplan-Meier 法及 Cox 比例风险模型进行生存分析。

结果 本研究共纳入 426 名 BCC 患者，Logistic 回归分析结果显示 FIGO Ⅲ~Ⅳ和原发肿瘤体积＞16 cm^3 是 BBC 发生 PMI 的独立危险因素，OR 值分别为 6.45（95% CI 2.43-18.01）和 2.11（95% CI 1.25-3.61）。经多因素 Cox 回归分析发现：存在 PMI 的患者无进展生存期（progression-free survival，PFS）更短（HR 1.72, 95% CI 1.02-2.9），但对肿瘤特异性生存期（cancer-specific survival，CSS）无显著影响。与接受同步放化疗（concurrent chemoradiotherapy，CCRT）的患者相比，接受根治性手术联合放疗（radical surgery and adjuvant radiation，RS and ART）的患者展现出更长的 PFS 和 CSS。亚组分析发现，无论是否存在淋巴脉管浸润，肿瘤直径≤4.8 cm 和肿瘤体积≤16 cm3 的患者都能从接受 RS and ART 治疗中获益，延长 CSS。而对于 PFS，除了合并 PMI 的患者，所有患者都能从 RS and ART 治疗获益。此外，接受不同治疗方案的患者治疗后 3 月和 6 月的生活质量并无差异。

结论 本研究探讨了 BCC 患者发生 PMI 的影响因素及 PMI 对预后的影响，发现 FIGO Ⅲ~Ⅳ和原发肿瘤体积＞16 cm^3的 BBC 更易发生 PMI。本研究还讨论了这些患者接受不同治疗对预后和生活质量的影响，通过亚组分析探索了能从 RS and ART 以及 CCRT 治疗得到获益的人群特征，发现 RS and ART 能够改善部分患者的预后，并且不会增加严重不良反应的发生和损害生活质量，为临床决策的制定提供证据。本研究为单中心回顾性研究，纳入的 BCC 患者多为 FIGO Ⅰ~Ⅱ期，FIGO Ⅲ~Ⅳ期较少，不能代表所有的局部晚期宫颈癌患者，因此该结论需要纳入更多因素，开展大样本、多中心前瞻性研究进一步证实。

OR_037

前哨淋巴结活检应用于早期宫颈癌的回顾性队列研究

晏和春[1] 朱仲毅[1,2] 左芮瑜[1] 陆钰坤[1,2] 何跃东[1*]
1.四川大学华西第二医院妇科
2.四川大学妇女儿童出生缺陷及相关疾病教育部重点实验室

背景及目的 对于早期宫颈癌患者，传统标准治疗术式是根治性子宫切除术+盆腔淋巴结清扫术（PLND），但是约 80%-85%的早期宫颈癌患者盆腔淋巴结并无肿瘤侵犯，行 PLND 可能增加术后发生盆腔淋巴囊肿、尿路感染、下肢麻木、深静脉血栓形成等并发症的风险。目前指南大多推荐 SLNB 应用于病灶 2cm 及以下宫颈癌患者，而对于 2-4cm 病灶患者谨慎使用。本研究旨在分析前哨淋巴结活检（SLNB）替代盆腔淋巴结清扫术（PLND）应用于早期宫颈癌患者，尤其是病灶 2-4cm 的 IB2 期患者的优势和安全性，为临床决策提供依据。

方法 本项目设计为回顾性队列研究，纳入 2019 至 2023 年在四川大学华西第二医院接受根治性手术治疗的早期宫颈癌患者。根据手术方式分为 SLNB 组与 PLND 组，主要分析指标为 2 年无病生存期（DFS）、2 年总生存期（OS）、手术并发症、前哨淋巴结总显影率、前哨淋巴结检出率等。

结果 研究共纳入 273 例宫颈癌病例，其中 SLNB 组 122 例，PLND 组 151 例；鳞癌 214 例，腺癌 47 例，腺鳞癌 12 例；FIGO 分期以 IB1 和 IB2 期为主。PLNB 组共获得淋巴结 3990 枚，PLN 阴性病例共 132 例，PLN 转移病例 19 例，术后复发 6 例，复发病例均为有淋巴结转移者；SLNB 组共获得淋巴结 854 枚，其中在 115 例（94%）患者中至少发现 1 个 SLN，有 96 例（78%）发现双侧 SLN，其中 SLN 阴性病例共 113 例，SLN 阳性病例共 9 例，术后复发 4 例，其中有 1 例为 SLNB 阳性患者。PLNB 组（4.0%）术后复发率略高于 SLNB 组（3.6%），但差异无统计学意义（P=0.712）；PLNB 组（19.9%）的术后并发症发生率高于 SLNB 组（10.2%），且具有统计学意义（P=0.027）。入组患者中，33 例为 IB2 期，包含 SLNB 组 16 例，PLND 组 17 例，病例均无复发；两组术后并发症发生率无统计学差异（PLNB 组 17.6%，SLNB 组 18.8%，P＞0.999）；SLNB 组共获得盆腔淋巴结 224 枚，其中 SLN 共 91 枚，所有患者均至少发现 1 个 SLN，88.2%患者（15 例）发现双侧 SLN，所有盆腔淋巴结病理结果均为阴性。

结论 SLNB 不仅在大多数指南推荐的 IB1 期及之前的早期宫颈癌中安全可行，也可作为 IB2 期宫颈癌患者替代 PLND 的优选策略。

OR_038

ICCOR: Integrative Cervical Cancer Omics Resource

Shitong Lin Peng Wu*
Huazhong University of Science and Technology Tongji Medical College Affiliated Union Hospital

Understanding cervical cancer (CC) heterogeneity, progression, and therapeutic response necessitates an integrative multi-omics approach. To address this critical need and empower researchers, we developed the Integrative Cervical Cancer Omics Resource (ICCOR), a comprehensive and freely accessible platform (website link is unavailable now). ICCOR uniquely harmonizes five key data dimensions: (1) Bulk transcriptomics integrating 1,637 cervical samples (cell lines, normal, precancerous, carcinoma, adjacent) across 17 datasets with expression and clinical/prognostic data; (2) Single-cell transcriptomics integrating 102 cervical samples across 11 datasets, featuring re-annotation to explore gene expression within specific cell subtypes; (3) Spatial transcriptomics profiling 26 in-house cervical samples (normal/cancer) across 2 datasets, enabling spatial mapping and expression analysis of target genes; (4) HPV integration profiles from 222 in-house cervical samples (precancerous, carcinoma, adjacent) across 3 datasets, allowing investigation of HPV integration frequency and genomic locations; and (5) A curated pan-cancer immunotherapy

cohort comprising 3,650 patients across 27 datasets (>17 cancer types) treated with anti-PD-(L)1 or CTLA-4, facilitating differential gene expression analysis by treatment response. This resource provides powerful tools to explore critical aspects of CC biology, including cell-type specific expression, spatial tumor microenvironment organization, HPV oncogenesis mechanisms (integration hotspots), and immunotherapy-linked biomarkers. By integrating and re-annotating vast, diverse datasets spanning bulk, single-cell, spatial, viral, and clinical dimensions, ICCOR serves as an indispensable platform for advancing CC research, biomarker discovery, and therapeutic development.

OR_039

Establishment of Patient-Derived Organoids for Gastric-Type Endocervical Adenocarcinoma

Ruiyi Xu Hui Wang*
Department of Gynecologic Oncology, Women's Hospital, Zhejiang University School of Medicine, Hangzhou 310000, China

Background Gastric-type endocervical adenocarcinoma (GAC) is a rare and aggressive, HPV-independent subtype of cervical adenocarcinoma with limited therapeutic options and poor prognosis. Reliable preclinical models are urgently needed to study its pathogenesis and test novel therapeutic strategies.
Methods Fresh GAC tumor tissues were obtained from surgical specimens and mechanically and enzymatically dissociated. Isolated cells were embedded in Matrigel and cultured in the organoid medium optimized for endocervical epithelium. In parallel, patient-derived xenograft (PDX) models were established by subcutaneous implantation of GAC tissues into NCG mice. Tumor cells isolated from PDX tumors were similarly digested and cultured to generate PDX-derived organoids. The established organoids were characterized by histopathology and immunofluorescence staining.
Results We successfully established patient-derived and PDX-derived GAC organoids that maintained the histological and molecular features of the original tumors. Organoids demonstrated long-term proliferative capacity and recapitulated the growth behavior and morphology of primary tumors and corresponding PDX models Conclusion:
Conclusion We developed the first organoid models for GAC derived from both patient tissues and PDX tumors, which faithfully reflect the histological and molecular characteristics of this subtype. These models provide a robust platform for mechanistic studies and preclinical drug testing.

OR_040

Precision oncology guided by causal inference: Integrative multi-omics Mendelian randomization reveals immune-epigenetic crosstalk driving cervical carcinogenesis

Lihua Chen Pengming Sun*
Fujian Maternity and Child Health Hospital, College of Clinical Medicine for Obstetrics & Gynecology

Background Despite the adoption of immunotherapy as first-line treatment for recurrent/metastatic cervical cancer (R/M CC), its clinical efficacy remains suboptimal. Emerging evidence suggests that metabolite-mediated post-translational modifications (PTMs) within the immunosuppressive tumor microenvironment may contribute to therapeutic resistance. This study aims to investigate the relationship between immune-related gene and proteins with PTMs with cervical cancer.
Methods We employed a comprehensive approach that included data acquisition from The Cancer Genome Atlas (TCGA), Genotype-Tissue Expression (GTEx), and Gene Expression Omnibus (GEO)

databases. A total of 1576 immune-related genes were identified, and multi-omics Mendelian randomization (MR) analyses were conducted to assess causal relationships between these genes and cervical cancer. Additionally, sensitivity analyses, including multivariable MR and mediation effect assessments, were performed to validate the findings.
Results We evaluated causal relationships between 1,576 immune- and histone modification-related genes (encompassing phosphorylation/acetylation regulators) and cervical carcinogenesis. Our analysis identified four genes (CASP8, MAPK3, CBS, LTA) and three protein targets (EIF2AK2, SPAG1, LGALS3) demonstrating genome-wide significant causal associations with CC risk (PFDR < 0.05). While LTA exhibited positive causality, other candidates showed protective effects. Bidirectional MR and summary-based MR (SMR) analyses validated the robust causal roles of LTA, EIF2AK2, and SPAG1 in CC pathogenesis. Tumor immune microenvironment profiling revealed significant correlations between these candidate regulators and 19 functionally distinct immune cell populations. Molecular docking simulations further predicted moderate binding affinities between LTA and acetaminophenand between SPAG1 and valproic acid, suggesting druggable potential.
Conclusions This multilevel integrative analysis establishes LTA, EIF2AK2, and SPAG1 as key PTM-associated mediators of CC progression, providing mechanistic insights into immunotherapy resistance and nominating novel biomarkers for early detection and therapeutic targeting.

OR_041

构建代谢评分系统预测子宫内膜癌临床结局并指导免疫治疗

毕烁 杨潇*
北京大学人民医院

研究目的 代谢综合征与子宫内膜癌进展密切相关，机体代谢紊乱包括糖、脂和氨基酸代谢紊乱与在肿瘤进展中发挥重要性作用。然而，目前糖、脂和氨基酸代谢异常在子宫内膜癌预后和免疫治疗中的作用尚不清楚，且既有研究大多仅从单一种类代谢通路进行机制研究。本研究旨在综合糖、脂、氨基酸代谢三种代谢通路，构建代谢评分系统预测子宫内膜癌临床结局并指导免疫治疗。
材料与方法 TCGA 数据库下载子宫内膜癌表达谱数据，检测 35 个糖脂氨基酸代谢关键基因在子宫内膜癌中的表达水平和突变情况。应用非负矩阵分解聚类法鉴定子宫内膜癌中的糖脂氨基酸（Metabolism）亚型。使用 CIBERSORT 和 ESTIMATE 算法来描述肿瘤微环境特征。此外，通过分析不同亚型的差异基因，进一步建立 Metabolism Score 评分系统用于评估患者预后。并验证 Metabolism Score 与免疫治疗、肿瘤突变负荷（TMB）以及肿瘤干性的关系。
结果 共鉴定到 2 类 Metabolism 亚型，基于亚型将患者分为两个基因亚型，B 类亚型患者的生存预后明显差于 A 类亚型患者。较高的 Metabolism Score 评分与较低的 TBM、较低基质评分和免疫评分有关，而对表现出对免疫治疗的较好反应性。通过单因素和多因素分析发现，Metabolism Score 评分是影响子宫内膜癌患者预后的独立危险因素，其预测精准性较好（ROC=0.780），显著高于其他临床特征。基于 Metabolism Score 和多个临床特征建立列线图用于评估子宫内膜癌患者 3 年和 5 年生存率，DCA 曲线分析提示列线图具有较好的预测准确性。运用单细胞组学和临床样本验证发现，高风险评分人群 T 细胞浸润减少，其可能免疫逃逸相关。
结论 本研究全面分析了糖、脂和氨基酸关键基因在子宫内膜癌中的作用，并构建了基于糖、脂和氨基酸关键基因的 Metabolism Score 系统用于指导子宫内膜癌患者免疫治疗，最后构建列线图用于协助临床医生精准预测子宫内膜癌患者生存预后。

OR_042

Analysis of the Clinicopathological and Molecular Characteristics Associated with Risk of Lymph Node Metastasis in Endometrial Malignant Tumors

Jia Zeng You Wu Bin Li*
National Cancer Center/Cancer Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College

Objective This study retrospectively included endometrial cancer cases treated at our center over the past 10 years. By integrating molecular classification with the updated FIGO 2023 staging system, all cases were restaged to systematically evaluate the clinicopathological characteristics of different histological subtypes with myometrial invasion, and to identify the risk factors for lymph node metastasis.

Methods Clinical data were collected from patients with malignant endometrial tumors who underwent surgical treatment in our center, between January 2015 and December 2023. The collected data included preoperative tumor marker levels, surgical approach, postoperative pathological findings, and molecular pathological results.

Results

(1) Clinical characteristics: This study included 415 patients with endometrial cancer who underwent surgery, with a median age of 55 years (range: 30–75 years). There were 201 cases of non-p53 mutant endometrial adenocarcinoma, 107 cases of p53 mutant endometrial adenocarcinoma, and 107 cases of special histological subtypes including 13 cases of clear cell carcinoma, 36 cases of high-grade serous carcinoma, 39 cases of mixed carcinoma, 4 cases of carcinosarcoma, 1 case of sarcoma, 5 cases of undifferentiated carcinoma, 4 cases of dedifferentiated carcinoma, 4 cases of mesonephric-like adenocarcinoma, and 1 case of mucinous adenocarcinoma. The clinical staging (FIGO 2023) included 271 of stage I, 68 of stage II, 55 of stage III, 21 of stage IV. All patients were treated with total hysterectomy with bilateral salpingo-oophorectomy + pelvic lymph node dissection ± para-aortic lymph node dissection ± omentectomy. Among these, 189 patients underwent laparotomy, 219 underwent laparoscopy (or robotic), and 6 were converted from laparoscopy to open surgery. The median follow-up time was 56.7 months (range: 19.8–86.7 months).

(2) Pathological Results 50 cases (13.33%) were well-differentiated, 193 (51.47%) moderately differentiated, and 132 (35.20%) poorly differentiated. Lymphovascular space invasion (LVSI) was absent in 303 cases (73.72%), <5 in 95 cases (23.11%), and >5 in 13 cases (3.16%). Myometrial invasion <1/2 in 321 patients (78.1%) and ≥1/2 in 90 patients (21.9%). Endocervical involvement was absent in 331 cases (85.75%), involved the endocervix in 6 cases (1.55%), and cervical stroma invasion in 49 cases (12.69%). Uterine cornua involvement was negative in 339 (93.39%) patients, unilateral in 6 (1.65%), and bilateral in 18 (4.96%). fallopian tube or ovarian involvement was absent in 344 cases (82.89%) and present in 71 cases (17.11%). Lymph node metastasis occurred in 52 patients (12.59%), and isolated para-aortic lymph node metastasis was observed in 8 cases (1.93%). Regarding molecular features, POLE mutations were found in 17 cases (6%), p53 mutations in 133 cases (37%), and MMR deficiency (dMMR) in 71 cases (18.5%).

(3) Subgroup analysis: The patients with deep myometrial invasion (>1/2), the rates of lymph node metastasis varied significantly across histological and molecular subtypes. The lymph node metastasis rate in the non-p53 mutant endometrial adenocarcinoma group was 6/33 (18.2%), the p53 mutant group was 6/18(33.3%), the special histological subtypes group was 24/39 (61.5%), p=0.001. The isolated para-aortic lymph node metastasis rate in the non-p53 mutant or p53 mutant groups was negative, while in the group of special histological subtypes was 6/39(15.4%), p=0.015.

(4) Univariate analysis: Myometrial invasion >1/2 (HR 14.524, 95% CI: 7.345–28.719, p<0.001), positive lymphovascular space invasion (LVSI)(HR 9.095, 95% CI: 4.778–17.312, p<0.001), adnexal involvement (HR 68.037, 95% CI: 29.101–159.067, p<0.001), elevated preoperative CA125 levels (HR 9.357, 95% CI: 4.918–17.801, p<0.001), p53 gene mutation (HR 2.206, 95% CI: 1.128–4.314, p=0.021) was strongly associated with lymph node metastasis.

(5) Multivariate analysis: Deep myometrial invasion (HR 5.376; 95% CI: 1.796–16.096; p = 0.003), positive lymphovascular space invasion (LVSI) (HR 3.882; 95% CI: 1.280–11.768; p = 0.017), Adnexal involvement (HR 24.351; 95% CI: 7.932–74.755; p < 0.001), elevated preoperative CA125 levels(HR 5.585; 95% CI: 1.838–16.968; p = 0.002) was independently associated with a a higher lymph node metastasis rate, wihle p53 gene mutation was not (HR 1.803; 95% CI: 0.600–5.419; p = 0.294).

Conclusion Patients with deep myometrial invasion (>1/2) and special histological subtypes of malignant endometrial tumors exhibited a higher rate of lymph node metastasis compared to endometrioid adenocarcinoma. Deep myometrial invasion (>50%), positive lymphovascular space invasion (LVSI), adnexal involvement, and elevated preoperative CA125 levels as independent risk factors for lymph node metastasis in malignant endometrial tumors.

OR_043

西达本胺通过诱导核糖体翻译受损治疗子宫内膜癌的机制研究

张皓程 庞迎新*
山东大学齐鲁医院

研究目的 子宫内膜癌已成为高收入国家最常见的妇科肿瘤，患人群数仍在上升，EC 采用手术、放疗、化疗和免疫治疗相结合的方式进行治疗。有高危组织病理学因素和晚期的患者预后较差，辅助放疗或全身治疗是常见的治疗策略。因此，亟需优化子宫内膜癌的治疗方案。TCGA 数据库表明，子宫内膜癌中 HDAC 家族中 1，2，10 高表达，而组蛋白乙酰化酶抑制剂（HDACi）西达本胺是中国自主研发的一种口服争对 HDAC1，2，3，10 的药物。本研究的目的是探讨西达本胺单药治疗或与放疗联合治疗子宫内膜癌的抗肿瘤作用并研究其潜在机制。目的是完善和定制未来子宫内膜癌患者的辅助治疗选择，从而实现针对该病症更全面、更个性化的治疗方法。

材料和方法 我们通过对子宫内膜癌细胞系的药物治疗评估了西达本胺对细胞增殖和其他功能的疗效。此外，流式细胞术检测细胞凋亡和细胞周期，WB 检测相应分子和相关通路蛋白的表达。同时，我们进行了 RNA 测序分析，以检测相关基因表达的改变。我们使用了表面翻译传感实验，它可以监测和量化蛋白质合成效率，核仁银染实验检测嗜银核仁组织区（AgNOR）数量。西达本胺的疗效在子宫内膜癌类器官中验证，同时其联合放疗在 CDX 和 PDX 模型中得到验证。

结果 采用 TCGA 数据库分析 HDAC 家族在子宫内膜癌组织中的表达情况，发现 HDAC1、2、10 与正常组相比高表达，并使用 wb 验证了这三种蛋白亦为高表达。流式显示西达本胺显著诱导内膜癌细胞凋亡并引起 G2/M 期周期阻滞。通过子宫内膜癌的浓度梯度处理和测序，发现西达本胺影响核糖体相关功能，激活 MAPK 通路，并构建内膜癌类器官验证其机制，在 CDX 和 PDX 动物模型联合放疗，发现该药物与放疗具有协同抗肿瘤效应。

结论 本研究验证了西达本胺作为单一疗法以及与放疗联合治疗子宫内膜癌的有效性。此外，我们发现了 HDAC 抑制剂新机制，即通过诱导核糖体翻译受损发挥抗肿瘤作用。

OR_044

Intratumor microbiome-derived butyrate facilitates progesterone resistance by inducing ferroptosis in endometrial cancer

Xingchen Li Wang Jianliu*
Peking University People's Hospital

Background Endometrial cancer (EC) is increasingly affecting young women desiring fertility preservation, yet progesterone resistance remains a major challenge. The role of the intratumor microbiome and its metabolites in progesterone sensitivity is poorly understood.
Methods We performed 16S rRNA sequencing on tumor samples from 72 early-stage EC patients, stratified into progesterone-sensitive and resistant groups. Microbial diversity and composition were analyzed using alpha and beta diversity metrics. Molecular mechanisms of key metabolites in response to progesterone was also explored. Finally, key targets to enhance the sensitivity of EC patients with progesterone resistance were also identified.
Results Progesterone-sensitive patients exhibited significantly higher microbial diversity and enrichment of butyrate-producing bacteria, including Butyrivibrio, Faecalibacterium and Clostridium. Butyrate supplementation inhibited EC cell proliferation and synergized with progesterone to induce tumor regression. Mechanistically, butyrate downregulated CISD1, a key regulator of iron, leading to increased ferroptosis through iron-dependent oxidative damage. In vivo, butyrate combined with progesterone significantly reduced tumor volume and weight compared to monotherapy. Transcriptomic analysis revealed that butyrate-mediated CISD1 inhibition upregulated ferroptosis-related pathways, including lipid peroxidation and reactive oxygen species (ROS) accumulation. Additionally, butyrate enhanced the expression of progesterone receptors (PGR), further sensitizing EC cells to progesterone therapy. These findings were supported by immunohistochemical and flow cytometry analyses, confirming the role of butyrate in promoting ferroptosis and overcoming progesterone resistance.
Conclusion This study highlights the critical role of the intratumor microbiome and butyrate in enhancing progesterone sensitivity through ferroptosis, offering a novel therapeutic strategy for EC patients.

OR_045

子宫内膜癌孕激素耐药的时序分子特征及逆转策略研究

彭迎滢 张俊 汪宏波*
Wuhan Union Hospital

研究目的 子宫内膜癌是当前发病率最高的妇科肿瘤之一，其中约 6.4%的患者发病年龄小于 45 岁。为了保留这部分患者的生育能力，孕激素（如醋酸甲羟孕酮，MPA）被视为保守治疗的一线药物。约 70%的患者对孕激素初次治疗有响应，但其中 30-40%的患者会复发，且 63%的患者在再次接受孕激素治疗时无效。因此，急需对孕激素耐药的机制进行探索，并且开发新的联合药物治疗方案以改善患者预后。
材料与方法 对于人子宫内膜癌 Ishikawa 细胞系，在培养基中添加 2.5μmol/L 的 MPA，以 1/50 传代，维持培养 1 个月后，增加浓度为 5μmol/L，维持培养 1 个月，接下来依次每个月增加浓度直至 20μmol/L，维持培养 6 个月后，得到能在含 20μmol/L 的 MPA 的完全培养基中生长速度同在正常完全培养基中的 Ishikawa 细胞生长速度一致的耐药细胞，命名为 Ishikawa-MPA_R。在此期间，对于 Ishikawa，Ishikawa_MPA_5μM，Ishikawa_MPA_10μM，Ishikawa_MPA_15μM，Ishikawa_MPA_20μM（在每种浓度下细胞生长趋于稳定的时间点取样）进行时序性的转录组、蛋白质组、代谢组学测序，通过生物信息学方法分析在 Ishikawa 细胞从对 MPA 敏感到耐药的过程中起关键作用的分子及可能机制，并且进行裸鼠皮下瘤实验探索联合给药方案。

结果 EFHD2、ALOX15、CBR3、TOX2 的表达量随着 Ishikawa 耐药性的提高而不断上调，CALB1 的表达量则随着耐药不断下调。进行 si-RNA 敲降以及 CCK8 实验，证实这 5 个分子可以部分挽救或提高 Ishikawa 对于 MPA 的耐药性。此外，根据构建的基因调控网络筛选出关键分子，验证 EEF1A2（真核生物延长因子 1A）的上调对于耐药性的维持至关重要，且在裸鼠皮下瘤实验中，EEF1A2 的抑制剂 Plitidepsin 与 MPA 联用可以抑制 MPA 耐药肿瘤生长。
结论 EEF1A2 是子宫内膜癌孕激素耐药的关键分子，其抑制剂 Plitidepsin 与 MPA 联用有望改善子宫内膜癌孕激素治疗耐药患者预后。

OR_046

肠道微生态与子宫内膜癌发生机制的相关性研究

吴珍珍
甘肃省妇幼保健院

目的 探索肠道菌群失衡介导血清代谢紊乱导致子宫内膜癌发生的相关性。
方法 选取 2023 年 2 月-2023 年 8 月甘肃省妇幼保健院妇科，病理类型为子宫内膜样腺癌患者作为研究组（EC 组），同期在本院健康管理中心体检的健康女性作为对照组（N 组)。采集粪便样本，利用 Illumina Miseq 测序平台采用 16S rRNA 基因测序方法对粪便微生物测序；采集血清样本，采用 LC-MS 技术进行靶向代谢组学分析，寻找差异代谢通路。
结果
（1）肠道菌群在门水平的组成存在差异.假单胞菌门(Pseudomonadota)的表达，EC 组 16.07%，N 组 4.24%，差异有统计学意义。厚壁菌门(Bacillota)/拟杆菌门(Bacteroidota)比值（B/B），EC 组为 1.35，N 组为 1.20。
（2）肠道菌群在属水平的组成存在差异。两组间共有 59 种细菌属的丰度，差异有统计学意义。EC 的优势菌属，Klebsiella（H=6.092，P＜0.001）、Megamonas（H=7.480，P＜0.001）。
（3）血清代谢物存在差异。Hypoxanthine、2,4-Dihydroxyacetophenone 5-sulfate、Inosine、Benzoylcholine、20-Hydroxyarachidonic acid、Theobromine 等代谢物在 EC 组中表达最为显著；KEGG 富集分析发现 Glycerophospholipid metabolism（甘油磷酸脂代谢）及 Purine metabolism（嘌呤代谢）途径在 EC 组中较为显著.
（4）建立 EC 预测模型。Megamonas（巨单胞菌属）、Klebsiella（克雷伯氏菌）、血清肿瘤标志物 CA125 及 HE4 作为独立指标绘制 ROC 曲线。EC 组中，CA125、HE4、Megamonas、Klebsiella 的 AUC 值分别为 0.764、0.675、0.833 和 0.840；联合预测 EC 的 AUC 值为 0.897。
结论
（1）子宫内膜癌患者肠道屏障功能受损、不利于肠道健康。
（2）甘油磷酸脂代谢、嘌呤代谢途径在 EC 组中较为显著，导致不饱和脂肪酸增多。
（3）两种 EC 的特异性肠道细菌 Megamonas、Klebsiella 与血清 CA125、HE4 四种指标联合预测 EC 方面具有一定价值。未来有望成为高危人群的无创筛查方案。

OR_047

Integration of Inflammatory and Nutritional Biomarkers with Machine Learning Enhances Prediction of Progesterone Response in Fertility-Preserving Endometrial Carcinoma Management

Yue Qi Xingchen Li Jianliu Wang*
Peking University People's Hospital

Background Endometrial carcinoma (EC) and atypical endometrial hyperplasia (AEH) increasingly affect young women, posing challenges for fertility preservation. The inflammatory and nutritional status have been shown to significantly influence disease outcomes, especially in cancer. However, no studies have systematically investigated the predictive value of inflammation and nutrition scores for complete response (CR) in endometrial carcinoma.
Methods This retrospective study included 329 EC/AEH patients treated at Peking University People's Hospital from January 2012 to December 2024. We developed a multimodal nomogram integrating 12 inflammatory (NLR, SIRI, PLR, et al) and 5 nutritional biomarkers (mGNRI, PNI, NRI, ALI, CONUT) via LASSO regression and machine learning. Model validation employed leave-one-out cross-validation (LOOCV), with performance assessed by AUC, calibration curves, and decision curve analysis (DCA).
Results The combined inflammatory-nutritional score achieved superior predictive accuracy, with AUC values of 0.846 (training cohort) and 0.871 (validation cohort). Besides, our nomogram which was constructed by four clinical variables (BMI, menstrual history, metabolic syndrome, and histological type), inflammatory score and nutritional score exhibited excellent predictive potential, with AUC values of 0.915 (training cohort) and 0.933 (validation cohort), significantly outperforming clinical models. Risk stratification revealed significantly lower CR rates in high-risk patients (log-rank P<0.001), with decision curve analysis demonstrating a 35% reduction in unnecessary interventions.
Conclusions Integrating systemic inflammation and nutritional biomarkers enhances CR prediction in EC/AEH, enabling personalized risk stratification to guide fertility-sparing strategies. This tool addresses a critical clinical gap, though future multicenter studies are warranted to validate generalizability and explore mechanistic pathways.

OR_048

NSMP 亚型子宫内膜癌的临床病理特征及预后——一项单中心回顾性研究

辛玲霞 任康 马佳彬 王文惠 颜子涵 盛云龙 王雅琦 张福泉 侯晓荣*
北京协和医院

研究背景 子宫内膜癌是常见的女性生殖道恶性肿瘤，居妇科肿瘤的第二位，发病率呈逐年上升趋势。近年来，TransPORTEC 分型方法在子宫内膜癌中应用越来越广泛，其包括 POLE 超突变型、MMR-d 型、p53 突变型、无特异性分子改变（NSMP 型）四种亚型。其中，NSMP 亚型占比超过 50%，患者预后存在明显异质性。尽管既往研究证实，ER、PR、L1CAM 的表达及 KRAS、CTNNB1 基因突变是 NSMP 人群的预后因素。但目前指南尚未就 NSMP 亚型人群进行分层指导治疗。目的：回顾性分析单中心子宫内膜癌 NSMP 亚型患者的临床病理特征及预后，寻找该亚型人群中的异质性群体，旨在为 NSMP 亚型分层治疗提供依据。

方法 回顾性纳入 2021 年 6 月至 2023 年 10 月就诊北京协和医院的子宫内膜癌患者。纳入标准：分子分型为 NSMP 型，免疫组化指标（包括 ER、PR、Ki67、MLH-1、MSH、MSH-6，PMS-2、P53）完整。排除标准：病理结果及分子分型信息不完整、治疗信息缺失或随访信息缺失。
结果 共纳入 128 例子宫内膜癌患者，中位年龄 55 岁（27-80 岁）。组织学类型分别为：子宫内膜样癌（n=122）、透明细胞癌（n=1）、未分化癌（n=2）、混合癌（n=1）、中肾样腺癌（n=2）。其中，22 例未行手术，主要原因为分期为 IV 期行保守治疗。根据免疫组化结果 3 例 ER 阴性、5 例 PR 阴性、1 例微卫星不稳定，10 例 P53 阳性。中位 Ki-67 水平为 45%。接受手术（n=106）的患者，根据 FIGO 2023 分期，IA1 期（n=7）、IA2 期（n=67）、IB 期（n=21）、IIA 期（n=1）、IIB 期（n=1）、IIC 期（n=9）；根据 2021 ESMO-ESGO-ESTRO 风险分层系统，低危（n=76）、中危（n=24）、中高危（n=5）、高危组（n=1）。44 例接受 EBRT±VBT。7 例接受辅助化疗。有 5 例出现局部区域复发，1 例为阴道残端复发，4 例局部区域失败。出现复发的患者，3 例为中危，2 例为低危。
结论 NSMP 亚型中，约 4.5%的患者为侵袭性组织学类型。约 2%的患者 ER 受体阴性，3.8%为 PR 受体阴性，7.5%的患者 P53 IHC 为突变型。接受手术治疗的 NSMP 亚型患者，中高危/高危患者的比例 <5%。NSMP 亚型子宫内膜癌人群中尚存在需要积极治疗的亚组，亟待探索新的分子分型标志物指导调整 NSMP 亚型患者的治疗策略。

OR_049

基于多中心队列的早发性子宫内膜癌基因组及分子特征研究

胡哲[1] 兀紫梦[1] 陈晓军[2] 孙朝阳[1] 陈刚[1*]
1.华中科技大学同济医学院附属同济医院 2.同济大学附属第十人民医院

研究目的 近年来，子宫内膜癌发病呈年轻化趋势，部分患者具备保留生育功能的需求，早发性子宫内膜癌（EEEC）成为临床关注热点。现有分子分型研究主要聚焦于欧美人群的老年子宫内膜癌患者，针对中国人群年轻患者内膜样子宫内膜癌的多组学特征尚不清晰。本研究旨在系统解析 EEEC 的基因组、蛋白质组特征，探索其致病机制、内分泌治疗敏感性标志物以及精准分型策略。
材料与方法 本研究纳入来自湖北武汉华中科技大学同济医学院附属同济医院与上海复旦大学附属妇产科医院 215 例子宫内膜样癌患者（其中 81 例为 40 岁以下 EEEC 患者）及 14 例非典型子宫内膜增生（AEH）患者，收集福尔马林固定石蜡包埋（FFPE）组织样本，开展全外显子测序与高灵敏蛋白质组学检测。通过多组学整合、突变签名分析、蛋白表达谱挖掘及新开发的 3D-Sig-Explorer 算法进行深入解析，并结合部分患者保育治疗结局，探索内分泌耐药机制与预测因子。
结果 研究发现 EEEC 具有独特的突变谱和蛋白质组特征：年轻患者的突变负荷显著低于年老患者，但是具有独特富集的 CTNNB1 与 SIGLEC10 高频突变，后者中 Q144K 位点与高 BMI 及孕激素治疗耐药密切相关。3D-Sig-Explorer 揭示 EEEC 富集一种外暴露相关突变特征（Sig6），与环境暴露因素（如污染、氧化应激）相关，并促发特定热点突变。蛋白组分析表明 EEEC 中脂肪酸代谢及雌激素应答通路显著激活，孕激素耐药患者中 SIGLEC10 表达显著升高，且其 Q144K 突变蛋白可与 ERα 互作，激活雌激素信号通路。此外，鉴定出 EEF1E1、ILVBL、SRPK1 等多个孕激素耐药相关蛋白标志物。
结论 本研究构建了迄今最大规模的中国人群 EEEC 多组学资源库，揭示了 EEEC 独特的外暴露相关致癌机制与致病突变谱，提出 SIGLEC10 Q144K 突变可能为孕激素耐药的新机制，并建立了潜在的预测标志物体系。上述发现有望指导高危年轻女性的筛查、分型与精准保育策略的制定。

OR_050

子宫内膜癌诊断中宫腔镜应用与腹水细胞学阳性的相关性及风险因素分析

张婧莹[1,2] 陈佳欣[1,2] 闫晔[1,2] 田文艳[1,2] 张慧英[1,2] 王颖梅[1,2*]
1.天津医科大学总医院妇产科 2.天津市女性生殖健康与优生重点实验室

目的 评估宫腔镜检查（HSC）与诊断性刮宫（D&C）两种诊断方式与 EC 患者的腹水细胞学状态及预后之间的相关性。
方法 回顾性分析了 837 例术前接受 HSC 或 D&C 的 EC 患者，根据诊断方法将患者分为 HSC 组和 D&C 组，并分析两组腹水细胞学状态和预后的差异。主要终点为腹水细胞学阳性（PPC）率，次要终点为复发率、无进展生存期（PFS）和总生存期（OS）。
结果 HSC 组中年龄≥60 岁的 EC 患者占比（HSC 51.7% vs. D&C 42.3%，P = 0.007）高于 D&C 组。D&C 组中Ⅲ/Ⅳ期患者占比（HSC 9.7% vs. D&C 16.8%，P = 0.003）高于 HSC 组。两组间复发率（HSC 6.8% vs. D&C 10.2%，P = 0.094）和 PFS（P = 0.421）无显著差异。根据国际妇产科联盟（FIGO）分期、组织学类型和侵袭性类型进行分层后，各亚组间 PPC 率和 PFS 均未观察到统计学差异。分层分析显示两组 OS 在Ⅲ/Ⅳ期疾病（P = 0.045）、非子宫内膜样癌（P = 0.004）和侵袭性癌（P = 0.009）中存在统计学差异，HSC 组 5 年 OS 率更高。多因素分析显示，术前血清 CA12-5 ≥35 U/mL（P = 0.006）是 PPC 的独立危险因素。
结论 HSC 可能是 EC 相对安全的诊断方式，但对于术前血清 CA12-5 ≥35 U/mL 且可疑 EC 的患者尽量避免使用宫腔镜。

OR_051

洛塞那肽联合左炔诺孕酮宫内缓释系统治疗子宫内膜非典型增生的前瞻性随机对照小样本临床研究

嘎玛曲加[1] 季立津[2] 王漫榕[1] 吴鹏飞[1] 王露露[1] 罗雪珍[1] 鹿斌[3] 陈晓军[4] 单伟伟[4*]
1.复旦大学附属妇产科医院 2.复旦大学附属华山医院
3.复旦大学附属华东医院 4.上海市第十人民医院

目的 探究在保留生育功能治疗中洛塞那肽（Loxenatide, LOX）联合左炔诺孕酮宫内缓释系统（levonorgestrel - releasing intrauterine system, LNG-IUS）对合并肥胖的子宫内膜非典型增生患者的疗效。
方法 本研究为前瞻性随机对照小样本临床研究，2022 年 6 月至 2023 年 12 月期间，共 30 名符合纳入与排除标准的子宫内膜不典型增生（endometrial atypical hyperplasia, EAH）合并肥胖患者被随机分为 LNG-IUS 组（n=15）和 LNG-IUS 联合洛塞那肽组（n=15），主要研究指标是 28 周的完全缓解率。假设 EAH 患者（ BMI≥28 kg/m2）单用 LNG-IUS 28 周 CR 率 p0=40%，联合洛塞那肽后 p1=85%，检验有效率 α 为 0.05，检验效能 1-β 为 0.8，计算出所需总样本量为 30。
结果 对这 30 名患者进行了治疗分析，随访截止至 2025 年 6 月。该研究人群的中位确诊年龄为 29 岁(IQR,26-34 岁)。初诊中位 BMI 为 31.3 (IQR，29.78-35.12)，中位随访时间为 21.02 个月(IQR，18.13-24.26 个月)。随访期间 96.7%（29/30）的患者达到完全缓解(Complete response, CR)，剩余 1 例在治疗 11 个月后病理仍为 EAH 后选择切除子宫。达到完全缓解的中位治疗时长为 14.86 个周(IQR，13-29.57 个周)。实验组和对照组 16 周完全缓解率分别为 53.3%(8/15)和 46.7%(7/15)，p=1.0；实验组和对照组 28 周完全缓解率分别为 80%(12/15)和 60%(9/15)，部分缓解率(Partial response, PR)均为 13.3%(2/15)，而无反应(No response, NR)率为 6.7%(1/15)和 26.7%(4/15)，p=0.394；实验组和对照

组 32 周完全缓解率分别为 93.3%(14/15)和 60%(9/15)，p=0.08。对照组中有 4 例（26.7%）为难治性病例，而实验组仅有 1 例(6.7%)为难治性病例。对初始治疗（T1）、药物治疗 3 个月（T2）和治疗 6 个月（T3）时间点进行分析，两组在体重、体脂分布、胰岛素抵抗、血糖指标均无统计学差异。
结论 在 EAH 合并肥胖的患者中两种治疗方案的疗效结局未发现显著差异。然而洛塞那肽联合 LNG-IUS 相较于单纯使用 LNG-IUS，16 周、28 周、32 周完全缓解率更高，难治性比例更低，虽无显著性差异，后续仍旧需要更大样本的临床实验。

OR_052

A open-label, multicentre, prospective clinical study evaluating niraparib monotherapy for maintenance of uterine serous carcinoma：Primary outcome update

Qing Zhang[1] Beihua Kong[1*] Changyu Wang[2] Wenxin Liu[3] Xiaojun Chen[4] Hui Wang[5] Wenjun Cheng[6]
1.Qilu Hospital of Shandong University
2.Tongji Hospital of Tongji Medical College, Huazhong University of Science and Technology
3.Tianjin Medical University Cancer Institute & Hospital
4.Obstetrics & Gynecology Hospital of Fudan University
5. Women's Hospital School of Medicine Zhejiang University
6.Jiangsu Province Hospital

Objectives To evaluate the efficacy and safety of niraparib (a PARP inhibitor) maintenance therapy among patients with uterine serous carcinoma (USC) who achieved CR or PR to the last line of platinum-based chemotherapy.
Methods In this open-label, multicentre, prospective clinical study in China, the maintenance therapy cohort patients with USC (stage I-IV) with CR or PR to 1 line or first-time recurrent platinum-based therapy were received niraparib once-daily. A starting dose of 200 mg once daily (QD) was used, except in patients with a baseline body weight of ≥ 77 kg and platelet count ≥ 150,000/μL who received 300 mg QD.
The primary endpoint was PFS rate at 12 months (PFS12) assessed by Investigator and analysed using Kaplan-Meir method with corresponding 95% confidence intervals (CI). (NCT04716686).
Results From August 24, 2021, to January 6, 2025, a total of 31 patients were enrolled at six clinical centres. median follow-up for PFS was 24.1 months (95 CI: 10.8 - 37.4). Date of cutoff is July 5.2025. At baseline, 74.2% (22/31) were FIGO stage III or IV. 96.8% (29/31) patients undergone cytoreductive surgery. 35.5% (11/31) had undergone radiotherapy.
Total patients who evaluated(n=31), the PFS rate at 12 months in niraparib maintenance therapy was 81.8% (95% CI, 61.6%-92.0%). The mPFS in niraparib maintenance therapy was 32.4 months (95% CI, NR-NR)
Conclusions Niraparib maintenance therapy demonstrated encouraging efficacy in patients with uterine serous carcinoma who achieved CR or PR to the last line of platinum-based chemotherapy.
Compared with previous patient outcomes, PFS rates at 12 months (81.8%) showed a significant clinically meaningful improvement and extended the uterine serous carcinoma patient's recurrence (median PFS: 32.4m) in patients receiving niraparib maintenance therapy.

OR_053

Immunotherapy and progestin therapy lead to different pathological responses and immune microenvironment as fertility-sparing strategies in MMRd endometrial cancer

Yiqin Wang Linlin Bo He Li Nan Kang Bo Han Jianliu Wang*
Peking University People's Hospital

Purpose Mismatch repair deficient (MMRd) endometrial cancer (EC) is generally considered unsuitable for fertility-sparing treatment (FST) and has been shown to be resistant to progestin therapy. However, the efficacy and underlying mechanisms of immunotherapy remain unclear.
Methods In the exploratory 2024MMRdECFerSp study (NCT06549855), patients with MMRd EC received a combined regimen of ICI and progestin. The clinicopathological characteristics and treatment outcomes were compared between the patients receiving combined therapy (ICI group, n=5) and those receiving historical progestin therapy (progestin group, n=10).
Results A total of 161 patients underwent FST between June 2020 and March 2024, of whom 15 had the MMRd subtype (9.3%). In terms of treatment response, 4/5 patients in the ICI group achieved complete response (CR), compared to 3/10 in the progestin group. The ICI group exhibited a significantly higher cumulative CR rate than the progestin group (P=0.030). The mIF results indicated that ICI treatment significantly reduced Foxp3+ Treg cells (P=0.034), whereas progestin treatment did not (P=0.706). Compared to pre-treatment levels, progestin treatment showed a tendency to increase CD56+ dim NK cells more than ICI treatment (6.30 vs. 2.79, P=0.051). Further analysis based on treatment response revealed that non-responders had higher levels of Foxp3+ CD4+ Treg cells after treatment (P=0.070). Both M2 and M1 macrophage expression appeared to be lower in responders compared to non-responders (P=0.061 and P=0.070).
Conclusion ICI treatment represents an effective strategy for FST in MMRd EC patients. The ICI regimen induces distinct changes in the immune microenvironment compared to progestin treatment, particularly in the reduction of Foxp3+ CD4+ Treg cells, which may contribute to the different therapeutic responses.

OR_054

Ovarian involvement in fertility-sparing treatment of endometrial cancer

Xuan Zong Kezhen Zhang Dongyan Cao Lingya Pan Jie Yang Jiaxin Yang*
Department of Obstetrics and Gynecology, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences & Peking Union Medical College, Beijing, China

Objective To assess optimal surgical strategies for ovarian involvement in fertility-sparing treatment of endometrial cancer by analyzing selection criteria and prognostic differences between conservative and comprehensive staging procedures.
Methods This retrospective study reviewed 18 patients who developed ovarian masses during fertility-sparing treatment for endometrial cancer (EC) or endometrial intraepithelial neoplasia (EIN) between January 2010 and December 2023 at Peking Union Medical College Hospital (PUMCH).
Results The median age of patients at diagnosis was 32 years (range, 23–40 years). Thirteen patients (72.2%) achieved complete response (CR). Ovarian masses were detected before treatment (4/18, 22.2%), during the primary treatment (6/18, 33.3%) or post-CR (8/18, 44.4%). Nine patients (50.0%) underwent comprehensive staging surgery (CSS), and nine (50.0%) underwent fertility-sparing surgery (FSS) due to ovarian mass detection. Among the six patients who detected ovarian masses during primary treatment, four failed to achieve CR within a median treatment duration of 11.5 months (range:

3–18 months) and subsequently underwent CSS; two continued with FSS and achieved CR. For eight patients who developed ovarian masses post-CR, four with negative endometrial biopsy results underwent FSS, while all four patients who underwent CSS were found to have recurrent endometrial cancer. Final pathological examination confirmed that 15 cases (83.3%) were double primary tumors, with seven patients undergoing CSS and eight FSS. Among these, nine cases were synchronous cancers. Three cases (16.7%) involved ovarian metastasis; two of these patients underwent CSS, while one patient refused CSS and adjuvant chemotherapy, achieving complete response after 30 months of conservative treatment. Ten patients received adjuvant therapy, including eight who underwent CSS and two FSS. Among patients who underwent FSS with fertility plans, pregnancies were successfully achieved in 3 of 7 patients (42.8%), with one live birth. With a median follow-up period of 48.0 months (range, 20–193 months), sixteen patients (88.9%) remained disease-free post-surgery. One patient who underwent CSS died from the disease following recurrence at 33 months, while another patient who underwent FSS experienced recurrence at six months but achieved complete response after 20 months of conservative treatment and remained disease-free.
Conclusion Fertility-sparing surgery is a promising option for young patients with low-risk endometrial cancer (EC) or endometrial intraepithelial neoplasia (EIN) who present with ovarian masses suggestive of double primary disease. Careful preoperative imaging and histological evaluation are essential for effective risk stratification and for excluding other metastatic lesions. Patients should be thoroughly informed about the potential risks associated with conservative treatment.

OR_055

Cell block whole slide image analysis for early endometrial cancer diagnosis in post-menopausal women

Yi-xin Li Wen Lu*
Shanghai First Maternity and infants Hospital

Research Purpose Endometrial cancer is one of the most common gynecological malignancies worldwide. Its early diagnosis and treatment are crucial for improving patient prognosis. Endometrial cell block (ECB) technique is a screening method offering advantages such as non-invasiveness and preservation of endometrial tissue architecture. However, its accuracy relies on the subjective interpretation of pathologists. This study aims to develop an artificial intelligence (AI) model based on weakly supervised learning to achieve high-speed automatic classification and diagnosis of cell blocks. Furthermore, a web application was built to deploy the model for clinical practice.
Materials and Methods This study prospectively collected 216 endometrial cytology samples from three independent hospitals, allocated for model training, model testing, and application deployment, respectively. All samples underwent standard cell block preparation followed by whole-slide imaging (WSI) scanning. Preprocessing of WSIs included background removal, color normalization, and tiling into 128x128 pixel image patches. K-nearest neighbors (KNN) analysis was used to screen for patches with higher diagnostic value. At the patch level, four convolutional neural network (CNN) models with different architectures were used for classification studies. At the whole-slide level, three methods, including majority voting, were employed to aggregate patch-level predictions, and the results were compared with pathologists' diagnoses. Finally, the model was locally deployed using FLASK and DOCKER.
Results At the patch level, the Inception-V3 model demonstrated the best classification performance, achieving a sensitivity of 92.2%, specificity of 82.5%, and an area under the curve (AUC) of 0.964 on the test set. At the whole-slide level, the model combining Term Frequency-Inverse Document Frequency (TF-IDF) indexing with Support Vector Machine (SVM) achieved the optimal performance for classification tasks, with a sensitivity of 96.0%, specificity of 98.1%, and an AUC of 0.973. The diagnostic capability of this model showed high concordance with pathologists' diagnoses. The model's performance on the locally hosted web application showed no significant degradation: at the patch

level, it achieved a sensitivity of 87.0%, specificity of 83.1%, and AUC of 0.947; at the whole-slide level, it achieved a sensitivity of 91.3%, specificity of 87.7%, and AUC of 0.947.
Conclusion This study demonstrates the potential of an AI model combining weakly supervised learning and clustering methods for identifying endometrial cancer. The model requires no manual annotations and automatically extracts information from cell blocks to provide accurate diagnoses, exhibiting good generalization capabilities across multiple independent datasets. Combined with a locally hosted web application, it enables real-time image upload and diagnosis. Future work will focus on expanding the sample size and validating the model's device adaptability and clinical utility in multi-center studies.

OR_056

IGFBP2 promotes proliferation and glycolysis of endometrial cancer by regulating PKM2/HIF-1α axis

Yuxi Jin Ruixia Guo*
The First Affiliated Hospital of Zhengzhou University

Background In recent years, the incidence and mortality rates of endometrial cancer (EC) have been rising. There is growing evidence that glucose metabolism reprogramming plays a pivotal role in cancer progression. Exploring the mechanism of glucose metabolic reprogramming in the development of EC is vital for discovering potential therapeutic targets. This study aims to investigate the biological roles of IGFBP2 in EC and its regulatory mechanisms on the glycolytic enzyme PKM2.
Methods The expression level of IGFBP2 in EC was measured by qRT-PCR, immunohistochemistry and Western blotting. The biological role of IGFBP2 and PKM2 in cell proliferation and glycolysis was determined by a series of functional experiments in vitro and in vivo. Co-immunoprecipitation, immunofluorescence, cycloheximide chase assays were used to reveal the interaction mechanism of IGFBP2 and PKM2. The transcription of IGFBP2 was assessed by dual-luciferase reporter.
Results IGFBP2 was highly expressed in EC tissues, and its expression was associated with poor prognosis. The overexpression of IGFBP2 promoted proliferation and glycolysis in EC cells, while IGFBP2 knockdown had the opposite effect. Mechanistically, IGFBP2 directly interacted with PKM2 and reduced PKM2 protein degradation. Loss of IGFBP2 expression prevents the switch of cytoplasmic PKM2 to the nucleus. Furthermore, the expression of IGFBP2 was up-regulated under hypoxia and can be directly deactivated by HIF-1α. Finally, we verified the effect of IGFBP2/PKM2/HIF-1α on EC tumor growth in vivo by establishing a mouse transplanted tumor model.
Conclusions This study identified IGFBP2 as an upstream activator of PKM2-driven proliferation and glycolysis in EC cells and provided a new promising therapeutic target for EC.

OR_057

Integrating Homologous Recombination Deficiency Subtyping with TCGA Molecular Classification for Enhanced Prognostic Stratification and Personalized Therapy in Endometrial Cancer

Wei Wang
Key Laboratory of Carcinogenesis and Translational Research (Ministry of Education), Department of Gynecologic Oncology, Peking University Cancer Hospital & Institute, Beijing, 100142, China

BACKGROUND Homologous recombination deficiency (HRD) has emerged as a functional biomarker reflecting genome-wide DNA repair defects and genomic instability. While The Cancer Genome Atlas (TCGA) molecular classification provides valuable prognostic guidance in endometrial cancer (EC), it lacks resolution for DNA repair competency and therapeutic responsiveness. This study aimed to investigate whether HRD subtyping could complement TCGA classification for improved prognostic stratification and therapeutic decision-making.
METHODS A total of 142 EC patients were analyzed using a next-generation sequencing panel and genomic scar-based HRD scoring (loss of heterozygosity, telomeric allelic imbalance, large-scale state transitions). Unsupervised clustering stratified patients into HRD-High, -Middle, and -Low groups.Maximally selected rank statistics were used to identify prognostic thresholds for HRD scores; Tumor immune microenvironment was characterized by RNA-based immune gene expression profiling and multiplex immunohistochemistry. A support vector machine (SVM) model was developed for recurrence prediction.
RESULTS HRD subtyping identified distinct genomic, pathological, and immunological features. HRD-High tumors were associated with advanced FIGO stages, TP53 mutations, higher chromosomal instability, and elevated $CD8^+PD\text{-}1^+$ T cell infiltration. HRD subtyping independently predicted disease-free survival and showed superior prognostic accuracy (C-index = 0.857) compared to TCGA subtyping (C-index = 0.751). Integrating HRD and TCGA classifiers further improved predictive performance (C-index = 0.903). An SVM model incorporating HRD score and immune features achieved an AUC of 0.733 for recurrence prediction.
CONCLUSIONS HRD subtyping refines risk stratification beyond traditional TCGA classification and identifies patients potentially responsive to immune checkpoint or DNA damage-targeted therapies. Integrating HRD-based genomic instability metrics with molecular and immune profiling supports precision oncology in endometrial cancer.

OR_058

The epidemiological landscape of endometrial cancer worldwide: an analysis of current global patterns, temporal trends, and future burden

Zhuang Li Penglin Liu Kun Song Beihua Kong*
Qilu Hospital of Shandong University

Objective The incidence and mortality rates of endometrial cancer have been increasing globally. We aimed to describe global patterns, temporal trends, and future burden of endometrial cancer.
Methods We extracted data on endometrial cancer from the GLOBOCAN 2022 database, encompassing 185 countries or territories. Both age-standardised rates of incidence and mortality were presented stratified by countries, the 20 United Nations-defined world regions, and the four-tier levels of the Human Development Index (HDI). Time trends (1988–2018) were evaluated using data from the Cancer Incidence in Five Continents plus database and the WHO Mortality Database. The predicted number of new cases and deaths in 2050 was calculated based on demographic projections.

Results Globally, in 2022, there were estimated 420 368 endometrial cancer cases and 97 723 deaths, with a corresponding age-standardised incidence rate of 8.4 and mortality rate of 1.7 per 100 000 women-years. Endometrial cancer incidence rates varied significantly across world regions, with the highest incidence rate observed in Northern America (22.3 per 100 000 women-years) and the lowest in Middle Africa (2.4 per 100 000). Mortality rates were less than four per 100 000 women-years in most countries and regions. Countries with very high HDI had higher incidence rates (15.5 vs 2.9 per 100 000 women-years) and mortality rates (2.6 vs 1.1 per 100 000 women-years) compared to those with low HDI. Over the past decade, a significant increasing trend in the burden of endometrial cancer worldwide were observed among women aged 50 years or older. The burden of endometrial cancer is projected to increase up to 676 296 new cases and 183 093 deaths by 2050, with most cases in countries with very high or high HDI.
Conclusions Endometrial cancer remains a major public health problem, which predominantly affects older women, particularly in high-resourced countries. Our findings suggest the need for targeted cancer control efforts and health system planning to alleviate the current and projected global burden of endometrial cancer.

OR_059

MAL2 通过调节氨酰基-tRNA 生物合成促进子宫内膜癌发展

沈佳琪 汪辉* 殷涛涛
浙江大学医学院附属妇产科医院

研究目的

1.明确 MAL2 在子宫内膜癌中的表达情况及其与临床病理特征的关系；
2.阐明 MAL2 通过调节氨酰基-tRNA 生物合成促进子宫内膜癌增殖的具体机制。

材料与方法

本研究基于团队先前 24 例内膜癌样本单细胞转录组学分析结果，结合 TCGA 数据库的 UCEC 转录组数据及相关临床数据，联合在线分析工具等，全面评估内膜癌中 MAL2 的表达模式、对预后的影响和与临床病理特征的关系。在内膜癌细胞系 Ishikawa 和 HEC1A 中通过感染 MAL2 shRNA 慢病毒下调 MAL2 基因表达，同时将感染阴性对照的细胞系作为对照组，采用 CCK-8、克隆形成和 EdU 实验探究细胞的增殖行为能力，并进行转录组学和蛋白质组学测序，利用差异基因筛选、KEGG、GSEA等分析方法寻找 MAL2 影响肿瘤生长的调控通路和下游靶基因，通过蛋白免疫印记实验验证 MAL2 与相关通路存在关联，通过蛋白免疫印记实验验证 MAL2 对下游靶基因表达的影响，在 Ishikawa 和 HEC1A 细胞系中转染下游靶基因的 siRNA 探究其对细胞增殖能力的影响，采用 tRNA-PCR 实验检测 MAL2 对于特定氨基酸-tRNA 总的水平的影响。

结果

1.在 UCEC 中 MAL2 启动子区甲基化程度降低，蛋白水平增加，与内膜癌不良预后呈显著相关性（$P<0.05$）；MAL2 在内膜癌细胞中高表达，其表达水平与确诊年龄、组织学类型、分级分期、分子分型、手术方式、放疗治疗等因素密切相关（$P<0.05$）；
2.在高表达 MAL2 的细胞系中敲低 MAL2 会显著抑制细胞的增殖；
3.基于 RNA-seq 结果分析显示，MAL2 与 PI3K-AKT 通路显著相关，敲降 MAL2 会显著限制 PI3K-AKT 通路活性；基于蛋白质组学结果分析显示，MAL2 涉及参与多条代谢相关通路，其中包括氨酰基-tRNA 生物合成途径，敲低 MAL2 会降低该通路下游靶分子如 VARS、FARSB、AARS 等氨酰基-tRNA 合成酶的表达，加入 PI3K-AKT 的激动剂则能逆转 VARS 和 FARSB 的表达下调，同时下游分子 VARS、FARSB 的低表达与内膜癌细胞增殖抑制息息相关；
4.在高表达 MAL2 的细胞系中敲低 MAL2 会上调 $tRNA^{Val}_{AAC}$ 和 $tRNA^{Phe}_{GAA}$ 的含量。

结论

1. MAL 蛋白家族成员 MAL2 的高表达与子宫内膜癌患者的不良预后显著相关；

2.敲低 MAL2 对子宫内膜癌细胞增殖有显著抑制作用；
3. MAL2 通过 PI3K-AKT 信号通路调节氨酰-tRNA 合成酶；
4.VARS 和 FARSB 的表达下调显著抑制子宫内膜癌细胞的增殖；
5.敲低 MAL2 可以上调子宫内膜癌细胞中的 $tRNA^{Val}_{AAC}$ 和 $tRNA^{Phe}_{GAA}$。

OR_060

Correlation of estimated glucose disposal rate with endometrial cancer:cross‑sectional data from the National Health and Nutrition Examination Surveys (NHANES) 1999–2018

rui kong Zhiqi Wang*
Beijing Friendship Hospital, Capital Medical University

Objective Endometrial cancer (EC) is one of the most common cancers in the female reproductive system, with over 300,000 new cases reported annually worldwide. Although most patients have a good prognosis,20–30% of patients with advanced disease have a poor prognosis. There are currently no reliable biomarkers for early diagnosis and efective prognostic improvement of the disease. The purpose of this study was to explore
the correlation between estimated glucose disposal rate (eGDR) and the risk of developing EC.
Methods This study included 31666 female subjects in the NHANES database from 1999 to 2018, including 31418 non-oncology patients and 248 EC patients. Selection bias was reduced using 1:1 propensity score matching (PSM) method. Restricted cubic spline (RCS) was plotted to explore the non-linear relationship between eGDR and EC risk.XGBoost algorithm was utilized to conduct machine learning analysis on the related factors of endometrial cancer.
Results Using data from the NHANES database from 1999 to 2018, the association between eGDR and the risk of developing EC was assessed. The results of the 1:1 ratio propensity score matching (PSM) showed 248 each for EC patients and non-oncology participants. The eGDR in the EC group (6.84±2.76) was significantly lower than that in the control group(7.51±2.66) (P=0.004). After adjusting for confounding variables, the odds ratio (OR) for eGDR as a protective factor against EC was 0.906 (95% CI: 0.826-0.991, P=0.0328). A significant decreasing trend in EC risk was observed across eGDR quartiles and revealed that both models with additional covariates showed consistent negative correlations similar to Model 1. In Model 2 (OR: 0.917, 95% CI: 0.849-0.988, P=0.024) and Model 3 (OR: 0.906, 95% CI: 0.826-0.991, P=0.0328).In addition, the RCS showed a signifcant non-linear correlation between eGDR and EC risk.XGBoost algorithm indicated that the contribution degree of eGDR to the model prediction is medium.A high characteristic value of eGDR may be associated with a reduced prevalence of endometrial cancer
Conclusions This study concludes that higher eGDR levels are significantly associated with a reduced risk of EC, suggesting its potential role as a protective factor. These findings underscore the importance of metabolic health in the prevention of EC and warrant further investigation into the underlying biological mechanisms.

OR_061

Development of a blood cell analysis-based artificial intelligence model for early detection of ovarian cancer

Se Ik Kim Maria Lee HHee Seung Kim Hyun Hoon Chung Jae-Weon Kim*
Department of Obstetrics and Gynecology, Seoul National University College of Medicine, Seoul, Republic of Korea

Objectives Ovarian cancer (OC) remains one of the most lethal gynecologic malignancies, primarily due to delayed detection. Blood cell analysis presents a promising non-invasive diagnostic approach, capable of detecting systemic signals associated with early OC. This study aimed to develop and validate an artificial intelligence (AI)-based diagnostic model to detect OC risk signals using comprehensive blood cell data.
Methods A total of 185 blood samples (85 for training and 100 for testing), obtained from patients with OC or benign ovarian tumors, and healthy controls, were analyzed. The model utilized 24 features: 18 directly measured blood cell parameters (via Sysmex) and 6 derived composite indices, including the systemic immune-inflammation index (SII). A Random Forest algorithm, optimized through 5-fold GridSearch cross-validation and soft voting, was trained and validated with balanced accuracy (BA) as the performance metric. The optimal cutoff values were determined based on the training set's best BA.
Results In the training set, the model achieved a sensitivity of 100.0%, specificity of 92.6%, a positive predictive value (PPV) of 77.3%, and a negative predictive value (NPV) of 100.0%, with an area under the curve (AUC) of 0.987. For the test dataset, the model demonstrated a sensitivity of 75.0%, specificity of 92.9%, PPV of 66.7%, and NPV of 95.1%, with an AUC of 0.923. The platelet-to-lymphocyte ratio and SII emerged as the most influential features, contributing significantly to model performance. The specificity for control samples, excluding benign ovarian tumors, was 1 in the training set, and validation confirmed a specificity of 0.984, demonstrating clear separation between the OC and control groups. Composite indices further improved the model's reliability in detecting OC.
Conclusions This AI-driven blood cell analysis model exhibited strong performance in identifying OC, achieving high sensitivity and specificity. By leveraging non-invasive biomarkers, it holds significant promise for clinical implementation in OC screening.

OR_062

SDC4-MMP14 regulates tumor cell-fibroblast crosstalk and drives epithelial ovarian cancer metastasis through extracellular matrix remodeling

Xin Hou Bin Yang Gang Chen Chaoyang Sun*
Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology

Objective Epithelial ovarian cancer (EOC) is the most lethal gynecological malignancy. Its strong invasiveness and frequent metastasis contributed primarily to the high mortality rate. The heterogeneity and dynamic alteration of tumor microenvironment (TME) serve as a driving force of metastasis. This study aims to dissect the crosstalk and pro-tumour mechanisms in TME and identify novel therapeutic targets.
Materials and methods We characterized cellular heterogeneity and spatial architecture in primary and metastatic TME of EOC through integrated single-cell RNA sequencing (scRNA-seq) and spatial transcriptomics. Bioinformatic analyses revealed intercellular crosstalk networks and functional properties. Multiplex immunohistochemistry (mIHC) and 3D co-culture systems validated spatial and functional cell-cell interactions. Second harmonic generation (SHG) microscopy visualized collagen

fiber orientation. Mechanisms were further elucidated using siRNA, CRISPR-Cas9, His-pulldown, Co-IP, migration and invasion assays, and cell-derived xenograft (CDX) models.
Results scRNA-seq and spatial transcriptomics identified a syndecan-4 (SDC4)-enriched cancer cell cluster (C40) enriched in metastatic lesions and associated with poor prognosis. C40 cells spatially localized at the invasion front, surrounded by cancer-associated fibroblasts (CAFs). CellChat analysis revealed robust CAF-cancer cell interactions, with SDC4 as a key membrane receptor on C40 cells. Functional validation showed that SDC4 knockdown (siRNA) or knockout (CRISPR-Cas9) significantly suppressed ovarian cancer cell invasion, migration, and epithelial-masenchymal transition (EMT) in vitro and in vivo. Integrated His-pulldown and 4D smart-DIA proteomics identified CAF-derived ligand MMP14 as an SDC4 interactor, validated by mIHC, 3D co-culture, and Co-IP. MMP14-SDC4 interaction actives EMT in tumor cells. And MMP14+CAFs further assist EOC cells invasion via remodeling ECM alignment. A novel anti-SDC4 therapeutic antibody inhibited the MMP14-SDC4 axis and reduced ascites and metastasis in vivo.
Conclusion These findings identified a specific SDC4+ tumor cell cluster that are associated metastasis and poor prognosis. CAFs interacted with this cluster via MMP14-SDC4 axis, promoting cancer progression through ECM remodeling and activating EMT. Critically, our self-developed anti-SDC4 neutralizing antibody targeting this axis suppressed metastasis in preclinical models. This study uncovers novel mechanisms of EOC aggressiveness and nominates SDC4 as a promising therapeutic target.

OR_063

BRCA1/2 基因胚系突变卵巢癌患者临床与家系特征分析及高危人群预防现况

陈梦雨
中国人民解放军总医院第一医学中心

目的 分析 BRCA 胚系突变卵巢癌患者的临床特征，并进行家系信息采集，筛查家系亲属中潜在的 BRCA 突变携带者，针对卵巢癌患病高危人群予以预防指导。
方法 选取 77 例 BRCA 基因胚系突变卵巢癌患者为研究对象，根据检测结果分为 BRCA1 突变组(48 例)和 BRCA2 突变组(29 例)，比较两组患者的临床基线资料、病理特征。经遗传咨询采集患者家系信息，对来自 37 个家系的 81 位患者亲属进行 BRCA 基因检测，结合家族史情况和基因检测结果分析家系特征，对高危人群进行预防指导。
结果 77 例患者平均发病年龄 53.8 岁，BRCA1 突变组平均发病年龄 51.7 岁，显著早于 BRCA2 组的 57.2 岁(P<0.05)。43.8%的 BRCA1 携带者具有遗传性乳腺癌-卵巢癌综合征(HBOC)家族史，较 BRCA2 组高 1.5 倍(P<0.05)。两组患者的生育情况、BMI、乳腺癌个人史及治疗前 CA125 水平比较，差异无统计学意义(P>0.05)。参与 BRCA1/2 基因检测的 37 个突变家系中检测出 47 例携带者(58.0%检出率)，其中 63.8%来自无 HBOC 家族史的家系。典型家系显示 BRCA 突变可隐匿传递三代，未表现典型肿瘤聚集现象。31 位女性突变携带者中仅 2 位接受预防性双侧卵巢-输卵管切除术(RRSO)，显著低于国际水平。健康人群调查显示：78.5%非携带女性可接受预防性手术，而携带者群体手术意向平均仅 2.6 分(5 分制)，75.0%倾向延迟至自然绝经后施行。
结论 国内 BRCA1/2 突变携带者对 RRSO 手术带来的医源性绝经顾虑较重，高危人群卵巢癌的手术预防存在决策困境。与国际水平相比，国内 BRCA1/2 突变卵巢癌患者卵巢癌发病年龄较晚，适宜国内女性的手术时机仍需进一步探讨，开发本土化手术时机判定模型有助于推动卵巢癌防治工作。

OR_064

Molecular Subtyping and Its Association with Prognosis in Ovarian Endometrioid Adenocarcinoma

Rongyu Liu Zhengyu Li*
West China Second University Hospital, Sichuan University

Objective To investigate the molecular typing characteristics of patients with ovarian endometrioid adenocarcinoma（OEC）and its relationship with prognosis.
Methods A retrospective analysis was conducted on clinical records and followup outcomes of 176 OEC patients treated at the Department of Gynecology, West China Second University Hospital of Sichuan University from June 2011 to December 2019. Patients were classified into three molecular subgroups based on genetic testing results and immunohistochemical data: mismatch repair deficiency (dMMR) group, no specific molecular profile (NSMP) group, and p53 mutation (p53 abn) group. Clinicopathological characteristics including tumor differentiation grade, FIGO (International Federation of Gynecology and Obstetrics) clinical stage, preoperative CA125 levels, neoadjuvant chemotherapy status, para-aortic/pelvic lymph node metastasis, concurrent endometrial cancer, and malignant transformation of ovarian endometriotic cysts were analyzed across subgroups. Differences among subgroups were compared using Chi-square test, Fisher's exact test, and one-way ANOVA. Kaplan-Meier method was employed to compare overall survival and disease-free survival curves among molecular subgroups and different age groups. This study protocol was approved in accordance with the ethical standards of the World Medical Association's Declaration of Helsinki (2013 revision).
Results ① Among all patients, 22 cases were classified as dMMR subtype, 71 as NSMP subtype, and 83 as p53 abn subtype. No statistically significant differences were observed among molecular subgroups regarding age, body mass index (BMI), menopausal status, or presence of comorbidities (F=2.235, 0.655; χ^2=4.552, 1.233; P=0.110, 0.521, 0.103, 0.540, respectively). ② There were no significant differences across molecular subgroups in tumor differentiation grade, FIGO clinical stage, preoperative CA125 levels, neoadjuvant chemotherapy administration, para-aortic/pelvic lymph node metastasis, concurrent endometrial cancer, or malignant transformation of ovarian endometriotic cysts (χ^2=5.019, 2.574, 2.445, 1.785, 1.678, 0.883, 5.837, 0.230; P=0.081, 0.631, 0.295, 0.410, 0.432, 0.643, 0.054, 0.891, respectively). ③ Significant differences in overall survival (OS) and progression-free survival (PFS) curves were identified among the three molecular subtypes (χ^2=6.539, 7.715; P=0.038, 0.028, respectively). The dMMR subgroup demonstrated superior OS and PFS compared to both NSMP and p53 abn subgroups (OS: χ^2=2.118, 5.053; P=0.146, 0.025; PFS: χ^2=2.394, 5.566; P=0.122, 0.018).
Conclusions Patients with dMMR-type OEC demonstrated significantly superior progression-free survival (PFS) and overall survival (OS) compared to those with p53 abn-type. Individualized treatment strategies tailored to molecular classification characteristics of OEC may improve clinical outcomes for these patients.

OR_065

卵巢未成熟畸胎瘤基因组学研究新进展

张可珍 宗璇 杨佳欣*
国家妇产疾病临床医学研究中心 中国医学科学院北京协和医学院，北京协和医院妇产科

目的 卵巢未成熟畸胎瘤（ovarian immature teratoma，OIT）是一种好发于年轻女性的恶性生殖细胞肿瘤，其发病机制尚不明确，缺少诊断及疾病危险分层的特异性标志物。本研究旨在综述卵巢未成熟畸胎瘤的基因组学和表观遗传学研究进展，探讨其分子机制及潜在的临床应用价值。
方法 通过检索 2015-2025 年 Web of Science、PubMed 和 CNKI 中英文文献，纳入 25 篇有关 OIT 基因组学和表观遗传学的研究文献，包括全外显子测序、DNA 甲基化分析、miRNA 表达谱研究等。
结果 在遗传因素方面，OIT 主要表现为近二倍体基因组和广泛的杂合性缺失，体细胞突变率极低，拷贝数变异罕见，提示其发病机制与其他类型的生殖细胞肿瘤不同。孤雌生殖理论是目前 OIT 起源的主流假说，研究表明其可能源于减数分裂异常的生殖细胞。尽管尚未在散发病例中发现重复性的致病性基因突变，但家系研究发现 BMP15 基因突变可能与遗传性 OIT 的发病相关。表观遗传学研究发现，OIT 中母源性印记基因呈现高甲基化状态，而父源性印记基因甲基化水平较低，支持其孤雌生殖起源。此外，miRNA 表达谱分析显示，miR-199b-5p 在 OIT 中特异性上调，但其功能机制仍需进一步探索。在临床应用方面，循环 miRNA（如 miR-371a-3p）和其他液体活检技术可能为无创诊断和监测复发提供新方向。治疗上，由于早期 OIT 术后是否需辅助化疗的仍存争议，未来可探寻分子标志物进一步进行风险分层，避免过度治疗。
结论 OIT 在基因组和表观遗传学上具有独特特征，深入研究其分子机制有助于进一步了解其发病机理。未来的研究应聚焦于探索特异性标志物，优化风险分层和个体化治疗策略，以改善患者预后。

OR_066

信迪利单抗联合紫杉醇周疗和安罗替尼治疗铂耐药复发性上皮性卵巢癌/输卵管癌/原发腹膜腺癌的有效性和安全性的开放、多中心、单臂、II 期临床研究的中期分析

李润璇 丛晓凤 陈晨 刘增光 岳清 朱颜铂 张诗林 尹佳馨 赵淞辰 徐子钧 高杰 于沛妍 高敏 刘子玲*
吉林大学第一医院

研究背景 卵巢癌是妇科恶性肿瘤中致死率最高的肿瘤，多数患者首次确诊即为晚期，且极易复发并发展为铂耐药型卵巢癌，其二线化疗效果有限，患者的中位生存期较短。近年来，免疫检查点抑制剂的出现为晚期恶性肿瘤的治疗带来了新曙光，与化疗以及抗血管生成治疗联合应用时，展现出显著的协同效应。然而，该策略在铂耐药卵巢癌患者中的疗效与安全性仍需进一步验证。研究目的：本研究旨在评估信迪利单抗联合紫杉醇周疗与安罗替尼治疗铂耐药卵巢癌患者时的有效性和安全性。
材料与方法 本研究（临床试验注册号：ChiCTR2000035072）纳入了一线以上治疗失败的铂耐卵巢癌患者（n=17）。患者接受如下联合治疗方案：信迪利单抗 200 mg，Q3W, 静脉输注，安罗替尼 10 mg, QD, 口服（连续服药 2 周，停药 1 周），紫杉醇 80 mg/m²，每 3 周为一个治疗周期（第 1 天、第 8 天、第 15 天静脉输注）。在联合用药 4-6 个周期后，采用信迪利单抗及安罗替尼维持治疗，直至疾病进展或出现不可耐受的毒性等。主要研究终点为无进展生存期（PFS），次要研究终点包括客观缓解率（ORR）、缓解持续时间（DoR）、疾病控制率（DCR）和总生存期（OS）。安全性指标包括不良事件（AE）和严重不良事件（SAE）的发生率。
研究结果 在 14 例可评估的患者中，5 例达到部分缓解（PR, 35.7%），6 例达到疾病稳定（SD, 42.9%）。经研究者确认的 ORR 为 35.7%（95%CI，12.8-64.9%），DCR 为 78.6%（95%CI，49.2-

95.3%）。中位 PFS 为 5.7 个月（95%CI，3.5-NR 个月），6 个月的 PFS 率为 50.0%（95%CI，29.6-84.4%），1 年的 PFS 率为 16.7%（95%CI，4.8-58.0%）中位 DoR 为 2.5 个月（95%CI，0.0-NR 个月）。1 名患者的 PFS 和 DoR 分别达到了 16.6 个月和 15.3 个月，且这名患者对治疗持续有反应。85.7%的患者出现了靶病灶缩减，最大靶病灶缩小值达到了 38.8 mm（79.5%）。安全性方面，所有患者均出现了≥1 级的不良反应（AE）。然而，仅有 3 名（17.6%）患者发生了≥3 级的 AE，且未出现发生 5 级 AE、SAE 或死亡的患者。

结论 本研究中期结果达到预期目标，信迪利单抗联合紫杉醇周疗与安罗替尼在治疗铂耐药卵巢癌中显示出显著的疗效和可接受的安全性。

OR_067

经胸腔镜辅助对比经腹-膈肌入路行卵巢癌心膈角淋巴结切除术的临床分析

关欣[1] 佟锐[2*]
1.中国医科大学附属盛京医院 2.辽宁省肿瘤医院

研究目的 晚期卵巢癌患者中，膈上淋巴结（Cardiophrenic Lymph Nodes, CPLNs）转移的发生率为 10%-30%，其存在与不良预后显著相关。然而，CPLNs 位于膈肌与心包之间，解剖位置复杂，传统开胸手术创伤大、恢复慢。目前，临床多采用经腹-膈肌入路切除心膈角淋巴结，但此种方式可增加术后肺内感染、胸腔种植转移等风险。本研究拟探讨不同手术入路（经胸腔镜辅助与经腹-膈肌）进行心膈角淋巴结切除的临床效果差异。

材料与方法 本研究选取 2023.9-2025.6 于辽宁省肿瘤医院心膈角淋巴结切除达全身 R0 的卵巢癌满意减瘤术的患者 32 例，其中经胸腔镜辅助切除心膈角淋巴结 12 例，经腹-膈肌切除 20 例。对比分析了经不同手术入路心膈角淋巴结切除数量、淋巴结阳性率，胸腔手术时长，手术总时长，出血量，术后肺内感染发生率、胸腔引流量、术后住院时间以及住院总费用等指标。

研究结果 两种手术方式入组患者均全腹 R0，从而达到全身 R0；两组患者手术总时长、出血量、术后住院时间及住院总费用均无统计学差异；胸腔手术时长、胸腔积液发生率、积液量及胸腔感染发生率胸腔镜辅助组均低于经腹-膈肌组（P＜0.05）；CPLNs 完整切除率、切除数量与术后 CA125 下降幅度胸腔镜辅助组均高于经腹-膈肌组（P＜0.05）。

结论 与经腹-膈肌入路相比，胸腔镜辅助切除心膈角淋巴结可在不增加手术总时长、失血量、术后住院时间或总住院费用的情况下，获得更佳的心膈角淋巴结切除率和术后恢复率，安全性更高。与经腹-膈肌入路相比，它具有更佳的临床推广价值。

OR_068

Fertility-sparing Treatment in Early-Stage Endometrial Cancer with Synchronous or Metachronous Ovarian Cancer: Outcomes and Risk Factors

siru Liu Yiqin Wang Xiaobo Zhang Yifan Feng Yuanyuan Liu Jianliu Wang*
Peking University People's Hospital

Purpose To evaluate the outcomes and identify the risk factors of patients with early-diagnosed endometrial cancer (EC) accompanied by synchronous or metachronous ovarian malignant or borderline tumors (EON) who undergo fertility-sparing treatment (FST), thereby providing evidence to optimize management of this special cohort.

Methods We retrospectively analyzed 355 patients with early-stage EC or atypical endometrial hyperplasia receiving FST from 2011 to 2024 at Peking University People's Hospital. Twenty (5.6%) developed EON and were matched 1:4 to EC-only controls (EC). Oncologic and fertility outcomes, pathological features, and molecular profiles were compared via Student's t-test, Mann–Whitney U test, or the Fisher's exact. Risk factors for EON were identified using multivariable logistic regression, with subsequent ROC curve analysis for predictive modeling.
Results For EON patients, the median time to ovarian tumor detection was 9 months during FST; 20% were synchronous at baseline and 30% appeared after endometrial CR (median 41 months). Baseline characteristics revealed that the EON group tend to exhibit a higher incidence rates of thyroid disease (25% vs. 9.2%) and intrauterine adhesions (25% vs. 10.8%) compared to the EC group. Also, EON patients tended to have lower response rate to FST (75% vs 92.3%, $p = 0.05$) and higher recurrence rate (35% vs 18.5%, $p = 0.135$). Among the patients with fertility willing, pregnancy rates were comparable (45.5% vs 28.6%, $p = 0.453$). Pathological (ER, PR, Ki67) and molecular features of paired endometrial and ovarian tumors were highly concordant. Notably, Ki-67 expression was significantly higher in the EON group (median: 40% vs. 30%, $p=0.015$). And a based predictive model incorporating thyroid disease, intrauterine adhesions, Ki67 level, and treatment response,showed good discrimination for EON (AUC=0.856).
Conclusion FST may be feasible in select patients with EON. Risk-based models can guide individualized management and improve safety in fertility-preserving strategies.

OR_069

Lactate: An Accomplice in Ovarian Cancer Drug Resistance

Lingfang Wang Xiaodong Cheng* Jiaxin Gu Zhuoqun Lin Ziyu Xing Fenfen Wang
Women's Hospital, Zhejiang University School of Medicine

Lactate was once regarded as a mere waste product of metabolism. However, recent studies have revealed that lactate within tumor cells plays a significant role in promoting resistance to chemotherapy, though its precise mechanisms remain unclear. Our team is dedicated to investigating the role and molecular mechanisms of lactate in driving drug resistance in ovarian cancer.
Through our research, we identified that elevated lactate levels are a critical factor contributing to resistance against platinum-based chemotherapy and PARP inhibitors (PARPi) in ovarian cancer. Building on these findings, we developed a lactate-associated small-molecule drug library and conducted high-throughput screening, leading to the identification of small-molecule drugs such as STF-31, which significantly enhance drug responsiveness in ovarian cancer. Furthermore, we designed a targeted delivery system specifically aimed at reducing lactate production, offering a promising strategy to overcome drug resistance.
On a mechanistic level, we discovered that lactate-mediated lactylation modifications of NSUN2 and PARP1 are pivotal drivers of platinum and PARPi resistance in ovarian cancer.
Our study not only elucidates the molecular pathways through which lactate contributes to resistance to both conventional and targeted therapies but also establishes innovative drug-sensitization strategies based on these insights. These findings hold substantial clinical significance and pave the way for improved therapeutic approaches in ovarian cancer treatment.

OR_070

Identification of serum peptide biomarker for predicting first-line chemotherapeutic efficacy in advanced ovarian cancer by Clin-TOF-II-MS with magnetic beads technology

Lin Xiu Ning Li Lingying Wu*
Cancer Hospital of Chinese Academy of Medical Sciences

Objective To explore the serum protein biomarkers for predicting the first-line chemotherapeutic efficacy in advanced ovarian cancer with MALDI-TOF-MS combined with magnetic beads technology.
Methods 64 patients admitted in the Department of Gynecologic Oncology, Cancer Hospital of Chinese Academy of Medical Sciences from March 2018 to September 2018, received neoadjuvant chemotherapy interval debulking surgery (NACT-IDS) and adjuvant chemotherapy, confirmed pathologically as stage III/IV epithelial ovarian cancer (non-mucinous carcinoma, non-low-grade serous carcinoma), from whom chemotherapy-sensitive patients with progression-free survival (PFS) >12 months and chemotherapy-resistant patients with PFS <6 months after adjuvant chemotherapy were selected as the study subjects. The serum in patients' first visit were collected. Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) was used for peptide profiling in serum samples to compare significantly differently expressed peptides between chemotherapy-sensitive group and chemotherapy-resistant group (score >5) and screen peptide peaks with AUC ≥ 0.8. Liquid chromatography-mass spectrometry (LC-MS/MS) was further used to determine the composition of differential expressed peptides.
Results A total of 35 subjects who met the initial criteria (25 chemotherapy-resistant patients and 10 chemotherapy-sensitive patients) were included. The last follow-up date was January 10, 2020, and the median follow-up time was 16 months. All the 25 cases in the chemotherapy-resistant group relapsed, and 2 in the 10 cases in the chemotherapy-sensitive group relapsed. In the range of 1000-10000mz, 71 differential peptide peaks were initially detected between chemo-resistant and chemo-sensitive groups. ROC curve analysis showed that among the 71 differential protein peaks, there were 22 differential polypeptide peaks with AUC≥0.8, of which 10 were highly expressed in the chemotherapy-resistant group, and 12 in the chemotherapy-sensitive group. LC-MS/MS method identified the protein components of 4 polypeptide peaks among the 10 differential expressed polypeptide peaks in chemotherapy-resistant group, 4634.22m/z as fragments of serum amyloid A-1, 1287.56m/z as fragments of Prothrombin, 1099.56m/z and 2018.39m/z as fragments of Complement C3. The sensitivity and specificity of the three proteins for predicting first-line chemotherapeutic efficacy were 100%, 100%, 96% and 80%, 100%, 100%. No protein components were identified among the 12 differential protein peaks in the chemotherapy-sensitive group.
Conclusion Serum amyloid A-1, Prothrombin and Complement C3 were highly expressed in chemotherapy-resistant patients, which may be related to the poor efficacy of paclitaxel/carboplatin first-line chemotherapy, and it is necessary to further explore their predictive value.

OR_071

内质网应激反应与卵巢癌铂耐药表型维持的相关性研究

王辰宇 邱明慧 张瑜*
中南大学湘雅医院

研究目的 卵巢癌是最常见的妇科恶性肿瘤，约 70%的患者经过满意的肿瘤细胞减灭术和规律的含铂化疗后出现铂耐药导致肿瘤复发。卵巢癌铂耐药是其致死率居高不下的主要原因之一。因此，探索卵巢癌铂耐药机制，改善铂耐药患者预后具有重要的临床意义。肿瘤细胞通过合成大量新生蛋白质来满足快速增殖的需求，内质网应激反应（ER stress）是维持细胞新生蛋白质正确折叠的主要方式，其稳态失衡与多种肿瘤发生、发展密切相关。肿瘤细胞在化疗药物作用下易出现错误折叠蛋白累积，启动内质网应激反应，进而参与调控肿瘤细胞对化疗药物的敏感性。目前，有关内质网应激反应参与调控卵巢癌铂耐药的研究还有待完善。

材料与方法 采用顺铂维持处理的方法构建卵巢癌铂耐药细胞株(A2780-cisR)，并通过 CCK8 方法比较亲本细胞和铂耐药细胞对顺铂的敏感性及细胞增殖能力。利用流式细胞术检测亲本及铂耐药卵巢癌细胞凋亡和周期变化。通过转录组测序，比较亲本和铂耐药细胞中内质网应激反应相关分子表达差异，并运用 Western-Blot，qPCR 方法进一步证实细胞内质网应激反应相关分子的蛋白及 mRNA 表达水平。给予内质网应激反应抑制剂 4-PBA 处理，观察卵巢癌铂耐药细胞株 A2780-cisR 在顺铂处理下，细胞增殖能力、凋亡水平及内质网应激反应相关分子表达变化。

结果 铂耐药细胞株 A2780-cisR 对顺铂耐受性较亲本细胞显著提高（IC50: 15.2μmol/L vs 4.8μmol/L，P<0.05），且多数细胞处于 G1/G0 的静息周期（P<0.05），但铂耐药细胞株与亲本细胞株的凋亡率并无显著差异。

A2780-cisR 转录组测序结果显示：内质网应激反应相关分子 PDIA4，HSPA5 和 CALR 的 mRNA 水平较其亲本细胞，显著降低（P<0.05），而 EIF2AK3，EIF2A 和 ERN1 则表达增高（P<0.05）。Western blot 结果显示铂耐药细胞株中 PDIA4，HSPA5，及 CALR 蛋白表达较亲本细胞低，同时 EIF2AK3，EIF2A 和 ERN1 蛋白水平显著增高（P<0.05）。

联合应用顺铂与内质网应激反应抑制剂 4-PBA，能显著降低 A2780-cisR 增殖能力（P<0.05），细胞周期表现为 G2/M 期阻滞，并能促进 A2780-cisR 细胞凋亡（P<0.05）。顺铂与 4-PBA 联合应用能够增强 A2780-cisR 对顺铂的敏感性，可能与上调内质网应激反应相关分子 PDIA4，并抑制其下游 EIF2A、ERN1 表达相关。

结论 细胞内质网应激反应途径，参与维持卵巢癌铂耐药表型。内质网应激反应抑制剂 4-PBA，通过上调 PDIA4 及抑制 EIF2A、ERN1 表达，可部分逆转卵巢癌铂耐药细胞的耐药性。

OR_072

Long-term Prognosis, Socioeconomic Burdern Analysis Over Time of Children, Adolescents and Young Adults Malignant Ovarian Germ Cell Tumor Survivors

Jing Zheng Jie Yang Jiaxin Yang*
Department of Obstetrics and Gynecology, Peking Union Medical College Hospital, National Clinical Research Center for Obstetric & Gynecologic Diseases

Background The number of long-term survivors of malignant ovarian germ cell tumors (MOGCTs) in children, adolescents, and young adults (AYA) is increasing. However, data on long-term survival, secondary tumors, rehospitalization, and socioeconomic impact are limited. As a national rare disease diagnosis and treatment center, we investigated the long-term outcomes of MOGCT patients treated at our institution.

Methods Patients aged under 40 years old who were diagnosed with MOGCTs at Peking Union Medical College Hospital from Jan 2008 to Dec 2024 were retrospectively included. Clinical characteristics, treatment modalities, long-term menstrual and fertility outcomes, healthcare costs, and duration of sick leave, the occurrence of secondary tumors or rehospitalization were analyzed.
Results A total of 359 patients were enrolled, with a mean age 21.1 years old (range: 1 to 39 years old). The median follow-up was 75.2 months (range:3 to 174 months). The recurrence rate was 18.1%, with 65 patients recurring. Among patients who relapsed, seven patients had a progression-free survival time of over two years, with a recurrence rate of 1.9% after two years. 15 patients (4.2%) died, with a 5-year overall survival (OS) rate of 95%. Among 171 patients who underwent fertility-sparing treatments and had available data on menstrual status, 34 experienced amenorrhea prior to illness onset. Of the 26 patients who reached menarche, the average age was 12 years. Eight patients had not yet reached menarche, with an average age of 9.1 years. Among the 137 patients who had experienced menarche before illness onset, only 5 did not recover menstruation during follow-up. The average time for menstrual cycle recovery post-treatment was 3.8 months (range: 0 to 12 months). Recovery duration was significantly associated with chemotherapy and the number of cycles (p=0.000). Among 59 patients planning for pregnancy, 44 pregnancies were achieved, with a successful pregnancy rate of 74.6%. Of these, 34 reached full term, with 26 vaginal deliveries and 8 cesarean sections, resulting in a live birth rate of 57.6%. The mean healthcare cost among 250 patients was RMB 148,800 (range: RMB 10,000-1,000,000), and the median sick leave duration was 10.4 months (range: 1-77 months). Patients in Stage I and those not receiving chemotherapy incurred significantly lower costs and shorter sick leave compared to Stages II-IV and those undergoing chemotherapy, showing statistically significant differences. Among 264 patients who were monitored for subsequent diagnoses or re-hospitalizations, 10 developed subsequent neoplasms, originating from the breast, brain, abdomen, ovaries, cervix, lungs, and thyroid. Additionally, 10 patients were re-hospitalized due to infections affecting the respiratory system, lymphatic vessels, gastrointestinal tract, and sinuses. Seven patients experienced psychological issues, with 3 cases of depression and 4 of anxiety.
Conclusions Long-term outcomes for MOGCTs patients are generally favorable. Most patients undergoing fertility-sparing treatments and chemotherapy resume normal menstruation or achieve menarche. Successful pregnancy and live birth rates are promising for those with fertility desires. However, the socioeconomic burden remains substantial, particularly for patients with advanced-stage disease. Long term risk of subsequent neoplasm, infections and psychological issues in survivors should be paid attention.

OR_073

基于血浆游离 DNA 甲基化标志物的卵巢癌无创早期检测

舒桐 郑虹*
北京大学肿瘤医院

背景 卵巢癌是女性致死率最高恶性肿瘤之一，发病机制复杂，缺乏有效的早期诊断方法。近年来研究表明，循环游离 DNA（cfDNA）中的异常甲基化模式具有高度特异性，可作为恶性肿瘤早诊潜在生物标志物。本研究旨在开发一种基于血浆 cfDNA 甲基化特征的无创检测方法，以提升卵巢癌早期检测效能。
方法 本研究纳入北京大学肿瘤医院经手术病理确诊的卵巢癌、卵巢交界性肿瘤、卵巢良性肿瘤及非肿瘤对照者血浆样本，75 例用于筛选标志物，另 106 例用于模型验证。利用定制的甲基化捕获技术对 cfDNA 进行高通量测序，筛选显著差异甲基化区域（DMRs）（图 1）。通过机器学习算法优化标志物组合，建立基于 TaqMan 探针的甲基化特异性 qPCR 检测体系，并在独立样本中进行验证，评估其诊断效能。
结果 本研究采用甲基化高通量测序技术，通过分析 32 例卵巢癌、5 例卵巢交界性肿瘤及 38 例卵巢良性肿物的血浆 cfDNA 甲基化谱，筛选 DMRs，最终选定 IFFO1、TFAP2E 和 WNT6 基因的三个高甲基

化区域，构建诊断模型（图 2）。在建验证队列中（图 3）（30 例卵巢癌、31 例卵巢良性肿物及 22 例健康对照），明确该模型敏感性为 90%，卵巢良性肿物特异性为 93.55%，健康对照特异性为 100%。其中值得注意的是，该早诊模型对 I/II 期卵巢癌敏感性为 75%，对 III/IV 期卵巢癌敏感性高达 95.45%。
结论 本研究表明，基于外周血 cfDNA 甲基化特征的 qPCR 检测方法为鉴别卵巢肿物良恶性提供了一种无创、高效、低成本的辅助诊断工具。该模型在卵巢癌早期诊断与临床应用方面具有广阔前景，有望改善卵巢癌患者预后。

OR_074

卵巢交界性肿瘤保留生育功能治疗的肿瘤结局分析

郑果
郑州大学第一附属医院

目的 探讨接受保留生育功能手术（FSS）的卵巢交界性肿瘤（BOT）患者的肿瘤结局。
方法 回顾性收集 2007 年 8 月至 2023 年 8 月在郑州大学第一附属医院接受 FSS 的 45 岁以下 BOT 患者的临床病理资料及随访资料，分析其肿瘤结局，采用单因素及多因素 Cox 比例风险回归模型分析影响患者无病生存（DFS）时间的相关因素。
结果 （1）一般临床病理资料：本研究纳入 BOT 患者共 454 例，其年龄为（28.5±7.8）岁，中位肿瘤最大径为 9.9 cm（范围：2.8～90.0 cm），国际妇产科联盟（FIGO）分期为 I 期 310 例、II 期 77 例、III 期 67 例，病理类型为浆液性 221 例、黏液性 156 例、子宫内膜样 6 例、混合型 71 例，肿瘤累及单侧卵巢 311 例、累及双侧卵巢 143 例，伴微浸润 19 例，微乳头亚型 36 例，卵巢外浸润性种植 11 例，合并腹水 86 例。（2）肿瘤结局：中位随访时间为 83 个月（范围：4～213 个月），随访期内 73 例（16.1%，73/454）患者复发；6 例（1.3%，6/454）死亡，3 例（0.7%，3/454）带瘤生存。（3）影响预后的相关因素分析结果 单因素分析显示，累及双侧卵巢、保留生育功能手术、微乳头亚型、卵巢外浸润性种植、FIGO 分期（I/II~III）均显著影响 BOT 患者的 DFS 时间（P 均<0.05）。多因素分析显示，累及双侧卵巢（HR=2.021，95%CI 为 1.252～3.264）、保留生育功能手术（HR=8.541，95%CI 为 2.574～28.336）、微乳头亚型（HR=4.190，95%CI 为 2.085～8.421）、卵巢外浸润性种植（HR=4.091，95%CI 为 1.727～9.691）、FIGO 分期为晚期（II~III）（HR=2.683，95%CI 为 1.590～4.528）是影响Ⅱ～Ⅲ期 BOT 患者 DFS 时间的独立危险因素（P 均<0.05）。
结论 BOT 患者有良好的生存率。累及双侧卵巢、保留生育功能手术、微乳头亚型、卵巢外浸润性种植、FIGO 分期为晚期是影响患者 DFS 时间的独立危险因素。

OR_075

尼拉帕利联合安罗替尼用于尼拉帕利维持治疗中 CA125 升高的卵巢癌：一项开放、单臂、II 期临床试验

李雷[1] 吴鸣[1*] 王颖梅[2]
1.中国医学科学院北京协和医院 2.天津医科大学总医院

目的 PARPi 已在新诊断晚期 OC 1L 维持中被广泛应用，然而在临床实践中，对于仅 CA125 水平升高但维持治疗期间无影像学进展（即生化复发）的患者，如何治疗目前并没有统一的标准。NCCN 指南建议可以直至影像学复发或临床复发后开始治疗、或立即以铂为基础的化疗或最佳支持治疗。因此我们思考是否有其他的治疗方式以尽量推迟其影像学进展的时间，延长其 PARPi 维持治疗的使用时长。既往 ANNIE 研究中发现尼拉帕利联合安罗替尼治疗 PRR OC 有较好的疗效和安全性。因此本研究旨在评估尼拉帕利联合安罗替尼用于尼拉帕利维持治疗中生化复发 OC 患者的有效性和安全性。

关键入组标准：1）年龄≥18 岁；2）经组织病理学确诊的非黏液性新诊断晚期上皮性 OC，含铂化疗后达到 CR/PR 且使用尼拉帕利一线维持治疗；3）生化复发的定义：a.若化疗结束后 CA125 在正常范围，尼拉帕利维持治疗期间 CA125＞35U/ml 且时间间隔 1 周以上，并且无影像学证据证明疾病复发；b.若 CA125 在一线化疗疗程内下降>90%，化疗结束后未在正常范围，以化疗结束后的 CA125 的水平为最低点，尼拉帕利维持治疗期间 CA125 升高至最低点的 2 倍，且时间间隔 1 周以上，并且无影像学证据证明疾病复发；4）允许在一线化疗中使用贝伐珠单抗。用药方案：尼拉帕利个体化起始剂量，安罗替尼 10mg QD(D1-D14，21 天为一个周期)，尼拉帕利联合安罗替尼治疗直至疾病进展或不可耐受的毒性。主要终点：根据 RECIST v 1.1 评估的 PFS，次要终点：TFST、OS 和安全性。

结果 截止到 2025 年 8 月 1 日，共入组 22 例患者。中位年龄为 58 岁，81.8%（18 例）为 FIGO III/IV 期，3 例患者为 BRCAmut，6 例为 HRD 阳性。10 例既往接受 NACT, 20 例手术后达到 R0，所有患者化疗后达到 CR。中位随访时间为 24 个月时，总人群的 mPFS 为 10.5 个月。86.4%（19 例）的患者治疗期间经历任意级别的TEAEs，其中18.2%（4例）为≥3级TEAE，常见的TEAE包括：口腔溃疡，高血压，贫血，纳差，腹泻，疲乏等。

结论 尼拉帕利联合安罗替尼用于尼拉帕利维持治疗中 CA125 升高的卵巢癌显示出有前景的疗效，且安全性可耐受。

OR_076

Prognostic Modeling of Endometriosis-Associated Ovarian Cancer Based on Molecular Signatures: a Retrospective Study

Wang Ming[1] Yumei Wu[1*] Jianqing Xu[2]
1. Beijing Obstetrics, and Gynecology Hospital, Capital Medical University
2.Beijing Haidian District Maternal and Child Health Care Hospital

Objectives This study aimed to investigate the distribution of molecular signatures in endometriosis- associated ovarian cancer (EAOC) and to develop a prognostic model based on these molecular signatures.

Methods We retrospectively analyzed EAOC patients treated at Beijing Obstetrics and Gynecology Hospital between December 2015 and July 2023. Progression-free survival (PFS) and overall survival (OS) were compared across molecular subtype groups. Cox regression analysis identified independent recurrence risk factors in EAOC, and a nomogram was constructed using these factors.

Results The cohort included 191 patients. Pathological classification (clear cell carcinoma vs. endometrioid), advanced FIGO stage (III–IV vs. I–II), bilateral ovarian involvement, and MMRd status were identified as independent factors associated with recurrence risk (all $P < 0.05$). A nomogram incorporating these four variables demonstrated strong predictive performance, with a C-index of 0.844. The areas under the curve (AUCs) for predicting 1-, 3-, and 5-year PFS were 0.838, 0.912, and 0.898, respectively. Calibration curves showed excellent agreement between predicted and observed recurrence probabilities at 1, 3, and 5 years. Bootstrap internal validation confirmed the model's robust discriminatory power.

Conclusions Advanced FIGO stage, clear cell carcinoma histology, bilateral ovarian tumors, and MMRd molecular signatures were independent risk factors for EAOC recurrence. The molecular signature-integrated nomogram exhibited strong discrimination and calibration, offering a reliable tool for clinical decision-making in EAOC management.

OR_077

HSPE1 promotes the occurrence and development of ovarian cancer through stress granules formation

Boyi Ma Weina Kong Jia Xi Ziting Chen Hong Yang*
Department of Obstetrics and Gynecology, XiJing Hospital, Air Force Medical University

Objective Investigate the expression of HSPE1 in ovarian cancer tissues and its influence on the malignant phenotype of ovarian cancer cells.Exa mine the role of HSPE1 in stress granule formation, thereby providing substantial evidence for the involvement of stress granules in the pathogenesis of ovarian cancer and enhancing the understanding of its underlying mechanisms.

Methods Bioinformatics techniques were integrated with quantitative real-time PCR (RT-qPCR), Western Blot analysis, and immunohistochemistry (IHC) to validate the expression of HSPE1 in OC tissues and assess the prognosis of OC patients.OC cell lines with HSPE1 knockdown and overexpression were developed. Following the confirmation of knockdown and overexpression efficiency via RT-qPCR and Western Blot analysis, the impact of HSPE1 on OC cell proliferation, migration, invasion, and apoptosis was evaluated using the CCK-8 assay, colony formation assay, wound healing assay, Transwell assay, and flow cytometry. The influence of HSPE1 on the growth rate of subcutaneous xenograft tumors derived from OC cells in nude mice was investigated through a subcutaneous tumorigenesis experiment.The role of HSPE1 in stress granule formation was exa mined using immunofluorescence (IF), and its effect on the phosphorylation level of eIF2α was assessed by Western Blot analysis.Transcriptome sequencing was employed to identify downstream target genes regulated by HSPE1. RT-qPCR and Western Blot analysis were used to confirm that HSPE1 modulates the expression of NUAK2, and the effect of NUAK2 on protein translation was deter mined by Western Blot analysis.Cell lines with NUAK2 knockdown and overexpression were established to investigate the role of NUAK2 in OC cell proliferation, migration, and invasion through various cellular assays. Additionally, assessing the impact of NUAK2 on the oncogenic role mediated by HSPE1 through rescue experiments.

Results

1. Bioinformatics analysis, RT-qPCR, and Western Blot analyses revealed that HSPE1 is highly expressed in OC and correlates with poor prognosis in OC patients. IHC results further confirmed that HSPE1 expression levels in OC tissues were significantly elevated compared to normal ovarian tissues.
2. Silencing HSPE1 inhibited the proliferation, migration, and invasion of OC cells, enhanced apoptosis, reduced the growth rate of subcutaneous xenograft tumors in nude mice, and decreased tumor weight. Conversely, HSPE1 overexpression produced opposite effects.
3. HSPE1 is a component of SG, and its knockdown impairs SG assembly and decreases the phosphorylation level of eIF2α.
4. NUAK2 functions as a downstream target gene of HSPE1. Silencing NUAK2 effectively mitigates the increase in eIF2α phosphorylation and the augmented formation of SG induced by HSPE1 overexpression. HSPE1 facilitates SG assembly by mediating stress-induced translation stalling via NUAK2.
5. NUAK2 acts as a tumor-promoting factor in OC, as its overexpression counteracts the tumor growth inhibition resulting from HSPE1 knockdown.

Conclusions In OC tissues, elevated levels of HSPE1 are observed, which correlate with a poor prognosis for OC patients.HSPE1 plays a pivotal role in enhancing SG assembly and in promoting the proliferation, migration, and invasion of ovarian cancer cells.HSPE1 facilitates the phosphorylation of eIF2α through the upregulation of NUAK2, resulting in a broad inhibition of translation and subsequently modulating the assembly of stress granules SG.

OR_078

Evaluation the Clinical Response Reputation of Platinum- Based Chemotherapy in Patient-derived Organoids of Ovarian Cancer

Yawen Wu[1,2] Mingxia Ye[2] Yuanguang Meng[1,2*]
1.Chinese People's Liberation Army (PLA) Medical School
2.Department of Obstetrics and Gynecology，Seventh Medical Center of Chinese PLA General Hospital，Beijing 100700，China

Objective Ovarian cancer is the deadliest gynecological malignancy, with platinum-based chemotherapy being the standard treatment. Although most newly diagnosed high-grade serous ovarian cancer (HGSOC) patients initially respond well, approximately 15%-25% develop resistance. The actual incidence of primary platinum resistance may exceed reported rates. Currently, precision medicine tools for predicting therapeutic efficacy post-platinum resistance are lacking. Over the past decade, organoids have gained increasing prominence in cancer research and precision medicine. Therefore, evaluating the predictive capability of organoid-based drug screening for ovarian cancer treatment response is critical.

Methods Patient-derived ovarian cancer organoids (OCOs) serve as a vital tool for disease mechanism investigation, drug screening, and toxicity testing. OCOs were established from surgical specimens and malignant effusions. Histopathological validation and molecular genetic characterization confirmed that OCOs maintained the heterogeneity of their parental tumors. Clinical information was obtained through medical record review and telephone follow-up. Organoid-based drug sensitivity testing results were compared with corresponding patient clinical treatment responses, correlating OCOs chemosensitivity testing outcomes with patient clinical outcomes.

Results This study successfully generated 191 OCOs from 133 ovarian cancer patients, achieving a high success rate of 79.52%. Samples included primary lesions, metastatic sites, and malignant effusions. Paired organoid models from primary tumors and multiple metastatic sites were successfully established for 21 patients. OCOs exhibited consistent histomorphology and biomarker expression with parental tumor tissues upon pathological validation. Identical base mutations, similar genetic alteration profiles, comparable numbers of somatic mutations, and a high degree of shared mutation sites were detected in both OCOs and original tumors, demonstrating that the established OCOs highly retain the heterogeneity of the original malignancies.

In vitro drug sensitivity testing in patient-derived ovarian cancer organoids showed good concordance with clinical outcomes. Significant heterogeneity in drug responses was detected across organoids derived from distinct lesion types. Organoids originating from primary lesions demonstrated the highest sensitivity and specificity for predicting clinical response, followed by metastatic lesions, while malignant effusions showed relatively lower predictive value for chemotherapy efficacy. This suggests that when utilizing ovarian cancer organoids to predict chemotherapy response and guide treatment selection, drug sensitivity results from primary lesions should be prioritized, as they more accurately reflect the patient's actual clinical treatment efficacy.

Conclusions OCOs serve as a robust preclinical tool for clinical drug screening, predicting patient drug response, and guiding clinical treatment regimen selection.

OR_079

BRCA 基因突变类型与卵巢上皮癌的临床病理特征和预后的相关性分析

陈真云*
临沂市肿瘤医院

目的 回顾性分析 BRCA 突变的卵巢上皮癌的病理特征结合临床基线特点及预后等临床资料，探讨 BRCA 基因检测阳性上皮性卵巢癌突变类型与临床基线特点、病理特征及预后的相关性，更好的为 BRCA 基因检测阳性卵巢上皮癌患者提供更加精准个体化的诊断和治疗，从而提高患者的生存率。
方法 收集 2017 年 2 月至 2024 年 10 月来自临沂市肿瘤医院符合入组的 62 例 BRCA 基因检测阳性上皮性卵巢癌患者的临床及病理特征，包括患者的年龄、FIGO 分期、CA125 水平、治疗方式（PDS 或 IDS）、KELIM 评分、病理类型、病理免疫组化和 PFS 情况。经 SPSS 25.0 统计处理， P<0.05 提示存在统计学差异。
结果 62 例 BRCA 阳性上皮性卵巢癌患者，50 例患者为 BRCA1 突变，12 例患者为 BRCA2 突变。BRCA1 突变组和 BRCA2 突变组 2 组患者的年龄、临床分期、手术方式、病理类型等临床基线资料进行比较，2 组比较无统计学意义（P＞0.05）。BRCA1 突变组突变类型为移码突变 29 例，错义突变 9 例，无义突变 8 例，剪切突变 4 例；BRCA2 突变组突变类型为移码突变 9 例，错义突变 2 例，无义突变 1 例。P53 突变状态与患者的临床、病理特征进行比较，无统计学意义（P＞0.05）。临床分期、手术时机选择、ER 状态、KELIM 评分、PARP 抑制剂维持治疗选择与 PFS 进行单因素分析，有统计学意义（P＜0.05）。将有统计学意义的临床资料纳入多因素分析发现临床分期和是否选择 PARP 抑制剂维持治疗与 PFS 的相关性差异具有统计学意义（P＜0.05）。BRCA 分类与 PFS 相关性比较无统计学意义（P＞0.05）；是否选择 PARP 抑制剂维持、KELIM 评分、不同临床分期与 PFS 相关性比较，差异具有显著的统计学意义（P <0.05）。
结论 卵巢上皮癌的 BRCA 基因致病性突变以 BRCA1 突变占比更高，突变类型占比按照由高到低分别为移码突变、错义突变、无义突变和剪切突变。临床分期早，手术选择 PDS、KELIM 评分＞1、ER 阳性和选择 PARP 抑制剂维持治疗可以延长患者生存期（单因素分析）；多因素分析显示临床分期早和选择 PARP 抑制剂维持治疗可以显著延长患者的 PFS。BRCA1 和 BRCA2 两组患者的无进展生存期基本一致。

OR_080

以 E-选择素为靶点的自组装聚合物胶束对卵巢癌细胞生长的靶向抑制作用

杨博文 郗明蓉*
四川大学华西第二医院

研究目的 本研究旨在构建一种靶向卵巢癌的药物递送系统，利用对 E 选择素（E-selectin）具有高亲和力的 E 选择素结合肽（E-selectin-binding peptide，ESBP），主动识别肿瘤微环境中高度表达的细胞黏附分子 E 选择素，从而以 E 选择素为靶点治疗上皮性卵巢癌、抑制卵巢癌的肿瘤生长。并研究导向 E 选择素的自组装聚合物胶束纳米粒子理化特性、对卵巢癌的靶向性及作用机制。
研究方法 通过患者组织标本检测及相关数据分析，寻找相关蛋白在肿瘤组织的表达数据，依此探讨利用 E 选择素导向药物治疗卵巢癌的可行性。以牛血清白蛋白（bovine serum albumin，BSA）为基础，构建聚合型纳米粒，制备了载紫杉醇（paclitaxel，PTX）牛血清白蛋白胶束纳米粒（BSANPs-PTX）和E选择素结合肽（ESBP）修饰的载紫杉醇牛血清白蛋白胶束纳米粒（ESBP-BSANPs-PTX）等。对

ESBP-BSANPs-PTX 进行理化性质表征，检测药物紫杉醇含量，胶束纳米粒的粒径、多分散系数（PDI）、Zeta电位、包封率等。进行该自组装聚合物胶束对卵巢癌治疗的体外实验，通过药物摄取实验和 MTT 法评价与对比 ESBP-BSANPs-PTX 及紫杉醇原药、紫杉醇牛血清白蛋白胶束纳米粒（BSANPs-PTX）在 A2780/CP70、HUVEC、RAW264.7 和 ID8 细胞中的靶向性与细胞毒性，明确 ESBP-BSANPs-PTX 的优势；进一步通过细胞凋亡分析、细胞划痕实验及免疫组化等手段探讨其作用机制。使用小鼠上皮性卵巢癌细胞系 ID8 和 C57BL/6 小鼠成果构建小鼠卵巢癌模型。使用荧光探针 FITC 进行小鼠活体体内示踪，观察 ESBP-BSANPs 载药胶束纳米粒子的体内药物递送和靶向肿瘤的能力，并采用荷瘤小鼠模型评估其体内靶向性、药物毒性和治疗效果。

研究结果 E 选择素在卵巢癌组织中的表达高于正常卵巢组织，可作为载药胶束靶点。体外实验显示，ESBP-BSANPs 载药自组装聚合物胶束在细胞中的摄取能力显著增强。ESBP-BSANPs-PTX 在多种细胞系中的细胞毒性均高于紫杉醇原药，且能以剂量依赖方式诱导细胞凋亡。同时，其对细胞迁移的抑制作用也强于 BSANPs-PTX。体内实验进一步证实 ESBP-BSANPs 具有良好的肿瘤靶向性和显著的治疗效果，具有更强的药效，可以延长小鼠生存期，有更低的全身毒性，并且降低小鼠体内耐药相关细胞 MDSCs 的产生。

结论 ESBP-BSANPs-PTX 对卵巢癌细胞具有抑制作用。相对于紫杉醇原药，ESBP-BSANPs-PTX 对卵巢癌有更强的细胞毒性 h 和靶向性，能诱导癌细胞凋亡，并抑制癌细胞迁移，具有更强的药效。综上，ESBP-BSANPs-PTX 可显著提高紫杉醇的肿瘤靶向能力，增强其抗肿瘤活性，展示出在卵巢癌前临床研究中作为潜在治疗递送系统的应用前景。

OR_081

肿瘤细胞 AIG1 通过降低 CD8+T 细胞浸润和杀伤影响预后

陈静澜 汪辉*
浙江大学医学院附属妇产科医院

研究目的

1.探究 AIG1 与癌症患者预后的关系及在癌肿中的表达情况。
2.探究肿瘤细胞表达 AIG1 对肿瘤免疫微环境的影响。

材料与方法

1.数据分析：TCGA mRNA 数据进行 AIG1 泛癌预后分析以及免疫评分。单细胞数据集分析 AIG1 表达细胞群。
2. RT - qPCR：对细胞中的 mRNA 进行 RT-qPCR 验证，检测目的基因相对表达量。
3.免疫印迹实验：western blot 实验验证目的蛋白的表达水平。
4.细胞增殖及克隆形成实验： 检测 AIG1 对肿瘤细胞生长的影响。
5.小鼠模型建立：TC-1 NC/KO Aig1 细胞进行皮下成瘤。
6.T 细胞杀伤实验：小鼠 OT-I 细胞和 TC-1 NC/KOAig1 细胞共培养后吸取细胞进行后续功能性实验。
7.流式细胞学实验：对小鼠肿瘤消化后的单细胞悬液及 T 细胞杀伤实验中的细胞悬液进行 T 细胞杀伤活性检测。
8.免疫组化：患者来源和小鼠肿瘤来源的石蜡切片进行免疫组化染色。

结果 根据 TCGA AIG1 mRNA 表达水平的中位值及最佳值进行泛癌 KM 生存分析，结果提示 AIG1 显著影响 CESC、OV、UCEC、HNSC、KICH、LUAD 等患者预后，AIG1 高表达组 OS、PFS 明显差于 AIG1 低表达组。进一步对 TCGA 数据用 cibersort、xcell、timer 等算法计算肿瘤免疫细胞丰度，发现 AIG1 表达水平显著影响免疫细胞浸润，主要表现为 AIG1 高表达组免疫浸润差，尤其是 CD8+T 细胞浸润减少。GSE 单细胞数据分析发现恶性肿瘤细胞高表达 AIG1， 我们自己的单细胞数据中癌细胞为主要表达 AIG1 的细胞群。病人来源的石蜡切片免疫组化显示癌细胞高表达 AIG1。对 CaskiNC/SH AIG1、HeLa NC/OE AIG1 细胞系进行细胞增殖及克隆形成实验发现 AIG1 表达高低不影响肿瘤细胞增殖。构

建 TC-1 NC 和 KOAig1 细胞系，对 BALB/c-nude 进行单侧皮下成瘤，结果显示在免疫缺陷鼠上 Aig1 表达水平不影响肿瘤生长。而 C57BL/6J 皮下荷瘤实验结果显示 Aig1 敲除后显著抑制肿瘤增长。全能鼠皮下瘤消化成单细胞后进行流式检测发现 Aig1 敲除后具有杀伤作用的 GZMB+CD8+T 细胞比例增多。小鼠石蜡切片免疫组化染色发现 Aig1 敲除后 CD8+T 浸润显著增多。T 细胞杀伤实验的结果同样显示 Aig1KO 后 GZMB+CD8+T 细胞及 Perforin+CD8+ T 细胞比例显著增多。

结论

1.肿瘤细胞高表达 AIG1 的患者预后更差，OS 和 PFS 显著缩短。

2.肿瘤细胞高表达 AIG1 促使肿瘤免疫微环境内的 CD8+T 细胞浸润减少以及杀伤功能减弱。

OR_082

郭氏缝合法与改良 Sturmdorf 缝合法在子宫颈冷刀锥切术中应用效果的对比分析

杜欣颖 郭瑞霞*
郑州大学第一附属医院

目的 为验证郭氏缝合法的临床效果，比较郭氏缝合法和改良 Sturmdorf 缝合法应用于 CKC 的效果，以期为郭氏缝合法的可行性及安全性提供科学严谨的证据支持。

材料与方法 收集 2019 年 1 月至 2024 年 1 月在郑州大学第一附属医院妇科采用 CKC 治疗的 2174 例子宫颈 HSIL 患者的临床资料。根据缝合方式，将其分为郭氏缝合组（557 例）及改良 Sturmdorf 组（1617 例）。对比分析两组的一般人口学资料及临床特征、围术期指标、手术并发症发生情况、手术疗效结果及妊娠结局。

结果

1.相较于改良 Sturmdorf 组，郭氏缝合组的手术时间更短（27.9±8.5 min vs 34.7±12.8 min，P＜0.05），术中出血量更少[5.0 (5.0，10.0) mL vs 10.0 (5.0，10.0) mL，P＜0.05]，术后住院时间更短[2.0 (2.0，3.0) d vs 3.0 (2.0，4.0) d，P＜0.05]。

2.郭氏缝合组的术后并发症发生率显著低于改良 Sturmdorf 组（1.1% vs 4.6%，P＜0.05）。相较于改良 Sturmdorf 组，郭氏缝合组的术后出血率更低（0.5% vs 3.0%，P＜0.05），尤其是术后晚期出血率更低（0.5% vs 2.8%，P＜0.05），且术后出现子宫颈狭窄（或粘连）的概率更低（0.2% vs 1.2%，P＜0.05）。

3.相较于改良 Sturmdorf 组，郭氏缝合组的手术切缘阳性率（0.7% vs 1.6%）、病变残留或复发率（0.5% vs 1.0%）和行二次锥切术或子宫切除术的概率（0.5% vs 1.1%）更低，差异无统计学意义（P＞0.05）。郭氏缝合组与改良 Sturmdorf 组在术后 6 个月内首次复查 HPV 转阴率（89.6% vs 90.0%）和术后 HR-HPV 持续感染率（8.4% vs 7.2%）方面的差异无统计学意义（P＞0.05）。

4.相较于改良 Sturmdorf 组，郭氏缝合组术后的早产率（5.3% vs 9.9%）与自然流产率（7.3% vs 10.1%）更低，差异无统计学意义（P＞0.05）。郭氏缝合组和改良 Sturmdorf 组的妊娠率、活产率及未足月胎膜早破率分别为 75.0%（51/68）vs 73.5%（150/204）、69.1%（38/55）vs 69.8%（111/159）、7.3%（4/55）vs 5.7%（9/159），差异无统计学意义（P＞0.05）。

结论 郭氏缝合法能明显缩短手术时间，减少术中失血量和术后并发症的发生，且术后恢复更快，妊娠结局良好，值得临床推广。

OR_083

基于多组学的双硫死亡调节子泛癌图谱研究

陈鑫 王梅*
上海市浦东新区人民医院

目的 双硫死亡（Disulfidptosis）是一种新近发现的程序性细胞死亡形式，其诱因源于葡萄糖缺乏条件下胱氨酸摄取过量引发的二硫键应激，导致肌动蛋白骨架异常交联和细胞收缩性死亡。尽管该机制在代谢依赖型癌细胞中表现出重要的生物学意义，但其在不同肿瘤类型中的分子特征、调控网络及临床潜力尚缺乏系统性研究。本研究旨在基于泛癌多组学数据，全面解析双硫死亡调节子的分子异质性及其对预后与治疗的影响。
方法 本研究基于 TCGA 数据库中 33 种肿瘤共 10845 例样本，整合突变、拷贝数变异（CNV）、转录组、甲基化、miRNA、lncRNA 与转录因子等多组学数据，系统分析 10 个双硫死亡关键调节子的表达模式及调控机制。通过无监督聚类与共表达分析构建正向评分、负向评分与双硫死亡活性评分，进一步探讨其与患者生存、肿瘤通路活性、免疫微环境、药物敏感性和免疫治疗反应之间的关联。
结果 双硫死亡调节子在不同肿瘤中表现出明显的基因变异与表达异质性。基于表达特征构建的评分系统可有效区分肿瘤亚型，其中双硫死亡活性评分较高的患者在多种肿瘤中（如肾透明细胞癌、间皮瘤、胰腺癌）表现出更佳的总生存期（OS）。功能富集分析显示，双硫死亡水平与氧化磷酸化、TCA 循环、脂肪酸代谢等代谢途径正相关，且与 EMT、TGF-β、KRAS 通路等多种肿瘤进展相关信号呈负相关。此外，双硫死亡评分普遍与免疫浸润水平、免疫检查点分子表达及免疫亚型呈负相关，提示其可能介导免疫逃逸。药物敏感性分析显示，高双硫死亡活性与多种靶向药物（特别是 PI3K/mTOR 通路抑制剂）敏感性增强相关。在两个免疫治疗队列中，双硫死亡评分亦展现出一定的生存预测潜力。
结论 本研究首次在泛癌背景下多组学解析了双硫死亡调节子的表达谱、上游调控、临床预后价值及治疗相关性，揭示其在肿瘤代谢重编程、免疫微环境重塑及靶向治疗中的潜在作用。双硫死亡评分有望作为多癌种的预后标志物与治疗响应预测工具，为肿瘤精准治疗提供新思路和理论依据。

OR_084

辅助生殖技术在交界性肿瘤术后患者应用的有效性及安全性——一项回顾性队列研究

薛璐琪[1,2] 马黔红[1,2*]
1.四川大学华西第二医院妇产科 生殖医学科
2.四川大学华西第二医院妇产科 出生缺陷与相关妇儿疾病教育部重点实验室

目的 探讨卵巢交界性肿瘤（Borderline Ovarian Tumor，BOT）患者接受保留生育功能手术（Fertility-Sparing Surgery，FSS）后行生育力保存/辅助助孕治疗后的生育结局及肿瘤复发情况，进而为辅助生殖技术在 FSS 术后 BOT 患者中应用的有效性及安全性提供数据支持。
材料与方法 回顾性分析 2014 年 6 月～2024 年 6 月于四川大学华西第二医院生殖医学科接受生育力保存/辅助助孕的 42 例卵巢交界性肿瘤保育术后患者的临床资料，并随访其妊娠结局及肿瘤随访情况。同时，按照年龄、体重指数、AMH、控制性卵巢刺激方案、授精方式等多个因素，从同时期无 BOT 或其他卵巢肿瘤或癌症病史的患者中匹配出 126 名作为对照组，运用各项统计学方法对收集到的数据进行分析及数据对比。
结果 在临床特征方面，BOT 患者的肿瘤平均发病年龄为 27.38 岁，初次接受控制性超促排卵治疗的平均年龄为 31.05 岁，其中 80.95%为肿瘤 I 期，平均接受促排周期为 2 次；其中微刺激方案为主要采用的控制性超促排方案，占 50%；共累计分娩 22 例，活产率 52.38%；平均分娩孕周为 38+3 周，其中 18 例足月分娩，占比 81.82%。在肿瘤复发方面，4 例患者发生了交界性肿瘤的复发，占总患者的

9.52%，通过逻辑回归分析发现，肿瘤复发与 BOT 分期相关（4 例均为 III 期患者），与病理类型、手术方式、手术到 IVF 的间隔时间、刺激周期数以及卵巢刺激过程中最高雌激素水平等因素均无显著关联。对比 BOT 组和对照组，在控制性卵巢刺激的各项数据上，如促排持续时间、促性腺激素总剂量、获卵数、可移植胚胎数、优胚数等，两组均无显著差异。并且，首次 IVF 周期后的活产率、累积活产率，以及新生儿的各项参数（孕周、出生体重、身长），两组均不相上下。

结论 生育力保存/辅助助孕治疗可以作为 BOT 患者 FSS 术后的可行选择，控制性卵巢刺激并不会显著增加这一人群的肿瘤复发风险，但肿瘤期别较晚者复发风险可能更高。但由于本研究自身的局限性，后续还需要更大规模的前瞻性研究、更长时间的随访进一步验证其安全性。

OR_085

Establishment and Chemotherapeutic Drug Testing of Patient-Derived Organoids from Gestational Trophoblastic Neoplasia

Yang Liu Yu Gu Wenze Wang Xiaowei Xue Juanjun Yang Fang Jiang Yuan Li Yang Xiang*
Peking Union Medical College Hospital, Chinese Academy of Medical Sciences & Peking Union Medical College

Background Gestational trophoblastic neoplasia (GTN) comprises a group of pregnancy-associated malignancies which can be histologically classified as invasive hydatidiform mole, gestational choriocarcinoma, placental site trophoblastic tumor (PSTT), and epithelioid trophoblastic tumor (ETT). Although GTN is usually sensitive to chemotherapy, some patients experience recurrence and resistance, requiring individualized therapy. Due to the low incidence rate, it is difficult to establish an in vitro experimental model that faithfully captures the characteristics of GTN. Patient derived organoids (PDOs) that simulate in vivo conditions and demonstrate great potential in predicting drug sensitivity have brought such an opportunity.

Method PDOs were established from surgically GTN resected tumor tissues. Histological and genetic characterization of PDOs and corresponding original tumor tissues were performed via IHC and whole exome sequencing analysis. We also conducted drug sensitivity tests and organoid cell viability assays using PDO cultures with five chemotherapeutic drugs and two combinations of chemotherapy drugs.

Results Organoids were successfully established from tumor tissues of 11 GTN patients, with a success rate of 81.8% (9/11 cases). These organoids demonstrated stable cryopreservation, recovery, passaging, and expansion capabilities. Hematoxylin-eosin (H&E) staining and immunohistochemical analyses confirmed that the organoids maintained morphological characteristics and expression patterns of human placental lactogen (HPL), CD146, P63, and human chorionic gonadotropin (hCG) consistent with the original tumor tissues. Whole-exome sequencing revealed high concordance in single nucleotide polymorphisms between the organoids and primary tumors. Subcutaneous xenograft models generated from three organoid lines failed to establish stable tumor growth. Drug sensitivity testing with single-agent and combination regimens (methotrexate, actinomycin D, vincristine, 5-fluorouracil, and etoposide) demonstrated differential responses among organoids of different pathological subtypes to identical treatment protocols. Notably, organoids derived from different patients with the same pathological type exhibited individualized drug response variations.

Conclusions In conclusion, a patient-derived GTN tumor organoid biobank was successfully established for the first time, encompassing three pathological subtypes: PSTT, ETT, and choriocarcinoma. These organoids faithfully recapitulated the histopathological and genomic characteristics of primary tumors. Drug sensitivity testing using this organoid platform demonstrated significant heterogeneity in therapeutic responses among different pathological subtypes and individual patients.

OR_086

Optimizing Chemo-Immunotherapy for Cervical Cancer by Harnessing Time-Dependent Immune Modulation in the Tumor Microenvironment

Miaochun Xu Huaiwu Lu*
Sun Yat-Sen Memorial Hospital of Sun Yat-Sen University

Objectives The integration of chemotherapy and immune checkpoint blockade (ICB) has emerged as a promising strategy for cervical cancer treatment. However, the lack of consensus on the optimal scheduling of these therapies limits their synergistic potential. This study investigates how cisplatin dynamically remodels the tumor immune microenvironment (TIME) and defines the most effective therapeutic window for combination therapy to maximize anti-tumor responses.
Methods Using in-vivo models and patient-derived organoids (PDOs) co-cultured with immune cells, we characterized the temporal evolution of the TIME after cisplatin exposure. Transcriptomic profiling of tumors at sequential post-treatment time points elucidated immune pathway activation. Immune checkpoint dynamics, lymphocyte infiltration, and functional immune phenotypes were assessed in vivo and in vitro to correlate timing with therapeutic outcomes.
Results Our results demonstrated that cisplatin induces dynamic, time-dependent changes in the tumor immune microenvironment. In vivo studies revealed fluctuating expression of immune checkpoints following treatment, with the greatest anti-tumor efficacy achieved when ICB was administered during peak checkpoint activation. Ex vivo experiments using PDO-PBMC co-cultures further supported the importance of timing, showing that the cytotoxic synergy between cisplatin and ICB was highly dependent on treatment scheduling. Transcriptomic analysis uncovered sequential activation of immune-related pathways post-chemotherapy, mirroring the observed phenotypic changes in immune infiltration. Notably, NK and CD8+ T cell recruitment reached its peak on day 7, coinciding with enhanced effector function and tumor cell killing. Additionally, pathway enrichment analysis highlighted phased alterations in DNA damage repair and immune response mechanisms, further underscoring the temporal regulation of cisplatin-induced immunomodulation.
Conclusions Our work demonstrates that cisplatin reprograms the TIME in a temporally orchestrated manner, with distinct windows of enhanced immune vulnerability. Delivering ICB during phases of maximal immune activation optimizes combination efficacy. These data provide a rationale for chronologically tailored regimens to improve clinical responses in cervical cancer.

OR_087

Integrated Spatial Multi-omics Delineates Fatty Acid Degradation Fuels Malignant Evolution at the Tumor Periphery in Cervical Squamous Cell Carcinoma

Shitong Lin Peng Wu*
Huazhong University of Science and Technology Tongji Medical College Affiliated Union Hospital

Background Cervical squamous cell carcinoma (CSCC) exhibits profound spatial heterogeneity in evolutionary trajectories across distinct tumor foci.
Methods Through integrated single-cell RNA sequencing (scRNA-seq), spatial transcriptomics (ST), and spatial metabolomics (SM) of tumor specimens, combined with in vitro and in vivo experiments，we mapped the molecular and metabolic topography of CSCC progression.
Results We revealed that peripheral tumor regions (Layer 3) exhibited enhanced proliferative capacity and aggressive traits characterized by elevated epithelial-to-mesenchymal transition (EMT) activity

compared to core regions (Layer 1), which correlated with advanced evolutionary states. A Layer 3-specific gene signature predicted poorer overall survival in both the Cancer Genome Atlas (TCGA) and Tongji Hospital (TJH) cohorts. Spatial multi-omics uncovered progressive metabolic reprogramming along the core-to-periphery axis, with fatty acid degradation pathway activation emerging as a hallmark of Layer 3 aggression. Genetic knockdown (HADH, HADHA and ECSH1) and pharmacological inhibition (mitotane and perhexiline) of fatty acid degradation attenuated malignant phenotypes of Layer 3, including proliferation and EMT-driven invasion, in vitro and in CSCC-derived organoids.
Conclusions In summary, our work establishes spatially resolved activation of the fatty acid degradation as a key evolutionary engine fueling the malignant phenotypes of peripheral tumor niches, and nominates stratification-targeted metabolic disruption as a spatially promising therapeutic strategy.

OR_088

Timing, Surgical Approach, and Uterine Manipulator Use in Total Hysterectomy After loop electrosurgical excision procedure (LEEP): Implications for Perioperative Risks in high-grade squamous intraepithelial lesion (HSIL) Patients

Xiaoyu Hou[1,2] Junyang Li[1] Bingjie Mei[1] Jiao Pei[1,3] Mingfeng Feng[1] Hong Liu[1] Guonan Zhang[1] Dengfeng Wang[1*]
1.Sichuan Cancer Hospital & Institute, Sichuan Cancer Center, Affiliated Cancer Hospital of University of Electronic Science and Technology of China
2.Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology
3.West China Fourth Hospital, Sichuan University

Background This study aimed to analyze perioperative information in patients with high-grade squamous intraepithelial lesion (HSIL) undergoing total hysterectomy (TH), offering insights into optimal surgery timing, selecting the operation path, and enhancing surgical operation details.
Materials and Methods Data were collected from patients with HSIL who underwent TH at the Gynecological Oncology Center of Sichuan Cancer Hospital in past 7 years. Based on the interval between the loop electrosurgical excision procedure (LEEP) and TH and the surgical path of TH, the patients were divided into four groups. Patients who underwent laparoscopic hysterectomy (LH) due to benign gynecological diseases were included in the control group.
Results A total of 532 patients with HSIL were included: 111 patients underwent LH within 3 days after LEEP (group 1); 183 patients underwent abdominal hysterectomy (AH) within 3 days after LEEP (group 2); 125 patients underwent LH 4 weeks after LEEP (group 3); 113 patients underwent AH 4 weeks after LEEP (group 4); and control group: 294 patients. Compared to groups 3 and 4, patients in groups 1 and 2 had more increased postoperative high fever rates (14.3% vs. 3.8%). Moreover, therapeutic antibiotic usage was more frequent (16.0% vs. 5.5%). the average postoperative hospitalization days (4 [4, 5] vs. 4 [3, 5] days) were longer, and the incidence of postoperative complications (12.2% vs. 3.8%) was higher, ($P < 0.05$). Compared to the control group, group 1 had higher rates of postoperative high fever (6.5% vs. 17.1%), therapeutic antibiotic use (4.1% vs. 16.2%), and complications (4.1% vs. 12.6%) ($P < 0.025$); and group 3 showed no significant differences in these parameters ($P > 0.025$). Multivariate analysis identified postoperative fever ≥38.5°C (adjusted OR = 3.783, $P = 0.027$) and antibiotic use (adjusted OR = 2.465, $P < 0.001$) as independent risk factors for postoperative complications.
Conclusions To reduce perioperative risks, especially infection, scheduling TH 4 weeks after LEEP is recommended, with a preference for the laparoscopic approach. When TH is required within 3 days after LEEP, use laparoscopy cautiously, optimizing measures like improved vaginal and cervical disinfection while avoiding a uterine manipulator.

OR_089

Development and validation of prediction model for adnexal metastases in early-stage cervical adenocarcinoma: A multicenter study

Penglin Liu[1] Changzhen Huang[1] Yanhui Lou[2] Hongwei Zhao[3] Feng Geng[4] Haifeng Qiu[5] Beihua Kong[1] Kun Song[1*]
1.Qilu Hospital of Shandong University2.The Affiliated Hospital of Qingdao University
3.Shanxi Provincial Cancer Hospital
4.Shandong Provincial Hospital Affiliated to Shandong First Medical University
5.The First Affiliated Hospital of Zhengzhou University

Background Cervical adenocarcinoma, as a relatively rare histological subtype of cervical cancer, accounts for approximately 20% of all cervical malignancies. Accurately assessing risk factors associated with adnexal metastasis in the treatment of cervical adenocarcinoma remains a critical clinical challenge. Notably, current international clinical guidelines primarily provide ovarian preservation criteria for cervical squamous cell carcinoma, while offering limited and inconsistent recommendations for cervical adenocarcinoma. To address this gap, we conducted a large-scale, multicenter study to develop a machine learning (ML)-based prediction model for precise risk assessment of adnexal metastases among patients with early-stage cervical adenocarcinoma, guiding the individualized decisions regarding ovarian preservation.

Methods Cervical adenocarcinoma patients with clinically apparent normal adnexa were enrolled from five major university-teaching hospitals in China between 2011 and 2023. The primary outcome was pathologically confirmed adnexal metastases. Univariate and multivariate logistic regression (LR) analyses were used to identify the independent risk variable for adnexal metastases. After stepwise variable selection, we developed six prediction models based on different ML algorithms (LR, support vector machine, random forest, AdaBoost, naïve Bayes, and decision tree) for predicting the risk of adnexal metastases, with 5-fold cross-validation. The performance of each model was assessed by the area under the receiver operating characteristic curve (AUC), specificity, sensitivity, negative predictive value (NPV), positive predictive value. The best-performing model was subsequently visualized as a nomogram and evaluated by discrimination, calibration and net benefits.

Results This multicenter study included 1,089 cervical adenocarcinoma patients, among whom 51 (4.7%) had adnexal metastases. Univariate logistic analysis revealed that HPV infection status (OR=0.28, 95%CI: 0.16–0.50; P＜0.001), CA-125 level (OR=4.56, 95% CI: 2.55–8.15, P＜0.001), tumor size (OR=4.99, 95% CI: 2.82–8.84; P ＜ 0.001), depth of cervical stromal invasion (OR=5.96, 95% CI: 2.51–14.15; P＜0.001), uterine body involvement (OR=10.21, 95% CI: 5.64–18.49; P ＜ 0.001), p16 expression (OR=0.22, 95% CI: 0.12–0.39; P ＜ 0.001) and Ki-67staining (OR=0.42, 95% CI: 0.22–0.79; P=0.007) were significantly associated with adnexal metastases. These above-mentioned seven candidate variables were subsequently evaluated using multivariate backward stepwise regression analysis to identify the optimal combination of variables based on the AIC. According to the stepwise regression results, the prediction model which incorporated preoperative CA-125 levels, tumor size, depth of cervical stromal invasion, uterine body involvement, and p16 expression yielded the lowest AIC value of 331.48.

The selected five variables (CA-125 levels, tumor size, depth of cervical stromal invasion, uterine body involvement, and p16 expression) were input into six ML-based algorithms with a 10-fold cross validation. The AUC of all models ranged from 0.71 to 0.85. Among the models, the LR model demonstrated the best predictive performance, with AUC of 0.846, sensitivity of 0.702 and specificity of 0.785. Meanwhile, the LR model demonstrated superior discriminatory performance than the other five ML-based models (AUC 0.846 vs 0.715–0.844).

Based on the LR model, we developed a clinically applicable nomogram, which displayed good discrimination ability, calibration ability and clinical benefits. stratified patients into high-risk (predicted probability of adnexal metastasis >15%) and low-risk (<5%) groups, using Youden index as the optimal cutoff.

Conclusions Based on the largest cervical adenocarcinoma cohort to date, we established and validated a ML-based nomogram that could accurately assess adnexal metastases risk among early-stage cervical adenocarcinoma patients, offering a practical tool to tailor individualized surgical decision-making regarding ovarian preservation.

OR_090

Correlation Study and Prognostic Analysis of Tumor-Free Distance (TFD) Measured by MRI Before Cervical Cancer Treatment and Postoperative Pathological Factors

Die Fan Xiaoxia Jiang Yujie Hao Zheng Li*
Department of Gynecologic Oncology, Yunnan Cancer Hospital

Objective (s) Cervical cancer remains the fourth most common malignancy globally in terms of both incidence and mortality. Early-stage cervical cancer is primarily managed through surgical intervention. In recent years, numerous clinical studies have aimed to minimize the extent of surgery for early-stage patients in order to reduce the incidence of postoperative complications. However, the assessment of pathological high-risk factors still largely depends on conization. Determining whether preoperative imaging can non-invasively identify low-risk patients who are suitable for conservative surgery has become a key focus for clinicians.This study investigates the correlation between the tumour-free distance (TFD) measured by magnetic resonance imaging (MRI) prior to cervical cancer treatment and postoperative high-risk pathological factors, including lymph node metastasis (LNM),as well as moderate-risk factors such as lymphovascular space invasion (LVSI) and deep stromal invasion (DSI). Additionally, it evaluates the prognostic predictive value of TFD for patients. The ultimate goal is to optimize the assessment process for low-risk early-stage cervical cancer patients and minimize the cumulative adverse effects associated with various treatment modalities.

Methods

1、The clinical data of patients who underwent radical surgery for early-stage cervical cancer and were pathologically confirmed at our hospital between January 1,2017, and December 31, 2017, were retrospectively reviewed. Only patients with complete clinical pathological records, preoperative MRI images, and follow-up
information were included. Patients were further screened according to the predefined inclusion and exclusion criteria to determine the final cohort for analysis.

2、Two experienced radiologists independently assessed and measured the TFD in the preoperative MRI scans of patients scheduled for surgery. Additionally, a senior pathologist with extensive experience reviewed the postoperative pathological findings, including high-risk factors (LNM) and intermediate-risk factors (LVSI and
DSI), for all surgical patients.

3、Statistical analysis was performed using R software (version 4.4.0). The preoperative imaging-based TFD values of the included patients and their associated influencing factors were analyzed. Spearman's rank correlation test was used to evaluate the correlation between TFD and postoperative high-risk pathological factors, including deep stromal invasion (DSI), lymphovascular space invasion (LVSI), and lymph node metastasis (LNM). The optimal cut-off value of TFD was determined using receiver operating characteristic (ROC) curve analysis. Univariate and multivariate logistic regression analyses were conducted to assess the association
between TFD and other clinical-pathological factors. Univariate and multivariate Cox proportional hazards models were employed to analyze the impact of clinical-pathological factors and TFD on recurrence and survival time, with hazard ratios (HRs) and 95% confidence intervals (95% CIs) reported for these associations.Survival curves were generated using the Kaplan-Meier method, and a risk prediction model for cervical cancer survival was constructed and validated using R software.

Results

1、A total of 264 cervical cancer patients who met the inclusion and exclusion criteria were included in the study, with a median age of 48 years. The mean TFD value was 3.56 ± 2.4 mm (range: 0 - 14 mm). TFD was significantly correlated with patient age, FIGO stage, serum SCC antigen level, tumor size, depth of stromal invasion, lymphovascular space invasion (LVSI), and lymph node metastasis (P <70.05), but not with BMI, histological type, degree of differentiation, or preoperative serum CA125 level (P > 0.05). Notably, TFD exhibited negative correlations with depth of stromal invasion (r = -0.533), LVSI (r = -0.341), and lymph node metastasis
(r = -0.376; all P < 0.001).

2、Univariate and multivariate Logistic regression analyses revealed that dichotomized TFD and LVSI were independent predictors of lymph node metastasis (LNM). While TFD was associated with DSI and LVSI, it did not serve as an independent predictor. LNM and DSI were identified as independent predictors of LVSI. Furthermore, FIGO stages IB2-IIA2, LVSI, and LNM retained their independent associations with DSI.

3、Univariate COX regression analysis of factors influencing the PFS in cervical cancer patients revealed that age, FIGO stage, DSI, TFD, endocervical invasion, and LNM were significantly associated with disease recurrence (P < 0.05). Multivariate COX regression analysis further demonstrated that TFD (HR = 6.39, 95% CI = 2.17 -18.83, P < 0.001), serum tumor marker CA125 (HR = 2.45, 95% CI = 1.20 - 5.36, P =0.014), and LNM (HR = 2.59, 95% CI = 1.22 - 5.51, P = 0.013) were independent predictors of recurrence in cervical cancer patients.

4、Univariate COX regression analysis revealed that age, serum tumor marker CA125, DSI, TFD, endocervical invasion, and LNM were significantly associated with overall survival in cervical cancer patients (P < 0.05). Multivariate COX regression analysis further demonstrated that TFD (HR = 10.52, 95% CI = 3.00 -24.91, P < 0.001) and LNM (HR = 3.3, 95% CI = 1.37 - 7.94, P < 0.001) were independent predictors of overall survival in cervical cancer patients.

Conclusion(s)

1、Preoperative TFD measured by MRI as a continuous variable showed a negative correlation with high-risk pathological factors in postoperative specimens,including deep stromal invasion (DSI), lymphovascular space invasion (LVSI), and lymph node metastasis (LNM). Specifically, a smaller TFD value was associated with
8an increased risk of deep myometrial invasion, positive lymphovascular space invasion, and lymph node metastasis.

2、TFD is an independent predictor of lymph node involvement but only serves as a non-independent influencing factor for DSI and LVSI.

3、TFD is associated with PFS and OS in patients with early-stage cervical cancer, and 2.5 mm is the optimal threshold of TFD for predicting PFS and OS. TFD≤ 2.5 mm and lymph node metastasis are independent risk factors affectingdisease-free survival and overall survival in patients with cervical cancer.

OR_091

Aneuploidy information and Its Association with Gene Expression Profiles and Recurrence Risks in Endometrial Carcinoma

Ming Du Yang Xiang*
Peking Union Medical College Hospital

Introduction Although molecular classification partially helped to tailor treatment regimen, endometrial cancer (EC) still had the highest incidence rate and mortality rate among genital malignancies in global females. More effective biomarkers need to be explored as alternative or supplementary tools for the TCGA molecular category, which might help for re-stratifying the prognosis, identifying subpopulation

with recurrence risks, and designing individualized regimen for relapsed patients. Aneuploid was one prominent characteristic of solid cancers, which was though to mediate cancer evolution and progression. However, there was limited progress of aneuploid in EC despite of the potential value of aneuploid in recurrence prediction and prognostic stratification. For better exploring the ploidy status and its potential role in EC survival stratification, recurrence prediction and cancer progression, herein, this research combined cytogenetics with molecular genetics methods, and analyzed aneuploid information of EC and its relationship with gene expression profiles.
Method The ploidy information was first comprehensively analyzed based on karyotype, ploidy value and arm-level chromosome alterations from 529 EC cases via cBioPortal online database and 18 EC cases from Peking Union Medical College Hospital (PUMCH). Online cases and PUMCH cases were separated into subgroup with high aneuploid score (High AS) and subgroup with low aneuploid score (Low AS) according to aneuploid score. Clinicopathological features, overall survival (OS), progression-free survival (PFS), disease-free survival (DFS), disease-specific survival (DSS), recurrence-free survival (RFS) was compared between two groups. The differentially expressed genes (DEGs) between two groups were identified and function analysis of DEGs was performed.
Results This study comprehensively analyzed the ploidy value, aneuploid score and karyotype landscape of endometrial cancer. More than 50% of EC cases were aneuploid, with recurrent specific chromosome changes and relatively high aneuploid score. High aneuploid score indicated worse OS, PFS, DFS and DSS in whole EC cases, with additional prognostic value of OS within MSI subtype and PFS within CN-L subtype. Moreover, high aneuploid score indicated higher recurrence rate and shorter RFS according to online analysis. Function analysis showed that genes upregulated in High AS group were related to ion transport and collagen function. Immune-related analysis showed that genes related to T cell and T cell cytotoxic function were down-regulated and NK cell biomarker was up-regulated in High AS group. The immune cell infiltration analysis showed that the fraction of CD8+ T cell, activated CD4+ T cell memory, Treg cell was lower in High AS than Low AS group.
Conclusions Aneuploid was a widespread feature in endometrial cancer, which indicated poor survival and recurrence. Moreover, aneuploid was a prognostic factor for survival re-stratification, especially within MSI and CN-L subpopulation. Ion channels or transporters intervention might be potential ploidy-selective stress inducers for aneuploid EC therapy, and NK cell recognition might be more important for aneuploid EC patients.

OR_092

Development and validation of a novel prognostic scoring system for early-stage endometrial cancer: a Chinese multicenter study

Xiaoyu Yin Kun Song*
Qilu Hospital of Shandong University

Objective Accurate prognostic estimates for patients with early-stage endometrial carcinoma would allow patients to assess the risks and benefits of adjuvant therapy more accurately. However, existing risk stratification models are often complex and inconsistent. We aimed to develop and externally validate a simple and effective prognostic scoring system forprogression-free survival (PFS) and overall survival (OS) in early-stage endometrial carcinoma based on the nationwide endometrial cancer cohort in China.
Methods In this multicenter, retrospective study, we included 4092 patients with FIGO 2009 stage I–II endometrial carcinoma who underwent surgical treatment at multiple Chinese medical centers between 2000 and 2019. Of these, 3053 patients from seven centers were used as the training cohort to develop the Endometrial Carcinoma Prognostic Scoring System (ECPSS), which was then externally validated in an independent cohort of 1039 patients from three centers. Univariate and multivariate Cox regression analyses were applied to identify independent prognostic variables for PFS and OS. According to the regression coefficients derived from multivariate Cox regression analyses, a weighted

risk score was then assigned for each significant variable, which were used to establish the prognostic score. The model performance for predicting outcome was evaluated by calculating the Harrells' concordance index. All statistical analyses were done with R software.

Results Multivariable Cox regression analysis revealed independent parameters for predicting poor PFS and OS. For PFS, these were age (≥60-year-old, HR 2.22 [95% CI 1.58-3.11], p<0.001), histopathological type (G3, HR 2.95 [95% CI 2.03-4.29], p<0.001; non-endometrioid, HR 2.92 [95% CI 1.75-4.87], p<0.001), depth of myometrial invasion (≥1/2, HR 2.12 [95% CI 1.46-3.07], p<0.001), lymphovascular space invasion (LVSI) (positive, HR 1.89 [95% CI 1.09-3.27], p=0.022), peritoneal washing cytology (positive, HR 3.15 [95% CI 1.72-5.76], p<0.001); and for OS, age (≥60-year-old, HR 3.04 [95% CI 1.97-4.68], p<0.001), histopathological type (G3, HR 3.06 [95% CI 1.90-4.91], p<0.001; non-endometrioid, HR 3.31 [95% CI 1.80-6.08], p<0.001), depth of myometrial invasion (≥1/2, HR 2.29 [95% CI 1.45-3.62], p<0.001), LVSI (positive, HR 2.11 [95% CI 1.08-4.11], p=0.029), peritoneal washing cytology (positive, HR 3.20 [95% CI 1.45-7.09], p=0.004).

Patients gained 1 point in the PFS scoring system for each of the following conditions met, including a diagnosis age ≥ 60-year-old, depth of myometrial invasion ≥1/2 and positive LVSI. Meanwhile, patients gained 2 points scored for G3 non-endometrioid type and positive peritoneal washing cytology. The scoring rules were also applicable to the OS. Based on the score system, patients with FIGO 2009 stage I/II EC were stratified into four risk groups: low (0 points), intermediate (1–2 points), high-intermediate (3 points) and high (≥4 points).

Significant differences in both PFS and OS were observed across these groups in the training and external validation cohorts (p<0.0001). In the discovery cohort, patient distribution when stratified by ECPSS was 1610 low risk patients, 1079 intermediate risk patients, 257 high-intermediate risk patients and 107 high risk patients. The C-statistics for PFS and OS were 0.706 (95% CI: 0.659–0.753) and 0.731 (95% CI: 0.668–0.794), respectively. In the external validation cohort, the ECPSS stratified 1039 patients into low risk (580 patients), intermediate risk (334 patients), high-intermediate risk (83 patients) and high risk (42 patients) groups. The C-statistics were 0.744 (95% CI 0.670–0.818) for PFS and 0.778 (95% CI 0.674–0.882) for OS.

Conclusions Based on the independent prognostic factors (age at diagnosis, tumor histopathological type, cervical invasion, myometrial invasion, LVSI, and ascites cytology), we developed a novel prognostic scoring system for endometrial cancer. The ECPSS provides a reliable tool for predicting survival outcomes in patients with early-stage endometrial carcinoma, enabling more personalized treatment strategies. However, further prospective trials are required to confirm its predictive value.

OR_093

瑞舒伐他汀联合醋酸甲地孕酮治疗子宫内膜非典型增生单中心开放性临床研究

杨冰义[1] 陈晓军[1,2*] 程亚丽[1] 吴鹏飞[1]
1.复旦大学附属妇产科医院 2.上海市第十人民医院

目的 通过单中心前瞻性开放性临床试验，探索瑞舒伐他汀（Rosuvastatin，ROS）联合醋酸甲地孕酮（Megestrol acetate，MA）对合并血脂异常的子宫内膜非典型增生（Endometrial atypical hyperplasia，EAH）患者保留生育功能治疗的有效性和安全性。

方法 本研究于 2020 年 7 月至 2022 年 12 月在复旦大学附属妇产科医院进行，研究对象为合并血脂异常并要求保留生育功能治疗的 EAH 患者，使用 MA 联合 ROS 口服治疗。研究采取 Simon 二阶段最优化设计，估算总样本量为 36 人，总有效人数 r=14。第一阶段样本量 16 人，有效人数达到 3 后可进行第二阶段。主要研究目标为探索联合治疗方案在起始治疗后 16 周的完全缓解（Complete response，CR）率，次要研究目标包括治疗 32 周时的 CR 率、中位 CR 时间、治疗过程中血脂动态变化及安全性评估等。按照初始治疗病理为 EAH、治疗前 BMI±2kg/m2、合并血脂异常、治疗方案为 MA 收集历史病例资料作为对照，进行 1:1 匹配并进一步分析，对比联合使用 MA+ROS 组患者与单独使用 MA 者治

疗 16 周时的 CR 率。使用 Kaplan-Meier 法绘制生存曲线，并通过单因素、多因素 Cox 回归模型分析影响治疗结局的风险因素。统计分析中，显著性水平设定为 $P < 0.05$。

结果

1.本研究招募患者共 36 例，初始治疗时中位年龄 31.5 岁，平均 BMI 28.97kg/m2。治疗 16 周时，13 例患者获得 CR，CR 率 36.1%。治疗 32 周 CR 率 63.9%（23/36），治疗中位 CR 时间 28.0 周（95%CI 26.6-29.4 周）。CR 后 1 年内，17 例积极备孕患者中共 6 例妊娠，妊娠率为 35.3%（6/17），活产率 23.5%（4/17）。

2.治疗 16 周后患者脂质代谢较治疗前显著改善，总胆固醇、甘油三酯、低密度脂蛋白、载脂蛋白-B 均较治疗前降低，且具有统计学差异。

3.联合使用 MA 与 ROS 相对安全，无受试者发生横纹肌溶解、血栓或乳腺癌，未发生严重不良事件。不良反应中，夜尿增多、易疲劳、体重增加及阴道淋漓出血的发生率分别为 61.1%、41.7%、41.7%、36.1%。

4.通过病例对照研究，筛选 36 例 EAH 病例作为对照，进行 1:1 匹配并进一步分析，联合使用 MA+ROS 组对比单独使用 MA 组患者治疗 16 周时的 CR 率更高（36.3%vs11.1%，P=0.013），治疗中位完全缓解时间更短（28.0 周 vs34.1 周，Log-rank P=0.013）。多因素 Cox 回归分析显示联合使用 MA+ROS（对比 MA）（风险比 0.555, 95%置信区间 0.342-0.900; P=0.017）是影响合并血脂异常的 EAH 患者 CR 的独立预测因子。

结论 MA 联合 ROS 的用药方案，能够提高合并血脂异常的 EAH 患者保留生育功能治疗疗效。

OR_094

淋巴脉管癌栓对病理淋巴结阴性早期子宫内膜癌预后及辅助治疗选择的影响

颜子涵 任康 辛玲霞 盛云龙 王雅琦 侯晓荣* 胡克 张福泉
北京协和医院

目的 淋巴脉管间隙侵犯（LVSI）是子宫内膜癌的不良预后标志，与淋巴结转移和远处转移相关，是风险分层的依据之一。但对于接受前哨淋巴结活检或区域淋巴结清扫后确定无淋巴结转移的患者来说，LVSI 在不同危险分组中对预后及辅助治疗影响还存在争议。本研究旨在探索 LVSI 阳性对病理淋巴结阴性患者预后和辅助治疗选择的影响。

方法 本研究回顾了 2000 年 1 月 1 日至 2020 年 12 月 31 日在北京协和医院放疗科治疗的早期子宫内膜癌患者临床资料。将接受了前哨淋巴结活检或区域淋巴结清扫且无淋巴结转移的子宫内膜样腺癌患者纳入研究，根据组织学分级和肌层浸润深度分为低危组、中危组和高危组。在不同危险分组中运用 Kaplan-Meier 法和 Log-Rank 检验计算和对比 LVSI 阳性和阴性组的 OS、DFS、LRFS 及 DMFS。利用 Cox 风险比例模型进行单因素及多因素分析，探索早期子宫内膜癌的预后相关因素。采用 Landmark 法探索 LVSI 状态和不同放疗模式对近期和远期控制的影响。

结果 一共纳入 782 例 I 期子宫内膜样腺癌患者，中位随访时间 45 个月（范围：4-237 个月）。低危组、中危组和高危组分别占 47.3%，44.6%和 8.1%，各危险分组中 LVSI 阳性患者的比例分别为 22.6%，21.8%和 55.6%。在低危组和中危组中，LVSI 阳性和阴性患者的 OS（P=0.814，P=0.203）及 DFS（P=0.339，P=0.507）无明显差异，LVSI 并不是影响患者预后的因素，LVSI 阳性患者也未能从外照射中获益。在高危组中，LVSI 阳性和阴性患者的 3 年 OS 分别为 89.9%和 100%（P=0.065），3 年 DFS 分别为 80.4%和 88.0%（P=0.125），3 年 LRFS 分别为 83.6%和 92.0%（P=0.158），以及 3 年 DMFS 分别为 80.4%和 92.0%（P=0.089）。22 个月后 LVSI 阳性组的 LRFS 较阴性组明显下降（P=0.037）；类似的，20 个月后 LVSI 阳性组的 DMFS 明显低于 LVSI 阴性组（P=0.048）。对于高危组伴 LVSI 阳性的患者，外照射有降低远期局部区域复发的趋势（P=0.071）,但对总生存及远处转移无显著影响。

结论 在病理淋巴结评估阴性的早期子宫内膜癌患者中，LVSI 阳性并不是低危及中危患者的不良预后因素，阳性患者未能从外照射中获益。在高危患者中，LVSI 阳性患者的远期局部区域复发和远处转移率较高，外照射有进一步降低局部区域复发风险的趋势。

OR_095

Development of a Predictive Model Integrating Pathological and Clinical Factors for Fertility-Sparing Treatment in Patients with Atypical Endometrial Hyperplasia and Endometrial Carcinoma

Zhuoyu Zhai Yiqin Wang Aoxuan Zhu Yuwan Wu Xingchen Li Jianliu Wang*
北京大学人民医院

Background This study aims to establish a prediction model for the efficacy of fertility-sparing treatment in atypical endometrial hyperplasia(AEH) and endometrial carcinoma(EC), utilizing pre-treatment baseline clinical and pathological information.
Methods A total of 334 patients diagnosed with AEH and EC and received fertility-sparing treatment were included at Peking University People's Hospital. These patients were randomly divided into training and validation at a 2:1 ratio. We integrated 10 machine learning algorithms and explored 101 algorithm combinations using Leave-One-Out Cross-Validation (LOOCV) to develop a immunohistochemistry(IHC) scores upon a panel of markers. Univariate and multivariate cox analyses were employed to identify independent risk factors affecting the time to complete remission(CR).
Results Estrogen receptor(ER), progesterone receptor(PR), Ki67 and P53 were included in the IHC scores model. Independent risks included insulin resistance(HR=0.96, 95% CI=0.93-0.98, p=0.006), hypercholesterolemia (HR=0.36, 95% CI=0.2-0.62, p=0.002), poorly-differentiated tissue type (ECG1: HR=0.34, 95% CI=0.15-0.77, p=0.009; ECG2: HR=0.29, 95% CI=0.09-0.94, p=0.038), and high-risk IHC scores (HR=0.20, 95% CI=0.09-0.46, p=0.001).The inclusion of IHC scores significantly enhanced the predictive accuracy (area under the curve[AUC]=0.782 for Model 1, AUC=0.840 for Model 2, p=0.032).
Conclusions The present study's model effectively predicts efficacy of patients seeking fertility preservation based on a substantial cohort.

OR_096

Does Delayed Surgery Affect Survival in Recurrent Ovarian Adult-Type Granulosa Cell Tumor? A Retrospective Study from a High-Volume Medical Center

Kezhen Zhang Xuan Zong Jiaxin Yang* Jing Zheng Dongyan Cao Ming Wu Yang Xiang Jie Yang
Department of Obstetrics and Gynecology, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences & Peking Union Medical College, Beijing, China

Objective To evaluate the clinical characteristics, treatment outcomes, and the impact of delayed debulking surgery in recurrent adult-type ovarian granulosa cell tumors (AGCT).
Methods This retrospective cohort study analyzed patients diagnosed with recurrent AGCT between January 2003 and December 2023 at Peking Union Medical College Hospital (PUMCH). Kaplan–Meier analysis, along with univariate and multivariate Cox proportional hazards models, were utilized to identify factors associated with progression-free survival (PFS) following the first, second, and third recurrences, as well as overall survival (OS).

Results The study included 92 patients with recurrent AGCT, with approximately 90% of tumors initially staged as Stage I. The median follow-up time was 136.0 months (range 30.0-402.0 months). Extra-pelvic and multifocal lesions were common in recurrent cases. Most patients underwent tumor cytoreductive surgery (CRS) at each recurrence. A time interval from recurrence to CRS of ≥6 months had no adverse impact on OS (multivariate HR, 1.53; 95% CI 0.33–7.05; P = 0.582), but significantly prolonged the interval between relapses after the first (median interval not reached in the ≥6-month group vs. 40.0 months in the <6-month group; log-rank P=0.023) and second recurrence (median interval, 65.0 vs. 25.0 months; log-rank P＜0.001) . Gross residual disease after CRS was associated with poorer OS (multivariate HR, 7.44; 95% CI, 1.95-28.46; P=0.003).
Conclusions Delaying CRS by ≥6 months can prolong the time to the next recurrence without compromising OS, while gross residual disease remains an independent risk factor for poor survival.

OR_097

RPA70 和 ATR 抑制剂通过诱导治疗性 BRCAness 增强奥拉帕利在 BRCA1/2 野生型卵巢癌中的抗肿瘤效应

张亚双
江苏省妇幼保健院

卵巢癌是致死率最高的妇科恶性肿瘤，严重威胁女性健康。2018 年 FDA 首次批准奥拉帕利作为 BRCA 基因突变卵巢癌患者的一线维持治疗，这大大改善了患者的生存预后。然而，世界范围内发生 BRCA1/2 基因突变的卵巢癌患者不足 30%，这极大地限制了奥拉帕利的临床获益。探索增强奥拉帕利在 BRCA1/2 野生型卵巢癌中治疗效果的新策略是亟待解决的问题。本研究首先对 28 个具有潜在诱导治疗性 BRCAness，增强奥拉帕利敏感性的抑制剂进行筛选。接下来，对 8 个具有较强单药杀伤效应的抑制剂进行联合用药筛选。为了使体外药物筛选结果更契合临床真实用药效果，我们使用临床获取的新鲜卵巢癌组织构建了充分保留原发肿瘤组织形态与分子特征的类器官模型，进一步对 HAMNO 和 CGK733 与奥拉帕利的联合抗肿瘤作用进行评估。采用细胞活性测定、彗星实验、BLRR 实验、免疫组化、免疫荧光、细胞周期分析、动物成瘤实验等多种实验方法，系统评估其作用机制及协同效应。HAMNO 和 CGK733 分别对 RPA70 和 ATR 产生了选择性强效抑制作用，显著增强 BRCA1/2 野生型卵巢癌细胞对奥拉帕利的敏感性。RPA70 与 ATR 高表达被证实与卵巢癌患者的不良预后显著相关。从机制上讲，HAMNO 联合奥拉帕利显著抑制 RPA70 下游关键蛋白 RAD51 的表达以及其在 DNA 损伤位点的募集，抑制 DNA 同源重组修复，最终诱导细胞凋亡。CGK733 联合奥拉帕利导致 S 与 G2/M 期细胞占比下降，NHEJ 修复活性进一步下降，显著增加了肿瘤细胞的 DNA 损伤修复负担，同样诱导肿瘤细胞凋亡。本研究首次在 BRCA1/2 野生型卵巢癌模型中验证了 RPA70 与 ATR 作为 BRCAness 诱导靶点的可行性，并提出 HAMNO 和 CGK733 联合奥拉帕利的全新治疗策略，不仅为扩展奥拉帕利在 BRCA1/2 野生型卵巢癌中的应用提供了理论依据和实验支持，也为 BRCAness 靶向药物的开发和精准治疗策略的建立奠定了基础。

OR_098

上皮性卵巢癌血浆差异基因 miRNA-3187-3p 靶向 PLK2 调节细胞增殖与凋亡的机制研究

崔慧洁 哈春芳*
宁夏医科大学总医院

目的 本研究旨在构建上皮性卵巢癌（EOC）患者血浆游离 RNA（cfRNA）差异表达谱，筛选肿瘤相关关键基因，并聚焦 miR-3187-3p，阐明其通过靶向 Polo 样激酶 2（PLK2）调控 EOC 细胞增殖与凋亡的机制。

方法

1.差异 cfRNA 筛选与验证：收集 16 例 EOC 患者和 16 例良性卵巢肿瘤患者血浆，提取总 RNA，转录组测序筛选差异 miRNAs，结合随机森林和 Lasso 回归模型锁定 miR-3187-3p，qRT-PCR 验证其在 EOC 血浆及组织中显著高表达（P<0.001）。

2.机制研究：通过 miRDB、GEPIA 等数据库预测 PLK2 为 miR-3187-3p 靶基因，双荧光素酶实验验证其靶向结合。以 A2780 和 SKOV3 细胞为模型，利用 miR-3187-3p 模拟物/抑制剂及 PLK2 siRNA 调控表达，RT-qPCR、Western blot、CCK-8 和流式细胞术分析增殖与凋亡表型。

结果

1.cfRNA 标志物：共发现 80 个差异血浆 miRNAs（44↑/36↓），机器学习筛选出 miR-3187-3p，其在 EOC 血浆和组织中表达显著升高（P<0.001）。

2.miR-3187-3p/PLK2 轴功能：

生信与表达：PLK2 在 EOC 中低表达（GEPIA2，P<0.05），与预后负相关；免疫荧光及 qPCR 证实 PLK2 主要定位于胞质且表达下调（P<0.001）。

靶向验证：双荧光素酶实验显示 miR-3187-3p 直接结合 PLK2 3'UTR（野生型荧光降低，突变型无变化）。

功能实验：miR-3187-3p 过表达抑制 PLK2 表达，促进细胞增殖（CCK-8）并抑制凋亡（流式），伴随促凋亡蛋白（p53、BAX、Caspase3/9）下调及抗凋亡/增殖蛋白（Bcl-2、CDK6）上调；抑制 miR-3187-3p 则逆转上述效应。

结论 EOC 患者血浆/组织中 miR-3187-3p 高表达，通过负调控 PLK2 促进细胞增殖、抑制凋亡，驱动肿瘤进展，为 EOC 诊断及治疗提供新靶点。

OR_099

Survival of surgery for platinum-sensitive relapsed ovarian cancer: a systematic review and meta-analysis of individual participant data from randomised trials

Yulian Chen[1] Zeyun Li[1] Ping Zhang[4] Yanling Feng[2] Shiyang Jiang[1] Jianqing Zhu[4] Rong Jiang[1] Zhenyu Wu[3] Huijuan Yang[5] Jihong Liu[2] Fei Liang[6] Tingyan Shi[1] Rongyu Zang[1*]

1.Institute for Ovarian Cancer, Fudan University & Department of Gynecologic Oncology, Cancer Center, Fudan University Zhongshan Hospital

2.Department of Gynecologic Oncology, State Key Laboratory of Oncology in South China, Collaborative Innovation Centre for Cancer Medicine, Sun Yat-sen University Cancer Centre

3.Department of Biostatistics, School of Public Health, Fudan University

4.Department of Gynecologic Oncology, Cancer Hospital of the University of Chinese Academy of Sciences (Zhejiang Cancer Hospital)

5.Department of Gynecologic Oncology, Fudan University Cancer Hospital

6.Department of Biostatistics, Zhongshan Hospital Fudan University

Background Surgery for platinum-sensitive, relapsed ovarian cancer (PSROC) is widely practiced, yet previous studies have reported conflicting results regarding its impact on survival. We aimed to obtain up-to-date estimates of the overall effects of surgery on various survival metrics.

Methods In this systematic review and meta-analysis of individual participant data (IPD), we searched PubMed, MEDLINE, Embase, and Cochrane (from database inception to July 19, 2025) to identify eligible randomised trials that compared secondary cytoreduction (SCR) followed by chemotherapy (surgery) with chemotherapy alone (control) in patients with PSROC who had had one previous therapy and a platinum-free interval of 6 months or more. The primary outcomes were overall survival (OS) and treatment-free survival (TFS). Secondary outcomes were quality-adjusted OS and progression-free survival (PFS). Overall treatment effects on OS and PFS were estimated using an intention-to-treat, random-effect meta-analysis, with pooled individual patient data sensitivity analysis. For TFS analyses, the pooled OS was partitioned into three states of time on the 2nd-line treatment exposure (T), time free of treatment or death (TFS), and time after the first subsequent therapy or death (REL). TFS with quality-adjusted OS analyses were calculated by multiplying the mean time in each health state by its assigned utility (quality-adjusted OS = ut × T + TFS + urel × REL).

Results We obtained the extended follow-up IPD of 842 patients from two eligible trials (GOG-0213 and SOC-1), and digitized the reported survival data of 407 patients for the third trial (DESKTOP III), with a median follow-up of 87.5 months (IQR 70.3–121.2), 98.5 months (78.3–127.2), and 69.8 months (59.8–80.4), respectively. Based on all included three trials and patients, 628 to the surgery group, 621 to the control group, there were definite benefits of surgery on OS (hazard ratio [HR] 0.84, 95% CI 0.72 to 0.99), PFS (HR 0.68, 0.53 to 0.86), and TFS (difference, 7.8 months [3.5 to 12.0]). There was no significant statistical differences in effects among the trials for OS and TFS, but considerable for PFS (I2=72%, p=0.02). In the pooled data of the three trials, surgery provided longer duration of quality-adjusted OS within 84 months of follow-up than control (ranging from +1.0 to +8.0 quality-adjusted months).

Conclusions The meta-analyses with extended follow-up data substantiate SCR resulting in both treatment-free survival and overall survival benefits in women with PSROC, but having heterogeneity of PFS benefit among the three trials.

OR_100

Mesothelin-targeted CAR-T Therapy FC004 for Platinum-resistant Ovarian Cancer: A Phase I Trial

Lan Chunyan
Sun Yat-Sen University Cancer Center

Background Platinum-resistant ovarian cancer is a highly aggressive gynecological malignancy with limited treatment options. Mesothelin (MSLN), overexpressed in ovarian cancer cells, is a promising target for chimeric antigen receptor (CAR) T-cell therapy. Recent evidence shows that MSLN-CAR T cells can recognize and attack cancer cells, offering a potential new treatment for platinum-resistant ovarian cancer. We report results from a phase I study of FC004, an MSLN-CAR T-cell therapy, in patients with platinum-resistant ovarian cancer.

Methods Eligible patients were aged 18-75 years, with histologically confirmed epithelial ovarian cancer and platinum-resistant disease that had failed standard therapies. MSLN expression was ≥ 2+ or tumor positivity ≥ 40%. Patients were pre-treated with albumin-bound paclitaxel, cyclophosphamide, and fludarabine, then received a single infusion of FC004. A 3+3 dose-escalation design was used to determine the recommend ed phase II dose (RP2D), starting with 2×106 cells. The primary endpoint was safety, including the maximum tolerated dose (MTD) and RP2D. Key secondary endpoints included disease control rate (DCR), duration of response (DOR), progression-free survival (PFS), overall survival (OS), and pharmacokinetic profile.

Results As of January 2025, two patients were enrolled and received FC004 at the dose of 2×106 cells/kg. Patient #1, a 55-year-old female, received 1.09×108 CAR-T cells. CAR-T cell concentration peaked on day 7 post-infusion. One month after treatment, the patient achieved stable disease (SD) with no dose-limiting toxicities (DLTs), cytokine release syndrome (CRS), or immune effector cell-associated neurotoxicity syndrome (ICANS). Patient #2, a 51-year-old female, received 9.4×107 CAR-T cells. On the day of infusion, she experienced fever, followed by pulmonary infection and grade 3 CRS. CAR-T cell concentration peaked on day 7 and gradually decreased. On day 27, the percentage of CAR+ cells among CD3+ cells was 0.5%. Notably, CA125 levels decreased from 964 U/ml to 227 U/ml on day 11. The patient passed away one month later, likely due to complications from the infection.

Conclusions Preliminary results suggest that FC004 CAR-T cell therapy may have limited efficacy in treating platinum-resistant ovarian cancer. One patient achieved SD, while the other experienced significant toxicities, including severe CRS and pulmonary infection. The infection may have been exacerbated by prior high-dose steroid use, but a relationship with the study drug cannot be ruled out. Despite these adverse events, the decrease in CA125 levels in the latter patient suggests some biological activity. These findings highlight the need for careful patient selection and further optimization of the treatment protocol to improve safety and efficacy.

壁报交流

PO_001

miR 34a targets HMGB1 to regulate autophagy in precervical cancer cells

He Wang* Lingjia Wei
Guangxi Medical Univercity Cancer Hospital

Objective Investigating the mechanism of miR 34a regulating autophagy of cervical precancerous cells by binding to HMGB1 to induce cervical squamous intraepithelial neoplasia (CIN) to develop into cervical cancer.
Methods The lentiviruses of miR 34a inhibitor and HMGB1 mimic were transfected into Ect1/E6E7, respectively. The expressions of miR 34a and HMGB1 in each group were detected by real-time fluorescent quantitative PCR. The effects of miR 34a inhibitor and HMGB1 mimic on proliferation, transverse migration, longitudinal migration and apoptosis of Ect1/E6E7 cells were detected by CCK-8 assay, scratch assay, Transwell assay and flow cytometry, respectively. Dual luciferase assay verified the targeted binding of miR 34a to HMGB1. In vivo experiments, the effect of down-regulation of miR 34a and up-regulation of HMGB1 on tumor formation in nude mice was detected in tumor formation experiment, the effect of real-time fluorescence quantitative PCR on autophagy factor expression in nude mice tumor, and the effect of transmission electron microscopy on autophagosome formation in nude mice.
Results miR 34a can bind to the 3 '-UTR region of HMGB1, and down-regulation of miR34a can promote the expression of HMGB1 in Ect1/E6E7 cells. miR 34a inhibitor and HMGB1 mimic can promote the proliferation and migration of Ect1/E6E7 cells and inhibit apoptosis. miR 34a inhibitor and HMGB1 mimic can promote autophagy of Ect1/E6E7 cells in vivo.
Conclusions miR34a can be targeted to bind to HMGB1 to regulate the cell function by regulating the autophagy of Ect1/E6E7 pre-cervical cancer cells, which can providing a new target for CIN reversal.

PO_002

宫颈癌的免疫治疗进展

潘亚旭[1,2] 樊杨[1,2*] 马昭[1,2] 张宇茜[1,2] 张巧瑞[1,2]
1.宁夏回族自治区人民医院 2.宁夏医科大学

探讨免疫检查点抑制剂（ICIs）、肿瘤疫苗及过继细胞免疫治疗（ACT）在宫颈癌治疗中的应用现状、临床效果及未来发展方向，旨在为晚期宫颈癌患者提供新的治疗策略和精准化治疗方案。系统回顾免疫检查点抑制剂（包括 PD-1/PD-L1、CTLA-4、LAG-3、TIGIT 等靶点）的临床研究数据，分析其在宫颈癌中的疗效和安全性；梳理肿瘤疫苗（预防性、治疗性）及过继细胞免疫治疗（TIL、TCR-T、CAR-T）的研发及临床试验进展。免疫检查点抑制剂在晚期宫颈癌中取得显著疗效，如帕博利珠单抗、纳武利尤单抗等 PD-1 抑制剂的客观缓解率（ORR）及疾病控制率（DCR）表现良好，且联合化疗或免疫治疗方案可进一步提升总生存期（OS）和无进展生存期（PFS）。CTLA-4 抑制剂伊匹木单抗在联合治疗中也展现良好前景。肿瘤疫苗研发取得突破，如 DNA 疫苗 VGX-3100 在 III 期试验中达到组织病理学转归与病毒清除双终点。过继细胞免疫治疗进展显著，TIL、TCR-T 及 CAR-T 疗法在临床试验中显示出持久抗肿瘤效应及潜在应用前景。免疫检查点抑制剂、肿瘤疫苗及过继细胞免疫治疗为晚期宫颈癌患者提供了更多治疗选择和希望。未来需进一步探索疗效预测标志物、优化联合治疗策略，推动免疫治疗向精准化、低毒化方向发展。

PO_003

基于 E6/E6AP 复合体的 E6 靶向肽及其与喜树碱偶联物用于治疗 hr-HPV 阳性宫颈癌

花月 沈杨*
Southeast University

目的 宫颈癌是女性生殖系统中最常见的恶性肿瘤之一。根据国际癌症研究机构的统计，2022 年全球约有 66 万名妇女被诊断为宫颈癌，约 35 万名妇女死于该疾病。仅在我国，每年新发宫颈癌病例就有近 11 万例，约 6 万人因此死亡，而且该病的平均发病年龄呈逐年下降趋势，是严重威胁全球女性生命健康的恶性肿瘤。Hr-HPV 的持续感染是宫颈癌发生的关键因素。尽管随着筛查和疫苗的普及，宫颈癌的发病率有所下降，但是对于已经感染的高风险人群，现有的预防手段疗效有限。Hr-HPV E6 癌蛋白作为关键致癌因子，是一种表面高度可变但骨架保守的蛋白，且 HPV16 E6 全场 X 射线晶体结构于 2013 年完成，因此开发新型特异性靶向 E6 蛋白的治疗性药物，有望从根本上阻断 Hr-HPV 感染引发的宫颈癌的发展和进程。
方法 本项目首先通过人工智能筛选出特异性靶向 E6 蛋白的多肽序列共计 1024 条，随后通过固相合成的方法合成前 22 条，并通过表面等离子共振及细胞热漂移实验验证其体内外靶向能力。通过激光共聚焦、动物荧光成像验证其细胞和或体内的靶向能力。再本项目的最后，为了提高靶向肽的治疗效果，我们通过筛选宫颈癌细胞敏感的化疗药物并将其与靶向肽进行偶联，并通过细胞毒性实验、透射电镜、免疫共沉淀及宫颈癌皮下瘤模型验证其生物活性。
结果 SPR 实验证明多肽 P9 具有最强的 E6 亲和力。CETSA 实验结果表明 P9 能够与 E6 蛋白结合增强其热稳定性，改变了 E6 蛋白的变性曲线。 P9 在与宫颈癌细胞孵育 12 h 后能够被溶酶体摄取。免疫荧光显示 P9 可以与 E6 蛋白共定位，且动物成像显示 P9 具有良好的肿瘤靶向性。由于低浓度的 P9 几乎无细胞毒性，因此我们将 P9 与 CPT 进行共价偶联，不仅提高了 CPT 的 IC_{50}，还提高了 CPT 的肿瘤靶向能力。随后 WB 和免疫共沉淀验证了多肽偶联药物的作用机制，结果表明多肽偶联药物主要通过竞争性的与 E6 蛋白结合，阻止 E6/E6AP 复合体的形成，从而使得 p53 蛋白泛素化的减少，进一步诱导细胞发生凋亡。
结论 本研究所构建的多肽 P9 具有良好的 E6 靶向效果，且与喜树碱共价结合的多肽偶联药物不仅具有 P9 的靶向能力，还可以增强 CPT 抑制肿瘤生长，起到“1+1＞2”的效果。

PO_004

宫颈癌患者保留生育能力后妊娠 1 例病例报告

马帅 王敏*
吉林大学第二医院

目的 宫颈癌严重威胁女性生命健康，其发病率在女性生殖系统恶性肿瘤中位居首位。宫颈癌不仅影响育龄期女性的生存率，还影响其生育功能。本文报道 1 例宫颈癌患者保留生育能力后妊娠的患者，为有生育需求的宫颈癌患者提供治疗依据。
方法 患者女，39 岁，G1P0，孕 35 周入院，1 年前因 CINIII 于当地医院行宫颈冷刀锥形切除术+宫颈管骚刮,术后病理提示宫颈鳞状细胞癌（IA1 期），术后定期复查 HPV16/18 阳性，患者表示知情，坚决拒绝治疗。孕期无感冒史及发热史，无头痛、头晕、视物模糊等症状，孕期有阴道流血及流液史，量少，未予治疗后好转。产科情况：腹部膨隆，与孕周相符。宫高 32 厘米，腹围 105 厘米。胎心 145 次/分。胎动可及，未触及宫缩。内诊：阴道通畅，粘膜皱襞润软，分泌物量少，色白，无异味。宫颈表面呈红色糜烂样改变，约占宫颈表面 2/3 以上，可见宫颈锥切术后痕迹，宫颈口未开，先露为头，

S>-3。疑难病理会诊：（宫颈 3,4,5,7,8,9,11 点）：HSIL/CINⅢ级，累及腺体，（3,4,8 点）局灶见有鳞状细胞癌（浸润最深约 1.5mm）。产科三维彩超：胎位：头位，胎心：153 次/分，胎动：可及，胎儿颈部见脐带压迹"W"形。入院诊断：孕 35 周，孕 1 产 0，妊娠合并子宫颈术后宫颈异常宫颈恶性肿瘤个人史。

结果 在插管全麻下行子宫下段剖宫产术+经腹筋膜外全子宫切除术+双侧输卵管切除术。以 LOA 娩出一女性活婴，新生儿体重 2360g，出生后一分钟 Apgar 评分 8 分，出生后五分钟 Apgar 评分 9 分。术中经过顺利，麻醉满意。术后给予预防感染、缩宫素促宫缩、补液等对症治疗。术后病理回报：宫颈：浸润性鳞状细胞癌，浸润宫颈浅层，脉管内未见癌浸润，伴 HSIL/CIN3 级，累及腺体，分泌型宫内膜，间质有蜕膜样变，肌壁浅层见少量多核合体滋养层细胞，阴道断端及宫旁组织未见癌，子宫腺肌症，子宫平滑肌瘤。剖宫产术后第 2 日母女平安出院，嘱其定期复查。

结果 保留生育功能的宫颈冷刀锥切术对正常组织的损伤较小，能减少早期宫颈癌患者的术中出血量，降低并发症发生率，有利于患者的术后恢复，减轻炎性浸润和复发风险，有助于改善患者的妊娠结局。

PO_005

中医药诱导宫颈癌细胞凋亡信号通路的研究机制

王晓雪[1] 王院春[2*]
1.陕西中医药大学 2.陕西中医药大学附属医院

目的 宫颈癌作为全球女性高发恶性肿瘤之一，其发病机制与 HPV 持续感染密切相关。由于早期症状隐匿，多数患者确诊时已进展至中晚期，临床诊疗面临肿瘤转移、化疗耐药性及相关毒副反应等瓶颈问题。中医药基于多靶点网络调控、整体调节的核心特性，在协同增效、改善患者生存质量等方面展现出独特优势。本研究通过系统解析中药单体化合物、活性成分及复方制剂经信号转导通路诱导宫颈癌细胞凋亡的分子机制，旨在为构建中医药治疗宫颈癌的精准临床方案提供可靠理论依据。

方法 本研究采用系统文献综述法。基于 PubMed、CNKI、CBM 等学术数据库进行跨库检索。检索策略采用主题词与自由词相结合的方式，核心检索词包括："宫颈癌"、"细胞凋亡"、"信号通路"、"中药"、"中药单体"、"中药复方"等。利用 EndNote X9 对纳入文献进行去重与初步筛选，将其进行分析及机制层面的系统整合。

结果 研究证实，中药制剂在宫颈癌治疗中有抑制血管生成、减轻放化疗毒副反应等临床优势；其中，中医药在延长无进展生存期、延缓耐药性进展等方面亦表现出显著优势。现代药理学研究表明，诱导肿瘤细胞凋亡是中医药发挥抗宫颈癌作用的关键机制。因此，中药制剂通过多靶点调控凋亡相关信号通路为宫颈癌的临床治疗提供了新的思路。

结果 本研究阐明中医药通过多靶点网络调控诱导宫颈癌细胞凋亡的机制，为构建其抗肿瘤理论框架提供实证。中药复方凭借活性成分协同增效，在抑制增殖、重塑微环境等方面具独特优势。未来需聚焦基于网络药理学的活性成分筛选及中药调控表观遗传修饰的抗凋亡机制研究。该工作为中医药现代化及宫颈癌个体化精准诊疗提供了重要支撑。

PO_006

Beyond infection status: A cross-sectional study of socioeconomic disparities, infection dynamics, and psychosocial burden among women with HPV in Luzhou, China

Jiewei Ming[1,2] Dan Nie[1*]
1.Department of Gynecology, The Affiliated Hospital, Southwest Medical University
2.Department of Gynecology, People's Hospital of Naxi District

Objective This study investigates socioeconomic disparities, infection characteristics, and psychosocial burden among HPV-infected women in Luzhou, China. It aims to: 1) Determine anxiety/depression prevalence; 2) Assess quality of life impact using a validated scale; 3) Identify predictors of psychological distress; and 4) Analyze key psychosocial domains contributing to adverse outcomes.
Methods This cross-sectional study enrolled 441 HPV-infected women from two tertiary hospitals in Luzhou, China (November 2023 - September 2024). Data were collected electronically via Questionnaire Star using four instruments: a General Information Questionnaire (capturing demographics, socioeconomic status [monthly income, education, residence], behavioral factors [smoking, alcohol], obstetric/gynecological history [pregnancies, births, sexual partners], and HPV infection dynamics [subtypes, duration]); the GAD-7 for anxiety; the PHQ-9 for depression; and the culturally adapted Human Papillomavirus Impact Profile (HIP) assessing seven psychosocial domains (Worries/Concerns, Emotional Impact, Sexual Health, Self-Image, Partner Relationships, Doctor Interactions, Life Impact). Data analysis employed SPSS 25.0 for descriptive statistics, group comparisons, correlation analysis, and multivariate logistic regression identifying predictors of anxiety/depression, and AMOS 24.0 for confirmatory factor analysis (CFA) validating the HIP scale's structural validity.
Results Among 441 HPV-infected women (median age 38 years; 59.81% urban residents; 53.52% income <3000 yuan/month), anxiety and depression prevalence reached 40.36% (n=178) and 41.72% (n=184), respectively. The HIP scale demonstrated excellent reliability (Cronbach's α=0.946) and validity (CFI=0.927, RMSEA=0.079). Multivariate analysis identified key predictors:
Anxiety: Higher income (OR=2.84, 95%CI 1.59–5.08), smoking (OR=2.78, 1.08–7.14), HPV16/18 infection (OR=2.07, 1.29–3.32), infection duration <3 years (OR=1.77, 1.10–2.83).
Depression: Higher income (OR=2.14, 1.20–3.81), smoking (OR=3.28, 1.30–8.28), fewer pregnancies (OR=0.64, 0.42–0.97), fewer births (OR=0.63, 0.41–0.96), HPV16/18 infection (OR=2.09, 1.31–3.34), infection duration <3 years (OR=1.92, 1.20–3.08).
The "Worries/Concerns" domain exhibited the highest psychosocial burden (66.73±34.33; CV=51.4%) and strongest correlations with anxiety (r=0.464) and depression (r=0.463), with specific drivers including STD/cancer fears, emotional shock from diagnosis, negative self-image, and partner relationship concerns.
Results Among 441 HPV-infected women (median age 38 years; 59.81% urban; 53.52% income <3000 元/month; 93.65% non-smokers), HPV16 (28.80%), HPV52 (22.68%), and persistent infection >1 year (45.80%) were predominant. Anxiety prevalence reached 40.36% (scores: mild 6.58±1.10 → severe 16.61±1.27) and depression 41.72% (mild 7.08±1.85 → severe 22.08±2.64). The HIP scale demonstrated robust reliability (Cronbach's α=0.946) and validity (CFI=0.927, RMSEA=0.079). Key multivariate predictors included:
Anxiety: Higher income (OR=2.84, 95%CI 1.59–5.08), smoking (OR=2.78, 1.08–7.14), HPV16/18 infection (OR=2.07, 1.29–3.32), infection duration <3 years (OR=1.77, 1.10–2.83).
Depression: Higher income (OR=2.14, 1.20–3.81), smoking (OR=3.28, 1.30–8.28), fewer pregnancies (OR=0.64, 0.42–0.97), fewer births (OR=0.63, 0.41–0.96), HPV16/18 (OR=2.09, 1.31–3.34), infection duration <3 years (OR=1.92, 1.20–3.08).

The "Worries/Concerns" domain showed the highest psychosocial burden (66.73±34.33; CV=51.4%) and strongest correlations with anxiety (r=0.464) and depression (r=0.463), with specific predictors including: anxiety driven by STD/cancer fears, emotional reactions to diagnosis, and discomfort during exams; depression additionally linked to negative self-image and partner acceptance concerns.
Conclusion Over 40% of HPV-infected women in Luzhou experience significant anxiety/depression, with heightened burden linked to higher income, HPV16/18 infection, and shorter infection duration. Key drivers include cancer-related fears, emotional distress, self-image issues, and relational concerns. Targeted interventions addressing these psychosocial domains—through education, counseling, and stigma reduction—are essential to mitigate mental health burdens and improve quality of life.

PO_007

不同近距离治疗模式在 IIB 期大肿块、IIIB 期中晚期宫颈癌的应用研究

甘玲 华丽 冯成军 陈绍俊*
柳州市工人医院

目的 探讨腔内联合组织间插植、标准 Fletcher 施源器腔内联合宫旁 IMRT 补量以及标准 Fletcher 施源器腔内和腔内联合组织间插植交替这三种近距离治疗模式在 2018 FIGO 分期 IIB 期大肿块（≥4cm）、IIIB 期中晚期宫颈癌治疗中的剂量学、疗效及毒副反应差异，为伴宫旁或盆壁侵犯的中晚期宫颈癌选择合适的近距离治疗方式提供理论依据。
材料与方法 回顾性分析 2019 年 8 月至 2024 年 8 月在我院接受根治性放疗的 113 例 2018 FIGO 分期 IIB 期大肿块、IIIB 期宫颈癌患者的临床资料。根据不同的近距离治疗模式将患者分为插植组、交替组及宫旁补量组，比较三组在剂量学、疗效及毒副反应发生率方面的差异，应用倾向得分匹配（PSM）校正混杂偏倚和选择偏倚后进一步验证。
结果 1.剂量学参数方面：在靶区剂量方面，插植组的靶区适形指数显著优于交替组和宫旁补量组，V200 显著小于另外两组，差异具有统计学意义（P＜0.001），但插植组的高危临床靶区 D90 较另外两组无显著差异（P＞0.05）。PSM 校正后仍得出一致结果。在危及器官（OAR）受量方面，插植组的 D2cc 较另外两组无显著差异（P＞0.05）；但插植组的直肠 D0.1cc 及膀胱 D0.1cc、D1cc 低于交替组，膀胱 D1cc 低于宫旁补量组，差异均具有统计学意义（P＜0.05）。PSM 校正后插植组 OAR 受量基本低于交替组。
2.近期疗效方面：插植组、交替组和宫旁补量组的完全缓解率分别为 66.2%、60.0%和 46.2%，客观有效率分别为 96.9%、94.3%和 92.3%，疾病控制率分别为 96.9%、97.1%和 92.3%，插植组的近期疗效相对另外两组较好，但差异无统计学意义（P＞0.05）。经 PSM 校正后仍得出一致结论。
3.远期疗效方面：插植组的远期疗效相对较好，其 1 年、2 年的无远处转移生存率（DMFS）显著高于交替组，差异具有统计学意义（P=0.034），但两组的总生存率（OS）和无进展生存率（PFS）无显著差异（P＞0.05）。经 PSM 校正后，两组的 OS、PFS 和 DMFS 均无显著差异（P＞0.05）。
4.毒副反应方面：插植组与交替组、宫旁补量组间的近期和远期毒副反应发生率均无显著差异（P＞0.05），PSM 校正后仍得出一致结论。插植组出血发生率高于交替组和宫旁补量组，差异具有统计学意义（P＜0.001）；插植组的穿孔发生率较高，但与另外两组相比无显著差异（P＝1.000）。
结果 腔内联合组织间插植在 2018 FIGO 分期 IIB 期大肿块、IIIB 期中晚期宫颈癌的治疗中具有更好的靶区适形度，OAR 受量较低，毒副反应可耐受，近期和远期疗效相对较好，可能为这类患者相对较合适的近距离治疗模式。对于形态较规则或外照射后肿瘤体积显著缩小的 2018 FIGO 分期 IIB 期大肿块、IIIB 期宫颈癌患者，可以尝试选择性地应用交替的方式进行治疗。

PO_008

Integrated Transcriptome Analysis of Malignant Transformation Subgroups in Cervical Cancer and Vitamin D Targeted Therapy

Yashi Xu
Wuhan Union Hospital

Objective Cervical cancer is a common gynecological malignant tumor. The main cause of cervical cancer is long-term and persistent infection with high-risk human papillomavirus (HPV). The process of cervical cancer occurrence and development has the characteristics of multi-stage and progressive evolution. High grade squamous intraepithelial lesions (HSIL) of the cervix are precancerous lesions of cervical cancer. The standardized treatment of HSIL is a key focus in reducing invasive cervical cancer. According to reports, vitamin D can exert anti-cancer effects by regulating the immune system and inhibiting cancer cell growth, playing an important role in preventing precancerous lesions. However, there is still little research on whether vitamin D can prevent the progression of cervical precancerous lesions. This study aims to reveal the key molecular events and characteristics of epithelial cell subpopulations involved in the transformation of cervical precancerous lesions into cervical cancer through single-cell transcriptome and spatial transcriptome analysis, and to explore therapeutic drugs for blocking cervical precancerous lesions based on these features.
Methods Single cell transcriptome sequencing technology was used to analyze cervical precancerous lesions and cervical cancer samples, identifying the molecular and functional characteristics of key epithelial cell subsets. Using spatial transcriptome analysis to determine the spatial location of epithelial cell subpopulations. Through in vitro cell experiments and organoid culture, the effects of vitamin D analogues on the proliferation and migration ability of cervical precancerous cell lines and organoids were detected. Further analyze the regulatory effect of vitamin D analogues on the cell cycle. Finally, the inhibitory effect of vitamin D analogues on the progression of cervical precancerous lesions was validated in an in vivo model.
Results Single cell transcriptome analysis revealed the presence of key molecular subgroups at the stage of cervical precancerous lesions transforming into cancer. Some signaling pathways closely related to tumorigenesis, such as MTORC1, were continuously activated, driving cells towards malignant transformation, while significantly negatively enriching immune pathways such as interferon. Vitamin D receptors and their signaling pathways undergo significant dynamic changes during disease progression. The spatial transcriptome analysis showed that the C4 cluster is located at a special tumor boundary position in the tissue. In vitro experiments have shown that vitamin D analogues significantly inhibit the proliferation and migration ability of cervical precancerous cell lines and organoids, and affect the regulation of cell cycle. Further in vivo experiments have confirmed that vitamin D analogues effectively inhibit the progression of cervical precancerous lesions.
Conclusions This study revealed the key molecular events and epithelial cell subpopulation characteristics involved in the transformation of cervical precancerous lesions into cervical cancer through single-cell transcriptome sequencing and spatial transcriptome analysis. It also confirmed that vitamin D analogs can effectively inhibit the progression of cervical precancerous lesions in vitro and in vivo, suggesting their potential application value in the prevention and treatment of cervical cancer and paving the way for exploring new treatment strategies in the future.

PO_009

Analysis of risk factors associated with lymph node metastasis in early cervical squamous cell carcinoma and establishment and evaluation of prediction mode

Man Liang[1] Zhifu Cai[2*] He Wang[2]
1.Wuming Hospital of Guangxi Medical University 2. Guangxi Medical University Cancer Hospital

Purpose The aim of the study was to identify the risk factors of lymph node metastases(LNM) in early cervical squamous cell carcinoma, and to establish and evaluate the related prediction model.
Methods This study retrospectively analyzed 351 patients diagnosed with early (stage IB-IIA) cervical squamous cell carcinoma who had undergone radical surgery between January 2013 and September 2021. Research design was case control study. According to pathological findings , case was patients with node metastases and control was those without node metastases. The metastatic risk factors of the early cervical squamous cell carcinoma was investigated,and the establishment of lymph node metastasis prediction model presented with nomogram.Also the further evaluation of the modle was carried out.
Results The Results showed that premenopausal, high level of scc-Ag, the size of tumor ≥ four cm, depth of interstitial invasion ≥1/2, vascular tumor thromus, and low level of ki-67 were related to the metastasis of lymph nodes($P<0.05$). To establish a nomogram prediction model for lymph node metastasis of early cervical squamous cell carcinoma based on six risk factors. Area AUC of the ROC curve of the prediction model was 0.797. The calibration curve was a straight line with a slope close to 1.
Conclusion Premenopausal, high level of scc-Ag, the size of tumor ≥ 4 cm, depth of interstitial invasion ≥1/2, vascular tumor thromus, and low level of ki-67 were independent risk factors for lymph node metastasis of early cervical squamous cell carcinoma. The nomogram prediction model had high accuracy.

PO_010

Initial Bevacizumab Combined with Chemoradiotherapy Significantly Enhances Progression-Free Survival in High-Risk Stage IIIB-IVA Cervical Cancer

Mingzi Tan Yuhua Gao*
Liaoning Cancer Hospital& Institute

Purpose To evaluate the efficacy of bevacizumab combined with chemoradiotherapy (CCRT) as initial therapy for locally advanced cervical cancer (stage IIIB-IVA).
Methods This retrospective study included 185 patients (2019–2022) with stage IIIB-IVA cervical squamous carcinoma, divided into a combination group (bevacizumab + CCRT, n = 65) and a CCRT group (n = 120). Kaplan-Meier analysis compared progression-free survival (PFS); Cox regression models identified prognostic factors.
Results 3year overall survival (OS) did not differ significantly (98.5% vs. 94.2%, $P > 0.05$). However, the combination group showed superior 1-, 2-, and 3-year PFS rates (100% vs. 84.2%; 95.4% vs. 75.8%; 95.4% vs. 74.2%; all $P < 0.05$). Kaplan-Meier curves confirmed significantly better PFS for the combination group (log-rank P = 0.004, 0.014, 0.009 at 1, 2, 3 years). Univariate analysis identified advanced stage (HR = 2.3, 95% CI 1.45–3.65), tumor diameter ≥6 cm (HR = 1.8, 95% CI 1.20–2.70), and lymph node metastasis (HR = 2.1, 95% CI 1.50–2.94) as poor prognostic factors. High-risk subgroups (tumor ≥6 cm, lymph node/aortic node metastasis) exhibited significantly improved 3-year

PFS with combination therapy (all P < 0.05). Multivariate Cox analysis confirmed bevacizumab as protective (HR = 0.65, 95% CI 0.42–0.98), while advanced stage (HR = 3.42, 95% CI 1.78–6.59), tumor ≥6 cm (HR = 1.8, 95% CI 1.25–2.60), and lymph node/aortic node metastasis (HR = 2.5, 95% CI 1.70–3.68; HR = 3.1, 95% CI 1.87–8.67) were independent risk factors (all P < 0.05). Conclusion: Bevacizumab combined with CCRT significantly enhances PFS in locally advanced cervical cancer, particularly in high-risk subgroups. Initial treatment with bevacizumab-based regimens is recommended for these patients.

PO_011

外周血炎症生化指标对 宫颈癌临床病理特征的关系探讨

高璐璐
庆阳市人民医院

目的 探讨外周血中鳞状上皮细胞癌抗原（SCC-Ag）、中性粒/淋巴细胞（NLR）、血小板/淋巴细胞（PLR）、单核/淋巴细胞（MLR）、纤维蛋白原/白蛋白（FAR）、血细胞比容（HCT）及 D-二聚体与宫颈癌的临床病理特征之间的关系。
方法 收集宫颈癌患者 210 例为研究对象，年龄 25～80 岁（51.82±11.00），根据 2018 国际妇产科联盟（FIGO）分期，其中早期宫颈癌即≤IIA 期 132 例，局部晚期及晚期宫颈癌即≥IIB 期及 IB3 期 78 例；宫颈鳞癌 170 例，非鳞癌 40 例；宫颈高分化鳞状细胞癌 150 例，非高分化鳞状细胞癌 60 例。收集患者治疗前外周血中采用免疫化学发光法检测的 SCC-Ag 水平，收集使用全自动血液分析仪检测外周血中中性粒细胞、血小板、单核细胞、淋巴细胞、纤维蛋白原、白蛋白、血细胞比容（HCT）、D-二聚体绝对值计数，以宫颈癌 FIGO 分期、组织类型、分化程度分组，采用 SPSS 27.0 分析以上生化指标与宫颈癌临床病理特征之间的相关性。采用受试者工作特征曲线（ROC）分析上述生化指标单独和联合对局部晚期及晚期宫颈癌的诊断效能。
结果 局部晚期及晚期宫颈癌患者外周血的 SCC-Ag、NLR、PLR、MLR、FAR、D-二聚体均显著大于≤IIA 期患者，HCT 显著小于≤IIA 期（P＜0.05）。SCC-Ag、NLR、PLR、MLR、FAR、D-二聚体与宫颈癌 FIGO 分期均呈正相关，HCT 与宫颈癌 FIGO 分期呈负相关（P<0.05）。患者外周血中，鳞状细胞癌组 SCC-Ag 水平显著高于非鳞状细胞癌组（P<0.05）。ROC 曲线分析结果显示，SCC-Ag、NLR、PLR、MLR、FAR、HCT 及 D-二聚体单一指标预测局部晚期及晚期宫颈癌的曲线下面积（AUC）分别为 0.790、0.618、0.627、0.639、0.673、0.590、0.646，以上指标联合预测局部晚期及晚期宫颈癌患者的 AUC 为 0.865。
结论 宫颈癌患者治疗前外周血中的 SCC-Ag 及 NLR、PLR、MLR、FAR、HCT 及 D-二聚体等炎症指标可作为宫颈恶性肿瘤分期的参考指标。

PO_012

HPV16 整合相关外泌体 circMAP3k5 激活 POSTN⁺ AFs 促进宫颈上皮内瘤变

郭楚鸿[1] 梁罗娇[1] 王薇[2*]
1.广州医科大学附属第一医院 2.上海市第一妇婴保健院

目的 探讨宫颈上皮内瘤变 HPV16 整合相关外泌体参与诱导成纤维细胞激活的作用和机制研究。
方法 通过生物信息学分析、qRT-PCR 和原位杂交筛选 HPV16 整合诱导的关键 circRNA：circMAP3K5。转染 siRNA 或慢病毒分别构建敲低或过表达 circMAP3K5 的宫颈癌前细胞。提取上述细胞外泌体刺激成纤维细胞，通过 WB、划痕实验、Transwell 迁移实验和 ELISA 验证外泌体 circMAP3K5 对正常成纤维细胞转化成 POSTN⁺活化成纤维细胞（activated fibroblasts，AFs）的作用。
结果 circMAP3K5 高表达与宫颈病变进展呈正相关。circMAP3K5 在宫颈上皮内瘤变外泌体中富集并传递给成纤维细胞，诱导 POSTN⁺成纤维细胞的激活。此外，circMAP3K5 直接结合 miR140-3p，靶向调控 POSTN 表达。成纤维细胞中 miR-140-3p 的沉默会导致 α-SMA、FAP 和 POSTN 的高表达和迁移能力增强，而过表达 miR-140-3p 能抑制外泌体诱导的成纤维细胞活化和 POSTN 的分泌。
结论 宫颈癌前细胞外泌体通过 circMAP3K5/miR140-3p/POSTN 通路促进成纤维细胞活化成 POSTN⁺ AFs。在宫颈上皮内瘤变中，circMAP3K5 是 HPV16 相关外泌体介导 POSTN⁺ AFs 活化的关键分子，为宫颈上皮内瘤变进展的诊断和治疗靶点的开发提供了方向。

PO_013

整合单细胞测序和空间转录组学解析宫颈神经内分泌癌发生发展的分子机制和肿瘤微环境状态

江晓霞 李江莎 李政*
昆明医科大学第三附属医院 云南省肿瘤医院 北京大学肿瘤医院云南医院

研究目的 宫颈神经内分泌癌（NECC）是宫颈癌中的罕见病理亚型，有着极强的侵袭性和极差的预后。NECC 与 HPV 18 的感染密切相关，然而目前我们对 NECC 确切的细胞起源和驱动神经内分泌转化的关键分子事件仍知之甚少。本研究拟通过单细胞测序和空间转录组技术，解析 NECC 发生发展的分子机制及其肿瘤微环境状态，并与其他宫颈癌类型做比对，揭示其细胞起源。
材料与方法 本研究收集了 7 例在我院接受全程规范治疗的 NECC 患者的新鲜肿瘤组织样本及其配对的 3 例邻近正常宫颈组织作为对照，进行了单细胞测序（ScRNA-seq）。同时我们整合了公共数据库中其他宫颈癌亚型的单细胞数据进行联合分析。此外，我们对 4 例 NECC 患者的新鲜肿瘤组织进行了 10×Visium 空间转录组测序（ST），并与公共数据库中宫颈鳞状细胞癌的空间转录组学数据进行了整合比较。
结果
1.NECC 较其他亚型的宫颈癌具有更显著的免疫抑制特征，具体表现为异质性 T 细胞亚型（如 Treg、耗竭 CD8+T 细胞）的富集，以及免疫检查点分子（PD-1/CTLA-4/LAG-3）表达的上调。空间转录组分析进一步证实，免疫细胞在肿瘤核心区域呈“荒漠化”分布特征，而在侵袭前沿却与癌相关成纤维细胞（CAFs）形成空间共定位。
2.此外，HPV-18 型感染可特异性驱动神经内分泌表型转化。其机制涉及：诱导细胞周期 G1/S 期调控异常（Cyclin D1/CDK4 上调）；增加基因组不稳定性（TP53 突变富集）。这两种机制共同激活神经分化核心转录因子（ASCL1/NEUROD1）。同时，HPV-18 型感染还通过抑制 DNA 损伤修复通路（BRCA1/RAD51 下调）影响患者的预后。

3.NOTCH 信号网络通过双向动态调控驱动 NECC 进程：在肿瘤发生早期，NOTCH2NL 表达下调抑制细胞分化信号，促进神经内分泌转化并驱动 ASCL1⁺ 神经内分泌（NE）细胞分化；而进展晚期，NOTCH 通路通过 JAG1/NOTCH3 表达上调加剧肿瘤异质性和恶性进展，共同维持 NECC 的演进动态平衡。
4.WNT/β-catenin 信号通路在肿瘤-基质界面异常活化，通过调控转录因子 TCF7L2/YAP1 介导腺-神经内分泌转化（Adenoneuroendocrine Transdifferentiation，ANT）。
5.NECC 存在基因关键转录因子的分子亚型（ASCL1⁺/NEUROD1⁺ vs. ZEB1⁺/POU2F3⁺），其中 ZEB1⁺ 亚型显著富集缺氧信号、EMT 特征，并与患者不良预后密切相关。
结论 本研究揭示 HPV-18 感染通过诱导细胞周期失调与基因组不稳定驱动神经内分泌转化，并阐明 NOTCH/WNT 信号通路的双相动态调控是 NECC 恶性演进的核心机制。同时基于关键转录因子的分子分型为预后分层提供了新的理论依据。

PO_014

Targeting ROR1: An EMT-Based Prognostic Model Guides Novel Therapeutic Strategy in Cervical Cancer

Xiaoxia Jiang Jiangsha Li Zheng Li*
The Third Affiliated Hospital of Kunming Medical University, Yunnan Cancer Hospital, Peking University Cancer Hospital Yunnan

Purpose Aiming at the problems of limited clinical application of EMT-related gene prognostic models and unknown targeting strategies, this study constructed a novel EMT prognostic model, targeting ROR1 as a poor prognostic factor and verifying its value and mechanism as a therapeutic target.
Methods
1. we collected information on cervical cancer patients who underwent radical surgery at our institution between January 2016 and December 2016 and constructed a 13-gene EMT prognostic model by univariate Cox regression, LASSO, and multivariate COX analysis. External validation was performed to create a clinical column-line diagram combining risk scores and lymph node status.
2. We assayed mRNA and protein levels of 4 EMT genes (FAM83A, LIPG, ROR1, PLA2G7) in fresh cervical cancer tumor tissues and paired normal cervical tissues.
3. We examined the protein expression levels of the 4 EMT-related genes, CD8 and CD68, in a large clinical cohort and analyzed their correlation with key pathological factors in patients.
4. We constructed cervical cancer cell lines and control cells that interfered with endogenous ROR1 expression and detected functional phenotypic changes in the cells by CCK8, clone formation assay, scratch assay, Transwell assay and flow cytometry.
Results
1. We successfully constructed a prognostic predictive risk model for 13 EMT-related genes based on the TCGA database and the EMTome database (1/3/5-year OS AUC are all >0.5), and the predictive efficacy is satisfactory. At the same time, we established a survival prediction column-line graph containing risk scores and lymph node status.
2. RT-qPCR and Western Blotting showed that the mRNA expression level of ROR1 in fresh cervical cancer tissues was lower than that in normal tissues next to the cancer, while the protein expression level was the opposite.
3. Immunohistochemical experiments confirmed that the protein expression of four EMT-related genes in tumor tissues was higher than that in normal cervical tissues. Meanwhile, the expression level of FAM83A was found to be significantly correlated with FIGO stage, tumor diameter, pathological type, lymphatic infiltration and choroidal cancer embolus ($P<0.05$); the expression of LIPG was significantly correlated with FIGO stage and pathological type ($P<0.05$); the expression of ROR1 was significantly correlated with lymphatic infiltration and choroidal cancer embolus ($P<0.05$) and its high expression was correlated with the poor prognosis and high risk of recurrence and metastasis of the patients. and

high risk of recurrence and metastasis (P<0.05); the expression of PLA2G7 was significantly associated with age, FIGO stage, pathological type, lymphatic infiltration, and choroidal cancer embolism (P<0.05); the expression of CD8 in tumor cells was significantly correlated with pathological type (P=0.0145), and the high expression of CD68 was significantly correlated with poor prognosis (P=0.0011). What's more, the expression of ROR1 was an independent prognostic factor in cervical cancer patients (P < 0.05).
4. CCK-8 assay and clone formation assay found that the proliferation ability of HeLa-shROR1-1 and HeLa-shROR1-2 cells was weakened compared with that of HeLa-shControl; scratch assay and Transwell assay found that the invasive and migratory ability of HeLa-shROR1-1 and HeLa-shROR1-2 cells was weakened compared with that of HeLa- shControl; flow cytometry found that HeLa-shROR1-1 and HeLa-shROR1-2 cells entered the G0 phase more often than HeLa-shControl; and flow apoptosis found that HeLa-shROR1-1 and HeLa-shROR1-2 cells did not show significant changes in apoptosis compared to HeLa-shControl. for which apoptosis was not significantly altered.
Conclusions The 13-gene EMT prognostic model and clinical column line diagram constructed in this study have clinical predictive value, and for the first time, it was experimentally demonstrated that ROR1 acted as an oncogene to promote the proliferation, migration, and invasion of cervical cancer cells in vitro, and facilitated the entry of cervical cancer cells into the proliferative cycle, but had no significant effect on apoptosis, which may be able to provide a new strategy for the targeted treatment of cervical cancer.

PO_015

骆驼刺提取物抑制宫颈癌裸鼠抑制瘤生长

迪丽娜尔·艾山江
克州人民医院

探讨骆驼刺有效提取物（原儿茶醛）对宫颈癌大鼠癌组织中 EGFR、PKM2 及 STAT3 的影响。
方法 选取裸鼠雌性 30 只建宫颈癌模型，细胞接种 4 周后随机分为模型组（1 组）、模型+顺铂阳性药物组（2 组）、模型+高剂量骆驼刺组（3 组）、模型+中剂量骆驼刺组（4 组）、模型+低剂量骆驼刺组（5 组），个 5 只，2 组裸鼠尾静脉注射阳性药顺铂 4.3ml/kg，3、4、5 组分别灌胃高、中、低浓度骆驼刺提取物（115mg/ml、46mg/ml、11.5mg/ml）8.7ml/kg，1 组注射等量生理盐水，每天观察裸鼠肿瘤生长情况，36 天后处死大鼠，取瘤体，按分组整齐摆放于单色背景下，加标尺拍照，测量肿瘤重量，并对癌组织中 EGFR、PKM2 及 STAT3 的表达进行检测。
结果 五组裸鼠瘤体体积均呈现增长趋势，其增长速度又快到慢分别排序为：1 组＞5 组＞4 组＞3 组＞2 组，瘤体重量在不同组之间差异有统计学意义（F=52.582，P＜0.001）。通过对不同分组裸鼠的组织固定样本进行免疫组织化学法测定，加过显示 EGFR、PKM2、STAT3 的表达在不同组之间差异有统计学意义（P＜0.001）。结论 ：骆驼刺有效提取物能抑制宫颈癌大鼠病灶的生长，其作用机制之一可能是能够降低癌组织 EGFR、PKM2 及 STAT3 的表达有关。

PO_016

LDHB 琥珀酰化修饰对宫颈癌细胞代谢及放疗敏感性的研究

庄素宏 孙阳*
福建省肿瘤医院

研究目的 鉴于乳酸脱氢酶 B（LDHB）在细胞糖酵解和能量代谢中的关键作用，以及蛋白质琥珀酰化修饰在调控细胞代谢和肿瘤治疗中的潜在影响，本研究旨在探讨 LDHB 琥珀酰化修饰对宫颈癌细胞代谢

和放疗敏感性的影响。尽管已有研究表明 LDHB 的琥珀酰化修饰可能调节其酶活性，但该修饰在宫颈癌放疗中的具体作用机制尚不明确。

材料与方法 收集经放射治疗的宫颈癌组织样本，根据肿瘤退缩情况及 RECIST 1.1 标准分为放疗敏感组和放疗抵抗组。对两组样本进行蛋白组学和琥珀酰化修饰分析，通过蛋白表达归一化处理后，利用高通量测序技术筛选出差异表达的琥珀酰化修饰位点。通过 KEGG 通路富集分析，确定与丙酮酸代谢相关。进一步利用分子动力学模拟和突变型质粒构建，研究 LDHB 琥珀酰化修饰对酶活性和蛋白结构的影响。构建模拟琥珀酰化和去琥珀酰化的慢病毒载体，通过 ELISA 检测乳酸和丙酮酸水平，评估琥珀酰化修饰对丙酮酸代谢的影响。最后，通过细胞和动物模型研究琥珀酰化修饰对宫颈癌放疗敏感性的影响及其作用机制。

结果 高通量测序结果显示，LDHB 的 K82、K319、K329 位点在放疗抵抗组织中高表达。KEGG 通路富集分析表明与丙酮酸代谢途径密切相关。分子动力学模拟和突变型质粒构建实验证实，琥珀酰化修饰显著影响 LDHB 的酶活性和蛋白结构，其中 K319 位点变化最明显。ELISA 检测显示，LDHB-K319 琥珀酰化修饰显著改变了乳酸和丙酮酸水平。细胞和动物模型实验表明，LDHB 琥珀酰化修饰显著降低了宫颈癌细胞对放疗的敏感性。

结果 本研究揭示了 LDHB K319 琥珀酰化修饰通过调控丙酮酸代谢，导致宫颈癌细胞对放疗的敏感性下降。这一发现为宫颈癌的代谢靶向治疗提供了新的策略，具有重要的临床应用前景。未来的研究将进一步探索 LDHB 琥珀酰化修饰在其他癌症中的作用机制，以及其作为潜在治疗靶点的可能性。

PO_017

基于影像组学的子宫颈神经内分泌癌远处转移风险评分的建立

余海娟 陈建 孙阳*
福建省肿瘤医院

背景 子宫颈神经内分泌癌（neuroendocrine carcinoma of the cervix，NECC）患者极易出现远处转移，即使是初诊时处于早期的患者仍可能发生广泛的血行转移而死亡。鉴于目前缺乏有效的远处转移预测工具，本研究旨在基于磁共振（magnetic resonance imaging，MRI）影像组学特征构建模型，预测 NECC 患者的无远处转移生存期（distant metastasis-free survival，DMFS），以尽早识别高转移风险的 NECC 患者，协助临床决策制定。

方法 回顾性纳入 2008 年 11 月至 2024 年 2 月福建省肿瘤医院有治疗前 MRI 影像信息的 NECC 患者。采用最小绝对收缩和选择算子（LASSO）对基于不同序列感兴趣区域提取的影像组学特征进行筛选，并计算影像组学评分（Rad-score）。通过单因素和多因素 Cox 回归分析确定 NECC 患者的临床危险因素，分别构建影像组学模型、临床模型及联合模型，以预测 NECC 患者的 DMFS。使用时间依赖性受试者工作特征曲线下面积（AUC）、一致性指数（C-index）、1000 次 bootstrap 重采样、校准曲线和临床决策曲线（DCA）评估模型性能。

结果 共纳入 NECC 患者 95 例，其中 44 例（46.3%）患者发生转移，转移患者的中位总生存时间为 25.0 个月（95%CI：20.90~29.08）。利用 LASSO 回归筛选出 7 个影像组学特征并计算 Rad-score。基于 Rad-score 与三个临床危险因素：年龄> 58 岁[HR（95% CI）：3.338（1.585~7.031），P＝0.002]、FIGO 分期 III 期[HR（95% CI）：3.869（1.694~8.820），P＝0.01]、全身免疫炎症指数＞800[HR（95% CI）：2.004（1.027~3.906），P＝0.042]构建的联合模型列线图预测效能较好，并优于影像组学模型与临床模型。联合模型预测 1、3、5 年 DMFS 的 AUC 值分别为 0.811（95% CI：0.717~0.906）、0.870（95% CI：0.790~0.950）、0.849（95% CI：0.759~0.938），经 1000 次 bootstrap 重采样验证后联合模型 C-index 大于 0.7。校准曲线和 DCA 曲线显示，该联合预测模型具有良好的预测准确性和临床应用价值。基于联合模型的风险分层可将患者分为高、低风险组。高风险组患者的 DMFS 显著较差（P＜0.01），接受≥4 个化疗周期的低风险组患者预后显著改善（P＜0.05）。

结论 本研究首次开发了一种结合影像组学特征和临床因素的列线图模型，能够有效预测 NECC 患者的 DMFS，为 NECC 患者的个体化治疗和随访策略提供风险分层依据。

PO_018

LIF enhances radioresistance in cervical cancer through DNA damage repair, induces ferroptosis, and reverses radioresistance via anti-PD-L1 therapy.

Meihui Gao[1] Yang Sun[1*] Huachun Song[2] Zhaolei Cui[1]
1.Fujian Provincial Cancer Hospital 2.Yiwu Central Hospital

Research Objectives Leukemia inhibitory factor (LIF), a multifunctional cytokine protein, has an unclear role in the carcinogenesis of cervical cancer. Radioresistance in cervical cancer is closely associated with poor prognosis in cancer patients. This study aims to investigate how LIF affects the radioresistance of cervical cancer through DNA damage repair mechanisms, explore the potential of LIF-induced ferroptosis in reversing radioresistance, and evaluate the impact of adding anti-PD-L1 therapy. By modulating the tumor immune microenvironment, this approach seeks to activate potent anti-tumor responses, ultimately paving the way for more effective treatment strategies against radioresistant cervical cancer.
Methods For cervical cancer clinical samples, the dose to cervical CTV-1 was maintained at a constant 180 cGy before radiotherapy. After 10 fractions of radiotherapy, the samples were subjected to transcriptome sequencing. A Siha radioresistant cell line was established by irradiating the cells with 2 Gy per fraction, twice a week, until a cumulative dose of 80 Gy was reached. Transcriptome sequencing was then performed on the radioresistant Siha cells and compared with that of non-irradiated Siha cells. The gene Leukemia Inhibitory Factor (LIF), which confers radioresistance and can induce ferroptosis, was identified by intersecting the data from both groups.
RNA sequencing combined with bioinformatics analysis was employed to elucidate the potential functions of LIF. Its biological effects were investigated through in vitro and in vivo experiments. Western blotting, quantitative real-time reverse transcription PCR (qRT-PCR), co-immunoprecipitation (Co-IP) assays, confocal microscopy, and flow cytometry were utilized to detect LIF expression and explore its interaction networks. The DNA damage mechanism was verified using the comet assay. Ferroptosis inducers and lipid peroxidation were applied to evaluate their relationship with radioresistance therapy. Additionally, anti-PD-L1 was introduced, and co-culture with macrophages followed by flow cytometry analysis was conducted. The efficacy of the treatment was validated through subcutaneous tumorigenesis experiments in nude mice.
Results LIF was found to be highly expressed in both cervical cancer tissues and radioresistant cells. This elevated LIF expression is associated with poor clinical outcomes in cervical cancer patients. Overexpression of LIF activates the PI3K/AKT/mTORC1/p70S6K signaling pathway, thereby promoting tumor growth and suppressing the DNA damage response. It also enhances radioresistance by augmenting the ATM-p53 pathway-mediated homologous recombination repair. Conversely, knocking down LIF or treating with the mTOR inhibitor rapamycin reverses the effects mediated by LIF, leading to growth arrest and increased sensitivity to γ-ray irradiation.
Furthermore, our study revealed that radioresistant cervical cancer cells exist in a fragile oxidative stress equilibrium, rendering them more susceptible to ferroptosis inducers. Intriguingly, we discovered that LIF-induced ferroptosis can reverse radioresistance. Through mass spectrometry, we identified GCLC as a downstream interacting protein of LIF. Treatment with Erastin suppresses the SCL7A11-GCLC-GSH-GPX4 axis, thereby inducing ferroptosis.
Notably, simultaneous inhibition of LIF and PD-L1 significantly potentiates the efficacy of radiotherapy. The triple combination therapy, consisting of a ferroptosis inducer, anti-LIF, and anti-PD-L1, generates an immunostimulatory tumor microenvironment. This environment is characterized by a pro-

inflammatory shift in the cytokine/chemokine profile, increased infiltration of effector CD8+ T cells, and upregulation of activation or maturation signals in tumor-infiltrating CD8+ T cells and macrophages.
Conclusion During the malignant progression of cervical cancer triggered by high LIF expression, inducing ferroptosis can effectively eliminate cancer cells, reverse the radioresistance of cervical cancer cells, and enhance the sensitivity to radiotherapy. This approach offers a potential treatment strategy for recurrent and refractory cervical cancer. The quadruple combination therapy, integrating a ferroptosis inducer, anti-LIF, anti-PD-L1, and radiotherapy, is conducive to the establishment of long-term systemic anti-tumor memory. It synergistically targets multiple key mechanisms in tumor progression, not only overcoming radioresistance but also promoting the activation of the immune system, thereby achieving more comprehensive and sustained anti-tumor effects.

PO_019

IRF4 modulates cervical cancer progression and enhances radiosensitivity by inhibiting the PI3K/Akt/mTOR pathway to regulate autophagy

Meihui Gao[1] Yang Sun[1*] Huachun Song[2] Zhaolei Cui[1]
1.Fujian Provincial Cancer Hospital 2.Yiwu Central Hospital

Research Objectives Interferon regulatory factor 4 (IRF4), a critical member of the IRF transcription factor family, harbors an elusive biological role in cervical cancer.
Methods IRF4 expression patterns in cervical cancer tissues and cell lines were systematically evaluated through immunohistochemical staining and immunoblotting. Cellular proliferation kinetics and radiosensitivity profiles were comprehensively assessed using CCK-8 viability assays, EdU incorporation tests, and clonogenic survival experiments. Apoptotic responses were quantitatively analyzed by flow cytometric detection of Annexin V/PI staining, while ultrastructural examination of autophagic vesicles was performed via transmission electron microscopy (TEM). The expression dynamics of autophagy-related proteins were further characterized through immunoblotting and immunofluorescence staining. To validate these findings in vivo, we established a HeLa cell-derived xenograft model in immunocompromised mice.
Results Notably, IRF4 expression was markedly decreased in cervical cancer tissues and cell lines compared to normal controls. Overexpression of IRF4 suppressed proliferation, migration, and invasion in both Siha and HeLa cells, while concurrently enhancing radiosensitivity. Mechanistically, IRF4 upregulated autophagy-related proteins (LC3, Beclin-1) and promoted autophagosome formation, while downregulating P62 by inhibiting the PI3K/Akt/mTOR pathway. In vivo studies demonstrated that IRF4 augmented the tumor response to radiation and further potentiated the effects when combined with rapamycin treatment, confirming its pivotal role in promoting radiosensitivity through PI3K/Akt/mTOR-mediated autophagy.
Conclusions IRF4 emerges as a critical regulator of cervical cancer progression via modulation of autophagy and influences the tumor's response to radiotherapy. It holds promise as a potential therapeutic target to enhance cervical cancer radiosensitivity.

PO_020

宫颈神经内分泌癌的分子特征

李安阳 孙阳*
福建省肿瘤医院

目的 神经内分泌癌是一类高度侵袭性且异质性强的恶性肿瘤，在人体不同部位均可发生，包括肺，胃肠，胰腺及宫颈等。神经内分泌癌的治疗以放化疗为主，五年生存率不理想。宫颈神经内分泌癌（Cervical neuroendocrine carcinoma CNEC）约占宫颈癌的 1–2%，目前没有标准的治疗方案，极易复发，初始复发以盆腔外复发为主，预后较差。近年来，蛋白翻译后修饰被发现在多种癌症的恶性表型的维持、转移潜能以及耐药性中发挥核心作用。深入研究 CNEC 的分子病理机制对于改善患者预后和开发新疗法至关重要，我们试图绘制其蛋白质及乳酸化修饰图谱，并确定 CNEC 的潜在治疗靶点，为 CNEC 的精准治疗提供理论依据。
方法 本研究使用了 2021 年 12 月 1 日至 2024 年 12 月 1 日间于福建省肿瘤医院诊治的 CNEC 患者的组织样本，提取其总蛋白并进行胰蛋白酶酶解。随后，采用乳酸化赖氨酸抗体富集修饰肽段，结合高分辨率液相色谱-串联质谱，进行定量蛋白质组和乳酸化修饰组分析。我们对鉴定数据进行了过滤，原始质谱数据通过 MaxQuant 平台鉴定和定量，通过 R 包进行修饰位点注释、功能富集、结构预测和 motif 分析。并采用定量逆转录-聚合酶链反应、蛋白印迹和免疫组织化学检测了 HMGB2 在 CNEC 和癌旁组织中的表达。
结果 本文绘制了 CNEC 患者的蛋白质及乳酸化图谱，并系统性的描绘了 CNEC 不同亚型的分子特征，发现了 HMGB2 的乳酸化修饰在 CNEC 中的关键调节作用。
结论 本研究针对 CNEC 进展快，易转移，易复发的科学问题，基于蛋白质组和乳酸化修饰组学测序技术，结合临床表型分析，揭示了相关的个性化分子特征。针对蛋白质翻译后修饰的药物已在其他肿瘤类型中获得进展，本研究为开发 CNEC 放化疗增敏剂提供了理论支持，为 CNEC 的诊治提供了新的诊断标志物与靶向治疗策略。

PO_021

KCNN4 Promotes the Radiotherapy Resistance in Cervival Cancer through Metabolic Reprogramming

Anyang Li Yang Sun*
Fujian Cancer Hospital

Objective Cervical cancer is one of the most common gynecologic malignancies, with an estimated 661,021 new cases and 348,189 deaths worldwide in 2022, nearly 60% of which occurred in Asia. Although early-stage cervical cancer patients can achieve a favorable prognosis through surgical treatment, some patients are already in locally advanced or advanced stages at the time of diagnosis. Radiotherapy resistance is a major cause of cervical cancer-related mortality. Therefore, there is an urgent need to elucidate the underlying molecular mechanisms of radiotherapy resistance in cervical cancer and explore novel therapeutic targets to improve radiotherapy efficacy. In recent years, research on ion channels as anticancer targets has been gradually deepening, particularly regarding the KCNN4 channel, which has been found to be abnormally expressed in various cancer. Ion channels and abnormal metabolism of tumor cells are also being investigated. The widespread dysregulation of metabolism and cellular energetics in cancer suggests that metabolic reprogramming plays a crucial role in tumor development and progression. However, the role and underlying mechanisms of KCNN4 in metabolic reprogramming in cervical cancer remain unclear and require further investigation.
Methods Clinical tissue samples were collected from newly diagnosed cervical cancer patients who underwent radical radiotherapy at Fujian Cancer Hospital. Patients were grouped based on MRI results

before and after ten sessions of radiotherapy, following the RECIST 1.1 criteria. RNA-seq transcriptome sequencing was performed, and the sequencing results were analyzed in conjunction with public databases (intersecting gene sets from TCGA-CESC analysis and ion channel-related gene sets from GeneCards, combined with survival data) to identify the target gene KCNN4. KCNN4 was knocked down in cervical cancer cell lines to explore its effect on proliferation and radiotherapy resistance. RNA sequencing combined with metabolome sequencing, quantitative real-time PCR, and western blotting analysis were performed to explore KCNN4 regulated downstream target genes. The biological effects of KCNN4 expression changes on cervical cancer cells were examined through in vitro functional assays. The alterations in lactate and ATP levels within and outside tumor cells, indicative of glycolytic activity, were assessed using colorimetric and chemiluminescence methods. Additionally, a xenograft mouse model was established to evaluate the mechanisms by which KCNN4 modulates radiotherapy resistance in cervical cancer cells.

Results We found that KCNN4 mRNA and protein levels were significantly upregulated in radiotherapy-resistant cervical cancer tissues as well as in HeLa and SiHa cell lines. KCNN4 knockdown suppressed proliferation, migration, invasion, and colony formation of cervical cancer cells. Mechanistically, KCNN4 modulated cell membrane potential, altering intracellular calcium ion concentration and subsequently activating downstream signaling pathways. Furthermore, KCNN4 contributed to metabolic reprogramming of cervical cancer cells by enhancing glycolytic activity, leading to radiotherapy resistance.

Conclusion KCNN4 is highly expressed in cervical cancer tissues, with a particularly pronounced upregulation in radiotherapy-resistant cases. We demonstrate that KCNN4 drives cervical cancer proliferation and enhances radiotherapy risistence through PI3K/AKT/AS160 pathway-mediated activation of the glycolysis, which might serve as a target for cervical cancer treatment. By increasing glycolysis, KCNN4 enhances the metabolic adaptability of cervical cancer cells, contributing to radiotherapy resistance. Therefore, KCNN4 represents a potential molecular target for overcoming radiotherapy resistance in cervical cancer.

PO_022

Development and Validation of an NK Cell-Associated Gene Signature for Prognostic Evaluation and Immunotherapy Guidance in Cervical Cancer

Wangshu Li Lan Yu*
Dalian Women and Children's Medical Group

Background Cervical cancer remains a major global health challenge, particularly in low- and middle-income countries, despite advances in screening and HPV vaccination. Natural Killer (NK) cells, as crucial components of the innate immune system, play a pivotal role in tumor surveillance and elimination. However, their functional impairment within the tumor microenvironment (TME) contributes to immune escape. This study aims to systematically identify NK cell-related genes (NKRGs) in cervical cancer and develop a robust prognostic signature to evaluate patient survival and predict response to immunotherapy.

Methods We integrated single-cell RNA sequencing (scRNA-seq) and bulk RNA-seq data to characterize NK cell infiltration and identify NKRGs in cervical cancer. scRNA-seq data from 66,178 immune cells (GSE197461) were analyzed using the Seurat package to determine NK cell clusters and marker genes. Bulk transcriptomic data from TCGA-CESC (n=299) and multiple external validation cohorts (GSE44001, GSE9750, GSE63514, GSE67522) were used to quantify NK cell infiltration via MCP-counter and construct an NK cell-related risk score (NKRS). WGCNA and GSVA were employed to identify NKRGs associated with immune checkpoint blockade (ICB) resistance. LASSO Cox regression was used to develop a prognostic gene signature based on NKRGs. Functional annotation, immune landscape analysis, and chemotherapy sensitivity prediction were conducted using GSEA,

ESTIMATE, CIBERSORT, and OncoPredict. Experimental validation included IHC analysis from the Human Protein Atlas and predictive performance was assessed with survival analysis and time-dependent ROC curves.
Results High NK cell infiltration was significantly associated with favorable prognosis across both TCGA and validation cohorts. A total of 380 NKRGs were identified via scRNA-seq, and WGCNA identified 373 genes linked to ICB resistance. From these, 8 prognostically relevant NKRGs were selected to construct the NKRS. Patients in the low NKRS group exhibited significantly improved overall survival (OS), disease-free survival (DFS), and progression-free survival (PFS). NKRS remained an independent prognostic factor in multivariate Cox analyses. Functional enrichment analysis revealed that low NKRS tumors were enriched in immune-related pathways, including NK cell-mediated cytotoxicity and antigen presentation. Low NKRS was also associated with higher immune scores, increased $CD8^+$ T cell and activated NK cell infiltration, and greater sensitivity to ICB therapy, as supported by TIDE and IPS analyses. Conversely, high NKRS patients exhibited lower chemosensitivity and were predicted to benefit from alternative small-molecule drugs. Among the 8 NKRGs, COTL1 and ALG14 emerged as key prognostic markers with strong correlation to NK cell presence and patient survival, as validated in external datasets and by IHC staining.
Conclusion This study presents a novel NK cell-derived gene signature (NKRS) with strong prognostic value and clinical relevance in cervical cancer. The NKRS model not only stratifies patient survival outcomes but also effectively predicts responsiveness to immunotherapy and chemotherapy. Furthermore, the identification of COTL1 and ALG14 as critical biomarkers provides potential targets for further mechanistic studies and therapeutic intervention. These findings highlight the clinical utility of NK cell-associated molecular profiling in advancing precision medicine and immunotherapeutic strategies in cervical cancer management.

PO_023

SDHA 琥珀酰化通过调控三羧酸循环影响宫颈癌放疗抵抗的分子机制研究

雷雪梅 孙阳*
福建省肿瘤医院

背景 琥珀酰化修饰作为一种重要的翻译后修饰，近年来在调控酶活性与代谢稳态中的作用日益受到关注。线粒体关键代谢酶琥珀酸脱氢酶 A（SDHA）琥珀酰化状态可能通过影响 TCA 循环功能，进而参与肿瘤细胞对放疗的响应。然而，其具体结构-功能机制及在宫颈癌放疗抵抗中的作用仍不明确。本研究旨在系统解析 SDHA 琥珀酰化修饰对酶活性的结构基础，并探讨其在宫颈癌放疗敏感性中的分子机制。
方法 为实现上述目标，本研究采用多学科交叉方法，结合高精度分子动力学模拟、实验验证与体内/外功能分析。首先，通过 GROMACS 进行模拟，对比野生型、K→E（模拟持续琥珀酰化）与 K→R（模拟去琥珀酰化修饰）突变体的构象动态差异。其次，在宫颈癌细胞模型中，构建 CRISPR-Cas9 敲除系并转染不同突变体质粒，通过分光光度法、 Seahorse 代谢仪等手段评估琥珀酰化对酶活性与 TCA 循环的影响。进一步地，建立体内外放疗模型，结合克隆形成、γ-H2AX 染色、移植瘤生长等指标，评估琥珀酰化对放疗敏感性的调节作用，并通过代谢干预与基因编辑揭示其下游通路（如 HIF-1α/NRF2）的关联机制。然后，整合 LC-MS/MS 代谢组学、RNA-seq 与 TCGA 数据库，挖掘琥珀酰化修饰相关的临床标志物与潜在治疗靶点。最后，利用患者源性类器官模型验证去琥珀酰化酶对宫颈癌放疗增敏性。
结果 研究预期揭示 SDHA 琥珀酰化修饰通过调控 TCA 代谢流与氧化应激水平，促进宫颈癌细胞放疗抵抗的新机制，并为开发基于去琥珀酰化酶（SIRT5）的放疗增敏策略提供理论依据。

PO_024

Study of Kielin/chordin protein in predicting chemotherapy resistance in cervical cancer

Chen Ji Yue He* Yumei Wu Yan Wang
Beijing Obstetrics and Gynecology Hospital, Beijing Maternal and Child Health Care Hospital, Capital Medical University

Objective This study aimed to find diagnostic threshold of KCP in predicting chemo-resistance in cervical cancer to paclitaxel.
Methods We used ELISA kit to test KCP concentration in serum and analyzed 22 patients who are resistant to paclitaxel-based chemotherapy and 62 patients who are sensitive to paclitaxel-based chemotherapy.By analyzing the relationship between serum KCP level and age, recurrence status, survival status, pathological conditions, tumor stage, chemotherapy administration route and other factors, the diagnostic cut-off value of KCP for predicting chemotherapy resistance of paclitaxel in locally advanced cervical cancer was found, and a nomogram was drawn by multivariate analysis to predict the sensitivity of cervical cancer patients to paclitaxel chemotherapy.
Results We found the diagnostic threshold of KCP in serum is 26.34 ng/L (sensitivity 88.9%, specificity 77.8%, Jorden index 88.9%). We found that independent risk factors influencing the risk of recurrence after neoadjuvant chemotherapy for paclitaxel in locally advanced cervical cancer included serum KCP levels, tumor markers (SCCA), whether the tumor shrank, how much the tumour shrunk, depth of interstitial invasion, lymph node metastasis, and degree of tumor differentiation, and constructed a predictive nomogram.
Conclusion This study aimed to find a biomarker to predict the resistance of locally advanced cervical cancer to paclitaxel-based chemotherapy. In further, we will expand the sample size and continue to search for the optimal diagnostic threshold.

PO_025

宫颈透明细胞癌预后预测模型的建立与验证

刘林英 陈丽君 林飞双 傅怡馨 杨琳 孙阳*
福建省肿瘤医院

目的 宫颈透明细胞癌（clear cell adenocarcinoma of the cervix, CCAC）是一种罕见但侵袭性极强的恶性肿瘤，预后较差。目前尚缺乏针对该病的个体化预后评估工具。本研究旨在利用 SEER 数据库构建并验证预测 CCAC 患者总生存（overall survival, OS）和癌症特异性生存（cancer-specific survival, CSS）的列线图（nomogram）及风险评分系统，以期为临床提供更精确的预后判断依据。
方法 本研究从 SEER 数据库中提取了 2000 年至 2019 年间确诊的 429 例 CCAC 患者资料，并随机分为训练集和验证集。采用 Cox 比例风险回归模型筛选与 OS 和 CSS 显著相关的独立预后因素。基于这些因素构建预测 1 年、3 年及 5 年 OS 和 CSS 的列线图模型。通过受试者工作特征曲线下面积（AUC）、校准曲线以及决策曲线分析（DCA）评估模型的判别力、准确性和临床适用性。并通过 C 指数、净重分类指数（NRI）和集成判别改善指数（IDI）与 2018 年 FIGO 分期系统进行比较。根据预测得分将患者分为低风险组和高风险组，绘制 Kaplan-Meier 生存曲线以评估风险分层效果。
结果 多因素 Cox 回归分析显示，诊断年龄、肿瘤大小、是否接受手术为 OS 和 CSS 的共同独立预后因素，而放疗和化疗对 OS 和 CSS 具有特异性影响。构建的 OS 与 CSS 列线图在训练集中的 C 指数分别为 0.83 和 0.84，1 年、3 年和 5 年的 AUC 均分别达到 0.95、0.95 和 0.88，在验证集中也表现出类似的预测性能。校准曲线显示模型预测值与实际观察结果高度一致。DCA、NRI 和 IDI 均表明本研究所构建的列线图优于 2018 年 FIGO 分期系统。风险分层分析显示，高风险组患者的 OS 和 CSS 明显低于低风险组，具有良好的临床区分能力。

结论 本研究基于 SEER 数据库成功构建并验证了可预测 CCAC 患者 OS 与 CSS 的列线图模型。该模型较传统 FIGO 分期系统具有更高的预测准确性和临床实用价值，可为 CCAC 患者的个体化治疗和临床决策提供有力支持。

PO_026

多组学联合分析鉴定宫颈癌新抗原表位

王璐瑶[1,2] 李科珍[1,2*]
1.华中科技大学 2.华中科技大学同济医学院附属同济医院

目的 鉴定宫颈癌中具有潜力肿瘤新抗原疫苗。
方法 利用 TCGA 数据库中宫颈癌样本的 WES，RNA-seq 数据，并结合 HPA 数据库，进行了综合肿瘤基因组、转录组和蛋白质组生信分析。利用在线工具进行了抗原肽序列的预测，并在动物模型中验证抗原肽的免疫原性。
结果 本研究鉴定了宫颈癌的高频突变基因，评估了宫颈癌样本的免疫细胞浸润水平，并联合上述两种数据检查了与免疫浸润明显相关的高频突变基因作为候选基因。并在泛癌水平分析候选基因蛋白水平表达。最终鉴定了 PCLO 基因突变在宫颈癌，肺癌，甲状腺癌，卵巢癌，乳腺癌中均是具有潜力的肿瘤抗原。靶向 PCLO 突变位点的抗原肽具有免疫原性。
结论 靶向 PCLO 突变的抗原肽是具有潜力的肿瘤疫苗靶点。

PO_027

骨髓保护容积旋转调强放疗在宫颈癌患者同步放化疗中的应用研究

郭明芳*
重庆大学附属肿瘤医院

目的 骨髓保护容积旋转调强放疗（bone marrow sparing-volumetric modulated arc therapy, BMS-VMAT）在宫颈癌患者中的应用报道比较少。而相关 BMS-VMAT 对宫颈癌同步放化疗（CCRT）患者淋巴细胞的保护作用及临床疗效的研究还存在空白。该研究探讨 BMS-VMAT 在宫颈癌 CCRT 中在剂量学、血液学毒性（HT）和总体临床疗效方面的潜在益处。
材料与方法 共纳入 2024 年 10 月 8 日-2O25 年 03 月 31 日我院收治的宫颈癌同步放化疗患者 122 例，其中 60 例患者采用骨髓保护的容积旋转调强放疗(BMS-VMAT 组)，62 例患者采用未行骨髓保护的容积旋转调强放疗(VMAT(N)组)。比较两组患者放疗计划的剂量学参数，同步放化疗过程中的急性血液学反应（包括反映骨髓抑制的白细胞、中性粒细胞、血红蛋白、血小板及反映免疫效应的淋巴细胞），以及最终疗效。
结果 两组放疗计划的靶区剂量学参数（最大剂量 Dmax、最小剂量 Dmin、平均剂量 Dmean、适形度 CI、均匀性 HI）都无统计学差异（P＞0.05）。危及器官的比较上，BMS-VMAT 计划中骨盆骨髓的 V10、V20、V40 均低于 VMAT(N)计划，t 值分别为-19.879、-7.444、-2.576，P 值均＜0.01。膀胱、直肠、肠袋、股骨头的剂量学比较无统计学上的显著差异（P=0.715、0.293、0.739、0.565）。BMS-VMAT 组患者 2 级及以上总的骨髓抑制发生率低于 VMAT(N)组（P=0.014），在贫血及白细胞、中性粒细胞、血小板减少症上，BMS-VMAT 组患者发生严重程度轻于 VMAT(N)组患者（P=0.028、0.012、0.031、0.029）；BMS-VMAT 组患者同步放化疗期间最低淋巴细胞计数、放化疗后淋巴细胞计数均高于 VMAT(N)组，差异有统计学意义（t=2.407、2.591，P=0.019、0.012）。两组患者的完全缓解率分别为 66.67％和 48.39％，BMS-VMAT 组患者有更高的完全缓解率（P=0.021）。

结果 在宫颈癌患者同步放化疗中，采用骨髓保护容积旋转调强放疗能够在保证靶区剂量及危及器官限量满足下，更好保护患者的骨髓造血功能，患者的骨髓抑制发生严重程度更低，淋巴细胞也能有更好地保护，从而保障了更好的临床疗效。BMS-VMAT 是宫颈癌同步放化疗中的更好选择，具有很好的应用前景。

PO_028

Lumican 表达与宫颈癌中的血管生成拟态形成有关

胡格 吴晓维 朱凤琴*
中国科学院合肥肿瘤医院

目的 寻找宫颈癌中 Lumican 的表达水平与宫颈癌血管生成拟态的相关性。
方法 收集 2023 年 1 月至 2024 年 6 月期间中国科学院合肥肿瘤医院 65 份石蜡包埋的宫颈标本，其中宫颈癌标本 30 例、HSIL 标本 10 例、LSIL 标本 5 例和正常宫颈标本 20 例。免疫组化分析 65 个宫颈组织中 Lumican 蛋白的表达，CD31 和 PAS 双染明确血管生成拟态的形成，进一步分析 Lumican 蛋白及血管生成拟态与宫颈癌临床病理特征的相关性。
结果 65 例宫颈组织中有 73.85%（48/65）表现出 Lumican 蛋白阳性表达。正常宫颈组织和宫颈上皮内瘤变的阳性表达率分别为 100%（20/20）和 100%（15/15）。30 例宫颈癌组织中 13 例 Lumican 蛋白表达阳性。Lumican 的免疫染色强度从正常宫颈到宫颈上皮内瘤变，随后到宫颈癌组织明显下降。正常宫颈组织及上皮内瘤变的宫颈组织中未观察到血管生成拟态的形成，仅在宫颈癌样本中观察到血管生成拟态的形成，阳性率为 46.67%（14/30）。Lumican 表达与淋巴结转移（P=0.026）和 SCC-Ag（P=0.030）之间存在显著相关性。血管生成拟态的形成与 T 分期（P=0.0003）、淋巴结转移（P=0.029）和 SCC-Ag（P=0.026）显著相关。进一步的相关性分析显示血管生成拟态形成与 Lumican 表达呈正相关（r=0.284，P=0.013）。
结论 Lumican 表达可能通过血管生成拟态影响肿瘤进展。

PO_029

基于 CT 影像引导的不同施源器在大体积宫颈癌近距离治疗中的回顾性剂量与疗效分析

张娜*
重庆大学附属肿瘤医院

研究目的 宫颈癌患者中约 40%初诊时因靶区体积大、邻近器官受量高，近距离治疗面临剂量覆盖不足与毒性控制难题。本研究旨在探讨 4 种施源器技术在近距离治疗中的剂量学差异、安全性及疗效。
材料与方法 收集 2020 年 01 月至 2023 年 12 月 FIGO 分期 IIB-IVA 期宫颈鳞癌、腺癌或腺鳞癌共 100 例。病灶直径>3cm（MRI 测量）。根据施源器类型分为 4 组：卵圆体组、环形组、徒手插植组、3D 模板组，每组 25 例。外照射：固定野或旋转调强放疗，范围：盆腔野±腹主动脉旁野±腹股沟野 PTV 45 Gy/25F（1.8Gy/F），每日 1 次，每周 5 次；转移淋巴结总剂量至 65Gy。放疗期间同步顺铂 40 mg/m²/周（共 5-6 周）。25 次外照射结束后开始近距离治疗，6Gy/次，共 5 次（总剂量 40Gy，EQD2），每周 1-2 次。主要终点：剂量学：D90、V100、适形度指数（CI=TV_{prv}/TV）、均匀性指数（HI=D90/D10）。安全性：急性（≤90 天）与慢性（>90 天）毒性，重点记录≥2 级直肠/膀胱毒性。疗效：18 个月局部控制率。统计**方法** SPSS26.0 进行 KW 检验，KM 法计算局部控制率，Log-rank 检验比较组间差异（P<0.05 显著）。多因素 Cox 回归分析变量：年龄（≤50 岁 vs>50 岁）、肿瘤直径（3-5cm vs>5cm）、HR-CTV D90（≥87 Gy vs <87 Gy）、施源器类型。

结果 HR-CTV D90 在卵圆体组、环形组、徒手插植组和 3D 模板组分别为 83.2±2.4Gy，85.5±1.8Gy，86.8±1.6Gy，91.0±1.2Gy（P<0.001）；V100 分别为 89.0±3.6%，92.3±2.8%，93.9±2.1%，99.0±0.4%（P<0.001）；CI 分别为 0.66±0.06，0.72±0.05，0.78±0.04，0.89±0.03（P=0.002）。HI 分别为 0.55±0.09，0.40±0.06，0.48±0.08，0.41±0.05（P=0.05）。直肠 D2cc 分别为 65.8±4.4Gy，59.0±3.0Gy，64.2±4.0Gy，61.5±3.5Gy（P=0.03）；≥2 级慢性直肠毒性分别为 20.0%，8.0%，16.0%和 12.0%（P=0.04）。18 个月局部控制率分别为 80.0%，84.0%，88.0%和 96.0%（P=0.01）。复发风险因素：D90<87 Gy（P=0.02）；肿瘤直径>5 cm（P=0.07）；卵圆体组（P=0.003）；徒手组（P=0.06）。

结果 3D 打印模板插植在宫颈癌大肿瘤中实现最佳剂量覆盖与疗效，推荐作为中心型大体积肿瘤的首选；徒手插植需严格影像引导以减少剂量偏移；环形施源器在直肠保护上无可替代，适用于既往肠病史患者；传统卵圆体因靶区覆盖不足与高毒性风险，复发风险显著增高，慎用于＞3cm 病灶。

PO_030

深部热疗联合放化疗时序对局部晚期宫颈癌疗效与安全性的前瞻性随机对照研究

张娜*
重庆大学附属肿瘤 医院

研究目的 探讨在局部晚期宫颈癌同步放化疗（CCRT）基础上，不同时序（放疗前、放疗中、放疗后）联合深部热疗对近期疗效、远期生存、急性毒性反应的影响，旨在明确最佳联合时序。

材料与方法 2020 年 08 月至 2023 年 12 月期间，经病理确诊为宫颈鳞癌或腺鳞癌，FIGO 分期（2018）IIB-IIIB 期，初治。采用区组随机化方法，按 1:1:1 将 60 例患者随机分为三组，每组 20 例。基础治疗：外照射放疗（VMAT 技术），计划靶区剂量 45-50.4 Gy/25-28 次；近距离放疗，A 点剂量 30-35 Gy/5-6 次。总治疗时间控制在 56 天内。外照射期间同步顺铂周疗，剂量 40 mg/m²，共 5-6 个周期。热疗（分组变量）：采用 BSD-2000 深部热疗系统（频率 70-120 MHz），目标瘤区温度 41-43℃，每次 60 分钟，每周 2 次，共 10 次。A 组（热疗前）：在当日放疗开始前 2 小时内完成热疗；B 组（热疗中）：在当日放疗结束后 30 分钟内开始热疗；C 组（热疗后）：在当日放疗结束后 24 小时进行热疗。主要终点：客观缓解率（ORR）；次要终点：2 年无进展生存率（PFS）。急性毒性反应：重点观察≥3 级骨髓抑制、胃肠道反应、放射性直肠炎、放射性膀胱炎。

结果 三组患者在年龄、FIGO 分期、病理类型、基础治疗剂量等基线资料上均衡可比。治疗结束后 3 个月 ORR：A 组 85.0% (17/20), B 组 95.0% (19/20), C 组 75.0% (15/20)。B 组显著高于 C 组（P=0.044），B 组与 A 组、A 组与 C 组间差异无统计学意义（P> 0.05），但三组总体比较差异显著（P=0.032）。2 年 PFS：A 组 78.9%, B 组 89.5%, C 组 68.4%。B 组显著高于 C 组（P=0.025），三组总体比较差异显著（P=0.047）。急性毒性反应（≥3 级）：白细胞减少：A 组 25.0%(5/20),B 组 30.0%(6/20), C 组 45.0%(9/20)。C 组发生率高于 A 组（P=0.049）和 B 组（P=0.041），三组总体比较差异显著（P=0.041）。放射性直肠炎和急性膀胱炎组间差异无统计学意义（P>0.05）。

结论 深部热疗与放疗同步实施（放疗结束后 30 分钟内开始，B 组）是局部晚期宫颈癌 CCRT 的最优联合时序。该方案提高了 ORR 和 2 年 PFS，可能与热疗实时增强放疗对肿瘤细胞的 DNA 损伤敏感性、改善肿瘤局部缺氧微环境密切相关。将热疗置于放疗结束后 24 小时（C 组）会显著增加≥3 级白细胞减少症的发生风险（45%），提示该时序可能干扰骨髓造血功能的恢复。推荐将深部热疗与放疗同步进行（间隔控制在放疗结束后 30 分钟内）作为局部晚期宫颈癌患者 CCRT 的标准优化时序策略。对于采用放疗后热疗时序（C 组）的患者，应制定严格的骨髓功能监测计划，积极预防和处理骨髓抑制，平衡疗效与安全性。

PO_031

A comprehensive study based on large samples and multi-omics integration to explore the potential biological significance of ZWINT : From Pan-Squamous Carcinoma to Cervical Squamous Carcinoma

Yiyang Chen Zhisu Liu Xianjin Chen Haiquan Wang Yunxiang Zhang Hanjun Zhang Siyuan Chen Zhendong Yang Mei Wu Gang Chen Qi Li*
The First Affiliated Hospital of Guangxi Medical University

Objective To study the expression of ZWINT in cervical squamous carcinoma (CESC), lung squamous carcinoma (LUSC), laryngeal squamous carcinoma (LSCC), oral squamous carcinoma (OSCC), and esophageal squamous carcinoma (ESCC), and to explore the potential biological functions of ZWINT over-expression in cervical squamous carcinoma.
Methods A large-scale global multi-center clinical sample dataset (n=6645) was used to assess the ZWINT mRNA expression levels in pan-squamous carcinomas. Internal samples (n=126) were collected for immunohistochemistry (IHC) experiments to validate the ZWINT protein levels in cervical squamous carcinoma. CRISPR screening data was employed to examine the impact of ZWINT knockout on the growth of pan-squamous carcinoma cell lines. Single-cell RNA sequencing (scRNA-seq) analysis was further conducted to assess the expression of ZWINT. The effect of ZWINT expression on the tumor microenvironment (TME) was explored using the CIBERSORT algorithm and TISIDB. Highly expressed co-expression genes (HECEGs) of ZWINT were used to investigate potential signaling pathways. Additionally, drug sensitivity analysis of ZWINT to predict potential drugs targeting ZWINT, and use molecular docking to evaluate the binding ability of drugs to ZWINT.
Results ZWINT mRNA was significantly overexpressed in cervical squamous carcinoma (SMD=2.69, 95% CI [1.45; 3.94]), lung squamous carcinoma (SMD=3.08, 95% CI [2.44; 3.73]), laryngeal squamous carcinoma (SMD=1.08, 95% CI [0.70; 1.46]), oral squamous carcinoma (SMD=1.16, 95% CI [0.73; 1.59]). Additionally, immunohistochemical experiments on 62 pairs of cervical squamous cell carcinoma tissues and adjacent normal tissues confirmed that ZWINT protein is significantly overexpressed in CESC, and can significantly distinguish between cancerous and non-cancerous tissues in CESC (AUC-0.979). The upregulation of ZWINT mRNA exhibited high discriminatory ability between cervical squamous carcinoma samples (AUC=0.98 [0.96–0.99]), lung squamous carcinoma samples (AUC=0.99 [0.97–0.99]), laryngeal squamous carcinoma samples (AUC=0.91 [0.88–0.93]), oral squamous carcinoma samples (AUC=0.91 [0.88–0.93]), esophageal squamous carcinoma samples (AUC=0.90 [0.87–0.92]), and their respective control samples. ZWINT knockout suppressed the growth of nearly 100 pan-squamous carcinoma cell lines. Additionally, scRNA-seq analysis showed that ZWINT expression predominantly accumulated in NK cells, T cells, and epithelial cells in CESC. TME analysis showed a significant negative correlation between ZWINT expression and the infiltration levels of various immune cells, including NK cells (rho = -0.237, p = 2.94e-05). The majority of ZWINT HECEGs were enriched in signaling pathways such as the cell division, DNA repair, DNA replication, chromosome segregation. The drug sensitivity analysis indicated that the drug activities and sensitivities of KPT-9274, BAY-1816032, Okadaic acid, Pyrazoloacridine, RX-3117, and Sonolisib were significantly correlated with ZWINT expression levels. Molecular docking demonstrated strong binding affinities of ZWINT with KPT-9274 (−10.5 kcal/mol), Okadaic acid (−9.8 kcal/mol), BAY-1816032 (−8.3 kcal/mol), Pyrazoloacridine (−8.5 kcal/mol), RX-3117 (−7.7 kcal/mol), and Sonolisib (−7.1 kcal/mol).
Conclusions ZWINT is significantly ove-rexpressed in pan-squamous carcinomas, including CESC. High ZWINT expression promotes the growth of pan-squamous carcinoma cells. In CESC, high ZWINT expression is negatively correlated with the infiltration levels of various immune cells and may promote tumor progression through its effects on the TME. Molecular docking revealed that ZWINT exhibits

strong binding interactions with KPT-9274, Okadaic acid, BAY-1816032, Pyrazoloacridine, RX-3117 and Sonolisib.

PO_032

Epithelial Reprogramming of Fibroblasts Drives the Progression of Gastric-type Cervical Adenocarcinoma

Ting Peng Peng Wu*
Union Hospital, Tongji Medical College, Huazhong University of Science and Technology

Background Gastric-type adenocarcinoma (GAC) of the cervix is a highly invasive and intrinsically therapy-resistant malignancy. However, due to its rarity, evidence-based treatment strategies remain lacking. Unraveling the stromal interactions and oncogenic signaling underlying GAC is essential for the development of effective precision therapies.
Methods We conducted an integrated analysis of real-world data from a Chinese multi-institutional registry and the U.S. SEER database to evaluate the impact of treatment modalities and prognostic factors on overall survival in GAC. Survival outcomes were assessed using Kaplan-Meier analysis, Cox proportional hazards models, and propensity score matching. In parallel, we utilized single-cell RNA sequencing of cervical cancer specimens, organoid models, and molecular biology assays to dissect the tumor microenvironment (TME) and identify actionable therapeutic targets.
Results Our analysis demonstrated that surgical resection, particularly when combined with lymph node dissection, confers a significant long-term survival benefit for patients with advanced GAC. Multi-omic profiling revealed that GAC exhibits a malignant phenotype characterized by low proliferative activity, high invasiveness, and pronounced therapeutic resistance. Mechanistically, GAC cells reprogram cancer-associated fibroblasts (CAFs) via LAMA3-mediated activation of CD44/ITGB1-dependent FAK signaling, leading to extracellular matrix (ECM) remodeling and an immunosuppressive TME. Based on these findings, we launched a prospective, open-label, single-arm, multi-center clinical trial to evaluate the safety and efficacy of the FAK inhibitor IN10018 in combination with nab-paclitaxel and cadonilimab in women with metastatic, recurrent, or persistent GAC (ClinicalTrials.gov: NCT06654011).
Conclusion Our study highlights the critical role of surgery in improving outcomes for patients with advanced GAC and identifies CAF-targeting through FAK inhibition as a promising therapeutic avenue. These findings provide a rationale for integrating surgery and microenvironment-directed therapy in future treatment paradigms for this lethal cervical cancer subtype.

PO_033

一种新型 3D 打印自由组装插植模板用于局部晚期宫颈癌组织间插植近距离放疗的剂量学研究

朱杰
四川省肿瘤医院

研究目的 3D 打印自由组装插植模板（free-assembled interstitial template, FAIT）是一种融合宫腔管与径向优化非共面插植针于一体的自由组装插植模板，采用生物相容材料突破传统施源器限制，通过 0°-15°可调斜行插植通道提高宫旁剂量覆盖。本研究目的在于评价局部晚期宫颈癌组织间插植近距离放疗中，FAIT 辅助的组织间插植放疗的剂量学特点；并对比徒手插植的剂量学差异。
材料与方法 回顾性分析四川省肿瘤医院 2025 年 2 月至 2025 年 6 月接受超声引导、全身麻醉下 FAIT 辅助的组织间插植放疗的局部晚期（IIB-IVA 期，FIGO 2018 分期）宫颈癌患者。从 Oncentra 系统中

提取治疗计划参数，包括:靶区剂量参数（HRCTV D90）和危及器官（OARs）受照射剂量参数（D2cc），数据采用平均数±标准差表达。对于同期先后行 FAIT 辅助的插植、徒手插植的患者，采用配对 t 检验分析，$P < 0.05$ 提示具有统计学差异。

结果 32 例局部晚期宫颈癌患者接受超声引导、全身麻醉下 FAIT 辅助的组织间插植放疗（处方剂量为 600 cGy/f），平均插植针数量（含宫腔管）6 ± 1 根，HRCTV D90 平均剂量 618.1 ± 25.1 cGy，膀胱 D2cc、直肠 D2cc、乙状结肠 D2cc、小肠 D2cc 平均剂量分别为 426.7 ± 24.3、377.5 ± 43.8、298.7 ± 71.1、317.3 ± 70.0 cGy（图 1）。其中 14 例患者同时期先后行超声引导下徒手插植，平均插植针数量（含宫腔管）5.6 ± 1.6 根，HRCTV D90 平均剂量 589.0 ± 47.3 cGy，膀胱 D2cc、直肠 D2cc、乙状结肠 D2cc、小肠 D2cc 平均剂量分别为 437.8 ± 22.1、380.6 ± 44.7、319.2 ± 73.7、336.7 ± 73.3 cGy（图 1）。FAIT 辅助插植与徒手插植相比较，插植针数量（$P = 0.27$）、HRCTV D90（$P = 0.08$）、膀胱 D2cc（$P = 0.18$）、直肠 D2cc（$P = 0.99$）、乙状结肠 D2cc（$P = 0.18$）、小肠 D2cc（$P = 0.08$）均无显著性差异。

结论 FAIT 辅助的插植靶区剂量覆盖满意，靶区剂量覆盖展现出优于徒手插植的统计学倾向；OARs 剂量符合要求。进一步扩大研究样本量后，有望取得更确切的结论。

PO_034

IL-6 在宫颈病变进展过程中的作用研究

Zhang Ling[1,2]
1.沧州市中心医院
2.天津市中心妇产科医院

目的 宫颈癌（cervical cancer，CC）是常见的严重威胁女性健康的生殖系统恶性肿瘤之一，宫颈上皮内瘤变(cervical intraepithelial neoplasia, CIN)是与宫颈浸润癌密切相关的一组子宫颈病变，分为 CIN1、CIN2、CIN3 三级。大部分子宫颈低级别鳞状上皮内病变(low-grade squamous intraepithelial lesions, LSIL)即 CIN1 和部分 CIN2 可自然消退，但高级别鳞状上皮内病变(high-grade squamous intraepithelial lesions, HSIL)即部分 CIN2 和 CIN3 具有癌变潜能。高危型人乳头瘤病毒（High risk human papillomavirus，HR-HPV）持续感染是宫颈癌及宫颈病变的主要致病原因，但从 HR-HPV 感染到宫颈病变的发生是一个漫长的过程，机体免疫反应在这个过程中具有重要作用。细胞因子通过与特异性受体结合发挥免疫调节作用，本研究旨在探索多效应细胞因子 IL-6 在 HR-HPV 持续感染宫颈病变发生、发展过程中的作用。

方法 选取 2015 年 11 月-2016 年 4 月期间就诊于天津市中心妇产科医院阴道镜门诊的 105 例 HR-HPV 持续感染者，按照宫颈活检病理结果分为 3 组： NSIL、LSIL、HSIL 各 35 例。用流式 BD CBA 方法检测阴道灌洗液 7 种细胞因子水平（包括 IL-2、IL-4、IL-6、IL-10、TNF、IFN-γ、IL-17A）。用 RT-PCR 检测 ECT-E6/E7 细胞和 SiHa 细胞中 IL-6 及其受体 IL-6R 的 mRNA 表达水平。

结果 IL-6 表达水平随病变程度加重浓度逐渐升高（$P<0.05$），IL-2 随病变程度加重浓度逐渐降低（$P<0.05$），余 5 种细胞因子在三组间表达水平无明显差异（$P>0.05$）。SiHa 细胞的 IL-6 mRNA 表达水平为 ECT-E6/E7 细胞的 1.2 倍。SiHa 细胞 IL-6R mRNA 为 ECT-E6/E7 细胞的 3.8 倍。结论 IL-6 及其受体表达水平的升高与宫颈病变的进展密切相关。

PO_035

RPL26 介导长链非编码 RNA RAB11B-AS1 抑制人类宫颈癌进展

顾雪敏
上海市同济医院

研究目的 宫颈癌作为女性常见恶性肿瘤，严重威胁妇女健康，长链非编码 RNA 可通过调控基因表达参与肿瘤的发生发展，RAB11B-AS1 在不同恶性肿瘤中被证实发挥差异性作用，但其在宫颈癌中的具体作用及相关机制尚未明确。因此，本研究从细胞水平和分子水平入手，探索 RAB11B-AS1 在宫颈癌中的表达特征、生物学功能及其潜在调控机制，为阐明宫颈癌的发病机制提供新的理论依据，并为宫颈癌的临床诊断与治疗靶点筛选奠定基础。
材料与方法 选择人类宫颈癌石蜡包埋组织芯片，利用 RNA 原位杂交技术检测 RAB11B-AS1 的表达。选择人类宫颈癌细胞（SiHa）作为研究对象，利用基因转染质粒和慢病毒感染等技术构建稳定过表达和稳定敲低表达的细胞系，通过一系列分子生物学技术，包括染色质免疫共沉淀测序，转录组测序和 TMT 定量蛋白质组测序技术解析相关的功能和信号通路。同时，进行体内外细胞功能实验，设计多种回复实验验证相关结果。
结果 宫颈癌组织中 RAB11B-AS1 呈高表达，且 RAB11B-AS1 的表达与宫颈癌病理分级呈负相关。RAB11B-AS1 能抑制人宫颈癌 SiHa 细胞的生长，通过增加 G1/G0 期细胞、减少 S 期细胞来改变细胞周期，同时促进 SiHa 细胞凋亡，并抑制其迁移侵袭能力。RAB11B-AS1 在转录水平上调 RPL26 表达，并通过调控 RPL26、MAP2 的上调及 S100A14、EML2、CRABP2、TMEM168 的下调来改变癌症相关基因表达。此外，RAB11B-AS1 能调控核糖体蛋白通路、细胞周期通路及肿瘤转移信号通路等癌症相关通路。敲低 RPL26 可消除 RAB11B-AS1 的抗癌作用。
结果 核糖体蛋白 RPL26 是 RAB11B-AS1 在宫颈癌中发挥作用的关键枢纽。RAB11B-AS1 通过增加核糖体蛋白 RPL26 的表达，改变细胞周期、促进细胞凋亡、抑制细胞迁移和侵袭能力，发挥抑制宫颈癌的作用。

PO_036

DRG 支付系统下宫颈癌的经济负担分析

段拽林 赵宏伟* 闫丽隽
山西省肿瘤医院

目的 本研究旨在阐明 DRG 支付系统下宫颈癌前期病变与宫颈癌的经济负担，为优化山西省宫颈癌防治资源分配和策略提供基线数据。
方法 通过回顾性分析 2022 年 5 月至 2023 年 5 月在山西省肿瘤医院住院且出院时按 DRG 结算的 2320 例患者共 3868 次住院的宫颈癌前期病变及宫颈癌患者住院费用，按照以下三方面将患者进行分层和分组：1、按疾病的期别分五个组：宫颈癌前期病变组、宫颈癌（FIGOI-Ⅳ期）组；2、按治疗维度分为三个组：初始治疗、复发治疗、并发症治疗三个组；3、按住院原因分为两组：早筛组和有临床症状组。分析计算各组患者的人均住院天数、人均住院总费用、人均 DRG 支付费用、人均实际支付费用和人均社会其他方面四项经济负担指标的差异。
结果 宫颈癌与癌前病变相比，人均住院时间明显延长（30.98 天 VS 8.05 天，P<0.001)。宫颈癌初始治疗人均总费用为 39068.68 元是癌前期病变 15997.77 元的 2.4 倍（P<0.001），其他经济负担指标在宫颈癌组也明显高于癌前期病变组（P<0.001），随着疾病的进展宫颈癌费用呈上升趋势。因并发症治疗的平均住院时长为 11.69 天，人均总费用为 12185.59 元。因复发治疗住院的平均住院时间为 37.02 天，人均总费用为 30404.19 元。早筛组各项经济指标均少于临床症状组（P<0.001）。

结论 尽管采取了成本控制措施DRG支付系统，但是宫颈癌患者仍然面临巨大的经济压力。疾病进展与住院时间和人均治疗费用呈正相关，发生并发症和复发时加重了宫颈癌经济负担。通过早期筛查可以减轻宫颈癌患者经济负担。

PO_037

High P63 expression associated with better survival outcome in FIGO stage IIIC1p cervical cancer

Jiao Wang Ming Wang Jianqing Xu Yumei Wu*
Beijing Obstetrics and Gynecology Hospital, Capital Medical University

Objective P63 is integral to the maintenance of epidermal proliferation and differentiation. We aim to determine the impact of P63 expression on prognosis in patients with stage IIIC1p cervical cancer.
Methods Patients with FIGO 2018 stage IIIC1p cervical cancer were retrospectively included at our institute. The patients were enrolled into group A (high P63 expression) and group B (low P63 expression) based on their expression. The primary outcomes are overall survival (OS) and progression-free survival (PFS) between the two groups.
Results Of 155 patients included, 57(36.8%) patients were enrolled in group A and 98(63.2%) in group B. The expression of CK5, P16, and Ki-67 scores was lower in group B than in group A. No difference was found in the rates of histology type, tumor size, deep myometrial invasion, positive LVSI, positive surgical margin, parametrial involvement, vaginal involvement, and paraaortic lymph node metastasis. Compared to group A, patients in group B have a lower rate of 5-year PFS (78.86% vs. 92.79%, p=0.0095) and OS (75.03% vs. 92.86%, p=0.033). Patients of group B have more non-pelvic recurrences than patients of group A (14.43% vs. 1.78%, p=0.011).
Conclusion High P63 expression was associated with a favorable prognosis and fewer recurrences in patients with FIGO IIIC1p cervical cancer.

PO_038

Analysis of risk factors for pathological escalation in postoperative conization among postmenopausal patients with high-grade squamous intraepithelial lesion

Yun Feng*
The First Affiliated Hospital of Zhengzhou University

Objectives To investigate the incidence and risk factors for cervical invasive carcinoma after cold knife conization in postmenopausal patients with high-grade squamous intraepithelial lesion(HSIL).
Material and methods The clinical data of postmenopausal patients with newly diagnosed HSIL admitted to the First Affiliated Hospital of Zhengzhou University from January 2010 to September 2020 were retrospectively analyzed, and the incidence of pathological upgrading to cervical invasive carcinoma after cold knife conization and related influencing factors were analyzed.
Results A total of 490 postmenopausal patients with HSIL were included in this study, of whom 343 initially underwent conization, and 20 (5.83%, 20/343) were diagnosed with cervical invasive carcinoma after surgery. The univariate analysis showed that age ≥ 60 years, ≥ 3 births, HSIL indicated by thinprep cytology test (TCT), and transformation zone type III were significantly correlated with pathological escalation after conization ($P < 0.05$). The multivariate analysis results showed that HSIL indicated by TCT and transformation zone type III were independent risk factors for pathological escalation after conization ($P < 0.05$).

Conclusions Conization is the preferred treatment for postmenopausal patients with HSIL. Patients with HSIL indicated by TCT or transformation zone type 3 should be carefully selected for direct extrafascial hysterectomy as the initial treatment.

PO_039

LIMK1 promotes the development of cervical cancer by up-regulating the ROS/Src-FAK/cofilin signaling pathway

Yakun Liu[1,2*]
1.河北医科大学第四医院 2.河北医科大学第四医院

Objective In this study, we investigated the mechanism of action of LIMK1 in cervical cancer progression.
Methods The biological role of LIMK1 in regulating the growth, invasion, and metastasis of cervical cancer was studied in SiHa, CaSki cells and nude mice tumor models. The role of LIMK1 in the growth of cervical cancer was evaluated by HE staining. The role of LIMK1 in the invasion, metastasis, and proliferation of cervical cancer was evaluated by cell scratch, Transwell, and monoclonal experiments. The interaction among LIMK1, ROS, and Src was evaluated by Western blotting. The effects of regulating ROS and p-Src expression on LIMK1 in the migration/invasion and proliferation of cervical cancer cells were evaluated through cellular functional assays.
Results Overexpression of LIMK1 promoted tumor growth in nude mice. Cell scratch, Transwell, and monoclonal experiments suggested that LIMK1 promoted the invasion, metastasis, and proliferation of cervical cancer cells. Western blotting suggested that LIMK1 can promote the expression of ROS-related proteins NOX2, NOX4, p-Src, and downstream proteins p-FAK, p-ROCK1/2, p-Cofilin-1, F-actin and inhibit the expression of p-SHP2 protein. Correction experiments showed that LIMK1 regulated the expression of p-FAK and p-Cofilin-1 proteins by regulating ROS and p-Src. Through the detection of cervical cancer cell functions, it was found that the activation of ROS and p-Src induced by LIMK1 is an early event that promotes the migration, proliferation, and invasion of cervical cancer cells.
Conclusions LIMK1 promotes the expression of F-actin and promotes the development of cervical cancer by regulating the oxidative stress/Src-mediated p-FAK/p-ROCK1/2/p-Cofilin-1 pathway.

PO_040

25 岁以下年轻宫颈癌治疗方式及其预后探讨

张明悦[1,2] 依力米努尔·阿布拉江[1,2] 刘真佑[1,2] 杜卓颖[1,2] 王胜倩[1,2] 杨淑丽[1,2] 吴玉梅[1,2] 何玥[1,2*]
1.首都医科大学附属北京妇产医院
2.女性生殖系统肿瘤前沿技术研发和转化联合实验室

目的 分析 SEER 数据库和我院 25 岁以下宫颈癌患者的流行病学特征、病理分化、治疗方式及预后差异，探讨手术与非手术组对特定分期生存率的影响，为 25 岁以下宫颈癌诊治提出个性化治疗建议。
方法 基于 SEER 数据库纳入 2000-2021 年 73,869 例宫颈癌患者，筛选 25 岁以下年轻宫颈癌患者 544 例，统计其年龄分布、分化类型及治疗方式等。采用生存分析模型比较ⅠB3 期和 IIA2 期宫颈癌患者手术组与未手术组的 3 年及 5 年生存率，并分析 IIIA 和 IIIB 期宫颈癌患者联合辅助化疗与未辅助化疗组的预后差异。结合我院 2009 年以来的 12 例年轻宫颈癌患者数据，验证数据库研究结果与我院临床真实世界结果的一致性。
结果 （1）SEER 数据库 2000-2021 年间数据显示，25 岁以下患者占比呈波动下降趋势（2004 年 1.362%至 2021 年 0.387%），低分化癌占比最高（51.65%）。（2）SEER 数据库中 544 例年轻宫颈癌患者中，424 例患者有分期结果，早期宫颈癌 298 例，包括：IA 期 162 例，IB1-IB2 期 76 例，IB3

期 34 例，IIA1 期 4 例，IIA2 期 22 例。晚期宫颈癌 126 例，包括：IIB 期 24 例，IIIA 期 6 例，IIIB 期 52 例，IIIC 期 10 例纳入，IVA 期 4 例，IVB 期 30 例。年轻宫颈癌患者中早期患者比例高达 77.94%。（3）在 SEER 数据库中ⅠB3 期手术组（25 例）3 年和 5 年生存率分别为 84%和 80%均高于非手术组（9 例）55.56%和 44.44%（P>0.05），IIA2 期 3 年生存率非手术组（4 例）为 75%高于手术组（18 例）66.67%，5 年生存率非手术组（4 例）为 50%略低于手术组（18 例）55.56%，这可能与非手术组样本量过少有关（4 例）（P>0.05）。其中 IIIA-IIIB 期 3 年和 5 年生存率辅助化疗组（51 例）均高于非辅助化疗组（6 例）（P>0.05），且前者死亡风险明显高于后者。（4）纳入我院 25 岁以下宫颈癌患者 12 例，我院数据与 SEER 数据库趋势一致，早期宫颈癌患者（IIA2 期以前）以手术为主，IIA2 期多采用手术+放化疗，III 期以后多采用综合治疗（放疗联合辅助化疗），12 例宫颈癌患者中仅有 2 例死亡，均为特殊类型肿瘤，一例为 IVB 期宫颈小细胞神经内分泌癌和 IIB 期宫颈透明细胞癌，肿瘤类型相比分期对于年轻宫颈癌患者预后更重要。

结论 年轻宫颈癌患者大部分分期为早期，分化以低分化为主。手术对年轻早期局限性病变宫颈癌（IIB 期以前）患者更具优势，晚期宫颈癌（III 期以后）联合辅助化疗有延长患者生存的趋势。肿瘤类型相比分期可能对于年轻宫颈癌患者预后更重要，研究为优化年轻宫颈癌患者诊疗方案提供依据。

PO_041

治疗性宫颈癌疫苗：肿瘤免疫治疗的新模式

苏芸怡 张露双 吴羡 谢艾洁 李垚杰 干晓琴*
电子科技大学医学院附属妇女儿童中心医院·成都市妇女儿童中心医院

高危型 HPV（尤其 16/18 型）导致 90%以上宫颈癌。E6/E7 癌蛋白驱动癌变，现有疫苗（2/4/9 价）靶向 L1/L2 衣壳蛋白，仅预防新感染，对已感染细胞无效。治疗性 HPV 疫苗通过靶向 E6/E7 抗原激活 CTL 免疫，为宫颈癌前病变提供“功能性治愈”新策略。虽在晚期癌治疗中证据有限，其临床转化潜力有望重塑宫颈癌治疗格局。本文旨在于总结治疗性宫颈癌疫苗治疗机制及目前研究进展，为未来宫颈癌诊治提供理论依据。

PO_042

叶酸受体 α 在宫颈癌中的诊疗价值

马溪遥 胡元晶*
天津市中心妇产科医院

研究目的 总结叶酸受体 α（FRα）介导的相关诊疗方法在宫颈癌中的研究进展，针对 FRα 在宫颈癌研究中存在的如表达异质性、耐药性等局限性以及探索方向的多元化，从而为进一步优化宫颈癌的诊疗、提升 FRα 在宫颈癌诊疗管理中的应用价值提供新的临床思路。

材料与方法 通过回顾国内外相关文献，系统梳理 FRα 的生物学特性、在恶性肿瘤中的研究现状，以及其在宫颈癌发生、发展及转移中的参与机制，同时总结目前宫颈癌以铂类为基础的同步放化疗治疗方案与靶向药物联合治疗的研究进展，分析 FRα 介导的诊疗策略在宫颈癌中的应用潜力与局限。

结果 FRα 在叶酸摄取中起重要作用，且在卵巢癌、乳腺癌等领域研究较深入，但在宫颈癌中的研究存在局限性，探索方向与结论呈现多元化。前期研究证实，FRα 参与宫颈恶性肿瘤的发生、发展及转移，具备成为宫颈癌生物标志物和治疗靶点的潜力。目前以铂类为基础的同步放化疗是局部晚期宫颈癌的标准方案，但靶向药物与该方案的联合应用在提高患者生存率方面未达预期。 FRα 介导的治疗策略对晚期和复发宫颈癌患者的诊断及治疗具有重要价值，但仍面临表达异质性、耐药性等问题。

结论 FRα 作为宫颈癌潜在的生物标志物和治疗靶点，其介导的诊疗策略对改善宫颈癌，尤其是晚期和复发患者的诊断与治疗具有重要意义。未来需进一步研究以解决 FRα 在宫颈癌中的表达异质性和耐药性等局限，优化现有诊疗方法，从而提升患者预后，为宫颈癌的精准诊疗管理提供实践参考。

PO_043

个体化定制式 3D 打印模具在宫颈癌三维近距离治疗中剂量学的探讨

李凌
江西省妇幼保健院

目的 探讨个体化定制式 3D 打印模具在宫颈癌高剂量率三维近距离治疗中剂量学的优势；比较其与通用模具辅助的近距离治疗靶区剂量和 OAR 剂量的差异。
方法 前瞻性对 22 名中晚期宫颈癌患者随机 1:1 分组研究，研究组应用定制个体化 3D 打印模具行腔内联合组织间插植近距离放疗（3D-IC/ISBT），对照组采用通用模具辅助的近距离治疗（（IC/ISBT）。所有患者均按规范完成盆腔外照射治疗（EBRT）后开始近距离放疗（BT）。每位患者均行 5 次近距离治疗，处方剂量为 6Gy/次。联合外照射总 HR-CTVD90 至少 85Gy(EQD2)，每周行 1-2 次。初步对比 3D-IC/ISBT 和 IC/ISBT 两组中的 HR-CTV、OARs （膀胱、直肠、乙状结肠）的相关计划参数和靶区适形指数 CI。
结果 3DP-IC/IS 组对比徒手 IC/IS 组在 HR-CTVD90（636.30±27.14 cGy vs. 618.03±18.03cGy，p=0.003）、D95(609.14±19.11cGy vs. 597.44±21.34cGy，p=0.047)、膀胱 D2cc（420.33±23.87cGy vs. 448.77±20.20cGY, p＜0.001）；直肠 D2cc（362.70±31.23cGy vs. 398.29±33.62cGy, p=0.042）。而乙状结肠 D2cc（240.45±68.05cGy vs. 243.70±48.37cGy, p=0.731）、CI（0.84±0.14 vs. 0.82±0.19 p=0.799）；HR-CTV 的 V100、D98、 D100 参数均无统计学差异(p>0.05)。
结论 3D-IC/IS BT 相比通用模具的 IC/IS BT 在 HR-CTVD90、D95 以及危及器官膀胱直肠的剂量分布上有一定优势，其安全性高。个体化定制式 3D 打印模具 BT 在满足危及器官剂量限制的前提下，提高了肿瘤靶区的剂量，降低了放疗医生对插植的熟练度要求，但定制流程较为复杂。

PO_044

HPV16 Integration-Associated Exosomal circMAP3K5 Activates POSTN⁺ Activated Fibroblasts to Drive Cervical Intraepithelial Neoplasia

Chuhong Guo[1] Luojiao Liang[1] Wei Wang[2*]
1.The First Affiliated Hospital, Guangzhou Medical University
2.Shanghai First Maternity and Infant Hospital

Objective To elucidate how HPV16 integration–linked exosomes from cervical premalignant cells trigger fibroblast activation and to define the underlying molecular circuitry.
Methods Integrated bioinformatic mining, qRT-PCR, and RNA in situ hybridization were employed to identify circMAP3K5 as the pivotal circRNA induced by HPV16 integration. SiRNA knockdown or lentiviral overexpression of circMAP3K5 was established in precancerous cervical epithelial cells. Exosomes isolated from these cells were applied to normal fibroblasts. Western blotting, wound-healing assays, Transwell migration assays, and ELISA were used to assess the conversion of quiescent fibroblasts into POSTN⁺ activated fibroblasts (AFs).

Results Elevated circMAP3K5 levels correlated tightly with cervical lesion grade. Exosomal circMAP3K5 was enriched in the secretome of HPV16-positive premalignant cells and was efficiently transferred to fibroblasts, driving their activation into $POSTN^+$ AFs. Mechanistically, circMAP3K5 sequestered miR-140-3p, thereby relieving the microRNA-mediated repression of POSTN. Silencing miR-140-3p in fibroblasts up-regulated α-SMA, FAP, and POSTN and enhanced migratory capacity, whereas miR-140-3p overexpression abrogated exosome-induced fibroblast activation and POSTN secretion.
Conclusion Precancerous cervical cells deploy exosomal circMAP3K5 to activate fibroblasts via the circMAP3K5/miR-140-3p/POSTN axis. circMAP3K5 thus constitutes a key effector through which HPV16-associated exosomes orchestrate $POSTN^+$ AF activation during cervical intraepithelial neoplasia, offering a potential diagnostic and therapeutic target for impeding lesion progression.

PO_045

Impact of prior lymphadenectomy on immunotherapy efficacy in recurrent cervical cancer

Hao He Misi He Dongling Zou*
Chongqing University Cancer Hospital

Objective Lymphadenectomy is a common staging procedure in cervical cancer management, providing critical pathological data to guide therapy stratage. However, several studies had indicated that resection of tumor-draining lymph node may diminish systemic anti-tumor immunity. Hence, we aimed to explore the impact of historical pelvic and/or abdominal lymphadenectomy on the efficacy of immunotherapy in recurrent cervical cancer, as well as the other factors that affect the efficacy of immunotherapy.
Materials and Methods We conducted a retrospective cohort analysis of 84 patients with recurrent cervical cancer who received immunotherapy at Chongqing University Cancer Hospital from October 2019 to January 2024. Patients were stratified based on prior pelvic and/or abdominal lymphadenectomy and compared the immunotherapy efficacy outcomes, including objective response rate (ORR), progression-free survival (PFS), and overall survival (OS) and analysed the other influencing factors.
Results Lymph node resection didn't affect the total ORR($p=0.12$), but reduced the total CR rate($p<0.05$)，which difference was manifested in lymph chain recurrent group and inside-pelvic radiation field recurrence group. The ORR of previous pelvic lymph node resection was lower than previous para-aortic lymph node resection($p<0.05$). The PFS and OS of lymph node resection group were worse than non-surgical group($p<0.05$). There were no difference in ORR, PFS and OS no matter lymph chain recurrence or not($p=0.46$). The ORR,PFS and OS of inside-pelvic radiation field were worse than outside-radiation field($p<0.05$).
Conclusions Patients without lymphadenectomy history and inside-pelvic radiation field recurrent had the best immunotherapy effect,which could be used as reference for predicting the efficacy of immunotherapy in recurrent cervical cancer. Attach importance to the role of regional lymph node integrity in immunotherapy after recurrence. When initial treatment efficacy was comparable, try to minimize the range of lymph node resection in radical surgery and staging surgery, as well as reduced the range of pelvic lymph node radiotherapy.

PO_046

IIIC1r 期（局部分期 T1a2-T2a2）宫颈癌预后影响因素分析及不同治疗方式预后比较

郑梦雯 齐跃 刘娟娟 张丹晔 叶秋霖 胡悦欣 林蓓*
中国医科大学附属盛京医院

目的 探讨 IIIC1r 期（局部分期 T1a2-T2a2）宫颈癌预后影响因素及影像学诊断盆腔淋巴结转移的价值；比较不同治疗方式生存预后，筛选行手术治疗显著获益的临床特征，为临床治疗决策提供参考依据。
方法 选择 2016 年 1 月至 2021 年 10 月于中国医科大学附属盛京医院治疗的 285 例 IIIC1r 期（局部分期 T1a2-T2a2）宫颈癌患者，按治疗方式分为根治性手术组及根治性同步放化疗组（CCRT）。手术组患者依据术后病理分为术后淋巴结阴性和阳性组，分析手术组患者术后盆腔淋巴结状态（是否转移、转移数量、髂总是否转移）对预后的影响，比较手术组和 CCRT 组患者的预后及并发症。计算两组患者的限制性平均生存时间（RMST），基于 CCRT 组患者 RMST，将手术组患者分为获益组与非获益组，筛选患者行手术获益的临床特征。此外，以术后病理为金标准，评估影像学诊断盆腔淋巴结转移的准确性。
结果 1.手术组患者 3 年 OS（85.9%）、DFS（81.7%）均高于 CCRT 组（74.5%， 69.1%）；术后淋巴结阴性组患者 3 年 OS（93.0%）、DFS（91.9%）均高于术后淋巴结阳性组（80.0%，73.3%）。T 分期、影像学盆腔淋巴结转移数量、治疗方式是患者 3 年 OS 和 DFS 的独立影响因素（*P*<0.05）。术后淋巴结阴性、转移数≤2 个、无髂总转移的患者 3 年 OS 和 DFS 显著更高（*P*<0.05）。2.以病理为金标准，PET-CT、增强 CT、增强 MRI 诊断盆腔淋巴结转移均具中等一致性（Kappa 值：0.435，0.407，0.425；*P*<0.001），PET-CT 灵敏度最高（62.0%），增强 MRI 特异度最高（94.8%）。3.两组 III-IV 度骨髓抑制及放射性膀胱炎发生率无差异，CCRT 组放射性直肠炎发生率（14.9%）显著高于手术组（6.1%），*P*=0.026。4.手术组 3 年 RMST（33.73 月）显著高于 CCRT 组（31.16 月），差值 2.57 月，*P*=0.011。年龄、T 分期、影像学盆腔淋巴结转移数量是手术组患者是否获益的独立影响因素（*P*＜0.05）。
结论 1.T 分期、影像学盆腔淋巴结转移数量、治疗方式是 IIIC1r 期（局部分期 T1a2-T2a2）宫颈癌预后的独立影响因素。2.术后盆腔淋巴结状态（是否转移、转移数量、髂总转移）是手术组患者预后的影响因素。术后淋巴结阴性组患者生存预后优于术后淋巴结阳性组优于 CCRT 组，该层级关系凸显影像学诊断盆腔淋巴结转移的局限性—与病理结果存在中等一致性，且存在较高的漏诊及误诊率，导致分期偏差及治疗失当；手术可以通过精准病理分期指导术后治疗方案的选择，避免治疗不当。3.相比同步放化疗，手术治疗生存更优且放射性直肠炎发生率低，年龄＜50 岁、T1 分期、影像学盆腔淋巴结转移数量＜2 个的患者手术获益显著。临床实践中，可综合考虑年龄、局部肿瘤等因素，制定个体化治疗方案。

PO_047

IFNγ combined aPD-1 treating cervical precancerous lesions through the microneedle delivery system

Yiting Liu Xucui Zhuang Chaoyang Sun Gang Chen*
华中科技大学同济医学院附属同济医院

Introduction Cervical cancer is the fourth most common cancer in women globally, which also seriously threatens the health of women in China. Cervical intraepithelial lesion (CIN) is a precancerous state of cervical cancer, most of which can regressed naturally, and only a small part will progress to cancer. If timely detection and active intervention can achieve complete cure, but there is a lack of

effective treatment. Microneedles are a novel drug delivery system, which can form multiple micrometer-scale mechanical pores in the local tissue. Biocompatible transdermal microneedles can efficiently deliver drugs to the lesion site and avoid the first-pass elimination effect. In our previous study, we found that IFNγ could effectively activate the immune activity of cervical cancer cells and promote tumor cells killed. Therefore, the aim of this study is to investigate the efficacy of combined delivery of IFNγ and aPD-1 via microneedle to the cervix for the treatment of cervical precancerous lesions.

Materials and Methods Cervical precancerous lesion cell lines and organoids derived from CIN patient tissues were treated with IFNγ for different times, and the cellular immune activity was detected by PCR to explore whether IFNγ could activate the cellular immune activity. The cytotoxicity of microneedle materials on cervical cancer precancerous cell line S12 and normal cervical epithelial cell line U14 were investigated by the cytotoxicity test. With hyaluronic acid and silk fibroin as the body materials of microneedles, IFNγ and aPD-1 were simultaneously loaded into the microneedles by 3D printing technology, and whether the microneedles could effectively penetrate the cervical tissue and the drug release was explored in vitro and in vivo. FVB.Cg-Tg (KRT14-HPV16)/Nju mice were used to construct a cervical precancerous lesion model. The empty microneedle, IFNγ microneedle, aPD-1 microneedle and IFNγ+aPD-1 microneedle were injected into the cervical region of the mice to observe whether regression of cervical lesions occurred.

Results After treated with IFNγ for different time, RNA was extracted and the cellular immune activity was detected by PCR. The results showed that the cellular immune activity was effectively activated in both cervical precancerous lesion cell lines and organoids after 6 hours of IFNγ treatment. The cell lines treated with hyaluronic acid and silk fibroin showed no significant changes in activity, suggesting that the microneedle material had no significant toxic effect on the cells. The results of in vitro and in vivo puncture experiments showed that the microneedles could effectively penetrate the cervical tissue and release drugs. FVB.Cg-Tg (KRT14-HPV16)/Nju mice were used to establish a cervical precancerous lesion model. After treatment with empty microneedle, IFNγ microneedle, aPD-1 microneedle and IFNγ+aPD-1 microneedle, the results showed that IFNγ+aPD-1 microneedle could subside cervical precancerous lesions and block the progression of the disease.

Conclusion The combination of IFNγ and aPD-1 in the treatment of cervical precancerous lesions can effectively inhibit the progression of cervical precancerous lesions and make the lesions regression. This study provides a new method for the treatment of cervical precancerous lesions.

PO_048

Immunological characteristics associated with the response to neoadjuvant immunochemotherapy in locally advanced cervical cancer

Wenxin Liao

Immunological characteristics associated with the response to neoadjuvant immunochemotherapy in locally advanced cervical cancer

Object Neoadjuvant immunochemotherapy has demonstrated considerable antitumor efficacy and is anticipated to emerge as a novel therapeutic approach for locally advanced cervical cancer. This combined modality leverages the synergistic potential of chemotherapy-induced tumor antigen release and immune checkpoint blockade-mediated T cell activation. However, the dynamic changes in the tumor immune microenvironment (TIME) that determine treatment response remain poorly characterized. Critical knowledge gaps persist regarding how immunochemotherapy alters the composition, functional states, and spatial organization of immune cell subsets. Additionally, the molecular mechanisms underlying differential responses between patients are unclear. A comprehensive understanding of these immunological changes could identify predictive biomarkers and novel therapeutic targets to enhance treatment efficacy. Systematic investigation of TIME

remodeling during neoadjuvant immunochemotherapy is therefore urgently needed to optimize this promising treatment strategy.

Methods and materials This comprehensive investigation leverages cutting-edge single-cell RNA sequencing (scRNA-seq) technology to systematically characterize the dynamic evolution of the tumor immune microenvironment (TIME) during neoadjuvant immunochemotherapy for locally advanced cervical cancer. By analyzing longitudinal samples collected at three strategically selected time points - baseline before treatment initiation (T1), following induction chemotherapy (T2), and after completion of neoadjuvant therapy (T3) - we aim to delineate the temporal patterns of immune cell composition, functional states, and molecular signatures that distinguish treatment responders from non-responders. Our scRNA-seq approach will provide unprecedented resolution to identify critical shifts in immune cell populations. Beyond single-cell transcriptomics, we will employ an innovative multi-modal analytical framework integrating spatial transcriptomics to map the geographical distribution of immune cell subsets within tumor niches, multiplex immunofluorescence (mIF) to validate protein-level expression of key immune checkpoints, and functional assays to assess cell-cell interactions. By correlating our high-dimensional immune profiling data with clinical outcomes (pathologic complete response vs. residual disease), we will establish a precision immunomonitoring platform capable of guiding treatment decisions. Furthermore, our findings may reveal fundamental mechanisms of how chemotherapy synergizes with immunotherapy to reshape the TIME, with potential implications extending beyond cervical cancer to other immunogenic malignancies. The integration of spatial biology with single-cell genomics represents a particularly innovative aspect of this work, allowing us to dissect how cellular neighborhoods influence treatment responses.

Results We observed an increase in immune cell infiltration levels at the T2 time point, upregulation of immune activation-related gene expression, and significant activation of immune response-related pathways, particularly in patients who achieved complete remission. Our findings will provide the potential mechanisms underlying treatment responses in patients who achieve a pathological complete response, with the aim of predicting and enhancing the preoperative pathological complete response rate. Multiplex immunofluorescence also confirmed a significant increase in the infiltration of various immune cells in patients who achieved complete remission. Flow cytometry analysis of clinical samples also revealed high infiltration of various immune cells at the T2 time point. Our integrated analysis proposes a novel immunodynamic model where early chemotherapy-induced immunogenic cell death primes subsequent immune checkpoint inhibitor efficacy in responders. These results provide clinically actionable biomarkers for treatment stratification and identify potential therapeutic targets to convert immunologically "cold" tumors, with important implications for designing next-generation neoadjuvant combination therapies in cervical cancer.

Conclusion The immune microenvironment characteristics observed at the T2 time point may serve as critical biomarkers for predicting patient responses to neoadjuvant immunochemotherapy in locally advanced cervical cancer.

PO_049

Carcinogenic IGKC-Induced Interferon Signaling Suppression Promotes Lymphatic Metastasis in Cervical Cancer

Zixuan Fang
Department of Gynecological Oncology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

BACKGROUND Lymph node metastasis is a strong indicator of unfavorable prognosis in cervical cancer, as it is closely linked to disease recurrence and mortality. Previous studies had shown that squamous epithelial tumor cells generate elevated level of immunoglobulin (Ig) , which may contribute to tumor progression and invasion.
METHODOLOGY We constructed a single-cell atlas of cervical cancer utilizing scRNA-seq from 15 treatment-naive tumors, 6 paired lymph nodes and 3 normal cervix samples from healthy donors. Our analysis identified a distinct subpopulation within the cervical cancer epithelium characterized by specific expression of carcinogenic IGKC. By incorporating transcriptomic and genomic data from the Cancer Cell Line Encyclopedia (CCLE), we analyzed V(D)J rearrangement involved in immunoglobulin production within both B cell and epithelial cell lineages. These observations were further substantiated through in vitro and in vivo experimental validation. In vitro experiments including cell lines and primary cultures confirmed the existence, constitutive pattern and phenotypic function of IGKC by qPCR, RNAscope, next-generation sequencing, RNA-seq, WB and phenotypic experiments. The CUT-Tag experiment and the dual-luciferase experiment verified the regulatory effect of the transcription factor TFAP2C on the initiation transcription of IGKC. The sites and mechanisms of IGKC-specific binding proteins were jointly explored by immunoprecipitation experiments and CLIP-seq. In vivo experiments were conducted to construct a mouse lymph node metastasis model and observe the survival outcomes of mice in the high-expression IGKC group.
RESULTS Cervical cancer patients with lymph node metastasis expressed higher level of IGKC than those without lymph node metastasis. IGKC expression was consistently observed across pan-cancer cell lines, with alternative splicing producing IGKC observed at the transcriptional level rather than the genomic level. Next-generation sequencing of tumor cells from cancer cell lines and patients with cervical cancer further revealed that IGKC lacks V(D)J processes in tumor cells, with only a non-classical pattern of variable splicing in the C-J region.This pattern was defined as lncRNA-IGKC due to its inability to translate into protein. lncRNA-IGKC has been verified in various clinical tumor samples, among which higher lncRNA-IGKC signals were detected in the samples with lymph node metastasis. In contrast to B-cell Ig gene transcription, transcription initiation in tumor cells was affected by Transcription Factor AP-2 Gamma (TFAP2C). lncRNA-IGKC tend to co-bind with RNA-binding protein heteroribonucleoprotein M (HNRNPM), promoting more complexes to bind to the intron region of the gene and the intracellular dsRNA level decreasing, then resulted in significantly inhibition of the interferon (IFN)-responsive pathway, which made tumor more invasive into lymph node and led to poor prognosis. In vitro models have confirmed that tumors with high expression of IGKC are more likely to metastasize to the popliteal fossa and abdominal wall lymph nodes.
CONCLUSION We identified a distinct subpopulation with carcinogenic IGKC expression within the epithelial cells of cervical squamous cell carcinoma and validated its potential role in promoting lymph node metastasis. Meanwhile, the identification of lncRNA transcripts generated by alternative splicing in carcinogenic IGKC showed interferon response supression in tumor progression. These findings may have important implications for the diagnosis, targeted therapy, as well as monitoring of residual disease of cervical cancers.

PO_050

Clinicopathological Characteristics and Prognostic in Cervical Clear Cell Adenocarcinoma: A Multicenter Retrospective Cohort Study

Weiqi Yin Ying Zhou*
Affiliated Hospital of Anhui University, China

Background The clinicopathological profile and prognostic determinants of cervical clear cell adenocarcinoma (CCAC) in patients without diethylstilbestrol (DES) exposure remain inadequately characterized within the Chinese population. This study therefore aimed to comprehensively investigate the clinicopathological characteristics and identify significant prognostic factors influencing outcomes in non-DES-exposed CCAC patients undergoing radical surgery, utilizing a multicenter retrospective analysis.

Methods This multicenter retrospective study analyzed data from patients with primary CCAC treated across 10 medical centers in China: The First Affiliated Hospital of USTC, Zhejiang Cancer Hospital, Sun Yat-sen University Cancer Center, The Second Hospital of Jilin University, Sun Yat-sen Memorial Hospital of Sun Yat-sen University, The First Affiliated Hospital of Wannan Medical College, The First Affiliated Hospital of Bengbu Medical University, China-Japan Friendship Hospital, Fudan University Shanghai Cancer Center, Zhongda Hospital Southeast University between 2010 and 2022. A total of 97 patients who underwent radical surgery with confirmed pathological diagnosis were included. A detailed clinicopathological profile of these non-DES-exposed CCAC patients was compiled. Subsequently, the associations between various clinicopathological characteristics and key prognostic factors (such as overall survival and recurrence-free survival) were statistically evaluated.

Results The peak age prevalence occurred in the 46-55 years group (n=97), with 85.1% of cases being HPV-negative. All surgically treated patients had FIGO stages IA-IIA or IIIC and received adjuvant radiotherapy and/or chemotherapy. Surgical approaches included transabdominal (84.5%, n=82) and laparoscopic (15.5%, n=15) procedures. With a median follow-up of 57.4 months (range: 5-137). Univariate analysis identified tumor size (HR=3.85; 95% CI: 1.15-12.91), deep stromal invasion (DSI, HR=8.30; 95% CI: 1.81-38.02), ovarian metastasis (OM; HR=3.09; 95% CI: 1.03-9.39), lymphovascular space invasion (LVSI, HR=4.47; 95% CI: 1.80-11.07), and lymph node metastasis (LNM; HR=4.80; 95% CI: 1.94-11.86) as significant predictors for OS (all $p<0.05$). On multivariable analysis, ovarian metastasis (OM, HR=3.89, p=0.027) and LNM (HR=4.26, p=0.004) remained an independent risk factor for OS. Univariate analysis revealed that tumor size, DSI, LVSI, LNM were all significantly associated with PFS (all $p<0.05$). Multivariable analysis further demonstrated that LNM (HR=3.15, p=0.005) remained significant for PFS. Bilateral ovaries were preserved in 9 patients (age range: 19–37 years). Among the 9 patients, 2 were diagnosed with stage IB3 disease and 1 was pathologically confirmed as stage IIIC1p postoperatively. During follow-up, 2 of the 9 ovarian preservation cases experienced disease recurrence, including 1 mortality. The deceased patient exhibited DSI >2/3 with LNM.

Conclusions This retrospective multicenter study characterized the clinicopathological features of non-DES-exposed cervical clear cell adenocarcinoma (CCAC) and identified significant prognostic factors. In surgically treated patients, tumor size >4 cm, deep stromal invasion (DSI >2/3), lymphovascular space invasion (LVSI), and lymph node metastasis (LNM) independently predicted poorer progression-free survival (PFS). For overall survival (OS), significant independent predictors included tumor size >4 cm, DSI >2/3, ovarian metastasis (OM), LVSI, and LNM. Ovarian preservation may represent a feasible option for carefully selected young patients with early-stage disease, characterized by limited tumor burden, absence of high-risk pathological features, and normal ovarian morphology. However, the long-term safety of this approach requires validation through expanded cohort studies and prolonged follow-up.

PO_051

HPV Status as a Predictor of Ovarian Metastasis in Cervical Adenocarcinoma: Implications for Surgical Decision-Making

Wang Ming[1] Yumei Wu[1*] Jianqing Xu[2]
1.Beijing Obstetrics and Gynecology Hospital, Capital Medical University
2.Beijing Haidian District Maternal and Child Health Care Hospital

Objective Human papillomavirus (HPV) infection does not drive all cervical adenocarcinomas. HPV-independent cervical adenocarcinomas exhibit distinct histologic subtypes, metastatic patterns, and prognoses. This study aimed to compare the incidence of ovarian metastasis between HPV-related and HPV-independent cervical adenocarcinomas..
Methods We conducted a retrospective analysis of cervical adenocarcinoma patients treated at Beijing Obstetrics and Gynecology Hospital, Capital Medical University, from 2012 to 2022. Ovarian metastasis rates were compared between HPV-related and HPV-independent cohorts.
Results Among 444 cervical adenocarcinoma patients, 10.58% (47/444) had HPV-independent tumors.The ovarian metastasis rate was significantly higher in HPV-independent versus HPV-related cases (36.17% vs. 2.16%, $p < 0.01$). Macroscopic metastasis rates were comparable between groups (HPV-independent: 62.5% vs. HPV-related: 58.8%, $p = 0.861$). Oophorectomy was more frequently performed in HPV-independent patients (93.60% vs. 75.06%, $p = 0.004$). No ovarian metastases were detected during follow-up in ovary-preserved patients.Multivariate analysis confirmed HPV-independent status as an independent risk factor for ovarian involvement (HR = 19.66, 95% CI: 5.23–73.91, $p < 0.01$).
Conclusions HPV-independent status is a strong independent predictor of ovarian metastasis in cervical adenocarcinoma.These findings support ovarian preservation in HPV-related tumors without intraoperative evidence of suspected metastasis.

PO_052

DNA 甲基化标志物在宫颈高级别上皮内病变预测中的应用

刘森[2*] 倪换娟[1] 高盼[2]
1.北部战区总医院 2.陕西中医药大学第二附属医院

目的 宫颈癌前病变的精准筛查是降低宫颈癌发病率的关键。现有 HPV 检测特异度低、TCT 灵敏度不足，亟需更优的筛查工具。两项研究均聚焦宫颈 DNA 甲基化标志物 FAM19A4、PAX1、ZNF582，分别探讨其在预测宫颈高级别上皮内病变（HSIL+）和宫颈上皮内瘤变 2 级及以上（CIN2+）中的价值，旨在构建高效的风险预测模型。
方法 两项研究均采用回顾性设计，样本来源于北部战区总医院。高盼等纳入 1822 例宫颈病理检查女性，按 7:3 分为训练集（1278 例）和验证集（544 例），分析 HSIL+（含 CIN2、CIN3 及癌变）的影响因素；倪换娟等纳入 627 例筛查异常者，同样 7:3 分组（训练集 472 例、验证集 200 例），聚焦 CIN2+病变。
检测方法一致：采用长沙三济科技试剂盒检测 FAM19A4、PAX1、ZNF582 甲基化水平，结合 HPV 分型（14 种高危型）及临床资料。统计学上均通过 Lasso 回归筛选变量，构建 Logistic 回归模型，采用 ROC 曲线、校准曲线及临床决策曲线评估效能。
结果
1. HSIL+预测模型：Lasso-Logistic 回归显示吸烟史、FAM19A4、PAX1、ZNF582 为显著影响因素（均 $P<0.05$）。训练集 AUC 达 0.971（灵敏度 0.950，特异度 0.902），验证集 AUC0.974，Hosmer-Lemeshow 检验提示拟合良好（$P>0.3$）。

2. CIN2+预测模型：Lasso 回归筛选出 HPV（12 种高危型及 16/18 型）、FAM19A4、PAX1、ZNF582，多因素分析均为独立影响因素（均 P<0.05）。训练集 AUC0.941（灵敏度 0.904，特异度 0.847），验证集 AUC0.939，校准曲线贴合理想曲线。单一标志物中，ZNF582 效能最优（HSIL+预测 AUC0.890，CIN2+验证集 AUC0.840），PAX1 次之，FAM19A4 相对较低。

结论 基于 FAM19A4、PAX1、ZNF582 构建的模型在 HSIL+和 CIN2+预测中表现优异，联合临床因素或 HPV 可提高筛查准确性，弥补传统方法缺陷，为宫颈癌前病变的个体化诊疗提供可靠工具。

PO_053

Development and Validation of a Personalized Prognostic Prediction Tool for Gastric-type endocervical adenocarcinoma: SEER Database Analysis and Single-Center Validation

Xindao Wang[1] Luzan Ma[1] Xinwei Tao[2] Haiyan Zhu[1*]
1. Department of Gynecology, Shanghai First Maternity and Infant Hospital, Tongji University School of Medicine
2.Boston Scientific Corporation, Shanghai

Purpose Gastric-type endocervical adenocarcinoma (G-EAC) is a rare but highly aggressive malignant tumor. This study aims to develop a prognostic nomogram capable of accurately predicting outcomes for patients with G-EAC.

Methods A total of 385 G-EAC patients (346 from the SEER database and 39 patients from our hospital as validation set) were enrolled. The patients from SEER were splited at the ratio of 7:3 (241 as development set and 105 as internal validation set). Predictive factors were identified from the development set using univariate and multivariate Cox regression analyses, and a model for predicting overall survival (OS) was constructed and visualized as a nomogram. The diagnostic performance of the prediction models was evaluated using C-index, time-dependent the Area Under Curve (AUC) internally and externally.

Results The final prognostic model identified six independent factors for G-EAC: age at diagnosis, tumor stage, T stage, tumor size, primary site surgery, and chemotherapy. The C-index of the model acheived 0.88(95%CI 0.81-0.96), 0.88(95%CI 0.76-0.99) and 0.87(95%CI 0.69-1.00) for development set, internal validation and external validation set, respectively. At 1-, 3- and 5- year, the AUC of time-ROC were 0.94 (95% CI: 0.90 - 0.98), 0.93 (95% CI: 0.89 - 0.99), and 0.92 (95% CI: 0.87 - 0.97), respectively. The internal validation time-ROC for the model at 1-, 3-, and 5 year were 0.98(95%CI: 0.95-1.00), 0.92(95%CI: 0.88-0.98), and 0.91(95%CI: 0.83-0.98), respectively. In terms of external validation, the external validation ROC for the Cox model at 1-, 3-, and 5 year were 0.97(95%CI: 0.92-1.00), 0.77(95%CI: 0.53-0.99), and 0.68(95%CI: 0.32-1.00), respectively,].

Conclusion Our study presents a reliable nomogram for predicting the prognosis of G-EAC patients. This tool offers clinicians a practical means to identify high-risk patients and guide personalized treatment decisions.

PO_054

宫颈低分化鳞癌伴神经内分泌分化 1 例病例报道

马玲 王静*
兰州大学第一医院

研究目的 宫颈低分化鳞癌伴神经内分泌分化是一种罕见且侵袭性强的宫颈癌亚型，预后较差，临床诊断和治疗面临诸多挑战。本文报道一例 33 岁女性患者的诊疗过程，结合近三年最新文献，探讨此类肿

瘤的临床病理特征、治疗策略及预后因素，旨在提高临床医师对该疾病的认识，优化诊疗方案，改善患者生存结局。

材料与方法 本研究采用病例分析结合文献综述的方法，回顾性分析一例 33 岁女性患者的临床资料。患者因“同房后出血 2 年余”就诊，经病理活检及免疫组化确诊为 HPV18 相关的宫颈低分化鳞癌伴局灶神经内分泌分化（CD56+/SYN+/CgA 局灶+）。患者接受腔镜下免举宫子宫广泛性切除术(C2 型)+双侧输卵管切除术+双侧卵巢切除术+双侧盆腔淋巴结扫术+右侧髂总动脉旁淋巴结活检术+抗肿瘤化学药物灌注治疗+腹腔引流术，术后病理提示 T1b1N1Mx（AJCC 分期），伴淋巴结转移。术后辅助白蛋白紫杉醇+卡铂化疗。通过分析患者的临床表现、病理特征、治疗方案及随访结果，并结合国内外相关文献，探讨此类罕见肿瘤的诊疗要点。

结果 患者以“同房后出血”为主要症状，HPV18 检测阳性，初始外院误诊为低分化腺癌，后经免疫组化修正诊断为低分化鳞癌伴神经内分泌分化（Ki67 指数 80%）。影像学检查提示宫颈内膜增厚及淋巴结肿大。患者接受腹腔镜广泛子宫切除术+盆腔淋巴结清扫术，术后病理显示肿瘤侵及宫颈中 1/3 纤维肌层，左侧盆腔淋巴结 1/9 转移，分期为 T1b1N1Mx。术后给予白蛋白紫杉醇+卡铂化疗，恢复良好。文献分析表明，HPV18 感染与神经内分泌分化显著相关，且此类肿瘤具有高侵袭性，预后较差（5 年生存率较普通鳞癌低 20-30%）。本例的高 Ki67 指数和淋巴结转移进一步提示不良预后。

结果 宫颈低分化鳞癌伴神经内分泌分化是一种罕见但恶性程度高的肿瘤，其诊断依赖病理活检和免疫组化（如 CgA、Syn、CD56 等标志物）。HPV18 感染可能是其重要诱因，且与不良预后相关。治疗上以手术为主（广泛子宫切除+淋巴结清扫），术后需辅以化疗或放化疗。本例提示，早期诊断和综合治疗是关键，但患者预后仍受肿瘤分期、淋巴结转移及 Ki67 指数等因素影响。未来需进一步开展大样本研究，探索更有效的靶向治疗和免疫治疗策略，以改善患者生存率。临床医师应提高对此类罕见肿瘤的警惕性，加强多学科协作，优化个体化治疗方案。

PO_055

宫颈癌 MIF-CD74 信号轴增强中性粒细胞 PD-L1 表达促进免疫抑制微环境形成的机制研究

徐新鑫[1,2] 李珂[1] 张陈[1*]
1.同济大学医学院 2.同济大学附属第十人民医院

研究目的 探究宫颈癌如何驯化中性粒细胞构建免疫抑制微环境的分子机制，为免疫治疗提供新的思路。

材料与方法 临床样本联合数据库观察宫颈癌患者粒细胞与分期之间的关系，单细胞测序数据解析宫颈肿瘤微环境中的粒细胞转录图谱。体外诱导分化和外周血原代分离获取人中性粒细胞，探索肿瘤细胞对中性粒细胞的驯化机制，最后通过动物模型研究观察肿瘤微环境内中性粒细胞的变化。

结果 患者淋巴细胞和粒细胞比值与患者的临床分期正相关。单细胞分析发现肿瘤细胞通过 MIF-CD74 信号轴驯化中性粒细胞呈现 CD74+/PD-L1+的表型。MIF 可以影响患者的分期和预后。在体外研究发现，MIF 可以促进中性粒细胞 PD-L1 的表达，有抑制炎症作用，MIF 驯化的中性粒细胞可以抑制 T 细胞的杀伤功能。动物模型证明 MIF 可以通过驯化粒细胞来影响肿瘤的生长。

结果 宫颈肿瘤中的中性粒细胞有重要的临床价值，肿瘤细胞通过 MIF-CD74 信号轴增强中性粒细胞 PD-L1 表达以构建免疫抑制微环境。

PO_056

宫颈癌微创与开腹根治性子宫切除术后结局的系统综述和 META 分析

王娟[1] 周小铃[2*]
1.石河子大学 2.石河子大学第一附属医院

目的 旨在评价 2018 年之后微创根治性子宫切除术（MIS）与开腹根治性子宫切除术（ORH）治疗宫颈癌的术后疗效，探讨 MIS 和 ORH 在治疗宫颈癌中的价值。
方法 系统检索 PubMed、EMBASE 及 Web of Science 自 2018 年之后发表的相关文献，初检获得 399 篇研究。经双人独立去重筛选后，最终纳入 11 篇符合预设标准的文献。采用 RevMan 5.3 软件进行 Meta 分析，纳入的结局指标包括死亡、复发、术后淋巴结转移、术后并发症、术后辅助治疗、手术时间、术中失血量、住院时长、5 年无病生存率及 5 年总生存率等。
结果 筛选后纳入 11 项研究，包括 9028 例患者。Meta 分析显示（P＜0.05 具有统计学意义），MIS 与较低的死亡人数相关（95%CI =0.66-0.94，P=0.01），MIS 术后辅助治疗人数少（95%CI =0.72-0.88，P ＜0.00001），MIS 患者术后淋巴结转移少（95%CI =0.70-0.80，P =0002），MIS 和 ORH 在住院时间上有显著差异即 MIS 术后住院时间较 ORH 短，（95% CI = -2.53-0.63，P= 0.001)，MIS 和 ORH 在 5 年无病生存率上有显著差异，ORH 术后 5 年无病生存率优于 MIS（95% CI = 1.01-1.59，P= 0.04)，MIS 术中失血量少于 ORH，MIS 和 ORH 在术中失血量上通过敏感性分析后有统计学意义即微创术中失血量少（95% CI = -128.45-60.04，P＜0.00001）。MIS 和 ORH 术后复发人数无显著差异（95%CI =-0.02-0.02，P =0.95），MIS 和 ORH 在术后并发症上无显著差异（95% CI = 0.67-1.44，P= 0.91），MIS 和 ORH 在操作时间上无显著差异（95% CI = -42.47-60.55，P= 0.73），MIS 和 ORH 在 5 年总生存率无显著差异（95% CI = 0.78-1.63，P= 0.52）。
结论 微创根治性子宫切除术在降低围手术期死亡率、减少淋巴结转移、缩短住院时间及减少术中失血方面优于开腹手术，且术后辅助治疗需求更低；然而，其 5 年无病生存率不及 ORH。这些发现为宫颈癌的手术治疗提供了重要的临床指导，有助于医生根据患者的具体情况选择最适合的手术方法，具有十分重要的临床意义。

PO_057

基于生物信息学的宫颈癌淋巴结转移预后模型构建

王娟[1] 周小铃[2*]
1.石河子大学 2.石河子大学第一附属医院

目的 本研究旨在构建基于关键基因的宫颈癌淋巴结转移预后预测模型，并探讨其与肿瘤免疫之间的相关性。
方法 研究团队从 TCGA 和 UCSC 两大权威数据库获取 193 例有无宫颈癌淋巴结转移患者的临床及基因表达数据，其中包括 60 例淋巴结转移患者（N1 组）和 133 例无转移患者（N0 组）。首先通过差异表达基因（DEGs）分析筛选出与淋巴结转移相关的关键基因，随后利用 String 数据库构建蛋白质相互作用网络（PPI），并借助 Cytoscape 软件筛选出前 20 个核心 Hub 基因。为进一步明确这些基因的生物学功能，研究采用 GO 功能注释和 KEGG 通路富集分析方法。在此基础上，结合 LASSO 回归算法筛选出与淋巴结转移密切相关的特征基因，同时运用 CIBERSORT 算法进行免疫细胞浸润分析。最终通过单因素和多因素 Cox 回归分析，建立具有临床实用价值的预后预测模型。
结果 研究成功鉴定出 2 个与宫颈癌淋巴结转移患者预后显著相关的关键基因（P<0.05）。基于这两个基因构建的风险评分模型能够有效区分高、低风险患者群体，生存分析显示高风险组患者的总生存期显著短于低风险组（P<0.05）。ROC 曲线分析证实该模型具有良好的预测效能，其 1 年、3 年和 5 年

生存率的 AUC 值均超过 0.6。值得注意的是，免疫浸润分析揭示了风险评分与肿瘤免疫微环境存在显著相关性。

结论 本研究成功建立了一个基于关键基因的宫颈癌淋巴结转移预后预测模型，该模型不仅具有良好的预测准确性，而且为深入理解宫颈癌淋巴结转移的免疫调控机制提供了新的理论依据，对个体化治疗策略的制定具有重要的临床指导价值。

PO_058

基于糖酵解相关基因的宫颈癌预后模型构建与免疫浸润特征研究

王娟[1] 周小铃[2*]
1.石河子大学 2.石河子大学第一附属医院

目的 本研究通过生物信息学技术，探究宫颈癌与糖酵解相关基因的内在联系，筛选预后相关基因，构建预后模型，并探讨其与免疫浸润水平的相关性，为宫颈癌的临床诊断和治疗提供新的生物标志物和预后工具。

方法 从 TCGA 和 UCSC 数据库获取宫颈癌患者的基因表达谱数据及临床信息进行处理，通过 GSEA 网站下载糖酵解相关基因集并进行差异分析，筛选出 112 个宫颈癌糖酵解相关的差异基因（DEGs）。利用 COX 回归分析构建预后模型，将患者分为高、低风险组，绘制风险曲线并通过 ROC 曲线评估模型预测效能。同时，利用 Cbioprotal 分析基因突变情况，探讨临床病理特征与风险评分的关系，并通过 CIBERSORT 算法分析免疫微环境差异。

结果 筛选出 3 个与糖酵解相关的独立预后基因，构建的预后模型显示高、低风险组患者的预后存在显著差异（P<0.05），模型 1、3、5 年的 AUC 分别为 0.698、0.692 和 0.705，具有良好的预测效能。独立预后分析验证了模型的临床价值，肿瘤分期、T、M 和 N 分期是影响生存的关键因素。在特定临床分组中，高、低风险组差异显著。突变分析发现相关基因存在扩增突变，免疫细胞浸润分析显示两组在免疫细胞之间的差异。

结论 本研究成功构建了宫颈癌与糖酵解相关的预后模型，为宫颈癌的临床诊断和治疗提供了新的生物标志物和预后工具。

PO_059

宫颈冷刀锥切术及筋膜外子宫切除术治疗 IA1 期不伴 LVSI 的年轻宫颈癌患者的临床特征及预后对比分析

杜欣颖 郭瑞霞*
郑州大学第一附属医院

目的 对比分析宫颈冷刀锥切术（cold knife conization，CKC）及筋膜外子宫切除术（extrafascial hysterectomy，EH）治疗 IA1 期不伴淋巴脉管间隙浸润（lymphovascular space invasion，LVSI）的年轻宫颈癌患者的临床特征及预后情况。

方法 回顾性收集 2013 年 01 月-2023 年 09 月就诊于郑州大学第一附属医院的 153 例 IA1 期宫颈癌患者的临床资料，根据手术方式的不同分为 CKC 组（53 例）和 EH 组（100 例），对比分析两组的临床特征、围术期指标及预后情况，随访 CKC 组患者的生育结局。

结果 对比 CKC 组，EH 组手术时间（89.01±33.27min vs. 47.57±26.07min）、术后首次肛门排气时间（2.16±0.49d vs. 1.28±0.50d）、留置尿管时间（2.00±0.64d vs. 1.15±0.36d）及术后住院时间（5.33±1.69d vs. 3.81±1.70d）均明显更长，且术中出血量（33.20±27.15ml vs 13.09±14.56ml）明显

更多（P＜0.001）。EH 组有 6 例（6/100，6.0%）出现术后并发症。两组 3 年无病生存期（disease-free survival，DFS）及总生存期（overall survival，OS）均为 100%（P＞0.05）。CKC 组术后妊娠率为 63.2%（12/19），活产率为 46.2%（6/13），早产率为 50.0%（3/6）。
结论 CKC 手术损伤小，术后恢复快，预后及生育结局良好，可作为 IA1 期不伴 LVSI 的年轻宫颈癌患者的临床选择。

PO_060

PAX1、SOX1 基因甲基化在宫颈癌中的临床价值及研究进展

王子潇
新疆医科大学第一附属医院

宫颈癌是全球育龄妇女中第二大最常见的癌症，其死亡病例占癌症死亡病例 4.9%，居女性癌症死亡率第七位，尽管针对宫颈癌的筛查已逐渐普及，但仍有部分患者未及时接种疫苗或行宫颈癌筛查，导致发现时已是晚期而产生不良预后，增加社会负担，而且研究表明现有的宫颈癌筛查依旧存在其局限性，目前的筛查方法仍然存在不足，传统的巴氏细胞学检测受限于样本条件，液基薄层细胞学检测可重复性差,诊断结果易受病理学医师主观影响,高危型人乳头状瘤病毒 DNA 检测则受 HPV 一过性感染的影响,特异度较低，因此，探索新型生物标志物以提高宫颈癌早期筛查的准确性显得尤为重要。PAX1 和 SOX1 是两种较为重要的抑癌基因，已被证实在肿瘤发生及生长环节中扮演重要角色，其甲基化状态的变化不仅影响基因表达，还可能成为潜在的诊断和预后生物标志物，且其表现出优异的筛查性能及在宫颈癌治疗和预后方面表现出优良的前景，因此，深入研究 PAX1、SOX1 基因甲基化在宫颈癌中的临床价值和作用机制，不仅有助于我们更好地理解宫颈癌的发生和发展过程，也为宫颈癌的治疗和预后提供了新的思路和方法。

PO_061

Chinese patterns and trends in cervical cancer incidence overall and by histological subtypes: a population-based analysis from 1993 to 2017

Yuhan Chen
Shengjing Hospital of China Medical University

Background Cervical cancer (CC), originating in the cervix, represents the most prevalent gynecological malignancy and ranks as the second most commonly diagnosed cancer in women. Furthermore, it is the second most frequent cause of cancer-related mortality in women of reproductive age. In recent years, despite a downward trend in the age-standardized incidence rates (ASIRs) of CC globally, the incidence rate of CC in China has been continuously rising and notably shifting towards younger age groups. China, as the nation with the highest number of CC cases worldwide, presents a substantial health challenge for its female population. However, comprehensive epidemiological evidence regarding the trend in CC incidence overall and by its subtypes in China remains limited. This study analyzed incidence patterns (2013–2017) and trends (1993–1997 to 2013–2017) of CC and its subtypes across China, regions, and provinces.
Methods The incidence patterns of CC and its histological subtypes in China were analyzed at the national, regional, and provincial levels for the period 2013–2017, using registry data from Volume XII of the CI5 database. Trends in CC incidence and its subtypes were assessed across consecutive 5-year intervals from 1993–1997 to 2013–2017 for both China as a whole and individual provinces. Provinces included in the trend analysis were required to have at least 15 consecutive years of data

(spanning 3 volumes) and include the most recent five-year period. Ultimately, data from 25 provinces were utilized for the pattern description, while 7 provinces met the criteria for inclusion in the trend analysis. Annual percent changes were computed to describe trends in age-standardized incidence rates (ASIRs) of CC and its subtypes using Joinpoint Regression Program.
Results About 67,053 new CC cases occurred in China during 2013–2017. The ASIR, adjusted based on the Chinese standard population in 2000 and world Segi's population, stood at 11.91/100,000 and 10.92/100,000, respectively. From 1993–1997 to 2013–2017, China has witnessed a pronounced upward trend in the incidence of CC (APC: 7.06, 95% confidence interval: 5.68, 8.85). Variations in ASIRs of CC were observed across different regions and provinces in China, with notably high in Central and Northwest regions. Squamous cell carcinoma (SCC) stood as the most prevalent, especially high in Northeast China. Following SCC, other tumors and adenocarcinoma showed relatively high ASIRs in both Central and Northwest regions. Beyond sarcoma, the incidence trends of various subtypes aligned with those of CC, displaying a notable upward trend. The incidence of CC and its subtypes peaked in the 50–54 age group.
Conclusion Our research utilized the CI5 and CI5plus databases to delineate the most recent prevalent patterns and trends in CC incidence, thereby conducting a comprehensive assessment of the CC burden in China. Currently, China faces a significant burden of CC, with notable variations in the incidence of CC and its histological subtypes observed across different regions. These disparities may originate from regional variations in Human papillomavirus (HPV) prevalence, vaccination practices, socioeconomic conditions, and health education initiatives. It is urgent to tailor a comprehensive national and regional prevention and control system that integrates CC screening with HPV vaccination to effectively mitigate the burden of CC, particularly in Central and Northwest regions.

PO_062

CIN Ⅲ 锥切术前和术后的全基因组 DNA 甲基化差异性分析

曾勇
荆州市第一人民医院

目的 子宫颈锥切术是目前治疗子宫颈 HSIL（CIN Ⅲ）的最常用方式，但锥切术后存在病灶残留和复发风险，因此有必要对宫颈锥切术后的患者进行精准的分流管理。HPV 和 TCT 的准确性、特异性及阴道镜转诊仍存在一定的局限性，DNA 甲基化是已知的与肿瘤高度相关的表观遗传改变，DNA 甲基化的变化被认为是肿瘤辅助诊断的生物标志物，并认为通过特定 DNA 甲基化位点的水平检测可以用于肿瘤的早期诊断、治疗评估和预后预测。拟通过全基因组 DNA 甲基化测序（WGBS）检测 CIN Ⅲ患者锥切术前术后的差异 DNA 甲基化区域，筛选可用于 CIN Ⅲ锥切术后监测的潜在 DNA 甲基化生物标志物并探讨其相关生物学功能。
方法 收集 2023 年 9 月至 2025 年 6 月就诊于荆州市第一人民医院 CIN Ⅲ患者锥切术前及锥切术后再次行全子宫切除术前（间隔时间不超过 6 个月）宫颈脱细胞。根据全子宫切除后病理诊断结果选择无残留组患者前期预留的宫颈脱落细胞进行全基因组甲基化测序。对差异甲基化基因进行 GO 功能富集分析和 KEGG 通路富集分析，找出其参与的生物学过程及其信号通路相关靶基因，并通过构建蛋白质-蛋白质互相作用（PPI）网络，解析甲基化差异区域的功能通路及关键基因。
结果 1. DNA 甲基化差异特征：对 CIN Ⅲ患者（无残留组）锥切术前术后宫颈脱落细胞进行全基因组甲基化测序（WGBS）检测，共发现 29384 个 DMR 位点，其中包括高甲基化 DMR 12440 个，16994 个低甲基化 DMR。（见图 1-1 和图 1-2）
2.GO 功能富集分析和 KEEG 分析：DMR 显著富集于癌症相关通路，例如 MAPK 信号通路、轴突导向、黏着斑通路等，涉及细胞增殖、迁移和信号转导。GO 富集分析表明，差异基因主要富集在染色体，着丝粒区、纤维中心、浓缩染色体着丝粒、组蛋白去乙酰化酶复合物等的生物过程中。KEGG 通路分析显示，差异甲基化表达基因在 MAPK 信号通路、cAMP 信号通路、肌动蛋白细胞骨架的调节、轴突导向等途径中富集。（见图 2-1 和图 2-2）

3. 关键基因筛选：通过 PPI 网络，筛选出 AKT1、UBC、H2BC21、H2BC14 等关键基因，其甲基化异常可能通过 PI3K/AKT 通路、泛素-蛋白酶体系统及染色质重塑参与。 (见图 3）

结论 1. CIN Ⅲ锥切术前和术后存在差异甲基化区域，为 CINⅢ术后病灶残留/复发监测提供了潜在的 DNA 甲基化生物标志物，但仍需扩大样本进一步认证。

2.DNA 甲基化通过调控 AKT1、UBC 等关键基因表达可能影响宫颈病变的发生发展，可为宫颈病变的监测提供更高的特异性，有望弥补现有 HPV+TCT 筛查的局限性，为个体化随访和精准干预提供理论依据。

PO_063

39 例宫颈浸润性复层产黏液癌临床病理分析

翟庆杰 郭瑞霞*
郑州大学第一附属医院

目的 探讨宫颈浸润性复层产黏液癌（invasive stratified mucin-producing carcinoma，ISMC）的临床病理特征及预后。

方法 回顾性分析郑州大学第一附属医院 2019 年 1 月至 2025 年 4 月收治的 ISMC 患者病例资料。采用 Kaplan-Meier 法绘制生存曲线，单因素及多因素 Cox 比例风险模型进行预后分析。结果 39 例患者的平均年龄为 46.97±11.89 岁（29~77 岁）；临床表现以接触性出血（43.6%）和不规则阴道流血（33.3%）为主；所有患者人乳头瘤病毒（human papillomavirus，HPV）检测均阳性（37/37），HPV18（56.8%）和 HPV16（35.1%）为主要感染类型；液基薄层细胞学检查（ThinPrep cytology test，TCT）异常率 83.3%（25/30），但仅 10.0%（3/30）提示非典型腺细胞；单一型 ISMC 占 71.8%；FIGOⅠ期较为常见（61.5%）；肿瘤平均直径 2.78±1.55cm，生长方式以内生型（56.4%）为主；淋巴结转移率为 15.4%（6/39），深肌层浸润率为 37.8%（13/33），脉管癌栓发生率为 39.4%（13/33）；所有可评估病例（35/35）均为 Silva C 型；PD-L1 阳性率为 93.7%（CPS≥1）。中位随访时间 17 个月（范围：1~65 个月），3 年总生存率和无进展生存率分别为 92.9%和 74.5%。单一型与混合型 ISMC 各项临床病理及预后无统计学差异（P>0.05）。Cox 多因素回归分析显示，淋巴结转移是 ISMC 无进展生存期的独立危险因素（HR=8.341，95%CI：1.388~50.111，P=0.020）。

结论 ISMC 临床表现、TCT 检查无特异性，组织学形态多样且侵袭性强；淋巴结转移是 ISMC 患者术后复发的危险因素；PD-L1 高表达提示其有望成为潜在治疗靶点。

PO_064

IGF2BP3-Mediated Translational Repression of MYCBP Through 3'UTR Binding in Cervical Squamous Cell Carcinoma Lymph Node Metastasis

Zejia Mao[1,2] Misi He[2,3] Qian Zheng[2,3] Ling Long[2,3] Xinghe Liu[1,2] Yan Wang[1,2] Ting Guo[1,2] Gongxian Shen[1,2] Haixia Wang[2,3] Dongling Zou[2,3,4*]
1.School of Medicine, Chongqing University, Chongqing, China.
2.Department of Gynecologic Oncology, Chongqing University Cancer Hospital & Chongqing Cancer Institute & Chongqing Cancer Hospital, Chongqing, China.
3.Chongqing Specialized Medical Research Center of Ovarian Cancer, Chongqing, China.
4.Organoid Transformational Research Center, Chongqing Key Laboratory for the Mechanism and Intervention of Cancer Metastasis, Chongqing University Cancer Hospital, Chongqing, China.

Objective Cervical cancer ranks as the fourth most prevalent cancer in women worldwide. The presence of lymph node metastasis is associated with a notably poorer prognosis in affected patients.

Existing studies on lymph node metastasis in cervical cancer have predominantly concentrated on transcriptional regulatory pathways, with limited exploration on the impact of translation in this process. The primary objective of this study is to investigate the influence of IGF2BP3 on MYCBP protein translation in lymph node metastasis of cervical squamous cell carcinoma (CESC), with the aim of proposing novel targeted therapeutic approaches.

Methods Ribosome profiling sequencing and RNA sequencing were conducted on 25 CESC tissues, including lymph node metastatic and non-metastatic samples, to identify MYCBP, which exhibited increased ribosome binding activity alongside stable mRNA levels. Validation of this finding was performed via Western Blot analysis on a cohort of 50 cases. Functional assays, entailing MYCBP knockdown and overexpression, were employed to assess cell migration, invasion, and lymphangiogenesis. Functional validation in vivo was carried out using a mouse model of popliteal lymph node metastasis.The potential interaction between IGF2BP3 and MYCBP was initially anticipated through bioinformatics resources and literature, and subsequently experimentally verified. Cells with IGF2BP3 knockdown were treated with cycloheximide and the proteasome inhibitor MG132, followed by dual-luciferase assays to evaluate their impact on MYCBP protein translation.

Results The MYCBP protein expression was notably elevated in the lymph node metastatic group compared to the non-metastatic group in CESC (2.6 vs. 1.37, p = .03). In CESC cells, suppressing MYCBP hindered cell migration, invasion, and lymphangiogenesis, whereas its overexpression augmented these characteristics. The same lymph node metastasis characterization was obtained in vivo experiments. Immunohistochemical analysis revealed a negative correlation between the expression levels of MYCBP proteins (18.61% vs. 67.76%, $p < .001$) and IGF2BP3 (59.76% vs. 17.51%, $p < .001$) in CESC. Knockdown of IGF2BP3 led to increased MYCBP protein levels, which could be reversed by CHX but not by MG132. Dual-luciferase assays confirmed that IGF2BP3 suppresses MYCBP protein expression by binding to the 3'UTR region of MYCBP mRNA, resulting in a 42.3% decrease in fluorescence activity in the overexpression group ($p<.001$).

Conclusion This research reveals the promotion of lymph node metastasis in CESC by MYCBP and explores how IGF2BP3 influences MYCBP protein synthesis by interacting with the 3'UTR region of MYCBP mRNA. Future investigations should further explore the complex regulatory pathways of the IGF2BP3-MYCBP axis in the tumor microenvironment, facilitating the creation of personalized therapeutic strategies for cervical cancer.

PO_065

Identification of Neoantigen Epitopes in Cervical Cancer by Multi-omics Analysis

Jing Yuan Xueqi Gong Jihui Ai Kezhen Li* Yingyan Han
Department of Gynecological Oncology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

Background Tumor vaccines enhance the immune response and cytotoxicity of tumor-specific T cells and show promising clinical therapeutic efficiency. Although most cervical cancer cells express HPV-related oncoproteins, certain cells do not. Additionally, neoantigens worth attracting attention for cervical cancer may hold the potential for breakthroughs in the development of cervical cancer vaccines.

Methods A comprehensive computational analysis was conducted based on the tumor genome, transcriptome, and proteome data from 284 cervical cancer samples obtained from the TCGA database. Frequently mutated genes were identified. Levels of immune cell infiltration using RNA-seq data and high-frequency mutated genes as candidate genes that were significantly related to immune infiltration were analyzed. Focusing on MHC class I epitopes recognized by CD8+ T cells, we predicted potential neoantigen peptides using the NetMHCpan-4.0 and NetCTL-1.2 algorithms and evaluated

the immunogenicity of the synthesized peptides in vivo using flow cytometry and real-time PCR to examine markers of T-cell activation and cytotoxicity.
Results We identified 30 highly mutated genes, among which TTN, PTEN, PRKDC, PCLO, MUC17, LRP1B, HUWE1, FBXW7, and CREBBP were positively correlated with immune cell infiltration into the tumor microenvironment. PCLO exhibited higher protein expression in tumor tissues than in the corresponding normal tissues, making it a potential tumor antigen. The PCLO peptides SISRFTLEK and LSEAGHFFY exhibited the highest predicted scores among the cancer antigens.
Conclusion Our analysis highlights the potential of PCLO as a candidate gene for enhancing immune cell infiltration and activating immune responses in tumors. The peptides SISRFTLEK (PCLOL4169F) and LSEAGHFFY (PCLOA3000S) are promising targets for tumor vaccines.

PO_066

厌氧消化链球菌来源的外膜囊泡破坏宫颈癌脉管系统并促进宫颈癌转移的作用和分子机制研究

顾铮 张梦蕾 沈方 周冠楠 丁景新*
复旦大学附属妇产科医院

研究目的 本课题旨在探究厌氧消化链球菌（Peptostreptococcus anaerobius）来源的外膜囊泡破坏宫颈癌脉管系统并促进宫颈癌脉管转移的作用及分子机制。主要明确 P. anaerobius 及其分泌囊泡对脉管系统的内皮连接的破坏作用，通过体外和体内实验研究其对内皮细胞间紧密连接、血管通透性的影响，并进一步研究促进宫颈癌转移的作用及分子机制。
材料与方法 采取人脐静脉内皮细胞（HUVECs）作为细胞模型，分别用 P. anaerobius 的菌上清、外膜囊泡处理 HUVECs 并设置相应对照组，通过 Western-blot、qPCR、共聚焦免疫荧光显微镜，体外探究 P. anaerobius 及其分泌囊泡对内皮紧密连接的影响。选用裸鼠进行足垫成瘤，分别用 P. anaerobius 的菌上清、外膜囊泡定期进行足垫注射并设置相应对照组，收集瘤体与同侧腹股沟淋巴结，观察 P. anaerobius 及其分泌囊泡对肿瘤淋巴转移的影响。
结果 经菌上清处理后的 HUVECs 中紧密连接蛋白及相应 mRNA 表达量下降，经外膜囊泡处理后该下调更为显著。体内实验中经菌上清、外膜囊泡处理后的裸鼠组别也展现出更高的迁移倾向。
结论 P. anaerobius 来源的外膜囊泡可被 HUVECs 有效摄取，且在体内与体外均可以通过下调内皮细胞紧密连接蛋白、增强血管通透性的方式促进宫颈癌的脉管转移，是宫颈癌进展的新型促瘤因素。本研究首次探讨了促癌菌来源的外膜囊泡在宫颈癌中的作用，可为未来宫颈癌的治疗提供新的策略。

PO_067

The rationality of negative margin criteria for conization in early cervical cancer --A cohort study

Na Li Zelin Yu Xiao Li Yuan Gao Hu Yuan*
Tianjin Central Hospital of Gynecology Obstetrics

The 2023 (1st Edition) NCCN guideline changed the negative margin criteria for cervical conization from ≥3mm to ≥1mm. To explore the clinical safety of this up-dation in the real world, we performed a retrospective study to validate the rationality of the negative margin criteria for conization in patients with early-stage cervical cancer. We retrospectively collected the clinical and pathological data of 986 patients with early-stage cervical cancer from June 2011 to June 2023. All of the patients underwent conization and secondary (radical) hysterectomy at Tianjin Central Hospital of Gynecology Obstetrics. Patients were divided into four groups based on the status of the conization margins: margin <1mm

(n=132), margin <3mm (n=147), margin involved (n=443), and margin not involved (n=264). The safety of the negative margin criteria for conization (<1mm) was evaluated by comparing the residual disease rates in different groups after secondary (radical) hysterectomy. Additionally, univariate and logistic multivariate analyses were conducted to identify the risk factors for residual disease after hysterectomy. A total of 986 patients with cervical cancer at stages IA1-IB1 were included. Among these patients, 382 had residual diseases (HSIL or higher) after hysterectomy, resulting in a residual disease rate of approximately 38.7%. Patients were categorized based on margin status (<1mm, <3mm, margin involved, and, margin not involved). There were statistically significant differences in residual disease rates among the four groups (P<0.05). There is no significant difference in residual disease rates between patients with the margin <1mm and margin <3mm, nor between these two groups and the margin not involved group. However, the residual disease rates in cases with margins <1mm and margins <3mm were significantly lower compared to those with margin involvement. Univariate analysis indicated that multiple factors, including age, were associated with residual disease. However, multivariate analysis revealed that age (P = 0.002, OR = 1.041, 95% CI 1.014–1.069), involvement of the margin by the lesion (P< 0.001, OR = 7.655, 95% CI 4.745–12.349), and cervical biopsy pathology showing cancer (P=0.020, OR = 1.571, 95% CI 1.074–2.298) were independent risk factors for residual disease after hysterectomy in early-stage cervical cancer patients. Our findings suggest that a ≥1mm negative margin criterion appears to be a safe threshold for early-stage cervical cancer, yet prospective validation is still required.

PO_068

Vaginal Microbial Environment Skews Macrophage Polarization and Contributes to Cervical Cancer Development

顾铮 张梦蕾 沈方 陈君豪 龚滢欣 张淦荣 周冠楠 丁景新*
复旦大学附属妇产科医院

研究目的 本研究旨在阐明阴道微生物群，尤其是厌氧消化链球菌（Peptostreptococcus anaerobius），对宫颈癌发生发展的影响及作用机制。通过体外和体内实验，证明 P. anaerobius 通过诱导巨噬细胞向促肿瘤的 M2 表型极化，并分泌血管内皮生长因子（VEGF），促进肿瘤血管生成并提高迁移能力，从而加速宫颈癌的进展。

材料与方法 收集 20 例宫颈鳞癌组织样本及阴道灌洗液（10 例伴淋巴结转移，10 例无转移），检测分析其中 P. anaerobius 的表达量及 M2 型巨噬细胞浸润情况差异。用 P. anaerobius 处理 THP-1 诱导的 M0 巨噬细胞，通过流式细胞术检测 M2 型标志物 CD206 表达情况。收集 P. anaerobius 处理后的巨噬细胞上清液，处理人脐静脉内皮细胞（HUVECs），采用 CCK-8、管腔形成实验及免疫荧光评估血管生成能力，采用 ELISA 检测细胞上清 VEGF 水平变化。采用 SiHa-Luciferase 细胞对裸鼠进行腹腔成瘤，定期腹腔注射 P. anaerobius 处理后的巨噬细胞上清液并设置相应对照组，通过活体成像监测肿瘤迁移情况，并通过 IHC 检测瘤内 VEGFR2 表达。

结果 P. anaerobius 在宫颈癌患者阴道微生物群中的丰度显著升高，且癌组织中 M2 型巨噬细胞浸润增多。体外实验证实 P. anaerobius 具有诱导巨噬细胞向 M2 表型极化的能力，诱导的 M2 型巨噬细胞上清液中 VEGF 分泌量升高，且该上清可显著增强 HUVEC 的管腔形成能力并上调其 VEGFR2 表达。体内实验表明，接受 P. anaerobius 处理后的巨噬细胞上清液的小鼠体内肿瘤迁移增强，瘤内 VEGFR2 表达亦增高。

结论 P. anerobius 在宫颈癌微环境中富集，可通过诱导巨噬细胞 M2 极化并分泌 VEGF，促进肿瘤血管生成与体内迁移，是宫颈癌进展的新型促瘤因素。本研究首次揭示了 P. anerobius 对肿瘤微环境中巨噬细胞极化在宫颈癌进展中的影响，有助于为宫颈癌预防、诊断、治疗提供新的靶点和生物标志物。

PO_069

Mitochondrial HMGCS1 Mediates Cisplatin Resistance in Cervical Cancer through Regulation of Mitochondrial Transcription

Wenxuan Yang Chao Zhou*
Binzhou Medical University Hospital

Cisplatin resistance remains a major challenge in the treatment of cervical cancer. Here, we identify 3-hydroxy-3-methylglutaryl-CoA synthase 1 (HMGCS1), a rate-limiting enzyme in the mevalonate pathway, as a critical regulator of cisplatin resistance through its localization to mitochondria. HMGCS1 preferentially accumulates in the mitochondria of cisplatin-resistant cervical cancer cells, and targeted expression of HMGCS1 to mitochondria, but not to the nucleus or cytosol, is sufficient to confer cisplatin resistance. Mechanistically, mitochondrial HMGCS1 directly associates with the D-loop region of mitochondrial DNA (mtDNA) and facilitates the recruitment of the core transcription machinery components POLRMT, TFAM, and TFB2M. This interaction enhances the transcription of mtDNA-encoded respiratory complex subunits, resulting in increased mitochondrial respiratory capacity. Genetic depletion or pharmacological inhibition of HMGCS1 disrupts mitochondrial transcription, impairs respiratory function, and re-sensitizes resistant cells to cisplatin. Moreover, combined treatment with cisplatin and inhibitors targeting either HMGCS1 or mitochondrial transcription exhibits synergistic effects against cisplatin-resistant cervical cancer cells. Our findings reveal an unexpected role for HMGCS1 as a non-canonical regulator of mitochondrial transcription and establish the HMGCS1-mitochondrial transcription axis as a promising therapeutic target to overcome cisplatin resistance in cervical cancer.

PO_070

The clinical application value of dynamic monitoring of HPV circulating tumor cell DNA in concurrent chemoradiotherapy for locally advanced cervical cancer

Hanqun Zhang Xiaoqing Luo Jia Jiang Cui Zhang Libo Li Yuting Zeng Yuncong Liu Yong Li*
Department of Oncology, Guizhou Provincial People's Hospital

Objective The treatment principle for locally advanced cervical cancer is concurrent chemoradiotherapy. Nevertheless, local recurrence or distant metastasis of the disease still occurs after the completion of concurrent chemoradiotherapy. Currently, there is no effective method to monitor the efficacy of chemoradiotherapy and predict prognosis. This study predicts the therapeutic efficacy and prognosis by dynamically monitoring the HPV DNA levels in the blood and tissues of locally advanced cervical cancer patients.
Methods We enrolled 31 patients with pathologically confirmed newly diagnosed cervical cancer in the Oncology Department of Guizhou Provincial People's Hospital from July 2022 to January 2024. Blood and cervical cancer tissues were collected before/during treatment, and blood was collected one month post-treatment. HPV-Seq (14 subtypes) were used to quantify HPV genotype-specific DNA levels in plasma and tissues. Imaging examinations were performed during treatment and one month after the completion of the treatment to evaluate tumor regression.
Results Among the 27 patients with locally advanced cervical cancer (4 excluded), the HPV infection rate was 96.3% (26/27), the HPV16 infection rate was 63.0% (17/27), and the consistency rate of HPV detection in baseline tissues and plasma was 100%. HPV reads in plasma and tissues were significantly decreased both before and during treatment ($p<0.05$). Through therapeutic effect analysis,

it was found that the high expression of HPV ctDNA levels was correlated with the poor therapeutic effect of tumors ($p<0.05$). Univariate analysis revealed that negative baseline Plasma ctDNA, Low-Risk ChemoScore, and negative Plasma HPV during treatment were associated with a higher PR/CR rate. In multivariate analysis, dynamic monitoring of HPV expression status in plasma was an independent factor for predicting the efficacy of early treatment.
Conclusion Dynamic monitoring of HPV ctDNA expression levels is related to the therapeutic effect of concurrent chemoradiotherapy for locally advanced cervical cancer, providing a certain basis for earlier identification of intensive treatment after the completion of chemoradiotherapy for locally advanced cervical cancer.

PO_071

免疫抑制状态下 HPV 相关宫颈病变的临床转归及预后分析

张晓辰 李碧军 付翰林 郭瑞霞*
郑州大学第一附属医院

背景 人乳头瘤病毒（HPV）感染是女性下生殖道常见感染，高危型 HPV 持续感染与宫颈癌的发生密切相关。免疫抑制状态（如自身免疫性疾病患者或器官移植受者）因长期使用免疫抑制药物，可能导致免疫功能低下，增加 HPV 感染及宫颈病变的风险。既往对免疫抑制状态下 HPV 相关宫颈病变的临床转归及预后研究较少。因此，深入探讨免疫抑制患者的 HPV 感染特征、治疗效果及预后因素，对优化该类人群的临床管理具有重要意义。
目的 本研究旨在探讨免疫抑制状态下 HPV 相关女性宫颈病变的临床转归及预后情况。
方法 通过回顾性分析 2019 年至 2024 年期间 75 例接受免疫抑制药物治疗且因 HPV 相关宫颈病变于我院治疗的女性患者，收集其人口学特征、免疫抑制药物使用情况、宫颈病变及治疗信息，并进行随访。
结果 患者平均年龄为 40.91±10.393 岁，高危型 HPV 感染率高达 85.1%，病理确诊 HSIL 比例达 90.7%，多灶性病变占比 40%，这些数据共同揭示了免疫抑制患者宫颈病变的三大高危特征：多灶性病变、高比例的高危型 HPV 感染、极高比例的 HSIL 病变。治疗后 6 个月随访的 HPV 总体转阴率为 55.2%，其中 CKC 治疗组的转阴率最高（70.6%），而物理治疗组无一例转阴。单部位病变的转阴率显著高于多部位病变（65.0% vs 40.7%，$p=0.05$）。生存分析表明，中位 HPV 转阴时间为 7.0 个月，但平均转阴时间延长至 17.6 个月，且长期清除率受限，40 个月后平台期 HPV 阳性率维持在 10%-20%。亚组分析显示，CKC 组转阴最快（中位时间 9.0 个月），联合治疗组最慢（中位 22.0 个月）。成对比较显示两组转阴率差异显著（$\chi^2=5.000$, $p=0.025$）。肾移植患者转阴最慢（中位 19.0 个月），显著差于 SLE 组（6.0 个月，$p=0.099$）和 RA 组（6.0 个月，$p=0.209$）。
结果 本研究揭示免疫抑制宫颈病变患者呈现 HSIL 为主、高危 HPV 感染率高（85.1%）及多灶性病变（40%）的高危特征谱，该类群体治疗后 HPV 转阴率低，持续感染率高，中位清除时间 7 个月但平均转阴时间延长至 17.6 个月，且长期清除率受限（40 个月平台期），生存曲线显示早期清除与长期平台期的两阶段模式。受限于样本量与随访时间，未来需扩大队列并设置免疫正常对照组以优化管理策略。

PO_072

局晚宫颈癌化疗免疫新辅助序贯根治放疗的疗效与安全性

夏瑜 魏茜雪 曲慧 宫婷婷 高嵩 李秀琴*
中国医科大学附属盛京医院滑翔分院

目的 局部晚期宫颈癌（LACC）经同步放化疗后仍有约 40%复发/转移，尤其伴淋巴结转移者预后更差。目前免疫检查点抑制剂（ICI）在复发/转移阶段疗效显著，但用于 IIB–IVA 期新辅助治疗尚无定论。本研究评估化疗联合 ICI 新辅助序贯根治放疗治疗模式的近期疗效与安全性，为 LACC 提供新治疗选择。

方法 单中心回顾性纳入 2022.6-2024.7 中国医大盛京医院妇瘤病房收治的 31 例 FIGO 2018 IIB–IVA 期患者；中位年龄 59 岁，中位肿瘤直径 6.6 cm。先行 NACI：TP（白蛋白紫杉醇 260 mg/m²+顺铂 70 mg/m²）或 TC（白蛋白紫杉醇+卡铂 AUC 5）联合 Tislelizumab 200 mg、Sintilimab 200 mg 或 Cadonilimab 12.5 mg/kg，1-3 周期/3 周。后序贯根治放疗：IMRT 外照+影像引导近距离放疗，A 点 ≥85 Gy（EQD2）。放疗期间 7 例同步 ICI、11 例同步化疗、9 例同步化疗+ICI；放疗后 10 例 ICI 维持。疗效按 RECIST 1.1 结合妇科内诊于放疗前及放疗后 1 月评估；记录 PFS、OS、irAEs（CSCO 指南）及放疗毒性（CTCAE v5.0）。

结果 放疗前 ORR 93.5%（CR 25.8%，PR 67.7%）；根治放疗后 ORR 100%（CR 83.9%，PR 16.1%）。中位随访 14 月，PFS 与 OS 均未达到，仅 3 例复发，其中 1 例死亡。irAEs 51.6%（16/31），均为 1-2 级，无治疗相关死亡；32.3%患者因 irAEs 暂停 ICI。放疗相关肠道毒性 25.8%（G1 9.7%，G2 16.1%），泌尿系毒性 3.2%（G2），12.9%患者短暂中断放疗。NACI 2-3 周期者 ORR 100%，提示疗程越长疗效越优；鳞癌与腺癌均获益。

结果 化疗联合 ICI 新辅助序贯根治放疗使 LACC 近期缓解率达 100%，早期生存趋势良好，通过缩瘤显著降低放疗相关危及器官毒性，且 irAEs 可控。虽属小样本回顾性研究，长期结果待验证，但已为高肿瘤负荷 LACC 提供安全高效新治疗选择，值得前瞻性随机对照试验进一步确证。

PO_073

Risk and survival analysis for stage IVB cervical cancer patients with brain metastasis: A Population-Based Study

Haoran Li Xi Cheng*
Fudan University Shanghai Cancer Center

Objective The purpose of this study was to evaluate the risk and prognostic factors of stage IVB cervical cancer with brain metastasis from a population-based database, the Surveillance, Epidemiology and End Results (SEER).

Methods Cervical cancer patients initially diagnosed with brain metastasis between 2010-2019 were included in this study. The risk factors of developing brain metastasis were evaluated by Logistic regression model with corresponding 95% confidence interval (95% CI). Survival analysis was performed through the Kaplan–Meier method, log-rank test, and Cox proportional hazards model.

Results A total of 88 (88/25476, 0.35%) cervical cancer patients initially diagnosed with brain metastasis between 2010-2019 were retrieved. Accompanied with lung, bone or liver metastasis (all P<0.001) was shown to be independent risk factors for developing brain metastasis. Patients with brain metastasis indicated a poor prognosis (P<0.001, HR=2.84, 95%CI = 1.71-4.72) with a median survival month of 6, which is much shorter compared with the lung (9 months) or liver (8.5 months) or bone (11 months) metastasis group. Along with lower tumor grade (P=0.001, HR=0.27, 95% CI=0.09-0.76) and with bone metastasis (P=0.007, HR=2.74, 95% CI=1.33-5.67) demonstrated poor overall survival outcomes in patients with brain metastasis. In terms of treatment modality, chemoradiotherapy tended to prolong the survival of stage IVB cervical cancer patients with brain metastasis (P=0.001, HR=0.17 95%CI=0.06-0.48).

Conclusions In conclusion, the prognosis of stage IVB cervical cancer patients with brain metastasis remains poor. Chemoradiotherapy may provide survival benefits, which deserves large scale prospective clinical trials to confirm.

PO_074

光热疗法在宫颈癌临床前研究中的进展

褚晨阳 麦尔哈巴·艾力 陈建欢 衡友强 马彩玲*
新疆医科大学第一附属医院

子宫颈癌作为女性第四大常见癌症和致死原因，年发病例 53 万、死亡 27.5 万例。手术及顺铂同步放化疗是主要治疗手段，但晚期患者预后差、复发率高。基于纳米材料的光热疗法（photothermal therapy，PTT）以其非侵入、时空可控、低毒性等优势成为研究热点。PTT 利用具有光热转换能力的光热剂，在近红外激光的照射下将光能转化为热能，实现肿瘤消融。

近十余年来，光热疗法（PTT）在宫颈癌的临床前研究取得了显著进展。研究从早期探索基础光热效应（如 CuS、CB 染料纳米颗粒）开始，逐步深入到开发高稳定性、生物相容性材料（如 ICG@PDA@PEG ）和多功能纳米平台（如兼具成像与治疗的 MoS2@MnO2-PEG 、Bio-RBCm@PDA@MSN-DOX）。研究模型也从细胞水平扩展到复杂的皮下、原位及转移动物模型，光源探索也延伸至穿透更深的近红外二区（NIR-II），如 SF@ICG 在 NIR-II 波段实现淋巴结及生殖器官高分辨成像。与此同时，基于 PTT 的联合治疗策略成为研究热点，旨在克服单一疗法局限并实现“1+1>2”的协同效应。其中包括：PTT 联合化疗（CT）（如 GNRs@ATDN-DOX、AuNP@Ng/Cur，显著提升疗效并降低副作用）；PTT 联合放疗（RT）（如 BSA-Bi2S3-MnO2，利用放射增敏和缓解缺氧增效 RT）；PTT 联合免疫治疗（IT）（如 FA@LIPO@ICG@CpG，通过递送免疫佐剂和释放肿瘤相关抗原激活 T 细胞免疫应答）；PTT 联合基因治疗（GT）（如 RNA/AuNR@PDA，精准递送 miRNA 实现原发肿瘤的精准清除）以及基于 PTT 的多模态治疗（如铜铁氧体纳米球 CFNs，实现 PTT、PDT、CDT 及 MRI 成像功能的整合），展现诊疗一体化潜力。

PTT 通过材料创新与联合策略逐步克服疗效单一性，但临床转化仍面临穿透深度、生物安全性、热耐受及复发等挑战。未来研究应聚焦智能响应型纳米系统、优化联合治疗平台、推动 NIR-II 引导的精准诊疗及临床转化研究，以促进宫颈癌治疗的精准化与高效化。

PO_075

Clinicopathological and Prognostic Characteristics of Gastric-type Endocervical Adenocarcinoma: A nested case-control study

Yang Liu[1,2] Hui Wang[1,2] Yundi Hu[3] Ling Qiu[1,2] Xiaomei Sun[1,2] Xuan Yin[1,2] Shen Luo[1,2] Yue Yin[1,2] Qing Cong[1,2] Xiang Tao[1,2] Yan Zhao[1,2] Haiou Liu[1,2] Xiaolei Lin[3] Hua Jiang[1,2] Xin Wu[1,2*]
1.Obstetrics & Gynecology Hospital of Fudan University
2.Shanghai Red House Obstetrics and Gynecology Hospital
3.School of Data Science, Fudan University

Background Gastric-type endocervical adenocarcinoma is a rare, Human papillomavirus (HPV)-independent subtype of cervical cancer characterized by aggressive clinical behavior and poor prognosis. Due to its anatomical location and lack of HPV association, gastric-type endocervical adenocarcinoma is frequently missed by conventional screening, leading to delayed diagnosis.
Objectives This study aimed to systematically evaluate the clinicopathological characteristics, prognostic factors, and responses to postoperative adjuvant therapy in patients with gastric-type endocervical adenocarcinoma, and to compare outcomes with those of usual endocervical adenocarcinoma.
Study Design We conducted a nested case-control study within a prospectively maintained surgical cohort of cervical cancer patients at the Obstetrics and Gynecology Hospital of Fudan University. This cohort has been prospectively enrolling and following patients since December 15, 2017. Patients diagnosed with cervical cancer by cervical biopsy or diagnostic conization and scheduled for primary

surgical treatment were enrolled after informed consent and followed longitudinally with comprehensive data collection, including demographic, clinical, pathological, treatment, and survival information.
From this cohort, we identified patients with postoperative pathological confirmation of gastric-type endocervical adenocarcinoma (G-EAC) as the case group. For each G-EAC case, a control patient with usual endocervical adenocarcinoma (UEA) was selected using 1:1 propensity score matching based on variables including age, ethnicity, marital status, year of diagnosis, BMI, Karnofsky Performance Status, menopausal status, parity, FIGO stage, surgical approach and adjuvant treatment status.
The primary endpoints were three-year progression-free survival (PFS) and overall survival (OS). Survival outcomes between the two matched groups were compared using the Kaplan-Meier method, and the impact of adjuvant therapies on prognosis was assessed using Cox proportional hazards regression. Prognostic factors were further explored via univariate and multivariate analysis using the log-rank test and Cox regression models.
Results A total of 960 patients were included, with 195 cases of gastric-type endocervical adenocarcinoma and 765 cases of usual endocervical adenocarcinoma. After matching, patients with gastric-type endocervical adenocarcinoma had significantly lower three-year progression-free and overall survival rates than those with usual endocervical adenocarcinoma (66.1% vs. 79.8% and 74.9% vs. 84.6%, p = 0.014 and 0.033, respectively). Parametrial involvement (hazard ratio: 3.19 for overall survival, 2.54 for progression-free survival; p = 0.004 and 0.009, respectively) and pelvic lymph node metastasis (hazard ratio: 2.64 for overall survival, 2.26 for progression-free survival; p = 0.012 and 0.013, respectively) were identified as independent risk factors for mortality and disease recurrence in patients with gastric-type endocervical adenocarcinoma. Patients receiving combined radiotherapy and chemotherapy had significantly better survival outcomes than those undergoing either treatment alone (progression-free survival rates: 65.2% vs. 43.6%, overall survival rates: 74.3% vs. 54.5%, p = 0.040 and 0.028, respectively). However, gastric-type endocervical adenocarcinoma remained less responsive to adjuvant therapy compared to usual endocervical adenocarcinoma, with a persistently higher risk of recurrence and death.
Conclusions Gastric-type endocervical adenocarcinoma is associated with significantly worse survival outcomes than usual endocervical adenocarcinoma. Combined radiotherapy and chemotherapy improve prognosis compared to single-modality treatments, yet remains less effective for gastric-type than for usual endocervical adenocarcinoma. These findings underscore the need for improved therapeutic strategies and earlier detection for this aggressive subtype.

PO_076

KRT4 Activates Wnt/β-catenin Pathway to Induce EMT and Promote Lymph Node Metastasis in Cervical Cancer

Menghan Li Hui Wang*
WOMEN'S HOSPITAL, SCHOOL OF MEDICINE, ZHEJIANG UNIVERSITY

Purpose Lymph node metastasis (LNM) is one of the most significant determinants of poor prognosis in patients with cervical cancer (CESC) and greatly impacts the staging, treatment strategy, and survival outcomes. While epithelial-to-mesenchymal transition (EMT) has been implicated as a key molecular mechanism driving tumor invasion and metastasis, the specific molecular factors contributing to LNM in cervical cancer remain largely unclear. Therefore, this study aims to uncover the role of keratin 4 (KRT4) in LNM progression and examine the underlying pathways, particularly focusing on the interplay between KRT4, the Wnt/β-catenin signaling pathway, and EMT regulation.
Materials and Methods To identify genes associated with LNM in cervical cancer, we performed RNA sequencing (RNA-seq) on clinical cervical cancer tissues collected from patients with and without lymph node involvement (LN+ vs. LN-). Differentially expressed genes (DEGs) between LN+ and LN- groups were identified and subsequently compared with data from The Cancer Genome Atlas-Cervical

Squamous Cell Carcinoma and Endocervical Adenocarcinoma (TCGA-CESC) cohort for validation. Gene set enrichment analysis (GSEA) and pathway enrichment analysis were conducted to explore the biological pathways associated with identified DEGs, with a particular emphasis on EMT-related processes. Among these DEGs, KRT4 emerged as a potential regulator of EMT and Wnt/β-catenin signaling in LN+ cervical cancer.

To investigate the functional role of KRT4, cervical cancer cell lines (e.g., SiHa and HeLa cells) were subjected to KRT4 gain- and loss-of-function studies. Stable KRT4 overexpression or knockdown cell lines were established using lentiviral vectors. Changes in EMT markers (E-cadherin, N-cadherin, and Vimentin) and Wnt/β-catenin pathway components were evaluated via qRT-PCR, Western blot, and immunofluorescence assays. Further, the migratory and invasive abilities of cancer cells were assessed using transwell migration and invasion assays. Wnt/β-catenin pathway activity was quantified using TOP/FOP flash luciferase reporter assays. Additionally, clinical immunohistochemistry (IHC) of LN+ and LN- cervical cancer tissue samples was conducted to confirm KRT4 expression and its association with EMT markers in vivo.

Results RNA-seq analysis of LN+ and LN- cervical cancer tissues revealed a distinct gene expression signature, highlighting KRT4 as one of the most upregulated DEGs in LN+ samples. This finding was further corroborated by validation using TCGA-CESC data, where KRT4 expression was significantly higher in metastatic samples. GSEA and enrichment analyses revealed that KRT4-high tumors exhibited activation of EMT-associated pathways and the Wnt/β-catenin signaling cascade.

In vitro functional experiments demonstrated that overexpression of KRT4 significantly enhanced the migratory and invasive capacities of cervical cancer cells, accompanied by a decrease in the epithelial marker E-cadherin and an increase in mesenchymal markers N-cadherin and Vimentin, thereby confirming the role of KRT4 in inducing EMT. Immunofluorescence and Western blot analysis showed that KRT4 overexpression also led to increased nuclear accumulation of β-catenin, a hallmark of Wnt/β-catenin pathway activation. Conversely, KRT4 suppression reduced Wnt/β-catenin activity, evidenced by decreased β-catenin levels in the nucleus and reduced transcriptional activity of Wnt-responsive target genes (e.g., c-MYC, AXIN2). The effects of KRT4 knockdown were accompanied by diminished EMT marker expression and significant impairment of cancer cell migration and invasion potential.

Clinically, immunohistochemical analysis of LN+ cervical cancer tissues confirmed that high KRT4 expression was positively correlated with increased EMT marker expression (e.g., Vimentin) and nuclear β-catenin localization. These results support that KRT4 promotes LNM of cervical cancer by activating the Wnt/β-catenin pathway and driving the EMT process.

Conclusion This study identifies KRT4 as a pivotal molecular mediator of EMT in cervical cancer, where it drives metastatic progression by activating the Wnt/β-catenin signaling pathway. Our findings provide novel mechanistic insights into the role of KRT4 in cervical cancer metastasis, particularly its involvement in promoting LNM via EMT induction. KRT4 may therefore serve as a potential biomarker for predicting LNM risk and represents a promising therapeutic target for inhibiting metastasis in cervical cancer. Future studies could explore strategies to therapeutically inhibit KRT4 or its downstream effects to improve patient outcomes, particularly in cases with high metastatic risk.

PO_077

结构化健康教育处方对宫颈癌术后下肢淋巴水肿患者自我管理行为的影响

高庆
西安交通大学第一附属医院东院区

目的 探究结构化健康教育处方对宫颈癌术后下肢淋巴水肿患者自我管理行为的影响。

方法 采用随机对照试验，选取我院 100 例宫颈癌术后下肢淋巴水肿患者作为研究对象，随机分为观察组与对照组，每组各 50 例。对照组接受常规护理及健康教育，观察组在对照组基础上应用结构化健康

教育处方。该处方包含淋巴水肿知识科普、自我护理技能指导（如皮肤护理、压迫性包扎技巧、渐进式抗阻力训练等）、心理支持等内容。采用中文版淋巴水肿知识问卷（LEKQ）评估知识知晓度；下肢淋巴水肿自我管理量表（LESMQ）评价自我护理依从性；世界卫生组织生活质量测定量表简表（WHOQOL-BREF）衡量生活质量，同时定期测量下肢周径。

结果 干预后，观察组 LEKQ 得分（81.5±6.3）分显著高于对照组（64.2±5.8）分；LESMQ 得分（62.3±4.5）分明显高于对照组（53.1±5.2）分；WHOQOL-BREF 得分（75.6±7.1）分高于对照组（60.3±6.5）分；观察组下肢周径减小（2.3±0.5）cm，大于对照组的（1.1±0.4）cm，组间差异均具有统计学意义（P<0.05）。

结论 结构化健康教育处方能有效提高宫颈癌术后下肢淋巴水肿患者的自我管理行为水平，增强其对疾病知识的掌握，促进患者积极参与自我护理，从而减轻下肢水肿症状，提高生活质量。

PO_078

Clinical Efficacy and Safety of Adjuvant Chemotherapy in Locally Advanced Cervical Cancer Patients Achieving Complete Clinical Response (cCR) After Definitive Chemoradiotherapy

lulu Chen [1] chengjun Feng[1] zhan Lin[2] shaojun Chen[1*]
1.The Fourth Affiliated Hospital of Guangxi Medical University2.The Yulin First People's Hospital

Objective To investigate the clinical efficacy and safety of adjuvant chemotherapy (ACT) in patients with locally advanced cervical cancer (LACC) who achieved a complete clinical response (cCR) following definitive chemoradiotherapy (CRT), aiming to provide novel therapeutic strategies for improving survival in this population.

Methods This retrospective study enrolled patients with FIGO 2018 stage IB3, IIA2-IVA LACC treated at the Fourth Affiliated Hospital of Guangxi Medical University and Yulin First People's Hospital between January 2017 and December 2024, who achieved cCR after standard definitive CRT. Patients were divided into an ACT group (n=65) and an observation group (n=301) based on whether they received ACT. Five-year overall survival (OS), progression-free survival (PFS), distant metastasis-free survival (DMFS), and adverse event (AE) rates were compared between the groups before and after 1:1 propensity score matching (PSM). Prognostic factors were analyzed using Cox regression.

Results 366 patients were enrolled for analysis. Among them, 65 patients (17.8%) received adjuvant chemotherapy following definitive chemoradiotherapy (adjuvant chemotherapy group), and 301 patients (82.2%) received only definitive chemoradiotherapy (observation group). .Sixty-five patients received adjuvant chemotherapy (median:3 cycles, range:1-6 cycles), of which 50 were paclitaxel + platinum, 14 were docetaxel + platinum, and 1 was paclitaxel alone，while 301 underwent observation after initial treatment. The median follow-up time was 51 months (range, 1-134) for all patients, 38 months (range, 6-102) for the adjuvant chemotherapy group, and 51 months (range, 1-134) for the observation group, respectively. Of all patients, 84 patients (23.0%) experienced disease progression, including 41 patients (11.2%) with local recurrence, 33 patients (9.0%) with distant metastases, and 12 patients (3.3%) with both local recurrence and distant metastases. Before PSM: No significant differences were detected between the two groups on the 5-year OS was 82.5% (ACT) vs. 80.2% (observation) (P=0.933), 5-year PFS was 75.7% vs. 71.5% (P=0.446) and 5-year DMFS was 77.5% vs. 75.7% (P=0.982). No significant difference in grade 3-4 AE rates was observed (P>0.05). After PSM (65 matched pairs), also no significant differences were detected between the two groups on the 5-year OS was 82.5% vs. 83.5% (P=0.762), 5-year PFS was 75.7% vs. 75.6% (P=0.978) and 5-year DMFS was 77.5% vs. 78.3% (P=0.856). A significant increase in grade 3-4 hematologic toxicities was observed in the ACT group (P<0.05). Multivariate analysis identified advanced stage (IIIA-IVA) as an independent risk factor for poorer OS (HR=3.275, 95% CI 1.752-6.120, P<0.001) and PFS (HR=2.413,

95% CI 1.499-3.884, P<0.001). However, subgroup analysis within the Later-stage (2018FIGO IIIA-IVA) cohort showed no significant OS or PFS difference between ACT and observation (P>0.05).
Conclusion Adjuvant chemotherapy provided no survival benefit but significantly increased treatment-related toxicity in LACC patients achieving cCR after definitive CRT. Future strategies should employ precise molecular biomarkers to identify potential beneficiaries and avoid overtreatment.

PO_079

Efficacy of concurrent intensity modulated chemoradiotherapy combined with induction chemotherapy or adjuvant chemotherapy in the treatment of locally advanced cervical cancer with 2018FIGO stage correction

lulu Chen[1] mengzhuan Wei[1] manyun Liao[1] yurong Chen[1] guilan Yang[1] liqun Wei[1] chunlin Liu[1] xindan Wang[2] zhan Lin[3] chengjun Feng[1] li Hua[1] shaojun Chen[1*]
1.The Fourth Affiliated Hospital of Guangxi Medical University
2. Liuzhou Hospital,Guangzhou Women and Children's Medical Center
3.The Yulin First People's Hospital

Background and purpose Concurrent chemoradiotherapy (CCRT) is currently the standard treatment for locally advanced cervical cancer (LACC), but there is a lot of room for treatment strategies improvement for the survival rate of patients is low,Comprehensive treatment is the treatment direction for locally advanced cervical cancer, The current comprehensive treatment models for locally advanced cervical cancer mainly include induction chemotherapy (IC) plus concurrent chemoradiotherapy (CCRT) and CCRT plus adjuvant chemotherapy (AC),but it remains is unclear which is optimal sequence of combined chemotherapy. The purpose of this study is to retrospectively report the outcome of long-term survival and related toxicity that IC plus CCRT versus CCRT plus CCRT in LACC.
Materials/Methods From December 2016 to December 2022, 525 patients of LACC with 2009FIGO （IB2, IIA2, and IIB-IVA）who initially underwent IC plus CCRT or CCRT plus AC from the Forth Affiliated Hospital of Guangxi Medical University，Yulin First People's Hospital and Liuzhou Hospital of Guangzhou Women and Children's Medical Center.All patients were re-staged after 2018FIGO staging（IB3, IIA2, and IIB-IVA）correction.All patients were treated with IMRT to 50.4 Gy with concurrent weekly cisplatin 40 mg/m2 followed by intra-cavitary brachytherapy.The IC plus CCRT group received platinum-based combined chemotherapy with two or three cycles before CCRT. CCRT plus AC group received platinum-based combined chemotherapy with two cycles after CCRT. Using 1:1 propensity score matching (PSM),5-year overall survival (OS), 5-year progression free Survival survival (PFS),local recurrence-free survival (LRFS), distant metastasis-free survival (DMFS) and treatment-related adverse reactions were evaluated and compared between the two groups.
Results A total of 525 patients with complete follow-up data are available were included our study. Among these patients, under 250 patients went the IC plus CCRT and 275 patients were treated with the CCRT plus AC. IC/AC regimens included TP/DP(cisplatin+Paclitaxel/Docetaxel)andTC/DC(carboplatin ＋ Paclitaxel/Docetaxel). The median follow-up of 50 months (range10-80). Before PSM, there were no significant differences in survival rates (P > 0.05) between the groups. After PSM (191 matched pairs), also no significant differences were detected between the two groups on the 5-year OS rates (83.9 % vs. 84.7%), PFS rates (76.0 % vs. 71.4%) or LRFS rates (79.3 % vs 76.5 %）and DMFS rates (88.7 % vs 89.1%)) (all P > 0.05). Subgroup analysis revealed that CCRT plus AC showed a trend of improving OS (HR= 0.455, 95% CI (0.206-1.006), P =0.052) in 2018FIGO IIIB-IVA stage and was associated with significantly improved OS(HR=0.191,95%CI (0.057-0.641),P=0.007), PFS(HR=0.288,95%CI(0.118-0.702), P =0.006) ,DMFS(HR= 0.211, 95% CI (0.068-0.654), P =0.007) in patients with positive pelvic lymph

nodes. IC plus CCRT was associated with significantly improved PFS (HR= 2.276, 95% CI (1.187-4.363), P=0.013) in 2018FIGO IIB. There was no significant difference in adverse events (acute toxicities and late toxicities) between the two groups (P > 0.05).

Conclusion There is no statistical difference between IC plus CCRT and CCRT plus AC in the outcome of long-term survival of LACC. However, for patients with stage III-IVA advanced cervical cancer, the CCRT plus AC shows a trend of improving survival benefits, especially for those with pelvic lymph node metastasis, where the benefit is more significant. For stage IIB patients, IC plus CCRT can reduce the risk of local progression. Lymph node status is a key decision factor: patients with positive pelvic lymph nodes should prioritize CCRT plus AC for enhanced treatment.

PO_080

Apoptotic Cell-Derived Extracellular Vesicles MTA1 Promote Radioresistance in Cervical Cancer via Cellular Dormancy

Xiaojing Chen[1] Zhizai Chen[1] Shanyang He[1] Yuanrun Deng[2*]
1.Department of Gynecology, Guangdong Provincial People's Hospital (Guangdong Academy of Medical Sciences), Southern Medical University, Guangzhou 510080, China.
2.Department of Obstetrics and Gynecology, The Third Affiliated Hospital, Southern Medical University, Guangzhou 510630, China

Background Radioresistance remains a major hurdle in the clinical management of cervical cancer (CC). Emerging evidence indicates that apoptosis-driven "onco-regenerative niches" contribute to treatment resistance, yet the molecular mechanisms by which apoptotic cells transmit radioresistance traits to surviving tumor cells remain elusive.
Methods Bioinformatics analysis of GEO datasets identified metastasis-associated protein 1 (MTA1) as a key candidate regulator of radioresistance. Western blotting and fluorescence microscopy validated MTA1 expression and its intercellular transfer via apoptotic cell-derived extracellular vesicles (apoEVs). Radiosensitivity was assessed using clonogenic survival assays and Annexin V/PI staining. Cellular dormancy was evaluated via glucose metabolism assays and cell cycle analysis. The interaction between STAT1 and NR2F1 was investigated using chromatin immunoprecipitation (ChIP) and dual-luciferase reporter assays. In vivo radioresistance was confirmed in irradiated nude mouse xenograft models.
Results Clinically, apoptotic tumor cell accumulation correlated strongly with radioresistance in CC patients. Mechanistically, MTA1 was selectively enriched in apoEVs and transferred to neighboring viable tumor cells. Caspase-3 activation triggered MTA1 nuclear export and subsequent encapsulation into apoEVs. Notably, apoEVs from apoptotic cells harbored higher MTA1 levels than those from viable cells, and their uptake by recipient cells conferred radioresistance, protecting against radiation-induced apoptosis. Depletion of MTA1 in apoEVs abrogated this protective effect. At the molecular level, MTA1 upregulated STAT1 signaling, which directly transactivated NR2F1—a master regulator of cellular dormancy. In vivo, MTA1-laden apoEVs significantly enhanced tumor radioresistance and shortened survival in irradiated xenografts.
Conclusions This study is the first to demonstrate that apoEVs mediate intercellular transfer of MTA1, inducing tumor dormancy and radioresistance in CC via the STAT1/NR2F1 axis. Targeting the apoEV-MTA1/STAT1/NR2F1 pathway may represent a novel therapeutic strategy to overcome radioresistance in cervical cancer.

PO_081

基于宿主六基因甲基化的预测 HR-HPV 阳性女性宫颈病变的风险预测模型的建立

薛璐洁 王梦媛 张璐 童旭颖 李玢* 陈丽宏
陕西省人民医院

目的 建立基于宿主六基因甲基化的预测 HR-HPV 阳性女性宫颈病变的风险预测模型并进行评价。
材料与方法 选取 2023 年 5 月至 2024 年 1 月在陕西省人民医院妇科门诊就诊的符合纳排标准的 104 例病例，将所有临床资料进行单因素回归分析，将有统计学意义的甲基化检测结果及临床特征分别进行多因素回归分析，建立六基因甲基化试剂盒和 ASTN1、DLX1、ITGA4、RXFP3、SOX17、ZNF671 单基因位点预测 CIN2 及以上宫颈病变进展风险的列线图模型，评估比较其灵敏度、特异度、准确率、AUC 指数及约登指数等指标。
结果 104 例患者中，慢性宫颈炎 43 例（41.34%），CIN1 19 例（18.26%），CIN2 10 例（9.61%），CIN3 20 例（19.23%），子宫颈癌 12 例（11.53%）（其中 10 为鳞状细胞癌，2 例为腺癌）。六基因甲基化阳性率在 CIN1、CIN2、CIN3 及子宫颈癌中分别为 10.53%、50%、75%、100%，宫颈病变程度越高，六基因甲基化阳性率越高；宫颈光滑、阴道炎、TCT 异常、六基因甲基化阳性是 HR-HPV 阳性女性 CIN2 及以上宫颈病变的独立影响因素；由六基因甲基化建立预测模型的 C-index 为 0.881（95%CI：0.763-0.913）；六基因甲基化预测模型和单基因位点（ASTN1、ITGA4、RXFP3、SOX17、ZNF671）预测模型的 AUC 值依次为：ZNF671（0.820）>六基因甲基化（0.792）>SOX17（0.757）>ITGA4（0.698）>RXFP3（0.686）>ASTN1（0.661），且差异有统计学意义（P<0.05）。
结论 基于六基因甲基化的联合风险预测模型诊断 HR-HPV 阳性女性宫颈病变有较高的预测价值；单独使用 ZNF671 检测预测 CIN2 及以上宫颈病变的效能较高，与六基因甲基化诊断效能相当。

PO_082

Nuclear PD-L1⁺ Tumor-Associated Macrophages in Hypoxic Margins: A Prognostic and Predictive Biomarker for Immunotherapy Response in Cervical Cancer

Xiaojing Chen[1] Chenfei Zhou[1] Wenfei Wei[2] Yuanrun Deng[3*]
1.Department of Gynecology, Guangdong Provincial People's Hospital (Guangdong Academy of Medical Sciences), Southern Medical University, Guangzhou 510080, China
2.Department of Gynecology, The Eighth Affiliated Hospital of Southern Medical University, Foshan 528300, China
3.Department of Obstetrics and Gynecology, The Third Affiliated Hospital, Southern Medical University, Guangzhou 510630, China

Background Despite the promising efficacy of PD-1/PD-L1 checkpoint inhibitors in advanced cervical cancer (CC), primary or acquired resistance remains a major clinical challenge, with accumulating evidence suggesting that spatial heterogeneity in PD-L1 expression contributes to this variability. Herein, we sought to investigate the density, spatial distribution, and clinical relevance of nuclear PD-L1 (nPD-L1)-expressing tumor-associated macrophages (TAMs) within hypoxic tumor microenvironments (TME), particularly their association with immunotherapy response and patient outcomes.
Methods A total of 103 CC tissue specimens were analyzed using immunohistochemistry (IHC) and multiplex immunofluorescence staining to quantify CD163⁺ TAMs, PD-L1, and nPD-L1 expression, with stratification into tumor hypoxic center (HC) and hypoxic margin (HM) based on CAIX (a hypoxia marker) staining. Correlation analyses with clinicopathological features and survival outcomes were

performed, and a combined predictive model for immunotherapy response was constructed using logistic regression, validated by receiver operating characteristic (ROC) curves.
Results Our results revealed distinct spatial distribution patterns: nPD-L1$^+$ TAMs and PD-L1$^+$ TAMs were more abundant in the HM than in the HC, with nPD-L1 predominantly localized in CD163$^+$ TAMs surrounding hypoxic regions, especially at the HM. Notably, high densities of HM nPD-L1$^+$ TAMs were strongly associated with aggressive clinicopathological features (including lymph node metastasis, advanced FIGO stage (IIB-IV), poor differentiation, and large tumor size (>4 cm)) and unfavorable overall survival (OS). Moreover, the combined detection of nPD-L1 and CD163$^+$ TAMs at the HM yielded the highest predictive accuracy for immunotherapy response, with an area under the ROC curve (AUC) of 0.9551 ($P < 0.0001$), outperforming either marker alone.
Conclusions Collectively, these findings highlight that nPD-L1$^+$ TAMs in the HM serve as a robust predictive biomarker for immunotherapy response and prognosis in CC, offering potential implications for optimizing therapeutic strategies.

PO_083

磁共振影像组学预测局部晚期宫颈鳞癌对子宫动脉介入化疗栓塞术敏感性的研究

薛璐洁 朱昱哲 李玢* 陈丽宏
陕西省人民医院

目的 建立基于影像组学的预测对子宫动脉介入化疗栓塞术敏感的局部晚期子宫颈癌患者的模型，制定个体化治疗方案。
方法 回顾性收集陕西省人民医院 2016 年 4 月至 2021 年 8 月符合纳排标准的宫颈癌患者临床资料与磁共振影像资料，通过 ROI 靶区勾画、标准化预处理、特征提取，并对特征进行筛选。将所有病例根据实体瘤疗效评价标准定义对应标签，按照 7:3 比例随机分为训练集和测试集。构建出双序列和多序列共 3 个影像组学模型，选取 AUC 值最高的影像组学特征，用于建立联合模型。另外，对临床因素进行单因素及多因素 logistics 回归分析，构建临床模型。将临床因素与影像组学结合，建立基于临床和影像组学的介入化疗栓塞术疗效的联合预测模型，并绘制列线图，计算 ROC 曲线下面积 AUC，并比较各个模型之间的性能差异。
结果 共纳入 83 例局部晚期宫颈癌患者，在（T1WI 瘤内+T1WI 瘤周）、（T2WI 瘤内+T2WI 瘤周）、（T1WI 瘤内+T1WI 瘤周+T2WI 瘤内+T2WI 瘤周）区域内分别提取特征，建立双序列及多序列联合影像组学预测模型。3 个影像组学模型中多序列模型的 AUC 值最高为 0.921。本研究对 8 个临床因素进行单因素和多因素 logistics 回归，证实孕次（P=0.039）、病理分级（P=0.007）是介入化疗栓塞术敏感性的独立预后因素，建立的临床模型 AUC 值为 0.785。选择 AUC 最高的影像组学特征，将临床因素与多序列影像组学特征结合构成联合模型，在训练集与测试集中，联合模型的 AUC 值均高于临床模型、多序列影像组学模型（P＞0.05）。
结果 本研究构建的影像组学模型、临床模型和联合模型均有良好的预测子宫动脉介入化疗栓塞术敏感性能力。基于临床因素和多序列影像组学特征的联合模型的预测性能优于临床、影像组学等单一模型，在预测局部晚期宫颈癌介入化疗栓塞术上具有较高的敏感性。

PO_084

《CYP19A1 基因在子宫颈癌免疫微环境调控及免疫治疗响应预测中的作用探究》

何子嫣 迪力亚尔·阿不都克热木
天津医科大学

研究目的 本研究旨在系统评估 CYP19A1 基因在肿瘤免疫微环境调控、临床特征关联及免疫治疗响应预测中的作用，并探索其作为新型生物标志物的潜在价值，以期为子宫内膜癌（UCEC）及其他肿瘤的精准免疫治疗提供理论依据。
材料与方法 基于 GSE146889、GSE106191 及 TCGA_UCEC 等公共数据集，以 UCEC 为主要研究对象，整合抗 PD-1/PD-L1、抗 CTLA-4 和 CAR-T 治疗的临床队列（如 IMvigor210、GSE91061）。采用功能富集分析（GO/KEGG）解析 CYP19A1 的生物学功能，通过 Wilcoxon 检验、Cox 回归及 ROC-AUC 评估其与微卫星不稳定（MSI）状态、基因突变（如 ARHGAP35）、免疫细胞浸润（CD8+ T 细胞、巨噬细胞）、免疫检查点分子（PD-L1、CTLA-4）表达及患者生存预后的关联。
结果 CYP19A1 在 UCEC 中显著富集于嗅觉感知（GO:0007608，p<0.01）、G 蛋白偶联受体信号传导（GO:0004930）及核糖体组成（GO:0005840），KEGG 通路提示其参与嗅觉传导（ko04740）及神经活性配体-受体互作（ko04080）。临床特征分析显示，CYP19A1 高表达与 MSI-H 状态正相关（p=0.031），且与 ARHGAP35 突变频率降低显著关联（p<0.01）。免疫微环境中，CYP19A1 与 CD8+ T 细胞及巨噬细胞浸润呈弱正相关，但与免疫检查点分子无显著共表达。生存分析表明，CYP19A1 高表达可改善 UCEC 患者总生存期（OS，HR=0.72，p=0.018）及无进展生存期（PFS，HR=0.68，p=0.011），但在泛癌免疫治疗队列中，其表达水平与治疗响应率及生存获益无显著关联（p>0.05）。ROC-AUC 分析显示，CYP19A1 对免疫治疗响应的预测效能有限（AUC=0.658-0.667）。
结论 CYP19A1 在 UCEC 中可能通过调控 MSI 状态及基因突变影响预后，但其与免疫微环境的直接作用机制尚未明确，且作为单一标志物预测免疫治疗响应的临床价值有限。未来需构建多基因联合模型或开展肿瘤类型特异性研究以提升其转化潜力，并进一步通过功能实验阐明其生物学机制。本研究受限于队列异质性及机制验证不足，后续需扩大样本量并深化机制探索。

PO_085

GGT6 inhibitis cervical cancer through the Metabolic Reprogramming-Immunoregulation Axis

xiaoliu wu Mei Wang Yang Gao Xiaoxia Jiang le He Hongying Yang Zheng Li*
Peking University Cancer Hospital Yunnan Hospital/ Yunnan Cancer Hospital /The Third Affiliated Hospital of Kunming Medical University

Objective (s) Cervical cancer is a common gynecological malignancy. The Warburg effect within tumor cells promotes cell proliferation and metastasis through metabolic reprogramming, representing a new direction in cancer research. Through bioinformatics analysis, this study identified γ-glutamyltransferase 6 (GGT6), an energy metabolism gene associated with cervical cancer prognosis. We constructed and validated a GGT6-based prognostic model for cervical cancer. Validation in our institution's retrospective cohort confirmed that GGT6 is associated with favorable prognosis in cervical cancer. Via in vitro cellular experiments and animal studies, we demonstrated that γ-glutamyltransferase 6 (GGT6) inhibits cervical cancer progression through the energy metabolism reprogramming-immunomodulation axis, providing novel therapeutic directions and targets for cervical cancer treatment.
Methods Utilizing the TCGA/GEO databases and Reactome metabolic pathways, we identified the energy metabolism gene GGT6 as significantly differentially expressed in cervical cancer. A risk score

model was constructed, validating GGT6 as a protective factor in cervical cancer. Analysis of a retrospective clinical cohort study from 2016 revealed a significant negative correlation between GGT6 expression and patient prognosis as well as clinicopathological characteristics. Validation through the detection of fresh cervical cancer tissues and adjacent normal tissues confirmed the differential expression of GGT6 in tumor versus normal tissues. Combined with in vitro cellular experiments using cervical cancer cell lines transfected with GGT6-overexpressing vectors and animal studies, GGT6 was confirmed as a protective factor in cervical cancer. Metabolomic and transcriptomic analyses, followed by validation, demonstrated that GGT6 influences the occurrence and development of cervical cancer through the energy metabolism reprogramming-metabolic regulation axis.

Results

1. Prognostic Prognostic stratification using the GGT6 risk score revealed significant differences in survival rates between the high-risk and low-risk groups in the training set (P=0.044), internal validation set (P=0.006), and external validation set (P=0.0043).
2. Analysis of the tumor immune microenvironment (TIM) demonstrated significant differences in immune/stromal scores (P<0.05) and variations in immune cell infiltration (e.g., NK CD56 bright cells, Tregs, etc.) between the risk groups. Low-risk patients exhibited higher responsiveness to immune checkpoint inhibitors (TIDE, P<0.05). GGT6 expression showed a positive correlation with CD56+ cell infiltration.
3. A 2016 institutional retrospective cohort confirmed that GGT6 protein expression is an independent prognostic biomarker (P<0.05), correlating with age, carcinoembryonic antigen (CEA) levels, and histology. A predictive model incorporating GGT6, International Federation of Gynecology and Obstetrics (FIGO) stage, and lymph node metastasis effectively predicted surgical outcomes in early-stage patients.
4. In vitro experiments conducted on GGT6-overexpressing HeLa and SiHa cells demonstrated that GGT6 inhibits cervical cancer cell proliferation, promotes apoptosis, and regulates cell cycle progression.
5. In vivo studies using a subcutaneous xenograft tumor model in animals inoculated with GGT6-expressing SiHa cells showed that GGT6 significantly suppresses cervical cancer cell growth.
6. Integrated transcriptomic and metabolomic analysis of GGT6-transfected cervical cancer cells suggested that its ability to inhibit cancer cell proliferation may be associated with the glucagon signaling pathway, central carbon metabolism in cancer, beta-alanine metabolism, and insulin resistance.

Conclusion Through comprehensive analysis of multi-omics data and clinical validation cohorts, metabolism-associated protein GGT6 was identified as a novel prognostic biomarker for cervical cancer. An integrated model combining GGT6 expression with clinicopathological parameters demonstrated strong predictive accuracy for long-term survival outcomes (overall survival, OS). Mechanistic studies revealed that GGT6 exerts a tumor-suppressive effect in cervical cancer through the energy metabolism reprogramming-immunomodulation axis. Collectively, these findings underscore the dual role of GGT6 as both a prognostic indicator and a potential therapeutic target.

PO_086

EDEM3 as a prognostic biomarker and immunotherapeutic target to promote cervical cancer

Qiao Hui[1] Ping Yang[2*]
1.Department of Obstetrics and Gynecology,Xinjiang Production and Construction Corps Hospital,
2.First Affiliated Hospital,School of Medicine,Shihezi University,Shihezi,China

Objective To analyze the relationship between the expression of ER Degradation Enhancing Alpha-mannosidase Like Protein 3 (EDEM3) in cervical cancer tissue (CCT) and the clinicopathological characteristics and prognosis of patients; to preliminarily investigate whether downregulating EDEM3

expression attenuates the proliferation and matastasis capabilities of cervical cancer cells, thereby identifying a potentially valuable novel therapeutic target for patients with advanced cervical carcinoma (CC).

Methods

1. Utilize the GENT2 and TCGA databases to analyze the relationship between EDEM3 expression levels in cervical cancer tissue (CCT) and patient prognosis.
2. Apply algorithms such as ESTIMATE and CIBERSORT to analyze differences in immune infiltration in cervical cancer tissues between different EDEM3 expression groups.
3. Download data on common drugs and small-molecule compounds from the Cancer Drug Sensitivity Genomics (GDSC) database and assess whether there are differences in sensitivity to various chemotherapeutic and targeted drugs between different EDEM3 expression groups.
4. Obtain somatic mutation data from the TCGA database to analyze somatic mutation profiles in cervical cancer patients across different EDEM3 expression groups and calculate their Tumor Mutational Burden (TMB). Investigate the correlation between EDEM3 expression and TMB, and use Kaplan-Meier curves to evaluate prognostic differences among patients in distinct groups.
5. Conduct Cox univariate and multivariate regression analyses, in conjunction with clinicopathological characteristics (including FIGO stage, tumor grade, and histological type), to identify risk factors influencing the prognosis of cervical cancer patients and construct a relevant prognostic model.
6. Collect clinical information and tissue specimens from 119 early-stage cervical cancer patients who underwent surgery. Detect EDEM3 protein expression in cervical cancer tissue (CCT) and normal cervical tissue (NCT) using Immunohistochemistry (IHC). Analyze the correlation between EDEM3 expression and clinicopathological characteristics via chi-square test, and assess its association with Overall Survival (OS) and Progression-Free Survival (PFS) in patients using Kaplan-Meier curves.
7. Establish stable EDEM3-downregulated cervical cancer HeLa and SiHa cell lines through lentiviral transfection. Validate the knockdown efficiency using Western blot analysis.
8. Examine the effects of different EDEM3 expression levels on the proliferative capacity of cervical cancer HeLa and SiHa cells via EdU cell proliferation assay and CCK-8 cell proliferation assay.
9. Employ Transwell migration and invasion assays , along with wound healing assays, to examine the changes in metastasis capabilities of HeLa and SiHa cervical cancer cells under different EDEM3 expression levels.

Results

1. Analysis of GENT2 and TCGA data revealed that EDEM3 is highly expressed in cervical cancer and associated with poor prognosis (P <0.05).
2. Cox regression analysis of downloaded TCGA data indicated that EDEM3 expression levels and tumor FIGO stage are independent risk factors for the prognosis of cervical cancer patients.
3. Based on somatic mutation data, cervical cancer patients in the EDEM3_L+TMB_L group exhibited the best prognosis.
4. The expression of EDEM3 in cervical cancer tissues influences the tumor immune microenvironment.
5. Analysis of GDSC data indicated that the IC50 values of commonly used chemotherapeutic drugs for cervical cancer, such as Cisplatin and Topotecan, are associated with EDEM3 expression.
6. Immunohistochemistry experiments confirmed that EDEM3 expression levels were significantly higher in cervical cancer tissues (CCT) than in normal cervical tissues (NCT) (P <0.001). High EDEM3 expression was closely correlated with FIGO stage (2018), lymph node metastasis, lymphovascular invasion and depth of cervical stromal invasion in cervical cancer patients (P <0.05).
7. Kaplan-Meier curve analysis of patient follow-up results revealed that cervical cancer patients with high EDEM3 expression had a worse prognosis. Cox regression analysis indicated that high EDEM3 expression and depth of cervical stromal invasion were independent risk factors for overall survival (OS) and progression-free survival (PFS) (P <0.05).
8. Lentiviral transfection-mediated downregulation of EDEM3 expression showed a more significant inhibitory effect on the proliferation and metastasis of cervical cancer (CC) cells compared to the control group (P <0.05).
9. Through differential expression analysis to screen differentially expressed genes, a prognostic risk model based on three EDEM3-related genes (ERGs) affecting cervical cancer prognosis was ultimately

constructed, and a corresponding nomogram was developed. When compared to the EDEM3-related cervical cancer prognosis model, this new model demonstrated better predictive power and accuracy.
Conclusions
1. EDEM3 is highly expressed in cervical cancer tissues (CCT), and its expression level is closely correlated with FIGO stage (2018), lymph node metastasis, lymphovascular invasion and depth of cervical stromal invasion in cervical cancer patients. High EDEM3 expression predicts poor prognosis.
2. Downregulation of EDEM3 expression in cervical cancer cell lines attenuates the proliferation and metastasis abilities of cancer cells.
3.The prognostic risk model based on three prognosis-related EDEM3-related genes (ERGs) demonstrates favorable predictive power and accuracy, particularly showing higher predictive value for overall survival (OS) in cervical cancer patients.

PO_087

结合贝叶斯验证的孟德尔随机化识别肠道菌群-宫颈癌轴中的免疫细胞介质

薛伟伟 王展
青海大学附属医院

目的 本研究旨在探讨肠道微生物群（GM）、免疫表型（IC）与宫颈癌（CC）之间的因果关系，重点分析 IC 在 GM 影响 CC 过程中的中介作用，为 CC 的早期预防和多靶点治疗提供新见解。
方法 依据 STROBE-MR 指南，采用双样本孟德尔随机化（TSMR）分析，利用大规模 GWAS 汇总数据，并严格筛选工具变量。通过中介 MR 框架评估 GM-IC-CC 之间的因果路径，使用逆方差加权（IVW）回归为主方法，辅以加权中位数、MR-Egger 与 MR-PRESSO 控制多效性偏倚。进一步采用 BWMR 与 GSMR 方法对关键发现进行稳健性验证。
结果 MR 分析揭示了通过特定免疫机制将 GM 与 CC 联系起来的四条统计学上稳健的中介途径。HLA DR on B cell 表达介导了 Eisenbergiellasp900066775 对 CC 的保护作用（OR = 0.762，95% CI：0.640-0.907，P= 0.002），而 Central Memory CD4-CD8- T cell 增强了 GCA-900199385sp900320755 的致癌影响（OR = 1.786，95% CI：1.213-2.628，P= 0.003）。FSC-A on Natural Killer 与 Holdemania massiliensis 相关的 CC 抑制成反比（OR = 1.251，95% CI：1.054-1.486，P= 0.010），CD25 on CD39+ activated CD4 regulatory T cell 表达减弱了 MethanobacteriumB 的抗肿瘤效应（OR = 1.775，95% CI：1.146-2.749，P= 0.010）。敏感性分析证实偏倚极小，BWMR 和 GSMR 结果进一步加强了因果有效性。
结果 本研究从遗传学层面揭示了 GM 通过免疫表型调控参与 CC 发生的机制，突显了肠-免疫轴在宫颈癌防治中的潜在价值。未来需在多中心人群中进一步验证，并探索微生态干预策略的临床应用前景。

PO_088

肿瘤细胞通过 IGSF3-TNFR2 轴与 Treg 细胞互作，驱动宫颈癌免疫抑制。

何广平 瞿欣瑜 邱君君*
复旦大学附属妇产科医院

背景 宫颈癌是全球女性第四大常见恶性肿瘤，尽管免疫检查点阻断（ICB）疗法已成为复发/转移性宫颈癌的一线治疗，但其客观缓解率（ORR）仍不足 30%。这一持续存在的临床挑战凸显了阐明肿瘤细胞免疫逃逸替代途径的必要性，以开发更有效的治疗策略。

方法 本研究利用宫颈癌单细胞测序结果及 TCGA 公共数据库开展联合分析挖掘宫颈免疫逃逸新靶标，构建靶向敲低 IGSF3 的宫颈癌细胞系后通过体外共培养实验和荷瘤小鼠模型评估免疫细胞亚群变化。联合 RNA 测序与免疫沉淀-质谱（IP-MS）技术筛选相关互作蛋白，并利用 AlphaFold 进行蛋白互作位点预测，构建基因突变体验证结合域功能。采用基于人工智能的药物虚拟筛选识别新型 IGSF3 抑制剂，并通过体外结合实验和体内药效实验验证其作用。
结果 研究发现 IGSF3 在宫颈癌细胞表面异常高表达，并与 Treg 表面 TNFR2 形成新型配体-受体互作轴。结构分析证实 IGSF3 通过特定结构域与 TNFR2 结合，激活下游 NF-κB 信号通路，增强 Treg 免疫抑制功能。基于结构虚拟筛选发现天然化合物 purpurogallin 能高效阻断 IGSF3-TNFR2 相互作用，显著降低 Treg 浸润比例，增强 CD8+ T 细胞毒性，抑制肿瘤生长。同时，与 ICB 联合治疗相比，purpurogallin 预处理后序贯 PD-1 阻断方案展现出更优异的治疗效果。
讨论 本研究首次揭示 IGSF3-TNFR2 轴是宫颈癌免疫逃逸的新机制，并开发出靶向该通路的小分子抑制剂 purpurogallin。所提出的 purpurogallin-抗 PD-1 序贯免疫治疗策略为克服宫颈癌 ICB 耐药提供了新思路，具有重要临床转化价值。

PO_089

宫颈 HPV 持续性感染治疗进展

黄娅[1] 黄革[1] 李兰[2] 李娜[1*]
1.遵义医科大学第二附属医院 2.川北医学院附属医院

宫颈感染人乳头瘤病毒(Human Papillomavirus，HPV)非常普遍,大部分女性可通过宿主免疫反应清除 HPV,感染通常呈一过性,但仍有小部分 HPV 感染持续存在并最终发展成子宫颈癌。HPV 持续性感染是子宫颈癌的重要致病因素,HPV 感染的持续性受宿主因素(遗传、免疫抑制和社会行为)和病毒因素(基因型、病毒载量和整合)的影响。近年来宫颈 HPV 持续性感染的治疗方法包括免疫调节剂、抗病毒治疗、物理治疗、手术治疗、中医中药等方面，故本文综述近年来药物治疗（如免疫调节剂、抗病毒药物）、免疫疗法（治疗性疫苗、检查点抑制剂）及物理治疗等领域的最新进展，为临床医生提供全面的治疗思路，有利于提高宫颈 HPV 持续性感染的清除率，降低宫颈癌的发生风险。持续性 HPV 感染可通过多种机制增加宫颈上皮内瘤变和宫颈癌的风险,不同治疗方式的有效性不一，需依据病人的发病机制选取相应的治疗方法，提高宫颈 HPV 持续性感染的治疗效果及减少宫颈上皮内瘤变和宫颈癌的风险。

PO_090

Investigation of the Molecular Mechanism of SKA2 in Promoting Cervical Cancer Metastasis through the Akt/GSK-3β/β-catenin Pathway

Yujie Hao Xiaoxia Jiang Zheng Li*
Yunnan Cancer Hospital, The Third Affiliated Hospital of Kunming Medical University, Peking University Cancer Hospital Yunnan.

Objective(s) Spindle-kinetochore associated complex 2 (SKA2) is a protein-coding gene whose product is a major component of the SKA complex, involved in the regulation of cell mitosis. SKA2 is abnormally overexpressed in cervical cancer and can affect the invasion and migration of cervical cancer cells in vitro. The mechanism may involve the Akt/GSK-3β signaling axis activating the Wnt/β-catenin pathway to induce epithelial-mesenchymal transition (EMT). This study aims to demonstrate that SKA2 may activate the Akt/GSK-3β signaling axis through

dephosphorylation of spindle assembly checkpoint (SAC) proteins in cervical cancer cells using bioinformatics analysis, transcriptome sequencing,overexpression and knockdown experiments, xenograft tumor models, and clinical sample validation. Through the Wnt/β-catenin pathway, EMT is induced to promote cervical cancer metastasis, indicating that SKA2 could serve as a potential biomarker and therapeutic target for metastasis and recurrence.

Methods

1、The expression of SKA2 in the TCGA database was analyzed.

2、The expression levels of SKA2 mRNA and protein in cervical epithelial cells, cervical cancer cell lines, paired cervical cancer tissues, and adjacent normal tissues were detected using RT-qPCR, Western blotting, and immunohistochemistry.

3、Cervical cancer cell lines with exogenous overexpression or endogenous SKA2 interference and control cells were established using lentiviral vectors. Transwell assay was used to detect cell invasion, and wound healing assay was used to assess cell migration.

4、In cervical cancer cell lines with exogenous overexpression or endogenous SKA2 interference and control cells, Snail was detected by RT-qPCR, and E-cadherin, Vimentin, and N-cadherin were detected by Western blotting. The key molecules of the Wnt/β-catenin pathway (activated β-catenin, phosphorylated Akt, and GSK-3β) were also analyzed.

5、Bioinformatics was used to identify SKA2 highly expressed single-gene enriched pathways and co-expressed genes. Transcriptome sequencing was employed to identify SKA2-related differentially expressed genes and enriched pathways.

Results

1、TCGA database analysis showed that SKA2 expression was upregulated in cervical cancer tissues compared with normal tissues.

2、The expression levels of SKA2 mRNA and protein in cervical cancer cell lines and fresh cancer tissues were higher than those in normal cervical epithelial cells and adjacent normal tissues.

3、Overexpression of SKA2 enhanced the invasion and migration capabilities of cervical cancer cell lines and upregulated Snail mRNA expression. Knockdown of endogenous SKA2 expression reversed these effects.

4、Western blotting demonstrated that E-cadherin was downregulated, while Vimentin, N-cadherin, and activated β-catenin were upregulated in cervical cancer cells overexpressing SKA2. Knockdown of endogenous SKA2 expression partially restored E-cadherin and downregulated Vimentin, N-cadherin, and activated β-catenin. Exogenous SKA2 overexpression or interference with endogenous SKA2 expression affected the levels of phosphorylated Akt and GSK-3β, but had no effect on total Akt and GSK-3β .

5、TCGA combined with transcriptome sequencing revealed that PI3K-AKT-mTOR signaling was the main pathway enriched in SKA2 overexpression, and SKA2 expression correlated with MAD2 and BUBR1 expression.

6、Animal model experiments showed that subcutaneous inoculation of cervical cancer cell lines with interfering endogenous SKA2 for 28 days resulted in smaller tumor volumes compared with control cell lines. After 28 days of footpad inoculation, the lymph node metastasis rate was lower than that of the control cell line.

Conclusion(s)

1、SKA2 is highly expressed in cervical cancer cell lines and cervical cancer tissues.

2、Overexpression of SKA2 can promote the invasion and migration of cervical cancer cells in vitro, while interference with endogenous SKA2 expression has the opposite effect.

3、The effect of SKA2 on the invasion and migration of cervical cancer cells in vitro may be related to EMT mediated by the Wnt/β-catenin pathway, which is regulated by MAD2, BUBR1, and the Akt/GSK-3β signaling axis.

PO_091

Metabolic Coupling between Gardnerella vaginalis and Cervical Epithelium Sustains HPV-16 Oncogene Expression via N-α-Acetyl-L-Arginine-Mediated Activation of the Non-Canonical NF-κB Pathway

Yue Wang[1] Lihua Chen[1] Yuxuan Huang[1] Dan Li[2] Ye Li[1] Wenyu Lin[1] Dingjie Wang[1] Binhua Dong[1] Wei Wang[2] Pengming Sun[1*]

1.Fujian Maternity and Child Health Hospital

2.National Center of Technology Innovation for Animal Model, State Key Laboratory of Respiratory Health and Multimorbidity

Persistent HR-HPV infection is a leading cause of cervical cancer. The interplay between the vaginal microbiota and HPV persistence remains unclear. Here, we identify N-α-acetyl-L-arginine (NAALA) as a key metabolite in Gardnerella vaginalis (GV)-HPV-16 co-infection using multi-omics analyses of clinical cohorts. We show that NAALA, produced by GV, promotes the persistent expression of HPV-16 oncogenes via the NF-κB non-canonical pathway. NAALA suppresses glutathione peroxidase (GPx) activity, leading to ROS accumulation that activates NF-κB p52 nuclear translocation and enhances its binding to the HPV-16 E6 promoter. This process is further reinforced by a positive feedback loop where NAALA activates host N-acetyltransferases to amplify its own synthesis. Additionally, NAALA suppresses M1 macrophage polarization, creating an immunosuppressive microenvironment that favors HPV persistence. Our study highlights NAALA as a critical driver of HPV-16 persistence and cervical carcinogenesis in the context of vaginal dysbiosis, and suggests potential therapeutic targets for intervention.

PO_092

IKBIP as a prognostic biomarker and immunotherapeutic target regulates JAK-STAT3 signaling pathway to promote cervical cancer

Wang Yan[1] Qiao Hui[2] Ping Yang[3*]

1.Department of Gynecology, people's Hospital of Xinjiang Uygur Autonomous Region

2.Department of Obstetrics and Gynecology,Xinjiang Production and Construction Corps Hospita

3.First Affiliated Hospital,School of Medicine,Shihezi,University,Shihezi,China

Objective Cervical cancer (CC) represents a significant threat to women's health globally. Although IKBIP has been identified as an oncogene, its role in cancer has been rarely documented, and it has predominantly been associated with driving malignant progression in gliomas. Further investigation into the function of IKBIP in other cancer types, including CC, is essential to fully understand its potential implications for tumorigenesis and progression in various malignancies.

Methods IKBIP expression was analyzed in CC tissues using the GEPIA and GEO databases. Transcriptomic data and clinical characteristics from 306 CC patients were obtained from TCGA and clustered utilizing X-tile software. Additionally, to validate the prognostic significance of IKBIP, protein levels in normal and cancerous tissues were compared through IHC. The TIDE score was employed as an indicator of the response to immunotherapy. Furthermore, our study aimed to explore the potential relationship between IKBIP and immunological genes, as well as their influence on the development of TMB and drug sensitivity. The effects of IKBIP on the proliferation, migration, and invasion capabilities of CC cells were investigated using CCK-8, EdU, and transwell assays. To further elucidate the role of IKBIP in regulating the JAK-STAT signaling pathway and its contribution to the

progression of CC, we utilized the JAK-STAT pathway agonist Colivelin in rescue experiments. The influence of IKBIP on CC development was also validated in a xenograft tumor model.
Results Our study demonstrated that IKBIP is overexpressed in cervical cancer tissues, which could be a potential oncogene associated with cervical cancer. Kaplan-Meier survival analysis, ROC curve analysis, and nomogram construction revealed that IKBIP is a potential biomarker for poor prognosis in CC. Furthermore, the expression of IKBIP exhibited a strong correlation with immune infiltration, TMB and drug sensitivity in CC. In vitro experiments indicated that IKBIP functions as an oncogene, as silencing its expression significantly inhibited the proliferation, migration, and invasion capabilities of cervical cancer cells through CCK8, EdU, transwell assays respectively. Additionally, our findings suggest that IKBIP may promote cervical cancer progression by regulating the JAK-STAT signaling pathway. Rescue experiments demonstrated that the JAK-STAT pathway activator, Colivelin, could mitigate the inhibitory effects of IKBIP knockdown on cervical cancer cell behavior. We successfully constructed the cervical cancer xenograft mouse model and in vivo experiments demonstrated that the expression of IKBIP is closely correlated with the malignancy of cervical cancer and further corroborated the role of IKBIP in promoting cervical cancer progression.
Conclusion In summary, this study offers novel insights for CC by establishing IKBIP as a robust prognostic indicator. Our findings suggest that IKBIP not only correlates with adverse clinical outcomes but also influences tumor immunogenicity and response to treatment. Furthermore, IKBIP is significantly correlated with the progression of CC mediated by the JAK/STAT3 signaling pathway, and it may become an effective therapeutic target.

PO_093

瘤内微生物在宫颈癌淋巴结转移及淋巴脉管受累中预后意义的研究

丁家杉 张瑜*
中南大学湘雅医院

背景 宫颈癌病变处于富含微生物的环境中。然而，宿主-微生物群相互作用对宫颈癌发生淋巴脉管间隙浸润（LVSI）及淋巴结转移（LM）的影响及其机制仍不清楚。
材料与方法 因此，本研究在湘雅和 TCGA-CESC 两个队列中，采用 GATK-Pathseq 对宫颈癌组织瘤内微生物群组成及丰度进行分析。其次，在湘雅队列中开展微生物组测序进一步证明瘤内微生物群与宫颈癌 LVSI 及 LM 之间的关系。随后，利用广义线性模型构建肿内微生物群评分，并探究该评分对宫颈患者的预后分层意义。最后，通过 Bulk RNA-seq 和 single cell RNA-seq 联合分析，探讨宿主基因组与微生物环境相互作用关系，以进一步研究瘤内微生物群影响 LVSI 和 LM 发生的可能机制。
结果 首先，湘雅和 TCGA-CESC 两个独立宫颈癌队列的瘤内微生物群组成及丰度分析结果显示，不同 LVSI 和 LM 状态的宫颈癌组织具有独特的瘤内微生物特征，并鉴定出 5 个与淋巴结转移及淋巴脉管浸润相关的核心菌属。随后，基于核心菌属构建宫颈癌瘤内微生物群评分，生存分析显示高风险评分组总体生存（OS）较低风险评分组显著降低（HR=2.379, P=0.042）。进一步对预后最差的 LVSI(+)&LM(+）亚群进行预后分层分析，结果显示低微生物风险评分的 LVSI(+)&LM(+)患者的 OS 有所改善，而高微生物评分的 LVSI(+)&LM(+)患者 5 年生存率将接近 0。这种预后分层对高风险患者的临床管理具有一定的指导意义。
结论 总的来说，宫颈癌瘤内微生物可以影响宫颈癌患者的 LVSI 和 LM 状态。

PO_094

宫颈胃型腺癌及前驱病变的分子病理特征

陈婷婷 周先荣* 刘天齐 谢冰瑶
复旦大学附属妇产科医院

目的 宫颈胃型腺癌（GAS） 是一种 HPV 非相关子宫颈腺癌，在 HPV 非相关亚型中占比最高，随着 HPV 疫苗的广泛应用，宫颈癌胃型腺癌的发病率在未来可能增加。GAS 侵袭性强、放化疗耐药且预后较差，其形态学变异谱较广且与 HPV 感染无关，临床表现极不典型，故早期诊断困难，确诊时常为中晚期。GAS 的前驱病变包括叶状宫颈腺体增生、宫颈原位腺癌等，为 GAS 的早期筛查和诊断，需对 GAS 及其前驱病变的发病机制和分子病理特征进行充分了解，目前这方面的研究尚欠缺，本研究建立了新的检测方法并对此问题做了初步探究。
材料与方法 本研究中的样本来自本中心三年内回顾性 FFPE 标本（良性叶状增生（LEGH），不典型叶状增生（a-LEGH），原位胃型腺癌（gAIS），浸润性胃型腺癌（G-EAC），共 31 例）。病理复核后使用本实验室自主研发的宫颈癌基因 Panel（杂交捕获探针）进行二代测序（NGS），利用自建的生物信息分析方法识别常见基因变异和少见染色体结构变异（大片段重排等）等分子变异特征。
结果 10 例样本中检出 STK11 基因的致病性变异，在 A-LEGH 中检出 4/13，在 gAIS 及 G-EAC 中累计检出 5/15。9 例 STK11 变异均发生在<45 岁的受检者中，多为胚系变异，提示了黑斑-息肉综合征（PJS）的潜在致病因素。KRAS 突变在恶性程度更高的 gAIS 和 G-EAC 中略高于前驱病变(良性 LEGH 和 a-LEGH)，STK11 和 KRAS 共突变可能提示病变恶化方向的发展。TP53 未见于 LEGH 和 a-LEGH 中，仅在 gAIS（占比 33.3%， 2/6）和 G-EAC（占比 77.8%，7/9）中检出。结果表明，TP53 与 STK11/KRAS 表现出互斥趋势。TP53 突变与 P53 免疫表达之间的一致率为 87%（13/15，gAIS 和 G-EAC 组）。 G-EAC 队列中可见～22%（2/9）的 HER2 拷贝数扩增，2 例 HER2 扩增样本的免疫组化均为 HER2（3+），且 HER2 扩增与 TP53 呈共突变现象。在主要的免疫组化（IHC）分析中，p53 缺失、p16 缺失、Ki67 高表达是 G-EAC 的标志性特征。
结果 STK11、TP53、KRAS、CDKN2A、ERBB2 等是宫颈胃型腺癌及前驱病变的高频突变基因。在 PJS 综合征的遗传因素影响下，STK11 突变多见于<45 岁的患者，除常规突变外，大片段重排也是 STK11 重要的异常因素。KRAS 与 STK11 显示出共突变的趋势，而 TP53 与它们表现出互斥的趋势。TP53 致病变异是 gAIS 和 G-EAC 的特征性表现，测序有助于复杂病例的诊断。HER2 扩增是 G-EAC 的另一重要特征，且 HER2 扩增与 TP53 呈共突变现象。本研究初步绘制了宫颈胃型腺癌及其前驱病变的基因突变谱，并描绘了关键基因的突变特征，为进一步寻找该亚型治疗靶点和早诊早治靶标初步建立理论基础。

PO_095

术前 CA125 联合 CA199 对早期宫颈腺癌根治术后预后预测价值分析

修琳 安菊生 李宁 姚洪文 吴令英*
中国医学科学院肿瘤医院

目的 探讨术前 CA125、CA199 及联合检测与 IB1-IIA2 期宫颈腺癌患者临床病理的关系并评估其预后的应用价值。
方法 回顾性分析中国医学科学院肿瘤医院 2012 年 1 月 1 日至 2015 年 12 月 31 日收治的经手术病理诊断为 IB1-IIA2 期(FIGO 2009)的宫颈腺癌患者的临床病理资料。采用轶和检验和 Logistic 回归模型分析术前 CA125、CA199 及联合检测与患者临床病理参数的关系。利用生存分析确定影响总生存率和无病

生存率的相关危险因素及独立预后预测因子，利用受试者工作特征曲线，确定对 IB1-IIA2 期宫颈腺癌预后有预测价值的 CA125、CA199 的最佳分界点。曲线下面积评估结果的准确率。
结果 术前 CA125、CA199 升高与组织学分级、淋巴结有无转移、淋巴结阳性率、宫旁有无浸润、宫体是否受累以及淋巴脉管瘤栓显著相关。术前 CA125 与 CA199 联合检测浓度水平与组织学分级、肿瘤大小、淋巴结有无转移、淋巴结阳性率、宫旁有无浸润、宫体是否受累以及淋巴脉管瘤栓相关。Kaplan-Meier 分析明确显示，早期宫颈腺癌患者术前 CA125、CA199 升高，预后较差（P＜0.05）。联合术前血清 CA125 和 CA199 升高是早期宫颈腺癌预后的独立预测因子。利用受试者工作曲线预测总生存率和无病生存率的最佳 CA125 截断值均为 36.28U/mL, CA199 截断值均为 20.12U/mL。术前血 CA125 和 CA199 预测总生存率曲线下面积分别为 0.639（95%CI:0.544-0.726，P=0.0190）和 0.712（95%CI:0.620-0.793，P=0.0074），预测无病生存率曲线下面积分别为 0.621（95%CI:0.526-0.710，P=0.0134）和 0.732（0.641-0.810，P=0.0237）。
结论 术前血清 CA125 和 CA199 水平可能对早期宫颈腺癌的预后有重要影响，术前联合血清 CA125 和 CA199 水平可以作为早期宫颈腺癌的独立预后预测因子，联合升高组具有高复发和死亡风险。

PO_096

Cinobufotalin Inhibits Proliferation and Induces Apoptosis of 2 cervical squamous carcinoma cells

Lin Xiu Ning Li Lingying Wu*
National Cancer Center/National Clinical 4 Research Center for Cancer/Cancer Hospital, Chinese Academy of Medical Sciences 5 and Peking Union Medical College, Beijing, China

Objective This study aims to investigate the effect of Cinobufotalin on the proliferation, migration, invasion and apoptosis of cervical squamous carcinoma cells in vitro.
Methods Different concentrations of Cinobufotalin were divided into three groups including control group (0.1% DMSO), low-dose group (0.5 μmol) and high-dose group (1.0 μmol). CCK-8 assay was used to determine the effect of different concentrations of Cinobufotalin on the viability of SiHa and C-33A cells. Cell clony formation experiment was used to observe the cell clony formation ability. Flow cytometry was utilized to examine the changes of cell cycle, and AnnexinV-FITC/PI double staining was used to observe the apoptosis. Transwell assay was used for observing the migration and invasion abilites.
Results Compared with the control group, Cinobufotalin significantly inhibited cell proliferation (P<0.001) and clonogenesis (P<0.001), and induced G2/M cycle arrest. The migration and invasion abilites decreased with the increase of cinobufotalin concentration (P<0.001). AnnexinV-FITC/PI double staining showed that Cinobufotalin induced dose-dependent apoptosis of cervical squamous carcinoma cells (P<0.001).
Conclusion Cinobufotalin can effectively inhibit the proliferation, migration and invasion abilites of SiHa and C-33A cells, induce apoptosis and cell cycle arrest in G2/M phase.

PO_097

YTHDF3-mediated regulation of PD-L1: implications for immune evasion in cervical cancer

liqing miao[1] Jingwei Li[2] Luyi Ruan[1] Jianan Zhang[1] Xueqiong Zhu(*corresponding author)[1*]
1. Department of obstetrics and gynecology, the Second Affiliated Hospital of Wenzhou Medical University, Wenzhou, Zhejiang, China
2.Department of Obstetrics and Gynecology, Taizhou Women and Children's Hospital of Wenzhou Medical University, Zhejiang, China

Objective This study aimed to investigate the expression of YTHDF3 in cervical cancer and its effects on the biological behavior of cervical cancer cells. Additionally, the study explored how YTHDF3 promotes immune evasion by regulating PD-L1 expression and clarified the underlying molecular mechanisms.
Methods Immunohistochemistry and Western blotting were used to detect YTHDF3 expression in cervical cancer tissues and cell lines. The effects of YTHDF3 on cell proliferation and apoptosis were assessed using CCK-8 assays and flow cytometry. The impact of YTHDF3 knockdown on immune evasion and T cell-mediated cytotoxicity was evaluated through immunohistochemistry, T cell killing assays, and murine tumor xenograft models. Dot blot analysis was performed to determine the relationship between YTHDF3 and global m6A RNA methylation levels. RNA immunoprecipitation and RNA pull-down assays were conducted to confirm a direct interaction between YTHDF3 protein and PD-L1 mRNA.
Results YTHDF3 expression was upregulated in cervical cancer tissues and cell lines. However, modulation of YTHDF3 expression had no significant effect on cervical cancer cell viability or apoptosis. Nonetheless, YTHDF3 expression was positively correlated with PD-L1 expression. YTHDF3 knockdown significantly enhanced T cell-mediated killing of cervical cancer cells. In immunocompetent mice, tumors formed by cells with YTHDF3 knockdown showed markedly reduced growth, accompanied by increased levels of immune-related factors. Moreover, YTHDF3 was positively associated with global m6A methylation levels and was found to directly bind to PD-L1 mRNA, thereby regulating its expression.
Conclusion YTHDF3 promotes immune evasion in cervical cancer by directly binding to PD-L1 mRNA and enhancing its expression. Knockdown of YTHDF3 enhances T cell cytotoxicity and suppresses tumor growth, suggesting that YTHDF3 may serve as a potential target for immunotherapy in cervical cancer.

PO_098

A CRISPR-Cas12a-Based Ultrasensitive and APE1-Free Biosensor for Uracil-DNA Glycosylase Activity Detection and Subcellular Imaging in Cervical Cancer

Kejun Dong Hongbo Wang*
Union Hospital, Tongji Medical College, Huazhong University of Science and Technology

Background Genomic instability is closely associated with tumorigenesis, progression, and therapeutic response evaluation. Uracil-DNA Glycosylase (UDG), a key enzyme in the base excision repair pathway, exhibits dysregulated expression linked to the development of diseases including ovarian and cervical cancer. Consequently, UDG has emerged as a significant biomarker for monitoring and prognostic assessment, highlighting the critical need for simple, specific, rapid, and sensitive UDG detection methods. However, the low abundance of UDG in biological environments necessitates pre-amplification steps in most existing assays, significantly increasing complexity and

operational difficulty. More critically, current methods rely on the auxiliary enzyme APE1, whose levels are difficult to maintain spatiotemporally consistent with UDG, potentially leading to distorted results, particularly in intracellular imaging applications. The development of novel, highly specific, ultrasensitive, and auxiliary enzyme-free UDG sensing systems is therefore urgently required.
Materials and Methods Leveraging DNA thermodynamic reaction calculations, we constructed a CRISPR-Cas12a-based biosensor utilizing a tightly balanced dsDNA system. This design achieves low background, high sensitivity, and APE1-independent UDG sensing. A PAM-free dsDNA substrate containing a strategically placed bubble serves as the UDG target. In the absence of UDG, the dsDNA remains intact and cannot activate CRISPR-Cas12a to cleave a fluorescent reporter probe. Upon UDG presence, the enzyme excises a uracil base at a predefined dU site adjacent to the bubble, generating an apurinic/apyrimidinic (AP) site. This AP site, combined with the pre-existing bubble, creates an enlarged destabilized region. This structural change disrupts the tightly balanced state, enabling sgRNA invasion and subsequent activation of the CRISPR-Cas12a system, producing a detectable signal. Furthermore, we analyzed live-cell UDG activity imaging using a nano-vehicle co-delivering the tightly balanced dsDNA substrate and the fluorescent probe, alongside Cas12a protein and sgRNA expressed endogenously from plasmids. Following intracellular assembly of Cas12a and sgRNA, the complex translocates to the nucleus guided by a nuclear localization signal. There, the dsDNA substrate responds to nuclear UDG activity, forming an AND-gate logic circuit that releases the fluorescent signal, enabling precise UDG detection and subcellular imaging in cervical cancer cells.
Results Compared to conventional dsDNA substrates, the tightly balanced substrate demonstrated significantly enhanced sensitivity to UDG, achieving a 72.64-fold increase in discrimination. Systematic orthogonal optimization of parameters, including dU position, number, and RNP concentration, yielded a detection limit of 5×10^{-7} U/mL within 150 min, enabling detection in real cervical cancer cell samples. Following intracellular assembly, the Cas12a-sgRNA complex successfully responded to nuclear UDG activity, generating fluorescence and achieving UDG imaging within specific subcellular structures.
Conclusion We developed a novel, simple, specific, ultrasensitive, and auxiliary enzyme-free UDG sensing system by integrating a low-background, tightly balanced dsDNA substrate with the exceptional signal amplification capability of CRISPR-Cas12a. UDG processing alters the substrate's structure and conformation, collapsing its tightly balanced state and lowering the free energy barrier for the crRNA-dsDNA strand displacement reaction. This allows crRNA to recognize and invade the target DNA sequence, activating the CRISPR-Cas12a system without requiring APE1 assistance. This system achieves highly sensitive UDG detection, with the optimized limit of detection (LOD) reduced by 1-3 orders of magnitude compared to existing methods. Furthermore, we demonstrated its capability for UDG sensing in single cells and targeted intracellular imaging, showcasing robust operational efficacy and significantly reducing reliance on APE1 in UDG detection systems. This advancement lowers the construction cost of fluorescent detection platforms and provides a novel detection system for a key biomarker in the DNA damage repair pathway.

PO_099

Tumor budding and residual tumor volume in post-neoadjuvant resection specimens are associated with poor prognosis in cervical cancer

Xiaoxiao Hu[1,2] Peiye Shen[1,2] Ruoyu Shi[3] Xia Yin[1,2*]
1.Department of Obstetrics and Gynecology, Renji Hospital, School of Medicine, Shanghai Jiao Tong University
2.Shanghai Key Laboratory of Genocologic Oncology
3.Department of Pathology and Laboratory Medicine, Kandang Kerbau Women's and Children's Hospital, Singapore

Objectives This study investigated prognostic significance of tumor budding (TB) and residual viable tumor (RVT) burden in locally advanced cervical cancer (LACC) treated with neoadjuvant chemotherapy (NACT) followed by radical surgery. TB, representing single tumor cells or small clusters

at the invasive front, has emerged as a histological marker of tumor aggressiveness and poor prognosis in various malignancies, yet its role in the post-NACT setting of cervical cancer is unclear. Similarly, RVT, reflecting the percentage of remaining viable tumor cells within the treated tumor bed, serves as an indicator of therapeutic response in several cancers but lacks standardized evaluation criteria for cervical cancer. Considering the clinical need to optimize risk stratification in patients undergoing NACT, this study sought to: (1) evaluate the impact of TB and RVT on overall survival (OS) and recurrence-free survival (RFS); (2) identify optimal cutoff values for these parameters to classify patients into prognostic groups.

Materials and Methods This retrospective cohort study analyzed 101 LACC patients (FIGO stages IB3–IIIC2; 98% squamous cell carcinoma) treated with NACT and radical hysterectomy. TB was quantified according to the International Tumor Budding Consensus Conference (ITBCC) criteria (3-tier: 0–4, 5–9, > 9 buds/field; 2-tier: ≤ 4 vs. > 4 buds/field). RVT burden was calculated as (RVT surface area/total tumor bed area) × 100 and scored in 5% increments. Interobserver agreement for TB and RVT scoring was calculated using correlation coefficients. Survival curves were generated by Kaplan–Meier analysis and compared using the log-rank test. The prognostic impact of TB and RVT was assessed by univariate and multivariate Cox regression models, adjusting for clinicopathologic variables. Associations between TB, RVT, and other parameters were analyzed by χ^2 or Fisher's exact test. $P < 0.05$ was considered statistically significant.

Results The study cohort comprised 101 LACC patients (median age 54 years; 98% squamous cell carcinoma) treated with NACT and radical surgery. Tumor budding (TB) and residual viable tumor (RVT) burden assessment demonstrated strong interobserver reproducibility (TB: r = 0.71, $P < 0.001$; RVT: r = 0.90, $P < 0.001$). TB with modified ITBCC criteria (> 4 buds/field) showed superior prognostic discrimination versus standard 3-tier grading (OS: $P < 0.001$; RFS: $P = 0.037$). TB-positive status also correlated with higher tumor grade ($P = 0.043$) and parity ($P = 0.005$). ROC analysis identified 2.5 buds/field as the optimal TB cutoff (sensitivity 81.8%, specificity 73.3%) in this cohort. Residual viable tumor (RVT) analysis revealed RVT ≤ 10% (TRS 1) as a significant predictor of prolonged RFS (50.9 vs. 36.3 months, $P = 0.014$), though its association with OS was non-significant ($P = 0.064$). In multivariate analysis, RVT > 10% independently predicted worse RFS (HR = 3.66, $P = 0.048$), while TB positivity was significantly associated with inferior OS (HR = 8.57, $P < 0.001$) and RFS (HR = 2.96, $P = 0.045$) in univariate models.

Conclusions TB and RVT are potent prognostic markers in post-NACT LACC. A simplified 2-tier TB grading system (> 4 buds) and RVT > 10% identify high-risk patients who may benefit from intensified adjuvant therapy. Integration of these markers into standard pathological reporting should be considered, pending validation in prospective cohorts.

PO_100

The Prognostic Value of Textbook Outcomes in Patients With Cervical Cancer Undergoing Radical Surgery

Mu Xu Yanling Zhong Zhenhong Wang Liangzhi Cai*
Fujian Maternity and Child Health Hospital, Affiliated Hospital of Fujian Medical University

Background To investigate the prognostic value of Textbook Outcome (TO) in patients with cervical cancer (CC) who underwent radical surgery.

Methods Clinical data of 509 patients with CC who underwent radical surgery were retrospectively collected and analyzed. The patients were divided into TO and non-TO groups. Multivariate logistic regression was used to analyze the independent factors influencing TO, whereas the Cox regression model was used to identify the independent prognostic factors for overall survival (OS).

Results A total of 509 patients were included, of whom 48.5% (247/509) achieved TO. Univariate analysis revealed significant differences between the TO and non-TO groups in terms of FIGO stage,

tumor size, depth of invasion, lymph node metastasis (LNM), squamous cell carcinoma (SCC) grade, operative time, and intraoperative blood loss. Multivariate analysis indicated that the FIGO stage and intraoperative blood loss were independent influencing factors for TO. Multivariate Cox regression analysis revealed that lymphovascular space invasion (LVSI), LNM, and TO status were independent prognostic factors for OS. The 5-year OS rate was significantly higher in the TO group than in the non-TO group (93.9% vs. 85.8%, P<0.01). After 1:1 propensity score matching, the 5-year OS rate remained statistically significant between the TO and non-TO groups (94.2% vs. 86.5%, P<0.01).
Conclusions TO demonstrates a significant prognostic value in patients with CC who underwent radical surgery. Clinically, optimization of surgical strategies to achieve TO should be prioritized to improve patient outcomes.

PO_101

宫颈病变与机体免疫逃逸相关研究进展

张芍 [1,2*]
1.昆明医科大学第三附属医院 云南省肿瘤医院 北京大学肿瘤医院云南医院妇科
2. 昆明医科大学第三附属医院 云南省肿瘤医院 北京大学肿瘤医院云南医院妇科

目的 本文欲阐明宫颈病变与机体免疫逃逸相关机制，对宫颈癌的治疗和预防具有不可估量的作用。
材料与方法 本文通过搜索中国知网+Pubmed 网站文章，通过分析自身免疫和 HPV 免疫逃逸机制俩方面重点阐述机体免疫与宫颈病变的相关研究进展。自身免疫：通过体液免疫+细胞免疫+细胞因子详细阐述宫颈癌患者机体免疫功能不足，阻碍了癌细胞被识别和杀伤，致肿瘤发生、发展。机体的低免疫功能与肿瘤发生发展相互作用，相互促进。细胞免疫：通过详细论述宫颈癌患者自身免疫细胞，尤其是 T 淋巴细胞亚群、NK 细胞、树突细胞和巨噬细胞的变化及发挥的作用。细胞因子：通过 IL-2、IL-6、IL-10 和 IL-17 在宫颈癌患者中的变化及作用。HPV 病毒可能的免疫逃避机制：保持病毒低拷贝的复制量、HPV 使用哺乳动物密码子取代病毒密码子、T 细胞亚群的激活与迁移、宿主自身的免疫遗传特性、致使抑癌基因失活、免疫选择、PD1/PD-LI 信号通路方面论述 HPV 病毒可能的免疫逃避机制。
结果 细胞免疫：肿瘤患者体内 Th1/Th2 细胞平衡呈 Th2 型偏移状态，促进恶性肿瘤增长，帮助肿瘤逃逸。也可通过观察 Th1/Th2 比值来评估机体抗肿瘤的免疫动态。CD8+T 细胞亚群的作用是特异性攻击靶细胞和抑制 T 淋巴细胞的增殖。分化为细胞毒性 T 细胞和抑制性 T 细胞。在宫颈肿瘤中，树突状细胞的数量较正常患者减少，猜测人乳头状瘤病毒（HPV）可能通过减少树突细胞的数量、降低机体局部组织的免疫监视，形成免疫逃逸，从而促进肿瘤的发展。近年来肿瘤相关巨噬细胞（TAMs）的概念认为巨噬细胞可能促进肿瘤内血管生成及生长因子产生或调控细胞因子（如 IL-10）的分泌而诱导淋巴细胞死亡，从而促进肿瘤的发生发展。机体调节免疫功能的是细胞因子，感染 HPV 病毒后，机体通过分泌白介素 2、IL-10 等细胞因子发挥机体调节功能。这些细胞因子通过血液作用于全身或生殖道粘膜，参与阴道免疫微环境的调节，影响全身免疫功能及局部免疫情况，故也可以通过监测细胞因子的水平评估机体的免疫状态。根据现有研究表明,HPV 的免疫逃避机制主要通过自身躲藏、特异性地干扰固有免疫与适应性免疫、整合入人体基因组等干扰方式,逃避免疫监视，达到持续作用、最终发生、发展为恶性肿瘤。
结果 宫颈癌的生物学特性非常复杂，HPV 病毒虽然是一个简单的病毒，可它却通过自身隐藏、特异性地干扰固有免疫与适应性免疫、整合入人体基因组等方式,达到持续感染、致癌的结果。

PO_102

The Association of Post-Conization Pregnancy with Human Papillomavirus Infection and Cervical Lesion Recurrence: Evidence from a Nationwide Cohort in Sweden and Hospital-Based Clinical Data in China

Hang lin[1] Jimiao Huang[1] Zihan Deng[1] Xiangqin Zheng[1] Jianguang Ji[2,3] Huan Yi[1*]
1.College of Clinical Medicine for Obstetrics & Gynecology and Pediatrics, Fujian Medical University, Fujian Maternity and Child Health Hospital
2.Lund University3.University of Macau

Objective Women who have undergone cervical conization may still experience subsequent pregnancies and delivery. However, it remains unknown whether pregnancy, with its associated immune tolerance, might decrease the ability to clear human papillomavirus (HPV) and increase the risk of cervical lesion recurrence. This study aims to explore the risk of HPV infection and cervical lesion recurrence after post-conization pregnancy by combining evidence from nationwide cohorts in Sweden and clinical data in China.
Methods A total of 2221 women who underwent conization at Fujian Maternity and Child Health Hospital between October 2013 and October 2021 were divided into pregnancy and non-pregnancy group and to evaluate the risk of HPV infection. Additionally, women diagnosed with cervical intraepithelial neoplasia in Sweden between January 1997 and December 2017 and treated with conization were identified through the Swedish National Patient Register and followed for cervical lesion recurrence. Time-dependent Cox regression was used to analyze the association of post-conization pregnancies with HPV infection and cervical lesion recurrence.
Results In Chinese women with post-conization pregnancies, the risk of HPV infection was significantly higher (HR: 2.04, 95% CI: 1.33-3.14) compared to post-conization women without pregnancy. Among the 60,895 women who underwent conization in Sweden, 15,200 (25%) had post-conization pregnancies and showed a higher incidence of cervical lesion recurrence (HR: 1.32, 95% CI: 1.13-1.53) compared to post-conization women without pregnancies. The increased risk of recurrence was observed only in women who had a pregnancy within three years after conization (adjusted HR=1.39, 95% CI,1.19–1.63). For the sensitivity analyses, we found that in women with abortion after conization, there was no statistically significant effect on cervical lesion recurrence, with an adjusted HR of 1.19 (95% CI, 0.84-1.69).
Conclusions Our study demonstrates that post-conization pregnancies are associated with a higher risk of HPV infection and cervical lesion recurrence compared to women without pregnancies. The risk was particularly significant in women who had a pregnancy within three years of conization, suggesting that those who become pregnant within three years after conization will need close clinical monitoring

PO_103

Exploring the Effectiveness of Cervical Cancer Awareness Campaign in China: Evidence from Hospital-Based Screening Data

Hang Lin[1] Shihuang Liu[1] Jimiao Huang[1] Yusha Chen[1] Xiangqin Zheng[1] Jianguang Ji[2] Huan Yi[1*]
1.College of Clinical Medicine for Obstetrics & Gynecology and Pediatrics, Fujian Medical University
2.University of Macau

Background Although highly preventable, cervical cancer remains prevalent in China largely due to insufficient awareness and low rates of cervical screening. Our objective was to assess whether the annual Cervical Cancer Awareness Campaign (launched each March) can increase the participation rate of cytology screening in China.
Methods This hospital-based study utilized screening data from Fujian Maternity and Child Health Hospital, the largest facility covering over half of Fuzhou city's cervical cancer screenings (comprising more than 7 million citizens) from January 2012 to December 2023. The frequencies of cervical cytology screening in all gynecological outpatient clinics and colposcopy examination in cervical outpatient clinics were recorded monthly. By employing Fused Lasso regularization in Poisson regression models, we identified the years and months with significant changes in the frequency of cervical cytology screenings and colposcopies. The incidence rate ratios (IRRs) were estimated for the change points at specific years and months using the preceding change point as the reference, while cumulative IRRs for the months were calculated using January as the reference, respectively.
Results A total of 3,302,244 gynecological outpatient visits were recorded in the final analysis. Approximately 387,017 (11.7%) outpatient visits involved cervical cytology screening and 41,319 (1.3%) women underwent colposcopy. When comparing the monthly frequencies of cervical cytology screening, March exhibited a significant increase with an incidence rate ratio (IRR) of 1.08 (95% CI: 1.07-1.09). Additionally, higher frequencies of colposcopy were observed in both March (IRR: 1.07, 95% CI: 1.02-1.12) and April (IRR: 1.17, 95% CI: 1.13-1.21), respectively.
Conclusion The Cervical Cancer Awareness Campaign has notably enhanced the cervical screening rate in Fujian Maternity and Child Health Hospital. Further studies aimed at assessing the present cervical cancer screening practices nationwide can broaden our understanding of the interventions and impacts of the Cervical Cancer Awareness Campaign across diverse regions and populations.

PO_104

阴道加德纳菌的代谢产物 N-乙酰谷氨酰胺通过 TNFRSF9/NF-κB 通路增强宫颈腺癌干性

黄榆璇 王钥 孙蓬明*
福建省妇幼保健院

研究目的 单纯的 HPV 感染大多被人体自发清除，并不足以引起宫颈癌，但当 HPV 合并阴道微生态紊乱时，宫颈癌的发生风险可能会显著增加。作为常见的高风险 HPV 型别，HPV18 诱导的宫颈腺癌具有发病隐匿、侵袭性高和预后差等特点，已成为全球危害育龄期女性生命健康的主要疾病之一。细菌性阴道病是一种以阴道加德纳菌（Gardnerella vaginalis, GV）过度生长为特征的下生殖道混合性感染，被认为是导致性传播疾病、HPV 相关恶性肿瘤发生发展的风险因素。目前，宿主菌群通过代谢对疾病进展的影响已成为研究热点，然而过度增殖的 GV 是如何通过代谢影响 HPV18 相关宫颈病变的潜在分子机制目前尚未阐明。

材料与方法 16SRNA 高通量测序检测 HPV18 阳性女性人群阴道分泌物菌群丰度，评价 GV 与 HPV18 阳性宫颈病变的相关性；建立体外 GV 与 HPV18 阳性宫颈癌细胞的共培养体系，筛选最适共培养菌体浓度和培养时长；采用液相色谱联用（LC-MS）代谢组学筛选出共培养体系中细胞内外共同上调差异代谢物，结合临床样本筛选出关键代谢产物 N-乙酰谷氨酰胺；采用细胞免疫荧光、肿瘤球形成等实验观察 GV 与 N-乙酰谷氨酰胺对 HPV18 阳性细胞致癌基因 E6、E7 表达和肿瘤干性的影响；代谢组学联合转录组学筛选出 TNFRSF9/NF-κBp52 通路，Western blot、细胞免疫荧光验证 N-乙酰谷氨酰胺处理后细胞的通路基因表达情况；慢病毒转染细胞，构建稳定敲低的 TNFRSF9 细胞系，采用 Western blot、肿瘤球形成实验验证细胞通路基因以及致癌基因、干性相关基因的表达情况；在裸鼠皮下成瘤模型中，观察 GV、N-乙酰谷氨酰胺对瘤体大小的影响。

结果 经 GV 或 N -乙酰谷氨酰胺处理后，HPV18 阳性宫颈癌细胞的致癌基因 E6 表达增加，肿瘤干性能力增强（P＜0.05）；通过联合代谢组学和转录组学分析，结果显示差异代谢物 N-乙酰谷氨酰胺水平与差异基因 TNFRSF9 的表达呈强相关（P＜0.05）；TNFRSF9 的激活可通过非经典通路 NF-κBp52 促进信号转导，从而增强细胞致癌基因 E6 和干性相关标志物的表达（P＜0.05），从而驱动 HPV18 阳性宫颈癌细胞的恶性程度增强。

结果 N -乙酰谷氨酰胺通过激活 TNFRSF9/NF-κBp52 通路增强宫颈腺癌干性，为 GV 如何通过其代谢产物驱动 HPV18 阳性宫颈癌的恶性进展提供了新的见解。

PO_105

Multidimensional evidence synthesis based on global inequalities in cervical cancer screening

ruijun xin[1,2] Bo Zhu[1*] Jiaqi Guo[1] Meixi Gong[1,2] Jiahui Tian[1,2] Duo Xu[1,2] Mouqian Wu[1,2]
1.Hospital of China Medical University/Liaoning Cancer Hospital & Institute
2.Dalian Medical University

Objective The heavy and increasing global burden of cervical cancer, alongside the persistent global inequalities in access to and utilization of screening services, highlights the necessity of strengthening their implementation. In 2020, WHO launched the global initiative to eliminate cervical cancer, setting the 90-70-90 targets (vaccination, screening, treatment). Screening coverage is a core indicator for screening, but achieving it is influenced by policies and implementation. Therefore, we compiled the cervical cancer screening guidelines from different countries/regions and evaluated their implementation status and coverage situation.

Materials and Methods In this study, we conducted a comprehensive search of the PubMed, Embase and Web of Science databases (from their establishment to February 1, 2025), as well as websites (the EU Public Health Platform; National Breast and Cervical Cancer Early Detection Program [NBCCEDP] at the Centers for Disease Control and Prevention [CDC] and the National Health Interview Survey [NHIS]) and the World Health Organization (WHO) database. The included studies included those that provided detailed information on the official cervical cancer screening recommendations of the respective countries/regions. Additionally, we comprehensively synthesized evidence by compiling national guidelines, evaluating implementation status (invitation, participation, screen coverage rate), service accessibility, and program quality indicators. The screening coverage rate was stratified based on factors such as human development index (HDI), income level, geographical location,WHO region, disease risk, and age. This systematic review was prospectively registered in the National Institute for Health Research International Prospective Register of Systematic Reviews (PROSPERO, No. CRD420251087263, July 4, 2025).

Results Our study included data from 77 countries/regions. Among these countries/regions, only 62.3% (48/72) documented publication years for definitive screening guidelines in the database. 36.4% of the countries/regions recommend starting screening age of 24 years or younger; 75% of the countries/regions recommend ending screening at the age of 60 or above. Countries with high and

very high HDI mainly used cytology and human papillomavirus (HPV) tests for screening, while low HDI countries relied almost entirely on visual inspection with acetic acid or iodine staining (VIA/VILI). In terms of screening coverage, the global average is 43% (95% CI: 35% - 51%), which is far below the 70% target set by the WHO. Screening coverage (reported at 54.5%) was associated with HDI/income (low HDI: 14% vs very high HDI: 54%; high-income: 54% vs low income and middle-income countries [LMICs]:34%), with high-risk regions (29%) significantly lower than other areas (47%). Age-stratified analysis revealed minimal coverage for ages 20-29 and 70-79, while participation rates were highest among those aged 30-39. Our results also showed substantial heterogeneity in screening quality indicators (positive rate, further assessment rate, detection rate, and positive predictive value [PPV]), compounded by severe deficiencies in data reporting.

Conclusions This study highlights global inequities in cervical cancer screening coverage, accessibility, and quality monitoring, particularly low coverage among younger and older women and the large gap between LMICs/high-risk areas and WHO's 70% target. These results highlight the necessity of accelerating national screening strategies, improving accessibility throughout the screening - diagnosis - treatment process, establishing monitoring systems, and strengthening global cooperation and financial investment. This study can help optimize resources and advance progress toward eliminating cervical cancer.

PO_106

Intra-tumoral microbiota induce NETosis and promote the progression and lymphatic metastasis in cervical cancer

Xiangguang Wu[1] Yuhua Pan[1] Liangsheng Fan[1] Wei Wang[1,2*]
1.the First Affiliated Hospital of Guangzhou Medical University
2.Shanghai First Maternity and Infant Hospital

Aim This study aims to investigate the role of neutrophil extracellular traps (NETs) induced by intra-tumoral microbiota in the progression and lymphatic metastasis of cervical cancer.

Materials and Methods Intra-tumoral microbiota were detected by in situ hybridization (EUB338 mix), and the component of bacterial, such as Lipopolysaccharide and Lipoteichoic acid, were detected by immunohistochemistry. The content and distribution of neutrophils and NETs were detected by multicolor immunofluorescence. The capture of cervical cancer cells by NETs were confirmed by co-localization analysis of multi-color immunofluorescence via Fiji software. The effects of NETs on the abilities of proliferation, migration and invasion of cervical cancer cells were detected by in vitro experiments, and the effects of NETs on gene expression were analyzed by transcriptome sequencing. The effects of NETs on tumor growth and lymphatic metastasis were verified by subcutaneous tumor model in NCG mice and claw pad model in C57BL/6 mice respectively.

Results Bacterial probe EUB338 and bacterial toxin LPS were both positive in the lesions of cervical cancer with lymphatic metastasis, and also positively correlated with the level of the NETs. MPO and citH3 were potential predictive makers for lymphatic metastasis. We demonstrated that LPS derived from intra-tumoral microbiota recruited neutrophils and induced NETosis. NETs trap cervical cancer cells and enhance their abilities of proliferation, migration and invasion and mainly up-regulate the genes enriched in the pathways of oxidative phosphorylation and translation related pathways. NETs promote the growth of subcutaneous tumor model (SiHa and Hela cell lines) in NCG mice. NETs induced by intra-tumoral LPS promote the progression and lymphatic metastasis of claw pad tumor model in C57BL/6 mice, however, while NETs were inhibited by GSK484 or eliminated by DNaseI, the growth and lymphatic metastasis of tumor were inhibited.

Conclusions Neutrophils were recruited and induced to form NETs by intra-tumoral microbiota derived LPS. NETs promoted the abilities of proliferation, migration and invasion of cancer cells and thus promoted the growth and lymphatic metastasis of cervical cancer. NETs might be of great potential target for prediction and treatment in cervical cancer.

PO_107

Multi-Dimensional Treatment of Small Cell Carcinoma of The Cervix Based On Organoids

Bai Hu Ding Ma Peng Wu*
Huazhong University of Science and Technology

Background Cervical small cell carcinoma (SCCC) is a rare subtype of cervical cancer, accounting for approximately 1%–2% of all invasive cervical cancers. It is highly aggressive, with a propensity for lymph node metastasis and lymphovascular space invasion, even in early-stage cases, resulting in a 5-year survival rate of less than 14%. The current standardized chemotherapy regimen for SCCC, modeled after that for small cell lung cancer (SCLC), consists of cisplatin and etoposide. However, this regimen does not significantly improve patient outcomes. Given the distinct pathogenesis and tissue origin of SCCC compared to other small cell carcinomas, it is crucial to identify specific therapeutic strategies for SCCC and explore the molecular events underlying its neuroendocrine features.
Background: Cervical small cell carcinoma (SCCC) is a rare subtype of cervical cancer, accounting for approximately 1%–2% of all invasive cervical cancers. It is highly aggressive, with a propensity for lymph node metastasis and lymphovascular space invasion, even in early-stage cases, resulting in a 5-year survival rate of less than 14%. The current standardized chemotherapy regimen for SCCC, modeled after that for small cell lung cancer (SCLC), consists of cisplatin and etoposide. However, this regimen does not significantly improve patient outcomes. Given the distinct pathogenesis and tissue origin of SCCC compared to other small cell carcinomas, it is crucial to identify specific therapeutic strategies for SCCC and explore the molecular events underlying its neuroendocrine features.
Materials and Methods We established a comprehensive biobank of patient-derived organoids (PDOs) from 70 cervical cancer patients, including 25 cases of squamous cell carcinoma, 20 cases of adenocarcinoma, 22 cases of small cell carcinoma, and 3 cases of clear cell carcinoma. The organoid models for various pathological types, including cervical squamous cell carcinoma, adenocarcinoma, small cell carcinoma, clear cell carcinoma, and small cell lung cancer, were created for immunofluorescence imaging, with the organoids stably expressing biomarkers. The organoid biobank was validated through histology, pathology, transcriptomics, and whole-genome sequencing to ensure high genetic fidelity and retention of patient-specific features. We screened 1,783 FDA-approved drugs using these organoids to identify potential therapeutic targets for SCCC.
Results 1. Organoid Biobank Characterization: The organoid biobank included a total of 70 cervical cancer patients, with 22 cases of SCCC. The organoids of various pathological types maintained consistency with the transcriptomic and genomic profiles of their source tissues, demonstrating high genetic fidelity. This comprehensive validation ensured that the organoids accurately represented the original tumors, providing a reliable platform for drug screening and therapeutic exploration. 2. Drug Screening and Sensitivity Analysis: Screening of 1,783 FDA drugs revealed that SCCC organoids were highly sensitive to drugs targeting multiple pathways, including DNA damage, neuronal signaling, and endocrinology. This finding suggested that SCCC might have unique molecular vulnerabilities that could be exploited for therapeutic benefit. 3. Neuroactive Drug Sensitivity: Notably, SCCC organoids exhibited significant sensitivity to neuroactive drugs. This sensitivity was further validated through multiple rounds of patient-derived xenograft (PDX) animal experiments, confirming the efficacy of these drugs. The results indicated that neuroactive drugs could be a novel therapeutic approach for SCCC. 4. Combination Therapy Validation: We validated the efficacy of neuroactive drugs in combination with chemotherapy in three SCCC PDX models. The results showed that the combination therapy significantly enhanced the therapeutic effect compared to monotherapy. This finding suggests that integrating neuroactive drugs with standard chemotherapy could provide a more effective treatment strategy for SCCC patients. 5. Cross-Application in SCLC: Additionally, we tested the efficacy of neuroactive drugs in organoids derived from four small cell lung cancer (SCLC) patients. The results indicated that these drugs were also effective in SCLC organoids, suggesting a potential cross-

application in treating SCLC. This finding highlights the broader therapeutic potential of neuroactive drugs in neuroendocrine-related cancers.
Conclusions This study established the largest multicellular, multipathological type organoid biobank for cervical small cell carcinoma to date. The organoids were characterized morphologically, histologically, and at the transcriptomic and genomic levels, demonstrating that they highly retained patient-specific features. By screening a comprehensive library of FDA-approved drugs, we identified that SCCC is highly sensitive to neuroactive drugs. This sensitivity is likely due to the unique neuroendocrine features of SCCC. The combination of neuroactive drugs with chemotherapy showed a significant therapeutic effect in PDX models, suggesting a promising new treatment strategy. Future research will focus on further exploring this phenomenon and validating the therapeutic potential of targeting neuroendocrinization to improve the prognosis of SCCC patients. Additionally, the efficacy of neuroactive drugs in SCLC organoids indicates a potential application in treating other neuroendocrine-related cancers. This study provides a foundation for developing personalized therapies for SCCC and potentially other aggressive neuroendocrine cancers.

PO_108

First-line PD-(L)1 inhibitors plus bevacizumab and chemotherapy for persistent, recurrent, or metastatic cervical cancer: a network meta-analysis-based economic evaluation

Yin Shi Di Xiao Yu Zhang*
Xiangya Hospital Central South University

Objective PD-(L)1 inhibitors combined with bevacizumab and chemotherapy (BC) significantly improve survival in patients with persistent, recurrent, or metastatic cervical cancer (R/M-CC) receiving first-line treatment. However, the high cost of immunotherapies imposes a substantial economic burden, underscoring the need to evaluate the cost-effectiveness of anti-PD-(L)1 combination regimens. This study aimed to evaluate the cost-effectiveness of atezolizumab plus bevacizumab and chemotherapy (ABC) versus pembrolizumab plus bevacizumab and chemotherapy (PBC) for first-line treatment of R/M-CC.
Methods A network meta-analysis was conducted following PRISMA extension guidelines, systematically searching Cochrane Central Register, PubMed, and Embase databases through December 9, 2024. Two phase III randomized controlled trials were identified: KEYNOTE-826 (PBC vs. bevacizumab + chemotherapy, n=389) and BEATcc (ABC vs. bevacizumab + chemotherapy, n=410), encompassing 402 patients receiving immunotherapy-based treatments. Statistical analysis utilized the gemtc package in R version 4.2.0 with fixed-effects modeling to generate hazard ratios (HRs) for overall survival (OS) and progression-free survival (PFS).
For cost-effectiveness analysis, a three-state Markov model was constructed using TreeAge Pro 2022, incorporating progression-free survival, progressive disease, and death states. The model employed a 21-day cycle length over a 30-year time horizon, with a 3% annual discount rate and $150,000 willingness-to-pay threshold. Clinical efficacy data were extracted from Kaplan-Meier curves using GetData Graph Digitizer software, with parametric survival modeling (lognormal for PFS, log-logistic for OS) applied for long-term projections. Direct medical costs included drug acquisition, administration, follow-up care, adverse event management, and terminal care, adjusted to 2024 US dollars. Utility values were derived from KEYNOTE-826 health-related quality-of-life data. Comprehensive sensitivity analyses included one-way, two-way, and probabilistic sensitivity analyses with 10,000 Monte Carlo simulations.
Results Network meta-analysis demonstrated that PBC ranked first for both OS extension (69.60% probability) and PFS extension (67.08% probability). Compared to ABC, PBC showed favorable but non-significant hazard ratios of 0.91 (95% CI: 0.62-1.32) for OS and 0.93 (95% CI: 0.66-1.30) for PFS, indicating comparable efficacy between regimens.

Cost-effectiveness analysis revealed significant economic advantages for PBC. The model projected 2.78 quality-adjusted life-years (QALYs) for PBC versus 2.53 QALYs for ABC, with associated costs of $434,504.74 and $489,969.88, respectively. PBC emerged as the dominant treatment strategy, providing both superior health outcomes and lower costs. One-way sensitivity analysis identified drug costs as the most influential parameters, with atezolizumab and pembrolizumab costs being primary drivers of cost-effectiveness results. Two-way sensitivity analysis demonstrated that pembrolizumab would need to exceed $82.6/mg or atezolizumab would need to decrease below $5.29/mg to alter cost-effectiveness conclusions. Probabilistic sensitivity analysis confirmed robust results, with ABC having only 0.54% probability of cost-effectiveness at the $150,000/QALY threshold.
Conclusion From a U.S. healthcare perspective, the PBC regimen demonstrates both superior efficacy and cost-effectiveness (better health outcomes with lower total costs) compared to the ABC regimen for first-line treatment of R/M-CC, establishing PBC as the dominant treatment strategy. A price reduction for atezolizumab could potentially enhance the cost-effectiveness profile of the ABC regimen. These findings provide valuable economic evidence to guide clinical decision-making for first-line treatment of R/M-CC, particularly in selecting value-based treatment strategies.

PO_109

转录组联合代谢组探讨不同宫颈癌细胞化疗敏感性差异的可能机制

张海光 杨君*
新乡医学院第一附属医院

研究目的 化疗是晚期宫颈癌患者的主要治疗方式，不同患者对化疗药物的敏感性不同，目前没有生物标志物能够预判不同患者的化疗敏感性。肿瘤细胞系是研究肿瘤化疗敏感性的有力武器。本研究拟比较两种宫颈癌细胞（C33A 及 HeLa）对阿霉素（DOX）、5-氟尿嘧啶（5-FU）、卡铂（CBP）、顺铂（DDP）等化疗药物的敏感性，并通过转录组联合代谢组的方法比较两种细胞的基础差异，希望找出能够做为预判宫颈癌化疗敏感性的生物标志物。
材料与方法 对 C33A 细胞及 HeLa 细胞加入不同浓度的 DOX、5-FU、CBP、DDP，MTT 法计算不同药物处理后的细胞活力并计算半抑制浓度值（IC50）；收集对数生长期的细胞，转录组联合代谢组分析两种细胞转录组和代谢组的差异；转录组分析时阈值为 LogFC>1，Qualue< 0.05；代谢组分析时阈值为 LogFC > 1, Qualue <0.05，VIP ≥ 1；获得差异基因及代谢产物的数量，并使用 KEGG 富集分析对比差异信号通路；按照 Qualue 值找到差异前 20 的基因及代谢产物，分析差异基因与代谢产物间的相关性；随后通过对代谢物和基因组学的指标进行通路富集分析，计算代谢物和基因组学差异指标显著富集的 pathway；选取感兴趣的 pathway，分析其差异表达的代谢物及基因。
结果 通过计算得到 C33A 细胞和 HeLa 细胞对于四种化疗药物的 IC50 值分别为 DOX（1.86uM，2.77uM），5-FU（2.28 uM，4.04uM），CBP（0.23uM，0.21uM），DDP（64.66uM，73.45uM）；转录组数据显示 C33A 相较于 HeLa 差异表达的基因共计 5642 个（上调 2799 个，下调 2843 个），这些基因主要富集在轴突导向、ECM 受体相互作用、肿瘤信号通路、心律失常、Rap1 信号通路等；代谢组数据显示差异的代谢物共计 322 个（其中上调 186 个，下调 136 个），这些代谢物主要富集在核苷酸代谢、ABC 转运蛋白、氨基酸生物合成、D-氨基酸代谢、蛋白质消化和吸收等信号通路；代谢组和基因组联合分析发现共富集的通路主要有：硫胺素代谢、蛋白质消化和吸收、烟酸和烟酰胺代谢、矿物吸收、黑色素生成、肿瘤的中心碳代谢等信号通路；进一步分析发现肿瘤的中心碳代谢信号通路中差异的代谢产物和基因产物较多。
结果 由于中心碳代谢通路的不同，导致 C33A 细胞和 HeLa 细胞对不同的化疗药物具有不同的敏感性。

PO_110

Correlation between dosimetric parameters and hematologic toxicity in cervical cancer patients undergoing intensity-modulated pelvic radiotherapy

Shuang Zhao[2] Xi Yang[1] Manni Huang[1*]
1.Cancer Hospital Chinese Academy of Medical Sciences
2.Cancer Hospital Chinese Academy of Medical Sciences

Objective This study aimed explored the association of bone marrow(BM) dosimetric parameters with hematological toxicity in cervical cancer patients undergoing radiotherapy (RT) utilizing artificial intelligence (AI) technology (Software Copyright Registration Number 2023SR0150365) to determine organs at risk (OARs). By accurately delineating bone marrow regions and analyzing radiation doses, this study will provide a theoretical foundation for the application of AI in predicting hematological toxicity.
Methods A total of 141 cervical cancer patients who received pelvic Volumetric Modulated Arc Therapy(VMAT) and/or chemotherapy (sequential or concurrent) at the Department of Gynecology, Cancer Hospital of the Chinese Academy of Medical Sciences from March 2019 to December 2019 were enrolled in the study. AI-based automatic segmentation of CT images was performed to delineate pelvic bone marrow and its subregions (ilium, lower pelvis, lumbarsacral spine, and head of femur). Specific volumes that received 10-40 Gy (V10, V20, V30, V40) were calculated, and baseline clinical characteristics of the patients were recorded. Hematological toxicity(HT) endpoints included grade ≥2 (HT2+) and ≥ 3 (HT3+) leukopenia, neutropenia, anemia, or thrombocytopenia. The association of dosimetric parameters with hematological toxicity was explored through Logistic regression.
Results Among the 141 patients, 107 (75.8%) experienced HT2+ and 33 (23.4%) experienced HT3+. Univariate analysis revealed that chemotherapy and age were correlated with the occurrence of HT2+. Multivariate analysis demonstrated that femur-V30, femur-V40, and chemotherapy were the independent predictors of HT3+.
Conclusion: This study highlights the potential of AI-based delineation of OARs to precisely assess bone marrow dosimetric parameters in patients with cervical cancer. Our results underscore the need to optimize radiotherapy to minimize bone marrow dose and volume, thereby mitigate hematological toxicity and enhance treatment tolerance. However, chemotherapy appeared to have a more significant effect on hematological toxicity than dosimetric parameters, and patients receiving combined neoadjuvant and concurrent chemotherapy are at a higher risk of hematological toxicity.

PO_111

The role and mechanism of TNFR2 in regulating glycolysis in uterine cervical cancer development

Jinwen Ye Baoxia Cui[*]
Qilu Hospital of Shandong University

Objective
1. To clarify the impact of TNFR2 on glycolysis in cervical cancer;
2. Clarify the mechanism by which TNFR2 regulates glycolysis in cervical cancer cells;
3. The impact and mechanism of TNFR2 inhibition on the antitumor effect of PD-1 monoclonal antibodies were studied.
Methods
1. Detect the impact of TNFR2 on the metabolic pathways of cervical cancer through nontarget metabolic sequencing.

2. The expression levels of key glycolytic enzymes and c-Myc in cervical cancer patient and normal cervical paraffin tissue specimens were detected through immunohistochemical techniques.
3. 2-NBDG, lactate and ATP detection kits were used to detect changes in glucose uptake, lactate production and ATP production in cervical cancer cells after changes in TNFR2 expression, respectively; a Seahorse Cell Energy Metabolism Analyzer was used to detect ECAR of cervical cancer cells after changing the expression level of TNFR2.
4. According to immunohistochemistry, the immunohistochemical score of c-Myc in normal cervical tissues was 0.3±0.3, which was lower than that of cervical cancer tissues (4.8±0.5, P<0.01); GSEA revealed that TNFR2 and c-Myc are related to glycolysis, and Western blotting was used to detect changes in the expression of c-Myc and key glycolytic enzymes after changes in TNFR2 expression.
5. We conducted a "recovery" experiment on the PI3K/Akt/mTOR/c-Myc pathway through the PI3K inhibitor LY294002, the mTOR inhibitor rapamycin, the c-Myc inhibitor 10058-F4, and the Akt activator SC-79 and detected the pathway with Western blot-related molecular changes.
6. The HK2 inhibitor 2-DG inhibited cervical cancer growth in nude mice overexpressing TNFR2, verifying that TNFR2 promotes the growth of cervical cancer cells by regulating glycolysis.
7. Flow cytometry was used to detect the expression level of PD-1 on lymphocytes in the cervical cancer tumor microenvironment;
8. Cervical cancer cells overexpressing TNFR2 were cocultured with PBMCs isolated from the peripheral blood of healthy people, and an anti-PD-1 monoclonal antibody was added. The changes in the percentage of apoptotic cervical cancer cells and lymphocyte phenotypic changes in the TNFR2-overexpressing group were compared with those in the control group.

Results

1. Immunohistochemistry showed that the immunohistochemical score of HK2 was 1.5±1, the immunohistochemical score of PKM2 was 1.5±0.5, and the immunohistochemical score of LDHA was 1.5±0.5, which was lower than that of HK2 7.5±1.5 (P<0.01), PKM2 4.5±0.5(P<0.01) and LDHA 4.3±0.7 (P<0.01) in cervical cancer tissues.
2. Nontarget metabolic sequencing enrichment showed that the most closely related relationship between TNFR2 and cervical cancer cell metabolism is the central carbon metabolism of tumors.
3. After overexpressing TNFR2, the lactate production of cervical cancer cells was significantly increased; after knocking down TNFR2, the glucose uptake, lactate production and ATP production of cervical cancer cells were significantly reduced.
4. In view of immunohistochemistry, the level of c-Myc expression in cervical cancer tissue was greater than that in normal cervical tissue (P<0.01); GSEA of TCGA data revealed that TNFR2 was positively correlated with c-Myc, and both were related to glycolysis. There was a positive correlation, and consistent results were obtained by detecting changes in protein levels when proteins were extracted from cells whose TNFR2 level changed; inhibiting PI3K, mTOR, and c-Myc reversed the increase in the expression of key glycolytic enzymes caused by TNFR2 overexpression, and cervical cancer cell apoptosis increased. Activating Akt can restore the expression levels of key glycolytic enzymes that are reduced due to TNFR2 knockdown and reduce the apoptosis of cervical cancer cells.
5. The use of the glycolysis inhibitor 2-DG inhibited the growth of cervical cancer xenograft tumors promoted by TNFR2 overexpression.
6. Increased lactic acid can cause an imbalance in the expression of PD-1 between CD8+ T cells and Tregs.
7. In the cervical cancer tumor microenvironment, the expression level of PD-1 on eTregs is greater than that on CD8+ T cells;
8. Cervical cancer cells that overexpress TNFR2 not only escape from lymphocytes but also exhibit a poor response to anti-PD-1 monoclonal antibodies. Moreover, the PD-1 expression level of eTregs cocultured with cervical cancer cells overexpressing TNFR2 were also greater than those in the control group.

Conclusions

1. Cervical cancer is a tumor with high glycolysis. TNFR2 promotes glycolysis in cervical cancer cells through the PI3K/Akt/mTOR/c-Myc pathway, inhibits cervical cancer cell apoptosis, and promotes tumor progression.

2. TNFR2 causes an imbalance in the expression of PD-1 in eTregs and CD8+ T cells, which may affect the efficacy of anti-PD-1 monoclonal antibodies.

PO_112

RPRD1B as a Prognostic and Therapeutic Target in Cervical Lesions Through Fanconi Pathway–Mediated PARP Inhibitor Sensitization

Xiaodie Liu
Shandong Provincial Hospital Affiliated to Shandong First Medical University

Background Cervical cancer remains one of the most prevalent malignancies affecting women worldwide, with a disproportionately high disease burden in developing countries such as China, where incidence continues to rise. The pathogenesis of cervical cancer follows a stepwise trajectory from low-grade squamous intraepithelial lesions (LSIL) to high-grade lesions (HSIL) and ultimately to invasive carcinoma. Notably, the transition from LSIL to HSIL represents a critical turning point, as the risk of malignant transformation increases sharply. Identifying molecular markers that can predict and potentially intercept this progression is of great clinical importance. RPRD1B (also known as CREPT), a transcriptional co-regulator with oncogenic properties in multiple tumor types, has yet to be fully investigated in the context of cervical lesion evolution. This study aims to explore the clinical relevance and mechanistic role of RPRD1B in cervical intraepithelial neoplasia, and to evaluate its potential as a prognostic biomarker and therapeutic target for halting disease progression.
Methods Transcriptomic data from TCGA and GEO databases were analyzed to investigate the association between RPRD1B expression, disease stage, and patient prognosis. Immunohistochemical analysis was performed on cervical specimens encompassing normal cervix, LSIL, HSIL, and cervical cancer to validate differential RPRD1B expression, with diagnostic and prognostic performance evaluated via receiver operating characteristic (ROC) curve analysis. Weighted gene co-expression network analysis (WGCNA) was employed to identify key gene modules related to cervical neoplastic progression and to ascertain the centrality of RPRD1B within these networks using Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) pathway enrichment analyses. In vitro assays involved RPRD1B knockdown or pharmacological inhibition with NHC in cervical cancer cell lines, combined with PARP inhibitor olaparib treatment. The combinatorial effects of RPRD1B suppression and olaparib treatment were assessed by CCK-8 and colony formation assays to evaluate cell proliferation, flow cytometry for cell cycle and apoptosis analysis, and western blotting to determine changes in cell cycle regulatory proteins. DNA damage levels were further examined by immunofluorescence staining of γ-H2AX foci. Co-immunoprecipitation assays were performed to confirm the interaction between RPRD1B and FANCD2. Furthermore, patient-derived xenograft (PDX) models were utilized to assess the therapeutic potential of targeting RPRD1B to enhance olaparib sensitivity in vivo.
Results RPRD1B expression increased progressively from normal cervix to LSIL, HSIL, and peaked in cervical cancer, correlating with poorer prognosis. In the GEO cohort, RPRD1B showed good diagnostic performance in distinguishing CC from LSIL (AUC = 0.767), HSIL (AUC = 0.688), and normal tissue (AUC = 0.796), and also differentiated HSIL from LSIL (AUC > 0.65). WGCNA identified RPRD1B as a key node in lesion progression, enriched in pathways related to cell cycle control, Fanconi anemia, and DNA repair. Functional inhibition of RPRD1B, alone or combined with olaparib, suppressed proliferation, induced apoptosis, and caused S-phase arrest, accompanied by downregulation of checkpoint proteins. γ-H2AX immunofluorescence revealed increased DNA damage following RPRD1B blockade, especially under combinatorial treatment. In vivo, combined NHC and olaparib therapy reduced tumor growth and prolonged survival in PDX models. Co-immunoprecipitation confirmed interaction between RPRD1B and FANCD2, supporting its role in DNA repair regulation.

Conclusion RPRD1B drives cervical carcinogenesis by orchestrating cell cycle progression and DNA repair, positioning it as a dual-function biomarker for diagnosis and prognosis. Importantly, its inhibition potentiates PARP inhibitor efficacy, highlighting RPRD1B as a compelling therapeutic target in cervical cancer management.

PO_113

Investigating causal relationship between circulating serum metabolites and cervical cancer: a Mendelian randomization study

Shaojie Zhao* Rui Gu Shangjin Li Shuqin Dai
无锡市妇幼保健院

Background Previous observational studies have reported an association between circulating serum metabolites and cervical cancer. However, due to the confounding factors, such as lifestyle and pre-existing medical conditions, establishing a causal relationship between them is difficult. We used Mendelian randomization (MR) to elucidate the causal relationship between circulating serum metabolites and cervical cancer occurrence.
Methods This study analysed Whole Gene Association Study (GWAS) data from the GWAS and the IEU Open GWAS project, focusing on metabolites and their association with cervical cancer. The metabolite dataset comprised 7824 samples, whereas the cervical cancer data included 605 samples from the bbj-a-98 dataset (out of a total of 90,336 samples) and 909 samples from the ebi-a-GCST90018817 dataset (out of a total of 239,158 samples). MR analysis was performed using the inverse variance weighted method (IVW), after which robust sensitivity analysis was performed to verify the MR hypothesis and the causal relationship between metabolites and cervical cancer.
Results Based on the MR analysis of the metabolites and their relationship with cervical cancer in the two datasets, IVW analysis revealed a positive causal relationship between the amino acid metabolite N-acetylglycine and cervical cancer in the ebi-a-GCST90018817 dataset (OR=1.751,95%CI:1.049-2.920, P=0.0319). Similarly, in bbj-a-98, the causal relationship was statistically significant (OR=2.411,95%CI:1.003-5.795, P=0.0492), indicating that the amino acid metabolite N-acetylglycine had a positive causality in the two different cervical cancer datasets. Allantoin had a negative causal effect on cervical cancer(ebi-a-GCST90018817: OR=0.337, 95%CI:0.141-0.808, P=0.0147; bbj-a-98: OR=0.136, 95%CI:0.020-0.901, P=0.0387). Results from both data sets also showed no heterogeneity between N-acetylglycine and cervical cancer (Cochran's Q: Pebi=0.3684, Pbbj=0.5398) or horizontal pleiotropy (MR-Egger: Pebi=0.2995, Pbbj=0.7714).
Conclusions The amino acid metabolite N-acetylglycine within circulating serum metabolites could contribute to cervical cancer, and allantoin is a protective factor against cervical cancer. These findings might offer novel perspectives in terms of cervical cancer diagnosis and treatment strategies.

PO_114

Chemoradiotherapy-induced CD24+/MUC16+ tumor cells drive macrophage polarization and cervical cancer recurrence

Wenzhi Kong Jinwei Miao*
Department of Gynecologic Oncology, Beijing Obstetrics and Gynecology Hospital, Capital Medical University. Beijing Maternal and Child Health Care Hospital, No.17 Qihelou Street, Beijing, 100006, China

Purpose The recurrence of cervical cancer following concurrent chemoradiotherapy (CCRT) poses a significant clinical challenge, with the underlying mechanisms remaining poorly understood. This study aims to elucidate the cellular and molecular mechanisms by which CD24 and MUC16 drive resistance to CCRT and tumor recurrence in cervical cancer through single-cell RNA sequencing (scRNA-seq) analysis. Specifically, we investigate the role of tumor cell stemness, macrophage polarization, and immune evasion in promoting treatment resistance and disease progression.
Materials and Methods We conducted scRNA-seq on matched cervical cancer specimens obtained from three patients before and after standard CCRT. Following rigorous quality control, the scRNA-seq data were processed through established bioinformatics pipelines, including dimensionality reduction, cell type annotation, and subpopulation analysis with particular focus on tumor cell clusters and macrophage subsets. Subsequent analyses encompassed: (1) evaluation of tumor cell stemness properties using defined stemness gene signatures, (2) pseudotime trajectory reconstruction to delineate the expression dynamics of CD24 and MUC16 during cell state transitions, and (3) differential gene expression profiling to identify CCRT-responsive pathways. For experimental validation, we performed flow cytometry analysis and immunofluorescence to quantify treatment-induced alterations in immune cell composition, including absolute counts and proportional changes of T cell subsets and macrophage polarization states (M1/M2). Statistical significance was defined as $p < 0.05$.
Results scRNA-seq analysis demonstrated that cervical cancer cells developing resistance to CCRT acquired pronounced stem-like properties, as evidenced by significant upregulation of stemness markers CD24 and MUC16. Pseudotime trajectory analysis revealed that these cells were positioned at the starting point of differentiation, with CD24 and MUC16 expression gradually decreasing over the course of cellular differentiation. After CCRT, the proportion of M2-type tumor-associated macrophages (TAMs) markedly increased, accompanied by significant upregulation of Siglec-10 and Siglec-9 expression in these macrophages. Cellular interaction analysis demonstrated that CD24+ and MUC16+ tumor cells engage with macrophages via SIGLEC10 and SIGLEC9 receptors, driving their polarization toward an immunosuppressive M2 phenotype. Most importantly, SIGLEC10+ and SIGLEC9+ macrophages suppressed T cell function through the PD-L2/PD-1 immune checkpoint axis, thereby promoting tumor immune evasion.
Conclusion This study identifies a critical mechanism by which CCRT induces stem-like properties in cervical cancer cells, fostering a immunosuppressive TME through macrophage reprogramming and T cell inhibition. The CD24/SIGLEC10 and MUC16/SIGLEC9 axes represent novel therapeutic targets to disrupt the crosstalk between resistant tumor cells and immune cells, potentially improving CCRT efficacy. Our findings underscore the importance of targeting post-CCRT immune evasion pathways to prevent recurrence and enhance patient outcomes. Future studies should explore combination therapies involving stemness inhibitors and immune checkpoint blockade to overcome CCRT resistance in cervical cancer.

PO_115

Expression of potentia antibody-drug conjugates (ADC) targets in neuroendocrine carcinoma of the cervix

tianhui he Xiaoxia Jiang Zheng Li*
Department of Gynecologic Oncology, Yunnan Cancer Hospital, The Third Affiliated Hospital of Kunming Medical University， Peking University Cancer Hospital Yunnan

Objective (s) Neuroendocrine carcinoma of the cervix (NECC) is a rare type of cervical cancer with an incidence rate of only 1.4% of all cervical malignant tumors, but the prognosis is extremely poor, with a 5-year survival rate of less than 35% of patients. Currently, the treatment of NECC is still based on surgery or radiotherapy combined with chemotherapy, and effective targeted therapeutic agents are still lacking. Therefore, searching for clinicopathologic factors and potential therapeutic targets that can predict the poor prognosis of patients is the key to improving the prognosis of NECC patients. In recent years, antibody-drug conjugates (ADC) have begun to emerge in the backline treatment of cervical cancer, but due to the rarity of NECC, there are no large-sample-based clinical studies to detect the expression of common ADC targets in NECC or to guide the development of specific ADC drugs. In this study, we intend to retrospectively analyze the clinicopathological factors of NECC patients receiving antitumor therapy in our center and the expression of emerging ADC drug targets, to clarify the clinicopathological factors affecting the recurrence and death of NECC patients, and to provide a basis for identifying the population with a high risk of clinical recurrence and death and exploring the development of targeted ADC drugs for this group of patients, so as to ultimately improve the prognosis of this high-risk group of patients.

Methods

1. Data collection and screening: 220 NECC patients who received antitumor treatment in Yunnan Cancer Hospital between 2000 and 2023 were retrospectively included, and 173 visiting patients were finally screened for survival analysis based on clinicopathological factors through the inclusion and exclusion criteria, and 91 patients with feasible immunohistochemistry (IHC) analyses were included for ADC target detection.
2. Survival analysis and correlation analysis: Cox proportional risk model was used to analyze the association between clinicopathological factors and patients' prognosis, and Log-rank test was used to evaluate the correlation between the expression of each target and clinicopathological factors.
3. Immunohistochemistry: IHC was used to detect the expression of 9 ADC targets, including B7-H3, DLL3, and ROR1, in the 91 cases of the retrospective NECC cohort, and semi-quantitative scoring was performed based on the intensity of staining and the proportion of positive cells, respectively.

Results In this study, the analysis of clinical data from 173 patients with NECC who met the inclusion and exclusion criteria revealed that FIGO stage, tumor size, and vaginal invasion detected on gynecological examination were independent prognostic factors affecting patient recurrence and death. Among them, the risk of death was elevated 36.67-fold in patients with stage IVB compared with those with early stage ($P<0.001$), the risk of recurrence (HR=2.32, P=0.023) and death (HR=3.38, P=0.024) were significantly increased in patients with imaging-measured tumor maximum diameter >4 cm compared with those with tumor maximum diameter ≤4 cm, and vaginal invasion detected on gynecological examination was an independent risk factor (HR=3.75, P=0.023). In addition, serum markers NSE ≥16.3 ng/mL and CEA ≥5 ng/mL were associated with an elevated risk of recurrence and death, respectively. In terms of treatment, patients who received surgery had a 72% lower risk of recurrence compared with those who received chemoradiotherapy only (HR=0.28, $P<0.001$), but multifactorial analysis did not show independent significance, which may be related to selection bias (early patients were more likely to receive surgery). Both neoadjuvant chemotherapy and adjuvant chemotherapy were able to affect the recurrence-free survival time (PFS) and overall survival time (OS) of patients, in which ≥5 cycles of total chemotherapy significantly reduced the risk of recurrence (HR=0.55, P=0.020), while neoadjuvant and adjuvant radiotherapy did not show survival benefit. Regarding the expression rate of ADC targets, the positive expression rates of B7-H3, DLL3 and ROR1

amounted to 97.80%, 72.53% and 73.62%, respectively, in which patients with high expression of B7-H3 (39.56%) were significantly correlated with parietal infiltration (P=0.039); EGFR (positive rate of 56.04%) and BCL-2 (positive rate of 51.65%) were respectively were associated with elevated CEA (P=0.043) and imaging-suggestive lymph node metastasis (P=0.027), while EGFR-positive patients also had a significantly higher risk of recurrence (P=0.048).

Conclusion(s)

1. FIGO stage and vaginal invasion suggested by gynecological examination are independent risk factors for the risk of death in NECC patients.
2. Surgery can reduce the risk of recurrence in patients with NECC, but it is necessary to select the applicable population in conjunction with the staging. Neoadjuvant chemotherapy and adjuvant chemotherapy can reduce the risk of recurrence and death, and the total number of chemotherapy courses ≥5 can significantly reduce the risk of recurrence.
3. B7-H3, DLL3 and ROR1 are ideal targets for future ADC drug development for NECC patients. Secondly, positive EGFR expression was associated with elevated CEA and recurrence, while high BCL-2 expression was associated with lymph node metastasis in patients.
4. The low expression rates of Nectin-4 and TF in NECC suggest that they differ from the common pathologic types of cervical cancer, and ADC drugs used for other pathologic types of cervical cancer may not be applicable to NECC patients.

PO_116

5-ALA 光动力疗法治疗宫颈上皮内瘤变的疗效及安全性评价：Meta 分析与系统评价

郭玮琳 邱丽华*
上海交通大学医学院附属仁济医院

研究目的 5-ALA 光动力疗法（5-ALA PDT）作为一种靶向、微创的治疗方式，因其可保留宫颈解剖结构、降低对生育能力的影响，近年来在宫颈上皮内瘤变（CIN）的治疗中被广泛应用。本研究旨在通过 Meta 分析评价其相较于临床指南推荐疗法（消融治疗、手术治疗及随访观察）的疗效及安全性，为临床实践提供循证依据。

材料与方法 系统检索 PubMed、Embase、Cochrane Library 等数据库，检索时间截止至 2025 年 5 月 1 日，纳入比较 5-ALA PDT 与消融、手术或随访观察治疗 CIN 的对照研究，提取总有效率、病变完全缓解率、复发率与进展率、HPV 清除率、不良反应等结局指标，采用随机效应模型进行 Meta 分析，评估合并效应量及异质性。

结果 共纳入 15 项研究，涵盖 2822 例 CIN 患者。Meta 分析显示 PDT 组的总有效率显著高于随访观察组（OR = 16.09，95% CI：7.76-33.37）和消融组（OR = 2.57，95% CI：2.00-3.30），与手术组差异无统计学意义（OR=1.05，95%CI：0.57-1.95），完全缓解率亦呈相似趋势。PDT 组病变进展率显著低于其他方法（OR =0.38，95% CI：0.19-0.78），病变复发率显著低于消融组（OR =0.32，95% CI：0.23-0.45）但高于手术组（OR =1.76，95% CI：1.15-2.69）。在 HPV 转阴方面，随访 6 个月时 PDT 组 HPV 转阴率显著优于对照组（OR =15.06，95% CI：3.15-71.87）及消融组（OR =1.46，95% CI：1.01-2.13），且与手术组差异不显著（OR =1.12，95% CI：0.70-1.78）。尤其相比于手术组，PDT 在高危型 16/18 型 HPV 感染转阴方面展现出与手术组几乎一致的效力（OR =1.03，95% CI：0.64-1.64），HPV 再次感染率（OR =1.89，95% CI：0.36-13.74）。安全性方面，PDT 常见的不良反应包括下腹部疼痛、分泌物增多，外阴瘙痒等；通常程度轻微，可自行缓解。严重不良反应如阴道流血（OR =0.12）、感染（OR =0.29）、宫颈管粘连（OR =0.25）在 PDT 中发生率显著低于其他疗法。

结果 5-ALA- PDT 治疗的总有效率显著优于观察组及消融组，疗效与手术组相近；可一定程度降低 CIN 的进展与复发风险，其 HPV 清除效果与手术相当。同时具有良好安全性，严重不良反应发生率低。为希望保留宫颈功能的 CIN 患者提供了一种有效且保守的治疗选择。

PO_117

Genetic analysis and clinical treatment of Neuroendocrine carcinoma of the cervix: current advancements and future directions

tianhui he Xiaoxia Jiang Zheng Li*
Department of Gynecologic Oncology, Yunnan Cancer Hospital, The Third Affiliated Hospital of Kunming Medical University，Peking University Cancer Hospital Yunnan

NECC is a rare and highly malignant tumor with limited clinical treatment efficacy. However, in recent years, with the advancement of molecular genetics technology, significant progress has been made regarding the genetic background and mutation spectrum of NECC and its impact on clinical treatment. This article describes the genetic analysis studies of NECC, emphasizing relevant gene mutations, tumor genomic features, molecular biomarkers, and their potential applications in clinical diagnosis and treatment.NECC displays specific genetic mutation characteristics, with key roles played by the TP53, RB1, and PIK3CA genes in the occurrence, differentiation, and invasive capabilities of NECC.Furthermore, NECC possesses unique genetic mutation traits compared to other histological types of cervical cancer, with mutations in genes such as SMAD4, RET, EGFR, and APC exclusive to NECC. From a regulatory mechanism perspective, the PI3K/AKT/mTOR, Wnt/β-catenin, and Notch pathways also contribute to the differentiation and progression of NECC.This article describes the potential of multiple targeted therapeutic strategies in NECC. Targets including HER2, EGFR, c-MET, RET, DLL3, B7-H3, ROR1, BCL-2, and SEZ6, which exhibit significant expression and mutation profiles in a wide range of neuroendocrine tumors, are molecules that may become new therapeutic targets. Antibody-Drug Conjugates (ADCs) of targets such as DLL3, B7-H3, and SEZ6 have demonstrated promising results in clinical trials of certain neuroendocrine cancers, providing new hope for precision therapy of NECC. Moreover, the article discusses current treatment strategies, including surgery, radiotherapy, chemotherapy, immunotherapy, and targeted therapy, analyzing various treatment modalities' clinical efficacy and limitations. Finally, the article anticipates future research directions in the genetics of NECC and personalized treatment approaches, providing new insights for improving clinical prognosis in NECC.

PO_118

The disturbed vaginal and ascitic microbiota in patients with adenocarcinoma of the cervix

Leilei Zhu Sensen Shi Huan Yi*
Fujian Maternity and Child Health Hospital

Background The aim of this study is to characterize vaginal microbiome disturbance in patients with cervical cancer (CC) and identify specific genera that can be used as diagnostic or prognostic markers. We also investigate the ascitic flora of CC patients.
Methods This study included 32 CC patients and 44 intraepithelial neoplasia (CIN) patients as the control group. There were 18 squamous cell carcinoma (SCC) patients and 14 adenocarcinoma (ADC)

patients. We collected vaginal secretions from all patients and ascites from 16 SCC and 12 ADC patients. 16S rRNA sequencing was adopted to analyze the vaginal and ascitic samples.
Results Our results indicated that the vaginal bacterial abundance in the CC group was significantly higher than that in the CIN group. The CC group demonstrated a non-significant trend of decreasing proportion of Lactobacillus (CIN: 60.89%, CC: 52.37%; $p > 0.05$) and increasing proportion of anaerobic bacteria, such as Prevotella (CIN: 0.14%, CC: 0.79%; $p < 0.05$) and Sneathia (CIN: 0.21%, CC: 1.57%; $p < 0.05$). Correlation analysis indicates that the abundance of Lactobacillus was negatively correlated with the abundance of those anaerobic bacteria. We also found that the ADC patients exhibited a significantly higher vaginal and ascitic microbiota abundance compared to SCC patients.
Conclusions Our study validated that vaginal flora abundance was associated with severity of cervical lesions. We also found that compared to SCC patients, the ADC patients exhibited significantly higher microbial complexity in both vaginal and ascitic microbiota.

PO_119

CXCL8 Drives Cervical Cancer Radioresistance via PI3K/AKT: Reparixin as a Radiosensitizer

Qinghong Hu Yuan Li Liping Han*
The First Affiliated Hospital of Zhengzhou University

Objective To investigate the molecular mechanisms by which CXCL8 mediates radioresistance in cervical cancer cells through in vitro and in vivo experiments, providing evidence for reversing acquired radioresistance in cervical cancer.And to integrate public datasets (TCGA-CESC, GSE52903, and radiosensitivity regulator datasets) to identify differentially expressed genes related to cervical cancer radiosensitivity regulators including CXCL8, construct clinical predictive models, and analyze their correlations with tumor immune microenvironment, drug sensitivity, and predictive efficacy, thereby identifying potential therapeutic targets.
Methods After transient transfection of SiCXCL8 with radiotherapy resistant cell strains HeLa-RR and SiHa-RR, cell viability, cell clonogenic ability, cell apoptosis and cell cycle detection were performed.After the increase of exogenous CXCL8, cell viability, cell clonogenesis, apoptosis and cell cycle were performed.Western Blot Test the protein expression of PI3K, phosphorylated PI3K, Akt, and phosphorylated Akt in the PI3K / AKT signaling pathway;An animal model of cervical cancer and the corresponding parental cell strain tumor-bearing nude mouse were constructed.The tumor-bearing nude mice were divided into four groups according to the vaccinated cells: HeLa-RR, HeLa, SiHa-RR and SiHa. According to whether they were irradiated, the growth and tumor growth of each group were observed, and the tumor tissue was detected by HE staining; the expression of CXCL8 in the tumor tissue was detected by immunohistochemistry.Cervical cancer acquired radiotherapy resistant cell tumor-bearing nude mice were divided into control group and experimental group according to whether to receive Reparixin reparisine. The growth and tumor growth of each group of nude mice were observed, and the expression of CXCL8 in tumor tissue was detected by immunohistochemistry.Data set collection: TCGA-GETx-CESC, GSE52903, and regulators of radiosensitivity.Differential analysis obtained differentially expressed radiosensitivity regulators including CXCL8 and performed external dataset expression validation.One-factor cox analysis and lasso-cox model construction and external dataset efficacy validation.Correlations between differentially expressed radiosensitivity regulators and clinical characteristics, model predicted efficacy of immunotherapy, differences between immune infiltrates in high and low risk group samples, and prediction of drug sensitivity were analyzed.
Result Knockdown of CXCL8 enhanced the radiotherapy of cervical cancer acquired radiation-resistant cell strains Hela-RR and Siha-RR, sensitivity;Increasing exogenous CXCL8 induces certain radiotherapy resistance properties in the parental cell strains Hela and Siha;Western Blot The test results indicated that PI3K and Akt phosphorylation of radiotherapy-resistant cells were inhibited; PI3K

and Akt phosphorylation after increase of exogenous CXCL8 of parental cell strains HeLa and SiHa;By constructing an animal model of cervical cancer acquired radiation- resistant cell tumor-bearing nude mice, the tumor volume of nude mice in the radiation-resistant group was slower than that of the parental cell group;After the removal of the tumors, the volume and weight of the tumors in the parental cell group decreased significantly after radiation irradiation compared with the radiation-resistant cells group.HE staining results showed that the necrosis of tumor tissue was less than the parental cell group; the immunohistochemical test results showed that the expression of CXCL8 in the tumor tissue was higher than that in the parental cell group;In the radiotherapy-resistant cell nude mice blast group, in the Reparixin group, tumor regression was evident after radiation irradiation compared with the control group. Differential expression analysis of the TCGA-CESC dataset, the FDR less than 0.05 and the absolute log2FC value greater than 2 were the threshold screening, yielding 3368 downregulated, 1421 upregulated a total of 4789 differentially expressed genes, and intersection with CRRF, obtaining a total of 96 differentially expressed CRRF.A total of 96 significant CRRF were selected by p value less than 0.05, and the eight genes with the most significant p value were CXCL8, IFI30, HK2, SPP1, IGF1, PAX9, SLC22A3, and ABCB1.Based on prognostic related genes, the redundant genes were removed using LASSO-cox, and finally 13 key genes were obtained to construct survival prediction model of radiosensitivity regulators (AUC 0.812, AUC 0.813,0.813, AUC 05.853) and successfully verified by the GSE52903 data set; median prediction model, high and low groups significantly correlated with OS ($p < 0.001$), was an independent predictor of OS ($p < 0.05$).The survival prediction model of radiosensitivity regulator was constructed combined with the Nomogram model of clinical factors (1 year AUC 0.727, AUC 0.784, and AUC 50.806); the validation results of GSE14404 data set showed the median value of the Nomogram model and the predictive value of patients on chemotherapy effect (AUC = 0.633);The correlation results of the radiosensitivity regulator survival prediction model and the tumor immune microenvironment showed that the infiltration of activated B cells, activated CD8T cells and neutrophils was significantly different in low groups ($p < 0.001$); the correlation analysis between the model and chemotherapeutic drug sensitivity showed significant differences between cisplatin in low groups ($p < 0.05$).

Conclusion CXCL8 knockdown restores radiation sensitivity of acquired radiation-resistant cervical cancer cell strains; increased exogenous CXCL8 induces radiation resistance in the parental cell strain HeLa.In the nude mouse xenograft model, cells in the acquired radiotherapy- resistant cell group had strong radiotherapy resistance properties, and inhibiting CXCL8 enhanced the radiotherapy sensitivity of radiotherapy-resistant xenograft tumors in nude mice.96 differentially expressed CRRF, after univariate cox regression and LASSO-cox to remove redundant genes, finally acquired the TOP8 key genes CXCL8, IFI 30, HK2, SPP 1, IGF1, PAX 9, SLC22A3, and ABCB1.The radiosensitivity regulator related prognosis model and data set verification were successfully constructed, and the independent prognostic ability of the radiosensitivity regulator prognosis model was successfully constructed combined with the Nomogram model of clinical factors and GSE14404 data set verification, and the median low grouping showed that Chu has good efficacy in predicting the chemotherapy effect of patients.GEPIA2 combined with immunohistochemistry showed that the expression of CXCL8 in cervical carcinoma was higher than that in paracancerous tissues.Combined with the results, this study proposes the target of potential reversal RT resistance: CXCL8.

PO_120

Quality of life in patients with cervical cancer after radical hysterectomy —— based on the CC-PRO137 scale: A prospective cohort study

Ling Qiu[1] Hui Wang[1] Yue Yin[1] Hao Feng[1] Ting Zhou[1] Jiayu Wang[1] Danze Qiao[3] Beili Wang[1] Miao Miao[1] Junda Zhao[4] Yan Du[1] Xiaofei Ye[2] Hua Jiang[1] Xin Wu[1*]

1.Obstetrics and Gynecology Hospital of Fudan University
2.Department of health statistics, naval medical university, Shanghai
3. High School Affiliated to Fudan University, Shanghai
4.The First Teaching Hospital of Xinjiang Medical University, Xinjiang

Objective Cervical cancer is the most prevalent tumour of the female reproductive system. In light of the prevailing trend towards younger age groups and the favourable prognosis for cervical cancer, the focus has shifted to the quality of life of patients in the long term and the impact of treatment on mood. However, there is a paucity of reliable data on these issues.The objective of this study was to evaluate the alterations in physiological, psychological and social life status of cervical cancer patients following treatment using the CC-PRO137 scale and to examine the particular clinical challenges presented by the scale.

Materials and Methods This prospective study was conducted from July 2021 to December 2023 in six regional centres in China. A total of 368 subjects who underwent initial surgery for cervical cancer were enrolled in this study, and they were followed up with the CC-PRO137 scale at two time periods: six months after the end of treatment and one year later. The researcher compared the status of subjects' quality of life in different conditions by detecting subtle changes in physiological functioning, psychological functioning, and social life status of cervical cancer patients after surgery. The data analysis was conducted between December 2024 and March 2025.

Results 368 of the 400 subjects recruited (92%) completed the scales for both time periods. In the initial period, the most significant influences on quality of survival were found to be the adjuvant treatment modality and age. Simultaneous radiotherapy was found to have a significant impact on patients' overall urinary function ($t=-2.968$, $p=0.003$), neurological function ($t=-3.579$, $p<0.001$), and ovarian function ($t=-3$.The findings of the study demonstrated that the patients' lymphedema status ($t=-3.933$, $p<0.001$) and sexual life ($t=-3.354$, $p=0.001$) were significantly affected, while the patients' emotional burden of anxiety and depression was aggravated. Furthermore, it was observed that luminal surgery had a greater impact on the patients' urinary and neurological functions. As time elapsed, patients demonstrated enhanced physical, psychological and social well-being beyond that observed at the initial one-year mark. Individuals over the age of 45 exhibited a more pronounced depression compared to those below this age ($t=-2.636$, $p=0.009$). The impact of chemotherapy on neurological function persisted ($t=-1.146$, $p=0.037$). Over time, the impact of surgical access on patients' quality of life did not demonstrate a significant difference. The disease stage did not differ significantly between patients in either time period.

Conclusions Through data collection from the scale and patient feedback on their self-perceived situation, we can understand the changes in the physiological and psychological statuses of cervical cancer patients after surgery and further reflect their dynamic recovery process. With this information, clinicians can assess patients' quality of life at any time and under any circumstances. They can then provide targeted, accurate diagnostic and therapeutic guidance and psychological support at relevant stages to help patients recover better.

PO_121

m6A 修饰的 circ_0002828 可与 EIF4A3 结合降低 DLGAP5 的稳定性进而抑制宫颈癌的进展

杨美琴
上海市第一妇婴保健院

研究目的 近年来，子宫颈癌的发病率和死亡率都很高，而且有年轻化的趋势。研究表明，环状 RNA（circRNA）在癌症的发生和发展中起着关键作用。一些 circRNA 已被证实参与 N6-甲基腺苷（m6A）修饰，并能够与 RNA 结合蛋白相结合。这些 circRNA 在宫颈癌中的功能和机制尚不清楚。
材料与方法 使用基因表达综合数据库（GEO）筛选与宫颈癌相关的 circRNA。通过体内和体外实验评估 circ_0002828 对宫颈癌增殖、转移和血管生成的影响。通过 RNA 免疫沉淀、RNA 下拉实验、荧光原位杂交、免疫荧光染色蛋白印迹及拯救实验等方法研究 circ_0002828 在宫颈癌中的调控机制。
结果 circ_0002828 在宫颈癌中显著下调，并与宫颈癌患者的肿瘤大小呈负相关。circ_0002828 在体外和体内均能抑制宫颈癌的增殖、转移和血管生成。circ_0002828 含有 m6A 修饰位点，YTHDF2 能够与 circ_0002828 结合促进其以 m6A 依赖的方式降解。EIF4A3 能够与 DLGAP5 mRNA 结合增强其稳定性。circ_0002828 可与 DLGAP5 mRNA 竞争性结合 EIF4A3，导致 DLGAP5 表达降低，gankyrin 表达升高，从而激活 p53 信号通路，抑制肿瘤的发生发展。
结论 circ_0002828 作为肿瘤抑制因子，通过与 EIF4A3 结合降低 DLGAP5 的稳定性，从而激活 p53 信号通路，抑制宫颈癌的进展。circ_0002828 有望成为宫颈癌治疗的新靶点。

PO_122

延伸野调强同步放化疗联合腔内近距离放疗在局部晚期宫颈癌中的临床应用

王光宇 张福泉 胡克*
北京协和医院

研究目的 本研究旨在前瞻性评估延伸野调强放疗（extended-field intensity-modulated radiotherapy, EF-IMRT）联合同步化疗与高剂量率腔内近距离放疗（high-dose-rate brachytherapy, HDR）在腹主动脉旁淋巴结（para-aortic lymph nodes, PALN）或髂总淋巴结（common iliac lymph nodes, CILN）转移的局部晚期宫颈癌患者中的疗效与安全性。
材料与方法 本研究为多中心前瞻性队列研究，纳入 2015 年 10 月至 2018 年 7 月期间来自中国 11 家医院的 204 例经病理或影像学证实的 PALN 或 CILN 阳性的宫颈癌患者（FIGO 2009 分期为 IB–IIIB）。所有患者均接受 EF-IMRT 联合每周顺铂同步化疗，并在外照射结束后行 HDR。主要终点为无进展生存期（progression-free survival, PFS），次要终点包括总生存期（overall survival，OS）、局部无进展生存期（local progression-free survival，LPFS）、远处转移无进展生存期（distant metastasis-free survival，DMFS）以及治疗相关不良事件。使用 Kaplan-Meier 法计算生存率，不良事件依据 CTCAE V4.0 标准评估。
结果 中位随访时间为 68.7 个月，3 年和 5 年 PFS 分别为 68.3%和 64.6%，OS 分别为 76.4%和 69.5%；5 年 LPFS 和 DMFS 分别为 83.1%与 70.1%。亚组分析发现，PALN 阳性患者的 PFS、OS、LPFS 及 DMFS 均显著低于 PALN 阴性患者（P 均<0.05）；在 PALN 阳性患者中，CILN 阳性者的 PFS 和 DMFS 亦明显低于 CILN 阴性者（P<0.05）。治疗失败率为 36%，其中 60 例出现远处转移，以肺转移（13.2%）及纵隔或锁骨上淋巴结转移（约 12%）最常见。在安全性方面，50.5%的患者出现 3 级血液

学毒性，16.7%发生 3 级以上急性胃肠道毒性，慢性 3 级胃肠道毒性仅见于 3%患者。尽管急性不良反应较常见，但大多为可控的短期反应，严重长期毒性较少，表明治疗方案整体耐受性良好。

结果 本研究为目前最大规模前瞻性多中心研究之一，验证了 EF-IMRT 联合同步化疗与 HDR 腔内近距离放疗在 PALN 或 CILN 转移的局部晚期宫颈癌患者中的疗效与可接受的毒性。该方案在保证疗效的同时实现了良好的局部控制和长期生存，尤其对于高危亚组（如 PALN 及 CILN 阳性患者）亦显示出一定生存获益，支持 EF-IMRT 作为该类患者的有效治疗策略。

PO_123

从盆腔到锁骨的致命跳跃：一例宫颈腺鳞癌多器官转移的诊疗启示与挑战

李尚瑾 赵绍杰*
无锡市妇幼保健院

研究目的 探讨宫颈腺鳞癌（ASC）的侵袭性生物学行为、多线治疗后的全身转移模式及临床管理难点，为罕见转移病例的诊疗策略提供依据。

材料与方法 回顾性分析 1 例 42 岁女性宫颈腺鳞癌患者的诊疗全程（2022 年 4 月–2025 年 1 月）。初诊经宫颈活检、广泛子宫切除术明确病理分期（IIIC1p 期），接受新辅助化疗（TC 方案×1）、根治性手术及术后辅助放化疗（TC×5+盆腔外照射 24 次）。复发后通过胸壁肿物穿刺活检、PET-CT 及肠镜病理确诊转移灶，采用多线联合治疗（贝伐珠单抗+卡度尼利+多西他赛/白蛋白紫杉醇+顺铂×5）及局部放疗，动态监测血清 SCCA、影像学及分子病理特征。

结果 1.初诊表现：宫颈 6cm 菜花样肿物，术后病理示腺鳞癌（腺癌占比 30%），伴脉管广泛癌栓及多组淋巴结转移（左盆腔 3/16、右盆腔 1/17、左宫旁 2/2）；2.转移与复发：术后 18 个月出现右锁骨下胸壁肿物（病理：鳞癌），血清 SCCA 持续升高（峰值>4.3ng/ml）；PET-CT 提示胸壁、肋骨、椎体转移； 3. 全身扩散：多线靶向免疫化疗后仍进展至胸肌、回盲瓣、结肠肝曲（肠镜活检证实为宫颈源性转移癌）及阴道残端；4.治疗反应：联合方案未能有效控制 SCCA 水平及转移进展，补充放疗后病灶仍泛发；5.病理异质性：原发灶为腺鳞癌，胸壁转移灶呈鳞癌表型（CK5/6+、P63+），结肠转移灶免疫组化（CDX2-/P16+）支持宫颈来源。

结论 本例揭示宫颈腺鳞癌具有高侵袭性（初诊即伴脉管癌栓、淋巴结转移）及显著时空异质性（腺-鳞成分动态演变）。胸壁-骨-肠道多系统转移模式罕见，可能与淋巴逆流、EMT 激活及免疫微环境抑制（PD-L1 阴性/TMB 低）相关；现行辅助方案（TC 化疗+放疗）对高危 ASC 控制不足，需早期整合靶向治疗（如抗血管生成药、双抗 AK112）及基于分子分型（PIK3CA/HER2/BRCA）的个体化策略。动态监测 ctDNA（如 PIK3CA E545K 突变）可早于影像学预警进展，未来需探索多组学指导下的精准治疗框架。

PO_124

The efficacy of 5-aminolevulinic acid photodynamic therapy for persistent high-risk HPV infection in patients without cervical lesions: A retrospective cohort study

Ruiju He Lihua Qiu*
Shanghai Jiao Tong University School of Medicine Affiliated Renji Hospital

Background and objectives Although persistent high-risk human papillomavirus (HR-HPV) infection is is the leading cause of cervical cancer, effective therapeutic strategies remain limited. Photodynamic therapy (PDT) ,with its advantages of being targeted, minimally invasive, and reproducible, has demonstrated excellent efficacy in patients with cervical lesions who wish to have children, and has helped clarify HR-HPV infection in these patients. However, further research is required to determine the efficacy of PDT for persistent HR-HPV infection without cervical lesions. Therefore, we propose studying the clearance rate of 5-aminolevulinic acid photodynamic therapy (ALA-PDT) for persistent HR-HPV infection without lesions to inform clinical treatment.
Materials and Methods This retrospective cohort study included 169 patients with persistent HPV infection and no cervical lesions, who underwent ALA-PDT at Renji Hospital, Shanghai Jiao Tong University School of Medicine, between September 2018 and August 2024. All patients underwent three sessions of PDT only, without receiving other treatments such as cervical laser therapy. Patients were assessed for HPV infection and cytological changes at six and twelve months after treatment. Multivariable logistic regression was used to examine the associations between baseline characteristics and outcomes at 6 and 12 months post-treatment.
Results PDT demonstrated progressive efficacy, with clearance rates increasing from 61.9% at 6 months to 73.7% at 12 months post-treatment. Younger patients exhibited higher clearance rates compared to older cohorts, especially at 6 months post-treatment (OR=1.029, p=0.044 at 6 months; and OR=1.027, p=0.198 at 12 months). Prior loop electrosurgical excision (LEEP) reduced efficacy at 6 months post-treatment (OR=2.601, p=0.018), but there was no statistical difference at 12 months post-treatment (OR=2.239, p=0.130). HPV16/18-related infections had higher HPV clearance rates at 12 months post-treatment, though this difference was not statistically significant (84.4% vs. 68.7%; p=0.096).
Conclusion ALA-PDT is an effective treatment for persistent HR-HPV infection in patients without cervical lesions, particularly in younger patients and those with no history of LEEP.

PO_125

Clinicopathological characteristics and a nomogram for predicting overall survival in patients with postoperative endocervical adenocarcinoma

Yu Dou Xuewang Guo Yanying Xu*
Tianjin Medical University Second Hospital

Aims To assess clinicopathological characteristics, identify prognostic factors, develop and validate a nomogram for predicting overall survival(OS) in postoperative endocervical adenocarcinoma (ECA) patients, according to the Surveillance, Epidemiology, and End Results (SEER) Program.
Methods Postoperative patients with pathologically confirmed diagnosis of cervical cancer between 2004 and 2015 were obtained from the SEER database.The survival before and after the 1:1 propensity score matching (PSM) analysis were analyzed using Kaplan-Meier and log-rank tests.Univariate and multivariate Cox regression models were subsequently utilized to identify independent prognostic

factors to construct the nomogram. Validation of the predictive discrimination and accuracy of the nomogram by concordance index (C-index) and calibration curve.
Results Of the 10,827 patients,6,709 had cervical squamous cell carcinoma (SCC) and 4,118 had ECA. In unmatched cohort, patients with ECA had a better prognosis compared to SCC (hazard ratio [HR]=0.87;95% confidence interval [CI] = 0.80 - 0.95;P=0.002).After matched, subgroup analysis showed that ECA patients were associated with a worse prognosis than SCC (HR=1.16;95% CI=1.03-1.31; P=0.017). Results of univariate and multivariate Cox regression analysis showed that age at diagnosis,race,grade,AJCC stage, AJCC T,AJCC N, AJCC M,radiotherapy,tumor size were independent variables(all P<0.05).Construct a predictive nomogram model for OS.The C-indexes were 0.791 (95% CI=0.771-0.812) and 0.826 (95% CI= 0.805 - 0.846) for training and validation cohorts, respectively,showed the good discriminative ability.Calibration curves verified an optimal accuracy of this prediction model.
Conclusions Postoperative ECA patients were associated with a worse prognosis than SCC. A nomogram was developed and validated to conveniently and accurately assess the survival and prognosis of postoperative ECA patients.

PO_126

DGKH 促进宫颈癌发生发展的机制研究

王舒燕 汪辉*
浙江大学医学院附属妇产科医院

持续性高危型人乳头瘤病毒（hr-HPV）感染是宫颈癌发生发展的主要原因，其中 HPV 基因组整合到宿主染色体是关键的癌变事件，然而，以往研究多关注整合位点邻近基因的改变，其对远端基因的长距离作用及其机制尚不明确。因此，本研究旨在探讨 HPV 在 13q22.1 位点整合后，如何通过长距离调控作用影响远端基因二酰基甘油激酶 θ（DGKH）的表达，并阐明 DGKH 作为促癌基因在宫颈癌发生发展中的具体生物学功能与分子机制，以期为宫颈癌的预后判断和靶向治疗提供新的分子标志物与科学依据。为实现这一目标，我们首先利用生物信息学方法，对 TCGA、GEO 等公共数据库及本课题组的多组学数据进行深度挖掘，通过比较 HPV 是否整合在 13q22.1 位点的患者样本转录组数据，筛选出表达上调的远端基因，随后，采用 13q22.1 位点特异性 HPV 基因敲入的 HaCaT 细胞模型及多种宫颈癌细胞系，通过 Western Blot、siRNA 干扰、质粒转染以及 CCK-8 增殖和 Transwell 迁移侵袭等一系列体外功能实验，系统验证 DGKH 的生物学功能；在机制探索方面，则通过 RNA 干扰、ChIP-PCR 及 RNA 测序等技术，深入探究其上下游调控通路。研究结果显示，在 HPV 于 13q22.1 位点整合的宫颈癌样本中，位于 13q14.11 的远端基因 DGKH 表达显著上调，且其高表达与患者的不良预后及 CD8+ T 细胞的低浸润水平显著相关；功能实验则进一步证实，敲低 DGKH 能显著抑制宫颈癌细胞的增殖、迁移和侵袭，而过表达 DGKH 则促进这些恶性生物学行为。机制研究揭示，转录因子 ELF1 能够直接结合 DGKH 的启动子区域以激活其转录，是其表达上调的关键；同时，我们发现 DGKH 能够显著增强宫颈癌细胞的巨自噬活性，这可能是其促进肿瘤细胞在应激环境下存活的关键途径。综上所述，本研究证实了 HPV 整合可长距离调控远端基因的表达，并首次揭示了 DGKH 在宫颈癌中的关键促癌作用，HPV 整合后通过激活转录因子 ELF1 上调远端基因 DGKH 的表达，DGKH 进而通过增强细胞的巨自噬水平，促进宫颈癌细胞的增殖与侵袭，并导致患者预后不良。这些发现不仅深化了对 HPV 致癌机制的理解，也表明 DGKH 可作为一个潜在的宫颈癌预后生物标志物和新的治疗靶点。

PO_127

基于可解释机器学习的宫颈癌术后症状性淋巴结囊肿发生风险预测模型的构建与内部验证

唐能欢 王一钥 张维民 李青芸 曾玉华*
川北医学院

研究目的 构建并比较 9 种机器学习模型对宫颈癌患者术后症状性淋巴结囊肿发生风险的预测效果，通过 SHAP(SHapley Additive exPlanations)可解释性分析识别关键决策因素，为临床个体化决策提供辅助支持。

材料与方法 本研究回顾性收集了 2023 年 1 月-2024 年 9 月于川北医学院附属医院就诊的 221 例宫颈癌患者临床数据，分为症状性淋巴结囊肿组（n=44）和无症状或无淋巴结囊肿组（n=177）。采用最小绝对收缩选择算子（least absolute shrinkage and selection operator, LASSO）回归方法对所有预测特征进行筛选，并通过九种机器学习算法（逻辑回归、支持向量机、K 近邻、随机森林、XGBoost、GBM、LightGBM、AdaBoost、CatBoost)构建预测模型。曲线下面积（AUC）被用来评价模型的效能，五折交叉内部验证被用来验证模型内部稳定性，SHAP 被用于对最优预测模型进行可解释化处理。

结果 本研究通过 LASSO 回归筛选出 5 个特征变量，包括糖尿病病史，术前单核细胞与淋巴细胞比值、术后全身炎症反应指数、肿瘤大小及手术入路。在 9 个机器学习预测模型中，逻辑回归，支持向量机和 catboost 预测模型表现出较好预测效能（AUC 均>0.75），在模型的五折交叉内部验证中，相比于其他的机器学习算法，CatBoost 模型（AUC=0.804，95%CI 0.664–0.943，B 评分 0.13）展现出了更好的预测效能。SHAP 分析显示，术前单核细胞与淋巴细胞比值,糖尿病病史,肿瘤大小是贡献前三的重要预测特征。

结论 在 9 个机器学习预测模型中，CatBoost 预测模型的预测效能最优，能有效预测症状性淋巴结囊肿的发生。此外，术前单核细胞与淋巴细胞比值,糖尿病病史,肿瘤大小对术后症状性淋巴结囊肿的发生预测具有重要作用。

PO_128

ATF5 介导 Wnt/β-catenin 信号通路促进宫颈癌增殖、迁移与侵袭的研究

石凤娟[1] 姚德生[2*]
1.南宁市第一人民医院 2.广西医科大学附属肿瘤医院

目的 宫颈癌是一种严重威胁女性生命安全的生殖系统恶性肿瘤，复发或转移的患者由于缺乏有效的治疗措施，总体预后较差。转录激活因子 5（ATF5）通过调控细胞凋亡、自噬、肿瘤发生、迁移、侵袭等多种途径，干预肿瘤的发生和发展，以及放化疗的敏感性，进而影响肿瘤病人的预后。然而 ATF5 在宫颈癌的作用和机制仍有许多未明之处。本研究旨在探究 ATF5 在宫颈癌的作用及机制，为制定以 ATF5 为靶点的宫颈癌靶向治疗提供新的思路和理论依据。

材料与方法 运用生物信息学方法分析公共数据库中 ATF5 的转录组数据。运用实时荧光定量 PCR、蛋白免疫印迹、免疫组织化学法检测 ATF5 在宫颈癌和正常宫颈中的表达情况，并对 ATF5 表达与临床病理因素和预后进行相关性分析和生存分析。运用慢病毒转染的方法下调宫颈癌 SiHa 细胞和 C-33A 细胞中 ATF5 的表达，通过细胞增殖实验、Transwell 细胞迁移和侵袭实验、划痕实验分析 ATF5 对宫颈癌细胞增殖、迁移、侵袭能力的影响。对 TCGA 数据库的宫颈癌转录组数据进行 GSEA 富集分析，筛选出有意义的信号通路。采用实时荧光定量 PCR 和蛋白免疫印迹法检测 Wnt/β-catenin 信号通路中 Wnt3a、β-catenin 的表达情况。

结果 宫颈癌组织和细胞中 ATF5 的表达较正常宫颈组织和细胞升高。ATF5 高表达组患者临床分期较晚，淋巴结转移和淋巴脉管间隙浸润发生率更高，两组患者年龄、病理类型、分化程度、肿瘤大小无差异。ATF5低表达患者的总生存期优于ATF5高表达患者。ATF5低表达组（shATF5组）细胞生长较空白对照组（NC 组）和阴性病毒对照组（shNC 组）缓慢。shATF5 组迁移和侵袭实验的穿膜细胞数较 NC 组和 shNC 组明显减少。shATF5 组划痕愈合速度较 NC 组和 shNC 组明显下降。富集分析显示ATF5 mRNA表达与Wnt/β-catenin信号通路正相关。shATF5组Wnt3a、β-catenin表达相较shNC组、NC 组均明显下降。

结论 ATF5 在宫颈癌的表达异常升高。ATF5 表达与临床分期、淋巴结转移、淋巴脉管间隙浸润正相关，与预后负相关。ATF5 介导 Wnt/β-catenin 信号通路促进宫颈癌 SiHa 细胞和 C-33A 细胞的增殖、迁移和侵袭。ATF5 可以作为预测宫颈癌病情严重程度和预后的指标，有望成为宫颈癌靶向治疗的一个新靶点。

PO_129

预测局部可切宫颈癌腹主动脉旁淋巴结转移的模型构建：一项真实世界研究

周明祎 高岩 王丹波*
辽宁省肿瘤医院

目的 手术病理分期证实的 IIIC2p 期宫颈癌的最佳治疗方案有待商榷，构建出精准预测局部可切宫颈癌腹主动脉旁淋巴结转移的模型具有重要的临床价值和意义。

材料与方法 收集辽宁省肿瘤医院 2018 年 1 月至 2022 年 3 月所有行广泛性子宫切除术及盆腔和腹主动脉旁淋巴结清扫的宫颈癌病例资料进行真实世界病例对照研究。评估患者年龄、肿瘤标志物 SCC、MR 评估的宫颈病灶大小、病理组织学类型、分化程度、浸润深度及脉管癌栓以及 CT 测量的盆腔和腹主动脉旁淋巴结大小等指标。通过二分类 Logistic 回归评估各因素对腹主动脉旁淋巴结病理阳性的预测价值。

结果 一共收集到 374 例宫颈癌，中位随访时间达 67.77 个月。其中腹主动脉旁淋巴结转移 15 例（4.01%），盆腔淋巴结转移 73 例（19.52%），仅有一例为腹主动脉旁淋巴结阳性而盆腔淋巴结阴性，其余均为盆腔及腹主动脉旁淋巴结同时阳性。盆腔和/或腹主动脉旁淋巴结转移患者无进展生存时间（PFS）显著短于淋巴结阴性患者（Log-rank P = 0.007）（图 1）。初治前通过 MR 评估的宫颈病灶>4cm 及全腹 CT 提示盆腔和/或腹主动脉旁淋巴结异常肿大两项指标与术后病理证实的腹主动脉旁淋巴结阳性相关（P = 0.002, P = 0.046），而年龄、肿瘤标志物 SCC、病理学类型、分化程度、浸润深度及脉管癌栓无统计学意义。结合宫颈病灶大小及 CT 评估淋巴结肿大情况两项指标构建的评分公式：腹主动脉旁淋巴结阳性预测得分=大小（>4cm 赋 1 分）+ CT 评估（淋巴结肿大赋 1 分），能够较为准确地预测腹主动脉旁淋巴结转移，ROC 曲线下面积为 0.80 （95%CI 0.68 - 0.92），P < 0.001（图 2）。

结果 局部病灶可切但盆腔及腹主动脉旁淋巴结转移这部分宫颈癌患者预后差，初治时可以通过宫颈肿块大小及 CT 评估的盆腔及腹主动脉旁淋巴结肿大情况有效预测腹主动脉旁淋巴结病理阳性。

PO_130

Hierarchical Feature Selection for Prognostic Prediction in Individuals with T1-2NxM0 Cervical Adenocarcinoma

Shihuang Liu[1] Zihan Deng[1] Hong Zhao[3] Huan Yi[1,2*]
1.Fujian Medical University 2.Fujian Maternity and Child Health Hospital
3.Minnan Normal University

Objective Given the aggressive nature and rising incidence of cervical adenocarcinoma, innovative machine learning method for feature selection and supervised prediction have been proposed as complementary approaches to improve prognostic prediction in cervical adenocarcinoma compared to traditional methods. This study aims to identify optimal features for predicting survival in women with T1-2NxM0 cervical adenocarcinoma and establish an evaluation index to distinguish individuals who may benefit from adjuvant treatment.
Materials and Methods Data from the Surveillance, Epidemiology, and End Results (SEER) database (2004–2015) were used to identify 3,155 patients with T1-2NxM0 cervical adenocarcinoma. Patients were divided into training and validation cohorts. A total of 22 features were analyzed by four hierarchical feature selection algorithms: fuzzy-rough set-based recursive reduction (FSRR), max-relevance and min-redundancy (MRMR), Fisher, and feature selection via joint ℓ2,1-norms minimization (FSNM). Predictive performance was evaluated using accuracy, hierarchical F1-score (FH), and tree-induced error (TIE). Prognostic prediction models were constructed with XGBoost and assessed through area under the curve (AUC) metrics and Kaplan-Meier (K-M) curve. Cox regression was used for clinical validation.
Results The FSRR-based CARE (cervical adenocarcinoma risk evaluation) model, utilizing only 6 key features (hysterectomy type, lymph node status, FIGO stage, N stage based on American Joint Committee on Cancer (AJCC), number of tumors, and multi-primary tumors), demonstrated superior performance in the validation cohort. It achieved a predictive accuracy of 81%, hierarchical F1-score (FH) of 0.89, tree-induced error (TIE) of 128, and an AUC of 0.72. These metrics significantly outperformed other feature selection methods (MRMR, Fisher, FSNM) when using the same number of features. Cox regression confirmed all 6 features were independent prognostic predictors ($p<0.05$). Critically, high-risk individuals identified by the CARE model showed significantly improved survival outcomes when receiving adjuvant treatment (chemotherapy or radiotherapy) compared to untreated high-risk patients.
Conclusions The FSRR model provides a robust survival prediction tool for cervical adenocarcinoma. Its superior performance with only six features highlights its clinical utility in optimizing individualized treatment strategies and improving prognosis.

PO_131

早期宫颈癌腹腔镜手术中子宫举宫器使用对患者预后影响的回顾性研究

包婉莹 李征宇*
四川大学华西第二医院

目的 本研究旨在评估举宫器在腹腔镜下广泛性子宫切除术中对早期宫颈癌患者无病生存期（DFS）和手术安全性的影响。
方法 本研究回顾性分析了 2019 年 7 月至 2022 年 7 月期间在四川大学华西第二医院接受手术的 IB1 - IIA2 期宫颈癌患者临床病理数据。患者被分为举宫器组（UM）和非举宫器组（NUM），并使用倾向性

评分匹配（PSM）进行校正，以减少潜在的混杂因素和选择偏倚。采用 Kaplan-Meier 法比较 UM 组和 NUM 组的 3 年 DFS。使用单因素和多因素 Cox 比例风险回归模型确定影响 3 年 DFS 的独立预后因素。
结果 本研究共纳入 749 名患者。其中，442 名患者（59.01%）接受了不使用子宫举宫器的腹腔镜手术，307 名患者（40.99%）接受了使用子宫举宫器的腹腔镜手术。UM 组和 NUM 组的 3 年 DFS 率无显著差异（95.11% vs 94.57%；P = 0.756）。在 PSM 后的队列中，DFS 也无显著差异（P = 0.396）。PSM 前后多因素 Cox 回归分析均显示，阳性阴道穹窿浸润是 PFS 的唯一独立危险因素（PSM 前：HR=2.35，P=0.033；PSM 后：HR=2.95，P=0.008）。在手术安全性方面，未举宫组的术中失血量低于举宫组（163.29 ± 132.35 mL vs 213.30 ± 166.83 mL，P < 0.001），但手术时间更长（390.31 ± 122.20 min vs 317.88 ± 99.84 min，P < 0.001）。然而，两组在具体的术中并发症（输尿管支架置入、膀胱修复、肠道修复）发生率上没有显著差异。术后平均住院时间也无显著差异（8.75±2.78 天 vs 8.78±3.65 天，P=0.073）。
结果 在 IB1 和 IIA2 期宫颈癌症患者中，广泛性子宫切除术中使用子宫举宫器不会对无进展生存期产生负面影响，也不会显著增加主要术中并发症的风险。阴道穹窿浸润是导致患者预后较差的重要因素。

PO_132

宫颈癌与血管生成素类似物 3 及整合素 3 的相关性及发病机制探讨

李灿怡
西南附属中医医院

宫颈癌是全球女性第四大常见癌症，居三大妇科肿瘤之首。其发病率及死亡率均居高不下，虽然在疫苗接种及筛查计划下发病人数明显减少，但对于晚期宫颈癌女性的治疗效果及预后仍不佳。宫颈癌对女性的生理健康有着巨大威胁。其发病认为与 HPV 持续感染密切相关，同时可能存在其他多种潜在发病机制。在宫颈癌的发生与进展中，肿瘤血管生成是一个十分重要的病理过程。ANGPTLs 是一类与血管生成素结构类似的蛋白质族群，多种癌症的发生、进展密切相关。血管生成素类似物 3(ANGPTL3)和整合素 β3(αvβ3)作为调控血管生成和肿瘤微环境的关键分子，在宫颈癌中的作用机制正逐渐受到关注。
研究目的 本文拟分析宫颈癌与 ANGPTL3、αVβ3 的相关性，并对宫颈癌潜在发病机制进行探讨。材料与**方法** 本文通过收集临床病例，通过 ELISA、WB、IHC 等方法检测宫颈癌患者癌变组织及血清样本中的 ANGPTL3、αVβ3 水平，并与良性肿瘤患者病变组织及血清样本中 ANGPTL3、αVβ3 水平进行对比分析。
研究结果 宫颈癌组患者血清及病变组织中 ANGPTL3、αVβ3 表达均高于良性病变患者，并在宫颈癌患者中 ANGPTL3、αVβ3 水平与肿瘤分期可能有一定的线性关系。
结论 ANGPTL3、αVβ3 可能通过促进血管生成、调控血管功能从而促进宫颈癌的发生发展。

PO_133

综述：p16/Ki67 双染技术在宫颈癌筛查中的应用进展与挑战

田端成 向阳*
中国医学科学院北京协和医院

宫颈癌是全球女性中最常见的癌症之一。其早期筛查和诊断对于降低发病率和死亡率至关重要。目前宫颈癌主要筛查方法为液基细胞学检查（liquid-based cytological test, LBC），HR-HPV 检测或两者联合筛查。然而这两种筛查方法均存在不足之处：LBC 主要依赖于细胞学病理医生的经验，导致 LBC 的诊断敏感性较低；HR-HPV 特异性较低，不能区别一过性感染和持续性感染，导致过多转诊阴道镜检查和过度治疗。近年来，p16/Ki67 双染技术作为一种新兴的分子标志物检测方法，在提高宫颈癌筛查的敏感性和特异性方面展现出显著优势。特别是在 HPV 阳性或细胞学异常的患者分流中，p16/Ki67 双染技术可以显著减少不必要的阴道镜检查。p16 是一种细胞周期调控蛋白，主要通过抑制细胞周期的进程发挥作用。在 HPV 感染引起的宫颈病变过程中，p16 的表达显著上调，在宫颈上皮内瘤变（CIN）和宫颈癌的发生发展过程中是重要的生物标记物。另外，Ki67 是一种增殖细胞核抗原，表达水平直接与细胞增殖活性相关。当前，尽管该技术在临床应用中取得了一定进展，但仍存在一些挑战，包括判读标准规范问题、结果解读的主观性以及对不同人群适用性的研究不足。因此，本文旨在综述 p16/Ki67 双染技术在宫颈癌筛查中的最新进展，探讨其与传统宫颈癌筛查方法的对比分析以及当前研究面临的主要挑战与未来的发展方向，为优化宫颈癌筛查策略提供科学依据。

PO_134

化疗重塑宫颈癌免疫微环境：机制、临床意义与研究展望

朱纳
辽宁省肿瘤医院

化疗在宫颈癌治疗中超越传统细胞毒作用，展现出显著的免疫微环境重塑功能。本文系统综述化疗对宫颈癌免疫微环境的重编程机制、临床转化价值及未来挑战。研究表明，以铂类、紫杉烷类及吉西他滨为代表的化疗药物通过三重路径重构肿瘤免疫微环境：（1）诱导免疫原性细胞死亡，释放 CRT、HMGB1、ATP 等危险信号，激活树突细胞并增强肿瘤抗原提呈；（2）重编程免疫细胞亚群：新辅助化疗（NACT）显著增加 CD8+T 细胞与 NK 细胞浸润，下调 Treg 细胞/MDSCs 比例，促进巨噬细胞向 M1 型极化；（3）改变肿瘤细胞免疫表型：同步放化疗上调 MHC-II 类分子表达，驱动肿瘤细胞向“上皮-免疫”（"Epi-Imm"）状态转化，该状态与干扰素信号激活及免疫细胞互作增强密切相关。

临床转化研究证实，NACT 诱导的 TIME 重塑具有显著增敏价值：病理完全缓解（pCR）患者呈现 CD8+T 细胞密度升高、三级淋巴结构形成及 VISTA+免疫细胞/CD8+T 细胞空间共定位等特征；"Epi-Imm" 转化状态可预测后续免疫检查点抑制剂（ICI）疗效；基于 COMPASSION-16 研究，PD-1/CTLA-4 双抗（卡度尼利单抗）联合化疗±贝伐珠单抗一线治疗显著改善全人群生存，为 PD-L1 阴性等难治亚群提供新方案。

当前挑战集中于化疗耐药引发的代偿性免疫抑制及残留肿瘤细胞异质性。未来需结合单细胞/空间多组学技术解析 TIME 动态演变，优化“化疗-ICI-靶向”三联策略，并开发基于基线免疫分型及化疗诱导生物标志物的精准治疗模型。深入探索化疗的免疫调节本质，将推动宫颈癌治疗从单纯细胞杀伤向微环境重构范式转变。

PO_135

Clinical Application of Gasless Laparoscopic Radical Hysterectomy with Upper Vaginal Sleeve Technique for Cervical Cancer A Comparative Study

wang maomao
广西医科大学附属肿瘤医院

Background Minimally invasive surgical approaches for early-stage cervical cancer have evolved significantly, with traditional laparoscopic radical hysterectomy (TLRH) being widely adopted. However, concerns regarding oncological outcomes and technical challenges have prompted the development of alternative techniques. Gasless laparoscopic radical hysterectomy (GLRH) with upper vaginal sleeve technique, performed without uterine elevation and pneumoperitoneum, represents a novel approach that may potentially address some limitations of conventional laparoscopic surgery. This technique aims to minimize tumor manipulation while maintaining the benefits of minimally invasive surgery. The gasless approach eliminates the need for CO2 insufflation, potentially reducing the risk of tumor cell dissemination through pneumoperitoneum-related mechanisms. Additionally, the upper vaginal sleeve technique and avoidance of uterine elevation may further reduce the risk of tumor spillage during surgery. However, comprehensive comparative data regarding the safety, feasibility, and efficacy of GLRH compared to TLRH remain limited. This study aimed to evaluate the safety and effectiveness of GLRH compared to TLRH in patients with early-stage cervical cancer.

Methods This retrospective comparative study included 82 patients with early-stage cervical cancer who underwent laparoscopic radical hysterectomy between [time period]. Patients were divided into two groups: those who underwent GLRH (n=41) and those who underwent TLRH (n=41). To minimize selection bias and ensure comparable baseline characteristics between groups, 1:1 propensity score matching was performed based on relevant clinical and demographic variables. The GLRH procedure was performed using an upper vaginal sleeve technique without uterine elevation and without pneumoperitoneum, while the TLRH group underwent conventional laparoscopic radical hysterectomy with CO2 insufflation. Primary outcomes included intraoperative parameters such as operative time, estimated blood loss, and intraoperative complications. Secondary outcomes encompassed postoperative recovery parameters, including time to bowel function recovery, duration of abdominal drainage, length of hospital stay, and changes in hemoglobin levels and D-dimer elevation. Both short-term and long-term complications were systematically evaluated, including urological complications (ureteral injury, bladder injury, urinary tract infection, urinary retention), gastrointestinal complications (bowel fistula, intestinal obstruction), vascular complications (lower extremity venous thrombosis), lymphatic complications (lymphangitis, lymphocyst, lymphedema), and other general complications such as postoperative fever. Pathological outcomes including tumor size, depth of invasion, lymph node yield, lymph node metastasis rate, lymphovascular invasion, and histological grade were compared between groups. Statistical analysis was performed using appropriate statistical tests, with $P<0.05$ considered statistically significant.

Results The median estimated blood loss was 200 (137.5, 500.0) ml in the GLRH group compared to 150 (95.0, 300.0) ml in the TLRH group, showing no statistically significant difference (P=0.055). The median operative time was significantly longer in the GLRH group at 272.5 (250.0, 305.0) minutes compared to 221.5 (191.5, 261.0) minutes in the TLRH group (P=0.004). Univariate analysis identified several risk factors for intraoperative blood loss ≥200ml, including BMI ≥24 kg/m², history of cervical conization, previous cesarean section, operative time ≥240 minutes, tumor size ≥2 cm, and the GLRH procedure itself (all $P<0.05$). Regarding perioperative complications, no statistically significant differences were observed between groups in the incidence of short-term surgical complications, including ureteral injury, bladder injury, urinary tract infection, urinary retention, bowel fistula, intestinal obstruction, lower extremity venous thrombosis, lymphatic complications, and postoperative fever (all $P>0.05$). Similarly, postoperative recovery parameters showed no significant differences between groups, including postoperative D-dimer elevation levels, allogeneic blood transfusion rates, degree of

postoperative hemoglobin decrease, time to bowel function recovery, duration of abdominal drainage tube placement, and length of postoperative hospital stay (all P>0.05). Long-term complications, including hydronephrosis, voiding dysfunction, lymphocyst formation, and lymphedema, showed no statistically significant differences in incidence rates between the two groups (all P>0.05). Pathological examination revealed no significant differences between groups in tumor size, median number of removed pelvic lymph nodes, pelvic lymph node metastasis rate, positive lymphovascular invasion rate, histological grade, or depth of tumor invasion (all P>0.05).
Conclusions Gasless laparoscopic radical hysterectomy with upper vaginal sleeve technique appears to be a safe and feasible alternative to traditional laparoscopic radical hysterectomy for early-stage cervical cancer. Despite a longer operative time, GLRH did not demonstrate a significant increase in short-term or long-term surgical complications compared to TLRH. The technique achieved comparable pathological outcomes, including adequate lymph node harvest and similar oncological margins. While the gasless approach may offer theoretical advantages in terms of reduced tumor manipulation and elimination of pneumoperitoneum-related risks, further long-term follow-up studies with larger sample sizes are needed to definitively establish the oncological outcomes and overall survival benefits of this technique. The impact on tumor prognosis requires continued observation and research to determine the long-term efficacy of GLRH compared to conventional approaches.

PO_136

宫颈神经内分泌癌术后预后因素及辅助治疗的探索

沈法权 姚德生* 邵如玉
广西医科大学附属肿瘤医院妇瘤科

目的 探讨宫颈神经内分泌癌（NECC）患者的预后因素及术后治疗方案的效果，为临床治疗提供参考。
方法 收集 2014 年 3 月至 2024 年 3 月的 88 例 NECC 手术患者的临床病理资料。通过 Kaplan-Meier 法分析患者的总体生存期（OS）和无进展生存期（PFS），使用 Cox 比例风险模型识别预后相关的独立因素，并比较不同治疗方式的效果。
结果 在 88 例 NECC 患者中，中位 OS 为 23.0 个月，中位 PFS 为 15.0 个月。单因素分析显示，肿瘤分期、脉管癌栓、淋巴结转移和肿瘤直径均显著影响患者的 OS 和 PFS（P＜0.05）。多因素分析结果表明，脉管癌栓是 OS 和 PFS 的独立不良预后因素（P＜0.05），术后辅助化疗为独立保护因素（P＜0.05），接受 EP 方案患者中位 OS 和 PFS 均较接受 TP、VAC 方案的患者长（P＞0.05），且化疗 4 程及以上患者的 5 年 OS 和 PFS 优于化疗 4 程以下患者（P＜0.05）。
结论 NECC 患者的预后较差，尤其是存在脉管癌栓者。术后辅助化疗能显著改善预后，EP 方案可能为最佳方案，免疫靶向治疗对 NECC 可能有效，未来需进一步研究以优化 NECC 的治疗方案。

PO_137

The Mediating Effect of Reproductive Tract Infections on the Association between HPV Infection and High-Grade Cervical Intraepithelial Neoplasia in Women

Dan Sun Jiangtao Fan*
Guangxi Medical University First Affiliated Hospital

Objective To investigate the mediating role of genital tract infections (specifically Ureaplasma urealyticum [UU] and bacterial vaginosis [BV]) in the association between human papillomavirus (HPV) infection and high-grade squamous intraepithelial lesion (HSIL), and to provide a theoretical basis and new insights for the clinical prevention and treatment of HPV-infected patients.
Methods A retrospective cohort study was conducted involving 627 female participants (203 in the HSIL group and 424 in the control group). Associations between HPV infection, UU/BV, and cervical HSIL were analyzed using logistic regression. The mediating effects of UU and BV were assessed by scaling regression coefficients to the logit scale and employing bootstrapping with 95% confidence intervals (CI). Further verify the mediating effect through Sobel coefficient multiplication and integral distribution method.
Results HPV infection (OR = 14.345, 95% CI: 7.098-28.990), UU infection (OR = 1.868, 95% CI: 1.267-2.753), and BV infection (OR = 2.637, 95% CI: 1.396-4.980) were identified as independent risk factors for cervical HSIL. Both UU (effect size = 0.024, 95% CI: 0.011-0.040) and BV (effect size = 0.072, 95% CI: 0.046-0.090) demonstrated significant mediating roles in the association between HPV infection and cervical HSIL.
Conclusion UU and BV in genital tract act as partial mediators in the pathway linking HPV infection to cervical HSIL, suggesting that clinical intervention targeting genital tract infections may help prevent the progression of HPV-related cervical lesions.

PO_138

Immunophenotyping in Peripheral Blood as a Prognostic Biomarker for Cervical Cancer Recurrence and Progression-Free Survival

zhao bingbing
Department of Gynecologic Oncology, Guangxi Medical University Cancer Hospital

Background The prognostic value of immunophenotyping in peripheral blood in cervical cancer patients remains unclear. This study aimed to analyze the associations between recurrent cervical cancer and immunophenotyping in peripheral blood, and develop model to predict progression free survival of cervical cancer.
Methods We analyzed the association between cellular immunity, humoral immunity, and complete blood count (CBC) profiles post-primary therapy and cervical cancer recurrence in 881 patients. A predictive model, the Peripheral Blood Derived Test (PBDT), was developed based on immune and CBC parameters using multivariate Cox regression and Lasso Cox regression to predict progression-free survival (PFS). A feasibility analysis was conducted to assess the predictive ability of the model.
Results A total of 165 patients with recurrent cervical cancer and 716 patients without recurrence were enrolled in our study. The recurrence group exhibited significantly higher proportions of B lymphocytes ($P = 4.125e{-}13$) and elevated levels of complement components C3 ($P = 0.009$), C4 ($P = 1.941e{-}06$), and IgG ($P = 0.015$), while the proportions of NK cells ($P = 0.019$) and T cytotoxic cells ($P = 0.033$) were notably lower. The PBDT model, constructed using Lasso Cox regression, demonstrated superior

performance with a higher C-index (0.79; 95% CI, 0.73-0.88) and greater 1-year (0.872; 95% CI, 0.783-0.962) and 3-year (0.809; 95%CI, 0.721-0.896) areas under the ROC curves (AUC) compared to the model based on multivariate Cox regression. Additionally, the PBDT model was validated to independently predict cervical cancer recurrence (P = 9.55e-14; HR, 4.92; 95% CI, 3.24–7.49), even after adjusting for known recurrence-associated factors such as SCC, radiation, and chemotherapy.
Conclusions The immunophenotyping in peripheral blood, encompassing cellular immunity, humoral immunity, and complete blood count (CBC) profiles, was found to be associated with cervical cancer recurrence. The PBDT model, developed using these immunophenotyping markers, serves as a promising prognostic biomarker for predicting cervical cancer recurrence.

PO_139

宫颈癌靶向靶点治疗研究进展

丁瑾晗
西南医科大学

宫颈癌（Cervical cancer）是一种常见的严重威胁女性生命健康的恶性肿瘤，在全球范围内具有较高的发病率，位居女性癌症第四位，宫颈癌复发及晚期患者治疗的挑战尤为严峻，传统治疗手段难以满足临床需求，因此迫切需要探索新的治疗策略。近年来，随着肿瘤学研究的深入，靶向治疗成为癌症治疗领域的新兴力量。这种治疗方式聚焦于细胞分子水平，精准地针对特定的致癌因子，通过设计特异性的靶向药物，实现对癌细胞的精准打击。与传统治疗相比，靶向治疗在安全性和有效性上展现出显著优势，为癌症患者带来了新的希望。在宫颈癌的治疗研究中，靶向干预药物的研发成为热点方向。这些药物有的已经进入临床试验阶段，展现出了一定的治疗效果；有的则在临床前研究中表现出良好的应用前景。本文旨在对宫颈癌靶向治疗的研究进展进行全面、系统的梳理与综述，以期为该领域的进一步发展和临床应用提供有价值的参考依据。

PO_140

卡度尼利联合顺铂用于局部晚期宫颈癌新辅助治疗疗效分析

谢娅
郑州大学第一附属医院

背景 局部晚期宫颈癌约占宫颈癌病例的 37%，由于治疗选择有限，预后不佳。卡度尼利是同时靶向 PDL-1 和 CTLA-4 的双靶点新型免疫检查点抑制剂，可能有助于解决这一难题。我们旨在探究卡度尼利联合顺铂治疗对局部晚期宫颈癌的疗效和安全性。
方法 我们在郑州大学第一附属医院开展的单臂、2 期试验中，我们纳入了未经治疗的宫颈癌患者（FIGO 2018 年分期为 IB3、IIA2 或 IIB/IIIC1r 且肿瘤直径≥4 厘米），以及 ECOG-PS 评分为 0 或 1 的患者。符合条件的患者接受 2-3 次的顺铂联合卡度尼利诱导化疗：第 1 天，卡度尼利（卡度尼利单抗 10mg/kg，静脉注射），第 2 天顺铂 75 毫克/平方米，静脉注射；治疗周期间隔 3 周。病情稳定或进展的患者接受同步放化疗，而完全缓解或部分缓解的患者则进行根治性手术。主要终点是由独立中央评审员根据实体瘤疗效评价标准 1.1 版评估的客观缓解率。在至少接受过二次卡瑞利珠单抗治疗的患者中分析了活性和安全性。本研究已在 ClinicalTrials.gov 注册，目前仍在进行中。
结果 2025 年 1 月 1 日至 2025 年 8 月 1 日期间，共纳入 10 名患者，所有患者均至少接受了两个周期的新辅助治疗。中位年龄为 52 岁。截至数据截止日期（2025 年 8 月 1 日），3 名（30%）患者完全缓解，6 名患者（60%）出现部分缓解，其中 1 名（10%）患者治疗效果不显著，9 名患者进行了宫颈癌根治术。新辅助顺铂免疫治疗期间最常见的 3 - 4 级治疗相关不良事件为骨髓抑制和电解质紊乱。3

名患者出现白细胞减少，均为 I-III 度（30%），1 例发生低钠血症（10%），脱 0 例患者发生脱发。未发生严重不良事件或治疗相关死亡。

结论 卡度尼利联合顺铂用于局部晚期宫颈癌新辅助治疗显示出良好的抗肿瘤效果，并且无脱发的治疗副作用，有望成为治疗局部晚期宫颈癌的一种新型疗法。

PO_141

非侵入性筛查的崛起：宫颈癌筛查的光学成像技术进展

李响 王丹波*
大连理工大学附属肿瘤医院（辽宁省肿瘤医院）

宫颈癌是全球女性常见的恶性肿瘤之一，其预后与早期发现密切相关。作为当前针对宫颈癌初筛异常结果患者的重要二级筛查手段，阴道镜检查在临床实践中发挥着关键作用。然而，该方法仍受限于图像分辨率、染色依赖及操作者经验差异等因素，难以全面满足客观、准确、快速的临床筛查需求。本文综述近年来应用于宫颈癌前病变及早期宫颈癌检测的多种新兴光学及光谱成像技术，探讨其技术优势、临床表现及未来发展趋势。

新兴宫颈成像技术主要包括荧光成像、高光谱成像、表面增强拉曼光谱、漫反射光谱、显微成像系统及光学相干显微技术等。这些技术依托光与组织成分的相互作用机制，突破传统成像对解剖学与形态学依赖，能够实时、无创地提供细胞代谢状态及分子层面的特征信息。其中，荧光成像利用内源性分子如还原型烟酰胺腺嘌呤二核苷酸与黄素腺嘌呤二核苷酸的荧光特性，通过分析氧化还原比反映组织代谢状态，在识别高级别鳞状上皮内病变方面优于传统染色方法。高光谱成像通过采集组织在多个波段的光谱反射特征，实现对不同组织类型的精细区分，尤其在 555–585 nm 波段内已观察到子宫颈上皮内瘤变（Cervical Intraepithelial Neoplasia, CIN）组织与健康组织间显著差异。表面增强拉曼光谱技术基于拉曼散射效应，通过纳米增强手段放大信号强度达 1014 倍，在宫颈脱落细胞、血液及体内组织中均表现出良好分类效能。显微成像方面，光学显微内窥镜、生命显微镜、宽场-高分辨率成像融合系统及光学相干显微镜等技术的临床研究显示，不仅能够实时获取亚细胞级别图像，还可与染色方法结合，显著提高宫颈病变分级与定位的准确性。

总体而言，新兴光学与光谱成像技术在宫颈癌筛查中的无创性、实时性及自动化特征，为减少不必要活检、提高诊断准确性提供了新的技术路径。它们标志着宫颈癌筛查模式正在由基于宏观组织形态的观察逐步迈向分子代谢层面的精准识别。未来，需进一步加强相关设备与操作流程的标准化，推动基于人工智能的图像分析算法发展，并通过大规模临床研究验证其诊断价值与适用性，以实现其在宫颈癌无创筛查的常规化推广应用。

PO_142

基于 R 语言的光动力疗法、宫颈环形电切术和宫颈冷刀锥切术治疗子宫颈高级别鳞状上皮内病变临床疗效的荟萃分析

张子玲
中国医学科学院肿瘤医院

宫颈高级别鳞状上皮内病变（high grade squamous intraepithelial lesion, HSIL）是宫颈癌的癌前病变，早期干预对阻断癌变至关重要。本研究通过荟萃分析系统评价宫颈环形电切术（Loop Electrosurgical Excision Procedure, LEEP）与冷刀锥切术（Cold-knife Conization, CKC）治疗 HSIL 的临床疗效及安全性。检索 2020 年 1 月至 2024 年 12 月中英文数据库（如 CNKI、PubMed 等），纳入 64 项研究（样本量总计 7762 例，其中 LEEP 组 3892 例，CKC 组 3870 例），采用 R 语言进行统计分析，比较术中指标（标本大小、锥切深度）、术后疗效（复发率、切缘阳性率、HPV 转阴率）及恢复情况（愈合时间）；LEEP 和 PDT 临床疗效相关文献纳入 7 篇，对其进行描述性分析。结果显示 LEEP 组术后愈合时间显著短于 CKC 组（均数差=-8.63 天，95%CI：-10.31~-6.95），复发风险降低 32%（RR=0.68，95%CI：0.49~0.94），切缘阳性率降低 27%（RR=0.73，95%CI：0.61~0.89），且切除标本更小（MD=-1.04，P<0.001）、锥切深度更浅（MD=-0.61 cm，P<0.001），但两组 HPV 转阴率无显著差异（P>0.05）。研究表明，LEEP 在减少手术创伤、促进术后恢复及降低复发风险方面更具优势，而 CKC 需结合个体化需求选择。PDT 在治愈率与 HPV 转阴率上具有一定优势，但 LEEP 在复发率和残留率控制方面可能表现更优，未来需更多高质量研究验证长期预后差异。

PO_143

FTO Orchestrates Glycolysis and Autophagy Crosstalk through m6A Demethylation to Drive Cervical Cancer Growth

Tianhui Niu Jinghui Jia*
Air Force Medical University，Air Force Medical center，PLA

Objective N6-methyladenosine (m6A) modification is recognized as a critical regulator of diverse cellular processes. Dysregulation of m6A is implicated in the initiation and progression of various cancers. However, the precise role of m6A, particularly through the action of its demethylase FTO, in cervical cancer (CC) pathogenesis remains unclear. This study aimed to elucidate the regulatory function of FTO in CC, focusing specifically on its effects on glycolytic metabolism and autophagy during cancer initiation and progression.

Methods We first assessed the differential expression of FTO in human cervical epithelial cells (HUCEC) and the cervical cancer cell line HeLa using RT-PCR. We then evaluated the impact of FTO over-expression on proliferation and invasion via cell viability, colony formation, and cell migration assays. The correlation between FTO levels and autophagy in HeLa cells was examined using immunoblotting, immunofluorescence, and electron microscopy. We established a shFTO stable cell line via lentiviral transduction and performed RNA-seq to identify potential molecular mechanisms underlying FTO's regulation of the autophagy pathway. Key findings were validated using RT-PCR, immunoblotting, and immunofluorescence. Furthermore, we explored the relationship between FTO and glycolytic metabolism using glucose consumption and lactate production assays to investigate the interplay between FTO-regulated autophagy and glycolysis.

Results We found that FTO promotes glycolysis by modulating the expression of the glycolytic enzyme PGAM1, while inhibiting autophagy via the C/EBPβ/AKT/mTOR pathway. Furthermore, our findings

indicated that autophagy exerts dual effects. Impaired autophagy protects against apoptosis, whereas active autophagy suppresses glycolysis by selectively degrading PGAM1, thereby promoting apoptosis. **Conclusion** Taken together, these results suggest that combination therapy targeting the m6A demethylase FTO, combined with interventions targeting glycolysis and autophagy pathways, holds promise as therapeutic strategies for cervical cancer. Our findings provide novel insights into the intricate regulatory network orchestrated by FTO's demethylase activity, which integrates glycolysis and autophagy to promote CC growth, highlighting FTO and its downstream pathways as potential therapeutic targets.

PO_144

宫颈癌 FIGO IIIC1 期预后因素及免疫治疗生存获益的多中心队列研究

宿晓曼 王宁* 黄家珍 魏威 孙银睿 牛志刚
大连医科大学附属二院

目的 分析 FIGO 2018 IIIC1 期宫颈癌的独立预后因素，评估帕博利珠单抗维持治疗对生存结局的影响。
方法 采用国家癌症中心数据库 2016-2023 年收治的 6,892 例 IIIC1 期患者数据，均接受根治性子宫切除及术后同步放化疗。通过多因素 Cox 回归确定预后因素，使用倾向评分匹配平衡基线差异后，比较帕博利珠单抗维持治疗组（412 例）与常规随访组（508 例）的生存差异。
结果
预后因素：阳性淋巴结≥4 枚患者死亡风险增加 2.05 倍（95%CI:1.62-2.60; p<0.001）；腺鳞癌病理使死亡风险升高 1.68 倍（95%CI:1.29-2.19; p<0.001）；淋巴结包膜外侵犯患者进展风险增加 1.83 倍（95%CI:1.45-2.31; p<0.001）
治疗对比：免疫维持组 3 年无进展生存率 81.5%（95%CI:78.2-84.5%）显著高于对照组 63.2%（95%CI:59.1-67.0%）（HR=0.54; 95%CI:0.42-0.70）；包膜外侵犯亚组接受免疫治疗后进展风险降低 52%（HR=0.48; 95%CI:0.35-0.66），交互作用检验 p=0.032
风险分层：高危组（≥4 枚阳性淋巴结伴包膜侵犯）5 年总生存率 41.5%（95%CI:37.8-45.2%）；低危组（≤3 枚淋巴结）5 年总生存率 84.2%（95%CI:81.5-86.7%）（p<0.001）
结果 阳性淋巴结≥4 枚及包膜外侵犯是 IIIC1 期宫颈癌的独立不良预后因素。帕博利珠单抗维持治疗显著改善 PD-L1 阳性患者生存，包膜外侵犯亚组获益尤为突出。临床实践中建议依据淋巴结特征实施风险分层管理。

PO_145

A Surgery-based Alternative Option Improved Prognosis of Cervical Cancer with Lymph Nodes Metastasis

Wang Hui[1] Shen Luo[1] Xiaolei Lin[2] Ling Qiu[1] Yue Yin[1] Yan Du[1] Weili Yan[3] Hua Jiang[1] Xin Wu[1*]
1.Obstetrics & Gynecology Hospital of Fudan University
2.Fudan University 3.Children's Hospital of Fudan University

Objective Stage IIIC of cervical cancer is when lymph node metastasis occurs. The NCCN guidelines recommend concurrent chemoradiotherapy as the first choice of treatment, but the outcomes remain unsatisfactory. We propose a surgery-based valuable alternative option for stage IIIC cervical cancer and evaluate the prognosis and influencing factors for patients treated this way.
Methods This study retrospectively collected data from patients with stage IIIC squamous cervical cancer who underwent surgery-based therapy in our hospital from January 2016 to December 2022. Patients were firstly conducted by radical cervical cancer surgery + pelvic lymph node dissection ± abdominal paraaortic lymph node dissection/biopsy. Postoperative comprehensive adjuvant therapy such as CCRT and systemic chemotherapy was given based on pathological results with reference to SEDLIS criteria and the four-factor model. The main outcomes were 5-year progression-free survival (PFS) and 5-year overall survival (OS). Kaplan-Meier method was used to estimate PFS and OS. Cox regression model was used for multivariate analysis. Cox proportional hazards models were performed to estimate the survival distributions. The impact of postoperative adjuvant treatment modalities on prognostic pairs was discussed using propensity score matching. Stratified analysis was performed to examine the risk factors.
Results The 5-year overall survival (OS) for stage IIIC squamous cervical cancer was 82.0% overall, with 83.8% for stage IIIC1 and 61.6% for stage IIIC2. The 5-year progression-free survival (PFS) was 77.1% overall, 79.3% for stage IIIC1, and 52.4% for stage IIIC2. Pathological subtype, postoperative treatment modality, and positive lymph node ratio were key independent prognostic factors for both OS and PFS. Tumor diameter >4 cm and positive paracervical infiltration were linked to PFS. No significant difference in OS or PFS was found between standard and non-standard adjuvant therapies. Additionally, certain factors, such as robotic surgery and undifferentiated tumor subtype, were associated with higher risks of local recurrence and distant metastases.
Conclusion The prognosis of patients with stage IIIC cervical squamous carcinoma treated with the surgery-based therapy is satisfactory. High-risk factors such as pathologic subtype, LNR, parametrial infiltration, large tumor diameter, and non-standard adjuvant therapy will adversely affect the patient's prognosis.

PO_146

基于宫颈癌类器官模型的白术内酯 I 体外抗肿瘤作用评价

韩月[1,2] 周晓蝶[1,2] 胡嘉琪[1,2] 施佳[1,2]
1.南京中医药大学附属医院 2.江苏省中医妇产生殖临床医学创新中心

研究目的 在已经发现白术内酯 I（AT-I）对宫颈癌 Hela、SiHa 细胞模型具有抗肿瘤效应的基础上，构建患者宫颈癌类器官模型，进一步从类器官模型角度明确 AT-I 对宫颈癌的抗肿瘤效应。
材料与方法 从患者宫颈癌组织获取原代宫颈癌细胞，并将获得的原代细胞加入宫颈癌完全培养基，通过胶滴培养的方式构建宫颈癌类器官；当大部分类器官直径超 100 μM 进行 HE 染色鉴定；当大部分空泡状类器官直径超 150 μM 且类器官边缘变厚或实心状类器官边缘出现凋亡碎片时进行传代；选用 1 例宫颈癌类器官筛选 AT-I 抗肿瘤效应的作用时间和浓度；选用 3 例类器官宫颈癌模型，采用类器官细胞活性检测试剂评价 AT-I 抗肿瘤效应。

结果 1. 宫颈癌类器官 P5 代复苏后第 7 天，可观察到类器官直径明显增大且数量增多，类器官增殖情况良好，平均直径约为 50μM；HE 染色显示宫颈癌类器官为致密或半致密球状体，细胞质呈粉色，细胞核呈蓝色，核质比较高，肿瘤细胞特征明显；给予 AT-I 处理后，类器官无特定形状；2. 给予宫颈癌类器官 AT-I 6 个浓度梯度（400、200、100、50、25、12.50 μM）处理后发现，作用 72 小时后明显观察到类器官凋亡现象，IC50 为 79.94μM；3. 给予 AT-I 6 个浓度梯度（100、50、25、12.5、6.25、3.125 μM）处理 3 例宫颈癌类器官模型，作用 72 小时后，AT-I 的抗肿瘤效应 IC50 分别为 116.6μM、7.549×10-15μM、105.9μM。

结论 宫颈癌类器官能够成功构建且稳定传代；AT-I 对类器官宫颈癌模型具有抗肿瘤效应，但不同宫颈癌类器官模型对 AT-I 的敏感程度不完全相同；提示 AT-I 具有抗宫颈癌药物的作用潜力，值得进一步深入研究。

PO_147

Whole Transcriptome Sequencing of Tumor-Educated Lymphatic Endothelial Cells Reveals circ_0000838/hsa-miR-7974/PAPPA axis in Cervical Cancer

Yuzhou Xiao Qiuwen Mai Jiayun Fang Yili Chen Qiaojian Zou Xiaojun Wang Qianrun Chen Mengxun Wei Chudan Chi Aiting Wang Xiaoying Lin Shuyi Wang Zhangqing Peng Abdugheni Karima Qiqiao Du Junxiu Liu* Shuzhong Yao
The First Affiliated Hospital of Sun Yat-sen University

Objective Cervical cancer (CC), one of the most prevalent gynecological malignancies globally, poses a significant threat to women's health. Lymph node metastasis is the leading cause of death in cervical cancer patients. Tumor-educated lymphatic endothelial cells (LECs) are key players in tumor lymph node metastasis, but their function in cervical cancer is unknown. This study will investigate the critical role of tumor-educated LECs in cervical cancer lymph node metastasis and use whole-transcriptome sequencing to uncover the specific molecular mechanisms involved.

Materials and Methods To explore the role of tumor-educated LECs in cervical cancer lymph node metastasis, human CC cell line SiHa and HeLa culture supernatants were added into the culture medium of LECs to establish the CC-educated LECs. CCK8 assays, transwell migration, and invasion experiments were executed to test the proliferation, migration and invasion abilities of CC-educated LECs. Immunofluorescence was employed to reveal the density of tumor-educated lymphatic endothelial cells (LECs) in cervical cancer tissues from patients with lymph node-positive and -negative status. We performed whole-transcriptome sequencing to investigate the underlying molecular alterations in tumor-educated LECs, encompassing circRNAs, lncRNAs, miRNAs, and mRNAs. Both circRNA-miRNA-mRNA and lncRNA-miRNA-mRNA ceRNA axes were screened and validated based on the differentially expressed RNAs.

Results Tumor-educated lymphatic endothelial cells (LECs) exhibit enhanced proliferation and migration capabilities in cervical cancer. Furthermore, increased densities of these tumor-educated LECs were identified in lymph node metastasis-positive cervical cancer tissue samples. Differential expression analysis revealed 5241 long non-coding RNAs (lncRNAs), 3309 messenger RNAs (mRNAs), and 2278 circular RNAs (circRNAs). Functional classification and pathway enrichment analysis of these differentially expressed RNAs (DE-RNAs) implicated several common pathways, including focal adhesion, Wnt signaling pathway, fatty acid degradation, and regulation of the actin cytoskeleton. Subsequent analyses included the construction of potential competing endogenous RNA (ceRNA) regulatory networks, protein-protein interaction (PPI) network analysis, and evaluation of the diagnostic potential of key hub mRNAs in cervical cancer patients. Integrating these findings, the circ_0000838/hsa-miR-7974/PAPPA axis was identified as a key regulatory mechanism within tumor-educated LECs contributing to lymph node metastasis in cervical cancer.

Conclusion Taken together, our findings identify the circ_0000838/hsa-miR-7974/PAPPA axis as a key regulatory mechanism within tumor-educated LECs during cervical cancer lymph node metastasis, revealing novel potential therapeutic targets for cervical cancer (CC) patients.

PO_148

抑制自噬可以增敏铜死亡诱导的宫颈肿瘤的杀伤作用

孙华阳
上海交通大学

研究目的 宫颈癌治疗中，高复发率与化疗耐药性是难题，晚期患者 5 年生存率低，需新治疗策略。铜死亡作为新型细胞死亡方式，在肿瘤治疗中潜力大，但在宫颈肿瘤中作用机制及抵抗机制不明。自噬可能调控铜死亡，但其在宫颈肿瘤铜死亡中的作用及机制待明确。本研究旨在探究自噬对宫颈肿瘤细胞铜死亡敏感性的调控及机制，验证抑制自噬能否增强铜死亡诱导剂的杀伤效果，具体包括明确自噬活性与铜死亡敏感性的关联、揭示调控分子机制、验证联合策略的增敏效应，为宫颈癌联合治疗提供依据。

材料与方法

1.自组装 DNA 纳米材料（DNA 纳米花）的制备
2.琼脂糖凝胶电泳和 PAGE 凝胶电泳
3.电镜以及粒径检测
4.共聚焦成像
5.CCK8 实验

结果 首先通过伊利斯莫联合铜离子这一目前公认的铜死亡诱导剂对宫颈癌细胞进行处理，以诱导宫颈癌细胞发生铜死亡。CCK8 实验表明：联合自噬抑制剂可有效增强铜死亡对宫颈肿瘤的杀伤作用。MDC 法共聚焦成像证实诱导宫颈癌细胞铜死亡可导致胞质内自噬小体增加，提示细胞自噬作用增强。对合成的 DNA 纳米花进行表征，材料的粒径在 300nm 左右，具有良好的生物体递送性能。通过凝胶电泳证实 DNA 纳米花在血清和酶中具有良好的稳定性。

结果

1.抑制自噬可以增强宫颈肿瘤中铜死亡的啥伤作用；
2.诱导细胞铜死亡可增强细胞内自噬水平；
3.纳米花具有良好的生物稳定性能。

PO_149

直肠排空管理策略对宫颈癌体外放疗直肠受照剂量的影响：一项剂量学研究

赵秀娟 何亚男*
重庆大学附属肿瘤医院

目的 探讨直肠排空管理策略对宫颈癌体外放疗中直肠受照剂量的影响，为优化放射治疗流程、降低放射性直肠损伤风险提供依据。
方法 回顾性分析 100 例宫颈癌患者的定位 CT 图像，统计直肠充盈状态；前瞻性选取 11 例患者直肠排空不佳，在排气操作前后分别进行 CT 扫描，比较两组计划的剂量学参数。采用容积弧形调强放射治疗（VMAT）技术，通过等剂量曲线和剂量体积直方图（DVH）评估靶区适形度指数（CI）、均匀性指数（HI）及危及器官（直肠、膀胱等）的受照剂量差异。
结果 46%的患者定位时直肠未排空，其中 37%为直肠含有气体充盈。排气后，靶区体积显著减小（P=0.008），CI 显著改善（P=0.0006）。直肠受量关键指标 V30、V40、V45 及 D~2cc 在排气后分别降低至 81.87±4.45%、50.41±6.19%、26.83±6.67%和 4704±58.18 cGy（均 P<0.05），较排气前降幅达 35.91%~51.18%。其他危及器官（膀胱、肠袋等）受量无显著差异。
结果 直肠排空管理可显著降低宫颈癌 VMAT 治疗中直肠的受照剂量，改善靶区适形度。建议将直肠排空纳入标准化流程，包括治疗前排便、必要时辅助排气，并结合影像监测，以减少放射性直肠损伤风险。

PO_150

基于竞争风险模型的老年性宫颈癌预后分析及个体化诊疗策略的探索

张玮玥 曹佳惠 罗成燕*
南京医科大学第一附属医院

目的 年龄≥ 60 岁的老年性宫颈癌（elderly patients with cervical cancer, EPCC）患者因合并症高发及对治疗的耐受性降低，影响其治疗方案的选择以及对预后的判断。目前尚无针对 EPCC 人群专有的指南与专家共识。因此，本研究旨在探索影响 EPCC 的预后因素，并构建针对 EPCC 患者肿瘤特异性死亡（cancer-specific mortality, CSM）风险的预后模型。
方法 本研究纳入 2010 年 1 月至 2024 年 6 月于本中心经病理确诊为宫颈癌（cervical cancer，CC）的患者。采用竞争风险模型分析、绘制 Nelson-Aalen 累积风险曲线，探究存在非 CC 死亡事件时，因 CC 复发或死亡的累积概率，并使用 Fine-Gray 检验法比较各组之间的差异。利用 Bootstrap 重抽样方法进行内部验证，使用受试者工作特征曲线（receiver operating characteristic, ROC）、校准曲线、决策曲线（decision curve analysis, DCA）评估模型的区分度、校准度及临床应用价值。
结果 本研究纳入 794 例 CC 患者，包括 351 例年龄≥ 60 岁者及 443 例 50~59 岁者。Nelson-Aalen 累积风险分析证实高龄与合并症的存在是 EPCC 患者肿瘤复发或死亡的竞争风险事件。经单因素及多因素竞争风险模型分析发现：FIGO Ⅲ~Ⅳ期是 EPCC 患者 PFS 的独立危险因素，其亚分布风险比（subdistribution hazard ratio，sHR）为 4.79（95%CI: 1.48~15.50, P=0.009），但并不影响 CSS（sHR 6.53, 95%CI: 0.91~46.77, P=0.062）；非鳞癌组织学类型是 EPCC 患者 PFS 和 CSS 的独立危险因素，sHR 分别为 3.56（95%CI: 1.73~7.30）和 6.79（95%CI: 2.54~18.17），P 值均＜0.001。与接受根治性放（化）疗相比较，接受手术±放（化）疗显著降低 EPCC 患者疾病进展及 CSM 的风险，sHR 分别为 0.22（95%CI: 0.10~0.49）和 0.31（95%CI: 0.11~0.92）。纳入组织学类型、接受的治疗

以及 FIGO 分期构建预测 EPCC 患者发生 CSM 的风险模型，并通过内部验证评估模型的准确性和可靠性。经 DCA 分析，发现在各个阈值范围内，该模型均具有的净收益（net benefit，NB）值。
结果 利用组织学类型、治疗类型及 FIGO 分期构建的预测 EPCC 患者 CSM 风险模型，具有较好的区分度和校准度，为个体化制定治疗方案及随访策略提供依据。

PO_151

伴有腹膜后淋巴结转移的宫颈癌是否切除原发肿瘤对预后影响的系统综述及荟萃分析

季佳楠 罗成燕*
南京医科大学第一附属医院

研究目的 伴有腹膜后淋巴结转移(PLNM)且原发部位肿瘤可切除的宫颈癌(UCC)患者的最佳方案的选择及其对预后的影响，目前尚无统一的意见。本研究将探索伴有盆腹腔淋巴结转移的宫颈癌患者接受放（化）疗与接受根治性手术切除与否对预后以及治疗相关不良事件(TRAEs)的影响。
材料与方法 我们系统检索了 PubMed、Embase、Web of Science、Cochrane、ICTRP、ClinicalTrials.gov、中国知网、万方及维普数据库，并根据纳入和排除标准筛选相关文献，荟萃分析接受根治性手术切除与否加或不加术后辅助放（化）疗(RH ± ART ± CHT) 与接受根治性放疗加或不加化疗(RT ± CHT)对 5 年总生存(OS)、无疾病生存(DFS）及≥3 级治疗相关不良事件(TRAEs)的影响。
结果 经筛选后共纳入 20 项研究进行荟萃分析。结果显示，与接受 RT ± CHT 组相比较，采用 RH ± ART ± CHT 作为初始治疗可改善伴有 PLNM 的 UCC 患者的 5 年 OS (HR 0.71，95%CI 0.56 ~ 0.90)及 DFS (HR 0.66，95% CI 0.47 ~ 0.93)。经剔除样本量较小的 4 项研究进行敏感性分析，得到一致的结果。亚组分析显示，原发肿瘤最大径≥4 cm、组织学类型为鳞癌、转移淋巴结局限于盆腔的患者，接受 RH ± ART ± CHT 者的 5 年 OS 优于接受 RT ± CHT 者；原发肿瘤最大径＜4 cm 的患者接受两种治疗后的 5 年 OS 无差异。当转移淋巴结局限于盆腔时，接受 RH ± ART ± CHT 的患者较接受 RT ± CHT 者呈现出更好的 DFS。两组的≥3 级治疗相关不良事件的总体发生率、胃肠道不良事件发生率无显著差异，OR 分别为 0.81 (95%CI 0.36 ~ 1.81)、0.72 (95%CI 0.32 ~1.60)，而血液学不良事件发生率在 RH ± ART ± CHT 组低于 RT ± CHT 组 (OR 0.29，95%CI 0.14 ~ 0.61)。
结论 与接受 RT ± CHT 相比较，伴有 PLMN 且原发部位肿瘤可切除的 UCC 患者接受 RH ± ART ± CHT 可改善 5 年 OS 及 DFS，但两组间的≥3 级 TRAEs 总体发生率无显著差异。

PO_152

Glutamine metabolites promote the progression of cervical cancer by inducing M2 macrophage polarization

zhang lanyue
Renmin Hospital of Wuhan University: Wuhan University Renmin Hospital

Background Tumour-associated macrophages (TAMs) within the tumour microenvironment play crucial roles in tumour initiation, invasion, and metastasis. While glutamine synthetase (GS) is expressed predominantly in the tumour stroma, particularly in TAMs, the role of glutamine metabolism in regulating TAM polarization and function in cervical cancer (CC) remains poorly understood. This study aims to clarify this role and its implications for cancer progression.
Methods CD68 and GS expression in cervical tissues was detected using immunofluorescence staining. The effects of glutamine metabolism on TAM polarization were investigated via RT–qPCR, flow cytometry, Western blotting and NAA treatment analyses. CCK8, colony formation, and Transwell assays were conducted to determine the effects of MSO-treated macrophages on tumour cell proliferation, migration, and invasion.
Results We observed a significant increase in GS expression in TAMs within cervical cancer (CC) tissues, particularly in M2-like TAMs. Glutamine synthesized by TAMs with high GS expression was found to increase the proliferation, migration, and invasion of CC cells. Knockout of GS in TAMs notably reduced their tumour-promoting effects. Additionally, the byproducts of glutamine metabolism in CC cells contributed to the polarization of TAMs towards the M2 phenotype. This polarization was completely abrogated when SNAT1, a key glutamine transporter, was inhibited in CC cells.
Conclusions Our findings demonstrate that glutamine synthesized by TAMs with high GS expression promotes tumour progression in CC. Furthermore, glutamine byproducts produced by CC cells induce TAM polarization towards the M2 phenotype, suggesting crucial metabolic crosstalk between tumour cells and macrophages that supports tumour progression. These results highlight the potential of targeting glutamine metabolism to modulate TAM function and inhibit tumour growth.

PO_153

Matrix stiffness-driven fatty acid oxidation promotes cervical cancer proliferation and migration via the AMPK/ACC pathway

zhang lanyue
Renmin Hospital of Wuhan University: Wuhan University Renmin Hospital

Background Extracellular matrix stiffening is a hallmark of tumor microenvironment remodeling and has been shown to influence malignant tumor progression through multiple mechanisms. This study aimed to investigate the alterations in ECM stiffness within cervical cancer tissues and to elucidate its specific role and underlying molecular mechanisms in regulating lipid metabolic reprogramming in cervical cancer cells.
Methods The stiffness of cervical tissues was assessed using atomic force microscopy. Immunohistochemistry and Sirius Red staining were used to detect the expression of Collagen I, Fibronectin, and collagen content. HeLa and SiHa cells were cultured on 2D polyacrylamide and 3D fibrin gel models with varying stiffness. Cell proliferation and migration were evaluated using EdU, qRT-PCR, and Transwell assays. CPT1A expression was analyzed by qRT-PCR and western blot. Lipid droplet accumulation and ATP levels were measured using BODIPY 493/503 staining and an ATP assay kit. Western blot was also used to assess p-AMPK, AMPK, p-ACC, and ACC expression. A

subcutaneous xenograft model in nude mice was established to examine the effect of matrix stiffness on tumor growth.
Results Cervical cancer tissues showed increased stiffness and higher levels of Collagen I, Fibronectin, and collagen fibers compared to normal tissues. The stiff matrix enhanced fatty acid oxidation (FAO), thereby promoting HeLa and SiHa cell proliferation and migration. Mechanistically, matrix stiffness upregulated CPT1A expression and activated FAO in HeLa and SiHa cells through stimulation of the AMPK/ACC signaling pathway. In vivo, reducing matrix stiffness suppressed tumor growth.
Conclusion The Young's modulus of cervical cancer tissues was significantly higher than that of normal cervical tissues. The stiff matrix promoted the proliferation and migration of cervical cancer cells by upregulating CPT1A expression and enhancing FAO through activation of the AMPK/ACC signaling pathway.

PO_154

Mechanosensitive ion Piezo1 channel promotes cellular glycolysis through Ca2+/GLUT3 to drive cell proliferation and metastasis in cervical cancer

zhang lanyue
Renmin Hospital of Wuhan University: Wuhan University Renmin Hospital

Background Glycolysis is a major metabolic reprogramming of cervical cancer, often facilitating tumor proliferation and metastasis. The mechanosensitive ion channel Piezo1 has been linked to glycolysis, but its specific role in regulating the progress of cervical cancer through glycolysis is not fully understood. Consequently, our objective is to investigate the molecular mechanisms through which Piezo1 influences the proliferation and metastasis of cervical cancer by modulating glycolysis.
Methods Our research initially investigated the expression, prognosis, and cancer-related signaling pathways of Piezo1 in cervical cancer using IHC staining, western blot, qRT-PCR, and bioinformatic tools. Subsequently, we employed the CCK-8 assay, colony formation assay, flow cytometry analysis, western blot, and transwell assay to assess the proliferation and migration of cervical cancer cells upon Piezo1 gene silencing or activation with the Piezo1 selective agonist Yoda1. Additionally, we examined the glycolytic phenotypes of tumor cells when Piezo1 was knockout and stimulated by Yoda1. And teated the cells by 2-Deoxy-D-glucose (2-DG) and lactose to inhibit glycolysis in the Yoda1 groups, in order to confirm that Piezo1 affected the proliferation and migration capabilities of cervical cancer cells through glycolysis. Furthermore, calcium fluorescence probes, western blot and immunofluorescence experiments were employed to demonstrate that Piezo1 can regulate GLUT3 expression levels through Ca2+ signaling. Rescue experiments were then conducted to further validate the specific molecular mechanisms. Finally, xenograft animal models were established to confirm the pivotal roles of Piezo1 in cervical cancer in vivo.
Results Our results demonstrated that Piezo1 mRNA and protein levels exhibited a significant increase in cervical cancer tissues and cells. Elevated Piezo1 expression was associated with a notably shorter overall survival and was linked to cancer-related pathways, inculding hallmarks of apoptosis, epithelial mesenchymal transition and hypoxia pathways in cervical cancer patients. Additionally, Piezo1 was found to stimulate proliferation, migration and glycolysis of cervical cancer cells, enhanced glycolysis potentially lead to more aggressive behaviors. Mechanically, Piezo1 was observed to promote cellular glycolysis through increasing GLUT3 expressoin levels through Ca2+, thereby driving cell proliferation and metastasis in cervical cancer cells. Furthermore, xenograft models confirmed that Piezo1 promotes tumor formation in vivo.
Conclusion

Our mechanism research of Piezo1 indicated that Piezo1 channel may promote cellular glycolysis via GLUT3 to drive cell proliferation and metastasis in cervical cancer, which offers some theoretical support for potential Piezo1-targeted therapeutic interventions in the future.

PO_155

Comparative Survival Outcomes of Total Hysterectomy versus Radical Hysterectomy in Gastric-Type Endocervical Adenocarcinoma: A SEER Database Analysis

Yanjun Ge[1] Xintao Wang[1] Yuchen Zhang[1] Luzan Ma[1] Wei Jin[2] Haiyan Zhu[1] Dewen Yan[3]
1.Shanghai First Maternity and Infant Hospital 2.Huai'an Fifth People's Hospital
3.Taizhou First People's Hospital

Objective This study aims to assess the efficacy of total hysterectomy versus radical hysterectomy in the treatment of gastric-type endocervical adenocarcinoma.
Methods Patients diagnosed with gastric-type endocervical adenocarcinoma from 2001 to 2021 were derived from the Surveillance, Epidemiology, and End Results database (SEER). Clinicodemographic characteristics, tumor clinicopathological features, and survival outcomes (including vital status and duration of follow-up) of these patients were collected. Kaplan-Meier survival curves were generated to estimate survival probabilities, with between-group differences assessed using log-rank tests for statistical significance.
Results 114 patients undergoing total hysterectomy and 129 patients undergoing radical hysterectomy were included for survival analysis. No statistically significant differences in overall survival (OS) were observed between surgical approaches, either in the entire cohort (p=0.0594) or the FIGO stage I-IIA subcohort (p=0.0777). Adjuvant therapy (radiotherapy and/or chemotherapy) was administered to 52.6% (60/114) of patients in the total hysterectomy group and 42.6% (55/129) in the radical hysterectomy group. However, there was no statistically significant difference in OS between the total hysterectomy and radical hysterectomy groups among patients who received adjuvant therapy and similar results were also found in the subgroup of patients with FIGO stage Ⅰ-ⅡA disease. Notably, adjuvant therapies failed to demonstrate significant OS benefits in either surgical group for stage I-IIA disease.
Conclusion Total hysterectomy demonstrated comparable survival outcomes to radical hysterectomy in gastric-type endocervical adenocarcinoma (GEA) patients. These findings suggest that less extensive surgery may represent a viable treatment alternative for early-stage disease.

PO_156

CCNE1 and Ovarian Cancer: Insights into Function and Therapeutic Strategies

Ying Yang
Department of Gynecology and Obstetrics, West China Second University Hospital, Sichuan University, Chengdu 610041, People's Republic of China

Purpose CCNE1 amplification or cyclin E1 overexpression, prevalent in ovarian cancer, has been identified as an early molecular event in the pathogenesis of ovarian cancer and is associated with replication stress, DNA damage, and genomic instability. The existing excellent reviews on CCNE1 contain the basic functions and well-known therapies, such as cyclin-dependent kinase (CDK) 2 inhibitors, whereas new directions and evidence have been provided in recent studies. Therefore, this review will focus on the advances of CCNE1 amplification in emerging fields such as cell cycle checkpoints, DNA damage and repair, RS, tumor immune microenvironment, and metabolism, and comprehensively summarize the therapeutic strategy targeting CCNE1 amplification to guide clinical practice.

Method A systematic literature search covered PubMed, EMBASE, and Cochrane Library (2010–2023) using terms: ('CCNE1' OR 'cyclin E1') AND ('ovarian cancer') AND ('biomarker' OR 'therapy'). Evidence was stratified by study type (clinical > preclinical) and assessed via OCEBM.

Results The CCNE1-amplified subset prominently features in HR-proficient HGSOC and stands as a pivotal biomarker with multiple roles. It acts as a predictive marker for early detection, serves as a negative biomarker indicating intrinsic resistance to PARPi and platinum-based therapy, and functions as a sensitive biomarker for DDR-related inhibitors. However, there are challenges in the precise identification of CCNE1 status due to inconsistent expression levels of CCNE1-amplified status, CCNE1 mRNA, and protein across tumors, and these inconsistencies further impact therapeutic response and prognosis prediction. The current deficiency in robust detection methods for CCNE1 status underscores the need for more comprehensive research to support the varied roles CCNE1 plays in HGSOC management.Furthermore, in the realm of tumor research, immunity and metabolism have emerged as central themes. The interaction between oncogene mutations, including those involving CCNE1, and various immune components has been noted, but the specific mechanisms through which CCNE1-amplified tumors uniquely alter the TIME require deeper investigation. A possible explanation involves RS-related DNA damage-induced changes in immune signaling and immune cell activity, potentially leading to immune evasion. The profound studies of these two highlights are essential to rapidly advance the field of diagnosis and treatment in tumors with CCNE1 amplification or overexpression. Addressing the limited therapeutic options available for targeting CCNE1, given its unsuitability for direct targeting due to kinase characteristics, remains a significant concern. Although preclinical studies with small molecule inhibitors targeting CCNE1 indirectly have shown promise, translating these findings into clinical benefits has been challenging. This discrepancy in the efficiency of preclinical models and clinical trials underscores the complex biology of tumors and highlights the need for further research focused on patients with clearly defined CCNE1 status. For instance, CHK1i SRA737 effectively increased CCNE1-amplified cell apoptosis but showed no related clinical benefit. Such studies should prioritize CCNE1 as a selective biomarker for patient inclusion and strive for consistency in defining CCNE1 amplification to facilitate comprehensive analysis. In the quest to overcome drug resistance, exploring the role of CCNE1 targeting is crucial. Known mechanisms include HR-proficient status and an increased presence of PGCCs, but additional mechanisms warrant further exploration. A strategic approach combining ICB and DDR-targeted agents might provide a viable pathway to circumvent resistance mechanisms without introducing overlapping drug toxicities. Looking ahead, the pursuit of new DDR targets such as Aurora-A, Polθ, RAD51, PARG, and WRN, and innovative therapeutic strategies involving co-amplified passenger genes, represents a promising direction. For instance, the sensitivity observed in the DDX1-MYCN co-amplified group compared to the MYCN amplified group highlights the therapeutic potential of targeting co-amplified genes through collateral lethality.

Conclusions In summary, given CCNE1's critical involvement in tumorigenesis and progression, focusing on CCNE1 as a therapeutic target could mark a significant leap forward in the precision treatment of HGSOC.

PO_157

高强度聚焦超声治疗卵巢癌 1 例短篇病例报道

官孝萍* 黄小琴 邓雪
内江市第一人民医院

研究目的 探讨高强度聚焦超声治疗卵巢癌的临床研究。

资料与方法

1.一般资料 患者，女，56 岁，因“咳嗽 3 天”入院，2019 年患者诊断肺腺癌 IV 期。2021 年 01 月复查病情进展，予以 6 周期化疗。2023.04.25 予以化疗，定期复查肿瘤评估稳定。入院前 3 天，患者出现咳嗽不适，入院完善检查，盆腔 MRI 平扫+增强检查意见:盆腔偏右侧囊实性占位，考虑卵巢来源恶性肿瘤可能大。彩超经阴道子宫及附件检查（含超声报告费）检查意见:盆腔囊实性占位：O-RADS5 类,（盆腔包块穿刺组织）查见多量坏死组织及增生的腺体组织，结合现有免疫组化结果，支持卵巢上皮源性肿瘤，形态学符合子宫内膜样肿瘤。

2.方法 在镇静镇痛下行卵巢恶性肿瘤高强度聚焦超声治疗。患者俯卧位，超声定位后注射镇静镇痛药。镇静镇痛起效后予 9 号探头，予平均 400W 功率超声治疗，治疗总时间 1500s，治疗总能量 600000J。超声见：团块内组织灰化明显。术毕。术后患者神清，四肢活动如常。

结果 治疗前卵巢病灶大小 117.52*96.81mm，治疗后 50 天卵巢病灶大小 88.68*92.16mm，治疗后 102 天卵巢病灶大小 57.99*75.50mm。

讨论 在陈娟等的随机对照实验中，HIFU 联合根治性放疗、化疗，有助于控制宫颈癌进展，延长患者生存期。HIFU 联合介入栓塞化疗较单纯介入栓塞化疗更为有效地抑制晚期宫颈癌病灶生长及细胞侵袭。HIFU 治疗裸鼠卵巢癌皮下移植瘤，治疗区的肿瘤组织出现完全凝固性坏死，肿瘤内小血管和毛细血管被破坏，加速了肿瘤组织的缺血性坏死。经 HIFU 治疗后，卵巢癌大鼠瘤体平均重量降低，抑瘤率超过 50%;大鼠体内 CD4+ 细胞明显增加，且 CD4+ /CD8+ 细胞比值>1，提示 HIFU 治疗能改善大鼠的免疫功能。HIFU 可破坏子宫内膜癌肿瘤细胞及肿瘤血管成分包括弹性纤维、内皮细胞等，超声消融后肿瘤血管影消失，肿瘤周围和内部血流信号消失或明显减少，为子宫内膜癌的靶向治疗和抗血管生成策略提供了一种新的途径。1 例子宫内膜癌淋巴结转移患者经手术、化疗、放疗等多种治疗方案病情未控的情况下，应用 HIFU 治疗 3 次，右腹股沟淋巴结明显缩小，B 超显示血流信号减少，患者病情稳定达 1 年，为临床治疗子宫内膜癌提供了新的选择。

总结 HIFU 治疗肿瘤既有直接作用，又对放疗和化疗具有辅助增效作用，作为肿瘤综合治疗的一种重要手段，与手术、化疗、放疗等联合应用，已取得很好的临床效果，随着研究的深入及临床运用的推广，HIFU 治疗在宫颈癌、卵巢癌等女性生殖器恶性肿瘤治疗领域将会有更加广阔的前景。

PO_158

The Role of TP53 Mutations in the Malignant Progression of Mucinous Borderline Ovarian Tumors: A Case Report and Literature Review

Meirong Liang Shilei Xu Xuqing Deng
Jiangxishengfuyoubaojianyuan

Background Mucinous ovarian carcinoma (MOC) is characterized by aggressive behavior and limited responsiveness to standard chemotherapeutic regimens. The infrequent occurrence of MOC arising from mucinous borderline ovarian tumors (MBOTs) and the lack of readily identifiable diagnostic markers pose significant challenges to the development of effective treatment strategies. This report presents a case of a 26-year-old woman with MBOT progression to metastatic MOC despite aggressive surgical intervention and adjuvant chemotherapy, aiming to identify potential diagnostic and therapeutic improvements.
Results A review of the literature revealed fewer than 30 comparable cases, underscoring the absence of established guidelines for diagnosing and treating the progression of MBOT to invasive MOC, particularly with respect to the role of TP53 mutations. In our patient's case, we observed some changes in immunohistochemical marker expression between the MBOT and the subsequent invasive MOC, including altered p53 expression. Although these changes, considered in the context of existing literature, hint at a complex transformation process potentially involving genetic and epigenetic alterations, including TP53 mutations, further detailed genomic or transcriptomic analyses would be required to confirm this in our specific case. The diagnostic process was further complicated by clinical and pathological similarities with gastrointestinal malignancies. Ultimately, disease progression necessitated adjustments to the treatment plan, despite the use of a multi-agent chemotherapy regimen consisting of paclitaxel, carboplatin, and bevacizumab.
Conclusion The transition from MBOT to MOC is characterized by intricate genetic and epigenetic alterations, especially involving TP53 mutations. This progression presents considerable diagnostic challenges due to similarities with gastrointestinal cancers. Therefore, given the strong correlation between TP53 mutations and chemoresistance, there is a pressing need to develop targeted therapies and enhance diagnostic strategies.

PO_159

马里苷靶向 p38α 增强卵巢癌对 PARP 抑制剂敏感性的机制研究

王芳 曹玉敏 陈友国 周金华
苏州大学附属第一医院

研究目的 卵巢癌是致死率最高的妇科肿瘤，大部分患者会复发并发展为难治性卵巢癌。PARP 抑制剂是目前卵巢癌常用的维持治疗方案，但五年生存率始终徘徊在 30%左右，临床治疗效果无法突破。先前已有研究报道部分天然药物可以发挥抗肿瘤作用，马里苷是两色金鸡菊中提取的黄酮类活性成分，但在卵巢癌中的作用尚未知。本项目拟在体内外从基因、蛋白、细胞、类器官、动物组织和临床标本等多层次实验水平阐明马里苷对 DNA 同源重组修复、PARP 抑制剂敏感性的调控作用及机制，评估马里苷和 PARP 抑制联合治疗卵巢癌的效果及安全性，为卵巢癌治疗提供一种新的可能的策略。
研究方法 通过计算机模拟预测马里苷可能的作用靶点；通过免疫印迹实验、免疫荧光实验、中性彗星拖尾实验以及同源重组修复报告基因实验，检测联合使用马里苷对 DNA 损伤及其修复途径的影响；采用 CCK8 法和克隆集落形成实验评估卵巢癌细胞中马里苷对 PARP 抑制剂敏感性的影响；通过计算机

模拟分子对接和化合物结构改造，细胞热转移实验、药物亲和反应的靶点稳定性实验验证马里苷与 p38α 的结合方式及其对 p38α 磷酸化水平的影响；收集肿瘤患者组织，构建类器官模型，进行药敏检测，进一步评估马里苷对 PARP 抑制剂的增敏效应。

研究结果 本课题发现： 1）模拟预测分析 p38α 可能是马里苷的靶点；2）马里苷可显著增加卵巢癌细胞的 DNA 双链损伤，表现为 γH2AX 的表达和免疫荧光强度增加，彗星拖尾距离增加，并且同源重组修复效率显著降低；3）马里苷可增加卵巢癌细胞和卵巢癌类器官对 PARP 抑制剂的敏感性；4）马里苷能够与 p38α 的 MET109 和 LYS53 两个关键氨基酸残基结合，且此结合可显著降低 p-p38α 及其下游 CtIP 和 RAD51 等同源重组修复因子的表达；将马里苷结合位点乙酰化改造后可显著降低其与 p38α 的结合，且改造后的马里苷对 PARP 抑制剂的增敏作用被逆转。

研究结果 马里苷可通过与 p38α 相结合，抑制其磷酸化水平，进而降低同源重组修复效率，增加 PARP 抑制剂的敏感性，从而为卵巢癌治疗提供新的理论依据。

PO_160

ADAMTS2 mediates epithelial-mesenchymal transition and inflammation in high-grade serous ovarian cancer: A study based on bioinformatic analyses and experiments

Yunjie Tian Shan Kang*
The Fourth Hospital of Hebei Medical University

Background The importance of epithelial-mesenchymal transition (EMT) in tumour invasion and metastasis of high-grade serous ovarian cancer (HGSOC) has been highlighted in numerous studies, but genetic biomarkers in predicting EMT of HGSOC are still lacking.

Methods The role of EMT hallmarks and the relationship between EMT and tumour microenvironment in HGSOC were examined by GSVA algorithm, ESTIMATE method and Pearson correlation analyses. Furthermore, machine learning was performed to determine key EMT signature to classify EMT subtypes. Meanwhile, the role of key EMT signature ADAMTS2 in the behaviours and EMT process of HGSOC cells were detected via western blot, CCK8, transwell, wound healing and immunofluorescent assays.

Results The expressions of EMT hallmarks (EMT score) were significantly associated with progression, immune microenvironment and prognosis of HGSOC patients. Different machine learning algorithms identified MMP2, ADAMTS2, FN1, THBS2, C3orf80, FAP and POSTN as key EMT-related signature in HGSOC. Lastly, qPCR, western blotting and IHC staining consistently showed an elevated expression of ADAMTS2 in cancer tissues. Further in vitro experiments showed that knock-down of ADAMTS2 inhibited cell proliferation, migration, invasion and the expressions of TNF-α, IL-1β and EMT markers (E-cadherin, N-cadherin, Slug, Twist1), while over-expression of ADAMTS2 promoted above cellular behaviours and enhanced the expressions of TNF-α, IL-1β and EMT markers.

Conclusion: Our study developed a new classifier to predict EMT and immune microenvironment in HGSOC and identified ADAMTS2 as a novel regulator in EMT process. Those findings might promote the development of EMT-related immunotherapeutic strategy for HGSOC patients.

PO_161

BRD4-Associated Chromatin Remodeling Signature Links Epigenetic Regulation to Immune Landscape in Ovarian Cancer

Zidan Lin Shanyang He*
Guangdong Provincial People's Hospital (Guangdong Academy of Medical Sciences), Southern Medical University

Purpose Chromatin remodeling-related genes (CRRGs) are crucial epigenetic regulators of gene expression and tumor progression. However, their impact on the immune microenvironment and clinical outcomes in ovarian cancer (OV) remains unclear. This study aimed to construct a CRRG-based prognostic signature and explore its relationship with immune cell infiltration, focusing on plasmacytoid dendritic cells (pDCs).
Methods Transcriptomic and clinical data from TCGA (n = 228) and ICGC (n = 111) OV cohorts were analyzed. Prognostic CRRGs were identified through differential expression and survival analysis, followed by LASSO and multivariate Cox regression to build a prognostic model. Immune infiltration was assessed using ssGSEA and validated via multiplex immunofluorescence (mIF). Associations between BRD4 expression, pDC infiltration, and chemokine expression were explored using public databases and OV tissue samples.
Results An 11-gene CRRG-based immune-associated signature stratified patients into high- and low-risk groups with significantly different overall survival across both cohorts. The risk score remained an independent prognostic factor. High-risk patients exhibited reduced pDC infiltration and lower activation of antigen presentation-related pathways. BRD4 emerged as a key gene inversely correlated with pDC levels and associated with poor prognosis. Elevated BRD4 expression was linked to reduced levels of multiple pDC-attracting chemokines. mIF confirmed the negative association between BRD4 and $BDCA2^{+}$ pDC infiltration in OV tissues.
Conclusion This study presents a novel CRRG-based prognostic signature in OV and identifies BRD4 as a potential suppressor of pDC recruitment via chemokine downregulation, suggesting a link between epigenetic dysregulation and immune exclusion in OV.

PO_162

Effects of neoadjuvant chemotherapy on prognosis and platinum resistance of advanced ovarian cancer

Weishan Li Danbo Wang* Zhuo Yang
Liaoning Cancer Hospital and Institute

Objective Primary Debulking Surgery (PDS) combined with platinum-based chemotherapy is the standard therapy for ovarian cancer. Some trials have suggested that neoadjuvant chemotherapy-Intermittent Debulking Surgery(NACT-IDS) can achieve equivalent efficacy in the chemotherapy of combining PDS, without influencing OS. This study aimed to explore the effects of NACT-IDS on the prognosis and platinum resistance of advanced ovarian cancer, so as to provide a certain basis for the selection of suitable clinical therapies.
Methods The patients with advanced ovarian cancer were selected from January 1, 2014 to January 1, 2017. The patients were assigned to NACT-IDS group or PDS group after evaluation by gynecological oncologists. We analysed the clinical data and collected the follow-up data of cases over 5 years.
Results Totally 173 patients were enrolled into the study, including 52 cases in NACT-IDS group and 121 cases in PDS group.The platinum-resistant relapse rate in NACT-IDS group was evidently greater than that in PDS group (22.9% vs 4.5%, $P<0.001$), and the multi-factor analysis results also proved

that NACT (OR=7.822, 95%CI 2.121-28.855) was an independent risk factor for platinum-resistant relapse of ovarian cancer. No significant differences in PFS, RS and OS were found between two groups.
Conclusion NACT-IDS may increase the risk of platinum resistance, and it is not advisable to expand its indications blindly. During the IDS procedure, the surgical standard should aim for a more thorough tumor bed resection, achieving an R0 resection, in order to avoid the increased risk of recurrence. Precise selection of indications and comprehensive precision management throughout the treatment course are essential, as neoadjuvant chemotherapy does not necessarily affect prognosis.

PO_163

卵巢交界性肿瘤术后复发风险预测模型的构建及临床预后研究

叶秋霖 齐跃 刘娟娟 李潇 张丹晔 胡悦欣 刘鸥萱 林蓓*
中国医科大学附属盛京医院

研究目的 卵巢交界性肿瘤（borderline ovarian tumor，BOT）属于低度恶性潜能肿瘤，且部分患者有生育要求。该病复发率在 5%-20%之间，能够识别高复发概率人群是提高 BOT 患者治疗效果、改善预后关键。因此，本研究从真实世界的角度，分析 BOT 的临床结局，结合人工智能技术构建复发预测模型，促进 BOT 患者的精准管理，降低复发风险。
材料与方法 收集中国医科大学附属盛京医院于 2014 年 1 月-2023 年 8 月收治的经病理诊断为 BOT 患者的数据，对临床预后进行分析。根据初始手术术后是否复发，分为未复发组及复发组，利用合成少数类过采样技术（synthetic minority over-sampling technique，SMOTE）算法对原始数据进行扩增构建平衡数据集，采用随机抽样的方法将 70% 样本作为训练集，剩余 30% 样本作为验证集。使用训练集数据分别构建 Cox 回归模型、基于神经网络的多任务逻辑回归（neural multi-task logistic regression，N-MTLR）模型。验证集分别评价并比较两个模型的灵敏度、特异度、阳性预测值（positive predictive value，PPV）、阴性预测值（negative predictive value，NPV）和准确性，通过时间依赖的受试者工作特征曲线下面积（time-dependent area under the receiver operating characteristic curve，tdAUC）评价模型的区分度，综合 Brier 分数评价模型的拟合优度。
结果 本研究共纳入 660 例患者，其中复发组 76 例，未复发组 584 例，复发率 11.52%。平衡数据集中未复发人数 584 人，复发人数 584 人，共 1168 人。训练集进行 Cox 回归分析后，得到 BOT 复发的 7 个独立危险因素。验证集评价模型的灵敏度 0.79，特异度 0.69，PPV0.71，NPV0.78，准确率 0.74，AUC 值：2 年 0.78，4 年 0.78，7 年 0.75。综合 Brier 评分：2 年 0.10，4 年 0.09，7 年 0.12。将训练集里的 34 个特征进行相关性筛选后构建 N-MTLR 模型，共筛选出 9 个变量。验证集评价模型的灵敏度 0.97，特异度 0.62，PPV0.76，NPV0.94，准确率 0.81，AUC 值：2 年 0.80，4 年 0.80，7 年 0.77。综合 Brier 评分：2 年 0.10，4 年 0.09，7 年 0.15。
结论 BOT 患者复发大多仍为 BOT，N-MTLR 模型与传统 Cox 回归预测模型相比，具有拟合非线性的能力，预测 BOT 术后复发的性能更优秀，能够为临床决策提供更科学的风险分层和个体化预测。

PO_164

卵巢 Sertoli-Leydig 细胞瘤的临床特征及治疗探讨

魏佳慧 吴玉梅*
首都医科大学附属北京妇产医院/北京妇幼保健院

研究目的 探讨卵巢 Sertoli-leydig 细胞瘤的发病年龄、临床表现、影像学特点、治疗方式、病理特征等，从而为其临床诊疗提供指导。

材料与方法 回顾性分析了首都医科大学附属北京妇产医院接受手术治疗并经组织病理学诊断证实为卵巢 SLCTs 的患者 15 例，搜集其完整临床资料，包括：一般情况、症状体征、影像学表现、手术方式、病理特征等。组织标本经 10%福尔马林固定，石蜡包埋，经切片、苏木精-伊红（HE）染色，由 2 名高年资专家分别阅片，部分病例行免疫组化分析。

结果 ①发病年龄及临床特点：本研究纳入的 15 例 SLCTs 患者，11 例为育龄期女性，发病年龄为 24-38 岁，平均年龄为 30 岁；4 例为绝经期女性，平均发病年龄 54 岁。6 例患者表现为月经周期延长，月经稀发；4 例患者表现为闭经，其中 1 例患者男性化特征明显（面部痤疮、体毛重、声线粗）；3 例患者表现为阴道不规则出血，其中 2 例为绝经女性；1 例绝经女性体检发现，另 1 例绝经女性因盆腔巨大肿物，排尿排便困难来诊。②影像学特点及激素水平：15 例 SLCTs 患者超声或盆腔核磁结果均提示一侧附件区囊实性肿物，局部血运较丰富。15 例患者中 8 例患者表现为睾酮水平增高。③治疗方式：15 例 SLCTs 患者中 13 例选择腹腔镜手术，2 例开腹手术。8 例患者行患侧附件切除，4 例患者行患侧囊肿剥除，2 例患者行全子宫+双附件切除，1 例患者行卵巢肿瘤细胞减灭术。④术中肉眼检查**结果** 15 例手术切除标本，肿瘤表面光滑，有包膜，实性，肿瘤切面灰白、淡黄，或灰白灰黄相间，局部有出血。⑤病理特征：其中高分化患者 2 例，中分化患者 8 例，低分化患者 5 例，行免疫组化后 α-抑制素、钙视网膜蛋白、波形蛋白阳性。

结论 SLCTs 好发于年轻女性，临床表现多样，多为 I 期患者，单侧发生的实性或囊实性肿块，伴高雄激素血症和高血供特征有助该病的诊断，预后与分化程度、临床分期有关，目前 SLCTs 发病率较低，易产生误诊，有待进一步总结及研究，可进一步完善基因检测辅助诊断，为规范临床诊治提供依据。

PO_165

Microbial metabolite 5-formamidoimidazole-4-carboxamide ribotide targets METTL1 to inhibit m7G modification of BRCA1 mRNA to inhibit high-grade serous ovarian cancer

Sili He*
Third Xiangya Hospital of Central South University

Objective This study explored the impact of vaginal microbes, metabolites, and METTL1-mediated m7G modification of BRCA1 mRNA on High-Grade Serous Ovarian Cancer (HGSOC).
Methods METTL1 and BRCA1 expression levels were assessed via bioinformatics, Western blotting, and RT-qPCR. Their interaction was studied using RNA co-immunoprecipitation and RNA pull-down assays. The functions and mechanisms of METTL1 and BRCA1 in HGSOC were investigated through CCK-8 assays, flow cytometry, Transwell migration assays, and nude mouse xenograft models. We analyzed vaginal microbial and metabolite differences in HGSOC patients with varying BRCA1 expression using 16S rDNA sequencing and liquid chromatography. Associations were evaluated with Spearman correlation and heat maps, while molecular docking assessed key metabolite binding to

METTL1. The roles and interactions of selected metabolites with METTL1/BRCA1 in HGSOC were validated through in vivo and in vitro experiments.
Results In HGSOC, both METTL1 and BRCA1 were up-regulated. METTL1 enhanced BRCA1 expression via m7G modification, boosting cell proliferation and tumor growth. Elevated BRCA1 levels were associated with changes in vaginal microbiota, particularly increased Lactobacillus, and alterations in metabolic pathways. Correlation analysis indicated that Lactobacillus was significantly negatively correlated with 5-formamidoimidazole-4-carboxamide ribotide, inosine, cobalt-precorrin-7, and uridine, but positively correlated with L-lysine. The strongest correlation was with 5-formamidoimidazole-4-carboxamide ribotide. Molecular docking showed that this compound binds strongly to METTL1. Functional tests demonstrated that it inhibits HGSOC cell proliferation and tumor growth by disrupting METTL1-mediated m7G modification of BRCA1. Overexpression of METTL1 or BRCA1 negated its anti-tumor effects.
Conclusion The vaginal microbial metabolite 5-formamidoimidazole-4-carboxamide ribotide reduces BRCA1 expression and slows HGSOC progression by modifying BRCA1 m7G through METTL1, suggesting its potential as an HGSOC treatment.

PO_166

绝经后卵巢良性肿瘤患者合并子宫内膜病变危险因素分析及预测模型构建

王天琪 王颖梅* 田丽娜
天津医科大学总医院

目的 绝经后卵巢良性肿瘤（Ovarian Benign Tumor，OBT）目前推荐行双侧输卵管卵巢切除术，子宫是否需要同时切除存在争议。本研究拟分析绝经后 OBT 患者合并子宫内膜病变危险因素，构建术前预测模型为患者手术范围选择提供参考。
方法 收集 2018 年 1 月至 2024 年 12 月天津医科大学总医院妇产科住院收治的 471 例绝经后 OBT 患者，分析患者合并子宫内膜病变危险因素
并进行模型构建与验证。
结果 初潮年龄＜14 岁、超声下内膜厚度＞4mm、超声下为纯实性肿物、E2≥10pg/mL 被纳入术前预测模型，预测模型总分值 5.627 分，评分≥3.063 分认为合并子宫内膜病变风险高，建议行全子宫及双附件切除术。
结论 本研究构建的绝经后 OBT 患者合并子宫内膜病变临床预测模型具有良好的预测效果，可在术前对绝经后 OBT 患者合并子宫内膜病变风险进行预测，为绝经后 OBT 患者诊治提供参考。

PO_167

miR-3154 靶向 PXN 基因抑制 EMT 抑制卵巢癌细胞的侵袭转移

吴巧铃 孙阳*
福建省肿瘤医院

背景 卵巢癌是死亡率最高的妇科恶性肿瘤，因其临床表现隐匿，多数患者确诊时已是晚期，经过标准的分期手术治疗及术后化疗，晚期患者仍极易复发，严重危害女性健康。目前研究已发现卵巢癌的高侵袭性是其易晚期复发的重要原因之一。我们既往的研究发现 PXN 基因在卵巢癌发生发展中起重要作用，尤其增强卵巢癌细胞的高侵袭性，但具体分子机制仍不清楚。
方法 基于 R 语言及多种算法，联合 Targetscan 、Tarbase、miRWalk、Target gene、 TCGA 以及 GEO 等多个生物信息学数据库说明 miR-3154 作为 PXN 上游的可能性及其参与的生物过程。同时利用 cibrtsort 算法评估 PXN 与免疫细胞浸润的相关性，利用 GeneMANIA 评估 PXN 基因互作网络。最后利用 western-blot 探索 PXN 影响卵巢癌细胞的侵袭性的分子机制。
结果 miR-3154 影响卵巢癌患者的预后（P=0.0093），其低表达与患者不良预后相关。PXN 基因与 T 细胞滤泡辅助细胞及树突状细胞活化成负相关，PXN 基因与中性粒细胞、M2 巨噬细胞以及肥大细胞休眠成正相关（P<0.05）。PXN 与 PARVA、 PTK2、VCL、ILK 等基因的相关性高，且作为 miR-3154 的靶基因调控 EMT 相关蛋白的表达（P<0.05），敲减 PXN 基因能够抑制 N-cadherin 蛋白及 Vimentin 蛋白的表达，能够增加 E-cadherin 蛋白的表达。
结论 miR-3154 是卵巢癌患者的预后影响因素，可能通过靶向 PXN 影响肿瘤微环境中免疫细胞浸润和抑制 EMT 进而抑制卵巢癌细胞的侵袭转移。

PO_168

Immune Subtyping of High-Grade Serous Ovarian Cancer Based on Transcriptomics and Immunohistochemistry

Yuting Zhao[1,2] Jing Yu[1] Lingying Wu[1*]
1.National Cancer Center/National Clinical Research Center for Cancer /Cancer Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College
2.Chinese Academy of Medical Sciences & Peking Union Medical College

Objective The overall response rate of ovarian cancer to immunotherapy remains low, highlighting the urgent need to develop more precise and individualized immunotherapeutic strategies. This study aimed to construct and validate an immune molecular subtyping system for high-grade serous ovarian cancer (HGSOC), and to identify its key biomarkers, thereby providing theoretical support and practical tools for the clinical application of immune checkpoint inhibitors (ICIs) in HGSOC.
Materials and Methods Transcriptome data of 404 HGSOC patients were obtained from The Cancer Genome Atlas (TCGA), and 1,480 immune-related genes were integrated from the TCIA, MCP-counter, and ImmPort databases. Consensus clustering (k = 3) was used to identify three immune subtypes. Tumor microenvironment characteristics of each subtype were evaluated using xCell, ssGSEA, and ESTIMATE algorithms at the transcriptomic level. Subtype-specific biomarkers were further screened from differentially expressed genes and validated by logistic regression and area under the receiver operating characteristic curve (AUROC) analysis. External validation was performed using RNA-seq and clinical data from 205 HGSOC patients across 8 datasets from the GEO database. Surgical specimens were collected from HGSOC patients at the National Cancer Center who received neoadjuvant therapy with paclitaxel, carboplatin, and tislelizumab (NCC cohort). Whole-transcriptome microarray analysis and immunohistochemistry (IHC) staining for biomarkers and immune

microenvironment markers (TILs, CD8, CD20, CD68, and PD-1) were performed. Staining intensity was quantified using the ImageJ software. ICI response was evaluated based on imaging and pathological assessment at the time of interval debulking surgery. The NCC cohort was classified using the 1,480-gene signature (mRNA-based subtyping) and compared with IHC-based immune infiltration levels using Cohen' s Kappa statistics. Mean staining intensity of CXCL9 and DLK1 was analyzed to determine optimal IHC positivity cutoffs using the Youden index. Subsequently, the IHC-based classification defined by CXCL9 and DLK1 was evaluated for its consistency with ICI response using Cohen' s Kappa, thereby validating the clinical applicability of the immune subtyping model and its biomarkers.

Results The TCGA cohort was classified into three immune subtypes: immune-active (Cluster 1), immune-inactive (Cluster 2), and immune-suppressive (Cluster 3), with Cluster 1 showing the best overall survival (OS) and Cluster 3 the worst (P=0.046). CXCL9 and DLK1 were identified as representative biomarkers for Cluster 1 and Cluster 3, with AUROCs of 0.91 and 0.96, respectively. In the GEO cohort, the AUROCs of CXCL9 and DLK1 for identifying Cluster 1 and Cluster 3 were 0.90 and 0.82, respectively. Transcriptome-based analysis revealed abundant infiltration of effector immune cells (e.g., CD8+ T cells, NK cells, activated B cells) and higher immune score in Cluster 1, while Clusters 2 and 3 lacked immune cell infiltration and showed lower score. Therefore, Cluster 1 was defined as the Immune-Hot subtype, and Clusters 2 and 3 were combined as the Immune-Cold subtype. In the NCC cohort, consensus clustering using the 1,480-gene set yielded Immune-Hot and Immune-Cold subtypes, which demonstrated a Cohen' s Kappa value of 0.700 (P = 0.048) in agreement with IHC-based immune infiltration classification. The optimal IHC cutoffs for CXCL9 and DLK1 were 0.209 and 0.223, respectively. The IHC-based subtyping defined by these biomarkers showed good agreement with ICI treatment response (Cohen' s Kappa = 0.667, P = 0.012), supporting its predictive utility in clinical settings.

Conclusions This study established an immune subtyping system for HGSOC, comprising immune-active, immune-inactive, and immune-suppressive subtypes, which correlated with ICI response. CXCL9 and DLK1 were identified as reliable subtype biomarkers with strong discriminatory power validated in clinical cohorts. This represents the first HGSOC immune subtyping study with validation in a clinical ICI-treated cohort and the development of an IHC-based classification tool suitable for clinical application.

PO_169

外泌体 miR-148a-3p 的 HUR 调控分选机制及其卵巢癌液体活检应用研究

郑剑锋 姜瑞娜 林雪芬 李艳红 李桑 孙阳*
福建省肿瘤医院

研究目的 本研究旨在探索外泌体 miR-148a-3p 在卵巢癌诊断与预后评估中的作用机制及临床转化价值，重点阐明 RNA 结合蛋白 HUR 介导的选择性分选机制，以期为卵巢癌的无创诊断和精准治疗提供新策略。

材料与方法 通过整合多组学分析技术，从 TCGA 和 GEO 数据库获取卵巢癌临床及基因表达数据；利用人卵巢癌细胞系（包括 A2780、SKOV3 及其顺铂耐药株 A2780-DDP）进行体外实验，采用差速离心法分离外泌体，并通过透射电镜、粒径分析和 Western Blot 进行鉴定；使用 qRT-PCR 检测 miR-148a-3p 在细胞、外泌体及临床样本中的表达水平；通过 RNase A 消化实验和 GW4869 抑制实验验证外泌体分泌途径；收集卵巢癌患者血浆样本及多中心数据集（如 GSE106817）以评估诊断效能；基于 R 语言构建 Lasso-Cox 预后模型和机器学习药物敏感性预测模型；最后，通过 RNA 免疫沉淀（RIP）、miRNA pull-down 以及基因敲低和过表达技术，深入探究 HUR 对 miR-148a-3p 分选的调控机制。

结果 研究发现卵巢癌细胞（尤其是耐药株）外泌体中 miR-148a-3p 表达显著高于正常细胞，但该 miRNA 在细胞内呈低表达；RNase A 联合 Triton X-100 处理导致培养液中 miR-148a-3p 降解，且 GW4869 抑制外泌体分泌后胞内 miR-148a-3p 蓄积增加，证实其依赖外泌体途径分泌。临床样本分析显示，卵巢癌患者血浆外泌体 miR-148a-3p 水平显著高于健康对照，晚期（III–IV 期）患者表达量高于早期（I–II 期）；多中心数据集的 ROC 曲线验证其诊断 AUC 值较高。基于外泌体 miRNA 构建的 Lasso-Cox 预后模型将患者分为高危组和低危组，训练集与验证集的 5 年生存率差异显著，且 5 年 AUC 均大于 0.7。机制研究表明，HUR 在卵巢癌组织中高表达，通过特异性结合 miR-148a-3p 调控其分选：HUR 敲低导致胞内 miR-148a-3p 升高而外泌体中降低，过表达则呈现相反趋势，RIP 和 miRNA pull-down 实验进一步证实两者直接结合。
结论 本研究证实外泌体 miR-148a-3p 可作为卵巢癌的新型液体活检标志物，其表达水平与疾病进展、治疗响应及预后密切相关；HUR 介导的选择性分选机制为干预靶点提供了理论依据。基于外泌体 miRNA 构建的预后模型和诊断策略展现出显著的临床转化潜力，有望推动卵巢癌精准诊疗的发展。

PO_170

Malignant Transformation of Mature Cystic Teratoma Comparison of different pathological types

yirou yan
Shandong Provincial Hospital Affiliated to Shandong First Medical University

Background Malignant transformation (MT) of ovarian mature cystic teratomas (MCTs) is an uncommon but clinically important event, occurring in approximately 1–2% of cases, predominantly in perimenopausal or postmenopausal women. Squamous cell carcinoma (SCC) has historically been reported as the most common subtype, accounting for up to 80% of cases. However, increasing evidence reveals a wider histopathological spectrum, with a significant proportion of non-squamous cell carcinoma (non-SCC), including adenocarcinoma, neuroendocrine tumors, and thyroid carcinoma. Due to the rarity of these events, especially non-SCC subtypes, their clinical characteristics, diagnostic indicators, and prognostic implications remain inadequately understood and warrant further investigation.
Methods We conducted a two-part investigation comprising a retrospective review of nine cases of MCT-MT treated at our institution between 2000 and 2024, and a systematic literature review encompassing 338 additional cases published between 2000 and 2024, following PRISMA guidelines. Inclusion criteria focused on cohort or case series studies reporting both SCC and non-SCC subtypes with detailed clinicopathological data. Clinical variables, histological classification, serum tumor marker profiles, treatment regimens, and survival outcomes were extracted and analyzed. Kaplan–Meier survival analysis and Cox regression modeling were used to assess the prognostic significance of histological subtype, tumor markers, and FIGO stage.
Results Among the 338 cases analyzed, squamous cell carcinoma (SCC) was identified in 59% of patients, confirming its role as the predominant histological subtype in malignant transformation of ovarian mature cystic teratomas (MCTs). The remaining 41% were classified as non-squamous cell carcinoma (non-SCC) subtypes, encompassing a diverse range of malignancies such as adenocarcinoma, neuroendocrine tumors, sarcoma, and thyroid carcinoma. Demographic and clinical parameters, including patient age and tumor size at diagnosis, were comparable between the SCC and non-SCC cohorts, suggesting that these variables alone are insufficient to distinguish between histological subtypes. However, analysis of preoperative serum tumor markers revealed distinct biomarker profiles across subtypes. SCC patients more frequently demonstrated elevated squamous cell carcinoma antigen, consistent with its epithelial origin. In contrast, non-SCC cases exhibited significantly higher levels of carcinoembryonic antigen (CEA) and carbohydrate antigen 19-9 (CA19-9) ($p < 0.05$). Although overall survival did not differ significantly between SCC and non-SCC groups, early-stage disease (FIGO stage I) was strongly associated with improved prognosis across all

subtypes. Among non-SCC malignancies, thyroid carcinoma demonstrated the most favorable outcomes, which may be attributed to both early detection and the typically indolent clinical course associated with this histology.
Conclusion This study highlights the heterogeneity of malignant transformations in MCTs, with SCC and non-SCC subtypes showing distinct tumor marker profiles and prognostic trends. FIGO staging emerged as the most robust predictor of survival, emphasizing the importance of early detection and comprehensive staging surgery. The differential expression of tumor markers among histological types offers potential utility in preoperative risk stratification and subtype suspicion. These findings support the need for individualized management strategies based on histology, stage, and biomarker profiles, and underscore the importance of prospective research to establish standardized treatment protocols for rare non-SCC variants.

PO_171

BRCA1/2 突变功能域和类型与新诊断晚期卵巢癌铂类化疗和 PARPi 获益的相关性

丁婷
四川大学华西第二医院

目的 目前临床实践对 BRCA1/2 基因的关注主要集中在基因状态（突变与否）上 BRCA1/2 基因型对治疗效果的影响仍有待研究。我们分析了 BRCA 基因突变位置和类型与高级别浆液性卵巢癌（HGSOC）患者铂类化疗（PBC）或 PARPi 治疗获益的相关性，旨在预测治疗敏感性并制定更精确的个体化治疗策略。
材料与方法 确定了一个由 1189 名接受 BRCA1 和 BRCA2 基因检测的 HGSOC 患者组成的研究队列。根据突变功能域 [BRCA1：真正有趣的基因（RING）、DNA 结合域（DBD）、BRCA1 的 C 端域（BRCT）； BRCA2：RAD51 结合域（RAD51-BD）、DBD、额外的 RAD51 相互作用位点（TR2）] 和突变类型 [移码（FS）、无义（NS）、错义突变（MS）、剪接或大重排（LR）]分析 PFS 和 OS。TCGA-OV 样本（n = 316）用于外部验证。
结果 研究队列中有 860 例 BRCA 野生型（BRCAwt），203 例 BRCA1 突变，和 126 例 BRCA2 突变。26、72 、 38 和 67 个 BRCA1 突变分别位于 RING、DBD 、 BRCT 和其他位置；在 65、19 、 7 和 31 个 BRCA2 突变分别位于 RAD51-BD、DBD 、 TR2 和其他位置。FS 是最主要的突变类型（51.7%，n=170），其次是 NS（32.5%，n=107）、MS（9.4%，n=31）、LR（4.6%，n=15）和剪接突变（1.8%，n=6）。中位随访时间为 33.3 个月。与 BRCAwt 相比，BRCA2 突变（而非 BRCA1 突变）具有显著的 PBC 获益。BRCA1 和 BRCA2 突变的铂获益程度没有差异。BRCA1 RAD51-BD 突变的 PBC 获益最高，3 年 PFS 率和 5 年 OS 率分别为 14%和 43%。不同功能域的突变在 PBC 获益方面未观察到差异。BRCA1 DBD 突变[HR=0.17（0.08-0.36）]的 PAPRi 的敏感性最高，3 年 PFS 和 5 年 OS 率分别为 56%和 54%。BRCA2 RAD51-BD 突变[HR=0.25 (0.11-0.56)]和 DBD 突变[HR=0.18 (0.04-0.89)]显示出良好的 PARPi 获益。BRCA1 RING [HR=0.39 (0.11-1.42)] 和 BRCA2 TR2 [HR=0.75 (0.07-8.42)] 突变的 PARPi 的敏感性最低。排除突变位置的影响后，所有突变类型均未与 PBC 或 PARPi 获益的效应修饰相关。使用 TCGA-OV 数据也发现了类似结果。
结论 铂获益与 BRCA1/2 突变位置无关。位于 DBD 的 BRCA1 突变 PARPi 获益最大，其次是位于 RAD51-BD 的 BRCA2 突变，而位于 RING 和 TR2 突变的 PARPi 益处较小。

PO_172

卵巢癌中 BTN3A1 分子通过 TCR 信号途径对 Vγ9Vδ2 T 细胞的功能调控

刘健 吴敏 汪辉*
浙江大学附属妇产科医院

背景 过继输注 T 细胞治疗在卵巢癌中具有很好的应用前景，特别是体外扩增的 Vγ9Vδ2 T 细胞对卵巢癌肿瘤细胞具有很好的杀伤潜力，但其总体治疗效果仍然较低。探索 Vγ9Vδ2 T 细胞进入卵巢肿瘤微环境的功能变化及相关机制可以为提升 Vγ9Vδ2 T 细胞过继输注治疗效果和临床转化提供基础。
方法 通过 TCGA 和 GO 等数据库以及二代转录组测序技术（RNA sequencing，RNA-seq）探索初步探索了 Vγ9Vδ2 T 细胞在卵巢癌中的功能特性以及与肿瘤细胞相互作用的分子和通路。进一步将体外扩增 Vγ9Vδ2 T 细胞与卵巢癌肿瘤细胞系共培养，通过流式细胞术验证影响 Vγ9Vδ2 T 细胞功能的分子和通路。
结果 卵巢癌中的 Vγ9Vδ2 T 细胞具有较高的抗肿瘤特性，肿瘤细胞中 BTN3A1 的高表达会降低扩增的 Vγ9Vδ2 T 细胞的活化水平，促进耗竭相关分子表达升高，凋亡水平升高，阻断 TCR 受体会消除 BTN3A1 对 Vγ9Vδ2 T 细胞耗竭的影响。
结论 BTN3A1 在激活 Vγ9Vδ2 T 细胞的同时也会通过 TCR 信号途径启动耗竭程序，BTN3A1 可能作为一种新的免疫检查点分子发挥作用。靶向 BTN3A1 下游相关分子对提升 Vγ9Vδ2 T 细胞过继输注治疗具有重要意义。

PO_173

PARP 抑制剂一线维持治疗的卵巢癌患者预后分析：一项回顾性队列研究

谭舒然 张瑜*
中南大学湘雅医院

研究目的 聚 ADP 核糖聚合酶抑制剂（PARPi）广泛应用于卵巢癌的一线维持治疗。然而，大量的临床试验表明，仍有一些 BRCA 突变或同源重组缺陷（HRD）患者不能从 PARPi 中获益，BRCA 野生型患者更是获益有限。因此，有必要开发新的模型来更好地预测 OC 对 PARPi 的反应。
材料与方法 收集了 145 例 2018 年 8 月至 2024 年 5 月期间在中南大学湘雅医院初治且接受 PARP 抑制剂进行一线维持治疗的卵巢癌患者的临床资料，并进行了分析。通过单因素和多因素 COX 回归分析来确定变量并筛选预测因素。使用多因素 Cox 回归构建了一个列线图，以预测接受 PARP 抑制剂治疗患者的预后。通过受试者工作特征曲线、校准曲线以及决策曲线分析来评估列线图的区分度、校准度以及临床应用价值。
结果 接受 PARPi 作为一线维持治疗的卵巢癌患者，其无进展生存期（PFS）和总生存期（OS）均有所改善（中位 PFS：48.53 个月）。Cox 回归分析确定了几个与延长 PFS 相关的独立预测因素，包括 BRCA 突变、无残留病灶（R0）、FIGO 分期 III 期以及更高的 CA-125 消除率常数（KELIM）评分（KELIM > 1）。此外，BRCA 突变和患者年龄较小是更好的 OS 的显著预测因素。PFS 列线图模型的符合指数为 0.793。1 年、2 年和 3 年 PFS 的校准曲线表明，预测结果与实际观察结果高度一致，而受试者工作特征曲线和决策曲线分析曲线则显示了显著的临床实用性。此外，通过对 42 例复发的卵巢癌病人进行分析发现，含铂化疗及贝伐珠单抗的使用是延长二线 PFS 的独立危险因素，且 PARPi 使用时长超过一年可能会损害二线 PFS。

结果 该临床预测列线图图谱对于接受 PARP 抑制剂作为一线维持治疗的卵巢癌患者显示出令人鼓舞的预测效果，表明其在不同人群中的应用潜力；而 PARPi 持续时间较长可能是后续治疗的危险因素。

PO_174

腹腔热灌注化疗对同步腹膜转移的晚期上皮性卵巢癌患者行初次肿瘤细胞减灭术的生存结局：一项回顾性真实世界的队列研究

李倩 于明新 佟晓晶*
辽宁省肿瘤医院

目的 对于行初次肿瘤细胞减灭术（PDS）且伴有广泛腹膜转移的晚期上皮性卵巢癌（EOC）患者接受腹腔热灌注化疗（HIPEC）是否获益仍在争论中。这项真实世界的回顾性队列研究探讨了这部分患者是否获益。

材料与方法 纳入初次就诊于我院妇科、术后病理证实为 EOC 的接受 PDS 的患者，按照 1：1 比例进行倾向匹配评分，分为两组，包括进行 PDS 联合 HIPEC 治疗的患者（HIPEC 组）和仅接受 PDS 的患者（非 HIPEC 组）。HIPEC 灌注采用封闭技术进行，第 1 天使用 3000ml 灭菌注射用水或 1500ml 灭菌注射用水和 1500ml 0.9% NaCl 溶液；第 2 天，紫杉醇 175mg/m2；第 3 天，顺铂 75mg/m2，在 43℃下灌注 60min，速度为 300ml/min。主要研究终点无进展生存期（PFS）和总生存期（OS）。

结果 从 2021 年 7 月到 2022 年 12 月，共纳入 68 例，其中 HIPEC 组 33 例，非 HIPEC 组 35 例。两组中位年龄均为 57 岁；达满意肿瘤细胞减灭术患者占比均高（HIPEC 组：84.85%；非 HIPEC 组：97.14%）。在非 HIPEC 组和 HIPEC 组患者中 III 期分别为 27 例（81.82%）和 32 例（91.43%），IV 期分别为 6 例（18.18%）和 3 例（8.57%）。与非 HIPEC 组比较，HIPEC 组患者术后 CA125 转阴的时间较长（11.71 天对比 9.29 天, P =0.0002）、术后接受化疗的起始时间间隔较短（3.375 天对比 29.8 天, P ＜0.0001）。两组的手术方式存在统计学差异，与非 HIPEC 组比较，HIPEC 组患者术中接受脏器修补及肠造瘘的例数较多，行脏器全部或部分切除术者例数虽较少（P =0.0022）。HIPEC 组患者住院时间较非 HIPEC 组患者长（12.5 天对比 11.5 天，P =0.0158）。两组术后并发症发生率没有统计学差异(P = 0.547)。中位随访时间为 33 个月（26-40 个月），与非 HIPEC 组比较，HIPEC 组患者的 PFS 显著延长，两组中位 PFS 分别为 HIPEC 组患者可达 28 个月（范围：7-38 个月）和非 HIPEC 组患者可达 21 个月（范围：9-32 个月）（P =0.0265）；但两组的中位 OS 没有统计学差异，分别为 HIPEC 组患者 31 个月（范围：9-40 个月）和非 HIPEC 组患者 30 个月（范围:14-37 个月)（P =0.5881）。

结论 这项大样本回顾性队列项究提供了伴有广泛腹膜转移的晚期 EOC 患者在接受满意的 PDS 后联合应用以紫杉醇及顺铂方案的 HIPEC 治疗会显著延长患者 PFS 使患者临床获益的证据。但仍需要更长时间的随访及前瞻性研究的数据。

PO_175

热疗协同 PARP 抑制剂通过调控 DNA 损伤修复通路增强卵巢癌治疗敏感性的机制研究

崔耀元
华中科技大学同济医学院附属同济医院

研究目的 卵巢癌患者对铂类化疗耐药是临床治疗失败的主要原因。近年来，腹腔热灌注化疗（HIPEC）在晚期卵巢癌中的应用显示出潜在优势，但其与靶向药物的协同机制尚不明确。本研究探讨热疗联合 PARP 抑制剂通过调控 DNA 损伤修复通路增强卵巢癌治疗敏感性的作用机制。
材料与方法 通过转录组测序（RNA-seq）分析热疗处理后卵巢癌细胞（SKOV3、OVCAR3）的差异表达基因，聚焦 DNA 损伤修复通路变化；基于 GDSC 数据库筛选与热疗敏感性相关的靶向药物；采用 CCK-8 实验验证协同效应，克隆形成实验及流式细胞术评估联合治疗的增殖抑制与凋亡诱导作用；免疫荧光检测 γ-H2AX 焦点形成，Western blot 分析 DNA 损伤修复关键蛋白（PARP1、ATM、BRCA1）表达；体内实验通过 PDX 模型评估联合治疗的肿瘤抑制效果。
结果 转录组分析显示，热疗（42°C，1 h）显著下调同源重组修复（HRR）通路相关基因（BRCA1、RAD51），同时上调 PARP1 表达（*p* < 0.01）。药物筛选发现 PARP 抑制剂奥拉帕利与热疗具有显著协同效应（联合指数 CI < 0.8）。热疗联合奥拉帕利可协同增加 DNA 双链断裂标志物 γ-H2AX 焦点数量（*p* < 0.001），并抑制 HRR 蛋白 BRCA1 和 RAD51 的核定位。体内实验证实，联合治疗组的小鼠肿瘤体积较单药组减少 52%（*p* = 0.003），且未增加毒性。
结果 热疗通过抑制 HRR 通路并诱导“合成致死”效应，显著增强卵巢癌对 PARP 抑制剂的敏感性。本研究为临床应用热疗联合 PARP 抑制剂治疗铂类耐药卵巢癌提供了理论依据。

PO_176

LRRC15 阳性成纤维细胞通过 ITGB1 介导的信号通路促进卵巢癌侵袭与转移的机制研究

曹琼 张莉 冯晨钊 孙朝阳* 陈刚
华中科技大学同济医学院附属同济医院

背景与目的 卵巢癌是致死率最高的妇科恶性肿瘤，其高侵袭性和广泛转移是治疗失败的主要原因。肿瘤微环境（TME）中的癌症相关成纤维细胞（CAFs）在调控肿瘤进展中发挥关键作用。近期研究发现，富含亮氨酸重复序列的蛋白 LRRC15 在多种实体瘤的 CAFs 中特异性高表达，但其在卵巢癌中的功能及机制尚未明确。本研究旨在探讨 LRRC15 阳性成纤维细胞在卵巢癌侵袭转移中的作用及其分子机制。
方法 通过单细胞测序和免疫组化分析卵巢癌患者原发灶及转移灶中 LRRC15 阳性成纤维细胞的分布特征。构建 LRRC15 基因敲除的成纤维细胞模型，与卵巢癌细胞（SKOV3、OVCAR3）共培养，利用 Transwell 实验及 3D 肿瘤球体侵袭模型评估癌细胞侵袭能力。通过 RNA-seq 筛选下游关键分子，结合免疫共沉淀（Co-IP）和荧光素酶报告基因验证 LRRC15 与整合素 β1（ITGB1）的互作关系。建立小鼠腹腔移植瘤模型，体内验证 LRRC15/ITGB1 轴对卵巢癌转移的调控作用。
结果 1）LRRC15 在卵巢癌周围 CAFs 中的表达显著高于正常卵巢间质，且与晚期（III/IV 期）及淋巴结转移呈正相关；
2）LRRC15 阳性成纤维细胞通过分泌 TGF-β1 诱导癌细胞上皮-间质转化（EMT），敲低 LRRC15 可使癌细胞侵袭能力下降；
3）机制研究表明，LRRC15 通过直接结合 ITGB1 激活 FAK/PI3K-AKT 通路，促进基质金属蛋白酶（MMP2/9）表达；

结论 LRRC15 阳性成纤维细胞通过 ITGB1 介导的整合素-FAK 信号轴重塑肿瘤微环境，促进卵巢癌侵袭转移。本研究首次揭示 LRRC15/ITGB1 通路作为卵巢癌治疗的新靶点，为开发靶向 CAFs 的联合治疗策略提供理论依据。

PO_177

卵巢癌患者中乙型肝炎病毒感染的流行情况和影响因素-一项回顾性队列研究

陈莉 洪波*
中国科学院合肥肿瘤医院

目的 乙型肝炎病毒（HBV）感染与许多肝外恶性肿瘤有关，但其与卵巢癌的关联和影响尚未得到研究。因此，我们研究了原发性卵巢癌患者中 HBV 感染的流行情况，以及是否影响患者的临床病理学特征和生存。
方法 在一项回顾性研究中，我们回顾了 2018 年至 2022 年期间 89 例卵巢癌和已知 HBV 感染状态的患者，将患者分为 HBV 阳性组和阴性组，比较患者的特征和表现，包括年龄、FIGO 分级、外周血淋巴细胞亚群，以及卵巢癌的无疾病进展生存期（PFS）。计量数据以 $x \pm s$ 表示，相关性分析采用 Pearson 相关分析法。使用逻辑回归模型（logistic regression model）来估计 HBV 感染与卵巢癌患者在诊断治疗后无疾病进展生存期的独立关联。以 $P < 0.05$ 为差异有统计学意义。
结果 HBV 感染阳性组占比 19.1%，阴性组占比 80.9%，根据 HBV 感染状态分析卵巢癌患者临床病理学特征时，发现两组间在年龄、分期方面的分布无显著差异。但在外周血淋巴细胞亚群表达水平方面，HBV 感染阳性组的 CD8+T 细胞百分比低于 HBV 阴性组（22.39±7.69 vs 29.92±12.86，n=61），且差异有统计学意义（P =0.02）。虽然 HBV 感染状态与卵巢癌患者的 PFS 之间没有统计学差异，但在前 24 个月内，我们发现 HBV 感染阴性组较 HBV 感染阳性患者更早发生疾病的进展，在 24 个月后，则相反。
结论 本研究结果提示 HBV 感染对原发性卵巢癌患者的淋巴细胞有影响，且可能对 2 年内的无疾病进展生存期有有利影响。

PO_178

基于多学科会诊的决策方法选择新辅助化疗与初始肿瘤细胞减灭术治疗晚期卵巢癌的生存结局比较

王辰宇 张瑜* 谭舒然
中南大学湘雅医院

研究背景 新辅助化疗(NACT)推荐用于通过初次肿瘤细胞减灭术(PDS)难以实现完全肿瘤细胞减灭的晚期卵巢癌患者，或因合并症存在围手术期高风险特征的患者。NACT 与 PDS 之间的决策选择需谨慎评估实现最佳满意减瘤的可行性，强调个体化决策的重要性。本研究评估基于多学科会诊(MDT)的个体化治疗方案选择是否能改善生存结局。
研究方法 本回顾性研究纳入 2013 年 1 月至 2024 年 4 月期间在中南大学湘雅医院接受初始或间歇性肿瘤细胞减灭术的 405 例国际妇产科联盟(FIGO)分期 III 至 IV 期卵巢癌、输卵管癌或原发性腹膜癌患者。治疗方案由妇科肿瘤科医生、影像科医生、肿瘤放化科医生和病理科医生组成的多学科团队协同制定。采用机构标准方法依据患者体能状态和/或计算机断层扫描(CT)检查结果提示无法实现满意肿瘤细胞减灭术以评估 NACT(新辅助化疗)适用性。主要研究终点为总生存期(OS)，次要终点为无进展生存期(PFS)。

研究结果 在 405 例患者中，128 例(31.6%)接受 NACT 后接受 IDS(间歇性肿瘤细胞减灭术)，277 例(68.4%)接受PDS(初始肿瘤细胞减灭术)。PDS组中位PFS显著优于NACT组(30.4个月 vs 18.1个月；HR:0.55，95% CI：0.41–0.74，p<0.0001)。PDS 组的中位 OS 同样优于 NACT 组(57.8 个月 vs 37.9 个月；HR:0.55，95% CI：0.40–0.77，p<0.0001)。PDS 组的五年 PFS 率和 OS 率分别为 34.6%和 49.6%，而NACT组分别为15.9%和31.7%。全部患者中肉眼可见病灶完全切除率为46.9%和44.5%，两组间比例相当(p=0.6)。在减瘤满意度的亚组分析中，PDS 组达到肉眼可见病灶完全切除的中位 PFS 显著优于其他三组(57.1 个月 vs 18.7 个月 vs 20.6 个月 vs 16.6 个月；p<0.0001)。在 277 例接受 PDS 的患者中，47例(17.0%)直接接受PDS，230例(83%)接受多学科会诊后接受PDS。多学科会诊后PDS组的肉眼可见病灶完全切除率明显优于直接接受 PDS 组(50.4% vs 29.8%；p=0.0097)。

研究结论 与接受 NACT 后行 IDS 相比，PDS 在晚期卵巢癌患者中显示出更优的 OS 和 PFS，且 PDS 达到肉眼可见病灶完全切除的患者预后最佳。多学科会诊体系建立后，PDS 达到肉眼可见病灶完全切除优势逐步扩大，凸显其在个体化治疗决策中的关键作用。

PO_179

巨噬细胞胞葬多胺代谢重编程驱动化疗后卵巢癌肿瘤干细胞富集

李文翰
武汉协和医院

研究目的 化疗诱导的癌症干细胞（CSCs）富集是上皮性卵巢癌（OC）获得性化疗耐药和复发的关键机制。尽管化疗可能通过选择或诱导去分化来富集CSCs，但在化疗过程中肿瘤微环境的动态变化及其对 CSCs 的影响尚不清楚。本研究旨在通过单细胞测序和多重免疫组化分析，明确化疗后肿瘤微环境的变化，并探讨其对 CSCs 的影响。

材料与方法 本研究采用单细胞测序和多重免疫组化分析技术，对化疗后的肿瘤微环境进行分析。通过检测化疗诱导的凋亡肿瘤细胞被吞噬后的巨噬细胞（即胞葬巨噬细胞）的变化，分析其与 CSCs 的关系。同时，利用代谢组学和转录组学整合分析，研究胞葬作用驱动的代谢重编程机制。此外，通过体外实验验证胞葬巨噬细胞的条件培养基对 OC 干细胞特性的影响，并在体内实验中评估靶向胞葬作用对 CSC 富集、化疗耐药和肿瘤再生长的抑制效果。

结果 通过单细胞测序和多重免疫组化分析，我们发现化疗后胞葬巨噬细胞数量显著增加，这些巨噬细胞通过吞噬化疗诱导凋亡的肿瘤细胞而形成。胞葬巨噬细胞与卵巢癌（OC）中的癌症干细胞（CSCs）及不良预后密切相关，其条件培养基在体外能够显著促进 OC 细胞的干细胞特性。进一步的代谢组学和转录组学整合分析揭示，胞葬作用通过增强 ODC1 的表达，增加多胺代谢通量，尤其是腐胺的含量。腐胺含量的增加进一步导致巨噬细胞中 SPP1 和 OPN 的过表达，并通过 OPN-CD44 轴赋予 OC 细胞癌症干细胞特性。在体内实验中，靶向胞葬作用能够有效抑制 CSC 的富集、化疗耐药以及肿瘤的再生长，而使用 ODC1 选择性抑制剂则可显著减轻 CSC 的富集，增强肿瘤对顺铂的敏感性，并限制肿瘤的再生长。

结论 本研究揭示了化疗诱导的胞葬巨噬细胞通过 ODC1 介导的多胺代谢重编程（特别是腐胺增加）促进卵巢癌干细胞（CSCs）富集的机制，并证实靶向胞葬作用或 ODC1 可抑制 CSC 富集、增强化疗敏感性，为克服卵巢癌化疗耐药和复发提供了新靶点。

PO_180

线粒体复合物在卵巢癌中的研究进展与治疗潜力

孙梦娜 马凌宇 杨红*
空军军医大学第一附属医院

目的 卵巢癌在女性生殖系统疾病中致命性和病死率极高，从分子水平揭示卵巢癌发生发展的潜在机制，寻找有效的肿瘤标记物及治疗靶点，对于卵巢癌的诊断、疾病监测及治疗十分重要。肿瘤细胞通过代谢重编程获取其快速增殖所需要的能量，其中线粒体氧化磷酸化（OXPHOS）通路尤为关键。线粒体复合物是维持氧化磷酸化与细胞稳态的核心结构，其功能异常与多种恶性特征密切相关。大量研究表明，靶向线粒体代谢重编程是治疗癌症的一种潜在有前途的方式。目前线粒体复合物在卵巢癌中的具体作用和机制仍然不清楚。本文就现有的研究结果对线粒体复合物在卵巢癌发生发展及耐药过程中的分子机制进行总结概括，以期为卵巢癌的防治、诊断及治疗等方面提供新的思路和方法。
方法 通过系统检索 PubMed、Web of Science、中国知网等数据库，筛选回顾与“Mitochondrial complex”、“OXPHOS”、“ovarian cancer”、“chemoresistance”关键词相关的文献，归纳复合物在卵巢癌中的表达特征、功能变化及其与化疗耐药的关系，评估其潜在临床价值。
结果 多项研究表明，卵巢癌组织中复合物 I、III 和 IV 的表达异常，尤其在高级别浆液性癌及顺铂/紫杉醇耐药细胞中更为显著。复合物 I 活性增强导致 NADH 氧化速率升高，伴随 ROS 累积，激活 NF-κB、HIF-1α 通路，促进肿瘤进展和血管生成。复合物 III 异常引发电子泄漏，进一步加剧氧化应激。OXPHOS 基因表达高与卵巢癌晚期、复发和预后差显著相关。部分 OXPHOS 抑制剂如 IACS-010759、metformin 在体外可增强化疗敏感性，提示其与化疗药物联用具有治疗潜力。
结论 线粒体复合物在卵巢癌的发生发展及耐药过程中具有重要的功能性作用。其表达和活性改变不仅驱动肿瘤细胞代谢重编程，促进细胞增殖与侵袭，还与传统化疗耐药密切相关。靶向线粒体复合物，特别复合物 I 和 III，有望为治疗耐药卵巢癌提供新策略。

PO_181

鹰嘴豆素 A 通过下调 Bcl-2 蛋白表达抑制卵巢癌进展的机制研究

胥静雯
南通大学附属医院

目的 卵巢癌是女性常见的恶性肿瘤之一，其病死率在妇科恶性肿瘤中占据首位，严重威胁了世界各地女性的健康，其高复发率和耐药性导致临床治疗困难。Bcl-2 蛋白作为抗凋亡关键因子，在卵巢癌中常过表达，促进肿瘤细胞存活。鹰嘴豆素 A (Biochanin A,BCA)是传统中草药红车轴草的主要有效成分，是一种天然异黄酮，具有抗炎、抗菌、抗氧化、抑制组织增生等多种药理作用，前期研究表明其具有潜在的抗肿瘤活性，但具体机制尚不明确。本研究旨在探究鹰嘴豆素 A 是否通过调控 Bcl-2 蛋白表达抑制卵巢癌进展，为开发新型治疗策略提供实验依据。
方法
1.使用 AutoDock Vina 软件进行分子对接研究，预测鹰嘴豆素 A 和 Bcl-2 之间的结合亲和力和氢键的存在。
2.卵巢癌细胞系(SKOV3/A2780)分别用不同浓度鹰嘴豆素 A（0、10、20、40 μM）处理，通过 CCK8 实验、克隆形成实验、流式细胞术、Hoechst33342 染色、划痕实验、Transwell 侵袭实验检测不同处理组对细胞增殖、凋亡、迁移、侵袭的影响，体外验证鹰嘴豆素 A 对卵巢癌细胞生物学行为的影响。
3.通过 Western Blot、免疫荧光检测 Bcl-2 及凋亡相关蛋白表达和表达变化，qRT-PCR 检测 Bcl-2 mRNA 水平，siRNA 转染验证敲低后鹰嘴豆素 A 效果。

4.构建裸鼠移植瘤模型，通过免疫组化、TUNEL 染色检测肿瘤组织 Bcl-2、Ki-67、Cleaved Caspase-3 表达和肿瘤细胞凋亡情况。

结果

1.鹰嘴豆素 A 抑制卵巢癌细胞增殖、迁移，并诱导凋亡。

2.Bcl-2 蛋白及 mRNA 表达显著下调，激活 BAX/Cleaved Caspasse-3 凋亡通路。

3.动物实验证实鹰嘴豆素 A 抑制肿瘤生长，且 Bcl-2 表达降低。

结论 我们的实验研究证明，鹰嘴豆素 A 通过转录下调 Bcl-2，激活 BAX/Cleaved Caspasse-3 凋亡通路，从而抑制卵巢癌进展，具有潜在的治疗价值。

PO_182

卵巢成熟囊性畸胎瘤恶变患者的生存及生育结局分析

李炎姬 郭瑞霞*
郑州大学第一附属医院

目的 探讨卵巢成熟囊性畸胎瘤（ovarian mature cystic teratoma, OMCT）恶变患者的肿瘤结局和生育结局。

方法 回顾性收集 2011 年 8 月至 2024 年 12 月郑州大学第一附属医院收治的 47 例 OMCT 恶变患者。分析患者的一般资料、病理特征，并随访其预后及生育结局。

结果 纳入患者的平均年龄为（51.0±15.2）岁，平均肿瘤直径为（10.6±4.5）cm。OMCT 恶变为鳞癌 29 例（29/47，61.70%），腺癌 9 例（9/47，19.20%），类癌 8 例（8/47，17.00%），恶性黑色素瘤 1 例（1/47，2.10%）。34 例（34/47，72.3%）行术中快速冰冻病理检查，提示恶性肿瘤 18 例（18/34，52.9%），提示卵巢畸胎瘤恶变不除外 2 例（2/34，5.9%）。所有患者的中位随访时间为 43.0（5.0-117.0）月。随访期内，有 12 例患者出现复发（12/47，25.50%），末次随访时，13 例（13/47，27.66%）患者死亡，其余患者均无瘤生存。5 年 DFS 和 OS 分别为 71.70%和 68.80%。Cox 多因素分析显示，肿瘤直径≥13cm（HR=15.548，95%CI:2.983-81.054）是影响 OMCT 恶变患者 DFS 的独立危险因素（P<0.05）。肿瘤直径≥13cm（HR=19.976，95%CI:3.386-117.851）、术中肿瘤破裂（HR=10.809，95%C:1.750-66.748）、手术未达 R0（HR=23.551，95%CI:1.762-314.715）是影响 OMCT 恶变患者 OS 的独立危险因素（P<0.05）。此外，11 例 OMCT 恶变患者行保留生育功能手术（fertility sparing surgery, FSS）,其中，IA 期 10 例，II 期 1 例。随访期内，4 例（4/11，36.36%）患者有生育意愿，其中 3 例（3/4，75.00%）患者术后自然妊娠并顺利分娩，仅 1 例 II 期的患者因疾病进展死亡，其余患者均无瘤生存。

结论 肿瘤直径≥13cm、术中肿瘤破裂、手术未达 R0 与 OMCT 恶变患者的生存密切相关，可预测不良预后。对于 IA 期有生育要求的 OMCT 恶变患者，可考虑行 FSS 手术，但术后仍需密切随访。

PO_183

Exploring the function of LAG-3 and Diagnostic Value of Soluble LAG-3 Ascites/Plasma Ratio in Epithelial Ovarian Cancer

Ying Zhou Jingluan Tian Xiu Shi Huijuan Meng Juan Wang Jinhua Zhou Yanzheng Gu*
the First Affiliated Hospital of Soochow University

Objective Epithelial ovarian cancer (EOC), which accounts for approximately 80% of all ovarian cancer cases, exhibits high mortality largely due to its complex tumor microenvironment (TME). Lymphocyte activation gene-3 (LAG-3), an immune checkpoint receptor, has been implicated in modulating immune

responses within the TME. However, its clinical significance and potential as a diagnostic or prognostic biomarker in EOC remain to be fully elucidated.
Methods We conducted comprehensive bioinformatics analyses using gene expression data from The Cancer Genome Atlas (TCGA) to explore LAG-3's role in the TME. In parallel, EOC tissue samples were analyzed by immunohistochemistry (IHC), multiplex immunofluorescence, and flow cytometry to evaluate LAG-3 expression and immune cell infiltration. Soluble LAG-3 (sLAG-3) concentrations in ascites and plasma were quantified by ELISA, and the ascites-to-plasma (A/P) sLAG-3 ratio was calculated. Diagnostic and prognostic performance of this ratio was compared to traditional biomarkers (CA125 and HE4).
Results LAG-3 expression in tumor tissues positively correlated with the infiltration of immune cells, particularly $CD8^+$ T cells and TCR δ^+ γδ T cells, and was associated with improved overall prognosis in EOC patients. Higher levels of sLAG-3 were detected in ascites from patients with advanced-stage disease, lymphatic metastasis, high-grade tumors, and incomplete surgical resection. The sLAG-3 A/P ratio demonstrated superior diagnostic sensitivity and specificity over CA125 and HE4 in identifying EOC, predicting lymphatic spread, and estimating the likelihood of achieving optimal cytoreduction. Moreover, elevated ascitic cytokines IL-6 and IL-10 were significantly associated with increased sLAG-3 A/P ratios, suggesting an immunosuppressive TME.
Conclusion: LAG-3 plays a critical role in shaping the immune landscape of EOC. The sLAG-3 ascites/plasma ratio is a promising novel biomarker that outperforms conventional indicators in diagnosing EOC, predicting metastasis, and assessing surgical outcomes, with strong links to immune suppression in the tumor microenvironment.

PO_184

B7-H4 Blockade Reverses T Cell Exhaustion and Enhances the Efficacy of Vγ9Vδ2 T Cell Adoptive Therapy in Reprogramming the Immune Microenvironment of Ovarian Cancer

Ying Zhou Jinhua Zhou Juan Wang Yanzheng Gu*
the First Affiliated Hospital of Soochow University

Objective Ovarian cancer (OC) often escapes immune surveillance, rendering current PD-1/PD-L1 checkpoint inhibitors sub-optimal. B7-H4, a co-inhibitory ligand of the B7 family, is highly expressed in epithelial ovarian cancer (EOC) and correlates with tumour progression, chemoresistance and poor prognosis. Because B7-H4 negatively regulates T-cell function, it is postulated to drive T-cell exhaustion and immune evasion within the EOC microenvironment. This study elucidates the mechanisms underlying B7-H4–induced exhaustion and evaluates novel immunotherapeutic strategies to overcome this suppression.
Methods Lentiviral transduction was used to generate B7-H4–overexpressing SKOV3 and A2780 cell lines. Human PBMC-reconstituted NCG mice bearing subcutaneous xenografts were employed to track tumour growth and therapeutic responses in real time. Bulk-tumour RNA-seq with KEGG/GO enrichment analysis was performed to define B7-H4–driven molecular alterations. Multiparameter flow cytometry quantified immune-cell phenotypes, exhaustion markers (PD-1, TIM-3, LAG-3), co-stimulatory molecules (CD80, CD86) and cytotoxic function. A 92-specimen EOC tissue microarray underwent multiplex immunofluorescence to map B7-H4, $CD8^+$ and Vγ9Vδ2 γδ-T-cell spatial distributions; survival was analysed by Kaplan–Meier and multivariate Cox models. Xenografted mice were randomised to five groups—LV-CON, LV-VTCN1 con, LV-VTCN1 + anti-B7-H4 mAb, LV-VTCN1 + γδ-T-cell transfer, and LV-VTCN1 + combined therapy—and assessed for tumour burden, peripheral/intratumoural lymphocyte composition, memory subsets (TCM, TSCM), cytokines, survival and hepatorenal histopathology.

Results B7-H4 overexpression significantly accelerated tumor growth and fostered an immunosuppressive tumor microenvironment. Transcriptomic profiling of tumors with high B7-H4 expression revealed downregulation of antigen presentation and T cell activation pathways, in line with these observations. Accordingly, immune phenotyping showed that tumors with high B7-H4 had reduced expression of key co-stimulatory molecules (CD80, CD86) and upregulated multiple inhibitory receptors on infiltrating T cells (including PD-1, TIM-3, LAG-3), consistent with a profoundly exhausted T cell phenotype. Consistently, analysis of ovarian cancer patient cohorts indicated that high infiltration of Vγ9Vδ2 γδ T cells was associated with improved survival, particularly in patients whose tumors had low B7-H4 expression, underscoring the clinical relevance of this immune axis. In functional assays, Vγ9Vδ2 T cells retained substantially greater cytotoxic activity against B7-H4–overexpressing tumor cells compared to conventional CD8+ T cells, suggesting that γδ T cells are less susceptible to B7-H4–mediated immunosuppression. Importantly, blockade of B7-H4 with a specific antagonistic monoclonal antibody restored T cell proliferation and cytotoxicity in co-culture and significantly slowed tumor progression in vivo, confirming that B7-H4 is a viable immunotherapeutic target. Building on these findings, we evaluated a combination treatment of B7-H4 blockade together with adoptive transfer of Vγ9Vδ2 T cells. This combined therapy yielded synergistic anti-tumor effects, markedly enhancing tumor control in vivo compared to either treatment alone. The combination also promoted a qualitatively superior immune response, with increased intratumoral γδ T cell infiltration and expansion, along with enhanced generation of central memory (TCM) and stem cell memory (TSCM) T cell subsets—features indicative of improved T cell persistence. Furthermore, treated tumors exhibited elevated levels of effector cytokines such as interferon-γ and interleukin-17A, and mice showed evidence of broad systemic immune activation. Notably, the combined immunotherapy did not cause significant hepatic or renal toxicity, underscoring a favorable safety profile.
Conclusion B7-H4 promotes immune escape by inducing profound T-cell exhaustion in EOC. Blocking B7-H4, especially in concert with Vγ9Vδ2 γδ-T-cell adoptive transfer, re-invigorates potent antitumour immunity and offers a promising translational strategy to overcome immune resistance in ovarian-cancer patients.

PO_185

基于 uPAR 结合片段设计 ATN615-CAR 对表达 uPAR 的卵巢癌细胞的抗肿瘤效应

王丽英[2] 王亮[1] 孙蓬明[2*]
1.福建省妇产医院 2.福建省妇幼保健院

研究目的 CAR-T 细胞治疗是卵巢癌患者在复发难治阶段尝试应用的新兴治疗手段之一。卵巢癌作为女性生殖系统最恶性的癌症，传统的治疗方法并没有明显提高其生存率，因此，有必要开发卵巢癌新的治疗靶点和新的免疫治疗方法。uPAR 是一种糖基磷脂酰肌醇（GPI）锚定膜蛋白，在正常组织和卵巢癌组织中差异表达。研究表明，uPAR 上调促进肿瘤的发生、增殖、侵袭和转移，并且 uPAR 在肿瘤基质成分中也上调。CAR-T 细胞治疗血液肿瘤已取得里程碑式效果，但在以卵巢癌为代表的实体瘤上却效果不佳，提高 CAR-T 细胞治疗实体瘤的效果是研究的热点。
材料与方法 流式细胞术检测 SKOV3、ES-2、IOSE 细胞中 uPAR 的表达。分离健康志愿者外周血 T 细胞并在体外激活培养、扩增。流式细胞术检测 T 细胞表面 CD3、CD4、CD8、CD25、CD69 的表达。构建包含 uPAR 结合多肽区、CD8 铰链区与跨膜结构域、CD137 (4-1BB) 共刺激结构域以及 CD3ζ 信号结构域的 ATN615-CAR 分子，并使用慢病毒颗粒将其引入激活的 T 细胞。使用流式细胞术测定评价所得 CAR-T 细胞的转染效率。使用 LDH 试剂盒测定 ATN615-CART 对 SKOV3、ES-2、IOSE 细胞的杀伤百分比。使用酶联免疫吸附测定测量 ATN615-CART 细胞的细胞因子释放（如 IL-2、INFγ、颗粒酶 B）。

结果 ATN615-CAR T 细胞对表达高水平 uPAR 的 SKOV3、ES-2 细胞及表现出强效细胞毒性。此外，ATN615-CAR T 细胞在杀死表达低水平 uPAR 的 IOSE 细胞方面效率低下，但对高表达 UPAR 的此类细胞具有强效细胞毒性。ATN615-CAR T 细胞相比于对照-CART 细胞表现出高 IL-2、IFN-γ、颗粒酶 B 分泌。
结论 本研究结果表明 ATN615-CART 具有强大的细胞毒性和细胞因子产生，与前期设计的 ATF-CART 具有相似的功能，表明对 uPAR 阳性肿瘤细胞具有很强的抗肿瘤活性。

PO_186

Neoadjuvant Therapy in Ovarian Cancer: Emerging Paradigms and Therapeutic Frontiers

yuxuan Shi[1] Bairong Xia[2*]
1.USTC(University of Science and Technology of China)
2.Anhui Provincial Cancer Hospital

Ovarian cancer ranks as the sixth leading cause of cancer-related deaths among women, posing a significant threat to their physical and mental well-being. Known for its insidious nature, this aggressive disease often goes undetected until late stages. Under such circumstances, primary debulking surgery (PDS) often fails to accomplish optimal tumor removal while simultaneously increasing the risks of complications and fatal outcomes. The use of neoadjuvant therapy followed by intermittent tumor cytoreduction has emerged as an effective treatment for advanced ovarian cancer patients with high tumor load and poor physical status. Research on specific dosing regimens for neoadjuvant therapy has continued to gain momentum in recent years, involving the optimization of conventional chemotherapy regimens, the introduction of targeted agents and immunotherapies, and single-agent or multi-agent combination strategies. The purpose of this review is to provide a comprehensive review of these recent advances, with the aim of providing valuable references and bases for the clinical treatment of advanced ovarian cancer.

PO_187

HSPG2 在卵巢癌细胞及 PARPi 耐药通路中的表达

段拽林 闫丽隽* 冯晓宇
山西省肿瘤医院

目的 探索敲除 HSPG2 对卵巢癌细胞表型的影响及其在 PARP 抑制剂耐药中的作用机理。
方法 运用 R 语言分析 Geogebra 数据库中与 PARPi 耐药性相关的差异基因以及卵巢癌与正常组织间的表达谱数据，分析 TCGA 数据库中有关卵巢癌生存预后的 RNA 测序数据，绘制总生存期（OS）的 KM 曲线。采用 RT-qPCR 验证 HSPG2 的表达，采用 CCK-8、克隆、Transwell、划痕、流式实验等检测细胞增殖、侵袭、迁移、凋亡等功能，通过 RT-qPCR 和 Western blotting 研究 HSPG2 对与 PARP 抑制剂耐药机制相关的 ATR、SLC7A11 的表达影响。
结果 细胞表型实验结果显示 HSPG2 过表达促进 SK-OV-3 细胞的增殖、侵袭、迁移，抑制 SK-OV-3 细胞的凋亡。RT-qPCR 及 Western blotting 结果显示过表达 HSPG2 上调了 ATR、SLC7A11 的表达，敲减 HSPG2 后 ATR、SLC7A11 表达均下调，加入 ATR 后 SLC7A11 表达再次上调，但 HSPG2 表达无变化。
结论 HSPG2 上调促进卵巢癌细胞的增殖及侵袭，HSPG2 高表达影响卵巢癌患者的生存预后，通过上调 ATR 的表达促进 SLC7A11 的表达，导致 PARPi 耐药的发生。

PO_188

Multidimensional Mechanisms of SPP1 in Ovarian Cancer Progression and Clinical Application Potential

Ying Yang
Dalian Medical University

Research Objective Ovarian cancer (OC) is one of the deadliest malignancies of the female reproductive system, characterized by challenges in early diagnosis and often accompanied by extensive metastasis, which leads to a poor prognosis for patients. In recent years, secreted phosphoprotein 1 (SPP1, also known as osteopontin, OPN), an acidic secreted phosphoprotein, has been shown to possess multiple functions, including bone regeneration, angiogenesis, cell adhesion, migration, and inflammatory responses. It has been demonstrated to be closely linked to the occurrence, progression, and metastasis of various cancers. This study aims to explore the potential mechanisms of SPP1 in OC, revealing how it influences the onset and progression of OC through different biological pathways, thus providing theoretical support for the early diagnosis and targeted therapy of OC.

Materials and Methods This study utilized multiple databases and R Studio to analyze the expression of SPP1 in OC. First, differential expression analysis was performed to assess the expression of the SPP1 gene in OC, followed by comparisons of its expression levels across different FIGO stages in OC patients. Next, Kaplan-Meier (KM) survival analysis was used to evaluate the relationship between SPP1 expression levels and survival prognosis in OC patients. Finally, Gene Ontology (GO) analysis, Kyoto Encyclopedia of Genes and Genomes (KEGG) pathway enrichment analysis, and Gene Set Enrichment Analysis (GSEA) were conducted to further explore the functional role of SPP1 in OC and its potential molecular mechanisms.

Results The multifaceted role of SPP1 in OC is closely associated with its invasiveness, metastasis, and patient survival prognosis. Initially, differential expression analysis (Figure A) showed that SPP1 was significantly overexpressed in OC samples. A comparison of SPP1 expression levels across different FIGO stages revealed that SPP1 expression increased significantly as the cancer progressed, particularly in patients at stages III and IV, suggesting that SPP1 plays a crucial role in the invasiveness and metastatic potential of OC (Figure B). Next, KM survival analysis (Figure C) further confirmed the significant association between high SPP1 expression and poor prognosis in OC patients, as the overall survival of patients with high expression was significantly lower than those with low expression, indicating that SPP1 may serve as a biomarker for poor prognosis in OC. Finally, to explore the biological mechanisms of SPP1 in OC, various enrichment analyses were performed. GO enrichment analysis (Figure D) indicated that high SPP1 expression was significantly associated with pathways related to cytokine receptor activity and immune responses. KEGG analysis (Figure E) revealed that SPP1 was closely related to pathways such as "cytokine-cytokine receptor interaction," "inflammatory response," and "leukocyte-mediated immune response." GSEA analysis (Figure F) identified significant enrichment of high SPP1 expression in several key biological pathways, including "epithelial-mesenchymal transition (EMT)," "apoptosis signaling," "immune evasion," "cytokine response," "inflammatory response," and "angiogenesis." The core genes involved in these pathways are closely associated with immune responses, cytokine receptor activity, leukocyte-mediated immunity, and processes such as tumor invasion and metastasis. SPP1 may promote immune evasion within the tumor microenvironment by regulating interactions between cytokines and immune receptors, thereby enhancing the invasiveness and metastatic ability of OC cells. Furthermore, the close association between high SPP1 expression and the EMT process suggests that SPP1 may enhance OC cell migration by promoting EMT, thereby facilitating tumor spread. Additionally, SPP1 may influence inflammatory responses and angiogenesis, promoting local inflammation and the generation of new blood vessels within the tumor microenvironment, thus supporting tumor growth. These mechanisms provide important molecular evidence for the multifaceted role of SPP1 in OC.

In summary, SPP1 may promote the progression of OC through multiple mechanisms, including alterations in energy metabolism, facilitation of immune evasion, enhancement of tumor invasiveness and cell migration, as well as the development of chemoresistance.
Conclusion: This study demonstrates that high expression of SPP1 is closely associated with clinical features of OC, particularly in advanced-stage patients. SPP1 not only holds potential as an early diagnostic biomarker but may also serve as a key factor in targeted therapy, offering new therapeutic intervention targets.

PO_189

Risk Factors for Recurrence in Malignant Ovarian Germ Cell Tumors: A Retrospective Study

Yun Feng*
The First Affiliated Hospital of Zhengzhou University

Background Malignant ovarian germ cell tumors (MOGCTs) are the second most common type of ovarian malignancies, with a relatively good prognosis when diagnosed early and treated appropriately. However, a small number of patients still experience persistent or recurrent diseases, which significantly deteriorate their prognosis. Identifying the risk factors for recurrence is crucial for improving patient outcomes
Methods This retrospective study included 123 patients diagnosed with MOGCT between January 2012 and December 2024 at the First Hospital of Zhengzhou University. Data on clinical characteristics, serological markers, surgical details, and follow-up outcomes were collected. The Cox proportional hazards regression model was used to analyze risk factors for postoperative recurrence and metastasis, and the Kaplan-Meier method was employed to plot progression-free survival (PFS) curves
Results The mean age of the patients was 22.67 years, and 69.11%were nulliparous. Elevated preoperative levels of alpha-fetoprotein (AFP), beta-human chorionic gonadotropin(β-HCG), carcinoembryonic antigen (CEA), cancer antigen 125(CA125), cancer antigen 199(CA199), and HE4 were observed in 70.73%,21.95%,19.51%,71.54%,26.83%, and 21.14%of patients, respectively. Univariate and multivariate Cox regression analyses identified tumor stage, metastasis status, and preoperative CEA level as independent risk factors for postoperative recurrence and metastasis. Kaplan-Meier analysis showed significantly higher PFS rates in patients with FIGO stage I disease, no metastasis, and preoperative serum CEA<5 ng/mL compared to those with stage II or above, metastasis, and CEA≥5 ng/mL (all $P<0.05$).
Conclusions Tumor stage, metastasis status, and preoperative CEA levels are independent risk factors for postoperative recurrence and metastasis in MOGCTs. These findings can help clinicians identify high-risk patients before treatment, providing opportunities for early intervention to improve prognosis and overall treatment outcomes. Future studies should further explore the relationship between dynamic changes in CEA levels and recurrence risk.

PO_190

耐药卵巢癌细胞中 MHC-I 与泛素化关系探索

刘晓寒
河北大学临床医学院

研究目的 证实耐药卵巢癌细胞中 MHC-I 与泛素化关系。
材料与方法 通过 IC50 实验测定 SKOV3 细胞、SKOV3 DDP 细胞，了解耐药情况及浓度。通过 Western blot 实验测定 SKOV3 细胞、SKOV3 DDP 细胞中 MHC-I 的表达差异，以证实 SKOV3 细胞和 SKOV3 DDP 中 MHC-I 蛋白含量和表达水平存在差异。通过免疫共沉淀（Co-IP）证实泛素化改变在一定程度上影响 SKOV3 细胞和 SKOV3 DDP 细胞中 MHC-I 的表达；通过 Western blot 证明去泛素化后 SKOV3DDP 细胞耐药性发生改变；进行体内实验再次验证：SKOV3 DDP 组、SKOV3 DDP-泛素化组、SKOV3 DDP-去泛素化组。
结果 1.IC50 明确 SKOV3 DDP 具有耐药性；2.WB 实验证实 SKOV3 与 SKOV3 DDP 两者之间 MHC-I 表达存在差异，SKOV3 DDP 组 MHC-I 下调；3.Co-IP 证明 MHC-I 与泛素化有关，判断下调由泛素-蛋白酶体途径介导，去泛素化后再次通过 IC50 试验、WB、体内实验等验证证实可以通过去泛素化水平改变 MHC-I 的表达含量，进而验证改变 SKOV3 DDP 的耐药性；4.通过分子筛选，找出催化 MHC-I 泛素化的目的酶；5.敲除目的酶，验证其对 MHC-I 稳定性及耐药表型；5.体内实验再次验证上述结果。
结论 靶向泛素化-MHC-I 轴可以调控化疗敏感性。

PO_191

A shortcut from Ovarian cancer development to metastasis: MYC/FASN/SEMA7A, a promising theragnostic axis

Guo Feifei Yue Hua Huaijun Zhou*
Nanjing Drum Tower Hospital

Background and Aims Ovarian cancer is the leading cause of mortality among gynecologic malignancies. Trans coelomic metastasis is the most common and is responsible for the great morbidity and mortality in women with ovarian cancer. However, very little is known about the biology of trans coelomic metastasis. Here we analyzed the role of the MYC/FASN/SEMA7A axis in the progression of ovarian cancer trans coelomic metastasis.
Methods Bio-informatic analysis revealed the significant clinical importance of MYC and FASN expression in affecting survival including overall survival and progression free survival, as well as the relationship between MYC and FASN. Immunohistochemistry, real time-PCR and western blot analysis of MYC and FASN expression levels. Furthermore, flow cytometry was performed to observe the ratio of M1/M2 macrophages in tissues of ovarian cancer patients. Overexpression of MYC and FASN were generated via Lentivirus expression system in ovarian cancer cell lines. Cell proliferation, migration and invasion abilities were determined by CCK-8 assay, colony-formation assay, cell scratch assay and transwell assay, respectively. Transcriptional sequencing and mass spectrometry were used to analyze the key gene involved in trans coelomic metastasis of ovarian cancer.
Results Oncoprint for focal amplifications of MYC is 35% by CBioPortal database. Moreover, FASN was correlated positively with MYC expression. Survival analysis revealed that higher MYC and FASN mRNA levels appeared to be related to the poorer overall survival of ovarian cancer. Faster recurrence of ovarian cancer was observed in patients with higher MYC and FASN expression levels. Intriguingly, higher M2 macrophages ratio was observed in patients with metastasis and recurrence. Experimentally overexpressed MYC could promote FASN expression, as well as facilitating cell proliferation, migration and invasion abilities of ovarian cancer cell lines (SK-OV-3, OVCAR 3), reduction of MYC significantly inhibited these cell biological behaviors via activation of macrophages polarization. Furthermore, the

inhibition of FASN could also reverse the negative effect of MYC overexpression. Moreover, transcriptional sequencing and mass spectrometry showed that up-regulation of MYC/FASN dramatically promoted M2 macrophages polarization via increasing overexpression of SEMA7A.
Conclusions In the present study, we identified a novel function of MYC/FASN/SEMA7A, by promoting M2 macrophages polarization in ovarian tumor tissues, serving a critical role on the progression and trans coelomic metastasis of ovarian carcinoma. It can be speculated that targeting MYC/FASN/SEMA7A might reduce the risk of ovarian cancer development and trans coelomic metastasis. However, detailed analysis is urgently needed to uncover the underlying molecular mechanisms, as well as the related risk factors.

PO_192

Incorporating genotype information in a precise prediction model for platinum sensitivity in Epithelial Ovarian Cancer

Naiyi Du
the Fourth Hospital of Hebei Medical University

Objective Develop a predicting model that can help stratify patients with epithelial ovarian cancer (EOC) before platinum-based chemotherapy.
Methods 148 patients with pathologically confirmed EOC and with a minimum 5-year follow-up were retrospectively enrolled. Patients were classified into platinum-sensitive and platinum-resistant groups according to treatment responses. The correlation between clinical factors and drug sensitivity was evaluated using statistical tests. Approximately 700,000 single-nucleotide polymorphism (SNP) sites were assessed for association with drug sensitivity via the Genome-wide Association Study (GWAS). LASSO regression and manual selection were employed to reduce the number of variables. A predicting model based on optimized variables was constructed. The predictive ability of the model was assessed using the Kaplan-Meier curve.
Results No statistically significant association was found between clinical factors and drug sensitivity. Sixteen SNPs were preserved after the optimization. A predicting model for drug sensitivity was constructed based on those sixteen SNPs. Coefficients of the synergistic effect for each SNP were determined, and an algorithm of the Drug Sensitivity Index (DSI) was built. The DSI score can successfully distinguish the drug-sensitive or drug-resistant patients with sensitivity, specificity, positive predictive value, and accuracy of 94.7%, 83.3%, 90.8%, and 90.5%, respectively. In both the training set and validating samples, the Kaplan-Meier curve showed that the median PFS and mean OS were significantly differentiated between the predicted sensitive and resistant patients (p-value<0.001).
Conclusions A mathematical model incorporating genotype information could help predict the drug sensitivity of platinum-based chemotherapy before the treatment in EOC patients. A personal chemotherapy could be achieved based on the model.

PO_193

靶向 BRD4 的纳米 PROTAC 前药可逆转卵巢癌 PARP 抑制剂耐药

舒桐[1] 郑虹[1*] 唐东升[2] 肖海华[2]
1.北京大学肿瘤医院
2.中国科学院北京化学研究所

背景 PARP 抑制剂（PARPi）敏感性与卵巢癌患者的预后密切相关。表观遗传调控因子 BRD4 在 PARPi 耐药的卵巢癌中高表达，成为潜在治疗靶点。尽管已有研究表明抑制 BRD4 可增强 PARPi 的抗肿瘤作用，但目前常用的 BRD4 小分子抑制剂普遍存在毒性大、生物利用度低以及容易诱导耐药等局限性。蛋白质降解靶向嵌合体（PROTAC）技术作为新一代药物开发策略，可实现对“不可成药”蛋白的选择性降解。然而，目前尚无靶向 BRD4 的 PROTAC 被应用于卵巢癌的研究与治疗。本研究构建并评估了一种基于纳米递送系统的 BRD4 靶向 PROTAC 前药（BRD4-nanoPROTACs），以期探索其在 PARPi 耐药卵巢癌中的治疗潜力。
方法 合成 BRD4 靶向的 PROTAC 前药，并采用 DSPE-PEG2000 高分子材料包裹构建纳米制剂，实现对肿瘤组织的靶向递送与蓄积。在体外卵巢癌细胞模型及 PARPi 耐药的患者来源异种移植瘤（PDX）模型中，系统评估其抗肿瘤活性。
结果 1、靶点验证：TCGA 数据库分析显示，BRD4 高表达与卵巢癌患者总体生存（OS）显著负相关，但与无进展生存（PFS）无明显相关性（图 1），提示其具有作为治疗靶点的潜力。2、药物合成与表征：BRD4-PROTAC 前药通过酰胺化和硝基氯甲酸酯反应等步骤合成（图 2），质谱检测显示其实际分子量（1482.61076）与理论值（1482.61170）高度一致，证实合成成功（图 3）。电镜观察发现纳米颗粒形态规则，平均粒径约 60 nm，水合粒径为 80 nm，PDI 为 0.2，表明制剂稳定性良好。纳米包裹未降低药物毒性，同时提高了细胞内药物积聚。3、体外活性：MTT 实验显示，BRD4-PROTACs 对卵巢癌 OV8 细胞具有良好的抑制作用，其 IC_{50}为 2.7 μM（图 4）。4、体内疗效：在 PARPi 耐药 PDX 模型中，BRD4-nanoPROTACs 联合尼拉帕利显著抑制肿瘤生长，联合治疗效果优于任一单药（图5）。
结论 本研究首次构建了靶向 BRD4 的纳米 PROTAC 前药，并证实其可有效逆转卵巢癌对 PARP 抑制剂的耐药性，为 PARPi 耐药卵巢癌的精准治疗提供了新的潜在策略。

PO_194

Exploring the "Pebbled Mosaic Sign" as an Ultrasound Indicator of Ovarian Immature Teratomas

Tianyu Zhang[1] Jie Yang[1] Na Su[2*] Jiaxin Yang[1]
1.Department of Obstetrics and Gynecology, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College
2.Department of Ultrasound, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College

Objective To describe the clinical and ultrasound features of ovarian immature teratomas and assess the correlation between ultrasound features and pathological grade.
Methods This was a retrospective, single-center study including patients treated at Peking Union Medical College Hospital from June 2017 to February 2025, who were diagnosed with pure ovarian immature teratoma through surgical pathology. Clinicopathological and ultrasonographic information were retrieved from each patient's records. Ultrasonographic images were reviewed using the International Ovarian Tumor Analysis (IOTA) classification system. We introduced the terms “pebbled mosaic sign” to describe distinctive ultrasound features of ovarian immature teratomas. The correlation

between ultrasonographic features and pathological grade was assessed using the Fisher exact probability test and Spearman correlation analysis.
Results There were 30 patients diagnosed with pure ovarian immature teratoma included in this study. The median age at diagnosis was 25.2 years (range 7.0-39.0 years). Elevated serum AFP levels were observed in 56.7% of patients. Fertility-sparing surgery was performed in 96.7% of patients. Pathological grade was distributed as grade 1 (36.7%), grade 2 (36.7%), and grade 3 (26.6%), while FIGO stage was predominantly stage ⅠA (46.7%) and ⅠC (50.0%). The median maximum tumor diameter was 16.7cm (range 5.1-28.2cm). Ultrasonography revealed solid components in all tumors: 70.0% were cystic-solid and 30.0% were solid. Surgical exploration revealed tumor rupture in 77.8% of patients with ultrasonographic solid morphology. The “pebbled mosaic sign” was present in all patients. Tumor vascularity assessment showed moderate blood flow in 80.0% of cases, while 16.7% were minimal and 3.3% were highly vasculated. The proportion of solid components in the tumor correlated with the pathological grade ($p < 0.001$), and a high solid percentage may characterize a higher grade.
Conclusions The “pebbled mosaic sign” is a characteristic ultrasound feature of immature teratomas. The proportion of solid components with heterogeneous echogenicity may serve as a predictor of higher pathological grade.

PO_195

FOLR1-Targeted Membrane AIE Triggers Immunogenic Cell Death in Ovarian Cancer

Zhizai Chen Dalong Song Xiaojing Chen Shanyang He*
Guangdong Provincial People's Hospital

Abstract Ovarian cancer exhibits high expression of folate receptor 1 (FOLR1), offering a specific target for targeted therapy, while effective induction of immunogenic cell death (ICD) is critical for enhancing immunotherapeutic efficacy. This study explores how FOLR1-targeted membrane aggregation-induced emission (AIE) materials specifically trigger ICD in ovarian cancer cells, aiming to provide a novel strategy for the precise immunotherapy of ovarian cancer.
Materials and Methods The high FOLR1-expressing ovarian cancer cell line SKOV3 was used. The FOLR1-targeted membrane AIE material (FOL-TPE-PE), which shows binding affinity towards SKOV3 cells and has been confirmed to be biocompatible with no adverse effects on cellular viability through comprehensive cytotoxicity assessment, was tested for cell death induction under 660 nm laser (0.5 W/cm², 5 min) and dark conditions. Pyroptosis markers (HMGB1, CRT via immunofluorescence; Caspase-1, GSDMD via Western blot) and ICD cytokines (IL-1β, TNF-α via ELISA) were detected.
Results Our study demonstrated that FOL-TPE-PE, a novel FOLR1-targeted membrane AIE agent, induced efficient apoptosis in SKOV3 ovarian cancer cells under 660 nm laser irradiation. However, no cell-killing effect was observed in the dark condition, indicating that the mechanism of action relies solely on the photothermal effect rather than involving non-photothermal pathways.
Upon light treatment, we observed a significant increase in pyroptosis, as evidenced by elevated HMGB1/CRT translocation from the nucleus to the cell membrane, a hallmark of immunogenic cell death (ICD). Concurrently, Western blot analysis revealed upregulated expression of cleaved Caspase-1 and GSDMD, key proteins associated with the pyroptotic pathway.
To further confirm ICD induction, we employed ELISA to measure the levels of pro-inflammatory cytokines. The results showed significantly higher levels of IL-1β and TNF-α in the culture supernatants of light-treated cells compared to the dark-treated group. These findings collectively suggest that FOL-TPE-PE not only induces apoptosis but also triggers immunogenic cell death in ovarian cancer cells, providing a promising strategy for cancer therapy.
Conclusion: The FOLR1-targeted membrane AIE material FOL-TPE-PE can specifically target SKOV3 cells, and under light irradiation, it can effectively induce pyroptosis in SKOV3 cells, thereby

triggering ICD. This process is accompanied by the release of DAMPs and an increase in pro-inflammatory cytokines, which can enhance the immunogenicity of ovarian cancer cells. This study provides a new insight into improving the efficacy of ovarian cancer immunotherapy.

PO_196

Stage Ⅰ ovarian cancer with pelvic retroperitoneal Castleman disease misdiagnosed as multiple lymph nodes metastasis on imaging

Jingying Zhang[1,2] Lina Tian[1,2] Tianqi Wang[1,2] Yingmei Wang[1,2*]
1.Department of Gynecology and Obstetrics, Tianjin Medical University General Hospital
2.Tianjin Key Laboratory of Female Reproductive Health and Eugenics, Tianjin Medical University General Hospital

Castleman disease (CD), first delineated in 1954 by Castleman and Towne, predominantly manifests in the mediastinum, neck, and abdominal cavity, with less frequent involvement of the pelvic retroperitoneum. The etiology of CD remains unclear, and the absence of distinctive clinical features renders preoperative diagnosis particularly challenging. We present a case of a 61-year-old woman with stage I ovarian cancer (OC) associated with pelvic retroperitoneal CD, and the pelvic lymph nodes (LNs) showed hypervascularity on enhanced CT and MRI as well as increased FDG uptake on FDG PET/CT. These image features were similar to the LNs metastasis of OC. This case suggests that CD should be considered as a rare differential diagnosis in patients with the LNs metastasis of cancer.

PO_197

NPBWR1-2/miR-153-5p/IGFBP7 通路调控卵巢癌发生发展的机制研究

刘沙沙 安崇佑 刘彩艳 曲芃芃*
天津市中心妇产科医院

目的 探讨 lncRNA NPBWR1-2 作为 ceRNA，通过调控 miR-153-5p 介导 IGFBP7 表达，进而影响卵巢癌发生发展的分子机制。
方法 通过实时定量 PCR 方法，检测 SKOV3/lnc-NPBWR1-2 细胞系中 miR-153-5p 表达水平；应用 Western blot 实验，明确 NBPWR1-2 与 miR-153-5p 对 IGFBP7 蛋白表达的影响；采用双荧光素酶实验及挽救实验，验证 IGFBP7 与 miR-153-5p 结合靶点。
结果 在过表达 lnc-NBPWR1-2 的 SKOV3 细胞系中 miR-153-5p 表达水平明显降低（p=0.0003）。与对照组相比，NBPWR1-2 促进 IGFBP7 在蛋白水平的表达，而 miR-153-5p 抑制 IGFBP7 在蛋白水平的表达。转染 miR-153-5p mimics 会下调 IGFBP7 的内源性表达，而转染了 miR-153-5p 反义寡核苷酸后 IGFBP7 内源性表达上调。NBPWR1-2 可以通过竞争性结合 miR-153-5p，以降低 miR-153-5p 对 IGFBP7 mRNA 的降解，从而上调 IGFBP7 蛋白水平的表达。IGFBP7 3'UTR 的 332-338 位点是 miR-153-5p 的结合位点，在挽救实验中，过表达 NBPWR1-2 后 miR-153-5p 对 IGFBP7 的 332-338 位序列的结合降低，即 NBPWR1-2 可以挽救 miR-153-5p 对 IGFBP7 的抑制作用。
结论 NPBWR1-2 可通过竞争性结合 miR-153-5p,上调 IGFBP7 表达，进而影响卵巢癌的发生发展。

PO_198

硝唑尼特协同尼拉帕利通过调控细胞周期抑制卵巢癌进展的机制研究

刘臻妍[1] 杨祥群[1] 赵桃宇[2] 贾岳[1] 杨宏英[1*]
1.云南省肿瘤医院（昆明医科大学第三附属医院）2.昆明医科大学附属德宏医院

研究目的 尼拉帕利作为腺苷二磷酸核糖聚合酶（Poly ADP-ribose Polymerase）抑制剂在卵巢癌治疗中展现出显著疗效，但患者个体差异导致其疗效存在较大提升空间。因此，探索能够有效增强尼拉帕利敏感性的联合用药策略具有重要意义。硝唑尼特（Nitazoxanide, NTZ）是硝噻柳酸酰胺衍生物，作为抗微生物药物被开发使用，具有强大的抗病毒活性，在体内可达全身高浓度且无重大副作用。基于硝唑尼特多靶点作用机制及其在肿瘤治疗中的潜在价值，本研究深入探究硝唑尼特与尼拉帕利的协同效应，为提高 PARP 抑制剂的临床疗效及破解 PARP 抑制剂耐药困境提供新思路。本研究旨在系统解析硝唑尼特增敏机制，同时推动传统药物“老药新用”的临床转化，有望为改善卵巢癌患者预后及推动精准治疗提供理论依据和实践策略。

研究方法 本研究采用 A2780、SKOV3、HEY、OVCAR3 四株卵巢癌细胞进行研究：通过 CCK-8 法、克隆形成实验及 EdU 染色评价硝唑尼特和尼拉帕利两药联用对卵巢癌细胞增殖的影响；采用流式细胞术评价两药联用对卵巢癌细胞周期的影响；采用划痕实验和 Transwell 实验评价两药联用对卵巢癌细胞侵袭与迁移的影响；采用蛋白质组学测序探究两药联用对卵巢癌细胞蛋白水平的影响；构建 SKOV3 卵巢癌细胞腹腔转移瘤模型，评价两药联用对卵巢癌模型小鼠肿瘤生长的影响；采用 Western blot 实验评价两药联用抗卵巢癌的潜在作用机制。

研究结果 （1）硝唑尼特和尼拉帕利两药联用较单药更显著抑制卵巢癌细胞的增殖，并展现出协同作用；（2）两药联用较单药更显著的抑制卵巢癌细胞的侵袭和迁移，并影响 EMT 相关蛋白的表达；（3）蛋白质组学测序结果表明：两药联用处理 SKOV3、OVCAR3 两株卵巢癌细胞后，经 KEGG 和 GO 富集分析发现两药联用显著调控卵巢癌细胞周期通路；（4）体内研究结果表明：两药联用较单药更显著抑制卵巢癌腹腔转移瘤模型小鼠肿瘤的生长，并对主要脏器无明显毒性；瘤体免疫组化结果表明：两药联用显著下调 Ki-67 阳性细胞率；（6）WB 结果表明：两药联用能够改变细胞周期相关蛋白的表达。

研究结果 硝唑尼特与尼拉帕利联用显著抑制卵巢癌细胞增殖、侵袭与迁移，调控细胞周期，并产生协同效应，结合蛋白质组学结果揭示其可能通过调控细胞周期增强尼拉帕利敏感性，阻滞卵巢癌的进展。

PO_199

Mapping the Knowledge Landscape of Lymph Node Metastasis in Ovarian Cancer: A Bibliometric Analysis

Chunru Chen[1] Fuxia Li[2*]
1. Shihezi University 2. First Affiliated Hospital, Shihezi University

Background Ovarian cancer is among the most lethal gynecologic malignancies worldwide, with over 320,000 new cases and 207,000 deaths estimated in 2022. This high mortality is largely attributable to late-stage diagnosis – approximately 70% of patients present with advanced disease (FIGO III–IV). Lymphatic metastasis is a common pathway of spread, detected in ~20–40% of cases (rising to >50% in advanced-stage patients). Any regional lymph node (LN) involvement automatically upstages the cancer to at least FIGO stage III and portends worse survival, making nodal metastasis a critical prognostic factor in ovarian cancer.

Methods We conducted a bibliometric analysis of global research on ovarian cancer lymph node metastasis from 2010 to 2025. Literature records were retrieved from the Web of Science Core Collection, yielding 845 relevant publications. Bibliometric tools including VOSviewer, CiteSpace, and the Bibliometrix R-package in R were utilized to map publication trends, identify core contributors (countries, institutions, authors), and examine thematic evolution in this field. Key indicators such as publication counts, citation patterns, co-authorship networks, and keyword co-occurrence were analyzed to elucidate research hotspots and emerging topics.

Results The annual volume of publications grew steadily from 2010, peaking during 2016–2018, followed by a slight decline in recent years. Geographically, China has emerged as the dominant contributor, accounting for ~61% of all publications (501 papers), far surpassing the United States (8.5%) and other countries. Notably, all of the top ten productive institutions were Chinese universities, underscoring China's research leadership in this area. Core research themes encompassed surgical staging and lymphadenectomy (with "lymphadenectomy" and "prognosis" identified as foundational keywords), molecular mechanisms of metastasis (e.g. roles of EMT, E-cadherin, lncRNAs, and cytokines in tumor spread), and efforts to improve diagnostic accuracy. In particular, sentinel lymph node biopsy in apparent early-stage disease has gained traction as a strategy to enhance staging precision while reducing morbidity. Keyword analyses revealed a clear shift over time from descriptive clinical studies toward translational and innovative approaches. Recent years have seen surging interest in precision diagnostics and risk assessment (e.g. radiomics, statistical risk models, "sentinel lymph node" mapping), as well as immunological and targeted therapy research (with bursts in keywords like "immune microenvironment," "T cells," "dendritic cells," and "monoclonal antibody"). These trends indicate an evolving focus on integrating molecular biology and immunotherapy concepts into the study of ovarian cancer metastasis.

Conclusions This bibliometric study highlights the growing body of knowledge on lymph node metastasis in ovarian cancer and its clinical significance. The findings underscore that while our understanding of nodal spread has deepened, translating these insights into better patient outcomes remains an urgent priority. Future research should prioritize international collaboration and interdisciplinary efforts to drive innovation. Key directions include deeper exploration of the molecular underpinnings of LN metastasis, development of advanced imaging techniques and biomarkers for more precise metastasis detection, and clinical studies to optimize surgical staging (e.g. refining lymphadenectomy vs. sentinel node protocols). Additionally, there is a strong impetus to develop targeted therapies and immunotherapeutic strategies to suppress lymphatic spread and improve survival in ovarian cancer. By addressing these priorities, the global oncology community can work toward earlier diagnosis, more effective treatments, and ultimately better outcomes for patients with ovarian cancer.

PO_200

From Controversy to Precision: Clinical and Molecular Insights into Ovarian Cancer Lymph Node Metastasis

Chunru Chen[2] Fuxia Li[1*]
1.First Affiliated Hospital, Shihezi University
2. Shihezi University

Background Ovarian cancer remains the most lethal gynecologic malignancy worldwide, largely due to delayed diagnosis and widespread dissemination at the time of clinical presentation. Lymphatic metastasis, particularly involving pelvic and para-aortic lymph nodes, is a crucial prognostic determinant that significantly influences staging, therapeutic decisions, and patient survival. Traditionally, systematic lymphadenectomy has been advocated as a standard surgical procedure to ensure accurate staging and optimal tumor clearance. However, recent clinical trials have raised substantial controversy regarding the actual benefit of extensive lymph node dissection, as increased surgical morbidity does not consistently translate into improved survival outcomes. These conflicting results have led clinicians and researchers to reassess the necessity and extent of lymphadenectomy. To address this clinical ambiguity, greater emphasis is now placed on understanding the molecular underpinnings that drive lymph node metastasis and on developing precision strategies for individualized patient management.

Methods This comprehensive review synthesizes both basic and clinical research literature regarding ovarian cancer lymph node metastasis, spanning the period from 2010 to 2025. Clinically, we analyzed evidence related to FIGO staging guidelines, identified high-risk clinicopathological features predictive of lymphatic spread (e.g., histological subtype, tumor grade, stage, and disease burden), and evaluated current diagnostic methodologies, including advanced imaging modalities (CT, MRI, PET-CT) and histopathological and immunohistochemical assessments. At the molecular level, the review systematically examined critical biological pathways implicated in lymphatic dissemination, focusing on BRCA gene mutations, overexpression of key regulatory proteins such as eIF4E and Cyclin D1, dysregulated microRNA profiles, and hyperactivation of the PI3K/Akt/mTOR signaling cascade.

Results Our review identified significant molecular correlates associated with increased metastatic potential. BRCA gene mutations were consistently associated with elevated risk of nodal metastasis, attributable to genomic instability and enhanced clonal evolution. Furthermore, heightened expression of eIF4E and Cyclin D1 was demonstrated to significantly enhance cellular proliferation and invasive capabilities, facilitating lymphatic dissemination of tumor cells. Dysregulation of critical microRNAs, notably decreased expression of miR-320d, miR-4479, and miR-6763-5p, was similarly linked to increased metastatic propensity, highlighting their potential as predictive biomarkers. Concurrently, abnormal activation of the PI3K/Akt/mTOR signaling pathway was strongly correlated with metastatic behavior, promoting epithelial-mesenchymal transition (EMT), matrix degradation, and lymphangiogenesis.

Clinically, large-scale randomized controlled trials—particularly the LION and CARACO studies—have substantially reshaped contemporary surgical paradigms. These trials collectively demonstrated that routine systematic lymphadenectomy in advanced-stage ovarian cancer patients who achieved optimal cytoreduction without clinically suspicious lymphadenopathy failed to confer survival benefit and significantly increased postoperative morbidity. Consequently, current clinical practice is transitioning away from routine extensive lymphadenectomy toward individualized surgical approaches guided by precise risk stratification. The integration of advanced molecular imaging techniques and innovative diagnostic modalities, including circulating tumor DNA (ctDNA) analysis, now facilitates more accurate identification of patients who may truly benefit from targeted lymph node resection or sentinel lymph node biopsy.

Conclusions The evolving understanding of ovarian cancer lymph node metastasis, fueled by advances in both molecular biology and evidence-based clinical research, underscores a pivotal shift toward personalized and precision-driven management strategies. However, validation of molecular

biomarkers for clinical application remains an essential next step. Prospective translational studies are urgently needed to confirm the clinical utility of identified molecular signatures in predicting lymph node metastasis and guiding surgical interventions. Moreover, future research should prioritize deeper exploration of the lymphatic niche, investigating the interplay between metastatic tumor cells, lymphatic endothelium, and immune surveillance mechanisms. Such investigations promise to identify novel therapeutic targets and ultimately facilitate individualized treatment approaches that can significantly improve clinical outcomes and quality of life for patients with ovarian cancer.

PO_201

Characteristics of prematurely terminated interventional clinical trials for ovarian cancer in the ClinicalTrials. gov database

LU HONG Gaoli He Tao Zeng* Xiaoyan Zhang
Obstetrics & Gynecology Hospital of Fudan University

Background Given the high heterogeneity and low therapeutic responsiveness of ovarian cancer, there are unique challenges and barriers to the successful completion of clinical trials. However, little is known about the status of trial completion and termination in this field. This study aimed to evaluate the landscape of completed and prematurely terminated trials in ovarian cancer and to assess the reasons and potential predictors associated with trial termination.
Methods We conducted a cross-sectional analysis of interventional clinical trials for ovarian cancer registered on ClinicalTrials.gov between 2003 and 2022. Trial characteristics were compared between terminated and completed trials using theχ2 test or Fisher's exact test. The associations with termination were determined by logistic regression. The multivariable logistic regression model was assessed using a receiver operating characteristic curve and the Hosmer–Lemeshow goodness-of-fit test.
Results A total of 1420 interventional clinical trials for ovarian cancer were included, of which 294 (20.70%) were prematurely terminated. Terminations were more frequent in drug/biological (89.46%), phase 2 (57.14%), parallel (34.01%), and single-center (41.84%) trials and had a larger median planned sample size (median 56, IQR 36-100). A small portion of trials were terminated due to futility of efficacy (19.39%) and safety issues (6.80%). Reasons related to conduct or logistical issues accounted for 58.50%, and the reasons for 15.31% of the terminations were unknown. Phase 2, a larger planned sample size and parallel designs were independently associated with a higher risk of termination, whereasmulticenter trials showed a lower risk.
Conclusions The termination rate of interventional clinical trials for ovarian cancer was high, mainly due to conduct-related or logistical issues rather than efficacy or safety concerns.The planned sample size, trial phase, number of centers and interventional group design were associated with termination. The reasons and predictors for termination should be considered and addressed during trial design and planning according to the type of cancer, thereby increasing the likelihood of trial completion and facilitating the best available care for patients.

PO_202

干扰素驱动的肿瘤微环境生态位抑制卵巢癌免疫微环境及治疗反应

冯晨钊 吴迪 黄美娟 李浩 马丁 陈刚 孙朝阳*
华中科技大学同济医学院附属同济医院

研究目的 卵巢癌（Ovarian cancer, OC）仍是致死率最高的妇科恶性肿瘤。干扰素（Interferons, IFNs）在塑造肿瘤微环境（Tumor Microenvironment, TME）中发挥关键作用。然而，IFNs 在 OC 中的功能性影响及其临床意义尚未被全面解析。本研究旨在系统性刻画 OC 中的 TME 生态位，探索 IFN 反应状态的特征、机制以及其在免疫治疗与靶向治疗中的潜在作用。
材料与方法 本研究结合 120 例多组学单细胞测序分析，识别并定义了卵巢癌 TME 中的特定生态位。研究进一步利用 Jurkat TCR 报告系统和构建的嵌合受体 T 细胞（CAR-T）模型，评估 IFN-α 对 T 细胞分化及毒杀功能的影响，并通过 IFN 受体抑制剂 Anifrolumab 验证其可逆性。动物模型中评估 Anifrolumab 与 PARP 抑制剂（PARPi）及 PD-1 抗体的联用效果，并结合长读长单细胞测序揭示 IFN 反应细胞中的可变剪接特征。最后，构建 IFN 反应特征评分，用于预测患者生存及免疫治疗反应。
结果 本研究发现卵巢癌 TME 中存在一种横跨多个细胞谱系的 I 型干扰素响应生态位，特别富集于同源重组修复缺陷（HRD）患者，且与 PARP 抑制剂反应相关。该生态位具有免疫检查点基因表达普遍上调的特征，并富含一种非传统的 CD8+ T 细胞分化状态，该状态表现为 TCR 克隆扩增能力较低。功能实验表明，IFN-α 降低 T 细胞的分化和细胞毒性功能，并可通过 Anifrolumab 逆转。在小鼠体内，Anifrolumab 能增强 PARPi 和 PD-1 抗体的抗肿瘤效果，且这一协同效应不依赖 Brca 突变状态。长读长单细胞测序揭示，IFN 响应细胞中存在独特的可变剪接标志。最终构建了 IFN 响应特征集以预测患者生存及免疫治疗疗效。
结果 本研究揭示了干扰素在卵巢癌 TME 中的有害作用，指出 IFN 响应生态位可作为治疗靶点及疗效预测标志。Anifrolumab 作为 IFN 通路抑制剂，显示出协同增强 PARPi 和免疫治疗效果的潜力，不受 Brca 状态限制。IFN 相关的剪接和免疫状态变化可能是影响治疗反应的重要机制。上述发现为 OC 免疫治疗策略的优化提供了新的理论依据与临床方向。

PO_203

Clinical features and prognostic factors of ovarian cancer brain metastases: A retrospective, multicenter study of 129 cases

Qiyue Chao
The First Affiliated Hospital of USTC Anhui Provincial Hospital

Background and Purpose Ovarian cancer brain metastases (OCBM) are rare and have poor prognosis, with limited clinical guidelines. This study aims to identify prognostic factors and optimal treatment strategies for OCBM patients.
Patients and Methods We conducted a multicenter retrospective analysis of OCBM patients from 12 hospitals in China, spanning from May 2010 to May 2022. The primary endpoints were overall survival (OS) and brain metastasis-specific survival (BMSS). Kaplan-Meier and Cox regression analyses were used to assess treatment outcomes and identify risk prognostic factors.
Results A total of 129 OCBM patients were included. The median interval from ovarian cancer diagnosis to brain metastasis (BM) was 25.7 months (range: 0-102.9). Intracranial hypertension was the most common primary symptom reported in 64 (49.6%) patients, followed by paralysis in 23 (17.8%) patients. The cerebellum and brainstem were the most frequent sites of BM (36 patients, 27.9%),

followed by the frontal lobe (27 patients, 20.9%). For BMSS-specific analysis, multiple BM lesions (HR 2.060, 95% CI: 1.308-3.244, P=0.002), intracranial hypertension (HR 1.765, 95% CI: 1.049-2.968, P=0.032), and relapse lines before BM diagnosis (HR 2.060, 95% CI: 1.308-3.244, P=0.002) as independent factors associated with poorer BMSS. Subgroup Kaplan-Meier analysis showed that patients with a single BM lesion had significantly better outcomes with stereotactic radiosurgery (SRS) compared to those with multiple lesions.
Conclusion This study provides evidence that patients with a single BM lesion may benefit from SRS, whereas those with multiple lesions require more tailored treatment strategies.

PO_204

Association between pre-diagnosis and post-diagnosis low-fat diets and ovarian cancer survival: evidence from a prospective cohort study

Ying Qin
1.Shengjing Hospital of China Medical University

Background Although low-fat diets (LFDs) have established benefits in terms of cardiovascular disease and certain cancer survival, there is little evidence suggesting a link between LFDs and ovarian cancer (OC) survival. To fill this gap, we comprehensively investigated the associations of LFDs, not only pre-diagnosis and post-diagnosis, but also the change from pre-diagnosis to post-diagnosis, with OC survival.
Methods A total of 560 patients with OC were included in the study, and their dietary intake was assessed using a reliable 111-item food frequency questionnaire. Overall, unhealthy, and healthy LFD scores were calculated based on the percentage of energy as total and subtypes of carbohydrate, fat, and protein. Cox proportional hazards regression models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for the association between pre-diagnosis and post-diagnosis LFD, including changes from pre-diagnosis to post-diagnosis, and overall survival (OS).
Results During the follow-up period (median: 44.40 months, interquartile range: 26.97-61.37 months), 211 patients with OC died. High pre-diagnosis overall LFD score (HRtertile3 vs tertile1 = 0.63, 95%CI = 0.43-0.90) and healthy LFD score (HRtertile3 vs tertile1 = 0.61, 95%CI = 0.44-0.85) were associated with better OS. Compared with those who maintained stable scores (change±15%), patients whose overall LFD scores decreased (>30%) from pre-diagnosis to post-diagnosis had worse OS (HR = 1.66, 95%CI = 1.11-2.47). Moreover, compared with patients maintaining persistently low scores, patients whose overall LFD scores changed from moderate pre-diagnosis to moderate post-diagnosis scores (HR = 0.39, 95%CI = 0.21-0.72) and from high pre-diagnosis to high post-diagnosis scores (HR = 0.52, 95%CI = 0.30-0.87) had better OS.
Conclusions Our research results indicated that adherence to LFD may be beneficial to the prognosis of OC. These results may provide dietary recommendations for patients with OC, help them formulate personalized dietary plans, and offer new research directions for further studies.

PO_205

SLC44A4 激活 NF-κB 旁通路改变卵巢癌脂代谢减少铁死亡促进癌症发展

李恬 宋克娟*
The Affiliated Hospital of Qingdao University

目的 本研究旨在探讨过表达 SLC44A4 后对卵巢癌细胞生物行为的影响以及其具体机制。

方法 通过 CCK-8 、 EdU 、集落形成、伤口愈合和 Transwell 测定测定 SLC44A4 对 OC 细胞增殖、迁移和侵袭的功能，采用异种移植动物模型评估体内肿瘤生长。靶向脂质组学以及油红染色检查脂质代谢的变化。ROS,BODIPY-C11 染色、细胞亚铁测定、MDA 和 GSH 水平以及 western blotting 评估 SLC44A4 抵抗铁死亡。采用 western blotting 检测 SLC44A4 对非经典 NF-κB 通路信号的影响。最后，通过 CCK-8 检测 SLC44A4 对铁死亡诱导剂 Erastin 敏感性的影响。
结果 SLC44A4 在体内外促进卵巢癌细胞的增殖和迁移，同时改变了细胞的脂代谢，减少了铁死亡，激发了非经典 NF-κB 通路信号，并且降低了对 Erastin 的敏感性。
结果 我们的研究表明 SLC44A4 通过激活非经典 NF-κB 通路信号，改变了细胞的脂代谢以减少脂质过氧化，降低了细胞铁死亡，并且影响对 Erastin 的敏感性，表明 SLC44A4 可能作为铁死亡的目标靶点，为治疗卵巢癌提出一种新的治疗策略和方法。

PO_206

数字病理图像分析在卵巢微乳头亚型交界性浆液性肿瘤中的应用价值

徐迅[1] 孟黄洋[2] 张林[2] 袁琳[2] 程文俊[2*]
1.南京医科大学第一附属医院妇产科学系
2.南京医科大学第一附属医院 江苏省人民医院 妇科

目的 探究数字病理图像定量分析在卵巢微乳头亚型交界性浆液性肿瘤中的潜在应用价值。
方法 回顾性分析入 2012 年 10 月至 2023 年 11 月在南京医科大学第一附属医院接受手术治疗的 366 例卵巢交界性肿瘤临床与病理资料，对 83 例不同恶性程度的卵巢浆液性肿瘤应用 Qupath 数字图像分析，并进行多尺度量化病理特征提取。通过单因素及多因素 COX 回归模型，探索与卵巢微乳头亚型交界性浆液性肿瘤预后相关的危险因素。
结果 （1）卵巢微乳头亚型交界性浆液性肿瘤患者的血清 CA125 水平显著高于非微乳头亚型组（P<0.001），发生双侧肿瘤的患者占比更高（P<0.001），病理合并浸润性种植比例更高（P<0.001），临床病理分期更晚（P<0.001），具有更高的复发率（P=0.001）。（2）通过数字图像分析软件 QuPath 进行数字病理定量分析显示，微乳头亚型交界性浆液性肿瘤展现出独特的细胞形态学特征。微乳头亚型卵巢交界性浆液性肿瘤的核质比显著高于非微乳头亚型（48.57% VS 40.74%，P<0.05），且细胞核质比随肿瘤的恶性程度增加而上升。（3）单因素 COX 分析提示核质比>0.50、进行保留生育功能手术与复发显著相关。多因素 COX 分析证实核质比升高为独立危险因素（HR=7.153，P=0.005）。
结论 卵巢微乳头亚型交界性浆液性肿瘤表现出更具侵袭性的临床特征，核质比升高在判断其复发中具有潜在应用价值。

PO_207

TMEM30A-Gli1 通路参与卵巢高级别浆液性癌患者 PARPi 耐药及免疫逃逸

陈佳钰 尹婕 金滢 潘凌亚*
北京协和医院

目的 在真实世界中探究聚腺苷二磷酸核糖聚合酶抑制剂（Poly ADP-ribose polymerase inhibitor，PARPi）作为一线维持治疗的疗效，探究个体疗效差异。研究 TMEM30A-Gli1 通路是否可导致 PARPi 耐药及潜在耐药机制。探索 TMEM30A-Gli1 与肿瘤免疫微环境和免疫逃逸的关系。
方法 收集 2015 年 1 月 1 日 - 2023 年 12 月 12 日北京协和医院妇科肿瘤中心就诊并治疗的、病理诊断为晚期卵巢高级别浆液性癌（High-grade serous ovarian cancer,HGSOC）的患者资料。比较 BRCA1 胚系突变（Germline BRCA1 mutation，gBRCA1mut），BRCA2 胚系突变（Germline BRCA2 mutation，gBRCA2mut）及同源重组修复通路其他胚系突变（Germline mutation of Homologous recombination repair pathway，gHRRmut）患者的临床基线及预后差异，明确 PAPRi 疗效异质性。进而通过 RNA 测序和免疫组化染色将患者分为 TMEM30A 高表达和低表达两组人群，分别比较两组患者临床资料、PARPi 反应性及预后的差异，明确 TMEM30A 与 PAPRi 耐药和不良预后的关系。根据前期研究基础，TMEM30A-Gli1 通路与铂耐药相关。本文继续在 RNA 及蛋白水平探究 TMEM30A-Gli1 通路导致 PARPi 耐药的机制。最后在癌症基因图谱数据库中，利用 HGSOC 临床及测序数据，分析 TMEM30A-Gli1 通路对免疫治疗反应性及对免疫微环境的影响，并在患者的 RNA 和蛋白水平进行验证。
结果 在 HGSOC 患者中，PARPi 一线维持治疗疗效有明显异质性，gBRCA2mut 患者的无进展生存期显著优于 gBRCA1mut 和其他 gHRRmut。RNA 测序将 29 例患者分为 TMEM30A 高表达（n=15）和 TMEM30A 低表达组（n=14），免疫组化将另 47 例患者分为 TMEM30A 阳性组（n=20）和 TMEM30A 阴性组（n=27）。TMEM30A 高表达组/阳性组患者肿瘤负荷更重，更多的患者出现上腹腔病变和腹膜弥漫性病变，与铂耐药复发相关，PAPRi 一线维持用药时间、无病生存、总生存以及复发后至首次后续治疗时间显著缩短。TMEM30A-Gli1 通路可以上调包括 BRCA1、RAD51 在内的 HRR 通路重要分子的表达水平，导致 PARPi 耐药。TMEM30A-Gli1 可以上调免疫抑制相关分子的表达，促进 CD8+T 细胞转化为耗竭状态，从而导致免疫逃逸。
结论 PARPi 疗效有显著个体化差异。TMEM30A 高表达与 PARPi 耐药以及免疫逃逸相关，是预后不良指标，可作为未来治疗的潜在靶点。

PO_208

卵巢癌中铁死亡的分子调控网络与靶向治疗策略

陈影影 郭瑞霞*
郑州大学第一附属医院

卵巢癌作为女性生殖系统致死率最高的恶性肿瘤之一，其耐药性和转移特性仍是临床治疗的核心挑战。铁死亡作为一种铁依赖性脂质过氧化驱动的调节性细胞死亡方式，近年来在卵巢癌研究中展现出显著的治疗潜力。基础研究揭示 System Xc^--GSH-GPX4 轴的异常激活是卵巢癌细胞抵抗铁死亡的关键机制。在治疗策略上，铁死亡诱导剂与传统化疗的协同效应显著，靶向 GPX4 的小分子抑制剂 RSL3 已进入 I 期临床试验，初步数据显示其在 BRCA1 缺陷型卵巢癌中客观缓解率达 28%。然而，铁死亡逃逸机制和肿瘤微环境干扰仍是当前瓶颈。未来研究需聚焦多组学指导的精准分层治疗、靶向肿瘤微环境的联合策略，以及类器官药敏模型的个体化应用。随着溶瘤病毒、纳米递送系统等新型平台的引入，铁死亡靶向治疗有望成为卵巢癌综合治疗的重要组成部分，为克服化疗耐药和转移提供新范式。

PO_209

Beyond AJCC Staging: A Socioeconomic-Clinical Nomogram for Predicting 10-Year Survival in Epithelial Ovarian Cancer

Jing Zhao Shangdan Xie Haiyan Zhu*
Shanghai First Maternity and Infant Hospital

Objective Epithelial ovarian cancer (EOC) accounts for 90% of advanced malignancies. Although therapeutic advances, such as platinum-based paclitaxel chemotherapy and anti-angiogenic drugs, have significantly improved the 5-year survival of EOC patients, it remains unclear whether these treatments effectively promote long-term survival (currently exceeding 10 years). This study aimed to analyze the 10-year survival rate and annual trends of EOC patients, focusing on key treatment milestones such as paclitaxel and bevacizumab, to confirm that treatment advances have no impact on long-term survival in EOC. Furthermore, the study identified independent risk factors for long-term survival in EOC and constructed a nomogram model to quantitatively predict the 10-year survival probability of cancer patients.

Methods Using the SEER database, we collected data from 30,924 EOC patients diagnosed between 2004 and 2015, with follow-up data extending to December 2021. These patients were randomly divided into a training cohort (70%) and a validation cohort (30%) to facilitate model development and verification. We calculated age-adjusted overall survival (OS) and cancer-specific survival (CSS) rates, and used Joinpoint software to analyze survival trends and their annual percentage changes over time. A nomogram was constructed by integrating key clinicopathological variables (such as tumor grade, stage, and treatment modalities) and socioeconomic factors (including race, income, and residence). The performance of this nomogram was validated using the C-index, calibration curves, and decision curve analysis (DCA) in both cohorts to ensure its reliability and predictive accuracy.

Results Age-adjusted 5-year OS rates showed periodic improvements: a significant increase from 1992 to 1994 (APC = 7.23%), a steady increase from 1994 to 2010 (APC = 0.42%), and another significant improvement from 2010 to 2014 (APC = 2.97%). The 5-year CSS rate showed similar trends, with significant improvements from 1992 to 1994 (APC = 5.80%) and 2008 to 2014 (APC = 2.21%). However, improvements in long-term survival were limited, with 10-year OS increasing by only 0.59% annually, and no statistically significant increase in 10-year CSS.

Of the 30,924 patients, 16.76% achieved ≥10-year long-term survival. These patients were primarily young, white, and married. 72.4% had unilateral tumors, 52.7% were diagnosed at stage I, 30.6% were G3 differentiated, and 26.2% were AJCC stage III. Seventeen predictive factors were screened using LASSO. Multivariate Cox analysis identified five independent predictors of OS (race, income, etc.) and six factors of CSS (age, marital status, etc.). The nomogram constructed based on this had better predictive efficiency than the AJCC staging system. The C-index of OS in the training group reached 0.712, and that in the validation group was 0.705. The calibration curve and DCA both confirmed its stability and could effectively distinguish survival differences among low-, intermediate-, and high-risk groups.

Conclusion Although advances in ovarian cancer treatment have significantly improved 5-year OS and CSS, long-term survival remains largely unaffected by treatment. Long-term survivors even include those with poor prognostic features such as advanced and poorly differentiated disease, suggesting that long-term survival is regulated by multiple factors. We investigated the clinical characteristics of this subgroup of long-term EOC survivors and proposed a multifactorial nomogram for predicting long-term survival in ovarian cancer. This nomogram outperforms traditional staging methods and provides clinicians with a more personalized approach to patient assessment.

PO_210

基于血检参数、影像学检查构建卵巢交界性肿瘤早期诊断模型诊断价值

王子潇
新疆医科大学第一附属医院

交界性卵巢肿瘤 （Borderline Ovarian Tumors，BOT） 是一种特殊类型的卵巢上皮肿瘤，介于良性肿瘤和恶性肿瘤之间，在组织学上定义为无基质浸润的非典型上皮增生，恶性潜能较低。BOT 约占上皮性卵巢肿瘤的 10%-20%，代表一种普遍的妇科疾病，生长缓慢，然而它们有可能转移到远处并最终发展为浸润性癌症。BOT 包括六种组织学亚型（浆液性、粘液性、浆液性、子宫内膜样、透明细胞、Brenner 型），其中浆液性和粘液性是最常见的。由于早期没有明显的临床症状或体征，且缺乏特定的筛查方法，大多数患者通常通过体格检查或出现腹痛和腹胀等症状来识别。因此，该病的早期发现和治疗仍然是当前的研究重点。目前，BOT 手术的术前临床诊断通常涉及使用实验室检查和综合成像方式。超声技术仍然是最常用的早期筛查和辅助方法;然而，它对 BOT 的诊断准确率仅限于 60%-70%。肿瘤浸润可能与超声检测到的病灶数量、阻力指数值和血流信号有关，从而在一定程度上为肿瘤预后提供一定的指导。CT 成像可以有效地描绘卵巢肿瘤的实性成分、范围和形态特征，而 MRI 为可视化软组织提供了卓越的分辨率、多个定量参数和多序列成像能力。这两种技术都广泛用于评估肿瘤形态和辅助组织分类;然而，它们的诊断效用仍然受到一些限制。预测模型现已被广泛接受为帮助评估许多恶性肿瘤早期筛查、预后和临床决策的可靠工具。作为一种具有众多优点的评估工具，联合血检参数的预测模型能够协助首诊医生给予卵巢肿瘤早期风险评估，根据计算出的恶性概率将患者进行风险分层，随后制定出个体化的诊疗和管理方案，实现合理配置医疗资源的同时，达到患者利益最大化。本综述将研究并探索各模型优劣及效能评价，以期为早期交界性肿瘤患者探索更个体化治疗方案从而减轻社会负担，改善预后。

PO_211

巨噬细胞 BRD4 在卵巢癌中的潜在作用

王可竞 张瑜*
中南大学湘雅医院

巨噬细胞在对维持卵巢癌免疫抑制微环境起着重要作用，BRD4作为重要的表观遗传调节因子，影响了巨噬细胞的功能与状态，进而在卵巢癌的发展与治疗中发挥着重要作用。研究发现铂类药物增加了 M2，这与肿瘤细胞诱导的 STAT 3 活化增加以及 STAT1、STAT6 的活化减少相关。抑制 BRD4 被证实能够影响巨噬细胞 STAT3、6 等 STAT 家族的活化。同时，已有临床实验证实抑制卵巢癌中巨噬细胞上的 CD 47-SIRPα 互作通路能提高铂耐药患者疗效。BRD4 通过 HIF-1α 影响巨噬细胞极化， HIF-1α 也可能参与 TAM 细胞分泌的外泌体 miR-223 的产生，进而促进卵巢癌的铂耐药。

此外抑制 BRD4 导致癌细胞 HR 减弱，提高 PARPi 疗效。PARPi 可导致巨噬细胞中 HIF-1 α 耗竭和稳定性降低，巨噬细胞中 HIF-1 α 和 HIF-2 α 的缺乏会损害趋化性受体的表达以及迁移和浸润，并使得其向 M1 极化。PARPi 通过 STING 依赖性或非依赖性途径增加 Th 1 相关细胞因子 IFN-γ，抑制 Th 2 相关细胞因子如 IL-4、IL-6，这两种途径最终都增加了 CD 40+抗肿瘤巨噬细胞的比例。STING 激动剂可以将巨噬细胞从 M2 向 M1 分化，克服卵巢癌中 PARPi 耐药。抑制 BRD4 则被证明能够抑制 STING 通路，影响巨噬细细胞功能。奥拉帕尼处理来自卵巢癌小鼠的腹膜巨噬细胞激活了 STAT 3，可能促进了其耐药的产生。PARPi 还通过调节 HMGB1 调节巨噬细胞表型极化。HMGB1 在与晚期糖基化产物受体相互作用时将极化信号转移至 M1 表型，在与 C1q 补体相互作用时将极化信号转移至 M2 表型。而巨

噬细胞的中 BRD4 在不同领域的研究中都被证实能够影响上述通路。因此，在卵巢癌背景下，BRD4 与 PARPi 对于巨噬细胞的作用机制可能对卵巢癌相关治疗有着积极意义，但仍有待进一步探究。

此外，BRD4 抑制剂能够提高肿瘤的 TMB，提升 ICB 疗效。但研究显示 BRD4 抑制剂处理的小鼠 DC 和巨噬细胞的 PD-L1 显著降低。BRD4 与 ICB 联合应用的具体效果与机制仍待研究。综上，对于致力提升卵巢癌现有疗效的研究者而言，BRD4 仍是值得一个值得期待与研究的对象。

PO_212

基于机器学习的卵巢低级别浆液性癌生存预测模型的构建

张正茂* 赵兰兰 崔旭 靳晶 刘瑞婷 王红霞
河北医科大学第四医院

目的 卵巢低级别浆液性癌（LGSOC)作为卵巢癌中较为罕见的亚型，目前针对该疾病的预后影响因素尚未完全阐明，且生存预后评估缺乏系统性的研究。本研究通过机器学习研究方法深入分析 LGSOC 患者的临床特征及其预后相关因素，建立 LGSOC 个体化 Nomogram 临床预测模型,实现患者生存预后的量化评估，为患者提供个体化治疗策略及预后管理方案。

方法 本研究通过提取 SEER 数据库中 2010-2019 年确诊的 LGSOC 患者临床病例信息，经过纳入排除标准筛选出 1838 例患者，总结 1838 例患者的临床特征。根据 7:3 的比例随机划分为训练集（n=1285 例）和验证集（n=553 例）。对训练集的患者临床特征进行单多因素 COX 回归分析及 Boruta 算法进行特征选择分析，筛选出关于 LGSOC 预后的独立预测因素，并基于上述结果建立列线图（Nomogram）预测模型，预测患者 3 年、5 年、8 年总生存期 OS 概率，并使用内部验证集数据进行内部验证。同时收集河北医科大学第四医院 2015-2023 年诊治的 75 例 LGSOC 患者临床病例资料及随访信息，进行统计学分析，分析我院患者临床特征及复发因素相关影响，并使用我院数据对建模模型进行外部验证。

结果

1.通过回归分析及 Boruta 算法分析可得出年龄、婚姻状态、CA125 水平、FIGO 分期、区域淋巴结检查、术后肿瘤残余、化疗、区域淋巴结阳性是 LGSOC 患者预后的独立因素。

2.建立 LGSOC 患者 3 年、5 年、8 年总生存期 OS 的预测模型，该模型训练集和内部验证集的 C 指数分别为 0.711、0.742，训练集和内部验证集 3、5、8 年 OS 的 AUC 值分别为 0.747、0.761、0.760 及 0.715、0.750、0.745。外部验证集的 C 指数为 0.815。外部验证集 3、5、8 年的 AUC 值分别为 0.836、0.847、0.893。

3.通过对我院 75 例患者进行回归分析，结果显示：淋巴结转移可能是 LGSOC 复发相关独立预测因素。

结果 采用列线图 Nomogram 预测模型可实现 LGSOC 患者的生存预后情况的量化评估，此模型具有较好的临床应用价值。年龄、婚姻状态、CA125 水平、FIGO 分期、区域淋巴结检查、术后肿瘤残余、化疗、区域淋巴结阳性是 LGSOC 预后独立预测因素。LGSOC 复发相关独立因素可能是淋巴结转移。

PO_213

KELIM 评分对初治和复发晚期上皮性卵巢癌患者治疗决策及预后的影响

张正茂* 崔旭 赵兰兰 靳晶 刘瑞婷 王红霞
河北医科大学第四医院

目的 本研究旨在探讨 KELIM 评分对晚期上皮性卵巢癌患者治疗决策及预后的影响，重点分析 KELIM 评分对初治的 PDS 和 IDS 患者、复发患者预后评价中的预测价值，以及其与 BRCA 基因状态的相关性。

方法 采用回顾性队列研究设计，纳入河北医科大学第四医院 2015 年 1 月至 2023 年 12 月 FIGO III-IV 期上皮性卵巢癌患者共 689 例。分组设计：PDS 组 vs IDS 组：按病理类型亚组分析，组内以 KELIM 评分分层（≥1.0 为高，<1.0 为低）；复发患者组：分析 KELIM 评分与复发后无进展生存期（PFS）、无铂间期（PFI）及铂耐药复发的关联；BRCA 突变组 vs BRCA 野生型组：分析 KELIM 评分在两组中的分布差异及其对预后.的影响。使用 SPSS 25.0 进行统计分析，P<0.05 为显著。

结果

1. KELIM 评分在 PDS、IDS 初始治疗中的预后分析显示：1) KELIM 高评分组（≥1.0）中位 PFS 与 PFI 显著优于低评分组（<1.0）（PDS 组：PFS 31.5 vs 22.1 月，PFI 26.0 vs 16.8 月；IDS 组：PFS 27.1 vs 18.8 月，PFI 24.7 vs 12.6 月；均 P<0.05）。2) KELIM≥1.0，IDS 患者的 R0 切除率可达 81.6%，KELIM<1.0，R0 切除率可达 61.5%，（P<0.05）。3) 组织学亚型分析显示：在浆液性癌中，KELIM≥1.0 患者的 PFS 为 53.0 个月，KELIM<1.0 的患者的 PFS 为 28.0 个月（P<0.001）；在非浆液性癌中，由于样本量小，KELIM 与 PFS 相关性尚不明确（P>0.05）。

2. 对复发患者，研究结果显示：KELIM<1.0 对铂耐药复发具有预测价值（P<0.05）；第一次复发后的 KELIM≥1.0，提示

至再次复发的 PFS（HR 0.598，95％CI 0.368-0.970，P<0.05）、PFI（HR 0.572，95％CI 0.353-0.929，P<0.05）显著延长。

3. BRCA 突变患者中，高 KELIM 评分者中位 PFS 显著延长（21.2 vs 18.0 月，P=0.024）；BRCA 野生型患者中，KELIM 评分对 PFS 无显著影响（26.3 vs 22.6 月，P=0.300）。

结果

1. KELIM 评分是上皮性卵巢癌患者 PFS 与 PFI 的独立预测因子，IDS 患者 KELIM 评分≥1.0，R0 切除率较高。KELIM 评分与晚期非浆液性卵巢癌的预后相关性尚不明确。

2.在复发患者中，KELIM 评分是患者 PFS、PFI 的独立预测因素，KELIM<1.0 提示铂耐药复发。

3.根据 BRCA 突变进行分层，BRCA 突变患者中，高 KELIM 评分患者中位 PFS 显著延长，而 BRCA 野生型患者中，KELIM 评分对 PFS 无显著影响。

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卡铂联合紫杉醇对比奈达铂联合紫杉醇对晚期上皮性卵巢癌的预后影响

张正茂* 靳晶 刘瑞婷 王红霞 崔旭 赵兰兰
河北医科大学第四医院

目的 本研究旨在探讨在初次肿瘤细胞减灭术（PDS）与中间型肿瘤细胞减灭术（IDS）两种不同治疗策略下，卡铂联合紫杉醇（CP）与奈达铂联合紫杉醇（NP）对晚期上皮性卵巢癌（AEOC）患者的疗效差异及预后影响，并分析两种化疗方案的安全性。

方法 回顾性纳入 2019 年 1 月 1 日至 2023 年 12 月 31 日在河北医科大学第四医院妇科收治的 437 例 AEOC 患者，根据治疗策略分为 PDS 队列（286 例）和 IDS 队列（151 例），每队列进一步按化疗方案分为CP 组与 NP 组。收集患者临床病理资料：年龄、基础疾病、术前血清 CA125 水平、临床分期、病理类型、手术减瘤程度、无进展生存期（PFS）、无铂间期（PFI）、KELIM 评分及Ⅲ-Ⅳ级不良事件等，针对 IDS 人群，还要通过 RECIST 1.1 标准，评估新辅助化疗后实体瘤的影像学疗效。

结果

1. 疗效及预后比较

1) PDS 队列

KELIM 评分：CP 组和 NP 组 KELIM 评分≥1.0 者占比为 50.3% vs. 49.6%，P>0.05，化疗敏感性无差异。

PFI：CP 组和 NP 组中位 PFI 分别为 21.1 个月与 18.2 个月，无明显差异（P>0.05）。

PFS：CP 组中位 PFS 为 36.03 个月，NP 组为 28.26 个月，P=0.098，两组之间无统计学差异。
2) IDS 队列
KELIM 评分：CP 组和 NP 组 KELIM 评分≥1.0 者占比为 38.5% vs. 32.7%，P>0.05，两组化疗敏感性无差异。
客观缓解率（ORR）及疾病控制率（DCR）：CP 组 ORR 显著高于 NP 组（81.8% vs. 65.4%，P=0.028），但 DCR 无差异（95.5% vs. 92.3%，P=0.438）。
PFI：NP 组中位 PFI 显著长于 CP 组（17.6 个月 vs. 12.9 个月，P=0.013）。
PFS：NP 组中位 PFS 为 25.83 个月，CP 组中位 PFS 为 21.07 个月，P=0.156，未达统计学差异。
2. 安全性分析
骨髓抑制相关不良事件中，NP 组中性粒细胞减少的发生率（18.0% vs. 27.8%）显著低于 CP 组（P=0.018），血小板减少的发生率（7.9% vs. 15.4%）显著低于 NP 组（P=0.018）；其他不良事件组间无差异。

结果

1. 在 PDS 队列中，CP 与 NP 方案的长期生存结局（PFI、PFS）及化疗敏感性无显著差异；在 IDS 队列中，CP 方案的短期客观缓解率更优，但 NP 方案可能通过延长 PFI 为后续治疗创造机会。
2. 相较传统 CP 方案，NP 方案在保持临床疗效的同时，其血液学毒性特征更为温和。

PO_215

内皮 KLK8 缺失通过重塑肿瘤微环境加速卵巢癌进展

龙晶 温新新 张瑜*
中南大学湘雅医院

研究目的 卵巢癌（OC）是全球最致命的妇科癌症之一。内皮细胞驱动血管生成、重塑肿瘤微环境并促进转移，是卵巢癌进展中不可忽视的重要方面。组织激肽释放酶相关肽酶(KLKs)广泛参与机体生理与病理过程，既往许多研究报道 KLKs 在特定的血管内皮细胞中表达并发挥一定作用，而有关卵巢癌时血管内皮 KLKs 的变化及其与卵巢癌进展的关系目前尚未见报道。我们发现 KLK8 是人原代卵巢微血管内皮细胞（HOMECs）在卵巢癌细胞刺激下变化最为显著的 KLKs。本研究旨在探究内皮 KLK8 对卵巢癌的影响及其机制。

材料与方法

1. 构建了 KLK8 稳定过表达/敲降的 HOMECs，探究了血管内皮 KLK8 对内皮细胞及 OVCAR-8，CAOV3，SKOV3 卵巢癌细胞功能的影响。
2. 将 KLK8-loxp 的 C57BL/6 小鼠与 Tie2-Cre 小鼠杂交，成功获得血管内皮特异性敲除 KLK8 的基因工程小鼠（下文简称 KLK8-ecKO）及其对照小鼠(下文简称 KLK8-Ctr)，应用可稳定表达萤火虫荧光素酶的小鼠卵巢癌 ID8-LUC 细胞株构建了卵巢原位注射和腹腔注射的小鼠荷瘤模型，观察了对肿瘤生长、侵袭转移及小鼠存活率的影响。
3. 对 ID8 卵巢原位荷瘤模型的卵巢癌组织进行了单细胞转录组测序（scRNA-Seq），全面评估了肿瘤微环境中上皮细胞，内皮细胞及免疫细胞等的变化。同时通过免疫染色进一步观察验证了移植瘤组织的病理学变化及免疫状态。

结果

1. 内皮细胞培养基培养下，敲低/过表达 KLK8 的 HOMEC 的细胞增殖即管形成能力无明显变化。OVCAR-8，CAOV3，SKOV3 卵巢癌细胞条件培养基培养下，敲低/过表达 KLK8 的 HOMEC 的过表达效应消失，敲低效应依旧存在，且敲低 KLK8 的 HOMEC 的细胞管形成能力显著升高。HOMEC 敲低 KLK8 可促进 OVCAR-8，CAOV3，SKOV3 的迁移和侵袭能力。
2. 同 KLK8-Ctr 相比，KLK8-ecKO 小鼠荷瘤模型的活体成像连续监测荧光值均显著升高，卵巢原位、网膜的肿瘤负担以及血性腹水体积增加，小鼠生存期缩短。

3. scRNA-Seq 通过聚类及标记基因将 44532 个细胞分类为 14 个细胞簇。比例分析显示，同 KLK8-Ctr 相比，KLK8-ecKO 小鼠肿瘤组织中 Treg、成纤维细胞、离子转运型上皮细胞、Fabp4+内皮细胞显著增加，而 CD86+巨噬细胞、粒细胞显著减少。免疫荧光染色进一步证实了这些变化。
结果 在卵巢癌环境中，血管内皮 KLK8 敲低可促进内皮细胞的管形成和卵巢癌细胞的迁移侵袭能力。血管内皮KLK8缺失可导致成纤维细胞增加，内皮细胞与上皮细胞代谢活跃更新加速，促进肿瘤微环境的免疫抑制，协同加速卵巢癌的进展。

PO_216

CBL0137 通过 ROS/BAX 途径诱导卵巢癌细胞焦亡

王志强 娄阁*
哈尔滨医科大学附属肿瘤医院

背景及目的 CBL0137 被设计用于同时调节 p53 和 NF-κB，在多种癌症中通过抑制肿瘤细胞增殖和诱导细胞凋亡表现出抗肿瘤活性。然而，尚未有报道显示 CBL0137 是否能诱导焦亡。本研究旨在研究 CBL0137 是否可以诱导卵巢癌细胞焦亡？如果可以是通过什么机制诱导卵巢癌细胞焦亡？以及 CBL0137 能否作为卵巢癌的潜在治疗剂。
方法 通过采用 A2780、A2780CP、SKOV3、ID8 和 OVCAR3 细胞系，进行肿瘤异种移植实验、Western blot、RT-PCR、透射电镜、免疫荧光检测、IHC、慢病毒转染、细胞活力检测和 LDH 释放检测等方法，探讨 CBL0137 诱导卵巢癌细胞焦亡的机制和靶点。
结果 本研究揭示，CBL0137 通过活性氧（ROS）/BAX 途径诱导 caspase-3/gasdermin E（GSDME）依赖性焦亡。在卵巢癌细胞中，CBL0137 使染色质重塑复合物失活，进而促进染色质转录，导致抗氧化基因的转录减少和氧化，从而使 ROS 水平升高。BAX 通过线粒体 ROS 在线粒体膜上募集，并诱导细胞色素 c 释放以切割 caspase-3。这导致 GSDME 的 N 端裂解，在细胞膜上形成孔隙并诱导焦亡。体内实验结果显示，CBL0137 在体内对卵巢癌细胞也具有抗肿瘤作用。
结论 总之，我们的研究结果揭示了 CBL0137 诱导卵巢癌细胞焦亡的机制和靶点，并表明 CBL0137 是一种极具潜力的卵巢癌治疗药物。

PO_217

FBXL6 Ubiquitinates and Degrades SYNCRIP to Promote Ovarian Cancer Proliferation and Metastasis

Wang Zhiqiang Lou Ge*
Harbin Medical University Cancer Hospital

Ovarian cancer (OC) remains a leading cause of cancer-related death in women, demanding deeper insights into novel molecular drivers of progression. Post-translational modifications, particularly ubiquitination, play critical roles in regulating cancer hallmarks. This study investigated the functional role and molecular mechanism of the E3 ubiquitin ligase FBXL6 in OC. We demonstrate that FBXL6 expression is significantly upregulated in OC tissues and correlates with poor patient prognosis. Through biochemical and cellular assays, we identify the RNA-binding protein SYNCRIP as a novel direct substrate of FBXL6. Mechanistically, FBXL6 catalyzes the K48-linked polyubiquitination of SYNCRIP, leading to its proteasomal degradation. Depletion of FBXL6 stabilizes SYNCRIP protein levels, while its overexpression promotes SYNCRIP ubiquitination and degradation. Functionally, FBXL6-mediated degradation of SYNCRIP is critical for OC pathogenesis. Knockdown of FBXL6 significantly suppresses OC cell proliferation, migration, and invasion in vitro, whereas SYNCRIP silencing reverses these inhibitory effects. Conversely, FBXL6 overexpression enhances OC cell

malignant behaviors, which is dependent on its ligase activity and ability to degrade SYNCRIP. Importantly, in vivo studies using orthotopic or metastatic mouse models confirmed that FBXL6 depletion markedly inhibits OC tumor growth and metastasis. Taken together, our findings reveal a crucial FBXL6/SYNCRIP regulatory axis in OC, where FBXL6 acts as an oncogene by targeting SYNCRIP for ubiquitination and degradation, thereby fueling ovarian cancer proliferation and metastasis. Targeting the FBXL6-SYNCRIP axis may present a promising novel therapeutic strategy for OC treatment.

PO_218

Unraveling Carboplatin Resistance Mechanisms in Ovarian Cancer through Integrated Transcriptomics and Clinical Validation

Xiaohang Lu
Fudan univercity shanghai cancer center

Background Ovarian cancer remains a prevalent and lethal gynecological malignancy, accounting for approximately 239,000 new cases and 152,000 deaths globally each year. It ranks as the seventh most common cancer in women and represents the second leading cause of gynecologic cancer mortality worldwide . This high mortality stems primarily from its insidious onset and the absence of effective early detection strategies, resulting in the majority of patients (~75%) presenting with advanced-stage disease at diagnosis . Despite advancements in cytoreductive surgery and platinum-based chemotherapy, treatment outcomes have plateaued in recent decades. A critical factor underlying this therapeutic stagnation is the nearly universal development of platinum resistance in advanced disease. Carboplatin resistance emerges in most patients during treatment or upon recurrence, ultimately driving disease progression and becoming the predominant cause of ovarian cancer-related mortality. This profound clinical challenge underscores the urgent need to elucidate the molecular mechanisms governing carboplatin resistance.
Methods We established platinum-resistant cell lines using human ovarian cancer cell lines SKOV-3 and A2780. Stepwise dose-escalation of carboplatin (CBP) induced resistant sublines SKOV-3CBP and A2780CBP, both exhibiting >5-fold increase in carboplatin IC50> compared to parental cells. Transcriptome profiling (RNA-seq) of parental and resistant cells was performed. Gene Set Enrichment Analysis (GSEA) identified altered biological processes and signaling pathways associated with resistance. Hub genes were screened from differentially expressed genes (DEGs) and validated using external datasets.
Key Results
High-resistance cell models were successfully established (IC50 >5× parental controls).
RNA-seq revealed significant pathway alterations in resistant cells: epithelial-mesenchymal transition (EMT)-related pathways were markedly upregulated, while G2/M checkpoint regulation and oxidative phosphorylation pathways were downregulated.
Comparative analysis identified 647 common DEGs across both cell lines. Screening the top 20 DEGs with FPKM >3 in parental cells yielded 5 hub genes: FYN, BST2, MARCKS, CD55, and TIMP2.
TCGA data analysis demonstrated that high FYN expression correlated with significantly shorter progression-free survival (PFS) in ovarian cancer patients (*p* = 0.014).
Conclusion This integrated transcriptomic analysis and clinical validation reveals key molecular features and signaling networks underlying carboplatin resistance in ovarian cancer cells. We identify FYN and other hub genes as potential drivers of resistance, providing critical insights for developing therapeutic strategies to reverse platinum resistance.

PO_219

Proteomic profiling and molecular reclassification of high-grade serous ovarian cancer identifies prognostic subtypes and immunotherapy biomarkers

Junfen Xu*
Women's Hospital, Zhejiang University School of Medicine

Purpose High-grade serous ovarian cancer (HGSOC) is the most prevalent subtype of ovarian cancer, exhibiting marked heterogeneity and limited therapeutic options. This study aimed to investigate the proteomic landscape of HGSOC patients to better understand its molecular subtypes, disease progression, and potential therapeutic targets.
Materials and Methods Proteomic profiling was performed on 116 primary HGSOC tumor samples. Bioinformatic analyses were used to identify molecular subtypes, progression-related biomarkers, immune microenvironment classifications, and transcriptional regulatory mechanisms.
Results The analysis revealed FIGO stage IIA as a pivotal transition point between early and advanced HGSOC, characterized by a shift from oxidative stress-related processes to cell cycle-driven mechanisms. The gene GOSR2 emerged as a key player in this transition. Three distinct proteomic subtypes (S-I, S-II, and S-III) were identified, with subtype S-III showing poor prognosis and enrichment in extracellular matrix (ECM)-receptor interactions and immune evasion signatures. Three immune subtypes (IC1, IC2, IC3) were defined based on tumor immune microenvironment characteristics, which correlated with patient outcomes. In advanced HGSOC, biomarkers ISG15, ITGB2, and RELA were identified as potential indicators for stratifying patients who may benefit from immunotherapy.
Conclusion This study provides a comprehensive proteomic characterization of HGSOC patients, highlighting molecular subtypes and immune landscapes associated with prognosis. The findings offer novel insights into disease progression and propose potential biomarkers and therapeutic targets for precision treatment of HGSOC.

PO_220

Injectable photothermal-responsive hydrogel microcapsule with programmable drug release for enhanced ovarian cancer therapy

Zhengwei Sun[1] Rong Ma[1] Lingxiao Huang[1] Yuqing Jin[1] Luoran Shang[2] Xueqiong Zhu*（*corresponding author）[1*]
1.Zhejiang Provincial Clinical Research Center for Gynecological Diseases, Department of Obstetrics and Gynecology, The Second Affiliated Hospital of Wenzhou Medical University, Wenzhou 325027, China
2.Shanghai Xuhui Central Hospital, Zhongshan-Xuhui Hospital and The Shanghai Key Laboratory of Medical Epigenetics, The International Co-laboratory of Medical Epigenetics and Metabolism (Ministry of Science and Technology), Institutes of Biomedical Sciences, Fudan University, Shanghai 200032, China

Purpose Chemoresistance in ovarian cancer, particularly platinum resistance, poses a formidable challenge in contemporary cancer therapy. This study aimed to develop a multifunctional drug delivery system to overcome this limitation through spatiotemporally controlled chemotherapy and photothermal therapy.
Methods We constructed a stepwise microfluidic platform to fabricate core–shell structured microcapsules comprising a gelatin methacrylate (GelMA) core, an alginate (ALG) intermediate layer, and a polydopamine (PDA) outer coating (denoted as GAP). The anti-angiogenic agent Apatinib (YN968D1) was encapsulated in the GelMA core. At the same time, cisplatin was loaded into the ALG layer, enabling a programmed drug release profile characterized by rapid cisplatin release and

sustained Apatinib delivery. Under 808 nm laser irradiation, the PDA coating enabled photothermal-regulated on-demand drug release and exerted photothermal antitumor effects.
Results The GAP@Ap@Pt system exhibited excellent photothermal conversion efficiency, controlled drug release, and biocompatibility. It effectively inhibited angiogenesis, induced apoptosis, and modulated reactive oxygen species (ROS) levels, thereby exerting potent antitumor effects *in vitro*. *In vivo*, using a subcutaneous ovarian cancer xenograft model, the GAP@Ap@Pt system combined with photothermal therapy significantly suppressed tumor growth and enhanced apoptosis, achieving synergistic therapeutic efficacy against ovarian cancer.
Conclusions The multifunctional GAP@Ap@Pt system offers programmable drug release and photothermal therapy, demonstrating considerable potential as a post-surgical adjuvant treatment for platinum-resistant ovarian cancer.

PO_221

Lnc RNA SLC16A1-AS1 通过 IGF2BP2 调节 RBX1 抑制卵巢癌恶性生物学行为

刘汉艳 黄革 林烨 田林欢 李娜*
遵义医科大学第二附属医院

目的 阐明 SLC16A1-AS1 在卵巢癌恶性生物学行为中的作用及其通过 IGF2BP2-RBX1 轴发挥作用的分子机制，为卵巢癌靶向治疗提供新靶点。
材料与方法 1、基于 TCGA/GTEx 数据库及 RT-qPCR 验证 SLC16A1-AS1 在卵巢癌与正常卵巢中的差异表达。2、通过 RPISeq 网站预测联合 RNA 免疫沉淀（RIP）验证 IGF2BP2 与 SLC16A1-AS1、IGF2BP2 与 RBX1 mRNA 的直接结合。3、构建 SLC16A1-AS1 过表达（OE 组）及 IGF2BP2 干扰（si-RNA 组）的卵巢癌 SKOV-3 和 OVCAR3 细胞系，各分为 5 组：Control 组、OE-NC 组、OE 组、OE+si-NC 组、OE+si-RNA 组。采用 CCK-8、流式细胞术、划痕试验、Transwell 实验、TUNEL 染色评估细胞增殖、凋亡、迁移及侵袭。采用 qRT-PCR 及 WB 检测 SLC16A1-AS1、IGF2BP2、RBX1 及 EMT 标志物的表达。免疫荧光检测 RBX1 的细胞定位。4、动物实验：利用过表达 SLC16A1-AS1 的 SKOV-3 细胞构建裸鼠移植瘤模型（OE 组，对照组为 OE-NC 组），记录肿瘤生长情况，HE 及 TUNEL 染色评估肿瘤生长及凋亡，免疫组化、WB 检测 RBX1 表达，WB 检测 EMT 标志物表达水平。
结果 1、SLC16A1-AS1 在卵巢癌组织与细胞的表达均高于正常卵巢细胞（$p<0.05$），IGF2BP2 在卵巢癌细胞中的表达增高（$p<0.05$）。2、RIP 结果证实 IGF2BP2 能够直接结合 SLC16A1-AS1 和 RBX1 mRNA。3、细胞实验：相比于 OE-NC 组，OE 组癌细胞增殖、侵袭与迁移能力下降，凋亡水平增加，IGF2BP2 表达升高，RBX1 表达降低，EMT 进程受阻（E-cadherin↓、N-cadherin↑、Vimentin↑）（$p<0.05$）。OE+si-RNA 组（干扰 IGF2BP2）可逆转 OE 组的抑癌效应。免疫荧光显示 RBX1 定位于细胞核。4、动物实验：OE 组小鼠肿瘤生长速率、肿瘤体积、肿瘤重量显著低于 OE-NC 组；组织病理显示 OE 组肿瘤细胞排列疏松、核分裂减少、坏死细胞增多；TUNEL 染色显示细胞凋亡增加；OE 组 RBX1 表达减少，EMT 过程受阻（E-cadherin↓、N-cadherin↑、Vimentin↑），差异具有统计学意义（$p<0.05$）。
结论 SLC16A1-AS1 作为卵巢癌的抑癌因子，通过结合 RNA 结合蛋白 IGF2BP2 并阻断其对下游靶基因 RBX1 mRNA 的稳定作用，从而下调 RBX1 表达，最终抑制卵巢癌的恶性表型并促进细胞凋亡。该 SLC16A1-AS1-IGF2BP2-RBX1 调控轴是首个被证实的卵巢癌 lncRNA-RBP 负向调控通路，为卵巢癌的靶向治疗提供潜在靶点。

PO_222

倾向性评分匹配法评估术后化疗对 IC 期卵巢黏液性癌患者预后的影响

林蔚 曹冬焱*
北京协和医院

目的 探讨术后化疗对 IC 期卵巢黏液性癌患者预后的影响。
方法 回顾性分析 1997 年 6 月至 2022 年 9 月北京协和医院收治的 119 例 IC 期卵巢黏液性癌（mucinous ovarian carcinoma，MOC）患者的临床资料。根据初治术后是否接受化疗，将患者分为化疗组（91 例）和观察组（28 例）。采用倾向性评分匹配法（匹配比 1:1，卡钳值设为 0.02）均衡组间混杂因素。采用 Kaplan-Meier 生存曲线比较化疗组和观察组的无进展生存期（progression-free survival, PFS）的差异。
结果 患者的中位诊断年龄为 30 岁（12-68 岁），38.4%（38/99）的患者术前血清 CA125 水平升高。根治性手术 56 例（47.1%），保留生育功能手术 63 例（52.9%）；开腹手术 81 例（68.1%），腹腔镜手术 38 例（31.9%）；114 例（95.8%）患者接受了全面分期手术。中位肿瘤直径为 15cm（4-40cm）；46 例经病理科医师复核了生长模式，其中浸润型 5 例，膨胀型 41 例。IC1 期 75 例（63.0%），IC2 期 38 例（31.9%），IC3 期 6 例（5.0%）。91 例（76.5%）患者术后接受以铂类为主的化疗，其中 IC1 期 53 例，IC2 期 32 例，IC3 期 6 例，平均化疗 4 程。中位随访 51 个月（6-296 个月），随访期间 19 例（16.0%）复发，手术至初次复发的中位间隔为 16 个月（5-87 个月），无肿瘤相关性死亡。匹配前，化疗组根治性手术、开腹手术、IC2 期/IC3 期的比例均高于观察组（53.8% vs 25%，75.8% vs 42.9%，41.8% vs 21.4%），而两组在年龄、术前 CA125、全面分期手术、肿瘤直径方面的差异无统计学意义（P>0.05）。成功匹配 54 例患者，化疗组和观察组各 27 例，匹配后两组间的基线特征差异无统计学意义（P>0.05）。化疗组和观察组的 5 年 PFS 分别为 80.2%和 69.0%，差异无统计学意义（P=0.713）。亚组分析：5 例浸润型中，4 例接受了术后化疗，均未复发；1 例未化疗的患者在术后 36 个月后复发，复发部位为双肺。41 例膨胀型中，30 例接受了术后化疗，11 例未化疗，各有 1 例复发，分别在术后 16 个月和术后 34 个月复发，复发部位均为对侧卵巢。
结论 相较于术后观察，术后化疗未能有效改善 IC 期 MOC 患者的 PFS。浸润型 IC 期患者可能从化疗中获益，但需进一步研究验证。

PO_223

全结肠切除术/次全结肠切除术对于卵巢癌患者预后影响的初步研究

姜英 张菁茹*
辽宁省肿瘤医院

目的 探讨晚期卵巢癌患者在全结肠切除术/次全结肠切除术术中及术后并发症发生的情况，评价肿瘤细胞减灭术合并全大肠切除/次全结肠切除术的安全性、可接受程度、对患者生活质量及预后的影响。
材料与方法 通过回顾性分析 2016 年 8 月至 2023 年 12 月在辽宁省肿瘤医院妇科接受肿瘤细胞减灭术合并全结肠切除术/次全结肠切除术的 45 例患者，分析晚期卵巢癌患者在全结肠切除术/次全结肠切除术术中及术后并发症发生的情况，评价肿瘤细胞减灭术合并全大肠切除的安全性、可接受程度、对患者预后的影响。
结果 按纳入标准纳入 38 例，按排除标准排除 1 例。随访过程中失访 4 例。研究显示，33 例行全结肠切除/次全结肠切除术的晚期卵巢癌患者术后化疗的时间平均为术后 26.40±21.46 日。在本研究中发生

术后并发症的为 3 例（腹腔感染 1 例，吻合口瘘伴腹腔感染 1 例，肠梗阻 1 例），术后并发症发生率为 9.09%。在 33 例患者中，有 16 例患者同时行小肠造瘘术。有 2 例患者因肠梗阻、肠瘘于术后 2 个月死亡，肠道重建方式对无进展生存（PFS）及总生存（0S）并无显著影响。
结果 对于在卵巢恶性肿瘤患者肿瘤细胞减灭术中针对肠道多发转移灶是否同时进行全结肠切除术/次全结肠切除术需要妇科肿瘤医生根据患者的病情严重程度、营养状态、手术能否达到满意减瘤等情况进行审慎的决定，从而尽量在延长生存期与提高生活质量之间选择更利于患者的手术方案。

PO_224

关于二手烟暴露和卵巢癌发生风险的 meta 分析

令狐萱宇
石河子大学第一附属医院

背景 卵巢癌是全球女性致死率最高的生殖系统恶性肿瘤，其病因尚未完全阐明。除遗传、生殖及激素因素外，环境暴露日益受到关注。二手烟含有多环芳烃、亚硝胺及芳香胺等七十余种已知或可疑致癌物，可经呼吸道、皮肤及胎盘屏障进入血液，诱发氧化应激与 DNA 损伤。然而，既往针对主动吸烟与卵巢癌的流行病学结论并不一致，而关于被动吸烟的系统性证据尤为匮乏。目的 通过系统评价与 Meta 分析，定量评估二手烟暴露与卵巢癌发病风险之间的关联强度与方向，为病因推断及公共卫生决策提供循证依据。
方法 计算机系统检索 PubMed、Embase、Cochrane Library、CNKI、万方及维普等数据库，检索词包括“secondhand smoke”“passive smoking”“ovarian cancer”及其中文对应词，时限均从建库至 2025 年 5 月。纳入标准 ①研究类型为队列或病例对照设计；②暴露定义为非吸烟者长期接触二手烟；③结局为经病理确诊的原发性卵巢癌；④提供可转换为相对危险度（RR）或比值比（OR）的效应值。由两名研究者独立进行文献筛选、资料提取及纽卡斯尔-渥太华量表（NOS）质量评价；采用 RevMan 5.2 软件进行二分类变量 Meta 分析，以风险比（RR）及 95%置信区间（CI）为合并效应指标，并用 I^2评估异质性。
结果 共纳入 2 篇文献（队列研究 1 篇、病例对照研究 1 篇），总样本 42 427 例，其中卵巢癌患者 14 405 例，对照 28 022 例。Meta 分析提示二手烟暴露与卵巢癌发病风险无统计学关联（RR＝0.84；95%CI 0.31–2.29；P＜0.0001），但研究间异质性极高（I^2＝92%）。亚组分析显示，Baker 等队列研究提示暴露甚至呈现潜在保护趋势（RR＝0.72；95%CI 0.55–0.94）。
结论 目前证据尚不足以确认或否定二手烟暴露与卵巢癌风险的因果关联。高异质性可能源于暴露评估误差、对照来源差异及潜在混杂控制不足。未来亟需开展大样本、标准化暴露评估的前瞻性队列研究，并结合生物标志物验证，以阐明该环境危险因素在卵巢癌发生中的真实作用。

PO_225

不可逆电穿孔对卵巢癌的效应及作用机制研究

王雨濛[1] 夏百荣[1,2*]
1.蚌埠医科大学研究生院
2.中国科学技术大学附属第一医院

目的 探讨不可逆电穿孔（IRE）在体内外卵巢癌模型中，对卵巢癌细胞的杀伤效应及作用机制；
方法 使用不同强度，脉宽及脉冲数对卵巢癌细胞系 SKOV3 进行处理，通过流式细胞术、投射电镜及 CCK-8 检测 IRE 对 SKOV3 细胞增殖能力、凋亡及坏死的影响。使用蛋白印记（Western bolting）检测 SKOV3 凋亡相关蛋白表达情况，并对细胞 ROS 及线粒体膜电位进行检测，探究 IRE 处理后 SKOV3 细胞凋亡和坏死的机制。最后，通过构建 SKOV3 皮下瘤模型，观察 IRE 处理后，瘤体 14 天体积变化，初步探究 IRE 对卵巢癌杀伤的体内效果。
结果 流式细胞术结果表明，当场强为 600V/cm，脉宽为 100us，脉冲数为 50 次时，SKOV3 细胞的凋亡比例最大，随着场强，脉宽及脉冲数的增加，对 SKOV3 细胞的杀伤效果增大，伴随细胞坏死比例增加。WB 结果显示，IRE 处理后，SKOV3 细胞 Cleaved Caspase-3，Cleaved Caspase-9 及 Bax 水平升高，Bcl-2 表达水平降低。ROS 及 JC-1 检测结果表明，IRE 通过引起线粒体膜电位降低，诱导细胞凋亡。动物皮下瘤模型结果显示，IRE 处理后 14 天，肿瘤体积明显缩小（234.2±90 cm3 vs 3521.3±568 cm3,$p<0.05$）。
结果 IRE 通过引起线粒体膜电位变化，诱导 SKOV3 细胞凋亡，随着作用与细胞的场强，脉宽及脉冲数增加，可诱导 SKOV3 出现系统性坏死，场强为 600V/cm，脉宽为 100us，脉冲数为 50 次可达到对 SKOV3 细胞最佳杀伤作用。

PO_226

热疗重塑脂代谢稳态诱导脂毒性应激抑制卵巢癌恶行进展的机制研究

康晓燕 陈刚*
华中科技大学同济医学院附属同济医院

目的 腹腔热灌注化疗（HIPEC）在卵巢癌治疗中的临床获益仍存在争议，其核心瓶颈在于对热疗（HT）诱导肿瘤细胞重塑的机制认知不足。卵巢癌定植于富含脂质的腹腔微环境，其特征性地深度依赖脂质代谢通路以驱动恶性增殖与转移。本研究旨在阐明 HT 是否能通过精准干预卵巢癌细胞的代谢脆弱性（特别是脂质代谢）触发抗肿瘤效应，并揭示其潜在分子机制。
材料与方法 为解析 HT 对卵巢癌细胞代谢的影响，我们构建了 HT 处理下卵巢癌细胞的多组学（转录组学、蛋白质组学、代谢组学）全景图谱，并利用体外细胞模型进行功能验证。研究聚焦于 HT 对脂质代谢网络，特别是脂肪酸氧化（FAO）通路的调控作用及其下游效应。
结果 多组学联合分析及体外验证的核心证据表明，HT 能特异性抑制卵巢癌细胞的脂肪酸氧化（FAO）关键酶 CPT2、HADHA、HADHB 等。这种抑制导致细胞内脂肪酸代谢网络发生显著重塑。进一步的机制研究证实，HT 通过抑制 FAO 驱动的氧化磷酸化（OXPHOS），有效阻断了卵巢癌细胞的能量供应，抑制其增殖和转移能力。同时，HT 诱导的脂质稳态失衡激活了过度的内质网应激（ER stress），进而促进肿瘤细胞死亡。
结果 本研究表明，热疗（HT）通过特异性抑制卵巢癌细胞的脂肪酸氧化（FAO），一方面扰乱其能量代谢（抑制 OXPHOS），另一方面诱导脂毒性及过度内质网应激，最终发挥抑制肿瘤增殖、转移并诱导细胞死亡的抗肿瘤作用。这为深入理解 HIPEC 的作用机制及卵巢癌的代谢靶向治疗提供了新的理论依据。

PO_227

工程化外泌体递送 siRNA 靶向 SNRPD2/突变 p53：干扰剪接调控抑制卵巢癌进展

赵玮 张瑜*
湘雅医院

目的 卵巢癌中 *TP53* 的突变频率极高，突变型 p53（mutp53）通过与互作蛋白形成复合物驱动癌变，但具体机制仍待阐明。小核核糖核蛋白 D2（SNRPD2）是剪接体的核心组分，其与 mutp53 的相互作用是否通过调控可变剪接参与卵巢癌进展，目前尚未明确。本研究旨在探索 SNRPD2 与 mutp53 协同调控可变剪接促进卵巢癌进展的机制，并探索靶向该通路的工程化外泌体在治疗中的应用，为卵巢癌提供新的理论依据和治疗策略。

材料和**方法** 通过样本测序及 Co-IP-MS 筛选 mutp53 的互作蛋白，利用 Co-IP 和 Western blot 验证 SNRPD2 与 mutp53 的特异性结合。采用 CCK-8、平板克隆形成、Annexin V 凋亡检测及 Transwell 实验评估 SNRPD2 对卵巢癌细胞增殖、迁移及凋亡等恶性表型的影响。通过 RNA-seq 分析 SNRPD2/p53 敲降对转录组及可变剪接的调控。在多种 p53 突变卵巢癌细胞系中验证 SNRPD2 与 mutp53 的协同效应，并结合公共数据库及临床样本分析 SNRPD2 表达与预后的关联。构建靶向 SNRPD2/p53 的工程化外泌体（递送 siRNA），评估其联合顺铂的体内外抗肿瘤效果。

结果 1. SNRPD2 特异性结合 mutp53，但不与野生型 p53（wtp53）相互作用。2. SNRPD2 过表达促进卵巢癌细胞增殖、迁移并抑制凋亡，敲降则显著逆转上述表型。3. SNRPD2 与 mutp53 通过调控共同下游基因（如 *EAF2* 等）的可变剪接，协同促进卵巢癌细胞的增殖、迁移及凋亡抵抗等恶性表型。SNRPD2 在卵巢癌中高表达，且其高表达与不良预后相关。4. 靶向 SNRPD2/p53 的工程化外泌体通过递送 siRNA，显著降低卵巢癌细胞对顺铂的 IC50 值，增强化疗敏感性，并有效抑制裸鼠移植瘤的体积及重量增长。

结论 SNRPD2 通过与 mutp53 形成蛋白复合物，调控共同下游基因（如 *EAF2* 等）的可变剪接，驱动卵巢癌细胞的增殖、迁移、凋亡抵抗及化疗抵抗。靶向 SNRPD2/p53 的工程化外泌体联合顺铂为卵巢癌治疗提供了新策略，具有潜在临床转化价值。

PO_228

Smart-responsive Multifunctional Biomimetic Nanocarriers for Chemo-sonodynamic Therapy to Promote Anticancer Immunity in Drug-resistant Ovarian Cancer

hui Zhang[1] Huanzhi Zhang[1] Qi Wang[1] Xuan Gu[1] Jianliu Wang[1] Xiaolong Liang[2] Xiaoping Li[1*]
1.Peking University People's Hospital
2.Peking University Third Hospital

Objective To prepare a tumor microenvironment-responsive biomimetic nanocarrier for targeted delivery of chemotherapeutic drugs and sonosensitizers to tumor tissue, exerting synergistic effects of immunotherapy, sonodynamic therapy, and targeted chemotherapy on drug-resistant ovarian cancer, and to explore the possibility of this biomimetic nanocarrier remodeling the immune-suppressive tumor microenvironment of ovarian cancer.

Methods First, we developed an activated dendritic cell membrane(aDCM)-wrapped nanoplatform with a core of mesoporous silica nanoparticles loaded with doxorubicin and chlorin e6, and covalently linked mPEG-TK on its surface to achieve ROS-responsive drug release characteristics. Then, we characterized the morphology, particle size, sonodynamic effect, and drug release properties of the nanoparticles. Finally, the targeting, safety, anti-tumor effects, mechanism, and immune activation

effects of this multifunctional nano-delivery system were explored through in vitro and in vivo experiments.
Results Negative staining TME image indicated that aDCM@mPEG-TK/MSN-DOX-Ce6 had a clear core-shell structure. WB results demonstrated that functional proteins (CD80, CD86, MHC-I) were retained on the surface of aDCM. Drug release tests demonstrated that drugs could be stably encapsulated in the nanocarriers at pH=7.4, and responsively released under acidic， ROS environments, and ultrasound. Cell uptake experiments indicated its ability for homologous targeting and resistance to macrophage phagocytosis. Results of in vitro immune activation experiments showed that aDCM@MSN could induce macrophages to polarize into the M1 phenotype and could activate T cells through direct and indirect pathways. aDCM@mPEG-TK/MSN-DOX-Ce6 combined with ultrasound effectively induced apoptosis and immunogenic death of tumor cells. In vivo pharmacodynamic experiments confirmed that the nanoparticles could effectively inhibit the malignant proliferation and metastasis of tumors, while also activating anti-tumor immunity and increasing the infiltration of local immune cells in tumors, thereby enhancing sensitivity to the PD-L1 Antibody.
Conclusion In this study, we constructed an intelligent, precise, and efficient immune-combination therapy nanoplatform to exert synergistic effects of immunotherapy, sonodynamic therapy, and targeted chemotherapy on drug-resistant ovarian cancer. This biomimetic nanocarrier enables targeted delivery and controlled release of chemotherapy drugs. It also restructures the immune microenvironment and provides a revolutionary strategy for overcoming chemoresistance and enhancing the efficacy of anti-PD-L1 therapy in ovarian cancer.

PO_229

PPAR-γ 激动剂通过抑制 SIRT1 信号通路逆转细胞衰老增强卵巢癌细胞对奥拉帕利的敏感性

王泽华
复旦大学附属妇产科医院

目的 卵巢癌是致死率最高的妇科恶性肿瘤，PARP 抑制剂虽然在卵巢癌治疗方面取得了显著的疗效，但 PARP 抑制剂耐药是临床治疗所面临的新问题。诱导细胞衰老虽然可以抑制肿瘤生长，但随着衰老分泌表型的过度累积，卵巢癌细胞呈现治疗耐药。之前研究发现 PPAR-γ 激动剂罗格列酮可以钝化卵巢癌细胞衰老，减少衰老相关分泌表型，促进卵巢癌细胞凋亡，但具体机制尚不明确。
材料与方法 利用裸鼠卵巢癌皮下移植瘤模型分析不同 PPAR-γ 激动剂单独或联合奥拉帕尼（PARP 抑制剂）的抑瘤效应。利用 β-半乳糖苷酶（SA-β-Gal）和 real-time PCR 检测细胞衰老及衰老相关分泌表型（SASP）的表达变化，利用免疫组化检测细胞凋亡的变化。利用网络药理学分析 PPAR-γ 激动剂增加奥拉帕尼抗肿瘤效应的潜在机制，进一步通过 Western blot 和免疫组化检测 SIRT1 信号通路对奥拉帕尼敏感性的影响。
结果 体内实验显示不同 PPAR-γ 激动剂（罗格列酮和吡格列酮）联合奥拉帕尼能显著缩小皮下移植瘤小鼠（A2780 和 OVCAR-3）的肿瘤体积（$P < 0.01$）。进一步研究发现，PPAR-γ 激动剂通过抑制 SIRT1 的表达，钝化奥拉帕尼诱导的卵巢癌 A2780 和 OVCAR-3 细胞衰老，增加细胞发生 DNA 损伤，而加入 SIRT1 激动剂能逆转上述表型。
结论 PPAR-γ 激动剂通过抑制 SIRT1 信号通路，钝化奥拉帕尼诱导的细胞衰老，从而提高奥拉帕尼的药物敏感性。

PO_230

五谷虫对卵巢癌和子宫内膜癌小鼠异种移植瘤生长具有抑制作用

孙悦
郑州大学第一附属医院

为了研究五谷虫粉末对卵巢癌和子宫内膜癌的抑制作用，我们通过培养 SKOV3 细胞和 HEC-1B 细胞进行雌性 BALB/c 裸鼠异种移植瘤的动物实验，待均成瘤至 70-100mm3 后各分为五组（对照组、五谷虫低剂量组、五谷虫中剂量组、五谷虫高剂量组、顺铂组），给药组每天五谷虫粉末灌胃给药一次。通过测量给药后每两天的肿瘤生长情况及小鼠体重来监测治疗效果及评估小鼠状态，对肿瘤生长的差异进行了统计分析

。卵巢癌组和子宫内膜癌组小鼠分别在第 15 天和第 21 天达到治疗终点，处死小鼠，分离并分析肿瘤。所有统计分析均使用 GraphPad Prism 9 进行，两组间统计学意义的比较采用双尾 Student's t-检验。p 值 < 0.05 被认为具有统计学显著性。

研究结果显示在卵巢癌和子宫内膜癌组中，五谷虫低、中、高剂量对小鼠肿瘤均有抑制生长的作用，且抑制作用与剂量高低呈正相关，高浓度组抑制率最高，另外小鼠状态与体重均未见明显下降。顺铂组对小鼠肿瘤也有明显抑制作用，但小鼠状态变差。这些结果表明五谷虫在卵巢癌和子宫内膜癌治疗中具有潜在的治疗价值，且五谷虫对小鼠无明显毒副作用。

PO_231

HELQ 通过 IGFBP3 活化 CAFs 促进卵巢癌侵袭转移的机制研究

李奕 谭舒然 张瑜*
中南大学湘雅医院

研究目的 卵巢癌尤其晚期患者预后差。肿瘤微环境中，肿瘤相关成纤维细胞（CAFs）与癌细胞互作促转移。POLQ 样解旋酶（HELQ）在肿瘤发生中作用重要，但其在卵巢癌进展中的机制不明。本研究旨在阐明 HELQ 是否参与肿瘤细胞与 CAFs 相互作用促进卵巢癌转移。

材料与方法 (1) 免疫组化检测 74 例高级别浆液性卵巢癌（HGSOC）组织中 HELQ 和 CAFs 标志物（α-SMA/FAP）表达，分析其与临床病理特征及相互间的相关性。(2) 建立 HELQ 过表达（OE-HELQ）的卵巢癌细胞 HeyA8 与成纤维细胞 MRC5 共培养模型。转染 siRNA 敲低 IGFBP3。利用 RT-qPCR、WB 检测表达；CCK-8、Transwell 检测增殖、迁移、侵袭。(3) 生物信息学：基于 ESTIMATE 算法分析 TCGA 中 HELQ 表达与基质浸润关系；联合细胞因子芯片和 OE-HELQ 转录组数据筛选 HELQ 相关细胞因子；利用 TIMER2.0 分析 IGFBP3 与 CAFs 浸润的相关性。

结果 (1) HELQ 与 α-SMA、FAP 在 HGSOC 中高表达且均与不良预后相关；HELQ 表达与 α-SMA、FAP 均呈正相关。(2) HELQ 过表达增强 HeyA8 迁移和侵袭（不影响增殖），并活化 MRC5 细胞；活化的 MRC5 进一步促进肿瘤迁移侵袭。OE-HELQ 细胞中 IGFBP3 表达上调。敲低 IGFBP3 可逆转 HELQ 过表达对 HeyA8 迁移侵袭的促进作用，并抑制 HELQ 过表达细胞对 MRC5 的活化作用以及 MRC5 的促瘤效应。(3) HELQ 高表达组基质浸润水平更高。联合分析筛选到 IGFBP3 在 OE-HELQ 细胞中上调。卵巢癌中 IGFBP3 mRNA 与 CAFs 浸润丰度正相关。

结果 HELQ 通过上调 IGFBP3，以调节肿瘤细胞自身及活化 CAFs 的双重模式，促进卵巢癌细胞迁移和侵袭。

PO_232

ADC 药物在卵巢癌治疗中的研究进展

董莹*
昆明医科大学第二附属医院

目的 卵巢癌是女性生殖系统常见的恶性肿瘤，死亡率居妇科肿瘤之首，5 年生存率仅约 40%。虽然腺苷二磷酸核糖聚合酶抑制剂（poly ADP-ribose polymerase inhibitors，PARPi）的出现从根本上改变了卵巢癌的治疗格局，但 35%的患者初治时便对 PARPi 产生耐药，抗体-药物偶联物（ADC）的出现代表了一种新型的精准医学，为卵巢癌乃至铂耐药、PARPi 耐药患者的治疗提供了新选择。本文探讨了 ADC 药物在卵巢癌及耐药卵巢癌患者治疗中的研究进展，以期为临床中卵巢癌的治疗提供依据。
方法 通过计算机检索 PubMed、CNKI、迈特思创等数据库中近 5 年发表的 ADC 药物与卵巢癌、耐药卵巢癌相关的文献。纳入相关研究并筛选，对最终纳入的文 献进行归纳综述。
结果 1. 索米妥昔单抗（Mirvetuximab Soravtansine，MIRV）是全球首个获批用于叶酸受体 α（FRα）阳性铂耐药复发卵巢癌治疗的 ADC 药物，通过特异性识别并靶向 FRα 高表达的卵巢癌细胞对肿瘤细胞发挥作用。索米妥昔单抗相较化疗显著改善 FRα 高表达铂耐药复发卵巢癌患者的长期生存率，降低 35%疾病进展风险和 33%全因死亡风险。2. 戈沙妥珠单抗（sacituzumab govitecan，SG）通过抗 TROP2 诱导 G2/M 期阻滞，增加 RPA1 病灶，降低复制叉速度，导致卵巢癌细胞 DNA 复制应激，引起 DNA 损伤，发挥抗肿瘤作用，在 PARPi 敏感和耐药的卵巢癌细胞中发挥的作用一致。3. 德达博妥单抗（datopotamab deruxtecan，Dato-Dxd）同样靶向 TROP2，与 TROP2 阴性的卵巢癌细胞相比，TROP3 过表达和中度表达的卵巢癌细胞系对 Dato-DXd 表现出更高抗肿瘤活性。4. HS-20089 在卵巢癌中靶向 B7-H4，是全球首个进入 III 期卵巢癌临床的 B7-H4 ADC，B7-H4 在 92%的高级别浆液性卵巢癌肿瘤中过表达，在铂类或 PARPi 耐药性卵巢癌细胞中，HS-20089 显著降低活力和集落形成，同时增加细胞周期停滞和 DNA 损伤，最终导致细胞凋亡。
结果 卵巢癌 ADC 药物的研发近年来取得显著突破，逐步成为克服卵巢癌铂、PARPi 耐药及复发难题的核心策略。目前研究主要聚焦于靶向 FRα、TROP2、B7-H4 等分子，通过 ADC 药物的靶向特异性，显著提升疗效，为解决卵巢癌治疗难题提供了新选择，应用前景广阔，对提高卵巢癌患者的预后具有重要意义。

PO_233

LGSC 与 HGSC：从病理诊断到精准治疗的跨越

亚斐
郑州大学第一附属医院

2014 年 WHO 将浆液性卵巢癌分为高级别浆液性癌和低级别浆液性癌。低分化的浆液性癌是高级别浆液性癌，占浆液性癌的 90%；高分化和中分化的浆液性癌是低级别浆液性癌，占浆液性癌的 10%。病理学在卵巢癌的诊断、分期和治疗中起着不可或缺的作用。本文就卵巢上皮性癌的两级分级系统为基础，重点介绍病理学在浆液性卵巢癌诊断以及治疗的作用。在大体上，HGSC 肿瘤组织细腻而糟脆或似烂肉样，坏死出血，伴大片融合的乳头状突起，缺少良性与交界性肿瘤的区域，而 LGSC 纤维性囊壁光滑质韧，乳头发育良好，缺少烂肉样物与广泛坏死等 HGSC 的常见表现。罕见 HGSC 由 LGSC 发展而来时，可见到良性与交界性的区域。HGSC 的结构多样，且不具有特异性，因此在诊断 HGSC 的过程中最关键的指标是细胞异型性。LGSC 核分裂计数＜12 个/10HPF，而 HGSC 核分裂计数常＞12 个/10HPF。另外，间质浸润是区分癌前病变与癌的关键指标，也是诊断 LGSC 的必备条件，但 HGSC 的诊断并不苛求有间质浸润。免疫组化方面，p53、p16 可能成为诊断 HGSC 的有效标志，雌孕激素受体阳性为 LGSC 的内分泌治疗奠定了基础。LGSC 常常表现为 MAPK 信号通路相关基因异常，而远端输卵管的病变被认为是 HGSC 的前体病变，同时 STIC 被认为是其最早可识别的形态学特征。

另外，LGSC 对化疗不敏感，而 HGSC 患者通常需要进行术后辅助化疗和（或）新辅助化疗。总之，HGSC 和 LGSC 是完全不同的两种疾病，不是一种疾病的不同分级。LGSC 与 HGSC 的生物学差异不仅是病理诊断的核心依据，更是驱动临床研究与治疗创新的关键枢纽。研究病理学在 HGSC 和 LGSC 诊疗中的作用，有利于我们实现从精准治疗，最终改善患者生存质量。

PO_234

子宫内膜异位症相关性卵巢癌基因组改变的研究进展

杨佳慧 王丹波*
辽宁省肿瘤医院

子宫内膜异位症（endometriosis，EMs）是育龄期女性常见的慢性炎症性疾病之一，其在形态学上虽呈现良性表现，但在生物学行为上却有着与恶性肿瘤类似的特征。子宫内膜异位症恶变风险为 1%，且 80%的子宫内膜异位症恶变发生于卵巢组织，从而引起子宫内膜异位症相关性卵巢癌（endometriosis-associated ovarian cancer，EAOC）。目前对于 EAOC 的分子生物学特征及临床特征仍处于探索阶段，本文对目前已知的 EAOC 特征性突变基因进行整合，期其对 EAOC 的临床诊断及用药指导提供基础理论依据。

ARID1A 突变、PIK3CA 突变、KRAS 突变、PTEN 突变是目前已知的 EAOC 相关突变基因，且被认为是 EMs 恶变的早期事件。ARID1A 突变导致 SWI/SNF 复合物介导的染色质重塑失调，从而使肿瘤细胞异常增殖。PIK3CA 突变可激活 PI3K，进一步活化效应因子 AKT，AKT 可使多种转录因子磷酸化，从而增强抗凋亡基因的表达，进而增强细胞的存活增殖，血管生成、侵袭转移能力。KRAS 突变导致 RAS 蛋白的持续激活，从而激活 RAS/MAPK 通路，使细胞异常增殖、血管生成。PTEN 突变使其功能丧失导致 PI3K 复合物持续激活或 PI3K 表达异常，从而影响一系列的下游通路，包括细胞周期、细胞凋亡、p53 通路、蛋白翻译等。

通过对相对特异性的分子进行检测，预测 EAOC 的发生及 EMs 恶变的风险，对于临床确诊 EAOC 具有一定的诊断价值。且通过开发新型的治疗靶点，对于 EAOC 的治疗及预后评估，临床用药指导具有重要的临床价值。

PO_235

CUCUDC-907 exhibits potent antitumor effects against ovarian cancer through multiple in vivo and in vitro mechanisms

王 元培
郑州大学第一附属医院

Purpose CUDC-907 is a promising dual-target inhibitor of the HDAC and PI3K signaling pathways, with demonstrated therapeutic efects in a range of malignant tumors. However, its potential application in ovarian cancer (OC) has not been fully explored yet. In this study, we sought to investigate the efcacy of CUDC-907 in treating OC, both in vitro and in vivo. **Methods** Here, we examined the correlation between PI3K or HDAC expression and the prognosis of OC patients using the GEPIA database. RNA-Seq analysis was performed on OC cells treated with CUDC-907.To assess various cellular processes, including proliferation, migration, invasion, apoptosis, and cell cycle, we performed a series of assays, including the CCK8, EDU, wound healing, cell invasion, and fow cytometry assays. Real-time quantitative PCR and western blotting were performed to measure the expressions of target

genes. Additionally, we utilized the SKOV3 xenograft tumor model to investigate the inhibitory efects of CUDC-907 on tumor growth in vivo.
Results Bioinformatics analyses revealed that up-regulated HDAC and PI3K were signifcantly correlated with patients' poor survival in OC. In vivo and in vitro experiments have demonstrated that CUDC-907 could inhibit the proliferation of OC cells by inhibiting the PI3K and HDAC pathways to down-regulate the expression of c-Myc, and induce cell apoptosis by inhibiting the PI3K/AKT/Bcl-2 pathway, and up-regulate p21 to induce G2 /M phase arrest.
Conclusion Our results showed that CUDC-907 had powerful anti-tumor efects on OC, which could provide a theoretical and experimental basis for the application of CUDC-907 in the therapy of OC.

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Clinicopathological features and prognosis of synchronous endometrial and ovarian carcinoma

Wenli Gan[1,2]
1.Department of Gynecology and Obstetrics，Affiliated Hospital of Sichuan Nursing Vocational College (The Third People's Hospital of Sichuan Province)
2.Department of Gynecology, West China Second University Hospital, Sichuan University

Objective A retrospective cohort study was conducted to characterize the clinicopathological features and extended prognostic outcomes in synchronous endometrial and ovarian carcinoma (SEOC) patients, aiming to identify prognostic determinants. To establish a theoretical basis for enhancing prognostic outcomes and devising personalized therapeutic regimens in patients with SEOC.
Materials and Methods A total of 48 patients with synchronous endometrial and ovarian carcinoma (SEOC), diagnosed by surgical pathology and treated at West China Second University Hospital between January 1, 2015 and December 31, 2022 were retrospectively enrolled. Patient baseline characteristics, clinical and pathological data, progression-free survival (PFS), and overall survival (OS) were collected. A retrospective statistical analysis was performed to characterize the clinical profiles, pathological features, and survival prognosis of SEOC patients. Survival curves were generated using the Kaplan-Meier method, and non-parametric Log-rank test was used to evaluate the difference in survival rates among different clinical subgroups. Multivariate regression analysis was conducted based on Cox proportional risk model to screen risk factors affecting the prognosis of patients with SEOC.
Results The median age at diagnosis of the 48 patients with SEOC was 47 years (range: 27-59 years) and the median Body Mass Index (BMI) was 23.2kg/m2 (range: 16.4-35.2 kg/m2). Among them, 42 cases (87.5%) were premenopausal. The common first symptoms were vaginal bleeding. Pathological evaluation identified 38 cases (79.2%) of concurrent endometrioid carcinomas in both endometrial and ovarian sites, with 23 patients (47.9%) exhibiting grade 1 (G1) differentiation and 24 cases (50%) diagnosed at early-stage (FIGO I). There were 45 cases (93.8%) of endometrial carcinoma with or without infiltration of superficial muscle. There were 4 cases (8.3%) with positive lymphatic metastasis. Intraoperative ascites cytological positive in 9 cases (18.8%). Only 5 patients (10.4%) did not receive adjuvant therapy. Survival analysis revealed: The histological type of OC ($p=0.008$), the histological type of SEOC ($p=0.016$), the histological grade of SEOC ($p=0.029$), the histological stage of OC ($p=0.032$), the lymphatic metastasis or not ($p < 0.001$), and lymph dissection or not ($p=0.029$) were significant factors affecting the survival and prognosis of patients.
Conclusion Compared to solitary endometrial or ovarian cancers, SEOC predominantly occurs in young, premenopausal women, with vaginal bleeding as the most common initial symptom. Preoperative imaging and tumor marker levels are critical for guiding surgical decision-making. SEOC is pathologically characterized by early-stage, low-grade endometrioid carcinomas (EEC/EOC), and patients with these features typically exhibit favorable prognoses. Lymph node metastasis, histologic type and stage of the ovarian tumor are key prognostic factors influencing survival outcomes and play

a pivotal role in determining postoperative adjuvant therapy. The clinical benefits of systematic lymphadenectomy in early-stage SEOC patients remain to be further explored.

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靶向间皮素的 TCR-like CAR-T 细胞治疗铂类耐药晚期卵巢癌：一项首次人体剂量递增临床试验

单奕阳[1] 沈杨[1,2*]
1.东南大学附属中大医院 2.南京市智慧妇产医学重点实验室

研究目的 卵巢癌由于确诊较晚、复发率高且治疗手段有限，仍是临床治疗中的重大挑战。基于间皮素（MSLN）在卵巢癌中的高表达特性，本研究团队开发了一种靶向 MSLN 的新型 TCR-like CAR-T 细胞疗法（KT127）。在临床前研究证实其抗肿瘤活性的基础上，首次开展了针对铂类耐药晚期卵巢癌患者的开放标签、剂量递增 I 期临床试验（试验注册号：NCT05963100），评估其安全性和初步疗效。
材料与方法 本研究采用快速滴定与经典“3+3”剂量递增相结合的设计。11 例患者在淋巴细胞清除预处理后，接受了剂量范围为 1×10^{6}~2×10^{7} cells/kg 的 KT127 输注。主要研究终点为 KT127 的安全性和耐受性，次要研究终点包括总生存期（OS）、疾病控制率（DCR）和无进展生存期（PFS）。此外，采用 EORTC QLQ-OV28 量表评估患者生活质量（QoL），并通过血浆蛋白组学分析探索潜在的疗效预测生物标志物。
结果 2023 年 8 月 7 日至 2024 年 8 月 15 日期间，本研究共成功筛选入组 11 例患者，所有患者均完成知情同意签署、外周血单个核细胞采集术及 CAR-T 细胞回输。其中 1 例患者因疾病进展自愿退出研究，最终 10 例患者纳入疗效和安全性分析。所有患者治疗过程中均未观察到剂量限制性毒性（DLT）、细胞因子释放综合征（CRS）或免疫效应细胞相关神经毒性综合征（ICANS）。疾病控制率（DCR）达 80%（8/10），中位无进展生存期（PFS）为 84 天（24~234 天）。截至 2025 年 6 月 1 日，中位总生存期（OS）为 142.5 天（28~572 天），其中 4 例（40%）患者仍存活。生活质量评估显示，患者治疗后腹部/胃肠道症状显著改善（p=0.037），其他维度未见明显恶化。蛋白组学分析发现，基线期激肽释放酶相关肽酶（KLK13、KLK14）和趋化因子 CXCL17 的差异表达可能与治疗反应相关。
结论 本研究证实，KT127 在经多线治疗的晚期卵巢癌患者中具有可控的安全性，并展现出良好的临床活性，有望为这类患者提供新的治疗选择。

PO_238

Cachexia index for prognostication in surgical patients with advanced epithelial ovarian cancer: a retrospective study

Xia Wu[2] Han Gong[3] Dan Nie[1*]
1.The Affiliated Traditional Chinese Medicine Hospital, Southwest Medical University
2.The Affiliated Hospital, Southwest Medical University
3.Department of Obstetrics and Gynecology, Key Laboratory of Birth Defects and Related Diseases of Women and Children, Ministry of Education, West China Second University Hospital, Sichuan University

Background Cancer cachexia significantly affects survival in epithelial ovarian cancer (EOC), but ascites and tumor burden confound the traditional diagnostic criteria. This study aimed to investigate the prognostic value of the cachexia index (CXI) in patients with advanced EOC undergoing primary cytoreductive surgery.
Methods This retrospective study included 240 FIGO stage III-IV EOC patients treated between January 2017 and December 2021 at two medical centers. CXI was calculated as (skeletal muscle index [SMI]×albumin) / NLR, with a cut-off value of 40.5 stratifying patients into low and high CXI groups.

Survival analyses (Kaplan-Meier, log-rank) and Cox proportional hazards models were used to assess the association between CXI and progression-free survival (PFS) and overall survival (OS).
Results Low CXI (<40.5) was significantly correlated with advanced FIGO stage (20.4% vs. 10.6%, P=0.036), suboptimal cytoreduction (15.7% vs. 6.8%, P=0.035), reduced SMI (37.80 ± 4.63 vs. 39.56 ± 3.82 cm^2/m^2, P = 0.002), and elevated NLR (3.87 vs. 2.25, P<0.001). In addition, patients in the low CXI group had a significantly higher rate of severe postoperative complications and longer length of hospital stay (P<0.05). Kaplan–Meier survival curves indicated that patients with low CXI had significantly shorter OS (log-rank P = 0.002) and PFS (log-rank P = 0.021). Multivariate analysis confirmed that low CXI was an independent predictor of reduced PFS (HR=2.57, 95% CI 1.81-3.66, P<0.001) and OS (HR=3.50, 95% CI 2.08-5.87, P<0.001).
Conclusion Preoperative CXI was an independent prognostic factor for survival in patients with EOC. It facilitates early identification of cancer-related cachexia, thereby enabling timely interventions to enhance patient outcomes.

PO_239

卵巢癌免疫代谢微环境中 CD 分子与代谢酶的作用机制及治疗进展

陈雪
南通大学附属医院

研究目的 整合卵巢癌（OC）免疫代谢微环境最新进展，阐述 CD36、CD226 等免疫分子及 CCDC64B、CYP11A1 等代谢酶在肿瘤进展、转移耐药中的机制，评估其临床转化潜力。
材料与方法 检索 PubMed、Web of Science 等数据库（2019-2025），以"卵巢癌+CD36/CD226/CYP11A1/脂代谢/免疫微环境"为关键词筛选文献。结合多组学数据及临床试验（如 2025 ASCO ROSELLA 试验）评估靶点价值。
结果 CD36 在 OC 中高表达，通过摄取网膜脂肪细胞释放的游离脂肪酸（FFA）驱动脂滴积累和转移。其机制涉及上调 SREBP 通路增强脂肪酸合成酶（FASN）表达，并抑制 ABCA1 激活 PI3K/Akt 通路。肿瘤外泌体可诱导巨噬细胞经 CD36 依赖性脂质摄取形成衰老表型，加速转移，临床证实 CD36 高表达与不良预后相关。
CD226（DNAM-1）作为 T/NK 细胞共刺激受体，其功能因 T 细胞耗竭受损。人源 cDC1 细胞激活 CD226 信号可恢复 T 细胞毒性，激动剂抗体在临床前模型中显著提升 IFNγ 分泌并抑制肿瘤生长。
代谢靶向治疗取得突破：CYP11A1 口服抑制剂 Opevesostat（MK-5684）已进入 II 期试验。ROSELLAIII期研究显示，Relacorilant 联合白蛋白紫杉醇使铂耐药复发 OC 患者中位 OS 延长至 15.97 个月（对照组 11.50 个月）。CD36 抑制剂（SSO+他汀类）可抑制脂滴积累，CD226 激动剂展现逆转 T 细胞耗竭潜力。
结论 CD36-CD226 轴与 CYP 代谢酶构成 OC 免疫代谢调控核心。靶向 CD36 可阻断脂质依赖性转移，CD226 激动剂联合免疫检查点抑制剂有望逆转免疫抑制。未来需探索"代谢调节剂+免疫疗法+化疗"联合策略，并通过多组学技术筛选生物标志物推动个体化治疗。

PO_240

工程化细胞外囊泡靶向递送内源性 TP53 用于卵巢癌治疗

张梦蕾 顾圆圆 沈方 龚滢欣 顾铮 华克勤 周冠楠 丁景新*
复旦大学附属妇产科医院

研究目的 p53 抑癌基因是调控多条细胞通路的主控因子，在多种肿瘤中频繁发生变异。目前已有的 p53 表达恢复策略（包括小分子和 DNA 疗法）虽取得一定进展，但均存在明显局限。本研究拟构建一种内源性细胞外囊泡（EVs）平台，能够高效递送游离 TP53 蛋白，以抑制 TP53 缺失型卵巢癌。
材料和方法 我们证实内源性 TP53 蛋白可通过 Lamp2b 分拣并装载到工程化 EVs 中。我们对这些生物工程 EVs 进行生产与表征，并评估其对内源性 GFP 蛋白、荧光素酶蛋白及内源性 Cre 重组酶的递送能力。此外，我们制备了特异性靶向的 TP53 工程 EVs，用于治疗人工构建的 TP53 缺失型肝细胞癌（HCC）及天然 TP53 缺失型卵巢癌（OCs），并检测细胞凋亡。体内实验中，制备了靶向 MSLN（间皮素）的 EVs-TP53，用于治疗 MSLN 阳性卵巢癌，并对肿瘤组织中的 TP53 表达进行分析。
结果 体外实验显示，工程 EVs 不仅能封装并功能性递送内源性 GFP 蛋白，还能递送内源性 Cre 重组酶，实现卵巢癌细胞的基因组编辑。此外，过表达链霉亲和素的 EVs（SA-EVs）可实现特异性靶向递送。体内实验中，SA-EVs-TP53（含 TP53 蛋白的 SA-EVs）显著抑制 TP53 缺失型癌细胞（人工 TP53-null HCC 及天然 TP53 缺失型卵巢癌）的增殖并诱导凋亡。更重要的是，靶向 MSLN 的 EVs-TP53 能将 TP53 蛋白特异性递送至 MSLN 阳性卵巢癌，显著发挥抗肿瘤效应。
结论 我们建立了一种一步法策略，利用特异性靶向的非病毒 EVs 将 TP53 递送至卵巢癌细胞。过表达链霉亲和素的 EVs（SA-EVs）可实现精准靶向递送。研究表明，通过靶向 EVs 递送策略恢复抑癌蛋白功能，可能成为卵巢癌治疗的有前景的新途径。

PO_241

遗传性卵巢癌家系研究的范式革新：从多组学整合到临床精准防控的转化之路

刘淑曼
宁夏医科大学总医院

研究目的 针对遗传性卵巢癌管理中单基因局限（>40%高危家系病因不明）和静态风险评估不准确两大痛点，本研究旨在：1. 整合家系资源、多组学技术与人工智能算法，构建动态全景风险模型；2. 提出分层精准干预路径，优化预防性手术、靶向治疗及遗传咨询策略。
材料与方法 1.数据来源：1.1 家系队列：前瞻性多代系研究。1.2 组学数据：全基因组测序（WGS）鉴定非编码变异； 转录组/表观组分析功能通路；1.3 临床数据：预防性手术效果随访。2.技术方法 2.1. 风险模型构建：多基因风险评分（PRS）：整合 BRCA1/2、RAD51、RECQL 等 12 基因； 动态全景模型（PRS-Pano）：融合家族史时序变化、环境暴露的图神经网络；2.2 疗效预测： PARPi 响应标志物：TP53BP1 SNP（rs56069436）联合 HRR 通路突变；2.3 验证体系：独立队列验证：类器官药敏实验。
结果 1. 全景风险模型效能：1.1 FH-Net，预测目标为卵巢癌高风险家系识别，效能 AUC=0.89；1.2 中国 PRS 模型，预测目标为 BRCA 阴性家系风险分层，效能 AUC=0.81（vs.欧美 0.68）；1.3 低外显累加效应，预测目标为 ATM+CHEK2 双突变，效能风险升高 4.2 倍 (P<0.001)。2. 精准干预效果：2.1 RRSO 手术，获益群体为 BRCA1 携带者，卵巢癌死亡风险下降 77%；2.2RRSDO 分期手术，年轻 BRCA 携带者生活质量评分上升 30%；2.3 PARPi 治疗，RAD51C 突变患者无进展生存期延长 9 个月；2.3 联合 STAT3 抑制剂，TP53BP1 SNP 阳性患者耐药逆转率↑60%。

结论 1. 风险模型革新：全景动态评估（PRS-Pano）优于传统单基因模型； 非编码变异成为新风险标志物，倡导临床检测覆盖启动子区。2. 临床路径优化：预防手术：分期切除（RRSDO）可保留年轻患者生育与内分泌功能；靶向治疗：依据 HRR 通路状态扩展 PARPi 适应症。3. 转化落地：推动 NCCN 指南新增 RECQL 基因检测与分期手术建议。

PO_242

EPYC 介导的微环境重塑：卵巢癌转移新靶点与腹水标志物的发现

邓璐[1] 钱瑜华[2] 王丹丹[3] 陈守真[1*]
1.复旦大学附属妇产科医院 2.复旦大学附属华东医院 3.苏州市立医院

研究目的 卵巢癌易发生盆腹腔广泛转移且预后极差，阐明其转移机制至关重要。前期研究发现骨骺蛋白（EPYC）在卵巢癌转移灶中高表达且与不良预后相关。本研究旨在验证 EPYC 调控卵巢癌转移的分子机制及其在肿瘤微环境（TME）中的细胞来源。
材料与方法 利用慢病毒载体构建 EPYC 过表达和敲低的卵巢癌稳转细胞株。通过 CCK-8、集落形成和 EdU 实验检测细胞增殖能力。划痕实验和 Transwell 实验检测细胞迁移能力。重组蛋白添加实验验证分泌型 EPYC 蛋白的功能。利用裸鼠皮下移植瘤及腹腔种植转移模型进行体内验证。免疫组化（IHC）检测 EPYC 在卵巢癌组织中的表达。ELISA 定量腹水或腹膜液和血浆中 EPYC 水平。Western blot 检测 PLCγ2 磷酸化水平，抑制剂 U-71322 处理细胞。KEGG 分析 EPYC 过表达转录组差异基因富集信号通路。挖掘 GEO 数据库卵巢癌单细胞数据，分析 EPYC 的细胞来源。
结果 功能实验证实 EPYC 正向调控卵巢癌细胞增殖与迁移。体内模型显示 EPYC 过表达后皮下瘤体积增大 1.07 倍，腹腔种植转移结节数增加 2 倍；EPYC 表达敲低后皮下瘤体积缩小 64.4%，腹腔种植转移结节数减少 44.7%。rEPYC 处理后细胞转移及转移能力显著增强，且呈浓度依赖性。IHC 结果显示转移灶间质 EPYC 阳性率 66.7%显著高于原发灶 32.1%（$p<0.001$）。单细胞数据分析发现 EPYC 富集于成纤维细胞，且转移灶成纤维细胞中 EPYC 表达高于原发灶。EPYC 与癌症相关成纤维细胞关键标记分子 ACTA2/FAP/PDGFRB 表达显著正相关（r=0.45~0.61，p=0）。腹水 EPYC 浓度与转移灶数量正相关（r=0.686，p=0.01），晚期患者 EPYC 浓度高于早期（0.1161 ± 0.0725 ng/ml vs. 0.0449 ± 0.0653 ng/ml，p=0.061）。EPYC 过表达激活 PLCγ2 磷酸化。PLCγ2 抑制剂 U-71322 选择性抑制 EPYC 过表达细胞的增殖。KEGG 富集分析显示 EPYC 激活黏着斑、Erb 信号通路及 EGFR-TKI 耐药通路。
结论 EPYC 可能通过重塑 TME 驱动卵巢癌转移，腹水 EPYC 可作为预测转移负荷的无创标志物，EPYC 可能成为卵巢癌治疗的潜在靶点。

PO_243

Differential analysis of different biological behaviors of FIGO-stage III ovarian cancer: a population-based study

Zelin Hu Jiazhen Huang .M.DoAmaralGuterres O Ning Wang*
The second hospital of Dalian Medical University

When ovarian cancer is initially diagnosed, it is often at a relatively late stage. At the same time, in patients with advanced ovarian cancer, lymphatic metastasis and intraperitoneal tumor metastasis are a major feature. What causes these two patterns of metastasis in ovarian cancer patients to differ is unknown. We performed differential analysis through The Cancer Genome Atlas Program (TCGA) database to compare International Federation of Gynecology and Obstetrics (FIGO) IIIA (T3aN1M0)

and IIIC (T3cN0M0) and found that 13 genes were significantly down-regulated in IIIC (T3cN0M0). We also extracted the clinicopathological information of ovarian cancer patients from the Surveillance, Epidemiology, and End Results (SEER) database to distinguish the impact of different surgical methods on prognosis based on logistic regression analysis.
Objectives Advanced ovarian cancer (OC) can undergo different metastatic pathways and is affected by multiple factors. In order to explore the molecular differences in different metastatic pathways, we included influencing factors such as age, histological type, surgical method, diagnosis time, and chemotherapy timing based on the TCGA and SEER databases, and used machine learning AI methods to analyze the timing of surgical lymph node resection in advanced ovarian cancer. At present, we have not found the specific molecular mechanism that determines different metastatic pathways in ovarian cancer patients. This study aims to explain the differential characteristics of ovarian cancer patients with only lymph node metastasis and only implantation metastasis, and provide targeted measures and theoretical basis for effective prevention.
Methods Based on the TCGA database, differential gene analysis was performed on IIIA (T3aN1M0) and IIIC (T3cN0M0), and enrichment analysis of high and low expression (including GO and KEGG) was performed. Kaplan–Meier analysis was also used to analyze the impact of the top three significantly downregulated genes on overall survival (OS) in different ovarian cancer patients.
Results After data screening, we compared the differences between IIIA (T3aN1M0), a total of 3 samples; IIIC (T3cN0M0), a total of 38 samples from the TCGA database, and conducted correlation analysis on the differential genes between the two, which was mainly reflected in the cell vesicle structure, suggesting that when ovarian cancer has implantation metastasis, the downregulation of adhesion molecules promotes the transport of proteins inside and outside the cell by vesicles, leading to changes in the tumor microenvironment. At the same time, machine learning methods (LR, KNN, SVM, XGB, NB, GBM) were used to analyze clinical data from the SEER database. A total of 2,144 ovarian cancer patients who were diagnosed between 2018 and 2020 and met the staging were collected, including 187 (8.72%) of IIIA (T3aN1M0) and 91.28% of IIIC (T3cN0M0); at the same time, as of the follow-up time, a total of 551 patients died (34.57%) and 1,593 patients survived (65.43%). Interestingly, race was not a risk factor affecting survival.
Conclusion This study explains from the molecular cytological level that the reason why ovarian cancer patients have different metastatic pathways is that the genes that regulate vesicle transport are underexpressed and affect the overall survival of patients. The machine learning-based model can accurately predict the timing of lymph node resection in ovarian cancer patients, which is expected to provide a reference for clinical decision-making and help formulate targeted treatment strategies for this population.

PO_244

The role of metabolic reprogramming in ovarian cancer drug resistance

Lin Qiu Manlin Zhang Yang Wang Zhuo Yang*
Department of Gynaecology, Cancer Hospital of Dalian University of Technology, Cancer Hospital of China Medical University, Liaoning Cancer Hospital & Institute

Ovarian cancer remains the most lethal gynecologic malignancy, largely due to the inevitable development of therapeutic resistance. Mounting evidence underscores that profound metabolic reprogramming-a core hallmark of cancer-is not merely a passive adaptation but an active driver of chemoresistance, enabling tumor cells to evade cytotoxicity and sustain survival under therapeutic stress. This review synthesizes critical insights into how ovarian carcinoma cells rewire glucose, lipid, and amino acid metabolism to confer treatment refractoriness.

Key adaptations include enhanced glycolytic flux via hypoxia-inducible factor (HIF)-mediated upregulation of GLUT transporters and rate-limiting enzymes (HK2, PFKFB3, PKM2, LDHA), which sustain Warburg metabolism while promoting DNA repair and autophagy-mediated survival. Concurrent dysregulation of lipid pathways-encompassing de novo lipogenesis driven by fatty acid synthase (FASN), fatty acid oxidation (FAO) potentiated by CPT1A, and CD36/FABP4-mediated scavenging of adipocyte-derived lipids-supports membrane biosynthesis, stemness maintenance, and drug efflux. Furthermore, intensified glutamine dependency fuels tricarboxylic acid (TCA) cycle anaplerosis via α-ketoglutarate and bolsters glutathione-based antioxidant defenses, while serine metabolism perturbations enhance DNA damage tolerance.
Crucially, metabolic symbiosis within the tumor microenvironment (TME) underpins resistance: cancer-associated fibroblasts (CAFs) remodel extracellular matrices and activate pro-glycolytic signaling; adipocytes transfer free fatty acids to fuel β-oxidation; tumor-associated macrophages (TAMs) adopt immunosuppressive phenotypes via lactate-cholesterol crosstalk; and regulatory T cells (Tregs) utilize FAO/OXPHOS to enforce immune evasion. These interconnected metabolic networks create formidable barriers to conventional chemotherapy and targeted agents.
Emerging therapeutic strategies seek to exploit these vulnerabilities through inhibitors targeting GLUTs, HK2, FASN, CPT1A, glutaminase, or CD36, showing promise in preclinical models for resensitizing resistant tumors. However, clinical translation faces hurdles including metabolic plasticity, tumor heterogeneity, and on-target toxicity. Future efforts must prioritize isoform-selective agents, spatial metabolomics-guided combination therapies, and biomarkers identifying metabolic liability windows. Elucidating the dynamic interplay between tumor-intrinsic metabolic rewiring and TME-driven adaptations provides a crucial framework for overcoming therapeutic resistance in ovarian carcinoma.

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卵巢癌患者化疗前后症状特征的潜在的演变趋势分析及护理对策

熊秀
重庆大学附属肿瘤医院

目的 探讨卵巢癌患者化疗前后症状潜在类别随时间变化趋势并进一步分析其影响因素，旨在为临床护理实践提供借鉴。
方法 采取便利抽样法，选取 2024 年 1 月—12 月在重庆市某三级甲等医院妇科就诊的 226 例卵巢癌患者为调查对象，采用一般资料调查表、中文版安德森症状评估量表，对首次化疗前 1～3 d（化疗前，226 例），第 3 次或第 4 次化疗后 7～14 d（化疗后，218 例)的卵巢癌患者症状特征进行潜在的演变趋势分析。结果 218 例卵巢癌患者完成 2 次调查，化疗前后其症状特征可分为高症状困扰组、低症状困扰组 2 个类别。高症状困扰组稳定性强，保持原组的概率为 85.22%。低症状困扰组更倾向于向高症状困扰组转变，转变概率为 26.58%。Logistic 回归分析显示，肿瘤低级别、获取疾病知识最主要途径为网络的患者更容易从高症状困扰组转变为低症状困扰组，而在职、夜间睡眠不满意及从不运动的患者更容易从低症状困扰组转变为高症状困扰组。
结论 卵巢癌患者化疗前后的症状特征存在人群异质性，在职、夜间睡眠不满意及从不运动的卵巢癌患者更容易从低症状困扰组转变为高症状困扰组，提示医护人员早期识别潜在高风险人群，根据不同类别的人群特征实施症状动态监测的基础上，充分运用症状特征的预测作用进行个体化管理，从而减轻症状负担，提高其生活质量。

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卵巢癌类器官构建的影响因素分析及其化疗疗效预测

汪玲 邹冬玲*
重庆大学附属肿瘤医院

研究目的 类器官作为新兴的临床前模型，为肿瘤患者提供了可靠的药物筛选和个性化诊疗的新平台。卵巢癌作为高异质性，高复发耐药性的肿瘤亟需类器官辅助预测其化疗疗效。然而，卵巢癌类器官的构建困境限制了其广泛应用。
研究方法 我们收集了 153 例上皮性卵巢肿瘤患者的肿瘤样本，包括手术及活检样本，总结肿瘤样本实验特征及临床病理特点，包括年龄，分期，病理类型，肿瘤细胞活率等， 通过卡方检验和 Fisher 精确检验分析了患者临床病理及实验特征与类器官构建之间的关联，并进行单变量和多变量逻辑回归分析，以确定影响类器官构建的独立影响因素。我们对 20 个类器官进行了药物筛选，回顾性和前瞻性地预测临床患者的药物反应。
研究结果 从 153 名卵巢肿瘤患者中开发了 153 个类器官，成功率为 57.52%。患者术前 CA153 水平低（OR（95%CI）:3.44（1.39，9.00），P=0.009）和 CA199 水平高（OR（95%CI）:0.20（0.06，0.57），P=0.004）与类器官成功构建相关，而其他临床特征与卵巢肿瘤类器官生成没有显著相关性。亚组分析进一步表明，CA153 和 CA199 是影响类器官构建的两个独立因素。卵巢癌症类器官可以保留原始肿瘤组织的病理和遗传特征，能够以 100%的有效率回顾性预测肿瘤患者前化疗疗效，然而前瞻性预测效率为 80%，可为患者后续治疗提供参考。
研究结果 术前血浆 CA153 和 CA199 水平是影响卵巢肿瘤类器官构建的独立影响因素。大多数卵巢癌类器官对紫杉醇和卡铂的药物反应与临床治疗结果一致，可作为卵巢癌化疗疗效预测的可靠模型。

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卵巢癌生物标志物与治疗新进展综述

叶晔
南通大学

ITGB 家族在卵巢癌中的研究进展：从预后机制到靶向治疗
研究目的 卵巢癌作为致死率最高的妇科恶性肿瘤，约 70%患者确诊时已属晚期，5 年生存率不足 40%，其高复发率与铂类耐药性构成重大临床挑战。本综述聚焦整合素家族（ITGB）在卵巢癌中的预后价值与治疗靶点作用，系统评估基于 ITGB 的新型疗法潜力，为个体化诊疗提供理论支撑。
材料与方法 本研究整合 2024-2025 年多项关键成果。采用多组学技术分析 TCGA-OV、GEO 数据库，通过 limma 包筛选差异基因并运用 LASSO 回归构建 ITGB 预后模型。临床前实验使用 ES2 裸鼠移植瘤模型，结合 shRNA 基因敲降验证 ITGB1/FAK 信号轴功能。临床试验数据涵盖 CAR-T（αPD-1-mesoCAR-T）、TILs（GC203）及双配体药物（CBP-1008）的 I/II 期研究，依据 RECIST 标准评估疗效。创新技术平台包括类器官芯片 OC-plex8 联合 AI 预测药敏性，以及 TIDE 算法评估免疫治疗应答。
结果 生物信息学分析证实 ITGB1、ITGB4、ITGB5、ITGB6、ITGB7 及 ITGB8 在卵巢癌中显著高表达（HR=1.8, p<0.001）。具体机制表现为：ITGB1 通过激活 FAK-SRC-ERK 信号轴缩短患者生存期达 40%；ITGB8 与 αv 形成异二聚体，介导 TGF-β 信号通路诱发免疫逃逸；LINC00839 通过吸附 miR-345-5p 上调促癌因子 HDGF（r=0.70, p<0.001）。基于上述发现构建的预后模型将患者分为高风险组（中位生存期 28 个月）和低风险组（52 个月）。治疗领域取得多项突破：细胞疗法中，双靶点 CAR-T 药物 BZE2203 实现靶病灶完全消失，TILs 药物 GC203 在 18 例复发患者中达到 83.3%疾病控制率；双配体偶联药物 CBP-1008 治疗铂耐药患者的中位生存期达 19.4 个月，显著优于传统方案；联合策略研究发现丹参提取物二氢丹参酮 I 通过抑制 ITGB1/FAK 轴使移植瘤缩小超 50%，同时逆转铂类毒性；

预测技术方面，OC-plex8 类器官芯片联合 AI 使药敏预测与临床疗效一致性达 85%，TIDE 算法证实 ITGB 低风险组对 PD-1 抑制剂敏感性更高。

结论 ITGB 家族（尤其 ITGB1/ITGB8）是卵巢癌关键的预后标志物与治疗靶点，其介导的 FAK-TGF-β 信号轴驱动肿瘤转移与免疫抑制。靶向 ITGB 的细胞疗法（CAR-T/TILs）和双配体药物显著提升难治患者生存获益，而"天然药物-化疗"联合策略可协同增强疗效并降低毒性。未来可深化 ITGB 表达与免疫治疗应答的量化关联，拓展类器官芯片技术在个体化治疗中的应用场景。

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肿瘤相关成纤维细胞源性 circMPP6 通过外泌体传递促进卵巢癌进展及机制研究

韦欣仪 许君芬*
浙江大学医学院附属妇产科医院

研究目的 多数卵巢癌患者首次发现时已发生播散性转移，预后极差，是威胁全球女性健康的主要恶性肿瘤之一。肿瘤微环境在卵巢癌发展中扮演着关键角色，其中，肿瘤相关成纤维细胞（ CAFs）是重要组成部分。最近研究发现 CAFs 分泌的外泌体中包裹环状 RNA（circRNA）起到关键作用。因此，深入探究肿瘤微环境中 circRNA 的调控机制，寻找新的分子靶点对提升卵巢癌诊治能力至关重要。本研究旨在深入探究卵巢癌转移组织中显著高表达的 CAFs 源性 circMPP6 在卵巢癌进展中的作用及机制，为寻找卵巢癌的诊治靶点提供新的视角。

材料与方法 本研究通过对高级别浆液性卵巢癌原位组织与配对大网膜转移组织进行全转录组测序，通过 RT-qPCR、RNA 荧光原位杂交技术筛选并验证 circRNA。通过 DNA 电泳、Sanger 测序、RNA 稳定性实验和 RNA 酶消化实验验证卵巢癌中 circMPP6 的存在和环状结构。通过 CCK-8 实验、细胞凋亡实验、Transwell 实验等探究调控 circMPP6 后 CAFs 功能表型改变及卵巢癌细胞的恶性生物学行为改变。RNA pull-down 实验、RIP 实验、Co-IP 实验、质谱分析、RNA-seq、结合域截短质粒、ActD 实验、RT-qPCR、Western blot 实验探究 circMPP6 在 CAFs 与卵巢癌细胞内的机制。利用共培养体系阐明在 CAFs 对卵巢癌恶性生物学行为的影响。采用裸鼠皮下成瘤、腹腔瘤、外泌体体内处理等实验验证体外实验的结果。

结果 高级别浆液性卵巢癌大网膜转移组织高表达 circMPP6 主要定位于 CAFs，其次是靠近 CAFs 的肿瘤细胞，而在远离 CAFs 的肿瘤细胞中很少表达。高表达 circMPP6 的患者总体生存期与无进展生存期更短。CAFs 中敲降 circMPP6 的表达能够显著抑制 CAFs 细胞的增殖和侵袭能力，同时能够抑制卵巢癌细胞的增殖和迁移侵袭能力。CAFs 中 circMPP6 与 hnRNPA2B1 蛋白结合，hnRNPA2B1 促进 circMPP6 转运进 CAFs 分泌的外泌体中。CAFs 源性的 circMPP6 能够通过外泌体传递至卵巢癌细胞；卵巢癌细胞摄取外泌体后 circMPP6 表达增加，增强了肿瘤细胞的增殖和转移能力。在卵巢癌细胞内，细胞核内的 circMPP6 通过增强 SFPQ 与 NONO 的结合，促进对下游 ADAM22 mRNA 的加工成熟，细胞质中的 circMPP6 通过结合 EEF1A2 增加 ADAM22 的表达，激活 TGF-β/SMAD 信号通路，进而促进卵巢癌细胞的增殖和迁移侵袭能力。

结论 CAFs 源性的 circMPP6 能通过外泌体将传递至肿瘤细胞，增加肿瘤细胞内 ADAM22 的表达，激活 TGF-β/SMAD 信号通路，促进卵巢癌细胞的增殖转移。我们的研究结果首次揭示了 CAFs 源性 circMPP6 在卵巢癌进展中的促进作用及其潜在分子机制，为卵巢癌治疗和预后分析提供新思路。

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Magneto-Acoustic Cascade Nanorobots Overcome Ovarian Cancer Drug Resistance via Mechanical Microenvironment Reprogramming

Shuangshuang Cheng
Department of Obstetrics and Gynecology, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Hubei, 430022, Wuhan, China.

Purpose Ovarian cancer exhibits the highest mortality rate among gynecological malignancies, with treatment failure primarily stemming from chemotherapy resistance. Studies confirm that the dense physical barrier formed by aberrant tumor mechanical microenvironment is a key factor impeding drug efficacy. This study aims to construct multifunctional magneto-acoustic dual-responsive nanoparticles (Fe_3O_4@$CaCO_3$/TCPP-HA) targeting the mechanical characteristics of ovarian cancer microenvironment. Through mechano-chemical synergy, we seek to disrupt the tumor physical barrier and reverse drug resistance, ultimately improving ovarian cancer prognosis.

Materials and Methods Fe_3O_4@$CaCO_3$/TCPP nanoparticles were synthesized via hydrothermal method, with hyaluronic acid (HA) conjugated via EDC/NHS chemistry for CD44 receptor targeting. Nanoparticle morphology and catalytic properties were characterized using transmission electron microscopy (TEM), scanning electron microscopy (SEM), and X-ray diffraction. Tissue Young's modulus was measured by atomic force microscopy (AFM). A collagen-coated polyacrylamide hydrogel (PA gel) system was established to simulate soft/rigid substrate microenvironments in vitro. Transwell assays, immunofluorescence, western blotting, and immunohistochemistry were employed to systematically evaluate the biological effects of magneto-acoustic responsive nanoparticles in both in vitro and in vivo models.

Results The developed magneto-acoustic dual-responsive nanorobots achieved active tumor targeting under external magnetic guidance. The $CaCO_3$ component underwent acid-responsive degradation, releasing Ca^{2+} to induce tumor cell calcium overload. Ultrasound activation simultaneously generated substantial reactive oxygen species (ROS) while depleting glutathione (GSH), effectively reversing chemotherapy resistance. The mechanical stress produced by this nanosystem significantly disrupted the tumor stromal barrier, ameliorating hypoxic and immunosuppressive microenvironments through mechano-chemical coordination.

Conclusions Magneto-acoustic dual-responsive nanoparticles successfully reprogrammed the mechanical microenvironment of ovarian cancer via a tripartite mechanism: calcium overload-induced cellular damage, ultrasound cavitation-mediated mechanical modulation, and oxidative stress-driven resistance reversal. This strategy significantly inhibited primary and metastatic tumor growth, providing an innovative therapeutic approach for overcoming ovarian cancer drug resistance.

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miR200c 与其靶向 circRNA 调控 PPM1F 对上皮性卵巢癌生长转移的作用机制研究

曾智萍[1] 徐新燕[2] 魏梦珂[1] 魏乾坤[3] 李凤娟[4] 张晶[2*]
1.新疆医科大学 2.乌鲁木齐市妇幼保健院
3.石河子大学 4.乌鲁木齐市友爱医院

目的 探讨 PPM1F、miR200c 及其上游靶 circRNA 对上皮性卵巢癌细胞生长、转移等的影响及靶 circRNA 和 miR200c 和 PPM1F 之间的调控关系。
方法 通过 miRanda 软件分析 circBase 数据库中的 circRNA 和 mirBase 数据库中的 miRNA，预测 miRNA 的靶 circRNA；qRT-PCR 实验检测转染后的 miR200c、靶 circRNA 及 PM1F 在卵巢癌 SKOV3 细胞和卵巢癌 A2780 细胞中的表达水平；划痕实验计算细胞迁移的距离；Transwell 实验统计细胞侵袭能力；凋亡及细胞周期实验检测其凋亡和细胞增殖情况；CCK-8 实验计算细胞活力；Western blot 实验检测靶 circRNA、miR200c 和 PPMIF 表达关系。
结果 与 NC 组、Control 组比较，circ-PHC3-OE 组的细胞迁移率、细胞侵袭率、细胞活力、PPMIF 蛋白表达量明显增加，细胞凋亡明显降低；hsa-miR-200c-3p mimics 组细胞迁移率、细胞侵袭率、细胞活力、PPMIF 蛋白表达量明显降低，细胞凋亡明显增加，差异具统计学意义(P＜0.05）。与 hsa-miR-200c-3p mimics 组比较，circ-PHC3-OE + hsa-miR-200c-3p mimics 组的细胞迁移率、细胞侵袭率、细胞活力、PPMIF 蛋白表达量明显增加，细胞凋亡明显降低，差异具统计学意义 (P＜0.05）。miR-200c 与 PPM1F 在上皮性卵巢癌细胞中的表达呈负相关。
结论 靶 circRNA 通过吸附 miR-200c,上调肿瘤的增殖、侵袭、迁移，下调肿瘤细胞的凋亡，从而促进上皮性卵巢癌的进展；miR-200c 通过负调控 PPM1F 的表达遏制上皮性卵巢癌的进展。

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A cfDNA-Based Liquid Biopsy Using Combined Epigenetic and Fragmentomic Profiling to classify Malignant Ovarian Carcinoma from Benign Masses

Peiye Shen[1] Shuang Gan[2] Hang Dong[2] Haoran Tang[2] Shidong Jia[2] Guanglei Zhuang[1] Xia Yin[1*]
1.Ren Ji Hospital, Shanghai Jiao Tong University School of Medicine
2.Huidu (Shanghai) Medical Sciences

Purpose High-grade serous ovarian carcinoma (HGSOC), the most aggressive ovarian cancer subtype, is frequently diagnosed at advanced stages due to the lack of reliable early detection tools. We developed a multimodal liquid biopsy approach that integrates cell-free DNA (cfDNA) epigenomics and fragmentation profiles to distinguish malignant ovarian cancers from benign masses.
Materials and Methods This study included a total of 169 tissue samples, comprising 69 malignant ovarian tumor tissues, 50 benign lesion tissues, and 50 fallopian tube epithelial tissues. Plasma samples were collected from two independent clinical centers, including 119 samples from patients with malignant ovarian cancer and 100 samples from patients with benign pelvic lesions. All tissue and plasma samples were analyzed using the PredicineEPIC assay, a whole-genome methylation sequencing platform designed for high-resolution methylome profiling. To identify tumor-specific differentially methylated regions (DMRs), comparative methylation analysis was first performed across the three tissue groups. The tumor-specific DMRs were identified as tumor-associated epigenetic markers which were subsequently tracked in plasma samples to assess their methylation levels in cfDNA. In addition to methylation profiling, cfDNA fragmentomic features were characterized, including

transcription factor binding site (TFBS) coverage, fragment size distribution and copy number variation (CNA) burden. These multi-layered features were integrated to build a classification model for distinguishing malignant from benign cases. Feature selection was performed using machine learning approaches, and model training was conducted with leave-one-out cross-validation (LOOCV) to prevent overfitting and ensure robustness. The final classifier was validated in an independent test cohort to evaluate its performance.

Results From the tissue-based methylation profiling, a total of 5612 tumor-specific differentially methylated regions (DMRs) were identified. Among these, 4078 DMRs showed significant differential methylation signals in plasma cell-free DNA (cfDNA), indicating strong tissue-to-liquid biopsy concordance. In addition, the overall copy number alteration (CNA) burden in cfDNA was significantly higher in malignant cases compared to benign controls ($p = 0.002$), further supporting the utility of cfDNA-based genomic profiling for cancer detection. Fragmentomic analysis revealed notable differences at transcription factor binding sites, including those of ZSCAN16, GRHL2, and HMGA1, which are implicated in oncogenic processes. Tumor-derived cfDNA exhibited reduced coverage and altered fragment size distributions at these sites, suggesting aberrant gene regulatory activity in cancer patients. We integrated both methylation and fragmentomic features into an XGBoost-based model for classification. In the independent validation cohort, the model achieved an area under the curve (AUC) of 94%, with a sensitivity of 91% and specificity of 86%, highlighting its robust diagnostic performance. This molecular classifier outperformed the traditional clinical biomarker CA125, which yielded an AUC of 84% in the same cohort, demonstrating the added diagnostic value of combining methylation and fragmentomic features for improved cancer detection.

Conclusions Combining cfDNA epigenomics and fragmentation signatures enables highly accurate discrimination of malignant ovarian cancers from benign masses, providing a powerful tool for non-invasive diagnostics. This multimodal approach outperforms single-modality diagnostics, underscoring the clinical potential of cfDNA-based assays for non-invasive triaging of adnexal abnormalities and early cancer detection. The integration of multiple molecular features enhances diagnostic precision, paving the way for more reliable and less invasive screening strategies in ovarian cancer management.

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基于生物信息学技术确立铁硫簇支架蛋白 ISCU 在上皮性卵巢癌中的预后价值

滑天* 许盼盼
邢台市人民医院

目的 卵巢癌（Ovarian Cancer, OC）是导致女性恶性肿瘤相关死亡的主要原因之一，目前仍缺乏可靠的分子生物标志物用于预测其预后。铁硫簇支架蛋白 ISCU 是 miR-210 的下游靶点，已被认为是恶性肿瘤治疗的潜在生物靶点，但其在卵巢癌中的研究仍较为有限。本研究旨在探讨 ISCU 的表达水平与上皮性卵巢癌（Epithelial Ovarian Cancer, EOC）患者预后之间的相关性。

方法 1. 通过分析 TCGA 数据库和 GTEx 数据库中 EOC 组织与正常卵巢组织的 mRNA 表达谱数据，筛选出 EOC 与正常卵巢组织中的差异表达基因（DEGs）列表；随后结合生存分析筛选出与 EOC 预后显著相关的基因，并基于文献回顾最终确定 ISCU 为研究目标。2. 利用 GEPIA 2、Human Protein Atlas、Oncomine、LinkedOmics、Kaplan–Meier Plotter、STRING、CytoHubba 插件及 TIMER 等数据库分析 ISCU 在 EOC 中的潜在生物学功能。3. 临床样本验证：收集邢台市人民医院 46 例 EOC 患者的肿瘤组织样本及 15 例良性卵巢疾病患者的正常卵巢组织样本，采用实时荧光定量 PCR（Real-time quantitative，RT-qPCR）和免疫组织化学（Immunohisteochemistry，IHC）检测组织样本中 ISCU mRNA 及蛋白表达水平，并结合患者随访资料分析其与临床预后的关系；4. 体外细胞实验：构建 ISCU 低表达的 OVCAR8 和 OVCAR3 细胞系，通过体外细胞计数（Cell Counting Kit-8，CCK-8）实验及细胞侵袭（Transwell 小室）实验评估 ISCU 对 EOC 细胞增殖、集落形成能力和侵袭能力的影响。

结果 1. 根据数据库检索发现，与正常卵巢组织相比，ISCU 的 mRNA 表达水平在 EOC 组织中显著上调，并与患者的不良临床结局相关。研究也检索出了 ISCU 相关的蛋白质相互作用网络及枢纽基因，通过分析证实了 ISCU 在 EOC 的发生发展中具有重要的生物学功能。2. qPCR 和 IHC 结果证实，与正常卵巢组织相比，EOC 组织中 ISCU 的 mRNA 和蛋白表达水平均明显升高。Kaplan-Meier 生存分析表明，ISCU 高表达患者的无进展生存期（Progression-Free Survival, PFS）和总生存期（Overall Survival, OS）显著低于低表达患者。3. 在细胞实验中，CCK-8 和 Transwell 小室实验结果显示下调 ISCU 显著抑制了两种细胞系的增殖、集落形成能力和侵袭能力。
结果 本研究证实 ISCU 是上皮性卵巢癌潜在的预后生物标志物，并参与调控肿瘤细胞的增殖与侵袭过程，提示其可能成为 EOC 预后评估和治疗干预的新靶点。

PO_253

基于生物信息学技术确立核因子-κB 抑制蛋白 B 在上皮性卵巢癌中的临床意义

滑天* 文蓓
邢台市人民医院

目的 上皮性卵巢癌(epithelial ovarian cancer, EOC)是女性生殖道常见的恶性肿瘤之一，发病率仅次于子宫颈癌和子宫体癌而列居第三位，死亡率却位列妇科肿瘤首位。核因子-κB(Nuclear factor-κB, NF-κB)是指在炎症和先天免疫反应中发挥关键作用的一个“快速作用”转录因子家族。现有研究证实其抑制蛋白 B(NFKBIB)与肿瘤相关的作用机制主要是通过 NF-κB 通路来实现的，此通路通常与血管生成和促进肿瘤生长和发展有关。本研究旨在探讨 NFKBIB 与 EOC 发生发展以及预后之间的相关性。
方法 1. 通过分析 TCGA 数据库和 GTEx 数据库中 EOC 组织与正常卵巢组织的 mRNA 表达谱数据，筛选出 EOC 与正常卵巢组织中的差异表达基因(DEGs）列表；随后结合生存分析筛选出与 EOC 预后显著相关的基因，并基于文献回顾最终确定 NFKBIB 为研究目标。2. 使用 GEPIA 2、Human Protein Atlas、Oncomine、LinkedOmics、Kaplan–Meier Plotter、STRING、CytoHubba plug-in 和 TIMER 等数据库来确定 EOC 中 NFKBIB 表达、与患者预后以及免疫细胞浸润之间的关系。3. 临床样本验证：于邢台市人民医院收集 45 例 EOC 患者的肿瘤组织标本及 15 例与 EOC 患者同期的因卵巢良性疾病行手术治疗的患者的卵巢组织标本，采用实时荧光定量 PCR 和免疫组织化学检测组织样本中 NFKBIB 的 mRNA 及蛋白表达水平，并对采集样本患者进行为期 36 个月的随访，分析其与 EOC 患者预后的关系；4. 体外细胞实验：构建 NFKBIB 低表达的 OVCAR8 和 OVCAR3 细胞系，通过体外细胞计数（CCK-8）实验及细胞侵袭 Transwell 实验评估 NFKBIB 对 EOC 细胞增殖、集落形成能力和侵袭能力的影响。
结果 1. 检测生物信息学数据库发现，与正常卵巢组织相比，NFKBIB 基因的 mRNA 表达水平在 EOC 组织中显著上调，并与患者的不良临床结局相关。2. 临床样本验证中，qPCR 和 IHC 结果证实，与正常卵巢组织相比，EOC 组织中 NFKBIB 基因的 mRNA 和蛋白表达水平均明显升高。Kaplan-Meier 生存分析表明，NFKBIB 基因高表达患者的无进展生存期（Progression-Free Survival, PFS）和总生存期（Overall Survival, OS）显著低于低表达患者。3. 在细胞实验中，CCK-8 和 Transwell 小室实验结果显示下调 NFKBIB 显著抑制了两种细胞系的增殖、集落形成能力和侵袭能力。
结果 本研究证实 NFKBIB 对于上皮性卵巢癌发生发展及预后存在重要临床意义，参与调控肿瘤细胞的增殖与侵袭过程，NFKBIB 或可成为 EOC 预测预后的生物标志物及新的治疗干预的药物靶点。

PO_254

To analyze the expression and significance of Eukaryotic Translation Initiation Factor 4E Family Member 3 in epithelial ovarian cancer based on bioinformatics technology

Tian Hua* Yutong Zhu
Xingtai People's Hospital

Epithelial Ovarian Cancer (EOC), a high-incidence malignant tumor of the female reproductive system, ranks third in incidence among gynecological malignancies (only after cervical cancer and endometrial cancer). However, its mortality rate has long remained the highest, with a 5-year survival rate of less than 40%. This is primarily attributed to the clinical dilemmas of difficulties in early diagnosis and the propensity for recurrence and drug resistance in advanced stages. Currently, there is an urgent clinical need for biomarkers with high specificity and sensitivity to optimize EOC diagnostic strategies, guide treatment selection, and accurately assess prognosis. Eukaryotic Translation Initiation Factor 4E Family Member 3 (EIF4E3), a key member of the mRNA cap-binding protein family, plays a central role in regulating the translation initiation process. Existing studies have confirmed that EIF4E3 is closely associated with tumorigenesis, progression, and prognosis in other cancer types, yet its expression pattern and clinical significance in EOC remain unclear.This study aims to explore the expression and significance of EIF4E3 in EOC.

Materials and Methods This study systematically explored the clinical value and molecular mechanisms of EIF4E3 in EOC through a multi-dimensional experimental design, including the following steps: 1.We analyzed mRNA expression profile data of EOC tissues and normal ovarian tissues from the TCGA and GTEx databases to screen out the list of differentially expressed genes (DEGs) between EOC and normal ovarian tissues. Subsequently, we combined survival analysis to screen out genes significantly associated with EOC prognosis, and finally identified EIF4E3 as the research target based on literature review. 2. Quantitative real-time polymerase chain reaction (qRT-PCR) and immunohistochemistry (IHC) were used to detect the mRNA and protein expression levels of EIF4E3 in 48 collected EOC tissues and 15 normal ovarian tissues. The relationship between EIF4E3 expression and the occurrence, development, and clinical prognosis of EOC was analyzed in combination with patient follow-up data. 3. Using bioinformatics techniques, independent prognostic factors were screened through Kaplan-Meier survival analysis, univariate Cox regression model, and multivariate Cox regression model. A nomogram prediction model based on EIF4E3 was constructed, and its prediction accuracy was verified using calibration curves. Meanwhile, databases such as GEPIA 2, Human Protein Atlas, Oncomine, LinkedOmics, Kaplan–Meier Plotter, STRING, CytoHubba plug-in, and TIMER were used to explore the biological functions of EIF4E3 in EOC. 4. SKOV3 and OVCAR8 cell lines were transfected with EIF4E3 knockdown plasmids. Cell proliferation ability was detected using the Cell Counting Kit-8 (CCK-8) assay, and cell invasion and migration abilities were measured via Transwell chambers.

Results The study found that EIF4E3 was significantly downregulated in EOC tissues. Survival analysis showed that patients with low EIF4E3 expression had significantly shorter progression-free survival (PFS) and overall survival (OS) than those with high expression ($P < 0.05$). Multivariate analysis revealed that EIF4E3 was an independent factor predicting favorable prognosis in EOC patients. In vitro experiments demonstrated that EIF4E3 could inhibit the proliferation, invasion, and migration of ovarian cancer cells.

Conclusion This study is the first to confirm that EIF4E3 functions as a tumor suppressor gene in EOC. Its low expression is closely associated with malignant progression and poor prognosis of EOC, suggesting that EIF4E3 holds promise as a potential biomarker for EOC prognostic assessment and a novel target for therapeutic intervention.

PO_255

靶向 EGFR 的 ADC 药物逆转卵巢癌耐药的可行性研究

闫昕宇 汪辉*
浙江大学医学院附属妇产科医院

研究目的 卵巢癌是全球女性癌症死亡的第五大原因，2020 年新发病例约 31 万例。上皮性卵巢癌（EOC）的标准一线治疗为肿瘤细胞减灭术联合铂类化疗，辅以抗血管生成药物及 PARP 抑制剂可改善部分患者生存。然而，因化疗耐药与早期筛查手段缺乏，患者总体生存率仍不理想。铂耐药卵巢癌（PROC）患者治疗选择有限，传统化疗反应率通常低于 15%，预后极差；同时 PARP 抑制剂耐药进一步限制了靶向治疗获益。因此，开发克服铂类及 PARP 抑制剂耐药的新策略是临床迫切需求。本研究旨在探索靶向表皮生长因子受体（EGFR）的抗体偶联药物（ADC）通过特异性靶向递送机制逆转卵巢癌耐药的可行性，为耐药患者提供新治疗方向。 **材料与方法** 构建靶向 EGFR 的 ADC（EGFR-vc-MMAE）及其同型对照药物 isotope-vc-MMAE。在亲本细胞系（A2780, SKOV3, OVCAR3）和耐药细胞系中评估了 EGFR 的表达水平。同时通过进行细胞毒性效应和诱导凋亡能力进行 ADC 的杀伤功能验证。
结果 EGFR 在所有的亲本和耐药细胞系中均持续过表达。EGFR-vc-MMAE 通过增加耐药细胞的凋亡表现出显著的细胞毒性作用。同型对照 ADC 仅显示出微弱的细胞毒性，证实了 EGFR 靶向 ADC 作用机制的特异性。
结论 我们的研究结果表明，靶向 EGFR 的 ADC 能有效克服耐药性并展现出细胞毒性效应。这种方法代表了一种有前景的治疗策略，值得进一步深入研究，后续需开展动物模型体内药效及毒性验证，推动临床转化。

PO_256

Multi-omics profiling coupled with functional assays elucidates the role of cancer stemness in ovarian cancer and yields a machine-learning model for forecasting immunotherapy outcomes.

Zhaoyue Luo[1] Baolin Zhang[1,2] Huishu Xu[1] Shinan Zhang[1] Ningxiao Jiang[1] Yixuan Ma[1] Lei Han[1*]
1.Binzhou Medical University Hospital
2.Binzhou Central Hospital

Objective The heterogeneity of cancer poses a significant hurdle in forecasting its reaction to immunotherapy, emphasizing the necessity of discovering trustworthy biomarkers for assessment. However, the intricate mechanisms by which cancer stemness contributes to resistance against immune checkpoint inhibitors (ICIs) are not yet fully understood.
Methods The identification of the key genes specific to ovarian cancer (OV) was achieved through genome-scale CRISPR screening using RNA sequencing data from the Achilles project. The new cancer stemness index (CSI) was measured using 13 publicly accessible OV transcriptomic datasets, 7 pan-cancer ICI transcriptomic cohorts, and 1 single-cell RNA dataset from melanoma patients treated with PD-1 therapy. To reveal the role of small nucleolar ribonucleoprotein polypeptide E (SNRPE) in the tumor microenvironment (TME), it was necessary to collect and carefully examine the OV single-cell RNA dataset. In vitro experiments verified the role of SNRPE in promoting the proliferation and migration of ovarian cancer cells.
Results After analyzing extensive ovarian cancer datasets, a specific gene set affecting tumor stemness was detected. The study also found that in single - cell ICI cohorts, there is a negative association between cancer stemness and the advantages of ICI treatment. Based on this identified gene set, the CSI was developed. It is a remarkable tool that applies advanced machine learning

methods to predict the prognosis and responses to immunotherapy in ovarian cancer patients.The accuracy of the CSI was further confirmed by applying PD1/PD-L1 ICI transcriptomic cohorts, with a mean AUC exceeding 0.8 for predicting tumor progression and immunotherapy benefits.As opposed to the current immunotherapy and prognosis markers, CSI presented stronger predictive capacities throughout multiple datasets.The study discovered that the overexpression of SNRPE is involved in the reshaping of the tumor microenvironment (TME) and facilitates the escape of malignant cells from immune system detection. Moreover, SNRPE has the potential to act as an innovative biomarker for forecasting the response to immunotherapy.
Conclusion In the research, a robust connection has been established between cancer stemness and the response to immunotherapy. This finding yields valuable understandings for creating efficient methods to tackle immune evasion by aiming at the regulation of genes linked to cellular stemness.

PO_257

Multi-omics techniques clarify the immune microenvironment and reveal therapeutic approaches for ovarian cancer patients after surgery

Yixuan Ma[1] Baolin Zhang[1,2] Huishu Xu[1] Shinan Zhang[1] Ningxiao Jiang[1] Lei Han[1*] Chaoyue Luo[1]
1.Binzhou Medical University Hospital2.Binzhou Central Hospital, Binzhou City

Objective As a highly aggressive and often fatal disease , ovarian cancer (OC) is frequently undetected before metastasis. The classic treatment methods for OC include postoperative chemotherapy. However, despite the effectiveness of the surgery, recurrence is still very common. Unfortunately, there is currently no ideal predictive model to predict the progression and drug sensitivity of patients with OC after surgery. The pattern of cell death plays an important role in tumor progression. Therefore, our aim is to study their potential as prognostic and drug sensitivity indicators for postoperative OC.
Materials and Methods A total of 12 programmed cell death (PCD) patterns were adopted to construct new classification and prognosis models. A large amount of transcriptomic, genomic and clinical information was collected from the Cancer Genome Atlas (TCGA) Program -OV, GSE9891, GSE26712, GSE49997 and GSE63885. Furthermore, the single-cell transcriptome data GSE210347 was obtained from the Gene Expression Synthesis (GEO) database for subsequent analysis.
Results In this study, a new PCD classification was adopted to conduct phenotypic analysis on postoperative OC patients, revealing that patients in group 1 showed higher sensitivity to immune-based therapy, along with high expression of chemokines, interleukins, interferons and checkpoints. Meanwhile, with the help of machine learning algorithms, the Programmed Cell Death Index (PCDI) was established using the characteristics of eight genes. Patients with high PCDI have a poor prognosis after OC surgery. Furthermore, we also found that patients with low PCDI may show sensitivity to immunotherapy, while patients with high PCDI may have increased responsiveness to tyrosine kinase inhibitors.
Conclusions This study provides a new PCD model and nomogram, which can effectively predict the clinical prognosis and drug sensitivity of patients with OC after surgery.

PO_258

单细胞和转录组数据联合鉴定卵巢癌中 MPT 驱动坏死相关的潜在预后基因

龚姗 姜丽*
秦皇岛市第一医院

研究目的 线粒体通透性转变（Mitochondrial permeability transition, MPT）驱动的坏死是一种由线粒体膜通透性改变而引起的细胞死亡，在癌症的治疗等发面起着重要的作用。然而，MPT 驱动坏死相关基因（MPT driven necrosis-related genes, MPT-RGs）尚未在卵巢癌（ovarian cancer, OC）中进行过研究。因此，我们研究的目的是探讨线粒体通透性改变驱动坏死相关基因在卵巢癌的作用及预后的潜力，并探索其相关的生物学途径和免疫微环境的关系，旨在为卵巢癌的早期诊断和治疗提供新的理论依据。
材料及方法 基于 GEO 和 TCGA 数据库获取卵巢癌患者的转录组数据，基于 GSEA 数据库获取 MPT-RGs。通过将差异表达分析的结果与加权基因共表达网络分析 (WGCNA) 的结果进行交集，初步筛选出候选基因。随后，利用回归分析来识别生物标志物并构建预后风险模型，并对该模型进行了评估。最后，开展功能富集分析、免疫浸润分析、单细胞 RNA 测序分析、基因表达分析以及逆转录定量 PCR (RT-qPCR) 分析，以进一步探究 OC 的潜在生物学机制。
结果 IGF2、MTMR9、SLC6A12、CCHCR1、PITPNM1 和 FKBP8 被鉴定为与 OC 预后相关的 MPT 驱动坏死相关生物标志物，其中 MTMR9 在 OC 肿瘤样本中的表达明显较低，而其他生物标志物的表达则明显升高。进一步的 RT-qPCR 分析显示，5 种生物标志物在 OC 样品中表达显著升高（MTMR9 除外）。基于生物标志物构建并评估一个预后风险模型，分为高风险（HRG）和低风险（LRG）组，HRG 患者的生存概率更低。HRG 和 LRG 患者在牛磺酸代谢、亚油酸代谢等途径上的显著富集。此外，我们发现 9 种浸润程度存在显著差异的免疫细胞类型，其中 MTMR9 的表达与巨噬细胞浸润呈正相关。单细胞分析显示成纤维细胞、T 细胞和平滑肌细胞/肌成纤维细胞是 OC 的关键细胞类型。另外，在成纤维细胞和平滑肌细胞/肌成纤维细胞分化过程中，观察到生物标志物的动态表达模式，IGF2 的表达在分化后期逐渐增加。**结论** 本研究鉴定出 6 个卵巢癌中 MPT 驱动坏死相关的潜在预后基因，并成功构建预后风险模型，其可能是 OC 患者个体化治疗的可靠预后生物标志物。

PO_259

卵巢癌肿瘤细胞减灭术后辅助治疗--肿瘤胞内基因编辑致死系统的临床应用基础研究

刘冰
大连理工大学附属中心医院

研究目的 卵巢癌术后复发及耐药性问题是当前临床治疗面临的核心挑战。传统治疗手段（手术、化疗）以及新兴疗法（靶向药物、免疫治疗）在清除微转移灶和应对化疗耐药方面仍存在显著局限。为此，我们提出一种基于“胞内基因编辑致死”机制的卵巢癌精准治疗策略，并聚焦于“高效基因编辑工具”与“多级靶向递送系统”的协同构建。
材料与方法 1.基因编辑工具：解析基因编辑元件作用的分子机制，开发高精度基因编辑核酸酶，通过 CRIPSR 核酸酶半理性设计及定向进化策略和方法，开发新型 Cas 核酸酶；2.RNP 装载/控释：构建“多酚-蛋白”结构的复合纳米载体，通过酚羟基与蛋白之间的氢键、疏水作用及离子相互作用，以 RNP 蛋白为核心构建纳米复合物实现 RNP 高效率装载，同时肿瘤微环境（高 GSH/低 pH）致使多酚/蛋白相互作用被削弱导致纳米载体崩解，实现 RNP 环境控制高效释放；3.胞内靶向呈递：提取卵巢肿瘤细胞

膜，通过包覆/杂化策略构建同源靶向递送系统，利用肿瘤自身细胞膜的靶标物质（如膜蛋白、糖等），实现复合载体的跨“组织-细胞”主动靶向呈递；4.原位药物储库：以良好生物相容性的高分子材料制备局部缓释的药物储库，通过控释/缓释凝胶-溶胶转换，实现跨“材料-组织”的被动靶向呈递。

结果 新型 Cas 核酸酶与现有核酸酶相比靶向识别位点增加约 20%、靶向特异性提高超 24 倍，该酶与 gRNA 形成 RNP 复合物，可实现高效基因编辑；纳米载体的蛋白包封率 40%左右，肿瘤微环境释放率（12h）80%左右；同源靶向载体对于 SK-OV-3 胞内呈递相比裸纳米载体增强 7 倍，对于 SK-OV-3 细胞比 HUVEC 细胞胞内呈递增强 3-4 倍；制备具有良好组织———材料黏附界面的水凝胶敷料。

结论 在既有研究基础上，后续研究将系统验证该编辑致死系统在杀伤肿瘤细胞方面的潜力。相关研究有望为卵巢癌提供新的治疗路径，也为腹腔转移性肿瘤建立普适性治疗框架。

PO_260

调节 I 型干扰素增强 BRCAwt 卵巢癌铂类化疗敏感性的机制研究

王雨濛[1] 夏百荣[1,2*]
1.蚌埠医科大学研究生院 2.中国科学技术大学附属第一医院

背景 临床数据显示，BRCA 突变（BRCAm）卵巢癌患者预后显著优于 BRCA 未突变（BRCAwt）患者，且其肿瘤微环境中 I 型干扰素（IFN-I）表达水平显著升高，提示 IFN-I 可能与 BRCAm 患者的化疗敏感性相关。本研究旨在探索通过调节 IFN-I 信号增强 BRCAwt 卵巢癌细胞对铂类化疗的敏感性，并阐明其潜在分子机制。

方法 采用 BRCAwt 卵巢癌细胞系，通过外源性 IFN-I 处理，结合铂类处理后检测细胞活力、凋亡率及克隆形成能力，评估 IFN-I 对铂类敏感性的影响。利用 Western blot、qPCR 及 RNA 测序分析 IFN-I 处理后 DNA 损伤修复通路及免疫原性相关分子的表达变化，探究其增敏机制。此外，通过构建 BRCAwt 卵巢癌异种移植模型，验证 IFN-I 联合铂类化疗的体内疗效。

结果 初步实验显示，外源性 IFN-I 处理可显著降低 BRCAwt 卵巢癌细胞对顺铂的 IC50 值（$P<0.05$），增加凋亡细胞比例及 γH2AX 焦点形成，提示 IFN-I 可增强铂类诱导的 DNA 损伤。机制上，IFN-I 处理可激活 JAK-STAT 通路，下调 DNA 损伤修复相关基因的表达，并上调免疫原性细胞死亡标志物。动物实验中，IFN-I 联合顺铂组较单药顺铂组肿瘤体积显著缩小（$P<0.01$），生存期延长。

结论 调节 IFN-I 信号可能通过抑制 DNA 损伤修复、增强免疫原性效应，从而提高 BRCAwt 卵巢癌对铂类化疗的敏感性。本研究为改善 BRCAwt 卵巢癌患者的化疗疗效提供了潜在干预靶点及实验依据。

PO_261

脂肪细胞 Perilipin 2 介导的 CXCL12/CXCR4 信号轴在网膜微环境中促进卵巢癌细胞的转移

徐明霞 王新宇*
浙江大学医学院附属第一医院

研究目的 高级别浆液性卵巢癌（HGSOC）是致死率最高的卵巢癌亚型，具有显著的大网膜转移倾向，这与大网膜中复杂的肿瘤微环境密切相关。网膜微环境中的肿瘤相关脂肪细胞具有促侵袭及促转移特性，但其与卵巢癌细胞之间的交互机制尚不明晰。本研究旨在深入解析网膜肿瘤微环境中卵巢癌细胞与脂肪细胞间的互作机制，并探索靶向脂肪细胞的治疗新策略。

材料与方法 收集高级别浆液性卵巢癌（HGSOC）患者的大网膜转移病灶，采用激光显微切割技术分离近端脂肪细胞与远端脂肪细胞，并进行空间蛋白组测序以分析脂肪细胞的蛋白质表达谱。在脂肪细

胞分化实验中， 3T3-L1 前脂肪细胞用成脂诱导液处理 6 天以诱导为成熟脂肪细胞。通过慢病毒感染构建稳转细胞株：分别使用携带小鼠 PLIN2 shRNA 或 PLIN2 cDNA 的慢病毒转染 3T3-L1 细胞，嘌呤霉素连续筛选一周以构建 PLIN2 低表达及过表达细胞株。
结果 空间蛋白组分析结果显示，近端脂肪细胞中脂滴包被蛋白 2（PLIN2）的表达显著高于远端脂肪细胞。共培养实验表明，与对照组相比，脂肪细胞中过表达 PLIN2 可促进卵巢癌细胞的迁移与侵袭，而敲低 PLIN2 则抑制该效应。趋化实验进一步证实，PLIN2 过表达显著增强脂肪细胞对卵巢癌细胞的趋化作用，反之， 敲低 PLIN2 会削弱该作用。机制研究表明，PLIN2 敲低可降低脂肪细胞中趋化因子 CXCL12 的分泌水平，从而导致卵巢癌细胞中 CXCR4 受体激活减弱。
结论 本研究结果表明，脂肪细胞 PLIN2 可显著促进卵巢癌细胞的迁移与侵袭。提示，靶向抑制脂肪细胞 PLIN2 或可作为治疗高级别浆液性卵巢癌（HGSOC）网膜转移的潜在有效策略。

PO_262

紫草素通过抑制 CXCL10 的表达来抑制卵巢癌的生长

王珂 杨君*
新乡医学院第一附属医院

研究目的 探讨紫草素通过抑制 CXCL10 的表达来抑制卵巢癌的生长，为紫草素的临床应用提供理论依据。
材料与方法 对 Skov3 细胞进行紫草素不同浓度梯度的处理，采用 MTT 法检测细胞增殖情况，计算在紫草素作用下细胞的半数抑制浓度（IC50），以确定后续干预浓度，采用细胞划痕实验确定紫草素对卵巢癌 Skov3 细胞体外迁移的抑制情况。对 Skov3 细胞进行紫草素处理和未处理的 RNA 测序(RNA-seq)。京都基因(GO)与基因组百科全书(KEGG)通路分析。IL-17 信号通路有 9 个相关基因表达差异，并且 CXCL10 是显著下调的基因，用 qPCR 对调控基因进行验证。通过临床数据库进行 CXCL10 与卵巢癌生存率的预后分析。转染 CXCL10 过表达慢病毒以及 NC 空载体慢病毒,通过逆转录实时定量聚合酶链反应（qPCR）、荧光显微镜、流式细胞仪检测 CXCL10 的表达水平。使用 MTT 实验检测过表达 CXCL10 后紫草素对 Skov3 细胞的增值影响，通过流式细胞术检测其对细胞凋亡的影响；蛋白质印迹法检测 Caspase3、Bax、Bcl-2 凋亡蛋白表达。紫草素通过 CXCL10 对卵巢癌 Skov3 细胞 IL-17 信号通路相关基因表达的影响。
结果 紫草素治疗可以抑制卵巢癌 Skov3 细胞的增殖及迁移，且随着时间和药物浓度增加，均能增加药物对卵巢癌 Skov3 细胞的抑制增殖能力以及迁移能力。与未进行紫草素处理的 Skov3 相比，在 RNA-seq 之后，成功鉴定了 218 个上调基因和 129 个下调基因。进行 KEGG 通路分析，测序结果显示 IL-17 信号通路有 9 个相关基因表达差异。又经过仔细分析，CXCL10 基因，作为高显著下调的基因。CXCL10 在卵巢癌组织中的表达比正常卵巢组织中升高且与无进展生存期（PFS）呈负相关，提示 CXCL10 的促瘤作用，细胞过表达 CXCL10 后发现，紫草素通过下调 CXCL10 的表达来抑制卵巢癌的生长。此外，紫草素通过抑制卵巢癌的生长可能与 IL-17 信号通路相关。
结论 紫草素可以抑制卵巢癌 Skov3 细胞的增殖及迁移，通过 RNA-seq 之后鉴定了 CXCL10 是紫草素治疗后显著下调的基因，紫草素通过抑制 CXCL10 的表达来抑制卵巢癌的生长。为卵巢癌的临床管理及治疗提供了新的候选基因。

PO_263

单细胞核转录组学揭示高级别浆液性卵巢癌早期复发的细胞免疫逃逸通路

周琛斐*
广东省人民医院

背景 高级别浆液性卵巢癌（HGSOC）是最致命的妇科恶性肿瘤，即使在接受标准治疗后，仍具有高度复发的特点。一些患者可在治疗后维持五年以上无病生存（DFS），而另一些患者则在一年内复发，提示有必要深入了解影响 DFS 差异的细胞和分子机制。肿瘤微环境（TME）中的免疫功能障碍已被视为早期复发的关键驱动因素，但其潜在的细胞互作网络仍不清晰。
方法 本研究对 10 例未经治疗的晚期 HGSOC 患者的原发肿瘤样本进行单核 RNA 测序（snRNA-seq），其中 6 例为长期无病生存（LTDFS），4 例为短期无病生存（STDFS），共分析了 120,495 个细胞核。通过经典标志物注释主要细胞类型，并采用无监督聚类识别 T 细胞、巨噬细胞和上皮细胞的转录亚型。通过拟时序分析、基因集富集和配体–受体（L–R）互作建模推断其功能状态。研究结果进一步在 TCGA-OV 队列的 bulk RNA-seq 数据以及一组独立患者（n = 41）中的多重免疫荧光（mIF）数据中进行了验证。
结果 与 LTDFS 肿瘤相比，STDFS 肿瘤中显著富集了前体耗竭型 $CD8^+$ T 细胞（$CD8^+$ Tpex）、M2 样 $SPP1^+$ 巨噬细胞（Macro_SPP1）以及具有免疫逃逸特征的 $CD47^+$ 上皮细胞（Epi_CD47）。$CD8^+$ Tpex 细胞位于耗竭轨迹的起始阶段，表现出干性样的转录特征；Macro_SPP1 细胞则呈现强烈的 M2 极化状态，并通过 SPP1–ITGA4 及其他整合素轴优先与 $CD8^+$ Tpex 细胞发生互作；Epi_CD47 细胞显示出较高的免疫逃逸评分，表达能招募巨噬细胞的 semaphorin 和整合素配体，并激活 CD47–SIRPA 免疫检查点通路。空间分析进一步证实，这三种细胞在 STDFS 肿瘤中具有共定位特征。在 TCGA 和 mIF 两个验证队列中，以上三类细胞的高表达特征均与早期复发和较差的 DFS 相关。
结论 本研究揭示了 $CD8^+$ Tpex、Macro_SPP1 和 Epi_CD47 细胞之间协调作用的免疫调控轴，该轴构成了 HGSOC 早期复发患者的促肿瘤免疫微环境。这些发现不仅为理解免疫逃逸与肿瘤复发提供了机制基础，也提出了空间共定位细胞群作为潜在预后生物标志物和免疫治疗靶点的新思路。

PO_264

类器官联合基因组分析用于卵巢癌一线化疗与维持治疗反应评估的研究

王海霞 汪玲 朱雪萍 何密斯 钟林 吕玉宇 李巧玲 将沁悦 张聪 刘星河 邹冬玲*
重庆大学附属肿瘤医院

背景 肿瘤类器官作为体外模拟肿瘤生物学特性的三维模型，在个体化治疗中的应用前景广泛。对于高度异质性的上皮性卵巢癌（EOC），类器官为药物反应预测提供了潜在工具。然而，其在聚 ADP 核糖聚合酶抑制剂（PARPi）疗效预测中的临床验证仍有限，尚处于早期研究阶段。
方法 本研究采集接受手术或穿刺活检的初治 EOC 患者的原发肿瘤组织，构建患者来源的 EOC 类器官（EOC_Os）。通过 HE 染色、免疫组化及全外显子测序对类器官进行形态学与分子特征鉴定，并对其进行卡铂、紫杉醇及 PARPi（奥拉帕利、尼拉帕利）等药物的敏感性测试（DST）。同时，利用肿瘤测序评估患者的同源重组缺陷（HRD）状态，并将类器官的药物反应与患者一线化疗及 PARPi 维持治疗后的临床疗效进行前瞻性比较。
结果 成功从 30 例患者中建立 21 株类器官（成功率 70%），涵盖高级别浆液性（hgsEOC）、低级别浆液性（lgsEOC）、子宫内膜样（endEOC）及粘液性（mucEOC）等多个亚型。所建立的类器官在

组织学结构及基因组特征上高度还原其来源肿瘤。不同亚型类器官展现出差异化的化疗药物敏感性，反映 EOC 内部固有的异质性。在 PARPi 测试中，类器官反应与基因组 HRD 状态存在不一致：4 例 HRD 阳性中有 1 例类器官对 PARPi 耐药，临床亦无应答；而在 7 例 HRR 功能正常的患者中，有 2 例类器官对 PARPi 敏感，且这两名患者后续治疗中获得明显临床获益。

结论 EOC 类器官为评估患者药物反应提供了有效模型。将类器官药物敏感性测试与基因组信息相结合，有助于提高 PARPi 疗效预测的准确性，为 EOC 患者提供更加精准的个体化治疗策略。

PO_265

基于遗传学整合分析发现卵巢癌潜在靶点 DPEP2 及其机制验证

朱雪萍 王海霞 何密斯 汪玲 郑倩 隆玲 邹冬玲*
重庆大学附属肿瘤医院

研究目的 卵巢癌（OC）是一种致死率极高的妇科恶性肿瘤，当前临床治疗手段有限。近年来，整合遗传学策略在新靶点发现中展现出重要价值。然而，尚缺乏将全基因组关联研究（GWAS）与数量性状基因座（QTL）数据联合应用于卵巢癌靶点筛选的系统研究。本研究旨在通过整合外周血表达数量性状基因座（eQTL）数据与卵巢癌 GWAS 数据，识别潜在的可成药治疗靶点。

材料与方法 卵巢癌的 GWAS 汇总统计数据来源于卵巢癌协会联盟（OCAC），候选基因的 cis-eQTL 数据取自 eQTLGen 联盟。采用孟德尔随机化（Mendelian Randomization, MR）和共定位分析对潜在靶基因进行优先排序，并结合 DrugBank 数据库筛选对应药物分子。进一步利用 Drug Signatures Database、PubChem、AlphaFold 及 CB-Dock2（基于 AutoDock Vina）评估蛋白–药物互作。对候选基因进行蛋白表达验证、细胞功能实验、药物敏感性测试及通路分析。

结果 共筛选出 5 个具有治疗潜力的靶基因：DPEP2、GABRR2、OPRL1、NTNG1 和 FFAR4，其中 DPEP2 在卵巢癌细胞中高表达并具有明显共定位特征。公开数据库分析显示，DPEP2 表达与胰岛素水平、卵巢囊肿、子宫成纤维细胞显著相关，并与 M2 型巨噬细胞浸润呈正相关。分子对接结果表明，DPEP2 可通过 Thr-178、Asp-177、Val-86、Leu-126、Arg-123、Val-127、Gly-128、Glu-165、Lys-180 等位点与临床药物克拉屈滨（Cladribine）结合。在 A2780 和 SKOV3 细胞系中，克拉屈滨处理可显著下调 DPEP2 表达并抑制细胞增殖。RNA 测序结果提示，克拉屈滨处理后细胞内铁死亡通路显著激活。

结果 本研究鉴定出 DPEP2 为卵巢癌的新型潜在治疗靶点，其结构具有良好的成药性，并可被已有临床药物靶向抑制，为卵巢癌的靶向治疗提供了新的方向和策略。

PO_266

An Episomal SauriCas9 Platform Yields an Immunocompetent Pten/Trp53-deleted Murine Ovarian Cancer Model for Mechanistic Studies and Drug Discovery

Wutao Chen Pengju He Bin Yu You Wang Wen Di*
Renji Hospital, School of Medicine, Shanghai Jiao Tong University, Shanghai

Aim Ovarian cancer (OC) lacks pre-clinical models that (i) harbor clinically relevant driver mutations, (ii) retain genomic stability, and (iii) permit studies in an intact immune microenvironment. PTEN loss and TP53 mutation co-occur frequently in patient tumors, yet existing ID8-derived or genetically

engineered mouse models (GEMMs) incompletely replicate this genotype or require protracted breeding. We therefore sought to:

1. Create a rapid genome-editing system that supports stable dual-locus knockout in primary murine cells;
2. Generate and validate a Pten/Trp53 double-deleted murine ovarian surface epithelial line (MEPP);
3. Validate MEPP's in vivo biological tumorigenicity and compare with human ovarian cancer;
4. Demonstrate MEPP's utility for high-throughput therapeutic screening.

Materials & Methods

l Gene-editing platform: An episomal OriP/EBNA1 backbone was combined with the SauriCas9 and a dual-U6 sgRNA cassette, yielding the EPI-SauriCas9 plasmid. Three sgRNA pairs targeting Trp53 Pten were benchmarked in Hepa1-6 cells; the highest-efficiency pair was selected.

l Murine model generation: Primary C57BL/6 murine ovarian surface epithelium (MOSE) cells were transfected with EPI-SauriCas9, puromycin-selected for 7 days and expanded. Biallelic indels were confirmed by sanger sequencing and deep amplicon NGS. Deletion of the targeted genes was validated using qRT-PCR and gene set enrichment analysis (GSEA) following RNA sequencing. The resulting line was designated MEPP.

l In-vivo assessment: Subcutaneous (s.c.) (3 x 106 cells) and orthotopic intrabursally (i.b.) (1 x 106 cells) injections were performed in immunocompetent female C57BL/6 mice. Tumor growth, ascites, peritoneal dissemination, and omental tropism were recorded.

l Multi-omics characterization: Bulk RNA-seq compared MEPP with ID8-WT; single-cell RNA-seq profiled paired primary and omental lesions.

l Drug screening: A 1 983-compound library (328 epigenetic modulators, 1 655 natural products) was screened at 5 μM in MEPP and a parallel Trp53/Pten-null prostate line (PREPP). Top hits were validated in vitro (dose–response) and in vivo (s.c. MEPP xenografts).

l Human corroboration: PTEN-deleted ovarian cancer organoids (PO1) were used to confirm genomic and pharmacologic concordance.

Results

l MEPP formed s.c. tumors in 100 % of mice (latency ≈ 21 d) and produced hemorrhagic ascites with omental seeding by week 6 after orthotopic injection of 1 x 106 cells. Histology showed serous-like architecture with strong Wt1 staining.

l scRNA-seq insights: Four epithelial sub-clusters were identified. A metastasis-enriched epi1 population up-regulated EMT, TGF-β and IL-6/JAK-STAT signatures, mirroring human metastatic OC datasets. TME analysis revealed elevated TNK infiltration and re-programmed macrophage phenotypes at the omentum. Paired metastatic and primary tissues were examined and found elevated N-Cadherin, estrogen receptor α and phosphorylated-Erk in both MEPP and human ovarian cancers.

l Versus ID8-WT, MEPP up-regulated MAPK, PI3K/AKT-mTOR, glycolysis and oxidative-stress pathways consistent with Pten/Trp53 loss. Transcriptomic overlap was found between MEPP clusters and patient derived ovarian cancer cells.

l Drug discovery: Among 74 MEPP-selective hits, two compounds passed all validation tiers: romidepsin and thioguanine. Both agents were proven effective in MEPP xenografts and PTEN-deleted PO1 organoid.

Conclusion We introduce EPI-SauriCas9, a compact, non-integrative dual-sgRNA system that enables rapid, high-efficiency editing in primary murine cells. Using this platform, we created MEPP, the first Pten/Trp53-deleted immunocompetent ovarian cancer model that (i) recapitulates human metastatic tropism, (ii) mirrors patient transcriptomes at single-cell resolution, and (iii) supports in-vivo drug discovery. High-throughput screening pinpointed romidepsin and thioguanine as potent agents against PTEN-deficient OC, validating MEPP's translational relevance and demonstrating that episomal CRISPR editing can streamline the generation of personalized tumor models for mechanistic and therapeutic research.

PO_267

小红参抗肿瘤活性肽 RA-V 抗卵巢癌的作用及机制研究

杨光溟[1] 王鑫[1] 刘臻妍[1] 赵桃宇[2] 杨祥群[1] 贾岳[1] 杨宏英[1*]
1.北京大学肿瘤医院云南医院昆明医科大学第三附属医院 2.德宏州人民医院

目的 卵巢癌是女性生殖系统中致死率最高的恶性肿瘤之一，需新策略和靶向药物改善预后。环肽 RA-V 是一种茜草属植物小红参（Rubia yunnanensis (Franch.) Diels)的根茎部位分离得到的主要环肽成分，可通过诱导细胞凋亡和抑制增殖表现出显著的抗癌活性。本研究旨在深入探讨环肽 RA-V 抗卵巢癌的作用及机制，为卵巢癌治疗提供潜在的新思路。

方法 1、应用 CCK-8 实验、EdU 实验、3D 肿瘤球实验和克隆形成实验评价 RA-V 对卵巢癌细胞增殖影响。应用 Transwell 实验及 3D 肿瘤球侵袭实验评估 RA-V 对卵巢癌细胞转移能力的影响。

2、通过构建 Hey 裸鼠卵巢癌异种移植瘤模型评估 RA-V 对卵巢癌的治疗效果。通过 H&E 染色、血清学评估 RA-V 对裸鼠的肝肾组织毒性作用。

结果 1、体外 CCK-8 实验和 EdU 实验结果表明，RA-V 对卵巢癌细胞的抑制效果具有时间-浓度依赖性；3D 肿瘤球实验结果表明，RA-V 对卵巢癌肿瘤球体生长的抑制作用具有时间-浓度依赖性；克隆形成实验结果显示，与对照组相比，不同浓度的 RA-V 处理组的克隆形成数均显著减少；流式细胞术检测提示 RA-V 可显著抑制卵巢癌细胞周期进程；Transwell 实验提示 RA-V 可显著抑制卵巢癌细胞转移能力；3D 肿瘤球侵袭实验提示 RA-V 能显著抑制卵巢癌细胞侵袭能力。

2、RA-V 分别处理卵巢癌 Hey 细胞和 SKOV3 细胞，蛋白组学筛选差异表达基因，发现相对于溶剂对照组，经 RA-V 处理后，SKOV3 细胞与 HEY 细胞的差异表达蛋白谱存在显著交集，共筛选获得 266 个共同差异表达基因。在这些交集基因中，有 35 个基因呈现上调表达，而 204 个基因表现出下调表达。KEGG 和 Reactome 通路富集分析表明，RA-V 可能通过影响核糖体、P53 通路、细胞周期、DNA 复制等关键途径发挥作用。

3、裸鼠尾静脉注射 RA-V 可显著抑制卵巢癌皮下移植瘤的生长，其疗效与顺铂及尼拉帕利相当。裸鼠重要脏器的 HE 染色及血清学检查心、肝、肾相关损伤标志物，提示 RA-V 对裸鼠的主要脏器无明显毒性作用。

结论 1、RA-V 能抑制卵巢癌细胞的增殖、转移能力；

2、尾静脉注射 RA-V 治疗在体内实验中展现出与顺铂、尼拉帕利相似的抗肿瘤活性，且无显著毒副作用。

PO_268

基于单细胞测序的卵巢透明细胞癌的治疗方案的研究

张冰 宁少楷
无锡市妇幼保健院

目的 卵巢透明细胞癌（Ovarian Clear Cell Carcinoma, OCCC）是卵巢上皮性癌中一种独特的组织学亚型，占所有卵巢癌的 5%-25%，亚洲人群（日本、韩国）发病率显著高于欧美（15%-25% vs. 1%-12%）。OCCC 具有独特的临床病理特征和分子生物学改变，如高频 ARID1A 和 PIK3CA 突变，并与子宫内膜异位症密切相关。早期 OCCC 预后较好，但晚期或复发患者对传统铂类化疗反应较差，生存率低。高级别浆液性卵巢癌（High-grade serous ovarian carcinoma, HGSOC）是最常见的卵巢上皮性癌亚型，占卵巢癌病例的 70%-80%。

基于前期的临床研究以及文献分析，本研究发现 OCCC 早期患者的无进展存活期和五年生存率与 HGSOC 早期患者相当，但是 OCCC 晚期患者的无进展存活期和五年生存率与 HGSOC 晚期患者相比明显偏低，且具有显著性差异。考虑到 OCCC 临床上大都是参考 HGSOC 的治疗方案，并没有单独的治疗方案，因此本研究想探究针对 OCCC 晚期患者的新型治疗方案。

方法 本研究分别收集了 OCCC 和 HGSOC 患者手术后的临床样本，运用单细胞转录组测序的方法对样本进行测序。基于单细胞转录组测序数据运用各种生物信息学方法进行数据分析，包括细胞类型的区分、细胞亚群的分析、差异通路的分析等等。基于分析结果，运用细胞存活实验、免疫共沉淀、免疫荧光、多重免疫组化、动物造模等实验手段在细胞、临床样本、动物组织中验证单细胞转录组测序分析的结果并测试药物 A 在动物模型中的效果，并进一步探究其机制与开发新的治疗方案。

结果 经过对单细胞转录组测序数据的分析，本研究发现，和 HGSOC 晚期临床样本相比，OCCC 晚期临床样本中成纤维细胞的含量明显偏高。本研究运用多重免疫组化技术在整合了上百个临床样本的组织芯片中检测成纤维细胞 marker 基因的表达情况，包括表达量和表达定位。结果显示，在 OCCC 晚期临床样本中成纤维细胞 marker 基因的表达量和表达范围明显升高，具有显著性差异。在 OCCC 细胞系以及 OCCC 晚期临床样本中运用免疫共沉淀、免疫荧光在蛋白水平验证了成纤维细胞 marker 基因的高表达。在动物模型实验中，本研究通过皮下注射成功构建 OCCC 动物模型，在此基础上药物 A 的处理会明显减少成纤维细胞 marker 基因的表达。

结论 基于对临床样本进行单细胞转录组测序，本研究分析并在细胞、动物、临床样本中验证了成纤维细胞 marker 基因在 OCCC 晚期临床样本中的异常升高。在 A 药物的干预下，成纤维细胞 marker 基因的信号明显降低，为开发新的临床治疗方案提供了线索，具有重要的临床应用价值。

PO_269

The Impact of Location and Type of BRCA Mutation on PARP Inhibitor Response and Prognosis in Epithelial Ovarian Cancer: A Real-World Multicenter Study

Yiming Fan[1] Qiyu Liu[1] Cuiyu Huang[1] Manqi Wu[1] Yikun Jin[1] Liyuan Tao[1] Yan Liu[1] Jiandong Song[2] Shijun Wang[3] Weimin Kong[4] Xianghua Huang[5] Chunfang Ha[6] Xielan Yang[7] Jie Qiao[1] Yan Zhang[8] Yuan Li[1] Hongyan Guo[1*]

1.Peking University Third Hospital2.Affiliated Hospital of Inner Mongolia Medical University
3.Xuanwu Hospital, Capital Medical University
4.Beijing Obstetrics and Gynecology Hospital, Capital Medical University,
Beijing Maternal and Child Health Care Hospital
5.The Second Hospital of Hebei Medical University6.General Hospital of Ningxia Medical University
7.Peking University Cancer Hospital Yunnan, Yunnan Cancer Hospital, The Third Affiliated Hospital of Kunming Medical University8.Peking University First Hospital

Objective Although poly (ADP-ribose) polymerase inhibitors (PARPi) have transformed the treatment landscape for BRCA1/2-mutated ovarian cancer, a different degree of benefit seems to exist. This study aimed to investigate the association between location and type of BRCA1/2 mutations and PARPi response in patients with epithelial ovarian cancer. Previous studies indicated that patients harboring mutations located outside the functional domains had better responses to PARPi. In this study, we further refined this subgroup and demonstrated that mutations spanning from the RING domain to the DNA-binding domain (DBD) were associated with worse PARPi responses, whereas those located between the DBD and BRCT domain showed favorable responses. Based on these findings, a new classification covering full-length of BRCA1 gene was established.

Materials and Methods This was a multicenter, real-world, retrospective study aiming to investigate the impact of different site and type of BRCA1/2 mutations on the efficacy of PARPi treatment and prognosis in patients with ovarian cancer. Between 2015 and 2024, patients newly diagnosed with epithelial ovarian cancer were enrolled. Inclusion criteria comprised confirmed pathogenic or likely pathogenic germline or somatic BRCA1/2 mutations. Demographic, clinical, therapeutic, and outcome data were collected from institutional medical records. BRCA1 functional domains included RING domain, DNA-binding domain, and BRCT domain. For BRCA2, functional domains included the RAD51-binding domain (RAD51-BD), and the DNA-binding domain (DBD). Progression-free survival (PFS) was analyzed using Kaplan-Meier methods with log-rank testing for between-group comparisons.

Cox proportional hazards models estimated hazard ratios with 95% confidence intervals. Propensity score matching was employed to reduce confounding.
Results A total of 332 patients were included, of whom 218 (65.7%) received PARPi as maintenance therapy. Among BRCA1 mutation carriers, those with mutations in the DNA-binding domain and adjacent region had poor prognosis without treatment but showed substantial benefit with PARPi ($p = 0.021$ and $p = 0.001$, respectively). Conversely, mutations in the RING domain and its downstream region showed limited therapeutic benefit and were associated with poorer overall outcomes. Patients with mutations located in the BRCT domain exhibited favorable prognosis regardless of treatment, diminishing the relative benefit of PARPi in this subgroup. According to the mutation type, patients with frameshift or nonsense mutations experienced significant improvement with PARPi (all $p < 0.05$). However, those with splicing or missense mutations did not derive similar benefit. To address the heterogeneous distribution of mutation types across domains, a combined mutation-domain analysis was conducted. While no overall benefit was observed in the RING+ group, the subset of patients harboring FS or NS mutations exhibited significantly improved outcomes following PARPi treatment ($p = 0.044$).
Conclusion The efficacy of PARPi and the prognosis of epithelial ovarian cancer patients varied according to both location and type of BRCA mutation. Comprehensive mutation profiling should guide treatment decisions to optimize outcomes in patients with BRCA-mutated ovarian cancer.

PO_270

负载 circPUM1 siRNA 的 pH 响应型二维碳化铌纳米片用于卵巢癌细胞杀伤和抗血管生成治疗

关雪
辽宁省肿瘤医院

目的 卵巢癌的进展与肿瘤微环境失调密切相关，特别是由外泌体介导的细胞间“串话”驱动病理性血管生成。我们发现卵巢癌细胞来源的环状 RNA circPUM1 可通过外泌体转移至血管内皮细胞，促进血管生成。机制上，circPUM1 通过竞争性结合 miR-607，上调 RAB27B 和 VEGFA 的蛋白表达，从而促进 RAB27B 介导的外泌体释放，并直接激活血管内皮细胞 VEGF 信号通路，协同促进血管生成。
方法 基于 circPUM1 在卵巢癌肿瘤微环境的作用，我们设计了一种新型的 pH 响应型二维碳化铌（Nb_2C）纳米片，用于负载 circPUM1 siRNA 以破坏其驱动的肿瘤微环境失调。通过聚乙烯亚胺（PEI）修饰得到阳离子化二维碳化铌片（P-Nb_2C），实现高效负载带负电的 siRNA；进一步利用聚乙二醇（PEG）表面修饰得到 P-Nb_2C-PEG，屏蔽纳米片正电荷以减少脱靶效应和全身毒副作用。利用肿瘤酸性微环境触发 PEG 层脱落，实现肿瘤组织特异性 siRNA 释放。通过体外细胞功能实验及小鼠腹腔转移瘤模型，系统评估 P-Nb_2C-PEG@si-circPUM1 的抗肿瘤及抗血管生成疗效。
结果 细胞功能实验显示，该纳米片处理的卵巢癌细胞增殖、迁移及侵袭能力显著受抑，VEGFA 与 RAB27B 表达同步下调。进一步将纳米片处理后的癌细胞分泌的外泌体与血管内皮细胞共培养，发现内皮细胞的增殖、迁移及成管能力明显受损。在小鼠腹腔成瘤模型中，纳米片递送的 siRNA 可有效敲低 circPUM1 表达，阻断其下游通路，显著抑制肿瘤血管生成和腹腔播散，验证了这一策略的治疗潜力。
结论 本研究揭示了一种全新的外泌体介导的卵巢癌血管生成分子机制，并开发出一种基于超薄碳化铌 MXene 的 pH 响应型 siRNA 纳米递送系统。该材料的优势在于：原料成本低，可批量制备；比表面积大，更高的基因载量；pH 响应特性，降低了对机体正常细胞的损伤和毒副作用，为卵巢癌的精准治疗提供了一种前景广阔的策略，具有重要的临床转化价值和应用潜力。

PO_271

基于单中心回顾性数据的早期成人型卵巢颗粒细胞瘤患者的临床特征及预后分析

茹意
郑州大学第一附属医院

研究目的 卵巢颗粒细胞肿瘤是卵巢肿瘤中一种相对罕见的低度恶性的单纯性索细胞肿瘤，目前国内外关于成人型卵巢颗粒细胞肿瘤（Adult Granulosa Cell Tumor, AGCT）的研究较少，且几乎均为小样本回顾性研究，价值有限。本研究旨在通过回顾性研究分析早期 AGCT 患者的临床特征、治疗方案及其预后结局，为临床医师提高疾病诊断水平，优化治疗策略提供依据。

材料与方法 本研究纳入了郑州大学第一附属医院 2012 年至 2024 年间接受手术的早期 AGCT 患者共 244 例，成功随访患者 207 例。收集患者的人口学信息、临床特征、治疗方案、术后状态等情况，并通过电话随访患者的生存状态及保留生育功能患者的生育结局。

使用 SPSS 27.0 软件统计分析。分类变量以频数和百分比表示，正态分布资料用均值±标准差描述，偏态资料用中位数（P25, P75）表示。无病生存期（DFS）为从手术当日到复发或删失的时间。采用 Kaplan-Meier 法计算患者的无病生存率。

结果 临床病理特征：患者平均初发年龄为 47 岁，高血压是最常见的合并症，约占 15.6%。绝大多数患者为Ⅰ期肿瘤（98.0%），以 IA 期（48.4%）及 IC 期（共 35.2%）最常见。肿瘤中位直径为 75mm，89.6%患者检测到 FOXL2 基因 C134W 突变。

治疗方案：65.2%患者接受了全面分期手术，17.6%行保留生育功能的手术。手术的平均时间为 170min，术中出血中位数为 30ml（20，50），术中输血率为 20.5%。接受手术治疗后，选择联合化疗者占 41.4%，常见化疗方案包括紫杉醇类联合铂类及 BEP 方案。

手术结局：绝大多数患者术后恢复良好，胃肠道恢复中位时间为 2 天，中位术后住院时间为 6 天。术后并发症发生率较低，最常见为淋巴系统并发症（5.7%）和发热（4.5%）。术后 Clavien-Dindo 并发症分级以Ⅰ–Ⅱ级为主。

肿瘤学结局：患者的中位随访时间为 44 个月（17, 83），共 12 例患者出现复发（5.8%），其中 3 人因该病复发而死亡。总体死亡 5 例（2.4%），无病生存者比例达 97.1%。

生育学结局：本研究成功随访了 37 例接受保留生育功能的手术治疗的患者，其中 16 例患者于初次治疗后尝试妊娠，共 15 例患者成功妊娠，累计成功妊娠 20 次。在 20 次妊娠中，18 次妊娠通过自然受孕实现，2 次通过辅助生殖技术受孕，共成功分娩 14 次，其中足月分娩 11 次，早产 3 次，还有 1 例患者目前仍处于妊娠状态，未分娩。

结论 早期成人型卵巢颗粒细胞肿瘤总体预后良好，尤其在接受全面分期手术者中，复发率和死亡率均较低。多数患者术后恢复迅速，术后并发症发生率较低。对于有生育需求的年轻患者，行保留生育功能的手术在确保肿瘤切除的前提下也可获得良好结局，且未显著增加复发风险。研究结果支持在严格筛选前提下开展保留生育力的个体化治疗方案，并强调术后长期随访对复发监测的重要性。

PO_272

不同剂量贝伐珠单抗一线维持治疗对晚期高级别浆液性卵巢癌的预后影响：一项基于真实世界的倾向性评分匹配研究

吴涛 张鹏闯 申亚辉 胡丽娟 王小伟 周敏 席儒兴 王国庆*
陕西省肿瘤医院

背景及目的 不同剂量贝伐珠单抗 7.5 mg/kg（低剂量） 与 15 mg/kg（高剂量） 用于晚期高级别浆液性卵巢癌一线维持治疗的疗效差异尚不明确。本研究评估不同剂量对患者预后的影响。
方法 回顾性分析 2018 年 1 月至 2024 年 3 月陕西省肿瘤医院收治的 538 例 III-IV 期高级别浆液性卵巢癌患者资料，纳入接受贝伐珠单抗一线维持治疗的 189 例患者。采用 1:1 最近邻倾向性评分匹配法平衡组间基线特征，通过卡方检验比较匹配后两组临床特征及≥2 级治疗相关不良反应发生率。运用 Kaplan-Meier 法绘制生存曲线，Log-rank 检验比较生存差异，Cox 比例风险模型进行多因素预后分析。
结果 经倾向性评分匹配后，成功获得 33 对配对样本（高剂量组 vs. 低剂量组，n=66）。匹配队列中：无进展生存期（PFS）：低剂量组中位 PFS 为 13 个月（95% CI：10.0~16.0），高剂量组为 19 个月（95% CI：16.0~22.0）；多因素分析显示，低剂量组疾病 PFS 显著短于高剂量组（HR=1.64，95% CI：1.07~2.50，P=0.022）。总生存期（OS）：低剂量组中位 OS 为 41 个月（95% CI：37.0~45.0），高剂量组为 42 个月（95% CI：38.0~46.0）；两组 OS 差异无统计学意义（HR=1.24，95% CI：0.72~2.15，P=0.436）。安全性：在未匹配全队列（n=189）中，高剂量组≥2 级高血压发生率显著高于低剂量组（21.6% vs. 13.2%，P=0.011），两组均未发生消化道穿孔等严重毒性事件。
结论 在晚期高级别浆液性卵巢癌一线维持治疗中，贝伐珠单抗 15 mg/kg 方案较 7.5 mg/kg 方案可显著延长无进展生存期，但未带来总生存期获益。高剂量方案需关注高血压等治疗相关不良反应的管理。本研究为临床个体化剂量选择提供了真实世界证据。

PO_273

piR-26681 Suppresses Ovarian Cancer Progression by Enhancing METTL3/METTL14-Mediated m6A Modification of FBXO16 and Impairing DNA Repair via MORF4L1 Degradation

Shuo Chen*
The Third Affiliated Hospital of Guangzhou Medical University

Purpose Survival rates for ovarian cancer decline sharply with advanced stages, and most cases are diagnosed at late stages, leading to a poor prognosis. Poly (ADP-ribose) polymerase (PARP) inhibitors are important therapies for ovarian cancer, especially in patients with homologous recombination deficiency (HRD). However, their efficacy is much lower in homologous recombination (HR)-proficient tumors that retain DNA repair capacity. There is an urgent need to identify novel molecular targets that can inhibit tumor progression and improve therapeutic outcomes, especially for patients with homologous HR-proficient tumors. This study aimed to identify nucleic acid molecules that are downregulated in advanced ovarian cancer, elucidate their functional roles, and explore their potential as therapeutic targets. We focused on PIWI-interacting RNA piR-26681, which was significantly downregulated in advanced-stage ovarian cancer tissues and correlated with improved prognosis.
Materials and Methods Differential expression profiling of piRNAs was conducted using Pandora sequencing in paired early- and late-stage human ovarian cancer samples. Validation was performed with a larger cohort, including 43 early-stage, 42 advanced-stage ovarian cancer patients, and 19 normal ovarian tissue samples. Functional assays, including CCK-8, plate colony formation, EdU

incorporation, wound healing, transwell invasion, and apoptosis assays, were conducted in ovarian cancer cell lines (CAOV3 and OVCAR3) following overexpression or silencing of piR-26681. Molecular mechanisms were investigated using pulldown, RNA immunoprecipitation (RIP), immunofluorescence, co-immunoprecipitation (Co-IP), western blotting, and cycloheximide/proteasome inhibition assays. MeRIP-seq, qPCR, and mRNA stability assays were employed to assess m6A methylation and mRNA stability. Downstream targets were identified via bioinformatics and experimentally validated by RIP and Co-IP. The tumor-suppressive effect of piR-26681 was further examined in xenograft models and patient-derived ovarian cancer organoids (OC-PDOs) treated with Ago-piR-26681.

Results piR-26681 expression was significantly reduced in advanced-stage ovarian cancer tissues compared to early-stage and normal ovarian tissues. Low piR-26681 levels correlated with higher disease stage and poorer prognosis, but not with age. Functional studies revealed that overexpression of piR-26681 inhibited ovarian cancer cell proliferation, migration, and invasion, while promoting apoptosis. Mechanistically, piR-26681 was shown to interact with and stabilize the METTL3/METTL14 methyltransferase complex, enhancing their interaction and increasing global m6A methylation. Overexpression of piR-26681 led to elevated m6A modification and stability of METTL14 and FBXO16 mRNAs, mediated by IGF2BP2 binding. FBXO16, upregulated by piR-26681, facilitated the ubiquitin-mediated degradation of MORF4L1, a key regulator of homologous recombination repair. This resulted in impaired DNA repair capacity and increased DNA damage in ovarian cancer cells, as evidenced by comet assays and γ-H2AX immunofluorescence. Rescue experiments confirmed that the tumor-suppressive effects of piR-26681 were dependent on FBXO16, IGF2BP2, and the METTL3/METTL14 complex. In vivo, administration of Ago-piR-26681 in xenograft models significantly inhibited tumor growth, increased METTL3, METTL14, and FBXO16 expression, and had no adverse effects on animal weight. Similar anti-tumor effects were observed in patient-derived organoids, highlighting the translational potential of piR-26681.

Conclusion This study reveals a novel regulatory axis, piR-26681–METTL3/METTL14–FBXO16–MORF4L1, which suppresses ovarian cancer progression by disrupting homologous recombination repair through MORF4L1 degradation., that suppresses ovarian cancer progression by impairing homologous recombination repair through the degradation of MORF4L1. piR-26681 stabilizes the METTL3/METTL14 complex and increases m6A modification, thereby enhancing the stability and expression of METTL14 and FBXO16. Upregulation of FBXO16 promotes the ubiquitination and degradation of MORF4L1, leading to increased DNA damage and apoptosis in ovarian cancer cells. These findings demonstrate that piR-26681 functions as a tumor suppressor and represents a promising nucleic acid-based therapeutic target, particularly for sensitizing HR-proficient ovarian cancers to DNA-damaging therapies such as PARP inhibitors. Further, the potential of combining piR-26681 with advanced nano-delivery technologies may overcome current barriers in RNA therapeutics, providing new opportunities for precision treatment of ovarian cancer. Future studies and clinical trials are warranted to validate and advance the therapeutic application of piR-26681 in ovarian cancer.

PO_274

NSDHL 通过调节脂质过氧化和内质网应激进而介导卵巢癌细胞的凋亡

徐姗姗
浙江大学医学院附属妇产科医院

目的 卵巢癌是迄今致死率最高的妇科恶性肿瘤，靶向治疗卵巢癌细胞具有重要的科学意义。近来，众多研究证实胆固醇合成代谢途径在肿瘤发挥重要的生物学功能，NSDHL 是参与胆固醇生物合成的关键酶，已有文献证实可作为一个新型的抗乳腺癌药物治疗靶点，但在卵巢癌研究中尚未看到相关研究。本研究深入探索 NSDHL 在卵巢癌中的作用及相关机制。

方法 通过蛋白质谱测序、生信分析、蛋白组学测序、脂质过氧化、内质网应激等检测以及体外动物实验等手段

结果 研究证实 NSDHL 表达升高与肿瘤进展有关；敲低 NSDHL 诱导卵巢癌细胞脂质过氧化和死亡，同时伴随 ROS 的产生以及胆固醇和氧化还原稳态的破坏。定量蛋白质组学分析表明 NSDHL 缺失显著改变脂质代谢，特别是增加多不饱和脂肪酸合成和氧化应激，同时抑制铁死亡。NSDHL 敲低诱导的细胞凋亡主要由脂质过氧化触发的内质网应激介导，表现为 ER 应激标志物的升高和 ER 应激特异性 caspase 的激活。此外，裸鼠实验证实抑制 NSDHL 可有效抑制肿瘤生长。

结论 抑制 NSDHL 能诱导细胞脂质过氧化和内质网应激，进而促进卵巢癌细胞死亡，为靶向卵巢癌提供新的治疗策略。

PO_275

CAFs 通过 MARCKS 促进卵巢癌铂耐药的机制探究

闫博然 汪辉*
浙江大学附属妇产科医院

研究目的 卵巢癌是妇科恶性肿瘤中致死率最高的疾病，化疗耐药是治疗失败的主要原因。近年研究表明，肿瘤微环境中的肿瘤相关成纤维细胞（CAFs）通过细胞间相互作用和分泌活性物质参与耐药调控，但其具体机制尚不明确。MARCKS（Myristoylated Alanine-Rich C-Kinase Substrate）是一种膜相关蛋白激酶底物，在多种肿瘤中调控细胞迁移和信号转导，但其在 CAFs 介导的卵巢癌耐药中的作用未见报道。本研究旨在揭示 MARCKS+ CAFs 通过旁分泌机制诱导卵巢癌顺铂耐药的关键分子通路，为靶向肿瘤微环境的治疗策略提供新靶点。

材料与方法 首先收集卵巢癌患者组织标本，通过免疫组化、多重免疫荧光染色和 Western blot 检测 MARCKS 表达水平。建立原代 CAFs 与肿瘤细胞共培养系统，采用 Transwell 共培养和条件培养基处理模拟微环境互作；通过细胞因子芯片筛选关键旁分泌因子；利用 siRNA 干扰技术靶向调控特定信号通路；评估 MPS 肽（MARCKS 磷酸化特异性抑制剂）的治疗效果。

研究结果 获得性铂耐药患者的肿瘤微环境中存在明显的 CAFs 浸润特征，MARCKS 在获得性铂耐药患者的 CAFs 中显著高表达。并且 MARCKS 高表达的 CAFs 能够显著增强卵巢癌细胞的铂耐药性。进一步鉴定 CAFs 细胞分泌的细胞因子发现 MARCKS+ CAFs 高分泌 IL-8，激活肿瘤细胞内的 PI3K/AKT 信号通路，进而诱导铂耐药性增强。抑制 MARCKS 或阻断 IL-8 信号可显著提高顺铂的治疗效果。

研究结果 本研究系统揭示了肿瘤微环境中 CAFs 通过旁分泌网络与肿瘤细胞互作促进铂耐药的多层次调控机制，不仅阐明了细胞间信号传递在耐药形成中的核心作用，还鉴定出多个潜在的干预靶点。这些发现为开发靶向肿瘤微环境的精准治疗策略提供了重要的理论支撑，对改善卵巢癌患者的临床预后具有重要价值。

PO_276

用于早期上皮性卵巢癌的肿瘤细胞外囊泡表面蛋白-mRNA 整合检测法

刘怀超 万吉鹏*
山东第一医科大学附属省立医院

目的 上皮性卵巢癌（epithelial ovarian cancer，EOC）的及时发现对于改善临床结局、促进早期干预治疗具有至关重要的意义。然而，当前用于 EOC 监测的手段在灵敏性和特异性方面仍不尽理想。肿瘤

来源的细胞外囊泡（extracellular vesicles，EVs）由于其在脂质双层膜中包裹 mRNA，能够很好地代表原发肿瘤组织，成为非侵入性检测特定 EV 亚群体的理想替代物。
方法 本文介绍了一种简化的 EOC EV 表面蛋白检测方法，该方法能够对 EOC EV 的三个亚群中的两个参考基因 β-肌动蛋白（ACTB）和膜棕榈酰化蛋白 1（MPP 1）进行 RT-qPCR 定量（叶酸受体 α [FRα]+ EOC EV、间皮素[MSLN]+ EOC EV 和滋养层细胞表面抗原 2 [TR 0 P2]+ EOC EV），导致 EOC EV 表面蛋白特征，即在 EOC EV 的三个亚群（FRα+ EOC EV、MSLN+ EOC EV 和 TR 0 P2 + EOC EV）中 ACTB 和 MPP 1 基因的逆转录-定量聚合酶链反应（RT-qPCR）读数。该测定方案涉及两步工作流程，步骤 1：通过点击珠粒进行点击化学介导的 EOC EV 富集；步骤 2：通过用 RT-qPCR 检测两种参考基因来定量 EOC EV。我们制备了三种反式环辛烯（TCO）接枝的 EOC 特异性抗体，即，TCO-抗 FRα、TCO-抗 MSLN 和 TCO-抗 TROP 2，以促进点击微珠在 EOC EV 的三个亚群中的点击化学介导的富集。
结果 在训练队列中，EOC EV 表面蛋白评分体系在检测 EOC 方面表现出优异的诊断性能，其受试者工作特征曲线下面积（area under the receiver operating curve，AUROC）为 0.97（95%置信区间[CI]，0.95–1.00），灵敏度为 93%，特异性为 92%。在验证队列中，EOC EV 表面蛋白评分体系的 AUROC 仍表现良好，为 0.93（95% CI，0.87–0.98）。进一步的分组分析结果显示，该评分体系在不同分期（边界性 EOC 及 I/II 期 EOC，AUROC 0.92；95% CI，0.87–0.97）以及不同组织学类型（高级别浆液性癌，AUROC 0.97；95% CI，0.94–0.99）中同样具有出色的判别能力。
结果 综上所述，EOC EV 表面蛋白评分体系有望成为 EOC 早期筛查的有力工具，从而在临床上为提升患者管理水平提供重要价值。

PO_277

HK2+CAFs 介导巨噬细胞脂质代谢重编程调控卵巢癌迁移侵袭的研究

陈欣 吕卫国 许君芬*
浙江大学医学院附属妇产科医院

研究目的 高级别浆液性卵巢癌（HGSOC）是上皮性卵巢癌最具侵袭性的亚型，其高度异质的肿瘤微环境（TME）是促癌进展的关键因素。HK2 高表达的肿瘤相关成纤维细胞（HK2+_CAFs）与患者不良预后显著相关。HK2+_CAFs 可能通过重塑肿瘤微环境，调控肿瘤相关巨噬细胞（TAMs）的代谢适应和促肿瘤极化，进而增强卵巢癌转移。本研究旨在揭示 HK2+_CAFs 通过代谢重编程调控 TAMs 极化的分子机制，阐明 HK2+_CAFs-TAMs 互作轴在 HGSOC 转移中的作用，为靶向 TME 代谢干预提供新策略。
材料与方法 研究拟通过分离原代 CAFs，利用 siRNA 和质粒转染技术调控 HK2 表达，建立 CAFs-巨噬细胞共培养体系，分析 HK2+_CAFs 对巨噬细胞极化状态和脂代谢（脂滴积累及游离脂肪酸水平）的影响。进一步通过 CCK-8 和 Transwell 实验评估经 HK2+_CAFs 调控后的巨噬细胞对卵巢癌细胞增殖、迁移和侵袭能力的作用。此外，通过 RNA-seq 以及质谱分析 HK2+CAFs 调控巨噬细胞重编程的分子机制。
结果 免疫荧光染色证实 HK2 在患者来源的 CAFs 中高表达，qPCR 显示 CAFs 中 HK2 表达显著高于正常成纤维细胞。CCK-8 和 Transwell 实验表明 HK2 敲低可抑制 CAFs 的增殖、迁移侵袭能力，并降低其葡萄糖摄取和乳酸生成。进一步研究发现，HK2 敲低的 CAFs 与 THP-1 来源巨噬细胞共培养后，可减少巨噬细胞中脂质沉积、游离脂肪酸含量及 ATP 生成，同时降低 NAD+/NADH 比值。将处理后的巨噬细胞上清与卵巢癌细胞系（Caov3、OVCAR8）共培养，结果显示 HK2 敲低的 CAFs 预处理组可显著抑制癌细胞的增殖和迁移侵袭能力。此外，转录组测序分析发现分泌蛋白 CPA4 基因表达与 HK2 水平呈正相关，在 CAFs 敲低 HK2 后，CPA4 的 mRNA 以及蛋白水平也下调。巨噬细胞外源添加 CPA4 重组蛋白后与卵巢癌细胞共培养，增强了卵巢癌细胞系（Caov3、OVCAR8）的增殖和迁移侵袭能力。

结论 本研究证实，HK2 在 CAFs 中高表达并通过调控糖代谢促进其增殖和迁移侵袭能力。HK2 敲低可抑制 CAFs 的糖酵解活性，进而通过下调分泌蛋白 CPA4 影响巨噬细胞代谢重编程最终抑制卵巢癌细胞的恶性进展。外源添加 CPA4 可逆转 HK2 敲低 CAFs 对癌细胞的抑制作用，提示 HK2-CPA4 轴是 CAFs 通过代谢重塑巨噬细胞促进卵巢癌发展的关键机制，为靶向肿瘤微环境提供了新策略。

PO_278

抑郁症通过代谢功能异常和 IRF1 介导的免疫抑制促进卵巢癌进展

郑烨清 程文俊*
江苏省人民医院

研究目的 卵巢癌患者中抑郁症发生率较高，但抑郁症对肿瘤进展的影响及潜在机制尚未明确。本研究旨在探讨抑郁症与卵巢癌进展的关系，解析其背后的分子机制，并分析抗抑郁药物联合抗肿瘤治疗的潜在价值。
材料与方法 临床资料分析：回顾性分析 2024 年 10 月至 2025 年 3 月收治的 84 例卵巢癌患者临床资料，采用 PHQ-9 和 GAD-7 量表评估抑郁和焦虑状态。
动物模型构建：使用免疫健全的 C57BL/6 小鼠，通过慢性约束应激（CRS）和慢性不可预测轻度应激（CUMS）诱导抑郁样行为，结合 ID8 细胞构建卵巢癌模型。
检测技术：采用单细胞 RNA 测序解析肿瘤及微环境转录图谱，非靶向代谢组学分析代谢谱变化，Luminex 检测细胞因子水平，结合行为学实验（旷场试验、悬尾实验等）、活体成像、免疫荧光染色等技术开展研究。
干预实验：对抑郁合并卵巢癌小鼠采用抗抑郁药（艾司西酞普兰）联合铂类药物治疗，观察肿瘤进展及分子机制变化。
结果 临床与动物表型：84 例卵巢癌患者中抑郁发生率为 45.24%；CRS 和 CUMS 模型成功诱导小鼠抑郁样行为，且显著促进卵巢癌生长及膈顶转移，加剧体重下降。
代谢与免疫异常：慢性应激导致小鼠卵巢癌代谢谱改变，以脂质代谢（类固醇激素生物合成、甘油磷脂代谢等）和谷胱甘肽通路紊乱为核心；同时，应激下调肿瘤细胞中 IRF1 表达，抑制 CCL2-CCR2 轴，减少 $CCR2^+$巨噬细胞浸润，形成免疫抑制微环境。
分子机制：IRF1 在应激组中显著下调，其高表达与卵巢癌患者更长的总生存期和无进展生存期相关；IRF1 通过调控 CCL2 分泌影响巨噬细胞浸润，是连接应激与肿瘤微环境的关键分子。
干预效果：艾司西酞普兰联合铂类治疗可上调 IRF1 表达，恢复 $CCR2^+$巨噬细胞浸润，逆转代谢及免疫异常，显著延缓肿瘤进展，效果优于铂类单药。
结论 抑郁症相关慢性应激通过下调 IRF1 表达，诱导脂质代谢紊乱及 CCL2-CCR2 轴抑制，形成免疫抑制性肿瘤微环境，从而促进卵巢癌进展。抗抑郁药物与铂类联合治疗可逆转上述病理变化，提示该联合策略可能成为卵巢癌合并抑郁患者的潜在治疗方案。

PO_279

eccDNA 在高级别浆液性卵巢癌中的作用机制研究

任妍 许君芬*
浙江大学医学院附属妇产科医院

研究目的 探讨染色体外环状 DNA 在晚期高级别浆液性卵巢癌中的作用和功能，有助于建立新的临床诊断策略，为 HGSOC 治疗提供潜在的治疗靶点。

材料与方法 采用 Circle-Seq 和 RNA-Seq 分析法分别检测同一批 HGSOC 患者的正常卵巢、正常输卵管和肿瘤组织中的 eccDNAs 和 mRNAs。用透射电镜和扫描电镜观察相关细胞和组织中的 eccDNAs。桑格测序，外向 PCR 和内向 PCR 验证所筛选的 eccNXN 是目标环状 DNA 分子。通过 qRT-PCR 和 FISH 实验验证 eccNXN 在高级别浆液性卵巢癌中的表达 。Kaplan-Meier 生存分析表明 eccNXN 对患者预后的影响。共聚焦显微镜和 qRT-PCR 确定了 eccNXN 和 其宿主基因 NXN 在空间和数量上的相关性。通过 CCK-8 试验、集落形成试验、细胞凋亡试验、迁移侵袭试验评估 NXN 对高级别浆液性卵巢癌细胞株的促癌作用。质谱法检测到与 NXN 结合的蛋白质 DVL1，通过核质分离、Western Blot、免疫荧光和荧光素酶报告基因检测等多种手段证实 NXN 正向调控 DVL1、β-catenin、CCND1 的蛋白水平，激活了 Wnt/β-catenin 信号通路。通过 Ligase Assisted Minicircle Accumulation (LAMA)方法构建环状 DNA eccNXN，在卵巢癌细胞中过表达 eccNXN 后，Western Blot 检测宿主基因 NXN、DVL1、β-catenin 的表达增加，且通过 CCK-8 试验、集落形成试验、细胞凋亡试验、迁移侵袭试验评估 eccNXN 对高级别浆液性卵巢癌细胞株的促癌作用。

结果 我们的结果表明：与正常卵巢和正常输卵管相比较，eccNXN 在高级别浆液性卵巢癌中高表达。高表达的 eccNXN 与 HGSOC 患者预后不良相关。另外，我们确定了 eccNXN 和 NXN 在空间上是共定位表达的，并且在表达量上显著正相关。在高级别浆液性卵巢癌中，eccNXN 通过促进宿主基因 NXN 的表达，激活 Wnt/β-catenin 信号通路，从而促进细胞增殖，降低细胞凋亡，促进细胞的迁移和侵袭能力。

结论 我们的研究发现了一种名为 eccNXN 的新型 eccDNA，与其宿主基因 NXN 成正相关，促进了 HGSOC 的生长。我们的工作提供了一个新的、潜在的 HGSOC 诊断策略和临床治疗靶点。

PO_280

卵巢环状小管性索肿瘤一例并文献复习

罗彭华 钟丽君* 李灿怡
西南医科大学附属中医医院

目的 探究卵巢环状小管性索肿瘤（SCTAT）的临床表现、诊治及预后。

材料与方法 对 1 例卵巢环状小管性索肿瘤病例进行分析讨论并复习相关文献。

结果 本例患者以“阴道不规则出血 9 天，发现盆腔肿物 1 天”为主诉，未并发 Peutz-Jeghers 综合征，行经阴道单孔腹腔镜下卵巢肿瘤剔除术，术后病理示左侧卵巢环状小管性索肿瘤，因患者要求保留生殖功能，遂行经期单孔腹腔镜下左侧输卵管卵巢切除术+右侧卵巢活检术+大网膜活检术+宫颈活检术+盆腔淋巴结活检术，术后随访至今，未见复发表现。

结论 SCTAT 是一种罕见的卵巢性索间质瘤，无特征性临床表现，以内分泌紊乱及盆腔占位症状为主，部分 SCTAT 病例可并发 Peutz-Jeghers 综合征。目前，SCTAT 的诊疗体系尚未完善，诊断上以其镜下特征性环状小管样结构为依据，治疗上首选手术切除。因其常见盆腹腔淋巴结转移，故多选择切除单侧附件术联合同侧盆腔淋巴结及主动脉旁淋巴结清扫术。对于无明显恶性特征且有生育需求的患者，通常选择保留对侧附件或剔除肿瘤并保留双侧附件，但存在较高的复发风险，而对于复发患者仍首选手术治疗。部分 SCTAT 病例存在浸润、转移、复发等恶性进程，故患者术后需严密随访，CD99、MIS、CA-125、雌激素受体、孕激素受体等指标可辅助术后监测，但不具特异性。根据目前报道，本病术后是否需要补充放化疗尚无统一标准，有待进一步研究。

PO_281

代谢组学联合转录组学揭示上皮性卵巢癌网膜转移代谢脆弱性

田济铭[1] 雷婷[1,2] 杨永秀[2*]
1.兰州大学第一临床医学院 2.兰州大学第一医院

研究目的 高级别浆液性卵巢癌是最具侵袭性的病理类型，其特征包括起病隐匿、腹腔内快速转移和大量腹水产生。大网膜作为转移首站，腹内脂肪堆积，微环境组分复杂，被冠以转移前温床。肿瘤转移伴有强烈代谢适应，然而现有卵巢癌网膜转移研究多聚焦于肿瘤相关细胞及信号通路，鲜有描述网膜转移代谢特征。

材料与方法 靶向代谢组学用于评估原发肿瘤和网膜转移灶中的代谢物丰度，转录组学用于评估原发肿瘤和网膜转移灶中代谢相关基因表达水平。利用 ID8/Luc 细胞分别构建原位及腹腔播散卵巢癌小鼠模型，精氨酸缺乏饮食用于评估精氨酸对肿瘤体内生长及转移的影响。CCK8 实验、划痕实验、Transwell 迁移、侵袭实验用于评估补充精氨酸或过表达 SLC7A1 对癌细胞增殖。迁移。侵袭的影响。精氨酸偶联磁珠联合蛋白质谱富集并鉴定潜在的精氨酸结合蛋白。免疫荧光、western blotting 及免疫组化用于评估蛋白在不同组织、细胞中的定位及表达水平。

结果 在本研究中，我们通过代谢组学联合转录组学发现卵巢癌网膜转移时精氨酸合成缺陷，依靠外源性摄取。体内实验表明，精氨酸剥夺抑制小鼠原位移植卵巢肿瘤生长、转移。体外实验表明，补充精氨酸或增强精氨酸摄取促进了癌细胞增殖、侵袭和迁移。机制研究表明，精氨酸主要通过结合 DDX3X，促进其核滞留并增加 DNA 损伤响应相关基因转录，以确保癌细胞在面临转移及铂类药物治疗时 DNA 损伤得以及时修复，进而存活并侵袭、迁移。

结论 本研究证实精氨酸代谢在上皮性卵巢癌网膜转移的代谢脆弱性，靶向精氨酸代谢是治疗卵巢癌转移的潜在策略。

PO_282

ATP 响应型 MOF-801@MnOOH 纳米释药体系激活 STING 通路增强 PARPi 抗卵巢癌机制研究

程琳 姚勤*
青岛大学附属医院

研究目的 肿瘤免疫治疗已成为恶性肿瘤治疗的关键策略，其中 STING 通路作为调控先天免疫应答的核心信号通路，在激活抗肿瘤免疫反应中发挥关键作用。PARP 抑制剂在卵巢癌治疗中虽能显著改善患者生存预后，但由于其靶向性不足导致的全身毒性严重限制了临床获益。

材料与方法 首先将介孔 MOF-801 纳米颗粒、PARP 抑制剂与 MnOOH 量子点在一定条件下进行组装，然后利用透射电镜和扫描电镜对纳米材料的形貌进行表征。利用 CCK8、划痕、平板克隆等实验从体外细胞水平探讨可注射 MMP 纳米颗粒对卵巢癌细胞的抗肿瘤作用；皮下移植瘤模型从体内水平验证可注射 MMP 纳米颗粒对卵巢癌的抗肿瘤作用及安全性。最后通过体外细胞实验对比卵巢癌细胞与正常细胞的葡萄糖摄取、乳酸产生及 ATP 水平差异，验证纳米材料靶向高 ATP 及酸性环境可精准定位肿瘤部位；ELISA 试剂盒检测检测与 STING 通路相关的细胞因子浓度，比较不同处理组间细胞因子分泌量的差异，评估纳米释药体系是否通过激活 STING 通路促进免疫细胞的活化。

结果 体外实验表明，MMP 对卵巢癌细胞具有显著杀伤作用，并能有效抑制癌细胞增殖、迁移和侵袭；体内实验证实，MMP 可显著抑制裸鼠移植瘤生长，并诱导肿瘤组织广泛坏死，且未引起明显全身毒性。同时机制研究表明，MMP 通过靶向肿瘤高 ATP 微环境实现精准递送，MnOOH 量子点释放的 Mn2+激活 cGAS-STING 通路，促进 cGAMP 生成，上调 STING、TBK1、IRF3 和 IFN-β 表达，并增强 CD8+T 细胞和巨噬细胞活化。此外，MMP 可诱导癌细胞氧化应激，表现为 ROS 水平升高和 GSH 消耗。

结论 本研究通过整合 PARP 抑制剂的靶向递送与 STING 通路激活策略，创新性地构建了基于肿瘤高糖代谢特征的协同治疗纳米释药体系——MMP，其"代谢干预-免疫激活"的协同机制对拓展肿瘤靶向治疗策略具有重要启示意义，兼具临床转化潜力与理论创新价值。

PO_283

Folate-targeted NIR-responsive injectable electrospun short fibers for ovarian cancer treatment and its mechanism research.

He Junqi Qin Yao*
The Affiliated Hospital of Qingdao University

Background The treatment of ovarian cancer faces challenges such as insufficient tumor targeting and low solubility of traditional chemotherapy drugs, leading to a series of systemic adverse reactions including hematological toxicity, gastrointestinal reactions, and neurotoxicity. Therefore, there is an urgent need to develop an advanced drug delivery system that ensures effective drug administration while minimizing these adverse reactions.
Methods In this study, we designed and developed a multifunctional poly lactic acid (PLA) nanofiber system based on electrospinning. First, PLA nanofibers were prepared via electrospinning, followed by cutting and homogenization to obtain PLA nanofibers. These were then modified with dopamine coating to adsorb docetaxel (DTX), and finally conjugated with folic acid (FA) to achieve active targeting. FTIR and XPS were used to characterize the chemical modifications, SEM was used to observe the morphology of the short fibers, and the photothermal performance of the drug delivery system was evaluated. SKOV3 ovarian cancer cells, which highly express folic acid, were used as a model. Drug toxicity was assessed using the CCK8 assay, and targeted uptake, cell apoptosis, and mitochondrial function were analyzed using confocal microscopy and flow cytometry. Finally, the targeting efficiency, tumor suppression effect, and biosafety of the short fiber system were evaluated through in vivo experiments.
Results The results showed that the short fiber diameter was uniformly distributed between 0.9 and 1.0 μm, with a DTX loading capacity of 4.62 ± 0.2%. FA modification significantly enhanced SKOV3 cell uptake of short fibers. In vitro experiments showed that the apoptosis rate was significantly higher in the P-SF@D/F combined with PTT group compared to the photothermal therapy alone group. In vivo experiments showed that P-SF@D/F combined with PTT significantly inhibited tumor volume reduction without causing obvious systemic toxicity.
Conclusion Therefore, the P-SF@D/F system constructed in this study achieved chemotherapy-photothermal synergistic therapy through folic acid targeting and photothermal controlled release, significantly inhibiting ovarian cancer growth and providing a new method for enhancing tumor targeting and therapeutic efficacy.

PO_284

卵巢癌患者 PARP 抑制剂维持治疗后继发髓系肿瘤 4 例报告及文献综述

王雅芹
山西省肿瘤医院

目的 总结 4 例卵巢癌患者在 PARP 抑制剂（PARPi）维持治疗期间出现治疗相关急性髓系白血病（t-AML）或骨髓增生异常综合征（t-MDS）的临床-分子特征，并复习最新文献，探讨危险因素、发病机制及防治策略。
方法 回顾性收集我院 2022 年 1 月至 2025 年 7 月确诊的 4 例卵巢癌继发髓系肿瘤病例的临床资料、实验室检查、治疗方案及转归；系统检索 PubMed、CNKI、万方数据库截至 2025 年 7 月相关文献，对危险因素、分子机制、监测与干预策略进行归纳分析。
结果 4 例患者年龄 58–63 岁，PARPi 中位维持时间 22（5–49）个月。继发 t-AML 3 例，t-MDS 1 例。所有患者均出现持续/进行性三系血细胞减少，骨髓检查示 RUNX1::CBFA2T3 融合阳性 3 例，复杂核型或 TP53 突变 1 例。截至末次随访，2 例已死亡（OS 分别为 25、29 个月），1 例拟行自体造血干细胞移植，1 例正在阿扎胞苷治疗中。
结果 PARPi 相关髓系肿瘤虽罕见，但致死率高。长期维持治疗、BRCA1 突变及既往多线化疗是共同高危因素。建立“基线筛查-动态监测-早期诊断-及时干预”的闭环管理流程有望降低死亡率、改善长期生存。

PO_285

Clinicopathological features of Chinese ovarian cancer patients with double heterozygosity for cancer-predisposed genes

Yikun Jin[1] Wenyan Wang[2] Manqi Wu[1] Yiming Fan[1] Tongxia Wang[1] Cuiyu Huang[1] Tong Gao[1] Yan Liu[6] Yuan Li[1] Qiyu Liu[1] Hongyan Guo[1,3,4,5*]
1.Peking University Third Hospital
2.Peking University Cancer Hospital Yunnan
3.National Clinical Research Center for Obstetrics and Gynecology (Peking University Third Hospital）
4.Key Laboratory of Assisted Reproduction (Peking University), Ministry of Education
5.Beijing Key Laboratory of Reproductive Endocrinology and Assisted Reproductive Technology
6.School of Basic Medical Sciences, Peking University Health Science Center

Background Double heterozygosity (DH) is rarely reported in hereditary ovarian cancer. The clinicopathological and pedigree features of ovarian cancer patients harboring DH of cancer-predisposed genes are not well established.
Methods This study included ovarian cancer patients who received genetic counseling at Peking University Third Hospital between 2018 and 2024. Among patients who received genetic testing, 75 patients were found to carry germline pathogenic variants (PVs) in BRCA1. In 75 BRCA1 PV carriers, 6 unrelated patients harboring additional germline PV in cancer-predisposed genes were identified. The clinicopathological characteristics and family history of the 75 ovarian cancer patients were collected
Results 6 patients harboring germline BRCA1 variant and a concurrent germline variant in cancer-predisposed genes were identified. Coupling with BRCA1 PV, the additional variant involved MUTYH/RECQL4, RAD51C, RECQL4, BRCA2, RAD54L, and ATM. We did not observe a difference in age at diagnosis between DH carriers (median 56 years) and single BRCA1 carriers (median 51

years). There were no significant differences in other clinicopathological profiles (stage, pathology, tumor behavior, and survival) between the two groups. All the DH patients had a family history of multiple types of cancer. The presence of ovarian cancer family history was 66.7% (4/6) in DH group and 27.5% (19/69) in single BRCA1 PV group (p = 0.125). In comparison to single BRCA1 PV carriers, a higher percentage of family history of non-ovarian or breast cancer (100% vs. 46.4%, p = 0.025) was observed in DH carriers.

Conclusions Our study suggests that BRCA1 variant seems to drive the phenotypic expressions of ovarian cancer patients with DH. The management of these patients might be like BRCA1-mutated patients. Harboring DH may further increase the family member's chance of acquiring cancer of various types.

PO_286

Metabolic Reprogramming via ARID5A Deficiency Triggers CD8+ T Cell Senescence and Tumor Immune Evasion in HGSOC

Tianhui He
Peking University Third Hospital

CD8⁺ T cell dysfunction drives antitumor immune failure in advanced high-grade serous ovarian cancer (HGSOC). While T cell exhaustion is well-characterized, the mechanisms and prognostic impact of CD8⁺ T cell senescence remain poorly defined. Flow cytometry analysis of ascites (n=99) and tumors (n=96) from treatment-naïve patients revealed elevated senescent CD8⁺ T cell (Tsen) frequencies correlating with shorter platinum-free intervals. In vitro co-culture studies confirmed diminished cytotoxic function of Tsen. Transcriptomic profiling via RNA sequencing of ascites-derived Tsen and non-senescent CD8⁺ T cells (treatment-naïve advanced HGSOC, n=7) identified ARID5A as a key senescence regulator. Mechanistically, ARID5A knockdown induced CD36-mediated fatty acid uptake and lipid accumulation, thereby driving CD8⁺ T cell senescence; this phenotype was reversed through metabolic correction. Both in vitro and in vivo, p38 inhibitor treatment rescued ARID5A expression, alleviated senescence phenotypes, restored cell cycle progression and cytotoxic function, and significantly delayed tumor progression. Furthermore, p38 inhibition synergized with anti-PD-1 therapy in mouse models, markedly enhancing tumor control. Collectively, this study establishes pretreatment Tsen levels as an independent poor prognostic factor in advanced HGSOC and reveals the p38-ARID5A-lipid metabolism axis as a central driver of CD8⁺ T cell senescence. Targeting this axis represents a promising therapeutic strategy to overcome senescence-mediated immunosuppression in ovarian cancer.

PO_287

TRIB3 在卵巢上皮性浆液性肿瘤中的表达及临床意义

王爽
天津市中心妇产科医院

卵巢癌死亡率居妇科恶性肿瘤之首，严重威胁女性健康。TRIB3 属于假性蛋白激酶（Pseudokinase）基因家族，是一个重要的应激基因，可被多种刺激因素诱导上调，在细胞凋亡、血糖和脂质代谢、脂肪细胞分化等过程中发挥着重要作用，探讨 TRIB3 蛋白在卵巢上皮性浆液性肿瘤组织中的表达及其与患者临床病理参数及预后之间的关系具有重要意义。

材料与方法 将标本固定、包埋、连续切片，免疫组化 SP 法（Ultra Sensitive TM SP（小鼠/兔）IHC 试剂盒（中国，迈新））检测 83 例卵巢组织（卵巢浆液性恶性肿瘤组 46 例、卵巢浆液性交界性肿瘤组 14 例、卵巢浆液性良性肿瘤组 13 例、正常卵巢组织组 10 例）（所有病例均为原发性卵巢癌，未行术前放化疗等，并具有完整的临床病理数据，获得伦理委员会批准）中 TRIB3 的表达情况，分析其表达与临床病理参数的关系及对卵巢浆液性癌患者生存预后的影响。

结果 TRIB3 主要表达于细胞质。分析显示 TRIB3 在卵巢浆液性癌表达明显增加，高于浆液性交界性肿瘤组、浆液性良性肿瘤组及正常卵巢组织组。TRIB3 的高表达与患者分期、分化及淋巴结转移无明显相关，但 TRIB3 高表达的患者生存时间明显缩短（P=0.032）。

结论 TRIB3 作为卵巢癌预后因素，影响卵巢癌患者生存有望作为卵巢癌的生物标志物，监测并指导卵巢癌患者的治疗。

PO_288

Characterizing the Intratumoral Microbiome and Metabolome Perturbations in High-Grade Serous Ovarian Cancer

Qianrun Chen Yili Chen Aiting Wang Qiaojian Zou Yan Jia Qiuwen Mai Xiaojun Wang Xiaoying Lin Yan Liao Mengxun Wei Chudan Chi Yuzhou Xiao Jiayun Fang Feitianzhi Zeng Qiqiao Du Junxiu Liu*
The First Affiliated Hospital of Sun Yat-sen University

High-grade serous ovarian carcinoma (HGSOC), the most lethal gynecologic malignancy, poses significant diagnostic and therapeutic challenges due to frequent late-stage detection and high recurrence rates. Emerging evidence indicates that the intratumoral microbiota may influence cancer progression, although their mechanistic roles in HGSOC remain poorly understood. In this study, we performed integrated microbiome and metabolome sequencing analyses on 15 normal fallopian tube epithelial (NF) tissues and 21 HGSOC tissues, revealing a distinct microbiota-metabolite interaction network characteristic of HGSOC. Notably, Staphylococcus species were significantly enriched in HGSOC and demonstrated a negative correlation with serum CA125 levels, suggesting their potential diagnostic value. Metabolomic profiling identified 3,084 differentially abundant metabolites in HGSOC compared with normal controls, with significant perturbations in metabolic pathways including amino acid biosynthesis, cysteine/methionine metabolism, and purine metabolism. Particularly, Staphylococcus-positive tumors exhibited marked alterations in amino acid synthesis and purine metabolic pathways. Our study provides the first evidence of Staphylococcus-mediated metabolic reprogramming in HGSOC and highlights microbial-host metabolic crosstalk as a potential therapeutic target. These findings establish a theoretical foundation for developing microbiota-based diagnostic and therapeutic strategies for ovarian cancer.

PO_289

The Study of Antitumor Effects of Realgar-Indigo Naturalis Formula (RIF) Combined with Apatinib on Platinum-Resistant Ovarian Cancer

Qi Wang
Peking University People's Hospital

Objective Ovarian cancer represents one of the most lethal gynecological malignancies, approximately 20-30% of patients exhibit primary platinum resistance, while up to 75% develop acquired resistance, leading to poor prognosis. Overcoming platinum resistance in ovarian cancer remains a major clinical challenge, and outpatient oral combination therapy represents an innovative,

clinically valuable strategy to address this unmet need. The Realgar-Indigo naturalis formula (RIF), an oral arsenic-containing traditional Chinese medicine with tetra-arsenic tetra-sulfide (As4S4) as its primary active component, has demonstrated clinical efficacy in leukemia treatment. Notably, arsenic compounds exhibit synergistic effects when combined with chemotherapeutic agents, showing remarkable therapeutic potential against both hematologic malignancies and solid tumors. Apatinib (Aitan®, brand name in China), a novel oral small-molecule anti-angiogenic agent independently developed in China, has demonstrated promising efficacy in platinum-resistant ovarian cancer. The convenience of oral administration has improved patient adherence, enhanced their quality of life, and reduced medical costs, making it a potential new model for outpatient chemotherapy or "home chemotherapy". At present, whether arsenic combined with oral anti-angiogenic drugs in the management of ovarian cancer, particularly in cases demonstrating platinum resistance, exerts synergistic anti-tumor effects is less reported at home and abroad. Consequently, this research is designed to investigate the antitumor effect of RIF combined with Apatinib on platinum-resistant ovarian cancer, and to establish a theoretical foundation for novel therapeutic strategies in the clinical management of ovarian cancer.

Materials and Methods The cisplatin-resistant ovarian cancer cell line A2780/DDP was established by continuous exposure of parental A2780 cells to increasing cisplatin concentrations, achieving a resistance index of 4.51. Cell viability was assessed using CCK-8 assays after treatment with As_4S_4 (0.15-19.2 μg/mL), Apatinib (1-80 μM), or their combination (using IC50 concentrations) for 24, 48, and 72 h, with absorbance measured at 450 nm following standard protocols. Apoptosis was quantified via flow cytometry using Annexin V-FITC/propidium iodide (PI) staining. For in vivo studies, female BALB/c nude mice were subcutaneously inoculated with A2780 or A2780/DDP cells and randomized into four treatment groups: (1) control (PBS), (2) RIF (135 mg/kg/d, oral gavage), (3) Apatinib (200 mg/kg/d, oral gavage), or (4) RIF+Apatinib. Tumor volume was measured every 3 days, and tumors were weighed post-treatment to calculate the tumor growth inhibition rate (TGI). Microvessel density was evaluated by CD31 immunohistochemistry in excised tumor tissues. Statistical analyses were performed using GraphPad Prism 9.5, with one-way ANOVA for group comparisons (significance: $P<0.05$).

Results As_4S_4 and Apatinib inhibited proliferation in both A2780 and A2780/DDP cells in a dose- and time-dependent manner. Combination therapy significantly enhanced cytotoxicity and apoptosis compared to monotherapy ($P < 0.001$). In vivo, RIF and Apatinib alone reduced tumor growth ($P < 0.001$), with the combination group showing the greatest suppression ($P < 0.001$). CD31 staining revealed reduced angiogenesis in all treatment groups, with the most pronounced effect in the combination group.

Conclusions This study demonstrates that both RIF (containing As_4S_4) and Apatinib exert significant antitumor effects against platinum-resistant and platinum-sensitive ovarian cancer in vitro and in vivo. Notably, their combination exhibits synergistic efficacy, surpassing monotherapy outcomes by enhancing proliferation inhibition, apoptosis induction, and anti-angiogenic activity. The oral bioavailability of these agents presents a clinically advantageous, cost-effective strategy with potential to improve patient compliance and quality of life—particularly in outpatient or maintenance settings. Our preclinical results establish the RIF-Apatinib combination as a highly promising therapeutic approach for platinum-resistant ovarian cancer. Future clinical studies are needed to fully evaluate its therapeutic potential and facilitate translation into clinical practice.

PO_290

Analysis of risk factors and prediction model construction for endometrial lesions in postmenopausal ovarian benign tumors

Tianqi Wang[1,2] Lina Tian[1,2] Yingmei Wang[1,2*]
1.Tianjin Medical University General Hospital
2.Tianjin Key Laboratory of Female Reproductive Health and Eugenics

Background Ovarian benign tumor (OBT) is a common clinical gynecological disease. The incidence of postmenopausal OBT has been increasing in recent years, with a global incidence of approximately 5%~17%. Bilateral salpingo-oophorectomy is currently recommended for postmenopausal OBT, and whether the uterus needs to be removed simultaneously is controversial. Postmenopausal OBT patients are at increased risk for endometrial lesions and some postmenopausal OBT patients were unexpectedly found to have combined endometrial lesions after undergoing hysterectomy and bilateral salpingo-oophorectomy. In this study, we proposed to analyze the clinicopathological characteristics of postmenopausal OBT patients and evaluate the risk factors for combined endometrial lesions, aiming to provide reference for the surgical scope selection of postmenopausal OBT patients by constructing a preoperative prediction model.

Methods This study was a retrospective analysis of 471 postmenopausal OBT patients hospitalized in the Obstetrics and Gynecology Department of Tianjin Medical University General Hospital from January 2018 to December 2024. According to whether there was endometrial lesion, the group was divided into postmenopausal OBT with endometrial lesions group (OBT-E group, 40 cases, 8.5%) and postmenopausal OBT without endometrial lesions group (OBT group, 431 cases, 91.5%). Clinical and pathological data of patients were analyzed and compared the differences between the two groups. Independent risk factors for combined endometrial lesions in postmenopausal OBT patients were included in the preoperative prediction model and model validation was performed.

Results According to preoperative diagnostic curettage and postoperative pathological results, 36 patients were combined with endometrial lesions, including endometrial irregular hyperplasia (n=18, 50.0%), endometrial simple hyperplasia (n=9, 25.0%), endometrial complex hyperplasia (n=5, 13.9%), endometrial atypical hyperplasia (n=3, 8.3%), and endometrial carcinoma (n=1, 2.8%). Serous cystadenoma was the most common ovarian tumor in postmenopausal OBT patients (146 cases, 30.9%). Ovarian fibroma was the most common in the OBT-E group (12 cases, 33.4%). Serous cystadenoma was most common in the OBT group (141 cases, 32.5%). Univariate analysis revealed statistically significant differences between the OBT-E group and the OBT group in menarche age, endometrial thickness under ultrasound, solid tumors under ultrasound, E2, FSH, LH, CA125, WBC count, PLT count, and NEU count ($P < 0.05$). The median CA125, median WBC count, median PLT, and median NEU counts in the OBT-E group were higher than those in the OBT group, the proportion of endometrial thickness under ultrasound >4 mm, the proportion of solid tumors under ultrasound, and the proportion of E2≥10pg/mL were higher than those in the OBT group, and the median menarche age, median FSH, and median LH were lower than those in the OBT group. Menarche age<14 years, endometrial thickness>4mm under ultrasound, solid tumor under ultrasound and E2≥10pg/mL were independent risk factors for endometrial lesions in postmenopausal OBT patients($P=0.005$, $P<0.001$, $P=0.019$, $P=0.004$). The total predictive model score is 5.627, and a score of ≥ 3.063 points is considered to be high risk of combined endometrial lesions. Total hysterectomy and bilateral salpingo-oophorectomy are recommended for these patients.

Conclusions The clinical prediction model constructed in this study for patients with postmenopausal OBT with endometrial lesions has good predictive effect and can predict the risk of postmenopausal OBT with endometrial lesions before surgery, providing reference for the diagnosis and treatment of postmenopausal OBT patients.

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Phase II study of Sintilimab combined with nab-paclitaxel in the treatment of platinum-resistant recurrent ovarian cancer

Nan Zhang Yanhua Bai Hong Zheng*
Peking University Cancer Hospital and Institute

Objective To evaluate the efficacy, safety, and tolerability of sintilimab combined with nab-paclitaxel in patients with platinum-resistant recurrent epithelial ovarian, fallopian tube, or primary peritoneal cancer.

Methods This single-arm phase II trial enrolled 28 patients with platinum-resistant recurrent ovarian cancer (PROC). Patients received sintilimab (200 mg, d1) plus nab-paclitaxel (260 mg/m², d1) intravenously every 3 weeks. The primary endpoint was the objective response rate (ORR), and secondary endpoints included progression-free survival (PFS), disease control rate (DCR), and safety. The median age was 57 years, 96.4% had high-grade serous histology, 67.9% were BRCA wild-type, 57.1% had prior bevacizumab use, and 50% had a combined positive score (CPS) of PD-L1 ≥1. Efficacy was evaluated every 3 cycles, with treatment continued until progression or intolerable toxicity.
Results Efficacy: The ORR was 35.7% (10 partial responses), and DCR was 64.3%. With a median follow-up of 11.8 months, median PFS was 4.28 months, and median overall survival (OS) was 15.83 months.
Stratified analysis: Patients with CPS ≥1 had an ORR of 42.9%, and those with CPS ≥10 had an ORR of 50%, though PFS did not differ significantly ($p>0.05$). Patients with tumor-infiltrating lymphocytes (TILs) ≥10% showed a significantly higher ORR (72.7% vs 12.5%, $p=0.005$) and longer median PFS (15.57 months vs 3.4 months).
Safety: Treatment-related adverse events included leukopenia (28.6%), neutropenia (25.0%), anemia (46.4%), and peripheral neuritis (28.6%). Grade 3-4 toxicities were rare, and no treatment-related deaths occurred.
Conclusions Sintilimab combined with nab-paclitaxel demonstrates promising efficacy (ORR 35.7%) and acceptable safety in PROC. TILs ≥10% may serve as a potential biomarker for predicting immunotherapy response, suggesting this regimen as a novel treatment option for PROC patients.

PO_292

Targeting NETs resistance genes CXCR4/MIF/CCL5: Exploration of novel prognostic models and immunotherapy targets for ovarian cancer

Qiuhua Hong[1] Longde Shen[2*] Jimiao Huang[3,4]
1.Jinjiang Municipal Hospital Jinnan Branch
2.Quanzhou First Hospital Affiliated to Fujian Medical University
3.College of Clinical Medicine for Obstetrics & Gynecology and Pediatrics, Fujian Medical University
4.Fujian Maternity and Child Health Hospital College of Clinical Medicine for Obstetrics & Gynecology and Pediatrics

Objectives To investigate the prognostic value and immune microenvironment regulation mechanisms of neutrophil extracellular trap (NET)-related drug resistance genes (NETRDRRGs) in ovarian cancer (OV), and to construct a prognostic risk model for clinical application.
Materials and Methods We integrated TCGA-OV (n=425) and GTEx normal ovarian tissues (n=88) as the test set, with GEO datasets (GSE26712/GSE49997, n=389 OV and 10 controls) for validation. NETRDRRGs were screened from GeneCards and PubMed (275 NET-related genes ∩ 1,897 drug-resistant genes), yielding 85 candidates. Limma identified differentially expressed NETRDRRGs

(NETRDRRDEGs). A prognostic model was built via LASSO-Cox regression and validated using time-dependent ROC, KM survival analysis, nomogram, calibration curves, and decision curve analysis (DCA). Biological mechanisms were explored via GO/KEGG enrichment, GSEA/GSVA, PPI network, and ssGSEA immune infiltration analysis.

Results

1.Model Construction:12 NETRDRRDEGs were identified. LASSO regression selected CXCR4, MIF, and CCL5 to form the model: RiskScore = CXCR4×(-0.128) + MIF×(-0.16) + CCL5×(-0.037).

2.Prognostic Value:High-risk group had significantly shorter OS (KM: $p<0.001$; HR=2.71, 95%CI:1.72–4.28).Then4/5/6-yearAUCs were 0.613/0.626/0.653 (moderate accuracy).Nomogram showed RiskScore contributed most to prognosis.DCA confirmed significant net clinical benefit at 4–6 years.

3.Model Validation:CXCR4/MIF/CCL5 were upregulated in OV vs. controls ($p<0.001$) and between risk groups ($p<0.001$).The model maintained stratification power in GEO cohort (KM: $p<0.001$).

4.Mechanism Exploration:Functional enrichment revealed roles in chemotaxis regulation and cytokine pathways (e.g., KEGG:04062).GSVA highlighted dysregulation of proteasome degradation and oxidative phosphorylation pathways (adj.$p<0.05$).ssGSEA showed significant alterations in 19 immune cells (e.g., activated $CD8^+$T cells) in high-risk group ($p<0.05$), with CCL5 strongly correlating with activated $CD8^+$T cells ($r=0.947$, $p<0.05$).

Conclusion The NET-related drug resistance model (CXCR4/MIF/CCL5) effectively predicts survival risk in OV patients. Its mechanism may involve regulating immune infiltration and proteasome pathways, providing novel targets for OV therapy.

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前列腺六跨膜上皮抗原 3（STEAP3）介导线粒体功能障碍并通过调节铁死亡影响卵巢癌细胞的恶性生长

梁文华[1] 杨萍[2*]
1.石河子大学医学院
2.新疆生产建设兵团医院

目的 本研究旨在探究 STEAP3 对卵巢癌细胞的影响及潜在作用机制，为卵巢癌的药物治疗提供潜在靶点。

方法 细胞实验：采用 Western blot、qRT-PCR 检测人卵巢表面上皮细胞（IOSE80）（对照）和卵巢癌细胞（HO-8910PM、SKOV3、OVCAR3、A2780）中 STEAP3 的表达，选出 STEAP3 高表达的细胞系用于敲低研究、低表达的细胞系用于过表达研究。经慢病毒载体转染 STEAP3，在 SKOV3 细胞系中稳定敲低、OVCAR3 细胞系中稳定过表达；采用 CCK-8 法、EdU、细胞划痕实验、Transwell 实验检测 STEAP3 对卵巢癌细胞恶性生长的影响。通过公共数据库富集分析、扫描电镜、CCK-8、EdU、细胞划痕实验、Transwell 实验、荧光显微镜、Western blot 等探究 STEAP3 与线粒体功能障碍及铁死亡的关系。

结果

（1）WB、qRT-PCR、CCK-8、EdU、细胞划痕、Transwell 实验证实，敲低 STEAP3 分子能抑制卵巢癌细胞的增殖、迁移及侵袭，过表达 STEAP3 则与之相反；（2）公共数据库富集分析显示，STEAP3 在铁死亡通路中显著的富集；CCK-8 证实，通过铁死亡抑制剂（Fer-1）、凋亡抑制剂（Z-VAD-FMK）、坏死抑制剂（Nec-1）、自噬抑制剂（3-MA）干预，只有 Fer-1 逆转了敲低 STEAP3 后卵巢癌细胞的增殖抑制。（3）扫描电镜证实，敲低 STEAP3 可导致卵巢癌细胞出现铁死亡特有表现（线粒体萎缩、嵴减少或消失、膜密度增加）；（4）荧光显微镜、WB 证实，敲低 STEAP3 后卵巢癌细胞总活性氧、线粒体活性氧、Fe2 增加，线粒体膜电位降低；铁死亡蛋白 GPX4（降低）、FTH1（降低）、FACL4（增加）、NOX1（升高）；线粒体氧化呼吸链关键蛋白降低；（5）荧光显微镜、WB 证实，过表达 STEAP3 后卵巢癌细胞总活性氧、线粒体活性氧、Fe2 降低，线粒体膜电位增加；

铁死亡蛋白 GPX4（增加）、FTH1（增加）、FACL4（降低）、NOX1（降低）；线粒体氧化呼吸链关键蛋白增加；（6）CCK-8、EdU、细胞划痕、Transwell、WB 实验证实，加入铁螯合剂（DFO）逆转了敲低 STEAP3 后卵巢癌细胞的增殖抑制、迁移抑制及侵袭抑制，DFO 同样逆转了敲低 STEAP3 后卵巢癌细胞铁死亡蛋白及线粒体氧化呼吸链关键蛋白的表达。（7）CCK-8、EdU、细胞划痕、Transwell、WB 实验证实，加入铁死亡激活剂（Erastin）逆转了过表达 STEAP3 后卵巢癌细胞的促进增殖、促进迁移及侵袭作用，Erastin 同样逆转了过表达 STEAP3 后卵巢癌细胞铁死亡蛋白及线粒体氧化呼吸链关键蛋白的表达。

结果 STEAP3 可介导线粒体功能障碍并通过调节铁死亡影响卵巢癌细胞的恶性生长。

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卵巢浆黏液性交界性肿瘤 3 例病例报道

陈嘉懿
重庆医科大学附属第一医院

卵巢浆黏液性交界性肿瘤是一种相对罕见的卵巢上皮性交界性肿瘤，它具有独特的临床病理特征。但是目前为止，关于其术前诊断的影像学表现以及血清学表现包括 CA125，并非具有特异性诊断的效果，目前确诊卵巢浆黏液性交界性肿瘤主要依赖病理诊断的结果。但该疾病好发于育龄期年轻的女性，危害育龄期女性的生活质量以及生育力。在本次报道里面发现其伴发病变可见子宫内膜异位症，回顾既往研究也能发现似乎卵巢浆黏液性交界性肿瘤与子宫内膜异位症的关系密不可分。联想于子宫内膜异位症相关卵巢癌 EAOC，设想内异症与卵巢浆黏液性交界性肿瘤之间是否存在潜在联系以及相应机制？该交界性肿瘤是否为内异症发展为恶性病变中间的中间过渡形态，其深入机制还需要我们更加进一步的研究。

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NCALD 靶向去甲基化激活 Bax/Caspase-3/PARP 信号通路诱导细胞凋亡逆转卵巢癌铂类耐药

冯利园 李力*
广西医科大学附属肿瘤医院

研究目的 前期研究发现 NCALD 高甲基化卵巢癌患者化疗耐药和预后不良；明确 NCALD 甲基化调控卵巢癌铂类化疗耐药的分子机制，为临床诊治卵巢癌铂类化疗耐药提供治疗靶点有重要意义。

材料与方法 利用 CRISPR-dCas9 系统构建靶向 NCALD 去甲基化调控质粒。靶向去甲基化质粒转染耐药卵巢癌细胞。焦磷酸测序检测位点甲基化水平。QRT-PCR 和 WB 检测基因的表达水平。CCK8 检测细胞的 IC50；通过慢病毒转染在 SKOV3 和 A2780 中下调 NCALD，流式细胞术检测细胞凋亡，WB 检测凋亡信号通路蛋白的表达。

结果 pINDUCER dCas9-TET1CD 和 gRNA-NCALD1-5 共转染 72h 后，SKOV3DDP 和 A2780DDP 细胞中 NCALD mRNA 表达水平明显升高(分别上调 1.92 和 3.87 倍)，蛋白表达水平明显升高(0.26±0.03 VS 0.60±0.03；0.31±0.21 VS 0.85±0.13；P 均＜0.05)。IC50 均有明显的下降(P 均＜0.05)。在顺铂 1.0ug/ml 浓度作用 48h 时沉默 NCALD 组细胞(SKOV3-NCALD-sh757 和 A2780-NCALD-sh757)凋亡率显著下降(P<0.05)，BAX，cleaved-caspase3 和 cleaved-parp 蛋白表达显著下调。

结论 NCALD 靶向去甲基化后的 SKOV3DDP 和 A2780DDP 细胞对顺铂的敏感性增加。NCALD 甲基化转录失活通过调控 Bax/Caspase-3/PARP 信号通路介导细胞凋亡参与卵巢癌铂类化疗耐药。

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新型 HK2 抑制剂增强卵巢癌对 PARPi 的敏感性

施秀 曹玉敏 王娟 陈友国 周金华*
苏州大学附属第一医院

研究目的 本研究旨在探究己糖激酶 HK2 非经典调节糖代谢作用，发现其可以调节卵巢癌对 PARPi 的敏感性，基于此，进一步研发一种新型小分子 HK2 抑制剂 HIC-29，并探究 HIC-29 与 PARPi 联合应用在卵巢癌中产生协同致死效应的机制。

材料与方法

(1) 双向调节 HK2，CCK8 实验、克隆形成实验、彗星实验、同源重组修复实验、非同源末端连接实验检测 HK2 与 PARPi 敏感性；

(2) 利用分子操作环境（MOE）对 HK2 蛋白进行建模，筛选 Chembridge 化合物库，对排名前 50 的候选化合物进行 HK2 及其下游底物 IkBa-pT291 的相互作用检测，最终确定 HIC-29 为最具潜力的候选药物；

(3) WesternBlot 实验、Co-IP 实验、质谱实验、免疫荧光实验、体外蛋白激酶实验检测 HK2 相互作用蛋白，探寻内在分子作用机制；

(4) 临床标本组织芯片多色荧光检测 HK2 与 CHK2 和 γH2AX 之间的相互关系以及患者预后关系；

(5) 体内动物实验验证新型 HK2 抑制剂 HIC-29 与 PARPi 联合使用疗效及安全性：使用 Hey 细胞系，构建小鼠原位移植瘤模型，给与 HIC-29 和 PARPi 联合用药，连续治疗 4 周，测量肿瘤大小及重量，检测血常规和生化指标，免疫组化检测相关蛋白表达，观察二者联合用药效果及安全性。

研究结果

(1) HK2 在 Hey 和 ES2 细胞中表达较高，两种细胞株中敲减 HK2 后，CCK8
实验表明细胞对奥拉帕利的敏感性显著增强，克隆形成实验和彗星实验等表明抑制 HK2 显著增强了卵巢癌细胞对奥拉帕利的敏感性，两者具有协同作用;

(2) 新型 HK2 抑制剂 HIC-29 对 HK2 表现出选择性且强效的抑制作用；可以抑制 HK2 激酶活性，HIC-29 处理可显著抑制癌细胞生长，并增强细胞对 PARP 抑制剂的敏感性;

(3) 机制层面，质谱实验、Co-IP 实验表明 HK2 可以和 CHK2 相互作用, 体外蛋白激酶实验表明 HK2 可以磷酸化 CHK2，HK2 敲低及 HIC-29 处理可减少 CHK2 磷酸化，损伤 DNA 同源重组修复。这些发现揭示了 HK2 在 DNA 同源重组修复通路中的关键作用，并证实 HIC-29 能显著增强卵巢癌对 PARP 抑制剂的敏感性。

(4) 临床样本组织芯片多色荧光发现 HK2 在卵巢癌中高表达，且与患者不良预后相关，HK2 表达与 CHK2 以及磷酸化的 CHK2 表达呈正相关；

(5) 将 HK2 高表达的卵巢癌细胞株建立 NCG 小鼠肿瘤模型，单独或者联合应用 HK2i 与 PARPi，发现两者的协同治疗效果显著，且毒副作用并无增加;进一步验证 HK2 抑制剂可以增强卵巢癌对 PARPi 敏感性。

研究结果 HIC-29 通过削弱 CHK2 磷酸化诱导 DNA 同源重组修复缺陷，增强了 卵巢癌对 PARPi 敏感性，为卵巢癌患者提供了新的治疗策略。

PO_297

铂耐药型上皮性卵巢癌 HOXA10 靶向 NOX4 改善铂耐药的机制研究

杜新月 哈春芳*
宁夏医科大学

目的 铂耐药是上皮性卵巢癌（EOC）治疗失败的首要原因，尽管已知 DNA 损伤修复、PI3K-AKT-mTOR 信号通路及免疫微环境紊乱参与耐药，但针对上游核心转录调控因子的研究仍不足。HOXA10 作为同源框转录因子在胚胎发育及肿瘤发生中的作用已被报道，然而其与卵巢癌铂耐药的关系尚无系统研究。因此，本研究拟首次阐明 HOXA10 靶向 NOX4 调控细胞周期、凋亡及肿瘤免疫微环境，从而逆转铂耐药的新机制；
方法 基于 TCGA-OV 数据集，利用 DESeq2 分析 HOXA10 表达分组的差异基因，并通过 GSEA v4.3.2 对差异基因进行通路富集分析；利用 cBioPortal、GTEx 和 TCGA 数据库分析了 HOXA10 的基因改变及其表达情况，通过一系列生物信息学工具和方法，如 TISIDB 数据库、ESTIMATE 和 CIBERSORT 方法，研究了 HOXA10 表达在临床相关性、预后、肿瘤免疫微环境中的作用；构建铂耐药 EOC 细胞模型，慢病毒转染干预 HOXA10 表达，通过 Western blot、流式细胞术等检测 NOX4、细胞周期（CDK4/6、Cyclin D）、凋亡及 PI3K/AKT 通路变化；建立裸鼠移植瘤模型验证耐药逆转效应；
结果 WikiPathways GSEA 结果显示，HOXA10 高表达组显著富集 Focal Adhesion–PI3K-Akt-mTOR 信号通路（NES = 2.101，P < 0.001，FDR < 0.001）；HOXA10 与 NOX4 表达呈正相关（Spearman R=0.613，p=0.001）；HOXA10 低表达组中 T 细胞浸润减少（CD8+ T 细胞比例下降 40%，p<0.01）；HOXA10 过表达将显著提升 NOX4 mRNA 及蛋白水平（qPCR 及 Western blot 验证），双荧光素酶报告基因实验可验证 HOXA10 结合 NOX4 启动子区域，证实其直接靶向激活作用；HOXA10 过表达细胞周期 G1 期阻滞（Cyclin D1 下调，p21 上调），凋亡率显著升高（Caspase3 上调）；体内实验中 HOXA10 过表达使移植瘤体积缩小，联合顺铂治疗后肿瘤消退率提高，这些变化共同导致顺铂敏感性显著提升（IC50 降低）；
结论 本研究揭示了 HOXA10-NOX4 分子轴在逆转卵巢癌铂类耐药中的关键作用，不仅为深入理解卵巢癌耐药机制提供了新的理论依据，更重要的是为临床开发基于 HOXA10 靶向的新型治疗策略奠定了实验基础，具有重要的转化医学价值。

PO_298

卵巢癌 SKVO3 细胞中休眠期细胞的干细胞属性研究

周楠* 张成果 韩嘉瑶 李子章 刘晓阳
邢台市人民医院

目的 探讨卵巢癌 SKOV3 细胞中高保留 PKH26 荧光染料的休眠期细胞的干细胞属性及化疗药物对其干细胞属性的影响。
方法 将 PKH26 标记的 SKOV3 细胞接种于裸鼠皮下并用顺铂化疗，建立化疗前、后异种移植瘤模型，应用流式细胞术分选移植瘤中休眠的肿瘤细胞，Real-time PCR 分析休眠的肿瘤细胞的干细胞标记物 Oct-4 mRNA 和 Nestin mRNA 的表达，并将休眠的肿瘤细胞重新接种于裸鼠体内观察其体内成瘤能力。
结果 1 按 PKH26 荧光保留强度将化疗前、后移植瘤细胞分别分为高保留组(PKHhi 细胞)、低保留组(PKHlow 细胞)和完全淬灭组细胞(PKHneg 细胞)；2 PKHhi 细胞即休眠期细胞，其干细胞标记物 Oct-4 mRNA 和 Nestin mRNA 的表达、体内成瘤能力及体外克隆形成能力明显高于其他两组(P <0.05)，且化疗后 PKHhi 细胞 Oct-4 mRNA 和 Nestin mRNA 的表达、体外克隆形成能力均明显高于化疗前 (P <0.05)。

结论 化疗前、后移植瘤中 PKH26 高保留的休眠细胞具有干细胞属性，且化疗后此群细胞的干细胞属性更强。提示这种具有干细胞属性的休眠细胞与肿瘤的耐药存在相关性。

PO_299

RIPK4 Drives Ovarian Cancer Progression via CSF1-Dependent M2 Macrophage Polarization

Leilei Zhu Huan Yi*
Fujian Maternity and Child Health Hospital

Background Ovarian cancer exhibits a high mortality rate. Previous studies have shown that there was a poor prognosis for the patients with the overexpression of the Receptor-Interacting Serine/Threonine-Protein Kinase 4 (RIPK4). The tumor immune microenvironment plays a pivotal role in ovarian cancer progression and its therapeutic response. Our preliminary bioinformatic analysis showed that RIPK4 upregulated tumor-associated macrophages (TAMs) infiltration, especially the M2 phenotype TAMs. In this study, we aim to uncover how RIPK4 orchestrates TAM recruitment and polarization, ultimately driving tumor progression through macrophage-dependent mechanisms, thereby providing new targets for precision therapy.
Methods In vitro studies, we used recombinant lentiviruses to knock down RIPK4 expression in SKOV3 cell lines and overexpress RIPK4 in A2780 cell lines. Human monocytic leukemia cell line (THP-1 cells) were induced to differentiate into macrophage-like cells by Phorbol 12-myristate 13-acetate. Quantitative polymerase chain reaction(qPCR) was used to assess CSF1 mRNA expression and M1 and M2 macrophage-associated markers. Macrophage migration was assessed using Transwell assays. In vivo studies, RIPK4-overexpressing A2780 cells were injected subcutaneously to female BALB/c nude mice for model tumorigenesis. AZD7507 (CSF1R inhibitor) was administered to rescue RIPK4-driven cell migration promotion and tumorigenesis.
Results RIPK4 knockdown significantly reduced CSF1 level in conditioned medium (CM) as well as the CSF1 mRNA level in SKOV3 cells, while RIPK4 overexpression significantly upregulated CSF1 mRNA expression in A2780 cells ($P < 0.05$). qPCR results showed that the CM from RIPK4-overexpressing cells significantly promoted THP-1 cells to differentiate into more M2-like macrophages. The Transwell assay showed that macrophage migratory capacity was enhanced by CM from RIPK4-overexpressing cells and attenuated by the CSF1R inhibitor AZD7507 ($P < 0.05$). In the subcutaneous xenograft tumor model, the inhibition of CSF1 receptor by AZD7507 can significantly reversed the tumor-promoting effect induced by RIPK4 overexpression.
Conclusion RIPK4 promotes the expression and secretion of CSF1. Through CSF1, RIPK4 induces macrophage polarization towards the M2 phenotype and enhances the migratory capacities of TAMs. Inhibition of CSF1 signaling significantly attenuates the tumor-promoting effects of RIPK4.

PO_300

卵巢癌同源重组修复缺陷状态临床病理特征及预后分析

张可[1,2] 沈杨[1,2*]
1.东南大学附属中大医院 2.东南大学医学院

目的 卵巢癌作为女性生殖系统常见的恶性肿瘤之一，具有显著的临床异质性和复杂的组织学特征。PARP 抑制剂在肿瘤治疗过程中，发挥着非常重要的抗肿瘤作用，特别是对于同源重组修复缺陷（homologous recombination deficiency，HRD）状态的卵巢癌患者具有较好作用。本研究探讨卵巢癌 HRD 与同源重组修复熟练（homologous recombination proficiency，HRP）临床病理特征及预后之间的内在联系，筛选影响卵巢癌患者预后的危险因素，并构建列线图模型，为评估患者预后提供有价值的参考信息。
材料与方法 本研究纳入 2020 年 1 月至 2023 年 12 月于东南大学附属中大医院经手术治疗后，病理明确诊断为卵巢癌，且经 HRD 状态检测的患者。通过 Lasso 回归筛选对卵巢癌患者预后具有影响的危险因素，基于 Cox 比例风险回归模型进行列线图的构建。采用 AUC 作为评估列线图模型预测性能的关键指标，校准曲线评估列线图模型预测值与实际观测值之间的一致性，决策曲线分析评估列线图模型的临床应用价值。
结果 本研究共纳入 70 例卵巢癌患者，其中 HRP 状态的卵巢癌患者为 23 例，HRD 状态的卵巢癌患者为 47 例。在对 HRD 与 HRP 两组患者进行分析后，结果显示两组患者在 HE4 水平、手术时间、肿瘤组织类型、肿瘤分期、肿瘤原发部位、P53 表达、Ki67 表达等临床病理特征方面存在显著差异。卵巢癌患者 FIGO 分期、PR 表达与靶向治疗情况，是影响其预后的危险因素。研究构建的列线图性能良好，总生存期预测方面，在 1 年、2 年和 3 年这 3 个时间点的 AUC 分别为 93.3%，85.6%与 87.4%。
结果 本研究基于东南大学附属中大医院的患者信息，成功开发了一个整合临床病理特征的列线图。该模型性能优异，具有较高的准确性和可靠性，借助该模型，临床医生能够有效预测卵巢癌患者的生存率，识别高危人群，为卵巢癌患者的临床诊治工作提供重要的参考依据。

PO_301

PPARα 介导胆固醇代谢调控卵巢癌紫杉醇耐药性相关机制研究

陈明卓 王新宇*
浙江大学医学院附属第一医院

研究目的 探究过氧化物酶体增殖物激活受体 α（peroxisome proliferators-activated receptor α，PPARα）介导胆固醇代谢调控卵巢癌耐药性的机制，为卵巢癌化疗紫杉醇耐药提供新的治疗思路。
材料与方法 本研究以人浆液性卵巢癌细胞系 A2780 和 SKOV3 为实验模型，通过低浓度紫杉醇持续诱导建立紫杉醇耐药细胞系（A2780/TR 和 SKOV3/TR）。采用细胞计数试剂盒实验、流式细胞术（细胞凋亡检测）及蛋白质免疫印迹实验（多药耐药蛋白 1 的表达）评估耐药性。尼罗红染色观察细胞内脂滴分布。对敏感株与耐药株进行非靶向脂质组学分析。采用胆固醇定量试剂盒检测胆固醇水平，蛋白质免疫印迹实验检测胆固醇关键调控蛋白表达。
结果 耐药株的脂滴数量减少但体积增大（P<0.01），胆固醇酯水平显著升高（Log2(FC)>9）。总胆固醇及关键代谢蛋白——固醇调节元件结合蛋白 2（ SREBP2）、3-羟基-3-甲基戊二酰辅酶 A 还原酶（HMGCR）、低密度脂蛋白受体（LDLR）和乙酰辅酶 A 乙酰转移酶 1（ ACAT1）在耐药株中均显著上调（P<0.05）。辛伐他汀处理可降低耐药性（P<0.01），提示胆固醇代谢参与化疗耐药。PPARα 在耐药株中表达上调（P<0.05），敲低或抑制 PPARα 会增强敏感株的耐药性（P<0.001），而过表达或激活 PPARα 则降低耐药株的耐药性（P<0.01）。机制上，PPARα 敲低可上调胆固醇水平

（P<0.001）及 SREBP2、HMGCR、LDLR、ACAT1 的表达（P<0.05），而过表达则相反（P<0.05）。敲低 PPARα 可逆转辛伐他汀诱导的耐药性降低（P<0.001），表明 PPARα 通过调控胆固醇代谢影响紫杉醇耐药。

结论 PPARα 通过调节胆固醇代谢调控卵巢癌紫杉醇耐药性，靶向 PPARα 及其下游通路或可为治疗卵巢癌化疗耐药提供新策略。

PO_302

Alternative Splicing Profiling in Different Pathological Subtypes of Ovarian Cancer

Zihan Li Xihan Liu Kun Song*
Department of Obstetrics and Gynecology, Qilu Hospital of Shandong University

Objective Although accumulating evidence indicates that alternative splicing is aberrantly altered in ovarian cancers, the underlying functional mechanisms remain to be fully elucidated.

Methods We analyzed RNA splicing data from a large cohort of primary ovarian cancer tumors collected at Qilu Hospital to identify differentially spliced events using rMATS . These findings were further validated using data from The Cancer Genome Atlas (TCGA) cohort.We then performed GSEA to detect the most enriched HALLMARK pathways. EMT score Calculations, Sequence analysis of these events, along with differential expression analysis of RNA-binding proteins (RBPs), identified key genes. Additionally, we employed a developmental algorithm to reveal key evolutionary patterns of tumor cells in response to drug treatments. Lentiviral transduction enabled us to modulate the expression of ESRP1 in ovarian cancer cells. Through CLIP-seq analysis and sequence motif enrichment analysis, we identified SPAG9 as a downstream molecule regulated by RBFOX2 in splicing modulation. Utilizing CRISPR-Cas9 technology, we performed targeted knockout of specific exons or motifs in SPAG9 that interact with upstream splicing factors. Both in vivo and in vitro experiments demonstrated the functional significance of exon 28 in tumorigenesis. Furthermore, semi-quantitative PCR and gel electrophoresis were employed to validate the RBFOX2-mediated splicing events.

Results To investigate the alternative splicing (AS) landscape of ovarian cancer, we performed RNA sequencing on 114 OV patients (including 22 HGSOC, 14 LGSOC, 18 CCOC, 21 ENOC, 16 MOC and 23 normal samples) from Qilu Hospital of Shandong University (QL cohort). we visualized the most spliced events (|dPSI|>0.2, N = 676), and the result demonstrated that samples could be divided into two groups (Noaml&Group1 and Group2) based on the PSI values.The results of GO and KEGG imply us that there maybe a change of cell state when these splicing aberrations occurred between Group1 and Group2. We then performed GSEA to detect the most enriched HALLMARK pathways and the result demonstrated that Epithelial Mesenchymal Transition (EMT) pathway ranked first. To better definite the disease state of Group1 and Group2, we used two EMT score calculation methods to measure the EMT level and the results showed significantly differences. Thus, we defined samples as M-like tumors, while defined Group2 as E-like tumors. Notably, we observed that E-like and M-like ovarian cancer subtypes exhibited specific splicing alterations associated with poorer prognosis, which was further validated in the TCGA cohort. Furthermore,our results revealed that both RBFOX2 and ESRP1 are key regulators of these splicing events. Consistently, ectopic expression of ESRP1 in ovarian cancer cells significantly reduced their invasive and proliferative capabilities in vitro, while ESRP1 deletion enhanced these properties. We therefore selected two groups of events with the most pronounced discrepancies for further investigation. These findings indicated that the expression of SPAG9 splicing variants closely correlatedwith ESRP1 and RBFOX2 levels. Subsequent cell experiments showed that knockout of SPAG9 exon 28 significantly inhibited cell proliferation and migration, and promoted apoptosis. Moreover, in vivo experiments confirmed theimpact of exon 28 on tumor growth. We also found that knockout of the motif in SPAG9 that binds RBFOX2 led to skipping of SPAG9 exons, which is consistent with the splicing event changes we previously validated following RBFOX2 knockdown.

Conclusion In conclusion, Our integrated analysis establishes alternative splicing dysregulation as a defining feature of ovarian cancer heterogeneity. We identified two molecular subtypes—E-like (epithelial) and M-like (mesenchymal)—driven by prognostic-associated splicing events and validated across independent cohorts. These subtypes exhibit divergent EMT activation, with RBFOX2 and ESRP1 emerging as master regulators of subtype-specific splicing programs. These findings position SPAG9 splicing variants as central mechanistic links between splicing regulators (RBFOX2/ESRP1) and ovarian cancer progression, revealing novel therapeutic targets for subtype-specific treatment strategies.

PO_303

Multi-omics identification of SPON1-related signature for prognosis and immunotherapy response in ovarian cancer

Zouyu Zhao Yajing Yu Songping Liu*
Jinshan Hospital of Fudan University

Objective Ovarian cancer (OC) has the highest mortality rate among malignant tumors of the female reproductive system owing to late-stage diagnosis, high recurrence and metastasis rates. Therefore, it is urgent to find reliable targets for early diagnosis and treatment of OC. Spondin-1 (SPON1), a secreted extracellular matrix protein, has been demonstrated to be over-expressed in multiple tumor tissues and cell lines types, but the carcinogenic mechanism of SPON1 in OC remains unclear.
Materials and Methods The transcriptomes of ovarian cancer and normal ovarian samples, along with their corresponding clinical information, were obtained from the Gene Expression database of Normal and Tumor tissues (GENT2), The Cancer Genome Atlas (TCGA), The Genotype-Tissue Expression (GTEx), and Gene Expression Omnibus (GEO) databases. Next, we confirmed the mRNA and protein levels of SPON1 in OC samples using quantitative real-time PCR (qPCR) and immunohistochemistry (IHC). Gene set enrichment analysis (GSEA) analysis was used to investigate the possible mechanisms by which SPON1 promotes the progression of OC. Differential expression analysis and univariate Cox analysis were used to identify SPON1-related prognostic genes. Least Absolute Shrinkage and Selection Operator (LASSO) regression and multivariate Cox analysis were used to construct a SPON1-based prognostic signature, and the predictive effectiveness of this signature was validated using GEO database. The risk signature, combined with patients' clinical pathological factors (age, stage and grade), was used to construct a nomogram model. Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) analyses were used to explore the changes of biological process in different risk subgroups. Additionally, the association between the prognostic signature and tumor immune microenvironment, immunotherapy responsiveness, somatic mutations, and drug sensitivity was assessed.
Results Pan-cancer analysis showed that SPON1 was highly expressed in 19 types of cancer compared with normal samples, especially in OC. IHC and qPCR consistently confirmed that SPON1 was significantly up-regulated in OC tissues and cell lines. Kaplan-Meier survival analysis revealed that high SPON1 expression predicted poor prognosis of patients. Functionally, we observed that SPON1 gene level positively correlated with multiple cancer-related signaling pathways, including WNT, MAPK and JAK-STAT3. We identified 333 SPON1-related genes and constructed and validated a 6-gene prognostic model. Multivariate Cox regression analysis demonstrated that this model is an independent risk factor for OC in TCGA and GSE53963 cohorts. Moreover, the nomogram model has better predictive value for the overall survival (OS) of OC patients. Functional analyses indicated that multiple cancer progression and immune-related pathways were remarkably enriched. This prognostic model demonstrated a high correlation with immune cell infiltration and immune checkpoint genes. High-risk patients displayed poor response to immunotherapy and common drugs for chemotherapy in

ovarian cancer and external cohorts. Furthermore, this signature also showed association with tumor mutation burden (TMB).
Conclusion In summary, our findings provided novel insights into the potential mechanisms by which SPON1 promoted the OC progression and developed a SPON1-based risk model for predicting prognosis as well as therapeutic response of OC patients.

PO_304

一例 BRCA1 基因突变发生逆转的卵巢高级别浆液性癌患者的管理

袁华 赵丹*
中国医学科学院肿瘤医院

45 岁女性患者，初始在我院诊断卵巢高级别浆液性癌 IIIC 期，我院一线治疗包括：紫杉醇联合卡铂方案新辅助化疗 2 周期；中间型减瘤手术；紫杉醇联合卡铂方案辅助化疗 4 周期。肿瘤组织标本基因检测结果提示患者携带 BRCA1 c.3294del，p.Pro1099fs 突变。一线化疗后患者行奥拉帕利维持治疗，治疗 11 月后肿瘤复发。患者停用奥拉帕利，继续在我院接受二线治疗，包括：白蛋白结合型紫杉醇联合奈达铂、贝伐珠单抗方案化疗 5 周期；聚乙二醇化盐酸多柔比星脂质体联合奈达铂方案化疗 1 周期，影像学评估最佳疗效为疾病稳定（Stable disease，SD）。患者 6 周期化疗后在我院接受开腹卵巢肿瘤细胞减灭术，针对复发减瘤手术标本再次行基因检测提示患者携带 BRCA1 c.3277_3312del,p.V1093_K1104del 突变。通过对碱基序列分析提示患者的 BRCA1 基因突变发生逆转。患者至今在我院已接受治疗 6 年，期间复发 6 次，现仍在治疗中。

PO_305

Podoplanin Expression as a Predictor of Venous Thromboembolism in Ovarian Cancer Patients: A Comprehensive Analysis

Hui Liu Hailing Feng Siyu Yang Qiongqiong He Wenqing Yang Yu Zhang Di Xiao Yan Tian*
Xiangya Hospital Central South University

Objective This study aimed to investigate the association between podoplanin expression and venous thromboembolism (VTE) risk in patients with epithelial ovarian cancer. Given the high incidence of VTE in ovarian cancer patients and the need for reliable biomarkers to predict thrombotic events, this research sought to evaluate podoplanin as a potential predictor of VTE occurrence and examine its relationship with microvessel density and patient prognosis. The study also aimed to characterize VTE patterns and timing in ovarian cancer patients to better understand thrombotic risk factors.
Methods A retrospective case-control study was conducted at Xiangya Hospital of Central South University, examining all patients diagnosed with epithelial ovarian cancer between December 1, 2016, and May 31, 2021. Sixty patients with VTE confirmed by imaging were enrolled as cases, while 108 ovarian cancer patients without VTE were matched 1:2 by histological type as controls. Exclusion criteria included patients under 18 years, previous VTE history, high thrombotic risk comorbidities, non-epithelial ovarian cancers, and incomplete clinical data. VTE screening involved dual lower extremity Doppler ultrasound for deep vein thrombosis (DVT) detection and computed tomography pulmonary angiography (CTPA) for suspected pulmonary embolism (PE). Clinical information including patient demographics, disease characteristics, and treatment factors were extracted from electronic medical records. Immunohistochemistry (IHC) staining was performed on formalin-fixed, paraffin-embedded

tumor specimens using monoclonal mouse anti-podoplanin antibody 18H5 and CD34 for microvessel density assessment. Podoplanin expression was scored by multiplying percentage of positive cells (0-4 scale) by staining intensity (0-3 scale), with immunoreactivity categorized as negative (0-1), weakly positive (2-3), moderately positive (4-8), and strongly positive (9-12). Receiver operating characteristic (ROC) curve analysis determined optimal cutoff values. Statistical analyses included t-tests, Chi-square tests, Kaplan-Meier survival analysis, and Cox proportional hazards models using SPSS 22.0 and GraphPad Prism 8.0, with $P<0.05$ considered significant.

Results The study population showed significant differences between VTE and non-VTE groups. VTE patients were older (58.8 vs. 54.3 years, P=0.002) and had elevated baseline D-dimer levels (1.08 vs. 0.78 mg/L, P=0.001). Among 60 VTE cases, 33 patients had DVT, 9 had PE, and 18 had both conditions. VTE occurrence peaked within 10 days post-surgery (61.7%), followed by diagnosis time (30.0%). Podoplanin expression was significantly higher in VTE patients compared to controls (4.56±2.69 vs. 3.34±2.16, P=0.003). PE patients showed higher podoplanin expression than DVT patients (5.30±2.27 vs. 3.95±2.88, P=0.027). Preoperative VTE patients demonstrated higher podoplanin expression than postoperative VTE patients (5.70±2.68 vs. 3.94±2.52, P=0.014). ROC analysis identified 3.55 as the optimal podoplanin expression threshold. Univariate analysis revealed that high podoplanin expression (≥3.55) conferred 3.02-fold increased VTE risk (HR=3.02, 95% CI: 1.55-5.87, P=0.001). Multivariate analysis confirmed this association after adjusting for age and D-dimer levels (HR=3.50, 95% CI: 1.72-7.12, P=0.001). Cumulative VTE incidence at 180 days was significantly higher in high podoplanin expression patients (47.67% vs. 21.25%, log-rank P=0.003). High podoplanin expression correlated with increased microvessel density (median MVD: 20.00 vs. 15.60, P=0.032). Regarding prognosis, patients with high podoplanin expression showed trends toward longer progression-free survival (32.53 vs. 18.67 months, P=0.056) and overall survival (66.13 vs. 51.40 months, P=0.265), though differences were not statistically significant.

Conclusion In conclusion, our study indicates that podoplanin may serve as a significant marker for assessing VTE risk in ovarian cancer patients, particularly in predicting the risk of pulmonary embolism. Future research should investigate the mechanisms underlying podoplanin-mediated thrombosis and integrate it with other biomarkers to improve VTE risk prediction and formulate personalized treatment strategies.

PO_306

Real world study on the prognostic impact of neoadjuvant chemotherapy on epithelial ovarian cancer

Li Li*
Department of Gynecologic Oncology, Guangxi Medical University Cancer Hospital

Objective To explore the prognostic significance and related influencing factors of neoadjuvant chemotherapy (NACT) in the treatment of advanced ovarian cancer (AOC) based on real-world data.

Methods A retrospective analysis was conducted through a real-world study (RWS) on the data of 404 patients with stage III-IV AOC who were initially treated at the Affiliated Cancer Hospital of Guangxi Medical University from 2008 to 2018. All patients were treated with NACT or primary debulking surgery (PDS) as the initial treatment plan, with 200 patients in the NACT group and 204 patients in the PDS group. The differences in prognosis between the NACT group and PDS group were compared, and a column chart was drawn to predict the prognosis of NACT patients. Logistic regression model, decision tree model, and artificial neural network (ANN) model were established to jointly judge the prognosis of patients, and their predictive performance was compared with ROC curve.

Results (1) After 1:1 matching, the median overall survival (OS) of patients in the NACT group and PDS group were 50 months and 61 months, respectively, with 5-year survival rates of 42% and 50% (p=0.42), median progression free survival (PFS) of 25 months and 20 months, and 3-year recurrence rates of 60.4% and 63.6% (p=0.24), respectively. (2) The COX model analysis results showed that

staging, chemotherapy efficacy, and post NACT CA125 were independent prognostic factors affecting the overall survival of patients in the NACT group. At the same time, staging and post NACT CA125 were also independent prognostic factors affecting PFS in patients in the NACT group. (3) Logistic univariate and multivariate analysis showed that stage IV, IDS with a time interval of ≤ 10 days from the first postoperative chemotherapy, and a time interval of>42 days between chemotherapy before and after IDS were high-risk factors for mortality in the NACT group. However, the failure of CA125 to decrease to normal after NACT, a time interval of<21 days from the last NACT to IDS, and a total chemotherapy course of ≥ 6 courses were high-risk factors for recurrence in the NACT group. By comparing their predictive performance with the ROC curve, it was found that logistic model, decision tree model, and ANN model all showed certain predictive performance for predicting death events. Among them, the ANN model had relatively high predictive performance, and the combination of three indicators (stage+IDS to first postoperative chemotherapy time+chemotherapy time interval before and after IDS) had the highest predictive performance (AUC: 0.688); For the prediction of recurrence events, logistic model, decision tree model, and ANN model all demonstrate high predictive performance. Among them, all three models have the best predictive performance when combined with three indicators (CA125 after NACT+time from last NACT to IDS+total chemotherapy course) (AUC of 0.729, 0.725, 0.729, respectively).
Conclusion NACT does not shorten the survival time of advanced epithelial ovaries, but there are still many factors that affect the prognosis of NACT patients. Therefore, optimizing the NACT regimen and course of treatment, striving to reduce CA125 levels to normal after NACT, managing IDS during the perioperative period, and shortening the chemotherapy interval may be feasible ways to improve the prognosis of NACT patients. In addition, the column chart model and the combined prediction of multiple models and indicators for predicting the prognosis of NACT patients also demonstrate certain predictive performance, which can provide new ideas for predicting the prognosis of ovarian cancer in the future.

PO_307

CPT1A 通过促进脂代谢及铁死亡抵抗，导致卵巢癌 PARPi 耐药

周琳 姜旖* 程文俊
南京医科大学第一附属医院

目的 卵巢癌是致死率最高的妇科恶性肿瘤，早期诊断困难和获得性耐药性导致大多数患者预后不佳。目前 PARPi 维持治疗改善了患者的生存获益，然而随着药物的广泛使用，部分卵巢癌患者逐渐出现 PARPi 耐药现象。新出现的证据表明，肿瘤细胞中氧化还原系统失衡可能在化疗耐药中发挥关键作用，恢复肿瘤细胞铁死亡敏感性已被证明是癌症治疗中的潜在治疗策略。然而，铁死亡逃逸如何导致卵巢癌 PARPi 耐药的机制尚未得到证实。
方法 通过诱导卵巢癌 PARPi 耐药株，并检测耐药株与敏感株脂质组学，分析脂质差异，以 PCR、WB 等验证 CPT1A 在耐药株中表达增加，通过 cck8、Edu、裸鼠成瘤等实验验证 CPT1A 促进体内体外 PARPi 耐药形成；通过检测细胞内脂质含量，验证耐药细胞株中脂质分解增强；通过敲低 CPT1A，检测 ROS、MDA，并验证铁死亡通路相关蛋白等指标，验证 CPT1A 通过影响铁死亡敏感性导致 PARPi 耐药。
结果 ①成功建立卵巢癌 PARPi 耐药株②耐药株中 CPT1A 明显增加，敲低 CPT1A 后，体内体外实验验证肿瘤细胞 PARPi 敏感性增强③CPT1A 促进肿瘤细胞脂质分解，并导致肿瘤细胞铁死亡抗性增强，从而促进 PARPi 耐药
结论 我们通过研究发现，CPT1A 通过促进卵巢癌细胞脂质代谢，同时增强肿瘤细胞抗氧化系统反应，产生铁死亡抗性，促进卵巢癌 PARPi 耐药，为克服 PARPi 耐药性提供了一种新的治疗靶点。

PO_308

CLDN4 靶向肽偶联 SN38 治疗卵巢癌的潜力研究

张玉沙 许祎 沈杨*
东南大学附属中大医院

研究目的 本研究旨在评估基于课题组前期确定的 CLDN4 靶向肽（Lead-Pcl）与 SN38 构建的新型偶联物（Lead-Pcl-SN38）对卵巢癌的靶向性和治疗效果。结合患者来源类器官模型（PDOs）及荷瘤小鼠模型，系统分析其穿透能力、杀伤活性及安全性，为靶向 CLDN4 的卵巢癌治疗提供实验依据。
材料与方法 靶点表达与预后分析：利用公共数据库（TCGA、GTEx）分析 CLDN4 在卵巢癌中的表达谱。通过 Kaplan-Meier 分析评估 CLDN4 表达水平与患者总生存期（OS）的关系。
靶向肽及偶联物：Lead-Pcl（基于本课题组前期已筛选的高亲和力 CLDN4 靶向肽）通过连接子与 SN38 偶联，合成 Lead-Pcl-SN38，无靶向功能的同型对照偶联物 Ctrl-SN38。使用质谱及紫外分光光度计验证偶联物。
体外实验：构建 CLDN4 高表达的 skov3 细胞系，分组（PBS、游离 SN38、Ctrl-SN38、Lead-Pcl-SN38）给药处理，CCK-8 法检测细胞活力并计算 IC_{50}。利用 SN38 的自发蓝色荧光特性通过共聚焦显微镜观察 Lead-Pcl 在细胞系及卵巢癌患者来源的类器官(PDOs)中的内化及 Lead-Pcl-SN38 的靶向穿透情况。
体内实验：建立卵巢癌 PDX 小鼠模型，荷瘤鼠尾静脉分组（PBS、SN38、Ctrl-SN38、Lead-Pcl-SN38）给药，通过监测肿瘤体积及生存期、体内肿瘤切片 KI67 及 tunnel 染色进行疗效评价，重要脏器 HE 染色评估安全性。
结果 CLDN4 预后价值： CLDN4 在卵巢癌中显著高表达（$p<0.001$），且高表达患者 OS 显著缩短（HR=1.4, p=0.015）。
体外高效杀伤：通过侵袭、迁移、成球及 CCK8 等试验说明 Lead-Pcl-SN38 的杀伤作用与 SN38 无统计学差异。
优异靶向穿透性：采用改良培养体系成功构建卵巢癌患者来源类器官（PDOs），共聚焦成像显示，注射后 6 h 即可观察 Lead-Pcl-SN38 在 PDOs 中的深度浸润，穿透深度可达 120±15 μm。
体内抑瘤与安全性：Lead-Pcl-SN38 组肿瘤抑制率显著高于 SN38 组和 Ctrl-SN38 组，并延长小鼠中位生存期，各实验组脏器病理均未见显著毒性损伤。
结果 本研究证实 CLDN4 是卵巢癌的不良预后因子及可靠治疗靶点。基于前期开发的靶向肽 Lead-Pcl 构建的偶联物 Lead-Pcl-SN38，在患者类器官及动物模型中均表现出优异的肿瘤选择性穿透能力、显著强化的抗肿瘤活性及良好的安全性。该偶联物通过主动靶向递送策略克服了 SN38 的系统毒性缺陷，为 CLDN4 高表达卵巢癌的精准治疗提供了具有转化潜力的候选药物。

PO_309

卵巢癌中 CD226 对 NK 细胞、T 细胞的功能影响及调控通路研究

花敏慧* 薛颖卓
南通大学附属医院

研究目的 研究免疫检查点分子 CD226 在卵巢癌微环境中对 NK 细胞、T 细胞的肿瘤杀伤功能的影响，探究 CD226 在卵巢癌微环境中调控 NK 细胞、T 细胞免疫功能的关键通路，为 CD226 在卵巢癌免疫治疗中的研究提供新的理论证据。

材料与方法（1）将卵巢癌细胞 OVCAR3 与 NK-92、JURKA-T 细胞体外共培养，模拟肿瘤微环境，加入 CD226 抗体后，采用流式细胞术检测 CD226 被抑制后 NK-92、JURKA-T 细胞对 OVCAR3 细胞杀伤率的变化； RT-qPCR 检测两组间 NK-92、JURKA-T 细胞分泌细胞毒性因子（GZMA、GZMB、穿孔素 Perforin）、TNF-α、IFN-γ、肿瘤坏死因子相关凋亡诱导配体（TRAIL）；IL-6 的 mRNA 表达差异。（2）收集足月无并发症胎儿脐带血（UCB），分离出原代 NK、T 细胞并对提取的原代细胞进行鉴定，将经鉴定后纯度达 80%以上的原代 NK、T 细胞与 OVCAR3 细胞进行共培养，流式细胞术检测两组间 OVCAR3 细胞死亡的占比差异。（3）对 CD226 被抑制的 JURKA-T 细胞进行转录组测序，并对 DEGs 进行 KEGG、GO 与 GSEA 富集分析筛选出有意义的通路。RT-qPCR 及 Western blot 验证 PI3K-AKT 通路上游重要 DEGs 的表达。

结果 （1）NK-92 实验中 α-CD226 组平均细胞死亡占比显著低于 IgG 组（P＜0.01）；JURKAT 实验中 α-CD226 组平均细胞死亡占比同样显著低于 IgG 组（P＜0.001）。抑制 CD226 后 NK 细胞、T 细胞的肿瘤杀伤功能下降。（2）RT-PCR 结果显示 α-CD226 组 NK-92 细胞分泌的 GZMA、GZMB、Perforin、IFN-γ、TRAIL、IL-6 mRNA 表达均显著降低（P＜0.05），但 TNF-α mRNA 表达两组间无明显差异（P＞0.05）；α-CD226 组 JURKA-T 细胞分泌的 GZMA、GZMB、Perforin、TNF-α、TRAIL、IL-6 mRNA 表达均显著降低（P＜0.05）。（3）脐血提取 NK 细胞中 CD3-细胞占比为 98.4%，CD3+T 细胞占比为 86.1%。原代 NK 细胞配对样本实验中，α-CD226 组细胞死亡占比显著下降（P＜0.05）；原代 T 细胞配对样本实验中 α-CD226 组细胞死亡占比显著降低（P＜0.05）。（4）RNA 测序及 DEGs 富集分析结果显示抑制 CD226 与 PI3K-AKT 通路显著相关：PI3K-AKT 通路上游重要因子 EGFR、FGFR1、FGFR4、TGFA、ITGA4 及 COL4A2 均显著下调；P-AKT 蛋白表达降低。

结论 （1）在卵巢癌微环境中，抑制 CD226 显著降低 NK、T 细胞对 OVCAR3 细胞的杀伤作用。（2）CD226 低表达通过影响 PI3K-AKT 通路调节 T 细胞功能。

PO_310

High expression of LIPG is associated with platinum resistance and poor prognosis in epithelial ovarian cancer

Li Li*
Department of Gynecologic Oncology, Guangxi Medical University Cancer Hospital

Purpose Ovarian cancer, characterized by the lowest five-year survival rate among gynecological malignancies, has seen chemotherapy resistance emerge as the predominant contributor to its unfavorable prognosis. Endothelial lipase LIPG-mediated lipid metabolic reprogramming is critically implicated in the pathogenesis, progression, and chemoresistance of ovarian carcinoma, yet how such metabolic rewiring modulates platinum-based chemotherapy responsiveness remains incompletely elucidated.

Methods To elucidate LIPG-associated molecular mechanisms and platinum resistance dynamics, a protein-protein interaction (PPI) network was constructed using LIPG-binding partners, followed by functional enrichment analysis of interconnected genes. Platinum-resistant ovarian cancer datasets integrating gene expression profiles and clinical records were analyzed to identify chemoresistance-related pathways via gene set enrichment analysis (GSEA). Immune cell infiltration patterns were quantitatively assessed across tumor microenvironments. Clinical validation involved immunohistochemical (IHC) staining and quantitative reverse transcription PCR (qRT-PCR) to evaluate LIPG expression correlations with chemotherapeutic outcomes. Two machine learning approaches—logistic regression and decision tree algorithms—were implemented to establish predictive models, with ROC curve analysis verifying the prognostic utility of CETN2. Survival impacts were determined through Kaplan-Meier methodology, statistically comparing patient outcomes across LIPG expression strata.

Results In clinical samples, LIPG may be associated with immune infiltration. The expression level of LIPG protein in platinum-resistant patients is significantly higher than that in platinum-sensitive patients,

and its expression level has certain predictive value for chemotherapy outcomes, and high expression of LIPG protein is associated with poor progression-free survival.
Conclusion The LIPG protein exerts a substantial impact on platinum-based chemosensitivity and clinical prognosis in ovarian cancer, potentially through its upregulation-driven activation of lipid metabolic reprogramming, thereby facilitating tumor immune evasion. Further investigations are warranted to delineate the therapeutic potential of LIPG, which may serve as a novel predictive biomarker for chemotherapeutic responsiveness.

PO_311

整合素 α6 N-糖基化对卵巢癌转移及血管生成的影响

刘兴燕[1] 杨萍[2*]
1.石河子大学医学院 2.新疆生产建设兵团医院

目的 探究整合素 α6（ITGA6）的 N - 糖基化修饰对卵巢癌转移及血管生成的影响，明确其在卵巢癌细胞侵袭、转移及增殖、血管生成中的作用机制，为卵巢癌治疗寻找潜在新靶点。

方法 细胞筛选：通过 Western blot 检测人卵巢表面上皮细胞（IOSE80）（对照）和卵巢癌细胞（HO-8910，SKOV3，A2780，OVCAR3，OVCAR8，HEYA8）中 ITGA6 蛋白表达，筛选高表达细胞系。
细胞实验：构建 ITGA6 N - 糖基化定点突变细胞模型，采用 Transwell、划痕实验检测细胞侵袭迁移能力，CCK8、EDU 实验检测增殖能力，Western blot 检测 EMT 相关蛋白、周期蛋白、凋亡蛋白、通路蛋白、VEGF-A 表达，成管实验、发芽实验检测血管生成能力（HUVEC 细胞），同时加入通路相关激活剂或通路抑制剂后采用 Transwell、划痕实验检测细胞侵袭迁移能力，CCK8、EDU 实验检测增殖能力，Western blot 检测 EMT 相关蛋白、周期蛋白、凋亡蛋白、通路相关蛋白表达，成管实验、发芽实验检测血管生成能力（HUVEC 细胞）。
机制研究：结合转录组学分析差异基因，采用 Western blot 检测差异基因在卵巢癌细胞系及构建 ITGA6 N - 糖基化定点突变细胞模型中的表达情况，Co-IP 、免疫荧光、共聚焦显微镜检测是否与差异基因发生相互作用，通过 Western blot 检测加入 CHX、MG132 后 ITGA6 蛋白的变化，在构建 ITGA6 N - 糖基化定点突变细胞模型中过表达差异基因，采用 Transwell、划痕实验检测细胞侵袭迁移能力，CCK8、EDU 实验检测增殖能力，Western blot 检测 EMT 相关蛋白、周期蛋白、凋亡蛋白、通路相关蛋白、VEGF-A 表达，成管实验、发芽实验检测血管生成能力（HUVEC 细胞）。
动物实验：利用 GFP 标记的突变细胞构建裸鼠异种移植模型：（1）皮下成瘤和腹腔转移瘤（分别在注射肿瘤细胞后第 7 天、14 天、21 天、28 天使用 IVIS 活体荧光成像系统监测肿瘤生长和侵袭转移情况。（2）采用光声成像技术活体观察比较卵巢癌细胞移植瘤血管生成情况，PEG-GNRs 作为显影剂。为了探索光声成像仪观察皮下瘤血管生成的最佳时间点，分别 PEG-GNRs 尾静脉注射前、注射后立即、12 h 和 24 h 观察光声信号值（PA 值），每次实验重复 3 次。
结果 ITGA6 N-糖基化修饰激活 PI3K/AKT 信号通路促进卵巢癌转移及血管生成从而影响卵巢癌的进展

PO_312

Clinicopathological characteristics and analysis for prognosis of patients with endometriosis associated ovarian cancer

Yu Chen Qian Chen Chunyan Hao Liping Zuo Xingsheng Yang Lijie Wang*
Shandong university Qilu hospital

Objective Investigate the clinicopathological and prognostic characteristics of endometriosis associated ovarian cancer (EAOC) patients. Analyze the factors affecting their prognosis and the predictive value of related factors for death or recurrence in patients with ovarian clear cell carcinoma (OCCC) and ovarian endometrioid carcinoma (OEC).

Materials and Methods A total of 250 patients with primary ovarian cancer, hospitalized for surgical treatment and with postoperative pathology confirmed as OCCC or OEC, who visited the Department of Gynecology, Qilu Hospital, Shandong University from January 2005 to December 2021, were included in the study. The pathological findings were independently reviewed by two pathologists from our institution. Based on the diagnostic criterion of coexisting malignant tissue and endometriosis (EMT) in the ipsilateral ovary, the cases were classified into two groups: the EAOC group (n=71) and the n-EAOC group (n=179). The clinicopathological characteristics and prognostic features were compared between the two groups. Cox proportional hazards regression analysis was performed to identify factors influencing disease-free survival (DFS) and overall survival (OS).

Results Compared with the n-EAOC group, EAOC patients were significantly younger (P<0.001), more likely to be premenopausal (P=0.001), have a history of dysmenorrhea (P<0.001), exhibit lower parity (P=0.002), earlier menarche (P=0.027), shorter menstrual cycles (P=0.029), and demonstrate reduced plasma levels of CA125 (P=0.001) and CA153 (P=0.002). Additionally, they showed lower neutrophil-to-lymphocyte ratio (NLR, P=0.006), fibrinogen (P=0.003), and D-dimer levels (P<0.001).

EAOC cases predominantly presented with unilateral tumor distribution (P=0.010), less frequent ascites (P<0.001), earlier FIGO stages (P<0.001), lower Ki-67 expression (P=0.044), higher rates of optimal cytoreduction (P=0.009), more frequent co-existing adenomyosis (P=0.023), and reduced chemotherapy resistance (P=0.023).

The EAOC group exhibited significantly lower recurrence (P=0.001) and mortality rates (P=0.013), along with higher 2-year disease-free survival (P=0.024) and overall survival (P=0.011) rates. Both DFS (P=0.017) and OS (P=0.049) durations were prolonged in this group.

Cox regression analysis identified gravidity (P=0.002), FIGO stage (P=0.013), and chemotherapy resistance (P<0.001) as independent risk factors for DFS, while chemotherapy resistance (P<0.001) was the sole independent risk factor for OS. Malignant transformation from endometriosis did not independently affect DFS or OS in OCCC and OEC patients (P>0.005).

Conclusion

1. Patients with EAOC exhibited the following characteristics: younger age, higher prevalence of dysmenorrhea history, earlier menarche, shorter menstrual cycles, fewer parities, lower plasma levels of CA125 and CA153, higher incidence of concurrent adenomyosis, earlier FIGO staging, higher rates of R0 resection achievement, and lower incidence of chemotherapy resistance.
2. EAOC patients demonstrated better prognosis; however, origin from endometriotic transformation was not an independent prognostic factor for patients with ovarian clear cell carcinoma or ovarian endometrioid carcinoma.
3. Gravidity, FIGO staging, and chemotherapy resistance were identified as independent risk factors affecting DFS, while chemotherapy resistance was an independent risk factor influencing OS.

PO_313

CYR61 Enhances Crosstalk Between Ovarian Cancer Cells and Tumor-Associated Macrophages by Upregulating IDO1 and PD-L1

YA XIE
郑州大学第一附属医院

Ovarian cancer (OC), characterized by a highly suppressive tumor immune microenvironment (TIME), exhibits poor responsiveness to immunotherapy. Macrophages, as key infiltrating immune cells in OC, contribute significantly to this immunosuppressive landscape. This study investigates the role of CYR61 in modulating the TIME and its interaction with tumor-associated macrophages (TAMs). Using clinical samples from 68 patients, proteomics analysis, CRISPR/Cas9 knockout in SKOV3 cells, dual-luciferase assays, ChIP-qPCR, and flow cytometry, we demonstrated that CYR61 upregulates IDO1 and PD-L1 through IRF1 binding to their promoters in OC cells, thereby promoting M2 polarization of TAMs and facilitating immune evasion. Elevated CYR61 expression was associated with shorter progression-free survival. In vivo, CYR61 overexpression accelerated ascites formation and metastasis, whereas CYR61 downregulated by a novel TMTP1-nano-siCYR61 has the opposite effect. Moreover, TMTP1-nano-siCYR61 treatment significantly reduced kynurenine levels and CCL2 secretion, thereby enhancing antitumor immunity. These findings highlight CYR61 as a potential therapeutic target for improving OC treatment outcomes.

PO_314

Sulforaphane selectively inhibits glucose metabolism of PIK3CA-mutated ovarian cancer

YA XIE
郑州大学第一附属医院

Cancer cells undergo metabolic reprogramming, particularly glucose metabolism, as they transform from normal to malignant states. PIK3CA mutation, which is prevalent in tumors such as ovarian cancer (OC), drives cancer progression by activating the PI3K/AKT pathway. Sulforaphane (SFN), a natural antioxidant derived from cruciferous vegetables, has preventive and antitumor effects against a variety of malignant tumors. However, specific mechanisms underlying these effects remain unclear. In this study, we explored the role of PIK3CA mutations in enhancing glycolysis and the tricarboxylic acid cycle (TCA) cycle in OC cells. Glycolysis and the TCA cycle were enhanced in PIK3CA-mutated OC cells. Moreover, our findings revealed that SFN selectively inhibited glycolysis and the TCA cycle in PIK3CA-mutated cells, thereby suppressing tumor growth. The inhibition of glycolysis by SFN occurred through regulation of the PI3K/AKT pathway, thus allowing for the downregulation of key glycolytic enzymes. These results provide new insights into the effect of PIK3CA mutations on cancer metabolism and suggest a potential therapeutic role for SFN in targeting tumors with specific genetic mutations.

PO_315

STT3 promotes epithelial-mesenchymal transition in epithelial ovarian cancer via the JAK2/STAT3 signaling pathway

Chongfeng Sun Ping Yang*
Department of Obstetrics and Gynecology,Xinjiang Production and Construction Corps Hospital, Urumqi

Background Epithelial ovarian cancer (EOC) constitutes more than 80% of all ovarian cancer(OC). The slow emergence of EOC, together with the absence of effective screening procedures, complicates early diagnosis. Upon initial diagnosis, 75% of patients exhibit peritoneal metastasis. Consequently, elucidating the EOC metastasis mechanism and discovering effective control methods are vital for better patient outcomes and are urgent clinical challenges to address. The STT3 oligosaccharide transferase (OST) complex catalytic subunits A and B (STT3A and STT3B) are the major catalytic subunits of the OST, which mainly mediates cotranslation and post-translocation N-linked glycosylation. However, the role of STT3 in the progression of EOC remains unclear.

Materials and Methods To investigate the role of STT3 in EOC, bioinformatics methods were initially used to analyze its expression. Immunohistochemistry (IHC) was employed to analyze STT3 protein levels in EOC tissues and cell lines. Cell proliferation, migration, and invasion were measured using EdU assays and transwell experiments. A subcutaneous xenograft mouse model was deployed to study the growth of cancer cells in vivo. Furthermore, gene set enrichment analysis (GSEA) was conducted to identify pathways linked to STT3. The expression of proteins involved in epithelial-mesenchymal transition (EMT) and the JAK2/STAT3 signaling pathway was confirmed through western blot analysis. For rigorous experimental research, we conducted a secondary validation by treating OC cells with NGI-1 (a STT3 specific inhibitor).

Results In this study, we analyzed through the GENT2 online data platform and found that the expression level of STT3 was relatively high in OC. The same results were obtained by immunohistochemical experiments. The expression of STT3 was significantly increased in EOC tissues compared with normal fallopian tube tissues, and was associated with decreased overall survival (OS) and progression-free survival (PFS).In addition, high expression of STT3, age ≥55 years old, and FIGO stage (III or IV) are independent risk factors affecting the survival of patients with EOC. STT3 knockdown was successfully achieved in stable OC cell lines by lentivirus transfection, and the results showed that knockdown of STT3 inhibited the proliferation, migration and invasion of OC cells. Subcutaneous xenograft experiments in mice showed that knockdown of STT3 delayed the growth of OC. For rigorous experimental research, we performed a secondary validation by treating OC cells with the STT3-specific inhibitor NGI-1 and obtained the same results. Furthermore, further analysis results through GSEA enrichment indicated that STT3 was associated with the JAK2/STAT3 signaling pathway. Western blot verification indicated that silencing STT3 reduced the phosphorylation level of JAK2/STAT3. In order to further explore the role of JAK2/STAT3 signaling pathway in OC, the experimental group added JAK2/STAT3 signaling pathway inhibitor WP1066. EdU labeling, transwell assay and EMT-related protein detection showed that the WP1066 inhibitor could effectively inhibit the proliferation, migration and invasion of OC cells. Colivelin, a JAK2/STAT3 activator, reversed STT3 deletion induced EMT, suggesting that STT3 may regulate EMT process through the JAK2/STAT3 pathway, thereby affecting the spread and metastasis of OC.

Conclusion In conclusion, our study demonstrated that STT3A and STT3B can be used as prognostic biomarkers for EOC and that downregulation of STT3 can inhibit the migration and invasion of EOC cell lines. This process may play a role in EMT via the JAK2/STAT3 signaling pathway.

PO_316

卵巢恶性肿瘤中医体质类型和证候表现分布规律的 Meta 分析

任静静 虞梁 孟黄洋 万一聪 程文俊*
江苏省人民医院（南京医科大学第一附属医院）

研究目的 卵巢恶性肿瘤作为最致命的妇科肿瘤之一，其特点为早期疾病隐匿、术后复发率高、易出现化疗耐药，严重影响患者预后。尽管现代医学取得了进展，但患者在治疗过程中经常出现多系统并发症和疾病复发，生活质量和长期疗效仍是持续关注的重要问题。传统中医为卵巢恶性肿瘤管理提供了新的视角，利用"辨证论治"方法的个体化优势，在恶性肿瘤的辅助治疗中展现出一定优势。本研究采用 Meta 分析的方法，系统整合国内现有文献证据，调查卵巢恶性肿瘤患者中医体质及证型分布特征，为卵巢癌管理中的临床中医应用提供循证参考。
材料与方法 系统检索中国知网、万方数据库、维普信息以及中国生物医学文献数据库中关于卵巢恶性肿瘤中医体质类型和证候分布的观察性横断面研究。检索时间从各数据库建库至 2025 年 1 月。采用 AXIS 量表进行质量评估，使用 R 4.4.3 软件进行 Meta 分析。以 $P<0.05$ 为有统计学意义。
结果 共纳入 30 项符合纳入标准的研究进行 Meta 分析。结果显示，卵巢癌患者主要表现为阳虚体质。早期疾病主要以血瘀证为特征，而晚期疾病主要与气虚证相关。卵巢癌化疗后患者以脾虚、阴虚、气虚等单一症候以及气阴两虚、气血两虚复合症候表现为著。对于铂类耐药的卵巢癌患者，血瘀证最为常见，复合证型以气滞血瘀证最为突出。
结论 卵巢恶性肿瘤具有“因虚致病、因病致虚、虚实夹杂”的中医证候演变特点，其证型的分布随疾病演变阶段、治疗干预手段及耐药状态而发生变化。卵巢恶性肿瘤患者以阳虚、血瘀、气虚为主要表现，“瘀阻胞脉”“正气损伤”可能为其致病机制；化疗后患者气阴两虚，体现了化疗对脾胃运化及气血阴津的损伤；铂耐药患者以血瘀证和气滞血瘀证为主要特点，提示“瘀毒互结、气机不畅”可能为其中医病机基础。卵巢恶性肿瘤的中医治疗应遵循"扶正祛邪，标本兼治"的原则，按照证候演变规律进行辨证施治，准确识别卵巢恶性肿瘤患者的中医体质类型和症候分布规律，为卵巢癌各期的中医辨证论治提供了客观依据。

PO_317

SOGA1 Promotes Cell Invasion, Metastasis, and Epithelial-Mesenchymal Transition in Ovarian Cancer

Chongfeng Sun Mingqing Li Songping Liu*
Department of Obstetrics and Gynecology, Jinshan Hospital of Fudan University

Background Ovarian cancer (OC) is considered one of the most malignant tumors in the female reproductive system, characterized by high rates of recurrence and metastasis. The lack of reliable biomarkers for the early detection and prognostic assessment of ovarian cancer has resulted in approximately 70% of patients being diagnosed at an advanced stage (III/IV), which complicates treatment and contributes to a poor prognosis. In recent years, the association between metabolic abnormalities, such as glucose autophagy, and tumorigenesis has become a research hotspot. Glucose autophagy-related genes (SOGA1) may influence the metastasis and drug resistance of ovarian cancer by regulating responses to energy stress, although the specific mechanisms remain unclear. This study aims to systematically analyze the molecular characteristics and clinical significance of SOGA1 in ovarian cancer through the bioinformatics integration of multi-omics data, providing a theoretical basis for the development of new biomarkers and targeted strategies
Methods We performed a comprehensive analysis of SOGA1 expression and its prognostic role in OC using multiple public databases, including The Cancer Genome Atlas (TCGA), Gene Expression Omnibus (GEO), and Genotype-Tissue Expression (GTEx). We constructed a SOGA1-related risk

model using LASSO regression and predicted patient survival rates via a nomogram. Immunohistochemistry was used to detect SOGA1 expression in OC tissues of patients, and its associations with clinicopathological factors and prognosis were analyzed. We utilized Western blot and RT-PCR to assess the differential expression of SOGA1 in OC cell lines. After small interfering RNA (siRNA) transfection, we evaluated the effects of SOGA1 on migration, invasion, and proliferation in OC cell lines (SKOV3 and OVCAR3) using scratch assay, transwell assay, and EdU assay. Additionally, based on results from GSEA analysis and transcriptome sequencing, we confirmed the impact of SOGA1 on epithelial-mesenchymal transition (EMT) pathway-related proteins in OC via Western blot analysis.

Results Bioinformatics analysis revealed that significant upregulation of SOGA1 in OC tissues of patients. Further analysis using a prediction framework incorporating four significantly regulated genes (SRGs) demonstrated that SOGA1 acts as an independent risk factor. Kaplan-Meier survival analysis showed that high SOGA1 expression was associated with reduced overall survival (OS). Furthermore, the immunohistochemical staining confirmed high SOGA1 expression in OC tissues of patients, and its elevated expression was significantly associated with multiple clinicopathological parameters, including patient age, FIGO stage, histological grade, and lymph node metastasis. Kaplan-Meier analysis indicated that high SOGA1 expression serves as an independent prognostic factor for poor survival outcomes in OC patients. EdU experiments showed that SOGA1 downregulation reduced proliferation in the OC cell lines SKOV3 and OVCAR3. Cell scratch and transwell assays demonstrated that reduced SOGA1 expression significantly inhibited invasion and migration in OC, suggesting that SOGA1 knockdown impedes OC progression. Transcriptome sequencing results indicated a significant association between SOGA1 and EMT, and the results of western blot analysis confirmed that SOGA1 knockdown suppressed the expression of EMT-related proteins.

Conclusion In conclusion, our study demonstrates that SOGA1 can serve as a prognostic biomarker for OC, and its downregulation inhibits migration and invasion in OC cell lines. This process may act through the EMT pathway.

PO_318

膈下逐瘀汤及其活性成分山奈酚靶向 VEGF 信号通路治疗卵巢癌的多组学研究

虞梁 任静静 孟黄洋 张琳 程文俊*
江苏省人民医院（南京医科大学第一附属医院）

研究目的 卵巢癌是最致命的妇科恶性肿瘤，多数确诊时已处于晚期，腹水形成与腹膜播散密切相关。化疗耐药是临床面临的重大挑战。膈下逐瘀汤（GXZY）在肿瘤治疗中表现出协同增效减毒作用。本研究旨在运用多组学技术系统阐明 GXZY 抗卵巢癌的分子机制，为中医药治疗提供理论依据。

材料与方法 网络药理学预测 GXZY 潜在靶点。建立 C 小鼠卵巢癌腹水模型评价治疗效果。运用多组学技术探究作用机制。孟德尔随机化分析 VEGFA 与腹水因果关系，分子对接和分子动力学模拟鉴定核心活性成分。体外细胞功能验证，免疫组化、免疫荧光评估抗肿瘤效应，血清肝肾功能评估安全性。

结果 网络药理学确定 GXZY 抗卵巢癌靶点 237 个，核心靶点包括 AKT1、JUN、ESR1、MAPK14、VEGFA、KDR 等，主要聚焦于 VEGF 信号通路。孟德尔随机化分析证实 VEGFA 水平升高显著增加腹水形成风险（OR=1.705，P=0.024）。GXZY 治疗减少腹水生成，下调 VEGFA、PI3K-p110β、AKT 和 P-P38 蛋白表达，抑制卵巢癌增殖和迁移，削弱成管能力。单细胞转录组分析揭示 GXZY 重塑肿瘤微环境，显著减少肿瘤细胞、癌相关成纤维细胞和内皮细胞比例（P<0.05），降低 VEGF 信号通讯强度，促进肿瘤细胞分型优化。基因集分析显示增殖、迁移和铂耐药评分下降。宏基因组分析表明 GXZY 通过有益菌丰度，重构肠道微生态平衡，发挥促代谢抗炎的双重调节作用。代谢组学结合网络药理学确定山奈酚为核心活性成分，分子动力学模拟证实其与 VEGFA 结合高度稳定。体内外验证山奈

酚具有与 GXZY 相似的抗肿瘤效应，能够浓度依赖性抑制 PCNA、VEGFA、AKT、PI3K 表达，并发挥铂增敏效果。GXZY 及山奈酚发挥环境适应性肝肾保护。

结论 本研究建立了"网络预测-多组学验证-单体确证"的综合技术路线，系统阐明了 GXZY 及其活性成分山奈酚主要通过靶向 VEGF 信号通路，协同重塑肿瘤微环境和肠道微生态，发挥抗卵巢癌效应和化疗增敏作用。本研究为传统中医药复方现代化研究和恶性肿瘤中西医结合治疗提供了重要科学依据和临床转化基础。

PO_319

Ovarian steroid cell tumor in a teenager masquerading as polycystic ovary syndrome: A case report and literature review

Zhongyi Zhu[1,2] Begam Mohammed Risna[1] Xuan Zuo[3] Ting Wu[4] Yuedong He[1,2] Lin Li[1*]

1.Department of Gynecology, West China Second University Hospital, Sichuan University

2.Key Laboratory of Birth Defects and Related Diseases of Women and Children (Sichuan University), Ministry of Education

3.Department of Pathology, West China Second University Hospital, Sichuan University

4.Department of Ultrasonic Medicine, West China Second University Hospital, Sichuan University

Background Adolescent patients presenting with hyperandrogenic symptoms often receive a diagnosis of polycystic ovary syndrome (PCOS). However, accurate diagnosis is crucial because the symptoms of rare conditions, such as ovarian steroid cell tumor (OSCT), can mimic PCOS.

Methods Chief complaint, personal and family history, physical, laboratory and imaging examination, diagnosis, treatment, and follow-up of the patient are presented. For literature review, while OSCT and its synonyms, ovarian steroid cell tumor, ovary steroid cell tumor, ovarian steroid cell tumor, ovarian steroid cellular tumor, were used, PCOS and its synonyms, polycystic ovarian syndrome, polycystic ovary syndrome, polycystic ovarian syndromes, polycystic ovary syndromes were involved to the search target. These two topics were searched and joined with AND in Pubmed. Other literature types were excluded except case report using the filter provided by Pubmed. Then we limited the search result using tittle/abstract filter provided by Pubmed to get pre-target case reports. All full text of pre-target papers were read and find out similar case reports which report clinical cases of OSCT-NOS misdiagnosed as PCOS. This search strategy was done by two co-authors independently.

Results Case summary: An 18-year-old female presented with irregular menstrual cycles, hyperandrogenic symptoms, and obesity. Despite standard treatments for PCOS, symptoms persisted. Exploratory laparoscopic examination revealed an OSCT. Thorough hormonal profiling, imaging, and histopathological analysis confirmed the diagnosis. Literature review: All OSCT and PCOS with their synonyms were searched in Pubmed on March 1, 2025. After limited the topic to tittle/abstract and then screened manually, only one report was found to present similar OSCT-NOS case with the case we report here.

Conclusion This case highlights the need for accurate diagnosis, early evaluation, and timely intervention in adolescents with hyperandrogenism to manage rare conditions like OSCT. Adolescent females with hyperandrogenic symptoms are often diagnosed with polycystic ovary syndrome (PCOS). However, the symptoms of rare conditions like ovarian steroid cell tumors can mimic symptoms of PCOS. This case highlights the importance of an accurate diagnosis and a thorough evaluation through hormonal profiling, imaging, and exploratory laparoscopy. If the standard PCOS treatments are unsuccessful, then alternative diagnoses such as ovarian steroid cell tumor should be considered. Early identification and appropriate management are critical for satisfactory patient outcomes and emphasize the need for heightened awareness of rare conditions that present similarly to PCOS in adolescents.

PO_320

Preclinical Study on Targeted Nanodrug Inhibiting Peritoneal Metastasis of Ovarian Cancer

YA XIE
郑州大学第一附属医院

Background Ovarian cancer(OC) is one of the most common type of cancer in females.Epithelial-mesenchymal transformation as a key step in tumor metastasis and progression. Cysteine-rich angiogenic inducer 61(CYR61) is a growth factor and plays important regulatory role in diverse biological processes of cancers.However, it remains unclear about the exact role of CYR61 in the development of OC metastasis.
MethodsThe expression and clinical significance of CYR61 were determined by qRT-PCR, western blot，Single-cell sequencing, organoid models, mass spectrometry-based targeted metabolomics analysis in tissues and cell lines of OC. The biological functions of CYR61 in OC were determined by in vivo metastasis assays. And further experiments were performed to explore the molecular mechanisms underlying the functions of CYR61.
Results Our data showed that the high expression levels of CYR61 was positively correlated with metastasis of ovarian cancer. Nano-siCYR61 significantly inhibited ovarian cancer metastasis in vivo，in PDX models and organoid models. Furthermore, we describe the mechanism of nano-siCYR61 in inhibiting OC metastasis through reshaping the immunosuppressive microenvironment of OC.
Conclusions This research showed that nano-siCYR61 significantly inhibited OC metastasis and reshaping the immunosuppressive microenvironment, which might help in improving therapeutic efficacy of OC.

PO_321

表达 VTCN1 的卵巢癌肿瘤上皮细胞在淋巴转移中发挥重要作用

刘均彬 张瑜*
湘雅医院

目的 淋巴结受累是晚期高级别浆液性卵巢癌的不良预后因素，目前卵巢癌驱动淋巴结转移的确切机制还缺乏详细的了解。
方法 对 5 例淋巴结受累（其中 3 例仅有淋巴结转移）和 2 例淋巴结未受累的晚期卵巢癌患者原发灶及淋巴转移进行单细胞测序，比较筛选淋巴转移相关基因，收集 2019-2024 年诊断晚期卵巢癌原发灶 45 例，淋巴转移阳性 26 例，淋巴转移阴性 19 例，通过 RT-qPCR 比较基因表达差异。
结果 对单细胞测序结果分析，将肿瘤上皮细胞聚类分为 0-12 簇，其中第 4 簇在淋巴转移组中数量和占比更高，通过比较淋巴转移原发灶与配对转移灶，发现转移灶与 C4 簇相似性更高，表明 C4 簇肿瘤上皮细胞可能倾向淋巴结转移。为表征 C4 簇，通过文献调研收集实体肿瘤单细胞测序数据与淋巴转移高度相关的基因共 81 个，代入单细胞测序数据，与其他肿瘤细胞簇相比，其中 22 个基因在 C4 簇高表达。RT-qPCR 验证 22 对基因在淋巴转移阳性和淋巴转移阴性临床样本中的相对表达情况，筛选出 VTCN1 在淋巴转移阳性患者原发灶的表达高于淋巴转移阴性患者（$p<0.05$）。
结论 表达 VTCN1 的卵巢癌肿瘤上皮细胞可能在淋巴转移中发挥重要作用，靶向 VTCN1 可能成为开发新疗法的目标。

PO_322

BRCA1/2 罕见序列变异对卵巢癌表型和治疗策略的影响：双杂合性、双突变和逆转突变

丁婷
四川大学华西第二医院

背景 尽管关于 BRCA1/2 单一突变携带者的临床行为已有大量数据，但关于罕见突变的临床数据却十分匮乏，通常仅反映单个病例报告，包括 BRCA1 和 BRCA2 的双杂合突变（DH）、BRCA1 或 BRCA2 的双突变（DM），以及逆转突（RM）。
方法 使用 156 例已发表的测序数据（41 例 DH、2 例 DM、114 例 RM），我们系统评估了罕见变异对卵巢癌表型和治疗策略的影响，
结果 DH 患者的诊断年龄和预后与 BRCA1 突变携带者更为相似，但疾病特征与单一突变患者无显著差异。目前仅有一例 DM 病例报告，该患者携带一个致病性突变（BRCA1，c.2457delC）和两个意义未明的突变（BRCA1，c.5209T>C；BRCA2，c.971G>C）。部分功能性的 BRCA1 等位基因和有害的 BRCA1 截断突变可能存在于同一个人身上。RM 通常呈累积性发生并具有位置依赖性。在铂类或 PARPi 耐药患者中，BRCA1-DBD 和 BRCA2-RAD51-BD 域可耐受较长的氨基酸缺失（分别为 2-390 个氨基酸和 4-468 个氨基酸），在赋予铂或 PARPi 抗性方面是可有可无的。在具有异构化功能域的蛋白质中，BRCA1-RING 和 BRCA2-TR2 域高度保守，是赋予铂和 PARPi 抗性所必需的。与截短突变不同，致病性错义突变的逆转极为罕见，且均为野生型逆转。未观察到致病性剪接突变的逆转事件。ctDNA 测序可有效检测逆转突变，并提供肿瘤多克隆异质性的信息。
结论 DH 的治疗可能更类似于 BRCA1 突变携带者。在临床实践中，应更加关注 BRCA 突变位点，以预测治疗效果并制定个性化治疗方案，从而改善该人群的预后。

PO_323

NF-κB p50/p52 在卵巢癌免疫逃逸中的作用及机制研究

卜岑岚
南通大学附属医院

研究目的 卵巢癌是女性高发恶性肿瘤，死亡率居高不下，亟待深入探究其发病机制以挖掘有效治疗靶点。NF-κB p50/p52 作为关键转录因子，能调控多基因表达，影响细胞存活、增殖及免疫逃逸。本研究旨在剖析 NF-κB p50/p52 在卵巢癌免疫逃逸中的作用及机制。
材料与方法 从 TCGA、GEO 数据库获取卵巢癌患者的转录组数据、临床及免疫细胞浸润信息。利用 DESeq2、limma 等工具筛选差异表达基因，重点聚焦 NF-κB p50/p52 相关及免疫基因，开展 GO、KEGG 富集分析。同时，借助 CIBERSORT、TIMER 算法分析免疫细胞浸润情况及其与 NF-κB p50/p52 表达的相关性。培养多种卵巢癌细胞系及正常卵巢上皮细胞，用 CRISPR-Cas9 技术构建 NF-κB p50/p52 基因敲除或过表达的卵巢癌细胞系。通过 CCK-8、Transwell 实验和流式细胞术分别检测细胞增殖、侵袭迁移能力及细胞周期、凋亡情况。将基因编辑后的癌细胞与免疫细胞共培养，观察免疫杀伤效果并用 ELISA 检测免疫因子水平。
结果 生信分析发现 NF-κB p50/p52 在卵巢癌组织中高表达，与部分免疫细胞浸润相关，免疫相关基因富集于免疫逃逸通路。湿实验中，改变 NF-κB p50/p52 表达后，卵巢癌细胞的增殖、侵袭迁移等生物学行为改变，与免疫细胞共培养时免疫杀伤作用及因子分泌水平变化显著。
结论 NF-κB p50/p52 在卵巢癌免疫逃逸中发挥关键作用，通过调控癌细胞功能和影响免疫细胞实现。该研究为卵巢癌免疫治疗提供了新靶点和理论支持，未来将进一步深入探究其分子机制，助力开发针对性药物。

PO_324

TIPARP mediated alternative splicing of MPRIP influences ovarian cancer progression and its reversal strategy

Yu Chen Xihan Liu Xiaoyu Yin Zihan Li Kun Song*
Shandong university Qilu hospital

Objective This study aims to investigate the key RNA-binding proteins (RBPs) influencing the pathogenesis and progression of ovarian cancer through multi-omics approaches, with a focus on elucidating the role and underlying mechanisms of the critical molecule TIPARP in ovarian cancer advancement, as well as identifying associated aberrant splicing events. By exploring its functional mechanisms, this research seeks to develop therapeutic strategies to reverse ovarian cancer progression, thereby providing novel therapeutic targets and approaches to improve patient prognosis.
Materials and Methods Through integrated analysis of RNA sequencing (RNA-seq) and whole-exome sequencing (WES-seq) data from ovarian cancer patient tissues, combined with our previously established ovarian cancer CRISPR library screening, RNA-binding proteins associated with ovarian cancer pathogenesis were identified. Following preliminary experiments and literature review, TIPARP was selected as a key candidate molecule. Isogenic ovarian cancer cell lines with TIPARP knockdown or overexpression were generated to investigate its functional relevance to malignant biological behaviors through in vitro and in vivo experiments. Further mechanistic exploration was performed via integrated analysis of RNA-seq and RNA immunoprecipitation sequencing (RIP-seq) data, revealing that TIPARP regulates alternative splicing events to promote ovarian cancer progression. Based on these findings, a therapeutic strategy was developed combining antisense oligonucleotides targeting the aberrant splicing events with RBN2397, a selective TIPARP inhibitor, to evaluate potential ovarian cancer reversal approaches.
Results Bioinformatic analysis revealed that high expression of TIPARP in ovarian cancer is associated with poor patient prognosis. Ex vivo experiments demonstrated that knockdown of TIPARP suppresses ovarian cancer cell proliferation and migration while promoting apoptosis, and vice versa. Integrated analysis of RIP-seq and RNA-seq data revealed that TIPARP binds to downstream RNA molecules and mediates aberrant splicing events. Enrichment analysis indicated that the abnormally spliced molecules are primarily associated with cytoskeletal regulation as well as tumor invasion and migration. Notably, TIPARP significantly modulates the alternative splicing of MPRIP, a key molecule in the RHO GTPase pathway, promoting the retention of exon 23 in MPRIP and thereby enhancing tumor cell invasion and migration. Conversely, treatment with a TIPARP inhibitor or antisense oligonucleotide drugs targeting MPRIP exon retention suppresses tumor progression.
Conclusion This study untangles the tumor-promoting role of TIPARP in ovarian cancer, demonstrating that it facilitates ovarian cancer progression by modulating the splicing event of MPRIP, a key gene in the RHO GTPase pathway, thereby increasing exon 23 retention in MPRIP. Both TIPARP and MPRIP represent novel therapeutic targets for ovarian cancer. Furthermore, we innovatively employed a targeted antisense oligonucleotide(ASO) drug against this splicing event and proposed a novel combination therapy strategy. These findings suggest that targeting TIPARP-mediated aberrant splicing may hold potential for cancer therapies.

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畸胎瘤相关自身免疫性脑炎 个案报道

李秀福
秦皇岛市第一医院

病例特点：1、青年女性。2、急性起病，病程 1 天，表现无明显诱因突发意识障碍，表现意识不清，呼之不应，言语混乱，伴有肢体抽搐，小便失禁，无恶心呕吐、高热、咳嗽咳痰、腹泻，8 小时前无其他诱因开始出现高热，体温最高可达 39℃，仍有频繁的肢体抽搐。3、平素健康状况一般，既往体健。否认肝炎、结核或其他传染病史。预防接种史不详。无药物及食物过敏史。否认外伤史。否认手术史。无输血史。4、查体：T：39.4℃，P：160 次/分，R：32 次/分，BP：120/84mmHg，神志昏迷，GCS 评分 E1VTM4 共 6 分，入科后进行气管插管呼吸机辅助呼吸，查体不合作，颈项强直，频繁抽搐，双侧瞳孔 2.5mm，对光反射灵敏，病理征未引出。

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Tumor Extracellular Matrix Remodeling Associated Bevacizumab Resistance in High – Grade Serous Ovarian Cancer

Guodong Zhang Xuyao Xu Haiou Liu*

Tumor Extracellular Matrix Remodeling Associated Bevacizumab Resistance in High - Grade Serous Ovarian Cancer

Background Bevacizumab, a monoclonal antibody targeting vascular endothelial growth factor (VEGF), has been confirmed by clinical trials such as GOG - 218 and ICON - 7 to delay progression - free survival (PFS) in the primary and recurrent treatment of ovarian cancer. However, it is only effective in some patients, and the benefit in overall survival (OS) is limited. At present, there is a lack of clear biomarkers for predicting its efficacy. Extracellular matrix (ECM) remodeling is a core feature of abnormal tumor microenvironment (TME). The dynamic changes of ECM components, such as abnormal deposition of collagen and changes in tissue hardness, can participate in the process of tumor resistance to bevacizumab by remodeling the tumor microenvironment and activating alternative signaling pathways.

Objective This study aims to clarify the correlation between key components of the extracellular matrix (such as type I collagen (COL1), lysyl oxidase (LOX), etc.) and bevacizumab resistance in high - grade serous ovarian cancer (HGSOC), so as to provide a basis for exploring the resistance mechanism and optimizing treatment strategies.

Materials and Methods 115 Patients with recurrent HGSOC who received bevacizumab combined with chemotherapy in Obstetrics and Gynecology Hospital of Fudan University from 2015 to 2022 were included. Immunohistochemical technology (IHC) was used to detect the expression levels of extracellular matrix components COL1 and LOX in tumor tissues. Meanwhile, the GSE140082 database was used to analyze the correlation between the expression of COL1, LOX, tissue stiffness and the prognosis of patients. Kaplan-Meier method was conducted to plot survival curves.

Results In the Fudan cohort, the IHC detection showed that COL1 and LOX had differential expression in the tumor which suggested different state of ECM in HGSOC. High expression of COL1 and LOX is the core marker of fibrosis, high hardness and functional abnormality in tumor ECM. This feature of ECM indicated a poor prognosis of bevacizumab treatment ($P < 0.001$, HR = 2.091). To investigate the association of the feature and tumor prognosis, we investigated it in the GSE140082 and found that the prognosis of patients with high expression of COL1 and LOX was not significantly worse. However, the group with low expression of COL1 and LOX could benefit from OS when using bevacizumab ($P < 0.001$, HR = 3.125), while the group with high expression of COL1 and

LOX had no significant benefit when using bevacizumab (P = 3.03, P for interaction <2.2e-16). COL1 is cross - linked by LOX, leading to an increase in stiffness, so we evaluated the stiffness in GSE140082. The stiffness was not correlated with prognosis in the whole database, while the use of bevacizumab also had a significant benefit in the low - stiffness group (P = 0.001,HR = 3.498), and there was no significant benefit in the high - stiffness group (P = 0.143, P for interaction <2.2e-16).
Conclusion The ECM remodeling characterized by high COL1 and LOX expression and increased tissue hardness is closely related to poor prognosis and bevacizumab resistance in HGSOC. This feature of ECM can be used as a potential biomarker to predict the efficacy of bevacizumab and the important elements of ECM may serve as potential therapeutic targets to reverse ECM remodeling and improve bevacizumab resistance.

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MAP3K14/BCL6/PLAAT4 信号通路参与高级别浆液性卵巢癌进展的机制研究

万安 彭程*
安徽省立医院

高级别浆液性卵巢癌是卵巢癌最常见、致死率最高的亚型，严重威胁着我国女性健康。虽然近年来治疗方案不断优化，患者 5 年生存率依然仅有 31%。因此有必要对 HGSOC 发病机制进行深入研究，阐明肿瘤免疫逃逸机制， 探寻新的治疗靶点，改善患者预后。

BCL6 是生发中心 B 细胞快速增殖和体细胞高频突变过程的守护者；肿瘤细胞利用了 BCL6 这种保护机制，逃避免疫监视，促进了肿瘤的进展。 目前 BCL6 在卵巢癌中的研究仅限于表型，其上下游调控机制尚不明确， 因此本研究以 BCL6 为切入点，探讨了 HGSOC 中 BCL6 的上下游调控机制及其在肿瘤进展中的作用。

（1）为阐明 BCL6 在 HGSOC 进展中的下游调控机制，本研究首先通过 RNA-seq 和 Western blot 实验发现 BCL6 可以激活 PI3K-AKT 信号通路。 然后利用 PI3K-AKT 信号通路抑制剂进行相关实验发现 BCL6 可以通过 PI3K-AKT 信号通路促进 HGSOC 细胞增殖和转移。 最后， 本研究通过联合分析 CUT&Tag 和 RNA-seq 数据探寻 BCL6 调控 PI3K-AKT 信号的途径并利用体内外实验进行了验证， 结果发现 BCL6 通过结合 PLAAT4 的基因启动子区域，抑制 PLAAT4 的转录表达， 导致 PLAAT4 在 HGSOC 中表达降低，从而激活 PI3K-AKT 信号通路， 促进 HGSOC 增殖和转移

（2）为探寻 HGSOC 中调控 BCL6 蛋白表达的上游机制，根据慢性炎症在肿瘤发病中的作用以及 MAP3K14、 BCL6 均可显著下调 PLAAT4 的研究发现，提出 HGSOC 中可能是 MAP3K14 上调了 BCL6 表达。为阐明这一机制，进一步研究发现 MAP3K14 可以促进 USP14 与 BCL6 结合降低 BCL6 泛素化水平， 延长 BCL6 蛋白的半衰期、 增强其稳定性，进而抑制 PLAAT4 的表达。关回复实验证明 MAP3K14 可以通过调控 BCL6 蛋白表达影响 HGSOC 增殖和转移能力综上，本研究证实了 BCL6 在 HGSOC 进展中起重要作用， BCL6 通过抑制 PLAAT4 的转录，从而激活 PI3K-AKT 信号通路，促进 HGSOC 细胞增殖和转移。 本研究同时发现， MAP3K14 可以促进 USP14 与 BCL6 结合， 抑制 BCL6 泛素化降解， 通过调控 BCL6/PLAAT4 轴来驱动 HGSOC 的进展。本研究为 BCL6 作为 HGSOC 潜在的预后生物标志以及可能的治疗靶点提供了理论依据

PO_328

卵巢原发平滑肌肉瘤临床病例报道并文献复习

凌开建 庞晓庆 王昭娣 冯翀 陈莹莹 王晓黎
海南省妇女儿童医学中心

背景 原发性卵巢平滑肌肉瘤（POLMS）是一种非常罕见的恶性肿瘤，占卵巢恶性肿瘤的 0.1%。平均无病生存期为 12.5 个月。其特征是生存率不佳且没有临床诊疗管理参考。将 POLMS 视为 ULMS 还是 EOC？像早期 EOC 一样执行分期淋巴结清扫术，还是像晚期 EOC 和 ULMS 一样去除肿大或可疑的淋巴结？

方法 我们回顾了分析了一例原发性卵巢平滑肌肉瘤的临床诊断过程及病理特征、不同的化疗方法的疗效并进行了该病例的文献复习

结果 50 岁女性，因下腹部胀痛伴尿频尿急入院，可自扪及腹部巨大肿物。化验 CA125：47，其余肿瘤标志物正常。核磁提示包块 20X12CM 大小，腹膜后淋巴结稍大，最大直径 1.6X0.85cm。术中探查见右附件巨大囊实性肿物，与网膜粘连。无腹水。行全子宫双附件及大网膜切除，未进行淋巴结清扫。术后与脂质体阿霉素化疗四个疗程。术后 1 年，右下腹阑尾区出现复发病灶。分叶状，5X5cm 3X3cm。MRI,PET-MRI 考虑复发转移病灶，不排除骨转移。复发后采取吉西他滨和紫杉醇化疗两周期，肿块增大。复发后 3 月行开腹肉瘤切除术，外观的特征是孤立的，小叶，柔软的多个肉瘤组织。肉眼无残留。术后患者行斑马鱼 CDX 模型筛选药物，脂质体阿霉素及异环磷酰胺提示有效。术后 1 月余复诊要求化疗，核磁提示术区片状软组织信号影，考虑复发。脂质体阿霉素及异环磷酰胺，患者拒绝安罗替尼联合。化疗 1 疗程后复查超声提示局部 4X3cm 包块，血流丰富。第二疗程化疗加用安罗替尼口服，复查超声提示包块大小稳定，无血流。

结论 POLM 通常在早期诊断出来，卵巢癌治疗方案效果不佳，多采用子宫肉瘤方案。文献报道提示疾病分期和有丝分裂数量与预后较差有关。手术切除+淋巴结清扫术联合术后化疗与生存率增加有关。CA125 肿瘤标记无特异，在正常范围内或轻度升高。发病机理尚不清楚，因为卵巢不包含平滑肌细胞，尽管已经提出了各种假设来解释其起源。它可能是由卵巢血管或韧带的平滑肌细胞引起的，或者是发育残留遗迹引起的。根据 Polms 的组织发生，可以鉴定出三种组织型：间充质，Teraidoid 和 Müllerian，不同组织型对治疗反应及药物敏感性可能存在不同。该病例多家会诊，免疫组化提示 P53 突变型，ER 及 PR 阳性。复发后按卵巢癌化疗方案无效，肉瘤方案敏感性也不尽如人意。安罗替尼或免疫靶向药物或激素治疗是否有效，有待进一步探讨和观察。

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Interleukin-27 potentiates Treg fragility and efficacy of immune checkpoint blockade in high grade serous ovarian cancer

Haiou Liu*
Gynecology & Obstetrics Hospital, Fudan university

Background The accumulation of immune-suppressive regulatory T cells (Tregs) is a critical determinant of resistance to anti-programmed death-1 (PD-1) therapy in high-grade serous ovarian carcinoma (HGSOC). Interleukins and related cytokines can nurture an environment that is conductive to cancer growth and generate tumor-derived immune responses, which can be explored to improve immunotherapy.

Methods In vitro and in vivo models were used to explore how IL-27 influences tumor growth and antitumor immune response. We also investigated the role of IL-27 in the differentiation of fragile Tregs

and its impact on T-cell function and antitumor immunity. The effects of IL-17 and the response to anti-programmed cell death protein-1 (PD-1) therapy were evaluated in syngeneic mouse models.
Results IL-27 retarded growth of murine ovarian cancers with enhanced accumulation of antitumor polarized tumor associated macrophages (TAMs) as well as the Treg fragility. Treg cells derived from IL-27 treated HGSOC organoids displayed less immunosuppressive function ex vivo. IL-27 and fragile Treg score was significantly associated with higher objective response rate and better overall survival. Importantly, treatment with IL-27 combined with dual immune checkpoint blockade (ICB) of PD-1 and CTLA-4 increased cytotoxicity of CD4+T cells from anti-PD-1 non-responsive HGSOC organoids ex vivo.
Conclusions Our findings offer mechanistic insight into how IL-27 can promote antitumor immunity and present a predictive value of improved response to ICB for patients, providing the rationale for the testing combination of IL-27 with ICB therapy in patients with HGSOC.

PO_330

铁调节在卵巢透明细胞癌中的作用

刘丽莹 王丹波*
辽宁省肿瘤医院

卵巢透明细胞癌（OCCC）与子宫内膜异位症密切相关，具有高铁、活性氧（ROS）和炎症的独特背景，并且对常规化疗不敏感、容易耐药，预后差。探索子宫内膜异位症早期启动癌变机制、卵巢透明细胞癌增殖进展机制、开发创新的靶向精准治疗策略非常重要。铁稳态的维持可以为细胞提供正常生长发育环境，铁可以促进活性氧物质的产生，并且铁超载和氧化损伤可导致细胞铁死亡，虽然多种铁死亡防御系统激活运行以调节该阈值，但如果铁死亡防御通路的过度表达则导致细胞的恶性改变。所以，铁调节紊乱可能是卵巢透明细胞癌发生发展的关键。本文综述了 OCCC 的病理生理学变化、分子生物学特征旨在分子水平上分析癌症驱动基因和途径的异质性；综述在铁代谢、铁死亡及其防御通路、肿瘤微环境中铁调节对 OCCC 的影响，研究探讨相关最新进展，围绕铁调节作为抑制早期癌变和抗肿瘤治疗的目标。

PO_331

卵巢癌类器官的应用：癌症建模与精准医疗

胡海瑶 孙崇恩 李征宇*
四川大学华西第二医院

研究目的 卵巢癌是致死率最高的妇科恶性肿瘤，在全球女性癌症发病率和死亡率中排名第八。大多数卵巢癌患者在晚期才被诊断出来，因此，迫切需要深入研究卵巢肿瘤的形成和演变机制，这要求实时且准确的预测模型以指导个体化和精准治疗策略。类器官是一种三维细胞培养模型，能够在体内体外培养的状态下保持肿瘤的组织学和基因组特征。本研究旨在总结类器官技术在卵巢癌疾病建模和精准医疗中的应用。
方法 以“卵巢癌”、“类器官”和“患者来源类器官”等为关键词，在 Medline、CINAHL 和 Embase 数据库进行搜索，共检索并综述了 75 项研究，对类器官技术与其他模型在不同方面进行了定性分析和比较。
结果 类器官技术能在体外和动物模型中模拟复杂的生物过程。体外培养的患者来源类器官（PDOs）能够再现组织结构和生物特征的主要方面，并在长期培养中保持基因稳定性和原始表型。本研究综述了类器官在卵巢癌模型建立和精准医疗中的应用与优势，特别关注其在再现肿瘤形成、肿瘤演变及肿瘤免疫微环境研究中的作用，以及患者来源类器官在预测患者临床反应、研究肿瘤启动、演变、转移和卵巢癌药物抗性机制方面的重要应用。

结论 近年来，类器官技术已成为肿瘤学研究领域的强大工具。我们总结了小鼠衍生类器官和患者来源类器官在卵巢癌研究中的应用。小鼠输卵管上皮/卵巢表面上皮类器官有助于建立一个同源免疫完整的原发性卵巢癌模型，能够再现不同基因型的肿瘤形成和演变，并作为研究肿瘤免疫微环境及利用免疫联合疗法的体内模型。患者来源类器官最常见的样本来源是手术中获得的肿瘤组织样本或腹水。这些类器官直接来源于卵巢癌患者，具有代表患者特异性的优势，因此在个性化医疗中有着重要的应用，可用于测试新药疗效，为进一步的临床试验提供依据，也是研究卵巢癌肿瘤形成、促进、转移及药物抗性机制的有力工具。类器官平台为癌症研究提供了广阔的前景，从深入研究相关机制到在个性化医疗中的应用，特别是与其他先进技术结合时，具有重要意义。

PO_332

基于外泌体技术的 OCS 评分在 CA125 轻度升高患者附件包块的良恶性辨别能力的双向队列研究：首个国产试剂盒的首批临床验证数据

陆钰坤 朱仲毅 綦小容 刘辉 杨帆 郭娜 廖光东 何跃东*
四川大学华西第二医院

背景 CA125 轻度升高患者的附件包块的良恶性辨别是临床面临的诊断难点。因影像学结合传统肿瘤标志物的诊断效能有限，这类患者常接受过度治疗或被误诊迟治。泌思优是中国首个国产上市的基于外泌体技术的卵巢癌诊断试剂盒，其应用被写入中国妇科肿瘤临床实践指南和卵巢癌早期筛查中国专家共识。通过测定人血清外泌体 CA125、HE4 和 C5a 浓度得到的受试者罹患卵巢癌的分值（OCS）可高效区分附件占位的良恶性。但除上市前研究外，目前 OCS 尚缺乏真实世界数据，尤其是在 CA125 轻度升高患者中的临床验证。

目的 评估 OCS 在 CA125 轻度升高患者附件包块良恶性鉴别中的诊断效能。

方法

1、病人队列构建：

采用前瞻性与回顾性相结合的双向队列研究设计：华西二院是国内首个临床使用 OCS 的医院，本研究筛选 2024 年 7 月至 2025 年 6 月于该院术前接受 OCS 检测的且血清 CA125 处于 35-200 U/ml 的附件包块患者，共 23 例作为回顾性队列。华西二院是国内首个针对 CA125 介于 35-200U/ml 的附件包块患者开展 OCS 上市后行效能检测的临床试验妇科肿瘤中心（国家备案编号：MR-51-25-047830），以入组该研究的样本作为前瞻性队列，现已积累 22 例。

2、数据分析方法

采用 ROC 曲线，以由两名高级职称妇科医生认定的临床诊断及手术病理结果为金标准，分析 OCS 鉴别卵巢癌的诊断效能，主要结局指标包括敏感性、特异性和 ROC 曲线下面积。所有数据均采用 SPSS 23.0 和 GraphPad PRISM version8.0 软件处理。

结果

1. 回顾性队列：纳入 23 例患者，其中良性 14 例，恶性 9 例。OCS 检测的敏感性：77.78%，特异性：85.71%。恶性组 OCS 中位值（0.674）显著高于良性组（0.258）。ROC 分析示，OCS 区分良恶性的 AUC 为 92.5，cutoff 值 0.581。

2. 前瞻性队列：22 例患者中，16 例为良性病变，2 例为交界性肿瘤，4 例为恶性肿瘤。恶性组 OCS 检测值显著升高（例 1=0.754, 例 2=0.804，例 3=0.702）或轻度升高（1 例 0.38），均明显高于本队列中良性组 OCS 中位值（0.088）。OCS 检测的敏感性：75.0%，特异性：94.74%。AUC 为 86.67，cutoff 值 0.362。

3、双向队列：已收集的 45 例患者中，30 例为良性病变，2 例为交界性肿瘤，13 例为恶性肿瘤。OCS 检测的敏感性：76.92%，特异性：93.75%。AUC 为 96.15，cutoff 值 0.362。

结论 本研究在真实世界背景下验证了基于外泌体的 OCS 检测在 CA125 处于 35-200 U/ml 患者的附件包块良恶性诊断中的潜在价值。目前前瞻性队列入组仍在继续，随着后续试验的进行，更大样本量将提供更可靠 OCS 诊断效能评估及亚组分析。此外，OCS 检测的阈值也有望根据本队列研究的结果进行优化。

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NRF2 与 SLC3A2 在卵巢透明细胞癌中的作用机制及其潜在治疗策略

刘丽莹 王丹波*
辽宁省肿瘤医院

卵巢透明细胞癌（OCCC）是一种相对少见且具有高度侵袭性的卵巢癌类型，近年来的研究表明，抗氧化转录因子 NRF2 与氨基酸转运蛋白 SLC3A2 在肿瘤的发生和发展中扮演着重要角色。OCCC 的高侵袭性及其耐药性使得传统治疗手段的效果有限，亟需新的治疗策略。当前的研究显示 NRF2 与 SLC3A2 在 OCCC 细胞的增殖、迁移和侵袭中存在复杂的相互作用，但对其具体的作用机制仍然了解不足。本文综述了 NRF2 和 SLC3A2 在卵巢透明细胞癌中的表达及其功能，分析了它们在细胞行为中的相互影响，并探讨了针对这两者的潜在治疗策略。通过对相关文献的综合分析，旨在为卵巢透明细胞癌的早期诊断和靶向治疗提供新的思路，并推动该领域的进一步研究。

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一例多次复发的卵巢颗粒细胞瘤病例报道并文献复习

段雪睿 张瑜*
中南大学湘雅医院

研究目的 卵巢颗粒细胞瘤是一类来源于性索细胞间质的肿瘤，约占所有卵巢恶性肿瘤的 5%。根据其病理类型可分为幼年型和成年型颗粒细胞瘤两种。卵巢颗粒细胞瘤预后较好，5 年总生存率为 80%-97%，但临床有晚期易复发的特点，并可多次复发。本文通过回顾性总结我院治疗的 1 例多次复发的卵巢颗粒细胞瘤患者的临床资料，分析其临床特点、治疗方式及预后情况，以期为此类患者的治疗及随访提供系统见解。
材料与方法 回顾性分析 1 例多次复发的卵巢颗粒细胞瘤临床资料并复习相关文献。
结果 患者女，70 岁。1987 年行“卵巢肿块切除”，具体病理不详。2015 年行腹膜后巨大肿块切除，术后病理提示“（腹膜后肿块）间叶源性肿瘤，性索间质肿瘤或午菲氏管来源的肿瘤未能排除”。2019 年 7 月出现腹部阵发性胀痛，向腰背部放射，外院腹部 CT 提示“胰腺后方、腹主动脉周围多发肿块，考虑肿瘤性病变，间叶源性肿瘤可能”，我院 CT 提示“约 L1-L2 水平腹膜后肿块，考虑复发可能性大”。行双侧腹膜后肿块切除术，术后病理考虑成人型颗粒细胞瘤。2020 年 4 月感腹胀，外院 B 超提示左附件区 5cm 大小实质性低回声包块，6 月外院复查 B 超示子宫左前方混合回声包块（104*94*69mm），行左侧卵巢切除术，术后病理我院会诊“（左卵巢）成年型颗粒细胞瘤”，2020 年 8 月于我院行补充手术卵巢癌肿瘤细胞减灭术，术中见膀胱窝、盆壁肿瘤结节，术后予“紫杉醇、顺铂”方案腹腔热灌注治疗 3 次，2020 年 9 月至 2021 年 1 月完成 6 次“多西他赛+卡铂”方案静脉化疗。2025 年 1 月复查 CT 提示“左侧腹膜后肿块，考虑转移来源”，我院 CT“左肾前筋膜增厚并肿块形成，复发？”，行腹膜后肿块切除术+主动脉旁淋巴结根治性切除术+左肾切除术，术中见肿瘤 10*7*4cm，部分侵犯左腰大肌及左输尿管，肿瘤以上输尿管及肾盂严重积水。术后病理“（腹膜后肿瘤）成年型颗粒细胞瘤”。术后 3 月复查 CT“腹膜后多发增大淋巴结，双侧肾上腺结节增厚，建议追踪复查”。

结果 由于肿瘤呈惰性生长，晚期卵巢成年型颗粒细胞瘤有着较高的复发率，且有着晚期复发的特点，因此需要坚持长期规律随访。CT 有助于监测肿瘤的复发情况。手术是卵巢颗粒细胞瘤复发乃至多次复发患者最重要的治疗手段，积极手术有助于改善症状和延长生命。

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Visual Analysis of Ovarian Cancer Immunotherapy: A Bibliometric Analysis From 2010 to 2025

Yingjie Zhang Yanyan Chen Chunru Chen Xiaohua Cheng Yanan Peng Juan Wang Fuxia Li Wenting Li*
THE FIRST AFFILIATED HOSPITAL OF SHIHEZI UNIVERSITY

Objective The study aims to conduct a comprehensive bibliometric analysis of global research on ovarian cancer immunotherapy from 2010 to 2025. By systematically evaluating 1,449 publications, we seek to: 1) map the knowledge structure and evolution of this field; 2) identify leading countries, institutions, and journals; 3) analyze research hotspots and emerging trends; 4) assess scientific collaborations; and 5) highlight future research directions. The findings will provide valuable insights for researchers to understand current progress, address existing challenges, and optimize future investigations in ovarian cancer immunotherapy.
Methods This bibliometric analysis utilized publications retrieved from the Web of Science Core Collection (WoSCC) through a targeted search strategy incorporating ovarian cancer and immunotherapy-related terms. Analytical approaches employed multiple specialized tools: Bibliometrix (R package) for statistical evaluation of publication outputs and citation metrics; VOSviewer for network visualization of institutional collaborations and thematic keyword clusters; and CiteSpace for temporal analysis of citation bursts and emerging research fronts. The investigation systematically examined quantitative indicators including annual publication trends, cross-national cooperation patterns, journal performance metrics, and keyword evolution to delineate the field's developmental trajectory.
Results The analysis identified 1,449 publications on ovarian cancer immunotherapy, with China (39.9%) and the U.S. (24.4%) emerging as the leading contributors, while Harvard University stood out as the most productive institution. Frequently occurring keywords such as "tumor microenvironment," "PD-1/PD-L1," and "immune checkpoint inhibitors" highlighted dominant research themes, alongside emerging foci including pyroptosis and combination therapies. Highly cited studies predominantly explored immune checkpoint inhibitors, CAR-T cell therapy, and tumor microenvironment modulation, reflecting key areas of scientific impact and clinical relevance in the field.
Conclusions Ovarian cancer immunotherapy research is rapidly growing, with a shift toward understanding the tumor microenvironment and overcoming resistance. Future directions include biomarker development, combination therapies, and international collaboration to enhance clinical translation. The field holds promise but requires further innovation to address challenges like tumor heterogeneity and immunosuppression.

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雷公藤甲素通过靶向 VDR/Nrf2/SLC7A11 轴诱导细胞铁死亡增强顺铂耐药型卵巢癌敏感性的作用机制

郭峻峰 王陈 丁忱欢 李鹤*
上海交通大学医学院附属仁济医院

目的 顺铂耐药是卵巢癌（OC）预后不良的核心瓶颈，亟需开发安全、高效的联合治疗策略。低剂量的雷公藤甲素（TP）可以有效增强铂耐药细胞对顺铂的敏感性，本研究旨在探讨 TP 与顺铂联合治疗的

协同细胞毒性如何在铂耐药细胞中发挥作用，揭示其增强耐药敏感性作用靶点与潜在机制，为克服卵巢癌铂耐药提供新范式。
方法 首先通过长期暴露于顺铂构建铂耐药卵巢癌细胞系（SKOV3/DDP-S 和 A2780/DDP-A），然后通过体内外实验包括 CCK-8、Edu、Transwell 实验和小鼠皮下瘤模型证实 TP 和顺铂联合治疗作用。随后通过转录组测序、ROS、GSH、MDA 定量以及使用 BODIPY (581/591) C11 和透射电镜确定铁死亡通路的核心作用并用铁死亡抑制剂反向验证。通过免疫共沉淀（Co-IP）、细胞热迁移技术、分子对接、分子动力学模拟、染色质免疫沉淀（ChIP）实验等，进一步探讨 TP 诱导铁死亡的潜在机制。
结果 我们证实了低剂量的 TP 与顺铂联合治疗在体内外可以增强铂耐药卵巢癌细胞敏感性、并显著抑制耐药 OC 细胞系的增殖与转移。联合治疗组 ROS、MDA 及脂质过氧化水平显著增加，GSH 耗竭，线粒体嵴形态塌陷，而铁死亡抑制剂可以阻断 TP 引起的上述表型。通过 Co-IP 等实验我们发现 TP 特异性靶向转录因子维生素 D 受体（VDR），抑制其表达及核转位，进而下调铁死亡核心防御轴 Nrf2/SLC7A11，最终启动铁死亡程序。
结果 TP 通过靶向 VDR/Nrf2/SLC7A11 信号轴诱导铁死亡，不仅抑制卵巢癌的恶性演进，更显著增敏铂耐药细胞对顺铂的应答，提示TP-顺铂联合策略为克服卵巢癌顺铂耐药提供了极具转化潜力的新范式。

PO_337

子宫内膜样腺癌合并卵巢性索间质瘤

谢秋芳
兴文县人民医院

子宫内膜小细胞神经内分泌癌是一种预后较差的少见疾病，常常诊断时为晚期，主要治疗方式是手术+基于铂类的化疗或放疗。由于此类肿瘤极其少见，术前往往难以诊断，诊断过程中，需注意与小细胞非角化鳞状细胞癌、淋巴上皮瘤样癌、腺样基底细胞癌等进行鉴别诊断。治疗方案综合考虑了手术、化疗及放疗等多模态治疗策略，根据病情进展和患者体质个性化调整。临床工作中应注意多总结和随访以提高对该病超声及临床特征的认识

PO_338

内源性催产素缺失抑制卵巢癌进展的机制研究

赵亚敏 常蕾*
郑州大学第一附属医院

研究目的 催产素（Oxytocin, OXT）是一种由下丘脑分泌、垂体释放的神经肽激素，广泛参与情绪、分娩及免疫调节等生理过程。近年来已有许多关于催产素及催产素受体在癌症中的作用，但其在卵巢癌中的功能尚未明确。本研究旨在探讨催产素对卵巢癌生长的影响及其潜在分子机制，为靶向治疗提供理论依据。
材料与方法 将催产素敲除组和野生型对照小鼠分别皮下接种人卵巢癌 A2780 细胞，持续观察肿瘤体积变化并于实验末期取材。利用免疫组化检测肿瘤增殖、凋亡及血管生成相关指标变化，如 Ki67、cleaved caspase-3、CD31 等。采用 qPCR 检测 OXTR、PI3K、Akt 等通路相关基因表达，Western blot 进一步评估 Akt 与 ERK 蛋白的磷酸化水平。
结果 与对照组相比，敲除组小鼠的肿瘤体积和重量显著升高（$P < 0.01$），具有更强的肿瘤生长能力；免疫组化结果显示 Ki67 水平上调，cleaved caspase-3 表达下调，提示肿瘤细胞增殖增强、凋亡减少，同时血管生成也显著增加。在分子水平上，OXTR、PI3K、p-Akt 和 p-ERK 表达上调，说明催产素缺失可能通过激活相关通路促进卵巢癌进展。

结论 内源性催产素缺失可显著促进卵巢癌的生长，初步表明催产素在卵巢癌中具有潜在的抑癌功能。本研究为催产素/催产素受体轴在卵巢癌中的保护性作用提供了实验证据，并为新型激素相关干预策略的开发提供理论基础。

PO_339

The Effect of AdipoRon on Biological Behavior of Ovarian Cancer Cells and Its Mechanism

Hengwei Du Ruiqi Hu Xueyuan Liu Jing Du Cuilan Liu*
Medical Research Center, Binzhou Medical University Hospital

Ovarian cancer is an endocrine tumor with numerous risk factors that significantly increase the likelihood of its development. Adiponectin, a unique adipose-derived factor encoded by the AdipoQ gene located on chromosome 3, is notably downregulated in obesity, making it a key focus in tumor-related metabolic research. AdipoRon, a novel adiponectin receptor agonist,offers advantages such as low molecular weight, high stability and a long half-life. Our study demonstrated that AdipoRon suppresses tumor phenotypes in ovarian cancer cell lines A2780 and SKOV3, including proliferation, clonogenesis, migration, stemmness, apoptosis, and autophagy. These findings were validated through CCK8 assay, colony formation assay, wound healing assay, Transwell experiments, sphere formation assay, and flow cytometry. In addition, we observed that LC3 and p-AMPK protein levels accumulated in A2780 and SKOV3 cell lines treated with AdipoRon, while AMPK and P62 were either reduced or unchanged. RNA-seq results further revealed that AdipoRon-treared ovarian cancer cell lines exhibited significant enrichment in autophagy-related signaling pathways, as indicated by KEEG analysis.In summary, our findings indicate that AdipoRon inhibits the migration and stemness of ovarian cancer cells, and suppresses their growth via inducing apoptosis or autophagy passway. Futher research will focus on elucidating the presise mechanisms of AdipoRon's action on ovarian cancer, with the goal of identifying novel therapeutic target for clinical treatment.

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Chronic stress-related neuroendocrine mediators and ovarian cancer

Hengwei Du[1,2,3] Guoxing Yu[2,4] Ruiqi Hu[1,2,3] Jing Du[1,2] Cuilan Liu[1,2*]
1.Department of Gynecology, Binzhou Medical University Hospital
2.Medical Research Center, Binzhou Medical University Hospital
3.The First School of Clinical Medicine of Binzhou Medical University, Binzhou Medical University Hospital
4.Department of Emergency Medicine, Qingdao Chengyang District People's Hospital

Various sources of stress act on the body, disrupting the stable state of the body's internal or external environment and producing pressure, also known as stress. In contemporary society, individuals are perpetually subjected to stress from emotions, career, family, and work. Although moderate pressure can be beneficial to the human body, long-term and chronic pressure often leads to depression, anxiety, and other mental disorders, making it an irreversible risk factor for diseases, such as cancer. Studies have confirmed that chronic stress promotes the occurrence and development of tumors. The impact of chronic stress-induced neuroendocrine mediators on cancer has garnered increasing attention, prompting ongoing exploration of related therapeutic strategies. In this review, we summarize the roles and mechanisms of chronic stress-related neuroendocrine mediators in the development and

progression of gynecological ovarian cancer and discuss potential therapeutic approaches for chronic stress-mediated ovarian cancer.

PO_341

GALNT10 affects O-glycosylation of IGFBP7 to promote tumor vascular remodeling and metastasis of ovarian cancer

Yanan Zhang Hongyan Guo*
Beking University Third Hospital

Purpose Metastatic ovarian cancer is characterized by significantly elevated glycosylation levels, yet the regulatory mechanism of glycosylation on tumor metastasis remains unclear. This study investigates the mechanistic role of the critical glycosyltransferase in ovarian cancer metastasis and explores its therapeutic targeting.
Materials and Methods Through high-throughput transcriptome sequencing analysis of metastatic and primary lesions of ovarian cancer, we identified the glycosyltransferase GALNT10 as a key regulator associated with tumor metastasis. Using both *in vitro* and *in vivo* ovarian cancer models, we investigated the functional role of GALNT10 through genetic manipulation, including knockdown and overexpression approaches. To elucidate the underlying functional mechanism, we employed O-GalNAc glycoproteomic profiling, co-immunoprecipitation, immunofluorescence staining, and molecular docking analysis. Vascular remodeling was assessed by co-culturing human umbilical vein endothelial cells (HUVECs) with cancer cells, as well as through histological analysis of tumor vasculature. GALNT10 inhibitors were screened and evaluated in metastasis models, both as monotherapy and in combination with anti-PD1 immunotherapy. Modulation of the tumor immune microenvironment (TME) was further analyzed using flow cytometry to characterize immune cell populations.
Results Here, we demonstrated that the glycosyltransferase GALNT10 facilitates the epithelial-mesenchymal transition (EMT) in ovarian cancer cells. Moreover, GALNT10 enhances the extracellular secretion of IGFBP7 through O-GalNAc glycosylation modification. IGFBP7 interacts with the CD93 receptor on endothelial cells, thereby promoting vascular remodeling, including aberrant vascular formation and deficient vascular maturity. These two mechanisms synergistically contribute to ovarian cancer metastasis. Furthermore, the GALNT10 inhibitor Luteolin effectively suppressed metastasis, modulated the immunosuppressive TME via vascular-immune crosstalk, and demonstrated synergistic efficacy with anti-PD1 therapy.
Conclusion: GALNT10 facilitates ovarian cancer metastasis through the induction of tumor cell EMT and tumor vascular dysfunction. Targeting GALNT10 with inhibitors like Luteolin represents a promising novel therapeutic strategy, capable of suppressing metastasis and enhancing response to immunotherapy.

PO_342

外泌体 TSPAN1 通过干性抑制、免疫浸润降低介导卵巢癌顺铂耐药

李思雨 常蕾*
郑州大学第一附属医院

研究目的 卵巢癌是女性生殖系统中致死率最高的恶性肿瘤之一，顺铂化疗虽为其标准治疗方案，但耐药性的发展严重影响疗效和预后。外泌体作为细胞间通讯的重要介质，在癌症耐药中的功能逐渐被揭

示。本研究旨在探讨顺铂耐药卵巢癌细胞来源的外泌体中 TSPAN1 的表达变化及其在干性调控与免疫逃逸中的作用，为顺铂耐药机制提供新的思路和潜在靶点。

材料与方法 我们构建了卵巢癌顺铂耐药细胞株 A2780DDP，并与其亲本细胞 A2780 共同提取外泌体，经 TEM 和 Western blot 验证外泌体纯度后，进行 TMT 定量蛋白质组学分析。差异蛋白经生物信息学分析筛选，发现 TSPAN1 在耐药细胞来源外泌体中显著上调。随后我们结合 TCGA 数据库分析 TSPAN1 在卵巢癌患者组织中的表达特征，并探索其与化疗反应、肿瘤干性以及免疫细胞浸润之间的关系。同时，我们利用 TCGA 数据库探讨 TSPAN1 表达水平与甲基化状态及免疫细胞亚群浸润程度之间的相关性。

结果 蛋白质组学结果显示，A2780DDP 与 A2780 差异蛋白 Top 10 为 TSPAN1、PCBP2、APOBEC3F、TM4SF1、IFITM1、ADGRG6、VAMP5、LHFPL2、MKRN1、CLDN3。A2780DDP 来源外泌体中 TSPAN1 表达显著高于敏感细胞。生信分析表明 TSPAN1 高表达患者对顺铂治疗的反应较差，提示其与耐药相关。相关性分析显示，TSPAN1 表达与干性标志基因表达呈负相关，表明其可能在抑制肿瘤干性表型中发挥作用。TIMER 分析结果指出，TSPAN1 的甲基化水平与多种免疫细胞丰度呈正相关，提示其表达可能受到表观遗传调控，并间接参与免疫微环境塑造。此外，蛋白互作网络提示 TSPAN1 可能通过与硫胺素转运蛋白 hTHTR-1 互作，影响肿瘤细胞能量代谢，为进一步研究其代谢调控作用提供线索。

结果 本研究初步证实了 TSPAN1 在顺铂耐药卵巢癌外泌体中的富集及其多维度的潜在功能。TSPAN1 的高表达与肿瘤干性抑制、免疫细胞浸润减少密切相关，提示其可能通过重塑细胞状态和免疫微环境协同驱动耐药过程。TSPAN1 有望作为预测化疗反应和开发逆转耐药策略的重要靶点。后续我们将进一步开展体内外功能实验，验证其在顺铂耐药中的致病机制。

PO_343

Diaphragm Hernia After Debulking Surgery in Patients with Advanced Ovarian Cancer: Case Series and Literature Review

Lina Shen Shiyang Jiang Rong Jiang Zhuozhen Sun Shuang Ye Libing Xiang*
Fudan University Zhongshan Hospital

Introduction Patients with advanced ovarian cancer frequently require extensive upper abdominal procedures, especially subphrenic peritonectomy. Postoperative diaphragmatic hernia represents an uncommon but clinically significant complication following this procedure.

Method Systemic research of the literature was conducted in PubMed/MEDLINE database. Clinical data on the presentation, diagnostic methods, management, and outcomes of postoperative diaphragmatic hernia following cytoreductive surgery for advanced ovarian carcinoma were analyzed. This study further incorporated cases of postoperative diaphragmatic hernia diagnosed at our center from January 2019 to December 2024.

Results In our center, left-sided diaphragmatic hernia developed in 4/167(2.4%) cases undergoing left diaphragmatic peritonectomy. Of the 55 cases who received diaphragmatic peritonectomy combined with splenectomy, 2 (3.6%) developed diaphragmatic hernia. The central tendon region represents the most common site for diaphragmatic hernia. A total of thirteen cases developing diaphragmatic hernia were analyzed in this study. The median time to diaphragmatic hernia diagnosis was 6 months (range 0.6-36 months) following primary cytoreductive surgery. Most diaphragmatic hernias occurred on the left side (12/13, 92.3%), and right-sided hernias was rare due to the protective shielding effect of the liver. Gastrointestinal symptoms were the most common clinical manifestations (6/13, 46.2%). Most cases were diagnosed by CT and all patients were cured by surgical management.

Conclusion Diaphragmatic hernia is a serious and rare complication following ovarian cancer surgery, primarily occurring after left diaphragmatic peritonectomy. Surgical repair is the primary approach to diaphragmatic hernia management. It is crucial to thoroughly inspect any diaphragmatic defects or weak areas and perform timely repair during diaphragmatic peritonectomy.

PO_344

LINC00240 Knockdown Suppresses the Proliferation, Migration, and Invasion of Ovarian Cancer Cells Through miR-30c-5p/P4HA2 Axis

Haibo Zhang* Yunjie Tian
The Fourth Hospital of Hebei Medical University

Objective This study investigates the expression and functional role of LINC00240, miR-30c-5p, and P4HA2 in ovarian cancer pathogenesis.
Materials and Methods Using datasets GSE66957 and the GEPIA database, we assessed LINC00240 expression levels and employed quantitative real-time polymerase chain reaction (qRT-PCR) to evaluate the expression of LINC00240, miR-30c-5p, and P4HA2 in ovarian cancer samples. Bioinformatics analysis via TargetScan software predicted interactions between these molecules, which were validated through dual-luciferase reporter assays. Functional assays, including clone formation and transwell assays, assessed the impact of LINC00240 and miR-30c-5p on cell proliferation, migration, and invasion.
Results Results indicate that LINC00240 and P4HA2 are significantly overexpressed, while miR-30c-5p is downregulated in ovarian cancer. Furthermore, LINC00240 modulates ovarian cancer malignancy by regulating P4HA2 expression through miR-30c-5p.
Conclusion These findings elucidate the role of the LINC00240/miR-30c-5p/P4HA2 axis in ovarian cancer and suggest new avenues for targeted therapeutic interventions.

PO_345

Association between tumor laterality and prognosis across different ovarian cancer subtypes: Results from SEER database and a Chinese multicenter cohort

Jiaqi Xu[1] Bingxin Zhang[1] Penglin Liu[1] Kezhen Li[2] Xiaodong Cheng[3] Yanci Che[4] Beihua Kong[1] Kun Song[1*]
1.Qilu Hospital of Shandong University
2.Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology
3.Women's Hospital, School of Medicine, Zhejiang University
4.The Affiliated Hospital of Qingdao University

Objective Left-right asymmetry of the body originates from embryonic development, causing huge heterogeneity between bilateral organs, concerning anatomy, blood and lymphatic circulation and genetic elements, which could further lead to different manifestations of cancer incidence, tumor progression and therapeutic response. Recently, tumor laterality has been regarded as a potentially significant prognostic factor due to the left-right heterogeneity of organs. Additionally, the association between tumor laterality and prognosis has been widely explored in various cancers, including breast cancer, renal cancer, lung cancer, colon cancer, and testicular germ cell tumor. To investigate the association between tumor laterality and prognosis across different histological subtypes of ovarian cancer.
Methods In this retrospective cohort study, we collected data on patients with ovarian serous cancer (SC), endometrioid cancer (EC), mucinous cancer (MC), clear cell cancer (CCC), sex cord-stromal tumor (SCST) and malignant germ cell tumor (MGCT) from the Surveillance, Epidemiology, and End Results (SEER) database between 1988 and 2017. Additionally, we collected data on patients with MGCT from a Chinese multi-institutional registry between 2003 and 2020. The primary outcomes were cancer-specific survival (CSS) and disease-free survival (DFS). CSS time was calculated as the time interval (months) from the date of the first diagnosis to the date of death due to

the cancer only. DFS was calculated as the time interval (months) from the date of the first diagnosis to the disease recurrence or death for any reason. Unadjusted CSS and DFS were estimated by Kaplan–Meier analyses, and the differences were explored by the log-rank test. Univariate and multivariate Cox proportional hazards regression models adjusted by previously reported confounding prognostic variables were performed to investigate the association of tumor laterality with CSS and DFS, using the left-sided disease as the reference. The results of Cox regression were represented using hazard ratios (HR) along with 95% confidence intervals (CI). Subgroup analyses were conducted by race (white, black, other) and the administration of chemotherapy (yes, no). Additionally, the sensitivity analyses were performed by excluding the patients with follow-up ≤6 months. A two-sided P value <0.05 was considered statistically significant.
Results In the SEER cohort, a total of 13597 ovarian cancer patients were included, with a median age of 56.0 years. The fully-adjusted multivariate Cox regression model showed the right-sided tumor was significantly associated with a better CSS than the left-sided tumor (hazard ratio [HR] 0.51, 95% confidence interval [CI]: 0.27–0.96; P=0.038) among patients with MGCT, whereas no significant association between tumor laterality and CSS was found among patients with SC, EC, MC, CCC and SCST (all P>0.05). Furthermore, among the 288 patients with MOGCT in the Chinese cohort, the significant association of right-sided disease with better DFS (HR: 0.18, 95% CI: 0.05–0.68; P=0.011) was also observed. Similar results were found in the Kaplan–Meier analyses.
Conclusion Patients with right-sided disease had a better prognosis than those with left-sided disease in MGCT, but not in the other ovarian cancer subtypes. Our findings suggested that the prognostic values of tumor laterality should be highlighted among patients with MGCT and more personalized treatment strategies based on the tumor laterality might be necessary.

PO_346

DDX42 调控卵巢癌 R-loop 代谢的研究

陈小兰 [1,2*]
1.重庆医科大学 2.重庆市妇幼保健院

在卵巢癌中，特别是高级别浆液性卵巢癌（HGSOC），R-loop 水平与卵巢癌的病理机制、 治疗敏感性和耐药性密切相关。卵巢癌细胞中具体的 R-loop 的代谢调控机制及 R-loop 代谢异常与卵巢癌发生发展的关系尚不清楚，本研究前期通过联合分析公共数据库寻找潜在的卵巢癌细胞 R-loop 代谢调控因子，发现 RNA 解旋酶 DDX42 在卵巢癌组织与正常组织中呈现差异表达，进一步细胞和分子生物学实验证明 DDX42 的缺失导致卵巢癌细胞 R-loop 水平显著升高，并引起 R-loop 相关的基因组不稳定性。
研究目的 探究 DDX42 调控卵巢癌细胞 R-loop 水平的深层次机制并揭示 R-loop 代谢异常与卵巢癌发生发展的关系
材料和**方法** 卵巢癌细胞，敲低，过表达，免疫荧光，细胞增殖检测，RNA-seq；
结果 DDX42 的敲低引起卵巢癌细胞中 R-loop 的聚集；DDX42 的缺失导致卵巢癌细胞的基因组不稳定性；DDX42 的缺失导致的基因组不稳定性与 R-loop 相关；DDX42 缺失介导的 R-loop 代谢异常调控卵巢癌的进展。
结论 DDX42 是卵巢癌细胞 R-loop 的代谢调控因子，可以引起 R-loop 相关的基因组不稳定性，进而影响卵巢癌的进展。

PO_347

miRNA 对卵巢癌诊断的 Meta 分析

魏妮[1] 赵荣伟[2*]
1.内蒙古医科大学 2.呼和浩特市妇幼保健院

目的 本研究旨在系统评价血液 microRNA（miRNA）对卵巢癌的诊断价值。
方法 通过检索 PubMed、Embase、Cochrane Library 等数据库，纳入探讨血液 miRNA 用于卵巢癌诊断的相关研究，检索时限为建库至 2024 年 3 月。由 2 名研究者独立筛选文献、提取资料（包括研究基本信息、样本量、miRNA 类型、诊断阈值及四格表数据等）并评价纳入研究的方法学质量（采用 QUADAS-2 工具）。采用 Stata 软件进行 Meta 分析，合并诊断效能指标包括合并灵敏度、特异度、阳性似然比、阴性似然比及综合受试者工作特征曲线（SROC 曲线）下面积（AUC）。
结果 最终纳入 28 项研究，涉及 3246 例卵巢癌患者及 2987 例对照（包括健康人群及良性卵巢疾病患者）。Meta 分析结果显示，血液 miRNA 诊断卵巢癌的合并灵敏度为 0.82（95%CI：0.78-0.85），合并特异度为 0.79（95%CI：0.75-0.82），SROC 曲线 AUC 为 0.83（95%CI：0.84-0.89）。亚组分析显示，多 miRNA 联合检测（AUC=0.90）诊断效能优于单一 miRNA（AUC=0.83），血清来源 miRNA（AUC=0.85）与血浆来源（AUC=0.86）无显著差异。发表偏倚检验（Deek's 漏斗图）提示无显著发表偏倚（P=0.12）。
结论 血液 miRNA 对卵巢癌具有较高的诊断价值，尤其多 miRNA 联合检测有望作为临床潜在的非侵入性诊断生物标志物，但受纳入研究异质性影响，上述结论仍需更多高质量研究验证。

PO_348

桑葚样化生在年轻子宫内膜增生及早期子宫内膜癌患者保留生育功能治疗中的临床意义

赵双双[1,2] 张东璨[1,2] 李甜[1,2] 王博宁[1,2] 王天琪[1,2] 张慧英[1,2] 田文艳[1,2] 王颖梅[1,2*]
1.天津医科大学总医院妇产科，天津，300052
2.天津市女性生殖健康与优生重点实验室，天津，300052

目的 评估桑葚样化生（morular metaplasia，MM）在年轻子宫内膜增生及早期子宫内膜癌（endometrial cancer，EC）患者保留生育功能治疗中的临床意义。
方法 回顾性收集 2018 年 1 月至 2022 年 12 月就诊于天津医科大学总医院妇产科并确诊为子宫内膜增生、小于 40 岁的早期 EC 患者的临床资料，对 MM 组（37 例）和非 MM 组（63 例）患者的临床特征、宫腔镜特征、治疗效果和生育结局进行比较分析。
结果 共纳入 100 例患者。根据病理结果分为 MM 组（共 37 例）和非 MM 组（共 63 例），其中，MM 组根据 MM 出现时间分为三个亚组：①第一次病理出现 MM，治疗后 MM 消失（MM only before treatment，BEF）组 10 例，②孕激素治疗前后持续出现 MM（sustained MM before and during treatment，SUS）组 14 例，③孕激素治疗前未出现 MM，治疗后出现 MM（MM after treatment，AFT）组 13 例。MM 组的 CR 率显著低于非 MM 组（P＝0.004）；MM 组的中位 CR 时间显著高于非 MM 组（P＝0.005）。宫腔镜下病灶砂砾样改变（P<0.001）在 MM 组发生率最高，差异具有统计学意义，MM 是保留生育功能治疗中 CR 的独立危险因素（HR 0.43，95%CI 0.22-0.86，P=0.016），亚组分析显示，SUS（HR 0.31，95%CI 0.11-0.82，P=0.018）和 AFT（HR 0.35，95%CI 0.14-0.84，P=0.020）是保留生育功能治疗中 CR 的独立危险因素。
结论 MM、SUS 及 AFT 是年轻子宫内膜增生及癌变患者保留生育功能治疗的危险因素，宫腔镜下病灶砂砾样改变是 MM 的宫腔镜特征性表现。

PO_349

雄激素受体在子宫内膜癌中的表达及其与临床病理特征之间的相关性

李建 周怀君*
南京鼓楼医院

目的 探讨雄激素受体（androgen receptor，AR）在子宫内膜癌（endometrial cancer，EC）中的表达水平及其与临床病理特征和预后的关系，评估其作为潜在预后指标的价值。
方法 回顾性分析 2018 年 1 月至 2019 年 12 月南京鼓楼医院收治的 68 例 EC 患者的临床资料与病理标本。采用免疫组织化学（IHC）方法检测 AR、p53、Ki-67、Vimentin 及错配修复蛋白（MLH1、MSH2、MSH6、PMS2）的表达水平。比较 AR 表达与患者的病理分期、分化程度、子宫外转移、脉管侵犯及分子标志物表达之间的关系，并进行 Kaplan-Meier 生存分析。结果 68 例 EC 患者中 AR 阳性表达率为 52.94%。AR 阳性表达与较早 FIGO 分期（P＜0.001）、高–中分化（P＜0.05）、无子宫外转移（P＜0.05）及无脉管侵犯（P＜0.001）显著相关。在分子水平上，p53 野生型（P＜0.001）与 Vimentin 阳性（P＜0.05）患者中 AR 阳性表达率均显著升高，而 Ki-67 表达水平及 MMR 缺陷状态与 AR 表达之间差异无统计学意义（P＞0.05）。Kaplan-Meier 生存分析显示，AR 阳性患者 5 年生存率为 94.44%，显著高于 AR 阴性患者的 65.63%（P＜0.01）。
结论 AR 阳性表达与子宫内膜癌的早期分期、高-中分化、无子宫外转移、p53 野生型、Vimentin 阳性表达及较好预后密切相关，提示 AR 可能为 EC 患者的重要预后指标及潜在治疗靶点。

PO_350

Non-invasive assessment of complete regression in endometrial cancer patients undergoing fertility preservation using MRI-based radiomics and immune heterogeneity

Xingchen Li Wang Jianliu*
Peking University People's Hospital

Background Fertility-preserving treatment using progestin is a crucial option for young women with atypical endometrial hyperplasia (AEH) or early-stage endometrial cancer (EC). However, the high rate of progestin resistance limits its effectiveness, necessitating a reliable, non-invasive tool to predict treatment response.
Methods A total of 146 patients with AEH or early EC treated conservatively were included. Radiomic features from pre-treatment contrast-enhanced MRI were extracted and used to build a radiomics signature associated with complete remission (CR) via LASSO regression. A radiomics-clinical nomogram was developed by combining clinical parameters and radiomics scores. Additionally, bulk RNA sequencing and prospective single-cell RNA sequencing (scRNA-seq) on patient samples were performed to validate the molecular landscape and immune cell distribution associated with progestin resistance.
Results The radiomics-clinical nomogram demonstrated strong predictive performance for CR, with ROC validation showing AUC values of 0.963 and 0.986 in the training and validation cohorts, respectively. Patients were stratified into high- and low-risk groups based on the radiomics score. The high-risk group exhibited significantly lower CR rates and was more likely to show progestin resistance. Additionally, single-cell sequencing analysis validated immune cell distribution and content, revealing immune alterations such as reduced $CD8^+$ T cells and M1 macrophages, and increased NK cells and Tregs in the high-risk group. Transcriptomic analysis further highlighted upregulated oxidative

phosphorylation and lipid metabolism pathways, indicating an immunosuppressive and metabolically active tumor microenvironment.
Conclusion This radiomics-based nomogram provides a promising non-invasive approach to predict progestin response and guide individualized fertility-preserving treatment in AEH and early EC.

PO_351

Lipid-driven OLR1/FOXM1/FGF19 axis orchestrates crosstalk in an epithelial-fibroblast positive feedback promoting endometrial cancer progesterone resistance

Xingchen Li Wang Jianliu*
Peking University People's Hospital

Background Progesterone resistance significantly limits the efficacy of fertility-preserving therapy in endometrial cancer (EC). Increasing evidence highlights the role of tumor–stroma interactions and metabolic reprogramming in driving resistance, but the molecular mechanisms remain incompletely understood.
Methods We integrated single-cell RNA sequencing of progesterone-sensitive and -resistant EC samples with bulk RNA-seq and lipid metabolism profiling. Functional validation was performed using knockdown and overexpression method, co-culture assays, ELISA, immunofluorescence, and oxidized low-density lipoprotein (oxLDL) neutralization. The roles of FOXM1 and FGF19 were evaluated using luciferase, qRT-PCR, proliferation，and resistance assays.
Results Our study identifies a positive feedback loop driven by the OLR1/FOXM1/FGF19 axis in progesterone-resistant EC. Tumor cells with high OLR1 expression respond to oxLDL stimulation by the transcription of FOXM1, which directly enhances transcription of FGF19. Secreted FGF19 activates adjacent fibroblasts, as evidenced by increased α-SMA expression and extracellular matrix remodeling. These activated fibroblasts generate reactive oxygen species (ROS), which in turn convert LDL to oxLDL, further amplifying OLR1-FOXM1 signaling in tumor cells. Disrupting this loop through FOXM1 knockdown or by resveratrol significantly reduced tumor cell proliferation and restored progesterone sensitivity.
Conclusion The reciprocal activation between epithelial cells and fibroblasts, mediated by oxLDL and FGF19, promotes a tumor-supportive microenvironment. Targeting oxidative stress or lipid signaling components may provide novel strategies to overcome resistance and improve the outcomes of hormone-based therapies in EC.

PO_352

基于病理形态学特征构建 POLE-Score 预测子宫内膜癌 POLE 突变状态的可行性及效能验证

胡阿锦[1] 刘岩[1,2] 刘从容[1,2*]
1.北京大学第三医院 2.北京大学而医学部病理系

目的 评估基于子宫内膜癌（EC）病理形态学特征预测 POLE 突变分子亚型的可行性。
方法 纳入 2016 年 1 月至 2023 年 9 月于本中心确诊的 1134 例 EC 患者，依据 WHO（2020）女性生殖系统肿瘤分类标准完成分子分型。由两位资深病理医生独立对肿瘤组织切片进行形态学特征评估。通过卡方检验和 Logistic 回归分析对比 POLE 突变型（POLEmut，n=182）与非 POLE 突变型（non-

POLEmut，n=952）EC 的形态差异。基于多因素 Logistic 回归分析中独立预测因子的效应量（OR 值）及临床可操作性分配参数权重。评分系统通过 ROC 曲线确定阈值，并利用混淆矩阵验证其预测性能。
结果 POLEmut 组与 non-POLEmut 组在肿瘤免疫微环境（TIME）、细胞学及组织结构特征上均呈现出显著差异。在 TIME 方面，POLEmut 组以 PTLs 重度浸润为主（54.4% vs. 15.0%），且 TILs 密度显著增加，同样以重度浸润为主（49.5% vs. 11.8%），总差异均具有统计学意义（p<0.001）。在细胞学特征上，POLEmut 组高级别核（40.1% vs. 15.9%）、奇异核（11.5% vs. 3.8%）、胞质嗜酸性（11.5% vs. 5.6%）及胞质透明（18.1% vs. 5.7%）的出现率均显著高于 non-POLEmut 组（p<0.001，p<0.001，p=0.014，p<0.001）。在组织结构方面，POLEmut 组瘤内异质性更为常见（14.3% vs. 4.0%，p<0.001），而其余特征在两组间无显著差异。此外，POLEmut 组肿瘤坏死率高于 non-POLEmut 组（15.9% vs. 9.3%，p=0.009）。评分系统的灵敏度为 82.4%（150/182），特异度为 68.3%（650/952），准确度 70.5%（800/1134），阳性预测值为 33.2%（150/452），阴性预测值为 95.3%（650/682）。AUC 为 0.808（95% CI: 0.773-0.842），临界值≥4 分时，可有效识别 POLEmut 病例，减少漏检（假阴性 n=32）。
结论 基于病理形态特征的 POLE-Score 评分系统，能够以较高灵敏度（82.4%）筛选 POLEmut 病例。该模型适用于资源有限场景下的初筛，可优先选择需分子检测的人群。

PO_353

Circular RNA intraflagellar transport 80 facilitates endometrial cancer progression through modulating miR 545-3p/FAM98A signaling.

Na Wang Yunfeng Guo Tong Tong Xiaomei Fan*
Department of Obstetrics and Gynecology, The Fourth Hospital of Hebei Medical University

Objective Endometrial cancer (ECa) is a common gynecological malignancy. Circular RNAs (circRNAs) have been identified as key regulators of human tumorigenesis and development. Herein, we explored the role and mechanism of circular RNA intraflagellar transport 80 (circ IFT80, also called circ_0067835) in ECa.
Methods Circ-IFT80, microRNA-545-3p (miR-545-3p), and family with sequence similarity 98 member A (FAM98A) were quantified by quantitative real-time polymerase chain reaction or Western blot. The biological characteristics of ECa cells were evaluated via Cell Counting Kit-8, 5-ethynyl-2′-deoxyuridine, transwell, tube formation and flow cytometry assays. Dual-luciferase reporter assay or RNA pull-down assay was employed to verify the binding relationship between miR-545-3p and circ-IFT80 or FAM98A. Xenograft assays were conducted to analyze the effect of circ-IFT80 in vivo.
Results Circ-IFT80 and FAM98A are upregulated in ECa tissues and cells, while miR-554-3p is downregulated. Circ-IFT80 contains a binding site for miR-545-3p, which can act as a sponge for miR-545-3p and inhibit its expression. FAM98A is a direct target of miR-545-3p, which has an inhibitory effect on FAM98A. Therefore, circ-IFT80 can indirectly promote the expression of FAM98A by inhibiting miR-545-3p, thereby promoting the proliferation of endometrial cancer cells，and enhancing the migration and invasion ability of endometrial cancer cells and contributing to tumor spread and metastasis.The growth and metastasis of endometrial cancer depend on angiogenesis, and circ-IFT80 can promote angiogenesis by regulating the miR-545-3p/FAM98A signaling pathway, providing more nutrition and oxygen to tumor cells and supporting tumor growth and development.Circ-IFT80 induces FAM98A expression by inhibiting miR-545-3p, which can affect the expression of cell cycle related proteins and apoptosis related proteins, accelerate the cell cycle process, inhibit cell apoptosis, and promote the survival and proliferation of endometrial cancer cells.
Conclusion: Circ-IFT80 may promote the malignant progression of ECa cells at least in part by modulating miR-545-3p/FAM98A axis, providing a potential therapeutic target for ECa.

PO_354

Weight Management Improves Fertility-sparing Treatment in Overweight Patients with Endometrial Cancer and Intraepithelial Neoplasia

Sijia Liu Li Li Pengfei Wu Lulu Wang Qujia Gama Manrong Wang Jinyu Zhang Liping Jin Weiwei Shan Xuezhen Luo*
Obstetrics and Gynecology Hospital of Fudan University

Background Endometrial cancer (EC) is increasingly diagnosed in younger women. Overweight may impair their fertility-sparing treatment (FST) efficacy. To evaluate the effects of weight management based on intensive lifestyle interventions (ILIs) in overweight patients with EC or its precursor, endometrial intraepithelial neoplasia (EIN).
Methods A total of 141 patients who received treatment at the Obstetrics and Gynecology Hospital of Fudan University from January 2020 to June 2022 were retrospectively analyzed. Body composition, oncological outcomes, and reproductive outcomes were compared between the intervention group (n=97, 69.8%) and the control group (n=44, 31.2%).
Results The intervention group demonstrated significant reductions in weight (−4.2% vs. +1.5%), body fat mass (BFM, −8.3% vs. +3.6%), and visceral fat area (VFA, −6.7% vs. 3.9%) (all $p<0.001$). The complete remission (CR) rate at 24 weeks was greater in the intervention group (44.3% vs. 25.0%, $p=0.026$), with weight management identified as an independent protective factor (HR 2.13, 95% CI 1.08–4.22, $p=0.030$). A parabolic relationship was observed between weight change and CR (p-nonlinear=0.038), peaking at a 3.0% weight reduction. Patients with BFM loss (48.5% vs. 28.8%, $p=0.047$) or VFA loss (51.8% vs. 27.3%, $p=0.010$) presented higher CR rates. Mediation analysis revealed that both VFA and BFM partially mediated the effects of weight management on the CR rate. Reproductive outcomes did not differ between the groups.
Conclusions Weight management, particularly targeting fat reduction, may enhance the therapeutic efficacy of FST for overweight patients with EC or EIN. Our findings advocate the integration of body composition-guided ILIs into standardized FST protocols.

PO_355

DIA-Based Identification of Potential Biomarkers in PCOS at High Risk for Endometrial Cancer

Meng Sun Ruofan Dong*
Affiliated Hospital of Jiangnan University

Objective To apply data-independent acquisition (DIA) analysis to screen for differentially expressed proteins (DEPs) in polycystic ovary syndrome (PCOS) patients at high risk of progressing to endometrial cancer, and to predict potential biomarkers and key molecular mechanisms.
Methods DIA analysis was conducted on a cohort of 15 PCOS patients and 15 endometrial cancer patients to identify DEPs that could serve as potential biomarkers. Subsequently, Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) analyses were employed for functional annotation of the DEPs, while protein-protein interaction (PPI) and gene set enrichment analysis (GSEA) were performed to detect key molecular modules and hub proteins. Candidate biomarkers were validated using enzyme-linked immunosorbent assay (ELISA).
Results A total of 122 DEPs were identified. GO and KEGG analyses revealed that these DEPs were primarily enriched in the PI3K-Akt signaling pathway, pathogenic Escherichia coli infection, dilated cardiomyopathy, and phagosome formation, among others. Furthermore, five core DEPs of interest were confirmed by ELISA: The expression of SRGN was found to be upregulated in blood plasma,

whereas ACTBL2, YWHAZ, and TUBA1B were downregulated, with no significant difference observed in THBS1.
Conclusion The combined DIA-ELISA analysis successfully screened and validated five novel and potentially valuable biomarkers for the early diagnosis of cancer in PCOS patients, as well as identified possible new pathogenic pathways and therapeutic targets.

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子宫内膜癌术前检查筛查研究进展综述

张宇茜[1,2] 樊杨[1,2*] 张巧瑞[1,2] 潘亚旭[1,2] 马昭[1,2]
1.宁夏回族自治区人民医院 2.宁夏医科大学

子宫内膜癌（EC）是女性生殖系统常见的恶性肿瘤之一，其早期诊断与精准术前评估对优化治疗决策和改善预后至关重要。尽管目前尚无理想的 EC 人群筛查方案，但术前个体化评估体系正快速发展，本文旨在通过系统综述 EC 术前检查的关键技术，如：经阴道超声（TVUS）技术是基础筛查手段，但存在较多局限；超声造影（CEUS）技术，特别是联合应用二维（2D-CEUS）与三维（3D-CEUS）技术，通过定量血流动力学分析显著提升了早期病变检出率、肌层浸润深度评估准确性；磁共振成像（MRI）技术在评估深肌层浸润和淋巴结转移方面具有优势；正电子发射断层扫描（PET-CT）对淋巴结转移检测特异性高，适用于高风险患者分期；宫腔镜引导活检（HEB）直视下评估和精准取样的金标准；分子标志物筛查（如 POLE 突变、p53 异常）有助于术前预测肿瘤侵袭性；以及放射组学和人工智能/机器学习等新兴技术在多模态影像分析、风险分层及预后预测中展现出巨大潜力，已逐渐应用于子宫内膜癌的术前影像分期中。通过分析这些技术在子宫内膜癌分期及诊疗中各有特点与价值，进而探究子宫内膜病癌的诊断最佳检查方法，对不同的子宫内膜癌患者给出更加个体化的术前指导。基于现有研究进展，系统梳理了子宫内膜癌术前检查的关键技术、临床应用价值及未来发展方向，并对其进行综述。

PO_357

Low-grade endometrial stromal sarcoma (LGESS): a case of spontaneous pregnancy following fertility-preserving treatment and literature review

Manrong Wang Pengfei Wu
Obstetrics and Gynecology Hospital of Fudan University,

Background Low-grade endometrial stromal sarcoma (LGESS) is a rare malignant tumor of the uterus, characterized by slow growth. Early-stage LGESS harbors a favorable survival but with a high recurrence rate. The primary treatment for this disease is full-staging surgery. In this report, we present a case to explore the potential for fertility-preserving treatment in young women with LGESS.
Case presentation A 29-year-old nulliparous patient diagnosed with stage IA LGESS underwent conservative treatment at the Obstetrics and Gynecology Hospital of Fudan University. After fertility-sparing surgery and three months of hormone treatment, no residual lesions were found. Subsequently, the patient conceived spontaneously and successfully delivered a healthy baby. However, she experienced recurrence eight months after delivery but declined hysterectomy and follow-up care.
Conclusions Currently, there are no established guidelines outlining the strategy for fertility-preserving treatment in LGESS patients. Both previously reported cases and our case suggest that fertility-sparing treatment may be an option for carefully selected patients with LGESS. Further research and larger clinical studies are necessary to explore fertility-preserving treatments for young nulliparous patients with LGESS in order to establish guidelines or consensus.

PO_358

Maching learning Model Combining Radiomics and Clinical Parameters for Predicting Type I and Type II Endometrial Carcinoma

Xiaoli Peng Lou Zhao Maochun Zhang*
Affiliated Hospital of North Sichuan Medical College

Objective To investigate the predictive value of combining ultrasound radiomics with clinical parameters for Type I and Type II endometrial Carcinoma.
Materials and Methods A retrospective analysis was conducted on patients (n=294) diagnosed with endometrial cancer confirmed by pathology in our hospital from 2019 to 2024. Patients were divided into training and testing sets using stratified random sampling at a ratio of 7:3. The region of interest (ROI) was manually delineated along the lesion margin using 3D Slicer (version 5.5.0). A total of 837 radiomic features, including morphology, gray level, texture, and wavelet features, were extracted from each ultrasound image using the radiomics software package. Features with an ICC <0.8 were removed to ensure reproducibility, and data were standardized using Z-score normalization. Important radiomic features were selected through LASSO regression with 5-fold cross-validation. Models were trained using the training set and validated with the test set. Five machine learning algorithms were employed to construct radiomic models, and the optimal model combined with clinical features formed the integrated clinical-radiomic model. Performance was evaluated using metrics such as AUC, accuracy, sensitivity, and specificity. Calibration curves and clinical decision curves were plotted to assess the calibration and clinical utility of the prediction models.
Results After applying inclusion and exclusion criteria, 294 cases were included (233 Type I and 61 Type II), aged between 32-82 years with a mean age of 54.65±8.641 years, all married females. Forty routine clinical and ultrasound semantic features were analyzed per patient. Seven routine clinical-ultrasound features were identified through univariate analysis and LASSO regression: Age, Menstrual disorders, Abdominal pain, Hepatic steatosis, Age at menarche, Gravidity, Maximum tumor diameter. These seven features were used to establish a clinical-only model. Among five machine learning models, the logistic regression (LR) model performed best, achieving AUC (95% CI), sensitivity, specificity, and accuracy of 0.835 (0.768-0.903), 0.738, 0.803, and 0.790 in the training set, and 0.713 (0.579-0.847), 0.631, 0.685, and 0.674 in the test set, respectively. Ten radiomic features were extracted for each patient post feature engineering, and a radiomic score (Radscore) was calculated to establish a radiomic-only model. The LR model outperformed other machine learning models, achieving AUC (95% CI), sensitivity, specificity, and accuracy of 0.834 (0.770-0.897), 0.785, 0.723, and 0.736 in the training set, and 0.811 (0.718-0.902), 0.736, 0.728, and 0.730 in the test set. Finally, an integrated clinical-radiomic model was constructed by combining the seven clinical-ultrasound features with Radscore using the LR algorithm, achieving AUC (95% CI), sensitivity, specificity, and accuracy of 0.916 (0.779-0.927), 0.833, 0.840, and 0.839 in the training set, and 0.803 (0.713-0.901), 0.736, 0.757, and 0.752 in the test set. Calibration curves and clinical decision curves indicated excellent calibration and high clinical utility.
Conclusion Ultrasound radiomics has the potential for predicting Type I and Type II endometrial cancer, contributing to the advancement of intelligent medical diagnosis and treatment.

PO_359

子宫内膜癌患者人表皮生长因子受体 2 表达及其相关因素分析

蒋诗阳 史庭燕*
复旦大学附属中山医院

目的 探讨子宫内膜癌患者的人表皮生长因子受体 2（human epidermal growth factor receptor 2，HER2）表达及相关临床、病理及影像特征。
方法 回顾性收集 2022 年 1 月至 2024 年 12 月复旦大学附属中山医院收治的 214 例初治子宫内膜癌患者，通过免疫组织化学（immunohistochemistry，IHC）评估 HER2 表达，分析其与临床参数、病理特征以及影像学指标的关系。
结果 子宫内膜癌患者 HER2 表达（IHC 评分 1＋/2＋/3＋）率为 37.4%（80/214）。HER2 表达与高龄、国际妇产科联盟（International Federation of Gynecology and Obstetrics，FIGO）分期进展、侵袭性组织学类型、分子分型 P53abn 以及术前盆腔磁共振成像（magnetic resonance imaging，MRI）病灶的低表观弥散系数（apparent diffusion coefficient，ADC）显著相关。多因素分析表明，侵袭性组织学类型（P＝0.002）以及术前盆腔 MRI 病灶的低 ADC 值（P＝0.047）是子宫内膜癌 HER2 表达的独立相关因素。
结论 子宫内膜癌术前 MRI 病灶 ADC 值可作为无创预测 HER2 状态的潜在指标，组织学类型是精准导向 HER2 检测的重要依据。

PO_360

Prognostic and immunotherapeutic potential of five autophagy-related lncRNAs in endometrial cancer

Wen Li Rutie Yin*
Department of Obstetrics and Gynecology, West China University Hospital 2, Sichuan University

Background The incidence and mortality of endometrial cancer (EC) have been steadily increasing. Autophagy and long non-coding RNAs (lncRNAs) are critically involved in EC pathogenesis. In this study, we aim to establish an autophagy-related lncRNA (AR-lncRNA)-based prognostic signature to predict prognosis and immunotherapy response in EC patients.
Methods Autophagy-related genes were obtained from the Human Autophagy Database (HADb), while transcriptomic data (lncRNA and mRNA expression profiles) were extracted from The Cancer Genome Atlas (TCGA) database. Using stringent Pearson correlation criteria ($|R| > 0.3$, $P < 0.001$), we identified autophagy-related lncRNAs (AR-lncRNAs). Then we employed a stepwise analytical approach to identify prognostic AR-lncRNAs and construct a predictive signature, sequentially performing univariate Cox regression, Kaplan-Meier survival analysis, LASSO regression, and multivariate Cox regression, which ultimately stratified EC patients into distinct high-risk and low-risk subgroups. To comprehensively evaluate the prognostic signature, we performed Kaplan-Meier survival analysis, ROC curve assessment, C-index calculation, and clinical nomogram construction. Additionally, we explored its predictive potential through GSEA, mutation profiling, and treatment response analysis (chemotherapy/immunotherapy), while independently validating AR-lncRNA expression patterns using GEPIA, Lnc2Cancer and UALCAN databases.
Results Five prognostic AR-lncRNAs were identified in EC patients, with AC017074.1, ELFN1-AS1 and LINC02585 as risk factors and LINC01871/VIM-AS1 as protective factors. The signature demonstrated strong predictive accuracy (1-/2-/5-year AUC: 0.737/0.735/0.762; C-index=0.727), further validated by a clinical nomogram (C-index=0.802). Mechanistically, dysregulated chemokine

signaling and PTEN/PIK3CA mutations were implicated through GSEA and mutation analyses. Notably, high-risk patients showed enhanced chemotherapy/immunotherapy responses, while independent database validations (GEPIA/Lnc2Cancer/UALCAN) confirmed all five AR-lncRNAs.
Conclusion Our study established a novel five-AR-lncRNA prognostic signature that demonstrated excellent predictive accuracy for clinical outcomes in patients with EC.

PO_361

高危和高-中危早期子宫内膜癌术后单纯近距离照射的复发模式和临床结局

颜子涵[1] 陈媛媛[2] 任康[1] 辛玲霞[1] 盛云龙[1] 王雅琦[1] 侯晓荣[1*] 胡克[1] 张福泉[1]
1.北京协和医院 2.河北省沧州中西医结合医院

目的 对于早期子宫内膜癌高-中危（HIR）组及高危（HR）组患者，盆腔外照射（EBRT）是推荐的辅助放疗方式。PORTEC-2 研究在 HIR 患者中发现，VBT 可获得与 EBRT 相似的生存结局，且不良反应更小。但由于入组患者 90%为 G1-2 级，因此该研究对 HIR 患者的评估并不充分，且 VBT 在 HR 组患者中的应用尚缺乏证据支持。本研究旨在探索接受单纯 VBT 治疗的 HIR 和 HR 患者的复发模式和临床结局。
方法 本研究为单中心回顾性研究，回顾了 1999 年 4 月至 2020 年 11 月在北京协和医院放疗科接受治疗的 1062 例早期子宫内膜癌患者资料，最终纳入 243 例术后仅接受 VBT 治疗的 HIR 和 HR 患者。利用 Kaplan-Meier 法计算生存率，Log-rank 检验比较组间差异，采用 Cox 模型进行单因素和多因素分析探索预后因素。
结果 中位随访 43 个月，全组 5 年 OS、DFS、LRRFS 和 DMFS 分别为 94.8%、82.4%、89.2%和 86.3%。HR 组 OS（87.9% vs 96.9%，P=0.047）和 DMFS（72.6% vs 90.4%，P=0.035）显著低于 HIR 组，但两组 DFS 和 LRRFS 无统计学差异。远处转移为主要复发模式（9.1%），HR 组远处转移率高于 HIR 组（16.9% vs 5.1%）。多因素分析显示，年龄≥60 岁（DFS: HR=2.12，P=0.036）和淋巴结切除≥20 枚（OS: HR=9.96，P=0.005；LRRFS: HR=3.52，P=0.020）是独立预后因素。淋巴结切除≥20 枚组与淋巴结切除＜20 枚组和未行淋巴结切除组相比，其死亡风险（P＜0.001，P=0.010）及局部复发风险（P=0.085，P=0.010）显著降低。
结果 对于经充分淋巴结评估的 HIR 和 HR 早期子宫内膜癌患者，术后单纯 VBT 可能提供较好的局部区域控制及生存结局。远处转移仍为主要的复发模式，且 HR 组风险更高，需探索更为有效的全身治疗策略。

PO_362

Glucagon-Like Peptide 1 Receptor Agonists Use and Mortality in Endometrial Cancer Patients: A Real-World Propensity Score-Matched Cohort Study

Wang Min*
The Ninth Medical Center of Chinese PLA General Hospital

Objective Endometrial cancer is associated with obesity and metabolic disorders. Glucagon-like peptide-1 receptor agonists (GLP-1RA), widely used to manage obesity and diabetes, have indicated potential anti-cancer properties, yet their impact on endometrial cancer mortality remains uncertain. We aimed to evaluate whether GLP-1RA use is associated with reduced all-cause mortality in patients with endometrial cancer.

Methods We conducted a retrospective cohort study, including patients diagnosed with endometrial cancer between January 2014 and December 2023. Patients receiving GLP-1RA were propensity score-matched 1:1 with non-users based on age, race, ethnicity, tumor stage, comorbidities, and medication usage. The primary outcome was all-cause mortality, evaluated via Kaplan-Meier curves and Cox proportional hazards models. Subgroup and sensitivity analyses were performed.
Results From 90,217 identified cases, 3,142 matched pairs were analyzed. After a median follow-up of 777 days (GLP-1RA) and 1,020 days (non-GLP-1RA), GLP-1RA use was associated with significantly lower mortality (4.50% vs. 11.38%; HR=0.46; 95% CI=0.38–0.56; P＜0.0001). This mortality reduction persisted across most high-risk subgroups, including elderly patients and those with overweight or obese, type 2 diabetes, or heart failure. Sensitivity analyses yielded similar results, confirming the robustness of the finding
Conclusion GLP-1RA use was associated with significantly lower all-cause mortality in patients with endometrial cancer, highlighting its potential as an adjunctive treatment strategy. Future prospective trials are warranted to confirm these findings and explore underlying mechanisms and clinical implications.

PO_363

IGF2BP3 通过 MUC1/HIF-1α/ENO1 通路调控子宫内膜癌代谢重编程的作用机制

姜瑞娜 郑剑锋 林雪芬 孙阳*
福建省肿瘤医院妇科

目的 本研究聚焦 RNA 结合蛋白 IGF2BP3，旨在阐明其通过 m6A 甲基化修饰调控 MUC1/HIF-1α/ENO1 信号通路，进而促进 EC 代谢重编程的具体分子机制。
方法 整合 TCGA-UCEC 的批量转录组数据和 GEO 数据库的单细胞 RNA 测序数据，筛选与表观遗传调控相关的差异表达基因（DEGs）。通过非负矩阵分解（NMF）对恶性肿瘤细胞进行亚型分类，分析其表观遗传特征。利用 CellChat 分析细胞间通讯网络，SCENIC 分析转录因子调控网络。通过体外实验（CCK-8、伤口愈合、Transwell 侵袭实验）和体内实验（裸鼠移植瘤模型）验证 IGF2BP3 对 EC 细胞增殖、迁移和侵袭的影响。RNA 免疫沉淀（RIP）和染色质免疫沉淀（ChIP）验证 IGF2BP3 与 MUC1、ENO1 mRNA 的结合及 HIF-1α 对 ENO1 的转录调控。检测葡萄糖消耗和乳酸产生，评估代谢重编程效应。基于表观遗传相关基因构建 6 种基因预后标志物（SDF2L1、ASPM、MUC1、MCM7、PHGDH、MSX1），并通过生存分析和 ROC 曲线验证其预测能力。
结果 鉴定出 20 个与 EC 预后显著相关的表观遗传基因，构建的 Epigenetics ssGSEA 评分可有效区分患者生存期（高风险组预后更差）。敲低 IGF2BP3 显著抑制 EC 细胞增殖、迁移和侵袭，并抑制裸鼠移植瘤生长；过表达则促进这些表型。干扰 IGF2BP3 后的转录组学分析显示，低氧反应效应物（ENO1、SLC7A5、DDIT4）表达下调，染色质免疫沉淀验证了 HIF-1α 与 ENO1 启动子的结合。研究发现 IGF2BP3 通过稳定 MUC1 mRNA 增强 HIF-1α 的表达，而 HIF-1α 转录激活 ENO1，其 mRNA 同时由 IGF2BP3 稳定，形成正反馈回路，驱动糖酵解通量（表现为葡萄糖消耗和乳酸生成的调节）。这种 IGF2BP3/MUC1/HIF-1α/ENO1 轴协调了一个 m6A 依赖的代谢级联反应，将表观遗传-代谢相互作用定位为子宫内膜癌微环境中的潜在治疗靶点。
结论 IGF2BP3 通过 m6A 依赖性机制稳定 MUC1 和 ENO1 mRNA，激活 HIF-1α/ENO1 通路，驱动 EC 的代谢重编程和恶性进展。该通路通过正反馈环路持续激活糖酵解，其构建的预后模型为 EC 的靶向治疗提供了新的表观遗传干预策略和潜在治疗靶点。

PO_364

ETV7 通过乳酸化调控子宫内膜癌代谢及恶性表型的机制研究

张容容 孙阳*
福建省肿瘤医院

目的 子宫内膜癌（Endometrial cancer, EC）是常见的三大妇科恶性肿瘤之一，其发病率逐年上升，对妇女健康构成严重威胁。乳酸化是一种蛋白质翻译后修饰，通过代谢重编程以及对免疫微环境的调控加速了肿瘤的发病、增殖、转移和耐药性的发展。一些研究表明，二甲双胍可降低组蛋白（H3K18）乳酸化，子宫内膜癌组织比正常子宫内膜组织含有更多的乳酸。乳酸化在子宫内膜癌中的生物调控仍未阐明，因此，探索乳酸组蛋白是否在 EC 的发展中发挥作用可能会导致未来治疗的新策略。

方法

1. 用二甲双胍处理 ISK 和 HEC1A 细胞，通过高通量转录组测序，结合生信分析，获取异常表达基因，筛选出 ETV7 基因。

2.检测子宫内膜癌细胞系中 ETV7 的 mRNA 和蛋白表达水平，构建 ETV7 敲低和过表达子宫内膜癌稳转株，通过 CCK8、克隆形成等实验检测 ETV7 对子宫内膜癌细胞的影响，通过 Westernblot 实验检测乳酸化水平的变化。

3.对 ETV7 敲低的细胞和对照组细胞进行转录组测序，通过生信分析获取与乳酸化有关的 ETV7 下游某基因。拟通过 CHIP-qPCR 验证 ETV7 与其下游基因的 DNA 是否互相结合，通过高/低乳酸化条件下培养癌细胞，利用 Westernblot 及免疫组化检测与基因产物乳酸水平的变化及对癌细胞表型的调控，并利用免疫共沉淀、质谱分析等方法确定目标蛋白上特异的乳酸化位点，构建下游基因关键位点突变体，检测乳酸化水平变化。

结果

1. 用二甲双胍处理 ISK 和 HEC1A 细胞之后，通过高通量转录组测序发现 ETV7 的表达均降低。通过 RT-qPCR 检测，在子宫内膜癌 ISK、HEC1a、RL952、KLE 细胞中，ETV7 在 RL952 细胞表达量最高，ISK 细胞仅次于 RL952 细胞。

2.拟通过对子宫内膜癌细胞 ETV7 过表达，与对照组细胞比较，预测 ETV7 过表达组促进子宫内膜癌细胞迁移、侵袭与增殖能力，而敲低 ETV7 的表达得到相反的结果。

结论

1.ETV7 在子宫内膜癌中高表达，其表达与患者的不良预后呈正相关。

2.ETV7 可能通过转录调节某基因通过乳酸化促进子宫内膜癌发生发展。

PO_365

Dual Regulation of MYC and p53 Drives Tumorigenesis in Patient-Derived Endometrial Organoids Model

Shuyan Cai[1] Yuzhen Huang[1] Zhenyi Yin[1] Xiaojun Ren[1] Guo Ji[2] Qian Wang[1] Yali Cheng[1] Bing Zhao[3] Xiaojun Chen[1,2*]
1.Obstetrics and Gynecology Hospital of Fudan University
2.Tenth People's Hospital of Tongji University3.Nanchang University

Endometrial cancer (EC) is one of the most common malignancies in women, yet the link between genetic alterations and tumor initiation remains unclear. This study evaluates the tumorigenic potential of commonly mutated genes in EC—CTNNB1, KRAS, MYC, PIK3CA, MLH1, LKB1, PTEN, and p53—using patient-derived endometrial organoids. Gene-specific effects on proliferation, polarity, and change in stemness were assessed in vitro. Among these, MYC showed the strongest tumorigenic activity but was insufficient alone to sustain in vivo tumor growth. Given its frequent co-mutation in

clinical cases, MYC was combined with p53 deficiency to evaluate synergistic effects in a xenograft model. Transcriptomic and single-cell RNA sequencing revealed activation of multiple tumor-associated pathways, with a marked increase in AGAP1+ cells, implicating them as key drivers of tumorigenesis. The cancerous origin aligned with findings from previous models, supporting the robustness of this system. Overall, MYC activation combined with p53 deficiency establishes a reproducible and continuously growing endometrial carcinoma model, offering new insights into EC pathogenesis.

PO_366

Fertility-Sparing Treatment Outcomes in Endometrial Cancer and Atypical Hyperplasia Patients with Inherited Genetic Syndromes

Xintong Yang[1] Xiaojun Chen[2*]
1.Obstetrics and Gynecology Hospital of Fudan University
2.Shanghai 10th People's Hospital, Tongji University School of Medicine

Introduction While most endometrial cancer (EC) cases are sporadic, approximately 10% are linked to hereditary syndromes, including Lynch syndrome (germline mutations in mismatch repair (MMR) genes: MLH1, MSH2, MSH6 and PMS2), PTEN hamartoma tumor syndrome (PTEN mutations), and hereditary breast/ovarian cancer syndrome (BRCA1/2 mutations). This study aimed to investigate fertility- sparing treatment outcomes in EC and endometrial atypical hyperplasia (EAH) patients with these inherited genetic alterations.

Materials & Methods We conducted a single-center retrospective analysis of 589 EC/EAH patients undergoing fertility-sparing treatment with available germline sequencing or molecular classification data. The distribution of inherited genetic syndromes was described. The outcomes of fertility-sparing treatment in EC and EAH patients with inherited genetic syndromes were analyzed.

Results Among 298 patients (50.6%) underwent germline sequencing, 23 harbored MMR mutations (Lynch syndrome), 7 had PTEN mutations, and 3 carried BRCA1/2 mutations. Molecular classification of 520 patients identified NSMP (89.0%), MMRd (6.7%), POLEmut (2.5%), and p53abn (1.7%). In MMRd patients without germline mutations (n=19), somatic MMR mutations were detected in 11 cases (MMRd-SomMut), while 8 lacked somatic alterations (MMRd-SomWT). Patients with PTEN mutations and NSMP tumors demonstrated comparable CR rates (16-/28-/40-week), 1-year relapse-free survival, and reproductive outcomes. The MMRd-SomWT group exhibited significantly higher diabetes prevalence (62.5% vs. 8.7% in Lynch syndrome and 9.1% in MMRd-SomMut; p=0.006 and p=0.041). Progesterone-treated MMRd-SomWT patients showed non-significant trends toward reduced cumulative 40-week CR rates (66.7% vs. 75.0% in Lynch syndrome and 33.3% in MMRd-SomMut, p≥0.05), prolonged treatment duration (44.00 vs. 32.14/30.43 weeks, p≥0.05), and shorter post-CR relapse intervals (36.53 vs. 59.60/52.42 weeks, p≥0.05). All 3 patients with BRCA1/2 mutations achieved CR within 24.0–50.1 weeks of treatment. One patient experienced recurrence at 85.9 weeks after CR, while the remaining two maintained remission through 30.4–74.4 weeks of follow-up.

Conclusion Germline mutations in MMR genes, PTEN, or BRCA1/2 do not constitute a contraindication for fertility-sparing therapy when managed through comprehensive assessment and enhanced surveillance. MMRd-SomWT tumors, which are potentially linked to MLH1 promoter methylation, may predict unfavorable fertility-sparing treatment outcomes. The integration of molecular profiling with germline sequencing could guide fertility-sparing treatment strategies, improving prognostic accuracy and clinical decision-making.

PO_367

基于类器官高通量药筛的不同分子特征子宫内膜癌精准治疗方案建立

程亚丽[1] 陈晓军[2*]
1.复旦大学附属妇产科医院 2.同济大学附属第十人民医院

研究目的 子宫内膜癌(Endometrial cancer，EC)的发病率及死亡率呈上升趋势，大部分晚期、复发性子宫内膜癌缺乏有效靶向治疗方案。患者来源的肿瘤类器官能够忠实复制原发肿瘤的关键特性，培养周期短、可稳定传代、冻存和复苏，是当前抗肿瘤药物研发和个体化药物筛选的绝佳模型。POLEmut 型 EC 预后最好，MMRd 型免疫检查点抑制剂治疗有效，p53abn 以及 NSMP 缺乏有效的个体化治疗方案。因此，本研究旨在建立不同分子特征内膜癌药敏数据库，指导临床精准治疗的开展。

材料与方法 本研究收集了 2021 年 9 月至 2024 年 1 月在复旦大学附属妇产科医院接受全子宫切除的 EC 的样本共 196 例用于构建类器官，通过 HE/IHC 染色、全外显子测序验证类器官与亲本肿瘤的组织病理学及分子标志物的一致性。基于 NCCN 指南及 FDA 获批药物库，搭建自动化高通量药敏平台。采用活细胞代谢荧光法检测 p53abn/NSMP 亚型类器官的药物敏感性并关联突变特征建立药物数据库。同步筛选 Clinical Trial 中 I 期临床小分子药物，通过靶向药敏试验拓展治疗策略。

结果

1.以成功传代 3 次以上、冻存、复苏后存活并具有类器官典型形态为标准，本研究成功构建了 152 例不同分子分型 EC 患者来源类器官，构建成功率为 77.5%；其中 POLEmut、MMRd、p53abn 及 NSMP 型成功率分别为 81.3%、86.8%、64.5%及 86.5%。HE/IHC 染色证明类器官能维持亲本肿瘤组织的病理形态及分子标志物特征，全外显子测序证明类器官与亲本肿瘤的微卫星状态一致率为 93.33%，分子分型一致率为 86.67%；通路分析证实不同分子分型类器官维持了特征性的信号通路改变。

2.构建包括 30 种抗肿瘤治疗方案的候选药物库，筛选出针对 p53abn 及 NSMP 型 EC 的 10 种临床治疗候选方案。进一步关联突变特征，具有 CTNNB1 突变的类器官对 TC、吉西他滨、拓扑替康、TC+曲妥珠单抗以及雌激素受体降解剂 FWD1802 具有一致的敏感性；KRAS 突变的类器官对 TC、吉西他滨、拓扑替康、紫杉醇+异环磷酰胺、TP+多柔比星以及 TC+曲妥珠单抗方案具有一致的敏感性；

3.选取 Clinical Trial 临床试验中 33 种候选药物进行药敏筛选，具有相同致病性突变的类器官中，双靶点抑制剂 Gedatolisib 显示出高度一致的敏感性；携带 KRAS G12D/G12V/G12C 等不同激活突变的类器官，对新型 pan-KRAS 抑制剂 RMC-6236 均表现出剂量依赖性敏感。

结论 本研究构建了包括不同分子类型的子宫内膜癌类器官生物样本库，证实子宫内膜癌类器官能长期大量培养及冻存复苏，且维持亲本组织的病理及分子特征，是理想的临床前药物筛选模型。通过整合类器官及高通量筛选技术，筛选了临床可行及潜在小分子药物的治疗方案，为 p53abn 和 NSMP 子宫内膜癌提供治疗选择。

PO_368

基于术前多模态盆腔增强 MRI 的子宫内膜癌多任务弱监督预测模型

王倩 陈晓军*
上海市第十人民医院

目的 盆腔增强 （Magnetic resonance imaging , MRI） 是子宫内膜癌（Endometrial cancer, EC）术前评估的首选影像学方法，可评估肌层浸润深度、淋巴结转移及子宫外累及等关键因素，但传统 MRI 诊

断高度依赖医师经验，存在观察者间差异显著等局限性。基于盆腔 MRI 的影像组学和深度学习模型在预测深肌层浸润及淋巴结转移中表现优异，但缺乏区分有无肌层浸润及子宫外累及的有效模型。
方法 本研究为回顾性研究，数据来源于复旦大学附属妇产科医院（中心 1）和上海市第十人民医院（中心 2）。中心 1 纳入术前行盆腔增强 MRI 的 EC 患者 3283 例，排除 358 例后最终纳入 2925 例，按 8:1:1 比例划分为训练集、验证集及内部测试集；中心 2 筛选 273 例 EC 患者，排除 35 例，最终纳入 238 例为外部测试集。纳入标准为接受全子宫+双附件切除术、术后病理确诊为 EC 且临床资料完整者，排除未行淋巴结评估或图像质量不达标的患者。影像数据为 1.5T MRI 的 T1 加权成像、脂肪抑制 T2 加权成像、表观扩散系数图及动态对比增强 - T1 加权序列。数据经标准化处理后，基于 3DViT 框架构建 MRnet-RF 模型：通过独立分支提取各序列深度特征，跨注意力机制融合多模态信息，最终通过三个分类头同步预测肌层浸润深度、淋巴结转移及子宫外累及。模型在 PyTorch 框架下训练，以术后病理为金标准进行评估。
结果 本研究共纳入 3163 例 EC 患者，各子集基线特征一致。在内部测试集中，MRnet-RF 模型区分肌层浸润≥50%、< 50%与无浸润判别的 AUC 分别为 0.842、0.802 和 0.763；预测淋巴结转移的 AUC 为 0.762，子宫外累及的 AUC 为 0.845。与影像科医生相比，模型显著提升淋巴结转移（敏感度 76.2% vs 14.3%）及子宫外累及（敏感度 90.9% vs 36.4%）的检测能力。外部测试集显示，模型区分无肌层浸润、肌层浸润 < 50% 及 50% 的 AUC 值较内部测试集下降 0.035-0.075，预测淋巴结转移和子宫外累及的 AUC 下降 0.051 和 0.120。
结论 基于多模态 MRI 构建的 MRnet-RF 模型在肌层浸润、淋巴结转移及子宫外累及评估中展现良好分类预测能力，其对淋巴结转移和子宫外累及的敏感度优于影像科医生。若与医生的高特异度诊断结合，可显著提升病灶检出效能。

PO_369

A Radiomics-Based Nomogram Model for Non-Invasive Prediction of Cervical Stromal Infiltration in Endometrial Cancer: A Multimodal Approach Integrating Ultrasound Radiomics and Clinical Parameters

Xiaoli Peng Maochun Zhang*
The Affiliated Hospital of North Sichuan Medical College

Objective To develop and validate a nomogram model integrating ultrasound radiomics features and clinical parameters for the non-invasive prediction of cervical stromal infiltration (CSI) in patients with endometrial cancer.
Materials and Methods A total of 294 patients with pathologically confirmed endometrial cancer (2019–2024) were retrospectively enrolled and randomly divided into a training set (n = 206) and a test set (n = 88) using stratified sampling (7:3 ratio). Regions of interest (ROIs) were manually segmented along tumor margins on transvaginal ultrasound images using 3D Slicer (version 5.5.0). A total of 837 radiomic features, including shape, gray-level, texture, and wavelet features, were extracted per case. Features with intraclass correlation coefficient (ICC) < 0.75 were excluded to ensure reproducibility. Data were normalized using Z-score transformation, and feature selection was performed via LASSO regression with 5-fold cross-validation. A radiomic score (RadScore) was calculated for each patient. Clinical parameters were also collected and selected through univariate analysis and LASSO regression. The final nomogram model was constructed by integrating significant clinical predictors and RadScore. Model performance was evaluated using area under the curve (AUC), accuracy, sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and F1 score. Calibration curves and decision curve analysis (DCA) were used to assess model calibration and clinical utility.

Results After exclusion criteria, 294 patients were included (CSI group: n=119; non-CSI group: n=175), aged 32–82 years (mean ± SD: 54.65 ± 8.641), all married women. Three clinical predictors—age, maximum tumor diameter, and years since menopause—were selected for the clinical-only model, which achieved an AUC (95% CI), sensitivity, specificity, accuracy, and F1 score of 0.741 (0.671–0.810), 0.674, 0.688, 0.682, and 0.632, respectively, in the training set, and 0.665 (0.546–0.784), 0.611, 0.679, 0.651, and 0.586 in the test set. Following feature engineering, 25 radiomic features were extracted from each patient's ultrasound images and used to calculate a radiomic score (RadScore). These features formed the basis of the radiomics-only model. The model demonstrated strong performance, with an AUC (95% CI), sensitivity, specificity, accuracy, and F1 score of 0.936 (0.904–0.967), 0.951, 0.737, 0.824, and 0.814, respectively, in the training set, and 0.835 (0.753–0.917), 0.944, 0.679, 0.786, and 0.781 in the test set. Finally, a clinical-radiomic nomogram model was developed by combining the three selected clinical-ultrasound features with RadScore. This integrated model showed excellent predictive performance, with an AUC (95% CI), sensitivity, specificity, accuracy, and F1 score of 0.935 (0.903–0.966), 0.891, 0.860, 0.873, and 0.850, respectively, in the training set, and 0.834 (0.751–0.917), 0.888, 0.716, 0.786, and 0.771 in the test set. Calibration curves indicated good agreement between predicted probabilities and actual outcomes, while decision curve analysis (DCA) demonstrated substantial clinical utility across a range of clinically relevant threshold probabilities.
Conclusion The ultrasound-based radiomics nomogram model demonstrated strong predictive performance for cervical stromal infiltration in endometrial cancer, offering a promising tool for non-invasive preoperative assessment. This approach supports the integration of artificial intelligence into clinical workflows and contributes to the advancement of precision oncology and intelligent diagnostic systems.

PO_370

靶向 SOX2-HK2-乳酸化-IGF2BP2/MCT4 通路调控子宫内膜癌乳酸代谢的治疗新策略

郑剑锋 姜瑞娜 林雪芬 谢宁 汪思萍 张容容 孙阳*
福建省肿瘤医院

研究目的 本研究旨在阐明 SOX2-HK2 轴介导的乳酸代谢通过组蛋白乳酸化修饰激活 IGF2BP2/MCT4 通路在子宫内膜癌进展中的分子机制，重点揭示 SOX2 调控 HK2 表达导致乳酸累积，进而通过 H3K18la 修饰激活 IGF2BP2 并稳定 MCT4 mRNA 的调控网络，为靶向干预该通路提供转化医学依据。
材料与方法 通过整合多维度实验体系：采用 CUT&Tag 测序、ChIP-qPCR 和双荧光素酶报告基因实验解析 SOX2 与 HK2 启动子的结合，结合基因编辑调控 SOX2 及 Seahorse 代谢分析评估糖酵解通量；运用 ChIP-qPCR 验证 H3K18la 对 IGF2BP2 启动子的特异性结合，辅以乳酸干预实验证实表观遗传效应；利用 MeRIP-seq 和 RIP-qPCR 检测 IGF2BP2 对 MCT4 mRNA 3'UTR m6A 位点的识别，并通过 RNA 稳定性分析阐明 m6A 依赖性调控机制。
结果 实验证实 SOX2 直接激活 HK2 启动子显著提升乳酸产量，累积乳酸诱导 H3K18la 修饰水平升高（组蛋白乳酸化组学验证），进而结合 IGF2BP2 启动子促进其转录激活；升高的 IGF2BP2 蛋白特异性识别 MCT4 mRNA 的 m6A 位点，通过延长 mRNA 半衰期增强 MCT4 介导的乳酸外排能力，形成"SOX2-HK2-乳酸化-IGF2BP2-MCT4"正反馈循环。关键治疗证据显示，SOX2 抑制可阻断通路活性，乳酸干预可逆转恶性表型。
结论 本研究首次揭示该代谢-表观遗传级联通路通过放大子宫内膜癌细胞乳酸代谢重编程驱动肿瘤进展，提出三重靶向治疗新策略：源头抑制 SOX2-HK2 轴阻断乳酸生成、干扰 H3K18la 修饰破解表观遗传开关、靶向 IGF2BP2-m6A-MCT4 模块抑制乳酸外排，为子宫内膜癌精准治疗提供创新理论思路。

PO_371

基于临床病理因素的 II 型子宫内膜癌预后列线图模型建立与效能评估

谢宁 孙阳*
福建省肿瘤医院妇科

背景 Ⅱ型子宫内膜癌 (Endometrial carcinoma, EC) 主要包括子宫内膜浆液性癌、透明细胞癌和癌肉瘤，其发病率占所有 EC 的 1%-10%。相较于 I 型 EC，II 型 EC 恶性程度高，侵袭性强、复发率高和预后差。本研究旨在寻找影响 II 型 EC 预后的临床病理危险因素并构建预后列线图模型，协助妇科肿瘤医生准确预测 II 型 EC 患者的总生存期(Overall survival, O S)，以进一步指导临床精准诊疗。
方法 本研究收集福建省肿瘤医院 2014 年 1 月至 2024 年 5 月经病理确诊的 II 型 EC 患者的人口学特征和临床病理资料，按 7:3 的比例随机分为训练组和验证组。采用 Cox 回归分析探究影响 II 型 EC 的独立预后危险因素，并建立预后列线图。利用时间依赖性受试者工作特征(tim eROC)曲线下面积(AUC)、校准曲线和决策曲线(DCA)评估列线图的预测能力和临床获益。
结果 研究共纳入 153 例 II 型 EC 患者(训练组:n=107，内部验证组:n=46)，全队列中位随访时间 60 个月，5 年 OS 为 65.4%，复发率 34.0%。多因素 Cox 回归分析示 FIGO 分期、淋巴结转移、预后营养指数(Prognostic Nutritional In dex,PNI)和 CA125 是 II 型 EC 患者结果预后的独立危险因素(P<0.05)。列线图在训练组 1 年、3 年和 5 年 OS 的 AUC 为 0.743、0.753 和 0.705，在内部验证组 1 年、3 年和 5 年 OS 的 AUC 为 0.738、0.703 和 0.738。训练组、内部验证组和外部验证组的校准曲线和 DCA 曲线均证实了预后列线图良好的预测能力和临床获益。
结果 本研究明确了 FIGO 分期、淋巴结转移、PNI 和 CA125 是 II 型 EC 患者预后的独立危险因素，以此构建一个全新的预后列线图，内部讨论验证证实该预后列线图具备良好的临床效能。基于本研究的预后列线图，妇科肿瘤医生可以更准确的预测 II 型 EC 患者的预后，为个体化治疗提供科学的诊疗依据。

PO_372

HMGA2 转录激活 ANT2 调控子宫内膜癌代谢及其恶性表型的机制研究

汪思萍 孙阳* 郑剑锋 林雪芬
福建省肿瘤医院

背景 子宫内膜癌 (Endometrial carcinoma, EC) 是一种非常普遍的妇科恶性肿瘤，尤其是在发达国家。近年来，由于生活方式的改变和人口老龄化，EC 的发病率不断增加。代谢异常是 EC 的重要危险因素，EC 中异常的线粒体自噬活性与肿瘤细胞代谢、增殖、存活和耐药性密切相关。因此，全面了解线粒体自噬在 EC 中的作用可能为开发新的治疗策略提供重要见解。
方法 首先检测 EC 细胞系中 HMGA2 的表达水平，经慢病毒转染建立稳定敲低及过表达 HMGA2 的 EC 细胞株， 体外细胞实验验证 HMGA2 对 EC 细胞增殖、迁移、侵袭、凋亡及线粒体自噬的影响，裸鼠皮下注射敲低 HMGA2 稳转株及对照株，观察小鼠成瘤情况。对敲低 HMGA2 稳转株和对照株进行高通量转录组测序，通过综合分析测序数据筛选出下游基因 ANT2 进行研究。通过染色质免疫共沉淀技术确认 HMGA2 蛋白和 ANT2 基因之间的相互作用。回复实验证明 HMGA2 通过 ANT2 影响子宫内膜癌细胞增殖、 迁移、侵袭、凋亡及线粒体自噬。
结果 我们发现，在 EC 不同的 GEO 数据集中，HMGA2 在 EC 组织中的表达高于正常组织，PCR 和 WB 结果提示 HMGA2 在 EC 细胞中表达显著升高，细胞实验表明下调 HMGA2 表达可抑制 EC 细胞的

增殖、迁移和侵袭，同时促进 EC 细胞凋亡，裸鼠成瘤实验中，下调 HMGA2 表达可抑制肿瘤的生长。通过分析高通量转录组学测序结果，我们发现 ANT2 可能是 HMGA2 潜在的下游分子，经过验证，发现 ANT2 和 HMGA2 表达水平正相关，CHIP-IP 实验表明转录因子 HMGA2 可结合于 ANT2 的启动子区，激活 ANT2 的转录表达，而敲低 HMGA2 以及加入自噬抑制剂 CQ 可降低 ANT2 mRNA 及蛋白水平，CHIP-qPCR 证明 HMGA2 可与 ANT2 启动子区域进行富集。ANT2 是一种参与 PINK1-PRKN 依赖性线粒体自噬的关键线粒体内膜蛋白，通过诱导 MMP 去极化，触发线粒体自噬。通过 WB 及回复实验验证，敲低 HMGA2 以后，线粒体自噬相关蛋白 PINK1 和 PRKN 表达下调，通过线粒体膜电位、脂质过氧化物检测等实验表明， HMGA2 的敲低抑制了 ANT2 转录和 PINK1/Parkin 介导的线粒体自噬。**讨论** 本研究结果提示 HMGA2 可以通过转录调控 ANT2 的表达水平增强线粒体自噬，从而促进 EC 恶性表型，促进肿瘤进展。

PO_373

CDCA3 调控 USP18 去泛素化修饰 MYC 稳定蛋白水平促进子宫内膜癌增殖及 DNA 损伤修复的机制研究

于佳源
烟台毓璜顶医院

子宫内膜癌（EC）是女性生殖系统高发恶性肿瘤之一，晚期及复发患者面临严重临床挑战，亟需解析其分子机制并开发新型诊疗靶点。细胞分裂周期相关蛋白 3（CDCA3）作为关键细胞周期调控因子，其异常高表达与多种癌症的恶性表型及不良预后密切相关，但其在 EC 中的作用机制尚不明确。本研究通过生物信息学分析发现 CDCA3 在 EC 中显著高表达且与不良预后相关，并经临床样本的 WB 实验、qRT-PCR 实验及 IHC 实验证实其表达高于正常内膜组织。通过构建 CDCA3 敲低及过表达的子宫内膜癌细胞模型，结合细胞功能学实验、γ-H2AX 检测及裸鼠成瘤实验，证实 CDCA3 通过促进细胞周期异常活化、增强 DNA 损伤修复能力及抑制凋亡，显著增强 EC 细胞恶性表型。机制层面，通过 GSEA 富集分析发现 CDCA3 相关分子在 MYC 相关通路显著富集，对两分子进行分子对接及 Co-IP 实验发现二者具有相互作用。在过表达 MYC 的 EC 细胞模型中，进行细胞功能学实验发现 MYC 本身可增强 EC 细胞恶性表型。进一步通过蛋白质及转录水平检测发现，CDCA3 影响 MYC 蛋白而非 RNA 表达水平，提示其可能调控 MYC 蛋白降解。通过 CHX 处理后，外源性添加泛素蛋白酶体抑制剂 MG132 及自噬溶酶体抑制剂 CQ 后检测蛋白表达水平发现，CDCA3 主要通过调控 MYC 泛素修饰从而影响蛋白水平表达。为进一步阐明具体机制，本研究筛选出与 CDCA3 显著相关的 MYC 泛素化酶（MID2、FBXL7、FBXO31）及去泛素化酶 USP18。WB 实验、qRT-PCR 实验及 Co-IP 实验等证明 USP18 与 CDCA3 及 MYC 存在强相关性。最终通过回复实验等得到**结果** CDCA3 通过调控去泛素化酶 USP18 减少 MYC 的泛素化降解。基于此，本研究通过生信分析及体内外实验验证：CDCA3 通过招募 USP18 介导 MYC 去泛素化修饰，稳定 MYC 的蛋白水平，进而促进细胞周期异常活化及 DNA 损伤修复，驱动 EC 恶性进展。本研究揭示 CDCA3 通过泛素化调控网络驱动 EC 进展的机制，为开发靶向 CDCA3 的精准治疗策略提供理论依据，具有重要临床转化价值。

PO_374

Collision tumor of endometrial hepatoid adenocarcinoma and endometrial mesenchymal sarcoma: A rare case and literature review

Zhang Jie Dengfeng Wang Guonan Zhang*
Sichuan Clinical Research Center for Cancer, Sichuan Cancer Hospital & Institute, Sichuan Cancer Center, Affiliated Cancer Hospital of University of Electronic Science and Technology of China

Background Endometrial hepatoid adenocarcinoma (HAC) is an exceedingly rare tumor. Histologically, the tumor is predominantly characterized by HAC with additional components, as documented in previous reports. The coexistence of endometrial HAC and endometrial mesenchymal sarcoma in collision tumors is exceptionally uncommon. To our knowledge, this case represents only the second documented instance of such a collision tumor.
Case presentation A 69-year-old postmenopausal woman presented with abnormal uterine bleeding, and her serum alpha‐fetoprotein (AFP) concentration was 8926 ng/mL. The initial biopsy revealed poorly differentiated adenocarcinoma. Subsequent postoperative histopathological evaluation confirmed the coexistence of endometrial HAC and endometrial mesenchymal sarcoma. Notably, according to imaging, gross examination, and microscopy, the two tumor components were separated by distinct contact areas without evidence of migration or intermingling. The patient developed pulmonary metastases within 40 days of surgery and eventually died of the disease.
Conclusion Endometrial hepatoid adenocarcinoma is an exceptionally rare tumor that frequently coexists with other pathological components. Preoperative diagnosis remains challenging, and definitive characterization relies predominantly on postoperative evaluation, particularly immunohistochemical analysis. These tumors exhibit marked heterogeneity and aggressiveness, with a propensity for lung metastasis and an overall poor prognosis. Due to the limited number of reported cases, there is no established consensus regarding the optimal treatment. Some case reports suggest that the development of hepatoid adenocarcinoma of the uterus may have some correlation with endometriosis. 18F-Fluorodeoxyglucose (18F-FDG) positron emission tomography/computed tomography (PET/CT) enables the comprehensive pretreatment assessment of disease extent and the early detection of metastases. Surgical intervention remains the mainstay of treatment, but accurate evaluation of pathological characteristics is critical for selecting the optimal chemotherapy regimen. In this context, the CAP regimen—comprising cyclophosphamide, adriamycin, and cisplatin—has demonstrated superior overall efficacy. Moreover, these tumors seem to be relatively resistant to radiation therapy.

PO_375

衰老微环境通过 CAFs 介导的 H3K18la 修饰激活 YY1-PHGDH 通路促进子宫内膜癌顺铂耐药的机制研究

张汤安苏 汪宏波*
华中科技大学同济医学院附属协和医院

研究目的 细胞衰老、癌症相关成纤维细胞（CAFs）及组蛋白乳酸化修饰在肿瘤化疗耐药中发挥重要作用，但其在子宫内膜癌（UCEC）顺铂（CDDP）耐药中的具体调控机制尚不明确。本研究评估衰老内膜癌细胞通过激活 CAFs 介导组蛋白乳酸化修饰在 UCEC 顺铂（CDDP）耐药中的具体分子机制。
材料与方法 构建 CDDP 耐药的衰老 UCEC 细胞模型，与 CAFs 共同培养。细胞活力和死亡通过细胞计数试剂盒-8（CCK-8）和碘化丙啶（PI）染色进行评估。通过 ELISA 检测衰老分泌相关表型（SASP）

的表达。组蛋白乳酸化通过乳酸的产生、pan-Kla 和 H3K18la 的表达来确定。通过染色质免疫沉淀（ChIP）和双荧光素酶报告系统确定分子机制。通过实时定量 PCR（qRT-qPCR）、Western Blot 印迹、免疫荧光和免疫组化染色检测分子和蛋白质的表达。建立裸鼠移植瘤模型，评估 CDDP 体内敏感性。

结果 CDDP 耐药的 UCEC 组织中衰老相关基因和信号通路显著激活，其中衰老 UCEC 细胞通过分泌 SASP 因子 TGF-β 和 IL-1β 有效激活 CAFs。机制研究发现，活化的 CAFs 通过 H3K18la 组蛋白乳酸化修饰促进 UCEC 细胞获得 CDDP 耐药性。进一步分析表明，衰老 UCEC 细胞中转录因子 YY1 表达水平显著上调，且其表达与组蛋白乳酸化修饰水平呈正相关。深入机制探讨揭示，CAFs 通过增强组蛋白乳酸化修饰上调 YY1 表达，而 YY1 可直接结合促癌基因 3-磷酸甘油酸脱氢酶（PHGDH）的启动子区域并促进其表达，从而介导 CDDP 耐药。功能回补实验表明，使用衰老细胞清除剂 ABT263 靶向清除衰老 UCEC 细胞可显著恢复 CDDP 敏感性；同时，采用葡萄糖类似物 2-脱氧葡萄糖（2-DG）和乳酸脱氢酶抑制剂草铵膦（oxamate）处理可有效降低组蛋白乳酸化水平，抑制 YY1 过表达，并显著增强 UCEC 细胞对 CDDP 的化疗敏感性。

结论 本研究系统阐明了衰老 UCEC 细胞通过 SASP-CAFs-H3K18la-YY1-PHGDH 信号轴介导 CDDP 耐药的新机制，并为临床克服 UCEC 化疗耐药提供了潜在的治疗策略：一方面可通过清除衰老细胞来阻断耐药起始环节，另一方面可通过靶向干预该信号通路中的关键节点来逆转耐药表型。这些发现为开发新型联合治疗方案以提高 UCEC 化疗敏感性提供了重要的理论依据和实验基础。

PO_376

RAB26 Promotes Endometrial Cancer Cell Progression Through the Sonic Hedgehog Signaling Pathway: Mechanisms and Therapeutic Implications

Chao Gao Xiaoyu Zhang Fengxia Xue Yingmei Wang*
Tianjin Medical University General Hospital

Objective Endometrial cancer (EC) is the most common gynecological malignant tumors since its etiology is still not clear. RAB26 has been shown to influence cell progression in cancers. Our previous study showed RAB26 was related to the cell cycle-associated protein IK, which has been shown to contribute to carcinogenesis. Herein, this study aimed to explore the clinical significance and biological function of RAB26 in EC, and to investigate the potential mechanism underlying its role in EC progression, with a focus on whether RAB26 influences EC cell behavior through the Sonic Hedgehog (Shh) signaling pathway.

Methods We first assessed RAB26 expression in 615 endometrial tissue samples using immunohistochemical staining and analyzed its correlation with clinical parameters. Then, we conducted in vitro experiments by attenuating or overexpressing RAB26 in EC cell lines (HEC-1B and HEC-1A) and evaluated the effects on cell proliferation, apoptosis, cell cycle distribution, migration, and invasion. RNA sequencing was performed to identify differentially expressed genes and signaling pathways affected by RAB26 attenuation. Western blotting was used to detect the expression levels of proteins related to the Shh pathway, cell cycle, and epithelial-mesenchymal transition (EMT). Additionally, we employed an in vivo tumor xenograft model in nude mice to further confirm the antitumor effects of RAB26 attenuation and the impact of Shh pathway activation on tumor growth.

Results RAB26 expression was significantly higher in EC tissues compared to normal endometrium and was positively correlated with tumor histological grade, FIGO stage, and tumor size. RAB26 attenuation suppressed EC cell proliferation, metastasis, and EMT, promoted apoptosis, and induced G0/G1 cell cycle arrest. RNA sequencing revealed enrichment of the Hedgehog signaling pathway in RAB26-attenuated EC cells. The Shh pathway agonist purmorphamine reversed the inhibitory effects of RAB26 attenuation on EC cell progression both in vitro and in vivo. In the tumor xenograft model,

RAB26 attenuation significantly inhibited tumor growth, and this effect was attenuated by purmorphamine treatment.
Conclusions Our findings demonstrate that RAB26 functions as an oncogenic protein in EC by promoting tumor progression through the Shh signaling pathway. RAB26 attenuation exerts antitumor effects by reducing the expression of Shh pathway-related proteins, and these effects can be reversed by activating the Shh pathway. These results suggest that targeting the RAB26-Shh axis may represent a promising therapeutic strategy for EC patients.

PO_377

CTMP 在子宫内膜癌孕激素抵抗中的作用机制研究

于新雨 尚凯悦 邢红鑫 张辉*
山东第一医科大学附属省立医院

目的 子宫内膜癌（Endometrial cancer, EC）患者近年来呈年轻化趋势。对于要求保留生育功能的早期 EC 患者，孕激素保守治疗为主要治疗方法。然而，在孕激素治疗过程中出现的耐药现象导致了孕激素疗效的难以预测性，孕激素抵抗是年轻 EC 患者保守治疗失败的关键因素，目前尚无有效的方法预测和逆转孕激素抵抗。已知 CTMP 参与 EC 的发生发展，但其机制尚未明确。
方法 通过 Western Blot 实验检测 CTMP 在不同 EC 细胞中的表达以及孕激素处理后不同 EC 细胞系中 CTMP 的表达水平，并探讨 CTMP 与孕激素敏感性的关系。通过 Western Blot、CCK-8 和 EDU 掺入法等实验检测 CTMP 敲低对 EC 细胞孕激素敏感性的影响，以及探究 CTMP 在 EC 孕激素抵抗中可能的作用机制。
结果 （1）孕激素对 Ishikawa 细胞和 KLE 细胞生长有抑制作用并具有浓度依赖性。孕激素对 Ishikawa 细胞系的抑制作用更加明显（IC 50 分别为 24.31μM 和 64.28μM）。经相同浓度孕激素刺激 48 小时后，Ishikawa 细胞中 CTMP 表达明显下降（P <0.05），KLE 细胞中 CTMP 表达明显升高（P <0.05）；（2）结果表明，在孕激素作用下，敲低 CTMP 能使 EC 细胞系的细胞存活率及增殖率降低（P <0.0001），凋亡蛋白表达明显升高（P <0.05），可增加 EC 细胞对孕激素的敏感性；（3）敲低 CTMP 可抑制 EC 细胞中 PI3K/AKT 信号通路活性，增强 EC 细胞对醋酸甲羟孕酮（MPA）的敏感性，阻断 PI3K/AKT 信号通路增加 EC 细胞对孕激素的敏感性。
结论 CTMP 可能与 EC 孕激素抵抗有关。敲低 CTMP 可增加 EC 细胞对孕激素的敏感性。CTMP 可能通过激活 PI3K/AKT 信号通路诱导 EC 发生孕激素抵抗。

PO_378

SMARCA4 缺陷型子宫肿瘤报告

刘群
首都医科大学附属北京妇产医院

SMARCA4 缺陷型肿瘤罕见，SMARCA4 基因变异和编码蛋白 BRG1 的表达缺失是其特征性的驱动性遗传事件。由 SMARCA4 失活性突变驱动的肿瘤可发生在全身多个脏器，发生于女性生殖系统的瘤种包括去/未分化子宫内膜癌（UDEC）、高钙血症型卵巢小细胞癌（SCCOHT）和 SMARCA4 缺陷型子宫肉瘤（SDUS）。尤其 SDUS 极为罕见，并具有高度侵袭性，患者预后极差。本文报道了 1 例 SMARC4 缺陷型 UDEC 和 1 例 SDUS。

病例 1 因“月经紊乱 2 年”就诊，MRI 提示宫颈管（3cm）及宫腔（8cm）内占位，DWI 高信号考虑恶性肿瘤侵犯肌层可能。根据子宫全切术后病理诊断为 SMARCA4 缺陷型子宫肉瘤 IB 期，术后补充吉西他滨+多西他赛化疗 3 程、帕博利珠单免疫治疗。目前术后 19 月，免疫维持治疗中，无复发迹象。

病例 2 因“下腹胀痛”就诊，既往：9 年前因宫颈低分化鳞癌 IIB 期行全程放疗+5 程增敏化疗，未规律复查。超声及 MRI 均提示宫腔内占位 8cm，不除外恶性可能，子宫切除病理为宫颈及子宫 SMARCA4 缺失性去分化癌（未分化癌与中分化子宫内膜样癌混合）。诊断为子宫 SMARCA4 缺失性去分化癌II期。术后补充白蛋白紫杉醇+卡铂化疗+帕博利珠单免疫治疗。术后4月再次出现阴道出血，查体见阴道残端见菜花样肿物，直径约 1cm，活检病理提示肿瘤复发。盆腔 MRI：阴道残端左前壁实性结节，大小 2.1*1.2*1.6cm，累及膀胱壁不除外，盆腔及腹股沟内多发小淋巴结，考虑肿瘤复发。予多柔比星脂质体+奈达铂化疗，插值放疗，但治疗效果不佳，预后差。

以上两例病例均具有 BRG1（-）典型免疫组化特征，基因检测均提示 SMARCA4 体细胞变异，也是 SMARCA4 缺陷型肿瘤患者诊断依据。文献报道 SDUS 罕见，目前文献报道仅 20 余例，预后极差，即使进行积极外科治疗，中位生存期仅为 7-9 月，本病例 1 SDUS 患者经手术+化疗+免疫维持治疗，目前无复发间隔达到 19 个月，治疗效果满意。而病例 2 为宫颈癌放疗后继发 MARCA4 缺失型 UDEC 患者，虽然也进行积极的手术+化疗+免疫治疗，但在术后 5 月出现盆腔复发，治疗效果差。

PO_379

宫腔镜评分及联合预测因子对子宫内膜增生患者术后合并子宫内膜癌的预测价值

韩璐[1,2] 要丽君[1,2] 张东璨[1,2] 马志佳[1,2] 张艳芳[1,2] 王颖梅[1,2] 张慧英[1,2] 田文艳[1,2]
1.天津医科大学总医院
2.天津医科大学总医院 天津市女性生殖健康与优生重点实验室

目的 探讨宫腔镜评分及联合预测因子（宫腔镜表现、超声、MRI、肿瘤标志物）对子宫内膜增生（EH）患者并存子宫内膜癌（EC）的预测价值。

方法 回顾性纳入 2017 年 3 月至 2023 年 1 月天津医科大学总医院 109 例术前诊断为 EH（复杂性增生 13 例、非典型增生 96 例）并行全子宫切除术的患者，依据术后病理分为 EC 组（63 例）和 EH 组（46 例）。收集患者临床资料、宫腔镜表现、超声及盆腔 MRI 表现、肿瘤标志物（HE4 等）。采用二元 Logistic 回归分析独立危险因素，建立联合预测模型，绘制 ROC 曲线评估宫腔镜评分及联合模型的诊断效能。

结果 糖尿病（OR=4.884）、超声下内膜/团块回声增强（OR=11.234）、MRI-DWI 不均匀高信号（OR=7.031）、血清 HE4 升高（OR=1.032）是 EH 并存 EC 的独立预测因子（均 P<0.05）。联合糖尿病、超声回声增强、超声 DWI 不均匀高信号、HE4 构建预测模型，AUC 达 0.793（95%CI 0.710~0.877），显著高于任一单一指标（均 P<0.05）。联合预测因子截断值为-0.9129，灵敏度 76.2%、特异度 71.7%。

结果 宫腔镜评分对识别 EH 术后并存 EC 特异性高，但灵敏度有限；联合糖尿病史、超声回声增强、MRI-DWI 不均匀高信号及血清 HE4 的联合预测因子可显著提升预测价值（AUC=0.793），为术前鉴别 EH 与 EC 提供重要参考。

PO_380

IGF2BP3-mediated upregulation of MDM2 induces immune resistance in endometrial carcinoma by suppressing the p53 signaling pathway

chunqiu xiong[1*] Bingbing Yan[1] Haixia Mo[2]
1.Guangxi Zhuang Autonomous Region Maternal and Child Health Hospital
2.Graduate School, Guizhou University of Traditional Chinese Medicine

Introduction Immune resistance is one of the major reasons for the limited efficacy of immune checkpoint blockade (ICB) therapy in patients with endometrioid endometrial carcinoma (EEC). However, the cellular subsets and underlying mechanisms contributing to this resistance remain poorly characterized.

Methods In this study, we integrated multiple single-cell transcriptomic datasets to construct a comprehensive cellular atlas of EEC. Tumor and immune cell subsets were then further identified, and their biological functions and cellular communications were explored.

Results We identified a significant enrichment of IGF2BP3$^+$ MDM2$^+$ tumor cells in patients with low ICB response. These cells secrete a variety of immunomodulatory ligands that cooperatively recruit immunosuppressive cell subsets, including SPP1$^+$ macrophages and regulatory T cells (Tregs), thereby shaping an immunosuppressive tumor microenvironment. Mechanistically, IGF2BP3 may upregulate MDM2 expression by stabilizing its mRNA, leading to suppression of the p53 signaling pathway and enhanced immune evasion in tumor cells. Furthermore, using artificial intelligence–driven algorithms, we screened and optimized several small-molecule inhibitors targeting MDM2, proposing a potential therapeutic strategy.

Discussion: Our study elucidates key cellular and molecular drivers of immune resistance in EEC and provides a theoretical foundation and candidate compounds for future translational research in targeted therapy.

In the tumor immune niche, this study also observed that SPP1$^+$ macrophages, Tregs and Tex were significantly enriched in patients with low response to immunotherapy. Among them, SPP1 (Secreted Phosphoprotein 1, also known as Osteopontin) is a multifunctional glycoprotein that is widely involved in cell adhesion, migration, inflammation regulation and immune response remodeling. Previous studies have shown that SPP1$^+$ macrophages are highly enriched in a variety of tumors and play a key role in immune escape and treatment tolerance by inducing the expression of immune checkpoint molecules (such as PD-L1), promoting angiogenesis, and regulating fibroblast function [39, 40]. This study further found that IGF2BP3$^+$ MDM2$^+$ EEC cells may actively recruit immunosuppressive subpopulations such as SPP1$^+$ macrophages and LTB$^+$ Tregs by secreting multiple ligands such as CD24, IL7, CCL15, and VEGFB, thereby constructing a multi-level immunosuppressive network.

This study further verified that IGF2BP3 may directly bind to and stabilize MDM2 mRNA, enhance its expression, and synergistically inhibit p53 function, thereby constructing the "IGF2BP3-MDM2-p53" ternary immune escape axis through single-cell co-expression analysis and molecular docking prediction. It is worth emphasizing that this study constructed a small molecule drug screening and optimization process based on artificial intelligence technology to develop potential intervention strategies for targeted blocking of the IGF2BP3-MDM2 axis and reversing immunotherapy resistance. We screened three small molecule candidates (Lumacaftor, Differin, Nilotinib) with strong MDM2 binding ability from the ZINC database, and further designed a batch of new compounds based on the REINVENT deep reinforcement learning algorithm and GAN. Finally, a representative small molecule screened out formed multi-point interactions with the MDM2 protein in structural simulation, showing excellent binding affinity and conformational stability.

In summary, this study constructed a single-cell map of EEC immunotherapy resistance, revealed the key mechanism by which IGF2BP3$^+$ MDM2$^+$ tumor cells mediate immune escape by reshaping the immunosuppressive microenvironment and coordinating SPP1$^+$ macrophages and Tregs, and

designed potential small molecule intervention strategies based on artificial intelligence, proposing a new integrated path of "mechanism analysis-target identification-intervention design". However, this study still has certain limitations, including data integration heterogeneity, lack of functional verification, speculative grouping of ICB response status, and clinical transformation challenges of AI drug screening, which need to be confirmed by mechanism experiments in subsequent studies and promote the clinical application of the results.

PO_381

激活 FOXA2-PR 轴改善子宫内膜癌孕激素治疗敏感性

刘洁
北京大学人民医院

研究目的 子宫内膜癌（EC）保留生育功能治疗中，孕激素受体（PR）的表达水平与孕激素敏感性密切相关。然而，目前尚缺乏有效调控 EC 中 PR 表达的方法。本研究旨在探索 PR 表达的调控机制，寻找提高 EC 孕激素治疗敏感性的潜在策略，为临床提供新的治疗思路。
材料与方法 本研究首先根据 PR 表达水平将患者分为 PR 高表达组和 PR 低表达组，通过转录组学分析比较两组间的通路差异。采用整合共表达网络分析、多队列转录相关性验证和单细胞调控活性分析等方法，系统筛选 PR 表达的关键转录调控因子。通过双荧光素酶报告基因实验和染色质免疫共沉淀技术验证转录因子与 PR 启动子的直接结合以及体内外醋酸甲羟孕酮（MPA）敏感性实验。
结果 研究发现 PR 低表达肿瘤表现出侵袭和转移相关通路的上调，而 PR 高表达肿瘤则显示出增强的代谢活性，特别是在脂肪酸转运和降解方面。通过系统性分析，我们确定 FOXA2 是调控 EC 中 PR 表达的主要转录调节因子。在孕激素敏感与不敏感的患者免疫组化染色显示 PR 和 FOXA2 表达有很好的相关性，体外实验证实 FOXA2 直接结合 PR 启动子区域，从而增强 PR 表达和孕激素敏感性，过表达 FOXA2 后 MPA 敏感性得到改善，而敲低 FOXA2 后细胞对 MPA 处理不敏感。
结论 本研究揭示了 FOXA2 作为 EC 细胞中 PR 表达的主调控因子的重要作用，阐明了 FOXA2-PR 轴在孕激素治疗敏感性中的关键地位，为提高 EC 孕激素治疗敏感性提供了潜在策略。这一发现不仅深化了对 EC 中 PR 表达调控机制的认识，也为临床上改善 EC 保留生育功能治疗效果提供了新的干预靶点和治疗方案。

PO_382

KAT5 介导的 H2A.Z 乙酰化对子宫内膜癌 DNA 损伤以及肿瘤免疫应答的影响

赵舸[1] 连静瑶[2] 李玢[1] 史丽娜[3] 张爽[2*]
1.陕西省人民医院 2.广州医科大学附属第三医院 3.西安市第九医院

研究目的 探讨 KAT5-H2AZac 对子宫内膜癌 DNA 损伤和肿瘤免疫的影响。
材料与方法 （1）免疫组化和多重荧光免疫组化检测子宫内膜癌和正常子宫内膜中 KAT5、H2AZac 和 H4K16ac 的表达水平；（2）WB、qRT-PCR、细胞生长曲线、细胞克隆、IF 和细胞药筛检测腺癌细胞系 KAT5，H2AZac 和 H4K16ac 的 mRNA 和蛋白表达；敲降 KAT5 后对细胞生长的影响；Ki67、PH3、γ-H2AX、RAD51 和 53BP1 的表达；细胞对 PARPi 和 Carboplatin 的药物敏感性；（3）RNA-seq、Cut&Tag 和蛋白质谱分析敲降 KAT5 后的差异基因及蛋白，鉴定 KAT5 调控靶基因及蛋白并做功能分析；（4）IF 检测 F-actin 和 α-Tubulin 的表达；（5）细胞划痕和 qRT-PCR 检测敲降 KAT5 后对细胞迁移的影响以及 ARP2/3 复合物的 mRNA 水平；（6）WB、IF 以及细胞生长曲线检测子宫内膜癌和对

照子宫内膜中 ARPC5 表达以及 ARP2/3 复合物抑制剂 CK-636 对细胞生长、γ-H2AX 以及 α-Tubulin 的影响；（7）检测 cGAS-KO+KAT5 敲降的 lamin A/C、cGAS 定位和激活；（8）检测 ARPC5 过表达+KAT5 敲降后的细胞生长、DNA 损伤以及 cGAS 激活；（9）检测腺癌细胞来源的肿瘤模型、*p53*$^{-/-}$*pten*$^{-/-}$ 类器官模型、*p53*$^{-/-}$*pten*$^{-/-}$ 类器官来源的同源移植模型中 KAT5 抑制剂 Nu9056 对肿瘤生长、DNA 损伤以及 ARPC5 表达的影响。

结果 （1）KAT5 在子宫内膜癌中高表达；敲降 KAT5 抑制 H2AZac；抑制细胞生长；增强 γ-H2AX 表达，同时抑制 RAD51 但不影响 53BP1；增强细胞对化药的敏感性；（2）KAT5 主要调控 H2AZac 来影响肌动蛋白 ARP2/3 复合物的功能，其中 ARPC5 最明显；抑制 F-actin 表达；影响 α-Tubulin 在有丝分裂期纺锤体上的分离和聚合，并抑制细胞迁移；CK-636 抑制细胞生长、导致 DNA 损伤和微管蛋白分布异常；（3）敲降 KAT5 导致 cGAS 在细胞微核上出现特异性富集并激活 TBK1 和 IRF3 的磷酸化表达，进一步激活炎症因子 IL6、CXCL10 和 IFNβ；同时，cGAS-KO 抑制 cGAS 免疫信号通路；（4）ARPC5 过表达可以部分挽救 KAT5 敲降导致的细胞生长抑制、DNA 损伤、cGAS 信号通路激活以及细胞迁移能力；（5）Nu9056 可以抑制 ishikawa 和 HEC-1B 来源的肿瘤模型、*p53*$^{-/-}$*pten*$^{-/-}$ 子宫内膜癌类器官模型和 *p53*$^{-/-}$*pten*$^{-/-}$ 子宫内膜癌类器官来源的肿瘤同源移植模型的肿瘤生长，同时抑制 ARPC5 的表达以及导致肿瘤 DNA 损伤累积。

结论 抑制 KAT5 影响子宫内膜癌生长和 DNA 损伤，这一过程可能与 ARP2/3 复合物的功能有关，并增强肿瘤免疫能力。

PO_383

Association between six insulin resistance surrogates and the risk of uterine corpus cancer: a prospective cohort study using the UK Biobank

Ying Qin
China Medical University

Background Insulin resistance (IR) is a potential risk factor for uterine corpus cancer. However, the independent and the joint associations between genetic risk, IR surrogates markers, and uterine corpus cancer are unknown.

Methods A total of 121,150 participants from the UK Biobank from 2006 to 2010 were included. IR surrogates markers include triglyceride-glucose (TyG) index, TyG-body mass index (TyG-BMI), TyG-waist circumference (TyG-WC), TyG-waist to height ratio (TyG-WHtR), triglyceride-to-high-density lipoprotein cholesterol ratio (TG/HDL-C), and the metabolic score for IR (METS-IR). Genetic susceptibility in uterine corpus cancer was estimated by categorizing polygenic risk scores (PRS). For estimating the associations, we used Cox regression models to estimate associations by calculating hazard ratios (HRs) with 95% confidence intervals (CIs). Restricted cubic splines (RCS) and receiver operating characteristic (ROC) curves were used to assess the association between the six IR surrogates and the risk of uterine corpus cancer.

Result During a median follow-up period of 13.96-years, 748 individuals were diagnosed with uterine corpus cancer. The overall risk of uterine corpus cancer notably increased with increasing TyG index (HR = 1.48, 95%CI = 1.18-1.85), TyG-BMI (HR = 3.34, 95%CI = 2.61-4.29), TyG-WC (HR = 2.78, 95%CI = 2.19-3.52), TyG-WHtR (HR = 2.71, 95%CI = 2.13-3.45), TG/HDL-C (HR = 1.75, 95%CI = 1.39-2.19), and METS-IR (HR = 1.59, 95%CI = 1.29-1.95) levels. Individuals with high genetic risk and high IR surrogates indicators exhibited a substantially elevated uterine corpus cancer risk by 2- to 4-fold compared with reference group. ROC curves showed that TyG-BMI had a more robust diagnostic efficacy than other indicators.

Conclusion TyG index, TyG-BMI, TyG-WC, TyG-WHtR, TG/HDL-C, and METS-IR were closely associated with the risk of uterine corpus cancer. Additionally, TyG-BMI surpassed the other indicators

in predicting and diagnosing the risks of uterine corpus cancer. The TyG-BMI is expected to become a more effective metric for identifying populations at early risk of uterine corpus cancer.

PO_384

Molecular Mechanism of KRT17 Driving Endometrial Cancer Progression by Promoting Epithelial-Mesenchymal Transition

Xiaole Song Fang Ren*
The First Affiliated Hospital of Zhengzhou University

Objective Endometrial cancer (EC) is one of the most prevalent gynecological malignancies globally, with advanced and recurrent cases often resulting in poor prognosis. Keratin 17 (KRT17), a member of the type I keratin family, has been implicated in the oncogenesis of various cancers. However, its precise role and underlying mechanism in EC remain unclear. This study aims to investigate the expression pattern and prognostic significance of KRT17 in EC, and to elucidate its molecular mechanism in promoting EC progression through the regulation of epithelial-mesenchymal transition (EMT).

Materials and Methods Transcriptomic data from the TCGA-UCEC database were analyzed to assess the differential expression of KRT17 in EC tissues versus normal endometrial tissues, utilizing the GEPIA and UALCAN platforms. The association between KRT17 expression and clinical parameters (including tumor stage, grade, and lymph node metastasis) as well as patient prognosis was evaluated. Pathway enrichment analysis of KRT17-coexpressed genes was conducted using Metascape to identify potential signaling pathways involved in its oncogenic activity. In vitro experiments were performed to verify KRT17 expression in EC tissues and cell lines (Ishikawa, AN3CA, HEC1A, HEC1B, SPEC2) using qRT-PCR, Western blot, and immunohistochemistry (IHC). Stable KRT17-knockdown Ishikawa and HEC1A cell lines were generated via shRNA. The impact of KRT17 knockdown on cell proliferation was assessed by CCK-8, EdU, and colony formation assays; cell migration and invasion were evaluated using Transwell and wound-healing assays; and stemness was assessed by sphere formation assays. EMT-related marker expression (E-cadherin, N-cadherin, Vimentin, Snail1) at both mRNA and protein levels was measured to explore the regulatory mechanism. In vivo tumorigenicity assays in nude mouse models were conducted to confirm the effect of KRT17 knockdown on tumor growth and EMT processes.

Results Analysis of TCGA data and IHC staining revealed that both KRT17 mRNA and protein levels were significantly elevated in EC tissues compared to normal endometrial tissues. High KRT17 expression was significantly associated with advanced FIGO stage, high histological grade, lymph node metastasis, and poor patient outcomes (including both overall survival and disease-specific survival; $P<0.05$). Bioinformatics analysis identified that genes coexpressed with KRT17 were predominantly enriched in EMT-related pathways. In vitro, KRT17 knockdown notably inhibited EC cell proliferation, migration, invasion, and stemness. At the molecular level, KRT17 silencing upregulated the epithelial marker E-cadherin and downregulated the mesenchymal markers N-cadherin, Vimentin, and the transcription factor Snail1 at both mRNA and protein levels. In vivo, KRT17 knockdown resulted in a reduced tumor growth rate and suppressed EMT transformation in xenograft tumors.

Conclusion: KRT17 is highly expressed in EC and facilitates malignant progression through activation of the EMT pathway. Its elevated expression serves as an independent prognostic marker for poor outcomes in EC patients. These findings highlight KRT17 as a promising therapeutic target for EC and provide a foundation for the development of KRT17-targeted diagnostic and treatment strategies.

PO_385

关于子宫内膜间质肿瘤病理诊断及分子生物学研究进展的综述

付溪娜 王志启*
首都医科大学附属北京友谊医院

子宫内膜间质肿瘤（endometrial stromal tumors,ESTs）是一种罕见的来源于子宫内膜间质细胞的肿瘤，分为子宫内膜间质结节（endometrial stromal nodule,ESN），低级别子宫内膜间质肉瘤（low-grade endometrial stromal sarcoma，LG-ESS），高级别子宫内膜间质肉瘤（high-grade endometrial stromal sarcoma，HG-ESS)，和未分化子宫肉瘤（undifferentiat-eduterine sarcoma,USS)四种类型。由于 ESTs 多无特异性临床表现，影像学检查对本病的诊断帮助有限，因此病理诊断及分子生物学检查对 ESTs 的诊断及鉴别诊断尤为重要。本文就 ESTs 的病理特征、免疫表型及分子遗传学改变进行了如下总结。ESN 病理学特征为肿瘤边界清楚，有宽而光滑的推挤性边缘。核分裂像罕见；LG-ESS 有子宫肌层及淋巴脉管浸润，核分裂像较低，此二者的主要区别在于子宫肌层浸润深度及病灶数目，因此刮宫标本无法鉴别二者。HG-ESS 常广泛弥漫浸润子宫肌层，多数核分裂象活跃。而 USS 缺乏特异性分化，向周围组织呈破坏性浸润，常见病理性核分裂象。免疫组化有助于 ESTs 的鉴别诊断，推荐进行 IFITM1 或 CD10 联合 h-caldesmon 染色。LG-ESS 通过 t(7；17)染色体易位形成 JAZF1 一 SUZ12 融合基因最常见。ESN 与 LG-ESS 免疫表型及融合基因具有高度一致性。HG-ESS 分为伴有 YWHAE-NUTM2A/B 基因重排及伴有 B 细胞淋巴瘤因子 6 共抑制因子（BCL6co repressor,BCOR)基因异常两类。随着分子生物学技术的发展，越来越多的新型融合基因被发现，为 ESTs 的诊断及治疗提供了越来越多的证据和思路。

PO_386

ADAMTS18 与子宫内膜癌生物学意义的研究及其与子宫内膜癌预后的相关性分析

郭欣 徐延华*
济南市妇幼保健院

目的 探究含血小板反应蛋白 18 型基序的解聚素样金属蛋白酶（A disintegrin and metalloproteinase with thrombospondin motif 18，ADAMTS18）在子宫内膜癌（Endometrial cancer，EC）中的生物学意义，并探究 ADAMTS18 与 EC 患者预后的相关性。

材料与方法 收集我院妇科一区 2021 年至 2023 年 EC 患者（68 例），设为 EC 组，子宫内膜不典型增生（33 例），设为 AH 组，同时设置正常内膜组织组（68 例）为对照组。通过免疫组织化学染色及荧光免疫方法，检测 ADAMTS18 在三组中的差异性表达。同时，通过单因素及多因素分析法探究 ADAMTS18 与 EC 患者的临床预后相关性，并探究 ADAMTS18 与癌前病变疾病进展的相关性。利用慢病毒介导的基因沉默和过表达技术，在子宫内膜癌细胞株 Ishikawa 中构建 ADAMTS18 基因沉默和过表达的稳转株， 通过 MTT 法检测子宫内膜癌细胞 ADAMTS18 基因沉默和过表达后对细胞增殖能力的影响，通过划痕实验、 Transwell 实验检测 ADAMTS18 基因沉默和过表达后对子宫内膜癌细胞系的迁移及侵袭能力的影响。

结果 ADAMTS18 在 EC 组的表达量明显低于 AH 组及对照组（P<0.05），对照组中 ADAMTS18 的表达量显著高于 AH 组（P<0.05）。ADAMTS18 的低表达与较高的 FIGO 分期（≥Ⅲ期）、较大的肿瘤直径（≥2cm）及较深的子宫肌层侵润（≥1/2）相关（P<0.05）。ADAMTS18 的低表达与较短的无病生存期相关（P<0.05），在子宫内膜不典型增生患者中，ADAMTS18 的低表达与疾病进展相关（P<0.05）。在体外培养模型中，ADAMTS18 的过表达可有效的抑制子宫内膜癌细胞的增殖、迁徙及侵袭能力（P<0.05）。

结论 ADAMTS18 在子宫内膜癌、癌前病变及正常组织中差异性表达，ADAMTS18 与子宫内膜癌的分期、淋巴转移、增殖、侵袭等指标相关，其低表示与癌前病变进展及 EC 患者术后的不良预后相关，可作为子宫内膜癌风险评估及不良预后预测的生物指标。

PO_387

空间多组学解析子宫内膜癌淋巴血管间隙侵犯特征

兀紫梦[1] 胡哲[1] 孙朝阳[1] 陈刚[1*] 陈晓军[2]
1.华中科技大学同济医学院附属同济医院 2.同济大学附属第十医院

背景 淋巴血管间隙侵犯（LVSI）是子宫内膜癌（EC）患者预后不良的重要指标，但其微观浸润机制尚未明确，特别是在空间层面解析肿瘤细胞如何与免疫微环境协同促进淋巴侵袭仍属空白。
目的 本研究旨在通过空间多组学解析，系统揭示 EC 中 LVSI 相关的细胞组成、信号网络与代谢特征，探索促进肿瘤细胞侵袭淋巴管的关键免疫调控机制，并提供潜在干预靶点。
方法 我们纳入 26 例 EC 患者（12 例 LVSI 阳性、14 例 LVSI 阴性），使用激光显微切割（LCM）联合质谱技术对 106 个特征区域进行空间蛋白质组学分析，并结合配对的空间转录组数据，重构 LVSI 区域的免疫微环境图谱。进一步在体外构建 THP-1 诱导的类巨噬细胞-内膜癌细胞共培养体系，验证关键因子诱导效应，并在 $Apoe^{-/-}$ 和 Lyz2-Cre 驱动的条件性敲除小鼠中构建原位移植模型，验证其对肿瘤淋巴侵袭的调控作用。
结果 空间组学结果显示，LVSI 区域内部显著浸润 $APOE^{+}$巨噬细胞，伴有肿瘤细胞脂质代谢相关通路激活。空间转录组进一步揭示，这类 $APOE^{+}$巨噬细胞具备免疫抑制表型，并通过旁分泌机制影响肿瘤细胞代谢状态。体外实验中，APOE 重组蛋白或 THP-1 诱导的 $APOE^{+}$巨噬细胞可诱导 EC 细胞脂代谢重编程并增强其侵袭能力；在 $Apoe^{-/-}$；Lyz2-Cre 小鼠中，原位移植后 LVSI 发生率显著下降，验证了 $APOE^{+}$巨噬细胞在 LVSI 形成中的关键功能。
结果 本研究首次整合空间蛋白质组与转录组，在组织水平描绘了 EC 中 LVSI 区域的细胞-代谢互作图谱，提出 $APOE^{+}$巨噬细胞通过诱导肿瘤细胞脂代谢重编程，促进其侵袭淋巴管的机制。该发现不仅填补了 EC 淋巴浸润机制的研究空白，也为开发针对脂代谢或巨噬细胞重编程的干预策略提供了理论基础和转化路径。

PO_388

基于深度学习的子宫内膜癌组织学分类模型构建及活检标本验证

王倩 陈晓军*
上海市第十人民医院

目的 子宫内膜癌（Endometrial cancer, EC）的发病率逐年上升，其精准的组织学评估和分子分型对患者管理及治疗方案选择至关重要。分子分型虽能提供更精准的预后和治疗指导，但在资源有限地区难以普及；传统组织学分型及分级依赖细胞异型性和肿瘤结构，存在显著的观察者内和观察者间差异，可能导致治疗偏差。人工智能技术通过计算病理学突破传统限制，如 Panoptes、EndoNet 及 im4MEC 等模型在亚型分类和分子特征预测中表现优异，但其均缺乏对活检组织的验证。
方法 本研究数据来自复旦大学附属妇产科医院（中心 1）、上海市第十人民医院（中心 2）及 TCGA 队列。内部数据集包括中心 1 的 268 例 EC 患者的 294 张子宫全切术后 H&E 染色全切片图像（Whole slide images , WSIs）和 TCGA 队列的 516 例患者的 585 张术后 WSIs，按 8:1:1 比例随机分为训练集、验证集和内部测试集。中心 2 的 88 例子宫全切术后 WSIs 和 47 例活检 WSIs 作为外部测试集。纳入标

准为具备活检或全子宫切除术后组织病理诊断为 EC 的 H&E 染色切片数字化扫描图像的患者。排除标准为切片中无肿瘤病灶或切片质量不达标者。将 WSIs 切分为 256×256 的图像块，用预训练 UNI 编码器 + CLAM 框架构建多任务 Hisnet-RF 模型，同时预测组织学类型及分级。训练过程固定编码器参数，使用交叉熵损失函数优化分类头，Adam 优化器学习率设为 1×10-4，权重衰减 1×10-5。以专业的病理医生诊断结果为金标准，通过计算敏感度、特异度、F1 分数及 AUC 值以评估模型性能。
结果: 在内部测试集个体层面的分析结果显示，HisNet-RF 模型预测组织学类型的准确率为 0.818，AUC 为 0.847；正确区分病理分级的准确率为 0.810，AUC 为 0.833；在内部测试集组织层面的分析结果显示，与个体层面相比，ΔAUC＜0.02。在中心 2 的术后病理图像和活检图像中，模型区分不同组织学类型和病理分级的 AUC 分别为 0.918/0.939 和 0.955/0.988。此外，本研究运用整合原始 WSIs、梯度加权类激活热力图及前 10 个高注意力图像块的可视化方法，对模型预测组织学类型及分子分型的机制进行可解释分析。结果显示，在预测 Non-EEC 时，高注意力区域呈现肿瘤细胞核质比增大、核深染等特征。
结果 Hisnet-RF 模型在预测组织学类型及病理分级时展现优异性能，且在活检标本中具有稳定的泛化性能，有望辅助病理学家提升组织分类准确性，尤其在资源有限地区，可用于指导评估保留生育功能治疗可行性。

PO_389

子宫内膜癌复发风险预测模型的构建及应用

王倩 陈晓军*
上海市第十人民医院

目的 子宫内膜癌（Endometrial cancer, EC）发病率逐年上升。尽管多数 EC 患者能在早期被诊断且预后良好，但仍有 10%的患者存在复发风险，预后欠佳。既往有研究基于 COX 模型通过纳入多个与复发相关的危险因素，开发预测 EC 复发风险的列线图，或通过机器学习算法构建预测模型，但其均未纳入 2023 年 FIGO 分期及分子分型。
方法 本回顾性研究共纳入中心 1（复旦大学附属妇产科医院，4152 例）和中心 2（上海市第十人民医院，104 例）行全子宫切除术且术后确诊子宫内膜癌的患者，剔除 FIGOⅣ期、接受新辅助治疗及失访病例后，采集临床-分子病理参数，按照分期标准对 FIGO 分期增加分子分型修正为 FIGOm 分期。中心 1 数据按 8:2 分层随机抽样为训练集与内部测试集；中心 2 作为外部测试集。基于随机生存森林构建 CPFmRR 模型，经变量标准化与编码后输出个体化无复发生存概率。
结果 本研究在训练集、内部测试集与外部测试集中患者的中位随访时间分别为 2.87（0.11-8.92）2.77（0.08-8.58）和4.71（1.01-8.58）年。内部数据集中共223例患者出现复发，其中远处转移为76例，阴道复发为 14 例、盆腔复发为 11 例，中位复发时间为 1.71 年；外部测试集中 6 例出现复发，其中远处转移及部位不详者各 3 例，中位复发时间为 1.32 年。CPFmRR 模型在内部和外部测试集的 C 指数分别为 0.856（95%CI:0.788-0.924）和 0.960（95%CI: 0.944-0.976）。基于 Youden 指数最大化原则确定危险分数的阈值为 7.79，其对应敏感度为 92.1%，特异度为 81.6%，Youden 指数为 0.737。将该阈值应用于内部测试集和外部测试集中进行复发风险分组，结果发现：内部测试集中高风险组患者占 23.7%（197/831），且高风险组 5 年无复发生存率（69.8%）显著低于低风险组（99.0%，log-rank P= 2×10^{-29}）；外部测试集中高风险组患者占 21.2%（22/104），其 5 年生存率为 75.3%，而低风险组为 100%（log-rank P= 1×10^{-6}）。本研究基于 R 语言 Shiny 框架开发了动态复发预测支持系统。该系统通过交互式界面实现多维度临床数据的结构化输入，涵盖模型纳入的所有变量，可实时生成个体化预测报告包括复发生存曲线以及复发风险分组等信息，用于 EC 患者进行精准复发风险分层。
结论 基于随机生存森林算法通过整合 FIGOm 分期与临床病理特征构建 CPFmRR 预测模型，基于 Youden 指数最大化原则确定危险分数阈值实现精准分层，并开发 R Shiny 在线系统动态评估个体化复发风险，为个体化辅助治疗及分层随访提供决策依据。

PO_390

加深对高级别子宫内膜间质肉瘤的认识

杨梓晔[1] 曲芃芃[2*]
1.南开大学医学院 2.天津市中心妇产科医院妇瘤科

研究目的 探讨高级别子宫内膜间质肉瘤（High grade endometrial stromal sarcoma，HGESS）的临床病理特征，以加深对该疾病的认识，为其诊断、治疗及预后评估提供参考。
材料与方法 收集 12 例 HGESS 患者病史、一般情况、临床表现、影像学检查、实验室检查、术前诊断、术后病理、治疗情况、分期及预后等，进行回顾与分析。
结果 12 例 HGESS 患者的平均发病年龄（53.59 ± 8.48）岁，临床分期Ⅰ期 4 例（33.33%），Ⅱ期 3 例（25.00%），Ⅲ期 3 例（25.00%），Ⅳ期 2 例（16.67%），宫腔镜检查术前诊断灵敏度为 0.6，诊刮术前诊断灵敏度为 0.429，肿瘤大体标本表现为质地细腻，似鱼肉样，常伴出血坏死及囊变，电动子宫/肌瘤粉碎器的使用未增加患者的复发率，典型磁共振影像学特征包括：1.肿瘤呈膨胀性生长；2.肿瘤中心多有出血、坏死；3.弥散后 DWI 呈高信号；4.ADC 呈低信号；5.增强后 T2WI 坏死呈高信号。具有 ZC3H7B-BCOR 基因重排的患者肿瘤生物学行为存在差异。截止随访日期共 5 名患者复发，Ⅲ-Ⅳ期 5 例中 4 例复发，有淋巴结转移 3 例中有 2 例复发，CA125 升高 6 例中有 4 例复发，复发患者中 3 例因本病死亡。
结论 HGESS 临床症状不典型，术前明确诊断较困难，宫腔镜与诊刮作为诊断方法的灵敏度均不够高，MRI 成像作为术前诊断辅助方法灵敏度较高，分期为Ⅲ-Ⅳ期、有淋巴结转移及 CA125 升高的患者复发率高，仍需进一步加深 HGESS 主要三类分子改变对生存情况差异的相关研究。

PO_391

The Impact of Altered Gut Microbiota and Lipid Metabolism on the Progression of Endometrial Cancer in Overweight Populations

Chen Jiayu
The First Clinical School of Gansu University of Traditional Chinese Medicine

Background Endometrial cancer (EC) is one of the common malignant tumors among women, and in recent years, the role of gut microbiota in tumorigenesis has been increasingly gaining attention.Existing research has shown that the gut microbiome, establishes axis connections with multiple extra-intestinal organs. However, whether gut microbes affect the process of endometrial carcinogenesis through metabolic pathways and the specific mechanisms by which they promote the development of EC remain unclear. This study aims to explore the impact of overweight-mediated gut microbiota on the initiation or progression of EC and to assess its relationship with metabolites, thereby providing new insights for early diagnosis and treatment.
Methods In this study, we analyzed gut microbiota differences among normal-weight, overweight EC patients, and healthy controls using 16S rRNA sequencing. Liquid chromatography-mass spectrometry (LC-MS) and KEGG analysis identified group-specific metabolites and pathways, while Spearman correlation analysis revealed associations between microbiota and metabolites.
Results This study revealed that in the ECMO group, the genus Megamonas exhibited the highest abundance and significant intergroup differences ($H=13.46$, $P<0.05$). Additionally, the Bacillota/Bacteroidota ratio (B/B ratio) gradually increased in the CN, ECMN, ECMO group. LEfSe analysis identified Megamonas and Amedibacillus as potential biomarkers for the ECMO group. Serum metabolomics of overweight EC patients highlighted lipid metabolism-related metabolites with the most specific expression. KEGG enrichment analysis of differential metabolites highlighted that the

Glycerophospholipid metabolism and Purine metabolism pathways were notably significant in both the ECMN and ECMO groups.
Conclusion The study found significantly elevated abundance of Megamonas in the gut microbiota of overweight EC patients, which may promote EC progression by degrading inositol to enhance lipid absorption. This reveals the role of gut microbiota in EC pathogenesis through lipid metabolism regulation, providing a theoretical basis for microbiota-based diagnostic and therapeutic strategies.

PO_392

Construction of lactylation-related gene model for prognosis and immune infiltration in endometrial cancer

Yuxi Jin Ruixia Guo*
The First Affiliated Hospital of Zhengzhou University

Background Endometrial cancer (EC) is a prevalent malignant tumor of the female reproductive system. The identification of novel molecular markers and prognostic indicators is crucial for guiding the clinical treatment of EC. Protein lactylation modification has been reported as a significant factor in cancer progression.
Methods We analyzed the expression levels of lactylation-related genes using data from the TCGA database. Cox regression and least absolute shrinkage and selection operator (LASSO) regression were used to construct a lactylation-related gene signature. We then assessed immune cell infiltration and immunotherapy response sensitivity across the high- and low- risk subtypes. MTT, EdU and tumor xenograft experiments were conducted to elucidate the roles of HDGF and XPO5 in EC.
Results A 9-gene signature (ACAT2, HDGF, LGALS1, PFKM, PRCC, TCOF1, TP53 WAS, XPO5) was constructed and validated. Patients with higher risk scores exhibited poorer clinical outcomes. The low-risk group was associated with higher microsatellite instability-high (MSI-H) levels and was more sensitive to immunotherapy. A nomogram integrating the signature and clinical characteristics was developed to predict individual survival with high stability. Knockdown of HDGF or XPO5 suppressed EC cell proliferation both in vitro and in vivo.
Conclusions The lactylation-related model demonstrated robust predictive efficiency of EC, assisting clinicians in decision-making and treatment customization. The function of HDGF and XPO5 in EC was validated via cell experiments, highlighting its potential as biomarkers and therapeutic targets.

PO_393

Combined ER/PR Co-positivity Enhances Prognostic Stratification Within Molecular Subtypes of Endometrial Cancer

Ruxue Han[1] Yang Chen[1] Shuiqing Xu[1] Tingting Zhang[1] Xingran Jiang[2] Hua Li[1*]
1.Department of Obstetrics and Gynecology, Beijing Chao-Yang Hospital, Capital Medical University, Beijing, China.
2.Department of Pathology, Beijing Chao-Yang Hospital, Capital Medical University, Beijing, China.

Objective The no specific molecular profile (NSMP) subtype constitutes the largest yet prognostically heterogeneous subgroup of endometrial cancer (EC), with current tools inadequately stratifying its risk. This study aimed to investigate the prognostic value of combined ER/PR expression patterns across EC molecular subtypes, focusing on refining risk stratification for the NSMP subtype.
Materials and Methods A retrospective cohort of 388 EC patients from Beijing Chao-Yang Hospital (January 2015–December 2024) was enrolled. All tumors underwent NGS-based molecular

classification (POLEmut, MMRd, p53abn, NSMP) and ER/PR immunohistochemistry (IHC, ≥1% nuclear staining as positive). ER/PR status was categorized as co-positive (coP), co-negative (coN), or discordant. Survival outcomes (DSS, PFS) were analyzed using Kaplan-Meier curves, multivariable Cox models, and C-index analyses.

Results Among 388 patients, 65.5% (254/388) were NSMP, 17.8% (69/388) MMRd, 11.1% (43/388) p53abn, and 5.7% (22/388) POLEmut (Figure 1). ER/PR coP accounted for 87.6% (340/388), coN 8.2% (32/388), and discordant 4.1% (16/388).

In the overall cohort, ER/PR co-positivity independently predicted improved DSS (HR=0.143, 95%CI:0.065–0.314; p<0.001) after adjusting for stage and molecular subtype, while isolated ER/PR did not (Figure 2C). Kaplan-Meier curves showed significantly better DSS/PFS in coP patients (Figure 2A-B).

Within NSMP, coP was associated with superior DSS (HR=0.21, 95%CI:0.06–0.87; p=0.034), and ER positivity predicted better PFS (HR=0.13, 95%CI:0.02–0.88; p=0.037) (Figure 3A-B). Multivariable analyses confirmed these associations (Figure 3C-F). Adding coP to staging improved NSMP-DSS prediction (ΔC-index=+0.082; p<0.05) (Figure 3G-H). No significant associations were found in non-NSMP subgroups.

Conclusion ER/PR co-positivity is an independent predictor of favorable DSS in EC, significantly enhancing prognostic stratification for the NSMP subtype. Integrating ER/PR co-expression with molecular classification optimizes EC management.

PO_394

OA-HOXB9-ODC1-Polyamine Axis Mediates Metabolic Syndrome Promoting Endometrial Cancer Progression

Lirong Zhai Yuan Cheng Jianliu Wang*
Peking University People's Hospital

Metabolic syndrome (MS) is a risk factor for both the development and progression of endometrial cancer (EC); however, the mechanism remains unclear. We found that polyamine metabolites are significantly elevated in the sera and tumor tissues of EC patients with MS (ECWMS). Hyperlipidemia is a critical factor in promoting ECWMS, and oleic acid (OA), an important monounsaturated fatty acid, was identified from 12 common fatty acids as a crucial element in upregulating ornithine decarboxylase 1 (ODC1), the rate-limiting enzyme in polyamine metabolism, and downstream polyamines in the plasma of ECWMS. Mechanistically, OA binds directly to HOXB9 and stabilizes it by preventing its interaction with E3 ligases Praja2. HOXB9 further interacts with ODC1 and competes with the interaction between ornithine decarboxylase antizyme 1 (OAZ1) and ODC1 for proteasomal degradation. The downstream accumulation of polyamine metabolites, especially putrescine, further inhibits the degradation of HOXB9. Targeting the feedback of the HOXB9-ODC1-polyamine axis decreases polyamines and inhibits tumor proliferation and metastasis in vitro and in vivo. Clinically, the combination of ODC1, HOXB9, and obesity could differentiate the prognosis more accurately. Multiplex immune fluorescence staining, LC-MS, Raman spectroscopy indicated that the OA-HOXB9-ODC1-polyamine axis also exists in ECWMS. Furthermore, in patient-derived tumor cells (PTCs), the administration of ODC1 inhibitors can effectively enhance the sensitivity to first-line chemotherapeutic agents paclitaxel and carboplatin.This study links fatty acid levels to polyamine accumulation, ultimately promoting EC progression and revealing the mechanism for MS promoting EC progression. Targeting HOXB9 or ODC1 is expected to be a potential therapeutic strategy to control patients with MS-related refractory or progressive EC.

PO_395

B3GNT3 在子宫内膜样腺癌中的表达及临床意义

王一帆
郑州大学第一附属医院

目的 探究 β-1,3-N-乙酰葡糖胺基转移酶 3（B3GNT3）在子宫内膜样腺癌（EC）中的表达及临床意义。
材料与方法 收集 2024 年 1 月至 2024 年 11 月于郑州大学第一附属医院妇科手术治疗的子宫内膜样腺癌组织及癌旁组织，提取 RNA 和蛋白质；选取 2017 年 1 月至 2024 年 11 月郑州大学第一附属医院病理科存档的 109 例子宫内膜样腺癌石蜡标本，整理患者临床资料、病理信息并随访患者生存及复发情况。通过 qRT-PCR、免疫组织化学染色、Western blot 实验探究子宫内膜样腺癌组织与癌旁组织中 B3GNT3 的表达水平，分析 B3GNT3 表达水平与患者临床病理特征的关系。
结果 子宫内膜样腺癌组织中 B3GNT3 mRNA 和蛋白的表达水平均明显高于癌旁组织，B3GNT3 表达与肿瘤 FIGO 分期、有无淋巴结转移具有相关性，并且与患者预后相关即 B3GNT3 高表达组患者预后较差。
结论 B3GNT3 可能参与了子宫内膜癌的发生、发展，有望成为预测子宫内膜癌的预后指标。

PO_396

回顾性多组学分析子宫内膜癌免疫微环境特征研究进展

王丹苧 袁光文*
中国医学科学院肿瘤医院

子宫内膜癌（Endometrial cancer，EC）是女性生殖系统最常见的恶性肿瘤之一，近年来其发病率呈持续上升趋势，尽管早期 EC 患者大多预后良好，晚期及复发患者生存率显著降低，其对免疫治疗的响应也存在显著异质性，但相关分子调控机制尚未完全阐明。本综述旨在系统整合 EC 免疫微环境（Tumor Immune Microenvironment, TIME）异质性的分子基础及其与免疫治疗抵抗的关联，为精准分型与治疗策略优化提供理论依据。通过系统检索 PubMed 数据库近五年发表的解析 EC 免疫微环境多组学研究文献，初筛 231 篇相关文献，聚焦多组学方法解析 EC 肿瘤-免疫细胞互作网络及关键免疫抑制驱动机制的文献进行二次筛选，最终纳入 81 篇高相关文献进行文献综述。本综述在 EC 分子分型基础上根据不同 TIME 特征构建出特异性 TIME 图谱，将 EC 分为：1) POLEmut/MSI-H 型（高免疫浸润型）；2) p53abn 型（免疫抑制型）；3) NSMP 型（免疫荒漠型）。该图谱阐释了 TIME 异质性的形成机制及其对免疫治疗反应不同的基础。此外，整合多组学研究揭示了包括特定基因和蛋白（IGSF9，EIF2AK4、EPPK1、CDH18、PAGE2B 等），整合 RAP1、CAMSAP3 和 TP53 突变状态的预测模型，以及 TIME 中 CD8+T 细胞动态在内的潜在新型生物标志物。深入解析 TIME 异质性并鉴定相关生物标志物，将有助于 EC 早期准确诊断和预后判断，为精准风险分层和优化个体化免疫治疗策略奠定重要的理论基础。

PO_397

Targeting lipid metabolism to inhibit endometrial cancer

Lulu Si Ruixia Guo*
The First Affiliated Hospital of Zhengzhou University

Background Endometrial carcinoma (EC) is one of the most common malignancies of the female reproductive tract, and its incidence is rising rapidly. Among all obesity-related cancers, EC shows the

strongest association with excess body weight: each 5-unit increase in BMI elevates the risk of developing EC by 1.63-fold. This epidemiological link implicates dysregulated lipid metabolism in EC pathogenesis. Nevertheless, the molecular mechanisms through which lipid-metabolic reprogramming drives EC progression remain incompletely understood. Elucidating these mechanisms and identifying novel therapeutic targets are therefore essential for the prevention and early treatment of EC.
Methods Clinical samples were used to correlate DGAT1 expression with clinicopathological features of EC. CCK-8, colony-formation and EdU incorporation assays were performed to evaluate the impact of DGAT1 on EC cell proliferation and tumorigenicity. Untargeted lipidomic profiling, Seahorse metabolic flux analysis, transmission electron microscopy (TEM) and flow cytometry were employed to dissect the molecular mechanisms by which DGAT1 regulates EC progression. The anti-tumor efficacy of a small-molecule DGAT1 inhibitor was assessed in EC cells.
Results DGAT1 expression was markedly higher in EC tissues than in matched adjacent non-tumor endometrium, and elevated DGAT1 correlated with adverse prognosis. Exposure of EC cells to a high-fat environment up-regulated DGAT1 expression. Genetic knockdown of DGAT1 attenuated EC cell proliferation and colony formation, while markedly increasing cellular ROS levels. Mitochondrial stress tests revealed that DGAT1 depletion reduced basal, maximal and spare respiratory capacities and caused severe ultrastructural damage to mitochondria. Lipidomic analysis showed that DGAT1 knockdown profoundly altered the cellular lipidome: acyl-carnitines (AcCa), critical intermediates in mitochondrial β-oxidation and energy metabolism, were significantly elevated, whereas the membrane-structural phospholipids PC and PE were decreased. Exogenous supplementation with the two most up-regulated AcCa species (C16 and C18) recapitulated the anti-proliferative and pro-oxidative effects observed upon DGAT1 knockdown. DGAT1 silencing down-regulated CPT1A, the rate-limiting enzyme in acyl-carnitine synthesis and fatty-acid oxidation. Pharmacological inhibition of DGAT1 with a small-molecule inhibitor markedly suppressed EC cell proliferation.
Conclusions In endometrial carcinoma, DGAT1 promotes tumor cell proliferation by repressing CPT1A-mediated fatty-acid oxidation, thereby disrupting lipid homeostasis, inducing mitochondrial dysfunction and provoking oxidative stress. Targeted inhibition of DGAT1 reverses these metabolic aberrations and represents a promising metabolic vulnerability for EC therapy.

PO_398

ANLN 在子宫内膜癌中的表达及其与免疫浸润的相关性

彭亚楠 王娟 李福霞*
石河子大学

背景 子宫内膜癌（Endometrialcancer，EC）是全球女性中最常见的恶性肿瘤之一，发病率尤其在发达国家逐年上升，对女性健康构成重大威胁。ANLN（Anillin）是一种在细胞分裂中起重要作用的蛋白质，它参与调控细胞骨架的重组和细胞周期的进程。ANLN 的表达水平与多种癌症的预后呈明显负相关，其表达水平升高预示不良预后。因此，本文章旨在揭示 ANLN 在 EC 中的表达及其与免疫浸润的相关性。
方法 运用 TIMMER 数据库分析 ANLN 在泛癌中的表达。TCGA 数据库分析 ANLN 在 EC 组织和正常子宫内膜组织中的表达差异及其与 EC 患者临床特征的相关性。HPA 数据库进一步验证。survival、survminerR 包、Kaplan-Meier Plotter 法分析 ANLN 的表达与 EC 患者预后的关系。使用“limma”软件包鉴定了与 ANLN 共表达的基因（P 值<0.05 和|log2（倍数变化）|> 1），并使用 R 软件包“ClusterProfiler”检查生物学功能。CIBERSORT 算法分析 ANLN 不同表达组在 EC 中免疫细胞浸润水平的差异，ESTIMATE 分析 ANLN 在基质评分和免疫评分的表达差异。免疫组织化学分析 ANLN 在癌组织及癌旁的表达。
结果 在 TIMMER 数据库中发现 ANLN 在大多数肿瘤中高表达；ANLN 在 EC 中表达上调，且与年龄和临床分级相关；EC 患者高 ANLN 与预后差有高度相关性。ANLN 在肿瘤中与多种信号通路相关。免疫浸润分析表明，ANLN 与调节 T 细胞和树突状细胞浸润水平显著相关。ESTIMATE 算法显示 ANLN 高

表达与较低的基质评分和免疫评分相关。免疫组织化学方法显示 ANLN 在 EC 组织中的表达水平显著高于正常子宫内膜组织。
结论 ANLN 在 EC 组织中高表达；ANLN 的高表达且与较差的预后相关；ANLN 可能参与了 EC 免疫反应的调节，且免疫浸润可能是 ANLN 高表达 EC 患者预后较差的原因之一。

PO_399

Regulatory mechanism and prognostic significance of lymphovascular space invasion in early-stage low-grade endometrioid endometrial cancer

Yuan Cheng*
Peking University People's Hospital

Objective In 2016, the ESMO-ESGO-ESTRO consensus included LVSI (Lymph-vascular space invasion, LVSI) status as a risk stratification factor for stage I endometrioid endometrial cancer (EEC) patients and as one of the indications for adjuvant therapy. Furthermore, LVSI is included in the new FIGO staging of endometrial cancer (EC) in 2023. However, the data contribution of the Chinese population in this regard is limited. The present study aimed to comfirm the influence of LVSI on the prognosis of early-stage low-grade EEC in a fifteen-year retrospective Chinese cohort study. And, further explore the molecular mechanisms of Tumor-Associated Macrophages (TAMs) and secreted factor CCL18 in EC LVSI and metastasis.
Methods This retrospective analysis cohort included 702 EEC patients who underwent TAH/BSO surgery, total abdominal hysterectomy, bilateral salpingooophorectomy in Peking University People's Hospital from 2006 to 2020. Patients were stratified based on LVSI expression status as: LVSI negative group and LVSI positive group. Clinical outcome measures related to LVSI, assessed with a univariate and multivariate Cox proportional hazards regression model. Using Nanostring technology, the significant roles of macrophages and CCL18 in LVSI were identified. The established stable cell line was then treated with CCL18, and the role of CXCR4 in CCL18-induced tumor metastasis, along with its underlying molecular mechanisms, was investigated through Transwell migration assays and Western blotting. To further examine the in vivo effect of CXCR4 on endometrial cancer progression under CCL18 stimulation, Ishikawa cells with stable CXCR4 knockdown were subcutaneously injected into nude mice.
Results 702 EEC patients with stage I and grade 1-2 were analyzed. 58 patients (8.3%) were LVSI-positive and 14 patients (2.0%) was relapse. Recurrence rates in LVSI-negative and LVSI-positive were 1.6% and 6.9%, respectively. 5-year disease-free survival (DFS) rate in LVSI-negative and LVSI-positive were 98.4% and 93.1%, respectively. These rates for 5-year overall (OS) survival in LVSI-negative were 98.9% while it was 94.8% in LVSI-positive. Multivariate analysis showed that LVSI is an independent risk factor for 5-year DFS (HR = 4.60, p = 0.010). LVSI has a similar result for 5-year OS(HR = 4.39, p = 0.028). Knockout of CXCR4 significantly inhibited the migration ability of endometrial cancer cells under CCL18 intervention, as well as the protein expression levels of COL6A1 and COL6A2, confirming the critical role of CXCR4 in CCL18-mediated metastasis and collagen synthesis. Animal experiments further demonstrated that CCL18 promotes theproliferation of endometrial cancer cells in vivo through the CXCR4 receptor signaling pathway.
Conclusions LVSI is an independent predictor of relapse and poor prognosis in early-stage low-grade endometrioid endometrial cancer in the Chinese cohort. Importantly, we for the first time revealed the crucial role of CXCR4 receptor in CCL18-mediated endometrial cancer LVSI and metastasis and in vivo proliferation, identifying it as a potential therapeutic target for intervening CCL18's pro-tumorigenic effects in EEC.

PO_400

KLF10 通过调控胆固醇生物合成抑制子宫内膜癌增殖的机制研究

张雯雯 曲芃芃*
天津市中心妇产科医院

研究目的 子宫内膜癌（Endometrial Cancer, EC）的发生发展与胆固醇代谢异常存在紧密关联。本研究聚焦于转录因子 KLF10，深入探究其在子宫内膜癌胆固醇代谢过程中的调控作用及潜在分子机制，以期为临床治疗提供全新的潜在靶点。

材料与方法 首先利用胆固醇检测试剂盒，精准分析 EC 肿瘤组织与正常子宫内膜组织之间的胆固醇水平差异。同时，运用 β- 环糊精处理 EC 细胞系，通过检测细胞内胆固醇水平以及细胞增殖活性（采用 CCK8 实验），初步探究胆固醇对 EC 细胞的影响。

为了明确 KLF10 的调控作用，采用慢病毒介导的基因干扰技术，实现对 KLF10 的敲低或过表达操作。在此基础上，结合 qRT-PCR 和 Western blot 技术，检测胆固醇合成关键基因的表达变化。进一步通过 CHIP 实验和启动子活性分析，验证 KLF10 对靶基因的转录调控作用。此外，利用小鼠皮下成瘤实验，评估 KLF10 对肿瘤生长的体内影响，并深入探讨其与二甲双胍的协同治疗效果。

结果 EC 肿瘤组织中胆固醇水平显著高于正常组织，β- 环糊精能够有效降低 EC 细胞内胆固醇含量，并抑制细胞增殖；敲低 KLF10 显著下调 HMGCR、MVK 和 PMVK 的表达，同时抑制 EC 细胞增殖，而过表达 KLF10 则呈现相反效果；CHIP 实验证实 KLF10 直接结合于 HMGCR、MVK 和 PMVK 启动子区域的 KLF 元件（KLFE）；启动子活性检测也证实，KLF10 直接调控 HMGCR、MVK 和 PMVK 启动子转录活性；体内实验显示，KLF10 敲低组肿瘤生长速度显著快于对照组，而过表达 KLF10 联合二甲双胍可协同抑制肿瘤生长。

结果 KLF10 通过直接抑制胆固醇生物合成关键基因（HMGCR、MVK、PMVK）的转录，降低 EC 细胞内胆固醇水平，从而抑制肿瘤增殖。本研究揭示了 KLF10 在 EC 胆固醇代谢中的核心调控作用，为靶向治疗提供了新的理论依据。

PO_401

脂质作为子宫内膜癌患者缓解的可靠预测因子：机器学习模型的建立和验证

毕欣怡
北京大学人民医院

目的 本研究旨在构建一个血脂相关的评分系统（lipid score Model），将其加入到子宫内膜非典型增生（AEH）和早期子宫内膜癌（EC）完全缓解（CR）预测模型中以提高对子宫内膜病变保育患者完全缓解的预测情况。

方法 纳入北京大学人民医院 2012 年 1 月至 2025 年 01 月期间确诊的 349 例 AEH 或 EC 患者。 所有患者按 2:1 的比例随机分为训练组和验证组。 收集临床病理资料和血脂相关指标数据。 通过留-交叉验证（LOOCV）框架建立了 101 种预测模型，用于筛选 C 指数最高的组合。采用单变量和多变量 COX 分析确定预测完全缓解的候选因素，构建列线图。利用拟合图、生存曲线、受试者工作特征（ROC）曲线和决策曲线分析（DCA）来估计 lipid score 加入到预测模型中对 CR 预测准确性。

结果 LASSO 回归识别四个独立预测因素，包括甘油三酯（TG）、高密度脂蛋白（HDL）、胆固醇（Cholesterol）和低密度脂蛋白（LDL）（统称为 lipid），ROC 曲线分析表明，训练组、验证组和总人群组对 1 年、3 年和 5 年随访的 CR 有很好的预测能力。通过单变量和多变量 COX 分析筛选出四个

独立预测因素，包括脂质评分（lipid score）、代谢综合征（MetS）、CA125、病理结果，建立无 lipidscore 的模型 1，并在模型 2 中添加 lipidscore，整个样本的曲线下面积（AUC）值得到了显著提高。AUC 值从 0.842 增加到 0.894，在验证组和总人群组中也观察到了同样的表现。Kaplan-Meier（K-M） 生存曲线显示不同风险组之间的累积 CR 率存在显著差异，高危患者 CR 率低，低危患者 CR 率高。因此，加入 lipid score model 能有效地提高子宫内膜癌保育患者 CR 的预测情况。

结果 建立 lipid score model 显著提高了预测子宫内膜病变患者的 CR 的准确性，并有助于做出更好的临床决策。

PO_402

年轻子宫内膜癌及非典型增生患者生育力保护的治疗探索

吴迪
解放军总医院第一医学中心

目的 回顾性分析早期子宫内膜癌（EC）及子宫内膜非典型增生（AEH）患者经保留生育功能治疗后的疾病结局和妊娠结局，探索符合精准治疗理念的个体化最佳治疗方案。

方法 选取 2014 年 1 月至 2024 年 12 月就诊中国人民解放军总医院第一医学中心妇产科有保留生育力需求的子宫内膜病变患者 181 例，其中经宫腔镜技术辅助诊刮活检病理确认 EC 患者 17 例和 AEH 患者 164 例，统计分析其临床病理特征和随访资料。根据内膜病变逆转情况和病变病理分组，探索治疗结局的影响因素和建立风险因子回归模型。

结果 比较子宫内膜病变逆转情况，分析显示 BMI（P=0.014）、糖尿病（P=0.023）、血脂异常（P=0.048）、宫腔镜技术联合孕激素治疗方案（P=0.012）、是否坚持保育意愿（P<0.001）以及首次 CR 时间（P<0.001）均有显著差异；多元回归分析得到首次 CR 时间大于 6 个月（OR=27.138,95%CI:1.127-653.741）,未进行维持治疗（OR=10.722,95%CI:1.397-82.298），治疗过程不规范（OR=20.542,95%CI:2.041-206.703）均为影响治疗结局的独立危险因素；对于子宫内膜不典型增生和早期子宫内膜癌保育疗效无显著差异（P>0.05）；迫切保育需求（P<0.001）、治疗依从性（P<0.001）以及孕激素与宫腔镜技术的联合方案（P=0.009）显著影响子宫内膜不典型增生患者的妊娠结局。

结论 年轻子宫内膜癌及非典型增生患者应用宫腔镜技术联合孕激素的个体化治疗方案，在实现精准治疗的同时有效保护生育功能，既保留了器官完整性又提高了患者生活质量。

PO_403

益元汤在子宫内膜癌中作用的初探

张晓玉 高超*
天津医科大学总医院

目的 子宫内膜癌（EC）作为女性生殖系统最常见的恶性肿瘤之一，其发病率和死亡率逐年上升，尤其在代谢异常人群中风险显著增加。尽管传统治疗手段（如手术、放疗和化疗）改善了早期患者的预后，但晚期或复发性 EC 的 5 年生存率仍不足 20%，其中多药耐药（MDR）是治疗失败的主要原因。肿瘤细胞通过多种机制（如药物外排泵上调、DNA 修复通路激活或凋亡抑制）产生耐药性，而传统单靶点药物难以全面应对。中医药因其多靶点、多途径的作用方式，在逆转 MDR 方面展现出显著潜力。本研究旨在探讨传统中药复方益元汤通过调控 PI3K-Akt 信号通路逆转 EC 多药耐药的具体分子机制，为临床提供新的治疗策略。

方法 本研究基于前期网络药理学和分子对接分析，筛选益元汤的活性成分及其作用靶点，并通过构建 EC 顺铂耐药细胞模型（Ishikawa 细胞系）进行实验验证。采用梯度浓度递增法诱导耐药株，检测耐药

相关指标（如 IC50 和 p-Akt 水平）。通过 Western blot 等技术分析益元汤对 PI3K-Akt/mTOR 通路关键蛋白（如 PI3Kα/β、PTEN、Akt、mTOR）的调控作用。此外，结合分子对接技术验证益元汤活性成分（如桑休苷、异塔拉定）与靶蛋白的结合能力，阐明其多成分-多靶点的协同作用机制。
结果 前期研究表明，益元汤中的 152 个活性成分可靶向 697 个 EC 相关基因，其中 PI3K-Akt 信号通路是核心调控通路之一。分子对接分析显示，桑休苷等关键成分与 Akt 等靶点具有较强结合亲和力。实验证实，益元汤可显著抑制耐药细胞中 PI3K-Akt 通路的过度激活（p-Akt 水平较对照组降低），并逆转顺铂耐药性（耐药株 IC50 降低）。此外，网络药理学分析揭示益元汤通过多靶点（如 PI3Kα/β、PTEN、mTOR）协同调控 PI3K-Akt 通路，从而抑制肿瘤细胞增殖、促进凋亡并改善耐药性。
结论 益元汤通过多成分-多靶点的协同作用，有效调控 PI3K-Akt/mTOR 信号通路，逆转 EC 的多药耐药性。其机制包括抑制 Akt 磷酸化、增强 PTEN 活性及调控下游 mTOR 信号，从而克服单一靶点药物的局限性。本研究为益元汤的临床应用提供了理论依据，并为其作为 EC 辅助治疗药物的开发奠定了基础。未来需通过临床试验进一步验证其疗效，推动中医药在肿瘤耐药治疗中的应用，改善患者预后并降低医疗负担。

PO_404

Association between metabolic disorders and clinicopathologic features in endometrial cancer

王 元培
郑州大学第一附属医院

Background In recent years, the incidence of Endometrial cancer (EC) has been on the rise due to high-fat, high-calorie diets and low-exercise lifestyles. However, the relationships between metabolic disorders and the progression of EC remain uncertain. The purpose of our study was to explore the potential association between obesity, hypertension, hyperglycemia and clinicopathologic characteristics in EC patients.
Methods In categorical variables, Chi-square tests were used to calculate P values. Univariate logistic regression and multivariate logistic regression were used to identify the risk factors of myometrial invasion>1/2 and lymph node metastasis. Overall survival (OS) was estimated using the Kaplan-Meier method.
Results The study included 406 individuals with EC, 62.6% had type I and 37.4% had type II. Hypertension was seen in 132 (32.5%), hyperglycemia in 75 (18.5%), and overweight or obesity in 217 (53.4%). Hypertension, hyperglycemia, and obesity are strongly associated with the clinicopathologic features of EC. Multivariate logistic regression revealed that hyperglycemia (OR=2.439,95% CI: 1.025-5.804, P = 0.044) was a risk factor for myometrial invasion depth >1/2 in patients with type I EC, and hypertension (OR=32.124,95% CI: 3.287-313.992, P = 0.003) was a risk factor for lymph node metastasis in patients with type I EC. Survival analysis found that hyperglycemia (P < 0.001) and hypertension (P = 0.002) were associated with OS in type I EC. Neither hyperglycemia, hypertension, nor obesity were associated with the prognosis in type II EC.
Conclusion Hyperglycemia was a risk factor for myometrial invasion depth >1/2 in patients with type I EC and hypertension was a risk factor for lymph node metastasis in patients with type I EC. Hypertension and hyperglycemia were associated with poor prognosis in patients with type I EC.

PO_405

术后不同辅助治疗方式对子宫浆液性癌患者生存预后影响的分析

王元培 wangyuanpei
郑州大学

研究背景 子宫内膜浆液性癌（Uterine Serous Carcinoma，USC）是一种罕见的子宫 内膜癌病理亚型，虽然它仅占子宫内膜癌的 10%，但它导致了子宫内膜癌相关性死亡的 39%。本研究拟纳入 SEER 数据库和本医院的 USC 患者，研究术后观察、化疗和放化疗对 USC 患者预后的影响。**方法** 通过访问 SEER 数据库及本医院的临床病例系统收集 USC 患者临床病理信息。将患者分为观察组、化疗组、放化疗组，使用 SPSS26.0 软件对数据进行统计和分析。**结果** 1. IA 期 USC 患者 SEER 数据库中观察组、化疗组、放化疗组的 3 年生存率 （Overall Survival，OS）分别为 85.5%、90.5%、91.9%，本医院为 100%、88.9%、 92.9%。SEER 数据库的结果显示在无肌层浸润的 IA 期（n=580）患者中，观察组、化疗组和放化疗组的 OS 无明显差异，而在存在肌层浸润的 IA 期 （n=512）患者中，与观察相比，化疗（P=0.002）和放化疗（P＜0.001）能改善患者的 OS。 2. IB-II 期 USC 患者 SEER 数据库中观察组、化疗组、放化疗组的 3 年 OS 分别为 55.9%、73.7%、75.4%，本医院为 60%、91.7%、91.7%。对于 IB-II 期 USC 患者，与观察相比，化疗（SEER 数据库 P＜0.001，本医院 P=0.028）和放化疗（SEER 数据库 P＜0.001，本医院 P=0.038）能够改善患者的 OS。但放化疗和单独化疗的 OS 无明显差异。3. III-IV 期 USC 患者 SEER 数据库中观察组、化疗组、放化疗组的 3 年 OS 分别为 27.2%、41.1%、60.8%，本医院为 33.3%、41.7%、77.8%。与观察相比，III-IV 期 USC 患者术后化疗（SEER 数据库 P＜0.0001，本医院 P=0.026）和放化 疗（SEER 数据库 P＜0.0001，本医院 P＜0.0001）能显著改善患者的 OS。放化疗（SEER 数据库 P＜0.0001，本医院 P=0.015）对于 III-IV 期 USC 患者 OS 的改善优于单独化疗。**结论** 1. 对于非浸润性 IA 期 USC 患者，观察组、化疗组和放化疗组的 OS 无明显 差异。 2. 对于浸润性 IA 期 USC 患者、IB-II 期 USC 患者，与观察相比，化疗和放 化疗能改善患者的 OS，但放化疗与单独化疗的 OS 无明显差异。 3. 对于 III-IV 期 USC 患者，与观察相比，化疗和放化疗能够改善患者的 OS， 且放化疗的疗效优于单独化疗。 4. 影响 USC 患者预后的其他因素包括年龄、肌层浸润、腹水细胞学阳性、 腹主动脉淋巴结切除、FIGO 分期。

PO_406

TP53 突变型子宫内膜癌术后辅助治疗的分期导向生存分析：基于 TCGA 数据库的队列研究

宿晓曼 王宁* 黄家珍 魏威 孙银睿 牛志刚
大连医科大学附属二院

目的 评估含铂化疗在根治术后 TP53 突变型子宫内膜癌不同分期中的生存价值，明确同步放化疗（CCRT）的临床地位。
方法 回顾性纳入TCGA数据库（2010-2015年）543例根治术后TP53突变患者（FIGO 2009分期）。按辅助治疗分组：单纯手术（S组，n=107）；手术+放疗（S+RT组，n=142）； 手术+含铂化疗≥4周期（S+CT 组，n=165）；手术+同步放化疗（S+CCRT 组，n=129），主要终点为总生存期（OS）。采用 Kaplan-Meier 法及分层 Log-rank 检验进行生存比较，多因素 Cox 模型校正年龄、分级及 MMR 状态。通过似然比检验分析化疗效益的分期异质性（检验水准 α=0.05）。
结果 1. III 期（n=198） S+CCRT 组中位 OS 54 个月（95%CI 49-60），对比 S+RT 组死亡风险降低 58%（HR 0.42, P<0.001）。 S+CCRT 较 S+CT 组降低 24%死亡风险（HR 0.76, P=0.028）、对比分

段放化疗（化疗后间隔>4 周放疗），同步方案额外降低 38%死亡风险（HR 0.62, P=0.004）2. IV 期（n=147）。含铂化疗组（S+CT/S+CCRT）中位 OS 25 个月（95%CI 23-29），较无化疗组（S/S+RT）死亡风险降低 51%（HR 0.49, P<0.001）。S+CCRT 与 S+CT 组生存无差异（HR 0.96, P=0.70）。3. I-II 期（n=198）。S+CT 组对比 S+RT 组 OS 未改善（HR 0.86, P=0.29）
结果 III 期：术后同步放化疗显著延长生存（中位 OS 54 个月），应作为标准方案； IV 期：含铂化疗是生存基石（HR 0.49），但增加同步放疗无获益； I-II 期：含铂化疗未改善 OS，辅助放疗价值需结合高危病理评估；TP53 突变型子宫内膜癌的化疗效益呈现明确分期梯度（III 期>IV 期>I-II 期）。

PO_407

经脐单孔腹腔镜腹膜外入路腹主动脉旁淋巴结切除在子宫内膜癌分期术中的应用

陈川 转胜男 陈纲*
中国科学技术大学附属第一医院（安徽省立医院）

目的 探讨经脐单孔腹腔镜腹膜外入路腹主动脉旁淋巴结切除术在子宫内膜癌全面分期术的可行性及安全性。
方法 回顾性分析中国科学技术大学附属第一医院妇科 2023 年 6 月至 2024 年 12 月，采用经脐单孔腹腔镜腹膜外入路下切除腹主动脉旁淋巴结的 10 例子宫内膜癌分期术临床资料。
结果 10 例子宫内膜癌手术方式均为筋膜外全子宫切除+双侧附件切除+腹膜后淋巴结清扫术，其中腹主动脉旁淋巴结均为经脐单孔腹腔镜腹膜外入路，手术均获成功。10 例患者中，年龄（58.00±5.56）岁，BMI（24.97±5.93）kg/m2，术前 CA125 水平（23.31±12.85）U/ml，术前病理：浆液性癌 1 例，透明细胞癌 2 例，腺鳞癌 1 例，子宫内膜样腺癌 6 例，FIGO 组织学分级 Ⅱ级 4 例，Ⅲ级 6 例；分子分型：6 例高拷贝数型，2 例 MSI-H 型，2 例低拷贝数型。
手术时间（320.29±82.61）min，术中出血量 100（50，275）ml，腹主动脉旁淋巴结阳性数 0（0，1.5）/总数（10.57±4.86），骶前淋巴结阳性数 0（0，0.25）/总数（3.29±1.70），左侧盆腔淋巴结阳性数 0（0，1.25）/总数（6.86±3.89），右侧盆腔淋巴结阳性数 0（0，0.5）/总数（10.71±6.96），尿管均术后第一天拔除，肛门排气时间 36（35.4，45）h，引流管拔除时间（40.08±9.54）h，其中 3 例患者术后关闭后腹膜切口，未放置引流管。住院时间（11.00±2.58）d。所有患者中，1 例出现肌间静脉血栓，1 例出现乳糜瘘，2 例出现术后感染，术后临床病理分期 IA 期 5 例，IIC 期 1 例，IIIB 期 1 例，IIIC 期 2 例，IV 期 1 例，5 例术后补充放化疗，术后 1 个月随访，阴道顶端无裂开，脐部切口愈合良好。
结果 具备娴熟单孔腹腔镜手术经验的主刀医生，实施经脐单孔腹膜外入路腹主动脉旁淋巴结切除在子宫内膜癌全面分期术中安全可行。

PO_408

肥胖与子宫内膜癌发生相关性的 meta 分析

陈红梅
石河子大学

研究目的 通过 meta 分析系统评价肥胖与子宫内膜癌发病风险的关系，探讨子宫内膜癌的危险因素。
材料与方法 计算机检索 CBM、知网、维普、万方、Pubmed、Embase、Web of Science 、Cochrane 等数据库及相关参考文献，检索时间为建库至 2025 年 6 月，共检索到 3280 篇。

纳入标准：①研究设计为病例对照研究；病例为经病理诊断的子宫内膜癌患者，对照为社区人群或同一医院的排除子宫肌瘤及其他恶性肿瘤的健康女性。②文献所提供的数据可以转化为 OR 值及 95%CI。③研究因素为肥胖（BMI ≥ 28kg/m²）。④结果 主要关注的结果是子宫内膜癌的发生率。⑤有开展研究的地点和时间。
排除标准：①不提供肥胖与子宫内膜癌之间关联的原始数据的干预研究、综述、荟萃分析、病例报告、信件或社论。②发表在非同行评审期刊上的研究③无法获取全文、质量较差、数据不完整的研究。
根据制定的纳入/排除标准筛选文献，共纳入 8 篇文献，并进行质量评价，提取肥胖与子宫内膜癌发病风险相关的数据。应用 Stata16.0、Review Manager5.3 统计软件采用随机或固定效应模式对数据进行合并分析。
结果 共纳入 8 篇相关文献（中英文各 4 篇），包括 17427 例患者，显示肥胖与子宫内膜癌发病风险显著相关，合并 OR 值为 2.32（95%CI：1.62-3.31），提示肥胖女性患子宫内膜癌的风险是正常女性的 2.32 倍。异质性检验表明研究间存在中度异质性（ I2=65.5％），故采用随机效应模型。
结论 肥胖是子宫内膜癌发病的重要危险因素，两者呈正相关。

PO_409

雌激素通过调节脂代谢促进子宫内膜癌发生的机制研究：Meta 分析

王伟娜 刘洁 单伟伟 陈晓军*
上海市第十人民医院

研究目的 子宫内膜癌是女性生殖系统最常见的恶性肿瘤之一，发病率呈上升趋势。雌激素在子宫内膜癌发生发展中起关键作用，脂代谢异常被认为是连接雌激素与子宫内膜癌发生的重要桥梁。本 Meta 分析旨在系统评价雌激素通过调节脂代谢促进子宫内膜癌发生的分子机制，探讨脂代谢相关生物标志物的临床价值。
材料与方法 系统检索 PubMed、Embase、Web of Science、中国知网等数据库至 2024 年 12 月，检索词包括"雌激素"、"脂代谢"、"子宫内膜癌"等。纳入标准：研究雌激素与脂代谢在子宫内膜癌中作用的原创性研究，包含明确的实验组和对照组，提供充分统计学数据。排除会议摘要、综述和重复发表研究。由两名研究者独立筛选文献、提取数据并评估质量。采用 RevMan 5.4 软件进行统计分析，计算合并效应量和 95%置信区间。
结果 最终纳入 32 项研究，涉及 18,756 例受试者。Meta 分析结果显示：雌激素水平升高显著增加子宫内膜癌发生风险（OR=2.34, 95%CI: 1.89-2.91, P<0.001）；脂代谢异常患者子宫内膜癌发生风险增加 1.76 倍（OR=1.76, 95%CI: 1.52-2.03, P<0.001）；雌激素通过激活 SREBP-1c/FASN 信号通路促进脂肪酸合成，FASN 表达在子宫内膜癌组织中较正常组织增加 2.8 倍（SMD=2.81, 95%CI: 2.34-3.28, P<0.001）；雌激素上调 HMG-CoA 还原酶表达促进胆固醇合成，胆固醇水平与子宫内膜癌分级呈正相关（r=0.45, 95%CI: 0.32-0.56, P<0.001）；脂代谢关键酶 ACLY、ACC1 在雌激素刺激下表达显著上调；绝经后女性中雌激素-脂代谢-子宫内膜癌关联性更显著。
结论 本 Meta 分析证实雌激素通过多重机制调节脂代谢促进子宫内膜癌发生发展。雌激素主要通过激活 SREBP-1c/FASN 和 HMG-CoA 还原酶等关键信号通路，促进脂肪酸和胆固醇从头合成，为癌细胞增殖提供必需的脂质成分和能量支持。脂代谢重编程不仅满足癌细胞快速增殖的代谢需求，还通过膜脂质组成改变、信号分子合成等方式直接参与肿瘤恶性转化。FASN、ACLY、ACC1 等脂代谢关键酶可作为潜在的诊断标志物和治疗靶点。临床上应重视雌激素水平和脂代谢状态的监测，特别是对高危人群的筛查。未来研究应进一步探索雌激素-脂代谢轴的精确调控网络，开发针对脂代谢的靶向治疗策略。

PO_410

The Cholesterol-Hormone-Immune Axis in Endometrial Carcinogenesis: Metabolic Reprogramming, Microenvironment Crosstalk, and Therapeutic Implications

Danhua Lu Xiaojun Chen* Jie Liu Weina Wang Weiwei Shan Yuanyuan Fu Xiaowen Shao Yifei Wang Xingran Wang
Shanghai Tenth People's Hospital

Endometrial cancer (EC) pathogenesis involves a complex interplay between dysregulated cholesterol metabolism, aberrant steroid hormone signaling, and the establishment of an immunosuppressive tumor microenvironment (TME). This review synthesizes current evidence elucidating the mechanisms underlying this cholesterol-hormone-immune axis. Key areas of focus include: (1) How hypercholesterolemia provides abundant precursors, fueling local intratumoral estrogen biosynthesis; (2) The role of tumor-intrinsic cholesterol accumulation in facilitating immune evasion; (3) Tissue-specific dysregulation of steroidogenic enzymes within the endometrium during carcinogenesis; (4) Mechanisms of hormone-driven immunosuppression within the TME; and (5) Emerging therapeutic strategies designed to target the critical crosstalk between metabolic reprogramming and immune dysfunction. This comprehensive analysis underscores the centrality of the cholesterol-hormone-immune axis in EC development and progression, highlighting novel avenues for understanding metabolic vulnerabilities and developing effective, targeted interventions.

PO_411

Cross-talk between endometrial cancer and polycystic ovary syndrome: The role and molecular mechanism of quercetin in inhibiting co-expressed genes via AKT

Lei Han[1] Mengyuan Li[2*]
1.Department of Reproductive Medicine, Binzhou Medical University Hospital
2.Department of Obstetrics and Gynecology, Southwest Hospital, Third Military Medical University

Objectives Endometrial cancer (EC) is a prevalent gynecological malignancy, and polycystic ovarian syndrome (PCOS) has been recognized as a significant risk factor for its development. Quercetin, a naturally occurring flavonoid widely distributed in nature, has shown promising therapeutic potential in the management of both PCOS and EC. However, the specific molecular targets through which quercetin exerts its effects in the context of PCOS comorbid with EC (PCOS-EC) remain largely unclear. This study seeks to explore the therapeutic potential of quercetin in treating PCOS-EC.
Materials and Methods The study employed network pharmacology, molecular dynamics simulations, and in vitro assays. Specifically, an intersection analysis was conducted between 379 PCOS-EC-associated targets and 361 quercetin targets. Gene Ontology enrichment analysis was used to reveal relevant biological functions, and the Kyoto Encyclopedia of Genes and Genomes was applied to identify pathways involved in quercetin's effects against PCOS-EC. Protein-protein interaction network analysis was performed to screen key targets, while molecular docking and molecular dynamics simulations were used to assess the binding affinity between quercetin and hub genes. In vitro experiments were conducted to verify the regulatory effect of quercetin on target gene expression in EC cells.
Results Through the intersection analysis, 47 potential therapeutic targets of quercetin for PCOS-EC were identified. Protein-protein interaction network analysis highlighted six overlapping targets: ACTB, AKT1, EGFR, ESR1, PTGS2, and TP53. Molecular docking and molecular dynamics simulations demonstrated that quercetin binds with high affinity to these hub genes, among which AKT1 emerged

as a central target. In vitro experiments further confirmed that quercetin treatment significantly downregulated AKT expression in EC cells.
Conclusion These findings elucidate the potential targets and molecular mechanisms underlying the therapeutic effects of quercetin in PCOS-EC. From a translational medicine perspective, they lay a foundation for the clinical translation of quercetin in the prevention and treatment of PCOS-EC. The identification of AKT1 as a key target provides a promising candidate for the development of targeted therapies, and the validation of quercetin's regulatory effect on AKT expression offers a potential avenue for designing combination treatment strategies in clinical practice, bridging the gap between basic research and clinical application.

PO_412

错配修复缺陷型子宫内膜癌及子宫内膜不典型增生患者保育治疗的肿瘤和生育结局

张天宇[1] 杨洁[1*] 曹冬焱[1] 吴焕文[2] 杨佳欣[1]
1.中国医学科学院北京协和医院 妇产科 2.中国医学科学院北京协和医院 病理科

研究目的 探讨错配修复缺陷型（MMRd）子宫内膜癌（EC）及子宫内膜不典型增生（AEH）患者接受保留生育功能治疗后的肿瘤及生育结局，为该亚型患者的临床管理提供参考。
材料与方法 回顾性分析 2020 年 1 月至 2023 年 12 月间北京协和医院收治的 14 例接受保留生育功能治疗的 MMRd 型 EC 及 AEH 患者的临床病理信息，总结其肿瘤治疗结局和生育结果。
结果 患者平均诊断年龄 33.6 岁，免疫组化中 MLH1/PMS2 联合缺失最为常见，其次为 MSH6 缺失、MSH2/MSH6 联合缺失和 PMS2 缺失。基因检测发现 4 例患者存在 MMR 基因致病/可能致病胚系变异。在 19 个月的中位随访期间，7 例（50%）患者达到完全缓解，从治疗开始达到完全缓解的中位时间为 8 个月；复发率为 57.1%，中位复发时间 11 个月。9 例患者在保育失败或复发后进行切除性手术治疗，其中 8 例术后病理较保育治疗前升级，显示出更高的分级或子宫外转移。仅 1 例患者通过辅助生殖成功妊娠并生育。
结果 MMRd 型患者接受保留生育功能治疗的缓解率较低、复发率较高，生育结局不佳。对有生育需求的患者需对肿瘤风险及妊娠可能性进行充分知情，如仍坚持保育治疗应加强监测。

PO_413

FOXA2 介导的脂质代谢重编程增强子宫内膜癌孕激素治疗敏感性的机制研究

刘洁
北京大学人民医院

目的 子宫内膜癌（EC）是常见的妇科恶性肿瘤，孕激素治疗是保留生育功能的重要手段，但部分患者对孕激素治疗产生耐药。本研究旨在探索转录因子 FOXA2 在子宫内膜癌中的作用机制，并评估脂肪酸合成抑制剂奥利司他（Orlistat）通过 FOXA2-PR 调控轴增强孕激素治疗敏感性的潜力。
材料与**方法** 采用转录组测序分析 FOXA2 过表达对 Ishikawa 细胞基因表达谱的影响。通过 CCK-8、流式细胞术、Transwell 等实验评估 FOXA2 对细胞增殖、凋亡、迁移和侵袭的影响。利用 BODIPY 染色、ChIP-seq 和 ChIP-qPCR 技术研究 FOXA2 对脂质代谢基因的调控。构建 FOXA2 敲低和 PR 过表达的稳定细胞系进行功能挽救实验。建立 B-NDG 小鼠皮下移植瘤模型，评估奥利司他联合醋酸甲羟孕酮（MPA）的治疗效果。

结果 FOXA2 过表达导致 809 个基因上调和 422 个基因下调，功能富集分析显示其抑制细胞增殖、转移相关通路。FOXA2 过表达显著增强 G0/G1 期阻滞和细胞凋亡，抑制细胞增殖、迁移和侵袭能力。在 TCGA 队列中，FOXA2 高表达与良好预后相关。机制研究发现，FOXA2 直接结合并调控脂肪酸代谢基因（ACOX1、ECHS1、HADHA 等）的启动子，促进脂肪酸氧化同时抑制脂肪酸合成。奥利司他处理可上调 FOXA2 表达并减少其 Thr156 位点磷酸化，进而增强 PR 表达。在 MPA 耐药的 FOXA2 敲低细胞中，PR 过表达可恢复 MPA 敏感性。体内实验证实，奥利司他与 MPA 联合治疗显著抑制肿瘤生长，但该协同效应在 FOXA2 敲低的肿瘤中变弱。

结果 本研究揭示了 FOXA2 通过重编程脂质代谢发挥肿瘤抑制作用的新机制。奥利司他通过稳定 FOXA2 蛋白并上调其表达，增强 PR 表达水平，从而提高子宫内膜癌对孕激素治疗的敏感性。FOXA2-PR 调控轴是维持孕激素敏感性的关键通路。本研究为克服子宫内膜癌孕激素耐药提供了新的治疗策略，奥利司他作为 FDA 批准的药物，具有良好的临床转化前景。

PO_414

Engineered exosomes with RNA Motif-Enhanced shRNA Loading and Rab4-Driven Amplified Production for Controlled Ferroptosis Induction in Endometrial Carcinoma

zhang jiarui Hongbo Wang*
Union Hospital, Tongji Medical College, Huazhong University of Science and Technology

Research Purpose Endometrial cancer (EC) remains a significant therapeutic challenge, particularly in advanced or recurrent cases where conventional treatments exhibit limited efficacy and high systemic toxicity. Chemotherapy and radiation, the mainstays of current management, often fail to control disease progression in these scenarios while causing severe side effects that compromise patients' quality of life. Although exosome-based RNA interference (RNAi) offers a promising avenue for targeted therapy, with the potential to deliver genetic payloads directly to tumor cells, clinical translation has been hampered by several critical limitations. These include inefficient cargo loading, which reduces the therapeutic potency of each exosome; low production yields, making large-scale manufacturing impractical; and insufficient tumor specificity, which increases the risk of off-target effects. To overcome these barriers, we developed a multi-functional exosome platform engineered to deliver shRNAs targeting key ferroptosis regulators—GPX4, FSP1, and FTH—with high precision. Ferroptosis, a form of iron-dependent lipid peroxidation-induced cell death, has emerged as a promising therapeutic target in EC, as tumor cells often rely on these regulators to evade this process. Our goal was to achieve three critical milestones: (1) enhance exosomal cargo loading efficiency to ensure sufficient delivery of therapeutic shRNAs, (2) amplify exosome production and tumor cell uptake to maximize the number of exosomes reaching their target, and (3) confer tumor-selective targeting to minimize damage to healthy tissues, ultimately enabling potent ferroptosis induction while minimizing off-target effects.

Materials and Methods Our approach combined three synergistic engineering strategies to optimize exosome performance. First, to address the issue of inefficient cargo loading, we designed RNA motifs (EXOmotifs) into shRNAs targeting the key ferroptosis regulators GPX4, FSP1, and FTH. These motifs were specifically chosen to interact with endogenous RNA sorting machinery, such as the RNA-binding protein hnRNPA2B1, which is known to facilitate the incorporation of RNAs into exosomes. This design aimed to leverage the cell's natural processes to enhance the loading of therapeutic shRNAs into exosomes beyond what is achievable with conventional methods. Second, to tackle both low production yields and poor uptake, we implemented shRNA-mediated Rab4 silencing in both producer and recipient cells—a dual strategy with complementary effects. In producer cells, Rab4 silencing disrupted endosomal recycling, a process that normally limits the number of exosomes secreted by diverting endosomal vesicles back to the cell membrane. By interfering with this recycling, we sought

to increase the number of endosomes that mature into exosomes and are released into the extracellular space. Concurrently, in recipient EC cells, Rab4 knockdown impaired early endosome maturation, which we hypothesized would slow the degradation of incoming exosomes and enhance their uptake by keeping them in a state where they can release their cargo into the cytoplasm. Third, to ensure tumor specificity, we functionalized exosome surfaces with cRGD peptides, small cyclic molecules that have a high affinity for αvβ3 integrins. These integrins are overexpressed in EC tumors and their associated vasculature, providing a unique molecular marker for targeted delivery. For evaluation, we systematically characterized the engineered exosomes using a range of techniques: qPCR and Western blot to verify the efficiency of cargo loading and the extent of target gene knockdown; Nanoparticle Tracking Analysis to quantify exosome production yields and assess their size distribution; Confocal microscopy and flow cytometry to visualize and quantify tumor cell uptake, respectively, and determine the specificity of this uptake compared to non-tumor cells; Lipid peroxidation assays, using fluorescent probes sensitive to lipid peroxides, and cell viability tests to measure the induction of ferroptosis; and Xenograft mouse models, where human EC cells were implanted into immunocompromised mice, to evaluate the therapeutic efficacy in terms of tumor growth inhibition and assess safety through histological analysis of major organs.

Results Our engineered exosome platform demonstrated significant improvements across all design parameters. The EXOmotif-mediated loading strategy proved highly effective, achieving more than 80-fold higher shRNA encapsulation efficiency compared to conventional methods such as electroporation or passive incubation. This marked increase was consistent across multiple experiments, confirming the reliability of the approach. The dual Rab4 silencing approach also delivered on its promise, synergistically enhancing both production and delivery. In producer cells, we observed a 1.8-fold increase in exosome yield, as measured by Nanoparticle Tracking Analysis, which translated to a greater number of therapeutic vehicles available for delivery. In recipient EC cells, Rab4 knockdown led to a 2.1-fold improvement in tumor cell uptake, as quantified by flow cytometry, indicating that more exosomes were successfully entering the target cells. Functionally, the engineered exosomes exerted a strong biological effect: they effectively downregulated the expression of GPX4, FSP1, and FTH, as shown by Western blot and qPCR analyses, leading to significantly elevated levels of lipid peroxidation—a hallmark of ferroptosis—and robust induction of this cell death pathway in EC cells, as evidenced by reduced cell viability in vitro. In vivo studies in the xenograft mouse models further validated the platform's therapeutic potential. Treated mice showed potent tumor growth inhibition, with a reduction in tumor volume that was significantly greater than in control groups receiving either non-engineered exosomes or vehicle alone. Importantly, there was no observable systemic toxicity, as indicated by normal body weight gain and the absence of histological abnormalities in major organs such as the liver, kidney, and heart. Additionally, the platform maintained structural integrity throughout the engineering process, with exosomes retaining their characteristic size and morphology as assessed by transmission electron microscopy, and showed excellent scalability potential for clinical translation, with consistent production yields even when scaled up in larger culture systems.

Conclusion We developed a modular exosome platform that integrates RNA motif-driven cargo loading, Rab4-mediated bioproduction optimization, and αvβ3-integrin-targeted delivery to overcome the key hurdles of RNAi-based therapy for EC. This system addresses the critical limitations that have hindered the clinical development of exosome-based therapeutics, not only enhancing their scalability—through increased production yields—and specificity—via targeted delivery—but also unlocking the clinical potential of ferroptosis induction in EC. By achieving spatiotemporal control over RNAi delivery, ensuring that therapeutic shRNAs reach the right cells at the right time, our work bridges the gap between the mechanistic precision of basic research and the translational feasibility required for clinical application. This platform thus offers a blueprint for next-generation exosome engineering in oncology, with potential applications beyond EC to other cancer types that overexpress αvβ3 integrins or rely on ferroptosis regulators for survival. Future studies will focus on optimizing clinical-scale manufacturing processes to ensure consistent quality and efficacy, as well as exploring combinatorial strategies—such as combining the engineered exosomes with chemotherapy or immunotherapy—to further improve therapeutic outcomes and overcome potential mechanisms of

resistance. Ultimately, this research represents a significant step forward in the development of targeted, effective, and safe therapies for patients with advanced or recurrent EC.

PO_415

The features and prognostic value of ARID1A mutation in endometrial cancer of no specific molecular profile (NSMP) subtype

Xinhui Yuan Mengyuan Cai Yulan Ren*
Fudan University Shanghai Cancer Center

Objective Endometrial cancer (EC) of no specific molecular profile (NSMP) subtype constitutes the majority of EC and shows significant heterogeneity. This study aims to explore the features and prognostic role of *ARID1A* in NSMP EC patients.
Methods. A retrospective analysis was conducted on patients who underwent primary surgery and next-generation sequencing (NGS) at the Fudan University Shanghai Cancer Center (FUSCC) between March 2020 and December 2023. The staging was determined according to 2023 FIGO staging and the molecular classification was conducted by the results of NGS. And the immunohistochemistry (IHC) for ARID1A was performed on the sections of endometrial cancer among these patients. Clinicopathologic features, *ARID1A* mutation and protein expression, and progression-free survival (PFS) were analysed in our study.
Results *ARID1A* mutations were identified in 49.0% (74/151) patients, with 85.1% (63/74) of them harboring loss-of-function (LOF) mutations and 27.0% (20/74) having multiple mutations. Compared to patients with *ARID1A* wild-type, those with *ARID1A* mutations were older (p=0.032) and had higher staging (p=0.022). The mutations of *PTEN* (79%), *PIK3CA* (53%), *CTNNB1* (27%) and *KRAS* (23%) were also observed in 151 NSMP EC patients. Aberrant PI3K pathway was more likely to occur in patients with *ARID1A* mutations (p=0.006). Of the 67 NSMP patients available for IHC, ARID1A positive expression was found in 82.1% (55/67) cases, including *ARID1A*-mutation tumors in 41.8% (23/55), and ARID1A negative expression was observed in 17.9% (12/67) tumors, all of which were complete loss expression, including *ARID1A*-mutation tumors in 91.7% (11/12) and *ARID1A*-wildtype tumors in 8.3% (1/12). The status of ARID1A protein expression was significantly associated with *ARID1A* mutation status (p=0.002). In all tumors with ARID1A positive expression, 69.1% (38/55) tumors were intact expression, heterogeneous partial diminution and diffuse partial diminution were found in 21.8% (12/55) and 9.1% (5/55) tumors respectively. Patients with *ARID1A* mutations were more likely to have a geographic pattern of ARID1A protein expression, while *ARID1A* wild-type tended to show a 'checkerboard' staining pattern. *ARID1A* mutation was associated with poorer PFS (Log-rank p=0.019; univariate: HR 8.86 (95% CI 1.03 to 76.41), p=0.047). Besides, PIK3CA hotspot mutations (p.E542K, p.E545K and p.H1047R) were associated with shorter survival in NSMP EC patients (Log-rank p=0.022), especially together with *ARID1A* mutations (Log-rank p=0.013). Additionally, ER and/or PR negative was associated with poorer prognosis in NSMP EC patients (Log-rank p=0.011), especially together with ARID1A mutations (Log-rank p<0.001).
Conclusion ARID1A mutations frequently occured in NSMP subtype, which was associated with loss of protein expression and shorter PFS. PI3K pathway was the most affected signaling pathway in NSMP EC patients with *ARID1A* mutations. Therefore, *ARID1A* gene might serve as a potential and critical predictive biomarker to subclassify NSMP EC patients and provide precise therapies.

PO_416

抑制 PRMT5 促进异常 R-loop 累积诱导 ARID1A 突变子宫内膜癌合成致死的机制研究

舒湾 董科均 沈小语 汪宏波*
华中科技大学同济医学院附属协和医院

研究目的 子宫内膜癌（EC）是女性生殖系统常见的恶性肿瘤，早期患者预后较好，但晚期患者 5 年生存率不足 20%，亟需新的治疗策略。ARID1A 作为染色质重塑复合体 SWI/SNF 的关键亚基，是子宫内膜癌中最常见的突变基因之一(30-40%)，这为靶向 ARID1A 突变型子宫内膜癌的合成致死治疗提供了潜在突破口。本研究旨在通过高通量药物筛选鉴定 ARID1A 突变子宫内膜癌的合成致死靶点，并阐明其分子机制，为临床治疗提供新思路。

材料与方法 通过筛选高通量化合物发现 PRMT5 抑制剂 JNJ-64619178 有效诱导 ARID1A 缺失子宫内膜癌细胞合成致死。通过 RNA-seq，彗星实验、免疫荧光及 Dot-blot 实验探究 PRMT5 抑制诱导 ARID1A 缺失子宫内膜癌细胞 DNA 损伤和 R-loop 积累。通过 IP-MS、CO-IP 及邻位连接实验检测 PRMT5-DHX9-R-loop 轴之间的相互作用及机制。进行 ChIP 实验以确认 ARID1A 直接调控 PRMT5 的转录。通过构建子宫内膜癌异种小鼠模型和类器官模型进一步验证 PRMT5 抑制与 ARID1A 缺失之间的合成致死效应。

结果 在本研究中，基于高通量药物筛选，我们鉴定出 PRMT5 抑制剂 JNJ-64619178 对 ARID1A 缺失子宫内膜癌细胞具有显著合成致死效应。shRNA 敲低 PRMT5 可抑制 ARID1A 缺陷子宫内膜癌细胞的增殖能力，并且 PRMT5 抑制促进 ARID1A 失活的子宫内膜癌 DNA 损伤、细胞凋亡、转录复制冲突及 R-loop 积累。这种合成致死性在 ARID1A 失活子宫内膜癌小鼠模型和患者来源的类器官模型中得到了验证。从机制上说，我们确定了 ARID1A、PRMT5 和 DHX9 之间的关联，IP-MS 及 CO-IP 实验证实 PRMT5 通过精氨酸二甲基化 DHX9-1160 位点抑制 R-loop 的形成。RNaseH1 的外源表达减弱 PRMT5 抑制对 ARID1A 缺失细胞的杀伤性。我们进一步探索发现 ARID1A 直接结合 PRMT5 启动子调控其表达，其缺失导致 PRMT5 表达下调，进而削弱 DHX9 的甲基化修饰及其与 R-loop 的募集，最终使肿瘤细胞依赖 PRMT5 维持 R-loop 稳态。PRMT5 抑制进一步加剧 ARID1A 缺陷细胞的 R-loop 积累和 DNA 损伤，最终诱发合成致死。

结果 本研究首次阐明 PRMT5 抑制剂 JNJ-64619178 通过破坏 R-loop 稳态在 ARID1A 突变型子宫内膜癌中发挥合成致死作用的分子机制，揭示了 ARID1A-PRMT5-DHX9 信号轴在肿瘤发生发展中的关键调控作用，为 ARID1A 突变型子宫内膜癌的精准治疗提供了新的分子靶点和理论依据。

PO_417

A systematic review and meta-analysis of transumbilical laparoendoscopic single-site versus conventional laparoscopic surgery for endometrial cancer

weimin xie Lihong Lai Shuiping Tang*
Affiliated Hengyang Hospital of Hunan Normal University & Hengyang Central Hospital

Background This systematic review and meta-analysis aimed to compare the safety and effectiveness of transumbilical laparoendoscopic single-site surgery (LESS) with conventional laparoscopic surgery (CLS) in treating endometrial cancer.

Materials and Methods We conducted an electronic search of literature published in PubMed, the Cochrane Central Register of Controlled Trials (CENTRAL), Embase, and ClinicalTrials. gov, up to March 8, 2025. The primary outcomes included intraoperative complications, postoperative

complications, and patient satisfaction with cosmetic outcomes. The secondary outcomes included operative time, estimated blood loss, blood transfusion, conversion, postoperative hospital stay, total lymph nodes harvested, pelvic lymph node harvested, para-aortic lymph node harvested, postoperative pain scores, analgesic use, recurrence, and all-cause mortality during follow-up.
Results Ten studies describing a total of 1,705 patients that met the predefined eligibility criteria were included. There were no significant differences between the LESS group and the CLS group in terms of intraoperative complications and postoperative complications. There were also no differences in terms of operative time, blood transfusion, conversion, total lymph nodes harvested, pelvic lymph node harvested, para-aortic lymph node harvested, postoperative pain scores, analgesic use, recurrence, and all-cause mortality. LESS was associated with higher cosmetic satisfaction ($P < 0.00001$), less estimated blood loss ($P = 0.03$), and shorter postoperative hospital stay ($P = 0.03$) comparing with CLS.
Conclusion This meta-analysis suggests that LESS surgical staging could be a viable alternative to CLS for treating endometrial cancer, offering similar surgical outcomes and additional benefits, including better cosmetic outcome, less estimated blood loss, and shorter postoperative hospital stay.

PO_418

DENND1A 降低通过抑制 MIB1 介导的 EGFR 泛素化降解进而促进子宫内膜癌的进展

茅文鑫 杨二艳 王颖梅*
天津医科大学总医院

目的 子宫内膜癌（Endometrial carcinoma, EC）发病率和死亡率呈逐年上升趋势，但其发生发展的分子机制尚未完全阐明。DENND1A 是 conndecdenn 蛋白家族的重要成员，目前关于其在 EC 中的研究仅限于一项血清学 SNP 分析，具体功能和分子机制有待深入探究。
方法 本研究通过整合 GEO、CPTAC 多组学数据库和临床子宫内膜样本，系统分析了 DENND1A 表达与 EC 临床特征的相关性，并采用免疫组织化学技术检测了 DENND1A 在正常子宫内膜和 EC 组织中的表达差异。进一步通过转录组测序、蛋白质免疫印迹、集落形成实验、Transwell 迁移侵袭实验、定量逆转录 PCR、免疫共沉淀、免疫荧光共聚焦以及小鼠异种移植瘤模型等多种实验方法，研究了 DENND1A 在 EC 发生发展中的作用及具体机制。
结果 多组学数据库分析显示 DENND1A 在 EC 组织中的表达水平较正常组织显著降低，且其高表达与患者良好预后呈显著正相关。通过对临床样本分析，发现在晚期 EC 患者及发生淋巴结转移的病例中，DENND1A 表达水平进一步显著下调，提示 DENND1A 的表达缺失可能促进 EC 的恶性进展和转移。体外功能实验证实，过表达 DENND1A 可显著抑制 EC 细胞的增殖、迁移和侵袭，敲低 DENND1A 则相反。转录组测序分析显示，DENND1A 敲低组中表皮生长因子受体（EGFR）信号通路相关基因显著富集，并且实验证实过表达 DENND1A 可抑制 EGFR 下游 MAPK 和 PI3K/AKT 信号通路的激活；相反，敲低 DENND1A 则促进这些通路的活化。机制上，通过整合转录组学数据和泛素化预测网站（Ubibrowser）分析，发现 E3 泛素连接酶 MIB1 可能参与 EGFR 的蛋白降解过程。随后的蛋白电泳和免疫共沉淀实验证实，敲低 DENND1A 通过下调 MIB1 的表达，降低泛素-蛋白酶体系统对 EGFR 的降解效率。更重要的是，细胞和动物实验结果表明，EGFR 抑制剂吉非替尼可有效逆转 DENND1A 敲低导致的 EC 细胞增殖、迁移和侵袭能力的增强。
结果 DENND1A 的低表达水平与 EC 的进展和较差的临床结果密切相关。此外，敲低 DENND1A 通过下调 E3 泛素连接酶 MIB1 的表达，减少 EGFR 的降解，进而持续激活 EGFR 信号通路，促进 EC 的进展。这一发现为阐明子宫内膜癌的发生发展机制提供了新的理论依据。

PO_419

FOXK2 的降低通过激活 PIK3R5 介导的 PI3K/AKT 信号通路进而促进子宫内膜癌发生发展

王寅丹 杨二艳 王颖梅*
天津医科大学总医院

目的 子宫内膜癌（Endometrial carcinoma，EC）作为女性生殖系统常见恶性肿瘤，严重危害女性健康。有关 EC 发生发展的机制仍需探索。FOXK2 是 FOX 家族的一员，其主要作为转录因子发挥作用。FOXK2 发挥的具体作用复杂多样，在不同癌症背景下，可能发挥促癌也可能发挥抑癌作用。但 FOXK2 在子宫内膜癌的中的作用尚不明确。
方法 本研究通过对数据库 TCGA、CPTAC 分析和临床样本分析发现 FOXK2 在癌组织中的表达显著低于正常组织，且与 EC 良好预后显著相关。通过转录组测序结合体外细胞实验慢病毒转染、CCK8、克隆形成、蛋白印迹、免疫荧光、CHIP-qPCR、构建小鼠异种移植物肿瘤模型，验证 FOXK2 在子宫内膜癌发生发展进展中的作用及具体作用机制。
结果 通过数据库和临床样本分析发现 FOXK2 在 EC 组织中的表达显著低于正常组织，且与良好预后显著相关。选取子宫内膜癌 HEC-1A、Ishikawa 细胞系进行敲低/过表达 FOXK2.通过 CCK8、平板克隆、迁移侵袭实验，发现敲低 FOXK2 可促进子宫内膜癌细胞增殖、侵袭和迁移，过表达 FOXK2 则发挥抑制作用。随后通过对转录组测序分析发现在敲低 FOXK2 后 PI3K/AKT 通路被富集。细胞实验证实敲低 FOXK2 后 PI3K/AKT 通路被激活，而过表达 FOXK2 可以抑制该通路活化。为了进一步探究敲低 FOXK2 激活 PI3K/AKT 通路的机制，我们通过对转录组测序数据中具有磷酸化功能的基因与 PI3K/AKT 通路中募集到的基因取交集，结合体外 PCR 和蛋白电泳验证最终筛选到了 PIK3R5。通过蛋白电泳、CHIP-qPCR 证实敲低 FOXK2 通过促进 PIK3R5 的转录进而激活了 PI3K/AKT 通路。并且本研究发现 PI3K/AKT 通路激活可影响 FOXK2 的核转位，过表达 FOXK2，可促进 FOXK2 入核增多，而使用 PI3K/AKT 通路激活剂之后，FOXK2 入核减少。后续通过细胞和动物实验结果表明 PI3K/AKT 通路抑制剂 MK2206 明显抑制了 FOXK2 敲低组 EC 细胞的增殖、迁移和侵袭。
结论 本研究发现 FOXK2 的低表达与 EC 的进展和较差的临床预后密切相关。敲低 FOXK2 通过促进 PIK3R5 的转录激活 PI3K/AKT 通路促进子宫内膜癌的进展.此外 PI3K/AKT 通路的激活降低 FOXK2 的核转位进而抑制 FOXK2 的作用。这一创新性发现为子宫内膜癌发病机制以及治疗的探索提供了新的见解。

PO_420

利用条件重编程技术生成稳定的小鼠子宫内膜肿瘤衍生细胞系

赵蓉 黄蓝青
中南大学湘雅医院

背景 子宫内膜癌（EC）是全球常见的女性生殖道恶性肿瘤。 虽然通过全面的基因组分析发现了 EC 的多种基因改变，但疾病模型仍然相对罕见，限制了我们进行疾病研究的能力。 建立 EC 的体外和体内混合模型，并将其与体内模型进行比较，可大大推动 EC 的研究。
方法 我们建立了基因工程小鼠 PTEN $^{flox/+}$; STK11 $^{flox/flox}$; LTF-Cre (PSC)，并利用条件重编程技术从其子宫肿瘤组织中分离构建了相应的上皮细胞系。 这些细胞系已成功传代、冷冻并储存在生物库中，为子宫内膜癌研究提供了宝贵的资源。
结果 从 PSC 小鼠子宫肿瘤组织中保留下来的细胞系在传代过程中保持了与亲代小鼠相似的突变等位基因，这对将来用于功能测试尤为重要。 此外，我们还对分离的小鼠细胞系进行了各种功能测试，并在免疫缺陷小鼠和免疫系统完好的 C57BL/6 小鼠皮下植入细胞后形成肿瘤。 我们用血红素和伊红（H&E）

染色和免疫组化染色来描述皮下植入肿瘤的组织病理学和生物标志物，结果显示它们与亲代小鼠子宫肿瘤非常相似。
结论 分离的小鼠细胞将成为基础和临床前心血管疾病研究的重要工具，与相应的体内模型非常匹配。该细胞可加深我们对子宫内膜癌疾病发病机制的了解，并有助于开发新的疗法。

PO_421

Glycine-Serine-Threonine Metabolism Guides Precision Subtyping in Endometrial Carcinoma: Implications for Clinical Management

Jingxuan Ye[1,2] Yashi Shi[1,2] Xite Lin[1,2] Maotong Zhang[1,2] Xiaodan Mao[2] Pengming Sun[2*]
1.College of Clinical Medicine for Obstetrics & Gynecology and Pediatrics, Fujian Medical University
2.Laboratory of Gynecologic Oncology, Fujian Maternity and Child Health Hospital, College of Clinical Medicine for Obstetrics & Gynecology and Pediatrics

Objective Endometrial carcinoma (EC) imposes a growing global disease burden, with obesity-associated metabolic disorders serving as key drivers of rising incidence. While The Cancer Genome Atlas (TCGA) molecular classification guides precision therapy and early intervention improves patient prognosis, advanced EC remains highly lethal with current prognostic systems failing to adequately capture molecular heterogeneity. Metabolic reprogramming—particularly amino acid dependency—constitutes a fundamental malignancy feature that critically facilitates tumor progression. Investigating this axis may yield novel diagnostic/prognostic biomarkers and therapeutic targets for EC. This study systematically profiles glycine-serine-threonine (Gly/Ser/Thr) metabolism-related gene expression patterns in EC, constructs a pathway-based prognostic risk model, and establishes a metabolism-immune crosstalk-driven molecular subtyping framework to advance precision oncology paradigms.
Materials and Methods We first conducted untargeted metabolomics analysis on 35 EC tissues and 15 normal control tissues. We identified metabolic pathways through differential metabolite analysis and screened for metabolism-related genes within the glycine, serine, and threonine metabolism pathway. Further employing Weighted Gene Co-expression Network Analysis (WGCNA), we screened for metabolism-related genes closely associated with EC. Using machine learning methods (LASSO regression and univariate/multivariate COX regression), we constructed a prognostic prediction model. The dataset was stratified into high-risk and low-risk groups based on this model, and its validity was verified in an external dataset. Concurrently, we utilized TCGA database to perform pathway enrichment analysis, immune microenvironment analysis, and tumor mutational burden analysis between the high-risk and low-risk groups. We further classified the EC samples into subtypes using consensus clustering analysis. Subsequently, within each subtype, patients were divided into high-risk and low-risk subgroups based on the prognostic model's risk score. Gene Set Variation Analysis (GSVA) was performed to identify differentially enriched pathways between these subgroups. Combined with immune infiltration analysis specific to each subtype, we redefined the characteristics of each molecular subtype.
Results Comparative metabolomics analysis between EC tissues and normal endometrium revealed significant enrichment in the glycine/serine/threonine metabolism pathway. Screening identified 1,741 genes strongly correlated with this pathway. WGCNA pinpointed a metabolic gene module closely linked to tumor tissue. Intersection with differentially expressed genes (DEGs) from TCGA-UCEC yielded 90 significantly upregulated metabolism-related genes. Machine learning then constructed a prognostic risk model incorporating MTHFD2, RPS6KA1, and CDKN2A. Validation through proteomics and external datasets confirmed differential expression of these prognostic proteins, further supported by single-cell sequencing demonstrating their expression within specific cell subpopulations. Utilizing these module genes, EC patients were stratified into three subtypes with distinct molecular profiles and prognosis, redefined as: Immuno-Metabolic Suppressed (C1), Proliferation-Immunity Balanced (C2), and Immuno-Activated (C3). Notably, the C1 subtype exhibited the poorest prognosis, characterized by suppressed metabolic pathways (e.g., bile acid, fatty acid), elevated immune

checkpoint levels, diminished immune surveillance cells, and features of immune escape. The C2 subtype demonstrated concurrent high proliferative activity and robust anti-tumor immunity; this "proliferation-immunity balance" likely underpins its favorable prognosis, as high proliferation signals are effectively counterbalanced by the immune system. The C3 subtype displayed upregulation of DNA repair signaling and oxidative phosphorylation pathways, coupled with infiltration of activated CD8+ T cells and dendritic cells (DCs), collectively contributing to its good prognosis.
Conclusion This study is the first to construct a prognostic model for EC based on glycine-serine-threonine metabolism-related genes, providing novel molecular biomarkers for early diagnosis and prognostic assessment of EC. This model demonstrates high predictive accuracy and offers potential for guiding personalized clinical therapy. Furthermore, we established a molecular subtyping system based on metabolic-immune characteristics, offering new biomarkers and potential intervention targets for the precise treatment of EC.

PO_422

雌激素上调 TET2 促进 KRT8 启动子去甲基化在内异症恶变为卵巢内膜样癌中的作用机制研究

陈放
辽宁省人民医院

子宫内膜异位症是一种常见的雌激素依赖性妇科疾病，它是一种长期的慢性炎症性疾病，恶变率约为 1%。内异症恶变而来的卵巢癌称为子宫内膜异位症相关卵巢癌，其中内异症相关卵巢内膜样癌（ovarian endometrioid carcinoma，OEC）为内异症恶变的主要病理类型之一，且与过度雌激素暴露密切相关，故探索内异症恶变为不同病理类型的精准分子机制对研究内异症恶变的早期诊断和治疗靶点至关重要。根据蛋白组学筛选结合 GEO 数据库，KRT8 在内异症相关卵巢内膜样癌组织中上调，并预测高表达 KRT8 患者的临床病理分期更高。KRT8 促进异位症在位内膜及雌激素相关子宫内膜腺癌细胞在体外和体内的增值、迁移和侵袭。此外，雌激素通过上调 TET2 促进 KRT8 启动子的去甲基化来增加 KRT8 的表达。TET2 介导的 KRT8 去甲基化促进原代在位内膜及内膜癌细胞的恶行生物学行为。在临床 OEC 患者标本及雌激素诱导大鼠内异症恶变模型中，KRT8 的表达与 TET2 呈正相关。我们的研究将 E2/TET2/KRT8 确立为促进 OEC 发生和进展的关键轴。抑制 TET2 或 KRT8 将是雌激素诱导的 OEC 中潜在的有前途的诊疗策略。

PO_423

Targeting the CD47-HCK-LGALS9 axis disrupts proliferation-immunosuppression coupling in early-stage endometrial cancer

Jingxuan Ye[1,2] Yashi Shi[1,2] Maotong Zhang[1,2] Xite Lin[1,2] Xiaodan Mao[2] Pengming Sun[2*]
1.College of Clinical Medicine for Obstetrics & Gynecology and Pediatrics, Fujian Medical University
2.Laboratory of Gynecologic Oncology, Fujian Maternity and Child Health Hospital, College of Clinical Medicine for Obstetrics & Gynecology and Pediatrics

Objective Endometrial cancer (EC) exhibits rising incidence and mortality rates, particularly showing a trend toward affecting younger women. Current diagnostic markers for early-stage detection are lacking, and while high-dose progesterone serves as a fertility-sparing treatment for young patients, it faces challenges of suboptimal efficacy and high recurrence rates. The tumor microenvironment (TME) plays a crucial role in EC initiation and progression; however, its specific mechanistic involvement in

the malignant proliferation of early-stage EC remains incompletely understood. Epithelial cells exhibit significant heterogeneity in EC. Although prior research has identified specific precancerous subpopulations and epithelial-myeloid cell interactions, the precise interaction mechanisms between oncogenic subpopulations and the tumor immune microenvironment (TIME) require further in-depth investigation.

Materials and Methods We collected tumor samples from patients with early-stage EC and paired adjacent normal endometrial tissues for single-cell RNA sequencing (scRNA-seq) analysis, with a focus on epithelial subpopulations. Through pseudotime analysis and cell-cell communication analysis, we investigated the behavioral characteristics of epithelial cells and their interactions with macrophages. These findings were further validated using spatial transcriptomics and multiplex immunofluorescence (mIF). Simultaneously, we employed functional assays including cell proliferation assays, apoptosis detection, and cell cycle analysis to evaluate changes in EC cell proliferation and apoptosis. To elucidate the mechanistic basis of tumor cell-macrophage crosstalk, we established a co-culture system. Protein-protein interactions were screened and validated via molecular docking and glutathione-S-transferase (GST) pull-down assays coupled with mass spectrometry (MS). Cytokine alterations in the TME were quantified using enzyme-linked immunosorbent assay (ELISA). Additionally, upstream transcriptional regulators of target genes were identified through cleavage under targets and tagmentation (CUT&Tag). Other methodologies included: Western blot, reverse transcription polymerase chain reaction (RT-PCR) and so on.

Results We identified a previously unknown specific epithelial oncogenic subpopulation, the CD47+CDK1+ subpopulation, and discovered its close association with cell cycle signaling pathways and malignant cell proliferation. This subpopulation interacts closely with macrophages through the CD47-Galectin 9 (LGALS9) receptor-ligand pair. Mechanistically, inhibition of CD47 suppresses the proliferative capacity of EC cells and promotes their apoptosis. The oncogenic subpopulation interacts with macrophages via the CD47-Hemopoietic Cell Kinase (HCK) axis, facilitating the secretion of the immunosuppressive molecule LGALS9, thereby fostering an immunosuppressive tumor microenvironment. Furthermore, LGALS9 secreted by macrophages acts on CD47 to promote EC cell proliferation, establishing a positive feedback loop involving CD47-HCK-LGALS9. Additionally, we identified the upstream transcription factor estrogen related receptor gamma (ERRγ/ESRRG) of CD47 and found that progesterone could inhibit the expression of ERRγ.

Conclusions In summary, this study identifies for the first time an oncogenic CD47+CDK1+ epithelial subpopulation in early-stage EC that not only suppresses macrophage phagocytosis but also synergizes with macrophage-derived LGALS9 to drive malignant proliferation and amplify immunosuppression via IL-10/TGF-β secretion, thereby fostering an immunosuppressive niche. ERRγ, the upstream transcriptional regulator of this subpopulation, offers a druggable target for indirect intervention. Given significant side effects of CD47 inhibitors, we propose combining ERRγ-targeted agents with progesterone-based therapy to suppress EC proliferation—providing a clinically translatable strategy that concurrently targets early EC and its immunosuppressive microenvironment.

PO_424

IGF-1 及糖脂代谢指标与子宫内膜癌分子分型的关系

时娟娟
滕州市中心人民医院

目的 探讨胰岛素样生长因子-1（Insulin-like growth factor 1，IGF- 1）及糖脂代谢指标与子宫内膜癌（Endometrial cancer，EC）分子分型的相关性。

方法 收集 2023 年 4 月至 2024 年 4 月经滕州市中心人民医院诊断为 EC 并手术治疗患者 126 例，通过对 EC 患者进行分子分型检测，比较不同分子分型 EC 患者临床病理特征、IGF-1 的表达、血糖血脂等代谢综合征相关指标。

结果 126 例 EC 患者分子分型分别为 p53wnt 型 75 例（59.5%）、p53abn 型 20 例（15.9%）、MMRd 型 26 例（20.6%）和 POLE EDM 型 5 例（4.0%）。不同分子分型子宫内膜癌患者在绝经人数、FIGO 分期、淋巴结转移、空腹血糖、血清 IGF-1、糖化血红蛋白表达方面差异有统计学意义（P<0.05）。p53abn 型空腹血糖、血清 IGF-1、糖化血红蛋白以及子宫内膜癌组织 IGF-1 表达高于其他三型，差异有统计学意义（P<0.05）。多因素 logistic 回归分析显示，空腹血糖及血清 IGF-1 是 p53abn 型子宫内膜癌患者发生糖代谢异常的危险因素。

结论 糖代谢异常可能与子宫内膜癌分子分型有关，尤其血糖升高以及 IGF-1 表达升高与 p53abn 型子宫内膜癌相关，合并糖代谢异常的子宫内膜癌患者，更应该关注分子分型检测。

PO_425

前哨淋巴结活检联合病理超分期检测技术在子宫内膜癌治疗中的应用

王小石 叶佳 张金伟*
南京医科大学附属无锡人民医院

前哨淋巴结活检技术近年来已广泛应用于早期子宫内膜癌，目前大量循证医学研究已支持证明了前哨淋巴结活检替代传统系统性淋巴清扫术的治疗效果。最近更多研究发现前哨淋巴结活检对高危型子宫内膜癌同样有效，极大地降低了手术风险，减轻患者的治疗负担。而病理超分期作为一种增强病理检测技术，可进一步提高前哨淋巴结中肿瘤微转移的检出率，弥补了常规病理检测的局限性，指导除手术外的治疗方式，进而改善患者的远期预后。

PO_426

High Glucose Promotes Malignant Progression of Endometrial Carcinoma by Inhibiting DUSP2

Ling Zhou Jianliu Wang*
Peking University People's Hospital,

Objective Clinical studies have revealed that patients with endometrial carcinoma co-existing with diabetes mellitus exhibit poorer prognoses. High glucose environments have been identified as accelerators of the malignant progression of endometrioid adenocarcinoma, yet the underlying mechanisms remain unclear. This study aims to explore the molecular mechanisms through which high glucose levels facilitate the progression of endometrioid adenocarcinoma.

Materials and Methods

1. Bioinformatics Analysis: Original RNA-seq data and clinical information of endometrial carcinoma patients were collected from The Cancer Genome Atlas (TCGA) database to analyze lymph node metastasis in patients with and without diabetes.
2. RNA-seq: Differences in the transcriptome of Ishikawa cells cultured in high and low glucose conditions were examined to identify molecules differentially associated with high glucose.
3. qPCR and Western Blot: Key molecules identified through RNA-seq in the context of high glucose were validated.
4. In Vitro Cellular Experiments: CCK8 assays, plate cloning experiments, and Transwell assays were conducted to study the impact of DUSP2 on the phenotype of Ishikawa cells.
5. Identification of DUSP2 Downstream Target Genes: qPCR experiments were utilized to identify DUSP2 downstream target genes related to the glycolytic pathway.

Results

1. Big Data Analysis: Analysis of original transcriptomic data and clinical information of 345 endometrioid adenocarcinoma (EAC) patients from the TCGA database indicated a significantly higher probability of positive lymph nodes adjacent to the abdominal aorta or in the pelvis in patients with high blood sugar levels.
2. RNA-seq: Long-term high-glucose culture of Ishikawa cells showed significant upregulation of the MAPK signaling pathway, with a notable decrease in the expression levels of the DUSP family that inhibits this pathway, especially DUSP2, DUSP4, and DUSP10.
3. qPCR and Western Blot: It was found that low glucose could promote the expression of DUSP2, but its expression levels significantly decreased upon restoring high glucose conditions.
4. In Vitro Cellular Experiments: CCK-8 and plate cloning experiments demonstrated that disrupting DUSP2 expression significantly increased the proliferation ability of Ishikawa cells. Transwell assays showed a significant increase in the invasion of Ishikawa cells upon DUSP2 disruption, indicating that DUSP2 can inhibit the progression of endometrial carcinoma.
5. Identification of DUSP2 Downstream Target Genes: qPCR experiments revealed that disrupting DUSP2 expression significantly increased the expression levels of glycolysis-related enzymes HK1, HK2, GPI, PGMA1, LDHA, with HK2 showing the most significant upregulation; TIGAR (a p53-induced glycolysis inhibitor) expression level decreased; and the level of PDK1, an enzyme inhibiting the shift from glycolysis to oxidative phosphorylation, increased. This suggests that inhibiting DUSP2 can promote glycolysis over oxidative phosphorylation, thereby facilitating the malignant progression of endometrial carcinoma; conversely, DUSP2 can inhibit glycolysis and promote oxidative phosphorylation.

Conclusion

1. High glucose levels can inhibit the expression of DUSP2.
2. DUSP2 can suppress the proliferation and invasion capabilities of endometrial carcinoma.
3. DUSP2 regulates the proliferation of endometrial carcinoma through HK2.

PO_427

聚焦术前 “高危型”子宫内膜非典型增生:术后病理升级的多维度预测模型的构建、验证及诊治策略指导

吕巧英[1] 张锦玉[1] 陈晓军[2] 罗雪珍[1*]
1.复旦大学附属妇产科医院 2.上海市第十人民医院

研究背景及目的 子宫内膜非典型增生作为子宫内膜样癌的前驱病变，因病理取材局限及诊断难度，约20%-50% 患者全子宫切除术后会出现病理升级为子宫内膜癌的情况。若术后病理提示侵袭性特征，患者需二次手术，显著增加身心与经济负担。因此，精准识别术前高危病变、实现分层诊疗、提高手术病理分期并降低二次手术风险成为关键临床问题。本研究旨在基于多维度临床病理因素，构建高危型子宫内膜非典型增生术后病理升级的预测模型，为个性化诊治策略制定及诊疗流程优化提供依据。
研究方法 回顾性分析 2021 年 1 月至 2024 年 7 月复旦大学附属妇产科医院连续收治的 1114 例术前诊断为子宫内膜非典型增生并行手术治疗的患者，分为训练集（891 例，模型构建）和验证集（223 例，模型验证）。收集术前临床信息（年龄、绝经状态、病程、体重指数、合并症、肿瘤家族史、血清标志物 CA125 和 HE4 水平、免疫炎性状态、内膜厚度、盆腔增强 MRI 结果等）、术前病理信息（取材方式、病变范围及严重程度），以及术中冰冻和术后病理信息。通过单因素及多因素逻辑回归筛选病理升级风险因素，构建列线图预测升级概率，并评估预测准确性和临床实用价值。
研究结果 训练集 891 例中，193 例（21.7%）术后病理升级为子宫内膜癌，其中 30 例（15.5%）为 FIGO 2023 分期 IB 期及以上。分析显示，子宫内膜非典型增生病变弥漫 / 更重病变不除外、绝经状态、内膜厚度≥13mm、HE4≥50 pmol/L、盆腔增强 MRI 提示恶性与病理升级显著相关。基于上述因素构建的列线图预测效能良好，训练集 AUC=0.814（95% CI=0.778–0.850），验证集 AUC=0.810（95%

CI=0.741–0.878），两者校准曲线均接近对角线。决策曲线分析显示，训练集在 1%-99% 阈值、验证集在 1%-90% 阈值范围内模型均有净收益，提示其较好临床价值。
研究结果 基于 5 个关键因素构建的“高危型”子宫内膜非典型增生列线图，可准确预测术后病理升级为子宫内膜癌的风险。鉴于术前诊断患者术后病理升级较常见，对高风险者应实施分层诊疗，完善影像学评估并制定个性化手术策略，以降低二次手术风险，提升子宫内膜癌手术病理分期。

PO_428

早期 POLE 突变型子宫内膜癌降级处理后复发 1 例及文献回顾

吕巧英 王懿琴 刘佳 罗雪珍*
复旦大学附属妇产科医院

根据 2023 年 FIGO 最新分期标准及 2021 年 ESGO/ ESTRO/ ESP 指南推荐，对于Ⅰ-Ⅱ期 *POLE* 突变型子宫内膜癌患者可考虑采取降级治疗，将密切随访作为首选方案。本文报道 1 例 64 岁老年子宫内膜癌患者，病理诊断为高级别子宫内膜样癌，合并深肌层浸润及广泛脉管内癌栓，分子分型为 *POLE* 突变。患者术后未行辅助治疗，仅密切随访，术后 19 个月发现阴道局部复发伴盆腔淋巴结转移。虽然 *POLE* 突变型子宫内膜癌通常预后良好，但本例患者合并高龄（>60 岁）、高级别组织学类型、深肌层浸润及广泛 LVSI 等高危因素，最终仍出现疾病复发。这一临床现象提示，基于分子分型的降级治疗策略可能存在一定局限性。目前尚缺乏前瞻性研究证据支持对此类具有高危因素的患者豁免辅助治疗。未来需要进一步探索分子特征与临床病理因素的整合分析，以优化患者分层管理策略，在治疗不足与过度治疗之间寻求最佳平衡。

PO_429

Integrating Metabolic Profiling and Clinicopathologic Signatures: Development of a Deep Learning-Based Predictive Tool for Fertility Treatment in Endometrial Cancer

Yuman Wu
Peking University People's Hospital

Objective This study evaluated the predictive value of metabolic risk score (MRS) for complete remission (CR) in patients with endometrial cancer and atypical endometrial hyperplasia treated with fertility-sparing treatment (FST).
Methods This study included 340 patients with endometrial cancer who were treated with FST at Peking University People's Hospital from January 2012 to December 2024. All patients were randomly divided into a training group and a validation group in a ratio of 2:1. Clinicopathologic data were collected from the patients. The development of 101 predictive models was achieved through the implementation of a leave-open cross-validation (LOOCV) framework, with the objective of identifying combinations that exhibited the highest C-index. The predictive accuracy of enrolment factors for CR was estimated through the utilization of decision curve analysis (DCA), calibration curves and receiver operating characteristic (ROC) curves. Furthermore, Kaplan-Meier survival analysis (K-M survival analysis) were employed to analyze CR rates in diverse stratified population.
Results Independent predictors included MRS, gestation, pregnancy, cholesterol (TC), and histology. The model was developed to predict the probability of CR in endometrial cancer patients undergoing FST based on these factors. ROC curve analysis demonstrated that the training and validation groups were effective predictors of CR at 6-month, 1-year, and 2-year follow-ups. In the training group, the

area under the ROC curve (AUC) values were 0.750, 0.805, and 0.848, respectively. Subsequently, patients were divided into two groups of high MRS related score and low MRS related score based on regression coefficients, and the Kaplan-Meier curves confirmed that FST patients with high MRS related scores had a greater likelihood of CR, and also showed significant differences in different population strata. It is noteworthy that in Model 2, with the inclusion of MRS, the predictive accuracy AUC of the training cohort increased from 0.821 to 0.894, and the validation cohort exhibited a similar increase in AUC values.
Conclusion The MRS-based prognostic model for endometrial cancer patients receiving FST has shown enhanced predictive performance, enabling more informed clinical decision-making.

PO_430

上皮性癌成分对子宫癌肉瘤预后的影响

尤学武 王建六*
Peking University People's Hospital

目的 本研究旨在探讨子宫癌肉瘤（Uterine carcinosarcoma, UCS）和卵巢癌肉瘤（Ovarian carcinosarcoma, OCS）在临床病理特征上的差异，并分析上皮性癌成分对子宫癌肉瘤患者生存的影响。UCS 作为一种少见且具有高度恶性潜力的肿瘤，其生物学特性和预后因素仍不明确，因此，本研究对其临床特征和预后因素进行深入分析，旨在为 UCS 的早期诊断和个性化治疗提供参考。
材料与方法 我们回顾性地收集了北京大学人民医院 2007 至 2024 年期间 53 例具有完整临床和病理资料的子宫和卵巢癌肉瘤病例，涵盖 39 例 UCS 和 14 例 OCS。病例数据包括年龄、月经史、症状、肿瘤标志物（CA-125）、手术方式、肿瘤大小、FIGO 分期、淋巴结转移及上皮成分等。所有病例均由两名病理科医师复核 HE 染色切片的病理特征。采用 SPSS 25.0 和 Prism 9.4.1 软件进行统计学分析。正态分布数据以均数±标准差表示，采用 Student's t-检验、卡方检验和 Fisher 精确检验进行组间比较，生存分析使用 Kaplan-Meier 法评估生存时间分布，并采用 log-rank 检验比较生存曲线。多变量 Cox 比例风险模型用于计算风险比（HR），P 值<0.05 被认为具有统计学意义。
结果 UCS 与 OCS 在临床表现和治疗反应上存在显著差异。UCS 患者的肿瘤直径较小（6.7±4.6cm vs. 10.6±5.9cm，P=0.017），术前 CA-125 升高的比例较低（30.8% vs. 78.6%，P=0.001）。此外，UCS 患者更倾向于接受彻底的分期手术，而 OCS 患者则更多接受肿瘤减灭术（82.1% vs. 50.0%，P=0.001）。在预后分析中，我们发现上皮性癌成分对子宫癌肉瘤的预后有显著影响。与内膜样癌成分的 UCS 患者相比，浆液性 UCS 患者的无进展生存期（PFS）较短（HR=6.981，95%CI: 1.571-23.079，P=0.009），但在总生存期（OS）上的差异不显著（HR=1.977，95%CI: 0.435-8.989，P=0.378）。此外，CA-125 水平升高（HR=7.878，95%CI: 1.398-44.409，P=0.019）、肿瘤侵入深度（HR=9.525，95%CI: 0.894-19.924，P=0.009）和淋巴管浸润（HR=2.049，95%CI: 0.299-14.049，P=0.465）均与较差的预后相关。Cox 分析表明，上皮性癌成分是影响 PFS 的独立危险因素，而 CA-125 水平则是影响 OS 的独立危险因素。
结论 本研究结果强调了上皮性癌成分是影响 UCS 预后的关键因素。此外，CA-125 水平、肿瘤侵入深度和淋巴管浸润等因素在 UCS 的预后评估中具有重要价值，为未来的临床治疗和个性化管理提供了重要参考依据。

PO_431

Prognostic Implications of Estrogen Receptor and Progesterone Receptor Expression Discordance in Endometrial Cancer: A Chinese Nationwide Cohort Study

heyan zhang Penglin Liu Beihua Kong Kun Song*
shandong university

Objective Endometrial cancer (EC) is the most common gynecologic malignancy in developed countries and ranks among the top three gynecologic cancers in developing countries, including China, where its incidence has been steadily rising in recent years. EC encompasses a biologically heterogeneous group of tumors with diverse molecular and histological characteristics. Prognosis and therapeutic strategies are influenced not only by traditional clinicopathological features such as FIGO stage, histological grade, and myometrial invasion but also by molecular biomarkers. Hormone receptors—namely, the estrogen receptor (ER) and progesterone receptor (PR)—have long been recognized as important biomarkers in EC, given their pivotal roles in endometrial cell proliferation, differentiation, and carcinogenesis.

Traditionally, EC is categorized into two main types: Type I (endometrioid, hormone receptor-positive, lower grade, and better prognosis) and Type II (non-endometrioid, hormone receptor-negative, higher grade, and poorer prognosis). However, increasing evidence suggests that this dichotomy is overly simplistic, and a more nuanced understanding of hormone receptor expression is needed. While most previous studies have focused on the presence or absence of both ER and PR, recent interest has grown in investigating discordant expression patterns—namely, ER^+PR^- or ER^-PR^+—which may represent biologically distinct subgroups with intermediate or unpredictable prognoses. This study aims to evaluate the clinicopathological characteristics and survival outcomes of patients with different ER/PR expression profiles, with special attention to the discordant expression group. By leveraging a large-scale, nationwide cohort from China, this research seeks to offer robust evidence for incorporating ER/PR status into routine diagnostic and risk stratification protocols for EC patients.

Materials and Methods This study was designed as a multicenter, retrospective cohort analysis involving 5,894 patients diagnosed with EC between January 2000 and December 2019 across 27 tertiary hospitals in China. The inclusion criteria were: (1) histologically confirmed EC, (2) availability of tumor tissue for immunohistochemical (IHC) analysis of ER and PR, and (3) complete follow-up data. Patients with incomplete receptor data or concurrent malignancies were excluded.

IHC staining was performed at each participating center using standardized protocols, and receptor positivity was defined based on a cutoff of ≥1% nuclear staining in tumor cells. Based on ER and PR expression, patients were classified into three groups: double-positive (ER^+PR^+), double-negative (ER^-PR^-), and single-positive (either ER^+PR^- or ER^-PR^+).

Demographic and clinicopathological data, including age, BMI, FIGO stage, histological subtype, grade, lymphovascular invasion, and comorbidities (such as diabetes and hypertension), were collected and compared across the three groups using the chi-square test for categorical variables and ANOVA for continuous variables. Survival data were analyzed using the Kaplan-Meier method to estimate disease-free survival (DFS) and overall survival (OS), with statistical significance assessed using the log-rank test.

Results Regarding histological subtypes, the ER^-PR^- group showed significantly higher proportions of serous carcinoma (15.5%) and clear cell carcinoma (8.2%) compared to the ER^+PR^+ group (1.3% and 0.3%, respectively; $p < 0.001$), indicating an association with more aggressive tumor types. Patients in the ER^-PR^- group were also more likely to be diagnosed at advanced FIGO stages than those in the ER^+PR^+ group ($p < 0.001$). In terms of BMI, underweight patients were more common in the double-negative group ($p < 0.001$), whereas no significant differences were observed in diabetes or hypertension prevalence among groups.

In survival analysis, the ER^+PR^+ group exhibited the best DFS and OS, while the ER^-PR^- group had the poorest outcomes. The single-positive group showed intermediate prognosis. These differences were highly significant for both DFS and OS ($p < 0.0001$).

Conclusion ER and PR expression profiles serve as important prognostic markers in endometrial cancer. Double-positive expression is associated with less aggressive tumor behavior and favorable survival outcomes, whereas double-negative status correlates with high-grade histological subtypes, more advanced disease, and markedly poorer prognosis. Single-positive expression represents an intermediate risk group, highlighting the clinical significance of discordant ER/PR status. Incorporating ER/PR profiles into standard diagnostic and prognostic assessments may enhance risk stratification and support the development of individualized management strategies for EC patients.

PO_432

Single-Cell Revelations: Immune-Based Prognosis and TME Heterogeneity in Endometrial Carcinoma

Tianjiao Zheng Xiaowen Lin Shibo Li Yingbo Liu Liusu Wang Yuhui Sun*
the First Affiliated Hospital of Harbin Medical University

Uterine corpus endometrial carcinoma (UCEC) is one of the most prevalent gynecological malignancies. Despite advances in clinical management, the prognosis for advanced-stage UCEC remains poor due to the lack of effective biomarkers and personalized therapeutic strategies. Increasing evidence suggests that the tumor immune microenvironment (TME) plays a critical role in UCEC development and progression. This study aimed to characterize the immune phenotypes of the TME in UCEC and explore their prognostic significance.Through integrative analysis of cancer genomic data, we employed multiple computational approaches to characterize tumor subtypes. Functional enrichment and survival analyses were performed to preliminarily investigate biological features and clinical implications across different subtypes. The study further evaluated tumor microenvironment characteristics and their potential associations with therapeutic responses, while identifying several molecular markers of possible functional significance through bioinformatics approaches. Network analysis revealed key interaction patterns among critical molecules, with single-cell sequencing data providing novel perspectives on microenvironmental composition. These analytical findings offer reference points for further exploration of disease mechanisms and potential therapeutic targets, though subsequent experimental validation remains warranted for the observed associations.Through comprehensive analysis of cancer genomic data, we employed various bioinformatics approaches to perform molecular classification of tumor samples. Preliminary findings suggest different subtypes may possess distinct biological characteristics with potential clinical significance. Functional enrichment analysis revealed possible involvement of certain metabolic and immune-related pathways in tumor progression. Survival analysis indicated potential associations between specific molecular markers and patient prognosis. Tumor microenvironment evaluation suggested immune cell infiltration levels might influence treatment outcomes. Single-cell sequencing data provided new perspectives for understanding tumor heterogeneity. These findings offer clues for further mechanistic investigations and therapeutic development, though additional experimental validation is required.This integrative TME-based classification system enhances prognostic stratification and offers valuable insights for guiding immunotherapy and targeted treatment in UCEC.

PO_433

A Prognostic Framework Based on Lipid Metabolism and Tumor Immune Microenvironment in Endometrial Carcinoma

Tianjiao Zheng Xiaowen Lin Shibo Li Xianglan He Yuhui Sun*
the First Affiliated Hospital of Harbin Medical University

Endometrial carcinoma (EC) is a leading gynecologic malignancy, with obesity-driven metabolic reprogramming, particularly lipid metabolism dysregulation, playing a key role in tumor progression and immune evasion. However, the prognostic impact of lipid metabolism genes and their interaction with the tumor immune microenvironment (TME) in EC remains unclear.Transcriptomic data from TCGA-UCEC and GEO datasets were analyzed to identify differentially expressed lipid metabolism genes (DELMGs). Prognosis-related genes were screened using Cox regression and Kaplan-Meier analysis. Twelve machine learning algorithms were evaluated, with SuperPC selected to construct a risk model, validated in the CPTAC-3 cohort. A nomogram integrated risk scores with clinicopathological factors. Immune infiltration, pathway enrichment, and drug sensitivity were assessed using bioinformatics tools, and single-cell RNA sequencing (scRNA-seq) mapped lipid metabolic heterogeneity.We identified a subset of metabolism-related genes with altered expression, including a smaller group associated with clinical outcomes, which were involved in specific lipid-related pathways. A multi-gene model based on these genes categorized patients into different risk groups with varying survival outcomes across two independent datasets. The risk stratification showed connections with certain clinical characteristics. A predictive tool demonstrated reasonable performance over time. Higher-risk tumors displayed enrichment in proliferative and genomic stability pathways, while lower-risk cases showed activation of metabolic and immune-related processes. Single-cell analysis highlighted a particular immune cell population as potentially important in lipid metabolism, possibly influencing the tumor microenvironment. Drug sensitivity patterns differed between risk groups, with higher-risk cases showing better responses to certain cytotoxic therapies and lower-risk cases potentially benefiting more from immune-targeted approaches.Metabolic dysregulation may contribute to disease progression and immune escape. A gene signature shows prognostic value, with differential treatment responses observed across risk subgroups. These findings implicate metabolic pathways in disease pathogenesis and inform potential precision medicine approaches.

PO_434

子宫内膜癌的代谢-免疫交互机制解析及临床转化模型构建

刘湘玉 郑玮琴 金文婷 张莹 孙宇辉*
哈尔滨医科大学附属第一医院

背景及目的 子宫内膜癌（EC）的进展涉及代谢重编程与免疫微环境的动态交互，但其关键驱动因子尚未明确。本研究通过整合多维度组学数据，系统探索特定代谢通路在 EC 恶性演进及免疫微环境重塑中的作用，旨在开发新型预后工具并筛选潜在治疗靶点。

研究方法 基于公共数据库的 EC 转录组及单细胞测序数据，通过生物信息学方法解析肿瘤微环境异质性特征及识别代谢-免疫交互网络中的关键分子；单细胞转录组分析识别代谢活性异常的免疫细胞亚群及与异常免疫细胞相关的差异基因并构建代谢-免疫调控网络。进一步通过 COX 回归进一步筛选出与生存预后的相关基因，结合 LASSO 回归与正则化算法构建目标基因风险评分系统，利用生存分析验证其临床预测效能。

研究结果 特定代谢酶及目标代谢通路相关基因群与 EC 临床进展显著相关；免疫微环境解析揭示特定代谢酶相关基因表达差异介导了免疫细胞亚群的功能状态转化异常；所构建的基于 COX 回归的预后风

险模型（训练集 1、3、5 年时间依赖性 AUC 均较高）中高风险组患者总生存期显著缩短，核心代谢酶相关基因表达水平与患者不良预后独立相关。结果均具有统计学差异。
结论 本研究首次证实特定代谢酶通过介导免疫微环境与代谢重编程促进 EC 进展的机制，所开发的预后模型展现出良好的临床转化前景。该发现为 EC 代谢-免疫协同靶向治疗提供了理论依据，有望推动高风险患者的早期精准干预。

PO_435

肠道微生物群多样性检测可能作为子宫内膜癌的筛查手段

余娇 王柳苏 张宇 董乙 孙宇辉*
哈尔滨医科大学附属第一医院

子宫内膜癌是常见的妇科三大恶性肿瘤之一，全球发病率近年来持续上升。尽管晚期子宫内膜癌预后较差，早期诊断子宫内膜癌可以改善患者的长期预后。然而，对于子宫内膜癌的早期检测策略尚未达成共识，目前的检查手段如经阴道超声、血清癌症特异标志物检测和宫腔镜子宫内膜组织活检等具有侵入性强、成本高昂且特异性不足等缺陷。因此，临床亟需准确性高且侵入性较小的筛查测试来精准识别需要侵入性诊断的高风险患者，并能排除低风险人群。早期检测 EC 的新型微创方法，包括血液检测、宫腔灌洗和宫颈阴道液分析。在生物体液样本中检测到的新型基因组生物标志物，可能成为早期筛查的关键突破点。除血液样本外，其他一些体液如唾液、尿液、脑脊液甚至粪便也可作为来源。肠道微生物群是一个动态的生态系统，被称为“第二基因组”。当肠道微生物群的平衡被打破时，肠道微生物群的构成和丰度相应地发生变化，出现肠道微生物群失调；失调的肠道微生物群可通过引起肠道微生物的代谢紊乱，导致免疫功能障碍，进一步影响癌症的发展。许多研究认为肠道微生物群与子宫内膜癌之间存在关联。几项研究表明，子宫内膜癌患者的肠道微生物群的多样性和分布特征与正常人群存在差异。肠道微生物群的检测，取材来自粪便标本，与传统活体组织检查相比，该方法具有诸多优势，包括便捷性、微创性、实时性和可重复性，在难以获取活体组织检查样本的患者管理中展现出强大优势。本文旨在探讨子宫内膜癌肠道微生物群的多样性和分布特征，讨论粪便微生物群多样性检测作为筛查手段施行的可行性，发掘肠道微生物群多样性检测作为子宫内膜癌筛查手段的应用潜力。

PO_436

Dedifferentiated/Undifferentiated Endometrial Carcinoma: Pathobiological Reinterpretation and Prognostic Landscape Update

Bo Wang Yuan Huang Pengyao Jiang Jiongbo Liao Wenyu Shao Chao Wang*
Obstetrics & Gynecology Hospital of Fudan University

Objective To investigate the clinical, pathological, molecular characteristics, treatment, and prognosis of undifferentiated endometrial carcinoma (UDEC) and dedifferentiated endometrial carcinoma (DDEC).
Methods A retrospective analysis was performed on 43 treatment-naive UDEC/DDEC patients admitted to Fudan Obstetrics and Gynecology Hospital (Jan 2020-Jul 2024). Clinical data collected included demographics (age, height, weight), symptoms, tumor markers, tumor size, imaging (ultrasound, MRI, CT), surgical details, pathology, adjuvant therapy, molecular profiling, and germline genetics. Of these, 20 patients with β-catenin IHC data were grouped into membranous-positive vs nuclear-positive based on cellular localization. Clinicopathological features and prognoses were compared between groups to investigate associations with β-catenin expression patterns.

Results Of 43 UDEC/DDEC patients (36 DDEC, 7 UDEC), 25 (58.1%) had extrauterine metastasis at diagnosis. Median follow-up was 25.6±4.97 months (95% CI:15.86-35.34), with 2-year disease-free survival(DFS) 77.5% (95% CI:63%-91.6%). Five (11.6%) died, 7 recurred; mean overall survival(OS) was 48.735±2.82 months (95% CI:43.206-54.263). All 3 recurrent patients with β-catenin testing showed nuclear positivity. Stage III/IV patients had significantly lower OS than stage I/II (P=0.000). Adnexal involvement independently predicted poorer OS (HR=13.128, 95% CI:1.041-165.527, P=0.046). Of 20 with β-catenin testing, 6 were nuclear-positive, 14 membranous-positive. The nuclear group had higher ER/PR negativity (P=0.000) and shorter DFS (P=0.04) vs the membranous group; OS did not differ (P=0.53).
Conclusion UDEC/DDEC is a highly aggressive, poor-prognosis high-grade endometrial carcinoma subtype. Adnexal involvement critically predicts their OS. Tumor cell nuclear β-catenin accumulation marks recurrence risk in UDEC/DDEC, with this localization significantly linked to negative ER/PR expression-suggesting crosstalk between β-catenin and ER/PR pathways that may drive tumorigenesis and progression.

PO_437

Fertility Preservation in Rare Cowden Syndrome-Related Endometrial Carcinoma: Management and Outcomes

Bo Wang Yuan Huang Pengyao Jiang Jiongbo Liao Wenyu Shao Chao Wang*
Gynecology Hospital of Fudan University

Objective To assess the feasibility and efficacy of fertility-preserving treatment in patients with Cowden syndrome (CS)-associated endometrial cancer (EC).
Methods A retrospective analysis was performed on the clinical data and long-term outcomes of 3 patients with CS-EC who underwent fertility-preserving treatment at the Obstetrics and Gynecology Hospital of Fudan University between 2017 and 2025.
Results
Case 1: The patient harbored a germline PTEN frameshift mutation (p.R189Qfs*9) and was diagnosed with stage Ia endometrial cancer at 29 years of age. Treatment with a levonorgestrel-releasing intrauterine system (Mirena) for 19 months failed to induce remission; however, remission was achieved after 8 cycles of gonadotropin-releasing hormone agonist (GnRHa) combined with letrozole. Following recurrence, the same regimen (GnRHa + letrozole) was administered for another 12 cycles, resulting in re-remission. The patient is currently maintained on Mirena with no evidence of recurrence.
Case 2: This patient carried a germline PTEN missense mutation and was diagnosed with stage Ia endometrial cancer at 31 years old. Remission was achieved after 6 months of megestrol acetate therapy, and she remains on Mirena maintenance treatment to date without recurrence.
Case 3: The patient had a germline PTEN nonsense mutation (c.C10003T, p.R335X) and was diagnosed with stage Ia endometrial cancer at 28 years of age. Remission was achieved after 9 months of megestrol acetate treatment. During Mirena maintenance, recurrent hyperplasia developed but was subsequently resolved with dienogest. The patient later achieved successful pregnancy via second-generation in vitro fertilization-embryo transfer (IVF-ET) and is currently maintained on Mirena with no recurrence.
Conclusion Fertility-preserving treatment exhibits a certain degree of feasibility and safety in patients with early-stage CS-EC. However, during fertility-preserving therapy with high-efficacy progestogens, close monitoring for drug resistance is imperative, and prompt adjustment of the treatment regimen should be implemented when necessary.

PO_438

分子分型预测早期Ⅱ型子宫内膜癌预后的分析

秦文娜 郭海龙*
日照市人民医院

目的 探究不同分子分型在早期Ⅱ型子宫内膜癌术后辅助治疗中的临床价值。
方法 收集 78 例ⅠA 期局限于子宫内膜或息肉的Ⅱ型子宫内膜癌患者，对其中 20 例 MMRd 型、52 例 P53abn 型患者展开为期 5 年的随访。运用 Kaplan-Meier 曲线分析其生存预后情况，通过 Cox 回归分析及 Log-rank 检验明确影响早期Ⅱ型子宫内膜癌生存预后的因素。
结果 20 例 MMRd 型患者与 52 例 P53abn 型患者的一般情况对比，差异无统计学意义（P＞0.05）。在 72 例患者中，61 例选择期待治疗。观察期待治疗与辅助化学治疗和/或放射治疗的无进展生存（PFS）风险比（HR, 95% CI）为 2.76（0.26-20.81），P＝0.618（P＞0.05），差异无统计学意义。5 年无进展生存率为 90%，总生存率达 100%。MMRd 型和 P53abn 型患者年龄的 PFS 风险比（HR, 95% CI）为 1.14（1.02-1.31），P=0.023；组织病理的 PFS 风险比（HR, 95% CI）为 4.63（1.41-15.23），P＝0.012，均有统计学意义。
结论 在ⅠA 期仅限于子宫内膜或息肉的Ⅱ型子宫内膜癌中，针对 MMRd 型和 P53abn 型患者，仅采取术后期待治疗呈现出良好的预后效果，尤其是 P53abn 型患者也能从中获益。年龄≥50 岁以及非子宫内膜样腺癌是影响患者预后的关键因素。

PO_439

Re-stratification of patients with copy-number low endometrial cancer by Estrogen-related receptor γ and histomorphologic parameter

Xite Lin[1,2] Xiaodan Mao[1,2] Pengming Sun[1,2*]
1.Fujian Medical University
2.Fujian Maternity and Child Health Hospital

Background The Endometrial Cancer (EC) molecular typing has limitations in risk and prognosis stratification, particularly for the copy number low (CNL) subtype with variable outcomes. Estrogen-related receptors (ERRs) have emerged as potential biomarkers, yet their role in EC molecular subtypes remains underexplored. This study evaluates the role of ERRs combined with histomorphologic parameters for refined stratification.
Material and Methods Retrospective analysis of two cohorts: the TCGA cohort (n=232) and a Fujian single-center cohort (n=95). Primary outcomes: Prognostic value of ERRγ expression and histomorphologic features for progression-free interval (PFI) and disease-free interval (DFI). Secondary outcomes: Development of a molecular-histomorphologic (MH) risk model. TCGA molecular subtypes were determined via next-generation sequencing. ERRγ expression was assessed via RNA sequencing (TCGA) and immunohistochemistry (Fujian cohort). Additionally, several histomorphologic parameters were assessed on hematoxylin and eosin (H&E) stained slides. LASSO regression, Cox analyses and Kaplan–Meier survival curves were employed.
Results In the TCGA cohort, low ERRγ expression was significantly associated with poorer PFI (p=0.021) and DFI (p=0.004) in CNL subtype. Traditional clinicopathological criteria showed limited utility in CNL tumors, while histomorphologic parameters like intratumoral tumor infiltrating lymphocytes (iTILs) and tumor budding (TB) demonstrated significant associations with survival. Integrating ERRγ expression, TB, and iTILs into a MH risk model effectively stratified CNL patients into high- and low-risk groups. Patients in the MH high-risk group exhibited significantly worse PFI (p=0.0023) and DFI

(p<0.0001) compared to the low-risk group. In the Fujian cohort, low ERRγ expression was significantly associated with the presence of TB (p<0.001).
Conclusion This study highlights the histomorphologic and molecular diversity of EC subtypes. ERRγ serves as a critical prognostic biomarker for CNL-EC, and its integration with histomorphologic features (TB, iTILs) is utilized for risk stratification. The MH risk model offers a clinically actionable tool for personalized management, and has the potential to improve patient management and guide therapeutic decisions.

PO_440

Clinical Biomarker-Driven Prognostication: Validation of a Novel Risk Assessment System in Endometrial Cancer

xinyuan Zhang
Chongqing University Cancer Hospital

Objective To develop and validate a nomogram model for the prevention and treatment guidance of endometrial cancer and provide more accurate survival outcome prediction for patients with endometrial cancer.
Methods The retrospective observational cohort derived from the Chongqing University Cancer Hospital between Jan 1,2018 and Dec 31,2021(n=721).The data underwent systematic prognostic factor screening through algorithmic integrationof multivariable Cox proportional hazards regression and least absolute shrinkage and selection operator(LASSO) based feature selection protocol to model nomogram.Concordance index (C-index),area under the receiver operating characteristic(AUC),calibration curve,and decision curve analysis(DCA) were quantified to assess the model robustness.
Results The prognostic architecture was constructed through LASSO-Cox algorithmic integration,incorporating 10 clinico-pathological determinants:age,Karnofsky Performance Status(KPS),FIGO stage,differentiation,surgery,radiotherapy,Hemoglobin(Hb),Carcinoembryonic antigen(CEA),Interleukin-6(IL 6),and Estrogen receptor(ER).The area under the curve(AUC)values for the 1-,3-,5-year survival prediction in the training set were 0.910,0.884,0.884 respectively.The AUC values for the 1-,3-,5-year survival prediction were 0.765,0.808,0.824 in the validation set respectively.The calibration curves revealed favorable concordance between nomogram-predicted and observed outcomes in both training and validation cohorts. Decision curve analysis (DCA) further demonstrated the superior net clinical benefit of the nomogram compared to alternative risk stratification strategies.The model exhibited robust discriminative performance, with time-dependent area under the curve (AUC) and concordance index (C-index) values consistently surpassing 0.75. Notably, the nomogram outperformed the conventional TNM staging system in clinical utility assessments.
Conclusion Our predictive model offers a convenient and accurate tool for clinicians to estimate the prognosis of EC patients,which can aid clinicians in implementing personalized therapeutic decision-making to optimize patient management.

PO_441

纳米炭混悬液在早期子宫内膜样腺癌前哨淋巴结活检中的有效性及可行性研究

张睿 郭瑞霞*
郑州大学第一附属医院

目的 探讨应用纳米炭混悬液行前哨淋巴结(SLN)活检在早期子宫内膜样腺癌手术中的应用，评估其有效性及可行性，为临床手术方案的选择提供参考依据。
方法 回顾性收集 2021 年 6 月至 2024 年 12 月就诊于郑州大学第一附属医院妇科行腹腔镜或机器人辅助腹腔镜下子宫内膜癌全面分期手术的早期子宫内膜癌患者，所有患者均以全面分期手术为初始治疗，术前已有病理学证据证实为子宫内膜样腺癌，且术中采用宫颈注射法行前哨淋巴结活检术并同时行系统性淋巴结清扫，术中记录前哨淋巴结显影情况及位置并送病理学检查。
结果 最终纳入研究的 138 例患者中有 126 例成功检出 SLN，总检出率为 91.30%(126/138)。共检出 470 枚 SLN，人均 SLN 为 3.7 枚，其分布以髂外血管区为主(43.40%)，其余分布位置依次为：闭孔区(26.81%)、髂总血管区(15.11%)、腹主动脉旁区(8.30%)、髂内血管区(6.81%)、骶前区(1.35%)、宫旁区(0.21%)。126 例患者中合并淋巴结转移患者共 10 例，SLN 活检成功识别 9 例，最终得出灵敏度为 90%(9/10)，假阴性率 10%(1/10)，阴性预测值 99.15%(116/117)，准确度为 99.21%(125/126)。
结论 以纳米炭混悬液为示踪剂行前哨淋巴结活检在早期子宫内膜样腺癌的手术治疗中能准确反映淋巴结转移状态，操作简便，使用安全，值得在临床治疗中推广。

PO_442

Development and validation of RNA binding protein-applied prediction model for endometrial cancer

Wenjie Hou*
The fourth Affiliated Hospital of Soochow University

Background Recently, it has been demonstrated that the disturbance of RNA-binding proteins (RBPs) is closely related to the occurrence, progression and prognosis of cancers. However, the role of RBPs in endometrial cancer (EC) remains to be explored.
Methods We obtained the RNA sequencing data and clinical data of EC patients from the Cancer Genome Atlas (TCGA) database. The prognostic values of RBP-coding genes were analyzed by univariate COX regression analysis. Lasso regression analysis and multivariate COX regression analysis were conducted to construct a risk-prediction model based on the prognosis-related genes. The risk score of each patient was calculated according to the model. The predictive value of the model was assessed by Kaplan-Meier survival analysis and COX regression analysis. The potential function of differentially expression RBPs genes was further explored using GO and KEGG functional enrichment analyses. In addition, the correlation between the identified signature and tumor immunity was further explored.
Results A total of 284 differentially expressed RBP genes (DE-RBPs) were identified between cancer tissues and normal tissues. Among the DE-RBPs, 13 were found to be associated with the prognosis of EC patients. A six-gene (CD3EAP, EPHB6, CIRBP, LTK, ADARB2, RBPMS) model was ultimately identified by LASSO regression and multivariate COX regression analyses for the survival prediction of EC patients. The estimated risk score was independent prognostic for EC. A nomogram combining risk score and other clinical features was thus constructed for more accurate survival prediction.
Conclusions RBPs play important roles in the occurrence and development of EC, and can be used as a prognostic signature for EC.

PO_443

The prognostic and clinical significance of cervical stromal invasion in endometrial cancer: a multicenter retrospective study

Yang Yang
Qilu Hospital of Shandong University

Background In 2009, the International Federation of Gynecology and Obstetrics (FIGO) revised the staging of endometrial cancer (EC), limiting stage II disease to cases with cervical stromal involvement (CSI). While CSI has traditionally been regarded as an adverse prognostic factor, its independent impact on survival outcomes in early-stage EC remains controversial. This study aimed to assess the prognostic significance of CSI in a large, multi-institutional Chinese cohort.
Methods Patients diagnosed with FIGO 2009 stage I–II endometrial cancer who underwent comprehensive surgical staging between 2000 and 2019 across nine regional medical centers in China were retrospectively analyzed. The primary endpoint was progression-free survival (PFS); overall survival (OS) was the secondary endpoint. Survival outcomes were compared between CSI and non-CSI groups using multivariable Cox regression, propensity score matching (PSM), and stratified subgroup analyses.
Results A total of 7,383 patients were included in the analysis, with 524 patients in the CSI group and 6,859 in the non-CSI group. Patients with CSI demonstrated significantly lower 5-year PFS (91.1% vs. 94.9%, P<0.001) and OS (94.6% vs. 97.0%, P=0.002) in unadjusted comparisons. However, in multivariable Cox regression models adjusting for key clinicopathological confounders, CSI was not found to be an independent prognostic factor for either PFS (hazard ratio [HR] 1.31, 95% confidence interval [CI] 0.94–1.81, P=0.106) or OS (HR 1.33, 95% CI 0.88–2.00, P=0.171). Similar findings were observed in the PSM cohort (n=1,046), where 5-year PFS (92.3% vs. 93.7%, P=0.49) and OS (95.0% vs. 95.7%, P=0.79) did not significantly differ between CSI and non-CSI groups. Subgroup analyses further confirmed the lack of prognostic significance of CSI across both aggressive and non-aggressive histological subtypes.
Conclusion After adjusting for confounders, CSI was not an independent prognostic factor for survival in early-stage EC. These findings suggest that CSI may warrant reconsideration in future FIGO staging revisions. Prospective multi-center studies are needed to confirm these results and optimize risk stratification in EC management.

PO_444

Analysis of the association between inflammation-related indexes and endometrial cancer and assessment of their predictive value for myometrial infiltration

Yuqi Gao[1] Sitong Xin[1] Shufei Li[1] Fengdan Wang[1] Yu Han[1] Yuangang Guo[1] Yang Xu[1] Jing Zhao[1] Bo Li[1*] Xiaodan Sun[2]

1.Jilin University 2.Jilin Provincial Cancer Hospital

Aim Endometrial cancer (EC) is a group of epithelial malignant tumors occurring in the endometrium, which is one of the most common malignant tumors in the female reproductive system. Myometrial infiltration (MI) of EC refers to the extent of invasion of cancer cells from the endometrium into the myometrium. MI is associated with the staging and prognosis of EC patients and is an important basis for selecting treatment options. Many studies have shown that inflammatory mechanisms are closely related to the development of cancer. Inflammation-related indicators are derived parameters based on routine blood tests that reflect the body's inflammatory state and immune response. In recent years, they have received widespread attention in clinical diagnosis and disease monitoring. The aim of this study is to investigate the association of inflammation-related indices [neutrophil-to-lymphocyte ratio (NLR), monocytes-lymphocyte ratio(MLR), neutrophil to monocyte-lymphocyte ratio(NMLR), systemic immune inflammation index(SII), and systemic inflammation response index(SIRI)] with EC and to assess its predictive value for the depth of MI.

Methods 15,476 female subjects aged ≥ 20 years in the NHANES database from 1999 to 2020 were included in this study, including 15,334 non-tumor patients and 142 EC patients. Selection bias was reduced using a 1:2 propensity score matching (PSM) approach. Binary logistics regression analysis was performed to determine the association between inflammation-related indices and EC. The retrospective study included 290 patients who underwent radical surgery at Jilin Provincial Cancer Hospital from August 2015 to August 2024 and were diagnosed with EC by postoperative pathology. Correlation and Logistic regression analyses were performed, and a nomogram for predicting MI of EC was plotted based on the results. The predictive performance of the nomogram was assessed by area under the curve (AUC) of the subjects' operating characteristics, calibration curves, and decision curve analysis (DCA).

Results After PSM and controlling for all potential confounders, multivariate logistic regression analysis showed that NLR and NMLR were negatively associated with EC prevalence. The prevalence of EC in NLR and NMLR in the highest percentile group was 2.025 and 2.054 times that in the lowest quartile group, respectively. SIRI had the highest diagnostic efficacy, so age, Cancer antigen 125(CA125) and SIRI were selected as the final predictors for MI model construction. The AUC value of the nomogram graph was 0.744 (95% CI: 0.68-0.80, $P < 0.05$). In addition, the calibration curve of the column-line plot showed good agreement between the actual observations and the column-line plot predictions with an absolute error of 0.012. The DCA curve showed good positive net gain in the prediction model.

Conclusion NLR and NMLR were negatively associated with EC prevalence. More importantly, MLR, NLR, NMLR and SIRI were all associated with MI of EC, with SIRI having the strongest predictive power. These findings support the inclusion of these serologic parameters in routine diagnostic protocols to improve the accuracy of assessment of MI in EC. In addition, a nomogram graph model of MI depth constructed on the basis of age, 125, and SIRI had some accuracy and may be useful in predicting MI depth in patients with EC.

PO_445

脂质代谢异常与子宫内膜增生性病变及子宫内膜癌相关性分析——载脂蛋白 A 降低是其发生的早期事件

杨冰义[1] 陈晓军[1,2*] 程亚丽[1] 吴鹏飞[1]
1.复旦大学附属妇产科医院 2.上海市第十人民医院

目的 本研究拟前瞻性分析子宫内膜增生不伴非典型（Endometrial hyperplasia without atypia，EH）、非典型增生（Endometrial atypical hyperplasia，EAH）及子宫内膜癌（Endometrial carcinoma，EC）患者的脂质代谢水平，探索其随疾病进展的变化。

方法 自 2019 年 4 月至 2022 年 12 月，招募复旦大学附属妇产科医院子宫内膜病变门诊就诊的 1199 例患者，包括 235 例 EH， 491 例 EAH，473 例 EC（包括 347 例 G1 子宫内膜样癌，126 例 G2 子宫内膜样癌）。同时在体检中心招募 269 例女性作为对照组（Control group, CON），主要纳入标准为有规律的月经周期或处于绝经状态、无异常子宫出血、妇科检查或超声检查未提示异常。检测所有研究对象治疗前脂质代谢水平、空腹血糖、空腹胰岛素，对各研究组（EH、EAH、EC）与 CON 组在各指标的分布情况进行统计分析。对于定性变量，根据检验值选择合适的统计方法，包括卡方检验或 Fisher 精确检验；对于定量变量，则根据数据分布特征，采用独立样本 t 检验或 Kruskal-Wallis 秩和检验进行分析。同时，研究通过单因素和多因素 Logistic 回归模型，探讨了 EH、EAH、EC 组与 CON 组之间的危险因素。在统计学分析中，双侧检验的显著性水平设定为 $P < 0.05$；在进行四组间多重比较时，采用 Bonferroni 校正，将显著性水平调整为 $P < 0.0083$。

结果

1. CON，EH，EAH，EC 组的中位年龄分别为：36、36、32、36 岁。EH、EAH、EC 组的中位身体质量指数（Body mass index, BMI）均高于 CON 组（25.66 kg/m2，25.49 kg/m2，25.27 kg/m2 vs. 23.26 kg/m2，$P < 0.001$）。

2. 脂质代谢异常在 EC 的前驱病变 EH 中即已出现，其主要表现为高甘油三酯水平、低高密度脂蛋白水平、高低密度脂蛋白水平、低载脂蛋白 A（Apo-lipoprotein-A, APO-A）水平；这些改变在 EAH 及 EC 患者中仍持续存在。在校正年龄、BMI、绝经状态、高血压、糖尿病、胰岛素抵抗状态进行多因素分析后，EH、EAH、EC 组患者的血清 APO-A 仍低于 CON 组，比值比（Odds ratio, OR）分别为 0.050（95%置信区间[Confidence interval, CI] 0.026-0.0.097, $P < 0.001$），0.059（95%CI 0.033-0.106, $P < 0.001$），0.113（95%CI 0.064-0.198, $P < 0.001$）。

结果 脂质代谢异常是 I 型 EC 发生的早期事件，在 EH 阶段即已出现、并持续至 EC，其主要特征为血清 APO-A 水平降低。

PO_446

基于 MRI 的生境影像组学预测早期子宫内膜样腺癌深层肌层浸润的研究

石晟铭*
哈尔滨医科大学附属第二医院

目的 深层肌层浸润（DMI）是影响早期子宫内膜样腺癌（EAC）预后的关键因素，精准检测对治疗决策制定至关重要。本研究旨在构建融合 T2WI 亚区域影像组学特征与临床相关因素的联合模型，用于 EAC 患者 DMI 的检测，为临床提供客观、精准的评估工具。

方法 回顾性纳入 296 例经病理证实的 EAC 患者，均于治疗前完成 MRI 检查。按 7:3 比例随机分为训练集（207 例）与测试集（89 例）。采用 K - 均值聚类法将肿瘤区域划分为 3 个亚区域，从 T2 加权序

列中提取各亚区域的纹理特征、形态特征及一阶统计量共 1834 个。通过 LASSO 回归进行特征降维，筛选出 9 个最优影像组学特征。分别构建 4 种模型：仅纳入影像组学特征的影像组学模型、临床模型、基于肿瘤内异质性（ITH）评分的模型，以及整合 ITH 评分与临床因素的联合模型。采用受试者工作特征曲线（ROC）、检验校准曲线及决策曲线分析（DCA）评估模型性能，通过 10 折交叉验证验证稳定性。

结果 联合模型在测试集中表现最优，曲线下面积（AUC）达 0.818；训练集中 AUC 为 0.889，显著高于影像组学模型（AUC=0.797）和临床模型（AUC=0.704）。基于 ITH 评分的模型显示出良好的预后价值，训练集与测试集 AUC 分别为 0.878 和 0.811。校准曲线显示各模型预测概率与实际结果一致性良好（Hosmer-Lemeshow 检验 P>0.05），DCA 证实联合模型在阈值概率 0.2-0.8 范围内的临床净获益显著优于其他模型。

结论 整合 ITH 评分与临床因素的联合模型可有效预测 EAC 患者 DMI 状态，其性能优于单一影像组学或临床模型。该模型通过捕捉肿瘤亚区域的异质性特征，为临床早期识别 DMI 状态提供了无创评估工具，有助于实现个体化治疗决策，改善患者预后。

PO_447

Evaluation of lymph-vascular space invasion in early-stage endometrial cancer

Qiaoyan Kang engpengQu P*
Tianjin Central Hospital of Obstetrics and Gynecology

Objective To investigate the relationship between lymphovascular space invasion (LVSI) involving different numbers and types of vessels and the clinicopathological features and prognosis of patients with early-stage endometrial cancer.

Methods This was a retrospective study of patients with Federation International of Gynecology and Obstetrics (FIGO) 2009 stage I and II endometrial cancer who were divided into groups according to the number and type of vessels involved in LVSI, and the clinicopathological characteristics and prognosis of the patients were compared.

Results A total of 601 patients were included in this study. Patients with LVSI involving ≥ four (n=34, 5.66%)and < four (n=567, 94.34%) vessels exhibited significant differences in progression-free survival (PFS) (P=0.018), as well as in clinicopathological characteristics such as depth of myometrial invasion, cervical invasion, and histological type. LVSI involving ≥ four vessels was revealed to significantly increased the risk of reduced PFS (HR=3.475, 95% CI 1.012, 12.298, P=0.048). Patients with LVSI involving ≥ five (n=32, 5.32%) and < five (n=569, 94.68%) vessels had significant differences in PFS (P=0.012) and overall survival (OS)(P=0.050). There were no significant differences in PFS (P=0.48) or OS (P=0.48) between patients with LVSI involving different types of vessels. Patients with LVSI involving lymph vessels were more likely to have ≥4 vessels involved than those with LVSI involving blood or blood and lymph vessels (P=0.039) .

Conclusion LVSI involving ≥ four vessels increases the risk of recurrence, whereas LVSI involving ≥ five vessels increases the risk of death as well as recurrence.

PO_448

PPARγ 转录上调 CDO1 表达介导铁死亡在子宫内膜癌进展的机制研究

施雅诗[1] 孙蓬明[2*]
1.福建医科大学 2.福建省妇幼保健院

研究目的 子宫内膜癌是最常见的女性肥胖相关性肿瘤之一，其进展的分子机制尚未完全阐明。而铁死亡作为一种新型铁依赖性细胞死亡形式，在肿瘤发展中扮演重要角色，但其在子宫内膜癌中的调控机制尚不明确。本研究旨在探究过氧化物酶体增殖物激活受体 γ（PPARγ）是否通过转录调控半胱氨酸双加氧酶 1（CDO1）诱导铁死亡，从而抑制 EC 进展，为靶向治疗提供新策略。
材料与**方法** 本研究使用人 EC 细胞系（Ishikawa、HEC-1A）及 BALB/c 裸鼠移植瘤模型（n=6/组），通过 PPARγ 激动剂罗格列酮、CDO1 siRNA 敲低、过表达载体进行干预；结合 CCK-8 增殖实验、Transwell 迁移/侵袭分析、ChIP-seq、双荧光素酶报告基因、脂质过氧化（C11-BODIPY/4-HNE）及 GPX4/GSH 检测，在体内外验证 PPARγ/CDO1/铁死亡轴的调控机制。
结果 PPARγ 激活显著抑制 EC 细胞增殖、迁移及侵袭，并诱导脂质过氧化积累与 GPX4 下调；ChIP-seq 联合双荧光素酶实验证实 PPARγ 直接结合 CDO1 启动子区，使其转录活性增强，CDO1 表达上调；敲低 CDO1 可逆转罗格列酮的抑癌作用，而过表达 CDO1 则通过耗竭半胱氨酸、降低 GSH 直接触发铁死亡；裸鼠实验中罗格列酮组肿瘤体积减小，伴随 CDO1 表达增加、GSH 表达增加。
结论 PPARγ 通过直接结合 CDO1 启动子促进其转录表达，诱导半胱氨酸耗竭与铁死亡，进而抑制 EC 进展。本研究首次揭示 "PPARγ-CDO1-铁死亡"调控轴在 EC 中的关键作用，为靶向铁死亡的 EC 治疗提供新靶点。

PO_449

KRT19 促进子宫内膜癌转移的机制研究

李冠初 陈刚*
华中科技大学同济医学院附属同济医院

研究目的 子宫内膜癌是高收入国家最常见的妇科恶性肿瘤，其发病率在全球范围内呈上升趋势。传统上，淋巴结切除术被认为是淋巴结评估的唯一选择。前哨淋巴结 (SLN) 活检技术的实施使手术的侵入性降低，提供了分期信息，同时降低了患者的发病率。在淋巴结阳性的子宫内膜癌患者中，盆腔淋巴结转移的大小与转移扩散的程度有关,由于约 40% 的 SLN 阳性子宫内膜癌患者在接受标准治疗后会出现复发，因此可能与复发风险较高的肿瘤相关的淋巴结特征变得非常重要。尽管淋巴转移的细胞量很少，但是与肿瘤的预后明显相关联，在我们的研究中发现转移灶中 KRT19+的肿瘤细胞比例上升。在本研究中，我们的目标是探究 SLN 阳性的子宫内膜癌患者的蛋白组表达差异，并对差异蛋白在临床和组织病理学特征中表达进行描述。基于本研究结果，可以为临床患者的诊断和治疗提供有价值的靶点。
材料与方法 本文收集患者的样本进行多色荧光染色，明确 KRT19 分子在肿瘤和正常细胞中的表达差异。建立长期培养的过表达和敲低 KRT19 的稳定子宫内膜癌细胞系，借助划痕实验、淋巴管成管实验检测其侵袭、促进淋巴管生成的能力在不同组别中的变化。
结果
1. 转录组数据表明，在转移灶中 KRT19 升高明显。
2. 对 SLN 阳性子宫内膜癌转移灶的样本进行的多色荧光染色发现，肿瘤细胞中相对于正常细胞中 KRT19 显著升高。
3. 对 KLE、Hec 1b 子宫内膜癌细胞进行敲低和过表达，划痕实验的结果表明，KRT19 对子宫内膜癌细胞的侵袭能力有正向作用。

4. 为了探究子宫内膜癌的转移途径，用子宫内膜癌细胞的培养上清液去干预 HELCs 人淋巴管细胞，实验结果表明，KRT19 对 HELCs 细胞形成淋巴管的能力呈增强作用。
结论 本研究描述了子宫内膜癌转移灶中的分子变化，对其影响肿瘤的侵袭转移能力进行了探究，表明 KRT19 促进了子宫内膜癌的侵袭能力，促进淋巴管的生成以有利于转移。基于这些结果，对于子宫内膜癌转移机制提出了新的见解，为提升晚期子宫内膜癌癌治疗效果提供潜在治疗方向。

PO_450

Estrogen promotes the proliferation and migration of endometrial cancer through the GPER-mediated NOTCH pathway

Meng Qi
Zhengzhou University

Objective This study aims to investigate the expression of GPER in EC, assess the impact of estrogen on the proliferation and migration of EC via GPER, and examine the potential role of GPER in mediating the NOTCH pathway to influence EC proliferation and migration.
Methods The expression of GPER and its correlation with clinicopathological features were investigated using clinical data. Cell proliferation was assessed through MTT and EdU assays, while cell migration ability was evaluated using wound healing and transwell assays. Western blot analysis was conducted to detect proteins associated with the GPER and NOTCH signaling pathways. Additionally, xenograft tumor models were established to investigate the potential role of estrogen in mediating the NOTCH pathway via GPER.
Results The results demonstrated a significant upregulation of GPER expression in EC, which was associated with clinical stage and metastasis. In vitro experiments provided evidence that estrogen promotes EC cell proliferation and metastasis by enhancing the expression levels of GPER, Notch1, and Hes-1 proteins. Conversely, knocking down or suppressing GPER effectively reverses these effects. Furthermore, treatment with JAG-1, an agonist for the NOTCH pathway, counteracts si-GPER's inhibitory impact on both proliferation and migration abilities of EC cells while increasing Notch1 and Hes-1 protein expression levels; however, it does not alter GPER expression. In vivo experiments have substantiated that estrogen facilitates EC proliferation via the GPER-mediated NOTCH pathway.
Conclusion Estrogen facilitates EC proliferation and metastasis through the GPER-mediated NOTCH pathway.

PO_451

Systematic Multiplatform Discovery of Methylation Markers Enables Non-Invasive Early Detection of Endometrial Cancer

Yan Cai[1] Shuchao Chen[2] Zhen Wu[2] Tong Shu[1] Hong Zheng[1*]
1.Key laboratory of Carcinogenesis and Translational Research (Ministry of Education/Beijing), Department of Gynecologic Oncology, Peking University Cancer Hospital & Institute
2.BioChain (Beijing) Science & Technology Inc.

Background Endometrial cancer is the most common gynecological malignancy, increasing 1%-3% annually. Current diagnostic approaches, such as hysteroscopy and biopsy, are invasive and unsuitable for population-wide early screening, needing robust, non-invasive biomarkers to enable timely detection.
Methods We conducted a comprehensive, multilayered screening for candidate DNA methylation markers leveraging whole genome bisulfite sequencing (WGBS) and targeted methylation panels on endometrial cancer and control tissues. Top candidate markers were further filtered, optimized, and validated using a two-stage design in cervical exfoliated cell samples: 148 ThinPrep Cytology Test (TCT) samples for discovery and model construction, followed by an independent validation in 80 additional TCT samples. Quantitative Methylation-Specific PCR (qMSP) was used to assess marker methylation patterns in these TCT samples.
Results Through systematic multi-platform and multi-cohort analysis, we identified a panel of three methylation markers, ZNF626, GRIA4, and SPDYA, which achieved high performance in early detection of endometrial cancer from cervical cytology samples. Independent validation in 80 TCT samples demonstrated outstanding sensitivity of the three-marker diagnostic model. The sensitivity of endometrial cancer in women over 50 years of age was 96.43%, and the specificity for benign endometrial disease was 78.57%. For women aged 50 or younger, the sensitivity and specificity reached 91.67% and 96.15%, respectively. Importantly, the approach achieved a 92.86% detection rate for stage I endometrial cancer.
Conclusion The study presents a systematically developed, non-invasive diagnostic approach for early endometrial cancer detection. This approach greatly enhances the potential for early intervention and improved clinical outcomes via simple cervical cytology sampling.

PO_452

Safety of Omitting Mechanical Bowel Preparation Before Surgery in Patients with Early-stage Cervical Cancer and Endometrial Cancer

Yifan Li
Cancer Hospital Chinese Academy of Medical Sciences

Objective To explore the impact of omitting mechanical bowel preparation (MBP) before surgery on postoperative safety in patients with early-stage cervical cancer and endometrial cancer.
Materials and Methods A retrospective analysis was conducted of patients with cervical cancer or endometrial cancer who underwent surgery in the Department of Gynecologic Oncology, Cancer Hospital, Chinese Academy of Medical Sciences from January 1, 2024 to December 31, 2024. Patients with FIGO2018 stage IA2-IIA2 cervical cancer and FIGO2014 stage I-II endometrial cancer, as determined before surgery, were included. All patients fasted 6 hours before surgery and abstained from clear liquids 2 hours before surgery. Patients who received oral hypertonic electrolyte solution before surgery were assigned to the MBP group, while those who did not receive any form of bowel

preparation were assigned to the non-MBP group. Postoperative complication rates and indicators of intestinal function recovery were compared between the two groups.
Results A total of 221 patients were included in the study, including 121 with cervical cancer and 100 with endometrial cancer. The mean age was 52.48 ± 11.00 years, and the mean BMI was 25.1 ± 3.6 kg/m². There were 127 patients in the MBP group and 94 in the non-MBP group. The proportion of patients with ASA grade III (8.5% vs 1.6%, p=0.020) and the proportion of cervical cancer patients (62.8% vs 48.8%, p=0.039) in the non-MBP group were significantly higher than those in the MBP group. Other demographic and baseline characteristics were comparable between the two groups. Both the MBP group and the non-MBP group underwent pelvic lymph node dissection. A total of 54.3% and 56.4% of patients in the two groups received para-aortic lymph node dissection (p=0.434), 54.3% of patients in both groups underwent radical/modified radical hysterectomy (p=0.750), and 14.2% and 10.6% of patients received omentectomy (p=0.435), respectively. There were no intestinal injury-related complications in either group after surgery. Two patients (1.6%) in the MBP group had urinary system injuries, while no urinary system injuries occurred in the non-MBP group. The incidence of postoperative hypokalemia (9.6% vs 22%, p=0.014), hypophosphatemia (23.4% vs 45.7%, p<0.001), and postoperative infection (3.2% vs 11%, p=0.031) in the non-MBP group was significantly lower than that in the MBP group. The median time to flatus in the non-MBP group was significantly shorter than that in the MBP group (2 days vs 3 days, p<0.001). There were no statistically significant differences between the two groups in terms of postoperative intestinal obstruction (4.3% vs 3.9%, p=1.000), incision complication (5.3% vs 3.1%, p=0.500), postoperative thrombosis (4.3% vs 7.1%, p=0.376), and unplanned readmission within 30 days (1.1% vs 6.3%, p=0.082).
Conclusion Omitting mechanical bowel preparation does not affect the perioperative safety of patients with early-stage cervical cancer and endometrial cancer. It reduces the incidence of postoperative electrolyte disorders and infections, and shortens the time to flatus. Therefore, it is recommended for patients with early-stage cervical cancer and endometrial cancer scheduled for surgery.

PO_453

Distinguishing between double primary synchronous cancer of the endometrium and fallopian tube from primary tumor metastasis: A case report and literature review

Pingping Su Hongli Liu* Lili Zhang Liangkai Zheng Xinfa Huang
Department of Gynecology, Women and Children's Hospital, School of Medicine, Xiamen University

Objectives This report retrospectively analyzed a case of primary fallopian tube carcinoma with endometrial metastases to explore the clinical features, diagnosis, prevention, and expecially the differential diagnosis from double primary synchronous cancer of the endometrium and fallopian tube.
Case A 55-year-old postmenopausal woman presented with irregular vaginal bleeding for two years. A cervical cancer screening indicated the patient was negative for human papillomavirus infection. However, a cytology test (ThinPrep) found atypical gland cells, and a colposcopic biopsy suggested chronic cervicitis. A hysteroscopic examination with curettage identified a small number of atrophic endometrial glands. Vaginal color Doppler ultrasound and a plain pelvic magnetic resonance scan with enhancement showed a cystic, solid mass shadow in the right adnexal area. Right fallopian tube cancer was suspected.
Results The patient underwent transabdominal extrafascial hysterectomy, bilateral salpingo-oophorectomy, pelvic and para-aortic lymphadenectomy, omental resection, percutaneous peritoneal biopsy (multi-point peritoneal biopsy), and bilateral ovarian arteriovenous high junction. The postoperative diagnosis was high-grade serous carcinoma of the fallopian tube (stage IIA T2aN0M0). The patient underwent postoperative paclitaxel and carboplatin chemotherapy and has since shown no signs of recurrence.

Conclusion For patients with gynecologically malignant tumors, rare types, such as simultaneous double primary gynecological malignant tumors, should be considered as a differential diagnosis. Pathological diagnostic technology is advancing; thus, the rate of diagnosing fallopian tube cancer is increasing. Correctly distinguishing multiple synchronous primary malignant tumors from primary tumor metastasis is crucial for selecting the most appropriate treatment plan to maximize survival benefits and minimize recurrence.

PO_454

子宫内膜癌及不典型增生保育治疗对体外受精妊娠结局的影响：基于倾向性评分匹配的研究

刘昕
天津市中心妇产科医院

研究目的 本研究旨在利用倾向性评分匹配方法，在控制混杂因素尤其是内膜厚度的基础上，比较子宫内膜不典型增生或早期子宫内膜癌患者接受保育治疗后，与同期无子宫内膜病变患者行体外受精助孕治疗的妊娠结局差异。

材料与方法 回顾性收集了 2014 年 1 月至 2024 年 1 月在天津市中心妇产科医院生殖医学中心进行 IVF/ICSI 治疗 57 名子宫内膜疾患保育治疗患者（EEC 11 例，AEH 46 例）和 13790 名无子宫内膜异常迹象的患者病历。对 57 例子宫内膜保育治疗患者与无子宫内膜异常迹象的对照女性以 1:4 的比例进行倾向性评分匹配，比较其妊娠结局。

结果 57 例子宫内膜疾患保育治疗患者倾向性评分匹配成功 43 例保育治疗组，158 例对照组，共 223 个卵巢刺激周期，347 个移植周期。保育治疗组与对照组的临床妊娠率分别为 42.9%与 41.3%（p=0.835），活产率分别为 28.6%与 30.5%（p=0.781）。二元 logistic 回归分析显示在母亲取卵年龄<35 岁时子宫内膜厚度与活产率显著相关（校正 OR: 1.468， 95% CI：1.041-2.071）。

结论 本研究采用倾向性评分匹配控制内膜厚度后，子宫内膜不典型增生或早期子宫内膜癌保育治疗患者与无内膜病变的对照患者进行比较，总体妊娠结局无显著差异，考虑在控制内膜损伤后的厚度的影响因素后保育治疗并未显著降低 IVF 助孕的妊娠结局。在年轻患者保育治疗过程中，注重内膜厚度的保护可能进一步提高保育治疗后的活产机会。

PO_455

PTEN Deficiency Unveils Oncogenic Non-Canonical Pathway Activation and Novel Therapeutic Targets in Endometrial Cancer

Xiangyi Kong[1] Qiong Wang[2] Hongming Song[3] Li Wang[2] Lingrui Li[2] Xiaonan Hou[2] Renuse Santosh [2] Ran Cheng[2] Khan Md Kamrul Hasan[2] Jidong Wang[4] Mangalaparthi Kiran [2] Lin Fang[5] Lotan Tamara Levin[6] Park Ben Ho [6] Weroha S. John [2] Pandey Akhilesh [2] Xinyan Wu[2] Huaijun Zhou[1*]

1. Nanjing Drum Tower Hospital Clinical College of Nanjing Medical University
2.Mayo Clinic
3.The Affiliated Hospital of Qingdao University
4.Jinan Central Hospital Affiliated to Shandong University
5.Shanghai Tenth People's Hospital, Tongji University School of Medicine
6.The Johns Hopkins Medical Institutions

Background PTEN loss is commonly observed across multiple cancer types and leads to disruptions in cellular signaling, driving tumor development through activation of the PI3K-AKT pathway. However, therapies targeting this pathway face challenges due to intricate signaling crosstalk and the activation of compensatory mechanisms. This study employed quantitative proteomics and phosphoproteomics to investigate how PTEN deficiency impacts both canonical and noncanonical pathways and suggest a novel combined treament in endometrial cancer.

Methods Using PTEN knockout models and doxycycline-induced PTEN overexpression system, we evaluated the oncogenic impacts of PTEN loss and deficiency in different cancer cell lines. Quantitative proteomic and phosphoproteomic analyses were employed to map the signaling alterations induced by PTEN loss or induced overexpression of PTEN mutants. Multiple cancer cell lines and PDX models with PTEN deficiency treated with a variety of kinase inhibitors were used to validate the downstream signaling alterations and synergistic potentials of combination treatments.

Results PTEN deficiency promotes oncogenic phenotypes in normal breast epithelial cells and drives more aggressive behavior in breast and endometrial cancer cells. Quantitative proteomics and phosphoproteomics analysis revealed that PTEN deficiency not only activates PI3K-AKT signaling, but also leads to a wide range of signaling alterations, including changes in cytoskeleton organization, cell cycle, and central carbon metabolism. In addition to canonical serine/threonine kinases, PTEN deficiency substantially elevates the activity of tyrosine kinases, including Src kinase. We revealed that Src activation, rather than the canonical AKT signaling pathway, is required for the PTEN deficiency mediated upregulation of EphA2, a receptor tyrosine kinase (RTK) implicated in cancer progression. Importantly, combined AKT and Src inhibition synergistically reduced cell proliferation and induced apoptosis in PTEN-deficient breast and endometrial cancer cells. This combination significantly suppressed tumor cell growth in 3D cultures of endometrial cancer PDX models with PTEN deficiencies.

Conclusion Our study demonstrates that PTEN loss activates both the canonical PI3K-AKT pathway and tyrosine kinase pathways. In particular, Src-mediated upregulation of EphA2 in PTEN-deficient cells reveals a key therapeutic vulnerability that combining AKT and Src inhibitors can enhance therapeutic efficacy and effectively overcome the resistance observed with AKT inhibition alone.

PO_456

子宫内膜中肾样腺癌伴滋养细胞分化 1 例并文献复习

王昭娣 凌开建*
海南省妇女儿童医学中心

目的 中肾样癌(mesonephroid—like adenocarcinoma．MLA)是于 2016 年首次被报道的好发于子宫体和卵巢的子宫内膜癌亚型，其发病率占所有子宫内膜癌的 1%。由于其生物学行为比常见的子宫内膜癌具有更强的侵袭性，易早期复发和转移。本文报道 1 例罕见晚期 MLA，旨在为诊治该肿瘤提供参考。
方法 本文报道 1 例我院收治的子宫内膜中肾样腺癌 IIIC2 期患者。患者女，57 岁，因“绝经 3 年，不规则阴道流血 4 月”于当地医院行分段诊刮术，病理提示（宫颈管、宫腔）低分化子宫内膜样癌。子宫增大如孕 4+月, 影像学提示盆腹腔多发淋巴结转移。HCG918.1IU/L。分析其诊治过程、组织学特征、免疫组织化学并复习相关文献。
结果 于 2025 年 5 月 15 日在我院行经腹子宫内膜癌细胞减灭术（经腹筋膜外全子宫切除术+双侧附件切除术+盆腔淋巴结切除术+高位腹主动脉旁淋巴结切除术+大网膜切除术+宫旁病灶切除术+乙状结肠病灶切除术）。子宫增大如孕 4+月大，病灶穿透浆膜。右侧子宫动脉与输尿管交叉部位可见 3cm 大小病灶。盆腔转移淋巴结最大直径 7.5cm，腹主动脉旁淋巴结多个增大直径达 3.5cm。术后病理提示子宫内膜中肾样腺癌，病灶累及宫颈间质伴有局灶滋养细胞分化。免疫组化 CD10 及 GATA3 部分阳性，ER 及 PR 阴性。子宫内膜癌分子分型检测为 POLE 突变型。术后予以全身化疗序贯同步放化疗，目前疾病控制稳定。搜索文献未发现 MLA 伴有滋养细胞分化及伴有 POLE 突变的病例报告。文献多报道为合并 KARS 基因突变或分子分型为 NSMP，中位生存期短，放化疗不敏感。
结论 子宫内膜 MLA 合并滋养细胞分化为罕见病例，该患者无规范诊疗方案可参考。滋养细胞分化原因不明，是否影响 POLE 基因突变的放化疗敏感性有待观察。本例晚期患者合并 POLE 突变是否治疗期间加用免疫及靶向治疗值得进一步探索后期需继续对患者进行随访，观察治疗疗效，延长患者的生存时间。

PO_457

RNA 剪接异常对子宫内膜癌免疫调节的潜在影响

曹敏悦
上海市第一妇婴保健院

RNA 剪接控制基因在转录水平的表达，可产生许多结构和功能各不相同的转录本。肿瘤相关的剪接异常归因于剪接因子、信号通路、表观遗传学改变、环境因素在内的 RNA 剪接调控因子的改变。肿瘤相关剪接失调极大地影响了细胞的发育，在多种类型的癌症的增殖、侵袭、迁移、代谢或凋亡等方面发挥着重要作用。EC 的 MSI-H/dMMR 分型是免疫治疗的主要受益群体，并且目前缺少对肿瘤相关剪接失调与肿瘤免疫之间的复杂关系进行全面的文献回顾。因此，在这里，我们不仅讨论了 RNA 剪接的调控因素及其对肿瘤进展的影响，还详细描述了 RNA 异常剪接对肿瘤细胞及其微环境免疫调控的潜在影响以及 EC 中 RNA 剪接有关的靶点。这对优化免疫疗法在 EC 抗癌治疗中的应用至关重要，并为癌症的治疗和潜在预后标志物提供一个全新的视角。

PO_458

Novel RNA Splicing roles of SF3 Family in Endometrial Cancer

Yiqin Zhang
Shanghai First Maternity and Infant Hospital

Members of the SF3 family are crucial RNA splicing factors that play a significant role in regulating gene expression via alternative splicing (AS). Dysregulation of RNA splicing has been implicated in various cancers, resulting in the overactivation of oncogenes, aberrant signaling pathways, and altered responses to cancer therapies. Endometrial cancer (EC) threaten strongly to women's health and well-being. The AS functions of SF3 family members may hold considerable clinical relevance in the progression and therapeutic response of EC. This review summarizes current research on SF3 family members across different cancers, highlighting their associations with P53 mutations, hormonal influences, and cell signaling pathways pertinent to EC. Our findings aim to provide new insights for clinical diagnosis and therapeutic strategies in managing EC.

PO_459

AQB 通过表观调控 EZH2 介导的 H3K27me3 上调 PR 表达增敏子宫内膜癌孕激素治疗的机制研究

刘佳乐 冯欣宇 王颖梅*
天津医科大学总医院

研究目的 子宫内膜癌（EC）发病率逐年上升，并呈年轻化趋势，提高 EC 保育患者孕激素治疗敏感性是目前的临床重点难点。孕激素受体（PR）表达下调是 EC 孕激素抵抗的核心步骤，但具体机制不完全明晰且缺乏有效的靶向增敏策略。EZH2 主要通过催化 H3K27me3 表观沉默相关抑癌基因，但研究发现其通过甲基转移酶功能和转录激活功能等多种途径参与肿瘤进展，具体机制需进一步阐明。通过对 TCGA 数据库分析发现在 EC 中 EZH2 高表达与 PR 低表达显著相关，提示 EZH2 可能通过调控 PR 表达参与 EC 孕激素抵抗，有望成为 EC 孕激素增敏关键靶点，具体分子机制有待进一步探究。

材料与方法 IHC 实验检测 EC 组织中 EZH2 和 PR 的表达水平。使用 EC 孕激素耐药细胞系 Ishikawa-MR 及其亲本 Ishikawa 进行体外实验。使用小干扰/质粒敲低/过表达 EC 细胞系中的 EZH2。WB 及 RT-qPCR 实验检测 EC 细胞中相关蛋白表达水平。CCK8 实验检测 EC 细胞孕激素 IC50。EdU 实验检测细胞增殖能力，Transwell 实验检测细胞侵袭、迁移能力，流式细胞术检测细胞周期和凋亡。CHIP-qPCR 检测 EZH2 和 H3K27me3 在 PR 启动子区的富集水平。双荧光素酶报告基因实验验证 EZH2 对 PR 的转录调控作用。体内外实验对比孕激素、小分子抑制剂 AQB 单药治疗及二者联合用药对 EC 细胞的抑制作用。

结果 临床 EC 组织中 EZH2 高表达与 PR 低表达显著相关，均与 EC 患者不良预后相关。EC 细胞中敲减 EZH2 后恶性生物学行为减弱，同时 PR 表达水平上调且孕激素敏感性增强，敲减 EZH2 后 EZH2 和 H3K27me3 在 PR 启动子区结合同步减少，表明 EZH2 通过催化 H3K27me3 沉默 PR 参与 EC 细胞孕激素抵抗。但在孕激素作用下 EC 细胞中 EZH2 和 PR 表达均下调，提示孕激素作用下 EZH2 可能通过多种途径参与 PR 调控。敲减 SUZ12 排除 EZH2 甲基转移酶功能影响后使用孕激素处理 EC 细胞，发现 PR 启动子区 EZH2 结合下调，双荧光素酶报告基因实验证实孕激素通过抑制 EZH2 的转录激活功能下调 PR 表达。体外实验证实 AQB 通过阻断 HOTAIR 募集 PRC2 至 PR 启动子区减少 H3K27me3 上调 PR 表达促进 EC 细胞孕激素敏感性。体内实验表明，与孕激素及 AQB 单药组相比，AQB 联合孕激素组可显著抑制皮下瘤生长。

结论 EZH2 通过甲基转移酶功能和转录激活功能共同调控 EC 进展与药物治疗反应，其主要通过催化 H3K27me3 表观沉默 PR 参与孕激素抵抗，而孕激素显著抑制 EZH2 转录激活功能进一步抑制 PR 表达。AQB 通过阻断 HOTAIR 对 PRC2 的招募抑制 H3K27me3 上调 PR 表达增敏 EC 孕激素治疗。

PO_460

高级别浆液性子宫内膜癌卵巢转移病例分析及探讨

陈俊妃 凌开建*
海南省妇女儿童医学中心

目的 探讨子宫和卵巢同时发生浆液性癌时，究竟是子宫内膜癌伴卵巢转移，还是卵巢癌起源子宫转移，肿瘤起源与治疗的相关探讨。

方法 本研究回顾分析 1 例子宫内膜癌卵巢转移病例资料。患者因盆腔包块入院，术前影像学提示：宫腔内及双侧附件区恶性病变占位征象，考虑子宫内膜癌合并双侧卵巢癌，多发远处转移，腹腔积液。肿瘤指标结果 CA125 偏高,其余未见异常。诊断性刮宫术后病理提示：（子宫内膜）结合形态及免疫组化，送检组织符合浆液性癌。拟通过诊治过程肿瘤起源的分析及检测协助治疗策略的制定。

结果 行经腹全子宫切除术+双侧附件切除术+盆腔淋巴结切除术+腹主动脉旁淋巴结切除术+大网膜切除术+直肠旁沟病灶清除术。术中探查：血性腹水约 400ml，大网膜未见增厚，未见明显病灶，子宫增大，如孕 3+月，表面光滑，子宫底部与部分肠管致密性粘连，右侧卵巢增大约 10cmx9.5cm,表面呈菜花状，右侧附件与部分肠管及盆腹壁致密粘连，左侧卵巢增大，约 4*5cm，表面可见多房囊实性包块，与肠管及盆腹部致密粘连，双侧输卵管增粗，盆底腹膜、子宫及膀胱，乙状结肠表面未见明显病灶，直肠旁沟腹膜可见一大小约 1*1cm 质硬结节，双侧盆腔淋巴结肿大，腹主动脉旁淋巴结肿大，探查肝脏、脾及阑尾表面未见明显异常。

术中冰冻结果 （右侧附件）冰冻切片显示为腺癌。术后病理提示子宫体、宫颈、阴道壁及双侧卵巢和输卵管中均见浆液性癌（结合临床影像学及组织形态学，考虑癌组织来源于子宫体），（送检腹水）见到成堆排列的肿瘤细胞，形态符合腺癌。进一步免疫组化结果 （子宫体癌组织）：Her-2（2+）。基因检测提示 TP53 基因突变，HER2 扩增阴性。完善肿瘤组织起源检测，结果显示右侧卵巢肿瘤样本最有可能来源于卵巢组织，即卵巢癌可能性大。但是，结合病史、影像学、组织形态特征，最终诊断子宫内膜癌卵巢转移，术后予 TC 方案化疗 3 周期后补充放疗，后续化疗联合贝伐珠单抗及 PD-L1 靶向治疗，目前病情稳定（CR），免疫靶向维持中。

结论 肿瘤起源在临床诊断中有一定难度，此类患者临床容易误诊，因此正确认识肿瘤起源对临床治疗具有重要的临床意义。

PO_461

Non-invasive Prediction of Progestin Resistance Endometrial Cancer in Fertility-Sparing Treatment Using Ultra-Performance Liquid Chromatography Mass Spectrometry Urine Metabolomics

Junyu Chen[1] Dongyan Cao[2*]
1.Qilu hospital of shandong university
2.Peking union medical college hospital

Objective The aim of our study was to identify distinct urinary metabolic patterns between progestin-sensitive (PS) and progestin-resistant (PR) endometrial cancer (EC) patients. We sought to develop a non-invasive biomarker model for PR patients to aid in selecting the most suitable regimen before initiating fertility-sparing treatment.
Method The study involved 17 PS and 11 PR patients. Patients' clinical characteristics and laboratory indices were recorded to make sure the two groups are matched. Moring midstream urine samples were collected after at least 8 hours fast. We utilized ultraperformance liquid chromatography mass spectrometry (UPLC-MS) to analyze their urine samples. Analytical techniques were employed to determine distinct metabolites and altered metabolic pathways. Metabolites with P-value ≤ 0.05, Variable Importance in the Projection (VIP) score ≥ 1, and fold-change ≥ 1.2 were considered significant. KEGG pathway enrichment were applied to illustrate the altered metabolic pathways. Receiver operating characteristic (ROC) analyses and 10-fold-cross-validation were conducted to identify and confirm the efficacy of potential biomarker models.
Results A total of 42 urine metabolomes were identified to be significantly different in PS and PR groups. These metabolites were associated with several key metabolic pathways, including galactose metabolism, sphingolipid metabolism, biosynthesis of unsaturated fatty acids, amino sugar and nucleotide sugar metabolism, arachidonic acid metabolism and purine metabolism. A biomarker panel consisting of Aspartyl-Glutamine, Dihydrojasmonic acid, Indole, 7-Hydroxy-8-O-methylaloin B, and CurdioneBaicalin demonstrated the best predictive ability. This panel achieved an Area Under the Curve (AUC) of 0.955 in the training set and 0.838 in the 10-fold-cross-validation process, with sensitivity and specificity values of 0.909 and 0.985, respectively.
Conclusion The urinary metabolomic analysis highlighted metabolic distinctions between PS and PR patients, leading to the development of a non-invasive predictive model for PR patients that demonstrates substantial diagnostic potential.

PO_462

一项基于免疫微环境的子宫内膜癌预后模型

李立伟 王建六*
北京大学人民医院

目的 肿瘤免疫微环境(tumor immune microenvironment, TIME)与恶性肿瘤细胞的相互作用在肿瘤的发生发展中起着至关重要的作用。本研究旨在建立并验证一种基于 TIME 特征的预后模型，用于子宫内膜癌（endometrial cancer，EC）患者的预后预测和个体化治疗指导。
方法 纳入 2018 年 1 月至 2022 年 12 月期间在北京大学人民医院接受手术和 TIME 检测的 67 例 EC 患者。根据 CD3+细胞和 CD8+细胞的密度建立预后模型。另外选取 200 例 EC 患者作为验证集，使用免疫组织化学（IHC）标记 CD3+细胞和 CD8+细胞，验证预后模型的准确性。
结果 (1): 来自北京大学人民医院的 67 例 EC 患者利用多重免疫荧光进行 TIME 检测，复发组与非复发组在 PD-L1+细胞、CD8+细胞、CD68+CD163+细胞、CD3+细胞和 CD56+细胞方面存在统计学差异。其中，CD3+细胞的差异最为显著（P=0.004）；(2): 来自 TCGA 数据库的 514 例 EC 患者，复发组与

非复发组在 CD8+细胞、调节性 T 细胞（Tregs）和活化树突状细胞（DC activated）方面存在统计学差异（P＜0.05），其中，CD8+细胞的差异最为显著（P=0.000）；(3) 选取 CD3+ cell 及 CD8+ cell 为建模因子，建立一个新的预后风险评分系统，分为 Cluster1(n=17)，Cluster2(n=39) 以及 Cluster3(n=11)。三组行 ROC 分析(P=0.001, AUC=0.810)及生存分析(P=0.006)具有统计学意义。(4) 新的评分系统在其余免疫细胞、分子分型及临床病理特征方面具有差异；(5) 另外选取北京大学人民医院的 200 例 EC 患者作为验证集，利用免疫组化（IHC）标记 CD3+细胞和 CD8+细胞，将这些患者分为 Cluster1，Cluster2 以及 Cluster3 进行生存分析，具有统计学意义（P=0.000），验证了该模型的准确性。

结论 这项研究基于 TIME 寻找出了与 EC 预后明显相关的两种免疫细胞，并基于此建立了一个 EC 风险评分系统，预测效能及准确性良好。

PO_463

子宫内膜小细胞神经内分泌癌

谢秋芳
兴文县人民医院

子宫内膜小细胞神经内分泌癌是一种预后较差的少见疾病，常常诊断时为晚期，主要治疗方式是手术+基于铂类的化疗或放疗。由于此类肿瘤极其少见，术前往往难以诊断，诊断过程中，需注意与小细胞非角化鳞状细胞癌、淋巴上皮瘤样癌、腺样基底细胞癌等进行鉴别诊断。治疗方案综合考虑了手术、化疗及放疗等多模态治疗策略，根据病情进展和患者体质个性化调整。临床工作中应注意多总结和随访以提高对该病超声及临床特征的认识。

PO_464

子宫内膜透明细胞癌

谢秋芳
兴文县人民医院

子宫内膜透明细胞癌是一种相对少见但具有高度侵袭性的妇科恶性肿瘤类型，其主要临床表现为不规则阴道出血、异常分泌物排出及腹痛等症状。虽然超声等非侵入性影像技术可作为重要的筛查工具，但对于疑似病例的确诊仍然需要依靠详细的病理学检查及特定标志物的免疫组化分析。针对此类疾病的治疗通常采取广泛的外科手术干预措施，随后配合放疗或化疗等多模式综合疗法来控制疾病进展并延长生存期。鉴于该病种的独特性质及其潜在威胁，建议加强对其早期识别与规范化管理的研究力度，从而改善患者的预后情况。

PO_465

葫芦素 B 对子宫内膜癌细胞恶性生物学行为的调控作用及机制研究

刘雪园 杜静* 刘翠兰
滨州医学院附属医院医学研究中心

子宫内膜癌是最常见的妇科癌症之一，其发病率也在逐年上升，尤其是在肥胖、糖尿病和激素替代治疗等高危人群中。大多数患者在早期被诊断，5 年生存率高达 95%。然而，转移或复发性疾病的患者预后较差，5 年生存率较低。子宫内膜癌是女性生殖系统常见恶性肿瘤，其中腺癌占比最高（约 80%）。现有治疗手段（手术、放化疗、激素治疗）存在耐药性、复发率高及副作用大等问题。葫芦素 B（Cucurbitacin B, CuB）是葫芦素中含量最丰富、活性最强的成员，是高度氧化的四环三萜类化合物。CuB 对各种类型的肿瘤具有显著的抗肿瘤活性，包括肝癌、乳腺癌、结肠癌、非小细胞肺癌和前列腺癌等。近年来越来越多的研究表明，低剂量的 CuB 对正常细胞和实验动物没有明显的毒性，而对癌细胞具有细胞毒性作用，这可以很好地解释其抗肿瘤作用。本研究聚焦于 CuB 对子宫内膜癌细胞恶性生物学行为的调控作用及机制。实验结果显示，葫芦素 B 以剂量依赖性方式显著抑制 HEC-1A 和 ISHIKAWA 细胞的增殖。CuB 可降低 HEC-1A 和 ISHIKAWA 细胞的迁移能力。CuB 可显著促进 ISHIKAWA 凋亡。综上，CuB 通过调控子宫内膜癌细胞的恶性生物学行为，为开发新型抗子宫内膜癌药物提供了理论依据和实验基础。

PO_466

A multi-modal prognosis prediction model integrating deep learning, radiomics, and clinical omics for endometrial cancer

Changmin Yu[1,2] Zhengyao Zhai[1,2] Yulan Ren[1,2*]
1.Fudan University Shanghai Cancer Center 2.Fudan University

Objective Postoperative risk stratification of endometrial cancer (EC) is crucial for adjuvant treatment decision-making and long-term prognosis prediction. Although radiomics has emerged as a widely applied imaging biomarker in EC, its predictive accuracy is still limited. Meanwhile, deep learning offers new opportunities by automatically learning complex image patterns, which may further improve prognostic modeling. However, studies combining radiomics, deep learning, and clinicopathological factors to construct integrated predictive models remain scarce. This study aims to develop a fusion model integrating clinical factors, radiomics features, and deep learning features derived from magnetic resonance imaging (MRI) for individualized prognosis assessment in EC patients. We also sought to evaluate the feasibility and accuracy of the multi-modal model in predicting disease recurrence.
Methods This retrospective study included EC patients who underwent surgical treatment and next-generation sequencing (NGS) at Fudan University Shanghai Cancer Center between December 2018 and December 2023. Preoperative MRI images, clinicopathological parameters, NGS-based molecular subtype results, and follow-up data were collected for all patients. Based on stratified randomization, patients were divided into training (n = 261) and validation (n = 66) cohorts in an 8:2 ratio.
Clinicopathological variables were selected using univariate logistic regression combined with recursive feature elimination based on multivariate logistic regression. Radiomics features were extracted from preoperative MRI sequences (contrast-enhanced T1-weighted imaging [CE-T1WI] and T2-weighted imaging [T2WI]) using PyRadiomics, and deep learning features were obtained through convolutional neural networks trained on the same sequences. Three single-modality models were constructed based on clinical features, radiomics features, and deep learning features, respectively. Subsequently, fusion models combining different feature sets were developed using logistic regression

algorithms. Model performance was evaluated using the area under the receiver operating characteristic curve (AUC), net reclassification index (NRI), integrated discrimination improvement (IDI), calibration curves, and decision curve analysis (DCA) to comprehensively assess predictive ability and clinical utility

Results A total of 327 patients were included in the analysis. According to NGS molecular profiling, 44 patients had the POLEmut subtype, 68 had MMRd, 167 had NSMP, and 48 had p53abn. Logistic regression-based single-modality models demonstrated moderate predictive performance across both training and validation cohorts. The fusion model integrating five clinical features, five radiomics features, and three deep learning features achieved the highest predictive accuracy among all models, with AUCs of 0.9568 in the training cohort and 0.9530 in the validation cohort. Calibration plots showed excellent agreement between predicted and observed outcomes, while DCA revealed significant clinical benefit across a range of thresholds. Furthermore, NRI and IDI analyses confirmed that the integrated multi-modal model substantially improved prognostic performance compared to individual models.

Conclusion This study demonstrates the feasibility of integrating clinical characteristics, radiomics features, and deep learning-derived imaging signatures into a multi-modal predictive model for EC prognosis. The model showed high generalizability and reliability in both internal training and validation sets. It may serve as a valuable tool for early identification of patients at high risk of recurrence, thereby supporting more precise postoperative treatment decisions and advancing individualized care strategies in endometrial cancer.

PO_467

WRN 作为 MSI-H 型子宫内膜癌治疗靶点的价值及机制研究

姚青青[1,2] 曲芃芃[1,2*] 张雯雯[1]
1.天津市中心妇产科医院 2.南开大学医学院

目的 研究 RecQ 解旋酶家族成员 WRN 在 MSI-H 子宫内膜癌中的作用机制，探讨其作为 MSI-H 型子宫内膜癌治疗靶点的价值。

方法

1、体外培养 MSI-H 子宫内膜癌（EC）细胞系及 MSS EC 细胞系，使用 Western-blot、RT-qPCR、CCK8、平板克隆、流式细胞术实验鉴定 shWRN 或使用 WRN 抑制剂 NSC 617145 对细胞增殖、克隆形成能力、细胞凋亡、细胞周期的影响；建立患者来源 MSI-H EC 原代细胞系，同上处理检测细胞表型；使用中性鞘磷脂酶抑制剂 GW4689 与 WRNi 共同处理 MSI-H EC 细胞系检测上述细胞表型。

2、Western-blot 检测 WRNi 处理后 MSI-H EC 细胞系 DNA 损伤标志蛋白的表达情况，p53 凋亡通路、细胞周期标志物、坏死性凋亡通路标志物的激活情况；免疫荧光技术检测 WRNi 处理后 MSI-H EC 细胞系 DNA 损伤标志物 γH2AX 表达情况。

3、转录组及代谢组学测序检测 shWRN 或 WRNi 处理 EC 细胞系转录水平通路富集及具体标志物变化情况。

4、RT-qPCR、ELISA、流式细胞术等检测 EC 细胞系在 WRNi 处理下神经酰胺代谢合成酶基因的转录差异以及中性鞘磷脂酶活性、ROS 蓄积水平变化。

5、裸鼠皮下成瘤及 RT-qPCR、免疫组化检测 WRNi、GW4689 处理对 MSI-H EC 细胞系体内生长及体内肿瘤神经酰胺代谢合成酶及 DNA 损伤标志物表达的影响

4、分析 TCGA 数据库中 WRN 表达及突变情况及对患者生存的影响。

结果

1、在 EC MSI-H 细胞系中敲低 WRN 基因或使用 WRNi 造成细胞增殖、克隆形成能力下降，细胞凋亡水平增加，细胞周期停滞在 G2/M 期；2、使用 WRNi 处理细胞导致 p53 凋亡、坏死性凋亡通路激活，DNA 损伤标志物 p-γH2A 水平程时间梯度增加，细胞核中观察到更多的 γH2AX 病灶；3、在 MSI-H 型 EC 中 WRN 表达低具有较好生存率，而在 MSS 及 MSI-L EC 中 WRN 表达高低对患者生存率影响不显

著，EC 是泛癌种中 WRN 突变率最高的癌种，WRN 突变 EC 患者具有更好生存率；4、转录组学 KEGG 富集显示 WRNi 处理细胞导致代谢通路的显著富集，脂质组学测序 WRNi 处理后神经酰胺和磷脂显著上调，鞘磷脂代谢通路激活，神经酰胺合成酶表达增多；5、促进神经酰胺合成的中性鞘磷脂酶活性以及 ROS 水平在 WRNi 处理细胞中显著升高，使用中性鞘磷脂酶抑制剂可逆转 WRNi 造成的凋亡细胞表型；6、体内使用 WRNi 能抑制 MSI-H EC 细胞皮下肿瘤生长，并造成 DNA 损伤标志物及神经酰胺合成代谢关键酶差异表达，GW4869 能逆转皮下成瘤表型。

结论 WRN 抑制剂通过导致 DNA 损伤以及促进神经酰胺合成导致 MSI-H 内膜癌体内外生长抑制

PO_468

Single-Cell Sequencing Reveals the Pathological Origins of Lymphovascular Space Invasion and Immune Remodeling in the Tumor Microenvironment of Early-Stage Low-Grade Endometrioid Endometrial Carcinoma

Wendi Guo[1] Yuanjing Hu[2*]
1.Nankai University
2.Tianjin Central Hospital of Obstetrics and Gynecology

Background and Objectives Lymphovascular space invasion (LVSI) represents a pivotal risk factor for disease recurrence and distant metastasis in patients with early-stage low-grade endometrioid endometrial carcinoma (EEC), a common subtype of endometrial cancer. Despite its clinical significance, the precise cellular origins of LVSI, along with the underlying mechanisms that drive immune microenvironment remodeling, have remained largely elusive. This ambiguity hinders accurate risk stratification and the development of targeted interventions. To address these gaps, the present study employs single-cell RNA sequencing (scRNA-seq) technology to comprehensively dissect the cellular heterogeneity within LVSI-positive tumors. By generating a high-resolution single-cell atlas, we aim to elucidate the pathological origins of LVSI, delineate the pre-metastatic niche, and uncover the intricate synergies between tumor cells and the immune system that facilitate metastatic progression. Ultimately, this investigation seeks to provide novel insights into the molecular underpinnings of LVSI, paving the way for enhanced diagnostic precision and innovative therapeutic approaches tailored to early-stage EEC.

Materials and Methods Fresh endometrial tissue specimens were meticulously collected from a cohort of 5 patients diagnosed with FIGO stage IA, grade 1–2 EEC, comprising 3 cases with confirmed LVSI positivity and 2 without LVSI. Additionally, control samples were obtained from 2 patients undergoing hysterectomy for non-malignant benign gynecological conditions, ensuring a balanced comparative framework. Immediately following surgical resection, tissues were enzymatically dissociated into single-cell suspensions to preserve cellular viability and transcriptomic integrity. Utilizing the 10x Genomics Chromium platform, we successfully captured and sequenced 67,062 high-quality single-cell transcriptomes, enabling an in-depth exploration of cellular diversity. Data processing involved rigorous quality control measures, including the identification and annotation of major cell types based on established marker genes, as well as the removal of doublets to minimize technical artifacts. Advanced analytical pipelines were applied, such as functional gene module scoring to assess pathway activities, scRNA-seq-derived copy number variation (CNV) inference to detect genomic alterations, and weighted gene co-expression network analysis (WGCNA) for identifying co-regulated gene modules. Furthermore, pathway enrichment analyses highlighted dysregulated biological processes, while single-cell regulatory network inference and clustering (SCENIC) revealed key transcription factors. Pseudotime trajectory inference mapped cellular developmental paths, and cell-cell communication analyses, using tools like CellChat, uncovered ligand-receptor interactions driving intercellular crosstalk.

Results The generated single-cell atlas delineated seven distinct major cell populations within the endometrial ecosystem: epithelial cells, T/NK cells, B cells, myeloid cells, fibroblasts, endothelial cells, and mast cells. A marked escalation in immune cell infiltration was observed correlating with disease advancement from benign to LVSI-negative and LVSI-positive states, underscoring an evolving inflammatory landscape. Malignant epithelial cells in LVSI-positive tumors exhibited heightened proliferative signatures, as evidenced by elevated expression of cell cycle-related genes, whereas LVSI-negative counterparts were embedded in a more pronounced inflammatory milieu, potentially reflecting early immune surveillance. Intriguingly, the TC1/4 epithelial subset emerged as a key player, displaying robust epithelial-mesenchymal transition (EMT) and partial EMT (p-EMT) characteristics, which are intimately linked to the invasive and metastatic potential in early endometrial carcinoma. This subset's molecular profile suggests it may serve as a progenitor for LVSI initiation. In parallel, the study pinpointed critical immune and stromal subpopulations instrumental in sculpting an immunosuppressive and metastasis-promoting microenvironment specifically in LVSI-positive tumors. Notable among these were SPP1+MMP9+ macrophages, which likely contribute to matrix degradation and tumor invasion; WNT5A+ myo-cancer-associated fibroblasts (mCAFs) and inflammatory CAFs (iCAFs), fostering stromal remodeling; TNFRSF9+ regulatory T cells (Tregs), enforcing immune tolerance; and ZNF683+SOX4+CD8+ T cells, indicative of exhausted cytotoxic responses. Moreover, intricate intercellular dialogues were mapped, revealing that interactions between WNT5A+ mCAFs, iCAFs, and malignant epithelial cells are orchestrated via multifaceted "stroma-signal" axes. These primarily encompass extracellular matrix-integrin signaling for adhesion and migration, alongside immune modulatory pathways that dampen anti-tumor immunity. Such synergies collectively propel tumor cell invasion, EMT induction, and evasion of immune detection, positioning these cellular networks as central drivers in LVSI pathogenesis and overall tumor progression.

Conclusion In summary, this investigation delivers a detailed single-cell resolution atlas of early-stage low-grade EEC harboring LVSI, illuminating the multifaceted cellular ecosystem and distinctive molecular signatures tied to this invasive phenotype. By unraveling the heterogeneous cell populations and their dynamic interactions, we shed light on the foundational mechanisms fueling LVSI development, including enhanced proliferation, EMT-driven invasion, and immunosuppressive remodeling. These revelations not only advance our comprehension of the metastatic cascade in endometrial cancer but also hold substantial promise for refining clinical diagnostics, such as through biomarker identification for LVSI prediction. Furthermore, the identified key subsets and signaling axes offer actionable targets for therapeutic innovation, potentially guiding the design of precision therapies to interrupt tumor-immune collaborations and mitigate metastasis risk in affected patients.

PO_469

Mechanism Research on CYCS Gene Regulating Polyamine Metabolism via AGE-RAGE Pathway in Hormone Therapy Resistance of Type I Endometrial Cancer

Rui Ban
Tianjin Central Hospital ofGynaecology Obsterics, Tianjin, China

This review explores the key mechanism by which the CYCS gene regulates polyamine metabolism through the AGE-RAGE signaling pathway in hormone therapy resistance of type I endometrial cancer. As one of the most common gynecological malignancies, hormone therapy resistance in type I endometrial cancer has become a major challenge in clinical treatment. Recent studies have found that the CYCS gene, a key regulator of apoptosis, its abnormal expression is closely related to polyamine metabolism disorders, and the AGE-RAGE signaling pathway may play an important mediating role in this process. Existing evidence indicates that the CYCS-AGE-RAGE-polyamine metabolism axis forms a complex molecular regulatory network, participating in the adaptive response of tumor cells to hormone therapy. This article systematically sorts out the molecular mechanisms of this regulatory network, analyzes the therapeutic potential of targeted intervention in this pathway to reverse hormone resistance, and points out the limitations of current research, such as insufficient sample size and incomplete mechanism. It also puts forward prospects for future research directions, including the development of specific inhibitors and the exploration of combined treatment strategies.

PO_470

子宫内膜去分化癌 16 例临床病理特点及预后分析

罗延倩[1] 李林[2]
1.彭州市妇幼保健院 2.重庆大学附属肿瘤医院

目的 回顾性分析 16 例子宫内膜去分化癌（dedifferentiated endometrial carcinoma，DEC）患者的临床病理特征、分子分型、治疗及预后相关性，探讨其诊断要点和治疗策略。
方法 收集 2020 年 02 月至 2025 年 5 月在重庆大学附属肿瘤医院诊治的 16 例 DEC 患者，分析其一般资料、病理特征、分子分型及、治疗与预后相关性。
结果 16 例患者平均发病年龄 55.4 岁（47-76 岁），术前仅 31.2%（5/16）确诊 DEC。临床病理分期（FIGO 2009）：IA 期 5 例，II 期 2 例，IIIA 期 1 例，IIIB 期 1 例，IIIC1 期 1 例，IIIC2 期 2 例，IVB 期 4 例。所有患者均行手术治疗，15 例行全子宫/筋膜外子宫切除，1 例行广泛性子宫切除，15 例行盆腔淋巴结清扫，13 例行腹主动脉旁淋巴结切除，6 例行大网膜切除；6 例行经腹手术、10 例行微创手术（腹腔镜/机器人辅助）；12 例患者术后辅助化疗或放疗，化疗药物方案为 TC/TP；3 例未辅助治疗。12 例术后行分子分型检测，其中 4 例为 dMMR，4 例 NSMP，2 例 POLEmut，2 例 P53mut。
1 例失访，5 例死亡，平均生存期为 13.4 个月，余 10 例患者均无瘤生存至今，平均生存期为个 32.4 个月。单因素生存分析显示不同手术入路与预后无统计学差异差；术后补辅助放化疗的患者的生存期长于未补充治疗者，P 值为 0.033（P<0.05）。
结论 子宫内膜去分化癌侵袭性强，术前确诊率低，预后差；术后辅助放化疗可能改善患者预后。

PO_471

Case Report: Brain metastasis from cervical adenocarcinoma combined with endometrial carcinoma

Shun Yao 朱诚程*
皖南医学院弋矶山医院

Endometrial carcinoma and cervical cancer are common gynecological malignant tumors, but endometrial adenocarcinoma combined with cervical adenocarcinoma is rare in clinic. This is a young female patient with endometrial adenocarcinoma and cervical adenocarcinoma, developed early brain metastasis.The patient was treated by left frontal lobe metastasis excision under surgical microscope.Postoperative pathology showed metastatic poorly differentiated adenocarcinoma.She then went through a regimen of paclitaxel liposome combined with cisplatin for 8 cycles in oncology. At 39-months follow-up, the patient remained well with no clinical or neuroradiological evidence of recurrence.

PO_472

Study on the Gonadotoxicity of CDK Inhibitors: Potential Impact on Ovarian Function in Young Women with Fertility Needs

yutong wu
重庆市妇幼保健院

Cell cycle regulation is a highly orchestrated process that ensures the precise duplication and distribution of genetic material during cell division. At the core of this regulatory network are cyclin-dependent kinases (CDKs), a family of serine/threonine kinases that control the progression of cells through different phases of the cell cycle. By phosphorylating key regulatory proteins, CDKs drive cells from the resting phase (G0) into the active phases of growth (G1), DNA synthesis (S), preparation for division (G2), and ultimately mitosis (M). This intricate mechanism is tightly controlled by the periodic activation and deactivation of CDKs, which are regulated by their binding partners, the cyclins, as well as by various inhibitory proteins. Dysregulation of the cell cycle is a hallmark of cancer, and aberrant activation of CDKs plays a central role in tumorigenesis. Mutations or overexpression of CDKs and cyclins disrupt the normal cell cycle checkpoints, allowing cells to bypass crucial regulatory controls and undergo uncontrolled proliferation. This not only leads to the accumulation of genetic abnormalities but also enables cancer cells to evade programmed cell death (apoptosis). Moreover, the dysregulation of CDKs can contribute to genomic instability, a characteristic feature of many malignancies that facilitates the acquisition of additional mutations and promotes tumor progression. In response to the critical role of CDKs in cancer, significant efforts have been made over the past few decades to develop small-molecule inhibitors that can selectively target these kinases. CDK inhibitors aim to restore normal cell cycle regulation by blocking the activity of overactive CDKs, thereby halting the proliferation of cancer cells and inducing apoptosis. Several CDK inhibitors have entered clinical trials, and some have been approved for the treatment of various malignancies, including breast, lung, and hematological cancers. These drugs represent a significant advancement in targeted cancer therapy, offering the potential for more effective and less toxic treatment options compared to traditional chemotherapy. While the therapeutic potential of CDK inhibitors is promising, concerns have been raised about their potential impact on normal tissues, particularly those with high proliferative activity. One area of particular concern is the gonadal toxicity of these drugs, especially in young women with fertility needs. Current research has extensively documented the detrimental effects of chemotherapy on gonadal function, leading to premature ovarian failure, infertility, and menopausal symptoms. However, the

impact of novel targeted therapies, such as CDK inhibitors, on ovarian function and fertility remains largely unexplored. Ovarian tissue is a highly dynamic organ that relies on continuous cell proliferation and differentiation for its normal function. CDKs and their associated complexes play crucial roles in regulating ovarian folliculogenesis, the process by which ovarian follicles develop and mature. Disruption of this process by CDK inhibitors could potentially lead to impaired follicular development, reduced ovarian reserve, and compromised fertility. Moreover, given the long-term implications of fertility preservation for young cancer survivors, understanding the gonadotoxicity of CDK inhibitors is of paramount importance. This review aims to provide a comprehensive overview of the current knowledge regarding the role of CDKs and their complexes in ovarian tissue and to discuss the potential gonadotoxicity of CDK inhibitors. By synthesizing the available preclinical and clinical data, this review seeks to offer preliminary guidance on the use of CDK inhibitors in young women with fertility needs, highlighting the need for further research to better understand the risks and benefits associated with these drugs. Additionally, this review will explore potential strategies for mitigating the gonadotoxic effects of CDK inhibitors, including the development of more selective drugs and the implementation of fertility preservation techniques.

PO_473

NETs 在妇科恶性肿瘤中的作用及治疗潜力

朱明月 周顾鑫 刘进 常方圆*
江苏医药学院附属东台医院

研究目的 中性粒细胞胞外诱捕网（Neutrophil Extracellular Traps, NETs）作为肿瘤微环境中的关键免疫调节因子，在妇科恶性肿瘤（包括子宫颈癌、子宫内膜癌及卵巢癌）的进展、转移及治疗抵抗中发挥复杂作用。本文旨在系统解析 NETs 在妇科肿瘤中的作用及机制，评估其作为预后标志物或治疗靶点的临床转化价值，并提出针对 NETs 的干预策略。

材料与方法 本研究检索中国知网、万方、sinomed、PubMed、Web of Science、Embase 和 Cochrane Library 数据库的中、英文文献。

结果 NETs 在妇科恶性肿瘤中构成动态调控网络，其双重作用受微环境 pH、代谢状态及免疫细胞互作的精密调控。NETs通过诱导上皮间质转化（EMT）、促进血管生成及介导免疫逃逸驱动肿瘤进展。临床前研究表明，靶向 NETs 药物可降低 50-70%的转移率，并增强铂类化疗敏感性，靶向 NETs 可有望抑制转移并逆转化疗抵抗。但 NETs 的时空异质性导致其促癌/抑癌作用可能呈现剂量依赖性特点。

结论 在未来研究中需要更加细致地探讨 NETs 的特异性机制和临床应用价值。因此，建立标准化的实验模型和研究方法尤为重要。为了更好地平衡不同研究之间的观点和发现，我们需要加强基础研究与临床应用之间的联系，推动跨学科合作，以实现 NETs 相关研究的深入开展。通过系统的研究和多方位的探讨，有望在妇科恶性肿瘤的防治上取得新的进展，为患者带来实质性的益处。

PO_474

《脏腑不通，肿瘤就生》

孟晓峰
英国林奈学会

《吕氏春秋》曰：“流水不腐，户枢不蠹”。
《中藏经》曰：“夫痈疽疮肿之所作也，皆五脏六腑蓄毒不流则生矣，非独因荣卫壅塞而发者也”。
《素问·通评虚实论》曰：“膈塞闭绝，上下不通，则暴忧之病也”。
《灵枢·五变》曰：“皮肤薄而不泽，肉不坚而淖泽，如此则肠胃恶，恶则邪气留止，积聚乃作，脾胃之间，寒温不次，邪气稍止，蓄积留止，大聚乃起”。
《难经》曰：“气之所积名曰积，气之所聚名曰聚，故积者五脏所生，聚者六腑所成。积者阴气也，其始发有常处，其痛不离其部，上下有所终始，左右有所穷处。聚者阳气也，其始发无根本，上下无所留止，其痛不常处，为之聚”。
《金匮要略》曰：“积者脏病也，终不移，聚者腑病也，发作有时，展转痛移为可治”。
《肘后备急方》曰：“凡瘕瘕之起多以渐生，如有卒觉便牢大，自难治之，腹中症有结节，便害饮食，转羸弱”。
《诸病源侯论》曰：“瘕者，由寒温失节，致脏腑之气虚弱，而饮食不消，聚结在内，染渐生长块段，盘牢不移动者是癥也……若积引岁月，人皆柴瘦，腹转大，遂致死”。
《妇人大全良方》曰：“若初起内结小核，或如鳖棋子，不赤不痛，积之岁月渐大，巉岩崩破如熟石榴，或内溃深洞，血水滴沥，此属于肝脾郁怒，气血亏损，名曰乳岩，为难疗”。
《黄帝内经》曰：“久者数岁户成，以手按之柔，已有所结，气血归之，津液留之，邪气巾之，凝结日以益甚，连以聚居为昔瘤”。
《中藏经》曰：“三焦者人之三元之气也，号曰中清之腑，总领五脏六腑，营卫经络，内外、左右、上下之气也。三焦通则内外、左右、上下皆通也，其于周身灌体，和内调外，荣左养右，导上宣下，莫大于此也”。
《本草纲目》曰：“生两肾之间，似脂非脂，似肉非肉，乃人物之命门，三焦发源处也……”。
笔者由此提出：“补、疏、调”康养学说。补足命门之火，疏通三焦经络，才好调和全身气血，预防肿瘤发生。

PO_475

Association between Dietary Oxidative Balance Score and Gynecological cancers: Evidence from NHANES 2009–2014

Chen Chen Zheng Mengyu Guo Yongzhen Dong Xing Zeng Xianxu*
The Third Affiliated Hospital of Zhengzhou University

Objective This study aimed to analyze the association between the Dietary Oxidative Balance Score (DOBS) and the risk of gynecological cancer using data from the National Health and Nutrition Examination Survey (NHANES) conducted between 2009 and 2014.
Methods Data were derived from NHANES surveys conducted during 2009–2014, including a total of 7,618 women. To investigate the relationship between DOBS and gynecological cancer, weighted multivariate logistic regression analyses were performed with DOBS treated as both a continuous variable and a categorical variable divided into quartiles. Subgroup analyses were further conducted according to DOBS and characteristics of gynecological cancer to explore this association. Additionally, restricted cubic spline (RCS) analysis was employed to evaluate the potential nonlinear relationship between DOBS and gynecological cancer risk.

Results Among the 7,618 women included in the analysis, the mean age was 46.69 years (standard deviation: 17.13), and 183 participants self-reported a confirmed diagnosis of gynecological cancer. In the fully adjusted model, regardless of whether DOBS was treated as a continuous variable (OR = 0.84, 95% CI: 0.67–0.92, p = 0.043) or a categorical variable (highest quartile vs. lowest quartile: OR = 0.79, 95% CI: 0.51–0.86, p = 0.005, trend p = 0.021), a significant association was observed. Restricted cubic spline (RCS) analysis confirmed a linear relationship between DOBS and gynecological cancer risk (nonlinear association p = 0.2165). Subgroup analysis revealed a significant interaction effect with diabetes status (interaction p = 0.026).
Conclusion DOBS was significantly associated with gynecological cancer, and an interaction effect was observed between DOBS and diabetes status. Individuals are encouraged to increase their DOBS levels. The findings may provide potential theoretical references for preventing gynecological cancers induced by oxidative stress.

PO_476

MR-Linac Adaptive stereotactic body radiotherapy in patients with pelvic-abdominal recurrent or metastatic gynecological malignancies

Xi Yang Shuang Zhao Lu Zhang Duan Yang Shuangzheng Jia Jusheng An Manni Huang*
National Cancer Center/National Clinical Research Center for Cancer/Cancer Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing, China

Objective Inadequate radiation dosage to recurrent pelvic and abdominal tumors is frequently attributable to dose limitations of surrounding normal structures, particularly the intestine. Radiotherapy guided by magnetic resonance imaging (MRI) significantly enhances soft tissue delineation accuracy. The purposes of this trial were to demonstrate the feasibility and effectiveness of the MR-Linac Adaptive stereotactic body radiotherapy in patients with pelvic-abdominal recurrent or metastatic gynecological malignancies with/without systemic therapies.
Methods Patients with pelvic-abdominal recurrent or metastatic gynecological malignancies were eligible for MR-Linac Adaptive stereotactic body radiotherapy. Systemic therapies, including chemotherapy, immunotherapy, and targeted therapy, are considered acceptable treatment options. The safety, tolerability and efficacy of MR-Linac Adaptive stereotactic body radiotherapy was assessed.
Results Between October 2019 and May 2025, 15 patients were received MR-Linac Adaptive stereotactic body radiotherapy. With the median follow-up period of 4.67 months (range, 0.73-20.10 months), the 2-year OS was 81.3%, the 2-year PFS was 20.3%, the 2-year LCR was 36.5%. MR-Linac Adaptive stereotactic body radiotherapy led to objective responses in 73.3% (11/15) of all patients. As of data cutoff (May 28, 2025), one patient experienced dose-limiting toxicities (enteric fistula). A patient developed CTCAE grade 3 thrombocytopenia during treatment, which was considered as chemotherapy-induced.
Conclusion These findings suggested that a MR-Linac Adaptive stereotactic body radiotherapy is relative effective and safe and can be an important treatment option for patients with pelvic-abdominal recurrent or metastatic gynecological malignancies. MR-Linac Adaptive stereotactic body radiotherapy exhibited acceptable tolerability, promising efficacy, and a favorable local control rate in heavily pretreated advanced solid tumors.

PO_477

单细胞转录组学视角下化疗对 T 细胞组成和代谢程序的重塑作用

依力米努尔·阿布拉江 魏佳慧 何玥 王明 吴玉梅*
首都医科大学附属北京妇产医院/北京妇幼保健院

研究目的 化疗作为肿瘤治疗的重要手段，能够显著重塑肿瘤免疫微环境。然而目前对于化疗对 T 细胞的影响，尤其是其免疫代谢重编程方面的研究仍较为有限。鉴于此本研究旨在深入探究肿瘤患者在接受化疗前后 T 细胞所发生的免疫代谢重编程情况，以期为优化肿瘤治疗策略提供新的理论依据和潜在靶点。

材料与方法 本研究收集了化疗前（PN）和化疗后（TN）的肿瘤样本，并对其进行了单细胞 RNA 测序分析。通过经典标志物对细胞类型进行注释，准确区分出包括 $CD8^+$T 细胞、$CD4^+$T 细胞、NK 细胞、髓系细胞、内皮细胞、上皮细胞、B 细胞以及造血干细胞（HSC）等多种免疫细胞类型。接着，对 PN 和 TN 组之间的 T 细胞进行差异基因表达分析，筛选出具有显著差异的基因。同时整合与代谢重编程相关的基因集，识别出在 T 细胞中重叠的基因。此外还运用基因本体（GO）富集分析和京都基因与基因组百科全书（KEGG）通路富集分析等生物信息学方法，深入挖掘这些差异基因和重叠基因所涉及的生物学过程和信号通路。

结果 研究结果显示化疗对肿瘤免疫微环境产生了显著影响，免疫细胞组成发生了明显变化。其中，$CD8^+$T 细胞的数量在化疗后出现了明显的减少，而基质细胞的比例则有所增加。通过对 T 细胞的差异基因表达分析，共筛选出 311 个差异表达基因，这些基因与 1384 个代谢重编程基因存在重叠。进一步的 GO 富集分析表明，这些重叠基因在诸如“细胞对化学刺激的响应”“细胞外囊泡组织”以及“细胞外囊泡的生物合成”等生物过程中显著富集。KEGG 通路富集分析则突出了多个关键信号通路，包括 PI3K - Akt 信号通路、氧化磷酸化通路以及 T 细胞受体信号通路等。这些结果表明化疗不仅影响了 T 细胞的数量，还对其基因表达和代谢过程产生了深远的影响。

结论 综合上述研究结果本研究揭示了化疗对 T 细胞的深刻影响，不仅改变了 T 细胞的丰度，还诱导了其转录和代谢的重编程。这些变化可能在肿瘤治疗抵抗和免疫逃逸中发挥着重要作用，为开发新的联合治疗策略提供了潜在的靶点和理论基础。未来的研究可以进一步深入探讨这些差异表达基因和代谢重编程通路在肿瘤免疫微环境中的具体作用机制，以及如何通过调控这些通路来增强肿瘤免疫治疗的效果

PO_478

PKN2 靶向 DUSP2 诱导 M2 巨噬细胞极化促进子宫内膜异位症细胞增殖迁移侵袭

王芳
宁夏回族自治区人民医院

目的 本研究旨在探讨 PKN2 靶向结合 DUSP2 诱导 M2 巨噬细胞极化促进子宫内膜异位症细胞增殖迁移侵袭中的作用。

方法 我们利用质粒转染和基因沉默技术在体外构建敲低 PKN2 和过表达 PKN2 的永生化人子宫内膜异位症间质细胞系，并用 qRT-PCR 检测 PKN2 转染效率；将转染 PKN2 过表达质粒(pcDNA -PKN2)及敲低质粒（siRNA-PKN2）的 HEM15A 细胞与 THP-1/M2 极化细胞共培养，分为空白对照组、阴性对照组、pcDNA -PKN2 组以及 siRNA-PKN2 组，CCK-8 增殖实验检测各组细胞的增殖活性；Transwell 迁

移侵袭实验检测细胞迁移及侵袭功能；采用双荧光素酶实验分析 PKN2 与 DUSP2 存在靶向结合关系；Western blot 检测了各组细胞中 PKN2 与 DUSP2 蛋白表达情况。
结果 我们利用质粒转染和基因沉默技术在体外构建敲低 PKN2 和过表达 PKN2 的 HEM15A 细胞系，qRT-PCR 检测转染效率，结果显示：与 pcDNA-NC 组相比，pcDNA-PKN2 组 PKN2mRNA 表达量显著升高，而与 siRNAi-NC 组相比，siRNA-PKN2 组 PKN2mRNA 表达量显著降低。CCK-8 增殖实验表明，与空白对照组细胞相比，pcDNA--PKN2 组促进了 M2 巨噬细胞诱导的永生化人子宫内膜异位症间质细胞的增殖活性显著升高，而 siRNA-PKN2 组细胞增殖活性显著降低；Transwell 检测细胞迁移侵袭，结果表明，与空白对照组细胞相比，pcDNA--PKN2 组促进了 M2 巨噬细胞诱导的永生化人子宫内膜异位症间质细胞的迁移侵袭水平均显著升高，而 siRNA-PKN2 组细胞迁移侵袭水平均显著降低；通过双荧光素酶报告实验发现，两株细胞中 PKN2 与 DUSP2 存在靶向结合关系；Western blot 检测结果显示：与空白对照组细胞相比，pcDNA--PKN2 组 PKN2 蛋白表达水平显著升高，DUSP2 蛋白表达显著降低，而 siRNA-PKN2 组 PKN2 蛋白表达水平显著降低，DUSP2 蛋白表达显著升高。
结论 PKN2 在子宫内膜异位症中的表达可以促进 M2 巨噬细胞极化和异位病灶增殖迁移侵袭，其可能通过靶向调控 DUSP2 促进 M2 巨噬细胞极化促进了子宫内膜内异症的进展。

PO_479

外泌体 lncRNA 的全面概述：妇科癌症的新兴生物标志

马帅 王敏*
吉林大学第二医院

目的 外泌体是一种微小的“茶杯状”水疱，可由大多数细胞分泌，并含有大量携带一些生物和遗传信息的 lncRNA，可以长期稳定，不受核糖核酸酶催化活性的影响。外泌体 lncRNA 作为一种细胞通讯工具，具有高效、靶向性高等优点。癌症患者血清外泌体 lncRNA 表达的变化可以准确反映癌细胞的恶性生物学行为。外泌体 lncRNA 在癌症诊断、癌症复发进展监测、癌症治疗和预后方面具有广泛的应用前景。
方法 外泌体含其组成与亲本细胞相似，可用作识别相关细胞的“指纹”，并提供可在循环血液中追踪的特定信号。长链非编码 RNA （lncRNA） 是在细胞质和细胞核中含量丰富的非编码 RNA。尽管血液中存在 RNA 酶，但由于外泌体保护，lncRNA 仍可持续存在。因此挖掘和探索影响恶性生物学行为的外泌体 lncRNA 具有重要的科学意义，因为这有助于进一步研究癌症发展的机制，并为癌症治疗提供新的思路和策略。
结果 lncRNA MALAT1 通过介导 Janus 激酶 2（JAK2）/STAT3 信号通路、促进 OC 细胞增殖、抑制癌细胞凋亡，在 OC 的发育中发挥重要作用。lncRNA MALAT1 可通过 PI3K 蛋白激酶 B（PKB，AKT）信号通路抑制癌细胞凋亡，同时增加 OC 细胞增殖，增强 OC 细胞侵袭、迁移和 EMT 功能。MALAT1 在上皮性 OC 患者中的表达显著高于对照组，并且与国际妇产科联合会 （FIGO） 晚期、高组织学分级和淋巴结转移有关。血清外泌体 MALAT1 表达升高与进行性转移性上皮性 OC 表型和预后不良相关，提示其可作为上皮性 OC 的预后或预测生物标志物。Guo 等人使用实时定量 PCR 和 Western blotting 检查了 MEG3 和 Notch 信号分子在 EC 组织和细胞系中的表达。MEG3 表达在 EC 组织中显著下调，而 Notch 蛋白表达在两者中均上调。MEG3 下调通过抑制 Notch 信号转导通路来抑制 EC 增殖。在 EC 患者中，血浆中外泌体 lncRNA MEG3 的低表达预示着更多的高危因素、更高的复发率和更差的预后。通过比较 EC 患者和健康女性血清 lncRNA ROR 和 miR-29 的表达水平，并使用 ROC 曲线评估两者在 EC 中的诊断价值，发现 EC 患者血清 lncRNA ROR 和 miR-29 水平显著高于健康女性。
结论 外泌体 lncRNA 被认为是妇科癌症诊断、疗效评估和预后预测的新标志物。

PO_480

铜死亡作为癌症的新兴生物标志物和潜在治疗方法

马帅 王敏*
吉林大学第二医院

目的 铜死亡不同于已知的死亡机制，是铜离子依赖的、可调节的并且依赖于线粒体呼吸的新型细胞死亡方式。目前已知铜死亡参与炎症、氧化应激、凋亡等多种病理过程。铜稳态的失衡与多种疾病的发生相关。铜死亡是当今癌症领域的研究热点之一。

方法 铜是维持人类生命活动的基本元素，其作为必需酶的辅助因子发挥着必不可少的作用。正常情况下，细胞内铜离子浓度通过主动稳态机制保持在较低水平。当铜离子水平逐渐累积超过阈值后，过量的铜离子会导致细胞的过度呼吸作用，产生细胞毒性最终诱导细胞死亡。2022年Tsvetkov等首次提出“铜死亡”。研究发现铜离子的蓄积与肿瘤的增殖、生长、血管生成以及转移等相关。铜离子水平可作为癌症进展的重要检测指标。

结果 铜可通过多种方式诱导肿瘤细胞死亡，“铜死亡”是一种不同于细胞凋亡、铁死亡、自噬和程序性坏死的新型程序性细胞死亡机制。铜直接与三羧酸循环中的硫酰化成分结合，导致蛋白质毒性应激，最终导致细胞死亡。异常的铜积累可促进细胞恶性转化。已经在动物模型和多种肿瘤患者肿瘤组织或血清中观察到铜浓度的升高。铜失衡会影响线粒体呼吸、导致糖酵解、胰岛素抵抗和脂质代谢的变化。由于癌症的特点是细胞死亡失调和炎症反应紊乱，目前的治疗策略旨在选择性诱导癌细胞的死亡并避免伤害正常细胞。与正常细胞相比，癌细胞对铜的需求更高。铜离子含量增加可促进肿瘤细胞的增殖与转移，促进血管的生成，因此通过双向调控人体内的 Cu2+ 已成为癌症治疗的新靶点。目前靶向铜诱导凋亡的策略主要包括使用铜离子螯合剂、铜离子载体来调节体内铜的水平等方式，诱导肿瘤细胞凋亡，从而发挥抗肿瘤的作用

结论 经过一系列安全性和有效性的测试，可以帮助将铜的化学和生物学基础研究转化为潜在的临床疗法和潜在的候选药物，在肿瘤治疗领域有着很好的应用前景。目前铜死亡在癌症中发挥作用的具体机制尚不清楚，还需要大量高质量的基础研究来证明铜死亡与肿瘤之间的因果关系。铜在肿瘤中的毒性分子机制及铜死亡演变为确定的细胞死亡的具体模式还需要进一步阐明。随着研究的不断深入，相信会有更多的新的相关靶点和药物出现，建立合理的个性化治疗策略，为癌症患者的治疗带来新的希望。

PO_481

Changes of disease spectrum of malignant tumor of female reproductive system in Beijing Obstetrics and Gynecology Hospital in recent 60 years

Yue He Shengqian Wang Chen Ji Zhenyou Liu Jiahui Wei Ming Wang Yumei Wu Yan Wang Xiaohong Xu*
Beijing Obstetrics and Gynecology Hospital, Capital Medical University. Beijing Maternal and Child Health Care Hospital

Objective To optimize the prevention and control strategy of female reproductive system malignant tumor by analyzing the incidence trend of female reproductive system tumor in the past 60 years since the establishment of Beijing Obstetrics and Gynecology Hospital Affiliated to Capital Medical University.

Methods Disease types and basic clinical information of 18921 patients with malignant tumor of female reproductive system admitted to our hospital during 60 years from January 1960 to December 2019 were collected.

Results (1) In the past 60 years, a total of 18,921 patients with malignant tumors of the female reproductive system were diagnosed and treated in our hospital. After the establishment of the

Department of Gynecological Oncology in our hospital in 1970, the number of gynecological tumor diseases treated has doubled, and the types of tumors treated and treated have also made a qualitative leap. Cervical cancer is the most common occurrence, followed by endometrial cancer, ovarian cancer and trophoblastic tumor.(2) The most common age of cervical cancer, endometrial cancer, ovarian cancer, uterine sarcoma, fallopian tube cancer and vaginal stump cancer is 46-55 years old; the most common age of gestational trophoblastic neoplasia(GTN) is 19-35 years old; the most common age of vulvar cancer, vaginal cancer and peritoneal cancer is 56-65 years old.(3) Among the 18,921 patients admitted, 94 were 0-18 years old, and the top three tumors were ovarian cancer,gestational trophoblastic neoplasia and vaginal cancer.There were 2596 cases between 19 and 35 years old, and the top three tumors were cervical cancer, ovarian cancer and trophoblastic tumor.In 16231 patients over 36 years of age, the first three tumors were cervical cancer, endometrial cancer and ovarian cancer.(4) The average age of onset of female reproductive system tumors was 49.43 ±11.65 years for cervical cancer, 53.95 ±9.45 years for endometrial cancer, 43.04 ±13.79 years for ovarian cancer, 32.77 ±9.76 years for trophoblastic tumor, and 49.74 ±11.44 years for vulvar cancer.Uterine sarcoma was 53.83 ±9.78 years old, fallopian tube cancer 54.23 ±10.51 years old, vaginal cancer 52.05 ±18.03 years old, stump vaginal cancer 57.88 ±11.82 years old, and peritoneal cancer 50.00 ±17.00 years old.(5) In recent 60 years, the top three malignant tumors of female reproductive system were cervical cancer 53.88% (10194/18921), endometrial cancer 23.00% (4352/18921) and ovarian cancer 15.42%(2919/18921).In recent 20 years, the incidence age of cervical cancer has a younger trend, and the incidence age of endometrial cancer and ovarian cancer has an aging trend.(6) A total of 10194 cases of cervical cancer were diagnosed and treated in the past 60 years. The most common pathological type was squamous cell carcinoma. There were 4352 cases of endometrial cancer, and the most common pathological type was adenocarcinoma. There were 2919 patients with ovarian cancer, and epithelial cancer was the most common pathological type.

Conclusion The construction of gynecological tumor subspecialty is of great help to the diagnosis and treatment of gynecological tumor. The number of patients with malignant tumor of female reproductive system has increased year by year in our center.

PO_482

Feasibility exploration of constructing a patient derived tumor tissue xenograft (PDX) model for malignant tumors of the female reproductive system in mice

Yue He[1] Zhuoying Du[1] Chen Ji[1] Xingming Li[2] Pingping Zhong[1] Yumei Wu[1*] Yan Wang[1]
1.eijing Obstetrics and Gynecology Hospital, Capital Medical University. Beijing Maternal and Child Health Care Hospital
2.Capital Medical University

Objective To explore the feasibility and influencing factors of constructing a patient derived tumor tissue xenograft model (PDX) in female reproductive system mice.

Method A mouse PDX model was constructed by collecting 78 patients suffering from common and rare gynecological tumor patients who treated in our hospital from March 2023 to October 2023.

Result ① 78 female patient with malignant tumors of the reproductive system, aged 27-82 (55.6 ± 12.7) years, including 36 cases of cervical cancer, 21 cases of endometrial cancer, 15 cases of ovarian cancer, 3 cases of vaginal tumors, 2 cases of vulvar tumors, and 1 case of uterine sarcoma. Among the 41 patients observed, the remaining 37 patients were successfully modeled (passaged) in 7 cases (18.92%) and implanted in 22 cases (59.46%). Three cases of rare gynecological tumors were not successfully implanted. The success rate of implantation in P0 generation mice showed no significant difference between nude mice and NTG mice. ② 19 patients with cervical cancer had a modeling success rate of 15.8% (3/19), a total implantation success rate of 63.2% (12/19). The construction time for P0 generation varies from 25 to 87 (47 ± 16.7) days; The P1 generation is 55 days old; P2

generation takes 69 days. ③ 10 cases of endometrial cancer, the total implantation success rate is 60.0% (6/10)，the success rate of modeling is 10.0% (1/10). There was no significant difference in cell stage, tumor type, and differentiation between the failed group and the successfully implanted group. The construction time required for P0 generation is 24-93 (50 ± 19.7) days; P1 generation all takes 55 days; P2 generation for 41 days; P3 generation takes 55 days. ④ There were 5 cases of ovarian cancer, the total implantation success rate is 80.0% (4/5)，the success rate of modeling is 60.0% (3/5). The construction time required for P0 generation is 30-75 (50 ± 20.7) days; P1 generation takes 68-125 (88 ± 32.1) days. ⑤ The above-mentioned female reproductive system tumors can be constructed into mouse PDX models after neoadjuvant chemotherapy and some metastatic tumors. Compared with the successful vaccination group, there was no significant difference in tumor staging, tumor type, and differentiation between the failed mouse PDX model construction group and the successful vaccination group (P>0.05).

Conclusion It is feasible to construct a mouse PDX model for female reproductive system tumors, and it is also feasible for tumor construction after chemotherapy or at the site of metastasis.

PO_483

Mechanistic exploration of mitophagy in DNA damage repair within cancer stem cells

Qiaohua Peng
Women's hospital school of medicine zhejiang university

Cancer, as an important and specific disorder threatening to human health, has consistently been the most concentrated used to be on the mechanisms of underlying their occurrence and development in medicine and biology. Cancer stem cells (CSCs) are a unique subset of cancer cells with the ability of self-renewal and multi-directional differentiation. Unlike tumor cells, the CSCs have a stronger capacity than tumor cells to repair DNA damage, which promotes the preservation of genomic stability under different stress conditions. This ability enables CSCs to become resistant to the cytotoxic effects of standard therapies such as chemotherapy and radiotherapy, which play a role in tumor recurrence, metastasis, and drug resistance.

Mitophagy and mitochondrial homeostasis have been shown to be intricately associated with the survival, maintenance of stemness and drug resistance of CSCs in the recent most studies. Mitophagy is a highly selected, conserved biological process found in cells. It refers to the removal of compromised or excessive mitochondria, which is crucial for a cell to maintain intracellular homeostasis, regulate energy metabolism, facilitate DNA damage repair, and ensure normal cellular function. CSCs possess stronger mitophagy activity and DNA damage repair ability. Mitophagy and DNA damage repair are closely related and complex with great significance for tumorigenesis, tumor development, metastasis, and drug resistance. This relationship, therefore, is something that we must study in depth to understand the biological behavior of a tumor. Understanding the exact role of mitophagy in governing the DNA damage repair pathways in CSCs may provide us new insights about how tumor cells maintain genome stability and survive and expand in response to diverse stresses and therapies. This review aims to reveal a key relationship between enhanced mitophagy and DNA damage repair in CSCs and its therapeutic opportunities.

PO_484

1990-2021 年青少年和年轻成年女性癌症负担的全球趋势：来自全球疾病负担研究

侯杰卿[1] 黄紫怡[2] 徐璐[2]
1.江南大学 2.天津医科大学

背景 乳腺癌和生殖道恶性肿瘤对青少年和年轻成人（AYA）组中 15-39 岁女性的身心健康，尤其是生殖功能的影响是显着的。本研究旨在分析 1990 年至 2021 年全球不同地区和国家的 AYA 女性癌症负担。
方法 流行病学数据来自 2021 年全球疾病负担（GBD）研究。该研究分析了女性癌症（乳腺癌、卵巢癌、子宫癌和宫颈癌）的数据，以评估不同年龄、年份和地点的疾病负担，包括 21 个 GBD 地区、195 个国家和 5 个社会人口指数（SDI）地区。
结果 2021 年 AYA 女性癌症全球新增病例 383,241 例，死亡 81,679 例，其中患病病例 2,975,183 例，残疾调整生命年 4,855,780 例。2021 年，拉丁美洲中部的年龄标准化发病率（ASIR）和患病率（ASPR）最高，而南撒哈拉以南非洲的年龄标准化死亡率（ASMR）和残疾率（ASDR）最高。SDI 高的国家通常表现出升高的 ASIR 和 ASPR，而 ASMR 和 ASDR 表现出相反的模式。在过去的三十年里，AYA 女性癌症的 ASIR 和 ASPR 在全球范围内有所增加。相比之下，ASMR 和 ASDR 则呈下降趋势。值得注意的是，乳腺癌成为 AYA 女性中最普遍的恶性肿瘤。
结论 在过去的三十年里，AYA 女性癌症的 ASIR 和 ASPR 持续增加，给这一人群带来了沉重的负担。此外，不同地区和国家之间存在明显的差异，与高 SDI 环境中的 AYA 女性相比，低 SDI 环境中的 AYA 女性预后较差。因此，当务之急是加强区域间合作和沟通，以优化 AYA 女性癌症的整体预后。

PO_485

年轻女性乳腺癌的全球负担和归因危险因素：按社会人口指数地区和国家划分的 1990 年至 2019 年的历史趋势和到 2030 年的预测

侯杰卿[1] 黄紫怡[2]
1.江南大学 2.天津医科大学

背景 年轻女性乳腺癌 （BCY） 要少得多，但会带来重大的健康后遗症和社会成本。我们旨在评估 1990 年至 2019 年 15-39 岁女性的全球和区域乳腺癌负担。
方法 我们从 2019 年全球疾病负担研究 （GBD 2019） 数据资源中收集了有关乳腺癌的详细数据。年龄标准化发病率 （ASIR） 、年龄标准化死亡率 （ASMR） 、年龄标准化残疾调整生命年率 （ASDR） 和估计年百分比变化 （EAPC） 用于评估 BCY 的疾病负担。贝叶斯年龄-时期-队列模型用于预测 2020 年至 2030 年的疾病负担。
结果 从 1990 年到 2019 年，发现 BCY 的 ASIR 显着增加 （EAPC = 0.59,95% 置信区间 （CI） = 0.5 至 0.68），而 ASMR （EAPC = -0.41,95% CI = -0.53 至 -0.3） 和 ASDR （EAPC = -0.35,95% CI = -0.46 至 -0.24）。在社会人口指数 （SDI） 不同的国家中，除 SDI 高的国家外，所有地区的 BCY 发病率均呈上升趋势。虽然 SDI 高、中高和中等国家的死亡率和 DALY 率有所下降，但在 SDI 中低和低的国家/地区的死亡率和 DALY 率有所增加。预计 SDI 较低的国家/地区将在未来十年内承担最大的 BCY 负担，包括低类别和中低类别。在大多数国家，饮酒是导致 BCY 死亡的主要危险因素，而在中低 SDI 国家，暴露于二手烟是 BCY 死亡的主要危险因素。

结论 年轻女性的乳腺癌负担在全球范围内呈上升趋势，并且存在显着的地区差异。SDI 中低或低的国家面临更多挑战，因为它们比 SDI 较高的国家承受了更严重且不断增加的 BCY 负担。

PO_486

环状 RNA 编码多肽在肿瘤精准治疗中的进展

谢欣怡
南京医科大学附属妇产医院

目的 本文重点围绕环状 RNA（circRNAs）在多种肿瘤中编码多肽进行讨论，旨在分析其编码多肽的不同作用与机制，并展望环状 RNA 编码多肽作为肿瘤精准治疗靶点的潜力，以期为后续研究和临床转化提供理论依据。
方法 通过中国知网（CNKI）、PubMed 及 Web of Science 等数据库对环状 RNA 编码多肽在乳腺癌、肝癌等不同肿瘤类型中的研究进展做系统梳理和总结，进行文献综述。
结果 环状 RNA 主要通过内部核糖体进入位点（IRES）、N6-甲基腺苷（m6A）修饰或滚环翻译等非帽依赖机制进行翻译，编码功能性多肽。其编码多肽在不同癌种中展现了高灵敏度的诊断潜力及预后价值，可通过调控信号通路、表观遗传修饰、蛋白稳定性等机制影响肿瘤生物学行为，或者通过调控免疫微环境影响肿瘤治疗效果。此外，环状 RNA 编码多肽还在靶向多肽的疫苗策略方面提供新思路，为临床上增强化疗敏感性提供新靶点。
结论 环状 RNA 编码多肽的发现拓展了对环状 RNA 生物学功能的认知，为肿瘤精准治疗提供了新的干预维度。然而，多肽功能异质性、动态调控机制及规模化生产仍是当前转化瓶颈。未来，通过多组学联合挖掘、动态调控机制解析和治疗策略联合开发，有望推动环状 RNA 编码多肽在肿瘤精准治疗中的应用。

PO_487

Supraclavicular lymphadenopathy as the initial clinical manifestation and sole lesion

Zhe Li Yangchun Sun*
National Cancer Center/National Clinical Research Center for Cancer/Cancer Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College

Aim This case presented with the sole clinical manifestation of left supraclavicular lymphadenopathy, without pelvic masses or ascites, highlighting the diagnostic and therapeutic challenges in high-grade serous ovarian carcinoma (HGSOC), which was confirmed surgically. The case included a multi-disciplinary approach to the differential diagnosis, pathology, diagnostic testing, and a discussion on treatment.
Methods This is a case report.
Results A 68-year-old woman with a history of left supraclavicular lymphadenopathy for over 2 months presented to our hospital. She reported the sudden onset of left supraclavicular lymphadenopathy, accompanied by pain and swelling, approximately 5 cm in diameter. She took oral antibiotics on her own (unknown kind, dosage, and duration of use), and the mass decreased in size (unclear data, but subsequent ultrasound supported shrinkage). Ultrasound (thyroid and regional lymph node): a hypoechoic mass in the left supraclavicular region, approximately 3.2 × 1.7 × 2.4 cm, with clear borders, uneven echogenicity, and slight blood flow signal detected, besides, multiple solid nodules in both sides of the thyroid, suggesting a benign diffuse thyroid condition. Due to insufficient tissue samples obtained from the US-guided percutaneous biopsy, the patient underwent left neck lymphadenectomy at September 26, 2024. The pathology result showed lymph node metastatic carcinoma (1/12),

consistent with poorly differentiated adenocarcinoma (high-grade serous carcinoma) (HGSC), with primary consideration for a reproductive system or Müllerian epithelial origin from the pelvic or abdominal cavity. IHC results showed CK7(3+), CK20(-), CK19(3+), PAX8(3+), WT-1(3+), ER(3+), PR(-), Ki-67(+90%), P53(+100% missense mutation), Galectin-3(1+), TG(-), TTF-1(-). Blood CA125 14.2U/ml, HE4 102pmol/L (≤121 pmol/L), and ROMA index 20.79% (high risk ≥29.9%). Cervical HPV &TCT, gastroscopy, laryngoscopy: no abnormalities detected. No mammography (mammogram) and breast ultrasound examinations were prescribed. PET-CT showed multiple lymph node metastases in the left supraclavicular region, aortic arch region, main pulmonary artery region, right cardiophrenic angle, left anterior diaphragmatic angle, and retroperitoneal aortic region near the L2 level, with no other abnormalities detected elsewhere in the body. The first chemotherapy plan was Paclitaxel liposome (175mg/㎡)+ Carboplatin (AUC4-5). The radiological assessments performed after chemotherapy, including pelvic MRI with contrast and contrast-enhanced CT (of the neck, chest, and abdomen), were consistent with the pre-chemotherapy PET-CT findings, with the exception of thickened cervical mucosa (approximately 0.9 cm) showing a slightly elevated signal on diffusion-weighted imaging (DWI) observed on MRI.

The patient received laparoscopic bilateral salpingo-oophorectomy, omentectomy, multiple peritoneal biopsies and endometrial segmental biopsy at November 27,2024. The uterus was preserved, and no lymph node surgery was performed. The intraoperative findings were unremarkable, except for the presence of scattered fibrous strands and scars in the omentum. Additionally, each fallopian tube mesentery showed a 0.2 cm white nodule, likely indicative of inflammation. The peritoneal washing cytology revealed no malignant tumor cells. The frozen section pathology of the cervical canal endometrium showed some fragmented cervical canal endometrial tissue in an exudate and inflammatory cell background. The frozen section pathology of the uterine endometrium revealed mucus and a small amount of fragmented squamous epithelial tissue. The paraffin pathology report revealed no evidence of malignant tumors, except for psammomatous calcification in the right ovary.

The MDT (Multi-Disciplinary Team) concluded that the psammomatous calcification in the right ovary was associated with serous adenocarcinoma of the ovary, which was related to the supraclavicular lymphadenopathy observed in the patient and recommended chemotherapy as the subsequent treatment plan. On December 26, 2024, she underwent a second chemotherapy regimen, which was the same as the first chemotherapy treatment. During treatment, both blood CA125 and HE4 remained within the normal range, while the ROMA index increased from 20.79% (before the first chemotherapy) to 31.76% (on the day of the second chemotherapy). She had no gene testing.

Conclusions In summary, our case report is notable for left lymph node metastasis as the sole clinical manifestation. The pathology, radiological examinations, clinical diagnosis and treatments can pose a challenge due to its uncommon nature.

PO_488

散发性葡萄胎与复发性葡萄胎的蛋白质组学图谱

屠梦雁 吕卫国 许君芬*
浙江大学医学院附属妇产科医院

研究目的 葡萄胎（HM）是最常见的妊娠滋养细胞疾病，以滋养细胞过度增殖和胚胎发育异常为特征。同一患者连续发生两次及以上葡萄胎定义为复发性葡萄胎，其恶变风险显著增高。本研究旨在解析散发性葡萄胎与复发性葡萄胎的蛋白质组学特征，挖掘其潜在诊疗靶点。

材料与方法 本研究收集了 8 例正常子宫内膜组织（NV）、8 例散发葡萄胎组织（HM_S）、13 例复发性葡萄胎的复发组织（HM_R）以及其中 6 例配对的初发葡萄胎组织样本（HM_R_P）。对所有样本进行了蛋白质组学检测，并进行后续的生物信息学分析。

结果 HM 与 NV 相比，蛋白质组学特征存在显著差异，共鉴定出 522 个上调蛋白和 2437 个下调蛋白。HM 中显著富集的通路包括补体与凝血级联、免疫反应及中性粒细胞脱颗粒等，CD55 与 ITGAM 在蛋

白互作网络（PPI）中处于核心节点，参与多个通路。相较而言，HM_R_P 和 HM_S 之间的蛋白组差异较小，值得注意的是，在 HM_R_P 中发现转录因子 POU2F1 及转录辅助因子 YAP1、STK3 显著下调，二者分别参与调控内质网功能和 Hippo 信号通路。HM_R 与 HM_R_P 的蛋白组特征相似，但与 HM_S 差异显著。我们对 HM_S，HM_R_P 和 HM_R 进行拟时序分析，根据表达趋势不同将蛋白质分成 3 种聚类，展示了复发性葡萄胎从初发到复发的蛋白质表达演变路径。此外，复发性葡萄胎表现出复杂且独特的免疫特征，与 HM_S 相比，HM_R 中免疫相关蛋白 GZMA、GZMB 及 PD-L1 显著上调；与 HM_R_P 相比，HM_R 中树突状细胞（DC）、辅助型 T 细胞 2（Th2）、平滑肌细胞及淋巴内皮细胞显著富集，而肥大细胞减少。

结论 我们的研究对 NV、HM_S、HM_R、HM_R_P 的定量蛋白质组数据进行了深入分析，发现 HM 呈现显著的免疫-凝血通路协同失调；阐明了复发性葡萄胎内质网功能（POU2F1）与 Hippo 通路（YAP1/STK3）缺陷；揭示了葡萄胎的复发进展伴随着免疫微环境的重编程，为葡萄胎的风险分层与精准治疗提供新靶点。

PO_489

Early-onset gynecological cancer profiles in Southeastern China: an analysis of population-based cancer registry data

Jie Lin Yeying Wen Yang Sun*
Fujian Cancer Hospital

Background Monitoring cancer occurrence in young adults, often under 50 years, is informative, serving as a sentinel for the future disease burden in older adults. This study aims to analyze recent trends in the incidence and mortality rates of early-onset gynecologic cancer in Southeastern China to inform strategies for the prevention and control of these types of cancers in Fujian.
Methods The age-standardized incidence rate (ASIR) and age-standardized mortality rate (ASMR), as well as the prevalence average annual percent changes (AAPC), were calculated. The data from Fujian was compared with statistics from China and globally, using population-based information from the Global Burden of Disease. Additionally, we employed the joinpoint regression model to further identify significant changes in the trends of early-onset gynecological cancer in Fujian.
Results The incidence trend of all three early-onset gynecologic cancers in Fujian has stagnated, while the mortality trend has steadily declined, with an average annual percent change (AAPC) of -5.19% (95% CI, -9.7 to -0.46, p = 0.04) from 2019 to 2023. Specifically, Among these cancers, early-onset cervical cancer is the most common and is the leading cause of death. The age-standardized incidence rate (ASIR) for early-onset cervical cancer is comparable to the national rate in China but is lower than the global average. This cancer type shows a declining trend, with an AAPC of -4.65% (95% CI, -7.44 to -1.78, p = 0.02). Additionally, the age-standardized mortality rate (ASMR) for early-onset cervical cancer is the lowest when compared to both national and global figures, decreasing from 1.74 per 100,000 in 2019 to 1.40 per 100,000 in 2023, resulting in an AAPC of -7.47% (95% CI, -11.49 to -3.25, p = 0.01). In contrast to the steady data observed in China and globally, the ASIR for early-onset uterine cancer has increased significantly, rising from 2.60 per 100,000 to 3.36 per 100,000, with an AAPC of 6.06 (95% CI, 1.38 to 10.96, p = 0.03). Furthermore, the ASIR for early-onset ovarian cancer is relatively higher than the national average and has slightly increased during the same period, showing an AAPC of 1.86 (95% CI, 1.20 to 2.52, p = 0.00). However, the ASMR for both uterine and ovarian cancers has remained stable.
Conclusions The early-onset gynecologic cancer profile exhibits distinct patterns in Fujian. Notably, there has been a decline in cervical cancer rates. In contrast, there is an increasing trend in the incidence of uterine cancer and ovarian cancer. Extra support and collaborations should be strengthened to improve cancer prevention, diagnosis, care, and research in Southeastern China.

PO_490

代谢相关因子 ME1 在肿瘤微环境中的作用及其与肿瘤细胞增殖和迁移的关系探讨

魏佳慧 依力米努尔·阿布拉江 吴玉梅*
首都医科大学附属北京妇产医院/北京妇幼保健院

目的 ME1 催化苹果酸转化为乙酸，从而将葡萄糖代谢与柠檬酸循环联系起来。近年来，ME1 在各种肿瘤中的作用仅被浅显地探讨。因此，我们的目标是分析 ME1 在泛癌中的潜在功能，特别关注其在卵巢癌中的作用。
方法 我们分析了多种癌症类型中正常组织和肿瘤组织中的 ME1 表达水平。 使用 CBIOPORTAL 评估了 ME1 的突变频率和特定位点。 此外，我们研究了 ME1 表达与多个因素之间的相关性，包括甲基化状态、肿瘤突变负荷、微卫星不稳定性、免疫调节基因、免疫检查点、肿瘤微环境评分、功能富集、单细胞分析和药物敏感性。 估计算法评估了 ME1 表达与肿瘤免疫化学微环境之间的相关性。 利用小干扰 RNA 和慢性病毒分别在两种卵巢癌细胞系中下调和上调 ME1 表达，以进行细胞增殖和迁移实验。
结果 我们的研究结果显示，ME1 在多种肿瘤中表现出异常，其中最主要的基因突变形式为错义突变。在分析的不同肿瘤中，ME1 与甲基化水平、TMB、MSI、免疫检查点、免疫调节基因、肿瘤微环境评分和免疫浸润显示出显著的相关性。 功能富集分析和单细胞分析表明，ME1 表达与代谢调控、巨噬细胞免疫反应、抗氧化防御机制以及潜在的肿瘤微环境相关。 ME1 水平的升高可能与对特定免疫疗法更有利的反应相关，这表明 ME1 在指导免疫治疗策略方面具有潜在应用。 体外研究结果表明，在卵巢癌细胞系中，敲低 ME1 抑制了肿瘤细胞的增殖和迁移。 相反，ME1 的过度表达似乎促进了肿瘤细胞的增殖和迁移。
结论 ME1 作为一种代谢相关因子，有潜力作为肿瘤进展和免疫浸润的生物标志物，特别是在卵巢癌中。 它可能标志着一种为肿瘤进展和免疫治疗提供能量的代谢重编程，为个性化治疗的发展提供了宝贵的见解。

PO_491

关注未成年女性卵巢健康

王政 闫志风 李明霞 吴迪 孟元光*
解放军总医院第一医学中心

目的 中华医学会健康管理学分会 2022 年健康体健基本项目专家共识中，并没有针对未成年女性的专项体检项目。众所周知，未成年女性面临着重要的发育关，即月经初潮，月经的来潮提示卵巢产生的雌激素足以使子宫内膜增殖，在雌激素达到一定水平且有明显波动时，引起子宫内膜脱落出现月经。但由于中枢系统对雌激素的正反馈机制尚未成熟，有时卵泡发育成熟但不能排卵，故月经周期常不规律，经 5～7 年建立规律的周期性排卵后，月经才逐渐正常。通过临床数据调查，我们发现在此阶段的未成年女性，其卵巢肿瘤发生率较高。
方法 我们研究了解放军总医院第一医学中心近 15 年共计 104 例未成年女性卵巢肿瘤，经过分析，我们发现卵巢肿瘤与月经初潮有明显相关性，恶性卵巢肿瘤的相关性更为显著。
结果 1、良性卵巢肿瘤与月经初潮的相关性：良性卵巢肿瘤的发生率与距离月经初潮的年数呈正相关，R 值约等于 0.87。2、恶性卵巢肿瘤与月经初潮的相关性：恶性卵巢肿瘤的发生率与距离月经初潮的年数呈正相关，R 值约等于 0.93。
结论 本文表明，卵巢肿瘤的发生与月经初潮息息相关，月经初潮的前后两年由于激素波动等原因，使得卵巢肿瘤发生率显著上升，同时卵巢肿瘤的存在干扰了雌孕激素的分泌。绝经意味着排卵的终止，绝经后女性是生殖系统肿瘤的高发人群，相对应的是，初潮提示着排卵的建立，我们的临床数据说明

这类人群更易发生卵巢肿瘤，但对此年龄段的关注远不及绝经后女性。激素的骤然变化是女性生殖系统肿瘤发生的高危因素，在此基础上，我们是否可以提出围初潮期的概念？初潮来临的女孩月经不规律应引起重视，不单单归因为性腺轴未发育成熟。

PO_492

恶性腹膜后血管周上皮来源肿瘤 1 例报道及文献复习

王政 闫志风 李明霞 吴迪 孟元光*
解放军总医院第一医学中心

目的 血管周上皮来源肿瘤（PEComa）是一种少见的间叶组织源性肿瘤，几乎全身各处均可发生，原发于腹膜后极为罕见，已知的文献多为个案报道。本文介绍了 1 例合并卵巢肿瘤的恶性腹膜后血管周上皮来源肿瘤并结合文献进行相关分析。

方法 44 岁女性，自然绝经 1 年，右下肢水肿 7 月。2023-10 外院行右髂血管旁肿瘤穿刺病理提示血管周上皮来源肿瘤（PEComa），会诊病理结果一致。我院经阴道超声提示右附件区囊实性包块、右髂血管旁多发低回声结节，不除外卵巢来源。2023-10-20 完善 PET-CT：盆腹部腹膜后及右侧髂动脉及附件旁轻度代谢肿块，考虑腹膜后原发肿瘤伴多发转移可能性大，间叶源可能。

结果

1、2023-11-22 手术：子宫色暗紫；囊性肿物包绕腹主动脉、肠系膜下动脉，肿物大小约 5*4*1cm，与周围组织联系密切，边界不清，质较软，内为糟粕质地，有囊液；右髂总血管分叉处内侧有一囊实性肿物，大小约 3*3*1cm，后为骶骨，联系密切；右附件区盆腔淋巴结位置可见一囊实性肿物，大小约 2*2*0.7cm。

2、术后病理：镜下见血管、脂肪及卵圆形或短梭形肿瘤细胞混杂生长，偶见核分裂像，局部见坏死，结合免疫组化结果，符合恶性血管周上皮样细胞肿瘤(PECom)，局部可见脉管内瘤栓；(左侧盆腔、右侧盆腔)淋巴结见肿瘤组织；免疫组化结果:CK(-)、Vimentin(+)、HMB45(部分+)、Melan-A(部分+)、S-100(-)、SMA(+)、TFE-3(+)、Ki-67(+3%)、ER（+3%）、PR（+40%）。行基因检测提示 TSC2 突变。

3、术后随访：术后放疗 12 次，末次放疗 2024-1-24。术后规律服用依维莫斯、醋酸甲羟孕酮。随访至 2024-7，患者一般情况良好。

结果 原发于腹膜后的 PEComa 临床表现缺乏特异性，手术切除是目前为止最可靠有效的方法，尽可能做到切缘阴性，术后辅助放化疗的意义上尚不能明确，肿瘤的基因学特征提示 mTOR 抑制剂可能有效。目前临床上关于腹膜后 PEComa 的治疗经验不足，更应针对临床表现、术中所见、术后病理及基因检测进行个体化诊疗。

PO_493

Research Advances in Mitochondrial-Derived Peptides and Their Association with Tumors

Ziting Chen Boyi Ma Chenlou Ren Yadong Yin Jia Xi Hong Yang*
Department of Obstetrics and Gynecology, XiJing Hospital, Air Force Medical University

Mitochondrial-derived peptides (MDPs) represent an emerging class of biologically active polypeptides (typically comprising less than 100 amino acids) that are encoded by short open reading frames (sORFs) within mitochondrial DNA (mtDNA). This finding represents a paradigm shift in our understanding of mitochondrial biology, as these novel regulatory peptides are synthesized through an unconventional pathway that circumvents the canonical mitochondrial translation machinery. Currently, eight distinct MDP family members have been characterized (including humanin, MOTS-c, and

SHLPs1-6), all of which are initially translated by cytoplasmic ribosomes before undergoing specific mitochondrial import – a biosynthetic route that fundamentally challenges traditional understanding of mitochondrial proteome generation. Extensive research has elucidated the multifaceted physiological roles of MDPs in maintaining cellular homeostasis. These peptides function as crucial metabolic modulators and stress response mediators through their ability to: The following three processes must be orchestrated: metabolic reprogramming, the fine-tuning of oxidative stress defense mechanisms, and the regulation of critical cell survival signaling cascades. In the context of oncology, the pleiotropic effects of MDPs are of particular significance, as they demonstrate context-dependent dual functionality by modulating metabolic adaptations specific to cancer, such as the Warburg effect, remodeling the tumor microenvironment and mediating cellular responses to chemotherapeutic agents. For instance, humanin exerts anti-apoptotic effects through direct interference with BAX activation, while SHLP2 demonstrates tumor-suppressive properties via negative regulation of the oncogenic IGF-1/AKT signaling axis. The pathological relevance of MDPs extends beyond cancer to encompass various age-related disorders, including neurodegenerative diseases, metabolic syndromes, and programmed cell death dysregulation. While the oncological implications of MDP dysregulation are increasingly recognized, several critical knowledge gaps persist. For example, the exact molecular mechanisms underlying their altered expression in cancer remain poorly characterized. The complete repertoire of MDP family members may not yet have been fully catalogued, and the precise signaling networks through which MDPs exert their tumor-modulating effects require systematic elucidation. This comprehensive review critically evaluates the current understanding of three principal MDP subfamilies (humanin, MOTS-c, and SHLPs), with particular emphasis on their emerging roles in malignant transformation and cancer progression. The objective of this study is to integrate recent advances in MDP biology with clinical oncology perspectives, with a view to highlighting promising translational opportunities for developing MDP-based diagnostic strategies and targeted anticancer therapies.

PO_494

基于 SEER 数据库的年轻外阴癌预后列线图构建与验证研究

傅怡馨 孙阳*
福建省肿瘤医院

外阴癌的发病率在持续上升，其中以年轻妇女的发病率上升尤为明显。与老年患者相比，年轻人往往表现出不同的临床特征、预后和治疗的反应。然而，目前对这一特定患者群体的研究仍然有限。本研究的目的是建立一个预测早发型外阴癌（EOVC）总生存期（OS）和癌症特异性生存期（CSS）的 nomogram。EOVC 患者从监测、流行病学和最终结果（SEER）数据库中抽取，并进一步随机分配到训练组和验证组。采用多变量 Cox 回归分析，识别对 OS 和 CSS 有显著影响的独立预后因素，构建 nomogram。采用 C 指数、校正曲线、受试者工作特征（ROC）曲线和决策曲线分析（DCA）曲线评价 nomogram 预测效果。共有 1386 例患者纳入生存分析，其中训练集 970 例，验证集 416 例。最终确定 OS 的预测因素包括年龄、婚姻状况、病理类型、分化程度、N 分期、SEER 分期、手术和放疗。而预测 CSS 的因素为病理类型、分化程度、N 分期、肿瘤部位、SEER 分期、手术、放疗及化疗。训练集中 OS 和 CSS 的 C 指数分别为 0.722 和 0.817。训练集 1、3、5、10 年 OS 的 AUC 分别为 0.821、0.801、0.772、0.738，CSS 的 AUC 分别为 0.891、0.884、0.854、0.822，与验证集的 AUC 基本一致。校正曲线显示预测值与实际观测值吻合较好。DCA 曲线表明改模型预测效能优于 TNM 分期。因此，在本研究中，我们建立并验证了一个能准确预测 EOVC 1、3、5、10 年 OS 和 CSS 的 nomogram，为临床提供了指导。

PO_495

腹膜播散性平滑肌瘤病的研究进展

许晓悦
广西医科大学

腹膜播散性平滑肌瘤病（LPD）是一种罕见的妇科疾病，其特点是腹膜表面散布着多个大小不一的平滑肌结节。虽然其确切病因尚不清楚，但相关研究表明激素、遗传和分子因素对疾病的发展有一定的影响。成像技术的进步，尤其是磁共振成像（MRI）和计算机断层扫描（CT），通过区分 LPD 和恶性肿瘤提高了诊断的准确性，而免疫组化标记物，如平滑肌肌动蛋白（SMA）、结蛋白和雌激素/孕激素受体（ER/PR），则提供了有价值的病理学见解。治疗策略包括激素治疗（如促性腺激素释放激素[GnRH]激动剂）和手术干预，治疗计划根据疾病严重程度和患者的具体情况量身定制。最新研究表明，靶向分子疗法在治疗难治性病例中可能发挥作用。尽管目前已经取得了这些研究进展，LPD 仍然是一种复杂的疾病，需要进一步研究以明确其发病机制，确定可靠的生物标志物并制定标准化的治疗方案。本综述总结了目前有关 LPD 的知识，以提高临床认识，并指导未来对其发病机制、生物标志物和治疗策略的研究。

PO_496

Salvia miltiorrhiza extract protects against cyclophosphamide-induced ovarian damage through multidimensional mechanisms

Xinyu Tang[1] Kezhen Li[2*]
1.华中科技大学同济医学院附属同济医院
2.华中科技大学同济医学院附属同济医院

Cyclophosphamide (CTX) is a high-risk ovarian injury agent due to its broad range of damaging mechanisms, but single compounds struggle to provide comprehensive protection, and there are currently no targeted protective agents. However, the multi-component nature of traditional Chinese medicine (TCM) offers hope for prevention and treatment against multi-mechanistic drugs causing ovarian damage. In this study, we aimed to investigate the protective effects of SM on the decline of ovarian function induced by CTX and explore its potential underlying mechanisms in mice. Primarily, SM had no impact on the anti-tumor effect of CTX. SM significantly increased the number of primordial follicles and decreased the number of atretic follicles. Additionally, SM increased serum levels of estrogen (E2) and anti-Mullerian hormone (AMH), while decreasing levels of follicle-stimulating hormone (FSH), resulting in improved fertility outcomes. Our findings showed that SM affected apoptosis through caspase pathway, attenuated oxidative stress induced by CTX by reducing accumulation of reactive oxygen species (ROS), enhancing the activity of antioxidant enzymes, and reversing glutathione (GSH) depletion, at the same time, alleviated vascular damage. In conclusion, this study provides multidimensional evidence that SM exerts protective effects on the ovaries through various mechanisms, thereby supporting its potential clinical efficacy in mitigating chemotherapy-induced decline of ovarian function.

PO_497

Paclitaxel exposure only transiently affected maternal oocyte quality and did not affect growth and development of offspring

Xinyu Tang[1] Kezhen Li[2*]
1.华中科技大学同济医学院
2.华中科技大学同济医学院附属同济医院

Paclitaxel (PTX) is a widely used chemotherapeutic agent, and there is no study to elucidate the effect of PTX exposure on the normal growth and development of offspring. To determine the effect and duration of PTX on parental oocyte quality, fertility and offspring, 7-week-old ICR female mice were given clinical doses of PTX in this study, in vitro maturation of oocytes and mating experiment were conducted separately on day 1, day 6, day 11 and day 16 after PTX administration, we assessed the impact of PTX on offspring growth and development, learning and memory abilities, as well as genomic stability. PTX not only reduced the number of mature oocytes but also impaired the developmental capacity of oocytes, with this effect lasting for 2 estrous cycles. The effect on fertility was mainly reflected in an increase in the number of stillbirths pups and a decrease in pregnant rate, which lasted 1 estrous cycle. In addition, the results showed that PTX exposure did not affect the normal growth and memory ability of offspring, but the impaired learning ability observed in offspring produced 1 day after maternal exposure to PTX may be associated with the exposure. Notably, whole-exome sequencing (WES) revealed a significant increase in genomic instability in offspring 1 day after maternal PTX exposure, which disappeared in offspring produced after 6 days or more of maternal exposure.

PO_498

Diagnostic Challenge: Functional Non-Communicating Rudimentary Horn with Ovarian Endometriosis and Ipsilateral Renal Agenesis

Manyu Li[1] Xin Zhang[2]
1.The Fourth Hospital of Hebei Medical University
2.The Bethune International Peace Hospital

A teenage girl presented with sudden lower abdominal pain following exercise, prompting initial consideration of ovarian cyst rupture by the attending emergency physician. However, the presence of unilateral renal absence on Computed Tomography (CT) imaging strongly suggested a reproductive tract malformation, later confirmed by Magnetic Resonance Imaging (MRI). Surgical exploration during laparotomy revealed a functional non-communicating rudimentary horn with associated ovarian endometriosis, in the setting of chronic dysmenorrhea. This rare case underscores the essentiality of comprehensive preoperative assessment to identify anatomical variations, guide surgical decision-making, and prevent diagnostic errors. Subsequent literature review focused on the co-occurrence of endometriosis in rudimentary horns and congenital urinary system abnormalities.

PO_499

米非司酮治疗子宫肌瘤的研究进展

薛惠 李倩倩 陈熠馨*
齐鲁医药学院

子宫肌瘤是育龄期女性常见的良性肿瘤，可引起月经异常、疼痛甚至不孕等症状，严重影响患者生活质量。米非司酮作为一种选择性孕激素受体拮抗剂，因其拮抗孕激素作用，已成为药物治疗子宫肌瘤的重要选择。但临床上米非司酮用药方案不一，本综述旨在系统梳理近年来米非司酮治疗子宫肌瘤的研究进展，以期为子宫肌瘤临床治疗方案提供借鉴及参考。

PO_500

1 例泪道阻塞合并妇科肿瘤患者长期糖皮质激素治疗致白内障的护理体会

崔小欢[1] 滕红飞[2] 芦岩双[1] 李英英[1] 吕秀丽[1]
1.河北省眼科医院 2.邢台医学院第二附属医院

目的 探讨在妇科肿瘤合并泪道阻塞患者中，长期使用糖皮质激素导致白内障的临床表现及护理干预策略，总结多病共存背景下个体化护理的关键要点，提升护理人员对慢性激素相关眼部并发症的识别与管理能力。

材料与方法 患者为一名既往宫颈癌术后复发病史的中老年女性，因“反复流泪、眼部分泌物增多”就诊于河北省眼科医院眼整形泪器病科，诊断为泪道阻塞并拟行泪道再通术治疗。护理过程中，患者多次诉说“视物模糊、畏光明显”，护理人员在评估中主动记录并报告医师，医师结合裂隙灯检查等辅助检查，发现患者双眼存在明显皮质性白内障表现。进一步了解病史发现，患者因妇科肿瘤复发，长期接受糖皮质激素治疗用于控制放化疗相关免疫反应，初步判断为药物性白内障。护理团队随后开展个体化护理措施，包括：泪道卫生指导、用药依从性教育、眼部症状持续评估、并发症健康宣教及心理支持干预，协助患者建立正确的疾病认知及就医路径。

结果 在护理干预与复查过程中，患者泪道术后恢复良好，流泪症状缓解，眼部分泌物明显减少，无感染并发症。护理人员定期观察患者视物情况变化，并指导患者及时接受白内障进一步治疗。患者在后续复查中反映护理服务专业、细致，能够及时发现并提醒潜在问题，积极配合后续眼部管理，表达高度认可。通过护理团队的系统干预，患者对糖皮质激素相关眼部不良反应有了明确认识，增强了自我管理意识，焦虑等负面情绪也逐渐缓解。

结论 本例显示，在眼整形泪器病科接诊的患者中，护理人员不仅要关注局部疾病本身，还应具备对系统性疾病史和药物使用背景的敏锐识别能力。对于接受长期糖皮质激素治疗的妇科肿瘤患者，尤其应警惕其可能诱发白内障等眼部并发症。通过早期识别、及时转诊及全程个体化护理管理，既保障了手术疗效，也提升了患者整体就诊体验与满意度。

PO_501

Clinicopathological characteristics of Fumarate hydratase-deficient leiomyomatosis and renal cell carcinoma including hereditary and sporadic forms

qi chen Yuanjing Hu*
Tianjin Central Hospital of Obstetrics and Gynecology

Objective To investigate the clinicopathological and molecular genetic characteristics of hereditary and sporadic leiomyomatosis and renal cell carcinoma with fumarate hydratase (FH) deficiency.
Methods 8 patients with FH-deficient uterine leiomyoma from May 2020 to August 2023 in Tianjin Central Hospital of Obstetrics and Gynecology. Fumarate hydratase (FH), S-(2-succino)-cysteine (2SC) and p16 was detected by immunohistochemistry in tumor tissues of all cases. The cell proliferation and immune cell infiltration in tumor tissues were observed by HE staining. The whole exon of FH gene was sequenced by next generation sequencing on tumor tissue and peripheral blood leukocytes.
Results The 8 patients with FH-deficient leiomyoma were 28-54 years old, with a mean age of (39.87 ± 7.54). 87.5% (7/8) had clinical symptoms, multiple fibroids and large size (median maximum volume was 75 mm). The leiomyoma patients with FH deficiency were divided into hereditary and sporadic FLRCC by FH gene sequencing. Patients with HLRCC had an earlier onset, and the serum tumor marker CA125 was more significant. Moreover, tumor tissues from patients with hereditary and sporadic FH-deficient LRCC differed in immunohistochemical and HE staining characteristics, including more positive p16 and susceptibility to invasion and metastasis in patients with HLRCC, and malignant proliferation in patients with sporadic FH-deficient LRCC.
Conclusion Our findings indicated subtle differences in the age of onset, immunohistochemical and histopathological features of hereditary and sporadic FH-deficient LRCC, facilitating the understanding and clinical diagnosis of FLRCC.

PO_502

泛凋亡的分子机制及其在肿瘤治疗中的研究进展

巩文建
武汉协和医院

研究目的 细胞死亡是生命体中的基本生物学现象，程序性细胞死亡（PCD）通过精确基因调控参与个体发育、机体稳态维持和病原体抵御等重要过程。目前研究最深入的三种 PCD 形式为焦亡、凋亡和坏死性凋亡。近年来研究发现这三种途径在多种疾病中可同时激活，基于此提出了泛凋亡（PANoptosis）概念，它整合三种细胞死亡途径形成复杂调控网络。泛凋亡在病毒感染、细菌感染和肿瘤等病理状态下发挥关键作用，其相关分子异常表达与肿瘤预后密切相关。本研究旨在系统阐述泛凋亡的分子机制，探讨其在肿瘤治疗中的应用前景。
材料与方法 本综述采用文献综述方法，系统回顾了泛凋亡的相关研究进展。通过 PubMed、Web of Science 和 CNKI 等数据库检索关键词“PANoptosis”、“PANoptosome”、“ZBP1”、“肿瘤治疗”等，筛选时间范围为 2019 年至 2025 年的英文和中文文献，共纳入 127 篇高质量论文，包括原创研究、综述和临床试验报告。文献筛选标准包括：（1）与泛凋亡分子机制直接相关；（2）涉及肿瘤治疗应用；（3）有明确的实验证据或临床数据。
结果 泛凋亡同时涉及焦亡、凋亡和坏死性凋亡的协同作用，形成复杂细胞死亡调控网络。泛凋亡复合体（PANoptosome）是其关键特征，包括以 ZBP1、AIM2、RIPK1、NLRP12 和 NLRC5 为核心的多种类型，均由传感器、接头蛋白和效应分子三个关键组分构成。在特定条件下，传感器识别刺激信号

后通过接头蛋白启动复合体形成，同步激活多种死亡通路。泛凋亡能有效抑制肿瘤细胞生长、增殖和耐药性，在先天免疫应答中发挥重要作用，可激活抗肿瘤免疫，促进"冷"肿瘤向"热"肿瘤转化。
结论 泛凋亡作为新型程序性细胞死亡形式，独特整合三种死亡途径形成复杂分子网络，在肿瘤治疗中显示重要应用价值。尽管取得进展，但其在肿瘤发生发展中的分子机制仍需深入研究。未来通过靶向泛凋亡复合体可直接促进肿瘤细胞死亡并增强免疫应答。本综述为肿瘤治疗策略开发提供新思路，有望推动更多临床转化研究。

PO_503

中国妇科医生的 HPV 疫苗接种状况及态度：一项横断面调查

黄月 何密斯 李巧玲 郭明芳 何昊 王晶 李秀英 隆玲 周琦 邹冬玲*
重庆大学附属肿瘤医院

背景 妇科医师是疫苗推广的主要群体，既往的研究较少关注他们 HPV 疫苗接种情况。了解该群体对疫苗的接受度及真实态度，以及未接种疫苗的原因，对推广疫苗具有重要意义。

方法 2024 年 6 月至 2024 年 8 月期间，依托中国抗癌协会肿瘤内分泌专业委员会这一平台，在中国的 6 个城市实施横断面调查。采用方便样本策略向这 6 个城市的三级公立医院妇科医师发放问卷。采用例数（百分比）形式进行描述，组间比较使用 χ^2检验或 Fisher 精确概率法，并通过 Logistic 回归方程分析影响因素。.
结果 总共收到问卷 577 份，其中有效问卷 563 份。中国女性妇科医师的疫苗接种率为 46.07%(223/484)。在所接种的疫苗中，四价 HPV 疫苗占比最高达 40.36%。未接种疫苗的主要原因是年龄超过疫苗限制，其次是性别为男性。12.35%的妇科医生因每年进行宫颈癌筛查而不愿接种疫苗。83.13%(468/563)的妇科医师支持男性接种疫苗。中国妇科医师对未成年女儿接种 HPV 疫苗的支持率显著高于其对未成年儿子接种该疫苗的支持水平，且差异具有统计学意义（87.74%（494/563）vs.68.92%（388/563），$p < 0.001$）。
结论 妇科医师对男性 HPV 疫苗的接受程度相对较低，且在一定程度上忽视了 HPV 感染可导致肛门、外阴、阴道、阴茎以及头颈部等部位肿瘤的风险。此外，HPV 疫苗预防上述部位肿瘤的作用被忽略了。

PO_504

3D 打印个体化阴道施源器在阴道癌腔内近距离治疗中的应用研究

郭玉萍
新疆肿瘤医院

研究目的 本研究采用回顾性分析，通过比较 3D 打印个体化阴道施源器与传统单通道阴道柱形施源器在原发性阴道癌近距离放疗中的计量学差异，确定阴道癌根治性放疗的体积剂量和局部控制率的关系。为原发性阴道癌患者的近距离治疗提供一定的指导依据。
方法 选取 58 例自 2020 年 1 月-2024 年 12 月接受 192 Ir 高剂量率近距离治疗的原发性阴道癌患者,38 例患者采用 3D 打印多通道施源器，28 例患者采用常规单通道阴道施源器放疗。进行 CT 扫描定位,并制定相应计划及评估,运用逆向剂量优化算法计算后得出剂量体积直方图,比较两组施源器用于近距离治疗时的高危临床靶区、膀胱及直肠受照剂量的差异。
结果 采用 3D 打印个体化多通道施源器计划的 D90%、D100%、V100%、V150%与常规单通道施源器设计的计划相比,差异均无统计学意义(P>0. 05)。两组 CTV D90 EQD2 的平均值分别为 86Gy 和 84Gy，

差异均无统计学意义(P>0. 05)。CTV D90 EQD2的平均值＞80Gy的患者的2年有效控制率分别为89%和86%，差异无统计学差异(P>0. 05)。采用3D打印个体化多通道施源器膀胱、直肠的D2cc与常规单通道施源器相比均明显降低,差异有统计学意义(均为P<0. 05)。
结论 阴道癌2年局控率与CTV D90有显著相关性。采用3D打印技术制作的多通道个体化施源器靶区适形性好,可更好地保护膀胱和直肠,有一定的治疗优势。

PO_505

Mechanism of Lactate Dehydrogenase A Regulating Immune Cell Functions to Influence the Progression of Gynecological Tumors

Xiaole Song Ruixia Guo Fang Ren*
The First Affiliated Hospital of Zhengzhou University

Objective This review elucidates the mechanisms by which lactate dehydrogenase A (LDHA) and its metabolic product lactate modulate immune cell functions within the tumor microenvironment of gynecological cancers including ovarian, cervical, and endometrial carcinomas. By dissecting LDHA-mediated interactions between metabolic reprogramming and immune dysregulation, this work establishes a theoretical foundation for novel therapies targeting antitumor immunity in gynecological malignancies.
Materials and Methods Comprehensive literature analysis evaluated both the direct effects of LDHA on immune cell intracellular signaling pathways such as PI3K-AKT and HIF-1α in tumors, and the indirect effects mediated by lactate—a key metabolite generated through LDHA-driven glycolysis—on tumor and immune cells within the tumor microenvironment. Key immune populations analyzed comprised lymphocytes ($CD4^+$ T, $CD8^+$ T, NK, and B cells) and myeloid cells (macrophages, dendritic cells, monocytes, neutrophils), with specific focus on functional alterations under LDHA modulation across gynecological cancer models.
Results LDHA overexpression consistently correlates with poor prognosis and metastatic progression across gynecological malignancies. It dually regulates antitumor immunity through cell-autonomous and non-cell-autonomous pathways. In lymphocyte populations, LDHA enhances Th1-mediated interferon-γ secretion yet simultaneously promotes regulatory T cell differentiation via lactate-MCT1-dependent 2-hydroxyglutarate production, accelerating PD-1 expression and conferring immunotherapy resistance. While initially boosting $CD8^+$ T cell cytotoxicity through perforin and granzyme B production, excessive lactate accumulation ultimately impairs interferon-γ expression and chemotactic capacity in advanced disease stages. Natural killer cells exhibit LDHA-dependent cytotoxicity maintenance under physiological conditions, but acidic tumor microenvironment conditions downregulate NKp46 receptors and induce apoptosis, compromising tumor surveillance. B cell differentiation into antibody-secreting plasma cells is impaired by LDHA knockdown, whereas Epstein-Barr virus-associated malignancies demonstrate LDHA-driven B cell proliferation. In myeloid compartments, lactate inhibits monocyte migration while promoting M2 macrophage polarization through AKT/ERK-HIF-1α-ARG1 axis activation, enhancing vascular endothelial growth factor and transforming growth factor-β secretion to drive tumor angiogenesis. Dendritic cells display maturation defects due to lactate-mediated interleukin-12 suppression, and tumor-associated neutrophils differentiate into pro-tumorigenic N2 subtypes under high-LDHA conditions. The acidic tumor microenvironment forms a self-reinforcing cycle wherein low pH upregulates LDHA expression via c-Myc transcriptional activation while sustained acidosis inhibits immune surveillance. Preclinical intervention studies demonstrate that pharmacological LDHA inhibitors effectively reduce lactate levels, restore $CD8^+$ T cell effector function, and potentiate programmed death-1 blockade efficacy in gynecological cancer models.
Conclusion LDHA critically shapes immunosuppressive microenvironments in gynecological cancers through metabolic reprogramming of both tumor and immune cells. Its dual immunoregulatory effects—enhancing specific antitumor responses while simultaneously establishing immunosuppressive

networks—highlight substantial therapeutic potential. Strategic targeting of LDHA activity, particularly when combined with immune checkpoint inhibition, may effectively reverse tumor-associated immunosuppression and improve clinical outcomes. Further clinical validation of LDHA inhibitors across gynecological cancer subtypes represents an essential next step, with this review underscoring metabolic pathway modulation as a fundamental strategy to overcome immunotherapy resistance in gynecological oncology.

PO_506

Synergizing Radiotherapy and Immune Checkpoint Inhibitors in Malignant Solid Tumors: Mechanistic Insights and Translational Frontiers

Dai Jiahui[1] Lingwei Ma[2] Xinyi Han[2] Wei Wang[2*]
1.1The First Affiliated Hospital of Guangzhou Medical University
2.Shanghai First Maternity and Infant Hospital

Radiotherapy (RT) and immune checkpoint inhibitors (ICIs) have each revolutionized the therapeutic landscape of malignant solid tumors. RT exerts its therapeutic effects not only through direct cytotoxicity but also by modulating the tumor microenvironment (TME). In contrast, ICIs function by targeting key inhibitory pathways—such as programmed cell death 1 (PD-1)/programmed cell death ligand 1 (PD-L1) and cytotoxic T lymphocyte antigen 4 (CTLA-4)—to unleash T cell–mediated antitumor immunity. A growing body of evidence indicates that RT can enhance tumor immunogenicity and synergize with ICIs to elicit systemic antitumor responses, particularly through mechanisms such as the abscopal effect, immunogenic cell death (ICD), and activation of the cGAS–STING pathway. This review offers a comprehensive analysis of the mechanistic interactions between RT and ICIs, and systematically summarizes recent clinical advances involving PD-1, PD-L1, and dual checkpoint blockade strategies. We will focus on the translational implications of these findings, the current challenges hindering their broader clinical application, and future research directions aimed at optimizing this combinatorial strategy to enhance therapeutic efficacy and achieve personalized cancer treatment.

PO_507

Organoid Models of Precancerous Lesions: Decoding Mechanisms and Advancing Precision Prevention

汉馨仪[1,2] 马菱蔚[1,2] 代佳卉[3] 王薇[1,2*]
1.同济大学 2.同济大学附属妇产科医院 3.广州医科大学

Purpose This study explores the application of organoid technology in precancerous lesion research, addressing limitations of traditional models in sample acquisition, molecular heterogeneity, and dynamic progression.
Methods As a comprehensive review, we searched for and analyzed established organoid models of precancerous lesions in multiple organs that have been used for research.
Results Organoid models successfully replicated key molecular and pathological features of precancerous progression, revealing critical roles of genetic alterations, inflammatory microenvironment interactions, metabolic reprogramming , and immune evasion mechanisms. High-throughput screening identified potential therapeutic targets and biomarkers, demonstrating the platform's utility for drug discovery and personalized intervention strategies.

Conclusion Organoids provide unprecedented insights into precancerous mechanisms and clinical translation. Despite challenges in standardization and microenvironment modeling, they represent a pivotal platform for early cancer interception and personalized medicine.

PO_508

靶向磁热响应型磁性纳米粒子诱导铜死亡并激活 JAK/STAT 通路以增强肿瘤免疫治疗的机制研究

张焕芝[1] 杨洋[2] 王垚[2] 张慧[1] 唐劲天[2] 李小平[1*]
1.北京大学人民医院 妇产科 2.清华大学 工物系

目的 卵巢癌是严重威胁女性健康的、致死率最高的妇科恶性肿瘤。卵巢肿瘤异质性高、易发生化疗耐药，具有免疫抑制性"冷"肿瘤的特性等，成为影响卵巢癌疗效及预后的关键因素，亟需开发高效、精准靶向及增强肿瘤免疫治疗的递药系统。本研究拟以四氧化三铁（Fe3O4）为核，以二氧化硅（SiO2）为壳，掺入铜的同时将铜离子载体伊利司莫（ES）载入二氧化硅中，最后偶联靶向肿瘤整合素受体的三肽（RGD），制备具有主动靶向、高磁热效应、诱导铜死亡及免疫原性细胞死亡（ICD）等多重效应的载铜的、新一代液态磁性抗肿瘤纳米递药系统（RGD/Fe3O4@SiO2-Cu/ES），构建磁热免疫调控体系，为克服耐药卵巢肿瘤治疗提供新策略。
材料与方法 采用醇热法合成粒径约为 50nm 的单分散四氧化三铁 Fe3O4；随后使用缩聚法合成带二氧化硅外壳及掺杂铜的 Fe3O4@SiO2-Cu；利用超声法将药物铜离子载体伊利司莫（ES）装载于二氧化硅的孔中，合成 Fe3O4@SiO2-Cu/ES；偶联肿瘤组织靶三肽 RGD，构建磁性抗肿瘤纳米递药系统 RGD/Fe3O4@SiO2-Cu/ES。体外和体内实验检测对化疗耐药卵巢癌的抗癌活性研究，进行磁感应热疗-铜死亡治疗耐药卵巢癌分子机制研究，同时联合免疫治疗 αPD-L1 进行感应热疗-铜死亡-免疫治疗调控机制研究。
结果 成功构建液态磁性抗肿瘤纳米递药系统（RGD/Fe3O4@SiO2-Cu/ES），该递药系统具有肿瘤主动靶向性、良好的磁热效应、诱导肿瘤细胞铜死亡以及免疫原性细胞死亡（ICD）。体内证实该磁性纳米材料具有较好的生物相容性和强大的肿瘤抑制能力。与 αPD-L1 结合能够促进肿瘤部位免疫细胞的浸润，促进树突状细胞（DC）的成熟，并使肿瘤巨噬细胞向 M1 型发生极化。
结论 本研究设计的液态磁性抗肿瘤纳米递药系统，将阐述磁热免疫治疗调控创新理论，为耐药性卵巢癌治疗提供新的治疗方案和治疗思路。

PO_509

Crosstalk between Fibroblast reticular cells and immune cells in the lymph node: A strained and close relationship

Peiyu Chen[1] Fanke Zeng[1] Shuangshuang Guo[1] Liangsheng Fan[1] Wei Wang[1,2*]
1.The First Affiliated Hospital of Guangzhou Medical University
2.Department of Gynecology, Shanghai First Maternity and Infant Hospital

Fibroblastic reticular cells (FRCs) are a vital component of the stromal cell network that supports the function and organization of secondary lymphoid organs (SLOs). In the past few decades, the function of forming structural scaffolds with various microenvironmental niches has been well characterized. Recent studies have demonstrated that FRCs are major players in modulating immune cell behavior and enhancing immune response. The interaction of FRCs and immune cells maintains homeostasis in lymph nodes. Once in a pathological state like bacterial, viral infection, and cancer, it is nonnegligible that the crosstalk between FRCs and immune cells, immune molecules present in lymph nodes, and the possible mechanisms by which FRCs induce immune suppression through these interactions.

FRCs regulate the function of immune cells through secret cytokines to induce immune response and immunosuppression. However, research into the interaction between FRCs and immune cells remains exploratory. The study of these can bring new directions and thinking for the diagnosis and treatment of diseases.
In this review article, we summarize recent findings on the relationship between FRCs and immune cells in the lymph node and discuss the possibility of insights into potential directions for developing novel treatment strategies for FRCs in the future.

PO_510

一例世界罕见巨大腹膜后肿瘤

周波 江大琼*
武汉大学中南医院

本研究报道一例世界罕见腹膜后巨大实性肿瘤——腹膜播散性平滑肌瘤，是妇科肿瘤中非常罕见的疾病。由于该病早期症状无特异性，因此容易被患者忽视，延误病情。大部分患者初次就诊时已经发生不同程度的全身各器官功能障碍，临床处理非常棘手。本文报道了 1 例腹膜后来源巨大肿瘤患者，通过术前妇科肿瘤牵头，细致查体并结合影像学检查结果充分评估，同时联合多学科团队协作诊疗，制定精准的手术方案后行开腹探查加经腹腹盆腔巨大腹膜后肿瘤切除术，术后患者压迫症状消失，经过围术期的精细化管理，各器官功能基本恢复正常。对该类诊疗困难的患者，术前进行充分评估、围术期加强循环管理以及营养支持是本例患者治疗成功的关键，经多学科联合诊治获得成功，本例患者的诊治经验值得借鉴。

PO_511

微生物易位与宿主 - 微生物共进化在肿瘤发生中的重要作用

张嘉鑫 刘书艺 陈薇好 孙朝阳*
华中科技大学同济医学院附属同济医院

尽管微生物易位和肿瘤内微生物组作为癌变的关键驱动因素日益受到认可，但目前仍缺乏一个能将这些过程与宿主 - 微生物共同进化整合起来的统一框架。为填补这一空白，本综述提出了一种新颖的理论模型 ——“微生物易位与宿主 - 微生物共同进化驱动癌变”，该模型系统地连接了微生物动态变化、宿主适应及肿瘤生态之间的关联。本综述归纳了三大核心机制：（1）微生物突破宿主屏障的策略（如突破黏膜防御、免疫监视）；（2）肿瘤微环境（TME）中的双向分子对话，这种对话会驱动微生物代谢重编程与免疫逃逸；（3）生态阈值概念，即当微生物累积产生的 “适应性副产品”（如基因毒素、免疫抑制代谢物）超出宿主稳态所能承受的范围时，会不可逆地触发恶性转化。重要的是，对结直肠癌、肝癌、子宫内膜癌和肺癌的组织特异性分析，揭示了保守的微生物特征（如具核梭杆菌、肺炎克雷伯菌）和菌群失调模式，这凸显了易位驱动癌变的普遍性。本综述还从三个维度对转化医学策略进行了评估：屏障修复（黏蛋白恢复、噬菌体疗法）、微生物组调控（合成群落、粪便微生物移植）以及共同进化干预（靶向免疫检查点、微生物代谢物）。通过整合多组学方法与临床前模型，该框架推动了精准肿瘤学的发展，强调了量化生态阈值和优化治疗协同作用的必要性。本综述将肿瘤定位为动态生态系统，呼吁开展跨学科研究以破解宿主 - 微生物的进化军备竞赛，并将生态学见解转化为临床突破。

PO_512

转录组测序筛选子宫内膜异位症患者差异表达基因

张瀚煜[1] 顾成磊[2] 孟元光[2]
1.解放军总医院第一医学中心 2.解放军总医院第七医学中心

研究目的 研究子宫内膜异位症（EMs）患者异位内膜及在位内膜组织与非 EMs 患者正常内膜组织转录组的差异基因表达，分析其所涉及的主要基因和关键通路，探索相关表达基因在子宫内膜异位症中的诊断价值。
材料与方法 收集 7 对病理诊断明确为卵巢型子宫内膜异位症患者的异位内膜及在位内膜组织及 5 例非 EMs 患者的正常内膜组织，提取 RNA 并构建文库，进行转录组测序，根据测序结果发现异位内膜、在位内膜、正常内膜的差异表达基因，并通过 GO、KEGG 对差异基因进行功能富集和信号通路的分析，采用 RT-qPCR 对部分差异表达基因进行验证。
结果 转录组测序及生物信息学分析发现，与在位内膜相比，异位内膜中有 3547 个差异表达基因，其中 1547 个差异基因下调，2000 个差异基因上调。GO 分析显示，差异表达基因在免疫系统调节、免疫反应、细胞或亚细胞组分的运动等功能富集明显。KEGG 分析显示，差异表达基因在神经递质受体-配体相互作用、细胞因子-细胞因子受体相互作用、细胞黏附因子等途径中明显富集。对部分差异表达基因行 RT-qPCR 验证，发现 ITGA11、C7、IGHG1、IGHG2、IGKC、JCHAIN、LTF 显著上调，APCDD1 显著下调。与正常内膜相比，在位内膜中有 1287 个差异基因，其中 578 个差异基因下调，709 个差异基因上调。GO 分析显示，差异表达基因在体液免疫应答、丝氨酸酶活性、补体激活调节等功能富集明显。KEGG 分析显示，差异表达基因在神经递质受体-配体相互作用、P53 信号通路、cAMP 信号通路、细胞周期等途径中明显富集。对部分差异表达基因行 RT-qPCR 验证，发现 ITGA11、IGHG1、IGHG2、IGKC、JCHAIN、LTF 显著下调，C7 显著上调。
结论 EMs 异位和在位内膜及非 EMs 正常内膜在转录组学表达方面存在明显差异，且差异表达基因（ITGA11、IGHG1、IGHG2、IGKC、JCHAIN、LTF）可能成为子宫内膜异位症诊断的候选标志物。

PO_513

Dual engagement of T cells to tumor and myeloid cells augments anti-tumor immunity

Changrui Yang Wen Di*
Department of Obstetrics and Gynecology, Renji Hospital, Shanghai Jiao Tong University School of Medicine

Objective Immunotherapy, particularly immune checkpoint inhibitors (ICIs), has revolutionized cancer treatment. However, immunologically “cold” tumors such as ovarian cancer often resist ICIs due to a scarcity of tumor-specific T cells and the immunosuppressive tumor microenvironment (TME), even though they may harbor substantial infiltration of functional bystander T cells. To address this therapeutic limitation, we developed a tri-specific T cell engager (Tri-TCE), designed to simultaneously redirect bystander T cells toward tumor cells and counteract the immunosuppression mediated by myeloid cells within the TME.
Materials and Methods Using our T-cell Redirecting Oncology Yukon Immunoglobulin (TROY-Ig) platform, we developed a novel Tri-TCE, B7H3xCD3xPDL1, in a modular format: (i) dual Fab arms targeting the pan-cancer antigen B7H3 with enhanced specificity and affinity, (ii) a low-affinity CD3ε binder to activate T cells while minimizing TAA-independent activation, and (iii) a weak PDL1-binding domain to counteract immunosuppressive signals. The functional activity of B7H3xCD3xPDL1 was assessed through a series of in vitro and ex vivo assays, including co-culture systems, patient-derived tumor suspensions (PDTSs), and patient-derived tumor fragments (PDTFs). After evaluating the stability and pharmacokinetics (PK) of B7H3xCD3xPDL1 to ensure its robustness and therapeutic

potential, in vivo efficacy was assessed using humanized mouse models bearing ovarian tumors. Mechanistic studies included high-dimensional imaging cytometry and single-cell RNA sequencing (scRNA-seq). Additionally, using an independent cohort of diverse clinical samples, we developed a machine learning (ML) model trained on ex vivo cytotoxicity results and transcriptomic profiles to predict the efficacy of B7H3xCD3xPDL1 treatment.
Results Our comprehensive studies utilizing co-culture systems, patient-derived tumor suspensions (PDTSs), patient-derived tumor fragments (PDTFs), and humanized mouse tumor models, demonstrate that B7H3xCD3xPDL1 effectively activates T cells and induces tumor cell lysis. Mechanistically, imaging cytometry and single-cell sequencing analyses reveal that B7H3xCD3xPDL1 facilitates interactions between T and myeloid cells, leading to rapid T cell activation and cytotoxic transition. Importantly, its robust therapeutic effects depend on B7H3 expression on tumor cells and PDL1 engagement on myeloid cells, triggering IFN-γ-mediated macrophage reprogramming and IL-15 secretion, thereby establishing a positive feedback loop that amplifies T cell functionality. Finally, application of our ML-based stratification model demonstrated the feasibility of data-driven identification of responders, providing a potential companion diagnostic strategy for clinical deployment.
Conclusions Our study presents a novel Tri-TCE in a unique format, B7H3xCD3xPDL1, which not only redirects a broad range of T cells to eliminate tumor cells, but also reprograms immunosuppressive myeloid cells in the tumor microenvironment, highlighting a promising immunotherapy approach for solid tumors.

PO_514

Near Infrared II Excitation Conjugated Polymers-based Phototheranostic Nanoplatform for Hyperthermia-Enhanced Ferroptosis and Immunotherapy

Yingying Chen Ruixia Guo*
the first affiliated hospital of zhengzhou university

Ovarian cancer stands as the most fatal gynecological cancer with poor prognosis, predominately due to the low immunogenicity and immunosuppressive microenvironment. Ferroptosis possesses the capacity to initiate immunogenic cell death (ICD) and trigger robust anti-tumoral immunity. Herein, a near infrared II (NIR-II) excitation and pH-sensitive nanoplatform DCP@Pt@Fe is meticulously constructed through the integration of phenylboronic acid-modified NIR-II conjugated polymers DPP-CPDT-PBA (DCP), along with cisplatin (Pt) and Fe2+, and encapsulated within an amphiphilic DSPE-mPEG2000. The formulated nanoparticles exhibit outstanding NIR-II photothermal property and NIR-II imaging tracking capability. The thermo- and acidic microenvironment-responsive release of Pt and Fe2+ subsequently induces ferroptosis in cancer cells through Pt-mediated glutathione (GSH) consumption by forming stable Pt-GSH complexes and Fe2+-driven reactive oxygen species (ROS) generation via Fenton reactions. Notably, ferroptotic tumor cells exhibit high immunogenicity, reprogram tumor-associated macrophage polarization, promote dendritic cell maturation, mobilize T cell activation, and significantly reverse the immunosuppressive tumor microenvironment. Simultaneously, interferon-γ (IFN-γ) produced by tumor-infiltrating immune cells inhibits SLC7A11 expression in cancer cells, which contributes to the ferroptosis therapeutic efficacy. Consequently, our nanoplatform effectively eradicates ovarian cancer via synergistic NIR-II-PTT/ferroptosis/immunotherapy, providing an exciting new avenue for therapeutic interventions.

PO_515

Association between pelvic inflammatory disease and gynecological cancer: Insights from the NHANES 2013-2018 and Mendelian randomization analysis

Chuqi Liu[1] Yongzhen Guo[1] Dongmei Lei[1] Ruixia Guo[2*]
1.Third Affiliated Hospital of Zhengzhou University
2.First Affiliated Hospital of Zhengzhou University

Background Pelvic inflammatory disease (PID) is an infection of the female reproductive organs that causes substantial morbidity for women worldwide. Gynecological cancers also pose serious threats to women's health. However, the relationship between them remains controversial, necessitating further investigation into the correlation and potential mechanisms involved.
Methods We adopted a three-pronged translational strategy. (1) Population analysis: weighted multivariable logistic regression was applied to data from 4 316 women in NHANES 2013–18 to quantify the relationship between self-reported PID and incident ovarian, cervical, and endometrial cancers. (2) Genetic analysis: a bidirectional two-sample Mendelian randomisation (MR) framework combined 23 independent SNPs associated with PID in FinnGen (6 181 cases, 226 439 controls) with large cancer-consortia GWAS summary statistics to infer potential causal directions. (3) Mechanistic exploration: two-step MR evaluated 731 peripheral immune-cell phenotypes to identify mediators of any PID–cancer association.
Results In NHANES, women with a history of PID showed a markedly higher prevalence of ovarian cancer than those without PID (adjusted OR 4·94, 95 % CI 2·49–9·80); no significant associations were found for cervical or endometrial cancer after full adjustment. MR supported a positive relationship between genetically predicted PID liability and overall epithelial ovarian cancer (inverse-variance–weighted OR per log-odds unit 1·06, 95 % CI 1·01–1·12), driven predominantly by HGSOC (OR 1·08, 95 % CI 1·01–1·15). Reverse MR provided no evidence that genetic liability to ovarian cancer increases the risk of PID. Two-step MR suggested that elevated CD45 expression on HLA-DR–positive T cells mediated approximately 9 % of the PID–HGSOC association (indirect effect β = 0·006, 95 % CI 0·001–0·013).
Conclusion Converging epidemiological and genetic evidence indicates that PID may increase the risk of HGSOC, with chronic pelvic inflammation and activated T-cell subsets emerging as plausible contributors. Strengthening PID prevention and timely treatment, alongside mechanistic work targeting inflammation-immune interfaces, could help mitigate the burden of HGSOC.

PO_516

外阴疣状癌一例并文献复习

王娟[1] 周小铃[2*]
1.石河子大学 2.石河子大学第一附属医院

患者，女性，76 岁，以“发现外阴赘生物 7 月”为主诉入院。完善相关检查排除手术禁忌症，给予患者外阴局部扩大切除术对症治疗，术后结合患者临床表现及辅助检查结果，诊断为外阴疣状癌。患者术后恢复良好，随访半年无复发转移。因有关外阴疣状癌的报道较少，因此本文就该病的病因、诊断及治疗方法进行探讨。

PO_517

基于中性粒细胞与淋巴细胞比值的妇科肿瘤术前焦虑预测模型构建及验证

孙星宇 熊旺 毛熙光*
西南医科大学附属中医医院

背景与目的 术前焦虑在妇科恶性肿瘤患者中较为常见，可能影响术后康复、免疫功能及远期心理健康。近年来，系统性炎症在心理障碍发生中的作用受到关注。中性粒细胞与淋巴细胞比值（NLR）作为易于获取的炎症指标，或可用于辅助识别高焦虑风险人群。本研究旨在构建并验证一项融合 NLR 等多因素的术前焦虑预测模型，并探讨其在妇科肿瘤人群中的临床实用价值。
方法 本研究回顾性纳入 2021 年 1 月至 2023 年 12 月在某三级医院接受手术治疗的 1,120 例宫颈癌、卵巢癌或子宫内膜癌患者。术前使用汉密尔顿焦虑量表（HAMA）评估焦虑状态，HAMA≥14 定义为临床显著焦虑。排除感染、自身免疫疾病、慢性炎症或免疫抑制治疗等干扰因素。通过单因素及多因素 Logistic 回归筛选预测变量，应用 AIC 优化模型，Bootstrap 方法进行内部验证，并评价模型的区分度（AUC）、校准性及决策净效益（DCA）。
结果 术前焦虑检出率为 36.1%。最终模型纳入 9 项独立预测因子，包括：年龄较小、低教育水平、独居、缺乏家庭支持、FIGO III–IV 期、低白蛋白、睡眠障碍、精神病史及 NLR>3.54。NLR 作为关键炎症指标，其预测力优于 PLR 和 SII（aOR = 2.45，P < 0.001）。最终模型 AUC 达 0.938（95% CI: 0.925–0.951），校准良好，DCA 分析显示模型具有较高的临床净受益。亚组分析提示 NLR 在晚期及宫颈癌患者中的预测效能更为显著。
结论 本研究构建的多因素预测模型融合了临床、心理及炎症指标，具有良好预测性能，适用于术前焦虑高风险人群识别。NLR 作为常规检查中可得的炎症参数，在模型中展现出稳定而显著的预测价值，提示其作为心理–免疫互动桥梁的潜力。该模型可为术前心理筛查和个体化干预提供参考依据，亦为未来基于 NLR 的心理管理策略探索奠定基础。研究结果具有一定推广前景，适宜在学术会议中进一步交流与探讨。

PO_518

滋肾育胎丸改善与年龄相关的卵巢功 能减退和生育力下降的机制探究

罗忠玉 李科珍* 艾继辉
华中科技大学同济医学院附属同济医院

目的 探究滋肾育胎丸改善年龄相关卵巢功能减退和生育力下降的效应和机制。
方法 应用网络药理学对滋肾育胎丸作用于卵巢衰老的潜在靶点进行基因本体 和京都基因和基因组百科全书富集分析。检测 12～40 周龄的 C57 小鼠卵巢功能和生育力， 确定卵巢功能和生育力开始出现显著下降的小鼠周龄。将高龄 C57 小鼠随机分为对照组和滋肾组，滋肾组小鼠滋肾育胎丸连续灌胃给药 28 天（2.25g/kg/d），对照组给予等量蒸馏水。通过检测卵巢指数，血清促卵泡激素、雌二醇和抗苗勒氏管激素水平、动情周期和各级卵泡计数，对滋肾育胎丸改善高龄小鼠卵巢功能的效应进行分析。通过体外受精、平均排卵数检测、平均胚胎种植数检测、妊娠率和平均产仔数检测，对滋肾育胎丸改善高龄小鼠生育力的效应进行分析。机制探究部分，检测两组小鼠卵巢组织磷脂酰肌醇 3-激酶和蛋白激酶 B 蛋白表达水平，线粒体 DNA 转录水平，以及线粒体融合和分裂相关蛋白表达水平。获取两组小鼠卵母细胞，检测线粒体膜电位、线粒体分布、活性氧和丙二醛水平、纺锤体组装、异常纺锤体样小

头畸形相关蛋白和核有丝分裂装置蛋白转录水平，以及细胞凋亡调节因子 Bax 和 B 细胞淋巴瘤-2 转录水平。

结果 网络药理学分析结果显示，滋肾育胎丸作用于卵巢衰老的潜在靶点与氧化还原反应、线粒体、细胞增殖和凋亡密切相关，且在 PI3K/Akt 通路上显著富集。12～40 周龄 C57 小鼠卵巢功能和生育力检测结果显示，在第 28 周龄，小鼠的卵巢功能和生育力开始出现显著下降。滋肾育胎丸改善高龄小鼠卵巢功能和生育力检测结果显示，与对照组相比，滋肾组小鼠卵巢指数显著升高，血清 E2 和 AMH 水平显著升高，动情周期更加规律，始基卵泡和窦卵泡计数显著增加，IVF 四细胞率和囊胚率显著增加，平均排卵数、平均胚胎种植数、妊娠率和平均产仔均显著增加。机制探究结果显示，滋肾组小鼠卵巢组织 PI3K 和 Akt 蛋白表达水平显著升高，线粒体 DNA 转录水平显著升高，视神经萎缩蛋白和线粒体融合素 1 表达水平显著升高。滋肾组小鼠卵母细胞线粒体膜电位显著增加，线粒体异常分布减少，ROS 水平和 MDA 水平降低，异常纺锤体组装减少，ASPM 和 NUMA 转录水平上升，Bax 转录水平下降，Bcl-2 转录水平上升。

结论 滋肾育胎丸可能通过上调 PI3K/Akt 通路表达，抑制卵母细胞凋亡，改善线粒体功能，减少异常纺锤体组装，从而提高卵母细胞质量，改善年龄相关的卵巢功能减退和生育力下降。

PO_519

Preventing the birth of BRCA1 heterozygotes using PARP inhibitors: a non-invasive method for hereditary disease screening

Wei Jianbo Lv Mingwei Deng Dongmei Liang Zhiqing Leng Kaijian Wang Yanzhou Wang Jianliu Yang Jie Ai Jihui Li Kezhen*

Department of Gynecological Oncology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

Hereditary tumors are approximately 5-10% of malignant tumors, the hereditary breast-ovarian cancer syndrome (HBOC) is caused by germline BRCA1 gene mutations. Here we used a PARP inhibitor, Veliparib, to eliminate mouse embryos with BRCA1 heterozygous mutations and avoided the birth of BRCA1 heterozygous mutant offspring. Compared with the wild-type embryos, BRCA1 heterozygous embryos were more sensitive to Veliparib and showed more severe DNA damage. Treatment with 7.5 μM Veliparib effectively inhibited the development of BRCA1 heterozygous embryos, and transferring Veliparib-treated embryos to the uterus resulted in no births of BRCA1-deficient offspring. The F1 generation's behavioral experiments showed that this treatment did not affect any physiological function of the wild-type offspring. This study creates a successful example of screening healthy embryos from the functional level of proteins using targeted drugs and provides a new direction for non-invasive screening of embryos for genetic diseases in vitro.

PO_520

Injectable adhesive hydrogel microspheres with VEGFA-high expression MSC exosomes encapsulation for intrauterine adhesions treatment

Rong Ma[1] Zhengwei Sun[1] Zhihui Xu[1] Yuqing Jin[1] Yujia Zhou[1] Luoran Shang[2] Xueqiong Zhu* (*corresponding author)[1*]

1.Zhejiang Provincial Clinical Research Center for Gynecological Diseases, Department of Obstetrics and Gynecology, The Second Affiliated Hospital of Wenzhou Medical University, Wenzhou 325027, China
2.Shanghai Xuhui Central Hospital, Zhongshan-Xuhui Hospital and The Shanghai Key Laboratory of Medical Epigenetics, The International Co-laboratory of Medical Epigenetics and Metabolism (Ministry of Science and Technology), Institutes of Biomedical Sciences, Fudan University, Shanghai 200032, China

Purpose Hydrogel-based therapeutic strategies hold significant promise in the treatment of intrauterine adhesions, while their performance still needs improvement in terms of restoring the structure and function of the endometrium. Exosomes, particularly through genetic engineering, can enhance the expression of specific factors in mesenchymal stem cells (MSC) and their derived exosomes, thereby amplifying their therapeutic potential. Thus, it was conceived that by utilizing microfluidics and combining genetically engineered MSC-derived exosomes, a novel hydrogel system with both unique structural features and multifaceted capabilities and action mechanisms could be established for the treatment of IUAs.
Methods We proposed an injectable and adhesive hydrogel GelMA&PDA microspheres from microfluidic electrospray. The microspheres were encapsulated with VEGFA-high expression mesenchymal stem cells exosomes (MSC^{VEGFA} EXOs) for IUAs treatment. Leveraging the adhesive capabilities of polydopamine (PDA), the microspheres were assembled to form a unique hydrogel system (microspheres scaffold, denoted as MPS) that possesses not only injectability but also shape adaptation.
Results The resulting microsphere system exhibited excellent injectability, shape adaptability, biocompatibility, and sustained drug release profile. Due to these merits, the hydrogel systems effectively promoted cell migration, enhanced angiogenesis, and showed antibacterial, infection-preventing, and reactive oxygen species (ROS) scavenging properties in vitro. Moreover, in vivo studies indicated that this hydrogel system not only promoted neovascularization and tissue regeneration but also enhanced fertility.
Conclusions Therefore, the MSC^{VEGFA} Exos loaded injectable adhesive microspheres introduced an innovative bioengineering method for applying multifunctional exosomes derived from MSC in biomedicine, while also broadening the clinical potential of the related therapeutics.

PO_521

Near-single-cell Mapping of Protein Expression Landscape of Oocytes and Granulosa Cells during Human Folliculogenesis

Qiuyue Liao Xingyuan Hu Xiaolin Meng Qian Zhang Xu Na Xie Qin Wenju Peng Junpeng Fan Jie Yang Jihui Ai Kezhen Li*

Department of Gynecological Oncology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

The rapid growth of mass spectrometry (MS) measurements has enabled proteomic analysis of individual cell, adding the possibility to map cellular heterogeneity of precious samples. Here we applied a near-single-cell proteomics strategy to elucidate the dynamic changes of human oocytes and

granulosa cells (GCs) from the primordial stage to the preovulatory stage. Samples containing 3~10 oocytes or ~100 GCs of five developmental stages were monitored and more than 3000 proteins were quantified in each kind of cell. The proteome landscape of oocytes and GCs reflected the stage-specific expression dynamics during human folliculogenesis. Analysis of oocyte-GC interactions identified vital roles of gap junctions and ECM components in follicular development. Further comparison between transcriptome and proteome revealed that proteomic studies provided distinct and complementary information to transcriptomic analysis. Notably, we estimated the transcription factor (TF) activities of follicles with different developmental stages and proposed new molecular subtyping, which enabled further understanding of the molecular basis during folliculogenesis. Novel oocyte-specific membrane proteins and disease-related proteins which could facilitate the exploring and understanding of reproductive science were discovered and validated, which may benefit the application of recent developed techniques such as in vivo therapeutic delivery and in vivo cell tracing. Our study provides the proteome dynamics during human folliculogenesis and offers valuable resources for further molecular studies regarding biology and disease of human follicle and ovary.

PO_522

Antigens of HPV related cancers: Viral antigens and Neoantigens

Dudu Zhang yingying Han Shuaiqingying Guo Jing Yuan Xiaolin Meng Yingjie Hu Jing Chen Kezhen li*
Department of Gynecological Oncology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

Therapeutic vaccines for HPV-associated tumors have always been the emphasis, and screening for the most potential tumor antigens is the key to the success of tumor vaccines. HPV protein has always been the first choice for antigen screening, but neoantigens should also be sources of HPV-related cancer vaccines. Both HPV proteins and neoantigens need antigen epitope screening through the methodology of biological information to identify peptides showing apparent immune responses. Thus, we introduce the best antigen screening method for cancer patients, including the steps of sample collection, mutation identification, antigen prediction, and vaccine design. During screening, attention must be paid to antigen homogeneity, high expression, high affinity and stability of antigen-MHC, and strong immunogenicity. Meanwhile, some bioinformatics algorithms for predicting antigens have been summarized, such as NetChop20S, NetChopCterm, NetChop-3.0Cterm, NetMHCstabpan, etc. In this review, we outline the conditions and corresponding strategies for screening optimal antigens to improve the efficacy of HPV-associated cancers.

PO_523

From Maternal Exposure to F1 Development: Unveiling Cyclophosphamide-Induced Reproductive Challenges

Xiaolin Meng jihui Ai Kezhen Li*
Department of Gynecological Oncology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

Various conclusions exist regarding genetic toxicity and pregnancy risks of cyclophosphamide, creating uncertainties about optimal conception timing, obstetric outcomes, and offspring safety. Using a mouse model to mimic clinical dosing, we examined whether female mice's reproductive function could recover after cyclophosphamide exposure and if their offspring could survive and develop normally long-term. Our findings revealed that despite ovarian reserve declines post-treatment, pregnancy remains possible, with outcomes influenced by the drug discontinuation interval. Fertility was compromised initially, transiently recovered at two weeks, declined again at four weeks, and ultimately led to infertility. Long-term, F1 offspring from the cyclophosphamide group displayed growth restrictions, higher mortality rates, impaired neurodevelopment, and delayed puberty. Extending the drug withdrawal interval did not alleviate developmental defects, however, among the CTX groups with different intervals after drug withdrawal, the two-week group showed the lowest mortality rate among the offspring. Cancer survivors should prioritize pre-treatment counseling and fertility preservation. For those missing fertility preservation before chemotherapy, waiting two or three menstrual cycles before conceiving may mitigate risks, as optimal pregnancy outcomes and reduced offspring mortality occur during this time. Delaying conception could lead to lifelong infertility, depriving patients of opportunities for motherhood. This study highlights effects of cyclophosphamide on fertility and offspring, offering critical insights for tumor survivors.

PO_524

Precision medicine in premature ovarian insufficiency: a focus on the precision therapeutic strategies for mesenchymal stem cells

Rou Wang Fengyuan Liu Qiuyue Liao Jiachen Wu Lingjuan Wang Kezhen Li*
Department of Gynecological Oncology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

Premature ovarian insufficiency (POI) is one of the most common disorders that reduces fertility and the quality of life in young women. And mesenchymal stem cells (MSCs) have gradually been regarded as a promising therapeutic approach for POI for their potential in remodeling impaired ovarian function. However, specific details regarding the application of MSCs in POI treatment remain unclear. Key issues include the selection of appropriate MSCs sources, the optimization of culture conditions, and the determination of optimal transplantation protocols (encompassing dosage, administration route, and timing), all of which pose significant barriers to clinical translation of MSCs. Here, this paper systematically reviews the researches on MSCs-based POI therapy from mechanism to clinical application, with a focus on the key therapeutic details and provides the first integrated analysis of MSCs' dose efficacy inverted U-shaped relationship. Additionally, it highlights the application potential of fetal adnexa sources, in site transplantation, hypoxic environment and three-dimensional cultivation strategy for MSCs in POI treatment. Given the crucial role of MSCs' targeting ability and therapeutic duration in precision medicine, this paper further explores the challenges, existing strategies, and future development directions of MSCs in this field, intending to provide a comprehensive reference for future research and clinical translation of MSCs in female ovarian diseases.

PO_525

Salvia miltiorrhiza extract ameliorated ovarian damage induced by cyclophosphamide in mice by multidimensional mechanisms

Lin Zhou Hualin Bai Lingjuan Wang Juan Xiao Yi Liu Jihui Ai Kezhen Li* Ding Ma Yingyan Han
Department of Gynecological Oncology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

Introduction Cyclophosphamide (CTX) can result in particularly severe ovarian damage by the broad range of damaging mechanisms, yet now single compounds struggle to provide comprehensive protection against CTX-induced ovarian damage. Salviae Miltiorrhizae Radix et Rhizoma (SM), as the traditional Chinese medicines (TCMs) with multi-component nature, offers hope for prevention and treatment against multi-mechanistic drugs causing ovarian damage. In this study, we aimed to investigate the protective effect of SM on ovarian hypofunction induced by CTX and explore its underlying mechanisms in mice.

Methods The Salvia miltiorrhiza Bunge with a final concentration of 1.33 g/mL was administrated daily by gavage in mice with tumor xenografts and/or CTX intraperitoneal intervention. The subcutaneous xenograft tumor volume of each mouse was recorded and calculated every 3 days by measuring its width and length. After drug administration, estrous cycle was monitored and follicle counting was performed by H&E staining, as well as ELISA assays for serum hormone and mating experiment for fertility assessment. The apoptosis inhibition, antioxidant capacity, vascular protective and anti-fibrotic effects of SM was respectively evaluated by TUNEL staining, ROS, DPPH scavenging and antioxidant enzymes assays, vascular morphological observation, masson staining, as well as immunofluorescence staining and western blotting for the related-protein expression.

Results SM didn't affect the anti-cancer efficacy of CTX and tumor growth rate in vivo. After SM administration for 41 days, the dropped body weight and decreased ovarian index caused by CTX gained obviously, the disturbed estrus cycle was markedly restored, a significant increase emerged in the levels of AMH and E2 and a significant decrease appeared synchronously in FSH levels, a significant increase in primordial follicles and a corresponding decrease in atretic follicles was showed with the same increasing trend in the primary follicles, secondary follicles and antral follicles. Our results also showed that CTX could lead to pregnancy rate decreased, especially the average stillbirth rate raised obviously, both of which could be improved by SM, but without effect on the average litter size and body weight of alive pups. In-depth research showed that SM could effectively attenuate CTX-induced granulosa cells apoptosis to a certain extent by blocking the activation of the apoptotic proteins, also alleviate oxidative stress damage caused by CTX through its anti-oxidative stress ability, even reverse CTX-induced ovarian vascular occlusion and alleviate ovarian tissue fibrosis.

Conclusion Our findings verified that SM could significantly reduce primordial follicle (PMF) loss and follicular atresia, and ameliorate CTX-induced ovarian dysfunction and poor fertility, but didn't affect the anti-tumor effect of CTX and tumor growth. Further compelling evidence was provided that, the protective effect of SM may be related to apoptosis inhibition, low oxidative stress, vascular protection and anti-fibrotic effect, which further confirmed the comprehensive protection of SM on CTX-induced ovarian damage, thereby supporting its potential clinical efficacy in mitigating chemotherapy-induced decline of ovarian function.

PO_526

Maternal exposure to paclitaxel caused a temporary adverse impact on oocyte quality but not developmental competence in offspring

Hualin Bai Mengge Cui Qiuyue Liao Dongmei Deng Wang Lingjuan Jihui Ai Kezhen Li*
Department of Gynecological Oncology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

Research Question What are the effects of paclitaxel (PTX) exposure on parental oocyte quality and offspring development?
Design In the study, the developmental capacity of oocytes were evaluated by in vivo and in vitro PTX administration to female ICR mice. Subsequently, a mating experiment was implemented, and the offspring growth and development was observed and analyzed, as well as the learning and memory abilities, genomic stability, which were respectively evaluated by Morris Water Maze (MWM) and whole exome sequencing (WES).
Results A combination of in vitro and in vivo studies confirmed for the first time that PTX primarily affected oocytes at the early stage of meiosis, metaphase I (MI) stage, and not the germinal vesicle (GV) stage. In vivo study suggested that these adverse effects soon returned to normal levels and lasted not up to two oestrous cycles. Consistent with this, pregnancy rate only dropped sharply for a short period of time and then gradually recovered, without obvious impact of PTX on the live-birth rate, average litter size and body weight of offspring. Most importantly, the results showed that PTX did not affect the normal growth, learning and memory ability, genomic stability in offspring, just a higher frameshift number was found on Day 1 after exposure.
Conclusions Our study findings revealed that paclitaxel exposure at MI stage exerted temporary adverse impact on oocyte quality, and no obvious abnormality was observed in their offspring, the above of which suggested the low and unabiding ovarian toxicity of paclitaxel, and could provide laboratory basis for the recommended gestation time after paclitaxel withdrawal.

PO_527

妇科恶性肿瘤继发性骨盆转移患者治疗及预后分析 (附 29 例临床分析)

张慧[1] 张焕芝[1] 王琪[1] 汤小东[2] 郭卫[2] 孙秀丽[1] 王建六[1] 魏丽惠[1] 李小平[1*]
1.北京大学人民医院 妇产科 2.北京大学人民医院 骨肿瘤科

目的 旨在分析妇科恶性肿瘤继发性骨盆转移患者的临床特点及治疗方法，探究预后影响因素。
方法 纳入 2010 年 06 月至 2025 年 01 月收治于北京大学人民医院妇产科的 29 例妇科恶性肿瘤继发骨盆转移的患者，回顾性分析其临床病理特点、治疗措施等，并随访患者的 NRS 疼痛数字分级、KPS 活动状态评分、骨肿瘤保肢术后 MSTS-93 评分及生存状况，以探讨预后影响因素。
结果 29 例患者均获全程随访，中位随访时间 48.3 个月(0-91.5 个月)。患者原发肿瘤类型包括：卵巢恶性肿瘤 7 例、子宫体恶性肿瘤 11 例和宫颈恶性肿瘤 11 例。FIGO 临床或手术病理分期以 III 期(6 例, 20.7%)、IV 期(14 例, 48.3%)为主，组织学分级以Ⅱ级(15 例, 51.8%)、Ⅲ级(9 例, 31.0%)为主。14 例(48.3%)患者确诊原发肿瘤时同时发现骨盆转移，15 例(51.7%)患者在治疗或随访复查期间发现骨盆转移，其中后者发现骨盆转移与确诊原发肿瘤的中位时间间隔为 38 个月(3~120 个月)。依据 Enneking 骨盆分区标准，骨盆转移灶多发生于 II+III 区(9 例, 31.0%)或 I 区(9 例, 31.0%)，多数为单侧骨盆转移(22 例, 75.9%)，且多数合并骨盆外转移(21 例, 72.4%)。治疗情况：所有患者均接受了全身系统治疗，包

括化疗、靶向治疗、免疫治疗及骨改良药物治疗。局部治疗情况：接受放疗的患者共 5 例，接受手术治疗的患者共 7 例。4 例 II+III 区及 2 例单纯 I 区骨盆转移患者采取骨盆肿瘤切除术+人工假体重建术，其中 4 例使用 3D 打印半骨盆假体、2 例使用骨水泥假体进行骨盆重建。1 例 IV 区骨盆转移患者进行了肿瘤切除术+骶椎钉棒系统内固定术。治疗后患者的 NRS 疼痛分级和 KPS 评分相较于治疗前均有改善(P＜0.01)，同时，接受手术治疗患者术后较术前 NRS 评分和 KPS 评分的差值均优于未接受手术的患者[(6.0±1.5)和(35.7±9.8) vs [(3.1±1.4)和(24.5±11.8)(P＜0.05)]。此外，采取 3D 打印假体重建骨盆的患者术后 MSTS-93 评分明显高于采取其他方式进行骨盆重建的患者[(18.8±1.7) vs (14.7±2.1)(P＜0.05)]。患者的骨盆转移中位生存时间为 20.0 个月(5.9-34.1 个月)。预后影响因素：多因素 Cox 比例风险回归分析结果显示，组织学分级 II & III 级、非初发骨盆转移、双侧骨盆转移的患者生存期更短，而异时性骨盆转移及采取综合治疗为患者预后的保护因素(均 P<0.05)。

结论 妇科恶性肿瘤出现骨转移患者的预后不良，组织学分级 II & III 级、非初发骨盆转移、双侧骨盆转移的患者预后更差。3D 打印假体骨盆重建对患者生存期的影响无统计学意义，然而，精准的假体设计、严格的手术程序和合理的术后管理有助于可以缓解局部疼痛和改善患者的生活质量。

PO_528

绝经后宫腔积脓合并子宫内膜病变的 308 例患者临床分析

朱蕊
中国医科大学附属盛京医院

目的 探讨绝经后宫腔积脓患者中合并子宫内膜非典型增生或子宫内膜癌患者（AEH/EC）的临床特征与相关危险因素，为高风险患者的早期识别和干预提供依据，增强临床对潜在恶性病变的识别能力与应对效率。

材料和方法 回顾性分析 2013 年 1 月至 2024 年 8 月中国医科大学附属盛京医院妇产科收治的 308 例绝经后宫腔积脓患者，收集并分析患者年龄、初潮与绝经年龄、孕产史、症状、BMI、影像学特征以及实验室指标等临床资料。采用 t 检验、Mann-Whitney U 检验、卡方检验进行组间比较。采用 Logistic 回归分析子宫内膜良恶性病变与绝经后宫腔积脓患者临床症状的相关性。

结果 308 例绝经后宫腔积脓患者，其中良性病变组 242 例（78.57%），AEH/EC 组 66 例（21.43%）。患者平均年龄为 66.37 岁，绝经年龄中位数为 50 岁，发病主要集中于绝经后 10~20 年。AEH/EC 组患者较良性组妊娠及生产次数较少，超重及肥胖比例较高，合并糖尿病比例增加，子宫内膜增厚及宫腔积液深度明显增加，CDFI 更易检测到血流信号，且绝经后阴道流血的发生率更高，宫内节育器的使用比例明显较低，差异有统计学意义（*P*＜0.05）。年龄、初潮年龄、绝经年龄、绝经年限、吸烟史、高血压、宫颈癌筛查、阴道分泌物异常及主要实验室指标在两组间无统计学差异。多因素 Logistic 分析进一步证实，合并糖尿病（OR=2.71）、子宫内膜增厚≥5 mm（OR=11.26）、CDFI 血流信号（OR=5.32）和绝经后流血（OR=2.85）为独立危险因素（*P*<0.05），生产次数（OR=0.65）增加为保护因素。宫腔积液深度接近边缘显著（*P*=0.058），可能为潜在危险因素。基于多因素分析建立诊疗路径图，强调宫腔积液较多时应先抗炎及引流，避免医源性穿孔，并依内膜厚度、CDFI 血流信号、糖尿病及绝经后流血等因素综合判断内膜病变风险，指导后续诊刮及治疗决策。

结论 绝经后宫腔积脓与子宫内膜恶性病变相关，合并糖尿病、子宫内膜增厚（≥5 mm)、宫腔积液增多、血流信号阳性、绝经后阴道流血及少产史为其独立危险因素。本研究为优化绝经后宫腔积脓的诊治流程、加强对子宫内膜病变的早期识别提供了重要参考。

PO_529

Peripheral Blood Biomarkers for Predicting Response to PD-1/PD-L1 Inhibitors: A Comprehensive Review and Synthesis

Shuting Chen Yingjie Hu Kezhen Li* Hanxu Cheng Shuaiqingying Guo Yingyan Han
Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology

Programmed death 1/ Programmed cell death-Ligand 1(PD-1/PD-L1) inhibitors have revolutionized the landscape of cancer treatment by improving survival rates and alleviating disease burdens. In identifying biomarkers for treatment response, such as PD-L1 expression in tumor tissue, tumor mutational burden (TMB), and microsatellite instability-high (MSI-H), the methods are invasive and present challenges for continuous sampling. Consequently, the validity of peripheral blood indicators as biomarkers has garnered significant attention. This systematic review aims to investigate advancements in peripheral blood components as predictive biomarkers for assessing therapeutic response to anti-PD-1/anti-PD-L1 immunotherapy. The latest achievements based on peripheral blood testing technologies have been compiled in this study, specifically including tumor-derived biomarkers such as tumor-specific markers, circulating tumor cells (CTC), circulating tumor DNA (ctDNA), lactate dehydrogenase (LDH), and blood tumor mutational burden (bTMB). Additionally, it covers the fine subsets of immune cells and their related indicators, including lymphocyte count and neutrophil-to-lymphocyte ratio (NLR), T cells, B cells, natural killer cells (NK cells) and myeloid cells, along with cytokines and multiparameter models. Our review underscores that tumor-associated components and immune-related cellular/molecular factors in peripheral blood can effectively predict tumor patients' responses to PD-1/PD-L1 inhibitors. However, universally applicable biomarker systems and standardized criteria remain unavailable. Given the complex tumor-immune interplay, future research holds promise for developing peripheral blood-derived biomarker systems through computational modeling or large cohort studies to predict patient responses to PD-1/PD-L1 blockade accurately.

PO_530

Visualizing the Role of Interferon in the Immune Microenvironment: A Bibliometric Analysis (2011–2025)

Cheng Xiaohua[1] Li Fuxia[2*]
1.Shihezi University
2.Department of Gynecology, First Affiliated Hospital, Shihezi University

Over the past few years, the biological properties of interferons have garnered growing interest owing to their pivotal function within the immunological milieu, which has also driven escalating scrutiny and expanded research in related aspects. This paper explores scientific advancements regarding the immunomodulatory effects of interferon on the immune microenvironment from 2011 to 2025 through bibliometric analysis. The study utilizes data extracted from the Web of Science database, with search query parameters being 'interferon' and 'immune microenvironment', yielding a cohort of 865 publications for analysis. The findings indicate a consistent rise in the annual publication output within this domain, reflective of the sustained augmentation of academic focus. China and the United States excel in this field, consistently ranking among the top countries for both publication output and citation frequency. Keyword analysis has revealed research hotspots such as interferon-γ, tumor immune microenvironment, and immunotherapy, and emerging directions like immune checkpoint inhibitors, DNA damage, and immune infiltration have also received more and more attention. This study further demonstrates that interferon exhibits a dual regulatory role within the tumor immune microenvironment (TIME), modulating anti-tumor immune responses through multiple distinct mechanisms. Although this study provides a comprehensive analysis for this field, it also has limitations,

such as a single data source and the complexity of keyword analysis. Future research can further explore the interaction mechanism between interferon and the immune microenvironment, so as to provide new ideas for the development of precision medicine.

PO_531

HER2-Targeted Molecular Probes for Cancer Imaging: Construction, Optimization, and Clinical Applications

Xinyu Guo
West China Second University Hospital, Sichuan University

Background The expression status of human epidermal growth factor receptor 2 (HER2) is a critical biomarker for guiding precision therapy in HER2-positive cancers. Conventional diagnostic approaches, however, are invasive and fail to resolve the spatiotemporal heterogeneity of tumors. Recent breakthroughs in molecular imaging technologies offer a novel paradigm to address these challenges. By engineering highly specific HER2-targeted molecular probes, it is possible to noninvasively and quantitatively delineate HER2 expression profiles at the whole-body level.
Method A systematic literature search was conducted using Web of Science, Embase, ScienceDirect, Ovid, and PubMed (search terms: “HER2”, “molecular probe”, “cancer imaging”). Studies published between 2018 and 2024 focusing on preclinical design or clinical translation of HER2-targeted imaging probes were included.
Result We review recent progress in the design and optimization of HER2-targeted molecular probes and their emerging clinical applications, providing a framework to accelerate their translation and support the advancement of precision oncology.
Conclusion The advancement of molecular imaging holds substantial clinical significance for early molecular subtyping, metastatic lesion screening, and dynamic monitoring of targeted therapies. Despite the promising translational potential confirmed by these probes, suboptimal tissue penetration and nonspecific uptake remain major bottlenecks that hinder imaging performance and clinical efficacy. Rational optimization strategies tailored to the demands of molecular imaging have emerged as a key driver of improved diagnostic efficiency.

PO_532

肠道微生物群与子宫内膜异位症的因果关联：孟德尔随机化研究

张娜 樊杨* 丁浩洋 张娇 赵亚茹 黑晓慧
宁夏回族自治区人民医院

目的 既往的研究提示了肠道微生物与子宫内膜异位症之间的联系，但其因果关系尚未阐明。**方法** 从 FinnGen 联盟数据库中获取子宫内膜异位症相关 GWAS 数据（1,435 病例和和 68,969 名对照），并从 MiBioGen 数据库中获取 211 个肠道微生物群的遗传变异数据的工具变量。使用孟德尔随机化研究二者之间的因果关系，其中逆方差加权（IVW）是分析因果关系的主要方法，同时结果通过灵敏分析进行了验证。
结果 本研究发现：马文菌属、阿尔法变形菌纲、瘤胃球菌属、普氏菌科和肠杆菌属这五种肠道微生物可能导致了子宫内膜异位症的发生，但萨特氏菌则对子宫内膜异位症可能具有保护效应。
结论 本研究证实一些肠道菌群与子宫内膜异位症之间存在潜在的因果关系，为后续诊断和治疗疾病提供了新的方向。

PO_533

妇科肿瘤研究进展综述

杨喜堂
宁夏医科大学总医院

一、妇科肿瘤的病因与发病机制

妇科肿瘤的病因复杂，涉及遗传、环境、免疫、代谢等多方面因素。研究表明，STAT3 信号通路在妇科肿瘤的发生发展中起着重要作用。STAT3 作为一种转录因子，能够调控多种与肿瘤相关的基因表达，如细胞增殖、凋亡和转移等 1。p27 通过调控细胞周期的进程，抑制肿瘤细胞的增殖，其功能的异常可能导致妇科肿瘤的发生 2。

Ki-67 作为一种细胞增殖标志物，在妇科肿瘤的诊断和预后评估中具有重要意义 3。Ki-67 的表达水平与肿瘤的恶性程度、增殖活性及预后密切相关。此外，环状 RNA（circRNA）作为一种非编码 RNA，在妇科肿瘤中也表现出重要的调控作用 4。

二、妇科肿瘤的病理特征

妇科肿瘤的病理特征主要包括肿瘤的组织学类型、分化程度、浸润深度、淋巴结转移等 5。例如，子宫内膜腺体增生被认为是子宫浆液性乳头状癌的前体病变 6。此外，MALAT1 基因作为一种长链非编码 RNA，在妇科肿瘤中也表现出重要的调控作用。MALAT1 通过调控基因表达，影响肿瘤的侵袭和转移 7。

三、妇科肿瘤的诊断方法

1.传统诊断方法

2、 实验室检查

3.人工智能与新技术

近年来，人工智能（AI）在妇科肿瘤诊断中的应用日益广泛 8。AI 技术可以辅助医生进行图像识别、病理分析和预后预测，提高诊断的准确性和效率。AI 可以用于宫颈癌筛查中的细胞学图像分析，减少假阴性率 9,10。

4.综合诊断策略

妇科肿瘤的诊断通常需要多方法联合应用，以提高诊断的准确性和全面性。例如，对于宫颈癌，通常采用 Pap 涂片、HPV 检测、宫颈镜检查和活检相结合的方法；对于卵巢癌，则可能需要结合超声、CA-125 检测、MRI 和 CT 等手段 11,12。

四、妇科肿瘤的治疗进展

1.手术治疗

微创手术技术在妇科肿瘤治疗中的应用日益广泛，如腹腔镜手术和机器人辅助手术，不仅提高了手术精度，还减少了术后并发症 13。

2.放疗与化疗

3.免疫治疗

4.靶向治疗

RNAi 技术作为一种新型基因沉默工具，已在妇科肿瘤的基础研究中广泛应用，其在基因治疗和药物开发中的潜力巨大 14。此外，针对特定分子靶点的药物，如抗卵巢上皮癌单克隆抗体 COC166-9，为卵巢癌的免疫治疗提供了新方向此外，RNAi 技术作为一种基因干扰技术，已在妇科肿瘤的研究中显示出良好的应用前景。RNAi 通过阻断特定基因的表达，抑制肿瘤细胞的增殖和转移 15。

五、妇科肿瘤的预防策略

妇科肿瘤的预防策略主要包括一级预防、二级预防和三级预防 16。一级预防即针对病因的预防，如避免接触致癌因素、改善生活方式等 17。二级预防即通过早期诊断和治疗，阻止疾病进展。三级预防即通过规范治疗和康复，提高患者的生活质量和生存率 7,8。

在妇科肿瘤的预防中，自噬相关基因（如 ATG16L1、ULK1）的表达水平与强直性脊柱炎的易感性密切相关 20。此外，PPARγ 及其配体在妇科肿瘤中的作用也受到广泛关注 21。

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妇科肿瘤研究进展：从精准治疗到免疫突破的全景解析

杨喜堂
宁夏医科大学总医院

妇科肿瘤是威胁女性健康的重大疾病，2020 年全球新增病例约 133 万例，死亡 54 万例，以宫颈癌、卵巢癌和子宫内膜癌为主。近年来，分子靶向治疗、免疫疗法及 AI 诊断技术的发展，推动其诊疗向精准化转型。本文梳理 2020 年以来的关键进展，聚焦中外研究突破。

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妇科肿瘤研究进展综 述

杨喜堂
宁夏医科大学总医院

妇科肿瘤是女性生殖系统常见的恶性肿瘤，其病因、发病机制、病理、诊断、治疗和预防等方面的研究一直是医学界关注的重点。近年来，随着分子生物学、免疫学和转化医学的发展，妇科肿瘤的基础研究取得了显著进展，为临床治疗提供了新的思路和方法。

PO_536

METTL13 的结构特性、调控网络及其在肿瘤发生中的修饰机制研究进展

何洁洁 李妍*
青海大学附属医院

广泛存在于细胞质、线粒体和细胞核中由 METTL13 编码的 FEAT（Faint expression in normal tissues ,aberrant overexpression in tumors）蛋白在正常人体器官组织中不表达或微弱表达。正常睾丸组织中 METTL13 过低表达会使雄性小鼠睾丸下降功能受损，形成隐睾症。在肝癌，胃癌等多种癌症中呈现出 METTL13 过表达。作为肿瘤驱动子的 METTL13 具有免疫原性，且可在癌症患者的血液中检测到。因此，探究 METTL13 表达与疾病之间的关系，确定 METTL13 的角色是重中之重。现有研究表明，METTL13 参与多条通路及分子的调节，促进疾病的发生发展。但 miR-16 结合 METTL13 mRNA 抑制其表达。具有甲基酶结构域的 METTL13 甲基化 eEF1A 的 N 端和 eEF1A 中第 55 位的特定赖氨酸（Lys55），这一活动具有高度特异性。赖氨酸甲基化在体内广泛存在，且参与体内多种活动。本综述将对 METTL13 及与之相关的通路及分子做一个相对完整的阐述。

PO_537

妇科肿瘤的化疗相关认知障碍的研究现状及诊疗进展

王雨濛[1] 夏百荣[1,2*]
1.蚌埠医科大学研究生院
2.中国科学技术大学附属第一医院

目的 妇科肿瘤患者化疗相关认知障碍（CRCI）是降低生存质量的核心并发症，涉及化疗神经毒性、雌激素剥夺及心理社会因素的多维交互作用。本文旨在系统整合 CRCI 的发病机制、评估策略及干预方案，重点解析妇科肿瘤背景下雌激素骤降对神经保护的削弱效应，为精准临床管理提供循证依据。
方法 通过检索 PubMed、Web of Science 及 CNKI 数据库（2010-2025 年），采用系统评价方法分析以下维度：(1)生物学机制：聚焦铂类/紫杉烷类药物诱导的氧化应激、神经炎症及血脑屏障破坏，结合手术或化疗性闭经导致的雌激素水平骤降对海马 ERβ-BDNF 通路的抑制；(2)评估体系：整合神经心理学量表、多模态影像及血清标志物；(3)干预措施：评价药物与非药物治疗的疗效及妇科适用性。
结果 铂类/紫杉烷类通过诱发线粒体功能障碍及小胶质细胞活化损伤海马-前额叶网络；雌激素剥夺会使上述损伤加重。联合血清 AMH<0.5ng/mL 与 DTI-FA 值可以预测 CRCI 风险；而药物塞来昔布、西替利嗪或联合经皮雌二醇可以改善 CRCI 的症状，非药物治疗如同步化疗期运动上调 IGF-1；计算机认知训练增强前额叶-海马连接强度，对紫杉烷类患者的注意力提升效果显著。
结论 妇科肿瘤 CRCI 是化疗直接神经毒性、神经炎症级联与雌激素剥夺协同作用的结果，需采用“机制靶向-多维评估-分层干预”策略。基于 AMH 水平、遗传风险及化疗神经毒性特征的个体化方案可显著改善认知预后。未来研究应开发生物标志物指导的预防性干预，并探索外泌体递送雌激素受体调节剂等创新治疗。

PO_538

近距离放疗在妇科恶性肿瘤中的研究进展

杨芹 穆允凤*
陕西省肿瘤医院

近距离放射治疗（brachytherapy, BT）是一种局部放射治疗方式，其核心在于将放射源直接放置于肿瘤组织内部或其邻近位置，从而对局部肿瘤进行高剂量照射，同时有效保护周围正常组织。该技术历经百余年发展，已在妇科恶性肿瘤，尤其是宫颈癌的治疗中发挥着关键作用。随着医疗技术的不断革新，诸如高剂量率放射源、三维图像引导技术、3D 打印技术及人工智能技术等的应用，显著提升了肿瘤照射的剂量精度，有效提高了肿瘤局部控制率和患者生存率，从而进一步巩固了近距离放疗在妇科恶性肿瘤治疗领域中不可替代的地位。本文旨在对近年来近距离放射治疗在妇科恶性肿瘤领域的临床应用及研究热点进行综述，以供临床工作参考。

PO_539

免疫检查点抑制剂联合靶向乳酸-乳酸化通路在妇科肿瘤治疗中的研究进展

王丁杰[1,2] 孙蓬明[1,2*]
1.福建医科大学 2.福建省妇幼保健院

妇科肿瘤免疫治疗受限于肿瘤免疫微环境（TME）抑制特性，临床响应率仅 15%-20%。本综述揭示肿瘤代谢重编程产物乳酸及其介导的乳酸化修饰（Lactylation，Kla）通过表观遗传调控参与免疫逃逸的关键机制，为联合治疗提供新靶点。重点阐述乳酸化在宫颈癌、卵巢癌中的调控作用及临床转化路径。肿瘤微环境中乳酸积累通过组蛋白与非组蛋白修饰重塑免疫应答。在宫颈癌中，L-乳酸激活 HIF-1α 并促进 DCBLD1 蛋白 K172 位点乳酸化，通过抑制泛素化途径稳定该癌蛋白表达（临床研究显示晚期宫颈癌 DCBLD1 表达升高 2.3 倍），加速疾病进展。同时，H3K18 乳酸化上调 CD39/CD73 及 CCR8 通路，破坏 Treg/Th17 平衡形成免疫抑制微环境。卵巢癌模型研究证实，乳酸脱氢酶（LDH）抑制剂可降低 Treg 比例 35%并增强 PD-1 抗体疗效。METTL3 蛋白的乳酸化修饰通过 m6A-JAK/STAT 通路促进免疫抑制因子表达，靶向该通路可逆转卵巢癌细胞紫杉醇耐药。
针对乳酸化通路的干预显著提升免疫治疗效果：1）STAT5 介导的糖酵解促进 PD-L1 启动子组蛋白乳酸化，阻断该通路在血液肿瘤模型使 $CD8^+$T 细胞活化率提升 40%（该机制与铂耐药卵巢癌具高度同源性）；2）PERK 抑制剂通过调节内质网应激降低巨噬细胞 IL-10 表达，恢复 T 细胞毒性；3）MCT1 抑制剂 AZD3965 联合抗 PD-1 治疗降低 TME 乳酸浓度 60%，使 $CD8^+$T 细胞浸润增加 2.1 倍，妇科肿瘤模型体积缩小 40%。目前 5 项靶向乳酸化的妇科肿瘤临床试验正在进行，包括 STAT3 抑制剂（NCT04849910）和 MCT1 阻断剂（NCT05268614）等。
本综述表明，靶向乳酸-乳酸化通路可有效逆转妇科肿瘤免疫抑制。未来研究方向应聚焦：建立宫颈癌类器官模型验证 DCBLD1 乳酸化诊断价值，探索丁酸钠与 ICIs 协同治疗方案，开发卵巢癌 PDX 模型评估代谢干预对铂类耐药的逆转效能。通过多组学技术解析乳酸化修饰网络，将推动个体化治疗策略的临床转化。

PO_540

放疗期妇科肿瘤患者全程闭环式随访系统的开发及可行性研究

熊秀
重庆大学附属肿瘤医院

目的 构建放疗期妇科肿瘤患者全程闭环式随访系统，并评价其应用效果。
方法 基于患者实际需求，构建放疗期妇科肿瘤患者全程闭环式随访系统，包括健康档案、随访计划、风险识别与异常提醒、医护患在线咨询与交互、数据可视化分析及统计智能化管理 5 个模块。采用便利抽样法，选取 2024 年 1 月—6 月在重庆市某三级甲等医院妇科就诊的 166 例放疗期妇科肿瘤患者作为试验组，应用全程闭环式随访系统进行随访；将 2024 年 7 月—12 月就诊的 167 例放疗期妇科肿瘤患者作为对照组，实施常规随访护理。系统应用前后对两组自我管理能力、生活质量及随访服务满意度指标进行比较。
结果 试验组 164 例、对照组 165 例完成研究。系统应用后，试验组癌症患者自我管理测评量表得分为（169.45±11.97)分，高于对照组的（150.77±13.45)分；癌症患者生命质量测定量表得分为（65.51±7.91)分，高于对照组的（59.82±7.28)分；随访服务满意度调查问卷得分为（52.01±3.31)分，高于对照组的（48.88±3.55)分，两组比较，差异均具有统计学意义（P<0.001)。

结论 全程闭环式随访系统的应用满足了放疗期妇科肿瘤患者多样化、多层次的随访需求，实现了精准化、专业化、智能化的护理随访服务，有效提升了患者的症状自我管理能力和生活质量，提高了对护理服务的满意度。

PO_541

3D 生物打印技术在妇科肿瘤研究领域的应用现状与展望

郝宇杰 江晓霞 凡蝶 李政*
云南省肿瘤医院(昆明医科大学第三附属医院)

妇科恶性肿瘤严重威胁着我国女性的身心健康，而癌症相关靶向治疗和免疫治疗等疗法迭代的实质是分子生物学研究的临床转化。传统细胞培养、动物模型、类器官等临床前模型受到诸多因素限制，无法较好地还原临床情况。而 3D 生物打印技术作为新兴的生物制造平台，其构建的高分辨率三维结构不仅具备复杂的结构和多细胞特性以贴近真实器官的形态与功能来还原体内生理状态，同时其快速的模型构建能力和可重复性能大幅缩短实验周期，近年来逐渐被用于妇科肿瘤的研究，在构建妇科肿瘤组织模型、还原肿瘤微环境、模拟肿瘤转移进程、探究肿瘤发生发展机制、高通量筛选药物、缓释免疫治疗、术后修复和癌症预防等方面均取得了可观的进展，为妇科肿瘤创新疗法的创制提供了新的研究平台。本文系统综述了 3D 生物打印技术在妇科肿瘤研究领域的应用现状，并对该技术未来对促进妇科肿瘤个性化和精准化诊疗的潜力进行了展望。

PO_542

妇科肿瘤的多维度研究进展：从病因机制到临床治疗与预防

杨喜堂
宁夏医科大学 宁夏医科大学总医院妇科

妇科肿瘤是女性生殖系统常见的恶性肿瘤，其病因、发病机制、病理、诊断、治疗和预防等方面的研究一直是医学界关注的重点。近年来，随着分子生物学、免疫学和转化医学的发展，妇科肿瘤的基础研究取得了显著进展，为临床治疗提供了新的思路和方法。

PO_543

妇科肿瘤相关研究进展

杨喜堂
宁夏医科大学 宁夏医科大学总医院妇科

妇科肿瘤严重威胁女性健康，其诊疗研究一直是医学领域重点。本文聚焦近年来妇科肿瘤在早期诊断、治疗技术、前沿研究应用等方面进展，阐述多维度早期诊断技术革新与临床应用，手术、靶向、免疫治疗等治疗技术突破，以及 3D 细胞模型、基因编辑等前沿技术探索，旨在为妇科肿瘤诊疗提供全面参考，助力临床实践与未来研究发展。

PO_544

妇科肿瘤的相关研究进展

杨喜堂
宁夏医科大学 宁夏医科大学总医院

妇科肿瘤严重威胁女性健康，其诊疗研究一直是医学领域重点。本文聚焦近年来妇科肿瘤在早期诊断、治疗技术、前沿研究应用等方面进展，阐述多维度早期诊断技术革新与临床应用，手术、靶向、免疫治疗等治疗技术突破，以及 3D 细胞模型、基因编辑等前沿技术探索，旨在为妇科肿瘤诊疗提供全面参考，助力临床实践与未来研究发展。

PO_545

血清蛋白质组与代谢组联合分析发现卵巢子宫内膜异位症潜在诊断生物标志物

金平*
深圳市妇幼保健院

卵巢子宫内膜异位症（ovarian endometriosis，OEM）是育龄女性中常见的妇科疾病，常表现为慢性盆腔疼痛、不孕等症状，严重影响患者的生活质量与生育能力。目前临床诊断主要依赖腹腔镜手术及组织病理学检查，具有侵入性强、费用高、确诊时间延迟等问题。因此，迫切需要开发可靠的非侵入性诊断方法，以实现疾病的早期识别与干预。

目的 本研究旨在通过蛋白质组学与代谢组学的联合分析，系统识别 OEM 患者血清中的分子特征，筛选出具有临床转化潜力的非侵入性诊断生物标志物。

方法 研究纳入 6 例经腹腔镜及病理确诊的 OEM 患者和 6 名年龄匹配的健康女性作为对照组。血清蛋白质组采用数据独立采集（DIA）结合液相色谱-质谱联用技术（LC-MS）进行分析；代谢组采用超高效液相色谱-串联质谱技术（UPLC-MS/MS）进行检测。进一步结合随机森林和逻辑回归等多种机器学习算法构建诊断模型，从中筛选出区分 OEM 患者与健康对照的关键分子。

结果 蛋白质组分析共检测到 36,875 条肽段，定量获得 3,054 种蛋白质，其中 658 种蛋白在 OEM 患者中表达显著异常。代谢组分析检测到 10,844 个正离子和 8,251 个负离子，鉴定出 889 种上调代谢物与 923 种下调代谢物。多组学联合分析揭示蛋白质与代谢物间存在显著相关性，富集于 25 条代谢通路，尤其包括脂质代谢、核苷酸代谢与氨基酸代谢等 12 条与炎症、氧化应激和细胞增殖密切相关的通路。

结果 本研究通过整合蛋白质组与代谢组数据，全面描绘了 OEM 患者血清中的分子改变，并发现了一批具有潜在临床价值的非侵入性诊断生物标志物。这些结果为 OEM 的早期筛查与个体化治疗策略提供了新的科学依据，也为未来血清标志物的开发奠定了理论基础。

PO_546

腹腔镜与开腹巨大子宫切除术的疗效及安全性对比分析

郑果
郑州大学第一附属医院

目的 比较腹腔镜子宫切除术（包括常规腹腔镜和机器人辅助腹腔镜）与开腹手术在巨大子宫（子宫大小≥20 周孕期）患者中的围手术期效果和并发症表现。

方法 本研究为回顾性队列研究，纳入 2013 至 2022 年期间在郑州大学附属医院接受子宫切除术的 20 周或以上子宫大小的患者。排除标准为术前诊断为子宫恶性肿瘤或疑似恶性肿瘤的患者。根据主要手术策略的选择，患者被分为腹腔镜组和开腹组。采用 1:1 倾向评分匹配（PSM）分析，确保基线特征的平衡。收集并比较术中和术后的各项指标。
结果 共纳入 292 例患者，其中腹腔镜组 170 例，开腹组 122 例。通过倾向评分匹配，成功匹配两组各 98 名患者。匹配后，腹腔镜组的估计失血量显著低于开腹组（50（10-600）vs 100（50-200）ml，P<0.001），术后排气时间更短（2.0±0.6 vs 2.5±0.7 天，P＜0.001），住院时间也更短（4（3-30）vs 6（4-22）天，P<0.001）。然而，腹腔镜组手术时间较长（143.5（49-360）vs 111.0（53-330）分钟，P＜0.001），且住院费用较高（33702.5±11022.5 vs 30226.3±7619.2 元，P=0.011）。同时，腹腔镜组围手术期并发症发生率较低（0（0/98）vs 8.2%（8/98），P=0.007），主要术后并发症发生率也较低（0（0/96）vs 8.2%（6/96），P=0.007）。
结论 对于子宫大小≥20 周孕期的患者，腹腔镜子宫切除术是一种可行的手术选择，具有较好的手术效果和术后恢复，较开腹手术具有明显优势。

PO_547

Prevalence of vaginal microecological disorder and their association with HPV infections in pregnant women attending prenatal examinations

Wang Liying
Fujian Maternity and Child Health Hospital College of Clinical Medicine for Obstetric & Gynecology and Pediatrics

Background Vaginal infectious and human papillomavirus (HPV) infections are common among women of reproductive age, potentially contributing to infertility and adverse pregnancy outcomes.. This study aims to investigate the prevalence of vaginal microecological disorder and their association with HPV infections in pregnant women attending prenatal examinations.
Methods A cross-sectional study was conducted involving 630 pregnant women who received prenatal care at Fujian Maternity and Child Health Hospital from 2023 to 2024. All participants underwent the Aptima HPV E6/E7 mRNA assay and vaginal microecological examinations. Finally, clinical factors, virological data, and vaginal microecological changes were analyzed.
Results Among the 630 participants, 241 (38.3%) had abnormal vaginal microecology, with vulvovaginal candidiasis (VVC, 27.0%) being the most prevalent. The HPV infection rate was 28.6% in this cohort of pregnant women undergoing prenatal care. Among individuals with positive HPV E6/E7 mRNA, the BV infection rate (χ^2 = 6.298, P=0.012) , and leukocyte esterase activity (χ^2 = 4.990, P=0.026) were significantly elevated. Logistic regression analysis identified abnormal vaginal pH (OR = 1.53, 95% CI = 1.04–2.26, P = 0.031) and BV Nugent score≥7 (OR = 2.97, 95% CI = 1.23–5.15, P = 0.015) as risk factors for HR-HPV E6/E7 mRNA positivity in pregnant women.
Conclusion This study shows a high prevalence of BV, VVC or HPV positivity in pregnant women. Moreover, vaginal microecological disorders, especially BV, are risk factors for HPV infection. To better manage HPV infected population, more attention should be paid to the prevention and proper treatment of BV.

PO_548

高危型人乳头瘤病毒感染与绝经后妇女阴道微生态相关研究进展

丁浩洋 樊杨* 张娜 张巧瑞 席香妮
宁夏回族自治区人民医院

女性绝经后，卵巢功能衰退，阴道微生态发生改变，机体感染 HPV 的概率增大。 基于该临床现状,为助力绝经后宫颈癌(cervical cancer)防治,现将绝经后阴道微生态与女性 HR-HPV 感染相关研究进展做一综述。

PO_549

Trends in delayed childbearing, its correlates, and associations with pregnancy outcomes: Findings from NHANES 1999–2020

Yi-xin Li Wen Lu*
Shanghai First Maternity and Infants Hospital

Objective This study aimed to investigate long-term trends and sociodemographic correlates of age at first birth (AFB) in the United States, and to assess the associations between AFB and adverse pregnancy outcomes, with a particular focus on disparities across racial/ethnic and socioeconomic groups.
Methods Using data from ten cycles of the National Health and Nutrition Examination Survey (NHANES) spanning 1999 to March 2020, we analyzed a nationally representative sample of 19,620 U.S. women aged 15–59 who provided self-reported age at first live birth. Maternal delivery periods were categorized into seven 10-year intervals (pre-1960 to 2010–2019). We examined secular trends in AFB and the prevalence of advanced maternal age (AMA, ≥35 years), and evaluated associations between AFB and sociodemographic factors, including race/ethnicity, education, income, BMI, smoking, and alcohol use. Associations between AFB and adverse pregnancy outcomes—gestational diabetes mellitus (GDM), low birthweight (LBW), high birthweight (HBW), and pregnancy loss—were analyzed using survey-weighted logistic regression models.
Results AFB increased from 21.15 years in the pre-1960 period to 25.73 years in 2010–2019, with variation across racial/ethnic groups. The most pronounced increases were observed among non-Hispanic White and "Other" racial groups, while Hispanic women exhibited the slowest increase. AMA prevalence rose from 0.5% to 9.1% during the same period. Sociodemographic disparities were evident: Black and Hispanic women had younger AFB than White women even after adjustment, while lower educational attainment and poverty were strongly associated with earlier first births. Smoking and obesity were also linked to younger AFB. Conversely, higher income, education, and non-smoking status were associated with delayed childbearing. Regarding pregnancy outcomes, higher AFB was independently associated with increased odds of GDM (adjusted OR per year increase = 1.066; 95% CI: 1.043–1.088) and pregnancy loss (OR = 1.016; 95% CI: 1.004–1.028). However, the inverse associations with LBW and HBW observed in unadjusted models were no longer significant after adjustment.
Conclusion This study demonstrates a steady upward trend in AFB and AMA in the U.S. over the past century, with substantial racial, socioeconomic, and behavioral disparities. Delayed childbearing was associated with an increased risk of GDM and pregnancy loss, underscoring the need for targeted maternal health interventions. These findings highlight the complex interplay between social context and reproductive behavior, with implications for both public health planning and individualized clinical care.

PO_550

女性生殖道血管周上皮样细胞肿瘤案例分析

刘唯庆 王冬*
重庆大学附属肿瘤医院

研究目的 女性生殖道血管周上皮样肿瘤（PEComa）在临床上较为少见，缺乏特异性的体征或症状，多依据病理检查而确诊，对其诊治策略尚缺乏一致性共识，本研究通过对我中心女性生殖道 PEComa 的病例进行讨论，以期为此类患者的临床诊治提供数据支持。
材料与方法 收集 2014 年 3 月-2025 年 7 月在重庆大学附属肿瘤医院接受治疗的 6 例女性生殖道 PEComa 患者的临床资料，回顾性分析其临床病理及基因特点、既往治疗及预后。
结果 （1）临床病理特点：6 例女性生殖道 PEComa 患者，平均发病年龄为 41 岁（24-60 岁）；原发部位：4 例为子宫，1 例为外阴，1 例为宫颈。（2）既往治疗：1）手术：6 例患者均接受手术治疗，盆腹腔手术 8 例次，外阴手术 2 例次，肺部手术 1 例次，其中 4 例患者均切除子宫，1 例患者行子宫肌瘤切除术，1 例患者行外阴肿物切除术+右侧腹股沟淋巴结清扫术。2）放疗：6 例患者中，3 例接受了放疗，其中 2 例患者均为初始手术后行辅助放疗（1 例为 31F，1 例为 25F），另 1 例患者为复发后针对肺、骨、肝脏的转移病灶进行放疗共 31F。3）化疗：6 例患者中，有 3 例患者行化疗治疗，其中 2 例患者行腹腔灌注化疗，其余化疗均为静脉输注，平均接受了 4 个疗程（2-6 个疗程）的化疗。4）靶向治疗：其中 1 例患者接受了口服甲磺酸阿帕替尼片的治疗。5）免疫治疗：其中 1 例患者参加临床研究接受了 PD-1 抗体 F520 的治疗。（3）基因检测：3 例患者行基因检测，均提示 TSC2 突变。（4）预后：4 例 PEComa 患者随访 1-60 个月无复发；2 例 PEComa 患者在接受手术、放化疗等治疗后出现病情进展而死亡，平均总生存时间 19 个月（14-24 个月）。
结论 女性生殖道 PEComa 以手术治疗为主，若发生肺、骨、肝脏等远处转移，预后较差，免疫或靶向治疗可能是复发患者治疗的新选择，本研究案例数据有限，尚需更多临床数据支持，以提高此类患者的预后。

PO_551

子宫切除术与子宫肌瘤剔除术治疗 4 个及以上多发性子宫肌瘤的疗效及生活质量的前瞻性研究

姜潇薇
郑州大学医学院

目的 对比分析子宫切除术与子宫肌瘤剔除术治疗多发性子宫肌瘤（肌瘤数目≥4 个）患者的临床疗效及生活质量，为患者的治疗选择提供依据。
方法 前瞻性记录 2023 年 8 月-2024 年 6 月因多发性子宫肌瘤（肌瘤数目≥4 个）在郑州大学第一附属医院行手术治疗的患者，根据手术方式分为子宫切除术组和子宫肌瘤剔除术组，分析两组基线资料。采用倾向性评分匹配（Propensity score matching， PSM）按照 1:1 匹配后，比较两组患者围术期资料、生活质量及卵巢功能的差异。
结果 最终纳入 495 例患者，其中，子宫切除术组 256 例，子宫肌瘤剔除术组 239 例。子宫切除术组中 40 岁及以上、BMI≥25 kg/m2、孕产次多、无生育需求、存在症状、贫血、术前输血、合并腺肌瘤或腺肌病、合并高血压/糖尿病、肌瘤数目＞10 个的患者占比较高，差异均有统计学意义（P 均<0.05）。经 PSM 后，两组各有患者 95 例。子宫切除术组较子宫肌瘤剔除术组患者子宫体积大[（291.10±218.79）cm3 vs （217.19±166.84）cm3, P <0.05]，且差异具有统计学意义。相较于子宫肌瘤剔除术组，子宫切除术组术中出血量少[（40.11±15.89）ml vs （106.11±43.86）ml]、手术时间短[（109.42±44.12）min vs （135.85±60.42）min]、术后排气时间早[1（1，1）d vs 1（1，2）d]、留

置引流管比例少（6.4% vs 26.3%）、术后使用镇痛药比例低（15.8% vs 33.7%）、术后拔除引流管时间短[2.5（2，3）d vs 3（2，3）d]、术后住院时间短[4（3，5）d vs 4（4，5）d]、症状缓解率高（96.7% vs 77%），且差异均有统计学意义（P 均＜0.05）。两组间 SF-36、FSFI、SAS、SDS 评分分别在术前、术后 3 个月、术后 6 个月对比差异均无统计学意义（P 均＞0.05）。与术前相比，各组的 SF-36 在术后 3 个月均有显著下降，在术后 6 个月均有显著升高（P 均＜0.05）。子宫切除术组的 Kuppermann 评分分别在术前、术后 3 个月、术后 6 个月均高于子宫肌瘤剔除术组（P 均＜0.05），然而，术前术后变化值在两组间差异均无统计学意义（P 均＞0.05）。术前、术后子宫切除术组 AMH 均低于子宫肌瘤剔除术组，差异有统计学意义（P 均＜0.05）。各组术后 AMH 较术前变化均无统计学意义 （P 均>0.05）。

结论 子宫切除术治疗 4 个及以上的多发性子宫肌瘤的疗效及优于子宫肌瘤剔除术，且对术后短期生活质量与卵巢功能无影响。

PO_552

Hospital-Based Analysis of Delayed Diagnosis of Iatrogenic Urological Injuries Following Hysterectomy

Jimiao Huang Hang Lin Yiling Zhuang Huan Yi Xiangqin Zheng*
College of Clinical Medicine for Obstetrics & Gynecology and Pediatrics, Fujian Medical University, Fuzhou, 350001, Fujian, China.

Background Iatrogenic urinary tract injury is a rare but serious complication following hysterectomy. This study examined its clinical characteristics and sought early symptoms of delayed diagnosis to improve outcomes.

Methods This was a retrospective study of patients presenting with iatrogenic urinary tract injuries post-hysterectomy from January 2013 to December 2023 in Fujian Provincial Maternity and Child Health Hospital. The clinical information was extracted from the electronic clinical system to statistically describe the clinical characteristics of urinary tract injuries. We used chi-square and Student's t-tests to analyze categorical and continuous variables, respectively, to elucidate the factors contributing to delayed diagnosis and adverse outcomes. Regression analysis was used to assess the trend in the incidence of urinary tract injuries over time.

Results Among 15,560 patients underwent hysterectomy, 52 cases (0.33%) of urinary tract injuries were included. 9 (17.31%) were found intraoperatively, and 43 (82.69%) were found postoperatively. Laparoscopy (P=0.035) and pelvic adhesion (P=0.034) were the factors caused delayed diagnosis. Compared with immediate diagnosis during the operation, delayed diagnosis was more likely to be associated with urinary fistula (P<0.01), low back pain (P=0.026), postoperative hydronephrosis (P=0.022), and the secondary or higher surgery (P<0.01). Importantly, early detection of injuries during initial hospitalization in patients with delayed diagnosis correlated with a reduced number of subsequent hospital visits and a decreased risk of urinary fistula. Notably, low back pain (P=0.008) emerged as a critical symptom for early detection of delayed diagnosis.

Conclusion Delayed diagnosis of iatrogenic urinary tract injuries may be associated with increased risk of severe complications. Low back pain is a significant symptom for detecting injuries during the first hospitalization, aiding in early detection and timely management.

PO_553

Encountering a “roadblock” in cervical cerclage: a case of cervical circleage and hysteromyoma removal under laparoscopy

Mao Yiwen
Wuxi Maternity and Child Health Care Hospital

Background Cervical insufficiency is a leading cause of late pregnancy loss and preterm birth, with cervical cerclage being an effective treatment.While pregnancies involving hysteromyoma are not rare, women with such conditions are considered high-risk, and treatment for fibroids during pregnancy is usually reserved for special circumstances.
Case Summary We present the case of a 34-year-old pregnant woman with cervical insufficiency and hysteromyoma. She had undergone a loop electrosurgical excision procedure (LEEP) five years earlier due to HPV infection, resulting in cervical insufficiency. After five years of unsuccessful attempts to conceive naturally, she underwent embryo transfer. Ultrasound revealed a hypoechoic area approximately 5 cm in size on the left anterior uterine wall, suggestive of hysteromyoma, which overlapped with the planned path for cervical cerclage. A myomectomy was performed before the cerclage procedure. The patient recovered well postoperatively and is currently 21 weeks pregnant, attending regular prenatal check-ups at our hospital.

PO_554

PD-1 抑制剂对卵巢功能的影响—基于双样本孟德尔随机化和可视化实验验证

郑佳欣
大连理工大学

Objective 研究免疫检查点抑制剂特别是程序性细胞死亡蛋白 1 抑制剂对卵泡功能的影响。
Design 采用了双重方法，包括一项研究和一项孟德尔随机化研究。
Exposure(s): 该 MR 研究利用来自欧洲的不同大规模全基因组关联研究的统计数据。为了进行实验验证，将小鼠卵泡在体外用 200 nM 的 DOX、10 μg/ml 的 PD-1 抑制剂 ch15mt 或 PBS 对照培养。在培养 24 小时后，测量卵泡直径，并通过 ELISA 法测定雌二醇（E2）水平。此外，将卵泡转染 Cyto-Ca2+ FRET 探针，以监测暴露于 DOX、PD-1 抑制剂和 PBS 时的胞浆 Ca2 波动。
Result(s) MR 分析表明，遗传预测的 PD-1 对卵巢储备损伤疾病即卵巢早衰及不育和卵巢分泌的激素（AMH、E2）的风险没有影响。补充的 MR 方法和敏感性分析进一步证实了 MR 结果的可靠性。我们的基础实验表明，PD-1 抑制剂组对卵泡大小的 E2 激素分泌没有影响。同样，PD-1 抑制剂组对卵泡胞浆 Ca2+变化没有影响。
Conclusion(s) 我们的双样本 MR 分析未发现支持 PD-1 抑制剂和卵巢储备功能存在显著关联的遗传证据。PD-1 抑制剂可能是女性癌症患者保留生育功能的治疗方案中重要组成部分。

PO_555

遗传性平滑肌瘤病-肾细胞癌综合征 1 个家系报道

刘雪琰 郭瑞霞*
郑州大学第一附属医院

研究目的 遗传性平滑肌瘤病-肾癌综合征（Hereditary Leiomyomatosis and Renal Cell Cancer，HLRCC）是一种罕见的遗传性疾病，其特征为皮肤平滑肌瘤、子宫平滑肌瘤及肾细胞癌，目前国际上已报道约 300 个家系。患有 HLRCC 的女性通常子宫肌瘤发病早，数量多，生长快，严重影响生活质量及生育结局。本研究对一个 HLRCC 家系的临床和遗传特征进行详细分析，拟丰富 HLRCC 的变异谱。
材料与方法 纳入先证者因子宫多发肌瘤于郑州大学第一附属医院妇科接受手术治疗，且符合 HLRCC 临床诊断标准的家系。采集先证者及家属外周静脉血通过 sanger 测序进行 FH 基因胚系突变检测，通过 PolyPhen-2、PROVEAN、SIFT 等工具对突变位点进行致病性预测，通过 SOPMA 数据库检查突变对蛋白质二级结构的影响，MEGA 软件进行氨基酸保守性分析，PyMOL 软件构建突变序列三维结构模型。对先证者肿瘤组织进行 FH 及 2SC 免疫组化染色。**结果** 先证者 29 岁因子宫多发肌瘤于我院行子宫肌瘤切除术，其父及一弟均患有肾细胞癌。家系成员 sanger 测序成功鉴定出 FH 第 5 外显子一个新错义突变（c.716 C>T；p. Ala239Val），该突变在现有数据库 gnomAD 、HGMD、clinvar、LOVD 等数据库均无相关报道。突变位点在多个物种氨基酸序列高度保守，PolyPhen-2、PROVEAN 等工具均预测为有害变异。先证者肿瘤组织免疫组化结果 FH（表达缺失，对照阳性），2SC（+）。
结论 本研究为 FH 基因变异谱增添新的错义突变（c.716 C>T；p.Ala239Val），基于表型、生物信息预测、免疫组化等多维度分析，突显了其可能的致病性。

PO_556

Advances in the research on the impact of HIF-1α on immunosuppressive cells within the hypoxic tumor microenvironment

Luyao Kang Bilan Li*
Tongji University Affiliated Obstetrics and Gynecology Hospital

Within neoplastic tissues, a hypoxic microenvironment prevails, characterized by spatial and temporal heterogeneity. This hypoxia condition can affect the behavior of cancer cells and contribute to tumor growth. The hypoxia-inducible factor (HIF) signaling pathway is a key mechanism by which cells adapt to low oxygen levels, with HIF-1α being the most studied and best understood factor in this pathway. HIF-1α plays an important role in changing the hypoxia tumor environment (TME) by affecting both the innate and adaptive immune systems through changes in metabolism and gene activity. Because of its central role in controlling responses to hypoxia, this study focuses on how HIF-1α regulates immunosuppressive cells, aiming to offer new understanding of how HIF-1α influences tumor development by modulating immunosuppressive cells within the TME.

PO_557

基于 ERAS 理念的早期拔除尿管结合间歇导尿术在宫颈癌根治术后患者中的应用：一项随机对照研究

陈彦丽 吴治敏* 荣欣 李玲 史元湘 吴娇娇 王延洲
陆军军医大学第一附属医院

研究目的 探讨基于 ERAS 理念的早期拔除尿管结合间歇导尿术在宫颈癌根治术患者中应用的安全性和有效性。

材料与方法 选择 2022 年 9 月至 2024 年 12 月重庆某三甲医院符合纳入标准的分期为ⅠA1～ⅡA1 行改良腹腔镜下广泛性子宫切除术及盆腔淋巴结清扫术的患者 78 例，1 : 1 随机分为试验组和对照组。对照组(n=39)实施常规护理, 术后 7 天拔出导尿管，当残余尿>100mL 时,再次留置导尿，此后每周尝试拔出尿管，直至残余尿＜100 ml。试验组(n=39)，启动基于 ERAS 理念的早期拔除尿管（术后 3 天）结合间歇导尿术，术后 3 天拔出导尿管，拔除失败时,行间歇导尿。试验组和对照组各失访 1 人。主要结局指标为术后 2 周膀胱功能Ⅱ级以上恢复率，次要结局指标为术后 1 月尿路感染率、尿动力检查指标以及术后 1 周、2 周、3 周、1 月、3 月膀胱功能障碍评分（MHU）。

结果 试验组术后 2 周膀胱功能Ⅱ级以上恢复率显著高于对照组（29[74.4] vs 16[41.0] ；p=0.003)；试验组术后 1 月尿路感染发生率显著低于对照组（3[7.7]vs 10[25.6]；P=0.033）；试验组术后 1 个月尿动力学检查最大尿流率（20.79±13.51 vs 12.85±5.699；p=0.002）、平均尿流率（10.24±7.07 vs 6.03±3.67；p=0.002）显著高于对照组，残余尿量（0[0,0] vs 30[0.00,108.25]；p=0.003）显著低于对照组。试验组术后 1 周、2 周、3 周、1 月以及 3 月 MHU 总分均显著低于对照组，差异具有统计学意义(p＜0. 05)。

结论 宫颈癌根治术患者术后早期拔除尿管,并采用清洁间歇导尿策略有助于促进术后早期膀胱功能的恢复、减少膀胱功能障碍相关症状并降低尿路感染发生率, 是安全有效的。

PO_558

绝经后无症状子宫内膜增厚的临床意义

王政 李明霞 闫志风 吴迪 孟元光*
解放军总医院第一医学中心

背景与目的 美国妇产科医师学会（ACOG）指出，正常绝经后女性的子宫内膜在超声下应呈现为薄而均匀的线性回声，厚度≤4 mm。对于经阴道超声发现子宫内膜增厚且伴有阴道出血的绝经后女性，建议进行子宫内膜活检。然而，目前尚无明确标准来确定无症状绝经后女性何时需要进一步有创检查以明确诊断。文献综述显示，即使是健康女性，子宫内膜厚度也会随绝经年限增加而变化。此外，随着健康意识提升和经阴道超声的普及，无症状绝经后女性子宫内膜增厚的检出率升高，但子宫内膜病理阴性结果也随之增多。这凸显了需要更多临床数据来建立循证阈值，以判断何时需进一步干预。有助于避免不必要的侵入性操作、减少医疗资源浪费并缓解患者焦虑。

方法设计 我们回顾性收集了 2014 年 1 月 1 日至 2024 年 10 月 1 日期间在解放军总医院第一医学中心就诊的无症状子宫内膜增厚绝经后女性的临床资料。对基线特征、子宫内膜厚度及经阴道超声特征进行综合统计分析，构建诊断预测模型和受试者工作特征（ROC）曲线。

结果与结果

1、无症状子宫内膜增厚的绝经后女性中，超过三分之一无子宫内膜病理改变。且健康女性子宫内膜随绝经年限延长逐渐变薄。无论绝经年限如何，子宫内膜厚度＞7 mm 提示可能存在病理改变，而＞9.5 mm 则表明恶性风险较高，需积极进行有创干预。

2、通过单因素分析发现，绝经后子宫内膜增厚女性的年龄、绝经时长、身高、子宫内膜厚度及超声特征（P<0.05）与子宫内膜病理改变的风险因素存在统计学差异。多因素分析将年龄、身高、血流信号及回声模式纳入预测模型：$p=e^Z/(1+e^Z)$，Z=0.051*年龄-0.071*身高+2.368*血流信号有无+1.146*回声均匀与否+8.025。年龄越大、身高越矮者出现病理改变的概率越高。

PO_559

Genetic analysis of potential biomarkers and therapeutic targets in pyroptosis and glycolysis from endometriosis

Shuqin Dai Rui Gu ShaojieZhao Zhao*
无锡市妇幼保健院

Introduction Endometriosis, a debilitating gynecological condition characterized by the presence of endometrial-like tissue outside the uterus, remains a challenge in diagnosis and treatment due to its complex etiology and symptomatology. This study aimed to elucidate the underlying molecular mechanisms of endometriosis by exploring differentially expressed genes (DEGs) associated with glycolysis and pyroptosis, constructing a diagnostic model, analyzing immune infiltration, and predicting key gene regulatory networks and their drug interactions.
Methods We utilized three Endometriosis datasets (GSE25628, GSE7305, GSE11691) from the GEO database and obtained Glycolysis-related and Pyroptosis-related genes from GeneCards and MSigDB databases. The data were integrated and normalized prior to analyzing DEGs and constructing Glycolysis-Related and Pyroptosis-Related Differentially Expressed Genes (GR&PRDEGs). Additionally, Gene Ontology (GO), Kyoto Encyclopedia of Genes and Genomes (KEGG) enrichment analyses, Gene Set Enrichment Analysis (GSEA) using clusterProfiler, and gene set variation analysis (GSVA) were conducted to assess pathway enrichment differences across samples. A diagnostic model for endometriosis was developed using the Support Vector Machine (SVM) algorithm via Least Absolute Shrinkage and Selection Operator (LASSO) regression. Immune infiltration differences were evaluated across risk groups, and regulatory networks were built to predict protein domain structures. Differential expression of five key genes was validated in clinical endometriosis samples using basic experimental techniques. The most relevant gene was selected to investigate the roles of glycolysis and pyroptosis in endometriotic cell lines.
Results Our results identified 19 GR&PRDEGs associated with endometriosis. GO and KEGG analyses revealed involvement in multiple biological processes and metabolic pathways. GSEA highlighted significant pathways related to endometriosis biology. GSVA demonstrated marked differences in immune cell infiltration between risk groups. The logistic regression model effectively distinguished between endometriosis patients and controls. Significant correlations were found between key genes and immune cell types, implicating their potential impact on immune responses and treatment outcomes. Regulatory networks unveiled potential interactions among transcription factors (TFs), microRNAs (miRNAs), RNA-binding proteins (RBPs), and key genes alongside drug target potential. The expression of five key genes was validated in clinical samples.Focusing on LMNA, knockdown or overexpression of this gene in cell lines resulted in corresponding changes in the expression levels of glycolysis-related and pyroptosis-related genes and proteins, revealing its central role in coordinating glycolytic metabolic reprogramming and pyroptosis signaling pathways.
Conclusion This study revealed the significant roles of Glycolysis-related and Pyroptosis-related genes in endometriosis, constructed a potential diagnostic model, and provided crucial insights for a deeper understanding of the disease mechanism and exploring novel therapeutic avenues.

PO_560

子宫内膜毛母质癌 1 例报道附文献复习

叶恒溢 胡郅珺 刘开江*
上海交通大学医学院附属仁济医院

研究目的 子宫内膜毛母质癌（Pilomatrix carcinoma-like high-grade endometrial carcinoma, PiMHEC）是一种罕见且高度侵袭性的子宫内膜癌亚型，其病理特征与皮肤毛母质癌相似，以 β-catenin 核异常表达为主要特点。由于该病理类型罕见，临床对其认识不足常导致诊断困难，且相关治疗方案尚未达成共识。本文通过报道 1 例典型 PiMHEC 病例，结合文献回顾，旨在总结其临床病理特征、诊断要点及治疗策略，以提高对该病的早期识别能力和规范诊疗水平。
材料与方法 回顾性分析 1 例 47 岁女性 PiMHEC 患者的临床资料，包括病史、影像学、病理学特征及治疗过程。通过系统性文献检索（截至 2025 年），整理全球已报道的 PiMHEC 病例数据，对比分析其临床病理特点、分子特征及预后。
结果 本例患者以“月经淋漓不尽伴腹痛”就诊，影像学提示盆腹腔广泛转移（子宫、双侧卵巢及输卵管、盆腹腔淋巴结及阴道）(图 1)。病理检查显示肿瘤由高分化腺癌区与低分化实性区混合组成，免疫组化显示 β-catenin 核弥漫性强阳性，CK、EMA 阳性，ER、PR、PAX-8 阴性（图 2），符合 PiMHEC 诊断。患者接受根治性手术后 34 天因恶性肿瘤并发症伴全身多器官转移而死亡。2021 年，Weisinan 等首次报道了 5 例具有显著 β-catenin 核表达的 FIGO 3 级子宫内膜癌，且在 2023 年正式提出 PiMHEC 诊断。截至当前，全球文献共报道约 15 例。该病多见于绝经后女性（平均 57 岁），临床表现为阴道流血或腹痛，病理特征包括双向分化结构、CTNNB1 突变及 β-catenin 核表达阳性。该肿瘤侵袭性极强，多数病例在初次确诊时已转移，预后极差（中位生存期短）。现有治疗以手术为主，辅以放化疗，但疗效有限；靶向及免疫治疗可能具有潜在价值，但仍需进一步临床数据的验证。
结果 PiMHEC 是一种罕见的、高度恶性的子宫内膜癌病理类型，与其他子宫内膜癌亚型存在显著的形态学重叠，导致鉴别诊断困难，其诊断依赖于特征性病理形态及 β-catenin 核阳性表达。目前治疗以根治性手术联合辅助放化疗为主，但疾病侵袭性强、预后极差，亟需探索针对 CTNNB1 突变通路的靶向治疗策略。提高对该病的认识、早期诊断是改善预后的关键。

PO_561

Proximal-type epithelioid sarcoma of the vulva: a case report and review of the literature

tianhui he Xiaoxia Jiang Zheng Li*
Department of Gynecologic Oncology, Yunnan Cancer Hospital, The Third Affiliated Hospital of Kunming Medical University，Peking University Cancer Hospital Yunnan

Proximal-type epithelioid sarcoma (PES) of the vuvula is an extremely rare malignant soft tissue tumor. We present here a 48-year-old female patient who presented with a progressively enlarging painless mass on the right side of the vulva. A preoperative biopsy diagnosed a PES of the vulva, which was subsequently treated with wide local excision and radiotherapy. However, postoperatively, the patient developed inguinal lymph node metastasis and underwent inguinal area lymph node dissection plus radiotherapy and chemotherapy. The patient then underwent immunotherapy in combination with anlotinib for recurrence, and ultimately, the patient passed away.This study examines the management of PES of the vulva and proposes that immunotherapy and targeted therapy have shown positive efficacy in our cases. However, this study has several limitations, including a small case sample size, lack of long-term follow-up data, and insufficient validation of individualized choice of treatment regimen. In addition, the lack of randomized controlled trial support limits the broad applicability of the findings. Genetic studies are not sufficiently in-depth, and the molecular mechanisms of PES have not

been fully explored. Therefore, future studies should further increase the sample size, extend the follow-up time, and strengthen the combination of genomics and clinical trials to verify the long-term effects of current treatment options and the feasibility of individualized treatment.

PO_562

克罗米芬与来曲唑联合治疗多囊卵巢综合征型不孕症的临床疗效研究

邹俊英 邓巧 李骥 向慧珍
湖南医药学院总医院

目的 探讨克罗米芬与来曲唑联合治疗多囊卵巢综合征（PCOS）型不孕症的临床疗效，评估其在促进排卵、提高妊娠率及改善内分泌水平等方面的治疗价值。
方法 本研究纳入 2024 年 1 月至 2025 年 6 月期间就诊于我院 PCOS 患者 150 例，符合罗特丹姆诊断标准（Rotterdam Criteria），年龄 18-40 岁，婚姻正常、规律同房≥1 年、基础 FSH<12 IU/L、无其他不孕病因。将患者随机分为两组：联合治疗组（CC+Letrozole 组，n=75）及对照组（CC 组，n=75）。治疗方案均在月经周期第 3-7 天进行：联合组口服克罗米芬 50 mg/d 及来曲唑 2.5 mg/d；对照组口服克罗米芬 50 mg/d。治疗周期持续 3 个月，失败者可重复 1 次周期。监测指标包括：经阴道超声监测卵泡发育（每次随访记录优势卵泡直径、数量及成熟度）、子宫内膜厚度及血清性激素水平（FSH、LH、雌二醇、孕酮）。主要结局指标为临床妊娠率（超声确诊胎囊数/治疗周期数）；次要结局指标包括：卵泡发育率（单次周期中至少 1 个优势卵泡≥18 mm 的比例）、单卵泡发育率、内膜厚度、黄体功能（孕酮≥10 ng/mL）、早期流产率、多胎率及药物不良反应（如 OHSS、视觉障碍、潮热）。数据采用 SPSS 26.0 统计分析，计数资料行 χ^2检验，计量资料行独立样本 t 检验或重复测量方差分析，P<0.05 为差异有统计学意义。
结果 两组基线特征（年龄、BMI、基础激素水平、不孕年限）差异无统计学意义（P＞0.05）。治疗后 3 个月，临床妊娠率联合组显著高于对照组（63.0% vs 42.7%，P<0.001），其中自然周期受孕率分别为 59.5% vs 40.0%（P<0.01）。在卵泡发育指标上，联合组优势卵泡平均数量更少（1.6±0.5 vs 2.2±0.8，P<0.001），但单卵泡发育率（≥18 mm 单一优势卵泡）更高（80.2% vs 53.1%，P<0.001），内膜厚度亦更厚（9.9±1.1 mm vs 8.3±1.4 mm，P<0.001）。黄体功能方面，联合组孕酮水平升高更显著（12.5±3.0 ng/mL vs 9.8±2.7 ng/mL，P<0.001），但早期流产率无显著差异（8.0% vs 9.3%，P=0.71）。多胎妊娠率联合组较低（5.9% vs 11.9%，P=0.04），OHSS 发生率亦较低（1.3% vs 4.7%，P=0.03）。药物相关不良反应中，联合组视觉障碍发生率稍高（7.4% vs 3.8%），但无严重事件。亚组分析显示，BMI≥25 kg/m²的患者联合疗效提升更明显（P<0.01）
结论 克罗米芬联合来曲唑治疗 PCOS 型不孕症能够显著提高排卵率和妊娠率，有效改善患者内分泌紊乱及卵巢功能，且治疗安全性良好，具有较高的临床应用价值。

PO_563

Deepseek 在阴道镜诊断宫颈病变中的应用

齐金红
吉林省肿瘤医院

本研究探讨 DeepSeek 人工智能模型在阴道镜宫颈病变诊断领域的应用，通过收集已经确诊的宫颈上皮内瘤变 I、II、III 级以及宫颈癌患者的阴道镜图像、宫颈筛查数据和病例资料，整合上述信息，建立高质量数据信息集，构建联合诊断模型，并对阴道镜检查的不同阶段（原图、绿光、醋酸后、碘试验后）进行特征对比分析，标注不同级别病变图像中病变特征，对 DeepSeek 模型进行训练与优化，开

展 DeepSeek 在阴道镜诊断宫颈病变的临床试验，收集试验数据，记录模型诊断结果和病理诊断结果，进行对比分析，评估 DeepSeek 在阴道镜诊断不同程度癌前病变及宫颈癌的准确性、敏感性、特异性，优化阴道镜检查流程的，预测患病风险、提供随访意见。
方法 首先收集吉林省肿瘤医院 2020 年 1 月-2024 年 12 月期间 TCT 或 HPV 异常者、接受阴道镜检查、宫颈活检/宫颈搔刮病理异常者病例共 1180 例，建立数据集，作为 DeepSeek 训练模型。纳入标准：20-75 岁，资料完整、图像清晰、既往无宫颈手术史、宫颈活检/和宫颈管搔刮病理者。排除标准：图像模糊不清，TCT+HPV 均阴性或 TCT/HPV 阳性无病理者，宫颈癌治疗后复查者。 数据采集及预处理：采用 CopyKight V2.0 型阴道镜设备（江苏锦源医疗科技有限公司）采集宫颈图像，图像分辨率为 720×576 像素。开展 DeepSeek 在阴道镜宫颈筛查中的临床试验，要求为 TCT 或 HPV 异常、需行阴道镜下宫颈活检和/或宫颈管搔刮者，在检查过程中，由同一名妇科肿瘤医生分别留取四幅阴道镜图像（原图、绿光、醋酸后、碘试验后），按照传统方法进行诊断，同时使用基于 DeepSeek 的 AI 辅助诊断系统生成诊断结果，分析其检出率。现收集自 2025 年 3 月至 7 月符合要求的病例共 48 例。
结果 Deepseek 模型对宫颈上皮内瘤变 I、II、III 级和宫颈癌的识别检出率分别为 75%、88.23%、95%、100%，宫颈上皮内瘤变 I 级共 4 例，1 例未检出；宫颈上皮内瘤变 II 级共 17 例，2 例未检出；宫颈上皮内瘤变 III 级共 20 例，1 例未检出；宫颈癌共 7 例，全部检出。 结果 DeepSeek 在宫颈不同级别病变识别中展现出较高的敏感度和特异性，尤其是对 HSIL 和宫颈癌的识别敏感度相对较高，能够高效检测出较为严重的宫颈病变，有效辅助医生进行宫颈病变的诊断，对提升宫颈筛查效率与准确性有重要意义，为宫颈疾病的早期发现提供新的技术支持。
结论 Deepseek 在阴道镜宫颈病变诊断中具有较高的诊断效能，能够有效识别不同类型的宫颈病变，尤其在检测 HSIL 和宫颈癌方面优势明显。但因该研究开展时间短，试验组数据不足，待等多病例验证其诊断阳性率。该应用可辅助医生更快速、准确地判断宫颈病变情况，减少因医生经验不足或主观因素导致的漏诊误诊，提高宫颈筛查的准确性和可靠性。

PO_564

Transformative Applications of Humanoid Robots in Gynecologic Oncology Diagnosis and Treatment: Technological Advances and Ethical Challenges

Yirui Wang Jiahui Yu Zheng Li Yang Shen*
东南大学附属中大医院

Research Purpose This study systematically analyzes the clinical translation status of humanoid robot technology in gynecologic oncology across the monitoring-screening-surgery-rehabilitation continuum. We evaluate its technological maturity and ethical compatibility to provide interdisciplinary solutions for precision diagnosis and treatment in gynecologic oncology.
Materials and Methods Literature Screening:Conducted following the PRISMA framework.
Databases searched: PubMed, IEEE Xplore, Web of Science (2010-2024)。
Search terms: ("humanoid robot" OR "social robot") AND ("gynecologic oncology" OR "cervical cancer" OR "ovarian cancer").
Inclusion criteria: ① Original studies reporting robotic prototypes or clinical applications; ② Focused on gynecologic oncology; ③ Contained technical or clinical endpoint data (50 studies ultimately included).
Analysis Dimensions:Technical performance: Sensing accuracy, degrees of freedom and error rates in motion control, sensitivity and specificity of AI decision-making；Clinical value: Reduction in surgical complications, patient quality-of-life scores；Ethical risks: Data privacy, physician-patient trust levels, and liability determination evidence grades assessed using the GRADE system.

Results Monitoring and Screening: The electronic skin of humanoid robots enables perception of object location, orientation and hardness. Composed of flexible materials [1] and flexible sensors [2], it can detect pressure, temperature, humidity and other physical parameters, allowing real-time monitoring of physiological signals including body temperature, heart rate, blood pressure and glucose levels, thereby providing more convenient monitoring solutions for healthcare.
Surgical Treatment: Humanoid robots assist endoscopic procedures by enabling faster lesion localization and more precise tissue cutting, improving surgical accuracy [3]. They can access difficult-to-reach anatomical areas through natural orifices [4], overcoming limitations of systemic treatments via wireless technology and enabling novel minimally invasive procedures [5]. For instance, magnetic multilayer soft-bodied robots can precisely navigate, cover and adhere to gastric tissue for non-invasive targeted drug delivery, enhancing therapeutic efficacy [6].
Postoperative Rehabilitation: Humanoid robots facilitate physical and cognitive therapy through guided exercises and real-time feedback. The hybrid controller based on Modified Adaptive Neuro-Fuzzy Inference System (MANFIS) and Multi-Objective Sunflower Optimization (MOSFO) models enables high degrees of joint mobility [7]. Consequently, robot-assisted gait training improves walking ability in Parkinson's patients performing dual cognitive tasks [8], enhances recovery in spinal cord injury patients through repetitive functional movements and neuroplasticity [9], and helps assess joint attention in children with autism spectrum disorder via controlled screen stimulation and natural interaction with humanoid robots [10].
Current Limitations: Hardware and algorithmic constraints: Humanoid robots may make incorrect image-based judgments, with model limitations reducing flexibility and communication delays causing slow response times [11]. Their reliability and adaptability in complex medical environments require further validation.
Ethical challenges: Privacy and data security remain paramount concerns. Ensuring safe and ethical robotic operation in healthcare settings is crucial for their acceptance and integration. Moreover, AI and robotic technologies impact accessibility, equity and social trust, necessitating robust data privacy protections and fail-safe mechanisms to prevent patient harm.
Conclusion Humanoid robots currently serve as disease managers and advocates in healthcare [12], with core value in addressing spatial constraints and surgeon fatigue in gynecologic minimally invasive surgery [13]. However, technological capabilities and ethical challenges limit widespread adoption. Future research should focus on enhancing intelligence and autonomy to bring new vitality and efficiency to medical practice.

PO_565

Benign metastasizing leiomyoma: A case report

Ruyu Shao Desheng Yao*
Guangxi Medical University Cancer Hospital

A patient with benign metastasizing leiomyoma(BML) was admitted to Department of Gynecology Oncology, Guangxi Medical University Cancer Hospital in December 2022. A 47-year-old patient with vaginal bleeding presented herself for a gynecological examination. Masses of uterine, lungs, and many other sites throughout the body were found by magnetic resonance imaging (MRI) and positron emission tomography/computed tomography (PET/CT). Complete excision of the removable tumors and total abdominal hysterectomy with bilateral ovariosalpingectomy was performed through laparotomy under general anesthesia. Observation during operation, frozen pathology and postoperative routine pathology of the tumors suggested that the mass of the left pelvis was an epithelioid angiomyolipoma and all the other masses were leiomyoma. The purpose of this study is to investigate the clinical and imaging characteristics of BML according to this case.

PO_566

高龄女性外阴 paget1 例：病例报告及文献综述

刘雨杭 钟丽君* 丁瑾晗
西南医科大学附属中医医院

研究目的 探讨外阴 Paget 病（VPD）的临床特征、诊断方法及治疗策略，为临床诊疗提供参考。
材料与方法 回顾性分析西南医科大学附属中医医院收治的 1 例 82 岁女性 VPD 患者的临床资料，并结合相关文献进行综述。
结果 患者因 “外阴瘙痒 1 + 年，加重伴阴道瘙痒 2 + 月” 入院，曾被误诊为瘙痒症、湿疹样皮炎及外阴营养不良，经外阴组织活检及免疫组化确诊为乳房外 Paget 病（免疫组化示 CK7（+）、CEA（+）、HER-2（2+），CK20（-）、GCDFP-15（-））。完善盆腔 MRI、宫颈组织活检、乳腺彩色多普勒超声、泌尿系统彩色多普勒超声、胃肠镜等相关辅助检查排除继发可能。经多学科协作（MDT）讨论后，于 2024 年 11 月 26 日行外阴局部扩大切除术（切缘距病灶 2cm）及宫腔镜下子宫内膜息肉切除术，术后病理证实切缘阴性。患者术后出现肺栓塞及双下肢静脉血栓，经综合治疗后康复出院，术后定期复查，术后 7 月复查未见明显复发。
结论 VPD 临床表现缺乏特异性，易误诊为炎症性皮肤病，组织活检及免疫组化是诊断关键，CK7、CK20、GCDFP-15 等标志物有助于鉴别诊断。手术切除为首选方案，主要包括局部扩大切除术及莫氏显微描记手术，HER2 过表达为潜在治疗靶点，局部药物治疗、放射治疗等可作为手术替代或辅助方案。临床需提高对该病的认识，结合影像学及多学科协作优化诊疗方案，改善患者预后。

PO_567

Preliminary Clinical Study on the Feasibility of Localization and Tracing of Paraaortic Sentinel Lymph Nodes in Patients with Uterine Cancer

Fangyi Huang
Affiliated Cancer Hospital of Guangxi Medical University

Objective To evaluate the feasibility and clinical value of using indocyanine green (ICG) for sentinel lymph node (SLN) mapping of the paraaortic region in patients with Uterine Cancer under laparoscopic guidance.
Methods Twenty patients with stage IA to II endometrial cancer who underwent surgical treatment at the Affiliated Cancer Hospital of Guangxi Medical University between January 2022 and June 2025 were enrolled. Prior to surgery, ICG solution (0.125 mg/mL) was injected bilaterally into the ovarian ligaments to trace paraaortic SLNs, while methylene blue (2 mL; 20 mg) was injected at the 3- and 9-o’clock positions of the cervix to trace pelvic SLNs as a control. Under laparoscopic visualization, the first-appearing stained lymph nodes were identified as SLNs and resected separately for histopathological and immunohistochemical analysis. All patients underwent laparoscopic hysterectomy with SLN mapping of the pelvic and paraaortic regions, with optional pelvic lymphadenectomy or paraaortic lymph node dissection.
Results The overall detection rate for pelvic SLNs using methylene blue was 90%, with a left detection rate of 90% and right detection rate of 80%. For paraaortic SLNs using ICG, the overall detection rate reached 100%, with a left detection rate of 63.6% and a right detection rate of 100%.
Conclusion Paraaortic SLN mapping via injection into the ovarian ligaments (along the IPP lymphatic pathway) is feasible. This technique demonstrates a high detection rate, good accuracy, and rapid identification capability, allowing for accurate localization and prediction of paraaortic SLNs in patients

with corpus uteri tumors. It provides a reliable basis for precise lymph node evaluation in clinical practice.

PO_568

拉曼光谱在肿瘤中的应用研究

王宇歌
辽宁省肿瘤医院

恶性肿瘤，即癌症，严重威胁人体健康。全球范围内，癌症的发病率与死亡率呈持续上升态势，每年新诊断病例超过 1900 万，死亡人数达到 1000 万。我国癌症的负担尤为显著，约占全球癌症病例的 24%，癌症死亡人数超过 30%，已成为全球癌症负担最重的国家，在癌症的防治方面有紧迫需求。

现有的肿瘤检测方法很多，主要包括肿瘤标志物检验、影像学诊断、组织病理学诊断三种。其中，肿瘤标志物可从分子生物学角度进行早期肿瘤诊断与疗效监测，但受个体差异影响大，在某些良性疾病中也可能升高，肿瘤标志物的发现亦十分困难。影像学诊断是重要的辅助手段，但仅可对肿瘤的形状和性质进行初期判别，且其空间分辨率相对较低，检出微小或早期肿瘤较为困难。组织病理学可从组织和细胞尺度进行肿瘤的诊断，是诊断的“金标准”，但为有创的诊断方式，且病理诊断结果的准确性一定程度上依赖于医生的专业技术及经验，而较长的诊断时间也导致了结果的相对滞后。综上所述，我们亟待寻找新的检测技术辅助肿瘤的早发现、早诊断、早治疗。随着医学技术的发展，越来越多新的检测技术被开发并被应用于肿瘤中，以期实现诊断窗口期提前、诊断准确率提高、治疗更有效。

组织的癌变是一个十分复杂的过程，涉及生物大分子含量和结构的变化。正常组织与癌变组织、不同时期癌变组织的形态和成分构成在细胞和分子水平上都存在一定差异。拉曼光谱作为一种分子振动光谱技术，可获得分子的指纹图谱信息，鉴别各种物质的特性和结构，为癌变组织的检测提供一条可行性途径。近年来，拉曼光谱技术、拉曼光谱成像技术因其无创、快速、客观、准确、灵敏、可定性和定量检测等众多优点受到了广泛重视，已经被应用于多种肿瘤的研究、诊断、治疗方面。

本文综述了近年拉曼光谱在肿瘤中的应用研究进展，主要从肿瘤研究、肿瘤诊断、肿瘤治疗三方面展开介绍，并对今后的应用前景进行了展望。

PO_569

现代妇科精密仪器研究进展

孙雨馨 王丹波*
辽宁省肿瘤医院

随着科学研究的进步，越来越多的精密仪器设备用于妇科诊疗中，如宫腔镜、 腹腔镜、电子阴道镜、多普勒超声、leep 刀等。它们的出现让许多患者获益。本 文研究了多普勒超声和宫腔镜在子宫内膜病变中的研究进展。

PO_570

VTCN1: A Key Regulator of Immune-Silent Phenotypes in Gynecologic Cancers

Yan Ding Bilan Li*
Shanghai Key Laboratory of Maternal Fetal Medicine, Shanghai Institute of Maternal-Fetal Medicine and Gynecologic Oncology, Shanghai First Maternity and Infant Hospital, School of Medicine, Tongji University, Shanghai 200092, China

VTCN1, also known as B7-H4, B7x, or B7S1, has emerged as a non-redundant immune checkpoint that plays a pivotal role in gynecologic malignancies—particularly those exhibiting immune-excluded ("cold") phenotypes and resistance to PD-1/PD-L1 blockade. Unlike classical B7 family members, B7-H4 lacks a defined receptor and canonical intracellular signaling motifs, yet exerts potent immunosuppressive effects by modulating effector T cells, regulatory T cells (Tregs), dendritic cells (DCs), and tumor-associated macrophages (TAMs).

The VTCN1 gene, located on chromosome 1p11.1, encodes a type I transmembrane glycoprotein comprising an N-terminal signal peptide, two extracellular Ig-like domains, a single transmembrane segment, and a minimal cytoplasmic tail. Although VTCN1 mRNA is widely detected in normal tissues, protein expression is tightly controlled and generally absent under physiological conditions, indicating robust post-transcriptional and post-translational regulation. In the tumor microenvironment, VTCN1 can be induced by proinflammatory cytokines (e.g., IL-6/JAK/STAT3 and IL-10), the aryl hydrocarbon receptor (AhR), hypoxia-inducible factor 1α (HIF-1α), and hormonal cues such as progesterone. Key post-translational modifications critically shape protein stability and function: extensive N-linked glycosylation shields B7-H4 from AMFR-mediated ubiquitination and proteasomal degradation; ZDHHC3-mediated palmitoylation inhibits lysosomal degradation; and proteolytic cleavage by NRD1 and ADAM family metalloproteases releases soluble B7-H4 (sB7-H4) into circulation. Elevated sB7-H4 correlates with advanced stage, lymph node metastasis, platinum resistance, and poor surgical outcomes in epithelial ovarian cancer (EOC), and may serve as a prognostic and diagnostic biomarker, especially in combination with CA125 and HE4.

Functionally, VTCN1 orchestrates a multi-layered immune suppression program. It directly inhibits $CD4^+$ and $CD8^+$ T-cell activation, proliferation, and cytokine production; promotes the expansion of immunosuppressive Tregs; impairs DC maturation and antigen presentation; and marks an M2-like TAM subset that sustains a tolerogenic tumor niche. Through these mechanisms, VTCN1 maintains immune exclusion in the absence of PD-L1 expression and adaptive immune pressure, contributing to resistance against conventional immune checkpoint inhibitors (ICIs).

Clinically, VTCN1 is broadly overexpressed across gynecologic cancers, with tumor-specific enrichment and minimal expression in normal tissues. In EOC, positivity rates range from 75–85%, with higher expression in poorly differentiated and metastatic tumors. In cervical cancer, VTCN1 expression exceeds 80% and independently predicts poor survival, correlating with elevated Treg infiltration and reduced IL-2 production. In endometrial carcinoma, VTCN1 is expressed across molecular subtypes, with particularly strong associations in the non-specific molecular profile (NSMP) subgroup, where it predicts recurrence and disease-specific mortality. Notably, in early-stage EOC, combined detection of B7-H4 and CA125 improves sensitivity over CA125 alone.

Therapeutically, VTCN1 has become an attractive target for next-generation immunotherapy in immune-cold, therapy-resistant tumors. Antibody–drug conjugates (ADCs) represent the most advanced strategy: XMT-1660 (dolasynthen-based, auristatin F-HPA payload) and SGN-B7H4V (vedotin-based, MMAE payload) have shown potent preclinical activity, including in PD-1–refractory models, and are in early-phase clinical trials. Novel B7-H4–targeted ADCs with pyrrolobenzodiazepine payloads demonstrate efficacy even in platinum- and PARP inhibitor–resistant ovarian cancer. Bispecific antibodies, such as HBM7008 (B7-H4 × 4-1BB), offer conditional T-cell activation within the tumor microenvironment, avoiding systemic toxicity while enhancing anti-tumor immunity.

Despite these advances, several challenges remain. The identity of the VTCN1 receptor is unknown, hindering full mechanistic understanding and rational drug design. mRNA–protein discordance and

spatial expression heterogeneity complicate biomarker-driven patient selection. Furthermore, dynamic regulation by microenvironmental cues may limit therapeutic durability. Addressing these gaps will require integrating spatial transcriptomics, single-cell profiling, and functional immunology to refine patient stratification and therapeutic combinations.
In summary, VTCN1/B7-H4 is a tumor-selective, immune-suppressive ligand that defines and sustains immune-silent phenotypes in gynecologic cancers. Its distinct biology—operating outside classical inflamed tumor contexts—makes it a compelling biomarker and therapeutic target in settings where PD-1/PD-L1 blockade fails. Continued mechanistic dissection and clinical validation of VTCN1-targeted strategies could unlock new avenues for effective immunotherapy in these historically resistant malignancies.

PO_571

髓源抑制细胞的免疫调节功能及其代谢重编程机制研究进展

迟楮丹
中山大学

髓源抑制细胞（MDSCs）是一类具有显著免疫抑制功能的未成熟髓系细胞群体，广泛参与肿瘤进展及自身免疫性疾病的免疫调节过程。本文综述了 MDSCs 的免疫抑制功能及其代谢重编程机制。在代谢层面，MDSCs 的糖代谢重编程，如有氧糖酵解增强，可减少活性氧积累，维持其增殖及免疫抑制功能。氨基酸代谢方面，精氨酸、谷氨酰胺、色氨酸等代谢途径对 MDSCs 的存活、增殖及免疫抑制功能具有重要调控作用。脂质代谢改变亦显著影响 MDSCs 功能，肿瘤微环境中的 MDSCs 通过上调脂质转运受体表达增强脂肪酸摄取，激活脂肪酸氧化途径增强免疫抑制功能，胆固醇代谢产物则可损害 T 细胞功能。深入探究 MDSCs 代谢调控机制，结合多组学技术解析其代谢网络，对开发针对 MDSCs 的免疫治疗策略意义重大，有望为癌症及自身免疫性疾病治疗提供新思路与靶点。

PO_572

Medical Education in Gynecologic Endoscopy: Lessons Learned from the Tri-tiered Training Architecture

Aiting Wang Yili Chen Qianrun Chen Qiaojian Zou Qiuwen Mai Xiaojun Wang Xiaoying Lin Yan Liao Mengxun Wei Chudan Chi Yuzhou Xiao Jiayun Fang Feitianzhi Zeng Qiqiao Du Linjing Yuan Junxiu Liu*
the First Affiliated Hospital of Sun Yat-sen University

In recent years, gynecologic endoscopy has rapidly developed in China and become a pivotal technology for diagnosis and treatment of gynecological diseases. However, the standardized training for grassroots physicians still faces multiple challenges: the disconnection between theoretical teaching and clinical skill development, the homogenization of training models that fail to meet the trainees' personalized needs, and lack of comprehensive training methods that integrate clinical practice, research, and teaching. This article focused on the training of gynecological grassroots physicians, and systematically summarized the training mode and assessment system of gynecologic endoscopy in the First Affiliated Hospital of Sun Yat-sen University (FAH-SYSU) through a multidimensional analysis of its Tri-tiered Training Architecture. This article revealed the current status of gynecologic endoscopy training in China, and highlighted the innovative elements of the training system at The Department of FAH-SYSU: the Tri-tiered Training Architecture, holistic learning ecosystem, and others. By analyzing the training effectiveness and existing problems, this article proposed 3 major improvement directions: technology integration, personalized evaluation, and resource optimization, promoting the popularization and application of technology, and providing

important guidance for the establishment of a standardized training system for gynecologic endoscopy in China.

PO_573

HPV integration detection as a novel and promising triage strategy for managing HPV-positive women

Xite Lin[1,2] Binhua Dong[1,2] Pengming Sun[1,2*]
1.Fujian Medical University
2.Fujian Maternity and Child Health Hospital

Background The clinical management of HPV-positive women remains challenging, as many HPV infections are transient. Universal colposcopy referral for all HPV-positive individuals risks over-referral. This study aimed to evaluate HPV integration status as a risk-stratification biomarker and its potential to optimize cervical cancer screening protocols.

Methods A cross-sectional study was conducted involving 949 HPV-positive women who underwent routine screening at Fujian Maternity and Child Health Hospital between October 2021 and July 2024. HPV integration was detected via high-throughput viral integration detection (HIVID). Multivariable logistic regression, bootstrap resampling (10,000 iterations), and Bayesian methods were employed to evaluate associations between HPV integration and risk of CIN3+. Diagnostic performance metrics and synergistic effects of combined strategies (cytology, HPV 16/18 genotyping, and HPV integration) were evaluated.

Results The HPV integration positivity rate was 6.6%, strongly linked to HPV16/18 infection ($p<0.001$). Women with HPV integration had significantly higher risks of severe cytological (17.5% vs. 4.5%) and pathological abnormalities (57.1% vs. 2.0%) (both $p<0.001$). Multivariate regression identified HPV integration as the strongest predictor of CIN3+ (Adjusted OR=59.52), with a notable synergistic effect with HPV16/18. HPV integration detection outperformed cytology in specificity (96.98% vs. 64.25%) and positive predictive value (57.14% vs. 10.36%) for CIN3+ diagnosis, while maintaining similar sensitivity and negative predictive value. Combining HPV integration detection with HPV16/18 genotyping improved risk stratification, enhancing specificity (69.50% vs. 44.25%) and PPV (14.69% vs. 9.11%) compared to cytology and HPV genotyping, without losing sensitivity (87.04% vs. 92.59%). The NILM/integration-negative group (59.2% of the cohort) had a minimal CIN3+ risk (0.7%). The HPV integration-based strategy reduced colposcopy referrals by 82% (6.6% for HPV integration vs. 37.6% for cytology，$p < 0.001$).

Conclusions HPV integration status is a robust biomarker for refining risk stratification in HPV-positive women. Its integration into screening protocols may enhance precision in colposcopy referrals and reduce unnecessary interventions.

PO_574

Long-term Effects of Unilateral Salpingo-oophorectomy: A Cohort Study Based on Data from Sweden and United Kingdom

Shihuang Liu[1,2] Chaohua Tang[1,2] Qiuhua Hong[1,2] Jimiao Huang[1,2] Jianguang Ji[3,4] Huan Yi[1,2*] Xiangqin Zheng[1,2]
1.College of Clinical Medicine for Obstetrics & Gynecology and Pediatrics, Fujian Medical University
2.Fujian Maternity and Child Health Hospital
3.Lund University 4.University of Macau

Background Opportunistic bilateral salpingo-oophorectomy (BSO) is recommended for individuals at high genetic risk of ovarian cancer (OC) who have undergone hysterectomy and do not intend to

conceive. Although BSO significantly reduces OC risk, premenopausal BSO induces iatrogenic perimenopausal states, elevating the risks of coronary artery disease (CAD) and osteoporosis. Thus, performing BSO in premenopausal women without malignancy indications remains controversial. Unilateral salpingo-oophorectomy (USO) preserves contralateral ovarian endocrine function while removing one adnexa. However, whether premenopausal USO prevents OC without increasing long-term risks of CAD, osteoporosis, or other health outcomes remains unclear.

Objective This multinational dual-cohort study aimed to evaluate the long-term effects of USO on OC, CAD, osteoporosis, and other health outcomes, comparing metabolic risks with BSO.

Methods Using the Swedish National Registry (1993–2018; n=42,306) and the UK Biobank prospective cohort (2006–2010; n=3,315), women undergoing USO before age 50 were included. Propensity score matching (1:5 ratio) selected non-USO controls. Follow-up spanned from the USO date to the first diagnosis of outcomes, death, or study end (median: 13 years in Sweden, 29 years in the UK). Multivariable Cox proportional hazards models calculated hazard ratios (HRs) and 95% confidence intervals (CIs) for OC subtypes, CAD, osteoporosis, and other outcomes, with age-stratified analyses.

Results In the Swedish cohort, USO significantly reduced risks of high-grade serous carcinoma (HR=0.65, 95% CI: 0.46–0.90) and clear cell carcinoma (HR=0.44, 95% CI: 0.20–0.99), though overall OC risk showed no statistical difference (HR=0.87, 95% CI: 0.69–1.09). Dual-cohort analyses revealed no increased risks of CAD (Sweden: HR=1.01, 95% CI: 0.94–1.08; UK: HR=1.17, 95% CI: 0.99–1.37) or osteoporosis (Sweden: HR=1.07, 95% CI: 0.98–1.16; UK: HR=1.06, 95% CI: 0.90–1.27). Age stratification in the UK cohort showed elevated CAD risk (HR=1.26, 95% CI: 1.03–1.54) and cognitive impairment (HR=1.72, 95% CI: 1.02–2.90) in women aged 18–40. Compared to USO, BSO in women <40 years significantly increased risks of CAD, osteoporosis, cognitive impairment, and venous thromboembolism.

Conclusion: USO before age 50 selectively reduces risks of high-grade serous and clear cell carcinomas without significant metabolic burden. However, younger women (<40 years) require vigilance for cardiovascular and cognitive risks. Compared to BSO, USO minimizes menopausal complications, underscoring the value of preserving the contralateral ovary in balancing oncologic prevention and long-term health.

PO_575

Unveiling the Lipid-Uterine Leiomyomas Connection: Mendelian Randomization Sheds Light on Novel Therapeutic Targets

Xite Lin[1,2] Pengming Sun[1,2*]
1.Fujian Medical University
2.Fujian Maternity and Child Health Hospital

Background Observational studies suggest a link between dyslipidemia and uterine leiomyomas (ULs), but confounding factors may affect this association. This research employs Mendelian randomization (MR) to assess causal relationships between lipid traits and UL risk. It also explores potential lipid-lowering drug targets for preventing and treating ULs.

Methods This study utilized a two-sample MR approach, incorporating lipid trait data from the Global Lipids Genetics Consortium (GLGC) GWAS. Lipid-lowering drug targets were identified using DrugBank and literature sources, while eQTLGen data were integrated to evaluate the impact of gene expression on ULs risk. Both univariate and multivariate MR analyses, along with SMR analysis, offered detailed insights.

Results The MR analysis identified significant causal associations between lipid traits and ULs risk, highlighting the critical role of lipid metabolism in the pathogenesis of ULs. Genetically predicted higher triglyceride (TG) levels were associated with an increased risk of ULs in European population and

higher low-density lipoprotein cholesterol (LDL-C) levels were associated with an increased risk of ULs in East Asian population. CETP inhibitor showed potential in lowering ULs risk among women from UK and Finnish cohorts, while no such association was found in East Asian cohorts. Additionally, LPL activator and APOC3 inhibitor were identified as protective factors against ULs in the East Asian cohort. These results suggest that CETP, LPL, and APOC3 may serve as promising therapeutic targets for ULs.

Conclusions This study provides genetic evidence linking lipid traits to ULs risk and highlights lipid metabolism as a novel therapeutic target. Findings underscore the need for personalized lipid management strategies tailored to genetic and environmental factors for UL prevention and treatment.

PO_576

The value of 3D reconstruction in the operation of complex uterine malignant tumor

Li Li*
Department of Gynecologic Oncology, Guangxi Medical University Cancer Hospital

In this study, 3d visual reconstruction technology was applied to the surgery of complex uterine malignant tumor, and the new technology was introduced to provide a new idea for the design of surgical plan of gynecological malignant tumor and a new auxiliary tool for individualized treatment. The application value of 3D reconstruction technique in preoperative evaluation, surgical plan determination and surgical quality control of gynecological malignant tumor was evaluated according to the practical application effect.

Methods The DICOM data of pelvic and abdominal cavity of 5 patients with uterine malignant tumor who underwent surgical treatment in the Gynecological Oncology Department of Guangxi Medical University Tumor Hospital were obtained by CT scanning. Mimics Research 19.0 software was used to perform 3D visualization reconstruction of pelvic internal structure. Three-dimensional reconstruction model was used to observe the tumor and its surrounding organs, related arteries and veins, lymph nodes. Combined with 3d reconstruction model, preoperative evaluation and surgical plan were made. The individual reconstruction model was compared with the actual intraoperative situation to observe the operation time, blood loss and other conditions to evaluate the impact of 3d visualization reconstruction technology on clinical surgery.

Results The three-dimensional models of pelvic and abdominal cavity of all patients were reconstructed successfully. The models can show the pelvic and abdominal vessels, some organs in pelvic cavity, the anatomical morphology of tumor, the position relationship between tumor and surrounding structures, and the relationship between lymph nodes and vascular structure. According to the results of three-dimensional reconstruction model, systematic lymph node dissection was performed in all 5 patients with cervical cancer, including 2 patients who underwent C2 type hysterectomy and3 patients who underwent laterally extended parametrectomy. One patient with uncontrolled endometrial cancer underwent total pelvic exenteration + rectal and ureterostomy. The intraoperative exploration was consistent with the 3D reconstruction model.

Conclusion Medical digital 3D reconstruction model technology can be applied to preoperative evaluation, surgical planning and preoperative communication of gynecological malignant tumors, which has certain guiding significance in the operation of complex gynecological malignant tumors.

PO_577

A glutamine-induced multifunctional injectable hydrogel for accelerating vaginal wound healing

Zhongyi Zhu[1,2] Yijing Zhang[1,2] Yi Wang[3] Yuedong He[1,2*]

1.Department of Gynecology, West China Second University Hospital, Sichuan University

2.Key Laboratory of Birth Defects and Related Diseases of Women and Children (Sichuan University), Ministry of Education

3.Sichuan Normal University, College of Chemistry and Materials Science- Key Laboratory of the Evaluation and Monitoring of Southwest Land Resources Ministry of Education, Chengdu, China

Background Vaginal reconstruction surgery is widely used in gynecology for patients with malignant tumors. The rapid wound healing after vaginoplasty remains a significant challenge globally. Conventional treatment methods often involve extended recovery periods and increased risk of retrograde infections, causing significant pain and discomfort for patients. To address this issue, an injectable, anti-swelling, stable hydrogel has been developed to facilitate vaginal wound repair.

Methods First, to confirm the design and characterization of hydrogel, the structure is characterized by scanning electron microscope (SEM), fourier transform infrared spectrometer (FTIR), small angle X-ray scattering (SAXS). Then, rheological tests, fatigue resistance test, mechanical and anti-swelling properties measurements were applied to elucidate the hydrogel properties. In order to evaluate the biocompatibility, we employed elution medium model, investigate the in vitro cytotoxicity of the hydrogel using live/ dead cell staining and cell counting kit-8 (CCK-8) cell viability assay. Meanwhile, to further investigate the in vivo biocompatibility, we implanted the hydrogel into the dorsal skin of rats and collected samples for hematoxylin and eosin (HE) staining and interleukin-6 (IL-6) immunohistochemical staining. Finally, to evaluate the wound healing effect of the hydrogel and molecular mechanisms involved in the healing process in vivo, we established a standardized rat vaginal defect model and captured wound photos followed Hematoxylin and Eosin (H&E) staining, Masson's trichrome staining, Ki-67 immunohistochemical staining, and TGF-β, CD31, vWF immunohistochemical staining.

Results This 2-hydroxyethyl methacrylate, sodium alginate/Mg2+, L-glutamine hydrogel (PHSG-Mg2+ hydrogel) has good injectability and can accurately fit the vaginal shape of different patients. By introducing the complexation of sodium alginate and Mg2+, a hydrogen bond/coordination bond dual physical cross-linking network structure is constructed, which significantly improves the mechanical properties and anti-swelling ability of the hydrogel, so that the hydrogel could basically maintain its initial shape within 30 days, providing continuous support and protection for vaginal wounds. At the same time, the hydrogel also has excellent biocompatibility, and has shown a significant therapeutic effect in promoting wound healing in the rat vaginal wound model by enhancing epithelium growth, accelerating extracellular matrix formation, and promoting vasculation.

Conclusion Collectively, our findings indicate that PHSG-Mg2+ hydrogel provides a new method for achieving satisfactory wound healing and supports the potential use of mechanically robust hydrogel as a drug-free therapy after vaginoplasty.

PO_578

Endometriosis in para-aortic lymph nodes without any pelvic endometriosis: the first case report, sequencing analysis and literature review

Yu Chen Chunyan Hao Qing Zhang Qiuli Teng Xiaoming Zhang Guowei Tao Lijie Wang*
Shandong university Qilu hospital

Objective This article introduces a novel case report of a patient exhibiting endometriosis within the para-aortic lymph nodes, concomitant with cystic adenomyosis and notably lacking pelvic endometriosis, alongside the sequencing analysis of her lesions. By synthesizing these findings with prior instances of lymph node endometriosis, the study seeks to offer innovative insights and identify potential targets for the diagnosis and treatment of this condition.

Materials and Methods A 36-year-old patient presented with escalating dysmenorrhea and a swiftly expanding abdominal mass, comparable to the size of a 5-month pregnancy. Imaging revealed the presence of a para-aortic mass measuring 4.2 cm, alongside a sizable cystic solid lesion in the pelvic abdomen featuring numerous internal grid-like patterns and a rich blood supply measuring approximately 23.5×12.4×27.0 cm3. Elevated levels of CA125, CA199 and prolactin were detected. The uterus and enlarged para-aortic lymph nodes were resected via laparotomy, with no additional endometriosis lesions detected in the pelvic cavity during the procedure. Pathological diagnosis confirmed endometriosis in the para-aortic lymph nodes and cystic adenomyosis. Sequencing analysis of this case was performed, and the similarities and differences between this case and previously reported cases of lymph node endometriosis were integrated to further explore the pathophysiology of this disease and identify potential therapeutic targets.

Results Due to the lack of reliable biomarkers, lymph node endometriosis is frequently misdiagnosed as a malignant tumor. Its definitive diagnosis heavily relies on the surgeon’s expertise and imaging evaluation. In contrast to previous studies on lymph node endometriosis associated with severe pelvic endometriosis (e.g. deep infiltrating or intestinal endometriosis), the present study demonstrates that retroperitoneal endometriosis can occur independently, suggesting a more invasive phenotype of endometrial tissue in this patient. Our study provides corroborative evidence for Halban's "Benign Metastasis" theory in endometriosis, which posits that benign endometrial stroma and glands can disseminate through lymphatic and vascular channels. Furthermore, RNA sequencing analysis of lesions indicated that the lesions exhibited up-regulation of genes and pathways associated with cell activation, adhesion, inflammation, and immunity. The WES analysis indicated that FOXA2 and SRRM2 were respectively candidate driver genes for endometriosis in para-aortic lymph nodes and cystic adenomyosis requiring validation in larger cohorts and functional studies. Consequently, these molecular alterations may contribute to an augmented presence of blood vessels and lymphatic vessels in the lesion, thereby facilitating the metastasis, colonization, and growth of ectopic endometrial cells in distant lymph nodes.

Conclusion

1. Retroperitoneal lymph node endometriosis can occur independently of other forms of endometriosis. The patient's unique imaging, high tumor markers, and swift disease progression necessitated careful preoperative differentiation from uterine malignancies. Therefore, clinicians should maintain a high index of suspicion and conduct comprehensive assessments for differential diagnosis.
2. We identified FOXA2 and SRRM2 as potential candidate driver genes for endometrial metastasis to lymph nodes and myometrium, respectively. These findings offer novel insights and directions for subsequent mechanistic studies on the pathophysiology of endometriosis.

PO_579

子宫腺肌病患者发生尿失禁的相关危险因素分析

胡昱如[1] 吴世豪[1] 彭程[1,2*]
1.安徽省蚌埠医科大学研究生院 2.中国科学技术大学附属第一医院

目的 子宫腺肌症是一种常见的慢性妇科疾病，其主要特征为进行性加重的痛经和月经过多。压力性尿失禁（stress urinary incontinence，SUI）指腹压突然增加导致的尿液不自主漏出，其严重程度影响患者生活质量。临床实践中，子宫腺肌症患者表现出显著的 SUI 高发倾向，但其潜在机制尚不明确。本研究旨在探讨子宫腺肌症患者发生 SUI 的相关风险因素。
材料与方法 本研究于 2023 年 1 月至 2024 年 4 月期间，共纳入 304 例确诊的子宫腺肌症患者。记录患者人口统计学资料、超声测量的子宫径线（长、宽、厚）、子宫体积（长×宽×厚×0.5233 cm^3）、血清 CA125 水平等变量。ICIQ-SF 评分≥1 分且具有 SUI 典型症状的患者被归入 SUI 组。采用 Logistic 回归分析探讨与子宫腺肌症患者发生 SUI 相关的风险因素，结果以比值比（OR）及其 95%置信区间（95% CI）表示。最终 Logistic 回归模型通过受试者工作特征（ROC）曲线和校准曲线进行评估。
结果 单因素分析显示，体重指数（BMI）、流产史、子宫体积以及子宫各径线（长、宽、厚）在两组间差异具有统计学意义（$p<0.05$）。多因素 Logistic 回归分析显示，BMI（OR=1.09, 95% CI: 1.01-1.18, p=0.04）、子宫长度（OR=1.03, 95% CI: 1.01-1.04, p<0.001）以及≥2 次阴道分娩史（OR=2.37, 95% CI: 1.26-4.45, p=0.008）是子宫腺肌症患者发生 SUI 的独立相关因素。该模型的 ROC 曲线下面积（AUC）为 0.69。Hosmer-Lemeshow 检验及校正曲线显示模型预测值与观测值具有良好的一致性。
结论 本研究结果与既往多项研究一致，提示子宫腺肌症与 UI 存在关联。在子宫腺肌症患者中，较高的 BMI、增大的子宫径线以及≥2 次阴道分娩史可能是增加 SUI 发病风险的潜在因素。临床实践中需重视对子宫腺肌症患者的早期识别与治疗，以降低其患 SUI 的风险，改善患者生活质量。

PO_580

巨噬细胞相关分子在子宫内膜异位症中的变化：一项系统评价和 Meta 分析

黄革 李娜*
遵义医科大学第二附属医院

目的 有大量证据表明，巨噬细胞以及相关分子在子宫内膜异位形成的机制中发挥作用。这项全面的系统评价和 meta 分析的目的是研究组织或者体液内巨噬细胞相关分子变化是否与子宫内膜异位症的机会增加以及分期有关。对巨噬细胞相关分子的研究为预测子宫内膜异位症以及严重程度的风险和对其治疗的易感性提供了新的可能性。
方法 系统检索 PubMed、EMBASE、Web of science、Cochrane Library 数据库，纳入比较子宫内膜异位症和对照组中巨噬细胞相关分子变化的原始研究，检索时间截止至 2025 年 04 月 30 日。采用相对危险度（OR）及 95%置信区间（CI）评估血清、腹液以及组织中巨噬细胞的配受体、分泌因子与子宫内膜异位症以及严重程度的风险。
结果 本 Meta 分析共纳入 179 项研究。分析结果显示，子宫内膜异位症患者的腹液及血液中 IL-1β、IL-4、IL-6、IL-8、IL-10、IL-12、IL-17A、IL-37、MCP-1、MIF、MMP-9、TNF-α、TGF-β、VEGF 明显高于对照组；IP-10 在血清中明显下降，在腹液中无明显变化趋势；IL-18、CD14%、HLA-DR%、CD14 MFI、HLA-DR MFI、CD54 MFI、GM-CSF、RANTES 在血清及腹液中均无明显差异。CD68 巨噬细胞在异位子宫内膜组织中出现明显数量聚集。此外，IL-10、IL-37 在子宫内膜异位症晚期患者的腹液及血液中较 EM 早期中均明显升高。

结论 子宫内膜异位症呈现明显的 M1/M2 并存，导致“高炎症-高免疫调节状态”，且在晚期子宫内膜异位症中这种状态更明显，未来需更大样本、标准化的 RCTs 进一步验证巨噬细胞指标的诊治意义。

PO_581

Cervi-LLM: A Multimodal Mixture-of-Experts Framework for Real-Time Colposcopic Lesion Detection and Interpretable Diagnosis

Zhijun Hu[1] Ling Ma[2] Suizhi Huang[2] Lihua Qiu[1*]
1.Renji Hospital, School of Medicine, Shanghai Jiao Tong University
2.Shanghai Jiao Tong University

Aim of the study Colposcopy is a cornerstone of cervical lesion screening but remains heavily dependent on physician expertise and lacks real-time, precise lesion localization and stratified diagnosis. This study aims to address these limitations by developing Cervi-LLM, an intelligent diagnostic framework based on multimodal Mixture-of-Experts.

Methods This study retrospectively analyzed 2,123 patients who underwent colposcopy at Shanghai Jiao Tong University School of Medicine’s Renji Hospital Cervical Outpatient Clinic between January 2018 and September 2022. The cohort included 126 cervical cancer cases, 692 HSIL cases, 306 LSIL cases, and 999 normal cases, with a mean participant age of 41.42 years (Ethics Approval: KY2023-009-B). To address diagnostic challenges, we propose Cervi-LLM featuring three core components: 1) The YOLOMed model enabling real-time, multi-scale detection and segmentation of three colposcopic staining modalities (saline, 3% acetic acid, iodine) for high-quality image feature extraction; 2) Joint fine-tuning of a large language model on clinical text and image features via LoRA adapters and three-stage progressive unfreezing; 3) A two-level Mixture-of-Experts architecture employing dynamic gating to fuse outputs from rule-based medical logic experts and data-driven experts, with clinical rule calibration generating interpretable diagnoses across categories (Normal, LSIL, HSIL(+)) (Figure 1).

Results We split the dataset into 70% training and 30% testing sets to evaluate model performance. In colposcopic image analysis, Cervi-LLM’s detection performance is slightly lower than YOLOv5s (mAP@0.5 of 0.69 vs. 0.72, mAP@0.95 of 0.32 vs. 0.33), but the latter only supports a single detection task (Table I). For segmentation, Cervi-LLM achieves a PA of 0.945 and a mean Intersection over Union (meanIoU) of 0.78, outperforming U-Net (0.918/0.72) and Polyp-PVT (0.936/0.75). In classification evaluation, we merged HSIL and cancer into a single HSIL(+) category, resulting in three classes: Normal, LSIL, and HSIL(+). On the validation set, Cervi-LLM achieves an overall accuracy of 0.9152 and an average AUC of 0.93, significantly outperforming Logic-Only (0.8279/0.84), YOLOMed (0.7121/0.75), and the Early Fusion MLP baseline (0.8346/0.84) (Table II). In comparison with clinical colposcopy experts, Cervi-LLM achieves superior diagnostic performance in the three-class classification task (Normal, LSIL, HSIL(+)), with an overall accuracy of 0.9152 versus 0.6588 for junior physicians and 0.7657 for senior physicians. It also attains a higher HSIL(+) detection rate (0.9485 vs. 0.6422 for juniors and 0.7947 for seniors) and a higher LSIL detection rate (0.8829 vs. 0.6769 for juniors and 0.7156 for seniors)(Table III). We further conducted a targeted analysis on HSIL(+). Cervi-LLM attains a diagnostic accuracy of 0.94 for HSIL(+) cases, significantly outperforming the expert cohort at 0.85. In terms of high-risk lesion detection, Cervi-LLM’s sensitivity and specificity reach 0.93 and 0.89 respectively, compared to 0.84 and 0.86 for the human experts (Table IV) (Figure 2). Regarding turnaround time, Cervi-LLM processes colposcopic images at approximately 30 fps and produces a final diagnosis in 0.30 Å} 0.05 minutes. By contrast, senior and junior colposcopists require on average X Å} Y minutes and A Å} B minutes per case, respectively. These results indicate that Cervi-LLM not only substantially accelerates the diagnostic workflow but also significantly enhances accuracy.

Conclusions Cervi-LLM effectively overcomes the limitations of conventional colposcopy, providing an accurate, real-time intelligent assistant for cervical lesion screening and biopsy guidance.

PO_582

The progress and dilemma of ovarian organoids: adopting strategies from various organoids of body systems

Yijing Zhang[1,2] Zhongyi Zhu[1,2*]
1.Department of Obstetrics and Gynecology, West China Second University Hospital, Sichuan University
2.Key Laboratory of Birth Defects and Related Diseases of Women and Children (Sichuan University), Ministry of Education

Objective Ovarian organoids that serve as an alternative approach for fertility preservation for cancer patients and ideal model for ovarian biological and pathological study, has made great progress for resent years. Here, we review the progress of ovarian organoids and draw advanced strategies and technologies from other organoids research to solve dilemma of ovarian organoids invention.
Methods The literature search was performed in PubMed databases. All studies relating to ovarian organoid and published in the English language were included. Researches of other organoids were search and reviewed in sections according to various obstacles in the invention of ovarian organoids.
Results As one of the most significant breakthroughs in the field of tissue engineering research during the last decade, organoid technology was rated as one of the top ten discoveries by Science magazine. It refers to a self-organized stereochemical tissue that is typically derived from pluripotent, fetal and adult stem cells, progenitor, and /or differentiated cells resembling the key function and structure of an organ through cell-to-cell and cell-to-matrix interactions. Although organoid technology has made great progress, thereby motivating the research and development of artificial ovaries, it continuously encounters practical obstacles: (1) low follicle survival rates and inferior oocytes competence after in vitro culture; (2) inadequate expanding and short-term maintenance; (3) the potential immunogenicity, and batch-to-batch variability of current matrices. The frontier research in other organoid of body systems other than reproductive system may solve the dilemma of ovarian organoid invention with advanced approaches such as widening cell sources, coculturing various types of cells to mimic crosstalk between cells and microenvironment, improving matrices by regulating signaling pathways and inventing synthetic hydrogels to obtain biocompatibility, structural support and degradability for follicle proliferation and migration. Furthermore, the newly arising bioengineering technologies such as 3D bioprinting, microfluidic and genetic tools facilitate ovarian organoids with more accurate, delicate and controllable technological means.
Conclusion The updated research and development in organoids of ovary and other body systems may provide practical experience and bring new light to optimize ovarian organoids' invention.

PO_583

First-line chemotherapy in low-risk gestational trophoblastic neoplasia: A network meta-analysis of randomized controlled trial

Yuquan Yuan Chengzhi Zhao*
chongqing health center for women and children

Background Gestational trophoblastic neoplasia (GTN) is a type of gestational trophoblastic disease that arises from the abnormal proliferation of placental trophoblastic cells. GTN with a FIGO stage I-III and a WHO score of six or below are defined as low-risk GTN. Unlike other malignancies, low-risk GTN exhibits significant invasiveness and metastatic capacity while maintaining a high complete remission rate. The latest National Comprehensive Cancer Network guidelines explicitly recommend methotrexate (MTX) and actinomycin-D (Act-D) as first-line single-agent chemotherapy for low-risk GTN patients. There are several MTX- and Act-D-based single-agent regimens used for low-risk GTN, and the optimal therapeutic strategy remains undefined. Several previous meta-analyses, incorporating both non-randomized and randomized controlled studies, have investigated this topic. However, a recent investigation suggests that using non-randomized studies in place of randomized trials may introduce additional uncertainty regarding the therapeutic effects of drugs. By incorporating the latest randomized controlled studies, this study aims to compare the efficacy and safety of various single-agent chemotherapy regimens, providing more reliable evidence-based guidance for clinical practice.

Methods We systematically searched the PubMed, Cochrane Library databases, Medline, EMBASE, and Web of Science, covering publications up to January 30, 2025. We used logarithm risk ratios for categorical outcomes with corresponding 95% credible intervals. These were pooled in Bayesian network meta-analysis models. The surface under the cumulative ranking curve (SUCRA) was used to illuminate the probability that each single-agent chemotherapy regimen would be the best for each outcome.

Results Eleven studies comprising 875 patients were included. The bi-weekly pulsed intravenous Act-D (Pulsed-IV Act-D) was superior to weekly intramuscular MTX (w-IM MTX) in the complete remission rate. The results indicated that five-day intravenous Act-D (5d-IV Act-D) (0.78) was likely to be the most effective treatment for low-risk GTN in complete remission rate, followed by five-day intravenous MTX (5d-IV MTX) (0.76). SUCRA rankings revealed that the three regimens (5d-IV MTX, 5d-IV Act-D, and pulsed-IV Act-D) achieving the high complete remission rates utilized the intravenous route, while the three regimens (w-IM MTX, 5d-IM MTX, and 8d-IM MTX FA) associated with the low efficacy were exclusively intramuscular routes. Eight-day intramuscular MTX with folinic acid (8d-IM MTX FA) was associated with lower severe adverse events and stomatitis with SUCRA values of 0.72 and 0.79, respectively. 5d-IV MTX demonstrated capability in controlling toxicity in nausea and vomiting with SUCRA values of 0.92 and 0.89, respectively. W-IM MTX had the highest SUCRA value in reducing neutropenia (0.71).

Conclusions The benefits and limitations of each single-agent chemotherapy regimen for low-risk GTN are obvious according to SUCRA values. The evidence suggested that the 5d-IV Act-D and 5d-IV MTX regimen might be the preferred option in terms of complete remission rate. Besides, 5d-IV MTX demonstrated the capability to control toxicity in nausea and vomiting. The intravenous route of administration may result in a higher rate of complete remission than the intramuscular route. Our conclusions should be verified through more high-quality randomized controlled trials involving a large sample size. There are currently multiple registered clinical studies in progress, and the results of these studies will give us more clinical guidance.

PO_584

妇科肿瘤精准诊疗的转化医学路径：从基础研究到临床实践的突破

王雨杭
天津医科大学研究生院

妇科肿瘤作为威胁女性健康的重要疾病，其诊疗面临诸多挑战。转化医学通过整合基础研究与临床实践，为妇科肿瘤诊疗带来了新的机遇。本文深入探讨了妇科肿瘤转化医学的核心领域，包括分子标志物与早期诊断、靶向治疗与免疫治疗、耐药机制与联合疗法；分析了转化医学研究模式的创新，如多组学整合分析、类器官与 PDX 模型、人工智能与大数据的应用；结合临床转化实践案例，阐述了成功经验与面临的挑战；并对未来妇科肿瘤转化医学的发展方向和政策支持提出了展望。

PO_585

ARID1A、CDKN2A、PBRM1 基因突变致子宫腺肌病恶变 1 例并文献复习

王雨杭
天津医科大学研究生院

本文报道一例 52 岁绝经后女性从子宫腺肌病进展为子宫内膜腺癌的罕见病例。患者因痛经进行性加重 10 年和间断非经期下腹痛 3 年入院，既往子宫腺肌病病史，本次入院诊刮病理考虑局灶子宫内膜复杂性增生，行全子宫切除，术后病理确诊为子宫腺肌病伴局灶腺癌。基因检测显示存在 ARID1A、CDKN2A 及 PBRM1 等突变。本病例首次报道了 ARID1A、CDKN2A、PBRM1 在子宫腺肌病恶变中的共突变现象，三者通过表观遗传重塑、血管生成、细胞周期失调和免疫逃逸形成协同致癌网络；共同促进免疫抑制性微环境形成，抑制抗肿瘤免疫应答。分子分型指导下的靶向治疗可能改善此类患者预后。无创性生物标志物有望实现早期诊断与干预。

PO_586

乳腺癌卵巢转移的临床分析

何力玲
宁夏医科大学总医院

目的 分析乳腺癌卵巢转移患者的临床特征、诊断方法、治疗方式及预后情况，为乳腺癌卵巢转移的防治提供临床参考。
方法 回顾性分析本院收治的 15 例乳腺癌卵巢转移患者的临床资料，包括患者的基本信息、乳腺癌原发灶特征、卵巢转移的诊断时间及方式、治疗方法及生存情况等。
结果 15 例患者中，年龄分布在 35 - 62 岁，中位年龄 48 岁。从乳腺癌手术到发现卵巢转移的时间间隔为 1 - 8 年，平均 3.5 年。主要临床表现为盆腔包块、腹胀、腹痛等。所有患者均通过影像学检查（超声、CT 或 MRI）及术后病理确诊。治疗方式包括手术、化疗、内分泌治疗等综合治疗。患者的生存时间存在较大差异，中位生存期为 18 个月。
结论 乳腺癌术后卵巢转移具有一定的临床特点，早期诊断较为困难。综合治疗有助于改善患者的预后，但总体预后仍较差，需进一步探索更有效的防治策略 。

PO_587

Vaginal Peptostreptococcus Anaerobius Promotes Foxp3+ Regulatory T Cells Differatiation through Tumoral TGF-β Production

陈君豪 张梦蕾 顾铮 龚滢欣 周冠楠 丁景新*
复旦大学附属妇产科医院

研究目的 本研究旨在探讨阴道厌氧消化链球菌（Peptostreptococcus anaerobius，P. anaerobius）在宫颈癌患者中的促瘤作用及其免疫调控机制，明确其是否通过诱导肿瘤源性 TGF-β 增多而促进 Foxp3+ 调节性 T 细胞（Treg）分化，从而加速宫颈癌进展。
材料与方法 收集 20 例接受手术的患者（宫颈癌 10 例，良性疾病 10 例）的手术标本及阴道冲洗液。流式细胞术检测肿瘤组织中各类免疫细胞浸润情况并通过免疫荧光染色了解 Foxp3+ Treg 比例。qPCR 测定冲洗液中阴道菌群丰度。用 P. anaerobius 培养上清处理 Hela 细胞，使用 Western blot 与 qPCR 检测 Hela 细胞 TGF-β 表达。收集经 P. anaerobius 培养上清处理后的 Hela 细胞上清液，作用于 C57 小鼠脾脏分离的 CD3+幼稚 T 细胞（经 CD3/CD28 激活），流式细胞术检测 T 细胞分化情况。建立 T 细胞-Hela 体外共培养体系，观察 P. anaerobius 介入对 T 细胞分化的影响。使用 TC-1 细胞系于腋窝处皮下成瘤（C57 小鼠），腹腔注射 TGF-β，监测肿瘤生长情况，流式及免疫组化检测瘤内 Treg 浸润水平。
实验结果 宫颈癌组肿瘤组织中 Foxp3+ Treg 浸润显著增多。宫颈癌患者阴道冲洗液中总细菌量下降，乳酸杆菌水平显著减少而 P. anaerobius 表达水平显著升高。经 PA 上清液处理 48 及 72 小时后，Hela 细胞中 TGFβ 水平显著提升。经 P. anaerobius 培养上清单独或 Hela 上清单独处理的 CD3/CD28 激活 T 细胞均不显著诱导 Treg 分化；而 P. anaerobius 处理的 Hela 上清或外源 TGF-β 均显著提高 Treg 比例。T 细胞-Hela 共培养中在加入 P. anaerobius 培养上清后 Treg 水平进一步升高。TGF-β 处理组肿瘤体积增大，瘤内 $Foxp3^+$ Treg 浸润显著增加。
结论 P. anaerobius 通过上调宫颈癌细胞的 TGF-β 分泌，诱导 Treg 分化，形成免疫抑制微环境，加速肿瘤进展。该菌可作为宫颈癌早期诊断、预后评估的潜在微生物标志物，并为靶向 P. anaerobius-TGF-β-Treg 轴的联合治疗提供新思路。

PO_588

心脏累及性静脉内平滑肌瘤病 6 例临床分析及多学科手术经验

朱三 杨凌云*
四川大学

研究目的 静脉内平滑肌瘤病（intravenous leiomyomatosis，IVL）累及心脏属妇科罕见危重并发症，本研究旨在总结其临床特点、多学科手术策略及预后，为临床诊治提供参考。
材料与方法 回顾性收集 2022 年 11 月至 2025 年 3 月确诊的 6 例心脏累及性 IVL 患者资料。纳入标准包括病理确诊 IVL、影像学或术中发现肿瘤延伸至右心系统（右心房/右心室），并接受多学科联合手术。分析指标涵盖人口学特征、临床表现、影像学检查、手术方案、病理结果及随访结局。
结果 6 例患者年龄 38-49 岁（中位 42 岁），均有生育史，其中 2 例存在子宫肌瘤挖除术病史。主要临床表现为活动后心慌/胸闷（4 例）、晕厥（1 例）及盆腔占位相关症状（3 例）。影像学检查（CTA/MRI/超声心动图）均显示肿瘤经卵巢静脉或生殖静脉侵入下腔静脉并延伸至右心房（6 例），其中 1 例进一步累及右心室；静脉内瘤栓呈多发条索状管道样，漂浮于血管内。所有患者均接受一期多学科联合手术：妇科团队行全子宫及单侧或双侧附件切除，心血管外科团队经胸骨正中切口开胸，6

例均建立体外循环，完整切除心内肿瘤且未发生术中肺栓塞；同期行下腔静脉修补（5 例）、肾静脉或髂静脉处理（4 例）。术后病理证实系静脉内平滑肌瘤，免疫组化显示雌激素受体（ER）及孕激素受体（PR）阳性率 100%（6/6），平滑肌标志物（SMA/Desmin）均阳性；1 例检出 HMGA2 基因不平衡易位。中位随访 5.8 个月（3-33 个月），1 例术后 3 个月出现盆腔复发，余 5 例无瘤生存，无围术期死亡。

结论 心脏累及性 IVL 以右心系统侵犯为特征，活动后心慌及晕厥是重要预警信号。多学科协作根治术（妇科肿瘤切除联合心血管外科体外循环下心内肿瘤摘除）可实现安全治疗，术中需强化栓塞风险防控。ER/PR 普遍阳性支持术后内分泌治疗。长期随访应关注盆腔、静脉及心脏复发可能。

PO_589

Gynecological surgery using the Kangduo robotic system

Yanan Pi Wei Huang Hongyan Wang Zhenxing Sun Meng Chi Liyuan Guo*
Harbin Medical University Cancer Hospital

Background Robotic surgery represents a notable advancement in the field of minimally invasive gynecological surgery. Although the Kangduo Robot® SR1000 (KD-SR-01) surgical system has shown improved stability and high efficiency, studies describing its use for gynecological surgery are limited. We aimed to review the safety and effectiveness of the KD-SR-01 system in gynecologic surgery and compare it with conventional laparoscopic operation.

Materials and Methods We compared patient characteristics and short-term outcomes in cases of gynecological surgery conducted using the KD-SR-01 system and laparoscopic minimally invasive procedures between March 2024 and October 2024. The short-term clinical efficacies of both surgical modalities were compared by performing statistical analyses.

Results The KD-SR-01 system was used for total hysterectomy due to benign uterine tumors in 144 cases, ovarian cyst removal surgery due to benign ovarian tumors in 25 cases, unilateral adnexectomy due to benign ovarian tumors or borderline tumors in 25 cases, staging of early endometrial cancer in 24 cases, and radical cervical cancer resection for early cervical cancer in 24 cases. None of the patients showed serious complications (Clavien–Dindo grade ≥ 3). In comparison with traditional laparoscopic surgery, Kangduo robotic surgery resulted in a lower duration of hospitalization, operation time, blood loss, and drainage volume, but the two surgical modalities showed no differences in the complication rate.

Conclusion The Kangduo robotic system was safe and feasible for gynecological surgery. Evidence from additional studies and more surgical experience are required to determine the long-term outcomes and indications for gynecological surgery using this robotic system.

PO_590

The Regulatory Role of COX-2 in Malignant Transformation of Ovarian Endometriosis via the WNT3a/β-Catenin Pathway and Its Underlying Mechanisms

Lin Deng Yu Zhang* Shumin Li
Cancer Hospital Chinese Academy of Medical Sciences

Objective To investigate the regulatory role of COX-2 in the malignant transformation of ovarian endometriosis and its underlying mechanism
Methods
1.Immunohistochemistry (IHC) was used to detect COX-2 and β-catenin expression in eutopic endometrium, ovarian endometriosis, and ovarian clear cell carcinoma tissues.
2.Serum levels of COX-2 and WNT3a were quantified using ELISA in patients with these conditions.
3.COX-2 was overexpressed in IOSE-80 and ES-2 cell lines, followed by celecoxib rescue treatment.
4.Cell proliferation was assessed by CCK-8 assay
5.Cell invasion and migration were evaluated using Transwell and wound healing assays
6.qPCR and Western blotting were employed to analyze gene and protein expression patterns.
Results
1.Compared with eutopic endometrium and ovarian endometriosis tissues, COX-2 and β-catenin expression were significantly upregulated in ovarian clear cell carcinoma tissues. .
2.The mean serum COX-2 level in ovarian clear cell carcinoma patients was 61.79 ± 14.27 U/L, significantly higher than in eutopic endometrium (47.93 ± 7.706 U/L, $P < 0.05$) and ovarian endometriosis (38.65 ± 5.481 U/L, $P < 0.05$) patients. Serum WNT3a levels in ovarian clear cell carcinoma patients (22.85 ± 5.072 ng/ml) were significantly elevated compared to ovarian endometriosis patients (17.09 ± 2.791 ng/ml, $P < 0.05$).
3.Overexpression of COX-2 significantly enhanced proliferation, invasion, and migration in IOSE-80 and ES-2 cells, upregulated WNT3a, β-catenin, Snail, and Vimentin expression, and downregulated E-cadherin. These effects were inhibited by celecoxib.
Conclusion Elevated expression of COX-2 and β-catenin in ovarian clear cell carcinoma may serve as potential indicators for malignant transformation of ovarian endometriosis and represent therapeutic targets. COX-2 likely promotes malignant transformation by regulating the WNT3a/β-catenin/EMT pathway.

PO_591

化疗所致早发性卵巢功能不全的中医药治疗的研究进展

刘思月[1] 蔡云朗[1,2*]
1.东南大学 2.东南大学附属中大医院

目的 早发性卵巢功能不全（premature ovarian insufficiency，POI）指女性 40 岁之前出现卵巢的内分泌功能丧失，可能导致不孕、泌尿生殖系统萎缩和骨质疏松症以及心血管疾病和神经退行性疾病风险增加[1,2]。以宫颈癌为例，宫颈癌好发于育龄期女性，在接受化疗的育龄期女性患者中，约 70%的患者会出现不同程度的不可逆性的卵巢功能损伤，最终导致早发性卵巢功能不全[3]。目前，针对 POI 的治疗，西医主要使用性激素替代、免疫调节、间充质干细胞移植和基因治疗等改善临床症状，以及使用辅助生殖技术解决生育问题，但这些方法均存在不同程度的局限性[4]。因此，本文将就 POI 的中医药治疗的潜在机制及临床应用价值的研究进展进行综述。
方法 通过早发性卵巢功能不全、中医药、研究等关键词在中国知网、Pubmed、Web of Science 等数据库中进行检索文献，经过阅读、整理，进行相关方面综述。

结果 中医药疗法具有安全性更高、不良反应小等优点，且在缓解临床症状、提高患者生育能力和生活质量等方面发挥显著优势，已成为治疗 POI 的重要手段。
近年来，临床和动物实验研究表明，中医药治疗 POI 作用机制复杂，主要涉及调节神经内分泌系统、改善卵巢功能、调控相关信号通路、增强机体免疫功能、提高抗氧化应激能力、维持线粒体能量代谢、抑制铁死亡、调节内质网应激等方面，中医药对调治 POI 所致的月经失调、不孕及情绪等问题发挥了重要作用。
资癸益冲方可以改善 POI 模型小鼠的血清激素水平，提高糖酵解水平,减少小鼠卵巢颗粒细胞的凋亡和卵泡闭锁，改善卵巢储备功能[5]；六味地黄丸通过 PGC-1α-TFAM-ROS 信号通路在一定程度上抑制早发性卵巢功能不全小鼠卵巢颗粒细胞凋亡，从而改善卵巢的内分泌功能，提高机体抗氧化能力，减轻氧化应激损伤程度[6]；补肾活血方可抑制卵泡闭锁，促进卵泡生长，改善内分泌紊乱，恢复卵巢储备，通过调节 VEGF 和 bFGF 的表达促进卵巢血管生成，促进骨髓间充质干细胞的增殖、迁移和归巢到卵巢[7]；育肾养卵方通过 HIF-1α/VEGFA 信号通路，改善卵巢血管生成，抑制卵巢颗粒细胞凋亡，从而对卵巢储备功能有改善作用[8]。
讨论 研究表明，育龄期女性接受具有性腺毒性的化疗方案可导致 POI，其发生机制主要涉及原始卵泡储备的不可逆耗竭，这一病理过程的发生发展与多种因素密切相关[9,10]。中医药凭借其整体观念、多靶点作用和个体化治疗策略，为女性提供了更安全、有效的治疗选择。未来研究应进一步探索中西医结合的治疗模式，深化中医药作用机制的分子水平研究，以推动其在 POI 及相关领域的临床应用。

PO_592

BRCA1 突变患者行预防性附件切除术后发生盆腔腺癌一例：病例报道及文献综述

左芮瑜[1] 朱仲毅[1,2] 晏和春[1] 陆玉坤[1,2] 何跃东[1,2*]
1.四川大学华西第二医院妇科
2.四川大学妇女儿童出生缺陷及相关疾病教育部重点实验室

背景及目的 BRCA1/2 为高风险卵巢癌遗传易感基因，其突变携带者发生卵巢癌、乳腺癌、输卵管癌的风险显著高于普通女性。目前，降低风险的输卵管卵巢切除术（risk-reducing salpingo-oophorectomy, RRSO）是预防此类患者发生卵巢癌最有效的方法。然而此类患者术后发生盆腔恶性肿瘤的风险仍长期存在。我们在此报道一例因 BRCA1 突变在四川大学华西二院接受 RRSO 术后发生盆腔高级别浆液性腺癌患者，并进行相关文献综述，以警示高风险 RRSO 患者术后随访的重要性。
方法 整理患者病历资料进行汇报，并在 Pubmed 数据库检索 2005-2025 年间所有关于 BRCA 和 RRSO 及其同义词的相关文章，限定文章类型为 case report，然后进行人工检索，筛选出与本例报道的病例临床特征相似的病例。
结果 本病例为一名 56 岁的女性，因卵巢癌家族史行基因检测确认为 BRCA1 突变携带者，在华西二院接受 RRSO。术后病理未见附件恶性病变，后规范随访。术后 18 个月时，患者因腹胀再次于华西二院就诊，腹腔穿刺腹水病理检查提示女性生殖道来源的高级别浆液性腺癌，影像学显示腹腔广泛种植转移灶及淋巴结肿大。我院予新辅助化疗联合间歇性肿瘤细胞减灭术 (IDS)，最终诊断为盆腔高级别浆液性腺癌 IIIc 期（T3cNxM0）。经过文献检索，筛选到 4 例与本报道的病例临床特征相似的病例。4 例既往报道的病例均为因 BRCA1/2 突变行 RRSO 后随访期间发生盆腔恶性肿瘤，其中原发性腹膜浆液性乳头状癌 1 例、高级别浆液性癌 2 例、高级别浆液性腺癌 1 例。与本病例一样，既往报道病例在随访期间发现盆腔恶性肿瘤时均已为晚期（III-IV 期）。本病例与既往报道类似情况病例患者共 5 例，均在 RRSO 后发生盆腹腔广泛转移，其中 2 例患者发生膀胱和肠道转移，1 例发生胸腔转移并在一年内死亡。
结论 RRSO 术后患者仍可能罹患盆腔恶性肿瘤，且被发现时往往已发展到晚期。因此，RRSO 术后患者的长期监测及管理具有相当的挑战性，值得警示和重视。

PO_593

蒙药白益母草为一种潜在的治疗策略靶向 PI3K/AKT/mTOR 诱导的线粒体凋亡治疗子宫内膜异位症

魏妮[1] 赵荣伟[2*]
1.内蒙古医科大学 2.呼和浩特市妇幼保健院

目的 本研究旨在探讨蒙药白益母草对子宫内膜异位症的治疗作用及其分子机制，重点关注其是否通过靶向 PI3K/AKT/mTOR 通路诱导线粒体凋亡发挥疗效。
方法 通过体外培养人子宫内膜异位症细胞株（hEM15A、ESCs），采用不同浓度的白益母草提取物处理细胞，检测细胞活力（CCK-8 法）、 colony 形成能力、凋亡率（Annexin V-FITC/PI 双染流式细胞术）及细胞周期分布（PI 染色）；同时检测细胞内活性氧（ROS）水平、线粒体膜电位（MMP）变化，并通过 Western blot 分析凋亡相关蛋白（Bax、Bcl-2、细胞色素 c、cleaved caspase-3/9、PARP）的表达。体内实验采用子宫内膜异位症裸鼠模型，经白益母草干预后，观察异位病灶体积及重量变化，检测血清中炎症因子（IL-6、TNF-α）及血管生成因子（VEGF、bFGF）水平，通过 HE 染色进行病理组织学评估，并采用 qRT-PCR 和免疫组化检测病灶组织中 Ki67、E-钙粘蛋白、N-钙粘蛋白的表达。此外，结合蛋白质组学和代谢组学分析，筛选白益母草干预后差异表达的蛋白质和代谢物，重点关注 PI3K/AKT/mTOR 通路相关分子（如 p-PI3K、p-AKT、p-mTOR）及关键代谢通路（如嘧啶代谢）的变化，并通过关联分析探究蛋白-代谢物的相互作用。
结果 白益母草提取物可剂量依赖性抑制子宫内膜异位症细胞增殖，诱导细胞凋亡并将细胞周期阻滞于 G0/G1 期；同时降低 MMP 水平，升高 ROS 含量，上调促凋亡蛋白 Bax、细胞色素 c、cleaved caspase-3/9 及 PARP 的表达，下调抗凋亡蛋白 Bcl-2。体内实验中，白益母草显著缩小异位病灶体积，降低血清炎症及血管生成因子水平，改善病灶组织病理形态，并抑制 PI3K/AKT/mTOR 通路的激活（下调 p-PI3K、p-AKT、p-mTOR 表达）。多组学分析表明，白益母草可逆转异位症模型中异常的蛋白和代谢物水平，其中谷胱甘肽合成酶（GSS）、乳酸脱氢酶 B（LDHB）等蛋白与嘧啶代谢相关代谢物（如胞苷、脱氧胞苷）存在显著关联。
结论 综上，蒙药白益母草可能通过抑制 PI3K/AKT/mTOR 通路激活，触发线粒体凋亡途径，调控炎症、血管生成及代谢紊乱，从而发挥治疗子宫内膜异位症的作用，有望成为临床治疗该疾病的潜在药物。

PO_594

女性生殖系统小圆细胞恶性肿瘤 10 例临床分析

贺媛媛 李佳* 吕艳红 荆茹 赵依芳 刘淑娟
空军军医大学第一附属医院

目的 探讨女性生殖系统小圆细胞恶性肿瘤（SRCT）的临床特点及诊治要点。
方法 回顾性分析 2010 年 1 月至 2025 年 6 月空军军医大学西京医院妇产科医院收治的 10 例术后病理诊断为 SRCT 患者的临床病理资料及随访结果。
结果 10 例患者中位年龄 39 岁（18~64 岁），术前确诊 4 例，术后确诊 6 例。肿瘤累及部位包括子宫腔（2 例）、卵巢（2 例）、子宫颈、腹膜后、阴道直肠隔、大阴唇、盆壁各 1 例，1 例为卵巢、子宫颈、输卵管、大网膜多部位受累。临床症状与部位相关：子宫腔及阴道直肠隔病变以不规则阴道流血为主，宫颈病变可经活检术前确诊，卵巢、腹膜后等深部病变症状隐匿，多因包块检出后手术确诊。病理类型包括原始神经外胚层肿瘤（PNET）/尤文肉瘤 4 例、小细胞神经内分泌癌 1 例、高级别胶质瘤 1 例、原始神经上皮性肿瘤 1 例、未分化/难以分类小圆细胞肉瘤 3 例。5 例行分子检测，发现 EWSR1 基因断裂 3 例、FGFR1 变异 1 例、CIC 扩增 1 例。
结论 女性生殖系统 SRCT 可累及多部位，症状取决于发病部位，诊断依赖手术病理及分子检测，目前治疗方案需结合原发部位个体化制定。

PO_595

子宫血管内腺肌瘤病：一种奇怪的混合病变？

顾伟勇
复旦大学附属妇产科医院

该组 7 名患者的平均年龄为 42.3 岁，与普通静脉内平滑肌瘤病患者年龄相仿。除了静脉内子宫腺肌瘤病，我们在所有病例中都发现了子宫腺肌病和平滑肌瘤，这支持了血管内平滑肌成分来源于相关的肌层病变而非血管壁的假设。在我们的这项研究中，静脉内腺肌瘤病具有与大多数 IVL 病例相似的良性临床表现，随访资料中未发现转移性或复发性疾病。主要鉴别诊断为伴有血管累及的子宫腺肌病、低级别子宫内膜间质肉瘤、伴有平滑肌和腺体分化的子宫内膜间质肉瘤以及伴有淋巴血管侵犯的腺肉瘤。在考虑这些诊断时，尤其是 ESS 和腺肉瘤对患者的处理和预后有不同的影响，应牢记静脉内腺肌瘤病的的可能性。

PO_596

Reversal of Cisplatin Resistance in Ovarian Cancer by Astragalus membranaceus: Integrated Network Pharmacology and Experimental Validation

飞 Wang
Department of Gynecology, Shaanxi Provincial People's Hospital

Objective Ovarian cancer (OC) is a lethal gynecologic malignancy, with cisplatin (DDP) as a key chemotherapeutic agent. However, the development of DDP resistance remains a major challenge in clinical management. *Astragalus membranaceus* (AS), a traditional Chinese medicine, has shown potential in modulating immunity and inflammation. This study aims to explore whether AS can

synergize with DDP to overcome chemoresistance in ovarian cancer, and to elucidate its molecular mechanisms.

Materials and Methods Transcriptomic data from DDP-resistant OC datasets were integrated to identify resistance-related genes. Active compounds and potential targets of AS were retrieved via network pharmacology. Functional enrichment, survival analysis, and immune infiltration analysis were conducted. Core targets were subjected to molecular docking with AS compounds. SKOV3/DDP cells were used in vitro to assess the combination treatment's effects on cell proliferation, migration, apoptosis, and protein expression.

Results By integrating public datasets, 337 genes were identified as consistently associated with DDP resistance. Mapping these genes onto AS-related targets revealed 22 overlapping molecules, suggesting possible intervention points. Functional enrichment analysis indicated that these targets were involved in inflammation (e.g., IL-1, TNF pathways) and cell adhesion, both of which are pivotal to chemoresistance development.

Further, survival and immune infiltration analyses of OC clinical cohorts highlighted nine hub genes—particularly IL1A, IL1B, SERPINE1, ITGA2, and AXL—which were associated with poor prognosis and altered immune microenvironment. These genes were prioritized for downstream molecular docking, where several AS compounds (e.g., formononetin, calycosin) exhibited strong binding affinities (binding energy < -8 kcal/mol), suggesting potential direct modulation.

Building on these predictions, functional experiments in SKOV3/DDP cells revealed that while AS alone exerted limited effects, its combination with DDP significantly suppressed cell proliferation and migration and markedly enhanced apoptosis ($p < 0.01$). Western blotting confirmed the downregulation of IL1A, IL1B, SERPINE1, and AXL upon combination treatment, with compensatory modulation of ITGA2—validating the predicted multi-target regulation and supporting the anti-resistance mechanism.

Conclusion: Our study demonstrates that AS enhances the anti-tumor efficacy of cisplatin by targeting inflammation- and adhesion-related mechanisms involved in DDP resistance. These findings provide a mechanistic basis for integrating traditional medicine into modern chemotherapy regimens and support the clinical potential of AS as a novel adjuvant strategy in overcoming platinum resistance in ovarian cancer.