The 11th Biennial Meeting of Society for Free Radical Research-Asia
Chinese National Conference of Redox Biology and Medicine 2024
第11届亚洲自由基研究国际会议暨2024中国氧化还原生物学与医学大会

SFRR-Asia 2024

October 21-23, 2024, Beijing

Organizers:
Society for Redox Biology and Medicine Branch of Biophysical Society of China (SFRR-China)
中国生物物理学会氧化还原生物学与医学分会
School of Chinese Medicine, The University of Hong Kong
香港大学中医药学院

Co-organizers:
The Material Biology and Intelligent Medicine Branch of Biophysical Society of China
中国生物物理学会材料生物学与智能诊疗技术分会
Shanghai Tissuebank Biotechnology Co.,Ltd
上海荻硕贝肯生物科技有限公司

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Welcome Message



Dear colleagues and friends:

On behalf of the organizing committee, I am honored to welcome you to "The 11th Biennial Meeting of the Society for Free Radical Research-Asia (SFRR-Asia) and the Chinese National Conference of Redox Biology and Medicine 2024" that will be held in Beijing, China from Oct. 21 to 23, 2024.

After two decades of ten past SFRR-Asia biennial meetings, we are marching into a new era to explore redox biology and medicine at precision, mechanistic and in vivo levels with innovative technology and multiple discipline collaborations. The SFRR-Asia 2024 with the theme "The new era of precision redox biology and medicine: from basic research to intervention of aging and diseases" will provide a great opportunity to address the updates on state-of-the-art research in these research fields. There will be a broad spectrum of topics concerning three aspects: Basic research on redox biology and medicine, Redox homeostasis in aging and diseases, Precision redox intervention and health management. For the first time, a special session entitled "Redox Future Perspective Forum" will be set aiming to draw the road map for future redox biology and medicine through open discussion with world-renowned leading scientists and experts. It's also exciting that Young Investigator Award (YIA) and Outstanding Poster/Oral Presentation Awards will be selected.

I sincerely welcome you to join the meeting and hope every participant will benefit from it in exchanging ideas, stimulating collaboration, developing friendship, and experiencing Chinese culture. We gratefully acknowledge the financial support from the SFRR-International, SFRR-Asia, and many sponsors listed in the program book and the conference website (SFRR-Asia2024.com.cn). I would like to thank all previous SFRR-Asia Biennial meeting organizers, Presidents and Secretariats for their outstanding contribution, dedication, and service to our society. My appreciation is extended to the Biophysical Society of China, the conference company and all volunteers for their cooperation and dedication. Finally, I cordially thank all SFRR-China council members and young investigators for their long-term strong support and solidarity.

I hope that the 11th SFRR-Asia will be a remarkable start for the next two decades!

Chang Chen

Chang Chen, PhD
President, SFRR-China
President-Elect, SFRR-Asia

About SFRR-Asia

The Society for Free Radical Research (Asian Region), founded in 1995, is an Asian regional branch of the Society for Free Radical Research International (SFRRI).

Current Officers of SFRR-Asia

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8th: Daniel Tsun-Yee Chiu (Taiwan, China)
9th: Shinya Toyokuni (Japan)
10th: Yang Liu (China)
11th: Yuji Naito (Japan)

Previous Biennial Meetings

The 1st SFRR-Asia Biennial Meeting

Date: November 6-8, 2003

Venue: Cultural Center of Seoul National University, Seoul, South Korea

Organizer: Myung-Hee Chung

The 2nd SFRR-Asia Biennial Meeting

Date: June 24-29, 2005

Venue: Galaxy Hotel, Shanghai, China

Organizer: Baolu Zhao

The 3rd SFRR-Asia Biennial Meeting: Emerging Trends in Free Radical and Antioxidant Research

Date: January 8-11, 2007

Venue: Fariyas Holiday Resort, Lonavala, India

Organizer: R.D. Lele

The 4th SFRR-Asia Biennial Meeting: Chemoprevention and Translational Research

(In conjunction with the 7th COSTAM/SFRR International Workshop)

Date: July 9-12, 2009

Venue: Langkawi Island, Malaysia

Organizer: Kalanithi Nesaretnam

The 5th SFRR-Asia Biennial Meeting

(In conjunction with the 8th Conference of Asian Society for Mitochondrial Research & Medicine and 11th Conference of Japanese Society of Mitochondrial and Medicine)

Date: August 31-September 4, 2011

Venue: Kagoshima Citizens' Culture Hall, Kagoshima, Japan

Organizer: Hideyaki Majima

The 6th SFRR-Asia Biennial Meeting: Oxidative Stress and Mitochondrial Alterations in Ageing and Disease

Date: October 16-19, 2013

Venue: The Second Medicine Building, Chang Gung University, Tao-Yuan, Taiwan, China

Organizer: Daniel Tsun-Yee Chiu

The 7th SFRR-Asia Biennial Meeting: Advanced Oxidative Stress Research for Health and Well-beings

Date: November 29-December 2, 2015

Venue: The Empress Hotel, Chiang Mai, Thailand

Organizers: Maitree Suttajit & Malyn Ungsurungsie

The 8th SFRR-Asia Biennial Meeting: Cross Talk between Free Radicals and Mitochondria in Health and Disease

(In conjunction with the 14th Conference of the Asian Society of Mitochondrial Research & Medicine and Chinese Mitochondrial Society 2017)

Date: September 8-11, 2017

Venue: Xi'an Nanyang Hotel, Xi'an, China

Organizer: Jiangkang Liu

Co-chairs: Yang Liu, Chin-San Liu, and Quan Chen

The 9th SFRR-Asia Biennial Meeting

Date: April 4-7, 2019

Venue: Kyoto International Community House (Kokoka), Kyoto, Japan

Organizer: Yuji Naito

The 10th SFRR-Asia Biennial Meeting: New Paradigm for Research on Oxidative Stress & Inflammation

Date: November 4-6, 2022

Venue: Center for New Drug Development, Seoul National University main campus (Gwanak), Seoul, Korea

Organizer: Young-Joon Surh

The 11th SFRR-Asia Biennial Meeting: New Research Horizons for Redox Biology and Medicine

(In conjunction with Chinese National Conference of Redox Biology and Medicine 2024)

Date: October 21-23, 2024

Venue: Kun Tai Hotel Beijing Wangjing, Beijing, China

Organizer: Chang Chen

Sponsor: Society for Redox Biology and Medicine (Branch of Biophysical Society of China)

Co-sponsor: The Material Biology and Intelligent Medicine (Branch of Biophysical Society of China)

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Liang-Jun Yan (The University of North Texas, USA)

Malyn Ungsurungsie (Silpakorn University, Thailand)

Osamu Handa (Kawasaki Medical School, Japan)

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Salmaan Hussain (Petroliam Nasional Berhad, Malaysia)

Shinya Toyokuni (Nagoya University, Japan)

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Bin Liu	Ke Liu	Yangping Liu	Yanlin Ma
Yulan Qiu	Moshi Song	Bo Tang	Jun Wang
Suhua Wang	Hai Wu	Jianqiang Xu	Li Xu
Xianquan Zhan	Hong Zhang	Yan Zhao	Zhongzheng Zhen
Li Zhong			

General Information

Conference Date

From 8:30 AM on Oct. 21 (Monday) to 22:00 PM on Oct. 23 (Wednesday), 2024.

Registration Open

Registration desk is located at the lobby of Kuntai Hotel and open during the following hours:

Oct. 20 (Sunday) 09:00-21:00;

Oct. 21 (Monday) to Oct. 23 (Wednesday): 8:00-18:00;

Group Photo Session

Date & Time: Oct. 21 (Monday) 10:00-10:20

Venue: The Ballroom

Name Badge

Participants are requested to wear their name badges during all scientific programs and social events. The conference staffs have the right to refuse entry to any session without the proper name badge.

Conference Policy

Please switch your mobile phone off or to the vibration mode in the conference room.

No photography and recording lectures or posters are allowed.

Internet Access

Free wireless internet service will be available in the conference venue. Ask the staff in the registration desk for the login information.

Official Language

The official language of the conference is English.

Weather

The average temperature in Beijing during the conference periods is about 9-19℃

Electricity

P.R.C operates on a 220V supply voltage and 50Hz.

Refreshments & Boxed Lunches

Beverages and light snacks will be provided during the coffee breaks and poster presentation session.

Boxed lunches will be provided for the registered participants.

Social Events

Welcome Reception (All registered participants are invited)

Date & Time: 18:30-20:00 on Oct. 21(Monday)

Venue: The Ballroom, Kuntai Hotel

Gala Dinner (By invitation Only)

Date & Time: 18:00-22:00 on Oct. 22 (Tuesday)

Venue: Hua's Restaurant (Dong Zhi Men)

Address: No.5 Dongzhimen Inner Street, Dongcheng Strict, Beijing.

Banquet:

Closing & Award Ceremony (All registered participants are invited)

Date & Time: 18:00-22:00 on Oct. 23 (Wednesday)

Venue: The Ballroom, Kuntai Hotel

Conference Venue

Kuntai Hotel (Beijing Wangjing)

Phone: (86-10) 84106666

Fax: (86-10) 84106688

Website: http://www.kuntaihotel.com

Address: No 2, Qiyang Road, Chaoyang District, Beijing

Zip code: 100102

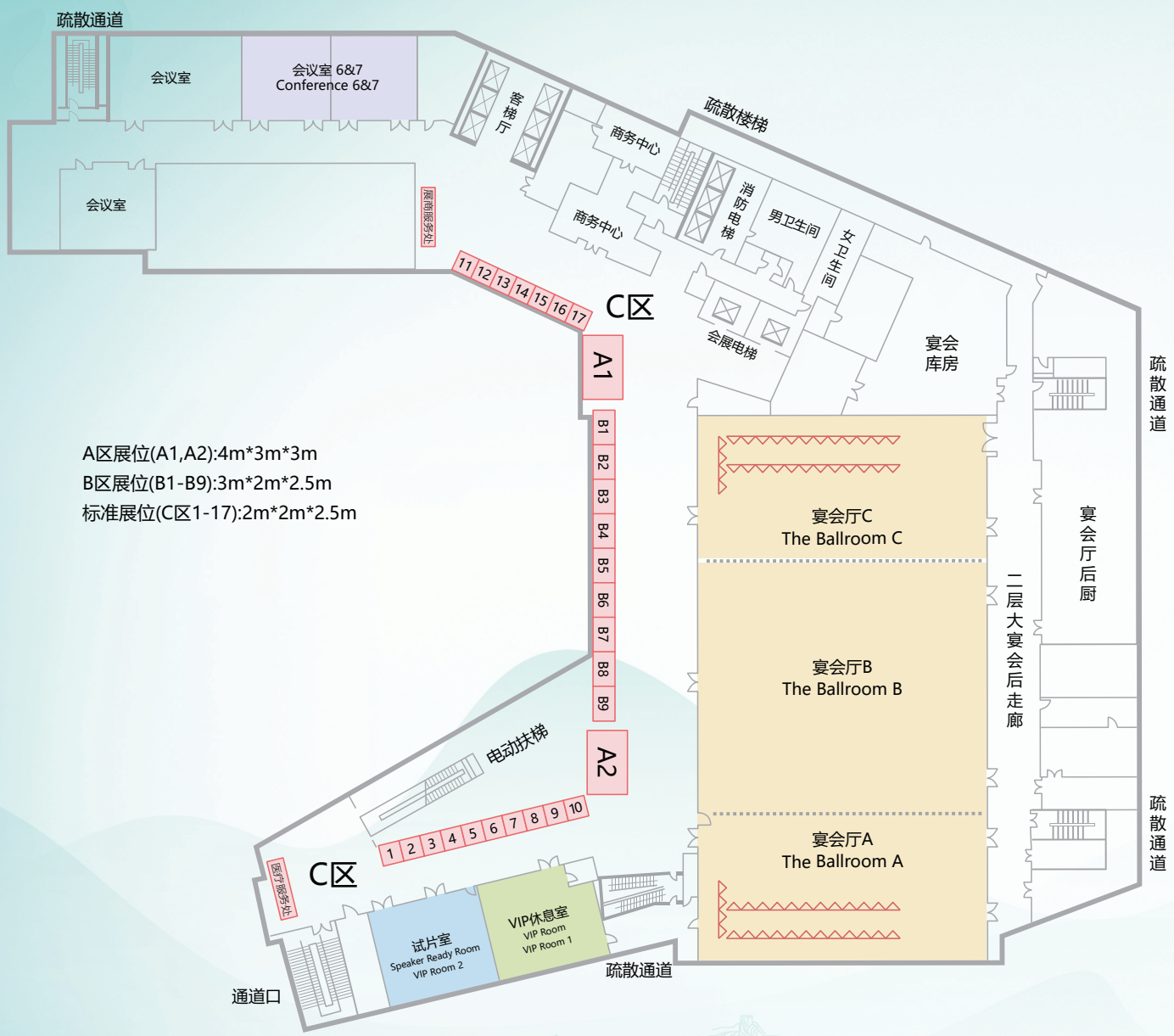
Email: Public@kuntaihotel.com

Map:




Wangjing Station
North Fifth Ring Road
Kuntai Hotel
(Beijing Wangjing)
Subway Line 13
Subway Line 15
Jingmi Road
Airport Expressway
North Fourth Ring Road
Subway Line 10
Subway Line 14

Conference venue floor plan & exhibition booth distribution map



A区展位(A1,A2):4m*3m*3m
B区展位(B1-B9):3m*2m*2.5m
标准展位(C区1-17):2m*2m*2.5m

Sponsors

A区展位

A1 北京同仁堂国药有限公司

Beijing Tong Ren Tang Chinese Medicine Company Limited

A2 上海荻硕贝肯生物科技有限公司

Shanghai Tissuebank Biotechnology Co.,Ltd

B区展位

B1 上海皓元生物医药科技有限公司（MCE 中国）

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Beijing Huawei Zhongyi Technology Co., Ltd

B6 MDPI

B7 《逆境生物学（英文）》编辑部

Editorial Office of Stress Biology

B8 上海漙美医疗科技有限公司

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B9 上海碧云天生物技术股份有限公司

Beyotime Biotech Inc

C区展位

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C3 山东安然纳米实业发展有限公司

Shandong Anran Nanometer Industry Development Co., Ltd

C4 日本生物压力研究振典速合

Japan Biostress Research Promotion Alliance (JBPA)

C5 北京隆福佳生物科技有限公司

Beijing Longfujia Biological Technology Co., Ltd

C6 杭州华安生物技术有限公司

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Bruker (Beijing) Technology Co., Ltd

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Shanghai Kuo Yun Instruments Co., Ltd

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JBKLAB（South Korea）

Timetable

Oct. 20, 2024 Sunday	
Open for Registration	**Open for Registration** Lobby of Kuntai Hotel 09:00-21:00 Oct. 20 08:00-18:00 Oct. 21 08:00-18:00 Oct. 22 08:00-18:00 Oct. 23

Date & Time	Day1 Oct. 21, 2024 Monday			Date & Time	Day2 Oct. 22, 2024 Tuesday			Date & Time	Day3 Oct. 23, 2024 Wednesday		
08:30-09:00	**Opening Ceremony** The Ballroom										
09:00-09:30	**Plenary Lecture-1 (PL-1)** The Ballroom			09:00-09:30	**Plenary Lecture-3 (PL-3)** The Ballroom			09:00-09:30	**Symposium-13 (S13)** The Ballroom A	**Symposium-14 (S14)** The Ballroom B	**Symposium-15 (S15)** The Ballroom C
09:30-10:00	**Plenary Lecture-2 (PL-2)** The Ballroom			09:30-10:00	**Special Lecture** The Ballroom			09:30-10:00			
10:00-10:20	**Group Photo** The Ballroom **Coffee Break** Poster Hall			10:00-10:20	**Coffee Break** Poster Hall			10:00-10:20	**Coffee Break** Poster Hall		
10:20-12:00	**Symposium-1 (S1)** The Ballroom A	**Symposium-2 (S2)** The Ballroom B	**Symposium-3 (S3)** The Ballroom C	10:20-12:00	**Symposium-7 (S7)** The Ballroom A	**Symposium-8 (S8)** The Ballroom B	**Symposium-9 (S9)** The Ballroom C	10:20-12:00	**Flash Talk-1 (FT-1)** The Ballroom A	**Flash Talk-2 (FT-2)** The Ballroom B	**Flash Talk-3 (FT-3)** The Ballroom C
12:00-12:30	**12:00-13:30 SFRR-Asia Business Meeting** Conference 2-6&7 on the second floor of Kuntai Hotel	**Lunch** The Ballroom		12:00-12:30	**Lunch** The Ballroom			12:00-12:30	**12:00-13:30 SFRR-China Business Meeting** Conference 2-6&7 on the second floor of Kuntai Hotel	**Lunch** The Ballroom	
12:30-13:30		**Poster Presentation** The Ballroom A		12:30-13:30	**Meet the Editors** The Ballroom			12:30-13:30		**Poster Presentation** The Ballroom C	
13:30-15:10	**Symposium-4 (S4)** The Ballroom A	**Symposium-5 (S5)** The Ballroom B	**Symposium-6 (S6)** The Ballroom C	13:30-15:10	**Symposium-10 (S10)** The Ballroom A	**Symposium-11 (S11)** The Ballroom B	**Symposium-12 (S12)** The Ballroom C	13:30-14:00	**Plenary Lecture-4 (PL-4)** The Ballroom		
								14:00-14:30	**Plenary Lecture-5 (PL-5)** The Ballroom		
								14:30-14:50	**Coffee Break** Poster Hall		
15:10-15:30	**Coffee Break** Poster Hall			15:10-15:30	**Coffee Break** Poster Hall			14:50-17:00	**Redox Future Perspective Forum** The Ballroom		
15:30-17:30	**Symposium-YIO-1 (Y-1)** The Ballroom A	**Symposium-YIO-2 (Y-2)** The Ballroom B	**Symposium-YIO-3 (Y-3)** The Ballroom C	15:30-17:30	**Symposium-YIO-4 (Y-4)** The Ballroom A	**Symposium-YIO-5 (Y-5)** The Ballroom B	**Symposium-YIO-6 (Y-6)** The Ballroom C				
17:30-18:30	**Exhibition Sharing Session** The Ballroom C			18:00-22:00	**Gala dinner (invited only)**			17:00-18:00	**Exhibition Exchange** Exhibition Area		
18:30-20:00	**Welcome Reception** The Ballroom							18:00-22:00	**Closing & Award Ceremony Banquet** The Ballroom		
20:00-22:00	**Early Career Researchers** The Ballroom C										

Scientific Program

08:30-09:00	**Opening Ceremony**	The Ballroom
Chair: Gunangjun Nie 聂广军 (National Center for Nanoscience and Technology, China)		
	Plenary Lecture -1 (PL-1)	The Ballroom
Chair: Chang Chen 陈畅 (Institute of Biophysics, CAS, China)		
09:00-09:30	Giovanni E Mann (King's College London, UK)	
	Roadblock for clinical translation: importance of physiological oxygen levels for high throughput screening of redox	
	Plenary Lecture -2 (PL-2)	The Ballroom
Chair: Malyn Ungsurungsie (Silpakorn University, Thailand)		
09:30-10:00	Young-Joon Surh (Seoul National University, Korea)	
	Warburg Effect Revisited: Role of NRF2 in Pseudohypoxic Stabilization of HIF-1α	
10:00-10:20	**Group Photo** **Coffee Break**	The Ballroom Poster Hall
	Symposium-1 (S1) **Redox signaling in organelles/cell fate/development/reproduction**	The Ballroom A
Chair: Taotao Wei 卫涛涛 (Institute of Biophysics, CAS, China) Hun Taeg Chung (Daegu Haany University, Korea)		
10:20-10:40	Ming-Yi Bai 白明义 (Shandong University, China)	
	H_2O_2 promotes stomatal development and opening through regulating SnRK1	

Oct. 21

10:40-11:00	Wenhua Zheng 郑文华 (University of MacauUniversity of Macau, China)
	Artemisinin attenuates astrocyte overactivation by inhibiting IRE1 phosphorylation and the downstream NF-κB pathway in Alzheimer's disease
11:00-11:20	Takaaki Akaike (Tohoku University, Japan)
	Redox Signal Regulation by Supersulfides
11:20-11:40	Hun Taeg Chung (Daegu Haany University, Korea)
	Carbon monoxide sensitizes cancer cell to erastin-induced ferroptosis via ROS-PERK-ATF4
11:40-12:00	Erich Gnaiger (Beijing Huawei Zhongyi Technology Co. Ltd)
	Oxidative phosphorylation, H_2O_2 production, mitochondrial membrane potential, coenzyme Q redox state, and calcium uptake: from tissue normoxia to deep hypoxia

Oct. 21

Symposium-2 (S2) **Redox and aging①"Targeting Redox and Mitochondria to delay aging and prevent age-related diseases"Forum**	The Ballroom B
Chair: Ke Liu 刘科 (Sichuan University, China) Malcolm Jackson (The University of Liverpool, UK)	
10:20-10:40	Min-Xin Guan 管敏鑫 (Zhejiang University, China)
	Vitamin A treatment rescues retinal cell-specific deficiencies caused by Leber's hereditary optic neuropathy-linked mtDNA mutation
10:40-11:00	Yuguang Shi 史裕光 (Barshop Aging Institute, UT-Health, San Antonio)
	Cardiolipin Remodeling byALCAT1 Controls the Mitochondrial Free Radical Clock
11:00-11:20	Zhiyin Song 宋质银 (Huazhong University of Science and Technology, China)
	A new mode of mitochondria-lysosome contacts under hypoxia
11:20-11:40	Yidong Bai (University of Texas Health San Antonio, USA)
	Mitochondrial electron transfer chain (ETC) in aging and longevity

11:40-12:00	Malcolm Jackson (The University of Liverpool, UK)
	Dysregulation of hydrogen peroxide-mediated responses to contractile activity in skeletal muscle loss associated with ageing

Symposium-3 (S3) **Redox and obesity, vascular function and metabolism**	The Ballroom C
Chair: Zhongbing Lu 陆忠兵 (University of Chinese Academy of Sciences, China) Juan Sastre (University of Valencia, Spain)	
10:20-10:40	Xiao-Wei Chen 陈晓伟 (Peking University, China)
	ER oxi-lipidosis drives MASH pathogenesis
10:40-11:00	Changtao Jiang 姜长涛 (Peking University, China)
	Gut microbial enzymes: new targets for intervention in metabolic diseases
11:00-11:20	Junli Liu 刘军力 (Shanghai Jiao Tong University, China)
	Obstructive sleep apnea syndrome and hepatic lipid metabolism disorders
11:20-11:40	Juan Sastre (University of Valencia, Spain)
	Redox signaling in acute inflammation
11:40-12:00	Michael Jonathan Davies (University of Copenhagen, Denmark)
	Oxidation and enzyme-mediated changes to the artery wall in cardiovascular disease

12:00-13:30	**SFRR-Asia Business Meeting** Lunch Provited	Conference 2-6&7 on the second floor of Kuntai Hotel
12:00-12:30	**Lunch**	The Ballroom

12:30-13:30	**Poster Presentation**	The Ballroom A

	Symposium-4 (S4) **New approach for precision redox research**	The Ballroom A
	Chair:Xiangliang Yang 杨祥良 (Huazhong University of Science and Technology, China) Kwang Pyo Kim (Kyung Hee University, Korea)	
13:30-13:50	Bo Tang 唐波 (Laoshan Laboratory, China)	
	Fluorescence Imaging for the Progression of Oxidative Stress-Related Diseases	
13:50-14:10	Yonggang Yao 姚永刚 (Kunming Institute of Zoology, CAS, China)	
	Primate Phenotype and Genetic Analyses – From Basic Research to Clinical Applications	
14:10-14:30	Huiru Tang 唐惠儒 (Fudan University, China)	
	Quantitative metabolomics for redox biology and medicine	
14:30-14:50	Youjun Yang 杨有军 (East China University of Science and Technology, China)	
	Near-infrared xanthene dyes and *in vivo* ROS sensing	
14:50-15:10	Young-Sam Keum (Dongguk University, Korea)	
	Leucine 305 and 309 residues contribute to the formation of two human NRF2 bands in SDS-PAGE	

	Symposium-5 (S5) **Discovery of new molecules in redox network**	The Ballroom B
	Chair: Qiang Zhao 赵强 (Nankai University, China) Ken-ichi Yamada (Kyushu University, Japan)	
13:30-13:50	Jingbo Pi 皮静波 (China Medical University, China)	
	NRF1 and NRF2 coordinate osteoclastogenesis and bone remodeling via ROS-dependent and independent mechanisms	

13:50-14:10	Akira Nakai (Yamaguchi University Graduate School of Medicine, Japan)
	HSF1 regulates nuclear/cytoplasmic and mitochondrial proteotoxic stress responses
14:10-14:30	Bo Xie 谢波 (Shanghai Tissuebank Biotechnology Co.,Ltd (TAB))
	Grasip the void of Redox——the new relic of Tissue Bank
14:30-14:50	Ken-ichi Yamada (Kyushu University, Japan)
	Structural library and visualization of endogenously oxidized lipids
14:50-15:10	Youngjoo Kwon (Ewha Womans University, Korea)
	Regulation of protein-protein interactions as a new paradigm in drug discovery: Targeting the oncogenic role of E74 Like ETS transcription factor 3 (ELF3) through modulation of its protein protein interaction

Symposium-6 (S6)
Redox modification of biomacromolecules
The Ballroom C

Chair: Zhonghong Gao 高中洪 (Huazhong University of Science and Technology, China)
Koji Uchida (The University of Tokyo, Japan)

13:30-13:50	Lee Jia 贾力 (Minjiang University, China)
	Biology and pharmaceutical development of S-nitrosylation
13:50-14:10	Huiyong Yin 尹慧勇 (City University of Hong Kong, China)
	Lipid Peroxidation and Cardiovascular Disease
14:10-14:30	Moran Benhar (Technion Israel Institute of Technology, Israel)
	The dynamic thiol redox proteome of macrophages and its role in the response to oxidative-inflammatory stress
14:30-14:50	Koji Uchida (The University of Tokyo, Japan)
	Immune memory against toxic aldehydes

14:50-15:10	Xingen Lei (Cornell University, USA)
	A protein protein interaction between SOD1 and YWHAZ and YWHAE

15:10-15:30	**Coffee Break**	Poster Hall

	Symposium-YIO-1 (Y-1) **Redox modification of biomacromolecules** **Redox and obesity, vascular function and metabolism**	The Ballroom A
	Chair: Li Xu 徐力 (Jilin University, China) Hyoung Kyu Kim (Inje University, Korea)	
15:30-15:45	Lei Chen 陈雷 (Peking University, China)	
	Activation mechanism of phagocyte NADPH oxidase	
15:45-16:00	Xueqing Ba 巴雪青 (Northeast Normal University, China)	
	OGG1 promotes iTreg differentiation and alleviates mouse IBD by facilitating Foxp3 transcriptional activation	
16:00-16:15	Ming Lu 鲁明 (Shanghai Institute of Nutrition and Health, CAS, China)	
	A lactate-lipid peroxidation-acetate metabolic axis between tumor-associated macrophages and cancer cells fuels hepatocellular carcinoma metastasis	
16:15-16:30	Shi Yuheng 石玉衡 (Fudan university, China)	
	STING: A Potential Target for Suppressing the Development of Clonal Hematopoiesis and Leukemia	
16:30-16:45	Wen Wang 王雯 (Capital Medical University, China)	
	Disorder of nitration/S-sulfhydration participates in hyperhomocysteinemia progression and liver damage	
16:45-17:00	Bin Liu 刘斌 (Shantou University, China)	
	Endothelium-dependent contraction, NO and cardiovascular disorders in the absence of prostacyclin synthesis	

17:00-17:15	Hou-Zao Chen 陈厚早 (Chinese Academy of Medical Sciences & Peking Union Medical College, China)
	SIRT2 governs a cytoplasm-mitochondrial signal to repress mitochondrial ROS and vascular ageing
17:15-17:30	Hyoung Kyu Kim (Inje University, Korea)
	Tetrahydrobiopterin is a Promising Target of Diabetic Cardiomyopathy via Restoring Mitochondria Function

Symposium-YIO-2 (Y-2)
Redox and cancer, infection and immunity
Redox and environmental challenge
The Ballroom B

Chair: Jianghong Man 满江红 (National Center of Biomedical Analysis, China)
Chung S. Yang (Rutgers, The State University of New Jersey, USA)

15:30-15:45	Jianqiang Xu 许建强 (Dalian University of Technology, China)
	Two sesquiterpene lactones inhibit TXNRD1 and induce endoplasmic reticulum stress in cancer cells
15:45-16:00	Xiu-Ping Chen 陈修平 (University of Macau, China)
	Novel anticancer drug discovery strategies by targeting NQO1
16:00-16:15	Kelong Fan 范克龙 (Institute of Biophysics, CAS, China)
	Structure-Activity Relationship and Biomedical Applications of Nanozymes
16:15-16:30	Cui-xia Di 狄翠霞 (Institute of Modern Physics, CAS, China)
	Electron FLASH Irradiation Ameliorates Radiation-induced Developmental and Neurological Toxicity in Zebrafish Model
16:30-16:45	Tomohiro Sawa (Kumamoto University, Japan)
	Supersulfide regulation of innate immune responses
16:45-17:00	Tianli Zhang (Akita University, Japan)
	Supersulfides regulate NLRP3 inflammasome activation through sensing homeostasis

17:00-17:15	Guoping Yin 尹国平 (Beijing Tsinghua Changgung Hospital, China)
	OSA induced multiple-system damage via oxidative stress
17:15-17:30	Jian-gang Long 龙建纲 (Xi'an Jiaotong University, China)
	HTHB: A Potential Therapeutic Agent for Cognitive Impairment and Inflammation

Symposium-YIO-3 (Y-3) Redox and neural function and mental health	The Ballroom C
Chair: Wenli Li李文丽 (Air Force Medical University, China) Changyang Gong 巩长旸 (Sichuan University, China)	
15:30-15:45	Jun Wang 王军 (Hubei University of Technology, China)
	Redox biomarkers for prognosis of infectious diseases
15:45-16:00	Yuming Zhao 肇玉明 (Capital Medical University, China)
	DDAH1, a key neuroprotective player, promotes neurogenesis and neural repair after cerebral ischemia insults
16:00-16:15	Danqian Liu 刘丹倩 (Institute of Neuroscience, CAS, China)
	A physiological role of H_2O_2 in sleep homeostasis
16:15-16:30	Ishii Tetsuro (University of Tsukuba, Japan)
	NRF2 translocation from dendrites to nucleus in glutamatergic pyramidal neurons induced by uncoupling of post-synaptic neuronal nitric oxide synthase via calcium influx through NMDAR
16:30-16:45	Meiling Wu 吴美玲 (The University of Hong Kong, China)
	Peroxynitrite reduces Treg cell expansion and function by mediating IL-2R nitration and aggravates multiple sclerosis pathogenesis
16:45-17:00	Xianhua Wang 王显花 (Peking University, China)
	ROMO1 shields the mitochondrial cysteinome from oxidations in diseases and aging

17:00-17:15	Xu Zhang 张栩 (Tianjin Medical University, China)
	Alox15/15-HpETE Aggravates Myocardial Ischemia-Reperfusion Injury by Promoting Cardiomyocyte Ferroptosis
17:15-17:30	Yan Zheng 郑焱 (The First Affiliated Hospital of Xi'an Jiaotong University, China)
	The Mechanisms of Plasma-Activated Solutions in Treating Atopic Dermatitis

17:30-18:30	**Exhibition Sharing Session**	The Ballroom C

18:30-20:00	**Welcome Reception**	The Ballroom

Chair: Jian Kang Liu 刘健康 (University of Health and Rehabilitation Sciences/Xi'an Jiaotong University, China)

20:00-22:00	**Early Career Researcher**	The Ballroom C

	Plenary Lecture-3 (PL-3)	The Ballroom
	Chair: Myung-Hee Chung (Seoul National University, Korea)	
09:00-09:30	Yuji Naito (Kyoto Prefectural University of Medicine, Japan)	
	Gut frailty: its concept and the role of dietary fiber	
	Special Lecture	The Ballroom
	Chair: Yang Liu 刘扬 (Institute of Chemistry, CAS, China)	
09:30-10:00	Chang Chen 陈畅 (Institute of Biophysics, CAS, China)	
	Carry forward the cause and forge ahead into the future —— Memory of past 20 years SFRR-Asia biennial meetings	
10:00-10:20	**Coffee Break**	Poster Hall
	Symposium-7 (S7) **Redox signaling in organelles/cell fate/development/reproduction**	The Ballroom A
	Chair: Dongyun Shi 施冬云 (Shanghai Medical College of Fudan University,Shanghai, China) Francisco Rafael Martins Laurindo (University of São Paulo, Brazil)	
10:20-10:40	Wenjun Ding 丁文军 (University of Chinese Academy of Sciences, China)	
	Airborne PM2.5-induced oxidative stress aggravates neurotoxicity in olfactory bulb	
10:40-11:00	Yanlin Ma 马燕琳 (The First Affiliated Hospital of Hainan Medical College, China)	
	Deciphering Umbilical Cord Blood Hematopoietic Stem/Progenitor Cell Alterations in Alpha-Thalassemia Using Single-Cell Transcriptomics	
11:00-11:20	Liron Bar-Peled (Harvard Medical School Department of Medicine, USA)	
	Identification of Druggable and Redox Vulnerabilities in Cancer	

11:20-11:40	Francisco Rafael Martins Laurindo (University of São Paulo, Brazil)
	An endoplasmic reticulum-based model of intercellular redox communication.
11:40-12:00	Kyung-Soo Chun (Keimyung University, Korea)
	Apoptotic Effect of Terfenadine, a Histamine H1 Receptor Antagonist, and Terfenadine-loaded Human Serum Albumin Nanoparticles in Colorectal Cancer and Glioblastoma Cells

Symposium-8 (S8)
Redox and aging ②"Targeting Redox and Mitochondria to delay aging and prevent age-related diseases"Forum — The Ballroom B

Chair: Young Zhang 张勇 (Tianjin University of Sport, Tianjin, China)
Liang-Jun Yan (University of North Texas Health Science Center, Fort Worth, TX, USA)

10:20-10:40	Han-Ming Shen 沈汉明 (University of Macau, China)
	Redox regulation of mitophagy by targeting PINK1
10:40-11:00	Xingguo Liu 刘兴国 (Guangzhou Biomedicine and Health Institute, CAS, China)
	NAD^+ dependent UPRmt activation underlies intestinal aging caused by mitochondrial DNA mutations
11:00-11:20	Quan Chen 陈佺 (Institute of Zoology, Chinese Academy of Sciences, China)
	Truncated oxidized phospholipids mediate synchronized ferroptosis and contribute to acute kidney injury
11:20-11:40	Liang-Jun Yan (University of North Texas Health Science Center, Fort Worth, TX, USA)
	NAD^+-dependent enzymes in health and disease: Our key findings on NADH-ubiquinone oxidoreductase in diabetic pancreas
11:40-11:55	Kanglin Wang 王康林 (Knature Bio-pharma Co., Ltd.)
	Mitochondrial drug NAD^+ anti-aging strategy

	Symposium-9 (S9) **Redox and cancer, infection and immunity**	The Ballroom C
	Chair: Pingping Shen 沈萍萍 (Nanjing University, China) Junji Yodoi (Kyoto University, Japan)	
10:20-10:40	Zigang Dong 董子钢 (Zhengzhou University, China)	
	The role of redox metabolism in drug resistance of cancer therapy	
10:40-11:00	Yoshiro Saito (Tohoku University, Japan)	
	Redox biology regulated by selenoproteins- significance for biological defense and its relation to cancer	
11:00-11:20	Martin Rottenberg (Karolinska Institutet, Sweden)	
	Immune Mechanisms and Oxidative Stress Underlying the Interaction of Tuberculosis and Diabetes	
11:20-11:40	Junji Yodoi (Kyoto University, Japan)	
	TRX Thioredoxin redox regulator of inflammasome: Redoxisome Concept	
11:40-12:00	Mee-Hyun Lee (Dongshin University, Korea)	
	A Systemic Effects of Herbal Medicine on Colon Diseases	
12:00-12:30	**Lunch**	The Ballroom
12:30-13:30	**Meet the Editors**	The Ballroom
	Chair: Huiyong Yin 尹慧勇 (City University of Hong Kong, China)	

Symposium-10 (S10) Redox and environmental challenge	The Ballroom A
Chair: Libo Du 杜立波 (Institute of Chemistry, CAS, China) Jin-Won Hyun (Jeju National University, Korea)	
13:30-13:50	Yinghui Li 李莹辉 (China Astronaut Research and Training Center, China)
	Research progress and thinking of space medicine omics
13:50-14:10	Qi Xie 谢旗 (Institute of Genetics and Developmental Biology, CAS, China)
	Gγ regulates PIP2 phosphorylation in ROS distribution to affect crop tolerant to alkaline stress
14:10-14:30	Jin-Won Hyun (Jeju National University, Korea)
	Particulate matter and reactive oxygen species
14:30-14:50	Jung-Hwan Kim (Gyeongsang National University, Korea)
	TCDD-induced Lysosomal SLC46A3 modulates hepatic cytosolic copper homeostasis resulting in triglyceride accumulation
14:50-15:10	Dae Young Kwon (Korea Food Research Institute, Korea)
	Redox History of Earth, and Life of Organisms and Foods Crops

Symposium-11 (S11) Traditional Medicine Prophylaxis-Therapeutics and Redox Balance (Sponsored by The University of Hong Kong & Beijing Tong Ren Tang Chinese Medicine Co. Ltd.)	The Ballroom B
Chair: Simon Ming-Yuen LEE 李铭源 (The Hong Kong Polytechnic University, Hong Kong, China) Motohiro Nishida (Kyushu University, Japan)	
13:30-13:50	Jing-yan Han 韩晶岩 (Peking University, China)
	Traditional Chinese medicine ameliorates cardiac and cerebral microvascular injury through regulating mitochondrial respiratory chain

13:50-14:10	Jiangang Shen 沈剑刚 (The University of Hong Kong, China)
	Niuhuang Qingxin Wan is a promising anti-depressive agent to attenuate chronic stress induced depressive- and anxiety-like behaviors through promoting hippocampal neurogenesis and modulating TrkB/ERK/CREB signaling pathway
14:10-14:30	Xinli Li 李新立 (The First Affiliated Hospital of Nanjing Medical University, China)
	Qiliqiangxin in the Treatment of Heart Failure with Reduced Ejection Fraction ------ Research Progress
14:30-14:50	Motohiro Nishida (Kyushu University, Japan)
	Cardiac stress resistance regulated by sulfur metabolism
14:50-15:10	Yibin Feng 冯奕斌 (The University of Hong Kong, China)
	The role of oxidative stress and antioxidants in liver disease therapy

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Symposium-12 (S12) The Ballroom C

Natural products and nutrition in anti-aging and health management

(Sponsored by The University of Hong Kong & Beijing Tong Ren Tang Chinese Medicine Co. Ltd.)

Chair: Bo Zhou 周波 (Lanzhou University, China)
Ae-son Om (Hanyang university, Korea)

13:30-13:50	Lina Qu 曲丽娜 (China Astronaut Research and Training Center, China)
	Research on Space Biological Rhythm Intervention Strategies Based on Redox Regulation
13:50-14:10	Qinghui Ai 艾庆辉 (Ocean University of China, China)
	Phosphatidylethanolamine alleviates OX-LDL-induced macrophage inflammation by upregulating autophagy and inhibiting NLRP1 inflammasome activation
14:10-14:30	Andrew Bulmer (Griffith University, Australia)
	Translation of bilirubin's redox potential to preventative and therapeutic medicine – use of models, and the development of therapies

14:30-14:50	Hye-Kyung Na (Sungshin Women's University, Korea)
	A Catechol Isoquinoline Salsolinol Induces Cell Death of Human Liver Cancer Cells by Regulating the STAT1/3 Signaling
14:50-15:10	Yun-Sil Lee (Ewha Womans University, Korea)
	From Target Identification to Early-Stage Therapeutic Discovery: Leveraging In Vivo Preclinical Models

15:10-15:30	**Coffee Break**	Poster Hall

Symposium-YIO-4 (Y-4)
New approach for precision redox research
Intelligence materials for precision redox intervention

The Ballroom A

Chair: Xianquan Zhan 詹显全 (Cancer Hospital and Institute, Shandong First Medical University, China)
Kenji Sato (Kyoto University, Japan)

15:30-15:45	Ling Fu 付玲 (Academy of Military Medical Sciences, China)
	Chemical proteomics reveals mechanisms of bacterial response to ROS mediated by antibiotics
15:45-16:00	Yuan Guo 郭媛 (Northwest University, China)
	Molecule-Guided Precise Identification and Intervention of Senescence
16:00-16:15	Yangping Liu 刘阳平 (Tianjin Medical University, China)
	Simultaneous Quantitation of Persulfides, Biothiols and Hydrogen Sulfide through Efficient Sulfur Exchange Reaction with Trityl Spin Probes
16:15-16:30	Ai-Hui Tang 唐爱辉 (University of Science and Technology of China, China)
	Spatial Transcriptome Profiling of a Huntington's Disease Mouse Brain with BASSFISH

16:30-16:45	Kenji Sato (Kyoto University, Japan)
	Generation of short chain aldehydes and increase of oxidative stress in mice by intake of fructose
16:45-17:00	Yue Yuan 袁月 (University of Science and Technology of China, China)
	GSH-induced in situ peptide self-assembly for precise tumor imaging and ROS-based therapy
17:00-17:15	Yang Li 李洋 (Shenzhen Institute of Advanced Technology, CAS, China)
	Molecular targeting nano drug candidates
17:15-17:30	Lizeng Gao 高利增 (Institute of Biophysics, CAS, China)
	Nanozybiotics: Advancing Antimicrobial Strategies Through Biomimetic Mechanisms

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Symposium-YIO-5 (Y-5)
Discovery of new molecules in redox network
Natural products and nutrition in anti-aging and health management

The Ballroom B

Chair: Jun Lu 陆军 (Southwest University, China)
Ock Jin Park (Hanyang university, Korea)

15:30-15:45	Zhi-Yong Mao 毛志勇 (Tongji University, China)
	Targeting DNA repair to extend lifespan
15:45-16:00	Jinzhi Lu 鲁锦志 (The First Affiliated Hospital, Yangtze University, China)
	Redox-Regulated Iron Metabolism and Ferroptosis in Ovarian Cancer: Molecular Insights and Therapeutic Opportunities
16:00-16:15	Moshi Song 宋默识 (Institute of Zoology, CAS, China)
	Restored PGAM5-mediated Oxeiptosis Eliminates ROShigh Cardiomyocytes and Improves Cardiac Function during Cardiac Aging

16:15-16:30	Jin Li 李瑾 (Beijing Hospital, China)
	Exploring the Neuroinflammatory Pathways of 8-oxoGTP and Their Effects on Cognitive Decline
16:30-16:45	Jin Meng 孟劲 (Capital Medical University, China)
	ATF-4 and hydrogen sulfide signaling mediate longevity in response to inhibition of translation or mTORC1
16:45-17:00	Guozhen Cui 崔国祯 (Zunyi Medical University, China)
	Network Medicine landscape on the Health-Enhancing Properties of Natural Antioxidants
17:00-17:15	Jing Qu 曲静 (Institute of Zoology, CAS, China)
	Cellular Senescence and Rejuvenation
17:15-17:30	Chalermpong Saenjum (Chiangmai University, Thailand)
	A hybrid sweet potato (Maejo 341) mitigates LPS-induced inflammation and RANKL-induced osteoporosis by regulating ROS-mediated pathways

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Symposium-YIO-6 (Y-6) Redox signaling in organelles/cell fate/development/reproduction	The Ballroom C
Chair: Zhangjian Huang 黄张建 (China Pharmaceutical University, China) Youngtae Jeong (Daegu Gyeongbuk Institute of Science and Technology, Korea)	
15:30-15:45	Jie He 何杰 (Institute of Neuroscience, CAS, China)
	cxcl18b-defined transitional state-specific nitric oxide signaling drives injury-induced Müller Glia proliferation in the zebrafish retina
15:45-16:00	Peng Huang 黄鹏 (Shenzhen University, China)
	Transforming nutrition into reactive oxygen species for tumor treatment

16:00-16:15	Weihua Yu 于卫华 (The Fourth Military Medical University, China)
	Mitochondrial dynamics and redox balance control macrophage cell fate
16:15-16:30	Ye Tian 田烨 (Institute of Genetics and Developmental Biology, CAS, China)
	Mitochondrial Superoxide Stress Response: Implications for Aging and Health
16:30-16:45	Lei Wang 王磊 (Institute of Biophysics, CAS, China)
	The ER redoxtasis: from basic research to the intervention of aging and diseases
16:45-17:00	Chao Li 李超 (East China Normal University, China)
	Regulation of plant development by peptides–receptor kinases–ROS signaling
17:00-17:15	Youngtae Jeong (Daegu Gyeongbuk Institute of Science and Technology, Korea)
	Reciprocal role of the Keap1-Nrf2 pathway in the self-renewal and differentiation of airway stem cells and tongue stem cells
17:15-17:30	Yong Yeon Cho (Catholic University, Korea)
	Involvement of UVB/ROS-mediated signaling pathway in karyoptotic cell death

18:00-22:00	**Gala dinner (invited only)** Meet at the lobby of Kuntai Hotel at 6 PM

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Symposium-13 (S13) Redox and neural function & mental health	The Ballroom A
Chair: Lin Mei 梅林 (Chinese Academy of Medical Sciences & Peking Union Medical College, China) Lin Mantell (St. John's University, USA)	
09:00-09:20	Chuanzhu Yan 焉传祝 (Qilu Hospital of Shandong University, China)
	Flavin adenine dinucleotide metabolism and related neuromuscular disorders
09:20-09:40	Ping Li 李平 (Shandong Normal University, China)
	In-situ Fluorescence Imaging of Brain Disease-associated Bioactive Molecules
09:40-10:00	Lin Mantell (St. John's University, USA)
	Mechanisms of α7 nicotinic acetylcholine receptor in modulating inflammatory lung injury and infection

Symposium-14 (S14) Intelligence materials for precision redox intervention	The Ballroom B
Chair: Fangyuan Li (Songjiang Hospital, Shanghai Jiao Tong University School of Medicine, China) Yong Sang Song (Seoul National University, Korea)	
09:00-09:20	Guangjun Nie 聂广军 (National Center for Nanoscience and Technology, China)
	Regulation and restoration of microenvironment homeostasis of intestinal diseases based on nanotechnology
09:20-09:40	Jun Zhou 周军 (Huazhong University of Science and Technology, China)
	Molecular mechanisms of selenium intervention in metabolic diseases through regulation of redox homeostasis
09:40-10:00	Yong Sang Song (Seoul National University, Korea)
	Next-generation RNA Sequencing-based Deep-learning Model to Predict Chemoresistance in High-grade Serous Ovarian Carcinoma

Symposium-15 (S15) Lifestyle and redox regulation	The Ballroom C
Chair: Cheng-Gang Zou 邹成钢 (Yunnan University, China) Osamu HANDA (Kawasaki Medical School, Japan)	

09:00-09:20	Tong-Jin Zhao 赵同金 (Fudan University, Shanghai, China)
	Surplus fatty acid synthesis increases oxidative stress in adipocytes and lnduces lipodystrophy
09:20-09:40	Hao Wu 吴昊 (Capital University of Physical Education and Sports, China)
	Effects of Hyperbaric Oxygen Intervention on Oxidative Stress in the Body after High-Intensity Interval Training
09:40-10:00	Osamu HANDA (Kawasaki Medical School, Japan)
	The role of ileal mucosa -associated microbiota in the patients with Crohn's disease

10:00-10:20	**Coffee Break**	Poster Hall

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	Flash Talk-1 (FT-1)	The Ballroom A

Chair: Suhua Wang 王素华 (Guangdong University of Petrochemical Technology, China)

10:20-10:25	Kishimoto Ayuta (Shibaura Institute of Technology, Japan)
	Detection and evaluation of novel oxidizing substances in sodium hypochlorite using Trolox
10:25-10:30	Ujihara Miyu (Kyoto University, Japan)
	High sensitive LC-MS/MS method for determining malondialdehyde in biological sample using thiobarbituric derivatization
10:30-10:35	Yan Wang 王炎 (Tianjin Medical University, China)
	A Chelator-linked Trityl Probe Enabling Highly Specific, Sensitive and Quantitative Detection of Cu(I) by EPR Spectroscopy
10:35-10:40	Wong Nai-Kei (Shantou University Medical College, China)
	Deciphering the RSS code in cellular senescence
10:40-10:45	Zhijuan Hu 胡志娟 (Guangzhou Institute of Biomedicine and Health, CAS, China)
	A novel protein CYTB-187AA encoded by the mitochondrial gene CYTB modulates mammalian early development

Chair: Ju Cui 崔菊 (Country Beijing Institute of Geriatrics, National Health Commission, China)

10:45-10:50	Zongmin Li 李宗敏 (Peking University First Hospital, China)
	Elucidating the reducibility of sulfur dioxide on cysteine proteomes
10:50-10:55	Mi Xinya (Kyushu University, Japan)
	TRPC6-mediated Zn^{2+} influx improves heart failure through supersulfide formation
10:55-11:00	Xinhua Qiao 乔新华 (Institute of Biophysics, CAS, China)
	Exploring the collaboration of redox and autophagy systems based on a genome-wide new redox genes screening
11:00-11:05	Chenlin Su (Kyushu University, Japan)
	TRPC6-mediated Zn^{2+} influx mitigates cardiac fibrosis through maintaining redox homeostasis
11:05-11:10	Jiao Meng 孟姣 (Institute of Biophysics, CAS, China)
	A novel redox gene atad-3 identified by whole genome RNAi screening in Caenorhabditis elegans

Chair: Chalermpong Saenjum (Chiangmai University, Thailand)

11:10-11:15	Yong Wang 王勇 (Ocean University of China, China)
	Ferrocene Correlates with Ferroptosis: Multiple Approaches to Explore Ferrocene-appended GPX4 Inhibitors as Anticancer Agents
11:15-11:20	Shuo Sun 孙硕 (Institute of Biophysics, CAS, China)
	Discovery of small molecule inhibitors specifically targeting the Ero1α-PDI oxidative protein folding pathway
11:20-11:25	Yongjie Zhang 张永杰 (China Pharmaceutical University, China)
	Unraveling the roles of Glutathione S-transferase P in protein S-glutathionylation modulation: Implications of therapeutic targets for oxidative organ injury
11:25-11:30	Jinwen Yang 杨劲文 (Huazhong University of Science and Technology, China)
	Detection of Protein Tyrosine Nitration or Amination
11:30-11:35	Siyu Tian 田丝雨 (Hebei Normal University, China)
	Brain-targeted liposomes with neuroprotective effects for precise therapy of ischemic stroke

11:35-11:40	Xi Hu 胡希 (Anhui University of Chinese Medicine, China)
	Nanomaterials for tumor-cell-specific catalytic therapy
11:40-11:45	Yingnan Liu 刘英楠 (University of Salzburg, Austria)
	Nano-assemblies overcome cancer multidrug resistance for effectively synergistic chemo-immuno-oncotherapy
11:45-11:50	Guofang Zhang 张国芳 (Shenzhen Institute of Advanced Technology, CAS, China)
	Nanomedicine by Modulating ROS for Oncotherapy
	Jing Mu 穆婧 (Peking University Shenzhen Hospital, China)
11:50-11:55	Protective effect of platinum nano-antioxidant and nitric oxide against hepatic ischemia-reperfusion injury
11:55-12:00	Xiaoyan Zhong 仲晓燕 (Soochow University, China)
	Scintillating nanodots as sonosensitizers for cancer sonodynamic therapy

Oct. 23

Flash Talk-2 (FT-2) The Ballroom B

Chair: Yan An 安艳 (Soochow University, China)

10:20-10:25	Yuanyuan Wang 王圆圆 (Institute of Biophysics, CAS, China)
	Targeting the integrated stress response and redox balance is a new strategy in meningioma inhibiting
10:25-10:30	Jiabin Yu 于佳斌 (Jeju National University, Korea)
	Loss of poly(ADP-ribose) polymerase 1 promotes catalase activation via the endothelin receptor
10:30-10:35	Chaorui Guo 郭朝瑞 (China Pharmaceutical University, China)
	Myeloperoxidase (MPO) plays a key role in mitophagy in murine macrophages
10:35-10:40	Lili Xin 信丽丽 (Soochow University, China)
	PM2.5 induced iron accumulation-associated liver injury via activation of ferroptosis and NLRP3 inflammasome
10:40-10:45	Huaiwei Liu 刘怀伟 (Shandong University, China)
	Polysulfides mediate multiple types of protein modification and tumor growth

Chair: Jinchuan Hu 胡晋川 (Fudan University, China)

10:45-10:50	Zhongwei Zhao 赵仲伟 (Beijing University of Chemical Technology, China)
	Physiologically relevant Fenton-like reactions and redox cycles of labile iron species: implications for ferroptosis and Alzheimer's disease
10:50-10:55	Seino Anna (Shibaura Institute of Technology, Japan)
	The changes of genes and protein which affects mitochondrial fusion and fission in AD transgenic mice
10:55-11:00	Yingmin Zhang 张英敏 (Beijing Hospital, China)
	The molecular mechanism study of oxidized microRNA regulating P21 and promoting aging
11:00-11:05	Zhongda Li 李忠达 (Hebei Normal University, China)
	The Beneficial Effects of Knockout of Astrocytic Ceruloplasmin on Learning and Memory Function in Aging Mice
11:05-11:10	Lvtao Zeng 曾律滔 (Beijing Hospital, China)
	Analysis of aging biomarkers and construction of a physiological age prediction model based on cytokine profiling

Chair: Yan Zhao 赵燕 (Institution, Country Harbin Institute of Technology (Weihai), China)

11:10-11:15	Jing Wu 武婧 (Soochow University, China)
	PM2.5-induced premature senescence in HUVECs through the SIRT1/ PGC-1α/SIRT3 pathway
11:15-11:20	Dong He 何东 (Shantou University, China)
	Disruption of E-prostanoid 3 receptor on cardiomyocytes protects against heart ischemia reperfusion injury
11:20-11:25	Shanzhuang Niu 牛善壮 (Yunnan University, China)
	The molecular mechanism of lysosome function impairment and promotes fat accumulation by loss of G6PD
11:25-11:30	Cuomao Niangji 娘吉措毛 (Qinghai University Affiliated Hospital, China)
	Metabolic reprogramming in placental mitochondria respiration contributes to the reproductive success of indigenous Tibetan women living at high altitude
11:30-11:35	Xiaolin Tian 田晓琳 (Shanxi Medical University, China)
	Fecal microbe transplantation ameliorates arsenic-and-fluoride-induced nephrotoxicity of offspring rats co-exposure to arsenic and fluoride through microbiota-gut-kidney axis

Chair: Tomohiro Sawa (Kumamoto University, Japan)

11:35-11:40	Zhang Lu (The University of Hong Kong, China)
	Ganoderma Lucidum Spore Lehuo Powder Attenuates Experimental Autoimmune Encephalomyelitis by Modulating Microglial Activation and Polarization through the NF-κB Signaling Pathway
11:40-11:45	Bingping Yang 杨冰萍 (Shantou University Medical College, China)
	Disruption of circadian rhythms promotes ventricular arrhythmia via oxidative stress and electrocardiography alternation
11:45-11:50	Fei Zhou 周飞 (University of Macau, China)
	Chrysanthemolide J mitigates acetaminophen-induced hepatotoxicity through LKB1 and PP2A-mediated mitochondrial hormesis
11:50-11:55	Mengchen Liu 刘梦晨 (Zhuhai campus of Zunyi Medical University, China)
	Network Medicine landscape on the Health-Enhancing Properties of Natural Antioxidants

Oct. 23

Flash Talk-3 (FT-3) — The Ballroom C

Chair: Julia Li Zhong 钟莉 (Chongqing University, China)

10:20-10:25	Xu Zhang 张旭 (Zhengzhou University, China)
	Hydrogen Peroxide Turn on Heat as Thermogenic agents and signals: Cellular Thermoregulation in Physiologies and Pathphysiologies
10:25-10:30	Qianlei Yang 杨乾磊 (Soochow University, China)
	Redox regulated Mitophagy in Arsenite-induced Malignant Transformation of Human Keratinocytes
10:30-10:35	Chenghua Luo 罗成华 (Shihezi University, China)
	Endogenous hydrogen sulfide promotes the proliferation and metastasis of breast cancer through PGK1 S-sulfhydration
10:35-10:40	Jia Han 韩佳 (Kanazawa Medical University Hospital, Japan)
	High PRDX4 Expression Can Predict Worse Pathological Characteristics in Cutaneous Squamous Cell Carcinom
10:40-10:45	Jie Chen 陈杰 (Shanghai Jiao Tong University School of Medicine, China)
	Radix Rehmanniae and its Active Ingredients Ameliorate CFA-Induced Inflammation by Attenuating Macrophage-Mediated Localized Response and Nitrative Damage

Chair: Junmin Zhang 张军民 (Lanzhou University, China)	
10:45-10:50	Pengfei Liu 刘朋飞 (The Second Affiliated Hospital of Xi 'an Jiaotong University, China)
	Pharmacological targeting of NRF2 represents a promising therapeutic approach for ferroptosis-related diseases
10:50-10:55	Qingyu Wang 王清宇 (Beijing Hospital, China)
	Increased oxidative stress induced by high-fat and high-fructose diets contribute to type 2 diabetes and its associated complications
10:55-11:00	Yau-Tuen Chan (The University of Hong Kong, China)
	Role of miR-3689a-3p in the regulation of mitochondrial oxidative stress in the sorafenib resistance of hepatocellular carcinoma
11:00-11:05	Guoquan Liu 刘国全 (Peking University Health Science Center, China)
	LPO-dependent lipid rafts inhibit immunogenic ferroptosis and pyroptosis in melanoma
11:05-11:10	Yusheng Lu 卢余盛 (Minjiang University, China)
	S-nitrosylation enhances RhoA activity and promotes tumor cell invasion and metastasis
Chair: Kuei-Hung Lai (Taipei Medical University, Taiwan, China)	
11:10-11:15	Xize Li 李析泽 (University of Health and Rehabilitation Sciences, China)
	The circ_0071616-miR-140-3p-USP34 axis mediates FoxM1 deubiquitination in Helicobacter pylori-induced gastric malignant transformation
11:15-11:20	Tingxu Jin 金庭旭 (Soochow University, China)
	A Bayesian benchmark concentration analysis for urinary fluoride and intelligence in adults in Guizhou, China
11:20-11:25	Qiong Wu 吴琼 (Hebei Normal University, China)
	Circadian-Cognitive Synchrony Disrupted: Iron's Influence on Rhythmic and Memory-Related Neural Functions
11:25-11:30	Qianjin Liu 刘前进 (Xuzhou Medical University, China)
	Mechanism analysis of oxidative stress and inflammation in brain diseases
11:30-11:35	Lingyan Su 苏凌燕 (Yunnan Agricultural University, China)
	S-nitrosoglutathione reductase alleviates morphine analgesic tolerance by restricting PKCα S-nitrosation

11:35-11:40	Zhen Li 李振 (Shenzhen Hospital of Integrated Traditional Chinese and Western Medicine, China)
	Phase separation of BRD2 promotes ferritinophagy in depression
11:40-11:45	Xiaoli Zhang 张小莉 (Shanxi Medical University, China)
	Mechanism of arsenic regulation of mitochondrial damage and autophagy induced synaptic damage through SIRT1 and protective effect of melatonin
11:45-11:50	Treethip Sukkho (Chiang Mai University, Thailand)
	Osteoprotective and osteoblastic potential of the Sambucus javanica Reinw ex Blume subsp. javanica leave

12:00-13:30	**SFRR-China Business Meeting Lunch Provided**	Conference 2-6&7 on the second floor of Kuntai Hotel
12:00-12:30	**Lunch**	The Ballroom
12:30-13:30	**Poster Presentation**	The Ballroom C

Oct. 23

Plenary Lecture-4 (PL-4)	The Ballroom
Chair: Jian Kang Liu 刘健康 (University of Health and Rehabilitation Sciences/Xi'an Jiaotong University, China)	
13:30-14:00	Rui-Ping Xiao 肖瑞平 (Peking University, China)
	Role of CaMKII in heart cell fate regulation
Plenary Lecture-5 (PL-5)	The Ballroom
Chair: Yan-Zhong Chang (Hebei Normal University, China)	
14:00-14:30	Zu-Hang Sheng (National Institute of Neurological Disorders and Stroke (NINDS), National Institutes of Health(NIH), USA)
	Energy Matters: Reprogramming of Redox Signaling and Mitochondrial Energy Metabolism in Aged Neurons

14:30-14:50	**Coffee Break**	Poster Hall

14:50-17:00	**Redox Future Perspective Forum** **——The road map for future redox biology and medicine**	The Ballroom

Topic 1, Redox is the basis of life and the common reason for diseases.
Topic 2, The main challenge of redox biology and medicine research in the future.
Topic 3, Advocating "International Redox-decode Project" with multidisciplinary global-level collaborations, in both basic and clinical research.

Chair: Rui-Ping Xiao 肖瑞平 (Peking University, China)

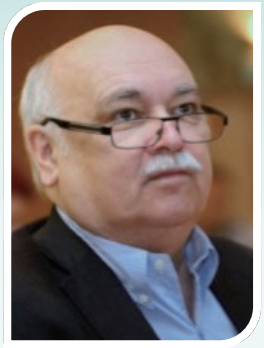
14:50-16:30	**Opening Message** (Video) from Prof. Helmut Sies, Heinrich-Heine-Universitat Düsseldorf, Germany **Panelist Discussion** Chang Chen, China / Andrew Bulmer, Australia / Francisco Laurindo, Brazil / Giovanni Mann, UK / Juan Sastre, Spain / Lin Mantell, USA / Malcolm Jackson, UK / Michael J Davies, Denmark / Xingen Lei, USA / Young-Joon Surh, Korea / Yuji Naito, Japan / Zu-Hang Sheng, USA
16:30-17:00	Open for Discussion

17:00-18:00	**Exhibition Exchange**	Exhibition Hall

18:00-22:00	**Closing & Award Ceremony & Banquet**	The Ballroom

Chair: Jiangang Shen 沈剑刚 (The University of Hong Kong, China)

Plenary Lecture - 1(PL-1)



Roadblock for clinical translation: importance of physiological oxygen levels for high throughput screening of redox therapeutics in live cell models

Giovanni E. Mann

School of Cardiovascular and Metabolic Medicine & Sciences, King's British Heart Foundation Centre of Research Excellence, Faculty of Life Sciences & Medicine, King's College London, UK

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Abstract

In vivo, vascular and other cell types are exposed to physiological oxygen levels ranging from to ~2-13 kPa O_2, while cells cultured in standard CO_2 gassed incubators are routinely exposed to hyperoxic O_2 levels (18 kPa O_2). Although the importance of studying cellular redox signalling under physiological O_2 levels is established, few studies have examined the effects of long-term adaptation of cells to different O_2 levels (Keeley & Mann, Physiol. Reviews 2019;99:161-234; Sies et al., Nature Rev Mol. Cell Biol. 2022;23:499-515). As molecular mechanisms regulating NRF2 mediated redox signaling have primarily been studied under hyperoxia, we characterised NRF2 gene targets in endothelial cells following 5d adaptation to 18 kPa, physiological normoxia (5 kPa) or hypoxia (1 kPa) using O_2 regulated Scitive workstations. Activation of NRF2 and induction of GSH-related genes were insensitive to changes in pericellular O_2 levels, whereas induction of HO-1 and NQO1 in response to electrophiles or NO was attenuated under 5 kPa O_2 due to enhanced expression of the NRF2 repressor Bach1 (Chapple et al., FRBM 2016;92:152-62). Furthermore, a PP2A-mediated feedback mechanism regulates Ca^{2+}-dependent endothelial NO synthesis under 5 kPa O_2 (Keeley et al., FASEB J. 2017;31: 5172-5183), with NO bioavailability increased significantly in cells adapted to 5 kPa O_2 (Keeley et al., FASEB J. 2017;31: 5172-5183; Sevimli et al., Redox Biology 2022;53:1023190; Altun et al., Free Radic Biol Med., 2024;221:89-97). Notably, enhanced SERCA activity under 5 kPa O_2 protects endothelial cells against calcium overload (Keeley et al., FASEB J. 2018;32:2531-2538). We recently employed ICP-MS and LA-ICP-MS to measure changes in total metal content in human coronary artery endothelial (Redox Biol., 2023;62:102712) and smooth muscle (Redox Biol., 2023:64:102777) cells cultured long-term under hyperoxia (18 kPa), physiological normoxia (5 kPa) and hypoxia (1 kPa O_2) and then subjected to ischemia-reoxygenation. We are currently investigating metabolome and lipidome profiles and redox phenotype of human brain microvascular endothelial cells (hCMEC/D3) adapted long-term to 18 kPa or 5 kPa O_2 and laminar shear stress. In summary, adapting cell models in vitro to physiological O_2 levels enhances the physiological relevance of high throughput screening of therapeutics and drugs for clinical translation.

Key words: Redox Biology, KEAP1/NRF2, Reactive Oxygen Species, Physiological Oxygen

Short CV

Prof. Giovanni Mann obtained his BSc in Zoology (1973) from George Washington University, Washington D.C., USA and MSc (1974) and PhD in Physiology (1978) from University College London. He was subsequently appointed to a 4-year postdoctoral Research Fellowship at Queen Elizabeth College London and then to a Lectureship in Physiology (1981), Readership in Physiology (1992) and as Professor of Vascular Physiology (1997-) at King's College London. He is an Associate Editor for Physiological Reviews, Reviews and Special Issues Editor for Free Radical Biology & Medicine and Chair of the FRBM Ethics Committee, President of the Society of Free Radical Research-International (SFRRI), and previously served as President-Elect and General Secretary of SFRRI, Chairman of The Physiological Society, President of the British Microcirculation Society, President of the European Microcirculation Society, President of the Society for Free Radical Research-Europe and President of the European Pancreatic Society. He was elected as a Fellow of The Physiological Society in 2018. He has previously served on Editorial Boards of The Journal of Physiology, Microcirculation and as Editorial Advisor for the Biochemical Journal. He has served as Chair of the Translational Sciences Panel of Heart Research UK, Medical Panel of the Henry Smith Charity and on grant panels of the British Heart Foundation, Guy's & St. Thomas' Hospital Charitable Foundation and Royal Society International Networks Panel. He is currently International Lead for the School of Cardiovascular and Metabolic Medicine & Sciences at King's College London. He has coordinated >45 research symposia at international conferences.



Chair: Chang Chen (陈畅)

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Short CV

Professor Chang Chen is presently Principal Investigator at Institute of Biophysics, Chinese Academy of Sciences (CAS), Professor of University of CAS, and Vice Director of the NationalLaboratory of Biomacromolecules (2012-2023). She received her BSc from Nankai University in 1990 and her PhD from Peking University in 1996. She then joined the Institute of Biophysics, CAS, and became an independent PI in 2000. She was a visiting scientist at the Institute of Food Research, Norwich, UK with The Royal Society K.C. Wong Research Fellowship from 1998 to 2000 and a visiting scientist at Center for Cancer Research, the Medical Research Council, Cambridge, UK from 2004 to 2005 and at NIH in 2018. Her major research interests are nitric oxide and S-nitros(yl)ation and other thiol modification in redox signaling transduction; redox regulation in aging and the related diseases; mechanism of traditional Chinese medicine.

Chen laboratory demonstrated the important roles of S-nitros(yl)ation in protein quality control, aging and aging-related diseases. They also developed a series of methods for S-nitrosation detection. Their work well illuminates the specificity and the important signalling roles of redox regulation. She proposed the concept of Precision Redox and the "5R" principles as the key for antioxidant pharmacology, i.e., Right species, Right place, Right time, Right level and Right target as guidelines for redox medicine development, defined Redox-stress Response Capacity (RRC) and identified the Redox-stress Signaling Threshold (RST) and discovered the insulin-resistance-like phenomenon in senescent cells, termed Redox-stress Response Resistance (RRR). Then based on RRC/RST/RRR, she advocates to increase RST through early stage exercise to enhance RRC, delay the occurrence of RRR, thereby delay aging as the proactive health strategy. Her lab has demonstrated the mechanisms of the effect of L. barbarum on "strengthening muscle and bone and anti-aging" as recorded in "Ben Cao Gang Mu".

Dr. Chen has been honored National Outstanding Young Scientist and also a receiver of Special Government Allowances of the State Council, China. She is the Chief Scientist of "National Basic Research Program of China, 973 Program" (2006-2010) and the Chief Scientist of "National Key Research and Development Program of China" (2017-2022, 2022-2027). Dr. Chen currently serves the President-elect of SFRR-Asia (Society for Free Radical Research, Asia) and the President of the Society for the Free Radical Biology and Medicine, China. She currently serves as the Associate Editor of Free Radical Biology & Medicine (FRBM)(2019-).

Homepage: http://english.ibp.cas.cn/en_sourcedb_ibp/rck/EN_xsszmA_G/202005/t20200519_341422.html

Plenary Lecture - 2(PL-2)



Warburg Effect Revisited: Role of NRF2 in Pseudohypoxic Stabilization o f HIF-1α

Young-Joon Surh

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Abstract

The 'Warburg effect' is defined as an increased rate of glucose uptake and glycoloysis even in the presence of oxygen. Hypoxia-inducible factor-1α (HIF-1α) is highly expressed/activated in most tumors including hepatocellular carcinoma (HCC). Another key transcription factor, nuclear factor erythroid 2-related factor 2 (NRF2) is also constitutively overactivated in HCC. In an attempt to determine whether HIF-1α and NRF2 could play complementary roles growth and progression of HCC, we investigated the crosstalk between these two transcription factors and underlying molecular mechanisms in cultured HCC cells and experimentally induced hepatocarcinogenesis as well as clinical settings. While silencing HIF-1α in HepG2 human hepatoma cells did not alter the protein expression of NRF2, NRF2 knockdown markedly reduced the nuclear accumulation of HIF-1α without influencing its mRNA expression. In diethylnitrosamine (DEN)-induced hepatocarcinogenesis, there was elevated NRF2 expression with concomitant upregulation of HIF-1α. However, this was abolished in Nrf2 knockout mice. NRF2 and HIF-1α co-localize and physically interact with each other which was verified by *in situ* proximity ligation and immunoprecipitation assays. In addition, the interaction between NRF2 and HIF-1α as well as their overexpression was found in specimens obtained from HCC patients. In normoxia, HIF-1α undergoes hydroxylation by a specific HIF-prolyl hydroxylase domain protein (PHD), which facilitates ubiquitination and proteasomal degradation of HIF-1α. However, direct interaction with NRF2 hampers the PHD2-mediated hydroxylation and subsequent recruitment of von-Hippel-Linda for ubiquitination of HIF-1α. This results in the stabilization of HIF-1α, even in the presence of oxygen (pseudohypoxia), which may account for the HIF-1α -mediated aerobic glycolysis (Warburg effect).

Key words: HIF-1α, Hepatocellular carcinoma, Hypoxia, NRF2, Pseudohypoxia, Warburg effect

Short CV

Prof. Young-Joon Surh graduated from College of Pharmacy, Seoul National University with Bachelor's and Master's degrees. Prof. Surh earned a PhD degree at the McArdle Laboratory for Cancer Research, University of Wisconsin-Madison. He had postdoctoral training at Massachusetts Institute of Technology (MIT). After spending three and half years as a tenure-track Assistant Professor at Yale University School of Medicine, Prof. Surh relocated to Seoul National University in 1996. Since then, he has been investigating the molecular mechanisms of cancer chemoprevention with anti-inflammatory and antioxidative natural products, with focus on intracellular redox and inflammatory signaling molecules as prime targets. Beside his role as Editor-in-Chief of Journal of Cancer Prevention, Prof. Surh is currently Associate Editor of Toxicology & Applied Pharmacology, Molecular Carcinogenesis, and Free Radical Research, and Editorial Board member of International Journal of Cancer, Cancer Letters, Cancer Prevention Research, Precision Oncology, Molecular & Cellular Biology, Free Radical Biology & Medicine, Antioxidants & Redox Signaling, Genes and Disease, Genes and Nutrition, Molecular Nutrition & Food Research, International Journal of Molecular Sciences, etc. He also co-edited following books: Oxidative Stress, Inflammation and Health (CRC Press), Molecular Targets & Therapeutic Use of Curcumin (Springer-Verlag), and Dietary Modulation of Cell Signaling Pathways (CRC Press). Prof. Surh has published more than 420 papers in peer-reviewed international journals and about 70 invited editorials, reviews and book chapters. The total number of citations of his publications is more than 30,000. The H-Index reported by Thomson Reuter of Web Knowledge is 90. Thomson Reuter selected him as one of the 16 Korean scientists whose publication is most highly cited. Prof. Surh received numerous awards including Elizabeth C. Miller and James A. Miller Distinguished Scholar Award from Rutgers University (2011), McCormic Science Institute Award from American Society for Nutrition (2009), Scientist of the Year Award from the Korea Science Reporters Association (2008), the Korea Science Award given by President of South Korea (2013), etc. He currently serves as President of Society of Free Radical Research-Asia (SFRR-Asia) and Chair of Division of Medical Sciences, Korean Academy of Science and Technology (KAST).



Chair: Malyn Ungsurungsie

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Short CV

Received a Bachelor Degree in Pharmacy from Faculty of Pharmacy, Chulalongkorn University with the Master and Doctorate Degrees in Science (Microbiology), International Program, from Faculty of Science, Mahidol University. Also received a Certificate of Management in Higher Education from Galilee College, Israel and Harvard University Extension School, United States. Had the post-doctorate training at School of Pharmacy, Robert Gordon University, United Kingdom. Followed was a research fellow at Institute of Medical Science, University of Tokyo, and Faculty of Agriculture and Veterinary Medicine, Nihon University, Japan. Had a few-year research experiences at Institute for Biochemical Technology and Microbiology, Vienna University of Technology, Austria.

Started working at Faculty of Pharmacy, Mahidol University and has accomplished to be a full professor. The most recent position is a director of S&J Research & Innovation Center. Additionally, has been appointed as a director and committee member to companies, associations and organizations e.g., S&J (UK) Co., Asian Association of Environmental Mutagen Societies, Silpakorn University, Chiang Mai University.

Received several awards and recognitions e.g., "Invention Award" from National Research Council of Thailand, "Outstanding Industrial Pharmacist" from Thai Industrial Pharmacist Association, "Outstanding Pharmacist" from The Pharmaceutical Association of Thailand Under Royal Patronage, "Outstanding Alumni" from Mahidol University Graduate Study Alumni Association, "Leading Scientists of the World" in the Area of Pharmaceutical Sciences from The Office of the International Biographical Centre, "Cooperation in Narcotic Prevention Award" from Office of the Narcotics Control Board, "Pharmacist Volunteer Certificate of Appreciation" from Her Majesty the Queen Rambhai Barni Medical Mobile Unit.

Plenary Lecture - 3(PL-3)



Gut frailty: its concept and the role of dietary fiber

Yuji Naito

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Abstract

There is still a considerable gap between average life expectancy and healthy life expectancy in Japan. Recent research has revealed that gut frailty may be an aggravated factor for various diseases, a cause of chronic inflammation, and a precursor to frailty. Among self-reported symptoms, constipation is particularly significant as one of the key symptoms of gut frailty. Studies have demonstrated that individuals with constipation have significantly lower survival rates and are also at a higher risk of developing various diseases such as chronic kidney disease, cardiovascular diseases, and neurodegenerative disorders like Parkinson's disease. Various molecular mechanisms could contribute to gut frailty, and the decrease in mucus secretion is an extremely early-stage pathology. Dysbiosis of gut microbiota has a major impact on many conditions associated with gut frailty. Prebiotics including dietary fibers, probiotics, post-biotics, and fecal microbiota transplantation are under investigation as a treatment option for gut frailty. Although the concept of gut frailty has not yet gained widespread recognition, we hope to propose more practical screening methods, diagnostic approaches, and specific interventions in the future.

Key words: Aging, Gut frailty, Microbiota, Dietary fiber

Short CV

Assistant Professor of Medicine, First Department of Medicine, Kyoto Prefectural University of Medicine, 1998-2000.

Visiting Professor, Department of Molecular and Cellular Physiology, LSU Health Sciences Center, 2001

Associate Professor, Molecular Gastroenterology and Hepatology, Kyoto Prefectural University of Medicine, 2008-2021

Chief, Department of Endoscopy and Ultrasound Medicine, Hospital of Kyoto Prefectural University of Medicine, 2015-2021

Professor, Department of Human Immunology and Nutrition Science, Kyoto Prefectural University of Medicine, 2021-



Chair: Myung Hee Chung

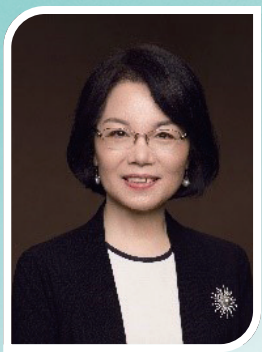
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Short CV

- 1982 ~ 2011: Professor, Pharmacology, Seoul national University College of Medicine
- 2002 ~ 2004: Vice President, Seoul National University
- 2011 ~ 2014: Chair professor, Samsung Medical Center
- 2014 ~ 2018: Vice President for medical Affair, Gachon University
- At Present: Science Adviser, Korea Health Functional Food

Plenary Lecture - 4(PL-4)



Role of CaMKII in heart cell fate regulation

Rui-Ping Xiao (肖瑞平)

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Abstract

Ca^{2+}/calmodulin-dependent kinase II (CaMKII), particularly its predominant isoform in the heart, CaMKII-δ, mediates multiple stress stimuli in the myocardium and is a key regulator of the fate of cardiomyocytes. The excessive activation of CaMKII leads to cardiomyocyte death in conditions such as myocardial infarction, cardiomyopathy, and heart failure. Our findings reveal that receptor-interacting protein 3 (RIP3) activates CaMKII through phosphorylation and oxidation, thereby promoting necroptosis in cardiomyocytes. Inhibition of CaMKII or RIP3 deficiency in mice reduces necroptosis and attenuates heart failure. Furthermore, the CaMKII-δ9 isoform contributes to cardiomyocyte death by impairing DNA repair. Targeting CaMKII oxidation and activity, including using the specific inhibitor hesperadin, presents a promising therapeutic strategy for reducing cardiac damage and related pathologies.

Key words: CaMKII, oxidation/phosphorylation, cardiomyocyte death, necroptosis

Short CV

Dr. Rui-Ping Xiao, a Peking University Chair Professor, is the Dean of the College of Future Technology at Peking University.

Dr. Xiao received her M.D. degree from Tongji Medical University in 1987 and her Ph.D. degree in Physiology from the University of Maryland in 1995. In 2003, she was appointed as a Principal Investigator with tenure of the National Institutes of Health, and in 2010, she returned to China to become the founding Director of the Institute of Molecular Medicine at Peking University.

Dr. Xiao's research has been focused on cardiovascular and metabolic diseases, with an emphasis on a translational approach to bring bench discoveries to clinically relevant situations. Ongoing research directions include signaling pathways involved in Cardiometabolic disease. She served as a Council Member of the International Society of the Heart (ISHR) from 2002 to 2021 and was elected a Fellow of the American Society for Clinical Investigation (ASCI) in 2004. Currently, Dr. Xiao serves as an Associate Editor of the New England Journal of Medicine and an Editorial Board Member of multiple international top journals.



Chair: Jiankang Liu (刘健康)

University of Health and Rehabilitation Sciences, Qingdao, China

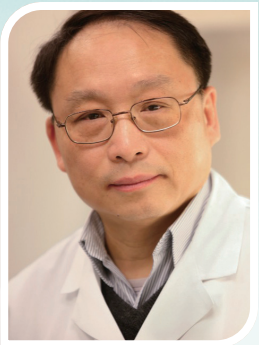
Email: jkliu@uor.edu.cn

Short CV

Dr. Jiankang Liu received his BS from Xi'an Jiaotong Unviersity and PhD of Medical Science from Okayama University School of Medicine, Japan. He completed post-doc training in Dr. Bruce Ames laboratory at University of California, Berkeley and worked as a faculty at University of California at Berkeley, Children Hospital Oakland Research Institute, University of California at Irvine, University of Kentucky College of Medicine, and Shanghai Institute for Nutritional Science, Chinese Academy of Sciences. Currently, he is a Professor of the University of Health and Rehabilitation Sciences at Qingdao and Xi'an Jiaotong University at Xi'an, China. Research interests include molecular and cellular mechanisms of aging, stress, and age-/stress-associated degenerative diseases with a focus on free radical and mitochondrial biology and medicine. Has proposed the "Mitochondrial Free Radical Hypothesis of Stress-induced Aging Acceleration" and "Mitochondrial Nutrients Concept" and published more than 270 peer reviewed papers in SCI journals, 2 books, and 20 book chapters; applied 24 patents; The total citations are 17000 times and H index is 81 (Google Scholar Data of April 10, 2024) and topped consecutively for 10 years (2014-2024) to the list of "Elsevier the Most Cited Chinese Researchers" and the World Top 1.5-2% Scientist (2019-2023).

He worked as Associate Editor for "Nutritional Neuroscience", "Antioxidants" and "Current Topics on Neutraceutical Research", and editorial board member for "Free Radical Biology and Medicine", "Antioxidants and Redox Signaling", "Mitochondrion" "Neurochemical Research", "Basic and Clinical Pharmacology and Toxicology" "Sport Medicine and Health Sciences" etc. As Co-Guest Editor for editing special issues for "Neurochemical Research", "Free Radical Biology and Medicine", "Antioxidants", and "Antioxidants and Redox Signaling".

Plenary Lecture - 5(PL-5)



Energy Matters: Reprogramming of Redox Signaling and Mitochondrial Energy Metabolism in Aged Neurons

Zu-Hang Sheng

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Abstract

Mitochondria are the cellular power plants that generate ATP to power various neuronal functions and regeneration. Chronic mitochondrial dysfunction and energy deficits are pathological hallmarks of aging-associated neurodegeneration, while acute mitochondrial damage triggered by brain injury leads to a local energy crisis that contributes to regeneration failure. Therefore, defects in mitochondrial maintenance have emerged as central issues in neurodegeneration and regeneration. I will first overview our recent investigations of reprogramming mitochondrial maintenance and transport and restoring bioenergetic metabolism to power neuronal regeneration and synaptic transmission **(1-13).**

Aging is a key risk factor in the development of neurodegeneration. As postmitotic cells, neurons face exceptional challenges in maintaining energy homeostasis over an organism's lifespan. Recovery of chronically stressed mitochondria is critical to energy maintenance in neuronal aging. Mitochondrial DNA (mtDNA) encodes 13 proteins essential for oxidative phosphorylation and ATP production. Mitochondrial nucleoids (mt-nucleoids) are composed of mtDNA and machineries for mtDNA replication and transcription. Since mitochondria are one of the primary sources of ROS, mt-nucleoids are constantly exposed to an oxidative microenvironment throughout a neuron's life. However, it remains elusive how chronic oxidative stress affects mt-nucleoid integrity and mitochondrial bioenergetics with age. I will introduce our recent work revealing the crosstalk between redox signaling and mitochondrial energy metabolism in aged neurons **(14)**. Using mouse dorsal root ganglion neurons and human iPSC-derived neuronal models, combined with cutting-edge live-cell imaging with mitochondria-targeted ROS and ATP sensors and Seahorse assays, we provide a comprehensive mitochondrial aging profile: neuronal mitochondria from aged mice display increased ROS levels, declined respiratory capacity, and reduced ATP production compared to neurons from young adult mice. Combining phase separation assays, MINFLUX nanoscopy imaging (1-3 nm resolution), AI-based machine learning, and mtDNA transcription/translation analysis, we found that neuronal mt-nucleoids from aged mice are strikingly disorganized both structurally and functionally. Triggering oxidative stress in young neurons recapitulates the aging mt-nucleoid phenotypes. With genetic targeting, we further identified oxidative response elements. Reprogramming oxidation-resistant signaling by replacing the targeted cysteine residue effectively attenuates

the disruption of mt-nucleoid condensates and reverses the declined mitochondrial energy metabolism in neurons from aged mice. Thus, our study provides new mechanistic insights into how chronic oxidative stress in aged neurons adversely affects mitochondrial bioenergetics, offering translational implications for restoring mt-nucleoid phase separation and integrity in normal aging and aging-associated neurodegeneration (Supported by the Intramural Research Program of NINDS, NIH).

Selected Sheng lab publications on neuronal mitochondria and energy metabolism:

1.Cheng XT, Huang N, Sheng ZH, **Neuron**, 2022, 110: 1899.

2.Li S, Sheng ZH, **Nature Reviews Neuroscience** 2022, 23:4.

3.Chamberlain KA*, Huang N* et al., **Neuron** 2021, 109:3456.

4.Huang N et al., **Current Biology** 2021, 31:3098.

5.Li S et al., **Nature Metabolism** 2020, 2:1077.

6.Han Q et al., **Cell Metabolism** 2020, 31:623.

7.Puri R et al., **Nature Communications** 2019, 10:3645.

8.Lin MY*, Cheng XT* et al., **Neuron**, 2017, 94: 595.

9.Zhou B et al., **Journal of Cell Biology**, 2016, 204:103

10.Morsci N et al., **Journal of Neuroscience,** 2016, 36:1373.

11.Xie Y, Zhou B et al., **Neuron** 2015, 87:355.

12.Sun T, Qiao H et al., **Cell Reports**, 2013, 4:413.

13.Kang JS et al., **Cell** 2008, 132:137.

14.Cheng XT*, Gao Y* et al., **unpublished**.

Short CV

Dr. Sheng received his Ph.D. in Biochemistry from the University of Pennsylvania in 1993 and completed his postdoctoral research with William Catterall at the University of Washington in 1996. He joined NINDS as an investigator in 1997 and is now a senior investigator and Chief of the Synaptic Functions Section at NINDS, NIH. Dr. Sheng was elected a Fellow of the AAAS in 2016 and a fellow of the ASCB in 2017. He received the 2021 Dr. Francisco S. Sy Award for Excellence in Mentorship from the US Department of Health and Human Service (HHS) and is the recipient of the 2023 NIH Director's Award for seminal contributions to the understanding of axonal mitochondrial and lysosomal transport and the maintenance of bioenergetics and cellular homeostasis in synaptic transmission and neural regeneration.



Chair: Yan-Zhong Chang (常彦忠)
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Short CV

Dr. Yan-Zhong Chang, Professor of College of Life Science in Hebei Normal University. He got his PhD degree in Hong Kong Polytechnic University. From 2008 to 2009, he was a Visiting Professor to study the regulation of brain iron metabolism in Dr. Tracey Rouault's Lab, NICHD. In 2003, he founded the Lab of molecular iron metabolism in Hebei Normal University. He major conducts his research in the mechanisms of iron metabolism, the mechanisms and treatment of iron misregulation and redox imbalance in Parkinson's disease, Alzheimer's disease, stroke, mental and emotional disorders; Preparation and safety evaluation of brain-targeted nanomedicines. As the first author or corresponding author Prof. Chang has published more than 100 papers on peer reviewed international journals such as European Heart Journal, ACS Nano, Redox Biology, and these papers have got more than 5000 citations. As the editor of 'Brain Iron Metabolism and CNS Diseases' was published by the Springer (2019). He is the Advisory Board member of the Journal- Cellular and Molecular Life Sciences. He won the First (2019), Second (2016) and Third (2013) Grade Awards for Natural Science of Hebei Provence.

Special Lecture



Carry forward the cause and forge ahead into the future ——Memory of past 20 years SFRR-Asia biennial meetings

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Taking this special opportunity, I would like to review the history and achievement of the Society for Free Radica Research-Asia (SFRR-Asia) and to recall the great memories of the past ten biennial meetings. At the same time, I will share my view of the future development of redox research to attract good ideas. After two decades, we are marching into a new era to explore redox biology and medicine at precision, mechanistic and *in vivo* levels with innovative technology and multiple discipline collaborations. It is the right time to think over the future of redox biology and medicine with regard to the conception, the essential questions, the new strategy and even the research pattern. Concerning the main challenge of redox biology and medicine research, I propose that seven layers (7L) should be explored, aiming "to know redox, to decode redox, and to utilize redox". L1, New technology for precision redox research, particularly an *in vivo*, in situ quantitative approach; L2, Exploring the redox network/family regarding discovery of new redox genes, species, noncoding RNAs, etc. L3, Biochemical mechanism of redox, concerning redox modification of biomacromolecules, redox relay, and redox architecture. L4, Redox regulation in organelle function, quality control and cell fate. L5, Redox physiology in development and reproduction and environmental challenge. L6, Redox stress in the pathogenesis of various diseases. Some uncultivated lands should be addressed, for example, redox signaling in mental health. L7, Precision redox intervention and health management, involving traditional medicine, intelligence materials, lifestyle, nutrition application, drug development, etc. Popularization of science and technology of redox biology and medicine is also one important part for improving public health. To stimulate the 7L research, multidisciplinary global-level collaborations, in both basic and clinical research, need to be implemented.



Chair: Yang Liu (刘扬)

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Short CV

Yang LIU, B.S. (1982) and M.S (1985) at Tsinghua Univ., Ph.D. (1988) at Inst. of Chem., the CAS; assist. Prof. (1988-1991); Associate Prof. (1991-1992) at Inst. of Chem., the CAS; visiting scientist at Institut für Pharmakol., Veterinämed. Univ. Wien (1992-1997); full Prof. (1997- present) in Inst. of Chem , the CAS & Univ. of CAS.

Awards:

The Natural Science Prize, 1991, in The Chinese Academy of Sciences on the contribution of spin trapping - ESR investigation;

Wang Tian-jiuan Prize, 2000, from the Magnetic Resonance Committee, Chinese Physical Society;

Contribution Award of EPR Development, 2022, from Xu Yuanzhi Award Funds of EPR Development;

Outstanding Contribution Award, 2023, from SFRR-China.

Research Interests

Ø Nanomedicine and drug delivery for oxidative stress-related diseases, such as ischemic stroke, Alzheimer's disease and cancer;

Ø *In vivo* and intracellular ROS/NOS assays with fluorescence probes and microelectrodes;

Ø *In vivo* and intracellular target spin trapping - ESR.

Selected Publications (total publications: 168)

1 Xiaojie Zhang, Xiaoxuan Kang, Libo Du, Lu Zhang, Yan Huang, Jihan Wang, Sihan Wang, Yanzhong Chang*, Yang Liu*, Yuming Zhao*, Tanshinone IIA loaded chitosan nanoparticals decrease toxicity of β-amyloid peptide in a Caenorhabditis elegans model of Alzheimers disease, Free Rad Biol Med, 193: 81–94(2022).

2 Yaru Li, Xiaojie Zhang, Zhifeng Qi*, Xueling Guo, Xiaopeng Liu, Wenjuan Shi, Yang Liu*, LiBo Du*, The enhanced protective effects of salvianic acid A: A functionalized nanoparticles against ischemic stroke through increasing the permeability of the blood-brain barrier, Nano Research , 13: 2791-2802(2020).

3 Xueling Guo, Xiaoxuan Kang, Yueqi Wang, Xiaojie Zhang, Changjian Li, Yang Liu*, Libo Du*. Co-delivery of cisplatin and doxorubicin by covalently conjugating with polyamidoamine dendrimer for enhanced synergistic cancer therapy, Acta Biomaterialia, 84: 367-377(2019)..

4 Shaipeng Huang, Rongchen Han, Qiaofen Zhuang, LiBo Du, Hongying Jia, Yangping Liu, Yang Liu*, New photostable naphthalimide-based fluorescent probe for mitochondrial imaging and tracking. Biosensors and Bioelectronics, 71: 313-321 (2015).

5 Qianfen Zhuang, Hongying Jia, Libo Du, Yanchao Li, Zhao Chen, Saipeng Huang, Yang Liu*. Targeted Surface-functionalized Gold Nanoclusters for Mitochondrial Imaging. Biosensors and Bioelectronics, 55: 76-82 (2014).

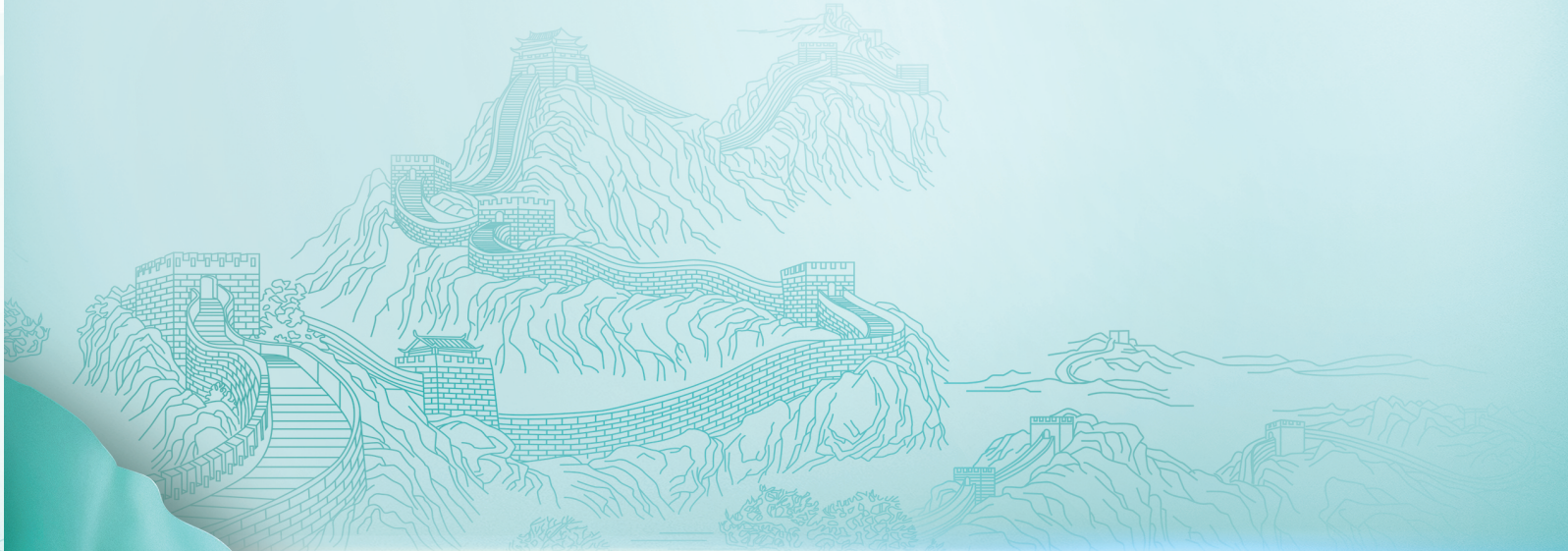
6 Lu Han, Libo Du, A Kumar, Hongying Jia, Qiu Tian, Guangjun Nie, Xinghe Liang, Yang Liu*, Inhibitory effects of trolox-encapsulated chitosan nanoparticles on tert-butylhydroperoxide induced RAW264.7 apoptosis, Biomaterials, 33: 8517-8528(2012).

7 Hong-ying Jia, Yang Liu*, Xue-ji Zhang*, Lu Han, Li-bo Du, Qiu Tian, Yuan- chao Xu, Potential Oxidative Stress of Gold Nanoparticles by Induced-NO Releasing in Serum, J Am Chem Soc, 131(1): 40-41 (2009).

8 Zhou Nie, Ke-jian Liu, Chuan-jian Zhong, Lan-fen Wang, Ying Yang; Qiu Tian; Yang Liu*, Enhanced radical- scavenging activity by antioxidant-functionalized gold nanoparticles: A novel inspiration for development of new artificial antioxidant, Free Rad Biol Med, 43: 1243–1254(2007)

Symposium-1(S1)

Redox signaling in organelles/cell fate/development/reproduction





Chair: Taotao Wei (卫涛涛)

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Short CV

1989 - 1993 BSc, Huazhong University of Science and Technology

1993 - 1996 MSc, Hubei Institute of Chemistry

1996 - 1999 PhD, Institute of Biophysics, Chinese Academy of Sciences

1999 - 2001 Assistant Professor, Institute of Biophysics, Chinese Academy of Sciences

2001 - 2008 Associate Professor, Institute of Biophysics, Chinese Academy of Sciences

2008 - Professor, Institute of Biophysics, Chinese Academy of Sciences

Mitochondrion is the center for energy metabolism machinery, and the major checkpoint of apoptotic regulation. The homeostasis of mitochondrial network plays essential role in many cellular processes; its dysregulation has been linked to many diseases. We aim to use combined approaches to investigate the mechanism and regulation of the mitochondrial network, and their implication in various human diseases.

Selected papers:

[1] CRISPR screening uncovers nucleolar RPL22 as a heterochromatin destabilizer and senescence driver. Nucleic Acids Research, (2024), doi.org/10.1093/nar/gkae740.

[2] Structural and biochemical insights into the mechanism of the Gabija bacterial immunity system. Nature Communications, 15(2024), 836.

[3] Airway relaxation mechanisms and structural basis of osthole to improve lung function in asthma. Science Signaling, 13(2020), eaax0273.

[4] KAP1-associated transcriptional inhibitory complex regulates C2C12 myoblasts differentiation and mitochondrial biogenesis via miR-133a repression. Cell Death and Disease 11(2020), 732.

[5] Transforming growth factor (TGF)-β1-induced miR-133a inhibits myofibroblast differentiation and pulmonary fibrosis. Cell Death and Disease 10(2019), 670.

[6] Detection of tBid oligomerization and membrane permeabilization by graphene-based dingle-molecule surface-induced fluorescence attenuation. Nano Letters 19(2019), 6937-6944.

[7] YWHA/14-3-3 proteins recognize phosphorylated TFEB by a noncanonical mode for controlling TFEB cytoplasmic localization. Autophagy 15(2019), 1017-1030.

[8] SIRT5 deacylates metabolism-related proteins and attenuates hepatic steatosis in ob/ob mice. EBioMedicine 36(2018), 347-357.



H_2O_2 promotes stomatal development and opening through regulating SnRK1

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Abstract

Stomata are plant-specific epidermal structures that function as the main conduit for water vapor and carbon dioxide exchange between plant and atmosphere. The formation and movement of stomata is regulated by multiple developmental and environmental signals. Here, we showed that spatially patterned hydrogen peroxide (H_2O_2) plays essential roles in stomatal development and light-induced stomatal opening through regulating the nucleocytoplasmic shuttling of KIN10, the catalytic α-subunit of energy sensor kinase SnRK1. H_2O_2 is remarkably enriched in meristemoids and guard cells, which is established by spatial expression patterns of H_2O_2-scavenging enzyme CAT2 and APX1. H_2O_2 interferes the interaction between KIN10 and the regulator subunit KINβ2, and then promotes the nuclear localization of KIN10. In stomatal lineage cells, KIN10 phosphorylates and stabilizes SPCH, a master regulator of stomatal formation, thereby promoting stomatal development. In guard cells, KIN10 phosphorylates a bZIP transcription factor KIP1, which specifically expresses in guard cells. KIN10-mediated phosphorylation of KIP1 enhances its transcriptional activity on BAM1 and KAT1, thereby promoting stomatal opening upon light exposure. The spatial distribution pattern of H_2O_2 in meristemoids and guard cells was found not only in Arabidopsis leaves but also in wheat leaves. H_2O_2 also plays an essential role for light-induced stomatal opening in wheat leaves. Overall, these evidences uncover the conservative roles of H_2O_2 in promoting stomatal development and stomatal opening in monocotyledon and dicotyledon plants.

Key words: H_2O_2, SnRK1, Brassinosteroid, Stomatal development, Stomatal opening

Short CV

Prof. Mingyi Bai earned his Ph.D. from the Institute of Botany, CAS, in 2007 and completed postdoctoral training at the Carnegie Institution for Science in Zhi-Yong Wang's lab. In 2014, he was appointed as a Principal Investigator at Shandong University. His research focuses on how the phytohormone brassinosteroid regulates plant growth and stress responses. Notable achievements include elucidating the role of brassinosteroids (BR) in the nitrate signaling pathway, detailing BR-mediated guard cell starch metabolism in stomatal movement, and uncovering the crucial role of hydrogen peroxide (H_2O_2) in BR signaling. His work has been published in top journals such as Nature Plants, Nature Communications, and Plant Cell, and has been featured in Preview. Prof. Bai has received several awards, including the National Science Fund for Distinguished Young Scholars, the Shandong Province Science Fund for Distinguished Young Scholars, and the Young Thousand Talents program. He also serves as an editor for JIPB, New Crops, and Frontiers in Plant Science.



Artemisinin attenuates astrocyte overactivation by inhibiting IRE1 phosphorylation and the downstream NF-κB pathway in Alzheimer's disease

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Abstract

Alzheimer's disease (AD) is characterized by the accumulation of amyloid-beta (Aβ) and is associated with neuroinflammation, endoplasmic reticulum (ER) stress and cognitive decline. Abnormal accumulation of β amyloid peptide (Aβ) induces ER stress, activating astrocytes through the nuclear factor kappa-B (NF-κB) pathway and ultimately causing neuroinflammation and neuronal injury. Astrocyte dysfunction can disrupt the normal neuronal environment, which is essential for maintaining cognitive function. Therefore, targeting the modulation of the ER stress-inflammatory cycle and normalizing astrocyte function could be a potential strategy for AD. Recent studies indicate that artemisinin has significant neuroprotective effects. However, the mechanism by which artemisinin regulates astrocyte activation to improve AD process requires further exploration. We investigated the impact of Aβ1-42 on astrocyte activation and explored the potential therapeutic effects of artemisinin (ART) *in vitro* and in a 3×Tg-AD mouse model.

Exposure of A172 cells to Aβ1-42 induced astrocyte activation, endoplasmic reticulum (ER) stress, and inflammatory responses. ART treatment attenuated these effects, specifically inhibiting IRE1 phosphorylation and downstream NF-κB signaling. ART also restored the neurotrophic function of astrocytes, protecting primary neurons from Aβ1-42 toxicity. The IRE1 kinase inhibitor KIRA6 reversed the toxic effects of Aβ1-42 on astrocytes, emphasizing the role of IRE1 in neuroinflammation.

In further studies, we demonstrated that ART's neuroprotective effects were mediated through the IRE1-ER stress pathway. It relied on IRE1 kinase activity to prevent Aβ1-42-induced astrocyte overactivation, confirmed by IRE1 wild-type and mutant plasmid experiments. Additionally, ART restored the phosphatase activity of PP2A, inhibiting IRE1 phosphorylation.

To validate these findings *in vivo*, 3×Tg-AD mice were treated with ART. ART reduced IRE1-mediated downstream inflammatory signals, alleviated astrocyte overactivation, and rescued neuronal apoptosis. It also ameliorated cognitive deficits in these mice. Pharmacological interventions in the mouse model further supported ART's therapeutic potential by demonstrating improvements in cognitive function and reduced neuroinflammation. Importantly, AAV-mediated IRE1 overexpression in astrocytes abrogated the beneficial effects of ART, highlighting the critical role of IRE1 in mediating ART's neuroprotective effects.

In conclusion, our study demonstrates that artemisinin exerts neuroprotective effects by modulating the IRE1-ER stress pathway in astrocytes, reducing neuroinflammation, and ameliorating cognitive deficits in an AD mouse model. These findings provide insights into the potential therapeutic value of artemisinin in Alzheimer's disease.

Short CV

Dr. WenHua Zheng, Professor, Principle Investigator in the Faculty of Health Science, University of Macau, leading a group of scientists working on aging and neuronal degenerative disorders, including Alzheimer's disease and degenerative retinal diseases; New functions and downstream targets of FoxO; Protective effect of Artemisinin and new drug developments. He is a Section Editor for Encyclopedia of Gerontology and Population Aging and a Lead Guest Editor and Editor for several journals. He is a grant Reviewer for NSFC, Poland, and CIHR in Canada. He is the Honorable Professor at the University of Queensland (QS45) and an Adjunct Professor/Visiting Prof at RMIT University in Australia and other universities at home and abroad. Dr Zheng has published >150 papers, which have been cited over 5000 times (Google >9000).



Redox Signal Regulation by Supersulfides

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Abstract

The major focus of redox biology has been on molecular oxygen, the most abundant element of the planet. The oxygen molecule accepts electrons from the respiratory chain in the mitochondria and is responsible for energy production in aerobic organisms. In addition, oxygen-derived reactive oxygen species that include hydrogen peroxide and oxygen- and nitrogen-centered free radicals, such as superoxide, hydroxyl radical and nitric oxide, undergo a complicated way of electron transfer reactions through their interaction with other biological substances, leading to alteration of their physiological functions, and cause diverse biological and pathophysiological consequences like oxidative stress. Discovery of supersulfides helped us to realize that the oxygen molecule itself accounts only partly for the redox reaction in many organisms, even under aerobic or hypoxic conditions, however, while it is much less biologically relevant in anaerobic and anoxic environments. My talk will deal with a brand-new venue of redox biology, which is governed by the redox-active supermolecules that are mostly consisted of supersulfides, i.e., sulfur-catenated molecular species. They are now found abundantly in all organisms but remain largely unexplored in view of the redox biology and life science research. In fact, accumulating evidence show that supersulfides are electron rich and thereby readily ionized or radicalized, so that they can actively participate in the energy metabolism, redox dependent signaling, and oxidative stress responses in the cellular and *in vivo* context. Moreover, the pharmacological intervention and medicinal manipulation of supersulfides has been shown to be beneficial in prevention as well as regulation of disease pathogenesis. The supersulfide biology now open up a new era of disease control that includes its potential application to clinical diagnosis, prevention, and therapeutics for various diseases.

Key words: Persulfides, polysulfides, supersulfides, redox signaling

Short CV

1992 Assistant Professor, Department of Microbiology, Kumamoto University School of Medicine;1993 Visiting Professor, Department of Microbiology and Immunology, Center for Neurovirology, Thomas Jefferson University; 1994 Associate Professor, Department of Microbiology, Kumamoto University School of Medicine; 2001 Visiting Professor at Center for Free Radical Research, University of Alabama at Birmingham; 2003 Program Officer at the Ministry of Education, Science, Sports and Culture (MEXT) of Japan; 2013 Full Professor, Department of Microbiology, Graduate School of Medical Sciences, Kumamoto University; 2013 Vice Dean & Director of Center for Medical Education and Research at Kumamoto University Medical School; 2019 Vice Dean, Tohoku University Graduate School of Medicine and Tohoku University Medical School; 2013-present Full Professor and Chair, Department of Environmental Medicine and Molecular Toxicology, Tohoku University Graduate School of Medicine



Carbon monoxide sensitizes cancer cell to erastin-induced ferroptosis via ROS-PERK-ATF4

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Abstract

Ferroptosis is an emerging form of regulated cell death, distinct from traditional apoptosis, characterized by iron-dependent lipid peroxidation. Carbon monoxide (CO), an endogenously gaseous molecule that is generated via the catabolism of heme by heme oxygenase 1 (HO-1), induces mitochondrial ROS-mediated activation of PERK, an arm of unfolded protein response (UPR). Because CO produces ROS and activates PERK which is known to be activated during ferroptosis, we tested whether CO could affect the ferroptosis inducer -mediated ferroptosis. We found that CO increases ferroptosis susceptibility of cancer cells even though its mechanism is not definitely identified. We observed that PERK was highly activated by CO in dose-dependent manner and in turn, sensitized cancer cells to erastin-induced ferroptotic cell death. Moreover, CO-mediated activation of PERK-ATF4 pathway leads to an increase in ferroptosis marker PTGS2 expression and lipid ROS. Additionally, ATF4-driven upregulation of CHAC1 and REDD1 resulted in the depletion of glutathione (GSH) and suppression of mTORC1 and GPX4, respectively, further promoting ferroptosis. The deficiency of PERK abrogated CO-induced ferroptosis sensitivity. These results reveal that the CO-PERK-ATF4 axis plays a crucial role in sensitizing cancer cells to erastin-induced ferroptosis, offering potential therapeutic avenues for enhancing ferroptosis-based cancer treatments.

Short CV

· 2024~ present: Professor, Daegu Haany University, Republic of Korea

· 2009~2023: Professor, University of Ulsan, Republic of Korea



Oxidative phosphorylation, H_2O_2 production, mitochondrial membrane potential, coenzyme Q redox state, and calcium uptake: from tissue normoxia to deep hypoxia

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Abstract

Mitochondrial respiration extends beyond ATP generation, with the organelle participating in many cellular and physiological processes. Parallel changes in components of the mitochondrial electron transfer system with respiration render it an appropriate hub for coordinating cellular adaption to changes in oxygen levels. How changes in respiration under functional hypoxia (i.e., when intracellular O_2 levels limit mitochondrial respiration) are relayed by the electron transfer system to impact mitochondrial adaption and remodeling after hypoxic exposure remains poorly defined. This is largely due to challenges integrating findings under controlled and defined O_2 levels in studies connecting functions of isolated mitochondria to humans during physical exercise. Here we present experiments under conditions of hypoxia in isolated mitochondria, myotubes and exercising humans. Performing steady-state respirometry with isolated mitochondria we found that oxygen limitation of respiration reduced electron flow and oxidative phosphorylation, lowered the mitochondrial membrane potential difference, and decreased mitochondrial calcium influx. Similarly, in myotubes under functional hypoxia mitochondrial calcium uptake decreased in response to sarcoplasmic reticulum calcium release for contraction. In both myotubes and human skeletal muscle this blunted mitochondrial adaptive responses and remodeling upon contractions. Our results suggest that by regulating calcium uptake the mitochondrial electron transfer system is a hub for coordinating cellular adaption under functional hypoxia.

Symposium-2(S2)

Redox and aging ① “Targeting Redox and Mitochondria to delay aging and prevent age-related diseases”Forum



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Short CV

Education

Ph.D. in Chemistry, Institute of Chemistry CAS, Beijing, China (2001)
M.S. in Biochemistry, Sichuan University, Chengdu, China (1998)
B.S. in Biochemistry, Sichuan University, Chengdu, China (1995)

Research Interests

My research focuses on understanding the molecular and cellular mechanisms of aging and age-related diseases, particularly exploring the connection between redox signaling and chronic diseases. I also work on developing methods to counteract aging using biochemical interventions.

Professional Experience

Professor: College of Life Science, Sichuan University (2005–Present)
Post-doctoral Scholar: The Jean Mayer USDA Human Nutrition Research Center, Tufts University (2010–2012)
Post-doctoral Assistant: Department of Biochemistry, University of Kentucky (2001–2005)

Selected Publications

1. Liu K, Zhang X, Lester RL, Dickson RC. The sphingoid long chain base phytosphingosine activates AGC-type protein kinases in Saccharomyces cerevisiae including Ypk1, Ypk2, and Sch9. J Biol Chem. 2005 Jun 17;280(24):22679-87.

2. Liu J, Huang X, Withers BR, Blalock E, Liu K, Dickson RC. Reducing sphingolipid synthesis orchestrates global changes to extend yeast lifespan. Aging Cell. 2013 Oct;12(5):833-41.

3. Liu K, Lyu L, Chin D, Gao J, Sun X, Shang F, Caceres A, Chang ML, Rowan S, Peng J, Mathias R, Kasahara H, Jiang S, Taylor A. Altered ubiquitin causes perturbed calcium homeostasis, hyperactivation of calpain, dysregulated differentiation, and cataract. Proc Natl Acad Sci U S A. 2015 Jan 27;112(4):1071-6.

4. Liu B, Wang W, Shah A, Yu M, Liu Y, He L, Dang J, Yang L, Yan M, Ying Y, Tang Z, Liu K. Sodium iodate induces ferroptosis in human retinal pigment epithelium ARPE-19 cells. Cell Death Dis. 2021 Mar 3;12(3):230.

Homepage

Google Scholar Profile： https://scholar.google.com/citations?user=bvAp5agAAAAJ&hl=en



Vitamin A treatment rescues retinal cell-specific deficiencies caused by Leber's hereditary optic neuropathy-linked mtDNA mutation

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Abstract

Leber hereditary optic neuropathy (LHON) is a paradigm for mitochondrial retinopathy due to mitochondrial DNA (mtDNA) mutations. However, the mechanism underlying retinal cell-specific effects of LHON-linked mtDNA mutations remains poorly understood and there has been no effective treatment or cure for this disorder. Using a mice model bearing a LHON-linked ND6P25L mutation, we demonstrated that the mutation caused retinal cell-specific deficiencies, especially in retinal ganglion cells (RGC), rods and Müller cells. Single-cell RNA sequencing revealed cell-specific dysregulation of oxidative phosphorylation and visual signaling pathways in the mutant retina. Strikingly, ND6 mutation-induced dysfunctions yielded abnormal vitamin A (VA) metabolism essential for visual function. VA supplementation remarkably alleviated retinal deficiencies, including reduced fundus lesion and retinal thickness, and increasing numbers of RGCs, photoreceptors and Müller cell neurites. The restoration of visual functions with VA treatment were further evidenced by correcting dysregulations of phototransduction cascade and neurotransmitter transmission and restoring electrophysiological properties. Interestingly, VA supplementation markedly rescued the abnormal mitochondrial morphologies and functions in the mutant retina. These findings provide new insight into retina-specific pathophysiology of mitochondrial retinopathy arising from vitamin A deficiency and mitochondrial dysfunction induced by mtDNA mutation and step toward for therapeutic intervention for LHON and other mitochondrial retinopathies.

Short CV

Dr. Min-Xin Guan graduated with BS in biology from Hangzhou University (previous and current Zhejiang University) in 1983. He did his postgraduate study at the Australian National University (Ph.D. 1993; Advisor: Professor G. Desmond Clark-Walker). Dr. Guan conducted postdoctoral research in the laboratory of Professor Giuseppe Attardi at the California Institute of Technology (1993-1999). In 1999, he started his independent research as an assistant professor at Cincinnati Children's Hospital Medical Center and the University of Cincinnati, eventually becoming a full professor in Division of Human Genetics, Cincinnati Children's Hospital Medical Center and University of Cincinnati College of Medicine in 2011. Since 2011, he has been joining the faculty at Zhejiang University as the founding Director of Institute of Genetics, Dean of College of Life Sciences (2011-2013), Associate Dean of Faculty of Medicine and Pharmaceutical Sciences (2015-2022). Dr. Guan's research interests focus on human mitochondrial genetics and biomedicine. Guan's pioneering work with mitochondrial diseases included the discoveries of the mitochondrial cause of maternally inherited nonsyndromic and aminoglycoside induced hearing loss. Dr. Guan's recent pioneering work were the finding how the interactions between mtDNA mutations and nuclear modifiers manifested the deafness and Leber's hereditary optic neuropathy. Currently, Dr. Guan's lab is focusing on investigating the mechanisms underlying the aberrant mitochondrial tRNA metabolisms including the synthesis, processing, maturation, CCA addition, posttranscriptional nucleotide modification and aminoacylation of tRNA, and their impact on human diseases including deafness, optic neuropathy and hypertension. Dr. Guan has published 184 manuscripts on mitochondrial diseases in the high impact journals. Dr. Guan served as the 4th president of Asian Society of Mitochondrial Research and Medicine (2011-2014).



Cardiolipin Remodeling byALCAT1 Controls the Mitochondrial Free Radical Clock

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Abstract

Aging is the primary cause for all age-related chronic disorders. Although the underlying causes remain poorly understood, aging increases oxidative stress that leads to the production of high level of reactive oxygen species (ROS). ROS cause cumulative damages to mitochondrial membrane proteins, phospholipids, and mitochondrial DNA, which is coined as the "the Mitochondrial Free Radical Aging Clock (MFRAC)" that links mitochondrial dysfunction associated with aging to the development of age-related chronic diseases, such as type 2 diabetes, heart failure, stroke, and neurodegenerative diseases. Despite of intensive efforts in recent years, what controls the MFRAC remains the last frontier in biomedical research. Our pioneering work in the field has identified ALCAT1 as the key regulator of the MFRAC. Our groundbreaking work show that induction of ALCAT1 by ROS accelerates the MFRAC by catalyzing pathological remodeling of cardiolipin with very long polyunsaturated fatty acids (PUFAs). Cardiolipin is the mitochondrial signature phospholipid that is required for every aspect of mitochondrial biology, from membrane structure, oxidative phosphorylation, mtDNA biogenesis, to mitochondrial fusion, fission, and mitophagy. Enrichment of cardiolipin with PUFAs renders cardiolipin highly sensitive to oxidative damages by ROS, leading to mitochondrial dysfunction in metabolic tissues with high energy demand from oxidative phosphorylation. Using mice with targeted deletion of ALCAT1 and Dafaglitapin, an extremely potent and highly selective ALCAT1 inhibitor, we demonstrated that age-related disorders can be treated as one disease, a paradigm-shifting concept for aging research.

Short CV

Dr. Roger (Yuguang) Shi is currently a Joe R. & Teresa Lozano Long Distinguished Chair Professor in Metabolic Biology at Barshop Institute for Longevity and Aging Studies, University of Texas Health Science Center at San Antonio (UTHSCSA). His led a unique career path that encompasses a pharmaceutical research experience at Eli Lilly and Company and academic positions at various academic institutions. His laboratory pioneered the cloning of the PERK kinase, a milestone work in ER-stress and translational control, and several first in class enzymes that catalyze the remodeling of phospholipids, including ALCAT1 and LPGAT1. His longstanding research interests in translation medicine has led to the identification of ALCAT1 as the key enzyme that controls mitochondrial etiology of aging and age-related metabolic diseases, including type 2 diabetes, obesity, diabetic complications, cardiovascular diseases, and neurodegenerative diseases. He is a co-founder of Perenna Pharmaceuticals Inc which successfully developed Dafaglitapin, an extremely potent and highly selective ALCAT1 inhibitor. In preclinical studies, Dafaglitapin demonstrated high efficacy in treating all age-related diseases. This pioneering work has validated a paradigm-shifting concept that all age-related metabolic diseases can be treated as ONE disease. His previous research work at Penn State University uncovered a novel signaling pathway by which GLP-1 regulates glucose-sensing by pancreatic beta cells. During his tenure at Lilly, he helped the company to build a robust drug pipeline for type 2 diabetes and obesity, including the successful launch of Byetta (Exenatide), the first-in-class treatment for type 2 diabetes.



A new mode of mitochondria-lysosome contacts under hypoxia

Zhiyin Song (宋质银)

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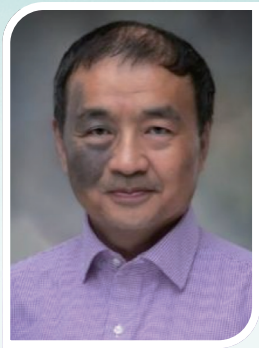
Abstract

Mitochondria physically and functionally interact with lysosomes to regulate cellular metabolism. However, the mode and biological functions of mitochondria-lysosome communication remain largely unknown. Here, we show that hypoxia remodels normal tubular mitochondria into megamitochondria by inducing broad inter-mitochondria contacts and subsequent fusion. Importantly, under hypoxia, mitochondria-lysosome contacts are promoted, and certain lysosomes are engulfed by megamitochondria, in a process we term "megamitochondria engulfing lysosome (MMEL)". Intriguingly, MMEL mediates a new mode of mitochondrial degradation, which we termed "mitochondrial self-digestion (MSD)". Moreover, MSD increases mitochondrial ROS production. Our results reveal a novel mode of crosstalk between mitochondria and lysosomes and uncover a new pathway of mitochondrial degradation.

Key words: mitochondria, lysosome, mitochondria-lysosome contacts, mitochondrial self-digestion

Short CV

Zhiyin Song received Ph.D. degree from University of Science and Technology of China in 2005, he then did her post-doctoral training in California Institute of Technology (Caltech) between 2005-2010. Zhiyin Song became a professor in College of Life Sciences at Wuhan University in between 2010-2023, and in Huazhong University of Science and Technology in 2024. Zhiyin Song's research interest is in the area of mitochondrial dynamics and quality control.



Mitochondrial electron transfer chain (ETC) in aging and longevity

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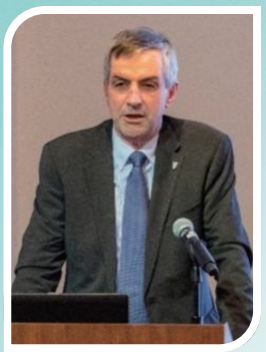
Abstract

Naked Mole-Rats (NMR, Heterocephalus glaber) are the longest-lived rodent species, with a maximum life span of more than 30 years. These long-lived mammals also exhibit delayed aging phenotypes and resistance to age-related pathologies including neurodegeneration. Multiple regulatory pathways have been proposed for the anti-aging mechanisms in NMR including enhanced mitochondrial function and suppressed oxidative stress. In this study, we investigated the assembly of the electron transfer chain (ETC) which constitute the structural base for the regulation of both oxidative phosphorylation and production of reactive oxygen species (ROS), in brains from young and old NMR and C57BL/6 mice. While ETC assembly declined with aging in C57BL/6 mice, we found that NMR displays a robust respiratory chain assembly at older ages in both males and females. Among them, individual complex IV and supercomplexes containing Complex I and III, and complex III and IV showed the most pronounced differences between two species. Our results indicate that a preserved robust assembly of ETC during aging contributes to enhanced mitochondrial oxidative phosphorylation and suppressed oxidative stress which may contribute to the longevity and resistance to age-related pathologies in NMRs.

Key words: electron transfer chain (ETC), aging, assembly, supercomplex

Short CV

Yidong Bai graduated from Fudan University with a bachelor's degree in microbiology and enrolled in graduate program in Shanghai Institute of Cell Biology, CAS before moving to Columbia University for the PhD program. After a postdoctoral fellowship at Caltech, he moved to the University of Texas Health as a faculty member where he is a professor in the department of Cell Systems and Anatomy.



Dysregulation of hydrogen peroxide-mediated responses to contractile activity in skeletal muscle loss associated with aging

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Abstract

Attenuated responses to redox stress are a common feature of aged organisms and these appear to present in skeletal muscle as a reduced ability to respond to contractile activity. Contracting skeletal muscle generates superoxide from membrane-localised NADPH oxidases and this is rapidly converted to hydrogen peroxide (H_2O_2) which acts to stimulate specific adaptive responses. The nature of these responses is extensive and includes increased generation of stress proteins and upregulation of mitochondrial biogenesis. The concentration of H_2O_2 generated within muscle fibres appears insufficient to directly oxidise redox-sensitive proteins in key response pathways and recent data indicate that effector proteins, such as peroxiredoxins, may play a key role in mediating adaptations. These pathways are disrupted in ageing models and conditions of disuse atrophy but appear amenable to manipulation through pharmacological approaches. Understanding the specific mechanisms involved therefore provides a potential route for interventions to maintain muscle mass and function in multiple degenerative skeletal muscle conditions.

Supported by UKRI Medical Research Council, US National Institute on Aging, Versus Arthritis and the UK Space Agency.

Key words: Skeletal muscle, Ageing, Redox Biology, Reactive Oxygen Species

Short CV

Professor Malcolm Jackson is a research professor at the University of Liverpool. He has an extensive research interest in ageing and frailty and has published over 200 original scientific papers. He has previously been Head of the Institute of Ageing and Chronic Disease (2010-15) and Director of the MRC-Arthritis Research UK Centre for Integrated Research into Musculoskeletal Ageing (CIMA), a UK Centre of Excellence. He has also been Treasurer and President of SFRR-Europe and President of SFRR-International. He has editorial roles with Free Radical Biology and Medicine, Redox Biology and Physiological Reviews.

Symposium-3(S3)

Redox and obesity, vascular function and metabolism





Chair: Zhongbing Lu (陆忠兵)

University of Chinese Academy of Science, China

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Short CV

Education:

2003–2006	Institute of Biophysics, CAS	Ph.D., Biophysics
2000-2003	Sichuan University	M.S., Biochemistry
1996-2000	Sichuan University	B.S., Biochemistry

Research Position:

2012-	University of Chinese Academy of Sciences, Professor	
2009-2012	University of Minnesota	Research Associate
2006-2009	University of Minnesota	Postdoctoral Associate

Our research is focused on the role of oxidative stress in the pathogenesis of cardiovascular or metabolic diseases.

Selected Publications:

1.Redox Biology. 2024, 70:103080

2.Acta Pharmaceutica Sinica B. 2023, 13(8): 3352-3364

3.Cellular & Molecular Immunology. 2022, 19(12):1333-1346

4.Redox Biology. 2022, 49:102224

Homepage: http://people.ucas.ac.cn/~0019068?language=en



ER oxi-lipidosis drives MASH pathogenesis

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Short CV

Dr. Xiao-Wei Chen is the Boya Distinguished Professor of Molecular Medicine at the Peking University. He originally obtained his BS and BA from the Peking University, and completed his Ph.D. training with Dr. Alan Saltiel on metabolic biology at the University of Michigan. He then pursued postdoctoral study on genetics and cardiovascular biology in the laboratory of Dr. David Ginsburg, before being recruited back to the Peking University in 2014. Dr. Chen's work focuses on the genetics and cell biology of lipoprotein biology and lipid homeostasis, particularly by elucidating a receptor-mediated export program for the lipoproteins and identifying the long-sought biogenic lipid scramblase. He has also discovered a messenger role of manganese in lipid control, and conceptualized manganese therapy for intensive plasma lipid lowering to reverse exiting atherosclerotic plaques in disease models. He has published ~70 scientific papers and authored two book chapters. He is the recipient of the Young Investigator Award from the Chinese American Diabetes Association and Special Recognition Award from the Society of Heart and Vascular Metabolism, as well as the Earl Stadtman Scholar finalist from the National Institute of Health, USA and the Distinguished Young Scholar Award from the National Natural Science Foundation, China. He serves as an associate editor at the Biochemical Journal and on the editorial boards of Life Metabolism, Journal of Lipid Research, and Cell Metabolism.



Gut microbial enzymes: new targets for intervention in metabolic diseases

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Abstract

Microbial enzymes are key functional molecules of gut microbiota. We have focused on gut microbial enzymes and metabolic diseases. We firstly proposed a new concept of "microbial-host isozymes". By establishing a high-throughput isozyme screening system, we found that microbial-host isozymes are widely present in human. Microbial DPP4, a highly active microbial-host isozyme, can degraded active GLP-1 and cause imbalances in glucose homeostasis, leading to inter-individual differences in host DPP4 inhibitor sitagliptin clinical efficacy. Further, we developed the first microbial DPP4 inhibitor and achieved encouraging results in clinical trials. Microbial bile acid converting enzymes serve as one of the key mediators of interactions between gut microbiota and human. Recently, by constructing a new microbial bile acid mining system based on click chemistry enrichment strategy and untargeted metabolomics, we discovered a new type of microbial bile acids modification—3-acylation modification. Taking advantage of our intestinal strains resource libraries, we identified Bacteroides uniformis as the producer of 3-susCA. Then, we analyzed and verified the key enzyme, BAS-suc, responsible for synthesis of 3-sucCA in B. uniformis based on activity-based protein profiling. Finally, we elucidated the mechanism that the B. uniformis-mediated microbial interactions can improve NASH outcomes through BAS-suc. All in all, we propose a new theory of "cross-kingdom regulation of host homeostasis by gut microbial enzymes".

Key words: metabolic disease; gut microbial enzymes; microbial-host isozymes; bile acid

Short CV

Changtao Jiang, Ph.D., is a tenured full professor at Peking University, serving as the chair of Department of Immunology and the deputy dean of the School of Basic Medical Sciences. He is the recipient of The National Science Fund for Distinguished Young Scholars and XPLORER PRIZE. His work aims at gut microbiota, their microbial enzymes and their impact on metabolic diseases. In short, he pioneers a new theory of "cross-kingdom regulation of host homeostasis by gut microbial enzymes". The research results include the following: proposing a new concept of "microbial-host isozymes"; revealing a novel bile acid modification type and elucidating the role of microbial bile acid converting enzymes in non-alcoholic steatohepatitis (NASH); uncovering a nicotine-degrading gut bacterium and its protective role in NASH. He has published more than 30 SCI papers in Cell (2024), Science (2023), Nature (2022), and other journals, among which, 6 papers are highly cited papers, one paper is picked up by F1000Research and 11 papers are highlighted and by internationally recognized scholars in the field. He is also invited to serve on the editorial board of Cell Metabolism. He has obtained Top 10 Chinese life scientific advances, First prize of Beijing Science and Technology (the first finisher), China Youth Science and Technology Award and C.C. Tan Life Science Awards. He is supported by Major Program of National Natural Science Foundation of China, and State Key Program of National Natural Science of China.



Obstructive sleep apnea syndrome and hepatic lipid metabolism disorders

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Abstract

Obstructive sleep apnea syndrome (OSAS), characterized by chronic intermittent hypoxia (CIH), is an independent risk factor for aggravating non-alcoholic steatohepatitis (NASH). The prevailing mouse model employed in CIH research is inadequate for the comprehensive exploration of the impact of CIH on NASH development due to reduced food intake observed in CIH-exposed mice, which deviates from human responses. To address this issue, we conducted a pair-feeding investigation with CIH-exposed and normoxia-exposed mice. We revealed that CIH exposure aggravated DNA damage, leading to hepatic fibrosis and inflammation. Our analysis of genome-wide association study (GWAS) data also disclosed the association between Eepd1, a DNA repair enzyme, and OSAS. Furthermore, we revealed that CIH triggered selective autophagy, leading to the autophagic degradation of Eepd1, thereby exacerbating DNA damage in hepatocytes. Notably, Eepd1 liver-specific knockout mice exhibited aggravated hepatic DNA damage and further progression of NASH. To identify a therapeutic approach for CIH-induced NASH, we conducted a drug screening and found that Retigabine dihydrochloride suppressed CIH-mediated Eepd1 degradation, leading to alleviated DNA damage in hepatocytes. These findings imply that targeting CIH-mediated Eepd1 degradation could be an adjunctive approach in the treatment of NASH exacerbated by OSAS.

Short CV

Junli Liu, Professor of Endocrinology
Shanghai Jiaotong University School of Medicine
Shanghai Jiaotong University affiliated 6th People`s Hospital
Shanghai Diabetes Institute

Dr. Junli Liu has received the "National Overseas High-level Talents" and "National Science Fund for Excellent Young Scholars" awards, and he also serves as the part-time Chief Editor of Metabolism Open (ESCI, Elsevier). He received his Ph.D. from the Shanghai Institute of Organic Chemistry, Chinese Academy of Sciences in 2009, under the supervision of Dr. Junyin Yuan (Harvard). He completed his postdoctoral training at MIT and Harvard Medical School. Dr. Liu's long-term research focuses on lipid and carbohydrate metabolism in the liver and adipose tissues, and their relationship with metabolic diseases. To date, Dr. Liu has published 21 papers in high-impact journals, including first author in Cell (3 papers) and additional papers as corresponding author in Cell Metabolism (2 papers), Science Translational Medicine, and Advanced Science. His research has been highlighted in 8 editorial reviews in prestigious journals such as Nature Medicine, with one paper recognized as an ESI Highly Cited Paper and another receiving "Most Picked Award" from Cell Press. Moreover, he was also invited to publish a highlight review (Voices) about his research findings in Cell Metabolism. Furthermore, Dr. Liu has obtained approval for one clinical trial, filed 10 patent applications, and been granted 2 patents.



Redox signaling in acute inflammation

Juan Sastre

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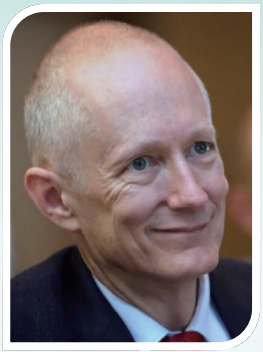
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Abstract

Acute inflammation is characterized by heat, erythema, pain, and swelling of the affected organ or tissue as well as by the respiratory burst of activated leukocytes. Acute pancreatitis has been studied in our lab as a model of acute inflammation. It is an inflammatory process of the pancreatic gland that eventually may lead to a systemic inflammatory response, and in severe cases to death by multiple organ failure. A key early event in pancreatic damage is glutathione depletion, which is transient in mild pancreatitis, but it is maintained over a long time in severe pancreatitis due to inefficient induction of glutamate cysteine ligase. In addition, there is blockade of the trans-sulfuration pathway due to nitration of cystathionine β-synthase. Interestingly, pancreatic inflammation is associated with protein cysteinylation, but not with glutathione oxidation or protein glutathionylation, leading to disulfide stress. Two types of targets of disulfide stress were identified: redox buffers, such as ribonuclease inhibitor or albumin; and redox-signaling thiols that include tyrosine and serine/threonine phosphatases, which markedly affect the inflammatory cascade. Protein cysteinylation is regulated by thioredoxin 1 and thioredoxin-related protein of 14 kDa (TRP14). The inflammatory cascade is also regulated by PGC-1α, which forms a complex with the NF-kB p65 subunit. This inhibitory complex markedly restrains specifically the up-regulation of interleukin 6 that triggers pulmonary inflammatory infiltrate and damage. Obesity causes marked PGC-1α deficiency in the pancreas promoting pulmonary damage. In the course of acute pancreatitis, p53 drives necroptosis of acinar cells via downregulation of sulfiredoxin and peroxiredoxin 3 and enhanced generation of mitochondrial reactive oxygen species. In conclusion, maintained glutathione depletion together with disulfide stress, PGC-1α deficiency, and p53-driven necroptosis decisively contribute to a severe outcome in acute inflammation.

Short CV

Juan Sastre is a Professor in Physiology at the University of Valencia, Spain. The most relevant scientific findings of his research group are related to oxidative stress and redox signaling in acute pancreatitis. He is currently President of European Society for Free Radical Research (SFRR-E) since January 2023. He was General Secretary of SFRR-E from 2013 till 2020. Juan Sastre has published > 150 articles, with currently > 11,000 citations, and an H-index of 58.



Oxidation and enzyme-mediated changes to the artery wall in cardiovascular disease

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Abstract

Cardiovascular disease (CVD) is a leading cause of morbidity and mortality worldwide. Atherosclerosis, a major underlying cause of CVD, is characterized by cholesterol and lipid accumulation in the artery wall and formation of plaques; these develop slowly and can be asymptomatic for decades. Destabilization and rupture of atherosclerotic plaques can be sudden and give rise to vascular occlusion and an acute myocardial infarction or stroke. Despite the importance of plaque stability, the mechanisms underlying rupture are poorly understood, though there is considerable evidence for extracellular matrix (ECM) alterations and a weakening of plaque structure. Compared to stable plaques, rupture-prone plaques typically contain higher levels of activated inflammatory cells that generate potent oxidants, such as hypochlorous acid and nitrating species which can both damage ECM proteins directly, or activate proteases that degrade ECM components.

In this presentation, data consistent with alterations to the nature and type of materials in plaque ECM will be presented, together with modifications to these materials, as determined by immunocytochemistry, immunoblotting and LC-MS/MS studies. Analysis of materials present in, or extracted from carotid plaques, has allowed identification of large numbers of differentially-abundant proteins between soft (rupture-prone) and hard (stable) plaques. Many of the overabundant proteins in soft plaques are involved in inflammation and ECM remodeling. LC-MS analyses have shown the presence of chlorinated, nitrated and oxidized species on ECM components, together with a marked increase in cleaved proteins, as judged by N-terminal proteomics, which allows detection of large numbers of cleaved peptides, consistent with extensive protein damage. The protein identities and the sites of cleavage have been characterized in some cases. These species are present at significantly higher abundance in unstable compared to stable plaques. These data offer a unique insight into the inflammatory and proteolytic mechanisms of plaque destabilization in CVD.

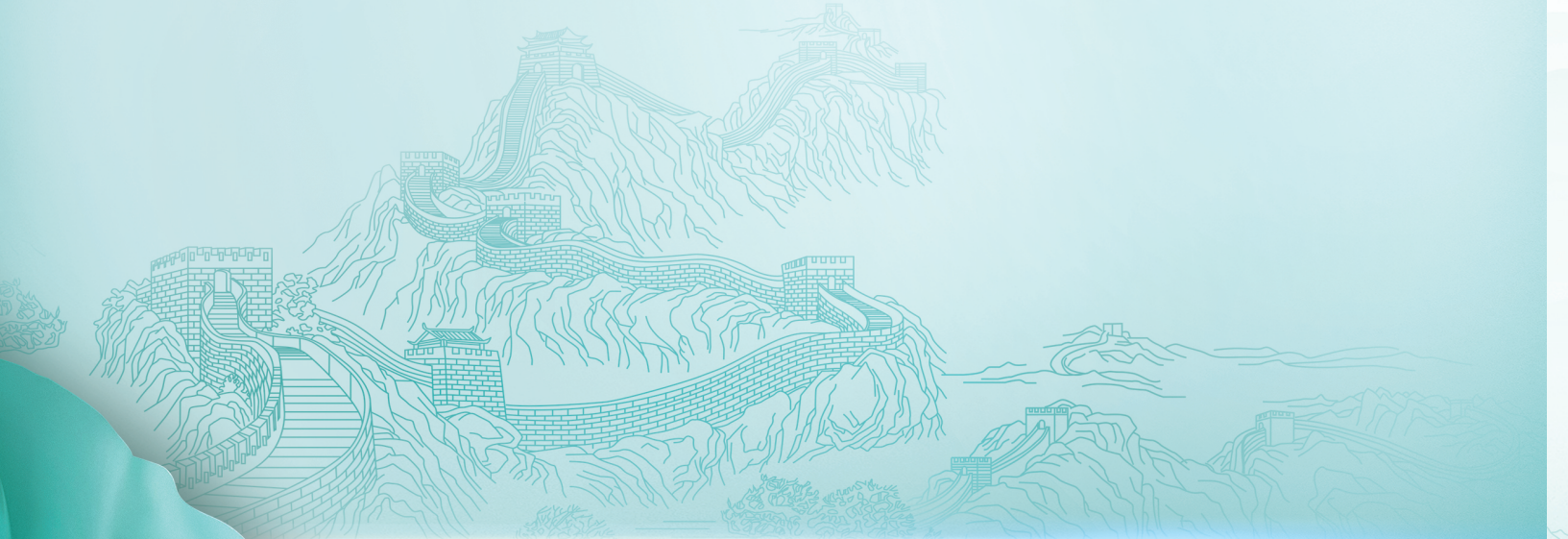
Key words: Atherosclerosis, Proteomics, Protein oxidation, Extracellular matrix, Myeloperoxidase, Peroxynitrite.

Short CV

Prof. Davies works at the University of Copenhagen, Denmark, and was previously Director of the Heart Research Institute, Sydney, Australia. He is joint Editor-in-Chief of 'Redox Biochemistry and Biology'. His research group is focused on the mechanisms and consequences of protein oxidation and extracellular matrix modification and in human disease and particularly cardiovascular pathologies.

Symposium-4(S4)

New approach for precision redox research





Chair: Xiangliang Yang (杨祥良)

Huazhong University of Science and Technology, China

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Short CV

He is the chair professor of the College of Life Science and Technology, Huazhong University of Science and Technology and the director of National Engineering Research Center for Nanomedicine. He is also the chief scientist of the "Nano Research" Project of the Major Scientific Research Program (973) and the leader of the Innovation Team for "Anti-tumor nanomedicine" in the key field of the Ministry of Science and Technology of China. Serving as vice-chairman in several key committees of national associations, including the Nanomedicine Committee of Chinese Pharmaceutical Association (CPA), the Nanomedicine Committee of China Anti-Cancer Association (CACA), the Nanomedicine and Engineering Committee of Chinese Society of Biomedical Engineering (CSBME). His research focuses on the fundamental study and clinical translation of nanomedicine. He has published over 480 peer-reviewed articles in Nature Nanotechnology, Nature Biomedical Engineering, Chemical Society Reviews, Nature Communications, Advanced Materials, etc, with over 20,000 citations and an H-index of 79, and has edited 3 books on nanomedicine and obtained 90 patents approved by NIPA of China. In addition, he has received several awards, including the first-tier prize of Science and Technology Progress Awards of Hubei Province of China. His research results in 4 approved new drug certificates, 17 drug registration approvals, 3 clinical approvals, and several products on the market.



Chair: Kwang Pyo Kim

Kyung Hee University, Korea

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Short CV

EDUCATION

University of Illinois at Chicago, Ph.D. Biochemistry, January, 2002.

Seoul National University, Korea, M.S. Chemistry, February, 1992.

Seoul National University, Korea, B.S. Chemistry, February, 1990.

EMPLOYMENT

Sep 2013-	Kyung Hee University Department of Applied Chemistry Professor
Mar 2013-Aug 2013	Konkuk University Department of Molecular Biotechnology, Professor
Mar 2004-Feb 2013	Konkuk University Department of Molecular Biotechnology, Assistant/Associate Professor
Jan 2002-Feb 2004	Harvard Medical School Dept of Cell Biology, Post-doctoral fellow (Research Advisor : Steven P. Gygi)

SELECTED PUBLICATIONS

1.Paraoxonase-2 agonist vutiglabridin promotes autophagy activation and mitochondrial function to alleviate non-alcoholic steatohepatitis. Shin GC, Lee HM, Kim N, Hur J, Yoo SK, Park YS, Park HS, Ryu D, Park MH, Park JH, Seo SU, Choi LS, Madsen MR, Feigh M, Kim KP, Kim KH. Br J Pharmacol. 2024 Jun 9. doi: 10.1111/bph.16438.

2.Enrichment and MALDI-TOF MS Analysis of Phosphoinositides in Brain Tissue. Le HT, Nguyen DPL, Jung GT, Kim E, Yang SH, Lee SM, Lee EA, Jung W, Kim TW, Kim KP. J Am Soc Mass Spectrom. 2024 Jun 5;35(6):1069-1075. doi: 10.1021/jasms.3c00364.

3.Constitutive activation mechanism of a class C GPCR. Shin J, Park J, Jeong J, Lam JH, Qiu X, Wu D, Kim K, Lee JY, Robinson CV, Hyun J, Katritch V, Kim KP, Cho Y. Nat Struct Mol Biol. 2024 Apr;31(4):678-687. doi: 10.1038/s41594-024-01224-7.

4.CREB-Regulated Transcriptional Coactivator 2 Proteome Landscape is Modulated by SREBF1. Lim JM, Anwar MA, Han HS, Koo SH, Kim KP. Mol Cell Proteomics. 2023 Aug 28;22(10):100637. doi: 10.1016/j.mcpro.2023.100637.



Fluorescence Imaging for the Progression of Oxidative Stress-Related Diseases

Bo Tang (唐波)

Laoshan Laboratory, China

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Abstract

Oxidative stress is an imbalance between oxidation and antioxidant processes within an organism, which can lead to damage of biomacromolecules and become one of the important factors in aging and disease. During the process of oxidative stress, the levels of reactive molecules within cells fluctuate, closely correlated with the occurrence and development of diseases. Therefore, precise detection of these biomolecules has attracted wide attention. In response to their low concentrations, continuous changes, short half-lives, and characteristics of interaction and conversion, we have developed a series of novel fluorescent probes. We established a new method for ultra-high sensitivity, real-time in situ, dynamic, and simultaneous imaging of cellular reactive molecules (such as reactive oxygen species, enzymes, etc.), obtaining important information on the involvement of these reactive molecules in the oxidative stress process of organisms, as well as their regulation of the progression of cardiovascular diseases and brain diseases.

Key words: ROS; fluorescence imaging; biomacromolecules

Short CV

Bo Tang, director of the Department of Health Oceans and Sustainable Resource Utilization Research at Laoshan Laboratory, and professor at Shandong Normal University. He serves as a member of the Science and Technology Committee of the Ministry of Education, was nominated for the First National Outstanding Science and Technology Worker Award (the only one in Shandong Province), serves as the chief scientist of the 973 Program, is a recipient of the National Distinguished Young Scientists Fund, and is a national-level candidate of the New Century National Talent Project and the "Ten Thousand Talent Program" of the Thousand Talents Plan. The team he leads has been selected for the Ministry of Education's Changjiang Scholars and Innovative Team Development Plan, and is recognized as the Huang Danian-style Teacher Team at the national level, the first outstanding innovation team in Shandong Province, and an excellent innovation team of the Ministry of Science and Technology. He is primarily engaged in research on the synthesis of molecular and nanoscale fluorescent probes and their applications in biological imaging, as well as marine chemical biology and the high-value utilization of marine resources. He has led multiple national-level research projects, including the 973 Program, National Natural Science Foundation Key Projects, and major scientific instrument development projects. He has published over 400 SCI-indexed papers in journals such as Nat. Synth., Nat. Commun., Chem. Soc. Rev., J. Am. Chem. Soc., Angew. Chem. Int. Ed., Adv. Mater., with more than 43,000 citations. He has filed for over 80 national invention patents. As the principal investigator, he has been awarded one second prize of the National Natural Science Award, two second prizes of the National Science and Technology Progress Award, two first prizes of the Shandong Natural Science Award, two first prizes of the Shandong Science and Technology Progress Award, and one first prize of the Shandong Technological Invention Award.



Primate Phenotype and Genetic Analyses – From Basic Research to Clinical Applications

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Abstract

Non-human primates (NHPs) have many advantages over other experimental animals in advancing biomedical research, especially the modeling of neurodegenerative and infectious diseases, and in understanding human beings, given the high degree of similarity in respect to genetics, anatomy, physiology, behavior, emotion, and cognitive function. NHPs constitute irreplaceable and in many ways superior models compared with common experimental animals such as rodents. Using NHPs to clarify the mechanisms underpinning genotypes and phenotypes will undoubtedly improve our understanding of complex traits and human diseases, as well as the responses of biological processes to environmental factors. On this point, NHPs constitute the perfect living template for us humans to understand ourselves. The establishment of the National Major Scientific and Technological Infrastructure for Primate Phenotype and Genetic Research provides researchers with a comprehensive and systematic platform that supports the translation from basic research to clinical applications. The creation of such facilities not only accelerates scientific research on primates but also offers new directions for addressing some of the challenges currently faced in life sciences and medical research.

Key words: Non-human primate, phenotype, genotype, clinical medicine, facility

Short CV

Dr. Yong-Gang Yao is the director general and principal investigator of the Kunming Institute of Zoology, Chinese Academy of Sciences (CAS). He obtained his bachelor's degree from Anhui Normal University in 1997 and his Ph.D. from the Kunming Institute of Zoology in 2003. He joined the School of Medicine at Johns Hopkins University as a post-doc in February 2003 and served as a visiting fellow at the National Heart, Lung, and Blood Institute (NHLBI), National Institutes of Health (NIH), in October 2004. He joined the Kunming Institute of Zoology as a principal investigator in December 2007.

Dr. Yao is involved in researching the genetic basis and molecular mechanisms underlying human diseases, with a particular focus on Alzheimer's disease. Furthermore, his team is also investigating the biology of the Chinese tree shrew, which is gaining prominence as a valuable laboratory animal. Currently, Dr. Yao is leading the establishment and construction of the National Research Facility for Phenotypic and Genetic Analysis of Model Animals (Primate Facility).

To date, Dr. Yao has published more than 300 peer-reviewed research articles and commentaries in various SCI-indexed journals, including Am J Hum Genet, PNAS, Autophagy, Alzheimers Dement, Natl Sci Rev, and Cell Discov. As of Auguest 30, 2024, his work has been cited over 11300 times (Web of Science), with an h-index of 54. He was recognized as one of the 2020-2023 ELSEVIER most-cited Chinese researchers. In addition to his research, Dr. Yao holds various editorial positions, including editor-in-chief of Zool Res and Zool Res Divers Conserv, associate editor of J Hum Genet, and editorial board member of J Genet Genomics and Mol Cell Neurobiol. He has received multiple awards, including the State Natural Science Award of China (second class) and Zhuliyuehua Award for Outstanding Teachers of the University of Chinese Academy of Sciences.



Quantitative metabolomics for redox biology and medicine

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Abstract

Human metabonome contains more than 20 thousand metabolites with a huge concentration dynamic range, diverse properties, matrices and many different functions. Quantitative metabolomic analysis is essential for understanding the molecular aspects of mammalian biology, physiology and pathophysiology of various diseases hence redox biology and medicines. During last decades, metabolomics science has made huge progress in both technical and application areas. To achieve accurate quantitative metabolomic analysis, however, developing efficient novel analytical technologies remains to be one of the most urgent and extremely challenging tasks. NMR and MS are the dominant analytical tools with complementary information from them. This presentation will deal with the requirements of quantitative metabonomics and strategies to fulfill such tasks followed with some recent methodological advances. We will also discuss the major challenges metabolomic analysis is facing and possible strategies to overcome such problems with some important applications related to redox biology and medicines.

Key words: Quantitative metabolomics, elementomics, redox homeostasis

Short CV

Dr. Huiru Tang is a Distinguished Professor (metabonomics and systems biology) in Fudan University (School of Life Sciences and Zhangshan Hospital). He has been developing novel metabonomics methods and studying metabolic aspects of important diseases including obesity and complications as well as the symbiotic interactions between mammalian hosts and their gut microbiota for decades. After earned his PhD in chemistry from University of London in 1994, he worked at Institute of Food Research, UK, and Imperial College London as a Senior Scientist before joining the Chinese Academy of Sciences in 2005 as a professor. He joined Fudan University in 2014. He authored over 220 peer-reviewed papers with over 14000 citations (h-index: 67). He has been a Fellow of the Royal Society of Chemistry since 2005 and received an Award for Outstanding Young Scholars in 2008. He is now editorial board members (or Associated Editors) for numerous international scientific journals, and is the President of the Metabolomics Society of China.



Near-infrared xanthene dyes and *in vivo* ROS sensing

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Abstract

Near-infrared light exhibits deep tissue penentration depth and is sought after for biomedical applications, i.e., intraoperative guidance, and photo dynamic therapy and more. The current gold standard is the indocyanine green (ICG) developed and approved in 1950s. ICG absorbs at 780 nm and is not stable chemically and photochemically. Development of bright and stable fluorophores absorbing and emitting beyond 800 nm is challenging.

In this presentation, our recent progress in development of near-infrared xanthenoid dyes (EC/ESi dyes) will be discussed. Briefly, benzannulation of xanthene scaffold is the key to redshift the spectral wavelengths and steric is the key to improves their resistance toward oxidation and bleaching. Currently, we have developed a series of EC/ESi dyes maximally absorbing at 740 nm, 780 nm, 820 nm, 835 nm, 860 nm, 880 nm, 920 nm, 1060 nm, and 1210 nm, respectively. Those dyes have the potentials for practical applications. Proof-of-concept biological *in vivo* imaging with mouse models will be presented. We further developed ROS-sensitive near-infrared fluorescent probes based on EC and ESi dyes. Their proof-of-concept *in vivo* applications were showcased with APAP-induced liver damages in mouse models.

Key words: Near-Infrared, Fluorescence, Redox, Sensing, *In vivo*

[1] J. Am. Chem. Soc., 2023, 145, 12013–12022.

[2] J. Am. Chem. Soc., 2022, 144, 14351–14362.

[3] Angew. Chem. Int. Ed. 2017, 56, 2979-2983.

[4] Angew. Chem. Int. Ed., 2024, e202402949.

[5] J. Am. Chem. Soc., 2022, 144, 2114-2119.

Short CV

Prof. Youjun Yang got his BS (2002) from University of Science and Technology of China and his PhD (2007) with Prof. Robert M. Strongin at Louisiana State University. Then, he joined the Anslyn group at the UT Austin as a postdoc. In 2010, he joined the faculty of the school of pharmacy, ECUST. His research falls within the area of dye chemistry. Current interests include NIR fluorescent dyes, molecular probes, photo-triggered drug release, and antibiotic xanthene dyes. He received the Czarnik Emerging Investigator Award (2018) and was supported by the NSFC Excellent Young Scientists program (2018).



Leucine 305 and 309 residues contribute to the formation of two human NRF2 bands in SDS-PAGE

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Abstract

Human NRF2 cDNA consists of 1,815 base pairs and encodes 605 amino acids. Therefore, human NRF2 is expected to appear around~65 KDa in SDS-PAGE based on the prediction of its molecular weight. However, human NRF2 appears around 110 KDa and exhibits two bands in SDS-PAGE. We have identified that leucine 305 and 309 residues existing in the Neh7 domain of human NRF2 are responsible for the formation of two bands in SDS-PAGE. While leucine 305 in primates is substituted into isoleucine in rodents, leucine 309 is conserved throughout the species. Moreover, we have identified that leucine 309 belongs to the LxxLL motif, which is essential for the binding to RXRα. We speculate that the intermolecular or intramolecular interaction of leucine 305 and 309 residues contributes to the formation of two bands of human NRF2 in SDS-PAGE.

Short CV

1991-2000 College of Pharmacy, Seoul National University, Korea (B.S. & M.S.)

2001-2007 Ernest Mario School of Pharmacy, Rutgers University, USA (Ph.D.)

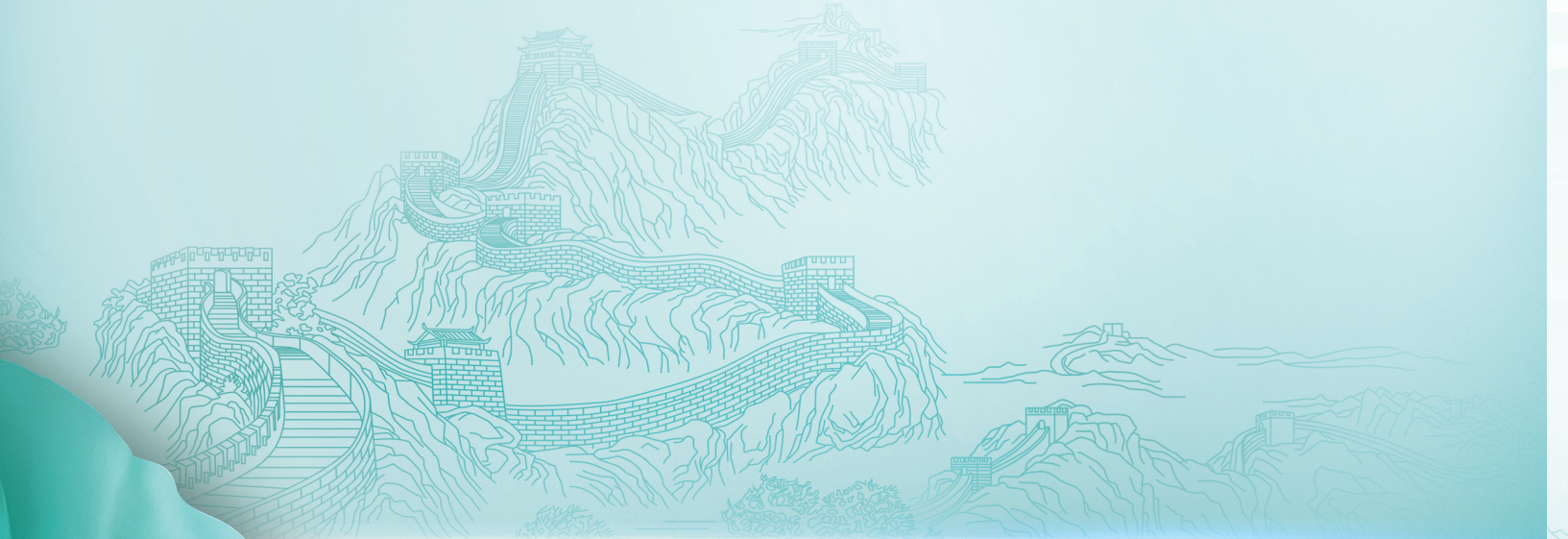
2007-2008 Post-doc, University of North Carolina at Chapel Hill, USA

2008-2010 Post-doc, The Hormel Institute, University of Minnesota, USA

2010-Present Professor, College of Pharmacy, Dongguk University, Korea

Symposium-5(S5)

Discovery of new molecules in redox network





Chair: Qiang Zhao (赵强)

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Short CV

Dr. Qiang Zhao received B. Eng degree from Northwestern Polytechnical University in 2001, and Ph.D. degree in Materials Science & Engineering from Tianjin University (China) in 2006. After three years of postdoctoral research at City University of Hong Kong, he joined College of Life Sciences, Nankai University (China) as associate professor in 2009, and was promoted to full professor in 2014. Dr. Zhao is the Director of Tianjin Key Laboratory of Bioactive Materials as well as the PI of State Key Laboratory of Medicinal Chemical Biology. He is the recipient of Distinguished Young Scientist Program of NSFC (2019) and Excellent Young Scientist Program of NSFC (2015). Currently his research interest focuses on biomaterials and regenerative medicine, including the development of novel biomaterials and therapeutic techniques for the treatment of cardiovascular diseases. He was awarded the First Class Prize of Natural Science Award of Tianjin (2019) as well as the Second Prize for Progress in Science and Technology of Tianjin (2016, 2021), and has authored over 100 peer-reviewed research papers (including Sci Transl Med, Nat Chem Biol, Nat Commun, Sci Adv, Cell Rep, Elife, Adv Mater, Circ Res, Adv Sci, J Am Soc Nephrol, Biomaterials, etc.), 6 book chapter, and >10 patents granted or pending.



NRF1 and NRF2 coordinate osteoclastogenesis and bone remodeling via ROS-dependent and independent mechanisms

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Abstract

While nuclear factor erythroid 2-related factor 1 (NRF1, also known as NFE2L1) and its CNC-bZIP family member NRF2 transcriptionally coordinate multiple stress responses via regulating a variety of antioxidant and cytoprotective genes, they play distinct roles in maintaining various cell metabolism and function, including bone remodeling and homeostasis. In the present study, we aimed to understand the molecular mechanisms underlying osteoporosis induced by aging, estrogen deficiency and various environmental stresses, focusing mainly on the roles of NRF1 and NRF2 in osteoclastogenesis. By employing a candidate gene associate study using the UK biobank cohort we found that multiple variants of human NFE2L1 gene are associated with heel bone mineral density. Knockout of all isoforms of Nfe2l1 transcripts specifically in the myeloid cell lineage in mice resulted in increased osteoclast number and activity, decreased bone mass and accelerated bone loss induced by ovariectomy and aging. Mechanistic investigations using bone marrow-derived osteoclast progenitor cells and RAW 264.7 cells revealed that deficiency of Nfe2l1 leads to accelerated and elevated osteoclastogenesis, which is attributed to enhanced expression of Nfatc1/α, a master regulator of osteoclast differentiation. Further studies postulated a new mechanism that NRF1 functions as a key factor controlling the transcription of Nfatc1/α and osteoclast differentiation in an isoform-specific manner. Specifically, long isoforms of NRF1 (L-NRF1) positively regulates the transcription of Nfatc1/α and promotes osteoclast differentiation, whereas the short isoform NRF1-453 competes with L-NRF1 for the same DNA binding site(s) to suppress the transcription of Nfatc1/α, highlighting that NRF1 is crucial in fine-tuning osteoclastogenesis and thus bone homeostasis. In contrast, ablation of Nrf2 globally or myeloid-specifically in mice resulted in a relatively minor phenotype in bone metabolism under non-stressed condition, but exacerbated osteoclast activation and bone loss induced by prolonged exposure to multiple environmental oxidative stressors, including cadmium (Cd) and arsenic, suggesting that NRF2-dependent osteoclast homeostasis plays a crucial role against the oxidative stressors-induced osteoclast overactivation and osteoporosis. Mechanistic in vitro studies revealed that Nrf2 deficiency aggravates the osteoclast differentiation provoked by low levels of Cd exposure, in which ROS-mediated L-NRF1 activation plays a crucial role in coordinating Nfatc1/α expression, osteoclastogenesis and thus bone remodeling and homeostasis. Together, NRF1 and NRF2 coordinatively respond to environmental oxidative stress and orchestrate osteoclastogenesis and bone remodeling. Mismatched and/or imbalanced activation of L-NRF1 and NRF2 in response to environmental cues may disrupt redox-sensitive signaling leading to impaired bone metabolism and function, bone remodeling in particular.

Key words: NFE2L1, osteoclast, NFATc1, osteoporosis, NRF2

Short CV

Jingbo Pi, MD, Ph.D.

Dr. Pi received M.D. (1990) and M.S. on Occupational Health (1995) from China Medical University, and Ph.D. in Medical Sciences from The University of Tsukuba, Japan in 2002. He had postdoctoral training at NIEHS, USA (2002-2004) and The Hamner Institutes for Health Sciences, USA (2004-2006). He worked as a Research Investigator, Assistant Investigator and Associate Investigator at The Hamner Institutes for Health Sciences (2006-2013). In 2013, Dr. Pi was recruited as a professor of China Medical University, and since then he has been serving as the Dean of School of Public Health. In 2008, Dr. Pi received the Outstanding New Environmental Scientist (ONES) Award, NIEHS, USA. Dr. Pi's research focus is on environmental oxidative stress and metabolic disorders. His research has been funded by NIDDK (USA), NIEHS (USA), Nature Science Foundation of China and the Ministry of Science and Technology, China. He has authored/co-authored over 200 peer-reviewed papers/book chapters with more than 10,000 citations. Dr. Pi has served as a board member and president/vice president of Stem Cell Specialty Section, SOT, USA and an advisor and subgroup co-chair of IARC/WHO Monographs. Currently, he is an Associate Editor of Toxicology and Applied Pharmacology and Toxicology Reports, and also serves as vice president of multiple Specialty Sections of Chinese SOT. In addition, he functions as the director of the Key Laboratory of Environmental Stress and Chronic Disease Control and Prevention, Ministry of Education, China.



HSF1 regulates nuclear/cytoplasmic and mitochondrial proteotoxic stress responses

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Abstract

Dysregulation of protein homeostasis (proteostasis) in different organelles is associated with age-related diseases including neurodegenerative disease and cancer. To cope with proteotoxic stresses, cells are equipped with adaptive mechanisms called proteotoxic stress response (PSR). Among these, the heat shock response (HSR) is evolutionarily conserved and is characterized by the induction of heat shock proteins (HSPs), which assist protein folding. HSR is regulated by heat shock transcription factor 1 (HSF1) in human cells and maintains proteotoxic capacity in the nucleus and cytoplasm. We recently showed that HSF1 also regulates mitochondrial unfolded protein response (UPRmt) and maintains mitochondrial function, which is related with redox homeostasis. To understand regulatory mechanisms of HSR, we have been studying regulation of HSF1-transcriptional complexes under proteotoxic stress conditions. I introduce mechanisms of heat shock gene transcription involving the stress-induced HSF1 complex formation and changes in chromatin states and show that aberrant regulation of these mechanisms is associated with cancer progression.

Key words: heat shock, proteostasis, mitochondria, transcriptional complex, cancer

Short CV

1981-1987 Undergraduate at Tottori University School of Medicine, Japan;

1987-1991 Graduate at Tottori University;

1991-1993 Northwestern University, IL, USA, Postdoctoral fellow;

1993-1998 Chest Disease Research Institute, Kyoto University, Assistant Professor;

1998-2000 Institute for Frontier Medical Sciences, Kyoto University, Assistant Professor;

2000-present, Yamaguchi University School of Medicine, Professor.



Grasip the void of Redox
——the new relic of Tissue Bank

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Abstract

Oxidative stress plays a critical role in the development and progression of various diseases, including cancer, aging, and metabolic disorders. Our team leverages a multi-omics platform, integrating proteomics, metabolomics, and genomics, to investigate the molecular mechanisms of oxidative stress, particularly its regulatory roles in specific disease models. Studies have shown that oxidative stress responses at the metabolic level differ among individuals, especially in energy metabolism pathways in insulin-resistant subjects, highlighting new avenues for personalized therapeutic strategies.

Our team members previously contributed to a study on metformin, which revealed the mechanism by which physiological concentrations of metformin activate AMPK through a lysosomal pathway. High concentrations of metformin inhibit mitochondrial activity and increase intracellular AMP levels, while physiological doses activate AMPK via a receptor-mediated pathway. The study also demonstrated that metformin regulates ROS (reactive oxygen species) in a dose-dependent manner, either reducing or increasing ROS levels, underscoring its dual-edged role. This finding provides new insights for further exploration of metformin's regulation of redox homeostasis.

Currently, our team is investigating the potential of N-acetylcysteine (NAC) in improving poor engraftment following hematopoietic stem cell transplantation. Preliminary results indicate that NAC enhances hematopoietic stem cell function by scavenging excess ROS in the bone marrow, promoting endothelial progenitor cell regeneration, and alleviating platelet engraftment delays.

In the future, we plan to build upon the findings of metformin's systemic regulation of ROS, integrating multi-omics and pharmacokinetics research to explore its mechanisms in various organs, tissues, and specific cell types. By combining multi-omics data, we aim to elucidate its impact on redox homeostasis and construct a network of cell fate regulation to advance research in disease diagnosis and treatment. Additionally, we will expand the application of our multi-omics platform to investigate the potential mechanisms of redox regulation in other diseases, further uncovering its role in various pathological processes. Through this platform, we aim to translate these research findings into clinical applications and actively seek collaboration with experts from other fields to foster innovation in clinical practice and disease research.



Structural library and visualization of endogenously oxidized lipids

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Abstract

Recently, oxidized phospholipids have been reported to be involved in various diseases. For example, lipid peroxides induce a new cell death form, "ferroptosis," and epoxidized ω3 fatty acids, an oxidized metabolite, are engaged in worsening allergies. In addition, the complex between lipid peroxide-derived aldehyde and protein is involved in angiogenesis. Thus, although the importance of oxidized phospholipids is widely recognized in the induction of inflammation and cell death, the number of oxidized phospholipids available or detectable is limited. This lower number would be due to the lack of appropriate detection techniques.

Here, we have developed a fluorescent probe to detect "lipid-derived radicals," key molecules during the chain reaction of lipid peroxidation. Furthermore, since this probe can covalently bind to lipid-derived radicals, we have constructed an LC/FL/HRMS/MS system and have successfully analyzed the structures of 132 lipid-derived radical species1). In addition, the involvement of lipid-derived radicals in the vitamin K cycle has been clarified recently.

Next, a non-targeted analysis of phosphatidylcholine-derived oxidized lipids (oxPCs) was performed using a high-resolution mass spectrometer, and a library of 465 oxPCs was constructed 2). Furthermore, we detected 70 kinds of oxPCs in mice with acetaminophen-induced acute liver failure, and mass imaging of oxidized lipids was successfully performed.

In this symposium, I would like to introduce our recent research, including the detection and structural analysis of oxidized phospholipids and their application using animal models.

<Yamada K, et al. Nat Chem Biol. 2016; 12:608-613. Matsuoka Y, et al. Nat Commun. 2021; 12:6339.>

Key words: Lipid radicals, Oxidized Phospholipids

Short CV

Ken-ichi Yamada is a Professor at the Faculty of Pharmaceutical Sciences, Kyushu University, Japan. He studied ESR and MRI imaging at Kyushu University and received his Ph.D. in Pharmaceutical Sciences from the Faculty of Pharmaceutical Sciences, Kyushu University, Japan. He did his postdoctoral work at NCI/NIH, USA, for two years, working on magnetic resonance imaging and radiation biology. Specific interests in Yamada's lab include the detection and regulation of oxidized lipids.



Regulation of protein-protein interactions as a new paradigm in drug discovery: Targeting the oncogenic role of E74 Like ETS transcription factor 3 (ELF3) through modulation of its protein-protein interaction

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Abstract

Protein-protein interactions (PPIs) form intricate networks essential for maintaining homeostasis under normal physiological conditions. Dysregulated PPIs are often implicated in the pathogenesis of various diseases, making them critical targets in drug discovery. However, disrupting these dysregulated PPIs remains challenging due to the large and shallow nature of PPI interfaces, which typically lack well-defined binding sites.

While some protein or peptide-based PPI inhibitors have been developed, their clinical utility is limited by drawbacks such as poor cellular internalization, low bioavailability, and high immunogenicity. Consequently, there is an ongoing effort to identify small molecule PPI regulators capable of binding to PPI interfaces with high specificity and affinity. Despite these efforts, only a limited number of small molecule PPI modulators have advanced to clinical use.

In our research, we have focused on developing small molecule modulators for specific PPIs by identifying 'hotspots'—small regions within PPI interfaces that are primarily responsible for the binding affinity between two proteins. Using a comprehensive approach that combines biochemical and analytical techniques with in silico structural studies, we have successfully identified small molecule inhibitors targeting these PPI hotspots. We have also elucidated their mechanisms of action and demonstrated their anticancer efficacy both in vitro and *in vivo*.

Short CV

2005.3-presnt: Assistant Professor, Associate Professor, and Professor, College of Pharmacy & Graduate School of Pharmaceutical Sciences, Ewha Womans University

2021.7- 2027.2: Director, Ewha Drug Development Research Core Center

2021.8- 2023.7: Chairman, Graduate School of Pharmaceutical Sciences, Ewha Womans University

2017.8- 2019.7: Associate Dean, College of Pharmacy, Ewha Womans University

2019: Director, Division of Pharmaceutical Analysis, Pharmaceutical Society of Korea

2023- 2025: Korea Drug Development Fund (KDDF), Investment Review Committe

Symposium-6(S6)

Redox modification of biomacromolecules





Chair: Zhonghong Gao (高中洪)

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Short CV

1998, PhD of Huazhong University of Science & technology; 2000, associate professor of Huazhong University of Science & technology; 2001.9.-2002.8., visiting scientist in The University of Texas, Medical School at Houston, U.S.A; 2004, professor of Huazhong University of Science & technology

Research interests

Extraction, identification, bioactivities (particularly on anti-oxidative and anti-nitrative activities) of nature products; the mechanism and the cellular effects of the redox-based post-translational modification of proteins, particularly on protein oxidation and tyrosine nitration; nanotoxicology based on oxidative stress. As first author or corresponding author, professor Gao published more than 60 papers on peer reviewed international journals, and these papers have got more than 2500 citations.



Biology and pharmaceutical development of S-nitrosylation

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Abstract

There are several approaches to delivering exogenous NO for the regulation of neuronal and cardiovascular functions. S-nitrosylation technology involves the covalent attachment of an NO group to the thiol group (-SH) of a cysteine residue in a protein, forming an S-nitrosothiol (SNO). This reaction can occur enzymatically, non-enzymatically, or be catalyzed by metal centers. Reversible S-nitrosylation of protein Cysteine residues has emerged as an important post-translational modification across a wide variety of living organisms, from bacteria to mammals, resulting in NO-like bioactivity.

We have synthesized and developed S-nitrosylated captopril (CapNO), a compound with significant pharmaceutical potential. Due to the inherent instability of the S-NO bond in CapNO, its solid form had never been successfully obtained before. Here, we present groundbreaking evidence that we are the first to synthesize CapNO crystals and formulate them as a cyclodextrin inclusion complex. By incorporating a single water molecule within the molecular structure, we stabilized the S-NO bond through ionic binding. The cyclodextrin inclusion complex further enhanced stability by providing spatial steric hindrance, protecting the vulnerable S-NO bond from degradation. This innovative approach overcomes the technical challenges of producing large quantities of stable CapNO crystals for pharmaceutical use. We have thoroughly characterized the physicochemical properties of CapNO.

Intravenous administration of CapNO in rats resulted in increased cerebral and vascular blood flow, along with an acute reduction in mean arterial pressure. In rats with acute hypertension induced by an iNOS inhibitor, CapNO significantly counteracted the hypertensive effects. Bolus injections of CapNO into the left atrium of awake dogs produced immediate epicardial vasodilation, increased coronary diameter and blood flow, a transient decrease in aortic pressure, and a transient increase in heart rate and left ventricular dP/dt. In contrast, intravenous administration of captopril alone did not produce these effects.

Subchronic treatment of spontaneous hypertensive rats (SHR), Dahl salt-sensitive hypertensive rats (SS/Jr), and two-kidney, one-clip Goldblatt hypertensive rats with oral CapNO significantly reduced mean arterial

pressure to normotensive levels without adverse effects on blood chemistry or hematology tests. Based on these results, the no observed adverse effect level (NOAEL) for CapNO can be safely established at 100 mg/kg/day. CapNO, which combines the properties of a nitric oxide donor and an angiotensin-converting enzyme (ACE) inhibitor, shows promise for beneficial effects on both the neuronal and cardiovascular systems.

Key words: NO, S-nitrosylation, cardiovascular effects

Short CV

Dr. Jia is well known for his pioneering research on 1) Nitric oxide (NO) and S-nitrosylation molecular biology and pharmaceutical innovation; 2) Cancer metastasis chemoprevention that prevents circulating tumor cells (CTCs) from germination into metastatic niches, and thus eliminating the root cause of cancer metastasis; 3) Innovative bionanomaterials used for molecular delivery, biosensing and precisely targeting blood CTCs; and 4) Biochemical basis of nutraceuticals used for long-term prevention and treatments of major diseases.

Jia received his Ph.D. in 1994 from the State University of New York, USA, advised by Robert Furchgott (the 1998 Nobel Laureate). He joined the prestigious National Cancer Institute/NIH, USA, at the tenure position (GS14/9) managing 6.3 millions of pharmacological contract research before recruited by "The China Recruitment Program of Global Experts". He then holds the rank of Distinguished Professor at the Fuzhou University and the Minjiang University, China, respectively. He is a Fellow of American Association of Pharmaceutical Scientists (AAPS; 2011) and a Section Chair of the AAPS (2009-2012), a Member of the International Eurasian Academy of Sciences (IEAS; 2020), and a Member-at-Large of the Chinese Chemical Society (2021), the Founding Chair of the Intelligent Functional Pharmaceutics Section of Chinese Pharmaceutical Association (CPA; 2021-) and the Council Member of CPA. He is the Associates Editor for Current Drug Metabolism, and for Drug Metabolism and Bioanalysis Letters (2023-), as well as Editors for other four Journals. He received many prestigious awards and recognitions. He dedicates to translating scientific discoveries into unmet needs of major diseases. He led teams to developing 3 candidate molecules for clinical trials. His 240+ publications are highly relevant to molecular biology and translational research with citations >16700, h-index 62 and i10-index 191 (Google scholar). He has collaborated with scientists from Switzerland, Sweden, Russia, the USA, the UK, Australia, South African.



Lipid Peroxidation and Cardiovascular Disease

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Abstract

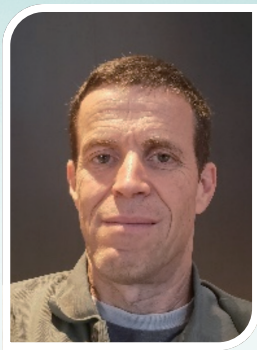
Background: As an iron-dependent form of regulated cell death caused by lipid peroxidation, ferroptosis has been implicated in ischemic injury but the underlying mechanisms in acute myocardial infarction (AMI) remain poorly defined. Acetaldehyde dehydrogenase 2 (ALDH2) catalyzes detoxification of lipid aldehydes derived from lipid peroxidation and acetaldehydes from alcohol consumption. The Glu504Lys polymorphism of ALDH2 (rs671, ALDH2 *2), affecting around 8% world population and 40% East Asians, is associated with increased risk of MI. This study aims to investigate the role of ALDH2 and ferroptosis in MI. Methods: A Chinese cohort of 177 acute heart failure patients with ALDH2 and ALDH2*2 were enrolled. MI mouse model of left anterior descending coronary artery ligation (LAD) was conducted on wild type, ALDH2*2, and mice with cardiomyocyte-specific knock down of eukaryotic translation initiation factor 3 subunit E (eIF3E) by adeno-associated virus. Lipid peroxidation products were measured by mass spectrometry-based lipidomics and metabolomics in human plasma and in mouse serum and heart tissues. Results: Human ALDH2 *2 carriers exhibit more severe heart failure post-AMI with features of ferroptosis in blood samples in lipidomic analysis, including increased levels of multiple classes of oxidized phospholipids, serum heme, and decreased levels of antioxidants, such as Coenzyme Q-10 (Co-Q10) and tetrahydrobiopterin (BH4). Similar features were observed in MI mouse models of ALDH2 *2, whereas ferroptosis inhibition by Fer-1 significantly improved heart functions and reversed ferroptosis markers. Importantly, ALDH2*2 led to significantly decreased protein levels of ALDH2, whereas ferroptosis related proteins including Transferrin receptor (TFRC), Acyl-CoA synthetase long chain family member 4 (ACSL4), and Heme oxygenase 1 (HMOX1) were upregulated specifically in the infarct heart tissues. Mechanistically, ALDH2 physically interacted with eIF3E to modulate translation of critical proteins involved in ferroptosis, and ALDH2 deficiency in ALDH2 *2 mutant predisposes cardiomyocytes to ferroptosis by promoting Tfrc/Acsl4/Hmox1 translation. Consistently, cardiomyocytes-specific eIF3E knock down restored ALDH2 *2 cardiac function by attenuating ferroptosis in MI. Conclusions: ALDH2 *2 aggravates acute heart failure in MI through promoting cardiomyocytes ferroptosis, and targeting

ferroptosis may be a potential therapeutic target for treating AMI, especially for ALDH2 *2 carriers.

Key words: Lipid peroxidation, Ferroptosis, Myocardial Infarction

Short CV

Dr. Huiyong YIN is a tenured Professor in the Department of Biological Sciences and Associate Dean （Research） of the Jockey Club College of Veterinary Medicine and Life Sciences at City University of Hong Kong. He also serves as the Associate Dean for Research for JCC and Chair of College Research Committee. Before joining CityU, Prof. Yin was the Distinguished Principal Investigator and Group Leader of Lipid Metabolism in Human Nutrition-related Diseases at Shanghai Institute of Nutrition and Health, Chinese Academy of Sciences, Shanghai, China. He was also the Distinguished Adjunct Professor in School of Life Sciences and Technology in ShanghaiTech University since 2013. Prof. YIN is one of the leading scientists in the field of redox regulation of glucose and lipid metabolism in human metabolic diseases including atherosclerosis, liver cancer, hyperuricemia and gout (http://www.cityu.edu.hk/bms/profile/huiyongyin.htm). He has published over 170 manuscripts in SCI journals, including Science, Nature, Cell Metabolism, Nature Cancer, JACS, Hepatology, JCI, Redox Biology, with > 14,600 citations and H-index of 66 (Google Scholar, Sept. 2024). He has been listed as the top 2% of the Most-Cited Scientists in the world by Stanford University and was awarded prestigious "Senior International Scientists" in 2021 by Chinese National Natural Science Foundation (NSFC).



The dynamic thiol redox proteome of macrophages and its role in the response to oxidative-inflammatory stress

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Abstract

Oxidative modifications of protein cysteine thiols regulate various physiological processes, including innate immune and inflammatory responses. We conducted proteomic and mechanistic studies to investigate the roles of protein thiol oxidation in inflammatory macrophages. Through these studies we uncovered new roles for thioredoxin in regulating the macrophage inflammatory response. We further characterized new thiol redox switches that regulate glutathione homeostasis and autophagy in activated macrophages. Overall, these studies have yielded new insights into the dynamic redox proteome of macrophages and the mechanisms by which macrophages adapt and fine-tune their responses according to a changing inflammatory and redox environment.

We next aimed to characterize how reactive sulfur species (RSS) and thiol persulidation influences macrophage oxidative-inflammatory response. We revealed that classical activation of mouse or human macrophages using lipopolysaccharide and interferon-γ (LPS/IFN-γ) triggers substantial production of RSS, leading to widespread protein persulfidation. Additional analyses revealed that this upsurge in cellular S-persulfidation engaged ~2% of total thiols and modified over 800 functionally diverse proteins, while in comparison the global proteome exhibited little changes. In this setting, S-persulfidation was largely dependent on the cystine importer xCT and the hydrogen sulfide-generating enzyme cystathionine γ-lyase. We further investigated the role of the sulfide-oxidizing enzyme sulfide quinone oxidoreductase (SQOR), and found that it acts as a negative regulator of S-persulfidation. Elevated S-persulfidation following LPS/IFN-γ stimulation or SQOR inhibition was associated with increased resistance to oxidative stress. Upregulation of persulfides also inhibited the activation of the macrophage NLRP3 inflammasome and provided protection against inflammatory cell death. These findings provide a better understanding of the effects of RSS in macrophages and highlight the crucial role of persulfides in enabling macrophages to cope with oxidative-inflammatory stress.

Key words: Redox Biology, Reactive Oxygen Species

Short CV

Dr. Benhar received his undergraduate degree in chemistry and graduate degree in biochemistry at the Hebrew University of Jerusalem, Israel. After a postdoctoral training at Duke University (USA) he joined in 2009 the Department of Biochemistry at the Technion-Israel Institute of Technology, as an Assistant Professor. In 2016 he became an Associate Professor at the Technion.

Dr. Benhar has a longstanding interest in oxidant signaling and in redox mechanisms involved in inflammation and cancer. Dr. Benhar and his group employ proteomic and biochemical tools to explore the roles of oxidative protein modifications in cellular signaling in macrophages and cancer cells. Research by Dr. Benhar has provided new insights into the crosstalk between nitric oxide and the thioredoxin antioxidant system in tumor and immune cells. His recent work revealed new roles and mechanisms by which of reactive sulfur species regulate inflammatory and cell death responses.



Immune memory against toxic aldehydes

Koji Uchida

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Abstract

Natural antibodies, predominantly IgM, play an important role in the defense against pathogens and in maintaining homeostasis against oxidized molecules known as oxidation-specific epitopes. Due to the complexity of oxidized products, very few individual epitopes have been characterized in detail. Based on the fact that the B cell repertoire contains cells producing IgM against oxidation-specific epitopes, we investigated the presence of innate B cells that respond to modified proteins with aldehydes. Among the aldehyde associated with lipid peroxidation, acrolein, the most reactive of all aldehydes, was shown to be as a potential source of the innate epitopes. We also established the presence of innate B-1 cells that specifically respond to the acrolein-modified proteins via a B cell receptor-dependent mechanism. The V-D-J gene usage of the VH and VL for the anti-acrolein IgM-producing hybridoma was 100% identical to the germline gene sequences, suggesting clonal expansion of IgM-producing B cell population. Our discovery of acrolein as a source of innate epitopes suggests that, besides our common concept of aldehydes as toxic molecules, they may also play a role as a crucial signal for cell survival (also called a tonic signal) mediating the homeostatic responses via binding to proteins.

Key words: Innate immunity, innate antigens, aldehydes, covalent modification of proteins

Short CV

Professor Uchida received his Ph.D. from Nagoya University in 1988 and immediately became Assistant Professor at the same institution. After completing his postdoctoral training at the N.I.H. in Bethesda from 1990 to 1992, he was promoted to Associate Professor in the Laboratory of Food and Biodynamics, Nagoya University in 1996 and to Professor in 2011. Currently, he is a professor at the Laboratory of Food Chemistry, Graduate School of Agricultural and Life Sciences, The University of Tokyo.



A protein protein interaction between SOD1 and YWHAZ and YWHAE

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Abstract

Background: Copper-zinc superoxide dismutase 1 (SOD1) is one of the major intracellular redox enzymes in scavenging superoxide radicals. Past research has been focused on that type of antioxidant protection of the enzyme.

Objective and methods: The current study was conducted to explore its non-canonical role and the metabolic implications. We applied protein complementation assay (PCA) and revealed novel protein-protein interactions (PPIs) between SOD1 and tyrosine 3-monooxygenase/tryptophan 5-monooxygenase activation protein zeta (YWHAZ) or epsilon (YWHAE). We also used site-directed mutagenesis of SOD1 to characterize the binding conditions of the two PPIs. We also determined impacts of the PPIs' on lipid metabolism and cell growth and survival of HEK293T and HepG2 cells

Results and Discussion: The formation of the SOD1 and YWHAE or YWHAZ protein complex elevated enzyme activity of purified SOD1 in vitro by 40% ($P < 0.05$) and protein stability of over-expressed intracellular YWHAE (18%, $P < 0.01$) and YWHAZ (14%, $P < 0.05$). Metabolically, these PPIs were associated with lipolysis, cell growth, and cell survival in HEK293T or HepG2 cells. In conclusion, our findings unveiled two new PPIs between SOD1 and YWHAE or YWHAZ and their structural dependence, responses to redox status, mutual impacts on the enzyme function and protein degradation, and metabolic implications. Overall, revealing the unorthodox role of SOD1 will provide new perspectives and insights for diagnosing and treating diseases related to this and other antioxidant proteins.

Reference: Z. Q. Sun and X. G. Lei. 2023. Evidence and metabolic implications for a new non-canonical role of Cu-Zn superoxide dismutase. Int. J. Mol. Sci. 24(4), 3230; https://doi.org /10.3390/ijms24043230.

Key words: Antioxidant enzyme, non-canonical role, tyrosine 3-monooxygenase/tryptophan 5-monooxygenase activation protein

Short CV

Xingen Lei is a Professor of Molecular Nutrition at Cornell University. He has developed a new generation of bacterial phytases that are used by the feed industry in 50 countries. Lei also pioneered nutritional genomics of selenium in animals and revealed dual roles of selenium in oxidative stress and diabetes. Lei is an international leader in applying agriculture to prevent "hidden hunger". He currently serves as the Editor-in-Chief of The Journal of Nutrition, President of Trace Elements in Man and Animals, and Associate Dean of Research and Innovation in College of Agricultural and Life Sciences at Cornell University. Lei was elected as a Fellow of the National Academy of Inventors in 2021.

Symposium-YIO-1 (Y-1)

Redox modification of biomacromolecules
Redox and obesity, vascular function and metabolism

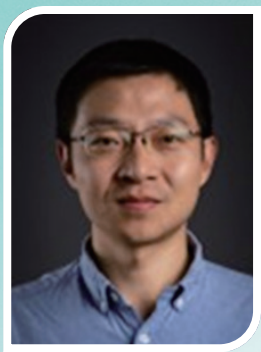




Chair: Li Xu (徐力)
Jilin University, China
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Short CV

Prof. Xu received her Ph.D. from Jilin University in 2002. She is currently a professor at the Key Laboratory of Molecular Enzyme Engineering, Ministry of Education, College of Life Sciences, Jilin University. From 2004 to 2005, she studied abroad as a national public scholarship visiting scholar at the INRA Institute in France. From November 2010 to March 2011, she was invited to serve as a visiting professor at Kwansei Gakuin University in Japan, where he conducted lectures and academic exchanges. From November 2012 to March 2013, she engaged in collaborative exchanges as a national public senior research scholar at George Washington University in the United States. Her research focuses on Nanomedicine: 1. Enhanced Targeted Nanocarrier for Hydrophobic Drug Delivery; 2. Designing a special functionalized nanozyme that depends on the interface effect of an inorganic nanocrystal. 3. Modification and assembly of nanoparticles with peptides and their applications in biotechnology. 4. Development of functional bioactive peptides in disease therapy.



Activation mechanism of phagocyte NADPH oxidase

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Abstract

Phagocyte NADPH oxidase, known as the NOX2-p22 complex, is responsible for transferring electrons from intracellular NADPH to extracellular oxygen. This process generates superoxide anions that are vital for killing pathogens. The activation of phagocyte NADPH oxidase requires membrane translocation and the binding of several cytosolic factors, including p47, p67, and Rac1. Our cryo-EM studies reveal that the p67-Rac1 complex clamps on the dehydrogenase domain (DH) of NOX2 and induces its contraction, stabilizing the binding of NADPH and resulting in a reduction of the distance between the NADPH-binding domain (NBD) and the FAD-binding domain (FBD). Additionally, DH docks onto the bottom of the transmembrane domain (TMD) of NOX2, leading to a shortened distance between the FAD and the inner haem. These structural rearrangements might facilitate the efficient electron transfer between the redox centers within NOX2, leading to the activation of phagocyte NADPH oxidase.

Key words: NOX, NOX2, NADPH, superoxide, ROS

Short CV

Lei Chen received his Ph.D. from Tsinghua University. He was working on the mechanism of AMPK. After that, he moved to Oregon Health and Science University as a postdoctoral researcher in the lab of Eric Gouaux, studying the mechanism of AMPA receptors. He started his own lab in Peking University in 2016. His lab focuses on the molecular mechanism of proteins involved in human diseases, especially metabolic diseases and cardiovascular diseases.



OGG1 promotes iTreg differentiation and alleviates mouse IBD by facilitating Foxp3 transcriptional activation

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Abstract

8-Hydroxyguanine (8-oxoGua) is one of the most prevalent forms of oxidative damage of DNA bases due to guanine's low redox potential. The 8-hydroxyguanine DNA glycosylase 1 (OGG1) is a cognate DNA repair enzyme that specifically recognizes 8-oxoGua and initiates base excision repair pathway to ensure genomic fidelity. However, guanine oxidation has an evolutionary bias within the genome and tends to occur in transcriptional regulatory regions. Thus, 8-oxoGua is beyond a lesion requiring repair, but may be an epigenetic-like modification in response to oxidative stress. Correspondingly, OGG 1 can serve as a specific "reader" of this base modification, playing function in transcriptional regulation of inflammatory genes independent of its repair activity.

Our present study further revealed that OGG1 deficiency reduces iTreg differentiation and aggravates mouse IBD colitis. Mechanically, the enzymatically inactive OGG1 binds to the promoter and CNS1 of Foxp3, promoting the recruitment of Smad3 to enhance Foxp3 transcription. Additionally, at the epigenetic level, binding of OGG1 to 8-oxoGua results in the demethylation of Foxp3 promoter and CNS2 via recruiting Tet1/2 and expelling Dnmt1, which in turn activates Foxp3 transcription. Furthermore, the OGG1S326C mutant, which has been taken as a susceptibility factor for many diseases such as lung diseases, with frequency up to ~20% in the human population, exhibits a stronger effect on iTreg differentiation induction than its wild-type counterpart, thus is negatively correlated with the incidence of IBD. Finally, OGG1 inhibitor O8, which inhibits imine formation in OGG1 without blocking its substrate binding, was able to promote mouse and human iTreg differentiation, and then effectively alleviate IBD in mice.

This work not only expands our understanding of the mechanism by which OGG1 promotes gene transcriptional activation and the dual roles of OGG1 in inflammation modulation, but also provides new targets for intervention in autoimmune diseases such as IBD.

Key words: oxidative stress, inflammation, OGG1, transcription regulation

Short CV

Xueqing Ba, Ph.D

Professor, the School of Life Science, Northeast Normal University. Research interest has long been focusing on the oxidative stress response of cells, especially the effect of DNA oxidation in chromatin-based biological processes. Among bio-marcromolecules, DNA is vulnerable to ROS due to the low redox potential of the nucleobases, and out of them, guanine is the most susceptible to being oxidized. The resultant base lesion 8-oxo-7, 8-dihydroguanine (8-oxoGua) is commonly regarded as a biomarker of oxidative stress and is repaired through 8-oxoguanine DNA glycosylase 1 (OGG1)-initiated base excision repair (BER) pathway. Our recent studies, together with the research by laboratories including Dr. Boldogh', Dr. Lloyd', Dr. Mitra', Dr. Burrow' and others', revealed the epigenetic role of 8-oxoGua and the "reader" function of OGG1 leading to gene expression during inflammation and tumorigenesis. This is supported by our original research (Li et al. BBA-Dis, 2024; Zheng et al., Redox Biol, 2023; Pan et al., Nucleic Acids Res, 2023; Hao et al, FASEB J, 2020; Hao et al. Redox Biol, 2018; Ba and Boldogh, Redox Biol (review), 2018; Wang et al, Cell Mol Life Sci, 2018; Wang et al, Cell Death & Dis, 2018; Pan et al. J Biol Chem, 2016).



A lactate-lipid peroxidation-acetate metabolic axis between tumor-associated macrophages and cancer cells fuels hepatocellular carcinoma metastasis

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Abstract

High abundance of acetyl-CoA is a critical metabolic feature in metastatic cancers. To sustain high level of acetyl-CoA, cancer cells actively uptake acetate for acetyl-CoA biosynthesis in various cancer types. However, the source of acetate in the cancer microenvironment remains largely undetermined. Here, using hepatocellular carcinoma (HCC) models, we demonstrate that tumor-associated macrophages (TAMs) promote acetate accumulation in HCC cells by secreting acetate to cancer microenvironment. Mechanistically, lipid peroxidation-ALDH2 pathway is responsible for the acetate production in TAMs. Inhibition of ALDH2 in TAMs suppresses the pro-migration effect of TAMs on HCC cells in vitro. In orthotopic HCC mice model, genetic ablation of ALDH2 in TAMs reduces acetate levels in primary HCC cells, and significantly diminishes HCC lung metastasis *in vivo*. Finally, we identify HCC cells-derived lactate as the upstream inducer of lipid peroxidation-ALDH2 pathway in TAMs. Collectively, our findings reveal a lactate-lipid peroxidation-acetate metabolic cross-talk between HCC cells and TAMs, which positions TAMs as an acetate reservoir to fuel HCC metastasis.

Key words: Tumor-associated macrophages, Lipid peroxidation, Acetate, HCC metastasis, Lactate

Short CV

Ming Lu, Principal Investigator in Shanghai Institute of Nutrition and Health (SINH), Chinese Academy of Sciences (CAS).

Brief Biography:

Ph.D./Postdoc Institute of Biochemistry and Cell Biology, CAS; Assistant/associated research fellow, Huashan Hospital, Fudan University; Visiting scientist, The Jackson Laboratory, US; Principal Investigator, SINH, CAS.

Research interests:

Our lab focuses on the roles of lipid metabolic aberrations in cancer metastasis and microenvironmental redox status.

Selected Publications:

Corresponding Author: Cell Metabolism 2019, 29(4): 886-900; Cancer Letters 2024, 592: 216903; Cell Rep, 2022, 39(3):110712.

First/co-first Author: Cancer Cell 2016, 30(3): 444-458. Nature Immunology 2020, 21(11): 1444-1455; Nature Communications 2020, 11(1): 4387.



STING: a potential target for suppressing the development of clonal hematopoiesis and leukemia

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Abstract

Clonal hematopoiesis (CH) is a significant risk factor for numerous diseases, including hematopoietic malignancies, atherosclerosis, ischemic stroke, gout, and chronic liver injury. Mutations in DNMT3A and TET2, which occur in approximately 70% of CH patients, are considered as initial triggers for leukemia. Despite this, effective strategies to prevent the progression of CH to leukemia are lacking. Our study demonstrates that targeting STING effectively prevents the development of CH harboring these mutations. Mechanistically, the loss of TET2 or DNMT3A activates the STING pathway, leading to chronic inflammation in DNA-modifying enzyme-deficient hematopoietic progenitor/stem cells. This inflammatory response in the bone marrow promotes increased self-renewal and skewed lineage differentiation of mutated HSPCs. Moreover, targeting STING activates FADS2 in AML1-ETO fusion leukemia cells, causing lipid-peroxidation-associated cell death. Collectively, our findings highlight STING as a potent target for preventing hematological diseases.

Key words: Clonal hematopoiesis, DNA modification, STING, Lipid peroxidation

Short CV

Dr. Yuheng Shi is an associate professor at the Institutes of Biomedical Sciences, Fudan University. His research primarily focuses on the role of inflammation in tumor initiation, the epigenetic regulation of leukemia development, and the identification of therapeutic targets. Dr. Shi has made series of progresses in understanding the mechanisms behind hematopoietic diseases related to mutations in DNA-modifying enzymes. His work has been published in journals such as Leukemia, Nature Communications, and Cell Reports.



Disorder of nitration/S-sulfhydration participates in hyperhomocysteinemia progression and liver damage

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Abstract

In recent years, studies have found that hyperhomocysteinemia (HHcy) has become an important risk factor for liver diseases. Meanwhile, homocysteine was mainly metabolized in liver. It is of great significance to clear out the relationship between HHcy and liver damage and reveal the underlying mechanism. Our study has found that HHcy increased the level of nitration of CBS and CSE, resulting in the inhibition of their activity. HHcy also decreased the expression level of CSE. *In vivo*, Hcy is negatively correlated with hydrogen sulfide (H2S) levels. HHcy decreased the S-sulfhydration level and activity of the Sp1-CSE-H2S pathway. The rise of nitration in HHcy led to insufficient S-sulfhydration. The decrease in H2S level inhibited MTHFR S-sulfhydration and its activity. This study proposed a vicious cycle of H2S signaling in Hcy metabolism, and emphasized the potential role of H2S in the treatment of HHcy. Furthermore, we found that HHcy promoted nitration of nuclear receptor coactivator 4 (NCOA4), up-regulate the level of ferritinophagy, cause a significant increase in intracellular free iron content, and increase the susceptibility to ferroptosis, thereby promoting the occurrence and development of liver injury. Our study also concluded that NaHS supplementation mitigates HHcy-induced liver injury by downregulating hepatic autophagy through the S-sulfhydration and activation of glucocorticoid-regulated kinase 1 (SGK1). In conclusion, the disorder of nitration/S-sulfhydration participates in HHcy progression and liver damage. The potential therapeutic application of H2S and anti-nitration in treating liver damage associated with HHcy presents a new avenue for research and clinical application.

Short CV

Professor, Doctoral Supervisor, Deputy Director of the Department of Physiology and Pathophysiology, School of Basic Medical Sciences, Capital Medical University, and Deputy Director of the Beijing Key Laboratory for Metabolic Disorder-Related Cardiovascular Diseases. Her research interest mainly focuses on abnormal oxidation-reduction modification and homocysteine metabolic disorder, whose work have been published in Cardiovasc Res, Antioxid Redox Signal and Free Radic Biol Med, et al as corresponding authors.



Endothelium-dependent contraction, NO and cardiovascular disorders in the absence of prostacyclin synthesis

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Abstract

Prostaglandin I2 (PGI2) synthesized by endothelial cyclooxygenase (COX) evokes potent vasodilation in some blood vessels but is paradoxically responsible for endothelium-dependent constriction (EDC) in others. However, how PGI2 synthase (PGIS) deficiency affects EDC and how this is implicated in the consequent cardiovascular pathologies remain largely unknown. Experiments were performed on WT, Pgis knockout (Pgis-/-) and Pgis/thromboxane-prostanoid receptor gene (Tp) double knockout (Pgis-/-Tp-/-) and Pgis-/- mice transplanted with unfractionated WT or Cox-1-/- bone marrow cells, as well as human umbilical arteries. PGF2α, PGE2 and a trace amount of PGD2, but not thromboxane A2 (TxA2), were produced in response to acetylcholine (ACh) in Pgis-/- or PGIS-inhibited arteries. PGIS deficiency resulted in augmentation of EDC ex vivo and *in vivo*. Endothelium-dependent hyperpolarization was unchanged, but phosphorylation levels of endothelial nitric oxide synthase (eNOS) at Ser1177 and Thr495 were altered and NO production and the NO-dependent relaxation evoked by ACh were remarkably reduced in Pgis-/- aortas. Blood pressure and the cardiac parameters remained normal in Pgis-/- mice at 8-10 weeks, but later the mice sequentially developed high blood pressure, vascular remodeling and cardiac hypertrophy. Additional ablation of TP not only restrained EDC and the downregulation of NO signaling in Pgis-/- mice, but also ameliorated the cardiovascular abnormalities. Stimulation of Pgis-/- vessels in the presence of platelets led to increased TxA2 generation. COX-1 disruption in bone marrow-derived cells failed to affect the development of high blood pressure and vascular remodeling in Pgis-/- mice though it largely suppressed the increase of plasma TxB2 (TxA2 metabolite) level. The non-TxA2 prostanoids/TP axis plays an essential role in mediating the augmentation of EDC, the decrease of NO and the cardiovascular disorders when PGIS is deficient, suggesting TP as a promising therapeutic target in diseases associated with PGIS insufficiency.

Key words: prostacyclin, hypertension, endothelial dysfunction, endothelium

Short CV

Dr. Bin Liu is a professor at Shantou University Medical College. He received his PhD degree from Institute of Chemistry, Chinese Academy of Sciences and completed his postdoctoral training at the Ohio State University. His research interests involve COX products, NO and ROS in heath and diseases and his recent work has been published in journals such as Circ Res and Kidney Int.



SIRT2 governs a cytoplasm-mitochondrial signal to repress mitochondrial ROS and vascular ageing

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Abstract

Cardiovascular diseases are the leading causes of death and disability in the world. A better understanding for the molecular mechanism in the development of cardiovascular diseases will provide more strategies for their diagnosis, treatment and prevention. Mammalian SIRTuins regulate metabolism and aging-related diseases, including diabetes and cardiovascular diseases. Among the SIRTuins, the cytosol member histone deacetylase SIRT2 is less characterized. Our group had previously shown that SIRT2 in the heart represses aging-related cardiac hypertrophy, at least in part, by maintaining signaling through the liver kinase B1 (LKB1)-AMPK pathway, the central pathway controlling various aspects of metabolism. Sirt2-KO promoted aging-related cardiac hypertrophy and caused cardiac dysfunction. However, Sirt2-KO attenuated metformin-induced activation of AMPK signaling and, subsequently, the cardioprotective functions of metformin in hypertrophic hearts. The results of recent studies from our lab showed that SIRT2 expression was highest in mouse aortas among the SIRTuin family and decreased in vascular smooth muscle cells (VSMCs) of aged aortas. Sirt2-KO promoted vascular remodeling in aged aortas. SIRT2 governs a cytoplasm-mitochondrial signal to repress mitochondrial ROS and vascular ageing. SIRT2 plays a protective role in age-related vascular dysfunction. Therefore, SIRT2 activation may represent a promising strategy for managing age-related cardiovascular dysfunction.

Short CV

Dr. Hou-Zao Chen is currently a professor of State Key Laboratory of Common Mechanism Research for Major Diseases, Department of Biochemistry and Molecular Biology, Institute of Basic Medical Sciences, Chinese Academy of Medical Sciences (CAMS) & Peking Union Medical College (PUMC). Dr. Chen's research expertise is molecular mechanisms of age-related cardiovascular diseases, particularly the role of epigenetic regulation in the development and progression of atherosclerosis, diabetic vascular disease, aortic aneurysm and cardiac hypertrophy. He has published more than 60 original research articles and invited reviews including Sci Immunol.2024; Nat Commun.2024,2022; Cell Rep.2024; Euro Heart J.2023,2017; Proc Natl Acad Sci U S A.2022; Circ.2021,2016; Nat Cell Biol.2019; J Exp Med.2016; Circ Res.2016,2011; Cell Syst.2015; Aging Cell 2014, which have been cited more than 5000 times. Throughout his academic career, Dr. Chen has received numerous awards including National Funds for Distinguished Young Scientists in China. He has served as Editorial Board Member for Free Radical Biology and Medicine and Cardiovascular Drugs and Therapy.



Tetrahydrobiopterin is a promising target of diabetic cardiomyopathy via restoring mitochondria function

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Abstract

Diabetic cardiomyopathy (DCM) is a major cause of mortality/morbidity in diabetes mellitus patients. Although tetrahydrobiopterin (BH4) shows therapeutic potential as an endogenous cardiovascular target, its effect on myocardial cells and mitochondria in DCM and the underlying mechanisms remain unknown. Here, we determined the involvement of BH4 deficiency in DCM and the therapeutic potential of BH4 supplementation in a rodent DCM model. We observed a decreased BH4:total biopterin ratio in heart and mitochondria accompanied by cardiac remodeling, lower cardiac contractility, and mitochondrial dysfunction. BH4 supplementation improved cardiac function, corrected morphological abnormalities in cardiac muscle, and increased mitochondrial activity. In the diabetic heart, a decrease in PGC1a, which is important for regulating mitochondrial biosynthesis, was confirmed, and BH4 significantly increased its level. Mechanistically, BH4 bound to calcium/calmodulin-dependent protein kinase kinase 2 (CaMKK2) and activated downstream AMP-activated protein kinase/cAMP response element binding protein/PGC-1α signaling to rescue mitochondrial and cardiac dysfunction in DCM. These results suggest BH4 as a novel endogenous activator of CaMKK2.

Key words: Tetrahydrobiopterin, mitochondria, diabetic cardiomyopathy

Symposium-YIO-2 (Y-2)

Redox and cancer, infection and immunity
Redox and environmental challenge





Chair: Jianghong Man (满江红)

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Short CV

The current focus of Dr. Man's research program is to identify specific protein targets and key regulators that control the maintenance of cancer stem cells (CSCs) in glioblastoma progression. The major purpose of his research is clarifying how CSC maintain its stemness and associate with the microenvironments to promote tumor growth, contribute therapeutic resistance and recurrence.

Education

2015 − now, Professor, National Center of Biomedical Analysis (NCBA), Beijing, China

2012 − 2015, Postdoctoral Fellow, Cleveland Clinic, Cleveland, OH, USA

2002-2008, Ph.D., National Center of Biomedical Analysis (NCBA), Beijing, China

1994-1999, M.D., China Medical University, Shenyang, China

Peer-reviewed Publications

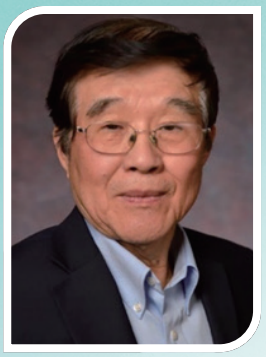
1. Dake Xiao, Haowen Ran, Lishu Chen, ..., Jianghong Man*. FSD1 inhibits glioblastoma diffuse infiltration through restriction of HDAC6-mediated microtubule deacetylation. SCIENCE CHINA Life Sciences. 2024, (accepted)

2. Lishu Chen, Qinghui Qi, Xiaoqing Jiang, Jin Wu, ..., Jianghong Man*. Phosphocreatine promotes epigenetic reprogramming to facilitate glioblastoma growth through stabilizing BRD2. Cancer Discovery. 2024, Aug 2;14(8):1547-1565.

3. Chen L, Zhou C, Chen Q, ..., Man J*. Oncolytic Zika virus promotes intratumoral T cell infiltration and improves immunotherapy efficacy in glioblastoma. Mol Ther, 2022 Feb 1;24:522-534.

4. Haohao Huang, Songyang Zhang, Yuanyuan Li, ..., Jianghong Man*. Suppression of mitochondrial ROS by prohibitin drives glioblastoma progression and therapeutic resistance. Nature Communications, 2021, Jun 17;12(1):3720.

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Chair: Chung S. Yang

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Short CV

Dr. Chung S. Yang is a Distinguished Professor Emeritus at Rutgers University, New Jersey, USA. He is noted for his research on disease prevention by dietary constituents such as tea, vitamin E, and other agents. His research group studied the fundamental mechanisms of cancer formation and prevention in animal models and extended the research to humans. Dr. Yang is one of the first group of scientists to conduct collaborative research in China in 1979 after US and China established normal diplomatic relationship and, soon afterwards, he helped to establish the large scale Linxian Nutritional Intervention Trial (LNIT). This unique US - China collaborative study found that supplementation with a combination of vitamin E, beta-carotene, and selenium for 63 months decreased mortality due to gastroesophageal cancer. His collaborative research and teaching in China have continued for 45 years today.

Dr. Yang has trained over 100 research students/associates and has authored more than 600 publications. He was elected Fellow of the American Association for the Advancement of Science in 2010 and received the First Lu Yu Award from the China Tea Science Society in 2021.



Two sesquiterpene lactones inhibit TXNRD1 and induce endoplasmic reticulum stress in cancer cells

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Abstract

Inhibiting selenoprotein TXNRD1 with lead compounds or effective drugs is beneficial for enhancing chemotherapy in clinicals. In this study, we identified two sesquiterpene lactone compounds, ergolide (Ergo) and eupalinolide K (EupK), are effective inhibitors of TXNRD1. TXNRD1 mutants' activity assay and LC-MS/MS analysis revealed that Ergo and EupK targeted the Sec498 residue of TXNRD1 through the Michael addition. The inhibition of TXNRD1 by Ergo and EupK, abolished the disulfide reductase activity but increase superoxide production via the inherent NADPH oxidase activity of the enzyme. In cellular condition, the cytotoxicity of Ergo and EupK is associated with oxidative stress and endoplasmic reticulum (ER) stress, and ultimately leading cancer cells to apoptosis in HCT116 cells. Meanwhile, we demonstrated that TXNRD1 inhibition may up-regulate NRF2, and Ergo's cytotoxicity was slightly increased when NRF2 activation was suppressed. Furthermore, we compared Ergo and EupK with another four sesquiterpene lactone compounds when incubating with TXNRD1, using LC-MS/MS analysis. Our results showed that the Cys64 residue is an effective binding site for the TXNRD inhibitor parthenolide (Part), and a notable preclinical TXNRD1 inhibitor TRi-1 can bind to Cys59 residue, indicating that N-terminal redox motif is also a target site for TXNRD1 inhibitors besides the redox active C-terminal motif. Taken all, this study may improve our understanding of TXNRD1 binding and inhibition, and provided new insights into the development of effective small molecules targeting TXNRD1.

Key words: thioredoxin reductase, sesquiterpene lactone, SecTRAPs, disulfide stress, ER stress, NRF2

Short CV

Xu is an associate professor in School of Chemical Engineering, Ocean Technology & Life Science @ Dalian University of Technology (DUT). He completed his B.S.(200207)/Ph.D.(200810) with Prof. Qing Yang in Dalian, and got his PostDoc training (200902-201409) with Prof. Elias Arner @ Karolinska Institutet in Stockholm. Since 201512, he was leading a research lab on selenobiology at Panjin Campus of DUT. His lab focuses on TXNRD1/2 & GPX1/4 regulating emerging PCDs & tumor cell drug resistance. He has published >50 peer reviewed research papers and the current H-index is 27.



Novel anticancer drug discovery strategies by targeting NQO1

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Abstract

NAD(P)H: quinone oxidoreductase 1 (NQO1) is an enzyme expressed in high levels in multiple solid tumors, making it an appealing prospect as an anticancer drug target, specifically in regard to NQO1 positive (NQO1+) tumors. β-lapachone (β-lap) is a drug targeting NQO1 that is presently undergoing clinical trials. β-lap selectively kills NQO1+ cancer cells by instigating reactive oxygen species (ROS) through catalytic activation of NQO1. Here, we found that cryptotanshinone (CTS), a natural compound, triggers NQO1-dependent necrosis without impacting NQO1 activity. The impact of CTS on NQO1 was measured using cellular thermal shift assay, enzymatic activity assay, and tryptophan fluorescence titration. The interaction between CTS and NQO1 was examined using molecular docking. The downstream signalings of NQO1, including iron, Ca2+, c-Jun N-terminal kinase 1/2 (JNK1/2), and poly(ADP-ribose) polymerase (PARP) in response to CTS were investigated using inhibitors, siRNA, and other methods. The results showed that CTS selectively kills NQO1+ cancer cells by inducing NQO1-dependent non-apoptotic necrosis. It is interesting to note that CTS directly binds to NQO1 but does not activate its catalytic activity. Moreover, CTS selectively suppressed the growth of the tumor in the NQO1+ xenograft model, which was reversed by the NQO1 inhibitor and NQO1 shRNA. In addition, the combination of CTS with β-lap shows a significant synergistic anticancer effect. This study demonstrates the non-enzymatic function of NQO1 in triggering cell death, offering new avenues for the creation of NQO1-targeted drugs against cancer.

Key words: NQO1, ROS, Cancer, Drug discovery

Short CV

Dr. Xiuping Chen obtained his Ph. D degree from Peking Union Medical College in 2007. Since Aug 2010, he worked as an assistant professor at University of Macau and was promoted to associate and full professor in 2015 and 2021, respectively. Dr. Chen's research focuses on pharmacology. He aims to screen and identify regulators of programmed cell death (PCD) and epithelial-mesenchymal transition (EMT) from natural compounds as leads or potential drugs for the treatment of cancer, cardiovascular diseases, and fibrotic diseases. He is the winner of The 17th SERVIER Young Pharmacologist Award in 2013 and the winner of Second prize of the Macao Science and Technology Awards in 2012 and 2014. Dr. Chen is a Member of the Royal Society of Biology since 2023. Dr. Chen has > 150 publications with >14,000 citations. The h-index is 63.



Structure-activity relationship and biomedical applications of nanozymes

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Abstract

Nanozymes, a novel class of nanomaterials exhibiting enzymatic properties, have gained significant attention in the biomedical field due to their multifunctionality, tunable catalytic activity, and exceptional stability. Despite these advantages, nanozymes still fall short in catalytic efficiency and diversity when compared to natural enzymes. This talk delves into the structure and function of nanozymes, with a focus on the active sites and catalytic microenvironments of natural enzymes as a blueprint. Through de novo design and biomimetic synthesis, we achieved rational design and activity optimization of iron-based and single-atom nanozymes, proposing a comprehensive structure-activity relationship for these materials. Leveraging this relationship, we pioneered new applications of nanozymes in tumor catalytic therapy, prodrug activation, and the construction of nanozyme organelles. These advancements pave the way for innovative approaches in the diagnosis and treatment of major diseases, offering fresh perspectives and technologies for the medical field.

Key words: Nanozymes, Structure-Activity Relationship, Biomedical Applications

Short CV

Kelong Fan received his Ph.D. degree in cell biology from the Institute of Biophysics, Chinese Academy of Sciences, in 2014. After this period, he stayed there to further pursue 3 years of postdoc training and 2 years of associate professor work experience before attaining a full professor position in 2019. He is interested in exploring the novel functions and applications of nanozymes in biomedicine, with a top priority to design functional nanozymes by learning from nature, and to develop novel strategies for disease theranostics. He is recognized as "Highly Cited Researcher" by Clarivate in 2022-2023 and now serves as Deputy Editor of Exploration.



Electron FLASH irradiation ameliorates radiation-induced developmental and neurological toxicity in zebrafish model

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Abstract

Ultra-high dose rate irradiation, also named Flash radiotherapy, emerged as a new milestone in the field of cancer radiotherapy, which has been shown to spare normal tissues while preserving the therapeutic effect on tumors compared to conventional irradiation (Conv). However, successful clinical translation of Flash radiotherapy depends on a better understanding of the underlying biological mechanisms. In this study, we leveraged the zebrafish model in radiobiological research to explore the amelioration of radiation-induced neurodevelopmental toxicity. The findings of this study demonstrated that electron Flash radiotherapy can reduce radiation-induced developmental and neurological toxicity and may provide long-term benefits, suggesting the potential utilization of this modality for the clinical management of patients requiring cranial radiation therapy.

Key words: Flash radiotherapy, Zebrafish model, Neurobehavior, Developmental neurotoxicity, Neuroinflammation

Short CV

Di Cuixia, Ph. D., researcher, professor, specializes in the mechanism of heavy ion therapy. Her work is to reveal the molecular mechanism of Alternative splicing in overcoming the radiosensitivity of cancer cells to heavy ions. Published more than 60 articles in well-known international journals such as Cell Death and Differentiation, J Exp Clin Cancer Res. Participated in Program 973 projects and key R & D projects of the Ministry of Science and Technology. Now responsible for the key R & D projects of the Ministry of Science and Technology. She has won provincial and ministerial awards for science and technology many times.



Supersulfide regulation of innate immune responses

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Abstract

Cysteine is an amino acid having thiol as a functional group, and plays an essential role in maintaining the structure of proteins through disulfide bonds. It also acts as an antioxidant, and enzyme activity center within cells. In recent years, cysteine persulfide and polysulfides, in which sulfur atoms are conjugated (sulfur catenation) to the cysteine thiol group, are abundantly present in cells as forms of glutathione and protein persulfides and polysulfides, and they have a wide variety of biological effects such as extremely strong antioxidant activity, anti-inflammatory effects, regulation of immune function, and control of energy metabolism. These diverse biological functions are not found in the original cysteine thiols, or are much stronger, so it is called supersulfides, meaning that it has functions that exceed those of sulfides. Currently, the metabolism and functional regulation of supersulfides are attracting a great deal of attention worldwide as targets for new drug discovery and diagnosis. We are currently investigating the roles of supersulifdes on innate immune responses. In this study, regulatory roles of supersulfides on neutrophil functions, particularly focusing on the effects of supersulfides on neutrophil mediated bacterial killing and host defense.

Key words: Innate immunity, neutrophil, supersulfides, bacterial infection, glutathione



Supersulfides regulate NLRP3 inflammasome activation through sensing homeostasis

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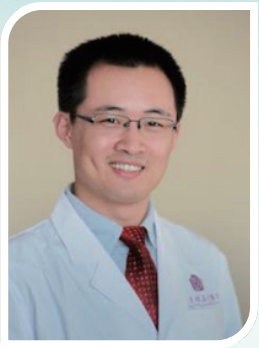
Abstract

The activation of the nucleotide-binding domain, leucine-rich repeat domain and pyrin domain-containing protein 3 (NLRP3) inflammasome is precisely regulated to prevent excessive innate immune and inflammatory responses. Despite numerous proposed mechanisms for regulating NLRP3 inflammasome activation, the checkpoints governing this process remain elusive. Here, we demonstrate that supersulfidation, a reversible post-translational modification of protein cysteine residues to persulfides or polysulfides, occurs endogenously to modulate NLRP3. This modification plays a crucial role in the NLRP3 inflammasome activation throughout whole processes, including priming, assembly and activation. Furthermore, supersulfidation of NLRP3 is implicated in homeostasis-altering molecular processes, such as potassium efflux and redox imbalance within macrophages. More importantly, enhancing supersulfides using a chemical donor significantly halts NLRP3 inflammasome activation both in vitro and *in vivo* by regulating NLRP3 supersulfidation. Our findings provide new insights into the endogenous mechanisms of NLRP3 inflammasome activation and constitute a potential therapeutic strategy for NLRP3 inflammasome-associated inflammatory diseases.

Key words: Reactive Sulfur Species, Supersulfides, NLRP3 inflammasome, Immune Responses, Inflammatory Responses

Short CV

I am an assistant professor at Akita University, Japan.Myresearch focuses on the regulatory mechanisms ofimmune and inflammatory responses from theperspective of supersulfides. I have developed supersulfide donors and demonstrated that supersulfides regulate immune responses by counteracting Toll-like receptor signaling. I also discovered that intracellular glutathione and its persulfides efflux are necessary for NLRP3 inflammasome activation.



OSA induced multiple-system damage via oxidative stress

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Abstract

Obstructive sleep apnea (OSA), affecting approximately 1 billion adults globally, is characterized by recurrent airway obstruction during sleep, leading to oxygen desaturation, elevated carbon dioxide levels, and disrupted sleep architecture. OSA-related complications include cardiovascular disorders, neurological impairments, metabolic dysfunction, and a potential link to cancer. OSA significantly impacts quality of life and is associated with increased morbidity and mortality, particularly in the cardiovascular and cognitive domains. The cyclic pattern of intermittent hypoxia in OSA triggers oxidative stress, contributing to the multiple-system damage. Understanding the role of oxidative stress in OSA will help to clarify the etiology and discover new treatment options, which will be of great signifcance for better clinical intervention.

Key words: Obstructive sleep apnea, Oxidative stress, Intermittent hypoxia, Multiple-system damage

Short CV

Guoping Yin, tenured associated professor of Tsinghua university.Member of the Sleep Medicine Professional Committee of the Chinese Medical Association, standing committee member and deputy secretary-general of Sleep Medicine Committee of the China International Healthcare Promotion Association, member of Otolaryngology Head and Neck Surgery Committee of Chinese Medical Association. Focus on the basic and clinical research of sleep disorder breathing. Up to now, has sponsored one general project of National Natural Science Foundation of China, as the key member participated 6 general project of National Natural Science Foundation of China and 1 National Key R&D Project of Science and Technology Ministry of China. As first or corresponding author published 13 high- level papers in this area, as co-author published 43 papers.



HTHB: A Potential Therapeutic Agent for Cognitive Impairment and Inflammation

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Abstract

Improving the function of central nervous system and peripheral metabolic organs simultaneously is an effective strategy to delay aging. 2-(3,4-Dihydroxyphenyl)ethyl 3-hydroxybutanoate (HTHB), a novel derivative of hydroxytyrosol (HT) synthesized by our laboratory, has shown promising metabolic and anti-inflammatory properties. We explored the mechanism of HTHB in improving brain function. In sleep deprivation (SD) rats, HTHB treatment significantly ameliorated behavioral disorders, reduced brain oxidative stress, and alleviated mitochondrial DNA (mtDNA) oxidation and release. This reduction in mtDNA oxidation and efflux was associated with decreased activation of pro-inflammatory cytokines and NF-κB. Research on senescence-accelerated mouse prone 8 (SAMP8) mice demonstrated that HTHB effectively mitigates memory decline, reduces inflammation in the brain cortex, intestine, and peripheral system, and modulates gut microbiota dysbiosis. Behavioral testing, biochemical detection, and 16S RNA analysis revealed that HTHB treatment enhances gut barrier integrity and rescues tight junction protein levels impaired by lipopolysaccharide (LPS) in vitro.

Together, these studies underscore the therapeutic potential of HTHB in addressing cognitive impairment and inflammation. The research on HTHB provides new experimental evidence for addressing disease- or aging-induced cognitive decline.

Key words: HTHB; Cognitive function; mitochondrial DNA; Gut microbiota; Inflammation

Short CV

- 2006-2009: Postdoctoral Researcher, University of California, Irvine and Sunhealth (Banner Health) Research Institute, USA
- 2009-2011: Associate Professor, School of Life Science and Technology, Xi'an Jiaotong University
- 2011-Present: Professor (Young Key Teacher) and Ph.D. Supervisor, School of Life Science and Technology, Xi'an Jiaotong University
- 2015-2016: Deputy Dean, School of Life Science and Technology, Xi'an Jiaotong University
- 2016-Present: Deputy Dean, Graduate School, Xi'an Jiaotong University

Symposium-YIO-3 (Y-3)

Redox and neural function and mental health





Chair: Wen li Li (李文丽)

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Short CV

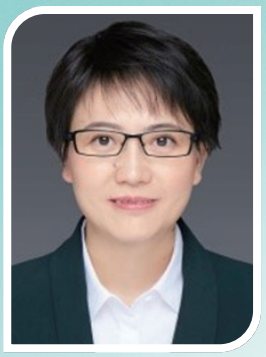
Wenli Li, Professor of Department of Toxicology, Expert for the International Organization for the Prohibition of Chemical Weapons. Prof. Li received her Master Degree in Preventive Medicine in 1996 and PhD in 2004 from The Fourth Military Medical University. She studied in the University of Tokyo in Japan for one year as visiting professor. Prof. Li cooperated research on phosgene with Inhalation Toxicology Laboratory of Bayer in Germany for ten years. She has been focusing on the study of the molecular mechanism and prevention of injury caused by industrial poisons, especially on lung injury induced by phosgene. She also focused on the studies of antioxidants in free radical biology and medicine. Prof. Li has received 4 projects of the National Natural Science Foundation, 2 international cooperation projects, 1 Key research and development project of Shaanxi Province and so on. She published more than 50 scientific papers in peer-reviewed journals and participated two books in the world, named Inhalation Toxicology and Chemical Warfare Agent as editor. She is on the editorial board of Inhalation Toxicology, the vice chairman of the Shaanxi Toxicology Society, the member of the International Phosgene Safety Group, a Committee Member of the Free Radical Biology and Medicine Branch of the Chinese Biophysical Society.



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Short CV

Prof. Changyang Gong received his Ph.D. degree in cell biology from Sichuan University, Chengdu, China, in 2010. He is currently a Professor of pharmaceutics at State Key Laboratory of Biotherapy, West China Hospital, Sichuan University, China. His current research focuses on novel drug and gene delivery system for tumor therapy, nanomedicine, and immune adjuvant for tumor vaccine. He has published three book chapters and more than 120 peer-review scientific papers in Nat Commun, J Am Chem Soc, Adv Mater, Adv Funct Mater, Adv Sci, ACS Nano, Adv Drug Deliver Rev, Biomaterials, J Control Release etc. He is Editorial Board of Chinses Chemical Letters and BioMed Research International. His scientific achievements have been honored by the National Natural Science Foundation for Excellent Young Scholars (2018) and the National Program for Support of Top-notch Young Professionals of China (2015).



Redox biomarkers for prognosis of infectious diseases

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Abstract

Oxidative stress is essentially caused by an imbalance between oxidizing reagents and reducing reagents in body. In viral infections, oxidative stress has been reported arising. However, whether oxidative stress still exists during recovery or under treatment is barely investigated. In one cohort study for patients recovered from COVID-19 or with long-COVID, blood and urine nitrate levels were found significantly higher than those who never got infected. In another cohort study for HIV-infected patients or patients with AIDS-related lymphoma (ARL), nitrate, malondialdehyde, total antioxidant capacity differentiated ARL and HIV-only patients. In addition, levels of nitrite, 3-nitrotyrosine, malondialdehyde and total antioxidant capacity were different between ARL patients who were still alive and those who passed away within one year after blood collection. In short, redox markers may provide useful insights for prognosis of virus-triggered diseases.

Key words: redox biomarker, prognosis, infectious disease, long-COVID, HIV

Short CV

Dr. Jun Wang graduated from The Johns Hopkins University with a Ph.D. degree in Chemistry. Jun joined Hubei University of Technology (Wuhan, China) in 2015 as a faculty in the Department of Biomedicine and Biopharmacology, and has been serving as a full professor and the director of the International Joint Research Center for Redox Biology Theory & Application of Hubei Province. Dr. Wang has published more than 50 peer-reviewed research papers in fundamental and translational studies of redox biology & medicine (h-index = 16), and served as a reviewer or editorial board member for several academic journals, including Inflammation, Journal of Alzheimer's Disease, Free Radical Biology & Medicine, Nitric Oxide, Journal of Biological Chemistry, EBioMedicine, Expert Opinion on Pharmacotherapy.



DDAH1, a key neuroprotective player, promotes neurogenesis and neural repair after cerebral ischemia insults

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Abstract

Choline acetyltransferase (ChAT)-positive neurons in neural stem cell (NSC) niches can evoke adult neurogenesis and restore impaired brain function after injury, such as acute ischemia stroke (AIS). However, the relevant mechanism by which ChAT+ neurons develop in NSC niches still remains poorly understood. Our RNA-seq analysis revealed that dimethylarginine dimethylaminohydrolase1 (DDAH1), a hydrolase for asymmetric dimethylarginine (ADMA), regulated genes responsible for the synthesis and transportation of ACh (e.g. Chat) post stroke insults. As expected, DDAH1 was clinically elevated in the peripheral blood of AIS patients which was positively correlated with AIS severity. By comparing the results among DDAH1 general knockout (KO) mice, transgenic (TG) mice and wild-type (WT) mice, we discovered that DDAH1 upregulated the proliferation and neural differentiation of NSCs in the subgranular zone (SGZ) under ischemic insults. As a result, it may promote cognitive and motor function recovery against the stroke impairments, while these neuroprotective activities are dramatically suppressed by NSC-specific conditional knockout of DDAH1 in mice.

Short CV

Dr ZHAO is the Associate Professor in Department of Pharmacology, School of Basic Medical Sciences, capital medical university, China. In Year 2005, she received the PhD degree from Chinese Academy of Medical Sciences and Peking Union Medical College. Currently, her group has focused on the preclinical pharmacological study of the antioxidants and neuroprotectants against the neurodegenerative insults. As the first author or corresponding author, Zhao has published more than 40 papers in peer reviewed international journals (e.g. Acta Pharmaceutica Sinica B, Free Radical Biology and Medicine, and Phytomedicine).



A physiological role of H_2O_2 in sleep homeostasis

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Abstract

Sleep is essential for animal survival and health, and one proposed fundamental function of sleep is to combat oxidative stress. Yet, the relationship between sleep and reactive oxygen species (ROS) in the mammalian brain remains unclear. Here we show that in mice, sleep deprivation caused an overall brain ROS accumulation, with stronger increases in sleep regulating regions. By real-time imaging of intracellular H_2O_2 across sleep-wake cycles, we found that cytosolic but not mitochondrial H_2O_2 in key sleep neurons regulates sleep initiation. Elevating H_2O_2 optogenetically or chemogenetically in these neurons enhances sleep generation, while reducing intracellular H_2O_2 by overexpressing antioxidant enzyme suppresses sleep. These results highlight a physiological role for H_2O_2 in sleep homeostasis and uncover a feedback mechanism that triggers sleep for antioxidant protection.

Short CV

Liu Danqian got her bachelor's degree from the University of Science and Technology of China (USTC) in 2010. In 2016, she completed her Ph.D. at the Institute of Neuroscience, Chinese Academy of Sciences, studying synaptic plasticity underlying fear memory. From 2016 to 2020, she got her postdoctoral training at the University of California, Berkeley. Since 2020, Danqian is a principal investigator at the Center for Excellence in Brain Science and Intelligent Technology, Chinese Academy of Sciences. Her main researches focus on the functions of sleep in maintaining cellular homeostasis in the brain.

NRF2 translocation from dendrites to nucleus in glutamatergic pyramidal neurons induced by uncoupling of post-synaptic neuronal nitric oxide synthase via calcium influx through NMDAR

Ishii Tetsuro

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Abstract

The transcription factor NRF2 plays a key role in maintaining high glutathione (GSH) levels in cells. In neurons, NRF2 is thought to control expression of the Na+-dependent amino acid transporter EAAT3, which takes up glutamate and cysteine, and γ-glutamyl-cysteine ligase for GSH synthesis. Hippocampal pyramidal glutamatergic neurons express both neuronal and endothelial nitric oxide synthases (nNOS and eNOS), and nitric oxide (NO) is important in potentiation of synaptic transmission. However, under synaptic depression, post-synaptic nNOS uncouples following glutamate stimulation of NMDA receptors to induce calcium influx. Subsequent reactive oxygen and nitrogen species generation would lead to GSH consumption. Uncoupled NOS-derived superoxide is converted to hydrogen peroxide (H_2O_2) via superoxide dismutase, and we previously proposed mechanisms for NRF2 nuclear translocation by low levels of hydrogen peroxide (Ishii T et al., Free Radic Biol Med (2022) 191: 191-202). The scaffolding protein PSD-95 binds nNOS in the post-synaptic membrane, while caveolin-1 tethers NRF2 in peri-synaptic lipid rafts. H_2O_2-activated ERK and p38 MAPKs, and calcium-activated PKC cooperate in membrane translocation and activation of the GSH sensor neutral sphingomyelinase 2 to generate the signaling molecule ceramide. Ceramide in turn activates the PKCζ/Casein kinase 2 signaling pathway to phosphorylates NRF2. ERK and cyclin-dependent kinase 5 phosphorylate NRF2 to induce a structural change by prolyl cis/trans isomerase Pin1 association to facilitate binding with importins. Subsequently, the dynein motor complex carries the NRF2 signaling complex toward nuclear pores along microtubules (Ishii T et al., Antioxidants (2023) 12:274).

Key words: NRF2, nitric oxide, superoxide, neuronal NO synthase, nuclear translocation, glutamate



Peroxynitrite reduces Treg cell expansion and function by mediating IL-2R nitration and aggravates multiple sclerosis pathogenesis

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Abstract

T-helper 17 cells and regulatory T cells (Treg) are critical regulators in the pathogenesis of multiple sclerosis (MS) but the factors affecting Treg/Th17 balance remains largely unknown. Redox balance is crucial to maintaining immune homeostasis and reducing the severity of MS but the underlying mechanisms are unclear yet. Herein, we tested the hypothesis that peroxynitrite, a representative molecule of reactive nitrogen species (RNS), could inhibit peripheral Treg cells, disrupt Treg/Th17 balance and aggravate MS pathology by inducing nitration of interleukin-2 receptor (IL-2R) and down-regulating RAS/JNK-AP-1 signalling pathway. Experimental autoimmune encephalomyelitis (EAE) mouse model and serum samples of MS patients were used in the study. We found that the increases of 3-nitrotyrosine and IL-2R nitration in Treg cells were coincided with disease severity in the active EAE mice. Mechanistically, peroxynitrite-induced IL-2R nitration down-regulated RAS/JNK signalling pathway, subsequently impairing peripheral Treg expansion and function, increasing Teff infiltration into the central nerve system (CNS), aggravating demyelination and neurological deficits in the EAE mice. Those changes were abolished by peroxynitrite decomposition catalyst (PDC) treatment. Furthermore, transplantation of the PDC-treated-autologous Treg cells from donor EAE mice significantly decreased Th17 cells in both axillary lymph nodes and lumbar spinal cord, and ameliorated the neuropathology of the recipient EAE mice. Those results suggest that peroxynitrite could disrupt peripheral Treg/Th17 balance, and aggravate neuroinflammation and neurological deficit in active EAE/MS pathogenesis. The underlying mechanisms are related to induce the nitration of IL-2R and inhibit the RAS/JNK-AP-1 signalling pathway in Treg cells. The study highlights that targeting peroxynitrite-mediated peripheral IL-2R nitration in Treg cells could be a novel therapeutic strategy to restore Treg/Th17 balance and ameliorate MS/EAE pathogenesis. The study provides valuable insights into potential role of peripheral redox balance in maintaining CNS immune homeostasis.

Key words: Peroxynitrite, Nitration, IL-2R, Experimental autoimmune encephalomyelitis, Multiple sclerosis



ROMO1 shields the mitochondrial cysteinome from oxidations in diseases and aging

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Abstract

Reactive thiols of proteinaceous cysteines are vital to cell biology by serving as sensor, effector and buffer of environmental redox fluctuations. Being the major source as well as the prime target of reactive oxygen species (ROS), the mitochondrion confronts great challenges in preserving its thiol pool. Here we identify ROS modulator 1 (ROMO1), a small inner mitochondrial membrane protein, as a master thiol-protector of the mitochondrial cysteinome. Being redox sensitive and reactive, ROMO1 shields functional thiols by scavenging ROS and forming intermolecular disulfides, thereby preventing irreversible thiol oxidations. Such ROMO1-mediated thiol protection exerts extensively beneficial effects on mitochondria, such as promoting energy metabolism and Ca2+ uniport while inhibiting vicious membrane permeability transition. Importantly, ROMO1 not only confers strong cardiac protection against oxidative injuries, but also reverses mitochondrial cysteinome oxidations in multiple organs and retards their functional declines in aging. These findings unravel an exquisite thiol-protection mechanism of the mitochondrial cysteinome, and mark ROMO1 as a potential target for combating oxidative stress and improving healthspan.

Key words: ROMO1, cysteine oxidations, mitochondria

Short CV

Dr. Xianhua Wang is an investigator of College of Future Technology, Peking University. She received her bachelor and Ph.D degrees in biochemistry and molecular biology from College of Life Sciences of Peking University, China, followed by postdoctoral training in Institute of Molecular Medicine, Peking University. Her research focuses on mitochondrial protection and mitochondrial signal transduction with the aid of cutting age *in vivo* mitochondrial imaging technique.



Alox15/15-HpETE Aggravates Myocardial Ischemia-Reperfusion Injury by Promoting Cardiomyocyte Ferroptosis

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Abstract

Myocardial ischemia-reperfusion (I/R) injury causes cardiac dysfunction to myocardial cell loss and fibrosis. However, the time point at which the various modes of cell death occur after reperfusion injury and the mechanisms underlying ferroptosis regulation in cardiomyocytes are still unclear. We found that apoptosis and necrosis occurred in the early phase of I/R injury, and that ferroptosis was the predominant form of cell death during the prolonged reperfusion. We demonstrated that Alox15 expression was specifically increased in the injured area of the left ventricle below the suture and colocalized with cardiomyocytes. Furthermore, myocardialspecific knockout of Alox15 in mice alleviated I/R injury and restored cardiac function. 15-Hydroperoxyeicosatetraenoic acid (15-HpETE) was identified as a trigger for cardiomyocyte ferroptosis. We explored the mechanism underlying its effects and found that 15-HpETE promoted the binding of Pgc1α to the ubiquitin ligase ring finger protein 34, leading to its ubiquitin-dependent degradation. Consequently, attenuated mitochondrial biogenesis and abnormal mitochondrial morphology were observed. ML351, a specific inhibitor of Alox15, increased the protein level of Pgc1α, inhibited cardiomyocyte ferroptosis, protected the injured myocardium, and caused cardiac function recovery.

Key word: 15-HpETE , Ferroptosis, ML351, Myocardium

Short CV

Xu Zhang, PhD, professor of Tianjin Medical University.

Mainly engaged in the research of "Pathophysiological Role and Regulatory Mechanisms of Bioactive Lipid Molecules".

Papers published in journals including Circulation, Circulation Research, Eur Respir J, Nature Communication, etc.



The Mechanisms of Plasma-Activated Solutions in Treating Atopic Dermatitis

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Abstract

Cold atmosphere plasma has been applied to many diseases and has been extensively studied in dermatology in recent years, addressing conditions such as wound healing, psoriasis, dermatitis, and vitiligo. Compared to direct plasma treatment, plasma-activated solutions (PAS) offer several advantages, including enhanced safety, better portability, higher patient acceptance, and greater convenience for application in sensitive areas, such as skin folds. These benefits have brought PAS increasing attention in the field.

Atopic dermatitis (AD) is a widespread chronic inflammatory skin condition that remains incurable, creating an urgent need for alternative therapies or daily management strategies with fewer side effects. Therefore, in this work we explore the potential of PAS as a promising treatment option for AD.

Our *in vivo* experiments demonstrated the therapeutic efficacy of PAS in a mouse model of AD, including its effectiveness in alleviating AD-like symptoms, reducing inflammatory markers, and reducing mast cell and macrophage infiltration. These benefits were linked to the activation of the antioxidant molecule Nrf2. Further, in vitro experiments using THP-1-derived macrophages showed that PAS reduced ROS levels and regulated cytokine expression in TNF-α/IFN-γ-stimulated keratinocytes and LPS-stimulated M1 macrophages. PAS also upregulated antioxidant stress molecules like Nrf2, HO-1, NQO1, and PPAR-γ in both cell types.

Overall, PAS demonstrated potent therapeutic potential for AD without notable side effects. Our research provided a promising approach to AD treatment and may open a novel avenue for treating.

Short CV

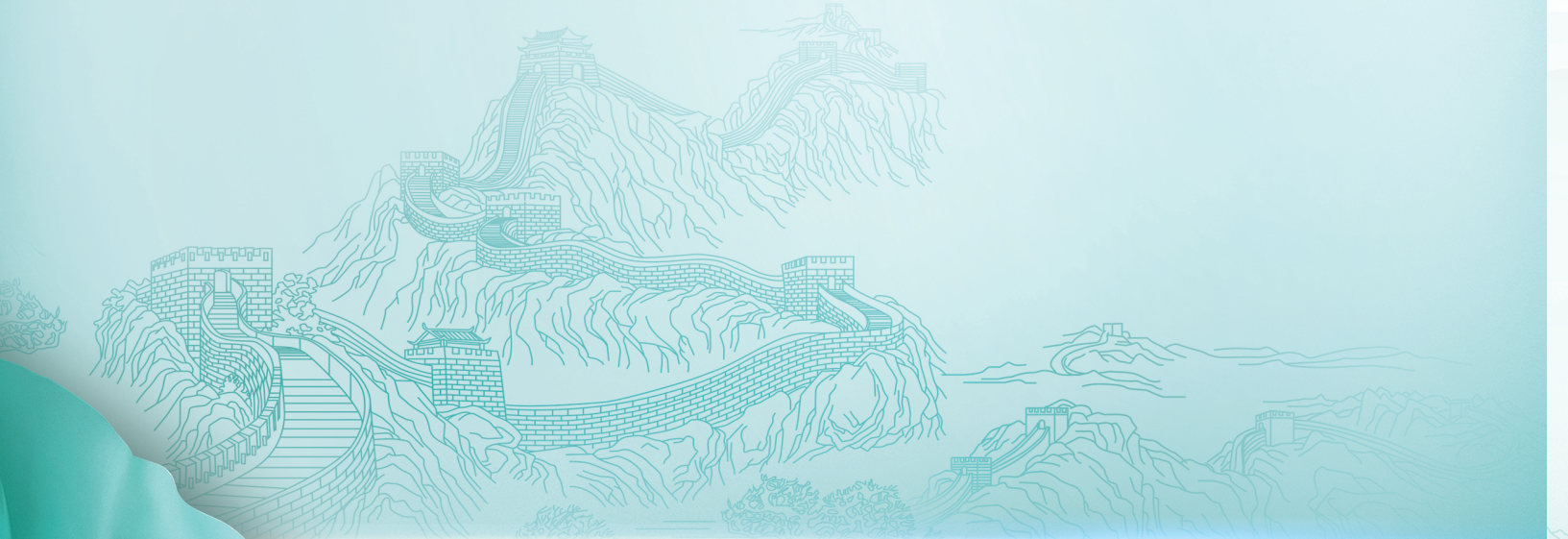
Department of dermatology, Xi'an Jiaotong University, Shaanxi, China, Professor

Department of dermatology, the First Affiliated Hospital of Xi'an Jiaotong University, Shaanxi, China, Director

Research Interests: Scientific research: pathogenesis of psoriasis and skin cancers; Clinical aspects: treatment of psoriasis and atopic dermatitis; pathological diagnosis of rare skin diseases; laser cosmetic treatment of skin

Symposium-7(S7)

Redox signaling in organelles/cell fate/development/reproduction





Chair: Dongyun Shi (施冬云)

Fudan University, China

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Short CV

Dr. Dongyun Shi received his BS and MS from Fudan University and PhD of Biochemistry and Cell Biology from Kings College, University of London, UK. Currently, she is a professor in the Department of Biochemistry, School of Basic Medicine, Fudan University, at Shanghai, China. Dr. Shi's research interests include the molecular and cellular mechanisms of reactive oxygen species in regulating metabolic diseases, focusing on the redox modulation of the metabolism in tumors and diabetes. She was selected as Pujiang Talent of Shanghai, and won the second prize of the Natural Science Award of the Ministry of Education of the People's Republic of China.



Airborne PM2.5-induced oxidative stress aggravates neurotoxicity in olfactory bulb

Wenjun Ding (丁文军)

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Abstract

Recent epidemiological studies have shown that exposure to atmospheric fine particulate matter (PM2.5) is associated with various central nervous system (CNS) diseases, including Alzheimer's disease and Parkinson's disease, etc. PM-induced microglia activation, neuroinflammation and oxidative stress are critical to the neurodegenerative diseases in the CNS. However, the cellular and molecular mechanisms by which PM2.5 induces neurotoxicity are not well understood. In our study, we found that PM2.5 entered the brain via olfactory pathway. PM2.5 significantly increased the concentrations of metal elements in mice olfactory bulb (OB). Compared with the controls, PM2.5 triggered oxidative stress and activated microglia in mice OB. Moreover, the release levels of glutaminase-containing EVs (40-200 nm) and the number of apoptotic cells were significantly increased following PM2.5 exposure. Furthermore, nuclear factor erythroid 2-related factor 2 (Nrf2) deficiency resulted in lower levels of antioxidant enzymes, greater induction of oxidative stress, microglia activation, inflammation and nuclear factor kappa B (NF-κB) activation in PM2.5-treated OBs. Taken together, glutaminase-containing EVs are crucial neurotoxic factors released by PM2.5-activated microglia. Nrf2-mediated defenses against oxidative stress will help develop new strategies for the prevention and treatment of diseases associated with airborne PM2.5 pollution.

Key words: PM2.5, oxidative stress, olfactory bulb, glutaminase, neuroinflammation, microglia, extracellular vesicles

Short CV

Wenjun Ding, Professor, Vice Dean, College of Life Sciences, University of Chinese Academy of Sciences (UCAS). At UCAS, he established and is leading a research group "Environment and Health". This group is focusing on the toxicological implications of Urban airborne fine particulate matter: sources, chemical composition, and possible underlying mechanism, especially cardiopulmonary diseases attributable to indoor and/or outdoor air pollution.



Deciphering umbilical cord blood hematopoietic stem/progenitor cell alterations in alpha-thalassemia using single-cell transcriptomics

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Abstract

Alpha-thalassemia represents one of the most globally widespread monogenic disorders. The main pathological features of α-thalassemia include ineffective erythropoiesis, hemolysis, and iron overload. Although the pathophysiology of α-thalassemia has been extensively studied in many aspects, it remains unclear how α-thalassemia affects the differentiation process of hematopoietic stem/progenitor cells (HSPCs). Here, we isolated CD34+ HSPCs from the umbilical cord blood of fetuses with α-thalassemia and conducted single-cell RNA sequencing. After stringent quality control, a total of 49,263 CD34+ HSPCs were included in downstream analysis. Based on known marker genes, 16 different cell clusters were annotated. Pseudotime analysis showed that CD34+ HSPCs from α-thalassemia and control exhibited similar differentiation trajectories, but there were differences in the number of cells differentiating along these paths. Compared to the control group, the MEP cell and Mk P cell proportion were significantly elevated in α-thalassemia, while the proportion of pro-B cells was generally reduced, with the differences being statistically significant.

We assessed the transcriptomic changes in each cell subtype in the thalassemia group and found that most of the differentially expressed genes were downregulated. Gene set enrichment analysis of these DEGs revealed significant enrichment in several key biological processes and signaling pathways, including positive regulation of GTPase activity, the PI3K-AKT signaling pathway, G protein-coupled receptor signaling pathway, and TGF-β receptor signaling pathway. The expression of genes associated with these processes was significantly reduced, suggesting that these biological functions may be suppressed or weakened. Moreover, in the HSC/MPP cell subtype, there was an observed increase in oxidative phosphorylation. An increase in oxidative phosphorylation is often accompanied by a rise in reactive oxygen species (ROS) levels, and excessive ROS can trigger oxidative stress. Oxidative stress can lead to cellular damage, impair cell function, and subsequently affect the self-renewal and differentiation potential of HSCs. These findings suggest that increased oxidative phosphorylation and ROS production may be key mechanisms underlying impaired HSC function and disrupted intracellular homeostasis in thalassemia. Elevated ROS levels may further exacerbate oxidative stress in

thalassemia patients, thus impacting overall hematopoiesis and normal immune system development.Further differentiation trajectory analysis revealed that, compared to the control group, the HSC/MPP cell population in α-thalassemia showed a marked increase in differentiation toward MEP.

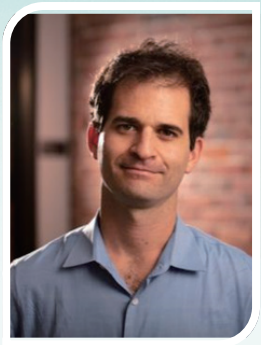
In pro-B cells of α-thalassemia, the expression of genes associated with programmed cell death, such as BAX and BAD, was significantly increased. The activation of the TP53 signaling pathway was accompanied by the upregulation of a series of direct effector genes, including CHEK1, PCNA, and PRMT1. Additionally, GO analysis indicated that pathways related to B cell proliferation and immature B cell differentiation were significantly downregulated in α-thalassemia, suggesting that pro-B cell apoptosis is significantly increased, and their differentiation process is severely impaired. These defects lead to a significant reduction in pro-B cell numbers, which may ultimately contribute to abnormal immune system function in α-thalassemia patients.

For the first time, we utilized single-cell transcriptomics to reveal the altered transcriptomic characteristics of CD34+ HSPCs in the umbilical cord blood of α-thalassemia patients. We discovered significant differences in the differentiation fate of HSC/MPP between α-thalassemia patients and controls. This differentiation tendency is likely a compensatory response by the patients to ineffective erythropoiesis and to address the insufficiency of red blood cell production in their bodies.

Key words: α-thalassemia, hematopoietic stem and progenitor cells, single-cell RNA sequencing, lineage differentiation bias;ROS

Short CV

Ma Yanlin is a distinguished expert recognized by the National Health Commission as an Outstanding Young and Middle-aged Expert for her significant contributions. She is also a recipient of the State Council Special Allowance and holds the title of Second-level Professor. Ma has long been engaged in clinical and laboratory work in the fields of reproductive medicine and genetic counseling.



Identification of druggable and redox vulnerabilities in cancer

Liron Bar-Peled

Harvard Medical School Department of Medicine, USA

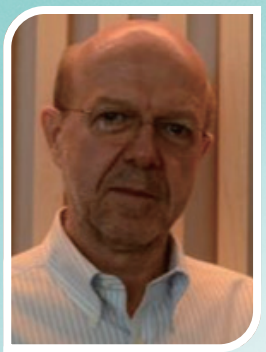
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Abstract

Multiple chemotherapies are proposed to cause cell death in part by increasing the steady-state levels of cellular reactive oxygen species (ROS). However, for most of these drugs exactly how the resultant ROS function and are sensed is poorly understood. In particular, it's unclear which proteins the ROS modify and their roles in chemotherapy sensitivity/resistance. To answer these questions, we examined 11 chemotherapies with an integrated proteogenomic approach identifying many unique targets for these drugs but also shared ones including ribosomal components, suggesting one mechanism by which chemotherapies regulate translation. We focus on CHK1 which we find is a nuclear H_2O_2 sensor that promotes an anti-ROS cellular program. CHK1 acts by phosphorylating the mitochondrial-DNA binding protein SSBP1, preventing its mitochondrial localization, which in turn decreases nuclear H_2O_2. Our results reveal a druggable nucleus-to-mitochondria ROS sensing pathway required to resolve nuclear H_2O_2 accumulation, which mediates resistance to platinum-based chemotherapies in ovarian cancers.

Short CV

Liron received his Bachelor of Science degree in Biochemistry from the University of Georgia in 2004. Liron received his PhD in Biology from the Massachusetts Institute of Technology, where he used advanced cellular and molecular techniques to uncover how nutrients are sensed by the mTORC1 pathway in the laboratory of David Sabatini. In 2013 as a Damon Runyon Postdoctoral fellow, he joined the laboratory of Ben Cravatt at the Scripps Research Institute to understand how cancer cells respond to oxidative stress. Employing chemical, proteomic and biochemical approaches, Liron revealed new druggable components of the NRF2 antioxidant response pathway uncovering new mechanisms by which NRF2 regulates metabolic pathways. In early 2019, Liron joined the Center for Cancer Research at the Massachusetts General Hospital and the Department of Medicine at Harvard Medical School. He is currently an Associate Professor of Medicine in Biological and Biomedical Sciences.



An endoplasmic reticulum-based model of intercellular redox communication

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Short CV

Francisco R. M. Laurindo graduated from the University of São Paulo School of Medicine, São Paulo, Brazil and underwent training in basic research in Physiology and Pharmacology at the Uniformed Services University of the Health Sciences, in Bethesda, MD, USA. Back to the Heart Institute, USP, he started an investigative research track on redox vascular biology, materializing into the Vascular Biology Laboratory at this institution, which he leads since 2008. His major research interests are mechanisms and regulation of oxidant and thiol signaling in vascular cells and their physiological implications for vessel remodeling in disease. Dr. Laurindo has authored or co-authored over 185 publications in peer-reviewed journals, and supervised over 25 PhD students and 25 post-doctoral fellows, in addition to several undergraduate trainees. Since 2008 (until present), he is the Vice-coordinator of Cepid-Fapesp Redoxoma, a large network project. He is a member of the Brazilian Academy of Sciences since 2012 and a member of its Board of Directors from 2016-2022; a member of Fapesp Research Agency Advisory Committee in Health Sciences from 2008-2024. He served as Council Member of the SfRBM (Soc. for Redox Biol Med) from 2010-2014, President-elect from 2020-22 and is current SfRBM President since 2022. He belongs to the Editorial Boards of Free Radical Biology and Medicine, Clinical Science and Circulation Research. He was vice-chair (2014) and chair (2016) of the Gordon Research Conference on Nox Family NADPH Oxidases.



Apoptotic Effect of Terfenadine, a Histamine H1 Receptor Antagonist, and Terfenadine-loaded Human Serum Albumin Nanoparticles in Colorectal Cancer and Glioblastoma Cells

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Abstract

Terfenadine is a second-generation histamine H1 receptor antagonist initially used for treatment of allergy. However, it was withdrawn from market due to serious adverse effects of the drug. Recently, we investigated that terfenadine exhibited marked antitumor effects against HCT116 colorectal cancer cells by inducing apoptosis via cleavage of caspase and PARP and release of cytochrome C. Moreover, terfenadine treatment decreased the phosphorylation of STAT3 and downregulated the expression of its target gene products such as cyclin D1, cyclin D2, cyclin D3 and survivin. In addition, terfenadine treatment led to aberrant reactive oxygen species generation (ROS) in HCT116 cells which could trigger apoptosis. Moreover, terfenadine showed prominent antitumor effects against U87MG glioblastoma cells. Addressing the challenge for revival of terfenadine in clinic, we attempted to develop human serum albumin (HSA) based nanoplatform incorporating terfenadine as payload which is speculated to minimize the adverse effects associated with the drug and increase the cell permeability in cancer cells. Indeed, terfenadine-loaded HSA-NPs (T-HSA-NPs) exhibited desired particle characteristics along with sustained release pattern. Incubation of cells with T-HSA-NPs showed enhanced cellular internalization, and higher cytotoxic effects in U87MG and HCT116 cells as compared to the free drug. In conclusion, our results illuminate on the potential of T-HSA-NPs for chemotherapy and represent efficient approach to enhance the permeability of terfenadine across blood brain barrier which is a key to the treatment of brain tumors.

<Baniya M.K.et al. Front. Pharmacol. 2024;15:118266.>

Short CV

ADDRESS: College of Pharmacy, Keimyung University, South Korea

EDUCATION: Ph.D. in Pharmacy, Seoul National University, Seoul, Korea, 2004

RESEARCH/PROFESSIONAL EXPERIENCE:

2011 – Present, Professor, College of Pharmacy, Keimyung University

2004 – 2011, Research Fellow, Laboratory of Toxicology and Pharmacology, NIEHS/NIH, Research Triangle Park, NC, USA

MAJOR: Molecular Toxicology, Cancer Biology

Symposium-8(S8)

Redox and aging ② “Targeting Redox and Mitochondria to delay aging and prevent age-related diseases”Forum



Chair: Yong Zhang (张勇)

Tianjin Key Laboratory of Exercise, Physiology and Sports Medicine, Tianjin University of Sport, Tianjin, China

Email: yzhang@tjus.edu.cn

Short CV

Dr. Yong Zhang got his BS from Hubei University in Biology and his PhD from Beijing Sports University, majoring in exercise physiology. He is a professor of Tianjin University of Sport and adjunct professor of Beijing Sport University and Xi'an Jiaotong University, and was International Visiting Professor of Southern Cross University, Australia. Prof. Zhang's Research interest is Cellular and Molecular Exercise Physiology: 1) Exercise-induced Oxidative Stress and Mitochondrial Biology, and 2) Mitochondrial Homeostasis Regulation and Integrative Physiology of Exercise. He has published more than 100 papers on exercise on health.



Redox regulation of mitophagy by targeting PINK1

Hanming Shen (沈汉明)

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Abstract

Mitophagy is a selective form of autophagy for clearance of damaged mitochondria via the autophagy-lysosome pathway. Among various mitophagy regulators, PINK1, a protein kinase, and Parkin, an E3 ligase, are two critical players, with important implications in neurodegenerative disorders such as Parkinson's disease (PD). In our recent studies, we focus on the upstream regulatory mechanisms of PINK1, including the following aspects: (1) Via a whole genome CRISPR-Cas9 screening, we identified glucose-6-phosphate dehydrogenase (G6PD), a key enzyme in glycolysis antioxidant defense mechanism, as an important positive regulator of mitophagy via direct interaction and stabilization of PINK1; (2) Establishing the regulatory function of reactive oxygen species (ROS) on mitophagy: H_2O_2 is capable of impairing the mitophagy process via PARP1 activation and PARylation of key mitophagy machinery. Our results thus provide a deeper insight into the molecular mechanisms in control of PINK1, the guardian of mitochondria and lay foundation for development of novel interventional strategies in PINK1- and mitophagy-related human diseases such as neurodegeneration and cancer.

Key words: Mitophagy, PINK1, G6PD, ROS, PARylation

Short CV

Dr. Han-Ming Shen is currently a Chair Professor and Associate Dean (Teaching), Faculty of Health Sciences, University of Macau, and a visiting Professor, Yong Loo Lin School of Medicine, National University of Singapore (NUS). He received his Bachelor of Medicine (1985), Master of Medicine (1988) and PhD (1996) from Zhejiang Medical University and NUS, respectively. He also received his postdoc training in National Cancer Institute (NCI), National Institutes of Health (NIH). His research is focused on the autophagy-lysosome pathway, mitophagy, as well as on glucose metabolism in cancer cell biology. Dr. Shen has published more than 250 papers, with more than 44,000 citations and H-index at 104 (Google scholar). Currently, he serves as the Associate Editor for Autophagy, and as member of editorial board for several other journals such as Life Metabolism, Burns & Trauma and Aging Cell.



NAD^+ dependent UPR^{mt} activation underlies intestinal aging caused by mitochondrial DNA mutations

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Abstract

Aging in mammals is accompanied by an imbalance of intestinal homeostasis and accumulation of mitochondrial DNA (mtDNA) mutations. However, little is known about how accumulated mtDNA mutations modulate intestinal homeostasis. We observe the accumulation of mtDNA mutations in the small intestine of aged male mice, suggesting an association with physiological intestinal aging. Using polymerase gamma (POLG) mutator mice and wild-type mice, we generate male mice with progressive mtDNA mutation burdens. Investigation utilizing organoid technology and *in vivo* intestinal stem cell labeling reveals decreased colony formation efficiency of intestinal crypts and LGR5-expressing intestinal stem cells in response to a threshold mtDNA mutation burden. Mechanistically, increased mtDNA mutation burden exacerbates the aging phenotype of the small intestine through ATF5 dependent mitochondrial unfolded protein response (UPRmt) activation. This aging phenotype is reversed by supplementation with the NAD^+ precursor, NMN. Thus, we uncover a NAD^+ dependent UPRmt triggered by mtDNA mutations that regulates the intestinal aging.

Key words: NAD, mitochondrial DNA, Aging, redox state, mitochondrial unfolded protein response

Short CV

Professor Xingguo Liu, was honored as "Distinguish Youth Foundation" of National Natural Science Foundation, Chief Scientist of the National Key Research and Development Program of China, 1st finisher of the first prize of the Guangdong Science and Technology Award in Natural Science, The Shulan Medicine Youth Award by the Academician Shusen Lanjuan Talent' Foundation, The Ying Ding Science and Technology Award, "2016 Stem cell Young Investigator Award" from Chinese Society for Cell Biology and "Young Bioenergeticist Award" of the International Biophysical Society. He is the Executive Editor of Science Bulletin, the council member of the Asian Society for Mitochondrial Research and Medicine, and the council member of the Biophysical Society of China. Dr. Liu has published more than 80 papers, which have been cited for more than 6000 times. Since 2015, he has published 32 research papers as corresponding author (4 IF>20, 22 IF>9), such as Cell Metabolism (2016,2018, 2024), Nature Metabolism, Nature Structural & Molecular Biology, Nature Communications (2022,2024), Science Advances (2019, 2022), Advanced Science, Hepatology. Among his papers, 3 were recommended by F1000, 9 were chosen as cover, and his findings have been listed in 17 books such as Encyclopedia of Biological Chemistry. He obtained 10 authorized patents (including one PCT). Dr. Liu has been the invited speaker in Keystone Symposia.



Truncated oxidized phospholipids mediate synchronized ferroptosis and contribute to acute kidney injury

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Abstract

Synchronized ferroptosis is suggested to play a significant role in nephron loss during acute kidney injury (AKI). However, the underlying mechanisms mediating synchronized ferroptosis in renal tubular injury remain unclear. Our study reveals that truncated oxidized phospholipids and platelet-activating factor (PAF) serve as mediators of synchronized ferroptosis, contributing to the pathogenesis of acute kidney injury (AKI). PAF initiates biomembrane permeabilization and triggers cell death in neighboring cells. PAF-acetylhydrolase (II) (PAFAH2), a phospholipase A2 (PLA2) family enzyme that specifically removes truncated acyl chains from phospholipids, along with PAF-specific antibodies that bind and neutralize PAF, effectively suppressed synchronized ferroptosis. Genetic or pharmacological inhibition of PAFAH2 led to increased PAF production, which augmented PAF-mediated synchronized ferroptosis and exacerbated ischemia/reperfusion (I/R)-induced AKI. Our findings uncover a novel mechanism underlying synchronized ferroptosis and propose a promising therapeutic strategy for the intervention of acute kidney injury (AKI).

Short CV

Dr. Quan Chen is the Professor and Director of Life Science in Nankai University (Tianjin, China) and Director of State Key Laboratory of Medicinal Chemical Biology. He works on the role of mitochondria in apoptosis, the molecular regulation of mitochondrial autophagy and the role of mitochondrial dysfunction in the occurrence of neurodegenerative diseases such as Alzheimer's disease, Parkinson's syndrome as well as cancer stem cells in tumorigenesis and metastasis. He serves on the editorial boards of JBC, Cell Research, Cell Death and Disease and Chinese Science journals. Professor Chen has received the American Association for Cancer Research (AACR-AFLAC) Young Scholar Award for Cancer Research, the 4th Tan Jiazhen Life Science Award Innovation Award, and the National Outstanding Scientific and Technological Worker. The research interests in Dr. Chen's laboratory: research apoptosis signal transduction and mitochondrial biology, focus on the role of mitochondria in apoptosis signaling, the role of apoptosis in tumorigenesis; analyze the molecular mechanisms of mitochondrial dynamic changes and quality control and theirs' function in neurodegenerative diseases; screen natural small molecule compound by targeting of key molecules of mitochondria and apoptosis; research on cancer stem cells.



NAD^+-dependent enzymes in health and disease: Our key findings on NADH-ubiquinone oxidoreductase in diabetic pancreas

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Abstract

It has been established that there is an oversupply of NADH in diabetes which can lead to cellular redox imbalance. As mitochondrial complex I is the major site accepting NADH electrons, how complex I responds to this redox imbalance and the consequences of this response have not been investigated, particularly in the pancreas. Therefore, we aimed to study the effects of NADH/NAD^+ redox imbalance on complex I and mitochondria in diabetic pancreas. Materials and Methods: Type 2 diabetic animal models such as Zucker diabetic rat and db/db mouse were from Charles River. Type I diabetes was induced by streptozotocin injection in rats. Complex I activity was determined by native gel electrophoresis. NADH/NAD^+, ATP concentrations, and apoptosis were measured using commercially available kits, respectively. Lipid peroxidation, protein oxidation, and H_2O_2 levels were also measured. Results: Pancreatic mitochondrial complex I was found to be highly activated by diabetic hyperglycemia in both type I and type 2 diabetes. Moreover, while NADH content was increased, the levels of ATP levels was decreased. We also discovered that the effect of NADH/NAD^+redox imbalance on complex I leads to mitochondrial oxidative stress and cell death. Conclusion: Our study suggests that complex I hyperactivity in diabetic pancreas is involved in the pathogenesis of diabetes.

Key words: Redox imbalance, mitochondria, oxidative stress, diabetes, redox imbalance, NADH-ubiquinone oxidoreductase

Short CV

Education	1996, PhD, University of California-Berkeley
	1990, MS, Institute of Biophysics, Chinese Academy of Sciences
	1987, BS, Peking University
Postdoc	2000, Southern Methodist University, Dallas, Texas
Membership	International Society of Nephrology
	Society for Redox Biology and Medicine
	American Society for Biochemistry and Molecular Biology
	International Society for Neurochemistry
	American Association of Advancement of Science
	Golden Bear Life Member-Cal Alumni Association, UC Berkeley
Positions	Tenured Professor, Pharmaceutical Sciences
Department/Affiliation	Pharmaceutical Sciences, College of Pharmacy
	Graduate School of Biomedical Sciences
	Institute for Aging and Alzheimer's disease Research

Education Program Involvement

Pharmacology and Medicinal Chemistry, PharmD; Pharmaceutical Sciences and Pharmacotherapy, PhD; Biomedical Sciences, PhD

Publications: https://www.ncbi.nlm.nih.gov/myncbi/1d7TqQRoGlw/bibliography/public/



Mitochondrial drug NAD^+ anti-aging strategy

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Abstract

With the coming of aging population in the world, the incidence of aging-related diseases such as heart failure, neurodegenerative diseases, tumors, etc. will increase sharply day by day. As an important substance in mitochondria within intracellular, NAD^+ plays a variety of important roles in intracellular, involving multiple biological processes such as energy metabolism, DNA repair, signal transduction, antioxidant, immune regulation, and apoptosis.

Through cardiomyocyte models, Sprague Dawley(SD) rats models, beagles models, and other animal models, compared with Novartis's LCZ696, it was confirmed that NAD^+ has an obvious advantage in anti-heart failure function key indicators such as left ventricular ejection fraction(LVEF) and myocardial infarction area. 60 candidate patients performed one Investigator-initiated clinical trial， and researchers also confirmed that NAD^+ can improve key indicators such as NT-proBNP and LVEF value in the heart-failure candidates patients group. Through pre-clinical research, a registered clinical trial of NAD^+ was carried out and approved by China Food and Drug Administration. The maximum ramping dose of the clinical trial human phase I reached 800 mg daily. It shows that the security in clinical trial phase I is fairly safe.

Compared with Memantine, Cholinesterase inhibitors such as Donepezil, Galantamine, and Rivastigmine, NAD^+ effectively inhibits reactive oxygen species (ROS) generated by free radicals, and increases the expression levels of Superoxide dismutase (SOD) mRNA and glutathione (GSH) mRNA. It improves neuronal cell viability. In a chronic cerebral ischemia rat model, NAD^+ reduces ROS damage and neuroinflammatory response in mitochondria, showing obvious anti-neuro- degenerative disease functions.

Immune checkpoint inhibitor drugs such as PD-1/L1 are primary clinical drugs used for cancer patients. However, the clinical tumor immune response rate is relatively low, at 15-20%, and PD-1/L1 is ineffective for most cancer patients. Research has shown that when NAD^+ is used in combination with PD-1/L1, NAD^+ can significantly improve the immune response rate of PD1/L1 in liver cancer and pancreatic cancer SD rats models. The tumor immune response rate increased from 42.2% to 82.8% in liver cancer SD rats models and from 18.6% to 64.7% in pancreatic cancer SD rats models. An Investigator-initiated clinical trial of the combination of NAD^+ and PD1/L1 drug is currently underway.

Symposium-9(S9)

Redox and cancer, infection and immunity





Chair: Pingping Shen (沈萍萍)

Nanjing University, China

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Short CV

Nanjing University, Jiangsu, P.R.C	BS	1980-1984	Biology
Nanjing University, Jiangsu, P.R.C	MS	1987-1990	Biology
Nanjing University, Jiangsu, P.R.C	Ph.D	1997-2000	Biochemistry and Molecular Biology

2018-	present, The Comprehensive Cancer Center, Nanjing Drum Tower Hospital
2011-2018	Head of Department, Department of Bioscience and Engineering, Jinling College, Nanjing University
2004-present	PI, State Key Laboratory of Pharmaceutical Biotechnology, Nanjing University; Professor, School of Life Sciences, Nanjing University
2003-2007	Associate Dean, School of Life Sciences, Nanjing University
2002-2003	Visiting Professor, University of California at San Diego

The research interests of my research group is to investigate he reprogramming mechanisms of macrophages within inflammatory microenvironments and their implications for human diseases. With a longstanding history of exploring the precise functions of specific macrophage subsets in the progression and prognostic outcomes of inflammation-related diseases, including cancer and metabolic inflammation. Our research integrates immunology, cell biology, and molecular biology to delineate the signaling transduction cascades, identify associated key regulatory nodes in macrophage function, and elucidate the exact mechanism by which these key nodes contribute to macrophage differentiation and polarization of immune responses. My lab employs engineering strategies, such as intracellular domain (ICD) assembly, metabolic/genetic editing, and chemical intervention, to develop chimeric antigen receptor macrophages (CAR-M) for solid tumors and autoimmune diseases. Additionally, leveraging synthetic biology, we have developed an intelligent CAR-M technology platform that allows for controllable assembly and tunable activity, endowing CAR-M with the capability of recognizing target antigens and precise manipulation of intracellular functional modules. Concurrently, we are advancing studies to design and implement in situ reprogramming approaches for *in vivo* macrophage engineering and CAR-M self-assembly.

Homepage: https://biopharm.nju.edu.cn//rcdw/yjry/20191126/i53941.html



The role of redox metabolism in drug resistance of cancer therapy

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Abstract

Many targeted drugs for tumor therapy primarily focus on redox reaction metabolism. During this process, sensitivity and resistance to these targeted drugs occured. Resistance to paclitaxel poses a major obstacle in esophageal squamous cell carcinoma (ESCC) treatment. A better understanding of the mechanisms underlying paclitaxel resistance could help identify prognostic biomarkers and improved therapeutic strategies. In this study, we established a patient-derived xenograft (PDX) model of acquired paclitaxel resistance and used RNA-sequencing to identify galectin-1, encoded by LGALS1, as a key mediator of resistance. Integrative analysis of clinical data and physiological studies indicated that serum galectin-1 levels were elevated in resistant patients and correlated with treatment outcomes before and during taxane therapy. Importantly, exposing cells to serum from resistant patients resulted in increased paclitaxel resistance compared to serum from sensitive patients, which was closely associated with galectin-1 concentrations in the serum. The specific clearance of galectin-1 from resistant patient serum significantly restored paclitaxel sensitivity, and inhibiting galectin-1, through knockdown or the pharmacologic inhibitor OTX008, increased sensitivity to paclitaxel. Galectin-1 inhibition reduced the activity of β-catenin, thereby inhibiting stem cell properties induced by the Wnt/β-catenin pathway. Furthermore, galectin-1 regulated MDR1 transcription through increased nuclear accumulation of β-catenin, thus increasing resistance to paclitaxel. Combining OTX008 with clinical taxane formulations effectively reversed paclitaxel resistance in vitro and *in vivo*. Elevated galectin-1 levels thus serve as an indicator of response to paclitaxel therapy in ESCC, offering a therapeutic intervention strategy to overcome drug resistance.

Short CV

Professor Zigang Dong, who got his PhD from Mailman School of Public Health, Columbia University, worked as the Director of The Hormel Institute, University of Minnesota for a long period of time, and currently is serving as a vice president of Zhengzhou University as well as the director of School of Medicine. Professor Dong focuses on the pathogenesis and prevention of cancer and is a leading figure in the field of cancer chemoprevention in the world. As of now, he has published more than 500 papers on journals such as Nature, Nature Reviews Cancer, Nature Cell Biology, etc. The total citations of his papers exceed 40,000. He also serves as an editorial board member or associate editor of journals including Cancer Research. He co-lunched npj Precision Oncology with Nature Publishing Group.

Professor Dong has presided over 50 plus projects funded by NIH and NSFC. Honors and awards won by him include McKnight Presidential Professor(the highest honor of University of Minnesota), MERIT Award(NIH, 2008), Stars in Nutrition and Cancer Lecturer Award(American Institute for Cancer Research, 2012), Yellow River Friendship Award (Henan Provincial Government, 2014) , Outstanding Contribution Award in Cancer Research(Society of American Asian Scientists, 2016), Contribution Award for overseas Chinese (All-China Federation of Returned Overseas Chinese, 2020) and Award for International Cooperation on Science and Technology (Henan Provincial Government, 2020). In 2021, professor Dong was included in the list of the top 100,000 scientists in the world. He ranks third among clinical researchers in China's mainland and is high on the list of scientists in Henan province. His name is frequently present in the lists of most cited Chinese researchers and the top 2% global researchers.



Redox biology regulated by selenoproteins- significance for biological defense and its relation to cancer

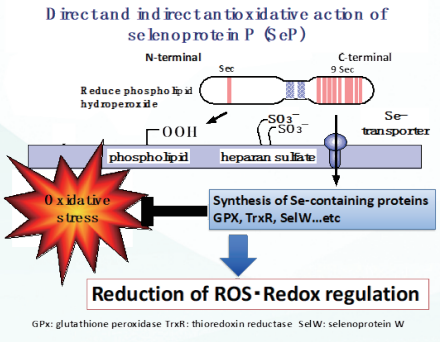
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Abstract

"Selenoprotein" is a general term for proteins that contain the essential trace element selenium in the form of selenocysteine (Sec), and 25 types are known in humans. Sec is encoded by one of the stop codons "UGA" and is called the 21st amino acid that can be translated. Selenoproteins play a significant role in redox reactions in the body, and representative selenoproteins are glutathione peroxidase (GPX), which reduces and detoxifies peroxides, and thioredoxin reductase, which is related to redox regulation. Selenoprotein P (SeP), which exists in plasma, is mainly synthesized in the liver and transports selenium to the periphery tissues. SeP has 10 Sec residues in the molecule, with one Sec residue on the N-terminus possessing GPX-like activity and nine Sec residues on the C-terminus functioning in selenium transport. In addition to its antioxidant action, SeP maintains cellular selenoproteins and plays an important role in antioxidant defense and redox regulation in the body (See Figure below). On the other hand, it has become clear that dysregulation of SeP expression is involved in various diseases such as diabetes and cancer. In this presentation, I will show the protective effect of SeP against electrophiles, which has been newly discovered as a biological function of SeP. Further, I introduce the latest findings on the relationship with severe brain cancer glioblastoma, associated with overexpression of SeP. In this lecture, I want to discuss the dual nature of selenoproteins from the view of redox biology.



Key words: Selenoprotein, Metabolism, Electrophile, Glioblastoma

Short CV

Yoshiro Saito, a professor at Tohoku University, received PhD from Hokkaido University in 2001 for his research on the structure and function of selenoprotein P. He became a postdoctoral researcher at AIST in 2002 and worked on oxidative stress biomarkers and their cellular responses. He researched oxidative stress and disease at Doshisha University since 2008 and has been in his current position since 2018. He has published 138 original papers with an H-index of 45 (Scopus). For details, see https://researchmap.jp/SY004680_MBM?lang=en.



Immune mechanisms and oxidative stress underlying the interaction of tuberculosis and diabetes

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Microbiology, Tumor and Cell Biology, Karolinska Instiutet, Sweden

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Short CV

Martin Rottenberg is a Professor of Infection Immunology and studies the role of the acquired immune response in infectious diseases, i.e. how the immune system reacts to infections such as tuberculosis and parasitic diseases.

Employments

Professor, Department of Microbiology, Tumor and Cell Biology, Karolinska Institutet, 2012

Degrees and Education

Docent, Karolinska Institutet, 2000



TRX Thioredoxin redox regulator of inflammasome: Redoxisome Concept

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Abstract

Thioredoxin (TRX) a small protein with reducing activity has TRX family members widely present from ancient organisms to humans. TRX plays various roles in intracellular signal transduction and resistance to bioistress including oxidative and chemical stress, opening a new research field of TRX-related "redox regulation". TRX has the protective effect of suppressing acute inflammation and cell death caused by oxidative stress, playing an important role in the prevention and treatment of stress-related diseases. In 2014, we proposed the concept that TRX forms a functional complex called "Redoxisome" with TRX-binding protein (TBP-2/VDUP1/TXNIP), which is involved in various pathological conditions. Oxidative stress is recognized as a critical factor influencing inflammasome activity, particularly NLRP-3, NLRP-1, NLRP-6, and NLRC-4. Recently, it has been continuously reported that TRX directly binds to the inflammasome to suppress or regulate inflammation. This research outcome has elucidated the anti-inflammatory mechanism of TRX connecting Redoxisome and Inflammasome, making it a promising target for the development of safe therapeutic drugs as an alternative to steroid preparations for respiratory inflammations, including COVID-19 infections, allergic diseases, skin hypersensitivity and psoriasis.

Key words: TRX, thioredoxin, redox, inflammation, inflammasome, redoxisome, ATL, TBP-2/TXNIP

Short CV

Junji Yodoi, M.D./Ph.D.,Prof.Em Kyoto University IVR/LiMe

1965 Tennoji High School Osaka

1971 Graduated from Kyoto University, Medical School

1971-1973 Postgraduate course of medicine, Kyoto University Hospital

1974-1975 Postgraduate course in Virus Research Institute, IVR

1975- Assistant Professor in The Institute for Immunology, Faculty of Medicine, Kyoto University

1977-1980 Research associate in Johns Hopkins University, Department of Medicine, Prof. Kimishige Ishizaka

1981- 1989 Assistant Professor The Institute for Immunology, Faculty of Medicine, Kyoto University

1989-1990 Professor in the Department of Prevention and Therapeutics, Institute for Virus Research, Kyoto University

1990-2000 Professor in the Department of Biological Responses, Institute for Virus Research, Kyoto University

2001- 2004 Human Stress Signal Research Center, BioMedical Special Research Unit, AIST at Kansai Head Joint Appointment

2003- 2008 Kyoto Univ, Translational Res Center, Thioredoxin Project, Group Leader

2003- Redox Bio Science Inc (Kyoto, Japan), Director CTO

2004- International Redox Network (IRN) , President

2010- Japan Biostress Research Promotion Alliance (JBPA), President

2010- Kyoto Univ, Prof.Em. IVR/LiMe

2011-2013 Invited Distinguished Professor in Ehwa Womans Univ. (WCU) Seoul.

2010 – Present Professor Em. Kyoto University, Chairman JBPA(Japan Biostress Research Promotion Alliance)



A systemic effects of herbal medicine on colon diseases

Mee-Hyun Lee

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Abstract

Traditional herbal medicine has shown many pharmacological activities such as anti-inflammations, antioxidant and anticancer. Each major component targets specific counterpart and mediates various signaling processes to regulate diseases. From the ancient period, Bi-Wi imbalance has been known to cause various diseases which is reflect to the microbiome imbalance in the present. Recently, the study of gut microbiome has getting attention cause of its important role in maintaining human health by immune system modulation, gut structural regulation and dietary nutrient metabolism. When the microbiome community profiles become to imbalance, it is caused to many kinds of diseases including, and inflammatory bowel disease, depression, arthritis, diabetes, obesity, atherosclerosis, non-alcoholic fatty liver disease and cancer. In the gut microbiome regulation, traditional herbal medicine can increase the beneficial gut microbial community profiles while decreasing the harmful microbial abundance. It is metabolized into active metabolites by the action of gut microbiota, restores the microbiome balances and enhance the fermentation products of the microbiomes, thus inhibits the gut microbiota dysbiosis-derived diseases. Taken together the traditional herbal medicine can be used for prevention and therapy in a disease by regulating the targets and gut microbiota balance. Therefore, the systemic regulation by traditional herbal medicine might be a new dimensional therapeutic strategy for disease treatment

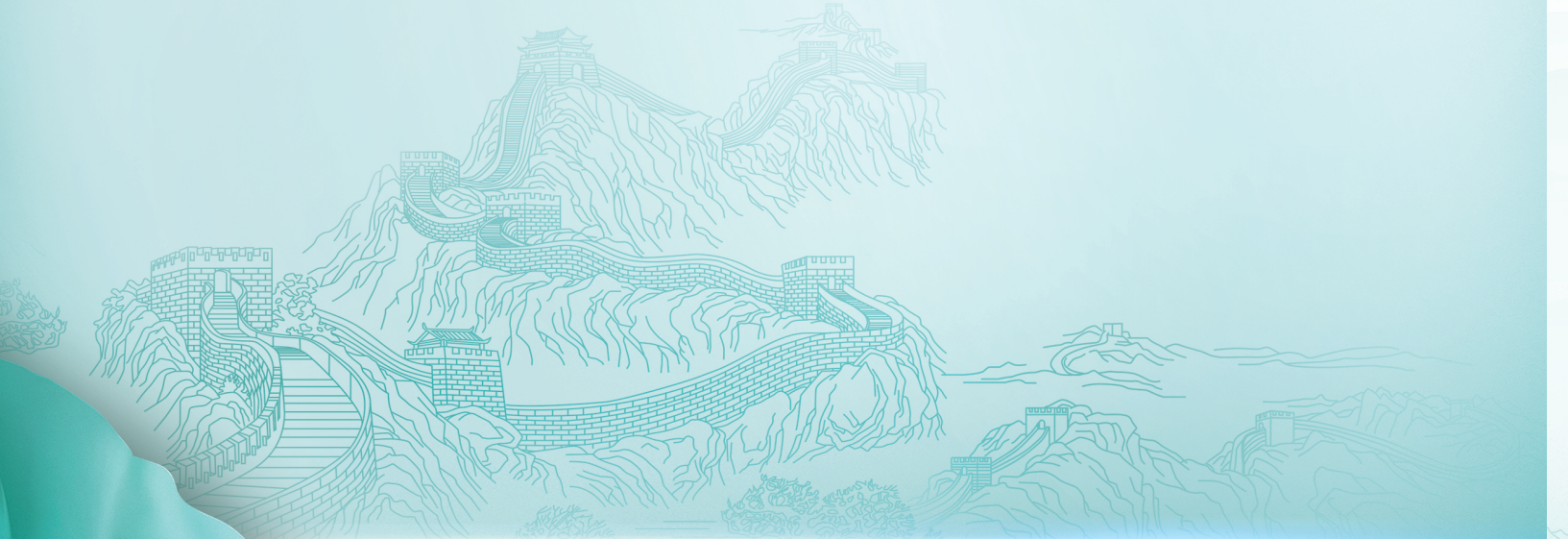
Key words: Traditional Herbal Medicine, Targeted therapy, Gut Microbiome Balance

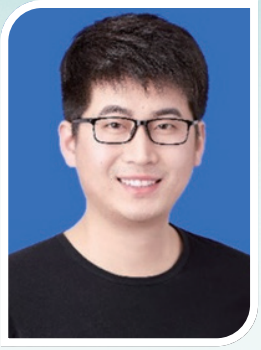
Short CV

Professor Lee is currently an Assistant Professor at Dongshin University and the Director of the Gut-Brain System Regulation Korean Medicine Research Center (GBRC) in South Korea. She received PhD from Seoul National University, where was supervised by Professor Young-Joon Surh. She also completed postdoctoral training under Professor Zigang Dong at The Hormel Institute, University of Minnesota, USA. Additionally, she has worked at the China-US (Henan) Hormel Cancer Institute in Zhengzhou, China. Her current research interests focus on investigating novel targets and natural compounds within cancer/carcinogenesis signaling networks, and the gut microbiome. (https://scholar.google.com/citations?user=IZy6bdEAAAAJ&hl=en)

Symposium-10(S10)

Redox and environmental challenge





Chair: Libo Du(杜立波)

Institute of Chemistry, Chinese Academy of Sciences, Beijing, China

Email: dulibo@iccas.ac.cn

Short CV

Dr. Libo Du is the associate professor of Institute of Chemisty, Chinese Academy of Sciences (CAS). He obtained his bachelor's degree from Shandong University of Technology in 2003 and M.D from Dalian University of Technology in 2006, and his Ph.D. from Institute of Chemistry, Chinese Academy of Science in 2009. Then, he joined Prof. Yang Liu's Lab as an assistant professor. In 2014, he became an associate professor of Institute of Chemistry, Chinese Academy of Science.

Dr. Du is involved in the design and synthesis of fluorescent probe that used for the monitoring the process of oxidative stress and reactive oxygen species. Furthermore, his team also focused on the nanodrug delivery system for the treatment of neurodegenerative diseases and skin disease. Recently, Dr. Du focused on the design and synthesis of peptides against skin anti-aging and anti-inflammatory.Dr. Du has published more than 80 peer-reviewed research articles and 12 authorized patents. Some patents have already been authorized for the use by the company.



Research progress and thinking of space medicine omics

Yinghui Li (李莹辉)

Astronaut Center of China; State Key Laboratory of Space Medicine, China

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Abstract

The completion of the China Space Station marks that Chinese strategic steps in manned spaceflight have moved from short-term flights to long-term in-orbit residence. By the end of 2022, China space station has entered the application development stage. China Space Laboratory has the capability to carry out systematic and large-scale space science research, providing a unique platform for space medicine and space life science to learn about life. The goal of the mission in the field of space medical experiments is to aim at the heights of the development of space medicine, breaking through the key technologies of space medicine, improve the ability of astronauts to provide medical support, healthy life and efficient work ability, solve the main medical problems of manned space flight, and provide theoretical support and technical reserves for the future manned moon landing and deep space exploration. Focusing on the physiological effects, mechanisms and protective technologies caused by space environment, multi-system physiological changes in cardiovascular, bone and muscle metabolism, glucose and lipid metabolism, visual function, nutrient metabolism, epigenetics and other related networks were systematically carried out. This paper introduces the latest research progress of China space station. Through system analysis of foundation simulation environment, isolation environment, pre-flight and post-flight DNA methylation, transcriptome, and phenomic data in ground simulation environment, space flight DNA methylation change characteristics, super baseline recovery phenomenon and its immune system in physiological system response, combined with the international dynamic, put forward the thinking and prospect of space medical research for long-term on-orbit operation of space station.

Short CV

Yinghui Li PhD, professor, director of State Key Laboratory of Space Medicine in China Astronaut Research and Training Center. Deputy Chief Designer of Astronautic system of China's manned spaceflight project. The member of international academy of astronautics life science. Vice president of Chinese Society of Space Science and the chairman of Space Life Specialized Committee. Focus on the mechanism and application research of microgravity physiology and countermeasure, the space experiment technology of space medicine. He presided over the establishment of the state Key Laboratory of Space Medicine, which has become an important technical support platform for space station missions. She is responsible for the organization and implementation of space medicine experiment projects, and systematically conduct the occurrence and development mechanism of space medical problems, published nearly 200 SCI papers. She has made outstanding contributions to the construction of China's space medical discipline.



Gγ regulates PIP2 phosphorylation in ROS distribution to affect crop tolerant to alkaline stress

Qi Xie (谢旗)

Institute of Genetics and Developmental Biology, Chinese Academy of Sciences, China

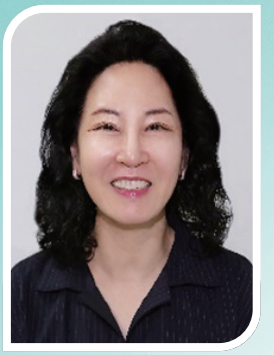
Email: qxie@genetics.ac.cn

Abstract

A scarcity of knowledge and breeding efforts for plant alkaline tolerance hinder the usage of alkaline-salt lands for crop production. Through genome association analysis of sorghum, a natural high-alkaline-tolerant crop, we detected a major locus, Alkaline Tolerance 1 (AT1), specifically related to alkaline-salinity sensitivity. An at1 allele with a C-terminal truncation increased sensitivity, while knockout of AT1 increased tolerance to alkalinity in sorghum, millet, rice and maize. AT1 encodes an atypical G protein γ subunit that affects the phosphorylation of aquaporins to modulate the distribution of H_2O_2. Several key factors are involved in the phosphorylation of aquaporins. These processes appear to protect plants against oxidative stress by alkali. Designing knockouts of AT1 homologues or selecting its natural nonfunctional alleles could improve crop productivity in sodic lands.

Short CV

Prof. Qi Xie, investigator of the Institute of Genetics and Developmental Biology at the Chinese Academy of Sciences. Focusing on the molecular mechanism of plant stress biology, while also working on sustainable agriculture, especially how to use saline-alkaline land for crop production.



Particulate matter and reactive oxygen species

Jin Won Hyun

Department of Biochemistry, College of Medicine and Jeju Natural Medicine Research Center, Jeju National University, Korea

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Abstract

Our previous studies showed that particulate matter 2.5 (PM2.5)-derived reactive oxygen species (ROS) caused skin cell damage, cellular senescence, and inflammation. Therefore, we sought to elucidate the mechanisms of how PM2.5-derived ROS are generated. The present studies demonstrated that PM2.5-drived ROS production in human keratinocytes was mediated via the NADPH oxidase (NOXs) system and the calcium signaling pathway. PM2.5 increased the expression of NOX1, NOX4, and a calcium-sensitive NOX, dual oxidase 1 (DUOX1). PM2.5 bound to aryl hydrocarbon receptor (AhR), and this complex bound to XRE sequences of NOX1 and DUOX1 promoter regions, suggesting that AhR acted as a transcription factor of NOX1 and DUOX1. PM2.5 increased the DUOX1 transcription through epigenetic modification in terms of DNA methylation and histone modification. A link between DNA demethylase and histone methyltransferase in DUOX1 promoter elevated the expression of DUOX1 mRNA. The increase in intracellular calcium level activated DUOX1, responsible for ROS production. Our findings provide evidence for a PM2.5-mediated ROS-generating system network, in which increased expression of NOX system serves as a ROS signal through AhR and calcium activation (RS-2023-00270936). <Kang KA, et al. Environ Pollut. 2024;347:123675.>.

Key words: Particulate Matter, Reactive Oxygen Species, NADPH oxidase

Short CV

2002-present	Professor	Jeju National University School of Medicine
2023-present	Center Director	Jeju Research Center for Natural Medicine of Core Research Institute (NRF)
2017-2020	BRL Director	Basic Research Laboratory (NRF)
2012-2014	Vice-Dean	Vice-Dean of Academic Affairs in Jeju National University
2005-2014	Research Director	Jeju Regional Cancer Center, Jeju National University Hospital



TCDD-induced lysosomal SLC46A3 modulates hepatic cytosolic copper homeostasis resulting in triglyceride accumulation

Jung-Hwan Kim

Department of Pharmacology, School of Medicine, Institute of Health Sciences, Department of Convergence Medical Science, Gyeongsang National University, Jinju, 52727, Korea

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Abstract

The environmental contaminant 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) causes hepatic toxicity associated with prominent lipid accumulation and oxidative stress. Here, the authors report that the lysosomal copper transporter SLC46A3 is induced by TCDD and underlies the hepatic lipid accumulation in mice, potentially via effects on mitochondrial function. SLC46A3 was localized to the lysosome where it modulated intracellular copper levels. Forced expression of hepatic SLC46A3 resulted in decreased mitochondrial membrane potential and abnormal mitochondria morphology consistent with lower copper levels. SLC46A3 expression increased hepatic lipid accumulation similar to the known effects of TCDD exposure in mice and humans. The TCDD-induced hepatic triglyceride accumulation was significantly decreased in Slc46a3 − / − mice and was more pronounced when these mice were fed a high-fat diet, as compared to wild-type mice. These data are consistent with a model where lysosomal SLC46A3 induction by TCDD leads to cytosolic copper deficiency resulting in mitochondrial dysfunction leading to lower lipid catabolism, thus linking copper status to mitochondrial function, lipid metabolism, and TCDD-induced liver toxicity. <Kim JH et al, Nature Commun, 2021;12:290.>

Key words: slc46a3, tcdd, copper, mitochondria, fatty liver

Short CV

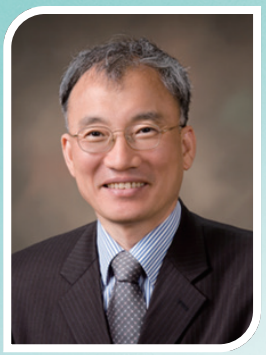
2024- present: Professor, Dept of Pharmacology, School of Medicine, Gyeongsang National. University, Republic of Korea

2018- 2024: Associate professor, Dept of Pharmacology, School of Medicine, Gyeongsang National. University, Republic of Korea

2014- 2018: Assistant professor, Dept of Pharmacology, School of Medicine, Gyeongsang. National University, Republic of Korea

2009-2014: Post-doctoral fellow in NCI/NIH, Bethesda, MD

2003- 2009: Ph.D, Pharmaceutical Science at the College of Pharmacy, Rutgers University, New. Jersey, USA



Redox history of earth, and life of organisms and foods crops

Dae Young Kwon

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Abstract

The history of the earth is largely based on the redox potential of hydrogen and oxygen. When the earth broke away from an asteroid 4.5 billion years ago (BYA), it was in the form of a magma sea, surrounded by high-temperature and high-pressure water vapor and carbon dioxide. At 3.8 billion years old, as the high temperature fell, the water vapor became water to form an ocean without any oxygen. As the sea was formed, the cyanobacteria (photosynthetic bacteria) emerged and absorbed carbon dioxide to form limestone and oxygem in 3.0 to 3.5 BYA, resulting redox potential increased high during 2.5 to 3.0 BYA, and resulted into mass extinction of 99% of existing life at 2.4 to 2.0 BYA. Subsequent evolution of Eukaryotes by dominating the photosynthetic solar driven and oxygen-based earth system in 1 BYA. Thus plants and animal were apeared in earth in 0.5 BYA. Extinction of large animals such as dinosaurs are by asteroid collision at 65 million years ago (MYA) that allowed to mammals emerge to dominate photosynthetic and oxygen rich world to hominids 1-4 MYA and Homo sapiens 60,000 to 250,000 years ago. In order for the Earth to create a sustainable environment in the future along with climate change, we need to know about these Earth's oxido-reduction ecological systems. Similarly, humans use these redox-potential to maintain the health human body. The mission is how we can reduce oxidative stress in human biological systems. For this purpose, along with the evolution of life on the Earth, human can reduce redox potential by foods. Plants and food crops produce antioxidant materials in themselves that remove many free radicals. Individual human health also becomes a problem when the redox potential changes due to the environment, fatigue, and disease. It is important how our body does something to lower the redox potential which goes up. First of all, it is important to relax and eat for resilience of body.

Key words: Redox story of earth, cyanobacteria, photosynthetic plants, Biology, Reactive Oxygen Species.

Short CV

DYK is a fellow of the Korean Academy of Science/Technology and was professor at Hoseo Univ. He acted as President of Korea Food Research Institute, where he worked almost 35 yr. He obtained his BS at Seoul National Univ with Food Science, and he received his MS and PhD from KAIST with Biological Science. He joined Whitehead Institute, MIT, USA as a post-doctoral fellow. He is now working as President of Institute of Food Science and Culture. He is also working as Editor in Chief of J. Ethnic Foods, by Springer-Nature.

Symposium-11(S11)

Traditional Medicine Prophylaxis-Therapeutics and Redox Balance





Chair: Simon Ming-Yuen Lee (李铭源)

The Hong Kong Polytechnic University, Hongkong, China

Email: simon-my.lee@polyu.edu.hk

Short CV

Simon Ming Yuen LEE is currently Chair Professor of Biomedical Sciences in Department of Food Science and Nutrition, and State Key Laboratory of Chemical Biology and Drug Discovery, The Hong Kong Polytechnic University. Simon obtained PhD degree in biochemistry from The Chinese University of Hong Kong. His research interests lie in the discovery of drug-like agents from natural products including small molecules and biologics for use in various therapeutic areas, including brain disorder and neurodegenerative diseases. His dedication to education and research in the fields of omics, pharmacology and toxicology has leaded to over 360 scholarly articles, including Nature, Nature Genetics, Nature Communications (4×) and Science Advances. Simon is in Stanford university's list of top 2% of most-cited scientists in Pharmaceutical Science and Biology (with h-index: 65 from Scopus). Simon is a life member of Clare Hall, University of Cambridge. He has served as an editorial board member for numerous international journals including Chinese Medicine, Antioxidants, Journal of Ethnopharmacology, and Water Biology and Security.



Traditional Chinese medicine ameliorates cardiac and cerebral microvascular injury through regulating mitochondrial respiratory chain

Jing-Yan Han (韩晶岩)

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Abstract

Cardiac microvascular effusion after myocardial infarction vascular recanalization and cerebral infarction vascular recanalization are both unsolved clinical problems. The method of supplementing plasma albumin and diuresis based on the theory of osmotic pressure and tissue pressure is not clinically effective. The clinical effect of traditional Chinese medicine in the treatment of cardiovascular and cerebrovascular exudation is obvious, but the mechanism of action of qi replenishment and intake is not clear.

The team of the presenters used a rat cardiac microvascular exudation model established by cardiac ischemia and reperfusion to confirm that Qishen Yiqi Dripping Pill, a compound Chinese medicine that replenishes Qi and invigorates blood, can inhibit cardiac microvascular exudation in rats caused by ischemia and reperfusion, which is related to improving the energy metabolism of vascular endothelial cells and inhibiting the opening of vascular endothelial cell gap links.

The reporter's team also used a mouse cerebral microvascular exudation model established by tPA thrombolysis after middle cerebral artery cerebral infarction to confirm that Qi Replenishing and Activating Blood Compound Chinese Medicine Qishen Yiqi Dripping Pill can inhibit cerebral microvascular exudation and hemorrhage in mice after tPA thrombolysis after middle cerebral artery cerebral infarction, which is related to improving the mitochondrial respiratory chain of vascular endothelial cells, improving energy metabolism, and inhibiting the opening of vascular endothelial cell gap links. , inhibition of vascular basement membrane injury.

In this report, we will introduce the mechanism of action of Qi Replenishing and Invigorating Blood Compound Chinese Medicine to improve the mitochondrial respiratory chain and improve the exudation of cardiovascular and cerebral microvessels.

Short CV

Professor Jing-Yan Han graduated from Liaoning University of traditional Chinese medicine in 1982. He obtained his doctor degree in the department of internal medicine at the Keio University School of Medicine in Japan in 2002. In 2004, he became the Director of the Tasly microcirculation research center at the Peking University Health Science Center. In 2008, he was appointed as the Chair and Professor of the department of integration of Chinese and Western medicine at the School of Basic Medical Sciences of Peking University. In 2009, he served as a visiting professor in the department of internal medicine at the Keio University School of Medicine. In 2010, he became the chair of the department of integration of Chinese and Western medicine at the Peking University Health Science Center. In 2019, he was appointed as the Dean of Academy of Integration of Chinese and Western Medicine, Peking University Health Science Center.



Niuhuang Qingxin Wan ameliorates depressive-like behaviors and improves hippocampal neurogenesis through modulating TrkB/ERK/CREB signaling pathway in chronic restraint stress or corticosterone challenge mice

Jiangang Shen

School of Chinese Medicine, University of Hong Kong, 3 Sassoon Road, Hong Kong, Hong Kong SAR, China.

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Introduction: Chronic stress-associated hormonal imbalance impairs hippocampal neurogenesis, contributing to depressive and anxiety behaviors. Targeting neurogenesis is thus a promising antidepressant therapeutic strategy. Niuhuang Qingxin Wan (NHQXW) is an herbal formula for mental disorders in Traditional Chinese Medicine (TCM) practice, but its anti-depressant efficacies and mechanisms remain unverified.

Methods: In the present study, we tested the hypothesis that NHQXW could ameliorate depressive-like behaviors and improve hippocampal neurogenesis by modulating the TrkB/ERK/CREB signaling pathway by utilizing two depression mouse models including a chronic restraint stress (CRS) mouse model and a chronic corticosterone (CORT) stress (CCS) induced mouse model. The depression-like mouse models were orally treated with NHQXW whereas fluoxetine was used as the positive control group. We evaluated the effects of NHQXW on depressive- and anxiety-like behaviors and determined the effects of NHQXW on inducing hippocampal neurogenesis.

Results: NHQXW treatment significantly ameliorated depressive-like behaviors in those chronic stress mouse models. NHQXW significantly improved hippocampal neurogenesis in the CRS mice and CCS mice. The potential neurogenic mechanism of NHQXW was identified by regulating the expression levels of BDNF, TrkB, p-ERK (T202/T204), p-MEK1/2 (S217/221), and p-CREB (S133) in the hippocampus area of the CCS mice. NHQXW revealed its antidepressant and neurogenic effects that were similar to fluoxetine. Moreover, NHQXW treatment revealed long-term effects on preventing withdrawal-associated rebound symptoms in the CCS mice. Furthermore, in a bioactivity-guided quality control study, liquiritin was identified as one of the bioactive compounds of NHQXW with the bioactivities of neurogenesis-promoting effects.

Conclusion: NHQXW could be a promising TCM formula to attenuate depressive- and anxiety-like behaviors against chronic stress and depression. The underlying anti-depressant mechanisms could be correlated with its neurogenic activities by stimulating the TrkB/ERK/CREB signaling pathway.



Qiliqiangxin in the treatment of heart failure with reduced ejection fraction ------ research progress

Xinli Li (李新立)

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Abstract

The latest findings from the QUEST study were published in Nature Medicine. This study evaluated the efficacy and safety of Qiliqiangxin capsules (QLQX), a traditional Chinese medicine, in treating heart failure with reduced ejection fraction (HFrEF). The results marked a significant achievement, highlighting the role of Traditional Chinese Medicine (TCM) in global cardiovascular treatment.

The QUEST study, a large-scale, multicenter, randomized controlled trial involving 3,110 HFrEF patients across 133 clinical centers, demonstrated that adding QLQX to standard treatments reduced major adverse cardiovascular events by 22%, heart failure hospitalizations by 24%, and cardiovascular deaths by 17%. Importantly, QLQX did not significantly increase adverse events, confirming its safety profile.

The systemic mechanisms suggest that QLQX may work by attenuating oxidative stress and modulating the PPARγ/PGC-1α signaling pathway. This groundbreaking research offers a promising future for integrating TCM with modern medical practices in heart failure treatment.

The study not only underscores the clinical value of QLQX but also sets a precedent for the internationalization and standardization of TCM, providing a pathway for future innovations in global healthcare.

Key words: Qiliqiangxin, Heart failure, Traditional Medicine, PPARγ/PGC-1α

Short CV

Prof. Xinli Li, Chief Cardiologist, Postgraduate & Doctorate Supervisor from State Key Laboratory for Innovation and Transformation of Luobing Theory, the First Affiliated Hospital of Nanjing Medical University, Nanjing, China.

Chinese special contribution expert of the state council, and Chief cardiologist and second-level professor of the First Affiliated Hospital of Nanjing Medical University.

Academic appointment in various academic groups including: Vice President of the Cardiovascular Branch of the Chinese Society of Gerontology, Vice Chairman of the Heart Failure Group of the Cardiovascular Branch of the Chinese Medical Doctor Association, Vice Chairman of the Hypertension Group of the Chinese Society of Cardiovascular Diseases, Vice Chairman of the Collateral Disease Branch of the Chinese Society of Chinese Medicine, Vice Chairman of the Precision Medicine Branch of the Chinese Society of Thoracic Cardiovascular Anesthesiology, etc.

Dedicated in the basic and clinical research on cardiology for decades; As the corresponding author, published more than 300 articles in various esteemed journal (Nature Medicine, JACC, Circulation, etc.); Supervised for over 100 post-graduate and doctorate students.



Cardiac stress resistance regulated by sulfur metabolism

Motohiro Nishida

Graduate School of Pharmaceutical Sciences, Kysuhu University, Japan

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Abstract

Sulfur-based redox signaling has long been attracted attention as critical mechanisms underlying the development of cardiac diseases and resultant heart failure. Especially, post-translational modifications of cysteine (Cys) thiols in proteins mediate oxidative stress-dependent cardiac remodeling including myocardial hypertrophy, senescence, and interstitial fibrosis. However, we recently revealed the existence of Cys persulfides and Cys polysulfides in cells and tissues, and these catenated sulfur molecules (supersulfide) substantially contribute to redox signaling and energy metabolism by exerting unique redox dynamics. We have established simple evaluation methods that can detect polysulfides in proteins and inorganic polysulfides in cells. We found that polysulfides in healthy hearts are dramatically catabolized by exposure to ischemic/hypoxic and environmental electrophilic stress, leading to vulnerability of the heart to mechanical load. Accumulation of hydrogen sulfide, a nucleophilic catabolite of persulfides/polysulfides, is well associated with myocardial remodeling, and perturbation of polysulfide catabolism can improve myocardial remodeling and dysfunctions after myocardial infarction in mice. These results suggest that prevention of supersulfide catabolism during ischemic/hypoxic stress becomes a new therapeutic strategy for the treatment of chronic heart failure.

Key words: Sulfur, heart, robustness, redox signaling, metabolism

Short CV

1996-2001 Ph. D., Graduate School of Pharmaceutical Sciences, University of Tokyo, Japan

2001.4-2001.5 JSPS Research Fellow, University of Tokyo, Japan

2001.5-2003.9 Assistant Professor, Okazaki Institute for Integrative Bioscience, Japan

2003.10-2006.7 Lecturer, Graduate School of Pharmaceutical Sciences, Kyushu University, Japan

2006.8-2013.7 Associate Professor, Graduate School of Pharmaceutical Sciences, Kyushu University, Japan

2013.8- Professor, National Institute for Physiological Sciences (NIPS) & Exploratory Research Center on Life and Living Systems (ExCELLS), National Institutes of Natural Sciences, Aichi, Japan

2013.10-2017.3 Research Fellow, JST, PRESTO, Japan

2020.4- Professor (80%), Graduate School of Pharmaceutical Sciences, Kyushu University, Fukuoka, Japan

Cross Appointment Professor (20%), NIPS&ExCELLS, NINS, Japan



The role of oxidative stress and antioxidants in liver disease therapy

Yibin Feng（冯奕斌）
The University of Hong Kong, China
Email: yfeng@hku.hk

Abstract

Over 10% of the world population is affected by various kinds of liver diseases. Liver diseases including acute virus hepatitis, chronic virus hepatitis, alcoholic and non-alcoholic fatty liver, liver fibrosis, cirrhosis and liver cancer etc., are serious health-threaten problems worldwide, especially in Asian region. In addition to various etiologic facts, base of liver diseases are inflammatory diseases and associate with oxidative stress. In fact, oxidative stress plays an important role in chain of liver diseases. Various pathological factors damaged liver tissues by increase reactive oxygen (ROS), nitrogen species (RNS) and peroxidation of lipids, DNA, and proteins. ROS affects fibrogenesis via increasing platelet-derived growth factor and ROS/RNS provokes hepatic stellate cells, which are presented by the enhanced production of extracellular matrix and accelerated proliferation, and most hepatocellular carcinomas occur in fibrotic and cirrhotic livers. There are complicated cross-talk among pathological factors, inflammatory, free radicals and immune responses. Finding network and mechanisms linking these relationship and process will play a major role in pathogenesis of liver diseases. It is still far to reach it, but targeting any specific etiological or pathological factor should be one of reasonable therapeutic strategy in liver diseases. For this purpose, antioxidative therapy maybe become a basic treatment for various liver diseases. Based on literature review and our original studies, I will review oxidative stress and relationship among other etiological and pathological factors in liver diseases, and then explore possibility of antioxidative therapy for liver diseases.

Key words: inflammatory, oxidative stress, antioxidants, liver disease, therapy

Short CV

Dr. Feng Yibin is currently a professor and Director of the School of Chinese Medicine, University of Hong Kong. Mainly focus on integrative Chinese and Western medicine in the prevention and treatment of cancer and metabolic diseases. He has published more than 600 various kind of publications, among which over 200 SCI papers in reputable international journals. According to InCites Essential Science Indicators (ESI), Dr. Feng has been listed as one of the top 1% of scholars in the world at the University of Hong Kong for 8 consecutive years, and is also listed as one of top 2% Scientists Worldwide by Stanford University. He contributes to academic community as the chairperson at the international conferences and has given keynote, plenary/ invited speeches over 200 around the world. He is the editor of several international journals in his field and reviewer for lots of international journals.

Symposium-12(S12)

Natural products and nutrition in anti-aging and health management





Chair: Bo Zhou (周波)

State Key Laboratory of Applied Organic Chemistry, Lanzhou University, China

Email: bozhou@lzu.edu.cn

Short CV

Bo Zhou obtained his BS degree in Chemistry from Gannan Normal University in China in 1993, followed by MS and PhD degrees in Organic Chemistry from Lanzhou University in 1997 and 2000, respectively. After completing his postdoctoral work at Life Science College and State Key Laboratory of Applied Organic Chemistry, Lanzhou University, he joined the college of chemistry and chemical engineering at Lanzhou University. In 2004, he was promoted to the position of professor. He received support from the Program for New Century Excellent Talents in University in 2006, and selected as a member of the Executive Committee for SFRBM of China in 2018.

His research interests primarily focus on developing pro-oxidative anticancer agents inspired by natural products to target abnormal redox homeostasis within cancer cells. Additionally, he is involved in designing fluorescent probes for monitoring intracellular redox-active molecules. He has published over 120 scientific papers across various international journals including J. Am. Chem. Soc., Anal. Chem., Free Radic. Biol. Med., Sens. Actuators B Chem., Chem. Eur. J., Antioxid. Redox Signaling, J. Org. Chem., with an H-index of 42.



Chair: Ae-Son Om

Hanyang University, Korea

Email: aesonom@hanyang.ac.kr

Short CV

EDUCATIONAL BACKGROUND

1996.09 - 1999.06 Michigan State University Department of Food Science & Human Nutrition Ph.D.
1993.08 - 1996.08 University of Oklahoma Department of Anatomy & Cell Biology Ph.D.
1988.03 - 1991.08 Hanyang University Department of Food and Nutrition Ph.D.
1983.09 - 1985.08 Hanyang University Department of Food and Nutrition M.S.
1979.03 - 1983.03 Hanyang University Department of Food and Nutrition B.A.

AWARDS AND HONORS

2016.12 Appreciation Award from Army Chief of Staff, Ministry of National Defense
2013.05 12th Food Safety Day Diligence Award, Ministry of Food and Drug Safety
2013.04 Commendation Award from Minister, Ministry of Health & Welfare

POSITIONS HELD TO DATE

2023.02 - Now Member of the Agriculture, Fisheries and Food Subcommittee of the Presidential Committee on Agriculture
2021.04 - Now Non-executive Director of Korea Food Safety Management Certification Institute (HACCP)
2021.03 - Now Chairman of the Agricultural and Fishery Products Hygiene and Safety Ingredients Division of the Ministry of Agriculture, Food and Rural Affairs
2021.01 - 2023.01 Non-executive Director of Korea National Cluster
2021.01 - 2021.12 Vice President, Korean Society of Food Science and Technology
2019.01 - 2021.01 Prime Minister's Office Food Safety Policy Committee
2019 - 2021 Vice Chairman of the Health Functional Food Advertisement Review Committee
2014.06 - 2022.12 Chief of Center for Children's Foodservice Management
2014.02 - Now President in the 11th Hanyang University HACCP education and training institution appointed by the Ministry of Food and Drug Safety
2008.01 - 2013.01 President in The Korean Society of Food Service Sanitation
2006 - 2009 Member of the Health Functional Food Advertisement Review Committee

PUBLICATIONS

Food Hygiene (Revised Edition) (Text Book), 2022
The impact of COVID-19 on older persons in Republic of Korea, 2020
Life Planning series 2, 2016
54 Korean Dietary Ingredients Against Cancer, 2013
Science of A Cup of Milk, 2010
Food Microbiology (Text Book), 2009
Current Food Sanitation (Text Book), 2005

International academic journal activities

2022.07 - 2023.03 Editor of Journal of Fungi
2021.03 - 2022.06 Editor of Toxins

RESEARCH PROJECTS

■ Hanyang University Food Tech Department Operation

2020 - Now Department Head, Department of Food Tech Functional Food, Hanyang University: Education for employees and students of about 40 small and medium-sized companies)

■ Industrial technical guidance/consultation (HACCP, expiration date setting experiment, etc.)

2022 - Now NST Bio, Our Bio Consulting

2019 - 2020 Medipresso, Shinan Tourism Co., Ltd., Doojin Food, Vitamin Tree HACCPConsulting (HACCP Certification Success)

2018 - 2018 Mac Bakery Co., Ltd., Hotel Rivera HACCP Consulting (Successful HACCP Certification)

2017 - 2018 Gwangjin Food Co., Ltd., International Comprehensive Consulting, Ubok-dong, Jinheung Agricultural Products, Hanul Comprehensive Food, Hana Architects & Engineers Co., Ltd. HACCP Consulting (HACCP Certification Success)

■ Military food service consulting and research and development

Combat Rations Research

2017 - 2022: L-type distribution period setting and ILS test

2016 - 2016: Combat ration utilization and stockpiling feasibility study of outdoor food

2015 - 2016: Preliminary research service for development of L-type personal combat rations

■ Hygiene and safety education (hygiene education and consulting for students, civil servants, companies, group meals, military meals, food processing companies, etc.)

2018 - 2018 Seoul Metropolitan City School Health Promotion Center Education: Specialized training for school meal hygiene and safety inspection personnel

2017 - 2017 Seoul Metropolitan Office of Education: Specialized training for school meal hygiene and safety inspection personnel

LIST OF JOURNALS AND PAPERS (10 Representative Papers)

1. Hong, J. Y., Kim, Y. M., Shin, M. H., Lee, Y. H., Om, A. S., & Kim, M. K. (2022). Development and validation of dietary atherogenic index using common carotid artery-intima-media thickness: A food frequency questionnaire-based longitudinal study in Korean adults. Nutrition Research, 104, 55-65.

2. Wei, X., Du, M., Hong, S. Y., & Om, A. S. (2022). Degradation of Patulin in Pear Juice and Apple Juice by Ascorbic Acid and the Combination of Ascorbic Acid and Ferrous Iron. Toxins, 14(11), 737.

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4. Sun, X., Xuan, X., Ji, L., Chen, S., Liu, J., Zhao, S., ... & Om, A. S. (2021). A novel continuous hydrodynamic cavitation technology for the inactivation of pathogens in milk. Ultrasonics sonochemistry, 71, 105382.

5. Choi, Y., Kim, D. S., Lee, M. C., Park, S., Lee, J. W., & Om, A. S. (2021). Effects of bacillus subtilis-fermented white sword bean extract on adipogenesis and lipolysis of 3T3-L1 adipocytes. Foods, 10(6), 1423.

6. Lee, M. C., Puthumana, J., Lee, S. H., Kang, H. M., Park, J. C., Jeong, C. B., ... & Lee, J. S. (2016). BDE-47 induces oxidative stress, activates MAPK signaling pathway, and elevates de novo lipogenesis in the copepod Paracyclopina nana. Aquatic Toxicology, 181, 104-112.

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10. Om, A. S., & Shim, J. Y. (2007). Effect of daidzein in rats on cadmium excretion. Bulletin of environmental contamination and toxicology, 78, 485-488.



Research on space biological rhythm intervention strategies based on redox regulation

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Abstract

During space exploration, astronauts expose consistently or intermittently to various environmental factors, including weightlessness, radiation, noise, isolation and confinement. All these factors combine to exert stress responses on many aspects of physiological systems, eliciting influence on astronaut's health. Long-term spaceflight is known to induce disruption in circadian rhythm and cognitive disturbance, but the underlying molecular mechanism remain unclear. More evidences have shown change of redox status and elicited increased oxidative stress during space flight.

Circadian rhythm is a widespread physiological phenomenon which exists in almost all of the life forms and comprises a system of interconnected transcriptional and translational feedback loops. It has been known that the oxidative stress involved in many diseases and injuries like aging, Alzheimer disease, diabetes and cancers. There have been growing literatures about the biological rhythm disorder caused by oxidative stress, but little is known about the effect of oxidants on circadian rhythm intervention and the potential mechanism.

In our research, we have found that H_2O_2 could modulate the circadian rhythm of Bmal1 via RORα, REV-ERBα (NR1D1) and REV-ERBβ (NR1D2). N-Acetyl cysteine could reverse the H_2O_2 induced up-regulation of CLOCK and BMAL1, period shorten and amplitude elevation of Bmal1-luciferase, providing evidences that the non-transcriptional oscillation may interplay with the transcriptional/translational feedback loops. In simulated microgravity and isolation condition, transcription factor Nrf2 might regulate biological rhythm by regulating the expression of the CLOCK, and mitophagy exerts circadian control by regulating NR1D1 degradation. In space simulated environment, rats were treated with urolithin A, a mitophagy activator derived from pomegranate nuts and berries, reversed the disruption of central body temperature, heart rate and activity rhythms by increasing mitophagy to decay NR1D1 and improving mitochondrial function. The activation of mitophagy holds great promise as a therapeutic strategy for long-term spaceflight as well as diseases with circadian disruption.

Key words: Space flight, Biological rhythm, Redox regulation, Oxidative stress

Short CV

Professor, State Key Laboratory of Space Medicine, China Astronaut Research and Training Center; Major in research of space medicine fundamental and application, especially on redox regulation in space medicine.



Phosphatidylethanolamine alleviates OX-LDL-induced macrophage inflammation by upregulating autophagy and inhibiting NLRP1 inflammasome activation

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Abstract

Oxidized low-density lipoprotein (OX-LDL)-induced inflammation and autophagy dysregulation are important events in the progression of atherosclerosis. Phosphatidylethanolamine (PE), a multifunctional phospholipid that is enriched in cells, has been proven to be directly involved in autophagy which is closely associated with inflammation. However, whether PE can influence OX-LDL-induced autophagy dysregulation and inflammation has not been reported. In the present study, we revealed that OX-LDL significantly induced macrophage inflammation through the CD36-NLRP1-caspase-1 signaling pathway in fish. Meanwhile, cellular PE levels were significantly decreased in response to OX-LDL induction. Based on the relationship between PE and autophagy, we then examined the effect of PE supplementation on OX-LDL-mediated autophagy impairment and inflammation induction in macrophages. As expected, exogenous PE restored impaired autophagy and alleviated inflammation in OX-LDL-stimulated cells. Notably, autophagy inhibitors reversed the inhibitory effect of PE on OX-LDL-induced maturation of IL-1β, indicating that the regulation of PE on OX-LDL-induced inflammation is dependent on autophagy. Furthermore, the positive effect of PE on OX-LDL-induced inflammation was relatively conserved in mouse and fish macrophages. In conclusion, we elucidated the role of the CD36-NLRP1-caspase-1 signaling pathway in OX-LDL-induced inflammation in fish and revealed for the first time that altering PE abundance in OX-LDL-treated cells could alleviate inflammasome-mediated inflammation by inducing autophagy. Given the relationship between OX-LDL-induced inflammation and atherosclerosis, this study prompts that the use of PE-rich foods promises to be a new strategy for atherosclerosis treatment in vertebrates.

Key words: Oxidized low-density lipoprotein, Macrophages, NLRP1 inflammasomes, IL-1β, Phosphatidylethanolamine, Autophagy

Short CV

Professor of Ocean University of China (OUC), Dean of Fisheries College, OUC. Distinguished professor of Changjiang Scholars, the recipient of the National Outstanding Youth Science Foundation of China and a national young and middle-aged science and technology innovation leader. He is mainly engaged in the research of lipid nutrition and immune metabolism in fish, and has explored the lipid metabolism, inflammatory response and its regulatory mechanism in marine fish, and proposed corresponding mitigation strategies, which has promoted the development of aquatic animal nutrition and the healthy and sustainable development of aquatic feed industry. The research results have been published in Progress in Lipid Research, Reviews in Aquaculture, Cell Death and Disease, Free Radical Biology and Medicine, iScience, FASEB and other journals, with more than 200 papers. Meanwhile, he has also obtained 28 national and international patents.



Translation of bilirubin's redox potential to preventative and therapeutic medicine – use of models, and the development of therapies

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Abstract

Bilirubin's antioxidant effects are well established, and protects against various pathologies underpinned by oxidative stress. Despite this, understanding whether bilirubin can prevent and/or treat pathologies that induce oxidative stress in humans remains unknown. A number of animal models are available to test these effects, however, very few induce physiologically relevant bilirubin concentrations, questioning the relevance of findings. Interestingly, the unique human model of Gilbert's syndrome provides a relevant model to test bilirubin's potential protective effects, however, can only provide correlative data. Therefore, our group developed a titratable inducible hyperbilirubinaemic mouse model, to test the potential beneficial effects of bilirubin, addressing issues relating to bilirubin concentration and providing a means to test preventative and therapeutic effects. This presentation will also discuss potential therapeutic strategies in humans and identify conditions that bilirubin is most likely protective against, based upon currently published literature. It is hoped that this presentation will stimulate research in the area, particularly concerning the development of therapeutics that can increase bilirubin, for prevention and treatment of age related pathologies.

Key words: Bilirubin, Haem Oxygenase, UGT1A1, cardiovascular disease, diabetes, animal model, Gilbert's Syndrome

Short CV

Professor Bulmer is a research intensive academic within the School of Pharmacy and Medical Sciences at Griffith University on the Gold Coast, Australia. Dr Bulmer has more than 20 years of experience in biochemistry, haematology and cardiovascular imaging, including their use in a variety of in vitro, *in vivo* small animal, pre-clinical human and human clinical trial research settings. Using this experience, his Experimental Laboratory Science (XLabS) research group aims to better understand and prevent the effects of vascular injury within the arterial and venous circulation. Dr Bulmer's research has two foci, the first includes demonstrating the protective effects of haem catabolism and bilirubin formation within inflammatory pathology, particularly in atherosclerosis. Secondly, his group aims to reduce vasculitis and thrombotic events in patients enduring invasive vascular access devices/procedures with the AVATAR group (https://www.avatargroup.org.au/).



A catechol isoquinoline salsolinol induces apoptosis of human liver cancer cells by regulating the STAT1/3 Signaling

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Abstract

Liver cancer is one of the most common malignancies and a leading cause of death worldwide. However, it is still very difficult to treat and prevent liver cancer. A catechol tetrahydroisoquinoline, salsolinol (SAL) is present in our daily diets, such as mushrooms, bananas, etc. It is also endogenously generated by the condensation of dopamine with acetaldehyde. In the present study, we found that SAL inhibited the growth and colony forming ability of human hepatic carcinoma SK-Hep1 cells. The phosphorylation at Tyr 705 of signal transducer and activator of transcription factor 3 (STAT3) and its dimerization, nuclear translocation, and transcriptional activity were inhibited by SAL. The expression of cyclin D1, the major target proteins of STAT3, was suppressed, whereas the expression of cell cycle regulator p21 and its upstream regulator p53 was enhanced by SAL. p53 regulates expression of genes involved in apoptosis. SAL induced intrinsic apoptotic signaling by enhancing Bax expression and proteolytic cleavage of caspase-9/3/7 and PARP. The proportions of cell population in the subG0/G1 fraction and TUNEL positive apoptotic cells were increased by SAL. SAL inhibited the mitochondrial STAT3 phosphorylation (Ser727) and induced disruption of mitochondria membrane potential, which led to the downregulation of cytochrome c in the mitochondria fraction. A general antioxidant N-acetyl cysteine (NAC) attenuated suppression of STAT3 at Tyr and Ser residues and blocked the phosphorylation of STAT1 (Tyr 701) and cell death as well as generation of reactive oxygen species induced by SAL. Moreover, SAL inhibited direct interaction between Annexin A2 and STAT3 (Ser727), thereby suppressing phosphorylation of STAT3 (Ser727). Furthermore, intraperitoneal injection of SAL significantly delayed the growth of tumor and reduced the tumor volume in a SK-Hep1 xenograft mouse model. Taken together, SAL regulates STAT1/3 signaling, thereby inducing apoptosis in SK-Hep1 cells, which may account for its anti-carcinogenic activity in liver cancer.

Key words: Salsolinol, STAT3, STAT1, Liver cancer, Cell Death

Short CV

Hye-Kyung Na is a professor of Food Science & Biotechnology, College of Knowledge-Based Services Engineering, Sungshin Women's University, Seoul, South Korea. Prof. Na's research focuses on molecular mechanisms underlying anti-oxidant, anti-inflammatory, and anti-carcinogenic activities of dietary and medicinal phytochemicals targeting STATs and 15-hydroxyprostaglandin dehydrogenase in liver, breast, and colon carcinogenesis models.



From target identification to early-stage therapeutic discovery: leveraging *in vivo* preclinical models

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Abstract

Idiopathic pulmonary fibrosis (IPF) is an interstitial lung disease of unknown cause, marked by irreversible damage to lung structure and function. Currently, the therapeutic drugs available for pulmonary fibrosis include pirfenidone and nintedanib. However, these drugs are not economically viable and require relatively high doses, which lead to significant side effects. Thus, it is necessary to discover novel targets and develop therapeutics based on these targets for IPF treatment. To identify the molecular signatures of lung fibrosis, we investigated the fibrotic process in irradiated areas of mouse fibrotic lung tissues, identifying GTSE1 and autophagy-senescence axis as targets for lung fibrosis. We have also developed novel therapeutics targeting these new discoveries, and we would like to take the opportunity to introduce these findings.

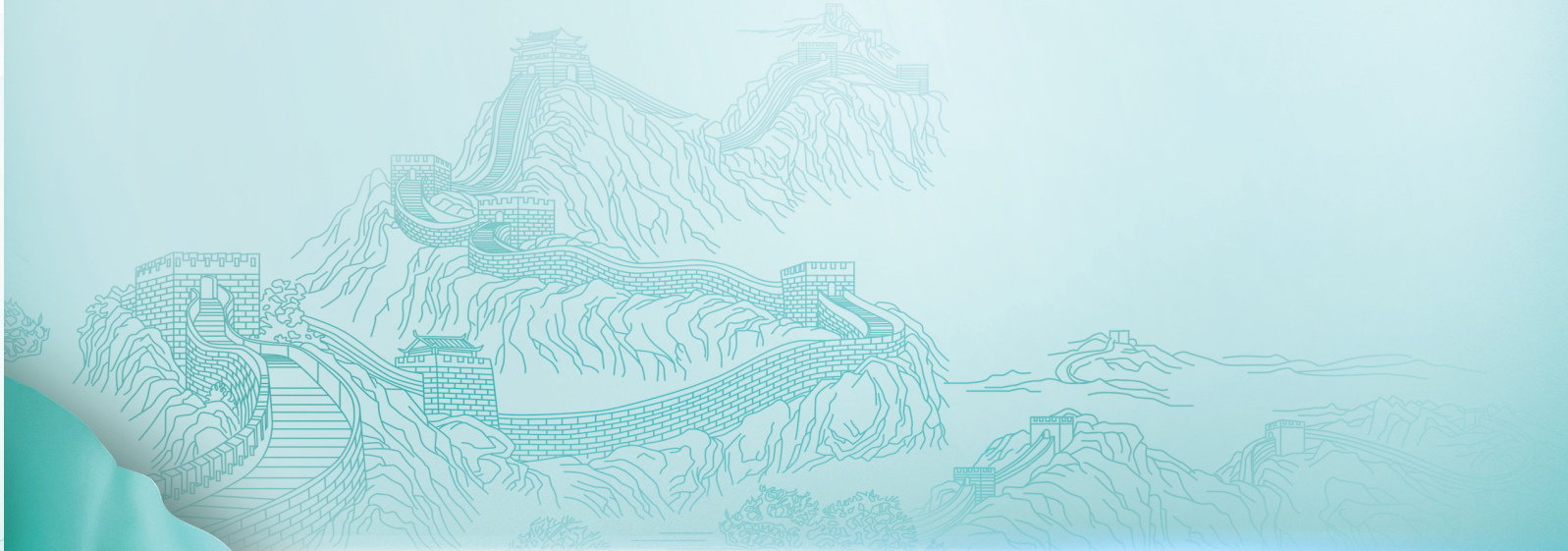
Key words: Idiopathic pulmonary fibrosis; Novel targets; Gtse1; Autophagy; Senescence

Short CV

Dr. Yun-Sil Lee is a full professor at the Graduate School of Pharmaceutical Sciences at Ewha Womans University. She earned her B.S., M.S., and Ph.D. degrees in the College of Pharmacy at Ewha Womans University. Following her academic journey, she pursued postdoctoral research at the National Cancer Institute (NCI) in the USA. For nearly two decades, she worked as a Principal Scientist at the Korea Institute of Radiological and Medical Sciences. Her research primarily revolves around the development of radiation protectors or sensitizers. More recently, her research interests have shifted towards the development of inhibitors for pulmonary fibrosis. Professor Lee's extensive research contributions are reflected in her publication record, with over 200 SCI(E) papers. Her expertise has also earned her invitations to international symposia as an invited speaker.

Symposium-YIO-4 (Y-4)

"New approach for precision redox research
Intelligence materials for precision redox intervention"





Chair: Xianquan Zhan (詹显全)

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Short CV

Prof. Dr. Xianquan Zhan received his MD, PhD training in preventive medicine at West China University of Medical Sciences during 1989–1999. He received his post-doctoral training in oncology and cancer proteomics at Central South University and University of Tennessee Health Science Center (UTHSC). He worked in UTHSC and Cleveland Clinic in United States during 2001–2012, and achieved the rank of Associate Professor at UTHSC. In 2012, he moved to Xiangya Hospital, Central South University as a Professor, and Advisors of MS/PhD graduate students and postdoctoral fellows. In 2020, he moved to Shandong First Medical University, where he is a Professor, Principal Investigator (PI), and advisors of MS/PhD graduate students and postdoctoral fellows at Medical Science and Technology Center. He is also the Fellow of Royal Society of Medicine, Fellow of World Academy of Productivity Science, Fellow of EPMA, European EPMA National Representative, Full member of ASCO, AAAS member, Shandong Province Taishan Scholar Distinguished Expert, Leader of medical disciplines in Hunan Province, Hunan Xiaoxiang Friendship Award, Hunan Provincial International Science and Technology Cooperation Award, Distinguished Professor of Hunan Provincial Hundred Talents Program, Jinan City class A high-level talents, Editor-In-Chief of IJCDT, Associate Editors of EPMA Journal, BMC Medical Genomics, and Frontiers in Endocrinology, and Guest Editors of Frontiers in Endocrinology, and Mass Spectrometry Reviews. He has published 180 articles, 10 academic books, 30 book chapters, and 3 international patents in the field of clinical proteomics and biomarkers, H-index 37. As the Guest Editor, he edited 18 special issues in SCI journals such as Mass Spectrometry Reviews, Frontiers in Endocrinology, EPMA Journal, Oxidate Medicine Cellular Longevity, and Med One. His main research interest focuses on the studies of cancer proteomics and proteoformics, multiomics and biomarkers, and the use of modern omics techniques and systems biology for predictive, preventive, and personalized medicine (PPPM; 3P medicine) and precision medicine (PM) in cancer.



Chemical proteomics reveals mechanisms of bacterial response to ROS mediated by antibiotics

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Abstract

Increasing bacterial resistance has become a major threat to human health, and a more comprehensive and in-depth understanding of the mechanisms of antibiotic sterilization can help us better address the problem of bacterial resistance. A variety of antibiotics have been proposed to cause bacterial death, in part by increasing the steady-state levels of reactive oxygen species (ROS) in bacteria. However, it remains unclear which proteins the ROS modify and their roles in antibiotic susceptibility/ resistance. To answer this question, we present a comprehensive, quantitative, and site-specific profile of E.coli cysteinome after 8 antibiotics treatment. Our data revealed antibiotic-mediated ROS targets modulate redox-sensitive events in nucleotide binding, cellular amino acid biosynthetic process, metabolic process, oxidative stress response, and etc. We also demonstrated that the tRNA-specific 2-thiouridylase MNMA_C102, the regulatory factor NEMR_C116, and TyrR_C337 are redox-regulated by antibiotic-mediated ROS, and that redox form shifts at these sites further affect enzyme activity, dimer formation, gene expression, etc., and ultimately the strain's susceptibility to antibiotics. Taken together, this study provides a molecular basis for understanding the relationship between antibiotic-mediated ROS targets and bacterial resistance, and offers a new perspective for finding new ways to enhance the bactericidal effect of existing antibiotics and developing novel antibiotics.

Key words: Antibiotic resistance, Reactive oxygen species, Cysteinome

Short CV

I joined Jing Yang's lab at the National Center for Protein Sciences • Beijing in 2015. Since then, I have been working on mapping protein S-sulfenylation (-SOH), S-sulfinylation (-SO2H) and S-Sulfhydration (-SSH) in various model organisms using state-of-the-art chemoproteomic technology. My interests mainly focus on redox proteomics to answer fundamental questions from a REDOX perspective, such as 1) Mechanism of antibiotic resistance in pathogenic bacteria; 2) Adaptation mechanism of microorganisms to extreme environments. I have published 10 research papers as the first/Co-first or co-corresponding authors in Nat Chem Biol, Nat Plants, Nat Commun, Nat Protoc, Antioxid Redox Signal and so on. Of these, 4 papers have been featured by Nature Research Journals in News & Views or Research Highlights.



Molecule-guided precise identification and intervention of senescence

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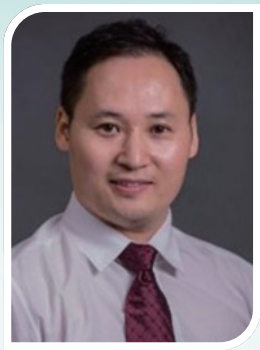
Abstract

Cellular senescence is believed to be a driver of aging. Guo's group designed and synthesized small-molecule based fluorescent probes with high spatiotemporal resolution to track senescence precisely, and developed prodrugs that destroys senescent cells by integrating multiple technologies that combine biomarker guidance with a fluorescence tag, target-site anchoring and photodynamic therapy. As such, the group created strategies for monitoring and specifically eliminating senescent cells to regulate aging. As such, they achieved: 1) the innovations in senescence/aging "tracing" approaches involving the species-specific identification of senescent-associated protein1, the two-dimensional senescence identification associated with senescent microenvironmen and the dynamic identification of aging of individuals under stress and 2) the breakthroughs in senescence/aging "intervention" technology involving the organ prodrug strategy to selectively eliminate senescent cells, and the individual prodrug strategy to eliminate senescent cells with single-cell resolution.

Key words: Aging, Senescence, Molecular Probes, Prodrugs, Identification and Intervention

Short CV

Yuan Guo is a professor at the Northwest University, China. She obtained her BSc in 2001 and PhD in 2006 from Northwest University. She spent two years in France, as a Post-doctoral Research Fellow (2012-2014) at Institut de Chimie Organique et Analytique, University of Orleans, UMR CNRS, and was an invited researcher at University of Orleans (2019), France. Her research interests include: chemical biology; molecular probes in biological systems; tracing and intervention of aging/senescence. Since she started her research work independently, more than 70 research papers have been published in academic journals as a correspondent author, including Nat. Aging, Angew. Chem. Int. Ed., Adv. Sci., Chem. Sci., etc. 10 national invention patents have been authorized and two of them have been transferred to companies. In addition, she serves as a member of the editorial board of several journals such as Chinese Chemical Letters, Acta Materia Medica, and Fine Chemicals.



Simultaneous quantitation of persulfides, biothiols and hydrogen sulfide through efficient sulfur exchange reaction with trityl spin probes

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Abstract

Reactive sulfur species (RSS) including persulfides (RSSHs), biothiols (RSHs) and hydrogen sulfide (H2S) are key regulators in various physiological processes. To better understand the symbiotic relationship and interconversion of these RSS, it is highly desirable but challenging to develop analytical techniques that are capable of detecting and quantifying them. Herein, we report the rational design and synthesis of novel trityl radical-based electron paramagnetic resonance (EPR) probes dubbed as CT02-TNB and OX-TNB for various RSS. CT02-TNB underwent fast sulfur exchange reactions with two reactive RSSHs (PS1 and PS2) which were released from their corresponding donors PSD1 and PSD2 to afford the specific conjugates. The resulting conjugates exhibit characteristic EPR spectra, thus enabling the discriminative detection and quantitation of the two RSSHs. Moreover, CT02-TNB showed good response towards other RSS including glutathione (GSH), cysteine (Cys), H2S and sulfite as well. Importantly, based on the updated EPR spectral simulation program, simultaneous quantitation of multiple RSS (e.g., PS1/GSH/Cys or PS1/GSH/H2S) by CT02-TNB was also achieved. Finally, the levels of the released PS1 from PSD1 and endogenous GSH in isolated mouse livers were measured by the hydrophilic OX-TNB. This work represents the first study achieving discriminative and quantitative detection of different persulfides and other RSS by a spectroscopic method.

Key words: persulfide; reactive sulfur species (RSS); redox; electron paramagnetic resonance (EPR); probe

Short CV

Yangping Liu received the PhD degree in Institute of Chemistry, Chinese Academy of Sciences in 2006 and then moved to The Ohio State University as a post-doc and research scientist from 2006 to 2013. In 2013, he joined Tianjin Medical University as a full professor and then promoted to vice dean in School of Pharmacy. Dr. Liu's research interest is to develop novel EPR probes for precise measurement of redox status in biological systems, stable radicals as magnetic resonance-related agents and gas signaling donors for cardiovascular diseases. Dr. Liu is active in the field of free radicals with more than 70 scientific papers. Dr. Liu is currently an executive member of Chinese Society of Free Radical Biology and Medicine and was awarded as Excellent Young Research Award from Xu Yuanzhi Award Funds of EPR Development in 2020.



Spatial transcriptome profiling of a Huntington's disease mouse brain with BASSFISH

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Abstract

Unraveling spatiotemporal dynamics in cells and molecules during disease progression is pivotal for elucidating disease mechanisms. We present BASSFISH, an innovative imaging-based spatial transcriptomics technique that leverages signal amplification and coded multiplex hybridization for rapid, cost-effective acquisition of high-resolution transcriptomics and multi-protein expression data within tissue samples. Our study employed BASSFISH to analyze over 1200 genes and multiple proteins in 6-month-old CAG-140 mice, a Huntington's disease model, using whole-brain sagittal sections. Integrating these findings with single-cell sequencing, we discovered cell-type and regional transcriptomic alterations during the early stages of HD, thereby generating a comprehensive cellular map of disease progression. BASSFISH stands as a powerful instrument for pinpointing the molecular and cellular underpinnings of disease pathology.

Key words: Spatial Transcriptome, FISH, Hunting's disease, imaging

Short CV

Professor at the School of Life Sciences and Medicine, University of Science and Technology of China, and Hefei National Research Center for Physical Sciences at the Microscale; Invesigator at the Institute of Artificial Intelligence, Hefei Comprehensive National Science Center; Recipient of the National Innovation Talent Youth Project.

He has been primarily engaged in the development of single-molecule imaging technology, including superresolution localization microscopy and imaging-based spatial transcriptomics, and their applications in neuroscience. His research in neuroscience focuses on the structural mechanisms of synaptic transmission and plasticity, and the pathogenesis caused by their abnormalities, including significant discoveries such as synaptic nanocolumns. He has published multiple papers in renowned journals including Nature, Nature Methods, PNAS, and Science Advances as the corresponding author.



Generation of short chain aldehydes and increase of oxidative stress in mice by intake of fructose

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Abstract

Aldehyde is reactive to amino, imino, guanidyl, and thiol groups, which deteriorates protein function and induce pathological responses such as inflammation, oxidative stress and consequently negative effects on human health. It is well known that alcohol intake produces acetaldehyde in the liver. In addition to alcohol-induced aldehyde, glucose, one of the carbohydrates that is the main source of energy, also contains an aldehyde group. However, most glucose has hemiacetal structure, and the amount of aldehyde derived from glucose in the body is not so high. Meanwhile, it is known that short-chain aldehydes such as glyceraldehyde and methylglyoxal are produced from the metabolic products of monosaccharides. Furthermore, it is known that malondialdehyde and other compounds are produced by the peroxidation of unsaturated fatty acids. Therefore, oxidative stress produces aldehydes. Conversely, it is possible that the generation of aldehydes may cause oxidative stress *in vivo*. However, the effect of the aldehydes generated in body on oxidative stress is not fully understood, as it has been difficult to quantify aldehydes *in vivo*. Recently, we have established a method for the quantitative determination of glyceraldehyde in body (Martin-Morales et al., J. Agric. Food Chem. doi. org/10.1021/acs.jafc.1c03177). We also developed the quantitative stable determination of methylglyoxal and malondialdehyde. As mentioned above, aldehydes are produced by the metabolism of monosaccharides. It has been reported that high intake of fructose damages the functions of the liver and small intestine. Therefore, in this study, we evaluated the production of aldehydes in the body of mice administered glucose and fructose (2 g/kg body weight).

The increase in blood fructose after administration of fructose was much smaller than that after administration of glucose. Unexpectedly, administration of ^{13}C-labeled fructose increased unlabeled glucose in the blood within a few minutes of administration. These results indicate that administration of fructose causes a rapid activation of the gluconeogenesis system. In the liver and kidney, where gluconeogenesis occurs, administration of ^{13}C-fructose significantly increased the production of ^{12}C-methylglyoxal compared with administration of glucose, indicating that methylglyoxal is produced by gluconeogenesis rather than glycolysis. In the small intestine, fructose administration resulted in significantly higher glyceraldehyde production than glucose administration. Administration of ^{13}C-fructose increased ^{13}C-glyceraldehyde in small intestinal tissue and lumen. These results indicate that fructose is metabolized to glyceraldehyde in the small intestine, and some of it leaks into the lumen. Malondialdehyde, a secondary product of lipid peroxidation, was significantly increased in the blood and kidneys by fructose administration compared to glucose administration. This showed that fructose also increases oxidative stress throughout the body.

Fructose administration has been shown to damage liver function and intestinal barrier function. This study revealed that fructose administration increases glyceraldehyde, a metabolic product of fructose, in the small intestine, and further increases methylglyoxal in the liver and kidneys by activating the gluconeogenesis. This suggests that even in cases other than excessive alcohol intake, fructose intake produces highly reactive short-chain aldehydes, which increases oxidative stress in the body and damages organ functions.

Key words: aldehyde, glyceraldehyde, methylglyoxal, fuructose, malodndialdehyde



GSH-induced in situ peptide self-assembly for precise tumor imaging and ROS-based therapy

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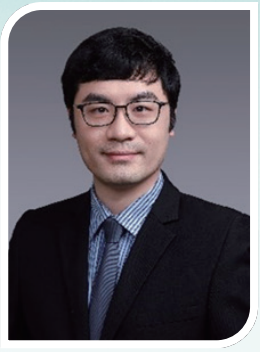
Abstract

Glutathione (GSH) is a key component of the cellular antioxidant system and is the primary nonprotein biothiol found at high concentrations within cells. It plays a crucial role in protecting cellular components from damage caused by reactive oxygen species (ROS) and toxins. In cancer cells, GSH concentrations are approximately 1,000 times higher than in normal cells, making it a significant biomarker for cancer diagnosis. Additionally, combining GSH with other tumor biomarkers, such as proteases and αv integrins, to develop dual-locked responsive probes and drugs can enhance the precision of tumor imaging and treatment. The in situ self-assembly strategy can enable small molecules to specifically accumulate at tumor sites, thereby increasing the local concentration of probes and drugs, improving imaging signals and therapeutic outcomes, and extending treatment duration. In this talk, I will introduce the GSH-responsive in situ peptide self-assembly strategy for enhanced imaging and ROS regulation-based therapy of tumors.

Key words: GSH; peptide self-assembly; tumor imaging; ROS-based therapy

Short CV

Yue Yuan is a Professor in the Department of Chemistry at the University of Science and Technology of China (USTC). She obtained her Ph.D. from the Department of Chemistry at USTC from 2011 to 2015. Following this, she served as a postdoctoral researcher at USTC from 2015 to 2016, and later at the School of Medicine at Johns Hopkins University from 2016 to 2020. She joined USTC in mid-2020. Dr. Yuan′ s current research focuses on redox and protease induced self-assembly for molecular imaging, with particular emphasis on peptide self-assembly for CEST MRI research.



Molecular targeting nano drug candidates

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Abstract

Currently, the clinical approved nanomedicines are mainly the delivery systems. To develop the nanomedicine that specifically targeting the biomolecules for disease treatment is still a challenging. The study of molecular targeting nanomaterials for drug candidates should be similar to study the small molecule chemical drugs to clearly evaluate and clarify their molecule-targets, revealing the binding sites with a purpose to disclose the potential biological/immunological/pharmacological mechanisms. Therefore, the development of molecular targeting nano drug candidates should be focused on exploring their intrinsic and specific biological properties, which could easily pave the way for future clinical translation. We have discovered several nanomaterials that could specifically/selectively target biomolecules for disease treatments. Black phosphorus (BP) could be used as DNA checkpoint inhibitor to specifically target and inactivate the PLK1 kinase, which consequently causes centrosome dysfunction, mitotic catastrophe, and ultimately leads to cell apoptosis. Thus, BP could be used as a potential anti-tumor drug. CuInP2S6 (CIPS) and cobalt hydroxide nanosheets (CHN) could selectively target the RBD region of SARS-CoV-2 spike protein. Such interaction caused a denaturation of the secondary structure of RBD and occupied the relevant binding sites of RBD-ACE2, thereby inhibiting the infection of SARS-CoV-2 in host cells. Thus, CIPS and CHN could be used as a potential anti-SARS-CoV-2 nano-drug. In addition, based on the three-dimensional structure of the spike protein, the cerium dioxide nanoparticles can selectively target this three-dimensional structure for function inhibition, achieving an effective antiviral effect. These studies revealed that nanomaterials could have specifically/selectively intracellular or extracellular molecular targets, analyzed the nano-bio interfaces and the detailed binding sites, and investigated the valence states of nanomaterials and the spatial effects of proteins for such nano-bio interactions. It clarifies the feasibility of molecular targeting nanomaterials and provides the fundamental strategies for future studies towards this direction.

Key words: Molecular targets, Nano-bio interface analysis, Nanomedicine development

Short CV

His group is now focusing on understanding the basis and mechanism of the nano-bio/immuno interactions and how these interactions modulate the immunological responses. With such basic information, his group is trying to develop efficient nano-formulated medicine to treat infectious/inflammatory diseases and cancer. Prof. Li has published over 50 scientific publications including Nature Nanotechnology (2021, 2022), etc. He awarded the "First Prize of Natural Science in Hebei Province", "Marie Curie Fellowship of European Commission" and "Chinese Government Award for Outstanding Self-financed Students Abroad". Currently, his research team is supported by National Natural Science Foundation of China, National Key R&D Program of China, Natural Science Foundation of Guangdong Province, and funds from Chinese Academy of Sciences, etc.



Nanozybiotics: advancing antimicrobial strategies through biomimetic mechanisms

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Abstract

Infectious diseases caused by microbes represent a global threat to human health. However, due to the abuse of antibiotics, antimicrobial resistance has evolved rapidly and led to the failure of antibiotics treatment. Thus, alternative antimicrobial strategies different to traditional antibiotics are urgently needed. Lysosomal redox enzymes-based bacteria killing plays a vital role in innate immune defense system, inspiring a novel antimicrobial strategy. However, due to their low stability, potential immunogenicity, and high cost, natural enzymes have limitations in practical antimicrobial therapy. In recent years, many nanomaterials with enzyme-like activities (Nanozymes) have been discovered as a new generation of artificial enzymes which have high activity, high stability, multifunctionality and low cost for large scale. In particular, nanozymes that can mimic the activities of lysosomal enzymes such as oxidase, peroxidase, demonstrated highly antimicrobial effects against pathological bacteria or viruses. To highlight the progress in the field of nanozymes-based antimicrobial strategy (Nanozybiotics), we summarized the antimicrobial mechanisms of action, versatile therapeutics and translational potentials of nanozybiotics in various infectious diseases. We believe that nanozybiotics will provide a new strategy by mimicking immune defense using nanozymes to combat antimicrobial resistance.

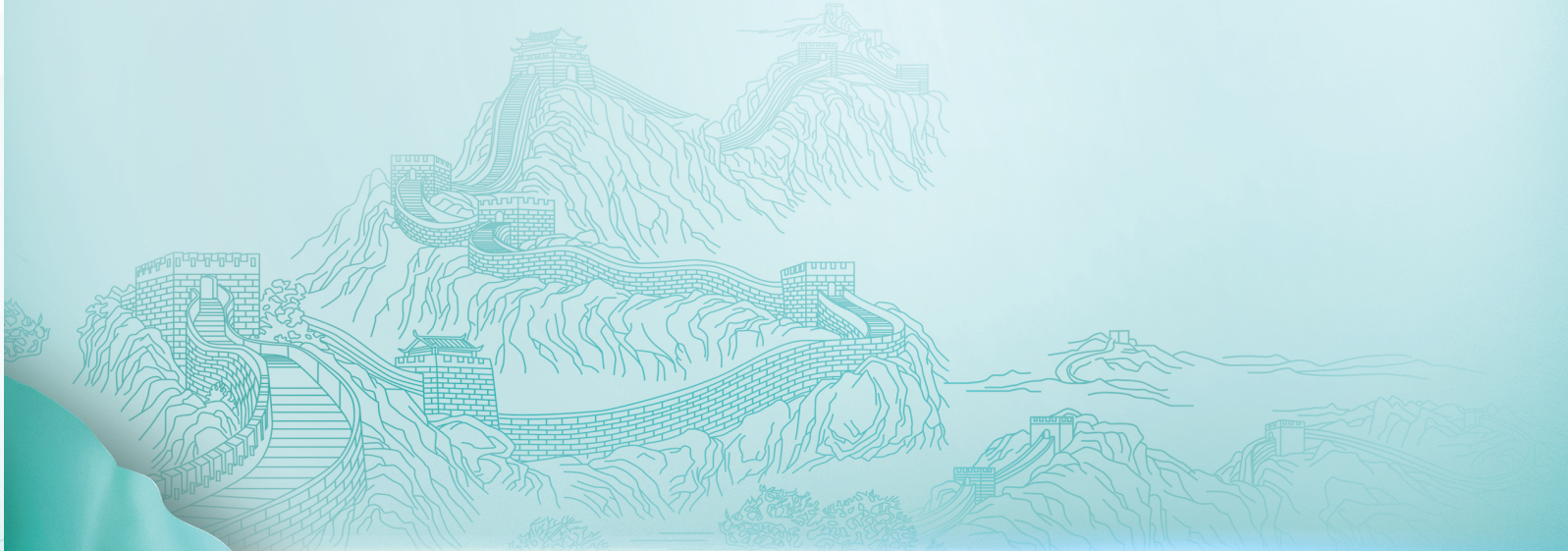
Key words: Nanozymes, Lysosome Redox, Biomimetic, Antimicrobial resistance, Nanozybiotics

Short CV

Lizeng Gao is a professor at Institute of Biophysics, Chinese Academy of Sciences (CAS). His research focuses on discovering intrinsic biological activities of nanomaterials (nanozymes) and developing biomimetic strategies against antimicrobial resistance including bacteria, fungi and viruses.

Symposium-YIO-5(Y-5)

"Discovery of new molecules in redox network
Natural products and nutrition in anti-aging and health management"





Chair: Jun Lu (陆军)

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Short CV

Prof. Dr. Jun Lu earned, from Sichuan University, his B.Sc. in Chemistry and his M.Sc. in Bioinorganic Chemistry in 1992 and 1995 respectively. He received his Ph.D. in Chemistry from the Changchun Institute of Applied Chemistry, Chinese Academy of Sciences in 1998. He had worked in The Institute of Physical and Chemical Research (RIKEN), Japan from 2000 to 2002, Karolinska Institutet, Sweden from 2003 to 2016. He currently works as the Director of the Teaching and Research in Pharmacology Section, College of Pharmaceutical Sciences, Southwest University, China. Additionally, he is a member of the editorial board of Antioxidants, Frontier in Physiology, and so on. He has published more than 70 scientific papers with a H-index of 40. His current research interests include the study of the mechanism and development of drugs based on targeting the regulation of thiol redox system, and the novel biopharmaceutical technology.

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Chair: Ock Jin Park

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Short CV

1966 -1970 Seoul National University College of Pharmacy B.S.

1970 - 1977 University of Minnesota Nutrition M.S. and Ph.D. Candidate

1984 - 1988 Ewha Womans University Nutrition Ph.D.

1980 - 2013 Hannam University College of Bionanoscience: Assistant Professor, Associate Professor, Full Professor

2013 - present Hannam University College of Bionanoscience: Honorary Professor

2021 - present Hanyang University Consulting Researcher

1980 - 2013 Korea Nutrition Society Member, Advisory Board Member and Scientific Committee Member

2001 - 2013 Korean Society of Cancer Prevention Member, Advisory Board Member, Vice President and Scientific Committee Member

2001 - 2013 Members of SFRR of Korea, Korea Toxicology Society, Korea Biomedical and Biochemistry Society, Korea Biochemistry and Molecular Biology



Targeting DNA repair to extend lifespan

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Abstract

Oxidative stress induces various DNA damages, and efficient repair is vital for delaying aging and preventing age-related diseases. While DNA repair deficiencies are known to hasten aging, targeted DNA repair for anti-aging purposes is an underexplored area. Our identification of several potential targetable DNA repair factors that could slow aging provides a theoretical basis for developing strategies to extend healthy lifespan. These factors may include specific repair enzymes or regulatory proteins, offering new avenues for interventions to enhance DNA repair and promote longevity.

Key words: DNA repair and aging

Short CV

• ACADEMIC EDUCATION:

Ph.D. Biology, University of Rochester, USA, 2010

M.S. Biology, Nanjing University, China, 2004

• RESERCH & PROFESSIONAL EXPERIENCE:

2012-present Professor, Tongji University, China

2010-2012 Postdoc, University of Rochester, USA

• MAJOR RESERCH INTERESTS:

DNA repair and aging



Redox-regulated iron metabolism and ferroptosis in ovarian cancer: molecular insights and therapeutic opportunities

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Abstract

Ovarian cancer (OC), known for its lethality and resistance to chemotherapy, is closely associated with iron metabolism and ferroptosis—an iron-dependent cell death process, distinct from both autophagy and apoptosis. Emerging evidence suggests that dysregulation of iron metabolism could play a crucial role in OC by inducing an imbalance in the redox system, which leads to ferroptosis, offering a novel therapeutic approach. This review examines how disruptions in iron metabolism, which affect redox balance, impact OC progression, focusing on its essential cellular functions and potential as a therapeutic target. It highlights the molecular interplay, including the role of non-coding RNAs (ncRNAs), between iron metabolism and ferroptosis, and explores their interactions with key immune cells such as macrophages and T cells, as well as inflammation within the tumor microenvironment. The review also discusses how glycolysis-related iron metabolism influences ferroptosis via reactive oxygen species. Targeting these pathways, especially through agents that modulate iron metabolism and ferroptosis, presents promising therapeutic prospects. The review emphasizes the need for deeper insights into iron metabolism and ferroptosis within the redox-regulated system to enhance OC therapy and advocates for continued research into these mechanisms as potential strategies to combat OC.

Key words: redox, iron metabolism, ferroptosis, ovarian cancer, tumor immune microenvironment, glycolysis

Short CV

Dr. Jinzhi Lu is an active researcher in the field of oncology, with a specific focus on ovarian cancer and the mechanisms of chemotherapy resistance. His academic interests lie in iron metabolism, oxidative stress, and the role of signaling pathways in cancer stem cell formation and drug resistance, particularly in relation to cisplatin. He has led and participated in multiple funded research projects, including studies on the mechanisms of ferroptosis and the regulatory roles of proteins like HIF-1α in ovarian cancer.Dr. Lu has authored numerous impactful publications in high-ranking journals, contributing to the understanding of TRAIL receptors in early pregnancy loss and the identification of critical genes for cisplatin resistance. His work contributes to the advancement of scientific knowledge and may help inform potential therapeutic interventions in cancer treatment.



Restored PGAM5-mediated oxeiptosis eliminates ROS high cardiomyocytes and improves cardiac function during cardiac aging

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Abstract

Cardiac aging is a major risk factor of CVDs as the incidence of cardiovascular disease as well as the rate of cardiovascular mortality and morbidity increase exponentially in the elderly population. It has been well established that cardiomyocyte death executed by diverse forms of cell death are linked with the adverse outcome of aged heart, it is unclear whether a certain form of cardiomyocyte death may serve a protective role in aged hearts. Here, we explored the role of PGAM5 and its mediated oxeiptosis in cardiac aging. We observed a decrease in PGAM5 levels in aged hearts. Further investigation in a cardiomyocyte-specific Pgam5 knockout (Pgam5 KO) mouse model revealed premature cardiac aging, characterized by increased oxidative stress and an accumulation of ROS high cardiomyocytes. In vitro and *in vivo* analysis revealed that Pgam5 KO led to the inhibition of oxeiptosis in cardiomyocytes. Specifically, we discovered that aged hearts exhibited suppressed PGAM5-mediated oxeiptosis, leading to the accumulation of ROS high cardiomyocytes. Subsequently, we found that restoring PGAM5-mediated oxeiptosis not only eliminated ROS high cardiomyocytes but also significantly improved cardiac function in aging hearts. Collectively, these findings underscore the role of PGAM5-mediated oxeiptosis in maintaining cardiac health and suggest potential therapeutic strategies for combating cardiac aging.

Key words: PGAM5, Cardiac Aging

Short CV

Prof. Moshi Song is a principal investigator in the Institute of Zoology of the Chinese Academy of Sciences. Prof. Song got her master's degree at Karolinska Institute (Sweden) and her Ph.D. at Washington University in St. Louis (USA), received her postdoctoral training at Stanford University (USA), and joined the Institute of Zoology as the leader of the Group of Mitochondrial Disease in 2018. Prof. Song has been focusing on the study of mitochondrial regulation of cardiac aging and related diseases and has published 44 papers in SCI journals including Cell, Cell Research (x2), Advanced Science (x2), Nature Communications (x2), Nature Aging (x2), Nucleic Acids Research (x2) during the last five years. Her major findings are as follows: 1) Elucidation of molecular mechanisms of cardiac aging; 2) Uncover of new biomarkers of cardiac aging; 3) Establishment of interventive strategies for cardiac aging and related diseases.



Exploring the neuroinflammatory pathways of 8-oxoGTP and their effects on cognitive decline

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Abstract

Neuroinflammation mediated by continuously activated glial cells may play a core role in neurodegenerative processes and cognitive deficits. Oxidative stress (OS) is considered to be one of the main underlying mechanisms of neurodegenerative diseases and is closely related to other pathological events. The cytoplasmic 8-oxoGTP generated by GTP oxidation increased during OS. On the one hand, 8-oxoGTP can serve as a substrate for RNA synthesis and cause RNA oxidation, leading to impaired protein synthesis, on the other hand, it can be used as a small molecule to regulate signaling. Given the susceptibility of 8-oxoGTP, free 8-oxoGTP as a signaling modulator requires more in-depth study. In this study, we administered multiple intracerebroventricular injections of 8-oxoGTP to SAMP8 mice and observed a significantly impaired performance in cognitive in behavioral experiments. Immunohistochemical analysis further demonstrated a marked reduction in the number of neurons in the cortical and hippocampal regions, accompanied by a significant increase in the number of activated microglia and elevated secretion of inflammatory cytokines. Cellular experiments indicated that 8-oxoGTP activates microglia by triggering the MAPK, AKT, and NF-κB signaling pathways, promoting an inflammatory phenotype. This mechanism highlights the critical role of microglia in 8-oxoGTP-induced neuroinflammation. Our study reveals the significant impact of 8-oxoGTP on neuroinflammation and cognitive decline, providing a theoretical foundation for exploring related therapeutic strategies.

Key words: Neuroinflammation, Oxidative stress, 8-oxoGTP

Short CV

Dr. Jin Li graduated from Peking University Health Science Center. Since 2018, she has been working in Beijing Hospital. She conducts research on nucleic acid oxidation and aging and aging-related diseases. She is supported by the National Natural Science Foundation and the Fundamental Research Funds for the Central Universities. She is a youth member of the free radical biology and free radical medicine branch.



ATF-4 and hydrogen sulfide signaling mediate longevity in response to inhibition of translation or mTORC1

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Abstract

The serine/threonine protein kinase complex mTORC1 (mechanistic target of rapamycin complex 1) is a master regulator of growth and metabolism. Inhibition of mTORC1 slows ageing across phyla, in part by reducing protein translation. Various stresses, including oxidative stress, nutrient deprivation, and loss of proteostasis, globally suppress protein synthesis through the integrated stress response (ISR), resulting in preferential translation of the transcription factor ATF-4. Here we show in C. elegans that inhibition of translation or mTORC1 increases ATF-4 expression independently of the canonical ISR signaling. Overexpression of ATF-4 is sufficient to extend healthspan and lifespan. ATF-4 not only activates canonical anti-ageing mechanisms, but also elevates expression of the transsulfuration enzyme CTH-2 to increase hydrogen sulfide (H2S) production. The ATF-4/CTH-2/H2S pathway also mediates longevity and increased stress resistance from mTORC1 suppression. Together, our results suggest that increasing H2S levels, or enhancing mechanisms that H2S influences through persulfidation on protein cysteines, may represent promising strategies for mobilising therapeutic benefits of the ISR, translation suppression, or mTORC1 inhibition.

Key words: aging, hydrogen sulfide, ATF-4, mTORC1

Short CV

2022 - Associate Professor, Capital Medical University

2021 - 2022 Senior Mary K. Iacocca Postdoctoral Fellow

2020 - 2021 Junior Mary K. Iacocca Postdoctoral Fellow

2018 - 2020 Research Fellow, Joslin Diabetes Center, Harvard Medical School

2018 PhD in Cell Biology, Yale University

2012 B.S. in Life Sciences, Peking University

Selective Publications:

[1] Meng, J. & Ferguson, S. M. J Cell Biol (2018).

[2] Meng, J., Fu, L. et al. Nat Commun (2021).

[3] Statzer, C., Meng, J. et al. Nat Commun (2022).



Network medicine landscape on the health-enhancing properties of natural antioxidants

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Abstract

Natural antioxidants have attracted increasing attention for their potential in promoting health and preventing disease, yet the mechanisms underlying their redox activities and their protective effects remain incompletely understood. Network medicine, an emerging field that analyzes complex molecular interactions, offers a promising framework for uncovering how these compounds function at a systems level. However, a gap exists in integrating network medicine with the study of natural antioxidants, particularly in understanding the molecular networks involved. This study addresses this gap by employing a network-based approach to investigate the interactions between natural antioxidant compounds and key regulatory nodes within biological signaling networks. We focus on the role of these interactions in maintaining cellular redox balance and mitigating the progression of chronic diseases associated with oxidative stress. Our analysis identifies specific compounds, including quercetin, palmitic acid, and linoleic acid, present in camellia oil, which modulate critical metabolic pathways related to phospholipids, fatty acids, and bile acids. These findings demonstrate the potential of network medicine to uncover novel antioxidant therapies and elucidate the molecular mechanisms that confer protection against tissue injury caused by oxidative stress. This study highlights the critical role of network medicine in advancing the understanding of natural antioxidants. Furthermore, it advocates for the integration of comprehensive evaluation criteria including chemical composition analysis, bioactivity assays, and animal study to assess the safety and efficacy of these compounds as health supplements. By bridging the gap between molecular insights and therapeutic applications, our study contributes to the development of innovative strategies for health promotion.

Key words: antioxidant, natural product, network medicine, health promotion

Short CV

Guozhen Cui currently serves as a professor and PhD supervisor at Zhuhai Campus of Zunyi Medical University. He received his PhD degree in Biomedical Sciences from University of Macau in 2013. He His research focused on the pharmacological studies of Chinese herbal medicine and functional foods, using integrated approach combining network medicine framework-based prediction with experimental validation. Dr. Cui has published over 60 papers in SCI-indexed journals and has obtained research fundings from various external agencies and industry partners, including 3 NSFC.



Cellular senescence and rejuvenation

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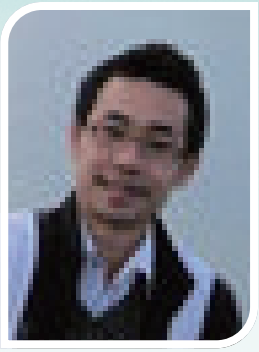
Abstract

In response to a variety of stress factors, certain cells in our organs undergo a transition into a state of senescence, contributing to the accumulation of such cells in different organs as part of the aging process. These senescent cells play a role in the structural and functional decline of organs and are associated with degenerative diseases. Targeting these cells is a critical aspect of the broader strategy to combat aging and help to rejuvenate. However, the heterogeneity of senescent cells and their ambiguous characteristics *in vivo* pose challenges, exacerbated by the lack of efficient methods for their detection and targeted intervention within the body. Dr. Jing Qu is comitted to investigating both the driving causes and *in vivo* impacts of senescent cells. Her research interest is to uncover novel biomarkers and develop intervention strategies to manage cellular senescence and the degeneration in organ structure and functionality.

Short CV

Jing Qu, researcher at the Institute of Zoology, Chinese Academy of Sciences, is mainly engaged in cellular senescence. Cells across different organs undergo a transition into a state of senescence, and the accumulation of such cells in different organs is a part of the aging process. These senescent cells play a role in the structural and functional decline of organs and are associated with degenerative diseases. Jing Qu is committed to investigating the properties of senescent cells, as well as their driving causes. Her research interest is to develop intervention strategies to manage cellular senescence and the degeneration in organ structure and functionality.

Jing Qu received her B.S. from Lanzhou University in 2002, and then the doctor degree from the Institute of Biophysics, CAS in 2007. Following that, she worked as an AFAR Postdoctoral Fellow at the Del E. Web Neuroscience, Aging, and Stem Cell Research Center at the Sanford/Burnham Medical Research Institute, and then as a Research Associate in the Gene Expression Laboratory at the Salk Institute for Biological Studies. In 2014, she initiated her research group focusing on " Stem Cell and Aging"at the Institute of Zoology, CAS. She is now the Chair of the Aging Genetics Elites of the Genetics Society of China, and also a member of Scientific Program Committee of ISSCR. She received the Chinese Young Women in Science Fellowship for her contributions to aging research.



A hybrid sweet potato (Maejo 341) mitigates LPS-induced inflammation and RANKL-induced osteoporosis by regulating ROS-mediated pathways

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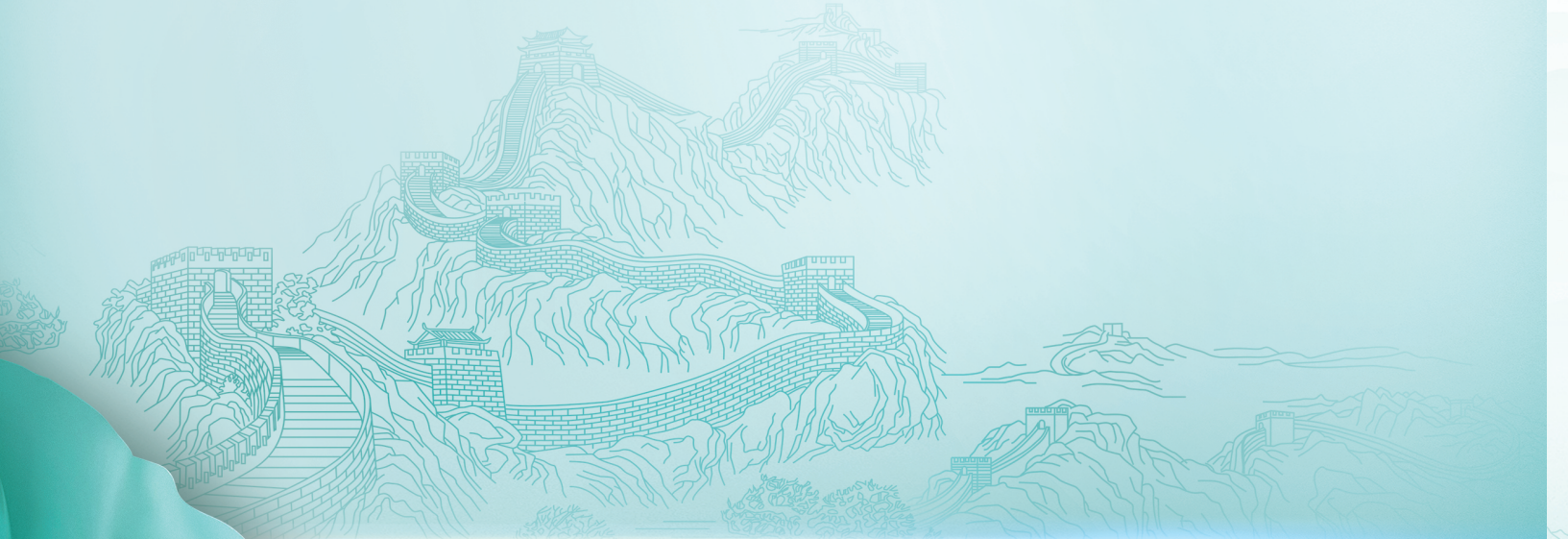
Abstract

"Maejo 341 sweet potato" is one of the new purple sweet potato varieties cultivated in Chiang Mai, Thailand through a crossbreeding of "Phichit 65-3" and "Mun-Khai Sukhothai". However, the health-related impacts of this hybrid crop remain largely unknown. The peel (P) and flesh (F) of Maejo 341 sweet potato were extracted with ethanol (E) and water (W) and denoted as PE, PW, FE, and FW, respectively. The extracts were used to investigate antioxidant, anti-inflammatory, and anti-osteoporotic activities as well as nutritional properties. PE and PW possess a higher phenolic and flavonoid contents, and antioxidant capacity than FE and FW. Therefore, we exclusively chose the peel extracts for further investigations. PE and PW inhibited lipopolysaccharide (LPS)-induced inflammation by suppressing the secretion of nitric oxide and matrix metalloprotein-9 in a dose-dependent manner. PE and PW at 50 mg/mL showed a significant reduction in mRNA levels of representative proinflammatory cytokines, TNF-α, IL-1b, and IL-6, and inhibited expression of two prototypic proinflammatory enzymes, cyclooxygenase-2 and inducible nitric oxide synthase and their mRNA transcripts. PE and PW also induced osteoclast differentiation by suppressing RANKL-stimulated TRAP activity, formation of TRAP-positive multinucleated cells, and expression of TRAP mRNA as well as down-regulation of the osteoclastogenic gene expression. Notably, PE and PW reduced production of reactive oxygen species and enhanced the antioxidant gene expression induced by both LPS and RANKL treatment. Furthermore, PE and PW also stimulated osteogenic activity by inhibiting the TNF-a-mediated decrease in osteoblast viability and alkaline phosphatase activity. The LC-MS and HPLC analyses revealed that the peel extracts contain substantial amounts of anthocyanins, specifically cyanidin-3-O-glucoside, peonidin-3-O-glucoside, and pelargonidin-3-O-glucoside. These compounds are notable for their powerful antioxidant properties, which contribute to anti-inflammatory and anti-osteoporosis effects. All these findings, taken together, suggest the potential use of anthocyanin-enriched Maejo 341 sweet potato peel as an alternative functional food to alleviate oxidative stress- and inflammation-associated osteoporosis, and its ingredients may be developed as the natural pharmaceutical formulations for bone health.

Key words: Maejo 341 sweet potato, Anthocyanins, Osteoporosis, Lipopolysaccharide (LPS), Receptor Activator of Nuclear Factor Kappa-B Ligand (RANKL)

Symposium-YIO-6(Y-6)

Redox signaling in organelles/cell fate/development/reproduction





Chair: Zhangjian Huang (黄张建)
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Short CV

Zhangjian Huang received his B.S. and Ph.D. degrees from China Pharmaceutical University (CPU) in 2004 and 2009, respectively. During 2009 to 2012, he worked as a postdoctoral fellow at University of Alberta in Canada. He joined the Center of Drug Discovery at CPU in 2013. He is now a Professor of Medicinal Chemistry at CPU. His research interests mainly focus on the discovery of novel gaseous signaling molecule nitric oxide (NO)-based agents for the intervention of cardiovascular diseases.

He has published about 80 SCI papers, including 25 in J Med Chem, a top journal in the field of medicinal chemistry. He won Distinguished Professor of the Ministry of Education's "Changjiang Scholars" program in 2024. He has hold 7 funds from National Natural Science Foundation of China including National Natural Science Foundation--Outstanding Youth Foundation (81822041, 2018). In addition, he held Jiangsu Province Funds for Distinguished Young Scientists (BK20160033 in 2016) and Program for New Century Excellent Talents in University (NCET-13-1033, in 2013). He has applied 28 Chinese patents and 10 PCT patent, and obtained 18 authorized patents. He won CPA-Servier Young Investigator Awards in Medicinal Chemistry in 2014.



cxcl18b-defined transitional state-specific nitric oxide signaling drives injury-induced Müller Glia proliferation in the zebrafish retina

Jie He (何杰)

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Center for Excellence in Brain Science and Intelligence Technology

Chinese Academy of Science, China

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Abstract

Zebrafish quiescent Müller glia (MG) can respond to the retina injury by re-entering the cell cycle, a critical step evolutionarily absent from their mammalian counterpart, which is essential for neuron regeneration program. MG could regenerate all retinal fates after the injury in the zebrafish retina by undergoing reprogramming and produced MG derived progenitors. However, the molecular and cellular mechanism driving this species-specific injury-induced MG proliferation may shed the light on new therapeutic strategy to repair human retina diseases but remains largely understood. In our study, single-cell transcriptome analysis reveals the landscape of injury-induced MG state progression from the quiescence to proliferation. We identified the injury-induced MG proliferation via cxcl18b-defined the transitional states. Notably, the cxcl18b-defined MG transitional states recapitulate molecular features of retinal developmental programs. Additionally, we discovered that nos2b is crucial for MG entry into the proliferation via nitric oxides signaling. Cell-specific knockout of nos2b in cxcl18b+ MG significantly reduced the MG proliferation after injury. In conclusion, our findings revealed the novel molecular and cellular mechanisms underlying the transition of MG from quiescence to proliferation after the cone ablation in the zebrafish retina.

Short CV

Dr. He, a researcher at the Center for Brain Intelligence Excellence, Chinese Academy of Sciences. primarily focuses on the generation mechanisms of cell lineage diversity in the central nervous system of vertebrates. Representative achievements include: revealing transcriptional and post-transcriptional regulatory mechanisms governing neuronal type determination across the entire brain; systematically analyzing the lineage structure and developmental program of retinal cell types in neural circuit in zebrafish by combining single-cell multi-omics techniques with *in vivo* cell lineage tracing technology. He has published 16 papers as the first or corresponding author in international academic journals such as Science, Neuron, Journal of Neuroscience, PLOS Biology, Journal of Cell Biology, EMBO Reports, Development, and eLife.



Transforming nutrition into reactive oxygen species for tumor treatment

Peng Huang (黄鹏)

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Abstract

Glucose is one of the main energy sources that tumor cells rely on for survival. Glucose oxidase (GOx), an emerging antineoplastic enzyme, has aroused great research interest in metabolism modulation for antitumor therapy due to its inherent biocompatibility and biodegradability, and its unique catalytic properties against β-D-glucose. GOx can effectively catalyze the oxidation of glucose into gluconic acid and hydrogen peroxide. This process depletes oxygen levels, resulting in elevated acidity, hypoxia and oxidative stress in the tumor microenvironment. All of these changes can be readily harnessed to develop a multimodal synergistic cancer therapy by combining GOx with other therapeutic approaches. In this talk, we will highlight our recent efforts on systematic design and construction of functionally specific GOx-based nanomaterials and present representative paradigms for effectively treatment of cancer, through transforming tumor nutrition (Glucose) into reactive oxygen species.

Key words: Reactive oxygen species, glucose oxidase, glucose, antitumor therapy, nanomedicine.

Short CV

Prof. Peng Huang is a Distinguished Professor, Director of Department of Molecular Imaging, Chief of Laboratory of Evolutionary Theranostics, at the School of Biomedical Engineering, Shenzhen University, China. His research interests are including molecular imaging, nanomedicine and theranostics. He has published many papers in this area in top journals such as Nature Biomedical Engineering, Nature Nanotechnology, Nature Communications, Science Advances, Chemical Reviews, Chemical Society Reviews, Accounts of Chemical Research, Advanced Materials, ACS Nano, Angewandte Chemie International Edition, Journal of the American Chemical Society, etc. Starting from 2008, Dr. Huang has authored over 290 peer-reviewed papers, which have received a total citation of > 34,000 times and given him an H-index at 97. He has been selected as a Global Highly Cited Researcher in the field of Cross-Field by Clarivate for four consecutive years (2020-2023).



Mitochondrial dynamics and redox balance control macrophage cell fate

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Abstract

Macrophages are monocyte-derived innate immune cells that participate in regulating inflammation response and various pathologies. Macrophages are usually divided into two categories, pro-inflammatory (M1) and anti-inflammatory (M2) macrophages. However, the mechanism involved remains obscure. Here, we aim to investigate the role of mitochondrial dynamics and redox balance in regulating macrophage polarization. In LPS-treated macrophages, mitochondria show increased mass, shorten length and looser cristae, which indicated enhanced mitochondrial fission. We prove that mitochondria in M1 macrophages shift their function from ATP synthesis to ROS generation, and ROS modulates NFκB-dependent pro-inflammatory response. Dynamin-related protein 1 (Drp1) is a key GTPase that plays critical role in regulating mitochondrial fission. Our data show that Drp1-dependent mitochondrial fission promotes macrophages pro-inflammatory differentiation. Knockdown or inhibition of Drp1 alleviates LPS-induced macrophages pro-inflammatory differentiation and mice sepsis. As a key member of the STAT family, signal transducers and activators of transcription 2 (Stat2) is indispensable for IFN-mediated anti-viral and anti-tumor. We prove that Stat2 enhances LPS-induced mitochondrial fission through regulating Drp1 S616 phosphorylation. Knockdown of Stat2 blunts the increase of pro-inflammatory cytokines in LPS-stimulated macrophages. Therefore, these results suggested that Stat2-Drp1 mediated mitochondrial fission modulates the initiation of macrophage pro-inflammatory response. Moreover, we observe an obvious increase of mitochondrial fusion and decrease of ROS generation in IL4-activated M2 macrophages. Enhance mitochondrial fusion also promotes the expression of anti-inflammation cytokines, including Arg1, Fizz1 and IL-10. It has been reported that ROS dose influences the inflammatory course, and ROS reduction promotes macrophages anti-inflammatory response. Hence, we prove that mitochondrial fusion boosts anti-inflammatory response through inhibiting mtROS generation. In conclusion, the crosstalk of mitochondrial dynamics and redox signaling may control macrophages polarization. Increased mitochondrial fission and ROS generation facilitates M1 macrophages pro-inflammatory response, while enhanced mitochondrial fusion and decreased ROS promotes M2 macrophages anti-inflammatory response.

Key words: Redox balance, Inflammation, macrophage, mitochondrial dynamics

Short CV

Yu Weihua, born in February 1988, Ph.D., is director and associate professor of the Toxicology Department of the Fourth Military Medical University.



Mitochondrial superoxide stress response: implications for aging and health

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Abstract

Mitochondria play pivotal roles as both energy producers and signaling centers within cells, profoundly impacting metabolism and aging through intricate communication with the nucleus. The maintenance of nuclear envelope (NE) integrity is crucial for cellular function, yet the mechanisms underlying its preservation during aging remain elusive. Here, we demonstrate that inhibiting mitochondrial electron transport chain (ETC) activity preserves NE integrity in aging Caenorhabditis elegans. Reduced ETC activity triggers elevated levels of mitochondrial superoxide, inhibiting polyunsaturated fatty acid (PUFA) biosynthesis through SBP-1-mediated lipid metabolism regulation. The lipidomic analysis uncovers a widespread reduction in PUFA, leading to diminished lipid peroxidation in organisms with elevated mitochondrial superoxide. Dietary supplementation of PUFAs abolished the NE protection. Moreover, interventions targeting lipid peroxidation effectively preserve NE integrity in monkey Hutchinson-Gilford Progeria Syndrome (HGPS) models and human BJ senescent cells. Additionally, we introduce an open platform utilizing image-based artificial intelligence (AI) algorithms for quantitative assessment of NE morphology. Thus, our work unveils unexplored mitochondria-NE crosstalk and underscores lipid peroxidation's pivotal role in NE integrity regulation and cellular aging.

Key words: Mitochondrial Superoxide Stress, Lipid homeostasis, Membrane integrity, Aging

Short CV

Dr. Ye Tian earned her B.S. degree in Biotechnology from Beijing Normal University in 2005, followed by a Ph.D. degree in Biochemistry and Molecular Biology from a joint program of Beijing Normal University and the National Institute of Biological Sciences, Beijing in 2010. Her academic journey continued with postdoctoral training at the Salk Institute and the University of California, Berkeley from 2010 to 2016. In 2016, she assumed the role of Principal Investigator at the Institute of Genetics and Developmental Biology, Chinese Academy of Sciences, Beijing.

Her research focuses on the regulatory mechanisms of mitochondrial stress and aging, resulting in several achievements. These include the discovery that neuronal mitochondria can transmit "stress memory" across generations by increasing mitochondrial DNA copy numbers in germ cells, enhancing offspring stress resistance and lifespan. Additionally, her work identified various cross-tissue mitochondrial signal exchanges influencing overall metabolism and aging. Furthermore, she uncovered the role of mitochondrial metabolites in regulating aging through epigenetic factors, providing a theoretical foundation for targeting metabolites to mitigate aging.



The ER redoxtasis: from basic research to the intervention of aging and diseases

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Abstract

The unique oxidizing environment of the endoplasmic reticulum (ER) facilitates the oxidative folding of secretory and membrane proteins, which are rich in disulfide bonds. The endoplasmic reticulum (ER) oxidoreductin-1α (Ero1α) and protein disulfide isomerase (PDI) constitute the pivotal oxidative protein folding pathway in the ER. Our previous work has shown that oxidative protein folding fidelity and ER redox homeostasis (redoxtasis) are maintained by both the precise control of Ero1α oxidase activity and the division of labor between PDI family members. Deregulated Ero1α-PDI functions contribute to aging and various diseases including cancers, thrombosis and inflammation. Our recent work identified two small molecule compounds, from an FDA-approved drug library, as highly selective Ero1α-PDI inhibitor, which providing new strategies for combating diseases associated with ER redox dysregulation.

Key words: ER, redox, disulfide bond, aging, disease

Short CV

Prof. Lei Wang is a Principal Investigator at the National Laboratory of Biomacromolecules, Institute of Biophysics, Chinese Academy of Sciences. His research focuses on the ER homeostasis and human health, including the mechanism of oxidative protein folding and redoxtasis regulation in the ER, and their roles in aging and related diseases. He has published more than 40 research and review papers in peer-reviewed journals.



Regulation of plant development by peptides–receptor kinases–ROS signaling

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Abstract

In angiosperms, successful sexual reproduction relies on complicated communications between male and female organs. In the study of pollen-stigma interaction, we established a lock-and-key mechanism that before pollination FERONIA receptor kinase and its homolog ANJEA (FER–ANJ) perceives the RAPID ALKALINIZATION FACTOR peptides RALF23/33, inducing the generation of reactive oxygen species (ROS) in stigma papillary cells via a ROP2–RBOHD pathway; during pollination, the POLLEN COAT PROTEIN B-class peptides (PCP-Bs) compete with RALF23/33 for binding to FER–ANJ, leading to a reduction in stigmatic ROS and subsequently facilitating pollen hydration. This study underscores the precise regulation of plant development by receptor kinases that perceive and switch between different types of peptide ligands. In the study of pollen tube growth, we found that GPI-anchored proteins LLG2/3 act as chaperones to facilitate the trafficking of AUX/BUPS receptor kinases from the endoplasmic reticulum to the plasma membrane of the pollen tube tip region; wherein LLG2/3 serve as coreceptors of ANX/BUPS receptor kinases to perceive the RALF4/19 peptides and activate a RAC/ROPs–NADPH oxidases–ROS pathway, which assures the cell wall integrity during the fast growth of pollen tubes. Furthermore, we demonstrate that auxin regulate the production of ROS and nitric oxide (NO) through FER receptor kinase–NADPH oxidase signaling pathway. Interestingly, ROS and NO initiate oxidative modifications in TIR1$^{C140/516}$ and AFB2$^{C135/511}$, facilitating their subsequent nuclear import. The oxidized forms of TIR1$^{C140/516}$ and AFB2$^{C135/511}$ play a crucial role in enhancing the function of TIR1 and AFB2 as auxin receptor in transcriptional auxin responses. These studies shed light on the complex signaling networks and molecular mechanisms that drive plant development, offering fresh insights into how receptor kinases, peptide ligands, and oxidative signaling work together to regulate essential developmental processes in plants. <Liu C, et al. Science. 2021; 372:171; Lu B, et al. Mol Plant. 2024; 17:772; Feng H, et al. Mol Plant. 2019; 12:1612.>

Key words: CrRLK1L receptor kinase, RALF peptides, reactive oxygen species, oxidative modification, plant development

Short CV

Prof. Chao Li, head of the Botany Department at the School of Life Sciences, East China Normal University. She has received several prestigious awards, including the Wei-Zhiming Young Innovation Award (CSPB), the Rising Star Award (SSBMB), and Shanghai Eastern Scholar Elite Program. She earned her Ph.D. from the Institute of Genetics and Developmental Biology, CAS and completed postdoctoral research at the University of Massachusetts. Since 2016, he has led an independent research group focusing on peptides-receptor kinases and plant development, with key findings published in top journals such as Science, Mol Plant, eLife.



Reciprocal role of the Keap1-Nrf2 pathway in the self-renewal and differentiation of airway stem cells and tongue stem cells

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Abstract

Mutations in the Keap1-Nrf2 pathway occur in more than 30% and 10% of lung and head and neck squamous cell carcinomas, respectively. However, the role of the Keap1-Nrf2 pathway in the stem cells in the airway and oral cavity, where those cancers are frequently found, has yet to be discovered. Here, we report the reciprocal role of the Keap1-Nrf2 pathway in regulating airway and tongue stem cell self-renewal and differentiation. To facilitate airway stem cell research, we established the tracheal organoid system and developed the airway stem cell-specific lineage tracing system, which revealed that the Keap1-Nrf2 pathway promotes airway stem cell self-renewal. We also established the tongue organoid system and a chemical tongue injury-recovery mouse model. In contrast to the airway stem cells, the Keap1-Nrf2 pathway promotes tongue stem cell differentiation. Further studies to elucidate these tissue-specific differences are currently ongoing. These data indicate that the Keap1-Nrf2 pathway has differential regulatory roles in stem cells in each tissue, which should be considered in developing regenerative medicine by modulating the Keap1-Nrf2 pathway in each tissue.

Key words: Keap1-Nrf2 pathway, airway, tongue, self-renewal, differentiation, stem cells



Involvement of UVB/ROS-mediated signaling pathway in karyoptotic cell death

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Abstract

Intracellular organelles enclosed by membranes play critical roles in maintaining cellular homeostasis. The nucleus, the largest organelle in the cell, is composed of two lipid bilayers and houses genomic DNA, where genetic information is stored. To accommodate the extensive length of genomic DNA within the confined space of the nucleus, it must be tightly packaged and highly organized. However, the nucleus is subjected to expansion pressure due to this tightly packed DNA, and any disruption in the forces counteracting this pressure can lead to a loss of nuclear integrity and ultimately, cell death. While cancer cells often develop resistance to apoptotic cell death, they may remain vulnerable to alternative forms of regulated cell death (RCD). Understanding these additional mechanisms is crucial for developing new therapeutic strategies.

Karyoptosis is a recently proposed form of RCD characterized by nuclear shrinkage, cytoplasmic atrophy, and abnormal nuclear morphologies such as herniations, folds, crevices, fragments, and lobules. This process, distinct from apoptosis and autophagy, involves the excessive excretion of nuclear components and is associated with terminal degradation events in cells. However, the intrinsic factors, extrinsic stimuli, and molecular mechanisms that trigger karyoptosis remain largely unknown.

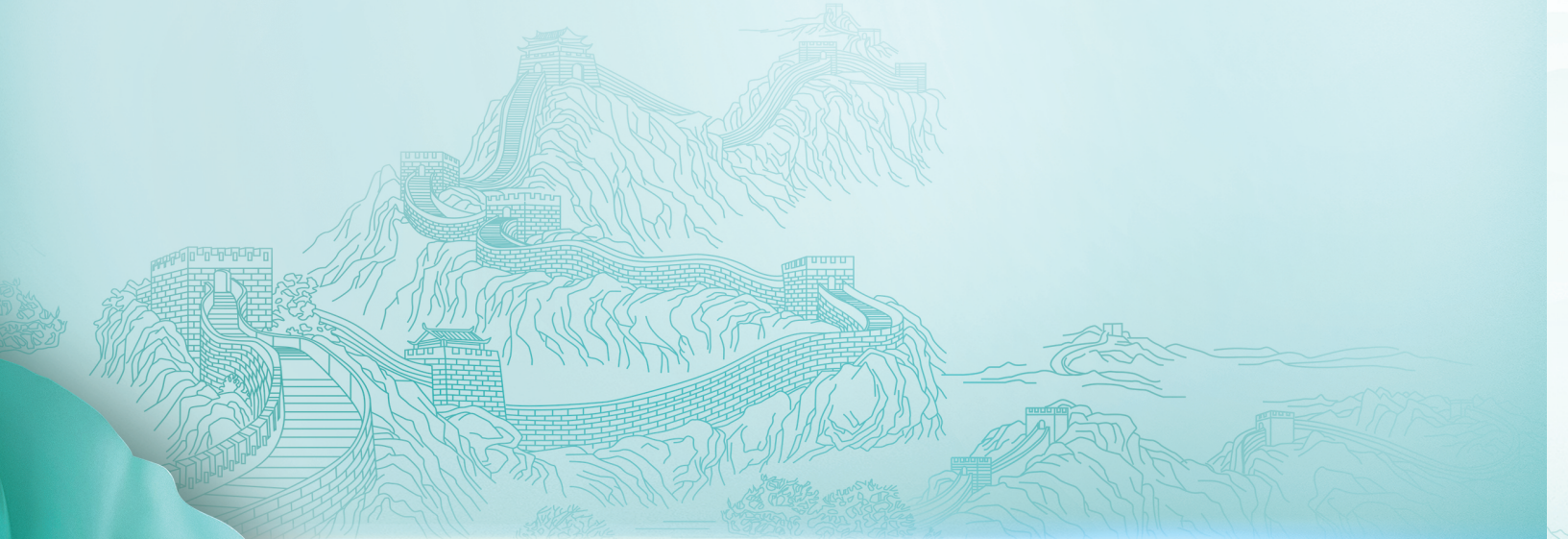
Our current research has uncovered that cyclic AMP response element-binding protein 3 (CREB3), traditionally recognized as an endoplasmic reticulum (ER)/Golgi-bound transcription factor, plays a pivotal role in this process. We found that CREB3-FL (full-length CREB3) is a type II membrane protein located at the inner nuclear membrane, where it undergoes cleavage by site-1 protease (S1P) and site-2 protease (S2P) to produce CREB3-CF (cleaved form). This cleavage event untethers CREB3-FL from the nuclear membrane, disrupting the balance between the outward expansion force of packed DNA and the inward force exerted by the nuclear membrane. As a result, the nuclear membrane undergoes explosive rupture, abnormal folding, and eventual herniation of nuclear DNA into the cytoplasm, leading to cell death.

Importantly, our findings suggest that karyoptosis can be modulated by external stimuli and by regulating the stability of CREB3. These insights reveal that targeting the regulatory mechanisms governing CREB3-CF accumulation and CREB3-FL cleavage could offer a novel therapeutic strategy to induce karyoptosis in cancer cells, providing a potential new avenue for cancer treatment.

Key words: CREB3, Nuclear membrane rupture, chromatin untethering, karyoptosis, regulated cell death

Symposium-13 (S13)

Redox and neural function & mental health





Chair: Lin Mei (梅林)

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Short CV

Lin Mei is a professor of Institute of Biomedical Engineering, Peking Union Medical College & Chinese Academy of Medical Sciences. He serves as principal investigator of State Key Laboratory of Advanced Medical Materials and Devices, and director of Tianjin Key Laboratory of Biomedical Materials. His research interests are focused on nanomedicine, molecular pharmaceutics and drug/gene delivery. He has published more than 180 SCI-indexed papers in such top journals as Science Translational Medicine, Nature Communications, and Science Advances, among which there have been 26 ESI-Highly Cited papers. He is also an associate editor of Smart Materials in Medicine and VIEW.



Flavin adenine dinucleotide metabolism and related neuromuscular disorders

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Abstract

FAD and the flavoproteins it supports are involved in a number of catabolic pathways which all converge on the mitochondrion.The re-oxidation of FADH2 from fat oxidation and branched anmino acid catalysis is mediated by the ETF-ETFQO system. Genetic defects in ETFA, ETFB and ETFDH result in multiple acyl-CoA dehydrogenation deficiency (MADD).On the other hand, disturbance of FAD homeostasis caused by genetic defect of riboflavin transporter (RFVT1-3), mitochondrial folate/FAD transporter (MFT) and FAD synthase (FADS) may lead to neuromuscular disorders biochemically mimicking MADD(MADD-like disorders）. Of note, recent reports showed that variant COASY gene codes Coenzyme A synthase and sertroline medication may also cause MADD-like phenotypes. Fortunately most patients with MADD or MADD-like disorders, except for MADD type1 and type2, are responsive to riboflavin. While nearly 200 flavoproteins have hitheto been identified, FAD homeostasis and re-oxidation of FADH2 may play an important role for cell metabolism and energy production. We advocate a collaborative study launched by clinical doctors and scientists in the field of redox biology and discover more potential disorders potentially related to FAD metabolism.

Short CV

- Professor and Chair of Department of Neurology and Neuromuscular Center in Qilu Hospital of Shandong University.
- President of Chinese Society of Neuromuscular Disorders
- President of China Alliance for Rare Diseases of Neurological Rare Diseases
- Vice president of Chinese Society of Neurology
- Broad member of AOMC(Asian and Oceanian Myology Center)



In-situ fluorescence imaging of brain disease-associated bioactive molecules

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Abstract

Brain diseases such as stroke and depression have a very high incidence and death and disability rates, bringing great mental and economic burdens to families and society. To effectively prevent and treat brain diseases, we must have a thorough understanding of the occurrence and development of brain diseases. However, at present, brain diseases have been a major challenge in the field of neuroscience due to their complex pathogenic factors, unclear etiology, and unknown pathogenesis. Existing findings suggest that bioactive molecules, including neurotransmitters, corresponding synthetic or hydrolytic enzymes, reactive oxygen radicals (ROS), reactive nitrogen radicals (RNS), and metal ions, play a crucial role in developing depression and stroke. To address the current bottleneck in detecting brain disease-related bioactive molecules in neurons in the living brain, we have developed a series of organic small molecule two-photon fluorescent probes that are stable, biocompatible, and fast-responding, and have realized the detection of ROS (hydroxyl radicals, superoxide anion radicals), neurotransmitter hydrolases (acetylcholinesterase), and metal ions (zinc ions), etc., in the cells of neurons in the living brain. Highly sensitive and specific two-photon fluorescence imaging analysis has systematically investigated the changes of disease-related bioactive molecules in mouse brain *in vivo* and preliminarily explored the related signaling pathways involved in the active molecules, which will provide important information and an ideal imaging probe for the study of molecular mechanisms related to the onset and development of depression and stroke.

Key words: Brain diseases, ROS, fluorescence imaging

Short CV

Ping Li obtained her doctorate degree from Shandong Normal University in 2008. Since 1998, she has been working at the College of Chemistry, Chemical Engineering and Materials Science, Shandong Normal University. Currently, she is a second-level professor, doctoral supervisor, and a member of the university's academic committee. She enjoys the special allowance of the State Council, is selected for the National Hundred, Thousand, and Ten Thousand Talent Program, is recognized as a young and middle-aged expert with outstanding contributions at the national level, is a specially appointed professor of Taishan Scholars, and serves as the leader of the innovation team under the "Changjiang Scholars and Innovative Research Teams in Universities" Program of the Ministry of Education. She is also a member of the 7th Committee of Shandong Provincial Committee of the China Zhi Gong Party, the principal of the Shandong Normal University Basic Committee of the China Zhi Gong Party, and a member of the Shandong Provincial Committee of the Chinese People's Political Consultative Conference.



Mechanisms of α7 nicotinic acetylcholine receptor in modulating inflammatory lung injury and infection

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Abstract

Supraphysiological concentrations of oxygen (hyperoxia) can compromise host defense, and increase susceptibility to bacterial and viral infections, causing ventilator-associated pneumonia (VAP). Compromised host defense and inflammatory lung injury are mediated, in part, by oxidative stress in the immune cells and high extracellular concentrations of HMGB1. Here, we report that agonists of α7 nicotinic acetylcholine receptor (α7nAChR), can significantly decreased animal mortality and markers of inflammatory injury in mice exposed to hyperoxia and subsequently infected with Pseudomonas aeruginosa. They can significantly decrease hyperoxia-induced extracellular HMGB1 accumulation and HMGB1-induced macrophage phagocytic dysfunction. Hyperoxia-compromised macrophage function was correlated with impaired mitochondrial membrane integrity, increased superoxide levels, and decreased manganese superoxide dismutase (MnSOD) activity. This compromised MnSOD activity is due to a significant increase in its level of glutathionylation. The α7nAChR agonists significantly decreases the levels of glutathionylated MnSOD, and restores MnSOD activity and mitochondrial membrane integrity. Overall, our results suggest that neuromodulation of lung inflammation plays a pivotal role in host defense.

Key words: Oxidative lung injury, pulmonary infection, neuromodulation, inflammatory reflex, nicotinic receptor

Short CV

After graduating from Beijing University School of Medicine, Lin pursued PhD at SUNY Stony Brook and trained as a NIH fellow at the Cold Spring Harbor Laboratory. She is a tenured full Professor at St. John's University College of Pharmacy and Health Sciences/Feinstein Institutes for Medical Research, Northwell Health System. She is the treasurer of the Society for Free Radical Research International and the Vice President for Finance and Advocacy of the Society for Redox Biology and Medicine. Her research focuses on the mechanisms underlying oxidative stress-induced lung diseases including cystic fibrosis, supplemental oxygen-induced acute inflammatory injury and secondary infection-induced pneumonia, and the role of neuromodulation in the pathogenesis of the disease.

Symposium-14 (S14)

Intelligence materials for precision redox intervention





Chair: Fangyuan Li (李方园)

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Short CV

Fangyuan Li is a Professor at the Shanghai Jiao Tong University School of Medicine. She is mainly engaged in research on the field of biomaterials, accumulating expertise in nanobiomaterials, molecular imaging, drug delivery, cancer and neurological disorders. In recent years, she has published numerous papers as corresponding/first author in academic journals such as Nature Nanotechnology、Nature Communications、Advanced Materials、Journal of the American Chemical Society、Angewandte Chemie International Edition、National Science Review、Nano Today. She has led projects funded by the National Science Fund for Excellent Young Scholars, the National Key Research and Development Program of China, and the Natural Science Foundation of Zhejiang Province. Prof. Li has been honored with the Outstanding Achievement Award for Female Scientists at the 2022 Biophysical Society Meeting, and recognition for provincial-level online first-class undergraduate courses. She is a secretary-general of the Materials Biology and Intelligent Diagnosis and Treatment Technology Branch of the Biophysical Society of China.

Selected Publications:

(1) An artificial protein modulator reprogramming neuronal protein functions，Nature Communications，2024, 15, 2039.

(2) Ligand-mediated magnetism-conversion nanoprobes for activatable ultra-high field magnetic resonance imaging, Angewandte Chemie International Edition, 2024, e202318948.

(3) Alpha-Synuclein Oligomers Driven T1-T2 Switchable Nanoprobes for Early and Accurate Diagnosis of Parkinson's Disease, Advanced Materials,2023, e2310404.

(4) Highly sensitive diagnosis of extracellular calcium ions associated brain diseases using Ca^{2+}-dependent T2-T1 switchable magnetic nanosensors, Advanced Functional Materials, 2023, 2313286.

(5) An immunomodulatory zinc-alum/ovalbumin nanovaccine boosts cancer metalloimmunotherapy through erythrocyte-assisted cascade immune activation, Advanced Science, 2023, 2307389.



Regulation and restoration of microenvironment homeostasis of intestinal diseases based on nanotechnology

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Abstract

The disorder of the intestinal microenvironment often goes with a series of intestine problems, such as functional diarrhea, inflammatory bowel disease (IBD), colon cancer, etc. A long period of disorder in the microenvironment will precipitate the development of significant chronic diseases, potentially posing a grave threat to life. Nanotechnology emerges as a promising avenue for precisely regulating and restoring homeostasis to the intestinal microenvironment, specifically targeting the remediation of compromised intestinal ecology, thereby ushering in novel treatments on intestinal diseases.

IBD is a chronic, recurrent inflammatory disorder of the digestive system. Traditional clinical medications are often limited to alleviating symptoms, yet they are prone to disease recurrence. Long-term treatment with these drugs can further complicate matters by inducing drug resistance, elevating the risk of infections, and even cancer. Recognizing these limitations, our team has developed a series of bioactive nanomaterials that aimed at transcending the boundaries of conventional drug therapy and promoting IBD treatment. The overabundance of reactive oxygen species (ROS) constitutes a pivotal pathological hallmark of IBD. To address this, we formulated an oral nano-antioxidant, SeNG, which targets the inflammatory cells in IBD. SeNG not only directly clears H_2O_2 but also upregulates the Nrf2/HO-1 signaling pathway, and then mitigates intracellular ROS level. Furthermore, we integrated a probiotic membrane into this antioxidant nanostructure, yielding another bionic-nanosystem, SeM@E.M. This system synergistically promotes cellular REDOX equilibrium, modulates the intestinal microbiota, influences the lamina propria immune response, and restores intestinal homeostasis. Recognizing the profound influence of intestinal symbiotic bacteria in the IBD microenvironment, we extended our strategy by precisely inhibiting the respiratory and energy metabolism of IBD-associated enterobacteriaceae using tungsten trioxide nanoparticles, which enhances the therapeutic efficacy against IBD.

Colorectal cancer (CRC), a highly aggressive intestinal malignancy, stands as the third most prevalent cancer worldwide and the second leading cause of cancer-related mortality. Leveraging the physiological features of intestinal tract, our team has developed an oral tumor vaccine that harnesses biologically modified Escherichia coli (E. coli) as a vector for delivering outer membrane vesicles (OMV) vaccine. These engineered bacteria produce OMVs, which express specific tumor antigens directly within the intestinal tract in response to exogenous signals. Subsequently, the OMVs infiltrate the lamina propria, prompting the maturation of

dendritic cells, which subsequently activate T cells to specifically eliminate tumor cells, thereby reinforcing the host's immune memory. In mouse models of subcutaneous colon tumors and lung metastases, this approach significantly suppressed tumor growth, demonstrating its promising potential.

Key words: intestinal disease, microenvironment, nanotechnology, intestinal microbiota

Short CV

Dr. Nie has a long-standing interest in nanomedicine, biomaterials, cancer biology, blood physiology and the pathophysiology of human disorders involving dysregulation of redox balance and metal metabolism. Currently, his main interests are in nanomedicines, nanovaccines and the design of biology-inspired materials to overcome the current barriers in tumor therapy. His group is working toward controlling the chemical properties of multi-functional nanoparticles to allow specific targeting and regulation of tumor cells and their microenvironment.

Dr. Nie's most recent research activity has generated a collection of interdisciplinary works in the fields of nanobiology, nanomedicine and blood physiology, comprising over 330 papers published in Nature Biotechnology, Nature Nanotechnology (2), Nature Biomedical Engineering (3), Nature Communications (5), Nature Protocols (2), Nature Reviews Cancer, Nature Reviews Materials, Science Translational Medicine (2), Cell Chemical Biology, Nature Materials, Acc Chem Res, Adv Mater, Angew Chem, Blood, Biomaterials, Br J Haematol, Cancer Res, Circulation Res, JACS, JBC, JCI, Mol Cancer Ther, Nano Letters and Trends Biotech. Additionally, he has filed over 50 patents on novel nanomedicines, over 30 of which have been granted. Three patents on antitumor drug development have been transferred to 3 biotechnology firms for further development.

Dr Nie was honored as "Fellow of FRSC" (2023), "AIMBE College of Fellows Class of 2022", "The 1st Class, Science and Technology Award, Beijing Municipal Science and Technology Commission", "Honorary Professor of University of Queensland, Brisbane, Australia", "The 2nd Class, Science and Technology Award, Chinese Pharmaceutical Association, China", "Chief Scientist, National Basic Research Program, Ministry of Science and Technology (MoST), China", "Yiling Pharma. Co. Young Scientist Award, Chinese Pharmaceutical Association, China", "Chief Scientist, National Basic Research Program, Ministry of Science and Technology (MoST), China", "Newton Advanced Scholar, Academy of Medical Sciences, UK", "National Distinguished Young Scholar Award, Natural Science Foundation of China", "Associated Full Member, Methodist Hospital Research Institute, Huston, US", "Hundred Talent Program Scholar, Chinese Academy of Sciences, China". He has been appointed as Associate editors or advisory editor for many prestigious academic journals including Nano Letters, Life Medicine, Nano Today, Exploration, Fundamental Research, Science China Chemistry, Materials Today Chemistry, Advanced Drug Development Reviews.



Molecular mechanisms of selenium intervention in metabolic diseases through regulation of redox homeostasis

Jun Zhou (周军)

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Abstract

Selenium, an essential trace element, plays an important role in the regulation of redox homeostasis and exerts a variety of important biological functions through selenoproteins. Selenium has both antioxidant and pro-oxidant functions, and is closely related to the onset and progression of a variety of diseases, including cardiovascular disease, diabetes, cataract and cancer. This paper describes relevant advances in our laboratory on selenium redox biology and its relationship with metabolic diseases, mainly including (1) the free radical mechanism of the two sides of selenium nutrient and selenium toxicity; (2) the molecular mechanism underlying selenium intervention in metabolic diseases, such as cardiovascular disease, cataract, diabetes, and cancer; (3) the biological functions and molecular mechanisms of selenoprotein S, selenoprotein F, selenoprotein K, and selenoprotein T.

Key words: selenium, selenoprotein, redox homeostasis, metabolic diseases

Short CV

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Education:

2010-2011 Postdoc, Oklahoma of University Health Sciences Center, USA
2005-2009 Ph.D., biochemistry and molecular biology, Huazhong University of Science and Technology, China
2000-2003 M.S., inorganic chemistry, Huazhong University of Science and Technology, China
1996-2000 B.S., applied chemistry, Huazhong University of Science and Technology, China

Professional Experience:

2016- Professor, School of Chemistry and Chemical Engineering, Huazhong University of Science and Technology, China
2010-2016 Associate Professor, School of Chemistry and Chemical Engineering, Huazhong University of Science and Technology, China
2010-2011 Research Scholar, Department of Medicine, University of Oklahoma Health Sciences Center, USA
2005-2010 Instructor, School of Chemistry and Chemical Engineering, Huazhong University of Science and Technology, China
2003-2005 Teaching assistant, School of Chemistry and Chemical Engineering, Huazhong University of Science and Technology, China

Research Areas:

1. The biological functions of selenium/selenoproteins and their roles in health and disease.
2. Molecular pathological mechanisms and therapeutics in dysregulation of glucose and lipid metabolism.

Professional Activities:

Memberships to professional organizations

2018- Member of the Standing Committee of the Biological Trace Elements Branch of the Chinese Biophysical Society
2024- Member of Specialized Committee on Biological Effects of Reactive Oxygen Species, Chinese Society of Environmental Mutagens
2022- Secretary General, Wuhan Academy of Trace Elements and Health
2022- Premium Member of Chinese Chemical Society



Next-generation RNA sequencing-based deep-learning model to predict chemoresistance in high-grade serous ovarian carcinoma

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Abstract

To fulfill precision cancer medicine in ovarian cancer, precise prediction of chemoresistance and subsequent recurrence is the first step. So we aimed to develop the next-generation RNA sequencing-based deep-learning model predicting chemoresistance risk in high- grade serous ovarian carcinoma (HGSOC).

We perfomed the next-generation RNA sequencing using fresh-frozen chemotherapy-naïve primary ovarian cancer tissues from HGSOC patients (n=86). Patients were randomly divided into training and test sets with a 2:1 ratio. In model development phase, transcriptomic data of both training set and HGSOC patients (n=208) from The Cancer Genome Atlas database were used. Using genes selected by differential gene expression analysis, we constructed and trained a deep neural network (DNN). Multiple DNN models were combined to build average ensemble models, which were further validated using the test set (validation phase). We assessed the predictive performance of the developed models based on the area under the receiver operating characteristic curve (AUC).

All patients in the study population received platinum-based combination chemotherapy: 14 and 72 were identified as chemoresistant and chemosensitive, respectively. Based on the differential gene expression between these two groups, we have identified 31 genes using two distinct methods. Machine learning algorithms were applied to develop and train DNNs of the 31 genes. Then, the five-fold average ensemble models were developed. Among different ensemble models, the chosen model demonstrated high accuracy in predicting chemoresistant cases (AUC, 0.85).

We successfully developed an RNA sequencing-based, deep-learning model to predict chemoresistance risk after first-line platinum-based chemotherapy in HGSOC. These newly developed models would help the individualized management of HGSOC patients. Incorporating targeted agents into the primary treatment and more intensive surveillance might be considered for patients at high-risk of developing chemoresistance.

Key words: ovarian cancer; high-grade serous carcinoma; prognosis; chemoresistance; deep learning; model.

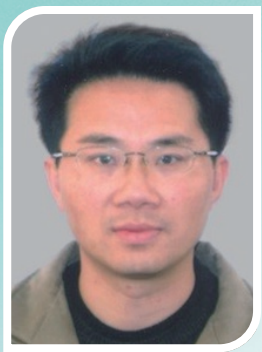
Short CV

Prof. Yong Sang Song is Professor Emeritus at Seoul National University, College of Medicine, and currently the Director of the Gynecologic Cancer Center at Myungji Hospital in Republic of Korea. He earned his MD and PhD from Seoul National University in 1983 and 1994, respectively, and served as a professor there from 1994 to 2023. His research focuses on the molecular mechanisms of chemoresistance in gynecologic cancers, particularly ovarian cancer. He has published over 400 papers in SCI journals and serves on the editorial boards of several scientific journals. Prof. Song has received numerous awards for his contributions to cancer research and prevention.

Symposium-15 (S15)

Lifestyle and redox regulation





Chair: Cheng-Gang Zou (邹成钢)

Yunnan University, China

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Short CV

Professor Cheng-Gang Zou graduated from Sichuan University with a bachelor's degree, Kunming Medical College with a master's degree, and the University of New England (UNE) with a doctor's degree. After completing postdoctoral training at the University of Nebraska-Lincoln (UNL), he moved to Yunnan University as a Professor in 2004 and now is Donglu Distinguished Professor of Yunnan University. He is currently the deputy director of State Key Laboratory for Conservation and Utilization of Biological Resources, Yunnan University.

Dr. Zou' research interests focus around the mechanism of microbe-host interaction and the role of metabolic pathways in aging. He published more than 50 scientific papers in peer-reviewed journals.



Surplus fatty acid synthesis increases oxidative stress in adipocytes and lnduces lipodystrophy

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Abstract

Adipocytes are the primary sites for fatty acid storage, but the synthesis rate of fatty acids is very low. The physiological significance of this phenomenon remains unclear. Here, we show that surplus fatty acid synthesis in adipocytes induces necroptosis and lipodystrophy. Transcriptional activation of FASN elevates fatty acid synthesis, but decreases NADPH level and increases ROS production, which ultimately leads to adipocyte necroptosis. We identify MED20, a subunit of the Mediator complex, as a negative regulator of FASN transcription. Adipocyte-specific male Med20 knockout mice progressively develop lipodystrophy, which is reversed by scavenging ROS. Further, in a murine model of HIV-associated lipodystrophy and a human patient with acquired lipodystrophy, ROS neutralization significantly improves metabolic disorders, indicating a causal role of ROS in disease onset. Our study well explains the low fatty acid synthesis rate in adipocytes, and sheds light on the management of acquired lipodystrophy.

Key words: fatty acid, oxidative stress, adipocytes, lipodystrophy, necroptosis, MED20

Short CV

Professor, Institute of Metabolism and Integrative Biology, Fudan University.

Research Interests

The focus of Zhao laboratory is to understand the underlying mechanism of these metabolic disorders including obesity, diabetes and fatty liver. They focus on: the physiological function of palmitoyl acyltransferases in regulating metabolism; identification of the key factors in adipogenesis and onset of obesity.



Effects of Hyperbaric Oxygen Intervention on Oxidative Stress in the Body after High-Intensity Interval Training

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Abstract

Purpose: This study aims to explore the impact of HBO intervention on oxidative stress levels following HIIT. Additionally, it seeks to investigate the corresponding mechanisms through the analysis of blood biochemical markers and metabolomics.

Methods: This study recruited 20 healthy male university students who are sports enthusiasts and employed a randomized controlled trial design. The participants were randomly divided into a control group (CON, n=10) and a hyperbaric oxygen group (HBO, n=10). Both groups followed the same exercise training program, with the CON group undergoing natural recovery after training and the HBO group undergoing hyperbaric oxygen recovery post-training. Blood biochemical markers and metabolomics data were collected at different time points before and after the experiment. Results: After the experiment, the SOD levels decreased and MDA levels increased in the CON group, while in the HBO group, SOD levels increased and MDA levels decreased. In terms of metabolomics, significant changes were observed in the metabolic pathways of arachidonic acid metabolism and oxidative phosphorylation. Enriched metabolites in these pathways included arachidonic acid, prostaglandin D2, and leukotriene D4.Conclusions:The HIIT training can induce a certain level of oxidative stress in the body, and HBO may improve oxidative stress levels to some extent, affecting arachidonic acid metabolism and oxidative phosphorylation, thereby reducing oxidative damage and promoting tissue repair.

Key words: hyperbaric oxygen; high-intensity interval training; oxidative stress; metabolomics

Short CV

Professor Wu Hao, Ph.D. in Exercise Physiology, doctoral supervisor. At present, Professor Wu Hao holds the following positions:Director of a key laboratory under the General Administration of Sport of China; Olympic technology expert at the General Administration of Sport of China; Distinguished researcher at the National Institute of Sport Science; Vice President and Secretary-General of the Hypoxia and Health Branch of the Chinese Biophysical Society; Executive Committee Member of the Exercise Physiology and Biochemistry

Branch of the Chinese Society of Sports Science; Vice Chairman of the Beijing Society of Sports Science; Expert Committee Member of the Beijing Health Security Association; Registered health manager; Director of a key laboratory in Beijing; Leader of the academic innovation team for "Strong Teaching of Talents" in Beijing; Deputy Director of the National Sports Industry Research Base; Founder and Curator of the Xuan Culture Museum; Expert Consultant Committee Member of the Beijing Community Sports Association; Co-founder of the Xi'an Rural Development Foundation; Secretary-General of the Collaborative Innovation Center for Sports, Fitness, and Leisure Development in the Beijing-Tianjin-Hebei region; Executive Vice Chairman of the Preservation Association of the Eight Temples outside the Summer Resort in China; National Level I Coach and Referee in Kabaddi.

Research focus on monitoring athletic performance, nutrition, and recovery. Engaged in Olympic research endeavors multiple times, such as serving as the head of the national team's research team during the preparation for the 2008 Beijing Olympics and the 2022 Beijing Winter Olympics, leading to Olympic gold medals and historic breakthroughs. Recognized with accolades including the "Outstanding Individual Contribution to the Beijing Olympics" from the General Administration of Sport of China, the Chinese Olympic Committee, the "Beijing Olympic Merit Award," the "Beijing May 1st Labor Medal," and titles like "Advanced Worker of Beijing."

Experience in teaching undergraduate, master's, and doctoral courses at the university level, covering subjects like "Sports Nutrition," "Specialized Courses in Exercise Physiology," "Youth Physical Fitness Training and Nutritional Recovery," and bilingual courses like "Health Promotion and Weight Control." Supervised and trained over 60 master's and doctoral students. Undertaken and completed various projects including key research and development projects from the Ministry of Science and Technology. Published 25 international SCI papers, authored or co-authored nine books on topics like "Sports Nutrition," "Health Promotion and Weight Control," "Cryotherapy and Exploration of Exercise Capacity," "Kayaking Sports," "Exploration of High-altitude Training in Chinese Rowing Projects," "Vegetarian Sports Nutrition," and "Safe and Effective Exercise for Overweight Adolescents." Holds 1 international invention patent in sports nutrition, 3 national invention patents, 1 utility model patent, and has published 189 papers domestically and internationally.



The role of ileal mucosa -associated microbiota in the patients with Crohn's disease

Osamu Handa

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Abstract

The intestinal mucus has multiple function of which it protects intestinal epithelial cells from various stimuli in intestinal lumen. Previously we found that aspirin induces small intestinal mucosal injury via superoxide production in mitochondria of small intestinal epithelial cells in vitro <Fukui A. et al. Am J Physiol Gastrointest Liver Physiol 2012. 303: G927-936>. We also found in an *in vivo* mice model that the presence of intestinal mucus significantly reduces the aspirin -induced small intestinal mucosal injury < Suyama Y. et al. Biochem Biophys Res Commun 2018. 498: 228-233>. Although the intestinal mucus itself plays protective role in healthy individuals, however, the microbiota in the intestinal mucus (mucosa -associated microbiota: MAM) plays important role in the pathogenesis of Crohn's disease (CD). In this study, we retrospectively examined the differences in the ileal MAM between CD patients and healthy controls and investigated the factors affecting MAM in CD patients to clarify potential therapeutic targets. As a result, CD patients had significantly reduced α-diversity in the ileum and a difference in β-diversity. The abundance of butyate-producing bacteria in the ileal MAM was significantly lower in CD patients with a history of abdominal surgery than in those without <Handa O. et al. Redox Rep 2023. 28: 2241615>, suggesting the important role of MAM in CD pathophysiology. Because butyric acid is a major energy source in the intestinal epithelium, its metabolism via β-oxidation increases oxygen consumption in epithelial cells, reducing oxygen concentration in the intestinal lumen and increasing the abundance of obligate anaerobic bacteria. The suppression of obligate anaerobes in CD patients caused an overgrowth of facultative anaerobes.

Key words: Crohn's disease, Mucosa -associated microbiota, butyrate, oxygen

Short CV

Osamu Handa is Associate Professor of Department of Gastroenterology and Hepatology, Kawasaki Medical School. He received his MD in 1994, and obtained his PhD in gastroenterology in 2003. He moved to Department of Gastroenterology and Hepatology, Kawasaki Medical School in 2019.

His clinical /basic research field is the oxidative stress in pathophysiology of gastrointestinal diseases. His current interests are (1) inflammatory bowel disease, (2) intestinal mucus, and (3) Helicobacter pylori and gastric carcinogenesis.

Flash Talk-1 (FT-1)



Chair: Suhua Wang (王素华)

Guangdong University of Petrochemical Technology, China

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Ph.D. from the Hong Kong University of Science and Technology and JSPS postdoc fellow. Research interests include basic theories and methods of analytical and bioanalytical chemistry, analysis of environmental pollutants and and detection of free radicals/reactive oxygen/nitrogen species related to life processes and environmental toxicology induction, such as hyroxyl radicals, nitric oxide, and NO_2. He has published over 140 relevant papers in journals including J. Am. Chem. Soc., Anal. Chem. , and Sensors Actuat. B. Chem., and etc.



From target identification to early-stage therapeutic discovery: leveraging in vivo preclinical models

Chair: Ju Cui (崔菊)

Beijing Institute of Geriatrics, National Health Commission, China

Email: cuiju4366@bjhmoh.cn

Short CV

Academic Qualifications

Ph.D. Department of Biochemistry, LKS Faculty of Medicine, The University of Hong Kong, Hong Kong, China. Feb. 2011

M.Phil. in Biotechnology, Department of Biology, The Chinese University of Hong Kong, Hong Kong, China. Dec. 2007

B.Sc. in Biotechnology, National Teaching Bases for Life Science and Biotechnology, Department of Biotechnology, Zhejiang University, Hangzhou, China. Jun. 2005

Academic Positions

Professor, The Key Laboratory of Geriatrics, Beijing Institute of Geriatrics, Beijing Hospital/National Center of Gerontology of National Health Commission, Beijing, China (Jun. 2022-present)

Research Interests

1. Molecular mechanisms and interventions of aging and aging related disease;

2. Cohort study of factors influencing health

Flash Talk-2 (FT-2)



Chair: Yan An (安艳)

Department of Toxicology, School of Public Health

Suzhou Medical College of Soochow University, China

Email: dranyan@126.com

Education

2005	PhD	Environmental Toxicology	Nihon University College of Pharmacy With Prof. Kenzo Yamanaka
2001	PhD	Radiomedicine	Jilin University Norman Bethune Medical College with Prof. Zenglin Gao
1996	MS	Industrial Hygiene and Occupational Diseases	China Institute for Radiation Protection With Prof. Rusong Chen
1993	B.S.Med	Preventive Medicine	Shanxi Medical University

Research statement

I am a toxicologist with a focus on Mechanism and Prevention of Xenobiotics Poisoning, Biochemical and Molecular Toxicology, and The Mechanism of Chemical Carcinogenesis on Redox Stress. I have been conducting the molecular mechanisms of the toxicities of environmental chemicals. Using cell lines and zebrafish embryos as model systems, and dissect the functions of signal pathways. I have raised the hypothesis that the metabolic process associated with the methylation of inorganic arsenicals, the subsequent metabolic sulfurization, and the chemical properties of their intermediate active metabolites play an important role in arsenic-induced Redox-stress Response Capacity in arsenic-induced cell transformation.

Major focuses of my current work are: (1) Regulative role of Redox-stress Response Capacity in arsenic-induced human cell transformation. (2) Environmentally relevant concentrations of chemicals induced developmental toxicity and Redox responses.

Representative Works

1. 'Environmental standard limit concentration' arsenic exposure is associated with anxiety, depression and autism-like changes in early-life stage zebrafish. Journal of Hazardous Material. 2024,469:133953.

2. A Bayesian Benchmark Concentration Analysis for Urinary Fluoride and Intelligence in Adults in Guizhou, China. Science of The Total Environment. 2024: 925: 171326.

3. Reductive Stress Induced by NRF2/G6PD through Glucose Metabolic Reprogramming Promotes to Malignant Transformation in Arsenite-Exposed Human Keratinocytes. Science of The Total Environment. 2023: 896: 165207.

4. Sustained expression of NRF2 and its target genes induces dysregulation of cellular proliferation and apoptosis is associated with arsenite-induced malignant transformation of human bronchial epithelial cells. Science of The Total Environment. 2021, 756: 143840.



Chair: Jinchuan Hu (胡晋川)

Fudan University, China

Email: hujinchuan@fudan.edu.cn

Short CV

Education

2004, Peking University, B.S.

2012, Institute of Microbiology, Chinese Academy of Science, Ph.D.

Work experience

2012-2017, University of North Carolina at Chapel Hill, Postdoc Associate,

2018- , Fudan University, Principal Investigator

Research Interests

1. The roles of DNA oxidative damage in cellular response to oxidative stress, especially how transcription is regulated by DNA oxidative damage during inflammation and other processes.

2. Molecular mechanisms of transcription-coupled nucleotide excision repair which can remove transcription-blocking lesions including oxidative damage.

Representative works

1. Jiao An#, Mengdie Yin#, Jiayong Yin#, ..., Maoxiang Qian*, Jinchuan Hu*. Genome-wide analysis of 8-oxo-7,8-dihydro-2'-deoxyguanosine at single-nucleotide resolution unveils reduced occurrence of oxidative damage at G-quadruplex sites. Nucleic Acids Research, 2021, 49(21): 12252-12267.

2. Jiao An#, Mengdie Yin#, Jinchuan Hu*. G-quadruplex and 8-oxo-7,8-dihydroguanine across the genome: methodologies and crosstalk. Genome Instability & Disease, 2022, 3: 241–254.

3. Liudan Jiang#, Jiayong Yin#, Maoxiang Qian#, ..., Jinchuan Hu*, Honghui Ma*, Yi-Han Chen*. UdgX-Mediated Uracil Sequencing at Single-Nucleotide Resolution. Journal of the American Chemical Society, 2022, 144(3): 1323–1331.

4. Yongchang Zhu#, Xiping Zhang#, Meng Gao#, ..., Jinchuan Hu*. Coordination of transcription-coupled repair and repair-independent release of lesion-stalled RNA polymerase II. Nature Communications, 2024, 15(1): 7089.

5. Yongchang Zhu#, Yuanqing Tan#, Lin Li#, ..., Maoxiang Qian*, Jinchuan Hu*. Genome-wide mapping of protein–DNA damage interaction by PADD-seq. Nucleic Acids Research, 2023, 51(6): e32.

Home page:

https://ibs.fudan.edu.cn/ibsen/05/a5/c39095a460197/page.htm



Chair: Yan Zhao (赵燕)

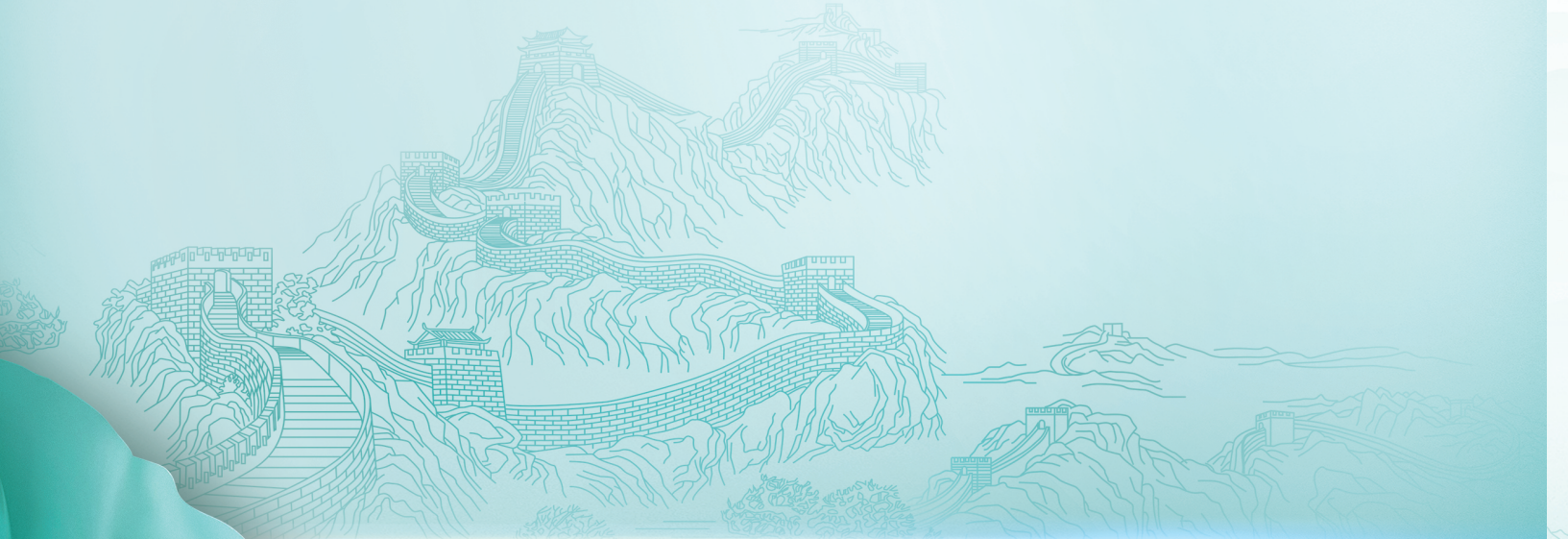
Harbin Institute of Technology (Weihai), China

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Short CV

Dr. Yan Zhao is a professor at the Department of Bioengineering, College of Marine Science and Technology, Harbin Institute of Technology (Weihai). She received B.S degree from University of Science and Technology of China, and Ph.D. degree from University of Kentucky. Her current research interest includes nutrition and gene interaction, bioactive compounds in intervention of aging and aging-related diseases.

Flash Talk-3 (FT-3)





Chair: Julia Li Zhong (钟莉)

Chongqing University, China

Email: jlzhonge@cqu.edu.cn

Short CV

Julia Li Zhong (Professor, PhD & MD)

Head of Experimental Dermatology of Chongqing, Chinese Society of Free Radical Biology and Medicine; Chinese Society of Photobiology;

E-mail: jlzhong@cqu.edu.cn; Website: Bioengineering.cqu.edu.cn.

Dr. Julia Li Zhong graduated from Clinical medicine (West China University of Medicine, Sichuan University; Ph.D. in the University of Bath, Photobiology-Pharmacology. Postdoctoral research at Kings College of London & University of London. University of Bath as RA2, 2008; then Chongqing University (Sept. 2009) to carry out teaching & research work. She has presided 4 NSFC(82373501, 2024.1-2027.12), related to UVA effects. & 2023YFC2508200: the National Key Research and Development Program of China.

She has published more than 60 papers in journals such as《Free Radical Biology &Medicine》; 《Oxid Med Cell Longev.》, etc. Moreover, she has joint applied and obtained UVA-LED patents of inventions. She also joint C Pourzand host SKIN@Bath Symposium, 2019, 2023 (China joint) &2024 Bath, UK;

【Research Interesting】

Photobiology. To study UVA radiation human skin cells and mouse model; Skin care (beauty products) & SPA medicine; UVA therapy and drug release: Iron chelation therapy. Skin diseases such as psoriasis and cancer related research etc.

【Publications】

1. Wang M, et al. Zhong JL. Bach2 regulates autophagy to modulate UVA-induced photoaging in skin fibroblasts. Free Radical Biology and Medicine, April, 2021. JCR/Zone2

2. Karisma VW, Wu W, et al Pourzand C, Zhong JL. UVA-Triggered Drug Release and Photo-Protection of Skin. Front Cell Dev Biol. 2021 Feb 11;9:598717.

3. Huang X, Nisar MF, et al, Zhong JL. UV-responsive AKBA@ZnO nanoparticles potential for polymorphous light eruption protection and therapy. Mater Sci Eng C Mater Biol Appl. 2020 Feb;107:110254.



Chair: Junmin Zhang (张军民)

School of Pharmacy & State Key Laboratory of Applied Organic Chemistry, Lanzhou University, Lanzhou 730000, China

Email: zhangjunmin@lzu.edu.cn

Short CV

Dr. Junmin Zhang is a Professor in the School of Pharmacy at Lanzhou University. He completed his Ph.D. in Medicinal Chemistry and Biology in 2017 from Lanzhou University, following which he pursued postdoctoral research at the State Key Laboratory of Quality Research in Chinese Medicine, Macau University of Science and Technology, Macau (S.A.R.), China. Dr. Zhang is a member of the Chinese Pharmacological Society and Chinese Chemical Society. His research interests lie in the discovery of small molecules that can target cellular redox systems or protein kinases, and their potential therapeutic applications, along with elucidation of their pharmacodynamics and pharmacokinetic mechanisms. Over the past 5 years, he has authored more than 30 papers in reputable Journals such as Trends Pharmacol Sci, Pharmacol Ther, Pharmacol Res, Med Res Rev, J Med Chem, Free Radical Biol Med, and others, either as the first author or corresponding author. His scholarly work has garnered over 1000 citations and includes 2 highly cited papers in the Essential Science Indicators (ESI). Additionally, Dr. Zhang has secured 2 authorized patents, successfully commercialized one, and obtained a registered software copyright. He has led and contributed to several national and provincial scientific research projects. Dr. Zhang's contributions have been recognized with accolades including a first prize for Military Scientific and Technological Progress and two second prizes for Scientific and Technological Progress in Gansu Province. Notably, he was selected for the 2019 International (Overseas) Exchange Program of the National Postdoctoral Management Committee of the Ministry of Human Resources and Social Security, as part of the "Macao Young Scholars Program."



Chair: Kuei-Hung Lai

Graduate Institute of Pharmacognosy, Taipei Medical University, Taiwan, China

Email: kueihunglai@tmu.edu.tw

Short CV

Kuei-Hung Lai is an Associate Professor in the Graduate Institute of Pharmacognosy, Taipei Medical University. After earning a B.S. in Life Science from National Chung Cheng University in 2010, he pursued Ph.D. degree in Pharmacognosy at Uppsala University, Sweden, completing in 2017. Following this, he gained experience in natural products chemistry and analytical chemistry at the National Museum of Marine Biology and Aquarium and the Chang Gung University of Science and Technology. Since 2020, he has been teaching at Taipei Medical University.

With a diverse research training and teaching background in pharmacognosy, he embraces different cultural perspectives to convey scientific knowledge and develop valuable scientific and technological research. Over four years at Taipei Medical University, he and the research team have focused on exploring lead compounds from marine and terrestrial natural products, primarily identifying and evaluating active ingredients with potential therapeutic effects against inflammatory diseases. The team has published 61 SCI papers since 2016, with 832 citations in Scopus and an h-index of 16.

The team also specializes in coral aquaculture, attempting to combine the drug development supply chain by utilizing characteristics of biological competition and defense to stabilize the growth conditions for mass-producing active components from corals sustainably. In recent years, the team has incorporated big data and databases to establish a mass spectrometry analysis application platform, building information on the active metabolic components of traditional Chinese medicine (TCM) and dedicating efforts to evidence-based research in TCM. The team is committed to the value of translational research, having developed a "TCM bioactive metabolite molecular network platform" to identify the active metabolite characteristics of TCM, influencing the selection of key components in Taiwan's TCM guidelines.

In recent years, his academic awards and scholarships include the Ta-You Wu Memorial Award from the National Science and Technology Council in 2024, the Young Scholar Award from the Society of Chinese Natural Medicine in 2023, and the 20th National Innovation Award from the Research Center for Biotechnology and Medicine Policy in 2023.

Poster Location

Venue: The ballroom A	
FT-01	A Chelator-linked Trityl Probe Enabling Highly Specific, Sensitive and Quantitative Detection of Cu(I) by EPR Spectroscopy Yan Wang 王炎 (Tianjin Medical University, China)
FT-02	Deciphering the RSS code in cellular senescence Wong Nai-Kei (Shantou University Medical College, China)
FT-03	Detection and evaluation of novel oxidizing substances in sodium hypochlorite using Trolox Kishimoto Ayuta (Shibaura Institute Of Technology, Japan)
FT-04	High sensitive LC-MS/MS method for determining malondialdehyde in biological sample using thiobarbituric derivatization Ujihara Miyu (Kyoto University, Japan)
FT-05	A novel protein CYTB-187AA encoded by the mitochondrial gene CYTB modulates mammalian early development Zhijuan Hu 胡志娟 (Guangzhou Institutes of Biomedicine and Health, Chinese Academy of Sciences, China)
FT-06	A novel redox gene atad-3 identified by whole genome RNAi screening in Caenorhabditis elegans Jiao Meng 孟姣 (Institute of Biophysics, Chinese Academy of Sciences, China)
FT-07	Elucidating the reducibility of sulfur dioxide on cysteine proteomes Zongmin Li 李宗敏 (Peking University First Hospital, China)
FT-08	Exploring the collaboration of redox and autophagy systems based on a genome-wide new redox genes screening Xinhua Qiao 乔新华 (Institute of Biophysics, Chinese Academy of Sciences, China)
FT-09	TRPC6-mediated Zn^{2+} influx improves heart failure through supersulfide formation Xinya Mi (Graduate School of Pharmaceutical Sciences, Kyushu University, Japan)
FT-10	TRPC6-mediated Zn^{2+} influx mitigates cardiac fibrosis through maintaining redox homeostasis Chenlin Su (Graduate School of Pharmaceutical Sciences, Kyushu University, Japan)
FT-11	Detection of Protein Tyrosine Nitration or Amination Jinwen Yang 杨劲文 (Huazhong University of Science and Technology, China)

FT-12	Discovery of small molecule inhibitors specifically targeting the Ero1α-PDI oxidative protein folding pathway Shuo Sun 孙硕 (Institute of Biophysics, Chinese Academy of Sciences, China)
FT-13	Ferrocene Correlates with Ferroptosis: Multiple Approaches to Explore Ferrocene-appended GPX4 Inhibitors as Anticancer Agents Yong Wang 王勇 (Ocean University of China, China)
FT-14	Unraveling the roles of Glutathione S-transferase P in protein S-glutathionylation modulation: Implications of therapeutic targets for oxidative organ injury Yongjie Zhang 张永杰 (China Pharmaceutical University, China)
FT-15	Loss of poly(ADP-ribose) polymerase 1 promotes catalase activation via the endothelin receptor Jiabin Yu 于佳斌 (Interdisciplinary Graduate Program in Advanced Convergence Technology & Science, Jeju National University, Korea)
FT-16	Myeloperoxidase (MPO) plays a key role in mitophagy in murine macrophages Chaorui Guo 郭朝瑞 (China Pharmaceutical University, China)
FT-17	PM2.5 induced iron accumulation-associated liver injury via activation of ferroptosis and NLRP3 inflammasome Lili Xin 信丽丽 (Soochow University, China)
FT-18	Polysulfides mediate multiple types of protein modification and tumor growth Huaiwei Liu 刘怀伟 (Shandong University, China)
FT-19	Targeting the integrated stress response and redox balance is a new strategy in meningioma inhibiting Yuanyuan Wang 王圆圆 (Institute of Biophysics, CAS, China)
FT-20	Analysis of aging biomarkers and construction of a physiological age prediction model based on cytokine profiling Lvtao Zeng 曾律滔 (Beijing hospital, China)
FT-21	Physiologically relevant Fenton-like reactions and redox cycles of labile iron species: implications for ferroptosis and Alzheimer's disease Zhongwei Zhao 赵仲伟 (Beijing University of Chemical Technology, China)
FT-22	PM2.5-induced premature senescence in HUVECs through the SIRT1/ PGC-1α/SIRT3 pathway Wu Jing 武婧 (Soochow University, China)

FT-23	The Beneficial Effects of Knockout of Astrocytic Ceruloplasmin on Learning and Memory Function in Aging Mice
	Zhongda Li 李忠达 (Ministry of Education Key Laboratory of Molecular and Cellular Biology, The Key Laboratory of Animal Physiology, Biochemistry and Molecular Biology of Hebei Province, College of Life Sciences, Hebei Normal University, China)
FT-24	The changes of genes and protein which affects mitochondrial fusion and fission in AD transgenic mice
	Seino Anna (Shibaura Institute of Technology, Japan)
FT-25	The molecular mechanism study of oxidized microRNA regulating P21 and promoting aging
	Yingmin Zhang 张英敏 (Beijing hospital, China)
FT-26	Disruption of E-prostanoid 3 receptor on cardiomyocytes protects against heart ischemia reperfusion injury
	Dong He 何东 (Shantou University Medical College, China)
FT-27	The molecular mechanism of lysosome function impairment and promotes fat accumulation by loss of G6PD
	Shanzhuang Niu 牛善壮 (Yunnan University, China)
FT-28	Endogenous hydrogen sulfide promotes the proliferation and metastasis of breast cancer through PGK1 S-sulfhydration
	Chenghua Luo 罗成华 (Medical College, Shihezi University, China)
FT-29	High PRDX4 Expression Can Predict Worse Pathological Characteristics in Cutaneous Squamous Cell Carcinom
	Jia Han 韩佳 (Department of Pathology, Kanazawa Medical University Hospital, Ishikawa, Japan)
FT-30	Hydrogen Peroxide Turn on Heat as Thermogenic agents and signals: Cellular Thermoregulation in Physiologies and Pathphysiologies
	Xu zhang 张旭 (Zhengzhou University, China)
FT-31	Increased oxidative stress induced by high-fat and high-fructose diets contribute to type 2 diabetes and its associated complications
	Qingyu Wang 王清宇 (Beijing Hospital, China)
FT-32	LPO-dependent lipid rafts inhibit immunogenic ferroptosis and pyroptosis in melanoma
	Guoquan Liu 刘国全 (Institute of Advanced Clinical Medicine, Peking University, China)

FT-33	Pharmacological targeting of NRF2 represents a promising therapeutic approach for ferroptosis-related diseases Pengfei Liu 刘朋飞 (National & Local Joint Engineering Research Center of Biodiagnosis and Biotherapy, The Second Affiliated Hospital of Xi'an Jiaotong University, China)
FT-34	Radix Rehmanniae and its Active Ingredients Ameliorate CFA-Induced Inflammation by Attenuating Macrophage-Mediated Localized Response and Nitrative Damage Jie Chen 陈杰 (Department of Orthopedics, Shanghai Institute of Traumatology and Orthopedics, Ruijin Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai, 200025, China)
FT-35	Redox regulated Mitophagy in Arsenite-induced Malignant Transformation of Human Keratinocytes Qianlei Yang 杨乾磊 (School of Public Health, Jiangsu Key Laboratory of Preventive and Translational Medicine for Geriatric Diseases, MOE Key Laboratory of Geriatric Diseases and Immunology, Suzhou Medical College of Soochow University, China)
FT-36	Role of miR-3689a-3p in the regulation of mitochondrial oxidative stress in the sorafenib resistance of hepatocellular carcinoma Yau-Tuen Chan (The University of Hong Kong, China)
FT-37	S-nitrosylation enhances RhoA activity and promotes tumor cell invasion and metastasis Yusheng Lu 卢余盛 (Fujian-Taiwan-Hongkong-Macao Science and Technology Cooperation Base of Intelligent Pharmaceutics, College of Material and Chemical Engineering, Minjiang University, China)
FT-38	The circ_0071616-miR-140-3p-USP34 axis mediates FoxM1 deubiquitination in Helicobacter pylori-induced gastric malignant transformation Xize Li 李析泽 (University of Health and Rehabilitation Sciences, China)
FT-39	A Bayesian benchmark concentration analysis for urinary fluoride and intelligence in adults in Guizhou, China Tingxu Jin 金庭旭 (Suzhou Medical College of Soochow University, China)
FT-40	Circadian-Cognitive Synchrony Disrupted: Iron's Influence on Rhythmic and Memory-Related Neural Functions Qiong Wu 吴琼 (Hebei Normal University, China)
FT-41	Mechanism analysis of oxidative stress and inflammation in brain diseases Qianjin Liu 刘前进 (Xuzhou Medical University, China)
FT-42	Mechanism of arsenic regulation of mitochondrial damage and autophagy induced synaptic damage through SIRT1 and protective effect of melatonin Xiaoli Zhang 张小莉 (School of Public Health, Shanxi Medical University, China)

FT-43	Phase separation of BRD2 promotes ferritinophagy in depression Zhen Li 李振 (Shenzhen Hospital of Integrated Chinese and Western Medicine, China)
FT-44	S-nitrosoglutathione reductase alleviates morphine analgesic tolerance by restricting PKCα S-nitrosation Lingyan Su 苏凌燕 (Yunnan Agricultural University, China)
FT-45	Fecal microbe transplantation ameliorates arsenic-and-fluoride-induced nephrotoxicity of offspring rats co-exposure to arsenic and fluoride through microbiota-gut-kidney axis Xiaolin Tian 田晓琳 (Shanxi Medical University, China)
FT-46	Metabolic reprogramming in placental mitochondria respiration contributes to the reproductive success of indigenous Tibetan women living at high altitude Jicuomao Niang 娘吉措毛 (Affiliated Hospital of Qinghai University (School of Clinical Medicine), China)
FT-47	Ganoderma Lucidum Spore Lehuo Powder Attenuates Experimental Autoimmune Encephalomyelitis by Modulating Microglial Activation and Polarization through the NF-κB Signaling Pathway Lu Zhang (School of Chinese Medicine, LKS Faculty of Medicine, the University of Hong Kong, China)
FT-48	Brain-targeted liposomes with neuroprotective effects for precise therapy of ischemic stroke Siyu Tian 田丝雨 (Hebei Normal University, China)
FT-49	Inorganic Nanosensitizers for Cancer Nanodynamic Therapy Xiaoyan Zhong 仲晓燕 (Soochow University, China)
FT-50	Nano-assemblies overcome cancer multidrug resistance for effectively synergistic chemo-immuno-oncotherapy Yingnan Liu 刘英楠 (University of Salzburg, China)
FT-51	Nanomaterials for tumor-cell-specific catalytic therapy Xi Hu 胡希 (Anhui University of Chinese Medicine, China)
FT-52	Nanomedicine by Modulating ROS for Oncotherapy Guofang Zhang 张国芳 (Shenzhen Institute of Advanced Technology, Chinese Academy of Sciences, China)
FT-53	Protective effect of platinum nano-antioxidant and nitric oxide against hepatic ischemia-reperfusion injury Jing Mu 穆婧 (Peking University Shenzhen Hospital, China)

FT-54	Disruption of circadian rhythms promotes ventricular arrhythmia via oxidative stress and electrocardiography alternation Bingping Yang 杨冰萍 (Shantou University Medical College, China)
FT-55	Chrysanthemolide J mitigates acetaminophen-induced hepatotoxicity through LKB1 and PP2A-mediated mitochondrial hormesis Fei Zhou 周飞 (University of Macau, China)
FT-56	Network Medicine landscape on the Health-Enhancing Properties of Natural Antioxidants Mengchen Liu 刘梦晨 (Zhuhai Campus of Zunyi Medical University, China)
FT-57	Osteoprotective and osteoblastic potential of the Sambucus javanica Reinw ex Blume subsp. javanica leave Treethip Sukkho (Department of Biotechnology, Multidisciplinary and Interdisciplinary School, Chiang Mai University, Chiang Mai, Thailand)
Venue: The Ballroom A	
PO-01	A New Approach for Treatment of Atopic Dermatitis: Plasma-activated Solutions Tingyi Yin 殷婷怡 (The First Affiliated Hospital of Xi'an Jiaotong University, China)
PO-02	An assessment of Redox-stress Response Capacity (RRC) Shilong Li 李世龙 (Institute of Biophysics, Chinese Academy of Sciences, China)
PO-03	Construction of the platform for precision redox detection and regulation Minghao Deng 邓明昊 (Institute of Biophysics, Chinese Academy of Sciences, China)
PO-04	Effect of electrolytic water generated by an alkaline ionizer on the concentration change of kelp extract Sato Takuma (Shibaura Institute of Technology, Japan)
PO-05	Exploring the Precision Redox Map of Cells and *C. elegans* under Different Treatment Conditions with High Glucose Yuyunfei Huang 黄雨云飞 (Institute of Biophysics, Chinese Academy of Sciences, China)
PO-06	Improvement of mouse bone marrow transplantation and chimerism analysis by qPCR of the modified gene Gang Yu 余钢 (Shantou University Medical College, China)
PO-07	One-step ligation of the phosphine-thioester elucidates the landscape of S-nitrosation proteome in lipopolysaccharide-related inflammation Hui Ye 叶辉 (China Pharmaceutical University, China)

PO-08	Trityl-based biradical as EPR probe for superoxide radical with enhanced sensitivity Yurui Leng 冷雨睿 (Tianjin Medical University, China)
PO-09	Browning effect of Inguinal White Adipose Tissue by a Novel Lignan (-)-Secoisolariciresinol 4-O-Methyl Ether, Modified from Arctigenin, Attenuates Diet-induced Obesity by Activating Mitochondria and Peroxisomes Wenjun Jiao (Department of Science in Korean Medicine, Graduate School, Kyung Hee University, Korea)
PO-10	CPD84: A Novel PPI inhibitor Targeting ELF3-PtnA Interaction to Modulate Angiogenesis in Ovarian cancer Moon Inhye (Ewha Womans University, Korea)
PO-11	Development of Nitric Oxide-Donating Netarsudil Derivatives as a Synergistic Therapy for Glaucoma with Reduced Ocular Irritation Cunrui Li 李存睿 (China Pharmaceutical University, China)
PO-12	Development of novel trityl radicals as efficient buffers for superoxide radical via unprecedented reversible reactions Longfei Gao 高龙飞 (School of Pharmacy, Tianjin Medical University, China)
PO-13	Discovery of Selective Cathepsin S Inhibitors as Potential Therapeutic Agents for Triple-Negative Breast Cancer Liu Yi (Ewha Womans University, Korea)
PO-14	Genome-wide CRISPR Screening of Genes Regulating Endoplasmic Reticulum H_2O_2 Qiaoli Zhu 朱乔丽 (Institute of Biophysics, Chinese Academy of Sciences, China)
PO-15	GTSE1 promotes pulmonary fibrosis through the induction of EMT Jin Hee (Graduate School of Pharmaceutical Sciences, Ewha Womans University, Korea)
PO-16	In Situ Generation and High Bioresistance of Trityl-based Semiquinone Methide Radicals under Anaerobic Conditions in Cellular Systems Xizi Du 杜习姿 (Tianjin Medical University, China)
PO-17	MS-based Exclusive Isolation Study Unveils a Novel Anti-Melanogenic Phenolic Glycoside from Idesia Polycarpa Maxim Jung-Eun Lee (Dongguk University, Korea)
PO-18	Optimization for Bioactive Compounds, Antioxidant Activity of Complex Extract Containing Three Herbs Grown in Korea Using a Simplex-Centroid Mixture Design Jeoung-Gyu Lee (Hanyang University, Korea)

PO-19	Protective roles of supersulfides on acetaminophen induced liver injury Chunyu Guo (Kumamoto University, Japan)
PO-20	Single-Cell Analysis Reveals Cell-Specific Patterns and Spatiotemporal Regulation of Nuclear Redox State Miaoling Yang 杨淼泠 (Institute of Genetics and Developmental Biology, Chinese Academy of Sciences, China)
PO-21	Supersulfides protect against SARS-CoV-2 infection via suppression of the viral thiol proteases Jia Yao (Department of Environmental Medicine and Molecular Toxicology, Tohoku University Graduate School of Medicine, Japan)
PO-22	The function of sulfite oxidase in mitochondrial supersulfide metabolism Yingchi Xia 夏应驰 (Tohoku University, Japan)
PO-23	TRPC3-Nox2 complex formation participates in the progression of striated muscle atrophy Di Wu (Department of Physiology, Graduate School of Pharmaceutical Sciences, Kyushu University，Japan)
PO-24	Blockade of the TP Receptor Ameliorates the Ischemic Renal Disorders in PGIS Deficient Mice Jiahui Ge 葛佳辉 (Shantou University, China)
PO-25	Effect and mechanism of oligodendrocyte knockout of Fpn1 in mice on depression-like behavior Na Zhang 张娜 (Hebei Normal University, China)
PO-26	Hydrogen Sulfide Targets S-Sulfhydrated-cAMP-response element binding protein (CREB) Cys286 Residues to Inhibit the epithelial-mesenchymal transition (EMT) in Chronic Renal Injury Shuai Chen 陈帅 (Capital Medical University, China)
PO-27	Impact of tyrosine amination on the aggregation and neurotoxicity of amyloid-β: Unveiling a potential defensive mechanism in Alzheimer's disease Ting Hu 胡婷 (Huazhong University of Science and Technology, China)
PO-28	Insufficient S-sulfhydration of serum and glucocorticoid-regulated kinase 1 participates in hyperhomocysteinemia-induced liver injury Xinyu Zhu 祝新宇 (Capital Medical University, China)
PO-29	Near-infrared fluorescent probes for imaging vimentin in the brain of ischaemic stroke mice Simiao Zhang 张思淼 (Shandong Normal University, China)
PO-30	Prostaglandin E2 promotes platelet aggregation and thrombogenesis via Thromboxane A2 receptor besides its canonic receptor EP3 Kaiqi Xie 谢恺麒 (Medical College of Shantou University, China)

PO-31	Redox-inducible Radiomimetic Photosensitizers Selectively Suppress Cancer Cell Proliferation by Damaging DNA through Radical Cation Chemistry Luo Wang 王洛 (Tianjin Medical University, China)
PO-32	S-nitrosation of CaMKIIα matters, a new mechanism mediating learning and memory Boyu Chu 褚博煜 (Institute of Biophysics, Chinese Academy of Sciences, China)
PO-33	The effect of water-soluble metalloporphyrin FeTPPS on membrane damage and cytotoxicity induced by hIAPP Zhilong Wang 王智龙 (Huazhong University of Science and Technology, China)
PO-34	The S-nitrosation of CKMT1 impedes intracellular energy shuttling by inducing its dissociation from octamer to dimer Tiepeng Wang 王铁鹏 (The Institute of Biophysics, Chinese Academy of sciences, China)
PO-35	Screenings of nanoantibodies against protein disulfide isomerase and its antiplatelet aggregation effects Guozhen Cui 崔国祯 (School of Bioengineering, Zhuhai Campus of Zunyi Medical University, China)
PO-36	Analysis of the translation stalling sensor protein GCN1 complex using the proximity-dependent biotinylation enzyme TurboID Kazuki Hasegawa (Hirosaki University Graduate School of Medicine, Japan)
PO-37	Carbon dots cause developmental toxicity in zebrafish embryos via endoplasmic reticulum stress-mediated lipid dysregulation Zeng Liwen 曾丽雯 (Soochow University, China)
PO-38	Ceruloplasmin deficiency in Leydig cells causes testicular dysfunction via iron-mediated oxidative stress in mice Lihui Wu 吴丽辉 (Hebei Normal University, China)
PO-39	Domperidone induces apoptosis via inactivation of β-arrestin2-dependent MEK/ERK/STAT3 pathway in of human colon cancer cells So Jin Sim (Keimyung University, Korea)
PO-40	Environmental toxin exposure-induced oxidative stress impairs chromosome cohesion and segregation in mammalian oocytes Yan Yun 云彦 (Shantou Central Hospital, China)
PO-41	Karyoptosis is a cyclic AMP-responsive element binding protein 3 driven novel regulated cell death Weidong Chen (The Catholic University of Korea, Korea)

PO-42	Loss of poly(ADP-ribose) polymerase 1 boosts catalase activation via endothelin receptors Jiabin Yu (Interdisciplinary Graduate Program in Advanced Convergence Technology & Science, Jeju National University, Korea)
PO-43	Myriocin regulates cellular redox homeostasis via mitochondrial hormesis Wenling Gu 顾文凌 (Key Laboratory of Bio-Resources and Eco-Environment of Ministry of Education, College of Life Science, Sichuan University, China)
Venue: The Ballroom C	
PO-44	Protective Effects of OM2 Plant Extract against Oxidative Stress and Inflammation in Arsenic Exposed RAW264.7 cells Hyejin Kim (Hanyang University, Korea)
PO-45	The effects of local iron treatment on intervertebral disc degeneration Chenchen Li 李晨晨 (Hebei Normal University, China)
PO-46	The Function of 2-Mercaptoethanol in the Repair of DNA during Kidney Ischemia and Reperfusion through GPX4 Upregulation Moon Daeun (Department of Anatomy, Jeju National University College of Medicine, Korea)
PO-47	The Role and Mechanisms of Prostaglandin E2 Receptor EP3 in Acute Lung Injury Zhengpeng Zeng 曾征鹏 (Shantou University Medical College, China)
PO-48	Water-soluble single molecular probe for simultaneous detection of viscosity and hydrazine Jiazi Yin 尹佳子 (Guangdong University of Petrochemical Technology, China)
PO-49	Assessing Myriocin and NAC for protection against cisplatin induced hearing loss and renal toxicity in Mice Zhiyi Liu 刘治义 (The College of Life Science, Sichuan University, China)
PO-50	Dimethyl α-Ketoglutarate Promotes the Synthesis of Collagen and Inhibits Metalloproteinases in HaCaT Cells Bo-Yeong Yu (Dongguk University, Korea)
PO-51	Dynamic proteostasis imbalance is a hallmark of aging Chang Shi 时畅 (Institute of Biophysics, Chinese Academy of Sciences, China)
PO-52	Increasing redox-stress signaling threshold (RST) through stress to delay aging Haoyang Shi 时浩洋 (Institute of Biophysics, Chinese Academy of Sciences, China)

PO-53	Inhibition of ER Stress as a Protective Strategy of 3, 3', 4', 5, 5', 7 Hexahydroxyflavone against PM2.5-induced Apoptosis in Skin Cells Herath Mudiyanselage Udari Lakmini Herath (Department of Biochemistry, College of Medicine, and Jeju Research Center for Natural Medicine, Jeju National University, Korea)
PO-54	Optimization of Brain Microvascular Endothelial Cell Extraction Ruikun Xie 谢睿坤 (Hebei Normal University, China)
PO-55	Quercetin 3-D-galactoside mitigates PM2.5-mediated keratinocyte cell senescence by alleviating the endoplasmic reticulum and mitochondrial stress Pincha Devage Sameera Madushan Fernando (Department of Biochemistry, College of Medicine, and Jeju Research Center for Natural Medicine, Jeju National University, Korea)
PO-56	Relationship between ferroptosis and cellular senescence caused by particulate matter 2.5 in skin keratinocytes Meijing Piao (Department of Biochemistry, College of Medicine, and Jeju Research Center for Natural Medicine, Jeju National University, Korea)
PO-57	The modification of 8-oxyguanine in the microRNA seed region regulates aging process Jiaqi Guo 郭佳琪 (Beijing Hospital, China)
PO-58	Txn1-F54L mutation in rats generates neurodegeneration and chronic kidney disease through mitochondrial dysfunction and multiple cell death modes Iori Ohmori (Faculty of Education, Department of Child Neurology, Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, Okayama University, Japan)
PO-59	Chemerin Activates the NLRP3 Inflammasome Through CMKLR1, Mediating Potassium Current Dysregulation and Obesity-Related Atrial Fibrillation Yating Chen 陈雅婷 (Medical School of Chinese PLA, China)
PO-60	Different roles of E-prostanoid 3 receptor on renal parenchymal cells and myeloid cells in acute oxalate nephropathy Jinwei Guo 郭锦伟 (Shantou University Medical College, China)
PO-61	Dysregulated peroxisomal metabolites in cardiac adipose tissue induce heart dysfunction Se Jin Jung (Department of Science in Korean Medicine, Graduate School, Kyung Hee University, Korea)
PO-62	GSNOR contributes to age-related obesity by regulating the S-nitrosation of Beclin-1 to promote adipose tissue whitening Ting Xie 谢婷 (Institute of Biophysics, Chinese Academy of Sciences, China)

PO-63	Hepatic Adenosine Kinase mitigates hepatic steatosis and insulin resistance in obese mice Kai Luo 骆开 (College of Life Sciences, University of Chinese Academy of Sciences, China)
PO-64	Inhibition of GCN2 alleviates hepatic steatosis and oxidative stress in alcoholic-related liver disease Ying Xu 徐颖 (University Network Teaching Platform, University of Chinese Academy of Sciences, China)
PO-65	Intestinal flora-bile acid-FXR axis regulates hepatic lipid metabolism induced by arsenic and fluoride co-exposure in rats Jinyao Chen 陈锦瑶 (Shanxi Medical University, China)
PO-66	Knocking out GCN2 exacerbates oxidative stress in the liver under zinc-deficient conditions by reducing Nrf2 expression Zhuoran Yu 于卓然 (University of Chinese Academy of Sciences, China)
PO-67	Machine learning based model for predicting coronary heart disease using dynamic triglyceride-glucose index: a Longitudinal study cohort CHARLS database Yi Yang 杨易 (I.T. Department of Faculty of Engineering and Information Technology of university of technology Sydney, Australia)
PO-68	Nfe2l1 deficiency exacerbates alcohol-induced liver injury in mice Jinghui Qu 曲景辉 (China Medical University, China)
PO-69	Obesity-induced Nox2 activation prolongs cardiac repolarization Bin Li 李彬 (The Eighth Medical Center of PLA General Hospital, China)
PO-70	Paeonol Induces Thermogenesis by Suppressing Endoplasmic Reticulum Stress via NRF2 Activation in Beige Adipocytes Ja Yeon Park (Department of Science in Korean Medicine, Graduate School, Kyung Hee University, Seoul, Korea)
PO-71	Suppression of O-GlcNAc transferase (OGT) inhibits adipogenesis in 3T3-L1 adipocytes through the modulation of PPARγ O-GlcNAcylation Hoang Hai Ngo (Dongguk University, Korea)
PO-72	YY1 nitration participates in T2DM induced-cardiomyocyte lipotoxicity by inhibiting Anxa3 transcription Jiayin Chai 柴嘉音 (Capital Medical University, China)
PO-73	α-Ketoglutarate pretreatment prevents hyperlipidemia-induced endothelial injury and fatty liver by ameliorating mitochondrial dysfunction and oxidative stress Danyu Cheng 程丹雨 (Xi'an Jiaotong University, China)

PO-74	A fluorescence-enhanced near-infrared fluorescent probe for real-time imaging of protein sulfenic acids in oxidative stress
	Zhixuan Feng 冯芷璇 (Institute of Chemistry, Chinese Academy of Sciences, China)
PO-75	Acute lung injury induced by acid aspiration or lipopolysaccharide leads to liver injury and hepatic regulated cell death in mice
	Cheng Peng 彭程 (Cardiovascular Research Center, Shantou University Medical College, China)
PO-76	COX7A1 heightens the susceptibility of human NSCLC cells to cystine deprivation-induced ferroptosis
	Rongrong Liu 刘蓉蓉 (The Second Affiliated Hospital of Xi'an Jiaotong University, China)
PO-77	Decomposable Nanodrugs Inducing Immunogenic Cell Death and cGAS-STING Pathway Activation for Enhanced Photodynamic Chemotherapy-Driven Immunotherapy
	Chenchen Li 李晨晨 (Hebei North University, China)
PO-78	Dehydrocostus lactone as a potential therapeutic agent for colorectal cancer and dextran sulfate sodium-induced colitis in mice
	Sun-Young Hwang (College of Korean Medicine, Dongshin University, Korea)
PO-79	Europium-modified carbon nitride nanosheets for smartphone-based fluorescence sensitive recognition of anthrax biomarker dipicolinic acid
	Wenhao Du 杜文浩 (Guangdong University of Petrochemical Technology, Korea)
PO-80	Exploring immune evasion mechanism mediated by superoxide anion of hepatic stellate cells by fluorescence imaging
	Yuantao Mao 毛元涛 (Shandong Normal University, China)
PO-81	Exploring Nitroproteomics in Cancer Biology: A Case Study of Early Onset Gastric Cancer
	Kwang Pyo Kim (Kyung Hee University, Korea)
PO-82	Hepatic Sinusoidal Endothelial Cells Targeted Delivery of Nitric Oxide via Glycosyl-modified Glycosidase to Treat Liver Fibrosis
	Jingjie Tan 谭靖节 (Tianjin Medical University, Tianjin, China)
PO-83	Investigating the Relationship Between Tumor Stem Cells, Oxidative Stress, and Cisplatin Resistance in Ovarian Cancer
	Jinzhi Lu 鲁锦志 (The First Affiliated Hospital of Yangtze University, China)
PO-84	Liposomes-encapsulated hERG Potassium Channel Probe for Glioblastoma Therapy and Imaging
	Li Liu 刘丽 (Shandong Normal University, China)

PO-85	Manipulating disulfide bond formation of the Spike protein to inhibit the SARS-CoV-2 infection Xinqian Li 李欣倩 (Institute of Biophysics, Chinese Academy of Sciences, China)
PO-86	New approach to tumor therapy: Targeting the destruction of redox balance Qiwen Luo 罗其文 (Key Laboratory of Metabolism and Molecular Medicine of the Ministry of Education, Department of Biochemistry and Molecular Biology, School of Basic Medical Sciences, Fudan University, China)
PO-87	Repurposing Flubendazole for Glioblastoma Ferroptosis by Affecting xCT and TFRC Proteins Wei Teng 滕炜 (Guizhou Medical University, China)
PO-88	Respiratory exposure to lithium nickel manganese cobalt oxide particles induces multi-organ damage and disrupts redox homeostasis in mice Junyi Wang 王君仪 (China Medical University, China)
PO-89	Role and mechanism of nanomedicine with ROS regulating ability for differentiation therapy of myeloid neoplasia Haiyan Xu 许海燕 (Institute of Basic Medical Sciences, Chinese Academy of Medical Sciences & Peking Union Medical College, China)
PO-90	Synthesis and study of β-glucuronidase controlled fluorescent probe Anqi Li 李安琪 (Tianjin Medical University, China)
PO-91	The acceleration of iron transport promotes the malignancy of glioma through Oxidizing 293 cysteine on sirt5 by the increased production of H_2O_2 Fei Wang 王飞 (Hebei Normal University, China)
PO-92	β-Lapachone Enhances NETosis through Reactive Oxygen Species Generation: Mechanistic Insight in Neutrophil-Mediated Innate Immunity Jisoo Han (Department of Science in Korean Medicine, Graduate School, Kyung Hee University, Korea)
PO-93	A Versatile Fluorescent Probe for Hydrogen Peroxide in Serotonergic Neurons of Living Mouse Brains with Depression Feida Che 车飞达 (School of Chemistry, Chemical Engineering and Materials Science, Shandong Normal University, China)
PO-94	DDAH1 attenuates MPTP-induced Parkinson's disease impairment via FOXO3 mediated signals Zhirui Li 李祉睿 (Capital Medical University, China)

PO-95	Discovery of potent LRRK2 inhibitors by ensemble virtual screening strategy and bioactivity evaluation
	Shuli Li 李淑黎 (University of Macau, China)
PO-96	Environmental standard limit concentration' arsenic exposure is associated with anxiety, depression, and autism-like changes in early-life stage zebrafish
	Yuanhui Zhu 朱原慧 (Department of Toxicology, School of Public Health, Jiangsu Key Laboratory of Preventive and Translational Medicine for Geriatric Diseases, China)
PO-97	Exploring the Neuroinflammatory Pathways of 8-oxoGTP and Their Effects on Cognitive Decline
	Jin Li 李瑾 (Institute of Geriatric Medicine, Chinese Academy of Medical Sciences, China)
PO-98	Functional study of TMPRSS6 in APP/PS1 mouse
	Hongtao Sun 孙洪涛 (HeBei Normal University, China)
PO-99	GSNOR, a new player in depression
	Chuanxin Sun 孙传鑫 (Institute of Biophysics, Chinese Academy of Sciences, China)
PO-100	Hydrogen peroxide in midbrain sleep neurons regulates sleep homeostasis
	Yujing Tian 田玉静 (Center for Excellence in Brain Science and Intelligence Technology, Chinese Academy of Sciences, China)
PO-101	Improving mitochondrial function with arachidonic acid supplementation to alleviate cognitive deficits in schizophrenia patients
	Yan Gao 高琰 (Shanghai Jiao Tong University, Bio-X Institutes, China)
PO-102	Melatonin derivative 6a protects Caenorhabditis elegans from formaldehyde neurotoxicity via ADH5
	Na Feng 冯娜 (School of Pharmacy and Food Engineering, Wuyi university, China)
PO-103	Nos2b regulates injury-induced cxcl18b-defined transitional state Müller Glia proliferation in the zebrafish retina
	Aojun Ye 叶傲君 (Institute of Biophysics, Chinese Academy of Sciences, China)
PO-104	Overexpressed of ferrous iron ions triggers Neutrophil extracellular trap formation and contributes to multiple sclerosis
	Shenyu Yan 颜深玉 (The University of Hong Kong, China)
PO-105	Oxidative stress increased β-galactosidase activity in the brains of mice with depression
	Deqiang Li 李德强 (Institutes of Biomedical Sciences, Shandong Normal University, China)

PO-106	Perturbation in mitochondrial quality control is associated with oxidative mitochondrial damage in patients with schizophrenia Shuhui Li 李书慧 (Shanghai Jiao Tong University, China)
PO-107	Pyran compounds 7r reduces α-synuclein aggregation and protects neuronal activity in Caenorhabditis elegans by modulating the oxidative stress pathway and enhancing the activation of autophagy Ruiting Han 韩瑞婷 (Wuyi University, China)
PO-108	Restoring the redox and norepinephrine homoeostasis in mouse brains promotes an antidepressant response Qi Ding 丁琪 (Shandong Normal University, China)
PO-109	Sodium Danshensu Attenuates Blood-Brain Barrier Disruption and Hemorrhagic Transformation in Ischemic Stroke Rats with Acute Hyperglycemia: Involvement of ONOO-NLRP3 Inflammasome Pathway Shuang Chen 陈霜 (The University of Hong Kong, China)
PO-110	Superoxide anion-mediated mitochondrial dysfunction in the hippocampus of depressed mice revealed by fluorescent sensing and labeling strategies based on tandem activity Xiwei Li 李玺威 (Shandong Normal University, China)
PO-111	Synergistic effects of Tanshinone IIA sustained release nano particles on Parkinson's disease insults Yuhao Kan 阚宇豪 (Capital Medical University, China)
PO-112	TFR Enhances α-Synuclein-mediated Ferroptosis in the Hippocampus of Parkinson's Disease Dementia Lijun Zhao 赵丽君 (Shenzhen Hospital of Integrated Traditional Chinese and Western Medicine, China)
PO-113	The beneficial effect of Angong Niuhuang Pill (AGNHP) on ischemic stroke via the regulation of gut microbiota Ao Shang 商奥 (The University of Hong Kong, China)
PO-114	The Deubiquitination of Erg1 by USP7 Regulates Nrf2 Redox Balance and Mitigates Fluoride-Induced Neurotoxicity Wenjin Qiu 仇文进 (The Affiliated Hospital of Guizhou Medical University, China)
PO-115	The effect and mechanism of knockdown Ferroportin1 in microglia on cerebral ischemia-reperfusion injury Qiaoya Zhao 赵悄雅 (The Laboratory of Iron Metabolism, College of Life Science, Hebei Normal University, China)

PO-116	Synthesis and characterization of Tanshinone IIA nanoparticles for the treatment of cerebral stroke Sihan Wang 王思涵 (Department of Pharmacology, School of Basic Medical Sciences, Capital Medical University, China)
PO-117	Fluorescence-enhanced detection of hypochlorite based on in situ synthesis of functionalization-free carbon spheres Tianhong Liu 刘天鸿 (Guangdong University of Petrochemical Technology, China)
PO-118	Liver injury induced by subchronic respiratory exposure to lithium nickel cobalt manganese oxide in mice Yongqin Xia 夏永钦 (China Medical University, China)
PO-119	Preparation of baicalin self-microemulsified preparation and study on its radiation protection effect Xinran Liu 刘欣然 (Huazhong University of Science and Technology, China)
PO-120	Biyuantong decoction reduces postoperative recurrence of chronic rhinosinusitis by inhibiting ferroptosis Yinyin Yao 姚茵茵 (Fujian University of Traditional Chinese Medicine, China)
PO-121	Coptisine Reduces Transformation Process From Chronic Atrophic Gastritis to Gastric Cancer via Inhibiting Hepcidin Expression Yashuo Zhao 赵亚硕 (Hebei University of Chinese Medicine, China)
PO-122	Dihydroisotanshinone I functions as an agonist of TRPV1 to alleviate lipopolysaccharide-induced neuroinflammation in vitro and in vivo Nan Xu 徐楠 (University of Macau, China)
PO-123	RSM for Revealing Traditional Chinese Medicine's Yun Qi Wisdom Based on the Theory of Superstrings Jiulong Chen 陈久龙 (Dahua Group Co., Ltd., China)
PO-124	A phosphatase-like nanomaterial promotes autophagy and reprograms macrophages for cancer immunotherapy Su Li 李苏 (Laboratory of Immunology and Nanomedicine, Laboratory of Inflammation and Vaccines, Shenzhen Institute of Advanced Technology, Chinese Academy of Sciences, China)
PO-125	In situ nitric oxide production for selective S-nitrosation as a promising synergistic cancer treatment strategy Chen Zhang 张宸 (China Pharmaceutical University, China)
PO-126	liver-targeted plasmid lipid nanomedicine treats liver fibrosis by ROS elimination Zhengxun Liu 刘正汛 (Hebei Normal University, China)

PO-127	NOS-like activity of CeO_2 nanozymes contributes to diminishing the vascular plaques Yuxiang Sun 孙玉祥 (Yangzhou University, China)
PO-128	Therapeutic effect of PN-CeO_2 on atopic dermatitis by regulating oxidative stress in keratinocytes and macrophages Ruimin Bai 白瑞敏 (Department of Dermatology, The First Affiliated Hospital of Xi'an Jiaotong University, China)
PO-129	A Catechol Isoquinoline Salsolinol Induces Apoptosis of Human Liver Cancer Cells by Regulating the STAT1/3 Signaling Jeong-Hwa Woo (Basic Science Research Institute, Sungshin Women's University, Korea)
PO-130	Acute Sleep Deprivation Induces Liver Damage and Protective Effects of Chalcone Analogue TAK Yifang Wang 王一方 (Xi'an Jiaotong University, China)
PO-131	Assessing Myriocin and N-Acetyl Cysteine on Age-Related Hearing Loss and disruption of Advanced Glycation End Products in mice Lin Cheng 程琳 (State Key Laboratory of Biotherapy, Sichuan University, China)
PO-132	Characterization of a polysaccharide from Amauroderma rugosum and its proangiogenic activities in vitro and in vivo Xin Nie聂欣 (University of Macau, China)
PO-133	Co-Treatments of Gardeniae Fructus and Silymarin Ameliorates Excessive Oxidative Stress-Driven Liver Fibrosis by Regulation of Hepatic Sirtuin1 Activities Using Thioacetamide-Induced Mice Model Jin A Lee (Daegu Haany University, Korea)
PO-134	Daphnetin ameliorates hepatic steatosis by suppressing peroxisome proliferator-activated receptor gamma (PPARG) in ob/ob mice Zhen Wang 王珍 (Xi'an Jiaotong University, China)
PO-135	Emodin, a major component of Cassia seed extract, exhibits potent anti-inflammatory effects in vitro and in vivo Kwanhwan Wi (College of Korean Medicine, Dongshin University, Korea)
PO-136	Exploring the Mechanisms of Action of Active Constituents in Schisandrae Fructus for the Management of Diabetic Cardiomyopathy Jihang Chen 陈吉航 (The Chinese University of Hong Kong, Shenzhen, China)
PO-137	Ferulic acid as a potent natural antioxidant: mechanisms and applications in animal health and production Yongquan Han 韩永权 (Guangzhou Cohoo Biotechnology Co., Ltd., China)

PO-138	Gaudichaudione H ameliorates liver fibrosis and inflammation by targeting NRF2 signaling pathway Mengjiao Shi 石梦姣 (National & Local Joint Engineering Research Center of Biodiagnosis and Biotherapy, The Second Affiliated Hospital of Xi'an Jiaotong University, China)
PO-139	Neem leaf extract exhibits anti-aging and antioxidant effects from yeast to human cells Jinye Dang 党劲野 (Sichuan University, China)
PO-140	Saikosaponin A suppresses inflammation in DSS-induced colitis mouse model Young-Gwon Kim (College of Korean Medicine, Dongshin University, Naju, Korea)
PO-141	Salsolinol Alleviates Tumor Formation and Anxiety Like Behaviors in the Diethyl Nitrosamine-induced Hepatocarcinogenesis in Mice Chan-Mi Park (Department of Future Applied Sciences, College of Natural Sciences, Sungshin Women's University, Korea)
PO -142	The ROMO1-LONP1-SIRT3 axis protects against hepatic steatosis via promoting mitochondrial lipid catabolism Kun Peng (Institute of Molecular Medicine, College of Future Technology, Peking-Tsinghua Center for Life Sciences, Peking University, China)
PO-143	Dexmedetomidine's Protective Effects and Mechanisms on Lung Injury Induced by Vesicant Sulfur Mustard SHI Minjie 师敏婕 (Air Force Medical University, China)
PO-144	Glutathione-sensitive mesoporous nanoparticles loaded with cinnamaldehyde for chemodynamic and immunological therapy of cancer Lichong Zhu 祝李冲 (National Engineering Research Center for Nanomedicine, College of Life Science and Technology, Huazhong University of Science and Technology, China)
PO-145	Hyperbaric oxygen augments probiotic-derived selenium nanoparticles-induced oxidative stress to enhance cancer immune checkpoint blockade therapy Puze Li 李璞泽 (National Engineering Research Center for Nanomedicine, College of Life Science and Technology, Huazhong University of Science and Technology, China)
PO-146	Engineered Extracellular Vesicles to Enhance Antigen Presentation for Boosting Light-Driven Tumor Immunotherapy Xuyu Li 李旭钰 (National Engineering Research Center for Nanomedicine, College of Life Science and Technology, Huazhong University of Science and Technology, China)
PO-147	Leveraging tumor-repopulating cell-derived microparticles for enhanced cancer therapy via targeted Akkermansia muciniphila modulation chemotherapy Shiyu Li 李诗雨 (National Engineering Research Center for Nanomedicine, College of Life Science and Technology, Huazhong University of Science and Technology, China)

PO-148	DNA-functionalized extracellular vesicles for efficient free radical prodrug activation in cancer therapy Zhiheng Cai 蔡之恒 (National Engineering Research Center for Nanomedicine, College of Life Science and Technology, Huazhong University of Science and Technology, China)
PO-149	Targeted intervention in nerve-cancer crosstalk enhances pancreatic cancer chemotherapy Jingjie Liu 刘婧杰 (National Engineering Research Center for Nanomedicine, College of Life Science and Technology, Huazhong University of Science and Technology, China)
PO-150	Glutaminolysis inhibition sensitizes cuproptosis by regulating oxidative stress to eliminate cancer stem cells Zitao Fan 樊子涛 (College of Life Sciences and Technology, Huazhong University of Science and Technology, China)

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免疫组是研究免疫相关的全套分子库，它们的作用靶分子及其功能。包括实体肿瘤HLA-loss检测、免疫组库、免疫肽组-肿瘤抗原肽，四聚体技术等产品。

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扩增子测序和宏基因组测序是两种常用的方法。扩增子测序获得环境样本中的微生物群落结构、进化关系以及微生物与环境相关性等信息。宏基因组主要研究微生物种群结构、基因功能、微生物之间的相互协作关系以及微生物与环境之间的关系。

代谢组

代谢组学主要目标是定性定量地研究生命体对外界刺激、生理病理变化以及基因突变而产生的体内代谢物水平的多元动态反应，研究对象大多是相对分子质量≤1000 Da的小分子物质，如有机酸、氨基酸、核苷酸、糖、脂质、维生素等。

单细胞转录组

在单细胞水平直接分析基因表达，并在单细胞水平分析细胞内群体异质性、定义细胞类型、细胞状态和细胞的动态转变，除了识别新的细胞亚型和稀有细胞群外，单细胞测序技术还能更好地理解转录动力学和基因调控关系。

蛋白组

蛋白质组学是指在大规模水平上探索蛋白质的特征，包括蛋白质的表达水平，翻译后的修饰，蛋白与蛋白相互作用等，由此获得在蛋白质水平上的关于疾病发生，细胞代谢等过程的整体而全面的认识。

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四聚体

微生物组

扩增子测序
宏基因组测序

单细胞转录组

单细胞3'转录组测序
单细胞5'转录组+免疫组库测序
单细胞全序列转录组测序
空间转录组测序

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Astral-DIA 深度血液蛋白组学
Astral-DIA 高深度血液蛋白组学
磷酸化修饰蛋白组

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代谢流

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单细胞转录组+空间转录组

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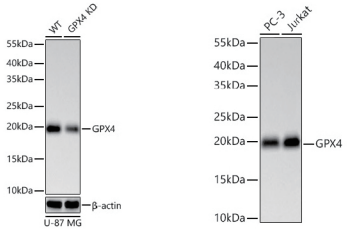
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精选产品

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A11243 [KD Validated] GPX4 Rabbit mAb

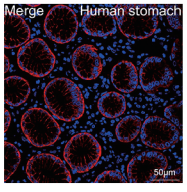
应用: IF/ICC, WB, ELISA
物种: H, M, R
文献引用(68篇)



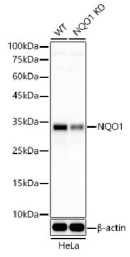
NQO1 KO/KD Validated 重组 兔单抗

A19586 [KD Validated] NQO1 Rabbit mAb

应用: IF/ICC, WB, ELISA
物种: H, M, R
文献引用(44篇)



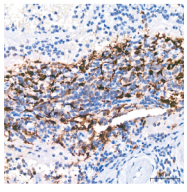
人胃组织



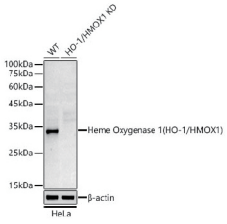
HO-1 KO/KD Validated 重组 兔单抗

A19062 [KD Validated] Heme Oxygenase 1 (HO-1/HMOX1) Rabbit mAb

应用: IF/ICC, IHC-P, WB, ELISA
物种: H, M, R
文献引用(42篇)



人脾脏组织



氧化还原研究精选产品列表

Target	Cat. No.	Product Name	Application	Reactivity
NADPH oxidase (NOX)	A19701	NOX2/gp91phox Rabbit mAb	WB, ELISA	H, M, R
	A3703	NOXA2/p67phox Rabbit mAb	WB, IHC-P, ELISA	M, R
	A22149	NADPH oxidase 4 (NOX4) Rabbit mAb	WB, IF/ICC, ELISA	H, M, R
Nitric oxide synthase (NOS)	A3774	iNOS Rabbit mAb	WB, IF/ICC, ELISA	M, R
	A20985	eNOS Rabbit mAb	WB, IHC-P, ELISA	H, M, R
Cycloxygenase (COX)	A3560	COX2/PTGS2 Rabbit mAb	WB, IHC-P, ELISA	H, M, R
Lipoxygenase	A2877	ALOX5 Rabbit mAb	WB, IHC-P, IF/ICC, ELISA	H, M, R
	A22908	ALOX15 Rabbit mAb	WB, IHC-P , ELISA	H, M, R
Xanthine oxidase (XOR)	A9022	Xanthine Oxidase (XDH) Rabbit mAb	WB, IHC-P, IF/ICC, ELISA	H, M, R
SOD2	A19576	[KO Validated] SOD2 Rabbit mAb	WB, IHC-P, IF/ICC, ELISA	H, M, R
HO-1	A19062	[KD Validated] Heme Oxygenase 1 (HO-1/HMOX1) Rabbit mAb	WB, IHC-P, IF/ICC, ELISA	H, M, R
GPX4	A25009	[KD Validated] GPX4 Rabbit mAb	WB, IHC-P, IF/ICC, ELISA	H, M, R
	A11243	[KD Validated] GPX4 Rabbit mAb	WB, IF/ICC, ELISA	H, M, R
Thioredoxin 1	A4024	Thioredoxin 1 (Trx1/TXN) Rabbit mAb	WB, IHC-P, ELISA	H, M, R
Thioredoxin 2	A4424	Thioredoxin 2 (Trx2/TXN2) Rabbit mAb	WB, IF/ICC, ELISA	H, M, R
NQO1	A19586	[KD Validated] NQO1 Rabbit mAb	WB, IF/ICC, ELISA	H, M, R
PRDX3	A2398	Peroxiredoxin 3 (PRDX3) Rabbit mAb	WB, IHC-P, IF/ICC, ELISA	H, M
PRDX4	A9131	Peroxiredoxin 4 (PRDX4) Rabbit mAb	WB, IHC-P, IF/ICC, ELISA	H, M, R
NRF2	A25327	[KO Validated] NRF2 Rabbit mAb	WB, IHC-P, IF/ICC, ELISA	H, M, R
	AP1133	Phospho-NRF2-S40 Rabbit mAb	WB, IHC-P, ELISA	H, M, R

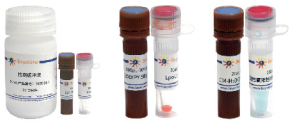
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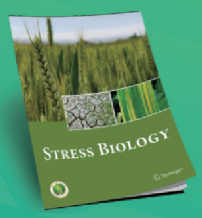
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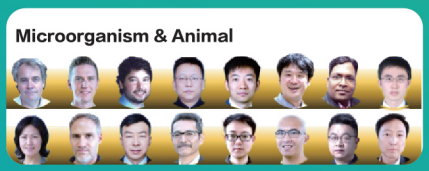
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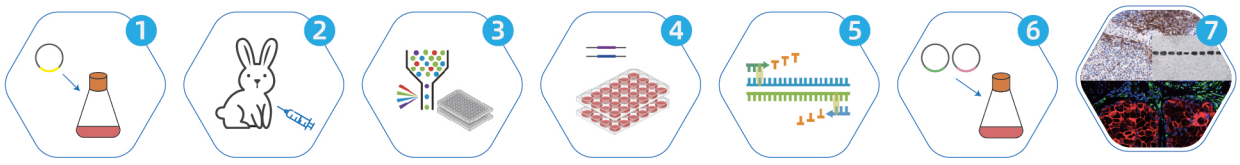
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1、高时空分辨率:单细胞及亚细胞水平(细胞质、细胞核)实时、原位、定量检测；
2、多种检测指标:开放试剂盒，单个活细胞多种小分子含量(如乳酸，ATP，ROS等)及酶活性(葡萄糖甘酶等)检测；
3、单细胞提取注射:提取细胞内容物或细的器(如溶酶体、线粒体)进行质谱分析、测序、酶活性分析;或注射药物、底物到细胞内进行药效评估等；
4、活体检测:可在活体水平上检测特定穴位处生化指标的变化，研究药物起效机理。

产品应用

细胞代谢　　肿瘤机制
新药研究　　生物纳米材料
神经科学　　中医针灸

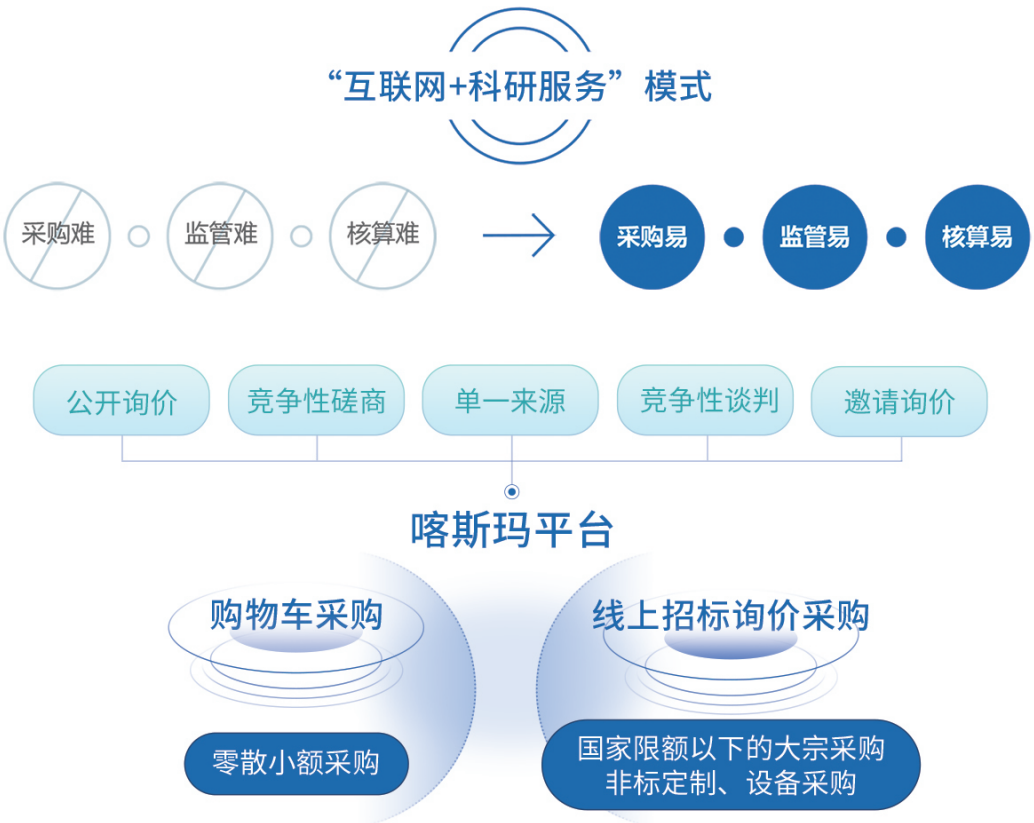


江苏瑞明生物科技有限公司
JIANGSU RAYME BIOTECHNOLOGY CO.,LTD

地址：江苏宜兴市经济开发区杏里路10号光电产业园1栋7楼
电话：0510-80328166
邮箱：sales@raymetech.com
网站：www.raymetech.com

扫码关注 了解更多

喀斯玛(北京)科技有限公司是中国科学院控股有限公司旗下国有企业。于2013年创建了服务科研领域的垂直型B2B+O2O第三方电子商务平台，集交易、管理、资讯为一体，以网络超市和信息化管理模式，实现了科研物资采购及管理的规范、安全、便捷。平台业务范围涵盖生物试剂及耗材、化学试剂、科研仪器及维修、技术服务、办公用品、元器件、农资农具、实验气体等。入网供应商9000余家，在线商品1.7亿条，全国超2600家科研、教育、医疗及产业机构应用本平台，服务了107个院士团队，保障了超254亿元国家科研经费的规范使用。十年来，喀斯玛获得了多项资质与荣誉，已成为国内重要的科技创新服务实体。



扫码关注
喀斯玛订阅号

BRS

BQC REDOX SYSTEM

总抗氧化能力检测仪

一种新型的电化学法、简单、快速、精确、多参数、便携式的设备
用于评估 **总抗氧化状态(TAC)** 和 **H_2O_2清除能力**

特点：

直接、快速

无有机溶剂

无自由基引发剂

无需样品预处理

轻松得到实验结果

无需其他昂贵的仪器

无需分光光度计和荧光计

无需专业技术人员

总抗氧化能力

BRS检测TAC仅需1min
且无需复杂的样品预处理。

一键测试多参数

饮料类：抗坏血酸、多酚、TAC
红酒类：抗坏血酸、多酚
油类：维生素E、TAC、氧化作用

H_2O_2清除能力

BRS只需两步即可检测H_2O_2清除能力，无需繁琐的光学法检测。

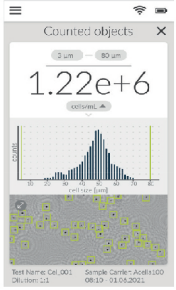


BRS适用样品

血液
血清
血浆
细胞和组织
尿液
唾液
饮料
提取物
粉末
其他

HI£
I'm fluidlad R-300
便携式细胞计数仪&光谱仪

anvajo



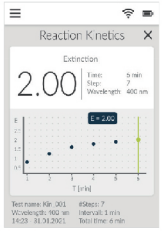
全自动细胞计数



光谱分析

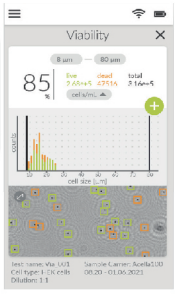


动力学测量



细胞计数

全息显微技术
免染色活率分析
大视场细胞计数
样本量仅需4-20ul



免染色
细胞存活率分析

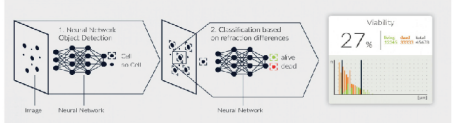
标准曲线

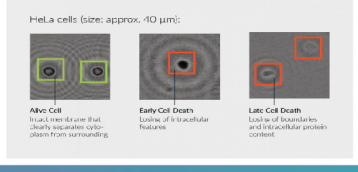


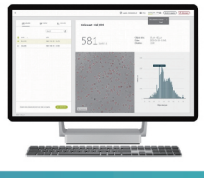
光谱分析

375-700nm波长
光谱扫描
OD600测定
动力学分析
小巧、便携









Bioquochem(BQC)总抗氧化能力检测仪

德国anvajo 免染色细胞计数仪&光谱仪

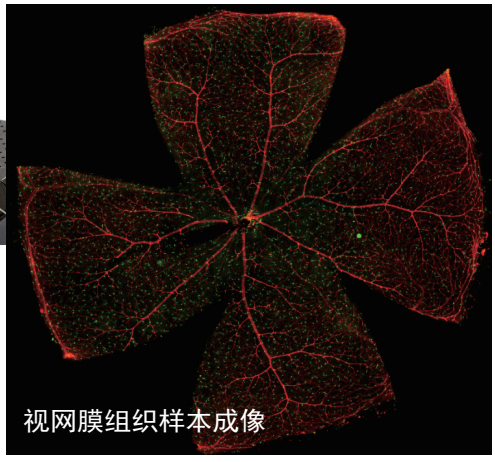
中国区总代理及技术服务中心：
上海怡赛科学仪器有限公司
Shanghai E-sci Scientific Instrument Co., LTD

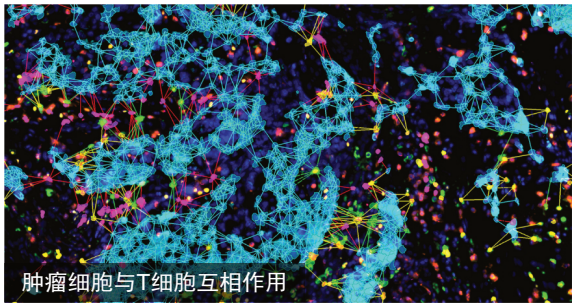
电话：021-67898557
邮箱：xpf@e-sci.com.cn
网址：www.e-sci.com.cn

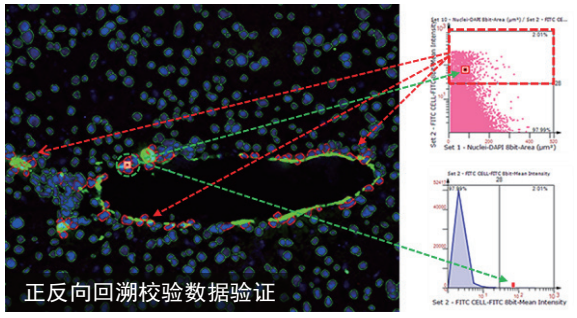
<<<TissueFAXS Q+ XY全景+Z轴全景深成像定量分析系统

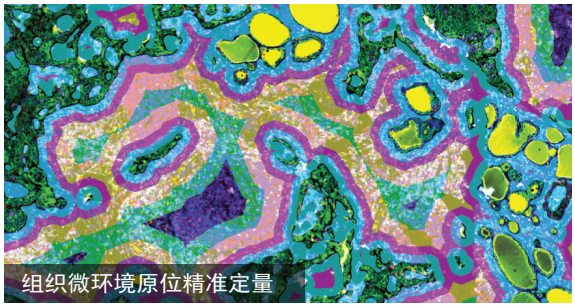
TissueFAXS Q+ 多维跨尺度高清超快速全景组织成像定量分析系统，可实现组织或器官层面跨模态、大尺度高清晰度共聚焦快速全景成像，以病人样本、实验动物样本、动物模型样本等为研究对象，深度挖掘组织（实体瘤、脏器、靶器官等）内或器官横切面蕴含的组织微环境分析大数据，构建临床研究辅助诊断体系，辅助临床科学研究实现精准诊疗，可以对医学实验动物（斑马鱼、大小鼠、实验猴、猪等）组织原位体内微环境进行深度解析和分析性研究，对体内蛋白或核酸原位精细定位量化分析、进行组织类流式分析，以及空间位置关系等微环境生物学研究。

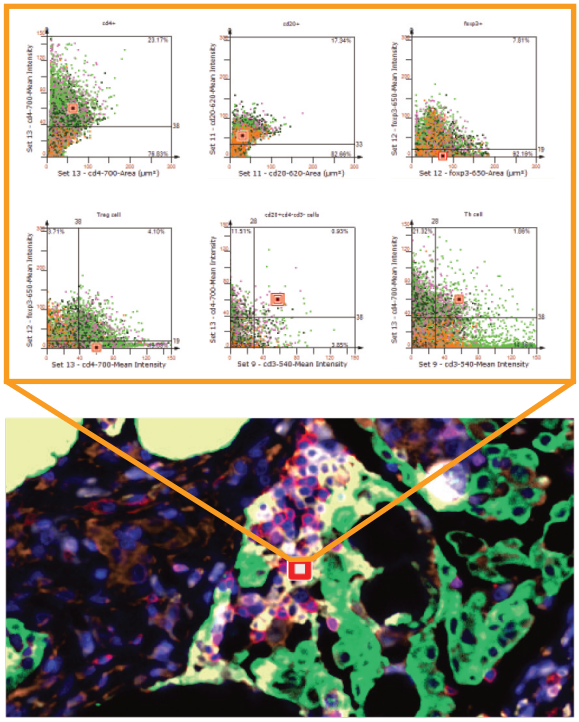



视网膜组织样本成像


肿瘤细胞与T细胞互相作用


正反向回溯校验数据验证


组织微环境原位精准定量



<<<神经学方向研究

神经干细胞、星形胶质细胞、脑功能、神经退行性疾病，如帕金森、阿尔兹海默等细胞体和神经突起的大小、长度、分支数量进行识别定量，并根据不同功能与不同区域，区分单极、多级神经元。利用形态学识别的方法，在多种不同样本类型中，识别出神经元胞体和突起。按照神经元外型的差异，在免疫组化样本中，对细胞体和神经突的大小、长度、分支数量进行识别定量，并根据不同功能与不同区域，区分单极、多级神经元。

北京华威中仪
展位号B5

Huawei
Better work . Better life

提供组织-细胞-线粒体多参数一站式实时检测方案

Provide a one stop real-time detection solution for tissue-cell-mitochondrial multi-parameters

OROBOROS INCYTON

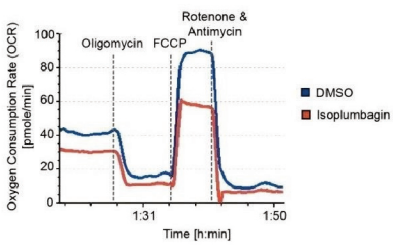
奥地利 OROBOROS O2k 高精度线粒体氧化磷酸化功能表征系统

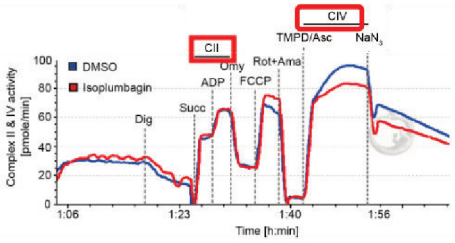
HIGHPRECISION MITOCHONDRIAL OXIDATIVE PHOSPHORYLATION FUNCTIONAL CHARACTERIZATION SYSTEM

多参数检测（Multi-parameter detection）：pO_2、pH、ATP、MMP、ROS、Ca^{2+}、CoQ、NADH、NO、H_2S、TPP^+
多样品适用（Suitable for multiple samples）：组织（Tissues）、细胞（cells）、线粒体（mitochondria）、血液（blood）、细菌（bacteria）
精准检测方式（Accurate detection method）：极谱氧电极传感器检测，精准度更高
Polarographic oxygen electrode sensor detection with higher accuracy
开放试剂（Open reagents）：可自行设计实验，同时提供 67 种 Protocol 可供选择
Design your own experiments and choose from 67 protocols
应用方向（Application direction）：Cancer、Cardiovascular、Neurological、Aging、Drugs、Exercise Physiology、 Mitochondrial Medicine etc

深度表征线粒体氧化磷酸化各个复合体的功能活性检测

In-depth characterization of mitochondrial oxidative phosphorylation of the functional activity of various complexes







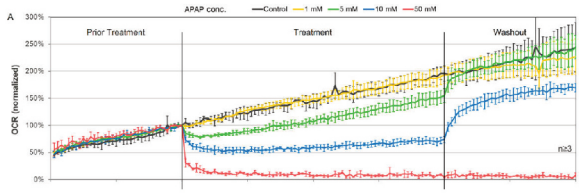
德国 INCYTON 智能多维度细胞长周期全息分析平台

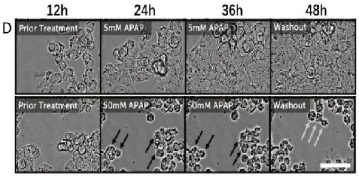
INTELLIGENT MULTI DIMENSIONAL CELL LONG CYCLE HOLOGRAPHIC ANALYSIS PLATFORM

技术特点（Technical features）：无标记检测（Mar freedetection）、多维度分析（multi dimensional analysis）、
长时间监测（long term monitoring）、全自动操作（fully automatic operation）
检测参数（Detection parameters）：pH、pO_2、Impedance、image
应用领域（Fields of application）：细胞毒理（Cytotoxicology）、安全性评价（safety evaluation）、细胞治疗研究（cell therapy research）、
肿瘤医学（Oncology），代谢组学（metabolomics）、药物筛选（Drug screening）
实时显微成像技术同步精确整合，实现几天、几周到数月的长时间细胞活性无标记监测与分析
Real-time microscopy technology is precisely integrated to enable label-free monitoring and analysis of cell viability over long periods of time, from days to months

对乙酰氨基酚(APAP)对人肝细胞(HepG2)毒性作用

Acetaminophen (APAP) toxic effects on human hepatocytes (HepG2)







OROBOROS INSTRUMENTS

INCYTON

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