Bridging Chinese Investigators Worldwide to Advance Life Sciences

Chinese Biological Investigators Society

11th Biennial Conference

July 29 – August 2, 2016

Chengdu, China

http://cbisociety.org/

Co-Organized by

State Key Laboratory of Biotherapy, West China Hospital, & Collaborative Innovation Center for Biotherapy, Sichuan University

with Support from the Municipal Government of Chengdu

Welcome Message

On behalf of the Board of Directors, we welcome you to Chengdu, China, for the 11th Biennial Conference of the Chinese Biological Investigators Society (CBIS). We have an outstanding program – prepared by the Program Committee with input from many of our members – that covers diverse topics in basic and translational research in life sciences as well as new technological developments. The program features 4 keynote speeches, 18 society (plenary) and special presentations, and 28 concurrent sessions. In addition, there will be 2 forums to share insights in career development, including managing laboratories, seeking research funds and publishing scientific papers, and to explore opportunities in entrepreneurial ventures.

Our goal is to provide a unique platform for you to connect with scientists who share the same passion in life sciences as you do, to interact with industrial leaders who are experienced in bringing scientific findings from bench to bedside, and to explore new prospects for academic and industrial collaborations.

At the meeting, we will announce the recipients of this year's Ray Wu Award to honor their scientific achievements and their efforts to promote life sciences in China as exemplified by Dr. Ray Wu. We will also present the CBIS Young Investigator Award, which recognizes our colleagues who are in the early career stages but have already made remarkable contributions in their respective fields, as well as the CBIS Teaching Award, which recognizes extraordinary contribution by a member to education in biomedical sciences in China.

We are particularly grateful this year for the support from the Municipal Government of Chengdu and the opportunity to co-organize this meeting with Sichuan University, specifically the State Key Laboratory of Biotherapy at West China Hospital and the Collaborative Innovation Center for Biotherapy under the leadership of Dr. Yuquan Wei. We are also very grateful to generous financial support from our sponsors, including the Cheerland Group, GenScript, AdvanTech, *Science China Life Sciences* and *Cell Research*.

We hope you will enjoy the hospitality of our local hosts and the interaction with students from Sichuan University. Chengdu is a vibrant city renowned for its food, cultural heritage and beautiful scenery. There are so many places to visit, both within the city and in the surrounding areas, for you and your family to enjoy.

We are delighted to have you here and hope you will have a memorable experience in Chengdu.

Weimin Zhong, Ph.D.
CBIS President 2014-2016

Guo-Min Li, Ph.D.
CBIS program Chair 2016

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Program at a Glance

(All meeting rooms are on the 3rd floor except the 6th floor Rose Hall/玫瑰厅)

Day 1, July 29, 2016, Friday

9:00 am – 10:00 pm	Arrival, registration and election ballot distribution (Hotel Lobby)
1:00 pm – 6:15 pm	CBIS and Ray Wu Memorial Fund (RWMF) Joint Presentation: "Ray Wu Symposium" (Lily Hall A/百合 A 厅)
1:15 – 2:15 pm	RWMF Keynote Lecture
2:15– 6:15 pm	RWMF Award Presentation and Lectures
6:30 pm – 10:00 pm	CBIS Reception, Banquet and Special Presentations (Lily Hall B/百合 B 厅)

Day 2, July 30, 2016, Saturday

8:45 – 9:25 am	11th CBIS Biennial Conference Opening Ceremony (Lily Hall A/百合 A 厅)
9:25 – 10:15 am	CBIS Keynote Lecture (Lily Hall A/百合 A 厅)
10:15 – 12:45 pm	Society Lectures I (Lily Hall A/百合 A 厅)
12:45 – 2:00 pm	*Lunch (1st Floor Cafeteria/一楼普罗旺斯西餐厅)*
2:00 – 4:00 pm	Concurrent Sessions 1: Non-Coding RNA (Lily Hall A/百合 A 厅) 2: Rewriting Genome (Lily Hall B/百合 B 厅) 3: Tumor Microenvironment and Metastasis (Peony Hall/牡丹厅) 4: Genome Maintenance (Hibiscus Hall/芙蓉厅) 5: Vision Research and Therapy (Crape Myrtle Hall/紫薇厅) 6: Infection and Host Defense (Daffodil Hall/水仙厅) 7: Inflammation and Autoimmune Diseases (Jasmine Hall/茉莉厅)
4:30 – 6:30 pm	Concurrent Sessions 8: Chromatin Remodeling (Lily Hall A/百合 A 厅) 9: New Frontiers in Gene Therapy (Crape Myrtle Hall/紫薇厅) 10: Signaling in Diseases (Daffodil Hall/水仙厅) 11: Development, Organ Formation and Physiology (Hibiscus Hall/芙蓉厅) 12: Late-Breaking Session I (Lily Hall B/百合 B 厅) 13: Metabolomics and Diseases (Peony Hall/牡丹厅) 14: Abstract Session I (Jasmine Hall/茉莉厅)
6:30 – 7:45 pm	*Dinner (on your own)*
7:45 – 10:30 pm	Panel Discussion I – Career Development (Hibiscus Hall/芙蓉厅)

Day 3, July 31, 2016, Sunday

8:30 – 10:45 am CBIS Keynote Lectures (6th Floor Rose Hall/六楼玫瑰厅)

11:15 – 12:45 pm Society Lectures II (6th Floor Rose Hall/六楼玫瑰厅)

12:45 – 2:00 pm *Lunch (1st Floor Cafeteria/一楼普罗旺斯西餐厅)*

1:00 – 7:00 pm Organized Sightseeing Tours

2:00 – 6:00 pm Group discussion with Sichuan provincial government officials, business leaders, and investors (Daffodil Hall/水仙厅)

8:00 – 10:00 pm Panel Discussion II – Entrepreneurship (Daffodil Hall/水仙厅)

Day 4, August 1, 2016, Monday

8:30 –10:30 am Society Lectures III (Lily Hall A/百合 A 厅)

10:50 - 12:45 pm CBIS Awards and Lectures (Lily Hall A/百合 A 厅)

12:40 – 2:00 pm *Lunch (1st Floor Cafeteria/一楼普罗旺斯西餐厅)*

2:00 – 4:00 pm Concurrent sessions

15: New Insights into Cancer Therapy (Lily Hall A/百合 A 厅)
16: RNA Modification and Chromosome Biology (Hibiscus Hall/芙蓉厅)
17: Structural Insights of Biology (Daffodil Hall/水仙厅)
18: New Insights into Neurodegeneration (Peony Hall/牡丹厅)
19: Brain Development and Diseases (Crape myrtle Hall/紫薇厅)
20: New Investigators Session I (Lily Hall B/百合 B 厅)
21: Abstract Session II (Jasmine Hall/茉莉厅)

4:30 – 6:30 pm Concurrent sessions

22: Plant Biology (Lily Hall A/百合A厅)
23: Stem Cell Biology (Peony Hall/牡丹厅)
24: Innovative Translational Medicine (Hibiscus Hall/芙蓉厅)
25: Late-Breaking session II (Lily Hall B/百合 B 厅)
26: New Investigators session II (Crape Myrtle Hall/紫薇厅)
27: Abstract Session III (Jasmine Hal/茉莉厅)
28: Abstract session IV (Daffodil Hall/水仙厅)

7:00 – 10:00PM Closing Banquet

Day 5, August 2, 2016, Tuesday *Departure*

Meeting Contacts: **Chong Chen (陈崇)** <chen_chong@yahoo.com> Tel: 189-8060-6532
Shangqin Guo <shangqin.guo@yale.edu>
Xiaoming Zhang <mzhang2@kumc.edu>

CBIS 11th Biennial Conference Program

Day 1, July 29, 2016, Friday

9:00 am – 10:00 pm	Arrival, registration and election ballot distribution (Hotel Lobby)
1:00 pm – 6:30 pm	CBIS and Ray Wu Memorial Fund (RWMF) Joint Presentation: "**2016 Ray Wu Symposium**" (Lily Hall A/百合 A 厅)
1:00 – 1:15	Introduction **Xiao-Hong Sun**, RWMF President, Oklahoma Medical Research Foundation
1:15 – 2:15	Keynote address **Jack Szostak**, Harvard University "The Origin of Cellular Life"
2:15 - 2:45	RWMF Award Presentation Moderators: **Junlin Guan**, University of Cincinnati **Qiang Yu**, Shanghai Institutes for Biological Sciences *Ray Wu Prize (RWP) of Excellence* *Gu Xiaocheng Lecture Award*
2:45 – 3:45	RWP Winner Presentations (I) Chair: Feng Shao, National Institute for Biological Sciences
2:45 – 3:00	**Yu Hou**, Peking University "Genome Analyses of Single Human Oocytes"
3:00 – 3:15	**Gaowen Liu**, Nanyang Technological University, Singapore "Adaptive evolution to the deletion of essential genes"
3:15 – 3:30	**Daisong Wang**, Shanghai Institutes for Biological Sciences "Identification of Multipotent Mammary Stem Cells by Protein C Receptor Expression"
3:30 - 3:45	**Lei Zhu**, Second Military Medical University "Effects of severing a normal S1 nerve root on the reconstruction of avulsed contralateral lumbosacral plexus"
3:45 -- 4:00	***Break***
4:00 – 6:15	RWP Winner Presentations (II) Chair: **Yimin Zou**, University of California, San Diego
4:00 – 4:15	**Biao Ma**, Nankai University "Hypoxia induced deactivation of the Hippo Signaling contributes to tumor growth and survival"
4:15 – 4:30	**Sai Luo**, Tsinghua University "Divergent lncRNAs regulate gene expression in pluripotent cells"
4:30 – 4:45	**Fan Zhou**, Academy of Military Medical Sciences "Tracing hematopoietic stem cell formation at single-cell resolution"

4:45 – 5:00 **Haibin Wang**, Second Military Medical University
"Biogenesis of Circular RNA and Its Potential Role of Diagnostic Biomarker in Spinal Osteoporosis"

5:00 – 5:15 **Xing Zhang**, Peking University
"Regulation on apical hook development by multiple hormones and light in Arabidopsis."

5:15 – 5:30 **Yulong Niu**, Sichuan University
"BnTR1 an E3 ligase from plant stabilizes heat shock factor σ32 of Escherichia coli by interacting with DnaK/DnaJ chaperon team"

5:30 – 6:00 **Gu Xiaocheng Lecture**
Bin Zhou, Professor, Shanghai Institutes for Biological Sciences
"Application of genetic lineage tracing in organ development and tissue regeneration"

6:00 – 6:15 GE China Presentation

6:15 Closing Remarks
Weimin Zhong, CBIS President, Yale University

6:30 – 7:00 pm Reception (all meeting participants and family members)
(Lily Hall Foyer/百合前厅)

7:00 – 10:00 pm Banquet and Special Presentations (registered participants and family)
(Lily Hall B/百合 B 厅)
Chair: **Weimin Zhong**, Yale University, weimin.zhong@yale.edu

7:00 – 8:50 Banquet and Video Presentation

8:50 – 9:20 CBIS Special Presentation
Chenjian Li, Peking University, li_chenjian@pku.edu.cn
"Daunting Tasks and Golden Opportunities in Reforming Chinese Education — A Global Citizen's Perspective"

9:20 – 9:50 CBIS Special Presentation
Ping Xiao (肖平), Deputy Director, Chengdu Library, cdtsg2007@126.com
"从照片看成都 (Chengdu History and Culture)"

Day 2, July 30, 2016, Saturday

8:45 – 9:25 am 11th CBIS Biennial Conference Opening Ceremony (Lily Hall A/百合 A 厅)
Chair: **Weimin Zhong**, Yale University, weimin.zhong@yale.edu

8:45 – 8:50 Opening Remarks
Weimin Zhong, CBIS President

8:50 – 9:00 Welcome Speech by Chengdu Government Representative

9:00 – 9:10 **Yan Shen (沈岩)**
Deputy Director, National Natural Science Foundation of China

9:10 – 9:20 **Heping Xie (谢和平)**
President, Sichuan University

9:25 – 10:45 am Keynote Session I (Lily Hall A/百合 A 厅)
Chair: **Guo-Min Li**, University of Southern California, guominli@usc.edu

9:25 – 10:15 **Richard Kolodner**, University of California, San Diego, rkolodner@ucsd.edu
"Genetic and Biochemical Dissection of Exonuclease 1-Dependent and -Independent Mismatch Repair"

10:15 – 10:45 **Wei Yang**, National Institutes of Health, weiy@niddk.nih.gov
"From DNA repair to a paradigm shift in enzyme catalysis"

10:45 – 11:15 am ***Coffee Break*** *(Lily Hall Foyer/百合前厅)*

11:15 – 12:45 pm Society Lectures I (Lily Hall A/百合 A 厅)
Chair: **Pan Zheng**, Children's National Health System
PZheng@childrensnational.org

11:15–11:45 **Chuan He**, University of Chicago, chuanhe@uchicago.edu
"RNA methylation in gene expression regulation "

11:45–12:15 **Bing Ren**, University of California, San Diego, biren@ucsd.edu
"Multi-dimensional analysis of the human genome"

12:15–12:45 **Feng Shao**, National Institute of Biological Sciences, shaofeng@nibs.ac.cn
"Pyroptosis in anti-bacteria immunity: sensing & execution."

12:45 – 2:00 pm *Lunch (speakers lunch with students and postdocs)*
(1st floor cafeteria/一楼普罗旺斯西餐厅)

2:00 – 4:00 pm **Concurrent Sessions**

Concurrent Session 1: Non-Coding RNA (Lily Hall A/百合 A 厅**)**
Co-Chairs: **Lianghu Qu**, Sun Yat-sen University, China, lssqlh@mail.sysu.edu.cn
Ling-Ling Chen, Shanghai Institute for Biological Sciences, linglingchen@sibcb.ac.cn

2:00 – 2:15 pm
Mofang Liu, Institute of Biochemistry and Cell Biology, CAS, mfliu@sibcb.ac.cn
"The new function of PIWI and piRNAs in mammalian spermatogenesis"

2:15 – 2:30 pm
Lin He, University of California at Berkeley, lhe@berkeley.edu
"miR-34a deficiency expanded the cell fate potential of pluripotent stem cells"

2:30 – 2:45 pm
Jun-An Chen, Institute of Molecular Biology, Academia Sinica, jachen@imb.sinica.edu.tw
"Exploring the functional role of non-coding RNA during motor neuron generation and degeneration"

2:45 – 3:00 pm
Yueqin Chen, Sun Yat-sen University, lsscyq@mail.sysu.edu.cn

"LncRNA in hematopoiesis and blood cancer"

3:00 – 3:15 pm
Liuqing Yang, the University of Texas MD Anderson Cancer Centre, LYang7@mdanderson.org
"Functional characterization of long noncoding RNAs in cancer signaling"

3:15 – 3:30 pm
Yijun Qi, Tsinghua University, qiyijun@mail.tsinghua.edu.cn
"Antisense transcripts regulates sense transcription and flowering time in Arabidopsis"

3:30 – 3:45 pm
Ge Shan, University of Science and Technology of China, shange@ustc.edu.cn
"Evolution of an lncRNA leads to a primate specific modulation of alternative splicing"

3:45 – 4:00 pm
Li Yang, CAS-MPG Partner Institute for Computational Biology, CAS, liyang@picb.ac.cn
"Genome-wide characterization of circular RNAs"

Concurrent Session 2: Rewriting Genome (Lily Hall B/百合 B 厅)
Co-Chairs: **Jingsong Li**, Shanghai Institutes for Biological Sciences, CAS, jsli@sibcb.ac.cn
Guangshuo Ou, Tsinghua University, guangshuoou@mail.tsinghua.edu.cn

2:00 – 2:20 pm
Weizhi Ji, Kunming University of Science and Technology
"Precision gene editing in nonhuman primates: approach to establishing human disease models"

2:20 – 2:40 pm
Liangxue Lai, Guangzhou Institutes of Biomedicine and Health, CAS, lai_liangxue@gibh.ac.cn
"Genome-editing pig models for biomedicine"

2:40 – 3:00 pm
Guangshuo Ou, Tsinghua University, guangshuoou@mail.tsinghua.edu.cn
"Using CRISPR-Cas9-assisted conditional knockout and knock-in to understand neuroblast migration in C. elegans"

3:00 – 3:12 pm
Ji-Long Liu, University of Oxford, ilong.liu@dpag.ox.ac.uk
"Knockdown of long noncoding RNAs by CRISPRi"

3:12 – 3:24 pm
Wensheng Wei, Peking University, wswei@pku.edu.cn
"CRISPR screening in the study of human disease"

3:24 – 3:36 pm
Zi-Long Qiu, Institute of Neuroscience, CAS, zqiu@ion.ac.cn
"Transgenic monkey as autism animal model"

3:36 – 3:48 pm
Haoyi Wang, Institute of Zoology, CAS, wanghaoyi@ioz.ac.cn
"CRISPR-Cas9 mediated genome editing in mouse model creation and Gene regulation"

3:48 – 4:00 pm
Hui Yang, Institute of Neuroscience, CAS,, huiyang@ion.ac.cn
"Chromosome engineering by CRISPR-Cas systems"

Concurrent Session 3: Tumor Microenvironment and Metastasis (Peony Hall/牡丹厅)

Co-Chairs: **Xiao-Fan Wang**, Duke University, xiao.fan.wang@duke.edu
Xiang Zhang, Baylor College of Medicine, xiangz@bcm.edu

2:00 - 2:30 pm
Xiang Zhang, Baylor College of Medicine, xiangz@bcm.edu
"CD4+ T cells restrict cancer cell intravasation via promoting vessel normalization"

2:30 – 3:00 pm
Min Yu, University of Southern California, minyu@usc.edu
"Understanding breast cancer metastasis and progression using patient-derived circulating tumor cells"

3:00 – 3:30 pm
Qing Chen, Wistar institute, USA, qichen@wistar.org
"Carcinoma-astrocyte gap junctions promote brain metastasis by cytosolic dsDNA response transfer"

3:30 – 4:00 pm
Guohong Hu, Shanghai Institute for Biological Sciences, China, ghhu@sibs.ac.cn
"The Role of small RhoGTPase inhibitor DLC1 in breast cancer bone metastasis"

Concurrent Session 4: Genome Maintenance (Hibiscus Hall/芙蓉厅)
Co-Chairs: **Junjie Chen**, MD Anderson Cancer Center, jchen8@mdanderson.org
Jean Wang, University of California, San Diego, jywang@ucsd.edu

2:00 – 2:20 pm
Zhou Songyang –Baylor College of Medicine,, songyang@bcm.edu
"Telomere maintenance and human diseases"

2:20 – 2:40 pm
Junjie Chen – MD Anderson Cancer Center, jchen8@mdanderson.org
"Regulation of DNA repair pathways"

2:40 – 3:00 pm
Jean Wang – University of California, San Diego, jywang@ucsd.edu
"DNA damage induced apoptosis"

3:00 – 3:12 pm
Huilin Zhou – University of California, San Diego, huzhou@ucsd.edu
"Sumoylation of replisome and genome instability"

3:12 – 3:24 pm
Dong Wang – University of California, San Diego, dongwang@ucsd.edu
"Transcription coupled repair and UV response"

3:24 – 3:36 pm
Lee Zou – MGH/Harvard University, USA, zou.lee@mgh.harvard.edu
"DNA damage checkpoint kinases and cancer therapy"

3:36 – 3:48 pm
Yong Wan –University of Pittsburgh, yow4@pitt.edu
"The role of ubiquitin-proteasome system in genome maintenance"

3:48 – 4:00 pm
Jun Huang – Zhejiang University, jhuang@zju.edu.cn

"New players in DNA damage response pathways"

Concurrent Session 5: Vision Research and Therapy (Crape Myrtle Hall/紫薇厅)

Co-Chairs: **Fu-Shin Yu**, Wayne State University, fyu@med.wayne.edu
Xiaohua Gong, University of California, Berkeley, xgong@berkeley.edu

2:00 – 2:16 pm
Fu-Shin Yu, Wayne State University, fyu@med.wayne.edu
"Dendritic Cell Dysfunction and Diabetic Sensory Neuropathy in the Cornea"

2:16 – 2:28 pm
Mingwu Wang, University of Arizona, mwang@eyes.arizona.edu
"Is the lacrimal gland indispensable?"

2:28 – 2:48 pm
Xian-Jie Yang, University of California, Los Angeles, yang@jsei.ucla.edu
"Neuroprotection of the retina, promises and challenges"

2:48 – 3:08 pm
Zhenglin Yang, University of Electronic Science and Technology of China, zliny@yahoo.com
"Molecular Genetics of Primary Open Angle Glaucoma"

3:08 – 3:24 pm
Lu Chen, University of California, Berkeley,, chenlu@berkeley.edu
"Novel insights into corneal lymphangiogenesis and beyond"

3:24 – 3:40 pm
Zhuo-Hua Pan, Wayne State University, USA, zhpan@med.wayne.edu
"Optogenetic gene therapy for vision restoration"

3:40 – 4:00 pm
Xiaohua Gong, University of California at Berkeley, xgong@berkeley.edu
"Gap junction and genetics variances in cataract formation and prevention"

Concurrent Session 6: Infection and Host Defense (Daffodil Hall/水仙厅)

Co-Chairs: **Feng Shao**, National Institute of Biological Sciences, shaofeng@nibs.ac.cn
Genhong Cheng, University of California, Los Angeles, gcheng@mednet.ucla.edu

2:00 – 2:15 pm
Xinnian Dong, Duke University, USA, xdong@duke.edu
"DNA damage repair, cell cycle regulators, and plant immunity"

2:15 – 2:30 pm
Lishan Su, University of North Carolina, Chapel Hill, lishan_su@med.unc.edu
"Plasmacytoid dendritic cells (pDC) and interferon in HIV-1 pathogenesis and therapy"

2:30 – 2:45 pm
Yang Liu, Children's National Medical Center, yaliu@childrensnational.org
"Sialoside-based pattern recognition, autoimmunity and metabolic syndrome"

2:45 – 3:00 pm
George F. Gao, Institute of Microbiology, CAS, gaof@im.ac.cn
"Ebola virus entry and fusion"

3:00 – 3:15 pm

Wenhui Li, National Institute of Biological Sciences, liwenhui@nibs.ac.cn
"HBV infection: from entry to persistence"

3:15 – 3:30 pm
Zhao-Qing Luo, Purdue University, luoz@purdue.edu
"Ubiquitination lessons taught by a bacterial pathogen"

3:30 – 3:45 pm
Xin Lin, Tsinghua University, linxin307@mail.tsinghua.edu.cn
"Innate immune response against fungal infection."

3:45 – 4:00 pm
Sudan He, Soochow University, hesudan@suda.edu.cn
"Necroptosis in host defense against viral infection"

Concurrent Session 7: Inflammation and autoimmune diseases (Jasmine Hall/茉莉厅)
Co-Chairs: **Chen Dong**, Tsinghua University, China, chendong@tsinghua.edu.cn
Bing Su, Shanghai JiaoTong University, bingsu@sjtu.edu.cn

2:00 – 2:20 pm
Xiaoyu Hu, Tsinghua University, xiaoyuhu@tsinghua.edu.cn
"Attenuation of neutrophil-mediated inflammation by transcription repressor Hes1"

2:20 – 2:40 pm
Hongyan Wang, Shanghai Institutes for Biological Sciences, hongyanwang@sibcb.ac.cn
"Identification of new effectors in the regulation of septic shock and multiple sclerosis"

2:40 – 3:00 pm
Wanjun Chen, National Institutes of Health, wchen@dir.nidcr.nih.gov
"In vivo generation of autoantigen-specific T regulatory cells to treat autoimmune diseases"

3:00 – 3:20 pm
Xinyuan Fu, National University of Singapore, fulabnus2012@yahoo.com
TBD

3:20 – 3:40 pm
Huji Xu, Changhai Hospital, Second Military University, huji.xu@uq.edu.au
TBD

3:40 – 4:00 pm
Bing Su, Shanghai JiaoTong University, Shanghai Institute of Immunology, bingsu@sjtu.edu.cn
"Mechanistic target of rapamycin in lymphocyte development and function"

4:00 - 4:30 pm ***Coffee Break*** *(Lily Hall Foyer/百合前厅)*

4:30 – 6:30 pm **Concurrent Sessions**

Concurrent Session 8: Chromatin Remodeling (Lily Hall A/百合 A 厅)
Co-Chairs: **Bing Zhu**, Institute of Biophysics, CAS, China, zhubing@ibp.ac.cn
Zhiguo Zhang, Mayo Clinic, USA, zhang.zhiguo@mayo.edu

4:30 – 4:47 pm
Zhiguo Zhang, Mayo Clinic, zhang.zhiguo@mayo.edu

"Impacts of histone mutations on cancer epigenomes"

4:47 – 5:04 pm
Zheng Zhou, Institute of Biophysics, CAS, zhouzh@ibp.ac.cn
"Structure basis for recognition of histone variant H2A.Z by histone chaperones"

5:04 – 5:21 pm
Jiemin Wong, East China Normal University, jmweng@bio.ecnu.edu.cn
Control of DNA maintenance methylation by UHRF1 and USP7

5:21 – 5:38 pm
Guohong Li, Institute of Biophysics, CAS, liguohong@ibp.ac.cn
"Structure and dynamics of the 30-nm chromatin fiber in gene regulation"

5:38 – 5:55 pm
Fei Lan, Fudan University, fei_lan@fudan.edu.cn
"Mis-regulation of Enhancer in Cancer"

5:55 – 6:12 pm
Bing Zhu, Institute of Biophysics, CAS, zhubing@ibp.ac.cn
"Dynamic regulation of DNA methylation"

6:12 – 6:30 pm
Jiang Liu, Beijing Institute of Genomics, CAS, liuj@big.ac.cn
"The inheritance and programming of parental DNA methylation in animals"

Concurrent Session 9: New Frontiers in Gene Therapy (Crape Myrtle Hall/紫薇厅)
Co-Chairs: **Xiao Xiao**, University of North Carolina, xxiao@email.unc.edu
Daowen Wang, University of Science and Technology, China, dwwang@tjh.tjmu.edu.cn

4:30 – 4:50 pm
Guangping Gao, University of Massachusetts Medical School, Guangping.Gao@umassmed.edu
"Gene therapy for neurological diseases"

4:50 – 5:10 pm
Xiao Xiao, University of North Carolina, xxiao@email.unc.edu
"From mouse to dog to human: A long journey of muscular dystrophy gene therapy"

5:10 – 5:30 pm
Yang Yang, Sichuan University, yang21@mail.med.upenn.edu
"Correction of a metabolic disease following in vivo delivery of CRISPR/Cas9"

5:30 – 5:50 pm
Daowen Wang, Tongji Hospital, Huazhong University of Science and Technology,,
dwwang@tjh.tjmu.edu.cn
"MicroRNA gene delivery for treatment of hypertension"

5:50 – 6:10 pm
Yang Lin, Sichuan University, yanglin0@hotmail.com
"Bioengineering of viral vectors for tissue-targeting"

6:10 – 6:30 pm
Chunbo Zhang, Nanchang University, zhangcbspring@gmail.com
"Regulation of transgene expression in mouse liver"

Concurrent Session 10: Signaling in Diseases (Daffodil Hall/水仙厅)

Co-Chairs: **Zhixiong Xiao**, Sichuan University, China, jimzx@scu.edu.cn
Xin-Hua Feng, Zhejiang University, China, xfeng@bcm.edu

4:30 – 4:45 pm
Xin-Hua Feng, Zhejiang University, xfeng@bcm.edu
"Introduction to signaling"

4:45 – 5:00 pm
Zhiyuan Shen, Rutgers University, shenzh@rutgers.edu
"Tumor suppressor genes rarely mutated in cancer"

5:00 – 5:15 pm
Qing Zhong, UT Southwestern Medical Center,, Qing.Zhong@UTSouthwestern.edu
"Biochemical dissection and reconstitution of mammalian autophagy"

5:15 – 5:30 pm
Xiao Yang, Beijing Institute of Biotechnology, yangx@bmi.ac.cn
"TGF-beta signaling and cardiovascular diseases"

5:30 – 5:45 pm
Pinglong Xu, Zhejiang University, xupl@zju.edu.cn
Dynamic phosphorylation governs cytosolic nucleic acid sensing and antiviral defense.

5:45 – 6:00 pm
Xi Chen, Baylor College of Medicine, xi.chen@bcm.edu
"Unfolding the Unfolded Protein Response in Breast Cancer"

6:00 – 6:15 pm
Dewang Zhou, Xiamen University, dwzhou@xmu.edu.cn
"Pharmacological targeting of kinases Mst1 and Mst2 augments tissue repair and regeneration"

6:15 – 6:30 pm
Zhixiong Xiao, Sichuan University, jimzx@scu.edu.cn
"Oncogenic signalings and cancer Metastasis"

Concurrent Session 11: Development, Organ Formation & Physiology (Hibiscus Hall/芙蓉厅)

Co-Chairs: **Xin Sun**, University of Wisconsin, xsun@wisc.edu
Tian Xu, Yale University, tian.xu@yale.edu

4:30 – 4:50 pm
Tian Xu, Yale University, tian.xu@yale.edu
"Regulation of growth and tissue size by size organizer, localized JNK"

4:50 – 5:10 pm
Yingzi Yang, Harvard University, yingzhi_yang@hsdm.harvard.edu
"Facing the wind: Wnt regulated planar cell polarity"

5:10 – 5:30 pm
Qiang Chang, University of Wisconsin, qchang@waisman.wisc.edu
"Astrocyte dysfunction in Rett syndrome"

5:30 – 5:45 pm
Jingsong Li, Shanghai Institutes for Biological Sciences, jsli@sibcb.ac.cn
"Artificial sperm: generation and application"

5:45 – 6:00 pm
Ting Xie, Stowers Institute, tgx@stowers.org
"Niche and intrinsic control of the self-renewal-to-differentiation switch"

6:00 – 6:15 pm
Yi Zeng, Shanghai Institutes for Bilogical Sciences, yzeng@sibcb.ac.cn
"Protein C receptor as a surface marker for mammary stem cells and its implication in breast cancer"

6:15 – 6:30 pm
Xin Sun, University of Wisconsin, xsun@wisc.edu
"The lung as a sensory organ: linking development to physiology:

Concurrent Session 12: Late-Breaking Session I (Lily Hall B/百合 B 厅)
Co-Chairs: **Xinnian Dong**, Duke University, xdong@duke.edu
Xiang-Dong Fu, University of California, San Diego, xdfu@ucsd.edu

4:30-4:54 pm
Jie Xiao, Johns Hopkins School of Medicine, xiao@jhmi.edu
"Probing transcription dynamics in live cells at the single molecule level"

4:54-5:18 pm
Y. Jessie Zhang, University of Texas, Austin, jzhang@cm.utexas.edu
"Mapping the phosphorylation pattern of eukaryotic RNA polymerase II"

5:18-5:42 pm
Zhiyong Wang, Carnegie Institution for Science, zywang24@stanford.edu
"Growth control under starvation through TOR-steroid crosstalk"

5:42-6:06 pm
Jing Chen, Emory University School of Medicine jchen@emory.edu
"Diet-fueled metabolic rewiring in human cancer"

6:06-6:30 pm
Wei Xu, University of Wisconsin-Madison, wxu@oncology.wisc.edu
"Protein arginine methylation in breast cancer"

Concurrent Session 13: Metabolomics and Diseases (Peony Hall/牡丹厅)
Co-Chairs: **Feng Liu**, University of Texas Health Science Center at San Antonio,, liuf@uthscsa.edu
Baoliang Song, Wuhan University, blsong@whu.edu.cn

4:30 – 4:45 pm
Baoliang Song, Wuhan University, blsong@whu.edu.cn
"Cholesterol transport through lysosome-peroxisome membrane contacts"

4:45 – 5:00 pm
Ming C. Gong, University of Kentucky, mcgong2@uky.edu
"Non-dipping circadian blood pressure in diabetes"

5:00 – 5:15 pm
Yi Yang, East China University of Science and Technology, yiyang@ecust.edu.cn
"Visualize and manipulate cellular metabolic states using optogenetic probes"

5:15 – 5:30 pm
Yingjie Wu, Mount Sinai School of Medicine (email?)
"Growth hormone receptor and β cell: Novel insight into mice models"

5:30 – 5:45 pm
Yi Tian, University of Louisville, y0tan002@louisville.edu
"Uncoupling of the mitogenic and metabolic FGF1 and the application in type 2 diabetes"

5:45 – 6:00 pm
Wenke Feng, University of Louisville, wenke.feng@louisville.edu
"Gut microbiota and alcoholic fatty liver disease"

6:00 – 6:15 pm
Aimin Xu, Hong Kong University, amxu@hku.hk
TBA

6:15 – 6:30 pm
Weiping Zhang, Second Military Medical University, wzhang@smmu.edu.cn
"Transcriptional regulation of hepatic lipogenesis by ZBTB20"

Concurrent Session 14: Abstract Session I (Jasmine Hall/茉莉厅)

Co-Chairs: **Jin Zhang**, University of California, San Diego, jzhang32@ucsd.edu
Dihua Yu, MD Anderson Cancer Center, dyu@mdanderson.org

4:30 - 4:45 pm
Dihua Yu, MD Anderson Cancer Center, dyu@mdanderson.org
"PTEN down regulation by astrocyte-derived exosomal microRNA promotes brain metastasis outgrowth"

4:45 - 5:00 pm
Lin-Feng Chen, University of Illinois at Urbana-Champaign, lfchen@life.illinois.edu
"Regulation of NF-kappaB signaling by Bromodomain-containing factor Brd4"

5:00 - 5:15 pm
Zhe Han, Children's National Medical Center, zhan@childrensnational.org
"Modeling Heart and Kidney Disease Genetics in Drosophila"

5:15 - 5:30 pm
Jian Zhang, Ohio State University, jian.zhang@osumc.edu
"Targeting Cbl-b as a Potential Therapeutical Approach for Disseminated Candidiasis"

5:30 - 5:45 pm
Xuewu Zhang, UT Southwestern Medical Center, xuewu.zhang@utsouthwestern.edu
"Regulation mechanisms of the guidance receptor plexin"

5:45 - 6:00 pm
Jing Wang, University of Nebraska Medical Center, jjwang@unmc.edu
"TGF-β mediates drug resistance by regulating PDK4 expression in colorectal cancer"

6:00 - 6:15 pm
Wen Xie, University of Pittsburgh, wex6@pitt.edu
"Transcriptional Regulation of Disease-Drug Interactions"

6:15 - 6:30 pm
Jin Zhang, UCSD, jzhang32@ucsd.edu
"Illuminating the Biochemical Activity Architecture of the Cell"

6:30 – 7:45 pm Dinner (on your own)

7:45 – 10:30 pm Panel Discussions I (Hibiscus Hall/芙蓉厅)
Career Development: Managing Lab, Seeking Funds & Publishing Papers
(Sponsored by ***Cell Research***, http://www.nature.com/cr/index.html)

Co-Chairs: **Feng Shao**, NIBS, shaofeng@nibs.ac.cn
Yimin Zou, University of California, San Diego, yzou@ucsd.edu

7:50 - 8:00 **Yan Shen**, China National Natural Science Foundation

8:00 - 8:10 **Boqin Qiang**, Peking Union Medical College

8:10 - 8:25 **Dangsheng Li**, Cell Research
"Publishing in high-profile journals"

8:25 - 10:30 Questions/Answers
Panelists:
Yan Shen, National Natural Science Foundation of China
Boqin Qiang, Peking Union Medical College
Yifan Cheng, University of California, San Francisco, YCheng@ucsf.edu
Xinnian Dong, Duke University, xdong@duke.edu
Dangsheng Li, Cell Research, dsli@sibs.ac.cn
Yanhui Xu, Fudan University, xuyh@fudan.edu.cn
Wei Yang, National Institutes of Health, weiy@niddk.nih.gov
Xiang Yu, Shanghai Institute for Biological Sciences, yuxiang@ion.ac.cn
Junying Yuan, Harvard University, junying_yuan@hms.harvard.edu
Qi Zhou, Institute of Zoology, CAS, qzhou@ioz.ac.cn

Day 3, July 31, 2016, Sunday

8:30 – 10:45 am Keynote Session II (6th Floor Rose Hall/六楼玫瑰厅)
Chair: **Yuquan Wei**, Sichuan University, yuquanwei@scu.edu.cn

8:30 – 9:15 am **Carlo Croce**, Ohio State University, carlo.croce@osumc.edu
"Causes and consequences of microRNA dysregulation in cancer"

9:15 – 9:45 am **Yuquan Wei**, Sichuan University yuquanwei@scu.edu.cn
"Translational Medicine in Sichuan University"

9:45 – 10:15 am **James Chen**, UT Southwestern Medical Center
,zhijian.chen@utsouthwestern.edu
"The dark side of DNA - immune and autoimmune responses to cytosolic DNA"

10:15 – 10:45 am **Don Cleveland,** University of California, San Diego, dclevelan@ucsd.edu
"Gene silencing therapy for human neurodegenerative disease"

10:45 – 11:15am ***Coffee Break*** *(Rose Hall Foyer/玫瑰厅外)*

11:15 – 12:45 pm Society Lectures II (6th Floor Rose Hall/六楼玫瑰厅)
Chair: **Lei Li**, MD Anderson Cancer Center, leili@mdanderson.org

11:15 – 11:45 **Junying Yuan**, Harvard University, jyuan@hms.harvard.edu
"Regulation of necroptosis and inflammation by RIPK1"

11:45 –12:15 **Qi Zhou.** Institute of Zoology, CAS, qzhou@ioz.ac.cn
"Progress of stem cell research and regenerative medicine in China"

12:15 – 12:45 **Changyu Wang**, Huamian Biotechnology Co., changyu_7963@yahoo.com
Cancer immunotherapy: PD-1 and beyond

12:45 – 2:00 pm *Lunch (1st Floor Cafeteria/一楼普罗旺斯西餐厅)*

1:00 – 7:00 pm Organized tours (select one; box lunch and dinner provided)
1) Sanxingdui (三星堆)
2) Dujiang Dam (都江堰)
3) Dayi Liu Family Museum (大邑刘氏庄园博物馆)

2:00 – 6:00 pm Group discussion with Sichuan provincial government officials, local business leaders, and investors about technology transfer and entrepreneurship (Daffodil Hall/水仙厅)

生物医药项目对接会 (14:00-18:00, 水仙厅)
主办：四川省人才办公室
承办：华人生物学家协会、省千人计划专家联谊会
议程：
（1）省人才办负责人介绍会议目的和相关人才政策
（2）华人生物学家协会负责人介绍协会情况
（3）重大科技成果转化项目路演
（4）自由讨论和项目对接

8:00 – 10:00 pm Panel Discussions II (Daffodil Hall/水仙厅)
Special forum on biopharmaceutical industry, entrepreneurship and opportunities

Co-Chairs: **Charlene Liao,** Immune-Onc Therapeutics, Inc., xcliao@yahoo.com
Li Zhu, GenScript Corporation, li.zhu@genscript.com

Moderator: **Min Li**, SVP, Global Head of Neuroscience and General Manager of R&D China, GSK, min.x.li@gsk.com

8:00 – 8:10 **Charlene Liao** (廖晓伶, PhD, President and CEO of Immune-Onc Therapeutics, Inc., xcliao@yahoo.com)
Innovative strategies to develop a combination therapy of two novel monoclonal antibodies

8:10 – 8:20 **Alex Wu** (吴越, PhD, President of Crown Bioscience, Inc.)
Building a bridge between academia and industry

8:20 – 8:30 **Jingyi Xiang** (向京宜, PhD, Head of Scientific Alliances, Eureka Therapeutics, Inc.)

Innovating antibody and adoptive T-cell therapies for targeting "undruggable" cancer antigens

8:30 – 8:40 **Qiang Lu** (吕强, PhD, SVP of CStone Pharmaceuticals Limited)
Building best biotech company in China under fast evolving innovation ecosystem

8:40 – 8:50 **Darren Ji** (纪晓辉, PhD, Vice President, Global Head, Asia and Emerging Markets Partnering, F. Hoffmann-La Roche)
The value of partnering early in science to accelerate drug innovation

8:50 – 9:00 **Li Zhu** (朱力, PhD, VP Strategy, GenScript Corporation)
li.zhu@genscript.com
How does GenScript evolve from a start-up to the number one gene synthesis company in the world?

9:00 – 10:00 Panel Discussion

Day 4, August 1, 2016, Monday

8:30 –10:30 am Society Lectures III (Lily Hall A/百合 A 厅)
Chair: **Hao Wu**, Harvard University, wu@crystal.harvard.edu

8:30 – 9:00 **Yi Rao**, Peking University, yrao@pku.edu.cn
"Molecular genetic analysis of sleep**"**

9:00 – 9:30 **Zhigang He**, Harvard University, Zhigang.He@childrens.harvard.edu
"Towards rebuilding functional neuronal circuits in the adult CNS"

9:30 – 10:00 **Yifan Cheng,** University of California, San Francisco, ycheng@ucsf.edu
"Structural biology of membrane protein by single particle cryo-EM"

10:00 – 10:30 **Min Li,** GlaxoSmithKline, min.x.li@gsk.com
"Academic, government and industry - partnership with excellence and scale"

10:30 – 10:50 am ***Coffee Break (Location: Lily Hall Foyer/百合前厅)***

10:50 - 12:45 pm CBIS Awards and Lectures (Lily Hall A/百合 A 厅)
Chair: **Yibing Kang**, Princeton University, ykang@Princeton.edu

10:50 – 11:50 Ray Wu Award Lectures

10:50 – 11:20 **Xiaoliang Sunney Xie**, Harvard University, xie@chemistry.harvard.edu
"Precision Genomics at the Single Molecule Level"

11:20 – 11:50 **Xiang-Dong Fu**, University of California, San Diego, xdfu@ucsd.edu
"Global Analysis of RNA-Chromatin Interactions Reveals Cell Type-Specific Transcriptional Hubs in 3D Genome"

11:50 – 12:45 pm Young Investigator Award Lectures
(Sponsored by ***Science China Life Sciences*****,** http://life.scichina.com**)**

11:50 – 12:15 **Ling-Ling Chen**, Shanghai Institutes for Biological Sciences,

linglingchen@sibcb.ac.cn
"Unusual processing generates long noncoding RNAs with new formats"

12:15 – 12:40 **Hai Qi**, Tsinghua University, qihai@tsinghua.edu.cn
"Visualizing cell-cell interactions in humoral immune responses"

12:40 – 2:00 pm *Lunch (speakers lunch with students and postdocs)*
(1st Floor Cafeteria/一楼普罗旺斯西餐厅)

2:00 – 4:00 pm Concurrent sessions

Concurrent Session 15: New Insights into Cancer Therapy (Lily Hall A/百合 A 厅)
Co-Chairs: **Yang Liu**, Children's National Medical Center, yaliu@childrensnational.org
Chong Chen, Sichuan University, chen_chong@yahoo.com

2:00 – 2:20 pm
Shengyong Yang, Sichuan University, yangsy@scu.edu.cn
"Discovery of a drug candidate against triple negative breast cancer"

2:20 – 2:40 pm
Yin Wang, The Children's Research Institute, ywang@childrensnational.org
"Therapeutic effect of echinomycin on acute lymphoblastic leukemia"

2:40 – 3:00 pm
Yu (Kiki) Liu, Sichuan University, yuliu_scu@yahoo.com
"Impact of chromosome large deletions on cancer"

3:00 – 3:15 pm
Penghui Zhou, Sun Yat-sen University, b01713@sysucc.org.cn
"Systemic Activation of Host Immunity against Cancer"

3:15 – 3:30 pm
Hanshuo Yang, Sichuan University, yhansh@126.com
"Eradication of established large tumor by bacterial nanoparticle as the vascular disruptor and drug carrier"

3:30 – 3.45 pm
Yong Peng, Sichuan University, pengyong10@hotmail.com
"ERK-mediated phosphorylation of exportin-5 suppress miRNA export to increase taxol resistance in liver cancer"

3:45 – 4:00 pm
Wei Wang, Sichuan University, weiwang@scu.edu.cn
"CAR-T based cancer immunotherapy: from hematological malignancies to solid tumors"

Concurrent Session 16: RNA Modification and Chromosome Biology (Hibiscus Hall/芙蓉厅)
Co-Chairs: **Hongtao Yu**, UT Southwestern Medical Center, Hongtao.Yu@UTSouthwestern.edu
Yungui Yang, Beijing Institute of Genomics, CAS, China, ygyang@big.ac.cn

2:00 – 2:15 pm
Yungui Yang, Beijing Institute of Genomics, CAS, ygyang@big.ac.cn
"Epitranscriptomic RNA modifications: Regulations and mechanisms"

2:15 – 2:30 pm

Chengqi Yi, Peking University, chengqi.yi@pku.edu.cn
"Sequencing nucleic acid modifications with epigenetic significance"

2:30 – 2:45 pm
Li Lan, University of Pittsburgh, lil64@pitt.edu
"RNA-templated recombination at active transcription sites upon damage"

2:45 – 3:00 pm
Hailin Wang, Research Center for Eco-Environmental Sciences, CAS, hlwang@rcees.ac.cn
"N-6-methyladenine DNA modification in high eukaryotes"

3:00 – 3:15 pm
Guoliang Xu, Shanghai Institute for Biological Sciences, CAS, glxu@sibcb.ac.cn
"Epigenetic regulation by enzymatic DNA oxidation"

3:15 – 3: 30 pm
Pumin Zhang, Baylor College of Medicine,, pzhang@bcm.edu
"The anaphase-promoting complex in DNA damage repair"

3:30 – 3:45 pm
Huiyan Li, National Center of Biomedical Analysis
"Molecular controls of mitotic progression and chromosome segregation"

3:45 – 4:00 pm
Yanbin Zhang, University of Miami, yzhang4@med.miami.edu
"Role of FANCA in DNA repair"

Concurrent Session 17: Structural Insights of Biology (Daffodil Hall/水仙厅)
Co-Chairs: **Wenqing Xu,** University of Washington, wxu@u.washington.edu
Yanhui Xu, Fudan University, xuyh@fudan.edu.cn

2:00 – 2:22pm
Eric Xu, Van Andel Research Institute, Eric.Xu@vai.org
"An X-ray laser structure of phosphorylated rhodopsin-arrestin complex"

2:22 – 2:44 pm
Ming Lei, National Center for Protein Science Shanghai, leim@sibcb.ac.cn
"Structural Insight into MLL family histone methyltransferases"

2:44 – 2:59 pm
Ning Gao, Tsinghua University, ninggao@mail.tsinghua.edu.cn
"Cryo-EM Structure of the late Nuclear Pre-60S particles at 3.0 Å"

2:59 – 3:14pm
Wenqing Xu, University of Washington, wxu@u.washington.edu
"Mechanism of PARylation-dependent polyubiquitination in Wnt signaling and beyond"

3:14 – 3:29pm
Xuelian Luo, UT Southwestern Medical Center, Xuelian.Luo@UTSouthwestern.edu
"Activation mechanisms of the Hippo kinase cascade"

3:29 – 3:44 pm
Yanli Wang, Institute of Biophysics, CAS, ylwang@sun5.ibp.ac.cn
"How does the CRISPR-Cas system defend against the invasive nucleic acids?"

3:44 – 4:00 pm

Yanhui Xu, Fudan University, xuyh@fudan.edu.cn
"Structural basis for the regulation of DNA methylation"

Concurrent Session 18: New Insights into Neurodegeneration (Peony Hall/牡丹厅)
Co-Chairs: **Xiao-Jiang Li**, Emory University, xli2@emory.edu
Fen-Biao Gao, UMass Medical School, fen-biao.gao@umassmed.edu

2:00 – 2:15 pm
Xiongwei Zhu, Case Western Reserve University, xiongwei.zhu@case.edu
"Abnormal mitochondrial dynamics as a potential therapeutic target of Alzheimer disease"

2:15 – 2:30 pm
Yong Shen, University of Science and Technology of China, yongshen@ustc.edu.cn
"BACE1 and neurodegenerative disorders"

2:30 – 2:45 pm
Zhenyu Yue, Icahn School of Medicine, Mount Sinai, zhenyu.yue@mssm.edu
"Vesicle trafficking and Parkinson's disease"

2:45 – 3:00 pm
Xiangdong William Yang, University of California, Los Angeles, xwyang@mednet.ucla.edu
"Integrated genetics and genomics to dissect Huntington's disease pathogenesis in mice"

3:00 – 3:15 pm
Xiao-Jiang Li, Emory University, xli2@emory.edu
"Genetically modified monkey models of neurodegenerative diseases"

3:15 – 3:30 pm
Fen-Biao Gao, University of Massachusetts Medical School, fen-biao.gao@umassmed.edu
"Novel insights from iPSCs and *Drosophila* models of *C9ORF72*-related frontotemporal dementia and ALS"

3:30 – 3:45 pm
Haining Zhu, University of Kentucky, haining@uky.edu
"RNA binding protein FUS and neurodegenerative disease Amyotrophic lateral Sclerosis"

3:45 – 4:00 pm
Yichang Jia, Tsinghua University, yichangjia@mail.tsinghua.edu.cn
"Disease mechanisms underlying neurodegeneration caused by RNA abnormalities"

Concurrent Session 19: Brain Development and Diseases (Crape Myrtle/紫薇厅)
Co-Chairs: **Wen-Cheng Xiong**, Augusta University, wxiong@gru.edu
Xiang Yu, Institute of Neuroscience, CAS, yuxiang@ion.ac.cn

2:00 – 2:20 pm
Zhiqi Xiong, Institute of Neuroscience CAS, xiongzhiqi@ion.ac.cn
"CDKL5 and synaptic plasticity"

2:20 – 2:40 pm
J. Julius Zhu, University of Virginia, jjzhu@virginia.edu
"Oncogenic Ras signaling at synapses"

2:40 – 3:00 pm
Zhihen Xu, Institute of Genetics and Developmental Biology, CAS, zhxu@genetics.ac.cn
"Crmp2 mutant mice display schizophrenia-like behaviors and hippocampal dysfunction"

3:00 – 3:20 pm
Jun Ding, Stanford University, dingjun@stanford.edu
"Curb your cravings-dopamine/GABA co-release in alcohol addiction"

3:20 – 3:40 pm
Riqiang Yan, Cleveland Clinic, yanr@ccf.org
"BACE in the control of neurogenesis and astrogenesis"

3:40 – 4:00 pm
Li Gan, University of California at San Francisco, lgu@gladstone.ucsf.edu
"Converging pathways in aging and neurodegeneration"

Concurrent Session 20: New Investigators Session I (Lily Hall B/百合 B 厅**)**

Co-Chairs: **Xiaoke Chen,** Stanford University, xkchen@stanford.edu
Zhongsheng You, Washington University at St. Louis, zyou@wustl.edu

2:00 – 2:15 pm
Zhongsheng You, Washington University at St. Louis, zyou@wustl.edu
"RNA decay in the DNA damage response"

2:15 – 2:30 pm
Dong Wang, Tsinghua University, dwang@biomed.tsinghua.edu.cn
"High Throughput Sequencing facilitated Drug Discovery"

2:30 – 2:45 pm
Shuli Xia, Johns Hopkins University, xia@kennedykrieger.org
"Methylated *cis*-regulatory elements mediate KLF4-denpendent gene transactivation and cell migration"

2:45 – 3:00 pm
Jian Xu, University of Southern California, xujian@usc.edu
"PRMT1-p53 pathway and epcardial differentiation"

3:00 – 3:15 pm
Qing Li, Peking University, China, li.qing@pky.edu.cn
"Depositing histones during DNA replication"

3:15 – 3:30 pm
Qing Zhang, University of North Carolina, qing_zhang@med.usc.edu
"Oxygen sensing pathways in cancer"

3:30 pm – 3:45 pm
Zilong Qiu, Shanghai Institute of Biological Sciences, zqiu@ion.ac.cn
"The neural mechanism and non-human primate model for autism"

3:45 – 4:00 pm
Xiaoke Chen, Stanford University, xkchen@stanford.edu
"Brain circuits mediating negative emotions"

Concurrent Session 21: Abstract Session II (Jasmine hall/茉莉厅)

Co-Chairs: **Xiao-Jing Wang**, University of Colorado, Denver, xj.wang@ucdenver.edu
Zhigao Wang, UT Southwestern Medical Center, zhigao.wang@utsouthwestern.edu

2:00 – 2:15 pm

Xiao-Jing Wang, University of Colorado, Denver, xj.wang@ucdenver.edu
Mechanisms of therapeutic effect of Smad7 on radiation-induced oral mucositis
2:15 – 2:30 pm
Jianqiu Wu, Ohio State University, wu.620@osu.edu
Plasma membrane deposition during cytokinesis

2:30 – 2:45 pm
Lei Xie, Hunter College, City University of New York, lxie@iscb.org
Toward precision medicines using structural systems pharmacology

2:45 – 3:00 pm
Chunbo Zhang, Nanchang University, cbzhang@ncu.edu.cn
The Histone Deacetylases Regulate the Non-viral Transgene Silencing *In Vivo*

3:30 – 3:45 pm
Bo Zhong, Wuhan University, zhongbo@whu.edu.cn
Regulation of innate antiviral immunity by deubiquitinating enzymes

3:45 – 4:00 pm
Winnie Shum, ShanghaiTech University, China, shumw@shanghaitech.edu.cn
Epithelial Cellular Communication for A Congenial Microenvironment

4:00 – 4:15 pm
Ping Hu, Shanghai Institutes for Biological Sciences, CAS, hup@sibcb.ac.cn
Pro-inflammatory cytokines promote muscle regeneration

4:15 – 4:30 pm
Zhigao Wang, UT Southwestern, zhigao.wang@utsouthwestern.edu
Ancient medicine, new cure? -Finding necroptosis inhibitors from Chinese traditional herbs

4:00-4:30pm ***Coffee Break*** *(Lily Hall Foyer/百合前厅)*

4:30 – 6:30 pm **Concurrent sessions**

Concurrent Session 22: Plant Biology (Lily Hall A/百合A厅)
Co-Chairs: **Yijun Qi**, Tsinghua University, qiyijun@mail.tsinghua.edu.cn
Jian-Min Zhou, Institute of Genetics and Developmental Biology, CAS, jmzhou@genetics.ac.cn

4:00 – 4:15 pm
Zhizhong Gong, China Agricultural University, gongzz@cau.edu.cn
"DNA replication- and repair-mediated transcription silencing"

4:15 – 4:30 pm
Xinjian He, National Institute of Biological Sciences, hexinjian@nibs.ac.cn
"RNA-directed DNA methylation and chromatin silencing"

4:30 – 4:45 pm
Jiawei Wang, Institute of Plant Physiology and Ecology, jwwang@sibs.ac.cn
"The clearance of repressive roadblocks drives shoot regeneration in plants"

4:45 – 5:00 pm
Yijun Qi, Tsinghua University, qiyijun@tsinghua.edu.cn
"Transcriptional regulation by Arabidopsis Argonautes"

5:00 – 5:15 pm
Yi Li, Peking University, liyi@pku.edu.cn
"MicroRNA-mediated antiviral defense in rice"

5:15 – 5:30 pm
Xueping Zhou, Zhejiang University, zzhou@zju.edu.cn
"Molecular arms race between viruses and plants"

5:30 – 5:45 pm
Yuanchao Wang, Nanjing Agricultural University, wangyc@njau.edu.cn
"The arms race in Phytopathora-plant interaction"

5:45 – 6:00 pm
Jian-Min Zhou, Institute of Genetics and Developmental Biology, CAS, jmzhou@genetics.ac.cn
"New strategy of bacterial phytopathogen virulence: strike better after the enemy has struck"

Concurrent Session 23: Stem Cell Biology (Peony Hall/牡丹厅)

Co-Chairs: **Shuo Lin,** University of California, Los Angeles, shuolin@ucla.edu
Chuxia Deng, University of Macau, cxdeng@umac.mo

4:30 – 4:48 pm
Shuo Lin, University of California, Los Angeles, shuolin@ucla.edu
"Production of endothelial cells from stem cells and somatic cells"

4:48 – 5:05 pm
Xiaohua Shen, Tsinghua University, xshen@tsinghua.edu.cn
"LncRNAs in stem cell function and transcription"

5:05 – 5:22 pm
Guokai Chen, University of Macau, guokaichen@umac.mo
"Improving cell culture consistency in human pluripotent stem cells"

5:22 – 5:39 pm
Renhe Xu, University of Macau, renhexu@umac.mo
"Marfan syndrome, mimicked and corrected in Petri dish"

5:39 – 5:56 pm
Xianming Mo, West China Hospital, Sichuan University, xmingmo@yahoo.com
"Capture and property of cancer stem cells from patients with gastric and rectal carcinoma"

5:56 – 6:13 pm
Chengjian Zhao, West China Hospital, Sichuan University, morning_zcj@163.com
"ETV2 Mediates the endo-transdifferentiation of glioblastoma stem-like cells"

6:13 – 6:30 pm
Chuxia Deng, University of Macau, cxdeng@umac.mo
"Cancer initiating cells in Brca1 associated cancer: drug resistance and metastasis"

Concurrent Session 24: Innovative Translational Medicine (Hibiscus Hall/芙蓉厅)

Co-Chairs: **Yi Sun**, Zhejiang University, yisun@zju.edu.cn
Hubing Shi, Sichuan University, shihubing77@sina.com

4:30 – 4:50 pm
Weiping Zou, University of Michigan, wzou@med.umich.edu

"Epigenetic control of T cell cancer trafficking and immunotherapy"

4:50 – 5:10 pm
Yuan Zhu, National Children's Medical Center, yzhu@childrennational.org
"Exploring therapeutic benefits of p53 activation in cancers that rarely have mutations in the p53 pathway"

5:10 – 5:30 pm
Hilda Ye, Albert Einstein College of Medicine, hilda.ye@ einstein.yu.edu
"STAT3 activation in ABC diffuse large B cell lymphoma: therapeutic consequences"

5:30 – 5:42 pm
Hubing Shi, Sichuan University, shihubing77@sina.com
"Tumor finds its way out: the lesson we learned from BRAF inhibition in melanoma"

5:42 – 5:54 pm
Bisen Ding, Sichuan University, dingbisen@scu.edu.cn
Hematopoietic and vascular niche regulates organ regeneration and fibrosis

5:54 – 6:06 pm
Lijun Jia, Fudan University, ljjia@fudan.edu.cn
"Targeting overactivated neddylation modification for anticancer therapy"

6:06 – 6:18 pm
Suling Liu, University of Science and Technology of China, suling@ustc.edu.cn
"The heterogeneity and regulation of breast cancer stem cells"

6:18 – 6:30 pm
Yi Sun, Zhejiang University, yisun@zju.edu.cn
"FBXW7 faciliates non-homologous end-joining repair via ubiquitylating XRCC4: therapeutic application."

Concurrent Session 25: Late-Breaking session II (Lily Hall B/百合 B 厅)
Co-Chairs: **Haifan Lin**, Yale University, haifan.lin@yale.edu
Ren Sun, University of California, Los Angeles, RSun@mednet.ucla.edu

4:30 – 4:50 pm
Hongtao Yu, UT Southwestern Medical Center & HHMI, hongtao.yu@utsouthwestern.edu
"Mitotic regulators in insulin signaling and metabolism"

4:50 – 5:10 pm
Haitao Li, Tsinghua University lht@tsinghua.edu.cn
"Sensing histone crotonylation for transcription control"

5:10 - 5:30 pm
Ren Sun, University of California, Los Angeles, RSun@mednet.ucla.edu
"Quantitative viral genetics at single nucleotide resolution"

5:30 - 5:50 pm
Ligan Wu, Shanghai Institute of Biochemistry and Cell Biology, CAS, lgwu@sibcb.ac.cn
"Sequencing Small RNAs in Mouse Oocytes and Early Embryos"

5:50 - 6:10 pm

Zhimin Lu, MD Anderson Cancer Center, zhiminlu@mdanderson.org
"Cancer metabolism and beyond"

6:10 - 6:30 pm
Hongyang Wang, National Center for Liver Cancer & Eastern Hepatobiliary Surgery Hospital/Institute, Shanghai, hywangk@vip.sina.com
"Tumour Heterogeneity and Precision Medicine"

Concurrent Session 26: New Investigators Session II (Crape Myrtle Hall/紫薇厅)
Co-Chairs: **Jun Lu**, Yale University, jun.lu@yale.edu
Yan Liu, Indiana University of School of Medicine, liu219@iu.edu

4:30 – 4:45 pm
Jian Yuan, Mayo Clinic, yuan.jian@mayo.edu
"Deubiquitination regulation in AKT pathway"

4:45 – 5:00 pm
Zhonghan Li, Sichuan University, Zhonghan.Li@outlook.com
"lncRNA-mediated regulation of innate immune response"

5:00 – 5:15 pm
Jia Chen, Shanghai Tech University, chenjia@shanghaitech.edu.cn
"APOBEC3s mediate mutagenesis in CRISPR/Cas9-induced genome editing"

5:15 – 5:30 pm
Yan Liu, Indiana University of School of Medicine, liu219@iu.edu
"Role of p53 in hematopoietic stem cells and leukemia"

5:30 – 5:45 pm
Jungseog Kang, New York University Shanghai, jungseog.kang@nyu.edu
"Phenotypic cancer drug screening by high-content single-cell analysis"

5:45 – 6:00 pm
Dongyi Xu, Peking University, xudongyi@pku.edu.cn
"A mitosis-specific MRN complex acts as a mitotic DNA damage checkpoint"

6:00 – 6:15 pm
Tieshan Tang, Institute of Zoology**,** CAS**,** tangtsh@ioz.ac.cn
"Calcium homeostatic maintenance in health and diseases"

6:15 – 6:30 pm
Jun Lu, Yale University, jun.lu@yale.edu
"The role of hematopoietic stem cell mutations on immune response"

Concurrent Session 27: Abstract Session III (Jasmine Hall/茉莉厅)
Co-Chairs: **Xiaobo Zhong**, University of Connecticut, xiaobo.zhong@uconn.edu
Xia Jin, Insititut Pasteur of Shanghai, xjin@ips.ac.cn

4:30 – 4:45 pm
Xia Jin, Insititut Pasteur of Shanghai, xjin@ips.ac.cn
"Subunit dengue vaccines"

4:45 – 5:00 pm

Yuanchao Xue, Institute of Biophysics, CAS, ycxue@ibp.ac.cn
"Sequential regulatory loops as key gatekeepers for neuronal reprogramming in human cells"

5:00 – 5:15 pm
Wenhui Li, National Institute of Biological Sciences, liwenhui@nibs.ac.cn
Hepatitis B virus and TLS polymerase

5:15 – 5:30 pm
Yi Shi, Institute of Microbiology, CAS, tianxn@biols.ac.cn
Ebola virus entry and fusion

5:30 – 5:45 pm
Xiangxi Wang, Institute of Biophysics, CAS, xiangxiwang@163.com
"Structure of Hepatitis A virus and Potent Neutralization of Hepatitis A virus"

5:45 – 6:00 pm
Xin-Hong Zhu, Southern Medical University, zhuxh@fimmu.com
"A perspective on astrocytes in major depressive disorder"

6:00 – 6:15 pm
Zhanxiang Zhou, University of North Carolina, Greensboro, z_zhou@uncg.edu
"Impact of adipose dysfunction on the pathogenesis of alcoholic liver disease"

6:15 – 6:30 pm
Xiaobo Zhong, University of Connecticut, xiaobo.zhong@uconn.edu
"A new concept in precision medicine: impact of human health by neonatal drug treatment"

Concurrent Session 28: Abstract session IV (Daffodil Hall/水仙厅)
Co-Chairs: **Junmin Peng**, St. Jude Children's Research Hospital, junmin.peng@stjude.org
Fei Li, New York University, fl43@nyu.edu

4:30 – 4:45 pm
Fei Li, New York University, fl43@nyu.edu
"Coordinated regulation of heterochromatin inheritance by DNA Polymerase epsilon complex"

4:45 – 5:00 pm
Yuying Liang, University of Minnesota, liangy@umn.edu
"Development of a novel viral vaccine vector based on a tri-segmented arenavirus"

5:00 – 5:15 pm
Qing Lu, Cincinnati Children's Hospital Medical Center, richard.lu@cchmc.org
"Targeting the Seeds of Brain Cancer"

5:15 – 5:30 pm
Zhong Wang, University of Michigan, zhongw@med.umich.edu
"Heart regeneration with engineered cell carrier-mediated cell transplantation and direct reprogramming"

5:30 – 5:45 pm
Chao Wan, The Chinese University of Hong Kong, cwan@cuhk.edu.hk
"The oxygen sensing pathway in bone regulates hematopoietic lineage differentiation"

5:45 – 6:00 pm
Meng Wang, Baylor College of Medicine, wmeng@bcm.edu

“Longevity as a Matter of Fat”

6:00 – 6:15 pm
Hinh Ly, University of Minnesota, hly@umn.edu
"Immune Evasive Mechanisms of Hemorrhagic Fever-causing Arenaviruses"

6:15 – 6:30 pm
Junmin Peng, St. Jude Children's Research Hospital, junmin.peng@stjude.org
“High Throughput Proteomics Approach to Understanding Common Human Diseases”

7:00 – 10:00PM *Closing Banquet*

- Announcing election results
- Speech by newly elected president
- Entertainment

Day 5, August 2, 2016, Tuesday ***Departure***

Bon Voyage!

Keynote and Plenary Session Speakers

Jack W. Szostak, PhD

Investigator, Howard Hughes Medical Institute

Professor, Harvard Medical School

The Alex Rich Distinguished Investigator, Massachusetts General Hospital

Boston, Massachusetts, USA

E-mail: szostak@molbio.mgh.harvard.edu

Education:
- BS (1972), McGill University, Montreal, Canada
- PhD (1977), with Prof. Ray Wu at Cornell University, Ithaca, NY

•Research Interests:
•synthesis of self-replicating systems and the origin of life
•Honors and Awards:
•2006 Albert Lasker Basic Medical Research Award
•2009 Nobel Prize in Physiology or Medicine
•Member, National Academy of Sciences, USA
•Member, American Academy of Arts and Sciences
•Member, the American Philosophical Society
•Fellow of the New York Academy of Sciences

Representative Publications (no more than 5):
- Prywes N, Blain JC, Del Frate F, Szostak JW. Nonenzymatic copying of RNA templates containing all four letters is catalyzed by activated oligonucleotides. eLife, 2016 Jun 28;5. pii: e17756. doi: 10.7554/eLife.17756.
- Engelhart AE, Adamala KP, Szostak JW. A simple physical mechanism enables homeostasis in primitive cells. Nat Chem 2016 May;8(5):448-53.
- Sheng J, Li L, Engelhart AE, Gan J, Wang J, Szostak JW. Structural insights into the effects of 2'-5' linkages on the RNA duplex. Proc Natl Acad Sci USA 2014 Feb 25;111(8):3050-5.
- Adamala K, Szostak JW. Nonenzymatic template-directed RNA synthesis inside model protocells. Science 2013 Nov 29;342(6162):1098-100.
- Engelhart AE, Powner MW, Szostak JW. Functional RNAs exhibit tolerance for non-heritable 2'-5' versus 3'-5' backbone heterogeneity. Nat Chem 2013 May;5(5):390-4.

Jon Clardy, PhD

Hsien Wu & Daisy Yen Wu Professor

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Harvard Medical School & Harvard University

Boston, Massachusetts, USA

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Education:
- BS (1964) Chemistry, Yale University
- PhD (1969), Chemistry, Harvard University

Other Positions:
• Co-Director, Infectious Disease Program, Broad Institute of Harvard and MIT
•Research Interests:
•Biologically active small molecules – discovery and mechanism of action
Honors and Awards:
•Clark Distinguished Teaching Award, Cornell University
•Fellow, American Association for the Advancement of Science
•Günther Award, American Chemical Society
•Member, American Academy of Arts and Sciences
•Fellow, American Academy of Microbiology, USA
•A. I. Scott Medal, American Chemical Society & Texas A&M University

Representative Publications (no more than 5):
•Isolation and Synthesis of a Bacterially-Produced Inhibitor of Rosette Development in Choanoflagellates, (2016) Cantley AM, Woznica A, Beemelmanns C, King N, Clardy J, J Am Chem Soc 138: 4326-9.
•Bacterial lipids activate, synergize, and inhibit a developmental switch in choanoflagellates (2016) Woznica A, Cantley AM, Beemelmanns C, Freinkman E, King N, Clardy J. Proc Natl Acad Sci USA 113: 7894-9
•A Rebeccamycin Analog Provides Plasmid-Encoded Niche Defense, (2015) Van Arnam EB, Ruzzini AC, Sit CS, Currie CR, Clardy J, J Am Chem Soc 137: 14272-4.
•Variable genetic architectures produce virtually identical molecules in bacterial symbionts of fungus-growing ants (2015) Sit CS, Ruzzini AC, Van Arnam EB, Ramadhar TR, Currie CR, Clardy J. Proc Natl Acad Sci USA 112: 13150-4

Chenjian Li, Ph.D.
Vice Provost, Peking University
Professor and Associate Dean,
School of Life Sciences, Peking University
li_chenjian@PKU.edu.cn

Dr. Chenjian Li is currently the Vice Provost of Peking University, and Professor and Associate Dean of School of Life Science, Peking University. Prior to his return to China, he was an assistant professor and associate professor at Weill Medical College of Cornell University (2003—2009), and then Aidekman Endowed Chair of Neurology at Mount Sinai School of Medicine (2010—2013). Dr. Li's scientific research focuses on exploring the molecular and cellular mechanisms of neurological diseases.

Parallel to research, Dr. Li is extremely devoted to education development and reforms, ranging from high school, undergraduate, graduate and medical student education. He was one of the organizers of the influential "*Science Outreach Program*" in USA. Since his return to China, he has played a leading role in important initiatives such as the establishment of *Cambridge-PKU Center for China Study*, inauguration of *Rhodes Scholar program in China*, reform of college admission by holistic evaluation, design and implementation of liberal education curriculum at PKU, etc. He received many awards for excellence in teaching, including a student-voted “Pied Piper Mater” at Weill Cornell Medical College in 2006, and a student-voted “Best Teacher” at PKU in 2015.

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研究馆员（教授）
作家

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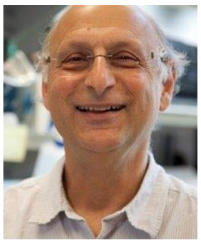

Richard D. Kolodner, PhD
Director and Distinguished Professor
Ludwig Institute for Cancer Research, San Diego Branch
University of California San Diego School of Medicine
San Diego, California, USA
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Education:
- BS (1971), Biological Sciences, University of California, Irvine
- PhD (1975), , Biological Sciences, University of California, Irvine
- Postdoctoral training (1975-1978), Harvard Medical School

Other Positions:
•Head, Scientific Review Council, Cancer Prevention and Research Institute of Texas (CPRIT)
•Research Interests:
•DNA mismatch repair, genome instability and cancer genetics
Honors and Awards:
•Charles S. Mott Prize, General Motors Cancer Research Foundation
•Member, National Academy of Sciences, USA
•Kirk A. Landon-AACR Award for Basic Cancer Research
•Member, American Academy of Arts and Sciences
•Member, National Academy of Medicine, USA
Representative Publications (no more than 5):
•Putnam, CD, Srivatsan, A, Nene, RV, Martinez, SL, Clotfelter, SP, Bell, SN, Somach, S, de Souza, JES, Fonseca, AF, de Souza, SJ, and Kolodner, RD. A genetic network that suppresses genome rearrangements in *Saccharomyces cerevisiae* and contains defects in human cancers. Nat. Communications. 2016;7:11256 doi/ncomms11256.
•Goellner, EM, Smith, CE, Campbell, CS, Hombauer, H, Desai, A, Putnam, CD, and Kolodner, RD. PCNA and Msh2-Msh6 activate an Mlh1-Pms1 endonuclease pathway required for Exo1-independent mismatch repair. Mol. Cell. 2014; 55:291-304.
•Hombauer, H, Srivatsan, A, Putnam, CD, and Kolodner, RD. Mismatch repair, but not heteroduplex rejection, is temporally coupled to DNA replication. Science. 2011;334:1713-1716.
•Hombauer, H, Campbell, CS, Smith, CE, Desai, A, and Kolodner, RD. Visualization of eukaryotic DNA mismatch repair reveals distinct recognition and repair intermediates. Cell. 2011;147:1040-1053.
•Putnam, CD, Hayes, TK, and Kolodner, RD. Specific pathways prevent duplication-mediated genome rearrangements. Nature. 2009;460:984-989.

Wei Yang, PhD
Distinguished Investigator
Laboratory of Molecular Biology, NIDDK
National Institutes of Health
Bethesda, MD, USA
E-mail: Wei.Yang@nih.gov

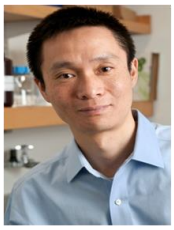
Education:
- BA (1985), Biochemistry, SUNY at Stony Brook, NY
- PhD (1991), Biochemistry and Molecular Biophysics, Columbia University, NY
- Postdoc (1991-2) Professor Wayne Hendrickson, Columbia University, NY
- Postdoc (1992-5) Professsor Tom Steitz, YaleUniveristy, CT

Other Positions:
- Arnold Professor in Molecular BioSciences, University of Texas at Austin, TX

Research Interests:
- Structural Biology and mechanism of DNA repair, replication and recombination

Honor and Awards:
- The 2002 Bea Singer Young Investigator Award (sponsored by the GRC)
- The 2011 Dorothy Crowfoot Hodgkin award (sponsored by the Protein Society)
- Member of the US National Academy of Sciences since 2013
- Member of the Americal Academy of Arts and Sciences since 2015

Representative Publications (no more than 5):
Gao, Y. & Yang, W. (2016) Capture of a third Mg2+ is essential for catalyzing DNA synthesis, **Science**, 352, 1334-1337.
Kim, M.S., Lapkouski, M., Yang, W. & Gellert M. (2015) Crystal structure of the V(D)J recombinase RAG1-RAG2, ***Nature***, 518, 507-511.
Li, C.-L., Golebiowski, F., Onishi, Y., Samara, N., Sugasawa, K. & Yang, W. (2015) Tripartite DNA lesion recognition and verification by XPC, TFIIH and XPA in nucleotide excision repair, ***Mol Cell***, 59, 1025-34.
Nakamura, T., Zhao, Y., Yamagata, Y., Hua, Y.J. & Yang, W. (2012) "Watching DNA polymerase h make a phosphodiester bond". **Nature**, *487*, 196-201.
Gupta, S., Gellert, M. & Yang , W. (2011) Mechanism of mismatch recognition revealed by human MutSb bound to unpaired DNA loops. ***NSMB.*** 19, 72-78.



Chuan He, PhD

Professor

Department of Chemistry, Department of Biochemistry and Molecular Biology, Howard Hughes Medical Institute, The University of Chicago

Chicago, IL, USA

E-mail: chuanhe@uchicago.edu

Education:
- BS (1994), Chemistry, University of Science and Technology of China
- PhD (2000), Chemistry, MIT
- Postdoctoral training (2000-2002), Harvard University

Other Positions:
•Director, Institute for Biophysical Dynamics, The University of Chicago
•Director, Synthetic and Functional Biomolecules Center (SFBC), Peking University

Research Interests:
•Epigenetics, RNA methylation, chemical biology

Honors and Awards:
•John T. Wilson Distinguished Service Professor

Representative Publications (no more than 5):

•Dominissini, D.; Nachtergaele, S.; Moshitch-Moshkovitz, S.; Peer, E.; Kol, N.; Ben-Haim, M. S.; Dai, Q.; Di Segni, A.; Salmon-Divon, M.; Clark, W. C.; Zheng, G.; Pan, T.; Solomon, O.; Eyal, E.; Hershkovitz, V.; Han, D.; Doré, L. C.; Amariglio, N.; Rechavi, G.; He, C. The dynamic N1-methyladenosine methylome in eukaryotic messenger RNA. ***Nature*** 2016, *530*, 441-446.

•Wang, X.; Zhao, B. S.; Roundtree, I. A.; Lu, Z.; Han, D.; Ma, H.; Weng, X.; Chen, K.; Shi, H.; He, C. N6-methyladenosine Modulates Messenger RNA Translation Efficiency. ***Cell*** 2015, 161, 1388-1399.

•Fu, Y.; Luo, G. Z.; Chen, K.; Deng, X.; Yu, M.; Han, D.; Hao, Z.; Liu, J.; Lu, X.; Doré, L. C.; Weng, X.; Ji, Q.; Mets, L.; He, C. N6-methyldeoxyadenosine marks active transcription start sites in chlamydomonas. ***Cell*** 2015, 161, 879-892.

•Wang, X.; Lu, Z.; Gomez, A.; Hon, G. C.; Yue, Y.; Han, D.; Fu, Y.; Parisien, M.; Dai, Q.; Jia, G.; Ren, B.; Pan, T.; He, C.* N6-methyladenosine-dependent regulation of messenger RNA stability. ***Nature*** 2014, 505, 117-120.

•Yu, M.; Hon, G. C.; Szulwach, K. E.; Song, C.-X.; Zhang, L.; Kim, A.; Li, X. K.; Dai, Q.; Shen, Y.; Park, B.; Min, J. H.; Jin, P.*; Ren, B.*; He, C.* Base-resolution analysis of 5-hydroxymethylcytosine in the mammalian genome. ***Cell*** 2012, 149, 1368-1380.

Bing Ren, PhD

Ludwig Institute for Cancer Research

Department of Cellular and Molecular Medicine

University of California, San Diego

La Jolla, California, USA

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Education:
- BS (1991), Biology, University of Science and Technology of China
- MS (1998), Computer Sciences, Harvard University
- PhD (1998), Biochemistry, Harvard University
- Postdoctoral training (1998-2001), Whitehead Institute for Biomedical Research

Other Positions:
•Reviewing Editor: eLife

Research Interests:
•Gene Regulation; Genomics; Epigenomics; Cancer Research; Stem Cell Research.

Honors and Awards:
•Distinguished Young Investigator Award, Chinese Biological Investigators Society (2007)
•Elected as Fellow of the American Association for the Advancement of Science (2013)
•Chen Award for Distinguished Academic Achievement in Human Genetic and Genomic Research (2016)

Representative Publications (no more than 5):

•J. R. Dixon, S. Selvaraj, F. Yue, A. Kim, Y. Li, Y. Shen, M. Hu, J. S. Liu and B. Ren. Topological domains in mammalian genomes identified by analysis of chromatin interactions. Nature. 2012. 485:376-80

•Y. Shen, F. Yue, D. F. McCleary, Z. Ye, L. E. Edsall, S. Kuan, U. Wagner, J. R. Dixon, L. Lee, V. V. Lobanenkov, and B. Ren. A map of the cis-regulatory sequences in the mouse genome. Nature, 2012. 488:116-20.

•F. Jin, Y. Li, J. R. Dixon, S. Selvaraj, Z. Ye, A. Y. Lee, A. C. Yen, A. C. Schmitt, C. Espinoza, and B. Ren. A High-resolution Map of Signaling-dependent, Dynamic Chromatin Interactome in Human Cells. Nature, 2013. 503: 290-4

•D. Leung, I. Jung, N. Rajagopal, A. Schmitt, S. Selvaraj, A. Y. Lee, C. A. Yen, S. Lin, Y. Lin, Y. Qiu, W. Xie, F. Yue, M. Hariharan, P. Ray, S. Kuan, L. Edsall, H. Yang, N. C. Chi, M. Q. Zhang, J. R. Ecker and B. Ren. Integrative analysis of haplotype-resolved epigenomes across human tissues. Nature, 2015. 518:350-4.

•J.R. Dixon, I. Jung, S. Selvaraj, Y. Shen, J. E. Antosiewicz-Bourget, A. Y. Lee, Z. Ye, A. Kim, N. Rajagopal, W. Xie, Y. Diao, J. Liang, H. Zhao, V. V. Lobanenkov, J. E. Ecker, J. A. Thomson, and B. Ren. Chromatin Architecture Reorganization during Stem Cell Differentiation. Nature, 2015. 518: 331-6.

Junying Yuan, PhD

Elizabeth D. Hay Professor of Cell Biology

Department of Cell Biology

Harvard Medical School

Boston, MA, USA

E-mail: jyuan@hms.harvard.edu

Education:
- BS (1982), Biochemistry, Fudan University
- PhD (1989), Neuroscience, Harvard University

Other Positions:
•Professor and Director: Interdisciplinary Research Center on Biology and Chemistry, Shanghai Institute of Organic Chemistry.
Research Interests:
•Mechanisms of cell death.
Honors and Awards:
•Fellow of the American Academy of Arts and Sciences and fellow of the American Association for the Advancement of Sciences.
Representative Publications (no more than 5):

a. Degterev A, Huang Z, Boyce M, Li Y, Jagtap P, Mizushima N, Cuny GD, Mitchison TJ, Moskowitz MA, & **Yuan J** (2005) Chemical inhibitor of nonapoptotic cell death with therapeutic potential for ischemic brain injury. *Nat Chem Biol* 1(2):112-119.
b. Degterev A, Hitomi J, Germscheid M, Ch'en IL, Korkina O, Teng X, Abbott D, Cuny GD, Yuan C, Wagner G, Hedrick SM, Gerber SA, Lugovskoy A, & **Yuan J** (2008) Identification of RIP1 kinase as a specific cellular target of necrostatins. *Nat Chem Biol* 4(5):313-321.
c. Hitomi J, Christofferson DE, Ng A, Yao J, Degterev A, Xavier RJ, & **Yuan J** (2008) Identification of a molecular signaling network that regulates a cellular necrotic cell death pathway. *Cell* 135(7):1311-1323.
d. Ofengeim D, Ito Y, Najafov A, Zhang Y, Shan B, DeWitt JP, Ye J, Zhang X, Chang A, Vakifahmetoglu-Norberg H, Geng J, Py B, Zhou W, Amin P, Lima JB, Qi C, Yu Q, Trapp B & **Yuan J** (2015) Activation of necroptosis in multiple sclerosis. *Cell Reports*. 10(11):1836-49.
e. Ito et al. (2016). RIPK1 Mediates Axonal Degeneration By Promoting Inflammation and Necroptosis in ALS. *Science. In press.*

Carlo M. Croce, M.D.

Professor/Director/Chair

Department of Cancer Biology and Genetics

The Ohio State University Wexner Medical Center

Columbus, Ohio, USA

E-mail: carlo.croce@osumc.edu

Education:
- BS (1967), Medicine, School of Medicine, University of Rome, Rome, Italy (Intern in Biochemistry)
- MD (1969), Medicine, Summa Cum Laude, University of Rome

Other Positions:
•Editorial Board: Signal Transduction and Targeted Therapy
Research Interests:
•Cancer Genetics
Honors and Awards:
•Distinguished University Professor
•John W. Wolfe Chair in Human Cancer Genetics
•Member, National Academy of Sciences and National Academy of Medicine
•Member, American Academy of Arts and Sciences
Representative Publications (no more than 5):

•Calin GA, Dimitru CD, Shimizu M, Bichi R, Zupo S, Noch E, Alder H, Rattan S, Keating M, Rai K, Rassenti L, Kipps T, Negrini M, Bullrich F, and **Croce CM**: Frequent deletions and down regulation of micro RNA genes miR15 and miR16 at 13q14 in chronic lymphocytic leukemia. ***Proc. Natl. Acad. Sci., USA***, 99: 15524-15529, 2002.

•Calin GA, Liu C-G, Sevignani C, Ferracin M, Felli N, Dumitru CD, Shimizu M, Cimmino A, Zupo S, Dono M, Del'Aquila ML, Rassenti L, Kipps TJ, Bullrich F, Negrini M, and **Croce CM**: MicroRNA profiling reveals distinct signatures in B-cell chronic lymphocytic leukemia. ***Proc. Natl. Acad. Sci., USA***, 101: 11755-11760, 2004.

•Calin GA, Ferracin M, Cimmino A, Di Leva G, Shimizu M, Wojcik S, Iorio M, Visone R, Sever I, Fabbri M, Iuliano R, Palumbo T, Pichiorri F, Roldo C, Garzon R, Sevignani C, Rassenti L, Wise T, Alder H, Volinia S, Liu C, Kipps T, Negrini M, and **Croce CM**. A Unique MicroRNA Signature Associated with Prognostic Factors and Disease Progression in B cell Chronic Lymphocytic Leukemia. ***N. Eng. J. Med***., 353:1793-1801, 2005.

•Calin GA and **Croce CM**. MicroRNA signatures in human cancers. ***Nature Reviews Cancer***. 6:857-866, 2006.

•Costinean S, Zanesi N, Pekarsky Y, Esmerina T, Volinia S, Heerema N and **Croce CM**. Pre B cell proliferation and lymphoblastic leukemia/high grade lymphoma in Eμ miR155 transgenic mice. ***Proc. Natl. Acad. Sci., USA***, 103:7024-7029, 2006.

Yuquan Wei, Ph.D.

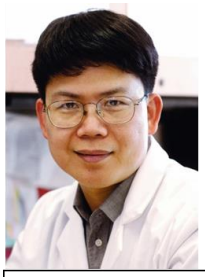
Academician of the Chinese Academy of Sciences,

Vice President of Sichuan University,

Director of State/National Key Laboratory of Biotherapy,

Director of Oncology Center in West China Hospital

Professor Wei is currently Academician of the Chinese Academy of Sciences, Vice President of Sichuan University, Director of State/National Key Laboratory of Biotherapy and Director of Oncology Center in West China Hospital. He is also deputy director of Human Gene Therapy. He focuses on research and development of biological medicine and small molecular targeted medicine. He was the distinguished professor of Chang Jiang Scholars System from 1999-2004. Prior to that, he was awarded by The National Science Fund for Distinguished Young Scholars in 1997. He was also poisoned as group leader of bio-engineering technology in the field of fifteen "863" biological and agricultural technology. He received his master degree in West China University of Medical Sciences in 1986, and received his Ph.D. in Medical School of Kyoto University in 1996, and came back to China in 1996 after 5-year study in Japan. He worked on basic researches of biological treatment of tumor and Application development and clinical practice, and the research results were published on Nature Medicine, PNAS, Blood, CancerRes., J.Immunol., J.Biol.Chem and other international journals.



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Education:

- BS (1985), Biology, Fujian Normal University
- PhD (1991), Biochemistry, SUNY Buffalo
- Postdoctoral training (1991-1992), Salk Institute

Other Positions:

•Senior Scientist, ProScript Inc, Cambridge, MA

Research Interests:

•Innate Immunity and Cell Signaling

Honors and Awards:

2012 National Academy of Sciences Award in Molecular Biology
2013 Fellow, American Association for the Advancement of Science
2014 Member, National Academy of Sciences, USA
2015 Merck Award, American Society of Biochemistry and Molecular Biology (ASBMB)

Representative Publications (no more than 5):

1.Liu, S., Cai, X., Wu, J., Cong, Q., Chen, X., Li, T., Du, F., Ren, J., Wu, Y., Grishin, N., and Chen, Z.J. (2015) Phosphorylation of innate immune adaptor proteins MAVS, STING and TRIF induces IRF3 activation. ***Science*** *347*, aaa2630.
2.Li, X., Wu, J., Gao, D., Wang, H., Sun, L., and Chen, Z.J. (2013). Pivotal roles of cGAS-cGAMP signaling in antiviral defense and immune adjuvant effects. ***Science*** *341*, 1390-1394
3.Gao, D., Wu, J., Wu, Y.T., Du, F., Aroh, C., Yan, N., Sun, L., and Chen, Z.J. (2013). Cyclic GMP-AMP Synthase Is an Innate Immune Sensor of HIV and Other Retroviruses. ***Science*** *341*, 903-906
4.Sun, L., Wu, J., Du, F., Chen, X., and Chen, Z.J. (2013). Cyclic GMP-AMP synthase is a cytosolic DNA sensor that activates the type I interferon pathway. ***Science*** *339*, 786-791.
5.Wu, J., Sun, L., Chen, X., Du, F., Shi, H., Chen, C., and Chen, Z.J. (2013). Cyclic GMP-AMP is an endogenous second messenger in innate immune signaling by cytosolic DNA. ***Science*** *339*, 826-830.

Don W. Cleveland, PhD

Distinguished Professor and Chair

Department of Cellular and Molecular Medicine

University of California, San Diego (Ludwig Cancer Research)

La Jolla, CA USA

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Education:

- BS (1972), Physics, New Mexico State University, Las Cruces, New Mexico
- PhD (1977), Biomedical Sciences, Princeton University, Princeton, New Jersey
- Postdoctoral training (1978-1981), University of California, San Francisco

Other Positions:

•Professor: Department of Neuroscience, Johns Hopkins University School of Medicine, Baltimore, Maryland

Research Interests:

•Mechanism and therapy for human neurodegenerative disease

Honors and Awards:

•Elected Member, National Academy of Sciences, 2006

•Elected Fellow, American Academy of Arts and Sciences, 2006

•Elected Fellow, American Association for the Advancement of Science (AAAS), 2009

•Elected Member, The National Academies Institute of Medicine, 2012

Representative Publications (no more than 5):

•Smith, R.A., Miller, T.M., Yamanaka, K., Monia, B.P., Condon, T.P., Hung, G., Lobsiger, C.S., Ward, C.W., McAlonis-Downes, M., Wei, H., Wancewicz, E.V., Bennett, C.F., and **Cleveland, D.W.** (2006). Antisense oligonucleotide therapy for neurodegenerative disease. J. Clin. Invest 116, 2290-2296.

•Polymenidou, M., Lagier-Tourenne, C., Hutt, K.R., Huelga, S.C., Moran, J., Liang, T.Y., Ling, S.C., Sun, E., Wancewicz, E., Mazur, C., Kordasiewicz, H., Sedaghat, Y., Donohue, J.P., Shiue, L., Bennett, C.F., Yeo, G.W., and **Cleveland, D.W.** (2011). Long pre-mRNA depletion and RNA missplicing contribute to neuronal vulnerability from loss of TDP-43. Nat. Neurosci. 14, 459-468.

•Kordasiewicz, H.B., Stanek, L.M., Wancewicz, E.V., Mazur, C., McAlonis, M.M., Pytel, K.A., Artates, J.W., Weiss, A., Cheng, S.H., Shihabuddin, L.S., Hung, G., Bennett, C.F., and **Cleveland, D.W.** (2012). Sustained therapeutic reversal of Huntington's disease by transient repression of huntingtin synthesis. Neuron 74, 1031-1044.

•Jiang, J., et al (2016). Gain of toxicity from ALS/FTD-linked repeat expansions in C9orf72 is alleviated by antisense oligonucleotides targeting GGGGCC-containing RNAs. Neuron 90:535-550.

Linheng Li, PhD

Investigator/Professor

Stowers Institute For Medical Research

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Education:

- BS (1985), Genetics, Fudan University
- MS (1993), New York University Medical Center
- PhD (1995), New York University Medical Center
- Postdoctoral training (1995-2000), University of Washington

Other Positions:

•Editorial Board Members: Cell Stem Cell, Stem Cell, Cancer Research, Cell Research

Research Interests:

•Stem cells and their associated niches in homeostasis and cancers

Honors and Awards:

•Fellow of the American Gastroenterological Association (AGA) 2013

•Fellow of the American Association for the Advancement of Science (AAAS) 2011

•Missouri Biotechnology Association Excellence in Life Sciences Award in Basic Research 2003

•Hudson Prize for excellence in basic biomedical research, presented by the M.R. and Evelyn Hudson Foundation 2004

Representative Publications (no more than 5):

•Qian P., He X., Paulson A., Li Z., Tao F., Perry JM, Guo F., Zhao M., Zhi L., Venkatraman A., Haug JS, Parmely T., Li H., Dobrowsky RT, Ding W-X, Kono T., Ferguson-Smith AC., **Li L**. The *Dlk1-Gtl2* Locus Preserves LT-HSC Function by Inhibiting the P13K-mTOR Pathway to Restrict Mitochondrial Metabolism. **Cell Stem Cell** 2016 Feb 4; (18):214-228

•Sugimura, R., X. C. He, A. Venkatraman, F. Arai, A. Box, C. Semerad, J. S. Haug, L. Peng, X. B. Zhong, T. Suda, and **L. Li**. Noncanonical Wnt signaling maintains hematopoietic stem cells in the niche. **Cell** 2012 Jul 20; 150:351-365. .

•**Li, L.,** and H. Clevers. 2010. Coexistence of quiescent and active adult stem cells in mammals. **Science** 2010 Jan 29; 327:542-545.

•Xie, Y., T. Yin, W. Wiegraebe, X. C. He, D. Miller, D. Stark, K. Perko, R. Alexander, J. Schwartz, J. C. Grindley, J. Park, J. S. Haug, J. P. Wunderlich, H. Li, S. Zhang, T. Johnson, R. A. Feldman, and **L. Li**. Detection of functional haematopoietic stem cell niche using real-time imaging. **Nature** 2009 Jan 1; 457:97-101 .

•Zhang, J., C. Niu, L. Ye, H. Huang, X. He, W. G. Tong, J. Ross, J. Haug, T. Johnson, J. Q. Feng, S. Harris, L. M. Wiedemann, Y. Mishina, and **L. Li**. Identification of the haematopoietic stem cell niche and control of the niche size. **Nature** 2003 Oct 23; 425:836-841.

Qi Zhou, PhD

Professor

State Key Laboratory of Stem Cell and Reproductive Biology

Institute of Zoology, Chinese Academy of Sciences (CAS)

Beijing, China

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Education:

- BS (1991), Developmental Biology of Embryo, Northeast Agricultural University, China
- PhD (1996), Histology and Embryology, Northeast Agricultural University, China
- Postdoctoral Fellow, then Associate Professor (1997-1999), Institute of Developmental Biology, CAS
- Postdoctoral Fellow and Project Leader (1999-2002), French National Institute for Agricultural Research (INRA)

Other Positions:

•Chinese delegate to and President of the International Stem Cell Forum (ISCF)

•President of the Chinese Society for Stem Cell Research, CSCB

Research Interests:

•Stem Cells and Regenerative Medicine

Honors and Awards:

•CAS Member

•Second Class Award of State Natural Science Award of China

Representative Publications (no more than 5):

•Li X, Cui XL, Wang JQ, Wang YK, Li YF, Wang LY, Wan HF, Li TD, Feng GH, Shuai L, Li ZK, Gu Q, Hao J, Wang L, Zhao XY, Liu ZH, Wang XJ, Li W, **Zhou Q**. Generation and application of mouse-rat allodiploid embryonic stem cells. ***Cell*** 164(1-2):279–292, 2016.

•Zhou Q, Wang M, Yuan Y, Wang X, Fu R, Wan H, Xie M, Liu M, Guo X, Zheng Y, Feng G, Shi Q, Zhao XY, Sha J, **Zhou Q**. Complete Meiosis from Embryonic Stem Cell-Derived Germ Cells In Vitro. ***Cell Stem Cell*** 18(3):330-340, 2016.

•Li W, Li X, Li T, Jiang MG, Wan H, Luo GZ, Feng C, Cui X, Teng F, Yuan Y, Zhou Q, Gu Q, Shuai L, Sha J, Xiao Y, Wang L, Liu Z, Wang XJ, Zhao XY, **Zhou Q**. Genetic modification and screening in rat using haploid embryonic stem cells. ***Cell Stem Cell*** 14(3):404-414, 2014.

•Li W, Teng F, Li T, **Zhou Q**. Simultaneous generation and germline transmission of multiple gene mutations in rat using CRISPR-Cas systems. ***Nature Biotechnology*** 31(8):684–686, 2013.

•Li W, Shuai L, Wan H, Dong M, Wang M, Sang L, Feng C, Luo GZ, Li T, Li X, Wang L, Zheng QY, Sheng C, Wu HJ, Liu Z, Liu L, Wang L, Wang XJ, Zhao XY, **Zhou Q**. Androgenetic haploid embryonic stem cells produce live transgenic mice. ***Nature*** 490(7420):407-411, 2012 .

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Huamian Biotechnology

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E-mail: changyu_7963@yahoo.com

Education:

- BS (1983), Biology, Wuhan University
- PhD (1994), Microbiology & Immunology, University of Colorado Medical Center
- Postdoctoral training (1994-1996), Harvard Medical School
- Postdoctoral training (1996-1998), MIT

Other Positions:

•Co-Founder, Immutics, USA

•Director, Cancer Immunology, Pfizer (2014-2016)

Research Interests:

•Cancer Immunotherapy

Representative Publications (no more than 5):

•**Wang C**., K. Thudium, M.. Han, X-T. Wang, H. Huang, D. Feingersh, C. Garcia, Y. Wu, M. Kuhne, M. Srinivasan, S. Singh, S. Wong, N. Garner, H. Leblanc, T. Bunch, D. Blanset, M. Selby, and A. Korman. 2014. In vitro characterization of the anti-PD-1 antibody nivolumab, BMS-936558, and in vivo toxicology in non-human primates. *Cancer Immunol Res.* 2:846-856.

•Brahmer J. R., C. G. Drake, I. Wollner, J. D. Powderly, J. Picus, W. H. Sharfman, E. Stankevich, A. Pons, T. M. Salay, T. L. McMiller, M. M. Gilson, **C. Wang**, M. Selby, J. M. Taube, R. Anders, L. Chen, A. J. Korman, D. M. Pardoll, I. Lowy, and S. L. Topalian. 2010. Phase I study of single-agent anti-programmed death-1 (MDX-1106) in refractory solid tumors: safety, clinical activity, pharmacodynamics, and immunologic correlates. *J Clin Oncol*. 28:3167-3175.

•Li B., M. Vanroey, **C. Wang**, T. H. Chen, A. Korman, and K. Jooss. 2009. Anti-programmed death-1 synergizes with granulocyte macrophage colony-stimulating factor-secreting tumor cell immunotherapy providing therapeutic benefit to mice with established tumors. *Clin. Cancer Res.* 15:1623-1634.

•Rutebemberwa A., S. C. Ray, J. Astemborski, J. Levine, L. Liu, K.A. Dowd, S. Clute, **C. Wang**, A. Korman, A. Sette, J. Sidney, D. M. Pardoll, and A. L. Cox. 2008. High-programmed death-1 levels on hepatitis C virus-specific T cells during acute infection are associated with viral persistence and require preservation of cognate antigen during chronic infection. *J. Immunol*. 181:8215-8225.

•Wong R. M., R. R. Scotland, R. L. Lau, **C. Wang**, A. Korman, W. M. Kast, and J. S. Weber. 2007. Programmed death-1 blockade enhances expansion and functional capacity of human melanoma antigen-specific CTLs. *Int. Immunol.* 19:1223-1234.

YI RAO, PhD

Chair Professor

Peking University

Beijing, China

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Education:

- BS(1983), Jiangxi Medical College
- MS(1985),Shanghai Medical University,
- PhD (1991), Neuroscience, University of California
- Postdoctoral Training (1991-1994), Harvard University

Other Positions:

• Editorial Boards: The Journal of Neuroscience, Developmental Biology, Neuroscience Research, Developmental Brain Research, NeuroSignals, Faculty of 1000 and Neuroscience Bulletin

Research Interests:

Neurogenesis (from 1985 to 1996), Axon Guidance (1997 to 2008), and Molecular Biology of Behaviors (2005 to present).

Representative Publications (no more than 5):

•Liu WW, Liang XH, Li YN, Gong JX, Yang Z, Zhang YH, Zhang JX and Rao Y .*(2011) Social regulation of aggression mediated by pheromonal activation of Or65a olfactory receptor neurons in Drosophila , Nature Neurosci , 7:896-902 .

•Liu Y, Jiang Y, Si Y, Kim J-Y, Chen Z-F, and Rao Y. * (2011) Molecular regulation of sexual preference revealed by genetic studies of 5-HT in the brain of male mice , Nature , 472:95-99 .

•Zhou C, Rao Y, and Rao Y .* (2008) A subset of octopaminergic neurons are important for Drosophila aggression , Nature Neurosci , 11:1059-1061.

•Li X, Gao X, Liu G, Xiong W, Wu J, Rao Y .* (2008) Netrin signal transduction and the guanine nucleotide exchange factor DOCK180 in attractive signaling , Nature Neurosci , 11:28-35.

•Jiang H, Guo W, Liang XH, and Rao Y .* (2005) Both the establishment and the maintenance of neuronal polarity require active mechanisms: critical roles of GSK-3b and its upstream regulators , Cell , 120: 123-135.

Zhigang He, PhD

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F.M. Kirby Program in Neuroscience

Boston Children's Hospital

Harvard Medical School

E-mail: zhigang.he@childrens.harvard.edu

Education:

- BS (1984), Nanjing Medical College
- PhD (1996), University of Toronto
- Postdoctoral training (1996-1999), University of California, San Francisco

Research Interests:

•Axon regeneration and neural repair

Representative Publications (no more than 5):

•Park K, Liu K, Hu Y, Smith P, Chen W, Cai B, Xu B, Connolly L, Kramvis I, Sahin M, He Z. Promoting axon regeneration in the adult CNS by modulation of the PTEN/mTOR pathway. Science 2008;322:963-966.

•Sun F, Park KK, Belin S, Wang D, Lu T, Chen G, Zhang K, Yeung C, Feng G, Yankner BA, He Z. Sustained axon regeneration induced by co-deletion of PTEN and SOCS3. Nature 480, 372-375, 2011.

•Liu K, Lu Y, Lee JK, Samara R, Willenberg R, Sears-Kraxberger I, Tedeschi A, Park KK, Jin D, Cai B, Xu B, Connolly L, Steward O, Zheng B, He Z. PTEN deletion enhances the regenerative ability of adult corticospinal neurons. Nat Neurosci 2010:13(9):1075-1081.

•Nawabi, H., Belin, S., Cartoni, R., Williams, P.R., Wang, C., Latremolière, A., Wang, X., Fu, X., Zhu, J., Taub, D.G., Yu, B., Gu, X., Woolf, C.J., Liu, J.S., Gabel, C.V., Steen, J. A., and He, Z. Doublecortin-like kinases promote neuronal survival and induce growth cone reformation *via* distinct mechanisms. Neuron 88, 704-719, 2015.

•Bei, F., Lee, H.H.C., Liu, X., Gunner, G., Jin, H., Ma, L., Wang, C., Hou, L., Hensch, T.K., Frank, E., Sanes, J.R., Chen, C., Fagiolini, M., and He, Z. Restoration of visual function by enhancing conduction in regenerated axons. Cell 164, 219-232, 2016.

Yifan Cheng, PhD

HHMI Investigator/Professor

Department of Biochemistry and Biophysics

University of California San Francisco

San Francisco, California, USA

E-mail: ycheng@ucsf.edu

Education:
- BS (1982), Physics, Wuhan University
- MS (1987), Physics, Wuhan University
- PhD (1991), Physics, Institute of Physics, Chinese Academy of Sciences

Research Interests:
• Structural Biology, cryo-EM

Honors and Awards:
•

Representative Publications (no more than 5):

Li X, Mooney P, Zheng S, Booth C, Braunfeld MB, Gubbens S, Agard DA* and **Cheng Y*** (2013) Electron counting and beam-induced motion correction enables near atomic resolution single particle cryoEM. *Nature Methods*, **10**, 584-590. PMCID: PMC3684049.

Liao M[§], Cao E[§], Julius D* and **Cheng Y*** (2013) Structure of the TRPV1 ion channel determined by electron cryo-microscopy. *Nature*, **504**, 107-112. PMCID: PMC4078027.

Cao E[§], Liao M[§], **Cheng Y*** and Julius D* (2013) TRPV1 structures in distinct conformations reveal activation mechanisms. *Nature*, **504**, 113-118. PMCID: PMC4023639.

Paulsen CE[§], Armache J-P[§], Gao Y, **Cheng Y*** and Julius D* (2015) Structure of the human TRPA1 ion channel suggests regulatory mechanisms. *Nature*, **520**, 511-517. PMCID: PMC4409540.

Gao Y, Cao E, Julius D* and Cheng Y* (2016) TRPV1 structures in nanodiscs reveal mechanisms of ligand and lipid action. Nature, doi:10.1038/nature17964. NIHMSID: NIHMS774422

Min Li, PhD (利民)
Senior Vice President
Global Head, Neuroscience R&D
GM of R&D China

GSK

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Education:

1984	B.S., Biochemistry, Wuhan University, China
1991	Ph.D., Molecular Biology and Genetics, Johns Hopkins School of Medicine, Baltimore, Maryland
1993	Post-Doctoral Fellow, Molecular Neurobiology and Physiology, University of California San Francisco (USCF)

Positions:
Assistant/Associate/Full Professor, Johns Hopkins School of Medicine (1994-2013)
Senior Vice President, Global Head of Neuroscience R&D, GM of R&D China, GSK (2014-present)

Honors and Awards:
AAAS Fellow, 2012; American Heart Association Established Investigator, 2000; American Heart Association Pfizer Awardee, 1996; Klingenstein Neuroscience Fellow, 1994 ; Sloan Foundation Neuroscience Fellow, 1994; National Institutes of Health Shannon Award, 1994; Helen Hay Whitney Foundation Fellow, 1991; Muscular Dystrophy Association Fellow, 1991

Representative Publications (no more than 5):

Zhang, H., Zou, B., Yu, H., Moretti, A., Yan, W., Babcock, J.J., McManus, O.B., Tomaselli, G., Laugwitz, K.L. & **Li, M.** Modulation of hERG potassium channel gating normalizes action potential duration prolonged by dysfunctional KCNQ1 potassium channel. ***Proc Natl Acad Sci USA*** 109(29), 11866-71. (2012).

Yu, H., Lin, Z., Mattmann, M.E., Zou, B., Terrenoire, C., Zhang, H., Wu, M., McManus, O.B., Kass, R.S., Lindsley, C.W., Hopkins, C.R. & **Li, M.** Dynamic subunit stoichiometry confers a progressive continuum of pharmacological sensitivity by KCNQ potassium channels. ***Proc Natl Acad Sci USA*** 110(21), 8732-7. (2013).

Zhou, P., Yu, H., Gu, M., Nan, F., Gao, Z. & **Li, M.** PIP_2 alters pharmacological selectivity for epilepsy-causing KCNQ potassium channels. ***Proc Natl Acad Sci USA*** 110(21), 8726-31. (2013).

Schreiber, S., Kotz, J., **Li, M.,** Aube, J., Austin, C., Reed, J.,. & NIH Molecular Libraries Project Team. Advancing Biological Understanding and Therapeutics Discovery with Small-Molecule Probes ***Cell*** 161, 1252 -1277 (2015)

Sun, H., Luo, L., Lal, B., Ma, X., Chen, L., Hann, C., Fulton, A., Leahy, D., Laterra, J. & **Li, M**. A monoclonal antibody against KCNK9 K^+ channel extracellular domain inhibits tumour growth and metastasis ***Nature COMMS*** 7, 10339-10344 (2016)

Xiaoliang Sunney Xie, PhD

Mallinckrodt Professor

Harvard University Cambridge, MA, USA

Director

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Education:
- BS (1984), Chemistry, Peking University, Beijing, P.R. China
- PhD (1990), Chemistry, University of California at San Diego, USA

Other Positions:
•Director, Beijing Innovation Center for Genomics, Peking University

Research Interests:
•Single molecule biology: single-molecule enzymology, live-cell gene expression, and single cell genomics

Honors and Awards:
• Albany Prize in Medicine and Biomedical Research
• U. S. Department of Energy E. O. Lawrence Award
• American Chemical Society's Peter Debye Award
• Biophysical Society's Founders Award
• National Institute of Health Director's Pioneer Award
• Sackler Prize for Physical Sciences

Representative Publications (no more than 5):
• Yan, Liying; Huang, Lei; Xu, Liya; Huang, Jin; Ma, Fei; Zhu, Xiaohui; Tang, Yaqiong; Liu, Mingshan; Lian, Ying; Liu, Ping; Li, Rong; Lu, Sijia; Tang, Fuchou; Qiao, Jie; Xie, X Sunney "Live births after simultaneous avoidance of monogenic diseases and chromosome abnormality by next-generation sequencing with linkage analyses" *PNAS* 112(52), 15964-15969 (2015).
• Zong, Chenghang; Lu, Sijia; Chapman, Alec R.; Xie, X. Sunney, "Genome-Wide Detection of Single-Nucleotide and Copy-Number Variations of a Single Human Cell" *Science* 338, 1622-1626 (2012).
•Yu, Ji; Xiao, Jie; Ren, Xiaojia; Lao, Kaiqin; Xie, X. Sunney "Probing Gene Expression in Live Cells, One Protein Molecule at a Time," *Science* 311, 1600-1603 (2006).
• Lu, H. Peter; Xun, Luying; Xie, X. Sunney "Single-Molecule Enzymatic Dynamics," *Science* 282, 1877-1882 (1998).
• Zumbusch, Andreas; Holtom, Gary R.; Xie, X. Sunney "Vibrational Microscopy Using Coherent Anti-Stokes Raman Scattering, " *Phys. Rev. Lett.* 82, 4142-4145 (1999).

Xiang-Dong Fu, PhD

Professor

Department of Cellular and Molecular Medicine

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Education:
- BS (1982), Virology, Wuhan University
- PhD (1988), Biochemistry, Case Western Reserve University
- Postdoctoral training (1988-1992), Harvard University

Research Interests:
•Regulated gene expression at transcriptional and post-transcriptional levels
•RNA processing mechanisms
•Regulatory RNAs and nuclear architecture

Honors and Awards:
•Searle Scholar (1994)
•Leukemia and Lymphoma Society Scholar (1997)
•Distinguished Alumnus of Wuhan University (2003)
•Elected AAAS Fellow (2010)

Representative Publications (last 3 years):
•Fu, X-D. and Ares, M. Jr. (2014). Context-dependent control of alternative splicing by RNA binding proteins. *Nature Review Genetics* 15:689-701.
•Wei, C., Qiu, J., Zhou, Y., Xue, Y., Hu, J., Ouyang, K., Bamerjee, I., Zhang, C., Chen, B., Li, H., Chen, J., Song, L-S., and Fu, X-D. (2015). Repression of the central splicing regulator RBFox2 is functionally linked to pressure overloading-induced heart failure. *Cell Reports* 10:1521-1533.
•Wang, L., Zhou, Y., Xu, L., Xiao, R., Lu, X., Chen, L., Chong, J., Li, H., He, C., Fu, X-D., and Wang, D. (2015). Molecular basis for 5-carboxycytosine recognition by RNA polymerase II elongation transcription complex. *Nature* 523:621-625.
•Xue, Y., Qian, H., Hu, J., Zhou, B., Zhou, Y., Hu, X., Karakhanyan, A., Pang, Z., and Fu. X-D. (2016). Sequential regulatory loops as key gatekeepers for neuronal reprogramming in human cells. *Nature Neuroscience* 19:807-815.
•Wei, C., Xiao, R., Chen, L., Cui, H., Zhou, Y., Xue, Y., Hu, J., Tsutsui, T., Qiu, J., Li, H., Tang, L., and Fu, X-D. (2016). RBFox2 binds nascent RNA to globally regulate polycomb complex 2 targeting in mammalian genomes. *Molecular Cell* 62:875-889.

Ling-Ling Chen, PhD

Principal Investigator

Shanghai Institute of Biochemistry and Cell Biology

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Education:
- BS (2000), Biology, Lanzhou University
- MS (2003), Pharmacology, Shanghai Institute of *Materia Medica*, Chinese Academy of Sciences
- MBA (2009), Management, University of Connecticut School of Business
- PhD (2009), Biomedical Science, University of Connecticut Health Center
- Postdoctoral fellow and Assistant Professor in residence (2009-2010), University of Connecticut Health Center

Other Positions:
- Editorial Board: Trends in Genetics (2015), Genome Biology (2014), Frontiers in noncoding RNAs (2011)

Honors and Awards:
- A-IMBN Research Young Investigator/Ken-ichi Arai Award (2016)
- Li Ruqi Award from the Chinese Genetics Society (2015)

Research Interests:
- Long noncoding RNA biogenesis and function

Representative Publications (no more than 5):

•**LL Chen**. 2016. The Biogenesis and emerging roles of circular RNAs. ***Nat Rev Mol Cell Biol***, 17:205-211.

•Hu SB, Xiang JF, Li X, Xu YF, Xue W, Huang M, Wong CC, Sagum CA, Bedford MT, Yang L, Cheng D and **Chen LL**. 2015. Protein arginine methyltransferase CARM1 attenuates the nuclear retention of mRNAs containing IRAlus at paraspeckles. ***Genes Dev***, 29: 630-645.

•Zhang XO, Wang HB, Zhang Y, Lu X, **Chen LL** and Yang L. 2014. Complementary sequence-mediated exon circularization. ***Cell***, 159:134-147.

•Zhang Y, Zhang XO, Chen T, Xiang JF, Yin QF, Xing YH, Zhu S, Yang L and **Chen LL**. 2013. Circular intronic long noncoding RNAs, ***Mol Cell***, 51: 792-806.

•Yin QF#, Yang L#, Zhang Y, Xiang JF, Wu YW, Carmichael GG and **Chen LL**. 2012. Long noncoding RNAs with snoRNA ends. ***Mol Cell***, 48: 219-230. (Best of Mol Cell 2012)

Hai Qi, PhD

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Education:
- BM (1996), Clinical Medicine, Beijing Medical University
- PhD (2003), Experimental Pathology, University of Texas Medical Branch, United States
- Postdoctoral training (2003-2009), Laboratory of Immunology, NIAID/NIH, United States

Other Positions:

•Vice Dean, School of Medicine, Tsinghua University

Research Interests:

• Humoral immune regulation and germinal center biology

Honors and Awards:

2014 "Distinguished Young Scientist", National Science Foundation of China
2014 "Young Talent in Science and Technology" Award, Ministry of Science & Technology of China
2015 "Tan Jiazhen Life Sciences Award" Innovation Award
2015 "Cheung Kong Scholar" Award, Ministry of Education, China

Representative Publications (no more than 5):

•Chen X*, Tang S*, Zheng JS*, Zhao R, Wang ZP, Shao W, Chang HN, Cheng JY, Zhao H, Liu L#, **Qi H#**. Chemical synthesis of a two-photon-activatable chemokine and photon-guided lymphocyte migration in vivo. ***Nature communications*** 6:7220, 2015.

•Liu D*, Xu HP*, Shih CM, Wan ZP, Ma XP, Ma WW, Luo D, **Qi H#**. T-B-cell entanglement and ICOSL-driven feed-forward regulation of germinal centre reaction. ***Nature*** 517:214-218, 2015.

•Xu HP, Li XY, Liu D, Li JF, Zhang X, Chen X, Hou SY, Peng LX, Xu CG, Liu WL, Zhang LF, **Qi H#**. Follicular T-helper cell recruitment governed by bystander B cells and ICOS-driven motility. ***Nature*** 496:523-527, 2013.

•**Qi H***, Cannons JL*, Klauschen F, Schwartzberg PL, Germain RN#. SAP-controlled T-B cell interactions underlie germinal centre formation. ***Nature*** 455:764-769, 2008.

•**Qi H**, Egen GJ, Huang AYC, Germain RN#. Extrafollicular activation of lymph node B cells by antigen-bearing dendritic cells. ***Science*** 312:1672-1676, 2006.

CBIS Announces Recipients of 2016 Awards

The Chinese Biological Investigators Society (CBIS) is pleased to announce the recipients of this year's Ray Wu Award, Young Investigator Award, and Teaching award.

The Ray Wu Award was established by the society to honor the late Dr. Ray Wu, who not only had a distinguished scientific career but also nurtured a new generation of Chinese scientists in life sciences through his tireless effort in promoting scientific and educational exchanges between China and the United States. The Award recognizes CBIS members who have made fundamental discoveries in life sciences and/or significant contributions in promoting life sciences in China. This year's recipients are:

Dr. Xiaoliang Sunney Xie, the Mallinckrodt Professor at Harvard University and the Director of BIOPIC at Peking University, for his contributions in single-molecule enzyme kinetics and gene expression, his transformative innovations in single-cell genomics, and his pioneering work on CARS microscopy that allows 3D imaging of live cells and organisms.

Dr. Xiang-Dong Fu, a Professor in the Department of Cellular and Molecular Medicine, University of California, San Diego, for his important contributions in elucidating the mechanism of RNA splicing as well as how regulatory RNAs and RNA binding proteins modulate gene expression.

The Young Investigator Award recognizes CBIS members who are in the early career stages but have already made remarkable contributions in their respective fields. This year's awardees are:

Dr. Ling-ling Chen, a Principal Investigator at the Shanghai Institute of Biochemistry and Cell Biology, Shanghai Institutes for Biological Sciences, Chinese Academy of Sciences, for her contributions to the field of RNA Biology, particularly for revealing new classes of broadly expressed new lncRNA species, including snoRNA-lncRNAs and circular RNAs, as well as their biogenesis and functional potential.

Dr. Hai Qi, a Professor in the Institute for Immunology, Department of Basic Medical Sciences, School of Medicine, Tsinghua University, for his important contributions in the field of humoral immune regulation and seminal discoveries in T follicular helper and germinal center biology.

The Teaching Award recognizes a CBIS member who has contributed extraordinarily to education in biomedical sciences, particularly in China. This year's awardee is:

Dr. Chenjian Li, a Professor in the School of Life Sciences, Peking University. He is also an Associate Dean of the School and a Vice Provost of the university.

Awardee Biographies

Xiaoliang Sunney Xie, PhD
Mallinckrodt Professor
Harvard University Cambridge, MA, USA
Director
BIOPIC, Peking University
E-mail: xie@chemistry.harvard.edu

Xiaoliang Sunney Xie received a B.S. from Peking University in 1984, Ph.D. from the University of California at San Diego in 1990, and did postdoctoral work at the University of Chicago. He joined Pacific Northwest National Laboratory in 1992 and rose to a Chief Scientist there. In 1999, he was appointed full Professor at Harvard University and became the first tenured professor at Harvard from the People's Republic of China since China's reform in 1978. He is currently the Mallinckrodt Professor of Chemistry and Chemical Biology at Harvard and the founding Director of Biomedical Institute of Pioneering Investigation via Convergence (BIOPIC) at Peking University.

Xie made major contributions to the emergence of the field of single-molecule biophysical chemistry and its application to biology. He also pioneered the development of coherent Raman scattering microscopy and single cell whole genome sequencing.

His honors include the Albany Prize in Medicine and Biomedical Research, the U. S. Department of Energy E. O. Lawrence Award, the Biophysical Society's Founders Award, the Sackler Prize for Physical Sciences and the American Chemical Society's Peter Debye Award. Xie is a fellow of the American Academy of Arts and Sciences and a member of the National Academy of Sciences.

Xiang-Dong Fu, Ph.D.
Professor
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Xiang-Dong Fu is a Professor of Cellular and Molecular Medicine at University of California at San Diego. He earned his B.S. degree from Wuhan University in 1982 and his Ph.D. degree from Case Western Reserve University in 1987. After postdoctoral training at Harvard, he joined the faculty of UC, San Diego as an Assistant Professor in 1992. He rose to the rank of Associate Professor in 1998 and Full Professor in 2002. He did pioneering research in RNA processing, regulated gene expression at both transcriptional and post-transcriptional levels, and RNA genomics. He discovered the founding member of the SR family of splicing factor and elucidated their roles in constitutive and regulated pre-mRNA splicing. He also discovered the SRPK family of splicing kinases that are highly specific for SR proteins and elucidated a dedicated signaling pathway via these kinases to transduce growth factor signaling to the nucleus to regulate alternative splicing. He pioneered studies on cell fate switches mediated by regulatory RNAs and RNA binding proteins and revealed diverse mechanisms for their functions in mammalian genomes to activate or repress transcription during cellular reprogramming. Dr. Fu is a founding member of the Ray Wu Society. In keeping Dr. Wu's inspirit, he has been actively promoting science, especially RNA research, in China by interacting, collaborating, mentoring junior scientists. He has extensively participated in institutional reviews, study sessions, and organization of scientific meetings in China. He is now serving as the Director of Chinese Academy of Science Key Laboratory for Nucleic Acid Research at the Institute of Biophysics.

Ling-Ling Chen, PhD

Principal Investigator

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Ling-Ling Chen is an Investigator at the Shanghai Institute of Biochemistry and Cell Biology (SIBCB), SIBS, CAS. She obtained a B.S. degree from Lanzhou University in 2000, a M.S degree from Shanghai Institute of *Materia Medica*, CAS in 2003 and a Ph.D-MBA dual degree from University of Connecticut in 2009. After one-year postdoctoral training at UConn Health Center, she was promoted as an Assistant Profession in residence there in 2010, and then joined SIBCB as a Principal Investigator in 2011. Dr. Chen studies long noncoding RNAs (lncRNAs), a giant and varied class of RNA molecules. She has made fundamental discoveries regarding the biogenesis and functional potential of several classes of lncRNA species. She pioneered methods for genome-wide characterization of non-polyadenylated RNAs, which led to the identification of broadly expressed intron-derived snoRNA-ended lncRNAs, circular intronic RNAs derived from intron lariats and circular RNAs produced from back-spliced exons by RNA pairing in flanking introns. These studies have uncovered unexpected types of lncRNAs with functional potential and roles for previously considered "junk" introns in shaping complex mammalian transcriptomes. She serves on Editorial Boards of Trends Genet and Genome Biol, and is the recipient of Liquqi Award from the Chinese Genetics Society (2015) and A-IMBN Research Young Investigator/Ken-ichi Arai Award (2016).

Hai Qi, PhD

Professor

Institute for Immunology

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Hai Qi is a Professor of Immunology in the School of Medicine at Tsinghua University. He obtained his B.M. degree from Beijing Medical University in 1996 and his Ph.D. degree from University of Texas Medical Branch at Galveston in 2003. After completing postdoctoral training at NIAID/NIH, he joined the faculty of Tsinghua University as a tenure-track Associate Professor in 2009 and became a full professor in 2015. Dr. Qi has made fundamental discoveries in humoral immune regulation, particularly in the area of follicular T-helper cells and germinal center biology. He has made creative use of intravital imaging, combined with sophisticated mouse models, to uncover novel cellular dynamics and molecular mechanisms for B cell activation, T-B lymphocyte interactions, and germinal center reaction. His findings are highly significant for our basic understanding of how long-lived humoral immune memory is formed and for our quest of future vaccines to induce durable antibody protection against microbial infection. Dr. Qi is the recipient of several prestigious awards, including a Tian Jiazhen Life Science Innovation Award (2015) and a Cheung Kong Scholar Award (2015).

Chenjian Li, Ph.D.
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Dr. Chenjian Li is currently the Vice Provost of Peking University, and Professor and Associate Dean of School of Life Science, Peking University. Prior to his return to China, he was an assistant professor and associate professor at Weill Medical College of Cornell University (2003—2009), and then Aidekman Endowed Chair of Neurology at Mount Sinai School of Medicine (2010—2013). Dr. Li's scientific research focuses on exploring the molecular and cellular mechanisms of neurological diseases.

Parallel to research, Dr. Li is extremely devoted to education development and reforms, ranging from high school, undergraduate, graduate and medical student education. He was one of the organizers of the influential "*Science Outreach Program*" in USA. Since his return to China, he has played a leading role in important initiatives such as the establishment of *Cambridge-PKU Center for China Study*, inauguration of *Rhodes Scholar program in China*, reform of college admission by holistic evaluation, design and implementation of liberal education curriculum at PKU, etc. He received many awards for excellence in teaching, including a student-voted “Pied Piper Mater” at Weill Cornell Medical College in 2006, and a student-voted “Best Teacher” at PKU in 2015.

CBIS 2016 Chengdu Meeting Abstracts

1 Chang Qiang University of Wisconsin-Madison qchang@waisman.wisc.edu

Rett syndrome (RTT) is a debilitating neurodevelopmental disorder caused by mutations in the X-linked human methyl-CpG binding protein 2 (MECP2) gene, which predominantly affects females. The disease is complex, as all key cell types (neurons, astrocytes, microglia, and oligodendrocytes) in the brain have been shown to contribute to the disease etiology. To fully understand the disease mechanism and develop effective treatments, it is essential to define the key phenotypes, link the phenotypes with loss of MeCP2 function, and reveal the consequences of the phenotypes in each cell type; and to study how these cells interact. While earlier research mostly focused on neuronal dysfunction in RTT, more recent studies have clearly demonstrated that astrocytes express MeCP2, loss of MeCP2 in astrocytes causes neuronal defects, and restoring MeCP2 expression to normal in astrocytes alleviates disease symptoms. Although a few studies have reported gene expression changes and phenotypes in Mecp2 mutant mouse astrocytes, it is not clear how these alterations contribute to RTT pathogenesis either by directly changing astrocyte functions or by indirectly changing neuronal functions. Therefore, it is necessary to systematically investigate the astrocyte cell autonomous phenotypes, the underlying mechanism of the phenotypes, and the functional consequence of the phenotypes. We have discovered significant changes in cytosolic calcium homeostasis in RTT astrocytes in the absence and presence of neurons, revealed potential molecular and cellular mechanisms underlying the abnormal calcium homeostasis, and identified major functional consequence of this astrocyte cell autonomous phenotype on neighboring neurons and the neural network. Our study employs various RTT mouse models (germline knockout mice, cell type specific conditional knockout mice, and cell type specific conditional reactivation mice), the RTT patient-specific induced pluripotent stem cell (iPSC) model, and genetically engineered human embryonic stem cell (hESC) model. By combining the strengths from these complementary models, we can cross-validate our findings between species and between in vitro and in vivo, therefore generating more valid insights. Our findings significantly advance the understanding of RTT disease mechanism and facilitate future development of therapies to treat RTT.

2 Chen Qing The Wistar Institute qichen@wistar.org

Brain metastasis represents a substantial source of morbidity and mortality in various cancers, and is characterized by high resistance to chemotherapy. Here we define the role of the most abundant cell type in the brain, the astrocyte, in promoting brain metastasis. We show that human and mouse breast and lung cancer cells express protocadherin 7 (PCDH7), which promotes the assembly of carcinoma-astrocyte gap junctions composed of connexin 43 (Cx43). Once engaged with the astrocyte gap-junctional network, brain metastatic cancer cells use these channels to transfer the second messenger cGAMP to astrocytes, activating the STING pathway and production of inflammatory cytokines such as interferon-α (IFNα) and tumour necrosis factor (TNF). As paracrine signals, these factors activate the STAT1 and NF-κB pathways in brain metastatic cells, thereby supporting tumour growth and chemoresistance. The orally bioavailable modulators of gap junctions meclofenamate and tonabersat break this paracrine loop, and we provide proof-of-principle that these drugs could be used to treat established brain metastasis.

3 Chen Xiaoke Stanford University xkchen@stanford.edu

Chronic opiate use induces opiate dependence, which is characterized by extremely unpleasant physical and emotional feelings after drug use is terminated. Both the rewarding effects of a drug and the desire to avoid withdrawal symptoms motivate continued drug use, and the nucleus accumbens is important for orchestrating both processes. While multiple inputs to the

nucleus accumbens regulate reward, little is known about the nucleus accumbens circuitry underlying withdrawal. Here we identify the paraventricular nucleus of the thalamus (PVT) as a prominent input to the nucleus accumbens mediating the expression of opiate-withdrawal-induced physical signs and aversive memory. Activity in the PVT to nucleus accumbens pathway is necessary and sufficient to mediate behavioural aversion. Selectively silencing this pathway abolishes aversive symptoms in two different mouse models of opiate withdrawal. Chronic morphine exposure selectively potentiates excitatory transmission between the PVT and D2-receptor-expressing medium spiny neurons via synaptic insertion of GluA2-lacking AMPA receptors. Notably, in vivo optogenetic depotentiation restores normal transmission at these synapses and robustly suppresses morphine withdrawal symptoms. This links morphine-evoked pathway- and cell-type-specific plasticity in the PVT to nucleus accumbens circuit to opiate dependence, and suggests that reprogramming this circuit holds promise for treating opiate addiction.

4 Chen Jia Shanghai Tech University chenjia@shanghaitech.edu.cn

CRISPR/Cas9 systems are robust and adaptable genome editing tools that rely on hybridization with guide RNAs (e.g., single guide RNA, sgRNA) to target specific DNA sequences. Once introduced by CRISPR/Cas9 system, DNA double strand breaks (DSBs) trigger homology-directed repair (HDR) or non-homologous end-joining (NHEJ), which could be exploited to replace or inactivate genes for therapeutic applications. Lots of efforts have been made to improve the efficiency and specificity of CRISPR/Cas9 systems, including a double nicking strategy. Surprisingly, it was later revealed that a single sgRNA/Cas9-nickase combination could still induce unwanted mutations through an unknown mechanism. We show here that the HDR-mediated gene correction events are not absolutely mutation free at on-target sites. Importantly, the observed mutations in both CRISPR/Cas9 triggered HDR and CRISPR/Cas9-nickase induced NHEJ are prone to occur on C of TpC dinucleotides, manifesting typical APOBEC3 cytidine deaminases mutagenesis patterns. In both cases, the mutation frequencies positively correlate with APOBEC3 expression level. In addition, the binding of APOBEC3 on genomic DNAs is specifically enriched in the regions near CRISPR/Cas9 target sites. Given that APOBECs are present in most cells and play an important role in endogenous mutagenesis, our findings not only shed new light on the mechanism of genomic mutagenesis in both Cas9-triggered HDR and Cas9-nickase-induced genome nicking, but also suggest a practical strategy to improve the specificity and preciseness of CRISPR/Cas9-induced genome editing by inhibiting APOBEC3 activity.

5 Chen Junjie The University of Texas MD Anderson Cancer Center jchen8@mdanderson.org

DNA double strand breaks (DSBs) are repaired by NHEJ and HR pathways in mammalian cells. It is believed that which pathway to use for DSB repair is mainly controlled by end resection process. While BRCA1 promotes end resection and therefore favors homologous recombination (HR) repair, 53BP1 inhibits end resection and engages nonhomologous end joining (NHEJ) pathway for DSB repair. We and others have shown previously that RIF1 is a major downstream effector of 53BP1 and participates in 53BP1-dependent inhibition of end resection. Interestingly, while RIF1 accumulation at DSBs is antagonized by BRCA1 in S and G2 phases, the translocation of BRCA1 to damage sites in G1 cells is inhibited by RIF1, indicating that 53BP1-dependent pathway and BRCA1 counteract each other in a cell cycle-dependent manner. Moreover, another DNA damage repair protein PTIP was also shown to act downstream of 53BP1 and counteract BRCA1 in DNA repair. Unfortunately, 53BP1, PTIP and RIF1 are adaptor proteins, which lack any enzymatic activities that would be directly involved in DNA repair. Therefore, it remained unknown precisely how the 53BP1-dependent pathway counteracts with BRCA1 in DNA repair until very recent studies from others and us. Excitingly, we discovered a nuclease SNM1C/Artemis that associates with PTIP and functions to prevent end resection and HR repair. In addition, two recent studies indicated that REV7/MAD2L2 acts downstream of RIF1 and inhibits HR repair. We are now

further investigating the regulation of DSB and other repair pathways. Together, these studies help us understand how DNA repair pathway choice is determined following DNA damage, which dictates therapeutic outcomes.

6 Chen Gong Penn State University gongchenpsu@yahoo.com

"Glial scar is widely associated with brain and spinal cord injury, stroke, glioma, and neurodegenerative disorders such as Alzheimer's disease. Reactive glia initially exert neuroprotective role but later form glial scar to inhibit neuronal growth. Currently, there is no effective way to reverse glial scar back to neural tissue. We have recently developed an innovative in vivo reprogramming technology to directly convert reactive glial cells into functional neurons inside the mouse brain (Guo et al., Cell Stem Cell, BEST of 2014 article). This is achieved through in vivo expression of a single neural transcription factor NeuroD1 in the reactive astrocytes in injured mouse brain or Alzheimer's disease mouse model. Our in vivo cell conversion technology makes use of internal glial cells to regenerate new neurons, making it possible for the first time in history to reverse glial scar back to neural tissue. Such internal cell conversion method will avoid transplantation of external cells and its associated immune rejection. We have further discovered a cocktail of small molecules that can directly convert cultured human astrocytes into functional neurons (Zhang et al., Cell Stem Cell, 2015), paving the way for a potential drug therapy for human brain repair. This project was supported by grants from NIH, Alzheimer's Association, and Charles H. Skip Smith Endowment Fund. G.C. is Verne M. Willaman Endowed Chair in Life Sciences at Penn State University."

7 CHEN LIN-FENG University of Illinois at Urbana-Champaign lfchen@life.illinois.edu

H. pylori infection causes chronic gastritis and peptic ulceration. H. pylori-initiated chronic gastritis is characterized by enhanced expression of many NF-kB-regulated inflammatory cytokines. Brd4 has emerged as an important NF-kB regulator and regulates the expression of many NF-kB-dependent inflammatory genes. In this study, we demonstrated that Brd4 was not only actively involved in H. pylori-induced inflammatory gene mRNA transcription but also H. pylori-induced inflammatory gene eRNA synthesis. Suppression of H. pylori-induced eRNA synthesis impaired H. pylori-induced mRNA synthesis. Furthermore, H. pylori stimulated NF-kB-dependent recruitment of Brd4 to the promoters and enhancers of inflammatory genes to facilitate the RNAPII-mediated eRNA and mRNA synthesis. Inhibition of Brd4 by JQ1 attenuated H. pylori-induced eRNA and mRNA synthesis for a subset of NF-kB-dependent inflammatory genes. JQ1 also inhibited H. pylori-induced interaction between Brd4 and RelA and the recruitment of Brd4 and RNAPII to the promoters and enhancers of inflammatory genes. Finally, we demonstrated that JQ1 suppressed inflammatory gene expression, inflammation, and cell proliferation in H. pylori-infected mice. These studies highlight the importance of Brd4 in H. pylori-induced inflammatory gene expression and suggest that Brd4 could be a potential therapeutic target for the treatment of H. pylori-triggered inflammatory diseases and cancer.

8 Chen Guokai University of Macau guokaichen@umac.mo

Human Pluripotent Stem Cells (hPSCs) are very sensitive to the culture environment and are a lot more prone to cell death in suboptimal conditions compared to undifferentiated cells. The cell death could lead to the accumulation of abnormal cells, inconsistency of cell culture results, and high probability of production failure. This is detrimental to various applications, such as disease modeling and cell-based therapies. At the same time, cell death is observed during differentiation, indicating potential connection between cell fate determination and cell death. The understanding of the death mechanisms at different conditions is critical for the improvement of stem cell technologies for different application. Here we report our new strategies to improve the cell culture conditions for stem cell maintenance and differentiation.

9 Chen Lu University of California at Berkeley chenlu@berkeley.edu

Lymphatic research has progressed rapidly in recent years. The cornea provides an ideal tissue for lymphatic research due to its accessible location, transparent nature, and inducible vascularity. Once induced by an inflammatory, infectious or chemical insult, corneal lymphatics enhance high volume delivery of antigens and immune cells, and accelerate transplant rejection. Our research goal is to elucidate the molecular and cellular mechanisms of lymphangiogenesis and to identify new targets for therapeutic intervention. This talk will introduce our recent advances in corneal lymphatic research, which bear broad implications both inside and outside the eye.

10 Chen Dong Feng Massachusetts Eye and Ear, Harvard Medical School dongfeng_chen@meei.harvard.edu

Some adult-onset disorders may be linked to dysregulated embryonic development, yet the mechanisms underlying this association remain poorly understood. Congenital retinal degenerative diseases are blinding disorders characterized by postnatal degeneration of photoreceptors, and affect nearly 2 million individuals worldwide, but ~50% do not have a known mutation, implicating contributions of epigenetic factors. We found that embryonic deletion of the histone methyltransferase (HMT) Ezh2 from all retinal progenitors resulted in progressive photoreceptor degeneration throughout postnatal life, via derepression of fetal expression of Six1 and its targets. Forced expression of Six1 in the postnatal retina was sufficient to induce photoreceptor degeneration. Ezh2, although enriched in the embryonic retina, was not present in the mature retina; these data reveal an Ezh2-mediated feed-forward pathway that is required for maintaining photoreceptor homeostasis in the adult and suggest novel targets for retinal degeneration therapy.

11 CHEN ZHIJIAN UT SOUTHWESTERN MEDICAL CENTER zhijian.chen@utsouthwestern.edu

The presence of DNA in the cytoplasm is a danger signal that alerts the host immune system to eliminate microbial infections, but inappropriate activation of these pathways can also lead to autoimmune diseases such as lupus. My talk will focus on our recent discoveries of the enzyme cGAS as an innate immune sensor for cytosolic DNA, and of cyclic GMP-AMP (cGAMP) as a novel second messenger that triggers the production of type-I interferons and other inflammatory cytokines.

12 Chen Jing Winship Cancer Institute, Emory University jchen@emory.edu

The terms metabolic "reprogramming" and "rewiring" have emerged to describe the increasingly emphasized metabolic changes in cancer cells. However, they have been used interchangeably without appreciation for their mechanistic distinction and biological implications. We were the first to clearly define and distinguish them. We have accomplished a series of work that lays the foundation for a novel notion that "metabolic reprogramming" should represent "software" changes in cancer cells and describe metabolic alterations that are normally induced by growth factors in proliferating cells, but "hijacked" by oncogenic signals; and "metabolic rewiring" should represent "hardware" changes and describe newly "forged" metabolic alterations due to "neo-function" of distinct oncogenic mutants, which are not found in normal cells. Our major conceptual contribution is that, besides “long-term” mechanisms involving gene expression, oncogenic signals also “reprogram” cancer cell in an “acute” manner involving diverse post-translational modifications (PTMs) including tyrosine phosphorylation and lysine acetylation of diverse key metabolic enzymes (Fan et al, Molecular Cell 2014; Shan et al, Molecular Cell 2014), and signal transduction mediated through metabolites that function as signaling molecules (Hitosugi et al, Cancer Cell 2012; Lin et al, Nature Cell Biology 2015). Moreover, oncogenic mutations may also “rewire” cell metabolism by forging a new pathway that is exclusive in cancers. This is supported by the recent identification of the mutations in isocitrate dehydrogenase (IDH) 1 and 2 in glioma and acute myeloid leukemia,

which confer a “neo-function” to IDH and enable the enzyme to produce the oncometabolite 2-hydroxyglutamate to regulate cancer epigenetics. Our recent study also supported this novel mechanism and showed that oncogenic BRAF V600E “rewires” ketogenesis to MAPK pathway, where a ketone body acetoacetate selectively promotes V600E-dependent MEK1 activation (Kang et al, Molecular Cell 2015). Thus, clearly distinguishing and characterizing metabolic "reprogramming" and "rewiring" in cancer cells will particularly inform therapy development. I will discuss our recent progress in development of small molecule inhibitors of key metabolic enzymes such as PGAM1 and 6PGD, as well as metal ion homeostasis such as a novel inhibitor of copper trafficking (Wang et al, Nature Chemistry 2015).

13 Dong Xinnian Duke University xdong@duke.edu

Effector-triggered immunity (ETI), a major defense mechanism in plants, is often associated with programmed cell death (PCD). However, plants lack close homologs of caspases, the key mediators of PCD in animals. In my talk, I will present our recent work showing that the Arabidopsis nuclear envelope protein, CPR5, negatively regulates ETI and PCD through a physical interaction with cyclin-dependent kinase inhibitors (CKIs). Upon ETI induction, CKIs are released from CPR5 to cause overactivation of another core cell-cycle regulator, E2F. In cki and e2f mutants, ETI responses induced by both TIR-NB-LRR and CC-NB-LRR classes of immune receptors are compromised. We further show that E2F is deregulated during ETI, probably through CKI-mediated hyperphosphorylation of retinoblastoma-related 1 (RBR1). This study demonstrates that canonical cell cycle regulators play important noncanonical roles in plant immunity. I will also present data on how CPR5 in the nuclear pore complex regulates this signaling pathway in response to NB-LRR induction."

14 Feng Jiaxuan Changhai hospital, the first affiliated hospital to the Second Military Medical University fengjiaxuan01@163.com

"Background and Objective: Endovascular stent-graft repair was introduced as an important alternative to conventional open surgery for the treatment of aortic dissections. But studies reported various complications caused by stent-grafts, among which, stent graft-induced new entry is not a rare complication and with high mortality. Despite of its importance, investigation on this aspect has been least carried out and the underlying mechanisms are still unknown. As an alien objective, the endograft is much stiffer than the host arterial tissues. Moreover, when it is deployed, it can induce high mechanical stress concentrations over the contact region. Therefore the hypothesis of this study is that the high stress concentration induced by the dismatch between the endograft and the aortic wall, and the local hemodynamic effects response for the onset of complications.
Methods: This study was conducted in three aspects: First, retrospectively analyze the clinical data to sieve out the targets for following biomechanical analysis; Second, accurately calculate and depict the stress concentration over the contact region on cases with complications, by computational hemodynamic simulation and the mechanical analyses of the interaction between the endograft and the aortic wall. These analyses will explain the associations between morphological features and the subsequent clinical events; Third, test and analyze the biomechanical effects on the aorta as a whole, as well as on the local artery tissue of different contact regions. At last, integrating these results into a hazard regression model will help to find out the histologic evidence of the mechanical and hemodynamic effects, and to identify the best stent-graft configuration for respective aortic dissection.
Result: In the Meta analysis study, we searched all published studies from January 1998 through December 2008 on endovascular stent-graft placement treating acute AD (type B-AD or retrograde type A-AD with an entry tear in the descending aorta) conducted by Chinese investigators. We found significantly higher post-operation complications in patients undergoing stent-graft placement with acute A than those with chronic AD. Patients with acute AD had significantly higher in-hospital mortality than patients with chronic AD. Then we study the single-center data of 674 Stanford type B

aortic dissections treated by TEVAR, From April 1997 to March 2010. We found that the preoperative mismatch rate and follow-up mismatch rate of the SIDR were significantly higher than that of the non-SIDR. Compared with the standard TEVAR, TEVAR+RBS was with lower incidence of SIDR and less secondary intervention. The placement of RBS significantly expanded the true lumen at the level of descending aorta with narrowest true lumen and at the level of distal end of stent-graft. Through the computational fluid dynamics study, we demonstrated that the RBS could reduce the high wall shear stress on the stenosis part of the true lumen of descending aorta. This would further make the wall pressure of the abdominal true lumen higher than false lumen in the same level, which could improve the morphological remodeling in the long term. Besides, RBS could improve the perfusion of the abdominal visceral arteries. In the study of solid mechanics exerted by stent-graft, we found that the stent structure with thinner struts and more crowns imposed smaller stress on the aortic wall. And the RBS could further reduce the wall stress. Through the mechanical properties study of aortic wall, we found the normal aortic wall is stiffer than aortic dissection and aneurysm wall.
Conclusions: The solid mechanics from stent-graft, the hemodynamic effect from the aortic flow, and the weakness of the aortic dissection wall all contribute the occurrence of SIDR. This study provides important in-sight on the mechanism of stent graft-related complications, and it will be essential for optimizing the stent-graft design to preferably match the aortic dissection configuration. As biomechanical rationales of endovascular treatment of aortic dissection, the outcomes of this series of study may have great clinical potential."

15 Gao Ning Tsinghua University ninggao@tsinghua.edu.cn

Ribosome biogenesis is a highly complex process in eukaryotes, involving temporally and spatially regulated ribosomal protein (r-protein) binding and rRNA remodeling events in the nucleolus, nucleoplasm and cytoplasm. Hundreds of assembly factors (AFs), organized into sequential functional groups, facilitate and guide the maturation process into productive assembly branches in and across different cellular compartments. However, the precise mechanisms by which these AFs function are largely unknown. Here, we use cryo-electron microscopy (cryo-EM), to characterize the structures of yeast nucleoplasmic pre-60S particles affinity-purified using the epitope-tagged AF Nog2. Our data pinpoint the locations and determine the structures of over 20 AFs, which are enriched in two areas, an arc region extending from the central protuberance (CP) to the polypeptide tunnel exit (PTE), and the domain including the internal transcribed spacer 2 (ITS2) that separates 5.8S and 25S rRNAs. These structural data suggest that the arc-located factors might function to chaperone formation of RNA helices found in the active sites of the subunit, including helices from the CP, peptidyl-transferase center (PTC) and intersubunit bridge. In particular, two regulatory GTPases, Nog2 and Nog1, act as hub proteins to interact with multiple, distant AFs and functional rRNA elements, manifesting their critical roles in structural remodeling checkpoints and nuclear export. Moreover, our snapshots of compositionally and structurally different pre-60S intermediates provide essential mechanistic details for three major remodeling events before nuclear export: rotation of the 5S RNP, construction of the active center, and ITS2 removal. Therefore, our structures provide a framework to understand the molecular roles of diverse AFs, and potentially the atomic information therein constitutes a resource to generalize principles governing the elusive functions nuclear RNA-binding proteins.

16 Gao Guangping University of Massachusetts Medical School guangping.gao@umassmed.edu

The central nervous system (CNS) is an important target for gene therapy of neurological disorders. Adeno-associated virus vector hold the promise for therapeutic gene transfer to treat a variety of diseases including the CNS disorders. Targeted infusion of AAV vectors into discrete structures in the CNS is highly effective for diseases with a defined localized lesion such as Parkinson’s disease, or those where local expression of the transgene is sufficient to modify the

overall disease phenotype such as CNS gene therapy for GM2 Gangliosidoses, which includes both Tay-Sachs and Sandhoff Diseases. However, for diseases that affect large areas of the CNS such as Canavan's disease and Spinal Muscular Atrophy (SMA), administration of gene therapy vectors through vasculature to target the CNS globally is an attractive approach but hindered by the blood-brain-barrier (BBB). Recent progresses in developing novel AAV vectors and delivery methods to cross the BBB and target the CNS systemically as well as their preclinical applications in gene replacement, gene silencing and genome editing therapy as well as animal modeling will be discussed.

17 Gong Ming University of Kentucky ming.gong@uky.edu

Blood pressure (BP) undergoes daily oscillations: BP is lowest at night (nocturnal dip) and rises before awakening (morning surge). Such BP circadian organization is essential for optimum health. Diabetic patients have a high prevalence of BP circadian rhythm disruption mostly manifested as increased night time BP or non-dipping profile. Accumulating clinical studies demonstrate a striking pathophysiological link between end-organ damage and non-dipping BP. Moreover, a decrease of non-dippers is associated with a decrease of cardiovascular episode. However, the mechanisms link diabetes to non-dipping BP is poorly understood and there is no effective strategy for prevention or treatment of diabetic non-dipping BP. We have found that the severely disrupted BP circadian rhythm in diabetic db/db mice is associated with vascular clock gene Bmal1 alterations, and loss of vascular contractility time-of-the-day variations. Selective deletion of Bmal1 from smooth muscle, but not from cardiomyocytes, compromised BP circadian rhythm and decreased blood pressure without affecting central SCN-controlled locomotor activity in murine models. In mesenteric arteries, BMAL1 bound to the promoter of and activated the transcription of Rho-kinase 2 (ROCK2), and Bmal1 deletion abolished the time-of-day variations in response to agonist-induced vasoconstriction, myosin phosphorylation, and ROCK2 activation. Moreover, restricting food intake time to only during the active phase recovered the normal circadian oscillations of BP and Bmal1 in db/db mice. Together, our studies indicate that Bmal1 plays a critical role in the daily control of vasoconstriction and BP circadian rhythm in physiology and dysfunction of Bmal1 significantly contribute to the non-dipping BP in diabetes.

18 Gong Xiaohua University of California, Berkeley xgong@berkeley.edu

Mammalian eye lenses mainly utilize intercellular gap junctions formed by Gja3 (Cx46) and Gja8 (Cx50). Our previous studies have demonstrated that Gja8 gap junctions are required for lens size and transparency while Gja3 gap junctions are critical for the fiber-to-fiber coupling and transparency in the lens core; in addition, increased lens gap junction proteins through knock-in Gja3 could prevent certain congenital cataracts. Morphological studies suggest that lens transparency relies on precisely packed lens fibers with unique surface interlocking structures such as ball-and- sockets (BS) and protrusions to minimize light scattering. Our recent data reveal abnormally packed fiber cells, decreased F-actin expression at tricellular vertices, and a loss of BS structures in Gja8 knockout (KO) lenses. Gja8 gap junctions likely provide structural domains for stabilizing BS structures. Loss of Gja8 communication and BS structures probably decreases metabolic exchanges between fibers, subsequently impairs fiber cell elongation and maturation, and ultimately results in small lenses. In contrast, Gja3 KO mice display severe nuclear cataracts in the 129 strains but mild opacities in the C57BL/6J (B6) strain. We have determined that periaxin (Prx), which encodes a scaffold protein with four amino acid variances between 129 and B6 strains, modulates cataractogenesis. The 129-Prx variant shows abundantly expressed proteins associating with F-actin to cause the formation of aberrant surface protrusions, impairing lens fiber cell packing and causing inner fiber degeneration to form severe cataracts. The B6-Prx variant leads to the absence of Prx proteins in inner fibers, resulting in mild cataracts. Therefore, lens transparency is achieved through the coordinative functions of gap junctions and cytoskeletal elements by controlling the morphogenesis of the precise surface architecture of lens fibers during development.

Genetic variances of these genes have been detected in the human genomes project, this further indicates that genetic variances in combination with fiber cell surface markers will be important for predicting or delaying the development and severity of cataracts.

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Pluripotency induction from somatic cell state by Yamanaka factors is a low efficiency event, which is considered to be stochastic and restricted by key bottleneck events. To reveal the molecular and cellular details of reprogramming, we devised a live cell imaging system, using which we have achieved direct visualization of pluripotency induction from hematopoietic progenitors at single cell resolution. Reconstruction of individual reprogrammed lineages led to the discovery that all successfully reprogrammed cells initiated fate transition from an ultrafast cell cycle averaging about 8 hours/cycle. These ultrafast cycling hematopoietic progenitors activate their endogenous Oct4 within 4-5 cell divisions, with all progeny progressing toward pluripotency. Thus, pluripotency induction from these somatic cells do not appear to be stochastic. We further demonstrate that their unique reprogramming behavior is related to their ultrafast cell cycle. Experimental acceleration or deceleration dramatically increase or decrease reprogramming efficiency, respectively, in hematopoietic progenitors as well as in mouse embryonic fibroblasts. Having recognized the unique nature of these naturally fast cycling hematopoietic progenitors, we attempt to address the molecular underpinning of this unique somatic cell state and its implication in additional cell fate transitions.

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Genomic sequencing from patients can implicate large numbers of genes and mutations as potential risk factors for diseases. However, the lack of a high throughput animal model system to validate the relevance of gene functions to tissue or organ pathology in vivo hinders progress in disease genetics, identification of therapeutic targets, and precision medicine-based approaches. We developed a Drosophila-based gene functional validation system to rapidly evaluate hundreds of candidate disease genes identified from genomic sequencing of Congenital Heart Disease (CHD) patients. Among the 223 candidate disease genes identified by bioinformatics analysis, 78% have a clear fly homolog and were analyzed for in vivo function in the heart in our study. We developed a series of quantifiable cardiac phenotypic readout to demonstrate the essential roles of more than 70 genes in the fly heart – many of them are involved in histone modification. We showed that one of these genes (Jmjd2a), when knocked out in mice, led to a striking cardiac septal defect, similar to the symptom of the corresponding patient. Therefore, our study identified ancestral roles of histone-modifying genes in heart development and disease, through functional genomics analysis in flies and specific gene validation in mice. More importantly, we present the first high throughput in vivo validation system to rapidly evaluate hundreds of candidate disease genes identified from patient genomic sequencing, which could facilitate precision medicine approach for a broad range of diseases.

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Cell death triggered by pathogens is a crucial component of mammalian host-defense system. Necroptosis has been identified as a regulated form of necrosis that can be invoked in the absence of caspase activity. Receptor-interacting kinase 3 (RIP3 or RIPK3) and its substrate MLKL are now recognized as the core cellular regulators of necroptosis. We show that human herpes simplex virus 1(HSV-1) triggers RIP3/MLKL-dependent necroptosis in mouse cells. HSV-1 protein ICP6 interacts with RIP3 and is sufficient to trigger necroptosis. Mice lacking RIP3 exhibit severely impaired control of HSV-1 replication and pathogenesis, supporting an essential role of necroptosis in host defense against HSV-1. Unexpectedly, HSV-1 is unable to induce programmed necrosis in human cells. Moreover, HSV-1 infection as well as ectopic expression of ICP6 in human cells

blocks tumor necrosis factor (TNF)-induced necroptosis by preventing the induction of a RIP1/RIP3 necrosome. Therefore, ICP6 has opposite impacts on RIP3 activation in mouse cells versus human cells. The molecular mechanism underlying species-specific modulations of necroptosis by ICP6 will be discussed as well.

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An RNA-directed DNA methylation (RdDM) pathway is responsible for establishment of DNA methylation in Arabidopsis. In this pathway, there are two plant-specific DNA-dependent RNA polymerases, Pol IV and Pol V. Single-stranded RNA produced by Pol IV is converted into double-stranded RNA by the RNA-dependent RNA polymerase RDR2 and is then cleaved into 24-nt small interfering RNA (siRNA) by the Dicer-like protein DCL3. Pol V is responsible for producing long noncoding RNA (lncRNA) that eventually facilitates recruitment of the methyltransferase DRM2 to RdDM target loci. However, it remains to be determined how Pol IV and Pol V are recruited to specific chromatin regions to mediate de novo DNA methylation. Our study revealed the mechanism underlying the recruitment of Pol IV and Pol V, indicating that repressive chromatin marks including histone H3K9 dimethylation and DNA methylation play important roles in recruitment of the polymerases to chromatin. The study suggests that generation of noncoding RNA including siRNA and lncRNA forms a feed-forward loop with repressive chromatin environment through the RdDM pathway. Additionally, we found that components of the RdDM pathway are involved not only in de novo DNA methylation but also in transcriptional silencing through an RdDM-independent pathway. The RdDM components SUVH9 and IDN2 associate with MORC6, thereby mediating transcriptional silencing through a mechanism that is different from the RdDM pathway.

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Breast cancer is one of the major causes of cancer-related deaths worldwide, mainly due to outgrowth of cancer cells in vital organs including the bone, lungs, liver and brain. The majority of patients with advanced breast cancer will develop bone metastases and suffer from severe pains and eventually deaths. Transforming growth factor-β (TGFβ) signaling plays a central role in breast-to-bone metastasis; however, little is known about how this pathway is regulated during cancer cell bone colonization. Recently, we identified deleted in liver cancer 1 (DLC1) as an important regulator of cancer cell TGFβ responses in the bone milieu. DLC1 suppresses bone metastasis by inactivating Rho GTPases, while Rho-ROCK signaling mediates SMAD3 linker region phosphorylation and TGFβ-induced expression of parathyroid hormone-like hormone (PTHLH), leading to osteoclast maturation for osteolytic colonization. Thus these findings define a stroma-dependent role of Rho signaling in cancer metastasis.

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"Skeletal muscle regeneration involves a series of physical responses after injury or disease, including activation of quiescent satellite cells (muscle stem cells), proliferation of satellite cells and myoblasts, differentiation of myoblasts, and formation of new myofibers. In recent years, more and more evidences suggested that inflammation plays important roles during muscle regeneration process. However, how inflammation affects muscle regeneration remains to be elusive.

Here we focused on T cells mediated inflammation and found that it is a required positive regulator at early stage of skeletal muscle regeneration. Upon muscle injury, we observed large amount of T cell infiltrated at injury site. In immunodeficient mice, where the T cell infiltration is diminished while other liphocytes such as macrophage infiltration remains normal, reparation of muscle injury was dramatically delayed. To further investigate the mechanism of T cell promoting muscle regeneration, we characterized the protein profile of activated T cells. A combination of four

factors was identified to be able to promote satellite cell proliferation and long term expansion dramatically in culture. The cultured expanded satellite cells continue to express muscle stem cell marker, and were able to regenerate functional myofiers in vivo. Furthermore, muscular injection of the four factor cocktail could rescue the muscle regeneration defects caused by T cell deficiency. Our results demonstrate that T cell mediated inflammation is required for muscle stem cell proliferation at early stage of post-injury regeneration."

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To systematically study protein function in a native cellular background, libraries of human cell lines expressing proteins tagged with a functional sequence at their endogenous loci would be very valuable. Here, using electroporation of Cas9/sgRNA ribonucleoproteins and taking advantage of a split-GFP system, we describe a scalable method for the robust, efficient and specific tagging of endogenous human genes with GFP. Our approach requires no molecular cloning and allows a large number of cell lines to be processed in parallel. We demonstrate the scalability of our method by targeting 48 human genes and show that the resulting GFP fluorescence correlates with protein expression levels. We next show that our GFP tagging approach also allows the biochemical isolation of native protein complexes for proteomic studies. Together, our results pave the way for the large-scale generation of endogenously tagged human cell lines for the proteome-wide analysis of protein localization and interaction networks in a native cellular context.

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Genetically modified (GM) animals represent a crucial tool for understanding gene function in development and disease. The type II bacterial CRISPR/Cas system has been demonstrated as an efficient gene-targeting technology to generate mice carrying mutations and reporter. However, GM animals generated by CRISPR/Cas9 system are mosaicism, in which two or more populations of cells with different genomes are present in an individual animal. It is costly and time consuming to produce single-gene knockout animal and even more so to make double-mutant animal through the germline transmission of chimeric animals, especially for non-human primate, due to its longer sexual maturity lengths (4 to 5years) as compared to rodents. Here we optimized CRISPR/Cas9 system and obtained fully functional knockout mice for one gene or multiple genes in one step. Using this optimized CRISPR/Cas9 system, we did high-throughput screen for genes located on mouse Y chromosome and risk genes for Alzheimer's disease. In future, we will aim at further optimizing CRISPR/Cas9 system to obtain fully functional knock out monkey at first generation.

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Recently, increasing evidence, including ours, suggested that dysfunction of RNA metabolism may play a key role in etiology of a range of neurodegenerative disorders, especially motor neuron disease. However, the basis of RNA metabolism in specific neuron types and their subcellular compartments during both physiological and pathological conditions is largely unknown. Currently, we focus on three RNA-binding proteins, IGHMBP2, TDP43, and FUS, and study how dysfunction of these genes results in motor neuron degeneration. In addition, we are interested to learn RNA species and their metabolism in specific cell type in the brain, including motor neurons, in different experimental paradigm and pathological conditions. To gain our understanding on these aspects, we combine neurobiology, molecular and cell biology, RNA biochemistry, high through-put sequencing, and mouse forward genetics.

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High-content, image-based screens enable the identification of compounds that induce cellular responses similar to those of known drugs but through different chemical structures or targets. A central challenge in designing phenotypic screens is choosing suitable imaging

biomarkers. Here we present a method for systematically identifying optimal reporter cell lines for annotating compound libraries (ORACLs), whose phenotypic profiles most accurately classify a training set of known drugs. We generate a library of fluorescently tagged reporter cell lines, and let analytical criteria determine which among them--the ORACL--best classifies compounds into multiple, diverse drug classes. We demonstrate that an ORACL can functionally annotate large compound libraries across diverse drug classes in a single-pass screen and confirm high prediction accuracy by means of orthogonal, secondary validation assays. Our approach will increase the efficiency, scale and accuracy of phenotypic screens by maximizing their discriminatory power.

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Regulation of enhancer activity is important for controlling gene expression programs. Here we report that a biochemical complex that contains a potential chromatin reader, RACK7 and the histone lysine 4 tri-methyl (H3K4me3)-specific demethylase KDM5C occupies many active enhancers, including almost all super-enhancers. Loss of RACK7 or KDM5C results in overactivation of enhancers, characterized by the deposition of H3K4me3 and H3K27Ac, together with increased transcription of eRNAs and nearby genes. Furthermore, loss of RACK7 or KDM5C leads to de-repression of S100A oncogenes and various cancer-related phenotypes. Our findings reveal a RACK7/KDM5C-regulated, dynamic interchange between histone H3K4me1 and H3K4me3 at active enhancers, representing an additional layer of regulation of enhancer activity. We propose that RACK7/KDM5C functions as an enhancer “brake” to ensure appropriate enhancer activity, which, when compromised, could contribute to tumorigenesis.

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Aneuploidy and chromosomal instability are major characteristics of human cancer. These abnormalities can result from defects in the spindle assembly checkpoint (SAC), which is a surveillance mechanism for accurate chromosome segregation through restraint of the activity of the anaphase-promoting complex/cyclosome (APC/C). This multi-subunit E3 ubiquitin ligase is a major regulator of protein degradation during mitosis. APC/C-Cdc20 activation causes polyubiquitination and degradation of securin and cyclin B1, and is critical for the anaphase onset.SAC is activated by improperly attached kinetochores, and then inhibits the activation of APC/C-Cdc20. Here, we show that a CUE-domain-containing protein, CUEDC2, is a cell-cycle regulator that promotes spindle checkpoint inactivation and releases APC/C from checkpoint inhibition. CUEDC2 is phosphorylated by Cdk1 during mitosis. Depletion of CUEDC2 causes a checkpoint-dependent delay of the metaphase–anaphase transition. Phosphorylated CUEDC2 binds to Cdc20, an activator of APC/C, and promotes the release of Mad2 from APC/C-Cdc20 and subsequent APC/C activation. CUEDC2 overexpression causes earlier activation of APC/C, leading to chromosome missegregation and aneuploidy. Interestingly, CUEDC2 is highly expressed in many types of tumours. These results suggest that CUEDC2 is a key regulator of mitosis progression, and that CUEDC2 dysregulation might contribute to tumour development by causing chromosomal instability。

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Biogeography refers to the study of spatiotemporal distribution of biological units across space and time. Traditionally, this discipline focuses on the pattern of plant or animal species in a defined habitat. More recently, DNA sequencing technology dramatically increased our ability to survey large numbers of evolving genomes at all scales. My own research has witnessed this data-driven transformation in at least three areas. One, in human population genetics, we now rely on ancient DNA and large-sample sequencing of extant groups to infer finer details of historical demography around major continents. Two, in cancer genomics, we perform multi-omic, multi-region analysis of somatic alterations to infer past evolutionary events underlying oncogenesis and recurrence after treatment. Three, in microbiome and infectious disease research, DNA-based

assays have superseded culture-based assays in documenting the dynamics of human-pathogen interaction at individual, cohort and global levels. These parallel lines of inquiry share similar concepts rooted in classic models of Darwinian evolution, and they also share common algorithms developed in population genetics to understand the interplay of mutation, genetic drift, selection, admixture, and migration. I will discuss recent applications, new opportunities, and emerging challenges in analyzing increasingly complex biogeography data. Possible topics include classification and prediction, and phylogeny estimation in single-cell/person or population pools.

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Eukaryotic DNA is hierarchically packaged into chromatin to fit inside the nucleus, in which the accessibility of DNA is dependent on the packing density of chromatin. Dynamic of chromatin structures plays a critical role in transcriptional regulation and all other DNA related biological processes. Previously, we reported the 11 Å resolution cryo-electron microscopy (cryo-EM) structures of 30 nm chromatin fibers reconstituted in the presence of linker histone H1, which reveals a left-handed double helix twisted by the repeating tetra-nucleosomal structural units. However, the dynamic organization of chromatin fibers and its regulation mechanisms remain poorly understood. Using single-molecule force spectroscopy, we reveal that the tetranucleosomes-on-a-string appears as a stable secondary structure during hierarchical organization of chromatin fibers. The stability of the tetranucleosomal unit is negatively regulated by the histone chaperone FACT (Facilitates Chromatin Transcription) in vitro. We also show via genome-wide analysis that FACT facilitates gene transcription by destabilizing the tetranucleosomal unit of chromatin fibers in yeast. Our study demonstrates that the tetranucleosome is a novel regulatory structural unit of chromatin fibers beyond the nucleosome, and provides crucial mechanistic insights into the structure and dynamics of chromatin fibers during gene transcription.

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Hepatitis B virus (HBV) is a small DNA virus with unique life cycle. HBV covalently closed circular (cccDNA) is the reservoir for the chronic HBV infection. Understanding the molecular base of cccDNA biogenesis holds the key for breaking the viral persistence. We previously identified a liver specific bile acid transport (Sodium taurocholate co-transporting polypeptide, NTCP) as the long sought-after functional receptor for HBV, and the receptor complemented human hepatoblasma HepG2 cells (HepG2-NTCP) has enabled a much-needed culture system for studying HBV. Recently, by taking advantages of efficient genetic screen of the HepG2-NTCP HBV infection system and combined recombinant HBV virus along with chemical inhibition studies, we identified that cellular DNA polymerase κ (POLK), a Y-family DNA polymerase with high activity in non-dividing cells, is a key factor for HBV cccDNA formation. We also found that upon HBV de novo infection of HepG2-NTCP cells, cccDNA can be effectively established from the HBV rcDNA precursor regardless of the presence or absence of HBV capsid protein expression. These studies elucidated a critical aspect of HBV life cycle. They also provide important clues for further investigation of viral and cellular factors in regulation of cccDNA biosynthesis and may contribute to the development of novel therapeutics to cure infection of HBV, which affects 1.5-fold more population than those infected by HIV and HCV combined worldwide.

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How epigenetic marks are inherited through the cell cycle remains poorly understood. Histone H3 lysine 9 methylation and histone hypoacetylation are conserved hallmarks of heterochromatin. We previously showed that the inheritance of H3K9me during DNA replication depends on the catalytic subunit of DNA Polymerase epsilon, Cdc20. Here we show that two histone-fold subunits of Pol epsilon, Daf1, and Dpb4, form a heterodimer that plays a crucial role in the inheritance of histone hypoacetylation. Our findings reveal a link between histone deacetylation

and H3K9 methylation, and suggest a mechanism for how two processes are coordinated during replication. We resolved the crystal structure of Daf1-Dpb4 that provides further insights into the role of the dimer in chromatin inheritance. Remarkably, we found that Dpb4 interacts with both silencing and anti-silencing factors. Our findings suggest that the Daf1-Dpb4 heterodimer serves as a platform for the recruitment of chromatin modifiers and remodellers that mediate the disassembly and re-assembly of heterochromatin, and ensure the faithful inheritance of epigenetic marks in heterochromatin.

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Nucleosome assembly is coupled with DNA replication, a process crucial for epigenetic inheritance and maintenance of genome integrity. However, the mechanism of coupling these two processes remains poorly understood. Here we show that replication protein A (RPA), a single-stranded DNA (ssDNA) binding protein that is essential for DNA replication, is required for the deposition of H3-H4 to replication forks and nucleosome formation on nascent chromatin. In vitro, RPA binds free histone H3-H4 but not nucleosomal histones, and a RPA coated ssDNA stimulates formation of DNA-H3-H4 complex. RPA mutants that cannot bind H3-H4 exhibit synthetic genetic interaction with mutations at key factors involved in replication-coupled nucleosome assembly, and are defective in assembly of replicating DNA into nucleosomes in cells. Taken together, our results reveal that RPA is a novel factor that couples nucleosome assembly with DNA replication.

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Stem cells are a special class of cells that occur naturally in the body but have amazing qualities that set them apart from other cells. Stem cells have a special capacity for repair and regeneration, coupled with their longevity, which makes stem cells potentially useful in medical applications. We are interested in hematopoietic and intestinal stem cells and focus on understanding how stem cells are maintained in vivo by cellular microenvironments (niches), signaling, and epigenetic regulations. On the other hand, when mutations accumulated in stem and progenitor cells, it can develop into tumors or cancers. Recently a new concept of cancer stem cells emerged to explain a role of CSC in tumorigenesis. We are testing a new strategy to target CSC in combination of conventional chemotherapy to treat leukemia.

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Vaccines are the most cost-effective measure to control infectious diseases. Many successful vaccines are based on live-attenuated pathogens. Live viral vectors usually induce strong and long-lasting vaccine immunity. However, their induction of a robust anti-vector immunity prevents repeated applications of the same vector. Pichinde virus (PICV) is a non-pathogenic Arenavirus, whose genome consists of two RNA segments (L and S) each encoding two viral genes in opposite orientation. By splitting the S segment into two RNAs (S1 and S2) that each can encode a foreign gene, we have developed a system to generate replication-competent tri-segmented PICVs (triPICV), which can be used to deliver up to two foreign antigens. Using influenza virus genes as model antigens, we have shown that this novel viral vaccine vector can be delivered to different animal hosts and through various routes of immunization, which can effectively generate antigen-specific humoral and cellular immune responses. Interestingly, the immune responses are significantly increased upon a booster dose and remain at high levels even after repeated boosting with the same viral vector. The unique features of this novel viral vector can be exploited for use as a vaccine platform against various human and animal diseases, particularly when strong cellular immunity and prime-boost vaccination strategy are desired, such as HIV, HCV, malaria, tuberculosis, and cancers."

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Blockage of blood vessels can lead to ischemia and tissue damage. One method to restore blood flow into ischemic tissues is to perform endothelial cell transplantation. To date, the most successful vascular repair for coronary or peripheral vascular damages are the use of patients' own blood vessels. However, such vessels are not easily available because they are either diseased or have already been harvested for previous surgery. Use of non-isogenic source of vascular endothelial cells has the problems of biocompatibility and growth deficiency. Ets variant 2 (ETV2) is transiently expressed in both zebrafish and mice and is necessary and sufficient for vascular endothelial cell specification. By controlled expression of this gene we have developed an efficient approach to produce functional endothelial cells from human embryonic stem cells or induced pluripotent stem cells as well as differentiated somatic cells. Thus it is now possible to generate patient-specific endothelial cells for therapeutics of ischemic diseases.

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Acute myeloid leukemia (AML) is thought to arise from leukemia-initiating cells (LICs); however, recent evidence suggest that the transforming events may initially give rise to pre-leukemic hematopoietic stem cells (HSCs), preceding the formation of fully transformed LICs. Pre-leukemic HSCs have been shown to contribute to normal blood development and harbor a selective growth advantage compared to normal HSCs. Recently, acquired somatic gain-of-function (GOF) p53 mutations were identified in the blood of aged healthy individuals as well as AML patients, suggesting that p53 mutations may be early events in the pathogenesis of AML. We found that some GOF mutant p53 proteins, including p53R248W, p53Y220C, and p53R273H, enhanced the self-renewal potential of normal HSCs without affecting terminal differentiation. Further, we discovered that HSCs expressing mutant p53 show enhanced clonal expansion following genotoxic stress. These findings demonstrate that GOF mutant p53 drives the development of pre-leukemic HSCs. Although p53 mutations are limited in AML, p53 mutations do co-exist with mutations of receptor tyrosine kinases and epigenetic regulators, including FLT3-ITD and ASXL1, in AML. To determine the synergy between mutant p53 and FLT3-ITD or ASXL1 mutations in the pathogenesis of AML, we have generated p53R248W/+Flt3ITD/+ and p53R248W/+Asxl1+/- mice. We found that the expression of mutant p53 rescued the self-renewal defect of Flt3ITD/+ and Asxl-1+/- HSCs, suggesting that mutant p53 may cooperate with FLT3-ITD or ASXL1 deficiency in the formation of leukemia-initiating cells. To investigate how GOF p53 regulates HSC self-renewal, we performed transcript profiling and ChIP-seq assays. We found that mutant p53 regulates the expression of several epigenetic regulators, including EZH1, EZH2, and SETD2, in HSCs. In addition, we found that there were increased levels of H3K27me3 and decreased levels of H3K36me3 in p53R248W/+ HSCs compared to that of the p53+/+ HSCs. In ChIP-seq assays, we found that Asxl1+/- HSPCs exhibited significantly low levels of H3K27me3 at promoters, and these defects were rescued in the mutant p53 background. Thus, we demonstrated that gain-of-function mutant p53 drives the development of pre-leukemic HSCs by a novel mechanism involving disruption of the epigenetic pathways.

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Long noncoding RNAs (lncRNAs) have emerged as regulators of gene expression across metazoa. Interestingly, some lncRNAs function independently of their transcripts - the transcription of the lncRNA locus itself affects target genes. However, current methods of loss-of-function analysis are insufficient to address the role of lncRNA transcription from the transcript which has impeded analysis of their function. Using the minimal CRISPR interference (CRISPRi) system, we show that coexpression of the catalytically inactive Cas9 (dCas9) and guide RNAs targeting the endogenous roX locus in the Drosophila cells results in a robust and specific knockdown of roX1 and roX2 RNAs, thus eliminating the need for recruiting chromatin modifying proteins for effective gene silencing. Additionally, we find that the human and Drosophila codon optimized dCas9 genes

are functional and show similar transcription repressive activity. Finally, we demonstrate that the minimal CRISPRi system suppresses roX transcription efficiently in vivo resulting in loss-of-function phenotype, thus validating the method for the first time in a multicelluar organism. Our analysis expands the genetic toolkit available for interrogating lncRNA function in situ and is adaptable for targeting multiple genes across model organisms.

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Mouse contains three PIWI homologs, MIWI, MILI and MIWI2, all are highly expressed in testes and required for male fertility. Spermatogenesis in mice is characterized by two waves of piRNA expression: one corresponds to classic piRNAs responsible for silencing retrotransponsons and the second wave is predominantly derived from non-transposon intergenic regions in pachytene spermatocytes, but the function of these pachytene piRNAs is largely unknown. Our recent findings show that a piRNA-induced silencing complex (pi-RISC) containing pachytene piRNAs, MIWI and deadenylase CAF1 is selectively assembled in elongating spermatids, which is responsible for inducing mRNA deadenylation and decay via a mechanism that resembles the action of miRNAs in somatic cells. Such a highly orchestrated program appears to take full advantage of the enormous repertoire of diversified targeting capacity of pachytene piRNAs, and is responsible for massive mRNA elimination at the late stages of spermiogenesis. In addition, we recently explored the metabolism of PIWI/piRNA machinery in mouse. We show that MIWI is degraded through the APC/C-26S proteasome pathway, and that piRNAs play an indispensable role in this process by enhancing MIWI interaction with the APC/C substrate-binding subunit. Interestingly, piRNA-triggered MIWI destruction occurs only in late spermatids, which in turn leads to piRNA elimination, suggesting a feed-forward mechanism for coordinated removal of the MIWI/piRNA machinery at a specific developmental stage. Most recently, by screening and sequencing human Piwi (Hiwi) in patients with azoospermia, we identified multiple germline mutations in the D-box element of Hiwi that prevent HIWI ubiquitination and degradation. We establish the causative role of such mutations in male infertility by knocking them into mouse Miwi. Strikingly, we discover that MIWI ubiquitination is coupled with histone ubiquitination, and defects in MIWI ubiquitination impair histone-to-protamine exchange during spermiogenesis. These findings provide direct evidence for Piwi as a vital gene for human male infertility gene, and reveal an unprecedented mechanism for regulated packaging of genomic DNA into functional sperm. Collectively, our studies, along with the findings by other labs, indicate that the function of PIWI/piRNA machinery is highly regulated during mammalian spermatogenesis.

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We recently reported that the T cell immunoglobulin and mucin domain (TIM) proteins inhibit release of HIV-1 and other enveloped viruses by interacting with virion- and cell-associated phosphatidylserine (PS) (Li et al, PNAS 111, 2014). In this study, we demonstrate that the Nef proteins of HIV-1 and other lentiviruses antagonize TIM-mediated restriction. We show that TIM-1 exhibits stronger inhibition of the release of Nef-deficient relative to Nef-expressing HIV-1 particles and that ectopic expression of Nef relieves this restriction. Consistent with this finding, knockdown of endogenous TIM-3 in human PBMCs effectively enhances the production of Nef-deficient HIV-1 particles. HIV-1 Nef does not appear to downregulate TIM-1 expression on the cell surface, nor does it disrupt TIM-1 incorporation into HIV-1 virions. Interestingly, we observed that coexpression of SERINC3 and SERINC5 potentiates TIM-1 inhibition of HIV-1 release, and that depletion of SERINC proteins in viral-producer cells rescues TIM-mediated inhibition of HIV-1 release. These results suggest that SERINCs are involved in TIM-mediated restriction of HIV-1 release. In addition to HIV-1 Nef, the Nef proteins of simian immunodeficiency virus (SIV) strains and HIV-2 also antagonize the antiviral activity of TIM-1, suggesting an evolutionarily conserved role of the lentiviral nef gene in antagonizing TIMs. Collectively, our work reveals a new role for lentiviral Nef in

antagonizing TIM, and highlights a complex interplay between lentiviral Nef and cellular restriction by TIMs and SERINCs.

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Malignant gliomas exhibit extensive heterogeneity and poor prognosis, which represents a significant hurdle for effective therapy. Here we identify mitotic Olig2+ cells as glioma-propagating cells in gliomas, elimination of which blocks tumor initiation and progression. Intriguingly, deletion of Olig2 resulted in tumors that grow, albeit at a decelerated rate. Genome occupancy and expression profiling analyses reveal that Olig2 directly activates cell proliferation machinery to promote tumorigenesis. Olig2 deletion causes a tumor phenotypic shift from an oligodendrogenesis-correlated proneural toward an astrocyte-associated gene expression pattern, manifest in down-regulation of PDGF receptor-alpha and reciprocal up-regulation of EGF receptor signaling. Olig2 deletion further sensitizes glioma cells to EGF receptor inhibitors and extends animal lifespans. Thus, Olig2-orchestrated receptor signaling drives mitotic growth and regulates glioma phenotypic plasticity. Targeting Olig2 may circumvent resistance to EGFR-targeted drugs.

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Somatic mutations that endow hematopoietic stem cells with competitive advantage accumulate during aging and are prevalent in human population. While these mutations underlie increased incidences of hematopoietic malignancies, they are also inherited in immune cells that derive from hematopoietic stem cells, thus leading to potential impact on immune response toward pathogens. We investigated the role of Ten Eleven Translocation 2 which is frequently mutated in loss-of-function patterns, and found aberrant response toward bacterial infection in a mouse model. This study supports “genetic reprogramming” of immune response by hematopoietic stem cell mutations.

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Pathogens have evolved various strategies to hijack the host ubiquitin network, which play essential roles in immunity. The bacterial pathogen Legionella pneumophila codes for numerous virulence factors (effectors) to subvert host function for its replication in phagocytes. A number of these effectors are predicted F-box or U-box proteins that together with components from the host, function as ubiquitin E3 ligases. Our investigation revealed that members of the SidE family of effectors hijack host processes by ubiquitinating several Rab small GTPases associated with the endoplasmic reticulum. Moreover, we found that these effectors catalyze the ubiquitination without the need for the E1, E2 enzymes and ATP. In this presentation, I will discuss our recent results in the mechanism of the E1/E2-independent ubiquitination and its regulation.

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Several pathogenic arenaviruses, including Lassa virus (LASV), cause hemorrhagic fever (HF) infections that can result in significant morbidity and mortality in humans with limited preventative and treatment options. A hallmark of severe HF is the high level of viremia coupled with generalized immune suppression of the hosts, the mechanisms of which are unknown. Recent studies in our laboratory using viral reverse genetics, cell/virus-based assays, structural/biochemical analysis, and animal modeling have revealed two specific molecular mechanisms arenaviruses use to evade host innate immune recognition that involve the viral nucleoprotein (NP) and matrix Z protein (reviewed in Meyer and Ly, 2016, J Virol). Our structurally directed functional studies of arenaviral NP proteins have demonstrated that they exhibit 3′–5′ exoribonuclease activity that is required for suppressing IFN-beta induction by degrading immune stimulatory RNAs (Qi, et al., 2010, Nature; Jiang, et al., 2013, JBC; and Huang, et al., 2015, J Virol.), whereas the Z proteins of all known pathogenic arenaviruses inhibit IFN-beta production by directly interacting with the CARD

domains of the pathogen-recognition proteins RIGi and MDA5 to inhibit their normal functions (Xing, et al., 2015a, 2015b, J Virol.). Novel insights learned from these studies of ours as well as those of other researchers can be exploited for the development of novel therapeutics and vaccines against deadly HF-causing viral infections (reviewed in McLay, et al., Antiviral Research, 2012, J Gen. Virol., 2013, and Shao, 2015, Pathogens)."

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We study the molecular and cellular mechanisms underlying Q neuroblast migration in C. elegans. We have developed live cell imaging techniques to document Q cell migration and somatic CRISPR-Cas9 technique to generate conditional mutations in Q cells. In a combination with imaging, genetic and biochemical approaches, we have identified a conserved transmembrane protein MIG-13 as a key regulator that functions autonomously during Q cell migration. We uncover two parallel pathways that respectively transduce the MIG-13 signal through the WASP and WAVE complexes to promote the nucleation of the actin cytoskeleton in the leading edge. Here I report our recent progresses in Q cell migration. Considering that these components have vertebrate homologs, our results can provide insights to mammalian neural development.

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Severe loss of photoreceptor cells in inherited or acquired retinal degenerative diseases, such as retinitis pigmentosa and age-related macular degeneration, can result in partial or complete blindness. Research in my lab is focused on the development of optogenetic approaches to restoring vision by expressing microbial rhodopsins to convert second- or third-order retinal neurons into photosensitive cells, thus imparting light sensitivity to the retina lacking photoreceptor cells. Proof-of-concept studies have demonstrated restoration of light responses in surviving retinal neurons and visually guided behaviors in animal models. The first Investigational New Drug (IND) application of the optogenetic gene therapy has been recently approved by US FDA for clinical trials. In this talk, I will review the current status of the field and outline our ongoing efforts to further improve the optogenetic gene therapy technology.

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"My research goal is to develop mass spectrometry-based proteomics and systems biology approaches to address biomedical challenges. We have developed a quantitative proteomics pipeline, capable of analyzing >10,000 proteins and dissecting tens of thousands of posttranslational modifications from relevant cellular and animal models as well as human clinical specimens. Integration of such large-scale omics data offers a holistic view for unbiased identification of central disease gene/protein networks. To understand the pathogenesis of Alzheimer disease (AD), the most common form of dementia, we profiled aggregated proteome in all common neurodegenerative diseases, including AD, mild cognitive impairment, Parkinson disease, Lewy body dementia, Ub-positive frontotemporal lobar degeneration, amyotrophic lateral sclerosis, and corticobasal degeneration. This comprehensive study identified the accumulation of U1-70K and other U1 snRNP spliceosome components in AD. Multiple U1 snRNP subunits form cytoplasmic tangle-like structures in AD but not in other diseases. RNAseq analysis reveals dysregulated RNA processing with accumulation of unspliced RNA species in AD. Thus, our results demonstrate unique U1 snRNP pathology and suggest abnormal RNA splicing in AD. Furthermore, we have generated a brain-specific transgenic mouse model for deregulating the U1-70K activity. The transgenic mice recapitulate many AD features, supporting the role of RNA dysregulation in Alzheimer's disease."

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Methyl-CpG binding protein 2 (MeCP2) primarily binds to methylated DNA and plays a critical role in transcriptional regulation. Mutations in mecp2 gene are found in 90% of patients with Rett syndrome, a severe form of developmental disorders. Duplications of mecp2–containing loci also cause the MeCP2 duplication syndrome that shares symptoms with autism spectrum disorders (ASD). Although the mecp2-null mice recapitulate most developmental and behavioral defects found in Rett syndrome patients, it has been difficult to identify autism-like symptoms in mouse model with MeCP2 overexpression. Here we report that lentivirus-based transgenic cynomolgus monkey (Macaca fascicularis) with MeCP2 overexpression in the central nervous system exhibited some behaviors and metabolic changes mimicking those of ASD. As compared to wild-type (WT) monkeys, the MeCP2 transgenic (TG) monkeys exhibited higher frequency of repetitive circular locomotion in the home cage, and impaired social interaction, as indicated by the time spent with WT monkeys within the same group (reared together for over six months) or with unfamiliar TG monkeys from a different group. The TG monkey also showed elevated stress responses, as shown by the number of defensive grunt induced by the human gaze during the threat-related anxiety and defensive (TAD) test for monkeys. The general health of TG monkeys was largely normal, but the level of their blood fatty acids was significantly higher than that of WT monkeys. Tests using the Wisconsin General Test Apparatus (WGTA) revealed largely normal cognitive functions in TG monkeys, although some of them showed signs of stereotypic cognitive behavior. These results suggest the feasibility of using non-human primates for studying neural mechanisms underlying ASD and related brain disorders and for developing therapeutic treatments.

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The genome of each person differs from one another at millions of nucleotides, and these sequence variants together are responsible for the spectrum of phenotypic traits and disease risks of that individual. While it has become commonplace these days to sequence one's genome, predicting the specific phenotypic traits of each individual from DNA still seems an insurmountable challenge. This is because over 98% of the human genome is non-protein-coding and generally without a clearly defined biological function. In particular, scattered in these noncoding sequences are millions of putative cis-regulatory elements responsible for spatiotemporal gene expression during development. Furthermore, a large number of sequence variants in the cisregulatory elements are believed to confer risks to various common human diseases. Therefore, identifying and characterizing the cis-regulatory elements in the human genome have the potential to significantly enhance our ability to link DNA variations to phenotypic traits. Thanks to the development of high-throughput technologies, hundreds of thousands of candidate enhancers have been annotated in the human genome, compromising at least 12% of the total DNA sequences. Recent experiments have suggested a role for the three-dimensional chromatin architecture in regulation of gene expression by distal cis elements. The presentation will discuss ongoing research in this area and future challenges in the context of precision medicine initiative.

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Noncoding RNAs, mobile elements, and alternative splicing are engaged in the evolution of life. Here we present a paradigm in which a relatively conserved noncoding RNA acquires novel function due to the insertion of a mobile element to regulate alternative splicing in human. The noncoding RNA we termed 5S-OT is transcribed from the 5S rDNA loci in eukaryotes from fission yeast to mammals. 5S-OT plays a cis role in regulating the transcription of 5S rRNA in both mice and human. In the anthropoidea suborder of primates, an insertion of antisense Alu element happens to 5S-OT. When examined in human cells, 5S-OT regulates alternative splicing of multiple genes in trans by Alu: anti-Alu pairing with target genes and by interacting with the splicing factor

U2AF65. 5S-OT may be involved in physiological events such as cell differentiation in human, and the trans effect of 5S-OT in splicing could be harnessed as a biotechnology.

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Much of the developmental complexity of higher eukaryotes is thought to arise from gene regulation rather than from an increase in the number of protein-coding genes. RNA may represent a hidden layer of regulatory information in complex organisms. Genome-wide transcriptome analyses have identified thousands of long noncoding RNAs (lncRNAs). It has been proposed that lncRNAs may serve as versatile regulators of diverse aspects of biology. However, the functionality of vast majorities of lncRNAs is unknown. Identifying functional lncRNAs and then inferring biological pathways in which they act in represent major challenges in understanding genome complexity and RNA-mediated gene regulation. I will discuss recent progresses we have made in lncRNA-mediated regulations of gene expression.

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Mutations of caretaker genes often lead to genomic instability that drives tumorigenesis by causing secondary genomic alternations on gatekeeper tumor suppressors or driver oncogenes. However, a substantial number of the caretakers are also indispensible for cell proliferation during certain stage of the tumor development, and their complete inactivation by mutations may also hinder tumor development. There is a little understanding on how dysfunctions of these essential caretakers uniquely contribute to tumorigenesis. In this presentation, I will use BCCIP gene as a platform to demonstrate how the cancer overcome the growth barrier posed by defective caretakers.

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"Although a large part of the genome is actively transcribed into RNA, a small portion can encode a protein, and most non-coding RNA is transcribed into different cell function (ncRNA). Small non-coding RNA (or small molecule RNA) is necessary for the regulation of gene expression in many organisms. Different small RNA have different ways of generating and function, but they all need bind with Ago proteins together to work. Ago proteins are RNA-induced silencing complex (RISC) core components, and it is necessary of the RNA interference (RNAi) for its participation in the targeted mRNA fracture, translational repression or chromatin modification, thus affecting gene silencing. Research work is mainly focused on the structural and biology studies of gene silencing mediated by Ago proteins. In the gene silencing complex (RISC) which consisted by RNA-Ago protein complex, small RNA molecules by complementary base pairing rules in a sequence-specific manner to guide Ago proteins bind to a target molecule mRNA. These target mRNA molecules will be cut or translation inhibition after being Ago proteins identification, and the cells will be degraded eventually. Our group made a research about the DNA enzyme activity of Ago proteins and the cleaving mechanism difference between the target RNA and DNA. We found that not only RNA interference exists in prokaryotes, there is also DNA-mediated DNA silencing phenomenon, and it clarified the cleavage mechanism guided by Ago protein from the bacteria to make the guiding DNA duplexes cleavage the target DNA duplexes. Ago is a key protein in the RNAi pathway of eukaryotic cells, and many prokaryotes also have genes encoding Ago, but their physiological role was not clear yet. Researches showed that prokaryotes can protect cells from DNA invasion."

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"Regarding the infection by Ebola virus, the virus entry process includes two key steps, virus attachment on the cell membrane and virus membrane fusion in the endosome. Following binding to the cell surface, Ebola viruses are internalized by a macropinocytosis-like process and

subsequently trafficked through early and late endosomes. The trimeric glycoprotein (GP) spike on the envelope of Ebola viruses mediates all stages of virus entry, including membrane fusion, and can be cleaved into GP1 and GP2 subunits by furin enzyme. In the late endosomes, GP needs to be primed to remove some “cap” components, thereby triggering the induction of the crucial membrane fusion event which leads to viral penetration. The functional EBOV GP priming is mediated by the cysteine proteases cathepsin B and cathepsin L, resulting in a primed form of GP (GPcl), which could bind to an indispensable endosomal membrane protein Niemann-Pick C1 (NPC1).

Here, for the first time, we have determined the crystal structures of free NPC1-C and its complex with GPcl. The results revealed that NPC1-C displays a helical structure core surrounded by several β-strands, and contains two extended loops protruding outwards for ligand interactions. Further GPcl/NPC1-C complex structure indeed shows that NPC1-C utilizes the two protruding loops to engage a hydrophobic cavity on the head of GPcl. Despite a low affinity between NPC1-C and GPcl as revealed by the biophysical analyses, upon enzymatic cleavage and NPC1-C binding, several conformational changes are shown to be induced in GPcl, including the uplift of the short 310 helix in the β3-α1 loop likely helps to release the N-terminal portion of the internal fusion loop (IFL), thereby triggering the membrane fusion. The mutagenesis work in NPC1-C further confirmed that the protruding loop 2 plays a more important role in the binding of NPC1-C to GPcl.

In conclusion, our results presented in this study for the structural basis of primed EBOV GP bound to its endosome-residing receptor NPC1 expands our understanding in the fusion trigger mechanism of enveloped viruses, and provides valuable information for the design of therapeutics against infections by Ebola viruses."

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Epithelia are formed by a well organized 3-dimentional-mosaic of different cell types and work as a barrier to separate the body from either the outside world or the lumen and cavities of organs. A complex intercellular architecture in specific epithelium allows its unique set of different cell types to operate in a concerted manner for the biological function of an organ. Proper cellular communication is essential to establish the congenial environment for epithelial health. Defects in the epithelial cell function are associated with a broad range of diseases, from minor disorders such as dry mouth to severe diseases including asthma, cancer and infertility. One organ that lines with a layer of epithelium is the epididymis in the male reproductive tract, which connects testis and vas deferens and is essential for sperm maturation and protection. It establishes a unique luminal environment, for instance, its fluid is acidic and low in calcium concentration, which also declines along the length of the epididymal tubule. Our previous studies have shown that the fluid secretion function of principal cells can be regulated by basal cells by releasing the COX-dependent PGE2 signaling pathway, and the proton-pumping function of clear cells by basal cells by the Angiotensin II type-2 receptor-NO-cGMP signaling pathway. Recently, in an attempt to better understand the calcium regulation in the epididymis, we used whole-cell patch-clamp electrophysiological method and pharmacological tools to characterize rat cauda epididymal principal cells. Our results revealed a TRPV6-mediated calcium conductance and TMEM16A-mediated calcium-activated chloride conductance in these cells. We also discovered the mRNA for TRPV6 and TMEM16A in the rat epididymis and showed that their proteins co-localized in the apical membrane of principal cells. These results provide evidence for a coupling mechanism between TRPV6 and TMEM16A in principal cells, which may play an important role in regulation of calcium homeostasis in the epididymis. Taken together, our studies demonstrate an intriguing cellular communication network underlying the barrier function of epididymal epithelium for sperm health and male reproduction.

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FBXW7 is a haploinsufficient tumor suppressor with loss-of-function mutations occurring in human cancers. FBXW7 inactivation causes genomic instability, yet the mechanism remains elusive.

Here we show that FBXW7 facilitates non-homologous end-joining (NHEJ) repair and FBXW7 depletion causes radiosensitization. In response to ionizing radiation, ATM phosphorylates FBXW7 at serine 26 to recruit it to DNA double-strand break (DSB) sites, while activated DNA-PKcs phosphorylates XRCC4 at serines 325/326 which promotes binding of XRCC4 to FBXW7. SCFFBXW7 E3 ligase then promotes polyubiquitylation of XRCC4 at lysine 296 via K63-linkage for enhanced association with the Ku70/80 complex to facilitate NHEJ repair. Consistent with these findings, a small molecule inhibitor that abrogates XRCC4 polyubiquitylation reduces NHEJ repair. Our study demonstrates one mechanism by which FBXW7 contributes to genome integrity and implies that inactivated FBXW7 in human cancers could be a strategy for increasing efficacy of radiotherapy.

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In an average human at resting, approximately 5-8 litters of air passes in and out of the lung. The air could carry allergen, pollutants, vary in oxygen level and mechanical pressure. How the information is sensed, processed and translated into distinct responses is not understood. In our recent study of the genetic mechanism of lung-associated birth defects, we have revealed new mechanisms of how the lung functions not only as a gas-exchange machine, but also a sensory organ.

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The recent discovery of metabolic roles for fibroblast growth factor 1 (FGF1) in glucose homeostasis has added a new facet to this classically known mitogen. To dissect the molecular basis for this functional pleiotropy of FGF1, we engineered an FGF1 partial agonist carrying triple mutations (FGF1△HS) that diminish the ability of FGF1 to induce heparan sulfate (HS)-assisted FGF receptor (FGFR) dimerization and activation. FGF1△HS exhibited a major loss in proliferative potential, while preserving the full metabolic activity of wild-type FGF1 in vitro and in vivo. Hence, submaximal FGFR activation by a weak FGF1-FGFR dimer is sufficient to evoke a metabolic response whereas full FGFR activation by stable and sustained dimerization is required to elicit a mitogenic response. In addition to providing a physical basis for the diverse activities of FGF1, our findings will also have major impacts on the ongoing drug discovery targeting FGF1 and related FGFs for the treatment of a variety of human diseases.

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"Calcium ion (Ca2+) is a highly versatile intracellular signal that controls many different cellular functions such as contraction, secretion, memory formation, gene transcription, cell growth and cell death. Maintaining homeostasis of Ca2+ stores in the endoplasmic reticulum (ER) is crucial for proper Ca2+ signaling and key cellular functions. The Ca2+- release-activated Ca2+ (CRAC) channel is responsible for Ca2+ influx and refilling after store depletion, but how cells cope with excess Ca2+ when ER stores are overloaded is unclear. We show that TMCO1 is an ER transmembrane protein that actively prevents Ca2+ stores from overfilling, acting as what we term a ""Ca2+ load-activated Ca2+ channel"" or ""CLAC"" channel. TMCO1 undergoes reversible homotetramerization in response to ER Ca2+ overloading and disassembly upon Ca2+ depletion and forms a Ca2+-selective ion channel on giant liposomes. TMCO1 knockout mice reproduce the main clinical features of human cerebrofaciothoracic (CFT) dysplasia spectrum, a developmental disorder linked to TMCO1 dysfunction, and exhibit severe mishandling of ER Ca2+ in cells. Our findings indicate that TMCO1 provides a protective mechanism to prevent overfilling of ER stores with Ca2+ ions. For more information, please see our recent paper in Cell journal: Wang QC et al. (2016) TMCO1 Is an ER Ca2+ Load-Activated Ca2+ Channel. Cell 165:1454-1466."

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Hematopoiesis is known to occur within a complex bone marrow microenvironment. Bone marrow stromal supportive cellular components including osteoblasts provide critical support for the survival and development of hematopoietic cells. The cells in the bone/marrow organ are located in a hypoxia microenvironment. Hypoxia inducible factor-alpha (HIF-α) is a key transcription factor that regulates gene programs involved in the adaptation and survival of cells in response to low oxygen tension. Our previous data indicate that HIF-α involves in the regulation of osteogenesis and hematopoiesis. However, the cellular and molecular mechanisms of HIF-α in bone in regulating hematopoiesis remain unclear. Here we show that mice with osteoblast conditional mutagenesis of the key components of the oxygen sensing pathway have altered osteogenesis, erythropoiesis or B lymphopoiesis. The phenotypes are accompanied by changes of the cellular or molecular niche components of the bone/marrow. Our results indicate that HIF-α in osteoblasts functions as an important mediator to control hematopoiesis.

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A mixed-mechanism dry eye (DE) model was created by excision of nictitating membrane, Harderian gland and main lacrimal gland (LG) in rabbits. DE phenotype developed after excision and was accompanied by elevation of inflammatory DE markers (MMP-9, IL-1β, and TNF-α). However, tear secretion as assessed by Schirmer test was never decreased in the absence of LG. In addition, DE phenotype improved in 3 months without intervention. Among many electrolyte and water transporters assessed, the expression of aquaporin 4 and 5 were found to increase in response to the development of DE phenotype. The expression of goblet cell-specific Muc5ac was also found to synchronize with that of aquaporins. Convincing laboratory, animal and human data was reviewed as supporting evidence that the main lacrimal gland is not indispensable for the maintenance of ocular surface tears. Spontaneous resolution of DE phenotype in this rabbit model further indicates that the conjunctiva potentially has an inherent defense machinery to combat external insults, at least in an acute setting.

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Bacteria obtain a memory of viral invaders by incorporating their DNA sequence elements into the host CRISPR locus, generating a new 33-nt spacer within the CRISPR array. We report on the crystal structure of a Cas1-Cas2-dual-forked DNA complex in efforts towards understanding how the protospacer is selected for insertion into the CRISPR locus. Our structure of the complex reveals a protospacer DNA containing a 23-bp duplex bracketed by tyrosine residues, together with anchored flanking 3'-overhang segments. The complementary PAM sequence in the 3'-overhangs are recognized by Cas1a catalytic subunits in a base-specific manner for protospacer selection and subsequent cleavage at positions 5-nts from the duplex boundary, thereby generating a 33-nt DNA intermediate for incorporation into the CRISPR array. Upon protospacer binding, the Cas1-Cas2 complex undergoes a significant conformational change, generating a flat surface conducive to proper protospacer recognition. Overall, our studies reveal unanticipated structure-based mechanistic insights into PAM-dependent spacer acquisition.

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"Cell-based therapy represents a highly promising approach for the treatment of heart diseases but its validation requires extensive preclinical studies. Mouse models have been most prevalent in such studies. However, many therapeutic strategies that showed promising results in mice have not been substantiated in human trials, raising concerns over whether mouse models are truly relevant in modeling and treating human diseases. Therefore, establishing reporter rabbit and pig models closely mimicking the physiology and pathogenesis of human heart disease will enable

us to derive invaluable preclinical insights. Other major challenges to cell-based therapy include the identification of ideal cell source and the extremely low retention and survival rates of transplanted cells. To address these critical challenges in heart regeneration, we have generated stable and high-level expression reporter swine and rabbit animals. For the first time in the field, we characterized and targeted the porcine ROSA26 (pROSA26) locus and generated ROSA26-EGFP swine reporters readily inducible by Cre expression (Li et al Cell Res 2014). These swine reporters will enable precise quantification of transplanted cells versus host cells. We also generated knock-in pigs containing Cre-T2A-tdTomato at endogenous ISL1 locus, which allow us to trace the specification and reactivation of ISL1+ CPCs in pig hearts. Meanwhile, we have established cutting-edge injectable cell microcarriers for tissue regeneration. In particular, we have developed new nanofibrous hollow microspheres (NF-HMS) that mimic the extracellular matrix architecture at the nanometer scale. Building on pioneering works in cardiac stem cell field, we are able to robustly generate embryonic cardiac progenitor cells (CPCs) from pluripotent stem cells for heart regeneration. Our results show that NF-HMS greatly enhance the CPC retention, survival, and integration in infarcted hearts of large animals. Our progress using these combined approaches in heart regeneration will be presented. Our integration of advanced cell source, biomimetic carrier, and large animal models for heart regeneration should provide general principles in developing an informative model system for regenerative medicine."

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"CRISPR-Cas9 system has become the tool of choice for genome engineering. Previously we established the method of microinjecting CRISPR-Cas9 system into zygotes to generate mouse models. To overcome the technically demanding and inherently low throughput method of microinjection, we devised the Zygote Electroporation of Nuclease (ZEN) technology, which employs electroporation to deliver CRISPR-Cas9 reagents to the zygotes and generated live mice carrying targeted NHEJ and HDR mutations with high efficiency. The general principles discovered and described in this study have implications for high efficiency, high throughput genome engineering in animals.

To extend the utility of the CRISPR-Cas9 system, we have taken advantage of the ability of Pumilio PUF domains to bind specific 8-mer RNA sequences. By combining these two systems, we established the Casilio system, which allows for specific and independent delivery of effector proteins to specific genomic loci. We demonstrated that the Casilio system enables independent up- and down-regulation of multiple genes, as well as live-cell imaging of multiple genomic loci simultaneously. Importantly, multiple copy of PUF binding sites can be incorporated on sgRNA backbone, therefore allowing for local multimerization of effectors. In addition, the PUF domain can be engineered to recognize any 8-mer RNA sequence, therefore enabling the generation and simultaneous operation of many Casilio modules."

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Drug resistance is one of the main causes of colon cancer recurrence. However, our understanding of the underlying mechanisms and availability of therapeutic options remain limited. Here we show that expression of PDK4 is positively correlated with drug resistance of colon cancer cells and induced by 5-fluorouracil (5-FU) treatment in drug-resistant but not -sensitive cells. Knockdown of PDK4 expression sensitizes colon cancer cells to 5-FU or oxaliplatin-induced apoptosis in vitro and increases the effectiveness of 5-FU in the inhibition of tumor growth in a mouse xenograft model in vivo. In addition, we demonstrate for the first time that TGFβ mediates drug resistance by regulating PDK4 expression and 5-FU induces PDK4 expression in a TGFβ signaling-dependent manner. Mechanistically, knockdown or inhibition of PDK4 significantly increases the inhibitory effect of 5-FU on expression of the anti-apoptotic factors Bcl-2 and survivin. Importantly, studies of patient samples indicate that expression of PDK4 and phosphorylation of

Smad2, an indicator of TGFβ pathway activation, show a strong correlation and that both positively associate with chemo resistance in colorectal cancer. These findings indicate that the TGFβ/PDK4 signaling axis plays an important role in the response of colorectal cancer to chemotherapy. A major implication of our studies is that inhibition of PDK4 may have considerable therapeutic potential to overcome drug resistance in colorectal cancer patients, which warrants the development of PDK4-specific inhibitors.

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"Essentially, the methylation of Cytosine at the C-5 position in genome provides a chemistry-orientated plasticity required for establishing functionally varying cells in mammals. By this modification, the gene expression can be comprehensively or specifically regulated and chromatin structures be dynamically manipulated. Meanwhile, the methylation of adenine at the N-6 position (N6-methyladenine, 6mA), which is the dominant DNA modification in genomes of bacteria and shows diverse functions, is absence in high eukaryotes. Intriguingly, 6mA was also found in unicellular eukaryotes. Until recently we and other groups1-3 discovered that this 6mA modification is also present in Caenorhabditis elegans and Drosophila melanogaster, respectively. These studies compulsively prove the presence of 6mA in invertebrate DNA1-4. Interestingly, in addition to the functions revealed in bacteria, 6mA may mark active transcription start sites and transposon activity and regulates embryonic development. These findings suggest that 6mA is a potential epigenetic mark in eukaryotes. However, it is not clear whether 6mA DNA modification is conservatory and present in mammalian genomes. These recent findings further prompted us to search 6mA DNA modification in mammals again. If so, how 6mA distributes in various tissues and how 6mA distributes in genome. Now we and other group showed the 6mA DNA modification in genomes of mice and human cells (approximately 1 6mA per 106 dA).

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Hepatitis A virus (HAV) infects ~1.4 million people annually and whilst there is a vaccine, there are no licensed therapeutic drugs. Hepatoviruses are unusual among picornaviruses, both in targeting the liver and also in their structure and life cycle. Here we show that nM concentrations of HAV-specific monoclonal antibody R10 neutralize virus infection efficiently. Cryo-EM structures of HAV full particles and empty particles at 3.4 Å and 3.8 Å resolution reveal a ‘layered’ RNA genome and altered inner capsid layer distinguishing the full and empty particles. The cryo-EM structure of full particles complexed with R10 Fab at 4.2 Å resolution, together with the crystal structure of R10 Fab allowed us to build a reliable model of the complex. R10 binds across the pentamer interface interfering with receptor attachment and viral uncoating, shedding light on the hitherto unknown mechanism by which this extraordinarily stable virus releases its RNA and demonstrating new opportunities for therapeutic intervention.

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Lipid molecules are one of the most important and structurally diverse components of the human body. They are not only crucial building blocks of the cellular architecture and of energy fuels, but also key signaling molecules actively involved in gene expression and signal transduction. During aging, lipid metabolism undergoes fundamental changes, and its homeostasis is tightly associated with organism physiology and pathology. Whether and how lipid metabolism actively regulates organism longevity remains unclear. In the first part of my talk, I will focus on a previously unknown signaling role of lipid metabolism in regulating longevity. Our work discovered a novel lysosome-to-nucleus retrograde signaling pathway in controlling healthy aging, and identified a longevity-promoting natural lipid metabolite. In the second part of my talk, I will introduce new technology platforms based on stimulated Raman scattering microscopy, as well as their

applications in tracking spatiotemporal dynamics of lipid molecules in living cells and organisms, to reveal the regulatory mechanism of lipid dynamics under physiological and pathological conditions.

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"Necroptosis is a programmed form of necrosis, which has been implicated in a variety of human diseases, including viral and bacterial infection, inflammation, ischemic tissue injuries as well as neurodegeneration. Understanding the mechanism of necroptosis pathway will provide valuable information for treating these diseases. The defining molecular player for necroptosis is Receptor Interacting Protein Kinase 3 (RIP3), which forms an amyloid like fiber with its close homolog RIP1 upon activation. Activated RIP1/RIP3 recruits MLKL and phosphorylates MLKL to form necrosome. Phosphorylated MLKL then homo-oligomerizes and translocates to membrane compartments and triggers cell death. However, how MLKL membrane translocation is regulated and how translocation causes cell death are still not clear. To identify novel necroptosis blocking compounds from natural products, we screened hundreds of Chinese Traditional Medicine herb extracts, and found several that had potent activities. We further purified one compound from herb Xanthium sibiricum and determined its chemical identity. Using a series of biochemical assays, we determined that this compound blocked necroptosis downstream of MLKL homo-oligomerization, suggesting that additional steps were required for MLKL oligomers to execute necroptosis. Using biotin conjugated compound we were able to identify its potential targets. How these targets might be involved in necroptosis execution is under investigation and will be discussed in the meeting."

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Plant cells are totipotent and competent to regenerate from differentiated organs. It has been known for six decades that high cytokinin:auxin ratio directs shoot regeneration. However, the underlying molecular mechanism remains elusive. We found that the homeodomain transcription factor WUSCHEL (WUS) is essential for de novo establishing shoot stem cell niche and the WUS-positive cell marks shoot progenitor during regeneration. High cytokinin:auxin ratio environment initially promotes the removal of repressive histone mark H3K27me3 at WUS locus in a cell-cycle dependent manner. Subsequently, the B-type ARRs, which encode the transcriptional activators in cytokinin signaling pathway, directly activate WUS expression through binding with HD-ZIP III transcription factors, the master regulators for apical/shoot fate commitment. Thus, our results reveal a two-step mechanism for cytokinin-directed shoot regeneration.

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Reprogramming somatic cells to generate iPSCs represents a powerful platform to develop new models and treatments for human disease. Our mRNA-based technology creates footprint-free iPSCs with ease and under cGMP-level control, in a process already set up for personalized medicine. The main portion of the presentation will be on pancreatic beta cells, which have unique potential in diabetes models, small molecule screening, and cell therapy. We have optimized a novel beta cell differentiation protocol that is faster and more efficient than all published methods. With mRNA transfections, we first convert iPSCs with nearly 100% efficiency into definitive endoderm cells, which can be further induced to pancreatic progenitor cells using a series of transfections of mRNA cocktails. Unlike most published methods, our protocol results in near100% NKX6.1 positive cells. Further differentiation results in detection of insulin producing cells in just 12-15 days. These beta cells naturally form islet-like structures and repeatedly perform glucose-stimulated insulin secretion (GSIS). These results indicate that our method of differentiating beta cells from iPSC is not only faster and more efficient than published methods, but is able to obtain high quality beta cells using our new method. I will also introduce our programs of differentiating personalized iPSCs to lung epithelial cells, hepatocytes, and mesenchymal stem cells for studying metabolic diseases, as well as muscle cells and neurons etc. for other applications.

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"RNA polymerase II (pol II) is responsible for synthesizing messenger RNA and non-coding RNA. High transcriptional fidelity is essential for many cellular functions. Errors in transcription can cause deleterious effect that can contribute to aging and human diseases such as cancer.
On the other hand, DNA lesions often affect transcriptional fidelity and arrest RNA polymerase II transcription elongation and signaling for DNA damage processing pathways such as transcription coupled repair or pol II ubiquitination. In addition, endogenous epigenetic DNA modifications, such as 5-Hydroxylmethylcytosine (5hmC), 5-formylcytosine (5fC) and 5-carboxylcytosine (5caC) that are involved in active DNA demethylation process, are also found to affect transcription. All these DNA modifications add additional potential regulatory layers. The roles of these oxidized species of 5mC in epigenetic and transcription regulation have been an area of intensive study recently. Here we report our effort in understanding the mechanism of pol II transcriptional fidelity control, functional interplays between these different forms DNA lesions and epigenetic DNA modifications on RNA polymerase II transcription elongation. We will report our unpublished result regarding to the molecular basis of how pol II sense modifications in the major groove and minor groove as well as crosstalk between transcription and DNA repair."

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TLR-triggered uncontrolled inflammation plays key role for the induction of sepsis and chronic inflammation-associated HCC (hepatocellular carcinoma). We have recently identified that (1) Vascular Endothelial Growth Factor Receptor-3 (VEGFR-3) and its ligand VEGF-C was upregulated by TLR4-NF-kB pathway, which then dampened TLR4-induced inflammatory response. Notably, ablation of the ligand-binding domain or tyrosine kinase activity of VEGFR-3 rendered mice more sensitive to septic shock. Aside from targeting lymphatic vessels, we suggest a key role of VEGFR-3-VEGF-C signaling in macrophages as a 'self-control' mechanism against bacterial infection (IMMUNITY, 2014; 40: 501-514). (2) Using murine chronic inflammation-associated HCC models, we found that the tumor suppressor serine/threonine protein kinase 4 (STK4) also regulates macrophage-mediated inflammatory responses to protect against HCC. Mechanistically, STK4 abrogated TLR-induced proinflammatory cytokine production by triggering the degradation of IL1R-associated kinase 1 (IRAK1). Treatment of STK4 KO mice with an IRAK1 inhibitor reduced serum IL-6 levels and liver tumors. Importantly, the reduced STK4 levels in macrophages from human HCC patients were inversely associated with the levels of IRAK1, IL-6. Our study suggests STK4 as a biomarker for inflammation-associated HCC (JCI, 2015; 125:4239-54). (3) Dock8 is a GEF for Cdc42 activation and regulates immune cell migration. We recently identified a new binding partner for Dock8 and examined their role in the regulation of T cell migration and the development of multiple sclerosis.

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We previously identified TGFβ activation in radiation-induced oral mucositis of oral cancer patients, and oral application of a TGF antagonistic protein, Smad7, with a cell permeable Tat-tag (Tat-Smad7) promotes healing of radiation-induced oral mucositis in mice (Han et al., Nat Med, 2013). In the current study, we assessed if Smad7-mediated cell protection is due to reduced DNA damage during radiation-induced oral mucositis healing. Wildtype mice orally treated with Tat-Smad7 or transgenic mice with Smad7 keratinocytes transduced with Smad7 were exposed to radiation. DNA damages, cell survival and oral mucositis healing were measured in vitro and in vivo. Irridiated mice with Smad7 transgene or Tat-Smad7 treatment had reduced ulcer sizes and significantly fewer cells positive for pH2AX, a marker for DNA damage and 8-OHdG, a marker for oxidative stress, compared to control mice. To determine if the above effects represent direct Smad7 functions in keratinocytes, we transduced human oral keratinocytes with an adenoviral Smad7 (adv.Smad7) or a control (adv.GFP) vector. Keratinocytes were irradiated 48h after

transduction. Smad7 transduced cells had less DNA breakage than control cells measure by alkaline comet assay. Reactive oxygen species (ROS) levels in irradiated keratinocytes were also reduced by Smad7 transduction. Further, Smad7 increased survival in irradiated normal but not cancer cells. Our study suggests that Smad7 functions as a radio-protector and -mitigator in radiation-induced oral mucositis, which contributes to its prophylactic and treatment effects on radiation-induced oral mucositis.

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The cleavage-furrow tip adjacent to the actomyosin contractile ring is believed to be the predominant sites for plasma-membrane insertion through exocyst-tethered vesicles during cytokinesis. Here we found that most secretory vesicles are delivered by myosin V on linear actin cables in fission yeast cytokinesis. Surprisingly, by tracking individual exocytic and endocytic events, we found that vesicles with new membrane are deposited to the cleavage furrow relatively evenly during contractile-ring constriction, but the rim of the cleavage furrow is the main site for endocytosis. Fusion of vesicles with the plasma membrane requires vesicle tethers. Our data suggest that the transport particle protein II (TRAPP-II) complex and Rab11 GTPase help to tether secretory vesicles or tubulovesicular structures along the cleavage furrow while the exocyst tethers vesicles at the rim of the division plane. We conclude that the exocyst and TRAPP-II complex have distinct localizations at the division site but both are important for membrane expansion and exocytosis during cytokinesis.

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Altered DNA methylation status is associated with human diseases and cancer; however, the underlying molecular mechanisms remain elusive. In a previous study, we identified numerous human transcription factors, including Krüppel-like factor 4 (KLF4), as sequence-specific DNA methylation readers that preferentially recognized methylated CpG (mCpG). An R458A mutation was identified created in KLF4 that abolished its binding activity to methylated DNA, ; but had no impact on binding to its canonical non-unmethylated motif. Taking advantage of this mutation, here we report report the biological function of mCpG-dependent gene activation of by KLF4 on the background of glioblastoma cells. We observed that induction of KLF4 promoted cell adhesion, migration, and morphological changes; however, R458A mutation completely abolished all of these phenotypes. Genome-wide profiling of gene expression and ChIP-seq analysis allowed us toSurprisingly, we identifiedy 116 genes that were directly activated via mCpG-dependent KLF4 binding activity. ImportantlyIn-depth mechanistic studies revealed that , recruitment of KLF4 to the methylated cis-regulatory elements of these genes resulted in chromatin remodeling and transcription activation. Our study demonstrates a new paradigm of DNA methylation-mediated gene activation and histone modificationchromatin remodeling, and provides a general framework to dissect the biological functions of DNA methylation readers and effectors.

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One of the key challenges of targeted cancer therapy is the lack of tumor-specific membrane targets. Tumor-specific antigens are mostly intracellular proteins, “undruggable” by traditional mAb therapy as well as the emerging CAR-T therapy. With Eureka’s technology, these previously inaccessible intracellular tumor-specific antigens can be effectively targeted through fully-human TCR-like antibodies that recognize an antigen-derived peptide presented by MHCI molecules on the cell surface. In collaboration with Memorial Sloan Kettering (and licensed to Novartis), Eureka’s ESK1 program successfully targets Wilms Tumor 1 (WT1) oncoprotein using different therapeutics options such as mAb, bispecific antibody, as well as CAR-T therapies. Eureka’s lead program ET1402, a CAR-T therapy targeting alpha-fetoprotein (AFP) for liver cancer, has also demonstrated effective anti-tumor activity in vitro and in multiple HCC xenograft mouse

models. Eureka's approach broadens the scope of cancer-specific antigens, especially for solid tumors.

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"Adeno-associated virus (AAV) vector-mediated gene therapy for FKRP deficiency.

Gene therapy has recently made great progress in clinical trials and attracted attention as a commercially viable treatment for genetic and chronic diseases and cancer, etc. In particular, the AAV vector has proven to be a vector of choice for gene replacement therapy by direct injection in patients. Numerous clinical trials, including the most recent spinal muscle atrophy (SMA) trial in infants by a simple intravenous (IV) injection of the therapeutic vectors, have shown encouraging safety and positive clinical outcomes. Thus, bodywide muscle and heart gene delivery by the IV injection route to replace the defective gene is the most plausible approach to treat muscular dystrophies.

We have been working on AAV-mediated gene therapy for dystrophin deficiency (Duchenne muscular dystrophy DMD) using different AAV capsids for gene delivery, testing different muscle-specific promoters and improving gene expression cassettes for safety and efficacy, etc, in mouse models of DMD. We have also carried out bodywide therapeutic minidystrophin gene delivery in the golden retriever muscular dystrophy (GRMD) dog model. Long term gene expression up to 8 years after a single dose IV injection and improvement in histopathology and muscle functions are observed. A local intramuscular injection of the AAV vector was tested in a human clinical trial 10 years ago. Armed with the newly improved therapeutic gene vector, we are preparing for a bodywide gene delivery clinical trial in DMD patients via a one-time simple IV injection. We will discuss the challenges on how to translate the preclinical results from the mouse models to dog model and to human clinical studies."

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"Oncogenic Signaling and Cancer Metastasis"

Activation of phosphatidylinositol 3 kinase (PI3K) and Ras signaling play a critical role in cancer progression. Oncogenic mutations in the p110α catalytic subunit of PI3K or Ras are frequently found in human cancers, which have been shown to promote cancer development and metastasis. We and others have shown that decreased expression of the p53 family member, ΔNp63α, promotes cancer metastasis. Our recent study demonstrates that the hotspot mutant p110α-H1047R or K/HRas-G12V significantly down regulates expression of ΔNp63α, the predominant p63 isoform expressed in epithelial cells. In addition, we shown that ΔNp63α is a direct FOXO3a transcriptional target, Furthermore, analyses reveal a significant correlation between FOXO3a and ΔNp63α expression in human cancer biopsy samples. Together, these results demonstrate new pathway that oncogenic PI3K or Ras signaling promotes cancer progression via down-regulation of ΔNp63α."

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Transcription, the process of converting genetic information stored in DNA to messenger RNA (mRNA), lies at the heart of gene expression. In the past half century, transcription has been studied extensively in-vitro to probe its mechanistic detail. However, the complex, heterogeneous cellular environment of a living cell is drastically different from the homogenous, well-mixed situation in in-vitro experiments, likely imposing different transcription kinetics. In recent years we have developed a set of new, single-molecule based gene expression reporters, chromosome conformation markers, and transcription activity indicators to probe the in vivo kinetics of transcription and its spatiotemporal organization in E. coli cells. Here I will present a case study in which we investigated the transcription factory model in bacterial cells.

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Drug metabolism and disposition are critical in maintaining the chemical and functional homeostasis of xenobiotics and endobiotics. Accumulating evidence suggests that many hepatic and systemic diseases can affect drug metabolism and disposition by regulating the expression and/or activity of DMEs and transporters in the liver. This presentation will focus on the recent progress in describing and understanding the hepatic injury and sepsis responsive regulation of DMEs in animal models. Liver ischemia and reperfusion (I/R) will be used as a typical example of a hepatic injury model, whereas lipopolysaccharides (LPS) and cecal ligation and puncture (CLP) will be used as the sepsis models. It is hoped that understanding the disease effect on drug metabolism will facilitate the efficient and safe use of drugs in the clinic.

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Precision medicine is an emerging method for disease treatment and prevention that takes into consideration individual genetic and environmental variability for each person. However, the advance of precision medicine is hindered by a lack of mechanistic understanding of the energetics and dynamics of drug-target and genetic interactions in the context of the whole human interactome. To address this challenge, we have developed a novel structural systems pharmacology approach to elucidate molecular basis and genetic biomarkers of drug action. Our approach combines big data analytics and mechanism-based modeling through integrating structural genomic, functional genomic, metabolomics, and interactomic data. By searching for all structurally-characterized human proteins and applying molecular modeling and machine learning, we are able to construct genome-scale high-resolution drug-target interaction models. Subsequently, we link the proteome-scale drug targets to genome-scale biological networks to identify drug modulation pathways and cryptic genetic factors. As proof-of-concept studies, we have applied our structural systems pharmacology approach to drug rescue and drug repurposing for precision medicine. We have identified cryptic genetic factors that account for the side effect of Torcetrapib, a cholesterol-lowering drug that failed in phase III clinical trial due to serious side effects. Recently, we have revealed molecular and genetic mechanisms of metformin, enabling us to repurpose metformin as a precision anti-cancer therapy. The predicted molecular targets of metformin were experimentally validated. Our results shed new light on repurposing metformin as safe, effective, personalized therapies, and demonstrate that structural systems pharmacology is a potential powerful tool to facilitate the development of precision medicine.

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Intellectual disability is a cognitive disability characterized by significant limitations both in intellectual abilities and in adaptive behaviors such as conceptual, social, and practical skills. It occurs naturally in 2%-3% of the population, as results of injury, disease, or genetics. Mutations in X-linked genes are important causes of intellectual disability. Many genes associated with intellectual disability have been identified, such as fmr1 in Fragile X Syndrome, MeCP2 and CDKL5 in Rett's syndrome and Ue3a in Angelman Syndrome. However, the molecular pathways that lead from the genetic mutations to intellectual disability are not clear. We use molecular biology, electrophysiological and imaging techniques in cultured neurons and knock-out mouse models to investigate how these genes affect neuronal development and functioning.

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Accumulative genome- and proteome-wide studies have consistently associated transcription and translation changes of CRMP2 (collapsing response mediator protein 2) with psychiatric disorders, yet little is known about its function in the developing or adult mammalian brain in vivo. Here we show that brain-specific Crmp2 knockout (cKO) mice display molecular, cellular, structural and behavioral deficits, many of which are reminiscent of neural features and symptoms associated with schizophrenia. cKO mice exhibit enlarged ventricles and impairments in

social behavior, locomotor activity, and learning and memory. Loss of Crmp2 in the hippocampus leads to reduced long-term potentiation with abnormal NMDA receptor composition, aberrant dendrite development and defective synapse formation in CA1 neurons. Furthermore, knockdown of crmp2 specifically in new neurons results in stage-dependent defects in their development during adult hippocampal neurogenesis. Our findings reveal a critical role for CRMP2 in neuronal plasticity, neural function, and modulation of schizophrenia-like behavior in mice.

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Cytosolic RNA/DNA sensing elicits primary defense against viral pathogens, by igniting cascades through MAVS or STING adaptor, the TBK1/IKKepsilon kinases, and the IRF3 transcriptional factor, as well as NF-κB signaling, to summon antiviral transcriptomes. Yet, how the activation status of MAVS, STING, TBK1 and IRF3 activation is controlled still remains obscure. Through a functional screen of the human kinome and analyses of interactomes, we identified that mammalian Sterile 20-like kinase 1 (Mst1), the metal ion-dependent phosphatase 1A (PPM1A), and a few other protein kinases and phosphatases, were the integral components of antiviral cascades to profoundly regulate host antiviral responses. In accordance, knockout of these kinases/phosphatases in cells, mice, and/or zebrafish altered host defense against infection of RNA/DNA viruses, while their ectopic expression endowed cells or zebrafish the vulnerable/invulnerable phenotype to resist viral pathogens. Furthermore, we elucidated the molecular bases underlying these regulations, including their targeting proteins, modifying residues, and the structural views. These findings identify a few physiological regulators of cytosolic nucleic acid sensing, and provide mechanistic insights into innate antiviral defense and potential antiviral prevention strategies.

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Emerging evidence show that epigenetic changes are causative for cancer etiology and progression. However, epigenetic therapy has not been widely used for breast cancer treatment due to the lack of defined targets and specific inhibitors for epigenetic enzymes. CARM1 is a protein arginine (R) methyltransferase that methylates histone H3 and a variety of non-histone substrates. CARM1 is overexpressed in various cancer types and the enzymatic activity is indispensable for its biological functions. We recently reported that CARM1 is highly expressed in triple-negative breast cancer and methylation of BAF155, a subunit of SWI/SNF chromatin remodeling complex, promotes breast cancer metastasis. This leads to the hypothesis that CARM1 methylation of cancer relevant substrates play essential roles in oncogenesis. Thus far, <15 CARM1 substrates are known. We knocked out CARM1 using Zinc Finger Nuclease (ZFN) in various breast cancer cell lines and showed that there was a massive loss of protein methylation detected by an anti-asymmetric dimethylarginine (ADMA) antibody. These hypo-methylated proteins, in response to loss of CARM1, are putative CARM1 substrates (Wang, L. et al., Cancer Cell, 2014). In this study, we successfully employed a novel quantitative mass spectrometry approach to deduce in vivo CARM1 substrates by comparing ADMA landscapes of WT and CARM1 KO model breast cancer cell lines. Over 140 novel substrates are identified in two-breast cancer cell lines and interestingly, the substrates share conserved proline rich motifs. Furthermore, we identified a novel module in CARM1 protein that recognizes the proline-rich sequence. In summary, this work systematically identified CARM1 substrates and regulons and provided molecular rationale for designing CARM1 inhibitors by targeting the new substrate-enzyme interface of CARM1 for breast cancer treatment.

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Epicardial epithelial-to-mesenchymal transition (EMT) is a vital process in embryonic heart development. During EMT, epicardial cells acquire migratory and invasive properties, and differentiate into new cell types, including cardiac fibroblasts and coronary smooth muscle cells. EMT is characterized by an increase in mesenchymal proteins such as Slug and Fibronectin, and a

decrease in cell-junction proteins such as E-Cadherin, and is dependent on TGF-β signaling. We have recently demonstrated that protein arginine methyltransferase-1 (PRMT1) regulates EMT in other cell types, such as HaCaT. To determine the role of PRMT1 in epicardial EMT, we used an epicardial cell line MEC1, ex vivo and in vivo mouse genetic models and established PRMT1-p53 pathway as a regulatory mechanism for epicardial EMT.

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Marfan syndrome (MFS) is a connective tissue disorder caused by mutations of a key extracellular matrix protein fibrillin-1 (FBN1). Mutated FBN1 impairs the integrity of extracellular matrix, which releases high amounts of TGFb ligands and inflammatory enzymes leading to pathological lesions in target organs and tissues, especially in the skeletal, ocular, skin, and cardiovascular systems. In the most severe case, patients can die of ruptured aortic aneurysms. Although animal models have been used to study the genetic disease, human induced pluripotent stem cells (iPSCs) offer a novel and homogeneic tool for dissection of the disease in vitro. Here we demonstrate that correction of a FBN1 mutation in iPSCs derived from a MFS patient allows detection of pathologic phenotypes in mesenchymal stromal cells and vascular smooth muscle cells derived from the iPSC line and isogenic comparison with its mutation-corrected counterpart. Inhibition of TGFb signaling reversed many of the phenotypes. Transcriptomic analysis revealed differentially expressed genes consistent with the phenotypic differences. Thus, we have generated a cellular model for pathologic phenotypes attributed to the FBN1 mutation, which may be developed as readout for screening of novel therapies of the disease.

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"Protein poly(ADP-ribosyl)ation (PARylation) regulates many biological processes, including DNA damage responses, transcriptional regulation and cell-death programs. Recent studies have shown that PARylation can serve as a signal for the polyubiquitination and degradation of several critical regulatory proteins, including Axin, 3BP2, PTEN and angiomotin. Tankyrase (TNKS) is the poly(ADP-ribose) polymerase (PARP) that earmarks substrates via PARylation, whereas the RING-type E3 ubiquitin ligase RNF146 (a.k.a. Iduna) is responsible for PARylation-dependent ubiquitination (PARdU). Our structural and biochemical studies have shown how TNKS recognizes protein substrates and how RNF146 subsequently polyubiquitinates PARylated substrates. Interestingly, iso-ADPr, the smallest internal poly(ADP-ribose) (PAR) structural unit, binds between the WWE and RING domains of RNF146 and functions as a robust allosteric signal that switches the RING domain from a catalytically inactive state to an active one. In the absence of PAR, the RING domain is unable to efficiently bind and activate an E2. Binding of PAR/iso-ADPr induces a major conformational change that creates a functional RING structure. Thus RNF146 represents a new mechanistic class of RING E3 ligases whose activities are regulated by non-covalent small-molecule binding.

Genetic manipulations at DNA and RNA levels (e.g. gene knockout, RNAi and CRISPR) have been the mainstream tools for functional studies of proteins. However, inducible protein knockout has a number of advantages over these traditional approaches, including the potential to induce fast and reversible response by the ligand, tunability (dosage-dependent quantitative depletion), depletion of the target protein from a specific cellular pool, and depletion of a specifically-modified form of the target protein. However, a general inducible protein knockout system that can deplete specific endogenous protein(s) remains to be developed. With well-defined molecular mechanism and structural basis for the RNF146 allosteric switch, we are using RNF146/iso-ADPr as the template to develop an inducible, tunable protein knockout system."

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Tissue and individual size is a fundamental feature of living organisms. It is essential for carrying out biological functions and yet is one of the most diverse and evolved characteristics of

the living world. In the past two decades, we have developed new forward genetic approaches to dissect the regulation of tissue size and growth and have identified the major growth control pathways including PTEN/TSC/mTor, Hippo/Lats, and JNK-JAK/STAT. Our recent studies start to reveal how these growth control pathways are integrated with morphogen signals to coordinate size and shape during development and how they are embedded in cell-cell communication mechanisms for monitoring and control tissue size. Interestingly, these developmental growth control mechanisms play critical roles in tumorigenesis. Studies of tissue size and growth in model organisms have not only contributed to our understanding of cancer biology, but also lead to development of new therapies.

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BACE1, a type I transmembrane aspartyl protease, cleaves amyloid precursor protein (APP) at the β-secretase site. Following this cleavage, gamma-secretase processes the membrane-bound APP C-terminal fragment to release amyloid peptides (Aβ). Since Aβ aggregation is the major component in amyloid plaques and excessive production of Aβ is linked to both familial and sporadic Alzheimer's disease (AD) pathogenesis, inhibition of BACE1 is widely pursued as an important target for AD therapy. A large volume of experimental results has shown that chemical inhibition of BACE1 in both mouse models and humans produces substantial inhibition of Aβ generation and amyloid deposition. Because of its important applications in human AD treatment, it is imperative to understand the potential side effects associated with mechanistic inhibition of BACE1. To address this practical question, we have used BACE1-knockout mice to explore the physiological functions of BACE1. We demonstrate that BACE1 can also process other membrane-bound substrates such as neuregulin-1 and Jagged-1 in addition to APP. The abolished cleavage of these two important substrates leads to hypomyelination of both central and peripheral nerves as well as altered neurogenesis. Collectively, we conclude that BACE1 is an important molecule that plays multiples roles in various neurological processes and that inhibition of BACE1 in the adult requires cautious monition of other neurological functions.

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Adeno-associated virus (AAV) has emerged as an important vector for human gene therapy. The evolutionary studies of AAV, both in its natural history and by genetic engineering, have greatly improved its application potentiality. We implemented DNA shuffling among capsid genes of AAV serotypes, and screened the resultant AAV library in a mouse in vivo model. An AAV variant named AAVM41 was enriched in the muscle and further characterized in in vivo and in vitro models. It exhibited the high transduction efficiency and improved tissue specificity in mouse myocardium after systemic administration. Furthermore, it has been successfully applied to delivery of the delta-sarcoglycan gene into a dystrophic hamster model to treat its cardiomyopathy. We are currently employing the directed evolution methods to engineer the AAV vector to enhance its transduction of the cardiac endothelial cells, which may further broaden its application to cardiac gene therapy.

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All living beings experience DNA damage during normal metabolism and exposure to natural environment. In an average day, tens of thousands of DNA lesions and adducts occur to each human cell. To avoid mutation and maintain genome integrity, multiple pathways have been evolved to identify DNA base lesions for excision or tolerance. In general there are two modes of lesion recognition, a lock-and-key in a one-to-one match and an ATP-dependent recognition of a broad range of lesions with high specificity for excision. I will highlight the nucleotide excision repair pathway and DNA translesion synthesis for UV lesion repair and tolerance as examples to illustrate the two modes of lesion recognition. Our detailed biochemical and structural analyses elucidate the molecular mechanisms for mutation avoidance and genome maintenance and provide insight to human diseases and cancer.

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Herein we report the sophisticated process of structural optimization towards a previously disclosed Src inhibitor, compound 1, which showed high potency in the treatment of triple negative breast cancer (TNBC) both in vitro and in vivo, but had considerable toxicity. A series of 3-(phenylethynyl)‐1H-pyrazolo[3,4-d]pyrimidin-4-amine derivatives were synthesized. In vitro cell-based phenotypic screening together with in vivo assays, and structure-activity relationship (SAR) studies finally led to the discovery of N-(3-((4-amino-1-(trans-4-hydroxycyclohexyl)-1H-pyrazolo[3,4-d]pyrimidin-3-yl)ethynyl)-4-methylphenyl)-4-methyl-3-(trifluoromethyl)benzamide (SKLB816). SKLB816 is a multikinase inhibitor, which potently inhibited Src (IC50 = 0.003 μM), KDR (IC50 = 0.032 μM), and several kinases involved in the MAPK signal transduction. This compound showed potent anti-TNBC activities both in vitro and in vivo, and good pharmacokinetic properties and low toxicity. Mechanisms of action of anti-TNBC were also investigated. Collectively, the data obtained in this study indicate that SKLB816 could be a promising drug candidate for the treatment of TNBC, hence merit further studies.

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Huntington's disease (HD) is one of the most common dominantly-inherited neurodegenerative disorders and is caused by a CAG repeat expansion translated into an elongated polyglutamine repeat near the N-terminus of mutant Huntingtin (mHtt) protein. HD is characterized by selective degeneration of neurons in the striatum and cortex, however the precise underlying mechanisms how a ubiquitously expressed disease protein could target specific types of neurons for degeneration and lead to clinical symptoms of HD remain unknown. To address this complex question, our laboratory has developed a series of Bacterial Artificial Chromosome (BAC) transgenic mouse models to study cellular and molecular mechanisms underlying HD pathogenic processes in vivo. We recently also applied integrative systems biology approaches to elucidate molecular networks that are perturbed by mHtt CAG repeat expansion in disease relevant brain regions. Our study provides novel paradigms to study pathogenesis and treatment of HD, and our approaches can be readily applied to study other neurodegenerative disorders such as Alzheimer's disease and Parkinson's disease.

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In humans, the visual system provides the majority of sensory inputs. In inherited and age-related retinal degenerative diseases, light sensing photoreceptor cells and the retinal projection neurons are prone to damages and death. Because some of the retinal degeneration diseases are slow progressing, intervention that delay cell loss and prolong neuronal survival can serve to preserve vision and improve quality of life. In recent years, a number of neuroprotective molecules have been tested in animal models either as broad-spectrum or cell-type specific neuroprotective agents. For example, ciliary neurotrophic factor (CNTF) has been used in clinical trials for several retinal degenerative diseases in humans, including age-related macular degeneration and glaucoma. However, cellular mechanisms of CNTF-mediated neuronal survival remain incompletely understood and severe unintended side effects may exist, thus hindering the therapeutic applications. Current and future research focusing on molecular pathways and effectors that lead to various cellular functions are necessary and ongoing to improve neuroprotective therapies in order to rescue vision.

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Over 100 types of chemical modifications have been identified in various types of RNAs including rRNA, tRNA, snRNA, snoRNA and mRNA, among which methylation is the most common

modification. The N6- methyl-adenosine (m6A) as the most common and abundant internal modification on mRNA molecules has been widely studied. The recent identification of m6A modifying enzymes including methyltransferase complex METTL3/METTL14/WTAP, two demethylases ALKBH5 and FTO and binding proteins such as YTHDC1, indicates that RNA methylation is reversible and represents a novel RNA epigenetic mechanism instead of micro-regulation in gene expression control. We will summarize recent progress in RNA m6A methylation and proposes its potential biological significance in this conference.

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Recent research into circRNA biogenesis has shown that exon back-splicing is enhanced by cis-complementary sequences in flanking introns. Strikingly, a single gene locus can produce multiple circRNAs through alternative back-splice site selection and/or alternative splice site selection; however, a detailed map of alternative back-splicing/splicing in circRNAs is lacking. Here, with the upgraded computational pipeline, we systematically annotated different types of alternative back-splicing and alternative splicing events in circRNAs from various cell lines. Compared with their linear cognate RNAs, circRNAs exhibited distinct patterns of alternative back-splicing and alternative splicing. Alternative back-splice site selection was correlated with the competition of putative RNA pairs across introns that bracket alternative back-splice sites. Unexpectedly, thousands of previously unannotated exons were detected in circRNAs from the examined cell lines. Although these novel exons had similar splice site strength, they were much less conserved than known exons in sequences. Finally, both alternative back-splicing and circRNA-predominant alternative splicing were highly diverse among the examined cell lines. Collectively, the annotation of alternative back-splicing and alternative splicing in circRNAs provides a valuable resource for depicting the complexity of circRNA biogenesis and for studying the potential functions of circRNAs in different cells.

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The spatiotemporal patterns of metabolite levels in living cells are pivotal in understanding signal transduction and metabolite flux in physiological and pathological conditions. To measure cellular metabolic states in single cell resolution and in vivo, we have developed a series of intensely fluorescent, rapidly responsive, genetically encoded sensors of wide dynamic range, which respond to subtle perturbations of various pathways of energy metabolism in real-time. The Frex sensors can be targeted to subcellular organelles and can be used to quantitate NADH concentrations inside living cells, while SoNar sensor can be used or to track cytosolic NAD+ and NADH redox states in living cells and in vivo. To precisely switch on/off gene expression at an exact location and time, we also developed an easy to use and robust light switchable gene expression system, termed as LightOn system, based on a synthetic light switchable transcription activator. The transgene system is easy to use, have high induction ratio and spatiotemporal resolution, and have minimal toxicity or interfere with cell signaling. Using these optogenetic approaches, we performed high-throughput metabolic screening of > 5,500 unique compounds and identified a potent NQO1-mediated redox cycling agent that produced extreme oxidative stress, selectively induced cancer cell apoptosis, and effectively decreased tumor growth in vivo. By using light, we have successfully controlled insulin gene expression and blood glucose level in living mice. We anticipate these optogenetic approaches, which allows visualizing and manipulation of cellular metabolic states, may not only be useful in studies of metabolic disorders, but also in the searching for metabolic diseases therapy.

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Therapy of established solid tumor remains a big challenge. Here, we showed that bacterial particle (fixation-inactive bacteria, fi-Bact) functioned as a biofunctional biomaterial for boosting innate immune response to disrupt tumor vascular system and for delivering drugs to tumor inner as a drug carrier. Intravenously injected fi-Bact made tumor shrinkage by inducing tumor vascular shutdown and extensive cell death. fi-Bact possessed a natural capability of carrying therapeutic drugs such as Doxorubicin, 5-Fu and Tax etc. fi-Bact loaded drugs (fi-Bact-Dox, fi-Bact-5-Fu, and fi-Bact-Tax) significantly inhibited the growth of established solid tumor and even made the complete regression of two-thirds tumors. Interestingly, the delivery of doxorubicin into tumor parenchyma was via neutrophils as the secondary carrier of fi-Bact-Dox.

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"STAT3 activation in ABC diffuse large B cell lymphoma: therapeutic consequences"

Diffuse large B cell lymphoma (DLBCL) is the most common type of aggressive B-cell lymphomas in the adult population. It contains two major molecular subtypes, namely, the germinal center B cell-like (GCB) and the activated B cell-like (ABC) DLBCLs. It is well documented that ABC-DLBCL cases have a significantly poorer survival response than GCB-DLBCLs in both the CHOP and the R-CHOP eras. However, the underlying cause of this subtype disparity is poorly understood. Nevertheless, these clinical observations raise the possibility for an ABC-DLBCL-specific resistance mechanism that is directed towards one of the CHOP components and inadequately addressed by rituximab. Here, we report that the main cytotoxic ingredient in CHOP, doxorubicin (Dox), has subtype-specific mechanisms of cytotoxicity in DLBCLs due to differences in the subcellular distribution pattern. Specifically, in cell line models of ABC-DLBCL, Dox is often enriched in the cytoplasm away from the nuclear DNA. As the result, Dox-induced cytotoxicity in ABC-DLBCLs is largely dependent on oxidative stress rather than DNA damage response (DDR). These findings are corroborated by gene signature analysis which demonstrates that basal oxidative stress status predicts treatment outcome among ABC- but not GCB-DLBCL patients. In terms of redox-related resistance mechanism, our results suggest that STAT3 confers Dox resistance in ABC-DLBCLs by reinforcing an antioxidant program featuring upregulation of the SOD2 gene. Furthermore, a small-molecule STAT3 inhibitor synergizes with CHOP to trigger oxidative stress and kill ABC-DLBCL cells in preclinical models. These results provide a mechanistic basis for development of novel therapies that target either STAT3 or redox homeostasis to improve treatment outcomes for ABC-DLBCLs. Our findings related to these new therapeutic approaches will be discussed at this conference."

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More than 100 different types of post-transcriptional modifications to RNA molecules have been characterized so far. Modifications in non-coding RNAs greatly impact their biological functions and have been extensively studied. In contrast, our knowledge regarding to the prevalence, mechanism and function of chemical modifications to mRNA and long non-coding RNA is limited. Several modified nucleotides in messenger RNA-namely 6-methyladenosine and inosine-have been demonstrated. The discovery of these RNA modifications has triggered an explosion of new information in the field of epitranscriptome. This rapid research progress has benefited significantly from timely developments of high-throughput sequencing technologies that enable identifications of these RNA modifications in the transcriptome. Because many RNA modifications form regular base pairs during reverse transcription and are of very low abundance, highly selective and sensitive methods with low background are required for their detection. In my lab, we utilize selective chemical/biochemical labeling to develop high throughput sequencing methods for these RNA modifications; in particular, we have developed two specific technologies for the transcriptome-wide sequencing of pseudouridine (Ψ) and N1-methyladenosine in RNA, respectively. With CeU-Seq, we identified thousands of Ψ sites in human cells and mouse tissues, showed that hPUS1 acts on mRNA and revealed inducible and stress-specific mRNA pseudouridylation events.

With m1A-ID-Seq, we identified ~900 m1A peaks in mRNA and ncRNA, revealed a prominent feature of enrichment in the 5'-untranslated region of mRNA transcripts and demonstrated that m1A in mRNA is reversible by hALKBH3, a known RNA/DNA demethylase. Such transcriptome-wide sequencing technologies will allow future functional studies of these RNA modifications.

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The surveillance systems that monitor macromolecules in cells such as DNA, RNA and protein are key to the faithful transfer of biological information and the well-being of all living organisms. Our lab studies both DNA and RNA surveillance mechanisms as well as their crosstalk in vertebrates using biochemical and cell biological approaches. In DNA surveillance, we are interested in understanding how the cell responds to DNA damage such as double-strand breaks (DSBs) and perturbations of DNA replication. In this direction, we have made significant contributions to the understanding of the molecular mechanisms of checkpoint activation, DNA end processing and repair after DSB damage or replication stalling. In the area of RNA surveillance, we have recently developed innovative tools to investigate the nonsense-mediated mRNA decay (NMD) pathway and discovered that NMD activity is regulated by intracellular calcium, a key signaling molecule that controls a wide range of cellular processes. Our ongoing work also suggests a novel link between NMD and the DNA damage response. These studies may have important implications for the understanding and treatment of cancer and other human diseases wherein the DNA and RNA surveillance systems are often broken.

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"Defeating Brain Metastasis: A Challenge of Cancer "Moonshot"

Of 1.6 million newly diagnosed breast cancer patients per year, about 10-16% will develop brain metastases. Among the different subtypes of breast cancer, HER2-overexpressing (HER2+) and triple-negative breast cancers (TNBC) have the highest incidence of brain metastasis. Recently, advances in targeted therapies for breast cancer (e.g., trastuzumab, T-DM1, and lapatinib) have prolonged patient survival through better control of the systemic disease; however, when patients have disease recurrence, their brain metastasis incidence doubles, which represents an emerging challenge. Current treatment options are limited and merely palliative for those patients with brain-metastatic breast cancer, and their one year survival is less than 20%. To develop effective treatments for brain metastasis, we strived to gain global understanding of the mechanisms of brain metastasis to guide rapid development of novel and clinically applicable targeted therapies.

We have made a major breakthorugh in understanding mechanisms of brain metastasis outgrowth that may be fast-track translated to the clinic to benefit patients. Metastasis to the brain depends on pathological cross-talks between metastatic tumor cells and brain microenvironment. We recently found that the brain astrocyte-derived exosomal miR-19a reversibly downregulates PTEN expression in metastatic tumor cells, thereby increasing their CCL2 secretion and recruitment of myeloid cell to promote brain metastasis. Importantly, CCL2 inhibitors have been used in clinical trials, our data showed stable ablation of CCL2 inhibits brain metastasis in vivo, demonstrating the potential of CCL2-targeting for therapeutic intervention of life-threatening brain metastases (Nature, 11/2015)."

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Diabetic peripheral neuropathy (DPN) often leads to neurotrophic ulcerations in the cornea and skin; however, the underlying cellular mechanisms of this complication are poorly understood. Here, we used post-wound corneal sensory degeneration and regeneration as a model and tested the hypothesis that diabetes adversely affects DC populations and infiltration, resulting in disrupted DC-nerve communication and DPN. In streptozotocin-induced type 1 diabetic mice, there was a substantial reduction in sensory nerve density and the number of intraepithelial DCs in unwounded

(UW) corneas. In wounded corneas, diabetes markedly delayed sensory nerve regeneration and reduced the number of infiltrating DCs, which were a major source of ciliary neurotrophic factor (CNTF) in the cornea. Exogenous CNTF or soluble CNTFRα partially restored the branching of diabetes-suppressed sensory nerve endings and regeneration in the diabetic corneas. Collectively, our data show that DCs mediate sensory nerve innervation and regeneration through CNTF and that diabetes reduces DC populations in UW and wounded corneas, resulting in decreased CNTF and impaired sensory nerve innervation and regeneration.

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The majority of breast cancer-related deaths are caused by metastasis – which occurs when circulating tumor cells (CTCs) are shed into systemic circulation and establish tumors in distant organs, such as the bones, lungs, liver and brain. We recently have established CTC lines derived from CTCs isolated from blood samples of patients with breast cancers. Using these CTC lines to assay their metastatic potency in xenografted mice, we are gaining new molecular insight on the metastatic tropisms of luminal type of breast cancers.

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The AKT pathway is a fundamental signaling pathway that mediates multiple cellular processes, such as cell proliferation and survival, angiogenesis and glucose metabolism. We recently reported that the immunophilin FKBP51 is a scaffolding protein that can enhance PHLPP-AKT interaction and facilitate PHLPP-mediated dephosphorylation of AKT at Ser473, negatively regulating AKT activation. However, the regulation of FKBP51-PHLPP-AKT pathway remains unclear. Here we report that a deubiquitinase, USP49, is a new regulator of the AKT pathway. Mechanistically, USP49 deubiquitinates and stabilizes FKBP51, which in turn enhances PHLPP's capability to dephosphorylate AKT. Furthermore, USP49 inhibited pancreatic cancer cell proliferation and enhanced cellular response to gemcitabine in a FKBP51-AKT dependent manner. Clinically, decreased expression of USP49 in pancreatic cancer patients was associated with decreased FKBP51 expression and increased AKT phosphorylation. Overall, our findings establish USP49 as a novel regulator of AKT pathway with a critical role in tumorigenesis and chemo response in pancreatic cancer.

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Approximately 66% of the gene mutations found in Fanconi Anemia patients are of the Fanconi Anemia complementation group A (FANCA). The 163kDa FANCA protein has been found to have important roles in DNA interstrand crosslink (ICL) repair and maintenance of chromosomal stability. Purified FANCA was shown to bind to DNA, recognize DNA ICLs, and regulate activity of the ICL-incision endonuclease MUS81-EME1. Due to the specific ICL recognition activity of FANCA, we hypothesized that FANCA is likely to catalyze strand separation in order to specifically recognize the crosslink damage. However, purified FANCA was unexpectedly found to catalyze single strand annealing (SSA) of complimentary ssDNA substrates in a DNA unwinding assay. Intriguingly, FANCA also promotes strand exchange between ssDNA and dsDNA. More importantly, disease-causing mutants of FANCA including Q772X, Q1128E, F1263Del, D598N, R951W, and R1117G are defective in annealing ssDNA. Finally, FANCA enhances the SSA of 3' overhang intermediate structures known to appear during DSB repair. Recent findings show a novel role for FANCA in class-switch recombination (CSR) involving the stabilization of short microhomology regions (Nguyen et al, 2014). In our study, we report biochemical evidence for the role of FANCA as a SSA factor. We propose the involvement of FANCA in CSR may be due to its ability to enhance the homology-mediated SSA at the ends of double strand breaks (DSB). Additionally, FANCA may be involved in the SSA pathway, as well as the synthesis dependent strand annealing (SDSA) pathway of the DSB repair process. Together, our data suggests FANCA may have multiple roles in the repair process of DNA ICLs and DSBs."

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Disseminated C. albicans infection in patients who have a weakened immune system is life-threatening. In hospitals 40% of bloodstream infections (candidemia) are caused by Candida spp. Despite the availability of several anti-fungal drugs, invasive candidiasis still has a high mortality rate ranging from 45 to 75%. The high morbidity and mortality associated with disseminated candidiasis are mainly due to the lack of early and accurate diagnostic tools, the limited anti-fungal drugs, and the emergence of drug resistance, thus highlighting the need to further understand host-pathogen interactions and the mechanisms of immune resistance to fungal spread, and to develop alternative immune-based strategies to combat candidemia. In normal hosts, C. albicans is controlled after activation of innate immune cells via cell surface pattern recognition receptors (PRRs) such as TLR2 and C-type lectin receptors (CLRs) that detect the infecting fungi. The CLRs Dectin-1 and Dectin-2/3 recognize C. albicans yeast cells and hyphae by binding to the surface β-glucans and α-mannans of the two fungal forms, respectively. Recognition of these molecules results in release of inflammatory cytokines from dendritic cells and macrophages, which is critical for anti-fungal immunity. The mechanisms that control this CLR-mediated pro-inflammatory response to fungal infection are completely unknown. In this study, we report that the E3 ubiquitin ligase Cbl-b targets K48-linked polyubiquitination of Dectin-1 and -2, two key pattern recognition receptors for sensing C. albicans, leading to Dectin internalization and degradation. Loss of Cbl-b function protects mice from systemic infection with a lethal dose of C. albicans and deficiency of Dectin-1, -2, or both in Cblb-/- mice negates this protection. Importantly, silencing Cbl-b gene in vivo protects mice from lethal systemic C. albicans infection. Therefore, our data reveals that Cbl-b's negative regulation of Dectin-1 and -2 is crucial for homeostatic control of innate immune responses against C. albicans infection. Our data also indicate that Cbl-b represents a potential host-directed therapeutic drug target for protection from disseminated Candidiasis.

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"Genome stability of mammalian cells is constantly challenged by DNA damage resulted from DNA replication errors and attacks by cellular metabolites, radiation and other environmental hazards. If not repaired, DNA damage can lead to gene mutations and even chromosome aberrations. Thus, dealing with damaged DNA is the utmost priority of a cell. Among many forms of DNA damage, double-strand breaks (DSBs) are the most dangerous to the integrity of the genome. DSBs are repaired through two main pathways, homologous recombination (HR) and non-homologous end joining (NHEJ). The choice between these two pathways is largely influenced by the cell cycle phases, with NHEJ primarily occurring in G1 and HR in S/G2 when homologous sequences are available from sister chromosomes.

We have been studying the function of anaphase-promoting complex (APC) in mitotic control and in maintaining chromosomal stability. APC is an E3 ubiquitin ligase that catalyzes the formation of K11-linked Ub chains on its substrates which are brought in for ubiquitination by two adaptor proteins Cdc20 and Cdh1. We have now accumulated data indicating that APC/Cdh1 is required for both HR and NHEJ repair of double strand breaks. Moreover, our data strongly suggest that Cdh1 plays a critical role in repair choice-making process in S/G2 cells by promoting BRCA1 recruitment."

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Hepatic de novo lipogenesis plays an important role in lipid homeostasis. However, its transcriptional regulatory network is still poorly defined. Here we demonstrate that the zinc finger protein ZBTB20 is required for the regulation of hepatic lipogenesis. The mice lacking ZBTB20 in the liver manifested hypolipidemia and reduced liver triglyceride contents, as well as decreased hepatic de novo lipogenesis and lipid secretion. ZBTB20 deficiency improved high carbohydrate diet (HCD)-induced hepatic steatosis and insulin resistance. Multiple critical glycolytic and lipogenic

genes were down-regulated in the mutant liver, including Glut5, Pklr, Fasn, Elovl6, Scd1, and ChREBP, while the expression or activation of SREBP1c was not affected. Furthermore, these affected genes failed to be fully activated in the mutant liver by HCD refeeding. ChIP analysis showed that ZBTB20 bound to the promoters of ChREBP□□in vivo. Lentivirus-mediated ChREBP overexpression in ZBTB20-deficient hepatocytes could largely rescue their lipogenesis and mRNA levels of the glycolytic and lipogenic genes. Put together, these findings indicate that ZBTB20 is an essential and unique regulator of hepatic de novo lipogenesis, and may serve as a therapeutic target for fatty liver.

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Hypoxia is associated with resistance towards radiation and chemotherapy. As tumors grow, they can sense the oxygen tension and reprogram critical pathways that are important for cancer cell survival and therapy resistance. One of examples is through upregulation of hypoxia inducible factor α (HIFα) and activation of HIF signaling downstream pathways. Dr. Zhang's lab is mainly interested in studying the oxygen-sensing pathway and how they contribute to the development of tumors as well as therapeutic resistance. One of the central players in this pathway is prolyl hydroxylase (EglN1, 2 and 3), a family of iron- and 2-oxoglutarate-depedent dioxygenases. EglNs can hydroxylate HIFα on critical proline residues, which will trigger von Hippel-Lindau (VHL)-associated E3 ligase complex binding and lead to HIFα degradation. Our lab currently studies hypoxia, prolyl hydroxylase and VHL signaling in cancer, especially breast and renal cell carcinomas. For example, we use proteomic and genomic approaches to screen for novel prolyl hydroxylase/pVHL substrates that play important roles in cancer.

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"Impacts on histone mutations on cancer epigenomes

Recent cancer genome sequencing efforts have revealed that many genes involved in chromatin regulation are mutated in a variety of cancers. Even more surprising are the discoveries that genes encoding histone proteins, the protein structural component of human chromatin, are mutated in different cancer types. For instance, one allele of the H3F3A gene, which encodes histone H3 variant H3.3, is mutated in 60% diffuse intrinsic pontine glima (DIPG), a high-grade pediatric brain tumors with dismal prognosis. The histone mutation found in all DIPG cases results in the replacement of histone H3 lysine 27 with methionine (H3.3K27M). In addition to H3F3A gene, one allele of H3F3B gene, another gene encoding H3.3, is mutated in over 90% chondroblastoma, a rare benign bone tumor. The histone mutation found in all chondroblastoma cases leads to replacement of histone H3 lysine 36 with methionine (H3.3K36M). In human genome, there are 14 genes encoding canonical histone H3 proteins that differ from H3.3 by four or five amino acids. H3K27 and H3K36 are conserved among all these histone proteins. Therefore, it is unknown how mutations at one allele of 16 histone H3 genes are linked to tumorigenesis. We have shown that the H3.3K27M mutation dominantly reprograms H3K27 di- and tri-methylation of (H3K27me2 and H3K27me3) in DIPG samples. In addition to a global loss of H3K27me3, H3K27me3 is retained at hundred loci. The alterations in H3K27me3 landscape is linked to changes in gene expression. In this meeting, I will first summarize our studies on H3.3K27M mutation promotes tumorigenesis of DIPG. I will then focus on discussing how the H3.3K36M mutation alters the H3K36 methylation landscape in chondroblastoma and promotes tumorigenesis of chondroblastoma. Based on these studies, we propose that different histone mutations reprogram epigenome of different progenitor cells, which in turn alters gene expression and transform different progenitor cells."

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Inhibitory leukocyte immunoglobulin-like receptors (LILRBs 1-5) transduce signals via intracellular immunoreceptor tyrosine-based inhibitory motifs (ITIMs) that recruit protein tyrosine

phosphatase non-receptor type 6 (PTPN6 or SHP-1), protein tyrosine phosphatase non-receptor type 11 (PTPN11 or SHP-2), or Src homology 2 domain-containing inositol phosphatase (SHIP), leading to negative regulation of immune cell activation. Certain of these receptors also play regulatory roles in neuronal activity and osteoclast development. The activation of LILRBs on immune cells by their ligands may contribute to immune evasion by tumors. Recent studies found that several members of LILRB family are expressed by tumor cells, notably hematopoietic cancer cells, and may directly regulate cancer development and relapse as well as the activity of cancer stem cells. LILRBs thus have dual concordant roles in tumor biology – as immune checkpoint molecules and as tumor-sustaining factors. Importantly, the study of knockout mice indicated that LILRBs do not affect hematopoiesis and normal development. Therefore LILRBs may represent ideal targets for tumor treatment. I will present recent progress about the roles of LILRBs and ITIM-receptors in leukemia development.

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It has become increasingly clear that cellular biochemical activities are compartmentalized in nanoscale domains that define the biochemical architecture of the cell. Despite advances in molecular sensors and optical imaging, direct interrogation of any minute activity domains at the molecular length scale remains a challenge. In this talk, I will focus on PI3K/Akt/mTOR and cAMP/PKA signaling pathways and present studies where we combined genetically encoded fluorescent biosensors, superresolution imaging, targeted biochemical perturbations and mathematic modeling to probe the biochemical activity architecture of the cell.

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Phosphorylation of the C-terminal domain of RNA polymerase II (CTD) plays an essential role in eukaryotic transcription by recruiting transcriptional regulatory factors to the active polymerase. However, the scarcity of basic residues and repetitive nature of the CTD sequence impose a huge challenge for site-specific characterization of phosphorylation, hindering our understanding of this crucial biological process. Herein, we apply LC-UVPD-MS methods to analyze post-translational modification along native sequence CTDs. Application of our method to Drosophila melanogaster CTD reveals the phosphorylation pattern of this model organism for the first time. The divergent nature of fly CTD allows us to derive rules defining how flanking residues affect phosphorylation choice by CTD kinases. Furthermore, our results reveal a novel role for Tyr1 as a crucial determinant for the placement of phosphorylation marks. Taken together our data supports the use of LC-UVPD-MS to decipher the CTD code and provides rules to program its function.

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Plexins are cell surface receptors for the guidance molecules semaphorins. Plexin signaling play critical roles in regulating neuronal and cardiovascular development. Malfunction of this pathway has been associated with neurological and cardiovascular disorders and cancer. We have shown that plexins transduce signal by acting as non-canonical GTPase Activating Protein (GAP) for the small GTPase Rap. Our structures show that the GAP normally adopts a closed inactive conformation, but can be activated through a conformational change by ligand-induced dimerization. Plexin signaling is mostly known to mediate repulsive guidance. However, binding of FARP1 to plexin has been shown to switch the signaling to promote dendrite growth. Our structural and functional analyses together elucidate the underlying mechanism for this switch.

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TALENs and Cas9/gRNA system have emerged as powerful reverse genetics tools in recent years to achieve targeted genome modifications in many organisms. We evaluated and

compared the success rate, targeting efficiency as well as specificity of these two genome editing tools in zebrafish (Danio rerio). By injecting in vitro transcribed mRNA of TALEN pairs into one-cell stage embryos, sixty seven out of 90 target sites could be effectively disrupted, indicating a success rate of 74% for TALENs in zebrafish. In the case of the Cas9/gRNA system, humanized Cas9 (hCas9) showed 32% success rate in generating indel mutations in zebrafish embryos, whereas a zebrafish-codon-optimized Cas9 (zCas9) could improve the success rate to 54%. Moreover, zCas9 showed 4% to 39% increase in efficiencies than hCas9 for the same target site. Comparing with TALENs, the Cas9/gRNA system has raised more concerns on its specificity and potential off-targeting effect, since the target site recognition for this system relies on base-pairing between the target sequence and the gRNA, where mismatches might be tolerated. By analyzing a relatively high efficienct (~95%) target site CNT05, we showed that a single-base mismatch in the 12-nt seed region but not in the 8-nt non-seed region of the target site could completely abolish the mutagenic activity of the Cas9/gRNA. To further evaluate the off-targeting effect of Cas9/gRNA at whole genome level, we designed a local tool called CasOT (http://eendb.zfgenetics.org/casot/) for searching of potential off-target sites in any given genome. Twenty one potential off-target sites for CNT05 in the zebrafish genome were predicted by using the CasOT and none of them showed detectable indels as revealed by high-throughput deep sequencing after PCR amplification. These results suggest that zCas9 is quite efficient in generating site-specific mutations in zebrafish, and the targeting effect could be specific if the target sites are selected carefully. I will also discuss our recent experience and improvement of gene targeting practice in zebrafish.

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The short duration of transgene expression represents a barrier in non-viral gene therapy and its mechanisms remain elusive. The objective of this study was to examine the role of histone deacetylases (HDACs) in regulating transgene silencing in mouse liver. By the method of hydrodynamic tail vein injection of a reporter plasmid (pCMV-SEAP), we demonstrated peak level of SEAP gene expression 36 hours after gene transfer followed by a quick decline reaching minimal level in 21 days. Transgene silencing was also observed in plasmid construct containing mouse IL10 gene. However, intra-peritoneal injection of HDAC inhibitors into animals with silenced reporter gene expression such as vorinostat, sodium butyrate or valproic acid resulted in an increase in serum concentration of reporter protein 24 hours after drug administration, suggesting a reactivation of silenced transgene. A dose response study showed reactivation of silenced gene is dose dependent and requires repeated drug administration to maintain reporter gene expression. Western blotting, real time PCR, and ChIP assays revealed that the reactivation of silenced reporter gene is associated with hyperacetylated histones (H3K9AC, H3K18AC, H3K27AC, H4K5AC, and H4K8AC) and increased binding to CMV promoter of acetylated histones (H3AC and H4AC) and TATA-binding protein. Inclusion of chromatin barrier sequences into promoter region of the plasmid prolonged reporter gene expression. These results suggest that HDACs play a critical role in transgene silencing. Inhibiting HDACs could be an effective approach to sustain transgene expression.

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We aim to overcome the challenge of eliminating microscopic metastases of breast cancer, so that distant recurrences and related deaths can be significantly reduced. We focused on bone micrometastases, which are precursors of overt bone metastases and possibly other metastases. Current adjuvant therapies intend to eliminate these cells. However, the therapeutic decisions and strategies are usually based upon pathological features of primary tumors. Micrometastases are likely to differ from their parental primary tumors either due to Darwinian selection or because of adaption. In both cases, the microenvironment in distant organs plays a critical role in driving the selection and/or in shaping the adaptive reaction. We envision that a critical barrier in curing breast cancer is the lack of knowledge about the crosstalk mechanisms between micrometastases and

their microenvironment niches. Key questions include the supporting pathways uniquely induced by cancer-niche interaction, and the differential therapeutic responses as compared to parental primary tumors. Our previous studies demonstrate that luminal-like breast cancer cells require the microenvironment niche that exhibits osteogenesis activity. The direct adherens junction-based interaction with osteogenic cells leads to increased cell proliferation of cancer cells, via activation of the mTOR pathway. Here, we presented a series of pre-clinical models that recapitulate the cellular nature of micrometastases, mimic their habitat and allow expedited testing of their drug responses, including 3D co-culturing systems that mimic cancer-niche interaction and an ex vivo bone-in-culture array (BICA) platform that recapitulate the microenvironment cellular constituents as well as therapeutic responses of cancer cells. These platforms are amenable to multiplex or even high throughput tests. Future studies will be performed to identify agents that can selectively eliminate micrometastases in the bone. These methodology and experimental platform here can be easily applied to bone metastases of other cancer types or pediatric osteosarcomas.

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STING (also known as MITA) critically mediates innate antiviral signaling and ubiquitination of STING is key to its function. However, the deubiquitination process of STING is unclear. Here we report that USP18 recruits USP20 to deconjugate K33- and K48-linked ubiquitination from STING and promotes stability of STING and DNA virus-induced expression of type I IFNs and proinflammatory cytokines. USP18 deficiency or knockdown of USP20 resulted in increased K48-linked ubiquitination and accelerated degradation of STING, and impaired activation of IRF3 and NF-□B as well as induction of downstream genes after infection with HSV-1 or transfection of various DNA ligands. In addition, Usp18-/- mice were more susceptible to HSV-1 infection compared to the wild-type littermates. Usp18-/-Ifnar1-/- mice produced decreased levels of type I interferons (IFNs) and proinflammatory cytokines and exhibited suppressed inflammation in the lungs compared to Usp18+/+Ifnar1-/- mice after HSV-1 infection. USP18 did not deubiquitinate STING in vitro but facilitated USP20 to mediate deubiquitination of STING in a manner independent of the enzymatic activity of USP18. In addition, reconstitution of STING into Usp18-/- MEFs restored HSV-1-induced expression of downstream genes and cellular antiviral responses. Our findings thus uncover previously uncharacterized roles of USP18 and USP20 in mediating virus-triggered signaling and contribute to the understanding of the complicated regulatory system of innate antiviral responses.

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Physicians use drugs to treat various diseases in neonates and infants. However, the current clinical practices have not considered the potential short- or long-term consequences on the patient's ability to metabolize drugs during adulthood who have received drug treatment early in life. A major reason is the lack of basic research to understand the consequences (short- or long-term) that drug exposure in early life has on drug metabolism and therapeutic efficacy. The goal in this study is to understand the short- or long-term impacts of drug exposure at early life on drug metabolism and therapeutic efficacy. Based on the previous publications and our preliminary data, we have formed a central hypothesis that "early life exposure to drugs that have the ability to activate nuclear receptors can result in either immediate (short-term) or persistent (long-term) alterations of expression and functions of certain drug metabolizing enzymes, further leading to alterations in therapeutic efficacy. This effect is dependent on the ability of the drug exposure with a certain dose at a specific age to alter epigenetic memory in liver." In an animal study, we used phenobarbital for drug exposure at early life of mice and omeprazole for therapeutic efficacy in adult life of mice and demonstrated that the dose of phenobarbital and age of treatment are the two key factors for the persistent induction of gene expression and consequential increases of enzyme activities of several tested P450 genes in adult liver. The increased P450 activities resulted in a decrease of proton pump inhibition in adult mouse stomach by faster metabolism of omeprazole.

These results may stimulate studies to evaluate the long-term impacts of drug treatment with different doses at neonatal and infant ages in human on drug metabolism and therapeutic efficacy for precision medicine."

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"Background: Alcohol consumption is a major cause of fatty liver disease. Mechanistic studies indicate that the pathogenesis of alcoholic fatty liver disease involves multiple factors including hepatic and extrahepatic factors. This study aims at determining the impact of adipose dysfunction on the development of alcoholic fatty liver.

Methods: A method of employing high-resolution mass spectrometry in combination with in vivo metabolite deuterium labeling was developed to investigate the effects of alcohol exposure on lipid homeostasis at the white adipose tissue (WAT)-liver axis in a mouse model of alcoholic fatty liver. Adipose triglycerides were first labeled by feeding heavy water for 5 weeks, following by 2 or 4 weeks of alcohol exposure, and the duterated triglycerides were measured in both the adipose tissue and liver. Adipose-specific lipin1 transgenic (aP2-LPIN1 Tg) mice along with wild type (WT) mice were then subjected to chronic alcohol exposure in order to determine if improving adipose lipid storage function leads to an attenuation of alcoholic fatty liver.

Results: Alcohol exposure decreased WAT mass but increased TAG concentration in the liver. A total of 14 deuterated TGs were significantly decreased in WAT with fold-change from 0.19 to 0.77. A total 13 and 10 duterated TAGs were significantly increased in the liver of alcohol-fed mice at 2 and 4 weeks, respectively, with fold-change of 1.7 to 6.3. Alcohol feeding to WT mice resulted in liver damage as indicated by elevation of plasma ALT/AST, which was alleviated in aP2-LPIN1 Tg mice. Alcohol feeding increased lipolysis and inhibited lipogenesis in WAT of the WT mice, which was attenuated in the aP2-LPIN1 Tg mice. Alcohol feeding induced hepatic lipid accumulation and down-regulation of beta-oxidation genes in the livers of WT mice. Overexpression of lipin1 in adipose tissue alleviated alcohol-induced lipid accumulation and abolished the down-regulation of beta-oxidation genes. Alcohol feeding also resulted in significantly hepatic ER stress and apoptosis in the WT mice. In contrast, overexpression of lipin1 in adipose tissue significantly attenuated alcohol-induced hepatic ER stress and apoptosis.

Conclusions: These results demonstrated that alcohol exposure induces reverse transport of adipose TAG to the liver, and enhancement of adipose lipid storage function leads to attenuation of alcoholic fatty liver. The study suggests a heathy WAT is required for maintaining hepatic lipid homeostasis."

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H2A.Z, a highly conserved histone variant in all species, is a universal mark of dynamic nucleosomes flanking gene promoters and enhancers. Histone chaperones and chromatin remodelers specifically recognize H2A.Z and facilitate the chromatin deposition and/or eviction of H2A.Z, but how they achieve this has been unclear. On the one hand, we identified Anp32e, a novel higher eukaryote-specific H2A.Z chaperone that promotes the H2A.Z removal from the chromatin, and determined the crystal structure of the Anp32e in complex with the H2A.Z-H2B dimer. On the other hand，we revealed the structural basis of the YL1(Swc2), a subunit of SRCAP(SWR1) chromatin remodeler, in recognition of H2A.Z–H2B. Our studies not only elucidated a highly conserved mechanism for H2A.Z recognition, but also provided insights into the important roles of H2A.Z chaperones they play in shaping the chromatin structure.

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Telomeres have been implicated in cancer and aging. In mammalian cells, the telomeres are bound by the core telomeric proteins TRF1, TRF2, RAP1, TIN2, TPP1, and POT1. These six telomeric proteins form large protein complexes and recruit different signaling molecules for

telomere length control and end protection. Genetic studies have been carried out using mouse knockout (KO) cells to illustrate how these telomeric proteins function in telomere maintenance. However, the core telomeric proteins in human appear to be different from mouse telomeric proteins. In addition, we have previously shown that human TIN2 can also regulate metabolism. Therefore, the function of human telomeric proteins in telomere maintenance and metabolic control requires further investigation by relevant genetic models. Here we report the generation of human cells that conditionally knockouts individual telomeric proteins. Our results indicate that human telomeric proteins differ in telomere maintenance and metabolic control.

128 Zhou Penghui Sun-Yat Sen University Cancer Center zhouph@sysucc.org.cn

The first application of cancer immunotherapy dates back to the 1890s, when William B. Coley treated cancer patients with a mixture of bacterial products and noticed regressions in a minority of those treated. However, this work was overshadowed by the advent of radiation therapy and chemotherapy. Throughout most of the last century cancer immunotherapy was a controversial and pessimistic field, though the concept of immune surveillance of cancer was proposed and many important discoveries occurred. The renaissance of tumor immunology began in 1990s, when the field entered the molecular era of immunology. Since then, the field of cancer immunology has been spurred by major discoveries such as the demonstration of cancer immune surveillance, the identification of tumor antigens, survival advantage of cancer immunity, the elucidation of different suppressive mechanisms in the tumor microenvironment and the establishment of multiple approaches of immunotherapy etc. In 2011, the FDA approved Ipilimumab (anti-CTLA4 Ab), the first drug with effect on metastatic melanoma，and Sipuleucel-T, the first therapeutic cancer vaccine. Recent clinical trial data of PD-1/PD-L1 blockade also showed great clinical benefit in many types of cancers. These remarkable successes of immunotherapies generated tremendous enthusiasm in the cancer immunotherapy field.

129 Zhou Dawang Xiamen University dwzhou@xmu.edu.cn

Tissue repair and regenerative medicine address the important medical needs to replace damaged tissue with functional tissue. Most regenerative medicine strategies have focused on delivering biomaterials and cells; yet, there is the untapped potential for drug-induced regeneration with good specificity and safety profile The Hippo pathway is a key regulator of organ size and regeneration by inhibiting cell proliferation and promoting apoptosis. Kinases MST1 and MST2 (MST1/2), the mammalian Hippo orthologs, are central components of this pathway and are therefore strong candidates to target in pharmacologically induced regeneration. Here, we report the discovery of a reversible and selective MST1/2 inhibitor, named XMU-MP-1, using an ELISA-base high-throughput biochemical assay. The co-crystal structure and the structure-activity relationship confirmed that XMU-MP-1 is ‘on-target’ to MST1/2. XMU-MP-1 blocked MST1/2 kinase activities, thereby activating downstream effector YAP and promoting cell growth. XMU-MP-1 displayed excellent in vivo pharmacokinetics and was able to augment mouse intestinal repair as well as liver repair and regeneration in both acute and chronic liver injury models at a dose of 1~3 mg/kg via intraperitoneal injection. Importantly, XMU-MP-1 treatment exhibited substantially greater repopulation rate of human hepatocytes in the Fah-deficient mouse model than the vehicle treatment control, indicating that XMU-MP-1 treatment might facilitate human liver regeneration. Thus, the pharmacological modulation of MST1/2 kinase activities might provide a novel approach to potentiate tissue repair and regeneration, with XMU-MP-1 as the first lead for the development of targeted regenerative therapeutics.

130 Zhu Xin-Hong Southern Medical University zhuxh@fimmu.com

Eicosanoids regulate a diverse set of homeostatic and inflammatory processes linked to health and diseases. However, little is known about the role of epoxyeicosatrienoic acid (EET) signaling in mood regulation. Here, we show that oligomerization of soluble epoxide hydrolase

(sEH), a key enzyme for EET signaling, was increased in human depression and mouse CSDS. Using pharmacologic and genetic approaches, we found that sEH modulated astrocytic ATP release in vitro and in vivo. Specific deletion of Ephx2 in astrocytes induced an antidepressant-like phenotype, whereas overexpression of hEPHX2 in mPFC prevented this phenotype and rapidly evoked depressive-like behaviors in response to stress. Our results indicate that EET signaling in astrocytes involves a homeostatic adaptation that helps an individual cope with stress.

131 Zhu Yuan Children's National Medical Center yuanzhu@gwu.edu

Medulloblastoma (MB), the most common malignant brain tumor in children, rarely harbors mutations in TP53 or other components of the p53 pathway. Individuals with Li-Fraumeni syndrome carrying germline TP53 mutations have increased risks of developing MBs, all of which are Sonic Hedgehog (SHH)-MBs. While the genetic studies from human tumors and mouse models support that p53 plays a role in suppressing SHH-MBs, it is not clear why most human SHH-MBs are not targeted for disrupting the p53 pathway and TP53-mutant SHH-MBs account for the majority of treatment failures within the SHH subgroup. Here we develop mouse SHH-MB models using different mutant p53 alleles with distinct capacities of activating apoptosis and/or cell-cycle arrest. While p53-dependent apoptosis, not cell-cycle arrest responses, eliminated most of tumorigenic cells induced by Ptch1 loss, some of Ptch1-deficient developing neural precursors formed tumors without activating the p53 pathway and appeared to generate p53-wild type SHH-MBs sensitive to p53-dependent apoptosis upon radiation. An experimentally increased response in p53-dependent cell-cycle arrest by radiation caused most non-Sox2+ MB cells to undergo neuronal differentiation, but enriched Sox2+ MB-stem cells, generating more aggressive SHH-MBs. In contrast, increased p53-dependent apoptosis largely eliminated Sox2+ MB-stem cells, significantly reducing the incidence of SHH-MBs. However, loss of wild type p53 occurred in Ptch1-loss driven SHH-MBs after radiation, mimicking human recurrent SHH-MBs. These results demonstrate an essential role of p53-dependent apoptosis in the suppression and therapeutic response of SHH-MBs.

132 Zhu Xiongwei Case Western Reserve University xiongwei.zhu@case.edu

Mitochondria play a critical role in neuronal function and, not surprisingly, mitochondrial dysfunction is a prominent feature in the brain of various neurondegenerative diseases including Alzheimer's disease (AD). Recent studies suggest that mitochondria are highly dynamic organelles characterized by a delicate balance of fission and fusion which revolutionizes our understanding of the regulation of mitochondrial structure, function and distribution. Our initial studies revealed an imbalance in mitochondrial fission and fusion in fibroblasts from sporadic AD patients compared with normal healthy fibroblasts from age-matched control patients. Later it was demonstrated that overexpression of familial AD (FAD)-causing AβPP mutant led to mitochondrial fragmentation and redistribution in neuronal cells along with altered expression of mitochondrial fission/fusion proteins. Soluble Aβ oligomers also induced mitochondrial fragmentation in primary hippocampal neurons which likely mediated Aβ-induced mitochondrial dysfunction and synaptic dysfunction. Genetic and pharmaceutical methods to rescue mitochondrial morphology and distribution could effectively restore Aβ-induced mitochondrial function and alleviate synaptic dysfunction both in vitro and in vivo, suggesting a causal involvement of mitochondrial dynamics in mediating Aβ-induced mitochondrial dysfunction. Importantly, we demonstrate significant changes in the expression and distribution of mitochondrial fission and fusion proteins in vivo in AD in consistent with a shifted mitochondrial dynamics towards excessive fission. Taken together, we suggest that such a fundamental shift in mitochondrial dynamics negatively impacts all aspect of mitochondrial function such as impaired bioenergetics, increased structural damage and ROS production and loss of mtDNA integrity which causes synaptic dysfunction and neuronal dysfunction that is critical to AD pathogenesis. Therefore, strategies to modify abnormal mitochondrial dynamics may be an attractive therapeutic intervention target for AD.

133 Zhu Bing Institute of Biophysics, CAS zhubing@ibp.ac.cn

TET family enzymes successively oxidize 5-methylcytosine (5mC) to 5-hydroxymethylcytosine (5hmC), 5-formylcytosine (5fC) and 5-carboxylcytosine (5caC), which may lead to eventual demethylation. 5hmC and TET proteins occupy distinct chromatin regions, suggesting unknown mechanisms controlling the fate of 5hmC at different chromatin regions. We report the identification of 5hmC binding proteins displaying preferential association with 5hmC in vitro. In mouse ESCs, the majority of these 5hmC binding proteins localize at distal regulatory regions and their chromatin association is largely TET1 dependent. Interestingly, their binding sites are generally depleted for 5hmC but enriched for 5caC. Knockout of the 5hmC binding protein results in an ectopic accumulation of 5hmC at the original binding sites, which suggests its stimulatory role in facilitating further oxidation of 5hmC at distal regulatory regions. Moreover, at these sites, the main enzyme responsible for further oxidation of 5hmC appears to be TET2, suggesting a stepwise regulation process for 5mC oxidation.

134 Zhu Li GenScript Corporation

How GenScript evolves from a start-up to the number one gene synthesis company in the world? GenScript is a China and US-based international biology CRO player. It was started by three Chinese scientists in New Jersey in 2002 with a few thousand dollars of out-pocket initial funding. By 2010, it had become the number one gene synthesis provider of the world. With synthetic biology reagent service as the core business, it has built a solid technology base and developed strong customer relationship in gene, protein, peptide, antibody, and cell line businesses. More recently, it has built a preclinical biologics service platform with some unique technologies such as single domain antibody and affinity maturation. At the end of 2015, GenScript went IPO in Hongkong stock market and started a new development stage. I will give a quick overview of the development path of GenScript and highlight the key elements of its rapid growth.

135 Zou Lee Harvard Medical School zou.lee@mgh.harvard.edu

The ATR checkpoint pathway is a key signal transduction pathway that controls the DNA damage response in human cells. On one hand, compromised ATR checkpoint could lead to genomic instability and promote tumorigenesis. On the other hand, the ATR pathway is required for the survival of cancer cells under genomic stress in specific contexts. Recent studies by others and us suggested that inhibition of the ATR checkpoint could be beneficial in specific therapeutic contexts. In my talk, I will discuss our recent efforts in identifying the vulnerabilities of cancer cells that can be exploited by inhibition of the ATR pathway. These studies may provide new therapeutic opportunities for the specific cancer types.

Ballot
CBIS Board
(2016-2018)

1. President, please select one.

- o Hao Wu
- o _____(fill in name)

2. Vice President, please select one.

- o Yibin Kang
- o _____(fill in name)

3. Board members, please select nine (9).

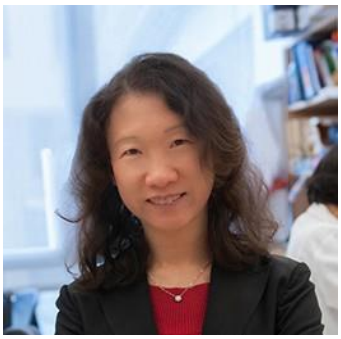
a. For re-election

- o Guo-Min Li
- o Lei Li
- o Yingzi Yang
- o Yimin Zou

b. New member

- o Lingling Chen
- o Yali Dou
- o Charlene Xiaoling Liao
- o Yijun Qi
- o Xin Sun
- o Yihong Wan
- o Yibin Wang
- o Zhenyu Yue
- o Shawn Xu
- o Wei Xu
- o _____(fill in name)

Hao Wu, Ph.D.



Personal Statement (Re-election, Candidate for President)

As a member of the expanding community of life scientists of Chinese origin, I am proud to have served on the CBIS board of directors during the past four years. I have learned a great deal from fellow board of directors and from interacting with all of you. As we move forward, we have many tasks in front of us to promote the missions of the society - career development of Chinese investigators in North America, China and around the world, enhancement of quality of science in China, and promotion of collaboration between academia and industry. I am seeking re-election to work with fellow board members, and most importantly, with all of you, to provide leadership for these society missions. Personally, I believe in the positive roles that professional organizations can play to assist their members and to advance the field, as my grandfather did for chemistry in China starting in the 1920s.

Education

1982-1985	B.S. (Biology)	Peking University, Beijing, China
1985-1988	M.D. candidate (Medicine)	Peking Union Medical College, Beijing, China
1988-1992	Ph.D. (Biochemistry)	Purdue University, West Lafayette, Indiana
1992-1997	Postdoc (Biochemistry)	Columbia University, New York, New York

Positions and Honors

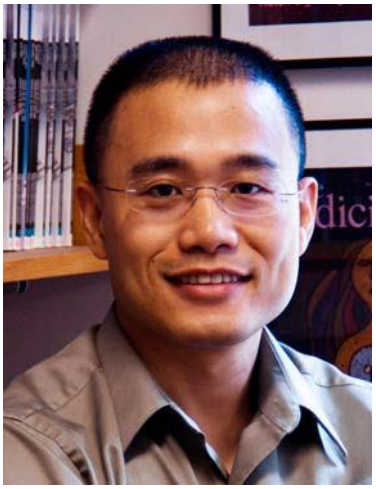
7/2012-	Asa and Patricia Springer Professor of Biological Chemistry and Molecular Pharmacology, Harvard Medical School, and the Program in Cellular and Molecular Medicine, Boston Children's Hospital
2003-7/2012	Professor of Biochemistry, Weill Cornell Medical College
2001-2003	Associate Professor of Biochemistry, Weill Cornell Medical College
1997-2001	Assistant Professor of Biochemistry, Weill Cornell Medical College

Election to the National Academy of Sciences, 2015
Pioneer Award from the National Institute of Health, 2015
Purdue University Distinguished Science Alumni Award, 2013
Elected Fellow of the American Association for the Advancement of Science, 2013
NIH Merit Award, 2012-2022
Editorial Board, Cancer Cell, 2012
Mayor's Award for Excellence in Science and Technology, 2003
Margaret Dayhoff Memorial Award, Biophysical Society, 2003
Rita Allen Scholar Award, 7/2002-6/2004
Pew Scholar Award, 7/2000-6/2004
Aaron Diamond Foundation Postdoctoral Fellowship, 6/1993-6/1996
Howard Hughes Medical Institute Predoctoral Fellowship, 4/1989-10/1992

Selected Recent Publications

1. Xing Liu, Zhibin Zhang, Jianbin Ruan, Youdong Pan, Venkat Giri Magupalli, **Hao Wu*** and Judy Lieberman* (2016). Inflammasome-activated Gasdermin D causes pyroptosis by forming membrane pores. ***Nature*** 535:153-8
2. **Wu H** and Fuxreiter M (2016). The Structure and Dynamics of Higher-Order Assemblies: Amyloids. Signalosomes, and Granules. ***Cell*** 165:1055-66.
3. H. Ru, M. G. Chambers, T. Fu, A. B. Tong, M. Liao, **H. Wu** (2015). Molecular Mechanism of V(D)J Recombination Captured by Structures of RAG1-RAG2 Synaptic Complexes. ***Cell*** 163:1138-52.
4. L. Zhang, C. Shuobing, R. Jianbin, J. Wu, A. B. Tong, Q. Yin, Y. Li, L. David, A. Lu, W. L. Wang, C. Marks, Q. Ouyang, X. Zhang, Y. Mao, **H. Wu** (2015). Cryo-EM Structure of the Activated NAIP2/NLRC4 Inflammasome Reveals Nucleated Polymerization. ***Science*** 350:404-9

Yibin Kang, Ph.D.



Personal Statement

As a proud member of CBIS since I became an independent investigator in 2004, I have benefited from the support and guidance from many senior Chinese biologists. I also had the privilege to serve in the CBIS Board of Directors in the last two years. In seeking the re-election to the CBIS board and as Vice President, I am committed to promoting the mission of CBIS, including helping the career development of young scientists, advancing the interest of the Chinese biological researchers (such as increasing our representation in AACR and other major conferences), connecting the academic and industrial research communities, and expanding the influence of CBIS in China.

Education

• 1991-1995	B.S. (Genetics)	Fudan University, Shanghai, China (June, 1995)
• 1996-2000	Ph.D. (Genetics)	Duke University, Durham, NC (May, 2000)
• 2000-2004	Postdoctoral	Memorial Sloan-Kettering Cancer Center, New York, NY

Positions and Employment

2004-2009	Assistant Professor
2010-2012	Associate Professor (tenured)
2012	Professor
2012-	Warner-Lambert/Parke-Davis Professor of Molecular Biology (endowed) Department of Molecular Biology, Princeton University, Princeton, NJ

Awards and Honors

2006	Era of Hope Scholar Award, Department of Defense Breast Cancer Program
2011	The Vilcek Prize for Creative Promise in Biomedical Science
2012	The 36th AACR Award for Outstanding Achievement in Cancer Research
2013	Young Investigator Award, Chinese Biological Investigator Society
2014	Fidler Innovation Award, Metastasis Research Society
2014	Fuller Albright Award, American Society for Bone and Mineral Research
2014	AACR Outstanding Investigator Award in Breast Cancer Research
2016	Komen Scholar

Professional Services

2016-2018	President, Metastasis Research Society
2008-2012	Board of Directors, Metastasis Research Society
2016-2018	Steering Committee of the AACR Tumor Microenvironment Working Group
2014-2016	Research Task Force, Metastatic Breast Cancer Alliance
2013-2015	Board of Directors, Chinese Biological Investigator Society
2011-2016	Scientific Committee, AACR Annual Meetings

Guo-Min Li, PhD

Personal Statement

If elected to a CBIS board member, I will do my very best to serve the society.

My research interests focus on DNA mismatch repair (MMR), an important cellular mechanism that maintains replication fidelity. I began to study this pathway when I was a postdoctoral fellow with Nobel laureate Paul Modrich (Chemistry 2015) at Duke University, where I discovered MMR defects as the genetic basis of certain hereditary and sporadic colorectal cancers, which contributed to Dr. Modrich's winning the 2015 Nobel Prize. During my over 20-year independent studies, I was able to systematically answer some timely questions in the field and have made some important observations contributing to our understanding of human MMR, which include 1) identification and characterization of 7 out of 10 essential MMR components; 2) reconstitution of human MMR in vitro; 3) discovery of the apoptotic function of MMR, where the MMR system triggers apoptosis in response to chemical and physical agents; 4) identification of the H3K36me3 histone mark as an essential factor for MMR in vivo by recruiting MMR proteins to replicating chromatin, and 5) discovery of MMR inhibition by EGFR-catalyzed PCNA phosphorylation. We are now targeting MMR factors for their potential roles in cancer therapy.

Education and training

1982	BS (Biology), Wuhan University, Wuhan, China
1985	MS (Cell Biology), Wuhan University, Wuhan, China
1991	PhD (Chemistry), Wayne State University, Detroit, MI
1991-1995	Post-doc (Biochemistry), Duke University, Durham, NC

Positions and Honors

Positions

1991-1995	Research Associate, Department of Biochemistry, Duke University
1995-1999	Assistant Professor, Department of Pathology, University of Kentucky College of Medicine
2000-2004	Associate Professor, Department of Pathology, University of Kentucky College of Medicine
2004	Associate Professor, Department of Toxicology, University of Kentucky College of Medicine
2006-2015	Professor, Department of Toxicology, University of Kentucky College of Medicine
2015-present	Professor, University of Southern California Keck School of Medicine

Honors

American Cancer Society Junior Faculty Award (1997)
Charles T. Wethington Research Award, University of Kentucky (2001)
Madeline F. James & Edith D. Gardner Endowed Chair in Cancer Research, UK (2001-2015)
Chang Jiang Scholar, The Ministry of Education of China (2006)

Adjunct Professor, Tsinghua University (2012-present)

Lei Li, Ph.D.

Personal Statement

We are interested in how DNA damage is sensed and transduced into checkpoint signals. We are investigating a number of genes that are critical in the generation of checkpoint signal and maintenance of genomic stability. By constructing genetic models both in animals and in somatic cells, we are able to elucidate their mechanism of function and their impact in tumorigenesis with particular interests in how checkpoint signals can be originated from DNA lesions and disrupted replication process. More recently, we have begun to explore how chromatin remodeling mechanisms interact with DNA repair and damage checkpoint pathways, since the highly compacted chromatin structure needs to be reconfigured to allowed access to DNA lesions. A second area of research deals with the repair of DNA interstrand cross-links, as many chemotherapy reagents are bifunctional DNA cross-linkers that covalently join the two strands of the double helix. We have identified a recombination-independent and error-prone pathway for the repair of DNA interstrand cross-links. Future studies will be focused on: 1. Characterization of the recombination- independent pathway of cross-link repair; 2. Identification of the essential components that carry out the error-free homologous recombination repair of interstrand DNA cross-links.

Education and training:

1984	B.S.	Beijing University, China
	Ph.D.	Beijing University Medical School
	Postdoctoral fellow	The University of Texas, MD Anderson Cancer Center

Current Positions:

Professor, Department of Experimental Radiation Oncology, the University of Texas MD Anderson Cancer Center
Joint Appointments: Department of Genetics, Department of Myeloma and Lymphoma

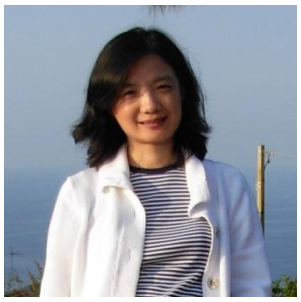
Previous Positions:

2009-present	Professor, Department of Experimental Radiation Oncology, Department of Genetics, UT MD Anderson Cancer Center
2004-2009	Associate Professor, Department of Experimental Radiation Oncology, Department of Genetics, UT MD Anderson Cancer Center
1998-2004	Assistant Professor, Department of Experimental Radiation Oncology, Department of Genetics, UT MD Anderson Cancer Center

Selected Honors and Services

American Federation of Aging Research Scholar, 1999
Editorial Board Member, Journal of Biological Chemistry, 2007-2012
NIH MGC Study section member 2004-2008
NIH NHGRI intramural program review committee

Yingzi Yang, Ph.D.



Personal Statement

It will be my great honor and pleasure to serve as a CBIS board member if elected. CBIS has provided a great platform for Chinese scientists in the US to interact with each other and with our colleagues in China. Such interactions have facilitated our career growth and promoted scientific excellence and political prominence both in the US and China. I would like to contribute time and effort to extend these traditions of CBIS and hope my service can make us a stronger group in many aspects.

Education and Training:

1988	BS (Biology)	Fudan University Shanghai, P. R. China
1996	PhD (Molecular Biology)	Weill Medical College of Cornell University, New York.
1996-2000	Postdoc	Harvard University, Dr. Andrew P. McMahon

Positions

February 2006-present Senior Investigator (tenured) Head of the Developmental Genetics Section Genetic Disease Research Branch National Human Genome Research Institute, NIH

August 2000- February 2006 Investigator (tenure track) Head of the Developmental Genetics Section, Genetic Disease Research Branch National Human Genome Research Institute, NIH

Awards and honors

2013:	Keynote speaker at the NIH ceremony of Asian-American month
2011:	NIH APAO Outstanding Achievements and Merit Scholarship Award
2011:	SCBA Young Investigator Award
2009:	NIH Award of Merit
2006:	US Government Service Award
2006:	NIH Award of Merit
2006:	NIH Director's Seminar Series
1996-1999:	Postdoctoral fellowship award from the Cancer Research Fund of the Damon Runyon-Walter Winchell Foundation.
1995:	Vincent du Vigneaud Award of Excellence from the Weill Medical College of Cornell University
1990-1995:	Graduate fellowship from the Weill Medical College of Cornell University

Yimin Zou, Ph.D.

Personal Statement

I am interested in serving as a CBIS Board Member and believe I can contribute to the efforts of the CBIS in promoting research excellence in the Chinese Bioscience Research Committee. As a former CUSBEA student, I currently serve on the Board of Directors of the Ray Wu Memorial Fund
and will rotate off from the Board in the next year or two. I have also served as the President of the Association of the Chinese Neuroscientists in America for two years and will rotate off from that role as well. I have organized the Chinese Social at the Society of Neuroscience Meeting for the last two years and have had great attendance. I think these experiences will help me assume a greater responsibility as a CBIS Board Member.

Education/Training

1984-1988	BS (Genetics)	Fudan University, Shanghai
1989-1995	PhD (Biochemistry)	Univ. of California, Davis and San Diego, Kenneth R. Chien
1995-1996	Postdoc (Dev. Biology)	University of California, San Diego, Kenneth R. Chien
1996-2000	Postdoc (Neuroscience)	University of California, SF Marc Tessier-Lavigne

Research Interests

My lab studies molecular and cellular mechanisms of axon guidance, synapse formation, and assembly, stability and regeneration of neural circuits. We identify molecular guidance cues that provide directional information for axon wiring *in vivo* as well as signal transduction pathways and cell biological mechanisms underlying growth cone turning. We also study synaptogenesis and
how specific synaptic connection patterns emerge from the interplay of molecular guidance system and neural activity. We study how central nervous system responds to traumatic injury and
develop therapeutic approaches to promote axonal and neuronal survival to combat degenerative disorders and improve axon regeneration and functional recovery following spinal cord injury.
These projects are coherently organized in the lab, providing broad training opportunities for postdoctoral fellows and also chances to collaborate with other lab members.

Positions and Employment

12/1996-10/2000 Postdoctoral Fellowship, University of California, San Francisco.
11/2000-04/2006 Assistant Professor, Dept of Neurobiology, Pharmacology and Physiology. The University of Chicago.
05/2006-06/2006 Associate Professor (with tenure), Dept of Neurobiology, Pharmacology and Physiology. The University of Chicago.
07/2006-06/2011 Associate Professor, Neurobiology Section, Biological Sciences Division. University of California, San Diego
07/2011-Present Full Professor, Neurobiology Section, Biological Sciences Division. University of California, San Diego
07/2012-Present Vice Chair, Neurobiology Section, Biological Sciences Division. University of California, San Diego

Ling-Ling Chen, PhD

Personal Statement (candidate for board member)

If elected, I will work closely with CBIS presidents and other board members to carry forward the goal and mission of CBIS to facilitate scientific interaction and personal friendship among our members. Efforts will also be made to strengthen the connection and to stimulate scientific exchanges among CBIS members and beyond for collaboration and education.

Education/ Training

2000	BS (Biology)	Lanzhou University, Lanzhou, China
2003	MS (Pharmacology)	Shanghai Institute of Materia Medica, CAS, China
2009	PhD (Biomedical Science)	University of Connecticut (Health Center), USA
2009	MS (Management)	University of Connecticut (Business School), USA
2010	Postodc (Stem cells and Mol. Biol)	University of Connecticut (Health Center), USA

Positions and Employment

1999-2000	Undergraduate Researcher with Dr. Ziren Wang, Lanzhou University, China
2000-2003	Graduate student with Dr. QIzhuang Ye, Shanghai Institute of Materia Medica, CAS
2003-2004	Research Associates at National Center for Drug Screening, CAS
2004-2009	Graduate student with Dr. Gordon Carmichael, UConn Health Center (UCHC), USA
2009-2010	Postdoc fellow with Dr. Gordon Carmichael, UCHC (supported by Connecticut Stem Cell Seed Award 09SCAUCHC16, PI/Ling-Ling Chen)
2010-2011	Assistant Professor in residence, Genetics & Developmental Biology, UCHC, USA
2011-	Principal Investigator, Institute of Biochemistry and Cell Biology, SIBS, CAS
2011-	Adjunct faculty, UConn Health Center, USA
2015-	Adjunct faculty, School of Life Science, Shanghai tech University, China

Services

2010-	Teaching, MEDS 5380-F40, Cell Biology, Graduate School, UCHC, USA
2011-	Journal Reviewer for Cell, Nature, Nature Reviews Genetics, Molecular Cell, Cell Metabolism, Trends in Biochemical Sciences, Nature Communications, Cell Research, Genome Biology, Nucleic Acids Research, Molecular and Cellular Biology, WIRES RNA, Genetics in Medicine, National Science Review, Stem Cells and Development, PloS One, Frontiers in Noncoding RNAs, and etc.
2012-	Guest Editor for Biomolecules
2013-	Member of CBIS; Member of ISSCR
2014-	Editorial Board of Genome Biology
2015-	Editorial Board of Trends in Genetics
2015-	Grant Reviewer for National Natural Science Foundation of China and European Research Council

Honors

1996-2000	First-Place Academic Excellence Awards, Lanzhou University, China
1999	Hui-Chun Chin and Tsung-Dao Lee Chinese Undergraduate Research Endowment
2000	Excellent Undergraduate Thesis Award, Lanzhou University, China
2000	Outstanding Graduate Award at Chinese Academy of Sciences, China
2008	Meeting Award at Workshop on Post-Transcriptional Regulation of Viral Gene Expression, Syria, VA, USA
2009	Best Poster Presentation Award at Gordon Research Conference, Galveston, TX, USA
2009	Best Oral Presentation Award at New England Stem Cell Consortium 2009 Junior Investigator Symposium, Worcester, MA, USA
2009	Connecticut Stem Cell Seed Award, USA
2012	Special Talents Program of SIBS, China
2012	National Outstanding Young Scholars Program, China
2013	National Excellent Young Scholars Program, NSFC, China
2015	Li RuQI Award from Chinese Genetics Society

Yali Dou, Ph.D

Personal Statement (candidate for Board Member)

Being a CBIS board member, I will commit to extend the great tradition of CBIS to younger generation of scientists, continue to build the platform that facilitates scientific collaborations and communications as well as seek new opportunities to increase the visibility and influence of CBIS in cutting-edge science, innovative education and societal contribution.

Education

1991-1996	BS, Basic Medicine, Beijing Medical University (now Peking University Health Center)
1996-1998	MS, Molecular Biology, University of Rochester
1996-2002	PhD, (Dr. Martin A. Gorovsky), University of Rochester
2002-2006	Postdoc (Dr. Robert G. Roeder), The Rockefeller University

Positions, Honors and Services

Positions

9/2012-present	Associate Professor, Department of Pathology and Biological Chemistry, University of Michigan
10/2006-8/2012	Assistant Professor, Department of Pathology and Biological Chemistry, University of Michigan

Honors and Awards

2014	Dean's Award in Basic Science, University of Michigan
2014	Inductee to UMMS League of Research Excellence
2012	Leukemia & Lymphoma Society Scholar Award
2011	Stand Up to Cancer IRG Award
2010	AACR Gertrude B. Elion Cancer Research Award
2010	American Cancer Society RSG Award
2007	Biomedical Science Scholar, University of Michigan
2004	The Irvington Institute for Immunological Research Fellowship

Services

2011-2013	Associate Member of Editorial Board, Journal of Clinical & Experimental Pathology
2013-2017	Member of Editorial Board, Molecular Cancer Research, AACR
2016-2021	Member of Editorial Board, Journal of Biological Chemistry
2013	NIEHS/NIDA, TARGET1 special R01 review panel (ZES1 LWJ-D),
2013	NIH, Special emphasis review panel ZRG1 BST-N (50) R: Functional Epigenomics: Developing Tools and Technologies for Manipulation of the Epigenome (R01)
2013	NIH MGA study session, ad-hoc
2014	National Science Foundation, 2014, ad-hoc
2015-2019	NIH MGA study section, regular member
2015	American Cancer Society, DMC study section, ad-hoc
2015	Special Emphasis Panel ZCA1 SRB-V (J1); NCI Omnibus R03 and R21, ad-hoc

Ad hoc reviewer for international agencies: National Science Foundation of China (NSFC, 2008), Breast Cancer Campaign (UK, 2009), National Health and Medical Research Council (NHMRC) Project Grants round, Australia, 2013, Foundation Against Cancer (Belgium, 2014), Leukemia and Lymphoma Foundation (UK, 2014)

Charlene Liao, PhD

Personal Statement (candidate for Board Member)

If elected as a CBIS board member with extensive leadership and industry experience, I will commit to fostering scientific collaborations among CBIS members, and between academics and biopharmaceutical industry. Together we will drive the translation of basic research and scientific progress into life-changing medicines and practices.

As one of the founding members of the CBIS, I helped draft its first bylaw. I will leverage my Board experience in non-profit and professional organizations to advance the mission of CBIS.

Charlene Liao graduated from Peking University in China with a Biochemistry majoring in 1986. She was among 30 students in China selected through the CUSBEA (China-United States Biochemistry Examination and Application) program to pursue Ph.D. in the United States in 1987. She holds Ph.D. in Biology from Brandeis University and conducted postdoctoral research in immunology at UCSF, first with Dr. Dan Littman, as a Fellow of the Damon Runyon Cancer Research Fund, and then with Dr. Art Weiss, as a Special Fellow of the Leukemia and Lymphoma Society of America. Since joining the industry and business world, Dr. Liao studied in the executive programs at Kellogg School of Management and at Stanford Graduate School of Business.

Dr. Liao began her bio-pharmaceutical industry career at Tularik Inc. (now Amgen) as a Scientist. Prior to joining Genentech Dr. Liao held various leadership positions at Rigel Inc., including Project Leader, Associate Director, and Director of Business Development. Dr. Liao joined Genentech in 2002 and has been a Project Team Leader since 2007. In near 14 years of her time at Genentech, Dr. Liao has contributed to numerous IND and/or CTA filings for 10 New Molecule Entities (NMEs), and has led drug development projects across therapeutic areas of oncology, immunology, neurology, inflammation, metabolic and infectious diseases. Recently Dr. Liao co-founded Immune-Onc Therapeutics, Inc. with Dr. Guo-Liang Yu and is serving as its President and CEO.

Dr. Liao has extensive leadership experience in non-profit organizations and served as their Board of Director or Executive Council/Committee Member:

- Dr. Liao is one of the founding members and Board of Directors of the **Ray Wu Memorial Fund (RWMF).** RWMF works closely with other professional organizations such as **CBIS** (Chinese Biological Investigators Society) and **SCBA** (Society of Chinese Bioscientists in America), and awards the Ray Wu Prize to recognize excellence in life science research by a graduate student in an academic institution located in mainland China, Hong Kong, Taiwan, or Singapore. At a Board of Director of RWMF, Dr. Liao led strategic planning and fund-raising efforts, and led the development and launch of its official website (http://www.raywumemorialfund.org)
- Dr. Liao served as Board of Director of **PRISMS** Inc. (Parents and Researchers Interested in Smith-Magenis Syndrome), an international patient supporting group that also sponsor research and foster partnerships with professionals (www.prisms.org)
- Dr. Liao was an Executive Council member of the **CABS** (Chinese-American BioPharmaceutical Society; www.CABSweb.org), and Co-Chair of the Career and Business Development Committee. She initiated and led the Career Advisory Network (CAN) program to bring 1:1 mentorship to CABS members, which is in its 7th year.
- Currently, Dr. Liao is an Executive Committee Member of **PKU Bio Teacher Fund** that provides financial support for retired teachers of the College of Life Sciences (including those from the original Department of Biology) at Peking University, China.

Xin Sun, Ph.D.

Personal Statement (Candidate for board member)

I am trained as a developmental biologist, first in *Drosophila* genetics, and later in mice genetics. Over the years, I have worked on many signaling pathways such as Notch and FGF, and many developmental processes, including fly wing and eye disc development, mouse gastrulation, somitogenesis and limb patterning. I established my lab at University of Wisconsin-Madison in 2002. Currently, we are funded to investigate a wide range of lung developmental processes and their links to neonatal and pediatric lung diseases.

Since 2007, I have given back my time each year to teach in China, both at the graduate level (Bio2000, Fudan University) as well as at the undergraduate level (Jiao-Tong University). In the US, I served for four years (2011-2014) as Director of the Cold Spring Harbor Laboratory summer course on Mouse Development, Stem Cells and Cancer, a flagship course for the Mouse research field. If elected, I will use my organizational experience to work with the CBIS Chair and other board members to establish a functional platform for scientific exchange between researchers in and outside of China.

Education and Training

1985-1989	B.S. Biochemistry, Fudan University.
1990-1996	Ph.D. dissertation with Dr. Spyros Artavanis-Tsakonas at Yale University.
1997-2002	Postdoctoral research with Dr. Gail Martin at UCSF.

Positions

2002-2009	Assistant Professor, Laboratory of Genetics, University of Wisconsin-Madison.
2010-2013	Associate Professor, Laboratory of Genetics, University of Wisconsin-Madison.
2014-now	Professor, Laboratory of Genetics, University of Wisconsin-Madison.

Honors and Services

2015-2021	NIH Lung Injury and Repair study section regular member.
2015	Romnes Faculty Fellowship, distinguished faculty award, University of Wisconsin.
2013-now	Editorial board, Developmental Biology.
2005-now	Editorial board, Developmental Dynamics.
2011-2014	Director of Cold Spring Harbor Laboratory summer course on Mouse Stem Cells, Development and Cancer.
2011,12,14	NIH Lung Injury and Repair study section ad hoc reviewer.
2012, 2014	NIH special emphasis panel reviewer.
2005-2007	Wisconsin Partnership Fund for a Healthy Future new investigator award.
2005	Organizing committee, International Society of Developmental Biology Annual meeting, San Francisco, CA.
2003-2005	March of Dimes Basil O'Conner award.
2001-2005	Burroughs-Wellcome career award.

Recent Selected Publications

Herriges JC, Zhang Z, Sui PF, Zhang Y, Anderson MJ, Swing DA, Zhang Y, Lewandoski M and **Sun X**. (2015) FGF-Regulated Etv Transcription Factors Control FGF-SHH Feedback Loop in Lung Branching. ***Developmental Cell***, 35(3)322-332.

Branchfield K, Lungova V and **Sun X**. (2015) A Three-Dimensional Study of Alveologenesis in Mouse Lung. ***Developmental Biology***, Epub ahead of print.

Branchfield K., Nantie, L., Verheyden, M., Sui, P., Wienhold, M. and **Sun, X.** (2016) Roundabout receptors control neuroendocrine cell clustering and lung sensory response. ***Science****,* in press.

Yijun Qi, PhD

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EDUCATION

Degree	Year	Major	Institution
Ph.D.	1998-2001	Plant Virology	Institute of Biotechnology, Zhejiang University
M.S.	1995-1998	Plant Virology	Institute of Biotechnology, Zhejiang Agricultural University
B.S.	1991-1995	Plant Pathology	Department of Plant Protection, Nanjing Agricultural University

PROFESSIONAL APPOINTMENTS

Year	Position	Institution
2016-present	Vice Dean	School of Life Sciences, Tsinghua University
2015-present	Professor	School of Life Sciences, Tsinghua University
2013-present	Director	Center for Plant Biology, Tsinghua University
2011-2015	Tenure-track Associate Professor	School of Life Sciences, Tsinghua University
2011-present	Investigator	Tsinghua-Peking Joint Center for Life Science (CLS)
2010-2011	Associate Investigator	National Institute of Biological Sciences, Beijing
2006-2010	Assistant Investigator	National Institute of Biological Sciences, Beijing
2004-2006	Postdoctoral Researcher	Cold Spring Harbor Laboratory
2001-2004	Postdoctoral Researcher	Department of Plant Biology, Ohio State University

RESEARCH INTERESTS

1. Mechanisms and functions of small RNAs in plants
2. Mechanisms and functions of long non-coding RNAs in plants

HONORS AND AWARDS

Year	Honors and awards
2013	Chinese Youth Science and Technology Award
2012	Recipient of The National Science Fund for Distinguished Young Scholars
2011	Changjiang Scholar, Ministry of Education of China
2010	Tanjiazhen Life Science Innovation Award

Yihong Wan, PhD

Personal Statement (candidate for board member)

If elected, I will contribute to the goal and mission of CBIS to facilitate scientific interaction and personal friendship among our members. Working with CBIS presidents and other board members, I will promote scientific collaboration and educational exchange among Chinese scientists worldwide. These efforts will significantly benefit our community and strengthen the tradition of CBIS.

Positions and Honors

Education/Training

1994	BS (Biochemistry)	Nankai University, Tianjin, China
1996	MS (Genetics)	Southern Illinois University, Carbondale, IL, USA
2002	PhD (Molecular Biology)	University of Colorado HSC, Denver, CO, USA
2008	Postdoc (Mol. Genetics)	The Salk Institute, La Jolla, CA, USA

Positions and Employment

1993-1994	Undergraduate Researcher with Dr. Dechang Gao, Institute of Molecular Biology, China
1994-1996	Graduate student with Dr. Thomas Schmidhauser, Southern Illinois University, IL
1996-2001	Graduate student with Dr. Steve Nordeen, Univ. of Colorado HSC, CO
2002-2008	Postdoctoral Research Associate with Dr. Ronald Evans, HHMI, Salk Institute, CA
2008-2015	Assistant Professor (tenure-track), Dept of Pharmacology, UT Southwestern Med Center, TX
2015-	Associate Professor with tenure, Dept of Pharmacology, UT Southwestern Med Center, TX
2012-	Adjunct faculty, Texas A&M Health Science Center, TX

Services

2009-	Journal Reviewer for Nature Medicine, Cell Metabolism, JCI, PNAS, Genes & Development, Endocrinology, Molecular Endocrinology, Molecular and Cellular Biology, Cell Reports, Expert Opinion on Therapeutic Targets, Journal of Leukocyte Biology, Cell Death and Differentiation, European Journal of Pharmacology, Journal of Cellular Biochemistry, Oncogene, International Journal of Biochemistry & Cell Biology, Clinical & Experimental Metastasis
2009-	Grant Reviewer for Swiss National Science Foundation, NIDDK-Diabetic Complications Consortium, NIH Study Sections (ODCS and INMP Study Section; ZRG1 MOSS-C (80) Special Emphasis Panel), Agency for Science, Technology and Research (A*STAR) in Singapore, Geneva University Hospitals and Faculty of Medicine Research Foundation in Switzerland
2009-	Member of Endocrine Society and American Society for Bone and Mineral Research
2012-	Member, American Society for Bone and Mineral Research Scientific Program Committee
2012-	Guest Associate Editor for PLoS Genetics and PNAS
2012-	Consultant for GlaxoSmithKline, Amgen, COI Pharmaceuticals, Acerta Pharmaceuticals, Guidepoint Global at New York, The Longevity Fund at San Francisco

Honors

1990-1994	ZhangShiChang Distinguished Scholar Award, NanKai University, China
1999	Univ. of Colorado HSC scholarship for the Annual Meeting of The Endocrine Society
2002	Keystone Symposia scholarship for the meeting “Chromatin Structrue and Activity”
1999-2002	U.S. Dept. of Defense Breast Cancer Research Program predoctoral fellowship
2003-2005	American Cancer Society postdoctoral fellowship
2008	Best Abstract Award for “Salk Science Day & Faculty Symposium”
2008-2012	Becton Dickinson Biosciences Research Grant Award
2008-2012	Virginia Murchison Linthicum Scholar Award in Medical Research
2009	ASBMR John Haddad Young Investigator Award
2010-2012	March of Dimes Basil O’Connor Starter Scholar Research Award
2010-2016	Cancer Prevention and Research Institute of Texas Individual Investigator Award
2011	Eugenia Rosemberg Award from The Endocrine Society
2012	Protégé of The Academy of Medicine, Engineering and Science of Texas (TAMEST)
2013-2014	The Charles and Jane Pak Center Innovative Research Award
2013	Awardee of Leadership Emerging in Academic Departments (LEAD) Program at UTSW
2014-2016	The Mary Kay Foundation Grant Award
2014	First GSK Pre-DPAc (Discovery Partnerships with Academia) Award at UTSW
2015	UTSW Friends of the Cancer Center Award

Wei Xu, PhD.

Personal Statement (candidate for board member)

If elected, I will assist the board members to carry out the mission of CBIS to enhance the professional interactions and collaboration of a diverse group of biologists representing many fields. I will help strengthen and optimize the operation of the society to provide the best education and research platform to bring the very best science and technology to most people possible. Working with board members, I will enhance the influence of the society to the policy makers of China to improve the financial situations of the investigators in biomedical science.

Education/Training

1987	BS (Chemistry)	Peking University, Beijing, China
1991	MS (Biophysics)	Institute of Biophysics, Academic Sinica, China
1994	PhD (Biochemistry)	University of Iowa, Iowa City, USA
1999	Postdoc	The Salk Institute for Biological Studies, La Jolla, USA

Positions and Honors

Positions

2005-2011	Assistant Professor, Department of Oncology, University of Wisconsin-Madison
2011-2014	Associate Professor, Department of Oncology, University of Wisconsin-Madison
2014-	Professor, Department of Oncology, University of Wisconsin-Madison

Honors and Services

David and Lucille Packard scholarship from the Keystone Symposia, 2002. FASEB MARC program travel award, 2003.
Endocrine Society Travel award, 2003.
Nuclear Receptor Keystone meeting: Orphan Brothers Travel Award, 2004. Susan G. Komen Breast Cancer Foundation; BCTR0600953 (2006-2009) NIH R01 CA125387 4/1/2008-3/30/2013
Elsa U. Pardee Foundation 12/31/06-12/30/07
Susan Komen Breast Cancer Foundation Spotlight, 2008
Shaw Scientist Award, 2008
Markos Family Breast Cancer Research Grant from Wisconsin Women's Health Foundation, 2010
DOD ERA of HOPE Scholar, 2010
Rush Basic Research Award from UW Comprehensive Cancer Center Retreat, 2011
Villas Associate of University of Wisconsin, 2012
Society of Toxicology Achievement Award, 2013
Villas Distinguished Achievement Professor, 2014
Member of the Scientific Advisory Committee on Alternative Toxicological Methods (SACATM) of NIEHS, 2014-present
Reviewer for National Natural Science Foundation of China, 2010
Reviewer for Italian Ministry of Health, 2010, 2013
Reviewer for MRC, UK, 2011, 2012, 2013
Reviewer for NIH, CBSS, 2011, regular member since July, 2012
Reviewer for National Science Center, Poland, April, 2014
Editorial Board Member, PPAR research, 2006-present
Editorial Board Member, Current BioData Epigenetic Regulators, 2006-present Editorial Board Member, Chemical Research in Toxicology, 2009-present Editorial Board Member, American Journal of Cancer Research, 2011-present Editorial Board Member, PLOS One, 2013-present

Yibin Wang, Ph.D.

Statement

I am honored to be considered as a CBIS board member and will strive to promote scientific exchange and professional success of its members, and to advance biomedical research and education in China.

Education/Training:

1986	Diploma (Biochemistry),	Fudan University, Shanghai, China
1988	B.S. (Bio. Sciences)	State University of New York, Albany, New York
1993	Ph.D. (Genetics/Cell Bio)	Baylor College of Medicine, Houston, Texas
1993-96	Postdoc (Neurobio)	Scripps Research Institute, Lo Jolla, CA
1996-99	Postdoc (Mol. Cardiology)	University of California, San Diego, CA

Positions:

1998-2003 Assistant Professor. Department of Physiology, University of Maryland School of Medicine, MD.

2003-2006 Associate Professor. Departments of Anesthesiology and Medicine, University of California, Los Angeles, CA.

2006- Professor. Division of Molecular Medicine, Departments of Anesthesiology, Physiology and Medicine, Member. Molecular Biology Institute, Cardiovascular Research Laboratories, UCLA, CA.

2011- Vice Chair for Research and Chief, Division of Molecular Medicine, Department of Anesthesiology, David Geffen School of Medicine, UCLA

2015- Chair, Cardiovascular Research Theme at David Geffen School of Medicine, UCLA

Honors:

1985-1986 , First Degree Academic Award, Fudan University; 1989-1990, Howard Hughes Medical Institute Pre-doctoral Fellowship; 1994, Visiting Scholarship Award. Chinese Natural Science Foundation; 1997, Ruth L. Kirschstein National Research Service Award; 1993, Student Scholarship Award. Association of Chinese American in Biosciences; 2005, Established Investigator Award, American Heart Association; 2009, Chang-Jiang Scholar Visiting Professorship, Ministry of Education, China; 2013- Fellow of AHA; 2013-, 1000 Talent Program, China (short-term);2013- Honorary Professor, Peking Union Medical College and Chinese Academy of Medical Sciences, Beijing, China; 2015-2018, Oversea's Expert, Chinese Academy of Science

Service:

1999-2015, Member, Study Section Pathophysiology, AHA, Mid-Atlantic, National, IRG, et al.; 2003-2015, Adhoc and Regular Member for NIH Study Section, CCHF, SEPs, MIM, PPG, T32; 2004, External Reviewer, Swiss National Science Foundation; 2006, 13, External Reviewer, Israel-American Research Foundation; 2008, Adhoc Reviewer for Fondazione Cassa di Risparmio di Padova e Rovigo, Italy; 2008, 13,14,15, Reviewer, Chinese National Science Award Selection Committee; 2008, 13,14,15, Member, NRPGM Research Program Review Committee, Taiwan; 2008, Adhoc Reviewer,Research Initiatives for Nevada System of Higher Education; 2009-15, Mail Reviewer, China Ministry of Education and Li Ka Shing Foundation, Chang Jiang Scholars Program; 2010.7, 12, Reviewer, Medical Research Council, United Kingdom; 2010, Reviewer, International Science and Technology Center (ISTC) and Science and Technology Center in Ukraine (STCU), US State Department; 2011. 2, Adhoc Reviewer, National Medical Research Council, Singapore; 2011. 5, Adhoc Reviewer, European Research Council, Brussels, Belgium; 2012. -, Member, International Evaluation Panel, Center for Life Sciences – PKU/Tsinghua, Beijing, China; 2013.6, Member, External Reviewer for Young 1000 Talents, China; 2013-, Member, Scientific Advisory Board, Keystone Symposium; 2014.5, Member, Pennsylvania Department of Health, Oakridge Associated Universities Performance Review Panel; 2014, Reviewer, Foundation for Prader-Willi Research; 2015, Member, DoD CDMDR Scientific Review Panel;

Officers and Committee Members of International Societies

2013-2016, Member, Scientific Advisory Board, Keystone Symposia, USA;

2014-2016, Member, Scientific Advisory Board,Translational and Clinical Research (TCR) Flagship Programme, National Research Council, Ministry of Health, Singapore.

2015-2017, Member, Research Committee of AHA.

Editorial Board Members:

2004-2007, 2009- Journal of Molecular and Cellular Cardiology; 2003- Circulation Research; 2011, Journal of Cardiac Failure; 2012 – 2017, Journal of Biological Chemistry,

2013-, Academic Editor, PlosOne.

Zhenyu Yue, Ph.D.

Personal Statement My goal is to work closely with the CBIS President and other members of the Board and serve the CBIS community with my best effort in promoting CBIS traditions, culture and spirit to pursue the best quality of science that has benefited the entire Chinese scientific community. I will also bring my best knowledge and experience in cell biology and neurodegenerative disease research to the Board and help structure and organize our scientific activities (conferences, collaboration, and fund raising) and improve education of the next generation scientists.

Education and Training

1988	BS (Cell Biology)	Wuhan University
1991	MS (Vertebrate Genetics)	Chinese Academy of Sciences, Wuhan
1997	PhD (Biochemistry/molecular Biology) Postdoc	Rutgers/Robert Wood Johnson MS Rockefeller University, USA
2003	(Molecular Neuroscience)	

Position and Honors

Positions

2000-2003	Postdoc/Research Associate, Howard Hughes Medical Institute, The Rockefeller University,
2004-2008	Assistant Professor, Department of Neurology/Neuroscience, Mount Sinai School of Medicine, New York, NY.
2004-present	Adjunct Faculty member of The Rockefeller University, New York, NY.
2008-2013	Associate Professor, Department of Neurology, Mount Sinai School of Medicine, New York, NY.
2013-present	Professor, Department of Neurology/Neuroscience, Icahn School of Medicine at Mount Sinai, New York, NY

Honors

1998-2001	Postdoc Fellow, Howard Hughes Medical Institute.
2008	Faculty Council Award for Academy Excellence, Mount Sinai School of Medicine
2013	Aidekman Endowed Professorship
2015 (5/13)	Keynote speaker, Friedman Brain Institute, Mount Sinai
2015 (9/25)	Keynote speaker, SfN Hudson-Berkshire Chapter, Albany,
2013	Honorary Professor, Xiangya School of Med., Central South University, Hunan Guest Professor, Tongji
2015-	Med. College, HuaZhong Sci. and Tech. University, Wuhan Guest Professor, Wuhan University
2015-	Medical College, Wuhan

Other Experience and Professional Memberships:

2012/10-	NIH study section, CMND, Regular. NIH
2011/06	study section, CMND, Ad Hoc.
2011	NIH study section, special review panel for the ZES1 LWJ-J MI NIH study
2011/06	section, invited to MBPP Ad Hoc
2010/10	NIH study section, ZRG1 CNNT-Q (02) S Ad Hoc. NIH study
2010/06	section, ZRG1 CNNT-Q (02) S Ad Hoc. NIH special panel
2010	RC4, Ad Hoc.
2011-	Parkinson Disease Foundation, Review committee, Scientific Advisory Board,
4/25/2011	Beijing Univ-Hsing Hua University Joint Life Center, Grant Review Panel, Beijing
2014－5	National Natural Science Foundation of China (life Science), (国自然基金二**审评**委）
2010-present	Research Grants Council, Hong Kong, China.
2009, 2011	National Science Foundation (NSF), Division of Molecular and Cellular Biosciences,

Editorial Board/Editor:

2007-present	Autophagy (Associate Editor)
2012	Chief Editor of the book Autophagy of the Nervous System, World Scientific. ent npj
2015-pre	Parkinson's Disease (editorial board member)
2015	guest editor for an issue "Autophagy in Neurodegeneration" (Brain Research)

X.Z. Shawn Xu

PERSONAL STATEMENT (candidate for board member)

CBIS is our own community. If elected, I will try every effort to service this community, expand this community, and further strengthen its role as a bridge and platform for facilitating scientific exchange, fermenting collaboration, and building friendship among all CBIS members. As a long-term member of CBIS and a recipient of the Young Investigator Award of CBIS, I have the passion, dedication, commitment and skills to service our community. Let's work together to make CBIS a warm home for all Chinese biological investigators.

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE	Completion Date	FIELD OF STUDY
Wuhan University, Wuhan	BS	1991	Biochemistry
Wuhan University, Wuhan	MS Ph.D.	1994	Biochemistry
Johns Hopkins University, Baltimore, MD California	Postdoc	2000	Neuroscience & Biochemistry
Institute of Technology, Pasadena, CA		2005	Neuroscience & Genetics

POSITIONS AND HONORS

Positions and Employment

1991 - 1993	Guest Researcher, National Laboratory of Biomacromolecules, Institute of Biophysics, Chinese Academy of Sciences, Beijing
2005 -	Assistant Professor, Associate Professor (with tenure), and Full Professor (with tenure), Department of Molecular & Integrative Physiology, Life Sciences Institute, University of Michigan, Ann Arbor, MI
2012 –	Bernard W. Agranoff Collegiate Professor of the Life Sciences, Department of Molecular & Integrative Physiology, Life Sciences Institute, University of Michigan, Ann Arbor, MI

Other Experience

2006 - 2012	Ad Hoc Reviewer for NIH study sections: CEBRA, 2013; ZDA1 SXC-E 2013; MNPS 2013; SYN 2012; ZDA1 SXC-E 2012; NTRC 2011; ZRG1 MDCN-C 2009; ZRG1 MDCN-C 2006.
2007	Ad Hoc Reviewer, Integrative Organismal Biology, NSF
2009 -	Ad Hoc Reviewer, NSF of China
2015-	Editorial board, Biophysics Reports
2012 - 2016	Permanent member of NTRC, NIH Study Section

Honors

2000	Harold M. Weintraub Graduate Student Award (Harold Weintraub Foundation)
2000	Michael Shanoff Young Investigator Award (Johns Hopkins University)
2001	Helen Hay Whitney Foundation Postdoctoral Fellowship (2001-2004)
2006	Sloan Fellow
2007	Pew Scholar
2009	Young Investigator Award, CBIS - Chinese Biological Investigators Society
2011	Basic Science Research Award, University of Michigan Medical School

CBIS Board Members

(December, 2013 to August, 2016)

Weimin Zhong, Ph.D., President

Hao Wu, PhD., Vice-President

Guo-Min Li, Ph.D., Secretary

Yingzi Yang, Ph.D., Treasurer

Yibin Kang, Ph.D.

Lei Li, Ph.D.

Xiangdong William Yang , MD, PhD.

Mingjie Zhang, Ph.D.

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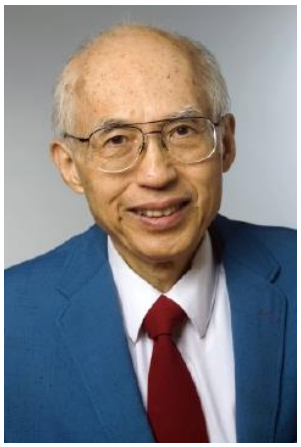
Pan Zheng, MD, PhD,

Yimin Zou, Ph.D.

CBIS 2016 Chengdu Meeting Sponsors



Dr. Ray Wu was a leading contributor to the development of the Sino-America overseas student program (CUSBEA) and to the establishment and expansion of CBIS.

We also acknowledge the following sponsors for supporting the 11th CBIS Biennial Conference

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http://www.cell-research.com/, http://www.nature.com/cr/index.html

CBIS 2016 Chengdu Conference Participants

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