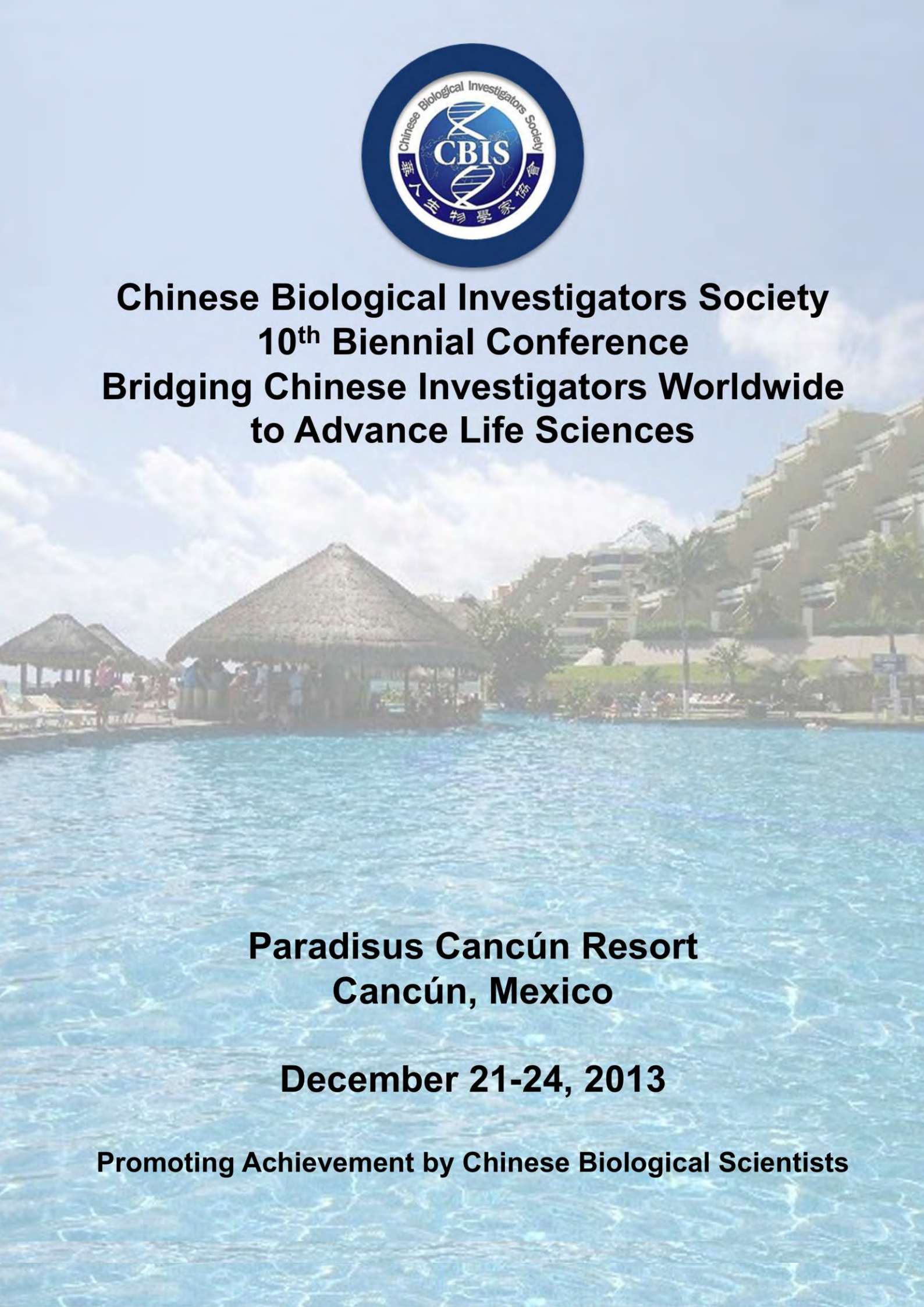

Chinese Biological Investigators Society
10th Biennial Conference
Bridging Chinese Investigators Worldwide to Advance Life Sciences

Paradisus Cancún Resort
Cancún, Mexico

December 21-24, 2013

Promoting Achievement by Chinese Biological Scientists

Welcome Message

Dear Colleagues and Friends,

On behalf of the CBIS board of directors, we are pleased to welcome you to our 10th Biennial Meeting in Cancún, Mexico. The Program Committee has prepared an outstanding line up that covers diverse topics in basic, technological, and translational research in life sciences. The program features 2 keynote speeches, 12 plenary presentations, and 11 concurrent sessions. In addition, there will be 3 special forums to celebrate the achievements of CBIS members, to share the skills in reviewing and handling grants and manuscripts, and to explore the 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 converting basic research into bio-products, 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 the late Dr. Ray Wu. We will present the CBIS Young Investigator Award to colleagues who are in the early career stages but have already made remarkable contributions in their respective fields.

We hope you will also enjoy Cancún's tropical weather and gorgeous beaches, as well as outstanding services and warm hospitality. There is much to appreciate about our host country Mexico, particularly its wonderful people and joyful culture. There are numerous activities in the surrounding areas for you and your family to enjoy.

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

Linheng Li
CBIS President 2011-2013

Pan Zheng
CBIS Program Committee Chair 2013

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Meeting Program at a Glance

Day 1, Dec. 21, 2013. Saturday. Arrival day

9:00 am – 6:00 pm	Meeting registration, ballot distribution (Main lobby)
6:00 pm – 7:00 pm	Reception for all meeting participants and family members (Pool area)

Day 2, Dec. 22, 2013. Sunday. (Morning in meeting rooms Greco, Dali and Goya combined)

8:00 – 8:30 am	Opening Ceremony and President Remark
8:30 – 9:30 am	**Keynote Presentation 1.** Stem Cells
9:30 – 10:30 am	**Society Lectures I**. Epigenetic Programming in Stem Cells
11:00 – 12:00 am	**Society Lectures II.** Epigenetic Regulation
1:30 – 3:30 pm	Breakout Sessions I – III (in rooms Picasso, Murillo, Miro)
	Breakout Session I. Neuroscience and Neurological Disorders (Picasso)
	Breakout Session II. Cardiovascular Medicine (Murillo)
	Breakout Session III. Metabolism and Diseases (Miro)
4:00 – 6:00 pm	Breakout Sessions IV – VI (in rooms Picasso, Murillo, Miro)
	Breakout Session IV. Immunology (Picasso)
	Breakout Session V. Stem Cells and Regenerative Medicine (Murillo)
	Breakout Session VI. Necrosis and Autophagy (Miro)
7:30 – 7:45 pm	Election Ballot Collection (Greco, Dali and Goya combined)
7:45 – 9:00 pm	**Panel Discussion I.** Achievement Celebration (Greco, Dali and Goya)
9:00 – 10:30 pm	**Panel Discussion II.** Academic Skill Development (Greco, Dali and Goya)
Session I:	How to deal with challenges of shrinking NIH funding and alternative opportunities?
Session II:	How to improve publication quality?

Day 3, Dec. 23, Monday. (Morning in meeting rooms Greco, Dali and Goya combined)

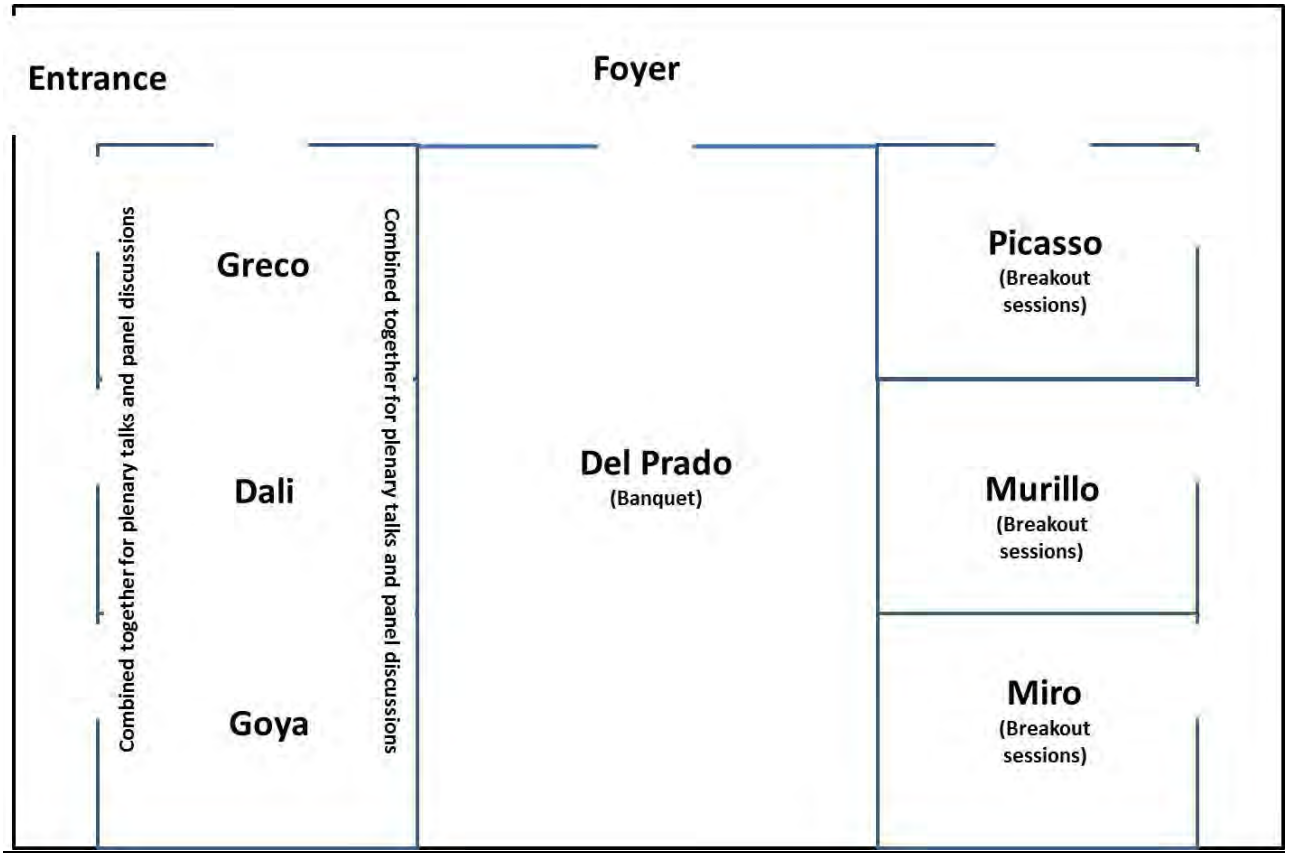
8:30 – 9:30 am	**Keynote Presentation 2**. Immunology and Cancer
9:30 – 10:30 am	**Society Lectures III.** Infection and Host Defense
11:00 – 12:00 am	**Society Lectures IV.** Neuronal Organization and Organ Size Control
1:30 – 3:30 pm	Breakout sessions VII – IX (in rooms Picasso, Murillo, Miro)
	Breakout Session VII. Epigenetic Regulation (Picasso)
	Breakout Session VIII. Cancer and Cancer Stem Cells (Murillo)
	Breakout Session IX. Cutting-edge Biotechnology (Miro)
4:00 – 5:40 pm	Breakout sessions X – XI (in rooms Picasso, Murillo)
	Breakout Session X. DNA Damage and Repair (Picasso)
	Breakout Session XI. Chemical Biology and Drug Screening (Murillo)
7:30 – 10:00 pm	**Panel Discussion III**. Special Forum on Entrepreneurship and Opportunities (Greco, Dali and Goya combined)

Day 4, Dec. 24, 2013. Tuesday, Christmas Eve. (Whole day in meeting rooms Greco, Dali and Goya)

8:30 – 9:30 am	**Society Lectures V.** Plant Biology
9:30 – 10:30 am	**Society Lectures VI.** Cancer and Immunity
11:00 am – 12:30 pm	Award Ceremony
12:45 – 2:00 pm	Special Luncheon, organized by Ray Wu Memorial Fund (Vento Terrace of Fuego Restaurant)
7:00 – 10:00PM	Closing Banquet and Christmas Party (Del Prado Banquet room)
	• Announce election results
	• Newly elected president speech

Day 5, Dec. 25, 2013. Wednesday, Departure day

Paradisus Cancún Conference Center Map



CBIS 10th Biennial Meeting Program

Day 1, Dec. 21, 2013. Saturday. Arrival day

9:00 am – 6:00 pm Meeting registration, ballot distribution (Main lobby)

6:00 pm – 7:00 pm Reception for all meeting participants and family members (Pool area)

Day 2, Dec. 22, 2013. Sunday. (Morning in meeting rooms Greco, Dali, and Goya combined)

8:00 – 8:30 am Opening ceremony and president remark
Linheng Li, CBIS president, Stowers Institute for Medical Research.

8:30 – 9:30 am **Keynote Presentation 1.** Stem Cells
Chairperson: Linheng Li

George Daley, Harvard Stem Cell Institute, Boston Children's Hospital, Harvard Medical School

"Stem cells in development and diseases"

9:30 – 10:30 am **Society Lectures I.** Epigenetic Programming in Stem Cells
Chairperson: Shijie Sheng, Wayne State University

9:30 – 10:00 am Haifan Lin, Yale University Stem Cell Center

"Life's tiniest architects: piRNAs guide a major epigenetic programming mechanism"

10:00 – 10:30 am Tian Xu, Yale University

"Cell-cell communication in regulation of growth and tissue size"

10:30 – 11:00 am Tea break (Foyer area at conference center)

11:00 – 12:00 am **Society Lectures II.** Epigenetic Regulation
Chairperson: Xin-Hua Feng, Baylor Medical School

11:00 – 11:30 am Yi Zhang, Harvard Medical School

"Mechanism and function of Tet-mediated 5mC oxidation"

11:30 – 12:00 pm Yang Shi, Harvard Medical School

"Investigation of the molecular mechanism of trans-generational epigenetic inheritance"

12:00 – 1:30 pm Lunch break

1:30 – 3:30 pm Breakout Sessions I – III (in rooms Picasso, Murillo, Miro)

Breakout Session I. Neuroscience and Neurological Disorders (Picasso)
Chairpersons: Xiangdong William Yang, University of California Los Angeles and Xiao-Jiang Li, Emory University

1:30 – 1:50 pm Ron Yu, Stowers Institute for Medical Research
"Molecular and circuit basis of mammalian courtship behavior"

1:50 – 2:10 pm Hongjun Song, Johns Hopkins University
"Circuit mechanisms regulating adult neural stem cells and neurogenesis"

2:10 – 2:30 pm Lin Mei, Medical College of Georgia
"Synapses, muscular dystrophy and schizophrenia"

2:30 – 2:50 pm Xiao-Jing Wang, NYU/Shanghai
"Neural circuit approach to mental disorders: function and mechanism"

2:50 – 3:10 pm Zhenyu Yue, Mount Sinai School of Medicine
"Autophagy mechanism in neurodegeneration"

3:10 – 3:30 pm Xiao-Jiang Li, Emory University
"Transgenic large animal models for neurodegenerative diseases"

Breakout Session II. Cardiovascular Medicine (Murillo)
Chairpersons: Eugene Chen, University of Michigan Health System and Yibin Wang, University of California Los Angeles

1:30 – 1:50 pm Hongliang Li, Wuhan University
"Genetic dissection of signaling mechanisms of heart failure"

1:50 – 2:10 pm Nanping Wang, PKU School of Medicine
"Cardiovascular effects of PPAR"

2:10 – 2:30 pm Jifeng Zhang, University of Michigan Health System
"Novel knockout, knockdown, and transgenic pig and rabbit models for cardiovascular research"

2:30 – 2:50 pm Zhong Wang, University of Michigan Health System
"Epigenetic regulation of cardiac stem cell differentiation"

2:50 – 3:10 pm Hong Wang, Temple University
"Epigenetic regulation in HHcy-induced vascular disease"

3:10 – 3:30 pm Xiao-feng Yang, Temple University
"Caspase-1 and endothelial cells"

Breakout Session III. Metabolism and Diseases (Miro)
Chairpersons: Yue Xiong, University of North Carolina Chapel Hill and Xiao-bo Zhong, University of Connecticut

1:30 – 1:50 pm Min Han, Howard Hughes Medical Institute and University of Colorado Boulder
"How lipid variants impact cell signaling for animal development and behavior?"

1:50 – 2:10 pm Kun-Liang Guan, University of California San Diego
"Nutrient signaling and autophagy regulation"

2:10 – 2:30 pm Dongsheng Cai, Albert Einstein College of Medicine
"Brain inflammation in obesity, diabetes, aging, and cardiovascular diseases"

2:30 – 2:50 pm Wen Xie, University of Pittsburgh
"Crosstalk between drug metabolism and energy metabolism"

2:50 – 3:10 pm Grace L. Guo, Rutgers University
"How liver and intestine coordinate lipid metabolism and liver regeneration"

3:10 – 3:30 pm Xiaochao Ma, University of Pittsburgh
"Metabolomic analysis reveals novel mechanisms of drug toxicity"

3:30 – 4:00 pm Tea break (Foyer area at conference center)

4:00 – 6:00 pm Breakout Sessions IV – VI (in rooms Picasso, Murillo, Miro)

Breakout Session IV. Immunology (Picasso)
Chairpersons: Yang Liu, Children's National Medical Center and Yang Xu, University of California San Diego

4:00 – 4:20 pm Yang Xu, University of California San Diego
"The immunogenicity of pluripotent stem cells"

4:20 – 4:40 pm Yang-Xin Fu, University of Chicago
"Targeting local tumor tissue for systemic immune protection"

4:40 – 5:00 pm Weiping Zou, University of Michigan
"Immune elements, cancer stemness and Knudson hypothesis"

5:00 – 5:20 pm Zhijian Chen, University of Texas Southwestern
"The dark side of DNA: innate immune sensing of cytosolic DNA"

5:20 – 5:40 pm Genhong Cheng, University of California Los Angeles
"Anti-Inflammatory gene program in balancing tissue protection and chronic infections"

5:40 - 6:00 pm Rongfu Wang, Houston Methodist Research Institute
"Innate immune signaling and inflammation in cancer"

Breakout Session V. Stem Cells and Regenerative Medicine (Murillo)
Chairpersons: Hongjun Song and Linzhao Cheng, Johns Hopkins University

4:00 – 4:20 pm Tao Cheng, Institute of Hematology, Chinese Academy of Medical Sciences/Peking Union Medical College
"Hematopoietic stem cells in response to leukemic cell expansion in vivo"

4:20 – 4:40 pm Qi-Qun Tang, Shanghai Medical School in Fudan University
"BMP4 on adipocyte development and metabolism"

4:40 – 5:00 pm Peng Jin, Emory University School of Medicine
"Cytosine modifications in stem cells and diseases"

5:00 – 5:20 pm Hongkui Deng, Peking University
"A new approach for cell reprogramming"

5:20 – 5:40 pm Kun Zhang, University of California San Diego
"Genetic integrity during cell fate reprogramming"

5:40 – 6:00 pm Linzhao Cheng, Johns Hopkins University School of Medicine
"Human cell reprogramming and genome engineering for blood disease modeling and treatment"

Breakout Session VI. Necrosis and Autophagy (Miro)
Chairpersons: Xin-Hua Feng, Zhejiang University and Qing Zhong, University of Texas Southwestern

4:00 – 4:20 pm Jiahuai Han, Xiamen University
"Molecular mechanisms of the switch between TNF-induced apoptosis and necrosis"

4:20 – 4:40 pm Francis Chan, University of Massachusetts Medical School
"Going up in flames: molecular regulation of necrosis in inflammatory diseases"

4:40 – 5:00 pm Qing Zhong, University of Texas Southwestern
"A novel signaling pathway involved in programmed necrosis"

5:00 – 5:20 pm Xue-Jun Jiang, Memorial Sloan-Kettering Cancer Center
"Dissecting the ULK complex dependency in mammalian autophagy"

5:20 – 5:40 pm Li Yu, Tsinghua University
"Autophagic lysosome reformation"

6:00 – 7:30pm Dinner

7:30 – 7:45 pm Election Ballot Collection (Greco, Dali, and Goya combined)

7:45 – 9:00 pm **Panel Discussion I.** Achievement Celebration (Greco, Dali, and Goya combined)
Chairpersons: Linheng Li and Weimin Zhong

Panelists:
- Liqun Luo, Stanford University
- Xinnian Dong, Duke University
- Xingwang Deng, Yale University
- Mingjie Zhang, Hong Kong University of Science and Technology
- Jiahuai Han, Xiamen University

9:00 – 10:30 pm **Panel Discussion II.** Academic Skill Development (Greco, Dali, and Goya)
Chairpersons: Xiao-Jing Wang, University of Denver

Session I: **How to deal with challenges of shrinking NIH funding and alternative opportunities?**

- Kun-Liang Guan, University of California San Diego
- Dihua Yu, MD Anderson Cancer Center
- Hua Lu, Tulane University
- Shijie Sheng, Wayne State University

Discussion topics:
- How to minimize the chance of unfair grant reviews (from choosing study sections to appeal)?
- Should a reviewer provide constructive comments to applicants?
- Opportunities for multi-PI/leadership grants to overcome funding cap for successful PIs.
- Alternative funding sources (foundation grants, fund raising efforts, collaborations with China).

Session II: **How to improve publication quality?**

- Xiao-Fan Wang, Duke University
- Yang Shi, Harvard Medical School
- Junjie Chen, MD Anderson Cancer Center
- Haifan Lin, Yale University
- Hao Wu, Harvard Medical School

Discussion topics:
- How to be a fair and constructive reviewer? How to be critical without being obnoxious?
- Should an editor help authors to improve their manuscripts and research?
- How to communicate with authors/editors transparently and efficiently?
- How to deal with unfair reviews, tips for how to exclude/suggest reviewers, appeal and rebuttal.

Day 3, Dec. 23, Monday. (Morning in meeting rooms Greco, Dali, and Goya combined)

8:30 – 9:30 am **Keynote Presentation 2.** Immunology and Cancer
Chairperson: Hao Wu, Harvard Medical School

Fred Alt, Boston Children's Hospital, Harvard Medical School

"Antibodies, Genome Instability and Cancer"

9:30 – 10:30 am **Society Lectures III.** Infection and Host Defense
Chairperson: Pan Zheng, Children's National Medical Center

9:30 – 10:00 am Chen Dong, Tsinghua University / MD Anderson Cancer Center

"Molecular control of T cell function"

10:00 – 10:30 am. Yang Liu, Children's National Medical Center.

"Sialoside-based pattern recognition and self-nonself discrimination in innate immunity"

10:30 – 11:00 am Tea break (Foyer area at conference center)

11:00 – 12:00 am **Society Lectures IV.** Neuronal Organization and Organ Size Control
Chairperson: Mingjie Zhang, Hong Kong University of Science and Technology

11:00 – 11:30 am: Liqun Luo, Stanford University

"Mapping olfaction"

11:30 – 12:00 pm: Duojia Pan, Johns Hopkins University

"Hippo signaling in development and cancer"

12:00 – 1:30 pm Lunch break

1:30 – 3:30 pm Breakout sessions VII – IX (in rooms Picasso, Murillo, Miro)

Breakout Session VII. Epigenetic Regulation (Picasso)
Chairpersons: Yujiang Shi, Harvard University and Yali Dou, University of Michigan

1:30 – 1:50 pm Xin Chen, Johns Hopkins University
"Epigenetic inheritance during asymmetric cell divisions"

1:50 – 2:10 pm Fei Lan, Fudan University
"A novel methylation site on histone H3"

2:10 – 2:30 pm Lingling Chen, SIBS Chinese Academy of Sciences
"Life without A tail: new long noncoding RNAs from excised introns"

2:30 – 2:50 pm Jinrong Min, University of Toronto
"Structural and functional studies of Chromatin binding proteins"

2:50 – 3:10 pm Zhiguo Zhang, Mayo Clinic
"Epigenetic inheritance: functions of Rtt proteins in nucleosome assembly"

3:10 – 3:30 pm Yali Dou, University of Michigan
"Targeting MLL1 histone methyltransferase activity in leukemia".

Breakout Session VIII. Cancer and Cancer Stem Cells (Murillo)
Chairpersons: Dihua Yu, MD Anderson Cancer Center

1:30 – 1:55 pm Zhimin James Lu, MD Anderson Cancer Center
"Warburg effect and beyond"

1:55 – 2:20 pm Yuan Zhu, University of Michigan
"Clonal evolution from neural stem cells to malignant glioma cells"

2:20 – 2:45 pm Wei Xu, University of Wisconsin
"CARM1 methylates chromatin remodeling factor BAF155 to enhance tumor progression and metastasis"

2:45 – 3:10 pm Yong-Jun Liu, Baylor Institute for Immunology Research.
"Targeting pDC and OX40 for cancer therapy"

Breakout Session IX. Cutting-edge Biotechnology (Miro)
Chairpersons: Tian Xu, Yale University

1:30 – 1:50 pm Jinsong Li, Shanghai Institutes for Biological Sciences, Chinese Academy of Sciences
"Cellular reprogramming and embryonic development"

1:50 – 2:10 pm Heng Zhu, Johns Hopkins University School of Medicine
"DNA methylation presents distinct binding sites for human transcription factors"

2:10 – 2:30 pm Feng Zhang, Massachusetts Institute of Technology
"Genome engineering: technologies and applications"

2:30 – 2:50 pm Jinmin Gao, University of Texas Southwestern
"UPS nanotechnology in biology and medicine"

2:50 – 3:10 pm Fuchou Tang, Peking University
"Preimplantation genomic screening by single cell genome sequencing"

3:10 – 3:30 pm Yanyi Huang, Peking University
"Microfluidics facilitated single cell genome sequencing"

3:30-4:00 pm Tea break (Foyer area at conference center)

4:00 – 5:40 pm Breakout sessions X – XI (in rooms Picasso, Murillo, Miro)

Breakout Session X. DNA damage and Repair (Picasso)
Chairpersons: Jun Qin, Baylor Medical College and Lei Li, Baylor Medical College

4:00 – 4:20 pm Chunying Du, University of Cincinnati
"Apoptotic proteins in DNA damage response"

4:20 – 4:40 pm Yi Wang, Baylor College of Medicine
"Mapping DNA damage-induced complexome"

4:40 – 5:00 pm Lee Zou, Harvard Medical School
"A new view of the ATR checkpoint"

5:00 – 5:20 pm Guo-Min Li, University of Kentucky College of Medicine
"New clues to cancer etiology: genome instability caused by abnormal epigenetic mark"

5:20 – 5:40 pm Junjie Chen, University of Texas MD Anderson Cancer Center
"Protein-protein interaction network in DNA damage response and tumorigenesis"

Breakout Session XI. Chemical Biology and Drug Screening (Murillo)
Chairpersons: Jun Liu, Johns Hopkins and Sheng Ding, University of California San Francisco

4:00 - 4:20 pm Sheng Ding, University of California San Francisco
"A chemical approach to controlling cell fate"

4:20 - 4:40 pm Zhong-Yin Zhang, Indiana University
"Drugging the undruggable: Therapeutic potential of targeting protein tyrosine phosphatases"

4:40 - 5:00 pm Jikai Liu, Yunnan Institute of Botany, Chinese Academy of Sciences
"Discovering new drug leads and chemical probes from fungi in southwest China"

5:00 - 5:20 pm Xin Xie, SIMM, Chinese Academy of Sciences
"Finding new tricks for old GPCR modulators - drug repositioning in autoimmune diseases"

5:20 - 5:40 pm Jun Liu, Johns Hopkins University School of Medicine
"Exploring existing drug space for novel biological activities"

06:00 – 07:30 pm Dinner

07:30 – 10:00 pm **Panel Discussion III.** Special Forum on Entrepreneurship and Opportunities (Greco, Dali and Goya combined)
Chair: Guo-Liang Yu and Jing-Shan (Jennifer) Hu

A short presentation (10 min + 2 min Q&A) from each speaker and a panel discussion.

Speakers and Panelists:

X. Charlene Liao, Project Team Leader, Portfolio Management & Operations, Genentech**. –** liao.charlene@gene.com
"Targeting membrane IgE with an antibody"

Alex (Yue) Wu, CEO, Crownbio Inc. – awu@crownbio.com
"From a CRO to a pipeline company, the evolving entrepreneurship"

Jing-shan (Jennifer) Hu, VP, Bayer Health Care. – Jennifer – hu@bayer.com
"What is Bayer looking for in China?"

Yingfei Wei, CEO, Elixirin Corp (former CSO, 3Sbio). – yingfei.wei@gmail.com
"Experience of taking a Chinese biotech company to NASDAQ"

Jiwu Wang, CEO, Allele Biotechnology, Inc. – jiwuwang@allelebiotech.com
"mRNA-iPSCs, nano Antibodies, and Superresolution Imaging--Technologies That Drive Innovation for Better Life Sciences"

Guo-Liang Yu, Chairman, Quantitative Health, Corporation. – guoliang.yu@gmail.com
"Biotechnology is for everyone: can we live 100 years?"

Topics for panel discussion:
1. What are the key drivers for today healthcare? How do various players position themselves in such an environment: large pharma vs. small biotech; drug discovery vs. diagnostics; academic vs. industry, platform vs. products?

2. What are the important roles China play? How do these important elements evolve over time? As Chinese professionals, how do we position?
3. Translating scientific innovations to people's life and health. What are the opportunities?
4. Entrepreneurship, drivers, nurturing and challenges.

Day 4, Dec. 24, 2013. Tuesday, Christmas Eve. (in meeting rooms Greco, Dali, and Goya combined)

8:30 – 9:30 am	**Society Lectures V.** Plant Biology Chairperson: Weimin Zhong, Yale University
08:30 – 09:00 am	Xinnian Dong, Duke University
	"Dynamic regulation of plant immune responses"
09:00 – 09:30 am	Xingwang Deng, Yale University
	"Getting out of darkness, a live-and-death decision-making during Arabidopsis seedling development"
09:30 – 10:30 am	**Society Lectures VI.** Cancer and Immunity Chairperson: Hao Wu, Harvard Medical School
09:30 – 10:00 am	Dihua Yu, MD Anderson Cancer Center
	"Tackling new challenges in cancer therapy"
10:00 – 10:30 am	Feng Shao, National Institute of Biological Sciences, Beijing
	"Biochemical dissection of bacterial virulence and macrophage innate immunity"
10:30 – 11:00 am	Tea break (Foyer area at conference center)
11:00 am – 12:30 pm	Award Ceremony
11:00am – 11:45 am	Ray Wu Award: Xiaofan Wang, Duke University
	"Find the best ways for one to make contributions to science and education in China".
	Ray Wu Award: Haifan Lin, Yale University
11:45am – 12:30 pm	CBIS Young Investigator Award: Yibin Kang, Princeton University
	"How tumor cells develop metastatic traits"
	CBIS Young Investigator Award: Feng Shao, National Institute of Biological Sciences, Beijing
12:45 – 2:00 pm	Special Luncheon, organized by Ray Wu Memorial Fund Chairperson: Xiao-Hong Sun, Oklahoma Medical Research Foundation (Vento Terrace of Fuego with existing buffet at NAOs)
2:00 – 7:00 pm	Free time
7:00 – 10:00PM	Closing Banquet and Christmas Party (Del Prado Banquet room at conference center) • Announce election results • Newly elected president speech

Day 5, Dec. 25, 2013. Wednesday, Departure day

Bon Voyage!

Keynote speakers

George Q. Daley, M.D., Ph.D. is the Samuel E. Lux IV Professor of Hematology/Oncology and the Director of the Stem Cell Transplantation Program at Boston Children's Hospital, Professor of Biological Chemistry and Molecular Pharmacology and Pediatrics at Harvard Medical School, and an investigator of the Howard Hughes Medical Institute. He has served the International Society for Stem Cell Research (ISSCR) as past-President ('07-'08), led the special task forces that produced the ISSCR Guidelines for Stem Cell Research (2006) and Clinical Translation (2008), and is currently the ISSCR Clerk. Dr. Daley received his Bachelor's degree *magna cum laude* from Harvard University (1982), a Ph.D. in biology from MIT (1989), and the M.D. from Harvard Medical School *summa cum laude* (1991). Dr. Daley has been elected to the Institute of Medicine of the National Academies, American Society for Clinical Investigation, American Association of Physicians, American Pediatric Societies, American Academy of Arts and Sciences, and American Association for the Advancement of Science, and has received the NIH Director's Pioneer Award, the Judson Daland Prize from the American Philosophical Society, the E. Mead Johnson Award from the American Pediatric Society, and the E. Donnall Thomas Prize from the American Society for Hematology. Dr. Daley's research exploits mouse and human disease models to identify mechanisms that underlie cancer and blood disease.

Frederick W. Alt, Ph.D. is the Charles A. Janeway Professor of Pediatrics and the Director of the Program in Cellular and Molecular Medicine at Boston Children's Hospital, Professor of Genetics and Pediatrics at Harvard Medical School, and Investigator of the Howard Hughes Medical Institute. Dr. Alt received his Ph.D. in Biology from Stanford University in 1977 with Robert Schimke and performed his postdoctoral work at MIT with David Baltimore. Dr. Alt has been elected to the U.S. National Academy of Sciences, the American Academy of Arts and Sciences, the American Academy of Microbiology, and the European Molecular Biology Organization. He has received the Clowes Memorial Award from the American Association of Cancer Research; the Rabbi Shai Shacknai Prize from The Hebrew University, the Pasarow Foundation Prize for Extraordinary Achievement in Cancer Research, the Leukemia & Lymphoma Society de Villiers International Achievement Award, the Irvington Institute Immunology Award, the National Cancer Institute Alfred K. Knudson Award for pioneering contributions that have revolutionized the field of Cancer Genetics, the American Association of Immunologists AAI-Huang Meritorious Career Award, the William B. Coley Award from the Cancer Research Institute, and the Novartis Basic Immunology Prize for his discoveries on B cell development and antigen responses. Dr. Alt has mentored over 100 students and research fellows, many of whom have become leaders in immunology, genetics, or cancer biology and has received the American Association of Immunologists Excellence in Mentoring Award. The Cancer Research Institute (New York) annually presents the Frederick W. Alt Award for New Discoveries in Immunology. The broad focus of Dr. Alt's research is the elucidation of mechanisms that maintain genomic stability in mammalian cells. More specifically, the lab studies V(D)J recombination in developing B and T lymphocytes and IgH heavy chain class switch recombination (CSR) and somatic hypermutation in mature B lymphocytes.

CBIS Announces Recipients of 2013 Awards

The Chinese Biological Investigators Society (CBIS) is pleased to announce the recipients of this year's Ray Wu Award and Young Investigator 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. Xiao-Fan Wang, Duke University, for his pioneering research in the field of TGF-β Signaling and for unparalleled efforts in shaping policy and promoting research in life sciences in China.

Dr. Haifan Lin, Yale University, for seminal contributions to the field of Stem Cell Biology and RNA Biology and for efforts in promoting life science research in China.

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. Yibin Kang, Princeton University, for contributions to the field of Cancer Biology, particularly for elucidating molecular mechanisms of cancer metastasis.

Dr. Feng Shao, National Institute of Biological Sciences, Beijing, for contributions to the field of Pathogen-Host Interaction, particularly for revealing novel biochemical mechanisms underlying bacterial virulence and host innate immunity.

Awardee Biography

Xiao-Fan Wang is the Donald and Elizabeth Cooke Professor of Experimental Oncology and a Professor of Pharmacology and Cancer Biology at Duke University Medical Center. He earned his B.S. degree from Wuhan University in 1982 and his Ph.D. degree from University of California at Los Angeles in 1986. After postdoctoral training at Massachusetts Institute of Technology, he joined the faculty of Duke University Medical Center as an Assistant Professor in 1992. He did pioneering research in cellular signaling, DNA repair and cancer metastasis. In particular, he identified the genes encoding the type I, type II and type III receptors for transforming growth factor beta (TGF-β) and further elucidated how this essential signaling pathway works as well as how it interacts with other major signaling pathways. Moreover, through his devotion and tireless efforts, Dr. Wang has been highly effective in China in shaping and implementing policy changes that have greatly improved the overall research environment, particularly in distributing and managing major research funds, in establishing fair and transparent evaluation systems for individual scientists and institutions, and in recruiting overseas talents. His efforts have also led to significant increases in stipends for graduate students and in the awareness of the importance of ethics and research integrity. He has received many awards, and his other academic activities include, among others, serving on the editorial boards of a number of scientific journals, such as an Associate Editor for the Journal of Biological Chemistry. He is also the past President of Society of Chinese Bioscientists in America (SCBA).

Haifan Lin is a Professor of Cell Biology, of Genetics and of Ob-Gyn and reproductive Sciences, and the Director of the Stem Cell Center at Yale University. He received his B.S. degree from Fudan University in 1982 and his Ph.D. degree from Cornell University in 1990. Following postdoctoral research at the Carnegie Institution of Washington, he joined the faculty of Duke University Medical Center in 1994, where he rose to the rank of Full Professor. He moved to Yale in 2006 to establish the Yale Stem Cell Center. Dr. Lin has made seminal contributions in elucidating the biology of stem cells, including providing experimental proof for the stem cell niche theory by demonstrating its existence and functional importance, the discovery of the Argonuate/PIWI gene family and their essential function in stem cell self-renewal and germline development, as well as the discovery and functional analysis of a novel class of non-coding small RNAs called PIWI-interacting RNAs (piRNAs), which was hailed by the *Science* magazine as one of the ten scientific breakthroughs of 2006. His findings have contributed significantly to a greater appreciation of the function of so-called “junk DNA” in the genome and a better understanding of epigenetic pathways. Dr. Lin has also devoted efforts in China by participating in peer review for distributing research funds, in evaluating research institutions, in mentoring graduate students and junior scientists, and in improving college and high school education. He has received many awards, and his other scientific activities include serving on the Editorial Boards of multiple scientific journals, and the Board of Directors or the Advisory Council of many societies, research institutions, and foundations, such as the Chinese Biological Investigators Society (2002-2008) and the International Society for Stem Cell Research (ISSCR, 2009- present).

Yibin Kang is the Warner-Lambert/Parke-Davis Professor of Molecular Biology at Princeton University. He obtained his B.S. degree from Fudan University in 1995 and his Ph.D. degree from Duke University in 2000. After completing postdoctoral training at the Memorial Sloan-Kettering Cancer Center, he joined the faculty of Princeton University as an Assistant Professor in 2004 and rose through the ranks to become endowed chair Professor in 2010. Dr. Kang has made fundamental discoveries in cancer biology, particularly in the area of tumor-stromal interaction in organ-specific metastasis of breast cancer. He established a series of elegant mouse models to dissect the molecular dynamics of tumor-stromal interaction as cancer cells disseminate to bone and other organs, break out of dormancy and establish overt lesions. These findings have not only elucidated the molecular mechanisms underlying cancer metastasis but also established new targets for therapeutic intervention. In recent years, he has expanded his research to the early phase of breast cancer progression and has uncovered the molecular connection between cell fate regulation, epithelial plasticity and metastatic progression. Dr. Kang is the recipient of several prestigious awards, including a Department of Defense Era of Hope Scholar Award (2006), the Vicek Prize for Creative Promise in Biomedical Sciences (2011), and the AACR Award for Outstanding Achievements in Cancer Research (2012).

Feng Shao is an Investigator at the National Institute of Biological Sciences, Beijing. He obtained his B.S. degree from Peking University in 1996 and his Ph.D. degree from University of Michigan at Ann Arbor in 2003. After postdoctoral trainings at the University of California, San Diego, and Harvard Medical School, he joined the National Institute of Biological Sciences as an Assistant Investigator in 2005. He has made fundamental discoveries regarding molecular mechanisms of bacterial infection and host innate immune defense. He discovered several novel enzymatic activities used by bacterial type III secretion virulence effectors to induce novel posttranslational modifications on crucial host signaling proteins. His work on the macrophage inflammasome pathway and its role in anti-bacterial innate immunity has led to the identification of the long-sought cytosolic inflammasome receptors for bacterial flagellin as well as the type III secretion apparatus. His discoveries are not only highly significant for understanding bacterial pathogenesis but also have led to new understandings of eukaryotic signal transduction systems and the innate immune system in mammals. He is the recipient of several prestigious awards, including the International Early Career Award from the Howard Hughes Medical Institute (2012), the DGHM Lecture Award from the German Society for Hygiene and Microbiology (2012), and the Young Investigator Award from the Protein Society (2013).

CBIS 2013 Cancún Meeting Abstracts

1. Cai, Dongsheng, dongsheng.cai@einstein.yu.edu, Albert Einstein College of Medicine
The hypothalamus is a converging point that integrates metabolic, neural, neuroendocrine, and neuroimmune signals to affect physiology. My research is to investigate the role of neural dysregulations, in particular in terms of neural inflammation in the development of metabolic syndrome and aging. Our observations demonstrated that the hypothalamus contains adult neural stem cells, and IKK/NF-kB activation affects the fate of these cells and cause disease consequences related to aging and metabolic dysfunctions. Taken together, we established several conceptual models addressing the central mechanism of metabolic diseases and aging, and identified the mechanisms that are mediated by the integrative actions of neuroendocrine and immunity, and generated strategies for combating aging-related and metabolic diseases through targeting the neural inflammatory molecular pathways.

2. Chan, Francis, Francis.Chan@umassmed.edu, University of Massachusetts Medical School
The focus of my research is to elucidate the signaling pathway of necrosis and its functions in healthy and disease states. Our group was one of the first groups to identify an obligate role for RIP1 and RIP3 in TNF-induced necrosis. We also identified the first class of viral inhibitors that target cell death by necrosis and to demonstrate a role for RIP kinase dependent necrosis in innate immune responses against different pathogens. Our long-term goal is to understand the molecular regulation of necrosis in inflammatory diseases, tissue homeostasis and cancers.

3. Chen, Eugene Yuqing, echenum@umich.edu, University of Michigan
The long-term goal of Dr. Chen"s research program in cardiovascular medicine is elucidating the molecular basis of obesity/diabetes-induced cardiovascular diseases (CVD) and stroke, and on developing new drugs/technologies to study and treat diabetes and CVD. A major bottleneck in cardiovascular research and drug development is the lack of larger animal models that more accurately simulate human disease in the pre-clinical stages of drug discovery and in the analysis of disease mechanisms. Although the study of the cardiovascular system has benefited significantly from the use of gene-targeted and transgenic mouse models, small rodents do not accurately reflect human cardiovascular physiology. To generate more appropriate and useful animal models for better mimicking the human cardiovascular system, it would be beneficial to explore larger mammalian models for CVD. Because of the compelling need to address this bottleneck in cardiovascular research and drug development, we have built a leading research team with multiple approached and expertise in the world to produce gene-targeted (knockout) and transgenic rabbits/pigs as CVD model animals.

4. Chen, Junjie, jchen8@mdanderson.org, The University of Texas MD Anderson Cancer Center
DNA double strand breaks (DSBs) are predominantly repaired by NHEJ and HR pathways in mammalian cells. It is believed that which pathway to use for DSB repair is predominantly 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 recently that RIF1 is a downstream effector of 53BP1 and participates in 53BP1-dependent inhibition of end resection. We are now investigating mechanistically how 53BP1, RIF1 and others function at DSB sites and the detailed roles of BRCA1 in DSB repair.

5. Chen, Ling-Ling, linglingchen@sibcb.ac.cn, Shanghai Institutes for Biological Sciences, CAS
One goal in my laboratory is to discover new RNA species and new modes of gene regulation. We have recently used deep sequencing to explore the repertoire of non-polyadenylated RNAs from human cells and have identified hundreds of long noncoding RNAs (lncRNAs) that are derived from excised introns. Such molecules would be expected to lack both 5' cap structures and 3' poly(A) tails. Further studies revealed there are at least two mechanisms for the processing of these excised introns, leading to the accumulation of two new classes of lncRNAs in the nucleus. I will discuss the processing mechanisms and functional implications of these RNAs discovered in the lab.

6. Chen, Xian, xianc@email.unc.edu, University of North Carolina-Chapel Hill
Developing and applying novel quantitative proteomics to dissect and discover new pathways involved in signal transduction and epigenetic regulation

7. Chen, Xin, xchen32@jhu.edu, Johns Hopkins University
We recently found that during the asymmetric division of Drosophila male germline stem cell (GSC), the preexisting histone 3 (H3) are selectively segregated to the GSC whereas newly synthesized histones during DNA replication are enriched in the differentiating daughter cell. Our studies provide the direct

evidence that stem cells retain preexisting canonical H3 during asymmetric cell divisions in vivo, which may contribute to maintain their epigenetic memory. We are continuing this exciting project by asking: (1) What are the molecular mechanisms underlying asymmetric segregation pattern of H3? (2) What are the consequences of the mis-regulation of such an inheritance mode? Our studies will shed light on a long-standing question regarding whether and how stem cells retain their epigenetic memory.

8. Chen, Yabing, ybchen@uab.edu, University of Alabama
Molecular regulation of vascular calcification

9. Chen, Zhijian, zhijian.chen@utsouthwestern.edu, University of Texas Southwestern Medical Center
Innate immune sensing and signaling of cytosolic DNA and RNA

10. Chen, Zhongping, z2chen@uci.edu, University of California Irvine
Optical coherence tomography (OCT), multiphoton microscopy (MPM), coherent anti-stokes Raman scattering (CARS) imaging, and photoacoustic tomography (PAT) are imaging technologies that have found many biomedical applications. My group's research focuses on the development of fiber based endoscopic OCT, MPM CARS, and PAT system that enable translation of these technology for in vivo and clinical applications. I will report several on-going research projects in my laboratory that focus on translating these technologies to solve specific clinical problems, including diagnosis of cancers in the gastrointestinal and respiratory tracts, and detection of vulnerable plaque in cardiovascular diseases. In addition, advances in multimodality endoscopic/intravascular imaging that combines morphological contrast of OCT with molecular contrast of MPM, CARS, PAT etc., will also be discussed.

11. Cheng, Tao, chengt588@gmail.com, Institute of Hematology, Chinese Academy of Medical Sciences
A direct cause for the death of leukemia patients is bleeding or infection, largely due to the interrupted generation of normal blood and immune cells from hematopoietic stem cells (HSCs) and hematopoietic progenitor cells (HPCs). However, the mechanism for which normal hematopoietic cells are overtaken by emerging leukemia cells within a diseased host is poorly understood. To systematically interrogate this important problem, we developed a simple, robust and clinically relevant method in which primary leukemia cells can be propagated in non-manipulated recipients, and then applied mathematical modeling in conjunction with gene expression profiling and functional validations. With our improved models and interdisciplinary approaches, this study has documented broad yet distinct suppression of the leukemic environment on different subsets of HSCs and HPCs. Moreover, our results also revealed that Egr3, Hey1 and Maff play distinct roles in inhibiting or protecting HSCs during leukemia cell outgrowth, thereby having implications for new therapeutic strategies against leukemia.

12. Cheng, Genhong, gcheng@mednet.ucla.edu, University of California Los Angeles
Research in Genhong Cheng's laboratory at UCLA is aimed at the process of innate and adaptive immune responses to infectious and inflammatory diseases. Our recent studies have defined a major type I interferon-mediated anti-inflammatory gene program, which proceeds through IFN-induced upregulation of IL-27, IL-27-induced upregulation of IL-10 and IL-10 induced suppression of inflammatory genes. This gene program plays a critical role in protecting host from tissue injuries during acute pathogen infections as well as inflammatory diseases such as Experimental Allergic Encephalomyelitis (EAE) and inflammatory bowel diseases. On the other hand, sustained activation of the IFN-IL-27-IL-10 gene program can also lead to influenza pneumonia as well as chronic infectious diseases such as Mycobacterium leprae and chronic MCMV viral infection.

13. Cheng, Linzhao, lcheng2@jhmi.edu, Johns Hopkins University
Human stem cell biology and engineering; Human genome biology and engineering; Human genome editing and targeting. Blood disease modeling and treatment; cell engineering and transplantation; transfusion

14. Cheng, Shiyuan, shiyuan.cheng@northwestern.edu, Northwestern University
Our research interests focus on the molecular mechanisms of human cancer tumorigenesis, progression, invasion/metastasis and angiogenesis. We have been investigating oncogenic receptor tyrosine kinase signaling, their mediators including guanine nucleotide exchange factors (GEFs), activators for Rac1 and Cdc42 in human glioblastomas and breast cancers. Glioblastomas are the most common malignant tumors in the brain. Breast cancer is the most common cancer in women. Recently, we started to study glioma stem cells that were derived from clinical glioma tumor tissues. We are also interested in developing novel approaches for cancer therapies using novel anti-tumor and anti-angiogenic inhibitors in combination with non-invasive evaluation methods.

15. Cui, Rutao, rutaocui@bu.edu, Boston University
I have a broad background in the field of UV-induced DNA repair, pigment and melanoma research.

16. Deng, Hongkui, hongkui_deng@pku.edu.cn, Peking University
Stem cell research & animal models

17. Deng, XingWang, xingwang.deng@yale.edu, Yale University
Over the last twenty years of independent research career at Yale University, Prof. Deng's research mainly centers on the molecular and biochemical mechanism for how light regulates plant development. Using Arabidopsis model, his group has identified a dozen of pleiotropic COP/DET/FUS loci responsible for mediating light control of plant development. They further demostrated that those COP/DET/FUS genes encoding proteins constitute three protein machineries involved in targeting photomorphogenesis-promoting factors for degradation by the 26S proteasome, in response to external stimuli. Two of those complexes defined two novel multiproetin complex E3 ligases (COP1 complexes and CDD complex) and the other is the well-conserved COP9 signalosome, a general eukaryotic Cullin-based E3 ligase regulator with de-rubbylation activity. Professor Deng lab are now applying both molecular/genetic and genomic approaches to further analyze the action mechanism of those novel cellular machineries, which are conserved among all multicellular organisms. Those discoveries made in plant model in Deng lab have significant implications in general biology research as well as those studies related to human health. Recently work from many groups including Deng lab have show the general conservation of those three complexes in all multicellular organism and their fundamental roles in a variety of developmental and cellular processes. Over the last few years, Deng lab has initiated another area of research: genome-wide analysis of the relationship between epigenetic modifications and chromatin structure/gene expression in rice and their role in rice heterosis. Deng lab has established several genomic platforms to examine the molecular and genomic basis of rice heterosis. In recent years, Dr Deng has also involved in Agribiotechnology Research and Development activities in China.

18. Ding, Sheng, sheng.ding@gladstone.ucsf.edu, Gladstone/University of California San Francisco
Recent advances in stem cell biology may make possible new approaches for the treatment of a number of diseases. A better understanding of molecular mechanisms that control stem cell fate as well as an improved ability to manipulate them are required. Toward these goals, we have developed and implemented high throughput cell-based screenings of chemical libraries, and identified and further characterized small molecules that can control stem cell fate in various systems. This talk will provide latest examples of discovery efforts in my lab that have advanced our ability and understanding toward controlling stem cell fate, including self-renewal, survival, differentiation and reprogramming of stem cells.

19. Dong, Chen, cdong@mdanderson.org, MD Anderson Cancer Center, Tsinghua University
We have been studying the function and regulation of CD4+ T lymphocytes in immune responses and diseases. Our work has led to the identification of Th17 differentiation lineage. I will discuss the genetic control of another T cell subset, T follicular helper cells, which functions to promote germinal center reaction and humoral immunity. The results from our studies have implications in infectious and autoimmune diseases as well as B cell lymphoma.

20. Dong, Xinnian, xdong@duke.edu, Duke University
Xinnian Dong's lab identified the signaling pathway involved in the perception and the transduction of the plant immune signal, salicylic acid, more recently, discovered surprising connections between plant defense with the circadian clock and with the DNA repair machinery.

21. Dou, Yali, yalid@med.umich.edu, University of Michigan
Our broad objectives are to study the epigenetic regulation of transcription. Epigenetics is used broadly to refer to heritable changes in cellular phenotypes without alterations in DNA sequence. It has emerged that histones, the basic building blocks of a eukaryotic genome, are major carriers of epigenetic information. The histone posttranslational modifications, 'written' by chromatin modifying enzymes, often define specific functional states in cells in response to extra- and intra-cellular signaling. The 'histone code' is proposed to regulate various cellular processes including transcription, cell cycle progression, DNA damage repair and replication. Overwhelming evidence also link epigenetic regulation to cell differentiation during development. The research in our lab has focused on three general but related areas in epigenetic regulation: 1) Biochemical characterization of histone methyltransferases and histone acetyltransferases 2) Develop small molecular inhibitors for the MLL methyltransferase activity. 3) Study epigenetic regulation in mouse embryonic stem cells (mESC).

22. Feng, GenSheng, gfeng@ucsd.edu, University of California, San Diego
Most recent studies in this and other labs have revealed dual roles of genes in promoting and suppressing hepatocellular carcinoma (HCC). Deciphering the "paradoxical" effects of these genes may provide a fresh view on the fundamental mechanisms of carcinogenesis, which can lead to development of mechanism-

based therapeutics for HCC. Our current focuses are on delineating genetic and epigenetic interactions and cell-cell communications that drive hepatooncogenesis. We are very interested in isolating and characterizing cancer stem cells, CSCs (or tumor-initiating cells, TICs) in the liver, and in dissecting the dynamic interplay between tumors and hepatic microenvironment. Efforts are also being devoted to elucidation of metabolic changes in tumor cells and to search of biomarkers for early diagnosis of liver cancers.

23. Feng, Qingping, qfeng@uwo.ca, Western University
Nitric oxide was initially identified as a vasodilator. We now know that its biological effects go far beyond the vascular system. Over the last decade, we have been studying the role of nitric oxide in adult heart physiology and embryonic heart development. Our studies have demonstrated that neuronal nitric oxide synthase (nNOS or NOS1) protects against ventricular arrhythmia after myocardial infarction (MI) while inducible nitric oxide synthase (iNOS or NOS2) contributes to the development of heart failure post-MI. We further demonstrated that endothelial nitric oxide synthase (eNOS or NOS3) plays a critical role in embryonic heart development and morphogenesis of coronary arteries and cardiac valves.

24. Feng, Xinhua, xfeng@bcm.edu, Baylor College of Medicine
TGF-beta; Protein phosphatase; Cell signaling; Cancer; Stem cells

25. Fu, Yang-xin, yfu@uchicago.edu, The University of Chicago
Antibodies (Abs) preferentially targeting oncogenic receptors are increasingly used for cancer therapy, but tumors often develop Ab-resistance after prolonged and costly treatment. Here, we arm Ab with IFN; as a next generation of biologics, is far superior to first generation of Ab for controlling Ab-resistant tumors independent of various resistance mechanisms. This new strategy controls Ab resistance by re-activating and re-bridging suppressed innate and adaptive immunity. Mechanistically, Ab-IFN therapy primarily targets intra-tumoral dendritic cells (DCs), greatly improving their CTL cross-priming function inside the tumor microenvironment. Blocking PDL-1 inside tumor induced by Ab-IFN treatment overcomes treatment-acquired resistance and completely eradicates larger established tumors. Therefore, this study establishes a next-generation Ab-based immunotherapy that targets and eradicates established Ab-resistant tumors.

26. Gao, Jinming, jinming.gao@utsouthwestern.edu, University of Texas Southwestern Medical Center
Biomaterials in drug delivery.

27. Gong, Ming, ming.gong@uky.edu, University of Kentucky
Vascular Clock Gene and Blood Pressure Circadian Rhythm in Normal and Diabetes Blood pressure exhibits a 24-hour circadian rhythm. Loss of BP circadian rhythm has been found in up to 75% of human diabetic patients and is associated with increased risks of cardiac, kidney, and vascular target organ injuries. Clock genes are expressed in vascular tissues; we and others have found their circadian oscillations were disrupted in diabetes. However, the physiological functional significance of vascular clock gene and the mechanisms underlying their dysfunction in diabetes are unknown. Moreover, to what extent the vascular clock gene dysfunction contributes to the blood pressure circadian rhythm disruption in diabetes remain unknown. Our current study aims to address these specific questions.

28. Gu, Liya, lgu0@uky.edu, University of Kentucky
DNA Damage and repair.

29. Guan, Junlin, jlguan@umich.edu, University of Michigan
Breast cancer; autophagy; normal and cancer stem cells; tyrosine kinase signaling

30. Guan, Kunliang, kuguan@ucsd.edu, University of California San Diego
AMPK and mTOR in nutrient signaling and autophagy regulation Kun-Liang Guan Department of Pharmacology University of California, San Diego Autophagy is a stress response protecting cells from unfavorable conditions, such as nutrient starvation. The class III phosphatidylinositol-3 kinase, Vps34, forms multiple complexes and regulates both intracellular vesicle trafficking and autophagy induction. The cellular energy sensor AMPK and key growth controller mTOR play major roles in autophagy regulation. We show that AMPK regulates different VPS34 complexes to induce autophagy in response to energy stress. Similarly, mTOR inhibits autophagy specific Vps34 complex to suppress autophagy under amino acid sufficiency. Our study reveals an intricate molecular regulation of Vps34 complexes by AMPK and mTOR in nutrient stress response and autophagy.

31. Guo, Grace, guo@eohsi.rutgers.edu, Rutgers University
Role of farnesoid X receptor (FXR) in regulating liver and intestine homeostasis. FXR is a ligand-activated transcription factor belonging to the nuclear receptor superfamily. We and others have identified that this

receptor is critical in mediating liver and intestine lipid and other energy homeostasis, and serving a critical role in mediating liver-intestine crosstalk. These novel discovery provide insight into understanding liver and intestine physiology and pathology, as well as providing novel targets in drug discovery.

32. Guo, Zhenheng, zguo2@uky.edu, University of Kentucky
With a long-term goal of identifying novel therapeutic targets for treatment of cardiovascular diseases, my research has focused on the role of vascular smooth muscle cells (VSMC) in cardiovascular diseases. In particular, I am interested in the physiological roles (e.g. the signal transduction signaling that regulates smooth muscle contraction and/or transcriptional regulation) of calcium independent phospholipase A2¦Â (iPLA2¦Â), CPI-17 (C-kinase-activated protein phosphatase-1 (PP1) inhibitor, 17kDa), mineralocorticoid receptor (MR), and BMAL1 and their pathological implications (e.g. vascular inflammation, oxidative stress, and vascular remodeling) in cardiovascular diseases, including hypertension, restenosis, aortic aneurysm, and diabetes vascular complications.

33. Han, Jiahuai, jhan@xmu.edu.cn, Xiamen University
My research interests are the mechanisms of intracellular signaling pathways in inflammatory responses. We study the signaling pathways that lead to necrotic cell death, cytokine production, etc.

34. Han, Min, mhan@colorado.edu, HHMI and University of Colorado
Fatty acids (FAs) are highly variable in their structures and these variations greatly contribute to the vast diversity in lipid structures. However, despite sporadic reports linking FA variants with human diseases and animal development, their functional specificities are, in general, poorly studied, and we know little about the mechanism by which these variants contribute to the lipid composition in specific tissues for cellular events under physiological conditions. We have been addressing the above questions by combining complex genetics with biochemistry in both worms and mice. I will describe our recent work that uncovered spectacular impacts of the rarely studied monomethyl branched-chain fatty acids (mmBCFAs) that abundantly present in our body on cell signaling, development and behavior.

35. He, Xi, cch@stowers.org, Stowers Institute. Studying cellular niche and niche signaling that regulates intestinal stem cells and targeting drug-resistant cancer stem cells in adenoma and colon cancer.

36. He, Zhigang, zhigang.he@childrens.harvard.edu, Boston Children's Hospital
Axon regeneration

37. Hu, Jianping, huji@msu.edu, Michigan State University
Our laboratory investigates molecular mechanisms that govern the dynamic behavior of plant organelles by focusing on peroxisomes and mitochondria - subcellular compartments tightly related to energy production and metabolism. We use molecular genetic, cell biological, biochemical, and proteomic approaches to identify factors involved in organelle division and protein degradation, and the interaction of these processes with environmental stresses. We have also begun to address the contribution of peroxisomal and mitochondrial metabolism to photosynthesis, the ultimate energy-producing process for life on earth.

38. Hu, Bo, bo.hu@northwestern.edu, Northwestern University
Our research interests focus on the molecular mechanisms of human cancer tumorigenesis, progression, invasion/metastasis and angiogenesis. We have been investigating oncogenic receptor tyrosine kinase signaling, their mediators including guanine nucleotide exchange factors (GEFs), activators for Rac1 and Cdc42 in human glioblastomas and breast cancers. Glioblastomas are the most common malignant tumors in the brain. Breast cancer is the most common cancer in women. Recently, we started to study glioma stem cells that were derived from clinical glioma tumor tissues. We are also interested in developing novel approaches for cancer therapies using novel anti-tumor and anti-angiogenic inhibitors in combination with non-invasive evaluation methods.

39. Hu, Jing-Shan, jingshan_hu@yahoo.com, Bayer Healthcare
Building Partnerships on Innovation with academia investigators and Biotech/CRO companies for global drug discovery of Bayer HealthCare

40. Huang, Yanyi, yanyi@pku.edu.cn, Peking University
My group is working on technology development for integrative biology researches. Our current research interests include genome sequencing technologies, microfluidics, and bioimaging. We are developing a few key advanced technologies to facilitate genome sequencing and its applications, especially the novel chemistry and instruments for DNA sequencing, single-cell genome and transcriptome analysis, as well as epigenomic studies of limited number of cells. We also apply large-scale integrated microfluidic devices to perform quantitative biological studies. We use microfluidic chips to culture cells and to apply the stimuli

precisely. By observing the cells' behavior on-chip, we are able to track the stochastic and dynamic life process with single-cell resolution. We are developing new imaging technologies and image processing algorithms to study the dynamics of cells and organisms. We use coherent Raman scattering microscopes to study the chemical distribution in living animals and cells without labeling. Currently we are also working on drug delivery and cancer diagnosis using this label-free technology.

41. Jiang, Xuejun, jiangx@mskcc.org, Memorial Sloan-Kettering Cancer Center
Cell death, autophagy, tumor suppressor PTEN

42. Jiang, Youxing, youxing.jiang@utsouthwestern.edu, UT Southwestern Medical Center
The research of our laboratory has been focusing on the structural and functional studies of ion channels and transporters, which control the flow of ions across the cell membrane. These proteins regulate many biological processes such as the excitation of nerve and muscle cells, the secretion of hormones, and sensory transduction. Our research approach is a combination of membrane protein X-ray crystallography, aimed at determining the three dimensional structure of the ion transporting proteins, and channel electrophysiology, to study the physiological functions of these proteins.

43. Jiang, Yu, yuj5@pitt.edu, University of Pittsburgh
Intracellular signaling pathways governing cell metabolism and growth in response to nutrients

44. Jin, Peng, peng.jin@emory.edu, Emory University School of Medicine
Epigenetics and noncoding RNAs in brain development and human diseases

45. Kang, Yibin, ykang@princeton.edu, Princeton University
The main research interest of our laboratory is to apply modern molecular biology, genomics, and computational biology approaches to understand the molecular basis of cancer metastasis. Major areas of research in our laboratory includes: identification and functional characterization of metastasis genes, pre-clinical evaluation of anti-metastasis therapeutics, development of advanced imaging technology and non-invasive detection of tumor-stroma interaction during metastasis, the role of miRNA in cancer progression and metastasis, molecular characterization of mammary gland stem cells and their link to breast tumor stem cells.

46. Kuang, Shihuan, skuang@purdue.edu, Purdue University
Muscle stem cells and tissue regeneration; Regulation of self-renewal and differentiation; Lineage origin of white and brown adipose tissues; Muscle-fat interaction and the prevention of muscle wasting and obesity

47. Lan, Fei, fei_lan@fudan.edu.cn, Fudan University
The differentiated state of a somatic nucleus can be reversed to an undifferentiated embryonic state, which is defined as somatic reprogramming. The successful of somatic reprogramming has offered exciting promises in basic and applied research, such as for testing nuclear potency, for distinguishing between genetic and epigenetic alternations of donor cells and for the potential use of custom-tailored cell therapy. Two experimental strategies have been successfully employed to induce the conversion of differentiated cells into functional pluripotent stem cells, i.e., iPS technology and NT. However, the efficiency of somatic reprogramming by these processes is very low, the quality of iPSCs is low and the underlying mechanisms are still not clear. Our research interests are focused on establishment of high-efficiency reprogramming strategies, improvement of the quality of iPSCs and elucidation of the molecular mechanisms involved in somatic reprogramming.

48. Li, Chenjian, li_chenjian@pku.edu.cn, Peking University
My laboratory is devoted to basic neuroscience and the understanding of neurodegenerative diseases such as Huntington's disease and Parkinson’s diseases at molecular, genetic, cellular and organismal levels. We work on establishing new methodology of animal modeling. The animal models we established are widely used in basic research and translational medicine.

49. Li, Guo-Min, gmli@uky.edu, University of Kentucky
DNA repair, genome instability, and cancer.

50. Li, Hongliang, lihl@whu.edu.cn, Wuhan University
My research is focusing on examining the novel role of interferon regulatory factors on cardivoascular diseases

51. Li, Jinsong, jsli@sibcb.ac.cn, Institutes for Biological Sciences, Shanghai
The differentiated state of a somatic nucleus can be reversed to an undifferentiated embryonic state, which is defined as somatic reprogramming. The successful of somatic reprogramming has offered exciting promises in basic and applied research, such as for testing nuclear potency, for distinguishing between genetic and epigenetic alternations of donor cells and for the potential use of custom-tailored cell therapy. Two experimental strategies have been successfully employed to induce the conversion of differentiated cells into functional pluripotent stem cells, i.e., iPS technology and NT. However, the efficiency of somatic reprogramming by these processes is very low, the quality of iPSCs is low and the underlying mechanisms are still not clear. Our research interests are focused on establishment of high-efficiency reprogramming strategies, improvement of the quality of iPSCs and elucidation of the molecular mechanisms involved in somatic reprogramming.

52. Li, Juan, juanL@email.unc.edu, University of North Carolina, School of Pharmacy
My research is focus on Adeno-associate virus vector (AAV) gene delivery and gene therapy for various genetic and acquired diseases. Our lab has developed AAV vectors to treat diseases like Duchenne muscular dystrophy (DMD), heart failure, diabetes, arthritis, hepatitis and cancer, etc. We have successfully worked with multiple animal models including Monkey, dogs, hamsters and mice.

53. Li, Lei, leili@mdanderson.org, M. D. Anderson Cancer Center
The core objective of my research has been to understand components and molecular mechanisms critical for the safe-guarding of genomic integrity. I am interested in repair of complex DNA lesions and related regulatory mechanisms and their translational implications in cancer therapy. Our efforts are focused in genetic instability syndromes that exhibit high predisposition to cancer. Many of these syndromes are direct consequences from deficient DNA damage repair or signaling. Currently, we are investigating the Fanconi anemia pathway and repair of DNA interstrand crosslink and DNA-protein crosslinks.

54. Li, Linheng, lil@stowers.org, Stowers Institute for Medical Research
Linheng Li lab studies the stem cell niche and stem cell development using mouse models and focusing on hematopoietic and intestinal systems. The hematopoietic system facilitates functional characterization of stem cells as bone marrow transplantation experiments can be readily performed. The intestinal system has a well-organized developmental architecture in which stem cell marking and lineage tracing can be used to investigate: • where and how stem cells are maintained by their microenvironment (niche), • how different niches provide different signals to maintain balance between long-term stem cell maintenance and daily self-renewal, proliferation, and lineage commitment, • what genomic changes and epigenetic alterations convert normal stem-progenitor cells into cancer stem cells, • what cellular and molecular mechanisms are underlying drug-resistance of cancer (especially stem) cells.

55. Li, Shawn, sli@uwo.ca, Western University
The main focus of the Li lab is to elucidate the molecular and epigenetic basis of cancer with the ultimate goal of developing protein- and peptide-based diagnostic or therapeutic agents for cancer.We use an integral approach that combines molecular, cellular, structural, and proteomic information to obtain a comprehensive picture of how proteins interact to transduce cellular signals and how aberrant changes in cellular signal transduction lead to cancer. We are particularly interested in understanding the role of post-translational modifications, including Tyr/Ser/Thr phosphorylation and Lys/Arg methylation on histone and non-histone proteins, in regulating such cellular processes as proliferation, differentiation, migration, apoptosis and DNA damage response. Because protein phosphorylation and methylation are intimately involved in tumorigenesis and metastasis, we hope our research to generate novel diagnostic and therapeutic agents that target these modifications.

56. Li, Shihua, sli@emory.edu, Emory University
My research interest focuses on the pathogenesis of polyglutamine (polyQ) diseases, in particular, the mechanisms by which mutant polyQ proteins affect gene transcription and induce selective neurodegeneration.In specific, we are studying the conditional knock-in mouse models of SCA-17 and glial specific transgenic mouse models of Huntington's disease.

57. Li, Song, sol4@pitt.edu, University of Pittsburgh School of Pharmacy
Farnesoid X receptor and Nitric Oxide Homeostasis The farnesoid X receptor (FXR) is a member of the nuclear receptor superfamily and was previously proposed to play an important role in the pathogenesis of cardiovascular diseases via regulating the metabolism and transport of cholesterol. We have previously shown that FXR is also expressed in vascular endothelial cells (EC) and that activation of FXR leads to inhibition of endothelin-1 expression. We have recently shown that activation of FXR in vascular ECs led to upregulation of eNOS expression and enhanced nitric oxide (NO) production. In addition, FXR further enhances NO signaling via facilitating the metabolism and clearance in liver and kidneys of asymmetric

dimethylarginine (ADMA), a potent endogenous inhibitor of eNOS. Our study suggests that activation of FXR enhances NO signaling via coordinated regulation of a number of FXR target genes in several different tissues/organs.

58. Li, Willis, willisli@ucsd.edu, University of California San Diego
My lab studies the JAK/STAT pathway in animal development, immunity, aging, and stem cell maintenance. We also investigate the role of JAK/STAT signaling in pathological conditions such as tumor formation and hyperactive immune responses. A current focus of the lab is how JAK/STAT signaling influences cellular epigenetic states, including heterochromatin formation and chromatin modification.

59. Li, XiaoJiang, xli2@emory.edu, Emory University School of Medicine
Neuropathology of transgenic HD animal models Identification of the polyglutamine expansion in huntingtin (htt) responsible for Huntington disease (HD) has allowed the establishment of a variety of transgenic mouse models of HD. However, most of these mouse models show no overt neurodegeneration in their brains. Expression of N-terminal mutant htt leads to more severe neurological phenotypes in transgenic pigs and monkeys than transgenic mice. Studies of different animal models of HD will reveal species-dependent neuropathology and help understand the pathogenesis of HD.

60. Liao, Charlene, liao.charlene@gene.com, Genentech Inc.
Targeting membrane IgE with an antibody against M1 prime: A novel method of reducing production of serum IgE. Elevated IgE levels are associated with atopic disease including allergic rhinitis and allergic asthma. In addition to the soluble form, IgE is found also as a membrane B cell receptor form, containing an additional extracellular 52-amino acid sequence â^' M1 prime. The M1 prime epitope is not found on secreted serum IgE, and has been detected in human IgE-switched B cells, IgE memory B cells, IgE plasmablasts and IgE myelomas. Here we report results from two clinical trials investigating the concept of targeting membrane IgE with an antibody against the M1 prime domain, with the aim of reducing production of newly stimulated allergen-specific IgE.

61. Lin, Haifan, haifan.lin@yale.edu, Yale University
Dr. Lin's work is focused on the self-renewing mechanism of stem cells, using Drosophila germline stem cells, mouse germline and embryonic stem cells, human embryonic stem cells, and Hydra stem cells as models. He also studies germline development and stem cell-related cancers.

62. Lin, Weichun, weichun.lin@utsouthwestern.edu, University of Texas Southwestern Medical Center
We have recently found that muscle dihydropyridine receptors (DHPRs) play a pivotal role in regulating the development of the neuromuscular junction (NMJ). Loss of DHPR function is followed by a disruption of muscle innervation and precocious maturation of the nerve terminals. This aberrant development of the NMJ in DHPR mutant mice is not due to the lack of synaptic or electrical activity; in fact, both synaptic and electrical activity are increased substantially in DHPR null muscles. These findings imply that electrical regulation of NMJ innervation patterning involves the activation of DHPRs. This, in turn, suggests that DHPR-mediated signaling is crucial for activity-dependent regulation of synapses. These unexpected findings provide a new avenue for investigating mechanisms of activity-dependent regulation of synapses.

63. Liu, Chunming, cli229@uky.edu, University of Kentucky
Beta-Catenin crosstalk in the intestine

64. Liu, Feng, liuf@uthscsa.edu, University of Texas Health Science Center San Antonio
Obesity and type 2 diabetes

65. Liu, Haoping, h4liu@uci.edu, University of California, Irvine
Candida albicans reversibly switches between yeast and hyphal morphologies, a major virulence attribute. We find that hyphal development consists of two phases, initiation and maintenance. Maintenance requires chromatin remodeling of promoters of hypha-specific genes. Interestingly, the combination of hypoxia and high CO2, but neither condition alone, maintains hyphal elongation, even in mutants lacking the nutrient responsive chromatin-remodeling pathway. The Ume6 transcriptional activator of hypha-specific genes is stabilized via regulation by Ofd1, a prolyl hydroxylase family member that is inhibited by hypoxia, and by an uncharacterized pathway that senses high CO2. The Ume6 stabilization and chromatin-remodeling pathways act parallel to govern hyphal maintenance and elongation. Virulence and hyphal elongation in vivo are attenuated only when both pathways are blocked.

66. Liu, Jikai, jkliu@mail.kib.ac.cn, Kunming Institute of Botany, Chinese Academy of Sciences
Bioactive natural products,chemical biology and drug discovery.

67. Liu, Jinsong, jliu@mdanderson.org, University of Texas MD Anderson Cancer Center
Polyploid giant cancer cells (PGCCs) have been observed by pathologists for over a century. PGCCs contribute to solid tumor heterogeneity, but their functions are largely undefined. Little attention has been given to these cells, largely because PGCCs have been generally thought to originate from repeated failure of mitosis/cytokinesis and have no capacity for long-term survival or proliferation. Here we report our successful purification and culture of PGCCs from human ovarian cancer cell lines and primary ovarian cancer. These cells are highly resistant to oxygen deprivation and could form through endoreduplication or cell fusion, generating regular-sized cancer cells quickly through budding or bursting similar to simple organisms like fungi. They express normal and cancer stem cell markers, they divide asymmetrically and they cycle slowly. They can differentiate into adipose, cartilage and bone. A single PGCC formed cancer spheroids in vitro and generated tumors in immunodeficient mice. These PGCCderived tumors gained a mesenchymal phenotype with increased expression of cancer stem cell markers CD44 and CD133 and become more resistant to treatment with cisplatin. Taken together, our results reveal that PGCCs represent a resistant form of human cancer using an ancient, evolutionarily conserved mechanism in response to hypoxia stress; they can contribute to the generation of cancer stem-like cells, and also play a fundamental role in regulating tumor heterogeneity, tumor growth and chemoresistance in human cancer.

68. Liu, Jun, joliu@jhu.edu, Johns Hopkins School of Medicine
Chemical Biology: Use of natural products and synthetic small molecules as probes of cell proliferation, differentiation and apoptosis and as leads for drug development.

69. Liu, Yang, yaliu@cnmc.org, Children's National Medical Center.
My research interests include regulation of inflammatory response in diseases such as sepsis, rheumatoid arthritis, multiple sclerosis and cancer. We also have active research in cancer genetics and experimental therapies, such as therapeutic targeting of cancer stem cells and immunotherapy.

70. Liu, Yi, yi.liu@utsouthwestern.edu, University of Texas Southwestern Medical Center
RNA interference,small and long non-coding RNAs, circadian clock

71. Liu, Yifan, yifan@umich.edu, University of Michigan
My research interest is to use the ciliate model system, Tetrahymena thermophila, to study epigenetic mechanisms in the regulation of transcription, DNA replication and DNA repair. I have probed into the mechanistic connections between the RNAi and Polycomb repression pathways in developmentally programmed nuclear differentiation events in Tetrahymena. I have characterized the prototypical Polycomb repressive complex in Tetrahymena, placed it in the same genetic network with RNAi machinery. I have also studied the epigenetic regulation of DNA replication, in particular the role played by H3 lysine 27 methylation. My research work has demonstrated that Tetrahymena deficient in H3 lysine 27 methylation accumulate single-stranded DNA as replication intermediates, which subsequently leads to massive DNA damage response.

72. Liu, Yong-Jun, yong-jun.liu@baylorhealth.edu, Baylor Research Institute
His 25 years of research has focused on human immunology, particularly dendritic cell biology, genomic approaches to the diagnosis of human diseases, the pathophysiology of autoimmune diseases and cancer, and the design of novel vaccines.

73. Lu, Hua, hlu2@tulane.edu, Tulane University School of Medicine
The Lu laboratory is interested in understanding the molecular and biochemical basis that underlies physiological and pathological signaling pathways (growth, metabolic, hypoxia, or DNA damage signals), which lead to gene expression and subsequent cell growth arrest, differentiation, senescence, autophagy, or apoptosis. Specifically, we focus on the pathways of p53 and c-Myc and try to identify molecule targets in these pathways for ultimate development of cancer therapy.

74. Lu, Zhimin, zhiminlu@mdanderson.org, MD Anderson Cancer Center
Pyruvate kinase M2 (PKM2) is highly expressed in multiple cancer types and contributes to the Warburg effect. Our research has illustrated the mechanisms underlying the upregulation of PKM2, nuclear translocation of PKM2, and metabolic and non-metabolic functions of PKM2 in promoting cancer cell metabolism and proliferation.

75. Luo, Liqun, lluo@stanford.edu, Stanford University
We study how neural circuits are organized to process information, and how they are assembled during development. To address these questions, we use fruit flies and mice as model organisms, and combine advanced molecular genetics with anatomical, physiological and behavioral approaches.

76. Luo, Wenqin, wenqinluo@gmail.com, University of Pennsylvania
Organization, Development, and Function of Mammalian Mechanosensory Dorsal Root Ganglion (DRG) Neurons, especially those sensing discriminative touch, pain, and itch.

77. Luo, Xuelian, Xuelian.Luo@UTSouthwestern.edu, University of Texas Southwestern Medical Center
Structural and biochemical characterization of the Hippo signaling pathway. The major goal is to determine the crystal structures of the core components of the Hippo pathway and elucidate the molecular mechanism of kinase activation and inhibition in organ size control with the combination of biophysics and biochemical approaches.

78. Ma, Minghong, minghong@mail.med.upenn.edu, University of Pennsylvania
G Protein-coupled Odorant Receptors Are Required for Mechanosensitivity in Olfactory Sensory Neurons Mechanosensitive cells are essential for organisms to sense the external and internal environments, and a variety of molecules have been implicated as mechanical sensors. Here we report that odorant receptors – a large family of G-protein coupled receptors – underlie the responses to both chemical and mechanical stimuli in mouse olfactory sensory neurons (OSNs). We demonstrate that knocking out key signaling proteins in odor transduction completely eliminates mechanical responses in OSNs. Furthermore, OSNs expressing different types of odorant receptors display significantly different responses to mechanical stimuli. Lastly, loss-of-function mutation or genetic ablation of an odorant receptor abolishes or reduces mechanical responses in the host OSNs. These results suggest that odorant receptors may serve as polymodal sensors of both chemical and mechanical stimuli, revealing a novel mechanism for mechanotransduction.

79. Ma, Xiaochao, mxiaocha@pitt.edu, University of Pittsburgh
Dr. Ma's research targets nuclear receptor-mediated drug-drug interactions and drug-induced liver injury. His laboratory has extensive experiences in studying human pregnane X receptor (PXR), a nuclear receptor involved in xeno- and endo-biotic metabolism. The laboratory utilizes the genetically engineered mouse models, including Pxr-null and PXR-humanized mice, to determine the role of human PXR in metabolism and toxicity in vivo. In addition, the laboratory is interested in identifying small molecule biomarkers of drug-induced liver injury using a metabolomic approach. Specific responding biomarkers are used for elucidating the mechanisms of drug-induced liver injury.

80. Mei, Lin, synapsesnmj@gmail.edu, Georgia Regents University
Synapse formation and plasticity and implications in muscular dystrophy and brain disorders.

81. Min, Jinrong, jr.min@utoronto.ca, University of Toronto
CpG islands contain a high density of CpG content and embrace the promoters of most genes in vertebrate genomes1. In the human genome, about 70% of promoters have high frequency of CpG dinucleotides. Generally, the CpG dinucleotides in the CpG islands of promoters are nonmethylated, irrespective of transcription status of the associated genes, with some exceptions, such as those CpG islands associated with X chromosome and imprinted genes. In spite of their conspicuous importance, the functional roles of the CpG islands in chromatin structure and transcription were elusive until recently. Recently we systematically characterized all of the known human CpG binding proteins by structural, systems and chemical biology approaches.

82. Ming, Guoli, gming1@jhmi.edu, Johns Hopkins University
The research of Dr. Guo-li Ming's laboratory centers on understanding the molecular mechanisms underlying neuronal development during embryonic stages and in the adult brain, with a particular focus on the signaling events involved in cell morphogenesis, cell migration, and axon/dendritic guidance.

83. Pan, Duojia, djpan@jhmi.edu, Johns Hopkins University
Duojia Pan studies the molecular and developmental pathways that control how organ size is determined in different organisms and the way these mechanisms regulate tissue homeostasis in normal and pathological conditions.

84. Peng, Junmin, junmin.peng@stjude.org, St. Jude Children's Research Hospital
Our research goal is to develop ultrasensitive, high throughput mass spectrometry-based proteomics technologies to address biological challenges in three fields: ubiquitin research, neuroscience and cancer biology. The lab seeks to identify the enzyme-substrate network of protein ubiquitination, and to investigate the structure and function of ubiquitin chains. Another focus is to profile tens of thousands of proteins and modifications directly from human disease specimens relevant to cancer and neurodegenerative disorders, and to integrate proteomic, genomic and clinical data. These studies allow identifying novel disease proteins

that may serve as biomarkers and provide new insights into pathogenesis as well as therapeutic intervention.

85. Qian, Brian, brian@cgenetool.com, Certified Genetool Inc.
I started the Certified Genetool Inc. in Bay Area to meet the needs for re-certified quality instruments for acdemic labs and biotech companies. We have grown into a sizable operation with offices in Bay Area, East Coast, China Beijing and Shanghai with combined 20 personnel. We have become the favored instrument provider for new labs and startup companies, with discounted equipment and solid after sale support. We provide re-certified equipment from GE, ABI, BD, MD, Beckman, Li-Cor, PE.....We have experienced service engineer team in the States and China to provide installation and warranty.

86. Shao, Feng, shaofeng@nibs.ac.cn, National Institute of Biological Sciences, Beijing
My laboratory studies molecular mechanisms of bacterial infection and host innate immunity. Using pathogens such as Shigella, Salmonella, Enteropathogenic E. coli (EPEC), Legionella and Burkholderia as the model, we have discovered several novel posttranslational modifications, including serine/threonine eliminylation, cysteine methylation, arginine GlcNAcylation and ubiquitin/NEDD8 deamidation, utilized by these pathogens to paralyze host innate immunity. Meanwhile, we are also interested in the role of the inflammasome pathway in macrophage immune defense against bacterial infections. Macrophage senses many kinds of pathogen-derived molecular patterns and activates the cytoplasmic inflammasome complex, leading to Il-1b production and macrophage pyroptosis. We are combining biochemical reconstitution, cell biology and bacterial/mouse genetics to identify new sensors and components involved in inflammasome signaling and further define their functional mechanisms.

87. Shen, Zhiyuan, shenzh@umdnj.edu, Cancer Institute of New Jersey-RWJMS
I am interested in genomic instability and cancer. Recently, we are particularly interested in how defective homologous recombination and DNA replication stress may contribute to numerical and structural chromosome instability. We also construct genetically engineered mouse models to address how dys-regualtion of DNA repair and cell cycle may lead to the development of cancer.

88. Sheng, Shijie, ssheng@med.wayne.edu, Wayne State University School of Medicine
Regulation of tumor cell plasticity in response to tissue micrenvironment duing tumor progression and metastasis.

89. Shi, Yujiang, Yujiang_shi@hms.harvard.edu, Brigham and Women's Hospital, Harvard Med Sch
We are interested in exploring novel mechanisms involved in regulation of eukaryotic gene expression. Mainly we try to understand how dynamic and coordinated changes of chromatin modifications including histone modification and DNA methylation (“epigenetic codes”) regulate gene expression in eukaryotic cells. We seek to identify novel epigenetic regulators and characterize their roles in controlling the patterns of epigenetic codes during normal cell differentiation and tissue development. We also try to understand how perturbation of these epigenetic processes could lead to complex human diseases. We hope that our studies will open new avenues in the histone and DNA demethylation field allowing us to quickly drive our research forward with great momentum in the new direction of translational research for epigenome medicine.

90. Shi, Yang, yang_shi@hms.harvard.edu, Harvard Medical School and Boston Children’s Hospital
My laboratory has a longstanding interest in understanding the molecular mechanisms that control mammalian transcription and chromatin regulation. We identified the first histone demethylase LSD1 in 2004, and subsequently many additional demethylases with different substrate specificity, thus demonstrating the dynamic nature of histone methylation regulation, a modification that was long considered irreversible. We continue to investigate the mechanisms that underlie dynamic regulation of histone methylation, as well as proteins that recognize various methylated and unmethylated states of histones, and the impact on both physiological and pathological conditions. Non-Mendelian inheritance influences a wide variety of phenotypes. How non-Mendelian information is transmitted from generation to generation is largely unknown. In *C. elegans,* loss of spr-5, which encodes the mammalian H3K4me2 demethylase LSD1, results in progressive sterility over multiple generations. To investigate the underlying molecular mechanism, we carried out an RNAi screen and identified both suppressors and enhancers of the trans-generational sterility phenotype. In this presentation, I will discuss the RNAi screening results and a possible working model.

91. Shi, Yanhong, yshi@coh.org, Beckman Research Institute of City of Hope
Stem cell technology holds great promise for the treatment of many diseases. Identifying factors that control stem cell maintenance and self-renewal could move stem cell technology from bench to bedside. My research is focused on defining molecular mechanisms regulating stem cell self-renewal and differentiation.

We aim to identify molecular cascades that program neural stem cells to self-renew, or induce them to differentiate. Furthermore, my laboratory aims to study pathological mechanisms and develop therapeutic candidates for neurological diseases using patient iPSCs. We have derived patient iPSCs for leukodystrophy, Alzheimer's and Parkinson's diseases. These patient iPSCs will be differentiated into neurons and/or glia based on disease phenotype. We will study disease mechanisms and perform drug screening using the iPSC platform.

92. Song, Hongjun, shongju1@jhmi.edu, Johns Hopkins University
We are interested in understanding novel mechanisms regulating structural and functional plasticity in the mature mammalian center nervous system. There are two major topics in the laboratory: one is on adult mammalian neural stem cells and neurogenesis; and the other is on epigenetic DNA modifications in the mature nervous system.

93. Su, Bing, Bing.Su@yale.edu, Yale University School of Medicine, Shanghai Institute of Immunology
1) We study the mammalian target of rapamycin (mTOR) pathway focusing on an adaptor molecule called Sin1. My lab a few years ago identified Sin1 as a critical regulator of the mTOR pathway. Following that initial discovery, we have continued our study on this complicated pathway by focusing on the molecular mechanisms and function of Sin1 by which it regulates mTOR complex (mTORC)2. My lab is also the first to discover a novel and highly conserved Sin1-mTORC2 function in regulating the stability of newly synthesized AGC kinases Akt and conventional PKC. We further demonstrated that this Sin1-mTORC2 function is associated with actively translating ribosomes. At the mechanistic level, we show that Sin1-mTORC2 acts as the kinase to phsophorylate Akt at the hydrophobic motif (HM) site, which is required for Akt substrate specificity, and at the TM site, which stabilizes the newly translated Akt. Furthermore, we discovered a negative regulatory role of Akt HM site phosphorylation, which led to the degradation of active Akt thus down regulating the PI3K-Akt signal. To understand the function of Sin1-mTORC2 in lymphocytes, we show that Sin1-mTORC2 plays a B cell specific role in regulating the FoxO1/3a activity during B cell development. Sin1-mTORC2 is required to suppress Rag1/2 gene expression when pre-BCR and BCR are capable of signaling, which is critical for B cell selection. In addition, we show that Sin1-mTORC2 negatively regulates nTreg expression but is largely dispensable for thymocyte development and peripheral T cell activation/proliferation. At the moment, we are working on the developmental role of Sin1 in embryonic angiogenesis and we also investigate the phosphorylation of Sin1 in regulating mTORC2 activity and function. We just generated Sin1 conditional knockout mice,

94. Sun, Luzhe, sunl@uthscasa.edu, University of Texas Health Science Center San Antonio
Both aging and exposure to environmental xenoestrogens such as bisphenol A (BPA) have been shown to be risk factors for breast cancer. We hypothesized that these risk factors may cause mammary gland susceptible to tumorigenesis by altering the number and function of mammary stem cells (MaSCs). Our studies showed that pubertal BPA exposure to BPA in mouse models altered the ratio of luminal to basal progenitor/stem cells and caused early neoplastic lesions in their regenerated glands indicating that MaSCs are susceptible to BPA-induced transformation. Interestingly, aging also caused similar changes of mouse MaSCs. Significantly, the early neoplastic transformation of MaSCs was inhibited by an anti-aging drug suggesting a potential utility of anti-aging drug for the prevention of breast cancer.

95. Sun, XiaoHong, Xiao-Hong-Sun@omrf.org, Oklahoma Medical Research Foundation
Myeloid Leukemia T cell biology

96. Sun, Yi, ysun@mednet.ucla.edu, University of California Los Angeles Medical School
Tissue specific stem cells often reside in highly complex cellular environment and are in close contact with specific stem cell niche and their progenies to maintain stem cell homeostasis via controlling the balance between quiescent and activated states. The scarcity of stem cells, particularly quiescent stem cells, and the enormous heterogeneity of stem cell surrounding environment had made characterization of the cellular and molecular identities of these cells extremely challenging. To face such a challenge, we have established technologies allowing RNA sequencing to be performed on single somatic cells in adult tissues. Using this technology, we profiled 17 single CD133 positive and negative cells isolated from the adult neurogenic zone, the ependymal and subependymal regions of the forebrain. We uncovered molecular signatures of critically staged cells along stem cell activation and subsequent neural lineage development. Moreover, using WGCNA (Weighed Gene Co-expression Network Analyses) we unearthed a tight link between CD133 expressing adult neural stem/progenitor cells and vasculature/angiogenesis related programs. This technology is likely applicable to the molecular identification of cancer stem cells, as well as revealing molecular features of complex biological systems such as the various functional neural circuits in the brain.

97. Sun, Zuoming, zsun@coh.org, Beckman Research Institute of City of Hope
T cell development, activation and differentiation

98. Tang, Fuchou, tangfuchou@pku.edu.cn, Peking University
As the primary cause for failure in human pregnancy and genetic disorders, aneuploidy increases drastically with women's age, and leads to low success rates in live birth and in vitro fertilization (IVF). To select ovum or embryo without aneuploidy in IVF, preimplantation genetic diagnosis (PGD) and preimplantation genetic screening (PGS) have been implemented with PCR, FISH, SNP and CGH arrays, but are limited in accuracy and resolution, thus often giving high false positives and negatives. Here we demonstrate highly accurate genome-wide PGS using multiple annealing and looping based amplification cycle (MALBAC) for single cell whole genome amplification and sequencing of oocytes and 8-cell embryos. By sequencing the two polar bodies of each oocyte, we deduced the ploidy of the female pronucleus, phased the maternal genome, and inferred the oocyte haplotype. In so doing, we show the proof of principle for selecting an embryo free of aneuploidy, particularly for women with recurrent implantation failure and miscarriages, as well as of maternal genetic diseases associated with point mutations.

99. Tang, Qi-Qun, qqtang@shmu.edu.cn, Fudan University
The Lab has been focused on the molecular mechanism under which pluripotent stem cell commit to preadipocyte; the mechanism how adipocyte is developed; how white adipocyte can be convert to brown adipocyte.

100. Wan, Chao, cwan@cuhk.edu.hk, The Chinese University of Hong Kong
Oxygen sensing and skeletal regeneration Chao Wan Oxygen is a fundamental requirement for organogenesis and tissue regeneration. Hypoxia inducible factors (HIFs) are essential mediators of cellular adaptation to oxygen fluctuations and critical for proliferation and differentiation of stem/progenitor cells. We showed that HIFá serves as a key to couple angiogenesis to osteogenesis during skeletal development and regeneration. Deletion of HIF-1á in the condensing mesenchyme of mouse bones impaired self-renewal and osteoblast differentiation of mesenchymal stem cells (MSCs). Hypoxia promoted the expansion of CD146+ human umbilical cord perivascular cells (HUCPVCs) and maintained the properties of MSCs in vitro and in vivo. The expansion of CD146+ HUCPVCs was associated with the inhibition of transcriptional suppression of Hif-2á by PPAR-ã under hypoxia.

101. Wang, Helen, Y, ywang4@tmhs.org, Methodist Hospital Research Institute
My research team studies the role of T cells in antigen recognition and immune regulation. The projects include identifying mechanisms of cytokine signaling in immune cells, tumor antigen discovery, and defining mechanisms of regulatory T cell function and innate immune signaling. A critical goal of my research is to identify antigens that are recognized by various T cells, including Th1, Th17 and Treg cells. We also work on understanding innate immune signaling pathways, in particular Toll-like receptor (TLR8) signaling in the reversal of regulatory T cell suppressive functions. We investigate how to improve immune responses by reducing the levels of these negative regulators in immune cells. These research findings have the potential to translate into novel therapeutics for diseases such as cancer, obesity and ALS.

102. Wang, Hong, hongw@temple.edu, Temple University School of Medicine
Dr. Wang's research focuses on identifying biochemical mechanisms responsible for hyperhomocysteinemia (HHcy) –induced cardiovascular disease, and discovering therapeutic targets and novel therapeutic strategies.

103. Wang, Hongbing, wangho@msu.edu, Michigan State University
My lab investigates the cellular and molecular mechanisms underlying synaptic modification and certain aspects of adaptive behavior. We aim to understand the function of calcium-stimulated signal transduction pathways and G protein-coupled receptors. We use molecular and cellular, transgenic, electrophysiological, and behavioral approaches to identify the molecular components involved in memory formation, anxiety, depression, behavioral flexibility, and emotional stability. Some results from these efforts have led to the development of new animal models of Schizophrenia and Bipolar disorder. Our mechanistic studies have also suggested several potential therapeutic approaches to treat Fragile X Syndrome and possibly autism.

104. Wang, Honglin, honglin.wang@sjtu.edu.cn, Shanghai Institute of Immunology
Forkhead box P3 (FoxP3)+ CD4+ regulatory T (Treg)-cells can be divided into natural Treg (nTreg)-cells and adaptive Treg (iTreg)-cells. Despite the mechanisms of iTreg-cell generation have been intensively focused in the past, our understanding of molecular mechanisms, particularly microRNA (miRNA)-mediated epigenetic regulation involved in the iTreg-cell generation is lacking. Here we show that microRNA-31 (miR-31), a small non-coding RNA, is diminished in TGF-beta1-induced iTreg-cells. MiR-31 limits iTreg-cell generation by directly repressing FoxP3. Specific miR-31 ablation in CD4+ T-cells promotes iTreg-cell differentiation, and ameliorates autoimmunity. Thus, our study has identified miR-31 as a negative post-transcriptional regulator for TGF-beta1-induced iTreg-cell generation in autoimmune disease.

105. Wang, Jian, jianwang@med.wayne.edu, Wayne State University
Cellular metabolism is often tailored to satisfy the specific needs of various cell types with particular functionalities. We are interested to study the metabolic changes and their roles in the processes of cell fate changes using embryonic stem cell renewal and differentiation and the epithelial-mesenchymal transition as experimental models. We previously demonstrated that mouse ES cells uniquely rely on threonine as metabolic fuel for self-renewal. We currently focus on studying the roles of mitochondrial amino acid and one-carbon metabolism in stem cell renewal and in cellular de-differentiation process such as EMT.

106. Wang, Jing (Jenny), jjwang@unmc.edu, University of Nebraska Medical Center
We are interested in studying molecular mechanisms and identifying therapeutic targets of colon cancer metastasis.

107. Wang, Jiwu, jiwuwang@allelebiotech.com, Allele Biotech/Scintillon Institute
mRNA reprogramming and differentiation, single domain camelid nanoantibodies, and fluorescent proteins for superresolution imaging.

108. Wang, Lianchun, lwang@ccrc.uga.edu, University of Georgia
Heparan sulfate proteoglycan in vascular development, cancer biology and stem cell biology

109. Wang, Qiming, qjw1@pitt.edu, University of Pittsburgh
My laboratory conducts basic and translational research on oncogenic protein kinases, particularly protein kinase D (PKD) and protein kinase C (PKC) families, in the area of cancer biology. My research group has pioneered the work on the discovery and development of novel chemical probes for PKD, which includes the identification and development of the first potent and specific small molecule PKD inhibitor – CID755673 and its analogs. Our previous work in prostate cancer has led to the identification of PKD as a potential therapeutic target for prostate cancer.

110. Wang, Rongfu, rwang3@tmhs.org, The Methodist Hospital Research Institute
I have a long-standing interest in tumor immunotherapy and cancer vaccine development. In particular, We have been interested in tumor antigen discovery, cancer stem cells and innate immunity and functional control of regulatory T cells by Toll-like receptors. My team studies the role and mechanisms of Toll-like receptors, NOD-like receptors, negative regulators of innate immune signaling, inflammation, epigenetics and cancer immunotherapy.

111. Wang, Xiao-Fan, xiao.fan.wang@duke.edu, Duke University Medical Center
The current research in the Wang laboratory mainly focuses on the elucidation of molecular nature and signaling mechanisms associated with tumor microenvironment that promotes tumor progression and metastasis. Particularly, we are interested in the roles of specific microRNAs as mediators of TGF-ß or hypoxia to affect the biological nature of tumor microenvironment, via the interactions with the immune system and the recruitment of various types of stromal cells, to enhance tumor metastasis. We are also studying the mechanisms underlying the phenomenon of cellular senescence. Ultimately, we hope that our studies in these areas to lead to the development of novel therapeutics for the treatment of various types of human cancer.

112. Wang, Xiao Jing, xjwang@nyu.edu, NYU Shanghai
Xiao-Jing Wang is the Provost of NYU Shanghai, and Professor of Neural Science at New York University. As Provost, Wang serves as NYU Shanghai's chief academic officer, setting the university's academic strategy and priorities, and overseeing academic appointments, research, and faculty affairs. Before joining NYU in the fall of 2012, Wang was Professor of Neurobiology at Yale University. At Yale he also served as the Director of the Swartz Center for Theoretical Neuroscience, and held secondary faculty appointments in Physics, Applied Mathematics and Psychology. Dr. Wang is an expert on the neurobiology of executive and cognitive functions. His group has pioneered neural circuit models of the prefrontal cortex, which is often called the "CEO of the brain". In particular, Dr. Wang is known for his work on the cellular basis of short-term memory, neural mechanisms for decision-making, communication and synchronization through inhibitory neurons in the brain. His research group is now embarking on a new initiative of developing neurobiologically-realistic large-scale brain circuit models of cognitively-controlled flexible behavior. He is a recipient of Alfred P. Sloan Research Fellow, National Science Foundation CAREER Award, John Simon Guggenheim Memorial Foundation Fellow, Chinese Government's 1000 Talent Award. Wang is also a Fellow of the American Association for the Advancement of Science.

113. Wang, Xiaojing, XJ.Wang@UCDenver.edu, University of Colorado Denver
We are using genetically engineered mouse models and human cancer specimens to study cancer stem cell properties in squamous cell carcinomas of the skin and oral cavity.

114. Wang, Yanzhuang, yzwang@umich.edu, University of Michigan
My research focuses on the biogenesis, function, and defects of the Golgi in diseases. The Golgi apparatus is a membrane-bound structure that serves as a central conduit for protein and lipid modification, trafficking and secretion. We want to understand how the unique stacked Golgi structure is formed and why this structure is important for its function. In the last few years, we found that stack formation, mediated by oligomerization of Golgi stacking proteins, ensures accurate protein modifications. We also discovered that monoubiquitination regulates Golgi membrane dynamics during the cell cycle. Most recently, we have gathered intriguing preliminary data concerning the structural and functional defects of the Golgi in cell culture and mouse models for cancer, and cardiovascular and Alzheimer's diseases.

115. Wang, Yibin, yibinwang@mednet.ucla.edu, University of California Los Angeles School of Medicine
Molecular pathways, genomics and genetics of cardiovascular diseases

116. Wang, Zhong, zhongw@umich.edu, University of Michigan
The research focus in the Wang laboratory is to dissect how epigenetic events determine stem cell self-renewal and differentiation. In particular, we are interested in deciphering the molecular mechanisms guiding directed differentiation of stem cells into desired mature cell types in heart. The Wang laboratory is also exploring novel strategies in producing cardiac progenitor cells and cardiomyocytes and establishing large animal models for preclinical studies. Many serious medical conditions, such as birth defects, are due to defective differentiation of stem cells into various human tissues. Our studies may help reveal the etiology of related heart diseases and provide clues to develop modern medical treatments such as small molecule drugs, gene or stem cell/progenitor cell therapies against these diseases.

117. Wang, Zhou, wangz2@upmc.edu, University of Pittsburgh

118. Wei, Yingfei, yingfei.wei@gmail.com, Elixirin Corporation
Bridging the innovative biotechnology across the multidisciplinary fields and areas, including eastern and western worlds and therapeutic and preventive medicines.

119. Wu, Hui, hwu@uab.edu, University of Alabama at Birmingham
Protein glycosylation and secretion, and their contribution to bacterial fitness and virulence

120. Wu, Jian-Qiu, wu.620@osu.edu, Ohio State University
The research interest of my laboratory is to understand the roles of cytoskeletal and signaling proteins in cytokinesis and cellular wound healing. Cytokinesis partitions cellular constituents into two new daughter cells at the end of the cell cycle. When coordinated with the generation of cellular asymmetry, cytokinesis can produce diverse cell types in multicellular organisms. Thus, cytokinesis plays a crucial role in both cell proliferation and cell differentiation. Contractile rings consisting of actin filaments and myosin-II motor proteins are the common machinery for cytokinesis and other processes including epithelial closure, epithelial wound healing, and apoptotic cell extrusion. We are studying contractile-ring assembly, constriction, and function in cytokinesis and single-cell wound healing using a combination of cellular, molecular, biochemical, genetic, and microscopic approaches.

121. Wu, Xiangwei, xwwu@mdanderson.org, University of Texas MD Anderson Cancer Center
Cancer chemoprevention approaches generally use long-term, continuous treatment, which can produce major preventive effects but which can also have unexpected serious adverse events. This raises the question of whether intermittent dosing schedules might reduce toxicity while retaining benefit, a concept that we call short-term intermittent therapy to eliminate premalignancy (SITEP). We have demonstrated that SITEP approach whereby short-term, intermittent therapy eliminates premalignant cells via apoptosis that is induced by synthetic lethal interactions, can be use for personalized and targeted chemoprevention.

122. Wu, Hao, hao.wu@childrens.harvard.edu, Harvard Medical School and Boston Children's Hospital
We are interested in elucidating the molecular mechanism of signal transduction by immune receptors, especially innate immune receptors.

123. Xiao, Xiao, xxiao@email.unc.edu, University of North Carolina
1) Genetic disease gene therapy; 2)AAV vector gene delivery in animal models; 3)Neuromuscular and metabolic diseases; 4)MicroRNA in nervous and muscle systems.

124. Xie, Wen, wex6@pitt.edu, University of Pittsburgh
Dr. Xie's laboratory studies orphan nuclear receptor-mediated regulation of genes encoding drug metabolizing enzymes and transporters. The same enzymes and transporters are also responsible for the homeostasis of endogenous chemicals (endobiotics) that include steroid hormones, cholesterol, lipids, bile acids and bilirubin. Research in Xie lab has helped to establish members of the orphan nuclear receptors, such as PXR (pregnane X receptor), CAR (constitutive androstane receptor), LXR (liver X receptor) and ROR (retinoid-related orphan receptor), as xeno- and endo-sensors that sense xeno- and endobiotics which, in turn, lead to enzyme and transporter gene regulation. This regulation has broad implications in drug metabolism and drug development. Moreover, these orphan receptors can be explored as therapeutic targets for the treatment and prevention of human diseases, such as cholestasis, jaundice, gallstone disease, breast cancer, prostate cancer, colon cancer, and metabolic syndrome.

125. Xie, Xin, xxie@simm.ac.cn, Shanghai Institute of Materia Medica
We are interested in the roles of G-protein coupled receptors (GPCRs) in the development of Multiple Sclerosis (MS). We found several GPCRs, which have been targeted to treat other diseased, are involved in the pathogenesis of MS, such as cysteinyl leukotriene receptor 1 (CysLT1) and adenosine receptor A2B. Several clinically used drugs targeting these receptors could effectively block the development of EAE, a mouse model of MS. Given our current lack of effective pharmacological targets for the treatment of MS, the continuous identification and study of GPCRs in MS pathogenesis may eventually lead to major breakthroughs and new pharmacological strategies. We are also interested in stem cell research from a chemical biology point of view.

126. Xiong, Yue, yxiong@email.unc.edu, University of North Carolina
Cell cycle control, ubiquitylation and tumor metabolism

127. Xu, Tian, tian.xu@yale.edu, Yale University
Xu lab is interested in utilizing model organisms to understand cancer biology and developmental mechanisms. In particular, the lab is developing and using new genetic approaches to identify genes that are involved in tumor growth and metastasis, and are exploring the developmental and biochemical functions of these genes.

128. Xu, Wei, wxu@oncology.wisc.edu, University of Wisconsin-Madison
Our laboratory is focused on the transcriptional regulation of estrogen receptor (ER) signaling pathways by nuclear receptor co-factors. Our major interest is on a protein arginine methyltransferase CARM1/PRMT4, a nuclear hormone receptor co-activator. Histone H3 methylation by CARM1 potentiates target gene activation by ER.

129. Xu, Yang, yangxu@ucsd.edu, University of California San Diego
Our laboratory is interested in understanding the signaling pathways involved in maintaining genetic stability in mammalian cells, particularly embryonic stem cells (ESCs). We are investigating the pathways that coordinate the DNA damage responses and self-renewal of ESCs and adult stem cells. We are testing the hypothesis that disruption of this coordination will induce genetic instability in adult stem cell population, leading to the generation of cancer stem cells. We are focusing on the roles of tumor suppressors such as ATM and p53, which are critical to maintain genetic stability in somatic cells. To facilitate the studies of gene function in human ESCs, we recently developed technologies that allow efficient knock-out and knock-in through homologous recombination in hESCs.

130. Yang, Li, liyang@picb.ac.cn, CAS-MPG Partner Institute for Computational Biology
I joined CAS-MPG Partner Institute for Computational Biology, Chinese Academy of Sciences to set up his own laboratory in 2011, where I and collaborators have successfully identified series of intron-derived non-polyadenylated lncRNAs, either processed and stablized by capping with snoRNAs at both ends, or stablized by forming circular structures. In the future, I will continue using the start-of-the-art deep sequencing technology and computational approaches to pursue his longstanding interest in exploring the complex transcriptome network, including but not limited to noncoding RNAs, RNA editing and alternative splicing(www.picb.ac.cn/rnomics).

131. Yang, Xiangdong William, xwyang@mednet.ucla.edu, University of California Los Angeles
Dr. Yang's laboratory at UCLA applies powerful mouse genetic approaches, including Bacterial Artificial Chromosome (BAC)-mediated transgenesis, to model Huntington's disease (HD) and other neurodegenerative disorders in mice. These novel BAC models allow sophisticated dissection of disease pathogenesis in vivo, and preclinical testing of novel candidate therapeutics. Moreover, the Yang lab is also using novel mouse models to study how the mammalian basal ganglia circuitry controls volitional behaviors

and how aberrant BG circuits may lead to pathological behaviors similar to those in neuropsychiatric disorders.

132. Yang, Xiao-Feng, xfyang@temple.edu, Temple University School of Medicine
Research projects in Dr. Yang's lab are to determine how immune cytokine pathways regulate high lipid- and high glucose-triggered endothelial cell activation and inflammation, which include hyperlipidemia-, hyperglycemia-, uremia-, endotoxemia-induced endothelial activation, endothelial progenitor cell survival pathways, epigenetic regulation of regulatory T cell apoptosis, microRNA regulation of endothelial cell activation.

133. Yang, Xiaoyong, xiaoyong.yang@yale.edu, Yale University School of Medicine
Metabolism drives all biological processes, dysregulation of which fuels a plethora of human diseases including diabetes, obesity, cancer, aging, cardiovascular and neurodegenerative diseases. The long-range goal of our research is to unravel temporal and spatial regulation of metabolic pathways in response to environmental and genetic cues, and to design strategies to battle metabolic diseases. Diet and the light/dark cycle are principle environmental cues that control intermediary metabolism. Nutrient flux into the cell triggers the posttranslational modification of intracellular proteins by the amino sugar called N-acetylglucosamine (O-GlcNAc). Our first goal is to elucidate how O-GlcNAc acts as a molecular switch that couples nutrient cues to cellular regulation of signal transduction, transcription and protein degradation. Both light and diet affect the body's circadian rhythms. Our second goal is to depict molecular pathways that couple the circadian clock to metabolic physiology. We are employing a combination of experimental approaches, including biochemistry, molecular and cellular biology, mouse genetics, genomics, proteomics and metabolomics, to accomplish our research goals.

134. Yang, Yong-Guang, yy2324@columbia.edu, Columbia University
My primary research interest has been in the area of transplantation immunology, with a focus on experimental hematopoietic cell transplantation for the treatment of blood malignancies and induction of transplantation tolerance. I am also interested in the development of humanized mouse models for the in vivo study of human immune function and human cancer immunotherapy.

135. Yao, Jun, jyao1@mdanderson.org, M. D. Anderson Cancer Center

136. Yu, Ron Congrong, cry@stowers.org, Stowers Institute for Medical Research
My research interest is to understand the neural circuitry that processes sensory information. In particular, I study the mammalian olfactory to understand how olfactory information is encoded by the patterns of activity. I also study the vomeronasal system to understand how pheromone input triggers innate reproductive and territorial behaviors.

137. Yu, Dihua, dyu@mdanderson.org, University of Texas MD Anderson Cancer Center
My laboratory functions as a bridge connecting basic/translational cancer research to important issues in cancer patient care. We focus on studying the molecular mechanisms of breast cancer initiation, progression, metastasis and therapeutic resistance. We are exploring the potential of marker-guided, rationally designed, combinatorial targeted therapies for treating human cancers using various preclinical cell and animal models. We use transgenic and knockout mouse models to study the role of 14-3-3zeta, PTEN, and ErbB2 in normal development and cancer progression. Our discoveries of PTEN loss-induced Herceptin-resistance (Cancer Cell 2004, cited >900 times) and strategies for overcoming Herceptin-resistance have led to efficacious Phase I/II clinical trials (J. C. O. 2011). We identified "key nodes" in the Herceptin resistance network and developed strategies of targeting the "key node" to overcome resistance from multiple resistance mechanisms (Nature Medicine, 2011). Currently, we are further boosting therapeutic efficacy of combinatorial targeted therapies by enhancing T cell activation. We have found that 14-3-3zeta cooperates with ErbB2 to promote the deadly transition from non-invasive DCIS to invasive breast cancer and 14-3-3zeta overexpression confers poor survival of breast cancer patients (Cancer Cell, 2009). Additionally, we study mechanisms and prevention strategies of ER negative, especially, triple negative, early stage breast cancers. Our recent research areas include identifying drivers and therapeutic targets of breast cancer brain metastasis, stem cells and breast cancer initiating cells, molecular imaging of breast cancer progression and metastasis, dysregulation of i) microRNA function and biogenesis; ii) metabolism, iii) tumor microenvironment; and iv) epigenetic modifiers, and their roles in breast cancer progression, metastasis and resistance to targeted therapies.

138. Yu, Guoliang, guoliang.yu@gmail.com, Telome Health Inc
Innovations that monitor general health and detect early sign of diseases. Biotechnology and entrepreneurship.

139. Yu, Hongtao, Hongtao.Yu@UTSouthwestern.edu, University of Texas Southwestern Medical Center
The long-term research interest of my laboratory is to study cellular mechanisms that govern chromosome inheritance and integrity, with a combination of cell biological, biochemical, and biophysical methods. Genomic DNA is packaged into highly compacted chromatin. The nucleosome core particle, the basic building block of chromatin, consists of 147 base pairs of DNA and a histone octamer. Histone modifications regulate chromatin structure and dynamics, which in turn affect all processes that need to access genomic DNA, including DNA replication, sister-chromatid cohesion and segregation, and DNA repair.

140. Yu, Jindan, jindan-yu@northwestern.edu, Northwestern University
FoxA1 is a pioneering transcription co-factor of the androgen receptor (AR) through which it defines prostate-specific gene expression. Surprisingly, we recently found that FoxA1 inhibits epithelial-to-mesenchymal transition, cell motility and tumor metastasis, exhibiting an AR-antagonizing role. Interestingly, through integrative genomic and bioinformatics analysis, we further found that FoxA1 defines the genomic landscape of AR not by direct recruitment, rather by inhibiting AR binding to non-target sites, via altering chromatin accessibility. Moreover, FoxA1 downregulation in prostate cancer activates AR signaling and contributes to castration resistance. Our data thus suggest a provocative model wherein FoxA1 acts as a suppressor, instead of a collaborator, of AR signaling, thereby providing important mechanisms to recurrent FoxA1 mutations found in prostate cancer.

141. Yue, Zhenyu, zhenyu.yue@mssm.edu, Mount Sinai School of Medicine
Autophagy Mechanism in Neurodegeneration

142. Zhang, Feng, zhang@broadinstitute.org, The Broad Institute of MIT and Harvard
The ability to introduce targeted modifications into genomes and engineer model organisms holds enormous promise for biomedical and biotechnological applications. Here we present the development of an RNA-guided nuclease adapted from the bacterial CRISPR-Cas immune mechanism for efficient and multiplexable mammalian genome engineering. Through heterologous expression of three minimal components in CRISPR-Cas, we show that Cas9 can be programmed by custom RNAs to induce DSB at endogenous mammalian loci with up to 59% cutting efficiency. Cas9 can be further converted into a nicking enzyme to facilitate template-directed homologous recombination while minimizing mutagenic indel formations. Finally, using a single crRNA array to encode a pair of guide sequences, we show that CRISPR can simultaneously and efficiently cleave multiple sites within the human genome. The tractability and multiplex capability of this system present unique possibilities for practical and therapeutic applications.

143. Zhang, Kun, kzhang@bioeng.ucsd.edu, University of California San Diego
We are developing genome technologies based on single-molecule sequencing, single-cell manipulation/amplification, and chip-based synthesis and manipulation of complex DNA libraries. We apply these novel technologies to stem cell genomics, epigenomics, personal genomes and human common diseases.

144. Zhang, JianTing, jianzhan@iupui.edu, Indiana University
Translational control of gene expression and tumorigenesis, specifically, regulation of protein synthesis by eIF3 in cancer biology. Mechanism of drug resistance in cancer chemotherapy including fatty acid synthase, 14-3-3sigma and ABC transporters. Drug discovery targeting STAT3, survivin, and fatty acid synthase

145. Zhang, Jifeng, jifengz@umich.edu, University of Michigan
A major bottleneck in translational research on cardiovascular (CV) system and drug development is the lack of large animal models that mimic human pathology in the pre-clinical studies. Although the study of the CV system has benefited significantly from the use of gene-targeted and transgenic mouse models, small rodents do not accurately reflect human CV physiology. To generate more appropriate and useful animal models for better representing the human CV system, it would be beneficial to explore larger mammalian models for cardiovascular diseases (CVD). In contrast to mice and rats, the rabbits are better models for CVD research. The CV system of the rabbits is similar to that of humans. However, the use of large animal models has been hindered by the lack of embryonic stem cell lines, which are highly amenable for genetic manipulations to create various necessary research models. Because of the compelling need to address this bottleneck in CV research and drug development, we have built a leading research team with multiple approaches and expertise to produce gene targeted transgenic rabbits. To date, we have successfully generated CETP knockout (KO), LDLR KO, ApoCIII KO, and human ApoAII knock-in rabbits using Zinc Finger Nucleases (ZFN) or Transcription Activator-like Effector Nucleases (TALEN) mediated methods. We provide service to UM faculties to generate rabbit models for their research.

146. Zhang, Mingjie, mzhang@ust.hk, Hong Kong University of Science and Technology
My laboratory mainly focuses on neuronal structural biology. We aim to understand the molecular mechanisms governing synaptic signal transduction complex organization, dynamic regulations of synaptic complex assemblies, trafficking of proteins involved in synaptic signaling, as well as cell polarity.

147. Zhang, Peijun, pez7@pitt.edu, University of Pittsburgh
Structural Mechanisms of HIV-1 Capsid Assembly and Maturation Mature HIV-1 particles contain a conical-shaped capsid that encloses the viral RNA genome and performs essential functions in the virus life cycle. In mature virion, the assembled capsid structure is best described by a fullerene cone model that is made up from a hexameric lattice containing hexameric and pentameric capsid protein (CA). We obtained a cryo-EM structure of HIV-1 capsid assembly at 8Å resolution(1). The density map clearly delineates all the α-helical motifs within the structure. The structure allowed unambiguous modeling and refinement by large scale molecular dynamics simulations, resulting in all-atom models of the HIV-1 capsid comprising 64 million atoms. The models revealed new hydrophobic interactions at the inter-subunit trimer interface. Further, cryoEM structural analysis of immature intermediate CA-NC assemblies revealed a marked conformational difference at this trimer interface compared to mature CA assemblies(2). The critical role of the trimer interface in HIV-1 maturation was verified via chemical crosslinking.

148. Zhang, Su-Chun, zhang@waisman.wisc.edu, University of Wisconsin

149. Zhang, Xuewu, xuewu.zhang@utsouthwestern.edu, University of Texas Southwestern Medical Center
We study the signaling and regulation mechanisms of a group of cell surface receptors named plexins, which bind semaphorins and transduce signals to control processes such as axon guidance and angiogenesis. We use X-ray crystallography in combination with biochemical and cell biological approaches to gain structural/functional understanding of these receptors.

150. Zhang, Yi, yzhang@genetics.med.harvard.edu, Harvard Med School and Boston Children's Hospital
The lab is interested in the role of epigenetic modifications, particularly DNA and histone methylation in gene expression, embryonic development, stem cell reprogramming and brain learning and memory. Epigenetic modifications play important roles in diverse biological processes that range from regulation of gene expression, embryonic development, stem cell reprogramming, and human diseases. One of the epigenetic modifications is DNA methylation. Although enzymes responsible for DNA methylation have been well characterized, enzymes that responsible for active DNA demethylation in mammalian cells have remained elusive. Recent studies have suggested that the ten-eleven translocation (Tet)-mediated oxidation of 5mC might be part of the DNA demethylation process. In my talk, I will present our recent studies on understanding the mechanism and function of this enzymatic process.

151. Zhang, Zhong-Yin, zyzhang@iu.edu, Indiana University School of Medicine
My research spans the disciplines of chemistry and biology with an emphasis on the structure and function of protein tyrosine phosphatases (PTPs); PTP-mediated signaling; mechanistic enzymology; and inhibitor design. Our group performed experiments that defined the precise catalytic mechanism, rate-limiting step, and the transition state of the PTP-catalyzed reaction; probed the molecule basis for PTP substrate specificity using both structural and biochemical approaches; pioneered a novel paradigm for acquisition of potent and selective bidentate PTP inhibitors; elucidated the biochemical mechanism for the PRL3 phosphatase-mediated cell proliferation and metastasis; developed activity-based PTP probes that can be used to interrogate PTP activity in the whole proteome under both normal and pathological conditions; and defined the signaling mechanisms by which mPTPB phosphatase, an essential virulence factor of Mycobacterium tuberculosis, subverts the host immune responses. In addition, I have established a unique academic chemical genomic program to carry out high-throughput screening, structure-based design, combinatory chemistry to obtain small molecule PTP probes that not only serve as powerful tools to elucidate PTP-mediated signaling mechanisms but also find use for therapeutic development.

152. Zhang, Zhiguo, zhang.zhiguo@mayo.edu, Mayo Clinic
The long-term goals are to determine how epigenetic states are inherited during S phase of the cell cycle and how alterations in epigenetic inheritance contribute to human disease. My laboratory has discovered histone chaperone Rtt106, histone acetyltransferase Rtt109 and the role of acetylation of H3 lysine 56 in the replication-coupled nucleosome assembly and found that phosphorylation of H4 serine 47 impacts replication independent nucleosome assembly in human cells and has identified genes involved in maintenance of inactivated X-chromosome in female mice. Currently the lab focuses on determining how nucleosome assembly pathways are regulated in budding yeast and human cells and addressing to what extent factors involved in the regulation of nucleosome assembly are altered in human cancer and cellular senescence.

153. Zheng, Jie, jzheng@ucdavis.edu, University of California Davis
The main research focus of the Zheng laboratory is on the molecular mechanisms of temperature-sensing ion channels. Human senses ambient temperature changes with “heat sensors” located at the sensory nerve terminals under the skin. When these sensors are activated upon heating or cooling, they open up transmembrane ion pathways, generating an excitatory current that initiates signaling of sensory neurons to the central nervous system. Several members of the TRP (transient receptor potential) channel family are known to serve as heat sensor, hence are termed “thermoTRP” channels. How thermoTRPs activate in response to temperature changes remains mysterious. In our laboratory, we work on this problem with a multidisciplinary approach that combines biophysical methods (patch-clamp current recording of macroscopic and single-channel currents, site-directed fluorescence recording, intracellular Ca2+ imaging, etc.) with molecular biology, pharmacology, cellular and system physiology, and protein structure modeling.

154. Zheng, Hui, huiz@bcm.edu, Baylor College of Medicine
We investigated the role of Transcription Factor EB (TFEB), which has been shown to mediate coordinated expression of autophagy and lysosomal target genes, in Alzheimer¡¦s disease mouse models. We report that mild TFEB overexpression has no untoward side effect on wild-type mice and does not affect Aƒ Ò pathology. In contrast, TFEB potently reduced neurofibrillary tangles (NFT) and rescued behavioral and synaptic deficits and neurodegeneration in rTg4510 Tau transgenic mice. TFEB specifically targets phospho- and misfolded Tau species present in both soluble and aggregated fractions and this is associated with an upregulation of lysosomal genes. While we did not detect a global transcriptional activation of autophagy targets, we identify phosphatase and tensin homolog (PTEN) as a direct target of TFEB, thus discovering a novel TFEB-mediated autophagy through the PTEN-Akt-mTOR signaling pathway. The specificity and efficacy of TFEB in mediating phospho-Tau clearance makes it an attractive therapeutic target for diseases of tauopathy.

155. Zheng, Pan, pzheng@cnmc.org, Children's National Medical Center
Dr. Zheng’s laboratory is studying the signal transduction molecules in hematopoietic stem cells in different physiological aging and pathological conditions. One signal transduction pathway they are interested is TSC-mTOR pathway. TSC is named after a pediatric genetic disease: Tuberous Sclerosis Complex. This is a disease affecting 1 in 6000 newborns. The affected children will have non-malignant tumors in the brain, kidneys, heart, eyes and lungs. The affected children have mutations in either TSC1 gene or TSC2 gene. The Children’s National Medical Center has a special clinic to treat children with this disease. The TSC-mTOR pathway consists of about 20 different molecules inside the cells that sensing the amino acids, glucose and other nutrients and give the signals to cells to start multiple cellular processes, including transcription, translation, autophagy, glucose and glycogen metabolism. The researchers in Dr. Zheng’s lab are trying to understand the role of each molecule in making new blood cells (hematopoiesis), new immune cells (thymopoiesis) and tumor formation. Bone marrow transplantation is the special tool to study the hematopoiesis. Their research work has found that TSC-mTOR pathway plays important roles in maintaining hematopoietic stem cell self-renewal ability and control the stem cell differentiation to different types of blood cells. We found that certain therapeutic reagents may rejuvenate aging hematopoietic process. Dr. Zheng’s laboratory also works on tumor immunology. They are working on activating our own immune system for better surveillance to prevent tumor formation.

156. Zhong, Qing, qing.zhong@utsouthwestern.edu, Univ of Texas Southwestern Medical Center
My lab is interest in dissecting the regulatory mechanism of autophagy, apoptosis and necrosis. One small-molecular compound, we named necrocide 1, kills a panel of breast, ovary and pancreatic cancer cells at nano molar concentrations, by inducing programmed necrosis rather than apoptosis or autophagy. A kinome siRNA screening identified several kinases involved in necrocide 1 induced necrosis. Biochemical analysis revealed that these kinases interact with PGAM5 and mitochondria fission/fusion machinery. We hypothesize that these kinases have newly recognized roles in programmed necrosis and tumorigenesis.

157. Zhong, Weimin, weimin.zhong@yale.edu, Yale University
Asymmetric cell division is a process by which a cell divides to produce two different daughter cells. Such divisions are an attractive means for stem cells to balance the needs of self-renewal and differentiation during organogenesis and tissue maintenance. We have been probing the behavior of stem/progenitor cells during neurogenesis in mice by manipulating the segregation of numb proteins, whose asymmetric presence can allow the two daughter cells to choose different fates after an asymmetric division. We present evidence that stem-cell numbers are strictly controlled in vivo and that Numb-mediated asymmetric cell division is a mechanism shared by stem cells in many tissues for their progeny to choose between self-renewal and differentiation.

158. Zhong, Xiaobo, xiaobo.zhong@uconn.edu, University Of Connecticut
Pharmacogeomics and personalized medicine

159. Zhou, Bin-Bing, binbing_s_zhou@yahoo.com, Shanghai Jiaotong University School of Medicine
We have been interested in cancer drug discovery and drug resistance mechanism over the years, and has contributed to one approved, one phase III and two Phase II clinical trial anti-cancer agents. Our translational research interests range from DNA damage response, ErbB signaling, Notch signaling to cancer stem cells with three Nature series and two Cancer Cell publications of total more than 3000 citations.

160. Zhu, Heng, hzhu4@jhmi.edu, Johns Hopkins School of Medicine
DNA methylation has been generally considered to prohibit transcription factor (TF) recruitment, resulting in transcription suppression. Here, we used a protein microarray-based approach to systematically survey the entire human TF family and found numerous TFs with methylated mCpG-dependent DNA-binding activities. Interestingly, some TFs exhibit specific binding activity to methylated and unmethylated DNA motifs of distinct sequences. To elucidate the underlying mechanism, we focused on KLF4, and decoupled its mCpG- and CpG-binding activities via site-directed mutagenesis. Furthermore, KLF4 binds specific methylated motifs in human embryonic stem cells in vivo. Our study suggests that mCpG-dependent TF binding activity is a widespread phenomenon and provides a new framework to understand the role and mechanism of TFs in epigenetic regulation of gene transcription.

161. Zhu, Jinsong, jinsong.zhu@me.com, National Center for Nanoscience & Technology
biosensors, high throughput screening, biomarker, drug reposition, cocktail therapy

162. Zhu, Yuan, yzhu@childrensnational.org, Children's National Medical Center
We are interested in understanding molecular and cellular mechanisms underlying the development of normal neural stem and progenitor cells as well as tumorigenesis in the nervous system. We are using the mouse as a model system to develop genetic engineering mouse (GEM) tumor models, which recapitulate human nervous sytem tumors both genetically and phenotypically. Particularly, we have been focused on the role of tumor suppressor genes in the nervous system.

163. Zong, Hui, hz9s@virginia.edu, University of Virginia
Efforts to develop molecularly targeted therapy toward cancer have encountered great setbacks due to drug resistance and tumor relapses. The toughness of tumor cells is not a new invention rather stems from the robustness of developmental programs. Our lab is focused on studying initiation process of tumorigenesis to reveal cellular mechanisms of malignancy. Using a mouse genetic system termed MADM (Mosaic Analysis with Double Markers) that limits TSG inactivation in very few GFP-labeled cells, we study brain tumor development in vivo at single cell resolution. We have found that transformation by TSG mutations is cell-type specific, suggesting permissive signaling context, and that transformed cells could trans-differentiate into niche cells to support tumor growth as a new mechanism for cancer robustness.

164. Zou, Lee, zou.lee@mgh.harvard.edu, Harvard Medical School
My lab is interested in the mechanisms by which human cells sense DNA damage and maintain genomic stability. We are also interested in understanding how the DNA damage response is altered in cancers, and how these alterations can be exploited in cancer therapy.

165. Zou, Weiping, wzou@med.umich.edu, University of Michigan
The research interest of the Zou lab is in tumor immunopathology and immunotherapy, with an emphasis on the cross-talk among immune cell subsets, stromal cells, tumor cells and tumor stem cells in the tumor microenvironment, and its impact on tumor immunity, tolerance and therapy.

166. Zou, Yue, zouy@etsu.edu, East Tennessee State University
ATM (ataxia telangiectasia mutated) and ATR (ATM and Rad3-related) are the two major DNA damage checkpoint kinases. Intriguingly, unlike ATM whose deficiency promotes carcinogenesis, inhibition of ATR has been shown to suppress UV carcinogenesis in mice. We hypothesized that ATR might contain an anti-apoptotic activity unrelated to its checkpoint functions. Indeed, here we show that besides its nuclear checkpoint functions, ATR in the cytoplasm is an anti-apoptotic protein. Upon UV damage, cytoplasmic ATR translocates to mitochondria, blocking the recruitment of pro-apoptotic Bcl-2–associated X (Bax) protein to mitochondria and preventing the loss of mitochondrial membrane potential and apoptosis. Consistently, Bax-depletion significantly reduced the effect of ATR;. Remarkably, the cytoplasmic ATR exhibits no checkpoint kinase activity, a hallmark function of nuclear ATR. Inhibition of ATR's kinase activity or silencing of ATRIP and Chk1 failed to revoke the negative impact of ATR on DNA damage-induced Bax localization to mitochondria. Furthermore, knocking down p53 did not change the effect of ATR on mitochondrial; indicating that the anti-apoptotic role of ATR is independent of p53. Our results reveal a novel checkpoint-independent anti-apoptotic function of ATR at mitochondria in the cellular response to UV damage.

CBIS board member candidates for re-election

Hao Wu (candidate for Vice President)

Xiangdong William Yang

Mingjie Zhang

Pan Zheng

Weimin Zhong (candidate for President)

CBIS board member candidates for new election

Yibin Kang

Guo-Min Li

Lei Li, Ph.D.

Xin Sun, Ph.D.

Wen Xie, MD, PhD

Wen-Cheng Xiong

Yingzi Yang, PhD

Yi Zhang

Weiping Zou

Yimin Zou

Positions for election: **President (1)**

Vice president (1)

Board members (9)

Hao Wu, Ph.D.

Personal Statement (re-election, candidate for Vice 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 two years and glad to have learned some of the operational logistics of the society. 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	BS (Biology)	Peking University, Beijing, China
1985-1988	MD candidate (Medicine)	Peking Union Medical College, Beijing, China
1988-1992	PhD (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 Medical College of Cornell University.
2001-2003	Associate Professor of Biochemistry, Weill Medical College of Cornell University.
1997-2001	Assistant Professor of Biochemistry, Weill Medical College of Cornell University.

Purdue University Distinguished Science Alumni Award, 2013
Elected AAAS Fellow, 2013
NIH Merit Award, 2012-2022
Editorial Board, F1000 Research, 2012-
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
Member of Gamma Sigma Delta
Top in class and outstanding academic achievement, Peking Union Medical College, 1982-1988
Highest entering grades to Peking Union Medical College in National College Entrance Examination, 1982
International Math Olympiad, 1981

Xiangdong William Yang, M.D., Ph.D.

Personal Statement (re-election)

It has been a great pleasure for me to serve on the board of CBIS in the past two years. I have learned tremendously from the leadership on the board, especially our President Linheng Li and Vice President Weimin Zhong, as well as the fellow board members. I have contributed to the society by helping to organize the neuroscience session at our bi-annual meeting. If I have the honor to be re-elected as a board member again, I will strive to serve all of our members to the best of my ability. I will particularly focus on recruiting new members in the area of neuroscience in both US and China, and to facilitate Sino-US collaboration in basic and translational neuroscience research.

Education/Training

1985-87	no degree (Biochemistry)	Peking University
1987-91	BS/MS (MB and B)	Yale University
1993-1998	PhD (Neuroscience)	Rockefeller University
1991-93,1999-2000	MD	Weill Medical College, Cornell Univ.
2000-01	Residency	New York Presbyterian Hospital
1998-99, 2001-02	Postdoc	Rockefeller University

Positions and Honors

Positions and Employment

1998-1999 & 2001-2002 Postdoctoral fellow (Mentor, Nathaniel Heintz), Rockefeller University;
2000-2001 Medicine Intern, New York-Presbyterian Hospital/Cornell Medical Center;
2002-2008 Assistant Professor, Department of Psychiatry & Biobehavioral Sciences, David Geffen School of Medicine at UCLA; Member, Center for Neurobehavioral Genetics, Semel Institute; Member, Brain Research Institute, UCLA;
2008- 2011 Associate Professor (Tenured), Department of Psychiatry & Biobehavioral Sciences; Member, Center for Neurobehavioral Genetics, Semel Institute; Member, Brain Research Institute, David Geffen School of Medicine at UCLA.
2011-Present Professor, Department of Psychiatry & Biobehavioral Sciences; Member, Center for Neurobehavioral Genetics, Semel Institute; Member, Brain Research Institute, David Geffen School of Medicine at UCLA.

Professional License

2001-present New York State Physician's License

Honors

1991 *Summa cum laude*, Distinction in the major, Dept. of Mol. Biophys. & Biochem., Yale University
1991 *Sigma Xi* Honor Scientific Research Society
1999-2003 Research Award, Cure Huntington Disease Initiative, Hereditary Disease Foundation
2003 Stein-Oppenheimer Award, David Geffen School of Medicine at UCLA
2003-2005 Research Award, Hereditary Disease Foundation
2008-2009 Michael J. Fox Foundation for Parkinson's Research: Rapid Response Innovation Award
2009-2012 McKnight Neuroscience of Brain Disorders Award
2011-2016 The Carol Moss Spivak Scholar in Neuroscience from UCLA Brain Research Institute
2013 Center for Excellence in Education Award to Outstanding Alumni in STEM and Business

Service to Professional Organizations

2005-2009 Scientific Advisory Board Member, Hereditary Disease Foundation
2011-present Scientific Advisory Board Member, Hereditary Disease Foundation
2011-1013 Board Member, Chinese Biological Investigators Society (CBIS)
2005-2007 *Ad hoc* member, Cell Death and Neuronal Injury Study Section (CDIN-D), CSR/ NIH
2008-2011 Chartered Member, Cell Death and Neuronal Injury Study Section (CDIN-D), CSR/ NIH
2005 Faculty member, Neurogenetics Branch, Faculty of 1000 Medicine
2006 Editorial Board Member, *Molecular Neurodegeneration*
2012 Neuroscience Section Editor, *Frontiers in Biology*

Mingjie Zhang, Ph.D.

Personal statement (re-election)

Being based in Hong Kong, I have unique opportunities to connect colleagues in North America with those in Asia Pacific Regions so as to enhance the impact of CBIS. Being legally inside the country but not being restrained by its bureaucratic system, I have some advantages of better connecting our society with the scientific community in our motherland. Finally, I will continue to spend time in influencing younger generation scientists (graduate students and undergraduate students in China in particular), as I believe that this is the best investments of my time in scientific education.

Education and Training:

- 1984-1988	B.Sc. (Chemistry)	Fudan University (Shanghai, P.R. China)
- 1989-1993	Ph.D. (Biochemistry)	University of Calgary (Calgary, Canada)
- 1994-1995	Postdoctoral Fellow,	Ontario Cancer Institute, University of Toronto (Canada)

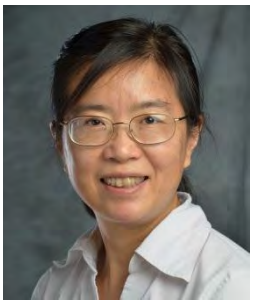
Professional Experience:

- Graduate student (Ph.D.), Department of Biological Sciences, 1989-1993 University of Calgary (Supervisor Dr. H. J. Vogel)
- Postdoctoral Fellow, Department of Biological Sciences, Jan. 1994- Jun. 1994 University of Calgary (Supervisor Dr. H. J. Vogel)
- Postdoctoral Fellow, Division of Structural and Molecular Biology Jun. 1994- Mar. 1995 Ontario Cancer Institute (Supervisor Dr. M. Ikura)
- Assistant Professor, Department of Biochemistry 1995-1999 Hong Kong University of Science and Technology
- Associate Professor, Department of Biochemistry 2000-2004 Hong Kong University of Science and Technology
- Professor, Department of Biochemistry 2004-2008 Hong Kong University of Science and Technology
- Chair Professor, Department of Biochemistry 2008-2012 Hong Kong University of Science and Technology
- Kerry Holdings Professor of Science, Division of Life Science Dec. 2012- Hong Kong University of Science and Technology
- Senior Fellow, Institute for Advance Study, HKUST Apr. 2012-

Awards and Honors:

- National Cancer Institute of Canada (NCI) Postdoctoral Fellowship Award 1994-1995
- Natural Sciences and Engineering Research Council of Canada (NSERC) Postdoctoral Fellowship Award (declined due to overlapping awards) 1994
- Medical Research Council of Canada (MRC) Postdoctoral Fellowship Award (Ranked the first out of 270 applications) (declined due to overlapping awards) 1994
- The Cancer Research Society Incorporated Canada Postdoctoral Fellowship Award (declined due to overlapping awards) 1994
- President's List, Natural Sciences and Engineering Research Council of Canada (NSERC) Doctoral Prize 1994
- Outstanding Overseas Young Scientist Award by the Natural Science 2002 Foundation of China (海外杰出青年基金奖)
- The Croucher Foundation Senior Research Fellow Award 2003
- The State Natural Science Award (Second Prize) 2006 (2006 年国家自然科学奖二等奖)
- The Ho Leung Ho Lee Foundation Science and Technology Advancement Award 2011 (2011 年何梁何利基金科技进步奖)
- Elected as a Member of Chinese Academy of Science 2011 (2011 年增选为中国科学院院士)

Pan Zheng, M.D., Ph.D.



Personal Statement (re-election)

I have been attending CBIS biennial meetings since 1989 Boston Meeting. I always treasure the experience to meet and to know the most talented and the top notch Chinese scientists in these meetings. I am currently serving the first term of CBIS board member. It is a great honor for me working together with other CBIS members to fulfill the CBIS mission in promoting research in life sciences and medicine and facilitating the interactions among Chinese scientists.

My laboratory is studying the signal transduction molecules in hematopoietic stem cells in different physiological aging and pathological conditions. The TSC-mTOR pathway senses the amino acids, glucose and other nutrients and gives the signals to cells to start multiple cellular processes, including transcription, translation, autophagy, glucose and glycogen metabolism. Our research work has found that TSC-mTOR pathway plays important roles in maintaining hematopoietic stem cell self-renewal ability and control the stem cell differentiation to different types of blood cells. We found that certain therapeutic reagents may rejuvenate aging hematopoietic process. My laboratory also works on tumor immunology, focusing on activating immune system for better surveillance to prevent tumor formation.

Education/Training:

1979 – 1987 MD. Peking Union Medical College, Beijing, P.R. China
1987 -- 1989 Resident, Internal Medicine, PUMC Hospital, Beijing, P.R. China
1989 -- 1994 PhD. Yale University, New Haven, CT
1994 -- 1998 Resident, Anatomic and Clinical Pathology, Department of Pathology, New York University Medical Center, New York, NY

Positions

1998 -- 2003 Assistant Professor, Department of Pathology, Ohio State University, OH
2003 -- 2006 Associate Professor, Department of Pathology, Ohio State University, OH
2006 -- 2011 Associate Professor, Department of Surgery, Department of Pathology, University of Michigan, Ann Arbor, MI.
2011-- 2013 Professor, Department of Surgery, Department of Pathology, University of Michigan, Ann Arbor, MI.
2013 – present Senior Investigator and McKnew Chair for Cancer Biology, Center for Cancer and Immunology Research, Children's Research Institute, Children's National Medical Center, Washington, DC

Honors and Services

1989-1993 Yale Fellowship
1991 Cold Spring Harbor Laboratory Summer Student Fellowship
2006 American Cancer Society Research Scholar Award
2003-2007: NIH study section, ZRG1 F07. Immunology.
2008-2013: DOD, Breast Cancer Research Program, Section of Cell Biology, Immunology, reviewer.
2009-2013: AIRC - Italian Association for Cancer Research, reviewer.
2010-2012. NIA Program Project Review Panel.
2012-2013: National Science Foundation fellowship panelist.
2011–2013 Secretary, Chinese Biological Investigators Society.

Weimin Zhong, MD, PhD.

Personal Statement (re-election, candidate for President)

If elected, I will continue and further strengthen the tradition of CBIS as a platform for scientific exchange and friendship among our members and as a welcoming home for next generation of scientists working both outside and inside of China. Working with the board and our members, I will explore avenues to deepen our collaboration with scientists and policy makers in China to facilitate the ongoing reform efforts there in undergraduate and graduate education and in strategic planning and funding of biological and biomedical sciences.

Education/Training

1984	BS (Biology/Premed)	Peking University, Beijing, China
1988	MD (Medicine)	Peking Union Medical College, Beijing, China
1993	PhD (Molecular Cell Biology)	The Rockefeller University, New York, USA
1994-98	Postdoc (Dev. Neurobiology)	University of California, San Francisco, USA

Positions and Honors

Positions

1981–1984 Undergraduate Student (Premed), Department of Biology, Peking University
1984–1988 Medical Student, Peking Union Medical College
1988–1993 Ph.D. Student, The Rockefeller University
1994–1998 Postdoctoral Fellow, Howard Hughes Medical Institute & Department of Physiology University of California, San Francisco
1999–2004 Assistant Professor, Department of Molecular, Cellular and Developmental Biology, Yale University
2004–present Associate Professor (without term since 2008), Department of Molecular, Cellular and Developmental Biology, Yale University

Honors and Services

1986 PUMC Friends Fund Award
1996–1998 Postdoctoral Fellowship, American Cancer Society
2001 Hellman Family Fellowship
2001 American Cancer Society Research Scholar Award ($949,000; gratefully declined)
2002–2003 Junior Faculty Fellowship, Yale University
2003–2005 Member, NIH Fellowship Review Group F03A
2003–2006 Member, Overseas Scientific Review Board, National Science Foundation of China
2004–2006 *Ad hoc* member, NIH MCDN6/Neurogenesis and Cell Fate (NCF) Study Section
2005 *Ad hoc* member, Development, Differentiation and Cancer Peer Review Committee, American Cancer Society
2005–2009Member, Board of Directors, Chinese Biological Investigators Society
2006 *Ad hoc* reviewer, National Science Foundation
2006–2010 Member, NIH NCF Study Section
2008 *Ad hoc* member, NIH CHHD-C Study Section
2009, 2010 *Ad hoc* member, NIH NICHD IDD Center Review Panel
2011 *Ad hoc* member, NIH DEV1 Study Section
2011 *Ad hoc* reviewer, Association for International Cancer Research, UK
2011 *Ad hoc* reviewer, Medical Research Council (England) Career Development Award
2012 *Ad hoc* reviewer, Qatar National Research Fund
2012 *Ad hoc* member, NIH DEV2 Study Section
2007–2010 Section Editor, *Neuroscience*
2011–2012 Guest editor, *Current Opinion in Cell Biology* special issue “Membranes and Organelles”
2011–present Member, Faculty *of* 1000
2014-2019 Member, Editorial Board, The Journal of Biological Chemistry
2011–present Vice President, Chinese Biological Investigators Society
2011–Present Scientific Advisory Panel, The Smith-Magenis Research Foundation

Yibin Kang, Ph.D.

Personal Statement

During Dr. Kang's career development as a young scientist in the field of metastasis research, he has benefited from the generous support and mentoring of many members of the Chinese Biological Investigator Society (CBIS). If he has the honor to be elected as a board member of the CBIS, he will help promote the society among young Chinese scientists, and facilitate their career development in the field of cancer research. He will also contribute to increasing influence of CBIS both in China and in the US.

Education

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

Professional Experience

- 1993-1995 Undergraduate Research Assistant at Dr. **Jianhua Chai**'s Laboratory, Department of Genetics, Fudan University, Shanghai, China
- 1996-2000 Graduate Research Assistant at Dr. **Bryan R. Cullen**'s Laboratory Department of Genetics and Howard Hughes Medical Institute, Duke University Medical Center, Durham, NC
- 2000-2004 Postdoctoral Research Associate at Dr. **Joan Massagué**'s Laboratory Cancer Biology and Genetics Program and Howard Hughes Medical Institute, Memorial Sloan-Kettering Cancer Center, New York
- 2004.9-2010.2 Assistant Professor, Princeton University
- 2010.2-2012.7 Associate Professor (Tenured)
- 2012.7 Professor
- 2012.7-present Warner-Lambert/Parke-Davis Professor of Molecular Biology (Endowed) Department of Molecular Biology, Princeton University, Princeton, NJ
- 2004- Member, Cancer Institute of New Jersey, New Brunswick, NJ

Honors and Awards

- 1990 First prize, National High School Chemistry Competition, China
  Member, National Select Team for the 22nd International Chemistry Olympiad
- 1991-1995 Fudan University Scholarships: People's Scholarship (91-95); Mao Cheng-Si Scholarship (93); Xu Zeng-Shou Scholarship (94)
- 2001-2004 Irvington Institute Postdoctoral Fellowship for Immunological Research
- 2004 AIMM-ASBMR John Haddad Young Investigator Award
- 2004 Memorial Sloan-Kettering Cancer Center Annual Postdoctoral Research Award
- 2005 The Padget Foundation Young Investigator Award
- 2005 American Cancer Society Research Scholar Award
- 2006 Department of Defense Breast Cancer Research Program Era of Hope Scholar Award
- 2009 Champalimaud Investigator, Champalimaud Foundation, Portugal
- 2010 Inaugural Oudang Distinguished Lectureship, Korean Pharmaceutical Society
- 2011 Vilcek Prize for Creative Promise in Biomedical Science
- 2012 AACR Award for Outstanding Achievement in Cancer Research
- 2012 Salvador E. Luria Lectureship, Massachusetts Institute of Technology
- 2013 Humphrey Oei Distinguished Professor, National Cancer Centre Singapore and Duke-NUS Graduate Medical School
- 2013 Sir Yue-Kong Pao Chair Professor, Zhejiang University, China

Guo-Min Li, Ph.D.

Personal Statement

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

My research interest focuses on DNA mismatch repair (MMR), a critical cellular mechanism that ensures replication fidelity by correcting mispairs created during DNA replication. I discovered MMR defects in hereditary and sporadic colorectal cancers that display microsatellite instability (MSI) during my postdoc training with Paul Modrich at Duke University. I am responsible for identifying the vast majority of proteins required for human MMR, which has led to our eventual effort to reconstitute the human MMR reaction in vitro. These studies have laid down a foundation for understanding the molecular basis of MMR and its role in cancer avoidance. My laboratory also discovered the apoptotic function of the MMR system, in which MMR proteins recognize a variety of DNA lesions and trigger apoptosis in cells with severe DNA damage. These observations establish a general concept that the MMR system acts as a DNA damage sensor, implicating the system in cancer chemo- and radiation-therapy. Recently, my laboratory has shown that the epigenetic histone mark H3K36me3 (trimethylated H3 lysine 36) is required for MMR in vivo by recruiting the MutSα mismatch recognition protein to chromatin through its interaction with the PWWP domain of MutSα and that cells lacking H3K36me3 display a classical MMR-deficient phenotype (e.g., MSI). This study reveals that epigenetic histone marks regulate genome-maintenance systems, suggesting that in addition to gene mutations, abnormal histone modifications can also cause genome instability and cancer development. In summary, I have a demonstrated record of successful and productive research projects in the area of DNA repair in cancer, and my expertise and experience has prepared me to lead the project proposed in this application.

Education

1982	BS (Biology)	Wuhan University, Wuhan, China
1985	MS (Cell Biology)	Wuhan University, Wuhan, China
1991	PhD (Biochemistry)	Wayne State University, Detroit, MI
1991-5	Post-doc (Biochem)	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-present	Professor, Department of Toxicology, University of Kentucky College 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-present)
Chang Jiang Scholar, The Ministry of Education of China (2006)

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 begin 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

Xin Sun, Ph.D.

Personal Statement:
I am a developmental biologist at University of Wisconsin-Madison, and my laboratory studies lung development. I obtained my bachelor's degree at Fudan University in 1989. I began my training in Developmental Biology as a graduate student in Dr. Spyros Artavanis-Tsakonas's laboratory at Yale University, where I studied Notch signaling in *Drosophila*. After I obtained my Ph.D. in 1997, I went on to postdoctoral training in Dr. Gail Martin's laboratory at University of California at San Francisco. There I studied FGF signaling in gastrulation and limb development. I started my laboratory at UW-Madison in 2002. After tenure, I transitioned from working on limb development to focusing on lung development and disease. Since 2007, I have devoted time each year to teach in China, both in graduate as well as in undergraduate courses. I am also currently the Director of the Cold Spring Harbor Laboratory summer course on Mouse Development, Stem Cells and Cancer.

Academic Appointments

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

Education and Training

1985-1989	B.S. (Biochemistry)	Fudan University, China.
1990-1997	Ph.D.	Yale University.advisor Dr. Spyros Artavanis-Tsakonas.
1997-2002	Postdoctoral Fellow	UCSF, Dr. Gail Martin at UCSF.

Fellowships and Awards

1990-1997	Yale University graduate fellowship.
1997-2000	American Cancer Society postdoctoral fellowship.
2001-2006	Burroughs-Wellcome career award in biological sciences.
2003-2006	March of Dimes Basil O'Conner award.

National Service

SDB International Annual Meeting organizing committee, San Francisco, 2005.
NIDDK beta Cell Consortium grants reviewer, 2011.
NHLBI workshop on lung development, 2011.
NIH Lung Injury and Repair study section ad hoc reviewer, 2011, 2012.
American Thoracic Society annual conference program committee member, 2012-2014.
Director, Cold Spring Harbor Laboratory mouse embryology course, 2011-2014.
Invited to serve as regular member on NIH Lung Injury and Repair study section, 2015-2019.

Wen Xie, MD, PhD

Personal Statement

I'm willing to serve as a CBIS board member. I'm committed to help the CBI Society to achieve its mission in promoting advancement of the frontiers in life sciences by providing Chinese and their peers of other nationalities with a platform of professional interactions and collaborations. I'm also enthusiastic to support the CBI Society's commitment to facilitate the development of life sciences in China.

Education

1993 – 1997 Ph.D. (Cell Biology) University of Alabama at Birmingham, USA
1985 – 1991 M.D Peking University Health Science Center (former Beijing Medical University)

Professional Experience

2011 – present: Professor with Tenure and Director, Center for Pharmacogenetics, Department of Pharmaceutical Sciences, University of Pittsburgh. Joseph Koslow Endowed Chair in Pharmaceutical Sciences
2007 – 2010: Associate Professor with Tenure and Associate Director, Center for Pharmacogenetics, Department of Pharmaceutical Sciences, University of Pittsburgh.
2003 – present: Assistant Professor (2003-2007), Associate Professor (2007-2010), Professor (2011-present), Department of Pharmacology and Chemical Biology, University of Pittsburgh School of Medicine (secondary appointment).
2002 – 2006: Assistant Professor, Center for Pharmacogenetics and Department of Pharmaceutical Sciences, University of Pittsburgh School of Pharmacy.
1998 – 2002: Postdoctoral Fellow with Dr. Ronald M. Evans, Howard Hughes Medical Institute, Salk Institute for Biological Studies, USA.
09/1993 - 12/1997: Graduate Research Assistant, Department of Cell Biology/Medicine, University of Alabama at Birmingham, USA. (Major Advisor Dr. Jeffrey E. Kudlow)
08/1991 - 08/1993: Research Associate, Division of Biochemistry, Institute of Sports Medicine, Beijing Medical University, Beijing, China.

Major Honors and Awards:

2013-: Guest Professor, Zhejiang University, Hangzhou, Zhejiang, China
2012: Distinguished alumnus, Peking University Health Science Center/Beijing Medical University
2012-: Joseph Koslow Endowed Chair in Pharmaceutical Sciences, University of Pittsburgh
2012-: Guest Professor, Shantou University School of Medicine, Shantou, Guangdong, China.
2012-: Guest Professor, Third Military Medical University, Chongqing, China.
2011-: Guest Professor, Peking University Health Science Center, Beijing, China.
2011: Visiting Professorship, University of Cagliari, Cagliari, Italy.
2009: American Society for Pharmacology and Experimental Therapeutics (ASPET) Division for Drug Metabolism Early Career Achievement Award.
2008: *James R. Gillette* International Society for the Study of Xenobiotics (ISSX) North American New Investigator Award.
2008: University of Pittsburgh Chancellor's Distinguished Research Award.
2008-: Guest Professor, Beijing Normal University, Beijing, China.
2008-: Guest Professor, Sun Yet-sen University, Guangzhou, China.
2003-: Guest Professor, Capital University of Medical Sciences, Beijing, China
1998: The Joseph Reeves Award for Excellence in Research by a Post-Doctoral Scholar, University of Alabama at Birmingham.
1996: The Samuel B. Barker Award for Excellence in Research by a Graduate Student, University of Alabama at Birmingham.

Wen-Cheng Xiong, M.D., Ph.D.

Personal Statement

1. I have enjoyed many activities organized by CBIS, such as scientific meetings. Wonderful service jobs have been done by previous members of the CBIS committee. It is unfair if I only enjoyed the services provided, but not contributing to the society. It is time for me to "pay" back.

2. CBIS provides a great platform for Chinese biologists from different fields to interact and exchange ideas. I have wide interests in research, and enjoy interacting with biologists from different fields, including neurobiology, cell biology, cancer biology, and bone biology fields.

Education:

1984	MD	The Third Military Medical University,Chongqin, China
1989	MS (Neuopharmacology)	University of Arizona, Tucson, Arizona
1994	PHD (Neuroscience)	Johns Hopkins Medical School, Baltimore, Maryland

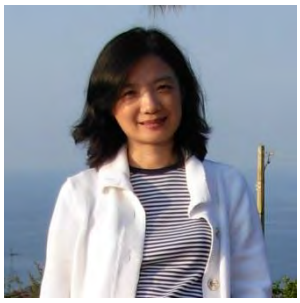
Academic Appointments:

1983-1984 Intern, First Affiliated Hospital, The Third Medical University, ChongQing, China
1984-1987 Graduate Research Assistant, Institute of Pharmacology & Toxicology, Beijing, China. Advisor: Prof. Jin-Chu Yan
1987-1989 Graduate Research Assistant, Department of Pharmacology & Toxicology, College of Pharmacy, University of Arizona, Tucson, Arizona. Advisor: Dr. David Nelson
1989-1994 Graduate Research Assistant, Department of Biological Chemistry & Neuroscience, Johns Hopkins Medical School, Baltimore, Maryland. Advisor: Dr. Craig Montell
1994-1999 Postdoctoral Fellow, Department of Microbiology, University of Virginia School of Med., Charlottesville, VA. Mentor: Dr. Tom Parsons
1999-2004.08 Assistant Professor, Department of Pathology, University of Alabama at Birmingham, Birmingham, Alabama
2004.08-2008.07 Associate Professor, Institute of Molecular Medicine & Genetics and Department of Neurology, Medical College of Georgia, Augusta, Georgia
2005.10- Weiss Research Professor, Institute of Molecular Medicine & Genetics & Department of Neurology, Medical College of Georgia, Georgia Health Sciences University, Augusta, Georgia
2008.07-present Professor, Institute of Molecular Medicine & Genetics and Department of Neurology, Medical College of Georgia, Georgia Health Sciences University, Augusta, Georgia

Awards/Honors:

1995-1998 National Research Service Award (NIH, USA)
4/2007 Outstanding Young Basic Science Faculty Award (MCG)
4/2009 Distinguished Research Award (MCG)

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

Yi Zhang, Ph.D.

Personal Statement

As a member of CBIS, I feel that the organization can play a positive role in many aspects of our community. It not only provides an avenue for its members to interact and foster collaboration, but also can provide suggestions and guidance for career development of its members, particularly the junior and young faculties. Furthermore, it can also play an important role in scientific exchange with scientists in China and help improve the scientific environment. I am willing to serve and contribute to the community to make CBIS a strong organization.

I started my lab in 1999 and have been continuously funded through NIH, private foundations, and now, as a Howard Hughes Investigator. I have the necessary expertise and skill to perform these studies as I am well trained in signal transduction, chromatin modification, and biochemistry. I am interested in how epigenetic-mediated dynamic changes in chromatin structure affect gene expression, cell lineage commitment, stem cell pluripotency and self-renewal, cancer and diabetes. My long-term goals are to apply this basic science research to studies of human diseases, like diabetes and leukemia, where epigenetic mutations are thought to affect and distort function.

Education

1984	BS (Biophysics)	China Agricultural Univ. College of Biological Sciences
1987	MS (Biophysics)	China Agricultural Univ. College of Biological Sciences
1995	PhD (Molecular Biophysics)	Florida State Univ. Institute of Molecular Biophysics

Positions

8/99 – 6/04	Assistant Professor, Dept. of Biochemistry & Biophysics, UNC at Chapel Hill
7/04 – 11/05	Associate Professor, Dept. of Biochemistry & Biophysics, UNC at Chapel Hill
12/05 - 06/12	Professor, Dept. of Biochemistry & Biophysics, UNC at Chapel Hill
05/09 - 06/12	Kenan Distinguished Professor, University of North Carolina at Chapel Hill
09/05 – pre	Investigator, Howard Hughes Medical Institute
07/12 – pre.	Fred Rosen Professor of Pediatrics and Professor of Genetics at Harvard Medical School; Senior Investigator Program in Cellular and Molecular Medicine at Boston Children's Hospital

Honors

2003	Gertrude B. Elion Cancer Research Award, AACR
2004	Hettleman Prize for Artistic and Scholarly Achievement, UNC-Chapel Hill
2005	Investigator, Howard Hughes Medical Institute
2008	Top 10 authors of high-impact papers by ScienceWatch (http://scientific.thomson.com/press/2008/8438330/)
2009	Kenan Distinguished Professorship, University of North Carolina-Chapel Hill
2012	Fred Rosen Chair, Harvard Medical School & Boston Children's Hospital

Weiping Zou, M.D., Ph.D.

Personal Statement

Weiping Zou obtained his MD from Tongji Medical School in Wuhan, China, and PhD from the University of Paris in France followed by postdoctoral training at Baylor Institute for Immunology Research in Dallas, Texas. After several years as a faculty member at Tulane University School of Medicine, he joined the University of Michigan Medical School in 2006. He is the Charles B. de Nancrede Professor of Surgery, Immunology and Biology, and Co-Director of the tumor immunology and host defense program at the University of Michigan. He has served on study sections for the National Institutes of Health, the Department of Defense and ten other scientific agencies and is/was on the editorial board of six immunological and cancer journals.

His research interests are in tumor immunopathology and immunotherapy, with an emphasis on the cross-talk among immune cell subsets, tumor cells and tumor stem cells in the tumor microenvironment and its impact on tumor immunity, tolerance and therapy. Over the past few years, his research team has achieved important insights into cancer immunopathogenesis in patients with cancer. His prior research efforts demonstrate that the tumor microenvironment is comprised of immune cells that have been reprogrammed by active tumor-mediated processes to defeat tumor-specific immunity and promote tumor growth in a highly effective manner. These studies have helped define the nature of immune responses in the tumor microenvironment and provide significant new insights into designing novel immune therapies to treat patients with cancer. As such, the Zou laboratory is one of the most cited research teams in the field of immunology.

Dr. Zou foresees a future in which CBIS faculty will need to place greater emphasis on patient-oriented basic and clinical research within their research programs in order to compete effectively for diminishing NIH funds and to accelerate therapeutic translation. Consequently, he believes that CBIS should duly promote and support collaboration in translational research efforts among its faculty. Dr. Zou is also aware of the increasing importance of the Chinese Investigators to medical research and advocates that CBIS pursue more efforts to identify valuable opportunities for collaboration with Chinese institutions. If elected, he will strive to uphold the standards of excellence and equitability on CBIS.

Education

1990	MD (Medicine)	Tongji Medical School
1997	PhD (Immunology)	University of Paris
2001	Postdoc (Immunology)	Baylor Research Institute

Professional Experience

1990-1993	Clinical fellow at Tongji University Hospital, China
1993-1994	Visiting scientist at Institut Pasteur; Institut Paris-Sud sur les Cytokines, France
1994-1997	PhD student at University of Paris, Institut Paris-Sud sur les Cytokines, INSERM U131, France
1997-1998	Postdoctoral fellow at Institut Paris-Sud sur les Cytokines, INSERM U131, France
1999-2001	Post-doctoral fellow at Baylor Institute for Immunology Research, Dallas, TX
2001-2004	Assistant Professor at Tulane University, New Orleans, LA
2004-2006	Associate Professor at Tulane University, New Orleans, LA
2006-2008	Associate Professor and Director for Translational Research Department of Surgery, U of Michigan, Ann Arbor, MI
2008-present	Professor and Director for Translational Research at the University of Michigan, Ann Arbor, MI
2012-present	Director, Tumor Immunology/Host Defense Program at the University of Michigan, Ann Arbor, MI

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

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