2017北京癌症研究国际研讨会

2017 International Symposium on Cancer Discovery, Beijing, China

会议手册

Meeting Manual

北京大学医学部 逸夫楼209报告厅

209, Yifu Building, Peking University Health Science Center

2017年5月4日-5日，中国．北京

May 4-5, 2017, Beijing, China

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5 月 5 日 日程安排 Agenda: May 5, 2017		
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14:50-15:10	Gregory F. Sonnenberg 教授报告 Speech by Dr. Gregory F. Sonnenberg **Title：Host-Microbiota Interactions during Homeostasis, Inflammation and Cancer**	Assistant Professor of Microbiology & Immunology in Medicine Weill Cornell Medical College
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16:10-16:30	魏文毅教授报告 Speech by Dr. Wenyi Wei **Title：Targeting Cell Signaling Pathways for Cancer Therapies**	Associate Professor Director, Biochemistry Program Cancer Research Institute Beth Israel Deaconess Medical Center Harvard Medical School
16:30-17:00	Nicholas B. La Thangue 教授报告 Speech by Dr. Nicholas B. La Thangue **Title：Epigenetics and Cancer Drug Discovery**	Professor of Cancer Biology Department of Oncology University of Oxford
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嘉宾 Speakers

PTEN: 20 Years of Discovery

Ramon Parsons, M.D., Ph.D.

Department of Oncological Sciences
Icahn School of Medicine at Mount Sinai , USA

The Parsons laboratory seeks to investigate tumor suppressor and oncogene signaling pathways that are altered in human solid tumors using a multidisciplinary approach that includes genetics, biochemistry, bioinformatics, and pathology. Thematic topics that are currently under investigation include dissection of the regulation and function of the PTEN tumor suppressor, determination of the impact of altered PTEN signals in cancer progression and altered energy metabolism, molecular pathogenesis of breast carcinoma initiation and progression, and examination of epigenetic and chromatin reprogramming as consequence of altered cancer signal pathways. Past accomplishments include discovery of PTEN, elucidation of the frequent alteration of the PI3K/PTEN pathway in a wide variety of cancers, generation of mouse models of cancer due to PTEN mutation, and identification of PREX2 as a regulator of PTEN function.

Brief Bio Sketch and General Research Area

Dr. Parsons received his MD and PhD degrees from the State University of New York at Stony Brook in 1992. Prior to his tenure at Columbia, was a Post-Doctoral Fellow at the Johns Hopkins University School of Medicine. He began his career at Columbia as Assistant Professor of Pathology in 1995, when he also became a member of the HICCC. He held several academic positions at the University and has served as the Leader of the Breast Cancer Program since 2005. Formerly a Professor of Breast Cancer Research, Medicine, Pathology, and Cell Biology in the Institute for Cancer Genetics and in The Herbert Irving Comprehensive Cancer Center (HICCC) at New York-Presbyterian Hospital, he also led the HICCC's Breast Cancer Program. Dr. Parsons has been professor and Chair of the Department of Oncological Sciences at the Icahn School of Medicine at Mount Sinai since 2013.

Representative Publications:

1.Hopkins BD, Fine B, Steinbach N, …, Parsons R. A Secreted PTEN Phosphatase that Enters Cells to Alter Signaling and Survival. Science 2013 Jun;.
2.Hodakoski C, Hopkins BD, Barrows D, …, Parsons R. Regulation of PTEN inhibition by the pleckstrin homology domain of P-REX2 during insulin signaling and glucose homeostasis. PNAS, 2014 Jan; 111(1).
3.Saal LH, Gruvberger-Saal SK, Persson C, …, Parsons R, Borg A. Recurrent gross mutations of the PTEN tumor suppressor gene in breast cancers with deficient DSB repair. Nature genetics 2008 Jan; 40(1).

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The Non-Coding RNA Revolution in Biomedical Research

Pier Paolo Pandolfi, M.D., Ph.D.

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Harvard Medical School , USA

The research carried out in Dr. Pandolfi's laboratory has been seminal at elucidating the molecular mechanisms and the genetics underlying thepathogenesis of leukemias, lymphomas and solid tumors as well as in modelling these cancers in the mouse. Dr. Pandolfi and colleagues have characterized the function of the fusion oncoproteins and the genes involved in the chromosomal translocations of acute promyelocytic leukemia (APL), as well as of major tumor suppressors such as PTEN and p53 and novel proto-oncogenes such as POKEMON. The elucidation of the molecular basis underlying APL pathogenesis has led to the development of novel and effective therapeutic strategies. As a result of these efforts, APL is now considered a curable disease. Novel therapeutic concepts have emerged from this work that are currently been tested in clinical trials.

Brief Bio Sketch and General Research Area

Pier Paolo Pandolfi received his M.D. in 1989 and his Ph.D. in 1995 from the University of Perugia, Italy, after having studied Philosophy at the University of Rome, Italy. He received post-graduate training at the National Institute for Medical Research and the University of London in the UK. He became an Assistant Member of the Molecular Biology Program and the Department of Human Genetics at Memorial-Sloan-Kettering Cancer Center in 1994. Dr. Pandolfi grew through the ranks to become a Member in the Cancer Biology and Genetics Program at the Sloan Kettering Institute; Professor of Molecular Biology and Human Genetics at the Weill Graduate School of Medical Sciences at Cornell University; Professor, Molecular Biology in Pathology and Laboratory Medicine, Weill Medical College at Cornell University; and Head of the Molecular and Developmental Biology Laboratories at MSKCC. Dr. Pandolfi was also the incumbent of the Albert C. Foster Endowed Chair for Cancer Research at Memorial Sloan-Kettering Cancer Center.
Dr. Pandolfi presently holds the Reisman Endowed Chair of Medicine, and is Professor of Medicine and Pathology at Harvard Medical School (HMS). He joined the HMS faculty at Beth Israel Deaconess Medical Center (BIDMC) in 2007 to serve as Scientific Director of the Cancer Center, the Director of the Cancer Genetics Program, and the Chief of the Division of Genetics in the Department of Medicine. He was recently appointed to serve as the Cancer Center Director and the Director of the Cancer Research Institute at BIDMC and HMS.

Representative Publications:

1.Matsumoto A, Pasut A, Matsumoto M, …, Pandolfi PP. mTORC1 and muscle regeneration are regulated by the LINC00961-encoded SPAR polypeptide. Nature 2016; 541:228-232.
2.Guarnerio J, Bezzi M, Jeong JC, …, Pandolfi PP. Oncogenic Role of Fusion-circRNAs Derived from Cancer-Associated Chromosomal Translocations. Cell 2016; 165:289-302.

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Autophagy and Secretion in Cancer

Jayanta Debnath, M.D.

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Traditionally viewed as an auto-digestive pathway, emerging evidence implicates autophagy as an important regulator of cellular secretion. ATGs are genetically required for the elaboration of various leaderless proteins. Our lab recently discovered new roles for ATGs in promoting the coordinate secretion of cytokines during tumor cell invasion, as well as in the efficient biogenesis and egress of exosomes. Because autophagy promotes pro-inflammatory cytokine and collagen secretion, we hypothesized that autophagy may enable pro-tumorigenic functions in cancer-associated fibroblasts (CAFs). Fibroblasts play prominent roles in cancer initiation and progression; they secrete factors that modulate pro-tumorigenic immune cell recruitment and angiogenesis, and deposit type I collagen and other extracellular matrix (ECM) components. This constellation of findings, pathologically termed desmoplasia, heralds poor prognosis and reduced patient survival. Using genetic mouse mammary cancer models and syngeneic transplantation assays, we find that autophagy ablation in stromal fibroblasts is necessary for tumor growth in vivo. Furthermore, the genetic ablation of stromal fibroblast autophagy significantly impedes fundamental elements of the tumor desmoplastic response required to support tumor growth. To more rigorously dissect the mechanistic roles of autophagy in secretion, we have developed a novel proximity-specific biotinylation strategy to label proteins that initially engage the autophagy machinery inside a cell, and thereafter, are trafficked and secreted outside of cells. Using quantitative proteomics, we have identified approximately 150 novel putative targets of autophagy-dependent secretion; these candidates lack N-terminal signal sequences and possess known functions in cytoskeletal dynamics, adhesion, metabolism, chromatin remodeling, RNA processing and immunity.

Brief Bio Sketch and General Research Area

Jayanta Debnath, M.D., Professor and Vice Chair for Research in the Department of Pathology at the UCSF, is internationally recognized for his expertise in autophagy and cancer. Dr. Debnath's laboratory focuses on how autophagy influences cancer progression, metastasis, and late recurrent disease in vivo, and how the core autophagy machinery can be targeted for therapeutic benefit. Dr. Debnath currently serves as an Associate Editor of Autophagy, Chair-Elect of the Programmatic Review Panel for the DOD Breast Cancer Research Program and Chair of the Tumor Cell Biology Study Section for NIH. His major honors include an AACR/Genentech Bio-Oncology Career Award, American Society of Cell Biology Keith Porter Fellow Award, and elected membership to the American Society of Clinical Investigation, etc.

Representative Publications:

1.R Lock, Kenific CM, Leidal AM, Salas E, Debnath J (2014). Cancer Discovery. 4 (4): 466-79.
2.L Murrow, Malhotra, R, Debnath J (2015). Nature Cell Biol. 17(3): 300-10.
3.Starobinets H, Ye J, Broz M, Barry K, Goldsmith J, Marsh T, Rostker F, Krummel M, and Debnath J. (2016) J Clin Invest. 126(12): 4417-4429.

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The Mitotic Regulator NIp in Genomic Instability and Tumorigenesis

Qimin Zhan, M.D., Ph.D.

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Disruption of mitotic events contributes greatly to genomic instability and results in mutator phenotypes. Indeed, abnormalities of mitotic components are closely associated with malignant transformation and tumorigenesis. Ninein-like protein (Nlp), a recently identified BRCA1-associated centrosomal protein involved in microtubule nucleation and spindle formation, is an oncogenic protein. Nlp was found to be overexpressed in approximately 80% of human breast and lung carcinomas analyzed. In human lung cancers, this deregulated expression was associated with NLP gene amplification. Further analysis revealed that Nlp exhibited strong oncogenic properties; for example, it conferred to NIH3T3 rodent fibroblasts the capacity for anchorage-independent growth in vitro and tumor formation in nude mice. Consistent with these data, transgenic mice overexpressing Nlp displayed spontaneous tumorigenesis in the breast, ovary, and testicle within 60 weeks. In addition, Nlp overexpression induced more rapid onset of radiation-induced lymphoma. Furthermore, mouse embryonic fibroblasts (MEFs) derived from Nlp transgenic mice showed centrosome amplification, suggesting that Nlp overexpression mimics BRCA1 loss. These findings demonstrate that Nlp abnormalities may contribute to genomic instability and tumorigenesis and suggest that Nlp might serve as a potential biomarker for clinical diagnosis and therapeutic target.

Brief Bio Sketch and General Research Area

Dr. Zhan got his M.D. in Suzhou Medical College (now Medical College of Soochow University) and PhD in Peking Union Medical College. After that he did his postdoctoral training in San Francisco Medical School of UCLA, Southwestern Medical Center of the University of Texas, and the National Cancer Institute (NCI) in USA. He worked as PI at NCI and University of Pittsburgh before he was nominated director of the State Key Laboratory of Molecular Oncology located at the Cancer Institute & Hospital, CAMS in 2004. Since May 2005 Dr. Zhan has been the vice-president of the Chinese Academy of Medical Sciences and the Peking Union Medical College. He was elected academician of Chinese Academy of Engineering in 2011. In 2016, Dr. Zhan was nominated president of Peking University Health Science Center.

Representative Publications:

1.Song Y, Li L, Ou Y, …, Zhan Q. Identification of genomic alterations in oesophageal squamous cell cancer. Nature. 2014 May 1;509(7498):91-5.
2.Shao S, Liu R, Wang Y, …, Zhan Q. Centrosomal Nlp is an oncogenic protein that is gene-amplified in human tumors and causes spontaneous tumorigenesis in transgenic mice. J Clin Invest. 2010 Feb;120(2):498-507.

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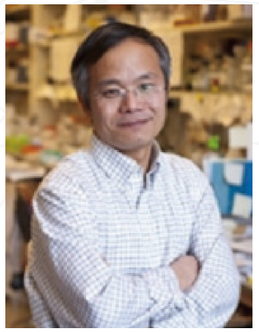
Isomerase Pin1: a Master Regulator of Cancer and a Unique Drug Target

Kun Ping Lu, M.D., Ph.D.

Cancer Research Institute, Beth Israel Deaconess Medical Center
Harvard Medical School, USA



Dr. Kun Ping Lu's lab has discovered that Pin1-catalyzed cis-trans conformational regulation after phosphorylation is a unique signaling mechanism that has the pivotal but opposite effects on the development of cancer and Alzheimer's disease, two major disease that were rarely studied together before. Importantly, this new disease mechanism may lead to novel diagnostic and therapeutic procedures. Notably, his lab has recently identified Pin1 inhibitors for stopping numerous cancer-driving pathways in cancer and cancer stem cells, and developed antibodies specifically against the cis P-tau conformation for early diagnosis and treatment of Alzheimer's disease and traumatic brain injury.

Brief Bio Sketch and General Research Area
Kun Ping Lu, M.D, Ph.D. received his medical training in China, passed USMLEs in the US, and obtained PhD degree from Duke University, followed by postdoctoral training at Salk Institute, where he cloned Pin1, before becoming as an independent investigator at Harvard. Currently, Dr. Lu is Professor of Medicine at Harvard Medical School and Chief and Director, Division of Translational Therapeutics, Cancer Research Institute at Beth Israel Deaconess Medical Center.

Representative Publications:
1.Zhou XZ, Lu KP. The isomerase PIN1 controls numerous cancer-driving pathways and is a unique drug target. Nat Rev Cancer 2016; 16:463-78.
2.Kondo, A., Shahpasand, K., Mannix, R., …, and Lu, K. P. 2015, Antibody against early driver of neurodegeneration cis P-tau blocks brain injury and tauopathy. Nature 523: 431-436
3.Wei, S., Kozono, S., Kats, L., …, and Lu, K. P. 2014, Active Pin1 as a target of ATRA in acute promyelocytic leukemia and breast cancer. Nature Med. 21: 457-466.

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PD-1 Cancer Immunotherapy

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Harvard Medical School, USA

Dr. Freeman's lab studies the role of costimulatory signals in the development of an immune response. T cell activation requires two signals. Specificity is provided by TCR recognition of peptide-MHC complexes but a second, costimulatory signal is required for full T cell activation. The B7 gene family, comprised of B7-1 and B7-2, provide the critical costimulatory signal for full T cell activation, clonal expansion, and development of effector function through their interaction with CD28 on T cells. After T cell activation, the interaction of B7-1 and B7-2 with the higher affinity ligand, CTLA4, expressed on activated T cells, leads to down-regulation of T cell activation. Stimulation of the TCR alone leads to T cell clonal anergy thus blockade of B7-1 and B7-2 can be used to establish antigen-specific tolerance for transplantation or the alleviation of autoimmunity. Conversely, expression of B7-1 and B7-2 can stimulate an immune response and the introduction of B7-1 or B7-2 into tumors can stimulate an anti-tumor response leading to tumor rejection and anti-tumor immunity. Recently, we have cloned two novel members of the B7 gene family. These new B7s bind to receptors expressed on activated T cells and further regulate the development of an immune response. We are currently focusing on the function of these novel B7 genes and their interactions with the B7/CD28-CTLA4 pathway.

Brief Bio Sketch and General Research Area
Dr. Freeman earned his BA in Biochemistry and Molecular Biology, and PhD in Microbiology and Molecular Genetics from Harvard University. His research has identified the major pathways that control the immune response by inhibiting T cell activation (PD-1/PD-L1 and B7-2/CTLA-4) or stimulating T cell activation (B7-2/CD28).
In 2000, Dr. Freeman discovered PD-L1 and PD-L2, and showed they were ligands for PD-1, thus defining the PD-1 pathway and the drug target: block the interaction. He showed the function of PD-1 was to inhibit immune responses and that blockade enhanced immune responses. He showed that PD-L1 is highly expressed on many solid tumors as well as some hematologic malignancies and allows these tumors to inhibit immune attack. He received the 2014 William B. Coley Award for Distinguished Research in Tumor Immunology for this work that led to development of PD-1 pathway blockade for cancer immunotherapy.

Representative Publications:
1.Im SJ, Hashimoto M, Gerner MY, Freeman GJ, etc. Defining CD8(+) T cells that provide the proliferative burst after PD-1 therapy. Nature 2016; 537:417-421.
2.Saha A, O'Connor RS, Thangavelu G, Freeman GJ, etc. Programmed death ligand-1 expression on donor T cells drives graft-versus-host disease lethality. J Clin Invest 2016; 126:2642-60.

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CAR T cell therapy: the CD19 Paradigm and Beyond

Isabelle Rivière, Ph.D.

Cell Therapy and Cell Engineering Facility
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Dr. Isabelle Rivierei is currently the Director of the Michael G. Harris Cell Therapy and Cell Engineering Facility where she investigates the genetic modification of hematopoietic cells to increase or retarget the immune response against tumors. Her laboratory has developed cell manufacturing platforms under cGMP conditions for several Phase I/II clinical trials and currently supports 8 CAR-T cell based clinical trials under 5 INDs at MSK. She actively participated in the National Cell Manufacturing Consortium Workshop that has led to the establishment of the Technology Roadmap to 2025 for Achieving Large Scale, Cost effective, Reproducible Manufacturing of High-Quality Cells.

Brief Bio Sketch and General Research Area

Dr. Isabelle Rivierei received her PhD in Cellular and Molecular Biology from the University of Paris. She initiated her graduate studies at the Institut Curie in Paris and completed her thesis in the laboratory of Dr.Mulligan at the Whitehead Institute in Cambridge. During this time, she developed novel retroviral vectors for in vivo long-term expression of transgenes in hematopoietic cells using MFG/SFG-based retroviral vectors that are widely used in clinical studies for the treatment of genetic and acquired disorders. She subsequently worked as a post-doctoral fellow in the laboratory of Dr. D. Littman at New York University. Her studies focused on the regulation of cytokines produce by T helper lymphocytes in vivo. Dr. Rivière joined the faculty of the Memorial Sloan-Kettering Cancer Center in 1999.

Representative Publications:

1.Davila ML, Riviere I, Wang X, …, Brentjens R. Efficacy and Toxicity Management of 19-28z CAR T Cell Therapy in B Cell Acute Lymphoblastic Leukemia. Sci Transl Med. 2014 Feb 19;6(224):224ra25.
2.Boulad F, Wang X, Qu J, …, Rivière I. Safe mobilization of CD34+ cells in adults with beta-thalassemia and validation of effective globin gene transfer for clinical investigation. Blood. 2014 Mar 6;123(10):1483-6.
3.Rivière I, Dunbar CE, Sadelain M. Hematopoietic stem cell engineering at a crossroads. Blood. 2012 Feb 2;119(5):1107-16.

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PTEN Family in Cancer and Beyond

Yuxin Yin, M.D., Ph.D.

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PTEN is essential for the maintenance of chromosomal stability and inhibition of cancer. We found that PTEN controls DNA replication progression through MCM2. PTEN also stabilizes replication forks through RPA1. Therefore, PTEN is a regulator of DNA replication and protector of replication forks. In our recent studies, we have revealed a mechanism of alternative protein translation, through which we identified two members of the PTEN family with novel functions. A CUG codon upstream of and in-frame with the coding region of canonical PTEN initiates translation of an N-terminally extended form of PTEN, PTEN. PTEN α induces cytochrome c oxidase activity and ATP production in mitochondria. Deletion of PTEN α impairs mitochondrial respiratory chain function. Recently, we have identified another N-terminal extended PTEN isoform, PTENβ. PTENβ localizes predominantly in the nucleolus and regulates rDNA transcription. Our data provide insights into the mechanism by which the PTEN family is involved in multiple cellular processes.

Brief Bio Sketch and General Research Area

Yuxin Yin received his Ph.D. in Genetics and Molecular Biology from the University of North Carolina at Chapel Hill in 1997 and got postdoctoral training in Princeton University. In 1999, he was appointed as a tenure-track assistant professor in Columbia University and was promoted to the associate professor in 2007. He was recruited as a National One-Thousand Talent Plan to the Peking University Health Science Center in 2010. His research field is cancer research with focus on the roles of tumor suppressors p53 and PTEN in cell cycle regulation, apoptosis and genomic stability. He was the first one to find a fundamental role for p53 in maintaining genomic stability. Also, it is first reported that PTEN plays a fundamental role in the maintenance of chromosome stability. In Peking University, Dr. Yin has made great progress in studying the PTEN pathway in genome stability and tumor suppression. Recently, his group revealed a novel mechanism of alternative translation of protein and discovered two new members of the PTEN protein family named PTENα and PTENβ. These studies may provide new insights into the mechanism by which PTEN maintains genomic stability and suppresses tumorigenesis.

Representative Publications

1) Shen, W. H., Balajee, A. B., Wang, J., Wu, H., Eng, C., Pandolfi, P. P., and Yin, Y.* (2007). Essential role for nuclear PTEN in maintaining chromosomal integrity. Cell,128: 157-170. Accompanying Minireview, Cell, 128: 25-28.
2) Liang, H., He, S., Yang, J., Jia, X., Wang, P., Chen, X., Zhang, Z., Zou, X., McNutt, M.A., Shen, W.H.,* and Yin, Y.*. (2014). PTENα is a PTEN isoform Translated through Alternative Initiation and Regulates Mitochondrial Function. Cell Metab. 19, 836-848.
3) Liang, H., Chen, X., Yin, Q., Ruan, D., Zhao, X., Zhang, C., McNutt MA, and Yin, Y. *(2017). PTENβ is an alternatively translated isoform of PTEN that regulates rDNA transcription. Nature Communications. 8:14771.

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Host-microbiota interactions in human health and disease

Gregory F. Sonnenberg, Ph.D.

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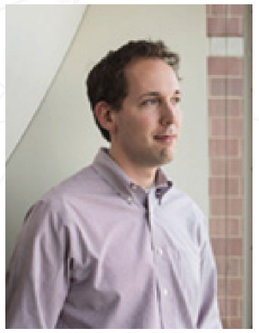

The human immune system is critical to protect against infection with pathogenic microorganisms. However, inappropriate immune responses against our own tissues or non-harmful environmental triggers such as beneficial commensal bacteria that normally colonize the body's barrier surfaces can promote autoimmune or chronic inflammatory diseases. Indeed, emerging studies in patient populations indicate that abnormal host immune responses to commensal bacteria are causally-linked to the pathogenesis and progression of numerous chronic infectious, inflammatory and metabolic diseases, such as HIV, inflammatory bowel disease (IBD) and cancer. The focus and long-term research goals of the Sonnenberg Laboratory are to interrogate functional interactions between the mammalian immune system and intestinal commensal bacteria in the context of health and disease. The laboratory employs cutting-edge immunologic and microbiologic approaches to interrogate these interactions in both basic mouse models and translational patient-based studies. Recent studies in the laboratory have identified a critical role for innate lymphoid cells in orchestrating host relationships to defined subsets of commensal bacteria. Delineating these complex interactions will lead to a better understanding of the pathogenesis of multiple chronic inflammatory diseases, and direct the future development of novel therapeutic strategies targeting commensal bacteria-dependent chronic inflammation.

Brief Bio Sketch and General Research Area

Dr. Gregory F. Sonnenberg received his Ph.D. from the University of Pennsylvania in 2011 and was a recipient of a NIH Directors Award that established the Sonnenberg Laboratory in 2012. In 2014, Dr. Sonnenberg was recruited to Weill Cornell Medicine where he is currently an Assistant Professor of Microbiology & Immunology in Medicine. Dr. Sonnenberg's laboratory interrogates the regulation and functional contributions of host-microbe interactions during health, inflammation and cancer.

Representative Publications:

1.Hepworth M.R., Monticelli L.A., Fung T.C., …, Sonnenberg G.F. (2013) Innate lymphoid cells regulate CD4+ T cell responses to intestinal commensal bacteria. Nature.
2.Hepworth M.R., Fung T.C., Masur S.H., …, Sonnenberg G.F. (2015) Group 3 Innate lymphoid cells mediated intestinal selection of commensal bacteria-specific CD4+ T cells. Science.

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Ferroptosis: A Missing Puzzle Piece in Tumor Suppression?

Wei Gu, Ph.D.

Institute for cancer Genetics, Dept. of Pathology and Cell Biology, HICCC Columbia University Cancer Center, Columbia University, New York, NY, USA

Dr. Gu is currently an Abraham and Mildred Goldstein endowed chair Professor and Vice-Chairman for Cancer Research at the department of Pathology and cell biology and Institute for Cancer genetics in Columbia University. He obtained his B.S. degree in Biological Science from Peking University and he received his Ph.D. in the area of Molecular Biology and Genetics from Columbia University in USA. Later he was a post-doctoral fellow of Dr. Robert Roeder at the Rockefeller University in New York City. Dr. Gu has received many honors; most recently including the Elected Fellow for AAAS, Ellison Medical Foundation Senior Scholar, The Stohlman Scholar, Leukemia and Lymphoma Society Scholar Award, Stewart Trust Award, Irma T. Hirshl Trust Scholar Award, Life Science Research Foundation Postdoctoral Fellowship and Dean's Award for Outstanding Research Achievement from Columbia University.

Dr. Gu is recognized internationally for the pioneering contributions to the regulation of p53-mediated tumor suppressor function. His lab has made several seminal discoveries in the area of protein modifications and tumor suppression. For example, he identified the first non-histone protein (p53) that can be regulated by acetylation and deacetylation; his studies of p53 acetylation laid the foundation for the current view that reversible acetylation is a general mechanism for regulation of non-histone proteins. Likewise, following his discovery of a deubiquitinase HAUSP-mediated stabilization of the p53 and Mdm2 polypeptides, deubiquitination has come to be accepted as a common mechanism of protein stabilization. Since p53 mutations are the most common genetic lesions associated with human cancer, a major objective of molecular oncology is to elucidate the mechanisms by which p53 is regulated. Gu's laboratory has identified the major molecules and pathways that regulate p53 activity. He discovered that p53 activity is controlled in large part by acetylation and deacetylation of the p53 polypeptide. In particular, he established that acetylation of specific p53 residues is required for its transcriptional function and acts as the primary signal to differentially induce its canonical functions in apoptosis, cell growth arrest and senescence. He identified "dynamic ubiquitination" (polyubiquitination, monoubiquitination and deubiquitination) as the major mechanism by which the stability and subcellular localization of p53 protein are determined. His findings on deacetylases in regulating non-histone protein regulation such as HDAC1 and Sirt1 provide the critical implication for the specificities in the usage of HDAC inhibitors in clinical trials. His findings on deubiquitinase activities directed to p53, Mdm2 and others have led to the usage of HAUSP inhibitors in preclinical trials. More recently, his work on metabolic regulation elucidates that p53-mediated tumor suppression can act in the absence of the classic tumor suppression mechanisms including cell-cycle arrest, apoptosis and senescence. Notably, his team has discovered a new mode of tumor suppression based on p53 regulation of cystine metabolism, ROS responses and ferroptosis. Of ~130 his papers, more than 20 of them were published in the top journals such as Science, Nature and Cell. He will present the latest work on ferroptosis, a novel tumor suppression mechanism of p53.

Targeting Cell Signaling Pathways for Cancer Therapies

Wenyi Wei, Ph.D.

Department of Pathology, Beth Israel Deaconess Medical Center
Harvard Medical School, Boston, MA, USA

My laboratory mainly focuses on understanding mechanistically how aberrant cell signaling events lead to altered protein homeostasis and cellular functions to facilitate the development of human disorders including cancer. Our research is uniquely poised to understand how post-translational modifications generate a complex coding system to transduce cellular messages, and crosstalk with other signaling pathways to govern various cellular processes. One of the major focus lies in understanding how aberrant cell cycle regulation leads to cancer development. To this end, proper cell cycle transitions are largely driven by waves of ubiquitin-dependent degradation of key cell cycle regulators by APC or SCF, the two major E3 ligase complexes. I am interested in elucidating the underlying mechanisms that timely regulate APC/Cdh1 and various SCF E3 ligase activities in different cell cycle phases, whether other layers of crosstalk between the APC and SCF complex exist, and identifying novel downstream targets for both APC and SCF complexes, which will help pinpoint their functions in both cell cycle control and tumor formation. I will present new information regarding our dedicated efforts in utilizing multidisciplinary approaches to understand the tumor suppressor role of Cdh1 or Fbw7 and the oncogenic potential of Skp2. These results will help to better understand the multilayer regulation of the delicate proteolysis pathways, which will lead us to the design of more efficient intervention strategies to combat cancer and other diseases.

Brief Bio Sketch and General Research Area

Dr. Wenyi Wei received his B.A. degree from Shandong University in 1993 and then obtained his M.S. training in Chinese Academy of Science from 1993 to 1996. Afterwards, Dr. Wei received his Ph.D. training in the MCB department at Brown University and his postdoctoral training in the laboratory of Dr. William Kaelin, Jr. at DFCI, Harvard Medical School. Dr. Wei became independent from October 2006 in Department Pathology at Beth Israel Deaconess Medical Center, Harvard Medical School. The major focus of the WEI laboratory is aimed at understanding how APC and SCF activities contribute to cell cycle regulation and subsequent tumor formation. To achieve these goals, the lab will use multidisciplinary approaches including biochemical and genetic analysis. In the long term, we hope that a better understanding of the multilayer regulation of the delicate proteolysis pathways will lead us to the design of more efficient intervention strategies to combat cancer, cardiovascular disorders and other human diseases.

Representative Publications:

1. Liu, P., Bagley, M., Inuzuka, H., … Wei, W. (2014) Cell cycle-regulated activation of Akt kinase by phosphorylation of its carboxyl-terminal terminus. Nature 508(7497):541-5
2. Guo, J., Chakraborty, A. K., Liu, P., … Wei, W. (2016) pVHL suppresses kinase activity of Akt in a proline-hydroxylation dependent manner. Science 353(6302):929-32.

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Epigenetics and Cancer Drug Discovery

Nicholas B. La Thangue, Ph.D.

Department of Oncology
University of Oxford, UK

In order to design better therapies that effectively treat cancer, it is essential to decipher the molecular and biological details of pathways that control proliferation in normal cells and thereafter understand how they become aberrant in cancer. The primary objective of our work is to explore the regulation of and control by pRb and p53 activity. Specifically, we have defined new levels of control in regulating pRb tumour suppressor activity, particularly novel post-translational signals. We have elucidated new members of the E2F family, and identified the key pathways through which they act. Functional characterisation of E2F in cell cycle control and apoptosis has identified a remarkable level of complexity that governs the switch to apoptosis. Our p53 research is principally focused on uncovering the diverse modifications that dictate the outcomes of the p53 response to stress.

Brief Bio Sketch and General Research Area

Nicholas La Thangue is Professor of Cancer Biology in the Department of Oncology, and was previously Cathcart Professor of Biochemistry at the University of Glasgow, and before that a scientist at the Medical Research Council. He is a Fellow of the Royal Society of Edinburgh, a Member of the European Molecular Biology Organisation (EMBO), a Fellow of the Academy of Medical Sciences, a Fellow of the European Academy of Cancer Sciences, a Fellow of the Lister Institute and Professorial Fellow at Linacre College Oxford. He has founded several biotech companies, most recently Oxford Cancer Biomarkers.

Representative Publications:

1.Carr SM, Munro S, Zalmas LP, …, La Thangue NB. (2014) Lysine methylation-dependent binding of 53BP1 to the pRb tumor suppressor. Proc Natl Acad Sci U S A. Jul 21. pii: 201403737.
2.Zheng, S., Moehlenbrink, J., Lu, Y.C., …, La Thangue, N.B. (2013) Arginine methylation-dependent reader-writer interplay governs growth control by E2F-1. Molecular Cell, 52 (1) 37-51
3.Cho, E-C., Zheng, S., Munro, S.,…, La Thangue, N.B. (2012) Arginine methylation controls growth regulation by E2F-1. EMBO Journal, 31: 1785-1797.

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北京大学基础医学院

北京大学基础医学院的前身是 1954 年 9 月成立的北京医学院基础医学部。1960 年 2 月改称基础医学系。1985 年 5 月成立北京医科大学后，更名为基础医学院。2000 年 4 月北京大学与北京医科大学合并后，更名为北京大学基础医学院至今。

学院现设 13 个系、2 个研究所及 1 个医学实验教学中心。拥有“生物学”和“基础医学”2 个博士学位授权一级学科（涵盖 12 个二级学科）、另有“中西医结合基础”二级学科，其中包括 7 个国家重点二级学科、1 个北京市重点一级学科、3 个博士后流动站、6 个省（部）级重点实验室，拥有国际先进水平的科研基地和实验技术平台。

学院现有教职工 403 人，其中教授 67 人、副教授 83 人。学院以师资力量雄厚、治学严谨著称，拥有一批国内外著名的专家学者。其中中国科学院院士 4 人；工程院院士 1 人；国家“千人计划”专家 4 人；“长江计划特聘教授”7 人；国家杰出青年基金获得者 7 人；国家“青年千人计划”专家 6 人；获得“国务院政府特殊津贴”13 人；获“国家人事部有突出贡献的中青年专家”称号 4 人；获“卫生部有突出贡献的中青年专家”称号 5 人；教育部跨世纪 / 新世纪优秀人才 19 人；获国家自然科学基金“优秀青年科学基金”资助 8 人；博士生导师 72 人。

在教学方面，学院每年为医学部多个专业的本科生、研究生开设必修、选修课程 100 余门；现有国家级精品课程 9 门，北京市级精品课程 10 门，国家级精品资源共享课 7 门。近年来，有两项教学成果获国家级教学成果二等奖和北京市一等奖，；学院被评为”北京市模范集体；生物医学实验教学中心被评为“国家级高等学校实验教学示范中心”；病理学、免疫学及人体解剖学分别被评为“教育部双语教学示范课程”；2 个教学团队”被评为北京市级优秀教学团队及国家级教学团队；“基础医学”专业被评为北京市特色专业；北京大学生物创新实践基地被评为北京高等学校示范性校内创新实践基地建设单位; 6 人获北京市教学名师奖; 1 人获宝钢教育基金会优秀教师特等奖; 1 人获“北京市师德先进个人”荣誉称号。学院培养的学生以学风严谨，实践综合能力强，开拓创新、潜力大等优点受到国内外教育和科研部门的赞誉。许多毕业生已成为国内外生物医学学科的学术带头人或学术骨干。

在科研方面，学院主要从事生物医学领域的基础及应用基础研究，拥有雄厚的科学研究综合实力。2012-2016 年获得部委和北京市科技成果奖 6 项，获得国家及国际发明专利 37 项，自主研发的 I 类新药“新型特异性肿瘤显像剂”等多项专利实现技术转让或产业化开发；2012-2016 年发表科研论文 1947 篇，以第一作者或通讯作者单位发表的 SCI 论文 1141 篇，其中 IF ≥ PNAS 期刊论文 55 篇。2012 年 -2016 年获批各类科技项目 476 项，共获科研经费约 42749 万元。

学院还十分重视国际间的交流与合作，注重借鉴世界先进的医学研究成果和教学经验，不断提高自身整体办学水平。已与几十所世界一流医学院校和科研机构开展了项目合作研究、人员交流、合作举办国际会议等多种形式的合作交流，建立了广泛的深层次联系。

历经 60 余载的发展历程，现在的北京大学基础医学院已成为国内著名的、以发展多层次基础医学教育、研究人类生命科学和防治疾病的基础理论为主要目标的教学科研中心之一，是国家基础医学领域高级专门人才的培训基地之一。

北京大学系统生物医学研究所

北京大学系统生物医学研究所 (Peking University Systems Biomedicine, PKUISB) 是 2010 年 6 月成立的生物医学研究机构，涵盖生物学、基础医学、药学、临床医学、生物信息学等学科，是集研究、教育、开发及服务于一体的新型科研机构。研究所主要针对肿瘤、心脑血管和神经精神疾病等威胁人类健康的重大疾病，用系统生物学的研究方法和手段将复杂疾病研究与临床实际密切结合，增进人们对肿瘤、心脑血管疾病等重大疾病的病理和机制的系统理解，寻找新途径预测个体对疾病以及药物的敏感性；探索对这些复杂疾病更为有效的诊断和治疗方法；为每个人提供维持健康和预防疾病的个性化方案。研究所的发展目标是：大力推动基础研究与临床医学相结合，推动新型诊断、治疗技术的研究和药物创新，通过引进国内外一流科研人才，多学科交叉融合、相互促进，将北京大学系统生物医学研究所建成具有国际一流水平的国家级研究基地，在培养多学科人才、促进学科发展及推动我国生物、医药、中医药事业等方面发挥领军作用。

北京大学系统生物医学研究所（系统所）设置 8 个研究室，目前已建立基因组学、分子遗传、计算生物学、结构生物学研究室，蛋白质组学和代谢组学研究室，拟建药物研发、转化医学研究室。系统所是北京市唯一一家致力于肿瘤系统生物医学研究的科研机构，在相关领域具备国际领先水平。2012 年 5 月，系统所牵头建设的“肿瘤系统生物学北京市重点实验室”成功获得北京市科委认定，这是基础医学院第一个成功获批的北京市重点实验室。

经过近七年的建设，系统所已经建立了高通量基因测序平台、转基因小鼠平台、蛋白质组学 / 代谢组学质谱分析平台、流式细胞分析平台和活细胞工作站等综合服务大平台。这些平台不仅可以满足系统所目前的科研需求，还可为北京大学及其他科研院所提供服务。

School of Basic Medical Sciences of Peking University

The School of Basic Medical Sciences was formerly called the Institute of Basic Medical Sciences of Beijing Medical College, and was established on September 14th, 1954. In 1960, it was renamed the Department of Basic Medical Sciences. After Beijing Medical University was established in 1985, it came to formally be called the School of Basic Medical Sciences. Following the integration of Peking University and Beijing Medical University in 2000, the school has been called the School of Basic Medical Sciences of Peking University.

The School of Basic Medical Sciences consists of 13 departments, 2 institutes and an educational center for biomedical research. There are 2 certified doctoral degree first-level disciplines and 12 second–level disciplines. There is one second-level discipline in “basic combined Chinese and Western medicine”; 7 national key disciplines; 1 Beijing key discipline; 4 provincial key laboratories and several international advanced research bases and experimental platforms.

The School is famous for its abundant teaching resources and meticulous scholarship, and its staff consists of well-known scholars and experts of both domestic and overseas origin. We have many outstanding and talented individuals in our school. There are 4 academicians of the Chinese Academy of Science, and 1 academician of the Chinese Academy of Engineering. 7 experts are Cheung Kong Scholar Distinguished Professors of The Cheung Kong Scholars Program. 4 experts are awarded the “1000 Talents Plan”. 7 faculty members have been awarded the National Fund for Distinguished Young Scientists; 6 have been awarded the “1000 Young Talents Plan”13 experts have received a special government allowance. The title “Young and Mid-aged Expert who have made Outstanding Contributions” has been conferred by the Ministry of Personnel on 4 faculty members; 5 experts have received the title “Young and Mid-aged Expert who have made Outstanding Contributions” from the Ministry of Health; 19 have been named as “Trans-century/New-century Outstanding Talents” by the Ministry of Education; 8 have received support from the “Excellent Young Scientist Foundation” of the National Natural Science Foundation of China (NSFC).

The school has 1126 undergraduate students who are majoring in either clinical medicine or basic biomedical research, and the school also oversees basic study for students in four majors including clinical medicine, and dental medicine. Since the year 2001, the length of the basic medical curriculum has been changed, and students of our eight-year system are now required to obtain undergraduate training and a PhD. The curriculum aims at training students who are well developed intellectually, morally, and physically in preparation to engage in high-level scientific endeavors. Students from our school have been accorded high praise by national and international education and research departments for being diligent and rigorous, as well as for their academic strengths and overall capacity, and for possessing great potential. Many students who have graduated have already become academic leaders in the field of biomedicine.Our school provides basic medical courses for students majoring in basic medicine, clinical medicine, dental medicine, preventative medicine, pharmacology, nursing, biomedical English, medical laboratory science and laboratory medicine.

The school now has 9 national level courses of excellence and 10 municipal level courses of excellence. There have been several outstanding teaching achievements at our school, two of which were awarded a first class teaching prize at the Beijing municipal level in the years 2009 and 2012; a second class teaching prize was also awarded at the national level in the year 2005. Our educational center for biomedical experiment work was given the honor of being designated as “The Experimental Teaching Demonstration Center of Beijing” and “The National Experimental Teaching Demonstration Center” in the years 2006 and 2008 respectively. In 2007, 2008 and 2010, Pathology, Immunology and Human Anatomy were cited as “Bilingual Teaching Demonstration Courses” by the Ministry of Education. In the years 2007 and 2010, the school’s “Team for training and teaching innovation talent in physiology” was honored as the Beijing and national outstanding teaching team. In 2008, the school’s basic medicine major was cited as Beijing’s special major, and 4 teachers were named Beijing Distinguished Teacher.One was awarded the grand prize for outstanding teacher by the Baosteel Education Foundation in 2011.
As regards scientific research, our school focuses primarily on basic and applied research in the field of biomedicine, and has strong overall strength in scientific research. From 2000 to 2012, the school received a variety of scientific and technical awards, which numbered 132 in total. Among these, there were 2 National Prizes for Natural Sciences, and 31 Science and Technology Progress Awards from national government ministries and the Beijing city government. In addition, there were 59 national and international patents awarded for inventions, and 40 other scientific and technical awards. From 2005 to 2012, there were a total of 883 scientific projects; 40 National Basic Research Programs of China were carried out; there were 15 “863 Projects" funded by the National High Technology Research and Development Program of China; 16 “National Science and Technology Major Program” projects received funds; 278 programs received Natural Science Funds of China; 125 programs received Science Funds from the Ministry of Education; and 65 projects received Beijing municipal science program funding.

Recently, the school has begun to augment research and development of biological technology and medical products, and engage in more extensive collaboration with large scale medical corporations. We have made significant progress in these areas of scientific development. A group of newly developed programs here has achieved outstanding results in animal experiments and clinical trials in areas of genetic engineering, pharmacology, tumor immunotherapy, therapy for autoimmune disease, gene therapy for cardiovascular disease, treatment of neurodegenerative diseases and corneal diseases with stem cell therapy, and development of diagnostic reagents.

Our school also places great emphasis on international collaboration and communication, and we are striving to learn from worldwide advances in medical research and education in order to raise the overall level of our medical education here. We co-operate with many top-flight medical colleges and institutes, and hold international conferences. With these kinds of activities, we have established ever better relationships with outside universities and other organizations.

After 60 years of development, the School of Basic Medical Sciences of Peking University has become a well-known educational research center whose main propose is to continue to develop multi-level basic medical education and study in life sciences, disease prevention and treatment. Our school now also serves as a training base for senior specialists in some fields of basic medicine.

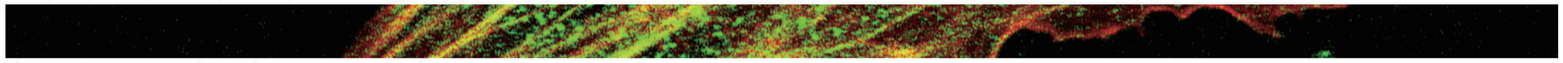
Peking University Institute of Systems Biomedicine

Peking University Institute of Systems Biomedicine (PKUISB) is an interdisciplinary research center which was established in 2010. The aim of this research center is development and application of advanced techniques in biology, basic medical sciences, pharmaceutical sciences, bioinformatics and clinical research to address fundamental and compelling problems in cancer, cardiovascular and neuronal diseases. PKUISB is equipped with state-of-the-art facilities, and focuses on three areas of research:

1) Identification of new molecular mechanisms underlying complex systemic disease;
2) Development of new clinical targets and novel therapeutic strategies;
3) Provision of health and care solutions specifically tailored for individuals.

As one of the leading systems biomedicine centers in the nation, our mission is fueled by collaboration in basic and clinical sciences. Our field of study thus spans a spectrum of research from the laboratory to the bedside. PKUISB faculty work to identify disease causation at the genetic and environmental levels, and translate these findings into pioneering and innovative clinical trials. Our institute currently conducts research in genomics, proteomics, metabolomics, molecular genetics, computational biology and structure biology. The institute has brought together some of the most exceptional scientists in the country to investigate and improve the prevention and early detection and treatment of cancer. We also provide outstanding services which serve to promote biomedical research at the systems level.

会议记录 Meeting Record



校园地图 Map

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