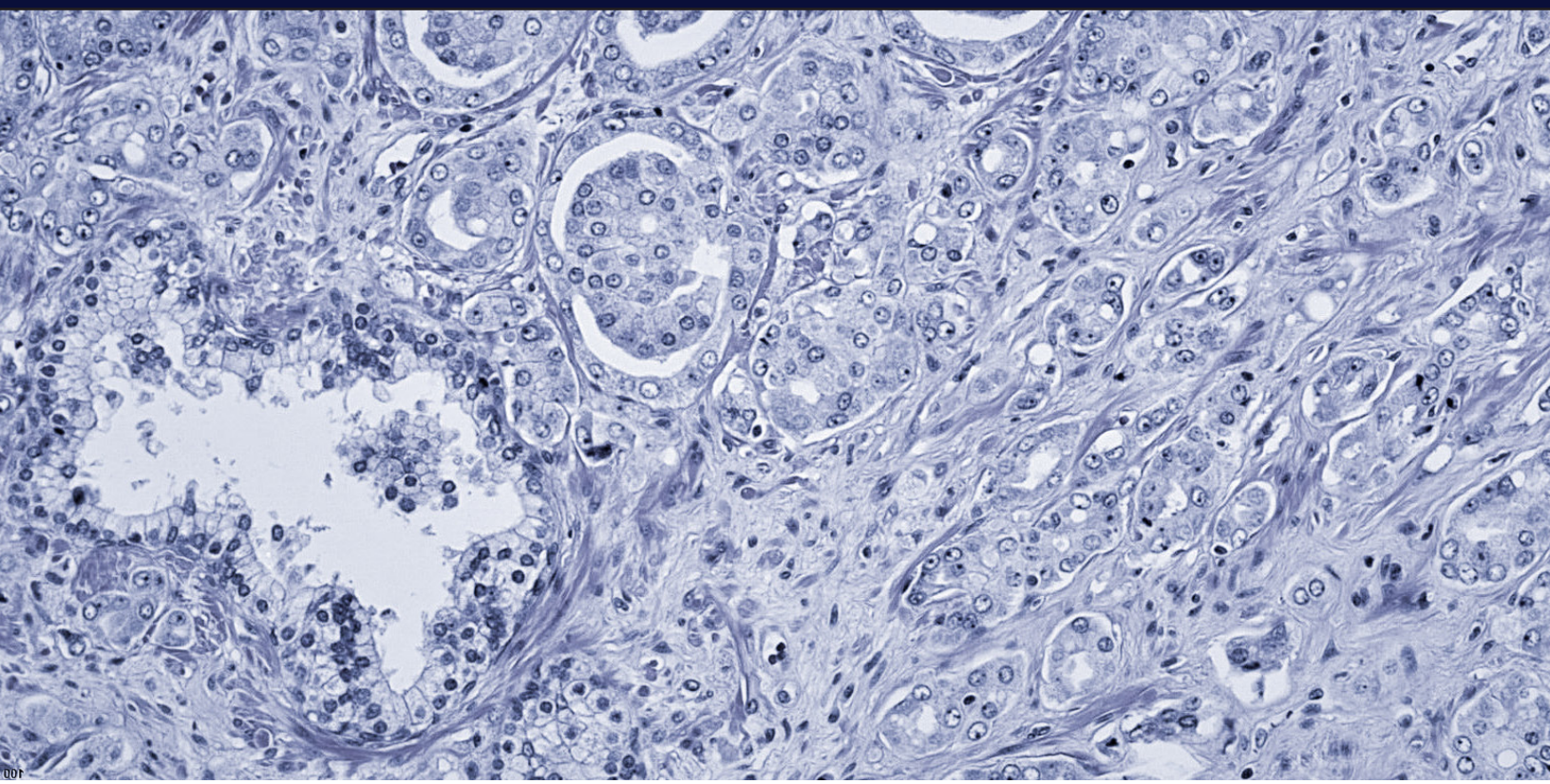
Henry Fok Ying Tung SYSU-UBC Medical Research Symposium — Cancer



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FUNDAÇÃO HENRY FOK

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Henry Fok Ying Tung SYSU-UBC Medical Research Symposium—Cancer

The Henry Fok Ying Tung SYSU-UBC Medical Research Symposium — Cancer is second in a series of three research symposia held collaboratively between Sun Yat-sen University in Guangzhou, China, and the University of British Columbia in Vancouver, Canada. The series aims to advance medical research by bringing together leading researchers in three areas: neuroscience, cancer, and infectious diseases and immunology. The symposium series is funded by the generous donation of UBC alumnus Ian Fok, who has also committed funds to promote collaborative research and student exchanges between the two institutions.

Symposium Details	
Date	May 7 & 8, 2019
Venue	Blusson Spinal Cord Centre - Lecture Hall & Atrium – 818 W. 10th Ave
Host	Faculty of Medicine, University of British Columbia
Chair	Dr. Rob McMaster, Executive Associate Dean, Research, Faculty of Medicine, UBC
Co-Chair	Prof. Erwei Song, Dean, Zhongshan School of Medicine, SYSU

About Henry Fok Ying Tung

Dr. Henry Fok Ying Tung was a prominent Hong Kong business tycoon and a noted philanthropist. He began his career in the civil and marine engineering contracting industry, and expanded into real estate investment, eventually founding the Fok Ying Tung Group, the Fok Ying Tung Foundation Ltd. and the Hong Kong Pei Hua Education Foundation. Long seen as one of Hong Kong's best-connected figures in Beijing, Fok was a trusted confidant of many mainland leaders and is often credited with invigorating China's economic development.

Fok was active in charitable work through the Fok Ying Tung Foundation, which he founded in 1984. He has named his philanthropic priorities as advancing education, reducing poverty, and furthering medical research in China and worldwide.

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Opening Program

Time	Agenda
8:00 – 8:10	Opening Speech by Dr. Dermot Kelleher, Dean, Faculty of Medicine, UBC
8:10 – 8:15	Remarks by Dr. Ian Fok, Henry Fok Foundation
8:15 – 8:20	Remarks by Prof. Erwei SONG, Dean, Zhongshan School of Medicine, SYSU
8:20 – 8:25	Remarks by Dr. Rob McMaster, Executive Associate Dean, Research, UBC
8:25 – 8:30	Group photo of speakers

DR. DERMOT KELLEHER

Dean, Faculty of Medicine & Vice-President, Health, University of British Columbia

Dermot Kelleher joined UBC in 2015 as Dean of the Faculty of Medicine. He is recognized internationally for innovation in academic health leadership and administration, clinical care, research and education. His research examines the immune response to many of the leading causes of gastrointestinal infectious disease worldwide. With a strong commitment to innovation and collaboration, Dr. Kelleher has worked to found several companies supporting both translational developments in biomedical science and fostering collaboration in biomedical research.

DR. IAN FOK

Chairman & CEO, Fok Ying Tung Group

Ian Fok is the son of Henry Fok Ying Tung. He is the Chairman and CEO of the Fok Ying Tung Group, and Director of the Fok Ying Tung Foundation and the Hong Kong Pei Hua Education Foundation. Named among the top 10 philanthropists of Guangzhou, he has received many prestigious awards and serves on numerous community boards. He credits his father with instilling in him strong philanthropic values, and has given his time, expertise and resources towards advancing education, reducing poverty, and furthering medical research in China and around the world. He is a proud UBC alumnus, with a BSc in Mathematics (1971) and an MBA (1973).

PROF. ERWEI SONG

Dean, Zhongshan School of Medicine, Sun Yat-sen University

Erwei Song is a Professor of Breast Surgery at Sun Yat-sen University, Distinguished Professor of China Medical Board (CMB) and Fellow of the Royal Society of Biology (UK). Currently, he serves as Dean of the Zhongshan School of Medicine and President of Sun Yat-sen Memorial Hospital at Sun Yat-sen University. He leads four ongoing national research projects, and serves as associate editor for BMC Cancer, Cancer Science and Science China-Life Sciences, and reviewing editor for the Journal of Biological Chemistry.

DR. ROBERT MCMASTER

Executive Associate Dean of Research, Faculty of Medicine, University of British Columbia

As the Executive Associate Dean, Research at the UBC Faculty of Medicine, Dr. McMaster leads the development of health and life sciences research strategies and ensures that research is translational and of the highest caliber. He provides guidance to health research program leaders and ensures resources are supported effectively across the Faculty on all campuses and health authorities. He has also played a key role in formulating the research component of the Faculty's strategic plan, and in ensuring that the Faculty's activities align with the plan's priorities.

Schedule

DAY 1 - MAY 7		
8:00 - 8:30	**Symposium Opening**	
Session 1 - Immuno-oncology & Tumour Microenvironment		
Time	Speaker	Topic
8:30 – 8:35	Dr. Janel Kopp	*Introduction to Immuno-oncology & tumour microenvironment*
8:35 – 8:55	Dr. Cal Roskelly	Podocalyxin is a driver of pre-metastatic invasive budding in solid tumours
8:55 – 9:15	Dr. Kelly McNagny	Podxl as a target for immunotherapy in metastatic cancers
9:30 – 9:50	Dr. François Bénard	Optimizing radiopharmaceuticals for cancer imaging and radioligand therapy
10:00 – 10:20	Prof. Erwei Song	Treat the cancer soil: Turn foes to friends
10:30 – 10:50	**Tea Break**	
Session 2 - Immuno-oncology & Tumour Microenvironment		
10:50 – 11:10	Prof. Jun Chen	The mechanism of macrophage-mediated cancer Immunology
11:20 – 11:40	Dr. Kevin Bennewith	Fertilizing the metastatic soil: Recruitment and function of regulatory T cells in promoting metastatic tumour growth in the lungs
11:50 – 12:05 12:05 – 1:00	**Lightning Round** **Lunch Break / Posters**	
Session 3 - Advanced Treatment & Resistance Pathways		
1:00 – 1:05	Dr. Gavin Stuart	*Introduction to advanced treatment and resistance pathways*
1:05 – 1:25	Prof. Musheng Zeng	The key cellular factors in EBV infection of epithelial cells
1:35 – 1:55	Dr. Poul Sorensen	Translational control of cell stress adaptation and tumor metastatic progression
2:05 – 2:25	Prof. Jingping Yun	Liver Cancer Progression Mediated by the Deregulated Metabolism of Glycogen and Lipid – From Abnormal Pathological Pattern to Molecular Mechanism
2:35 – 2:55	Dr. Amina Zoubeidi	Tackling hormone therapy resistance in prostate cancer
3:05 – 3:25	Prof. Bo Li	FBP1-mediated metabolic disorder in cancer
3:35 – 3:50	**Tea Break**	
Session 4 - Comparative Genomics		
3:50 – 3:55	Dr. Chris Maxwell	*Introduction to comparative genomics*
3:55 – 4:15	Prof. Miaoxin Li	A powerful statistical approach for the estimation of cancer-driver genes by accurately modelling background mutation rate
4:25 - 4:45	Dr. Philipp Lange	BRAvE - A pheno-proteogenomics pipeline for prospective personalized treatment stratification in childhood cancer
4:55 - 6:00	**Networking Wine & Cheese**	

DAY 2 - MAY 8		
9:00 – 10:00	**Networking Breakfast**	
Session 5 - Artificial Intelligence & Pathology		
10:00 – 10:05	Dr. David Schaeffer	*Introduction to artificial intelligence & pathology*
10:05 – 10:25	Dr. David Huntsman	Reducing death and suffering for gynecologic cancer: a multidisciplinary team approach
10:35 – 10:55	Prof. Haotian Lin	The AI application in clinical medicine
11:05 – 11:25	Dr. Mike Byrne	The potential of Artificial Intelligence in screening of GI cancer
11:35 – 11:50 12:00 – 1:00	**Lightning Round** **Lunch Break / Posters**	
Session 6 - Stem Cells & Biomedical Engineering		
1:00 – 1:05	Dr. Xiaoyan Jiang	*Introduction to stem cells & biomedical engineering*
1:05 – 1:25	Dr. Florian Kuchenbauer	From tumorsupressor to oncogene and back: plasticity of noncoding RNAs
1:35 – 1:55	Dr. Connie Eaves	Modeling Leukemia in Human Cells
2:05 – 2:25	Dr. Shane Duggan	Epithelial-Cell-Intrinsic Inflammatory Factors, Initiated by Gastro-Esophageal Reflux Disease, support Barrett's-associated Stem Cell Emergence and Esophageal Adenocarcinoma cell growth
2:35 – 3:00	**Tea Break**	
Session 7 - Artificial Intelligence & Pathology		
3:00 – 3:20	Dr. Tony Ng	Molecular diagnostics for cancer pathology – state-of-the-art tools for the practicing pathologist
3:30 – 3:50	Dr. Janel Kopp	Cellular origin affects the phenotype of pancreatic ductal adenocarcinoma
4:00 – 4:20	Dr. Raymond Ng	Data Science for Cancer and Chronic Disease Care
4:30 – 4:35	Closing Remarks by Dr. Rob McMaster & Poster Awards	
4:35 – 5:30	**Networking Wine & Cheese**	

Funding for the Henry Fok Ying Tung SYSU-UBC Medical Research Symposium—Cancer was graciously provided by the Henry Fok Foundation.

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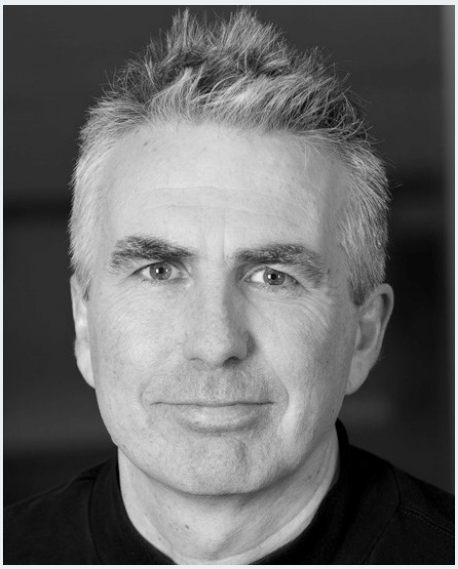
Speakers



Dr. Calvin Roskelley, PhD

- Professor, Cellular & Physiological Sciences, and Obstetrics & Gynaecology, UBC
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Education
Ph.D., University of British Columbia, Canada

Podocalyxin is a driver of pre-metastatic invasive budding in solid tumours

Podocalyxin is a single-pass transmembrane sialomucin that, when overexpressed, is an independent indicator of poor outcome in a number of solid tumor types, including invasive breast carcinoma. In the tumor microenvironment, podocalyxin overexpression is often observed in tumor cells that form invasive, cohesive tumor buds—a histopathological manifestation of collective invasion. Experimentally, we have shown that the forced overexpression of podocalyxin drives the formation of invasive tumor buds in orthotopic breast tumor xenografts without the los of junctional E-cadherin. This overexpression also promotes the collective migration and invasion of these tumor cells in 2D and 3D culture, respectively, independent of an epithelial to mesenchymal transition (EMT). Pathological observation and recent experimental findings have led to suggestions that an overt EMT is not required for breast tumor metastasis. Thus, it is becoming apparent that a mechanistic understanding of collective invasion will be important for detecting and therapeutically targeting metastatic disease in some contexts. Hence we sought to identify the underlying cellular mechanisms driving podocalyxin-dependent increases in collective invasion. Treatment of podocalyxin-overexpressing MCF7 cells with blebbistatin revealed that podocalyin's ability to drive collective invasion was dependent on actomyosin contractility and the activation of the scaffolding protein ezrin. Together, this suggests that the ability of the cytoplasmic tail of podocalyxin to interact with the actin cytoskeleton may contribute to its ability to increase collective tumour cell motility.

Biography

Dr. Roskelley did his graduate training at UBC with Nelly Auersperg, and his post-doctoral training with Mina Bissell at University of California at Berkeley. He is a cell biologist who works with multidisciplinary teams in an effort to convert fundamental research findings into clinically relevant tools that can be used to target breast and ovarian cancer metastasis. Specifically, his group is identifying molecular determinants that regulate changes in adhesion dynamics that drive tumor cell invasion. His laboratory is funded by the Canadian Cancer Society, the Cancer Research Society and the Canadian Institutes for Health Research. He is currently the Chair of the National Advisory Committee on Research for the Canadian Cancer Society Research Institute.

Podxl as a target for immunotherapy in metastatic cancers

Podocalyxin (gene name PODXL, protein name Podxl) is a CD34-related sialomucin implicated in the regulation of cell adhesion, migration and polarity. Upregulated expression of Podxl is linked to poor patient survival in a wide variety of cancers. However, it is not known if podocalyxin has a functional role in tumor progression. Recently, we found that suppression of Podxl expression in breast and ovarian cancer cell lines (through either lentiviral shRNA mediated-suppression or CRISPR-mediated gene inactivation) cripples their ability to form tumors and clonal metastases in xenografted mice. Likewise we find that these cell lines lose their ability to form tumor spheres in vitro, suggesting a suppression of tumor initiating cell activity. Accordingly, in collaboration for the Centre for Drug Research and Development, we developed a novel panel of monoclonal antibodies to human Podxl and have explored their utility in suppressing tumor cell growth in vitro and in vivo. One antibody, Podo83, shows a potent ability to suppress primary tumor growth and the development of distal metastases. Thus, this antibody appears to block a functional epitope on the Podxl core protein. In addition a second antibody, Podo447, recognizes an exquisitely tumor-specific glycoform of Podxl on a subset of tumors. When coupled to a toxic payload this antibody, too, exhibits a strong ability to suppress tumor growth in vivo. Similarly, grafting of the Podo447 antibody V regions onto a chimeric antigen receptor (CAR) and expression in primary T cells, yields T cells with a potent ability to kill Podxl+ tumor cells in vitro. In summary, we find that the stem cell marker Podxl is upregulated on a variety of human tumors, facilitates disease progression and is a promising target for immunotherapy as an approach to blocking metastatic disease.

Biography

Dr. Kelly McNagny obtained his Ph.D. in Cellular Immunology at the U. of Alabama at Birmingham, working with Dr. Max D. Cooper on cell surface proteins that regulate B cell maturation and homing. He then moved to the European Molecular Biology Laboratory in Heidelberg, Germany for his postdoctoral studies in the lab of Dr. Thomas Graf. There his work focused on transcriptional control of hematopoietic stem cell maturation and cell fate. He performed some of the first studies to identify transcription factors that regulate the gene expression and differentiation of eosinophils, which are known to play a major role in allergic and asthmatic responses. In addition, he identified a number of novel hematopoietic stem cell surface proteins (the CD34 family) and began analyzing their function. He continued his studies at the EMBL as a Visiting Scientist prior to starting his own laboratory at UBC in 1998. He is currently a full professor in Medical Genetics at The Biomedical Research Centre where his work focuses on stem cell behavior, innate immune responses, inflammatory disease, cancer biology and therapeutics. In 2015 he also served as the Scientific Director of the Centre for Drug Research and Development (CDRD), a National Centre of Excellence aimed at translating early stage scientific discoveries into therapies. Kelly fills leadership roles in the Canadian Stem Cell Network Centre of Excellence, and is the Associate Director of the Canadian AllerGen Network Centre of Excellence, and Co-Director of its Biomarkers and Bioinformatics Platform.

Dr. Kelly McNagny, PhD

- Professor, Medical Genetics, UBC
- Canadian Stem Cell Network Centre of Excellence (Sub-Chair of the Trainee Education Committee)
- Associate Director, Canadian AllerGen Network Centre of Excellence
- Co-Director, Canadian AllerGen Network Biomarkers and Bioinformatics Platform
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Education:

Ph.D., Cellular Immunology, University of Alabama at Birmingham, USA (1990)

Post-doctoral Fellow, European Molecular Biology Laboratory, Heidelberg, Germany (1991-1996)

Dr. François Bénard, MD

- Vice President, Research, BC Cancer
- Distinguished Researcher, BC Cancer Research Centre
- Professor, UBC Department of Radiology
- BC Leadership Chair in Functional Cancer Imaging
- fbenard@bccrc.ca

Education

Fellowship, Radiology, University of Pennsylvania (1995-1998)

M.D., Université de Sherbrooke (1991)

Optimizing radiopharmaceuticals for cancer imaging and radioligand therapy

The widespread adoption of positron emission tomography for cancer imaging has stimulated the development of improved approaches to radiolabel and optimize cancer targeting molecules, including small molecules, peptides and antibodies. In turn, this has led to a resurgence in interest in therapeutic radiopharmaceuticals using beta or alpha particles to treat advanced metastatic cancer. We have developed several new optimized radiolabeled peptides targeting g-coupled receptors and enzymes overexpressed in breast, prostate and lymphoid cancers. This presentation will showcase technologies such as improved radiofluorination approaches, peptide stabilization to decrease metabolic inactivation, and the use of albumin binders to optimize the delivery of radiopharmaceuticals to cancer cells. We will also highlight how the tumour microenvironment affects the distribution of radiopharmaceuticals and how it can be exploited to deliver a radioactive payload to cancer cells.

Biography

François Bénard is the Vice President, Research at BC Cancer, a distinguished scientist at the BC Cancer Research Center and Professor in the Department of Radiology at the University of British Columbia. He holds the BC Leadership Chair in Functional Cancer Imaging. As a clinician scientist, his research interests are in positron emission tomography (PET), nuclear medicine, cancer imaging and targeted radionuclide therapy. His team developed several new radiopharmaceuticals targeting tumour receptors, notably peptides and small molecule ligands. He initiated the program that developed cyclotron production of 99mTc, now in clinical trials at multiple sites in Canada. He has established extensive multidisciplinary collaborations, and he and his colleagues were awarded the 2015 Brockhouse Canada Prize for Interdisciplinary Research in Science and Engineering by NSERC.

Treat the cancer soil: Turn foes to friends

Current paradigms of cancer-centric therapeutics are usually not sufficient to eradicate the malignancy, while cancer stroma may prompt tumor relapse and therapeutic resistance. Among all the stromal cells that populate the tumor microenvironment, tumor-associated macrophages (TAMs), cancer-associated fibroblasts (CAFs) and tumor-infiltrated lymphocytes (TILs) are the most abundant and are critically involved in cancer progression. Tumor stromal cells regulate the biology of tumor cells via cell-cell contact, numerous regulatory factors and synthesizing or remodeling the extracellular matrix, and thus affect cancer initiation and development. Therefore, recent characterization of tumor microenvironment brings stroma-targeting therapies for cancer treatment onto the agenda. In our studies, we not only underscore the contribution of tumor stroma components to cancer progression, but also highlight the relevant translational advances and potential therapeutic strategies that target tumor soil for cancer treatment.

Biography

Erwei Song is Professor of Breast Surgery at Sun Yat-sen University (SYSU), Distinguished Professor of China Medical Board (CMB) and Fellow of the Royal Society of Biology (UK). Currently, he serves as Dean of Zhongshan School of Medicine and President of Sun Yat-sen Memorial Hospital at Sun Yat-sen University.

Prof. Song has focused his research on the microenvironment of malignant tumors, mainly on breast cancers, and unraveled the regulatory mechanisms of non-coding RNAs (ncRNAs) related to tumor microenvironment. He has published 126 research articles in SCI-indexed journals, including Cell, Cancer Cell, Nature Immunology, Nature Medicine, Nature Biotechnology, Science Translational Medicine, Cell Research and so on. The total SCI citation frequency is more than 7000 so far. As a corresponding author, one of his articles has been cited 1137 times. Also, he is leading four ongoing national research projects, and serves as associate editor for BMC Cancer, Cancer Science and Science China-Life Sciences, and reviewing editor for Journal of Biological Chemistry. He has received numerous honors, including the "Ho Leung Ho Lee Foundation Award" in 2013, and the "National Natural Science Award" by the State Council of China in 2015.

Prof. Erwei Song, MD, PhD

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- President, Sun Yat-sen Memorial Hospital, SYSU
- Distinguished Professor, China Medial Board (CMB)
- Fellow, Royal Society of Biology (UK)
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Education:

M.D., Sun-Yat-Sen University of Medical Science, China (1993)

Ph.D., Experimental Hepato-billiary Surgery, Sun-Yat-Sen University, China (2000)

Postdoc Research Fellow, Transplant Immunology, Essen University Hospital, Germany (2001)

Postdoc Research Fellow, CBR Institute of Biomedical Research, Harvard Medical School, USA (2003)

Prof. Jun Chen, PhD

- Professor, Zhongshan School of Medicine, SYSU
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Education

Ph.D.,Institute of Biophysics, Chinese Academy of Sciences, China (2013)

Postdoc Research Fellow, Montreal Clinical Research Institute, Canada (2018)

The mechanism of macrophage-mediated cancer immunology

Cancer cells elude anti-tumour immunity through multiple mechanisms, including upregulated expression of ligands for inhibitory immune checkpoint receptors. Phagocytosis by macrophages plays a critical role in cancer control. Therapeutic blockade of signal regulatory protein (SIRP)-a, an inhibitory receptor on macrophages, or of its ligand CD47 expressed on tumour cells, improves tumour cell elimination in vitro and in vivo, suggesting that blockade of the SIRPa-CD47 checkpoint could be useful in treating human cancer. However, the pro-phagocytic receptor(s) responsible for tumour cell phagocytosis is(are) largely unknown. Here we find that macrophages are much more efficient at phagocytosis of haematopoietic tumour cells, compared with non-haematopoietic tumour cells, in response to SIRPa-CD47 blockade. Using a mouse lacking the signalling lymphocytic activation molecule (SLAM) family of homotypic haematopoietic cell-specific receptors, we determined that phagocytosis of haematopoietic tumour cells during SIRPa-CD47 blockade was strictly dependent on SLAM family receptors in vitro and in vivo. In both mouse and human cells, this function required a single SLAM family member, SLAMF7 (also known as CRACC, CS1, CD319), expressed on macrophages and tumour cell targets. In contrast to most SLAM receptor functions, SLAMF7-mediated phagocytosis was independent of signalling lymphocyte activation molecule-associated protein (SAP) adaptors. Instead, it depended on the ability of SLAMF7 to interact with integrin Mac-1and utilize signals involving immunoreceptor tyrosine-based activation motifs. These findings elucidate the mechanism by which macrophages engulf and destroy haematopoietic tumour cells. They also reveal a novel SAP adaptor-independent function for a SLAM receptor. Lastly, they suggest that patients with tumours expressing SLAMF7 are more likely to respond to SIRPa-CD47 blockade therapy.

Biography

Dr. Jun Chen have been engaged in research on tumor immunology since his PhD training. During Dr. Chen's postdoctoral work at the Montreal Clinical Research Institute in Canada, he focused on the mechanism of macrophage-mediated cancer immunotherapy (CD47-Sirp pathway). By studying 20 different knockout mouse strains and establishing multiple macrophage-specific mouse tumor models, the first phagocytic receptor SLAMF7 and the whole macrophage phagocytic receptor signaling pathway were first discovered internationally. (Nature, 2017; Trends in Immunololy, 2018). During Dr. Chen's PhD training, he identified a new mechanism by granzyme K-mediated ER stress to kill tumor cells by NK cells. In addition, he identified a novel gene named ERAdP and analyzed its new pathway to promote anti-tumor and anti-infection functions of NK cells.

For now, his main research interest is macrophage-associated tumor immunology. The main research directions include: (1) the mechanism and application of CD47-Sirp immune checkpoint pathway in cancer immunotherapy; (2) the discovery of novel immune checkpoint pathways on macrophages; (3) Function study of macrophages in tumor microenvironment.

Fertilizing the metastatic soil: Recruitment and function of regulatory T cells in promoting metastatic tumour growth in the lungs

It is estimated that over 90% of cancer-related deaths are associated with metastatic disease. The development of tumour metastases requires collaboration between the metastatic tumour cells (seeds) and host tissue (soil), with recent evidence indicating primary tumours can promote the development of a pro-metastatic microenvironment in some organs. We and others have shown that metastatic tumours can produce cytokines that stimulate the expansion and accumulation of immune suppressive myeloid cells (including myeloid-derived suppressor cells and macrophages) in metastatic target organs. Immune suppressive cells normally mediate peripheral tolerance, prevent autoimmunity, and limit chronic inflammation, but the aberrant accumulation of immune suppressive cells in tissues can protect metastatic tumour cells from immune attack and ultimately promote metastatic tumour growth. We are interested in understanding how primary tumours affect the accumulation and function of immune suppressive cells in the lungs with the goal of developing novel therapeutic strategies to treat metastatic cancer. This talk will focus on our recent work studying regulatory T cell (Treg) recruitment to the lungs and the influence of Tregs on metastatic tumour growth, including strategies to disrupt Treg accumulation and function to decrease metastatic tumour growth.

Biography

Dr. Kevin Bennewith is a Senior Scientist at BC Cancer and an Associate Professor in Pathology and Laboratory Medicine at UBC. After completing his PhD at UBC studying poorly oxygenated (hypoxic) tumour cells, he pursued post-doctoral training at Stanford University where he studied the role of the hypoxia-induced secreted proteins connective tissue growth factor and lysyl oxidase in solid tumour growth and metastasis. Dr. Bennewith's current research interests include the role of the solid tumour microenvironment and poorly oxygenated (hypoxic) tumour cells in promoting resistance to cancer treatment, tumour progression, and metastasis. He is also interested in how immune suppressive cells protect metastatic tumour cells from immune attack in distant tissues. Dr. Bennewith is a Michael Smith Foundation for Health Research Scholar and research in his laboratory has been funded by the Terry Fox Foundation, the BC Cancer Foundation, the Cancer Research Society, and the Canadian Institutes of Health Research.

Dr. Kevin Bennewith, PhD

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- Associate Professor, Pathology and Laboratory Medicine, UBC
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Education:
Ph.D., Pathology and Laboratory Medicine, University of British Columbia (2004)

Prof. Musheng Zeng, PhD

- Professor and Vice-President, Sun Yat-sen University Cancer Center
- zengmsh@sysucc.org.cn

Education

Ph.D., Sun Yat-sen University of Medical Sciences (1998)

Postdoc Research Fellow, Tennessee State University of Medical Center and New England Medical Center, Tufts University, USA (2003)

The key cellular factors for Epstein-Barr virus infection of nasopharyngeal epithelial cells

Epstein-Barr virus (EBV) is implicated as an etiological factor in B lymphomas and undifferentiated nasopharyngeal carcinoma (NPC), a malignant epithelial cancer occurring frequently in South China and Southeast Asia. However, the mechanisms of cell-free EBV infection of nasopharyngeal epithelial cells (NEPCs) remain elusive, mainly due to the deficiency of highly susceptible NPECs model. We focus on establishing the highly susceptible NPECs model and identifying cellular factors responsible for EBV infection. We found EGF treatment and premalignant NPECs-Bmi1 cells grown as sphere-like cells (SLCs) significantly enhanced EBV infection. We then used cDNA array, siRNA library and pull-down assay followed by liquid chromatography-tandem MS to screen the key cellular factors involved in EBV infection of epithelial cells. We found that non-muscle myosin heavy chain IIA (NMHCIIA) interacts with EBV gH/gL and mediates EBV attachment with the epithelial cells. EphA2 interacts with both gH/gL and gB and mediates EBV fusion of cellular membrane, while neuropilin 1 (NRP1) interacts with EBV gB and triggers the activation of EGFR/RAS/ERK signaling induced by EBV. Taken together, NMHCIIA, EphA2 and NRP1 are identified as cellular factors in EBV infection of NPECs, indicative of their capacity to serve as targets for blocking EBV infection.

Biography

Prof. Mu-Sheng Zeng is a vice president and professor of Sun Yat-sen University Cancer Center. He gained his Ph.D. degree at Sun Yat-sen University of Medical Sciences in 1998 and had his Post-doc training in Tennessee State University and New England Medical Center, Tufts University from 1999 to 2003. Prof. Mu-Sheng Zeng moved back to China to start his lab in 2003. He was granted the funding of "National Distinguished Young Scholar" in 2010, and was honored as "Yangtze Scholar Distinguished Professor" in 2014. He was invited to give a talk in various international conference including Gordon Research Conference (NPC) in both 2016 and 2018, and was elected as a co-chairman of 2022 Gordon Research Conference for NPC.

Zeng's laboratory majorly engages in the etiology, pathogenesis of Nasopharyngeal Carcinoma (NPC). He characterized the epithelial cell receptors for EBV infection, and illustrated the mechanism of EBV infection and transformation in nasopharyngeal epithelial cells. He has published more than 70 papers in the area of virology and cancer research, including journals such as: Nature Microbiol, PNAS, J Clin Invest, JNCI, Nature Commun, Cancer Res, Clin Cancer Res, et al.

Translational control of cell stress adaptation and tumor metastatic progression

In aggressive sarcomas such as Ewing sarcoma (EwS), the single-most powerful predictor of poor outcome is metastatic disease, highlighting the critical need to identify new factors driving sarcoma metastasis. Sarcoma cells are continually exposed to acute stress in the tumor microenvironment, including oxidative stress and hypoxia. Each is potentially lethal unless tumor cells can acutely adapt to them. Successful adaptation can then lead to emergence of clones with aggressive behaviour, including metastatic capacity. We posit that stress-induced cell plasticity acquired through acute changes in mRNA translation drives tumor fitness for metastasis. YB-1, an RNA binding protein (RBP), translationally activates HIF1A mRNAs to activate HIF1 synthesis under hypoxia, and mRNAs encoding the stress granule (SG) nucleating factor, G3BP1, under oxidative stress to mediate SG formation. SGs are cytosolic structures containing stalled translation initiation complexes, RBPs, 40S ribosomes, and silenced mRNAs, induced under diverse stresses to reduce translation. We found that blocking YB-1 translational activation of its cytoprotective mRNA targets markedly reduces EwS metastasis in vivo. We hypothesize that this decreases the fitness of sarcoma cells to metastasize. Small molecule screens to search for inhibitors of YB-1 translational activity, using fluorescent detection of SG formation in vitro as a tractable readout, identified several classes of compounds that potently block SGs. These agents reduce acute synthesis of multiple YB-1 induced cytoprotective factors and dramatically inhibited metastasis of sarcoma cells to lungs in mouse models. Targeting this process therefore offers a promising strategy to reduce the burden of metastatic disease in high-risk sarcomas.

Biography

Dr. Poul Sorensen is a molecular pathologist and cancer biologist specializing in the genetics and biology of pediatric cancers. He is a principal investigator on the recently awarded Stand Up 2 Cancer Pediatric Cancer Dream Team grant focused on immunotherapeutic approaches to targeting high-risk childhood cancers. His group runs several major components of this prestigious award, including the Pathology Core.

Dr. Sorensen's laboratory focuses on using both genetic approaches (such as next-generation sequencing) and biochemical methods (e.g. proteomics) to identify deregulated signaling cascades in childhood cancer cells. His group has discovered many novel translocation associated alterations in childhood cancer. Moreover, the group has extensive renown in utilizing genetic findings as a means to characterize relevant cancer biology. Current work is focused on how cancer cells respond to acute stress. The overarching hypothesis is that adaptation to such stresses through altered mRNA translation and protein synthesis leads to tumour cell clonal selection and metastasis. The group therefore utilizes a variety of proteomic and other techniques to probe the "translatome" of stressed tumour cells to identify new targets for therapy in aggressive human solid tumours.

Dr. Poul Sorensen, MD, PhD

- Distinguished Scientist, Molecular Oncology, BC Cancer Research Centre
- Professor, Pathology & Laboratory Medicine, UBC
- Johal Chair in Childhood Cancer Research, UBC
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Education:

Ph.D., University of British Columbia, Canada (1990)

M.D., University of British Columbia, Canada (1984)

Fellow of the Royal College of Physicians and Surgeons of Canada (FRCPC), Anatomical Pathology (1991)

Prof. Jingping Yun, PhD

- Director, Dept of Pathology, Sun Yat-sen University Cancer Center
- Vice-Chairman, Committee of Tumor Pathology, Chinese Society of Clinical Oncology
- Chairman, Committee of Pathology of Guangdong Provincial Anticancer Association
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Education
Ph.D., Chinese University of Hong Kong (2001)

Liver Cancer Progression Mediated by the Deregulated Metabolism of Glycogen and Lipid --From Abnormal Pathological Pattern to Molecular Mechanism

Hepatocellular carcinoma (HCC) is a rapidly proliferating malignant tumour that requires a large amount of energy supply but usually under nutrition deprivation stress. However, the metabolic features of HCC cells and hepatocytes remains largely unknown. In the present study, we found two pathological patterns that concerning the abnormal glycogen regulation and dysregulation of lipid metabolism in HCC. In the first session, we showed that glycogen content is noticeably decreased in HCC tissues. Glycogen synthase 2 (GYS2) was significantly downregulated and correlated with decreased glycogen content and unfavorable patient outcomes in HCC. GYS2 overexpression suppressed, whereas GYS2 knockdown facilitated cell proliferation in vitro and tumor growth in vivo via modulating p53 expression. Acetylation of p53 at K373/382, which in turn inhibited the transcription of GYS2 in the support of HBx/HDAC1 complex. In the second part, we demonstracted that de novo lipogenesis in hepatocytes could be markedly enhanced by HCC-derived insulin growth factor 2 (IGF2), resulting in an increased secretion of fatty acids (FAs). The FAs were taken up by cancer cells via the coordination of fatty acid binding protein 4 (FABP4) and CD36 to induce leptin-mediated metabolic reprogramming in favour of fatty acid oxidation, and consequently to support the rapid growth of HCC cells. Our findings may broaden our insights of HCC metabolism, which provided novel therapeutic targets for the clinical management of HCC.

Biography

Professor Jing-Ping Yun graduated in medicine of Hunan Medical College, Changsha city in 1985 and then worked as pathologist for nearly 30 years, and received his Ph.D. in Pathology from the Chinese University of Hong Kong in 2001. Dr. Yun is the director of Department of Pathology of Sun Yat-sen University Cancer Center, Guangzhou. He was elected the vice chairman of Committee of Tumor Pathology of Chinese Society of Clinical Oncology, and the chairman of Committee of Pathology of Guangdong Provincial Anticancer Association. The research interests of Yun laboratory focus on the identification of potential biomarkers in differential diagnosis of cancers, and the aberrant signaling pathways in the progression of gastrointestinal malignancies, mainly in liver cancer. Dr. Yun is fascinated with and actively engaged in moving his laboratory findings to translational applications, with the overarching goal to establish novel strategies for routine pathological examination and clinical management of liver cancer. He has published over 70 articles in the international journals including Hepatology, Cancer Research and Oncogene.

Tackling hormone therapy resistance in prostate cancer

Resistance to newly developed androgen receptor pathway inhibitors (ARPIs), such as abiraterone and enzalutamide, rapidly emerges and patients generally die within two years. In particular, a subset of patients who relapse following ARPI therapy exhibit lineage switching whereby tumours shed their dependence on AR signaling and emerge with neuroendocrine features. These tumours, termed treatment induced neuroendocrine prostate cancer (t-NEPC), carry an extremely poor prognosis and, to date, treatment remains decades old cytotoxic chemotherapy which carries a short-lived response at the cost of significant toxicity. Thus, the need to develop targeted treatments for this devastating disease is of paramount importance. Dr Zoubeidi will discuss how cell plasticity including cancer stem cells and neuroendocrine are mechanisms of hormone therapy resistance and how this is driven epigenetically and why the transcription factor BRN2 is a major regulator/driver and a promising target for t-NEPC.

Biography

Dr. Zoubeidi's research program aims to uncover how standard care of prostate cancer therapy targeting the androgen receptor (AR) induces treatment resistance and controls phenotypic plasticity, which has been associated with the clinically relevant problem of drug resistance in prostate cancer. Her multifaceted research program addresses the importance of various signaling pathways in this inherently heterogeneous disease. She has published over 85 peer-reviewed manuscripts in Cancer Discovery, Cell, Nature communication, Cell Report, Cancer Research and others. She is a Michael Smith Scholar and has been awarded the Terry Fox Young Investigator and Prostate Cancer Foundation USA Young Investigator awards. She received substantial funding awards from national and international funding agencies as a principal investigator or co-investigator that together total over $16 million. Her track record of research excellence is underscored by numerous accolades from the American Association for Cancer Research, the American Urological Association, the Northwest Urological Society and others. She has been invited to speak at numerous national and international conferences on molecular mechanisms and drug targets of prostate cancer, and as a visiting professor at different Universities. She serves on several grant panel review committees including NIH, CIHR, PCa Canada and Prostate Cancer Foundation USA. She is a member of the editorial board of Endocrine Related Cancer and an ad hoc reviewer for numerous journals including Science Translational Medicine, Nature Cell Biology, EMBO, Oncogene, the AACR journals to name a few. In recognition of her meritorious achievements, she was awarded twice the UBC Faculty of Medicine Distinguished Achievement Award for excellence in basic science.

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Prof. Bo Li, PhD

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Education

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FBP1-mediatedm metabolic disorder in Cancer

Cancer cells are frequently subjected to tumor-associated metabolic disorders. We discovered that expression of the gluconeogenic enzyme fructose-1, 6-bisphosphatase (FBP1) is uniformly suppressed in renal tumors compared to normal kidneys. FBP1 inhibits glycolysis in renal cancer cells through its metabolic activity, and suppresses hypoxia-inducible factors in a catalytic activity-independent manner. In addition, FBP1 is universally silenced in hepatocellular carcinoma, and hepatocyte-specific Fbp1 deletion disrupts liver metabolism leading to fibrosis and steatosis in mice. Mechanistically, FBP1 loss accelerates hepatic tumor progression by eliciting a senescence-associated secretory phenotype in hepatic stellate cells. Furthermore, FBP1-deficient hepatocytes signal to stellate cells by releasing HMGB1, whereas blocking HMGB1 release limits the stellate cell senescence and tumor progression. These results identified underappreciated oncogenic events in renal and hepatic tumorigenesis, and provide new mechanistic link between cell metabolism, gene transcription, and immune responses.

Biography

Cancer cells are frequently exposed to intratumoral stresses including nutrient and oxygen deprivation due to poorly-formed vasculature in solid tumors. Dr. Bo Li's goal is to investigate the molecular mechanisms whereby cancer cells reprogram their metabolism to survive within nutrient-limited environments. Recently Dr. Li determined that expression of the gluconeogenic enzyme fructose-1, 6-bisphosphatase (FBP1) is uniformly suppressed in renal cell carcinoma compared to normal kidneys. FBP1 inhibits glycolysis in RCC cells through its metabolic activity, and suppresses hypoxia-inducible factors in a catalytic activity-independent manner. These results identified an underappreciated oncogenic event in RCC and defined a novel feedback loop from cell metabolism to gene transcription. Dr. Li's future plan is to build upon these findings and dissect the molecular details underlying FBP1 loss-mediated metabolic reprogramming and tumorigenesis.

A powerful method for the estimation of cancer-driver genes using a weighted iterative regression accurately modelling background mutation rate

Genomic identification of driver mutations and genes in cancer cells are critical for precision medicine. Due to difficulty in modeling distribution of background mutations, existing statistical methods are often underpowered to discriminate driver genes from passenger genes. Here we propose a novel statistical approach, weighted iterative zero-truncated negative-binomial regression (WITER), to detect cancer-driver genes showing an excess of somatic mutations. By solving the problem of inaccurately modeling background mutations, this approach works even in small or moderate samples. Compared to alternative methods, it detected more significant and cancer-consensus genes in all tested cancers. Applying this approach, we estimated 178 driver genes in 26 different cancers types. In silico validation confirmed 90.5% of predicted genes as likely known drivers and 7 genes unique for individual cancers as very likely new drivers. The technical advances of WITER enable the detection of driver genes in TCGA datasets as small as 30 subjects, rescuing more genes missed by alternative tools. The tool is available at http://grass.cgs.hku.hk/limx/witer/.

Biography

Dr. Miaoxin Li is a full professor of Sun Yat-Sen University and an honorary assistant professor of the University of Hong Kong. He obtained his PhD degree at the University of Hong Kong in 2009. He mainly focuses on two research fields: 1) methodological innovations in bioinformatics and statistical genetics for identification of genetic factors of human diseases; and 2) genetic mapping studies of specific human diseases (including schizophrenia and liver cancers), in which he has spent over 15 years and published over 50 peer-reviewed papers (some first-authored ones in the top professional journals, including American Journal of Human Genetics, Genome Biology, PLOS Genetics and Nucleic Acids Research). He has developed several bioinformatics and statistical software tools for genetics mapping of human traits and diseases [e.g., KGGSeq].

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Dr. Philipp Lange, PhD

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Education
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BRAvE – towards personalized treatment stratification in childhood cancer

Personalized treatments have significant potential to improve therapy options for high-risk cancers and reduce late effects in general. The approach is still in its infancy and much work is needed to improve its utility and efficacy. This is particularly true for childhood cancers, that, as we are only beginning to appreciate, are fundamentally different from adult malignancies.

Simply applying the lessons learned from adult malignancy is not necessarily an effective approach to pediatric disease.

Here I discuss how BRAvE (**B**etter **R**esponses through **Av**atomics **E**vidence), a research-based precision medicine initiative, addresses these challenges to develop an effective precision medicine pipeline for pediatric cancer. I will focus on our recent advances in three key areas: (1) Rapid identification of actionable changes and associated targeted therapies by genomics and proteomics; (2) Development of xenograft (avatar) models that enable proactive deep characterization of individual patient cancers before the relapse occurs; (3) Treatment stratification and response validation incorporating evaluation phenotypic and proteomic responses to single- or combination-therapies in patient-derived cell based and xenograft models.

With the vision to deliver tomorrow's treatments today, BRAvE is set up to change precision medicine for pediatric oncology from an approach that is reactive to disease progression to one that is proactive by initiating analysis of each child's cancer sample at diagnosis.

Biography

Dr. Philipp Lange received his PhD in Biochemistry from the Free University Berlin, Germany after earning an MSc in Molecular Biology, Microbiology and Computer Sciences from the University of Hamburg, Germany. During his PhD at the Max Delbruck Centre for Molecular Medicine, Berlin, Germany he studied the molecular causes of hereditary osteopetrosis in children and patented a new drug target for the treatment of osteoporosis in women.

In 2009 he moved to Vancouver, BC for his postdoctoral work with Dr. Christopher Overall. At the Centre for Blood Research, UBC he advanced mass spectrometry based proteomics to study post-translational protein modification in breast cancer. He is also the developer and curator of the biological knowledgebase and data analysis platform TopFIND.

Since 2015 Dr Lange is Assistant Professor in the Department of Pathology and Laboratory Medicine at UBC and Investigator at the BC Children's Hospital and BC Cancer Research Centre. As Canada Research Chair in 'Translational Proteomics of Pediatric Malignancies' he heads an integrated proteomics and bioinformatics driven research program investigating the molecular basis and new treatment avenues in childhood cancer.

Reducing death and suffering for gynecologic cancer: a multidisciplinary team approach

Our team is motivated by our goal of reducing death and suffering for gynecologic cancers by 50%. We have coalesced around three programs: i) biologically informed prevention strategies, ii) precision diagnostics and oncology and iii) biologically and socio-culturally informed survivorship strategies. All are considered within a population based context. The first two objectives will be described and my own research program which used normal tissue derived organdies to determine how cell context, mutation and microenvironment interact to create oncogenic opportunity.

Biography

Dr. David Huntsman is a Professor in the Departments of Pathology and Laboratory Medicine and Obstetrics and Gynaecology at The University of British Columbia (UBC) and is the Dr. Chew Wei Memorial Professor of Gynaecologic Oncology. He is a a Staff Pathologist at the BC Cancer Agency (BCCA), and a Consulting Pathologist at the Vancouver General Hospital (VGH).

Dr. Huntsman is currently the Director of the BC multidisciplinary ovarian cancer research team (OvCaRe), Medical Director of the Centre for Translational and Applied Genomics (CTAG) at the BCCA, and co-Director of the Genetic Pathology Evaluation Centre (GPEC) at the Jack Bell Research Centre, VGH.

Dr. Huntsman research has led to development of predictive and prognostic tissue based cancer biomarkers for ovarian cancer and a wide variety of other tumour types. His team created a blueprint for subtype specific ovarian cancer control and have been leaders in the application of novel genomics technologies to ovarian cancer. As collaboration is critical in his field, Dr. Huntsman happily leads and engages in a wide number of multidisciplinary research groups. Most recently he has been working on the creation of broad based personalized medicine initiative for British Columbia.

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Prof. Haotian Lin, MD

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Education
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AI Application in Clinical Medicine

High quality medical resources remain unevenly distributed and hard to access, but traditional reforms in medical system have little effect in improving it. An efficient and reliable medical decision making platform is needed to prove more high-level medical services and meet the demands of different kinds of patients. The artificial Intelligence (AI) offers an opportunity to achieve it. Currently, AI has been adopted in multiple medical scenarios and exhibited its superiority, including diagnosis, treatment, prognosis evolution, disease prediction, and clinician workflow. The AI has been adopted in the diagnosis of ocular diseases, cardiovascular diseases, orthopedic diseases, and cerebral disease with an accuracy of more than 95% based on the images and literal data. In some more functional models, the AI platforms can give therapeutic regimens and potential prognosis according the diagnosis results. Moreover, the AI systems are utilized to achieve disease prediction, involving the prediction of high myopia, and the prediction of cardiovascular diseases based on fundus images. Besides, the data entry of forms and text fields are improved with the use of machine-learning techniques such as voice dictation, and automatic summarization, which increase efficiency, ease documentation, improve automated clinical workflow, and allow clinicians to spend more time with their patients. In brief, the current achievements of medical AI are abundant and rewarding, and further advancements are required to extend the applications of AI in clinic medicine and increase the supplement of high quality medical resources.

Biography

Prof. Haotian Lin is a PhD supervisor, the winner of Outstanding Youth Science Foundation, the chief scientist of National Key R&D Program of China, the "Youth Pearl River Scholar" of Universities and Colleges of Guangdong Province, and the leader of technology innovation of "special support plan" of Guangdong Province. Prof. Lin also serves as the director of Department of Artificial Intelligence and Big Data of Zhongshan Ophthalmic Center of Sun Yat-Sen University, the deputy director of the Intelligent Medical Committee of the Chinese Artificial Intelligence Society, the young member of the Ophthalmology Academy of Chinese Medical Association, the director of the Translational Medicine of Guangdong Province, and in the editorial board of several international SCI journals. Prof. Lin is active in clinical practice as well as academic activity. He has published more than 60 SCI papers as the first and corresponding authors in the top international journals like "Nature", "Science", "Lancet", "BMJ", and "PLoS Med" in the past five years. Among them, a new cataract therapy successfully applied in clinical was published (Nature 2016), and was named as one of the "eight breakthroughs in life medicine in 2016" by Nature Medicine. He built the first cloud platform of intelligent diagnosis and treatment of cataract in the world (Nat. Biomed. Eng. 2017, cover paper), and is the creator of the world's first intelligently ophthalmic clinic in Guangzhou, which was selected as one of "the 11 major AI events affecting the global medical community" by IEEE Spectrum and was the only selected one completed by a Chinese team.

The potential of Artificial Intelligence in screening of GI cancer

As a tool for the screening and diagnosis of diseases in the gastrointestinal (GI) cancer, artificial intelligence (AI) is advancing rapidly. There are many emerging technologies to improve screening and diagnosis of GI cancers, but these will struggle if they do not also have some kind of built-in AI or machine intelligence.

Due to the limitations of the human eye, a form of AI known as deep learning is increasingly considered the answer for improving the accuracy of diagnostic screening tests such as colonoscopy in colon cancer and polyp screening. Deep learning employs neural networks that use a large number of features in the task of discrimination. For example, a computer can consider a thousand features when evaluating a polyp, which is way beyond what the human eye can do.

Several groups have developed advanced machine learning systems for screening colonoscopy. In a study from my own group, we found that AI had a greater than 94% accuracy in distinguishing adenomas from hyperplastic polyps using histopathology as a gold standard.

However, the work in screening colonoscopy for colon cancer is just the beginning for AI. The same principles are relevant and being pursued for other GI malignant conditions, such as dysplasia screening in patients with Barrett's esophagus (BE), and the assessment of early gastric cancer, to name just two opportunities.

Overall, there is abundant evidence that optical biopsy with AI is feasible. Clinical applications are approaching quickly. The technology will need a "human in the loop" as it enters clinical practice initially, but will play a significant role in investigation of GI cancers.

Biography

Specializing in gastroenterology and interventional endoscopy, Dr. Byrne is currently a Clinical Professor of Medicine in the Division of Gastroenterology, Director of the Advanced Endoscopy Fellowship at VGH, and was the Director of Endoscopy at Vancouver General Hospital/University of British Columbia from 2012-2014.

His research interests range from basic science to clinical medicine, and he has hands-on research training in molecular science and pharmacology. His clinical experience and research interests include many aspects of interventional endoscopy (especially in pancreaticobiliary disease), and general gastroenterology and the overlaps with oncology and diagnostic imaging, as well as expertise in optical biopsy and artificial intelligence.

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Education

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From tumorsupressor to oncogene and back: plasticity of noncoding RNAs

Based on their binding properties, miRNAs are able to target more than one signaling cascade, thus affecting many physiological and pathophysiological processes. Recent studies showed that the amount and the presence of targets determine the function of a single miRNA. Here, we review how non-coding RNAs can tip the balance between normal and aberrant hematopoiesis and highlight this through recent research projects of our group.

Biography

As a scientist, the work of Dr. Kuchenbauer's laboratory has focused on non-coding RNAs, especially microRNAs (miRNAs) in acute myeloid leukemia (AML) and normal hematopoiesis. As a physician, Dr. Kuchenbauer specialized in hematopoietic stem cell transplantation (HSCT) in adults, where he developed a clinical interest in optimizing current treatment protocols such as conditioning regimens for HSCT.

His team's current research is mainly translational and focuses on interactions between the stem cell niche and allogenic stem cell transplantation, mechanisms of leukemogenesis as well as innovative treatment strategies for multiple myeloma. He and his team are driven to improve patients´ lives by addressing clinical "every day" challenges with state-of-the-art research including preclinical animal models and to translate our research into clinically applicable therapies.

Modeling Leukemia in Human Cells

Human acute leukemias comprise a largely lethal group of malignancies with different mutations and biological features. Nevertheless, they are all characterized by the presence of a rapidly expanding clone of "blasts" that are unable to differentiate into normal blood cells. Different mutations in the leukemic cells allow approximately half of the diseases to be subdivided into groups that have different prognoses. However, to date, very few of these mutations have enabled more effective treatments to be identified. To address this challenge, we have focused on developing systems in which human acute leukemias are generated de novo from primary sources of human cells that are first transduced with selected transforming genes and the cells are then transplanted into permissive immunodeficient mice. Although this approach has been largely unsuccessful in the past, we have now used it to develop several models of human leukemia that have features of significant clinical relevance, starting either with cells from patients with chronic phase chronic myeloid leukemia, or from completely "normal" cord blood or adult bone marrow cells. The efficiency and reproducibility of generating these models make them attractive systems for investigating the initial stages of leukemic transformation and identifying and testing new vulnerabilities.

Biography

Dr. Eaves holds a BA in Biology & Chemistry and a MSc in Genetics from Queen's University, and a PhD in Immunology from the University of Manchester, UK. Following post-doctoral training at the Ontario Cancer Institute under Dr. James Till in collaboration with Dr. Ernest McCulloch, she joined the faculty of the BC Cancer Agency and the University of British Columbia in 1973. In 1981, she and her husband co-founded the Terry Fox Laboratory at the BC Cancer Agency where they jointly developed an internationally recognized research program that continues to address fundamental questions in normal and cancer stem cell biology. Dr. Eaves' contributions include the development and use of quantitative methods to molecularly and biologically characterize rare, functionally defined cells of the blood-forming system, the mammary gland, leukemia, and breast cancer that have become gold standards. More recently, her group has pioneered the creation of new models of human leukemia and breast cancer starting from primary sources of human cells. She has published more than 500 papers and has a long track record as a scientific leader and devoted mentor of >100 postgraduate trainees at all levels in multiple disciplines. She has also been an energetic lifelong contributor to the development of science policy and management in Canada and abroad, and maintains an active role as scientific editor and reviewer. She has received many prestigious national and international awards for her numerous and diverse accomplishments, including serving as President of the former National Cancer Institute of Canada, election as a Fellow of the Royal Societies of Canada and Edinburgh, and most recent selection as a 2019 Inductee into the Canadian Medical Hall of Fame and recipient of the 2019 Gairdner-Wightman Award.

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Epithelial-Cell-Intrinsic Inflammatory Factors, Initiated by Gastro-Esophageal Reflux Disease, Support Barrett's-Associated Stem Cell Emergence and Esophageal Adenocarcinoma Cell Growth

Esophageal cancer patients suffer some of the poorest survival rates of all cancer types. Esophageal squamous cell carcinoma (ESCC) and esophageal adenocarcinoma (EAC) differ dramatically in their cell of origin deriving from either squamous- or intestinal metaplasia-associated cell lineages respectively. EAC originates from within the precursor intestinal type metaplasia, Barrett's esophagus (BE), which purportedly emerges from a stem cell pool resident at the gastro-esophageal junction. Underlying the development of both BE and EAC is a significant history of gastro-esophageal reflux disease (GERD) resulting in acidic exposure, inflammation and esophageal ulceration. Consequently, GERD directs the reparative or regenerating esophageal epithelium, through ill-defined mechanisms, towards a metaplastic intestine-like cellular lineage with genomic instabilities and increased cancer risk. In this seminar the continuous mechanistic links between GERD, associated inflammation, and metaplastic or oncogenic transformations of the lower esophagus will be explored through genomic studies, single cell sequencing of stem cell derived organoids of BE, functional genomic screening of BE and EAC cells and cancer genome atlas data of esophageal cancers. Collectively our studies will demonstrate that targetable immune-associated factors, intrinsic to epithelial cell responses to GERD, become constitutively activated during the esophageal metaplasia-dysplasia-adenocarcinoma sequence. Functional studies will highlight that these immune and GERD-associated signals support increased BE and EAC cell growth, elicit dynamic morphological changes conducive to BE stem cell colonisation of the lower esophagus and metastasis of EAC cells in later stages. Thus EAC is dependent upon inflammatory signals throughout its development with implications for therapeutic outcomes and responses to cancer immunotherapies.

Biography

Dr. Shane Duggan earned his PhD from Trinity College Dublin and continued with post-doctoral training at Imperial College London where he investigated the intestine-like nature of intestinal metaplasias, such as Barrett's esophagus, at a genomic level defining the GERD responsive transcription factors central to the maintenance of the intestine-like gene signature prevalent throughout esophageal adenocarcinogenesis.

Upon transition of the Kelleher Laboratory to UBC, Dr. Duggan has continued his post-doctoral studies utilising siRNA library screening strategies to discover novel therapeutic options for esophageal cancer patients, further defining the inflammatory and GERD-associated pathways underlying esophageal cancer development. In collaboration with international research groups, Dr. Duggan aims to capitalise upon a fundamental understanding of the interaction between immune-associated factors and the cellular plasticity underlying Barrett's esophagus, and adenocarcinoma, to transform clinical care and patient outcome.

Moleculr diagonostics for cancer pathology - state-of-the-art tools for the practicing pathologist

Pathologists have always played an important role in the cancer diagnosis, but this role has evolved immensely over the last decade, with the pathologic classification and ancillary testing of cancer tissues now playing a central role in oncologic treatment decisions. In particular, the array of tools for the molecular analysis of tumor tissues has expanded greatly, allowing for determination of genetic markers that are increasingly important in guiding targeted therapies. This recent evolution in "molecular pathology" has been driven by the development of numerous novel high-throughput technologies that screen for large panels of genetic alterations in a cost-efficient manner and in a timescale suitable for clinical utility. Although older methods such as PCR and FISH still play an important role in molecular diagnostics, these novel technologies such as next-generation sequencing and digital hybridization techniques have paved the way for an efficient approach to comprehensive characterize tumors for targetable mutations, including low-frequency but highly actionable mutations, as well as characterization of rare cancer types. In this talk, I will give an overview of the molecular diagnostic tools available to the current era of pathologists, including clinical vignettes to illustrate their role in cancer care.

Biography

Tony Ng is a consultant head and neck pathologist and sarcoma pathologist at Vancouver General Hospital and BC Cancer, and a clinical associate professor at the University of British Columbia. He is also an investigator at the Genetic Pathology Evaluation Center, and the head of the VGH Molecular Diagnostics laboratory. His main clinical and research interest is in molecular pathology, particular the development of molecular diagnostic assays for sarcomas and head and neck tumors using novel genomic technologies. He has developed a clinically validated diagnostic assay for sarcoma fusion gene detection based on the Nanostring platform, which is being actively used for cases across British Columbia and referral cases from various sites in Canada, and is developing a corresponding assay for the head and neck tumors.

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Cellular origin affects the phenotype of pancreatic ductal adenocarcinoma cells

The cellular origins of pancreatic ductal adenocarcinoma (PDAC) could affect both the genetic changes needed for tumor development, as well as the phenotype of the tumors themselves, potentially creating inter-tumoral heterogeneity. Recent studies have demonstrated that murine pancreatic ductal and acinar cells can both be cellular origins of PDAC. Using Sox9CreER;KrasG12D;Trp53f/f (Duct:Kras-p53) or Ptf1aCreER;KrasG12D;Trp53f/f (Acinar:Kras-p53) mice to specifically target ductal and acinar cells, respectively, we recently demonstrated that ductal cells initiate PanIN3 lesions that progress to PDAC earlier than those from acinar cells. The Duct:Kras-p53 mice also reach humane endpoint earlier than Acinar:Kras-p53 mice. We found that acinar-cell-derived PDAC maintained more of the characteristics of low-grade PanIN lesions, with higher Keratin 20, Mucin 5AC, and Alcian blue positivity than ductal-cell-derived PDAC. We have extended our analysis of the phenotype of acinar- and ductal-cell-derived PDAC by generating ductal- and acinar-cell-derived PDAC cell lines from tumors from Duct:Kras-p53 and Acinar:Kras-p53 mice, respectively. Consistent with previous data, RNA sequencing showed acinar-cell-derived PDAC cell lines maintain the expression of genes associated with a gastric phenotype, which is usually associated with low-grade PanIN lesions. In addition, we found that acinar-cell-derived PDAC cell lines proliferated slower than ductal-cell-derived PDAC cell lines and were more resistant to the cytotoxic chemotherapeutic agent, gemcitabine. Altogether our data suggest the cellular origin can affect the characteristics of PDAC. Future studies to identify markers or functional traits consistently associated with cellular origin across multiple genetic contexts are needed to determine the clinical importance of identifying cellular origin in human disease.

Biography

Dr. Kopp received her PhD from the University of Nebraska and undertook post-doctoral training at the University of California at San Diego. She is broadly interested in the processes involved in the development of the mature pancreatic structure, as well as the development of pancreatic ductal adenocarcinoma. Her group's current work in cancer is focused on the contribution of cellular origin to the heterogeneity in pancreatic cancer. More specfically, three different precancerous, or precursor, lesions are thought to precede the formation of pancreatic cancer. Whole genome sequencing of these lesions have provided insights into the mutations that are associated with the disease, but it is unclear what role these mutations have in that initiation and progression of each lesion, particularly the large cystic intraductal papillary mucinous neoplasia lesions (IPMN). Dr. Kopp's group recently developed a mouse model of IPMN and they are currently utilizing this unique model to examine the impact of these human gene mutations on IPMN development.

Data Science for Cancer and Chronic Disease Care

Big data and artificial intelligence methods have made fundamental changes to medical research and patient care in the past decade. In the first half of this talk, we will give an overview of the UBC Data Science Institute and some of the interdisciplinary medical projects sponsored by the Institute. In the second half, we will introduce some of the text analytics methods we have developed for identifying the changing needs of cancer patients. We will also briefly describe machine learning methods for copy number analysis, which is pivotal for recommending targeted cancer treatment.

Biography

Raymond's main research area for the past two decades is on data mining, with a specific focus on health informatics and text mining. He has published over 200 peer-reviewed publications on data clustering, outlier detection, OLAP processing, health informatics and text mining. He is the recipient of two best paper awards – from the 2001 ACM SIGKDD conference, the premier data mining conference in the world, and the 2005 ACM SIGMOD conference, one of the top database conferences worldwide. For the past decade, he has co-led several large-scale genomic projects funded by Genome Canada, Genome BC and industrial collaborators. Since the inception of the PROOF Centre of Excellence, which focuses on biomarker development for end-stage organ failures, he has held the position of the Chief Informatics Officer of the Centre. From 2009 to 2014, Dr. Ng was the associate director of the NSERC-funded strategic network on business intelligence.

Dr. Raymond Ng, PhD

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Posters • May 7

	Day 1			
#	Title	Name	Supervisor(s)	Email Address
1	The MicroRNA-17~92 Cluster in CD8 T Cell Function and Response to Lung Adenocarcinoma	Etienne Melese	Dr. Ninan Abraham	meleseet@mail.ubc.ca
2	Tumour-derived G-CSF induces immunosuppression and poses a barrier to immunotherapy	Israel Matos	Dr. Ken Harder	
3	Agents that decluster centrosomes are effective against aneuploid B-cell precursor ALL cells	Maria Guo	Dr. Poul Sorensen	mguo@bcchr.ca
4	How does Memory Retrieval Enhance versus Extinguish Emotional Memories?	Rachel Cederberg	Dr. Kevin Bennewith	
5	Interleukin-5 alters the lung B cell compartment and decreases pulmonary metastasis	Raunak Shrestha	Dr. Colin Collins	
6	Identifying Secreted Proteins that Promote Immune Evasion during Malignant Lung Transformation	Jennifer Luu	Dr. Will Lockwood	jluu@bccrc.ca
7	A link between microRNAs and mRNA translation elongation: The let7-eEF2K axis in MYC-driven pediatric tumors adaptation to nutrient deprivation	Alberto Delaidelli	Dr. Poul Sorensen	adelaidelli@bccrc.ca
8	Osteoclast promotes the outgrowth of dormant breast cancer cells under estrogen-deprivation condition	An Su	Dr. Erwei Song	48233908@qq.com
9	MYC activation through multiple mechanisms as a pathway of acquired TKI resistance in METex14-driven tumourigenesis	Daniel Lu	Dr. William Lockwood	
10	Loss of Ataxin-1-Like (ATXN1L) destabilizes the tumour suppressor Capicua (CIC) promoting dysregulation of the cell cycle	Derek Wong	Dr. Stephen Yip	derekwong90@gmail.com
11	Cystathionine γ-lyase (CTH) promotes oxidative stress adaptation and lung metastasis in Ewing sarcoma	Haifeng Zhang	Dr. Poul Sorensen	hzhang@bccrc.ca
12	The AHI-1-BCR-ABL-DNM2 Complex Mediates Mitochondrial Dynamics in Drug-Resistant BCR-ABL+ Cells	Ryan Yen	Dr. Xiaoyan Jiang	
13	Targeting the Core Autophagy Protein ATG4B in a Leukemic Xenotransplantation Mouse Model	Yueyang Shen	Dr. Xiaoyan Jiang	yshen@bccrc.ca
14	The Long Noncoding RNA Landscape of Neuroendocrine Prostate Cancer and its Clinical Implications	Rohan Ramnarine	Dr. Colin Collins	vramnarine@prostatecentre.com

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	Day 2			
#	Title	Name	Supervisor(s)	Email Address
20	Deep Learning-Based Approach to Predict Gene Regulating Effects of Small Molecules	Godwin Woo	Dr. Artem Cherkasov	godwinwoo@gmail.com
21	MOLI: Multi-Omics Late Integration with deep neural networks for drug response prediction	Hossein Sharifi Noghabi	Dr. Colin Collins	hsharifi@prostatecentre.com
22	Endogenous insulin contributes to pancreatic cancer development	Anni Zhang	Dr. James Johnson & Dr. Janel Kopp	annizubc@gmail.com
23	Lymphoma cancer internet patient information: a systematic evaluation of the quality of online resources for lymphoma patients and characterization of internet usage patterns	Donna Liao	Dr. Paris Ingledew	
24	Elucidating the molecular pathways that control daughter cell-size during cell division	Lin Mei	Dr. Poul Sorensen	lmei@bcchr.ca
25	Protein tyrosine phosphatase alpha (PTPα) as a potential target for triple-negative breast cancer cell invasion.	Lisa Decotret	Dr. Kevin Bennewith	ldecotret@bccrc.ca
26	Disruption of TACC2 epigenetically represses CDKN1A and confers sensitivity to CDK2 inhibitors in oesophageal squamous cell carcinoma	Tianliang Xia		xiatl@sysucc.org.cn
27	Modelling Initiation Events of Serous Ovarian Cancers with Organoid Cultures and Single Cell Sequencing	Joyce Zhang	Dr. David Huntsman	joycezhang1014@hotmail.com
28	Overcoming the Clinical Barriers of Protein Nanoparticles: Creating a Tunable and Reproducible Triggered Release System Using a Microfluidic System	Courtney van Ballegooie	Dr. Donald Yapp & Dr. Marcel Bally	cballegooie@bccrc.ca
29	Investigation of the molecular process of human B-lineage restriction.	Fangwu Wang	Dr. Connie Eaves	fwwang@bccrc.ca
30	Reticulon and CLIMP-63 control nanodomain organization of peripheral ER tubules	Guang Gao	Dr. Ivan Robert Nabi	gaoguang@mail.ubc.ca
31	Targeting Leukemic Stem Cells: Inhibition of Integrin-Linked Kinase in the Niche	Katharina Rothe	Dr. Xiaoyan Jiang	krothe@bccrc.ca
32	BRCA1 supports luminal features of mammary cells through suppressing NF-kB and maintaining cell-cell contacts	Zhengcheng He	Dr. Christopher Maxwell	zche@bcchr.ca
33	Global transcriptome profiling identifies a key miR-185-PAK6 axis that promotes survival of leukemic stem cells and drug-insensitive blasts in BCR-ABL+ human leukemia	Andrew Wu	Dr. Xiaoyan Jiang	awu@bccrc.ca

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