香港城市大學
City University of Hong Kong

CityU-NUS Joint RNA Symposium

06 November 2018 (Tuesday)

9:00am - 6:30pm

Venue: City University of Hong Kong

Li Dak Sum Yip Yio Chin Building [AC2]

3/F, Joseph Lee Hall LT3505

Keynote Speakers:

Anna Marie PYLE

Daniel G. TENEN

Invited Speakers:

TingFung CHAN
Polly Leilei CHEN
Melissa FULLWOOD
Sudhakar JHA
Dennis KAPPEI
Chun Kit KWOK
Jiguang WANG
Jack Chun Ming WONG

Organizers

Hong Kong RNA Club

NUS-CSI RNA Biology Centre

Participating Institutions

香港中文大學
The Chinese University of Hong Kong

香港大學
THE UNIVERSITY OF HONG KONG

香港科技大學
THE HONG KONG UNIVERSITY OF SCIENCE AND TECHNOLOGY

SCIENTIFIC PROGRAM

9:00am - 10:00am	REGISTRATION and RECEPTION
10:00am - 10:07am	OPENING REMARKS **Chun Kit KWOK** *City University of Hong Kong, Hong Kong*
10:07am - 10:15am	OPENING REMARKS **Daniel G. TENEN** *Cancer Science Institute of Singapore, NUS, Singapore*
	SESSION I (Chair: Chun Kit KWOK)
10:15am - 11:15am	**KEYNOTE TALK 1** **Anna Marie PYLE** *Yale University, USA* Harnessing the RIG-I innate immune sensor to control antiviral and antitumor responses
11:15am - 11:45pm	**Jiguang WANG** *Hong Kong University of Science and Technology, Hong Kong* Computational study noncoding transcriptome reveals the role of noncoding regions in cancer
11:45pm - 12:15pm	**Polly Leilei CHEN** *Cancer Science Institute of Singapore, NUS, Singapore* Understanding the mechanisms for fine-tuning A-to-I RNA editing in cancer
12:15pm - 1:45pm	GROUP PHOTO AND LUNCH
	SESSION II (Chair: Polly LeiLei CHEN)
1:45pm - 2:15pm	**Chun Kit KWOK** *City University of Hong Kong, Hong Kong* RNA structures, interactions and G-quadruplexes
2:15pm - 2:45pm	**Dennis KAPPEI** *Cancer Science Institute of Singapore, NUS, Singapore* ZBTB48 is both a vertebrate telomere-binding protein and a transcriptional activator
2:45pm - 3:15pm	**Jack Chun Ming WONG** *The University of Hong Kong, Hong Kong* *Deregulation of RNA methylation in liver cancer*
3:15pm - 3:45pm	**Melissa FULLWOOD** *Cancer Science Institute of Singapore, NUS, Singapore* Machine learning to understand patient-specific variations in chromatin interactions involved in transcription regulation in cancer
3:45pm - 4:15pm	COFFEE/TEA BREAK
	SESSION III (Chair: Minh LE)
4:15pm - 4:45pm	**TingFung CHAN** *Chinese University of Hong Kong, Hong Kong* The usefulness of junk: noise modelling improves transcriptome-wide identification of RNA G-quadruplexes
4:45pm - 5:15pm	**Sudhakar JHA** *Cancer Science Institute of Singapore, NUS, Singapore* TIP60 keeps the endogenous oncogenic demons repressed
5:15pm - 6:15pm	**KEYNOTE TALK 2** **Daniel G. TENEN** *Cancer Science Institute of Singapore, NUS, Singapore* KATs, SPEARS, HSCs, and weird RNA biology
6:15pm - 6:30pm	CLOSING REMARKS, SOUVENIRS, AND SPEAKERS' PHOTO **Minh LE** *City University of Hong Kong, Hong Kong*
6:30pm - 8:00pm	DINNER FOR SPEAKERS AND ORGANIZING CHAIRS

KEYNOTE TALK 1

Prof Anna Marie PYLE
Sterling Professor of Department of Molecular, Cellular & Developmental Biology
Professor of Chemistry
HHMI Investigator
President of RNA Society
Yale University

Harnessing the RIG-I innate immune sensor to control antiviral and antitumor responses

Abstract:

Host cell invasion in vertebrates is detected by a set of proteins that are known as pattern recognition receptors (PRRs). Each of these endogenous biosensors are designed to activate the innate immune defense machinery upon recognition of a cognate "pathogen associated molecular pattern" (PAMP), which is usually a peptide, carbohydrate or nucleic acid moiety that is characteristic of the invading organism. The RIG-I like receptor (RLR) family of PRRs includes three proteins (RIG-I, MDA5, and LGP2) that are responsible for sensing infection by intracellular pathogenic RNA, such as that produced by common RNA viruses (i.e. Dengue virus, influenza, and Ebola). All RLR proteins are built around a central ATPase core that is homologous to that found in canonical Superfamily 2 (SF2) RNA helicases, and which has been modified through the addition of novel accessory domains that recognize duplex RNA and 5'-triphosphate moieties. Using a combination of cell biology, structural biology and animal studies, we have attempted to understand the structural basis for pathogen-specific dsRNA binding and ATPase activation in RLRs, and the differential RNA recognition by RLR family members. Through this work we have identified a structurally-defined set of small, synthetic RNA molecules that are exceptionally active stimulators of the RIG-I response. We are now leveraging this information to design a potent new class of anticancer therapies, antivirals and vaccine adjuvants.

Biosketch:

Prof. Anna Pyle obtained her B.A. in Chemistry (1985) from the Princeton University in New Jersey. She completed her PhD (1990) in Columbia University, New York under the supervision of Professor Jacqueline Barton. She then work as Jane Coffin Childs Fellow in Prof. Thomas R. Cech's group at the University of Colorado from 1990-1992. In 2018, Dr. Pyle became the Sterling Professor of Molecular, Cellular and Developmental Biology and Professor of Chemistry at the Yale University, and she has been HHMI Investigator since 1997. Over the years, she has received numerous pretigious awards, including Endowed Chair: The William Edward Gilbert Professorship, Yale University in 2005, Fellow of the American Association for the Advancement of Science in 2007, and more recently Blavatnik Fund for Innovation Award, New Haven in 2017 and Jerry A. Weisbach Memorial Lecture, Rockefeller University in 2018 and many more. She has published more than 170 papers, including Nature, Science, Cells. She currently serves as the editorial board for the Journal of Molecular Biology, board of reviewing editor for elife, and editor for Methods in Enzymology.

Dr. Pyle's laboratory studies RNA structure and RNA recognition by protein enzymes. They use a combination of experimental biochemistry and crystallography to study the architectural features of large RNA molecules, such as self-splicing introns, noncoding RNAs, and viral genomes. This is accompanied by complementary work on RNA-dependent ATPase enzymes that bind and remodel RNA structures, with an emphasis on proteins that are involved in viral replication and host innate immune response.

KEYNOTE TALK 2

Prof Daniel G. TENEN
Director, Cancer Science Institute of Singapore
Distinguished Professor in Medicine
National University of Singapore

KATs, SPEARS, HSCs, and weird RNA biology

Abstract:

Hematopoietic stem cells (HSC) have the potential to replenish the blood system for the lifetime of the organism. Their two defining properties, self-renewal and differentiation, are tightly regulated by the epigenetic machineries. Here, using conditional gene knockout models, we demonstrate a critical requirement of lysine acetyltransferase 5 (Kat5, also known as Tip60) for murine HSC maintenance both in the embryonic and adult stages, which depends on its acetyltransferase activity. Genome-wide chromatin and transcriptome profiling revealed that Tip60 co-localizes with c-Myc and that Tip60 deletion suppress the expression of Myc target genes, which are associated with critical biological processes for HSC maintenance, cell-cycle and DNA repair. Notably, acetylated H2A.Z (acH2A.Z) was enriched at the Tip60-bound active chromatin and Tip60 deletion induced a robust reduction in the acH2A.Z / H2A.Z ratio. These results uncover a critical epigenetic regulatory layer for HSC maintenance through Tip60 dependent H2A.Z acetylation to activate Myc target genes.

The mechanisms by which epigenetic modifications, such as acetylated H2A.Z, are established in gene regulatory regions of active genes remain elusive. Independently, we demonstrate that the establishment of a major epigenetic mark, the acetylated form of the replacement histone H2A.Z, is regulated by cell cycle-specific long noncoding RNAs encoded in regions adjacent to the promoters of active genes. These transcripts, termed SPEARs (S Phase EArly RNAs), are induced in early Sphase: their expression precedes that of the downstream genes on which they exert theirregulatory action. SPEARs set the stage for the modification and deposition of the acetylatedform of histone H2A.Z by bringing together the replacement histone and the histone acetyl transferase Tip60. This widespread bimodal interaction constitutes a novel RNA-mediated mechanism for the establishment of epigenetic marks and cell-specific epigenetic profiles, providing a unifying mechanistic explanation for the accuracy and persistence of epigenetic marks on chromatin.

Biosketch:

Over the past 34 years my laboratory has made seminal contributions to understanding the role of gene regulation in cell differentiation and the role of disruption of these pathways in leukemia, lung cancer, and liver cancer. I have been PI on many individual US NIH R01 grants and Program grants. Since 2008 I have been Director of the Cancer Science Institute of Singapore. My recent studies have focused on noncoding RNAs, and include published findings on antisense RNAs, RNA editing, and noncoding RNAs in gene regulation, methylation, and cancer. Our most recent studies demonstrated that RNA can regulate DNA methylation, and that RNA can be utilized to induce demethylation in a gene-specific manner.

Dr Jiguang WANG
Assistant Professor in Division of Life Science
Hong Kong University of Science and Technology

Computational study noncoding transcriptome reveals the role of noncoding regions in cancer

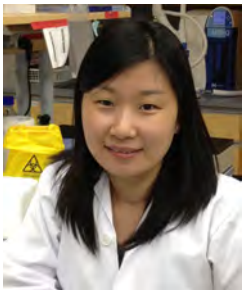
Abstract:

The human genome project has shown that only a small fraction (<2%) of human genome can be transcribed into mRNA that is further translated into protein, and the vast majority of the mammalian genome might express non-coding RNA (ncRNA). Although a number of long non-coding RNAs (lncRNAs) have been recently shown to play significant roles in the regulation of gene expression or protein activity in critical signaling pathways, the total number of ncRNAs and the fraction of functional ncRNAs within mammalian genome are still mysteries. To reveal the landscape of ncRNA expression and specifically, to capture the expression of transient RNAs, we have developed an RNA-seq Analysis pipeline of Transcriptome Reconstruction and Annotation to Identify Novel non-coding RNAs from exosome deficient cells. Using this approach we have identified a vast number of novel ncRNAs, including short TSS RNA (xTSSRNA), long non-coding RNAs (xlncRNAs), enhancer RNAs (xeRNAs), and others. We further applied this approach in studying the role of noncoding RNA in Gastric cancer. We found lncRNA-based classification can cluster gastric cancer into clinical-relevant subtypes.

Biosketch:

Prof Jiguang Wang joined HKUST in 2016, having previously spent five years as a research scientist at Columbia University, where he focused on studying cancer genomics and developed a computational method for tracing the evolution of chronic lymphocytic leukemia. In 2015, he was named as an Irving Institute Precision Medicine Fellow. He received his Ph.D. in Applied Mathematics from the Chinese Academy of Sciences. He has the substantial contribution to the discovery of *MET*ex14 (currently in Press in *Cell*) and *LTBP4* mutations (published by *Nature Genetics*) in brain tumors, and the elucidation of xTSS-RNA (published by *Nature*) and Exotome (published by *Cell*).

Dr Polly LeiLei CHEN
Principal Investigator, Cancer Science Institute of Singapore
NUS President Assistant Professor, Department of Anatomy
National University of Singapore



Understanding the Mechanisms for Fine-Tuning A-to-I RNA Editing in Cancer

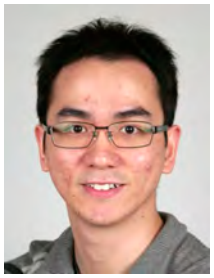
Abstract:

Conventionally, cancer is driven by a clonal accumulation of somatic mutations, referred to as "driver mutations", conferring a selective growth advantage to cancer cells. RNA editing, is an epigenetic mechanism, introduces changes in the RNA sequences encoded by the genome, contributing to "editing/epigenetic mutations". In humans, the most frequent type of editing is the conversion of adenosine to inosine (A-to-I), which is catalyzed by ADAR (Adenosine Deaminase Acting on RNA) proteins, ADAR1 and ADAR2. Inosine (I) essentially mimics guanosine (G), therefore ADAR proteins actually introduce a virtual A-to-G substitution in transcripts. Such changes can lead to specific amino acid substitutions, alternative splicing, altered microRNA seeds or targets, or changes in transcript localization, expression and degradation. Up until the past 5 years we and others highlighted the role of RNA editing dysregulation in cancer development, changes in the information are being investigated almost exclusively at the DNA level. In this talk, I will discuss our recent findings on the mechanisms for fine-tuning A-to-I RNA editing in cancer.

Biosketch:

Dr Polly Chen got her Bachelor of Medicine in 2002 from Medical school, Jiangsu University, China, followed by 2-year specialized training in Obstetrics & Gynecology. She completed her PhD in Cancer Genetics in Prof. Xin-Yuan Guan's laboratory at the University of Hong Kong in 2010. After 2-year postdoctoral training in the same lab, she joined Cancer Science Institute of Singapore as a Special fellow in 2012, and was promoted to a Principle Investigator and joined the Department of Anatomy, NUS, as an Assistant professor in 2014. Dr. Chen's research has centered on transcriptome alterations in human cancers and in particular on understanding how A-to-I RNA editing contributes to cancer initiation and progression. She currently places focus on the regulators of A-to-I RNA editing and the crosstalk between RNA editing and other RNA processes (e.g. alternative splicing) in cancer.

Dr Chun Kit KWOK
Assistant Professor in Department of Chemistry
City University of Hong Kong



RNA structures, interactions and G-quadruplexes

Abstract:

RNA adopts diverse structural motifs, such as stem-loop, pseudoknot, G-quadruplex, and is capable of long-range interactions, contributing to its basic biological functions. These structural elements can form through cis (intra-molecular) interactions within the same RNA molecule, or through trans (inter-molecular) interactions with other biomolecules such as RNA, DNA and proteins, to regulate fundamental cellular processes. Identifying RNA structures and interactions that are involved in gene regulation and function is thus critical for the elucidation of the underlying biochemical mechanisms. Over the past 5 years, we and others have witnessed the dawn of the *in vivo* RNA structurome and interactome. In this talk, I will present our lab's recent efforts to decipher the *in vivo* RNA structures and interactions in Zika virus. In addition, the formation and role of RNA G-quadurplexes in human mature microRNA will be discussed.

Biosketch:

Dr. Kit Kwok obtained his B.Sc. in Chemistry (2009) from the Chinese University of Hong Kong, after completing an exchange program at University of California, Los Angeles in 2007-2008. He completed his PhD in Pennsylvania State University (2014), mentored by Professor Philip C. Bevilacqua and Professor Sarah M. Assmann. In Apr 2014, Dr. Kwok commenced on a postdoctoral fellowship as a Croucher Fellow in University of Cambridge under Professor Sir Shankar Balasubramanian FRS FMedSci. In Oct 2016, Dr. Kwok's joined the faculty team at the City University of Hong Kong as an Assistant professor. Dr. Kwok's current research focus is to explore the role of RNA structures and interactions in biology, especially the functions of G-quadruplex structures in the human transcriptome and their relevance to health and diseases. Dr. Kwok has recently written a review "Detecting RNA G-quadruplexes (rG4s) in the Transcriptome." that will be featured as a book chapter in the upcoming book "RNA Worlds: New tools for deep exploration, 5th Edition".

To cultivate a stimulating learning environment for students and to promote RNA sciences in Hong Kong, Dr. Kwok, together with Dr. Minh Le, founded the Hong Kong RNA Club in Aug 2017. (http://www.kitkwok.com/hk-rna-club.html)

Dr Dennis KAPPEI
Principal Investigator, Cancer Science Institute of Singapore
National University of Singapore

ZBTB48 is both a vertebrate telomere-binding protein and a transcriptional activator

Abstract

Telomeres constitute the ends of linear chromosomes and together with the shelterin complex form a structure essential for genome maintenance and stability. Due to the end replication problem and active end processing, telomeres shorten with every cell division, ultimately leading to cellular senescence. Cancer cells would eventually share this fate. However, they (re-)activate mechanisms to defy telomere shortening that are in part orchestrated by telomere-binding proteins. In addition to the constitutive binding of the shelterin complex, other direct, yet more transient interactions are mediated by the CST complex and HOT1, while subtelomeric variant repeats are recognized by NR2C/F transcription factors. Recently, we systematically investigated telomere-binding proteins in 16 vertebrate species using label-free quantitative mass-spectrometry based proteomics, creating a phylointeractomics map of telomeres and identified ZBTB48 as a novel telomere-associated factor throughout the vertebrate lineage. Here, we show that ZBTB48 binds directly both to telomeric as well as to subtelomeric variant repeat sequences *in vitro* and *in vivo*. ZBTB48 is found at telomeres of human cancer cells regardless of the mode of telomere maintenance and it acts as a negative regulator of telomere length. In addition to its telomeric function, we demonstrate through a combination of RNAseq, ChIPseq and quantitative label-free expression proteomics experiments that ZBTB48 acts as a transcriptional activator on a small set of target genes, including mitochondrial fission process 1 (MTFP1). This discovery places ZBTB48 at the interface of telomere length regulation, transcriptional control and mitochondrial metabolism. We are currently exploring the mechanism behind ZBTB48's role at telomeres and as a transcriptional activator through various mass spectrometry-based interactomics approaches.

Biosketch:

Dr Dennis Kappei is a Principal Investigator and the Head of the Quantitative Proteomics Core at the Cancer Science Institute of Singapore (CSI). He obtained his MS degree from Ecole Normale Supérieure and University Paris VI and pursued his graduate studies at the Max Planck Institute of Molecular Cell Biology and Genetics under the roof of the Dresden International Graduate School for Biomedicine and Bioengineering. For his graduate work he was awarded the Georg Helm Prize by TU Dresden. Dr. Kappei currently also serves as a member of the Steering Committee for the International Biology Olympiad.

Dr Jack Chun-Ming WONG
Associate Professor in the Department of Pathology
University of Hong Kong

Deregulation of RNA methylation in liver cancer

Abstract:

Traditionally, epigenetic regulation refers to diverse and reversible chemical modifications on DNA and histones, which regulate gene expression in a way independent to genome sequences. Besides DNA and histones, cellular RNAs (mRNA, tRNA, snRNA etc.) also carry hundreds of distinct post-transcriptional modifications. These modifications are thought to moderate RNA structures, functions, and stability. Some of these post-transcriptional RNA modifications are reversible and dynamic controlled, indicating that they might have potential regulatory functions similar to modifications on DNA and histones. In this regard, investigating the landscapes and functions of these reversible RNA modifications is now emerging as a new frontier of research, known as “RNA epigenetics” or “epi-transcriptomics”. Given the fact that epigenetic alterations contribute immensely to carcinogenesis, we reasoned that RNA modifications might also represent a new layer of epigenetic alteration in cancer progression. In this talk, I will discuss our recent findings on delineating the implications of RNA modification deregulations in liver cancer.

Biosketch:

Dr. Jack Chun-Ming Wong is an Associate Professor in the Department of Pathology, and a Principal Investigator of the State Key Laboratory for Liver Research, the University of Hong Kong. Dr. Wong obtained his PhD degree from the University of Hong Kong and conducted his postdoctoral researches with Prof Irene Ng in studying liver cancer. His research focuses on genetic alterations and epigenetic deregulations in liver cancer.

Dr Melissa FULLWOOD
Principal Investigator, Cancer Science Institute of Singapore
National University of Singapore
Nanyang Assistant Professor, School of Biological Sciences
Nanyang Technological University, Singapore

Machine learning to understand patient-specific variations in chromatin interactions involved in transcription regulation in cancer

Abstract:

Chromatin interactions are two or more regions of the genome that come together in close proximity and can regulate gene transcription. Superenhancers are long stretches of enhancers that regulate key oncogenes in cancer. We found that superenhancers are highly associated with chromatin interactions. CRISPR excision of superenhancers results in reduction of gene expression of genes to which the superenhancers loop by chromatin interactions. Analysis of superenhancer-promoter contacts in individual chronic myelogeneous leukemia patient samples by Oxford Biodynamics' EpiSwitch method showed that certain interactions are similar between all patients, whereas chromatin interactions show individual variation, prompting us to develop methods to interrogate large datasets of patient samples. We developed a machine learning-based prediction method to predict chromatin interactions based on open chromatin datasets. We then re-analyzed a published set of open chromatin datasets in Chronic Lymphocytic Leukemia (CLL), which revealed that there exists individual heterogeneity in chromatin interactions, corroborating our previous observations of chromatin interaction heterogeneity in the chronic myelogenous leukemia patient samples. Study of the predicted chromatin interactions between two different CLL subtypes revealed the existence of systematic differences between chromatin interactions in the two cancer subtypes. Using the predicted chromatin interactions, we also showed that differential expression of important CLL prognostic markers may be associated with changes in distal regulatory regions. Our method may allow for the design of chromatin interaction-based biomarkers for diseases such as cancer.

Biosketch:

Dr Melissa J. Fullwood is a Junior Principal Investigator in the Cancer Science Institute with a joint appointment as a Nanyang Assistant Professor at School of Biological Sciences in Nanyang Technological University. She completed her undergraduate degree in Biological Sciences at Stanford University and her PhD with the National University of Singapore Graduate School for Integrative Sciences and Engineering (NGS) and the Genome Institute of Singapore. She worked as a Lee Kuan Yew Post-doctoral Fellow in the Duke-NUS Graduate Medical School. She has 24 publications, which have been cited more than a thousand times, and is a co-inventor on several patents. She is a recipient of the Agency for Science, Technology and Research (A*STAR) National Science Scholarships, the L'Oreal-UNESCO for Women in Science National Fellowships in Singapore, and was the international winner of the GE and Science prize in 2010. She is a National Research Foundation (NRF) fellow. Her research examines 3D genome organization and RNA biology in cancer.

Dr. Ting Fung CHAN
Associate Professor in the School of Life Sciences
Chinese University of Hong Kong

The usefulness of junk: noise modelling improves transcriptome-wide identification of RNA G-quadruplexes

Abstract:

Noise exits in all data, and RNA-seq data are widely known to have intrinsic noise originated from the underlying chemistry. However, since they usually do not critically affect conventional RNA-seq analyses such as differential expression and transcript isoform discovery they are simply being tolerated. In the past decade, many sequencing-based methods have been developed to interrogate various RNA elements in the transcriptome, and these intrinsic noises have suddenly become non-negligible at single-nucleotide resolution, when precision in locating an RNA element along a transcribed region is frequently needed. And even more importantly, unlike biological or technical variations, these noises are systematic cannot be effectively removed by increasing the number of replicates.

In this talk, I will introduce how noise-modelling could be applied to rG4-seq, and how understanding platform-specific noise could be used to improve transcriptome-wide identification of RNA G-quadruplexes.

Biosketch:

Dr. Ting-Fung Chan studied in the United States in his early years and received his Bachelor degree in computer sciences from the University of Wisconsin – Madison. He then pursued postgraduate training in genomics and obtained his PhD at the Washington University School of Medicine. He received postdoctoral training in genomics at the University of California – San Francisco with an NIH fellowship. He is currently an Associate Professor at the School of Life Sciences, CUHK. And because of his cross-disciplinary background, he is affiliated with many research units, including the Hong Kong Bioinformatics Centre (Co-director), State Key Laboratory of Agrobiotechnology, CUHK, the Hong Kong Institute of Diabetes and Obesity, the Gerald Choa Neuroscience Centre, and the CUHK-BGI Innovation Institute of Trans-omics. His current research interests are on RNomics and bioinformatics, and their broader importance across various fields of study in biology and medicine.

Dr Sudhakar JHA
Principal Investigator, Cancer Science Institute of Singapore
Assistant Professor in Department of Biochemistry, YLL School of Medicine
National University of Singapore

TIP60 keeps the endogenous oncogenic demons repressed

Abstract:

TIP60 is a lysine acetyltransferase which belongs to the MYST family of acetyltransferases and is known to be a haplo-insufficient tumor suppressor. TIP60 downregulation is an early event in tumorigenesis which has been observed in several cancer types including breast and colorectal cancers. Colorectal cancers are characterized by inflammation wherein inflammatory bowel disease greatly increases the risk for colorectal cancer. In this symposium, I will discuss the role of TIP60 in silencing of endogenous retroviral elements (ERVs). We have identified a unique mechanism of ERV regulation in cancer cells mediated by TIP60 and BRD4 through regulation of Histone H3K9 trimethylation. I will also discuss our efforts to exploit this pathway to sensitize colorectal cancer to reverse transcriptase inhibitors.

Biosketch:

Dr Sudhakar Jha is a Principal Investigator at Cancer Science Institute of Singapore, and Assistant Professor in Department of Biochemistry, YLL School of Medicine at the National University of Singapore. His group is interested in understanding the regulation of chromatin remodeling complexes and their role in cancer prevention and intervention (Mol Cell 2009, 34: 521-533). Dr. Jha's group focuses on identifying the role of TIP60, a lysine acetyltransferase in transcription (J Mol Cell Biol 2016, 85: 384–399) and DNA damage response pathway (Mol Cell Biol 2008, 28: 2690-2700 and Mol Cell Biol 2013, 33: 1164-74). Among various regulators of TIP60, Dr. Jha's group has discovered human papillomavirus (HPV) E6 and Adenovirus (AdV) oncogenes to destabilize TIP60 (Mol Cell 2010, 38: 700-711, Oncogene 2013, 32: 5017-25 and Oncogene 2016, 35:2062-74). In addition, his group has also identified a new cellular regulator of TIP60 and have demonstrated its role and significance in epithelial-mesenchymal transition and breast cancer progression (Oncotarget 2015, 6:41290-306 and J Mol Cell Biol 2016, 85: 384–399).

How to get to Joseph Lee Hall -

Location: 3/F, Li Dak Sum Yip Yio Chin Academic Building

Arrived at Pedestrian Subway by MTR

1. When you get off the MTR, look for Festival Walk exit. In Festival Walk, on Level LG1 (next to Cova), there is an escalator which lead to a Pedestrian Subway to CityU campus

2.Please walk to Yeung Kin Man Academic Building

3. Take the escalator next to bookshop to 4/F and walk straightly.

4. Then get out from the building at the doors

5. Walk towards to Bank of China (HK) Complex and go straightly.

6. Walk up the staircases

7. You will arrive the road lead to Li Dak Sum Yip Yio Chin Academic Building (LI)

8. Turn left to find lift lobby of Li Dak Sum Yip Yio Chin Academic Building (LI) to 3/F

9. Turn left when you come out from lift at 3/F

10. Open the exit door and find the Joseph Lee Hall at far end of your left hand side

Arrived at University Circle by Taxi or by Car

1. When you drop off at the University Circle, go long the covered walkway to Bank of China (HK) Complex

2. Follow the above instructions pt. 5 to pt. 10 to the Joseph Lee Hall

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