

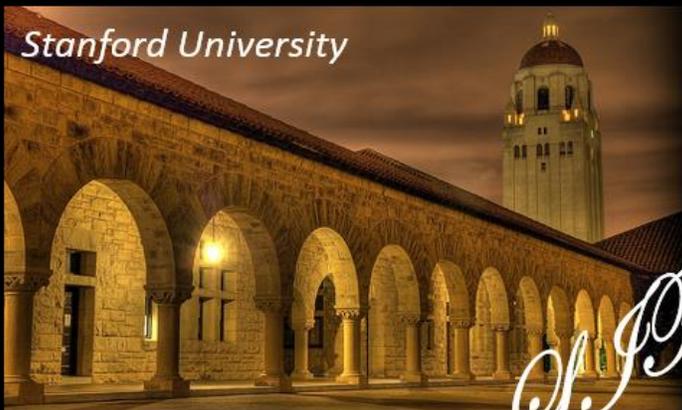
KOLIS

Korean Life Scientists in the Bay Area

KOLIS Spring Conference 2015

일시: 2015년 5월 09일

장소: Genentech Hall, Mission Bay, UC San Francisco



*2015
KOLIS
CONFERENCE*

Programs

10:30-11:00

Registration

11:00-11:05

Opening Remarks

11:05-11:45

Luke P. Lee, Ph.D.

*Arnold and Barbara Silverman Distinguished Professor
Dep. of Bioengineering, Electrical Engineering & Computer Science, and
Biophysics Program, UC Berkeley*

11:45-12:25

Lydia Sohn, Ph.D.

Associate Professor, Dept. of Mechanical Engineering, UC Berkeley

12:25-13:10

Lunch

13:10-13:50

Jin Hyung Lee, Ph.D.

*Assistant Professor, Neurology & Neurological Sciences and Bioengineering,
Stanford University*

13:50-14:30

Il-Jin Kim, Ph.D.

Assistant Professor, Director of Applied Genomics, UCSF

14:30-14:45

Coffee Break

**Symposia I
(Genentech Hall)**

**Symposia II
(N-114)**

14:45-15:15

Bomsoo Cho, Ph.D.

Stanford University

Gunsoo Kim, Ph.D.

UCSF

15:15-15:45

Sekyung Oh, Ph.D.

Stanford University

Taeho Kim, Ph.D.

UC Davis

15:45-16:00

Coffee Break

16:00-16:30

Anna Joe, Ph.D.

UC Davis

Jaecheol Lee, Ph.D.

Stanford University

16:30-17:00

Minwoo Lee, Ph.D.

UCSF

Jongmin Kim, Ph.D.

Stanford University

17:00-17:15

Messages from our sponsors

17:15-17:30

Raffle & Closing Remarks

17:30-

Dinner

KOLIS SPRING CONFERENCE 2015

PLENARY TALKS (Genentech Hall)

Plenary Talk 1

Precision Medicine in the Palm of Your Hand

(Time) 11:05~11:45



Luke P. Lee
UC Berkeley

Abstract

In this talk, I will present how to see the world's health care crisis in a grain of *iSAND* (integrative Science, Arts, Nanomedicine, and Digital technology), and find solutions for preventive precision medicine and healthy environment. Since the future of healthcare is in the palm of our hands, a few examples of creative healthcare innovations will be discussed along with the vision of digital healthcare in both developing and developed countries: *iMDx* (integrated *Molecular Diagnostic* systems) for personalized medicine, *iMAPs* (integrated *Microphysiological Analysis Platforms*) for regenerative medicine and *in vitro* organogenesis, and *iHIT* (integrative *Healthcare Information Technology*) for the prevention and treatment of infectious and neurodegenerative diseases.

Plenary Talk 2

Node-pore sensing: A label-free platform to screen single cells for their phenotypic profile

(Time) 11:45~12:25



Lydia Sohn
UC Berkeley

Abstract

Flow cytometry is one of the cornerstones of biomedical research and clinical diagnostics. With its ability to screen individual cells for multiple protein epitopes simultaneously and subsequently identify sub-populations of cells, flow cytometry has had a profound impact in a broad range of areas including immunology, cancer, and regenerative medicine. While the current state-of-the-art is 18 parameters, flow cytometry is often “practically” limited to 6-10 parameters, as emission spectral overlap is of concern and highly complex analysis to decouple such overlap is needed. We have developed a *rapid, label-free* method of screening cells for their phenotypic profile, which we call Node-Pore Sensing (NPS). NPS involves measuring the modulated current pulse caused by a cell transiting a microchannel that has been segmented by a series of inserted nodes. When the segments between the nodes are functionalized with different antibodies corresponding to distinct cell-surface antigens, cells whose antigens interact specifically with the functionalized antibodies in a particular segment will travel more slowly through that section of the channel than that through the isotype-control. Surface-marker identification is accomplished by comparing transit times within the modulated pulse. I will show the versatility of NPS and discuss how we are using this method in a pilot clinical study to phenotype acute myeloid leukemia patient samples.

Plenary Talk 3

Optogenetic fMRI (ofMRI): *In Vivo* Visualization and Control of Neural Circuits

(Time) 13:10~13:50



Jin Hyung Lee
Stanford University

Abstract

Understanding the functional interactions of the whole brain has been a long sought-after goal of neuroscientists. However, due to the widespread and highly interconnected nature of brain circuits, the dynamic relationship between neuronal networks often remains elusive. The recent development of optogenetic functional magnetic resonance imaging (ofMRI) provides a key technological advancement in addressing this problem. Using ofMRI, it is possible to observe whole-brain level network activity that results from modulating with millisecond-timescale resolution the activity of genetically, spatially, and topologically defined cell populations. The significance of ofMRI lies in its ability to map global patterns of brain activity that result from the precise control of distinct neuronal populations. No other method exists to bridge this gap between whole-brain dynamics and the activity of genetically, spatially, and topologically defined neurons. Advances in the molecular toolbox of optogenetics, as well as improvements in imaging technology, both stand to benefit ofMRI and bring it closer to its full potential. In particular, the integration of ultra-fast data acquisition, high SNR, and combinatorial optogenetics will enable powerful systems of closed-loop ofMRI to modulate and visualize brain activity in real-time. Further research into the nature of the ofMRI BOLD response may also make it possible to extract detailed information about the neural activity underlying a given signal. Finally, the application of ofMRI to translational research has the potential to fundamentally transform how therapies are designed for neurological disorders. Therefore, ofMRI is anticipated to play an important role in the future dissection and treatment of network-level brain circuits. In this talk, the ofMRI technology will be introduced with advanced approaches to bring it to its full potential, ending with some examples of dissecting neurological disease circuits and stem cell integration visualization utilizing ofMRI.

Plenary Talk 4

Bench to Bedside and Beyond

(Time) 13:50~14:30



Il-Jin Kim
UC San Francisco

Abstract

Dr. Il-Jin Kim is a Principal Investigator and Director of Applied Genomics in the UCSF Thoracic Oncology Program. His work focuses on the identification of novel diagnostic and therapeutic markers in lung cancer and mesothelioma. Dr. Kim investigates human cancers using state-of-the-art technologies including next-generation sequencing (NGS) and system genetics, Dr. Kim has developed several innovative microarrays (RET, beta-catenin, K-ras, and BRAF) and pioneered new methods (including NGS) of high-throughput mutation screening for which he holds numerous patents.

Dr. Kim graduated the College of Veterinary Medicine, Seoul National University with a B.S. and D.V.M. (1999), and Ph.D. (2003) in Tumor Biology from the College of Medicine at Seoul National University. Dr. Il-Jin Kim served as a group leader of the Korean Hereditary Tumor Registry from 2003-2006, an organization of which he has been a member since 1999. As group leader, he led a project that screened approximately 1,000 patients from 400 families, looking at 12 inherited cancer syndromes. He has three times received awards for development of genetic assays by the AACR (American Association for Cancer Research). In 2007, Dr. Kim came to UCSF as a post-doctoral fellow and joined the UCSF faculty as an Assistant Professor in the Department of Surgery (2010).

The Kim lab studies genome-wide gene expression, DNA copy number, mutation, fusions, and any other genetic changes in lung cancer. His lab has identified several novel molecular targets in normal and matched adenocarcinoma tissue, and is now working on functional and biological validation of these markers. This research is being used to develop novel therapeutic drugs and diagnostic assays for lung and other types of human cancers.

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SYMPOSIA I (Genentech Hall)

Clustering and Negative Feedback by Endocytosis in Planar Cell Polarity Signaling is Modulated by Ubiquitylation of Prickle

(Time) 14:45–15:15

Bomsoo Cho

Jeff Axelrod Lab, Dept. of Pathology, Stanford



Abstract

The core components of the planar cell polarity (PCP) signaling system, including both transmembrane and peripheral membrane associated proteins, form asymmetric complexes that bridge apical intercellular junctions. While these can assemble in either orientation, coordinated cell polarization requires the enrichment of complexes of a given orientation at specific junctions. This might occur by both positive and negative feedback between oppositely oriented complexes, and requires the peripheral membrane associated PCP components. However, the molecular mechanisms underlying feedback are not understood. We find that the E3 ubiquitin ligase complex Cullin1(Cul1)/SkpA/ Supernumerary limbs(Slimb) regulates the stability of one of the peripheral membrane components, Prickle (Pk). Excess Pk disrupts PCP feedback and prevents asymmetry. We show that Pk participates in negative feedback by mediating internalization of PCP complexes containing the transmembrane components Van Gogh (Vang) and Flamingo (Fmi), and that internalization is activated by oppositely oriented complexes within clusters. Pk also participates in positive feedback through an unknown mechanism promoting clustering. Our results therefore identify a molecular mechanism underlying generation of asymmetry in PCP signaling.

Altered translation response to stress by medulloblastoma-associated DDX3X mutation

(Time) 15:15–15:45

Sekyung Oh

Yoon-Jae Cho Lab, Dept. of Neurology and Neurological Sciences, Stanford



Abstract

DDX3X encodes an ATP-dependent RNA helicase (DDX3) that has been identified as one of the most commonly mutated genes in medulloblastoma, a highly aggressive cerebellar tumor affecting both children and adults. Here, we show that disease-related alterations in DDX3 diminish its helicase and ATPase activities and result in deregulated translation in primary medulloblastomas. By systematically indexing the proteins and RNAs that DDX3 associates with and, in parallel, assessing the global influence of DDX3 on translation, we unveil an extensive collaboration between DDX3 and the translation initiation machinery. This includes direct binding of DDX3 to multiple components of the translation initiation complex, specific sites on the 18S rRNA, 5' leaders of mRNAs, and GC-rich sequences positioned upstream of ribosome-dense regions. Furthermore, we demonstrate how, under stress, these physical interactions promote the general repression of translation and how disruption of DDX3 catalytic activity via a cancer-related mutation blunts the translation response to stress.

Activated Type 2 Innate Lymphoid Cells Regulate Beige Fat Biogenesis

(Time) 16:00–16:30

Minwoo Lee

Dept. of M_Cardiovascular Research Inst, UCSF



Abstract

Type 2 innate lymphoid cells (ILC2s), an innate source of the type 2 cytokines interleukin (IL)-5 and -13, participate in the maintenance of tissue homeostasis. Although type 2 immunity is critically important for mediating metabolic adaptations to environmental cold, the functions of ILC2s in beige or brown fat development are poorly defined. We report here that activation of ILC2s by IL-33 is sufficient to promote the growth of functional beige fat in thermoneutral mice. Mechanistically, ILC2 activation results in the proliferation of bipotential adipocyte precursors (APs) and their subsequent commitment to the beige fat lineage. Loss- and gain-of-function studies reveal that ILC2- and eosinophil-derived type 2 cytokines stimulate signaling via the IL-4Ra in PDGFRa APs to promote beige fat biogenesis. Together, our results highlight a critical role for ILC2s and type 2 cytokines in the regulation of adipocyte precursor numbers and fate, and as a consequence, adipose tissue homeostasis.

The molecular interaction of plants and bacterial pathogens

(Time) 16:30–17:00

Anna Joe

The Pam Ronald laboratory, Plant pathology department, UC Davis



Abstract

An important aspect of innate immunity is the perception of pathogen-associated molecular patterns (PAMPs) by specific pattern recognition receptors (PRRs) leading to PAMP-triggered immunity (PTI). Plant PRRs are key to immunity, as their inhibition or loss of function leads to enhanced susceptibility to adapted and non-adapted pathogens. Pathogens must block or avoid PTI to cause disease. A potent strategy to inhibit PTI is via the action of secreted effectors delivered into the host cells leading to effector-triggered susceptibility (ETS). Corresponding host targets have been identified only for a few of them, but they revealed that effectors interfere with key components of PTI. I will present about each well-developed story of rice receptor kinase and an effector from *Pseudomonas syringae*.

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SYMPOSIA II

(N-114)

Cortical representation of complex spectrotemporal features in songbirds

(Time) 14:45–15:15

Gunsoo Kim

Center for Integrative Neuroscience, UCSF



Abstract

Understanding how the auditory cortex processes complex communication signals remains a major challenge. With their rich vocal communication behaviors, songbirds can offer insights into this question. We investigated the neural representation of sound features in the cortical auditory areas of zebra finches. In the primary cortical area field L, our systematic mapping of spectrotemporal receptive fields revealed a highly organized representation in which sharpness of frequency and temporal tuning of sound is mapped along two separate anatomical axes. The clustering of temporally or spectrally selective neurons suggests that filtering for basic perceptual qualities such as tempo and pitch occurs in a spatially organized and segregated manner. Moreover, using information theoretic technique, we find that many neurons encode a second sound feature that carries significant additional information about the neural response. Together, our data reveal a systematic mapping of sound features onto songbird cortical neurons.

A patient-derived mutant RAD51 fails to protect blocked replication forks and results in Fanconi anemia-like syndrome

(Time) 15:15–15:45

TaeHo Kim

UC Davis



Abstract

Repair of DNA interstrand crosslinks requires the concerted action of multiple DNA repair pathways, including homologous recombination. Defective crosslink repair can result in Fanconi anemia, a bone marrow failure and cancer predisposition syndrome. Here, we report a *de novo* heterozygous T131P mutation in *RAD51*, the key recombinase essential for homologous recombination, in a patient with Fanconi anemia-like phenotype. *In vitro*, RAD51-T131P displays DNA-independent ATPase activity, no DNA pairing capacity and a co-dominant negative effect on RAD51 recombinase function. The patient cells are sensitive to crosslinking agents and display hyperphosphorylation of Replication Protein-A due to increased activity of DNA2 nuclease at the DNA crosslink. These findings demonstrate that proper RAD51 function is essential to protect the replication fork from DNA degradation during crosslink repair. Our study provides a molecular basis for how RAD51 and its associated factors may operate in a homologous recombination-independent manner to maintain genomic integrity in order to prevent tumorigenesis.

Identification the role of histone methyltransferase SETD7 in epigenetic dynamics during cardiac lineage specification

(Time) 16:00~16:30

Jaecheol Lee

Joseph C. Wu Lab, Dept. of Medicine, Stanford



Abstract

Coordinated epigenetic regulation is fundamental for differentiation of human embryonic stem cells (hESCs) into cardiomyocytes (CMs). However, the precise mechanism how epigenetic modifying enzyme modulates epigenetic alterations in specific lineages of cardiac differentiation is still unclear. We identified histone methyltransferase, SET domain containing protein 7 (SETD7), as a key regulator of cardiac differentiation. SETD7 expression is up-regulated during CM differentiation, which is crucial for the differentiation of hESCs into CMs. Chromatin immunoprecipitation sequencing (ChIP-seq) revealed that the SETD7 directly binds at the intragenic region of cardiac-specific genes, and its enrichment patterns were correlated with coordinated alterations of H3K36me3 during CM differentiation. The peptide array and immunoprecipitation conjugated with mass spectrometry showed the interaction of SETD7 with H3K36me3 and several chromatin remodeling factors suggesting the function of SETD7 in gene activation during CM differentiation. Collectively, our results demonstrated a critical role of SETD7 in CM differentiation, and provided novel insights into the regulation of epigenetic dynamics by histone modifying enzymes during CM differentiation.

Stage-specific transcriptional repression directs selective gene activation in an adult stem cell lineage

(Time) 16:30~17:00

Jongmin Kim

Margaret T. Fuller Lab, Dept. of Chemical and Systems Biology, Stanford



Abstract

A single cell uses only a subset of its genome to establish and maintain its unique identity. A great mystery of metazoan development is how such cell type specific gene expression programs are set up during differentiation. Much attention has focused on the role of gene selective transcriptional activators in initiating cell type specific transcriptional programs. Here we show that proper terminal differentiation in an adult stem cell lineage requires stage-specific transcriptional repression mechanisms that prevent promiscuous activation of transcripts characteristic of alternate cell fates. A novel gene, *tZnF*, encoding a 6 C2H2-type zinc finger protein, was expressed specifically in male germ cells starting in early spermatocytes and localized to chromatin. Loss of *tZnF* by knock down as well as CRISPR-Cas9 mediated knock out resulted in strong defects in differentiation and *tZnF*^{-/-} spermatids failed to elongate to make functional sperm. Analysis of transcripts in *tZnF* knock down testes showed dramatic upregulation of over 500 genes normally expressed in specific somatic cell types or organs but not in testis. Surprisingly, for spermatocytes in which the repressor was knocked down, testis Meiotic Arrest Complex (tMAC), the transcriptional activator of spermatocyte-specific genes was responsible for the misexpression of somatic genes. Thus through cell-type specific expression of a transcriptional repressor, tZnF, the developmental program prevents collateral activation of somatic specific transcripts inappropriate for the lineage by restricting the action of tMAC to testis transcripts. Our findings highlight the importance of cell type and gene selective transcriptional repression mechanisms for proper differentiation in stem cell lineages.

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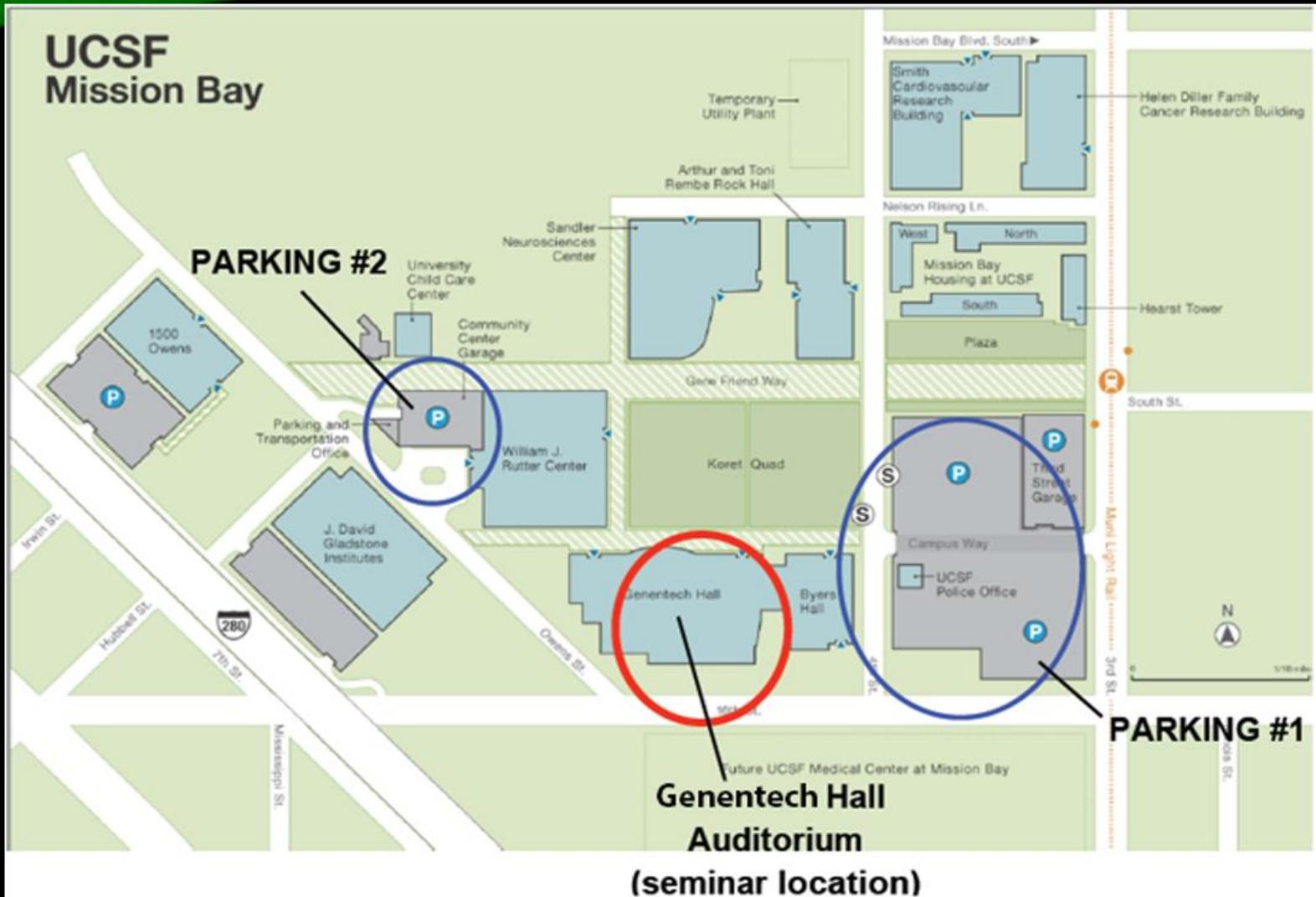
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Directions and Parking
UCSF Mission Bay, Genentech Hall
Auditorium
600 16th St., San Francisco, CA 94158



Direction to Mission Bay Campus, UCSF

From East Bay:

- 1: Take the I-80 W toward San Francisco
- 2: Take the 5TH ST exit, EXIT 1B, on the LEFT toward US-101 N/ GOLDEN GATE BR.
- 3: Turn SHARP LEFT onto 5TH ST.
- 4: Turn RIGHT onto TOWNSEND ST.
- 5: Turn LEFT onto 7TH ST.
- 6: Turn SLIGHT LEFT onto 16TH ST.
- 7: Turn LEFT onto 4TH ST. The parking lot is on your right side.

From South San Francisco:

- 1: Start out going NORTH on US-101 or I-280.
- 2: Merge onto I-280 N toward PORT OF SF.
- 3: Take the MARIPOSA STREET exit.
- 4: Turn RIGHT onto MARIPOSA ST.
- 5: Turn LEFT onto 3RD ST.
- 6: Turn LEFT onto 16TH ST.
- 7: Turn RIGHT onto 4TH ST. The parking lot is on your right side.

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