2016. 12. 10.

KOLIS Winter Conference

University of California, Berkeley

Morgan Hall

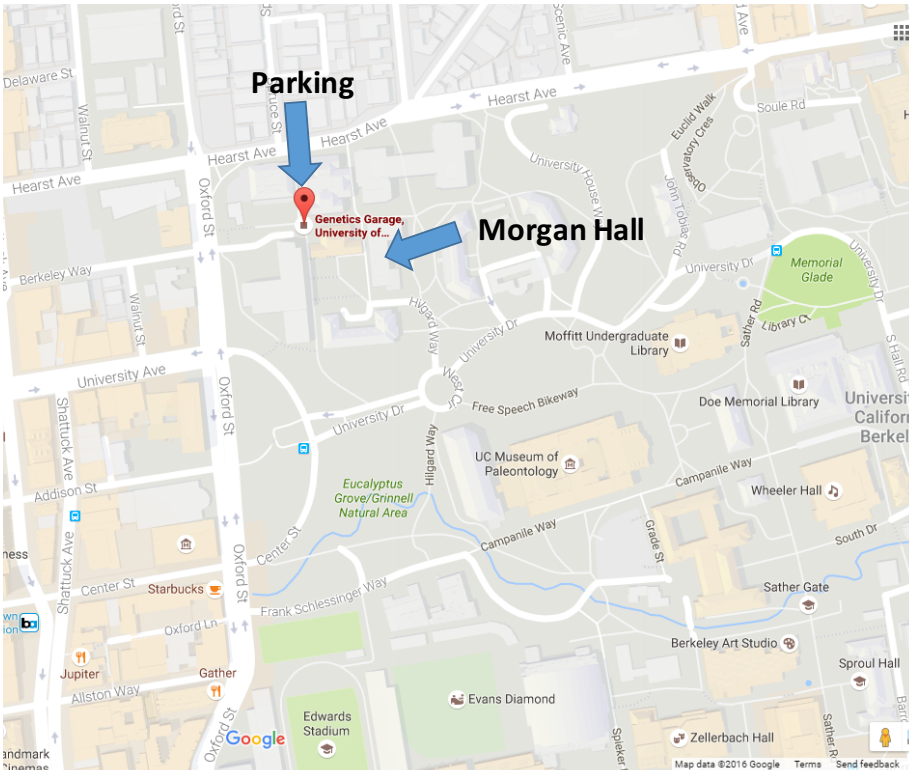
University of California
San Francisco

KOLIS Korean Life Scientists In the bay area

Directions

Parking (Berkeley Way and Oxford Street, Berkeley): Genetics Garage, UC Berkeley

[Click here for a direct link to Google Maps]



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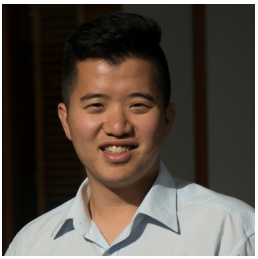

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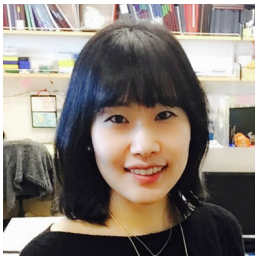


Heenam Kwon, Ph.D.
UC Davis

Kicheol Kim, Ph.D.
UC San Francisco

Siyeon Rhee, Ph.D.
Stanford

Agenda

Welcome

12:00 PM - 01:00 PM — Registration

01:00 PM - 01:10 PM — Opening by Narae Lee, Ph.D.
KOLIS President
Rm 101

Plenary Talks

01:10 PM - 01:50 PM
Rm 101 — Young Shin Kim, M.D., MPH, Ph.D.
UCSF Weill Institute for Neurosciences; Department of Psychiatry, UCSF
Title: Know what you see rather than see what you know: prevalence of ASD

01:50 PM - 02:30 PM
Rm 101 — Jinoh Kim, Ph.D.
Division of Genomic Medicine, Department of Pediatrics, UC Davis Medical Center
Title: SEC23A and SEC24D play essential roles in ER export of a large cargo molecule, procollagen

02:30 PM - 03:10 PM
Rm 101 — Taek Soon Lee, Ph.D.
Joint BioEnergy Institute, Lawrence Berkeley National Laboratory, Emeryville
Title: Biological Design and Engineering of Biofuels and Biochemicals Producing Microbial System

03:10 PM - 03:30 PM — Coffee Break

Agenda

Session Talks

	Symposia I (Rm 101)	Symposia II (Rm 138)
03:30 PM - 04:00 PM	Kyuho Han, Ph.D. Title: A CRISPR-based genetic interaction map identifies synergistic drug combinations for cancer	Youngchool Choe, Ph.D. Title: 20 things young Korean Job seekers in the Bay area's Biotech Industry may want to know. Assorted tips on how to search for open positions, power up your CV and maintain balanced perspectives on life
04:00 PM - 04:30 PM	Karam Kim, Ph.D. Title: The interplay between the kinase and structural functions of CaMKII during synaptic plasticity of hippocampal pyramidal neuron	Young-Jun Jeon, Ph.D. Title: MiRNA mediated TS3 deficiency enhances metastatic potential of NSCLC by regulating UPR and ERAD
04:30 PM - 05:00 PM	Jung Kim, Ph.D. Title: Mechanical stress regulates insulin sensitivity through integrin-dependent control of insulin receptor localization	Jaehak Oh Ph.D. Title: MARCH1 ubiquitin ligase supports regulatory T cell development and repertoire expansion by rescueing thymic dendritic cells from MHCII proteotoxicity

Sponsor Talks and Dinner

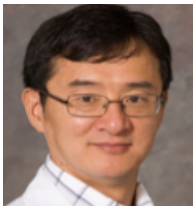
05:10 PM - 06:00 PM Rm 101	Sponsors Info Session Doeul Law LLP, Korea Innovation Center, Bioneer, Korea Evaluation Institute of Industrial Technology, Genius Factory
06:05 PM - 06:20 PM Rm 101	Current and new KOLIS board introduction & budget report
06:30 PM Rm 101	Raffle, dinner, and networking

Plenary Speakers

Young Shin Kim, MD, MS, MPH, PhD.

Associate Professor, UCSF Weill Institute for Neurosciences
Department of Psychiatry, UC San Francisco

Know what you see rather than see what you know: prevalence of ASD



Jinoh Kim, Ph.D.

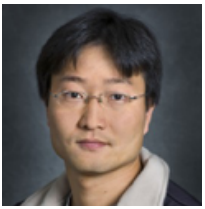
Associate Professor
Division of Genomic Medicine, Department of Pediatrics,
University of California Davis Medical Center, Sacramento, California

SEC23A and SEC24D play essential roles in ER export of a large cargo molecule, procollagen

Most secretory and cell surface membrane proteins are synthesized in the endoplasmic reticulum (ER) and traffic through the secretory pathway. These cargo molecules exit the ER in a transport vesicle. Assembly of this transport vesicle is catalyzed by cytosol coat complex II (COPII). COPII proteins consist of SAR1, SEC23/SEC24 complex, and SEC13/SEC31 complex. Typical COPII vesicles are about 60-80 nm in diameter. Interestingly, cargo molecules such as procollagen and chylomicron are too large to be packaged into typical COPII vesicles. Thus, it is unclear how COPII proteins expand the dimension of COPII vesicles.

Our laboratory has contributed to discovery of two human diseases caused by mutations in COPII genes. Patients with mutations in SEC23A or SEC24D show strong craniofacial and skeletal defects. Our in vitro and in vivo results indicate that SEC23A or SEC24D mutations compromise ER export of procollagen, which partially accounts for bone phenotypes in the patients. Our results suggest that SEC23A and SEC24D are necessary for packaging procollagen into large COPII vesicles.

Plenary Speakers



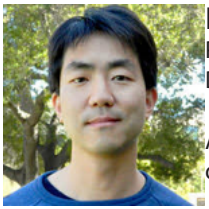
Taek Soon Lee, Ph.D.
Director of Metabolic Engineering and Deputy VP of Fuels Synthesis
Joint BioEnergy Institute, Lawrence Berkeley National Laboratory, Emeryville, CA,

Biological Design and Engineering of Biofuels and Biochemicals Producing Microbial System

Single-cell microbes, such as E. coli and yeast, can be redesigned to be miniature chemical reactors that transform sugars into biofuels and biochemicals. With the development of Synthetic Biology, we can design biological system, introduce biosynthetic pathways from one organism into a host microbe, and engineer metabolic pathways using genetic manipulation to optimize the production of target biofuels and biochemicals that the host microbe does not naturally generate. The goal of this study is to create highly efficient microbial factories for green, cost-effective and sustainable production of advanced biofuels and other valuable products.

In this talk, I will present two synthetic E. coli systems that have been designed and engineered for production of two important compounds. The first example is a microbial isopentenol producer, and the other is a microbial producing system for hydroxytyrosol, a strong antioxidant extracted from olive.

Session Speakers



Kyuho Han, Ph.D

Postdoctoral Fellow at Bassik Lab
Department of Chemical & Systems Biology, Stanford

A CRISPR-based genetic interaction map identifies synergistic drug combinations for cancer

Identification of effective combination therapies is critical to address the emergence of drug-resistant cancers. Although millions of drug combinations might be created by repurposing existing drugs, direct screening of these combinations is infeasible. Here, we designed a scalable CRISPR-based double knockout (CDKO) system to generate a mammalian genetic interaction (GI) map at unprecedented scale, comprised of 490,000 double-sgRNAs directed against 21,321 pairs of drug targets. We first developed an efficient strategy for cloning and sequencing the libraries, as well as a robust statistical scoring method for calculating GIs from CRISPR-deleted gene pairs. We then extensively validated this system in a second GI-rich CDKO map by demonstrating its ability to identify known and novel genetic interactions in the ricin pathway, as well as established protein-protein interactions (PPIs). Using this validated system, we searched for rare synthetic lethal drug target pairs in K562 leukemia cells and identified a number of potent combinations for which corresponding drugs exhibit synergistic killing. These included the BCL2L1 and MCL1 combination, which was exceptionally effective even in imatinib-resistant cells. Together, this work demonstrates an effective strategy to screen synergistic drug combinations in high throughput, and a powerful CRISPR-based tool to dissect functional genetic interaction networks.

Youngchool Choe, Ph.D.

Bio-Rad Laboratories Inc.

20 things young Korean Job seekers in the Bay area's Biotech Industry may want to know. Assorted tips on how to search for open positions, power up your CV and maintain balanced perspectives on life.

Session Speakers

Karam Kim, Ph.D.

UC Davis Assistant Project Scientist
Department of Pharmacology, UC Davis

The interplay between the kinase and structural functions of CaMKII during synaptic plasticity of hippocampal pyramidal neuron

The structural modification of dendritic spines plays a critical role in synaptic plasticity. CaMKII (Ca2+/calmodulin-dependent protein kinase II) is a pivotal molecule involved in this process through both kinase-dependent and independent structural functions but respective contribution of these two remains unclear. We demonstrate that the transient interplay between the kinase and structural functions of CaMKII during the induction of synaptic plasticity temporally gates the activity-dependent modification of the actin cytoskeleton. Inactive CaMKII binds F-actin, thereby limiting access of actin regulating proteins to F-actin and stabilizing spine structure. CaMKII-activating stimuli trigger dissociation of CaMKII from F-actin through specific autophosphorylation reactions within the F-actin binding region and permit F-actin remodeling by regulatory proteins followed by reassociation and restabilization. Blocking the autophosphorylation impairs both functional and structural plasticity without affecting kinase activity. These results underpin the importance of the interplay between the kinase and structural functions of CaMKII in defining a time window permissive for synaptic plasticity.

Young-Jun Jeon, Ph.D.

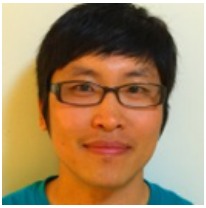
Associate Specialist at Diehn Lab
Stanford Cancer Institute & Stem Cell Bio Regenerative Medical Institute, Stanford

MiRNA mediated TS3 deficiency enhances metastatic potential of NSCLC by regulating UPR and ERAD

Lung cancer is the most common cause of cancer-related deaths in the world. Non-small cell lung carcinoma (NSCLC) accounts for about 85% of all lung cancers. Non-coding RNAs are functional RNA molecules not translated into protein. There are now different types of non-coding RNAs which have been identified and characterized: 1. Small non-coding (snc) RNAs, 10-40 nucleotides in length. 2. Long non-coding (lnc) RNA, more than several hundred nucleotides. Recently, a numerous paper has convinced that miRNAs, the largest group of sncRNAs, play a critical role in tumorigenesis and cancer metastasis.

Cells have Endoplasmic Reticulum (ER) quality control machineries to monitor the proper folding status of a polypeptide. The major response to the accumulation of unfolded and/or misfolded proteins referred to as ER stress is an activation of Unfolded Protein Response (UPR) pathway. As a consequence of the failure to reach a native conformation, the misfolded proteins are frequently subjected to Endoplasmic Reticulum Associated Degradation (ERAD) pathway. ERAD is highly activated upon unfolded protein accumulation and ER stress induction, which is closely associated with several diseases including cancer by degrading numerous substrates including not only ER-resident proteins but also cytoplasmic proteins. However the exact roles and regulating factors of ERAD in cancer metastasis remain unexplored. The presentation will briefly discuss the roles of miRNAs to characterize a novel tumor suppressor associated with ERAD and cancer metastasis.

Session Speakers



Jung Kim, Ph.D.

Postdoctoral Fellow at Bilder Lab
Department of Molecular and Cell Biology, UC Berkeley

Mechanical stress regulates insulin sensitivity through integrin-dependent control of insulin receptor localization

'Insulin sensitivity' describes the cellular activity of the Insulin pathway in the presence of ligand, and misregulation of Insulin sensitivity leads to metabolic diseases including type-2 diabetes. Mechanical stress resulting from e.g. exercise is a known regulator of Insulin sensitivity, but the underlying molecular mechanisms are unclear. Here, we use the Drosophila larval fat body to address this question. We find that Insulin signaling activity is abolished in the absence of mechanical stress, even when excess Insulin is present. Mechanical stress is required for activation of Insulin signaling in a new ex vivo assay as well as in vivo. Interestingly, Insulin Receptor (InR) and other downstream components are cytoplasmically localized in the absence of mechanical stress, but are recruited to the plasma membrane upon stress initiation. Stress sensing is mediated by Integrins, whose activation is sufficient as well as necessary for recruitment and signaling thought InR; Integrin activation is also necessary and sufficient for mechanical stress-dependent activation of TOR. Together, our data suggest that Integrin-mediated mechanical stress controls Insulin sensitivity by altering localization of InR.

Jaehak Oh, Ph.D.

Associate Specialist at Shin Lab
Deptartment of Microbiology and Immunology, Sandlers Asthma Basic Research (SABRE) Center, UC San Francisco

MARCH1 ubiquitin ligase supports regulatory T cell development and repertoire expansion by rescueing thymic dendritic cells from MHCII proteotoxicity

Membrane-Associated RING-CH1 (MARCH1) is an ubiquitin ligase preferentially expressed in antigen-presenting cells and mediating ubiquitination and degradation of MHCII and CD86. MARCH1 deficiency results in a significant reduction in the number of thymic regulatory T (Treg) cells in mice, implicating an important role of this molecule in Treg cell development. However, neither the mechanism by which MARCH1 supports Treg cell development nor its impact on Treg cell function has been clearly defined. Here we report that the role of MARCH1 in Treg cell development critically depends on its expression in dendritic cells and its activity to ubiquitinate MHCII. Importantly, not the MHCII ubiquitination per se but its consequences to MHCII proteostasis is essential for dendritic cells to organize surface proteins appropriately, make a stable conjugate with thymocytes, and provide them with sufficient signal for Treg cell differentiation. When MARCH1-mediated MHCII proteostasis in dendritic cells fails, Treg cells develop to a markedly reduced number and a substantially restricted TCR repertoire in the thymus, resulting in the function of these cells to suppress graft-versus-host-disease severely impaired. Thus, MARCH1-mediated MHCII proteostasis plays an essential role for thymic dendritic cells to select regulatory T cells in abundance and diversity required for proper immune suppression.

Sponsors

On behalf of KOLIS, we would like to thank these sponsors for their support: