2013 KOLIS Annual Meeting

Sponsored by KRIBB

Helen Diller Cancer Center HD-160
UCSF Mission Bay Campus
1450 3rd St., San Francisco, CA 94158

April 27, 2013
12:00 pm – 18:00 pm
Committee

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Stanford University

Stanford University

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UC San Francisco

Stanford University

UC Davis

UC Berkeley
Dear KOLIS members,

KOLIS is pleased to announce our first annual meeting in 2013, which will be held on April 27th, 2013 at Helen Diller Cancer Center HD-160, UCSF Mission Bay Campus. This annual meeting is supported by KRIBB (Korea Research Institute of Bioscience and Biotechnology).

For this meeting, we have invited Dr. Sung Uk Kim (Vice Present) and Dr. Kyu-Sun Lee (Senior Research Scientist) from KRIBB. Through this interaction with KRIBB, our KOLIS members would have a great opportunity to learn not only about KRIBB but also about general biomedical research in South Korea. After two presentations from KRIBB, four KOLIS members from each university who have recently achieved great progress in their fields will present their on-going exciting research. As always, we encourage our members to participate in scientific discussion and networking between members.

On behalf of all KOLIS members, I would like to thank all the speakers who have generously agreed to contribute to this KOLIS annual meeting by sharing their expertise and experiences with others. Furthermore, I deeply appreciate the continued support and interest of our valued sponsors throughout this year. Their generosity makes our KOLIS programming possible and allows us to bring the bay area scientists together. Please join us for this wonderful annual meeting. It will be a great opportunity to interact with fellow researchers in the bay area and communicate with each other in regard to emerging trends and cutting-edge issues in science. I look forward to meeting all of you there.

Sincerely yours,

Won-Suk Chung, Ph.D.

2013 KOLIS President
Directions and Parking
Helen Diller Cancer Center HD-160
UCSF Mission Bay Campus
1450 3rd 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.

* Please park your car at surface parking lot located at 4th street and Gene Friend Way for $2.50/day (blue circle on the map)
2013 KOLIS Annual Meeting
Sponsored by KRIBB, April 27, 2013

Helen Diller Cancer Center HD-160
UCSF Mission Bay Campus
1450 5th St., San Francisco, CA 94158

12:00 – 13:00 Registration

13:00 – 13:20 Opening Remarks
Won-Suk Chung, Ph.D. (2013 KOLIS President)

13:20 – 13:50 Talk 1: 김성욱 (KRIBB; Vice President)
About KRIBB

13:50 – 14:30 Talk 2: 이규선 (KRIBB)
Drosophila model is a genetic tool kit for human age-related diseases

14:30 – 15:10 Talk 3: 이혜영 (UC San Francisco)
Dendritic potassium channel regulation by Fragile X Mental Retardation Protein (FMRP)

15:10 – 15:30 Coffee Break

15:30 – 16:10 Talk 4: 이호영 (UC Berkeley)
Investigating RNA-protein interaction networks using a conditional CRISPR nuclease

16:10 – 16:50 Talk 5: 김은배 (UC Davis)
Comparative genomics to understand animal physiology

16:50 – 17:30 Talk 6: 김성연 (Stanford)
Diverging neural pathways assemble a behavioural state from separable features in anxiety

17:30 – 18:00 경품추첨 & Closing Remarks

18:00 – Dinner & Networking
Invited speaker

ABOUT KRIIBB

*(Korea Research Institute of Bioscience and Biotechnology)*.

Sung Uk Kim, Ph.D.
Vice President, KRIIBB

- 연세대학교 응용 미생물학 박사 (1986)
- 한국화학연구소 생물공학연구실 선임연구원 (1986~1991)
- 한국생명공학연구원 책임연구원 (1992~현재)
- 한국생명공학연구원 선임연구본부장 (2012~현재)
- 기초기술연구회 기획평가위원회 자문위원 (2009~2011)
- 한국한의학연구원 자문위원 (2009~2011)
Drosophila model is a genetic tool kit for human age-related diseases

Kyu-Sun Lee, Ph.D.
Senior Research Scientist
Aging Research Center, KRIIBB

According to develop modern society, increased lifespan is accompanied by an increased prevalence of age-related diseases like cancers, metabolic diseases and neurodegenerative diseases. Drosophila melanogaster is a highly tractable genetic model organism for understanding molecular mechanisms of human diseases. Many basic biological, physiological, and neurological properties are conserved between mammals and Drosophila, and over 75% of human disease-causing genes are evolutionary and functionally conserved in Drosophila. In addition, Drosophila neuroblasts, the stem cell in developing fly brain, have emerged as a key model system for neural stem cell biology and brain tumor formation. Based on the advantage of Drosophila, we investigated the pathogenic mechanisms for metabolic diseases and brain tumor using Drosophila model systems. Feeding behavior is one of the most essential activities in animals, which is tightly regulated by neuroendocrine factors. We suggest that DYRK1A-Sirt1-FOXO-NPY signaling pathway regulated feeding both in mammalian and Drosophila model and fasting-feeding cycles are controlled by the post-translational modification of the dFOXO transcription factor. In addition, I will introduce that the molecular mechanisms on balancing between self-renewal and differentiation in neural stem cells. Especially, we suggest that overactivated notch pathway triggered the transition from normal neural stem cell to cancer stem cells result in brain tumor formation. These results propose that Drosophila models of human metabolic diseases and brain cancer can effectively be used for mining the new therapeutic approaches as well as drug discovery.

Education and position

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<tr>
<th>Year</th>
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<td>2006.2 – 2008.5</td>
<td>Postdoc.</td>
<td>Chung-Ang University</td>
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<td>2006</td>
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<td>2006.2 – 2008.5</td>
<td>Visiting Scholar –Dept. of Pathology</td>
<td>Stanford University</td>
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<td>2008.5 – Present</td>
<td>Senior Research Scientist</td>
<td>KRIIBB</td>
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<td>2010.3 – Present</td>
<td>Adjunct Professor</td>
<td>University of Science and Technology</td>
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<td>2002</td>
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Dendritic potassium channel regulation by Fragile X Mental Retardation Protein (FMRP)

Hye Young Lee, Ph.D.
UC San Francisco

Fragile X syndrome (FXS) is a common form of mental disability and one of the known causes of autism. The mutation responsible for FXS is a large expansion of the trinucleotide CGG repeats that leads to DNA methylation of the fragile X mental retardation gene 1 (FMR1) and transcriptional silencing, resulting in the absence of fragile X mental retardation protein (FMRP), an mRNA binding protein. Although it is widely known that FMRP is critical for metabotropic glutamate receptor (mGluR)-dependent long-term depression (LTD), which has provided a general theme for developing pharmacological drugs for FXS, specific downstream targets of FMRP may also be of therapeutic value. Since alterations in potassium channel expression level or activity could underlie neuronal network defects in FXS, we will describe our recent findings on how the dendritic potassium channel, Kv4.2 might be altered in mouse models of FXS and the possible therapeutic avenues for treating FXS.

How transmitter receptors modulate neuronal signaling by regulating voltage-gated ion channel expression remains an open question. Recently we reported dendritic localization of mRNA of Kv4.2 voltage-gated potassium channel, which regulates synaptic plasticity, and its local translational regulation by FMRP. FMRP suppression of Kv4.2 is revealed by elevation of Kv4.2 in neurons from fmrl knockout (KO) mice, and in neurons expressing Kv4.2-3’UTR that binds FMRP. Moreover, treating hippocampal slices from fmrl KO mice with Kv4 channel blocker restores long-term potentiation (LTP) induced by moderate stimuli. The evaluation of Kv4.2 potassium channels as potential therapeutic targets for FXS will allow us to be extended to include additional proof-of-principle tests for genetic reduction of Kv4.2 as potential ways to reduce fmrl KO mutant phenotypes, with a strong interest in pursuing translational research to develop treatments and improve outcomes for individuals with FXS.

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<td>2013–Present</td>
<td>Associate Specialist, PI: Lily Jan</td>
<td>UC San Francisco</td>
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<td>2008–2013</td>
<td>Postdoc, PI: Lily Jan</td>
<td>UC San Francisco</td>
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<td>2006–2008</td>
<td>Postdoc, Signal Transduction Lab</td>
<td>POSTECH</td>
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<td>2001–2006</td>
<td>Ph.D. in Life Science, PI: Dr. Sung Ho Ryu</td>
<td>POSTECH</td>
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<tr>
<td>1997–2006</td>
<td>B.S in Life Science</td>
<td>Ewha Womans University</td>
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Investigating RNA-protein interaction networks using a conditional CRISPR nuclease

Ho Young Lee, Ph.D.
UC Berkeley

RNA binding proteins control the fate and function of the transcriptome in all cells. Here we present a technology for isolating RNA-protein partners efficiently and accurately using an engineered CRISPR endoribonuclease. An inactive version of the Csy4 nuclease binds irreversibly to transcripts engineered with a 16-nucleotide hairpin sequence at their 5’ ends. Once immobilized by Csy4 on a solid support, contaminating proteins and other molecules can be removed by extensive washing. Upon addition of imidazole, Csy4 is activated to cleave the RNA, removing the hairpin tag and releasing the native transcript along with its specifically-bound protein partners. This conditional Csy4 enzyme enables recovery of specific RNA binding partners with minimal false-positive contamination. We use this method, coupled with quantitative mass spectrometry, to identify cell type-specific human pre-miRNA binding proteins. We also show that this technology is suitable for analyzing diverse size transcripts, and that it is suitable for adaptation to a high-throughput discovery format.

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<td>2008.8 – Present</td>
<td>Postdoc, PI: Prof. Jennifer Doudna</td>
<td>UC Berkeley</td>
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<td>1996.3 – 2000.2</td>
<td>B.S. in Chemistry</td>
<td>Seoul National University</td>
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Recent sequencing technology has had many genome sequences of animals, plants, and microorganisms released. Although many genomes are available for now, we frequently experience difficulties in translating it. In this presentation, I will show examples of genome analysis using a genome sequence from the Naked Mole Rat (NMR, *Heterocephalus glaber*), and how to get interesting biological inferences from the analysis. The NMR is an animal belonging to the Rodentia Order, who lives in East Africa. The NMR is as small as a mouse, but it lives 10-fold longer than a mouse. It has little hair in the wrinkled pink skin, is strongly resistant to cancer formation, and shows defective phenotypes in body temperature control, visual perception, tasting, and pain sensation in the skin. The NMRs lived together in a harsh underground environment, and exhibit unique eusociality shown in ants and bees, which are not common in mammals. Living in the closed underground environment have influenced resistance to hypoxia and high level of CO₂, poor visual perception, and feeding on tubers found underground. Research groups including me used comparative genomic approaches to reveal that the most phenotypes are genetically associated with their genome contents. The comparative genomics is extensively applied to genome research on not only animals and plants but also microorganisms. For now, the second generation sequencing platforms are applied in many research areas because of high throughput capacities with low error rates at reasonably low costs. Moreover, the 3rd generation sequencing platforms are currently available for longer sequencing despite of higher error rates at reasonable cost. These advanced technologies will lead us to do genome analyses more aggressively. Currently, many research groups are sequencing genomes of individual human subjects and a diversity of pathogens and microorganisms, which will give us better opportunities for healthier and safer lives.
Diverging neural pathways assemble a behavioural state from separable features in anxiety

Sung-Yon Kim, Ph.D. Candidate
Stanford University

Behavioural states in mammals, such as the anxious state, are characterized by several features that are coordinately regulated by diverse nervous system outputs, ranging from behavioural choice patterns to changes in physiology (in anxiety, exemplified respectively by risk-avoidance and respiratory rate alterations). Here we investigate if and how defined neural projections arising from a single coordinating brain region in mice could mediate diverse features of anxiety. Integrating behavioural assays, in vivo and in vitro electrophysiology, respiratory physiology and optogenetics, we identify a surprising new role for the bed nucleus of the stria terminalis (BNST) in the coordinated modulation of diverse anxiety features. First, two BNST subregions were unexpectedly found to exert opposite effects on the anxious state: oval BNST activity promoted several independent anxious state features, whereas anterodorsal BNST-associated activity exerted anxiolytic influence for the same features. Notably, we found that three distinct anterodorsal BNST efferent projections—to the lateral hypothalamus, parabrachial nucleus and ventral tegmental area—each implemented an independent feature of anxiolysis: reduced risk-avoidance, reduced respiratory rate, and increased positive valence, respectively. Furthermore, selective inhibition of corresponding circuit elements in freely moving mice showed opposing behavioural effects compared with excitation, and in vivo recordings during free behaviour showed native spiking patterns in anterodorsal BNST neurons that differentiated safe and anxiogenic environments. These results demonstrate that distinct BNST subregions exert opposite effects in modulating anxiety, establish separable anxiolytic roles for different anterodorsal BNST projections, and illustrate circuit mechanisms underlying selection of features for the assembly of the anxious state.

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<tr>
<td>2009 – Present</td>
<td>Ph.D. in Neuroscience PI: Karl Deisseroth</td>
<td>Stanford University</td>
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<tr>
<td>2003 – 2009</td>
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바이오니어

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대구과학기술대학교 (DGIST)

재미한인과학기술자협회 (KSEA)

한국한의학연구원