KOLIS

KOREAN LIFE SCIENTISTS IN THE BAY AREA

2022 KOLIS
Virtual Mini Conference

NOVEMBER 28th
6 pm (PST)

Berkeley UNIVERSITY OF CALIFORNIA
UCDAVIS UNIVERSITY OF CALIFORNIA
Stanford University
UCSF University of California San Francisco

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KOLIS KOREAN LIFE SCIENTISTS IN THE BAY AREA

2022 1st KOLIS Virtual Mini Conference Nov 28 (Mon) 6 pm

Registration and Opening remarks	
18:00 – 18:05	Registration: check-in for dinner coupon
18:05 – 18:10	Opening remark: **Min Gee Cho, Ph.D.** President of KOLIS
Session I (moderator: Min Gee Cho)	
18:10 – 18:45	**Keynote Lecture: Hijai Regina Shin, Ph.D.** (UC Berkeley) *"How lysosomal cholesterol sensing mechanism translates to growth signaling"*
18:45 – 19:00	Flash talk 1: **Jungwoo Wren Kim, Ph.D.** (UC Berkeley) *"Dissecting neuronal activity via calcium-dependent protein labeling"* Flash talk 2: **Byeonghak Park, Ph.D.** (Stanford University) *"Spider's pad inspired dynamic noise free bioelectronics"*
19:00 – 19:10	Break: Trivia
Session II (moderator: Serim Yang)	
19:10 – 19:40	Flash talk 3: **Gwanggyu Sun** (Stanford University) *"The E. coli Whole-Cell Modeling Project"* Flash talk 4: **Huijeong Jeong, Ph.D.** (UC San Francisco) *"Mesolimbic dopamine release conveys causal associations"* Flash talk 5: **Heonseok Kim, Ph.D.** (Stanford University) *"Single cell CRISPR engineering and characterization using long-read sequencing"* Flash talk 6: **Mangyu Choe, Ph.D.** (UC Berkeley) *"Leveraging genetic tool to manipulate mitochondrial physiology"*
19:40 – 19:55	Voting and networking
Flash talk winner announcement and Closing remarks	
19:55 – 20:00	Announcement of flash talk winner Closing remark: **Hyuncheol Lee, Ph.D.** Vice President of KOLIS

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KOLIS KOREAN LIFE SCIENTISTS IN THE BAY AREA

About KOLIS

KOLIS (Korean LIfe Scientists in the bay area)는 미국 캘리포니아주 샌프란시스코 인근, Bay area에 위치한 네 학교 (UC Berkeley, UC Davis, UC San Francisco, and Stanford)를 중심으로 생명과학 관련 Postdoc, 대학원생 및 연구원들이 결성한 학술 단체입니다. 1980년대 중반 bay area의 생명공학자들간의 교류회에서 시작되어 2022년 현재 300 여명의 회원들이 생명과학 관련 학업과 연구에 매진하고있으며, 2022년 한 해에만 70여명, 매해 30 - 40명의 회원들이 새로이 유입 및 배출되는 매우 역동적인 모임입니다.

KOLIS는 여러 분야에서 세계적인 연구 성과를 내고 있는 우수한 회원들간의 인적 네트워크 형성과 학술 정보 공유를 통해, KOLIS의 회원들이 향후 한국 생명 과학계의 큰 선도자로 자리매김할 수 있도록 최선을 다해 지원할 것을 약속드립니다.

KOLIS 임원진 일동 올림

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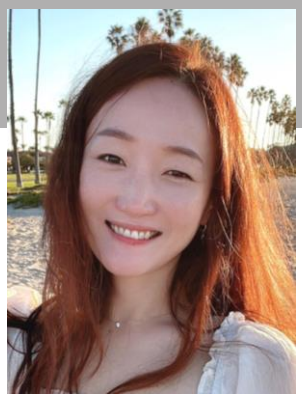
Welcome remarks



Min Gee Cho, Ph.D.
President, 2022 KOLIS

KOLIS 회원 여러분 안녕하세요!

2022년 하반기 KOLIS 회장을 맡고 있는 조민지입니다.
제1회 겨울 미니 총회에 참석해주신 여러분, 모두 환영합니다!

COVID-19로 인한 판데믹이 시작되던 2020년 3월을 생각하면 올해는 봄 총회와 가을 소풍을 대면으로 개최할 수 있게 되어 감회가 새로웠습니다. 수많은 생명과학자들의 뛰어난 연구로 백신과 치료제가 신속하게 개발되어, 올해는 pre-pandemic 모습을 대부분 되찾았습니다. 지난 3년은 생명과학자들의 역할이 인류의 생존과 직결된 문제들을 해결하고 기본적인 삶을 살 수 있게 해주는 데 필수적임을 새삼 느끼고 감사할 수 있었던 시간이었습니다.

최근 몇 달 동안 KOLIS에 새롭게 참여하시는 분들이 더욱 많아지는 것을 보면 판데믹으로 힘든 시기가 거의 끝나가고 있음을 실감합니다. 판데믹 기간 동안 우리는 가상공간을 통해 물리적 공간의 한계를 극복하고 교류할 수 있는 방법을 배웠습니다. 이번 미니 총회는 연구에 매진하느라 바쁜 KOLIS 회원들께서 거리의 제약 없이 각자의 자리에서 학술 교류를 하시고 지적 자극을 즐기시도록 온라인으로 준비했습니다.

9월부터 하반기 회장을 맡게 되어 가을 소풍과 겨울 미니 학회를 준비하는 동안 함께 애써준 회장단과 각 학교 대표님들께 진심으로 감사드립니다. 든든하게 도와준 저희 2022년 모든 임원진들 덕분에 무사히 개최하게 되었습니다. 또한 올 한 해에도 KOLIS의 다양한 활동을 지원해주신 여러 후원사들께 감사의 말씀을 드립니다. 짧은 기간 준비하며 많이 부족했지만, 봄, 여름 활동에 이어 가을과 겨울에도 KOLIS 회원분들께서 활발하게 교류하실 수 있는 장을 만들어 드리고자 열심히 준비했습니다. 판데믹으로 힘들고 어려운 시기임에도 훌륭한 연구를 하신 자랑스러운 KOLIS 회원분들을 연사로 모셨습니다. 모두 자유롭게 교류하고, 즐겁게 배우는 시간 되었으면 좋겠습니다.

앞으로도 저희 KOLIS 활동에 많은 관심 부탁드립니다! 귀한 시간 내어 참석해주신 모든 분께 진심으로 감사드립니다.

Minjee

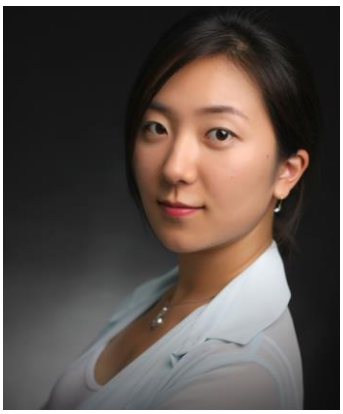
2022 KOLIS 회장 조민지 올림

KOLIS 2022 MINI Conference

Keynote Lecture

18:10 – 18:45

"How lysosomal cholesterol sensing mechanism translates to growth signaling"



Hijai Regina Shin, Ph.D.
Department of Molecular and Cell Biology, University of California at Berkeley, USA
Innovative Genomics Initiative at the University of California, Berkeley, USA

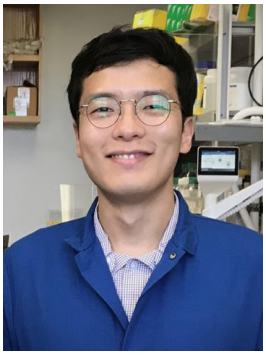
Cholesterol, a key building block for cellular membranes and a precursor for steroid hormones, was recently identified as a major nutrient input that activates the master growth regulator, mammalian Target of Rapamycin Complex 1 (mTORC1) kinase. A cholesterol pool on the lysosomal limiting membrane regulates the nucleotide loading state of the heterodimeric Rag guanosine triphosphatases (GTPases). In turn, under high cholesterol the Rag GTPases promote mTORC1 recruitment from the cytosol to the lysosomal membrane, where mTORC1 triggers downstream programs for biomass production and suppression of catabolism. Whereas genetic epistasis experiments place cholesterol sensing upstream of the Rag GTPases, our mechanistic understanding of how lysosomal cholesterol levels translates to modulation of the Rag GTPase nucleotide state and subsequent mTORC1 recruitment is limited. Combining organelle immune-isolation and proteomics with bioinformatic analysis, we identify a novel lysosomal transmembrane protein, which we name LYsosomal CHOlesterol Signaling (LYCHOS), as an essential factor that enables cholesterol-dependent activation of mTORC1. Biochemical analysis shows that LYCHOS binds to cholesterol and, in response, undergoes a conformational change that involves a large cytoplasmic domain. In turn, the cytoplasmic domain of LYCHOS undergoes cholesterol-dependent interactions with protein complexes that regulate Rag GTPase nucleotide status. Through these interactions, LYCHOS promotes Rag GTPase activation and mTORC1 recruitment to the lysosome. Through bioinformatics and gene expression analysis, we find that that LYCHOS is metabolically regulated in multiple tissues and downregulated upon fasting, and that its activity is required for a vast anabolic transcriptional program. In summary, we identify LYCHOS as a novel component of a lysosomal pathway that transduces cholesterol levels into activation of mTORC1 signaling and regulation of cell growth.

Session I

18:45 – 19:00

Flash Talk 1

Dissecting neuronal activity via calcium-dependent protein labeling



Jungwoo Wren Kim, Ph.D.
Department of Molecular and Cell Biology, UC Berkeley

The remarkable ability of neural circuits to change over time relies on structural and functional changes of neurons and their connections. These changes require precise control of activity-associated gene expression to fulfill the need for new protein synthesis. Defects in such control causes multiple neurological disorders including epilepsy and autism spectrum disorders. However, how global and local regulation of activity-associated gene expression are orchestrated to support neuroplasticity is not fully understood. To address this question from a new angle, I envisioned a molecular recorder that generates permanent biochemical traces in response to calcium transients. These biochemical marks would provide selective access to molecular events from active neurons. I have implemented this idea and begun a stepwise approach to interrogate the activity-associated gene expression. First, I engineered a calcium-dependent proximity labeling enzyme, Cal-ID, and showed that it can selectively label active neurons in the brain. Next, I developed the tool into a Calcium-dependent, Translating Ribosome Affinity Purification system (CalTRAP) that provides a new and robust way to investigate the activity-associated gene expression program. CalTRAP-seq applied to mouse epilepsy models discovered rapidly and translationally induced genes in response to seizure. I will use these tools to tackle the molecular mechanisms of translational regulation supporting neuroplasticity, and study how defective activity-associated gene expression can lead to neuronal disorders.

Session I

18:45 – 19:00

Flash Talk 2

Spider’s pad inspired dynamic noise free bioelectronics

Byeonghak Park, Ph.D.
Department of Chemical Engineering, Stanford University

As the demand for continuous biodiagnosis increases, many studies that can measure biosignals with a high signal-to-noise ratio are being conducted. A method that is currently being actively studied is a method of monitoring by inserting or attaching a small electronic device into the body. The potential of successful products such as smartwatches and pacemakers are already being recognized in the market. However, the chronic problem is external mechanical noise, and various signal interferences such as the user's unconscious contact or walking and running make signal analysis difficult and reduce accuracy. Post-processing methods such as signal processing cause signal distortion and require a lot of data and standards for accurate classification of signals.. The method proposed in this seminar is to develop a device that selectively damps only noise according to the frequency of the signal by designing a viscoelastic polymer to fit the biological system. This is using biomimetic technology, and in the case of the viscoelastic pad on the spider's leg, it is known that it detects the location of prey or enemies well even with external stimuli such as wind by selectively damping the signal according to the frequency. Viscoelastic materials that mimic these properties selectively block external noise in advance, enabling continuous monitoring. The selective reading of biomechanical and electrical signals under external noise suggests that biosignals can be read more successfully when biodevices are slightly softer and more viscous. (B. Park et al., Science 376, 6593, 624-629 (2022)). This seminar will cover materials, damping band control, and application possibilities, and I would like to invite all interested researchers.

Session II

19:10 – 19:40

Flash Talk 3

The E. coli Whole-Cell Modeling Project

Gwanggyu Sun, Ph.D. candidate
Department of Bioengineering, Stanford University

The Escherichia coli whole-cell modeling project seeks to create the most detailed computational model of an E. coli cell in order to better understand and predict the behavior of this model organism. Currently, the model includes the functions of 43% of characterized genes, with ongoing efforts to include additional data and mechanisms. As additional information is incorporated in the model, its utility and predictive power will continue to increase, which means that discovery efforts can be accelerated by community involvement in the generation and inclusion of data. This project will be an invaluable resource to the E. coli community that could be used to verify expected physiological behavior, to predict new outcomes and testable hypotheses for more efficient experimental design iterations, and to evaluate heterogeneous data sets in the context of each other through deep curation.

Session II

19:10 – 19:40

Flash Talk 4

Mesolimbic dopamine release conveys causal associations

Huijeong Jeong, Ph.D.
Department of Neurology, UC San Francisco

Learning to predict rewards based on environmental cues is essential for survival. It is widely believed that animals learn to predict rewards by updating predictions whenever the outcome deviates from expectations, and that such reward prediction errors (RPEs) are signaled by the mesolimbic dopamine system—a key controller of learning. However, instead of directly learning prospective predictions of outcomes following cues from RPEs, animals can infer predictions by learning the retrospective cause of rewards (i.e., whether a cue consistently precedes reward). Hence, whether mesolimbic dopamine instead conveys a causal associative signal that sometimes resembles RPE remains unknown. Here, we develop an algorithm for retrospective causal learning and show that mesolimbic dopamine release conveys causal associations but not RPE, thereby challenging the dominant theory of reward learning in the brain. Our results provide a new conceptual and biological framework for associative learning.

Session II

19:10 – 19:40

Flash Talk 5

Single cell CRISPR engineering and characterization using long-read sequencing

Heonseok Kim, Ph.D.
School of Medicine, Stanford University

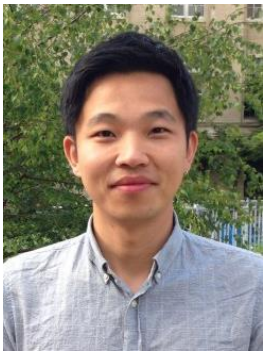
Large scale genomic studies are cataloguing thousands of genetic mutations. With the sheer number of discovered mutations, determining their phenotype and functional characterization remains an enormous challenge. Conventional CRISPR screening have been used to determine the phenotypic effects of genetic mutations by analyzing altered cellular fitness. However, these methods were not able to determine CRISPR perturbed cells' state in detail. Single-cell CRISPR screening enabled transcriptome changes induced by CRISPR engineering. However, they had relied on short-read sequencing which are not able to detect full-length transcripts. Herein, we developed novel single-cell technologies to directly introduce mutations into the human genome and determine their transcriptional phenotype with integrated long- and short-read sequencing among individual cells. These enable introduction of more diverse genome engineering (i.e., point mutations, gene fusions, etc.) and investigation of their effect in depth (i.e., genetic mutations, transcript isoform usage, etc.) in a single-cell resolution. This will generate rich and valuable dataset about complex phenotypes of various genetic mutations which were not attainable before with previous methods.

Session II

19:10 – 19:40

Flash Talk 6

Leveraging genetic tool to manipulate mitochondrial physiology



Mangyu Choe Ph.D.
Department of Nutritional Sciences & Toxicology, UC Berkeley

Metabolism is compartmentalized in subcellular organelles and its regulation is interconnected in a complex manner. To better understand the regulation of metabolism, we need a new generation of tools that allow direct and compartment-specific manipulation of a single metabolite or metabolic pathway/enzyme. Our lab has been interested in developing genetic tools to specifically manipulate a single metabolic pathway in live cells, and we call these tools Genetically encoded tools for the manipulation of metabolism (GEMMs). Our work is mainly focused on bioenergetic parameters which are known as master regulators of metabolism and physiology and are directly involved in the regulation of metabolic enzymes, epigenetic modifiers, ROS production, transcription factors, kinases, and ion channels. Various small molecules have been used to study the role of bioenergetic parameters in metabolism, but compartment- and tissue-specific manipulation of relevant parameters cannot be achieved with chemical drugs.

Here we show that tuning the expression of UCP1, a mitochondrial protein that carries protons across the inner mitochondrial membrane, can be a powerful genetic tool to specifically manipulate mitochondrial membrane potential. UCP1 is only expressed on the mitochondrial membrane and is activated when extracellular oleic acid is available. Unlike FCCP, one of the most common chemical drugs used for dissipating mitochondrial membrane potential, UCP1 specifically decreases mitochondrial membrane potential but not plasma membrane potential and does not affect cell proliferation. We then use UCP1 to demonstrate that increased mitochondrial membrane potential causes oligomycin-driven mitochondrial stress signaling in cells. In summary, we validated UCP1 as a selective reagent for the manipulation of mitochondrial membrane potential and we believe will have a wide range of applications for studying bioenergetics.

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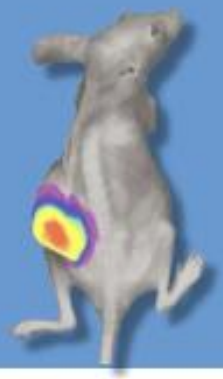

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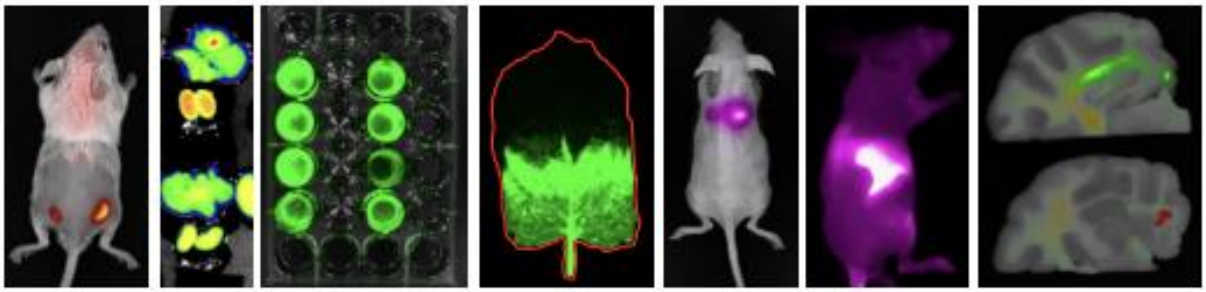

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다가올 100년에는 창업자 유일한 박사의 숭고한 정신을 바탕으로
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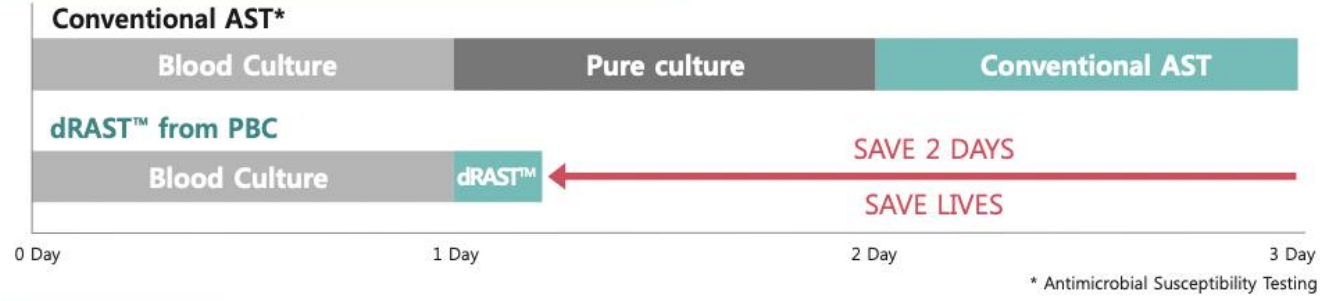
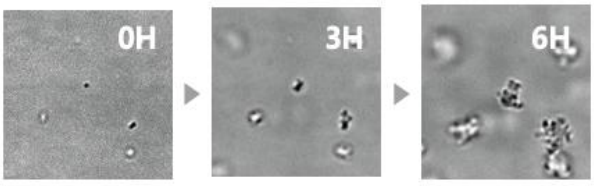


* Antimicrobial Susceptibility Testing

핵심기술

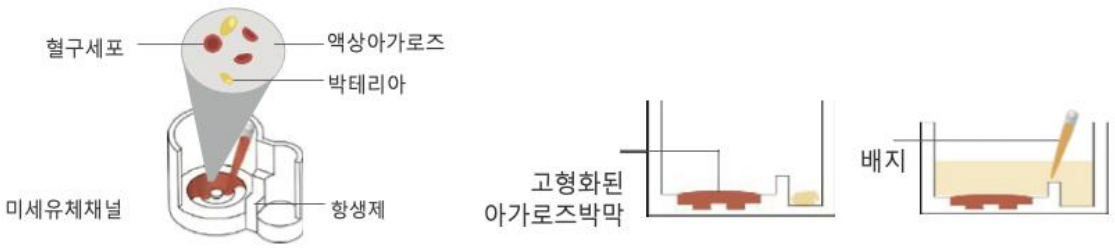
• 이미지 분석 및 알고리즘 기술

- 현미경 이미지로 단일 세포의 분열을 관찰
- 균의 성장 여부를 정확히 판단
- 3천 만개 데이터 포인트에 기반한 AI 알고리즘 분석



• 미세유체 아가로즈 채널 기술

미세유체 아가로즈 채널에 균 고정 후, 세포의 분열 여부를 판단



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Seongnam-si, Gyeonggi-do, South Korea

"제약회사가 할 수 있는 최고의 사회공헌은 신약개발이다"

왜, 동아쏘시오그룹은 수십년 동안 끝없는 암의 비밀을 풀고있는 걸까?

아직도 완치의 희망을 놓지 않는 수많은 암환자와 가족들의 간절함을 알기에 동아쏘시오그룹은 연구실의 불을 끌 수가 없습니다.

암보다도 더 고통스럽다는 항암 치료, 많은 좋은 약이 개발되고 있지만 암은 아직도 풀지 못한 인류의 가장 큰 숙제입니다.
지난 수십 년 동안 신약개발에 앞장서온 동아쏘시오그룹은 생명연장과 완치를 위해 면역항암에 기초한 신약개발에 노력을 기울이고 있습니다.
2016년 애브비와 면역항암제 기술 수출 및 공동연구 계약,
2017년 아스트라제네카와 공동연구개발 계약 등
암 정복에 한발 먼저 다가가고 있습니다.

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