MAY 12th 2012 9AM - 5PM
Columbia University, New York, NY
Havemeyer Hall

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4th New York Korean Biologists

Annual Conference

Albert Einstein College of Medicine
Cold Spring Harbor Laboratory
Columbia University
Cornell University
Memorial Sloan-Kettering Cancer Center
Mount Sinai School of Medicine
New York University
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모시는 글

안녕하십니까.

저희 NYKB (New York Korean Biologists) 는 뉴욕 지역에 위치한 8 개의 학교 및 연구소 (Albert Einstein College of Medicine, Cold Spring Harbor Laboratory, Columbia University, Cornell University, Memorial Sloan-Kettering Cancer Center, Mount Sinai School of Medicine, New York University, Rockefeller University)에서 연구하고 있는 한인 생명과학자들로 구성된 학술 단체입니다. 최근에는 저희 NYKB 의 구성단체 외에도 뉴욕지역과 롱아일랜드 그리고 뉴저지 인근의 많은 한국인 생명과학자들께서도 저희 모임에 적극적으로 참여해 주시고 계십니다. 저희 NYKB 에 소속되어 있는 8 개의 학교 및 연구소에서는 매월 1 회의 세미나를 개최하고 있으며, 이를 저희 홈페이지 (www.nykb.org) 에 사전 공지하여 다른 학교와 연구소의 관심있는 회원도 자유롭게 참여하고 있습니다.

저희는 이러한 1 년간의 활동을 바탕으로 올해 5 월 12 일 (토요일) 제 4 회 NYKB 연례 컨퍼런스를 컬럼비아 대학교에서 개최합니다. 작년 한해 훌륭한 연구를 해오신 뉴욕지역의 교수님, 포스트닥, 학생 위주로 발표연사가 꾸려 졌습니다. 그리고 우수 포스터 2 분에게도 우수 포스터상을 수여할 예정입니다. 저희는 올해 컨퍼런스에서 최근의 우수한 연구 성과 발표를 통해 최신 생명과학 연구동향을 파악하고 회원 상호간의 연구 협력의 장을 더욱 활성화시켜 나가고자 합니다.

저희 NYKB 는 더 나아가서 볼티모어 생명과학자협회 (BLSA), NIH 한인과학자 협회 (NIH-KSA), 뉴잉글랜드 생명과학협회(NEBS), 재미과학기술자협회 (KSEA), 한-미 과학협력센터 (KUSCO)등과 긴밀한 상호 협력 체제를 구축해 나가고 있습니다. 최근에는 한국의 생물학연구정보센터 (BRIC)와 한국분자세포생물학회 (KSMCB), 한국생명공학연구원 (KRIBB) 등과 지속적인 상호협력체제를 구축하여 저희 회원님들께 더 많은 유익한 정보와 연구 네트워크 기회를 제공해 나가고자 노력하고 있습니다.

마지막으로 저희 NYKB 를 후원해 주고 계시는 많은 회원님들과 후원자 여러분, 그리고 이번 컨퍼런스 준비를 위해 진심으로 노고하고 계시는 저희 NYKB 임원진 여러분들께 진심으로 감사의 말씀을 드립니다. 저희 NYKB 에 궁금한 점이 있으시면 언제라도 저희 대표 메일 (nykb2008@gmail.com) 로 연락을 주시기 부탁드리며, 올해 NYKB 컨퍼런스가 전체 NYKB 회원님들간의 훌륭한 교류의 장이 되고, 지난 한해 훌륭한 연구 활동의 중요한 연장선이 될수 있도록 많은 회원님들의 적극적인 참여를 부탁드립니다.

감사합니다.

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CONFERENCE PROGRAM

*Host: **Yongsoo Kim**, Ph.D. (Cold Spring Harbor Laboratory)*

09:00 - 09:20 am	**Registration**
09:20 - 09:25 am	**Welcoming Address** **Yongsoo Kim**, Ph.D. (Cold Spring Harbor Laboratory) Director of Academic Affairs
09:25 – 09:30 am	**Opening Remark**

Morning Session

Chair of Morning Session: Su-Hwan Kwak, Ph.D. (Long Island University)

09:30 - 10:10 am	**Su-Hwan Kwak**, Ph.D. (Long Island University) "To be a hair, or not to be: cell fate determination in Arabidopsis roots"
10:10 - 10:50 am	**Soon Ju Park**, Ph.D. (Cold Spring Harbor Laboratory) "Dynamics of meristem maturation and the evolution of tomato inflorescence architecture"
10:50 - 11:10 am	Coffee Break
11:10 - 11:50 am	**Ja Young Kim-Muller**, Ph.D. (Columbia University) "The functional consequences of acetylation status of FoxO1 in regulating glucose and lipid metabolism"
11:50 - 12:40 pm	Photo & Lunch

Afternoon Session

Chair of Afternoon Session 1: Cheolho Cheong, Ph.D. (Rockefeller University)

12:40 - 01:20 pm **Jun R. Huh**, Ph.D. (New York University)
"Small molecule inhibitors of RORγt: their development to study the function of inflammatory immune cells"

01:20 - 01:50 pm **Sung Ho Park**, Ph.D. candidate (Weill Cornell Medical College)
"Tumor necrosis factor induces GSK3 kinase–mediated cross-tolerance to endotoxin in macrophages"
(Awardee of NYKB student award)

01:50 - 02:50 pm Poster session

Chair of Afternoon Session 2: Daesoo Kim, Ph.D. (KAIST)

02:50 - 03:30 pm **Daesoo Kim**, Ph.D. (KAIST)
"Physiological and Pathophysiological Role of T-type Calcium Channels in the Brain"

03:30 - 04:10 pm **Laibaik Park**, Ph.D. (Cornell University)
"Scavenger receptor CD36 is essential for the cerebrovascular oxidative stress and neurovascular dysfunction induced by amyloid-β"

04:10 - 04:40 pm **Jiwon Choi**, Ph.D. candidate (Columbia University)
"Miniature Neurotransmission Regulates the Structural Maturation of *Drosophila* Synapses"
(Awardee of NYKB student award)

04:40 - 04:50 pm **Excellent Poster Awards**

04:50 - 05:00 pm **Closing Remark & Announcement**
Sae Woong Park, Ph.D. (Cornell University)

Banquet

05:00 - 08:00 pm 700 Conference Room in Fairchild Center

Abstracts of oral presentations

To be a hair, or not to be: cell fate determination in Arabidopsis roots

Su-Hwan Kwak, Ph.D. (Assistant Professor)

Long Island University

The epidermal cell pattern of Arabidopsis roots provides a simple and fascinating model for studying cell fate determination in plants. The epidermal cells of Arabidopsis roots have two distinct fates: root-hair cell and nonhair cell. Binary fates in Arabidopsis root epidermis are determined by lateral inhibition fulfilled by WEREWOLF (WER) and CAPRICE (CPC). Although the lateral inhibition has a critical role in cell fate determination, the position of epidermal cells dictates which epidermal cell would inhibit and which epidermal cell would be inhibited. Developing epidermal cells on two underlying cortical cells preferentially differentiate as root-hair cells (H position), whereas epidermal cells on a single cortical cell adopt the nonhair cell fate (N position). It implies that epidermal cells recognize their position somehow. It has been discovered that SCRAMBLED (SCM), a leucine-rich repeat receptor-like kinase (LRR RLK), enables epidermal cells to interpret their position and adopt appropriate cell fate. After perception of positional cue, it is proposed that SCM represses the expression of *WER* triggering the initial difference in the level of WER between cells in H position and N position. This initial bias is thought to be amplified by lateral inhibition between epidermal cells on different positions. However, the molecular mechanism responsible for this preferential SCM signaling is unknown. We analyze the distribution of the SCM receptor and the biological effect of altering its accumulation pattern. We find that SCM expression and accumulation in the epidermis is necessary and sufficient to generate the cell-type pattern. Further, SCM preferentially accumulates in H cells, and this accumulation pattern is dependent on the downstream transcription factors. Thus, SCM participates in an auto-regulatory feedback loop, enabling cells engaged in SCM signaling to maintain high levels of SCM receptor, which provides a simple mechanism for reinforcing a bias in receptor-mediated signaling to ensure robust pattern formation. Together with the results of previous research on SCM signaling, searching for ligand of SCM receptor and pleiotropic functions of SCM in Arabidopsis development will be addressed as well.

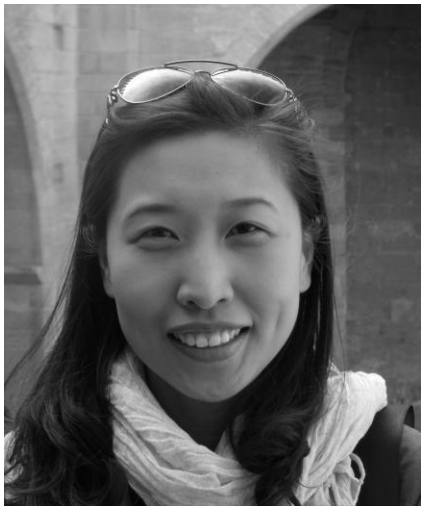
Dynamics of meristem maturation and the evolution of tomato inflorescence architecture

Soon Ju Park, Ph.D.

Cold Spring Harbor Laboratory

In plants such as tomato, new branches are born to replace older branches that terminate in flowers. Differences in the organization of these 'sympodial shoots' produce a remarkable array of inflorescence architectures. Tomato inflorescences range from single sympodial inflorescence in a zigzag pattern in domesticated varieties to two or multi branched sympodial inflorescences with dozens of flowers in related, but genetically incompatible, wild species. To dissect sympodial inflorescence variation, we captured transcriptome dynamics from gradually maturing meristems in branched and unbranched tomato species, as well as the highly branched mutant *compound inflorescence* (*s*). We show that widespread and developmentally coordinated changes in gene expression foreshadow a branching program in *s* that is based on delaying floral termination in two sequentially developing meristems. Applying algorithms to quantify and compare molecular maturation states, we find that wild species, despite sharing identical meristem ontogenies with *s*, are delayed only in the first termination within each developing inflorescence, and consequently experience a prolonged reproductive transition that enables modest branching. In addition, to providing the first gene expression atlas underlying tomato meristem ontogeny, our study exposes a surprisingly early developmental window for sympodial inflorescence branching that was likely adopted by thousands of plants to maximize flower production and boost reproductive success.

The functional consequences of acetylation status of FoxO1 in regulating glucose and lipid metabolism



Ja young Kim-Muller, Ph.D.

Columbia University

FoxO1 integrates multiple metabolic pathways. Nutrient levels modulate FoxO1 acetylation, but the functional consequences of this posttranslational modification are unclear. To answer this question, we generated mice bearing alleles that encode constitutively acetylated and acetylation-defective FoxO1 proteins. Homozygosity for an allele mimicking constitutive acetylation (Foxo1(KQ/KQ)) results in embryonic lethality due to cardiac and angiogenesis defects. In contrast, mice homozygous for a constitutively deacetylated Foxo1 allele (Foxo1(KR/KR)) display a unique metabolic phenotype of impaired insulin action on hepatic glucose metabolism but decreased plasma lipid levels and low respiratory quotient that are consistent with a state of preferential lipid usage. Moreover, Foxo1(KR/KR) mice show a dissociation between weight gain and insulin resistance in predisposing conditions (high fat diet, diabetes, and insulin receptor mutations), possibly due to decreased cytokine production in adipose tissue. Thus, acetylation inactivates FoxO1 during nutrient excess whereas deacetylation selectively potentiates FoxO1 activity, protecting against excessive catabolism during nutrient deprivation.

Small molecule inhibitors of RORγt: their development to study the function of inflammatory immune cells

Jun R. Huh, Ph.D.

New York University

CD4+ T helper lymphocytes that express interleukin-17 or IL-17 (Th17 cells) and innate lymphoid cells (ILCs) are distinguished by expression of the retinoic acid receptor-related orphan nuclear receptor RORγt. These cells have critical roles in mouse models of autoimmunity, and there is mounting evidence that they also influence inflammatory processes in humans. By performing a chemical screen with an insect cell-based reporter system, we recently found that the cardiac glycoside digoxin and its derivatives function as specific inhibitors of RORγt transcriptional activity. Digoxin inhibited Th17 cell differentiation without affecting the differentiation of other T cell lineages and was effective in delaying the onset and reducing the severity of autoimmune disease in mice. We also demonstrated that RORγt is important for the maintenance of IL-17 expression in mouse and human effector T cells. From a large-scale chemical screen with more than 300,000 small molecules, we identified a second series of RORγt inhibitors. One compound (NIHJH-N2) in this series exhibited lower than 100 nM IC50 (the half maximal inhibitory concentration) in an *in vitro* RORγt competition assay. In addition, NIHJH-N2 suppressed human Th17 cell differentiation at sub-micromolar concentrations. To elucidate RORγt function in human cells, we identified downstream RORγt targets by transcriptome deep-sequencing of human Th17 cells following treatment with two different classes RORγt inhibitors, the digoxin derivatives and NIHJH-N2. Since these RORγt inhibitors have distinct chemical structures, genes commonly affected by both inhibitors likely represent true downstream targets rather than the off-target artifacts of one particular compound. Using these highly specific inhibitors, we also demonstrated that RORγt activity is required for human ILCs to produce IL-22.

Tumor necrosis factor induces GSK3 kinase–mediated cross-tolerance to endotoxin in macrophages

Sung Ho Park, Ph.D. candidate
(Awardee of NYKB Student award)

Cornell University

Endotoxin tolerance, a key mechanism for suppressing excessive inflammatory cytokine production, is induced by prior exposure of macrophages to Toll-like receptor (TLR) ligands. Induction of cross-tolerance to endotoxin by endogenous cytokines has not been investigated. Here we show that prior exposure to tumor necrosis factor (TNF) induced a tolerant state in macrophages, with less cytokine production after challenge with lipopolysaccharide (LPS) and protection from LPS-induced death. TNF-induced cross-tolerization was mediated by suppression of LPS-induced signaling and chromatin remodeling. TNF-induced cross-tolerance was dependent on the kinase GSK3, which suppressed chromatin accessibility and promoted rapid termination of signaling via the transcription factor NF-kB by augmenting negative feedback by the signaling inhibitors A20 and IkBa. Our results demonstrate an unexpected homeostatic function for TNF and a GSK3-mediated mechanism for the prevention of prolonged and excessive inflammation.

Physiological and Pathophysiological Role of T-type Calcium Channels in the Brain

Daesu Kim, Ph.D. (Professor)

KAIST

My specific interest has focus on the physiological and pathophysiological role of T-type calcium channels in the brain. During postdoctoral period, I reveal that CaV3.1 T-type channels play a critical role in thalamic "sensory gating" which controls the flow of sensory information influx from sensory world: complete closing of thalamic gate causes a loss of consciousness during absence seizures (Neuron, 2001) and while regulated focal closing of it contributes to novel anti-nociceptive mechanism for visceral pain (Science, 2003). In KAIST as a principal investigator, I am doing the systems neuroscience for motor circuits in the brain. Recently, we revealed the role of CaV3.1 channels in tremorogenesis (PNAS, 2010) in the development of prefrontal dysfunctions in mediodorsal thalamus (J Neuro, 2011). I hope that my research will contribute to the understanding of how complex cognitive functions of brain eventually lead to a specific pattern of behaviors.

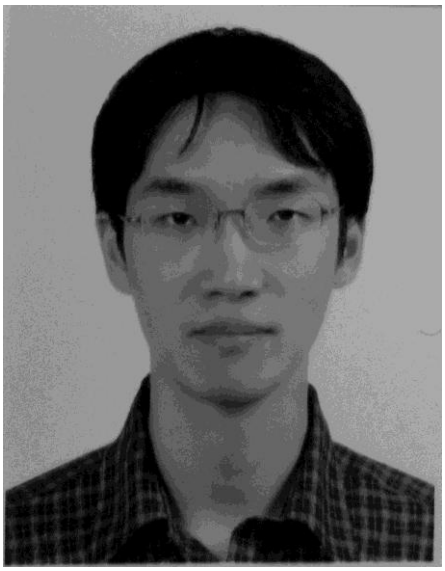
Scavenger receptor CD36 is essential for the cerebrovascular oxidative stress and neurovascular dysfunction induced by amyloid-β

Laibaik Park, Ph.D. (Assistant Professor)

Cornell University

Accumulating evidence suggests that cerebrovascular dysfunction plays a pathogenic role in the Alzheimer's disease (AD). Amyloid-β (Aβ), a peptide central to the pathogenesis of AD, has profound vascular effects mediated, for the large part, by reactive oxygen species produced by the enzyme NADPH oxidase. The mechanisms linking Aβ to NADPH oxidase-dependent vascular oxidative stress have not been identified, yet. We report that the scavenger receptor CD36, a membrane glycoprotein that binds Aβ, is critical for the vascular oxidative stress and neurovascular dysfunction induced by $A\beta_{1-40}$. Hence, superfusion of $A\beta_{1-40}$ onto the somatosensory cortex attenuates the increase in cerebral blood flow elicited by neural activity or by endothelium-dependent vasodilators in WT mice, but not in CD36-null mice ($CD36^{0/0}$). CD36 deficiency prevents the neurovascular dysfunction and behavioral alterations observed in transgenic mice overexpressing the Swedish mutation of the amyloid precursor protein Tg2576 despite elevated levels of brain $A\beta_{1-40}$. In addition, CD36 is required for the vascular oxidative stress induced by exogenous $A\beta_{1-40}$ or observed in Tg2576 mice. These observations establish CD36 as a critical link between $A\beta_{1-40}$ and the NADPH oxidase-dependent cerebrovascular oxidative stress underlying the neurovascular dysfunction and suggest that CD36 is a therapeutical potential to counteract the cerebrovascular dysfunction associated with Aβ.

Miniature Neurotransmission Regulates the Structural Maturation of *Drosophila* Synapses



Jiwon Choi, Ph.D. candidate
(Awardee of NYKB Student award)

Columbia University

In the process of miniature neurotransmission, miniature Excitatory Post-Synaptic Potentials (mEPSPs), often called 'minis', are induced by the spontaneous release of single synaptic vesicles (SVs) from presynaptic neurons. mEPSPs are a general property of every excitatory chemical synapse studied since their discovery over fifty years ago, however the *in vivo* necessity for these small amplitude, low probability events has remained enigmatic. Here we show that miniature neurotransmission is required for the structural growth and maturation of *Drosophila* glutamatergic synaptic terminals while evoked neurotransmission is not. We demonstrate that miniature neurotransmission bi-directionally regulates the cellular process of bouton expansion and genetically interacts with BMP signaling. Our results establish that miniature neurotransmission has an essential role *in vivo* as an instructive trans-synaptic signal critical for synaptic development and plasticity.

Posters list

1. Fgf signaling is required for mutual exclusion but not onset of primitive endoderm program in the mouse blastocyst.
Minjung Kang, Ph.D. candidate, Cornell University

2. Regulation of multiple DNA repair pathways by the Fanconi anemia protein, SLX4.
Yonghwan Kim, Ph.D., The Rockefeller University

3. Nicotinamide Riboside and Its Derivatives Increase SIRT5 Deacetylase Activity.
Dou Yeon Youn, Ph.D. candidate, Cornell University

4. The Role of Elf4 in DNA Damage Response.
Narae Bae, Ph.D. candidate, Cornell University

5. Genome-wide analysis identify molecular regulators of metastasis.
Seongho Ryu, Ph.D., Cornell University

6. Foxo1 Control of Memory CD8+ T cell Differentiation.
Myoungjoo Kim, Ph.D. candidate, Cornell University

7. MicroRNA transcriptome profiles in B-cells and plasma of human centenarians.
Hwa Jin Jung, Ph.D., Albert Einstein College of Medicine

8. Regulation of translation of the serotonin transporter in non serotonergic cells via a RNA binding protein.
Yone Jung Yoon, Ph.D., Cornell University

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