3rd New York Korean Biologists

제 3회 뉴욕 한인 생명과학자

Annual Conference

2011

연례 컨퍼런스

9am-7pm, Mar 19

Columbia University

Davis Auditorium
Schapiro Center 412

Design Jinhee An

NYKB New York Korean Biologists

재미한인과학기술자협회 KSEA Korean-American Scientists and Engineers Association

KUSCO KOREA - U.S. SCIENCE COOPERATION CENTER

BRC Brain Research Center

KGSA Korean Graduate Students Association at Columbia University Since 1967

3rd New York Korean Biologists Annual Conference 2011

Davis Auditorium (Morris Schapiro Center 412)

Columbia University

Saturday, March 19, 2011, 9am- 7pm

Organizers

New York Korean Biologists

Korean-American Scientists and Engineers Association

Korea-U.S. Science Cooperation Center

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 Cold Spring Harbor Laboratory 의 한국인 모임이 저희 NYKB 의 새로운 구성원이 되었으며, 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 3 월 19 일 (토요일) 제 3 회 NYKB 연례 컨퍼런스를 컬럼비아 대학교에서 개최합니다. 작년 한해 훌륭한 연구를 해오신 뉴욕지역의 교수님, 포스트닥, 학생 위주로 발표연사가 꾸려 졌으며, 올해 처음으로 한국분자세포생물학회 (KSMCB) travel grant 수상자 1 분과 NYKB 학생 어워드 1 분도 이번 발표연사에 포함되어 있습니다. 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) 등과 지속적인 상호협력체제를 구축하여 저희 회원님들께 더 많은 유익한 정보와 연구 네트워크 기회를 제공해 나가고자 노력하고 있습니다.

마지막으로 저희 NYKB 를 진심으로 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.

제 2 대 NYKB 회장 김현수 올림

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NYKB Homepage

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CONFERENCE PROGRAM

*Host: **Sung Hyun Kim**, Ph.D. (Weill Cornell Medical College)*

09:00 - 09:20 am	**Registration**
09:20 - 09:25 am	**Welcoming Address** **Sung Hyun Kim**, Ph.D. (Weill Cornell Medical College) Vice-President of NYKB
09:25 – 09:30 am	**Opening Remark** **Chin Ok Lee**, Ph.D. (Rockefeller University) Senior Advisor of NYKB

Morning Session

*Chair of Morning Session: **Yousin Suh**, Ph.D. (Albert Einstein College of Medicine)*

09:30 - 10:10 am	**Tae-Wan Kim**, Ph.D. (Columbia University) "Chemical Modifiers of β-Amyloid Pathology in Alzheimer's Disease"
10:10 - 10:50 am	**Sunghee Cho**,Ph.D. (Weill Cornell Medical College) "Road to Stroke Recovery"
10:50 - 11:00 am	Coffee Break
11:00 - 11:40 am	**Yonghwan Kim**, Ph.D. (Rockefeller University) "Mutations of *SLX4* gene in Fanconi Anemia" **(Awardee of KSMCB travel grant)**
11:40 - 11:50 am	**NYKB Award**
11:50 - 12:50 pm	Lunch

Afternoon Session

Chair of Afternoon Session: ***K. Stephen Suh****, Ph.D. (Hackensack Univ. Medical Center)*

12:50 - 01:30 pm	**Jihye Paik**, Ph.D. (Weill Cornell Medical College) "FOXOs in Cancer and Stem Cells"
01:30 - 02:10 pm	**Miook Cho**, Ph.D. candidate (Albert Einstein College of Medicine) "A molecular Pathway Regulating Stress-Induced Recruitment of CTCF to the SIRT1 Gene Promoter is Genetically Linked to Risk of Myocardial Infarctions in Humans" **(Awardee of NYKB student award)**
02:10 - 03:10 pm	Photo & Poster session
03:10 - 03:50 pm	**Cheolho Cheong**, Ph.D. (Rockefeller University) "Monocyte-derived dendritic cells in vivo"
03:50 - 04:30 pm	**Yoon-Chi Han**, Ph.D. (Memorial Sloan Kettering Cancer Center) "Genetic dissection of the miR-17~92 cluster in mouse development and lymphomagenesis"
04:30 - 04:35 pm	**Excellent Poster Awards**
04:35 - 04:50 pm	**Sponsor Session** **Jong-Deok Kim**, Ph.D. (The Director of KUSCO) **Taek Jin Kwon**, Ph.D. (TG/APS director of KSEA)
04:50 - 05:00 pm	**Closing Remark & Announcement** **Hyun Soo Kim**, Ph.D. (Albert Einstein College of Medicine) President of NYKB

Banquet

05:00 - 08:00 pm	Carleton cafeteria (Mudd Building 4th floor)

Abstracts of oral presentations

Chemical Modifiers of β-Amyloid Pathology in Alzheimer's Disease

Tae-Wan Kim, Ph.D. (Associate Professor)

Department of Pathology and Cell Biology, Columbia University Medical Center, New York, NY 10032

Alzheimer's disease (AD) represents one of the highest unmet needs in human medicine today. Current AD drugs only offer minor symptomatic relief but do not provide any disease-modifying benefits. Aberrant elevation and accumulation of amyloid β-peptide (Aβ) are critically associated with both pathological and behavioral/clinical manifestations of AD. Thus, discovery of drug-like compounds that can effectively and safely reduce the Aβ levels in the brain constitute a promising approach for therapeutic and prophylactic development in AD. Aβ is produced by sequential proteolytic cleavages of the β-amyloid precursor protein (APP) by a set of membrane-bound proteases termed β- and γ-secretases. The majority of the current therapeutic approaches appear to have some limitations due to potential adverse side effects, such as the target-associated toxicity, or inability to counteract the progression of the disease. Thus, there is an unprecedented need for safe and effective therapeutic targets in AD research. In search of new targets, we employed a 'chemical genetics' approach which ventures to identify chemical probes that regulate Aβ levels in the brain via a novel mechanism. This approach requires unbiased screening of small molecule compounds using a cell-based assay system which reliably detects AD-relevant phenotypic changes. Our chemical genetic approaches toward the identification and validation of new druggable target of AD will be discussed.

Road to Stroke Recovery

Sunghee Cho, Ph.D. (Associate Professor)

Department of Neurology/Neuroscience, Weill Cornell Medical College at Burke Medical Research Institute, White Plains, NY 10605

Stroke is the leading causes of adult disability and impose tremendous burden on health care cost in the U.S . In spite of the paucity of treatment strategies in a setting of acute ischemia, increasing evidence suggests that synaptic plasticity and remodeling occur weeks after stroke. Stroke induces unique microenvironments that allow for regenerative events. Understanding the mechanism of restorative responses that occurs long after injury and identification of compounds that promote the process would be clinically relevant. I will discuss current issues raised in the field of stroke, quantity vs quality in the injury size, underlying potential mechanism to enhance synaptic plasticity and functional recovery in stroke.

Mutations of *SLX4* gene in Fanconi Anemia

Yonghwan Kim, Ph.D.
(Awardee of KSMCB travel grant)

Laboratory of Genome Maintenance, The Rockefeller University, New York, NY 10065

Fanconi anemia (FA) is a rare recessive disorder characterized by genome instability, congenital malformations, progressive bone marrow failure, and predisposition to hematologic malignancies and solid tumors. At the cellular level, hypersensitivity to DNA interstrand crosslinks (ICLs) is the defining feature in FA. Mutations in thirteen distinct FA genes have been shown to interfere with the DNA-replication dependent repair of lesions involving crosslinked DNA at stalled replication forks. SLX4 is a multidomain scaffold protein interacting with three distinct nucleases SLX1, XPF-ERCC1, and MUS81-EME1, and has been recently identified as a Holliday junction resolvase. Depletion of SLX4 leads to enhanced sensitivity of the cells to crosslinking agents and camptothecin, a DNA topoisomerase I inhibitor. These findings prompted us to screen for mutations in SLX4 in the families from the International Fanconi Anemia Registry (IFAR) with unassigned FA complementation groups. Here, we report the identification of biallelic *SLX4* mutations in two patients with typical clinical features of FA, such as increased mitomycin C sensitivity and cell cycle arrest at late S and G2 phase and show that the cellular defects in the patients' cells are complemented by wild-type SLX4, demonstrating that biallelic mutations in *SLX4/FANCP* cause a new subtype of Fanconi anemia, FA-P.

FoxOs in Cancer and Stem Cells

Jihye Paik, Ph.D. (Assistant Professor)

Department of Pathology and Lab Medicine, Weill Cornell Medical College, New York, NY 10032

Activation of the PI3K-AKT signalling network is an obligate genetic event in the development of cancer. The mammalian FoxO transcription factor family represents one of several downstream effector arms of PI3K-AKT signalling. In one series of genetic experiments, we sought to define the specific contribution of the FoxOs in mediating the wide-range of neoplastic phenotypes linked to PI3K-AKT activation. Employing both germline and somatic deletion strategies, deletion of one or two FoxO genes was associated with a remarkably modest neoplastic phenotype in the context of advancing age or carcinogen exposure. In contrast, broad somatic deletion of all FoxOs engendered a progressive and penetrant cancer-prone condition characterized by thymic lymphomas and widespread hemangiomas. To understand the biological actions of FoxO in these processes, an integrated approach was implemented, combining in vivo phenotypic filters, comparative transcriptional profiling, in silico and biochemical promoter analysis, and gain-of-function and loss-of-function hypothesis testing. Specifically, in silico promoter analysis uncovered evidence of lineage-specific FoxO signaling as reflected by distinct and non-overlapping sets of direct targets regulated by the FoxOs in different cell types. We also explored the role of FoxOs in the maintenance of tissue stem cells. In hematopoietic stem cells (HSCs), FoxOs play key roles in quiescence regulation, survival and oxidative defense. Pharmacological reduction of ROS levels reverses a progressive decline in HSCs with advancing age and enhances repopulating capacity in competitive repopulation assays. In the CNS, particularly neural stem cells (NSCs), deletion of all three FoxOs reveals a role for the FoxOs in the proliferation, renewal and differentiation of NSCs. Analysis of the FoxO deficient brain shows initial increased brain size and proliferation of neural progenitor cells during the early adult life, followed by precocious decline in the NSC pool and neurogenesis in more mature brains. Integrated transcriptomic, promoter and functional analyses of FoxO deficient NSC identified direct gene targets with known links to the regulation of human brain size and the control of cellular proliferation, fate determination, and oxidative defense. Together, these genetic studies demonstrate that the mammalian FoxOs serve to suppressor cancer and to maintain long-lived cells such as stem cells in advancing age.

A Molecular Pathway Regulating Stress-Induced Recruitment of CTCF to the SIRT1 Gene Promoter is Genetically Linked to Risk of Myocardial Infarctions in Humans

Miook Cho, Ph.D. candidate
(Awardee of NYKB Student award)

Department of Genetics, Albert Einstein College of Medicine, Bronx, NY 10461

Accumulation of DNA damage has been considered an important causal factor in age-related diseases and death. There is strong evidence that genome maintenance is a major longevity assurance pathway because genetic defects in this pathway cause a shorter life span and premature aging in humans and mice. We hypothesize that genetic variation at loci involved in genome maintenance may contribute to individual differences in the rate and severity of aging. We conducted a systematic multidisciplinary study to discover "functional" variants in eighty five candidate genes acting in genome maintenance pathways among 353 Mexican American individuals of San Antonio Longitudinal Study of Aging (SALSA). We established a genetic association between SIRT1 SNPs and risk to myocardial infarction (MI), which was replicated in an independent cohort. SIRT1 is an evolutionarily-conserved protein deacetylase that modulates life span and stress resistance in model organisms. We discovered a functional variant in the SIRT1 promoter associated with MI that has permitted the delineation of a pathway by which genotoxic stress leads to activation of SIRT1 gene expression. This is based on stress-induced removal of the polycomb repressor complex and a histone repression mark (H3K27me3) and binding of CTCF in the SIRT1 promoter, a mechanism that is abrogated in the risk genotype carriers. Our study suggests that regulation of the SIRT1 gene expression in response to genotoxic stress, to which all individuals are constantly exposed in many organ systems, is a component of the resilience program that protects against diseases including myocardial infarction.

Monocyte-derived dendritic cells in vivo

Cheolho Cheong, Ph.D.

Chris Browne Center for Immunology and Immune Diseases, The Rockefeller University, New York, NY 10065

Dendritic cells (DCs), critical antigen-presenting cells for immune control, normally derive from bone marrow precursors distinct from monocytes. It is not yet established if the large reservoir of monocytes can develop into cells with critical features of DCs in vivo. We now show that fully differentiated monocyte-derived DCs (Mo-DCs) develop in mice and DC-SIGN/CD209a marks the cells. Mo-DCs are recruited from blood monocytes into lymph nodes by lipopolysaccharide and live or dead gram-negative bacteria. Mobilization requires TLR4 and its CD14 coreceptor and Trif. When tested for antigen-presenting function, Mo-DCs are as active as classical DCs, including cross-presentation of proteins and live gram-negative bacteria on MHC I in vivo. Fully differentiated Mo-DCs acquire DC morphology and localize to T cell areas via L-selectin and CCR7. Thus the blood monocyte reservoir becomes the dominant presenting cell in response to select microbes, yielding DC-SIGN(+) cells with critical functions of DCs.

Genetic dissection of the miR-17~92 cluster in mouse development and lymphomagenesis

Yoon-Chi Han, Ph.D.

Cancer Biology and Genetics Program, Memorial Sloan-Kettering Cancer Center, New York, NY 10065

MicroRNAs (miRNAs) are small, non-coding RNAs that regulate gene expression and play important roles in development and disease. miR-17~92 is an oncogenic cluster that has been implicated in the pathogenesis of various human cancers, including several types of lymphomas. Interestingly, in vivo studies have also uncovered a role for this cluster in mouse development. The cluster encodes 6 distinct miRNAs that can be grouped into four "seed families" predicted to modulate largely non-overlapping gene sets, suggesting that the various members of the cluster are not functionally equivalent. Consistent with this hypothesis, we have recently demonstrated that miR-19a and miR-19b are the critical oncogenic determinants of miR-17~92 in c-Myc induced-lymphomas. Nonetheless, it is unknown how each miRNAs within the cluster individually and cooperatively contribute towards mammalian development and cancer.

To address these important questions, an "allelic series" of miR-17~92 knock-in mice carrying targeted deletions of individual members of the cluster were generated. These mice will be used to dissect the function of individual miRNAs in the miR-17~92 cluster in mouse development and tumorigenesis. The results of these experiments will greatly advance our understanding of the role of miR-17~92 in normal and pathologic hematopoiesis.

Posters list

1. The Ab - Fibrinogen Interaction as new therapeutic target for the Alzheimer's disease.
Hyung Jin Ahn, Ph.D. Laboratory of Neurobiology and Genetics, The Rockefeller University, New York, NY 10065

2. HSPG Facilitation of Sema-1A-Mediated Repulsion During Drosophila Embryonic Motor Axon Guidance.
Joong Youn Cho, Department of Neuroscience, Howard Hughes Medical Institute, The Johns Hopkins University School of Medicine, Baltimore, MD 21205
(Baltimore Life Scientists Association, BLSA)

3. Miniature Neurotransmission Regulates the Structural Development of Drosophila Synapses.
Ben Jiwon Choi, Ph.D. candidate, Department of Physiology, Columbia University, New York, NY 10032

4. High-throughput, multiplexed microwell assays for characterizing multicellular interactions among individual tumor cells, NK cells or MSCs.
Jonghoon Choi, Ph.D., Department of Chemical Engineering, MIT, Cambridge MA 02139

5. Probing antigen specific responses of human immunological cells on microwell arrays.
Jonghoon Choi, Ph.D., Department of Chemical Engineering, MIT, Cambridge MA 02139

6. Profiling of altered localization of REST in the hippocampal CA1 of rats subjected to global ischemia via ChIP-chip analysis.
Jee-Yeon Hwang, Ph.D., Department of Neuroscience, Albert Einstein College of Medicine, New York, NY 10461

7. System Identification of the DM4 Glomerulus in Drosophila Antennal Lobes.
Anmo J. Kim, Ph.D., Department of Electrical Engineering, Columbia University, New York, NY 10032

8. CD36-deficient bone marrow stem cells transfer reduces stroke-induced brain injury in hyperlipidemic ApoE KO mice.
Eunhee Kim, Ph.D., Department of Neurology/Neuroscience, Weill Cornell Medical College at Burke Medical Research Institute, White Plains, NY 10605

9. A role for spleen CCR2+ monocytes in stroke-induced injury in hyperlipidemic condition.
Eunhee Kim, Ph.D., Department of Neurology/Neuroscience, Weill Cornell Medical College at Burke Medical Research Institute, White Plains, NY 10605

10. Enhancement of myogenic differentiation of VEGF-treated adipose stem cell graft by using thermo sensitive hydrogel.
MiJung Kim, Ph.D. (Visiting Scholar), Department of Genetics, Albert Einstein College of Medicine, New York, NY 10461

11. Tet-off conditional AIMP2 transgenic mice recapitulate progressive dopaminergic neurodegeneration via PARP1 overactivation.
Yunjong Lee, Ph.D. candidate, Department of Cellular and Molecular Physiology, The Johns Hopkins University School of Medicine, Baltimore, MD 21205
(Baltimore Life Scientists Association, BLSA)

12. Genome-wide analysis identify molecular regulators of metastasis
Seongho Ryu, Ph.D., Department of Cardiothoracic Surgery, Weill Cornell Medical College, New York, NY 10065

13. Identification of a conserved vertebrate peptidoglycan receptor
Jin Seo, Ph.D., Department of Microbiology and immunology, Columbia University, New York, NY 10032

14. The effects of manipulating two visual stimuli on firing patterns of entorhinal grid cells.
Eunyoung Song, Ph.D., Department of Physiology and Pharmacology SUNY Downstate Medical Center, Brooklyn, NY 11203

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The Korean Graduate Students Association at Columbia University
(KGSA, www.columbia.edu/cu/kgsa/)

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- Address: **2960 Broadway, New York, NY 10027**
- Bus Lines: **M4**, **M5**, **M11**, **M60**, **M104**
- Subway Line: **#1 Line (Red)**

Conference: **Davis Auditorium** (**Morris Schapiro Center 412**)
Banquet: **Carleton cafeteria** (**Mudd Building 4th floor**)