New York Korean Biologists

2nd Annual Conference 2010

Jerome Greene Hall, Room 101

Columbia University

8:30 AM- 8 PM, March 27. 2010, Saturday

Organizers

New York Korean Biologists

Korean-American Scientists and Engineers Association

Korea-U.S. Science Cooperation Center

개 회 인 사

지난 제 1 회 NYKB Annual Conference 가 여러분의 커다란 관심 속에 성공적으로 마무리되었습니다. 작년 Conference 는 우리 주위에 계신동료 생명과학자분들의 훌륭한 연구들을 접할 수 있는 좋은 기회가 되었고, 또한 후배들을 격려해주시기 위하여 Manhattan 일대에 계신 많은 선배 과학자분들께서도 참석해주셨습니다. 그리고 성공적인 행사를 위해서 KSEA 와 KUSCO 를 비롯한 여러 기업과 단체에서도 아낌없는 후원을 해주셨습니다. Conference 후 NYKB 에 대한 인지도가 높아졌으며, 작년 9 월 1 일에는 NYKB 홈페이지(www.nykb.org)를 개장하여 Manhattan 일대 곳곳에 흩어져 있는 연구기관과 학교들 간의 상호 교류와 소모임 활동의 활성화에도 도움이 되고자 노력하고 있습니다.

작년의 성공을 바탕으로 3 월 27 일 Columbia University 에서 제 2 회 NYKB Annual Conference 를 개최합니다. 이번 conference 에는 Oral presentation Session 과 함께 Poster Session 도 마련하여 능동적으로 의견을 교환할 수 있는 기회를 준비하였습니다. 또한 Special Lecture 에서는 작년 Cornell University 의 조동협 박사님에 이어서 Rockefeller University 의 이진옥 박사님께서 후배 과학자 들을 위하여 소중한 말씀을 해주실 예정입니다. 올해 NYKB Annual Conference 소식은 New Jersey 및 다른 지역의 학교들에도 알렸으며 Manhattan 지역 중심으로 다른 여러 지역에 계신 분들도 참석하실 예정입니다. 이번 Conference 가 앞으로 NYKB Annual Conference 가 미국에 계신 한인생명과학자들을 위한 뜻 깊은 행사로 거듭나는 계기가 되기를 기원합니다. 대단히 감사합니다.

2010 년 3 월 27 일

박희준 올림.

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CONFERENCE SCHEDULE

08:30 - 09:20 AM Registration

09:20 - 09:25 AM Welcoming Address
Heuijoon Park, Ph.D. Candidate (Columbia University)
President, New York Korean Biologists

09:25 – 09:30 AM Opening Remark
Tong Hyup Joh, Ph.D. (Cornell University)
Senior Advisor, New York Korean Biologists

Morning Session

Chair: **Tae-Wan Kim,** Ph.D. (Columbia University)

09:30 - 10:00 AM **Stephen Suh**, Ph.D. (Hackensack University Medical Center)
- Translational biomarker discovery based on clinical outcomes

10:00 - 10:30 AM **Jae-Kyun Ko**, Ph.D. (UMDNJ)
- Single plasmid system for a tissue-specific and inducible gene knockdown using 2nd-generation shRNA

10:30 - 11:00 AM **Ki Bum Lee**, Ph.D. (Rutgers University)
- Nanotechnology approaches for identifying microenvironmental cues regulating stem/cancer cell fate

11:00 - 12:00 PM SPECIAL LECTURE
Chin Ok Lee, Ph.D. (Rockefeller University)
- Function and regulation of Na-K ATPase (Na-K pump): Story of my scientific career

12:00 - 12:30 PM Lunch

12:30 - 01:30 PM Poster Session

Afternoon Session

Chair: **Kyonsoo Hong,** Ph.D. (New York University)

01:30 - 02:00 PM Sponsor Session

02:00 - 02:30 PM **Greg S. Suh**, Ph.D. (New York University)
- CO_2 avoidance behavior is mediated by two distinct populations of olfactory sensory neurons

02:30PM - 03:00 PM Photo Session & Coffee Break

03:00 - 03:30 PM **Ja Wook Koo**, Ph.D. (Mount Sinai School of Medicine)
- Role of BDNF in morphine actions on gene expression and place conditioning

03:30 - 04:00 PM **Sang Ryong Kim**, Ph.D. (Columbia University)
- Protective and Restorative effects of Rheb/mTOR pathway in experimental models of Parkinson's disease

04:00 - 04:20 PM Coffee Break

04:20 - 04:50 PM **Yousin Suh**, Ph.D. (Albert Einstein College of Medicine)
- Genetic variation in genome maintenance and aging

04:50 - 05:20 PM **Eun Young Yu**, Ph.D. (Cornell University)
- Structural and functional analysis of telomeric proteins in *Candida albicans*

05:20 - 05:50 PM Closing Remark & Announcement
Heuijoon Park, Ph.D. Candidate (Columbia University)
President, New York Korean Biologists

06:00 - 08:00 PM Banquet
800 Conference Room in Sherman Fairchild Center
in Biology Department

ORAL PRESENTATION ABSTRACTS

1. Translational Biomarker Discovery Based on Clinical Outcomes

Stephen Suh[1]

[1]Tissue Bank, The Cancer Center, Hackensack University Medical Center, Jurist Research Building, 30 Prospect Avenue, Rm RC-326 and 328; office RC-334, Hackensack, NJ 07601

Developing novel therapies for “personalized” treatment on cancer requires systematic scientific approaches that will lead to identification of robust and stratified biomarkers. First and foremost important, but often neglected, step in biomarker discovery is the biospecimen procurement methods that will consequently provide bench scientists with high quality clinical specimens. A combination of specimens with high integrity and innovative research significantly shortens time in discovery phase. Second, a good set of biomarkers are necessary to generate effective molecular platforms that are highly applicable to diagnostic, prognostic and translational clinical studies for achieving “personalized" chemotherapeutic interventions.

This laboratory has developed a bioinformatics-guided, real time and web-based tissue procurement workflow that ensures maximum and transparent communications between personnel from multiple departments of the hospital, including phlebotomy, surgery and pathology. For a medical center that ranks 4th in US for patient volume and performs over 40,000 surgical procedures per year, high quality tissue procurement is a difficult task. We have constructed a workflow that utilizes information technology and telecommunication departments for automated paging and email alert systems to secure tight communication network throughout the tissue procurement process. For biomarker studies, we have used bioinformatics approach that is substantially different from ‘global screening’ by using microarray or proteomics technologies. High-end data mining and knowledge management software was used to extract putative biomarkers for cancer type of interest in publicly available databases, including literatures. These putative markers were then extensively tested against a library of cancer cell lines by using qRT-PCR methods to identify only robust biomarkers. For the purpose of this lecture, ovarian cancer and lymphoma project will be presented. The final sets of biomarkers were validated with patient samples and associate them with drug responses and clinical outcomes. As an independent approach, retrospective patient samples with specific clinical outcome of interest will be analyzed for genomic and proteomics markers. These biomarkers will be used to preselect the patients with a high probability to respond to the conventional first-line therapies versus the patients that are likely to relapse. We postulate a combination of these approaches would be a good model to identify translational biomarkers to achieve "personalized" therapeutic options to improve response rate and outcome for multiple human cancer types.

2. Single plasmid system for a tissue-specific and inducible gene knockdown using 2nd-generation shRNA

Jae-Kyun Ko[1], Kyoung-Han Choi[1], Noah Weisleder[1] and Jianjie Ma[1]

[1]Department of Physiology and Biophysics, University of Medicine and Dentistry of New Jersey, Robert Wood Johnson Medical School, Piscataway, NJ.

Junctophilins (JPs) play an essential role in muscle excitation-contraction coupling by contributing to the formation of junctional membrane complexes (JMCs). However, the lethality associated with germ-line ablation of JP genes prevents futher evaluation of JPs in adult striated muscles. To investigate the physiological role of JP genes in mouse development and cardiovascular function, we have developed a novel plasmid system for RNA interference by combining three unique approaches: first, shRNA probe containing Dicer and Drosha-RNase processing sites was designed for the target gene; second, transcription of the shRNA probe was controlled in a inducible and reversible manner by the tetracycline-on expression system; and third, Cre-loxP system was used for tissue-specific expression of the shRNA probe. We have tested this plasmid system, named pTLcG-mirJP, in cell cultures, and found efficient silencing of JP expression that was tightly controlled by the Tet-on machinery. Transgenic mice carrying pTLcG-mirJP were produced and mated with mice containing muscle-specific Cre to generate offspring that carry both transgene in a single mouse. Our studies show that this system can be efficiently switched to an active expression cassette for shRNA against JP mRNAs from an inactive cassette in skeletal muscle. While this system tightly regulated leaky expression of shRNA in the absence of doxycycline, it induced robust knockdown of JPs in the skeletal muscle by treatment with doxycycline. Moreover, knockdown of JPs significantly reduced store-operated Ca2+ entry (SOCE) and induced abnormal structures of triad junction in the skeletal muscle, indicating that JPs play an important role in both the regulation of intracellular Ca2+ homeostasis and the formation of JMCs. Biochemical and phenotypic assays indicate that this system is very useful tool with high potential of multiple applications to down-regulate specific target gene in a tissue specific and inducible manner in both *in vitro* and *in vivo* studies.

3. Nanotechnology approaches for identifying microenvironmental cues regulating stem/cancer cell fate

Ki Bum Lee[1]

[1]Dept. of Chemistry & Chemical Biology, Rutgers, The State University of New Jersey 610 Taylor Road, Piscataway, NJ 08854-8087, USA

This talk will focus on the interface of micro-/nano science and cell biology. Even though cell fate (e.g. stem cell differentiation and cancer cell apoptosis) is regulated by interactions with microenvironment cues and intrinsic cellular programs, understanding the functions of microenvironments and manipulating gene expression in stem/cancer cells are hampered by limitations of conventional methods and the lack of extensive knowledge of multiple regulatory signals. If the complex cell behaviors are to be fully investigated, both approaches from nanotechnology—the "top-down" patterning of extracellular matrix (ECM) and signal molecules in combinatorial ways (e.g. ECM compositions, pattern geometry, pattern density and gradient patterns), and the "bottom-up" synthesis of multifunctional nanoparticles and their surface modification with specific signal molecules—should be combined synergistically. To address the aforementioned challenge, our research mainly focuses on three approaches: i) development of combinatorial arrays of microenvironmental signal molecules for investigating cell behaviors; ii) synthesis and utilization of multifunctional nanoparticles as chemotherapeutic reagents against glioblastoma multiforme (GBM); and iii) development of a microfluidic assay platform to identify the optimal conditions for stem cell differentiation and self-renewal.

More specifically, we have applied the combinatorial signal arrays to study the temporal/spatial effect of microenvironmental cues on adhesion, growth, differentiation of functional cells (e.g. neural stem cells and glioblastoma cells). Furthermore, novel synthetic approaches for anti-cancer drugs [e.g. Erlotinib and Histone deacetylase inhibitors (HDAC inhibitors)] and modified siRNA to be linked with nanoparticles have been developed. In parallel research efforts, we have developed a high throughput screening method based on microfluidics to study human embryonic stem cell (hESCs) responses toward multiple microenvironmental cues at the single cell level. In this talk, a summary of the results from these efforts and future directions will be discussed.

SPECIAL LECTURE

4. Function and regulation of Na-K ATPase (Na-K Pump): Story of my scientific career

Chin-Ok Lee[1]

[1]Laboratory of Cardiac/Membrane Physiology, The Rockefeller University, New York, N.Y., U.S.A.

The effects of forskolin, the adenylyl cyclase activator on Na-K pump and Na-Ca exchange current-voltage (I-V) relationships were investigated in isolated guinea pig ventricular myocytes by using wide-tipped, perfused pipettes to record whole cell currents. The steady-state Na-K pump I-V relationship was obtained by subtracting the I-V relationship determined in the presence of 0.5 mM strophanthidin to inhibit the pump from that determined just before application of stropanthidin. The steady-state Na-Ca exchange outward I-V relationship was obtained by subtracting the I-V relationship determined at 0 mM $[Ca^{++}]_o$ from that determined at 2 mM $[Ca^{++}]_o$. Forskolin increased the Na-K pump current (by roughly 30%) over the entire membrane voltage range examined from -100 mV to +30 mV. The voltage dependence of Na-K pump current in the presence of forskolin was identical to that of Na-K pump current in the absence of forskolin. The increase (by roughly 60% at +20 mV) of Na-Ca exchange current by forskolin became more pronounced as membrane voltage was depolarized from -100 mV to +100 mV. The voltage dependence of Na-Ca exchange current was altered in the presence of forskolin. The results indicate that the increase of Na-K pump current measured in forskolin is voltage-independent whereas the increase of Na-Ca exchange current measured in forskolin seems voltage-dependent. A simple interpretation of this difference is that forskolin action results in an increase in the number of active Na/K pumps without any change in their functional characteristics, whereas forskolin action somehow alters the functional characteristics of already active Na/Ca exchangers. Whether the latter effect of forskolin is direct or indirect (perhaps reflecting altered local concentration of activating ions, e.g. Ca^{2+}) remains to be determined. (NIH HL36783)

5. CO_2 avoidance behavior is mediated by two distinct populations of olfactory sensory neurons

Minrong Ai[1], Soo-hong Min[1], Yael Grosjean[2], Rati Bell[2], Richard Benton[2], **Greg S. Suh**[1]

[1]Molecular Neurobiology Program, Skirball Institute, NYU School of Medicine, New York, NY. [2]Center for Integrative Genomics, University of Lausanne, CH-1015, Lausanne, Switzerland

How specific sensory stimuli evoke defined behaviors is a fundamental problem in neurobiology. Most odorants elicit attraction or avoidance depending on their concentrations and identity, as well as the nature of neural circuits they activate. Carbon dioxide (CO_2), in contrast, elicits avoidance over a wide range of concentrations in the fly, *Drosophila melanogaster*. With a dissected fly brain preparation and calcium imaging technique, we previously identified a population of olfactory sensory neurons (OSNs) expressing Gr21a/Gr63a receptors, which mediates detection of and avoidance to low concentrations of CO_2. However, in a new live fly preparation that allows imaging analysis of previously inaccessible antennal nerves, we were able to identify an additional population of OSNs activated by CO_2. Genetic silencing experiments showed that these OSNs, together with Gr21a/Gr63a expressing OSNs, are required for avoidance to high concentrations of CO_2. Our results reveal the existence of a combinatorial mechanism for CO_2 detection and behavioral response. Tracing the projection of these two distinct neural circuits into the brain may identify a center encoding avoidance behavior in general.

6. Role of BDNF in morphine actions on gene expression and place conditioning

Ja Wook Koo[1], Quincey LaPlant[1], Michelle S. Mazei-Robison[1], Deveroux Ferguson[1], David M. Dietz[1], Mary Kay Lobo[1], Scott J. Russo[1], Matthew B. Wilkinson[1], and Eric J. Nestler[1]

[1]Department of Neuroscience, Mount Sinai School of Medicine, New York, NY

Repeated exposure to opiates produces long-term adaptive biochemical and morphological alterations in the mesolimbic dopamine system, which comprises dopaminergic neurons in the ventral tegmental area (VTA) and their projections to the nucleus accumbens (NAc) and other forebrain structures, and these changes are believed to contribute to the addiction-related behavioral phenotypes. Our laboratory has shown that intra-VTA infusions of brain-derived neurotrophic factor (BDNF) prevent or reverse the size reduction of VTA dopamine neurons induced by chronic morphine administration and that specific down-streams of BDNF signaling pathway such as IRS2 and AKT in the VTA mediate the morphological and behavioral responses to morphine. To further understand how BDNF is epigenetically regulated in the VTA by morphine treatment, we used a chromatin immunoprecipitation assay and found that chronic morphine treatment decreases in the amount of the trimethyl modification on lysine 4 of histone 3 at the *Bdnf* promoters P1, P2, and P4, as well as its functional receptor (TrkB) gene *Ntrk2*. To investigate the role of VTA BDNF signaling in genome-wide change in gene expression in the NAc, we performed microarray analysis with NAc tissues from floxed BDNF mice in which VTA BDNF was knockdowned via localized viral-mediated Cre recombinase expression. We identified clusters of genes that are regulated by morphine, but of which expression patterns are vanished by knockdown of VTA BDNF (e.g., *Ptpn2*). In contrast, some other genes are not regulated by morphine but knockdown of VTA BDNF reveals the expression changes by morphine (e.g., *Zbtb16*). However, some genes display common regulation regardless of BDNF knockdown (e.g., *Camk1g*). Further behavioral study revealed that localized BDNF or TrkB knockdown in the VTA enhances morphine reward in place conditioning, suggesting that VTA BDNF signaling may have an antagonizing effect to behavioral actions of morphine. These findings implicate the BDNF signaling as a critical regulator of morphine addiction in transcriptional, epigenetical, behavioral, as well as cellular levels. Supported by NIDA.

7. Protective and Restorative effects of Rheb/mTOR pathway in experimental models of Parkinson's disease

Sang Ryong Kim[1], Tatyana Kareva[1], Nikolai Kholodilov[1], Robert E. Burke[1,2]

[1]Department of Neurology, The College of Physicians and Surgeons, Columbia University, 650W 168th St. BB-307, New York, NY10032 [2]Departments of Pathology, The College of Physicians and Surgeons, Columbia University, New York, New York 10032

Rheb is a member of the Ras family of small GTP-binding proteins, expressed at relatively high levels in the brain, and inducible by growth factor stimulation. Rheb plays a crucial role in the activation of mammalian target of rapamycin (mTOR), a serine/threonine kinase that is involved in the activation of protein synthesis and growth, and in neuron survival. However, little is known of the role of Rheb in dopamine (DA) neurons in the brain in vivo. To determine whether activation of Rheb signaling can induce trophic effects on DA neurons, we constructed adeno-associated virus (AAV) vectors packaging a wild type human Rheb (WT-Rheb) or constitutively active mutants carrying a single amino acid change (N153T or S16H). Each of the vectors was injected into the substantia nigra (SN) of wild type adult mice. Mice were sacrificed four weeks later, and brain sections were stained for tyrosine hydroxylase (TH), neuron-specific nuclear protein (NeuN), and the dopamine transporter (DAT). WT-Rheb did not show any significant effect on DA neurons. However, N153T- and S16H-Rheb induced a 19% and 35% increase in DA neuron size in the SN pars compacta, respectively. In addition, S16H-Rheb induced a 42% and 14% increase in general neuron size in the SN reticulata and SN volume, respectively. S16H-Rheb also induced an increased density of TH- (35%) and DAT-immunopositive fibers (22%) in the ipsilateral striatum. In addition to these trophic effects on DA neurons, our results demonstrate that S16H-Rheb could induce neuroprotective and restorative effects in the unilateral intrastriatal 6-hydroxydopamine (6-OHDA) lesion model of Parkinson's disease (PD). Our findings suggest that regulation of Rheb expression in the brain could be a useful approach to the DA system in PD.

8. Genetic variation in genome maintenance and aging

Miook Cho[1], Xiaoyuan Song[2], Jeehae Han[1], Michael G. Rosenfeld[2], and **Yousin Suh**[1 3*].

[1]Department of Genetics, [3]Department of Medicine, Albert Einstein College of Medicine, 1301 Morris Park Avenue, Bronx, NY 1046; [b]Institute for Aging Research, Diabetes Research and Training Center, Albert Einstein College of Medicine, Bronx, NY 1046 [2] HHMI, Dept. of Medicine, University of California, San Diego School of Medicine, 9500 Gilman Dr., La Jolla, CA 92093-0651 * yousin.suh@aecom.yu.edu

Aging is a major risk factor for the most common human diseases and one of the most complex phenotypes that we know. The identification of genetic variation and their potential functional impact on aging-related phenotypes will be important in assessing genetic components of aging, including exceptionally healthy aging, ultimately contributing to our understanding of functional diversity in aging human populations. We hypothesize that genetic variation at loci involved in genome maintenance can be related to individual differences in the rate and severity of aging. We are conducting a systematic multidisciplinary study to discover "functional gene SNP haplotypes", i.e., allelic variation caused by multiple SNPs in the same gene, among over a hundred candidate genes acting in genome maintenance pathways. These candidate genes include all DNA repair genes in which heritable mutations have been found associated with accelerated aging in humans or mice as well as genes interacting with these key genes and other genes acting in the same pathway. To ascertain the functional relevance of observed positive associations, candidate gene-SNP haplotypes are screened for various parameters of cellular fitness in short-term cell culture studies. Functionally relevant gene variants will then be further studied for their *in vivo* effect during aging by modeling them in mouse. In this symposium, we will present results on identification of functional gene variants in the human *Sirt1* promoter associated with myocardial infarction and delineation of a novel molecular pathway by which genotoxic stress activates *Sirt1* gene expression. We will provide evidence how defects in this response caused by genetic variation leads to increased susceptibility to myocardial infarction, a major aging-related disease.

9. Structural and functional analysis of telomeric proteins in *Candida albicans*

Eun Young Yu[1], Jia Sun[2], Wei-Feng Yen[1], Laura A. Confer[2], Stephen Sun[2], Ming Lei[2], and Neal F. Lue[1].

[1]Department of Microbiology and Immunology, Weill Medical College of Cornell University, New York, NY; [2]Department of Biological Chemistry, University of Michigan Medical School, Ann Arbor, MI

The telomere maintenance machinery appears to have undergone rapid evolutionary divergence in the *Saccharomycotina* subphylum of budding yeast. In particular, the telomere repeat sequence in this group of yeast is extremely variable. To gain further understanding of how such variability could have been generated and maintained, we performed molecular genetic and biochemical analysis of telomere proteins in *Candida albicans*.

In stark contrast to the irregular, G-rich telomeres of the well-studied *S. cerevisiae*, the *C. albicans* telomere repeat unit is unusually long, regular, and non G-rich. However, despite the telomere repeat sequence divergence, homologues of the *S. cerevisiae* telomere binding proteins can be readily identified in the *C. albicans* genome database. These homologues include components of the Cdc13-Stn1-Ten1 (CST) complex, which is thought to protect the telomeric 3' overhang, as well as Rap1, which is thought to bind double stranded telomeres. Attempts to generate a *cdc13* null mutant were unsuccessful, suggesting that this gene is essential in *C. albicans*. In contrast, *stn1*, *ten1* and *rap1* null mutants can be readily constructed. Consistent with similar roles in telomere regulation, all three null mutants were found to possess extremely long telomeres and high levels of t-circles. However, in contrast to several *S. cerevisiae CST* mutants, the accumulation of G-tails was not observed in the *C. albicans stn1, ten1,* and *rap1* mutants. The drastic increase in telomere length in the *ten1* mutant was dependent on both telomerase (*TERT*) and recombination (*RAD50*) genes, indicating that both pathways of telomere elongation are abnormally active in the mutant. Biochemical analysis revealed high affinity and sequence specific recognition of *Candida* telomere repeat sequences by *C. tropicalis* Cdc13 and *C. albicans* Rap1, indicating that these proteins are likely to regulate telomere structure and function through direct binding. We conclude that in *C. albicans*, Rap1 and the CST complex are both required to suppress excessive telomerase activity and recombination. In addition, the OB fold of Cdc13 and the MYB domain of Rap1 are flexible scaffolds that can readily evolve diverse DNA binding specificities.

To elucidate the structural basis of Stn1 and Ten1 function, we solved the crystal structure of a Stn1-N/Ten1 complex from the closely related *Candida tropicalis*. As predicted from earlier bioinformatic analysis, these proteins resemble RPA32 and14 and mediate mutual binding primarily through their C-terminal alpha helices. In support of the critical importance of this interaction, we found that multiple point mutations in residues located at the Stn1-Ten1 interface caused severe loss of function in *vivo*. These findings provide direct confirmation of the hypothesized similarity between the RPA and CST complex.

POSTER LIST

1. **2D encoding of concentration and concentration gradient in Drosophila ORNs**

Anmo J. Kim[1], Aurel A. Lazar[1], Yevgeniy Slutskiy[1]
[1]Department of Electrical Engineering, Columbia University, New York, New York 10025

2. **Simulation-based Perturbation Studies: Genome-Wide Cause and Effect predictions of mRNA Expression under Perturbation**

In Sock Jang[1,2], Andrea Califano[2-4]
[1]Department of Electrical Engineering, Columbia University, New York, New York 10025; [2]Center for Computational Biology and Bioinformatics; [3]Department of Biomedical Informatics; [4]Institute for Cancer Genetics, Columbia University, New York, New York 10032

3. **Lineage and Birth Date Specify Motor Neuron Targeting and Dendritic Architecture in Adult *Drosophila***

Myungin Baek[1], Cesar Mendes[2], and Richard S. Mann[2]
Departments of [1]Biological Sciences and [2]Biochemistry and Molecular Biophysics, Columbia University, New York, New York 10032

4. **A mouse model for gene-environment interaction in the holoprosencephaly spectrum: synergy between loss of *Cdo* and fetal alcohol exposure**

Mingi Hong[1] and Robert S. Krauss[1]
[1]Department of Developmental and Regenerative Biology, Mount Sinai School of Medicine, New York, NY 10029

5. **The role of XMAP215, a microtubule binding protein on controlling cell shape by regulating microtubule dynamics in fission yeast**

Hwajin Kim[1], Fred Chang[1]
[1] Departments of Microbiology, Columbia University, New York, New York 10032

6. **Nicotinamide riboside protects cells from MMS genotoxicity by increasing NAD+ levels in mammalian cells**

Dou Yeon Youn[1], Anthony Sauve[1]
[1]Department of Pharmacology, Weill Cornell Medical School, New York, New York, 10065

7. **Group II metabotropic glutamate receptor stimulation triggers production and release of Alzheimer's amyloid beta 42 from isolated intact nerve terminals**

Soong Ho Kim[1], Paul E. Fraser[4], David Westaway[5], Peter H. St George-Hyslop[4,6], Michelle E. Ehrlich [1,3,] Sam Gandy [1,2,7]
[1]Neurology, [2]Psychiatry, [3]Pediatrics, Mount Sinai School of Medicine, New York, NY 10029; [4]Center for Research in Neurodegenerative Diseases, Departments of Medical Biophysics and Medicine (Neurology), University of Toronto, Toronto, ON, Canada; [5]Center for Prions and Protein Folding Diseases, University of Alberta, Edmonton, AB, Canada; [6]Clinical Neurosciences, University of Cambridge, Cambridge, United Kingdom; [7]James J. Peters VA Medical Center, Bronx, NY 10468

8. **HDAC1 nuclear export induced by pathological conditions is essential for the onset of axonal damage**

Jin Young Kim[1,2], Siming Shen[1], Karen Dietz[2], Ye He[2], Owain Howell[3], Richard Reynolds[3] & Patrizia Casaccia[1,2]
[1] Graduate Program in Neuroscience at the Graduate School of Biomedical Sciences, University of Medicine and Dentistry of New Jersey-Robert Wood Johnson Medical School, 675 Hoes Lane, Piscataway, NJ 08854, [2] Department of Neuroscience and Genetics & Genomics, Mount Sinai School of Medicine. One Gustave Levy Place. Box 1065. New York NY 10029, [3] Department of Cellular and Molecular Neuroscience, Division of Neuroscience and Mental Health Imperial College Faculty of Medicine, Charing Cross Hospital Campus, London, UK

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B. Banquet: 800 Conference Room in Sherman Fairchild Center in Biology Department
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