

2015 **KASBP** Fall Symposium

Oct. 30-31, 2015

**Sheraton Edison Hotel Raritan Center
Edison, NJ 08837**

Hosted by

KASBP

 **대웅제약**


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녹십자

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1967년 설립 이래, 모두가 가고 싶어 하는 편한 길을 선택하기보다 힘들지만
누군가 반드시 가야할 길을 외면하지 않고 묵묵히 걸어온 녹십자가 이제
대한민국을 넘어 세계인의 건강을 지키는 새로운 희망으로 도약하고 있습니다.
이미 세계적으로 인정받은 백신과 혈액제제 등을 개발한 탁월한 R&D 역량을 바탕으로 세포배양,
유전자재조합 등의 차원 높은 솔루션을 더해 세계로 나아가는 녹십자, 만들기는 힘들지만
꼭 필요한 약품으로 세계인의 건강과 행복을 지키는 믿음직한 이름이 되겠습니다.

건강 산업의 글로벌 리더 - 녹십자

Invitation Letter from KASBP Fall Symposium Organizing Committee

Korean American Society in Biotech and Pharmaceuticals (KASBP) cordially invites all members and professionals to the 2015 KASBP Fall Symposium, hosted by KASBP, Daewoong, Green Cross and KUSCO. Following the success of previous symposiums, this symposium continues to focus on current trends and issues in drug discovery and development.

The symposium organizing committee is delighted to announce the recruitment of outstanding speakers and panels. Invited speakers will share their experience and expertise with the attendees regarding cutting-edge science, early discovery to clinical science and biopharmaceuticals with further details shown in agenda.

This year, KASBP delightfully announces Dr. Jong Sung Koh, CTO of Genosco as 2015 KASBP-Daewoong Achievement Award recipient based on his contribution to drug discovery and development. Also, KASBP will present KASBP Recognition Award to Dr. Jong Wook Lee, Vice Chairman and CEO of Daewoong to recognize his significant and steady contribution and commitment to the collaboration with KASBP. As another meaningful event, the KASBP-Daewoong,-Green Cross and KASBP fellowship awards will be presented to young scholars such as graduate students and post-docs who exhibit excellence in their research.

This symposium also provides an opportunity for members to establish professional networks, and share information and experience in the pursuit of excellence in research and development.

The symposium organizing committee is looking forward to meeting all members and participants associated with pharmaceutical and biotech industry as well as academics.

2015 KASBP Fall Symposium Organizing Committee

Symposium Schedule at a Glance

October 30 (Fri), 2015

03:30 pm –	Job Fair
05:00 pm – 06:00 pm	Registration
06:00 pm – 06:20 pm	Opening & Congratulatory Remarks
06:20 pm – 07:20 pm	Dinner
07:20 pm – 07:30 pm	Award Ceremony
	<ul style="list-style-type: none"> • KASBP Recognition Award: Jong Wook Lee • KASBP-Daewoong Achievement Award: Jong Sung Koh
07:30 pm – 07:50 pm	Keynote Speech: Jong Wook Lee
07:50 pm – 08:40 pm	Keynote Lecture: Jong Sung Koh
08:40 pm – 09:00 pm	Sponsor Presentation: Green Cross
09:00 pm – 11:00 pm	Networking
09:30 pm – 10:30 pm	Round Table Discussion (Pharma industry - Academia)

October 31 (Sat), 2015

07:30 am – 08:30 am	Registration & Breakfast
08:30 am – 08:35 am	Opening Remarks
08:35 am – 09:45 am	Session A Presentations
09:45 am – 10:00 am	Sponsor Presentation: ST Pharm
10:00 am – 10:20 am	Coffee Break
10:20 am – 11:30 am	Session B Presentations
11:30 am – 12:10 pm	Fellowship Award Presentations
12:10 pm – 12:20 pm	Photo Time
12:20 pm – 02:30 pm	Lunch & Poster Presentations
02:30 pm – 03:55 pm	Session C Presentations
03:55 pm – 04:15 pm	Coffee Break
04:15 pm – 05:25 pm	Session D Presentations
05:25 pm – 05:35 pm	Closing Remarks
06:00 pm – 09:00 pm	Dinner

Symposium Schedule in Detail

October 30 (Fri), 2015

Job Fair 3:30 pm ~ Organizer: *DongWeon Song, Novartis*

Registration

5:00 pm ~ 6:00 pm

Coordinators: *Jun Hyuk Heo (Merck), Sahee Kim (RevHealth), Dahea You (Rutgers University)*

Opening & Congratulatory Remarks

6:00 pm ~ 6:20 pm

Moderator: *KASBP President-Designated: Yun Choe, Lucas & Mercanti*

Opening Remarks

KASBP President: *Jae Uk Jeong, GlaxoSmithKline*

Congratulatory Remarks

Daewoong Vice Chairman and CEO: *Jong Wook Lee*

Mogam Institute President: *Senyon (Teddy) Choe*

KHIDI USA Director General: *Jung Hoon Woo*

KOTRA NY Executive Director: *Su Jung Lee*

Dinner

6:20 pm ~ 7:20 pm

Award Ceremony

7:20 pm ~ 7:30 pm

KASBP President: *Jae Uk Jeong, GlaxoSmithKline*

KASBP Award Committee: *Young-Choon Moon, PTC Therapeutics*

- KASBP Recognition Award: *Jong Wook Lee, Daewoong*
- KASBP-Daewoong Achievement Award: *Jong Sung Koh, Genosco*

Keynote Speech

7:30 pm ~ 7:50 pm

Jong Wook Lee, Ph.D., Vice Chairman and CEO, Daewoong

“인생의 꿈과 신약개발 (Dream in my life and drug development)”

Keynote Lecture

7:50 pm ~ 8:40 pm

Jong Sung Koh, Ph.D., CTO, Genosco

“Joyful journeys to create, add and promote value as a new drug discoverer”

Sponsor Presentation

8:40 pm ~ 9:00 pm

Introduction to Green Cross R&D

Senyon (Teddy) Choe, Ph.D., President, Mogam Institute

Networking

9:00 pm ~ 11:00 pm

Round Table Discussion: Pharma industry - Academia

9:30 pm ~ 10:30 pm

Moderator: *Stephen Suh, Hackensack University Medical Center*

October 31 (Sat), 2015

Registration & Breakfast

7:30 am ~ 8:30 am

Opening Remarks

8:30 am ~ 8:35 am

KASBP 1st Vice President: *Chang-Sun Lee, PTC Therapeutics*

Session A-----Chair: *Sung-Kwon Kim, Alexion*

8:35 am ~ 9:45 am

- **A-1: “Opportunities and challenges in immunological diseases”**
Hyun-Hee Lee, Ph.D., Merck

- **A-2: “Small molecule inhibitors of ROR γ t: their development to study the function of inflammatory immune cells”**
Jun Huh, Ph.D., University of Massachusetts Medical School

Sponsor Presentation

9: 45 am ~ 10:00 am

Introduction to ST-Pharm

Kyung-Jin Kim, Ph.D., Vice President, ST Pharm

Coffee Break

10:00 am ~ 10:20 am

Session B -----Chair: Sang Tae Park, Macrogen Clinical Laboratory

10:20 am ~ 11:30 am

- **B-1: “The role of clinical pharmacology in oncology drug development”**
Eunju Hurh, Ph.D., Novartis

- **B-2: “Oncogene addiction and the development of targeted cancer therapy”**
Andrew Joe, M.D., Merck

Fellowship Award Ceremony & Presentations

11:30 am ~ 12:10 pm-----Chair: *Eunsung Junn, Rutgers University*

Photo time

12:10 pm ~ 12:20 pm

Lunch & Poster Presentations

12:20 pm ~ 2:30 pm

Session C -----Chair: Sean Kim, Novartis

2:30 pm ~ 3:55 pm

- **C-1: “Oncology for the 21st century”**
Geoffrey Kim, M.D., FDA

- **C-2: “Translational and clinical cancer research in Korea vs USA: Pros and Cons”**
Kyu-Pyo Kim, M.D., Asan Medical Center

- **C-3: “Directives to support biopharmaceuticals' advance to global market”**

Nam Soo Kim, Deputy Director, Ministry of Food and Drug Safety

Coffee Break

3:55 pm ~ 4:15 pm

Session D -----Chair: *KernHee Chang, GlaxoSmithKline*

4:15 pm ~ 5:25 pm

- **D-1: “Biopharmaceuticals producing platforms: developments, optimizations and challenges”**

Yong Hwan Jin, Ph.D., GlaxoSmithKline

- **D-2: “New and enabling technologies for accelerated process development of biologics”**

Jun Hyuk Heo, Merck

Closing Remarks

5:25 pm ~ 5:35 pm

KASBP President: *Jae Uk Jeong, GlaxoSmithKline*

Dinner

6:00 pm ~ 9:00 pm

Ichiumi Restaurant, Menlo Park Mall (tel. 732.906.2370)

352 Menlo Park Dr., Edison, NJ 08837

Abstracts

Joyful journeys to create, add and promote value as a new drug discoverer

Jong Sung Koh, Ph.D., Chief Technology Officer, Genosco



New drug discovery and development is like a marathon that requires endurance and commitment. Success in making new medicine requires immense resources and a deeper understanding of rigorous R&D process. It also takes persistence and sometimes, luck. Ultimately, though, discovery of new medicine brings hope and relief to millions of patients. In that sense, the process of drug discovery is a joyful journey if you enjoy the scientific process of making a new medicine. However, there are also often many dead ends and setbacks. To overcome the agonies in the dead ends and setbacks, drug discoverer needs high adversity quotient (AQ) to turn obstacles into opportunities. Over 25 years, I have been involved in new drug discovery in the areas of anti-HIV, anti-diabetes, anti-cancers, and rheumatoid arthritis. This talk presents my journey as a drug discoverer and immense satisfaction it has given me over the years.

The audacious journey from Daeduk Science town to Cambridge is also presented.

2008 ~ present CTO, Genosco

2007 ~ 2008 Head, Korea Research Institute of Chemical Technology (Anticancer)

2005 ~ 2007 Managing Director, Vice President, LG Life Sciences, Ltd.

2001 ~ 2005 Vice President, Drug Discovery, LG Life Sciences, Ltd. (Antidiabetes)

1991 ~ 2000 Group Leader, LG Chem, Ltd. (Anticancer & HTS)

1990 ~ 1991 Senior Research Associate, The Scripps Research Institute

Opportunities and challenges in immunological diseases

Hyun-Hee Lee, Ph.D., Merck

The immune system is orchestrated by many cells and protein factors involved in physiological and pathological processes. Controlled inflammation would be beneficial (e.g., Infection) but could be damaging if uncontrolled such as in rheumatoid arthritis and asthma. The causes for such diseases are not well elucidated and clinically heterogeneous, which makes it difficult to identify specific targets and treatment for the diseases. Therefore, it is critical to find the key pathways and targets through the investigation on disease mechanism utilizing translational animal models or human tissue and cell based platform to validate and optimize the inflammatory disease targets.

Small molecule inhibitors of ROR γ t: their development to study the function of inflammatory immune cells

Jun R. Huh, Ph.D., University of Massachusetts Medical School

CD4⁺ T helper lymphocytes that secrete interleukin-17 (Th17 cells) and innate lymphoid cells are distinguished by expression of the retinoic acid receptor-related orphan nuclear receptor ROR γ t. 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 previously identified and reported that the cardiac glycoside digoxin and its derivatives function as specific inhibitors of ROR γ t transcriptional activity. Digoxin inhibited Th17 cell differentiation without affecting 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 ROR γ t is important for the maintenance of IL-17 expression in mouse and human effector T cells. From a large-scale chemical screen covering more than 300,000 small molecules, we identified a second series of ROR γ t inhibitors. One compound (ML209) in this series exhibited lower than 100 nM IC₅₀ in an *in vitro* ROR γ t competition assay. In addition, ML209 suppressed human Th17 cell differentiation at sub-micromolar concentrations. In order to elucidate ROR γ t function in human cells by identifying the downstream targets of ROR γ t, deep-sequencing analyses were performed. We analyzed the gene expression profiles of human Th17 cells following treatment with two different ROR γ t inhibitors, the digoxin derivatives and ML209. Using these highly specific inhibitors, we have identified novel downstream targets of ROR γ t that play critical roles in human Th17 cells.

The role of clinical pharmacology in oncology drug development

Eunju Hurh, Ph.D., Novartis

Clinical development of new oncology drugs start with phase 1 dose-escalation studies in patients with advanced cancer as single agent or in combination, frequently with an expansion cohort in the target patient population. Dose-ranging studies, often as part of a phase 1 study for acceleration, evaluate 2 or more dose levels or regimens in order to select the optimal dose and regimen for subsequent pivotal study. Population pharmacokinetic-pharmacodynamic modeling aids in the selection of the optimal dose and regimen besides statistical analysis of dose-response and exposure-response relationships. At the conclusion of the pivotal study, similar tools are employed to justify the recommended therapeutic dose.

Various clinical pharmacology studies are conducted during clinical development of oncology drugs. They are food effect study for oral drugs and mass-balance, drug interaction, organ impairment, bioavailability/bioequivalence, and thorough or dedicated QTc study. These studies are common across therapeutic areas although the studies required for registering oncology drugs are typically limited in scope compared to those required for drugs treating non-life threatening diseases. Certain clinical pharmacology characteristics may be evaluated via population pharmacokinetic modeling rather than in a dedicated clinical study.

These studies and analyses provide important information for safe and effective use of drugs in special populations and in the presence of concomitant medications. The need for dose adjustments for these special circumstances is determined based on the outcome of individual clinical pharmacology studies and exposure-response relationships for efficacy and key adverse effects. Clinical pharmacology program ultimately aims to maximize benefit-risk ratio for a new drug by understanding the factors influencing drug exposure and minimizing exposure variability within the target patient population.

Oncogene addiction and the development of targeted cancer therapy

Andrew Joe, M.D., Merck

There has been considerable progress in the systemic treatment of cancer due to the rapid development and clinical application of molecular targeted agents. Although patients with a particular type and stage of cancer are often treated as a single group, more specific and personalized therapy has been developed, as subsets of cancer patients who are more likely to benefit from treatment with particular agents have been identified based on cancers with specific genetic abnormalities. Thus, vemurafenib and ceritinib are standard treatments for patients with

BRAF-mutated melanoma and ALK-rearranged lung cancer, respectively. We previously described the concept of “oncogene addictions for patients with BRAF-mutated melanoma and ALK-rearranged lung cancer, respectively. We previously described the concept of “oncogene addiction and clinical application of molecular targeted agents. Thus, reversal of only one or a few of these abnormalities can inhibit cancer cell growth and in some cases translate to improved survival rates. The challenge in drug development is to identify the state of oncogene addiction, i.e., the “Achilles' hill” in specific cancers.

Oncology for the 21st century

Geoffrey Kim, M.D., FDA

The treatment paradigm for many cancer types has changed dramatically due to the increased understanding of the biology of the disease and the development of novel therapeutic products. Can the pace of scientific discovery be sustained and how will therapy be optimized in the years to come?

Translational and clinical cancer research in Korea vs US: Pros and Cons

Kyu-Pyo Kim, M.D., Asan Medical Center

In 2010, over 200,000 cases of cancer were diagnosed, there were 72,000 deaths due to cancer, and one out of four patients died of cancer in Korea. The number of Korea Food and Drug Administration (KFDA) registered investigational new drug clinical studies numbered only 45 cases in 2001, but increased to over 670 cases in 2012. In addition, the Korea National Enterprise for Clinical Trials was established in 2007 and expanded the clinical trial infrastructure for new drug development through collaboration with government, academic society and industry. As cancer clinical trials require more information regarding translational science and digital data collection, Korea will encounter a new era. With translational science, institutes are initiating pre- & post-biopsies and genomic profiling. Korea is pioneering digitalization of clinical trials based on a firm IT infrastructure. However, investigators may have to understand the difference between Korea and the United States when considering and designing collaborative and/or co-clinical trials. Korea is an emerging site for translational and clinical trials with various strengths and challenges.

Directives to support biopharmaceuticals' advance to global market

Nam Soo Kim, Ministry of Food and Drug Safety

The importance of the biopharmaceutical industry is increasing as it is helping to enhance the health and quality of life of the humanity. Korean biopharmaceutical industry is growing leaps and bounds. With aging, a growing number of incurable diseases, and the progress of diagnostic technologies, a market for tailored treatment using advanced biopharmaceuticals is expected to expand dramatically. The world's drug market was about 781 billion USD in 2014, and of it, the biopharmaceuticals accounted for 179 billion USD in 2014 with an annual average growth rate of 9% in the last five years from 2010 to 2014, and the biopharmaceuticals are expected to see its share increase in the market. Korea's biopharmaceutical market was approximately 1.88 billion USD in 2014 which takes up around 10% of the entire drug market of 19.8 billion USD and it is growing 11.5% on average annually.

The MFDS continues to implement measures to globalize the biopharmaceutical industry with an aim to protect public health, strengthen biopharmaceutical safety control, and further grow relevant industries. To help biopharmaceuticals better advance to overseas markets, the MFDS is putting in place measures to better self-supply vaccines, acquire WHO PQ, give access to overseas regulations, guidelines, provide commercialization support by developmental stage, streamline review/authorization criteria, and strengthen private-public communications. Additionally, a global bio conference, an annual international meeting that will be held to share biopharmaceutical issues and recent research and development trends, will be utilized to lay the foundation of establishing a global biopharmaceutical network. The MFDS will exert utmost efforts to put in place necessary measures and policies, and lend technical support in R&D, commercialization and an advance to the global market of safe and high quality biopharmaceuticals.

Biopharmaceuticals producing platforms: developments, optimizations and challenges

Yong Hwan Jin, Ph.D., GlaxoSmithKline

The dawn of recombinant DNA technology commenced the modernization of protein-based pharmaceuticals, known as biopharmaceuticals or biologics. The first therapeutic biologics produced by recombinant DNA technology was human insulin, which obtained the first marketing authority in 1982. Since then, more than 250 recombinant drug substances have been approved in the markets of United States and EU.

With the introduction of the new class “recombinant drugs” into the markets, a new paradigm of substance definition was introduced as well. While classical low molecular weight compounds

were defined on the basis of their chemical and physical characteristics, protein drugs were classed based not only on these characteristics but also on the related production process. “The product defined by the process” became the new paradigm with the understanding that all recombinant drugs are isolated from an extremely complex matrix: a living cell.

Variety of living cells as expression platforms have been developed and used to manufacture biopharmaceuticals, which are from microbial cells (yeast and *E. coli*), plant, insect cells and mammalian cells (mainly Chinese hamster ovary; CHO). The selection of an expression platform for recombinant biopharmaceuticals is often centered upon suitable product titers and critical quality attributes, including post-translational modifications. In this presentation, considerations and challenges during the development of expression platforms for biopharmaceuticals will be discussed.

New and enabling technologies for accelerated process development of biologics

Jun Hyuk Heo, Merck

This talk will give introduction to bioprocess development and new technologies for rapid process development. The biotechnology and pharmaceutical industries continue to face pressure to reduce the time from discovery to product launch and minimize the costs associated with manufacturing and process development. Innovation through automation, single-use, and continuous processing technologies have led to efficiency and cost improvements of process development.

Combination of perfusion bioreactor and continuous purification technologies save space, resources, and time compared to traditional batch process. In addition, automated and high throughput analytical technologies with fast turn-around time is critical for supporting such accelerated process development. Technologies such as UPLC-PATROL and ASL Spectrum allow for real-time monitoring of upstream perfusion process and automated immunoassay systems such as Gyrolab give downstream monitoring capability. All in all, combination of new technologies developed in process area will enable us to build “Facility of the Future” for manufacturing of biotherapeutics.

2015 Fall KASBP Fellowship Recipients

Unconventional pathways of nitrogen metabolism in lung cancer

Jiyeon Kim, Children's Medical Research Institute, Univ. of Texas Southwestern Medical Center

Lung cancer is the leading cause of cancer death worldwide. The critical barrier in lung cancer treatment is a lack of effective therapies at later stages of the disease. Lung cancer genome sequencing supports the idea that combinations of mutations act in concert to foster malignancy and disease progression. Among these, *KRAS* and *LKB1* mutations represent “driver” changes in tumor development. Importantly, mutations in these genes also perturb signal transduction to promote a form of metabolism conducive to tumor growth. Metabolic reprogramming is considered to be fundamental to malignant transformation; thus, identifying molecular links between these mutations and metabolism, and targeting the resulting metabolic pathways, may produce better therapies. To examine metabolic signatures uniquely found in *KRAS^{mut}/LKB1^{mut}* cancer cells, we performed metabolomics analysis using *KRAS^{mut}/LKB1^{mut}* versus *KRAS^{mut}* human lung cancer cells. Notably, 5 out of 7 key metabolites differentiating *KRAS^{mut}/LKB1^{mut}* from *KRAS^{mut}* were involved in nitrogen metabolism, including nucleotides, cysteine, arginine and arginine-derived polyamines. Supporting the metabolomics data, gene set enrichment analysis (GSEA) of whole-genome transcript profiles returned ‘the urea cycle and arginine metabolism’ genes as significantly associated with *KRAS^{mut}/LKB1^{mut}* status. Furthermore, *LKB1* deficiency in the context of *KRAS^{mut}* regulates the expression of specific urea cycle genes, and a focused RNAi screen showed that *KRAS^{mut}/LKB1^{mut}* cells are selectively vulnerable to silencing of the urea cycle enzyme CPS1. These experiments establish functional significance of the urea cycle – previously not known to be relevant to cancer cell survival– in a select population of lung cancer cells harboring a particularly potent combination of oncogenic mutations. This work will elucidate a novel mechanism of urea cycle regulation and may point towards a new therapeutic strategy in *KRAS^{mut}/LKB1^{mut}* lung tumors.

Ligand-dependent conformational dynamics of ghrelin receptor

Minyoung Park, Lab. of Chemical Biology and Signal Transduction, The Rockefeller University

As obesity is associated with many medical conditions and continues to be a socioeconomic burden, there is urgent need for developing anti-obesity therapeutics. Ghrelin receptor (GhrR), a class A G protein-coupled receptor (GPCR), is a key player in regulating appetite and food intake, and thus has been a promising drug target. Previous drug discovery efforts targeting GhrR, however, failed to yield selective and potent therapeutics. By combining unnatural amino acid

(uaa) mutagenesis and bioorthogonal labeling chemistries, we developed resonance energy transfer (RET)-based assays that facilitate studies of ligand-specific conformational states of GhrR. GhrR variants modified with an azide-containing uaa were functionally assessed in cell-based bioassays and subsequently labeled with an alkyne-bearing fluorophore via strain-promoted alkyne-azide cycloaddition. With a panel of fluorescent ligands, we designed multiple fluorescent donor-acceptor pairs and measured specific RET signals that reflect ligand-binding behavior and ligand-directed structural changes in GhrR. Our bioorthogonal labeling approach and RET-based assays can be applied for screening drug candidates including orthosteric/allosteric ligands and also for preparing site-specifically labeled GhrR in single-molecule fluorescence studies. With facile applicability to other GPCRs and effector proteins, RET-based approaches also can facilitate studies of signaling events during GhrR activation, ultimately allowing us to fine-tune drug targets for obesity and related metabolic disorders.

p204 is a dual regulator critical for microbial sensing and inflammatory responses in macrophage-mediated innate immunity

Young-Su Yi, Hospital for Joint diseases, School of Medicine, New York University

p204, a member of murine p200 family, is a close homolog of human interferon-inducible protein 16 (IFI16) reported to have an anti-viral function through direct sensing intracellular viral DNA. Although several emerging studies have been actively focusing on the role of p204 as a viral sensor like IFI16, its role is still poorly understood. In this study, we report p204 senses both viral DNA and bacterial components in macrophages. Moreover, we first report p204 is also a modulator of inflammatory responses in macrophage-mediated innate immunity. IFN- γ level in macrophages challenged with various viral DNAs was first examined, and as expected, its level was significantly decreased in p204 KO macrophages. Next, macrophages were also challenged with various bacterial pathogen-associated molecular patterns (PAMPs), and interestingly, LPS mainly induced IFN- γ level while its level was significantly reduced in p204 KO macrophages. Levels of various pro-inflammatory cytokines critical for inflammatory responses were measured in LPS-stimulated macrophages, and their levels were dramatically decreased in p204 KO macrophages. In accordance with the *in vitro* data, serum levels of these cytokines and IFN- γ were also significantly reduced in p204 KO mice. Mouse survival rate under septic shock was further measured, and p204 KO mice were more resistant to LPS shock than WT mice. Next, molecular mechanisms of p204 functions were examined in macrophages. The activities of signaling molecules, including TBK1, a key player in IRF-3 pathway and PI3K/p85, AKT and IKK α/β , I κ B α , key players in NF- κ B pathway were significantly reduced in p204 KO macrophages, moreover, nuclear translocation and transcriptional activities of NF- κ B/p65 and p-

IRF-3 were dramatically decreased p204 KO macrophages. This study confirms the role of p204 as a murine counterpart of IFI16. More importantly, this study first reports novel functions of p204 as a key player in both microbial sensing and inflammatory responses in macrophage-mediated innate immunity. This study deepens our knowledge for emerging functions of p204 in host defense mechanisms against microbial infection and could provide molecular basis and new insight of p204 as a potential target for the development of anti-microbial and anti-inflammatory drugs.

Immune cell-mediated drug delivery system with biodegradable fluorescent nanoparticles for brain cancer

*Gloria B. Kim, Department of Biomedical Engineering, Materials Research Institute,
Huck Institutes of Life Sciences, The Pennsylvania State University*

Brain cancer is a life-threatening disease due to its rapid development and the difficulty in its treatment. Even after an aggressive resection followed by concurrent or sequential radiation and chemotherapies, the median survival time of patients with the most common primary brain tumor, glioblastoma, is less than 15 months. Unfortunately, the delivery of drugs to the brain is extremely challenging due to the presence of the blood-brain barrier (BBB). To overcome the BBB and achieve targeted drug delivery, we developed an immune cell-based nanoparticle (ICNP) delivery system using human monocytic cells as delivery vehicles while taking advantage of their innate targeting, penetrating, and therapeutic functions. We demonstrated their ability to transmigrate across the *in vitro* BBB and effectively deliver our biodegradable photoluminescent polymer (BPLP) nanoparticles. The intrinsic photoluminescence of BPLP nanoparticles enabled facile tracking and quantification of the migrated monocytic cells. The results suggest that our ICNP system can potentially offer advantages over traditional brain drug delivery strategies as a transformative platform technology.

MSI2 RNA binding protein target IKZF2 plays an oncogenic role in myeloid leukemia.

*Sun Mi Park, Molecular Pharmacology and Chemistry Program and Center for Cell
Engineering, Memorial Sloan-Kettering Cancer Center*

A dysregulated developmental epigenetic program is considered to be a general feature of many cancers, and mutations or chromosomal translocations with histone methyltransferases contribute to transformation in myeloid leukemias. Post-transcriptional regulation of leukemia has recently been highlighted as a novel way for maintaining the leukemia stem cell (LSC) program. We have

recently demonstrated that Msi2 is required for LSC function in a murine MLL-AF9 leukemia model. We determined that MSI2 directly maintains the mixed-lineage leukemia (MLL) self-renewal program by interacting with and retaining efficient translation of critical MLL regulated transcription factors including *Hoxa9*, *Myc*, and *Ikzf2*. Despite extensive studies implicating *Myc* and *Hoxa9* in leukemia, the role for *Ikzf2* in myeloid leukemia is not known. *Ikzf2* is a member of the *Ikaros* transcription factor family and regulates lymphocyte development by controlling regulatory T-cell function and chromatin remodeling. To find out the role of *Ikzf2* in the MLL-AF9 model, we utilized mice that have a specific deletion of *Ikzf2* in the hematopoietic system through the Vav-cre system. *Ikzf2* deficiency resulted in delayed leukemia progression and disease burden. Secondary transplantation exhibited a pronounced delay in leukemogenesis in the *Ikzf2* knockout cells. Furthermore, generating inducible deletion of *Ikzf2* using the puromycin-inducible Cre system revealed that *Ikzf2* deletion leads to cell death within 24hrs after 4-hydroxytamoxifen treatment. Flow cytometric analysis showed that *Ikzf2* deficient cells were more differentiated confirmed by increased Mac1 expression. In contrast to its known tumor suppressor role in hypodiploid B-ALL and T-ALL, these results suggest that *Ikzf2* contributes to MLL leukemia cell initiation and maintenance. Thus, we provide evidence that MSI2 maintains the oncogenic LSC epigenetic program by regulating genes such as *Ikzf2*, a chromatin remodeler which could help in maintaining the stem cell self renewal program in LSCs.

Genomic redistribution of GR monomers and dimers mediates transcriptional response to exogenous glucocorticoid in vivo

Hee-Woong Lim, The Institute for Diabetes, Obesity, and Metabolism, Perelman School of Medicine at the University of Pennsylvania

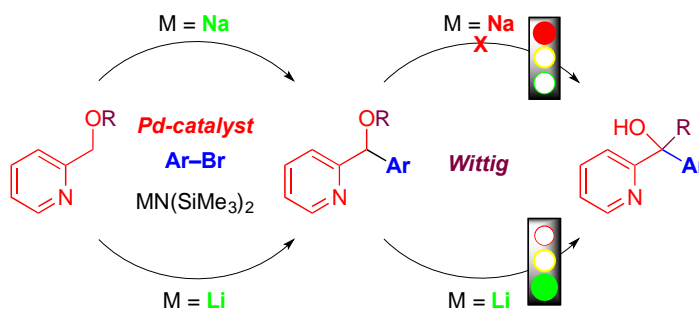
Glucocorticoids (GCs) are commonly prescribed drugs, but their anti-inflammatory benefits are mitigated by metabolic side effects. Their transcriptional effects, including tissue-specific gene activation and repression, are mediated by the glucocorticoid receptor (GR), which is known to bind as a homodimer to a palindromic DNA sequence. Using ChIP-exo in mouse liver under endogenous corticosterone exposure, we report here that monomeric GR interaction with a half-site motif is more prevalent than homodimer binding. Monomers colocalize with lineage-determining transcription factors in both liver and primary macrophages, and the GR half-site motif drives transcription, suggesting that monomeric binding is fundamental to GR's tissue-specific functions. In response to exogenous GC in vivo, GR dimers assemble on chromatin near ligand-activated genes, concomitant with monomer evacuation of sites near repressed genes. Thus, pharmacological GCs mediate gene expression by favoring GR homodimer occupancy at classic palindromic sites at the expense of monomeric binding. The findings have important

implications for improving therapies that target GR.

Chemoselective palladium-catalyzed deprotonative arylation/[1,2]-Wittig rearrangement of pyridylmethyl ethers

Byeong-Seon Kim, Department of Chemistry, University of Pennsylvania

Control of chemoselectivity is one of the most challenging problems facing chemists and is particularly important in the synthesis of bioactive compounds and medications. Herein, the first highly chemoselective tandem $C(sp^3)$ -H arylation/[1,2]-Wittig rearrangement of pyridylmethyl ethers is presented. The efficient and operationally simple protocols enable generation of either arylation products or tandem arylation/[1,2]-Wittig rearrangement products with remarkable selectivity and good to excellent yields (60–99%). Choice of base, solvent, and reaction temperature play a pivotal role in tuning the reactivity of intermediates and controlling the relative rates of competing processes. The novel arylation step is catalyzed by a $Pd(OAc)_2$ /NIXANTPHOS-based system via a deprotonative cross-coupling process. The method provides rapid access to skeletally diverse aryl(pyridyl)methanol core structures, which are central components of several medications.



2015 Fall Symposium Poster Presentations

Investigating fertility functions of *lin-28* in *C. elegans* hermaphrodites

Sungwook Choi, Program in Molecular Medicine, University of Massachusetts Medical School

lin-28 was first characterized as a developmental timing regulator in *C. elegans*. *lin-28* encodes an RNA-binding protein whose functions include downregulation of the level of *let-7* microRNA. We found that *C. elegans lin-28* loss of function (*lin-28(lf)*) mutants exhibit temperature sensitive fertility defects. *lin-28(lf)* hermaphrodites have an average brood size of 20 progeny at 20°C (~10% of normal), and are essentially sterile at 25°C. Our data show that *lin-28(lf)* mutants not only produce fewer embryos than wild type, but *lin-28(lf)* embryos also exhibit about 72% lethality. Some *lin-28(lf)* oocytes contain endomitotic DNA, which is a characteristic of defective ovulation. Many *lin-28(lf)* embryos become trapped in the spermatheca, suggesting defects in spermathecal exit. Searching for the causes of these defects, we found that the defects stem from abnormal somatic gonad development of *lin-28(lf)* mutants. In particular, abnormal structure of spermathecal uterine valve prohibits the exit of embryo from spermatheca to uterus in the mutants. In addition, *lin-28(lf)* embryos are more permeable to lipophilic dye than wild type embryos, indicating an abnormal egg shell integrity, which contributes to the embryonic lethality of *lin-28(lf)* mutants. Genetically, *let-7* microRNA acts downstream of *lin-28* in fertility function. We discovered that *lin-28(lf)let-7(lf)* partially suppress defects of ovulation, spermathecal exit, embryo production and embryonic lethality. Loss of *lin-29* function, which is positively regulated by *let-7* in heterochronic pathway, also partially rescues *lin-28(lf)* mutant's fertility defects. Other than *let-7* and *lin-29*, we found *lin-46* is also involved in fertility function of *lin-28* because *lin-28(lf)lin-46(lf)* double mutants show enhanced fertility phenotypes than *lin-28(lf)* mutants. Currently, we are determining the timing and tissue that *lin-28* is required for the fertility functions.

SLC46 family members are novel Tracheal cytotoxin transporters

Donggi Paik, Division of Infectious Diseases, University of Massachusetts Medical School

Peptidoglycan (PGN), a glyco-polymer of alternating N-acetylglucosamine and N-acetylmuramic acid, is the main component of prokaryotic cell wall and provides rigidity through cross-linked stem peptides. Small PGN fragments, known as muropeptides, are highly inflammatory. Cytosolic innate immune receptors such as NOD1, NOD2 and NLRP1 recognize these muropeptides and activate NF- κ B signaling and/or inflammasome, to elicit robust innate immune responses. However, the molecular mechanisms by which small PGN fragments gain

access to these cytoplasmic innate immune sensors are not fully understood, yet. Previous studies have revealed that members of SLC15, a family of oligonucleotide transporters, transport MDP from endosomal compartment to cytoplasm for NOD2-mediated recognition. However, no transporter for larger muropeptides, such as Tracheal Cytotoxin (TCT), has been identified. TCT, a disaccharide tetrapeptide derived from DAP-type PGN, is implicated in the cytopathology caused by *B. pertussis* and *N.* To identify transporters for TCT and potential related muropeptides, we have used unique *Drosophila* model system. The *Drosophila* innate immune system robustly responds to TCT through pattern recognition receptors, PGRP-LC and PGRP-LE. Here we report that SLC15 homologs are not responsible for the transport of TCT into the cytoplasm. Instead, SLC46 family transporters are involved with the intracellular delivery of TCT and smaller muropeptides in *Drosophila* and human cells. Overexpression of SLC46A2 is sufficient to promote NF- κ B signaling in a NOD1-dependent manner. Publicly available gene expression databases show that lung is one of the human tissues where SLC46A2 is highly expressed. Since TCT causes damage to the ciliated epithelial cell in the lung, our data suggest one of the underlying molecular mechanisms in the pathology of pertussis by TCT.

The Crosstalk between complement and TLR signaling in murine cardiac transplantation

Joong Hyuk Francis Sheen, Icahn School of Medicine at Mount Sinai

Heart transplantation is a therapy of choice for end stage heart failure. Inducing donor specific allograft tolerance in humans has remained the optimal goal and understanding the immunological mechanisms underlying “tolerance resistance” using mouse models can provide insights for prolonging the allograft survival in human transplant. We and others showed Toll-like receptor 9 (TLR9) activation by CpG DNA treatment can break MR1 (anti-CD40L mAb)-induced graft tolerance in the murine heart transplant model but the mechanisms are incompletely understood. Based on the observation that immune cell-derived complement enhances effector T cell (Teff) and suppresses regulatory T cell (Treg) responses similar to reported inflammatory effects of TLR signaling led us to hypothesize that TLR signaling and complement activation are linked. Real time qPCR data showed that CpG-stimulated WT splenic DCs significantly upregulated C3 and factor B gene expression at 24 hours. Also, TLR9 stimulation further enhanced the production of complement cascade activation product C5a during the cognate interactions between antigen presenting cells and T cells. Next, we showed that CpG-prestimulated WT DCs augmented alloreactive T cell proliferation/expansion in MLRs (mixed lymphocyte reactions) whereas similar MLR containing C3/C5 KO DCs and C3 KO allo-T cells showed ~30-50% reduction in allo-T cell responses. Also, genetic deficiencies of C3aR/C5aR (receptors for C3a/C5a respectively) on responding T cells or in vitro blockade of

C3a/C5a by neutralizing antibodies significantly impaired the TLR9-induced T cell responses. Moreover, when we compared DC phenotype in WT and C3/C5 KO DCs upon CpG stimulation, C3/C5 deficiency did not affect DC maturation but production of IL-6, TNF- α and IL-12 in DCs was modestly reduced in the KO cells. Building upon the previous observation that local complement signaling on CD4+T cells inhibits Treg generation, we showed while CpG-stimulated DCs reduced in vitro induction of Tregs, absence of C3aR/C5aR on CD4+ T cells increased the generation of Tregs despite CpG treatment. Finally, we tested whether our in vitro findings apply in a transplantation system as well. As mentioned above, CpG treatment of WT recipients after transplant induced rapid rejection of cardiac allografts (day 20, n=5) despite MR1. However, the absence of C3/C5 in the recipients significantly prolonged the allograft survival (>60 days) despite CpG stimulation post transplant (n=7 p<0.0001). Together our work reveals a novel mechanistic link between TLR signaling and immune cell derived complement and suggests that targeting complement cascade has the potential to alleviate TLR-induced inflammation amplifying alloimmunity in human transplant recipients.

Ube4b, a U-box ubiquitin ligase, is required for ubiquitination of paternal mitochondria

Seung-Wook Shin, Laboratory of Cellular and Developmental Biology, NIDDK, NIH

In many eukaryotes mitochondrial DNA is maternally inherited, but not paternal. In mice, there are several reports showing that paternal mitochondria are degraded by crosstalk between the ubiquitin-proteasome system and autophagy after fertilization. However, further studies are needed to understand the mechanism that eliminates paternal mitochondria in mammals. To elucidate this clearance mechanism, we tried to identify a E3 ligase that ubiquitinates paternal mitochondria and found polyubiquitination of sperm using cytosol of various mouse tissues and cultured cells, suggesting that E3 ligase(s) required for the ubiquitination of paternal mitochondria might be expressed in the cytosol of most organs. Next, to identify responsible E3 ligases, we fractionated cytosolic lysates from mouse liver, and evaluated each fraction for ubiquitination activities toward paternal mitochondria. Finally, we identified a U-box E3 ubiquitin ligase, Ube4b. We then tried to test the ubiquitination activity by reconstituting *in vitro* ubiquitination assay using recombinant proteins, showing that the U-box E3 ligases ubiquitinated paternal mitochondria. Further *in vivo* analyses of the mechanism in which paternal mitochondria are ubiquitinated and degraded after fertilization are currently under way included drug development of Parkinson's disease.

MDR1 transporter protects against Paraquat-induced dopaminergic neurodegeneration

Dahe You, Graduate School of Biomedical Sciences, Rutgers University

Parkinson's disease (PD) is a chronic, neurodegenerative disorder affecting around seven million people worldwide. However, its etiology has not been fully understood. The interaction of genetic and environmental factors including the exposure to pesticides such as paraquat may contribute to the pathogenesis of PD. Prior studies have observed a loss-of-function genetic polymorphism and overall reduction in the expression and function of the multidrug resistance protein 1 (MDR1, *ABCB1*) transporter in PD patients. Therefore, MDR1 may be a key element in the pathogenesis of PD. In this study, we evaluate the role of MDR1 in the transport of neurotoxicants and determine whether loss of Mdr1 function in mice altered the susceptibility to neurotoxicity. In a human brain capillary endothelial cell line (hCMEC/D3) which endogenously expresses transporters, the reduction of MDR1 transport using the antagonists PSC833 or siRNA transfection resulted in up to 200% greater accumulation of paraquat. *In vivo* studies assessed the accumulation and toxicity of paraquat in the midbrains of wild-type and Mdr1a/1b knockout mice. The knockout mice showed increased susceptibility to paraquat-induced neurotoxicity. One week after a single dose of paraquat (10mg/kg i.p.), Mdr1a/1b knockout mice had a 40% reduction in tyrosine hydroxylase-positive dopaminergic neurons in the substantia nigra pars compacta as compared to wild-type mice, which had similar staining as vehicle-treated controls. In addition, the Mdr1a/1b knockout mice treated with paraquat also experienced a more profound reduction in the expression of the dopamine transporter (DAT) and greater accumulation of alpha-synuclein compared to the wild-type mice. DAT and the organic cation transporter 3 are uptake transporters responsible for the entry of paraquat into cells; there were no basal differences in their expression between the genotypes. Collectively, these results suggest that the MDR1 transporter plays an important role in the efflux of paraquat and protection against paraquat-induced neurotoxicity.

BioID: A new cellular screen for protein-protein interactions

Daein Kim, Sanford Children's Health Research Center

In the post-genome era there is an increased emphasis on understanding of protein-protein interactions (PPIs) to identify new drug candidates. Current methods to screen for PPIs have been successful but have substantial limitations. To overcome some of these limitations and provide a complementary approach, we developed a novel method called BioID (for proximity-dependent biotin identification). This method is a fundamentally unique method to screen PPIs by using a promiscuous biotin ligase (BirA*) fused to a bait protein. When expressed in live cells, the BirA* fusion protein biotinylates proteins interacting with the bait overtime in a proximity dependent manner, which permits biotinylation selective isolation and detection of PPIs. Thus,

unlike other approaches, BioID is capable to generate a profile of PPIs irrespective of stability or affinity. To improve the BioID method, we generated a substantially smaller promiscuous biotin ligase, called BioID2. While functionally comparable to the promiscuous biotin ligase employed in the BioID method, BioID2 enables more efficient detection of PPIs with substantially less biotin. Collectively, our studies on engineered biotin ligase will further our understanding of PPIs, thus opening new horizons in therapeutics providing new targets for drug development.

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