2020 KASBP FALL eSYMPOSIUM

NEW THERAPEUTIC MODALITIES

KASBP-DAEWOONG AWARD RECIPIENT
LARRY KWAK, City of Hope
Overcoming Tumor Escape From Immunotherapy

Junghae Suh, Biogen
Future is Now for Gene Therapy

Sang Mok Chung, FDA
Clinical Trials R Us

Sangyeul Han, Ingenia
Landscape Overview of Bispecific Antibody Development

Danny Lee, Abbvie
Strategies for Ab-drug Conjugates

Spencer Nam, KSV Global
Oncolytic Viruses: A Novel Approach to Immunotherapy

Kyung Sung, FDA/CBER
Regulatory Science Insights into Cell-Based Products and Microphysiological Systems for Their Assessment

Kern H Chang, Janssen
What You Should Know About CMC When Licensing-Out Your Biopharmaceutical Product
- Licensing Antibody Products

Jiyoung Min, GSK
Rapid COVID-19 Countermeasure Development - How Vaccine Development Platforms Work for Pandemic Preparedness

3-DAY eSYMPOSIUM
EST Oct 29th - Oct 31st 7PM-11PM
PST Oct 29th - Oct 31st 4PM-8PM
KST Oct 30th - Nov 1st 8AM-12PM

No Early Bird Registration
Undergraduate Students: FREE; Graduate Students: $10; Regular members & Attendees from Korea: $20

충청북도 DAEWOONG ISU ABXIS
KPBMA NanoEntek Embassy of the Republic of Korea KHIDI GC Pharma MDimune Seegene SK biopharmaceuticals KUSCO

INVITATION LETTER

Korean American Society in Biotech and Pharmaceuticals (KASBP) invites you to the "2020 KASBP Fall sSymposium" which will be online from October 29 to October 31, 2020. Despite the threat of COVID-19 pandemic, the KASBP members will meet and stay connected at the virtual venue. This Fall Symposium focuses on "New Therapeutic Modalities" and is cohosted by Daewoong Pharmaceuticals with our other honorable sponsors.

This year the extraordinary speakers will be joining the symposium, including our distinguished keynote speaker Dr. Larry Kwak, Professor of City of Hope National Medical Center. We are excited to announce that Dr. Kwak is selected as the recipient of "KASBP-Daewoong Achievement Award" for his long-standing contribution to the immuno-oncology clinical practice and research.

Eight distinguished speakers are invited from industry who will share their experiences and expertise in cutting-edge science from early discovery research to clinical trials of the new therapeutic modalities. These modalities include bispecific antibody, antibody-drug conjugate, oncolytic viruses, gene therapy, siRNA and the cell-based products.

We are also delighted to announce that awardees were selected for KASBP Fellowships, which was possible by generous contributions from our sponsors. For the Job fair event, the committee members have put a lot of efforts to schedule for success. Many qualified applicants will have an opportunity to interview with prominent Korean pharmaceutical and biotechnology companies.

With the amazing theme and the topics, the symposium organizing committee is truly looking forward to meeting all the members and colleagues.

October 29th, 2020

2020 KASBP Fall Symposium Organizing Committee
Program Chair Hanjo Lim
KASBP President Soo-Hee Park

2020 KASBP FALL eSYMPOSIUM SPONSORS

2020 KASBP SPONSORS

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SUMMARY OF SYMPOSIUM SCHEDULE (US EST)

All sessions are accessible through Whova App. Please create an account in Whova App. using the same email for Symposium Registration to gain access to the eSymposium.

October 26-30, 2020, 6:00 – 10:00 PM, Monday to Friday **YG Program**

October 27-30, 2020, Tuesday to Friday **Job Fair**

EST	October 29, Thu	October 30, Fri	October 31, Sat
7 PM	7:00 – 7:30 PM Opening & Congratulatory Remarks	7:00 – 8:45 PM Scientific Session B • Sangyeul Han (Ingenia): Bi-specific antibody therapy • Danny Lee (AbbVie): Antibody drug conjugate • Kern Chang (Janssen R&D): Biologics CMC	7:00 – 7:20 PM Sponsor Presentation III • Seegene • MDimune
	7:30 – 8:30 PM KASBP-Daewoong Award Ceremony and Keynote Lecture • Larry Kwak (City of Hope)		7:20 – 8:30 PM Scientific Session C • Junghae Suh (Biogen): Gene therapy • Spencer Nam (KSV Global): Oncologic viral therapy
8 PM	8:30 – 8:40 PM Break		8:30 – 8:40 PM Break
	8:40 – 9:10 PM Sponsor Presentation I • Daewoong	8:45 – 9:00 PM Break	8:40 – 9:20 PM Fellowship Awards Ceremony and Presentation II
9 PM	9:10 – 10:10 PM Chungcheoungbuk-do Session	9:00 – 9:20 PM Sponsor Presentation II • IsuAbxis • NanoEntek 9:00 – 11:00 PM KHIDI/Embassy/KPBMA-KASBP Session	
		9:20 –10:00 PM Fellowship Awards Ceremony and Presentation I	9:20 – 10:30 PM Scientific Session D • Sang Mok Chung (FDA): Clinical trials • Kyung Sung (FDA): Regulatory science insights
10 PM	10:10 – 10:20 PM Break	10:00 –11:00 PM Industry-Pharma Networking Session & YG Networking/Mentor-Mentee Session	10:30 – 10:40 PM Closing Remarks
	10:20 – 11:00 PM Scientific Session A Jiyoung Min (GSK): Corona Virus		
11 PM			

➢ The rest of the program is in US Eastern Standard Time (US EST).

SUMMARY OF SYMPOSIUM SCHEDULE (한국시간 KST)

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October 27-30, 2020, Tuesday to Friday (EST) **Job Fair**

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	8:30 – 9:30 AM KASBP-Daewoong Award Ceremony and Keynote Lecture • Larry Kwak (City of Hope)			8:20 – 9:30 AM Scientific Session C • Junghae Suh (Biogen): Gene therapy • Spencer Nam (KSV Global): Oncologic viral therapy
9 AM	9:30 – 9:40 AM Break			9:30 – 9:40 AM Break
	9:40 – 10:10 AM Sponsor Presentation I • Daewoong	9:45 – 10:00 AM Break		9:40 – 10:20 AM Fellowship Awards Ceremony and Presentation II
10 AM	10:10 – 11:10 AM Chungcheoungbuk-do Session	10:00 – 10:20 AM Sponsor Presentation II • IsuAbxis • NanoEntek	10:00 AM – 12:00 PM KHIDI/Embassy/KPBMA-KASBP Session	
		10:20 –11:00 AM Fellowship Awards Ceremony and Presentation I		10:20 – 11:30 AM Scientific Session D • Sang Mok Chung (FDA): Clinical trials • Kyung Sung (FDA): Regulatory science insights
11 AM	11:10 – 11:20 AM Break	11:00 AM –12:00 PM Industry-Pharma Networking Session & YG Networking/Mentor-Mentee Session		11:30 – 11:40 AM Closing Remarks
	10:20 – 11:00 PM Scientific Session A Jiyoung Min (GSK): Corona Virus			
12 PM				

- 나머지 프로그램은 미국 동부시간 기준 입니다.

SYMPOSIUM SCHEDULE IN DETAIL

October 26 – 30, 2020, Monday – Friday

YG Program *Organizer: KASBP YG Director, Amy Boyoung Kim*

6:00 pm ~ 10:00 pm

1:1 Mocking Interview/Resume Correction Session via Zoom Meeting

October 27 – 30, 2020, Tuesday – Friday

Job Fair *Organizer: Career Development Director, Min-Kyung Choo, Ingenia Therapeutics*

October 29, 2020, Thursday

Opening & Congratulatory Remarks

7:00 pm ~ 7:30 pm *Moderator: KASBP President Designated, Hanjo Lim, Genentech*

- **Opening Remark** *KASBP President, Soo-Hee Park, Novartis*
- **Congratulatory Remarks**
  - **Hee-Mok Won** *Chairman of KPBMA (Korea Pharmaceutical and Bio-Pharma Manufacturers Association)*
  - **Sengho Jeon** *CEO and President of Daewoong Pharmaceuticals*
  - **Jae-Young Lee** *Director General of Chungcheongbuk-do Bureau of Bio Industry*

KASBP-Daewoong Achievement Award and Keynote Lecture *Moderator: Jong Sung Koh, CEO, Genosco*

7:30 pm ~ 8:30 pm

- **Overcoming Tumor Escape from Immunotherapy**
  Larry Kwak, City of Hope National Medical Center

Sponsor Presentation I *KASBP President Designated, Hanjo Lim, Genentech*

8:40 pm ~ 9:10 pm

- **Daewoong: Status of Development of COVID-19 Therapeutics by Daewoong Pharmaceuticals** *(Jumi HAN, Director, Clinical Development Center)*

Choongcheongbuk-do Session *Moderator: KASBP Councilor,* Yun Choe, Lucas & Mercanti

9:10 pm ~ 10:10 pm

Scientific Session A *KASBP-IL Chapter President, Seungwon Chung, AbbVie*

- **Rapid COVID-19 Count Measure Development – How Vaccine Development Platforms Work for Pandemic Preparedness**
  Jiyoung Min, GSK

October 30, 2020, Friday

Scientific Session B

7:00 ~ 8:45 pm *KASBP-CT Chapter President, Sung Kwon KIM, Alexion*

- **A-1: Landscape Overview of Bispecific Antibody Development**
  Sangyeul Han, Ingenia
- **A-2: Strategies For Ab-Drug Conjugates**
  Danny Lee, AbbVie
- **A-3: What You Should Know About CMC When Licensing Out Your Biopharmaceutical Product – Licensing Antibody Product**
  Kern H Chang, Janssen R&D

Break *8:45 pm ~ 9:00 pm*

Sponsor Presentation II *KASBP-IL Chapter President, Seungwon Chung, AbbVie*

9:00 pm ~ 9:20 pm

- **IsuAbxis: Innovative Immuno-Oncology Biologics Company**
  Donggoo Bae, VP, R&D
- **NanoEntek: Sharing Innovation for Life Science and Cell Therapy**
  Woo Young Sim, Business Development team

Fellowship Award Ceremony and Presentation I

Moderator: KASBP Fellowship Director, Hakryul Jo*, Kimera Therapeutics*

9:20 pm ~ 10:00 pm

Industry-Pharma Networking Session

10:00 pm ~ 11:00 pm

- **Immunology-Oncology/Autoimmune/Inflammatory:** *Moderator - Kern H Chang (Janssen R&D)*
- **Respiratory/Metabolic/Cardiovascular/Aging/Mental/Neurodegenerative:** *Moderator - Hakryul Jo (Kimera Therapeutics)*
- **Cell and Gene Therapy/Viral Infection/Rare Disease:** *Moderator - Younghoon Oh (Sarepta Therapeutics)*
- **Business Development/Legal/Venture Capital:** *Moderator - Junghoon Woo (BW Biomed)*
- **PK/PD/Pre-clinical/Clinical Science:** *Moderator - Joonyul Kim (Ciscovery)*
- **Chemistry:** *Moderator - Seungwon Chung (AbbVie)*
- **Quality/Regulatory Affairs:** *Moderator - Sung-Yong Hwang (FDA)*
- **Process/Manufacturing/Quality Control:** *Moderator - Jun Young Choi (Avecia)*
- **Bioinformatics/Quantitative Science:** *Moderator - Min Young Lee (*Takeda)
- **Clinical Supply Chain/Program Management:** *Moderator - Sahee Kim (RevHealth)*

October 30, 2020, Friday

[Public Session] *KHIDI/Embassy-KPMBA-KASBP Webinar*

9:00 pm ~ 11:00 pm

Moderator: Junghoon Woo ,CEO, BW Biomed

October 31, 2020, Saturday

Sponsor Presentation III *Moderator: KASBP-Boston Chapter President, Joon Young Choi, Avecia*

7:00 pm ~ 7:20 pm

- **Seegene: Seegene R&D** *(Dae-Hoon Lee, Managing Director)*
- **MDimune: Smart Drug Delivery Platform** *(Seung Wook Oh, CSO)*

Scientific Session C *KASBP-Phila Chapter President, Younghoon Oh, Sarepta Therapeutics*

7:20 pm ~ 8:30 pm

- **B-1: Future Is Now For Gene Therapy**
  Junghae Suh, Biogen
- **B-2: Oncolytic Viruses: A Novel Approach to Immunotherapy**
  Spencer Nam, KSV Global

Break

8:30 pm ~ 8:40 pm

Fellowship Award Presentation II *Moderator: KASBP Fellowship Director,* Hakryul Jo*, Kimera Therapeutics*

8:40 pm ~ 9:20 pm

Scientific Session D

9:20 pm ~ 10:30 pm *KASBP-Washington DC Chapter President, Sung-Yong Hwang, FDA*

- **C-1: Clinical Trials R Us**
  Sangmook Chung, FDA
- **C-2: Regulatory Science Insights into Cell-Based Products and Microphysiological Systems for Their Assessment**
  Kyung Sung, FDA/CBCR

Symposium Closing Remarks

10:30 pm ~ 10:40 pm *KASBP President, Soo-Hee Park, Novartis*

KEYNOTE LECTURE

Overcoming Tumor Escape from Immunotherapy

Larry Kwak, Hematologist - Oncologist; Dr. Michael Friedman Professor in Translational Medicine, City of Hope

Dr. Kwak knew from an early age that we wanted to pursue a career as a dual scientist and physician; to make the scientific discoveries in the laboratory and also offer them to patients in the clinic. He graduated from the highly selective 6-year combined B.S.-M.D. Honors Program in Medical Education from Northwestern University Medical School in 1982 and earned his Ph.D. in tumor cell biology there in 1984. He then completed a residency in internal medicine and a fellowship in medical oncology at Stanford University. Prior to coming to City of Hope in 2015, Dr. Kwak served for a decade as Chairman of the Department of Lymphoma and Myeloma and Co-Director of the Center for Cancer Immunology Research at M.D. Anderson Cancer Center in Houston, Texas. He began his career in basic discovery and invention of novel cancer therapies as Head of the Vaccine Biology Section at the National Cancer Institute in 1992. Few people have single-handedly taken original inventions from laboratory to clinic that have been fundamental for highly successful cancer treatments in man, but over 30 years, Dr. Kwak has serially generated disruptive technologies for novel immunotherapies and pioneered their first-in-human clinical testing. Overall, more than 500 cancer patients have been treated on first-in-human clinical trials of four unique "homegrown" therapeutic agents invented by Dr. Kwak. In 2010 Dr. Kwak was named to the TIME100, one of the world's 100 most influential people by TIME magazine, for his 20 year commitment to the science of cancer immunotherapy. In 2016 he was awarded the Ho-Am Prize in Medicine for his pioneering research in cancer immunotherapy.

He is scientific founder of Pepromene Bio, Inc., a biotech company currently developing a novel chimeric antigen receptor ("CAR") T-cell therapy. A committed physician, scientist, and mentor, he and his wife live in Pasadena, California, and they recently published a book in Korea on the subject of teamwork in parenting, drawing from their experience raising four children.

Dr. Kwak's seminal contributions to the field of cancer immunotherapy are the result of perseverance, beginning long before immuno-oncology became popular. For example, he developed the first efficacious blood cancer vaccine producing molecular remission in first-in-human trials. With the revolution of immuno-oncology in the clinic, groups worldwide are replicating personalized therapeutic vaccine approaches for solid tumors based on mutation-generated neoantigens first utilized by Dr. Kwak. His serial scientific accomplishments which highlight progress in the field of immunotherapy of cancer include:

- Pioneering first-in-human clinical trials of a personalized lymphoma vaccine, championing the technology transfer from his academic laboratory to commercial sector, and design of an international Phase III randomized clinical trial proving its efficacy. This trial became a hallmark in the field and one of the first controlled cancer vaccine trials with positive results ever. As a controlled clinical experiment, this trial fundamentally validated the cancer vaccine hypothesis, was an important breakthrough, and presaged the FDA approval of the first therapeutic cancer vaccine in 2011. (N Engl J Med 1992, Nature Med 1999, J Clin Oncol 2011)
- Demonstration of the first successful transfer of vaccine-induced anti-cancer immunity in man, forming the foundation for the FDA's approval of adoptive T-cell therapies which are now curing refractory hematologic cancers. More recently, Kwak patented and successfully licensed chimeric antigen receptor T cells (CAR-T)

against a novel leukemia target, overcoming the devastating clinical barrier of tumor relapse from antigen loss after treatment with current FDA-approved CAR-T therapies. (Lancet 1995, Sci Transl Med 2019)

- Invention of a new genetic vaccine delivery platform for cancer, AIDS, and SARS-Cov2, that targets antigen presenting cells to elicit potent T cell immunity by fusion to chemokines. Kwak's team recently completed a first-in-human clinical trial of a novel, personalized cancer vaccine, based on this technology, resulting in objective clinical responses and evidence for favorable perturbation of the tumor immune microenvironment by single-cell RNA sequencing analysis. (Nature Biotech 1999, Science 2002, BMC Cancer 2018)

References

1. Qin H et al., CAR T cells targeting BAFF-R can overcome CD19 antigen loss in B cell malignancies. Sci Transl Med. Sep 25;11(511), 2019

2. Thomas SK et al., Phase I study of an active immunotherapy for asymptomatic phase Lymphoplasmacytic lymphoma with DNA vaccines encoding antigen-chemokine fusion. BMC Cancer 18;187, 2018

3.Qin H et al. Generation of a new therapeutic peptide that depletes myeloid-derived suppressor cells in tumor-bearing mice. Nature Med. 20(6):676-81, 2014 (105 citations).

4. Schuster SJ et al. Vaccination with Patient-specific Tumor-derived Antigen in First Remission Improves Disease-Free Survival in Follicular Lymphoma. J Clin Oncol 29:2787-94, 2011. (185 citations)

5. Biragyn A et al. Toll like receptor 4 activation of dendritic cells mediated by the endogenous antimicrobial peptide B-defensin-2. Science 298:1025-9, 2002. (755 citations)

6. Bendandi M et al. Complete molecular remissions induced by patient-specific vaccination plus granulocyte-monocyte colony-stimulating factor against lymphoma. Nature Med 5(10):1171-7,1999. (525 citations)

7. Biragyn A, et al. . Genetic fusion of chemokines to a self tumor antigen induces protective, T-cell dependent anti-tumor immunity. Nature Biotech 17:253-58, 1999. (231 citations)

8. Kwak LW et al. Transfer of myeloma idiotype-specific immunity from an actively immunized marrow donor. Lancet 345:1016-20,1995 (264 citations)

9. Kwak LW et al.. Induction of immune responses in patients with B-cell lymphoma against the surface-immunoglobulin idiotype expressed by their tumors. N Engl J Med 327(17):1209-15, 1992. (497 citations)

SCIENTIFIC SESSION

SESSION A

A-1: Rapid COVID-19 Countermeasure Development- How Vaccine Development Platforms work for Pandemic Preparedness

Ji-Young Min, GSK

Biography

Global project director for new vaccines at GSK, lead cross-functional teams to drive project strategy. Collaborate effectively with internal and external stakeholders. Responsibility includes managing all aspects of vaccine project from research to launch. Maintain a high-performance team through effective team building, managerial and leadership skills. Over 20 years' experience in R&D, with 13+ years' experience serving as a program/project leader, PI, and Study Director of government and commercial contracts focusing on various infectious diseases programs. Have a proven record for creative research and directing diverse collaborative teams. Managed multiple complex collaborations and provide strategic and technical advice to improve project outcomes. Multi-task oriented with demonstrated abilities to organize, mentor staff, and make prioritization decisions. Communicate with all levels of management and scientific collaborators effectively. Professional interests include leading the development of efficacious vaccines and therapies against infectious agents, understanding the role of antibodies, chronic diseases and cancer with a keen interest to establish global portfolio to have timely public-health impact.

Educational Background & Professional Experiences:

- 2017-Current: Director, Global Vaccines, GlaxoSmithKline, Rockville, MD, USA
- 2011-2017: Head, Preparedness to Emerging Virus Group, Institut Pasteur Korea, South Korea
- 2007-2011: Staff Scientist, Research Fellow, Laboratory of Infectious Diseases, NIAID, NIH, Bethesda, MD
- 2006-2007: Postdoctoral Fellow, Institute for Cellular and Molecular Biology, Univ. of Texas, Austin, TX
- 2005: Ph.D.: Molecular Genetics and Microbiology, University of Texas, Austin, TX

Abstract

While the frequency of pandemic threats seems to be increasing, we fortunately have new tools and technologies to make vaccines with more precision and speed and that support a more proactive approach to pandemic preparedness and response. There are ~25 virus families associated with human infection from which the next pandemic threat will likely arise. Within each relevant virus family, a database of information with accompanying reagents, assays, and animal models could be developed for prototypic viruses based on properties of tropism, transmission routes, and other distinguishing features of pathogenesis. Candidate vaccine approaches could be designed based on virus structure, transmission dynamics, entry requirements, and replication strategy. Here in this talk, vaccinology for the 21st century addressing emerging infectious diseases and update on the development of COVID-19 therapeutics and vaccine candidates will be discussed.

SESSION B

B-1: Landscape Overview of Bispecific Antibody Development

Sangyeul Han, Ingenia

Biography

Sangyeul Han is currently the Co-Founder & CEO at INGENIA Therapeutics Inc., a biotech startup, established to develop innovative therapeutic antibodies. He received his B.S., M.S., and Ph.D. degrees in Molecular Biology from Seoul National University, South Korea, where his research focused on investigating the regulatory mechanism of secretory protein trafficking. In 2002, he started his postdoctoral research at the Massachusetts General Hospital/Harvard Medical School where he studied the pathological mechanisms of several tumor suppressor syndromes, including Tuberous Sclerosis Complex (TSC), Neurofibromatosis (NF), and chordomas as a research fellow/instructor. In 2010, he then joined the Biotherapeutics lab at Samsung Advanced Institute of Technology, South Korea, where he led a project for the development of therapeutic antibodies for multiple diseases. In 2016, he moved to Cell Signaling Technology, USA, where he led Antibody Technology group & Molecular Biology group. He is an author of 20+ publications in scientific journals and an inventor on multiple patents in the area of therapeutic antibody development. His expertise lies in R&D of therapeutic antibodies, vascular biology, & cancer biology.

Abstract

Bispecific antibody designed to strike two targets in one molecule is becoming a popular approach to obtain better efficacy in developing immune checkpoint modulators, as well as in the non-oncology field. In this presentation, the landscape of bispecific antibody development, will be discussed, including popular/emerging targets, prevailing formats, and challenges.

B-2: Strategies for Ab-Drug Conjugates

Danny Lee, Abbive

Biography

Danny Lee is a Senior Scientist in the medicinal chemistry department at AbbVie-Sunnyvale, California. He obtained his Ph.D. in organic chemistry at the University of Auckland (New Zealand) where he worked on immune glycopeptide vaccines and total chemical synthesis of proteins. He then joined the Stephen Kent lab at the University of Chicago to develop D-proteins as potent, non-immunogenic protein antagonists against various targets including VEGF-D. After joining AbbVie in 2015, Danny has worked on various aspects of ADCs, especially in linker and payload design and how these influences the efficacy and safety profile of ADCs.

Abstract

Antibody-drug conjugates (ADCs) are a class of biopharmaceutical drugs that have gained considerable traction in the recent years for the treatment of various cancers. There are three critical components that require fine optimization for successful ADCs: 1) monoclonal antibodies that target specific antigens on cell surfaces; 2) anti-cancer agents that drive the pharmacology; and 3) chemical linkers that play vital roles in terms of payload release, drug stability, and physicochemical properties of the resulting ADCs. In this presentation I will share key learnings from successful ADCs that have been approved in the FDA, and provide recommendations as to how one might further optimize the existing technologies for the discovery and development of next generation ADCs.

B-3: What You Should Know About CMC When Licensing Out Your Biopharmaceutical Product – Licensing Antibody Product

Kern H Chang, Janssen J&J

Biography

Kern H. Chang, Ph.D. is currently an Associate Director of Janssen R&D, a Johnson & Johnson Company with focus on CMC and analytical development of biotherapeutic products. He obtained his Ph.D. degrees in South Korea from KAIST in Biological Engineering in 2000. After finishing the graduate school, he joined Samsung in 2000 to be part of Life Science Business team for the Strategic Planning and Business Development. In 2004, he moved to US to take the post-doctoral training at Johns Hopkins Medical School in immunology and vaccines development under Professor J. Thomas August. After the post-doctoral training, he joined Bristol-Myers Squibb in 2006 to develop biologics products such as Orencia (Abatacept), Yervoy (Ipilimumab). In 2011, he joined GSK to lead a group of people to support development and the regulatory filing of monoclonal antibody products (Benlysta, Nucala) in emerging markets countries. From 2017, he has been working for Janssen R&D as project leader/scientific integrator to develop large molecule products (antibody, ADC, fusion proteins) for immunology and metabolic therapeutic areas.

Abstract

CMC (Chemistry, Manufacturing and Control) is a set of activities required for the supply of the pharmaceutical product for both clinical study and commercialization. It ranges from raw material management to manufacturing, shipping the final product to the site. These activities are defined so that the product is safe, efficacious and consistent between batches. When the CMC issues were underestimated or not properly remediated, some issues can jeopardize the licensing deal or may cause the delay to the clinical development. In this talk, the presenter will elaborate about the licensing process from CMC perspective (including due diligence, post-deal activities) and highlight the specifics which companies might want to pay attention when out licensing the products. This talk will be focusing on the biopharmaceutical products such as monoclonal antibody, antibody-drug conjugate, cell & gene therapy products.

SESSION C

C-1: Future Is Now For Gene Therapy

Junghae Suh, Biogen

Biography

In 2019, Dr. Junghae Suh joined Biogen as head of the Gene Therapy Accelerator Unit (GTxAU) to develop transformative gene therapies for the treatment of neurological diseases. Dr. Suh received her S.B. in Chemical Engineering from MIT in 1999 and a Ph.D. in Biomedical Engineering from Johns Hopkins School of Medicine in 2004. She then completed a two-year postdoctoral fellowship in the Laboratory of Genetics at the Salk Institute for Biological Studies. She is a tenured member of the faculty in the department of Bioengineering at Rice University. She was awarded the NSF CAREER Award and the Outstanding New Investigator Award from the American Society for Gene and Cell Therapy for her innovative work on reprogramming viruses as therapeutic platforms. Her academic work has been funded by the National Institutes of Health, National Science Foundation, and the American Heart Association.

Abstract

Biogen's purpose is clear: we are pioneers in neuroscience. We aim to bring transformative medicines to patients suffering from devastating diseases. Gene therapy is the newest therapeutic modality to join Biogen's strategy. As

with any new modality, there is still much work to do as we work to bring to bear its full potential. We are working hard to help build the industrial foundation for gene therapy, aiming to tackle the key technological challenges in the field in order to accelerate gene therapy investigational programs through the preclinical and clinical pipeline. Industry advancements in the field of gene therapy coupled with our expertise in neurology help to bring us closer to a day where we help make a difference in some of the most serious diseases.

C-2: Oncolytic Viruses: A Novel Approach to Immunotherapy

Spencer Nam, KSV Global

Biography

Spencer Nam is a Managing Partner and co-founder of KSV Global, a Boston-based US-Korea partnership fund between SV Investment Corporation of Korea and Kensington Capital Holdings of Boston. Since opening SV Investment's US office in early 2017, Spencer has led SV Investment's US activities and formation of KSV Global. Spencer brings more than 20 years of financial and strategic advisory experiences primarily in the U.S. health care industry. Prior to founding KSV Global, Spencer worked as a senior research fellow at the Christensen Institute for Disruptive Innovation where he researched disruptive innovation models in the health care industry. Prior to his tenure at the Christensen Institute, Spencer spent 10 years as a licensed securities analyst for several Wall Street investment banks where he had research coverage on publicly traded companies in medical devices, diagnostics and life science tools sectors. Prior to his tenure on Wall Street, Spencer was an associate at TDI Capital where he conducted investment analysis on companies in life sciences and technology sectors. Prior to TDI, Spencer was a consultant at Bain & Company where he advised senior management teams of Fortune 500 companies.Spencer is a member of the Board of Directors of Oncorus, Inc., a Cambridge, MA biotech company developing oncolytic virus immunotherapies. Spencer holds B.A. in Mathematics from Harvard College and an MBA from Harvard Business School.

Abstract

While the field of cancer immunotherapy has made significant progress over the past decade, robust opportunities still exist in developing and delivering less toxic and more efficacious therapies to patients. The oncolytic virus therapy has long been considered a possible answer to this challenge, but its potential as one of the key modalities in cancer treatment has only emerged in the last few years. Since T-Vec's FDA approval in the United States (2015), several novel approaches to make the therapy more effective have emerged. The session will survey the scientific fundamentals of oncolytic viruses and overview the current clinical developments of the viral immunotherapy. Genetic modifications and engineering methods to improve tumor targeting and anti-tumor efficacies while enhancing the safety of the therapy will be discussed. The potential for combination therapies involving immune checkpoint inhibitors and/or cellular immunotherapies will be explored. Next generation oncolytic virus models will be introduced, and we will discuss how some of the leading developers are approaching the opportunities. The participants will get a broad view of the current research and clinical advances and develop general perspective on key issues of the therapy.

SESSION D

D-1: Clinical Trials R Us

Sangmook Chung, FDA

Biography

Dr. Chung has dedicated his professional development at building expertise in regulatory science* of drug development. Dr. Chung has extensively reviewed regulatory submissions such as Investigational New Drug, New Drug Application or Biologic License Application from clinical pharmacology perspectives as part of multidisciplinary team. Dr. Chung's review experiences cover conventional small molecules, peptides, oligonucleotides, biologics and biosimilars. Dr. Chung has therapeutic backgrounds in the areas of diabetes, lipid disorders, obesity and endocrinology (e.g., growth hormone deficiency, Cushing's disease, or thyroid hormone) with further expansion in many other diseases (e.g., pulmonary and rheumatology), which can indicate for conventional or rare diseases. Dr. Chung has actively participated in regulatory researches as PI or co-PI, and presenting those results at scientific meetings. Dr. Chung has given lectures related the clinical development to colleges and universities. Dr. Chung earned Ph.D. in Pharmacokinetics, College of Pharmacy, University of Illinois at Chicago.

Abstract

Clinical development has been the significant challenge for drug developers due to high uncertainty as indicated by a low likelihood of success (e.g., 9.6% from Phase I for all developmental candidates[1]). Regulatory science has evolved to support drug development as exemplified by the Critical Path Initiative[2] and clinical pharmacology information has played increasingly roles of pivotal bridging gaps between science and the practice of medicine. Model-Informed Drug Development Pilot Program[3] is the representative initiative in those regards.
To illustrate its role in the clinical development, clinical pharmacology trials will be introduced and discussed how to execute them for bridging uncertainty in the clinical development from regulatory science perspectives.

1. Clinical development success rate 2006-2015, BIO Industry Analysis
2. Critical Path Initiative;
3. Model-Informed Drug Development Pilot Program; https://www.fda.gov/drugs/development-resources/model-informed-drug-development-pilot-program

D-2: Regulatory Science Insights into Cell-Based Products and Microphysiological Systems for Their Assessment

Kyung Sung, FDA/CBCR

Biography

Kyung Sung is a Principal Investigator in the Cellular and Tissue Therapies Branch, Division of Cellular and Gene Therapies in the Office of Tissues and Advanced Therapies. Her research focuses on developing new quantitative assays using microscale tools to understand cell-biomaterials interactions and to explore cell behavior in various tissue microenvironmental conditions. She received her Ph.D. in Chemical Engineering from the University of Michigan, Ann Arbor and did her postdoctoral training at the University of Wisconsin, Madison. She also worked as a patent examiner in Biotechnology at the US Patent and Trademark Office before she joined the FDA in 2015.

Abstract

As described in the 21st Century Cures Act, products eligible for Regenerative Medicine Advanced Therapy (RMAT) designation include cellular therapies, therapeutic tissue engineered products, human cell and tissue products, or any combination products that use such therapies or products. Multipotent stromal cells (MSCs) and induced

Pluripotent Stem Cells (iPSCs) have been popular sources for manufacturing RMAT products due to their ability to undergo lineage-specific differentiation. For successful clinical translation of such cell-based products, there is a paucity of reliable markers that can predict the products' *in vivo* performance. For instance, MSCs are very heterogeneous and responsive to their surrounding environment, resulting in distinct subpopulations of cells with potentially different qualities needed for product potency. Since there are numerous biochemical and biomechanical factors regulating the functions of MSCs, it is critical to develop reliable high-throughput assays that enable the efficient exploration of large and complex parameters for evaluating cellular function. Microscale *in vitro* systems offer the practicality to fulfill this unmet need. Several simple microfluidic channel arrays have been successfully implemented in screening the influence of paracrine mediators and various tissue microenvironment components in the regulation of cellular functions. In addition, microphysiological three-dimensional organoids and tissue-like structures such as chondrogenic cell aggregates and blood vessels have been incorporated into high-throughput, cell-based screening platforms in efforts to provide functionally relevant *in vivo*-like conditions. This presentation will give an overview of practical microscale technologies that are simple to operate while enhancing throughput, relevance, and reliability. How such technologies could be employed in the assessment of cell-based products will be discussed.

POSTER SESSION

2020 FALL FELLOWSHIP AWARDEES

AWARD NAME	AWARDEE	AFFILIATION
KASBP–Daewoong Fellowship	Jun Young Hong	Yale University
KASBP–Daewoong Fellowship	Heeseon An	Harvard Medical School
KASBP–Daewoong Fellowship	Yoon Seok Kim	Stanford University
KASBP–Choongcheongbuk-do Fellowship	Su Bin Lim	Johns Hopkins University
KASBP–Choongcheongbuk-do Fellowship	Brandon Suh	Harvard University
KASBP–GC Pharma Fellowship	Namgyu Lee	University of Massachusetts
KASBP–IsuAbxis Fellowship	Jongwoo Son	University of Wisconsin
KASBP–IsuAbxis Fellowship	Won Dong Lee	Princeton University
KASBP–KHIDI Fellowship	Haejin Yoon	Harvard Medical School
KASBP–Seegene Fellowship	Hye Jin Kim	Columbia University
KASBP–MDImune Fellowship	Young Jae Woo	Icahn School of Medicine at Mount Sinai
KASBP–NanoEntek Fellowship	Jongkyun Kang	Brigham and Women's Hospital

P-1: Developmental programming of long-term immunity by perinatal stress hormone

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Early life environmental exposure, particularly during perinatal period, can have a life-long impact on organismal development and physiology. The biological rationale for this phenomenon is to promote physiological adaptations to the anticipated environment based on early life experience. However, perinatal exposure to adverse environments can also be associated with adult-onset disorders. Multiple environmental stressors induce glucocorticoids, which prompted us to investigate their role in developmental programming. Here, we report that perinatal glucocorticoid exposure had long-term consequences and resulted in diminished CD8 T cell response in adulthood and impaired control of tumor growth and bacterial infection. We found that perinatal glucocorticoid exposure resulted in persistent alteration of the hypothalamic-pituitary-adrenal (HPA) axis by modifying mineralocorticoid receptor (type I corticosteroid receptor) expression in the hippocampus. Consequently, the level of the hormone in adults was significantly reduced, resulting in decreased CD8 T cell function. Inhibition of GR signaling in CD8 T cells acted primarily on CD25 signaling and mTOR pathway, resulting in decreased effector function and survival. Moreover, chromatin accessibility of T-bet regulated loci was significantly decreased in naïve CD8 T cells with perinatal glucocorticoid exposure. Our study thus demonstrates that perinatal stress can have long-term consequences on CD8 T cell immunity by altering HPA axis activity.

P-2: Systematic Quantitative Analysis of Ribosome Inventory During Nutrient Stress

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There is a significant interest in selective turnover of proteins and organelles via autophagy. Defects in selective removal of damaged organelles via autophagy have been implicated in various human diseases including Parkinson's,

Alzheimer's, and infectious diseases. Although many studies have revealed molecular mechanisms underlying mitophagy (removal of damaged mitochondria) and xenophagy (autophagic degradation of bacterial pathogens), our understanding on selective removal of ribosomes through autophagy is still rudimentary, and the selectivity of the ribophagy process in human cells remains controversial. Ribosomes are targets of translational control during nutrient stress and have also been suggested to be a source of amino acids and nucleotide precursors via autophagy. However, the precise contributions of biosynthetic and degradative mechanisms to ribosomal protein (r-protein) abundance is poorly understood. In this study, we investigate how the ribosome concentration in mammalian cells is maintained or altered after nutrient stress. We employ systematic quantitative methods including 1) total proteomics analysis for ribosome abundance change, 2) Ribo-Halo for simultaneous measurement of r-protein translation and dilution in single cells, 3,4) quantitative proteomics of protein synthesis and turnover, and 5) Ribo-Keima for autophagic turnover. Collectively, the methods provide a comprehensive inventory of r-proteins in response to nutrient stress. r-protein abundance during nutrient stress is primarily shaped by translational suppression, with dilution and non-autophagosomal turnover playing contributing roles. Ribophagic flux, however, accounts for a very small fraction of r-protein turnover. Our data is inconsistent with a role for NUFIP1, a previously reported ribophagy receptor, in mediating autophagic turnover of a large fraction of ribosomes. The quantitative inventory of r-proteins in this study provides a framework for examining the interplay between nutrient availability and cellular biosynthetic and degradative systems. This work is central to understanding how various organelles are remodeled due to the depth of proteome analysis which encompasses the contributions from global protein translation, the UPS- and autophagy- mediated degradations, and cell division rate control. The usefulness of our integrated dataset extends far beyond our focus on ribosomes.

P-3: Structural Foundations of Optogenetics

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The brain is a remarkably complex structure, composed of hundreds of neurons in simple organisms and up to hundreds of billions of neurons in large mammals. The recent advent of optically-modulated, molecular tools for neuroscience ('optogenetics') has allowed unprecedented access to simultaneously modulate and observe the activity of hundreds of genetically-defined neurons with millisecond resolution. However, while optogenetics has enabled rapid advances in neuroscience, this powerful toolset remains constrained by a limited mechanistic understanding of light-gated molecules, including channelrhodopsins (ChRs). Here, I describe my efforts to understand structural and dynamical mechanisms of ChRs, using three complimentary approaches. First, to extend the available high-resolution ChR structural insights, I employed X-ray crystallography to determine the structures of natural and designed anion-conducting ChRs (ACRs). Next, I used a combination of these atomic-resolution structures, molecular dynamics (MD) computational simulation, and in vitro electrophysiology to assess functional dynamics of ACRs, leading to the identification of a variant with improved channel-closing kinetics. Finally, I used structure-guided genome mining, whole-cell patch clamp electrophysiology, and two-photon imaging to identify and characterize a new red-shifted excitatory channelrhodopsin with large photocurrents and high light sensitivity. Taken together, this work provides a framework for the engineering and discovery of better optogenetic tools and lays a foundation for future studies of channelrhodopsin biology.

P-4: RNA velocity of single nuclei in Parkinson's Disease

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Parkinson's Disease (PD) is prion-like disorder characterized by the spread of pathologic α-Synuclein (α-syn) from cell to cell. It is thus clinically pertinent to identify modulators of α-syn transmission and aggregation as potential disease-modifying or neuroprotective targets. Yet, the mechanism underlying α-syn PFFs triggered aggregation of

endogenous α-syn - and the effect of PD-causing mutations on this process - is still largely unexplored particularly at the single-cell or single-nuclei resolution. Consequently, it remains unclear whether such genetic variants can be targeted for therapeutic interventions. While most PD is idiopathic, genetic models of PD have provided deep insights into the more common sporadic form of the disease, uncovering novel candidate biomarkers. Mutations in the leucine-rich repeat kinase 2 (LRRK2) gene are the most common cause of late onset PD, with similar clinical features and neurochemical changes in idiopathic PD including α-syn pathology. In this proposal, I aim to address the aforementioned gaps by leveraging in vitro model of PFF-induced α-syn aggregation and single nuclei RNA-sequencing (HiF snRNA-seq), coupled with the newly established concept of RNA velocity, using human-induced pluripotent stem cells (hiPS)-derived dopamine neurons from PD patients with LRRK2 mutations. Since the role of G2019S mutation in LRRK2 on α-syn uptake, transport, aggregation and neurodegeneration are being elucidated, and the deletion of LRRK2 has been reported to rescue α-syn PFF-induced pathology in mouse models and human neurons, it is hypothesized that LRRK2 modifies α-syn pathology, with potential clinical implications for LRRK2 inhibitors, which are currently in clinical trials. Unlike bulk-cell profiling, sc/snRNA-seq analyses enable a more holistic interrogation of distinct cell subpopulations and cellular trajectories through pseudotime analysis, revealing temporal and evolutionary processes defining PD. Here, the concept of 'RNA velocity' is being applied to HiF snRNA-seq data, in combination with subclustering, to provide insights into the transcriptional dynamics of different cellular states and PFF-induced α-syn pathology related to LRRK2 mutations. Supported by exciting preliminary data, and through high-level cross-disciplinary collaborations, I aim to achieve a deep molecular understanding of the effect of PD-causing mutation on α-syn PFF-induced pathology at the single-nuclei level. The proposed research will shed light on how early biological changes in transcriptomic profiles influence later disease phenotype through the study of molecular trajectories and transition status. In addition to unbiased classification of cell types and states, the study will enable construction of systems biology models that predict the behavior of degenerating cells during dynamic processes.

P-5: Building Homogenous Organoids using microfluids and optical stimulation

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Organoids, which mimic tissue architecture, have been emerging as powerful tools for disease modeling and drug discovery. A prominent challenge with using organoid for testing mechanisms of drug is heterogeneity of organoids. Typically, cells are seeded at high density to form an organoid culture and this usually creates organoids with a heterogeneous mix of cells. Heterogenous cultures make it difficult to study both effects and action mechanisms of a drug. In addition, heterogeneous cultures in each experiment leads to poor reproducibility. We aim to overcome this challenge by utilizing microfluidics, laser sorting, and by optical stimulation of signaling pathway. Our study will reduce the variability in organoid populations by precisely controlling the initial composition of organoids and will results in building more robust platform for drug discovery and personalized medicine.

P-6: Selenium detoxification is required for cancer-cell survival

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The micronutrient selenium is incorporated via the selenocysteine biosynthesis pathway into the rare amino acid selenocysteine, which is required in selenoproteins such as glutathione peroxidases and thioredoxin reductases. Here, we show that selenophosphate synthetase 2 (SEPHS2), an enzyme in the selenocysteine biosynthesis pathway, is essential for survival of cancer, but not normal, cells. SEPHS2 is required in cancer cells to detoxify selenide, an intermediate that is formed during selenocysteine biosynthesis. Breast and other cancer cells are selenophilic, owing to a secondary function of the cystine/glutamate antiporter SLC7A11 that promotes selenium uptake and selenocysteine biosynthesis, which, by allowing production of selenoproteins such as GPX4, protects cells against

ferroptosis. However, this activity also becomes a liability for cancer cells because selenide is poisonous and must be processed by SEPHS2. Accordingly, we find that SEPHS2 protein levels are elevated in samples from people with breast cancer, and that loss of SEPHS2 impairs growth of orthotopic mammary-tumour xenografts in mice. Collectively, our results identify a vulnerability of cancer cells and define the role of selenium metabolism in cancer.

P-7: Electrocyclizations of N-Vinylnitrones and Sigmatropic Rearrangements of N,O-Divinylhydroxylamines: Novel Access to Highly-Substituted Morpholines and Furans

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Our research group has developed new transformations involving N-vinylnitrone and N,O-divinylhydroxylamine intermediates to facilitate the synthesis of functionalized molecules and to access novel heterocyclic structures. Recently, we have expanded the scope of these methods to include the synthesis of oxazine-N-oxides through the 6π-electrocyclization of transient N-vinylnitrones, azetidine N-oxides through 4π-electrocyclization of N-vinylnitrones and the preparation of N-furanylamides through the addition of N,O-divinylhydroxylamines to electron-deficient allenes. The optimization, scope, and limitations of these new transformations will be discussed to emphasize new fundamental reactivity patterns and a detailed description of the synthetic versatility of these heterocyclic products will be presented to illustrate their utility in facilitating the preparation of sophisticated structures.

P-8: Tumor reliance on cytosolic versus mitochondrial one-carbon flux depends on folate availability

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Folate metabolism supplies one-carbon (1C) units for biosynthesis and methylation and has long been a target for cancer chemotherapy. Mitochondrial serine catabolism is considered the sole contributor of folate-mediated 1C units in proliferating cancer cells. Here, we show that under physiological folate levels in the cell environment, cytosolic serine-hydroxymethyltransferase (SHMT1) is the predominant source for 1C units in a variety of cancers, while mitochondrial 1C flux is overly repressed. Tumor-specific reliance on cytosolic 1C flux is associated with poor capacity to retain intracellular folates, which is determined by the expression of SLC19A1, which encodes the reduced folate carrier (RFC). We show that silencing SHMT1 in cells with low RFC expression impairs pyrimidine biosynthesis and tumor growth in vivo. Overall, our findings reveal major diversity in cancer cell utilization of the cytosolic versus mitochondrial folate cycle across tumors and SLC19A1 expression as a marker for increased reliance on SHMT1.

P-9: PHD3 loss promotes exercise capacity and fat oxidation in skeletal muscle

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Rapid alterations in cellular metabolism allow tissues to maintain homeostasis during changes in energy availability. The central metabolic regulator acetyl-CoA carboxylase 2 (ACC2) is robustly phosphorylated during cellular energy stress by AMP-activated protein kinase (AMPK) to relieve its suppression of fat oxidation. While ACC2 can also be hydroxylated by prolyl hydroxylase 3 (PHD3), the physiological consequence thereof is poorly understood. We find that ACC2 phosphorylation and hydroxylation occur in an inverse fashion. ACC2 hydroxylation occurs in conditions of high energy and represses fatty acid oxidation. PHD3-null mice demonstrate loss of ACC2 hydroxylation in heart and skeletal muscle and display elevated fatty acid oxidation. Whole body or skeletal muscle-specific PHD3 loss enhances exercise capacity during an endurance exercise challenge. In sum, these data identify an unexpected link between AMPK and PHD3, and a role for PHD3 in acute exercise endurance capacity and skeletal muscle metabolism.

P-10: Bisretinoids: Lipofuscin to Watch in Retina

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The presence of inherent autofluorescence in retina enables imaging ophthalmoscopically as fundus autofluorescence and examination of the latter aids in the diagnosis and monitoring of many retinal disorders in clinical practice. Fundus autofluorescence originates from the complex mixture of bisretinoid fluorophores that are amassed by retinal pigment epithelial (RPE) cells as lipofuscin. The formation is attributable to non-enzymatic irreversible reactions by two molecules of vitamin A aldehydes in photoreceptor cells; daily phagocytosis of photoreceptor outer segments results in deposition of bisretinoids in RPE. These fluorescent pigments accumulate even in healthy RPE and are a hallmark of aging retina. Nevertheless, the generation of this material is amassed at an accelerated rate, particularly in some retinal disorders including recessive Stargardt disease (STGD1) and age-related macular degeneration (AMD); a multifactorial retinal disorder that leads cause of irreversible vision loss on elderly populations. Evidence available from mouse models and clinical studies of ATP-binding cassette transporter 4 (ABCA4)-related disease, indicate that accelerated levels of bisretinoid are sufficient to negatively impact on the cells has been linked to the pathogenesis of retinal degeneration. Intervention aimed at bisretinoids would be benefit in the treatment of diseases such as STGD1. As bisretinoids impart chronic damage to retina, therapeutic approaches that target bisretionoids are likely to be appropriate for suppressing early and intermediate stages of cellular damage but less likely to be as effective for alleviating existing disease. Efforts have given consideration to intracellular deposits of visual cycle adducts that could modulate the development of AMD. Understanding the interplay among bisretinoid lipofuscin, lifetime light exposure, and oxidative stress provides insights into therapeutic implications.

P-11 Multi-scale models of neurological diseases based on big biomedical data

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Human brain is complex and interconnected structurally. Brain connectome changes are associated with wide range of neurological conditions. However, the relationship between brain connectivity and genomic changes is not well understood due to inability to acquire brain samples in living humans. I have pursued big data analysis integrating neuroimaging (in vivo) and other OMICs data (bio-samples) and have developed methods to dissect how genetics and genomics relate to human neuroanatomical changes in the context of Alzheimer's Disease (AD) and neurodevelopmental disorders: imaging genetics and imaging transcriptomics. Furthermore, these techniques are

useful at constructing multi-scale models of neurological diseases (Fig. A). Multi-scale models are based on converging evidences from different biological levels and they strengthen the validity of the model. These models are useful for 1) identifying novel disease associated genes that can serve as a therapy target, and 2) providing biomarkers as effective clinical trial endpoints for the developed drugs. The two developed integrative methods, Imaging genetics and Imaging transcriptomics, can be used to construct multi-scale models for other neurological diseases. Using Imaging genetics, how copy number variation at a four-gene region at 15q11.2 increases risk for neurodevelopmental disorders is investigated to identify causal gene contributing to the clinical outcome. Imaging transcriptomics is a network study utilizing neuroimaging features and post-mortem brain transcriptome data. Its purpose is to understand how the spatial patterning of gene expression relates to spatial variations in brain structure or function. Utilizing Diffusion Tensor Imaging (DTI) and multi-region brain transcriptome data, novel methods to integrate DTI and multi-region brain transcriptome data have been developed, and its application led to identification of gene modules that were synchronized between brain regions. This work showed that AD patients suffer white matter abnormalities proportional to disease severity, and DTI based biomarkers were replicated in an independent cohort and the molecular synchronization was found to occur more frequently between brain regions connected by white matter connections, and both synaptic and immune signaling were involved especially ionotropic glutamate receptor signaling and Toll-like receptor signaling pathways. Lastly, innate immune response measures in the blood was associated with white matter integrity for hippocampal connections, suggesting blood biomarkers can capture microstructural change in the brain and may be useful for AD diagnosis.

P-12: APP Family Regulates Neuronal Excitability and Synaptic Plasticity but Not Neuronal Survival

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Amyloid precursor protein (APP) is associated with both familial and sporadic forms of Alzheimer's disease. Despite its importance, the role of APP family in neuronal function and survival remains unclear due to perinatal lethality exhibited by knockout mice lacking all three APP family members. Here we report that selective inactivation of APP family members in excitatory neurons of the postnatal forebrain results in neither cortical neurodegeneration nor increases in apoptosis and gliosis up to ~2 years of age. However, hippocampal synaptic plasticity, learning and memory are impaired in these mutant mice. Furthermore, hippocampal neurons lacking APP family exhibit hyperexcitability, as evidenced by increased neuronal spiking in response to depolarizing current injections, whereas blockade of Kv7 channels mimics and largely occludes the effects of APP family inactivation. These findings demonstrate that APP family is not required for neuronal survival, and suggest that APP family may regulate neuronal excitability through Kv7 channels.

P-13 Small molecule-induced polymerization triggers degradation of BCL6

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Effective and sustained inhibition of non-enzymatic oncogenic driver proteins represents a major pharmacologic challenge. The clinical success of thalidomide analogs demonstrates the therapeutic efficacy of drug-induced degradation of transcription factors and other cancer targets, but a significant subset of proteins are recalcitrant to targeted protein degradation using current approaches. Here we report an alternative mechanism, whereby a small molecule induces highly specific, reversible polymerization, sequestration into cellular foci, and subsequent degradation of a target protein. We used cryo-EM to reveal how the solvent-exposed moiety of a BCL6 inhibitor contributes to a composite ligand/protein surface that engages BCL6 homodimers to form a supramolecular

structure. Drug-induced formation of BCL6 filaments facilitates ubiquitination by the E3 ubiquitin ligase. Our findings demonstrate that a small molecule can induce polymerization coupled to highly specific protein degradation, which in the case of BCL6 leads to superior pharmacological activity. These findings create new avenues for the development of therapeutics and synthetic biology.

P-14 Non-cell autonomous hyperexcitability underlies focal epileptogenesis mediated by low-level brain somatic mutations in MTOR

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Low-level of brain somatic mutations (e.g. less than ~1% of mutated neurons) are recently known as a major genetic cause of intractable focal epilepsies. However, how such sparse mutation-carrying neurons induce focal epileptogenesis remains poorly understood. Here, using computation simulation of neural network followed by electrophysiological and biochemical experiments in the focal cortical dysplasia (FCD) mouse model with brain somatic mutations in MTOR, we found that seizure triggering hyperexcitability was originated from non-mutated nearby neurons rather than mutation-carrying dysmorphic neurons. Interestingly, mutation-carrying neurons were less excitable and had no significant change in the net balance of the excitatory and inhibitory synaptic property. Furthermore, we found that the inhibition of adenosine kinase (ADK), which is known to affect adenosine metabolism and neuronal excitability, reduced hyperexcitability of non-mutated surrounding neurons. Thus, this study shows that low-level brain somatic mutations in MTOR lead to focal epileptogenesis via non-cell autonomous hyperexcitability of surrounding neurons.

P-15 Loss of epigenetic information as a cause of mammalian aging

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All living things experience entropy, manifested as a loss of inherited genetic and epigenetic information over time. As budding yeast cells age, epigenetic changes result in a loss of cell identity and sterility, both hallmarks of yeast aging. In mammals, epigenetic information is also lost over time, but why it is lost and whether it is a cause or a consequence of aging is not known. To test this, we generated a system called ICE (for inducible changes to the epigenome), which allows for the transient induction of genomic instability, in the form of a low number of non-mutagenic DNA breaks. Here we provide evidence that faithful DNA repair induces the erosion of the epigenetic landscape, a loss of cellular identity, and advancement of the epigenetic clock. As assessed by Hi-C and HiChIP, higher-order chromatin architecture, CTCF-mediated chromatin insulation, and spatial chromatin contacts are altered. ICE mice show physiological, cognitive, and molecular changes normally seen in older mice, including

accelerated DNA methylation age. These data support a model in which the cellular response to DNA breaks perturbs multiple layers of epigenetic information to help drive aging in mammals.

P-16 Functionally distinct population of immune suppressive macrophages differentiate from monocytic myeloid-derived suppressor cells

Taekyoung Kwak, Ph.D.
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Macrophage is one of basic immune cell types that have functions of clearing toxic substance such as bacteria and virus in our body. Because of its plasticity, macrophage is frequently changing its characteristics by nearby environments including cytokines and chemokines. Although macrophage works as first-defense of innate immunity in our body, depending on physiological situation like cancer, this heterogeneous macrophage population turns its role as pro-tumor effect. Tumor associated macrophages (TAM) are heterogeneous group of cells with variable immune suppressive activity. In this study, we report that functional heterogeneity of macrophages in cancer could be determined by the nature of their precursors: monocytes (Mon) or monocytic myeloid-derived suppressor cells (M-MDSC). Macrophages differentiated from M-MDSC, but not from Mon were immune suppressive with genomic profile matching that of M-MDSC. Immune suppressive activity of M-MDSC derived macrophages was dependent on the persistent expression of S100A9 protein in these cells. S100A9 promoted M2 polarization of macrophages. Tissue resident and Mon derived macrophages lacked S100A9 protein. S100A9 dependent immune suppressive activity of macrophage was mediated via transcription factor CEBP/β. S100A9 positive TAM were associated with shorter survival in patients with head and neck cancer and poor response to PD-1 antibody treatment in patients with metastatic melanoma. Thus, this study revealed the pathway of the development of immune suppressive TAM and suggested approach to their selective targeting in clinical manner.

P-17 *Humanized mouse model to test host immune response to iPSC-derived dopaminergic cells for personalized cell therapy of Parkinson's disease*

Tae-Yoon Park[1,2], Nayeon Lee[1,2], Pierre Leblanc[1,2], Jeffrey Schweitzer[3], and Kwang-Soo Kim[1,2]
[1]Department of Psychiatry, McLean Hospital, Harvard Medical School, Belmont, MA 02478, USA, [2]Molecular Neurobiology Laboratory, McLean Hospital, Program in Neuroscience and Harvard Stem Cell Institute, Harvard Medical School, MA 02478, USA, [3]Department of Neurosurgery, Massachusetts General Hospital, Harvard Medical School, MA 02114, USA
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Cell replacement therapy using human iPSC- or ESC-derived dopaminergic cells is a promising therapeutic strategy for the treatment of Parkinson's disease (PD). Because the brain has traditionally been considered an "immune-privileged" organ, the necessity for immunosuppression with allogeneic grafts has been controversial. More recent information has revealed the complex interactions between the brain and the immune system which raised the possibility of immune rejection of allogeneic cells over time. While autologous cell transplants would theoretically circumvent the requirement for immunosuppressive agents, it has been argued that the in vitro differentiation processing of autologous iPSC-derived cells could result in immunogenicity and therefore possible immune rejection by the host. This would negate the theoretical advantages of autologous cell replacement therapy. In order to investigate whether autologous iPSC-derived dopaminergic progenitor cells differentiated in vitro would be rejected or not, we sought to establish a NOD/SCID/IL2rγ^{null} humanized mouse model. The model is reconstituted by injecting human PBMCs into the peritoneal cavity of the mouse followed by transplantation of either autologous or allogeneic dopaminergic progenitor cells into the striatum of the humanized mice. In this study, we demonstrate that transplantation of PD patient-derived midbrain dopaminergic progenitor cells (C4-mDAP), differentiated in vitro from autologous iPSCs (C4-iPSC), were not rejected by humanized mice reconstituted with the patient's PBMCs. In contrast, humanized mice reconstituted with allogeneic PBMCs rejected the C4-mDAP, demonstrating that they can be recognized by a foreign immune system. In addition, mDAPs differentiated from human ESCs were

rejected by both mice humanized from either source. These data support the hypothesis that personalized cell transplantation therapy for PD using autologous iPSC-derived differentiated neurons without the use of immunosuppression is indeed a viable strategy.

P-18 PGE1 and PGA1 bind to Nurr1 (NR4A2) and activate its transcriptional function

Woori Kim[1,15], Sreekanth Rajan[2,15], Yongwoo Jang[1,3,15], Chun-Hyung Kim[1,4,15], Hui Ting Toh[2,5], Jeha Jeon[1], Bin Song[1], Aida Serra[2], Julien Lescar[2,6], Jun Yeob Yoo[2], Serap Beldar[2], Hong Ye[2], Congbao Kang[7], Xue-Wei Liu[8], Melissa Feitosa[1], Yeahan Kim[1], Dabin Hwang[1], Geraldine Goh[9], Kah-Leong Lim[9,10], Hye Min Park[11], Choong Hwan Lee[11], Sungwhan F. Oh[12], Gregory A. Petsko[13], Ho Sup Yoon[2,6] and Kwang-Soo Kim[1,14]

[1]Molecular Neurobiology Laboratory, Department of Psychiatry, McLean Hospital, Harvard Medical School, Belmont, MA, USA, [2]School of Biological Sciences, Nanyang Technological University, Singapore, Singapore, [3]Department of Biomedical Engineering, Hanyang University, Seoul, Korea, [4]Paean Biotechnology, Daejeon, Korea, [5]Nanyang Institute of Technology in Health and Medicine, Interdisciplinary Graduate School, Nanyang Technological University, Singapore, Singapore, [6]NTU Institute of Structural Biology, Nanyang Technological University, Singapore, Singapore, [7]Experimental Drug Development Centre, Agency for Science, Technology and Research, Nanos, Singapore, Singapore, [8]Division of Chemistry and Biological Chemistry, School of Physical and Mathematical Sciences, Nanyang Technological University, Singapore, Singapore, [9]National Neuroscience Institute, Singapore, Singapore, [10]Lee Kong Chian School of Medicine, Singapore, Singapore, [11]Department of Bioscience and Biotechnology, Konkuk University, Gwangjin-gu, Seoul, Republic of Korea, [12]Department of Anesthesiology, Perioperative and Pain Medicine, Brigham andWomen's Hospital, Harvard Medical School, Boston, MA, USA, [13]Ann Romney Center for Neurologic Diseases, Department of Neurology, Brigham andWomen's Hospital, Harvard Medical School, Boston, MA, USA, [14]Program in Neuroscience and Harvard Stem Cell Institute, McLean Hospital, Harvard Medical School, Belmont, MA, USA, [15]These authors contributed equally

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The orphan nuclear receptor Nurr1 is critical for the development, maintenance and protection of midbrain dopaminergic (mDA) neurons. Here we show that prostaglandin E1 (PGE1) and its dehydrated metabolite, PGA1, directly interact with the ligand-binding domain (LBD) of Nurr1 and stimulate its transcriptional function.We also report the crystallographic structure of Nurr1-LBD bound to PGA1 at 2.05 Å resolution. PGA1 couples covalently to Nurr1-LBD by forming a Michael adduct with Cys566, and induces notable conformational changes, including a 21° shift of the activation function-2 helix (H12) away from the protein core. Furthermore, PGE1/PGA1 exhibit neuroprotective effects in a Nurr1-dependent manner, prominently enhance expression of Nurr1 target genes in mDA neurons and improve motor deficits in 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine-lesioned mouse models of Parkinson's disease. Based on these results, we propose that PGE1/PGA1 represent native ligands of Nurr1 and can exert neuroprotective effects on mDA neurons, via activation of Nurr1's transcriptional function.

P-19 Lymphangiogenesis near the Cribriform Plate after Cerebral Ischemia

Yun Hwa Choi[1], Martin Hsu[2], Collin Laaker[2], Matyas Sandor[3], Zsuzsanna Fabry[3]

[1]Pharmaceutical Sciences Division, School of Pharmacy, University of Wisconsin-Madison, [2]Neuroscience Training Program, University of Wisconsin-Madison, [3]Department of Pathology and Laboratory Medicine, University of Wisconsin-Madison

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Conventional lymphatic vessels typically reside within the tissue parenchyma and facilitate drainage to local lymph nodes. However, the central nervous system (CNS) has traditionally been described as "immune privileged" because there are no conventional lymphatic vessels or peripheral immune cells within the CNS parenchyma. Recently, it has been hypothesized that antigens, antigen-presenting cells (APCs), and cerebrospinal fluid (CSF) may drain from the CNS into lymphatics near the cribriform plate or dura in order to provide antigen drainage and fluid homeostasis.

This recent re-discovery of lymphatic vessels surrounding the CNS under steady-state conditions ignited an intense debate about mechanisms of how antigens or immune cells drain from the CNS parenchyma to lymphatic organs. Additionally, the role of lymphatic vessels in the CNS during neuroinflammation is not well understood yet. Here, we study how lymphangiogenesis near the cribriform plate contributes to recovery of brain tissues following transient ischemic stroke. We employ the transient middle cerebral artery occlusion (tMCAO) model of ischemic stroke by placing a filament into the middle cerebral artery, followed by reperfusion after removing the filament after 60 minutes. We show that lymphangiogenesis occurs near the cribriform plate, which peaks at day 7 and decreases after 14 days following tMCAO. The lymphangiogenesis occurred through interaction of vascular endothelial growth factor receptor (VEGFR)-3 and vascular endothelial growth factor (VEGF)-C which are produced by $CD11b^{+}$, $CD11c^{+}$ dendritic cells. These lymphangiogenic vessels could transport CSF and immune cells which were confirmed by injection of Evans Blue dye through cisterna magna and flow cytometry, respectively. Flow cytometry analysis showed robust leukocyte accumulation, which include macrophages ($CD45^{hi}$, $CD11b^{+}$, $CD11c^{low}$), dendritic cells ($CD45^{hi}$, $CD11b^{+}$, $CD11c^{hi}$), CD8 T cells ($CD45^{hi}$, $CD11b^{-}$, $CD11c^{-}$, $CD8^{+}$), and CD4 T cells ($CD45^{hi}$, $CD11b^{-}$, $CD11c^{-}$, $CD4^{+}$) near the cribriform plate after 7 days of tMCAO, which also correlates with lymphangiogenesis ($CD45^{-}$, $Podoplanin^{+}$, $CD31^{+}$). Inhibition of VEGFR-3 reduced lymphangiogenesis near the cribriform plate and improved motor ability at earlier time points following tMCAO. Understanding the biology and mechanisms of CNS lymphatic drainages may lead to novel therapeutics for neuroinflammatory diseases.

KASBP 2020 FALL SYMPOSIUM YG PROGRAM

1:1 Mocking Interview / Resume Correction Session via Zoom Meeting

October 26-30, 2020 (6pm-10pm)

Purpose

To support KASBP's important goal: identifying and nurturing young Korean researchers to grow up to become future leaders for Korean Biotech and Pharmaceutical industries, YG is aiming to provide practical help for young professionals with job searching. By this program, our goal is to successfully reduce the pain of starting the networking, prevent one-time occurring short conversation, and provide efficient guides in specific fields.

Who can participate?

Students, young professionals, even semiprofessionals, and everyone who needs advice.

How it works?

It is mocking interview session, so the mentors will actually be interviewing mentees. But the difference is that they give reviews and tips after the interview. Here is how mentor-mentee matching processed:

1) Mentees get notification through KASBP email RSVP to the pull
2) YG team send individual email to Mentees
3) Mentees have to submit the job description and Resume
4) Upon confirming, YG will look for Mentors based on Mentee's goal and field of interest
5) Once mentor-mentee matched, Mentees' resume, job descriptions and basic info are sent to Mentors
6) YG schedules the meeting
7) Mentees have to create Zoom meeting links
8) YG sends zoom link to mentor and Cc the final email to mentees
9) Mentor-Mentee 1:1 sessions are held following schedule

More questions? Need mentor? Contact KASBP YG director: amy.boyoung.kim@gmail.com

2020 KASBP FALL ONLINE JOB FAIR

October 27-30, 2020 (Prearranged Meeting Time)

The 2020 KASBP Fall Job Fair will provide an online meeting for conversation between applicants and hiring managers at Korean pharmaceutical companies. 2020 KASBP Fall eSymposium Registration is required, but no additional fees are required to participate in Job-Fair as an applicant.

For the past few years, the KASBP Job Fair has established itself as a direct channel for many Korean scientists who are researching or working in pharmaceutical companies in the US to be recruited by Korean pharmaceutical companies. In addition, the hiring managers of Korean pharmaceutical companies consider it an opportunity to meet and recruit talented people from overseas, and have been constantly participating. Currently participating companies are Daewoong Pharmaceutical, Isu Abxis, GC Pharma, Seegene, Samyang Biopharm, and MDimune. Candidates may apply multiples companies.

The job-fair application was closed on October 2, 2020. For a late-break application, please contact the KASBP Job-Fair coordinator (jobfair@kasbp.org) to check the availability. The job description of the companies is available from KASBP website (under 2020 Fall Job Fair).

Interview time is pre-arranged during 8:00 PM ~ 11:00 PM, Tuesday-Friday, October 27-30, 2020. The interview meeting details are sent to each candidate and interviewer via email.

More information about 2020 KASBP Fall eSymposium Job Fair can be found at:

- 2020 KASBP Fall Job Fair Info.: https://kasbp.org/2020-Fall-Job-Fair
- Application Link: https://forms.gle/ce5a9gBmEGzjtZAw6
- 2020 KASBP Fall eSymposium Registration: https://kasbp.org/event-3963171
- Contact: jobfair@kasbp.org

Sincerely,

2020 KASBP Fall eSymposium Organizing Committee

[PUBLIC SESSION] KHIDI/Embassy/KPMBA/KASBP WEBINAR

October 30, 2020, 9:00 pm ~ 11:00 pm

GLOBAL LICENSING STRATEGY OF KOREAN BIO-PHARMA BY EACH STEP
제약바이오 단계별 글로벌 라이센스 전략

AS A PART OF THE

2020 KASBP FALL eSYMPOSIUM

SPEAKERS

BW Biomed
우정훈 대표

Jounce Therapeutics
김민지 박사 BD 총괄상무

Nelson Mullins
김공식 파트너

Solasta Ventures
윤동민 대표

패널 토론으로 진행:
사업개발, 특허/법률, 투자 분야에서 바라본 라이센스 단계별 전략

Available via Zoom Link:
https://us02web.zoom.us/j/84783925973?pwd=Q2lqdG1XbVdqZzFoRlArMkVidDQwZz09

온라인 웨비나
EST Friday Oct 30, 9:00PM-11:00PM
PST Friday Oct 30, 6:00PM-8:00PM
KST Saturday Oct 31, 10:00AM-12:00PM

※ 참석자: KASBP 등록자 외에 모든 국내외 제약바이오 기업 종사자 참여 가능

Korean American Society in Biotech and Pharmaceuticals 재미한인제약인협회

Embassy of the Republic of Korea

KHIDI Korea Health Industry Development Institute

KPBMA

[PUBLIC SESSION] KHIDI/Embassy/KPMBA/KASBP WEBINAR (con'd)

※ 참석자: KASBP 등록자 외에 모든 국내외 제약바이오 기업 종사자 참여 가능

1. 개요

1) **발표자**

- 좌장: BW Biomed 우정훈 대표
- 연사
  - BD 분야 : Jounce Therapeutics BD Head 김민지 상무
  - 특허/법률 분야 : Nelson Mullins 김공식 파트너
  - 투자 분야 : Solasta Ventures 윤동민 대표

2. 행사 순서 및 발표 내용

1) 개회사
   - 주미대한민국 대사관 신꽃시계 복지관
2) 연사 소개 및 주제별 진행 방향 설
   - 좌장 - 우정훈
3) 주제 발표 및 패널 토론

3. 세부 발표 및 토론 내용

1) 우정훈 대표 (**글로벌 제약바이오 산업 동향**)
   - Covid 이전과 이후 질환별 투자 및 전략적 제휴 동향
   - 단계별 해외 라이센스 인/아웃 프로세스
2) 김민지 BD 총괄 상무 (**글로벌 라이선스 전략**)
   - 기술 초기 검토 (NDA 체결) 단계 주요 이슈
   - Term Sheet 작성 등 협상 시 주요 이슈 및 유의 사항
   - Due Diligence 및 계약 단계 핵심 사항
3) 김공식 파트너 (**글로벌 진출을 위한 특허 및 법률 전략**)
   - 기술 검토 시 특허 부분 주요 이슈
   - Term Sheet 작성 & 계약서 작성 시 체크 리스트
4) 윤동민 대표 (**제약바이오 글로벌 투자 전략**)
   - 기술 투자 검토 시 주요 포인트
   - 투자자 입장에서 Valuation 등 협상 단계 핵심 사항
5) 패널 토론
   - 미국 라이센스 관련 투자, 시장, 특허 분야 현황
   - 전세계 (미국포함) 제약 바이오 산업 전망
   - 한국 제약바이오 기업의 미국 기업/연구소와의 전략적 제휴 방안

PAST FELLOWSHIP AWARDEES

KASBP-DAEWOONG ACHIEVEMENT

2009 Jong Eun Kim (Gilead Sciences, Inc.), (Kainos Medicine Inc, Korea, Current)
2010 David C. Chu, (University of Georgia)
2011 Sung Ho Kim (University of California, Berkeley)
2012 Dennis Choi (Stony Brook Medicine and Stony Brook University)
2013 Joseph Kim (Inovio Pharmaceuticals)
2014 Kinam Park (Purdue University)
2015 Jong Sung Koh (Genoscco)
2016 Jang-Ho Cha (Novartis)
2017 Peter Park (Bicycle Therapeutics)
2018 Jong Wook Lee (Daewoong Pharmaceuticals)
2019 Kwang-Soo Kim (Harvard Medical School)

KASBP RECOGNITION AWARD

2015 Jong Wook Lee, Daewoong

KASBP-DAEWOONG FELLOWSHIP

2006 Jaeki Min (New York University), Hahn Kim (Princeton University), Hyejin Park (Rutgers University)
2007 Jisook Moon (Harvard University), Sungyeon Park (Rutgers University), Seokgeun Lee (Columbia University)
2008 Heungkyu Lee (Yale University), Junghwan Kim (Rutgers University), Minsik Kang (Columbia University)
2009 Jinah Park (Harvard University), Jaemin Choi (Yale University), Deokho Kim (Johns Hopkins University)
2010 Jungmin Kee (Rockefeller University), Hyungwook Kim (Nih), Sejin Ahn (Harvard University)
2011 Moori Han (University Of California, La), Hwanjong Jang (Boston College)
2012 Jeongho Jang (Columbia University), Jaewoo Choi (Oregon State University)
2013 JangEun Lee (University of Pennsylvania), Eun Chan Park (Rutgers University)
2014 Kimberly H. Kim (Harvard University), Seung Koo Lee (Weill Cornell Medical College), Min-Sik Kim (Johns Hopkins University)
2015 Jiyeon Kim; UT Southwestern; Sun Mi Park, Memorial Sloan-Kettering Center); Byeong Seon Kim, (University of Pennsylvania)
2016 Sang Bae Lee (Columbia University), Junil Kim (University of Pennsylvania), Ho-Keun Kwon (Harvard Medical School)
2017 KyeongJin Kim (Columbia University Medical Center), Min-Ji Bak (Ernest Mario School of Pharmacy), Heung Sik Hahm (Free University Berlin)
2018 Jung Ho Hyun (Max-Plank Florida Institute for Neuroscience), Seung Hoon Lee (Harvard Medical School), Jang Hwan Cho (Boston University)
2019 Hyunyong Koh (Boston children's Hospital), Young Cha (MeLean Hospital), Hojong Yoon (Harvard University)

KASBP-GREEN CROSS FELLOWSHIP

2011 HanSang Cho (Harvard Medical School), SungWoong Kang (Johns Hopkins University), MiYeon Kim (Columbia University), JaeYoung Soh (Rutgers University), SungYong Hwang (NIEHS/NIH)
2012 WonJin Cho (Drexel University), HyoJung Kang (Yale University), JungHyun Lee (Columbia University), YongJae Lee (Yale University), JaeHyun Yoon (NIH)
2013 Yunjong Lee (Johns Hopkins University), Jun-Dae Kim (Yale University)
Bae-Hoon Kim (Yale University), Ja Young Kim-Muller (Columbia University)

2014 Catherine Rhee (University of Texas at Austin), Ji-Seon Seo (The Rockefeller University), Sehyun Kim (New York University)
2015 Young-Su Yi (New York University), Hee-Woong Lim (University of Pennsylvania), Gloria Bora Kim (The Pennsylvania State University)
2016 Eui Tae Kim (University of Pennsylvania), Kihyun Lee (Weill Cornell Medical Science)
2017 Seung-Yeol Park (Harvard medical school), Young Bok Abraham Kang (Harvard medical school)
2018 Jae Yeon Hwang (Yale University), Youngjin Kim (Rockefeller University)

KASBP-HANMI FELLOWSHIP

2011 HyungJin Ahn (Rockefeller University), ChangHoon Cho (Abramson Research Center)
2012 YuNa Kim (University of North Carolina), HyunSeob Tae (Yale University), InHye Lee (NIH)
2013 JooHee Lee (Memorial Sloan-Kettering Cancer Center), KyungRyun Lee (Rutgers University), ManRyul Lee (Indiana University)
2014 Young Chan Cha (Wistar Institute), Min-Kyu Cho (New York University), Lark Kyun Kim, (Yale University), Yu Shin Kim (Johns Hopkins University)
2015 Seonil Kim (New York University), Peter B. Kim (Yale University)
2016 Sungwhan Oh (Harvard Medical School), Won-Gil Lee (Yale University), Hee-Jin Jeong (Harvard Medical School)
2017 Seungkyu Lee (Harvard Medical School), Soo Seok Hwang (Yale University), Heeoon Han (University of Pennsylvania)
2018 Jae Yeon Hwang (Yale University), Yeong Shin Yim (MIT), Dahea Yu (Rutgers University)

KASBP-LG CHEM FELLOWSHIP

2017 Kyoung-Dong Kim (Wistar Institute), Seok-Man Ho (Icahn School of Medicine at Mount Sinai)

KASBP-QURIENT FELLOWSHIP

2018 Soeun Kang (University of Illinois at Chicago), Do Hyung Kim (Johns Hopkins University)
2019 Jae Hyun Baek (Biogen), Donggi Paik (Harvard Medical School)

KASBP-YUHAN FELLOWSHIP

2011 KiYoung Kim (Boston University), JoongSeop Shim (Johns Hopkins University)
2012 YeMin Huh (University of Michigan), SookHee Bang (University of Pennsylvania), JungHo Baik (Columbia University)
2013 Dong Jun Lee (University of Chicago), Ingyu Kim (Yale University), Ja Yil Lee (Columbia University)
2014 Seouk Joon Kwon (Rensselaer Polytech Institute), Jeongmin Song (Yale University), Jae-Hyun Yang (Harvard Medical School), Wan Seok Yang (Columbia University)
2015 Min-Joon Han (Harvard Medical School), Minjung Kang (Cornell University)
2016 Ki Su Kim (Harvard Medical School), Hongjae Sunwoo (Harvard Medical School), Seo-Young Park (University of Massachusetts)
2017 Hanseul Yang (Rockefeller University), Ji-Hoon Park (NIH), Hong-Yeoul Ryu (Yale University)
2018 Sangdoo Kim (Harvard Medical School), Baehyun Shin (Harvard Medical School), Mikyung Yu (Harvard Medical School)

KASBP-ST PHARM FELLOWSHIP

2016 Jung-Eun Jang (New York University), Byungsu Kwon (MIT)

KASBP FELLOWSHIP

2009 Sangho Choi (NIH)
2010 Sangryung Kim (Columbia University), Taesook Yoon (Rutgers University), Eunmi Huh (Cal. Tech.)
2015 (Spring) Mi Jung Kim (Duke University)
2015 (Fall) Minyoung Park (The Rockefeller University)

KASBP-KSEA FELLOWSHIP

2013 Sung In Lim (University of Virginia)
2014 Keun-woo Jin (Temple University)

KASBP-KUSCO FELLOWSHIP

2008 Hyunho Kim (National Institutes of Health), Taekbeom Ohn (Harvard Medical School), Wonah Joo (Wistar Institute)

KASBP-KRICT FELLOWSHIP

2009 Seungsik SHIN (Rutgers University), Eunjoo JEONG (Columbia University), Kyuwon BAEK (University of Pennsylvania)

KASBP-SAMSUNG FELLOWSHIP

2019 Eunju Im (Nathan S. Kline Institute for Psychiatry Research), Jongho Park (Massachusetts General Hospital)

KASBP- KRIBB FELLOWSHIP

2019 Sooong Min (Harvard Medical School), Eun-Ik Koh (University Of Massachusetts Medical School)

KASBP-KHIDI FELLOWSHIP

2010 Jaehyun BAE (Yale University), Heeyeon CHO (Boston College)

KASBP FELLOWSHIP

2019 Kyusik Kim (University of Massachusetts Medical School)

KASBP-DAEWOONG SCHOLARSHIP

2006 Jin K. Pai, Schering-Plough (Handok Pharmaceuticals, Korea, Current)
2007 Youngwhan Park, Merck (National Cancer Center, Korea, Current)
2008 Young-Choon Moon (PTC Therapeutics)
2009 Hongyong Kim (Novartis)

END OF THE PROGRAM

THANK YOU FOR ATTENDING 2020 KASBP FALL eSYMPOSIUM!