



# 2022 KASBP FALL SYMPOSIUM

**POST-PANDEMIC PHARMACEUTICAL INDUSTRY;**  
Forecasting the Trend in life-Science, Technology, and Innovation

**November 4 (FRI) – 5 (SAT), 2022**  
**Hilton Boston-Dedham** | 25 Allied Dr, Dedham, MA 02026



## KASBP-DAEWOONG ACHIEVEMENT AWARD

**JAE PARK** (Memorial Sloan Kettering Cancer Center)

## KASBP - KHIDI USA GENE AND CELL THERAPY PANEL DISCUSSION



**DANIEL C. CHUNG**  
SpringVision



**JUNGHAЕ SUH**  
Biogen



**BYOUNG RYU**  
Umoja Biopharma



**JOOYOUNG LEE**  
Vertex

## SCIENTIFIC SESSION SPEAKERS



**YANG LEE**  
GSK (GlaxoSmithKline)



**MATT LEE**  
Beam Therapeutics



**TAEYOON KYUNG**  
Ginkgo Bioworks



**TAEGYO LEE**  
Pfizer



**JOONGHYUK FRANCIS SHEEN**  
BioNTech SE



**MOONHYEONG SEO**  
KIST (Korean Institute of Science and Technology)

**CV CLINIC**  
(NOV 4, 4:00 - 5:00 PM)

**5:00-6:30**  
**MEET WITH SPONSORS:**  
SK BIOSCIENCE/  
DAEWOONG PHARMACEUTICAL/  
GC BIOPHARMA

**CAREER WORKSHOP**  
(NOV 4, 8:30 - 9:30 PM)  
**FOR STUDENTS AND  
YOUNG PROFESSIONALS**



SPONSORED BY



## 1 차세대 위식도 역류질환 치료제 FEXUCLU (Fexuprazan)

- 과제시작부터 신약 허가단계까지 개발 전 과정 자체 역량으로 수행

### 대웅제약 '펙수클루정' 허가...국산 34호 신약 탄생

\* 편집부 | © 입력 2021.12.30 14:04 | 쉐닷컴 D

글로벌 기술수출 1.1조원 달성

미국, 중국 임상 3상 진행중

## 2 Well-balanced 신약 파이프라인 보유

- 소분자, biologic 신약 및 줄기세포 치료제

- First-in-class 위주의 프로그램으로 도전적 연구 수행

	연구단계	전임상	임상 1상	임상 2상	임상 3상	허가/발매
24 NCEs	DWJ218 <sup>1)</sup> 신약	DWP213235 <sup>2)</sup> 자기면역, crwn-β	DWP213388 <sup>2)</sup> 자기면역 질환(신약)	DWP305401 <sup>2)</sup> 백인신 대항염(신약)	Fexuprazan <sup>3)</sup> 위산	Fexuprazan <sup>3)</sup> 복합제(신약)
	DWP216 <sup>2)</sup> 포스페라	DWN12088 <sup>2)</sup> 진상/과다	DWP17061 <sup>2)</sup> #비면역 과다(신약)	DWN12088 <sup>2)</sup> 백인신 대항염(신약)	Fexuprazan <sup>3)</sup> NSC-391(신약)	inavoglican <sup>4)</sup> 2형당뇨
	DWP217 <sup>2)</sup> 면역질환	DWN12088 <sup>2)</sup> 과다, LD	DWP306 <sup>2)</sup> 4인		Enavo + Mel <sup>5)</sup> 2형당뇨	1)  대웅바이오시스
	DWP218 <sup>2)</sup> 면역성 근력	DWN12088 <sup>2)</sup> 면역성 근력(신약)	Fexuprazan <sup>3)</sup> 4인(신약)		Enavo + DPP4 i <sup>5)</sup> 2형당뇨	2)  코리아바이오 테라퓨틱스
	DWP220 <sup>2)</sup> 신약	DWP219 <sup>1)</sup> 수술용, 신약				3)  bridgebio
	A2A 공동연구 신약	Enavogilixozin <sup>6)</sup> 신약				4)  CC 바이오사이언스
7 Biologics - 6 Stem cells	HL190 <sup>7)</sup> 면역-신약	DWP457 <sup>2)</sup> 자기면역 질환(신약)	DWP710 <sup>2)</sup> 비면역성 질환(신약)	DWP700 <sup>2)</sup> 4인 신약	HL036 <sup>8)</sup> 면역-신약(신약)	5)  HANALL
	HL197 <sup>7)</sup> 면역-신약	DWP6203001 <sup>2)</sup> 자기면역 질환(신약)	Fireslem <sup>9)</sup> 신약	DWP706 <sup>2)</sup> 신약		6)  신성신약
	DWP458 <sup>7)</sup> 면역-신약	DWP6203009 <sup>2)</sup> 면역성 질환(신약)		Fireslem <sup>9)</sup> 면역성 질환(신약)		7)  코리아바이오 테라퓨틱스
	DWP700 <sup>2)</sup> 신약					8)  코리아바이오 테라퓨틱스

1) First-in-class 2) IND-ready (US FDA)

## 3 연구원 창업 활발 (사내벤처, 스피노프, Joint venture)



Cell & Gene  
Joint venture  
with Avacta (UK)  
New co (2019.12)



Ion-Channel based NCE  
New co (2020.05)



Depot DDS  
Start-up (2021.02)



Microbiome 신약  
Start-up (2021.05)



# GC Biopharma to the world right now! We bring new hope to patients around the world

**1983** - The world's 3rd hepatitis B vaccine

**1988** - The world's 1st epidemic hemorrhagic fever vaccine

**1993** - The world's 2nd varicella vaccine

**2010** - The world's 6th WHO prequalified pandemic (H1N1) influenza vaccine

**2011** - The world's 4th WHO prequalified seasonal trivalent influenza vaccine

**2016** - The world's 2nd WHO prequalified seasonal quadrivalent influenza vaccine



Since its establishment in 1967, GC Biopharma has consistently maintained a philosophy of taking the difficult but essential path, rather than the easier path. Now, GC Biopharma is going that extra mile by aiming to give new hope to people all around the world, not just those living in Korea. By combining its outstanding R&D capability for developing globally-recognized vaccines and blood derivatives with its differentiated solutions, GC Biopharma has set itself a new challenge to discover novel and much needed medicines and to become a trusted name, synonymous with protecting the health and happiness of people across the world.

A global leader in the healthcare industry - GC Biopharma Corporation



## Welcome to 2022 KASBP Fall Symposium

Korean American Society in Biotech and Pharmaceuticals (KASBP) welcomes you to the “2022 KASBP Fall Symposium” held in person from November 4<sup>th</sup> to November 5<sup>th</sup>, 2022 at Hilton Boston/Dedham. The theme of the symposium is the “**Post-Pandemic Pharmaceutical Industry; Forecasting the Trend of Life-Science, Technology, and Innovation**” and the event is hosted by Daewoong, GC Biopharma and SK Bioscience with the other honorable sponsors.

This year, exceptional speakers join the symposium in person, including our distinguished keynote speaker, Dr. Jae Park, Acting Chief of Cellular Therapeutics at Memorial Sloan Kettering Cancer Center. We are excited to announce that Dr. Park is selected as the recipient of “KASBP-Daewoong Achievement Award” for his contribution to the clinical research and development on CAR-T therapy. In addition, six great speakers and four panel discussion members from the industry also join us to share their experience and expertise in cutting-edge science from the early discovery research to the clinical development of the products.

We are also delighted to announce that eleven awardees are selected for KASBP Fellowships and eight awardees are selected for MOGAM-KASBP Scholarship, by generous support from our major sponsors. At the Job fair event, the qualified candidates have opportunities to interview with prominent Korean pharmaceutical and biotechnology companies.

With an inspiring theme and the exciting topics, the symposium organizing committee is genuinely looking forward to meeting all the members and colleagues.

November 4<sup>th</sup>, 2022

2022 KASBP Fall Symposium Organizing Committee

Program Chair, Ik-Hyeon Paik  
KASBP President, Seungwon Chung

## 2022 – 2023 KASBP Officers

Title	Name	한글 이름	Affiliation
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Advisory Committee	Past KASBP Presidents		

## Symposium Schedule at Glance (U.S. Eastern TIME)

		November 4 (Friday)	November 5 (Saturday)
AM	7		7:00 am – 8:00 am Registration / Breakfast
	8		8:00 am – 9:20 am    Scientific Session A
	9		9:20 am – 09:45 am    Sponsor Presentation II
			09:45 am – 10:00 am    Coffee Break
	10		10:00 am – 11:00 am Gene/Cell Therapy Panel session
	11		11:00 am – 12:30 pm Fellowship Awards Ceremony and MOGAM Scholarship Ceremony
	12		12:30 pm – 12:45 pm    Group Photo
	PM	1	
2			2:15 pm – 3:15 pm    Sponsor Presentation II
3		2:00 pm – 5:00 pm Job Fair / onsite interview  CV Clinic for YG (4:00 – 5:00 pm)	3:15 pm – 3:30 pm    Coffee Break
4			3:30 pm – 4:45 pm    Scientific Session B
			4:45 pm – 5:00 pm    Closing Remarks
5		5:00 pm – 6:20 pm Registration and Networking	5:00 pm    Networking and Dinner out (Registration required)
6		6:20 pm – 7:30 pm	
7		Opening & Congratulatory Remarks and Dinner	
8		7:30 pm – 8:30 pm Award Ceremony & Keynote Presentation	
9		8:30 pm – 9:30 pm Sponsor Presentation I	YG Career Workshop
10		9:30 pm – 11:30 pm	
11	Networking Session		

## Symposium Schedule In Detail (U.S. Eastern TIME)

### November 4 – 5, 2022, Friday – Saturday

**Job Fair** Organizer: Career Development Director, Min-Kyung Choo / Jooyoung Lee  
2:00 pm ~ 5:00 pm

### November 4, 2022, Friday

#### **CV Clinic for YG (Students/Young Professionals)**

4:00 pm ~ 5:00 pm Moderator: KASBP YG Director, Hyunjin Jung, Woodbridge Pharmacy

#### **Registration and Networking**

5:00 pm ~ 6:20 pm

#### **Opening & Congratulatory Remarks and Dinner**

6:30 pm ~ 7:30 pm Moderator: KASBP President Designated, Ik-Hyeon Paik, Wave Life Sciences

- **Opening Remark** KASBP President, Seungwon Chung, AbbVie
- **Congratulatory Remarks** 유기준 보스톤 총영사, 허은철 대표, 박준석 센터장, 이은정 팀장
- **Dinner**

#### **KASBP-Daewoong Award Ceremony and Keynote Lecture**

7:30 pm ~ 8:30 pm Moderator: KASBP President Designated, Ik-Hyeon Paik, Wave Life Sciences

- **Optimizing Performance of CAR T Cells in Hematologic Malignancies: Lessons Learned and Road Map for the Future**  
*Jae Park, MD. Acting Chief of Cellular Therapeutics at Memorial Sloan Kettering Cancer Center*

#### **Sponsor Presentation I**

8:30 pm ~ 9:30 pm Moderator: KASBP Philadelphia Chapter President, Kern Chang, Janssen R&D

- **Daewoong Pharmaceutical : Vision of Daewoong Pharmaceutical: Discovery and development of innovative drugs**  
*Joon Seok Park/ Head of New Drug Discovery Center*
- **GC Biopharma : Three Pillars of R&D at GC Biopharma**  
*Jae Uk Jeong/ SVP, Head of Research and Early Development*
- **SK Bioscience : SKBS 3.0 Next Generation: Our Science and Technology**  
*Hak Kim / Team Leader*

**YG Program/Career Development Workshop (in parallel)**

8:30 pm ~ 9:30 pm

Moderator: KASBP YG Director, Hyunjin Jung, Woodbridge Pharmacy

**Networking**

9:30 pm ~ 11:30 pm

Moderator: KASBP Washington DC Chapter President, Sungyong Hwang

- NW-01: Medicinal Chemistry / Drug Discovery & Delivery / Preclinical (Mod. Ik-Hyeon Paik, Wave Life Science)
- NW-02: Immunology-Oncology / Autoimmune / Inflammatory Diseases (Mod. Hyungwook Lim, Novartis)
- NW-03: Metabolic Diseases / Cardiovascular / Diabetes / Respiratory Diseases (Mod. Soo-Hee Park, Novartis)
- NW-04: Infectious Diseases / Vaccines / RNA Therapeutics (Mod. Ji-Young Min, GSK)
- NW-05: Neurological Disorders / Alzheimer's Disease / Parkinson's Disease / Aging (Mod. Seungkyu Lee, BMS)
- NW-06: Cell and Gene Therapy / Rare Diseases (Mod. Daniel C. Chung, SparingVision)
- NW-07: Business Development / Venture Capital / Legal / Consulting / Government Relations (Mod. Tae Heum Jeong, SV Bio Ventures)
- NW-08: Bioinformatics / A.I. / Machine Learning / Quantitative Science (Mod. Hyunjin Shin, MOGAM Institute)
- NW-09: CMC / Quality Assurance / Regulatory Affairs / Project Management (Mod. Sang Mok Chung, LG Chem)
- NW-10: Medical Device / In Vitro Diagnostics / Biomedical Engineering / Analytical Method Development (Mod. Stephen Kim, Qiagen)
- NW-11: Clinical Trial & Development / Clinical Pharmacology / Biostatistics (Mod. OhKyu Yoon, Gilead)

**November 5, 2022, Saturday****Registration and Breakfast**

7:00 am ~ 8:00 am

**Scientific Session A**

8:00 am ~ 09:20 am

Moderator: KASBP San Francisco Chapter President, Oh Kyu Yoon, Gilead Science

**A-1: Synthetic lethality: accelerating for personalized cancer treatment***Yang Lee, GSK***A-2: Directed evolution and engineering of CBE-T: next-generation cytosine base editors with minimized off-target editing***Matt Lee, Beam Therapeutics***A-3: Pooled screening platform for discovering the next generation Chimeric Antigen Receptors***Tae Yoon Kyung, Ginkgo Bioworks*



## Sponsor Presentation II

9:20 am ~ 9:45 am Moderator: KASBP Connecticut Chapter President, Sung Kwon Kim, Alexion

● **JW Pharma : JW's R&D strategy for innovative drugs.**  
*Kwangseok Ko/ Director of Research Strategy office*

● **KAIST-GCC**  
*Video presentation*

● **ITP-Yonsei**  
*Video presentation*

**Break** 09:45 am ~ 10:00 am

## KASBP- KHIDI USA Session - Gene and Cell Therapy Panel Discussion

10:00 am ~ 11:00 am Moderator: Jooyoung Lee, Senior Scientist at Vertex Pharmaceuticals

*Dan C. Chung, Chief Medical Officer at SparingVision*

*Junghae Suh, Vice President at Biogen*

## Award Ceremony and Presentation

11:00 ~ 12:30 pm Moderator: KASBP Fellowship Director, Hyung wook Lim

● **KASBP-Fellowship**

● **MOGAM-KASBP Scholarship**

## Group Photo

12:30 pm ~ 12:45 pm Moderator: KASBP Web Director, Hyelim Kim, PineTree Therapeutics

## Lunch, Poster session and Networking

12:45 pm ~ 02:15 pm

## Sponsor Presentation III

2:15 pm ~ 3:15 pm Moderator: KASBP Career Development Director, Min-Kyung Choo, Ingenia Therapeutics

- **Myriad Life Sciences : How to turn your science into a business- Corporate Venturing with Myriad Life Sciences**  
*Mina Lee / AC head*
- **NanoEntek : From the Lab to the Clinic (cell counting method and issue)**  
*Woo Young Sim, Head of Business Development*
- **CJ-Batavia Bioscience : Current application and development of novel adenovirus vectored vaccines**  
*Peter Abbink, Head or R&D / Director of Operations*
- **Dong-A ST**  
*Jaehong Park, President and CSO, Video presentation*
- **Deargen**  
*Video presentation*

**Break** 3:15 pm ~ 3:30 pm

## Scientific Session B

3:30 pm ~ 4:45 pm Moderator: KASBP Technical Director, Dooyoung Lee, Morpnic Therapeutics

- B-1: Process Development for Nirmatrelvir, a COVID-19 Oral Antiviral: Milligrams to Metric Tons in 18 Months**  
*Taegyo Lee, Pfizer Inc.*
- B-2: Proteomic approaches for identification and regulation of protein-protein interactions**  
*Moon Hyeong Seo, KIST*
- B-3: Translational research workflow for in-depth characterization of immune cells derived from the clinical trials**  
*Joong hyuk Sheen, BioNTech US*

## Symposium Closing Remarks

4:45 pm ~ 5:00 pm KASBP President, Seungwon Chung, AbbVie

## Dinner and Networking

5:00 pm ~

## Keynote Lecture

### Optimizing Performance of CAR T Cells in Hematologic Malignancies: Lessons Learned and Road Map for the Future

*Jae Park, MD, Acting Chief of Cellular Therapeutics at Memorial Sloan Kettering Cancer Center*

#### Biography

Dr. Park, widely recognized as one of the world's experts in cell therapies, is the leading principal investigator of several clinical trials in patients with non-Hodgkin lymphoma, chronic lymphocytic leukemia and acute lymphoblastic leukemia. Dr. Park's groundbreaking research and experience treating patients with CAR-T and other novel immunotherapies lead the Cancer Center to be the leader in developing a treatment platform for patients with blood cancers and solid tumors. Dr. Park received his medical degree from the Johns Hopkins School of Medicine and completed an internal medicine residency at Massachusetts General Hospital and a hematology/oncology fellowship at Memorial Sloan Kettering Cancer Center. Dr. Park has published numerous peer-reviewed articles appearing in *New England Journal of Medicine*, *Science Translational Medicine*, *Blood*, *Cancer Discovery* and *Journal of Clinical Oncology*. His research focused on establishing novel targeted and immunotherapies for patients with hematologic malignancies has been recognized and funded by American Society of Hematology, American Association for Cancer Research, American Society of Clinical Oncology, Leukemia and Lymphoma Society, Geoffrey Beene Research Foundation, and National Comprehensive Cancer Network.

#### Abstract

Tremendous progress has been made in the field of CAR T cell therapy, now with several approved autologous CAR T cell products for treatment of lymphoma, leukemia and myeloma. However, only a small subset of patients are able to achieve long-term remissions. In this talk, I will review the history and initial development of CD19 CAR T cell therapy in various hematologic malignancies, clinical data to date, current unmet need and emerging next generation products including armored CAR T cells and allogeneic CARs as well as approaches to improve the toxicity of these cellular immunotherapeutic.

## Scientific Session

### SESSION A

#### A-1: Synthetic lethality: accelerating for personalized cancer treatment

*Yang Lee, GSK.*

#### Biography

Yang Lee is a drug discovery professional with experience in planning, designing, and executing preclinical and translational projects in oncology drug discovery programs in GSK. He has been working on the drug discovery process, including target selection, in vitro and in vivo pharmacology, pharmacodynamic marker identification, pharmacokinetic/pharmacodynamic relationship, and patient stratification strategies. Before joining GSK, Yang was a postdoctoral research fellow in Vascular Biology Program at Boston Children's Hospital and studied how lymphangiogenesis protected against metabolic disease. During his postdoctoral training, he received an AHA postdoctoral fellowship and NIH F32 fellowship. He completed his Ph.D. in Medical Science/ Lymphatic Biology at Texas A&M Health Science Center. He received a Lymphatic Biology Fellowship and doctorate work focused on the molecular mechanisms of lymphatic dysfunction and immune cell alteration in metabolic diseases.

#### Abstract

Synthetic lethality occurs between two genes when silencing of either gene alone enables viable, but inhibition of both is lethal. Synthetic lethality is a precision treatment that provides safe and sustainable results for cancer patients. It has been showing signs of helping various cancers that were previously thought of as "undruggable." The synthetic lethality concept was established in early 1920 by Calvin Bridges from observation of fruit flies (*Drosophila melanogaster*) that showed normal phenotype in single mutation, but a certain combination of a non-allelic gene was lethal. It is only since 2005 that synthetic lethal interactions have been translated into the clinic for cancer patients. Synthetic lethality displays a new wave of genetically selective cancer medicines. Precision medicine could help treat cancer safely by selective targeting cancer cells, leaving a patient's healthy cells unaffected. We have developed new technologies that change our approach and enhance our understanding of the research to discover new synthetic lethal targets. We have used CRISPR-based tools that provide a path forward where 'hits' (i.e. potential drug targets) can be generated, and the patient population is expected to benefit from inhibiting each target. We have discovered compelling combinations and the opportunity to build an industry-leading pipeline targeting several major solid tumors. Our strategy and innovative precision medicine therapy platforms via synthetic lethality could have a meaningful and safe impact for patients with, but not limited to, lung, prostate, breast, colorectal, and ovarian cancers.



**A-2: Directed evolution and engineering of CBE-T: next-generation cytosine base editors with minimized off-target editing**

*Matt Lee, Beam Therapeutics Inc.*

**Biography**

Matt Lee is a Director and the Head of Protein sciences at Beam Therapeutics Inc. He received a B.S. degree in 2002 from University of Canterbury, Christchurch, New Zealand. He then obtained a Ph.D. degree from Case Western Reserve University, Cleveland, Ohio under the guidance of Prof. Vivien C. Yee. During his undergraduate and Ph.D. studies Matt investigated structural consequence of disease-related mutations in human prion protein using X-ray crystallography, AFM, and various biophysical methods. Currently he is co-leading Beam Therapeutics' Gene Editing Platform Technology group, working on CRISPR/Cas9-based gene editing enzyme and other therapeutically relevant proteins.

**Abstract**

Base-editing is a next-generation genome editing technology that conducts programmable chemistry on the genome without inducing double-stranded breaks. Base editors are composed of a modified form of the CRISPR-associated nuclease Cas9 and a deaminase, and chemically convert one base into another via hydrolytic deamination of an exo-cyclic amine of a targeted nucleobase. There are two main classes of base editors: adenine base editors (ABEs) and cytosine base editors (CBEs) that catalyze irreversible conversion of A:T base pair into G:C, and C:G base pair into T:A, respectively. CBEs typically utilize a naturally occurring cytidine deaminase from APOBEC enzyme family, which are, unlike ABEs, known to cause unguided genomic cytosine deamination. Recently, new variations of CBEs that reduce guide-RNA independent off-targets have been reported, but they also show lower on-target editing efficiency. Here, we report next-generation cytosine base editors, CBE-Ts, that utilize an evolved and engineered Tada enzyme capable of catalyzing C-to-T conversion at levels comparable to APOBEC-derived CBEs with no substrate sequence biases and no detectable guide-independent off-target. Together with ABEs, CBE-Ts enable the programmable installation of all transition mutations using laboratory-generated Tada enzyme variants with greatly reduced off-target deamination relative to naturally occurring enzymes.

**A-3: Pooled screening platform for discovering the next generation Chimeric Antigen Receptors**

*Taeyoon Kyung, Ginkgo Bioworks*

**Biography**

Dr. Kyung is currently leading Chimeric Antigen Receptor (CAR) engineering group at Ginkgo Bioworks where his team is focused on early discovery work of novel CAR designs in various immune cell types. Previous to Ginkgo, Dr. Kyung contributed to optimizing synthetic molecule designs and function of off-the-shelf CAR-NK cell product at Catamaran Bio. Dr. Kyung received his B.S. and Ph.D. at KAIST where he developed optogenetic calcium channel switch to elucidate how calcium ions in neurons contribute to memory formation in the hippocampus. Then, he moved to MIT as a Postdoctoral Fellow to apply his expertise in synthetic molecule design and screening in cell therapy space where he generated large-scale CAR lentiviral library to screen for the next generation CAR-T cells.

**Abstract**

Signaling cascades triggered by intracellular domains (ICDs) of Chimeric Antigen Receptor (CAR) in immune cells drive cell behaviors that correspond to different therapeutic outcomes. While canonical CAR-T ICD combinations 4-1BB-CD3z (BBz) and CD28-CD3z (28z) have achieved clinical success in a limited number of hematologic indications, CAR-based cell therapies have not yet been successful in more challenging indications. Systematic discovery of novel ICD combinations that drive more favorable T cell phenotypes has been onerous due to technical constraints in high-throughput screening and scaling. Here, we developed a versatile platform that allows parallel comparison of 10,000 different 2nd generation CAR ICD combinations in primary CD8+ T cells. We utilized combinatorial genetics en masse (CombiGEM) to assemble multiple DNA-barcoded lentiviral libraries of CAR ICDs distinguished by the affinity of extracellular binder and the length of hinge/spacer. We screened CAR ICD libraries for T cell persistence and fitness in a serial tumor rechallenge assay in a pooled manner. Precisely tracking enriched barcodes over the course of 20 days allowed us to identify novel CAR clones with superior T cell proliferation and survival relative to conventional BBz and 28z CARs. We believe our approach is readily applicable to diverse immune cell types, including T cell subsets, NK cells and macrophages, as well as different CAR domains including binders, structural components, and armoring components in addition to ICDs. Rapid and systematic generation and comparison of synthetic molecules at scale in pooled libraries via our platform will further elucidate design principles to guide next generation cell therapies engineering strategies.

**SESSION B****B-1: Process Development for Nirmatrelvir, a COVID-19 Oral Antiviral: Milligrams to Metric Tons in 18 Months**

*Taegyo Lee, Pfizer Inc.*

**Biography**

Taegyo Lee is a Principal Scientist at Pfizer Chemical Research and Development in Groton, CT. He received a B.S. degree in 2013 from Seoul National University in South Korea where he performed undergraduate research with Prof. Chulbom Lee. He then obtained a Ph.D. degree from the University of California, Berkeley under the guidance of Prof. John F. Hartwig. During his undergraduate and Ph.D. studies, Taegyo developed transition metal-catalyzed reactions and studied their mechanisms. Since he joined Pfizer in 2018, he has contributed to successful development of manufacturing processes on multiple programs ranging from early clinical space through commercialization.

**Abstract**

nirmatrelvir is a potent, selective and orally bioavailable inhibitor of SARS-CoV-2 M<sup>pro</sup>. The successful development and scale-up of an efficient chemical synthesis of nirmatrelvir allowed for progression of Paxlovid™ (nirmatrelvir tablets; ritonavir tablets) from first laboratory synthesis to emergency use authorization in just 18 months, a new record for Pfizer and the pharmaceutical industry. During this seminar, I will discuss our development efforts for the commercial synthesis of nirmatrelvir. Key synthetic and logistical challenges will be presented.

Paxlovid™ has not been approved but has been authorized for emergency use by the FDA to treat mild-to-moderate COVID-19 in patients 12 and older, weighing at least 40 kg, with positive results of SARS-CoV-2 viral testing, who are at high risk for progression to severe COVID-19, including hospitalization and death. Authorized only for the duration of the declaration that circumstances exist justifying the authorization unless the declaration is terminated, or authorization revoked sooner

## **B-2: Proteomic approaches for identification and regulation of protein-protein interactions**

*Moon-Hyeong Seo, KIST*

### **Biography**

Dr. Seo received B.S. and Ph.D. degree at KAIST. During his doctoral studies, he developed novel methods for efficient analysis of protein-protein and protein-ligand interactions, which he used to investigate the role of protein dynamics in protein-ligand interactions. After he joined Dr. Philip M. Kim's lab at University of Toronto as a postdoctoral fellow, he integrated computational and experimental approaches for proteome-wide identification and regulation of protein-protein interactions. Dr. Seo is currently a senior research scientist at KIST Gangneung, and his primary research interests are the development of immunomodulatory peptide therapeutics and protein-based biosensors.

### **Abstract**

As proteins perform pivotal roles in cells, tightly and accurately controlled interactions among proteins orchestrate essential cellular processes, from proliferation to cell death. Thus, not surprisingly, protein-protein interactions (PPIs) have caught particular attention for deeper understanding of cellular mechanisms as well as for the drug discovery to various types of diseases. Nevertheless, the motif-mediated protein-protein interactions are the most underrepresented types of interactions in available interaction data, largely due to technical difficulties inherent in the properties of short motifs. As the increasing recognition of the vital roles of PPIs in biology, systematic approaches have recently been applied to detect novel binding surfaces by short linear motifs. In the presentation, peptide-based proteome-scale strategies to identify novel PPIs are introduced. Which not only prove to be powerful for identifying low-affinity interactions, but are further harnessed to develop immunomodulatory peptide therapeutics.

## **B-3: Translational research workflow for in-depth characterization of immune cells derived from the clinical trials**

*Joong Hyuk Francis Sheen, BioNTech US*

### **Biography**

Dr. Sheen is a trained immunologist with global experience in the bio-pharma industry. He has been interested in deciphering mechanisms of disease states associated with inflammation ranging from organ transplantation to immuno-oncology. He is a senior scientist in the Translational Immunology team at the BioNTech US since 2020, contributing to establishing and expanding biomarker and translational research

strategy employing multidisciplinary approaches. He actively participates in organizations such as the American Association for Cancer Research (AACR), Society for Immunotherapy of Cancer (SITC) and American Association of Immunologists (AAI). He received the Young Investigator Awards at the American Transplant Congress for two consecutive years (2015, 2016), and the Achievement Fellowship Award at the Icahn School of Medicine at Mount Sinai (2017). Prior to joining BioNTech US, he was an investigator at the MOGAM Institute for Biomedical Research in South Korea. He received a B.S. degree from the University of Washington and a Ph.D. degree in immunology from the Icahn School of Medicine at Mount Sinai.

### **Abstract**

Studying the impact of immunotherapies on the patients in the clinical trials and underlying their mechanisms of response and resistance are the key goals of implementing translational research strategies. In a Ph1B clinical study, a personalized neoantigen peptide vaccine, in combination with chemotherapy and pembrolizumab, shows safety and immunogenicity in patients with metastatic nonsquamous non-small-cell lung cancer. The regiment induces durable vaccine-specific T cell responses. Single-cell RNA-sequencing analysis further reveals thorough profiling of immune cells derived from the patient samples at the post treatment time point where neoantigen-specific CD4<sup>+</sup> T cells have an activated effector phenotype, as well as a cytotoxic gene signature. This multiomics-based immune monitoring workflow can be implemented for deeper understanding of phenotype and function of a given cell within the context of diverse biological samples across multiple indications.

### **KASBP- KHIDI USA Session - Gene and Cell Therapy Panel Discussion**

*Moderator: Jooyoung Lee, Vertex Pharmaceuticals*

Dr. Jooyoung Lee received her B.S. in Biomedical Sciences with a minor in Biochemistry at Minnesota State University. She then obtained a Ph.D. from University of Massachusetts Medical School in the Graduate School of Biomedical Sciences. During her doctoral training with Dr. Erik Sontheimer at the RNA Therapeutics Institute, Dr. Lee co-discovered the first anti-CRISPR proteins which inhibit CRISPR-Cas9. While a Nobel-prize-winning CRISPR technology revolutionized various fields of science from a gene drive to potential therapeutics, safety issues demand development of a system to control Cas9. Being part of the co-discovery of anti-CRISPR proteins, Dr. Lee's thesis research was to repurpose these inhibitors as potent off-switches for Cas9. In 2020, Dr. Lee joined Vertex in the department of Cell and Genetic Therapies as a gene editing research scientist to develop transformative gene therapies for hereditary diseases.

*Panel : Daniel C. Chung, Chief Medical Officer at SparingVision*

Dr. Chung is the Chief Medical Officer for SparingVision, an ocular genomic medicine company, focusing on gene agnostic gene therapy and CRISPR gene editing approaches to combat blinding diseases. Prior to his recruitment to SparingVision, Dr. Chung was the Ophthalmology Therapeutic Leader for Spark Therapeutics, where he led the medical affair group and contributed to areas of clinical development and operations, marketing, commercial, patient advocacy, pre-clinical research and development and business



development. Dr. Chung was intimately involved with the development of Luxturna, the first gene therapy approved by the FDA and EMA for use in a blinding genetic disease.

Prior to joining Spark Therapeutics, he was a senior investigator/instructor at the FM Kirby Center for Molecular Ophthalmology at the Scheie Eye Institute at the Perelman School of Medicine of the University of Pennsylvania, working in retinal gene transfer and therapy. Concurrently, he served as the scientific advisor on the *RPE65* gene therapy study team for phase 1 and 3 of the clinical trial at the Children's Hospital of Philadelphia (CHOP).

He was the lead designer of the Phase 3 MLMT novel endpoint, PI of the MLMT study, and PI of the RPE65 Natural History Study. He completed his ophthalmology residency in Akron, Ohio. He then completed fellowships in pediatric ophthalmology and ocular genetics research at the Cole Eye Institute at the Cleveland Clinic, and in retinal gene therapy at the National Eye Institute/NIH in Bethesda, MD

*Panel : Junghae Suh, Vice President at Biogen*

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.

*Panel : Byoung Ryu, Executive Vice President at Umoja Biopharma*

Byoung Ryu is the Executive Vice President of Discovery Research and Vector Biology at Umoja. Byoung brings more than two decades of experience in cell and gene therapy research and product development. Before joining Umoja, Dr. Ryu was the Vice President of Vector biology at Lyell Immunopharma where he established lentiviral vector system and manufacturing. Dr. Ryu received his Ph.D. from the University of Tennessee, Health Science Center and trained as a postdoc at St. Jude Children's Research Hospital working on lentiviral safety for gene therapy application. He joined bluebird bio in 2010 where he developed two clinical lentiviral vectors, LentiGlobin BB305 (ZYNTEGLOTM) for b-thalassemia and Sickle Cell Disease and bb2121 CAR vector for multiple myeloma, both which are in clinical trials and under BLA licensing review at FDA.

## Poster Session

### 2022 Fall KASBP Fellowship Awardees

Award Name	Awardee	Affiliation
KASBP- DAEWOONG Fellowship	Inyoung Jung	University of Pennsylvania
KASBP- DAEWOONG Fellowship	Lee Joon Kim	Lawrence Berkeley National Laboratory
KASBP- DAEWOONG Fellowship	Benjamin Lew	University of Illinois Urbana-Champaign
KASBP- GC Biopharma Fellowship	Thomas TaeHyun Kim	Picower Institute (MIT)
KASBP- GC Biopharma Fellowship	Jaehyeok Jin	Columbia University
KASBP- GC Biopharma Fellowship	Seung Hun Park	Massachusetts General Hospital (Harvard Medical School)
KASBP-SK bioscience Fellowship	Woo Yong Park	National Cancer Institute
KASBP-SK bioscience Fellowship	Sungwook Jung	Brigham and Women's Hospital (Harvard Medical School)
KASBP-SK bioscience Fellowship	Yunju Jeong	Brigham and Women's Hospital (Harvard Medical School)
KASBP- KAIST-GCC Fellowship	Hyejoong Jeong	University of Pennsylvania
KASBP- ITP-Yonsei Fellowship	Jae Kyo Yi	Dana-Farber Cancer Institute (Harvard Medical School)

### 2022 Fall MOGAM-KASBP Scholarship Awardees

Awardee	Current Institution
Yehlin Cho, PhD Graduate student	Massachusetts Institute of Technology
Jayoung Ryu, PhD Graduate student	Harvard University
Jee Won Yang, PhD Graduate student	California Institute of Technology
Hyoann Choi, PhD Graduate student	Georgia Institute of Technology
Sookyung Kim, MD/PhD MSTP student	University of Massachusetts Medical School
HyeRin, Leah Yim, PhD Graduate student	Icahn School of Medicine at Mount Sinai
Byunggik Jason Kim, PhD Graduate student	Johns Hopkins University
Sally Chung, Undergraduate student	Johns Hopkins University

**P-1: BLIMP1 and NR4A3 Transcription Factors Reciprocally Regulate Tumor-directed CAR T-cell Stemness and Exhaustion****Inyoung Jung, University of Pennsylvania**

Chimeric antigen receptor (CAR) T-cells have not induced meaningful clinical responses in solid tumor indications. Loss of T-cell stemness, poor expansion capacity and exhaustion during prolonged tumor antigen exposure are major causes of CAR T-cell therapeutic resistance. scRNA sequencing analysis of CAR T-cells from a first-in-human trial in metastatic prostate cancer identified two distinct and independently validated cell states associated with antitumor potency or lack of efficacy. Low levels of the PRDM1 gene encoding the BLIMP1 transcription factor defined highly potent TCF7+ CD8+ CAR T-cells, while enrichment of TIM3+CD8+ T-cells with elevated PRDM1 expression predicted poor outcome. PRDM1 single knockout promoted TCF7-dependent CAR T-cell stemness and proliferation resulting in marginally enhanced leukemia control. However, in the setting of PRDM1 deficiency, a negative epigenetic feedback program of NFAT-driven T-cell dysfunction characterized by compensatory upregulation of NR4A3 and multiple other genes encoding exhaustion-related transcription factors hampered effector function in solid tumors. PRDM1 and NR4A3 combined ablation skewed CAR T-cell phenotypes away from TIM3+CD8+ and toward TCF1+CD8+ to counter exhaustion of tumor-infiltrating CAR T-cells and improve in vivo antitumor responses, effects that were not achieved with PRDM1 or NR4A3 single disruption alone. These data reveal a novel molecular targeting strategy to enrich stem-like CAR T-cells resistant to exhaustion and underscore dual inhibition of PRDM1/NR4A3 expression or activity as a promising approach to advance adoptive cell immuno-oncotherapy.

**P-2: Aiding Drug Discovery and Developmental Efforts Using Scattering-Based Methods****Lee Joon Kim, Lawrence Berkeley National Laboratory**

Structural knowledge of molecules has guided drug discovery and development for decades, enabling rational design of novel therapeutics. As increasingly challenging biomolecules are targeted with highly complex drug molecules, integrating different techniques becomes necessary to develop a holistic understanding of their structures and interactions. To this end, numerous methods have been utilized, such as nuclear magnetic resonance (NMR), X-ray crystallography, and cryo-electron microscopy (cryoEM), each possessing unique advantages and disadvantages for various applications. One method that can complement these existing techniques is electron diffraction, which uses crystals that are orders of magnitude smaller than those used in single crystal X-ray diffraction studies. Electron crystallography has demonstrated great synergy with genome mining to unambiguously determine full structures of biologically relevant natural products, such as fischerin, an orphan natural product that has eluded complete structural characterization since its isolation in 1993. By elucidating structures of these small molecules, electron diffraction can accelerate the rates of novel natural product discovery and, consequently, drug discovery campaigns that rely on novel scaffolds and mechanisms of action. The high sensitivity of electron diffraction could also be utilized to screen for trace amount of impurities contained in a sample. Finally, small angle X-ray scattering (SAXS) will be briefly introduced as another complementary technique for studying samples in solution rather than in crystalline phases, enabling detections of biomolecules' flexibility or environmentally induced conformational changes to develop a better understanding of drug targets. Combined, these two scattering-based methods provide additional advantages and information that can help develop a more comprehensive knowledge of chemical and biological systems for discovery and development of pharmaceuticals.

**P-3: Tumor-specific fluorophores and stimulus-responsive nanoprobes for intraoperative imaging of primary tumors****Benjamin Lew, University of Illinois Urbana-Champaign**

Cancer is one of the leading causes of death worldwide. Surgery is the oldest kind of cancer treatment, and it remains the primary curative option for localized tumors. For the successful treatment of various cancers including breast, colon, and lung, it is crucial to detect and remove malignant tissues in their early-stage development. In this regard, fluorescence-guided surgery (FGS) using near-infrared (NIR) molecular imaging provides invaluable information on the location of primary tumors and their metastatic progression to ensure complete tumor resection. The advancement in imaging sensors has contributed to the development of intraoperative NIR imaging modalities with high spatial resolution and fluorescence sensitivity to aid surgeons in the operating room in real-time. Various fluorophores in a form of antibodies, peptides, affibodies, and nanoparticles have emerged as promising NIR imaging tracers to highlight tumors' unique features including overexpressed biomarkers, low extracellular pH, and upregulation of tumor-associated enzymes. However, employing a single fluorophore is often not sufficient to anticipate consistent clinical outcomes in FGS due to the spontaneous and heterogeneous nature of cancer development. For instance, different stages of metastatic progression could lead to the upregulation of biomarkers different from those of the primary tumors. Furthermore, it is critical to accurately highlight the tumor margins to avoid damaging any adjacent vital organs and vasculatures while removing residual tumors. Hence, the ability to detect fluorescence signatures from different tumor-targeted fluorophores is a highly desirable feature in FGS. Herein, we present an overview of our latest results regarding multiple fluorophores for intraoperative tumor imaging. Various NIR fluorophores conjugated with different types of tumor-targeted agents (affibody, cyclic-peptide, and nanoparticle) were prepared and their uptake behavior in different cancer cell lines were monitored using the In Vivo Imaging System (IVIS). Subsequently, we employed an intraoperative NIR imaging sensor to detect multiple fluorophores with a single excitation source and monitored the contrast between the tumors and adjacent healthy tissues in small animal xenograft models.

**P-4: Gamma entrainment using sensory stimuli alleviate chemo brain pathology and cognitive impairment induced by chemotherapy agents****Thomas TaeHyun Kim, Picower Institute (MIT)**

Cancer patients undergoing chemotherapy treatments often suffer from a neurological condition called chemotherapy related cognitive impairment, or chemo brain, which may persist for the rest of their life. Despite the increasing number of chemo brain patients, neither mechanism nor treatment of chemo brain is well explored. Recent findings indicate that chemo brain shares multiple features with neurodegenerative diseases, such as chronic neuroinflammation, DNA damage, and synaptic loss. We tested if Gamma ENtrainment Using Sensory stimuli (GENUS), which has been shown to ameliorate Alzheimer's disease (AD) pathology in mouse models of AD, can be utilized to treat chemo brain. When given with cisplatin, GENUS alleviated cisplatin-induced pathology, promoted oligodendrocyte survival, and led to significant improvement of cognitive functions in a mouse model of chemo brain. Furthermore, we show that the effect of GENUS is not limited to cisplatin-induced chemo brain, but also applies to methotrexate (MTX)-induced symptomology, suggesting that GENUS could be a versatile treatment approach for a wide range of chemo brain patients.



**P-5: Overcoming the Multiscale Simulation Challenge for Biomolecular Systems Toward Drug Discovery and Pharmaceutical Design****Jaehyeok Jin, Columbia University**

Recent advances in theoretical and computational methodologies have aimed at studying biomolecular systems across multiple length and time scales. Since experimentally probing detailed mechanisms (e.g., drugs or proteins) remains a great challenge, predictive multiscale modeling is necessary to fill in the gaps in our understanding. The present approach provides a systematic design principle for high-fidelity multiscale computational models derived from underlying atomistic information. At the heart of the approach is the coarse-graining method by integrating away the unnecessary details from atomistic simulations. Nevertheless, several challenges significantly affect the quality and performance of coarse-grained models applied for complex (biomolecular and other soft matter) systems. Here, I will address these problems using advances in modern computer architecture with fundamental theories from biology, chemistry, and physics. This presentation will delineate how the bottom-up multiscale approach can be faithfully applied from the smallest molecular scale of self-assembly to mesoscopic phenomena of large biomolecule complexes. Particularly, applications to experimentally important targets in pharmaceuticals and drug discoveries such as a polypeptide, endophilin amphipathic helix, integrin protein, and HIV-1 capsid protein will be discussed.

**P-6: Injectable thermosensitive hydrogels for a sustained release of iron nanochelators****Seung Hun Park, Massachusetts General Hospital (Harvard Medical School)**

Deferoxamine (DFO) is an FDA-approved iron-chelating agent and shows good therapeutic efficacy; however, its short blood half-life has been a challenge that requires repeated injections or continuous infusions. Considering the lifelong need of chelating agents in iron overload patients, a sustained-release formulation that can reduce the number of chelator administrations is essential. Here, we report injectable hydrogel formulations prepared by integrating crosslinked hyaluronic acid into Pluronic F127 for an extended release of DFO nanochelators. The subcutaneously injected hydrogel showed thermosensitive sol-gel transition at the body temperature and provided a prolonged release of renal clearable nanochelators over 2 weeks, of which half-life is 47-fold longer than that of the nanochelator alone. In addition, no chronic toxicity of the nanochelator-loaded hydrogel was confirmed by biochemical and histological analyses. This injectable hydrogel formulation with DFO nanochelators has the potential to be a promising formulation for the treatment of iron overload disorders.

**P-7: Apoptosis-induced nuclear expulsion in tumor cells drives S100a4-mediated metastatic outgrowth through the RAGE pathway****Woo Yong Park, National Cancer Institute**

Metastasis is a process marked by massive amounts of cell death, with only the fittest tumor cells surviving to colonize distant organs. There are different forms of cell death with distinct morphological, molecular, and genetic features. Apoptotic cell death, a seemingly beneficial therapeutic outcome, however, can be harnessed by tumor cells to enhance metastatic functions. The underlying mechanisms have been perplexing, and it is now increasingly apparent that apoptotic cells influence nearby tumor cells through

the release of mitogen signals, extracellular vesicles, inflammatory mediators, and metabolites. Yet, it remains unclear what happens to chromatin that is released from dying tumor cells and how it might affect nearby live tumor cells. Here we discovered that apoptotic cancer cells enhance the metastatic outgrowth of surviving cells through Padi4-mediated nuclear expulsion, a process we term exsporosis deriving from the Greek word *ekspo*, meaning to break out and *sporos*, meaning seed or nucleus. The resulting extracellular complex of chromatin and its associated proteins will be referred to as exsporosi. We demonstrate chromatin bound S100a4 in exsporosi activates the RAGE receptor on neighboring tumor cells and enhances their metastatic outgrowth through Erk activation in the lung. In addition, we identified exsporosi in human tumor lesions from breast, bladder, and lung cancer patients and an exsporosis signature, consisting of genes regulated by exsporosi, correlated with poor prognosis of metastasis. Collectively, our studies uncover a novel mechanism where apoptotic tumor cells undergo a previously unreported nuclear expulsion which enhances the metastatic outgrowth of surviving tumor cells through chromatin-bound RAGE ligand, S100a4. This provides the insight that cancer cells could constitute populationbased survival mechanisms in addition to ways for individual survival, as well as the full repertoire of apoptotic signals that dying cells give as “last words” to the cancer cell community.

#### **P-8: Epitopic high endothelial venule-targeted nanodelivery for type 1 diabetes**

**Sungwook Jung, Brigham and Women’s Hospital (Harvard Medical School)**

Targeted drug delivery systems hold the remarkable potential to improve the therapeutic index of 5 diabetes medications. Herein, we developed a targeted delivery platform for type 1 diabetes (T1D) 6 treatment using high endothelial venule (HEV)-targeted nanoparticles. We encapsulated anti-CD3 7 in PLGA nanoparticles (NPs) and conjugated mAb on the surface of NPs. Our mAb-NP localized 8 to pancreatic lymph nodes (PLNs) and pancreata in NOD mice and encapsulation of anti-CD3 in 9 MECA79-NP enhanced its delivery to these organs. Treatment of hyperglycemic NOD mouse 10 model with mAb-anti-CD3-NP resulted in significant reversal of T1D, as compared to non11 treatment, empty NP, and free anti-CD3. mAb-anti-CD3-NP treatment caused a marked increase 12 of regulatory T (Treg) cells in pancreata. Our data suggested that ectopic HEV expressed in 13 pancreata of T1D patients. Our study demonstrates that HEV-targeting nanovehicles constitute a 14 novel drug delivery platform that can augment the effects of immunosuppression as well as reduce 15 the inflammatory risk in treating T1D. Moreover, HEV-targeting therapeutics may be used as 16 means by which drugs are delivered specifically to PLNs and pancreata, thereby prolonging the 17 reversal of T1D patients.

#### **P-9: Autocrine LIF-LIFR loop in fibroblasts drives recruitment of Granzyme K+ T cells in earlier-stage ILD**

**Yunju Jeong, Brigham and Women’s Hospital (Harvard Medical School)**

**Rationale** To define molecular mechanisms in early ILD, we first applied single-cell RNA sequencing (scRNA-seq) to surgical lung biopsies from patients with earlier stage of ILD and validated candidate mechanisms in vitro in lung fibroblasts from patients with early ILD. **Methods** We obtained lung tissues from patients with early ILD (N=7, Video-assisted thoracoscopic surgery [VATS] biopsies) or late-stage Idiopathic Pulmonary Fibrosis (IPF, N=4, explant); or from donor controls (N=4). We applied scRNA-seq (10X Genomics) to disaggregated lung tissue, and analyzed data with Seurat v4 and CellPhoneDB. For in vitro validation, we cultured primary lung fibroblasts from early ILD and healthy lung tissues. We profiled primary early ILD lung fibroblasts in vitro by Q-PCR, ELISA, and ultra-low input (ULI)-RNA-seq with standard analysis pipelines. **Results** Differential abundance analysis in the scRNA-seq data demonstrated that T cells

were expanded in early ILD lung compared to control or late-stage IPF. CellPhoneDB interactome analysis suggested that fibroblasts produce CXCL12 to recruit CXCR4+ T cells to early ILD lung. We validated this axis with in vitro migration assays of T cells and lung primary fibroblasts. Moreover, primary lung fibroblast cell lines from ILD patients exhibit an autocrine LIF-LIFR loop that drives expression of CXCL12. In differential gene expression analysis, CXCR4+ T cells in early ILD had increased expression of granzyme K (GZMK), IFNG, and TNFA. To understand the potential effector role of the CXCR4+ T cells, we treated primary early ILD fibroblasts in vitro with combinations of GzmK, IFNg and TNFa for analysis by ULI-RNA-seq. GzmK synergized with IFNg and TNFa to drive a specific inflammatory program in early ILD fibroblasts. This GzmK-dependent inflammatory program features CCL7 and CCL8. The GzmK-dependent, inflammatory gene program in early ILD fibroblasts is inhibited in vitro by baricitinib and ruxolitinib, two oral JAK inhibitors in wide clinical use for other diseases. **Conclusion** Our study defines the global transcriptome of earlier stage ILD lung at single-cell resolution. Autocrine LIF drives CXCL12 expression by fibroblasts, which recruit CXCR4+ GZMK+ T cells. GzmK synergizes with IFNg and TNFa to drive a gene signature that may inform biomarkers and therapeutic targets for early ILD.

### **P-10: Nanocloak for Endothelial Cells Delivering Nitric Oxide and Growth Factor for Angiogenesis**

**Hyejoong Jeong, University of Pennsylvania**

Myocardial infarction (MI) is a representative disease that requires mesenchymal stem cell (MSC) transplantation to resolve myocardial ischemia by improving angiogenesis. To improve the angiogenic effect, the cell coating technique can apply functional molecules to individual cells. Layer-by-layer (LbL) assembly technique is based on molecular interactions among macromolecules and creates very thin nanocoating—"nanocloak"—on cells. Nitric oxide (NO)—an intrinsic cellular signaling molecule—plays an essential role in maintaining homeostasis of the cardiovascular system and enhancing vasodilation, angiogenesis, and inhibition of platelet aggregation. Vascular endothelial growth factor (VEGF) is the most important factor in angiogenesis. In this study, we reported nanocloak developed by the LbL assembly technique on individual endothelial cells (ECs), which is leveraged as a platform for applying VEGF and NO to cells. The nanocloak was composed of VEGF, fibronectin, heparin, and synthesized S-nitrosothiol-bound polyacrylic acid which provides an endothelial level of NO in physiological conditions. As angiogenic factors, the synergistic effects of VEGF and NO were investigated through cell viability, proliferation, cell migration, and tubule formation—prerequisites of angiogenesis. This is the first report on nanocoating that delivers NO and VEGF to cells simultaneously and investigates their synergistic effects in terms of angiogenesis. For future follow-up studies, this nanocoating system can be applied to MSCs for MI treatment.

### **P-11: Enhancement of anti-tumor immunity in ER+ breast cancer through dual inhibition of MCL1 and caspases**

**Jae Kyo Yi, Dana-Farber Cancer Institute (Harvard Medical School)**

While immunotherapy has revolutionized cancer care in recent years, there has been very limited success in breast cancer with only a small subset showing efficacy to immune checkpoint inhibitors. One strategy that has been proposed to enhance anti-tumor response is acute upregulation of Type I Interferon (IFN) response, which promotes increased expression of MHC Class I molecules. Type I IFN response can be highly activated by releasing the inner contents of mitochondria inhibition of Myeloid Cell Leukemia-1 (MCL1). A body of literature has suggested that Type I IFN response may be activated by release of damage-associated molecular patterns (DAMPs) from mitochondria which can be induced through. However, when

DAMPs are released from mitochondria, Type I IFN response is silenced due to cleavage of pattern recognition receptors (PRRs). Our study showed that the combination treatment with MCL-1 and caspase inhibitors resulted in a substantial increase in Type I IFN response and immunogenic potential of multiple breast cancer cell lines. Moreover, all clinical studies have shown that caspase inhibitors and MCL1 inhibitors are well-tolerated and safe. Consequently, the results of this study will lay the foundation for a clinical trial assessing the combination treatment over a shorter period of time.

### **P-12: Investigation of inherited and secreted CAGE antigen as a target for evading chemotherapy resistance in cancer patients**

**Min Jeong Yeon, The Wistar Institute**

Cancer is one of the major concerns of health over the world for a long time. Even though promising cancer therapies are being developed, both traditional therapy, radiotherapy and chemotherapy, are being still used. Particularly, in the case of chemotherapy, it is the last treatment option for late-phase patients with some kinds of cancers. However, chemotherapy resistance is a big hustle for treatment with its non-specificity. For several decades, cancer biologists have revealed some of the crucial proteins that mediate chemotherapy resistance, but it still needs to be investigated. CAGE, also known as DDX53, is known as an RNA-helicase protein. Surprisingly, it is one of the proteins expressed widely among tumor tissues while limited to only testis among normal tissues. I figured out that the expression level of CAGE is increased in various types of chemo-resistant cancer cells. Also, CAGE accelerates malignant properties of cancer cells with chemo-resistance by increasing autophagic flux. Malignant cancer cells tend to secrete extracellular vesicles (EVs) and develop a tumorsupportive microenvironment. Exosomes from CAGE overexpressed aggressive mouse cancer cells mediate M2 polarization of tumor-associated macrophages (TAMs) and degranulation of tumor associated mast cells in the tumor microenvironment. These innate immune cells promote tumor in vivo. I revealed CAGE is one of the ingredients of an exosome secreted from chemo-resistant cancer cells. In addition, exosomal CAGE mediates chemotherapy resistance and aggressive properties to chemo-sensitive cancer cells by increasing autophagic flux. To prevent chemo-resistance, I investigated CAGE-targeted strategies such as CAGE 3'UTR binding specific microRNAs and peptides. They inhibit malignant properties and confer sensitivity to chemotherapy in CRCs by decreasing autophagic flux both in vivo and in vitro. Even though there are some challenges including delivery materials, degradation, and toxicity problems, it would be a promising approach for solving chemotherapy resistance. In summary, I figured out CAGE is a mediator of chemotherapy resistance by increasing autophagic flux, and CAGE contained exosomes support a chemo-resistant tumor microenvironment. My study suggested that development of practical CAGE-targeted therapy is a promising task for supporting late-phase cancer patients.

### **P-13: Emerging of small molecules inducing targeted protein degradation as a novel therapeutic modality**

**Kwang-Su Park, Icahn School of Medicine at Mount Sinai**

Since Proteolysis Targeting Chimeras (PROTACs) has emerged, various approaches including Lysosome Targeting Chimeras (LYTACs), autophagy-targeting chimera (AUTAC) and hydrophobic tag have been tried to achieve the degradation of targeted proteins. Those protein degradation technologies have been increased an interest, owing to potential advantages over conventional inhibitors with respect to off-target effect, drug-resistance and targeting undruggable targets. Although there are some of barriers to

overcome as therapeutic agent, it is very promising approach for crossing the hurdles of present drug discovery. Here, I would like to share lessons learned from TPD projects based on our current publications.

#### **P-14: Molecular networking-guided strategy for the pharmacokinetic study of herbal medicines: *Cudrania tricuspidata* leaf extracts**

**Jeong In Seo, Harvard University/Hanyang University**

In this study, the pharmacokinetic profiles of the bioactive components in the leaf extract of the medicinal herb, *Cudrania tricuspidata*, were investigated using an MS/MS-based molecular networking system. To identify the major active components of the *C. tricuspidata* leaf extract (CLE), HPLC-DAD analysis was conducted with a standard mixture of six flavonoids (rutin, isoquercitrin, nicotiflorin, kaempferol 3-O-glucoside, quercetin, and kaempferol). The unknown peaks were determined via molecular networking analysis using the mass dataset obtained by liquid chromatography quadrupole time-of-flight mass spectrometry (LC-QTOF/MS). For the subsequent pharmacokinetic study, CLE (1 g/kg) was orally administered to rats, and plasma samples were collected. The product ion mass data of plasma samples using LC-QTOF/MS were obtained and subjected to molecular networking analysis. The resulting molecular networking map indicated that the glucuronide metabolites of quercetin and kaempferol were the major circulating species. Accordingly, quercetin and kaempferol were determined following  $\beta$ -glucuronidase treatment, and their pharmacokinetic parameters were calculated. These findings indicate that the proposed molecular networkbased approaches are potential and efficient methods for the pharmacokinetic study of herbal medicines.

#### **P-15: Ultrasonic Reporter for Kinase Activity (UReKA)**

**Jee Won Yang, California Institute of Technology**

Although protein kinases control nearly every facet of cellular function, current approaches to understand their tightly controlled spatiotemporal regulation of multiple cellular processes are limited to *in vitro* assays due to the lack of robust, deep-tissue *in vivo* biomolecular tools. As aberrant kinase signaling is linked to numerous pathologies and is a major target for drug discovery, novel tools for imaging live kinase activity are increasingly needed to study the rapid dynamics of and complex mechanisms behind aberrant signal transduction pathways. While advances in fluorescent proteins and live-cell optical imaging have allowed spatiotemporally precise kinase activity measurements in living cells, they do not enable noninvasive deep-tissue imaging due to the physical limitation of light scattering. Alternatively, ultrasound has unique advantages for cellular response imaging due to its ability to penetrate much deeper into tissue (several cm) than light with relatively high spatial and temporal resolution (<100  $\mu\text{m}$  and 1 ms). Additionally, genetically encoded contrast agents for ultrasound based on gas vesicles (GVs), a unique class of air-filled protein nanostructures derived from buoyant microbes, have been developed as acoustic reporter genes and as acoustic biosensors for enzymatic activity. Here, we have engineered GVs as the first ultrasonic reporter for kinase activity (UReKA), enabling dynamic monitoring of Protein Kinase A (PKA) activity reversibly and repeatably when UReKA are administered. Our hypothesis is that upon phosphorylation by PKA, the outer structural proteins of UReKA undergo an allosteric conformational change with a loss of binding to GV shells, resulting in reduced GV shell stiffness, increased mechanical deformation behavior (e.g., buckling), and enhanced detection of non-linear signal (d). Future experiments include demonstrating functionality of UReKA *in vitro* in mammalian cells and *in vivo* to measure

upregulated PKA activity, such as that in tumor resident macrophages, to study pharmacological response to various kinase inhibitors.

### **P-16: Potent and Selective Mitogen-Activated Protein Kinase Kinase 1/2 (MEK1/2) Heterobifunctional Small-molecule Degraders**

**HyeRin (Leah) Yim, Icahn School of Medicine at Mount Sinai**

Previously, we reported a first-in-class von Hippel-Lindau (VHL)-recruiting mitogen-activated protein kinase kinases 1 and 2 (MEK1/2) degrader, MS432. To date, only two MEK1/2 degrader papers have been published and very limited structure-activity relationships (SAR) have been reported. Here, we describe our extensive SAR studies exploring both von Hippel-Lindau (VHL) and cereblon (CRBN) E3 ligase ligands and a variety of linkers, which resulted in two novel, improved VHL-recruiting MEK1/2 degraders, 24 (MS928) and 27 (MS934), and the first CRBN-recruiting MEK1/2 degrader 50 (MS910). These compounds potently and selectively degraded MEK1/2 by hijacking the ubiquitin-proteasome system, inhibited downstream signaling, and suppressed cancer cell proliferation. Furthermore, concurrent inhibition of BRAF or PI3K significantly potentiated the antitumor activity of degrader 27, suggesting that the combination of MEK1/2 degradation with BRAF or PI3K inhibition may provide potential therapeutic benefits. Finally, besides being more potent, degrader 27 displayed improved plasma exposure levels in mice, representing the best MEK1/2 degrader to date for in vivo studies.

### **P-17: Integrated Stress Response regulates mitochondrial biogenesis**

**Sookyung Kim, University of Massachusetts Medical School**

Mitochondrial function declines with aging, and the decline is further aggravated in age-associated diseases. However, it remains unclear if improving mitochondrial function can reduce the physiologic decline associated with aging. To test this relationship, we explored the impact of a translation control pathway known as the Integrated Stress Response (ISR) on mitochondrial function in aging worms. Importantly, the ISR inhibitor (ISRIB) has been shown to reduce age-associated decline in physiologic functions including memory formation in mice (Krukowski et al., eLife. 2020). However, the affected cellular processes responsible for the physiologic improvement observed upon ISR inhibition remain unknown. Using *C. elegans* mutants that mimic ISRIB, I found that mitochondrial function starts to deteriorate as worms reach reproductive maturity, and this mitochondria dysfunction leads to age-associated decline in physiologic function. Worm strains with loss of function mutations in the ISR activation pathway harbor significantly increased mitochondrial mass much later into their adulthood. In addition to living considerably longer than wildtype worms (Denzel et al. Nat. Comm. 2021), these animals also exhibit increased fitness in their muscular and neuronal functions. Intriguingly, I have found these improved physiologic functions observed in ISRIB-like mutant worms require ATFS-1, a transcription factor that has been recently shown to coordinate mitochondrial expansion with growth-factor stimulated protein synthesis (Shpilka et al., Nat. Comm. 2021). Furthermore, I demonstrated that elevated protein synthesis is required for the ATFS-1-dependent mitochondrial expansion observed in ISRIB-like mutant worms. Together, these findings suggest that increased mitochondrial activity may be sufficient to extend muscular and neuronal function further into adulthood.



**P-18: NanoMEA-based In-Vitro assay for predictive assessment of Drug-induced Cardiac Toxicity using human iPSC-derived cardiomyocytes****Byunggik Jason Kim, Johns Hopkins University**

Drug toxicity is a major cause of post-market drug withdrawals. More than 464 medicinal products were withdrawn between 1950 and 2017 due to cardiotoxicity and neurotoxicity, responsible for approximately 16% and 10%, respectively. Also, the drug discovery pipeline of new cardiovascular drugs is limited due to the complexity and cost of evidence generation, with the high rate of failure during the development and clinical trial process. Previously, preclinical drug screening considerably relied on immortalized cell lines and animal models. However, the recent development of human-induced pluripotent stem cells (hiPSCs) allows a more physiologically relevant human model. In this study, we used a nanotopographically-patterned MEA (NanoMEA) with an action potential analysis tool called local extracellular action potential analysis (LEAP) to longitudinally measure and analyze electrophysiological alterations of cardiomyocytes (CM) in vitro, especially for cardiotoxicity screening. The proposed nanoMEA assay provided credible data from 14 of the 15 test compounds (13 FDA-approved and two withdrawn compounds) under seven different dose groups. Beating period (BP), field potential duration (FPD), spike slope (SS), and spike amplitude (SA) were analyzed and compared by double subtraction methods (pre- and post-drug treatment first, and then the difference with the change of DMSO group). Our data showed that Ranolazine, Metformin, Quinidine, Sotalol, Epirubicin, and Domperidone significantly increased BP. In the case of FPD, we found that Ranolazine, Metformin, Quinidine, Sotalol, Epirubicin, and Domperidone prolong the FPD. SS and SA results showed corresponding changes with the BP results. For example, Ranolazine and Sotalol showed significantly prolonged BP but reported a significant decrease in both SS and SA. Irregular beating activity was analyzed to identify potential arrhythmogenic effects of each compound, and we found that 11 of 14 compounds showed early afterdepolarization occurrence from at least one replicate. Finally, our LEAP analysis revealed that action potential waveform kinetics largely follow the trend of BP/FPD changes. The compounds showed prolongation of BP, and FPD also showed increased repolarization in LEAP analysis, and those that showed no difference in FP measurement reported no significant changes between low and high-dose drug treatment either. Our data suggest that our novel hiPSC-CM cardiotoxicity screening platform, NanoMEA plus LEAP assay, may report the effects of test drugs on hiPSC-CMs reliably.

**P-19: Crispr Base Editor Reporter Screen With Bayesian Network Accurately Identifies Causal Variants Of Cellular Low-Density Lipoprotein Uptake****Jayoung Ryu, Harvard University**

CRISPR base editors can induce single-nucleotide transitions and have emerged as powerful tools to test the phenotypic impacts of disease-associated variants in the human population. However, base editing often results in incomplete efficiency at the intended target base as well as bystander edits at proximal bases. The variability in genotypic outcomes and editing efficiency hinders the identification of causal variants in high-throughput base editor screens. We, along with other studies, have recently developed base editor reporter techniques to address this problem by incorporating a reporter, i.e., a target site surrogate sequence with the same spacer and PAM sequence as the endogenous target site into the guide RNA (gRNA) construct. As the gRNA introduces base edits in both endogenous target site and reporter sequence, the overall editing pattern in the endogenous target site can be inferred. We show that our reporter design faithfully recapitulates endogenous target site editing. To utilize the reporter editing pattern to improve the resolution of base editor screens, we have built a novel computational method that

explicitly models the data generation process using the observed editing rate and pattern of the reporter sequence. We have shown this model accurately estimates the effect size of variants in a simulated dataset whereas an existing method, MaGECK, suffers when gRNA editing rate is variable. We employed this model to analyze base editor reporter screen data from two screens measuring the effects of over 10,000 gRNAs on uptake of fluorescent LDL-cholesterol. These screens include a saturation tiling screen of LDLR and a survey of LDL-cholesterol GWAS-associated variants. Our computational method shows better performance in identifying the effective of noncoding variants compared to previous approaches. This work provides a novel and widely applicable approach to improving the power of base editor screens.

### **P-20: $\beta$ -arrestin 2 has anti-tumorigenic and anti-angiogenesis activities via HIF-1 $\alpha$ downregulation in glioblastoma**

**Woom-Yee Bae, The Wistar institute**

Glioblastoma (GBM) is the most common malignant brain tumor and characterized by increase in angiogenesis. Hypoxia inducible factor-1 $\alpha$  (HIF-1 $\alpha$ ) is a critical transcription factor promoting cancer angiogenesis in response to a hypoxic environment. Therefore, HIF-1 $\alpha$  is an attractive therapeutic target in cancer. The basic function of  $\beta$ -arrestin 2 (Arrb2) is a negative regulator of G-protein-coupled receptor. In recently, Arrb2 has been reported to play important roles in cancer pathology including cell proliferation, angiogenesis and metastasis. However, there no report regarding the function of Arrb2 in GBM. We examined the function of Arrb2 associated with regulation of HIF-1 $\alpha$  in GBM. Degradation of HIF-1 $\alpha$  is increased by Arrb2 through promoting the interaction with PHD2 and pVHL. overexpression of Arrb2 in GBM cell line suppresses tumorigenesis and angiogenesis. Furthermore, low Arrb2 expression is correlate with high HIF-1 $\alpha$  expression and is associated with poor GBM patient survival. Collectively, these results indicate that Arrb2 is negative regulator of HIF-1 $\alpha$  and suggest Arrb2 as a new potential therapeutic target for GBM.

### **P-21: Synthetic studies towards 4 $\alpha$ ,9 $\alpha$ ,10 $\alpha$ -trihydroxyguaia-11(13)en-12,6 $\alpha$ -olide**

**Hyejin Lee, Duke University**

Fungal infections have been a severe threat to patients whose immune system is weakened. Several types of antifungal drugs have been developed over the past few decades, but there are still unmet clinical needs including the emergence of antifungal drug resistance. Recently, a guaianolide-type natural product was reported to possess potent antifungal activity against *C. albicans* and *C. parapsilosis*, suggesting it has great potential as a novel template for antifungal agent development. Herein, we report our efforts towards the total synthesis of 4 $\alpha$ ,9 $\alpha$ ,10 $\alpha$ -trihydroxyguaia-11(13) en 12,6 $\alpha$ -olide from (*R*)-(-)-carvone. Currently, our research is investigating a synthetic route for the stereoselective introduction of a  $\gamma$ -butyrolactone followed by the construction of seven-membered ring system to afford a 5,7,5-tricyclic ring system with seven stereocenters. Upon completion of the total synthesis, mode of action studies will be conducted, and structure-activity relationships (SARs) will be explored to develop a new antifungal drug.

**P-22: Biocompatible Gelatin-gallic acid microcomplexes release GO/Cu nanomaterials to eradicate antibiotics-resistant microbes and their biofilm****Jiwon Kim, University of Pennsylvania**

When a wound is formed on the skin, various pathogenic bacteria proliferate, wound healing may be delayed, and if wound infection worsens, life-threatening complications such as sepsis may occur. Wound-infecting bacteria are typically *Pseudomonas aeruginosa* and *Staphylococcus epidermidis*, both forming biofilms and becoming resistant to antibiotics. To solve this problem, graphene oxide (GO)/copper nanocomposites are used as antibacterial materials. However, since the excessive use of antibacterial substances is fatal to normal tissues, to reduce the cytotoxicity of the nanocomposite, GO/Cu was encapsulated with a gelatin complex. Also, among the catechol-based substances, gallic acid, which has anti-inflammatory and antibacterial properties, was used in this study to impart stability to the gelatin complex. Gelatin and gallic acid were combined by a crosslinking method using EDC/NHS as a crosslinker, and this was confirmed by FT-IR, <sup>1</sup>H NMR, and fluorescence. And the synthesized GO/Cu nanocomposites were placed in a GE-GA conjugate solution to fabricate a microcomplex using a water-in-oil method. As a result, it has been proven that it has an antibacterial and antibiofilm effect against Gram-positive bacteria (*Staphylococcus epidermidis*) and Gram-negative bacteria (*Pseudomonas aeruginosa*), even with a small amount. And it has biocompatibility in human cells (HDF) even at a concentration having antimicrobial activity. Reported work showed the potential of antimicrobial Gelatin micro complex in prohibiting the infectious bacteria even though biocompatible for cells and controlling the release of antimicrobial substances.

**P-23: Identifying Ice-Philic Patches to Inform the Ice Binding Sites of Antifreeze Proteins****Jeongmoon Choi, University of Pennsylvania**

Creatures living in cold environment have developed unique strategies to survive. One of their tactics is to evolve antifreeze proteins (AFPs) which lower the freezing temperature by binding to ice. AFPs bind to ice through specific regions on their surface known as ice binding sites (IBS). Each AFP has its own IBS which binds to specific ice planes. Identifying IBS is crucial for understanding how AFPs bind to ice. Here, we use specialized molecular simulation wherein an external potential is used to stimulate ice formation in the AFP hydration shell. We find that the most ice-philic AFP regions, where ice nucleates most readily in response to the external potential, display a strong correspondence with the experimentally determined IBS (using site-directed mutagenesis). In addition to identifying the IBS, our specialized simulations also shed light on ice polymorph and facet that optimally binds the AFP IBS.

**P-24: Nicotinamide phosphoribosyltransferase (Nampt) potentiates antioxidant defense in diet-induced diabetic cardiomyopathy****Jaemin Byun, Hackensack-Meridian Health, Center**

Consumption of a western diet and obesity promote insulin resistance and the development of diastolic dysfunction and fibrosis in the heart, termed diabetic cardiomyopathy. Diabetic cardiomyopathy is accompanied by decreases in NAD<sup>+</sup>/NADH, leading to the increasing production of NADH, consequently increases in oxidative stress. The role of nicotinamide phosphoribosyltransferase (Nampt), the rate-

limiting enzyme of the salvage pathway of NAD<sup>+</sup>-synthesis, in the development of diabetic cardiomyopathy is poorly understood. NADP is converted to NADPH, an electro donor for glutathione (GSH) and thioredoxin (Trx) and is generated from NAD<sup>+</sup> in the presence of NAD kinase (NADK). We investigated the role of endogenous and exogenous Nampt during the development of diabetic cardiomyopathy in response to high fat diet (HFD) consumption. We asked if Nampt effects the development of diabetic cardiomyopathy, if Nampt plays an important role in the regulation of NADP(H) and consequent activation of the GSH and Trxs systems, and if the protective effect of Nampt is mediated through NADP(H) production in the heart. HFD consumption upregulated endogenous Nampt, and HFD-induced cardiac diastolic dysfunction, fibrosis, apoptosis and pro-inflammatory signaling were alleviated in transgenic mice with cardiac-specific overexpression of Nampt. The alleviation of diastolic dysfunction observed in these mice was abolished by inhibition of NADP(H) production via NAD kinase (NADK) inhibition. Nampt overexpression decreased the GSSG/GSH ratio, oxidation of thioredoxin 1 (Trx1) targets, dityrosine, and the accumulation of toxic lipids, including ceramides and diglycerides, in the presence of HFD consumption. Nampt overexpression upregulated not only NAD<sup>+</sup> but also NADP<sup>+</sup> and NADPH in the heart and in cultured cardiomyocytes (CMs), which in turn stimulated the GSH and Trx1 systems and alleviated oxidative stress in the heart induced by HFD consumption. In cultured CMS, Nampt-induced upregulation of NADPH was abolished in the presence of NADK knockdown, whereas that of NAD<sup>+</sup> was not. Nampt overexpression attenuated H<sub>2</sub>O<sub>2</sub>-induced oxidative inhibition of Prdx1 and mTOR in an NADK-dependent manner in cultured CMs. Nampt overexpression also attenuated H<sub>2</sub>O<sub>2</sub>-induced cell death, an effect that was partly abolished by inhibition of NADK, Trx1 or GSH synthesis. In contrast, oxidative stress and the development of diabetic cardiomyopathy in response to HFD consumption were exacerbated in Nampt<sup>+/-</sup> mice. Nampt-mediated production of NAD<sup>+</sup> protects against oxidative stress in part through the NADPH-dependent reducing system, thereby alleviating the development of diabetic cardiomyopathy in response to HFD consumption. Nampt is necessary and sufficient protect the heart against the development of diabetic cardiomyopathy by HFD consumption and the protective effect of NAMPT in the diabetic heart is mediated in part through NADK-dependent-mechanisms and stimulation of GAH and Trx1.

### **P-25: A Fluorescence-Polarization-Based Lipopolysaccharide–Caspase-4 Interaction Assay for the Development of Inhibitors**

**Jinsu An, Boston Children's Hospital, Harvard University**

Recognition of intracellular lipopolysaccharide (LPS) by Caspase-4 (Casp-4) is critical for host defense against Gram-negative pathogens. LPS binds to the N-terminal caspase activation and recruitment domain (CARD) of procaspase-4, leading to auto-proteolytic activation followed by pro-inflammatory cytokine release and pyroptotic cell death. Aberrant hyper-activation of Casp-4 leads to amplification of the inflammatory response linked to sepsis. While the active site of a caspase has been targeted with peptide inhibitors, inhibition of LPS–Casp-4 interaction is an emerging strategy for the development of selective inhibitors with a new mode of action for treating infectious diseases and sepsis induced by LPS. In this study, a high-throughput screening (HTS) system based on fluorescence polarization (FP) was devised to identify inhibitors of the LPS and Casp-4 interaction. Using HTS and IC<sub>50</sub> determination and subsequently showing inhibited Casp-4 activity, we demonstrated that the LPS–Casp-4 interaction is a druggable target for Casp-4 inhibition and possibly a non-canonical inflammatory pathway.

**P-26: Rapid analysis of gene therapy vectors with Nanopore MinION sequencer****Suk Namkung, UMass Chan Medical School**

Adeno-associated virus (AAV) is a non-enveloped single-stranded DNA virus. AAV is primarily studied as a vector for gene therapy because of its non-pathogenic nature and ability to deliver engineered transgene cassettes as recombinant (r)AAV. There are currently three commercially available rAAV-based therapies, and over 200 clinical trials. However, quality control strategies for rAAV vectors fall short in detecting non-unit length genomes and different contaminants that can impact the efficacy and safety of these therapies. Furthermore, Sanger sequencing platforms cannot accurately quantify the proportion of contaminant genomes in vectors, nor can they identify their exact compositions. To develop a means to analyze rAAV genomes, we evaluated the capacity of long-read sequencing by Oxford Nanopore Technology's MinION platform to sequence both clinically relevant forms of rAAV genomes: the single-stranded (ss) and the self-complementary (sc)AAV vector genomes. We used both MinION R9 and R10 flow cells to: 1) evaluate differences in sequencing ssAAV and scAAV genomes, 2) validate whether reads accurately reflect the vector genome population, and 3) assess the accuracy of nanopore sequencing across the inverted terminal repeats (ITRs), which are characterized by T-shaped hairpins that interfere with the processivity of polymerases. Our results show that both R9 and R10 can identify and quantify DNA contaminants and heterogeneous vector populations; however, R9 showed better coverage of the ITRs. Sequencing of the parental plasmids validated that abnormal vector genomes are not an artifact of the sequencing platform. Additionally, through MinION's ability to sequence through the ITRs, we were able to reveal different conformations of rAAV genomes during production, which until now have remained uncharacterized. In conclusion, we find that the ONT platform excels in characterizing each individually packaged vector genomes and can serve as a rapid and lower cost alternative for analyzing vectors.

**P-27: Bulk RNA-seq analysis with unique machine learning algorithm provides accurate deconvolution of tumor microenvironment****Taekyoung Kwak, BostonGene Co.**

Cancer remains one of the major leading causes of death that need to be overcome for better quality of human life. The tumor microenvironment is the newly forming heterogeneous environment by tumor derived cytokines, chemokines, and is closely interacted with tumor and other components including immune cells, fibroblasts, blood vessels, and the extracellular matrix. Although recent understanding on cancer immunology has shed light on mechanisms for cancer initiation, progression and metastasis, the dynamics of tumor and microenvironment is still unclear in each individual cancer case and furthermore it has not been fully addressed which mechanism can improve the overall response rate for immunotherapy. Precision medicine, known as "Personalized medicine", is a revolutionized approach for disease treatment and prevention that consider individual variability by analyzing integrated genomics, epigenomic, transcriptomics, proteomics and metabolomics. Unprecedented technology development in next generation sequencing (NGS), flow cytometry and artificial intelligent (AI) driven machine learning (ML) technique is able to provide better resolution on tumor and microenvironments. Cellular deconvolution algorithms have been developed to determine the proportions of different cell types in collected samples by gene expression profile and applied to a variety of samples e.g., PBMC, saliva, hair follicle and cancer sample. Since deconvolution algorithm concept was introduced in 2018, 16 different types of algorithms are actively being used in the field of precision medicine. Although deep learning-

based deconvolution methods have also been developed recently, these approaches often require data from single-cell RNA seq (scRNA-Seq) of the same tissue type and/or paired flow cytometry data, indicating there is still limitation in terms of clinical utility. In addition, scRNA-Seq has a blind spot for addressing importance of small fraction of tumor microenvironment subset, such as natural killer (NK) cells that is barometer for response of immunotherapies. It is required to minimize technical noise during cellular deconvolution for providing accurate cellular composition from bulk RNA-seq. Here we present the decision tree machine learning algorithm, *Kassandra*, trained on a broad collection of >9,400 tissue and blood sorted cell RNA profiles incorporated into millions of artificial transcriptomes to accurately reconstruct the tumor microenvironment. Performance was validated on 4,000 H&E slides and 1,000 tissues by comparison with cytometric, immunohistochemical, or single-cell RNA-seq measurements. *Kassandra* algorithm accurately deconvolved TME elements, showing the role of these populations in tumor pathogenesis and other biological processes. Digital TME reconstruction revealed that the presence of PD-1-positive CD8<sup>+</sup> T cells strongly correlated with immunotherapy response and increased the predictive potential of established biomarkers, indicating that *Kassandra* could potentially be utilized in future clinical applications of precision medicine.

### **P-28: Effectiveness of famotidine on the risk of poor prognosis in COVID-19 patients: A nationwide cohort study in Korea**

**Roise Kwon, University of Michigan**

**Introduction:** Famotidine has been proposed as a promising candidate for the treatment of coronavirus disease 2019 (COVID-19). However, there is limited research on the association of famotidine with the poor prognosis of COVID-19. **Methods:** The Korean nationwide cohort included 6,556 patients who tested positive on RT-PCR for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The poor COVID-19-related outcomes were defined on the basis of having encountered with the composite outcome of high oxygen therapy, intensive care unit admission, administration of mechanical ventilation, or death. In addition, we performed exposure-driven propensity score matching for no H<sub>2</sub>-blocker use versus current famotidine use, and other H<sub>2</sub>-blocker use versus current famotidine use. **Results:** 4,785 (73.0%) patients did not use a H<sub>2</sub>-blocker, 393 (6.0%) patients were currently used famotidine, and 1,292 (19.7%) patients currently used H<sub>2</sub>-blocker other than famotidine. In multivariable analysis after matching (no H<sub>2</sub>-blocker use versus current famotidine use), there was no significant association between current famotidine use and composite outcomes (adjusted odd ratios [aOR]: 1.30, 95% confidence interval [CI]: 0.55-3.06). On the other hand, another matched cohort (other H<sub>2</sub>-blocker use versus current famotidine use), demonstrated a positive association between current famotidine use and composite outcomes (aOR: 3.56, 95% CI: 1.03-12.28). **Conclusions:** Our study results did not support the potential of famotidine as a therapeutic agent for COVID-19. A rather unexpected result could be observed in the comparisons between current famotidine use and other H<sub>2</sub>-blocker use; it was observed that current famotidine use increased the risk of poor COVID-19 related outcomes. Further studies are needed to clearly prove the causal relationship with several H<sub>2</sub>-blocker, including famotidine.



## 2022 KASBP Fall Job Fair

### Nov 4-5, 2022 (Prearranged Meeting Time)

For the past few years, the KASBP Job Fair has established itself as a direct channel for many Korean professionals 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 as an opportunity to meet and recruit talents from overseas and have been continuously participating throughout the years. This year, participating companies include Daewoong Pharmaceutical, GC Biopharma, GC Cell, SK Bioscience, JW Pharmaceutical, Dong-A ST, Amyloid Solution, Myriad Life Sciences, KIST, Samyang Holdings, and Samsung Biologics. Candidates may apply to multiple companies.

The 2022 KASBP Fall Job Fair will provide an in-person or virtual job interview for applicants and hiring managers from Korean pharmaceutical companies. 2022 KASBP Fall Symposium Registration is required, but no additional fees are required to participate in Job Fair as an applicant.

The 2022 KASBP Fall Job Fair application was closed on Oct 16, 2022, however, late-applications may be considered. Please contact the KASBP Job Fair coordinator ([jobfair@kasbp.org](mailto:jobfair@kasbp.org)) to check the availability. The job description of the companies is available on the KASBP website (under 2022 Fall Job Fair).

In-person interview will take place during Friday, Nov 4 - Saturday, Nov 5, 2022. The interview meeting time will be prearranged and details will be sent to each candidate and interviewer via email by KASBP Job Fair coordinator. Virtual interviews are arranged and notified directly by the participating companies.

More information about 2022 KASBP Fall Symposium Job Fair can be found at:

- 2022 KASBP Fall Job Fair Info.: <https://kasbp.org/page-1863526/12923524>
- 2022 KASBP Fall Job Fair Application Link: <https://forms.gle/5riMGG7ohacQPbjZA>
- 2022 KASBP Fall Symposium Registration: <https://kasbp.org/event-4975133>
- Contact: [jobfair@kasbp.org](mailto:jobfair@kasbp.org)

## Past 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)
- 2020 Larry Kwak (City of Hope)
- 2021 Daniel Chung (SparingVision)

### 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)
- 2020 Jun Young Hong (Yale University), Heeseon An (Harvard Medical School), Yoon Seok Kim (Stanford University)
- 2021 Youngeun Kim (Harvard Medical School), Juhyun (Julie) Oh (Harvard Medical School), Rafael Taeho Han (University of California, San Francisco)

**KASBP-GC Biopharma 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), Bloria 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)
- 2020 Namgyu Lee (University of Massachusetts)

**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)
- 2021 Taekyung Kwak (The Wistar Institute), WooriKim (Harvard Medical School), Tae-Yoon Park (McLean Hospital)
- 2022 Hyuna Jo (UC Irvine), Changrim Lee (Harvard Medical School), Danielle (Hyun Jun) Kim (UC Davis)

**KASBP-LG CHEM Fellowship**

- 2017 Kyoung-Dong Kim (Wistar Institute), Seok-Man Ho (Icahn School of Medicine at Mount Sinai)
- 2019 Jea Hyun Baek (Biogen Inc.), Donggi Paik (Harvard Medical School)

**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 Kiyong 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)
- 2021 Sekyu Choi (Harvard University), Sungyun Cho (Weill Cornell Medical College), Dongheon Lee (Duke University)
- 2022 Yongmin Cho (Harvard Medical School), Ho Namkung (Johns Hopkins University), Hyeonglim Seo (UCSD)

#### **KASBP-Dong-A ST PHARM Fellowship**

- 2016 Jung-Eun Jang (New York University), Byungsu Kwon (MIT)
- 2021 Min Jae Lee (Yale University)

#### **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), (Fall) Minyoung Park (The Rockefeller University)
- 2019 Kyusik Kim (University of Massachusetts Medical School)

#### **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. Line Institute for Psychiatry Research), Jongho Park (Massachusetts General Hospital)

#### **KASBP- KRIBB Fellowship**

- 2019 Song Min (Harvard Medical School), Eun-Ik Koh (University of Massachusetts Medical School)

#### **KASBP-KHIDI Fellowship**

- 2010 JaeHyun Bae (Yale University), HeeYeon Cho (Boston College)
- 2020 Haejin Yoon (Harvard Medical School)
- 2022 Se-Yeong Oh (Emory University), Soojin Lee (UMass Medical School)

#### **KASBP-CHOONGCHEONGBUK-DO Fellowship**

- 2020 Su Bin Lim (Johns Hopkins University), Brandon Suh (Harvard University)

**KASBP-ISUABXIS Fellowship**

2020 Jongwoo Son (University of Wisconsin-Madison), Won Dong Lee (Princeton University)

2021 Yong-Woo Jun (University of Massachusetts Medical School), Yu Young Jeong (Rutgers University)

**KASBP-SEEGENE Fellowship**

2020 Haejin Kim (Columbia University)

**KASBP-MDIMUNE Fellowship**

2020 Young Jae Woo (Icahn School of Medicine at Mount Sinai)

**KASBP-NANOENTEK Fellowship**

2020 Jongkyun Kang (Brigham and Women's Hospital)

**KASBP-ENZYCHEM Fellowship**

2021 Jinwoo Kim (Stony Brook University)

**KASBP-SAMYANG Fellowship**

2021 Kyongman An (Johns Hopkins University)

**KASBP-SK BIOPHARM Fellowship**

2021 Sung-Hee Yoon (Harvard Medical School)

**KASBP-ABTIS Fellowship**

2021 Yunju Yang (University of Texas Health Science Center at Houston)

**KASBP-EUTILEX Fellowship**

2021 Yun Hwa Choi (University of Wisconsin-Madison)

**KASBP-KPBMA Fellowship**

2021 Dahye Kang (Harvard Medical School), Bumjun Kim (Princeton University)

**KASBP-ITP/Yonsei Fellowship**

2021 Jaeho Shin (University of Notre Dame), Jeonghwan Kim (OSU & OHSU)

**KASBP-KAIST/GCC Fellowship**

2021 Seungbeom Ko (Medical University of South Carolina), Annie J. Lee (Columbia University Medical Center)

**KASBP-DAEWOONG Scholarship**

2006 Jin K. Pai (Handok Pharmaceuticals, Korea)

2007 YoungWhan Park (National Cancer Center, Korea)

2008 Young-Choon Moon (PTC Therapeutics)

2009 HongYong Kim (Novartis)

## 2022 KASBP Fall Symposium Attendees (as of 10/24/2022)

	First name	Last name	이름	근무처	Networking Group
1	Jae Eun	Ahn	안재은	Pfizer, Inc.	Clinical Trial & Development / Clinical Pharmacology/Biostatistics
2	Hyun Ji	An	안현지	Harvard Medical School	Medical Device/In Vitro Diagnostics/Biomedical Engineering/Analytical Method Development
3	Jinsu	An	안진수	Boston Children's Hospital/Harvard Medical School	Immunology-Oncology/Autoimmune/Inflammatory Disease
4	Kazuko	Aoyagi	Kazuko	Celerion	Metabolic Diseases/Cardiovascular/Diabetes/Respiratory Diseases
5	Jungho	Back	백정호	AccessBio Inc	Medical Device/In Vitro Diagnostics/Biomedical Engineering/Analytical Method Development
6	Alexis	Bae	배지은	Massachusetts Biomed Lab	Business Development/Venture Capital/Legal/Consulting/Government Relations
7	Alice	Bae	배리스	University of Chicago Medicine	Cell and Gene Therapy/Rare Disease
8	Woomyee	Bae	배움이	The Wistar Institute	Immunology-Oncology/Autoimmune/Inflammatory Disease
9	Juhyun	baek	백주현	Bio Marketing Lab	Business Development/Venture Capital/Legal/Consulting/Government Relations
10	Jaemin	Byun	변재민	Hackensack Meridian Hospital	Metabolic Diseases/Cardiovascular/Diabetes/Respiratory Diseases
11	Jun Hyung	Cha	차준형	Harvard t.h. chan school of public health	Bioinformatics/A.I./Machine Learning/Quantitative Science
12	Joonyeon	Chang	장준연	Korea Institute of Science and Technology	Business Development/Venture Capital/Legal/Consulting/Government Relations
13	C-HONG	CHANG	장시홍	Alexion AstraZeneca Rare Disease	Cell and Gene Therapy/Rare Disease
14	Yeonji	Chang	장연지	Whitehead Institution	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
15	Hyun-Kyung	Chang	장현경	Columbia University	
16	Jintae	Chang	장진태	Amyloid Solution	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
17	Jin Hyuk	Chang	장진혁	NEXEL USA	Business Development/Venture Capital/Legal/Consulting/Government Relations
18	Kern	Chang	장건희	Janssen R&D	Business Development/Venture Capital/Legal/Consulting/Government Relations



	First name	Last name	이름	근무처	Networking Group
19	Min-Kyu	Cho	조민규	Novartis	CMC/Quality Assurance/Regulatory Affairs/Project Management
20	Ji-Hoon	Cho	조지훈	Johnson & Jonhson	
21	Yehlin	Cho	조예린	MIT	Medical Chemistry/Drug Discovery&Delivery/Preclinical
22	Junhyo	Cho	조준효	University of Massachusetts Amherst	Metabolic Diseases/Cardiovascular/Diabetes/Respiratory Diseases
23	Mi Hyeon	Cho	조미현	Massachusetts General Hospital/ Harvard Medical School	Medical Chemistry/Drug Discovery&Delivery/Preclinical
24	Yong	Cho	조용성	YC Consulting	Business Development/Venture Capital/Legal/Consulting/Government Relations
25	Yun H.	Choe	최윤	ArentFox Schiff LLP	Business Development/Venture Capital/Legal/Consulting/Government Relations
26	Hyunsop	Choe	최현섭	The Catholic University of Korea	Immunology-Oncology/Autoimmune/Inflammatory Disease
27	Gayeon	Choi	최가연	MCPHS	Clinical Trial & Development / Clinical Pharmacology/Biostatistics
28	Young Joon	Choi	최영준	SK bioscience	Cell and Gene Therapy/Rare Disease
29	Doo Eun	Choi	최두은	Massachusetts General Hospital	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
30	Dongsub	Choi	최동섭	GC 녹십자	Clinical Trial & Development / Clinical Pharmacology/Biostatistics
31	Sung Hugh	Choi	최성휴	Pinetree Therapeutics	Bioinformatics/A.I./Machine Learning/Quantitative Science
32	Hee June	Choi	최희준	(Former) Boston Children's Hospital/ Harvard Medical School	
33	Jonggil	Choi	최종길	SK Inc	Business Development/Venture Capital/Legal/Consulting/Government Relations
34	Hannah	Choi	최혜수	Massachusetts General Hospital	Business Development/Venture Capital/Legal/Consulting/Government Relations
35	Hak Soo	Choi	최학수	Mass General Hospital	Medical Chemistry/Drug Discovery&Delivery/Preclinical
36	Sofia	Choi	최예원	Merck	Clinical Trial & Development / Clinical Pharmacology/Biostatistics
37	John	Choi	최 존	New Target Health	
38	Hyoann	Choi	최효안	Georgia Tech	Bioinformatics/A.I./Machine Learning/Quantitative Science

	First name	Last name	이름	근무처	Networking Group
39	Eunseo	Choi	최은서	Oregon State University	Immunology- Oncology/Autoimmune/Inflammatory Disease
40	Jeongmoon	Choi	최정문	University of Pennsylvania	Medical Chemistry/Drug Discovery&Delivery/Preclinical
41	Soon Gang	Choi	최순강	Ginkgo Bioworks	Business Development/Venture Capital/Legal/Consulting/Government Relations
42	Hyeong- wook	Choi	최형욱	Eisai Inc.	Medical Chemistry/Drug Discovery&Delivery/Preclinical
43	Jeonghoon	Choi	최정훈	Wave Life Sciences	Metabolic Diseases/Cardiovascular/Diabetes/Resp iratory Diseases
44	Hanmaru	Chon	전한마루	Access Bio	Medical Device/In Vitro Diagnostics/Biomedical Engineering/Analytical Method Development
45	Min-Kyung	Choo	추민경	Ingenia Therapeutics	Immunology- Oncology/Autoimmune/Inflammatory Disease
46	Alex	Chung	정관호	Perkins Coie LLP	Business Development/Venture Capital/Legal/Consulting/Government Relations
47	Younghoon	Chung	정영훈	MCPHS Worcester Campus	Clinical Trial & Development / Clinical Pharmacology/Biostatistics
48	Sally	Chung	정혜린	Johns Hopkins University	
49	Daniel	Chung	정다니엘	Sparingvision	
50	Sang Mok	Chung	정 상목	LG Chem Life Sciences Innovation Center	CMC/Quality Assurance/Regulatory Affairs/Project Management
51	HaeWon	Chung	정해원	Asimov	Clinical Trial & Development / Clinical Pharmacology/Biostatistics
52	Seungwon	Chung	정승원	AbbVie	Medical Chemistry/Drug Discovery&Delivery/Preclinical
53	Taesun	Eom	엄태선	Beam Therapeutics	Infectious Diseases/Vaccines/RNA Therapeutics
54	Seungjin	Ha	하승진	Nano-Ditech	Medical Device/In Vitro Diagnostics/Biomedical Engineering/Analytical Method Development
55	Sangyeul	Han	한상열	Ingenia Therapeutics	Metabolic Diseases/Cardiovascular/Diabetes/Resp iratory Diseases

	First name	Last name	이름	근무처	Networking Group
56	James	Han	한성준	Harvard University	Immunology- Oncology/Autoimmune/Inflammatory Disease
57	Daehee	Han	한대희	Standigm	Bioinformatics/A.I./Machine Learning/Quantitative Science
58	JUMI	HAN	한주미	Daewoong, HQ	
59	Karsten	Holm	Karsten	Medicilon	CMC/Quality Assurance/Regulatory Affairs/Project Management
60	Ted	Hong	Ted Hong	AstraZeneca	
61	Eun Chul	Huh	허은철	GC Pharma	
62	Sunyoung	Hwang	황선영	UMass Chan medical school	Medical Chemistry/Drug Discovery&Delivery/Preclinical
63	Hee	Hwang	황희	MCPHS University	
64	MinEun	Hwang	황민은	MCPHS University	
65	Sunil	Hwang	황선일	Sonata Therapeutics	Bioinformatics/A.I./Machine Learning/Quantitative Science
66	Sungyong	Hwang	황성용	US FDA	Immunology- Oncology/Autoimmune/Inflammatory Disease
67	SEUNGJO	HYUN	현승조	GCCell	Cell and Gene Therapy/Rare Disease
68	HYO YOUNG	IM	임효영	Curachem	
69	Eunju	Im	임은주	Nathan S. Kline Institute for Psychiatric Research / NYU Grossman School of Medicine	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
70	NanoEnTek	Inc	나노엔텍		
71	Sang Eun	Jee	지상은	Xtalpi Inc.	Bioinformatics/A.I./Machine Learning/Quantitative Science
72	Youngha	Jeon	전영하	University of rhode island	
73	HEYKYEONG	JEONG	정혜경	GSK	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
74	Yunju	Jeong	정윤주	Brigham and Women's Hospital & Harvard Medical School	Metabolic Diseases/Cardiovascular/Diabetes/Resp iratory Diseases
75	Tae Heum	JEONG	정태흠	SV Bio Ventures	Business Development/Venture Capital/Legal/Consulting/Government Relations
76	Hyejoong	Jeong	정혜중	University of Pennsylvania	Medical Device/In Vitro Diagnostics/Biomedical Engineering/Analytical Method Development

	First name	Last name	이름	근무처	Networking Group
77	Jae Uk	Jeong	정재욱	MOGAM Institute for Biomedical Research	
78	Mi Ho	Jeong	정미호	Massachusetts General Hospital	Medical Device/In Vitro Diagnostics/Biomedical Engineering/Analytical Method Development
79	Eunmi	Jeong	정은미	Clark University	Business Development/Venture Capital/Legal/Consulting/Government Relations
80	Sinyoung	Jeong	정신영	Intek Scientific Inc	Bioinformatics/A.I./Machine Learning/Quantitative Science
81	Euijoon	Jeong	정의준	NeuBase Therapeutics	Business Development/Venture Capital/Legal/Consulting/Government Relations
82	Sun-Gou	Ji	지선구	BridgeBio Pharma Inc.	Bioinformatics/A.I./Machine Learning/Quantitative Science
83	Joon Yung	Jin	진준영	Hanwha Impact	
84	Jaehyeok	Jin	진재혁	Columbia University	Bioinformatics/A.I./Machine Learning/Quantitative Science
85	Yonghwan	Jin	진용환	Samsung BioLogics	Infectious Diseases/Vaccines/RNA Therapeutics
86	Seunghee	Jo	조승희	Blueprint Medicines	Medical Chemistry/Drug Discovery&Delivery/Preclinical
87	Hakryul	Jo	조학렬	Kymera Therapeutics	Medical Chemistry/Drug Discovery&Delivery/Preclinical
88	Yongwoo	Jun	전용우	UMASS MEDICAL SCHOOL	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
89	Young Chun	Jung	정영춘	Mersana Therapeutics Inc.	
90	Hyunkyung	Jung	정현경	Medic Life Sciences	Medical Chemistry/Drug Discovery&Delivery/Preclinical
91	Hyun Jin	Jung	정현진	Woodbridge pharmacy	Infectious Diseases/Vaccines/RNA Therapeutics
92	Sang Hoon	Jung	정상훈	Korea Institute of Science and Technology	Medical Chemistry/Drug Discovery&Delivery/Preclinical
93	Hosun	Jung	정호선	CRISPR Therapeutics	
94	Da-Jung	Jung	정다정	Brigham and Women's Hospital	Immunology-Oncology/Autoimmune/Inflammatory Disease
95	Inyoung	Jung	정인영	University of Pennsylvania	Cell and Gene Therapy/Rare Disease
96	Jinhyuk	Jung	정진혁	Bridge Biotherapeutics	Medical Chemistry/Drug Discovery&Delivery/Preclinical

	First name	Last name	이름	근무처	Networking Group
97	Sungwook	Jung	정성욱	Harvard Medical School	
98	Jaeyong	Jung	정재용	Rutgers University	Immunology- Oncology/Autoimmune/Inflammatory Disease
99	Eunsoo	Jung	정은수	Bridge Biotherapeutics	Metabolic Diseases/Cardiovascular/Diabetes/Respiratory Diseases
100	Dahee	Jung	정다희	University of Illinois at Chicago	Immunology- Oncology/Autoimmune/Inflammatory Disease
101	Hyunki	Kang	강현기	KHIDI USA	Business Development/Venture Capital/Legal/Consulting/Government Relations
102	Byunghak	Kang	강병학	Novartis	Bioinformatics/A.I./Machine Learning/Quantitative Science
103	Mikyung	Kang	강미경	Mass General Hospital	
104	hyelim	kang	강혜림	dana farber cancer institute	Medical Chemistry/Drug Discovery&Delivery/Preclinical
105	Jongkyun	Kang	강종균	Harvard Medical School/BWH	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
106	Pilsoo	Kang	강필수	Sanofi	Cell and Gene Therapy/Rare Disease
107	Jin-Chul	Kim	김진철	Korea Institute of Science and Technology	Infectious Diseases/Vaccines/RNA Therapeutics
108	Sungjin	Kim	김성진		
109	Hyeonsuk	Kim	김현숙	Access Bio Inc.	Medical Device/In Vitro Diagnostics/Biomedical Engineering/Analytical Method Development
110	HoWon	Kim	김호원	K2B Therapeutics	Immunology- Oncology/Autoimmune/Inflammatory Disease
111	Seongwon	Kim	김성원	Harvard University	Cell and Gene Therapy/Rare Disease
112	Cheoljin	Kim	김철진	MST	Medical Device/In Vitro Diagnostics/Biomedical Engineering/Analytical Method Development
113	Hyungchul	Kim	김형철	Novartis	Medical Chemistry/Drug Discovery&Delivery/Preclinical
114	Hyoungsoo	KIM	김형수	Access bio	Medical Device/In Vitro Diagnostics/Biomedical Engineering/Analytical Method Development

	First name	Last name	이름	근무처	Networking Group
115	Junyong	Kim	김준용	ModernaTX	Medical Chemistry/Drug Discovery&Delivery/Preclinical
116	Jinah	Kim	김진아	KHIDI USA	Business Development/Venture Capital/Legal/Consulting/Government Relations
117	Olivia	Kim	김해정	Accessbio inc	Medical Device/In Vitro Diagnostics/Biomedical Engineering/Analytical Method Development
118	Hak	Kim	김학	SK bioscience	Infectious Diseases/Vaccines/RNA Therapeutics
119	hyeonkyeon g	kim	김현경	GC Biopharma	
120	Minjeong	Kim	김민정	Hongik University	
121	Younghoon	Kim	김영훈	Tome BioSciences	Cell and Gene Therapy/Rare Disease
122	Myo-Kyoung	Kim	김묘경	University of the Pacific	Clinical Trial & Development / Clinical Pharmacology/Biostatistics
123	Yurim	Kim	김유림	MCPHS University	Clinical Trial & Development / Clinical Pharmacology/Biostatistics
124	SungKwon	Kim	김성권	Alexion Pharmaceuticals	Immunology-Oncology/Autoimmune/Inflammatory Disease
125	Sookyung	Kim	김수경	UMass Med	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
126	Hylim	Kim	김혜림	PineTree Therapeutics	CMC/Quality Assurance/Regulatory Affairs/Project Management
127	Jeonghyeon	Kim	김정현	DFCI	Bioinformatics/A.I./Machine Learning/Quantitative Science
128	WonHee	Kim	김원희	EdiGene Biotech	Infectious Diseases/Vaccines/RNA Therapeutics
129	Dae-Shik	Kim	김대식	Eisai Inc	Medical Chemistry/Drug Discovery&Delivery/Preclinical
130	hyejung	kim	김혜정	MCPHS University	CMC/Quality Assurance/Regulatory Affairs/Project Management
131	Kyusik	Kim	김규식	Tessera Therapeutics	Cell and Gene Therapy/Rare Disease
132	Yoon	Kim	김윤아	Weill Cornell Medicine	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
133	Kiel	Kim	김카일	SK Inc.	Cell and Gene Therapy/Rare Disease
134	Mi-Sook	Kim	김미숙	Takeda	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
135	Kyung	Kim	김경효	AbbVie	Bioinformatics/A.I./Machine Learning/Quantitative Science
136	Byunggik	Kim	김병직	Johns Hopkins University	

	First name	Last name	이름	근무처	Networking Group
137	Byungchan	kim	김병찬	Xtalpi Inc	Medical Chemistry/Drug Discovery&Delivery/Preclinical
138	Stephen	Kim	김스티븐	Qiagen	Medical Device/In Vitro Diagnostics/Biomedical Engineering/Analytical Method Development
139	Eun Mi	Kim	김은미	University of Illinois at Urbana-Champaign	Medical Device/In Vitro Diagnostics/Biomedical Engineering/Analytical Method Development
140	Jiwon	Kim	김지원	University of Pennsylvania	
141	Minji	Kim	김민지	Cross Border Partners, LLC	
142	Miji	Kim	김미지	MCPHS University	
143	Hannah	Kim	김한나	Temple University	
144	Heesun	Kim	김희선	Vertex Pharmaceuticals	Cell and Gene Therapy/Rare Disease
145	insil	kim	김인실	Repare Therapeutic	Clinical Trial & Development / Clinical Pharmacology/Biostatistics
146	Seongjun	Kim	김성준	MCPHS University	
147	Sean	Kim	김승빈	Valo	Clinical Trial & Development / Clinical Pharmacology/Biostatistics
148	Sung ki	Kim	김성기	Massachusetts Biomed Lab, Inc.	Business Development/Venture Capital/Legal/Consulting/Government Relations
149	Dongkyoon	Kim	김동균	Initium Therapeutics	
150	JINWOO	KIM	김진우	Akebia Therapeutics	Medical Chemistry/Drug Discovery&Delivery/Preclinical
151	Boram	Kim	김보람	Rice University	Immunology- Oncology/Autoimmune/Inflammatory Disease
152	TaeHyun	Kim	김태현	MIT	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
153	Hye In	Kim	김혜인	Pfizer	Bioinformatics/A.I./Machine Learning/Quantitative Science
154	Lee Joon	Kim	김이준	Lawrence Berkeley National Laboratory	Medical Chemistry/Drug Discovery&Delivery/Preclinical
155	Tae-Hyung	Kim	김태형	University of New Mexico	Immunology- Oncology/Autoimmune/Inflammatory Disease
156	Najung	Kim	김나정	Biotech consulting	
157	Deok-Ho	Kim	김덕호	Johns Hopkins University	Infectious Diseases/Vaccines/RNA Therapeutics



	First name	Last name	이름	근무처	Networking Group
158	Nam Cheol	Kim	김남철	United States Pharmacopeia	Business Development/Venture Capital/Legal/Consulting/Government Relations
159	Taeg S	Kim	김택수	HotSpot Therapeutics	Immunology-Oncology/Autoimmune/Inflammatory Disease
160	Jeong Hoon	Ko	고정훈	MIT	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
161	Kwangseok	Ko	고광석	Jw pharmaceutical	Medical Chemistry/Drug Discovery&Delivery/Preclinical
162	Byunghee	Koh	고병희	Brigham and Women's hospital	
163	JONG SUNG	KOH	고종성	GENOSCO	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
164	Kwi Hye	Koh	고귀혜	Morphic Therapeutic	Medical Chemistry/Drug Discovery&Delivery/Preclinical
165	Young Don	kwak	곽영돈	Novartis Institutes for Biomedical Research	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
166	Taekyoung	Kwak	곽태경	BostonGene	Immunology-Oncology/Autoimmune/Inflammatory Disease
167	Minji	Kwon	권민지	KHIDI USA	
168	Kyenghee	Kwon	권경희	Dongguk University	Business Development/Venture Capital/Legal/Consulting/Government Relations
169	Rosie	Kwon	권로지	University of Michigan	
170	Taeyoon	Kyung	경태윤	Ginkgo Bioworks	
171	YUNSUP	LEE	이윤섭	LG CHEM LIFE SCIENCES	
172	Jongha	Lee	이종하	Axoft	
173	Dayeoun	Lee	이대연	mcphs pharmacy	
174	Jaekyoo	Lee	이재규	GENOSCO	
175	Taegyo	Lee	이태교	Pfizer Inc.	
176	Soojin	Lee	이수진	University of Massachusetts Medical School	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
177	Jaemin	Lee	이재민	Samsung Ventures	Cell and Gene Therapy/Rare Disease
178	Suji	Lee	이수지	Access Bio	
179	JUNG KUG	LEE	이정국	KHIDI USA	Business Development/Venture Capital/Legal/Consulting/Government Relations
180	Eun Joung	Lee	이은정	SK bioscience	

	First name	Last name	이름	근무처	Networking Group
181	Jung Hwa	Lee	이정화	Eisai Inc	Medical Chemistry/Drug Discovery&Delivery/Preclinical
182	Joohee	Lee	이주희	Fox Rothschild LLP	Business Development/Venture Capital/Legal/Consulting/Government Relations
183	TAE HYUNG	LEE	이태형	Beam Therapeutics	
184	Matt	Lee	이승주	Beam Therapeutics	
185	Hyejin	Lee	이혜진	Duke University	Medical Chemistry/Drug Discovery&Delivery/Preclinical
186	Dooyoung	Lee	이두영	Morphic Therapeutic	Clinical Trial & Development / Clinical Pharmacology/Biostatistics
187	Doheon	Lee	이도현	MCPHS University	
188	Eun-Joo	LEE	이은주	BIDMC	Metabolic Diseases/Cardiovascular/Diabetes/Respiratory Diseases
189	Mina	Lee	이미나	Myriad Life Sciences Co.,LTD	Cell and Gene Therapy/Rare Disease
190	JU-HYUN	LEE	이주현	Nathan Kline Institute/NYU Langone Medical Center	Medical Chemistry/Drug Discovery&Delivery/Preclinical
191	Jisun	Lee	이지선	Umass Medical School	
192	Seungkyu	Lee	이승규	Bristol Myers Squibb	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
193	You-Kyung	Lee	이유경	Icahn school of medicine at mount sinai	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
194	Yoojung	Lee	이유정	Northeastern University	Business Development/Venture Capital/Legal/Consulting/Government Relations
195	Yang	Lee	이양	GSK	
196	Jooyoung	Lee	이주영	Vertex Pharmaceuticals Incorporated	
197	Yejin	Lee	이예진	Yuhan USA	Business Development/Venture Capital/Legal/Consulting/Government Relations
198	Agatha	Lee	이아가타	Batavia Biosciences ( CJ Biosciences)	Cell and Gene Therapy/Rare Disease
199	Jisun	Lee	이지선	University of Massachusetts Chan Medical School	Cell and Gene Therapy/Rare Disease
200	Hyunjoo	Lee	이현주	LG Chem	Cell and Gene Therapy/Rare Disease
201	Juneyoung	Lee	이준영	Moderna	Medical Chemistry/Drug Discovery&Delivery/Preclinical

	First name	Last name	이름	근무처	Networking Group
202	Joon-Hyuk	Lee	이준혁	Brigham and women's hospital	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
203	Yunjin	Lee	이윤진	Harvard medical school	Neurological Disorders/Alzheimer's Disease/Parkinson's Disease/Aging
204	Hyunho	Lee	이현호	Houston Methodist Research Institute	Metabolic Diseases/Cardiovascular/Diabetes/Respiratory Diseases
205	Dongho	Lee	이동호	BioDesigners	Cell and Gene Therapy/Rare Disease
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296	Jeemin	Yoo	유지민	Northeastern University	
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298	Cheol-min	Yook	육철민	Bridge Biotherapeutics	Medical Chemistry/Drug Discovery&Delivery/Preclinical
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304	Chongwoo	Yu	유종우	US Food and Drug Administration	Clinical Trial & Development / Clinical Pharmacology/Biostatistics
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- 자체 기술력을 바탕으로 글로벌 기관 및 기업들과 협력해 코로나19 백신 등 자체 백신 연구 개발
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행복과 성공에 대한 간절한 소망을 명확하게 그리며 모든 일을 열정을 가지고 즐겁게 실천하는 SK바이오사이언스의 인재상을 가리킵니다.



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