KHUPO 2025 BUSAN

10.15.-10.17. BPEX

BON VOYAGE TO THE NEXT GENERATION PROTEOMICS

ABSTRACT BOOK

KHUPO
The Korean Human Proteome Organization

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KRISS

한국표준과학연구원 창립 50주년

50년의 신뢰 세상의 기준

KRISS 한국표준과학연구원
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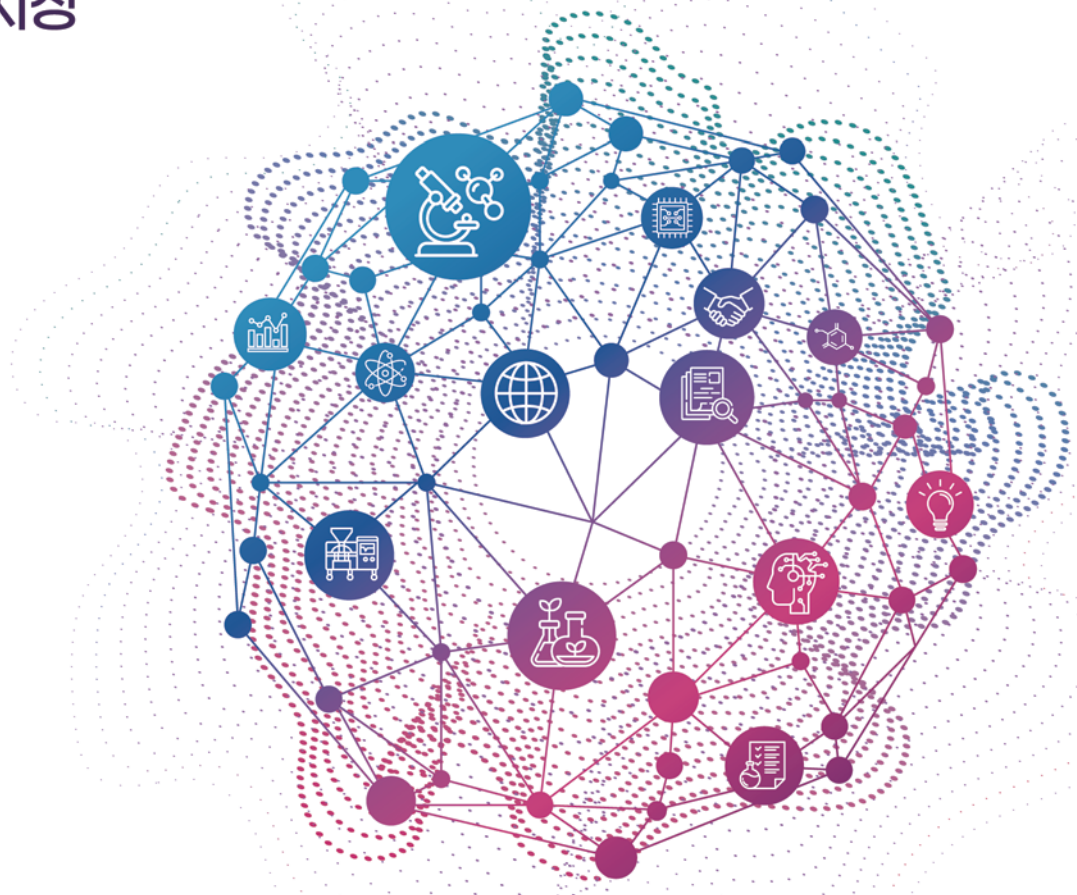
IRIC 2025 국제 연구산업 컨벤션 2025

International R&D Industry-Con

연구산업 AI 융합으로 R&D 혁신과 국가 경쟁력 강화

2025.**10.28**화 – **29**수

코엑스 마곡 전시장



▲홈페이지 바로가기

주최 _ 과학기술정보통신부 Ministry of Science and ICT

공동주관 _ 한국연구산업협회 KOREA R&D INDUSTRY ASSOCIATION

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Exosome
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전체 단백체에 대한 정성 및 정량 분석

TARGETED PROTEOMICS

MRM/PRM법을 적용한
타겟 단백질에 대한 정량 분석

LIPIDOMICS

20종의 lipid 표준물질을 이용한 lipid 정성 및
정량 분석

SINGLE CELL PROTEOMICS

단일 세포에서의 단백질 정성 및 정량 분석

BIOINFORMATICS

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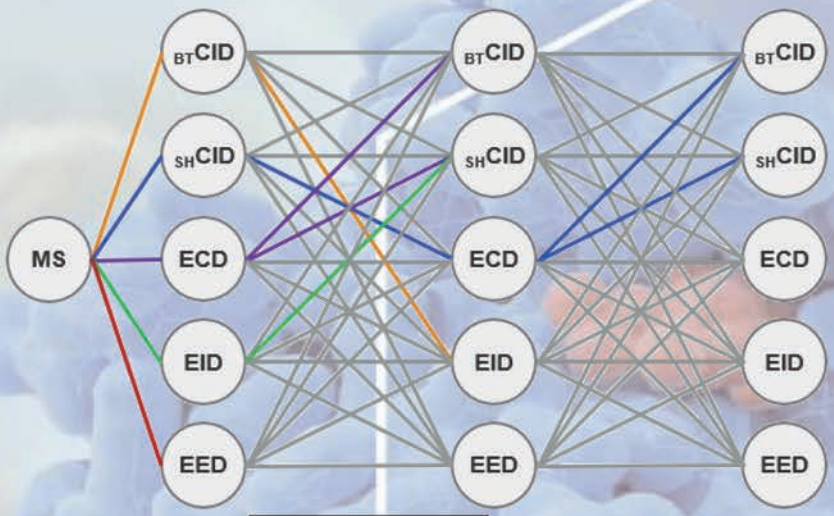

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- Structural characterization of glycopeptides

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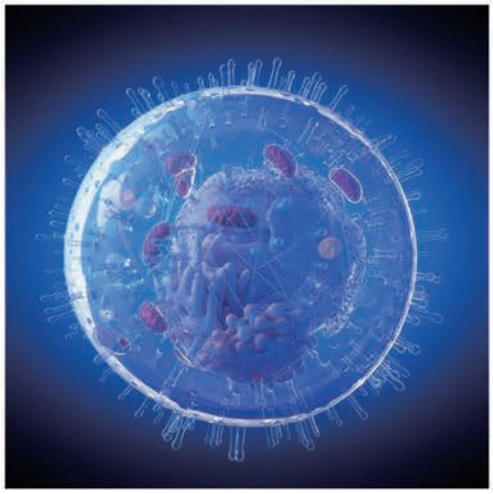

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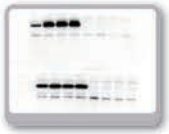

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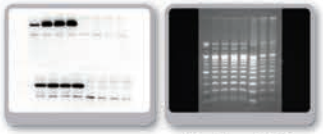
(자동 조리개 및 필터휠 조절)



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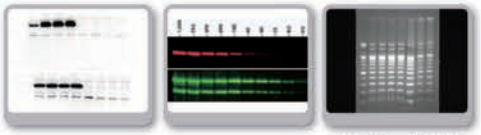

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KHUFO 2025 BUSAN

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BON VOYAGE
TO THE NEXT
GENERATION
PROTEOMICS

CONTENTS

Welcome Message

존경하는 한국단백체학회 회원 여러분께,

한국단백체학회의 회장직을 맡게 되어 깊은 영광과 함께 큰 책임감을 느끼고 있습니다. 회원 여러분과 함께 미래의 대한민국 단백체학 발전을 이끌어갈 수 있게 되어 매우 기쁘게 생각합니다.

우리 학회는 단백체학을 통해 생명의 본질을 이해하고, 이를 바탕으로 보건 분야에서 새로운 가능성을 열어가는 중요한 역할을 하고 있습니다.

우리 학회가 그동안 보여준 성과는 단지 학회원 개인의 연구뿐만 아니라 협력과 협업의 결과입니다. 서로 다른 전문성과 지식을 바탕으로 집단 지성을 발휘하고, 창의적이고 혁신적인 아이디어를 통해 많은 과학적 성과와 혁신을 이루어냈습니다. 앞으로도 협력의 중요성을 더욱 강조하고, 다양한 분야와의 융합을 통해 새로운 도전과 기회를 창출해 나가겠습니다.

또한, 연구와 교육, 사회 공헌 활동을 더욱 활발히 전개해 나가며, 학회의 역량을 극대화하여 전 세계적으로 우리 학회의 영향력을 넓혀가겠습니다. 이를 통해 인류의 건강과 복지에 기여하는 의미 있는 성과를 이루어낼 수 있도록 노력하겠습니다.

끝으로, 그동안 학회 발전을 위해 헌신해주신 선배님들과 동료 과학자 여러분께 깊은 감사의 말씀을 드리며, 여러분과 함께 나아갈 새로운 여정에 대한 기대와 설렘을 안고, 우리의 목표를 향해 힘차게 나아가겠습니다.

감사합니다.

한국단백체학회 12기 회장

이 진 환 드림

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	김나리	인제대학교

General Information

Overview

Title	The 23rd Annual International Proteomics Conference (KHUPO 2025)
Date	October 15 (Wed) – October 17 (Fri), 2025
Organized by	The Korean Human Proteome Organization (KHUPO)

Venue

5F, Busan Port International Exhibition & Convention Center (BPEX)

Program at a Glance

October 15 (Wed)	Satellite Session, ES 1, ES 2, Opening Ceremony, PL 1
October 16 (Thu)	SYM A1(Biomolecular Interactomics), SYM B1(Multi-Omics and Proteogenomics), SYM A2(Clinical Proteomics), SYM B2(Dark Proteome and Cancer Research), SYM A3(Recent Advances in Technology), SYM B3(KHUPO-KSEV Joint Session), PL 2, PL 3, CW 1-3, 3 Minutes Talk 1, Poster Session 1(Odd Numbers), Banquet
October 17 (Fri)	SYM A4(Single-cell and Spatial Omics), SYM B4(Glycosylation and PTMs), SYM A5(Drug Discovery), SYM B5(Young Scientist), PL 4, CW 4-6, 3 Minutes Talk 2, Poster Session 2(Even Numbers), Closing Ceremony

Secretariat (The PlanB Co., Ltd.)

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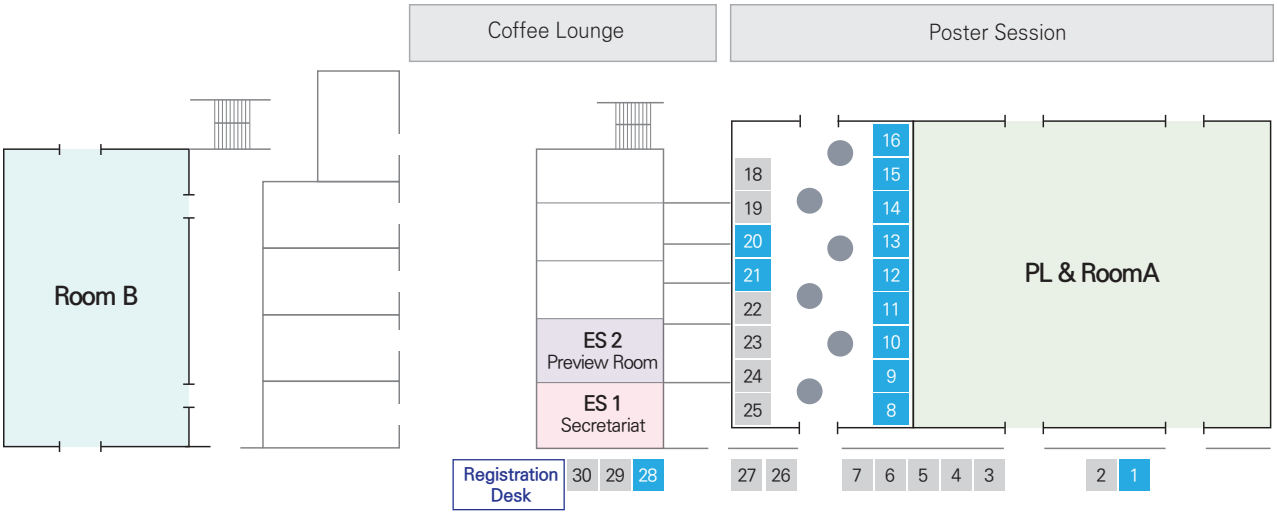
Exhibition Information



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	6
	7
소마로직 Somalogic	8
시마즈사이언티픽코리아 Shimadzu	9
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Program at a Glance

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(10.16 Thu)</th><th colspan="2">Day 3
(10.17 Fri)</th></tr>
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(Conference Hall)</td><td>Room B
(Event Hall A)</td><td>Room A
(Conference Hall)</td><td>Room B
(Event Hall A)</td></tr>
<tr><td>09:00 – 09:30</td><td colspan="2">Registration</td><td colspan="2">Registration</td></tr>
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Biomolecular Interactomics
(Korean)</td><td>SYM B1
Multi-Omics and Proteogenomics
(Korean)</td><td>SYM A4
Single-cell and Spatial Omics
(English)</td><td>SYM B4
Glycosylation and PTMs
(Korean)</td></tr>
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SCIEX
Agilent</td><td rowspan="2">illumina
BRUKER
DAON BIOSCIENCES</td></tr>
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<tr><td>12:45 – 13:05</td><td colspan="2">Coffee Break</td><td colspan="2">Coffee Break</td></tr>
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<tr><td>14:30 – 15:45</td><td rowspan="2">Registration</td><td>SYM A2
Clinical Proteomics
(English)</td><td>SYM B2
Dark Proteome and Cancer Research
(English)</td><td>SYM A5
Drug Discovery
(English)</td><td>SYM B5
Young Scientist
(English)</td></tr>
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<tr><td>16:00 – 16:10</td><td rowspan="3">ES 1
Educational Session 1
(Korean)
ES 2
Educational Session 2
(Korean)</td><td rowspan="3">SYM A3
Recent Advances in Technology
(English)</td><td rowspan="3">SYM B3
KHUPO-KSEV Joint Session
(Korean)</td><td colspan="2">General Assembly</td></tr>
<tr><td>16:10 – 16:45</td><td colspan="2">Closing Ceremony</td></tr>
<tr><td>16:45 – 17:00</td><td colspan="2" rowspan="2"></td></tr>
<tr><td>17:00 – 17:15</td><td>Coffee Break</td><td colspan="2">Coffee Break</td></tr>
<tr><td>17:15 – 17:30</td><td>Opening Ceremony</td><td colspan="2" rowspan="2">PL 3</td><td colspan="2" rowspan="3"></td></tr>
<tr><td>17:30 – 18:00</td><td>PL 1
(17:30 ~ 18:15)</td></tr>
<tr><td>18:00 ~ 20:00</td><td colspan="3">Banquet</td></tr>
</table>

Program by Date

Day 1 (10.15 Wed)

<table>
<tr><th>Time</th><th>Room A (Conference Hall)</th><th>Room B (Event Hall A)</th></tr>
<tr><td rowspan="3">Satellite Session</td><td rowspan="3">11:30–14:30
@Meeting Room 1+2
SIMPLE planet.</td><td>10:00–13:00
SPOT</td></tr>
<tr><td>13:30–15:30
SCIEX</td></tr>
<tr><td>15:30–16:30
PDCC</td></tr>
<tr><td>14:30–16:00</td><td colspan="2">Registration</td></tr>
<tr><td></td><td>[ES 1] (Korean) @Meeting Room1</td><td>[ES 2] (Korean) @Meeting Room2</td></tr>
<tr><td>16:00–16:30</td><td>“A Simple Guide to Proteomics”
Introduction to Proteomics
: Mass Spectrometry and Bioinformatics
Kwang Hoe KIM
CellKey AI Co., LTD</td><td rowspan="2">“How to Visualize My Proteomic Data?”
Integrative Proteogenomic Analysis for Revealing Biological Insight and Mechanisms
Jason Kyungha SA
Korea University College of Medicine</td></tr>
<tr><td>16:30–17:00</td><td>“Choosing Your Tool: MS or Olink?”
Exploring the Human Proteome through Affinity–Based Proteomics
Byung–Hee SHIN
Olink, part of Thermo Fisher Scientific</td></tr>
<tr><td>17:15–17:30</td><td colspan="2">Opening Ceremony</td></tr>
<tr><td>[Plenary Lecture 1]</td><td colspan="2">Chair. Cheolju LEE Korea Institute of Science and Technology (KIST)</td></tr>
<tr><td>17:30–18:15</td><td>From Organoid Genetics to Mosaic Genetics</td><td>Bon–Kyoung KOO Institute for Basic Science (IBS)</td></tr>
</table>

Program by Date

Day 2 (10.16 Thu)

Room A (Conference Hall)

Symposium A1
Biomolecular Interactomics (Korean)

Chair. Jong-Seo KIM
Seoul National University

09:30-09:50 **Development of Proximity Labeling Technologies for Local Proteome Mapping**
Song-Yi LEE Daegu Gyeongbuk Institute of Science and Technology (DGIST)

09:50-10:10 **Subcellular Proteomes and Beyond: Application of Proximity Labeling**
Kwang-eun KIM Yonsei University Wonju College of Medicine

10:10-10:30 **Ultra-specific and Efficient Bioconjugation to Nonengineered Proteins for Therapeutic Applications**
Seung Soo OH Pohang University of Science and Technology (POSTECH)

10:30-10:45 **DARTS-LC-MS/MS Target Deconvolution Reveals VDAC1 as a Key Mediator of Metformin-Induced Autophagy**
Minjeong KO Yonsei University

[Plenary Lecture 2]

Chair. Sang-Won LEE Korea University

11:00-11:45 **Precision Medicine : Omics and Clinical Data** Daehee HWANG Seoul National University

11:45-12:45 ThermoFisher SCIENTIFIC

Symposium A2
Clinical Proteomics (English)

Chair. Tadashi Kondo
National Cancer Center, Japan

14:30-14:50 **LC-MS based Blood Biomarkers for Early Detection of Alzheimer's Disease**
Zee-Yong PARK Gwangju Institute of Science and Technology (GIST)

14:50-15:10 **Exploring Extracellular Vesicle-Derived Biomarkers from Human Biofluids for Male Infertility**
Xuejiang Guo Nanjing Medical University

15:10-15:30 **Cancer Cachexia Research Using Patient-Derived Models: A Multi-Omics Approach for the Molecular Background of Disease and Novel Therapeutic Strategies**
Julia Osaki National Cancer Center, Japan

15:30-15:45 **Poly-sialylated Glycan of Cervicovaginal Fluid can be a Potential Marker of Preterm Birth**
Gun Wook PARK CellKey AI Co. LTD

Program by Date

Room A (Conference Hall)

Symposium A3
Recent Advances in Technology (English)

Chair. Je-Hyun BAEK Seegene Medical Foundation
R&D Center for Clinical Mass Spectrometry

16:00-16:20 **Mechanisms and Phygiology of the Golgi Complex**
Seung Yeol PARK Pohang University of Science and Technology (POSTECH)

16:20-16:40 **Role of Reverse Phase Protein Array Platform in Advancing Oncology at National Cancer Center Institute, Japan**
Mari Masuda National Cancer Center Research Institute, Japan

16:40-17:00 **PepQueryMHC: Rapid and Comprehensive Prioritization of Tumor Antigens Using Immunotranscriptomics Data**
Seunghyuk CHOI School of Software, College of Computer Science, Kookmin University

[Plenary Lecture 3]

Chair. Je-Yoel CHO Seoul National University

17:15-18:00 **Organelle-Targeted Chemical Proteomics : From Mechanism to Metabolic Therapy**
Ho Jeong KWON Yonsei University

Program by Date

Day 2 (10.16 Thu)

Room B (Event Hall A)

Symposium B1
Multi-Omics and Proteogenomics (Korean)

Chair. Min-Sik KIM
Daegu Gyeongbuk Institute of Science and Technology (DGIST)

09:30-09:50	**Insights from Public Multi-Omics Resources in Cancer** Hee Jin CHO Kyungpook National University
09:50-10:10	**Multi-dimensional Omics Analysis for a Comprehensive Understanding of Disease-Disease Associations** Ji Hwan PARK Ajou University
10:10-10:30	**Exploring the Regulation of Hematopoiesis Using Multi-Omics Approaches** Hye Ji CHA Dankook University
10:30-10:45	**A Multi-Omics Landscape of pQTLs in Autism Reveals Immune and Synaptic Dysregulation** Jae Won OH Kyung Hee University
11:45-12:15	SCIEX
12:15-12:45	Agilent
13:05-13:30	**3 Minutes Talk 1 (English)**

Symposium B2
Dark Proteome and Cancer Research (English)

Chair. Heeyoun HWANG
Korea Basic Science Institute (KBSI)

14:30-14:55	**There is no Dark Side of the Proteome... as a Matter of Fact, its all Dark** Christopher M Overall University of British Columbia
14:55-15:20	**Human Proteome Data as a Sustainable Public Resource** Yasushi Ishihama Graduate School of Pharmaceutical Sciences, Kyoto University
15:20-15:45	**Network-based Clinical Proteogenomics and Discovery Bioinformatics** Toshihide Nishimura St. Marianna University School of Medicine

Program by Date

Room B (Event Hall A)

Symposium B3
KHUPO-KSEV Joint Session (Korean)

Chair. Yong Song GHO / Jisook MOON
Pohang University of Science and Technology (POSTECH) / CHA University

16:00-16:20 **Extracellular Vesicle Engineering**
Jaesung PARK Pohang University of Science and Technology (POSTECH)

16:20-16:40 **Exosomes as Precision Biomarkers: From Liquid Biopsy to Next-Generation Therapeutics**
Jisook MOON CHA University

16:40-17:00 **Metabolic Reprogramming of Prostate Cancer Cells Into a Glycolysis Phenotype Induced by Extracellular Vesicles**
Dongsic CHOI Soonchunhyang University

Program by Date

Day 3 (10.17 Fri)

Room A (Conference Hall)

Symposium A4
Single-cell and Spatial Omics (English)

Chair. Youngsoo KIM
CHA University / Bundang CHA Hospital

09:30-09:55 **Filter-Aided Expansion Proteomics for Spatial Analysis of Single Cells and Organelles in FFPE Tissues**
Zhen Dong Westlake Laboratory of Life Sciences and Biomedicine

09:55-10:20 **Spatially Resolved Single-Cell Transcriptomics for the Tumor Microenvironment of Pancreatic Cancer**
Sung Hwan LEE CHA university school of medicine

10:20-10:45 **Repurposable Neuroactive Drugs Target an AP-1/BTG-centric Neural Vulnerability of Glioblastoma**
Sohyon LEE Korea Advanced Institute of Science & Technology (KAIST)

[Plenary Lecture 4]

Chair. Jin Young Kim Korea Basic Science Institute(KBSI)

11:00-11:45 **High-Quality Peptide Evidence for Annotating Non-Canonical Open Reading Frames as Human Proteins**
Robert Moritz ISB, USA

11:45-12:45 illumina®

Symposium A5
Drug Discovery (Korean)

Chair. Junho PARK
CHA University School of Medicine

14:30-14:55 **Introduction of Time-dependent Integrative Omics Research for New Drug Target Discovery**
Sangkyu LEE Sungkyunkwan University

14:55-15:20 **Integrative Proteomics Identifies MASTL as a Master Regulator in Gastric Cancer Signaling Networks**
Jae-Young KIM Chungnam National University

15:20-15:45 **Open Microfluidic Systems for 3D Vascular Proteomics**
Jihoon KO Gachon University

16:00-16:10 **General Assembly**

16:10-16:45 **Closing Ceremony**

Program by Date

Day 3 (10.17 Fri)

Room B (Event Hall A)

Symposium B4
Glycosylation and PTMs (Korean)

Chair. Sangkyu LEE
Sungkyunkwan University

09:30–09:50	**Disrupted N-Glycosylation Drives Cognitive Dysfunction in Schizophrenia** Hyun Joo AN Chungnam National University
09:50–10:10	**Prefrontal O-glycan Sialylation as a Modulator of Depressive-like Behaviors** Boyoung LEE Institute for Basic Science (IBS)
10:10–10:30	**Histone PTMs in Glioma** Chan CHUNG Daegu Gyeongbuk Institute of Science and Technology (DGIST)
10:30–10:45	**Spray-type Modifications: Shaping the Spatial Proteome** Yun-Bin LEE Seoul National University
11:45–12:15	BRUKER
12:15–12:45	DAON BIOSCIENCES
13:05–13:30	**3 Minutes Talk 2 (English)**

Symposium B5
Young Scientists (English)

Chair. Hyun Woo PARK
Yonsei University

14:30–14:45	**Narrow-Window Data-Independent Acquisition with TMT for High-Throughput and Deep Proteomics Study** Chaewon KANG Korea University
14:45–15:00	**Development of a Quantitative Proximity Labeling Method for Mapping the Intracristal Space Proteome** Myeong-Gyun KANG Seoul National University
15:00–15:15	**Serpina1e Mediates the Exercise-induced Enhancement of Hippocampal Memory** Hyunyoung KIM Korea Brain Research Institute (KBRI)
15:15–15:30	**The Chloroplast-targeted Long Noncoding RNA CHLORELLA Mediates Chloroplast Functional Transition Across Leaf Ageing via Anterograde Signaling** Myeong Hoon KANG Department of New Biology, Daegu Gyeongbuk Institute of Science and Technology (DGIST)
15:30–15:45	**Organ-Specific Diversity of Immunogenic Glycosylation in Pigs for Xenotransplantation** Ji Eun PARK Chungnam National University

KHUFO 2025 10.15.-10.17. BUSAN BPEX

PARTNERS

- Platinum

ThermoFisher SCIENTIFIC

- Star

KRISS 한국표준과학연구원 Korea Research Institute of Standards and Science

- Gold

한국연구산업협회 KOREA R&D INDUSTRY ASSOCIATION

illumina

- Silver

BERTIS

BRUKER

SCIEX

Agilent

- Bronze

Waters™

- Others

SHIMADZU Excellence in Science

SIMPLE planet.

SPOT Spatial Proteomics & Omics Technology Consortium

PDCC

National Science Challenge Initiative AST Metastasis Research Center

다빈치-K

Plenary Lecture

Plenary Lecture 1

Bon-Kyoung KOO

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■ Education

2002.03-2006.02	Ph.D.	POSTECH
2000.03-2002.02	M.S.	POSTECH
1996.03-2000.02	B.S.	POSTECH

■ Professional Experience

2023.08-Present	Director	Institute for Basic Science
2021.12-2023.08	Associate Director	Institute for Basic Science
2017.10-2023.06	Group Leader	Institute of Molecular Biotechnology
2013.04-2017.09	Group Leader	Cambrdige Stem Cell Institute
2009.03-2013.03	PostDoc	Hubrecht Institute
2006.03-2009.02	PostDoc	POSTECH/SNU Prof Kong's lab

■ Academic Society

2025.03-Present	President	Korean Association for Genome Editing

■ Publications

- H Kim, S-Y Heo, Y-I Kim, ···, Koo BK†, YK Choi†, Diverse bat organoids provide pathophysiological models for zoonotic viruses, Science Advances. 2025 May;
- Ahn JY, Kim S, ···, Kim IS†, Kim JK†, Koo BK†, Baek SH†. Dual function of PHF16 in reinstating homeostasis of murine intestinal epithelium after crypt regeneration. Dev Cell. 2024 Aug 27.
- Wu SS, Kim S, ···, Colozza G†, Koo BK†. Red2Flpe-SCON: a versatile, multicolor strategy for generating mosaic conditional knock-out mice. Nat Comm. 2024 Jun 11;15(1):4963.
- G Colozza, H Lee, A Merenda, S Wu, ···, Koo BK †. Intestinal Paneth cell differentiation relies on asymmetric regulation of Wnt signaling by Daam1/2. Science Advances. 2023 Nov 24. 9, 47.
- S-H Sam Wu, H Lee, R Szép-Bakonyi, G Colozza, ···, Koo BK†. SCON—a Short Conditional intrON for conditional knockout with one-step zygote injection. Experimental & Molecular Medicine. 2022 Dec 09, 54,2188-2199.

From Organoid Genetics to Mosaic Genetics

Bon-Kyoung Koo

Center for Genome Engineering, Institute for Basic Science

■ Abstract

Genetics has shaped the fundamental basis of modern biology, and continuous advancements in genetic manipulation have significantly expanded our understanding of biological systems. The human organoid system is a groundbreaking, human-based model that allows the study of human-specific physiology in an in vitro setting. Various genetic engineering methodologies have been adopted to maximize the potential of this system. At the same time, organoid models do not yet fully replicate the physiological complexity of in vivo systems, maintaining the continued relevance of experimental animals such as mice. Mosaic genetics, extensively utilized in fly models, has now been adapted to mammalian systems through modifications of the Confetti allele, originally introduced to track clonal cell behavior in mouse tissues. This novel system enables red clone-specific genetic loss- and gain-of-function analyses, facilitating systematic comparisons between mutant and wild-type behaviors and their cellular interactions. A number of examples will be presented to illustrate how both organoid and mosaic genetics tools can be leveraged to gain deeper insights into the fundamental nature of biological systems.

Plenary Lecture 2

Daehee HWNAG

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Homepage	https://bioinfo.snu.ac.kr/

■ Education

1999–2003	Ph.D.	Chemical Engineering, MIT
1996–1998	M.S.	Chemical Engineering, POSTECH
1990–1996	B.S.	Chemical Engineering, POSTECH

■ Professional Experience

2019.03–present	Professor	Seoul National University, School of Biological Sciences
2013.10–2019.02	Associate Director	Institute for Basic Science
2013.10–2019.02	Professor	DGIST, Department of New Biology
2010.09–2011.08	Director	System Bio-dynamics NCRC, POSTECH

■ Publications

- A systems approach for decoding of mitochondrial retrograde signaling pathways. Science Signaling, 6, 264, rs4. doi: 10.1126/scisignal.2003266 (2013)
- Proteogenomic characterization of human early onset gastric cancer. Cancer Cell, 35, 111–124 (2019) .
- Proteogenomic landscape of human pancreatic ductal adenocarcinoma in an Asian population reveals tumor cell-enriched and immune-rich subtypes. Nature Cancer 4, 290–307 (2023)
- Identification of MYH9 as a key regulator for synoviocyte migration and invasion through secretome profiling. Annals of the Rheumatic Diseases (ARD),82(8): 1035–1048 (2023))
- Targeted deletion of CD244 on monocytes promotes differentiation into anti-tumorigenic macrophages and potentiates PD-L1 blockade in melanoma, Molecular Cancer, 23(1) :45 (2024)

Precision Medicine : Omics and Clinical Data

Daehee Hwang

School of Biological Sciences, Seoul National University, Seoul 08826, Republic of Korea

Abstract

As huge amounts of global data (genomic, epigenomic, transcriptomic, proteomic, and metabolomic) generated from a broad spectrum of specimens collected from human patients have been accumulated in public repositories, together with electronic health records and drug treatment information, biology is now becoming an informational science. Accordingly, there have been significant needs for bioinformatic methods that can effectively extract useful information from these data. In this talk, I will present two different precision medicine approaches using multi-omics data and clinical big data, respectively.

Plenary Lecture 3

Ho Jeong KWON

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■ Education

1995. 04–1998. 03	Post Doc.	Harvard University, USA
1992. 04–1885. 03	Ph.D.	University of Tokyo, Japan
1990. 04–1992. 03	M.S.	University of Tokyo, Japan
1980. 03–1984. 02	B.S.	Seoul National University

■ Professional Experience

2005–present	Professor	Department of Biotechnology, Yonsei University, Seoul, Korea
2021–present	Director(리더사업단 단장)	NRF Leader Research Initiative, Korea
2018–present	Guest Professor	Lund University, Medical School, Lund, Sweden
2015–2021	Director	NRF Global Research Lab (GRL) Initiative, Korea
2011–2019	Scientific Advisory Board	Institute Pasteur Korea (IPK)
2011–2012	Visiting Professor	Dept. of Chemical & Systems Biology, Stanford University, USA
1999–2005	Professor	Dept. of Biotechnology, Sejong University, Seoul
1983–1989	Chief Researcher	Pacific Chem. R/D Center, Seoul

■ Academic Society

2020–present	Council	Human Proteome Organization (HUPO)
2024–2024	President	KSBMB
2020–2022	President	AOHUPO
2018–2019	President	KVBM
2010–2011	President	KHUPO

■ Publications

- Published over 240, citation: 27274, h-index: 59, i-10 index: 179 (2025.07)
- Jang Y, Ko M, Lee JY, Kim JY, Lee EW, Kwon HJ*, Autophagy. (2025) 한빛사 논문
- Ko M, Kim J, Lazi R, Ym J, Lee JY, Kim JY, Gosu V, Lee Y, Choi S, Kwon HJ*, EMM 56 (12), 2714 (2024) 한빛사 논문
- Park SI, Cho SM, Atsumi S, Kawada M, Shibuya M, Lee JY, Kim JY, Kwon HJ*, J Proteome. Res., 23 (1), 130–141 (2023)
- Jung Y, Lee SE, Kang I, Cho SM, Kang KS*, Kwon HJ*, Clin Transl Med. 12(4), (2022) 한빛사 논문

Organelle-Targeted Chemical Proteomics : From Mechanism to Metabolic Therapy

Ho Jeong KWON

Chemical Genomics Leader Research Laboratory, Department of Biotechnology,
College of Life Science & Biotechnology, Yonsei University, Seoul 03772, Republic of Korea. kwonhj@yonsei.ac.kr

Abstract

Recent advances in chemical proteomics have opened new avenues for mapping drug–target interactions within complex cellular environments, particularly in relation to metabolic organelle signaling. Our laboratory has focused on defining the “disease–organelle–metabolism axis” as a conceptual framework for identifying actionable targets in inflammatory and metabolic disorders.

Using DARTS–LC–MS/MS–based profiling, we recently identified VDAC1 as a previously unrecognized effector of metformin, mediating its autophagy–inducing and anticancer effects through mitochondrial membrane remodeling (Exp Mol Med, 2024). In a separate study, we uncovered LAMTOR1 as a lysosome–anchored scaffold that governs autophagy signaling in fatty liver disease and identified Acacetin as a functional small–molecule regulator of this pathway (Autophagy, 2025).

These findings suggest that organelle–specific metabolic nodes such as lysosomal scaffold proteins and mitochondrial channels can serve as therapeutic entry points across diverse diseases. In this lecture, I will present an integrated view of how chemical proteomics approaches can elucidate spatially confined metabolic signaling and enable the rational design of modulators that act through organelle–level precision. I will also discuss the broader implications of this strategy for translational medicine, including target expansion pipelines and emerging disease models.

Keywords: Chemical Proteomics, Organelle–Metabolism Axis, Autophagy, VDAC1, LAMTOR1, Metformin, Acacetin, Mitochondria, Lysosome.

Plenary Lecture 4

Robert L. Moritz

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Dr. Robert Moritz is a Professor and Head of Proteomics Research at the Institute for Systems Biology (ISB) in Seattle, Washington, USA. He received his PhD in Biochemistry from the University of Melbourne while beginning his full-time career at the Ludwig Institute for Cancer Research in Melbourne, Australia, where he worked from 1983 to 2008. In 2008, he moved to the United States to join ISB as a faculty member.

The Moritz group develops and applies cutting-edge proteomics technologies to drug discovery, protein interaction studies, and biomarker analysis for early disease detection, enabling the investigation of complex biological questions. Dr. Moritz has published more than 330 research articles, contributed to numerous book chapters, and holds multiple patents in proteomics technologies.

Dr. Moritz has served as Chair of the Human Proteome Project (HPP), Vice President of the Human Proteome Organization (HUPO), and as a committee member of USHUPO initiatives, where he plays a significant role in advancing the field and expanding the organizations. He also initiated the HUPO HPP Grand Challenge to define “a function for every protein.” He contributes to several scientific journals as a Scientific Chair and editorial board member and serves on the scientific advisory boards of several life science companies. Dr. Moritz is an entrepreneur, having established a number of life science tools companies. He is actively involved in teaching and disseminating proteomics technologies, supporting educational exchanges, and creating forums for collaborative relationships centered on the proteome.

High-Quality Peptide Evidence for Annotating Non-Canonical Open Reading Frames as Human Proteins

Robert L Moritz[1], Eric W Deutsch[1], Leron W Kok[2,3], Jonathan M Mudge[4], Jorge Ruiz-Orera[5], Ivo Fierro-Monti[4], Zhi Sun[1], Jyoti Choudhary[6], Michal Bassani-Sternberg[7,8,9], Juan Antonio Vizcaíno[4], Nicola Ternette[10,11], John R Prensner[12,13], Sebastiaan van Heesch[2,3]

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[13]Department of Biological Chemistry, University of Michigan Medical School, Ann Arbor, MI, 48109, USA

Abstract

A major scientific drive is to characterize the protein-coding genome as it provides the primary basis for the study of human health. But the fundamental question remains: what has been missed in prior genomic analyses? Over the past decade, the translation of non-canonical open reading frames (ncORFs) has been observed across human cell types and disease states, with major implications for proteomics, genomics, and clinical science. However, the impact of ncORFs has been limited by the absence of a large-scale understanding of their contribution to the human proteome. Here, we report collaborative in proteomics, immunopeptidomics, Ribo-seq ORF discovery, and gene annotation, to produce a consensus landscape of protein-level evidence for ncORFs. We show that substantial ncORFs give rise to translated gene products, yielding over 3,000 peptides in a pan-proteome analysis encompassing 3.8 billion mass spectra from 95,520 experiments. With these data, we developed an annotation framework for ncORFs and created public tools for researchers through GENCODE and PeptideAtlas. This work will provide a platform to advance ncORF-derived proteins in biomedical discovery and, beyond humans, diverse animals and plants where ncORFs are similarly observed.

KHUFO

2025 10.15.-10.17.

BUSAN BPEX

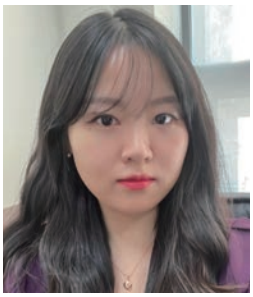
BON VOYAGE TO THE NEXT GENERATION PROTEOMICS

Symposia A

KHUPO
The Korean Human Proteome Organization

Symposia A

SYM-A1-1



Song-Yi LEE

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2014.03-2019.02	Ph.D.	Department of Chemistry, UNIST

■ Professional Experience

2019.09-2024.09	Post Doc.	Department of Genetics, Stanford

■ Publications

- Lee SY†, Roh H†, Gonzalez-Perez D, Mackey MR, Hoces D, McLaughlin CN, Lin C, Adams SR, Nguyen K, Kim KY, Luginbuhl DJ, Luo L, Udesh ND, Carr SA, Hernandez-Lopez RA, Ellisman MH, Alcalde M, and Ting AY*. Directed evolution of LaccID for cell surface proximity labeling and electron microscopy. Nature Chemical Biology, 2025, In press (†equally contributed)
- Shin SH†, Lee SY†, Kang MG, Jang DG, Kim JS, Rhee HW* and Kim JS* (2024). Super-resolution proximity labeling with enhanced direct identification of biotinylation sites. Communications Biology, 2024, 7, 554. (†equally contributed)
- Lee SY†, Cheah JS†, Zhao B, Xu C, Roh HG, Kim CK, Cho KF, Udeshi ND, Carr SA, and Ting AY. Engineered allostery in light-regulated LOV-Turbo enables precise spatiotemporal control of proximity labeling in living cells. Nature Methods 2023, 20, 908-917. (†equally contributed).

Development of Proximity Labeling Technologies for Local Proteome Mapping

Song-Yi Lee

Department of New Biology, DGIST

Abstract

Proximity labeling involves enzyme-driven chemical reactions that occur within the localized environment of living cells, facilitating the high-resolution proteomic mapping of cellular organelles and interaction networks. The two major enzyme classes used in proximity labeling are peroxidases, such as APEX2, and biotin ligases, including BioID and TurboID. While these enzyme types offer distinct benefits, they also come with notable limitations. Peroxidases require hydrogen peroxide, which is toxic to living cells and organisms. Biotin ligases can lead to high background noise in experimental results due to the presence of endogenous biotin and biotinylated proteins. Additionally, the spatial accuracy of these enzymes is constrained by the limitations of current genetic targeting techniques. To address these limitations, we have employed structure-guided protein engineering and directed evolution to develop two new proximity labeling enzymes: LOV-Turbo and LaccID.

LOV-Turbo, a novel single-chain variant of TurboID, can be reversibly turned on and off with visible light, offering controlled temporal dynamics in labeling processes. LaccID, an engineered Laccase, generates biotin-phenoxyl radicals like APEX2 but uses non-toxic oxygen instead of hydrogen peroxide, thus significantly reducing cytotoxicity. Moreover, LaccID is selectively active on the cell surface, enabling cell type-specific surface proteome analysis. We demonstrate multiple applications for each enzyme: Proximity labeling in various organelles and cell types, pulse-chase labeling to map proteome trafficking, and electron microscopy. These new PL enzymes expand the toolkit of PL methods and enable novel applications in cell and organismal biology.

Symposia A

SYM-A1-2

Kwang-eun KIM

Position	Assistant Professor
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■ Education

2017.03–2022.02	Ph.D.	Grad. Sch. of Med. Sci. & Eng., KAIST
2010.03–2015.02	B.S.	Dept. of Chem., Seoul National University

■ Professional Experience

2024.03–Present	Assistant Professor	Yonsei University Wonju College of Medicine
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■ Academic Society

2024.03–Present	Member	Kor. Soc. Biochem. Mol. Biol. (KSBMB)
2024.03–Present	Member	Korean Chemical Society (KCS)

■ Publications

- Suh Y, Kim KE. Sex- and strain-specific plasma proteomic signature in C57BL/6 and BALB/c mice. In submission
  Lee JG, Jeong I, Kim KE. Bridging molecular and cellular neuroscience with proximity labeling technologies. Exp. Mol. Med. 2025 Jul; Online
- Kim HB, Kim KE. A straightforward interpretation of proximity labeling through direct biotinylation analysis. ACS Omega 2025 Jun; 10: 26098-26105.
- Kim HB, Kim KE. Precision proteomics with TurboID: mapping the suborganelle landscape. Korean J. Physiol. Pharmacol. 2024 Nov; 28: 495-501.
- Park I*, Kim KE*, Kim J*, Kim AK, Bae S, Jung M, Choi J, Mishra PK, Kim TM, Kwak C, Kang MG, Yoo CM, Mun JY, Liu KH, Lee KS, Kim JS, Suh JM, Rhee HW. Mitochondrial matrix RTN4IP1/OPA10 is an oxidoreductase for coenzyme Q synthesis. Nat. Chem. Biol. 2024 Feb; 20: 221-233. – Featured in News & Views, Reducing mitochondrial mysteries, Nat. Chem. Biol. 2024 Feb
- Park A*, Kim KE*, Park I, Lee SH, Park KY, Jung M, Li X, Sleiman MB, Lee SJ, Kim DS, Kim J, Lim DS, Woo EJ, Lee EW, Han BS, Oh KJ, Lee SC, Auwerx J, Mun JY, Rhee HW, Kim WK, Bae KH, Suh JM. Mitochondrial matrix protein Letmd1 maintains thermogenic capacity of brown adipose tissue in male mice. Nat. Commun. 2023 Jun; 14: 3746.
- Kim KE*, Park I*, Kim J, Kang MG, Choi WG, Shin H, Kim JS, Rhee HW, Suh JM. Dynamic tracking and identification of tissue-specific secretory proteins in the circulation of live mice. Nat. Commun. 2021 Sep; 12: 5204. – Featured in Research Highlights, Revealing the secretome, Nat. Methods 2021 Sep

Subcellular Proteomes and Beyond : Application of Proximity Labeling

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Abstract

To better understand metabolic changes associated with disease processes, we have developed novel tools for tissue-specific profiling of subcellular proteomes. First, we introduce a method for in vivo mitochondrial proteome profiling using transgenic mice (MAX-Tg) that express a proximity-labeling enzyme targeted to the mitochondrial matrix. Under labeling conditions, MAX-Tg mice biotinylate proteins proximal to the enzyme in muscle mitochondria. Mass spectrometry of biotinylated proteins confirmed highly specific and efficient labeling of mitochondrial proteomes and revealed tissue-specific patterns within the matrix proteome. Our analysis identified RTN4IP1 as a mitochondrial matrix protein required for coenzyme Q biosynthesis. In addition, we developed another in vivo proximity-labeling tool, iSLET (in situ Secretory protein Labeling via ER-anchored TurboID), which targets the ER lumen to label secretory pathway proteins. Expression of iSLET in mouse liver enabled efficient labeling of the liver secretome, which could be subsequently tracked and identified in circulating plasma. Together, MAX-Tg and iSLET mice provide powerful platforms for investigating mitochondrial function and inter-organ communication networks in disease models. Finally, I will highlight the potential of proximity labeling in neuroscience, emphasizing its utility for elucidating the molecular basis of neural connectivity in both health and disease.

Symposia A

SYM-A1-3

Seung Soo OH

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■ Education

2007.09–2012.12	Ph.D.	Materials Department, University of California, Santa Barbara, U.S.A.
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1998.03–2005.02	B.S.	Department of Materials Science and Engineering, Seoul National University, South Korea

■ Professional Experience

2020.09–present	Associate Professor	Department of Materials Science and Engineering, POSTECH, South Korea
2016.06–2020.08	Assistant Professor	Department of Materials Science and Engineering, POSTECH, South Korea
2014.09–2016.06	Research Fellow	Massachusetts General Hospital/Harvard Medical School, Boston, U.S.A.
2012.12–2014.08	Post Doc.	Institute for Collaborative Biotechnologies, Santa Barbara, U.S.A.

■ Academic Society

2025–present	Chairman of Public Relations	The Korean BioChip Society
2025–present	General Director–Acad. and Edu.	The Polymer Society of Korea
2024–present	Associate Editor	BioChip Journal
2022–2024	Associate Editor	Macromolecular Research

■ Publications

- Lee M, Woo S, Oh SS. Electrochemical pan-variant detection of SARS-CoV-2 through host cell receptor-mimicking molecular recognition. Biosens Bioelectron. 2025; 278:117311
- Jo H, Ju So, Kim M, Beon J, Jang SY, Pack SP, Son CY, Kim JS, Oh SS. Aptamer-guided, hydrolysis-resistant deoxyoxanosine enables epitope- and moiety-selective conjugation to non-engineered proteins even in complex environments. J Am Chem Soc. 2025; 147:9328
- Kim J, Yoo H, Woo S, Oh SS. Aptasensor-encapsulating semi-permeable proteinosomes for direct target detection in non-treated biofluids. Biosens Bioelectron. 2024; 251:116062
- Kim M, Jo H, Jung GY, Oh SS. Molecular complementarity of proteomimetic materials for target-specific recognition and recognition-mediated complex functions. Adv Mater. 2023; 35:2208309

Ultra-Specific and Efficient Bioconjugation to Nonengineered Proteins for Therapeutic Applications

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Abstract

In protein engineering, researchers have extensively explored the incorporation of nonprotein entities into proteins to extend their functionalities to various applications; however, achieving precise modifications of proteins is still challenging. In this talk, I demonstrate epitope- and moiety-selective conjugation of nonengineered proteins by integrating "slow-reactive and hydrolysis-resistant" guanosine analogues into a "target- and epitope-selective" aptamer. The amine-reactive nucleotide-containing aptamers are dominantly single-lysine-selective at recognition sites, achieving significantly high conjugation yields with remarkably low off-target reactions in complex environments under near-physiological conditions through a catalyst-free, one-pot reaction. When stoichiometrically controlled protein-DNA conjugates are efficiently produced for various proteins, high conjugation selectivity enables semipermanent regulation of enzymatic functions, targeted labeling in a protein mixture, and even heterofunctionalization of a single protein. As our guanosine analogue-containing aptamers selectively react with the recognition sites of target proteins among nontargets, we demonstrate bioorthogonal labeling of live-cell surface nucleolin and PTK7 in amine-rich cell media, displaying their distinct distributions. Aptamer-guided positioning of amine-reactive guanosine analogues offers a promising strategy for site-specific modification of native proteins in complex environments, opening new avenues for the synergistic collaboration between nucleic acids and proteins.

Symposia A

SYM-A1-4

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■ Education

2018.03–2023.08	Ph.D. in Biotechnology (Advisor: Prof. Ho Jeong Kwon)	Yonsei University
2013.03–2018.02	B.S. in Systems Biotechnology	Chung-Ang University

■ Professional Experience

2025.03–present	Research Professor	Yonsei University
2023.08–2025.02	Postdoctoral researcher	Yonsei University

■ Academic Society

2021–2025	Member	Human Proteome Organization (HUPO)
2018–2025	Member	Korean Society for Biochemistry and Molecular Biology (KSBMB)

■ Publications

- Ko M, Kim J, Lazim R, Lee JY, Kim JY, Gosu V, Lee Y, Choi S, Kwon HJ. The anticancer effect of metformin targets VDAC1 via ER-mitochondria interactions-mediated autophagy in HCC. Exp Mol Med. 2024 Dec;56(12):2714–2725.
- Ko M, Jung HY, Lee D, Jeon J, Kim J, Baek S, Lee JY, Kim JY, Kwon HJ. Inhibition of chloride intracellular channel protein 1 (CLIC1) ameliorates liver fibrosis phenotype by activating the Ca2+-dependent Nrf2 pathway. Biomed Pharmacother. 2023 Dec;168:115776.
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- Ryu G, Ko M, Lee S, Park SI, Choi JW, Lee JY, Kim JY, Kwon HJ. Urolithin A Protects Hepatocytes from Palmitic Acid-Induced ER Stress by Regulating Calcium Homeostasis in the MAM. Biomolecules. 2024 Nov 26;14(12):1505.
- Kwak C, Park C, Ko M, Im CY, Moon H, Park YH, Kim SY, Lee S, Kang MG, Kwon HJ, Hong E, Seo JK, Rhee HW. Identification of proteomic landscape of drug-binding proteins in live cells by proximity-dependent target ID. Cell Chem Biol. 2022 Dec 15;29(12):1739–1753.e6.

DARTS–LC-MS/MS Target Deconvolution Reveals VDAC1 as a Key Mediator of Metformin-Induced Autophagy

Minjeong Ko[1], Jiho Kim[1], Raudah Lazim[2], Ju Yeon Lee[3,4], Jin Young Kim[3,4], Vijayakumar Gosu[2], Yoonji Lee[2], Sun Choi[2,*] & Ho Jeong Kwon[2,*]

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Abstract

Metformin (MetF), a widely prescribed first–line therapy for type 2 diabetes, exerts diverse pharmacological activities, including anticancer effects. Nevertheless, its direct molecular target and the interactome–level mechanisms underlying autophagy induction remain unclear. Here, we applied an unbiased, label–free Drug Affinity Responsive Target Stability (DARTS)–LC–MS/MS strategy to deconvolute protein targets of high–dose MetF in hepatocellular carcinoma (HCC) cells. This analysis identified mitochondrial voltage–dependent anion channel 1 (VDAC1) as a previously unrecognized MetF–binding protein. Binding was validated through computational alanine scanning mutagenesis, which highlighted D9 and E203 as electrostatic interaction hotspots, and further confirmed by in vitro biophysical assays. Mechanistically, MetF–VDAC1 interaction disrupted the IP_3R–GRP75–VDAC1 tethering complex at mitochondria–associated ER membranes (MAMs), thereby altering inter–organelle Ca^{2+} flux, reducing mitochondrial ATP production, and activating the AMPK–mTOR–TFEB signaling cascade. These events converged to induce robust autophagy, phenocopied by VDAC1 knockdown. Collectively, our study identifies VDAC1 as a direct ionic–binding partner of MetF, linking disruption of the IP_3R–GRP75–VDAC1 tethering complex at MAMs to autophagy induction in HCC. These results provide mechanistic insight into the anticancer actions of MetF and highlight its therapeutic potential in diseases characterized by VDAC1 overexpression.

Symposia A

SYM-A2-1

Zee-Yong PARK

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■ Education

1996.09–2001.08	Ph.D.	Texas A&M University
1991.03–1993.02	M.S.	Seoul National University
1987.03–1991.02	B.S.	Seoul National University

■ Professional Experience

2003.09–Present	Professor	Gwangju Institute of Science & Technology
2001.09.–2003.08	Post Doc.	The Scripps Research Institute

■ Academic Society

2010.03–Present	Member	Korean Human Proteome Organization
2003.09–Present	Member	Korean Society for Mass Spectrometry

■ Publications

- Pak B, Han O, Oh S, Cho H, Kim M, Lee G, Park JH, Park ZY, Kim TY, Jin SW. Lipid-Rich diet protects aquatic vertebrates by reducing polystyrene nanoparticles deposition and alleviating harmful effects from exposure. Aquat. Toxicol. 2025 Oct. 287:107516.
- Seong KJ, Park S, Jang SH, Park ZY, Jung JY, Kim WJ. Dental pulp stem cell secretome inhibits mitophagy-induced hippocampal neural injury during hypoxia. Free Radic Biol Med. 2025 Oct. 238:36-51.
- Koo H, Park KC, Sohn HA, Kang M, Kim DJ, Park ZY, Park S, Min SH, Park SH, You YM, Han Y, Kim BK, Lee CH, Kim YS, Chung SJ, Yeom YI, Lee DC. Anti-proteolytic regulation of KRAS by USP9X/NDRG3 in KRAS-driven cancer development. Nat Commun. 2025 Jan 16;16(1):628.
- Oh S, Jeong J, Park B, Kang B, Kim JY, Park S, Lee DH, Jung S, Lee M, Lee W, Yasin M, Seo J, Park ZY, Shin KH, Müller V, Choi IG, Chang IS. Acetogenesis to ethanologenesis: facilitating NADH oxidation via reductive acetate uptake. Trends Biotechnol. 2025 Mar;43(3):696-714.
- Kim SJ, Lee KM, Park SH, Yang T, Song I, Rai F, Hoshino R, Yun M, Zhang C, Kim JI, Lee S, Suh GSB, Niwa R, Park ZY, Kim YJ. A sexually transmitted sugar orchestrates reproductive responses to nutritional stress. Nat Commun. 2024 Oct 1;15(1):8477.

LC-MS based Blood Biomarker Discovery for Early Detection of Alzheimer's Disease

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[1] Department of Life Sciences, GIST,
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Abstract

The plasma phosphoproteome, like the plasma proteome, is a valuable noninvasive and economical biomarker source for various diseases. Nonetheless, the discovery and verification of plasma phosphoprotein biomarkers have been hampered by the inherent characteristics of plasma that are detrimental for phosphopeptide enrichment. Here, we expanded the coverage of the plasma phosphoproteome by optimizing existing serial phosphopeptide enrichment methods. This method identified 3,533 phosphopeptides corresponding 867 phosphoproteins from plasma samples of healthy individuals. Our plasma phosphoproteome provides the most comprehensive human plasma phosphoproteome to date. Notably, our plasma phosphoproteome showed the strongest functional association with the phosphoproteome from brain rather than from other blood components or major organs. Importantly, Alzheimer's disease-related phosphorylated brain proteins, including Tau, were detected in our plasma phosphoproteome. We finally demonstrate that Alzheimer's disease-related brain proteins in the plasma phosphoproteome can be used as a biomarker for Alzheimer's disease diagnosis by targeted mass spectrometry analysis of pooled plasma samples from patients at normal or preclinical stages, and those with mild cognitive impairment and dementia.

Symposia A

SYM-A2-2

Xuejiang Guo

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■ Education

2006.09–2009.07	Ph.D.	Nanjing Medical University
1999.09–2006.07	Bachelor and Master degrees	Nanjing Medical University

■ Professional Experience

2015.09–Now	Professor	Nanjing Medical University
2014.06–2015.06	Visiting scholar	Pacific Northwest National Laboratory (PNNL)
2011.09–2015.08	Associate Professor	Nanjing Medical University
2009.09–2011.08	Lecturer	Nanjing Medical University

■ Academic Society

2023.07–Now	Vice Chair	Young Anatomists Working Committee of the Chinese Society for Anatomical Sciences
2022.11–Now	Vice Chair	Reproductive Science Branch of the Chinese Association for Physiological Sciences
2023.12–Now	Chair-Elect	Reproductive Biology Branch of Jiangsu Society of Developmental Biology

■ Publications

- Li M, ⋯, Xie Z*, Li Y*, Guo X*. A functionally tunable magnetic nanochains platform for N-glycoproteomic analysis of extracellular vesicles from ultratrace biofluids. *Nat Commun*. 2025 Aug 16;16(1):7646.
- Zhu T,⋯, Zhu H*, Li Y*, Guo X*. Stage-Resolved Phosphoproteomic Landscape of Mouse Spermiogenesis Reveals Key Kinase Signaling in Sperm Morphogenesis. *Adv Sci (Weinh)*. 2025 Sep 3:e08538.
- Wang Y, ⋯, Guo Y*, Zhu H*, Guo X*. Single-Cell Proteomics Using the One-Step Droplet-in-Oil Digestion Method Reveals Proteins Important for Male Meiotic Progression. *Anal Chem*. 2025 Jun 3;97(21):11007-11015.

Extracellular Vesicle-derived Biomarkers from Human Biofluids for Male Infertility

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State Key Laboratory of Reproductive Medicine and Offspring Health, Department of Histology and Embryology, Nanjing Medical University, Nanjing, China

Abstract

Male infertility remains a global health challenge, with oligo–astheno–teratozoospermia being prominent etiological factors and azoospermia as the most severe form. Extracellular vesicles (EVs) in biofluids have emerged as a highly promising source of diagnostic biomarkers for male infertility. Here, we report a magnetic nanochains–based platform (EVGpro) that enables tandem EV capture, in–situ protein digestion, and N–glycopeptide enrichment from ultratrace biofluids. Compared with traditional ultracentrifugation, EVGpro demonstrates superior performance in identifying a greater number of glycoproteins across diverse biofluids. When applied to seminal plasma analysis, this platform uncovered disease–specific glycoprotein signatures in patients with asthenozoospermia (AZS). Based on EV N–glycoprotein profiles, we further classified AZS into two molecular subtypes (AZS–C1 and AZS–C2), which may deepen our understanding of disease pathogenesis and facilitate precision therapy development. For differential diagnosis of azoospermia, we purified EVs from human seminal plasma and performed integrated proteomic/phosphoproteomic analyses. Two proteins, SLC5A12 and HIST1H2BA, were identified as potential biomarkers. Notably, SLC5A12 showed 100% specificity and sensitivity in distinguishing non–obstructive azoospermia (NOA) from obstructive azoospermia (OA), while HIST1H2BA emerged as the optimal marker for subclassifying NOA subtypes. Collectively, these findings highlight the diagnostic potential of EV–derived biomarkers in male infertility, paving the way for the development of more accurate diagnostic tools and personalized therapeutic strategies.

Symposia A

SYM-A2-3

Julia Osaki

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■ Education

2018.04 – 2022.07	Ph.D.	The University of Tokyo, Graduate School of Medicine, Japan
2010.04 – 2016.03	DDS	Tokyo University of Science, Faculty of Dentistry, Japan

■ Professional Experience

2022.09 – present	Project Researcher	Division of Rare Cancer Research, National Cancer Center Research Institute, Tokyo, Japan
2018.04 – 2021.03	Clinical Staff	Oral and Maxillofacial Surgery Orthodontics The University of Tokyo Hospital, Tokyo, Japan
2016.04 – 2018.03	Resident	Oral and Maxillofacial Surgery Orthodontics The University of Tokyo Hospital, Tokyo, Japan

■ Academic Society

2023.04 – present	Board Member	Japanese Association of Patient-Derived Cancer Models
2022.06 – present	Member	Human Proteome Organization (HUPO)
2022.06 – present	Member	Japanese Human Proteome Organization (JHUPO)

■ Publications

- Noguchi R, Yanagihara K, Iino Y, Komatsu T, Kubo T, Ono T, Osaki J, Adachi Y, Iwata S, Shiota Y, Seyama T, Kondo T. Establishment and characterization of novel cancer cachexia-inducing cell line, Aku60GC, of scirrhous gastric cancer. Hum Cell. 2025 Apr 3; 38(3):82. doi:10.1007/s13577-025-01208-9. PMID: 40178664.
- Osaki J, Noguchi R, Ono T, Adachi Y, Iwata S, Toda Y, Funada T, Kojima N, Yoshida A, Kawai A, Kondo T. Establishment and characterization of NCC-MLPS4-C1: a novel cell line of myxoid liposarcoma. Hum Cell. 2025 May 26; 38(4):109. doi:10.1007/s13577-025-01241-8. PMID: 40418421.
- Noguchi R, Shiota Y, Osaki J, Kosako H, Kondo T. Proteomic analysis data of 35 patient-derived sarcoma cell lines and their original tumor tissues. J Proteome Data and Methods. 2025 Jun; accepted.

Cancer Cachexia Research Using Patient-Derived Models: A Multi-Omics Approach for the Molecular Background of Disease and Novel Therapeutic Strategies

Julia Osaki[1], Yomogi Shiota[1], Kazuyoshi Yanagihara[1], Kenta Kono[1], Shu Tingting[1], Ikumi Fijita[2], Masaki Matsumoto[3], Sumio Ohtsuki[4], Tadashi Kondo[1]

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Abstract

Cancer cachexia is a severe paraneoplastic syndrome characterized by progressive weight loss and skeletal muscle wasting. It affects approximately one third of cancer patients and is a direct cause of nearly 20% of cancer-related deaths. This syndrome leads to poor tolerance to anticancer therapies and shortened survival. Despite its high prevalence and clinical impact, no curative treatment currently exists. The condition involves multiple organs and metabolic systems, yet the molecular mechanisms underlying this systemic damage remain poorly understood.

To investigate the molecular basis of cancer cachexia, we established patient-derived cancer cell lines from gastric, pancreatic, and duodenal tumors, and generated cachexia-inducible sublines from each parental line. These sublines were used to develop murine xenograft models. From these models, we collected four cachexia-affected organs—skeletal muscle, liver, kidney, and heart—for comprehensive transcriptomic and proteomic analyses. We also performed proteomic and transcriptomic analyses on both cachexia-inducing sublines and their corresponding parental cell lines.

These xenograft models consistently exhibited marked body weight loss, skeletal muscle atrophy, and reduction of adipose tissue. The weights of multiple organs were also decreased. Comprehensive multi-omics analyses revealed molecular signatures associated with organ weight loss and functional impairment. In skeletal muscle, activation of proteasome-mediated protein degradation and suppression of mitochondrial oxidative phosphorylation were observed. The liver showed induction of acute-phase and complement-coagulation pathways coupled with metabolic rewiring. The kidney exhibited injury-response programs, and the heart demonstrated downregulation of contractile and calcium-handling proteins. Proteomic and transcriptomic profiles of cachexia-inducing tumor cell lines were further used to explore potential interactions with molecular pathways in affected organs. Several secreted proteins, including both established cachexia mediators and novel candidates, were highlighted based on higher expression in cachexia-inducing sublines compared with their parental counterparts.

Our findings demonstrate the value of combining patient-derived models with multi-omics profiling to elucidate tumor–organ communication in cancer cachexia. This framework refines the molecular landscape of cachexia and highlights actionable signaling pathways, providing a foundation for targeted biomarker development and therapeutic intervention strategies.

Symposia A

SYM-A2-4

Gun Wook PARK

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■ Education

2016.08	Ph.D.	Chungnam National University
2004.02	M.S.	Soongsil University

■ Professional Experience

2020.11-Present	CBIO	CellKey AI Co. LTD
2004.01-2020.10	Researcher	Korea Basic Science Institute (KBSI)

■ Publications

- Go YY, Park GW, Kim YJ, et al. Poly-sialylated glycan of cervicovaginal fluid can be a potential marker of preterm birth. Scientific Reports 15:11456 (2025)
- Kawahara R, Chernykh A, Park GW, Bern M, et al. Community evaluation of glycoproteomics informatics solutions reveals high-performance search strategies for serum glycopeptide analysis. Nature Methods 18:1304–1316 (2021).
- Park GW, Lee J, Lee HK, Shin JH, Kim JY, Yoo JS. Classification of Mucin-Type O-Glycopeptides Using Higher-Energy Collisional Dissociation in Mass Spectrometry. Analytical Chemistry, 25 June (2020)
- De Leoz MLA, Duewer DL, Park GW, Liu L, et al. NIST Interlaboratory Study on Glycosylation Analysis of Monoclonal Antibodies: Comparison of Results from Diverse Analytical Methods. Molecular & Cellular Proteomics 19:11–30 (2020).
- Park GW, Kim JY, Hwang H, Lee JY, et al. Integrated GlycoProteome Analyzer (I-GPA) for Automated Identification and Quantitation of Site-Specific N-Glycosylation. Scientific Reports 6:21175 (2016).

Poly-Sialylated Glycan of Cervicovaginal Fluid can be a Potential Marker of Preterm Birth

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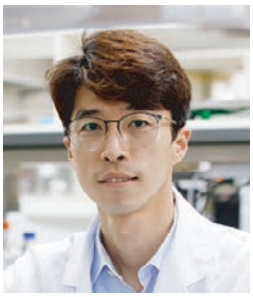
Abstract

Preterm birth remains a leading cause of neonatal mortality and lifelong morbidity, yet accurate risk stratification tools are limited. We investigated whether site-specific N-glycosylation of cervicovaginal fluid (CVF) proteins can predict preterm birth in a case-control cohort. Using high-resolution LC-MS/MS, we identified 862 N-glycoproteins and quantified 6,595 N-linked glycopeptides at a 1% FDR across 20 pregnancies. Comparative analyses revealed lower fucosylation and higher sialylation in the preterm group compared with term controls ($p<0.05$). Three polysialylated glycopeptides—CTTNBP2 N-terminal like (EENRTK+HexNAc5Hex6NeuAc3), ORM2 (NEEYNK+HexNAc5Hex6NeuAc3), and SERPINA1 (YLGNATAIFFLPDEGK+HexNAc4Hex5NeuAc2)—showed high discriminative performance (AUC 0.802–0.875), and a two-marker model further improved prediction; inclusion of clinical covariates (age, BMI) enhanced accuracy, sensitivity, and specificity. For automated, large-scale glycopeptide identification and quantification, we employed the SpAC9 glycoprotein search software to streamline site-specific N-glycopeptide calling and control false discovery at scale. Our findings indicate that polysialylated glycan signatures in CVF are robust candidates for early prediction of preterm birth and motivate translation to targeted assays for clinical validation.

Symposia A

SYM-A3-1



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■ Education

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■ Professional Experience

2019-present	Assistant /Associate Professor	POSTECH
2017-2019	Instructor	Brigham and Women's Hospital/Harvard Medical School
2011-2017	Post doc.	Brigham and Women's Hospital/Harvard Medical School
2008-2009	Visiting scholar	PNRI/University of Washington

■ Publications

- Park K, Ju S, Gao P, Chan KC, Choi H, Bang G, Choi JH, Ma X, Jang J, Morris AJ, Fan J, Kang BH, Hsu VW#, Park SY#, PITPβ promotes COPI vesicle fission through lipid transfer and membrane bending, J Cell Biol. 2025 May 5;224(5):e202407166.
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Mechanisms and Phygiology of the Golgi Complex

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Abstract

The Golgi complex serves as a central hub organelle in the secretory pathway by regulating protein sorting, trafficking, and glycosylation. Dynamic changes in architecture and function of the Golgi have been observed under various physiological conditions, including cancers and neurodegenerative diseases. However, due to both technical limitations and conceptual complexity, understanding the molecular mechanisms and functional implications of this unique organelle has remained a significant challenge in cell biology for the past decades. Here, I introduce our recent findings on bi-directional transport at the Golgi, which is coordinately regulated by lipid geometry, membrane contacts, and Golgi-specific signaling pathways. I will also present how the Golgi functions as a QC organelle acting in various biological events including immune response, neuronal development, and cellular senescence. Finally, I wish to present my ultimate goal of not only understanding fundamental biology from the perspective of organelles and membrane dynamics but also advancing its applications in bioindustry.

Symposia A

SYM-A3-2

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■ Education

1998.4–2000.9	Ph.D. in Medicine	Grad. School of Medicine, The University of Tokyo, Japan
1991.1–1995.5	M.S. in Biomedical Science	Grad. School of Hood College, Frederick, MD, USA
1980.4–1984.3	B.S. in Chemistry	Gakushuin University, Tokyo, Japan

■ Professional Experience

2021.4–present	Staff Scientist	Dep. Proteomics, NCCRI, Tokyo, Japan
2022.4–present	Lecturer	Grad. School of Rikkyo Univ., Tokyo, Japan
2017.4–2021.3	Group Leader	Div. of Cellular Signaling, NCCRI, Tokyo, Japan
2008.4–2017.3	Senior Staff Scientist	Div. of Chemotherapy, NCCRI, Tokyo, Japan

■ Academic Society

2020.4–present	Councilor	Japan Society of Clinical Proteogenomic
2017.4–present	Member	The International Cancer Proteogenome Consortium (ICPC)
2011.4–present	Member	Global Reverse-Phase Protein Array Society
2001.4–present	Councilor (2017–) Member (2001–)	Japanese Cancer Association (JCA)

■ Publications

- Masuda M*, et al., Cancer Science. 2024. May; 115(5):1378–1387. PMID:38409909.
- Hirozane T, Masuda M, et al., JCI Insight. 2021 Feb 8;6(3):e137245. PMID: 33400690
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Role of Reverse Phase Protein Array Platform in Advancing Oncology at National Cancer Center Institute, Japan

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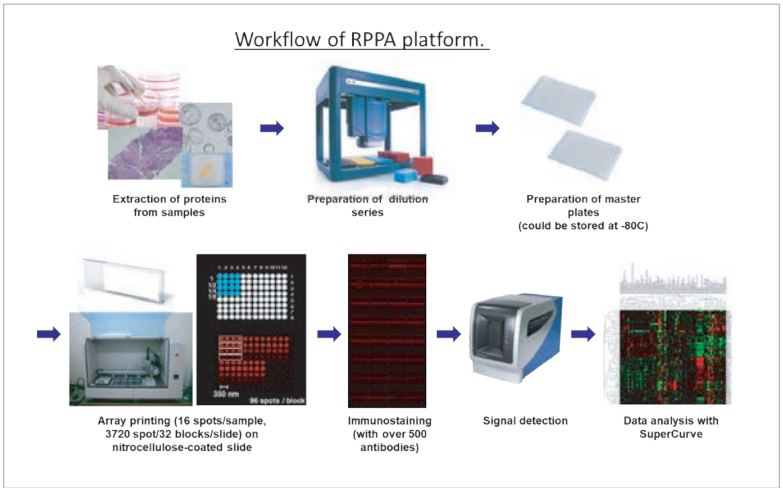
Abstract

Recent decades have brought significant advances in oncology. Comprehensive genomic profiling (CGP) has become standard for solid tumor treatment, immune checkpoint inhibitors have transformed treatment paradigms, and approved anticancer drugs continue expanding. However, genomics-driven precision oncology currently benefits only ~10% of cancer patients, underscoring the need for complementary stratification approaches. Unlike genomics, proteomics captures cancer pathogenesis more accurately, making proteomic analysis essential for precision oncology. Reverse-phase protein array (RPPA) represents one promising technology, enabling quantitative detection of post-translational modifications (PTMs) from limited clinical material. RPPA's cost-effectiveness and rapid turnaround time make it ideal for clinical applications. This talk will begin by introducing the overview of our reverse-phase protein array (RPPA) platform at the Fundamental Innovative Oncology Core (FIOC) in NCCRI and the strengths and limitations of RPPA analysis. Then, I will discuss the complementary role of reverse-phase protein array (RPPA) in improving precision oncology and its usefulness in preclinical research, including my own research, collaborative research, and its application to clinical trials in the United States. Despite its quantitative accuracy and flexibility in handling diverse samples, making it applicable across various research fields, RPPA has seen slow adoption due to the required technical expertise and challenges in kit development. In Japan, only FIOC at the National Cancer Center Research Institute offers analytical services based on collaborative research. RPPA effectively quantifies PTMs, such as phosphoproteins involved in signalling pathways and methylated/acetylated histones, often reflecting transcriptional activity. We are also establishing the workflow of comprehensive analysis by mass spectrometry analysis, with validation via RPPA. This presentation aims to contribute meaningfully to addressing current challenges while cultivating innovative opportunities for interdisciplinary collaboration.



Masuda, M. et al, Cancer Sci, 2024 May;115(5):1378-1387

Symposia A

SYM-A3-3

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2016.03–2022.08	Ph.D.	Hanyang University, Republic of Korea
2009.03–2016.02	B.S.	Hanyang University, Republic of Korea

■ Professional Experience

2025.09–current	Assistant Professor	Kookmin University, Republic of Korea
2023.12–2025.07	Postdoctoral Associate	Baylor College of Medicine, TX, USA
2022.11–2023.10	Postdoctoral Associate	Hanyang University, Republic of Korea

■ Publications

- Jangho Seo*, Seunghyuk Choi#, and Eunok Paek#. NovoRank: Post-processor for De Novo Peptide Sequencing Using Spectral Clustering and Deep Learning. Journal of Proteome Research. (2025)
- Kyu Jin Song*, Seunghyuk Choi*, Kwoneel Kim*, Hee Sang Hwang*, Eunhyong Chang, Ji Soo Park, Seok Bo Shim, Seunghwan Choi, Yong Jin Heo, Woo Ju An, Dae Yeol Yang, Kyung-Cho Cho, Wonjun Ji, Chang-Min Choi, Jae Cheol Lee, Hyeong-ryul Kim, Jiyoung Yoo, Hee-Seong Ahn, Gang-Hee Lee, Changwoong Hwa, Seoyeon Kim, Kyunggon Kim, Min-Sik Kim, Eunok Paek, Seungjin Na#, Se Jun Jang#, Joon-Yong An#, and Kwang Pyo Kim#. Proteogenomic Analysis of a Korean Cohort Reveals Lung Cancer Subtypes Predictive of Metastasis, Chromosome Instability, and Tumor Microenvironment. Nature Communications. (2024)
- Seunghyuk Choi*, and Eunok Paek#. pXg: Comprehensive identification of noncanonical MHC-I associated peptides from de novo peptide sequencing using RNA-Seq reads. Molecular & Cellular Proteomics. (2024)
- Seunghyuk Choi* and Eunok Paek#. MutCombinator: identification of mutated peptides allowing combinatorial mutations using nucleotide-based graph search. 28th Conference on Intelligent Systems for Molecular Biology (ISMB) 2020 (Bioinformatics 36, 203-209). (2020)
- Seunghyuk Choi*, Shinyeong Ju*, Jinwon Lee*, Seungjin Na, Cheolju Lee# and Eunok Paek#. Proteogenomic Approach to UTR Peptide Identification. Journal of Proteome Research. (2019)
- Seunghyuk Choi*, Hyunwoo Kim, and Eunok Paek#. ACTG: novel peptide mapping onto gene models. Bioinformatics. (2017)

PepQueryMHC: Rapid and Comprehensive Prioritization of Tumor Antigens Using Immunotranscriptomics Data

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Abstract

T cell–mediated immunity is a central component of the adaptive immune system, responsible for eliminating infected or malignant cells through recognition of specific antigens. This immune surveillance depends on the presentation of peptides bound to major histocompatibility complex (MHC) molecules (pMHCs) on the cell surface. Harnessing this mechanism, cancer immunotherapy has achieved major breakthroughs by targeting tumor–specific pMHCs, including tumor–specific antigens (TSAs) and tumor–associated antigens (TAAs). Such pMHCs may arise from mutations, gene fusions, aberrant expression, dysregulated splicing, or non–canonical translation events, and can be detected through mass spectrometry (MS)–based immunopeptidomics. However, distinguishing TSAs and TAAs from other pMHCs remains challenging due to the lack of comprehensive tumor–specificity assessment. To overcome these challenges, we developed PepQueryMHC, a computational tool that enables ultra–fast, memory–efficient comparison of class I and II pMHC sequences against translated RNA–seq reads from large–scale tumor and normal datasets using a multiple–pattern matching algorithm. This approach supports comprehensive prioritization of tumor antigens, including those originating from unannotated genomic regions. We benchmarked PepQueryMHC against the state–of–the–art tool BamQuery using immunopeptidome data from ten lung adenocarcinoma (LUAD) tumors and matched RNA–seq from tumor and adjacent normal tissues. PepQueryMHC demonstrated substantial performance gains, running 1.79–57.90 times faster and using 49.25–149.41 times less memory than BamQuery. In addition, it showed higher sensitivity in matching pMHC sequences to reads, successfully detecting pMHCs from hypermutated genomic regions that BamQuery missed, underscoring its superiority in speed, memory efficiency, and comprehensiveness. As a practical application, we used PepQueryMHC to prioritize tumor antigens from previously identified class I and II immunopeptidomes in LUAD. By integrating 2,403 RNA–seq datasets from 54 normal tissues in GTEx, 10 normal lung and 8 mTEC RNA–seq datasets, we stringently filtered candidate pMHCs. This analysis identified 18 class I and 3 class II pMHCs as potential tumor antigens: three class I neoantigens (CIC, HUWE1, KRAS), six class I non–canonical antigens, nine class I testis (or likely testis) antigens, and three class II canonical antigens. Of the 21 prioritized antigens, 12 had been reported in earlier immunopeptidomics studies. Notably, two pMHCs derived from a non–coding transcript of WDR72 were recurrently observed in glioblastoma, melanoma, and lung cancer in IEAtlas–cancer, suggesting their potential as shared TAAs. Importantly, none of the 21 pMHCs were present in normal immunopeptidome databases (HLA Ligand Atlas or IEAtlas–normal), confirming their tumor specificity. Collectively, these results demonstrate that PepQueryMHC enables efficient and comprehensive prioritization of tumor antigens, providing a powerful computational framework for the discovery of novel cancer immunotherapeutic targets.

Symposia A

SYM-A4-1

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■ Education

2017.03–2020.12	Ph.D.	University of Otago, Dunedin, New Zealand
2012.09–2016.07	M.S.	Chinese Academy of Agricultural Sciences, Beijing, China
2008.09–2012.07	B.S.	Northwest A&F University, Yangling, China

■ Professional Experience

2023.03–	Research Assistant Professor	Westlake Laboratory of Life Sciences and Biomedicine, Hangzhou, China
2021.03–2023.02	Post doc.	Westlake University, Hangzhou, China

■ Academic Society

2025.07–	Guest editor of High Resolution Spatial Proteomics at Single-Cell Levels collection	Clinical Proteomics
2024.12–	Member of Single Cell Proteomics Initiative	HUPO

■ Publications

- Dong Z#, Wu C#, Chen J, Jiang W, Piatkevich K*, Zhu Y*, Guo T*. Nat Protoc. In press.
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Filter-Aided Expansion Proteomics for Spatial Analysis of Single Cells and Organelles in FFPE Tissues

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Abstract

Understanding tissue heterogeneity at the subcellular level is critical for uncovering disease mechanisms and identifying potential therapeutic targets. High-throughput spatial proteomics with single-cell and organelle resolution provides essential technical support for such studies. Antibody-based imaging methods such as CODEX can achieve up to subcellular resolution but are limited by antibody availability and throughput, while mass spectrometry (MS) approaches such as MALDI-MS are antibody-independent yet constrained in protein coverage and resolution. Laser capture microdissection (LCM) combined with high-resolution MS has improved spatial proteomic analysis, but challenges remain below the 10–20 μm scale. To overcome these limitations, we developed Filter-Aided Expansion Proteomics (FAXP), which integrates tissue expansion with data-independent acquisition MS (DIA-MS), tailored for spatial proteomics of formalin-fixed paraffin-embedded (FFPE) tissues. Embedding tissue sections in a hydrogel followed by 4–5× linear expansion, combined with super-resolution imaging, LCM, and filter-aided sample processing, enables the collection of single-cell and even subcellular samples. Coupled with high-throughput DIA-MS, FAXP significantly surpasses the resolution limits of conventional LCM. FAXP achieves a volumetric resolution increase of up to 14.5-fold, enhances peptide yield, increases protein identifications by ~250%, and reduces sample processing time by half. In colorectal cancer FFPE tissues, more than 6,700 proteins can be identified; from single hepatocyte nuclei, nearly 2,400 proteins are quantified. Optimized expansion further enables super-resolution imaging with standard confocal microscopes to visualize subcellular structures. FAXP has been successfully applied to cholangiocarcinoma, breast cancer, and colorectal cancer FFPE samples, and has been integrated with other spatial omics methods, demonstrating broad clinical applicability. FAXP provides a robust and efficient platform for single-cell and subcellular spatial proteomics in FFPE tissues, bridging imaging and MS. Looking forward, tissue-expansion-based technologies such as FAXP may serve as the structural foundation for Artificial Intelligence Virtual Cell (AIVC), supporting multiscale biological integration and modeling. This presentation will outline the principles, applications, advantages, and limitations of FAXP, and emphasize its potential contribution to AIVC development.

Symposia A

SYM-A4-2

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■ Education

2009.09-2017.08	Ph.D.	Yonsei University College of Medicine
1997.03-2005.02	M.D.	Yonsei University Wonju College of Medicine

■ Professional Experience

2020.01-Present	Associate Professor	CHA University School of Medicine
2017.10-2019.10	Postdoctoral Fellow	Department of Systems Biology, University of Texas MD Anderson Cancer Center
2015.03-2017.08	Research Fellow	Department of Pathology, Yonsei University College of Medicine
2013.03-2015.02	Clinical Fellow	Department of Surgery, Severance Hospital

■ Academic Society

2020.03-Present	Academic Committee Member	Korean Association of Hepato-Biliary-Pancreatic Surgery (KHBPS)
2021.03-Present	Academic Committee Member	Korean Cancer Association (KCA)
2021.09-Present	Academic Committee Member	Korean Genome Organization (KOGO)

■ Publications

- Lee, S. H. et al (2024). Genomic biomarkers to predict response to atezolizumab plus bevacizumab immunotherapy in hepatocellular carcinoma: Insights from the IMbrave150 trial. Clinical and molecular hepatology, 30(4), 807.
- Lee, S. H. et al (2023). Clinically conserved genomic subtypes of gastric adenocarcinoma. Molecular Cancer, 22(1),147.
- Lee, S. H., Yim, S. Y., Jeong, Y. S., Li, Q. X., Kang, S. H., Sohn, B. H., ... & Lee, J. S. (2022). Consensus subtypes of hepatocellular carcinoma associated with clinical outcomes and genomic phenotypes. Hepatology, https://doi.org/10.1002/hep.32490
- Lee, S. H., Jeong, Y. S., Lee, S., & Lee, J. S. (2022). Two distinct stem cell-like subtypes of hepatocellular carcinoma with clinical significance and their therapeutic potentials. Cancer communications, https://doi.org/10.1002/cac2.12251

Spatially Resolved Single-Cell Transcriptomics for the Tumor Microenvironment of Pancreatic Cancer

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CHA Future Medicine Research Institute, Seongnam, KOREA

Abstract

Tumor heterogeneity and complexity are well-known causes of therapeutic resistance and poor prognosis in solid cancers. Pancreatic cancer, mainly, is well-known for its unique tumor microenvironment showing a desmoplastic reaction. Recently, new emerging technology, especially spatial transcriptomics platform, has appeared in cancer research to reveal complex tumor biology harboring in situ pathologic information of tumor microenvironment. This technology allows us to decipher the inter and intra-heterogeneity of tumor microenvironments using the spatial transcriptomic technique with collected patient samples. The formalin-fixed paraffin-embedded (FFPE) blocks, the principal repository form of clinical tumor samples, can be utilized as valuable samples to apply the spatial transcriptomic technique. Recent advances in this platform enable single-cell level sequencing for small locations in patients' high-resolution sample slides. Various subsets in the tumor microenvironment, including tumor cells, immune cells, and other cell types, can be analyzed vigorously and comprehensively, integrating with spatially resolved information. This presentation will introduce and discuss translational studies using spatial transcriptomics technology focusing on clinical usability in pancreaticobiliary cancer. The inter- and intra-tumoral heterogeneity showing different therapeutic opportunities that can be identified from spatial transcriptomic techniques warrant translational research for clinically relevant molecular deciphering with clinical samples in the era of precision oncology.

Symposia A

SYM-A4-3

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■ Education

2012.08–2018.05	Ph.D.	The Scripps Research Institute, U.S.A.
2008.03–2012.02	B.S./B.A.	Yonsei University, South Korea

■ Professional Experience

2025.01–current	Adjunct Research Fellow	Center for Genome Engineering, Institute for Basic Science (IBS)
2024.12–current	Adjunct Professor	Graduate School of Stem Cell & Regenerative Biology, KAIST
2024.10–current	Assistant Professor	Department of Biological Sciences, KAIST
2018.11–2024.08	Postdoctoral Fellow	Institute of Molecular Systems Biology, ETH Zurich, Switzerland

■ Academic Society

2025.04–2027.04	Research Committee Secretary	Korean Society for Neuro-Oncology (KSNO)
2024.10–current	Member	Korean Society for Brain and Neural Sciences (KSBNS)

■ Publications

- Lee, S.*, Weiss, T.*, Bühler, M., Mena, J., Lottenbach, Z., Wegmann, R., Sun, M., Bihl, M., Augustynek, B., Baumann, S. P., Goetze, S., van Drogen, A., Pedrioli, P. G. A., Penton, D., Festl, Y., Buck, A., Kirschenbaum, D., Zeitlberger, A. M., Neidert, M. C., … Snijder, B. (2024). High-throughput identification of repurposable neuroactive drugs with potent anti-glioblastoma activity. Nature Medicine, 30(11), 3196–3208.
- Tsunemoto, R.*, Lee, S.*, Szűcs, A., Chubukov, P., Sokolova, I., Blanchard, J. W., Eade, K. T., Bruggemann, J., Wu, C., Torkamani, A., Sanna, P. P., & Baldwin, K. K. (2018). Diverse reprogramming codes for neuronal identity. Nature, 557(7705), 375–380.

Repurposable Neuroactive Drugs Target an AP-1/BTG-Centric Neural Vulnerability of Glioblastoma

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Abstract

Glioblastoma, the most aggressive brain cancer, exhibits neural properties that can be therapeutically targeted. Our research identifies neuroactive drugs, such as vortioxetine, that exploit glioblastoma's intrinsic neurophysiological vulnerabilities, triggering AP-1/BTG-driven tumor suppression. By performing image-based high-throughput ex vivo drug screening in patient samples and machine learning of drug-target networks, we map glioblastoma's drug responses at a single-cell level, revealing a new class of neuro-oncological therapies. This approach challenges traditional cancer treatment paradigms: framing glioblastoma as a disease of dysregulated neural lineage rather than solely uncontrolled proliferation. This aligns with our lab's broader goal of understanding how gene regulatory networks shape cell identity and plasticity, influencing disease progression. By studying the intricate coordination of biology across scales and integrating deep multimodal profiling and personalized treatment strategies, we aim to restore normal cellular identity in diseased states. Our work also connects to broader questions in cancer neuroscience, where cell fate dysregulation links brain tumors to aberrant neurodevelopmental pathways. Bridging neuroscience and oncology, our research advances a framework for targeting brain cancers based on their neural origins.

Symposia A

SYM-A5-1

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■ Education

2004.03-2007.02	Ph.D.	Yeungnam University
2002.03-2004.02	M.S.	Yeungnam University
1998.03-2002.02	B.S.	Yeungnam University

■ Professional Experience

2023.02-	Professor	Sungkyunkwan University
2011.03-2023.02	Assistant Professor -Professor	Kyungpook National University
2009.03-2011.02	Post doc.	The University of Chicago
2007.03-2009.02	Post doc.	Doping Control Center, KIST

■ Academic Society

2011.03-	member	Korean Society for Mass Spectrometry
2011.03-	member	The Pharmaceutical Society of Korea

■ Publications

- Yu HC, Jeon YG, Na AY, Han CY, Lee MR, Yang JD, Yu HC, Son JB, Kim ND, Kim JB, Lee S*, Bae EJ*, Park BH*. p21-activated kinase 4 counteracts PKA-dependent lipolysis by phosphorylating FABP4 and HSL, Nature Metabolism. 2024; 6(1):94-112
- Na AY, Lee H, Ki Min E, Paudel S, Young Choi S, Sim H, Liu KH, Kim KT, Bae JS, Lee S*. Novel Time-dependent Multi-omics Integration to Explore Mechanisms of Disease Progression in sepsis-associated Liver Dysfunction, Genomics Proteomics Bioinformatics, 2023. S1672-0229(23)00068-2. Online ahead of print.
- Gao Y, Sheng X, Tan D, Kim S, Choi S, Paudel S, Lee T, Yan C, Tan M, Kim KM, Cho SS, Ki SH, Huang H*, Zhao Y*, Lee S*. Identification of Histone Lysine Acetoacetylation as a Dynamic Post-Translational Modification Regulated by HBO1. Adv Sci. 2023;10(25):e2300032.
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Introduction of Time-dependent Integrative Omics Research for New Drug Target Discovery

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Abstract

Sepsis-associated acute kidney injury (SA-AKI) is a rapidly progressing and life-threatening complication of sepsis, yet its early-stage molecular mechanisms remain poorly understood. In this study, a strategy integrating in vivo multi-omics analysis at different time points was utilized to investigate the toxic mechanism of SA-AKI. Phosphoproteomic (CLP 4 h), transcriptomic (CLP 8 h), proteomic (CLP 12 h), and metabolomic/lipidomic (CLP 24 h) datasets were collected following cecal ligation and puncture surgery, to sequentially identify the molecular mechanisms of sepsis progression. Differentially expressed molecules from each omics layer were integrated using xMWAS and Reactome for network-level analysis. Consequently, the integrated analysis suggested ferroptosis as a novel mechanism of SA-AKI. The potential involvement of ferroptosis was further supported by in vitro and in vivo validation using ferrostatin-1, a ferroptosis inhibitor. These findings suggest that time-dependent multi-omics integration provides insights into the pathological mechanisms and molecular interactions underlying SA-AKI driven by multiple pathogeneses.

Symposia A

SYM-A5-2

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■ Education

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2001.03-2003.02	M.S.	Catholic University, College of Medicine
1994.03-2001.02	B.S.	Seoul National University, College of Agriculture and Life Sciences

■ Professional Experience

2022.03-Present	Associate Professor	Chungnam National University
2016.10-2022.02	Assistant Professor	Chungnam National University
2010.08-2016.09	Post doc	H. Lee Moffitt Cancer Center & Research Institute, Tampa, FL USA
2009.02-2010.07	Post doc	Sanford-Burnham Medical Research Institute, La Jolla, CA USA

■ Academic Society

2017.03-Present	Member	KHUPO
2016.10-Present	Member	KSMCB

■ Publications

- Kim SJ, Lee SM, YJ Seo, Kim JY (2025) Proteomic characterization and bioinformatic insights into MUC18 function in gastric cancer. J Anal Sci Technol ; 16, 16.
- Han J, Shi YH, Kim E, Park HM, Kim JY (2025) Proteomic Characterization of NEDD4 Unveils Its Potential Novel Downstream Effectors in Gastric Cancer. J Proteome Res ;24(2):891-902.
- Seong H, Lee CH, Park SG, Choi KM, Lee SM, Han J, Bae HS, Han SB, Kim SJ, Kim E, Kim JY*, Song JY*(2025) Proteomic profiling of the serum of patients with COVID-19 reveals key factors in the path to clinical improvement. Clin Transl Med 15(2):e70201. (* : co-corresponding authors).

Integrative Proteomics Identifies MASTL as a Master Regulator in Gastric Cancer Signaling Networks

Sung-Jin Kim[1], Kyoung-Min Choi[1], Yoon-Hee Shin[2], Hyun-Mee Park[2], Jae-Young Kim[1]

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Abstract

Gastric cancer (GC) remains a major global health challenge, especially at advanced stages where treatment options are limited. To identify novel therapeutic targets, we profiled HSP90-dependent kinases in GC using mass spectrometry-based activity-based protein profiling (ABPP). Among the candidate kinases, microtubule-associated serine/threonine kinase-like (MASTL) was upregulated in GC and correlated with poor prognosis. Functional studies revealed that MASTL knockdown significantly reduced GC cell migration, invasion, and proliferation. Proteomic profiling of MASTL-silenced cells identified NEDD4-1, an E3 ubiquitin ligase, as a potential downstream effector. Like MASTL, NEDD4-1 was overexpressed in GC and contributed to tumor cell aggressiveness. Notably, NEDD4-1 exerted its oncogenic effects independently of the canonical PTEN/AKT pathway. Comprehensive proteomic and bioinformatic analyses following NEDD4-1 knockdown uncovered its regulatory role in cancer-associated metabolic processes and highlighted poor-prognosis proteins potentially governed by this pathway. Expanding beyond tumor-intrinsic functions, we investigated MASTL's role in the tumor microenvironment. We found that conditioned media from GC cells upregulated MASTL in primary cancer-associated fibroblasts (CAFs). Silencing MASTL in CAFs diminished their tumor-promoting functions, as assessed by impaired GC cell migration. Secretome analysis of MASTL-silenced CAFs identified key secreted proteins and signaling pathways that may mediate these effects. Together, our integrative proteomics studies identify MASTL as a central regulator of both tumor cell-intrinsic and stromal signaling in GC, offering a promising therapeutic target that spans cancer and its supportive microenvironment.

Symposia A

SYM-A5-3

Jihoon KO

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■ Education

2021.02	Ph.D. in Mechanical Engineering	Seoul National University, Seoul, Korea
2015.02	B.S. in Mechanical Engineering	Hanyang University, Seoul, Korea

■ Professional Experience

2023.03-present	Assistant Professor	Department of BioNano Technology, Gachon University
2021.09-2023.02	Research Professor	Department of Medicine, Sungkyunkwan University
2021.01-2023.02	Postdoctoral Research Associates	Division of Hematology and Oncology, Samsung Medical Center
2018.09-2023.02	Research Associates	Institute of Advanced Machines and Design, Seoul National University

■ Publications

- S. Jung, S. Cheong, Y. Lee, J. Lee, J. Lee, M-S. Kwon, Y.S. Oh, T. Kim, S. Ha, S.J. Kim, D.H. Jo, J. Ko*, N.L. Jeon*, "Integrating Vascular Phenotypic and Proteomic Analysis in Open Microfluidic Platforms", ACS Nano (2024)
- J. Ko†, S. Hyung†, M. An†, S. Jung, S.T. Kim, S.H. Park, J.O. Park, H.Y. Lim, W.K. Kang, K-M. Kim, S. Yoo, N.L. Jeon, J. Lee, "Patient-derived tumor spheroid-induced angiogenesis preclinical platform for exploring therapeutic vulnerabilities in cancer", Biomaterials (2024)
- J. Ko, D. Park, J. Lee, S. Jung, J. Lee, K. Sung, K. Baek, N.L. Jeon, "Microfluidics for High-throughput 3D Cell Culture Systems", Nature Reviews Bioengineering (2024)
- S. Hyung†, J. Ko†, Y.J. Heo, S.M Blum, S.T. Kim, S.H. Park, J.O. Park, W.K. Kang, H.Y. Lim, S.J. Klempner, J. Lee, "Patient-derived exosomes facilitate therapeutic targeting of oncogenic MET in advanced gastric", Science Advances (2023)
- J. Shin†, J. Ko†, S. Jeong, P. Won, Y. Lee, J. Kim, S. Hong, N.L. Jeon, S.H. Ko, "Monolithic digital patterning of polydimethylsiloxane with successive laser pyrolysis", Nature Materials (2021)

Open Microfluidic Systems for 3D Vascular Proteomics

Jihoon Ko

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Abstract

We present a high-throughput 3D cell culture platform based on an open microfluidic system designed for efficient vascular morphogenesis assays and functional analysis. The open-channel architecture allows easy access to the culture chamber, enabling direct manipulation, sample retrieval, and high-resolution imaging without device disassembly. By aligning endothelial cells within a 3D extracellular matrix, the platform supports robust angiogenic sprouting, which can be quantitatively analyzed using confocal microscopy. Screening of antiangiogenic compounds—including bevacizumab, ramucirumab, cabozantinib, regorafenib, wortmannin, chloroquine, and paclitaxel—revealed dose-dependent inhibition of vascular formation, with over 50% suppression at higher concentrations. This platform offers a scalable and accessible approach to study vascular responses to drugs and can be broadly applied to multi-organ models and downstream omics workflows.

KHUFO

2025 10.15.-10.17.

BUSAN BPEX

BON VOYAGE TO THE NEXT GENERATION PROTEOMICS

Symposia B

KHUPO
The Korean Human Proteome Organization

Symposia B

SYM-B1-1

Hee Jin CHO

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■ Education

2012.03-2017.08	Ph.D.	Health Sciences and Technology, Sungkyunkwan University
2007.09-2011.05	B.A.	Chemistry; Biochemistry, Boston University

■ Professional Experience

2025.04-present	Associate Professor	Department of Biomedical Convergence Science and Technology, Kyungpook National University
2021.03-2025.03	Assistant Professor	Department of Biomedical Convergence Science and Technology, Kyungpook National University
2018.01-2021.02	Senior Scientist	Precision Medicine Research Institute, Samsung Medical Center
2017.09-2017.12	Post doc. Researcher	Institute for Refractory Cancer Research, Samsung Medical Center

■ Publications

- Cho HJ*, Yun K-H, Shin S-J, Kim HS et al., Durvalumab plus pazopanib combination in patients with advanced soft tissue sarcomas: a phase II trial, Nat Comm. 15(1): 685.
- Hong JY, Cho HJ*, Sa JK, Liu X, Ha SY, et al., Hepatocellular carcinoma patients with high circulating cytotoxic T cells and intra- tumoral immune signature benefit from pembrolizumab: results from a single-arm phase 2 trial, Genome Med. 2022 Jan 6;14(1):1.
- Sa JK, Chang N, Lee HW, Cho HJ*, et al., Transcriptional regulatory networks of tumor-associated macrophages that drive malignancy in mesenchymal glioblastoma, Genome Biol. Aug 26 2020;21(1):216.
- Oh S, Yeom J, Cho HJ*, et al., Integrated pharmaco-proteogenomics defines two subgroups in isocitrate dehydrogenase wild-type glioblastoma with prognostic and therapeutic opportunities, Nat Comm. Jul 3 2020;11(1):3288.
- Cho HJ*, Zhao J, Jung SW, et al., Distinct genomic profile and specific targeted drug responses in adult cerebellar glioblastoma, Neuro-oncology, Jan 1 2019;21(1):47-58.

Insights from Public Multi-Omics Resources in Cancer

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Abstract

Although transcriptomic profiling has advanced our understanding of cancer biology, mRNA expression does not always correlate with protein abundance. This transcriptome–proteome discordance arises from post–transcriptional, translational, and post–translational regulations, and can have significant implications for biomarker discovery and therapeutic target identification. We utilized publicly available multi–omics datasets from the Clinical Proteomic Tumor Analysis Consortium (CPTAC) and The Cancer Genome Atlas (TCGA) to systematically evaluate the relationship between mRNA and protein expression across multiple cancer types. We are performing a series of statistical analyses, including correlation and regression analyses, at both the gene and pathway levels. Discordant genes are being further examined using functional enrichment analysis. Our analysis of the multi–omics data has identified a substantial subset of genes exhibiting low transcript–protein concordance. Furthermore, our findings indicate that several discordant proteins are associated with patient survival and therapeutic responses, highlighting their potential clinical relevance. This work underscores the limitations of transcriptome–only analyses and highlights the necessity of integrative multi–omics approaches for reliable biomarker discovery in cancer. By leveraging public proteogenomic resources, we demonstrate how clinically relevant insights can be uncovered that would be missed by transcriptomic profiling alone.

Symposia B

SYM-B1-2

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■ Education

2012.09–2017.08	Ph.D.	Department of Chemical Engineering, POSTECH
2008.03–2012.08	B.S.	Department of Chemical Engineering, POSTECH

■ Professional Experience

2024.09–Current	Assistant Professor	Department of Biological Science, Ajou University
2022.09–2024.08	Associate Professor	Department of Bioscience, UST
2019.03–2024.08	Senior Researcher	KOBIC, KRIBB
2017.08–2019.02	Postdoctoral Fellow	Center for Plant Aging Research, IBS

■ Academic Societies

2025.01–Current	Trustee	Korea Genome Organization (KOGO)
2023.01–Current	Trustee	Korean Society for Bioinformatics (KSBi)

■ Publications

- Kim J*, Park JL*, Yang JO*, Kim S*, Joe S*, Park G, Hwang T, Cho MJ, Lee S, Lee JE, Park JH#, Yeo MK#, Kim SY#. Highly accurate Korean draft genomes reveal structural variation highlighting human telomere evolution. Nucleic Acids Res. 2025; 53(1):gkae1294.
- Ko G*, Lee JH*, Sim YM*, Song W, Yoon BH, Byeon I, Lee BH, Kim SO, Choi J, Jang I, Kim H, Yang JO, Jang K, Kim S, Kim JH, Jeon J, Jung J, Hwang S#, Park JH#, Kim PG#, Kim SY#, Lee B#. KoNA: Korean Nucleotide Archive as A New Data Repository for Nucleotide Sequence Data. Genomics Proteomics Bioinformatics. 2024; 22(1):qzae017.
- Lee S*, Lee KY*, Park JH*, Kim DW, Oh HK, Oh ST, Jeon J, Lee D, Joe S, Chu HBK, Kang J, Lee JY, Cho S, Shim H, Kim SC, Lee HS, Kim YJ, Yang JO#, Lee J#, Kang SB#. Exploring the DNA methylome of Korean patients with colorectal cancer consolidates the clinical implications of cancer-associated methylation markers. BMB Rep. 2024; 57(3):161–166.

Multi-Dimensional Omics Analysis for a Comprehensive Understanding of Disease-Disease Associations

Ji-Hwan Park[1], Jung Hee Koh[2], Jung Gon Kim[3], Seung-Ah Yoo[4]

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[3] Division of Rheumatology, Department of Internal Medicine, Inje University Ilsan Paik Hospital, Goyang, Republic of Korea.
[4] Department of Medical Life Sciences, College of Medicine, The Catholic University of Korea, Seoul, Republic of Korea

Abstract

Understanding comorbidities and other associations between diseases is clinically important but still challenging. With the drastic improvement of omics technologies, including single-cell transcriptome analysis, diverse similarity measures between diseases have been developed based on qualitative or quantitative changes in cell types, cellular processes and signaling pathways, proteins, genes, and genetic variants. To introduce this multidimensional approach for identifying potential disease-disease associations, here, we will present one of the case studies, which is a single-cell transcriptome approach to post-COVID pulmonary fibrosis (PCPF) and idiopathic pulmonary fibrosis (IPF). Furthermore, we will also introduce a brief research plan for expanding these case studies into a generalized model by using a large-scale of biological data. Describing the multidimensional nature of diseases using this omics approach facilitates the evolution of conventional concepts for a disease-disease network, as well as the identification of uncharted clinical implications and potential therapeutic targets.

Symposia B

SYM-B1-3

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■ Education

2008.08-2014.08	Ph.D.	University of Texas at Austin
2006.03-2008.02	M.S.	Korea Advanced Institute of Science and Technology (KAIST)
2002.03-2006.02	B.S.	Korea Advanced Institute of Science and Technology (KAIST)

■ Professional Experience

2023.03-present	Assistant Professor	Dankook University
2022.04-2023.02	Instructor	Harvard Medical School
2014.09-2022.03	Research Fellow	Boston Children's Hospital, Dana-Farber Cancer Institute, and Harvard Medical School

■ Academic Societies

2025.01-present	Member	Korean Society for Molecular and Cellular Biology
2025.01-present	Member	Korean Society for Biochemistry and Molecular Biology
2024.01-present	Board Member	Korean Society of Integrative Biology
2024.01-2024.12	Council Member	Korean Society for Stem Cell Research
2024.01-2024.12	Member	Korean Society of Developmental Biology
2023.01-present	Board Member	Genetics Society of Korea
2023.01-2023.12	Member	American Society of Hematology
2013.01-2013.12	Member	Society for Developmental Biology
2011.01-2011.12	Member	American Society for Cell Biology

■ Publications

- Cha HJ. Erythropoiesis: Insights from a Genomic Perspective. Experimental & Molecular Medicine. Oct 1, 2024. 56(10):2099-2104. PMID: 39349824.
- Cha HJ, Uyan Ö, Kai Y, Liu T, Zhu Q, Tothova Z, Botten GA, Xu J, Yuan G-C, Dekker J, Orkin SH. Inner Nuclear Protein Matrin-3 Coordinates Cell Differentiation by Stabilizing Chromatin Architecture. Nature Communications. Oct 29, 2021. 12(1):6241. PMCID: PMC8556400.

Exploring the Regulation of Hematopoiesis Using Multi-Omics Approaches

Hye Ji CHA

Department of Biomedical Science & Engineering, Dankook University, Cheonan 31116, Korea

Abstract

The regulation of hematopoietic cell identity relies on the interplay between transcription factor networks and nuclear structural components that together shape the chromatin landscape. While transcription factors orchestrate lineage-specific gene expression through selective enhancer binding, the spatial organization of chromatin provides an additional layer of control over transcriptional accessibility. To investigate this coordination, we disrupted a conserved nuclear scaffold protein in human and mouse erythroid progenitor cells. Its loss resulted in widespread chromatin reorganization and acceleration of differentiation programs, accompanied by destabilization of CTCF and cohesin binding to chromatin. Similar regulatory effects were observed in muscle and embryonic stem cells, suggesting a conserved role for nuclear structural components in maintaining higher-order chromatin organization and transcriptional control. Genome-wide analyses further revealed that lineage-determining transcription factors operate in dynamic combinations to regulate distinct enhancer modules during hematopoiesis, beginning from hematopoietic stem cells. Transcriptional outputs were shaped not by individual factors alone but through context-dependent cooperativity, enabling precise integration of developmental and environmental signals. This combinatorial regulation serves as a flexible mechanism to fine-tune gene expression programs in response to differentiation cues. Together, our findings highlight how transcription factor cooperativity and nuclear architecture function in coordinated yet distinct regulatory layers to control chromatin dynamics and transcription during hematopoietic lineage progression.

Symposia B

SYM-B1-4

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■ Education

2015-2023	Ph.D.	Kyung Hee University, Seoul, Korea
2011-2015	B.S.	Konkuk University, Seoul, Korea

■ Professional Experience

2023 -	Post doc	Kyung Hee University, Seoul, Korea
2019-2022	Researcher (B.I)	Macrogen

■ Publications

- Metabolomics and proteomics analyses of zebrafish exposed to mesaconitine from Aconitum plants reveal marked reductions in lipid, glycolytic pathways. Ecotoxicology and Environmental Safety. Volume 302, 1 September 2025, 118769
- Assessing Long-Term Stored Tissues for Multi-Omics Data Quality and Proteogenomics Suitability. J Proteome Res. 2025 Aug 10. doi: 10.1021/acs.jproteome.5c00289
- KRASG12D Selective VHL-PROTAC with sparing KRASWT and Other KRAS Mutants. European Journal of Medicinal Chemistry. 5 July 2025, 117928. doi:10.1016/j.ejmech.2025.117928
- Proteomic Analysis of Aqueous Humor Identified Clinically Relevant Molecular Targets for Neovascular Complications in Diabetic Retinopathy. Mol Cell Proteomics. 2025 Mar 19;24(4):100953. doi: 10.1016/j.mcpro.2025.100953
- Peripheral blood proteome biomarkers distinguish immunosuppressive features of cancer progression. Mol Oncol. 2025 Feb 12. doi: 10.1002/1878-0261.13817
- Extracellular vesicle proteins as breast cancer biomarkers: Mass spectrometry-based analysis. Proteomics 2024 Jun
- Proteomics Analysis of Aqueous Humor and Rejected Graft in Pig-to-Non-Human Primate Corneal Xenotransplantation. Front Immunol. 2022 Mar 24;13:859929. doi: 10.3389/fimmu.2022.859929. eCollection 2022.
- Downregulation of miR-122-5p Activates Glycolysis via PKM2 in Kupffer Cells of Rat and Mouse Models of Non-Alcoholic Steatohepatitis. Int J Mol Sci. 2022 May 7;23(9):5230. doi: 10.3390/ijms23095230.

A Multi-Omics Landscape of pQTLs in Autism Reveals Immune and Synaptic Dysregulation

Jae Won OH

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Abstract

Autism spectrum disorder (ASD) is a neurodevelopmental condition shaped by both genetic and non-genetic influences, yet the protein-level mechanisms linking genetic variation to ASD remain incompletely understood. To address this, we performed deep plasma proteomics and used WGS (Whole Genome Sequencing) data in a cohort of 227 individuals, including 48 ASD cases and 42 unrelated neurotypical controls, enabling mapping of cis and trans protein quantitative trait loci (pQTLs). We identified 8,880 significant cis-pQTLs and over one million trans-pQTLs, with ASD-specific pQTLs predominantly associated with proteins upregulated in ASD. These proteins were enriched in pathways related to adaptive immune responses and extracellular matrix organization, whereas downregulated proteins were linked to adhesion and actin cytoskeleton processes.

To refine our analysis toward ASD-specific signals, we excluded proteins that showed family-shared expression patterns. We then integrated three independent approaches (ASD GWAS-based Mendelian Randomization (MR), Fisher's exact test across the full proteomics cohort, and family signal exclusion) and identified nine proteins consistently supported across all methods. Many of these top risk proteins were originally identified in human brain single nuclei transcriptomics datasets, and this revealed their regional and cell-type specificity, particularly in glia cells in the prefrontal cortex. These proteins are likely key mediators of neuroinflammatory processes that contribute to circuit dysfunction in ASD.

Our study delineates a proteogenomic landscape of pQTL in ASD risk, uncovering peripheral protein signatures mechanistically linked to neurodevelopment and immune signaling, and proposes novel blood-based biomarkers for ASD.

Symposia B

SYM-B2-1

Christopher M Overall

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■ Education

1989–1992	Centennial Post Doc	Biotechnology Lab, Dept. Biochem & Mol Biol, UBC
1984–1989	PhD	University of Toronto (Biochemistry)
1984–1989	Fellowship	MRC and J.L. Eustace Fellow, University of Toronto
–1985	MDS	University of Adelaide, Australia (Oral Biology)
–1980	BSc (Hons)	University of Adelaide, Australia (Immunology)

■ Professional Experience

2018–2020	Scientist	Creative Destruction Lab West, UBC Sauder School of Business Research
2015–2025	Associate Editor	Journal of Proteome Research
2011–	Editorial Board	Molecular Cellular Proteomics
2008	Visiting Scientist	Centre for Proteomic Chemistry, Novartis, Basel, Switzerland
2005–2008	Director	Centre for Blood Research Proteomics Hub, UBC

■ Academic Societies

2024–27, 2014–2020	Executive Committee	Human Proteome Organization (HUPO)
2019–2020	Treasurer	Human Proteome Organization (HUPO)
2023–2027	Councilor, π-HuB	π-HuB, Proteomic Navigator of the Human Body
2023–2027	Membership Committee	π-HuB, Proteomic Navigator of the Human Body
2015–2024	Chair, C-HPP	Human Proteome Organization (HUPO)

■ Publications

- 320 publications, with an h-index = 110 and 〉42,800 citations—including 37 high-impact papers in Nature (2), Science (2), Cell and their daughter journals (33), most as senior PI. Presented 〉311 keynote, plenary, and invited talks at international conferences, 250 invited seminars at universities, research institutes, and mentored 61 graduate students and PDFs (40 PDF/14 Ph.D./7 M.Sc.): 9 are Full Professors, 5 are Associate Professors, and 6 are Assistant Professors.

There is no Dark Side of the Proteome... as a Matter of Fact, its all Dark

Christopher M Overall

Centre for Blood Research, The University of British Columbia, Life Sciences Centre, Vancouver, BC Canada

Abstract

I will focus on broad consideration of the role of proteolysis as the most pervasive PTM irreversibly affecting every protein in our proteomes. There are 560 proteases in humans, many more than needed just for degradation··· in fact proteases perform proteolytic processing on virtually every protein, at one or two sites only, with high efficency and specificity, and, in virtually every case, alter processing alters protein function, e.g., activating, inactivating, maturing, switching receptors or cell location, shedding, rapidly altering protein complex formation and composition, as well as in signaling. The talk will illustrate this with key examples from inflammation, viral infection, and knockout mice and the proteomic technologies (Terminal Amino Isotopic Labelling of Substrates, TAILS, and ATOMS) to identify the neo (protease–generated) termini. As will become evident, one can not understand protein function simply from the full–length / mature protein identification. Since processing is omnipresent and clouds the functional interpretation of physiological and pathological events, without considering the protein termini, this can lead to completely wrong hypotheses or drug target identification. Indeed, many proteases are beneficial and protective ("anti–targets"). Therefore, just because a protease is elevated in a disease, it does not necessarily mean it's a target for new drugs, and this has, in fact, led to the failure of many clinical trials. Until we shed light on proteolysis, which is irreversible and affects all proteins, the entire proteome is somewhat dark or at least dim. There are many lessons to be learned and new approaches to employ to shed light on the dark aspects of the proteome to truely understand the interplay of protein and proteome composition with human growth and development and pathology.

Symposia B

SYM-B2-2

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■ Education

1998.05	Ph. D.	Kyoto University
1990.04-1992.03	M. Engr.	Kyoto University
1986.04-1990.03	B. Engr.	Kyoto University

■ Professional Experience

2010.10-present	Professor	Grd Schl of Pharm Sci, Kyoto University
2006.04-2010.09	Associate Professor	Institute for Advanced Biosciences, Keio University
2001.07-2003.07	Guest Researcher	University of Southern Denmark
1992.04-2006.03	Principal Researcher	Eisai Co. Ltd.

■ Academic Societies

2025-present	Chair	Asia-Oceania Mass Spectrometry International Committee (AOMSC)
2025-present	President	Division of Physical Sciences, the Pharmaceutical Society of Japan
2023-present	Vice President	Asia Oceania Human Proteome Organization (AOHUPO)
2023-2025	President	Mass Spectrometry Society of Japan
2022-Present	Editorial Board	Journal of Pharmaceutical and Biomedical Analysis Open
2021-present	Editor	DNA Research
2019-present	Editor-in-chief	Journal of Proteome Data and Methods
2013-present	Editorial Board	Molecular and Cellular Proteomics

■ Publications

- Tabata T, Yoshizawa AC, Ogata K, Chang CH, Araki N, Sugiyama N, Ishihama Y*, UniScore, a Unified and Universal Measure for Peptide Identification by Multiple Search Engines, Mol Cell Proteomics. 2025 Jul;24(7):101010.
- Nakai-Kasai A, Ogata K, Ishihama Y*, Tanaka T*, Leveraging pretrained deep protein language model to predict peptide collision cross section, Commun Chem. 2025 May 6;8(1):137.

Human proteome data as sustainable public resource

Yasushi Ishihama

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Abstract

In order to collect valuable proteome data scattered across various databases in Japan, standardize and integrate it, and share it with researchers for effective use, the Japanese Proteomics Society began development of jPOST (Japan Proteome Standard Repository/Database) in April 2015. The data repository part was publicly released in May 2016, and jPOST officially joined the ProteomeXchange Consortium (PXC) in July of the same year. Now celebrating its 10th anniversary, jPOST has established itself as a global standard repository for data deposits from around the world [1].

Currently, jPOST contains more than 3,500 projects, approximately 200,000 files, and 132 TB of data (half of which is human-derived), covering 450 species. In addition to global proteome data, it also includes PTM information such as phosphorylation and glycosylation, as well as isoforms/variants information. User registration is accepted from 54 countries, including the US, Europe, and Asia-Oceania, with 14% of projects coming from Japan. Over the past 10 years, jPOST has established reanalysis protocols and developed various sub-databases. Currently, in collaboration with the Journal of Proteome Data and Methods (JPDM), we are promoting the AI-READY conversion of registered data and advancing metadata collection. We are also actively promoting the jPOST Prime Project to further promote data utilization in collaboration with researchers outside the jPOST project.

Recently, we introduced UniScore as a re-scoring scale for accurate error rate control in ultra-large-scale data [2]. UniScore can be calculated solely from peak annotation information for individual spectra, enabling easy processing of ultra-large-scale data on small laptop PCs without the need for massive computing resources. In this presentation, I will also discuss the requirements for databases in the AI era.

1. Okuda S, Yoshizawa AC, Kobayashi D, Takahashi Y, Watanabe Y, Moriya Y, Hatano A, Takami T, Matsumoto M, Araki N, Tabata T, Iwasaki M, Sugiyama N, Kodera Y, Tanaka S, Goto S, Kawano S, Ishihama Y.*, jPOST environment accelerates the reuse and reanalysis of public proteome mass spectrometry data, Nucleic Acids Res. 2025 Jan 6;53(D1):D462-D467.
2. Tabata T, Yoshizawa AC, Ogata K, Chang CH, Araki N, Sugiyama N, Ishihama Y.*, UniScore, a Unified and Universal Measure for Peptide Identification by Multiple Search Engines, Mol Cell Proteomics. 2025 Jul;24(7):101010.

Symposia B

SYM-B2-3

Toshihide Nishimura

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■ Education

1980.3	Doctor of Philosophy	University of Tsukuba, Japan

■ Professional Experience

2019.4-present	Visiting Professor	Department of Chest Surgery, St. Marianna University School of Medicine, Japan
2015.12-2019.3	Specially Appointed Professor	Department of Translational Medicine Informatics, St. Marianna University School of Medicine, Japan
2014.10-present	Senior Scientist & Honorary Member	Center for Biomedical Mass Spectrometry,Lund University, Sweden; European Cancer Moonshot Center, Lund, Sweden
2002.12-2006.7	Professor	Clinical Proteome Center, Tokyo Medical University, Tokyo, Japan

■ Academic Societies

2021.7-present	Representative Director	Japanese Society of Clinical Proteogenomics (J. C-ProG)
2010.4-2014.3	Principal Investigator	Ch3 Project, Human Proteome Organization (HUPO)
2001.4-2011.3	Board of Directors	Japanese Proteomics Society (4th Conference Chairman, July 2006)

■ Publications

- Végvári A, Nishimura T. Editorial: Mass spectrometry-based proteogenomics – advances in mutant proteomics and clinical oncology. Front Oncol. 2024; 14:1383838.
- Nishimura T., Végvári Á, Nakamura H, Fujii K, Sakai H, Naruki S, Furuya N, Saji H. Cancer cell immunity-related protein co-expression networks are associated with early-stage solid-predominant lung adenocarcinoma. Front Oncol. 2024; 14:1273780.
- Toshihide Nishimura, Haruhiko Nakamura, Developments for Personalized Medicine of Lung Cancer Subtypes: Mass Spectrometry-Based Clinical Proteogenomic Analysis of Oncogenic Mutations, in Proteogenomics (edited by Ákos Végvári), pages 115-137 (2016).

Network-based Clinical Proteogenomics and Discovery Bioinformatics

Toshihide Nishimura[1], Yasuhiko Bando[2], Tetsuya Fukuda[2], Takeshi Kawamura[3]

[1] Chest Surgery, St. Marianna University School of Medicine, Kanagawa, Japan
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Abstract

Proteogenomics integrates genomic and transcriptomic data, including DNA mutations, epigenetic regulation, and RNA expression, and proteomic data including posttranslational modifications (PTMs), to comprehensively understand complex biological phenotypes such as tumors. Recent mass-spectrometry-based proteomics has been powered by its remarkably high capability of qualitative and quantitative protein sequencing. Diseases are complex systems with multi-component, hierarchical feedback loops that dynamically change over different timescales. A pivotal issue is the cancer heterogeneity in inter-patient, intra-patient, and intra-tumor, for which single cell sequencing technology has been developed in recent years. Spatial proteomics, a critical validation technique, is also attracting attention.

Since 2005 we have been developing the single-cell tissue proteomics, utilizing laser-microdissection to collect targeted cancer cells from formalin-fixed paraffin-embedded (FFPE) tissue specimens, and have successfully applied to numerous cancers, including the subtypes of lung cancer (large-cell neuroendocrine lung carcinoma, small-cell lung carcinoma, eight subtypes of lung adenocarcinomas, squamous-cell lung carcinoma), breast cancer, resectable pancreatic cancer, pancreatic ductal adenocarcinoma, extrahepatic cholangiocarcinoma, prostate cancer, and pediatric neuroblastoma (currently on-going project), etc.

Statistical approaches based on group comparisons, by their very nature, are unable to capture the "system" at the center of disease mechanisms. Even next-generation sequencing (NGS) analysis of genetic mutations makes it extremely difficult to identify the "system" underlying disease. Proteins play a vital role as dynamic functional molecules. Therefore, it is important to identify data-driven co-expressed protein networks as a "functional system", that is responsible for the abnormality of a disease. Understanding disease as a system encompassing many distinct types of networks, which centers on the cellular and molecular level of protein-protein interactions, is essential. Moreover, we have been developing our novel bioinformatics discovery analysis, named as the Discovery Bioinformatics (DB), utilizing Articicial Intelligence (AI)-driven inference engines with different algorithms to predict disease molecular mechanisms via identification of upstream regulators & causal networks, together with transcription factors (TFs), which target data-driven co-expression protein networks significantly associated with a disease.

This presentation will briefly exemplify our Discovery Bioinformatics following the single-cell tissue proteomics.

Symposia B

SYM-B3-1

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■ Education

2002	Ph.D.	University of Wisconsin-Madison, Mechanical Engineering
1997	M.S.	Seoul National University, Mechanical Engineering
1995	B.S.	BS, Pohang University of Science and Technology (Postech), Mechanical Engineering

■ Professional Experience

2007	Research Associate Post doc.	Harvard Medical School/ MGH
2023	Visiting Professor	University of Michigan

■ Academic Societies

2023	President	KSEV
2022	President	Micro-Nano System (KMEMS)

■ Publications

- S Cho, J Yi, Y Kwon, H Kang, C Han, J Park," Multifluorescence Single Extracellular Vesicle Analysis by Time-Sequential Illumination and Tracking," ACS nano 15 (7), 11753-11761
- C Han, H Kang, J Yi, M Kang, H Lee, Y Kwon, J Jung, J Lee, J Park , "Single-vesicle imaging and co-localization analysis for tetraspanin profiling of individual extracellular vesicles," Journal of Extracellular Vesicles 10 (3), e12047
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Extracellular Vesicle Engineering

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Abstract

Extracellular vesicles (EVs) are known to mediate intercellular communications and have drawn wide attention due to their potential potency in therapy and diagnostics. Although the results in therapy and diagnostics with EVs are promising, there have been inconsistencies in the findings. More reliable methods for EV isolation and analysis are needed to overcome the inconsistencies. Density-gradient ultracentrifugation is widely used for EV isolation, but the recovery efficiency is estimated to be much less than 20%. Although several methods have been tried to overcome this poor recovery, the results have not been satisfactory in terms of recovery and purity. Conventional methods for the isolation of EVs will be discussed and compared. In addition to isolation, a new method for analyzing EVs will be introduced. Most EV studies are based on ensemble EVs, even though EVs are heterogeneous. To understand the heterogeneity of EVs, a single EV analysis is required. However, the size of EV (~100nm) is below the diffraction limit, and phase-contrast microscopes cannot be used to observe EVs properly. Additionally, the fluorescence of EVs labeled with markers quenches very rapidly because the small size of EVs limits the number of fluorescence molecules on EVs. To overcome this technical challenge, a system for the visualization of single EVs has been developed; the results obtained with this system will be discussed. Unlike ensemble analysis methods, this system can count the number of EVs and type EVs simultaneously.

Symposia B

SYM-B3-2

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■ Education

2001.08-2006.08	Ph.D./ M.S.	Division of Nutritional Science/Neurosience, graduation / Bioinformatics and Statistics

■ Professional Experience

2009.01-	Associate Prof	CHA University
2006.09-2008.12	Post doc.	Harvard Medical School

■ Academic Societies

2023.01-2025.12	Chairperson of the General Affairs Committee	KSEV
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■ Publications

- Yuri Choi, Jae Hyun Park, Ala Jo, Chul-Woo Lim, Ji-Min Park, Jin Woo Hwang, Kang Soo Lee, Young-Sang Kim, Hakho Lee, Jisook Moon. Blood-derived APLP1+ extracellular vesicles are potential biomarkers for the early diagnosis of brain diseases. Science Advances. 2025, Jan (Corresponding Author)
- Kim J, Inbo H, Kim HS, Kim W, Jang SJ, Min K, Kim SH, Bae SH, Jeong YH, Kim B, Kim C, Schwarz SC, Schwarz J, Cho KG, Chung SS, Moon J. First Clinical Report on the Treatment of Parkinson's Disease with Fetal Midbrain Precursor Cells. Mov Disord. 2023 Apr (Corresponding Author)
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- Park JH, Moon J Conserved 3′ UTR of Severe Acute Respiratory Syndrome Coronavirus 2: Potential Therapeutic Targets. Front. Genet. 2022 June (Corresponding Author)
- Jae Hyun Park, Yuri Choi, Chul-Woo Lim, Ji-Min Park, Shin-Hye Yu, Yujin Kim, Hae Jung Han, Chun-Hyung Kim, Young-Sook Song, Chul Kim, Jisook Moon. Antiviral effects of miRNAs in extracellular vesicles against severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and mutations in SARS-CoV-2 RNA virus. BioRxiv. 2020. (Jisook Moon)

Exosomes as Precision Biomarkers: From Liquid Biopsy to Next-Generation Therapeutics

Yuri Choi[a]*, Jae Hyun Park[a], Ala Jo[b], Chul-Woo Lim[a], Ji-Min Park[a], Jin Woo Hwang[a], Kang Soo Lee[c], Young-Sang Kim[d], Jaejoon Lim[e], Hakho Lee[b], Jisook Moon[a,†]

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Abstract

The early detection of neurodegenerative diseases necessitates the identification of specific brain-derived biomolecules in peripheral blood. In this context, our investigation delineates the role of amyloid precursor-like protein 1 (APLP1) – a protein predominantly localized in oligodendrocytes and neurons – as a brain-specific marker in extracellular vesicles (EVs). Through rigorous analysis, $APLP1^+$ EVs from human sera were unequivocally determined to be of cerebral origin. This assertion was corroborated by distinctive small RNA expression patterns intrinsic to $APLP1^+$ EVs. Remarkably, the miRNAs' putative targets within these EVs manifested pronounced expression in the brain, fortifying their neurospecific provenance. We subjected our findings to stringent validation utilizing Thy-1 GFP M line mice, transgenic models wherein GFP expression is confined to hippocampal neurons. The congruence of GFP and APLP1 expression in plasma EVs across wild-type and transgenic models further authenticated APLP1's neural derivation.

Building on these discoveries, we propose that $APLP1^+$ EVs not only serve as robust biomarkers for early diagnosis of brain diseases but also provide a therapeutic avenue through the development of $APLP1^+$ targeting peptides. Such peptides can be engineered to guide EVs selectively toward neural cells, enabling the design of precision EV-based therapeutics for neurodegenerative disorders. An amalgamation of these results with comprehensive data analysis accentuates the dual potential of $APLP1^+$ EVs—as diagnostic markers and as platforms for targeted therapeutic development.

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Symposia B

SYM-B3-3

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■ Education

2006.03 – 2012.02	Ph.D.	Department of Life Sciences, POSTECH
2002.03 – 2006.02	B.S.	Department of Life Sciences, POSTECH

■ Professional Experience

2021.03–present	Assistant Professor	Departments of Biochemistry, Soonchunhyang University, College of Medicine
2015.12–2021.01	Research associate	The Research Institute of the McGill University Health Center
2012.03–2015.11	Postdoctoral Fellow	Department of Life Sciences, POSTECH

■ Academic Societies

2022.01–present	Board member	Korean Society of Extracellular Vesicles
2025.01–present	Board reviewer	Journal of Extracellular Vesicles
2025.01–present	Board reviewer	Journal of Extracellular Biology

■ Publications

- YJ Lee, CW Seo, S Chae, CY Lee, SS Kim, YH Shin, HM Park, YS Gho, S Ryu, SH Lee, D Choi, Metabolic Reprogramming into a Glycolysis Phenotype Induced by Extracellular Vesicles Derived from Prostate Cancer Cells, Mol Cell Proteomics. 2025 Mar 13;24(4):100944.
- Lee YJ, Chae SW, Choi D, Monitoring of single extracellular vesicle heterogeneity in cancer progression and therapy, Front Oncol 2023;13
- Choi D, Montermini L, Meehan B, Lazaris A, Metrakos P, Rak J*, Oncogenic RAS drives the CRAF-dependent extracellular vesicle uptake mechanism coupled with metastasis, J Extracell Vesicles 2021 June 10;10:e12091.
- Choi D, Go G, Kim DK, Lee J, Park SM, Di Vizio D, Gho YS*, Quantitative proteomic analysis of trypsin-treated extracellular vesicles to identify the real-vesicular proteins, J Extracell Vesicles 2020 Apr 30;9(1):1757209.
- Choi D, Montermini L, Jeong H, Sharma S, Meehan B, Rak J. Mapping subpopulations of cancer cell-derived extracellular vesicles and particles by nano-flow cytometry. ACS Nano. 2019 Sep;13(9):10499-10511.

Metabolic Reprogramming of Prostate Cancer Cells Into a Glycolysis Phenotype Induced by Extracellular Vesicles

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Abstract

Most cancer cells adopt an inefficient yet advantageous metabolic process known as aerobic glycolysis, characterized by elevated glucose uptake and lactic acid production–commonly referred to as the Warburg effect. This metabolic shift enables cancer cells to enhance survival and proliferation within the harsh, low–oxygen tumor microenvironment. Furthermore, the resulting acidic environment can suppress immune responses, including T–cell function, thereby promoting immune evasion. While numerous studies have demonstrated that tumor–derived extracellular vesicles (EVs) transfer oncogenic materials to neighboring cells, their role in regulating energy metabolism remains poorly understood. In this study, we developed acid–tolerant prostate cancer cells (PC–3AcT) capable of resisting cell death in lactic acid–enriched culture conditions. Quantitative proteomic analysis of EVs from parental PC–3 and PC–3AcT cells identified 935 high–confidence EV proteins, among which 159 were differentially expressed in response to lactic acidosis. These proteins were primarily associated with energy metabolism, cellular architecture, and the extracellular matrix. Notably, EVs derived from PC–3AcT cells were enriched in glycolytic enzymes and apolipoproteins, particularly apolipoprotein B–100 (APOB). APOB facilitated EV uptake by recipient PC–3 cells through interactions with the low–density lipoprotein receptor (LDLR), leading to enhanced cellular proliferation and survival under acidic conditions. This was accompanied by increased expression and activity of key glycolytic enzymes, including hexokinases and phosphofructokinase, resulting in elevated glucose consumption and ATP production–hallmarks of a metabolic shift toward the Warburg phenotype. Our findings provide the first evidence that prostate cancer–derived EVs can drive metabolic reprogramming in recipient cells, promoting adaptation and survival within the tumor microenvironment.

Symposia B

SYM-B4-1

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■ Education

1999–2004	Ph.D.	Chemistry, University of California, Davis
1995–1997	M.S.	Chemistry, Inha University
1991–1995	B.S.	Chemistry, Inha University

■ Professional Experience

2024.04–present	Vice President for International Affairs	Graduate School of Analytical and Science Technology (GRAST), Chungnam National University, Daejeon, Korea
2019.09–2023.09	Dean	GRAST, Chungnam National University
2011.03–present	Professor	GRAST, Chungnam National University
2011.08– present	Director	Asia-Pacific Glycomics Reference Site, Daejeon, Korea

■ Academic Societies

2011–present	Regular member	Korean Human Proteome Organization (KHUPO)
2025–present	Regular member	HUPO Diversity Representative
1999–present	Regular member	American Society for Mass Spectrometry (ASMS)
2004–present	Regular member	Korean Society for Mass Spectrometry (KSMS)

■ Publications

- MJ Oh, Y Seo, N Seo, HJ An, MS-Based Glycome Characterization of Biotherapeutics With N-and O-Glycosylation. Mass Spectrometry Reviews, (2025).
- J Lee, D Yin, J Yun, M Kim, SW Kim, H Hwang, JE Park, B Lee, CJ Lee, HS Shin, HJ An, Deciphering mouse brain spatial diversity via glyco-lipidomic mapping. Nature Communications 15, 8689 (2024).
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Disrupted N-Glycosylation Drives Cognitive Dysfunction in Schizophrenia

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Abstract

Schizophrenia is a devastating psychiatric disorder marked by profound cognitive deficits that remain resistant to current antipsychotic treatments. Post-translational modifications (PTMs), particularly protein N-glycosylation, have emerged as critical regulators of neuronal communication and brain homeostasis, yet their mechanistic contribution to schizophrenia remains poorly defined. Here, we report a direct causal link between aberrant N-glycosylation and cognitive dysfunction in a well-established schizophrenia model. Glycomic profiling of Plcβ1 knockout (KO) mice revealed a significant reduction in high-mannose-type N-glycans within the prefrontal cortex (PFC), a region central to cognitive processing. Transcriptomic analysis identified downregulation of Man1a2, an essential enzyme in high-mannose glycan maturation. Targeted knockdown of Man1a2 in the medial PFC (mPFC) reproduced severe spatial working memory impairments, while viral-mediated overexpression restored both cognitive function and neuronal excitability. Mechanistically, the glycoprotein ATP1B2, a subunit of the Na^+ /K^+ ATPase, exhibited site-specific loss of high-mannose N-glycans. Notably, combined overexpression of Man1a2 and Atp1b2 synergized with haloperidol to ameliorate both positive symptoms and cognitive deficits in Plcβ1 KO mice. Our findings establish MAN1A2-mediated N-glycosylation as a novel molecular determinant of schizophrenia-related cognitive impairment. This study provides the first mechanistic evidence that glycosylation remodeling can restore cognition, highlighting MAN1A2 and ATP1B2 as promising therapeutic targets for addressing the major unmet need of cognitive dysfunction in schizophrenia.

Symposia B

SYM-B4-2

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■ Education

2007	Ph.D. Department of Neuroscience	College of Medicine, The Ohio State University (USA)
2001	M.S. Department of Molecular Biology	Plant Molecular Biology and Biotechnology Center, Gyeongsang National University (Korea)
1999	B.S. Department of Biology	College of Natural Science, Kon-Kuk University (Korea)

■ Professional Experience

2023.09~Present	Adjunct Professor	Department of Integrative Biotechnology, Sungkyunkwan University
2021.12~Present	Adjunct Professor	Biomedical Engineering, UNIST
2021.03~Present	Associate Professor	Basic Science, IBS campus, University of Science & Technology
2020.11~Present	Senior Research Fellow	Center for Cognition and Sociality, IBS
2019.04~2020.09	Associate Research Scientist	Yale University (Supervisor: Dr. Ronald S. Duman)

■ Publications

- Seo Y, Song I, Kim KJ, Chang B, Pradeep P, Roh WS, Won W, Joo J, Kim M, Jeong JC, Lee CJ†, Lee B†. Abnormal O-Glycan Sialylation in the mPFC Contributes to Depressive-like Behaviors in Male Mice, Science Advances. In press †Corresponding authors
- Lee JH*, Jang HJ*, Hwang IY*, Lim J, Jung J, Kim M, Lee E, Won W, Lee EH, Kim KJ, Park KD, Jung W, Lee B†, Ryu S†, Lee CJ† (2025) H2O2-induced astrocytic collagen triggers neuronal death via fucosylation-dependent glial barrier formation upon ischemic stroke. bioRxiv, 2025.05. 01.651594, †Corresponding authors
- Jang HJ*, Shin J*, Lee S, Lee B†, Kim DW† (2025) Deciphering distinct spatial alterations in N-glycan expression profiles in the spinal cord and brain of male rats in a neuropathic pain model. Cell Mol Biol Lett 30, 31. https://doi.org/10.1186/s11658-025-00709-7, † Corresponding authors

Prefrontal O-Glycan Sialylation as a Modulator of Depressive-Like Behaviors

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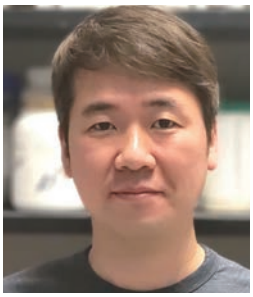
Abstract

Understanding the biological underpinnings of depression and creating effective treatments remain significant challenges. Although post-translational modifications are known to influence disease-related molecular processes, their involvement in depression is still not well defined. Among these, glycosylation has been particularly understudied due to its structural diversity and technical complexities, despite its potential to reveal novel disease mechanisms. In this work, we show that chronic stress leads to brain region-specific alterations in O-linked glycosylation, especially within the prefrontal cortex (PFC). We observed marked shifts in sialylated O-glycan profiles driven by St3gal1, a key enzyme responsible for O-glycan sialylation. Strikingly, reducing St3gal1 levels in control animals triggered depressive-like behaviors, while enhancing its expression in stressed animals mitigated these behaviors, highlighting its role in promoting stress resilience. Furthermore, we identified candidate glycoproteins regulated by St3gal1 in the medial prefrontal cortex (mPFC), such as neurexin 2 (NRXN2), pointing to specific molecular circuits that may connect O-glycosylation dynamics to mood-related outcomes.

Symposia B

SYM-B4-3



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■ Education

2010.09–2016.04	Ph.D.	University of Michigan, Medical School
2005.03–2007.08	M.S.	Seoul National University
1998.03–2005.02	B.S.	Sogang University

■ Professional Experience

2021.05–present	Assistant Professor	DGIST
2020.05–2021–04	Research Investigator	University of Michigan, Medical School
2016.01–2020.04	Postdoc. Res. fellow	University of Michigan, Medical School

■ Academic Societies

2024–present	Publicity Committee	Genetics Society of Korea
2025–present	Executive Committee	Korean Society for Neuro-Oncology (KSNO)
2024–2025	Organizing Committee	The Federation of Asian and Oceanian Biochemists and Molecular Biologists (FAOBMB)
2023–2024	Publicity Committee	Korean Society for Molecular and Cellular Biology

■ Publications

- Mbah NE,⋯, Chung C,⋯, Lyssiotis CA. Therapeutic targeting of differentiation-state dependent metabolic vulnerabilities in diffuse midline glioma. Nature Comm. 2024 Oct; 15(1): 8983.
- Venneti S,⋯, Chung C,⋯, Koschmann C. Clinical Efficacy of ONC201 in H3K27M-Mutant Diffuse Midline Gliomas Is Driven by Disruption of Integrated Metabolic and Epigenetic Pathways. Cancer Discovery. 2023 Nov; 13 (11): 2370~2393.
- Mota M,⋯, Chung C,⋯, Chinnaiyan A.M, Venneti S. Targeting SWI/SNF ATPases in H3.3K27M diffuse intrinsic pontine gliomas. PNAS of the USA. 2023 May; 120(18): e2221175120.

Histone PTMs in Glioma

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Abstract

The 2021 WHO classification of brain tumors has highlighted the importance of molecular diagnostics in defining new tumor subtypes, many of which are characterized by histone protein mutations, dysregulation of histone methyltransferase (HMT) regulators, or accumulation of onco-metabolites that alter histone-modifying enzyme activity. These epigenetic alterations drive tumor initiation and progression while maintaining a microenvironment favorable for tumor growth. Among them, changes in histone H3 lysine 27 trimethylation (H3K27me3) have emerged as a critical hallmark and therapeutic target in several brain tumors. We investigated posterior fossa group A ependymomas (PFAs), an aggressive pediatric brain tumor subtype exhibiting global loss of H3K27me3 due to overexpression of EZHIP (enhancer of Zeste homologs inhibitory protein). Through integrated multi-omics approaches combining quantitative proteomics, metabolic profiling, single-cell RNA sequencing, and noninvasive metabolic imaging of patients, we demonstrate that EZHIP-mediated loss of H3K27me3 results in profound metabolic reprogramming that enhances tumor cell survival and proliferation. Our proteomic analyses revealed dysregulated pathways linking epigenetic alterations to glycolysis, TCA-cycle metabolism and PPP, suggesting an intimate crosstalk between chromatin state and metabolic adaptation. Furthermore, we identified a small-molecule compound that reduces EZHIP levels via a histone-mediated mechanism, partially restores H3K27me3, and effectively suppresses PFA tumor growth in both patient-derived cells and in vivo models. These results underscore the value of integrating proteomic and metabolomic platforms to elucidate tumor-specific vulnerabilities and provide preclinical evidence that targeting EZHIP may serve as a promising therapeutic strategy for PFAs.

Symposia B

SYM-B4-4

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■ Education

2019.08-2025.08	Integrated M.S./Ph.D.	Chemistry, Seoul National University (Principal Investigator : prof. Hyun-Woo Rhee)
2015.03-2019.08	B.S.	Chemistry, Seoul National Univeristy

■ Professional Experience

2025.08-	Postdoctoral Reseracher	Chemistry, Seoul National University (Principal Investigator : prof. Hyun-Woo Rhee)

■ Publications

- Lee YB, Rhee HW* Spray-type modifications: an emerging paradigm in post-translational modifications. 스프레이 효소화학 반응: 단백질 변형 (PTM) 연구의 새로운 패러다임. CHEMWORLD (화학세계) of Korean chemical society (KCS, 대한 화학회), written in Korean, 2024, May, 24-31, https://ebook.kcsnet.or.kr/ecatalog5.php?Dir=205&catimage=
- Lee YB, Rhee HW* Spray-type modifications: an emerging paradigm in post-translational modifications. Trends Biochem. Sic., 2024, 49, 3, 208-223, https://doi.org/10.1016/j.tibs.2024.01.008
- Lee YB, Jung M, Kim J, Charles A, Christ W, Kang J, Kang MG, Kwak C, Klingström J, Smed-Sörensen A, Kim JS*, Mun JY*, Rhee HW* Super-resolution proximity labeling reveals antiviral protein network and its structural changes against SARS-CoV-2 viral proteins. Cell Reports, 2023, 42, 8, 112835, https://doi.org/10.1016/j.celrep.2023.112835

Spray-type Modifications : Shaping the Spatial Proteome

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Abstract

Post-translational modifications (PTMs) play a critical role in regulating protein localization and function. Previously, we proposed the concept of "spray-type" PTMs—proximity-driven modifications that occur independent of strict sequence motifs. In this study, we introduce a generalizable proximity-based platform that enables the local delivery of diverse PTMs—including acetylation, biotinylation, ubiquitination, SUMOylation, and ADP-ribosylation—by fusing PTM-catalyzing enzymes to a GFP-binding protein (GBP). These "spray-type" enzymes promiscuously modify proximal proteins in a compartment-specific manner, revealing both shared and distinct substrate preferences.

Among these, acetylation exhibited a particularly robust impact on subcellular localization. Using GBP-p300KAT, we identified thousands of local acetylation events across various compartments, called as Local Acetyl Spray. Notably, lysine residues within nuclear localization signals (NLS), including those of SV40 and TDP-43, were found to be acetylation hot spots. This modification impaired nuclear import and promoted cytoplasmic mislocalization also into stress granules. We further demonstrate that endogenously localized proteins such as NCL, and PARP1 also undergo mislocalization in an acetyl spray-dependent manner, with effects abolished by catalytically inactive p300KAT mutants. These findings reveal that lysine acetylation can act as a regulatory switch for spatial protein distribution and may contribute to pathological mislocalization events.

Our study provides a proximity-targeted framework for exploring the spatial consequences of PTMs, highlighting acetylation as a key modulator of protein localization and identifying PTM "hotspots" with potential implications for disease and therapeutic targeting.

Symposia B

SYM-B5-1

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2014.03–2018.08	B.S. in Chemistry	Sejong University, Korea

■ Publications

- Kang C, Hong J, Kim H, Jo J, Seo J.-H, Lee J.-W, Lee S.-W. A Robust Strategy for High-Throughput and Deep Proteomics Combining Narrow-Window Data-Independent Acquisition and Isobaric Mass Tagging, J. Proteome Res., 2025, under review.
- Kang C, Huh S, Nam D, Kim H, Hong J, Hwang D, Lee S.-W. Novel Online 3-Dimensional Separation Expands the Detectable Functional Landscape of Cellular Phosphoproteome, Anal. Chem., 2022, 94 (35), 12185–12195.
- Huh S, Kang C, Park J.E, Nam D, Kim S.I, Seol A, Choi K, Hwang D, Yu M.-H, Chung H.H, Lee S.-W, Kang U.-B. Novel Diagnostic Biomarkers for High-Grade Serous Ovarian Cancer Uncovered by Data- Independent Acquisition Mass Spectrometry, J. Proteome Res., 2022, 21 (9), 2146–2159.
- Kang C, Kim H, Yang H, Bok K.H, Bae J, Hong J, Lee S.-W. Wideband PRM: Highly Accurate and Sensitive Method for High-Throughput Targeted Proteomics, Anal. Chem. 2024, 96 (25), 10219–10227.
- Chae S.Y, Nam D, Hyeon D.Y, Hong A, Lee T.D, Kim S, Im D, Hong J, Kang C, Lee J.W, Hwang D, Lee S.-W, Kim H. I. DNA repair and cholesterol-mediated drug efflux induce dose-dependent chemoresistance in nutrient-deprived neuroblastoma cells, iScience., 2021, 24 (4), 102325.
- Hyeon D.Y, Nam D, Shin H.-J, Jeong J, Jung E, Cho S.Y, Shin D.H, Ku J.-L, Baek H.J, Yoo C.W, Hong E.-K, Lim M.C, Lee S.-J, Bae Y.- K, Kim J.K, Bae J, Kim S.-J, Back S, Kang C, Madar I.H, Kim H, Kim S, Kim D.K, Kang J, Park G.W, Park K.S, Shin Y, Kim S.S Jung K, Hwang D, Lee S.-W, Kim J.-Y. Proteogenomic characterization of molecular and cellular targets for treatment-resistant subtypes in locally advanced cervical cancers, Molecular Cancer. 2025, 24 (77), 1–27

Narrow-Window Data-Independent Acquisition with TMT for High-Throughput and Deep Proteomics study

Chaewon Kang[1,†], Jiwon Hong[1†], Hokeun Kim[1], JeongSu Jo[1], Jun-Hyeong Seo[2], Jeong-Won Lee[2] and Sang-Won Lee[1,*]

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Abstract

Data-independent acquisition (DIA) mass spectrometry has become a powerful strategy for comprehensive proteome analysis by systematically fragmenting all precursor ions within defined isolation windows across a selected m/z range. In contrast to data-dependent acquisition (DDA), which prioritizes high-intensity precursors, DIA expands proteome coverage by improving the detection and quantification of low-abundance peptides. However, its application to isobaric labeling has been limited, largely due to reporter-ion interference from co-isolated, co-eluting peptides, which compromises quantitative precision and accuracy.

Here, we introduce an ultra-narrow-window DIA workflow with 18-plex TMTpro labeling. Leveraging the Orbitrap Astral mass spectrometer operating at 200 Hz MS/MS scan speed and 80,000 resolution (m/z 200), we implemented 0.6 Th isolation windows that achieve near DDA-level.

Applied to ovarian cancer tissue, this workflow identified substantially more peptides and protein groups than DDA, while maintaining high quantification accuracy and precision. These advances allow deeper proteome coverage without sacrificing quantitative robustness, positioning this approach as a promising tool for multiplexed proteomics in clinical and large-scale population studies.

Symposia B

SYM-B5-2

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■ Education

2014.03~2020.02	Ph.D.	Ulsan National Institute of Science and Technology
2009.03~2014.02	B.S.	Ulsan National Institute of Science and Technology

■ Professional Experience

2020.03–	Postdoctoral associate	Seoul National University

■ Publications

- Kang MG†, Shin S†, Jang DG, Kwon O, Lee SY, Mishra PK, Jung M, Mun JY, Kee JM*, Kim JS*, Rhee HW* Intracristal space proteome mapping using super-resolution proximity labeling with isotope-coded probes. Nat Commun. 2025, 16, 7757 (†equally contributed)
- Kang MG†, Kim HR†, Lee HY†, Kwak C, Koh H, Kang BH, Roe JS*, Rhee HW*, Mitochondrial thermogenesis can trigger heat-shock response in the nucleus, ACS Cent. Sci. 2024, 10, 1231-1241 (†equally contributed)

Development of a Quantitative Proximity Labeling Method for Mapping the Intracristal Space Proteome

Myeong-Gyun Kang[1†], Sanghee Shin[2,3,4†], Dong-Gi Jang[2,5], Ohyeon Kwon[6], Song-Yi Lee[1,7,8], Pratyush Kumar Mishra[1], Minkyo Jung[9], Ji Young Mun[9], Jung-Min Kee[6*], Jong-Seo Kim[2,5*], Hyun-Woo Rhee[1,2*]

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Abstract

The intracristal space (ICS) is a distinct sub-domain of the mitochondrial intermembrane space (IMS) where oxidative phosphorylation occurs. This place is expected to control mitochondrial respiration in response to dynamic demands; however, the molecular components of the ICS have not been identified due to the lack of an effective purification method. Recently, proximity labeling has emerged as a powerful approach for identifying proteomes within various subcellular compartments. Here, we develop isotope-coded phenol probes for APEX labeling (ICAX), enabling the quantitative analysis of the spatial proteome at nanometer resolution between two distinctly localized APEX enzymes. From this novel proximity labeling strategy, we identified that TMEM177 is specifically localized to the ICS. Moreover, TMEM177 offers a unique ICS targeting modality of various fluorescent sensor proteins for measuring local pH, redox states, and temperature within the ICS. ICAX analysis further reveals unexpected dynamics of the mitochondrial spatiome under conditions of disrupted mitochondrial contact site and cristae organizing system and mitochondrial uncoupling, respectively. Overall, these results demonstrate the importance of ICS for mitochondrial quality control under dynamic stress conditions.

Symposia B

SYM-B5-3

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■ Education

2017.09-present	Ph.D.	Dept of Brain science, Daegu Gyeoungbuk Institute of Science & Technology (DGIST)
2011.03-2017.02	B.S.	Dept of Biotechnology, Yeungnam university

■ Professional Experience

2018.08-present	Student researcher	Korea Brain Research Institute (KBRI)
2017.09-present	MS/Ph.D. student	Daegu Gyeoungbuk Institute of Science & Technology (DGIST)
2017.02-2017.07	Intern researcher	Korea Brain Research Institute (KBRI)
2016.03-2016.12	Student researcher	Yeungnam university
2013.07-2013.07	Student internship	Korea Research Institute of Bioscience and Biotechnology (KRIBB)

■ Academic Societies

2024.10-present	Student	Korean Society for Biochemistry and Molecular Biology (KSBMB)
2022.06-present	Student	Society for Neuroscience (SFN)
2021.06-present	Student	Korean Society for Biochemistry and Molecular Biology (KSBMB)
2019.02-present	Student	The Korean Society for Brain and Neural Sciences (KSBNS)

■ Publications

- Park S*, Kim H*, Choi M, Lee J, Kim J, Kim K, Park H, Choe H†. Defective primary cilia in hypothalamic paraventricular nucleus oxytocin neurons leads to social amnesia. (in preparation)
- Kim H*, Shin S*, Han J, Yun K, Kim J-S†, Park H†. Serpina1e mediates the exercise-induced enhancement of hippocampal memory. (in revision)

Serpina1e Mediates the Exercise-induced Enhancement of Hippocampal Memory

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Abstract

The exercise-induced enhancement of learning and memory is thought to be regulated not only by improved cardiometabolic and psychosocial health but also by body–brain interactions mediated by metabolites and proteins derived from peripheral organs. Given the prominent role of skeletal muscle during exercise, muscle-derived factors are believed to mediate the cognitive enhancements induced by exercise. However, the specific myokines that exert beneficial effects on cognitive functions remain to be elucidated. Here, we reveal that a novel myokine, Serpina1e, acts as a molecular mediator that directly supports long-term memory formation in the hippocampus. Using an in vivo myokine-labeling mouse model, proteomic analysis revealed that the Serpina1 family of proteins are the myokines whose levels increased the most in plasma after 4 weeks of chronic aerobic exercise in male mice. Systemic delivery of recombinant Serpina1e or intramuscular overexpression of Serpina1e in sedentary male mice was sufficient to reproduce the enhancement of hippocampus-associated cognitive functions. Conversely, mice with a muscle-specific reduction in Serpina1e did not exhibit exercise-induced hippocampal memory enhancement, indicating a requirement of muscle-derived Serpina1e for exercise-induced cognitive enhancement. Moreover, our data showed that an increase in plasma Serpina1e level led to upregulated neurogenesis, brain-derived neurotrophic factor (BDNF), and neurite growth in the hippocampus, suggesting a positive role of exercise-induced Serpina1e in boosting hippocampal function. Because plasma Serpina1e can cross the blood–cerebrospinal fluid (CSF) and blood–brain barrier to reach the brain, our findings reveal that Serpina1e is a myokine that migrates to the brain and mediates exercise-induced memory enhancement by triggering neurotrophic growth signaling in the hippocampus. This discovery elucidates the molecular mechanisms underlying the beneficial effects of exercise on cognitive function and may have implications for the development of novel therapeutic interventions for alleviating cognitive disorders.

Symposia B

SYM-B5-4

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2018.03-2025.02	Ph.D.	Daegu Gyeongbuk Institute of Science and Technology (DGIST)
2012.03-2018.02	B.S.	Kyung Hee University

■ Professional Experience

2022.11.10	KSPB	Molecular genetic study on the long noncoding RNA-mediated regulatory network in leaf senescence

■ Publications

- Kang, M.H., Lee J., Kim, J., Hazara, B.M., Park, J., Jung, H.J., Kim, S., Lee, H., Yang, S.W., Kwak, J.M., Kim. M.S., Lee, J-C., & Lim, P. O. (2025). The chloroplast-targeted long noncoding RNA CHLORELLA mediates chloroplast functional transition across leaf ageing via anterograde signaling. Nat.Plants (Accepted)
- Lee, J., Kang, M.H., Choi, D. M., Marmagne, A., Park, J., Lee, H., Gwak, E., Lee, J. C., Kim, J. I., Masclaux-Daubresse, C., & Lim, P. O. (2024). Phytochrome-interacting factors PIF4 and PIF5 directly regulate autophagy during leaf senescence in Arabidopsis. J Exp Bot. https://doi.org/10.1093/jxb/erae469 (Co-First Author)
- Jung, S., Kim, H., Lee, J., Kang, M.H., Kim, J., Kim, J. K., Lim, P. O., & Nam, H. G. (2024). The genetically programmed rhythmic alteration of diurnal gene expression in the aged Arabidopsis leaves. Front Plant Sci, 15, 1481682. https://doi.org/10.3389/fpls.2024.1481682
- Kim, J. Y., Lee, J., Kang, M.H., Trang, T. T. M., Lee, J., Lee, H., Jeong, H., & Lim, P. O. (2022). Dynamic landscape of long noncoding RNAs during leaf aging in Arabidopsis [Original Research]. Frontiers in Plant Science, 13. https://doi.org/10.3389/fpls.2022.1068163 (Co-First Author)
- Lee, J., Kang, M.H., Kim, J. Y., & Lim, P. O. (2021). The Role of Light and Circadian Clock in Regulation of Leaf Senescence. Front Plant Sci, 12, 669170. https://doi.org/10.3389/fpls.2021.669170 (Co-First Author)

The Chloroplast-targeted Long Noncoding RNA CHLORELLA Mediates Chloroplast Functional Transition Across Leaf Ageing via Anterograde Signaling

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[6] These authors contributed equally: Myeong Hoon Kang, Juhyeon Lee, Jinkwang Kim.
[7] These authors jointly supervised this work: Min-Sik Kim, Jong-Chan Lee and Pyung Ok Lim.

Abstract

Leaf senescence is the last stage of the development of plants. During the developmental transition from mature to senescent leaves, chloroplast, which acts factory of metabolic intermediates that support plant growth, undergoes destruction to facilitate the relocation of nutrients to reproductive organs. However, how the timing of this transition is regulated through communication between chloroplast and the nucleus remains to be elucidated. Here, we report a novel regulatory mechanism underlying this transition. Through co-expression analysis and genetic screening, we identified *CHLOROPLAST-RELATED LONG NONCODING RNA (CHLORELLA)*, which is highly co-expressed with genes supporting chloroplast function during leaf lifespan. Lacking *CHLORELLA* transcripts accelerate leaf senescence as well as downregulating chloroplast genes. Moreover, we found that it translocates into chloroplasts and contributes to the accumulation of the plastid-encoded RNA polymerase (PEP) complex. As the expression of *CHLORELLA* decreases during aging, PEP accumulation is reduced, leading to reduction of transcription of photosynthesis-related genes, which may trigger leaf senescence. Our study unravels a long noncoding RNA-based anterograde signaling mechanism that facilitates timely leaf senescence.

Symposia B

SYM-B5-5

Ji Eun PARK

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■ Education

2023.03 – Present	Ph.D. candidate	Graduate School of Analytical and Science Technology Chungnam National University
2020.09 – 2023.02	M.S.	Graduate School of Analytical and Science Technology Chungnam National University
2014.03 – 2020.08	B.S.	Biochemistry, Yeungnam University

■ Professional Experience

2020 – Present	Graduate Researcher	Asia-Pacific Glycomics Reference Site, Graduate School of Analytical Science and Technology, Chungnam National University, Korea

■ Academic Societies

2024 – Present	The Korean Human Proteome Organization (KHUPO)
2023 – Present	American Society for Mass Spectrometry (ASMS)
2022 – Present	Korean Society for Mass Spectrometry (KSMS)
2021 – Present	The Korean Chemical Society (KCS)
2021 – Present	The Korean Society for Glycoscience (KGS)
2021 – Present	Korean Society for Biochemistry and Molecular Biology (KSBMB)

■ Publications

- Pham, D. T., Hoang, T. X., Park, J., Tieu, M. V., Tran, T. M., Park, S., ... & Cho, S. (2025). Novel Siglec-1/β-CD/rGO Impedimetric Biosensor for Detecting Neu5Ac Expression in Red Blood Cells. Biosensors and Bioelectronics, 117235.
- Lee, J., Park, J. E., Lee, D., Seo, N., & An, H. J. (2024). Advancements in protein glycosylation biomarkers for ovarian cancer through mass spectrometry-based approaches. Expert Review of Molecular Diagnostics, 24(4), 249-258.

Organ-Specific Diversity of Immunogenic Glycosylation in Pigs for Xenotransplantation

Ji Eun Park[1,2], Myung Jin Oh[1,2], Joohyun Shim[3], Kimyung Choi[3], and Hyun Joo An[1,2]

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Abstract

Xenotransplantation holds promise for addressing the organ shortage, with gene editing enabling genetically modified pigs for clinical use. However, immune rejection remains a challenge due to species-specific glycans like α-Gal, NeuGc, and Sda, which trigger strong antibody-mediated rejection in humans. While gene-editing effectively eliminates certain immunogenic glycans, the broader landscape of organ-specific glycosylation patterns and their immunogenic potential remains insufficiently understood. Given that glycosylation is a complex and dynamic posttranslational modification (PTM) influencing immune compatibility, a systematic characterization of glycan structures in genetically modified pigs is crucial. In this study, we utilized PGC-based LC-MS/MS to comprehensively map immunogenic glycans and delineate organ-specific glycosylation patterns in organs (kidney, heart, and pancreas) of wild and transgenic pigs. N-glycan compositions were identified based on accurate mass, retention times, and established glycan biosynthetic pathways. Immunogenic glycan structures were further characterized by CID-MS/MS. The relative abundance of each N-glycan was normalized against the total ion counts for quantitative comparison. The key factor in hyperacute rejection, α-Gal, was the predominant glycan in the kidney, constituting approximately 55% of the total glycan pool, whereas its abundance was more than twofold lower (~21%) in other organs. Conversely, NeuGc associated with acute and chronic rejection exhibited minimal expression in the kidney (~0.3%) but was more abundant in the heart (~5%) and pancreas (~14%). The Sda antigen, a blood group-related glycan structure, was absent across all tested organs. These immunogenic glycans were expressed both independently and concurrently, thereby significantly increasing structural diversity. A total of 85 glycans were detected in the kidney, including 36 immunogenic structures, exhibiting the highest structural diversity among the three organs. Although the heart showed the lowest diversity of immunogenic glycans, a unique heart-specific structure was observed, characterized by NeuAc linked to HexNAc rather than the typical galactose-linked structure. In knockout pig organs where terminal immunogenic glycans were removed, glycan composition shifted toward NeuAc-conjugated structures. However, the distribution of high-mannose, hybrid, and complex type glycans retained organ-specific characteristics. By elucidating the organ-specific diversity of immunogenic glycans in pig models, this study provides critical insights for xenotransplantation. Our findings contribute to refining gene-editing strategies and improving transplant compatibility, ultimately enhancing transplant safety.

KHUFO

2025 10.15.-10.17.

BUSAN BPEX

BON VOYAGE TO THE NEXT GENERATION PROTEOMICS

BON VOYAGE TO THE NEXT GENERATION PROTEOMICS

Corporate Workshop

KHUPO
The Korean Human Proteome Organization

Corporate Workshop ThermoFisher SCIENTIFIC

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■ Education

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Redefining Multi-Omics Research with the Next Generation Mass Spectrometer

■ Professional Experience

2025.01 –	LSMS /HRMS Technical Specialist	Thermo Fisher Scientific / Chromatography & Mass Spec Division
2024.03 – 2025.01	LSMS /HRMS Technical Specialist	Thermo Fisher Scientific / Chromatography & Mass Spec Division
2015.01 – 2024.03	LSMS Application Manager	Thermo Fisher Scientific / Chromatography & Mass Spec Division
2002 – 2015.01	Application & Marketing Manager	SCINCO CO. LTD.

Redefining Multi-Omics Research with the Next Generation Mass Spectrometer

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Abstract

At ASMS 2025, Thermo Fisher Scientific introduced two new Orbitrap platforms that significantly advance Multi-Omics research. The Orbitrap Astral Zoom delivers high-throughput DIA enabled by ultrafast scan speed, enhanced sensitivity for single-cell proteomics, and robust TMT 32-plex workflows for large-scale multiplexed analysis. The Orbitrap Excedion Pro integrates ETD, Hybrid-DIA, and eDR capabilities, enabling PTM studies with ETD, improved LFQ performance through Hybrid-DIA, and enhanced qualitative and quantitative metabolomics with superior S/N from eDR. Together, these innovations provide versatile opportunities to accelerate discovery across diverse biological applications.

Corporate Workshop ThermoFisher SCIENTIFIC

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■ Education

March 2009 – August 2011	Master's degree	Korea University
March 2003 – February 2005	Bachelor's degree	Konkuk University

■ Professional Experience

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March 2012 – June 2014	Researcher	KAHPA, Research center, Seohyeon-dong,Bundang, Gyeonggi

Advancing multiplex imaging for spatial proteomics research with EVOS S1000 Spatial Imaging System

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Thermo Fisher Scientific, Seoul, Republic of Korea

Abstract

Spatial proteomics, selected as the 2024 Method of the Year by Nature Methods journal, is an emerging scientific discipline that focuses on studying proteins within their spatial contexts, providing comprehensive insights into cell types and their interactions within the native tissue microenvironment. Protein tissue mapping is crucial for understanding processes such as metastasis, immune evasion, malignant cell growth, angiogenesis, and therapeutic drug resistance in immunology and oncology research. Spectral imaging, a key technology in spatial proteomics, allows capturing signals emitted by several fluorophores in a single imaging round, thus offering visualization of the tissue spatial diversity while preserving sample integrity. However, spectral imaging has the disadvantage of necessitating algorithmic processing, known as unmixing, in order to resolve overlapping fluorescent signals, especially when detecting more than six protein targets. Unmixing often lacks transparency and is limited to certain proprietary dyes, significantly reducing the choices available to researchers.

The Invitrogen EVOS S1000 Spatial Imaging System addresses these spectral imaging challenges by providing a simplified workflow that supports up to 9–plex imaging in a single round, with fast, integrated, automated and reliable unmixing. It is a flexible platform, that has compatibility with commonly available labelling methods and dyes, that can be easily selected by the user to configure acquisition protocols.

Corporate Workshop

ThermoFisher SCIENTIFIC

Seong cheol HONG

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■ Professional Experience

2018 – Present	Application Scientist	CMD, Thermofisher Scientific
2014 – 2018	Ph.D.	Korea Institute of Science and Technology (KIST)
2005 – 2012	B.S.-M.S.	Pusan National University

■ Research interests

1. Profiling of organic impurities in industrial field
2. HRMS based multi-omics analysis
3. High-throughput characterization of combinatorial chemistry

■ Publications

- Hong, S. C.; Murale, D. P.; Lee, M.; Lee, S. M.; Park, J. S.; Lee, J.-S* : Bulk aggregation based fluorescence turn-on sensors for selective detection of progesterone in aqueous solution. Angew. Chem. Int. Ed. 2017, 56, 14642-14647.
- Hong, S. C.; Murale, D. P.; Jang, S.-Y.;Haque, M. M.;Seo, M.; Lee, S.; Woo, D.; Kwon, J; Song, C.-S.; Kim, Y. K.; Lee, J.-S. *: Discrimination of Avian Influenza Virus using Host-cell Infection Fingerprinting by Sulfinate-based Fluorescence Superoxide Probe. Angew. Chem. Int. Ed.2018, 57, 9716-9721.
- Hong, S. C.*; Koh, H.; Kwon, H.; Hackhusch, S.; Morin, S.: Rabeprazole impurity identification using a trap heart-cut 2D-LC system coupled to Orbitrap-based HRAM-MS. Thermofisher Application note 002622.

Orbitrap Astral Zoom and Excedion Pro in Multi-Omics Research: Demonstrating Sensitivity and Throughput

Seong cheol HONG

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■ Abstract

Recent advances in mass spectrometry have expanded the scope of multi-omics research through the development of Thermo's latest platforms, the Orbitrap Astral Zoom and the Excedion Pro. The Orbitrap Astral Zoom combines ultra-fast scan speed with high sensitivity, while the Excedion Pro offers versatile collision modes to maximize analytical flexibility. These capabilities enable robust and comprehensive data acquisition from a single injection, thereby improving research productivity and enhancing the depth of biological insights. In this seminar, we present representative case studies demonstrating how these instruments can be applied in multi-omics research.

Corporate Workshop SCIEX

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Education

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Professional Experience

2022–2025	Proteomics Technical Marketing Manager	SCIEX
2013–2022	Applications Support Manager	SCIEX
2005–2013	Applications Support Scientist	SCIEX
2003–2005	Postdoctoral Fellow	University of Maryland (USA) in the laboratory of Catherine Fenselau

Redefining sensitivity in accurate mass spectrometry with the SCIEX ZenoTOF 8600 system

Patrick Pribil

Abstract

The ZenoTOF 8600 system represents a seismic shift from what was achievable using previous generation systems, with a profound improvement to the system sensitivity equal to or better than industry–leading triple quadrupole platforms. The ZenoTOF 8600 system combines state–of–the–art ion generation, ion transfer technology, complementary MS/MS fragmentation techniques, robustness improvements, and photomultiplier–based ion detection, enabling this platform to offer the sensitivity, speed, and versatility needed for the highest levels of qualitative and quantitative analysis. This presentation will focus on proteomics applications using the ZenoTOF 8600 system, including both data–dependent and data–inde–pendent methods for the in–depth detection, identification, characterization, and quantitation of peptides and proteins for biological research.

Corporate Workshop Agilent

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■ Education

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■ Professional Experience

2020.03-Current	LC/MS Product Specialist	Agilent Technologies Korea, South Korea
2013.05-2020.03	Field Application Chemist	Bruker Daltonics, Bruker Korea, South Korea

Unlocking the Power of Automated Sample Preparation: Seamless Quantitation and Identification with LC/TQ and LC/Q-TOF

Jin Nyoung CHOI

■ Abstract

This presentation showcases automated LC/MS workflows for seamless quantitation and identification using LC/TQ and LC/Q–TOF systems. The first workflow integrates metabolomics, lipidomics, and proteomics in plasma and cell samples, enabling high–throughput analysis with attomole sensitivity and reduced sample prep variability (~50%). Over 500 metabolites, 763 lipids, and 375 peptides are quantified with high reproducibility. The second workflow demonstrates automated phosphopeptide enrichment and identification in the HUPO Phosphopeptide Challenge, achieving 〉92% selectivity and recovering 93 of 94 spiked phosphopeptides with the highest recovery among participating labs. Together, these automated solutions highlight robust performance and versatility for biomarker discovery and precision medicine applications.

Corporate Workshop illumina

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Education

March 2010	Master of Science Biochemistry and Bioinformatics	Georgetown University, Washington DC
April 2021	Master of Business Administration	HAAS School of Business, Univ of California, Berkley, California, USA

Professional Experience

June 2022 – Present	Associate Director Product Management, Proteomics	Illumina, San Diego, USA
June 2020 – June 2022	Associate Director Product Management, Proteomics Software	Quantum Si, San Diego, USA
August 2017 – June 2022	Associate Director Product Management, Clinical Informatics	QIAGEN, Redwood City, USA
Jan 2013 – August 2017	Bioinformatics Scientist	DxTerity, Los Angeles, USA

Publications

- Mazumder R, Morampudi KS, Motwani M, Vasudevan S, Goldman R. Proteome-wide analysis of single-nucleotide variations in the N-glycosylation sequon of human genes. PLoS One. 2012;7(5):e36212. doi: 10.1371/journal.pone.0036212. Epub 2012 May 7. PMID:
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  A Novel Technology for Multiplex Gene Expression Analysis Directly from Whole Blood Samples Stabilized at Ambient Temperature Using an RNA-Stabilizing Buffer Chang Hee Kim · Majid Abedi · Yenbou Liu · Sree Panuganti · Francisco Flores · Kevin R. Shah · Hannah Catterall · Krishna S. Morampudi · Robert Terbrueggen The Journal of Molecular Diagnostics, Volume 17, Issue 2, 118 – 127

Expanded Content & Improved Performance of Illumina Protein Prep

Andrew Slatter[2]; Mike Dorwart[1]; Mike Mehan[1]; Simon Freedman[1]; Izabel Cavassim Alves Ines[1]; Nithya Subramanian[2]; Dave Jones[2], Yang Cao[2], Carlo Randise-Hinchliff[1], Krishna Morampudi[1], Fiona Kaper[1]

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Abstract

The Illumina Protein Prep™ (IPP) is a comprehensive high-throughput proteomics solution combining Illumina's next-generation sequencing with Standard BioTool's SOMAmer™ (Slow Off-rate Modified Aptamers) technology. SOMAmers are modified single-strand DNAs that bind to proteins with high affinity. Here we present a newly updated version of IPP with an expanded menu of 9,580 unique human proteins and improved overall performance. 2.5 Days Sample-To-Results, ~4 Hrs Hands-on Time

Matrix	Median Intra-Run CV	Median Inter-Run CV	Median Total-Run CV	90th %ile Total-Run CV	Median Pop. Counts > LoD*
Plasma	5.1%	3.1%	5.9%	10%	94%
Serum	4.8%	2.9%	5.6%	9.7%	97%

Corporate Workshop 

Ph.D. Unbeom Kang

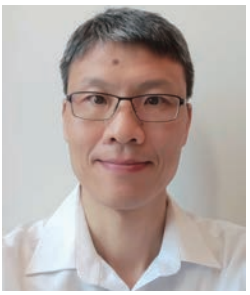
Affiliation BERTIS

- Title

  Deep Profiling for Translational Insights in Clinical Proteomics

MEMO.

Corporate Workshop



Chuan-Wei (David) Jang

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■ Education

2003.08 – 2009.08	Ph.D.	Program in Developmental Biology Baylor College of Medicine, Houston, TX, USA
1999.09 – 2001.06	M.S.	Institute of Molecular Medicine, College of Medicine National Taiwan University, Taipei, Taiwan
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■ Professional Experience

2025.03 –	Sr. Field Application Scientist	Syncell Inc.
2024.02 – 2025.02	Sr. Scientist	TFBS Bioscience Inc.
2022.12 – 2024.02	Principal Scientist/Project Leader	Mycenax Biotech Inc.

■ Publications

- Chen YL, Yen YC, Jang CW, Wang SH, Huang HT, Chen CH, Hsiao JR, Chang JY, Chen YW. Ephrin A4-ephrin receptor A10 signaling promotes cell migration and spheroid formation by upregulating NANOG expression in oral squamous cell carcinoma cells. Sci Rep. 2021 Jan 12;11(1):644.
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Microscoop_Webinar

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Abstract

Understanding the spatial distribution of proteins within cells and tissues is key to revealing their roles in health and disease. Conventional spatial proteomics approaches are often constrained by complex genetic manipulations or insufficient resolution to study subcellular features. We present Microscoop®, a microscopy-guided proteomics platform that enables high-resolution proteomic analysis across diverse biological samples. The system uses a photoactivatable probe (Synlight-Rich™) and automated image-guided two-photon laser illumination to achieve biotinylation of all proteins within user-defined regions of interest. Labeled proteins are then enriched (Synpull™) and identified by mass spectrometry analysis, providing proteomic insights with submicron precision. This optoproteomics approach offers new opportunities to map molecular interactions and uncover previously unrecognized proteins in complex biological systems.

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BUSAN BPEX

BON VOYAGE TO THE NEXT GENERATION PROTEOMICS

Educational Session

KHUPO
The Korean Human Proteome Organization

Educational Session 1-1

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2012.03–2019.02	Ph.D.	Graduate School of Analytical Science and Technology, Chungnam National University

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2021.01–	Head of Bio R&D Center	CellKey AI Co., Ltd.
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■ Publications

- Ji, E. S., Lee, B. S., Mun, J., Jeon, S. Y., Hong, H. R., Kim, H. J., ... & Kim, K. H. (2025). Quantification of salmon nasal cartilage extracts using liquid chromatography–tandem mass spectrometry. Food Chemistry, 143280.
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Introduction to Proteomics : Mass Spectrometry and Bioinformatics

Kwang Hoe Kim

Bio R&D Center, CellKey AI Co., Ltd., Seoul, 06571, Republic of Korea

Abstract

Mass spectrometry–based proteomics has emerged as a powerful approach for exploring the complexity of proteins, post–translational modifications, and their biological functions. This session, Introduction to Proteomics: Mass Spectrometry and Bioinformatics, will provide an overview of how proteomics leverages advanced MS technologies for large–scale protein identification, quantification, and the characterization of modifications such as glycosylation. Applications will be discussed across diverse fields, including biomarker discovery, systems biology, and translational research for clinical diagnostics. In addition, the session will highlight key challenges such as the complexity of protein regulation and the interpretation of high–dimensional datasets. The importance of bioinformatics will be briefly introduced, emphasizing its role in enabling efficient, accurate, and scalable analysis of MS data. By outlining recent developments and integrated approaches, the lecture aims to give participants both a conceptual foundation and a forward–looking perspective on how proteomics and bioinformatics together can drive innovations in biomedical research.

Educational Session 1-2

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■ Education

2001.09-2004.02	M.S.	Korea University

■ Professional Experience

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2018.07-2023.12	General Manager	SCIEX Korea
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2011.07-2015.08	Manager	FAS MD Team, SCIEX Korea
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■ Publications

- Shin, B., Jung, H., Hyung, S., Kim, H., Lee, D., Lee, C., Yu, M., Lee, S. "Postexperiment Monoisotopic Mass Filtering and Refinement (PE-MMR) of Tandem Mass Spectrometric Data Increases Accuracy of Peptide Identification in LC/MS/MS" Molecular & Cellular Proteomics, 2008, 7, 1124-1134

Exploring the Human Proteome through Affinity-Based Proteomics

Byung-Hee Shin

Olink, part of Thermo Fisher Scientific

Abstract

The human proteome represents a dynamic and highly complex network of molecular interactions that underpins both physiological processes and disease mechanisms. Mass spectrometry has long been recognized as the most powerful system for analyzing diverse proteoforms, including post-translational modifications and sequence variants, thereby providing unmatched depth in proteome profiling. Nevertheless, despite its remarkable strengths, mass spectrometry continues to face significant challenges when applied to biofluids such as plasma and serum, where proteins are present across an extremely wide dynamic range. This limitation has motivated the exploration of complementary approaches that can overcome dynamic range barriers and enable large-scale studies.

One of the most impactful solutions has been the refinement of traditional affinity-based protein detection and quantification methods. By improving assay robustness, increasing multiplexing capacity, and reducing sample requirements, these platforms now allow thousands of proteins to be measured across hundreds or even thousands of samples within a short timeframe. Importantly, such developments have transformed proteomics from small-scale, discovery-oriented projects into population-scale investigations, where meaningful biological insights can be derived from microliter volumes of plasma or serum. These innovations have also accelerated the integration of proteomic datasets with genomics, thereby providing a multidimensional view of human biology that connects genetic variation, protein expression, and clinical outcomes.

As a result, affinity-based proteomics has rapidly emerged as a powerful complement to mass spectrometry, not only by expanding the measurable space of the proteome but also by enabling scalable studies that are essential for biomarker discovery, disease stratification, and precision medicine. In this session, we will present and discuss some of the most important global research achievements that highlight the combined potential of mass spectrometry–based and affinity-based proteomics technologies in advancing our understanding of the human proteome and its role in health and disease.

Educational Session 2

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■ Education

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2011.08–2013.06	M.S.	Sungkyunkwan University (SAIHST)
2005.09–2009.06	B.S.	University of California Santa Barbara

■ Professional Experience

2023.09–Present	Associate Professor	Korea University College of Medicine
2019.09–2023.08	Assistant Professor	Korea University College of Medicine
2017.01–2019.08	Senior Scientist	Samsung Medical Center

■ Academic Society

2025.03–Present	Editorial Board	Genome Biology

■ Publications

- Lee JY, Lee EJ, Seo BY, Kim J, Song Y, Lee D, Moon N, Koo H, Park CK, Kim MS, Park SI, Nam DH, Kong DS, Sa JK* (2025) Single-cell analysis reveals a longitudinal trajectory of meningioma evolution and heterogeneity, Nature Communications, 16(1):5481
- Kim KH, Migliozzi S, Koo H, Hong JH, Park SM, Kim S, Kwon HJ, Ha S, Garofano L, Oh YT, D'Angelo F, Kim CI, Kim S, Lee JY, Kim J, Hong J, Jang EH, Mathon B, Di Stefano AL, Bielle F, Laurenge A, Nesvizhskii AI, Hur EM, Yin J, Shi B, Kim Y, Moon KS, Kwon JT, Lee SH, Lee SH, Gwak HS, Lassorella A, Yoo H, Sanson M*, Sa JK*, Park CK*, Nam DH*, Iavarone A*, Park JB* (2024) Integrated proteogenomic characterization of glioblastoma evolution, Cancer Cell, 42(3):358-377
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Integrative Proteogenomic Analysis for Revealing Biological Insight and Mechanisms

Kyung-Hee Kim[1], Harim Koo[1], Jun-Hee Hong[1], Seung Min Park[1], Young Taek Oh[2], Jason K. Sa[3], Chul-Kee Park[4], Do-Hyun Nam[5], Jong Bae Park[1]

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Abstract

Integrative proteogenomic analysis has emerged as a profound approach for advancing precision oncology. While genomic profiling has significantly deepened our understanding of cancer biology, it often falls short in capturing the functional consequences of genomic alterations. Proteogenomic analysis bridges this gap by linking genetic variants to protein-level changes, thereby providing a more comprehensive and actionable view of tumor biology. Here, we perform an integrative proteogenomic analysis of 123 longitudinal glioblastoma pairs and identify a highly proliferative cellular state at diagnosis and replacement by activation of neuronal transition and synaptogenic pathways in recurrent tumors. Proteomic and phosphoproteomic analyses reveal that the molecular transition to neuronal state at recurrence is marked by post-translational activation of the wingless-related integration site (WNT)/ planar cell polarity (PCP) signaling pathway and BRAF protein kinase. Inhibition of B-raf proto-oncogene (BRAF) kinase impairs both neuronal transition and migration capability of recurrent tumor cells, phenotypic hallmarks of post-therapy progression. Combinatorial treatment of temozolomide (TMZ) with BRAF inhibitor, vemurafenib, significantly extends the survival of PDX models. This study provides comprehensive insights into the biological mechanisms of glioblastoma evolution and treatment resistance, highlighting promising therapeutic strategies for clinical intervention.

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Satellite Session

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■ Professional Experience

2013.09-2019.08	Assistant prof.	College of medicine, Kyung Hee University/Kyung Hee Institute of Regenerative Medicine (KIRM), Kyung Hee University Hospital
2012.09-2013.08	Post Doc.	Kyung Hee Institute of Regenerative Medicine, Kyung Hee University
2007.04-2009.3	Team manager	Drug development. Biosolutions. Co. Ltd
2003.04-2007.03	Researcher	Korea Institute of radiological and medical science (KIRAMS)

■ Publications

- Gabee Park, Dae Yeon Hwang, Do Young Kim, Ji Young Han, Euiseon Lee, Hwakyung Hwang, Jeong Seop Park, Dae Wook Kim, Seonmin Hong, Sung Vin Yim1, Hyun Sook Hong 2*, Youngsook Son*. Identification of
- CD141+vasculogenic precursor cells from human bone marrow and their endothelial engagement in the arteriogenesis by co-transplantation with mesenchymal stem cells. Stem cell research & therapy (2025)
- Jeong Seop Park, Do Young Kim, Hyun Sook Hong, FGF2/HGF priming facilitates adipose-derived stem cell-mediated bone formation in osteoporotic defects, Heliyon (2024)
- Doyoung Kim†, Jiyuan Piao†, Jeong Seop Park, Dahyun Lee, Dae Yeon Hwang, Hyun Sook Hong* Substance P-mediated vascular protection ameliorates bone loss. Oxidative medicine and cellular longevity (2023)
- Jeong Seop Park, Doyoung Kim, Hyun Sook Hong*. Priming with a combination of FGF2 and HGF restores the impaired osteogenic differentiation of adipose-derived Stem cells. Cells (2022)
- Dahyeon Lee, Jeong Seop Park, Doyoung Kim, Hyun Sook Hong*. Substance P hinders Bile Acid-induced hepatocellular injury by modulating oxidative stress and inflammation. Antioxidants (2022)
- Jiyuan Piao, Jeong Seop Park, Dae Yeon Hwang, Hyun Sook Hong*, Youngsook Son* Substance P blocks β-aminopropionitrile-induced aortic injury through modulation of M2 monocyte-skewed monocytopoiesis. Translational research (2020)
- Sang Min Baek†, Kiyoung Kim†, Suna Kim, Youngsook Son, Hyun Sook Hong*, Seung-Young Yu*. SP prevents T2DM complications by immunomodulation. Scientific reports (2020)

Aging-Associated Changes in Stem Cell Function and Their Implications for Regenerative Medicine

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Abstract

Stem cells are actively explored as novel therapeutic modalities for critical diseases, cosmetic applications, and even alternative meat production. Adipose tissue represents a rich and easily accessible source of stem cells.

Adult stem cells derived from adipose tissue (ADSCs) can be expanded ex vivo and exhibit characteristics comparable to those of bone marrow-derived stem cells. However, the quality and functionality of ADSCs can be influenced by donor-related factors such as age, underlying disease, and lifestyle. The present study aimed to investigate the relationship between donor age or age-related disease and ADSC activity.

Our results demonstrated that aging and age-associated diseases significantly reduced ADSC activity and prolonged their doubling time without inducing profound morphological changes. Moreover, the paracrine functions of ADSCs were markedly altered by aging or disease, and their differentiation potential toward osteogenic or adipogenic lineages was likewise compromised.

Taken together, these findings indicate that donor conditions are critical determinants of ADSC quality and function, and should be carefully considered in divese applications.

Satellite Session SIMPLE planet.

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■ Education

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2011-2013	MBA	Kon-kuk University
1997-2001	BS	Kon-kuk University

■ Professional Experience

2016-present	Head of research center	Small-animal vet nutrition laboratory, JeilFeed.Co.Ltd
2014-2015	General Manager	Forza10 Korea
2011~2013	Director	Korea Pet Production Manufacture Association; KPPMA
2005-2013	S&C director	Royal-Canin Korea

■ Academic Society

2024-present	Director	Korea Society of Veterinary Clinical Dermatology (KSVCD)
2012-present	Director	Korea Society of veterinary Nutrition
2009-present	Director	Korea Society of feline medicine (KSFM)

■ Publications

- Woo Jae Cho3 2022 Effects of Synbiotic Preparation Containing Lactobacillus gasseri BNR17 on Body Fat in Obese Dogs: A Pilot Study. Animals (Basal) 2022 Mar 3;12(5):642.
- Woo Jae Cho2 2021 Anti-obesity effects of Celastrus orbiculatus extract containing celastrol on canine adipocytes. Can J Vet Res 2021 Jul 85(3) 177-185
- Woo Jae Cho1 2019 The effectiveness of wavy kibble with propolis coating dental therapeutic diet in beagle dog. KALAS PS-E-22
- Woo Jae Cho 2013 Feline clinical nutrition. Journal of Veterinary Clinics 61-61
- Cat Nutrition, Donguranmi, 2021.04.22. Writer Page 256, ISBN 9791196688356
- 반려동물영양학, Life science. 2024.03.01. Writer 조우재 236쪽, ISBN 9788961544467

Veterinary Clinical Evaluation of Prescription Diets Utilizing Insect Protein for Companion Animals and Proposals for Future Novel Protein Sources

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Abstract

In recent years, insect protein has garnered significant attention as a promising alternative to conventional animal-derived proteins. Within the pet food industry, insect protein has been introduced as a novel protein source, particularly for animals suffering from food allergies. Its clinical efficacy has been demonstrated, leading to the development of therapeutic diets. Amino acid profiling of various insect species reveals a diverse composition, and research in this area is actively expanding. In terms of essential amino acid fulfillment, insect protein has shown superior scores compared to fish and poultry proteins. For dogs and cats, amino acid adequacy is assessed using the Dog Amino Acid Score (Dog AA) and Cat Amino Acid Score (Cat AA), respectively. Certain insect proteins exhibit exceptionally high Cat AA values, indicating their suitability for feline nutrition.

Despite these advantages, the production of insect protein faces several challenges, and it has yet to achieve widespread commercialization as a pet food ingredient due to limitations in scalability. Cultured meat presents a compelling alternative to traditional animal proteins. Similar to insects, cultured meat can be engineered to meet the specific amino acid requirements of dogs and cats. If scalable production is achieved, the application of cultured meat in pet food could offer substantial nutritional value.

Developing cultured meat tailored to the amino acid profiles of companion animals enables the formulation of single-protein diets, thereby facilitating the creation of high-value functional pet foods. Moreover, the use of certain protein sources in pet food is increasingly restricted to avoid competition with human consumption. Cultured meat designed specifically for dogs and cats has the potential to become a key driver in the expanding pet food market, offering numerous benefits in veterinary medicine, including the management of food allergies, atopic conditions, and the development of single-protein therapeutic products.

Keyword: pet food R&D, real in vivo, insect-proten

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Satellite Session SIMPLE planet.

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Education

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Professional Experience

2008 – Present	Product Manager	Roche Diagnostics Korea

Biography

Anne Lee joined Roche Diagnostics Korea in 2008 as an application specialist and has been the product manager of Custom Biotech since 2012. She graduated from Kyunghee University in 2012 and obtained a master's degree in biochemistry from Ulsan University, College of Medicine. She is responsible for applications and marketing at Roche CustomBiotech, which focuses on the supply of raw materials to diagnostic manufacturers and biopharma reagents and instruments to biopharmaceutical companies.

Optimizing Bioprocess with Cedex Bio : A Core Strategy for Yield and Efficiency

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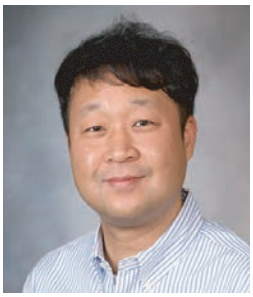
■ Abstract

Maintaining optimal cell culture conditions throughout the cell culture and fermentation process is vital to producing high yields of top-quality bioprocess products. Accurate monitoring of complete culture physiology inside bioreactors is simple and fast with Cedex® Analyzers. This instruments performs precise measurements and can detect subtle changes in nutrients, metabolites, or cell morphology and growth, enabling informed decisions and fast time response.

Leveraging this accurate data minimizes media waste and reduces unnecessary media changes. Furthermore, the acquisition of reliable data accelerates R&D cycles, ultimately contributing to a reduction in production costs and an enhancement of competitive advantage. This methodology represents a core strategy for the sustainable commercialization of bioprocess products.

Satellite Session SIMPLE planet.



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2022.01-Present	CTO	Simple Planet
2019.04-2021.07	Assistant Professor	Mayo Clinic College of Medicine and Science
2016.05-2021.07	Principal Research Technologist	Mayo Clinic
2013.01-2016.04	Post Doctoral Fellow	Johns Hopkins University
2012.03-2012.12	Post Doctoral Fellow	Kyung Hee University

■ Academic Society

2007.02-2019.01	Member	American Association for Cancer Research
2015.03-2016.02	Journal Reviewer	Cancer Translational Medicine
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2006.04-2019.01	Member	The Korean Society for Applied Biological Chemistry
2006.04-2019.01	Member	The Korean Society of Medical Crop Science

■ Professional Experience

- Yoo KH, Tang JJ, Rashid MA, Cho CH, Corujo-Ramirez A, Choi J, Bae MG, Brogren D, Hawse JR, Hou X, Weroha SJ, Oliveros A, Kirkeby LA, Baur JA, Jang MH. Nicotinamide mononucleotide prevents cisplatin-induced cognitive impairments. Cancer Res. 2021 Jul 1; 81(13):3727-3737 PMID: 33771896 DOI: 10.1158/0008-5472.CAN-20-3290
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Trends in Green Biotechnology : Focusing on Cell-based Foods as Future Foods

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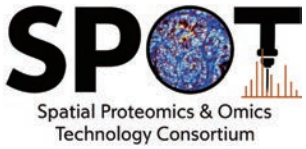
R&D Center, Simple Planet Inc., Seoul, Republic of Korea

Abstract

Food insecurity and the global protein crisis have become pressing challenges, driven by complex factors such as climate change, pandemics, armed conflicts, and economic instability. These disruptions compromise food supply chains and limit production capacity, making it difficult to meet the nutritional demands of a growing global population. Cell-cultured foods have emerged as a promising alternative, representing a novel, science-driven technology for sustainable protein production within the broader field of alternative foods. The sector is expanding rapidly, with multiple market analyses projecting annual growth rates exceeding 10%, and numerous companies now entering the commercial sector with the aim of providing cell-derived products. This technology is fundamentally science-driven, requiring advanced cell culture methods similar to those applied in cell therapeutics. Consequently, regulatory approval is essential, and an increasing number of start-ups are actively submitting applications and obtaining clearances from relevant authorities. Since the first approval of chicken-based cell-cultured meat by GOOD Meat in Singapore in 2020, regulatory progress has continued, exemplified by Believer Meats' FDA approval in 2025 for chicken-derived fibroblastic cells. In this presentation, we provide an overview of the current market trends in cell-cultured foods, focusing on global technological advancements, current regulatory frameworks, and the evolving landscape of companies applying for and obtaining approval worldwide

Satellite Session



Satellite Session

2025년 10월 15일 (수) 10:00–13:00 / BPEX 이벤트홀A

- 주 최 SPOT 공간단백체 기술개발사업단
- 후 원 ㈜ 다온비에스, Waters Korea, Bruker Korea, MeteoBiotech, ThermoScientific Korea
- 문 의 김경곤 / kkkon1@amc.seoul.kr

시 간	내용	연 자
10:00–10:10	개회사	김경곤 / 울산의대
10:10–10:30	Why and How we try spatial omics approach	김경곤 / 울산의대
10:30–10:50	Discover the proteome with cellular and subcellular spatial resolution	David Jang / Syncell Inc.
10:50–11:10	DESI MS를 이용한 공간단백체 연구 동향	김현우 / Waters Korea
11:10–11:30	Coffee break / sandwich	
11:30–11:50	Spatial lipidomicsand proteomics using TIMS Flex	강신권 / Bruker Korea
11:50–12:10	SLACS 기술을 이용한 공간오믹스 연구	장해욱 CTO / MeteoBiotech
12:10–12:30	Spatial Omics platform for translational research	김경미 / ThermoScientific Korea
12:30–12:35	폐회사	김경곤 / 울산의대

Satellite Session SCIEX

Doyoung CHOI

Position	Application Specialist
Affiliation	SCIEX Korea
Domain	Proteomics, Metabolomics, Lipidomics, Marker quantification, Biopharma Characterization

Education

2015. 7–2020. 5	Researcher	CJ Healthcare
2011. 3–2014. 9	Ph.D.	Kyung Hee University
2009. 3–2011. 2	Master	Konkuk University
2002. 3–2009. 2	Bachelor	Konkuk University

Abstract

The ZenoTOF 8600 is a powerful and easy–to–use mass spectrometer that helps researchers get fast and accurate results. It supports a wide range of applications, making it a valuable tool for scientific discovery and innovation. In today's presentation, we highlight its performance across several key areas, including pharmaceutical and biopharmaceutical development, advanced quantification workflows, lipidomics, metabolomics, and proteomics.

With its strong and reliable design, user–friendly interface, and flexible features, the ZenoTOF 8600 is a great fit for laboratories that need dependable and future–ready solutions. Whether used for routine testing or complex research, it delivers consistent results that help scientists work more efficiently and make confident decisions.

Satellite Session SCIEX

Kyunggon KIM

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■ Education

2000	BS	Yonsei University, Korea
2006	MS	Seoul National University, Korea
2010	Ph.D	Seoul National University, Korea
2013	Senior Researcher	Institute of Medical Research, SNU
2015	Post doctoral Researcher	Northwestern University, IL, USA
2015~	Associated professor	Asan Medical Center, Korea
2023~	Associated professor	University of Ulsan, Korea

■ Academic Society

2025~Present	Secretary of General, Korean Human Proteome Organization (K-HUPO)
2022~2023	Secretary General, 2023 HUPO Busan World Congress
2022~Present	Editor of Journal of Analytical Science & Technology
2024~Present	Director of Academic board of Korean Society of Mass Spectrometry (KSMS)
2023~Present	Director of ProteoXplore unit in AMC Sciences
2022~Present	President of Young Scientist Forum in Mass spectrometry
2024~Present	Boarder member in International Committee in Korean Cancer Association

■ Publications

- Generating Detailed Spectral Libraries for Canine Proteomes Obtained from Serum and Urine, Scientific Data, 2023 (Corresponding, IF:8.5)
- Magnetic transferrin nanoparticles (MTNs) assay as a novel isolation approach for exosomal biomarkers in neurological diseases, Biomaterial Research, 2023 (Corresponding, IF:11.3)
- Advanced 3D dynamic culture system with transforming growth factor-β3 enhances production of potent extracellular vesicles with modified protein cargoes via upregulation of TGF-β signaling, Journal of Advanced Research, 2023 (Corresponding, IF:12.88)

Satellite Session SCIEX

Hyun Joo AN

CV

Hyun Joo An is a Professor at the Graduate School of Analytical Science and Technology (GRAST), Chungnam National University, South Korea, and Director of the Asia-Pacific Glycomics Reference Site (AGRS). She earned her Ph.D. in Chemistry from the University of California, Davis, where she specialized in mass spectrometry-based glycomics. Professor An is internationally recognized for her pioneering contributions to glycomics, glycoproteomics, and glycolipidomics, with a strong focus on developing advanced analytical platforms for biopharmaceutical quality assessment and brain research. She has led numerous international collaborations with academic, clinical, and industrial partners, and her group has published widely in leading journals including Nature Communications and PNAS. Currently serving as Vice President of the Korean Human Proteome Organization (KHUPO) and as a Diversity Representative on the HUPO Council, Professor An continues to advance innovation at the interface of technology development and biomedical application.

Bridging Innovation and Application: Combined CID–EAD LC-MS/MS for Site-Specific Glycosylation in Advanced Biotherapeutics

Abstract

Accurate site-specific glycosylation analysis is essential for assessing immunogenicity risk in advanced biotherapeutics and xenotransplantation products. Traditional fragmentation methods often struggle to balance glycan structural information with precise localization at the peptide backbone. Here, we present a combined CID–EAD LC–MS/MS strategy that integrates complementary fragmentation modes to achieve both comprehensive glycan characterization and accurate site-specific assignment. CID provides glycan-derived fragment ions, while EAD preserves intact glycans and yields extensive c- and z-type peptide fragments, enabling confident site localization. By fine-tuning kinetic energy, diagnostic ions unique to EAD spectra were observed, allowing reliable detection of immunogenic glycans and differentiation of isomeric glycoforms. This approach was applied to cetuximab and porcine tissues, revealing site-specific immunogenic epitopes, with α-Gal completely absent in α1,3-galactosyltransferase knockout pigs. Together, these results demonstrate how combining CID and EAD enhances the precision of glycopeptide analysis and provides a powerful analytical framework for immunogenicity assessment in advanced biotherapeutics.

Satellite Session

Robin PARK

Position	CEO
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Education

May 2004	Master of Science	San Diego State University
Jan 2000	Bachelor of Science	University of Tennessee
Mar 1995	Bachelor of Science	Pusan National University

Professional Experience

Current	member	HUPO
Current	member	US HUPO
Current	member	ASMS
NA	Ad hoc reviewer	Nature methods
NA	Ad hoc reviewer	Journal of Proteome Research
NA	Ad hoc reviewer	American Chemical Society (ACS)
NA	Ad hoc reviewer	Analytical Chemistry
NA	Ad hoc reviewer	Bioinformatics

Academic Society

Feb 2024-current	CEO	Chaparral Labs
Aug 2020-current	Professional Scientific Collaborator	Scripps Research
Aug 2020-Feb2024	Director	Bruker Scientific LLC
Nov 2008-Aug 2020	CEO	Integrated Proteomics Applications
Nov 2004-Aug 2020	Director of Bioinformatics	Scripps Research
Mar 2003-Oct 2004	Bioinformatician	University of California, San Diego
May 2002-Mar 2003	Bioinformatician	ActivX BioScience, San Diego

Chaparral Bioinformatics Solutions: Accelerating Proteomics with Scalable, High-Performance Tools

Robin Park, Chaparral Labs

Department of Herbal Crop Research, National Institute of Horticultural and Herbal Science,
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Abstract

Modern proteomics has made data analysis more complicated and time-consuming due to increasing complexity and large-scale datasets, requiring scalable and efficient computational tools. We have developed core bioinformatics tools—SageDDA, SageDIA, and SagePRM—designed to meet the evolving needs of data-dependent, data-independent, and targeted proteomics analysis. These tools are accessible via standalone binaries, cloud-based SaaS, and local high-performance computing (HPC) environments, making them flexible to access or integrate into existing platforms. The Chaparral API further simplifies automation and integration into users' existing computational workflows.

SageDDA, built as a complete rewrite of the open-source Sage, is a high-performance, memory-efficient search engine designed for large search-space proteomics, including multiple PTMs, HLA, metaproteomics, and microbiome studies. It features XGBoost-based FDR estimation and robust global FDR control for confident peptide and protein identification.

SageDIA adopts a hybrid approach that combines spectrum-centric and peptide-centric strategies, leveraging their complementary strengths to handle large search spaces. It identifies peptides from both perspectives and applies a rescoring step to improve accuracy and confidence in results.

SagePRM is designed for high-throughput targeted proteomics, making it easy to analyze PRM data quickly and accurately. In a test run with 427 Steller files and 166 targets (data provided by the Jennifer Van Eyk Lab at Cedars-Sinai Medical Center), it completed the entire analysis in about 3 minutes on an AMD Ryzen 9 7950X 16-Core Processor. It's a fast and reliable tool built for large-scale clinical and single cell proteomics applications.

Together, these tools empower researchers to perform comprehensive proteomics analyses with speed, flexibility, and reproducibility. SageSuite represents a major step forward in accelerating scientific discovery through scalable and high-performance bioinformatics.

Satellite Session

Shinyeong JU

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■ Education

2004.02–2013.02	B.S. in Life Science	Hanyang University, Seoul, Republic of Korea
2013.08–2020.08	Integrated Ph. D. in Life Science	Hanyang University, Seoul, Republic of Korea

■ Academic Society

2020.09–present	Postdoctoral Researcher	Korea Institute of Science and Technology, Seoul, Republic of Korea

■ Publications

- Nawale, L.*, Ju, S.*, Kim, J. G., Soung, N. K., Kim, B. Y., Lee, C. & Cha-Molstad, H. ATE1 promotes breast cancer progression via arginylation-dependent regulation of MAPK-MYC signaling. Cell Commun. Signal. 23, 390 (2025). (*Co-first Author)
- Ju, S., Cha-Molstad, H. & Lee, C. Tracing the mark of arginine. Nat. Chem. Biol. 1–2 (2025). (Commissioned Article)
- Ju, S.*, Nawale, L.*, Lee, S.*, Kim, J. G., Lee, H., Park, N., Kim, D. H., Cha-Molstad, H. & Lee, C. Implementing N-terminomics and machine learning to probe in vivo Nt-arginylation. bioRxiv 2025.04.09.646507 (2025). (*Co-first Author) (in review)
- Park, S.-J.*, Ju, S.*, Goh, S.-H.*, Yoon, B.-H.*, Park, J.-L., Kim, J.-H., Lee, S., Lee, S.-J., Kwon, Y., Lee, W., Park, K. C., Lee, G. K., Park, S. Y., Kim, S., Kim, S.-Y., Han, J.-Y. & Lee, C. Proteogenomic Characterization Reveals Estrogen Signaling as a Target for Never-Smoker Lung Adenocarcinoma Patients without EGFR or ALK Alterations. Cancer Res. OF1–OF13 (2024). (*Co-first Author)
- Ju, S. & Lee, C. Protein Arginylation, Methods and Protocols. Methods Mol Biology 2620, 229–241 (2023).
- Ju, S., Kwon, Y., Kim, J.-M., Park, D., Lee, S., Lee, J.-W., Hwang, C.-S. & Lee, C. iNrich, Rapid and Robust Method to Enrich N Terminal Proteome in a Highly Multiplexed Platform. Anal. Chem. 92, 6462–6469 (2020).

Implementing N-terminomics and machine learning to probe in vivo Nt-arginylation

Shinyeong Ju[1,7], Laxman Nawale[2,4,7], Seonjeong Lee[1,7], Jung Gi Kim[2], Hankyul Lee[1,5], Narae Park[1,5], Dong Hyun Kim[3], Hyunjoo Cha-Molstad[2,4*] and Cheolju Lee[1,6*]

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*Co-correspondence

Abstract

N-terminal arginylation (Nt-arginylation) is a multifunctional post-translational modification (PTM) with roles in protein quality control, organelle homeostasis and stress signaling, but its study has been limited by technical challenges. Here, we developed an integrated approach combining N-terminomics with machine learning-based filtering to identify in vivo Nt-arginylation. Using Arg-starting missed cleavage peptides as proxies for ATE1-mediated arginylation, we trained a transfer learning model to predict mass spectra and retention times. By applying the prediction models with an additional statistical filter, we identified 134 Nt-arginylation sites in thapsigargin-treated HeLa cells. Arginylation was enriched in proteins from various organelles, especially at caspase cleavage and signal peptide processing sites. 8/12 tested proteins were further validated for their interaction with p62 ZZ domain. Temporal profiling revealed that ATF4 increased early post-stress, followed by arginylation at caspase-3 substrates and ER signal-cleaved proteins. Our approach enables sensitive detection of rare N-terminal modifications, offering potential for biomarker and drug target discovery.

Satellite Session

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Education

2007.03-2012.02	Ph.D.	Mechanical and Information Engineering, University of Seoul, Seoul, Korea
2004.03-2006.02	M.S.	Mechanical and Information Engineering, University of Seoul, Seoul, Korea
1997.03-2004.02	B.S.	Mechanical and Information Engineering, University of Seoul, Seoul, Korea

Professional Experience

2023.12-present	Senior Researcher	Digital Omics Research Center, Korea Basic Science Institute, Cheongju, Korea
2018.04-2023.12	Research Professor	Institute for Artificial Intelligence Research, Hanyang University, Seoul, Korea
2012.03-2018.02	Postdoctoral Scholar	Dept. of Computer Science and Engineering, University of California, San Diego, La Jolla, US

Publications

- Song KJ, Choi S, Kim K, Hwang HS, Chang E, Park JS, Shim SB, Choi S, Heo YJ, An WJ, Yang DY, Cho KC, Ji W, Choi CM, Lee JC, Kim HR, Yoo J, Ahn HS, Lee GH, Hwa C, Kim S, Kim K, Kim MS, Paek E, Na S*, Jang SJ*, An JY*, Kim KP*. Proteogenomic analysis reveals non-small cell lung cancer subtypes predicting chromosome instability, and tumor microenvironment. Nat Commun. 2024 Nov 23;15(1): 10164.
- Lee C, Park M, Wijesinghe WCB, Na S, Lee CG, Hwang E, Yoon G, Lee JK, Roh DH, Kwon YH, Yang J, Hughes SA, Vince JE, Seo JK, Min D, Kwon TH. Oxidative photocatalysis on membranes triggers non-canonical pyroptosis. Nat Commun. 2024 May 13;15(1): 4025.
- Na S, Choo Y, Yoon TH, Paek E. CyGate provides a robust solution for automatic gating of single cell cytometry data. Ana Chem. 2023 Nov; 95(46): 16918-16926.
- Son J, Na S*, Paek E*. DbyDeep: Exploration of MS detectable peptides via deep learning. Anal Chem. 2023 Aug; 95(30): 11193-11200. Na S, Choi H, Paek E. Deephos: Predicted spectral database search for TMT-labeled phosphopeptides and its false discovery rate estimation. 2022 May; Bioinformatics. 38(11): 2980-2987.

From Repository to Innovation: Reusing KPOP Proteomics Data

Seungjin Na[1], Jingi Bae[2], Hokeun Kim[2], Shinyeong Ju[3], Heeyoun Hwang[1], Cheolju Lee[3], Jin Young Kim[1], Sang-Won Lee[2], Eunok Paek[4]

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[2]Department of Chemistry, Korea University, Republic of Korea
[3]Chemical and Biological Integrative Research Center, Korea Institute of Science and Technology, Republic of Korea
[4]Department of Computer Science, Hanyang University, Republic of Korea

Abstract

With the rise of mass spectrometry (MS)-based proteomics, we introduce KPOP (https://kbds.re.kr/KPOP), a public repository for collecting and sharing raw data, identifications, and standardized metadata from proteomics experiments. KPOP provides user-friendly interfaces, fast file transfers, and flexible file management, thereby enhancing the accessibility and value of proteomics data for scientific research. Its user-centric structure organizes data by projects and datasets, with each dataset representing a distinct experimental setup (e.g., biological replicates, technical replicates, or clinical samples). In compliance with ProteomeXchange (PX) Consortium guidelines, KPOP enables registered users to submit their data in either public or private mode. Private submissions allow controlled sharing with designated collaborators, ideal for peer review or pre-publication work, while public submissions provide unrestricted community access.

Beyond serving as a repository, KPOP promotes global data sharing and reuse by curating high-quality datasets that support big data approaches, including machine learning and computational modeling. By reusing public proteomics data, we demonstrate deep learning studies that predict peptide fragment intensities, peptide detectability in MS-based experiments, and protein phosphorylation sites. These examples highlight how repository-driven reuse can generate new biological insights and foster innovation in proteomics.

KHUFO

2025 10.15.-10.17.

BUSAN BPEX

BON VOYAGE TO THE NEXT GENERATION PROTEOMICS

Poster Session

KHUPO
The Korean Human Proteome Organization

Poster Session A. Biomolecular Interactomics

PS-A001

Comparative IP–MS Analysis of HTT Interactomes : Construct- and Cell-Type–Dependent Network Signatures

Eunseo Kim[1,2], Hong-Beom Park[1,2], Gyuri Park[2,3], S inae Lee[2,4], Jin Lee[1,2], Minhyeok Kang[2,3], Yerin kim[2], and Dohyun Han[2,5]

[1] Department of Biomedical Sciences, Seoul National University Graduate School, Seoul, 03080, Korea,
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[3] Molecular Medicine and Biopharmaceutical Sciences, WCU Graduate School of Convergence Science and Technology Seoul National University, Seoul, 03080, Korea,
[4] Cancer Research institute, Seoul National University college of medicine, Seoul, 03080, Korea,
[5] Proteomics core facility, Biomedical Research Institute, Seoul National University Hospital, Seoul, 03082, Korea

Huntingtin (HTT) is a 3,144-residue scaffold that regulates vesicle trafficking, proteostasis and cytoskeletal dynamics in neurons, and expansion of its N-terminal polyglutamine (polyQ) tract (〉36Q) causes Huntington's disease (HD); early toxicity involves both full-length (FL) HTT and proteolytic fragments such as exon1. Although numerous affinity-purification mass-spectrometry (AP-MS) studies catalog HTT interactors, few distinguish domain architecture—FL versus exon1—or cellular context—non-neuronal versus neuronal—so how truncation reshapes the interactome in disease-relevant settings remains unclear, hindering mechanism-based target discovery. To bridge this gap, we built a quantitative framework dissecting HTT interactomes along two axes—domain (FL vs exon1(Q23)) and cell type (HEK293T vs SH-SY5Y)—and, through AP-MS with structural modelling, aimed to (i) identify condition-specific interactors, (ii) characterize their functional and spatial signatures and (iii) propose structural hypotheses for domain-selective binding. Tagged HTT-FL and exon1(Q23) were transiently expressed in each cell line alongside untransfected controls; lysates underwent anti-FLAG/HA immunoprecipitation, S-Trap digestion and Orbitrap LC-MS/MS, and interactors were selected via moderated t-tests ($log_2FC \geq 1$; FDR 〈 0.05). Identified proteins were functionally interpreted by over-representation analysis of GO Biological Process and Reactome pathways and by clustering a high-confidence STRING protein-protein interaction network. To add structural context, we built AlphaFold3 hetero-complex models for HTT-partner pairs and first screened model-level confidence (mean pLDDT, ipTM, and global PAE). We then computed interface-resolved metrics—median pLDDT and PAE within the interface—and overlaid IUPred2A (ordered if pLDDT ≥ 70 & IUPred ≤ 0.3) and PScore to flag LLPS-prone contacts. These features prioritize high-confidence HEAT-groove bindings versus fuzzy, disorder-rich engagements. The resulting dataset comprises several hundred proteins across conditions; volcano plots, network maps and analysis pipelines will be presented. Preliminary models suggest that FL interacts through defined HEAT-repeat grooves, whereas exon1 preferentially engages LLPS-prone intrinsically disordered surfaces, nominating a subset of candidates for co-IP, mutagenesis and polyQ- or stress-dependent validation. By integrating quantitative interactomics with structure prediction, this study delivers a domain- and cell-resolved map of HTT network organization, supporting future efforts to decode how polyQ expansion or cleavage perturbs proteostasis and to guide interface-specific degradation strategies

PS-A002

Defining the Distinct Proteomic Pool Targeted by 6SGTP through Photo-Crosslinking and Mass Spectrometry

Myeongji Jin[1,2], Jeongbin Yi[1], and Jong-seo Kim[1,2]

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Thiopurine drugs such as 6-mercaptopurine and 6-thioguanine are widely used antimetabolites whose therapeutic efficacy and toxicity are mediated by conversion into thioguanine nucleotides, including 6-thioguanosine triphosphate (6SGTP). While 6SGTP is classically recognized for incorporation into DNA or inhibition of purine metabolism, its potential to engage with cellular proteins and thereby modulate signaling pathways has not been systematically explored. In this study, we employed 6SGTP as a photoactivatable probe under 365 nm UV irradiation to define the proteomic pool targeted by this metabolite. Mass spectrometry analysis revealed characteristic probe-derived shifts of 313 Da after alkaline phosphatase (ALP) treatment and 393 Da without ALP treatment, consistent with labeling via 6SGMP. Importantly, the protein pools enriched under both conditions were largely overlapping, indicating that hydrolyzed 6SGMP is the predominant binding species. To overcome this limitation, we are testing non-hydrolyzable SGTP analogs and λ-phosphatase-IMAC workflows to enhance recovery of the intact 393 Da-modified species. These results reveal that 6SGTP enriches a distinct subset of nucleotide-binding proteins, separate from the canonical GTP-binding proteome, thereby linking thiopurine drug metabolism to previously unrecognized protein interactions. This work highlights a novel proteomics approach for uncovering off-targets and mechanistic pathways of thiopurine action beyond DNA incorporation and metabolic inhibition.

Poster Session A. Biomolecular Interactomics

PS-A003

Spatiotemporally-Resolved Profiling of Protein Dynamics via Proximity-Labeling

Minsang Hwang[1,2] Jae-yoon Park[1], and Jong-Seo Kim[1,2,*]

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Protein proteostasis and subcellular localization are tightly regulated by protein–protein interactions (PPIs) and modulated by post-translational modifications (PTMs). Disruption of this balance impairs protein function and contributes to disease pathogenesis. Biomolecular condensates, increasingly recognized as key regulators of cellular pathophysiology, are thought to be governed by dynamic PTM regulation. However, due to their membraneless nature, such condensates are inherently difficult to isolate physically; at best, traditional affinity-based purification methods have been applied, but these often yield limited and inaccurate results—underscoring the pressing need for more refined and spatially precise techniques. To elucidate the molecular mechanisms driving condensate-associated diseases, we employed proximity labeling–based proteomics to profile the protein composition and PTM landscape associated with condensate assembly and disassembly. We first adopted the conventional strategy of enriching biotinylated proteins followed by tandem enrichment of specific PTMs, aiming to identify localization-specific proteins and their regulatory PTMs. However, widely used statistical models for biotinylation data analysis frequently yield false positives, particularly in biologically ambiguous systems such as biomolecular condensates, thereby undermining the reliability of proximity labeling studies. To address these limitations, we introduce a robust analytical strategy that enables precise identification and accurate quantification of biotinylated proteins, independent of statistical inference. This approach facilitates high-resolution spatial profiling of PTM landscapes. After enriching biotinylated proteins and performing on-bead digestion, unlabeled peptides were collected using a conventional protocol. A portion of these peptides was analyzed to quantify biotinylated proteins, while the remainder was subjected to tandem enrichment for targeted PTMs. In parallel, biotinylated peptides retained on streptavidin beads were eluted and analyzed by mass spectrometry according to the eSPOT-ID protocol. Our strategy significantly reduces the false positive rate compared to conventional approaches, while maintaining quantification accuracy and offering direct spatial evidence. Furthermore, we elucidated the PTM dynamics of proteins involved in stress granule assembly/disassembly—structures difficult to purify due to their membraneless nature. Altogether, our methodology establishes a new standard for resolving protein interactomes and PTM landscapes at suborganellar resolution, including within membraneless subcellular organelles. It provides critical insights into how PTMs regulate protein dynamics in a spatially confined manner and advances our understanding of condensate-associated pathological mechanisms at the molecular level.

PS-A004

Formaldehyde Crosslinking Based Poly(ADP-ribose) Interactome Capture(PARIC) for in Situ Analysis of PAR Related Proteins and Their Role in Stress Granule Formation

Sanghyuk Lee[1], Seonmin Ju[1,2] and Jong-Seo Kim[1,2,*]

[1] School of Biological Sciences, Seoul National University, Seoul, South Korea, [2] Center for RNA Research, Institute of Basic Science (IBS), Seoul, South Korea

ADP-ribosylation is a post-translational modification (PTM) in which ADPribose moiety from NAD+ is covalently attached to target proteins. ADPribosylation can give rise to negatively charged poly(ADP-ribose) (PAR) chains composed of multiple ADP-ribose units, which can extend up to 200mers. Due to these properties, PAR not only functions as a PTM that regulates the function of specific proteins, but also serves as a scaffold for the recruitment of other proteins, thereby acting as a trigger for the formation of biomolecular condensates. However, the understanding of the underlying mechanisms remains incomplete. In particular, these condensates have been implicated in diseases such as cancer and neurodegeneration. Given the clinical use of PAR synthesis inhibitors, a comprehensive understanding of the PAR interactome is essential. However, no proteomic tool has been available to profile these interactions in situ without introducing in vitro artifacts. In this study, we developed the formaldehyde crosslinking based PAR Interactome Capture pipeline. As a result, we identified 42 out of 92 known PAR binders and 2,119 PAR-binding candidates in total. Quantitative comparison following arsenite treatment, which induces stress granule formation, enabled the identification of more than 600 stress granule associated proteins and revealed dynamic changes in the PAR interactome under stress conditions. This study demonstrates that in situ proteomic analysis of PAR binding and its role in condensate dynamics is feasible.

Poster Session A. Biomolecular Interactomics

PS-A005

Cereblon–TRPC1 Axis in the Regulation of Cardiac Ca^{2+} Influx

Nammi PARK[1], Jubert MARQUEZ[1] , Hyoung Kyu KIM[1], Jin Han[1,2,3]

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Cereblon (CRBN), a substrate receptor of the E3 ubiquitin ligase complex, selectively degrades ion channels such as large-conductance calcium- and voltage-activated potassium channels, while transient receptor potential channel 1 (TRPC1) regulates intracellular calcium and plays a key role in cardiac remodeling; however, the cellular mechanisms by which CRBN targets TRPC1 in the heart remain largely unknown. In this study, we demonstrate that CRBN functions as a regulator of TRPC1 to modulate Ca^{2+} signaling in the heart. Cardiac tissues from patients with heart failure with reduced ejection fraction (HFrEF, EF 〉 55%) showed no significant change in TRPC1 expression compared with controls (EF ≤ 55%), but CRBN deficiency under physiological conditions upregulated TRPC1 expression both in vitro and in vivo. Mechanistically, the Lon domain of CRBN directly interacted with both the C-terminal and N-terminal regions of TRPC1, and increased CRBN levels promoted TRPC1 ubiquitination and proteasomal degradation. Furthermore, TRPC1 overexpression in HEK293 CRBN-knockout cells enhanced TRPC1 current density and Ca^{2+} transients, while CRBN-deficient mice exhibited increased cardiac TRPC1 expression during ex vivo ischemia/reperfusion. Collectively, these findings identify CRBN as a selective regulator of TRPC1 degradation that controls intracellular calcium homeostasis in the heart, suggesting that targeting this mechanism may provide a novel cardioprotective therapeutic strategy for heart failure.

PS-A006

Profiling of the Glyceraldehyde-3-Phosphate Interacting Proteome and its Binding Sites

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Protein homeostasis and cellular functions are tightly regulated by protein–protein interactions and post-translational modifications (PTMs). While most studies to date have focused on enzyme-mediated PTMs such as phosphorylation, acetylation, and ubiquitination, recent reports suggest that certain metabolites can directly and non-enzymatically modify proteins through covalent reactions. Such metabolite-derived modifications have the potential to influence protein stability, interactions, and activity, yet the comprehensive scope and physiological significance of these modifications remain poorly understood. In this study, we focused on glyceraldehyde-3-phosphate (G3P), a central glycolytic intermediate. Beyond its well-established role in energy metabolism and nucleotide biosynthesis, G3P contains a reactive aldehyde group that can form Schiff bases with lysine residues of proteins. To investigate this possibility, we treated HepG2 cell lysates with G3P, stabilized the adducts via sodium borohydride reduction, and performed tryptic digestion followed by IMAC-based phosphopeptide enrichment coupled with mass spectrometry. As a structural comparator, we included dihydroxyacetone phosphate (DHAP), which harbors a ketone group, to evaluate the aldehyde-specific reactivity of G3P. Our results demonstrate that G3P induces extensive, concentration-dependent protein modifications under denaturing (urea) conditions and, importantly, also promotes significant modifications under near-native conditions (Triton). Notably, glycolytic enzymes such as fructose-bisphosphate aldolase A(ALDOA), phosphoglycerate mutase 1(PGAM1), and glyceraldehyde-3-phosphate dehydrogenase (GAPDH) exhibited robust G3P modifications, many of which overlapped with known regulatory PTM sites or substrate-binding regions. In contrast, DHAP showed substantially weaker reactivity under identical conditions, highlighting the aldehyde-specific nature of G3P-driven modifications. Protein-pool analyses further revealed that modifications persisting under Triton conditions were enriched in metabolic enzymes, suggesting that G3P can act as a physiological modulator of enzyme activity. Together, these findings establish G3P as more than a simple metabolic intermediate, positioning it as a potential functional metabolite capable of directly modulating protein function through non-enzymatic covalent modifications. This work highlights a previously underappreciated mechanism by which fluctuations in cellular metabolism can influence protein regulation and disease pathogenesis, thereby opening new avenues for exploring metabolite–protein interactions in health and disease.

Poster Session B. Multi-Omics and Proteogenomics

PS-B001

Comprehensive In-Silico Database and Spectral Libraries for The Global, Secretome, and Phosphoproteome of Bacteroides Fragilis

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Bacteroides fragilis (B. fragilis) is a gram-negative anaerobic bacterium that constitutes up to 2% of the healthy human colonic microflora. It is classified into non-enterotoxigenic (NTBF) and enterotoxigenic (ETBF) strains based on secretion of Bacteroides fragilis toxin (BFT), a key virulence factor. BFT has been associated with acute diarrheal disease, inflammatory bowel disease, and colorectal cancer. ETBF facilitates biofilm formation and chronic intestinal inflammation through BFT expression. Despite their clinical importance, proteomic differences between ETBF and NTBF remain poorly understood. This study integrates the global proteome, secretome, and phosphoproteome of B. fragilis to generate the first comprehensive spectral libraries and DIA-based datasets. Global and secretome peptide samples were prepared using filtered-aided sample preparation, while phosphopeptides were enriched with TiO2 and FeNTA following urea-based in-solution digestion. To generate high quality spectral library for DIA-MS, peptides obtained from whole cell and secreted proteins were fractionated using high-pH reversed-phase HPLC. The fractionated peptides were analyzed using Q-Exactive HF-X with DDA mode, and individual samples were acquired with variable-window DIA. Spectral libraries were generated using DIA SpecLib Quant workflow in Fragpipe (v23.0) against the combined ETBF and NTBF sequence database, and DIA data were processed with DIA-NN (v2.0.2) using default settings. Sequence similarity analysis for in-silico database was performed with BLASTP (v.2.16.0). To generate a in-silico database of B. fragilis orthologous proteins, BLASTP analysis identified 3,514 orthologs. However, we could not find ETBF orthologs for 675 NTBF proteins, and 584 ETBF proteins had no NTBF orthologs, resulting in a total of 4,773 proteins. The global proteome spectral library, obtained by DDA analysis using 24 fractions, comprised 1,013,289 precursors, 58,742 peptides, and 3,652 proteins, covering 67.1% proteome coverage of the B. fragilis in-silico database. Global DIA-MS quantified 43,849 peptides corresponding to 3,011 protein groups. The secretome library, constructed from 12 fractions, contained 30,267 precursors and 15,418 peptides across 1,732 protein groups, representing 31.8% coveraged of the B. fragilis database. DIA-MS analysis of the secretome identified 1,348 protein groups comprising 8,960 peptides. Complementing these datasets, a phosphoproteome library was established from six samples, covering 592 phosphopeptides and 565 phosphosites. DIA-MS further identified 411 phosphopeptides mapping to 272 phosphosites. These datasets were analyzed using principal component analysis and volcano plots to visualize strain-specific distinctions. Collectively, these enabled the construction of an integrated, multi-modal proteome resource for B. fragilis strains, facilitating detailed comparative analyses of global, secretome, and phosphoproteomic profiles. Importantly, this resource provides foundational proteomic data to investigate ETBF-NTBF biological differences, elucidate ETBF-associated pathogenic mechanisms, and support proteomic investigations of other microorganisms.

PS-B002

Multi-Omics Profiling Reveals Bioactive Compounds in Warm-Water Extracts of Cereal Grains

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Low-molecular-weight proteins (LMWPs) have garnered significant attention due to antioxidant, anti-inflammatory, and antibacterial properties, and their enhanced ability to traverse cell membranes enables targeted health benefits. A recent study demonstrated that warm-water extraction can effectively extract proteins associated with bioactive materials synthesis in crop seeds. Therefore, in this study, we soaked barley, oat, and wheat grains at 55°C to obtain warm-water extracts (WWEs) and sequentially applied size exclusion filtration using 10KD filter unit to explore the bioactive materials. At first, we performed proteomic analysis using WWEs extracted from each grain, and this approach led to the identification of 570 differentially modulated proteins (DMPs). Further functional annotation of DMPs revealed that these are primarily associated with various cellular metabolic processes such as amino acid metabolism, stress response, and redox regulation. Subsequent metabolomic and peptidomic analyses using LMWPs fractions, enriched by 10KD filter unit from total WWE samples, were conducted to investigate potential bioactive materials in each grain. From these analyses, 747, 2,096, and 366 significant peptides, associated with antioxidant activity, stress response, and amino acid transport, were identified in barley, oat, and wheat grains, respectively. Additionally, we identified a total of 310 metabolites, of which 9 were mainly related to antioxidant, anti-inflammatory, and antibacterial activities with up-regulation exclusively in barley grain. For further verification, cell viability tests were conducted on normal human astrocytes (NHA) and bronchial epithelial cells (16HBE14o). Particularly, we observed that cell viability significantly improved after co-treatment for 72 h with barley LMWPs compared to other extracts. Furthermore, barley LMWPs enhanced the survival rate of 16HBE14o cells when co-treated with the insecticide hydramethylnon (HM). These findings strongly suggest that soaking cereal grains at 55°C results in the accumulation of diverse proteins and bioactive materials that may help prevent cell damage by regulating the cell cycle during insecticide exposure.

Poster Session B. Multi-Omics and Proteogenomics

PS-B003

Multi-Omics in The Gut of Infant Atopic Dermatitis

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An understanding of the phenotypes and endotypes of atopic dermatitis (AD) is needed for the future development of targeted therapies and prevention strategies. Based on recent evidence for the gut-skin axis in AD, this study investigated the underlying mechanisms of AD phenotypes using multi-omics analyses. AD phenotypes were classified using latent class analysis, and multi-omics analyses (gut epithelial cell transcriptome, short-chain fatty acids, microbiome) were performed using stool samples obtained at 6 months of age. Short-chain fatty acids and transcriptome analyses showed a decreased gut acetate level in the early-onset persistent phenotype with potential pathways of Wnt/Ca2+ and acetate/ acetyl-CoA conversion, leading to Th2 immune responses. Early-onset transient phenotype was linked with signaling pathways of AMPK, chemokine, and FcεRI, whereas the late-onset phenotype was associated with pathways of barrier function and IL-17F in allergic inflammatory airway diseases. In additional prediction analysis using the conditional inference tree model, HNRNPA2B1, TMEM88B, CEACAM5, CLCA4, and BROX expression levels showed an ability to subtype the AD phenotypes with an accuracy of 91.1%. Also, correlation analysis between potential genes and microbiota showed an association between HNRNPA2B1 and Enterococcus faecium in the early-onset persistent phenotype. Despite needs of further replication and functional studies, our findings suggest that multi-omics profiles in early life could provide underlying pathophysiological mechanisms of AD phenotypes, with enabling their prediction and therefore a more optimal treatment choice.

PS-B004

Integrated Proteomic and Glycomic Profiling Reveals Cell-Type Specific Diversity in the Mouse Brain

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The brain contain diverse neuronal and non-neuronal cell types with distinct functions. Although the brain has been studied for decades, cell-type specific functions and underlying molecular mechanisms remain incompletely understood, largely due to extensive cellur heterogeneity. Brain cell-type proteomics has expanded our knowledge of neuronal and glia proteins. However, protein abundance alone cannot fully explain protein regulation, especially since more than half of brain proteins are glycosylated. Glycosylation, one of the most common post-translational modifications, playsciritcal roles in early cell-cell interactions. Because glycans respond sensitively to cell state and environmental conditions, they are emerging as highly informative targets for studying cellular diversity. In this study, we integrated LC-MS/MS based proteomics and N-glycomics to compare mouse synaptosomes, astrocytes, and whole brain tissue. We identified approximately 2,500 proteins and 140 distinct N-glycan structures across these compartments. Label free quantitative proteomics revealed that whole brain tissue was enriched in structural and maintenance proteins such as myelin proteins, tubulins, and histones, whereas synaptosomes were dominated by transport and signaling proteins including ATPases, translocases, and synaptic vesicle proteins essential for neurotransmission. At the glycan level, synaptosomes showed glycosylation patterns resembling those of whole brain tissue, with abundant bi-, tri-, and tetra-antennary structures carrying LacNAc extensions. By contrast, astrocytes were enriched in sialylated complex/hybrid glycans. Notably, fucosylation emerged as a defining feature of synaptic glycans, while astrocytes exhibited marked increases in sialylation. These findings demonstrate that integrated proteomic and glycomic analyses reveal hidden layers of brain cell-type diversity beyond protein abundance alone. To further elucidate the molecular mechanisms underlying these differences, we are extending this study with N-glycoproteomics, employing complementary fragmentation strategies including CID, HCD, and EAD.

Poster Session B. Multi-Omics and Proteogenomics

PS-B005

Systematic Proteogenomic Analysis Identifies Causal Plasma Proteins and Subtype-Specific Biomarkers for Alzheimer's Disease

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Alzheimer's disease (AD) is a multifactorial neurodegenerative disorder with high heritability and growing public health impact. Although recent studies have identified numerous blood-based biomarkers for early AD detection, the lack of causal evidence has limited their translational utility. We aimed to identify blood proteins with a potential causal role in AD using large-scale proteomic and genetic datasets. We conducted Mendelian randomization (MR) analyses using plasma pQTL summary statistics from 54,219 individuals and AD-related GWAS datasets, collectively covering over 1.1 million individuals. These GWAS covered diverse AD phenotypes, including clinically diagnosed AD, late-onset AD, tau levels, hippocampal volume, and cardiovascular comorbidities. To evaluate causality and directionality, we employed genetic epidemiology approaches and assessed genetic colocalization to identify shared causal signals. Among approximately 25,000 protein-trait pairs, 71 candidates showed consistent evidence of a directional relationship with AD-related phenotypes. Of these, 15 proteins were further supported by shared genetic architecture, highlighting their potential causal relevance. This study demonstrates a comprehensive proteogenomic approach to nominate and prioritize plasma proteins as putative biomarkers or therapeutic targets for AD.

PS-B006

Emerging Physical Function Indicators of Midlife Cognitive Decline: Insights from the Korean Medicine for Aging Cohort (KOMAC) Multi-Omics Analysis

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The growing global burden of dementia highlights an urgent need for effective tools to detect early cognitive decline. While traditional physical indicators like BMI, muscle mass, and grip strength are linked to cognitive health in older adults, their predictive value is limited in middle-aged populations. This study, leveraging data from the Korean Medicine for Aging Cohort (KOMAC), sought to identify more sensitive physical markers for early cognitive vulnerability in midlife. We evaluated 1,000 community-dwelling adults between the ages of 50 and 65. Alongside conventional measures, we assessed novel indicators of physical capacity. To explore the underlying biological mechanisms, we conducted a comprehensive multi-omics analysis, including proteomic and metabolomic profiling, on participants grouped by their cognitive and physical function. This revealed distinct molecular signatures associated with different states of physical function and cognitive health. These findings offer new insights into the biological pathways that link physical deterioration to an increased risk of cognitive decline. In conclusion, our research supports core endurance as a practical and non-invasive screening tool for identifying individuals at risk of cognitive decline in midlife. The integration of multi-omics data provides a deeper understanding of the biological connections between physical performance and cognitive aging, which could pave the way for future targeted interventions and preventative strategies.

Poster Session B. Multi-Omics and Proteogenomics

PS-B007

Porphyran Protects Against PCM-Induced Calcification by Restoring Mitochondrial Function in hVICs

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Calcific aortic valve disease (CAVD) is a prevalent and progressive valve disorder marked by osteogenic reprogramming and calcium deposition in valvular interstitial cells (hVICs). Despite its clinical burden, no pharmacological therapy exists, and valve replacement remains the only option. In this study, we investigated the protective role of porphyran, a sulphated galactan from edible red seaweed (Pyropia/ Porphyra), in PCM-induced calcification of patient-derived hVICs. Porphyran markedly reduced calcium deposition, down-regulated osteogenic markers (RUNX2, OPN, BMP2), decreased mitochondrial ROS, and improved oxygen consumption rate. Quantitative proteomics revealed 100 differentially expressed genes (DEGs) and 36 differentially expressed proteins (DEPs) suppressed under PCM, with 38 proteins rescued following porphyran treatment. GO enrichment identified cellular respiration and protein folding as the principal restored pathways. Key mitochondrial components including COX6B1, UQCRC2, HSPD1, and the antioxidant enzyme GSTK1 were restored (p value < 0.05 / log2(FC) < -1.5). Proteomic results were independently validated by RT-qPCR, demonstrating consistent directional changes in gene expression. Collectively, these findings demonstrate that mitochondrial genes are disrupted in calcifying hVICs and that porphyran attenuates calcification by preserving mitochondrial function and redox balance, indicating it as a promising candidate for pharmacological intervention in CAVD.

Keywords: CAVD; Porphyran; Mitochondrial function; Oxidative stress; proteomics.
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PS-B008

Integrative Proteomic Approach to Reveal Altered Signaling Modules During Alzheimer's Disease Progression in Tau P301S Mice

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Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by cognitive, functional, and behavioral impairments. Its neuropathological hallmarks include the accumulation of extracellular amyloid-β plaques and the formation of intracellular neurofibrillary tangles composed of hyperphosphorylated tau protein. In addition to these features, dysregulation of neurotransmitter systems, including dopamine, contributes to impaired synaptic transmission and neuronal communication in AD. Although the link between neurotransmitter alterations and AD pathology is recognized, comprehensive profiling of neurotransmitters and their associated proteomic changes remains limited. To address this gap, we conducted an integrated proteomic and neurotransmitter analysis across seven brain regions of PS19 (Tau P301S) transgenic mice at different stages of AD progression. Our proteomic analysis revealed distinct alterations in canonical pathways, including metabolic dysfunction, that varied across brain regions. Furthermore, profiling of neurotransmitters identified significant alterations in six neurotransmitter systems during AD progression. Through integrative analysis, we uncovered specific neurotransmitter-related signaling modules that exhibit AD progression-dependent associations with neurotransmitter alterations, particularly within the hippocampus and cerebellum. This integrated approach provides novel insights into the molecular mechanisms underlying AD progression and highlights potential signaling pathways involved in disease development.

Poster Session B. Multi-Omics and Proteogenomics

PS-B009

Proteometabolomic Profiling of Starved Mouse Liver by Tandem Mass Spectrometry

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Nutritional status is a regulator of metabolic homeostasis, and fluctuations in nutrient supply can have a profound effect on physiological functions. While prolonged deprivation is harmful, controlled restriction–such as intermittent fasting–has been linked to lifespan extension and delayed age-related diseases. Starvation is a form of acute and extreme nutritional stress that induces systemic metabolic adaptations, in which the liver plays a central role. In this study, we performed proteometabolomics based on mass spectrometry to investigate liver-specific molecular changes in response to starvation. Male mice were starved for 48 h, and liver tissues were analyzed. A total of 5,632 proteins and 259 metabolites were quantified, of which 270 proteins and 153 metabolites were significantly altered. The integrated omics analysis revealed major pathway alterations such as lipid metabolism (e.g., glycerophospholipids), energy metabolism (TCA cycle, glycolysis/gluconeogenesis), amino acid and antioxidant systems (cysteine and methionine metabolism), and sugar mobilization (starch and sucrose metabolism). These changes reflect energy balance, oxidative stress response, and structural remodeling as adaptive responses to starvation. The findings of this study offer insights for understanding the metabolic adaptation mechanisms of the liver during acute starvation and may aid in the developing of novel therapeutic strategies for the management of metabolic diseases.

PS-B010

Multi-Omics for Plasma: A Three-in-One End-to-End Automated Sample Preparation and LC/MS Metabolomics, Lipidomics, and Proteomics Workflow

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This study presents a comprehensive, automated, and reproducible LC/MS-based multi-omics workflow for plasma and cell samples, integrating metabolomics, lipidomics, and proteomics. Utilizing Agilent's Bravo Metabolomics Sample Prep Platform and Infinity II Bio LC coupled with the 6495D LC/TQ system, the workflow enables high-throughput analysis with attomole-level sensitivity and precision across hundreds of analytes. The platform supports both standard and low-flow methods, allowing simultaneous positive and negative mode detection in a single injection. Over 500 polar metabolites, 763 lipids across 44 classes, and 375 mouse plasma peptides can be quantified using reproducible chromatographic methods. Automation significantly reduces sample preparation variability (~50%) compared to manual protocols, enhancing statistical power for large-scale biological studies. This integrated solution demonstrates robust performance, transferability across labs, and suitability for pathway discovery, biomarker quantification, and precision medicine applications.

Poster Session C. Clinical Proteomics

PS-C001

Revitalization of Visual Senescence by Application of Pluripotency Genes

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Age-related macular degeneration (AMD) is a main cause of central blindness among the working-age population. In 2020, it affected more than 190 million people world-wide with the prevalence expected to increase to 288 million people by 2040. According to the Federal Aviation Administration (FAA) Federal Air Surgeon's bulletin, people whose occupation is air maneuvering or flight control are under strict FAA regulation and may lose their license when AMD develops. To overcome the disease, rejuvenating factors could be potential solutions of revitalization. Yamanaka factors are transcription factors that can convert somatic cells into pluripotent stem cells. Yamanaka factors consist of OCT4, SOX2, KLF-4, and c-MYC (OSKM). Because induced pluripotent stem cells (iPSCs) can propagate indefinitely, and have capacity to transform into every other cell type in the body, they hold promise in the field of regenerative medicine. In various bioinforamtic studies, quantitative proteomics is an extremely powerful primary approach used to understand global proteome abundance and dynamics in a cell, tissue, or organism. The main methodology in quantitative proteomics is ultra high-resolution mass spectrometry combined with nano-liquid chromatography. Although this strategy is more complex and lower-throughput than the current genomic approaches represented by RNAseq, quantitative proteomics overcomes many limitations of RNAseq by directly quantifying global protein expression/modifications. The proteomic markers obtained are used to reconstruct underlying the molecular mechanisms of biological processes. In this study, we propose to test 1) the rejuvenation anti-senescence potential of Yamanaka factors in an AMD cell line model, which has not been attempted, 2) probe the proteomic landscape of the rejuvenate/unrejuvenated AMD cells to understand the molecular mechanism of rejuvenation, and 3) validate these findings in the mouse AMD model. To this date, application of state-of-art quantitative proteomics in the field of rejuvenation studies has been rarely attempted. If successful, insights obtained from this study could provide a platform to develop pharmaceutical solutions against AMD. We hope that these lines of investigations will lead us to a deeper understanding of how the induction of pluripotency inhibits AMD, and open the door to a new line of studies.

PS-C002

Automated Sample Preparation coupled with MALDI-TOF Mass Spectrometry for High-Throughput M-Protein in Multiple Myeloma

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Monoclonal proteins (M-proteins) are critical biomarkers for diagnosing plasma cell disorders, including multiple myeloma. Conventional electrophoresis-based methods are limited in resolution, specificity, and speed. Additionally, distinguishing M-proteins from co-existing drugs remains a diagnostic challenge. We developed an automated sample preparation coupled with MALDI-TOF mass spectrometry to improve qualitative detection of M-proteins, reduce processing time, and evaluate the ability to distinguish M-proteins from potential interfering substances. Clinical serum samples included M-protein positive, M-protein positive with spiked drug, and M-protein negative (healthy control) specimens. The Hamilton ML prep system was used to automate the sample preparation and immunoprecipitation of M-proteins was performed using CaptureSelect nanobeads, followed by MALDI-TOF mass spectrometry for qualitative analysis. The resulting spectral profiles were compared to evaluate qualitative detection and discrimination between M-protein and drug signals. Using the Hamilton ML prep system, we achieved a 16-fold reduction in sample preparation time compared to manual workflows. Distinct M-protein peaks were clearly observed in the positive samples. In the drug spiked samples, additional peaks corresponding to the drug were detected, while the M-protein peaks remained unchanged. This result demonstrats the platform's ability to distinguish protein signals from drug interferences based on m/z values. No significant peaks were observed in negative control samples. The automated workflow reduced hands-on time while maintaining reproducibility and accuracy. Our automated MALDI-TOF-based approach enables rapid and reliable qualitative detection of M-proteins, with the added benefit of distinguishing them from potential drug interferences. This platform shows promise for integration into routine clinical diagnostics and may serve as a basis for future quantitative development.

Poster Session C. Clinical Proteomics

PS-C003

Sensitive Detection of Patient-Specific M-Protein Peptides for MRD Monitoring in Multiple Myeloma Using Targeted LC-MS

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Multiple myeloma (MM) is a hematological malignancy characterized by clonal plasma cell proliferation. Monitoring minimal residual disease (MRD) is essential for assessing treatment efficacy and detecting disease recurrence. However, detecting extremely low levels of M-protein in clinical settings remains challenging, especially when conventional assays fail to identify such low concentrations. This study aims to develop a targeted LC-MS method for the sensitive detection of patient-specific M-protein peptides, enabling precise MRD monitoring in multiple myeloma patients. Immunoglobulin enrichment was performed on patient serum samples, followed by a patient specific M-protein sequencing to identify clonotypic peptide sequences. A targeted LC-MS method was then established to analyze these patient-specific peptides. To simulate clinical conditions, patient serum was diluted with negative serum to create monitoring sets. The processed samples were injected to LC-MS instrument, and the M-protein peptides were detected. The targeted LC-MS analysis successfully detected patient-specific M-protein peptides in samples diluted under 0.004 g/dL. The method demonstrated the ability to monitor M-protein levels at concentrations below the detection limits of conventional clinical assays, enabling the detection of disease recurrence at early stages. The developed targeted LC-MS assay provides a highly sensitive and specific approach for monitoring MRD in multiple myeloma patients. This method has the potential to be integrated into clinical practice, offering an effective tool for early detection of disease relapse and informing timely therapeutic decisions.

PS-C004

M-Protein Quantification Using Non-Linear Calibration Curves in MALDI-TOF Mass Spectrometry

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An MALDI-TOF MS-based method for multiple myeloma diagnosis has been developed by a top-class hospital, Mayo Clinic. However, it has notable challenges, including limited quantitative stability under low M-protein concentrations due to high sensitivity to variation in peak width, and potential diagnostic bias due to not accounted for gamma globulin levels. Here, we develop an enhanced MALDI-TOF MS-based diagnostic method that employs non-linear calibration curves for accurate quantification up to low concentration (〈0.1 g/dL), making it potentially suitable for follow-up monitoring. M-protein quantification was performed using non-linear calibration curves, with monoclonal-to-polyclonal antibody area ratio (mAb-to-pAb ratio) as the response variable. The calibration curves, reflecting gamma globulin concentrations, was generated by non-linear curve fitting. To construct calibration curves, three serum types and nine calibration points were used (hypogammaglobulinemia, normogammaglobulinemia, and hypergammaglobulinemia). All spectral analyses were conducted using R (version 4.3.2). Each spectrum was functionalized using Gaussian KDE, and the mAb area was mathematically calculated through derivative-based peak detection and spline regression-based identification of tangential skim points. Our method allowed the measurement of mAb-to-pAb ratios with a coefficient of variation (CV) below 7% across a wide range of gamma globulin levels and across mAb concentrations ranging from 0.016 to 1.0☒g/dL. Using non-linear calibration curves, M-protein concentrations were estimated in clinical MM-positive samples of various isotypes with known reference values, with relative errors of 0.27% for IgG-kappa, 9.57% for IgG-lambda, and 14.25% for IgM-kappa. Furthermore, the non-linear calibration curves method maintained acceptable accuracy even at high dilution (final concentration: 0.0182 g/dL). Additionally, when the drug and M-protein coexisted, our method successfully detected the drug (Avelumab), identified its isotype, and quantified the M-protein. Non-linear calibration curve maintained reliable quantification performance by the lowest level of MALDI-based M-protein quantification cases. Moreover, it allows the distinction between therapeutic drugs and M-proteins. These capabilities are expected to be effective for accurate diagnosis and prognostic monitoring in the future.

Poster Session C. Clinical Proteomics

PS-C005

Large-Scale Proteomic Profiling Identifies Progression-Specific Signatures and Biomarkers in Anaplastic Thyroid Cancer

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Thyroid cancer is a prevalent type of endocrine cancer, and it was ranked as the ninth most common cancer in the world. However, the thyroid cancer proteome remains largely unexplored despite identifying oncogenes and their associated mutations at the mRNA level. Anaplastic thyroid cancer (ATC) is aggressive, highly metastatic, and difficult to treat, with a poor prognosis. Given the importance of studying ATC, identifying proteins contributing to tumor progression from differentiated thyroid cancer (DTC) to ATC is essential. These proteins may serve as crucial biomarkers for predicting DTC prognosis and facilitating early diagnosis of ATC. Accordingly, we conducted a proteomic analysis using a cohort of 267 formalin-fixed, paraffin-embedded (FFPE) tissue samples: normal; follicular thyroid cancer (FTC); papillary thyroid cancer (PTC) with or without BRAF V600E mutation; and ATC with or without the mutation. Proteins were selectively extracted from targeted regions of FFPE, followed by enzymatic digestion using the S-Trap protocol. The peptide samples were analyzed by OE480 MS coupled to a Vanquish Neo using a data-independent acquisition (DIA) method. DIA data were processed using Spectronaut software. Statistical analyses were performed using Perseus and R, and feature selection was carried out using a machine learning-based approach. Quantitative proteomic analysis identified a total of 8,844 protein groups. Uniform manifold approximation and projection (UMAP) visualization demonstrated clear sepearation between clinical subtypes, and the thyroid differentiation score (TDS) was lower in poorly differentiated cancers, consistent with prior reports. ANOVA test (FDR < 0.05) yielded 6,667 proteins showing significant differences among groups. Pathway enrichment analysis revealed key biological processes associated with ATC and tumor groups. By analyzing protein expression changes across Normal, DTC, and ATC samples stratified by subtype and mutation, we identified protein signatures that were progressively altered along the disease trajectory. Notably, functional enrichment showed that up-regulated signatures were mainly involved in ribosome biogenesis and RNA processing pathways, while down-regulated signatures were enriched for gas transport pathways. Using random forest combined with grid search-based hyperparameter optimization, we ranked features by importance and selected a progression-specific biomarker panel. The marker panel's ability to distinguish ATC from other groups was confirmed in an external validation cohort using GSVA scoring, and its discriminatory performance was evaluated by AUROC analysis, yielding an AUC of 0.887. In conclusion, our large-scale proteomic study of thyroid tissues suggests a progression-specific biomarker panel for ATC, exhibiting high discriminative power and capturing key molecular pathways involved in tumor progression.
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PS-C006

Evaluation of a Universal Reference Sample for Enhancing Reproducibility in Proteomic Studies: Assessing the Optimal Sample Size

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Integration and comparison of proteomic data across large cohort studies remain major challenges in clinical research. In multiplexed proteomics, internal reference samples (IRSs) are commonly prepared by pooling all samples under investigation for quantitative normalization and comparison. However, variability in IRSs across studies complicates data harmonization. Protocols for using the same reference sample across studies as a universal reference sample (URS) are not yet well established. We hypothesized that pooling a sufficiently large number of samples would minimize differences between mixture samples from different groups, enabling reliable quantitative comparisons across different case groups. Using data-independent acquisition (DIA)-based proteomics, we investigated the number of pooled samples required to ensure reproducibility and consistency. In the discovery cohort, 180 paired gastric cancer and normal adjacent tissue (NAT) samples from the National Cancer Center Biobank were pooled into mutually exclusive groups of 5, 15, 45, 60, 90, and 180 pairs. The validation cohort included 135 paired samples from four independent biobanks, pooled into groups of 5-90 pairs. The coefficient of variation (CV) of quantified proteins notably decreased up to the 45 pairs, with no further significant reduction beyond 45 pairs. In groups of 45 or more pairs, principal component analysis (PCA) showed indistinguishable scattering, and hierarchical clustering analysis (HCA) revealed no group-specific separation based on 1,617 proteins significantly different by analysis of variance (ANOVA, $p < 0.05$). Pearson correlation supported these findings, showing strong correlations in the discovery ($r = 0.994$–0.999) and validation cohorts ($r = 0.989$–0.999). These results suggest that pooling more than 45 pairs provides a stable and reproducible URS, and the possibility of comparing and integrating data from multiple experiments by using a URS, despite the data being obtained from different laboratories. Our findings will facilitate reliable data integration and comparison across multiple studies and laboratories, ultimately supporting large-scale, multi-center gastric cancer proteomic investigations.

Poster Session C. Clinical Proteomics

PS-C007

Identification of Plasma Biomarkers for CADASIL Diagnosis and Monitoring: Insights from Inflammation Pathway Analysis

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Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL) presents a formidable challenge in clinical management due to its wide array of neurological symptoms and genetic complexity. While genetic testing for NOTCH3 mutations serves as the primary diagnostic method, there exists a pressing need for supplementary blood-based biomarkers, especially in cases where genetic testing is impractical or inconclusive.

This study aims to identify such biomarkers through plasma protein analysis, aiming to enhance CADASIL diagnosis and treatment approaches. Plasma samples were meticulously collected from CADASIL patients, stroke patients, and healthy controls, ensuring demographic parity among the groups. Employing the proximity extension assay (PEA), we meticulously examined 92 inflammation-related proteins, seeking potential biomarkers that could effectively differentiate CADASIL from stroke. Our comprehensive analysis unveiled notable differential expression of three inflammation-related proteins in CADASIL patients compared to stroke patients, suggesting their candidacy as novel biomarkers for CADASIL diagnosis.

These findings not only deepen our understanding of CADASIL pathophysiology but also hold promise for the development of targeted therapeutic interventions. The identification of plasma biomarkers for CADASIL diagnosis and monitoring represents a significant stride forward in the field, furnishing clinicians and researchers with invaluable diagnostic tools. Further validation studies are imperative to ascertain the clinical utility of these biomarkers and explore their potential in tailoring personalized treatment regimens for CADASIL patients.

PS-C008

Proteomic Analysis of FFPE Pathological Placental Tissues to Identify Proteins Associated with Fetal Growth Restriction

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The placenta is a critical organ that mediates the transfer of oxygen, nutrients, hormones, and immune factors from the mother to the fetus, playing a central role in fetal organ development. Dysfunction of the placenta can adversely affect fetal development and is associated with long-term health issues such as fetal growth restriction (FGR) and neurodevelopmental delays. However, studies investigating placental pathologies related to poor pregnancy outcomes at the molecular level remain limited, highlighting the need for further research. This study aims to investigate proteomic alterations in formalin-fixed paraffin-embedded (FFPE) pathological placental tissues associated with FGR and poor neonatal prognosis and to identify unique proteins potentially associated with these adverse outcomes. FFPE placental samples from normal controls and from FGR cases exhibiting distinct pathological features were subjected to proteomic analysis. Proteins were extracted from FFPE samples and subjected to in solution digestion. Peptides derived from each sample were then labeled using tandem mass tags (TMT) for multiplexed quantification, followed by fractionation using high-pH reversed-phase chromatography. LC-MS/MS analysis is being conducted to compare protein expression levels across samples, allowing for the identification of proteins potentially enriched in placentas with lesion-specific pathological features. These lesion-associated proteins may provide insight into the pathophysiological mechanisms underlying FGR-related placental pathology and could serve as a preliminary resource for biomarker discovery with potential clinical applications.

Poster Session C. Clinical Proteomics

PS-C009

Glycoproteomic Workflow for the Discovery of PSA-Based Biomarker for Accurate Early Diagnosis of Prostate Cancer

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This study aims to develop a glycoproteomic workflow for the discovery of prostate cancer (PCa)–specific prostate specific antigen (PSA) that enables more accurate early diagnosis. Serum PSA test used in clinical practice is not cancer–specific, as PSA levels are also elevated in benign prostatic diseases, leading to a high rate of false–positive results. Nevertheless, PSA remains the gold standard for PCa screening, and various screening strategies based on PSA have been developed. PSA has only one N–linked glycosylation site, and its N–glycosylation is known to undergo aberrant alterations in cancer compared with non–cancer groups, highlighting its clinical importance. In this study, various workflows will be attempted using PSA standard samples in order to achieve high sequence coverage of PSA, including its N–linked glycopeptide. All workflows are designed to include trypsin and PNGase F treatment for digestion and deglycosylation, and the order of detailed preprocessing steps will create differences among the workflows. The resulting peptides will be analyzed by nLC–MS/MS, and proteins will be identified using protein database search software. Finally, the optimal workflow evaluated with PSA standard samples will be further assessed for its applicability to cancer tissue and plasma from PCa patients.

PS-C010

Deep Serum Proteome Profiling with Mag-Net Identifies Novel Diagnostic Biomarkers for Prostate Cancer

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Prostate cancer (PCa) diagnosis remains challenging, particularly within the clinical “gray zone” of prostate–specific antigen (PSA, 3–20 ng/mL), where the limited specificity of PSA often leads to unnecessary biopsies. In this study, we developed and evaluated a serum–based proteomic biomarker panel for PCa diagnosis using a liquid chromatography–mass spectrometry (LC–MS) workflow. Serum samples from 30 PCa and 30 benign prostatic hyperplasia (BPH) patients within the PSA gray zone were analyzed. Mag–Net–based protein enrichment enabled the identification of 2,343 proteins, among which 837 proteins with less than 20% missing values were selected for downstream analysis. Differential expression analysis revealed 15 candidate biomarkers, including 12 up–regulated (e.g., SELENOF, PSMA7, MMP19, HGFAC) and 3 down–regulated proteins (IGKV1–5, ECM2, CAVIN2) in PCa compared to BPH. A multi–marker predictive model constructed from these candidates achieved an area under the ROC curve (AUC) of 0.8044, demonstrating significantly higher diagnostic performance than PSA alone (AUC = 0.5567). Notably, combining PSA with the biomarker panel yielded an AUC of 0.7000, which did not improve upon the panel alone, indicating that the selected proteins provide additional discriminatory power beyond PSA.

Poster Session C. Clinical Proteomics

PS-C011

Strategic Enrichment of Serum-Derived Extracellular Vesicles and Particles Enables In-Depth Serum Proteome Profiling

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Comprehensive profiling of the serum proteome is crucial for identifying clinically relevant biomarkers. However, this is hindered by serum complexity and a broad dynamic range, dominated by highly abundant proteins such as albumin, which mask low-abundance proteins and limit proteome coverage. To address this, we developed a cost-effective, depletion-free, and scalable workflow that integrates polyphenolic biomolecule-conjugated magnetic bead-based extracellular vesicle and particle (EVP) enrichment with S-Trap digestion. This enables rapid and high-throughput isolation of serum-derived EVPs, while SDS-based S-Trap digestion ensures efficient lysis of lipid membranes and improved protein extraction. To evaluate the strategy, we performed a parallel comparison of native serum, top 14-depleted serum and EVP-enriched serum. DIA-MS identified 273 proteins from native serum, 433 from depleted serum and 735 from EVP-enriched serum. EVP enrichment markedly improved proteome coverage by enabling detection of low-abundance proteins not observed in other conditions, many of which showed EV-associated features. Albumin abundance was also strongly reduced, and an additional PBS washing step lowered the albumin ratio to below 1 percent, demonstrating an effective depletion effect. Reproducibility was confirmed with technical replicates of the same serum. DIA consistently identified approximately 860 proteins with correlation coefficients above 0.99, and EVP marker proteins also showed correlations above 0.99, demonstrating high qualitative and quantitative reproducibility. The optimized workflow was applied to 50 serum samples from pancreatic, bile duct, gallbladder and liver cancer patients (n = 10 per group) and controls. Enriched samples were labeled using TMT 32plex and analyzed by Orbitrap-based LC-MS/MS, enabling multiplexed quantification of cancer-associated proteomic alterations across two TMT sets. In total, 1,070 proteins were identified, with 824 consistently quantified after excluding missing values. Robust detection of all 13 EVP marker proteins across samples further supports the reliability of the workflow. This EVP enrichment-based serum proteomics strategy enhances proteome coverage by overcoming dynamic range limitations, ensures reproducibility, and provides a robust platform for future discovery of cancer-specific biomarkers.

PS-C012

LC-MS/MS-Based Integrated Proteomic and N-Glycoproteomic Approach to Identify Immunotherapy Response Biomarkers in Cervical Cancer

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Cancer immunotherapy has revolutionized oncology by leveraging the body's immune system to selectively target and eradicate cancer cells. While it has shown remarkable clinical efficacy in a subset of patients, therapeutic responses remain highly variable, emphasizing the urgent need for robust and predictive biomarkers to stratify patients and guide personalized treatment decisions. In this study, we conducted an integrated serum proteomic analysis using liquid chromatography-tandem mass spectrometry (LC-MS/MS) to explore proteome- and N-glycoproteome-level alterations associated with immunotherapy response in cervical cancer. A total of nine longitudinal samples (n=9 per patient) were obtained from each patient throughout the course of treatment. Through label-free quantification and site-specific N-glycopeptide analysis, we identified distinct patterns of protein expression and aberrant N-glycosylation events that correlated with therapeutic responsiveness. These molecular changes suggest differential immune activation states and hold potential as predictive biomarkers. Our results demonstrate that integrated analysis of the serum proteome and N-glycoproteome provides mechanistic insights into immunotherapy response and may contribute to the development of clinically actionable biomarkers in precision oncology. This study highlights the utility of high-resolution LC-MS/MS-based multi-dimensional proteomics for translational cancer research.

Poster Session C. Clinical Proteomics

PS-C013

Nasal Proteomic Signatures for Canine Cognitive Dysfunction Syndrome

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Canine Cognitive Dysfunction Syndrome (CDS) in elderly dogs shares many clinical similarities with human neurodegenerative diseases (NDDs) such as Alzheimer's Disease and Parkinson's Disease, hence often referred to as 'Dog Dementia'. It is therefore plausible to assume that CDS also shares a common pathological feature of NDDs, namely protein accumulation in the brain parenchyma. Based on the CSF outflow route toward the nasal area, it has been proposed that molecular alterations within the brain could be reflected in the nasal mucosa. Therefore, we aimed to differentiate CDS by identifying CDS-specific proteomic alterations in canine nasal discharge. We performed LC/MS-based proteomic profiling of nasal discharge from dogs (n=3 for CDS group and n=3 for healthy controls (HCs)), and each sample was labelled with TMT reagent. Differentially expressed proteins (DEPs) were identified, providing potential marker candidates for CDS. Functional enrichment on DEPs revealed similarities to pathways implicated in human NDDs, supporting the classification of CDS as a neurodegenerative disorder. Further validations were performed with a larger cohort (n=48 for CDS group and n=17 for HCs), using dot blot and enzyme-linked immunosorbent assay (ELISA). This study provides initial molecular insights into CDS pathophysiology and suggests nasal proteomics as a non-invasive approach for biomarker discovery and disease stratification.

PS-C014

Optimizing Semi-Automated Plasma Proteome Sampling Using the KingFisher system

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Plasma proteomics is widely applied for purposes such as biomarker discovery and clinical screening. Despite the presence of nearly 10,000 proteins in plasma, only a few hundred are typically detectable due to the high abundant proteins. To overcome this limitation, various depletion strategies have been proposed. Among them, the Mag-Net protocol offers advantages in automation, cost efficiency, and throughput. However, magnetic bead-based protocols often suffer from increased missed cleavage rates, which compromise the reliability of protein quantitation. In this study, we sought to retain the benefits of Mag-Net automation while reducing the missed cleavage rate to improve data reliability. We systematically compared different digestion conditions, including digestion time, temperature, reduction agents, and digestion methods. DTT proved to be the most effective reduction agent, yielding the lowest missed cleavage rate. Digestion temperature had only minor effects, with a slight improvement observed at 47 °C. Digestion time showed the best outcome with overnight incubation; interestingly, missed cleavage rates improved up to 2 hours and then plateaued. Additional digestion further reduced missed cleavage rates. In terms of digestion methods, S-Trap-based digestion consistently outperformed on-bead digestion. Ultimately, elution from beads followed by S-Trap digestion achieved the lowest missed cleavage rate. These findings suggest that optimization of digestion conditions, particularly through the combination of bead elution and S-Trap digestion, can significantly enhance the reliability of plasma proteomics while preserving the advantages of automated protocols such as Mag-Net.

Poster Session C. Clinical Proteomics

PS-C015

Hypertrophic Cardiomyopathy as a Multi-System Disorder: Insights from Organ Aging and Proteomics

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Hypertrophic cardiomyopathy (HCM) is increasingly recognized as a systemic disorder rather than a purely cardiac disease. Using proteomics-based biological aging estimates across multiple organs, we demonstrate that HCM patients exhibit marked acceleration of organ-specific aging compared to healthy controls. Cardiac tissue showed the greatest age gap, consistent with the primary pathology of HCM, but accelerated aging was also evident in immune, vascular, renal, and metabolic systems. Nearly half of patients displayed accelerated aging in at least one organ, and nearly a third demonstrated multi-organ involvement, far exceeding healthy reference populations. Distinct phenotypes emerged, including "heart agers" and "multi-organ agers," reflecting heterogeneity in biological aging patterns. Importantly, organ aging correlated with adverse clinical outcomes. Patients with major adverse cardiac events (MACE) exhibited significantly greater aging in adipose, arterial, cardiac, and renal tissues, with adipose tissue aging surpassing cardiac aging as the strongest predictor of risk. Machine learning models based on organ age outperformed those using raw proteomic profiles, underscoring the translational potential of this approach for risk stratification. Protein-level analysis identified TNFRSF11B (osteoprotegerin) and NPPB (brain natriuretic peptide) as key predictors of MACE, implicating vascular inflammation, calcification, and diastolic stress, while the inclusion of adiponectin highlighted the role of adipose–vascular crosstalk. Disease enrichment analyses revealed overlap with coronary artery disease, myocardial infarction, diabetes, and heart failure, suggesting shared mechanisms such as endothelial dysfunction, metabolic imbalance, and inflammation. Together, these findings expand the conceptual framework of HCM, highlighting accelerated and heterogeneous biological aging across organ systems, and identify molecular signatures with potential utility for precision risk assessment and therapeutic targeting beyond conventional metrics of hypertrophy or obstruction.

PS-C016

Integrative Surface Proteomics and Computational Analysis for Molecular Profiling in Acute Myeloid Leukemia (AML)

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Acute myeloid leukemia (AML) is a heterogeneous hematologic malignancy characterized by the rapid proliferation of immature myeloid cells, leading to bone marrow failure and high mortality. Despite advances in chemotherapy, targeted therapies, and hematopoietic stem cell transplantation, treatment outcomes remain suboptimal, with frequent relapse and considerable variability in patient response. In this context, precise molecular characterization of AML is critical for improved diagnosis, risk stratification, and personalized therapeutic strategies. Here, we present an integrative workflow that combines selective enrichment of membrane proteins from peripheral blood mononuclear cells (PBMCs) with high-resolution mass spectrometry and computational analysis to identify candidate biomarkers in AML. Comparative proteomic profiling of patient and healthy donor samples revealed distinct surface protein expression patterns, enabling the detection of disease-associated signatures. Further computational analysis highlighted subtype-specific proteomic profiles that correspond to clinically recognized categories, with particular relevance for aggressive monocytic subtypes. This study demonstrates that mass spectrometry-based surface proteomics, integrated with data-driven analysis, provides a robust platform for uncovering mechanistic insights and clinically actionable biomarkers, offering new opportunities to advance precision diagnostics and targeted interventions in AML. Supported by the National Research Foundation of Korea (NRF), funded by the Korean government (MSIT) (No. RS-2024-00454407).

Poster Session C. Clinical Proteomics

PS-C017

Proteome Characterization of Lymphocyte-Rich Hepatocellular Carcinoma and the Prognostic Role of Tertiary Lymphoid Structures Based on Spatial Proteomics

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Lymphocyte-rich hepatocellular carcinoma (LR-HCC) is a rare and largely uncharacterized subtype of HCC featuring an immune-rich stroma. Tertiary lymphoid structures (TLS), which are frequently observed in LR-HCC, are known to be prognostically significant in various malignancies, but their role in HCC remains unstudied. We retrieved clinicopathologic data from 191 cases of surgically resected conventional HCC (C-HCC, n = 160) and LR-HCC (n = 31). We conducted immunohistochemistry, multiplex immunofluorescence staining, RNA sequencing, and proteomic analysis. Differences between the subtypes were evaluated statistically. LR-HCC was significantly correlated with larger tumor size, higher Edmondson-Steiner grade, and the presence of TLS. Compared to C-HCC, LR-HCC also showed higher expression of $CD3^+$, $CD8^+$, and $FOXP3^+$ T cells, along with high PD-1 and PD-L1 expression ($p < .001$ for all). Patients with LR-HCC exhibited significantly better overall survival (OS) ($p = .044$) and recurrence-free survival (RFS) ($p = .025$) than those with C-HCC. Proteomic analysis revealed that LR-HCC demonstrated TLS signatures with significantly higher proteomic-based immune scores in 14 of 17 types of tumor-infiltrating immune cells. Furthermore, C-HCC with secondary follicles, the most mature form of TLS, showed significantly better OS ($p = .031$) and RFS ($p = .033$) compared to those without. Across the global proteome, LR-HCC was well-differentiated from C-HCC. We also completed a map of protein-protein interactions between tumor-infiltrating lymphocytes and HCC in the tumor microenvironment. In conclusion, LR-HCC is clinicopathologically and molecularly distinct and shows a better prognosis than C-HCC. The presence of secondary follicles can be an important prognostic marker for better prognosis in both LR-HCC and C-HCC.
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PS-C018

Comparative Proteomic Profiling of FFPE Tissues Reveals Molecular Differences Between Neoadjuvant Chemotherapy and Surgery in Pancreatic Cancer

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Pancreatic ductal adenocarcinoma (PDAC) is a highly aggressive malignancy with limited treatment options, and understanding therapy-induced molecular alterations remains essential for improving patient outcomes. To investigate the proteomic consequences of neoadjuvant chemotherapy (NACT), we performed a comparative mass spectrometry-based analysis of formalin-fixed paraffin-embedded (FFPE) tissues from two patient cohorts: NACT-treated tumors (n=34) and upfront surgery tumors (n=30). Proteins were extracted using adaptive focused acoustics (Covaris S220), digested with S-Trap microcolumns, and analyzed by high-resolution LC-MS/MS on a Thermo Scientific Orbitrap Eclipse Tribrid system. Data were processed with MaxQuant, and after normalization, log2 transformation, and missing value imputation, a total of 3,870 proteins were consistently quantified. Multivariate statistical analyses, including partial least squares-discriminant analysis (PLS-DA), volcano plots, and variable importance projection (VIP) scoring, revealed distinct proteomic clustering between NACT and upfront groups. Functional annotation and gene ontology enrichment highlighted apoptosis and immune-modulatory pathways in NACT-treated tumors, whereas upfront surgery tumors were enriched for cell adhesion, migration, and proliferation-related proteins, reflecting higher metastatic potential. In addition, normal tissues from both groups displayed unique proteomic alterations, suggesting systemic molecular remodeling following chemotherapy exposure. Immune deconvolution using CIBERSORTx further revealed significant differences in immune cell composition, including mast cells and CD4 T-cell subsets, indicating that chemotherapy may reshape the tumor-immune microenvironment. Together, these findings underscore the ability of advanced quantitative proteomics to capture therapy-induced molecular reprogramming in PDAC. By integrating deep proteome coverage, robust statistical modeling, and functional bioinformatics, this study provides a comprehensive resource for biomarker discovery and highlights potential molecular targets that may inform personalized treatment strategies in pancreatic cancer. This research was supported by the NRF (Project No. RS-2024-00454407, Spatial Proteomics Project).

Poster Session C. Clinical Proteomics

PS-C019

Proteomic Signatures for Differentiating Follicular-Patterened Thyroid Tumors

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Preoperative differentiation between thyroid cancer and benign nodules, particularly in follicular-patterned tumors, remains challenging and often necessitates diagnostic surgery. Developing biomarkers for more accurate and rapid diagnosis could reduce unnecessary surgeries and support personalized treatment strategies. This study aimed to analyze the protein profiles of patients with follicular-patterned thyroid tumors, including follicular adenoma (FA), follicular thyroid carcinoma (FTC), follicular nodular disease (FND), and follicular tumor of uncertain malignant potential (FT-UMP), to identify potential biomarkers capable of distinguishing these entities.

Proteins were extracted from formalin-fixed, paraffin-embedded (FFPE) thyroid tissue samples obtained from 26 FND, 66 FA, 48 FT-UMP, 81 FTC, and 50 normal thyroid tissues. The extracted proteins were analyzed using liquid chromatography-tandem mass spectrometry (LC-MS/MS)-based global proteomics. Proteomic data were filtered with a 50% presence threshold per group, followed by normally distributed imputation and quantile normalization.

To identify potential protein biomarkers for distinguishing disease groups, proteins showing significant differences from normal tissues were first identified using Student's t-test, and decision tree analysis was subsequently applied to these differentially expressed proteins to explore candidates with high discriminatory power. Statistical analyses were performed using R software (version 4.3.3), and pathway analysis was based on the Gene Ontology (GO) database.

UMAP analysis of proteomic expression patterns revealed that normal tissues were clearly separated from all disease groups. FTC and FT-UMP showed relatively distinct distributions, while FA was broadly dispersed. FND exhibited a distribution pattern partially similar to FA. This study demonstrates that specific proteins are differentially expressed among various follicular-patterned tumors, highlighting their potential as biomarkers for differential diagnosis in clinical practice.

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PS-C020

A Rapid and Economical Workflow for Protein Biomarker Discovery Using Agilent 6495D Triple Quadrupole LC/MS System

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Recent advancements in triple quadrupole LC/MS systems and the availability of public peptide MS/MS spectral libraries have enabled the rapid development of comprehensive MRM-based assays without the need for extensive proteome identification. In this proof-of-concept study, we utilized the Uniprot human reference proteome, PeptideAtlas plasma spectral libraries, Skyline software, and the Agilent 6495D LC/TQ system to establish a robust peptide quantification workflow in human plasma samples. Notably, the assay was developed without the use of stable isotope-labeled standard peptides (SILs), streamlining the process and reducing cost. The workflow was applied to plasma samples from 40 healthy individuals (20 males and 20 females) for comparative analysis. Results demonstrated the capability of this rapid and economical approach to identify potential protein biomarkers in complex biological matrices.

Key features of the Agilent 6495D LC/TQ system that supported this workflow include simplified MRM method development via Skyline with Agilent Automation Plugin and MassHunter software, accelerated MS acquisition enabling up to 500 concurrent dMRMs, enhanced analytical sensitivity, and excellent reproducibility in LC and MS signals—facilitating label-free quantification across cohort samples.

Poster Session C. Clinical Proteomics

PS-C021

PGS Catalog–Driven Genetic Stratification of OMICS Cohorts Identifies Plasma Proteomic Signatures for Early Alzheimer's Disease

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Biomarker discovery for Alzheimer's disease (AD) increasingly relies on multi-omics integration, yet existing datasets often lack clinical diagnosis or longitudinal follow-up information, limiting their utility for early disease research. To overcome this, we applied polygenic risk stratification using the open PGS Catalog, computing AD polygenic scores (PGS) in participants without clinical AD diagnoses. Plasma proteomics data were subsequently analyzed to identify protein signatures distinguishing high- versus low-PGS groups.

High-risk individuals demonstrated elevated levels of established AD-associated proteins (CHI3L1/YKL-40, YWHAE, YWHAZ) as well as platelet-related proteins (MPIG6B, MYL12B, PLEK) previously implicated in AD pathology. Moreover, proteins linked to protein folding, ER stress, and proteolysis (CALR, CCT5, PSMA7) showed statistically significant alterations. These proteomic patterns in genetically defined, non-diagnosed individuals mirror known AD phenotypes and highlight potential preclinical plasma biomarkers.

Our results demonstrate that coupling genetic risk stratification with proteomic profiling enables exploratory biomarker discovery in legacy OMICS cohorts lacking clinical annotation. This integrative strategy expands the utility of existing proteomic datasets and provides a scalable framework for early-stage biomarker identification in neurodegenerative disease research.

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PS-C022

Role of Cereblon in Diabetic Cardiomyopathy: Evidence from db/bd Mice and Clinical Patient Data

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The high incidence of heart failure in patients with type 2 diabetes mellitus (T2DM) can be attributed to myocardial fibrosis, weakening of ventricular function, and damage to myocardial muscles, such as mitochondrial dysfunction. We studied physiological changes in the heart, which are early characteristics of diabetic myocardial disease, using 6- and 8-week old obese T2DM model mice (db/db, BKS.Cg-Dock7m+/+Leprdb/J) and wild type mice. We confirmed mitochondrial dysfunction in 8 weeks old db/db mice, which was not accompanied by any changes in heart function. Metabolomic analysis performed on heart tissues revealed that the levels of 12 metabolites changed substantially. The levels of glucose and leucine increased considerably, and lipid metabolites changed. Protein expression analysis of the blood and heart tissues confirmed a remarkable increase in cereblon (CRBN) levels and a decrease in AMP-activated protein kinase, a negative regulator. Furthermore, in silico studies on data in the blood of diabetic cardiomyopathy (DCM) patients. These results show that an increase in CRBN, without any abnormal heart function, in early T2DM mice roles an important role in the reduction of mitochondrial function and metabolomic changes. Therefore, CRBN level in blood and heart tissues may serve as a diagnostic biomarker for the detection of early DCM.

Poster Session C. Clinical Proteomics

PS-C023

Prognostic Significance of Tumor-Infiltrating Neutrophils in Pancreatic Ductal Adenocarcinoma by Proteomic Analyses

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Pancreatic cancer is one of the most lethal malignancies in Korea, ranking 4th in cancer-related mortality among men and 3rd among women. Pancreatic ductal adenocarcinoma (PDAC), the most common histological type, has a 5-year survival rate of about 8%, remaining as low as 15–25% even after surgical resection. While PDAC typically exhibits extensive desmoplasia, a subset of tumors shows softer consistency with abundant tumor-infiltrating neutrophils (TINs). These neutrophils act as myeloid-derived suppressor cells (MDSCs), exerting strong immunosuppressive effects that facilitate immune evasion and metastasis. In this study, to investigate the prognostic role of TINs in PDAC with formalin-fixed paraffin-embedded (FFPE) specimens from pancreatic cancer paticent, proteome from normal, neutrophil-derived niche and fibrotic stroma based pancreatic cancer tissue were quantitatiely analyzed with LC-MS/MS system coupled with TMT labeling. MPO, ELANE and CEACAM8 as marker neutrophil-derived niche were highly expressed in TIN and the group of neutrophil-rich tumors enriched the neutrophil degranulation, responsed to bacterium, defense responsed to fungus and so on pathway, on the other hand translation, metabolism of RNA, vesicle-mediated transport and so on were significantly different among three groups. Findings from this study may clarify the role of TINs in PDAC progression and provide a foundation for developing immunotherapeutic strategies.

Poster Session D. Dark Proteome and Cancer Research

PS-D001

Secretome-Based Dissection of the Mechanism of Dasatinib in Gastric Cancer–Associated Fibroblasts

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Gastric cancer (GC) remains one of the leading causes of cancer-related mortality worldwide, with poor prognosis largely attributed to the tumor microenvironment (TME) that supports tumor growth, invasion, and therapeutic resistance. Among stromal components, cancer-associated fibroblasts (CAFs) play a central role by remodeling the extracellular matrix and secreting paracrine factors that promote malignant progression. Targeting CAF function has therefore emerged as a promising therapeutic strategy. In this study, we investigate the effects of dasatinib, a broad-spectrum SRC family kinase inhibitor, on CAF phenotype and secretory profile, as well as the subsequent influence on GC cell behavior. Treatment with dasatinib exerted minimal effects on CAF viability and activation marker expression but markedly reduced their motility. Conditioned media (CM) collected from dasatinib-treated CAFs are subjected to comprehensive secretome profiling using liquid chromatography–mass spectrometry (LC–MS) to identify key secreted proteins that may mediate the suppression of gastric cancer cell progression and tumor microenvironment remodeling. Functional assays further demonstrated that CM from dasatinib-treated CAFs attenuated GC cell migration compared with CM from untreated CAFs. Bioinformatic analysis of differentially expressed proteins is expected to identify novel candidates mediating these effects, thereby providing new insights into CAF-driven TME remodeling. Collectively, our findings indicate that dasatinib modulates the CAF secretome in a manner that may suppress GC cell aggressiveness, highlighting potential secretory targets for combinational cancer therapies. Undated works will be presented.

PS-D002

Elucidating the Role of Fucosylation in Cancer-Associated Fibroblasts (CAFs) of Gastric Cancer

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Gastric cancer (GC), which originates from epithelial cells, is known to involve stromal cells within the tumor microenvironment (TME), particularly cancer-associated fibroblasts (CAFs), that play crucial roles in tumor progression and the development of drug resistance. Targeting components of the tumor microenvironment such as CAFs for combination cancer therapies has been proposed as a new paradigm in cancer research. Although the importance of fucosylation in tumors has recently gained attention, research on fucosylation in CAFs remains insufficient. Furthermore, no studies have yet examined how fucosylation in gastric cancer CAFs contributes to cancer progression. This study aims to elucidate the functional role of fucosylation in gastric CAFs during gastric cancer progression and to propose fucosylation-related therapeutic targets within gastric CAFs. We analyzed public gastric cancer TME single-cell RNA sequencing (scRNA-seq) datasets (GSE183904) to compare the expression patterns of fucosylation-related genes between normal fibroblasts and tumor fibroblasts, further to identify genes selectively upregulated in tumor fibroblasts. In cell-based assays, we optimized the treatment conditions of the fucosylation inhibitor 2-Deoxy-2-fluoro-L-fucose (2FF) in gastric CAFs using lectin blotting, and confirmed that fucosylation was effectively suppressed. Transwell migration assays showed that the ability of 2FF-treated CAFs to promote motility of gastric cancer cells was significantly reduced compared with the control group. Currently, we are further characterizing the phenotypic and signaling alterations followed by 2FF exposure. Further, conditioned media obtained from 2FF-treated gastric CAFs are to be analyzed using high-resolution liquid chromatography–tandem mass spectrometry (LC–MS/MS) to investigate the role of fucosylation in regulating CAF secretome. Updated works will be presented.

Poster Session D. Dark Proteome and Cancer Research

PS-D003

Secretome Profiling to Elucidate MASTL Function in Gastric Cancer-Associated Fibroblasts

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Gastric cancer (GC) remains a significant global health burden due to its high incidence and poor prognosis, especially in advanced stages where therapeutic options are limited. Increasing evidence highlights the pivotal role of cancer-associated fibroblasts (CAFs) in shaping the tumor microenvironment (TME) to support tumor progression. However, the molecular mechanisms underlying CAF activation remain incompletely understood. Microtubule-associated serine/threonine kinase-like (MASTL), a mitotic kinase known for regulating the cell cycle via ENSA phosphorylation, has been previously implicated in GC cell proliferation. In this study, we reveal a novel role for MASTL in the activation of CAFs. Conditioned media from GC cells induced MASTL expression in CAFs, and functional assays—transwell migration and wound-healing—demonstrated that MASTL silencing in CAFs significantly impairs their tumor-promoting capacity. To elucidate the molecular mechanism underlying this phenotype, we employed high-resolution liquid chromatography-tandem mass spectrometry (LC-MS/MS) to profile the secretome of MASTL-silenced CAFs. This advanced proteomic approach enabled the identification of differentially secreted proteins regulated by MASTL. Subsequent bioinformatic analyses, including pathway enrichment and protein-protein interaction network analysis, highlighted the VEGFA-VEGFR2 signaling pathway as a key effector mechanism. These findings suggest that MASTL modulates the secretion of angiogenesis-related factors, thereby contributing to tumor progression. To further validate the involvement of MASTL in modulating angiogenic signaling, we performed tube formation assays using the HUVEC cell line. The results demonstrated that MASTL-regulated secreted factors promote angiogenesis within the tumor microenvironment, providing additional evidence for its role in tumor progression.

PS-D004

Characterizing Kinase Signaling Networks Driven by ATP6AP1 in Triple-Negative Breast Cancer

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Triple-negative breast cancer (TNBC) is a highly aggressive subtype characterized by the absence of hormone receptors and HER2 expression, with limited therapeutic options and poor clinical outcomes. We found that nucleolin-targeting aptamer AS1411 inhibits TNBC cell migration and viability, and reduces ATP6AP1 expression, as shown by proteomic analysis. ATP6AP1 knockdown selectively impairs proliferation and migration of TNBC cells without significantly affecting normal breast epithelial cells, highlighting its potential as a cancer-specific therapeutic target. Although kinase signaling pathways are frequently dysregulated in cancer and represent attractive therapeutic targets, the specific role of ATP6AP1 in these pathways remains poorly understood. Emerging evidence suggests that ATP6AP1 may promote tumor progression through mechanisms independent of its classical role in proton transport as part of the V-ATPase complex, implicating alternative regulatory functions in cancer signaling. To investigate the signaling functions of ATP6AP1 in TNBC, we performed phosphoproteomic profiling following ATP6AP1 knockdown in TNBC cells. These studies aim to uncover ATP6AP1-regulated kinase networks and downstream effectors that drive TNBC progression. Our findings are expected to provide new mechanistic insights and identify potential therapeutic targets in TNBC. Updated results will be presented.

Poster Session E. Recent Advances in Technology

PS-E001

Temporal Proteomic Profiling of Senescent Preadipocytes via TMT-Based Quantification

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Cellular senescence in preadipocytes plays a key role in age-associated adipose dysfunction and chronic inflammation; however, the dynamics of the cell surface proteome during this process remain poorly understood. Here, we optimized a surface protein enrichment protocol compatible with tandem mass tag (TMT)-based quantitative proteomics to improve the detection and quantification of membrane proteins. To enhance the identification of low-abundance surface proteins, we designated a TMT channel for an enriched surface protein sample, allowing consistent peptide matching across all study samples. Applying this strategy to senescence-induced preadipocytes, we conducted temporal proteomic profiling to characterize dynamic changes in the surface proteome during senescence progression. Our analysis revealed a distinct set of membrane-associated proteins that were progressively upregulated in senescent cells, implicating their potential roles as surface markers or mediators of cellular aging. Our approach provides candidate surface markers for investigating cellular senescence.

PS-E002

Improved Analysis of Hydrophilic Peptides Using C30 Reverse-Phase LC-MS/MS

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Hydrophilic peptides often exhibit limited retention on conventional C18 reverse-phase columns, which can reduce chromatographic resolution and MS/MS identification efficiency. Here, we evaluate the use of a C30 reverse-phase column for the analysis of hydrophilic peptides. Our results show that C30 columns provide enhanced retention for these peptides, improving their detection and identification in LC-MS/MS workflows. This improved performance suggests that C30-based chromatography can be broadly applied to the analysis of hydrophilic peptide classes, including those bearing post-translational modifications (PTMs), offering a valuable strategy for enhanced proteomic coverage of challenging peptide populations.

Poster Session E. Recent Advances in Technology

PS-E003

Combined CID-EAD LC-MS/MS Enables Site-Specific Glycosylation Analysis for Immunogenicity Assessment in Advanced Biotherapeutics

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Glycosylation is a critical quality attribute of therapeutic glycoproteins, influencing efficacy, stability, and immunogenicity. In particular, The terminal immunogenic glycan epitopes, present in mammalian but absent in humans, requires careful characterization in biopharmaceuticals and organ transplants. Because immune responses can vary with the localization of non-human glycans, accurate risk assessment demands site-specific glycosylation analysis. In this study, we established a site-specific glycopeptide analysis strategy for advanced biotherapeutics using LC-MS/MS with integrated collision-induced dissociation (CID) and electron-activated dissociation (EAD). CID provides glycan-derived fragment ions, enabling broad glycopeptides identification, while EAD preserves intact glycans and produces extensive peptide backbone fragments (c- and z- ions), allowing precise localization of glycosylation sites. By leveraging tunable kinetic energy, EAD yielded distinct distinct diagnostic ions that enhanced confidence in immunogenic glycan identification and enabled the differentiation of isomeric glycoforms. Applying this approach, we profiled immunogenic glycans at site-specific resolution from cetuximab, wild-type porcine tissue and porcine plasma. Importantly, comparison with α1,3-galactosyltransferase knockout pigs confirmed the complete loss of α-gal epitope at previously identified immunogenic glycosylation sites in wild type samples. Overall, the combined CID-EAD fragmentation strategy provides a reliable framework for accurate site-specific glycosylation analysis and immunogenic glycan identification. This approach supports quality attributes assessment and immunogenicity risk evaluation for advanced biotherapeutics and xenotransplantation products.

PS-E004

Production of Chondroitin Sulfate as a Functional Ingredient Using 3D Chondrocyte Spheroids Derived from Bovine Adipose-Derived Stem Cells

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Chondroitin sulfate (CS) is a well-established natural bioactive compound that has been demonstrated to improve joint function and is extensively utilized in medical applications, including eye health, skin regeneration, cardiovascular diseases, and inflammatory disorders. However, current methods of CS production primarily depend on the extraction of cartilage obtained from the slaughter of bovine, swine, poultry, and sharks, which raises ethical concerns and contributes to environmental burdens associated with livestock rearing. In this study, we established an alternative approach for CS production through the culture of chondrocyte spheroids differentiated from bovine adipose-derived stem cells (bADSCs). Chondrogenic differentiation was induced in low-attachment AggreWell plates by seeding equal numbers of bADSCs, and six different differentiation protocols were compared to optimize CS production. The spheroids successfully formed three-dimensional structures, and among the tested conditions, a serum- and antibiotic-free medium supplemented with insulin-transferrin-selenium, ascorbic acid, dexamethasone, L-proline, and TGF-β3 (Differentiation Method 1) yielded the highest levels of glycosaminoglycans (GAGs), which include chondroitin sulfate. Other differentiation media also enhanced GAG production compared with spheroids maintained in standard growth medium, which served as a negative control. Furthermore, spheroids cultured under Differentiation Method 1 expressed key chondrocyte markers, including collagen II, RUNX2, and cartilage oligomeric matrix protein (COMP). Collectively, these findings demonstrate a robust and reproducible in vitro platform for the generation of 3D chondrocyte spheroids from bADSCs capable of producing GAGs, underscoring the potential for future large-scale CS production using suspension bioreactor systems. This research is supported by research funding from Simple Planet and the Technology Development Program, funded by the Ministry of SMEs and Startups (MSS, RS-2024-00507800).

Poster Session E. Recent Advances in Technology

PS-E005

Technical Comparison of Plasma Sample Preparation Methods: Exploring Alternatives to Antibody-Based High-Abundance Protein Depletion

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Blood is widely regarded as an ideal biofluid for clinical proteomics due to its minimally invasive collection and its potential as a rich source of biomarkers, as it circulates throughout the body. However, comprehensive blood proteome analysis is challenged by the wide dynamic range of protein abundances. To address this issue, antibody-based affinity columns have been employed to deplete high-abundance proteins that can mask signals from low-abundance proteins. While this strategy enhances the detection of low-abundance proteins, it also has limitations, including high cost, substantial labor for large-scale studies, variability in sample preparation, and the unintended co-depletion of low-abundance proteins that interact with high-abundance proteins. To explore alternative methods to antibody-based depletion, we compared various blood sample preparation techniques with highly sensitive LC-MS workflows. A total of seven methods were evaluated: in-solution digestion of neat plasma (Neat), Multiple Affinity Removal Column Human 14 (MARS14), perchloric acid precipitation with neutralization (PCA-N), PreOmics ENRICH-iST, MagNet, Nanomics ProteoNano, and Seer Proteograph XT. Among them, Seer Proteograph XT achieved the highest number of protein identifications (〉6,000), while other enrichment-based methods identified approximately 1,800~2,400 proteins. All methods demonstrated good reproducibility, with median coefficients of variation (CV) below 20%. Notably, the neat and enrichment-based methods showed comparable or even better reproducibility than MARS14, with ENRICH-iST achieving the lowest median CV (9.30%) and ProteoNano the highest among them (15.59%). This study provides a technical evaluation of plasma sample preparation methods and offers guidance for selecting approaches that achieve deep proteome coverage, low quantitative variability, and high efficiency in labor and time for clinical proteomics.

PS-E006

Development of Ultrasensitive and Robust Metabolomics Platform

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Biological functions arise from complex interactions among multiple "-omes," such as proteomes and metabolomes. To address the demand for high-sensitivity metabolomic profiling, we developed a capillary-flow LC-MS platform that significantly improves detection sensitivity while maintaining excellent retention time (RT) reproducibility. Flow rates were systematically reduced from 200 μL/min to 450 nL/min while adjusting column inner diameters to preserve constant linear velocities. Using a reverse-phase particle with exceptionally low back pressure, we fabricated 50-cm-long columns that achieved narrow peak widths (FWHM), superior isomer separation, and effective retention of hydrophilic phosphate-containing metabolites. Rigorous column quality control ensured highly reproducible home-packed columns with RT variability below 0.5% CV and enabled the construction of a reference spectral library for 200 compounds. Operating at a flow rate of 1 μL/min resulted in an average ~100-fold enhancement in signal intensity for 45 metabolite derivatives compared with conventional flow rates (200 μL/min). This optimized platform achieves sensitivity comparable to state-of-the-art nano-flow LC-based proteomics systems and provides a robust foundation for high-resolution and high-sensitivity metabolomic profiling.

Poster Session E. Recent Advances in Technology

PS-E007

Emerging Approaches in Mass Spectrometry-Based Proteomics for Decellularized Extracellular Matrix

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Decellularized extracellular matrices (dECM), produced by removing cellular constituents from native tissues, are anticipated to serve as novel biomaterials due to their inherent attributes such as robust tissue regenerative potential, low immunogenic response, controlled degradability, and excellent hemocompatibility. Proteomic profiling of dECM can reveal and quantify the ensemble of ECM proteins that mediate extracellular signaling; however, detailed analysis is challenging because of the limited solubility of matrix proteins. The main objective of our research team is to establish analytical platforms for dECM proteome characterization and to elucidate proteomic variations among dECMs derived from distinct tissue sources. We have implemented chemical digestion and high-pressure extraction utilizing Pressure Cycling Technology (PCT) within our sample preparation workflow. Following optimization with dECM isolated from rat brain, chemical cleavage led to marked enhancement of peptide yields from ECM components such as collagen proteins. Moreover, applying high-pressure extraction shortened the chemical cleavage duration from 17 hours to only 3 hours. We further explored stepwise extraction approaches, combining chemical cleavage with phase-transfer surfactants (PTS). This sequential extraction protocol improved the overall protein identification coverage from dECM samples. Ultimately, we identified 3,316 proteins via chemical cleavage, 2,388 through PTS extraction, and 3,502 proteins using sequential extraction after normalization in rat brain dECM (n = 3). The optimized proteomic methodology for dECM characterization developed herein will be broadly applicable to various ECM-related investigations.

PS-E008

Enhanced Proteomic Profiling via Multiple Affinity Removal System (MARS)-Based Depletion of Serum High-Abundance Proteins

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Serum is one of the most widely used biofluids for biomarker discovery, but its proteomic analysis is challenged by the extreme dynamic range of protein concentrations. Highly abundant proteins such as albumin and immunoglobulins account for more than 95% of total serum protein content, masking low-abundance biomarker candidates. In this study, we employed a Multiple Affinity Removal System (MARS) Human-14 column to deplete 14 major abundant proteins from over 300 human serum samples using high-performance liquid chromatography (HPLC). We developed a quantitative screening method based on chromatogram peak areas to assess sample suitability prior to mass spectrometry (MS) analysis. Comparative analysis of cancer versus normal serum revealed greater variability in unbound/bound protein ratios in cancer samples, suggesting potential diagnostic utility. Data-independent acquisition (DIA) nanoLC-Orbitrap Exploris 480 MS was used to evaluate the efficiency of depletion. Our results demonstrated reproducible removal of high-abundance proteins, enabling enhanced detection of low-abundance proteins. This workflow provides a robust and scalable approach for serum sample preparation and quality control, facilitating reliable biomarker discovery through MS-based proteomics.

Poster Session E. Recent Advances in Technology

PS-E009

Hanwoo Serum as a Potential Alternative to Fetal Bovine Serum: Cost-Effective, Stable, and Ethical

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Fetal bovine serum (FBS) is widely used in cell culture; however, its high cost, ethical concerns, and dependence on overseas supply have raised the need for alternative serum sources. In this study, adult bovine serum derived from Hanwoo (Korean native cattle) blood, which is typically discarded after slaughter, was developed to achieve a stable supply and cost reduction. The effects of Hanwoo serum (HWS) on cell proliferation were evaluated using chicken embryonic muscle-derived fibroblasts (cMDF) and bovine myoblasts (bMYO). The cells were cultured in media supplemented with 10% FBS, 10% HWS, or 20% HWS, and proliferation was measured using a CCK-8 assay. Both cMDF and bMYO maintained over 70% and 80% of the proliferation rates observed with FBS, respectively. To further assess cell characteristics, qPCR analysis was performed on cMDF-specific markers, including FAP, Thy-1, α-SMA, and Vimentin. The results showed that FAP and Vimentin expression levels were comparable to those observed with FBS, whereas Thy-1 and α-SMA, which are associated with cell proliferation and adhesion, were reduced in cells cultured with HWS. This decrease in marker expression may explain the slightly lower culture performance of HWS relative to FBS. While FBS is prone to batch-to-batch variation due to physiological differences among fetuses, HWS, derived from adult blood, was expected to exhibit less variability due to relatively uniform physiological status. To verify this, the quality of serum collected from three different Hanwoo individuals was evaluated, confirming consistent culture performance and quality uniformity. Taken together, these findings indicate that HWS possesses potential as an alternative to FBS, while also revealing inherent limitations that currently preclude its full substitution. To overcome the aforementioned limitations of HWS, research on supplementation strategies is required, and once these limitations are addressed, it is expected that a stable supply and commercialization of domestic cell culture serum will be possible. This research is supported by research funding from Simple Planet and the Korea Institute of Marine Science & Technology Promotion (KIMST) funded by the Ministry of Oceans and Fisheries (MOF, RS-2024-00405273).

PS-E010

Development of a Scalable Suspension Culture System for Muscle-Derived Fibroblasts in Food Industry Application

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The escalating global food demand and the environmental burden of livestock farming have positioned cell-based alternative proteins as a pivotal solution for sustainable protein supply and the expansion of the high-value-added food industry. However, the development of scalable cell culture platforms is hindered by the surface area limitations and high production costs associated with adherent cell cultures. Although research utilizing immortalized cells or stem cells has progressed, overcoming the limitations of cell division, the inefficiencies of adherent culture systems in scaling up remain unresolved. This study aimed to establish suspension culture system for chicken embryonic muscle-derived fibroblasts (cMDFs), an economically viable and stable cell source, and to integrate these conditions into bioreactor systems for large-scale production. For this purpose, isolation method of cMDFs was developed, and cell purity was confirmed through polymerase chain reaction (PCR) and quantitative PCR (qPCR) analyses to confirm interspecies (chicken cytochrome c oxidase subunit Ⅰ; COⅠ), gender (XhoI within W chromosome of female chicken) and fibroblast-specific markers (FAP, Thy-1 and Vimentin). To obtain cMDFs with superior cell proliferation capacity, population doubling time (PDT) and population doubling level (PDL) were evaluated. Optimal suspension culture conditions for the cMDFs with superior proliferation ability were established by regulating parameters such as cell inoculation density, agitation speed, and pH regulation. The developed suspension culture system offers significant potential for industrial applications in cultured meat production and high-value-added food development, contributing to sustainable protein supply chains. Future research aims to implement this system in commercial-scale bioreactors to enhance its industrial feasibility and support diverse productization and commercialization initiatives. This research is supported by research funding from Simple Planet and the Korea Institute for Advancement of Technology (KIAT) through the International Cooperative R&D Program, funded by the Ministry of Trade, Industry and Energy (MOTIE, P0028257).

Poster Session F. Exosome

PS-F001

The Proteomic Studies in the N-Degron Pathway-Mediated Protein Metabolism

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Protein homeostasis is maintained by an unending and dynamic balance between the synthesis of native proteins and their metabolism. This intricate process is mediated by precisely coordinated pathways governing protein biogenesis, folding, transportation, secretion, and degradation. Proteins are fundamental components of cellular activity, and proteostasis ensures that proteins maintain their essential roles by preserving their specific 3D conformation, concentration, and localization. Cytosolic proteins serve as structural support, catalytic, enzymatic, and regulatory functions. Membrane proteins are associated with transport and receive the signaling molecules. Secretory proteins play a critical role in cell-to-cell communication, which serves various physiological processes such as development, homeostasis, the immune system, and signal transduction in multicellular organisms. Therefore, precise regulation of both protein degradation and secretion is critical to maintaining proteostasis. In this study, we investigated the molecular mechanisms linking protein degradation and secretion, focusing on the N-degron pathway. Our proteomic analyses revealed that components of the N-degron pathway modulate the composition of exosomes, the key mediators of unconventional protein secretion. Through O-link proteomics, we further characterized the secretion routes of exosome-associated cytokines. Additionally, we performed comparative proteomic profiling to distinguish between selective and bulk autophagy, highlighting unique substrates and regulatory modules involved in each pathway. Taken together, our findings establish a connection between the N-degron pathway and exosomal protein metabolism using the proteomic analysis. These results may provide a framework for understanding how proteostasis contributes to pathological states such as inflammation and cancer.

PS-F002

Exosome Isolation Methods for Individual Proteomics in Aqueous Humor

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Aqueous humor (AqH) derived exosomes have emerged as promising diagnostic biomarkers and therapeutic vehicles for ocular diseases. However, the limited volume and low concentration of AqH present significant challenges in obtaining sufficient exosomal material for analysis. Consequently, studies on AqH-derived exosomes at the individual patient basis remain exceedingly uncommon. This study aimed to compare exosome isolation methods to determine the optimal technique for individual-level proteomic analysis. Exosomes were isolated from 150 µL of AqH using three distinct isolation techniques: size exclusion chromatography (qEV 20 nm), tangential flow filtration (ExoDisc D20), and precipitation (ExoQuick). The yields and purity were assessed using nanoparticle tracking analysis and ExoView assay. To characterize the protein content of AqH-derived exosomes, proteomic profiling was performed using liquid chromatography-tandem mass spectrometry (LC-MS/MS). Among the tested isolation techniques, qEV 20 nm method produced the highest yield of exosome particles from AqH. Proteomic analysis of exosomes isolated via qEV 20 nm identified 146 proteins. These findings demonstrate that the qEV 20 nm method is the most effective for isolating AqH-derived exosomes in individual proteomic studies, providing a foundation for future biomarker discovery and therapeutic development in ocular disease.

Poster Session F. Exosome

PS-F003

Proteomic and Small RNA Profiling of APLP1+ Extracellular Vesicles Reveals Cerebral Origin and Diagnostic Potential for Neurodegenerative Diseases

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Early detection of neurodegenerative diseases requires the identification of brain-derived biomolecules in peripheral blood. In this study, we propose amyloid precursor-like protein 1 (APLP1), predominantly expressed in oligodendrocytes and neurons, as a novel biomarker in extracellular vesicles (EVs). Analysis of APLP1+ EVs isolated from human sera revealed brain origin, supported by distinctive small RNA expression patterns and the brain-specific expression profiles of their predicted target genes. Proteomic analysis identified numerous proteins directly associated with brain functions, such as synaptogenesis, neurotransmission, and myelin maintenance, some of which are closely linked to the pathology of Alzheimer's disease, Parkinson's disease, and other neurodegenerative disorders. Furthermore, using Thy-1 GFP M line transgenic mice, in which GFP is expressed in hippocampal neurons, we validated the neural origin of APLP1 by demonstrating colocalization of APLP1 and GFP in plasma EVs. Collectively, these findings suggest that APLP1+ EVs are not merely diagnostic markers but potential key tools for the early diagnosis and mechanistic elucidation of neurodegenerative diseases. In particular, our integrated proteomic and small RNA analysis strategy provides a promising foundation for the development of brain-derived EV-based precision diagnostic platforms.
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PS-F004

Isolation Strategies for Proteomic Analysis of Neuron-Derived Extracellular Vesicles from Human Blood

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Extracellular vesicles are lipid bilayer-enclosed vesicles released from all cells and circulating in the blood. These vesicles carry diverse molecular cargo and play an important role in the inter-cellular communication. Notably, neuron-derived extracellular vesicles (NDEVs), released specifically from brain neuronal cells, can cross the blood-brain barrier and have emerged as key biomarkers in neurodegenerative diseases like Alzheimer's disease. However, cleary isolating the neuron-derived extracellular vesicles (NDEVs) present in blood and comprehensively analyzing EV proteome remains challenging. Furthermore, current studies on NDEV have relied on enzyme-linked immunosorbent assay or transcriptomics. For in-depth and comprehensive understanding of the proteins in NDEVs from limited amount of human blood via mass spectrometry (MS)-based proteomic approach, we firstly evaluated and compared three exosome isolation methods (ultracentrifugation, ExoQuick polymer-based precipitation, and EVtrap magnetic bead precipitation). Subsequently, NDEVs were specifically enriched by immunoprecipitation targeting the neuronal cell adhesion molecule (L1CAM). The isolated NDEVs were characterized by nanoparticle tracking analysis (NTA) to assess size and concentration and validated by western blotting using antibodies against L1CAM and the canonical exosome markers. Subsequently, Proteins extracted from NDEVs were digested with Trypsin/LysC enzymes and peptides were analyzed by the Orbitrap Exploris 480 instrument employing data-independent acquisition mode. The resulting MS raw data underwent analysis via Spectronaut software. Firstly, we compared different exosome isolation methods and identified that the polymer-based method yields the highest exosome particle number from minimal blood volume. Also, NTA assay of NDEVs isolated from exosomes by polymer-based method showed that 100 to 140 nm size aligning with the expected exosome size, and the expression of exosome markers such as CD9, CD63, and CD81 and L1CAM were confirmed by western blotting. These results indicate and validate the robust enrichment of NDEVs from peripheral blood for subsequent proteomic analysis. And then, in-depth proteome analysis of NDEVs led to the quantification of about 400 proteins and most proteins were included in ExoCarta and Vesiclepedia database. Furthermore, peptides corresponding to the transmembrane domain or intracellular regions of L1CAM, as well as the neural cell-specific marker CD56 (NCAM1), were identified by MS analysis. These results showed that highly effective isolation and comprehensive MS-based proteomic profiling of NDEVs isolated from human blood. Collectively, we compared isolation strategies for overall proteome profiling of NDEV from human blood, and MS-based NDEV analyses could be an important tool for investigating the pathogenesis of neurodegenerative and psychiatric disorders.

Poster Session F. Exosome

PS-F005

Multi-Omics Analysis for Characterization of Extracellular Vesicle

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Extracellular vesicles (EVs), or more frequently known as exosomes, are membrane-surrounded vesicles released by numerous cell types into the extracellular microenvironment. EVs contain multiple classes of molecules, including nucleic acids, proteins, and lipids. EVs are involved in cell-cell communication, coagulation, inflammation, immune response modulation, and disease progression. EV have recently emerged as novel therapeutics in various clinical applications. Despite the growing number of evidences demonstrating that EVs are a crucial mediator of intercellular communication, challenges to commercialize EVs in clinical applications remain largely unresolved. Here we report the multi-omics characterization of vesicles included lipidomic, proteomic, and small RNA assessments. Our multi-omics platform with Liquid chromatography-mass spectrometry (LC-MS/MS) can be used in a number of ways in the quality control (QC) of EVs as a new therapeutic and carrier.

PS-F006

Serum Exosomal Proteomics Identifies Biomarkers for Surgical Response and Recurrence Risk in Pancreatic Cancer

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Pancreatic cancer is one of the most lethal malignancies, with most cases diagnosed at an advanced stage. Early detection and accurate recurrence prediction are essential for improving clinical outcomes. However, current diagnostic tools—such as imaging and biopsy—are invasive and often insufficient for detecting early-stage or minimal residual disease. Reliable biomarkers for recurrence monitoring are also lacking in clinical practice. Circulating exosomes are a promising, minimally invasive biomarker source that encapsulates molecular signatures reflective of the tumor's physiological and pathological state.

In this study, we aimed to identify serum-derived exosomal protein biomarkers associated with surgical intervention. Our goal was to support early diagnosis and recurrence risk stratification in pancreatic cancer. Exosomes were isolated from paired pre- and post-operative serum samples of pancreatic cancer patients and subjected to mass spectrometry-based proteomic profiling using label free quantitation. Exosomes were isolated from paired pre- and post-operative serum samples and analyzed using label-free LC-MS/MS proteomics, identifying 78 differentially expressed proteins (DEPs). Multivariate analyses including PLS-DA, volcano plots, and hierarchical clustering revealed clear separation between surgical states. ROC curve and boxplot validation further demonstrated the robust predictive performance of selected proteins, with AUC values approaching 1.0. Functional enrichment indicated pathways related to immune regulation, extracellular matrix remodeling, and inflammatory responses. For recurrence risk, separate analyses of pre- and post-operative samples identified 64 and 18 DEPs, respectively, with representative proteins showing high discriminatory ability (AUC up to 1.0 in pre-operative and 0.99 in post-operative groups).

Although further validation in larger patient cohorts is required to overcome overfitting issues in model performance, this study highlights the clinical applicability of exosome-based proteomic profiling for monitoring surgical outcomes and predicting recurrence in pancreatic cancer. Given the current limitation in sample size, we are actively expanding the cohort for validation. These efforts aim to support precision oncology and guide more personalized therapeutic decisions for patients with this aggressive disease.

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Poster Session F. Exosome

PS-F007

Understanding Molecular Characteristics of Extracellular Vesicles and EV-Depleted Secretome Fraction Derived from Different Types of Thyroid Cancer Cell Lines

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Thyroid cancer is one of the most common endocrine malignancies derived from the epithelial cells of the thyroid gland, and its incidence is increasing worldwide. Papillary Thyroid Carcinoma (PTC) is the most prevalent subtype and has a favorable prognosis, whereas Anaplastic Thyroid Carcinoma (ATC) is rare but highly aggressive. The tumor microenvironment (TME) is a complex ecosystem composed of tumor cells and non-malignant cells such as immune cells and stromal cells. PTC and ATC induce non-malignant cells within the TME to exhibit distinct phenotypes. For instance, recent study has reported that immune cells in ATC display a more immunosuppresive phenotypes. Within the TME, tumor cells secrete a variety of molecules to communicate with surrounding cells, among which secretome—including extracellular vesicles (EVs) and soluble protein—have been reported to to promote tumor survival and immune suppression. Due to their highly diverse molecular composition, single-molecule analyses are insufficient to capture their full functional landscape. An omics-based approach enables comprehensive profiling and reveals complex signaling networks and interactions. We therefore hypothesized that differences in secretory molecules drive the variations in phenotypes of TME cells across thyroid cancer subtypes and aimed to investigate this using mass spectrometry-based proteomics. To explore molecular differences in secretory molecules between PTC and ATC, we performed a comparative proteomic analysis of two secretome fractions—EVs and EV-depleted secretome fraction(SF)—isolated from the PTC cell line TPC-1, ATC cell line Cal-62 and normal thyroid epithelial cell line Htori-3. TPC-1 and Cal-62 were starved in serum-free media for 24 and 48 hours, whereas Htori-3 was starved for 18 hours to obtain the conditioned media (CM). Crude EVs were isolated by ultracentrifugation (UC), and the remaining CM was then subjected to ultrafiltration followed by acetone precipitation to isolate the SF. Subsequently, all samples were digested via S-trap digestion and the peptides were analyzed by data-independent acquisition-based label-free quantification using Q Exactive HF-X mass spectrometer. Most of the main EV markers were detected in the EV samples by western blotting, and the particle size was confirmed to be within the EV range using Nanoparticle Tracking analysis. Proteomic analysis identified approximately 1,300 proteins in EVs and 2,400 proteins in the SF, and ANOVA-based differential analysis revealed distinct secretion patterns. Regardless of starvation time, differentially expressed proteins (DEPs) were identified in thyroid cancer cell lines compared with normal thyroid cell line, and unique DEPs were also detected in TPC-1 and Cal-62 cell lines. Gene ontology and KEGG pathway analyses were performed on these DEPs, revealing that their characteristics differ between cancer subtype. STRING database-based protein-protein interaction (PPI) analysis further revealed that the interaction networks of EVs and SF proteins are distinct between TPC-1 and Cal-62, highlighting subtype-specific functional modules. For the EVs, additional TMHMM anlysis was conducted to determine whether the proteins were localized to the cargo or the membrane. Our study suggests that EVs and SF derived from thyroid cancer cells exhibit distinct molecular characteristics compared with those from normal thyroid cells, and that difference between PTC and ATC indicate the presence of subtype-specific secretory features. These molecular variations in secretory components may underlie subtype-dependent heterogeneity within the TME.

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Poster Session G. Single-cell and Spatial Omics

PS-G001

Single-Cell Mass Cytometry Reveals Cell Type- and Cluster-Specific Heterogeneity in Silver Nanoparticle Responses in a 3D Alveolar

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Silver nanoparticles (AgNPs) are widely used in medicine, environmental science, and industry. However, their heterogeneous interactions with complex biological systems, especially at the single-cell level, are not fully understood yet. Conventional toxicity assessment methods are typically conducted on oversimplified in vitro models that fail to replicate actual physiological conditions, and measure collective responses across cell populations, obscuring differences among individual cells. To overcome these limitations, we utilized single-cell mass cytometry (CyTOF) to investigate individual cell responses to AgNP-induced stress, combined with a 3D alveolar tetra-culture model designed to better reflect the complexity of biological systems. Single-cell mass cytometry of a 3D alveolar model revealed heterogeneous, cell type-specific responses to AgNP exposure. Specifically, PMA-differentiated THP-1, A549 and EA.hy926 cells exhibited high AgNP association but limited cytotoxicity, indicating activation of stress-mitigation pathways, while THP-1 cells showed early inflammatory activation despite minimal AgNP association, suggesting an indirect mechanism. Single-cell analysis and FlowSOM clustering revealed distinct subpopulations exhibiting diverse intracellular signaling profiles of inflammatory cytokines, anti-inflammatory mediators, and stress-response proteins, which unveiled common cellular responses and unique cell-type specific pathways determining cell fate (survival, transitional states, or apoptosis) upon AgNP exposure. This study introduces a novel framework for studying heterogeneous interactions of nanoparticles with complex biological systems by integrating a 3D alveolar tetra-culture model with single-cell mass cytometry analysis, enabling the dissection of nanoparticle-induced stress responses at an unprecedented level of detail. These insights have broad implications for nanotoxicology and nanomedicine, underscoring the need to account for cellular heterogeneity when evaluating nanoparticle-induced toxicity.

PS-G002

Single-Cell Multi-Omic Analysis Reveals Divergent Characteristics of Different Cell Types in Triple-Negative Breast Cancer

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Triple-negative breast cancer (TNBC) accounts for approximately 20% of all breast cancer cases and has shown a steady increase in mortality due to its high metastatic potential and lack of targeted therapies. TNBC exhibits cancer stem cell (CSC)-like characteristics at functional, molecular, and transcriptional levels, identified by markers such as CD24, CD44, CD133, and ALDH1. Recently, spatial multi-omics technologies have emerged as powerful tools for high-resolution and unbiased characterization at the single-cell level.
Our study used CSC markers to isolate regions of interest (ROIs) from immunofluorescence-stained TNBC tissue, captured by spatially resolved laser-activated cell sorting (SLACS). $ALDH1^+$, $CD44^+$, $ALDH1^+$ /$CD44^+$, and $ALDH1^-$ /$CD44^-$ cell populations were isolated and analyzed for proteomic and transcriptomic profiles at the single-cell level. A total of 6,523 proteins and 36,978 genes were identified across ROIs from four groups, followed by bioinformatic analyses to identify differentially expressed proteins and genes. Furthermore, we also performed integrated interpretation using proteome-transcriptome co-profiling.
In conclusion, the integration of SLACS with mass spectrometry represents a robust and precise strategy for the spatial analysis of ROIs in tissue. These approaches enhance our understanding of the molecular mechanisms underlying tumor complexity and offer valuable insights for developing subtype-specific preventive and therapeutic strategies.

Poster Session G. Single-cell and Spatial Omics

PS-G003

Mass Spectrometry-Based Proteomic Analysis of LMD Monkey Brain Tissue Using One-Pot Digestion Method

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Spatial proteomics (SP) has emerged as a powerful approach for investigating mechanisms of health and disease, providing unprecedented insights into the spatial organization of proteins within cells and tissues. As a rapidly advancing field, SP has garnered significant attention. Ultrasensitive mass spectrometry provide the enabled multiscale spatial proteomics, integrating complementary imaging techniques with exploratory mass spectrometry approaches to achieve highly detailed spatial protein analysis.
In this study, to ensure compatibility with low-input samples, we confirmed the reproducibility and data reliability of an one-pot digestion workflow using nanogram-scale proteome sample preparation and LC-MS/MS systestm consisting of an Ultimate 3000RSLnano System and Orbitrap Fusion Lumos Tribrid mass spectrometer. And then, this protocol was appiled to monkey brain tissue spatially sectioned via laser microdissection (LMD) and the resulting proteome smaples were analyzed using LC-MS/MS systestm achieving high sensitivity and consistent quantification from minimal sample input.

PS-G004

Molecular Heterogeneity and Prognostic Biomarker Discovery in Gastric Neuroendocrine Neoplasms through Spatial Proteomics

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Gastric neuroendocrine neoplasms (GNENs), which encompass neuroendocrine tumors (NETs) and neuroendocrine carcinomas (NECs), are rare malignancies that arise from neuroendocrine precursor cells. Although genomic studies have advanced our understanding of these tumors, the protein-level mechanisms underlying their biology are still poorly understood. To address this gap, we performed spatial proteomic profiling on formalin-fixed, paraffin-embedded (FFPE) tissue slides from 20 NEC and 24 NET specimens. Using the Vanquish Neo system coupled with the Orbitrap Exploris™ 480 mass spectrometer, we applied data-dependent acquisition (DDA) to identify and quantify differentially expressed proteins (DEPs) between tumor tissues and adjacent normal tissues. Our analysis revealed distinct DEPs that discriminated NECs from NETs, as well as unique protein alterations between tumor and normal counterparts. These findings highlight protein signatures potentially associated with the aggressive biological behavior of NECs and identify novel prognostic biomarkers for GNENs. This study represents the first application of spatial proteomics to GNENs, uncovering molecular heterogeneity at the protein level. The results provide a foundation for biomarker validation and the development of future targeted therapeutic strategies.

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Poster Session G. Single-cell and Spatial Omics

PS-G005

Identification of Inflammation-Associated Proteomic Markers in Gastric Neuroendocrine Carcinoma Using LC-MS/MS and Machine Learning

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Gastric neuroendocrine carcinomas (GNECs) are highly heterogeneous tumors in which the inflammatory status of the tumor microenvironment plays a critical role in prognosis and treatment response. However, proteomic alterations distinguishing inflamed from non-inflamed phenotypes remain insufficiently understood. We conducted spatial proteomic profiling with label-free LC-MS/MS on 30 tumor sections from 20 patients, classified as inflamed (n = 20) or non-inflamed (n = 10). Proteins demonstrating statistically significant associations with overall survival were advanced for subsequent evaluation. The dataset was divided into training (70%) and validation (30%) cohorts, and class imbalance in the training set was corrected using SMOTE. Four machine learning approaches—logistic regression, support vector machine, random forest, and XGBoost—were subsequently evaluated for their ability to classify tumor phenotypes. Model performance was assessed using accuracy, precision, recall, F1 score, and AUC-ROC, and robustness was further confirmed in the validation set. A subset of proteins was identified as reliably distinguishing between the two inflammatory phenotypes. At the same time, unsupervised spatial clustering revealed pronounced molecular differences between inflamed and non-inflamed tumors, providing further biological support for the computational analyses. In summary, our findings show that integrating LC-MS/MS proteomic profiling with machine learning provides novel insights into the biological complexity of gastric neuroendocrine carcinomas. The protein signatures uncovered here may serve as prognostic indicators and could inform the design of individualized therapeutic approaches.

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PS-G006

Manipulation of Single Living Cell's Fate Using on-Demand and Spatioselective Gene-Delivering Nanowire

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Recently, we have witnessed dramatic advancements in genetic engineering, allowing us to edit genetic functions of cells either temporarily or permanently. However, current genetic manipulations, which are mainly conducted at the bulk-level rather than the single-level, often result in heterogeneous gene uptake and obscure single-cell-level insights. Here, we introduce a nanowire waveguide-based platform for ultrafast, site-specific on-demand gene delivery into individual living cells while minimizing structural and biochemical disruption. A polymeric nanowire (~400 nm in diameter), chemically and mechanically functionalized with high-density genetic cargos via photocleavable linkage, can be spatiotemporally positioned within a target cell. Upon transmission of the light at the wavelength of linkage cleavage to the nanowire through a coupled optical fiber, genetic cargos are rapidly released (< 10 s) at precisely defined locations within a living cell via a confined UV evanescent field, thereby achieving highly efficient genetic manipulation of a target living cell.

Poster Session G. Single-cell and Spatial Omics

PS-G007

Comparative Single-Cell Proteomics Revealed Molecular Alterations of in Vitro and in Vivo Oocyte Aging

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Oocyte aging is characterized by a decline in oocyte quality, which in turn represent a major cause of female infertility. However, the molecular mechanisms underlying oocyte aging and their impact on oocyte competence remain poorly understood, in part due to the heterogeneous nature of single oocytes. To address this, we established a robust single-cell proteomics (SCP) workflow and applied it to compare the proteome of fresh, in vitro-aged (post-ovulatory aged, POA), and in vivo-aged (chronologically old) mouse oocytes. Our SCP identified a total of 6,423 proteins covering a dynamic range of seven orders of magnitude and the overall coefficient of variation (CV) for protein identifications was approximately 3%, demonstrating the reliability of the proteome data obtained by our SCP workflow. Principal component analysis separated samples into three distinct groups. Interestingly, consensus clustering further partitioned the in vivo-aged group into two subpopulations, one of which clustered with the fresh group. These findings indicate that oocytes with the same chronological age can differ in biological age, underscoring the capacity of SCP to resolve intercellular heterogeneity. To determine whether biological processes underlying oocyte aging differ between in vitro and in vivo conditions, we conducted Gene Ontology (GO) term enrichment analysis of proteins exclusively downregulated in each aging condition. Mitochondria proteins were predominantly downregulated in the in vitro-aged group, supporting the concept that POA compromises mitochondrial pathways. In the in vivo-aged group (the subpopulation not clustering with fresh), proteins enriched in spindle assembly and microtubule-based processes were dramatically downregulated, indicating heterogeneous biological aging trajectories between the two aging conditions. In conclusion, our study established a robust SCP workflow that enables deep profiling of single-oocyte proteome to elucidate intercellular heterogeneity. Furthermore, our data identified candidate targets that may influence oocyte quality in both in vitro and in vivo aging and revealed distinct biological differences between the two conditions.

PS-G008

Proteomic Reprogramming and Immune Remodeling Drive Endometrial Cancer Aggressiveness

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Endometrial cancer (EC) is the most common gynecologic malignancy, with rising incidence and mortality. To understand drivers of tumor aggressiveness, we conducted integrative proteomic and immune profiling of two genetically engineered mouse models: mild (MC; Pgrcre/+Ptenf/f) and aggressive (AC; Pgrcre/+Ptenf/fMig-6f/f). FFPE tumor tissues were processed using adaptive focused acoustics and analyzed via high-resolution LC-MS/MS with DIA-PASEF. Over 6,400 proteins were quantified, with 285 differentially expressed between AC and MC. AC tumors showed enrichment in extracellular matrix remodeling, EMT, and inflammatory pathways, while MC retained immune-related signatures. Immune deconvolution revealed immunosuppressive cell infiltration in AC and pro-inflammatory immune subsets in MC. Cross-species comparison with human EC datasets identified 47 conserved differentially expressed proteins associated with tumor progression. These findings highlight molecular and immune mechanisms underpinning EC invasiveness and support the relevance of murine models for translational biomarker discovery and therapeutic development.

Poster Session G. Single-cell and Spatial Omics

PS-G009

Comparison of Data Analysis Pipelines for Single-Cell Proteomics

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Single cell proteomics(SCP) is emerging as a critical approach in biological research by overcoming the limitations of bulk proteomics, which averages protein signals across thousands of cells and masks cellular heterogeneity. However, SCP presents significant analytical challenges due to the extremely limited protein content within individual cells, the enormous dynamic range of protein expression levels, and the inability to amplify proteins. These technical limitations result in difficulties for accurate protein detection and quantification, while conventional bulk proteomics computational frameworks are not well-suited for the characteristically low-signal and sparse datasets generated by SCP experiments. In response, several specialized SCP tools such as SCeptre, SCoPE2, scplainer have been developed, employing different strategies including reference-based normalization, KNN averaging, and linear modeling. However, these approaches can yield different biological interpretations, and no standardized SCP pipeline has been established to date. To address these multifaceted challenges, we have pursued comprehensive advancements across the entire analytical pipeline, including implementation of robust normalization approaches for data interpretation, imputation methods to handle missing values, and strategies to mitigate batch effects. We compared the analysis workflows of SCoPE2, SCeptre, and scplainer using a public TMT-labeled DDA dataset and a label-free DDA/DIA dataset, evaluating cell type consistency, protein quantification reproducibility, clustering patterns, PCA organization, and the extent of residual batch effects. In conclusion, this comparison of computational pipeline strengths and limitations provided valuable guidance for selecting appropriate pipelines based on dataset-specific characteristics in SCP data analysis.

Poster Session H. Glycosylation and PTMs

PS-H001

Implementing N-Terminomics and Machine Learning to Probe in Vivo Nt-Arginylation

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N-terminal arginylation (Nt-arginylation) is a degradation signal in the ubiquitin-proteasome and autophagy-lysosomal pathways, but its study has been limited by technical challenges. Here, we developed an integrated approach combining N-terminomics with machine learning-based filtering to identify in vivo Nt-arginylation. By using Arg-starting missed cleavage peptides as proxies for ATE1-mediated arginylation, we trained a transfer learning model to predict MS2 spectra and retention times. By applying the prediction models with an additional statistical filter, we identified 134 Nt-arginylation sites in thapsigargin-treated HeLa cells. Arginylation was enriched in proteins from various organelles, especially at caspase cleavage and signal peptide processing sites. Several proteins were further validated for their interaction with p62 ZZ domain. Temporal profiling revealed that ATF4 increased early post-stress, followed by arginylation at caspase-3 substrates and ER signal-cleaved proteins. Our approach enables sensitive detection of rare N-terminal modifications, offering potential for biomarker and drug target discovery.

PS-H002

Utilizing Mass Errors of Fragment Ions to Distinguish Near-Isobaric N-terminal Modifications

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Alpha-N-terminal methylation is an understudied post-translational modification involved in protein-protein or protein-DNA interactions. Its global profiling by mass spectrometry is challenging due to low abundance and interference from near-isobaric modifications like Nt-acetylation, even after N-terminome enrichment. To address this problem, we assume that a, b- and y-ions will exhibit different mass error distributions in MS2 spectra if falsely assigned to a near-isobaric Nt-modification. We exploit this statistically to correct the Nt-modification, a procedure we name as mass error test (MET). We confirmed effectiveness of MET by manual inspection of chemically methylated BSA peptides. MET was further confirmed by comparing a,b-ion proportion and predicted retention time between Nt-methylation and Nt-acetylation in chemically modified cell lysates. We apply MET to potentially Nt-methylated spectra from a repurposed dataset and reassign correct Nt-modification. By implementing MET to HCT116 N-terminome, we were able to reassign Nt-modified PSMs with net change of ~17.1% reduction in falsely assigned Nt-trimethyl PSMs. These results indicate that MET is a useful tool for detection of Nt-methylated proteins in complex proteomes.

Poster Session H. Glycosylation and PTMs

PS-H003

Identification of 0-Series Gangliosides as a Distinct Cerebellar Signature by LC-MS/MS

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Gangliosides, essential glycosphingolipids in the nervous system, play critical roles in cellular signaling, synaptic plasticity, and recognition. Their biosynthesis is tightly linked to neuronal maturation and synapogenesis, emphasizing their importance in brain function. Traditionally, brain gangliosides have been described in terms of the a- and b-series pathways, which dominate the ganglioside composition of the brain. However, structural complexity introduced by glycan isomers and ceramide heterogeneity suggests that additional biosynthetic diversity may exist. In this study, we applied our established high-resolution LC-MS/MS platform to profile ganglioside expression across five brain regions in mouse, rat, and monkey models. Comparative analysis revealed a striking cerebellum-specific biosynthetic pathway not accounted for by the canonical a- and b-series. Using diagnostic ions (e.g., m/z 292.10 for NeuAc; m/z 657.22 for GalGalNAcNeuAc), along with isomer-specific separation, we clearly discriminated GD1a, GD1b, and GD1c. Importantly, we identified robust accumulation of the 0-series gangliosides in the cerebellum, a pathway absent in other brain regions and not previously characterized in the brain. The selective detection of GD1c, confiremed by its characteristic diagnostic ion at m/z 948.33, further highlighted this unique cerebella signature. These findings represent the first characterization of the 0-series gangliosides in the cerebellum, extending the conventional framework of brain ganglioside biosynthesis. This region-specific discovery opens new avenues for investigating how divergent ganglioside pathways contribute to neural function and cerebellar specialization.

PS-H004

N-Glycan-Dependent ER Quality Control Modulates Extracellular Vesicle Biogenesis and Virulence Factor Export in Cryptococcus Neoformans

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The endoplasmic reticulum quality control (ERQC) system is essential for proper folding of N-glycosylated proteins and proteostasis in eukaryotic cells. We investigated how disruption of ERQC affects both canonical protein secretion and extracellular vesicle (EV)-mediated export in the human pathogen Cryptococcus neoformans. We focused on mutants lacking Ugg1, a misfolded glycoprotein sensor, and the α1,2-mannosidases Mns1 and Mns101, core components of a conserved yet evolutionarily tailored N-glycan-dependent ERQC pathway. These mutants exhibited defective N-glycosylation patterns, compromised cell surface integrity, and impaired secretion of key virulence factors, including capsule polysaccharides, urease and laccase. Transcriptomic analysis of the ugg1⊿ mutant revealed induction of ER stress-responsive genes without major changes in classical secretion or capsule biosynthesis pathways. Notably, ugg1⊿ cells displayed pronounced defects in EV biogenesis, including reduced vesicle yield and altered size distribution. Comparative proteomics profiling revealed alterations in EV cargo composition in ugg1⊿, compared to the overall cellular proteome. Complementary lipidomic analysis if the ugg1⊿ mutant showed altered lipid profiles, suggesting disrupted membrane organization as a contributing factor to defective EV biogenesis. Together, these findings establish ERQC as a central regulator of EV biogenesis and protein cargo loading processes essential for the secretion of virulence-related molecules, providing new insights into how ERQC dysfunction can diminish the pathogenic potential of C. neoformans.

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Poster Session H. Glycosylation and PTMs

PS-H005

A Machine Learning Model for Site-Specific Classification of N-Glycoprotein Fucosylation Using Tandem Mass Spectrometry and Deep Neural Network

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Protein fucosylation is a key post-translational modification affecting protein structure, stability, and interactions. N-glycopeptide complexity arises from various combinations of HexNAc, Hex, Fuc, and Sia. Fucosylation is classified into core and outer types, both linked to cancer, immune responses, and protein regulation, requiring precise structural analysis. This study presents a method to classify N-glycopeptide fucosylation into none, core, outer, and dual types using deep neural networks (DNN) and support vector machines (SVM). To classify fucosylation types, we selected training and test sets from over 1,320 N-glycopeptide MS/MS spectra derived from immunoglobulin G (IgG) and alpha-1-acid glycoprotein (AGP). The N-glycopeptide MS/MS spectra were identified characteristic fragment ions of N-glycopeptides, and peak m/z and intensity values were applied to machine learning models. Various hyperparameters were tested to optimize performance, and the model was validated on human plasma samples to classify fucosylated N-glycopeptides. DNN and SVM approaches accurately predicted fucosylation types in complex plasma samples, demonstrating the effectiveness of combining machine learning with MS/MS analysis.

PS-H006

Characterization of Site-Specific N- and O-Glycopeptides from Recombinant Spike and ACE2 Glycoproteins Using LC-MS/MS Analysis

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The COVID-19 pandemic, driven by SARS-CoV-2, continues to threaten global health as new variants emerge. The viral spike (S) protein and its human receptor ACE2 are heavily glycosylated, and these glycans modulate folding, receptor binding and immune recognition, making site-resolved glycoproteomic mapping essential. Here we profiled N- and O-glycosylation of recombinant spike RBD and ACE2 produced in Expi293F cells, and the S2 subunit expressed in Nicotiana benthamiana. Using an Orbitrap Eclipse Tribrid MS coupled to Ultimate 3000 RSLCnano and our Integrated GlycoProteome Analyzer (I-GPA), we identified 148 N- and 28 O-glycopeptides on RBD, 71 N-glycopeptides on S2, and 139 N-glycopeptides on ACE2. Novel PTMs—mannose-6-phosphate and GlcNAc-1-phosphate-6-O-mannose on RBD and ACE2 N-glycans, and O-acetylation on RBD O-glycans—were detected for the first time. Quantitative site-specific analysis revealed dominant occupancy at Asn331 (RBD), Asn1098 (S2) and Asn103 (ACE2); complex glycans prevailed on RBD and ACE2, whereas S2 was enriched in high-mannose structures. These data provide a high-resolution atlas of SARS-CoV-2 spike and ACE2 glycosylation, informing vaccine design and antibody- or lectin-based therapeutics.

Poster Session H. Glycosylation and PTMs

PS-H007

Modulation of Keratinocyte N-Glycosylation by Atopic Induction and the Functional Cosmetic Lipimoide

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Atopic dermatitis (AD) is a chronic skin disease characterized by barrier dysfunction, immune imbalance, and persistent inflammation, yet therapeutic efficacy is still primarily assessed by clinical observations, lacking objective molecular biomarkers. Glycans on keratinocytes play critical roles in signaling and immune regulation, and their alterations in AD may provide important insights into disease mechanisms and treatment effects. In this study, we investigated keratinocyte and disease-model cell lines to examine the molecular effects of the functional cosmetic Lipimoide (LM) on atopic induction and to explore glycan-based efficacy indicators. Four groups were prepared: Control (Con), Atopic induction (AD), LM treatment (LM), and Atopic induction with Lipimoide treatment (AD+LM). For each group, approximately 1×106 cells were used to isolate cell membranes, from which N-glycans were extracted. To enhance sensitivity, glycans were derivatized by Instant PC labeling and subsequently analyzed by UHPLC/Q-TOF-MS. Based on an in-house library, about 80 N-glycan compositions were identified across all groups. Clustering analysis revealed distinct group separation. In particular, the Con and LM groups showed similar patterns, whereas the AD and AD+LM groups clustered independently. In the analysis by glycan types, sialylated complex/hybrid (CH-S) glycans were specifically increased in AD group compared with Con, but decreased upon LM treatment (AD+LM), indicating a recovery effect. Further narrowing the analysis identified bi-antennary Neu5Ac-containing complex N-glycans as candidate biomarkers that reflect this recovery trend. To quantify the effect, we introduced a Rescue Index (R.I.), which confirmed that LM treatment attenuates abnormal glycosylation in atopic keratinocytes. Notably, several glycans exhibited R.I. values ≤ 1 (e.g., 0.79, 0.38, 0.41), indicating recovery levels comparable to the normal state. These findings demonstrate that Lipimoide ameliorates glycosylation abnormalities in AD models and highlight bi-antennary Neu5Ac-containing complex N-glycans as potential structural biomarkers for evaluating the efficacy of functional cosmetics.

PS-H008

Tyrosine Phosphorylation of Mitochondrial Creatine Kinase Protects the Heart from Ischemic Injury

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Heart failure due to ischemic cardiomyopathy (ICM) is considered as one of the main causes of cardiovascular disease-related deaths worldwide. Patient mortality due to ICM still remains high. Ischemic preconditioning (IPC) has been found as an effective mitigator of ICM. This is done by short-time ischemia is applied before ischemia/reperfusion injury (I/R). The brief time applied for IPC may contribute to a rapid change in protein expression and regulation wherein protein function modulation by post-translational modifications is important. Mitochondria play a significant role in heart disease progression and so, it is a good target for ICM treatment. In this study, we focused on mitochondrial creatine kinase (CKMT2) under I/R injury. Ex vivo Langendorff system on Sprague-Dawley rat hearts were used to simulate normal perfusion, I/R, and IPC condition and used for phosphoproteomic analyses. In vitro study using human cardiomyocyte AC16 cells were used to determine the cardioprotective role of mitochondrial creatine kinase through overexpression and how CKMT2 site-directed mutagenesis can affect cardioprotection by CKMT2 protein activity, mitochondrial function, and protein expression. CKMT2 was dephosphorylated during ischemia and I/R but remained phosphorylated under IPC conditions. CKMT2 overexpression show increased cell viability and mitochondrial ATP level against hypoxia/reoxygenation confirming the cardioprotective effect of CKMT2. Conversely, there was decreased cell viability and increased ROS production during H/R when CKMT2 is phosphomutated, specifically in Y368. We also confirmed increased mitochondrial function via the proliferator-activated receptor γ coactivator-1α/estrogen-related receptor-α pathway during CKMT2 overexpression. CKMT2 regulation and phosphorylation may be used for future ICM therapeutics.

Poster Session H. Glycosylation and PTMs

PS-H009

Phosphorylation of MDH2 at S246 Confers Cardioprotection During Hypoxia/Reoxygenation

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Mitochondrial malate dehydrogenase (MDH2) regulates the conversion of malate/NAD+ to oxaloacetate/NADH. Proteomic studies show that MDH2 is involved in cardiovascular diseases, wherein the expression of certain mitochondrial proteins was altered under ischemia/reperfusion conditions. Screening of phosphorylation sites of MDH2 revealed several posttranslational modifications occurring in the protein. However, it is still unclear how modifications of these sites lead to the altered activity and function of the system under pathological models. This study focuses on the importance of MDH2 phosphorylation and how it regulates mitochondrial biogenesis and mitochondrial function. Phosphorylation occurs at various conserved sites, specifically Y56, Y80, Y161, and S246. Phosphorylation mutants based on these sites were manufactured using expression vectors and transfected into cellular models for analysis. Phosphomutants decreased cell viability, ATP production, and MDH2 activity. Mitochondrial biogenesis marker expressions were also altered by phosphomutants under hypoxia/reoxygenation conditions. The protective mechanism that MDH2 confers cardioprotection remains unclear. Further studies on the role of MDH2 phosphorylation in mitochondrial biogenesis can elucidate the importance of MDH2 in cardiovascular disease models and treatment modalities.

PS-H010

Rapid Phosphopeptide Enrichment and Fractionation for High Efficiency Phosphoproteomics

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Phosphorylation is a key post-translational modification (PTM) that regulates various biological processes such as signal transduction, cell cycle progression, stress response, and metabolism. Due to its dynamic nature, phosphorylation is closely associated with diseases including cancer and neurological disorders. As a result, phosphoproteomics has become a powerful tool for deciphering signaling pathways and disease mechanisms. However, the inherently low abundance and poor ionization efficiency of phosphopeptides make mass spectrometry (MS)-based detection challenging and necessitating effective enrichment strategies.
Conventional approaches to enhance analytical depth have relied on offline HPLC-based fractionation or online 2D-LC systems. While these methods provide high resolution and deep proteome coverage, they require specialized instrumentation such as multidimensional HPLC setups and often involve complex workflows.
In this study, we developed a simple phosphopeptide enrichment workflow tailored for low-input samples. This method integrates IMAC-based phosphopeptide enrichment with stepwise acetonitrile (ACN) gradient fractionation (10%, 25%, 50%) during the desalting step, enabling both enrichment and fractionation without the need for additional instrumentation.
We applied this workflow to both TMT-labeled and label-free samples and evaluated its performance across various input amounts. The results demonstrated high reproducibility and effective enrichment, supporting the utility of this approach for phosphoproteomic analysis of low-abundance samples.

Poster Session H. Glycosylation and PTMs

PS-H011

Quantitative Phosphoproteomic Profiling Reveals Subtype-Specific Signaling Signatures in Thyroid Cancer

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Thyroid cancer subtypes, including papillary (PTC), follicular (FTC), and anaplastic (ATC) carcinomas, exhibit marked differences in molecular characteristics and clinical aggressiveness.

Protein phosphorylation is a key post-translational regulatory mechanism governing cell cycle progression, cytoskeletal organization, and stress responses, and its dysregulation is a well-recognized hallmark of tumorigenesis. Phosphoproteomic analysis offers a direct readout of signaling pathway activity, enabling the identification of active kinases and regulatory nodes that are not apparent from genomic or transcriptomic data alnoe. Characterizing subtype-specific phosphorylation patterns is therefore crucial to elucidate the signaling networks that drive tumor heterogeneity and progression.

Five thyroid-derived cell lines were analyzed : Htroi (Normal), B-CPAP (PTC), Uhth-7 (ATC), CAL-62 (ATC), and FTC-133 (FTC). Cell pellets were lysed in 4% SDS buffer. Proteins were digested using S-trap method with trypsin. Peptides were desalted with Sep-Pak C18 cartridges, labeled with TMTpro 16-plex reagents, and pooled in equal amounts after confirming labeling efficiency. Phosphopeptides were enriched using Zr-IMAC HP magnetic beads and fractionated by high-pH reversed-phase stagetip into six fractions. Each fraction was analyzed on an Orbitrap Exploris 480 mass spectrometer operating in data-dependent acquisition (DDA) mode. Raw data were processed with Fragpipe (v.23) using the Uniprot human reference proteome as the database.

TMT-based quantitative phosphoproteomic profiling identified 22,179 modified peptides, including 12,735 phosphopeptides mapping to 12,132 unique phosphosites. Among these, 8, 868 class Ⅰ sites (localization probability ≥ 0.75) were selected for downstream analyses. Principal component analysis revealed clear separation between normal and cancer subtypes with tightly clustered replicates, confirming high data reproducibility. Differential expression and ANOVA defined numerous regulated phosphosites and robust subtype-specific signatures. Kinase-substrate enrichment and motif analyses uncovered distinct regulatory patterns among subtypes. Functional enrichment demonstrated a gradual shift in signaling, from cytoskeletal remodeling in PTC to extracellular matrix reorganization and metabolic adaptation in FTC, and to enhanced motility, angiogenesis, and rapid proliferation in ATC, suggesting a stepwise intensification of oncogenic signaling.

This study provides a comprehensive phosphorylation map across thyroid cancer subtypes, highlighting distinct patterns of signaling network utilization. These results constitute a valuable resource for identifying subtype-specific biomarkers and potential therapeutic targets.

Poster Session I. Drug Discovery

PS-I001

Investigation of Neuroprotective Effect and Proteomic Alterations of Prasugrel Treatment in Parkinson's Disease Model

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The exact etiology of Parkinson's disease (PD) is still not understood and current medications of PD are palliative; therefore, alternative treatments to overcome these hurdles are urgently required. Drug repurposing offers new therapeutic alternatives for incurable diseases, including PD, and is less costly and time-consuming, in addition to ensuring drug safety compared with traditional drug development strategies. This study aimed to repurpose a novel alternative medication from the U.S. Food and Drug Administration-approved drug library using two in vitro PD models, 1-methyl-4-phenylpyridinium-induced primary cortical neurons and lipopolysaccharide-induced BV2 microglial cells. We discovered that prasugrel, an antiplatelet agent used to treat acute coronary syndrome, has neuroprotective and anti-inflammatory effects via inhibition of the mitogen-activated protein kinase signaling pathway. Using integrative pathway analysis results, we found that the expression of various proteins related to apoptotic cell death and neuroinflammation were decreased following prasugrel treatment in in vitro PD models. In the protein-protein interaction analysis, caseinolytic peptidase P and leucine-rich pentatricopeptide repeat containing, both of which are involved in neurotoxicity and PD pathogenesis, were core proteins in the interactome of primary cortical neurons. Furthermore, ribosomal protein L35, which is involved in microglial activation, was identified as the core protein-protein interaction protein in the BV2 proteome. Finally, we confirmed the neuroprotective effect of prasugrel in an MPTP-induced PD mice model through behavioral and histological analyses. Therefore, this study demonstrates that prasugrel can potentially be used to treat PD.

PS-I002

Quantitative Proteomic Profiling Identifies Functional Signatures of PARP Inhibitor Response in HGSOC

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Ovarian cancer is the most lethal gynecologic malignancy affecting women worldwide. Among its subtypes, high-grade serous ovarian cancer (HGSOC) accounts for approximately 70–75% of cases and shows the highest mortality rate. Despite standard treatment, which includes cytoreductive surgery and platinum-based adjuvant chemotherapy, HGSOC has shown high recurrence and mortality rates. The introduction of Poly (ADP-ribose) polymerase inhibitors (PARPi) as targeted therapies has significantly improved the prognosis of patients with HGSOC. PARPi, such as Olaparib, induce the accumulation of DNA single-strand breaks that lead to DNA double-strand damage, eventually resulting in tumor cell death in cancers with homologous recombination deficiency (HRD). This selective cytotoxicity occurs via a mechanism known as "synthetic lethality." However, treatment with PARPi continues to exclude HGSOC patients who retain proficiency in homologous recombination (HR), and the development of resistance to PARPi remains a major clinical challenge. Recent studies have sought to elucidate the mechanisms underlying PARPi resistance and to extend the application of PARPi to HR-proficient patients. However, further investigations are required to expand the pool of potential therapeutic targets, thereby increasing the likelihood of translating these findings into effective clinical strategies. With the rapid advancements in mass spectrometry (MS) technologies, both sensitivity and proteome depth have been markedly improved, and omics approaches offer the advantage of integrating vast datasets to facilitate efficient target discovery. Thus, MS-based proteomics represents a powerful and essential tool for identifying key molecular targets among thousands of proteins. To address these challenges using this platform, it is important to establish new therapeutic targets and strategies. Accordingly, we performed quantitative proteomic profiling to characterize functional signatures of PARPi response in HGSOC. To analyze proteomic differences in PARPi response, we used two HGSOC cell lines, OVCAR3 and KURAMOCHI. Cell viability assays revealed that both cell lines responded to Olaparib in a time- and dose-dependent manner, with distinct sensitivities observed between them. IC50 measurements further confirmed differential sensitivity and resistance responses to PARPi. Immunoblotting demonstrated the induction of DNA damage and early apoptosis following treatment with Olaparib at each cell line's respective IC50 concentration. Cells were harvested after 48 hours of treatment with Olaparib at each cell line's respective IC50 concentration. Proteins were extracted, reduced, and alkylated, then processed on S-Trap micro spin columns and digested on-column with trypsin according to the manufacturer's instructions. Peptides were labeled using tandem mass tag (TMT) 16-plex reagents and analyzed on a Q-Exactive high-resolution mass spectrometer in data-dependent acquisition (DDA) mode. The MS raw data were analyzed using FragPipe software, and the resulting protein data were analyzed using Perseus software. We identified approximately 9,000 proteins per TMT experiment, among which approximately 1000 differentially expressed proteins (DEPs) were identified. Subsequently, Gene Ontology (GO) and pathway enrichment analyses of the DEPs highlighted pathways associated with PARPi resistance. A protein-protein interaction (PPI) network was constructed based on the identified DEPs using the STRING database. Network analysis revealed several hub proteins, which may function as key molecules in PARPi resistance mechanisms. Therefore, the integration of DEPs, pathway analyses, and PPI network analyses enabled the identification of candidate targets, which may serve as potential therapeutic targets and provide insights into novel strategies for HGSOC treatment. Future studies will focus on validating key targets and exploring therapeutic approaches to extend PARPi applicability in HR-proficient HGSOC and to overcome resistance mechanisms. Identifying and validating clinically meaningful targets will be an important step toward advancing therapeutic strategies in HGSOC.

Poster Session I. Drug Discovery

PS-I003

DARTS-LC-MS/MS-Based Target Discovery of Acacetin in MAFLD

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Autophagy is a critical regulator of cellular homeostasis, and its dysregulation is associated with diverse diseases. In particular, impaired autophagy accelerates the progression of metabolic-associated fatty liver disease (MAFLD). However, the mechanistic link between autophagy dysfunction and MAFLD progression remains incompletely understood. To explore the therapeutic potential of autophagy regulation, we identified acacetin (ACA) as a novel autophagy-inducing compound with anti-MAFLD activity. ACA reduced lipid accumulation without cytotoxic effects and enhanced autophagic degradation in vitro, while also alleviating hepatic steatosis in vivo. To investigate the molecular mechanism of ACA, we applied a label-free proteomic target identification approach using drug affinity responsive target stability (DARTS) combined with LC-MS/MS. This analysis identified late endosomal/lysosomal adaptor, MAPK and MTOR activator 1 (LAMTOR1) as a direct target of ACA. Conformational changes in LAMTOR1 upon ACA binding were validated by DARTS and cellular thermal shift assay (CETSA). Collectively, these findings demonstrate that ACA exerts anti-MAFLD effects by directly targeting LAMTOR1 and modulating the mTORC1-AMPK signaling axis to restore autophagy, thereby correcting lipid dysregulation and improving intracellular steatosis.

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PS-I004

Target Identification and Binding Site Mapping of CDK Inhibitors Using Limited Proteolysis-Based Chemoproteomics

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Cyclin-dependent kinases (CDKs) are critical regulators of cell cycle progression and are often dysregulated in cancer and other diseases, making them major targets for therapeutic intervention. Despite the development of several CDK inhibitors, challenges remain in accurately identifying their cellular targets and binding sites, which is essential for understanding their mechanisms of action and optimizing clinical efficacy. Here, we systematically profiled the target landscape of CDK inhibitors in human cell lysates using a limited-proteolysis based chemoproteomics. By employing massive trypsinization methods, we demonstrate the binding site-specific mapping of CDK inhibitors across the CDK family and their other target. Our workflow also allows high-resolution localization of ligand-able hotspots on CDKs, facilitating rational drug design and potential identification of novel regulatory mechanisms. Consequently, this work advances chemoproteomic strategies for comprehensive target deconvolution and binding site elucidation, offering new insights for the development and optimization of next-generation CDK-targeted drugs.

Poster Session I. Drug Discovery

PS-I005

Neuroprotective Effect of Pimobendan in Amyloid-Beta Treated Primary Neurons and 5xFAD Alzheimer's Disease Mice Model

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Alzheimer's disease (AD) is the most common neurodegenerative disorder, characterized by the accumulation of amyloid-β (Aβ), for which no curative treatment is available and the etiology remains poorly understood. In this study, we explored the potential of PDE3 inhibitors—previously reported to have antioxidant and anti-inflammatory effects—as part of a drug repurposing approach to discover new therapeutic agents for AD. Through cell-based screening using an FDA-approved drug library, we identified pimobendan, a selective PDE3 inhibitor, as a potential candidate with beneficial effects for AD. In Aβ-induced in vitro AD model, pimobendan significantly attenuated Aβ-induced apoptosis in a dose-dependent manner. In addition, pimobendan dramatically inhibited MAPK-induced reactive oxygen species production and mitochondrial dysfunction. Neuroprotective effect of pimobendan also validated in 5xFAD in vivo AD model. Mice were administered pimobendan (10 or 100 mg/kg, intraperitoneally) for two weeks. Interestingly, administration of pimobendan at a dose of 100 mg/kg showed a tendency to improve learning and memory performance in the passive avoidance test. These findings suggest that pimobendan may enhance cognitive function by modulating oxidative stress and promoting mitochondrial recovery, rather than by reducing Aβ accumulation. Further investigation is required to elucidate its underlying mechanisms and assess its potential as a repurposed therapeutic candidate for AD.

PS-I006

Cathepsin B-Activated Prodrug Nanoparticles with a Tumor-Implantable Optofluidic System (LED-SC) for Chemo-Photodynamic Glioma Treatment

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Glioma remains one of the most aggressive brain tumors, with therapeutic progress hindered by the blood-brain barrier (BBB) and the skull. To overcome these challenges, we developed a minimally invasive tumor-implantable optofluidic device (LED-SC) that integrates a microscale light-emitting diode (microLED) with a microsyringe chip. This system enables localized delivery of both light and prodrug nanoparticles (PNPs) directly into glioma tissue. The PNPs are self-assembled conjugates of verteporfin (VPF) and doxorubicin (DOX), designed with a cathepsin B-cleavable linker to ensure tumor-selective activation. Once internalized, the nanoparticles release active DOX and VPF, inducing combined chemotherapeutic and photodynamic effects under microLED irradiation. In vitro, glioma cells exhibited efficient nanoparticle uptake and pronounced cytotoxicity following light activation. In vivo, implantation of the PNP-loaded LED-SC achieved a 3.9-fold higher drug delivery efficiency compared with intravenous administration, ensuring deeper tumor penetration, uniform intratumoral distribution, and reduced systemic exposure. Collectively, this strategy provides a promising platform for glioma therapy by enabling dual light/drug administration in a tumor-specific and minimally invasive manner.

Poster Session I. Drug Discovery

PS-I007

Comparative Study of Cathepsin B-Cleavable Linkers for the Optimal Design of Cathepsin B-Specific Doxorubicin Prodrug Nanoparticles for Targeted Cancer Therapy

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Carrier-free prodrug nanomedicine provides a powerful strategy to improve the therapeutic index of anticancer agents by maximizing tumor selectivity while minimizing systemic burden. In this work, we designed and compared a series of five cathepsin B-responsive peptide-doxorubicin (DOX) conjugates, namely FRRG, FRRL, FRRLL, FRRLG, and FLRRG. These conjugates spontaneously organized into nanoparticles through intrinsic amphiphilic balance and π-π interactions, yielding distinct supramolecular architectures. Comprehensive physicochemical analysis revealed that the FRRL-DOX conjugate formed uniform and stable nanoparticles (~167 nm) with prolonged colloidal stability under physiological conditions. Upon cathepsin B-mediated cleavage, FRRL-DOX nanoparticles efficiently liberated DOX, enabling selective nuclear delivery in HT29 colon carcinoma cells while sparing normal cardiomyoblasts. Pharmacokinetic and biodistribution studies demonstrated markedly enhanced tumor accumulation (2.3-16.3-fold relative to free DOX and other prodrug analogues) with minimal hepatic sequestration. Repeated systemic administration of FRRL-DOX nanoparticles at doses up to 10 mg/kg (DOX equivalent) induced no overt systemic or hematologic toxicity. In tumor-bearing mice, FRRL-DOX exhibited superior therapeutic benefit, achieving over twofold higher antitumor efficacy and improved survival compared with free DOX. Collectively, these findings identify FRRL as an optimized cathepsin B-cleavable motif for carrier-free prodrug nanoparticle engineering, providing a rational design principle for next-generation tumor-selective chemotherapy.

PS-I008

Cancer-Targeted Aposomes Delivering SMAC Peptide–Doxorubicin Prodrug for Enhanced Immunotherapy

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Immunogenic cell death (ICD) represents a promising strategy to remodel the immunosuppressive tumor microenvironment into an immune-active state, thereby enhancing the efficacy of immune checkpoint blockade (ICB) therapies. Nonetheless, many chemotherapeutic ICD inducers suffer from limited tumor selectivity and undesired toxicity to immune cells, which restrict their clinical potential. To address this, we developed cancer-targeted PEGylated liposomes, termed Aposomes, encapsulating a cathepsin B-cleavable prodrug composed of a SMAC mimetic peptide (SMAC-P) and doxorubicin (DOX). Upon accumulation in tumors via the enhanced permeability and retention effect, Aposomes release SMAC-P-FRRG-DOX, which is selectively cleaved within cathepsin B-overexpressing cancer cells into active SMAC-P and DOX. This dual mechanism triggers potent apoptosis while synergistically promoting ICD, leading to dendritic cell maturation, cytotoxic T cell activation, and suppression of regulatory T cells. In murine colon tumor models, treatment with Aposomes elicited robust immune responses, inhibited tumor growth, and significantly improved survival compared to free DOX or liposomal DOXIL. Notably, combination therapy with anti-PD-L1 antibody achieved a high rate of complete tumor regression and established durable immunological memory that prevented tumor recurrence. These findings highlight Aposomes as a versatile and clinically relevant nanoplatform for enhancing ICB therapy through the coordinated induction of apoptosis and ICD in tumor cells.

Poster Session I. Drug Discovery

PS-I009

Self-Assembled Peptide PROTAC Nanoparticles Enable Tumor-Targeted and Sustained PD-L1 Degradation

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Proteolysis-targeting chimeras (PROTACs) have emerged as a transformative modality for targeted protein degradation; however, their therapeutic potential has been constrained by poor solubility, limited cellular uptake, and insufficient tumor selectivity. To overcome these challenges, we developed self-assembled peptide-derived PROTAC nanoparticles (PT-NPs) that enable precise and durable degradation of programmed death-ligand 1 (PD-L1) for cancer immunotherapy. The amphiphilic PROTAC peptide (CLQKTP-KQC-FF-ALAPYIP) integrates a PD-L1-binding motif, a phenylalanine-based self-assembly linker, and an E3 ligase-recruiting sequence, yielding ~212 nm nanoparticles with high stability and biocompatibility. PT-NPs multivalently engage PD-L1 on tumor cells, undergo receptor-mediated endocytosis, and induce dual degradation mechanisms: direct lysosomal degradation of membrane PD-L1 and PROTAC-mediated ubiquitin-proteasome degradation of cytosolic PD-L1, effectively preventing its recycling. In CT26 colon tumor models, intravenously administered PT-NPs exhibited enhanced tumor accumulation, prolonged retention, and robust PD-L1 suppression, leading to significant tumor regression, increased cytotoxic T lymphocyte infiltration, and reduced regulatory T cell populations. Importantly, PT-NPs demonstrated enhanced efficacy and a favorable safety profile compared with anti-PD-L1 antibodies, avoiding immune-related toxicities. Collectively, this work establishes a rational nanoplatform that unites self-assembly and PROTAC technologies, offering a versatile strategy for precise and durable checkpoint protein degradation in cancer immunotherapy.

PS-I010

Tumor-Directed BRD4 Degradation via Albumin-Binding PROTACs

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Proteolysis-targeting chimeras (PROTACs) have recently emerged as a transformative therapeutic modality for targeted protein degradation in cancer. However, despite their remarkable potential, the clinical translation of PROTACs has been considerably hindered by unfavorable physicochemical properties, such as high hydrophobicity, poor cell permeability, and limited tumor accumulation. To overcome these intrinsic limitations, we designed and synthesized an esterase-cleavable maleimide-modified BRD4 PROTAC (ECMal-PROTAC), which was engineered to exploit plasma albumin as a natural drug carrier for tumor-targeted delivery. ECMal-PROTAC rapidly and selectively conjugated to endogenous albumin through thiol-maleimide chemistry, thereby forming Alb-ECMal-PROTAC complexes with an average hydrodynamic diameter of approximately 7 nm, which is comparable to native albumin and showed no evidence of denaturation or aggregation. In vitro experiments using 4T1 breast cancer cells revealed that Alb-ECMal-PROTACs were efficiently internalized, achieving maximal intracellular accumulation at 12 h. Following cellular uptake via endocytosis, the maleimide linker was enzymatically cleaved by intracellular esterases, leading to the release of free BRD4-PROTAC. This liberated PROTAC effectively degraded BRD4, suppressed oncogenic downstream effectors including Bcl-2 and c-Myc, and ultimately induced apoptotic cell death. In vivo, intravenous administration of ECMal-PROTACs into tumor-bearing mice resulted in a striking 16-fold increase in tumor accumulation relative to free BRD4-PROTAC. This enhanced biodistribution, mediated by the albumin hitchhiking effect, translated into more than a 5-fold improvement in antitumor efficacy, without any evidence of systemic toxicity. Importantly, the favorable pharmacokinetic profile, together with the observed tumor selectivity, highlights the potential of this strategy to overcome critical barriers to PROTAC drug development. Taken together, these findings establish in situ albumin binding via esterase-cleavable linkers as a powerful and versatile platform for improving the pharmacokinetics, tumor targeting, and therapeutic efficacy of PROTAC-based therapies. This approach provides a broadly applicable strategy for enhancing the clinical translation of diverse protein degradation modalities and may serve as a foundation for next-generation targeted cancer therapeutics.

Poster Session I. Drug Discovery

PS-I011

Neopetroside A Prevents Post-Ischemic Cardiac Fibrosis through GSK-3β Inhibition

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Natural compounds are emerging as potential treatments for cardiovascular disease. Neopetroside A, a pyridine nucleoside with an α-glycosidic bond, was tested for its effects on mitochondrial metabolism and heart function and assessed its ability to protect against ischemia/reperfusion injury in mice. The compound preserved cardiac hemodynamics and mitochondrial respiratory capacity and markedly reduced cardiac fibrosis. Mechanistically, these benefits appear to come from maintained cellular and mitochondrial function via inhibition of glycogen synthase kinase-3β (GSK-3β), which shifts the NAD^+ /NADH balance through activation of the Nrf2-NQO1 pathway in a phosphorylation-independent way.

PS-I012

Spinochrome D Alleviates Doxorubicin-Induced Cardiotoxicity by Modulating Glutathione Metabolism and Oxidative Stress

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A naphthoquinoid pigment from sea urchins, echinochrome A has been known to possess antioxidant effect against ROS-generating environment. We produced 12 echinochrome A derivatives by replacing its potentially reactive groups. In this study, we investigated the cardiomyocyte protective role of echinochrome derivatives in the presence of doxorubicin (Adriamycin, Rubex), an anticancer drug with cardiotoxicity. We found that echinochrome A derivatives protected cardiotoxicity of doxorubicin by reducing reactive oxygen species (ROS) in AC16 cells, isolated human cardiomyocytes. We also found that echinochrome A derivatives protect cardiomyocytes in high glucose condition and cobalt chloride addition which could induce endoplasmic reticulum (ER) stress. Among these derivatives, SpD (2, 3, 5, 6, 8- pentahydroxy-1, 4-naphthoquinone) showed the most significant effect by reducing ROS stress in AC16 cells. We demonstrated that SpD increased ATP ratio and oxygen consumption rate (OCR) from mitochondria. Our study might give light on protecting cardiomyocytes against cancer drugs with severe cardiotoxicity by reducing their ROS generation.

Poster Session I. Drug Discovery

PS-I013

Protective Role of β-Lapachone Against Cardiac Fibrosis and Lipotoxicity in a High-Fat Diet/Streptozotocin-Induced Diabetic Cardiomyopathy Model

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Diabetic cardiomyopathy (DCM) is one of the major causes of end-stage heart failure (HF), contributing to mortality and morbidity in type 2 diabetes. This study aims to evaluate the potential of β-lapachone (B-Lap), a natural compound that enhances antioxidant activity in tissues, as a treatment for DCM by reducing cardiac fibrosis and lipotoxicity in high-fat diet (HFD) and streptozotocin (STZ)-induced diabetic mice. C57BL/6 male mice (seven weeks old) were used to establish a diabetic model through a high-fat diet and low-dose streptozotocin (40 mg/kg/day). Mice were randomly divided into six groups: WT (wild-type mice), WT+HK80, DM (diabetic mice), DM+HK20, DM+HK80, and DM+Met. B-Lap was administered at 20 mg/kg/day (HK20) or 80 mg/kg/day (HK80), while Metformin (Met) was given at 200 mg/kg/day. HFD treatment lasted 10 weeks, and STZ was injected intraperitoneally for five consecutive days after two weeks of treatment to evaluate the protective effects of B-Lap on DCM. B-Lap treatment significantly improved cardiac function and metabolic parameters, including reductions in heart and body weight, blood glucose, HbA1c and insulin resistance. Notably, B-Lap reduced fatty acid uptake in cardiomyocytes by attenuating CD36 expression, leading to decreased triglyceride accumulation and improved mitochondrial fatty acid oxidation. Our findings indicate that B-Lap mitigates diabetic cardiomyopathy by attenuating cardiac fibrosis and lipotoxicity in high fat diet-streptozotocin-induced diabetic mice.

PS-I014

Exercise Attenuates Cisplatin-Induced Anorexia-Cachexia via Modulation of Hypothalamic Inflammation and Orexigenic Signaling

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Chemotherapy is a common cancer treatment, but it often induces chemotherapy-induced anorexia-cachexia (CAC), which negatively impacts prognosis, treatment outcomes, and may lead to treatment discontinuation. However, effective treatment strategies for CAC are currently lacking. The aim of the present study was to evaluate the potential effects of an exercise intervention on the hypothalamus in a mouse model of CAC. Eight-week-old C57BL6 mice were divided into three groups: normal control (CON, n=7), cisplatin injection control (Cis-CON, n=7), and cisplatin with aerobic exercise (Cis-EXE, n=7). CAC was induced using intraperitoneal cisplatin injection at a dose of 3.5 mg/kg/day. The Cis-EXE group underwent treadmill running at a speed of 14-16m/min for 45 min daily, three times per week, for 12 weeks. Our results showed that exercise ameliorated the reduction in food intake and body weight induced by cisplatin and increased the mRNA expression of orexigenic peptides such as neuropeptide Y (NPY) and agouti-related protein (AgRP), while suppressing the expression of anorexigenic peptides, including cocaine-and amphetamine-regulated transcript (CART) and pro-opiomelanocortin (POMC) in the hypothalamus. Additionally, exercise intervention significantly reduced the levels of pro-inflammatory cytokines IL-6, TNF-α, and NF-kB. Exercise also increased AMP-activated protein kinase (AMPK) activity while decreasing the expression of protein kinase B (Akt) and phosphatidylinositol-3 kinase (PI3K). In conclusion, the findings suggest that exercise provides a potential therapeutic strategy for CAC, at least in part via increasing neuropeptide and inhibiting pro-inflammatory in the hypothalamus.

Poster Session I. Drug Discovery

PS-I015

β-Lapachone Prevents Isoproterenol-Induced Cardiac Hypertrophy and Fibrosis through Regulation of NQO1/NRF2/HO-1 Pathway

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This study examined the potential of β-lapachone to attenuate isoproterenol-induced cardiac hypertrophy and dysfunction, while elucidating its underlying cardioprotective mechanisms. Eight-week-old male C57BL/6 mice were used in this study. Cardiac hypertrophy was induced by subcutaneous injection of isoproterenol (ISO, 100 mg/kg/day) for 7 days following a 1-week pretreatment with intraperitoneal β-lapachone (β-lap) at either 20 mg/kg/day or 80 mg/kg/day. The mice were divided into five groups: control, ISO, ISO+β-lap (20 mg), ISO+β-lap (80 mg), and control+β-lap (80 mg). After ISO administration, the β-lapachone groups continued receiving the respective doses, while the other groups received vehicle treatment for an additional 5 weeks. Body weight was monitored weekly, and echocardiography was performed to assess cardiac function at pre- and post-treatment. After the last week of treatment, blood and hearts were collected and further analyses were performed. Results showed that neither ISO nor β-lap treatment affected body weight or indices of liver and kidney function (AST, ALT, and creatinine). However, ISO administration induced cardiac injury, hypertrophy, and dysfunction, as evidenced by elevated serum CK-MB levels, increased heart weight, enlarged myocyte cross-sectional area, reduced ejection fraction (EF) and fractional shortening (FS) on echocardiography, and impaired mitochondrial function. In contrast, β-lap treatment in ISO-challenged mice not only attenuated cardiac hypertrophy and fibrosis but also improved mitochondrial function, thereby enhancing overall cardiac performance. Furthermore, Western blot analysis revealed that β-lap upregulated NQO1 expression and activated the NRF2/HO-1 signaling pathway. These findings suggest that β-lap mitigates isoproterenol-induced cardiac hypertrophy and dysfunction by enhancing NQO1/NRF2/HO1 signaling-mediated mitochondrial function, highlighting its potential as a therapeutic candidate for cardiac hypertrophy and heart failure.

PS-I016

Neopetroside-B alleviates doxorubicin-induced cardiotoxicity via mitochondrial protection

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Doxorubicin, a member of the anthracycline family, is a widely prescribed anticancer chemotherapy drug. Unfortunately, cumulative doses of doxorubicin can cause mitochondrial dysfunction, leading to acute or chronic cardiotoxicity. This study demonstrated that Neopetroside-B (NPS-B) protects cardiomyocytes in the presence of doxorubicin. NPS-B improved mitochondrial function in cardiomyocytes by increasing ATP production and oxygen consumption rates. On the other hand, NPS-B negatively influenced cancer cell lines by increasing reactive oxygen species. We analyzed NPS-B-influenced metabolites (VIP 〉 1.0; AUC〉0.7; p〈0.05) and proteins (FC 〉 2.0) and constructed metabolite-protein enrichment, which showed that NPS-B affected uracil metabolism and NAD-binding proteins (e.g., aldehyde dehydrogenase and glutathione reductase) in cardiomyocytes. However, for the cancer cells, NPS-B decreased the NAD+/NADH balance, impairing cell viability. In a xenograft mouse model treated with doxorubicin, NPS-B reduced cardiac fibrosis and improved cardiac function. NPS-B may be a beneficial intervention to reducing doxorubicin-induced cardiotoxicity with anticancer effects.

Poster Session J. Others

PS-J001

Integrating Subcellular Fractionation and N-Terminomics to Identify Dual-Localized Alternative Translational Isoforms in Mitochondrial Proteome

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Proteins in eukaryotic cells must be accurately targeted to specific organelles to perform their functions. Most mitochondrial proteins are encoded by nuclear genes, translated in the cytoplasm, and subsequently imported into mitochondria. Recent studies suggest that the selection of alternative translation initiation sites (aTIS) plays a critical role in determining the subcellular localization of proteins. In this study, we quantitatively assessed whether protein isoforms derived from aTIS are differentially localized between the cytoplasm and mitochondria. HCT116 cells were SILAC-labeled, fractionated into organelles, followed by N-terminal peptide enrichment and LC-MS/MS analysis. Our results revealed that inclusion of a mitochondrial targeting sequence (MTS) varies depending on aTIS usage in numerous nuclear-encoded mitochondrial proteins, generating isoforms specifically localized to distinct organelles. This indicates a regulatory mechanism by which a single gene produces protein isoforms differing in subcellular localization and function, similar to that reported for the FUM1 gene. This study highlights the functional significance of aTIS at the proteomic level, providing fundamental insights into mitochondrial protein targeting with potential implications for disease.

PS-J002

Quantitative Proteomic Analysis of Brain Proteomic Changes Induced by Repeated and Passive Δ9-Tetrahydrocannabinol Exposure

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Cannabis consumption has become increasingly widespread, raising concerns about the long-term consequences of exposure to its major psychoactive component, ⊿9-tetrahydrocannabinol (THC). Chronic THC exposure has been associated with addictive behaviors, neurocognitive impairment, and respiratory dysfunction; however, the molecular mechanisms underlying repeated and passive exposure remain poorly understood. In our previous work, we characterized proteomic alterations following acute THC exposure at 1 and 14 days. Building on these findings, the present study investigates proteomic alterations in the brain of mice subjected to repeated or passive THC inhalation. Mice were exposed to controlled vaping of 100 mg THC, with repeated exposures conducted over 30, 60, and 90 days, while passive exposure was modeled under identical conditions. Repeat oil control (ROC) groups were exposed to cannabis-free base oil vapor for 30 days under the same vaping protocol. Brain tissues were subjected to proteomic analysis through tandem mass tag (TMT) labeling and liquid chromatography–tandem mass spectrometry (LC-MS/MS), which enabled multiplexed quantification of protein abundances. Our results reveal distinct proteomic signatures and pathway alterations associated with chronic and passive THC exposure, highlighting brain-specific molecular responses. This study provides a proteomic framework for understanding the molecular mechanisms underlying chronic and passive THC exposure.

Poster Session J. Others

PS-J003

Proteomic Analysis of Mouse Brain Tissue Following Acute Inhalation Exposure to Illegally Distributed Cannabis Oil via Vaping

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The illegal distribution and recreational abuse of cannabis among adolescents and adults is increasing worldwide, posing a serious public health risk due to uncertainties in product composition and lack of safety regulation. This study investigated proteomic alterations in mouse brain tissue following inhalation of illegally distributed cannabis oil products, with the aim of identifying specific protein expression signatures. Mice were exposed through a controlled vaping protocol consisting of a 2-second puff followed by a 10-second rest, repeated for 20 sets with a 6-minute interval afterward, for a total duration of 60 minutes. Control groups were exposed only to base oil. On day 1 post-exposure, brain tissues were collected from both control mice and cannabis oil-exposed mice (n = 5 per group), and on day 14, additional samples were obtained from the cannabis oil group (n = 5). Proteins were extracted from brain tissues and subjected to in-solution digestion, and the resulting peptides were labeled using tandem mass tags (TMT) for multiplexed quantification, followed by fractionation using high-pH reversed-phase chromatography.The liquid chromatography-tandem mass spectrometry (LC-MS/MS) analysis revealed significant alterations in brain protein expression following exposure to illegally distributed cannabis oil, indicating distinct biological responses to its components. The proteomic framework established in this study will be extended to other major organs in future work, thereby contributing to a comprehensive understanding of organ-specific biological responses induced by the inhalation of illegally distributed cannabis oil.

PS-J004

Global Proteome Analysis of Early-Stage TDP-43 Proteinopathy in Caenorhabditis Elegans

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TAR DNA-binding protein 43 (TDP-43) is predominantly localized in the nucleus and plays essential roles in RNA metabolism, including splicing, transport, and stability. However, abnormal cytoplasmic mislocalization and aggregation of TDP-43 are pathological hallmarks of a spectrum of neurodegenerative disorders collectively termed TDP-43 proteinopathies. Among these, amyotrophic lateral sclerosis (ALS) and frontotemporal lobar degeneration (FTLD) are the most prominent. Cytoplasmic aggregates of TDP-43 are detected in more than 90% of ALS patients and approximately 50% of FTLD cases, highlighting the central role of TDP-43 in disease pathogenesis. Despite extensive research, no effective therapeutic strategies have been developed, and the molecular mechanisms underlying the early stages of TDP-43 proteinopathy remain unclear. In this study, we employed an optogenetic Caenorhabditis elegans model, OptoTDP, in which hTDP-43 fused to Cry2olig irreversibly aggregated upon blue light stimulation. Using nLC-Orbitrap MS-based global proteomic analysis, we identified 107,815 peptides and 9,501 proteins across four sample groups. Through comparative analyses, we identified 58 DEPs specifically altered upon TDP-43 aggregation. Functional annotation analysis revealed that upregulated proteins (n=32) were associated with metabolic processes, intracellular anatomical structure, catalytic activity, and metabolism of xenobiotics by cytochrome P450. In contrast, downregulated proteins (n=26) were associated with lipid transport/localization, lipid transporter activity, and signaling by VEGF. Especially, among the upregulated proteins, gst-4 and gst-5 were identified as being involved in cytochrome P450-mediated xenobiotic metabolism and prostaglandin (PG) synthesis. HPGDS, the human ortholog of these proteins, is an enzyme that converts arachidonic acid to PGD2. PGD2 is known to induce motor neuron cell death by acting on the DP1 receptor, suggesting that these proteins play a key role in the early stages of TDP-43 proteinopathy. These results provide clues to the changes in proteins associated with lipid metabolism in the early stages of TDP-43 proteinopathy.

Poster Session J. Others

PS-J005

Comparative Proteomic Profiling of Thyroid Cancer Cell Lines Reveals Subtype-Specific Signatures

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Thyroid cancer is the most common endocrine malignancy. It comprises heterogeneous subtypes, including papillary (PTC), follicular (FTC), and anaplastic thyroid carcinoma (ATC), which display distinct biological behaviors and clinical outcomes. While genomic studies have revealed critical subtype-specific alterations, proteomic characterization remains limited. Global proteomic analysis enables comprehensive identification of differentially expressed proteins (DEPs) and functional pathways, thereby providing insights into tumor heterogeneity. Cell line-based proteomic studies offer a valuable platform for systematic investigation of tumor-intrinsic molecular characteristics and serve as the foundation for subsequent clinical investigations, despite inherent limitations including genetic instability and loss of in vivo complexity. In this study, we conducted a comparative proteomic analysis of 15 thyroid cell lines (5 PTC, 2 FTC, 7 ATC, and 1 normal) to define subtype-specific DEPs and characterize their associated molecular pathways. Proteins were extracted using SDS lysis buffer, digested using the S-trap method with trypsin, and peptides desalted with SDB-RPS tips. Peptides were then analyzed on an Orbitrap Exploris 480 mass spectrometer in data-independent acquisition mode, and the resulting data were searched against our in-house spectral library using Spectronaut. This library, generated from the same thyroid cell lines by combining spectral libraries obtained through high-pH and gas-phase fractionation, encompasses 457,090 precursors, 345,063 modified peptides, and 11,958 proteins. Using this resource, we achieved comprehensive proteome profiling and quantified over 9,000 proteins from our samples. Based on the numbers of DEPs relative to normal thyroid cells, FTC showed the most extensive changes, followed by PTC and ATC. This was unexpected, as FTC is generally considered the most similar to normal cells due to its relatively well-differentiated phenotype. Subsequent pairwise comparisons between the subtypes further revealed numerous DEPs, indicating distinct molecular signatures underlying each subtype. While proteins associated with cell migration were overexpressed across all subtypes, Gene Ontology enrichment analysis highlighted distinct subtype-specific processes: vascularization in PTC, cell developmental processes in FTC, and negative regulation of multicellular organismal processes in ATC. Consistently, hierarchical clustering with heatmap analysis based on DEPs revealed that cell lines within each subtype cluster together, exhibiting similar protein expression patterns. Across subtypes, ATC was clearly separated, while PTC and FTC showed more comparable clustering patterns, reflecting the undifferentiated nature of ATC and the differentiated phenotype of PTC and FTC. Taken together, our comprehensive proteomic dataset provides detailed molecular profiles of thyroid cancer cell subtypes. These findings suggest the distinct molecular mechanisms driving each subtype and emphasize the importance of subtype-specific analyses in understanding thyroid cancer. Using our dataset together with a high-quality spectral library, we establish a robust proteomic platform that serves as a foundational resource for thyroid cancer research and further aim to address emerging biological and clinical questions in the field.

PS-J006

Identification of Pannexin-3 as a Missing Protein in the Human Proteome Using Liquid Chromatography-Tandem Mass Spectrometry

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The Human Proteome Project (HPP) has classified Pannexin-3 (PANX3) as a "missing" protein due to insufficient evidence of its existence. PANX3, a member of the pannexin family, is known to form functional cell surface channels and play roles in various physiological processes. This study finds the PANX3 (NX_Q96QZ0, Protein Evidence Level 2) located on chromosome 11 using liquid chromatography-tandem mass spectrometry. We discovered PANX3 from cancer patients, and subsequently validated through comparison with corresponding synthetic peptides. This approach not only confirmed the presence of PANX3 but also demonstrated the effectiveness of LC-MS/MS in identifying previously undetected proteins. We provide an evidence for PANX3's existence and establishes a foundation for further investigations into its biological functions and potential clinical significance. Our findings contribute to the ongoing efforts of the HPP to comprehensively characterize the human proteome and highlight the importance of advanced analytical techniques in protein identification.

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Poster Session J. Others

PS-J007

Three-Dimensional Culture of Bovine Adipose-Derived Stem Cells for Alternative Fat

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Growing global population, the environmental impact of greenhouse gas emissions, and the rising costs associated with conventional meat production are driving an urgent need for sustainable alternative protein sources. In this context, cultivated meat, produced through advanced cell culture methodologies, is gradually being recognized as a promising and environmentally friendly protein alternative capable of closely replicating the sensory and nutritional characteristics of traditional meat. Among the various components that contribute to meat quality, fat is a key determinant of flavor and overall palatability, making its accurate reproduction a critical challenge for cultivated meat development. In the present study, adipose-derived stem cells isolated from intermuscular adipose tissues of bovine (bADSCs), a subtype of mesenchymal stem cells (MSCs) obtained from adipose tissue, were employed to generate fat tissue in vitro as a functional substitute for animal-derived fat. To confirm the stemness and multipotent characteristics of bADSCs, we evaluated the expression of canonical MSC surface markers (CD29, CD73, and CD105) and assessed their colony-forming efficiency. The cells were subsequently induced to undergo adipogenic differentiation under both two-dimensional (2D) and three-dimensional (3D) culture conditions, allowing for the formation of lipid-rich adipocytes in environments that more closely mimic native tissue architecture. Fat samples harvested from these cultures were analyzed using gas chromatography, revealing a fatty acid composition closely aligned with that of natural bovine adipose tissue, including comparable ratios of saturated and unsaturated fatty acids. Moreover, the use of a textured vegetable protein (TVP) scaffold during adipogenic induction supported robust lipid droplet formation within the scaffold matrix, demonstrating the feasibility of combining plant-based matrices with cell-cultured fat for hybrid meat products. Taken together, these findings establish a robust and reproducible in vitro platform for the production of bovine-derived fat from bADSCs and underscore the potential application of this system in the development of hybrid cultivated meat products that offer improved sustainability without compromising flavor or nutritional quality. This research is supported by research funding from Simple Planet and the Korea Institute of Planning and Evaluation for Technology in Food, Agriculture and Forestry (IPET) through the High Value-added Food Technology Development Program, funded by the Ministry of Agriculture, Food and Rural Affairs (MAFRA, RS-2024-00402798).

PS-J008

Maximizing Proteome Insights: A Comparative Study of Adipose Tissue Sample Processing

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Adipose tissue plays a key role in metabolism and immune regulation; therefore, its proteomic characterization is essential for understanding diseases such as metabolic dysfunction-associated steatotic liver disease (MASLD). However, due to its high lipid content and low protein yield, adipose tissue poses significant technical challenges for in-depth proteomic analysis. To improve proteome coverage, we aimed to investigate a sample preparation method that maximizes lipid removal while minimizing protein loss by comparing two distinct strategies: removal of excess lipids (RELi) and a chloroform-based two-phase extraction (C2PE). Proteins obtained from each approach were identified and quantified using the TMT 16-plex labeling method to assess the strengths of each strategy and their suitability for analyzing lipid-rich tissues, murine adipocytes. A total of 5,686 proteins were identified across all samples, with notable differences depending on the sample preparation method. RELi-based processing enabled the detection of a greater number of proteins such as Pigu, Plaat3 and Lpcat1, which are associated with cellular metabolic process. In contrast, the C2PE-based processing was more effective in capturing proteins such as Col4a2, Fn1, and Cma1, which are involved in cellular component organization. Furthermore, a comparative analysis of retroperitoneal and mesenteric adipocytes revealed differentially expressed proteins related to metabolic processes and translational activity.

Poster Session J. Others

PS-J009

Multi-Fluid Quantitative Proteomics Defines Divergent Host Responses and Mechanisms in Viral and Bacterial Infections

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Viral and bacterial infections remain leading causes of global morbidity and mortality, with influenza and pneumococcal pneumonia representing major respiratory threats. While both pathogens elicit strong host responses, the underlying mechanisms differ substantially, and comparative studies across local and systemic compartments remain limited. In this study, we applied quantitative proteomics in mouse infection models to investigate the distinct responses induced by influenza virus and Streptococcus pneumoniae. Bone marrow wash fluid, bronchoalveolar lavage fluid, and plasma exosomes were analyzed to capture early immune activation, local pulmonary changes, and systemic alterations. Comparative profiling revealed clear differences in protein expression patterns between viral and bacterial infections, as well as between biological fluids within the same infection type. Functional characterization of infection-specific protein sets highlighted both unique and partially overlapping pathways, indicating that while some immune processes are commonly activated, the overall response patterns diverge significantly between pathogens. Furthermore, fluid-specific signatures underscored the importance of tissue context in shaping immune responses. Collectively, this integrative analysis delineates the distinct host mechanisms employed during viral and bacterial infections, providing insights into pathogen-specific immune modulation that may inform improved diagnostic, preventive, and therapeutic strategies. Supported by the National Research Foundation of Korea (NRF), funded by the Korean government (MSIT) (No. RS-2024-00454407).

PS-J010

Integrative Analysis of Striatal Proteome Remodeling in zQ175 Knock-In Mouse of Huntington's Disease

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Huntington's disease (HD) is an autosomal dominant neurodegenerative disorder caused by abnormal CAG repeat expansion in the huntingtin (HTT) gene on chromosome 4p16.3. The mutant HTT is translated into huntingtin protein containing an expanded polyglutamine tract (polyQ). Proteolytic N-terminal fragments of huntingtin that include the expanded polyQ are prone to misfolding and aggregation, leading to the formation of pathological inclusion bodies (IBs) that sequester other cellular proteins. The striatum is one of the most severely affected brain regions in HD, because it is rich in medium spiny neurons that are highly vulnerable to mutant HTT. In this context, understanding the dynamics of both the soluble proteome and IBs is significant for elucidating HD progression. Here, we employed the zQ175 knock-in mouse model, which mimics human HD pathology more accurately than other transgenic models. Proteins extracted from striata of wild-type and heterozygous zQ175 mice at 6 and 22 months were fractionated into radio immunoprecipitation assay (RIPA)-soluble supernatants and insoluble pellets. After tryptic digestion, the resulting peptides from both fractions were subsequently analyzed by liquid chromatography-tandem mass spectrometry (LC-MS/MS). In total, we identified 9,156 soluble and 8,760 insoluble proteins in the striata of wild-type and zQ175 mice, respectively. To the best of our knowledge, this represents the highest proteome coverage reported for the zQ175 model. With deep proteomic profiling of this model, we could capture stage- and fraction-specific proteomic alterations during HD progression. Differentially expressed proteins (DEPs) were few at 6 months, indicating limited proteomic alterations at the early stage. In contrast, a substantial increase in DEPs was observed at 22 months, reflecting extensive remodeling in both soluble and insoluble proteomes. Functional enrichment analysis demonstrated that DEPs at 22 months in both fractions were involved in synaptic signaling, motor coordination, and memory, showing consistent perturbations of neuronal functions. Although both fractions implied neuronal dysfunction, soluble DEPs were mainly linked to transcriptional regulation and neurotransmission, whereas insoluble DEPs were more strongly associated with translation, neurodegeneration, and neuroinflammation. In addition to these functional differences, integrative analysis of the soluble and insoluble proteomes enabled the identification of candidate proteins that are potentially sequestered into IBs by mutant HTT. Such candidates could serve as novel molecular contributors in the pathophysiology of HD and offer promising targets for therapeutic intervention. Our findings provide novel molecular insights into the poorly understood huntingtin-containing IBs as well as the soluble proteome.

Poster Session J. Others

PS-J011

Genome- Wide Association Study for Milk Production Traits in Korean Holstein Cattle

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Holstein cattle are the foundation of Korea's dairy industry and have been selectively bred for more than a century. A deeper understanding of the genetic factors influencing yield and composition is essential for making further progress. This study aimed to identify quantitative trait loci (QTL) and candidate genes associated with 305-day total milk yield (Milk305), 305-day total milk fat yield (Fat305), and 305-day total milk protein yield (Prot305) in Korean Holsteins across first and second lactation. Phenotypic data came from 5,289 cows provided by the National Agricultural Cooperative Federation and the Korean Livestock Improvement Association. These included 2,795 cows in the first lactation and 2,494 in the second lactation. Genotypic data for 2,016 cows were obtained using the Illumina Bovine 50K SNP BeadChip. Heritability estimates for Milk305, Fat305, and Prot305 were 0.31, 0.22, and 0.16 for the first lactation and 0.37, 0.36, and 0.25 for the second lactation. A total of 531 significant SNPs were identified for first-lactation traits and 396 for second-lactation traits. Notable candidate genes included POU1F1, STMN1, HSP90B1, EPHA7, and DGAT1 for Milk305; MOGAT1, SLC52A2, SREBF1, GPS1, FASN, and DGAT1 for Fat305; and BOD1L1, CCL2, and DGAT1 for Prot305. These findings contribute to a clearer understanding of the genetic architecture of milk traits and provide a foundation for marker-assisted selection and genomic breeding programs aimed at improving dairy efficiency and sustainability in Korea.

PS-J012

A Proteomic Analysis of Mouse Retina Revealed Molecular Changes Induced by Drug Addiction

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Morphine and lysergic acid diethylamide (LSD) are major psychoactive drugs that contribute to drug addiction, posing a global public health concern. Morphine is an opioid drug that acts on the central nervous system (CNS) to relieve pain but is associated with a high risk of addiction and severe side effects such as confusion. LSD, a powerful serotonin receptor agonist is known for its hallucinogenic activity. Recent studies demonstrated that chronic exposure to these drugs disrupt not only mind and behavior but also fundamental molecular networks within the CNS, further contributing to neurotoxicity. The retina, an extended part of the CNS, is known to be vulnerable to the side effect of psychoactive drugs and provides a uniquely accessible site for the observation of drug-induced neurotoxic processes. Specifically, it has been reported that morphine and LSD addiction is associated with various retinal diseases including maculopathy, Saturday night retinopathy, and talc retinopathy. However, the molecular mechanisms how these drugs contribute to the retinal dysfunction remain largely unknown. To address this, we performed a proteomic analysis of mouse retinas following morphine or LSD administrations. A high-throughput Data Independent Acquisition (DIA) strategy resulted over 10,000 protein identifications and subsequent statistical analysis discovered hundreds of differentially expressed proteins (DEPs). Furthermore, we observed dose-dependent molecular changes in LSD-administrated mouse models, providing molecular evidence for its direct pharmacological action on the retina. Further bioinformatics analyses revealed that dysregulation of cellular proteostasis and neurotoxic pathways were prominent in both models. In conclusion, our study generated an in-depth retinal proteome dataset and uncovered significant biological pathways connecting morphine and LSD to retinal dysfunction, providing better understanding of the molecular insights regarding drug-induced organ degeneration.

Poster Session J. Others

PS-J013

Evaluation of Heavy Metal Content and Food Safety of High Value-Added Fish Distributed in East Sea

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Seafood offers substantial nutritional benefits, yet heavy-metal accumulation in marine environments can lead to human exposure through consumption, raising public-health concerns—such as neurodevelopmental effects of methylmercury, renal toxicity of cadmium, and cardiovascular or cognitive impacts of lead—and eroding consumer confidence. To address these issues, this study quantified seven elements—arsenic (As), cadmium (Cd), copper (Cu), mercury (Hg, total), manganese (Mn), lead (Pb), and zinc (Zn)—in high-value fish species widely consumed in Korea, namely olive flounder (Paralichthys olivaceus), Japanese eel (Anguilla japonica), and rainbow trout (Oncorhynchus mykiss). For each species, three specimens (total n = 9) were obtained at the retail stage in Gangneung, a representative coastal city in the East Sea region, and then processed and homogenized. Mn and Zn were quantified by ICP-OES, Pb, Cd, As, and Cu by ICP-MS, and Hg by a direct mercury analyzer, following standardized procedures with quality control. Element- and species-specific distributions were observed: Mn and Zn occurred at comparatively higher levels consistent with their biological roles, Cd and Pb were present only in trace amounts, and Hg was consistently low across all species. Overall, all measured concentrations were within both domestic (MFDS, Korea) and international (FAO/WHO, EU, US FDA) food-safety standards, demonstrating that consumption of these fish species poses a low risk of heavy-metal exposure to consumers. This study provides a critical baseline for assessing dietary exposure to heavy metals and contributes to the development of sustained monitoring frameworks that account for seasonal variability, supply-chain differences, and species-specific accumulation. Furthermore, the findings support the refinement of quantitative risk-assessment tools such as THQ and HI, and offer a scientific foundation for residue and transfer evaluations across raw materials, cell-derived materials, process inputs, and intermediate or final products-thereby strengthening safety assurance during the transition to cultivated seafood.

PS-J014

TRPA1 Downregulation Decreases Fibrotic Markers in TGFB1-Induced in vitro Fibrotic Model

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Background: Transient receptor potential Ankyrin 1 (TRPA1) is a non-selective cation channel primarily studied for its nociceptive role in neurons. However, more recent studies confirmed its expression in other tissues such as cardiac tissues where it facilitates ion transport. This study aimed (1) to evaluate the regulation of TRPA1; and (2) to determine the role of TRPA1 in the physiology of cardiac fibrosis in vitro.

Methods: Western blot was performed to check the TRPA1 protein levels in vitro (mouse cardiac fibroblasts) fibrotic model. RT-PCR analysis was used to measure gene expression while immunocytochemistry was used to visualize the protein expression in cells. To create an in vitro fibrotic model, mouse cardiac fibroblasts (MCF) were treated with rhTGFβ1 (5 ng/mL). On the other hand, to create a TRPA1 knockdown model, siRNA targeting TRPA1 was treated to the cells.

Results: TRPA1 levels in MCF were found to increase under fibrotic conditions. Meanwhile, knocking down TRPA1 using siRNA decreased the expression of fibrotic markers, α-SMA and col1a1. Accordingly, the decreased levels of TRPA1 under fibrotic conditions attenuated the phosphorylation of SMAD2 and ERK1/2 proteins which led to decreased fibrotic markers.

Conclusions: The findings of this study showed that inhibition of TRPA1 is a potential strategy in addressing against TGF-β1-induced cardiac fibrosis. The study also provided evidence that TRPA1 affects the progression of fibrosis through the fibrotic ERK1/2 signaling pathway.

Poster Session J. Others

PS-J015

FABP3 and IGFB7 as Potential Biomarkers for the Early Detection Diabetic Cardiomyopathy

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Objectives: Diabetic cardiomyopathy is a disorder of myocardial function that affects diabetics. The initial stage of the disease is reversible, but the later stages progress to systolic dysfunction and heart failure. The main objective of this study was to evaluate the ability of FABP3, IGFBP7, and MYH7, alone or in combination, to function as biomarkers for predicting the early onset of diabetic cardiomyopathy.

Methods: To characterize the phenotype of animals with diabetic cardiomyopathy, we determined when serum levels of the proposed biomarkers began to rise above the normal range, and compared with serum levels of NT-proBNP. Male C57BL/6J and db/db mice aged 5 weeks were studied without intervention for 13 weeks of age. Echocardiography was performed at 7, 9, 11, and 13 weeks, 3 days before termination. Serum levels of FABP3, IGFBP, MYL7, and NT-proBNP were determined by ELISA; FABP3, IGFBP7, MYL7, and MYH7 protein and mRNAs levels were quantified by Western blot and RT-PCR assays.

Results: Echocardiography data showed that both systolic and diastolic functions were not significantly different in WT and db/db mice groups throughout at 7, 9, 11, and 13 weeks of age. However, protein levels of FABP3 and IGFBP7 gradually increased in the db/db group following the same pattern as weight and fasting blood sugar. In contrast, levels of MYL7 were found to be lower and declined over time. Plasma levels of FABP3 and IGFBP7 increased over time in the db/db group, while levels of NT-proBNP did not differ between the groups.

Conclusion:
Overall, these findings show that FABP3 and IGFBP7 can be considered early markers for diagnosing diabetic cardiomyopathy.

PS-J016

Aerobic Exercise Decreases the Levels of CRBN in the Skeletal Muscle of Type 1 Diabetes Rats

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Cereblon (CRBN) has been reported as a negative regulator of adenosine monophosphate-activated protein kinase (AMPK). Aerobic exercise training has been shown to increase AMPK., which resulted in glucose regulation in skeletal muscle. However, the expression level of CRBN and its association with the physiological modulation of glucose are still unclear. Male Sprague-Dawley rats (5-week-old, n=18) were assigned to control, streptozotocin (STZ, 65mg/kg)-induced diabetic group, and STZ+Exercise (STZ+EXE) group with six rats in each group. Rats in the STZ+EXE group exercised by treadmill running (20m/min, 60 min, 4 times/week) for 8 weeks. Compared with the STZ group, blood glucose was significantly decreased in the STZ+EXE group. The skeletal muscle of rats in the STZ+EXE group showed a significant decrease in CRBN levels and an increase in AMPK, protein kinase B, peroxisome proliferator-activated receptor gamma coactivator 1-alpha, fibronectin type III domain-containing protein 5, glucose transporter type 4, superoxide dismutase 1, and uncoupling protein 3 levels. These results suggest that CRBN is a potential regulator of glucose homeostasis in the skeletal muscle. Moreover, our results suggest that aerobic exercise training may provide an important physiological treatment for type 1 diabetes by decreasing CRBN and increasing AMPK signaling in skeletal muscle.

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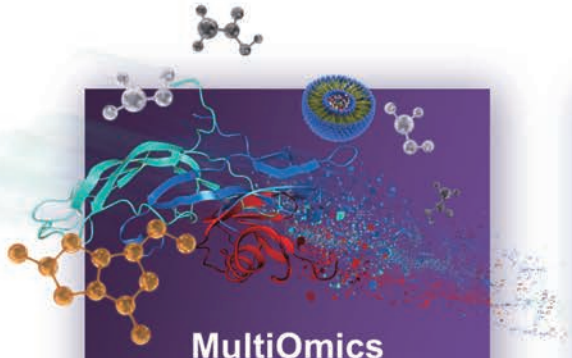
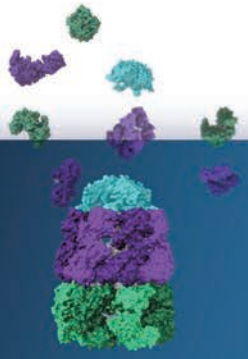


MultiOmics

Powerful versatility from metabolomics to proteomics



Structural Biology

Unmatched combination of low and high mass range on a single high-resolution platform

BioPharma

Versatile platform from metabolite ID to native biotherapeutic analysis

thermo scientific