

구룡포 제1회 대한미생물학회 동계 네트워킹

WINTER NETWORKING

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경북대학교·경북대학교병원 인재원

주관·주최 |



대한미생물학회
Korean Society for Microbiology



경북대학교
난치성감염병연구소



충남대학교
CHUNGNAM NATIONAL UNIVERSITY
감염제어 리더연구단

대한미생물학회는 감염성 질환으로부터 국민을 보호하고, 국가보건 및 산업의 원동력을 함양을 목적으로 전국의 의과대학을 중심으로 1946년 5월 1일에 설립된 대한민국에서 가장 오래된 전국규모의 학술 단체입니다. 학회 회원들이 의과대학을 포함한 다양한 학계, 의료기관, 연구소, 바이오 제약 등의 산업계에서 병원성 미생물 및 감염성 질환과 관련된 연구와 개발에 종사하고 있는 학회 회원들의 노고로 현재에 이르렀습니다.

금번 제1회 대한미생물학회 동계 네트워킹은 학회회원간의 학술적 소통, 교류 및 강화를 통해 의학 미생물학 및 감염 면역학 분야의 학문적인 발전을 도모 할 수 있는 계기를 만들고자 포항 구룡포 경북대학교 경북대학교병원 인재원에서 2월 10일부터 11일까지 개최를 하게되었습니다.

학회 회원님들의 적극적인 참여가 이번 제1회 대한미생물학회 동계 네트워킹의 성공적인 개최의 핵심입니다. 회원님들의 적극적인 참여를 통하여 학회 회원님들의 활발한 학문적인 소통 및 교류가 이루어져 학회가 더 발전할 수 있는 기회가 되기를 바랍니다.

2026. 2. 10

대한미생물학회장 기 선 호

Program

02.10 Tue

13:00 -

등록

사회: 인하의대 신민혜 교수 | 학술부위원장

13:50 - 14:00

환영사

고려의대 기선호 교수

대한미생물학회 회장

14:00-15:45 Young Scientist (PI)

14:00-14:15	김석호	경북대학교 의과대학	Advanced Engineering of Bacteriophage Therapy: Integrating Minimal Pegylation and Nanoclay-based Detoxification for Enhanced Efficacy Against Multidrug-resistant Enteric Infections
14:15-14:30	김우섭	고려대학교 의과대학	Antibody-based Understanding of Human B Cell Immunity Following Vaccination and Infection
14:30-14:45	김의태	제주대학교 의과대학	HSV-1 Exploits Non-degradative Ubiquitin Signaling to Concentrate Antiviral Factors in VCP Condensates
14:45-15:00	김진경	계명대학교 의과대학	Control of Hypervirulent and Antibiotic-Resistant Infectious Diseases through Host-Directed Therapeutic Modulation
15:00-15:15	손문일	부산대학교	No Brain, but Neurodegenerative Disease: Prions in Microbes and Its Relevance to Microbiology
15:15-15:30	신민혜	인하대학교 의과대학	Integrated Strategies for Disease Control: Next-Generation Antimicrobials, Bioactive Metabolites, and Synthetic Live Biotherapeutics
15:30-15:45	신혜진	충남대학교 의과대학	Viral Hijackers: Virus-host Molecular Interactions, Antiviral Drug Discovery, and Environmental Determinants of Viral Infection

Program

15:45 - 17:00 Young Scientist (PI)			
15:45-16:00	오태환	단국대학교 의과대학	Advances in Spatial Transcriptomics for Infectious Disease Research
16:00 - 16:15	이민호	한림대학교 의과대학	New Therapeutic Approaches to Overcome Antibiotic Resistance
16:15 - 16:30	이용욱	계명대학교 의과대학	Diet, Organism, Omics, Metabolism laboratory
16:30 - 16:45	이충용	경북대학교 의과대학	Evolution of Influenza A Virus and its Cross-species Transmission
16:45 - 17:00	최은영	건국대학교 의과대학	Oncogenic Human Herpesvirus Hijacks Host Metabolism for Viral Pathogenesis and Tumorigenesis
17:00 - 18:00 Special Lecture			
17:00 - 17:30	조남혁	서울대학교 의과대학	IL-27-mediated Hematopoietic Dysregulation Exacerbates Disease Severity in Severe Fever with Thrombocytopenia Syndrome Virus Infection
17:30 - 18:00	이준행	전남대학교 의과대학	Flagellin: A Engineerable Immune Modulator and Adjuvant

18:00 - 18:10 **맺음말** 충남의대 김화중 교수 **대한미생물학회 이사장**

18:10 - **만찬**

▣ 02.11 Wed

10:00 - 12:00 **개별토론**

Advanced Engineering of Bacteriophage Therapy: Integrating Minimal Pegylation and Nanoclay-based Detoxification for Enhanced Efficacy Against Multidrug-resistant Enteric Infections

Shukho Kim

Department of Microbiology, School of Medicine, Kyungpook National University, The Republic of Korea

Background: The rise of multidrug-resistant (MDR) bacteria necessitates innovative alternatives to conventional antibiotics. However, clinical application of phage therapy faces challenges such as rapid systemic clearance by the immune system and the "endotoxin dilemma"—the inflammatory surge caused by toxin release during bacterial lysis.

Objectives: This study proposes a multi-faceted approach to optimize phage therapy by combining minimal pegylation (mpeg-s-nhs) for improved pharmacokinetics and bentonite-based functional nanoclay for in situ detoxification and microbiome restoration.

Methods: Lytic phages (vb_abast_w16 and ec.W2-6) were characterized for their efficacy against *acinetobacter baumannii* and ETEC. Minimal pegylation was applied to enhance serum stability and reduce immunogenicity. Simultaneously, bentonite nanoclay was integrated to sequester lipopolysaccharides (LPS), outer membrane vesicles (omvs), and enterotoxins released during lysis.

Results: Minimal pegylation significantly extended the systemic circulation of phages and improved survival rates in immunocompetent murine models without compromising infectivity. Concurrently, the addition of bentonite nanoclay effectively neutralized lysis-induced toxins and protected phages from gastric acidity. This dual strategy not only ensured 100% survival in lethal infection models but also promoted gut microbiome recovery by suppressing *proteobacteria* and restoring beneficial microbial diversity.

Conclusions: Synergizing chemical surface modification (pegylation) with mineral-based adsorbents (nanoclay) addresses the critical limitations of phage therapy. This integrated platform provides a robust, non-antibiotic intervention that enhances phage longevity and safety, offering a comprehensive solution for treating severe MDR infections and maintaining intestinal homeostasis.

Antibody-based Understanding of Human B Cell Immunity Following Vaccination and Infection

Wooseob Kim

Department of Microbiology Korea University College of Medicine, The Republic of Korea

B cell immunity, characterized by the production of pathogen-specific antibodies, is a cornerstone of long-term protection against infectious diseases. While foundational insights into B cell biology have largely been derived from animal models, these systems often fail to fully capture the complexity and heterogeneity of human immune responses. Recent advances in high-resolution experimental and analytical technologies have enabled the direct interrogation of the human immune system, revealing previously inaccessible dynamics of human B cells following vaccination and infection.

Central to these responses is the germinal center (GC) reaction, where antigen-activated B cells undergo rapid proliferation and diversify their B cell receptor (BCR) repertoires through somatic hypermutation. Within the GC, B cells are subjected to stringent selection pressures that favor clones with superior antigen affinity, driving affinity maturation and the generation of long-lived plasma cells and memory B cells.

In this study, we leverage an evolutionary framework to dissect human B cell responses at both clonal and functional resolutions. By analyzing mutation frequencies and patterns in paired heavy- and light-chain immunoglobulin sequences, alongside V(D)J gene usage, we define clonal architectures and reconstruct B cell developmental trajectories. Furthermore, we characterize monoclonal antibodies derived from these sequences to assess the functional properties of individual clones directly. Collectively, this antibody-based approach provides critical insights into the dynamics, selection, and fate of B cell responses, offering an essential knowledge base for the development of next-generation vaccines.

HSV-1 Exploits Non-degradative Ubiquitin Signaling to Concentrate Antiviral Factors in VCP Condensates

Eui Tae Kim

**Department of Microbiology and Immunology, Jeju National University
College of Medicine, The Republic of Korea**

Herpes simplex virus 1 (HSV-1) has evolved to exploit the ubiquitin system beyond conventional proteasomal degradation. The viral E3 ubiquitin ligase ICP0 harnesses non-degradative ubiquitin signaling to efficiently neutralize host antiviral factors through a novel mechanism involving biomolecular condensates.

ICP0 induces the formation of nuclear condensates enriched with the AAA+ ATPase VCP (valosin-containing protein). These structures exhibit properties of liquid-liquid phase separation (LLPS) and serve as specialized compartments that concentrate and clear diverse host restriction factors. This virus-driven condensate formation enables spatiotemporal control of antiviral factor clearance, representing an efficient strategy to suppress host immunity without relying solely on proteasomal degradation.

This work reveals a paradigm shift in viral immune evasion: non-proteolytic ubiquitination drives the assembly of functional condensates for coordinated clearance of multiple restriction factors. I will discuss how HSV-1 co-opts phase separation principles to reshape the nuclear landscape and explore therapeutic implications of targeting this mechanism.

Control of Hypervirulent and Antibiotic-Resistant Infectious Diseases through Host-Directed Therapeutic Modulation

Jin Kyung Kim

Department of Microbiology, Keimyung University School of Medicine, Daegu, The Republic of Korea

This research focuses on hypervirulent and antibiotic-resistant infectious diseases, aiming to overcome the limitations of pathogen-centered therapies through host-directed therapeutic strategies. In particular, the work investigates how host immune cell functions are disrupted during infection and mechanistically elucidates how the interplay among autophagy, inflammatory responses, and immunometabolic reprogramming influences pathogen survival and disease severity.

Using cellular and animal infection models based on clinically isolated hypervirulent and antibiotic-resistant strains, this research demonstrates that modulation of host immune pathways can enhance intracellular bacterial clearance while attenuating excessive inflammatory responses. Special emphasis is placed on infection models of hypervirulent *Klebsiella pneumoniae*, in which host immunometabolic remodeling and autophagy dysregulation are quantitatively analyzed for their contributions to infection persistence and therapeutic responsiveness.

Through these studies, this work aims to establish mechanism-based therapeutic strategies to overcome antibiotic resistance and to develop preclinical evaluation frameworks using infection animal models, thereby contributing to a paradigm shift in infectious disease treatment from pathogen targeting toward host-centered immune modulation.

No Brain, but Neurodegenerative Disease: Prions in Microbes and Its Relevance to Microbiology

Moonil Son

Department of Microbiology, Microbiological Resource Research Institute, Pusan National University, Busan, 46241, The Republic of Korea

Prion is simply an infectious protein, and it occurs spontaneously without any well-defined reason. Once prions occurred, they mostly propagated in biophysically very stable amyloid form with self-templating mechanism. Human and mammalian prion results in untreatable and even fatal brain disorders, thus study of prion and its pathology extremely difficult. However, since the first discovery of two prions in model microorganism *Saccharomyces cerevisiae*, studies about prion biology, host physiology affected by prion or prion disease have facilitated. Moreover, many attempts to understand human and mammalian prion diseases were applied based on what have done from yeast-prion system. In this review, progressive advances, from early experiment recognitions about prion even before actual proof to current advances in prion research, will be discussed, and from the fundamentals, such as yeast prion manipulation, prion biology including prion domain and transmission, to in-depth achievements of prion amyloid structure and anti-prion systems will be presented. Lastly, the impact of yeast prion study on other kingdom such as bacteria and human biomedical research, and recent development of basic cellular physiology.

Integrated Strategies for Disease Control: Next-Generation Antimicrobials, Bioactive Metabolites, and Synthetic Live Biotherapeutics

Minhye Shin

Department of Microbiology, College of Medicine, Inha University, 22212, The Republic of Korea

The Inha Microbial Biochemistry Laboratory is dedicated to the control of pathogenic bacterial infections and the development of microbe-derived pharmaceuticals for human health. These goals are based on comprehensive studies of microbial physiology, genetics, metabolism, and biochemistry. Utilizing multi-omics, genome engineering tools, animal models, and protein-based biochemical analyses, our main research topics include:

- 1) The development of next-generation antimicrobials and vaccine antigens** against multi-drug resistant (MDR) bacteria, including methicillin-resistant *Staphylococcus aureus* (MRSA), carbapenem-resistant Enterobacteriaceae (CRE), and vancomycin-resistant Enterococci (VRE), to inhibit their growth and virulence expression.
- 2) The discovery of novel microbial metabolites** for disease prevention and treatment via metagenome and metabolome profiling, targeting cancer, metabolic diseases, and intractable infectious diseases.
- 3) The development of live bio-therapeutic products (LBPs) by engineering probiotic strains**, particularly *Lactobacillus* and *Bifidobacterium* species, using CRISPR-Cas genome editing and bio-delivery toolkits for enhanced therapeutic effects.

Viral Hijackers: Virus–host Molecular Interactions, Antiviral Drug Discovery, and Environmental Determinants of Viral Infection

Hye Jin Shin

Department of Microbiology, Chungnam National University School of Medicine, Daejeon, 35015, Republic of Korea

Respiratory viruses have evolved complex strategies to utilize host cellular pathways to support their replication and sustained infection. Respiratory viruses, including severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and respiratory syncytial virus (RSV), hijack host signaling networks and mitochondrial functions to optimize viral propagation. Viral infection reprograms mitochondrial dynamics and bioenergetic metabolism, resulting in enhanced adenosine triphosphate (ATP) production and maintenance of cellular homeostasis that favor viral replication. Intracellular epidermal growth factor receptor (EGFR) signaling serves as a central regulatory node linking virus-induced signaling cascades to mitochondrial regulation. Pharmacological inhibition of EGFR activation disrupts virus-induced mitochondrial remodeling and suppresses viral propagation, indicating that host EGFR signaling is a critical determinant of infection outcome. Furthermore, virus–environment interactions are evaluated using in vitro respiratory disease models, including asthma- and chronic obstructive pulmonary disease (COPD)-derived airway epithelial cells, as well as exposure-related airway conditions, to clarify how the host microenvironment modulates viral replication and inflammatory responses. Overall, an integrated framework is presented in which respiratory viruses hijack host signaling and organelle homeostasis for survival, supporting host-directed antiviral strategies as a promising approach for future pandemic preparedness.

Advances in Spatial Transcriptomics for Infectious Disease Research

Taehwan Oh

Department of Microbiology, College of Medicine, Dankook University, Republic of Korea

Spatial transcriptomics (ST) enables genome-wide gene expression profiling while preserving tissue architecture, bridging the gap between bulk, single-cell, and histo-logical analyses. Originating in 2016 and rapidly evolving since, ST has transformed infectious disease research by mapping host–pathogen interactions directly within in-tact tissues. Current platforms fall into two categories: Sequencing-based methods (Visium, GeoMx, Stereo-seq) offering whole-transcriptome coverage at modest resolution, and imaging-based platforms (Xenium, CosMx, MERFISH) providing single-cell or subcellular detail with targeted gene panels. These technologies reveal spatially organized immune responses, local tissue remodeling, and pathogen niches across viruses, bacteria, and parasites. In viral infection, ST uncovered heterogeneity in COVID-19 lung microenvironments, spatial immune activation in lymphoid tissues, and variant-specific inflammatory patterns. In bacterial disease, ST delineated granuloma architecture in tuberculosis and mapped vaccine-induced lung responses in *Shigella* studies. Parasitic infection studies identified localized inflammatory hotspots and microenvironmental control of T cell differentiation in malaria. Despite powerful insights, ST faces constraints including RNA quality limitations, trade-offs between resolution and transcript breadth, high cost, and analytical complexity. Nonetheless, ST increasingly informs vaccine design by identifying tissue-specific immune programs and protective micro-environments, and is poised to become a standard tool for infectious disease biology.

New Therapeutic Approaches to Overcome Antibiotic Resistance

Minho Lee^{1,2}

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Antimicrobial resistance (AMR) is an escalating global health threat, and carbapenemase-producing carbapenem-resistant *Klebsiella pneumoniae* (CPKP) is a high-risk pathogen associated with treatment failure and high mortality. This study evaluated the antibacterial activity and mechanism of a novel antimicrobial peptide, hirunipin 2, against clinical CPKP isolates. MIC testing showed activity across multiple isolates (8–64 µg/mL). Checkerboard assays revealed strong synergy with rifampicin (FICI ≤ 0.5), and time-kill experiments confirmed rapid bactericidal effects within 4 h when combined with rifampicin or meropenem. Membrane permeabilization assessed by SYTOX Green increased faster and to higher levels with hirunipin 2 than with melittin or colistin, indicating potent membrane disruption. Binding assays demonstrated interactions with LPS and cardiolipin, with higher affinity for cardiolipin, suggesting preferential targeting of the inner membrane. These findings support hirunipin 2 as a promising AMP candidate and a synergistic partner for repurposed antibiotic combinations against CPKP.

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Diet, Organism, Omics, Metabolism laboratory

Yong-Uk Lee

Department of Microbiology, Keimyung University School of Medicine, The Republic of Korea

Host–microbe interactions range from mutualism to infection, but complex ecosystems such as the human gut microbiota make it hard to isolate microbe-specific effects on the host. Using *C. elegans* fed with defined bacterial diets, we connect microbial inputs to cellular and molecular mechanisms. Prior work indicates that different bacterial foods alter host physiology and life-history traits, including development, reproduction, longevity, and transgenerational effects. We aim to identify molecular determinants that specify these interactions and to discover host/microbial factors that buffer or exacerbate phenotypes under genetic or environmental perturbations.

Evolution of Influenza A Virus and its Cross-species Transmission

Chung-Young Lee

Kyungpook National University, School of Medicine, Daegu, The Republic of Korea

Influenza A viruses (IAVs) are major pathogens that frequently cross the interface between animals and humans. Historical data from the last four pandemics highlight that the introduction of novel influenza strains is primarily driven by the cross-species transmission of animal-origin viruses. However, our understanding of how these viruses overcome host-specific barriers remains incomplete. This talk introduces our laboratory's ongoing research and future strategies aimed at elucidating the mechanisms of viral evolution and interspecies transmission.

Oncogenic Human Herpesvirus Hijacks Host Metabolism for Viral Pathogenesis and Tumorigenesis

Un Yung Choi ^{1,2}

¹Department of Infection and Immunology, Konkuk University School of Medicine, Seoul 05030, ²Department of Microbiology, Konkuk University School of Medicine, Chungju 27478, The Republic of Korea

Oncogenic γ -herpesviruses such as Kaposi's sarcoma-associated herpesvirus (KSHV) exploit host metabolic pathways to support persistent infection, episodic reactivation, and ultimately the malignant transformation of infected cells. Using 3D spheroid cultures that better mimic the *in vivo* metabolic environment, we performed metabolomic analyses and observed marked increases in proline and spermidine levels. Mechanistic studies based on these findings revealed that KSHV reprograms proline biosynthesis through activation of pyrroline-5-carboxylate reductase (PYCR) by the viral K1 oncoprotein. This interaction elevates intracellular proline levels, thereby promoting three-dimensional spheroid growth and tumorigenesis *in vivo*. More recently, we further demonstrated that KSHV infection enhances spermidine synthesis and consequently increases eIF5A hypusination, which stabilizes latent nuclear antigen (LANA) expression essential for episome maintenance during latency. Collectively, these studies highlight a multi-layered metabolic hijacking strategy in which KSHV first amplifies amino acid anabolic pathways to drive viral transformation and then engages the polyamine–hypusination axis to sustain viral latency and long-term persistence.

IL-27-mediated Hematopoietic Dysregulation Exacerbates Disease Severity in Severe Fever with Thrombocytopenia Syndrome Virus Infection

Nam-Hyuk Cho

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College of Medicine, Seoul 03083, The Republic of Korea**

Severe Fever with Thrombocytopenia Syndrome (SFTS) is a life-threatening tick-borne viral infection characterized by high fever, thrombocytopenia, and severe inflammation, with no effective treatments currently available. Through comprehensive analysis of bone marrow and peripheral blood mononuclear cells from SFTS patients, and validation in a mouse model, we identified IL-27 as a key driver of disease progression. Elevated IL-27 levels promote emergency hematopoiesis and impair protective B cell development and antibody production, resulting in worsened inflammation and ineffective viral control. IL-27 induction in monocytes and B cells triggers a vicious inflammatory cycle, exacerbating the severity of SFTS. Neutralizing IL-27 in the mouse model significantly improved survival, reduced viral loads, and restored anti-viral immunity, highlighting IL-27 as a promising therapeutic target for SFTS.

Flagellin: An Engineerable Immune Modulator and Adjuvant

Joon Haeng Rhee

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Modern vaccines became safer than old conventional vaccines by improving purity and regulating composition with defined constituents. Consequently, new vaccines confer lower immunogenicity because of the removal of built-in adjuvant components through more refined manufacturing and purification processes. Hence, newly developed vaccines require coformulation with effective adjuvants. TLR ligands are considered attractive adjuvants for vaccines and immunotherapy. Flagellin is the cognate ligand for Toll-like receptor 5 (TLR5) of host cells. TLR stimulation leads to activation of innate immunity and subsequently modulates adaptive immune responses. Flagellin has an excellent adjuvanticity for mucosal vaccines. Mucosal co-administration of a *V. vulnificus* flagellin (FlaB) with microbial antigens induced significantly enhanced antigen-specific IgA responses in both mucosal and systemic compartments and IgG responses in the systemic compartment. Mucosally administered FlaB targets TLR5 expressing CD11c+ DCs in draining lymph nodes and stimulate induction of antigen-specific T and B cell responses. Flagellin could be engineered as a component of built-in adjuvanted vaccines as a fusion partner of antigens or building blocks of multivalent nanoparticle formulations. The built-in adjuvanted vaccines could be further engineered to target antigen presenting cells and enhance cross-presentation resulting in stronger cellular immune responses. Recently, we have engineered flagellin for repeated administration, targeted immune response generation, mRNA development, etc.