Curriculum Vitae

Name	**First Name**	Moon Jung	**Last Name**	Song
Country	Republic of Korea			
Affiliation	Korea University College of Life Sciences and Biotechnology			

Educational Background	
1990-1994	B.S., Department of Pharmacy, College of Pharmacy, Seoul National University, Seoul, Republic of Korea (magna cum laude)
1994- 1996	M.S. in Pharmacology, College of Pharmacy, Seoul National University, Seoul, Republic of Korea
1997- 2002	Ph.D. in Molecular and Medical Pharmacology, David Geffen School of Medicine, University of California at Los Angeles, U.S.A.

Professional Career	
2002-2004	Postdoctoral Researcher, Department of Molecular and Medical Pharmacology, David Geffen School of Medicine, University of California at Los Angeles, California, USA
2004-2006	Assistant Professor, Department of Microbiology, College of Medicine, Hallym University, Chuncheon, Republic of Korea
2006-2009	Assistant, Associate, & Full Professor, Division of Biotechnology, College of Life Sciences and Biotechnology, Korea University, Seoul, Republic of Korea
2021-present	Director, Korea Bio-Defense Research Institute, Korea University, Seoul, Republic of Korea
2024-2025	Vice President, Graduate Office, Korea University, Seoul, Republic of Korea
2025-present	Director, Korea University Library, Seoul, Republic of Korea

Research Field

Virus–Host Interactions Laboratory: From Mechanisms to Therapeutics

My research focuses on the molecular virology of oncogenic gammaherpesviruses and emerging RNA and respiratory viruses, emphasizing virus–host interactions, innate immunity, and host-directed therapeutic strategies. The central goal of my laboratory is to understand how viruses manipulate host defense systems to establish infection, persistence, and pathogenesis, and to leverage this knowledge toward the development of broad-spectrum and host-directed antivirals.

Using a genome-wide functional genomics platform for murine gammaherpesvirus 68 (MHV-68), a small-animal model closely related to EBV and KSHV, we have identified viral genes essential for replication and immune evasion, and elucidated mechanisms of interferon antagonism and inflammasome regulation. We further demonstrated that NLRC3 acts as a critical regulator constraining viral latency through a reciprocal NF-κB–dependent loop, conserved across gammaherpesvirus infections.

Expanding beyond oncogenic herpesviruses, we utilized reverse genetics systems for emerging RNA viruses, including major respiratory pathogens, to define viral gene functions, host dependency factors, and antiviral mechanisms. In parallel, we discovered conserved viral strategies counteracting the host restriction factor PARP1 and uncovered the link between viral reactivation, PARP1 degradation, and NAD^+ metabolism. Our research also integrates chemical and structural biology approaches, identifying bioactive natural products and G-quadruplex (G4) ligands that modulate replication of herpesviruses and SARS-CoV-2. Recently, together with the Broad Institute, we embarked on a collaborative effort to develop host-directed therapies against pan-respiratory viruses, building upon our expertise in molecular virology and virus–host interaction research.

Papers, Books, etc. presented or published by your name

1. Dynamic bidirectional regulation of NLRC3 and gammaherpesviruses during viral latency in B lymphocytes. (2024) *Journal of Medical Virology*; e29504.
2. Suppression of SARS-CoV-2 nucleocapsid protein dimerization by ISGylation and its counteraction by viral PLpro. (2024) *Frontiers in Microbiology* 15:1490944
3. Stabilization of RNA G-quadruplexes in the SARS-CoV-2 genome suppresses viral infection via translational repression. (2023) *Archives of Pharmacal Research* 46(7):598-615
4. G-quadruplexes formed by Varicella-Zoster virus reiteration sequences suppress expression of glycoprotein C and regulate viral cell-to-cell spread. *PLoS Pathogens* 19(1):e1011095
5. Epstein-Barr Virus Viral Processivity Factor EA-D Facilitates Virus Lytic Replication by Inducing Poly(ADP-Ribose) Polymerase 1 Degradation. (2022) *Journal of Virology* 96(21):e0037122
6. Kaposi's sarcoma-associated herpesvirus processivity factor (PF-8) recruits cellular E3 ubiquitin ligase CHFR to promote PARP1 degradation and lytic replication. (2021) *PLOS Pathogens* 17(1): e1009261.