SUNG KYUN KWAN UNIVERSITY(SKKU)

BAE Yoe-Sik

Professor
Department of Biological Sciences

- **Office** 32302A, Science Building 2, Sungkyunkwan University (SKKU) Natural Sciences Campus, 2066 Seobu-ro, Jangan-gu, Suwon, Gyeonggi-do, Republic of Korea
- **Phone** 82-31-290-5914
- **Website**
- **E-mail** yoesik@skku.edu
- **Social Media**

Key Words Leukocyte, Inflammation, Sepsis, Chemotaxis, Rheumatoid arthritis

Research Area

We are interested in the development of bioactive molecules that can be used to treat infectious or inflammatory diseases. Our major target diseases include polymicrobial sepsis, rheumatoid arthritis, inflammatory bowel disease, and colon cancer. To generate useful target molecules that can be used to develop therapeutic agents against these infectious or inflammatory diseases, we are focusing on severa chemoattractant G-protein coupled receptors (GPCRs). Since leukocyte trafficking in vivo is a crucial event that modulate immune and inflammatory responses, and chemotactic GPCRs mediate these response, the receptors can be considered as important primary targets to control infectious or inflammatory disorders. Currently, we are testing the effects and mechanism of action of chemotactic GPCR ligands against these diseases in experimental animal models

Education

• 2000	PhD	Dept. of Life Science, POSTECH
• 1998	MSc	Dept. of Life Science, POSTECH
• 1996	BSc	Dept. of Animal Sci. & Technol., Seoul National University

Experience

• 2014 - Present	Professor, Dept. of Biological Sciences, Sungkyunkwan University
• 2010 - 2014	Associate Professor, Dept. of Biological Sciences, Sungkyunkwan University
• 2008 - 2009	Visiting Associate Professor, Stanford University School of Medicine
• 2002 - 2010	Full-time Instructor, Assistant Professor, Associate Professor, Dong-A University College of Medicine
• 2000 - 2002	Postdoc, POSTECH

Position

• 2015 - 2016	Chief of academic affair, Korean Peptide Protein Society
• 2014 -	Senior Editorial Board Member, American Journal of Clinical and Experimental Immunology
• 2015 -	Editorial Board Member, International Journal of Cancer Immunology & Immunotherapy

Selected Publication

- Lee et al., Phospholipase D2 drives mortality in sepsis by inhibiting neutrophil extracellular trap formation and down-regulating CXCR2. J Exp Med. 2015;212(9):1381-90.
- Kim et al., Activation of CXCR2 by extracellular matrix degradation product acetylated Pro-Gly-Pro has therapeutic effects against sepsis. Am J Respir Crit Care Med. 2011;184(2):243-51.
- Kim et al., Phospholipase C activator m-3M3FBS protects against morbidity and mortality associated with sepsis. J Immunol. 2012;189(4):2000-5.
- Lee et al., Sphingosylphosphorylcholine stimulates CCL2 production from human umbilical vein endothelial cells. J Immunol. 2011;186(7):4347-53.
- Kim et al., The agonists of formyl peptide receptors prevent development of severe sepsis after microbial infection. J Immunol. 2010 ;185(7):4302-10.
- Kim et al., Functional expression of formyl peptide receptor family in human NK cells. J Immunol. 2009;183(9):5511-7.
- Lee et al., Serum amyloid A induces CCL2 production via formyl peptide receptor-like 1-mediated signaling in human monocytes. J Immunol. 2008;181(6):4332-9.

Others

- Several patents on therapeutic agents against sepsis or rheumatoid arthritis