



• Field Immunology, Virology  
• Name Park, Jin-seo  
• Title Professor

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## Education background

- Undergraduate Degree  
Dept. of Food Engineering, Yonsei University, Korea; B.S., 1981.
- Graduate Degree  
Dept. of Biological Engineering, Korea Advanced Institute of Science and Technology, Korea; M.S., 1983.  
Dept. of Microbiology, University of Alabama at Birmingham, USA; Ph.D., 1992
- Postdoctoral Training  
Harvard Medical School, Dana-Farber Cancer Institute, Division of Human Retrovirology, 1992-1994.

## Major careers

- 1) 2016- Dean, Graduate School
- 2) 2013-2016 Dean, College of Natural Science
- 3) 2012-2013, Chairman, Department of Biomedical Science, Hallym University
- 4) 2009-2012, Vice President for Research Affairs, Hallym University
- 5) 2005-, Professor, Department of Biomedical Science, Hallym University
- 6) 2001-2002, Visiting Professor, Department of Cell Biology, University of Alabama at Birmingham
- 7) 2003-2005, Professor, Department of Genetic Engineering, Hallym University
- 8) 1998-2003, Associate Professor, Department of Genetic Engineering, Hallym University
- 9) 1994-1998, Assistant Professor, Department of Genetic Engineering, Hallym University
- 10) 1992-1994, Research Fellow, Department of Pathology, Dana-Farber Cancer Institute Harvard Medical School

## Studies & Books

### ■ Representative Publications (2011 ~ present)

- 1) Jo H, Jang HY, Youn GS, Kim D, Lee CY, Jang JH, Choi SY, Jun JG, Park J. 2018. Hindsiiopropane B alleviates HIV-1 Tat-induced inflammatory responses by suppressing HDAC6-NADPH oxidase-ROS axis in astrocytes. *BMB Rep.* 51:394-399.
- 2) Youn GS, Cho H, Kim D, Choi SY, Park J. 2017. Crosstalk between HDAC6 and Nox2-based NADPH oxidase mediates HIV-1 Tat-induced pro-inflammatory responses in astrocytes. *Redox Biol.* 12:978-986.
- 3) An SY, Youn GS, Kim H, Choi SY, Park J. 2017. Celastrol suppresses expression of adhesion molecules and chemokines by inhibiting JNK-STAT1/NF- $\kappa$ B activation in poly(I:C)-stimulated astrocytes. *BMB Rep.* :25-30
- 4) Youn GS, Lee KW, Choi SY, Park J. 2016. Overexpression of HDAC6 induces pro-inflammatory responses by regulating ROS-MAPK-NF- $\kappa$ B/AP-1 signaling pathways in macrophages. *Free Radic Biol Med.* 97:14-23.
- 5) Kim H, Youn GS, An SY, Kwon HY, Choi SY, Park J. 2016. 2,3-Dimethoxy-2'-hydroxychalcone ameliorates TNF- $\alpha$ -induced ICAM-1 expression and subsequent monocyte adhesiveness via NF- $\kappa$ B inhibition and HO-1 induction in HaCaT cells. *BMB Rep.* 49(1):57-62.
- 6) Park SH, Shin MJ, Kim DW, Park J. Choi SY, Kang YH. 2016. Blockade of monocyte-endothelial trafficking by transduced Tat-superoxide dismutase protein. *Int J Mol Med.* 37(2):387-397.

- 7) Youn GS, Ju SM, Choi SY, [Park J](#). 2015. HDAC6 mediates HIV-1 Tat-induced proinflammatory responses by regulating MAPK-NF-kappaB/AP-1 pathways in astrocytes. *Glia*. 63(11):1953-1965.
- 8) Kim YN, Kim DW, Jo HS, Shin MJ, Ahn EH, Ryu EJ, Yong JI, Cha HJ, Kim SJ, Yeo HJ, Youn JK, Hwang JH, Jeong JH, Kim DS, Cho SW, [Park J](#), Eum WS, Choi SY. 2015. Tat-CBR1 inhibits inflammatory responses through the suppressions of NF-kB and MAPK activation in macrophages and TPA-induced ear edema in mice. *Toxicol Appl Pharmacol*. 286(2):124-134.
- 9) Seo WY, Youn GS, Choi SY, [Park J](#). 2015. Butein, a Tetrahydroxychalcone, Suppresses Pro-inflammatory Responses in HaCaT Keratinocytes. *BMB Rep*. 48(9):495-500.
- 10) Kim MJ, Park M, Kim DW, Shin MJ, Son O, Jo HS, Yeo HJ, Cho SB, Park JH, Lee CH, Kim DS, Kwon OS, Kim J, Han KH, [Park J](#), Eum WS, Choi SY. 2015. Transduced PEP-1-PON1 proteins regulate microglial activation and dopaminergic neuronal death in a Parkinson's disease model. *Biomaterials*. 64:45-56.
- 11) Yoon CH, Kim SY, Byeon SE, Jeong Y, Lee J, Kim KP, [Park J](#), Bae YS. 2015. p53-Derived Host Restriction of HIV-1 Replication by Protein Kinase R-Mediated Tat Phosphorylation and Inactivation. *J Virol*. 89(8):4262-4280.
- 12) Ju SM, Youn GS, Cho YS, Choi SY, [Park J](#). 2015. Celastrol ameliorates cytokine toxicity and pro-inflammatory immune responses via suppression of NF-kB activation in the RINm5F beta cells. *BMB Rep*. 48(3):172-177.
- 13) Youn GS, Kwon DJ, Ju SM, Rhim H, Bae YS, Choi SY, [Park J](#). 2014. Celastrol ameliorates HIV-1 Tat-induced inflammatory responses via NF-kappaB and AP-1 inhibition and heme oxygenase-1 induction in astrocytes. *Toxicol Appl Pharmacol*. 280:42-52.
- 14) Kwon DJ, Bae YS, Ju SM, Youn GS, Choi SY, [Park J](#). 2014. Salicortin suppresses lipopolysaccharide-stimulated inflammatory responses via blockade of NF-kB and JNK activation in RAW 264.7 macrophages. *BMB Rep*. 47(6):318-323.
- 15) Kwon DJ, Bae YS, Ju SM, Youn GS, Choi SY, [Park J](#). 2014. Salicortin suppresses lipopolysaccharide-stimulated inflammatory responses via blockade of NF-kB and JNK activation in RAW 264.7 macrophages. *BMB Reports*. 47(6):318-323.
- 16) Shin MJ, Kim DW, Lee YP, Ahn EH, Jo HS, Kim DS, Kwon OS, Kang TC, Cho YJ, [Park J](#), Eum WS, Choi SY. 2014. Tat-glyoxalase protein inhibits against ischemic neuronal cell damage and ameliorates ischemic injury. *Free Radic Biol Med*. 67:195-210.
- 17) Youn GS, Kwon DJ, Ju SM, Choi SY, [Park J](#). 2013. Curcumin ameliorates TNF-alpha-induced ICAM-1 expression and subsequent THP-1 adhesiveness via the induction of heme oxygenase-1 in the HaCaT cells. *BMB Reports* 46(8):410-415.
- 18) Kwon DJ, Ju SM, Youn GS, Choi SY, [Park J](#). 2013. Suppression of iNOS and COX-2 expression by flavokawain A via blockade of NF-kB and AP-1 activation in RAW 264.7 macrophages. *Food Chem Toxicol*. 58:479-486.
- 19) Kim MJ, Kim DW, Park JH, Kim SJ, Lee CH, Yong JI, Ryu EJ, Cho SB, Yeo HJ, Hyeon J, Cho SW, Kim DS, Son O, [Park J](#), Han KH, Cho YS, Eum WS, Choi SY. 2013. PEP-1-SIRT2 inhibits inflammatory response and oxidative stress-induced cell death via expression of antioxidant enzymes in murine macrophages. *Free Radic Biol Med*. 63:432-445.
- 20) Ju SM, Goh AR, Kwon DJ, Youn GS, Kwon HJ, Bae YS, Choi SY, [Park J](#). 2012. Extracellular HIV-1 Tat induces human beta-defensin-2 production via NF-kappaB/AP-1 dependent pathways in human B cells. *Mol Cells*. 33(4):335-341.
- 21) Kwon DJ, Bae YS, Ju SM, Goh AR, Youn GS, Choi SY, [Park J](#). 2012. Casuarinin suppresses TARC/CCL17 and MDC/CCL22 production via blockade of NF-kB and STAT1 activation in HaCaT cells. *Biochem Biophys Res Commun*. 2012 417(4):1254-1259.
- 22) Lee SH, Kim DW, Kim HR, Woo SJ, Kim SM, Jo HS, Jeon SG, Cho SW, Park JH, Won MH, [Park J](#), Eum WS, Choi SY. 2012. Anti-inflammatory effects of Tat-Annexin protein on ovalbumin-induced airway inflammation in a mouse model of asthma. *Biochem Biophys Res Commun*. 417(3):1024-1029
- 23) Song HY, Ju SM, Seo WY, Goh AR, Lee JK, Bae YS, Choi SY, [Park J](#). 2011. Nox2-based NADPH oxidase mediates HIV-1 Tat-induced up-regulation of VCAM-1/ICAM-1 and subsequent monocyte adhesion in human astrocytes. *Free Radic Biol Med*. 50(5):576-584.
- 24) Seo WY, Goh AR, Ju SM, Song HY, Kwon DJ, Jun JG, Kim BC, Choi SY, [Park J](#). 2011. Celastrol induces expression of heme oxygenase-1 through ROS/Nrf2/ARE signaling in the HaCaT cells. *Biochem Biophys Res Commun*. 407:535-540.
- 25) Kwon DJ, Bae YS, Ju SM, Goh AR, Choi SY, [Park J](#). 2011. Casuarinin suppresses TNF-alpha-induced ICAM-1 expression via blockade of NF-kB activation in HaCaT cells. *Biochem Biophys Res Commun*. 409:780-785.
- 26) Kim SY, Sohn EJ, Kim DW, Jeong HJ, Kim MJ, Kang HW, Shin MJ, Ahn EH, Kwon SW, Kim YN, Kwon HJ, Kim TY, Lee KS, [Park J](#), Eum WS, Choi SY. 2011. Transduced PEP-1-FK506BP Ameliorates Atopic Dermatitis in NC/Nga Mice. *J Invest Dermatol*. 131:1477-1485.
- 27) Kwon SW, Sohn EJ, Kim DW, Jeong HJ, Kim MJ, Ahn EH, Kim YN, Dutta S, Kim DS, [Park J](#), Eum WS, Hwang HS, Choi SY. 2011. Anti-inflammatory effect of transduced PEP-1-heme oxygenase-1 in Raw 264.7 cells and a mouse edema model. *Biochem Biophys Res Commun*. 411:354-359.

## I Others

### ■ Awards

- 1) Fellowship, 1987-1991, Department of Microbiology  
University of Alabama at Birmingham
- 2) Korean National Fellowship, 1981-1983, Department of Biological Sciences  
Korea Advanced Institute of Science and Technology
- 3) Academic Award, 2015. 11. 13, 2015 Annual Meeting of The Korean Society for AIDS  
The Korean Society for AIDS

### 주요연구주제

- Pathogenesis of AIDS by HIV, Development of therapeutic tools using protein transduction technology, Development of therapeutic materials against inflammatory diseases

### ■ Societies

- The Korean Society of Virology
- The Korean Society for Biochemistry and Molecular Biology
- The Korean Society for Molecular Biology and Cell Biology
- The Korean Society for AIDS

### ■ Research interests and current research (Laboratory of Virology and Immunology)

#### 1) Background

The global spread of HIV infection and AIDS continues while current therapies are very limited and effective vaccines are nonexistent. Accordingly, novel therapeutic approaches must be devised to target viral gene functions that regulate virus replication and produce disease. Thus, understanding of viral gene function may not only lead to the elucidating the viral pathogenesis, but also may provide new approaches to inhibit HIV replication and/or preventing disease progression.

#### 2) Elucidation of the role of Tat in the pathogenesis of HIV

Tat is a transcription transactivator produced by the human immunodeficiency virus type 1 (HIV-1) at the early phase of infection and plays an essential role in the expression and replication of the viral genome. Tat protein, which can be secreted from the infected cells, has the ability to enter uninfected cells and exert its activity upon the variety of cellular genes, suggesting Tat may affect various cell types in the central nervous system, potentially contributing to the development of AIDS-associated neurological disease. We are currently studying the biological effects of exogenous Tat on cells from the central nervous system (astrocyte and neuronal cell) and the lymphoid T cells (Jurkat). This study will elucidate the critical role of Tat in molecular pathogenesis of AIDS dementia leading to development of therapeutic tools for this disease.

#### 3) Development of protein transduction technology

Protein therapy is based on the delivery of target proteins into cells instead of traditional chemotherapy to treat human diseases. Based on the recent findings that abnormal activity of cellular proteins is responsible for the generation of a variety of human diseases, development of therapeutic agents could be achieved by regulating the biological activity of these proteins. Nevertheless proteins are superior to other chemicals in the selectivity and efficacy of their action mode, utilization of these proteins as the therapeutic drugs is severely limited due to the difficulty of delivery of therapeutic proteins into cells. To overcome this problem, it is necessary to develop a novel technology by which a variety of biologically active proteins could be delivered into cells. This protein transduction technology is based on the previous findings that exogenous HIV-1 Tat protein was able to translocate through the plasma membrane and to reach both the cytosol and nucleus in the cell. By exploiting this property of Tat, we are currently developing a system for the delivery of biologically functional peptide/protein into target cells or tissues.

#### 4) Directions for the anti-inflammatory drug discovery

- Utilize cell-based assays and in vivo testing strategies for the development of anti-inflammatory drugs.
- is initially directed towards discovering inhibitors from natural products and recombinant proteins.

### ■ Patents (2015~ 현재)

#### 1) 미국특허등록

- (1) Eye drop composition for prevention and treatment of ophthalmic diseases containig fusion protein of FK506 Binding Protein  
등록일 2015.2.24 등록번호 8,962,567

## 2) 국내특허등록

- (1) 세포투과성 NQO-1 융합단백질을 포함하는 뇌허혈 치료용 약학조성물  
등록일: 2015.11.14 등록번호: 10-1677449
- (2) 글루타레독신 1 융합단백질을 함유하는 관절염 예방 및 치료용 약학 조성물  
등록일: 2016. 10. 26 등록번호: 10-1671197
- (3) 세포투과성 NQO1 융합단백질을 함유하는 뇌허혈 치료용 약학 조성물  
등록일: 2016. 11. 14 등록번호: 10-1677449
- (4) 세포투과성 PIM2 융합단백질을 포함하는 파킨슨병 예방 및 치료용 약학 조성물  
등록일: 2017. 04. 26 등록번호: 10-1732349
- (5) PEA15 융합단백질을 함유하는 관절염 예방 및 치료용 약학 조성물  
등록일: 2017. 07. 20 등록번호: 10-1761550
- (6) 사이토카인 유도 세포자기사멸 저해제 1 융합단백질을 포함하는 항염증 약학 조성물  
등록일: 2018. 06. 15 등록번호: 10-1869686