induced by those supernatants. Separately, we assessed their potential as a diagnostic method to identify the presence of VacA by immunohistochemistry in both the liver tissue and the stomach tissue of patients with the corresponding cancer, respectively. These data indicate that this recombinant VacA has function and structure similar to those of native VacA, which could be useful in exploring the pathogenic role, especially correlation with hepatic diseases and developing a potential vaccine and a diagnostic kit.

*Corresponding Author : Professor, Kyu Shik Jeong, D.V.M., Ph.D.,
Department of Pathology, College of Veterinary Medicine, Kyungpook National University, 702-701, #1370, Sangyeok-dong, Buk-ku, Daegu City, Republic of Korea
Phone +82+53+950+5975, Fax +82+53+950+5955, E-mail jeongks@mail.knu.ac.kr

P#37

JNK1 Regulates α-Smooth Muscle Actin Expression by Transcriptional Activation and Ubiquitin-Dependent Degradation During Renal Fibrosis

Sang-Joon Park[1,2], Sun Hee Do[2], Da-Hee Jeong[2], IL-Hwa Hong[2], Eun-Joo Kim[1], Si-Yun Ryu[3], Tae-Hwan Kim[2] and Kyu-Shik Jeong[2]*

[1]Korea Institute of Toxicology, Korea Research Institute of Chemical Technology, Daejeon, Republic of Korea, [2]Department of Veterinary Pathology, College of Veterinary Medicine, Kyungpook National University, Daegu, Republic of Korea, [3]Department of Veterinary Anatomy, College of Veterinary Medicine, Chungnam National University, Daejeon, Republic of Korea

Myofibroblast, a specialized fibroblast characterized by expression of α-smooth muscle actin (α-SMA), play a major role in many tissue injuries and participate actively in the fibrotic process through extracellular matrix(ECM) production. Previously, we reported that phosphorylated form of c-Jun N-terminal kinase (JNK) was colocalized with α-SMA in myofibroblast during progressive renal disease. In the present study, to elucidate relationship between JNK and α-SMA in renal fibrosis, we investigated the kinetics of myofibroblast during the progression of renal fibrosis in rat using specific phosphorylated JNK (p-JNK) and α-SMA antibody, and elucidated regulatory mechanisms of α-SMA using wild type or deficiency fibroblast of JNK1 and JNK2. In a time-course in vivo study, a marked increase in p-JNK and α-SMA in both interstitial myofibroblast was shown in the progression stage of renal fibrosis, but decreased in end-stage renal fibrosis. Interestingly, overexpression of JNK1 and JNK2 reduced the expression of α-SMA gene, but increased α-SMA promoter activity in transfected cell line. While, JNK1-/- and JNK2-/- fibroblast increased the expression of α-SMA. Furthermore, we demonstrated that both the degradation and ubiquitination activity of α-SMA were more significantly increased by overexpression of JNK1 than JNK2. Collectively, our results suggest that α-SMA by JNKs is activated in the transcription level, but degradated in posttranslation level

by ubiquitin-dependent pathway. Regulation of α-SMA by JNK signaling pathway may lead to new approaches in the treatment of progressive renal fibrosis

*Corresponding Author :Professor, Kyu Shik Jeong, D.V.M., Ph.D.,
Department of Pathology, College of Veterinary Medicine, Kyungpook National University, 702-701, #1370, Sangyeok-dong, Buk-ku. Daegu City, Republic of Korea
Phone +82+53+950+5975. Fax +82+53+950+5955, E-mail jeongks@mail knu ac kr

P#38

A Functional Genomic Screen for Cardiogenic Genes Using RNA Interference in Developing Drosophila Embryos

Sang-Joon Park, Yong-Ou Kim, Robert S. Balaban, Marshall Nirenberg, Yongsok Kim
Office of the Director, Laboratory Research Program, Laboratory of Cardiac Energetics, and Laboratory of Biochemical Genetics, National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, MD 20892

Identifying genetic components is an essential step toward understanding complex developmental processes. The primitive heart of the fruit fly, the dorsal vessel, which is a hemolymph-pumping organ, has provided a unique model system to identify cardiogenic genes and to further our understanding of the molecular mechanisms of cardiogenesis. Using RNA interference in developing *Drosophila* embryos, we performed a genomewide search for cardiogenic genes. Through analyses of the >5,800 genes that cover 40% of all predicted *Drosophila* genes, we identified a variety of genes encoding transcription factors and cell signaling proteins required for different steps during heart development. Analysis of mutant heart phenotypes and identified genes suggests that the *Drosophila* heart tube is segmentally patterned, like axial patterning, but assembled with regional modules. One of the identified genes, *simjang*, was further characterized. In the *simjang* mutant embryo, we found that within each segment a subset of cardial cells is missing. Interestingly, the *simjang*gene encodes a protein that is a component of the chromatin remodeling complex recruited by methyl-CpG-DNA binding proteins, suggesting that epigenetic information is crucial for specifying cardiac precursors. Together, these studies not only identify key regulators but also reveal mechanisms underlying heart development and disease.

P#39

Overexpression of SMP30 Inhibit Radiation-Induced Apoptosis in Smad3-Knockout Mice Liver

Da-Hee Jeong[(1)], Il-Hwa Hong[(1)], Sun-Hee Do[(1)], Hai-Jie Yang[(1)], Dong-Wei Yuan[(1)], Mi Na Seo[(1)], Jae-Chung Jo[(1)], Seung-Sook Lee[(2)], Won-Suk Jang[(2)], Tae-Hwan Kim[(1)] and Kyu-Shik Jeong[(1)]
[(1)]Department of Veterinary Pathology, College of Veterinary Medicine, Kyungpook National University, Daegu, Republic of Korea and