Abstract

Chronic inflammation caused by persistent infection of hepatitis virus inside hepatocyte is one of the main causes of the hepatic cellular carcinoma, even though the direct role of inflammation on the hepatic carcinogenesis has not been clearly elucidated. Bacteria or virus infection evokes systemic response of the body, which involves multi-cellular interactions with diverse inflammatory mediators. One of the well-known signal transducer and transcription factors of inflammation, Stat3, has been suggested to cause cellular malignancy, as constitutively activated Stat3 expression frequently coincides with tumor development in human cancer patients. Interestingly, forced expression of Stat3\(\beta\), an alternatively spliced isoform of Stat3\(\alpha\), has been reported to inhibit cellular proliferation and induce programmed cell death in several human tumors and tumor-derived cell lines. Stat3\(\beta\) acts as transcriptional suppressor or activator, depends on the context of promoter region where it binds. To assess the contribution of aberrant Stat3 activation, we have generated Stat3\(\beta\) knockout mice. Specific ablation of Stat3\(\beta\) production altered the patterns of global gene regulation during systemic inflammation and elicited severely perturbed inflammatory response against pathogenic infection in the mouse. As the results, Stat3\(\beta\) knockout mice exhibited hyper- and chronic inflammatory response upon LPS administration. Both chronic inflammation caused by persistent viral infection and the development of cancer must evade from the immune surveillance capability of the body. The molecular mechanism of the immune tolerance caused by pathogenic infection or tumorigenic antigen has not been fully characterized, though accumulating data suggests that the inflammatory signal mediated by NFKB and Stat3 of the innate immune response play critical roles for this regulation.

The ultimate goal of our study is to develop a novel therapeutic approach that breaks immune tolerance, through the understanding of the molecular mechanism of inflammatory intervention of cancer development in the liver.
Dr. Joo-Yeon Yoo joined the Department of Life Science at POSTECH as an assistant professor in 2004, after serving as a research associate at the Johns Hopkins University. Dr. Yoo was trained as a postdoctoral fellow at the same school and was a research fellow of Howard Hughes Medical Institute. Dr. Yoo is a graduate of Seoul National University and earned her Ph.D. degree at the University of Maryland in 1997.

Present Position
Assistant Professor, Department of Life Sciences, Pohang University of Science and Technology (POSTECH)

Education
1989 B.A. Department of Botany, Seoul National University, Korea.
1991 M.S. Department of Botany, Seoul National University, Korea
1997 Ph.D. Program in Molecular and Cellular Biology, University of Maryland, USA

Professional experiences
1997-2002 Post-doctoral fellow, HHMI/Department of Molecular Biology and Genetics,
The Johns Hopkins University, USA
2003 Research Associate, Department of Molecular Biology and Genetics,
The Johns Hopkins University, USA
2004-present Assistant professor, Department of Life Science, POSTECH

Selected publications
S. A. 99:9015-9020. (* Both authors share co-first authorship). 