World Academy of Science, Engineering and Technology
International Journal of Biomedical and Biological Engineering
Vol:10, No:05, 2016

Role of Tyrosine-Phosphorylated STAT3 in Liver Regeneration: Survival, DNA Synthesis, Inflammatory Reaction and Liver Mass Recovery

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Abstract : In liver regeneration, quiescent hepatocytes need to be primed to fully respond to growth factors such as hepatocyte growth factor. To understand the priming process, it is necessary to analyze patterns of gene expression that occur during liver regeneration after partial hepatectomy (PHx). Recently, tyrosine phosphorylation of signal transducer and activator of transcription 3 (pYSTAT3) has been shown to play an important role in initiating liver regeneration. In order to evaluate the role of pYSTAT3 on liver regeneration after PHx, we used an intrabody which can selectively inhibit pYSTAT3. In our previous studies, an intrabody had been shown that it bound specifically to the pYSTAT3. Adenovirus-mediated expression of the intrabody in HepG2 cells, as well as mouse liver, blocked both accumulation of pYSTAT3 in the nucleus and downstream target of pYSTAT3. In this study, PHx was performed on intrabody-expressing mice and the expression levels of liver regeneration-related genes were analyzed. We also measured liver/body weight ratios and the related cellular signaling pathways were analyzed. Acute phase response genes were reduced in an intrabody-expressing mice during liver regeneration than in control virus-injected mice. However, the time course of liver mass restoration in intrabody-expressing mice was similar to that observed in control virus-injected mice. We also observed that the expression levels of anti-apoptotic genes, such as Bcl2 and Bcl-xL were decreased in intrabody-expressing mice whereas the expression of cell cycle-related genes such as cyclin D1, and c-myc was increased. Liver regeneration after PHx was partially impaired by the selective inhibition of pYSTAT3 with a phosphorylation site-specific intrabody and these results indicated that pYSTAT3 might have limited role in liver mass recovery.

Keywords : STAT3, pYSTAT3, liver regeneration, intrabody
Conference Title : ICMBB 2016 : International Conference on Molecular Biology and Biomedicine
Conference Location : Tokyo, Japan
Conference Dates : May 26-27, 2016