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

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P#38

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

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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.

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

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Apoptosis occurs early after irradiation, which may be a better indicator of radiation damage in various tissues. Recent evidence has indicated that apoptotic cells may be actively involved in suppression of inflammatory responses by inducing the anti-inflammatory cytokines and TGF-β1. Because elevated levels of TGF-β are associated with radiation-induced inflammation and fibrosis, the mice null for Smad3, a key downstream mediator of TGF-β, show accelerated healing of cutaneous injury with reduced inflammation and accumulation of matrix. Therefore, we hypothesized that loss of Smad3 will decrease liver damage induced by irradiation. To evaluate resistance to the radiation-induced liver injury in the Smad3-null mice, we determinate the incidence of apoptosis and expression of senescence marker protein-30 (SMP30), as an anti-apoptotic marker after irradiation to the liver.

Livers of Smad3-mutant mice were exposed to local irradiation of 0 and 15 gray (Gy), at a dosage rate of 1.95 Gy/min from a Co^{60}-gamma radiation source unit. In Smad3-WT mice of irradiated group body weights were significant decreased at 1week after irradiation. In Smad3-KO mice of irradiated group, however, change of body weight was mild compared to those of irradiated WT mice. At 1 week after irradiation, radiation-induced apoptosis of Smad3-KO mice was produced lower levels than that of WT mice liver. These findings were correlated with expressions of CYP2E1, which play as a role in hepatic injury produced by oxidative stress. In addition, antioxidant related protein, SMP30 levels were reduced by gamma irradiation in both irradiated mice. However, highly increased expression of SMP30 in Smd3-KO mice liver was preserved higher level than that of WT mice after irradiation.

Therefore, these results suggest that interruption of TGF-β signaling by deletion of Smad3 bring about inhibition of hepatic apoptosis after exposure to ionizing irradiation. Moreover, protective effect to ionizing radiation might be correlated with overexpression of SMP30 in the Smad3 null mice, act as an anti-apoptotic signaling molecule. Alteration of SMP30 by interruption of Smad3 might be useful therapeutic target for the radiation-induced liver injury.

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Canine Multiple Intestinal Lymphomatous Polyposis

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