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Response gene to complement 32 in cardiovascular diseases

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Abnormal smooth muscle cell (SMC) differentiation and phenotypic modulation play important roles in the development of several prominent cardiovascular diseases such as atherosclerosis, intimal hyperplasia, vein graft stenosis, restenosis following angioplasty, and aortic aneurysm. SMC phenotypic modulation is an important process in vascular remodeling following injury. Response gene to complement 32 (RGC-32) is originally identified as a cell cycle regulator in SMC, endothelial, and various cancer cells. We find that RGC-32 is an important regulator of smooth muscle differentiation and phenotypic modulation. RGC-32 promotes injury-induced lesion formation through stimulation of SMCproliferation and migration. In addition, RGC-32 is involved in epithelial-mesenchymal transition (EMT) and fibroblast activation, two important processes implicated in organ fibrosis. Knockout of RGC-32 in mice reduces the infarcted area in heart and decreases collagen expression in the infarcted area after ischemia-induced myocardial infarction. It appears that RGC-32 plays a critical role in cardiac fibroblast activation, leading to an enhanced collagen deposition, which eventually causes cardiac fibrosis.

Biography

Shiyou Chen has completed his Ph.D. from Nanjing Agricultural University in China and postdoctoral studies from University of Iowa School of Medicine and National Institutes of Health. He is an Associate Professor in the department of Physiology and Pharmacology, University of Georgia. He has published more than 40 papers in reputed journals and serving as a reviewer for several reputed scientific journals and grant agencies including American Heart Association and National Institutes of Health.

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