INHIBITION OF NF-KB P65 BY RIBOZYME GENE TRANSFER WITH ADENO-ASSOCIATED VIRUS SEROTYPES 9 ATTENUATES POST-INFARCT LEFT VENTRICULAR RUPTURE AND REMODELING IN AGED MICE

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Authors: Yi Tong Ma, Yang Xiang, Yining Yang, Fen Liu, Bangdang Chen, Xia Gao, Lei Du, Department of Cardiology, The First Affiliated Hospital, Xinjiang Medical University, Urumqi, Xinjiang, People’s Republic of China, Cardiovascular Research Institute, The First Affiliated Hospital, Xinjiang Medical University, Urumqi, Xinjiang, People’s Republic of China

Background: Activation of NF-κB is involved in cardiac remodeling, we examined whether inhibition of NF-κB p65 would prevent post-infarct left ventricular rupture and remodeling in aged mice.

Methods: Recombinant adeno-associated virus serotypes 9 carrying ribozyme gene (rAAV9-R65), which inhibiting NF-κB activity, was injected into 18-month-old C57BL/6 mice via tail vein. Sixteen days later myocardial infarction (MI) was induced by ligation of the left coronary artery. Incidence of rupture, cardiac remodeling by echocardiography, NF-κB activity, expression of p65, TNF-α, MMP-9 were examined.

Results: Administration of rAAV9-R65 inhibited NF-κB activity at day 7 post MI, which was associated with a lower rupture incidence (15.2% vs 32.8%, P=0.018), reduced LV dilation and preserved FS when compared to untreated group (P<0.05). MMP-9 and TNF-α were decreased in rAAV9-R65-treated mice (P<0.05).

Conclusions: Cardiac rupture and remodeling were attenuated in aged mice by rAAV9-R65. It maybe caused by decreased collagen degradation as the result of decreased MMP-9, TNF-α which proved that NF-κB signal pathway may be associated with cardiac rupture and remodeling in aged mice.