P24 Compensatory Effect of IGF-I Survival Signaling Is Reduced by Aging to Accelerate Apoptosis in Cardiac Cells Exposed to Second-Hand Smoke

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Cardiac hypertrophy is an enlargement of the heart relative with in cardiac myocyte volume in secondhand smoke exposure and age. However, pathological and physiological cardiac hypertrophy are regulated by hormone-insulin like growth factor-I (IGF-I) and cell death program (TNFα and Fas-L) in the heart. Physiological hypertrophy is up-regulation in early advance age and pathological is up-regulated in secondhand smoke exposure. IGF-I induced cardiac growth in physiological hypertrophy, but failed to show induction of cardiac pathological hypertrophy. In contract, we found that cell death program including through mitochondrial was expressed in pathological hypertrophy. Our results support the hypothesis that IGF-I induced cell growth in old age-induced hypertrophy and down-regulation in secondhand smoke exposure, and downstream PI3K/AKT signaling pathway. Interestingly, we found physiological program and pathological is resistant to each other in secondhand smoke exposure and in old age-induced myocardium hypertrophy.

Key Words: cardiac hypertrophy, physiological hypertrophy

IGF-I的生存信號減少老化加速細胞凋亡的心肌細胞暴露於二手煙的補償效果

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