Aging reduces the IGF-I Compensated signaling and accelerate the cardiac apoptoic effects induced by Second-hand smoke exposure

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Abstract:

Background:

Exposure to secondhand smoke (SHS) increased the risk of heart diseases including atherosclerosis and coronary disease. Aging is a physiology process involving progressive impairment of normal heart functions, due to an increasing vulnerability, which reduces the ability of survive. However, it is not clear pathological condition in aging exposure to SHS. The aim of this study was to examin SHS exposure in aging-related death-survival imbalance of rat hearts.

Methods:

The young SD rats (3 months) and aging SD rats (24 months) were subjected into non-smoking and smoking exposure. All animals were divided into four groups: MYC (male-young-non-smoking group), MYS (male-young-smoking

group), MOC (male-old-non-smoking group) and MOS (male-old-smoking group). The smoking groups were placed in SHS exposure chamber and exposed to 10 cigarettes for 30 min, twice a day, 5 days per week for 1 month. After 4 weeks secondhand smoke exposure, rats left ventricular (LV) underwent morphological and function study with echocardiography. Histopathologic of left ventricular sections were stained with Hematoxylin-Eosin staining and related death-survival protein expression levels evaluated by Western blot analysis.

Results:

After 4 weeks SHS exposure, LV weight showed significantly increas in MYS and MOC groups and showed greater synergistic effect in MOS group. Similarly results were observed from echocardiography analysis, The EF (%) and FS (%) were apparently decrease in young SHS exposure and aging group, and even synergistic enhanced in MOS group. The IVS, LVID and LVPW displayed the similar findings. Morever, we found the upregulation of Fas-dependent apoptosis pathway, TNFα-Fas-L-Fas-FADD-cleaved caspase 8 and mitochondrial-dependent apoptosis related proteins, cytochrome c, t-Bid, Bid, Bad, cleaved-caspase 9 in MYS and MOC groups and synergistically enhanced in MOS group. In addition, the IGF-I/IGFIR and p-PI3K/p-Akt survival signaling pathways were compensated increase in MYS and MOC groups, but totally suppressed in MOS group.

Conclusions:

Our study strongly suggest that aging and SHS synergistically enhanced apoptosis related pathways. However, aging under SHS exposure totally compromised the compensative survival signalings of rat hearts.