Aging reduces the IGF-I compensated signaling and accelerates the cardiac apoptotic effects induced by Second-hand Smoke

Jia-Ping Wu[1], Wei-Wen Kuo[2,3,4], Yu-Lan Yeh[3,4], Mu-Hsin Chang[1], Fuu-Jen Tsai[5], Chang-Hai Tsai[6], Chih-Yang Huang[1,9,10]

[1]Granduate Institute of Basic Medical Science, China Medical University, Taichung, Taiwan, R. O. C.

[2]Department of Biotechnology, China Medical University, Taichung, Taiwan

[3]Department of pathology, Changhua Christian Hospital, Changhua

[4]Department of Medical Technology, Jen-The Junior College of Medicine, Nursing and Management, Miaoli, Taiwan.

[5]Department of Pediatrics, Medical Research and Medical Genetics, China medical University Taichung, Taiwan

[6]Department of Healthcare Administration, Asia University, Taiwan

[9]Graduate Institute of Chinese Medical Science, China Medical University, Taichung, Taiwan.

[10]Department of Health and Nutrition Biotechnology, Asia University, Taichung, Taiwan

Cardiac hypertrophy is an enlargment of the heart relative with in cardiac myocyte cell volume in secondhand smoke exposure. The aim of this study was to examine the association between the age rats exposure to secondhand smoke and cardiovascular disease death. And to determine whether the age is affected on ventricular remodeling process induced by SHS (secondhand smoke) exposure in rats. However, it is not clear pathological condition in elderly age by SHS exposure. In this study, we investigated the relations of survival and apoptosis signals in SHS exposure and old age. The rats were placed in exposure chamber and exposed to 10 cigarettes and smoke for 30 min, twice a day, 6 days/ week for 1 month. Left ventricular morphological variables assessed on echrocardiography, histopathologic of left ventricular sections was stained with Hematoxylin-eosin staining, and protein expression levels results is detected by Western Blot. Left ventricular internal dimension at end diastolic(LVIDd) in young age and old age exposure to SHS for 4 weeks increased to 4.3±0.03($p<0.05$)(MYC; 2.6±0.3, MOC; 3.72±0.4), left ventricular internal dimension at end systemic(LVIDs) in MYS increased to 3.0±0.00($p<0.01$), in MOC increased to 2.5±0.3($p<0.05$), in MOS increased to 3.3±0.03($p<0.05$) for 4 weeks SHS exposure(MYC; 1.8±0.1). Left ventricular posterior wall thickness in systemic(LVPWs) and Interventricular septal in systemic(IVSs) in MOS increased to 2.0±0.0($p<0.05$)(MYC; 1.7±0.0 and 1.6±0.0, respectively). However, EF(%) and FS(%) are decreased in young and old age. In addition, Western blot represent survival signaling pathway(IGF-I-IGF-IR-p-PI3K/p-Akt) are compensatory cardiac growth to show cardiac hypertrophy. In contrast, apoptosis signaling pathway (TNFα, Fas-L, Fas, FADD, active caspase 8, active caspase-3, active caspase-9, bad, cytochrome c, and bid, t-bid) are increased their expression in SHS-exposured in young and old age. Overall, we think SHS and aged bloth enhanced left ventricular hypertrophy and cell death.

Keyword: apoptosis, survival signaling pathway, pathological hypertrophy, left ventricular.

年齡減少 IGF-I 的代償訊號並加速抽煙所誘導的心臟凋

吳嘉平 [1]，郭薇雯 [2,3,4]，蔡輔仁 [5]，蔡長海 [6]，玉蘭 [3,4]，木新 [1]，黃志揚 [9,10]

[1] 台中，中國醫藥大學，基礎醫學研究所

[2] 台中，中國醫藥大學，生物技術

[3] 彰化市，彰化基督教醫院，病理

[4] 台中市，輔仁的醫學，護理和管理

[5] 台中市，中國醫藥大學，醫學研究和醫學遺傳學

[6] 台中縣，亞洲大學，台灣醫務管理

[9] 台中市，中國醫藥大學，中國醫學科學研究所

10 台中縣，亞洲大學，保健營養生技