EXPERIMENTAL RESEARCH

1. Expression of Calcium Sensing Receptor in Atherosclerosis Rat Vascular Endothelial Cells. PEI Tian-Xian, WANG Jing-Jing, GUO Jing-Yue, GUO Chuan-Min, HU Man, and XU Chang-Qing. CHINESE JOURNAL OF ARTERIOSCLEROSIS 2011; 19(3):176


4. Proprotein Convertase Subtilisin/Kexin 9 siRNA Repressed Inflammation Factor Expression of THP-1 Macrophage Cells Induced by Oxidized Low Density Lipoprotein. TANG Zhi-Han, JIANG Lu, REN Zhong, LIU Lu-Shan, and JIANG Zhi-Sheng. CHINESE JOURNAL OF ARTERIOSCLEROSIS 2011; 19(3):192

5. Effect of Shensongyangxin Capsule on QT Dispersion and Connexin43 Expression in Heart Failure Rats. YANG Jun, ZHOU Xian-Ling, CHU Chun, PENG Wei-Rui, TAN Fang, DING Sai-Liang, and TAN Xiao-Jin. CHINESE JOURNAL OF ARTERIOSCLEROSIS 2011; 19(3):197

6. Effect of Water Extract Propolis on Apoptosis of Human Umbilical Arterial Smooth Muscle Cells Induced by ox-LDL. WANG Jian-Li, XU Xing-Hua, SI Yan-Hong, SANG Hui, YU Feng-Xiu, WANG Jia-Fu, and SHANG Zhan-Ping. CHINESE JOURNAL OF ARTERIOSCLEROSIS 2011; 19(3):202


CLINICAL RESEARCH

9. The Analysis of the Risk Factor for Asymmetric Leukoaraiosis. LI Bei-Bei, WEI Wei, ZHANG Wei-Wei, CHEN Hui, ZHU Guang-Ming, HUANG Yong-Hua, ZHAO Xiu-Xin, LI Juan, and
1. Expression of Calcium Sensing Receptor in Atherosclerosis Rat Vascular Endothelial Cells

PEI Tian-Xian1, WANG Jing-Jing1, GUO Jing-Yue1, GUO Chuan-Min1, HU Man1, and XU Chang-Qing2
(1. Tianjin Institute of Pharmaceutical Research, Tianjin 300193, China; 2. Department of Pathophysiology, Harbin Medical University, Harbin, Heilongjiang 150086, China)

[KEY WORDS] Calcium Sensing Receptor; Rat; Atherosclerosis; Apoptosis; Endothelial Cell

[ABSTRACT] Aim: To observe the expression of calcium sensing receptor (CaSR) in atherosclerosis rat vascular endothelial cells and the relationship with apoptosis.

Methods: 144 Wistar rats were randomly divided into control group (n=72) and atherosclerosis groups (n=72). Early atherosclerosis models were copied by injection of vitamin D3 360 million units/kg body weight, and then fed with high fat diet for 6 weeks. Serum triglyceride (TG) and total cholesterol (TC) were detected by automatic biochemistry analyzer. The morphological changes were observed under optical microscope. The mRNA and protein expressions of CaSR, Bcl2, Bax and Caspase3 were analyzed by Western blotting and RTPCR. Apoptotic cells were measured by TUNEL staining.

Results: Compared with control group, the apoptosis index and the expression of CaSR, Bax and Caspase3 were increased, Bcl2 expression were decreased, the changes in aorta thoracica were aggravated in the atherosclerosis group.

Conclusion: CaSR may participate in the apoptosis of vascular endothelial cell, and then accelerate the formation of atherosclerosis.

2. Effect of 2,3,5,4′ Tetrahydroxystilbene2-O-β-D Glucoside on Hydrogen Peroxide Induced Apoptosis of Human Umbilical Vein Endothelial Cell

LONG Shi-Yin, HUANG Liang-Zhu, QIAO Xin-Hui, ZHANG Cai-Pin, GAO Xi-Qiang, TONG Li, and TIAN Ying
(Department of Biochemistry and Molecular Biology, University of South China, Hengyang, Hunan 421001, China)

[KEY WORDS] Tetrahydroxystilbene2-O-β-D Glucoside; Human Umbilical Vein Endothelial Cells; Apoptosis; Caspase3

[ABSTRACT] Aim: To investigate the role and mechanism of TSG on the apoptosis of human umbilical vein endothelial cells (HUVEC) induced by H2O2. Methods: HUVEC were treated with TSG (0, 1, 10 and 100 μ mol/L) for 24 hours then exposed to H2O2 (100, 200, 300, 400 and 500 μ mol/L) for 24 hours, the optimal concentration of H2O2 and TSG were deducted by MTT and Flow Cytometry. Morphology of apoptosis and the protective effect of TSG on HUVEC induced by H2O2 were observed by Hoechst33258
staining. The mRNA and protein expression of Caspase3 were detected by RT-PCR and Western blot analysis respectively. Results: According to the MTT and Flow Cytometry, the optimal concentration for H2O2 to establish apoptosis model and for TSG to protect HUVEC induced by H2O2 were 300 μmol/L and 10 μmol/L respectively. Compared with the control group, the group of 300 μmol/L H2O2 inhibited the cell proliferation, increased the number of apoptotic cells and the expression of Caspase3 significantly. Compared with H2O2 group, 10 μmol/L of TSG improved the rate of cell proliferation, inhibited cell apoptosis, and decreased expression of the caspase3 significantly (P<0.05). Conclusion: TSG could inhibit H2O2 induced apoptosis of HUVEC, and its mechanism was associated with the inhibition of Caspase3 expression.

3. Effects of Astragali Radix Extract on the Vascular Cell Adhesion Molecule Expression of Mice Vascular Endothelial Cell Induced by Tumor Necrosis Factorα

YOU Yang1, 2, DUAN Yan2, ZHANG Xiao-Lin2, KANG Jian2, YAN Cheng-Hui2, ZHANG Xiu-Li3, FENG Jia-Tao3, and HAN Ya-Ling2

(1. The Affiliated Hospital of Liaoning University of TCM Shenyang, Liaoning 110032, China; 2. Cardiovascular Research Institute, General Hospital of Shenyang Military Region Shenyang, Liaoning 110840, China; 3. Dalian Institute of Chemical Physics, Chinese Academy of Sciences Dalian, Liaoning 116023, China)

[KEY WORDS] Astragali Radix Extract; Tumor Necrosis Factorα; Nuclear Factor-κ B; Vascular Cell Adhesion Molecule

[ABSTRACT] Aim: To investigate the protective effect of Astragali radix extract on vascular cell adhesion molecule expression of mice vascular endothelial cell against tumor necrosis factorα (TNFα). Methods: Adhesion model was established by THP-1 cells and mice endothelial cell in vitro. The cells were pretreated by different dose and different time of Astragali radix extract before induced by TNFα, and the adhesion rate were detected. The levels of vascular endothelial cell adhesion molecule VCAM1 in the cell culture were determined with ELISA. The expression of VCAM1 and NF-κ B subunit (p65) were evaluated by Western blot. Results: The expression of VCAM1 and NF-κ B was increased obviously after induced by TNFα; While the expression of VCAM1 and the effect of NF-κ B protein nuclear translocation induced by TNFα were inhibited after pretreatment of Astragali radix extract in a dose and time dependent manner. The reduction of adhesion of monocytes to endothelial cells, the down regulation of the expression of VCAM1 and reduction of the expression of NF-κ B were apparent (P<0.05) at the concentration of 120 mg/L preincubated 4~8 h. Conclusion: Astragali radix extract can inhibit the TNFα induced expression of VCAM1 and reduce the adhesion of monocytes, by which the damage to vascular endothelial cells was relieved. The mechanism may be related to the role of inhibiting the activation of NF-κ B.

4. Proprotein Convertase Subtilisin/Kexin 9 siRNA Repressed Inflammation Factor Expression of THP-1 Macrophage Cells Induced by Oxidized Low Density Lipoprotein

TANG Zhi-Han, JIANG Lu, REN Zhong, LIU Lu-Shan, and JIANG Zhi-Sheng

(Institute of Cardiovascular Disease, Key Laboratory for Arteriosclerology of Hunan
Aim: To investigate the effect of proprotein convertase subtilisin/kexin 9 (PCSK9) siRNA on inflammatory factor expression in oxidized low density lipoprotein (ox-LDL) induced THP-1 macrophage cells.

Methods: The siRNAs for PCSK9 were designed and synthesized, then THP-1 macrophages were transfected with 80 nmol/L PCSK9 siRNA by positive ion liposome Lipofectamine 2000 for 24 h and then a high level of ox-LDL (80 mg/L) for an additional 24 h. Reverse transcription polymerase chain reaction (RT-PCR) was conducted to detect interleukin-1α (IL1α) mRNA, interleukin-6 (IL6) mRNA and tumor necrosis factor-α (TNF-α) mRNA.

Results: Ox-LDL increased IL1α mRNA, IL6 mRNA and TNF-α mRNA expression. The expression of IL1α mRNA, IL6 mRNA and TNF-α mRNA in the macrophage pretreated with PCSK9 siRNA was decreased significantly, in comparison with ox-LDL treated group (P<0.05). Conclusion: PCSK9 siRNA can repress inflammation factor expression of THP-1 macrophage cells induced by ox-LDL. PCSK9 may be involved in adjustment for inflammation.

5. Effect of Shensongyangxin Capsule on QT Dispersion and Connexin43 Expression in Heart Failure Rats

YANG Jun, ZHOU Xian-Ling, CHU Chun, PENG Wei-Rui, TAN Fang, DING Sai-Liang, and TAN Xiao-Jin
(Department of Cardiology, the First Affiliated Hospital, University of South China, Hengyang, Hunan 421001, China)

Aim: To investigate the effect of Shensongyangxin capsule on QT dispersion and connexin43 expression in heart failure rats. Methods: Heart failure rats models were built by constricting abdominal aorta, and were lavaged with Shensongyangxin capsule for 8 weeks. Ventricular electrophysiology were measured by inserting homemade electrode into subcutaneous, left ventricular morphostructure, myocardial fibrosis, and connexin43 distribution were respectively observed by HE staining, Masson staining, immunohistochemical staining. Results: Heart failure rats QT dispersion were significantly longer (37.20±9.94 ms, P<0.05), cardiomyocytes were misaligned, myocardial fibrosis area were significantly increased (101217.30±33970.02 μm², P<0.05), and connexin43 distribution were significantly decreased (55.93±11.61, P<0.05). Shensongyangxin capsule can shorten QT dispersion (25.50±8.21 ms) of heart failure rats, increase connexin43 distribution (69.09±16.59) and decrease myocardial fibrosis area (13580.64±8213.73 μm²) in myocardium of heart failure rats. Conclusion: Shensongyangxin capsule can shorten QT dispersion of heart failure rats, increase connexin43 distribution and decrease myocardial fibrosis area in myocardium of heart failure rats.

6. Effect of Water Extract Propolis on Apoptosis of Human Umbilical Arterial Smooth Muscle Cells Induced by ox-LDL

WANG Jian-Li, XU Xing-Hual, SI Yan-Hong2, SANG Hui2, YU Feng-Xiu2, WANG Jia-Fu2,
and SHANG Zhan-Ping2
(1. Department of Pathophysiology, Jining Medical College, Jining, Shandong 272067, China; 2. Department of Pathophysiology, Taishan Medical College, Taian, Shandong 271000, China)

[KEY WORDS] Smooth Muscle Cell; Oxidized Low Density Lipoprotein; Water Extract Propolis; Cholesteryl Ester; Apoptosis

[ABSTRACT] Aim: To investigate the effect of water extract propolis (WEP) on apoptosis in human umbilical arterial smooth muscle cell (HUASMC), and explore the mechanism of water extract propolis on inhibition of atherosclerosis and it’s implications. Methods: HUASMC were cultured and identified by immunocytochemistry technique. Cultured HUASMC were randomly divided into 5 groups: control group, model group and WEP groups (treated with 50 mg/L, 100 mg/L and 200 mg/L WEP respectively). Intracellular total cholesterol (TC) and free cholesterol (FC) were measured by high performance liquid chromatogram, the content of cholesterol ester (CE) was obtained by subtracting the FC from TC. Cellular apoptosis index was tested by flow cytometry. Results: The content of intracellular CE in model group was more than that in control group (P<0.01). The content of intracellular CE in 50 mg/L, 100 mg/L and 200 mg/L WEP groups were less than that in the model group (P<0.01), and with the increase of the concentration of WEP the content of intracellular CE showed the decreasing tendency. The cell apoptosis index in model group was higher than that in control group (P<0.01). The cell apoptosis index in 50 mg/L, 100 mg/L and 200 mg/L WEP groups were lower than that in the model group (P<0.01), and with the increase of the concentration of WEP the cell apoptosis index showed the decreasing tendency. The content of intracellular CE was correlated with the cell apoptosis (r=0.964, P<0.01). Conclusion: WEP can reduce the apoptosis of HUASMC induced by ox-LDL which may be concerned with the inhibition of intracellular CE accumulation.

7. Knockdown of Soluble Epoxide Hydrolase by RNA Interference in Mice Cardiomyocytes
DU Guang-Sheng1,2, WEN Yuan1, and MA Ye-Xin1
(1. Department of Cardiology, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, Hubei 430030, China; 2. Department of Cardiology, the First Affiliated Hospital of Medical College, Shihezi University, Shihezi, Xinjiang 832000, China)

[KEY WORDS] Cardiomyocytes; Soluble Epoxide Hydrolase; RNA interference

[ABSTRACT] Aim: To construct the soluble epoxide hydrolase (sEH) gene specific recombinant vectors pSUPER retro neo, and selectively knockdown the expression of sEH in mice cardiomyocytes by RNA interference (RNAi), and to select the plasmids having the best silence effect to sEH. Methods: Two pairs of siRNA that target at sEH gene were designed, and to construct the siRNA expression vectors specific to sEH gene (EH1 and EH2). There were plasmids carrying a nonspecific siRNA coding sequence (pCN) as the negative control group and the blank control group involved. Then the plasmids were transfected into primary cultured mice cardiomyocytes by FuGENE HD. The mRNA and protein expressions of sEH were analyzed by RTPCR and Western Blotting respectively. Results: The expressions of mRNA and protein of sEH in EH2 group (0.202
±0.017 and 0.212±0.029, P<0.01) were significantly decreased compared with that of blank control group, negative control group, and EH1 group. Conclusion: Recombinant expression vectors have been constructed, and RNA interference can selectively knockdown sEH expression in cultured cardiomyocytes. This lays a foundation for further research of RNAi on cardiomyocytes.

8. Effect of Flavonoids from Cuscuta Chinensis on Myocardial Cells Apoptosis in Ischemic / Reperfused Rat Hearts
ZHAI Hong-Ying, and WANG Gui-Min
(Traditional Chinese Medicine, the First Affiliated Hospital of Liaoning Medical College, Jinzhou, Liaoning 121001, China)

[ KEY WORDS ] Flavonoids from Cuscuta Chinensis; Ischemia Reperfusion Injury; Apoptosis; Bcl2 Protein; Bax Protein

[ ABSTRACT ] Aim: To observe the effect of flavonoids from Cuscuta Chinensis on myocardial cell apoptosis and the related gene expression in ischemic/reperfused myocardial infarction. Methods: The left anterior descended coronary artery was ligated for 30 min and reperfused for 2 h to make ischemia reperfusion injury model. 50 SD rats were randomly divided into the sham operation group, ischemia reperfusion group, the low and high dose groups of flavonoids from Cuscuta Chinensis and isosorbide dinitrate group. Creatine kinase (CK) and lactate dehydrogenase (LDH) were assayed. The apoptotic index (AI) was analyzed by TUNEL staining. The protein expression of Bcl2 and Bax were measured by immunohistochemistry and Western Blotting in each group, the ratios of Bcl2 and Bax were calculated. Results: Compared with the sham operation group, the ischaemia reperfusion group of enzymes CK and LDH, AI, and Bcl2 and Bax protein expression were significantly higher (P<0.01); The enzyme CK and LDH, and AI in the low and high dose group of flavonoids from Cuscuta Chinensis were decreased compared with those of the ischaemia reperfusion group (P<0.01). The expression of Bcl2 was increased and the expression of Bax was decreased (P<0.01). The isosorbide dinitrate group showed no significant difference when compared with the high dose group of flavonoids from Cuscuta Chinensis (P>0.05). Conclusion: The pretreatment of flavonoids from Cuscuta Chinensis could dose dependently decrease enzymes CK, LDH levels, increase the expression of Bcl2 protein and Bcl2/Bax, reduce the expression of Bax protein, and inhibit the apoptosis of myocardial cell. The therapeutic effects was similar to isosorbide dinitrate.

9. The Analysis of the Risk Factor for Asymmetric Leukoaraiosis
LI Bei-Bei1,2, WEI Wei1, ZHANG Wei-Wei1, CHEN Hui1, ZHU Guang-Ming1, HUANG Yong-Hua1, ZHAO Xiu-Xin1, LI Juan1, and ZHAO Xiao-Ping1
(1. The Department of Nerology in the General Hospital of Beijing Amy Area, Beijing 100700; 2. The Second Clinical Institute of Shanxi Medical University, Taiyuan, Shanxi 030001, China)

[ KEY WORDS ] Asymmetric Leukoaraiosis; Symmetric Leukoaraiosis; Incidence; Risk Factor

[ ABSTRACT ] Aim: To analyze the risk factor of asymmetric leukoaraiosis (LA) and
explore its incidence and its pathogenesis. Methods: 266 consecutive patients elder than 40 years were included prospectively. Their clinical data were analyzed respectively. All of them had accepted a magnetic resonance imaging (MRI) and a Ultrasonic examination. The white matter hyperintensities (WMH) volume of fluid attenuated inversion recovery (FLAIR) were quantified with a semi automated method, Leukoaraiosis patients with a ratio of WMH volumes in heavy side and in light side >1.5 were considered as asymmetric LA, and numbers of lacunes were counted. Local factors were records for the analysis. Results: 32 cases of asymmetric leukoaraiosis were found, the incidence of asymmetric LA was 12.03%. Comparing the risk factor of asymmetric LA patients and symmetric LA, the two groups had significant difference in age, history of hypertension, diabetes, carotid artery plaques/stenosis, ischemic stroke, and the heavy side of asymmetric LA had more lacunar infarction, higher Crouse scale. Binary Logistic regression showed age, history of hypertension, lacunar infarction and carotid artery plaques/stenosis were included in the model. Conclusion: The risk factors for asymmetric LA are age, history of hypertension, lacunar infarction and carotid artery plaques/stenosis.

10. The Effect of Cyclosporine A on Serum Interferonγ, NO and NOS in Patients with Chronic Aplastic Anemia

Xiang Yong-Sheng, and Wang Long
(Department of Hematology, the First People’s Hospital of Jingmen, Jingmen, Hubei 448000, China)

[KEY WORDS] Aplastic Anemia; Interferonγ; Cyclosporine A

[ABSTRACT] Aim: To observe the effect of cyclosporine A on serum interferonγ (IFNγ), nitric oxide synthase (NOS) and nitric oxide (NO) in patients with chronic aplastic anemia (CAA). Methods: 60 CAA patients were randomly divided into two groups. Control group was given Yixuesheng, while observation group was given cyclosporine A on the basis of control group. Two groups serum IFNγ, NOS and NO before and after treatment were compared. Results: Control groups serum IFNγ (27.4±3.3 ng/L), NOS (30.8±4.0 kU/L) and NO (28.1±2.7 μ mol/L) after treatment had no obvious change. Observation groups indexes (21.7±2.5 ng/L, 22.3±3.6 kU/L and 9.5±1.8 μ mol/L) were obviously lower than before treatment and control group (P<0.05). Conclusion: Cyclosporine A can decrease serum NO through inhibition of bone marrow failure, and thus reduce the damage degree of oxygen free radical and protect vascular wall in a certain degree.