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吉首大学学报自然科学版 » 2008, Vol. 29 » Issue (6): 104-106 DOI:

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## 黄芪甲甙对大鼠阿霉素心肌细胞凋亡的影响

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### Effects of Astragaloside on Cardiomyocyte Apoptosis in Adriamycin-Induced Cardiomyopathy in Rats

(Medical College of Jishou University, Jishou 416000, Hunan China)

- 摘要
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**摘要** 目的 探讨黄芪甲甙对大鼠阿霉素心肌病心肌细胞凋亡及其端粒酶活性表达的影响.方法 雄性SD大鼠, 体重250 g左右.建立阿霉素心肌病模型, 随机分为黄芪甲甙干预组、模型组(阿霉素组)、正常对照组、正常大鼠黄芪甲甙对照组.用原位末端标记法(TUNEL)标记凋亡的心肌细胞, 用TRAP-PCR-ELISA法检测端粒酶活性.结果 阿霉素组凋亡指数明显高于对照组( $P < 0.05$ ), 黄芪甲甙干预组凋亡指数明显低于阿霉素组( $P < 0.05$ ), 但仍高于对照组( $P < 0.05$ ); 黄芪甲甙干预组与阿霉素组比较, 端粒酶活性明显升高( $P < 0.05$ ), 但仍低于对照组( $P < 0.05$ ).结论 心肌细胞凋亡是阿霉素心肌病的重要机制, 黄芪甲甙干预治疗可减少阿霉素心肌病的心肌细胞凋亡, 可能与黄芪甲甙能提高端粒酶活性有关.

**关键词:** 黄芪甲甙 阿霉素 心肌 凋亡 端粒酶

**Abstract:** Objective To explore the effects of Astragaloside on cardiomyocyte apoptosis and the expression of telomerase in adriamycin (ADR)-induced cardiomyopathy in rats. Methods Male SD rats weighing about 250 g were used to establish the model of Adriamycin-induced Cardiomyopathy, then randomized into groups: Astragaloside group, ADR group, control group and control+Astragaloside group. Apoptotic cardiomyocytes were detected using the terminal deoxynucleotidyl transferase mediated dUTP nick end labeling method (TUNEL). The expression of telomerase was determined by TRAP-PCR-ELISA method. Results Compared with control group, ADR group had significantly higher index of apoptotic cardiomyocytes ( $P < 0.05$ ). The apoptotic index in Astragaloside group was less than that in ADR group ( $P < 0.05$ ), however significantly higher than that in control group and control+Astragaloside group ( $P < 0.05$ ). The expression of telomerase in Astragaloside group was significantly higher than that in ADR group ( $P < 0.05$ ), however significantly lower than that in control group and control+Astragaloside group ( $P < 0.05$ ). Conclusions Myocardial apoptosis is an important mechanism of adriamycin-induced cardiomyopathy. Astragaloside therapy can inhibit cardiomyocyte apoptosis in adriamycin-induced cardiomyopathy, partly because it might increase expression of telomerase.

**Key words:** astragaloside adriamycin myocardium apoptosis telomerase

#### 基金资助:

湖南省卫生厅资助项目(2007171); 吉首大学校级重大项目(08ZDWT001)

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#### 引用本文:

钟飞,李辉,周卫华. 黄芪甲甙对大鼠阿霉素心肌细胞凋亡的影响[J]. 吉首大学学报自然科学版, 2008, 29(6): 104-106.

ZHONG Fei, LI Hui, ZHOU Wei-Hua. Effects of Astragaloside on Cardiomyocyte Apoptosis in Adriamycin-Induced Cardiomyopathy in Rats[J]. Journal of Jishou University (Natural Sciences Edit), 2008, 29(6): 104-106.

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- [1] 章敏, 吕宝经, 荣辉之.福辛普利对阿霉素中毒心肌的保护作用 [J].上海第二医科大学学报, 2001, 22(1):19-21.
- [2] OGAMI M, IKURA Y, OHSAWA M, et al.Telomere Shortening in Human Coronary Artery Diseases [J].Arteriosclerosis, Thrombosis, and Vascular Biology, 2004, 24:546-549. 
- [3] SIVESKI-ILISKOVIC N, HILL M, CHOW D A, et al.Probucol Protects Against Adriamycin Cardiomyopathy Without Interfering with Its Antitumor Effect [J].Circulation, 1995, 91(1):10-15.
- [4] 尹瑞兴, 杨德素, 李佳荃.心肌营养素-1对心肌梗死大鼠心功能和心肌细胞凋亡的影响 [J].中华心血管病杂志, 2005, 33(3):273.
- [5] MORIN G B.The Implications of Telomerase Biochemistry for Human Disease [J].Eur. J. Cancer,1997, 33(5):750-760. 
- [6] TATSUMOTO N, HIYAMA E, MURAKAMI Y, et al.High Telomerase Activity is an Independent Prognostic Indicator of Poor Outcome in Colorectal Cancer [J].Clin. Cancer. Res., 2000, 6(7):2 696-2 701.
- [7] NAKAMURA T, UEDA Y, JUAN Y, et al.Fas-Mediated Apoptosis in Adriamycin-Induced Cardiomyopathy in Rats:In Vivo Study [J].Circulation, 2000, 102(5):572-578.
- [8] KRUPP G, KLAPPER W, PARWARESCH R.Cell Proliferation,Carcinogenesis and Diverse Mechanisms of Telomerase Regulation [J].Cell Mol. Life Sci., 2000, 57(3):464-486. 
- [9] CHIANG Y J, HEMANN M T, HATHCOCK K S, et al.Expression of Telomerase RNA Template, But Not Telomerase Reverse Transcriptase, is Limiting for Telomere Length Maintenance in Vivo [J].Mol. Cell Biol., 2004, 24(16):7 024-7 031.
- [10] 任晓庆, 王方正, 浦介麟, 等.心肌细胞再生与心肌移植修复 [J].中华心律失常学杂志, 2004, 4(3):125-128.
- [11] KAJSTURA J, PERTOLDI B, LERI A, et al.Telomere Shortening is an in Vivo Marker of Myocyte Replication and Aging [J].Am. J. Pathol, 2000, 156(3):813-819. 
- [12] HAMEZ P, THORIN-TRESCASES N, MOREAU P, et al.Workshop:Excess Growth and Apoptosis:Is Hypertension a Case of Accelerated Aging of Cardiovascular Cells? [J].Hypertension, 2001, 37(2 Part 2):760-766.
- [13] BLASCO M A, GASSER S M, LINGNER J.Telomeres and Telomerase [J].Genes. Dev., 1999, 13(18):2 353-2 359.
- [14] ANVERSA P, KAJSTURA J.Ventricular Myocytes are Not Terminally Differentiated in the Adult Mammalian Heart [J].Circ. Res.,1998, 83(1):1-14.
- [15] 钟飞, 杜九中, 于小华.黄芪甲甙对病毒性心肌炎细胞凋亡作用的研究 [J].南华大学学报: 医学版, 2004, 32(1):182-183.
- [16] 李丽, 陶辉宇, 陈杰斌,等.黄芪甲苷保护阿霉素心肌损伤的抗氧化机制研究 [J].临床儿科杂志, 2007, 25(1):58-61.
- [1] 李双杰, 钟飞, 张召才, 陈瑞珍, 杨英珍, 陈灏珠, 葛均波. **病毒性心肌疾病的基因时空表达特征**[J]. 吉首大学学报自然科学版, 2008, 29(6): 107-111.