

论著

扩展功能

本文信息

- ▶ [Supporting info](#)
- ▶ [PDF\(410KB\)](#)
- ▶ [\[HTML全文\]\(0KB\)](#)

▶ 参考文献

服务与反馈

- ▶ [把本文推荐给朋友](#)
- ▶ [加入我的书架](#)
- ▶ [加入引用管理器](#)
- ▶ [复制索引](#)
- ▶ [Email Alert](#)
- ▶ [文章反馈](#)
- ▶ [浏览反馈信息](#)

相关信息

▶ [本刊中包含“六价铬”的相关文章](#)

▶ 本文作者相关文章

- [杨渊](#)
- [刘新民](#)
- [肖芳](#)
- [李鹏](#)
- [邹悦](#)
- [戴璐](#)
- [钟才高](#)

六价铬对肝细胞内活性氧和腺苷酸转运体1转录水平的影响

杨渊^{1, 2}, 刘新民¹, 肖芳¹, 李鹏¹, 邹悦¹, 戴璐¹, 钟才高¹

1. 中南大学公共卫生学院卫生毒理学系, 湖南 长沙 410078;

2. 湖南省怀化医学高等专科学校 医学检验系, 湖南 怀化 418000

收稿日期 2011-6-28 修回日期 2012-7-13 网络版发布日期 2012-10-11 接受日期

摘要 目的 探讨重金属六价铬[Cr(VI)]的体外肝毒性。方法 L-02肝细胞经Cr(VI) 0, 2, 4, 8, 16和32 $\mu\text{mol} \cdot \text{L}^{-1}$ 分别染毒12, 24或36 h后, 采用逆转录-荧光定量聚合酶链反应(RT-qPCR)和荧光光度法分别对腺苷酸转运体1(ANT1)mRNA表达水平和活性氧簇(ROS)水平进行检测。结果 Cr(VI) 32 $\mu\text{mol} \cdot \text{L}^{-1}$ 处理细胞12和24 h后, ROS水平明显升高, 而处理36 h后, ROS水平明显下降。Cr(VI) 2~32 $\mu\text{mol} \cdot \text{L}^{-1}$ 处理细胞12和24 h后, 细胞内ANT1 mRNA呈明显低表达水平, 而处理36 h后, 细胞内ANT1 mRNA表达水平明显增高, 达正常对照组的2倍左右。结论 Cr(VI)在早期(12, 24 h)可使L-02肝细胞内ROS水平升高, 发生氧化应激, 在后期(36 h)可诱导ANT1 mRNA表达水平升高, 发生能量代谢应激, 可能是Cr(VI)诱导细胞线粒体损伤的分子毒性机制之一。

关键词 [六价铬](#) [活性氧簇](#) [腺苷酸转运体1](#) [毒性](#)

分类号 [R994.6](#)

Effect of hexavalent chromium on reactive oxygen species level and adenine nucleotide translocator mRNA expression in L-02 hepatocytes

YANG Yuan^{1,2}, LIU Xin-min¹, XIAO Fang¹, LI Peng¹, ZHOU Yu¹, DAI Lu¹, ZHONG Cai-gao¹

1. Department of Health Toxicology, School of Public Health, Central South University, Changsha 410008, China;

2. Department of Clinical Laboratory, Huaihua Medical College, Huaihua 418000, China

Abstract

OBJECTIVE To explore the interference effect of hexavalent chromium(Cr(VI)) on liver cells. METHODS Cultured L-02 hepatocytes were treated with Cr(VI) 0, 2, 4, 8, 16 and 32 $\mu\text{mol} \cdot \text{L}^{-1}$ for 12, 24 and 36 h. The level of adenine nucleotide translocator 1 (ANT1) mRNA, or reactive oxygen species (ROS) was measured by reverse transcription qualitative PCR or fluorometry, respectively. RESULTS Compared with normal control group, the level of ROS increased significantly ($P<0.05$) after Cr(VI) 32 $\mu\text{mol} \cdot \text{L}^{-1}$ was treated for 12 or 24 h, while the ANT1 mRNA level decreased significantly ($P<0.05$). Compared with 24 h treatment, Cr(VI) treatment for 36 h decreased the ROS level significantly($P<0.05$) while the ANT1 mRNA level increased about two-fold compared with the normal control group. CONCLUSION At the early stage of Cr(VI) exposure (12 or 24 h), the ROS level increases significantly in cells and oxidative stress occurrence. At later stages of Cr(VI) exposure (36 h), the ANT1 mRNA level is elevated significantly, indicating an activated energy metabolism. The increased ANT1 mRNA level may be one of the molecular toxicities of mitochondrial damage induced by Cr(VI).

Key words [hexavalent chromium](#) [reactive oxygen species](#) [adenine nucleotide translocator](#) [toxicity](#)

DOI: 10.3867/j.issn.1000-3002.2012.05.012

通讯作者 钟才高 zcg54@xysm.net