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论文

三氧化二砷通过Bcl-2相关机制诱导哮喘患者T细胞凋亡

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摘要：

本研究观察了三氧化二砷对哮喘患者T细胞凋亡、白细胞介素-4分泌的影响，并探讨了Bcl-2的作用。分离哮喘患者(n＝21)和健康对照者(n＝20)的外周血T细胞，分别加入三氧化二砷和地塞米松培养24 h。用荧光显微术、流式细胞仪DNA含量分析法和细胞色素c ELISA试剂盒检测T细胞凋亡，用ELISA的方法测量血清和细胞培养上清液白细胞介素-4水平，用免疫荧光流式细胞分析法测定Bcl-2基因表达。与健康对照者比较，哮喘患者T细胞体外培养24 h后自发凋亡减慢。地塞米松使哮喘患者和健康对照者的T细胞凋亡率均增加，两试验组间增加幅度无显著差异。三氧化二砷显著增加哮喘患者T细胞凋亡率，但对健康对照者T细胞凋亡影响不明显。哮喘患者血清白细胞介素-4水平升高，T细胞Bcl-2表达上调。而且，在体外培养24 h后，哮喘患者T细胞比健康对照者T细胞自发分泌的白细胞介素-4及Bcl-2的表达水平均有所提高。三氧化二砷显著减少哮喘患者T细胞白细胞介素-4分泌，下调Bcl-2表达，但对健康对照者T细胞无明显影响。地塞米松可使两试验组T细胞释放白细胞介素-4显著减少，但对两试验组T细胞Bcl-2基因表达影响不明显。结果提示：三氧化二砷在体外可诱导哮喘患者T细胞凋亡，减少白细胞介素-4分泌，下调Bcl-2基因表达可能是其重要机制之一。

关键词： 三氧化二砷 T细胞 细胞凋亡 白细胞介素-4 哮喘 Bcl-2

In vitro arsenic trioxide induces apoptosis in T cells of asthmatic patients by a Bcl-2 related mechanism

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Abstract:

This study examined the effects of arsenic trioxide on apoptosis and interleukin-4 release in T cells of asthmatic patients *in vitro* and investigated the role of Bcl-2 in the active mechanism. T cells were isolated from asthmatic patients (n=21) and healthy controls (n=20), and then treated with arsenic trioxide and dexamethasone. Cell apoptosis was measured using fluorescence microscopy, flow cytometry and a cytochrome c ELISA kit. Interleukin-4 levels in the serum and in supernatants from T cells were quantified by ELISA. Flow cytometric analysis and immunofluorescence studies were performed to determine Bcl-2 expression. T cells of the asthmatic patients (*i.e.* without treatment) exhibited decelerated spontaneous apoptosis after 24 h incubation *in vitro* when compared to T cells of the healthy controls. With dexamethasone treatment, an increase in apoptosis of T cells was not significantly different between both groups, irrespective of the method used. Arsenic trioxide treatment, however, significantly increased the apoptosis of T cells of the asthmatic group and showed a slight effect on the control group. In asthmatic patients, elevated levels of interleukin-4 and up-regulated Bcl-2 expression were detected. Moreover, *in vitro*, T cells of asthmatic patients spontaneously released more interleukin-4 and exhibited more Bcl-2 expression than T cells from the control group. Arsenic trioxide treatment significantly decreased interleukin-4 release and down-regulated Bcl-2 expression in asthmatic patients, while it only slightly affected healthy controls. Dexamethasone treatment decreased interleukin-4 release in both groups examined. It did not significantly influence Bcl-2 expression. These results suggest that arsenic trioxide induces T cell apoptosis and decreases interleukin-4 release in T cells of asthmatic patients *in vitro* and that down-regulation of Bcl-2 expression may be an important mechanism.

Keywords: T cell apoptosis interleukin-4 asthma Bcl-2 arsenic trioxide

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参考文献：

本刊中的类似文章

1．薛英威;韩继广;李宝馨;杨宝锋.三氧化二砷对胃癌细胞SGC7901多药耐药的逆转作用及其机制[J]. 药学学报, 2007,42(9): 949-953

2．邓友平;林晨;张雪艳;陈德权;肖培根;吴旻.三氧化二砷(As2O3)诱导人胃腺癌SGC7901细胞程序化死亡并降低c-myc基因的表达[J]. 药学学报, 1999,34(5): 333-337

3．董德利;宋维华;马培林;杨宝峰.钾通道阻断剂部分抑制三氧化二砷诱导的HeLa细胞死亡[J]. 药学学报, 2005,40(7): 644-648

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