

论文

二氢青蒿素下调粒系白血病细胞转铁蛋白受体表达

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摘要:

通过建立常铁HL60和K562细胞以及富铁K562细胞体外模型, 研究二氢青蒿素对粒系白血病细胞转铁蛋白受体(transferrin receptor, TfR)的调控作用。采用流式细胞术检测二氢青蒿素对粒系白血病细胞TfR密度的调控作用, Western blotting和RT-PCR法检测二氢青蒿素对粒系白血病细胞TfR表达的调控作用, 原子吸收分光光度法检测二氢青蒿素对常铁和富铁K562细胞铁含量的影响, 以及MTT法和台盼蓝拒染法分析二氢青蒿素对粒系白血病细胞增殖的作用。结果显示, 二氢青蒿素能显著降低常铁HL60和K562细胞TfR的密度和下调TfR蛋白的表达, 且呈浓度和时间依赖性, 并能有效地抑制细胞增殖, IC₅₀值分别为1.74和11.33 μmol·L⁻¹。二氢青蒿素对富铁K562细胞的TfR蛋白和mRNA表达能进一步增强下调作用, 与常铁培养组比较, 10 μmol·L⁻¹二氢青蒿素对富铁K562细胞TfR蛋白和TfR mRNA表达量分别下调了28.1%(P<0.01)和26.2%(P<0.05), 并能显著下降富铁K562细胞铁的含量(P<0.05), 更有效地抑制富铁K562细胞增殖。由此可见, 二氢青蒿素能下调粒系白血病细胞TfR密度以及TfR蛋白和mRNA的表达, 有效抑制常铁HL60和K562细胞的增殖, 对富铁K562细胞增殖的抑制作用能进一步增强。

关键词: 二氢青蒿素 转铁蛋白受体 K562细胞 HL60细胞 铁饱和转铁蛋白

Dihydroartemisinin down-regulates the expression of transferrin receptor in myeloid leukemia cells

WANG Zeng; ZHOU Hui-jun

Abstract:

This article reports the effect of dihydroartemisinin (DHA) on transferrin receptor (TfR) in myeloid leukemia cells by establishing the model of normal iron HL60 and K562 cells and iron overload K562 cells *in vitro*. The TfR content of myeloid leukemia cells was determined by flow cytometry, and the effect of DHA on iron content in K562 cells was determined by atomic absorption spectrophotometric analysis. Furthermore, the inhibitory effect of DHA on the anti-proliferation and expression of TfR protein and mRNA in myeloid leukemia cells was studied. As a result, DHA effectively decreased the TfR content and down-regulated TfR protein expression in normal iron HL60 and K562 cells in a dose- and time-dependent manner and inhibited the cell proliferation. The IC₅₀ were 1.74 and 11.33 μmol·L⁻¹, respectively. DHA exerted more pronounced inhibitory action on expression of TfR protein and mRNA in iron overload K562 cells. Compared to normal iron K562 cells, the TfR protein and mRNA levels were lowered by 28.1% (P<0.01) and 26.2% (P<0.05), respectively, after DHA treatment for 48 h in iron overload K562 cells. Moreover, DHA decreased the iron content of iron overload K562 cells and inhibited the proliferation of iron overload K562 cells more potently. DHA effectively down-regulated the TfR content as well as expression of TfR protein and mRNA in normal iron myeloid leukemia cells. DHA also inhibited the proliferation of HL60 and K562 cells. The anti-proliferation effect of DHA on iron overload K562 cells was more striking.

Keywords: transferrin receptor K562 cell HL60 cell holotransferrin dihydroartemisinin

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