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论文

五味子酚对氧自由基引起大鼠脑突触体和线粒体损伤的保护作用

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摘要：

以Fe^{2+}-半胱氨酸(Cys)为氧自由基生成系统,在体外模仿脑出血或脑外伤引起的氧自由基损伤的模型,观察五味子酚是否对Fe^{2+}-Cys引起的大鼠脑突触体和线粒体损伤有保护作用,以探讨Sal用于延缓衰老、防治某些神经系统疾病的可能性。结果显示,与Fe^{2+}-Cys共温孵可使脑突触体和线粒体MDA生成量显著增加,线粒体ATPase活性下降。而预先加入Sal($10^{-6}mol\cdot L^{-1}$)可抑制MDA生成,防止线粒体ATPase活性降低。Sal对Fe^{2+}-Cys引起的线粒体肿胀和膜流动性降低也有明显的保护作用,并能防止Fe^{2+}-Cys所致线粒体和突触体形态的病理性损伤。结果提示,Sal对氧自由基引起的大鼠脑突触体和线粒体损伤有明显保护作用。

关键词： 五味子酚 氧自由基 突触体 线粒体

PROTECTIVE EFFECTS OF SCHISANHENOL AGAINST OXYGEN FREE RADICAL INDUCED INJURY OF RAT CEREBRAL MITOCHONDRIA AND SYNAPTOSOMES

Li Li and Liu Gengtao

Abstract:

The cerebral tissue injury induced by ischemia and reperfusion or trauma has been considered to be due to over production of oxygen free radicals (OFRs). The aim of this study was to evaluate the effects of schisanhenol (Sal) on Fe^{2+}-cysteine (Cys) induced injury of rat cerebral mitochondria and synaptosomes *in vitro*. Incubation of cerebral mitochondria or synaptosomes with Fe^{2+}-Cys at 37℃ resulted in an increase of malondiadehyde (MDA) formation and decrease of ATPase activity. Sal(10^{-4} $mol\cdot L^{-1}$) completely inhibited Fe^{2+}-Cys induced increase of MDA formation of mitochondria and synaptosomes as well as the loss of ATPase activity of mitochondria. The swelling of mitochondria and reduction of membrane fluidity of mitochondria and synaptosomes induced by Fe^{2+}-Cys were also prevented by Sal. Sal($10^{-5}mol\cdot L^{-1}$) was shown to siganificantly inhibit the decrease of synaptosomal GSH content induced by H_2O_2. The electron micrographs also showed that Sal markedly reduced the pathological damage of mitochondria and synaptosomes induced by Fe^{2+}-Cys. The results suggest that Sal has protetive action against Fe^{2+}-Cys induced injury of rat cerebral mitochondria and synaptosomes.

Keywords: Oxygen free radicals Mitochondria Synaptosomes Glutathione(GSH) Schisanhenol (Sal)

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