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论著

盐酸右美托咪定对H_2O_2诱导的肺泡巨噬细胞氧化应激的影响

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摘要:

目的: 观察α2肾上腺素受体激动剂盐酸右美托咪定是否能够对抗过氧化氢(H_2O_2)诱导的肺泡巨噬细胞氧化损伤。方法: 选择合适浓度H_2O_2和作用时间建立细胞氧化损伤模型,应用0.01,0.10,1.00 μmol/L浓度盐酸右美托咪定分别处理24 h后,再应用MTT比色法检测H_2O_2诱导的损伤细胞的存活率;用相应试剂盒测定细胞乳酸脱氢酶(lactate dehydrogenase,LDH)和肿瘤坏死因子-α(TNF-α)释放量。结果: 50~300 μmol/L H_2O_2浓度依赖性地引起肺泡巨噬细胞氧化损伤,降低细胞存活率,增加LDH和TNF-α释放。0.01~1.00 μmol/L盐酸右美托咪定可以浓度依赖性地对抗H_2O_2诱导的细胞氧化损伤,使细胞存活率明显增加,减少LDH和TNF-α释放,这种作用具有剂量依赖性。α2受体拮抗剂育亨宾能够完全拮抗盐酸右美托咪定的这种保护作用,并且育亨宾本身对细胞的氧化损伤没有影响。结论: 盐酸右美托咪定能保护肺泡巨噬细胞对抗H_2O_2诱导的氧化应激损伤,此作用可能通过α2肾上腺素受体发挥作用。

关键词: 右美托咪定 氧化应激 肺泡巨噬细胞

Effect of dexmedetomidine hydrochloride on H_2O_2-induced oxidative stress in alveolar macrophages

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Abstract:

Objective: To evaluate whether dexmedetomidine hydrochloride, an α2-adrenergic receptor agonist, can prevent oxidative damage to alveolar macrophages induced by H_2O_2.
Methods: We used methyl thiazolyl tetrazolium (MTT) colorimetry to test the effect of different concentrations and action time of H_2O_2 on the survival rate of alveolar macrophages, and then we chose the appropriate H_2O_2 concentration and action time to build NR8383 cell oxidative damage model. After pre-conditioning of 0.01, 0.10, and 1.00 μmol/L dexmedetomidine hydrochloride for 24 hours, MTT colorimetry was used to demonstrate the survival rate of NR8383 cells damaged by H_2O_2, and the release of lactate dehydrogenase (LDH) and TNF-α by H_2O_2-damaged NR8383 cells was detected by corresponding kit.
Results: At 50-300 μmol/L, H_2O_2 caused concentration-dependent oxidative damage in the alveolar macrophages, decreased the cell survival rate, and increased LDH and TNF-α release. At 0.01-1.00 μmol/L dexmedetomidine hydrochloride concentration-dependently protected NR8383 cells from oxidative damage induced by H_2O_2, significantly increased the cell survival rate, decreased LDH and TNF-α release, and this effect of dexmedetomidine hydrochloride was dose-dependent. Yohimbine, an α2 - adrenergic receptor antagonist, completely neutralized the protective effect of dexmedetomidine hydrochloride on NR8383 cells without affecting the oxidative damage of NR8383 cells.
Conclusion: Dexmedetomidine hydrochloride can prevent alveolar macrophages from oxidative damage induced by H_2O_2, which may play a protective role through α2 - adrenergic receptors.

Keywords: dexmedetomidine oxidative stress alveolar macrophages

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