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论著

ROS介导线粒体ATP敏感性钾通道开放剂对缺氧脑的保护作用

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摘要 目的：观察线粒体ATP敏感性钾通道(mitoKATP)及活性氧（ROS）在缺氧脑保护中的作用及其相互关系。 方法： 采用脑片灌流及电生理学技术，细胞外记录海马CA1区的群体锋电位(PS)和缺氧去极化电位(HD)。 结果： 用mitoKATP开放剂diazoxide (300 μmol/L) 预处理海马脑片，可延长HD的潜伏期及缺氧后PS消失的时间，提高复氧后PS的恢复率。该作用可被mitoKATP阻断剂5-hydroxydecanoic acid (200 μmol/L) 所阻断。以ROS清除剂N-2-mercaptopropionyl glycine (MPG) (500 μmol/L) 预处理海马脑片，可减弱diazoxide 的作用。单独使用MPG对PS及HD无明显影响。 结论： ROS介导了mitoKATP开放剂对缺氧脑的保护作用。

关键词 线粒体； 钾通道； 缺氧,脑； 活性氧

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Reactive oxygen species mediate neuroprotection induced by mitochondrial ATP-sensitive potassium channel opener in rat hippocampal slices during hypoxia

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Abstract

<FONT face=Verdana>AIM: To examine whether reactive oxygen species (ROS) is involved in the neuroprotection by mitochondrial ATP-sensitive potassium channel (mitoKATP) in rat hippocampal slices during hypoxia. METHODS: The technique of electrophysiology was used, and the latency to hypoxic depolarization (HD) and the amplitude of population spike (PS) in the stratum pyramidale of the CA1 region were measured. RESULTS: Pretreatment of the slices with diazoxide (DIA, a mitoKATP opener, at concentration of 300 μmol/L), prolonged the latency to HD, delayed the onset of PS disappearance and improved the recovery of PS after reoxygenation. The effects induced by DIA were attenuated by 5-hydroxydecanoic acid (a mitoKATP blocker, at concentration of 200 μmol/L). Pretreatment with N-2-mercaptopropionyl glycine (MPG, a ROS scavenger, at concentration of 500 μmol/L), also abrogated the effects induced by DIA, while treatment of MPG alone had no effect on PS and HD. CONCLUSION: ROS participates in neuroprotection offered by mitoKATP opener during hypoxia.</FONT>

Key words Mitochondria Potassium channels Hypoxia brain Reactive oxygen species

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