Neuroprotection and Inhibition of Apoptosis by Mitrochondrial Potassium Channel Openers

Published date: Oct. 7, 2014

Technology description

Technical Details:
Mitochondria can either enhance or suppress cell death. Cytochrome C release triggers apoptosis, but activation of mitochondrial ATP-sensitive potassium (mitoPotassiumATP) channels prevents lethal ischemic injury in vivo. To clarify the mechanism of the protection against ischemia, Johns Hopkins University researchers probed the relationship between mitoPotassiumATP channels and apoptosis in neonatal cardiomyocytes. Oxidative stress induced mitochondrial depolarization, cytochrome c release, caspase activation, PARP cleavage and DNA fragmentation. Pharmacological opening of mitoPotassiumATP channels by diazoxide preserved mitochondrial integrity and suppressed the markers of apoptosis; these effects were blocked by the mitoPotassiumATP channel antagonist 5-hydroxydecanoate. Diazoxide had similar cytoprotective properties in cerebellar granule neurons. The finding that mitoPotassiumATP channel activation inhibits apoptosis rationalized the protective roles of the channels in ischemia.

Institution

Johns Hopkins University

Inventors

Brian O'rourke
Professor
Medicine SOM
Eduardo Marban
Director, Heart Institute
Outside

联系我们

叶先生

电 话：021-65679356

手 机：13414935137

邮 箱：yeyingsheng@zf-ym.com