

亚硒酸钠诱导SW480细胞凋亡过程中活性氧的作用

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为探讨亚硒酸钠诱导人结肠癌SW480细胞凋亡的机理. 将荧光探针2',7'-二氯荧光黄乙二脂(2',7'-DCFH-DA)、罗丹明123(rhodamine123)负载人结肠癌细胞, 利用多光子成像系统测定胞内活性氧(ROS)、线粒体跨膜电位($\Delta\Psi_m$)的变化。结果发现(1)Na₂SeO₃作用SW480细胞, 可导致细胞凋亡和胞内的ROS增加, SOD、过氧化氢酶可降低凋亡率并抑制ROS的增加。(2)线粒体电子传递链抑制剂鱼藤酮及氰化钠可抑制ROS增加。(3) Na₂SeO₃可导致线粒体的跨膜电位的下降。表明Na₂SeO₃作用细胞可导致来源于线粒体的ROS增加, ROS介导亚硒酸钠诱导细胞凋亡。

EFFECT OF REACTIVE OXYGEN SPECIES ON APOPTOSIS INDUCED BY Na₂SeO₃ IN SW480 CELLS

To investigate the mechanism of apoptosis induced by sodium selenite in a human colonic cancer cell line SW480. SW480 cells were loaded with fluorescent probes 2',7'-DCFH-DA, and rhodamine123, respectively. The changes of intracellular ROS, and mitochondrial transmembrane potential ($\Delta\Psi_m$) was detected by using multiphoton imaging system. The results showed that (1) Na₂SeO₃ elevated SW480 cells apoptotic rate and intracellular ROS; SOD, catalase evidently suppressed ROS increase and reduced apoptotic rate, (2) Inhibition of mitochondrial electron transport with NaCN or rotenone almost blocked selenite-induced ROS production, (3) Na₂SeO₃ induced disruption of the mitochondrial transmembrane potential ($\Delta\Psi_m$). The data suggest that Na₂SeO₃ results in the increase intracellular ROS which originates from mitochondria and mediates Na₂SeO₃-induced apoptosis.

关键词

亚硒酸钠(Sodium selenite); 活性氧(ROS); 细胞凋亡(Apoptosis); 信号转导(Signal transduction)