SODIUM FLUORIDE INDUCES APOPTOSIS IN H9C2 CARDIOMYOCYTES THROUGH CHANGING MITOCHONDRIAL MEMBRANE POTENTIAL AND INTRACELLULAR ROS LEVEL

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SUMMARY: Chronic excessive fluoride intake is known to be toxic and effects of long-term fluorosis on different organ systems have been examined. However, there are really a few studies about the effects of fluorosis on cardiovascular systems. Here, we studied the fluoride induced apoptosis in H9c2 cells and determined the underlying molecular mechanisms including the cell viability, intracellular ROS level, the changes of mitochondrial membrane potential (MMP) and the cell apoptosis. Sodium fluoride (NaF) at concentrations of 0, 2, 4, 8 and 16 mg/L was administered to cultured H9c2 cells for up to 48h. After the treatment period, H9c2 cells were collected and the associated parameters were measured by flow cytometry. Our study found that fluoride not only inhibited H9c2 cell proliferation but also induced cell apoptosis. With the increase of NaF concentration, the apoptotic rates and ROS generation were up-regulated, while the MMP was decreased. In summary, this data suggested that NaF-induced H9c2 cell apoptosis is mediated by direct increased intracellular ROS and down-regulated MMP.

Key words*:* H9c2 cells；Sodium fluoride；ROS；Apoptosis

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