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The relationship between the MAPK signaling pathway with Kashin-Beck Disease, an endemic osteochondropathy and the protective effect of Selenium

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Extracellular signal regulated kinases (ERKs) and C-Jun N-terminal kinase (JNK) are members of the mitogen-activated protein kinase (MAPK) family and are activated by environmental stress. Selenium plays an important role in the biological pathways by forming selenoprotein. Selenoproteins have been shown to exhibit a variety of biological functions, including antioxidant functions, maintaining cellular redox balance and compromise of such important proteins would lead to oxidative stress and apoptosis. We examined the expression levels of ERKs and JNK in KBD patients, tested the potential protective effects of sodium selenite on tert-butyl hydroperoxide (tBHP) induced oxidative injury and apoptosis in human chondrocytes as well as its underlying mechanism in this study. We produced an oxidative damage model induced by tBHP in C28/I2 human chondrocytes to test the essential anti-apoptosis effects of selenium in vitro. The results indicated that the protein levels of pRaf-1, pMek1/2 and pErk1/2 decreased significantly in KBD patients, the expression level of phosphorylated JNK was significantly increased in KBD patients. Cell apoptosis was increased and molecule expressions of JNK signaling pathway were activated in the tert-butyl hydroperoxide (tBHP) injured chondrocytes. Na2SeO3 treatment improved the reduction of proteins in ERK signal pathway, Na2SeO3 protects against tBHP induced oxidative stress and apoptosis in cells by increasing cell viability, reducing ROS generation, increasing GPX activity and down-regulating JNK pathway. These results demonstrated that apoptosis induced by tBHP in chondrocyte might be mediated via up regulation of JNK pathway, Na2SeO3 has an effect of anti-apoptosis by down regulating of JNK signaling pathway, Selenium stimulates the phosphorylation of the ERK signaling pathway.

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