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KBD pathogenesis from Kashin-Beck disease and a rat model of Kashin–Beck disease using T-2 toxin and selenium deficiency conditions

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Kable and MMPs/TIMPs ratio between KBD children and a novel model, in which rats were administered a seleniumdeficient diet for 4 weeks prior to their exposure to T-2 toxin for 4 weeks. Chondronecrosis in deep zone of articular cartilage of knee joints was seen in T-2 toxin plus selenium-deficient diet groups, which were very similar to chondronecrosis observed in human KBD and decreased anabolic enzymes of GAG metabolism, PAPSS2, PAPST1 and CHST15, and increased catabolic enzymes ARSB and GALNS in cartilage were seen in KBD and in the novel rat model. Further, the levels of MDA, and IL-6, IL-1 β , TNF- α in serum and cartilage were increased in KBD and in the novel rat model. And MMP-1 and -13 levels increased, while TIMP-1 levels decreased in KBD and in the novel rat model. And MMP-1 and -12 toxin treatment under selenium deficient conditions can be used as a suitable animal model for studying etiological factors contributing to the chondro-necrosis observed in human KBD. This pathological change which results from oxidative stress/ cytokines/ MMPs pathways by T-2 toxin and selenium deficiency may result in progressive chondrocyte cell death, and ECM degradation and destruction in the deep zone of cartilage in KBD. Alterations of enzymes involved in cartilage CS GAG metabolism on PGs play an important role in the onset and pathogenesis of KBD.

Biography

Jing-hong Chen completed her PhD from Xi'an Jiaotong University in China and Post-doctoral studies from Hospital for Special Surgery, Cornell University School of Medicine. She is the Vice-Director of Institute of Endemic Diseases, Xi'an Jiaotong University, a premier Kashin-Beck disease research organization in China. She has published more than 50 papers in international journals and Chinese journals.

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