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A Study on The Mechanism of Oxidative Stress, Screening of Protective Agents and Signal Transduction of Cell Differentiation in Cultured Osteoblast and Osteoclast Damaged by Reactive Oxygen Species

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It is well known that osteoblasts and osteoclasts play a key role in bone metabolism. They involve in osteoformation or bone destruction which are ragulated by various factors such as thyroid hormone, parathyroid hormone, estrogen, growth factor and cytokine. Recently, it is demonstrated that oxidative stress is one of pathological factors in bone metabolism, but it is left unknown about mechanism between oxidative stress and bone metabolism. In this study, we examined oxidative stress on osteoblasts and osteoclasts, and also the protective effects of NMDA and AMPA/kainate receptor antagonists against xanthine oxidase(XO)/hypoxanthine(HX)-induced oxidative stress, were evaluated in these cultures. XO/HX decreased the cell viability by MTS assay or INT assay and glutathione peroxidase(GPx) activity in these cultures, but, it increased lipid peroxidase(LPO) activity by TBARS assay in these cultures. In the protective effect, NMDA receptor antagonist, APV or CKA increased protein synthesis by SRB assay, cell viability or ALP activity, but decreased LDH activity in cells injured by XO/HX-induced oxidative stress. While, AMPA/kainate receptor antagonist such as CNQX or DNQX did not show any protective effect in these cultures. NMDA receptor antagonist also regarded to have a diminutive tendancy of

c-fos expression or TNF-α activity. From these results, it is suggested that oxidative stress is toxic to osteoblasts or osteoclasts, and also, NMDA receptor antagonist is effective in the prevention of oxidative stress.