PB-5

Effect of High glucose on JNK/ERK signaling pathway in UMR106 cells

In Ok Jung, Mei Hua Jin and Sung Jin Kim*
Dept of Pharmacology, School of Dentistry, Kyung Hee University Seoul Korea 130-701

Recently diabetes has been found to be associated with metabolic bone diseases such as osteoporosis. In the present study, attempts have been made to explore the effect of high glucose in bone formation. Osteoblast-like UMR 106 cells were treated with high glucose (22mM, 33mM, 44mM) for 1 or 2 days. High glucose significantly inhibited proliferation of UMR106 cells in a time- and dose- dependent manner as evidenced by MTT assay. For the evaluation of collagen synthesis, UMR 106 cells were cultured in high glucose media (44mM) for 24 h and the ratio of collagen content to total protein was measured. In addition, gene expression pattern of type I collagen was assessed by RT-PCR. The high concentration of glucose inhibited a collagen synthesis, a marker of bone formation activity. JNK, c-Jun N-terminal Kinase, is known to play an important role in stress-associated cell death. In this regard, we tested to determine whether high glucose has any effect on JNK activity. It has been found that treatment of high glucose induced phosphorylation of JNK. On the other hand, ERK phosphorylation was inhibited by high glucose in a dose-dependent manner. Taken together, Therefore these results indicate that inhibition of proliferation in UMR 106 cells following high glucose is related to JNK/ERK containing signal pathways. This study showed high glucose concentration could alter the bone metabolism leading to defective bone formation, suggesting that high glucose due to diabetes may play a significant role in the development of metabolic bone disease.