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Treatment with hydrogen molecule alleviates TNFα-induced cell injury in osteoblast

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Abstract

Tumor necrosis factor-alpha (TNFα) plays a crucial role in inflammatory diseases such as rheumatoid arthritis and postmenopausal osteoporosis. Recently, it has been demonstrated that hydrogen gas, known as a novel antioxidant, can exert therapeutic anti-inflammatory effect in many diseases. In this study, we investigated the effect of treatment with hydrogen molecule (H(2)) on TNFα-induced cell injury in osteoblast. The osteoblasts isolated from neonatal rat calvariae were cultured. It was found that TNFα suppressed cell viability, induced cell apoptosis, suppressed Runx2 mRNA expression, and inhibited alkaline phosphatase activity, which was reversed by co-incubation with H(2). Incubation with TNFα-enhanced intracellular reactive oxygen species (ROS) formation and malondialdehyde production increased NADPH oxidase activity, impaired mitochondrial function marked by increased mitochondrial ROS formation and decreased mitochondrial membrane potential and ATP synthesis, and suppressed activities of antioxidant enzymes including SOD and catalase, which were restored by co-incubation with H(2). Treatment with H(2) inhibited TNFα-induced activation of NFκB pathway. In addition, treatment with H(2) inhibited TNFα-induced nitric oxide (NO) formation through inhibiting iNOS activity. Treatment with H(2) inhibited TNFα-induced IL-6 and ICAM-1 mRNA expression. In conclusion, treatment with H(2) alleviates TNFα-induced cell injury in osteoblast through abating oxidative stress, preserving mitochondrial function, suppressing inflammation, and enhancing NO bioavailability.

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