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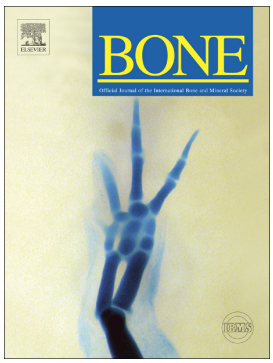

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ACCEPTED MANUSCRIPT

Interleukin-6 Deletion Stimulates Revascularization and New Bone Formation Following Ischemic Osteonecrosis in a Murine Model

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ABSTRACT

Legg-Calvé-Perthes disease (LCPD) is a childhood form of ischemic osteonecrosis of the femoral head which can produce a permanent femoral head deformity and early osteoarthritis. The femoral head deformity results from increased bone resorption and decreased bone formation during repair and remodeling of the necrotic femoral head. A recent study showed that a pro-inflammatory cytokine, interleukin-6 (IL-6), is significantly elevated in the synovial fluid of patients with LCPD. We hypothesized that IL-6 elevation decreases bone formation during the repair process following ischemic osteonecrosis and that IL-6 depletion will increase new bone formation. To test this hypothesis, we surgically induced ischemic osteonecrosis in the wild-type (n=29) and IL-6 knockout (KO) mice (n=25). The animals were assessed at 48 hours, 2 weeks and 4 weeks following the induction of ischemic osteonecrosis using histologic, histomorphometric and micro-CT methods. IL-6 immunohistochemistry showed high expression of IL-6 in the osteonecrotic side of the wild-type mice at 48 hours and 4 weeks following ischemic osteonecrosis, but not in the IL-6 KO mice. We also confirmed an undetectable level of IL-6 expression in the primary osteoblasts of the IL-6 KO mice compared to the readily detectable level in the wild-type mice. Furthermore, we confirmed that IL-6 deletion did not affect the extent of bone necrosis in the IL-6 KO mice compared to the wild-type mice by performing histologic and terminal deoxynucleotidyl transferase mediated dUTP nick-end labeling (TUNEL) assessments

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