

The 49th Biomechanics Seminar

2025年11月12日(水)11:00~12:00

京都大学 医生物学研究所

南部総合研究 1 号館・医生研 1 号館 1 階セミナー室 3

http://www.kyoto-u.ac.jp/ja/access/campus/yoshida/map6r_b.html

演 題: STAT3 in Mechanotransduction and Bone Healing

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冒: Signal Transducer and Activator of Transcription 3 (STAT3) is a key transcription factor that integrates biochemical and biomechanical cues to regulate bone remodeling and repair. Emerging evidence indicates that STAT3 serves as a crucial mediator of mechanotransduction, linking external mechanical stimuli to intracellular gene expression that governs osteogenesis, angiogenesis, and tissue regeneration. Using conditional STAT3 knockout mouse models, studies have demonstrated that loss of STAT3 in osteoblasts or osteocytes leads to impaired bone formation, reduced responsiveness to mechanical loading. Conversely, mice with STAT3 overexpression display enhanced bone formation and accelerated bone healing. Elevated STAT3 signaling promotes osteoblast differentiation, increases bone formation rate, and improves callus maturation following injury. Together, these genetic models highlight STAT3 as a central regulator of bone mechanotransduction and bone repair, acting as a molecular hub that translates mechanical and cytokine signals into coordinated transcriptional programs. Modulating STAT3 activity - either through genetic or pharmacologic means - offers a promising strategy to enhance skeletal adaptation and promote fracture healing in pathological conditions where mechanosensitivity is compromised.

講演言語:英語

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