



GUEST SEMINAR SERIES

Department of Musculoskeletal Tissue Regeneration

Date Wednesday 18th November 2026 at 16:30-17:30 o'clock

Location Hörsaal der Orthopädischen Klinik im Zentrum für Seelische Gesundheit am König-Ludwig-Haus • Brettreichstraße 11 • 97074 Würzburg

Guest-Speaker **Prof. Makarand Risbud,, Ph.D.**
Department of Orthopaedic Surgery,
Thomas Jefferson University, Philadelphia, USA

Lecture “Epigenetics, senescence and spine aging – what we know so far ?”



**For seminar abstract and presenter's biosketch,
please refer to next page**



Presentation abstract

Cellular senescence is increasingly recognized as a major contributor to skeletal aging and degeneration. The accumulation of senescent cells and their inflammatory SASP disrupts tissue homeostasis and promotes progressive degeneration of the intervertebral disc. Experimental studies show that eliminating or modifying senescent cells can mitigate pathological changes. The lecture will focus on emerging evidence on the mechanisms driving senescence and the utility of senotherapeutics as a new treatment approach

Biosketch

Prof. Risbud's research focuses on understanding the molecular, cellular, and microenvironmental mechanisms that regulate intervertebral disc homeostasis, degeneration, and associated pain. His laboratory has developed an integrative research program investigating how the unique hypoxic, osmotic, mechanical, and ageing-associated environments of the disc regulate cell function and tissue integrity. The laboratory has four interconnected research themes: (1) hypoxia-inducible factor (HIF) signaling and its roles in nucleus pulposus cell survival, metabolism, autophagy, extracellular matrix regulation, and adaptation to the hypoxic disc niche; (2) osmotic and mechanical regulation of disc cell function, with a particular focus on tonicity-regulated enhancer binding protein (TonEBP) and its role in disc development and homeostasis; (3) mechanisms of age-associated disc degeneration, including cellular senescence, progenitor cell biology, inflammation, and neuroinflammatory pathways contributing to disc pathology and pain; and (4) the development and characterization of genetically defined animal models of spontaneous disc degeneration, herniation, and calcification to identify disease mechanisms and therapeutic targets. Collectively, this work seeks to establish how niche-specific environmental factors govern disc biology and to translate these insights into mechanism-based therapeutic strategies for disc degeneration.