

PhD Viva Presentation

Title: Study of immune signatures in the pathogenesis of osteoporosis

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Department: Molecular Immunodiagnostics

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Abstract

Osteoporosis is a systemic skeletal disorder characterized by reduced bone mass and increased fracture risk, particularly in postmenopausal women. While aging and estrogen deficiency are established risk factors, growing evidence recognizes osteoporosis as an immune-mediated inflammatory condition. Activated T cells, especially T helper (Th) subsets, influence bone remodeling by regulating osteoclastogenesis through cytokine secretion. The present work aimed to delineate the role of T-cell subsets in bone loss by comparing premenopausal and postmenopausal women with varying bone mineral density. Our findings demonstrate an increased Th17 frequency, elevated IL-17 levels, and an altered Th17/Treg balance in postmenopausal women, particularly those with low bone mass. Th17 cells predominantly exhibited an activated effector memory phenotype, accompanied by reduced Th1 cells. Correlation and partial correlation analyses revealed that immune–bone associations are age-dependent but strongly modulated by estrogen deficiency. Overall, these findings highlight the interplay between immune dysregulation and bone health during the menopausal transition.