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Title:	Exploring the effect of post-menopausal inflammatory conditions on osteoclast differentiation
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Abstract:	<p>Osteoporosis, a systemic skeletal disorder characterized by decreased bone density and increased susceptibility to fractures, poses a significant public health concern globally, particularly among aging populations. Among the various forms of osteoporosis, post- menopausal osteoporosis stands out as one of the most prevalent types, affecting a large number of post-menopausal women. Following menopause, hormonal changes, particularly the decline in estrogen levels, contribute to accelerated bone loss, leading to an imbalance in bone remodeling processes. In post-menopausal osteoporosis, the delicate balance between bone formation by osteoblasts and bone resorption by osteoclasts is disrupted, tipping the scales towards increased bone resorption. Osteoclasts, multinucleated cells derived from the monocyte/macrophage lineage, play a pivotal role in bone resorption by secreting enzymes such as tartrate-resistant acid phosphatase (Trap) and cathepsin K (Ctsk), which degrade the bone matrix. Understanding the underlying mechanisms driving osteoclast differentiation and activity in the context of post-menopausal osteoporosis is crucial for developing targeted therapeutic interventions to mitigate bone loss and reduce fracture risk in affected individuals. In this study, we aimed to elucidate the intricate interplay between inflammatory signals and osteoclast differentiation in the context of post-menopausal osteoporosis. Through a series of in vitro and in vivo experiments, we investigated the regulatory roles of key molecules such as Nfatc1, Toll-like receptor 2 (TLR2), and CD36 in modulating osteoclastogenesis under inflammatory conditions. Moreover, in an ovariectomy-induced osteoporosis model, we observed a priming effect of pre-existing RANKL exposure on osteoclast differentiation, emphasizing the complex interplay between inflammatory signals and bone remodeling. Overall, these findings offer valuable insights into the pathophysiology of post-menopausal osteoporosis.</p>
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