發炎與細胞素對於蝕骨細胞之調控

Inflammation and cytokines in regulation of osteoclast activation

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Human osteoclast formation from mononuclear phagocyte precursors involves interactions between tumor necrosis factor (TNF) ligand superfamily members and their receptors. Bone and immune systems are tightly linked. In the past years, many molecules originally believed to belong to the immune system were found to function in bone cells. It is now evident that the two systems are coregulated by many shared cytokines and signaling molecules. We exemplify the complex interaction between bone metabolism and immune response focusing on the multifaceted role of receptor activator of NF-κB ligand (RANKL). RANKL is expressed by cells of both systems, is an essential regulator of bone degradation and exerts either pro or anti-inflammatory effects on the immune response. Our recent studies demonstrated that TRAIL, a new TNF superfamily member induces osteoclast differentiation via a TRAF-6-dependent signaling pathway. This study suggests TRAF6-dependent signaling may be a central pathway in osteoclast differentiation, and that TNF superfamily molecules other than RANKL may modify RANK signaling by interaction with TRAF6-associated signaling. In this review, we summarize the multiple functions of RANKL in bone and in the immune systems, aiming to provide an overview of the field of osteoimmunology and the targets for development of new therapy in inflammation associated bone destruction.