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SIRT3 regulates bone remodeling in mice by regulating AMPK activity

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The mitochondrial sirtuin 3 (SIRT3) is involved in suppressing the onset of multiple pathologies, including cardiovascular disease, fatty liver, breast cancer and age-related disorders. However, its role in bone metabolism is not known. Here we show the involvement of SIRT3 in osteoclast differentiation. Receptor activator of nuclear factor-κB ligand (RANKL), an essential cytokine for osteoclastogenesis, induces the expression of both the transcription co-activator peroxisome proliferator-activated receptor-γ co-activator-1β (PGC1β) and the nuclear receptor estrogen receptor-related receptor α (ERRα), which coordinately up-regulated SIRT3 during osteoclast differentiation from bone marrow-derived monocytes/macrophages (BMMs). SIRT3-deficient mice exhibit decreased bone mass compared with wild-type mice due to increased numbers of osteoclasts. Consistently, *Sirt3*$^{-/-}$ osteoclast precursor cells underwent increased osteoclastogenesis in response to RANKL, whereas SIRT3 overexpression in osteoclast precursor cells exhibited reduced the formation of osteoclasts. Strikingly, *Sirt3*$^{-/-}$ osteoclast precursor cells reduced AMP-activated protein kinase (AMPK) phosphorylation through down-regulating the expression of AMPK that plays a key role in regulating cellular energy metabolism during RANKL-induced osteoclast differentiation. These data demonstrate that a mitochondrial SIRT3 is an intrinsic inhibitor for RANKL-mediated osteoclastogenesis.

Biography

Jeong-Eun Huh working in the Research Center for Cellular Homeostasis in Ewha Womans University. A major in molecular and cellular bone biology, she has published 3 peer-reviewed paper. Her primary research interests are in the field of osteoimmunology, especially the mechanisms of osteoclastogenesis. She received a research fellowship grant from the National Research Foundation of Korea (NRF). Her long term goal is to target signaling pathways as a novel approach to therapy

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