A new role for HERPUD1 and ERAD activation in osteoblast differentiation and mineralization

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 Bone integrity depends on a finely tuned balance between bone synthesis by osteoblasts and resorption by osteoclasts. The secretion capacity of mature osteoblasts requires strict control of proteostasis. Endoplasmic reticulum?associated degradation (ERAD) prevents the accumulation of unfolded ER proteins via dislocation to the cytosol and degradation by the proteasome. The ER membrane protein, homocysteine-inducible endoplasmic reticulum protein with ubiquitin-like domain 1 (HERPUD1), is a key component of the ERAD multiprotein complex which helps to stabilize the complex and facilitate the efficient degradation of unfolded proteins. HERPUD1 expression is strongly up-regulated by the unfolded protein response and cellular stress. The aim of the current study was to establish whether HERPUD1 and ERAD play roles in osteoblast differentiation and maturation. We evaluated preosteoblastic MC3T3-E1 cell and primary rat osteoblast differentiation by measuring calcium deposit levels,