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Bone Metabolism and Atherosclerosis: Interaction between Bone and Vessel in Aging Process

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Bone loss is a representative change with aging and atherosclerosis is one type of vascular aging. Several epidemiological studies, including cross-sectional and longitudinal studies, have reported that decreased bone mass and atherosclerosis are significantly associated. Although there are still controversies about the biological basis of these associations, severe potential mechanisms have been proposed: common mediators or specific signaling pathways between these two conditions have been suggested as one mechanism that could underlie these associations. Accumulation of oxidative stress and increased levels of pro-inflammatory cytokines are common factors affecting both the bones and vasculature. Therefore, higher levels of shared factors that accelerate both bone loss and atherosclerosis have been proposed as one potential mechanism. Meanwhile, several osteogenic factors released from bone, such as osteocalcin and sclerostin, are known to be related to vascular calcification. Osteocalcin is a protein synthesized by osteoblasts. Recent human studies have demonstrated negative associations between osteocalcin levels and atherosclerosis. Sclerostin, a endogenous negative regulator of Wnt signaling, is also known to be positively associated with vascular calcification. Moreover, disturbed calcium metabolism, including elevated parathyroid hormone levels, is also expected to partially contribute to the relationships between low bone mass and atherosclerosis. Further studies are warranted to evaluate further the plausible association between low bone mass and atherosclerosis, and the clear biological mechanisms that could explain the association as well.