Mitochondrial regulator DRP1 promotes vascular calcification, report Rogers et al.

Blood vessel calcification is associated with adverse hemodynamics and cardiovascular conditions. Once believed to be a passive process of aging, it is now known that calcification involves the active differentiation of smooth muscle cells (SMCs) and valve interstitial cells (VICs) into bone-like cells (osteoblasts). Many of the biological processes related to vessel calcification—such as cell differentiation, apoptosis, and calcium homeostasis—involve mitochondrial dynamics, and it has been shown that mutations to the mitochondrial regulator dynamin-related protein 1 (DRP1) are associated with calcification of heart tissue in mice. Rogers and colleagues have examined the potential link between DRP1 and calcification in humans. They found that in human carotid artery plaques and in calcified valve tissue—from patients undergoing aortic valve replacements—DRP1 staining was elevated in regions of calcium deposition. Furthermore, DRP1 levels were also high in cultures of human SMCs and VICs undergoing osteoblast differentiation, and inhibition of DRP1 attenuated both SMC and VIC calcification. The findings suggest that human DRP1 promotes vascular calcification and that this condition may be prevented or attenuated by therapeutic inhibition of DRP1.