The Role of Peroxiredoxins in Autophagic Flux in the Pathogenesis of Atherosclerosis

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Reactive oxygen species (ROS), which include superoxide anions and peroxides, induce oxidative stress, contributing to the initiation and progression of cardiovascular diseases involving atherosclerosis. The endogenous and exogenous factors hypercholesterolemia, hyperglycemia, hypertension, and shear stress induce various enzyme systems such as nicotinamide adenine dinucleotide (phosphate) oxidase, xanthine oxidase, and lipoxygenase in vascular and immune cells, which generate ROS. Besides inducing oxidative stress, ROS mediate signaling pathways involved in monocyte adhesion and infiltration, platelet activation, and smooth muscle cell migration. A number of antioxidant enzymes (e.g., superoxide dismutases, catalase, glutathione peroxidases, and peroxiredoxins) regulate ROS in vascular and immune cells. Atherosclerosis results from a local imbalance between ROS production and these antioxidant enzymes. In this lecture, I would like to discuss 1) ROS-dependent atherogenic signaling in endothelial cells, macrophages, and vascular smooth muscle cells, and 2) roles of peroxidases in autophagic flux.