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The Functional Roles of APE1/Ref-1 on Vascular Inflammation

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Oxidative stress plays an important role in the pathogenesis of cardiovascular disease. Oxidative stress is a state of imbalance between oxidants and antioxidants, leading to damaging effects. Monocyte adhesion to endothelial cells is the initial event of vascular inflammation, and the cellular adhesion molecules plays some important mediators in monocyte adhesion. Physiologically, antioxidant defense systems are capable of regulating to levels of oxidants in order to maintain the oxidant-antioxidant balance. An apurinic apyrimidinic endonuclease 1/redox factor-1 (APE1/Ref-1) is involved both in the base excision repair of abasic DNA lesions and in eukaryotic transcriptional regulation including AP-1, NF-kB etc. Recently, the presence of extracellular plasma APE1/Ref-1 was reported in endotoxemic rats. The biological significance and the extracellular function of APE1/Ref-1 is uncovering. Recently, it confirmed that APE1/Ref-1 protein with reducing activity induced a conformational change in TNF-α receptor by thiol-disulfide exchange as well as inhibition of Toll-like receptor signaling. Therefore, APE1/Ref-1 plays as thiol-dependent antioxidant system in defense against oxidative stress through it disulfide activity regulating protein dithiol/disulfide balance. These results strongly indicate that anti-inflammatory effects of APE1/Ref-1 and its usefulness as therapeutic biomolecules against vascular inflammation.