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A NOVEL ENDOTHELIAL PROTEIN TYROSINE PHOSPHATASE IN VASCULAR WALL

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Understanding the molecular networks that regulate vascular inflammation is crucial for gaining insight into atherosclerosis and identifying medicinal targets thereof is necessary for pharmacological interventions. However, molecular actions of regulators that control the early development of vascular inflammation are still largely unknown. Herein, we demonstrate that a novel endothelial protein tyrosine phosphatase (ePTP) serves as a potent regulator of inflammatory signaling in the vascular wall. Endothelial PTP expression was significantly down-regulated in aortic endothelium of apoE-deficient mice fed an atherogenic diet. Loss of ePTP in artery endothelial cells (ECs) markedly activated inflammatory cytokines-activated NF-κB signaling via down-regulation of A20 expression at the transcriptional level. In addition, depletion of ePTP in artery ECs prominently potentiated inflammatory cytokines-induced cell adhesion molecules (CAMs) expression and subsequently resulted in a remarkable enhance of leukocyte adhesion. In contrast, transduction of ePTP prevented inflammatory cytokines-induced NF-κB signaling, CAMs expression, and leukocyte adhesion. Consistently, EC-specific ePTP transgenic/apoE-deficient mice displayed decreased atherosclerotic plaque formation compared to wild-type littermates fed an atherogenic diet for 12 weeks. Collectively, these findings demonstrate that ePTP controls NF-κB-mediated EC activation in response to proinflammatory stimuli and that ePTP may be a potential therapeutic target for treatment of atherosclerosis and vascular inflammation-related diseases.

Biography

Min Ji Cho has undergraduate in B.S in Biology (2009-2014) from Dong-A University, Busan, Korea in February, 2014.

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