
Abstract:
Vascular smooth muscle cells (VSMC) play a key role in the pathogenesis of atherosclerosis, the globally leading cause of death. The transmembrane orphan receptor endosialin (CD248) has been characterized as an activation marker of cells of the mesenchymal lineage including tumor-associated pericytes, stromal myofibroblasts, and activated VSMC. We, therefore, hypothesized that VSMC-expressed endosialin may display functional involvement in the pathogenesis of atherosclerosis. Expression of endosialin was upregulated during atherosclerosis in apolipoprotein E (ApoE)-null mice and human atherosclerotic samples analyzed by quantitative real-time polymerase chain reaction and immunohistochemistry. Atherosclerosis, assessed by Oil Red O staining of the descending aorta, was significantly reduced in ApoE/endosialin-deficient mice on Western-type diet. Marker analysis of VSMC in lesions induced by shear stress-modifying cast implantation around the right carotid artery identified a more pronounced contractile VSMC phenotype in the absence of endosialin. Moreover, in addition to contributing to neointima formation, endosialin also potentially regulated the proinflammatory phenotype of VSMC as evidenced in
surrogate cornea pocket assay experiments in vivo and corresponding flow cytometry and ELISA analyses in vitro. The experiments identify endosialin as a potential regulator of phenotypic remodeling of VSMC contributing to atherosclerosis. The association of endosialin with atherosclerosis and its absent expression in nonatherosclerotic samples warrant further consideration of endosialin as a therapeutic target and biomarker.

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