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Polymeric immunoglobulin receptor (PIGR) is a transmembrane protein involved in the transcytosis of polymeric IgA and IgM immunoglobulins. Recent data from our group described that systemic deficiency of Pigr reduces atherosclerosis development, which is associated with increased plasma IgA and IgM levels. To analyse the role of Pigr deficiency in hematopoietic cells in an experimental model of atherosclerosis in mice. We analyzed PIGR distribution in early atherosclerotic plaques compared to healthy aortic samples, as well as PIGR expression in macrophages derived from human peripheral blood mononuclear cells (PBMCs). The contribution of hematopoietic Pigr to experimental atherosclerosis was assessed by performing bone marrow transplantation in Ldlr-/- mice fed an atherogenic diet for 10weeks. PIGR expression was observed in the intima of early human atherosclerotic lesions, showing colocalization with macrophages. In addition, PIGR levels increased during the in vitro differentiation of human PBMCs into macrophages. Analysis of the hematopoietic lineage-specific Pigr deficiency in atherosclerosis revealed that Pigr-/- mice displayed significantly reduced plaque size in the aortic sinuses as well as a strong decrease in macrophage infiltration, while no changes in contractile vascular smooth muscle cells and collagen content were observed. These effects occurred independently of systemic changes in lipids, IgA and IgM levels. Hematopoietic Pigr deficiency attenuates experimental atherosclerosis, although the molecular mechanisms underlying this protective effect remain to be fully elucidated.