Protective effect of the polarity of macrophages regulated by IL-37 on atherosclerosis

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ABSTRACT. As an anti-inflammatory cytokine, interleukin-37 (IL-37) provides certain protective effects against inflammatory and autoimmune diseases. Recent reports indicate that IL-37 is expressed in foam cells of atherosclerotic plaques in both the coronary and carotid arteries of humans, suggesting the possible involvement of IL-37 in the pathogenesis and progression of atherosclerosis. Current evidence supports the protective role that IL-37 plays against atherosclerosis via the regulation of different subtypes of macrophage. Atherosclerosis was induced in apolipoprotein E -/- mice through diet, and the mice were then given IL-37 to observe patterns in the aorta plaque. Furthermore, human peripheral blood-derived monocytes were cultured for seven days to induce the differentiation of macrophages. Specifically, we observed the effect of IL-37 on oxygenated low density lipoprotein (ox-LDL)-induced macrophage polarity, in addition to conducting an expression assay of the M1 cell markers tumor necrosis factor (TNF)-α and CD86 and the M2 marker CD206. IL-37 effectively decreased the area ratio between the aorta plaque and vascular cavity. We also observed that M1 macrophages can be induced from peripheral monocytes by ox-LDL, with significant elevation of marker molecules.
TNF-α and CD86. The co-stimulation of IL-37 and ox-LDL, however, inhibited the induction of M1 cells and facilitated the transformation of macrophages into M2 cells, as supported by the elevation of cell-specific marker CD206. These results indicate that IL-37 can prevent atherosclerosis by modulating macrophage polarity.

Key words: IL-37; Macrophage polarity; Atherosclerosis; Artery plaque