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## The innate chemokine MIF goes adaptive in atherosclerosis

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Macrophage migration inhibitory factor (MIF) proteins (MIF and MIF-2) are chemokine-like inflammatory mediatorswith unique structural properties distinct from classical chemokines. MIF proteins play arole in the control of both physiological and pathophysiological immune responses. With MIF-2 only very recently identified, most evidence currently is available for MIF. In fact, MIF is a pivotal upstream mediator of innate immunity, while some contribution to the adaptive response has been reported. When dysregulated, MIF is an exacerbating promoter of several inflammatory diseases including atherosclerosis, a chronic inflammatory condition of large and medium-sized arteries and the major underlying cause of cardiovascular morbidity and mortality worldwide. MIF orchestrates the atherogenic recruitment of monocytes/macrophages and T lymphocytes through non-cognate interaction with the CXC chemokine receptors CXCR2 and CXCR4, respectively, and contributes to the inflammation and destabilization in atherosclerotic lesions. These processes have been considered as the effects of an innate chemokine on innate inflammatory cells in the atherosclerotic lesion area.Here we show that MIF also controls adaptive immune cells in atherosclerotic pathogenesis. We present data that MIF is a novel B cell chemokine that promotes B cell migration and proliferationvia the chemokine receptors CXCR4 and CXCR7 as well as CD74, the surface form of MHC class II invariant chain. MIF-driven B cell responses are mediated through the ZAP-70 and ERK1/2 signaling pathways and encompass the activation of calcium transients. We also studied the impact of Mif gene deletion in the pro-atherogenicApoE-/-genetic background in mice and have unraveled a surprising atherogenic phenotype with a previously unrecognized link between MIF and B cell pathobiology. This suggeststhat MIF could be a potential therapeutic target to induce protective B cell responses in such diseases.

**Notes:**