Stimulatory interactions between human coronary smooth muscle cells and dendritic cells

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Despite inflammatory and immune mechanisms participating to atherogenesis and dendritic cells (DCs) driving immune and non-immune tissue injury response (1, 2), the interactions between DCs and vascular smooth muscle cells (VSMCs) possibly relevant to vascular pathology including atherogenesis are still unclear. To address this issue, DCs were generated from peripheral blood CD14 $^+$ cells isolated from healthy donors and co-cultured (ccDCs) with human coronary artery VSMCs (CASMCs). Co-culture induced DC functional maturation, as demonstrated by mixed lymphocyte reaction. In turn, factors from mature DCs and ccDCs induced CASMC migration. Among secreted factors there were IL-6 and MCP-1 from CASMCs, and MCP-1 and TNF α from DCs. DCs adhesion to CASMCs ?was enhanced by CASMC pre-treatment with IFN γ and TNF α ? and was inhibited by CASMC pre-treatment with atorvastatin and rosiglitazone. The findings indicate that DCs and VSMCs can interact with reciprocal stimulation, possibly leading to perpetuate inflammation and vascular wall remodeling, and that the interaction is enhanced by a cytokine-rich inflammatory environment and down-regulated by HMGCoA-reductase inhibitors and PPAR γ agonists.

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