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CXCR7: A potential target for anti-angiogenesis treatment

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Angiogenesis is an important pathogenic process implicated in rheumatoid arthritis (RA), and anti-angiogenesis treatment is potential strategy for RA therapy. Recent studies suggest that endothelial progenitor cells (EPC) are involved in synovial vascularization, and may contribute to the increased cardiovascular morbidity and mortality in RA. EPC is trigger of angiogenesis. During angiogenesis, EPC are mobilized from bone marrow, recruited to angiogenic site, incorporate and finally form neo-vessel. In those processes, stromal cell-derived factor-1 (SDF-1) is a main regulator. For a long time, SDF-1 was considered to act via its unique receptor CXCR4. CXCR7 is a recently-identified alternative receptor of SDF-1. Western blotting and flow cytometry assay results showed that considerable intracellular CXCR7 was expressed in EPC derived from human cord blood. We investigated the role of CXCR7 in EPC by blocking CXCR7 with antagonist CCX733. Multiple functional assays revealed that CXCR7 mediated human EPC survival exclusively, mediated tube formation and matrix metalloproteinase-2 (MMP-2) production along with CXCR4. Blocking CXCR7 with CCX733 impaired EPC adhesion to active HUVEC and trans-endothelial migration induced by SDF-1, but had no effect on EPC migration, proliferation or nitric oxide (NO) production. These results indicated that CXCR7 plays a critical role in EPC homing and participating in angiogenesis, and may be a potential target molecule in anti-angiogenesis therapy for RA.

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