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Regulation of T cell chemotaxis by CXCL4

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Directed migration of cells along a chemotactic gradient is a fundamental cellular process involved e.g. in host defense, tissue development, and wound repair. Surprisingly, although a broad spectrum of different mediators are able to induce a chemotactic response, only very few regulators or inhibitors of this function are known. CXCL4 (platelet factor 4; PF4), a platelet-derived CXC-chemokine, modulates long-term immunregulatory functions in T cells but lacks the capacity to induce chemotaxis in these cells. However, in the current study we are able to show for the first time, that CXCL4 acts as a potent inhibitor of T cell chemotaxis induced by CXCR3 ligands CXCL11 and CXCL9, but not CXCL10. CXCL4 did neither interfere with ligand binding to CXCR3 nor with ligand-induced internalization or calcium signaling of CXCR3. By several lines of evidence we could rule out the participation of known CXCL4-receptors, like proteoglycans or CXCR3-B, in CXCL4-mediated inhibition of chemotaxis. We, thus, claim the presence of a further, so far unidentified receptor for this chemokine, which is present on T cells. Intriguingly, CXCL4 also reduces the chemotaxis of T cells and neutrophils induced by the CXCR3-independent ligand CXCL12 (SDF-1alpha). Taken together, our results identify CXCL4 as the first chemokine, which acts as an inhibitor rather than an inducer of chemotaxis on T cells and neutrophils in vitro.