

Modulation of macrophage differentiation and activation by decoy receptor 3

邱文祥

Chang;Yung-Chi;Hsu;Tsui-Ling;Lin;His-Hsien;Chio;Chung-Chung;Chiu;Allen W

摘要

Abstract

Decoy receptor 3 (DcR3) is a soluble receptor of the tumor necrosis factor receptor superfamily and is readily detected in certain cancer patients. Recently, we demonstrated that DcR3.Fc-treated dendritic cells skew T cell responses to a T helper cell type 2 phenotype. In this study, we further asked its ability to modulate CD14⁺ monocyte differentiation into macrophages induced by macrophage-colony stimulating factor in vitro. We found that DcR3.Fc was able to modulate the expression of several macrophage markers, including CD14, CD16, CD64, and human leukocyte antigen-DR. In contrast, the expression of CD11c, CD36, CD68, and CD206 (mannose receptor) was not affected in the in vitro culture system. Moreover, phagocytic activity toward immune complexes and apoptotic bodies as well as the production of free radicals and proinflammatory cytokines in response to lipopolysaccharide were impaired in DcR3.Fc-treated monocyte-derived macrophages. This suggests that DcR3.Fc might have potent, suppressive effects to down-regulate the host-immune system..