Enhanced Adhesion of Monocyte via Reverse Signaling Triggered by Decoy Receptor 3 (DcR3)

許銘仁

Hsu MJ;Lin WW;Tsao WC;Hsu TL;Chang YC;Hsiao HW;Chiu AW;Chio CC;Hsieh SL

摘要

Abstract

Decoy receptor 3 (DcR3), a newly identified soluble protein belonging to the tumor necrosis factor receptor (TNFR) superfamily, is a receptor for Fas ligand (FasL), LIGHT and TL1A. It has been demonstrated that DcR3 is frequently overexpressed by malignant tumors arising from lung, gastrointestinal tract, neuronal glia and virus-associated leukemia. Recently, we demonstrated that DcR3 is able to modulate the differentiation and activation of dendritic cells (DCs), and that DcR3-treated DCs skew naive T cell differentiation towards a Th2 phenotype. In this study, we further demonstrate that DcR3 is able to induce actin reorganization and enhance the adhesion of monocytes and THP-1 cells by activating multiple signaling molecules, such as protein kinase C (PKC), phosphatidylinositol 3-kinase (PI3K), focal adhesion kinase (FAK) and Src kinases. This provides the first evidence that the soluble DcR3, like other immobilized members of TNFR superfamily, is able to trigger 'reverse signaling' to modulate cell function.