Modulation of dendritic cell differentiation and maturation by decoy receptor 3

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摘要

Abstract

Decoy receptor 3 (DcR3), a soluble receptor belonging to the TNFR superfamily, is a receptor for both Fas ligand (FasL) and LIGHT. It has been demonstrated that DcR3 is up-regulated in lung and colon cancers, thus promoting tumor growth by neutralizing the cytotoxic effects of FasL and LIGHT. In this study, we found that DcR3.Fc profoundly modulated dendritic cell differentiation and maturation from CD14(+) monocytes, including the up-regulation of CD86/B7.2, and the down-regulation of CD40, CD54/ICAM-1, CD80/B7.1, CD1a, and HLA-DR. Moreover, DcR3-treated dendritic cells suppressed CD4(+) T cell proliferation in an allogeneic MLR and up-regulated IL-4 secretion of CD4(+)CD45RA(+) T cells. This suggests that DcR3.Fc may act not only as a decoy receptor to FasL and LIGHT, but also as an effector molecule to skew T cell response to the Th2 phenotype.