DcR3/TNFRSF6B Protein, Human (sf9, hFc)

Cat. No.:	HY-P75306
Synonyms:	Tumor necrosis factor receptor superfamily member 6B; Decoy receptor 3; TNFRSF6B; DCR3; TR6
Species:	Human
Source:	Sf9 insect cells
Accession:	O95407 (V30-H300)
Gene ID:	8771
Molecular Weight:	Approximately 65 kDa

PROPERTIES

Biological Activity	Measured by its ability to inhibit Fas Ligand induced apoptosis of Jurkat human acute T cell leukemia cells and the ED_{50} for this effect is typically ≤25 μg/mL in the presence of 200 ng/mL human Fas ligand.
Appearance	Solution
Formulation	Supplied as a 0.2 μm filtered solution of 100 mM Glycine, 10 mM NaCl, pH 7.0.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	N/A.
Storage & Stability	Stored at -80°C for 1 year. It is stable at -20°C for 3 months after opening. It is recommended to freeze aliquots at -80°C for extended storage. Avoid repeated freeze-thaw cycles.
Shipping	Shipping with dry ice

DESCRIPTION

Background	Decoy Receptor 3 (DcR3), a secreted member of the Tumor Necrosis Factor (TNF) receptor superfamily, neutralizes three different TNF ligands/pro-apoptotic molecules: FasL, LIGHT, and TL1A. Each of these ligands engages unique signaling receptors which direct distinct and critical immune responses. Dcr3 has competitive binding with the functional receptors to the respective cytokines (Fas for FasL, DR3 for TL1A, and LTbR and herpesvirus entry mediator/HVEM for LIGHT), thus preventing downstream pro-apoptotic signaling. DcR3 is encoded by the Tnfrsf6b gene, which does not encode a cytoplasmic or transmembrane segment, resulting in an obligate secreted protein of 300 amino acids including the signal peptide. However, mouse and rat genomes lack a Tnfrsf6b gene[1][2]. DcR3 is overexpressed in a wide variety of malignancies and is correlated with tumorigenesis and progression. Therefore, it has been considered a potential biomarker to predict cancer invasion and progression of inflammation. Specifically, DcR3 induces epithelial-mesenchymal transition through activation of the TGF-β3/SMAD signaling pathway in human colorectal cancer (CRC)[3][4]. In addition, it increases the expression levels of several components of the PI3K/AKT/GSK-3β/β-catenin signaling pathway, such as p-AKT, GSK-3β, p-GSK-3β and β-catenin. Additionally, DcR3 also enhances the expression of N-cadherin and Vimentin and decreases the expression of E-cadherin[3]. DcR3 also promotes proliferation and invasion of pancreatic cancer via a DcR3/STAT1/IRF1

feedback loop. It promotes the phosphorylation of signal transducers and activators of transcription 1 (STAT1), leading to a dramatic increase in interferon regulatory factor 1 (IRF1). IRF1 then increased the transcriptional activity of DcR3, forming a positive feedback loop to reinforce DcR3 expression[5]. The homology of human DcR3 protein is low with that of other animals, and the sequence similarity with rats, mice and pigs is 20.49%, 20.82% and 20.00%, respectively.

REFERENCES

[1]. Zhan C, et al. Decoy strategies: the structure of TL1A:DcR3 complex. Structure. 2011 Feb 9;19(2):162-71.

[2]. Lagou S, et al. The Role of Decoy Receptor DcR3 in Gastrointestinal Malignancy. Cancer Diagn Progn. 2022 Jul 3;2(4):411-421.

[3]. Ge H, et al. DcR3 induces proliferation, migration, invasion, and EMT in gastric cancer cells via the PI3K/AKT/GSK-3β/β-catenin signaling pathway. Onco Targets Ther. 2018 Jul 19;11:4177-4187.

[4]. Liu YP, et al. DcR3 induces epithelial-mesenchymal transition through activation of the TGF-β3/SMAD signaling pathway in CRC. Oncotarget. 2016 Nov 22;7(47):77306-77318.

[5]. Gao L, et al. DcR3, a new biomarker for sepsis, correlates with infection severity and procalcitonin. Oncotarget. 2017 Dec 28;9(13):10934-10944.

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