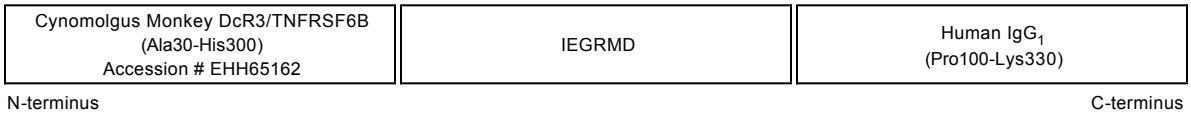
Recombinant Cynomolgus Monkey DcR3/TNFRSF6B Fc Chimera

Catalog Number: 9297-DC

DESCRIPTION

Source

Chinese Hamster Ovary cell line, CHO-derived



N-terminal Sequence Analysis	Ala30
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	56 kDa

SPECIFICATIONS

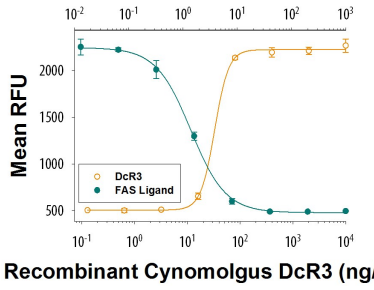
SDS-PAGE	58-67 kDa, reducing conditions
Activity	Measured by its ability to inhibit Fas Ligand-induced apoptosis of Jurkat human acute T cell leukemia cells. Cheng, J. *et al.* (1994) Science **263**:1759. The ED_{50} for this effect is typically 15-75 ng/mL in the presence of 20 ng/mL Recombinant Human Fas Ligand/TNFSF6 (Catalog # 126-FL).
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in MES and NaCl. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 500 µg/mL in water.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

DATA

Bioactivity



Recombinant Cynomolgus Monkey DcR3/TNFRSF6B Fc Chimera (Catalog # 9297-DC) inhibits Recombinant Human Fas Ligand/TNFSF6 (Catalog # 126-FL) induced apoptosis of Jurkat human acute T cell leukemia cells. The ED_{50} for this effect is 15-75 ng/mL.

bio-techne
bio-techne.com
Global info@bio-techne.com techsupport@bio-techne.com
North America TEL 800 343 7475 Europe | Middle East | Africa TEL +44 (0)1235 529449
China info.cn@bio-techne.com TEL +86 (21) 52380373
Rest of World bio-techne.com/find-us/distributors TEL +1 612 379 2956

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BACKGROUND

DcR3 (Decoy Receptor 3), also known as TR6 and TNFRSF6B is a 35-40 kDa secreted member of the TNF Receptor superfamily (1). Mature cynomolgus DcR3 contains four tandem TNFR cysteine-rich domains and shares 92% amino acid sequence identity with human DcR3. It binds to the TNF superfamily ligands Fas Ligand, TL1A, and LIGHT (2-5) and interferes with their respective interactions with Fas, DR3, or HVEM and Lymphotoxin βR (2-4). It blocks apoptosis triggered through either Fas Ligand, TL1A, or LIGHT (2, 3, 5, 6). DcR3 is up-regulated in a variety of cancers and enhances tumor cell immune evasion (2, 3, 7). It also promotes immune suppression by inducing dendritic cell apoptosis (8), inhibiting NK cell and $CD8^+$ T cell activity (2, 4), and inhibiting the production of inflammatory cytokines during viral infection or autoimmunity (9, 10). In humans, proteolytic removal of the C-terminal 53 amino acids generates a shortened DcR3 that retains the ability to block LIGHT but not Fas Ligand induced apoptosis (11). DcR3 can also induce osteoclast formation from monocytes (12).

References:

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bio-techne.com
Global info@bio-techne.com techsupport@bio-techne.com
North America TEL 800 343 7475 Europe | Middle East | Africa TEL +44 (0)1235 529449
China info.cn@bio-techne.com TEL +86 (21) 52380373
Rest of World bio-techne.com/find-us/distributors TEL +1 612 379 2956