



Product Data Sheet

TNFRSF3/LTBR Protein, Cynomolgus (HEK293, Fc)

Cat. No.: HY-P77445

Synonyms: Tumor Necrosis Factor Receptor Superfamily Member 3; TNF-RIII; TNFCR; TNFR3

Species: Cynomolgus HEK293 Source:

F6V995 (S28-M225) Accession:

Gene ID: 712550

Molecular Weight: Approximately 68 kDa

PROPERTIES

AA Sequence

SQPQVVPPYG	SENQTCRDQE	KEYYEPRHRI	CCSRCPPGTY
VSAKCSRSRD	TVCATCAENS	YNEHWNYLTI	$C\ Q\ L\ C\ R\ P\ C\ D\ P\ V$
MGLEEIAPCT	SKRKTQCRCQ	PGMFCAAWAL	ECTHCELLSD
CDDCTEAELK	DEVCKCNNHC	V D C K A C H E O N	TSSDSADCOD

HTRCEDQGLV EAAPGTAQSD TTCRNPSESL PPEMSGTM

Biological Activity Measured by its binding ability in a functional ELISA. Immobilized Cynomolgus TNFRSF3 at 5 μg/mL (100 μL/well) can bind Human LIGHT. The ED_{50} for this effect is 13.86 ng/mL.

Lyophilized powder **Appearance**

Lyophilized from a 0.2 µm filtered solution of PBS, pH 7.4. Formulation

Endotoxin Level <1 EU/µg, determined by LAL method.

Reconsititution It is not recommended to reconstitute to a concentration less than $100 \, \mu g/mL$ in ddH_2O . For long term storage it is recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose).

Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is Storage & Stability recommended to freeze aliquots at -20°C or -80°C for extended storage.

Shipping Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background

Lymphotoxin beta receptor (LTBR), also known as tumor necrosis factor receptor superfamily member 3 (TNFRSF3), is a member of the tumor necrosis factor receptor superfamily and a cell surface receptor for lymphotoxins involved in apoptosis and cytokine release. LTBR is expressed on the surface of most cell types, including breast, colorectal, lung, gastric, melanoma, and bladder cancers, while its ligands lymphotoxin (LT) a1b2 and TNF superfamily member 14

(TNFSF14; also known as LIGHT), are mainly expressed on the surface of immune cells. The LTBR signaling pathway may be involved in the activation of responses that control cell differentiation, growth and death, as manifested by the formation of peripheral lymphoid-like organs, especially secondary and tertiary lymphoid structures critical for tissue, dendritic cell homeostasis, liver regeneration, interferon response to pathogens and death in mucosa-derived carcinomas. LT β R signaling may facilitate communication between infiltrating immune cells and tumor cells. Triggering LT β R induces typical and atypical nuclear factor (NF)- κ B signaling pathways that are associated with inflammation-induced oncogenic effects. Sustained LT β R signaling also leads to NF- κ B-mediated chronic inflammation and the development of hepatocellular carcinoma (HCC)^{[1][2]}.

REFERENCES

[1]. Norris PS, et al. The LT beta R signaling pathway. Adv Exp Med Biol. 2007;597:160-72.

[2]. Mo Shen, et al. Lymphotoxin β receptor activation promotes mRNA expression of RelA and pro-inflammatory cytokines TNF α and IL-1 β in bladder cancer cells. Mol Med Rep. 2017 Jul;16(1):937-942.

Caution: Product has not been fully validated for medical applications. For research use only.

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