TNFRSF3/LTBR Protein, Rat (HEK293, Fc)

Cat. No.:	HY-P75917
Synonyms:	Tumor Necrosis Factor Receptor Superfamily Member 3; TNF-RIII; TNFCR; TNFR3
Species:	Rat
Source:	HEK293
Accession:	Q5U2S8 (M1-A218)
Gene ID:	297604
Molecular Weight:	Approximately 66 kDa

PROPERTIES

Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	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 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. LTβR signaling may facilitate communication between infiltrating immune cells and tumor cells. Triggering LTβR induces typical and atypical nuclear factor (NF)-κB signaling pathways that are associated with inflammation-induced oncogenic effects. Sustained LTβR signaling also leads to NF-κB-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 TNFα and IL-1β in bladder cancer cells. Mol Med Rep. 2017 Jul;16(1):937-942.

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