BioLegend
The path to legendary discovery™

Product Data Sheet

Recombinant Human CD255 (TWEAK) (carrier-free)

Catalog # / Size: 566402 / 5 µg

Source: Human TWEAK, amino acids Lys97-His249 (Accession# NM_003809.2), was expressed in *E. coli*.

Molecular Mass: The 154 amino acid N-terminal methionylated recombinant protein has a predicted molecular mass of 17 kD.

Purity: >98%, as determined by Coomassie stained SDS-PAGE.

Endotoxin Level: Less than 0.01 ng per µg cytokine as determined by the LAL method.

Activity: ED_{50} < 10 ng/ml, corresponding to a specific activity of > 1 x 10^7 units/mg, as determined by the dose dependent stimulation of production of IL-8 by human PBMC.

Formulation: Lyophilized

Storage: Unopened vial can be stored at -20°C or -70°C for 12 months. For maximum results, quick spin vial prior to opening. Stock solutions should be prepared at no less than 10 µg/mL in buffer containing carrier protein such as 1% BSA or HSA or 10% FBS, and storage at -20°C or -70°C. **Avoid repeated freeze/thaw cycles.**

Applications:

Applications: Bioassay

Application Notes: BioLegend's Recombinant Human CD255 (TWEAK) has been reported to induce cardiomyoctye proliferation *in vivo*.[1]

Application References:
1. Novoyatleva T, *et al.* 2010. *Cardiovasc. Res.* 85:681. PubMed

Description: TWEAK (TNFSF12) is a "TNF-likeweak inducer" of apoptosis through a non-deathdomain-dependent mechanism. TWEAK is a type II membrane protein which exhibits a single internal hydrophobic domain of 27 amino acids in the N-terminal region. TWEAK is proteolytically cleaved to produce a soluble cytokine that signals as a trimerized molecule. Fibroblast growth factor-inducible 14 (Fn14)/TWEAKR has been described as a receptor for TWEAK, and it is associated with proliferation of endothelial cells and angiogenesis. However, TWEAK mediates signal transduction and linear differentiation of monocyte/macrophage cells lacking Fn14/TWEAKR, suggesting that such cells contain an alternative TWEAK receptor. Elevated levels of TWEAK and/or Fn14 have been found to be associated with the pathogenesis of rheumatoid arthritis, skeletal muscle wasting, systemic lupus erythematosus, multiple sclerosis, stroke, neuroinflammation and neurodegeneration, and several types of cancer. The pathological functions of TWEAK are primarily attributed to its ability to induce the expression of several proinflammatory cytokines, chemokines, cell adhesion molecules, and matrix-degradingenzymes mainly through the activation of NF-B, a major proinflammatorytranscription factor. More recently, it has been described that CD163 (a scavenger receptor) might be acting as a receptor "decoy" for the ligand TWEAK.

Antigen References:
1. Chicheportiche Y, *et al.* 1997. *J. Biol. Chem.* 272:32401.
2. Wiley SR, *et al.* 2001. *Immunity* 15:837.
3. Jakubowski A, *et al.* 2002. *J. Cell. Sci.* 115:267.
4. Bover LC, *et al.* 2007. *J. Immunol.* 178:8183.
5. Kumar M, *et al.* 2009. *J. Immunol.* 182:2439.
6. Van Gorp H, *et al.* 2010. *Mol. Immunol.* 47:1650.

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