

DESCRIPTION

Source *E. coli*-derived
Arg93-His249, with an N-terminal Met and 6-His tag
Accession # Q4ACW9

N-terminal Sequence Analysis Met

Predicted Molecular Mass 18.3 kDa

SPECIFICATIONS

Activity Measured in a cell proliferation assay using HUVEC human umbilical vein endothelial cells.
The ED₅₀ for this effect is typically 2-8 ng/mL.

Endotoxin Level <0.10 EU per 1 µg of the protein by the LAL method.

Purity >95%, by SDS-PAGE under reducing conditions and visualized by silver stain.

Formulation Lyophilized from a 0.2 µm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

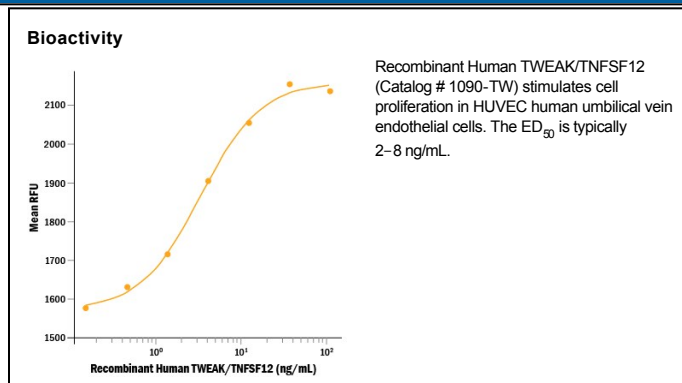
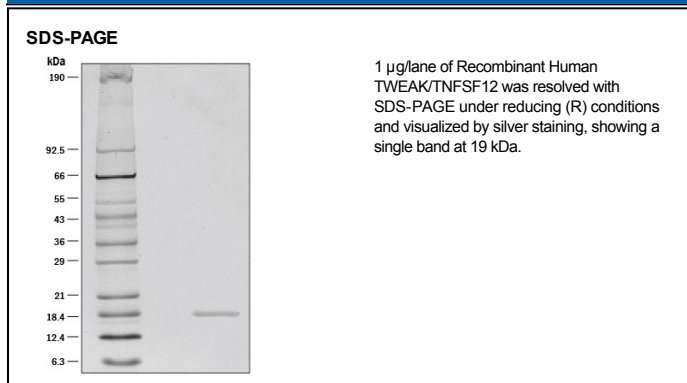
Reconstitution Reconstitute at 100 µg/mL in sterile PBS containing at least 0.1% human or bovine serum albumin.

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



BACKGROUND

TWEAK is a type II transmembrane protein belonging to the TNF superfamily (1). It contains a short cytoplasmic domain (aa 1-18), the transmembrane domain (aa 19-42) and an extracellular domain (aa 43-249). The extracellular domains of human and murine TWEAK share 89% aa sequence identity. A soluble form of TWEAK is generated from the membrane-associated molecules by proteolytic cleavage after Arg 93 suggesting that TWEAK may have long-range effects. TWEAK is expressed widely in many tissues and cells. At least two receptors that bind TWEAK have been identified (2-4). Death Receptor 3 (DR3), also known as TNFRSF12, Apo-3, LARD, WSL-1 or TRAMP, is a TNF receptor superfamily member that is expressed predominantly in tissues with high lymphocyte content (2). It has been suggested that induction of cell death by TWEAK-DR3 interaction involves the activation of NF-κB. In cells that lack DR3, alternate pathways of TWEAK-induced cell death mediated by receptors distinct from DR3 have been suggested (5, 6). TWEAK receptor (TWEAKR, alternatively known as FN14), is a novel TNF receptor superfamily member that also binds TWEAK (3, 4). It is a mitogen-inducible gene that is expressed in fibroblasts, hepatic carcinomas and endothelial cells. TWEAK-TWEAKR interaction has been shown to play a role in endothelial cell growth and migration. This effect of TWEAK is not mediated by an up-regulation of VEGF (7).

References:

1. Chicheportiche, Y. *et al.* (1997) *J. Biol. Chem.* **272**:32401.
2. Marsters, S. *et al.* (1998) *Current Biol.* **8**:525.
3. Wiley, S. R. *et al.* (2001) *Immunity*, **15**:837.
4. Feng, S.-L.Y. *et al.* (2000) *Am J. Path.* **156**:1253.
5. Nakayama, M. *et al.* (2002) *J. Immunol.* **168**:734.
6. Schneider, P. *et al.* (1999) *Eur. J. Immunol.*, **29**:1785.
7. Lynch, C. *et al.* (1998) *J. Biol. Chem.* **274**:8455.