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RDSYSTEMS

Recombinant Human TWEAK/TNFSF12

Catalog Number: 1090-TW

DESCRIPTION		
Source	<i>E. coli</i> -derived human TWEAK/TNFSF12 protein 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 ED ₅₀ for this effect is 0.200-4.00 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.



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 is expressed in fibroblasts, hepetocellular 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:

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- 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.
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Global bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL +1 612 379 2956 USA TEL 800 343 7475 **Canada** TEL 855 668 8722 **China** TEL +86 (21) 52380373 **Europe | Middle East | Africa** TEL +44 (0)1235 529449