

## DESCRIPTION

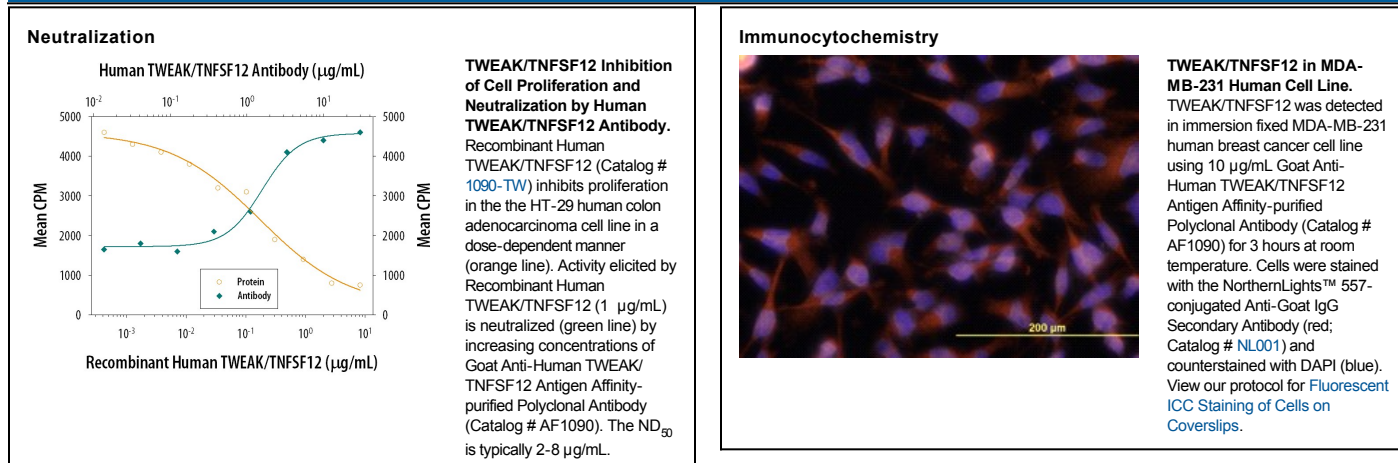
|                           |  |
|---------------------------|--|
| <b>Species Reactivity</b> | Human  |
| <b>Specificity</b>        | Detects human TWEAK in direct ELISAs and Western blots. In direct ELISAs, less than 5% cross-reactivity with recombinant human (rh) BAFF, rhFas Ligand, rhGITR Ligand, rhOX40 Ligand, rhTRAIL and less than 1% cross-reactivity with rhAPRIL, rhLIGHT, rhTNF- $\alpha$ , rhVEGI, and rhTRANSE is observed. |
| <b>Source</b>             | Polyclonal Goat IgG  |
| <b>Purification</b>       | Antigen Affinity-purified  |
| <b>Immunogen</b>          | <i>E. coli</i> -derived recombinant human TWEAK<br>Arg93-His249<br>Accession # Q4ACW9  |
| <b>Endotoxin Level</b>    | <0.10 EU per 1 $\mu$ g of the antibody by the LAL method.  |
| <b>Formulation</b>        | Lyophilized from a 0.2 $\mu$ m filtered solution in PBS with Trehalose. See Certificate of Analysis for details.<br>*Small pack size (-SP) is supplied as a 0.2 $\mu$ m filtered solution in PBS.  |

## APPLICATIONS

**Please Note:** Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

|                            | Recommended Concentration  | Sample  |
|----------------------------|--|---|
| <b>Western Blot</b>        | 0.1 $\mu$ g/mL   | Recombinant Human TWEAK/TNFSF12 (Catalog # 1090-TW) |
| <b>Immunocytochemistry</b> | 5-15 $\mu$ g/mL  | See Below   |
| <b>Neutralization</b>      | Measured by its ability to neutralize TWEAK/TNFSF12-induced inhibition of proliferation in the HT-29 human colon adenocarcinoma cell line. Yu, K. Y. <i>et al.</i> (1999) <i>J. Biol. Chem.</i> <b>274</b> :13733; Harrop, J. A. <i>et al.</i> (1998) <i>J. Biol. Chem.</i> <b>273</b> :27548 The Neutralization Dose (ND <sub>50</sub> ) is typically 2-8 $\mu$ g/mL in the presence of 1 $\mu$ g/mL Recombinant Human TWEAK/TNFSF12. |   |

## DATA



## PREPARATION AND STORAGE

|                                |   |
|--------------------------------|---|
| <b>Reconstitution</b>          | Reconstitute at 0.2 mg/mL in sterile PBS.   |
| <b>Shipping</b>                | The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.<br>*Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C  |
| <b>Stability &amp; Storage</b> | Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> <li>• 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>• 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>• 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul> |

**BACKGROUND**

TWEAK is a type II transmembrane protein belonging to the TNF superfamily (1). It contains a short cytoplasmic domain (amino acids (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- $\kappa$ B. 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, hepatocellular 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.