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Product datasheet for TA320503

TWEAK (TNFSF12) Mouse Monoclonal Antibody [Clone ID: CARL-2]

Product data:

Product Type:	Primary Antibodies
Clone Name:	CARL-2
Recommended Dilution:	ELISA, Functional Assay
Reactivity:	Human
Host:	Mouse
Clonality:	Monoclonal
Formulation:	Aqueous buffer, 0.09% sodium azide, may contain carrier protein/stabilizer
Concentration:	lot specific
Purification:	Affinity purified
Conjugation:	Unconjugated
Storage:	Store at -20°C as received.
Stability:	Stable for 12 months from date of receipt.
Gene Name:	tumor necrosis factor superfamily member 12
Database Link:	NP_003800 Entrez Gene 8742 Human O43508

View online »

Background: The CARL-2 monoclonal antibody reacts with human TWEAK, a type II transmembrane TNF superfamily member with high identity to TNF in its extracellular portion. TWEAK transcript is expressed broadly in many adult and fetal tissues, however, the staining of human peripheral blood mononuclear cells with monoclonal antibodies shows a more restricted pattern. While freshly isolated PBMCs do not express detectable levels of TWEAK on their surface, IFN gamma-stimulated blood monocytes rapidly upregulate TWEAK surface expression. TWEAK is expressed as membrane bound and secreted forms. Interaction of TWEAK with its counter-receptor promotes secretion of IL-8, activation of NF-kappaB, proliferation of endothelial cells, and apoptosis in a number of human cell lines. Initially, DR3 was thought to be a receptor for TWEAK, but further studies have shown that TWEAK could induce apoptosis via receptors distinct from DR3. While TWEAK exhibits overlapping signaling functions to TNF, it is generally less effective in inducing apoptosis, giving rise to its name, TNF-like weak inducer of apoptosis. For detection of human TWEAK by sandwich ELISA, a combination of purified CARL-2 for capture and biotinylated CARL-1 for detection is recommended.

Synonyms: APO3L; DR3LG; TWEAK

Protein Families: Druggable Genome, Secreted Protein, Transmembrane

Protein Pathways: Cytokine-cytokine receptor interaction