

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse DR3 in direct ELISAs and Western blots. In direct ELISAs, approximately 30% cross-reactivity with recombinant mouse (rm) DR6, rmTrail R2, and recombinant human (rh) Mer is observed, and approximately 20% cross-reactivity with rhDR3, rhTrail R4, rmCD30, and rmHVEM is observed, and less than 5% cross-reactivity with rhTrail R3, rm4-1BB, rhTrail R1, rmGITR, and rmTNF R1 is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse DR3/TNFRSF25 Gln31-Phe199 Accession # AAK11256
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

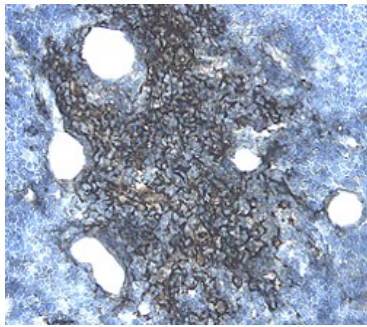
APPLICATIONS

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
Western Blot	0.1 µg/mL	Recombinant Mouse DR3/TNFRSF25 Fc Chimera (Catalog # 2437-D3)
Immunohistochemistry	5-15 µg/mL	See Below

DATA

Immunohistochemistry



DR3/TNFRSF25 in Mouse Thymus.

DR3/TNFRSF25 was detected in perfusion fixed frozen sections of mouse thymus using 5 µg/mL Goat Anti-Mouse DR3/TNFRSF25 Antigen Affinity-purified Polyclonal Antibody (Catalog # AF2437) overnight at 4 °C. Tissue was stained with the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS008) and counterstained with hematoxylin (blue). View our protocol for [Chromogenic IHC Staining of Frozen Tissue Sections](#).

PREPARATION AND STORAGE

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

BACKGROUND

Death receptor 3 (DR3), also known as TNFRSF25, LARD, WSL-1, APO3, TRAMP, and TR3, is a 55 kDa TNF receptor superfamily protein that is predominantly expressed by lymphocytes. TNF receptor superfamily members have varying numbers of extracellular cysteine-rich domains (CRDs) with conserved cysteine spacing (1, 2). DR3 contains four CRDs and one cytoplasmic death domain (3, 4). Alternative splicing of mouse DR3 generates an isoform that lacks the fourth CRD and a secreted isoform that consists of only the extracellular domain (ECD) (3). Human DR3 exists in at least eleven alternate splice forms (5). Within the ECD, mouse and human DR3 share 59% amino acid (aa) sequence identity. DR3 shares 20-28% aa sequence identity with the ECD of death domain receptors DR5, DR6, EDAR, Fas, NGF R, and TNF RI. Naïve B and T cells preferentially express truncated soluble isoforms of DR3, whereas stimulated lymphocytes preferentially express transmembrane DR3 (5). TL1A/TNFSF15, a high affinity DR3 ligand which also exists in membrane bound and soluble forms, is expressed by activated endothelial cells and T cells (6, 7). TL1A additionally binds to DcR3/TNFRSF6B, a soluble decoy receptor that interferes with DR3 activation (8). DR3 signaling triggers either apoptosis or NFκB-induced anti-apoptotic effects depending on the cellular setting (9). Apoptosis is partially impaired during negative selection of thymocytes in DR3-null mice (10). TL1A interactions with DR3 augment T cell proliferation and pro-inflammatory cytokine secretion (6, 7, 11, 12). DR3 is upregulated by inflammatory stimulation of CCR9⁺ T cells, a T cell subset important in mucosal immunity (11). T cell and macrophage DR3 expression is prominent in several inflammatory disorders such as Crohn's disease, inflammatory bowel disease, and atherosclerosis (7, 11-15). DR3 activation on IFN-γ treated THP-1 cells induces the production of TNF-α, CXCL8, CCL2, MMP-1, -9, and -13 (14, 15).

References:

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