

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human LAP in direct ELISAs and Western blots. In direct ELISAs and Western blots, this antibody is specific for the LAP derived from the TGF-β1 precursor but does not react with LAP from the TGF-β2 precursor.
Source	Polyclonal Goat IgG
Purification	Protein A or G purified
Immunogen	<i>S. frugiperda</i> insect ovarian cell line Sf 21-derived recombinant human LAP (TGF-β1) and Chinese hamster ovary cell line CHO-derived recombinant human LAP (TGF-β1) Leu30-Ser390 Accession # P01137
Endotoxin Level	<0.10 EU per 1 µg of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS and NaCl with Trehalose. See Certificate of Analysis for details.

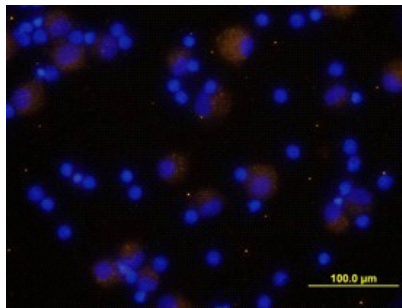
APPLICATIONS

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	1 µg/mL	Recombinant Human LAP TGF-β1 (Catalog # 246-LP)
Immunocytochemistry	5-15 µg/mL	See Below
Immunohistochemistry	5-15 µg/mL	Immersion fixed paraffin-embedded sections of human prostate cancer tissue
Neutralization	Measured by its ability to neutralize LAP TGF-β1 inhibition of TGF-β1 growth inhibition in the HT-2 mouse T cell line. Tsang, M. <i>et al.</i> (1995) <i>Cytokine</i> 7:389. The Neutralization Dose (ND ₅₀) is typically 15-30 µg/mL in the presence of 125 ng/mL Recombinant Human LAP TGF-β1 and 0.25 ng/mL TGF-β1.	

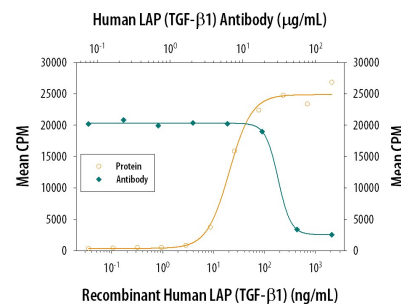
DATA

Immunocytochemistry



TGF-β1 in Human PBMCs. TGF-β1 was detected in immersion fixed human peripheral blood mononuclear cells (PBMCs) using Goat Anti-Human LAP TGF-β1 Polyclonal Antibody (Catalog # AB-246-NA) at 10 µg/mL for 3 hours at room temperature. Cells were stained using the NorthernLights™ 557-conjugated Anti-Goat IgG Secondary Antibody (yellow; Catalog # NL001) and counterstained with DAPI (blue). View our protocol for [Fluorescent ICC Staining of Non-adherent Cells](#).

Neutralization



LAP TGF-β1 Inhibition of TGF-β1 Activity and Neutralization by Human LAP TGF-β1 Antibody. Recombinant Human LAP TGF-β1 (Catalog # 246-LP) inhibits Recombinant Human TGF-β1 (Catalog # 240-B) growth inhibition activity in the HT-2 mouse T cell line in a dose-dependent manner (orange line). Inhibition of Recombinant Human TGF-β1 (0.25 ng/mL) activity elicited by Recombinant Human LAP TGF-β1 (125 ng/mL) is neutralized (green line) by increasing concentrations of Goat Anti-Human LAP TGF-β1 Polyclonal Antibody (Catalog # AB-246-NA).

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 1 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <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

TGF-β1 (transforming growth factor beta 1) and the closely related TGF-β2 and -β3 are members of the large TGF-β superfamily. TGF-β proteins are highly pleiotropic cytokines that regulate processes such as immune function, proliferation and epithelial-mesenchymal transition (1-3). Human TGF-β1 cDNA encodes a 390 amino acid (aa) precursor that contains a 29 aa signal peptide and a 361 aa proprotein (4). A furin-like convertase processes the proprotein within the trans-Golgi to generate an N-terminal 249 aa (aa 30-278) latency-associated peptide (LAP) and a C-terminal 112 aa (aa 279-390) mature TGF-β1 (4-6). Disulfide-linked homodimers of LAP and TGF-β1 remain non-covalently associated after secretion, forming the small latent TGF-β1 complex (4-8). Purified LAP is also capable of associating with active TGF-β with high affinity, and can neutralize TGF-β activity (9). Covalent linkage of LAP to one of three latent TGF-β binding proteins (LTBPs) creates a large latent complex that may interact with the extracellular matrix (5-7). TGF-β activation from latency is controlled both spatially and temporally, by multiple pathways that include actions of proteases such as plasmin and MMP9, and/or by thrombospondin 1 or selected integrins (5, 8). The LAP portion of human TGF-β1 shares 91%, 92%, 85%, 86% and 88% aa identity with porcine, canine, mouse, rat and equine TGF-β1 LAP, respectively, while mature human TGF-β1 portion shares 100% aa identity with porcine, canine and bovine TGF-β1, and 99% aa identity with mouse, rat and equine TGF-β1. Although different isoforms of TGF-β are naturally associated with their own distinct LAPs, the TGF-β1 LAP is capable of complexing with, and inactivating, all other human TGF-β isoforms and those of most other species (9). Mutations within the LAP are associated with Camurati-Engelmann disease, a rare sclerosing bone dysplasia characterized by inappropriate presence of active TGF-β1 (10).

References:

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