

DESCRIPTION

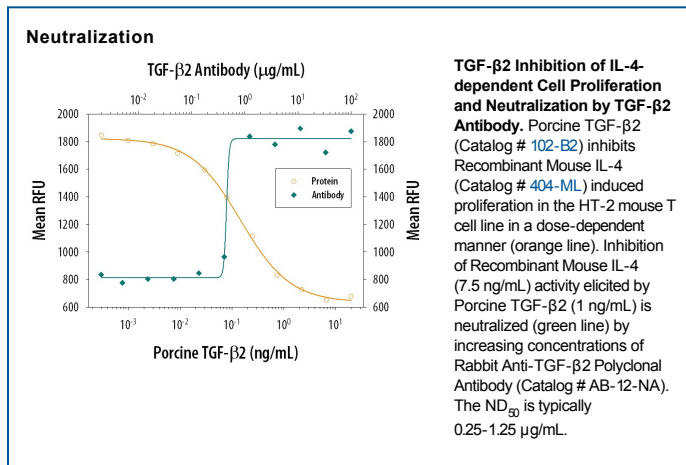
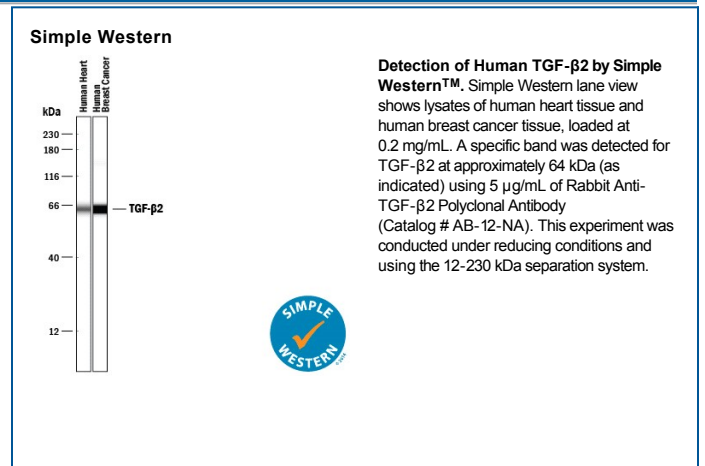
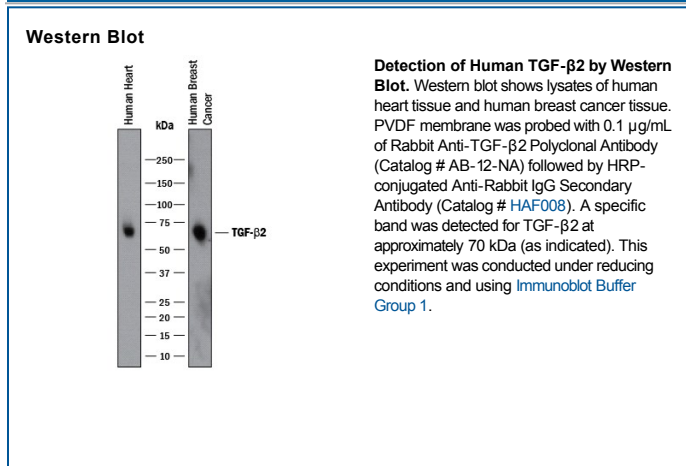
Specificity	Detects TGF-β2 and TGF-β1.2 in direct ELISAs and Western blots. In direct ELISAs and Western blots, less than 2% cross-reactivity with TGF-β3 and TGF-β1 is observed.
Source	Polyclonal Rabbit IgG
Purification	Protein A or G purified
Immunogen	Porcine platelet-derived TGF-β2
Endotoxin Level	<0.10 EU per 1 µg of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS 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	0.1 µg/mL	See Below
Simple Western	5 µg/mL	See Below
Neutralization	Measured by its ability to neutralize TGF-β2 inhibition of IL-4-dependent proliferation 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 0.25-1.25 µg/mL in the presence of 1 ng/mL Porcine TGF-β2 and 7.5 ng/mL Recombinant Mouse IL-4.	

DATA



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- β 2 (transforming growth factor beta 2) is one of three closely related mammalian members of the large TGF- β superfamily that share a characteristic cysteine knot structure (1-7). TGF- β 1, -2 and -3 are highly pleiotropic cytokines proposed to act as cellular switches that regulate processes such as immune function, proliferation and epithelial-mesenchymal transition (1-4). Each TGF- β isoform has some non-redundant functions; for TGF- β 2, mice with targeted deletion show defects in development of cardiac, lung, craniofacial, limb, eye, ear and urogenital systems (2). Human TGF- β 2 cDNA encodes a 414 amino acid (aa) precursor that contains a 19 aa signal peptide and a 395 aa proprotein (8). A furin-like convertase processes the proprotein to generate an N-terminal 232 aa latency-associated peptide (LAP) and a C-terminal 112 aa mature TGF- β 2 (8, 9). Disulfide-linked homodimers of LAP and TGF- β 2 remain non-covalently associated after secretion, forming the small latent TGF- β 1 complex (8-10). 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 (9, 10). TGF- β is activated from latency by pathways that include actions of the protease plasmin, matrix metalloproteases, thrombospondin 1 and a subset of integrins (10). Mature human TGF- β 2 shows 100% aa identity with porcine, canine, equine and bovine TGF- β 2, and 97% aa identity with mouse and rat TGF- β 2. It demonstrates cross-species activity (1). TGF- β 2 signaling begins with binding to a complex of the accessory receptor betaglycan (also known as TGF- β RIII) and a type II ser/thr kinase receptor termed TGF- β RII. This receptor then phosphorylates and activates another ser/thr kinase receptor, TGF- β RI (also called activin receptor-like kinase (ALK)-5), or alternatively, ALK-1. The whole complex phosphorylates and activates Smad proteins that regulate transcription (3, 11, 12). Use of other signaling pathways that are Smad-independent allows for disparate actions observed in response to TGF- β in different contexts (11).

References:

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