

DESCRIPTION

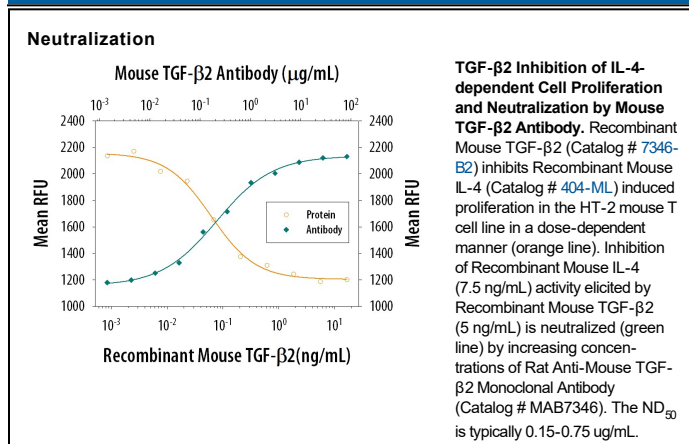
Species Reactivity	Mouse
Specificity	Detects mouse TGF- β 2 in direct ELISAs. In direct ELISAs, approximately 50% cross-reactivity with recombinant human (rh) TGF-beta 2 and rhTGF-beta 3 is observed.
Source	Monoclonal Rat IgG _{2B} Clone # 771213
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Chinese hamster ovary cell line CHO-derived recombinant mouse TGF- β 2 Ala303-Ser414 Accession # P27090
Endotoxin Level	<0.10 EU per 1 μ g of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 μ m filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 μ 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.

Neutralization	Measured by its ability to neutralize TGF- β 2 inhibition of IL-4-dependent proliferation in the HT-2 mouse T cell line. Tsang, M. <i>et al.</i> (1995) Cytokine 7:389. The Neutralization Dose (ND ₅₀) is typically 0.15-0.75 μ g/mL in the presence of 5 ng/mL Recombinant Mouse TGF- β 2 and 7.5 ng/mL Recombinant Mouse IL-4.
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DATA



PREPARATION AND STORAGE

Reconstitution	Sterile PBS to a final concentration of 0.5 mg/mL.
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

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. 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. 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. Mouse TGF- β 2 cDNA encodes a 414 amino acid (aa) precursor that contains a 19 aa signal peptide and a 395 aa proprotein. A furin-like convertase processes the proprotein to generate an N-terminal 283 aa latency-associated peptide (LAP) and a C-terminal 112 aa mature TGF- β 2. Disulfide-linked homodimers of LAP and TGF- β 2 remain non-covalently associated after secretion, forming the small latent TGF- β 2 complex. 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. TGF- β is activated from latency by pathways that include actions of the protease plasmin, matrix metalloproteases, thrombospondin 1 and a subset of integrins. Mature mouse TGF- β 2 shares 100% aa identity with rat TGF- β 2, and 97% aa identity with human, porcine, canine, equine and bovine TGF- β 2. It demonstrates cross-species activity. In most cells, 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, which 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. In bone-related cells, however, TGF- β 2 also signals through TGF- β RIIB (a splice variant of TGF- β RII), independently of TGF- β RIII. Use of other signaling pathways that are Smad-independent allows for disparate actions observed in response to TGF- β in different contexts.