

## DESCRIPTION

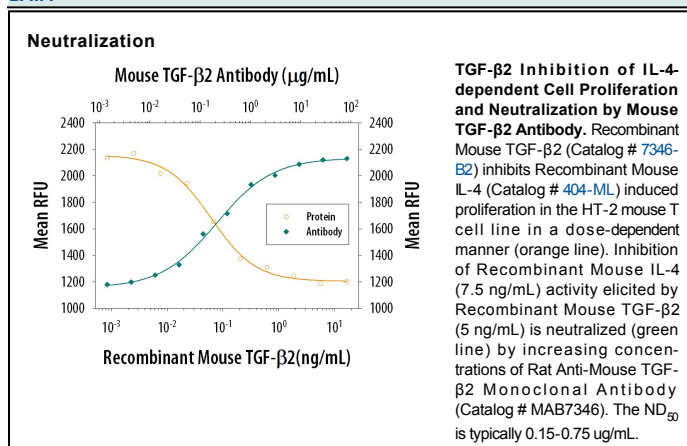
<b>Species Reactivity</b>	Mouse
<b>Specificity</b>	Detects mouse TGF-β2 in direct ELISAs. In direct ELISAs, approximately 50% cross-reactivity with recombinant human (rh) TGF-beta 2 and rhTGF-beta 3 is observed.
<b>Source</b>	Monoclonal Rat IgG <sub>2B</sub> Clone # 771213
<b>Purification</b>	Protein A or G purified from hybridoma culture supernatant
<b>Immunogen</b>	Chinese hamster ovary cell line CHO-derived recombinant mouse TGF-β2 Ala303-Ser414 Accession # P27090
<b>Endotoxin Level</b>	<0.10 EU per 1 μg of the antibody by the LAL method.
<b>Formulation</b>	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.

<b>Neutralization</b>	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 <sub>50</sub> ) is typically 0.15-0.75 μg/mL in the presence of 5 ng/mL Recombinant Mouse TGF-β2 and 7.5 ng/mL Recombinant Mouse IL-4.
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## DATA



## PREPARATION AND STORAGE

<b>Reconstitution</b>	Sterile PBS to a final concentration of 0.5 mg/mL.
<b>Shipping</b>	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
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>• 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>• 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>• 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

## BACKGROUND

TGF-β2 (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-β1, -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-β2, mice with targeted deletion show defects in development of cardiac, lung, craniofacial, limb, eye, ear and urogenital systems. Mouse TGF-β2 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-β2. Disulfide-linked homodimers of LAP and TGF-β2 remain non-covalently associated after secretion, forming the small latent TGF-β2 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-β2 shares 100% aa identity with rat TGF-β2, and 97% aa identity with human, porcine, canine, equine and bovine TGF-β2. It demonstrates cross-species activity. In most cells, TGF-β2 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-β2 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.