

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived Ala303-Ser414 Accession # P61812
N-terminal Sequence Analysis	Ala303
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	12.7 kDa (monomer)

SPECIFICATIONS

SDS-PAGE	12 kDa, reducing conditions 24 kDa, non-reducing conditions
Activity	Measured by its ability to inhibit the IL-4-dependent proliferation of HT-2 mouse T cells. Tsang, M. <i>et al.</i> (1995) Cytokine 7:389. The ED ₅₀ for this effect is 0.025-0.25 ng/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>97%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in Acetonitrile and TFA. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute 2 µg vial at 5 µg/mL in sterile 4 mM HCl. Reconstitute 10 µg or larger vials at 100 µg/mL in sterile 4 mM HCl.
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. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

DATA

<p>Bioactivity</p> <p>Recombinant Human TGF-β2 (Catalog # 302-B2/CF) inhibits Recombinant Mouse IL-4 (Catalog # 404-ML) induced cell proliferation in the HT-2 mouse T cell line. The ED₅₀ for this effect is 0.025-0.25 ng/mL.</p>	<p>SDS-PAGE</p> <p>1 µg/lane of Recombinant Human TGF-β2 was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by silver staining, showing single bands at 12 kDa and 24 kDa, respectively.</p>
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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:

1. Sporn, M.B. (2006) Cytokine Growth Factor Rev. **17**:3.
2. Dunker, N. and K. Kriegelstein, 2000, Eur. J. Biochem. **267**:6982.
3. Wahl, S.M. (2006) Immunol. Rev. **213**:213.
4. Chang, H. *et al.* (2002) Endocr. Rev. **23**:787.
5. Lin, J.S. *et al.* (2006) Reproduction **132**:179.
6. Hinck, A.P. *et al.* (1996) Biochemistry **35**:8517.
7. Mittl, P.R.E. *et al.* (1996) Protein Sci. **5**:1261.
8. deMartin, R. *et al.* (1987) EMBO J. **6**:3673.
9. Miyazono, K. *et al.* (1988) J. Biol. Chem. **263**:6407.
10. Oklu, R. and R. Hesketh (2000) Biochem. J. **352**:601.
11. de Caestecker, M. *et al.* (2004) Cytokine Growth Factor Rev. **15**:1.
12. Zuniga, J.E. *et al.* (2005) J. Mol. Biol. **354**:1052.