**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. et al. (1995) Cytokine 7:389. The ED₅₀ 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 with BSA as a carrier protein. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

Reconstitution: Reconstitute at 20 μg/mL in sterile 4 mM HCl containing at least 0.1% human or bovine serum albumin.

Shipping: The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage:

- 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**

Bioactivity: Recombinant Human TGF-β2 (Catalog # 302-B2) 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.

SDS-PAGE: 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.
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 (1-7). 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 (1-4). 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 (2). Human TGF-β2 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-β2 (8, 9). Disulfide-linked homodimers of LAP and TGF-β2 remain non-covalently associated after secretion, forming the small latent TGF-β1 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-β2 shows 100% aa identity with porcine, canine, equine and bovine TGF-β2, and 97% aa identity with mouse and rat TGF-β2. It demonstrates cross-species activity (1). 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. 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: