

DESCRIPTION

Source Chinese Hamster Ovary cell line, CHO-derived
Leu30-Ser390
Accession # P01137

N-terminal Sequence Analysis Leu30 & Ala279

Predicted Molecular Mass 41.3 kDa (monomer)

SPECIFICATIONS

SDS-PAGE 40-43 kDa & 12 kDa, reducing conditions
80-90 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 20-60 ng/mL before acid activation and 0.15-0.5 ng/mL after acid activation.

Endotoxin Level <1.0 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 Supplied as a 0.2 µm filtered solution in PBS and Glycerol with BSA as a carrier protein. See Certificate of Analysis for details.

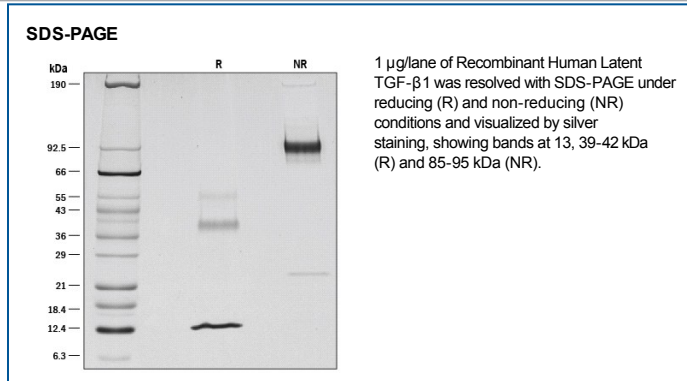
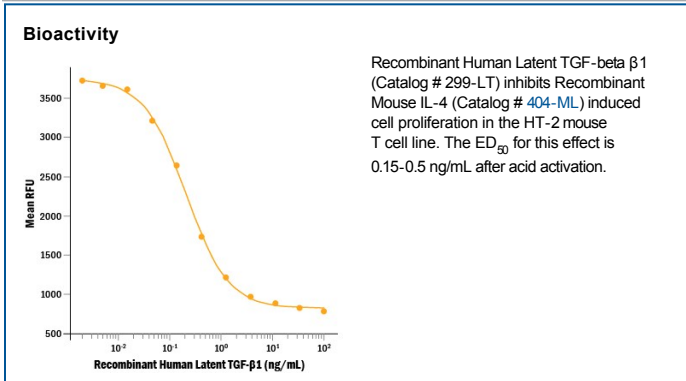
PREPARATION AND STORAGE

Shipping The product is shipped with dry ice or equivalent. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after opening.

DATA



BACKGROUND

TGF-β1 (transforming growth factor beta 1) and the closely related TGF-β2 and -3 are members of the large TGF-β superfamily. TGF-β proteins are highly pleiotropic cytokines that regulate processes such as immune function, proliferation and epithelial-mesenchymal transition (1-3). Human TGF-β1 cDNA encodes a 390 amino acid (aa) precursor that contains a 29 aa signal peptide and a 361 aa proprotein (4). A furin-like convertase processes the proprotein within the trans-Golgi to generate an N-terminal 249 aa latency-associated peptide (LAP) and a C-terminal 112 aa mature TGF-β1 (4-6). Disulfide-linked homodimers of LAP and TGF-β1 remain non-covalently associated after secretion, forming the small latent TGF-β1 complex (4-8). Purified LAP is also capable of associating with active TGF-β with high affinity, and can neutralize TGF-β activity (9). 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 (5-7). TGF-β activation from latency is controlled both spatially and temporally, by multiple pathways that include actions of proteases such as plasmin and MMP9, and/or by thrombospondin 1 or selected integrins (5, 8). The LAP portion of human TGF-β1 shares 91%, 92%, 85%, 86% and 88% aa identity with porcine, canine, mouse, rat and equine TGF-β1 LAP, respectively, while the mature human TGF-β1 portion shares 100% aa identity with porcine, canine and bovine TGF-β1, and 99% aa identity with mouse, rat and equine TGF-β1. Although different isoforms of TGF-β are naturally associated with their own distinct LAPs, the TGF-β1 LAP is capable of complexing with, and inactivating, all other human TGF-β isoforms and those of most other species (9). Mutations within the LAP are associated with Camurati-Engelmann disease, a rare sclerosing bone dysplasia characterized by inappropriate presence of active TGF-β1 (10).

References:

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3. Chang, H. *et al.* (2002) *Endocr. Rev.* **23**:787.
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5. Dabovic, B. and D.B. Rifkin (2008) "TGF-β Bioavailability" in *The TGF-β Family*. Derynck, R. and K. Miyazono (eds): Cold Spring Harbor Laboratory Press, p. 179.
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