**DESCRIPTION**

**Source**  
Spodoptera frugiperda, Sf 21 (baculovirus)-derived  
Leu30-Arg278 (Cys33Ser)  
Accession # P01137.2

**N-terminal Sequence Analysis**  
Leu30

**Structure / Form**  
Disulfide-linked homodimer

**Predicted Molecular Mass**  
27 kDa (monomer)

**SPECIFICATIONS**

**SDS-PAGE**  
28-36 kDa, reducing & 60-70 kDa, non-reducing conditions (variably glycosylated)

**Activity**  
Measured by its ability to inhibit TGF-β1 activity on HT-2 mouse T cells. Tsang, M. et al. (1995) Cytokine 7:389. The ED₅₀ for this effect is 50-300 ng/mL in the presence of 1 ng/mL of Recombinant Human TGF-β1 (Catalog # 240-B).

**Endotoxin Level**  
<0.10 EU per 1 μg of the protein by the LAL method.

**Purity**  
>97%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

**Formulation**  
Lyophilized from a 0.2 μm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

**Reconstitution**  
Reconstitute at 100 μg/mL in sterile PBS 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**  
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 reconstitution.
- 3 months, 20 to -70 °C under sterile conditions after reconstitution.

**BACKGROUND**

TGF-β1 (transforming growth factor beta 1) and the closely related TGF-β2 and -β3 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-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-β1 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 and bovine TGF-β1, respectively, while 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:**