Recombinant Human Latent TGF-β1

Catalog Number: 299-LT

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

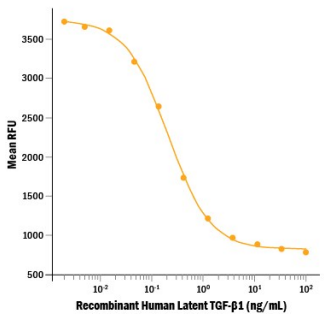
SDS-PAGE	40-43 kDa & 12 kDa, reducing conditions 80-90 kDa, non-reducing conditions
Activity	Measured by its ability to inhibit the IL-4-dependent proliferation of HT-2 mouse T cells. Tsang, M. *et al.* (1995) Cytokine **7**:389. The ED_{50} for this effect is 20-60 ng/mL before acid activation and 0.15-0.5 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.

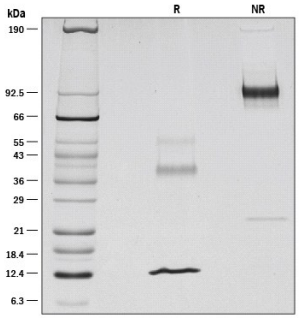
DATA

Bioactivity



Recombinant Human Latent TGF-beta β1 (Catalog # 299-LT) inhibits Recombinant Mouse IL-4 (Catalog # 404-ML) induced cell proliferation in the HT-2 mouse T cell line. The ED_{50} for this effect is 0.15-0.5 ng/mL after acid activation.

SDS-PAGE



1 µg/lane of Recombinant Human Latent TGF-β1 was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by silver staining, showing bands at 13, 39-42 kDa (R) and 85-90 kDa (NR).

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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 procine, 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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