

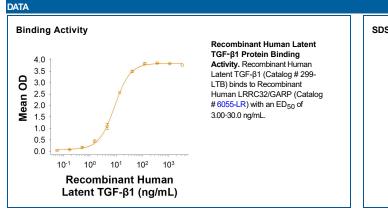
Recombinant Human Latent TGF-β1

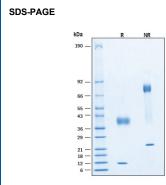
Catalog Number: 299-LTB

DESCRIPTION	
Source	Chinese Hamster Ovary cell line, CHO-derived human TGF-beta 1 protein Leu30-Ser390 Accession # P01137
N-terminal Sequence Analysis	Leu30 (LAP) & Ala 279 (Mature)
Predicted Molecular Mass	29 kDa (LAP) & 13 kDa (Mature)

SPECIFICATIONS	
SDS-PAGE	36-42 kDa & 9-13 kDa, under reducing conditions
Activity	Measured by its binding ability in a functional ELISA. Recombinant Human Latent TGF-β1 (Catalog # 299-LTB) binds to Recombinant Human LRRC32/GARP (Catalog # 6055-LR) with an ED ₅₀ of 3.00-30.0 ng/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Supplied as a 0.2 µm filtered solution in PBS with Trehalose. 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, -70 °C as supplied.	
	 2 weeks, 2 to 8 °C under sterile conditions after opening. 3 months, -20 to -70 °C under sterile conditions after opening. 	





Recombinant Human Latent TGF-β1 Protein SDS-PAGE. 2 µg/lane of Recombinant Human Latent TGF-β1 Protein (Catalog # 299-LTB) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 36-42 kDa & 9-13 kDa, and 70-80 kDa and 18-26 kDa, respectively.

Rev. 11/6/2024 Page 1 of 2



Recombinant Human Latent TGF-β1

Catalog Number: 299-LTB

BACKGROUND

TGF- β 1 (transforming growth factor beta 1) and the closely related TGF- β 2 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- β 1 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- β 1 (4-6). Disulfide-linked homodimers of LAP and TGF- β 1 remain non-covalently associated after secretion, forming the small latent TGF- β 1 complex (4-8). Purified LAP is also capable of associating with active TGF- β 8 with high affinity, and can neutralize TGF- β 5 activity (9). Covalent linkage of LAP to one of three latent TGF- β 6 binding proteins (LTBPs) creates a large latent complex that may interact with the extracellular matrix (5-7). TGF- β 8 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- β 1 shares 91%, 92%, 85%, 86% and 88% aa identity with porcine, canine, mouse, rat and equine TGF- β 1 LAP, respectively, while the mature human TGF- β 1 portion shares 100% aa identity with procine, canine and bovine TGF- β 1, and 99% aa identity with mouse, rat and equine TGF- β 1. Although different isoforms of TGF- β 1 are naturally associated with their own distinct LAPs, the TGF- β 1 LAP is capable of complexing with, and inactivating, all other human TGF- β 1 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- β 1 (10).

References:

- 1. Dunker, N. and K. Krieglstein (2000) Eur. J. Biochem. 267:6982.
- 2. Wahl, S.M. (2006) Immunol. Rev. 213:213.
- 3. Chang, H. et al. (2002) Endocr. Rev. 23:787.
- 4. Derynck, R. et al. (1985) Nature 316:701.
- 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.
- 6. Brunner, A.M. et al. (1989) J. Biol. Chem. 264:13660.
- 7. Miyazono, K. et al. (1991) EMBO J. 10:1091.
- 8. Oklu, R. and R. Hesketh (2000) Biochem. J. 352:601.
- 9. Miller, D.M. et al. (1992) Mol. Endocrinol. 6:694.
- 10. Janssens, K. et al. (2003) J. Biol. Chem. 278:7718.