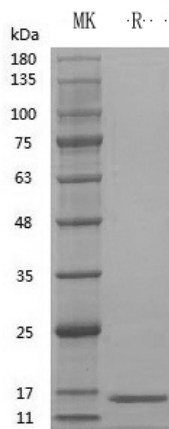


Summary

Name	TGF- β 1
Purity	Greater than 95% as determined by reducing SDS-PAGE
Endotoxin level	\leq 10 EU/mg
Construction	Recombinant Human TGF- β 1 is produced by our Mammalian cell expression system and the target gene encoding Ala279-Ser390 is expressed.
Accession #	P01137
Tag	Tag free
Host	Mammalian cell
Species	Human
Predicted MW	12.8 kDa
Form	Lyophilized
Buffer	100 mM Glycine, 150 mM NaCl, 5% mannitol and 0.01% Tween 80, pH 4.0
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature listed below.
Stability&Storage	Store at \leq -70°C, stable for 6 months after receipt. Store at \leq -70°C, stable for 3 months under sterile conditions after opening. Please minimize freeze-thaw cycles.
Reconstitution	Always centrifuge tubes before opening. Do not mix by vortex or pipetting. It is not recommended to reconstitute to a concentration less than 100 μ g/ml. Dissolve the lyophilized protein in distilled water. Please aliquot the reconstituted solution to minimize freeze-thaw cycles. Always centrifuge tubes before opening. Do not mix by vortex or pipetting. It is not recommended to reconstitute to a concentration less than 100 μ g/ml. Dissolve the lyophilized protein in distilled water. Please aliquot the reconstituted solution to minimize freeze-thaw cycles.

SDS-PAGE image

Product Name: GMP Recombinant Human TGF- β 1
Catalog#: PHH90058



Background

Alternative Names

Transforming Growth Factor Beta-1; TGF-Beta-1; Latency-Associated Peptide; LAP; TGFB1; TGFB

References

Transforming Growth Factor β -1 (TGF β -1) is a secreted protein which belongs to the TGF- β family. TGF β -1 is abundantly expressed in bone, articular cartilage and chondrocytes and is increased in osteoarthritis (OA). TGF β -1 performs many cellular functions, including the control of cell growth, cell proliferation, cell differentiation and apoptosis. The precursor is cleaved into a latency-associated peptide (LAP) and a mature TGF β -1 peptide. Disulfide-linked homodimers of LAP and TGF-beta 1 remain non-covalently associated after secretion, forming the small latent TGF-beta 1 complex. Purified LAP is also capable of associating with active TGF-beta with high affinity, and can neutralize TGF-beta activity. Covalent linkage of LAP to one of three latent TGF-beta binding proteins (LTBPs) creates a large latent complex that may interact with the extracellular matrix. TGF-beta 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. Although different isoforms of TGF-beta are naturally associated with their own distinct LAPs, the TGF-beta 1 LAP is capable of complexing with, and inactivating, all other human TGF-beta isoforms and those of most other species. Mutations within the LAP are associated with Camurati-Engelmann disease, a rare sclerosing bone dysplasia characterized by inappropriate presence of active TGF-beta 1.

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Note

For research use only .