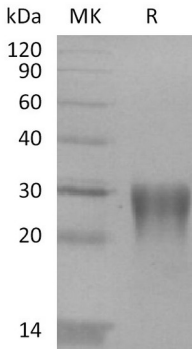
Product Name: Recombinant Human LAP (TGF-beta 1)
Catalog #: PHH2193

Summary

Name	LAP (TGF-beta 1)
Purity	Greater than 95% as determined by reducing SDS-PAGE
Endotoxin level	<1 EU/μg as determined by LAL test.
Construction	Recombinant Human Transforming Growth Factor beta 1 is produced by our Mammalian expression system and the target gene encoding Leu30-Arg278(Cys33Ser) is expressed.
Accession #	P01137
Host	Human Cells
Species	Human
Predicted Molecular Mass	28.5 KDa
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature listed below.
Stability&Storage	Store at ≤-70°C, stable for 6 months after receipt. Store at ≤-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 100μg/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 100μg/ml. Dissolve the lyophilized protein in distilled water. Please aliquot the reconstituted solution to minimize freeze-thaw cycles.

SDS-PAGE image



Background

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Alternative Names	Transforming Growth Factor Beta-1; TGF-Beta-1; Latency-Associated Peptide; LAP; TGFB1; TGFB
Background	Transforming Growth Factor β-1 (TGFβ-1) is a secreted protein which belongs to the TGF-β family. TGFβ-1 is abundantly expressed in bone, articular cartilage and chondrocytes and is increased in osteoarthritis (OA). TGFβ-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 TGFβ-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.

Note

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