ELK Biotechnology

Recombinant Human LAP (TGF-beta 1)

Catalog #	EPT048
Expression Host	Human Cells
DESCRIPTION	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
Synonyms	Transforming Growth Factor Beta-1; TGF-Beta-1; Latency-Associated Peptide; LAP; TGFB1; TGFB
Mol Mass	28.5 KDa
AP Mol Mass	20-30 KDa, reducing conditions
Purity	Greater than 95% as determined by reducing SDS-PAGE.
Endotoxin	Less than 0.1 ng/μg (1 EU/μg) as determined by LAL test.
FORMULATION	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4.
RECONSTITUTION	Always centrifuge tubes before opening.Do not mix by

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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.

SHIPPING

The product is shipped at ambient temperature.

Upon receipt, store it immediately at the temperature listed below.

STORAGE

Lyophilized protein should be stored at < -20 ° C, though stable at room temperature for 3 weeks.

Reconstituted protein solution can be stored at 4-7°C for 2-7 days.

Aliquots of reconstituted samples are stable at < -20° C for 3 months.

BACKGROUND

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 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.

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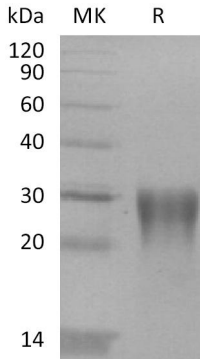

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