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Product Datasheet

Recombinant Human LAP (TGF-beta 1)
EBT-EPT048

Artikelname	Recombinant Human LAP (TGF-beta 1)
Artikelnummer	EBT-EPT048
Hersteller Artikelnummer	EPT048
Alternativnummer	EBT-EPT048-10
Hersteller	ELK Biotechnology
Kategorie	Proteine/Peptide
Produktbeschreibung	Recombinant Human Transforming Growth Factor beta 1 is produced by our Mammalian expression system and the target gene encoding Leu30-Arg278(Cys33Ser) is expressed....
Molekulargewicht	Molecular weight: 28.5 KDa. Apparent molecular weight: 20-30 KDa, reducing conditions
UniProt	P01137
Reinheit	Greater than 95% as determined by reducing SDS-PAGE.

Anwendungsbeschreibung	Redissolve: 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.. Endotoxin: Less than 0.1 ng/µg (1 EU/µg) as determined by LAL test. Background: Transforming Growth Factor beta-1 (TGFbeta-1) is a secreted protein which belongs to the TGF-beta family. TGFbeta-1 is abundantly expressed in bone, articular cartilage and chondrocytes and is increased in osteoarthritis (OA). TGFbeta-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 TGFbeta-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