Recombinant Human Transforming Growth Factor Beta-1/TGFB1

Catalog No. PKSH033947

Note: Centrifuge before opening to ensure complete recovery of vial contents.

Description		
Synonyms	Transforming Growth Factor Beta-1;TGF-Beta-1;Latency-Associated Peptide;LAP;TGFB1;TGFB	
Species	Human	
Expression Host	HEK293 Cells	
Sequence	Leu30-Arg278(Cys33Ser)	
Accession	P01137	
Calculated Molecular Weight	28.5 kDa	
Observed molecular weight	20-30 kDa	
Tag	None	
Bioactivity	Not validated for activity	
Properties		
Purity	> 95 % as determined by reducing SDS-PAGE.	
Endotoxin	< 1.0 EU per μ g of the protein as determined by the LAL method.	
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.	
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.	
Formulation	Lyophilized from a 0.2 µm filtered solution of PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.	
Reconstitution	Please refer to the printed manual for detailed information.	
Data		

kDa	МК	R
120 90		
60		
40		
30	-	
20	-	107
- 1		
14	anar-	

> 95 % as determined by reducing SDS-PAGE.

Background

Transforming Growth Factor β -1 (TGF β -1) is a secreted protein which belongs to the TGF- β family. TGF β -1 is

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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.