

## Recombinant Human Transforming Growth Factor Beta-1/TGFB1

Catalog Number: PKSH033947

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

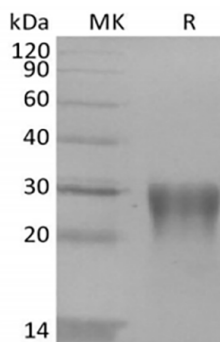
### Description

<b>Species</b>	Human
<b>Source</b>	HEK293 Cells-derived Human TGFB1 protein Leu30-Arg278(Cys33Ser)
<b>Calculated MW</b>	28.5 kDa
<b>Observed MW</b>	20-30 kDa
<b>Accession</b>	P01137
<b>Bio-activity</b>	Not validated for activity

### Properties

<b>Purity</b>	> 95 % as determined by reducing SDS-PAGE.
<b>Endotoxin</b>	< 1.0 EU per µg of the protein as determined by the LAL method.
<b>Storage</b>	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.
<b>Shipping</b>	This product is provided as lyophilized powder which is shipped with ice packs.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution of PBS, pH 7.4. Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
<b>Reconstitution</b>	Please refer to the specific buffer information in the printed manual. Please refer to the printed manual for detailed information.

### Data



> 95 % as determined by reducing SDS-PAGE.

### Background

Transforming Growth Factor  $\beta$ -1 (TGF $\beta$ -1) is a secreted protein which belongs to the TGF- $\beta$  family. TGF $\beta$ -1 is abundantly expressed in bone, articular cartilage and chondrocytes and is increased in osteoarthritis (OA). TGF $\beta$ -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 $\beta$ -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.