

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived human PDGF R beta protein Leu33-Lys531, With a C-terminal 6-His tag & Avi-tag Accession # AAA36427.1
N-terminal Sequence Analysis	Leu 33
Structure / Form	Biotinylated via Avi-tag
Predicted Molecular Mass	59 kDa

SPECIFICATIONS

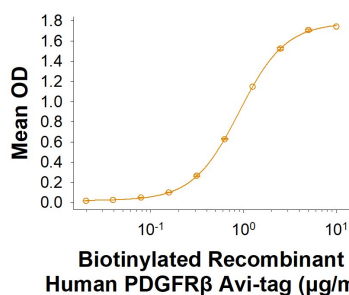
SDS-PAGE	100-110 kDa, under reducing conditions
Activity	Measured by its binding ability in a functional ELISA. Biotinylated Recombinant Human PDGFR β His-tag Avi-tag binds to Recombinant Human PDGF-BB Protein (Catalog # 220-BB) with an ED ₅₀ of 0.150-1.50 μ g/mL.
Endotoxin Level	<0.10 EU per 1 μ g of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Supplied as a 0.2 μ m filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Shipping	The product is shipped with dry ice or equivalent. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

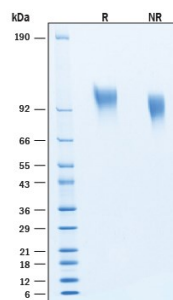
DATA

Binding Activity



Biotinylated Recombinant Human PDGF R β His-tag Avi-tag Protein Binding Activity. Measured by its binding ability in a functional ELISA. Biotinylated Recombinant Human PDGFR β His-tag Avi-tag Protein (Catalog # AV110676) binds to Recombinant Human PDGF-BB Protein (Catalog # 220-BB) with an ED₅₀ of 0.150-1.50 μ g/mL.

SDS-PAGE



Biotinylated Recombinant Human PDGF R β His-tag Avi-tag Protein SDS-PAGE. 2 μ g/lane of Biotinylated Recombinant Human PDGF R β His-tag Avi-tag Protein (Catalog # AV110676) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 100-110 kDa, under reducing conditions.

BACKGROUND

The platelet-derived growth factor (PDGF) family consists of proteins derived from four genes (PDGF-A, -B, -C, and -D) that form disulfide-linked homodimers (PDGF-AA, -BB, -CC, and -DD) and a heterodimer (PDGF-AB) (1, 2). These proteins regulate diverse cellular functions by binding to and inducing the homo- or heterodimerization of two receptors (PDGF R α and R β). Whereas α/α homo-dimerization is induced by PDGF-AA, -BB, -CC, and -AB, α/β hetero-dimerization is induced by PDGF-AB, -BB, -CC, and -DD, and β/β homo-dimerization is induced only by PDGF-BB, and -DD (1 - 4). Both PDGF R α and R β are members of the class III subfamily of receptor tyrosine kinases (RTK) that also includes the receptors for M-CSF, SCF and Flt3-ligand. All class III RTKs are characterized by the presence of five immunoglobulin-like domains in their extracellular region and a split kinase domain in their intracellular region. The extracellular domain of human PDGF R beta contains 4 disulfide bonds and shares a 79% sequence identity with mouse and rat PDGF R beta. Ligand-induced receptor dimerization results in autophosphorylation in trans resulting in the activation of several intracellular signaling pathways that can lead to cell proliferation, cell survival, cytoskeletal rearrangement, and cell migration. Many cell types, including fibroblasts and smooth muscle cells, express both the α and β receptors. Others have only the α receptors (oligodendrocyte progenitor cells, mesothelial cells, liver sinusoidal endothelial cells, astrocytes, platelets and megakaryocytes) or only the β receptors (myoblasts, capillary endothelial cells, pericytes, T cells, myeloid hematopoietic cells and macrophages). A soluble PDGF R α has been detected in normal human plasma and serum as well as in the conditioned medium of the human osteosarcoma cell line MG-63 (5). Both the recombinant mouse and human soluble PDGF R α bind PDGF with high affinity and are potent PDGF antagonists. Our Avi-tag Biotinylated human PDGF R beta features biotinylation at a single site contained within the Avi-tag, a unique 15 amino acid peptide. Protein orientation will be uniform when bound to streptavidin-coated surface due to the precise control of biotinylation and the rest of the protein is unchanged so there is no interference in the protein's bioactivity.

References:

1. Betsholtz, C. *et al.* (2001) *BioEssays* **23**:494.
2. Ostman, A. and A.H. Heldin (2001) *Advances in Cancer Research* **80**:1.
3. Gilbertson, D. *et al.* (2001) *J. Biol. Chem.* **276**:27406.
4. LaRocheells, W.J. *et al.* (2001) *Nature Cell Biol.* **3**:517.
5. Tiesman, J. and C.E. Hart (1993) *J. Biol. Chem.* **5**:9621.