

DESCRIPTION

Source Chinese Hamster Ovary cell line, CHO-derived human PDGF R alpha protein
Gln24 - Glu524, with a C-terminal 6-His tag
Accession # P16234.1

N-terminal Sequence Analysis Gln24, deduced from Leu25 upon deblocking

Predicted Molecular Mass 59 kDa

SPECIFICATIONS

SDS-PAGE 89-100 kDa, under reducing conditions.

Activity Measured by its binding ability in a functional ELISA.
When Recombinant Human PDGF-AA Protein (Catalog # 221-AA) is presented at 2 μ g/mL (100 μ L/well), Recombinant Human PDGF R α His-tag (Catalog # 10382-PR) binds with an ED₅₀ of 2.00-16.00 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 Lyophilized from a 0.2 μ m filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 500 μ g/mL in PBS.

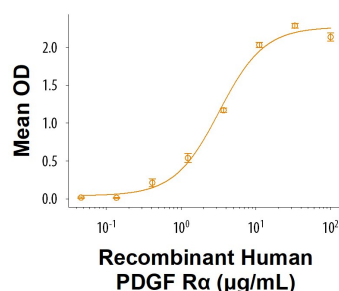
Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

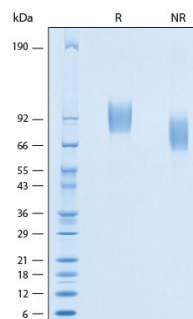
DATA

Binding Activity



Recombinant Human PDGF R alpha His-tag Protein Binding Activity. Measured by its binding ability in a functional ELISA. When Recombinant Human PDGF-AA Protein (Catalog # 221-AA) is presented at 2 μ g/mL (100 μ L/well), Recombinant Human PDGF R α His-tag Protein (Catalog # 10383-PR) binds with an ED₅₀ of 2.00-16.00 μ g/mL.

SDS-PAGE



Recombinant Human PDGF R alpha His-tag Protein SDS-PAGE. 2 μ g/lane of Recombinant Human PDGF R alpha His-tag Protein (Catalog # 10383-PR) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 89-100 kDa.

BACKGROUND

PDGF R α (platelet-derived growth factor receptor alpha) is a type I transmembrane glycoprotein in the class III subfamily of receptor tyrosine kinases (RTK) (1 - 4). PDGF R α and PDGF R β can form homo- or hetero-dimeric receptors when engaged by dimers of the PDGF family of growth factors, which include disulfide-linked homodimers of PDGF-A, B, C or D, or the heterodimer PDGF-AB that is mainly found in human platelets. While multiple *in vitro* ligand-receptor combinations have been identified, *in vivo* evidence indicates that PDGF R α primarily binds PDGF-AA and PDGF-CC, while PDGF R β primarily binds PDGF-BB and probably PDGF-DD. Like all class III RTKs, the extracellular domain (ECD) of human PDGF R α (aa 24 - 524) contains five immunoglobulin-like domains, while the intracellular region contains a split tyrosine kinase domain (aa 593 - 954) (1 - 4). Within the ECD, human PDGF R α shares 85%, 83%, 95%, 93%, and 88% aa sequence identity with mouse, rat, equine, canine and bovine PDGF R α respectively. PDGF R α autophosphorylates upon dimerization, activating signaling cascades in PI 3-kinase Ras-MAP kinase, and PLC- γ pathways (1, 2). Signaling is down-regulated by SHP-2 phosphatase activity and by receptor endocytosis and lysosomal degradation. PDGF R α is expressed at low levels in most mesenchymal cells, but is strongly expressed in oligodendrocyte, lung, skin and intestinal progenitor cells and induced by inflammation or growth in culture (1 - 4). During development, mesenchymal cells expressing PDGF R α respond to local gradients of epithelially produced PDGF-AA or PDGF-CC during formation of the cranial and cardiac neural crest, retina, gonads, lung alveoli, intestinal villi, skin, hair follicles, skeleton, teeth, palate, and interstitial kidney mesenchyme (1, 5). Deletion of PDGF R α in mice severely impairs mesenchymal derivatives in both embryo and extraembryonic tissues, and high or low PDGF R α signaling in humans may result in spina bifida or cleft palate-type malformations. Postnatally, PDGF R α is implicated in gliomas and fibrotic disorders of lung, heart and skin (scleroderma) (6 - 8).

References:

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2. Heldin, C-H. and B. Westermark (1999) *Physiol. Rev.* **79**:1283.
3. Claesson-Welsh, L. *et al.* (1989) *Proc. Natl. Acad. Sci. USA* **86**:4917.
4. Matsui, T. *et al.* (1989) *Science* **243**:800.
5. Klinghoffer, R.A. *et al.* (2002) *Dev. Cell* **2**:103.
6. Martinho, O. (2009) *Br. J. Cancer* **101**:973.
7. Olson, L.E. and P. Soriano (2009) *Dev. Cell* **16**:303.
8. Baroni, S.S. *et al.* (2006) *N. Engl. J. Med.* **354**:2667.