

## Background

PDGF R $\alpha$  (platelet-derived growth factor receptor alpha) is a type I transmembrane glycoprotein in the class III subfamily of receptor tyrosine kinases (RTK) (1 - 3). PDGF R $\alpha$  and PDGF R $\beta$  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 $\alpha$  primarily binds PDGF-AA and PDGF-CC, while PDGF R $\beta$  primarily binds PDGF-BB and probably PDGF-DD. Like all class III RTKs, the extracellular domain (ECD) of mouse PDGF R $\alpha$  (aa 25 - 525) contains five immunoglobulin-like domains, while the intracellular region contains a split tyrosine kinase domain (aa 593 - 954). Within the ECD, mouse PDGF R $\alpha$  shares 85%, 93%, 84%, 84%, and 81% aa sequence identity with human, rat, equine, canine and bovine PDGF R $\alpha$  respectively. PDGF R $\alpha$  autophosphorylates upon dimerization, activating signaling cascades in PI 3-kinase Ras-MAP kinase, and PLC- $\gamma$  pathways (1, 2). Signaling is down-regulated by SHP-2 phosphatase activity and by receptor endocytosis and lysosomal degradation. PDGF R $\alpha$  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 (1-3). During development, mesenchymal cells expressing PDGF R $\alpha$  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, 4). Deletion of PDGF R $\alpha$  in mice severely impairs mesenchymal derivatives in both embryo and extraembryonic tissues, and high or low PDGF R $\alpha$  signaling in humans may result in spina bifida or cleft palate-type malformations. Postnatally, PDGF R $\alpha$  is implicated in gliomas and fibrotic disorders of lung, heart and skin (scleroderma) (5 - 7).

## References:

1. Andrae, J. *et al.* (2008) *Genes Dev.* **22**:1276.
2. Heldin, C-H. and B. Westermark (1999) *Physiol. Rev.* **79**:1283.
3. Do, M.S. *et al.* (1992) *Oncogene* **7**:1567.
4. Klinghoffer, R.A. *et al.* (2002) *Dev. Cell* **2**:103.
5. Martinho, O. (2009) *Br. J. Cancer* **101**:973.
6. Olson, L.E. and P. Soriano (2009) *Dev. Cell* **16**:303.
7. Baroni, S.S. *et al.* (2006) *N. Engl. J. Med.* **354**:2667.

## Description

<b>Source</b>	Murine myeloma cell line, NS0-derived		
	Mouse PDGF R $\alpha$ Leu25 - Glu524 (Asp65Glu, Gly439Ala, Thr440Ala) Accession # P26618.3 N-terminus	IEGRMD	Human IgG <sub>1</sub> (Pro100 - Lys330) C-terminus
<b>N-terminal Sequence Analysis</b>	Leu25		
<b>Structure / Form</b>	Disulfide-linked homodimer		
<b>Predicted Molecular Mass</b>	82.5 kDa (monomer)		

## Specifications

<b>SDS-PAGE</b>	125 - 140 kDa, reducing conditions
<b>Activity</b>	Measured by its ability to inhibit the biological activity of PDGF-AB or PDGF-AA using NR6R-3T3 mouse fibroblasts. Raines, E.W. <i>et al.</i> (1985) <i>Methods Enzymol.</i> <b>109</b> :749. The ED <sub>50</sub> for this effect is typically 0.01 - 0.04 $\mu$ g/mL in the presence of 10 ng/mL recombinant human PDGF-AA.
<b>Endotoxin Level</b>	<1.0 EU per 1 $\mu$ g of the protein by the LAL method.
<b>Purity</b>	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
<b>Formulation</b>	Lyophilized from a 0.2 $\mu$ m filtered solution in PBS. See Certificate of Analysis for details.

## Preparation and Storage

<b>Reconstitution</b>	Reconstitute at 100 $\mu$ g/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 3 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

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NOT FOR USE IN HUMANS