

## DESCRIPTION

<b>Species Reactivity</b>	Mouse
<b>Specificity</b>	Detects mouse PDGF R $\alpha$ in Western blots. In Western blots, less than 15% cross-reactivity with recombinant human PDGF R $\alpha$ is observed and less than 1% cross-reactivity with recombinant mouse PDGF R $\beta$ is observed.
<b>Source</b>	Polyclonal Goat IgG
<b>Purification</b>	Antigen Affinity-purified
<b>Immunogen</b>	Mouse myeloma cell line NS0-derived recombinant mouse PDGF R $\alpha$ Leu25-Glu524 (Asp65Glu, Gly439Ala, Thr440Ala) Accession # P26618
<b>Formulation</b>	Lyophilized from a 0.2 $\mu$ m filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

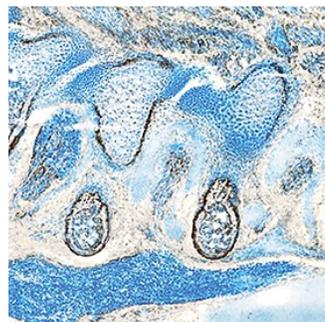
## APPLICATIONS

**Please Note:** Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Western Blot</b>	0.1 $\mu$ g/mL	Recombinant Mouse PDGF R $\alpha$ Fc Chimera (Catalog # 1062-PR)
<b>Immunohistochemistry</b>	5-15 $\mu$ g/mL	See Below

## DATA

### Immunohistochemistry



**PDGF R $\alpha$  in Mouse Embryo.** PDGF R $\alpha$  was detected in immersion fixed frozen sections of mouse embryo (13 d.p.c.) using Goat Anti-Mouse PDGF R $\alpha$  Biotinylated Antigen Affinity-purified Polyclonal Antibody (Catalog # BAF1062) at 15  $\mu$ g/mL overnight at 4 °C. Tissue was stained using the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS008) and counterstained with hematoxylin (blue). Specific staining was localized to developing cartilage. View our protocol for [Chromogenic IHC Staining of Frozen Tissue Sections](#).

## PREPARATION AND STORAGE

<b>Reconstitution</b>	Reconstitute at 0.2 mg/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>	<p><b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b></p> <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>• 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

## 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 hetero-dimerization of two receptors (PDGF R $\alpha$  and R $\beta$ ). Whereas  $\alpha/\alpha$  homo-dimerization is induced by PDGF-AA, -BB, -CC, and -AB,  $\alpha/\beta$  hetero-dimerization is induced by PDGF-AB, -BB, -CC, and -DD, and  $\beta/\beta$  homo-dimerization is induced only by PDGF-BB, and -DD (1-4). Both PDGF R $\alpha$  and R $\beta$  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. 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  $\alpha$  and  $\beta$  receptors. Others have only the  $\alpha$  receptors (oligodendrocyte progenitor cells, mesothelial cells, liver sinusoidal endothelial cells, astrocytes, platelets and megakaryocytes) or only the  $\beta$  receptors (myoblasts, capillary endothelial cells, pericytes, T cells, myeloid hematopoietic cells and macrophages) (1, 2). Recombinant mouse and human soluble PDGF R $\beta$  bind PDGF with high affinity and are potent PDGF antagonists.

### 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. LaRochells, W.J. *et al.* (2001) *Nature Cell Biol.* **3**:517.