

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse PDGF R α in direct ELISAs
Source	Monoclonal Rat IgG _{2B} Clone # 1039504
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived mouse PDGF R α Leu25-Glu524 Accession # P26618.3
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide
*Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.	

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.

Immunocytochemistry	Optimal dilution of this antibody should be experimentally determined.
Immunohistochemistry	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

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) (1-3). 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 mouse PDGF R α (amino acids 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 α shares 85%, 93%, 84%, 84%, and 81% amino acid sequence identity with human, 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 (1-3). 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, 4). 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) (5- 7).

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