Recombinant Mouse PDGF Rα Fc Chimera

Catalog Number: 1062-PR

DESCRIPTION

Source

Mouse myeloma cell line, NS0-derived mouse PDGF R alpha protein

N-terminal Sequence Analysis	Leu25
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	82.5 kDa (monomer)

SPECIFICATIONS

SDS-PAGE	125-140 kDa, reducing conditions
Activity	Measured by its ability to inhibit the biological activity of PDGF-AB or PDGF-AA using NR6R-3T3 mouse fibroblast cells. Raines, E.W. *et al.* (1985) Methods Enzymol. **109**:749. The ED_{50} for this effect is 0.01-0.04 µg/mL in the presence of 10 ng/mL recombinant human PDGF-AA.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in sterile 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.

BACKGROUND

PDGF Rα (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 Rα and PDGF Rβ 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 Rα primarily binds PDGF-AA and PDGF-CC, while PDGF Rβ primarily binds PDGF-BB and probably PDGF-DD. Like all class III RTKs, the extracellular domain (ECD) of mouse PDGF Rα (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 Rα shares 85%, 93%, 84%, 84%, and 81% amino acid sequence identity with human, rat, equine, canine and bovine PDGF Rα respectively. PDGF Rα 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 Rα 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 Rα 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 Rα in mice severely impairs mesenchymal derivatives in both embryo and extraembryonic tissues, and high or low PDGF Rα signaling in humans may result in spina bifida or cleft palate-type malformations. Postnatally, PDGF Rα is implicated in gliomas and fibrotic disorders of lung, heart and skin (scleroderma) (5- 7).

References:

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7. Baroni, S.S. *et al.* (2006) N. Engl. J. Med. **354**:2667.

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