Recombinant Human PLGF/PGF Protein (His Tag)

Catalog Number: PDEH100881

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

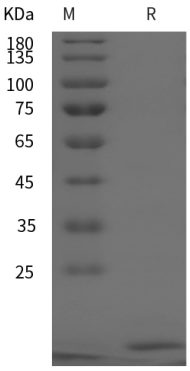
Description

Species	Human
Source	E.coli-derived Human PLGF protein Leu19-Arg149, with an C-terminal His
Calculated MW	14.3 kDa
Observed MW	16 kDa
Accession	P49763-2
Bio-activity	Not validated for activity

Properties

Purity	> 95% as determined by reducing SDS-PAGE.
Endotoxin	< 10 EU/mg of the protein as determined by the LAL method
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis.

Data



SDS-PAGE analysis of Human PLGF/PGF proteins, 2 μg/lane of Recombinant Human PLGF/PGF proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 16 kDa.

Background

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Placenta growth factor (PlGF) is a member of the PDGF/VEGF family of growth factors that share a conserved pattern of eight cysteines . Alternative splicing results in at least three human mature PlGF forms containing 131 (PlGF-1), 152 (PlGF-2), and 203 (PlGF-3) amino acids (aa) respectively . Only PlGF-2 contains a highly basic heparin-binding 21 aa insert at the C-terminus . Human PlGF-1 shares 56%, 55%, 74% and 95% aa identity with the comparable isoform of mouse, rat, canine, and equine PlGF, respectively. PlGF is mainly found as variably glycosylated, secreted, 55-60 kDa disulfide linked homodimers . Mammalian cells expressing PlGF include villous trophoblasts, decidual cells, erythroblasts, keratinocytes, and some endothelial cells . Circulating PlGF increases during pregnancy, reaching a peak in mid-gestation, this increase is attenuated in preeclampsia . However, deletion of PlGF in the mouse does not affect development or reproduction. Postnatally, mice lacking PlGF show impaired angiogenesis in response to ischemia . PlGF binds and signals through VEGF R1/Flt-1 but not VEGF R2/Flk-1/KDR, while VEGF binds both but signals only through the angiogenic receptor, VEGF R2. PlGF and VEGF therefore compete for binding to VEGF R1, allowing high PlGF to discourage VEGF/VEGF R1 binding and promote VEGF/VEGF R2-mediated angiogenesis . However, PlGF (especially PlGF-1) and some forms of VEGF can form dimers that decrease the angiogenic effect of VEGF on VEGF R2 . PlGF-2, but not PLGF-1, shows heparin-dependent binding of Neuropilin (Npn)-1 and Npn-2 . PlGF induces monocyte activation, migration, and production of inflammatory cytokines and VEGF. These activities facilitate wound, bone fracture, and cardiac repair, but also contribute to inflammation in active sickle cell disease and atherosclerosis . PlGF can also inhibit TIMP3 expression in the spleen, leading to immune triggering of hypertension .

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