Recombinant Human PlGF

Catalog Number: 264-PGB

DESCRIPTION

Source	*E. coli*-derived Ala21-Arg149 Accession # P49763-2
N-terminal Sequence Analysis	Ala21
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	15 kDa

SPECIFICATIONS

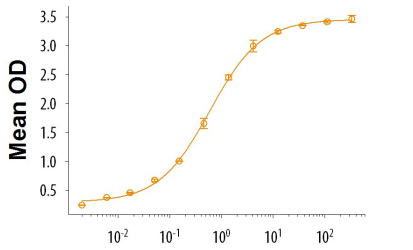
SDS-PAGE	12-15 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human VEGF R1/Flt-1 Fc Chimera (Catalog # 3516-FL) is immobilized at 0.5 µg/mL, 100 µL/well, the concentration of Recombinant Human PlGF that produces 50% of the optimal binding response is approximately 0.15-0.9 ng/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in Acetonitrile and TFA with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 200 µg/mL in 4 mM HCl.
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.

DATA

Bioactivity



When Recombinant Human VEGF R1/Flt-1 Fc Chimera, aa 27-328 (Catalog # 3516-FL) is coated at 0.5 µg/mL, Recombinant Human PlGF (Catalog # 264-PGB) binds with a typical ED_{50} of 0.15-0.9 ng/mL.

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BACKGROUND

Placenta growth factor (PlGF) is a member of the PDGF/VEGF family of growth factors that share a conserved pattern of eight cysteines (1, 2). 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 (1, 2). Only PlGF-2 contains a highly basic heparin-binding 21 aa insert at the C-terminus (1). 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 (3). Mammalian cells expressing PlGF include villous trophoblasts, decidual cells, erythroblasts, keratinocytes, and some endothelial cells (1, 4-6). Circulating PlGF increases during pregnancy, reaching a peak in mid-gestation; this increase is attenuated in preeclampsia (7). However, deletion of PlGF in the mouse does not affect development or reproduction. Postnatally, mice lacking PlGF show impaired angiogenesis in response to ischemia (8). 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 (1, 4, 8, 9). However, PlGF (especially PlGF-1) and some forms of VEGF can form dimers that decrease the angiogenic effect of VEGF on VEGF R2 (3, 4). PlGF-2, but not PLGF-1, shows heparin-dependent binding of Neuropilin (Npn)-1 and Npn-2 (10, 11). 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 (5, 6, 8, 12-15). PlGF can also inhibit TIMP3 expression in the spleen, leading to immune triggering of hypertension (16).

References:

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