Recombinant Human PlGF-4

Catalog Number: 8987-PL/CF

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived Leu19-Arg242 Accession # P49763-4
N-terminal Sequence Analysis	Leu19
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	25 kDa

SPECIFICATIONS

SDS-PAGE	35-40 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human VEGF R1/Flt-1 Fc Chimera, aa 27-328 (Catalog # 3516-FL) is immobilized at 0.5 µg/mL, 100 µL/well, the concentration of Recombinant Human PlGF-4 that produces 50% of the optimal binding response is approximately 4-24 ng/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>85%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in HCl. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 250 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.

BACKGROUND

Placenta growth factor (PlGF or PGF) is an approximately 55-60 kDa member of the PDGF/VEGF family of secreted growth factors that share a conserved pattern of eight cysteines (1). Alternative splicing generates multiple human PlGF isoforms containing 131 (PlGF-1), 152 (PlGF-2), 203 (PlGF-3), or 224 (PlGF-4) amino acids (aa) (2, 3). Mature human PlGF shares 66% and 63% aa sequence identity with comparable regions of mouse and rat PlGF, respectively. PlGF is expressed as a variably glycosylated disulfide linked homodimer by villous trophoblasts and decidual cells, with smaller amounts in erythroblasts, keratinocytes and some endothelial cells (3-6). Circulating PlGF increases during pregnancy, reaching a peak in mid-gestation; this increase is attenuated in preeclampsia (7). Postnatally, mice lacking PlGF show impaired angiogenesis in response to ischemia (8). PlGF binds and signals through VEGF R1/Flt-1 and Neuropilins (some isoforms), but not VEGF R2/Flk-1/KDR (8-10). In contrast, VEGF binds both VEGF R1 and R2, but signals mainly through the angiogenic receptor, VEGF R2. PlGF and VEGF therefore compete for binding to VEGF R1, resulting in a PlGF inhibition of VEGF/VEGF R1 binding coupled to a subsequent promotion of VEGF/VEGF R2-mediated angiogenesis (8, 9). However, PlGF (especially PlGF-1) and some forms of VEGF can form heterodimers that alter the angiogenic effect of VEGF on VEGF R2 (4, 9). PlGF induces monocyte activation, migration, and production of inflammatory cytokines and VEGF (1). These activities facilitate wound and bone fracture healing and also contribute to inflammation in active sickle cell disease and atherosclerosis (1, 5, 6, 8, 11-13).

References:

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