

## Recombinant Human PlGF

### Information

Accession #	Q07326
Alternate Names	PlGF; PlGF-2; PLGFplacental growth factor-like; PGFL; placenta growth factor; placental growth factor
Source	Human embryonic kidney cell, HEK293-derived human PlGF protein
Protein sequence	Ala21-Arg149
M.Wt	14.5 kDa
Appearance	Solution protein
Stability & Storage	Avoid repeated freeze-thaw cycles. It is recommended that the protein be aliquoted for optimal storage.- 3 years from date of receipt, -20 to -7°C as supplied.
Concentration	0.2 mg/mL
Formulation	Dissolved in sterile PBS buffer.
Reconstitution	We recommend that this vial be briefly centrifuged prior to opening to bring the contents to the bottom. This solution can be diluted into other aqueous buffers.
Biological Activity	When Recombinant Human VEGF R1/Flt-1 Fc Chimera is immobilized at 0.5 $\mu$ g/mL, 100 $\mu$ L/well, the concentration of Recombinant Human PlGF that produces 50% of the optimal binding response is approximately 0.05-1 ng/mL. Measured by its binding ability in a functional ELISA.
Shipping Condition	Shipping with dry ice.
Handling	Centrifuge the vial prior to opening.
Usage	For Research Use Only! Not to be used in humans.

### Quality Control

Purity	> 95%, determined by SDS-PAGE.
Endotoxin	<0.010 EU per 1 $\mu$ g of the protein by the LAL method.

### Description

Placenta growth factor (PlGF) is a member of the PDGF/VEGF family of growth factors that share a conserved pattern of eight cysteines<sup>[1, 2]</sup>. 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<sup>[1, 2]</sup>. Only PlGF-2 contains a highly basic heparin-binding 21 aa insert at the C-terminus<sup>[1]</sup>. 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<sup>[3]</sup>. Mammalian cells expressing PlGF include villous trophoblasts, decidual cells, erythroblasts, keratinocytes, and some endothelial cells<sup>[1, 4-6]</sup>. 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].

## Reference

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