

**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** 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 PIGF 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. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

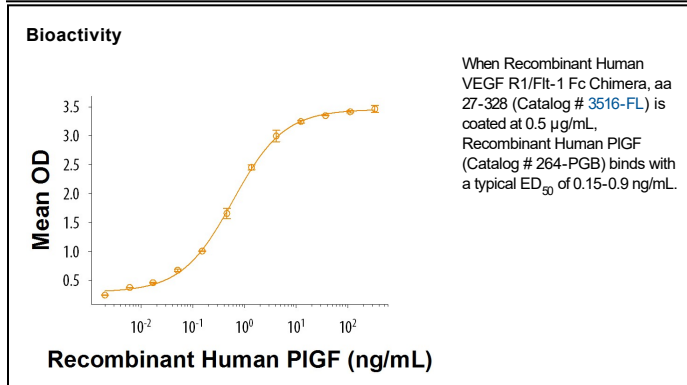
**Reconstitution** Reconstitute at 200 µg/mL in 4 mM HCl.

**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**



**BACKGROUND**

Placenta growth factor (PIGF) 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 PIGF forms containing 131 (PIGF-1), 152 (PIGF-2), and 203 (PIGF-3) amino acids (aa) respectively (1, 2). Only PIGF-2 contains a highly basic heparin-binding 21 aa insert at the C-terminus (1). Human PIGF-1 shares 56%, 55%, 74% and 95% aa identity with the comparable isoform of mouse, rat, canine, and equine PIGF, respectively. PIGF is mainly found as variably glycosylated, secreted, 55-60 kDa disulfide linked homodimers (3). Mammalian cells expressing PIGF include villous trophoblasts, decidual cells, erythroblasts, keratinocytes, and some endothelial cells (1, 4-6). Circulating PIGF increases during pregnancy, reaching a peak in mid-gestation; this increase is attenuated in preeclampsia (7). However, deletion of PIGF in the mouse does not affect development or reproduction. Postnatally, mice lacking PIGF show impaired angiogenesis in response to ischemia (8). PIGF 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. PIGF and VEGF therefore compete for binding to VEGF R1, allowing high PIGF to discourage VEGF/VEGF R1 binding and promote VEGF/VEGF R2-mediated angiogenesis (1, 4, 8, 9). However, PIGF (especially PIGF-1) and some forms of VEGF can form dimers that decrease the angiogenic effect of VEGF on VEGF R2 (3, 4). PIGF-2, but not PLGF-1, shows heparin-dependent binding of Neuropilin (Npn)-1 and Npn-2 (10, 11). PIGF 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). PIGF can also inhibit TIMP3 expression in the spleen, leading to immune triggering of hypertension (16).

**References:**

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