

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human PIGF-3 in direct ELISAs.
Source	Monoclonal Mouse IgG _{2B} Clone # 1039018
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>Spodoptera frugiperda</i> Sf21 (baculovirus)-derived human PIGF-3 Leu19-Arg221 Accession # P49763
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

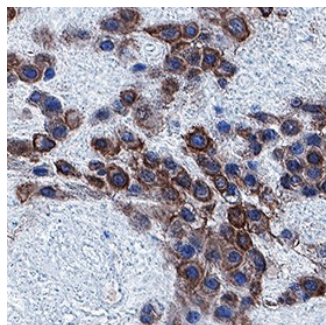
APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

	Recommended Concentration	Sample
Immunohistochemistry	5-25 µg/mL	Immersion fixed paraffin-embedded sections of human placenta

DATA

Immunohistochemistry



PIGF-3 in Human Placenta. PIGF-3 was detected in immersion fixed paraffin-embedded sections of human placenta using Mouse Anti-Human PIGF-3 Monoclonal Antibody (Catalog # MAB7758) at 5 µg/mL for 1 hour at room temperature followed by incubation with the Anti-Mouse IgG VisUCyte™ HRP Polymer Antibody (Catalog # VC001). Before incubation with the primary antibody, tissue was subjected to heat-induced epitope retrieval using Antigen Retrieval Reagent-Basic (Catalog # CTS013). Tissue was stained using DAB (brown) and counterstained with hematoxylin (blue). Specific staining was localized to decidual cells. Staining was performed using our protocol for IHC Staining with VisUCyte HRP Polymer Detection Reagents.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
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. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Placenta growth factor (PIGF or PGF) is a member of the PDGF/VEGF family of growth factors that share a conserved pattern of eight cysteines (1-3). Alternative splicing likely results in four human mature PIGF forms containing 131 (PIGF-1), 152 (PIGF-2), 203 (PIGF-3), or 224 (PIGF-4) amino acids (aa) (1-3). The PIGF-3 form is limited to humans. PIGF-3 and PIGF-1 do not contain a heparin binding insert at the C-terminus (1, 2). Within the region shared with other PIGF isoforms (aa 18-131), human PIGF-3 shares 68%, 66%, 96%, 96%, 87% and 77% aa sequence identity with mouse, rat, porcine, equine, canine and bovine PIGF, respectively. PIGF is mainly found as a variably glycosylated, secreted, 55 - 60 kDa, disulfide linked homodimer (1, 4). Mammalian cells expressing all forms of PIGF include villous trophoblasts and decidual cells, with smaller amounts in erythroblasts, keratinocytes and some endothelial cells (1-3, 5, 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, which expresses only PIGF-2, does not affect development or reproduction (3, 8). Postnatally, mice lacking PIGF show impaired angiogenesis in response to ischemia (8). PIGF binds and signals through VEGF R1/Flt-1 and Neuropilins (some isoforms), but not VEGF R2/Flk-1/KDR (3, 8-10). In contrast, VEGF binds both VEGF R1 and R2, but signals mainly through the angiogenic receptor, VEGF R2. PIGF and VEGF therefore compete for binding to VEGF R1, resulting in a PIGF inhibition of VEGF/VEGF R1 binding coupled to a subsequent promotion of VEGF/VEGF R2-mediated angiogenesis (1, 3, 8, 9). However, PIGF (especially PIGF-1) and some forms of VEGF can form dimers that can alter the angiogenic effect of VEGF on VEGF R2 (3, 4, 9). PIGF induces monocyte activation, migration, and production of inflammatory cytokines and VEGF (3). These activities facilitate wound and bone fracture healing, and also contribute to inflammation in active sickle cell disease and atherosclerosis (5, 6, 8, 11-14). Circulating PIGF often correlates with tumor stage and aggressiveness (3, 14, 15).

References:

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