Human PIGF Alexa Fluor® 405-conjugated Antibody

Monoclonal Mouse IgG_{2B} Clone # 1038864

Catalog Number: FAB2643V

100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human PIGF in direct ELISAs.
Source	Monoclonal Mouse IgG_{2B} Clone # 1038864
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	*E. coli*-derived human PlGF Ala21-Arg149 Accession # P49763-2
Conjugate	Alexa Fluor 405 Excitation Wavelength: 405 nm Emission Wavelength: 421 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

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.*

Immunocytochemistry	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

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).

PRODUCT SPECIFIC NOTICES

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