Human PIGF Alexa Fluor® 594-conjugated Antibody

Monoclonal Mouse IgG_{2A} Clone # 1038928

Catalog Number: FAB11199T

100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human PIGF-1,2,3 and 4 in direct ELISAs.
Source	Monoclonal Mouse IgG_{2A} Clone # 1038928
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	CHO-derived human PIGF-2 protein Leu19-Arg170 Accession # NP_002623
Conjugate	Alexa Fluor 594 Excitation Wavelength: 590 nm Emission Wavelength: 617 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.*

ELISA	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 (PIGF) is a member of the PDGF/VEGF family of growth factors that share a conserved pattern of eight cysteines (1 - 3). Alternate 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 - 3). Only PIGF-2 contains a highly basic heparin-binding 21 aa insert at the C-terminus (1). In the mouse, only one PIGF that is the equivalent of human PIGF-2 has been identified (3). Human PIGF-2 shares 60%, 56%, 82%, 95% and 95% aa identity with mouse, rat, canine, equine and porcine PIGF-2. PIGF is mainly found as a variably glycosylated, secreted, 55 - 60 kDa disulfide linked homodimer (4). Mammalian cells expressing PIGF include villous trophoblasts, decidual cells, erythroblasts, keratinocytes and some endothelial cells (1, 5 - 7). Circulating PIGF increases during pregnancy, reaching a peak in mid-gestation; this increase is attenuated in preeclampsia (8). However, deletion of PIGF in the mouse does not affect development or reproduction. Postnatally, mice lacking PIGF show impaired angiogenesis in response to ischemia (9). 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, resulting in a PIGF inhibition of VEGF/VEGF R1 binding coupled to a subsequent promotion of VEGF/VEGF R2-mediated angiogenesis (1, 5, 9, 10). However, PIGF (especially PIGF-1) and some forms of VEGF can form dimers that decrease the angiogenic effect of VEGF on VEGF R2 (4, 5). PIGF-2, like VEGF164/165, shows heparin-dependent binding of neuropilin (Npn)-1 and Npn-2, and can inhibit nerve growth cone collapse (11, 12). PIGF induces monocyte activation, migration, and production of inflammatory cytokines and VEGF. These activities facilitate wound and bone fracture healing, and also contribute to inflammation in active sickle cell disease and atherosclerosis (6, 7, 9, 13 - 16). Circulating PIGF often correlates with tumor stage and aggressiveness, and therapeutic PIGF-2 antibodies are being investigated for their ability to inhibit tumor growth and angiogenesis (5, 13).

PRODUCT SPECIFIC NOTICES

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