Epoto Biotech 南京艾璞拓生物科技有限公司

Recombinant Human PIGF, Tag Free

Catalog Number: HF-2047

General Information		
Synonyms	PIGF; PIGF-2; PLGFplacental growth factor-like; PGFL; placenta growth factor; placental growth factor	
Accession #	Q07326	
Source	Human embryonic kidney cell, HEK293-derived human PIGF protein	
	Ala21-Arg149	
Predicted Moleucular we	eight 14.5 kDa	
Components and St	orage	
Formulation	Solution protein.	
	Dissolved in sterile PBS buffer .	
	This solution can be diluted into other aqueous buffers. Centrifuge the vial prior to opening.	
Storage and Stability	orage and Stability Avoid repeated freeze-thaw cycles. It is recommended that the protein be aliquoted for optimal storage.	
	12 months from date of receipt, -20 to -70 °C as supplied.	
Shipping	Shipping with dry ice.	
Quality		
Purity	> 95%, determined by SDS-PAGE.	
Endotoxin Level	<0.010 EU per 1 ug of the protein by the LAL method.	
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human VEGF R1/FIt-1 Fc Chimera is	
	immobilized at 0.5 μ g/mL, 100 μ L/well, the concentration of Recombinant Human PIGF that produces 50% of the	
	optimal binding response is approximately 0.05-1 ng/mL.	

SDS-PAGE

Bioactivity



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/FIt–1 but not VEGF R2/FIk–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).

Reference

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