Recombinant Human ANGPTL4 Protein(hIgG1 Fc Tag)

Catalog Number: PDMH100444

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

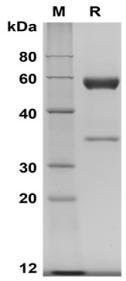
Description

Species	Human
Source	Mammalian-derived Human ANGPTL4 protein Arg164-Ser406, with an C-terminal hIgG1 Fc
Calculated MW	51.6 kDa
Observed MW	58 kDa
Accession	Q9BY76
Bio-activity	Not validated for activity

Properties

Purity	> 90% as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU/mg of the protein as determined by the LAL method
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis.

Data



SDS-PAGE analysis of Human ANGPTL4 proteins, 2μg/lane
of Recombinant Human ANGPTL4 proteins was resolved
with SDS-PAGE under reducing conditions, showing bands
at 58 kDa

Background

ANGPTL4, also known as ANGPTL2, is a protein with hypoxia-induced expression in endothelial cells. It contains 1 fibrinogen C-terminal domain and is expressed at high levels in the placenta, heart, liver, muscle, pancreas and lung but expressed poorly in the brain and kidney. ANGPTL4 inhibits proliferation, migration, and tubule formation of endothelial cells and reduces vascular leakage. It may act as a regulator of angiogenesis and modulate tumorigenesis. It inhibits proliferation, migration, and tubule formation of endothelial cells and reduces vascular leakage. It may also exert a protective function on endothelial cells through an endocrine action. ANGPTL4 is directly involved in regulating glucose homeostasis, lipid metabolism, and insulin sensitivity. In response to hypoxia, the unprocessed form of the protein accumulates in the subendothelial extracellular matrix (ECM). The matrix-associated and immobilized unprocessed form limits the formation of actin stress fibers and focal contacts in the adhering endothelial cells and inhibits their adhesion. It also decreases motility of endothelial cells and inhibits the sprouting and tube formation.

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