Recombinant Human Angiopoietin-like Protein 3/ANGPTL3 aa 17-220

Catalog Number: 4954-AN

DESCRIPTION

Source	Chinese Hamster Ovary cell line, CHO-derived human Angiopoietin-like Protein 3/ANGPTL3 protein Ser17-Pro220, with a C-terminal 6-His tag Accession # Q9Y5C1
N-terminal Sequence Analysis	Ser17
Structure / Form	Oligomer
Predicted Molecular Mass	24.6 kDa

SPECIFICATIONS

SDS-PAGE	35-43 kDa, reducing conditions
Activity	Measured by its ability to inhibit lipoprotein lipase activity. Yoshida, K. *et al.* (2002) J. Lipid Res. **43**:1770. The IC_{50} value under conditions in which Recombinant Human Lipoprotein Lipase/LPL (Catalog # 9888-LL) and p-nitrophenyl butyrate are present in 0.1 M Sodium Phosphate, 0.15 M NaCl, 0.5% Triton® X-100, pH 7.2, is approximately <15 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS and NaCl. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 200 µg/mL in sterile PBS.
Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
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. • 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Angiopoietin-like 3 (ANGPTL3) is a secreted glycoprotein that is structurally related to the angiopoietins (1-3). Mature human ANGPTL3 contains an N-terminal coiled coil domain and a C-terminal fibrinogen-like domain (4). Within the N-terminal region, human ANGPTL3 shares 15%-21% amino acid (aa) sequence identity with ANGPTL1, 2, 4, 5, 6, and 7, and 82% with mouse and rat ANGPTL3. ANGPTL3 is expressed in the liver from early in development through adulthood (4, 5). Full length ANGPTL3 circulates in the plasma as do the proteolytically separated N- and C-terminal segments containing the coiled coil domain and fibrinogen-like domains, respectively (6, 7). ANGPTL3 is found as 70 kDa, 50 kDa, and 32 kDa species and can form weakly-associated, noncovalent multimers *in vitro* (5, 6). ANGPTL3 directly inhibits lipoprotein lipase (LPL) and endothelial lipase (EL), enzymes responsible for hydrolyzing circulating triglycerides and HDL phospholipids (8, 9). This activity requires a putative heparin-binding motif which is N-terminal to the coiled coil domain (6). Proteolytic removal of the fibrinogen-like domain from the N-terminal fragment serves to activate ANGPTL3 and increase its ability to inhibit LPL *in vitro* and function *in vivo* (6). ANGPTL3 promotes an increase in circulating triglyceride levels without altering VLDL or HDL secretion or uptake (6-8). ANGPTL3 knockout mice are hypolipidemic and have elevated LPL activity (10). ANGPTL3 expression *in vivo* is up-regulated by LXR agonists and down-regulated by insulin, leptin, and agonists of TRβ or PPARβ (11-14). Dysregulated ANGPTL3 expression and elevated plasma triglyceride levels are characteristic of some strains of obese and diabetic mice (7, 8, 12). ANGPTL3 does not bind Tie1 or Tie2, but its fibrinogen-like domain interacts with integrin αVβ3 to induce endothelial cell adhesion, migration, and neovascularization (15). ANGPTL3, secreted by fetal liver cells, also promotes the expansion of hematopoietic stem cells (16).

References:

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