

DESCRIPTION

Source Chinese Hamster Ovary cell line, CHO-derived
Ser17-Thr206, with a C-terminal 6-His tag
Accession # Q9R182

N-terminal Sequence Analysis Ser17

Predicted Molecular Mass 23 kDa

SPECIFICATIONS

SDS-PAGE 23-37 kDa, reducing conditions

Activity Measured by its ability to promote the expansion of E16 rat liver mononuclear cells *in vitro*, in the presence of Recombinant Mouse SCF/c-kit Ligand (Catalog # 455-MC), Recombinant Mouse Thrombopoietin/Tpo (Catalog # 488-TO), and Recombinant Mouse Flt-3 Ligand (Catalog # 427-FL).
The ED₅₀ for this effect is typically 20-100 ng/mL in the presence of a cross-linking antibody, Mouse Anti-polyHistidine Monoclonal Antibody (Catalog # MAB050).

Endotoxin Level <0.10 EU per 1 µg of the protein by the LAL method.

Purity >95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 µm filtered solution in MOPS,NaCl and CHAPS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

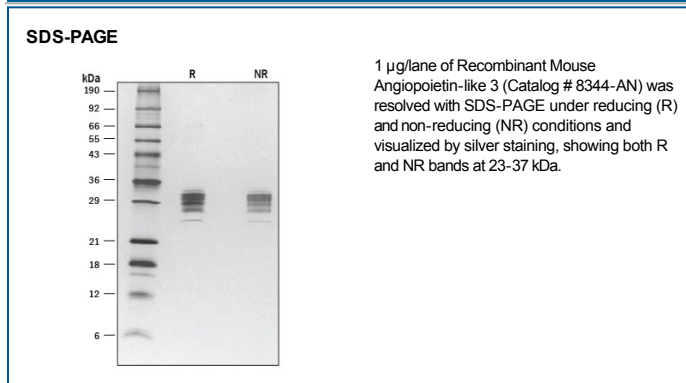
Reconstitution Reconstitute at 100 µg/mL in PBS.

Shipping The product is shipped at ambient temperature. 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.

DATA



BACKGROUND

Angiopoietin-like Protein 3 (ANGPTL3) is a secreted glycoprotein that is structurally related to the angiopoietins (1). Mature mouse ANGPTL3 contains an N-terminal coiled coil domain and a C-terminal fibrinogen-like domain (2). Within the N-terminal fragment, mouse ANGPTL3 shares 83% and 92% aa sequence identity with human and rat ANGPTL3, respectively. ANGPTL3 is expressed in the liver from early in development through adulthood (2, 3). Full length ANGPTL3 circulates in the plasma as do the proteolytically separated N- and C-terminal fragments containing the coiled coil domain and fibrinogen-like domains, respectively (4, 5). The cleavage of ANGPTL3 by Furin and Proprotein Convertase 5/6 is enhanced by its interaction with the related ANGPTL8 (6, 7). ANGPTL3 is found as 70 kDa, 50 kDa, and 32 kDa species and can form weakly associated noncovalent multimers *in vitro* (3, 4). 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 (4). 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* [Ono 41804]. ANGPTL3 promotes an increase in circulating triglyceride levels without altering VLDL or HDL secretion or uptake (4, 5, 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 TR β or PPAR β (11-14). Dysregulated ANGPTL3 expression and elevated plasma triglyceride levels are characteristic of some strains of obese and diabetic mice (5, 8, 12). ANGPTL3 does not bind Tie1 or Tie2, but its fibrinogen-like domain interacts with Integrin α V β 3 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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