

## DESCRIPTION

**Source** *Trichoplusia ni*, *T. ni* (baculovirus)-derived cynomolgus monkey Angiotensin-like Protein 3/ANGPTL3 protein  
Ser17-Glu460, with a C-terminal 10-His tag  
Accession # XP\_005543242

**N-terminal Sequence Analysis** Ser17

**Predicted Molecular Mass** 53 kDa

## SPECIFICATIONS

**SDS-PAGE** 56-65 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<sub>50</sub> for this effect is 1.5-9 µg/mL.

**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 PBS, NaCl and CHAPS with Trehalose. See Certificate of Analysis for details.

## PREPARATION AND STORAGE

**Reconstitution** Reconstitute at 250 µg/mL in water.

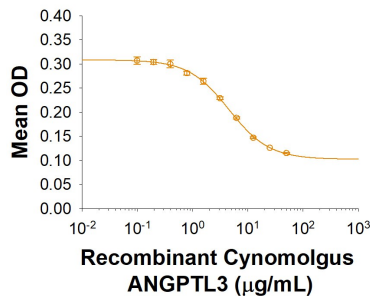
**Shipping** The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

**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 °C under sterile conditions after reconstitution.

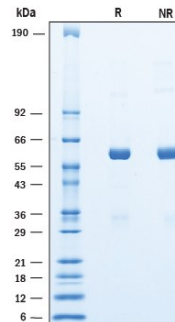
## DATA

### Bioactivity



Recombinant Cynomolgus Monkey Angiotensin-like Protein 3/ANGPTL3 (Catalog # 10052-AN) dose dependently inhibits Recombinant Human LPL (Catalog # 9888-LL) activity with an IC<sub>50</sub> of 1.5-9 µg/mL.

### SDS-PAGE



2 µg/lane of Recombinant Cynomolgus Monkey Angiotensin-like Protein 3/ANGPTL3 was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 56-65 kDa.

## BACKGROUND

ANGPTL3 is a secreted glycoprotein that is structurally related to the Angiotensins (1-3). Mature ANGPTL3 contains an N-terminal coiled-coil domain and a C-terminal fibrinogen-like domain (4). ANGPTL3 is expressed in the liver from early in development through adulthood (4, 5). Full length ANGPTL3 circulates in the plasma as do proteolytically separated N- and C-terminal fragments containing the coiled-coil domain and fibrinogen-like domains, respectively (6, 7). 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 TR beta or PPAR beta (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  $\alpha V \beta 3$  to induce endothelial cell adhesion, migration, and neovascularization (15). ANGPTL3, secreted by fetal liver, also promotes the expansion of hematopoietic stem cells (16). Mature Cynomolgus Monkey ANGPTL3 shares 97%, 77%, and 78% amino acid sequence identity with human, mouse, and rat ANGPTL3, respectively.

## References:

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