

DESCRIPTION

Source Mouse myeloma cell line, NS0-derived
Arg26-Pro281, with a C-terminal 6-His tag
Accession # Q9BXJ1

N-terminal Sequence Analysis Arg26

Predicted Molecular Mass 30 kDa

SPECIFICATIONS

SDS-PAGE 33-41 kDa, reducing conditions

Activity Measured by its binding ability in a functional ELISA.
When Recombinant Human BAI3 is coated at 4 µg/mL, Recombinant Human C1qTNF1 (Catalog # 9268-TN) binds with an ED₅₀ = 0.6-3.6 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 HEPES and NaCl. See Certificate of Analysis for details.

PREPARATION AND STORAGE

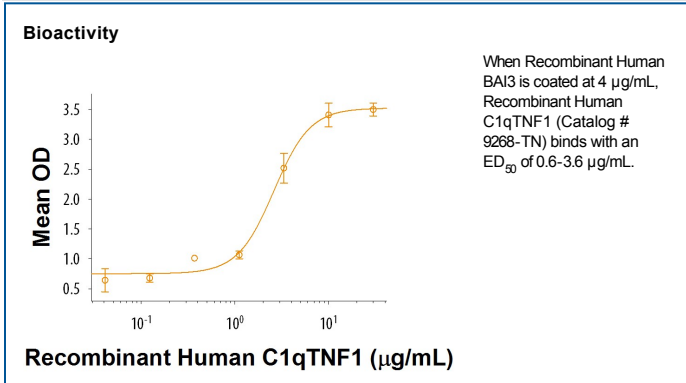
Reconstitution Reconstitute at 250 µg/mL in PBS.

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

C1qTNF1 (CTRP1) is an approximately 35 kDa member of the C1q family of secreted proteins and plays a role in energy metabolism and inflammation (1, 2). C1qTNF1 contains a collagen-like region and one C1q-like domain (3). Mature human C1qTNF1 shares 80% aa sequence identity with mouse and rat C1qTNF1. Circulating levels of C1qTNF1 are elevated in obesity, hypertension, and diabetes but can be decreased in the serum of diet-induced obese mice (4-6). C1qTNF1 expression is up-regulated in atherosclerotic plaques or adipose tissue by oxidized LDL or inflammatory cytokines (3, 7, 8). In turn, it induces the expression of inflammatory cytokines (7, 9) and the up-regulation of adhesion proteins on vascular endothelial cells (8). Systemically administered C1qTNF1, in contrast, can limit tissue damage following myocardial infarction (9). In skeletal muscle, C1qTNF1 promotes fatty acid oxidation, energy expenditure, insulin sensitivity, and glucose uptake and glycolysis (6, 10). It also induces the proliferation of immature chondrocytes (11) and aldosterone synthesis in the adrenal cortex (4). R&D Systems in-house testing indicates that C1qTNF1 binds to BAI3, consistent with the reported interactions between BAI3 and C1qL proteins (12).

References:

1. Grebrehwet, B. *et al.* (2012) *Front. Immunol.* **5**:3.
2. Seldin, M.M. *et al.* (2014) *Rev. Endocr. Metab. Disord.* **15**:111.
3. Kim, K.-Y. *et al.* (2006) *FEBS Lett.* **580**:3953.
4. Jeon, J.H. *et al.* (2008) *FASEB J.* **22**:1502.
5. Xin, Y. *et al.* (2014) *Endocr. J.* **61**:841.
6. Peterson, J. M. *et al.* (2012) *J. Biol. Chem.* **287**:1576.
7. Wang, X.Q. *et al.* (2016) *Atherosclerosis* **250**:38.
8. Lu, L. *et al.* (2016) *Eur. Heart J.* **37**:1762.
9. Yuasa, D. *et al.* (2016) *FASEB J.* **30**:1065.
10. Han, S. *et al.* (2016) *J. Nutr. Biochem.* **27**:43.
11. Akiyama, H. *et al.* (2013) *Mol. Cell. Endocrinol.* **369**:63.
12. Bolliger, M.F. *et al.* (2011) *Proc. Natl. Acad. Sci. USA* **108**:2534.