

DESCRIPTION

Source Mouse myeloma cell line, NS0-derived
Asn16-Pro333, with an N-terminal 6-His tag
Accession # AAH40438

N-terminal Sequence Analysis His

Predicted Molecular Mass 34 kDa

SPECIFICATIONS

SDS-PAGE 36-40 kDa, reducing conditions

Activity Measured by its ability to induce CXCL10 secretion by RAW 264.7 mouse monocyte/macrophage cells.
The ED₅₀ for this effect is 1.5-7.5 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

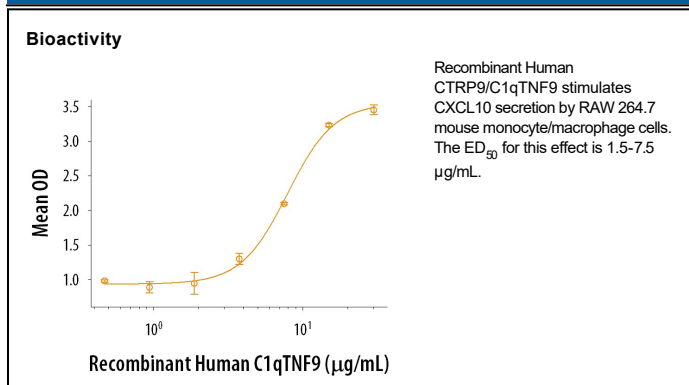
Reconstitution Reconstitute at 250 µg/mL in sterile PBS.

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

C1qTNF9, also known as CTRP9, is an approximately 40 kDa member of the C1q and TNF-related protein family (1). Like all members of this protein family, C1qTNF9 consists of a short variable region, a collagenous domain that can be hydroxylated, and a C1q-like globular domain (1). Human C1qTNF9 shares 85% amino acid sequence identity with the mouse and rat orthologs. Both the mouse and human C1qTNF9 proteins are expressed in adipose tissue, but the mouse protein has also been detected in the heart, lung, muscle, kidney, testis, lymph node, smooth muscle, prostate, thymus, and uterus (1, 2). They have both also been shown to be secreted as trimers and higher order multimers and also to form hetero-oligomers with Adiponectin (1, 2). Mouse C1qTNF9 can stimulate the phosphorylation of AMPK, Akt, and eNOS (1, 3, 4). Also in mice, C1qTNF9 may have an important role in cardiac and metabolic health. Its expression has a cardioprotective effect following acute myocardial infarction that may be dependent on AMPK activation (4-6). Additionally, transgenic mice overexpressing C1qTNF9 are resistant to high fat diet-induced obesity (7). This metabolic role may be conserved, since C1qTNF9 serum levels have been shown to inversely correlate with metabolic syndrome in humans (8).

References:

1. Wong, G.W. *et al.* (2009) *FASEB J.* **23**:241.
2. Peterson, J.M. *et al.* (2009) *Biochem. Biophys. Res. Commun.* **388**:360.
3. Zheng, Q. *et al.* (2011) *Arterioscler. Thromb. Vasc. Biol.* **31**:2616.
4. Kambara, T. *et al.* (2012) *J. Biol. Chem.* **287**:18965.
5. Su, H. *et al.* (2013) *Basic Res. Cardiol.* **108**:315.
6. Sun, Y. *et al.* (2013) *Circulation* **128**:S113.
7. Peterson, J.M. *et al.* (2013) *Am. J. Physiol. Regul. Integr. Comp. Physiol.* **305**:R522.
8. Hwang, Y.C. *et al.* (2013) *Int. J. Obes. (Lond)* [Epub ahead of print].