Recombinant Human CTRP5/C1qTNF5

Catalog Number: 9510-TN

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived human CTRP5/C1qTNF5 protein Ser16-Ala243, with Gln44Arg substitution and a C-terminal 6-His tag Accession # Q9BXJ0
N-terminal Sequence Analysis	Ser16
Predicted Molecular Mass	25 kDa

SPECIFICATIONS

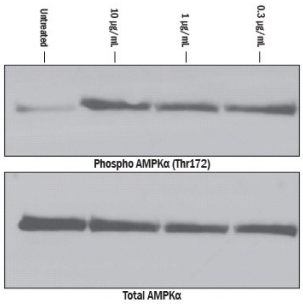
SDS-PAGE	26-32 kDa, reducing conditions
Activity	Measured by its ability to induce phospho AMPK activation in C2C12 mouse differentiated myocytes. 300 ng/mL of Recombinant Human CTRP5/C1qTNF5 induces phosphorylation of AMPK. Measured by its binding ability in a functional ELISA. When Recombinant Human MFRP (Catalog # 1915-MF) is immobilized at 2 µg/mL, 100 µL/well, the concentration of Recombinant Human CTRP5/C1qTNF5 that produces 50% of the optimal binding response is 0.3-1.8 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>85%, 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 200 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

Bioactivity



Recombinant Human CTRP5/C1qTNF5 Induces Phosphorylation of AMPK in C2C12 Differentiated Myocytes. C2C12 differentiated myocytes were treated with Recombinant Human CTRP5/C1qTNF5 (Catalog # 9510-TN) for 1 hour prior to Western blot analysis

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BACKGROUND

CTRP5, also known as C1qTNF5, belongs to the highly conserved family of Acrp30/Adiponectin paralogs known as C1q and TNF-related protein family (1). All family members share a modular organization comprising an N-terminal signal peptide, a short variable region with conserved cysteine residues, a collagenous domain for coiled coil structure, and a C-terminal globular domain (2, 3). CTRP proteins are predicted to have trimeric structures that can assemble into higher order molecular forms (1). Human and mouse CTRP5 share 94% amino acid sequence identity. CTRP5 is highly expressed in the eye, testis and adipose tissue (4). A mutation (S163R) in C1qTNF5 impairs secretion and is associated with early-onset long anterior zonules (LAZ) and late-onset retinal degeneration (L-ORD) (5). In both mouse and human, the 3' untranslated region of the MFRP transcript contains the complete open reading frame of C1qTNF5, suggesting that these genes are dicistronic. MFRP and C1qTNF5 have been shown to directly interact in the retinal pigment epithelium and are likely functionally related (6). Like other C1qTNF family members, C1qTNF5 shares similarities to adiponectin in structure and function and has been shown to stimulate glucose uptake and increase fatty acid oxidation through activation of AMPK in skeletal muscle (3). Conversely, administration of rhC1qTNF5 was shown to attenuate insulin-induced Akt activation in adipocytes and skeletal muscle (7).

References:

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