

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human CTRP3/C1qTNF3/CORS26 in direct ELISAs.
Source	Monoclonal Mouse IgG ₁ Clone # 860115
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived human CTRP3/C1qTNF3/CORS26 Gln23-Lys246 Accession # Q9BXJ4-1
Conjugate	Alexa Fluor 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

ELISA Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

CTRP3 (Complement C1q TNF-related Protein 3/C1qTNF3) also known as CORS-26 (Collagenous Repeat-containing Sequence of 26 kDa protein), Cartnnectin and Cartducin, is a 30-32 kDa, secreted member of the C1q and TNF-related (CTRP) superfamily of molecules (1). The mature protein is 224 aa in length. It contains an N-terminal collagen-like domain followed by a C-terminal globular region. Human CTRP3 shares 99% aa sequence identity with the mouse CTRP3 (2). Like other CTRP members, CTRP3 has a trimeric structure and can assemble into hexameric or higher order molecular forms (3). It is expressed by a variety of cells, including adipocytes, cartilage, fibroblasts, monocytes and proliferating chondrocytes (4). The inflammatory effects of LPS, TLR-4 and fatty acids have been shown to be inhibited by CTRP3 in adipocytes and monocytes (5). In mouse models, CTRP3 has been shown to lower glucose levels and decrease gluconeogenic gene expression (6). Inhibition of 3T3-L1 pre-adipocyte differentiation to adipocytes is associated with CTRP3 treatment, demonstrating potential anti-obesity effects (7). Treatment with CTRP3 results in the proliferation of skeletal muscle C2C12 cells and inhibition of C2C12 myotube differentiation, mediated by the ERK pathway (8). Due to the variety of functions in metabolism and inflammation, CTRP3 is a potential new target of type 2 diabetes treatment.

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