

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human CTRP1/C1qTNF1 in direct ELISAs.
Source	Monoclonal Mouse IgG _{2B} Clone # 395520
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived human CTRP1/C1qTNF1 Arg26-Pro281 Accession # Q9BXJ1
Conjugate	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 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.

Western Blot	Optimal dilution of this antibody should be experimentally determined.
Immunohistochemistry	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

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).

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