SANTA CRUZ BIOTECHNOLOGY, INC.

C1q-A (7H8): sc-58920

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

C1q, a subcomponent of the classical complement pathway, is composed of nine subunits that mediate classical complement activation and thereby play an important role in the immune response. Six of these subunits are disulfide-linked dimers of chains A and B, while three of these subunits, designated C1q-A through C1q-C, are disulfide-linked dimers of chain C. The presence of receptors for C1q on effector cells modulates its activity, which may be antibody-dependent or independent. Macrophages are the primary source of C1q, while anti-inflammatory drugs as well as cytokines differentially regulate expression of the mRNA as well as the protein. However, its ability to modulate the interaction of platelets with collagen and immune complexes suggests C1q influences homeostasis as well as other immune activities, and perhaps thrombotic complications resulting from immune injury. Defects in C1q-A, C1q-B and C1q-C cause inactivation of the classical pathway, leading to a rare genetic disorder characterized by lupus-like symptoms.

REFERENCES

1. Peerschke, E.I. and Ghebrehiwet, B. 1998. Platelet receptors for the complement component C1q: implications for hemostasis and thrombosis. Immunobiology 199: 239-249.
2. Hiepe, F., et al. 1999. C1q: a multifunctional ligand for a new immunoadsorption treatment. Ther. Apher. 3: 246-251.
3. Kishore, U. and Reid, K.B. 2000. C1q: structure, function, and receptors. Immunopharmacology 49: 159-170.
4. Faust, D. and Loos, M. 2002. *In vitro* modulation of C1q mRNA expression and secretion by interleukin-1, interleukin-6, and interferon-γ in resident and stimulated murine peritoneal macrophages. Immunobiology 206: 368-376.
5. Faust, D., et al. 2002. Anti-inflammatory drugs modulate C1q secretion in human peritoneal macrophages *in vitro*. Biochem. Pharmacol. 64: 457-462.
6. Petry, F. and Loos, M. 2005. Common silent mutations in all types of hereditary complement C1q deficiencies. Immunogenetics 57: 566-571.

CHROMOSOMAL LOCATION

Genetic locus: C1QA (human) mapping to 1p36.12; C1qa (mouse) mapping to 4 D3.

SOURCE

C1q-A (7H8) is a rat monoclonal antibody raised against serum-derived complement subcomponent C1q of mouse origin.

PRODUCT

Each vial contains 200 µg IgG_1 in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

C1q-A (7H8) is available conjugated to either phycoerythrin (sc-58920 PE) or fluorescein (sc-58920 FITC), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

APPLICATIONS

C1q-A (7H8) is recommended for detection of precursor and mature C1q-A of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and flow cytometry (1 µg per 1 x 10^6 cells).

Suitable for use as control antibody for C1q-A siRNA (h): sc-43651, C1q-A siRNA (m): sc-44962, C1q-A shRNA Plasmid (h): sc-43651-SH, C1q-A shRNA Plasmid (m): sc-44962-SH, C1q-A shRNA (h) Lentiviral Particles: sc-43651-V and C1q-A shRNA (m) Lentiviral Particles: sc-44962-V.

Molecular Weight of C1q-A: 29 kDa.

Positive Controls: NIH/3T3 whole cell lysate: sc-2210 or RAW 264.7 whole cell lysate: sc-2211.

SELECT PRODUCT CITATIONS

1. Heesemann, L., et al. 2011. Studies on galactofuranose-containing glycostructures of the pathogenic mold *Aspergillus fumigatus*. Int. J. Med. Microbiol. 301: 523-530.
2. Qiao, S., et al. 2012. Mimosine-induced apoptosis in C6 glioma cells requires the release of mitochondria-derived reactive oxygen species and p38, JNK activation. Neurochem. Res. 37: 417-427.
3. Hao, X., et al. 2022. Periodontal infection aggravates C1q-mediated microglial activation and synapse pruning in Alzheimer's mice. Front. Immunol. 13: 816640.
4. Zhang, C., et al. 2022. Paraquat induces microglial cause early neuronal synaptic deficits through activation of the classical complement cascade response. Immunobiology 227: 152275.
5. Xu, F., et al. 2023. Prolonged anesthesia induces neuroinflammation and complement-mediated microglial synaptic elimination involved in neurocognitive dysfunction and anxiety-like behaviors. BMC Med. 21: 7.
6. Wen, L., et al. 2023. The complement inhibitor CD59 is required for GABAergic synaptic transmission in the dentate gyrus. Cell Rep. 42: 112349.
7. Butler, M.J., et al. 2023. Dietary fatty acids differentially impact phagocytosis, inflammatory gene expression, and mitochondrial respiration in microglial and neuronal cell models. Front. Cell. Neurosci. 17: 1227241.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.