SANTA CRUZ BIOTECHNOLOGY, INC.

C1q (1A4): sc-53544

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. Samuel, D.J., et al. 1986. An efficient one-step method for isolating immune complexes from whole serum using a monoclonal anti-C3G affinity immunosorbent. Clin. Exp. Immunol. 65: 458-464.
2. Peerschke, E.I., et al. 1998. Platelet receptors for the complement component C1q: implications for hemostasis and thrombosis. Immunobiology 199: 239-249.
3. Kishore, U., et al. 2000. C1q: structure, function and receptors. Immunopharmacology 49: 159-170.
4. Faust, D., et al. 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. Zabaleta-Lanz, M.E., et al. 2006. Further description of early clinically silent lupus nephritis. Lupus 15: 845-851.

SOURCE

C1q (1A4) is a mouse monoclonal antibody raised against C1q of human origin.

PRODUCT

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

C1q (1A4) is available conjugated to agarose (sc-53544 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-53544 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-53544 PE), fluorescein (sc-53544 FITC), Alexa Fluor® 488 (sc-53544 AF488), Alexa Fluor® 546 (sc-53544 AF546), Alexa Fluor® 594 (sc-53544 AF594) or Alexa Fluor® 647 (sc-53544 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-53544 AF680) or Alexa Fluor® 790 (sc-53544 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

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APPLICATIONS

C1q (1A4) is recommended for detection of complement subcomponent C1q of human origin by immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Molecular Weight of C1q: 29 kDa.

RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml).

SELECT PRODUCT CITATIONS

1. Hong, E.S., et al. 2015. The amount of C1q-adiponectin complex is higher in the serum and the complex localizes to perivascular areas of fat tissues and the intimal-medial layer of blood vessels of coronary artery disease patients. Cardiovasc. Diabetol. 14: 50.
2. Kiriakidis, S., et al. 2017. Complement C1q is hydroxylated by collagen prolyl 4 hydroxylase and is sensitive to off-target inhibition by prolyl hydroxylase domain inhibitors that stabilize hypoxia-inducible factor. Kidney Int. 92: 900-908.
3. Devasundaram, S., et al. 2020. Priming with DNA expressing trimeric HIV V1V2 alters the immune hierarchy favoring the development of V2-specific antibodies in rhesus macaques. J. Virol. 95: e01193-20.
4. Kuppan, J.P., et al. 2021. A morphological transformation in respiratory syncytial virus leads to enhanced complement deposition. Elife 10: e70575.
5. Klingler, J., et al. 2021. Detection of antibody responses against SARS-CoV-2 in plasma and saliva from vaccinated and infected individuals. Front. Immunol. 12: 759688.
6. Feng, P., et al. 2021. Early pregnancy regulates expression of complement components in ovine liver. Anim. Sci. J. 92: e13660.
7. Zhang, L., et al. 2022. Complement regulation in ovine lymph nodes during early pregnancy. Exp. Ther. Med. 23: 166.
8. Zhang, L., et al. 2022. Effects of early pregnancy on the complement system in the ovine thymus. Vet. Res. Commun. 46: 137-145.
9. Han, X., et al. 2022. Selection of early pregnancy specific proteins and development a rapid immunochromatographic test strip in cows. Theriogenology 187: 127-134.
10. Klingler, J., et al. 2022. Immune profiles to distinguish hospitalized versus ambulatory COVID-19 cases in older patients. iScience 25: 105608.

STORAGE

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

RESEARCH USE

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