

C1QBP (74.5.2): sc-23885

BACKGROUND

The human complement subcomponent C1q associates with C1r and C1s in order to yield the first component of the serum complement system (SCS). The SCS contains over 30 glycoproteins that influence physiological mechanisms of the body in response to immune complex (the classical pathway), carbohydrate (the lectin pathway) or bacterial (alternative pathway) initiation. C1q binding protein (C1QBP), also designated gC1q-R, p32 (p33) or HABP1 (hyaluronan-binding protein 1), is known to bind the globular heads of C1q molecules and inhibit C1 activation. C1QBP has been described as a complement receptor for C1q on B cells, neutrophils and mast cells. The C1QBP protein may form homodimers. C1QBP is expressed in vascular endothelial cells and has been found to be a multifunctional protein interacting with elements of complement, coagulation and kinin systems. In addition, C1QBP is a subunit of pre-mRNA splicing factor SF2/ASF.

CHROMOSOMAL LOCATION

Genetic locus: C1QBP (human) mapping to 17p13.2; C1qbp (mouse) mapping to 11 B4.

SOURCE

C1QBP (74.5.2) is a mouse monoclonal antibody raised against recombinant C1QBP corresponding to amino acids 74-282 of mature C1QBP.

PRODUCT

Each vial contains 200 µg IgG₁ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

C1QBP (74.5.2) is available conjugated to agarose (sc-23885 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-23885 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-23885 PE), fluorescein (sc-23885 FITC), Alexa Fluor® 488 (sc-23885 AF488), Alexa Fluor® 546 (sc-23885 AF546), Alexa Fluor® 594 (sc-23885 AF594) or Alexa Fluor® 647 (sc-23885 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-23885 AF680) or Alexa Fluor® 790 (sc-23885 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

APPLICATIONS

C1QBP (74.5.2) is recommended for detection of mature and truncated form of C1QBP 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), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and flow cytometry (1 µg per 1 x 10⁶ cells).

Suitable for use as control antibody for C1QBP siRNA (h): sc-42880, C1QBP siRNA (m): sc-42881, C1QBP shRNA Plasmid (h): sc-42880-SH, C1QBP shRNA Plasmid (m): sc-42881-SH, C1QBP shRNA (h) Lentiviral Particles: sc-42880-V and C1QBP shRNA (m) Lentiviral Particles: sc-42881-V.

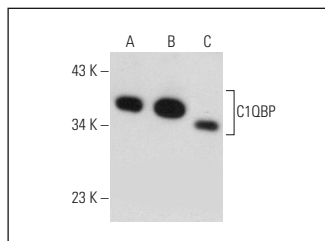
Molecular Weight of C1QBP: 33 kDa.

Positive Controls: MCF7 whole cell lysate: sc-2206, HeLa whole cell lysate: sc-2200 or NIH/3T3 whole cell lysate: sc-2210.

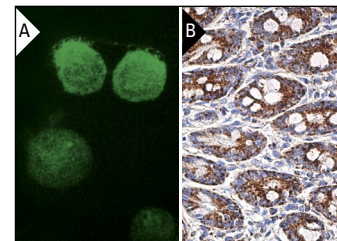
STORAGE

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

DATA



C1QBP (74.5.2): sc-23885. Western blot analysis of C1QBP expression in MCF7 (A), HeLa (B) and NIH/3T3 (C) whole cell lysates.



C1QBP (74.5.2): sc-23885. Immunofluorescence staining of methanol-fixed HL-60 cells showing cytoplasmic localization (A). Immunoperoxidase staining of formalin fixed, paraffin-embedded human duodenum tissue showing cytoplasmic staining of glandular cells (B).

SELECT PRODUCT CITATIONS

1. Sano, E., et al. 2008. Novel tyrosine phosphorylated and cardiolipin-binding protein CLPABP functions as mitochondrial RNA granule. *Biochim. Biophys. Acta* 1783: 1036-1047.
2. Lood, C., et al. 2009. C1q inhibits immune complex-induced interferon- α production in plasmacytoid dendritic cells: a novel link between C1q deficiency and systemic lupus erythematosus pathogenesis. *Arthritis Rheum.* 60: 3081-3090.
3. Irvine, M., et al. 2010. Amino terminal hydrophobic import signals target the p14^{ARF} tumor suppressor to the mitochondria. *Cell Cycle* 9: 829-839.
4. Wu, S., et al. 2016. Herpes simplex virus 1 induces phosphorylation and reorganization of Lamin A/C through the γ _{134.5} protein that facilitates nuclear egress. *J. Virol.* 90: 10414-10422.
5. Changotra, H., et al. 2016. Epstein-Barr virus glycoprotein gM can interact with the cellular protein p32 and knockdown of p32 impairs virus. *Virology* 489: 223-232.
6. Inturi, R., et al. 2018. Human adenovirus infection causes cellular E3 ubiquitin ligase MKRN1 degradation involving the viral core protein pVII. *J. Virol.* 92: e01154-17.
7. Phan, Q.T., et al. 2021. The globular C1q receptor is required for epidermal growth factor receptor signaling during *Candida albicans* infection. *mBio* 12: e0271621.
8. Phan, Q.T., et al. 2022. Serum bridging molecules drive candidal invasion of human but not mouse endothelial cells. *PLoS Pathog.* 18: e1010681.

RESEARCH USE

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

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