

CERTIFICATE OF ANALYSIS – TECHNICAL DATA SHEET

Product name C1q, Mouse, clone JL-1

Catalog number HM1096-5MG

Lot number -

Expiry date -

Volume xx ml

Amount 5 mg

Formulation 0.2 µm filtered in PBS

Concentration >0.5 mg/ml

Host Species Mouse IgG2b

Conjugate None

Endotoxin <24 EU/mg

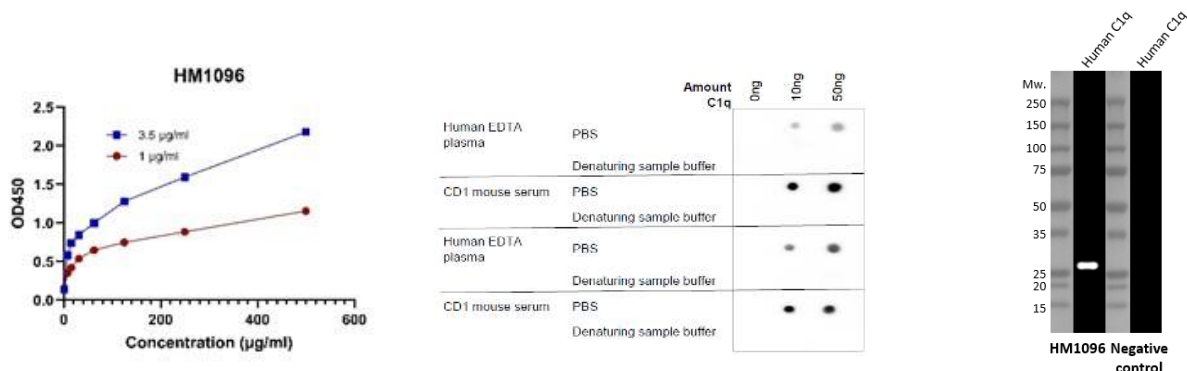
Purification Protein G

Storage 4°C

Application notes

	IHC-F	IHC-P	IF	FC	FS	IA	IP	W
Reference #	1		1		1	1,2,4		5
Yes	•		•		•	•		•
No								
N.D.		•		•			•	

N.D.= Not Determined; IHC = Immuno histochemistry; F = Frozen sections; P = Paraffin sections; IF = Immuno Fluorescence; FC = Flow Cytometry; FS = Functional Studies; IA = Immuno Assays; IP = Immuno Precipitation; W = Western blot



IA: Specificity test of HM1096. HM1096 was used as coat antibody in different concentrations.

Dot blot with HM1096 with mouse serum and human plasma with different amounts of C1q. (Ref 3)

W: Purified C1q protein was resolved on gel reducing conditions and detected with antibody HM1096 at 2 µg/ml.

Dilutions to be used depend on detection system applied. It is recommended that users test the reagent and determine their own optimal dilutions. The typical starting working dilution is 1:50.

- IA: HM1096 can be used as coat or detection antibody (Ref. 1, 2 & 4).
- W: A reduced sample treatment and SDS-PAGE was used. The band size is ~26 kDa under reducing conditions for purified C1q. Please note that C1q is known to form immune complexes, which could vary in larger sizes. (Ref. 5.)
- IHC-F: Antibody JL-1 was used to stain tissue sections which were fixed in acetone. As positive control a polyclonal anti-C1q antibody was used and as negative control an isotype matched monoclonal antibody (Ref.1).
- FS: Antibody JL-1 was administered to mice resulting in depletion of circulating C1q, glomerular deposition of C1q and induction of anti-C1q autoantibodies in susceptible mice. As a negative control an isotype matched monoclonal antibody was used (Ref.1).
- Positive control: Spleen and kidney tissue of wild-type mice (Ref.1); Negative control: Spleen and kidney tissue of C1q -/- mice (Ref.1).

General Information

Description

The monoclonal antibody JL-1 recognizes the collagen-like region (CLR) of mouse C1q, a 459 kDa molecule consisting of three individual polypeptide chains. The antibody has been generated by immunization of C1q^{-/-} C57BL/6 mice with purified mouse C1q.

C1q forms together with C1r and C1s the C1 macromolecule, the first component of the classical complement pathway. Interaction of immune complexes with C1q induces a conformational change within the C1 complex, which results in activation of the classical pathway. C1q functions as recognition unit by binding to the heavy chain of IgG or IgM (Fc gamma and Fc micro) provided that the immunoglobulins are bound to their antigen. Furthermore, C1q can also recognize molecular patterns associated with pathogens and it can bind to apoptotic blebs, where it activates the

classical complement pathway and mediates phagocytosis. As such, C1q promotes the clearance of apoptotic cells and subsequent exposure of auto antigens, thereby preventing stimulation of the immune system.

C1q is predominantly produced by macrophages but also by follicular dendritic cells, interdigitating cells and cells of the monocyte-macrophage lineage. C1q deficiency has a profound effect on host defence and clearance of immune complexes. Absence of C1q may cause autoimmunity by impairment of the clearance of apoptotic cells. Inherited C1q deficiency is also associated with the development of systemic lupus erythematosus (SLE).

The monoclonal antibody JL-1 is reactive with the collagen-like region (CLR) only, which is the same region to which autoantibodies in mice and humans are binding. Anti-C1q autoantibodies deposit in glomeruli together with C1q but induce overt renal disease only in the context of glomerular immune complex disease. This provides an explanation why anti-C1q antibodies are especially pathogenic in patients with SLE.

Immunogen	Purified mouse C1q
Aliases	Complement component C1q, Complement C1q subcomponent subunit A
Cross reactivity	Human: Yes; Rat: Yes.
References	1. Trouw, L et al; Anti-C1q autoantibodies deposit in glomeruli but are only pathogenic in combination with glomerular C1q-containing immune complexes. J Clin Invest 2004, 114: 679 2. Li, M et al; Development of a humanized C1q A chain knock-in mouse: assessment of antibody independent beta-amyloid induced complement activation. Mol Immunol 2008, 45: 3244 3. Erlich, P et al; Complement protein C1q forms a complex with cytotoxic prion protein oligomers. J Biol Chem 2010, 285: 19267 4. Kiriakidis, S et al; 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. Kid Int 2017, 92: 900. 5. Meng, J. et al; Microglial Tmem59 Deficiency Impairs Phagocytosis of Synapse and Leads to Autism-Like Behaviors in Mice. J Neurosci, 2022, 42 (25), 4958-4979
Storage&stability	Product should be stored at 4°C. Under recommended storage conditions, product is stable for at least one year.
Precautions	For research use only. Not for use in or on humans or animals or for diagnostics. It is the responsibility of the user to comply with all local/state and federal rules in the use of this product. Hycult Biotech is not responsible for any patent infringements that might result from the use or derivation of this product.

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