

Datasheet for ABIN2191847
anti-C1q antibody (FITC)

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Overview

Quantity:	100 µg
Target:	C1q
Reactivity:	Mouse
Host:	Mouse
Clonality:	Monoclonal
Conjugate:	This C1q antibody is conjugated to FITC
Application:	Flow Cytometry (FACS), Immunofluorescence (IF), Immunohistochemistry (Frozen Sections) (IHC (fro)), Functional Studies (Func)

Product Details

Clone:	JL-1
Isotype:	IgG2b
Cross-Reactivity (Details):	Cross reactivity: Human : Yes, Rat : Yes
Sterility:	0.2 µm filtered

Target Details

Target:	C1q
Alternative Name:	c1q (C1q Products)
Background:	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

Target Details

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.

Aliases Complement C1q subcomponent subunit A Immunogen Purified mouse C1q

Application Details

Application Notes:	For immunohistochemistry, and Western blotting, 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. For functional studies, in vitro dilutions have to be optimized in user's experimental setting. Positive Spleen and kidney tissue of wild-type mice (Ref.1) control Negative Spleen and kidney tissue of C1q -/- mice (Ref.1) control
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Restrictions:	For Research Use only
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Handling

Buffer:	PBS, containing 1 % bovine serum albumin.
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Storage:	4 °C
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Storage Comment:	Product should be stored at 4 °C. Under recommended storage conditions, product is stable for at least one year. The exact expiry date is indicated on the label.
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Product cited in: Müller, Peri, Doni, Perruchoud, Landmann, Pasqualini, Mantovani: "High circulating levels of the IL-1 type II decoy receptor in critically ill patients with sepsis: association of high decoy receptor levels with glucocorticoid administration." in: **Journal of leukocyte biology**, Vol. 72, Issue 4, pp. 643-9, (2002) ([PubMed](#)).

Penton-Rol, Orlando, Polentarutti, Bernasconi, Muzio, Introna, Mantovani et al.: "Bacterial lipopolysaccharide causes rapid shedding, followed by inhibition of mRNA expression, of the IL-1 type II receptor, with concomitant up-regulation of the type I receptor and induction of ..." in: **Journal of immunology (Baltimore, Md. : 1950)**, Vol. 162, Issue 5, pp. 2931-8, (1999) ([PubMed](#)).