



Mouse Anti-C1qA [MD314]: MC0615, MC0615RTU7

Intended Use: For Research Use Only

Description: 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.

Specifications

Clone: MD314
Source: Mouse
Isotype: IgG2b
Reactivity: Human

Immunogen: Recombinant fragment aa 104-237 of human C1qA protein

Localization: Cytoplasm, secreted

Formulation: Antibody in PBS pH7.4, containing BSA and ≤ 0.09% sodium azide (NaN3)

Storage: Store at 2°-8°C

Applications: IHC

Package:

Description	Catalog No.	Size
C1qA Concentrated	MC0615	1 ml
C1qA Prediluted	MC0615RTU7	7 ml

IHC Procedure*

Positive Control Tissue: Liver, kidney Concentrated Dilution: 50-200

Pretreatment: Tris EDTA pH9.0, 15 minutes Pressure Cooker or 30-60 minutes water bath at 95°-99°C

Incubation Time and Temp: 30-60 minutes @ RT

Detection: Refer to the detection system manual * Result should be confirmed by an established diagnostic procedure.

FFPE human liver tissue stained with anti-C1qA using DAB

References:

- 1. Type I IFNs Regulate Inflammation, Vasculopathy, and Fibrosis in Chronic Cutaneous Graft-versus-Host Disease. Delaney TA, et al. J Immunol 197:42-50, 2016.
- 2. In vitro modulation of C1q mRNA expression and secretion by interleukin-1, interleukin-6, and interferon-γ in resident and stimulated murine peritoneal macrophages. Faust, D. et al. Immunobiology 206: 368-376, 2002.
- 3. Anti-inflammatory drugs modulate C1q secretion in human peritoneal macrophages in vitro. Faust, D., et al. Biochem. Pharmacol. 64: 457-462, 2002.

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