

FITC Goat Anti-Human C1q Polyclonal: IF0005-2ML

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:	Polyclonal
Source:	Goat
Isotype:	IgG
Reactivity:	Human
Localization:	Cytoplasm, membrane
Formulation:	Purified Antibody in PBS pH7.3, with $\leq 0.09\%$ sodium azide (NaN ₃), lyophilized
Storage:	Store at 2°- 8°C
Applications:	IF-FFPE, frozen tissue
Package:	

Description	Catalog No.	Size
FITC Goat Anti-Human C1q Polyclonal	IF0005-2ML	2 ml

IHC Procedure*

Positive Control Tissue:	Liver, kidney, lupus positive tissue
Reconstitution:	Reconstitute product with 2 ml of deionized or distilled water. Gentle swirling may be used to speed rehydration. Avoid vigorous shaking of the reconstituted material
Concentrated Dilution:	100-400
Pretreatment:	Proteinase K, 30 minutes @ 37°C (FFPE)
Incubation Time and Temp:	30-60 minutes @ RT

* Result should be confirmed by an established diagnostic procedure.

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. Vasculitis and crescentic glomerulonephritis in a newly established congenic mouse strain derived from ANCA-associated vasculitis-prone SCG/Kj mice. Yoshitomo Hamano, et al., Autoimmunity, Jul 06, 2020.
3. 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.
4. Anti-inflammatory drugs modulate C1q secretion in human peritoneal macrophages in vitro. Faust, D., et al. Biochem. Pharmacol. 64: 457-462, 2002.

Doc. 100-IF0005
Rev. A