


FITC Anti-C3c antibody ab4212

★★★★★ [1 Abreviews](#) [9 References](#) [1 Image](#)

Overview

Product name	FITC Anti-C3c antibody
Description	FITC Rabbit polyclonal to C3c
Host species	Rabbit
Conjugation	FITC. Ex: 493nm, Em: 528nm
Specificity	This antibody reacts with human C3c complement and with the C3c part of C3 and C3b.
Tested applications	Suitable for: ICC/IF
Species reactivity	Reacts with: Human Predicted to work with: Mouse, Rat, Sheep, Goat, Guinea pig, Cow, Cat, Dog, Pig, Kangaroo, Mink 
Immunogen	Full length native protein (purified) corresponding to Human C3c. C3c complement isolated from complement activated human serum.
General notes	<p>The Life Science industry has been in the grips of a reproducibility crisis for a number of years. Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets your needs before purchasing.</p> <p>If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be found below, along with publications, customer reviews and Q&As</p>

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C.
Storage buffer	Preservative: 0.05% Sodium azide Constituent: 1% BSA
Purity	IgG fraction
Purification notes	Traces of contaminating antibodies have been removed by solid phase absorption with human plasma proteins.
Clonality	Polyclonal
Isotype	IgG

Applications

The Abpromise guarantee

Our **Abpromise guarantee** covers the use of ab4212 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
ICC/IF		Use a concentration of 1 µg/ml.

Target

Function

C3 plays a central role in the activation of the complement system. Its processing by C3 convertase is the central reaction in both classical and alternative complement pathways. After activation C3b can bind covalently, via its reactive thioester, to cell surface carbohydrates or immune aggregates.

Derived from proteolytic degradation of complement C3, C3a anaphylatoxin is a mediator of local inflammatory process. It induces the contraction of smooth muscle, increases vascular permeability and causes histamine release from mast cells and basophilic leukocytes.

Tissue specificity

Plasma.

Involvement in disease

Defects in C3 are the cause of complement component 3 deficiency (C3D) [MIM:613779]. A rare defect of the complement classical pathway. Patients develop recurrent, severe, pyogenic infections because of ineffective opsonization of pathogens. Some patients may also develop autoimmune disorders, such as arthralgia and vasculitic rashes, lupus-like syndrome and membranoproliferative glomerulonephritis.

Genetic variation in C3 is associated with susceptibility to age-related macular degeneration type 9 (ARMD9) [MIM:611378]. ARMD is a multifactorial eye disease and the most common cause of irreversible vision loss in the developed world. In most patients, the disease is manifest as ophthalmoscopically visible yellowish accumulations of protein and lipid that lie beneath the retinal pigment epithelium and within an elastin-containing structure known as Bruch membrane.

Defects in C3 are a cause of susceptibility to hemolytic uremic syndrome atypical type 5 (AHUS5) [MIM:612925]. An atypical form of hemolytic uremic syndrome. It is a complex genetic disease characterized by microangiopathic hemolytic anemia, thrombocytopenia, renal failure and absence of episodes of enterocolitis and diarrhea. In contrast to typical hemolytic uremic syndrome, atypical forms have a poorer prognosis, with higher death rates and frequent progression to end-stage renal disease. Note=Susceptibility to the development of atypical hemolytic uremic syndrome can be conferred by mutations in various components of or regulatory factors in the complement cascade system. Other genes may play a role in modifying the phenotype.

Sequence similarities

Contains 1 anaphylatoxin-like domain.

Contains 1 NTR domain.

Post-translational modifications

C3b is rapidly split in two positions by factor I and a cofactor to form iC3b (inactivated C3b) and C3f which is released. Then iC3b is slowly cleaved (possibly by factor I) to form C3c (beta chain + alpha' chain fragment 1 + alpha' chain fragment 2), C3dg and C3f. Other proteases produce other fragments such as C3d or C3g.

Phosphorylation sites are present in the extracellular medium.

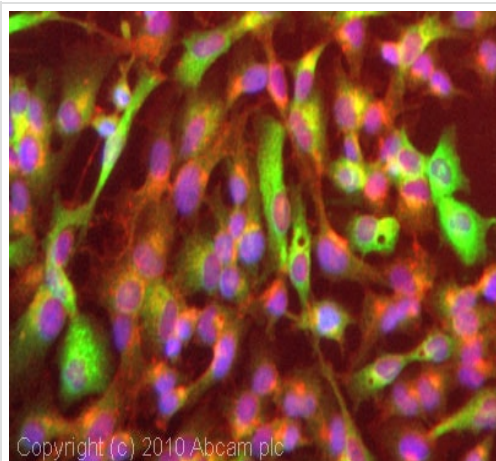
Cellular localization

Secreted.

Form

Cleaved into the following 10 chains: 1) Complement C3 beta chain 2) Complement C3 alpha chain 3) C3a anaphylatoxin 4) Complement C3b alpha' chain 5) Complement C3c alpha' chain fragment 1 6) Complement C3dg fragment 7) Complement C3g fragment 8) Complement C3d fragment 9) Complement C3f fragment 10) Complement C3c alpha' chain fragment 2

Images



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Immunocytochemistry/ Immunofluorescence - FITC
Anti-C3c antibody (ab4212)

ICC/IF image of ab4212 stained HepG2 cells. The cells were 4% formaldehyde fixed (10 min) and then incubated in 1%BSA / 10% normal goat serum / 0.3M glycine in 0.1% PBS-Tween for 1h to permeabilise the cells and block non-specific protein-protein interactions. The cells were then incubated with the antibody (ab4212, 1µg/ml) overnight at +4°C. Alexa Fluor® 594 WGA was used to label plasma membranes (red) at a 1/200 dilution for 1h. DAPI was used to stain the cell nuclei (blue) at a concentration of 1.43µM.

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