abcam

Product datasheet

FITC Anti-C3c antibody ab182878

1 References

Overview

Product name FITC Anti-C3c antibody

Description FITC Goat polyclonal to C3c

Host species Goat

Conjugation FITC. Ex: 493nm, Em: 528nm

Tested applications Suitable for: IHC-Fr, ICC, ICC/IF

Species reactivity Reacts with: Mouse

Immunogen Full length native protein (purified) corresponding to Mouse C3c. (Isolated and purified from

pooled normal mouse serum).

Database link: P01027

General notesThe 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.

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

Properties

Form Lyophilized:Reconstitute by adding 1 ml sterile distilled water. Spin down to remove insoluble

particles

Storage instructions Shipped at 4°C. Store at +4°C. Store In the Dark.

Storage buffer pH: 7.20

Constituent: 100% PBS

No preservative added.

Purity lgG fraction

Clonality Polyclonal

Isotype IgG

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The Abpromise guarantee

Our **Abpromise guarantee** covers the use of ab182878 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
IHC-Fr		Use at an assay dependent concentration.
ICC		Use at an assay dependent concentration.
ICC/IF		Use at an assay dependent concentration.

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

Involvement in disease

Plasma.

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 extracelllular 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 C3d fragment 10) Complement C3c alpha' chain fragment 2	

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