

Product datasheet

Anti-C3 antibody [10A1] ab36989

1 References 1 Image

Overview

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<b>Product name</b>	Anti-C3 antibody [10A1]
<b>Description</b>	Mouse monoclonal [10A1] to C3
<b>Host species</b>	Mouse
<b>Tested applications</b>	<b>Suitable for:</b> ELISA, WB, Flow Cyt
<b>Species reactivity</b>	<b>Reacts with:</b> Human
<b>Immunogen</b>	Complement C3 protein purified from Human plasma.
<b>Positive control</b>	Human plasma.
<b>General notes</b>	<p>This product was changed from ascites to tissue culture supernatant on 18th September 2017. Lot numbers higher than GR191331 will be from tissue culture supernatant. Please note that the dilutions may need to be adjusted accordingly.</p> <p>Abcam is committed to meeting high standards of ethical manufacturing and as such, we will be discontinuing this product, which has been generated by the ascites method, within the next year. We are sorry for any inconvenience this may cause. If you would like help finding an alternative product, please do not hesitate to contact our scientific support team.</p>

Properties

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<b>Form</b>	Liquid
<b>Storage instructions</b>	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
<b>Storage buffer</b>	Preservative: 0.03% Sodium azide Constituents: 50% Glycerol, 0.87% Sodium chloride, 0.01% BSA
<b>Purity</b>	Tissue culture supernatant
<b>Clonality</b>	Monoclonal
<b>Clone number</b>	10A1
<b>Isotype</b>	IgG2b
<b>Light chain type</b>	kappa

Applications

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Our [Abpromise guarantee](#) covers the use of **ab36989** 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
ELISA		Use at an assay dependent concentration.
WB		Use a concentration of 0.5 µg/ml. Predicted molecular weight: 187 kDa.
Flow Cyt		Use at an assay dependent concentration. PubMed: 19129916ab170192-Mouse monoclonal IgG2b, is suitable for use as an isotype control with this antibody.

## 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:120700]. 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.

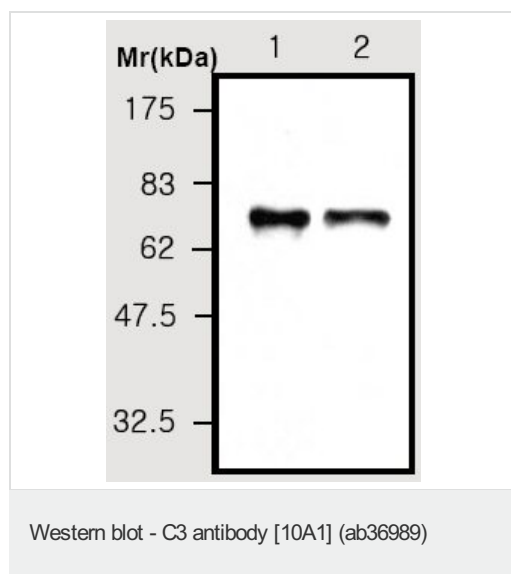
### Post-translational modifications

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.

## Images



**All lanes :** Anti-C3 antibody [10A1]  
(ab36989)

**Lane 1 :** Complement C3 isolated from  
Human plasma

**Lane 2 :** Human plasma

**Predicted band size:** 187 kDa

**Observed band size:** 70 kDa

**Please note:** All products are "FOR RESEARCH USE ONLY AND ARE NOT INTENDED FOR DIAGNOSTIC OR THERAPEUTIC USE"

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