

Product datasheet

Anti-C3 α / C3 α des Arg antibody [2991] ab11873

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Overview

Product name	Anti-C3 α / C3 α des Arg antibody [2991]
Description	Mouse monoclonal [2991] to C3 α / C3 α des Arg
Host species	Mouse
Specificity	ab11873 reacts with a neo-epitope (des-Arg) on C3 α that is formed when C3 is cleaved into C3 α and C3 β . ab11873 recognizes C3 α and C3 α des arg only. It has the most affinity with C3 α des arg and least affinity with C3 α . The des arg variant has an about 5x higher affinity than C3 α . The antibody does not recognize C3 β or full C3, as it recognizes a neo-epitope that is not available on C3.
Tested applications	Suitable for: WB
Species reactivity	Reacts with: Human
Immunogen	Other Immunogen Type corresponding to Human C3 α / C3 α des Arg. Database link: P01024
Positive control	Activated human serum or purified human C3 α desArg protein
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. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
Storage buffer	Preservative: 0.02% Sodium azide Constituents: PBS, 0.1% BSA
Purity	Protein G purified
Purification notes	0.2 μ m filtered
Clonality	Monoclonal

Clone number 2991
Isotype IgG1

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab11873 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
WB		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 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.

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