abcam

Product datasheet

Anti-C3a / C3a des Arg antibody [2991] ab11873

★★★★★★ <u>1 Abreviews</u> <u>5 References</u>

Overview		
Product name	Anti-C3a / C3a des Arg antibody [2991]	
Description	Mouse monoclonal [2991] to C3a / C3a des Arg	
Host species	Mouse	
Specificity	ab11873 reacts with a neo-epitope (des-Arg) on C3a that is formed when C3 is cleaved into C3a and C3b. ab11873 recognizes C3a and C3a des arg only. It has the most affinity with C3a des arg and least affinity with C3a. The des arg variant has an about 5x higher affinity than C3a. The antibody does not recognize C3b 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 C3a/ C3a des Arg. Database link: P01024	
Positive control	Activated human serum or purified human C3adesArg protein	
General notes	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.	
	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	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 µm filtered
Clonality	Monoclonal

Clone number	2991
lsotype	lgG1

Applications

Our <u>Abpromise guarantee</u> covers the use of ab11873 in the following tested applications. The Abpromise guarantee

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 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.

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