abcam

Product datasheet

Anti-C3 antibody [bH6] ab90814

Overview

Product name	Anti-C3 antibody [bH6]
Description	Mouse monoclonal [bH6] to C3
Tested applications	**Suitable for:** IHC-P, IHC-Fr, ELISA
Species reactivity	**Reacts with:** Human
Immunogen	zymosan-activated serum(human)
Epitope	Recognizes a neo epitope expressed on the cleavage fragments of C3b, iC3b and C3c but not C3dg and C3f.

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: 0.1% BSA, PBS
Purity	Protein G purified
Purification notes	0.2 µm filtered antibody solution
Clonality	Monoclonal
Clone number	bH6
Isotype	IgG2a

Applications

Our Abpromise guarantee covers the use of **ab90814** 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-P		
IHC-Fr		
ELISA		

Application notes	ELISA: Use at an assay dependent dilution. IHC-P: 1/50. IHC-Fr: 1/50. Not yet tested in other applications. Optimal dilutions/concentrations should be determined by the end user.

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 extracelllular medium.
Cellular localization	Secreted.

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