Product datasheet

Anti-C3a / C3a des Arg antibody [K13/16] ab36385

Overview

Product name: Anti-C3a / C3a des Arg antibody [K13/16]
Description: Mouse monoclonal [K13/16] to C3a / C3a des Arg
Host species: Mouse
Specificity: Does not cross react with C4a or C5a.
Tested applications: Suitable for: ELISA, WB, IHC-P
Species reactivity: Reacts with: Human
Immunogen: Full length native protein (purified) corresponding to Human C3a/ C3a des Arg.
Epitope: Recognizes an epitope present on human C3, C3a and C3a(desArg).
General notes: Effectively inhibits the biological activity of C3a in a guinea-pig platelet activation assay.

Properties

Form: Liquid
Storage buffer: Preservative: 15mM Sodium Azide
Constituents: 0.5M Sodium chloride, 0.01M PBS, pH 7.4
Purity: Protein A purified
Primary antibody notes: Effectively inhibits the biological activity of C3a in a guinea-pig platelet activation assay.
Clonality: Monoclonal
Clone number: K13/16
Isotype: IgG1
Light chain type: kappa

Applications

Our Abpromise guarantee covers the use of ab36385 in the following tested applications.
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.
**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.

### Application

<table>
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<tr>
<th>Application</th>
<th>Abreviews</th>
<th>Notes</th>
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</thead>
<tbody>
<tr>
<td>ELISA</td>
<td></td>
<td>1/32000.</td>
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<tr>
<td>WB</td>
<td></td>
<td>Use at an assay dependent concentration. Predicted molecular weight: 75, 110 kDa. In vivo, human C3 exists as a 185kDa protein, formed of two smaller proteins linked together via a disulfide bond. In Western blot the proteins will separate and be detected as bands at molecular weights of approximately 75kDa and 110kDa.</td>
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<tr>
<td>IHC-P</td>
<td></td>
<td>1/150.</td>
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</table>

**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.
Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) analysis of human kidney tissue labelling C3a / C3a des Arg with ab36385 at a dilution of 1/150. Strong staining is visible in the veins of nephritis.

Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) analysis of human liver tissue labelling C3a / C3a des Arg with ab36385 at a dilution of 1/150. Strong staining is visible in the veins.

Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) analysis of human tonsil tissue labelling C3a / C3a des Arg with ab36385 at a dilution of 1/150. Strong staining is visible in the lumen of veins.
Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) analysis of human kidney tissue labelling C3a / C3a des Arg with ab36385 at a dilution of 1/75. Strong staining is visible in the veins of nephritis.

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