## Anti-C3 antibody [8G4] ab17455

### Overview

<table>
<thead>
<tr>
<th>Product name</th>
<th>Anti-C3 antibody [8G4]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Description</td>
<td>Mouse monoclonal [8G4] to C3</td>
</tr>
<tr>
<td>Host species</td>
<td>Mouse</td>
</tr>
<tr>
<td>Specificity</td>
<td>Ab17455 is specific for an allotypic marker on human C3 (Leu instead of Pro in codon 314 of exon 9), which occurs regularly in the beta-chain of C3F and occasionally in the beta-chain of C3S. F and S mean fast and slow, respectively, on agarose gel electrophoresis. Ab17455 reacts with the 75-kDa beta-chain band on SDS-PAGE immunoblotting of reduced C3, and reacts with 20-kDa and 17-kDa beta-chain fragments produced by cyanogen bromide cleavage.</td>
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</tbody>
</table>

### Tested applications

**Suitable for:** ELISA, WB

### Species reactivity

**Reacts with:** Human

### Immunogen

Full length native C3 protein isolated from human plasma.

### Epitope

The exact epitope is not known, but the antibody is specific for the allotypic human C3 where Leu is found instead of Pro in codon 314 of exon 9.

### Properties

<table>
<thead>
<tr>
<th>Form</th>
<th>Liquid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Storage</td>
<td>Shipped at 4°C. Store at +4°C short term (1-2 weeks). Store at -20°C or -80°C. Avoid freeze / thaw cycle.</td>
</tr>
</tbody>
</table>
| Storage buffer| Preservative: None  
Constituents: 0.5M Sodium chloride, 0.01M PBS, pH 7.4 |
| Purity        | Protein A purified |
| Clonality     | Monoclonal |
| Clone number  | 8G4 |
| Myeloma       | x63-Ag8.653 |
| Isotype       | IgG2a |
| Light chain type | kappa |

### Applications
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.
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