

## Product datasheet

# Biotin Anti-C3c antibody ab182909

### Overview

<b>Product name</b>	Biotin Anti-C3c antibody
<b>Description</b>	Biotin Goat polyclonal to C3c
<b>Host species</b>	Goat
<b>Conjugation</b>	Biotin
<b>Specificity</b>	Biotin-conjugated IgG fraction of polyclonal Goat antiSerum to C3c fragment of Rat complement factor C3.  The antiSerum does not cross-react with any other component of Rat plasma. Inter-species cross-reactivity is a normal feature of antibodies to plasma proteins since they frequently share antigenic determinants. Crossreactivity of this antiSerum has not been tested in detail.
<b>Species reactivity</b>	<b>Reacts with:</b> Rat
<b>Immunogen</b>	Full length native protein (purified) corresponding to Rat C3c. C3c is isolated and purified from pooled normal Rat serum. Freund's complete adjuvant is used in the first step of the immunization procedure.  Database link: <a href="#">P01026</a>
<b>General notes</b>	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

<b>Form</b>	Lyophilized: Add 1 mL of sterile distilled water.
<b>Storage instructions</b>	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C long term. Avoid freeze / thaw cycle. Store In the Dark.
<b>Storage buffer</b>	pH: 7.20 Constituent: 100% PBS
	No preservative added, as it may interfere with the antibody activity.
<b>Purity</b>	IgG fraction

<b>Clonality</b>	Polyclonal
<b>Isotype</b>	IgG
<b>Target</b>	
<b>Function</b>	<p>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.</p> <p>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.</p>
<b>Tissue specificity</b>	Plasma.
<b>Involvement in disease</b>	<p>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.</p> <p>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.</p> <p>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.</p>
<b>Sequence similarities</b>	<p>Contains 1 anaphylatoxin-like domain.</p> <p>Contains 1 NTR domain.</p>
<b>Post-translational modifications</b>	<p>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.</p> <p>Phosphorylation sites are present in the extracellular medium.</p>
<b>Cellular localization</b>	Secreted.
<b>Form</b>	Cleaved into the following 10 chains: 1) Complement C3 beta chain 2) Complement C3 alpha chain 3) C3a anaphylatoxin 4) Complement C3b alpha' chain 5) Complement C3c alpha' chain fragment 1 6) Complement C3dg fragment 7) Complement C3g fragment 8) Complement C3d fragment 9) Complement C3f fragment 10) Complement C3c alpha' chain fragment 2

**Please note:** All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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