



## Rabbit Anti-Complement C3g fragment antibody

SL4876R

<b>Product Name:</b>	Complement C3g fragment
<b>Chinese Name:</b>	补体C3g片段抗体
<b>Alias:</b>	Acylation stimulating protein; ASP; C3; C3a anaphylatoxin; Complement C3 alpha chain; Complement C3; Complement C3 precursor; Complement component 3; Complement component C3; Complement factor 3; Plp; Complement C3g fragment; CO3 HUMAN.
<b>Organism Species:</b>	Rabbit
<b>Clonality:</b>	Polyclonal
<b>React Species:</b>	Human,
<b>Applications:</b>	ELISA=1:500-1000IHC-F=1:400-800IF=1:100-500 (Paraffin sections need antigen repair) not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
<b>Molecular weight:</b>	5/181kDa
<b>Cellular localization:</b>	Secretory protein
<b>Form:</b>	Lyophilized or Liquid
<b>Concentration:</b>	1mg/ml
<b>immunogen:</b>	KLH conjugated synthetic peptide derived from human Complement C3g fragment:955-1001/1663
<b>Lsotype:</b>	IgG
<b>Purification:</b>	affinity purified by Protein A
<b>Storage Buffer:</b>	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
<b>Storage:</b>	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
<b>PubMed:</b>	<a href="#">PubMed</a>
<b>Product Detail:</b>	The complement factor C3 consists of an alpha and a beta chain. C3 is a central factor in the complement cascade. It is central to the alternative pathway that leads to the C3 convertase C3bBb. The classical mannose binding lectin activation pathway leads to

the C3 convertase C4b2a. These convertases cleave C3 resulting in C3a and C3b. Further degradation leads to the formation of the alpha chain products C3d, C3g and C3c. C3 is an acute phase protein that is produced by a wide range of tissues, including renal epithelial cells and hepatocytes.

**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.

Acylation stimulating protein (ASP): adipogenic hormone that stimulates triglyceride (TG) synthesis and glucose transport in adipocytes, regulating fat storage and playing a role in postprandial TG clearance. Appears to stimulate TG synthesis via activation of the PLC, MAPK and AKT signaling pathways. Ligand for GPR77. Promotes the phosphorylation, ARRB2-mediated internalization and recycling of GPR77.

**Subunit:**

C3 precursor is first processed by the removal of 4 Arg residues, forming two chains, beta and alpha, linked by a disulfide bond. C3 convertase activates C3 by cleaving the alpha chain, releasing C3a anaphylatoxin and generating C3b (beta chain + alpha' chain). C3dg interacts with CR2 (via the N-terminal Sushi domains 1 and 2). During pregnancy, C3dg exists as a complex (probably a 2:2:2 heterohexamer) with AGT and the proform of PRG2. Interacts with VSIG4. C3b interacts with herpes simplex virus 1 (HHV-1) and herpes simplex virus 2 (HHV-2) envelope glycoprotein C; this interaction inhibits the activation of the complement system. Interacts with S.aureus immunoglobulin-binding protein sbi, this prevents interaction between C3dg and CR2. Interacts with S.aureus fib.

**Subcellular Location:**

Secreted.

**Tissue Specificity:**

Plasma. The acylation stimulating protein (ASP) is expressed in adipocytes and released into the plasma during both the fasting and postprandial periods.

**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. C3a is further processed by carboxypeptidases to release the C-terminal arginine residue generating the acylation stimulating protein (ASP). Levels of ASP are increased in adipocytes in

the postprandial period and by insulin and dietary chylomicrons. Phosphorylation sites are present in the extracellular medium.

**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. Note=Increased levels of C3 and its cleavage product ASP, are associated with obesity, diabetes and coronary heart disease. Short-term endurance training reduces baseline ASP levels and subsequently fat storage.

**Similarity:**

Contains 1 anaphylatoxin-like domain.

Contains 1 NTR domain.

**SWISS:**

P01024

**Gene ID:**

718

**Database links:**

[Entrez Gene: 718](#)Human

[Omic: 120700](#)Human

[SwissProt: P01024](#)Human

**Important Note:**

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

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