abcam

Product datasheet

Human Complement C3 ELISA Kit ab108823

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Overview

Product name Human Complement C3 ELISA Kit

Detection method Colorimetric

Precision

Intra-assay

Sample	n	Mean	SD	CV%
Overall				4.3%

Inter-assay

Sample	n	Mean	SD	CV%
Overall				9.8%

Sample type Cell culture supernatant, Saliva, Milk, Urine, Tissue, Cell Lysate, Cerebral Spinal Fluid

Assay type Sandwich (quantitative)

Sensitivity = 0.32 ng/ml

Range 0.625 ng/ml - 40 ng/ml

Recovery 97 %

Assay time 4h 00m

Assay duration Multiple steps standard assay

Species reactivity **Reacts with:** Human

Product overview Human Complement C3 ELISA kit is designed for the quantitative measurement of Complement C3 concentrations in Human urine, milk, saliva, cerebrospinal fluid and cell culture supernatants.

A Complement C3 specific antibody has been precoated onto 96-well plates and blocked. Standards or test samples are added to the wells and subsequently a Complement C3 specific biotinylated detection antibody is added and then followed by washing with wash buffer. Streptavidin-Peroxidase Conjugate is added and unbound conjugates are washed away with wash buffer. TMB is then used to visualize Streptavidin-Peroxidase enzymatic reaction. TMB is catalyzed by Streptavidin-Peroxidase to produce a blue color product that changes into yellow after adding acidic stop solution. The density of yellow coloration is directly proportional to the amount of Complement C3 captured in plate.

The entire kit may be stored at -20°C for long term storage before reconstitution - Avoid repeated freeze-thaw cycles.

Platform Microplate

Properties

Storage instructions Store at -20°C. Please refer to protocols.

Components	1 x 96 tests
50X Biotinylated Human Complement C3 Antibody	1 x 120µl
100X Streptavidin-Peroxidase Conjugate	1 x 80µl
10X Diluent N Concentrate	1 x 30ml
20X Wash Buffer Concentrate	2 x 30ml
Chromogen Substrate	1 x 7ml
Complement C3 Microplate (12 x 8 well strips)	1 unit
Complement C3 Standard	1 vial
Sealing Tapes	3 units
Stop Solution	1 x 11ml

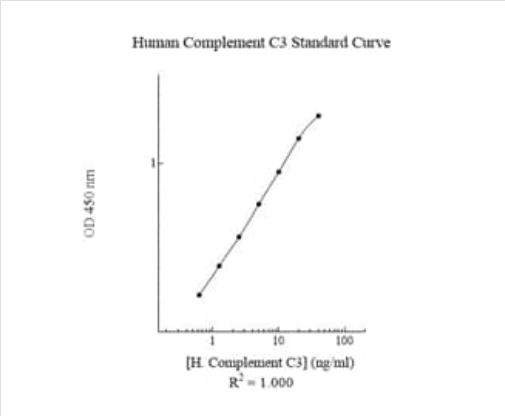
Function C3 plays a central role in the activation of the complement system. Its processing by C3 convertase is the central reaction in both classical and alternative complement pathways. After activation C3b can bind covalently, via its reactive thioester, to cell surface carbohydrates or immune aggregates.
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

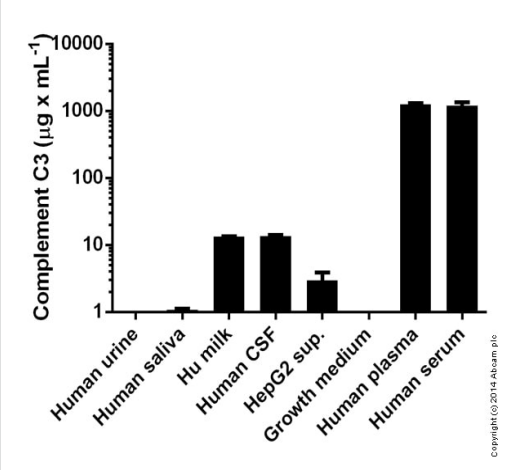
	absence of episodes of enterocolitis and diarrhea. In contrast to typical hemolytic uremic syndrome, atypical forms have a poorer prognosis, with higher death rates and frequent progression to end-stage renal disease. Note=Susceptibility to the development of atypical hemolytic uremic syndrome can be 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.

Images



Human Complement C3 ELISA Kit

Representative Standard Curve using ab108823



Sandwich ELISA - ab108823 Complement C3 Human ELISA kit

Complement C3 measured in various samples showing quantity (microgram) per mL of tested sample

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