Complement Factor H, CFH (CFH) Antibody – Rabbit Polyclonal

PRODUCT OVERVIEW

Product Name	complement factor H, CFH, Polyclonal Antibody
Full Product Name	Rabbit anti-human complement factor H, CFH polyclonal Antibody
Product Gene Name	anti-CFH antibody
Product Synonym Names	complement factor H; CFH; ARMD4; ARMS1; CFHL3; FH; FHL1; HF; HF1; HF2; HUS; MGC88246
Clonality	Polyclonal
Isotype	IgG
Host	Rabbit
Species Reactivity	Human
Purity/Purification	Antigen Affinity Purified
Molecular Weight	7,746 Da
Immunogen	Human CFH
Storage Buffer	PBS with 0.1% Sodium Azide, 50% Glycerol, pH 7.3. -20 degree C, Avoid freeze / thaw cycles.
Santa Cruz Alternative	Potential replacement for Santa Cruz Biotechnology antibody catalog# sc-17949 / sc-33157
ISO Certification	Manufactured in an ISO 9001:2015 Certified Laboratory.
Applications Tested/Suitable for anti-CFH antibody	ELISA (EIA), Western Blot (WB)
Related Product Information for anti-CFH antibody	MBS714336 is an antibody targeting CFH. Also known as Rabbit anti-human complement factor H, CFH polyclonal Antibody. Reactive in Human. Host: Rabbit.
Other Notes	Small volumes of anti-CFH antibody vial(s) may occasionally become entrapped in the seal of the product vial during shipment and storage. If necessary, briefly centrifuge the vial on a tabletop centrifuge to dislodge any liquid in the container`s cap. Certain products may require to ship with dry ice and additional dry ice fee may apply.

ADDITIONAL DETAILS

NCBI/Uniprot data below describe general gene information for CFH. It may not necessarily be applicable to this product.

NCBI GI #	555431917
NCBI Accession #	AGZ15310.1
NCBI Official Full Name	complement factor H, partial
UniProt Protein Name	Complement factor H
UniProt Synonym Protein Names	Complement factor H
Protein Family	Complement factor
UniProt Gene Name	CFH
UniProt Entry Name	U5TZG9_HUMAN

NCBI Summary for CFH	This gene is a member of the Regulator of Complement Activation (RCA) gene cluster and encodes a protein with twenty short consensus repeat (SCR) domains. This protein is secreted into the bloodstream and has an essential role in the regulation of complement activation, restricting this innate defense mechanism to microbial infections. Mutations in this gene have been associated with hemolytic-uremic syndrome (HUS) and chronic hypocomplementemic nephropathy. Alternate transcriptional splice variants, encoding different isoforms, have been characterized. [provided by RefSeq, Oct 2011]
UniProt Comments for CFH	CFH: Factor H functions as a cofactor in the inactivation of C3b by factor I and also increases the rate of dissociation of the C3bBb complex (C3 convertase) and the (C3b)NBB complex (C5 convertase) in the alternative complement pathway. Genetic variations in CFH are associated with basal laminar drusen (BLD); also known as drusen of Bruch membrane or cuticular drusen or grouped early adult-onset drusen. Drusen are extracellular deposits that accumulate below the retinal pigment epithelium on Bruch membrane. Basal laminar drusen refers to an early adult-onset drusen phenotype that shows a pattern of uniform small, slightly raised yellow subretinal nodules randomly scattered in the macula. In later stages, these drusen often become more numerous, with clustered groups of drusen scattered throughout the retina. In time these small basal laminar drusen may expand and ultimately lead to a serous pigment epithelial detachment of the macula that may result in vision loss. Defects in CFH are the cause of complement factor H deficiency (CFHD). A disorder that can manifest as several different phenotypes, including asymptomatic, recurrent bacterial infections, and renal failure. Laboratory features usually include decreased serum levels of factor H, complement component C3, and a decrease in other terminal complement components, indicating activation of the alternative complement pathway. It is associated with a number of renal diseases with variable clinical presentation and progression, including membranoproliferative glomerulonephritis and atypical hemolytic uremic syndrome. Defects in CFH are a cause of susceptibility to hemolytic uremic syndrome atypical type 1 (AHUS1). 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. 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. Genetic variation in CFH is associated with age-related macular degeneration type 4 (ARMD4). 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 (known as drusen) that lie beneath the retinal pigment epithelium and within an elastin- containing structure known as Bruch membrane. 2 isoforms of the human protein are produced by alternative splicing. Protein type: Secreted, signal peptide; Secreted Chromosomal Location of Human Ortholog: 1q32 Cellular Component: extracellular space; extracellular region Molecular Function: heparan sulfate proteoglycan binding; heparin binding; protein binding Biological Process: complement activation, alternative pathway; regulation of complement activation; innate immune response; complement activation Disease: Complement Factor H Deficiency; Basal Laminar Drusen; Hemolytic Uremic Syndrome, Atypical, Susceptibility To, 1; Macular Degeneration, Age-related, 4
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