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Perspective

An Overview of Complement System

Andrew K Godwin*, **Mitchell W Braun**

Department of Pathology and Laboratory Medicine, University of Kansas Medical Center, Kansas City, USA

DESCRIPTION

The complement system, also known as the complement cascade, is an immune system component that boosts antibodies' and phagocytic cells' ability to eliminate pathogens and damaged cells from an organism, stimulate inflammation, and assault the pathogen's cell membrane. It's a part of the body's innate immune system, which isn't flexible and doesn't alter with time. Antibodies produced by the adaptive immune system, on the other hand, can recruit and activate the complement system. The complement system is made up of a variety of tiny proteins that are generated by the liver and circulate as inactive precursors in the blood. Proteases in the system cut certain proteins to release cytokines and start an amplifying cascade of additional cleavages when prompted by one of several triggers. The stimulation of phagocytes to clear foreign and damaged material, inflammation to attract extra phagocytes, and activation of the cell-killing membrane assault complex are all outcomes of this complement activation or complement fixation cascade. The complement system is made up of about 50 proteins and protein fragments, including serum proteins and cell membrane receptors. They make up roughly 10% of the globulin portion of blood serum.

The complement system is activated by three biochemical pathways: the classical complement pathway, the alternative complement pathway, and the lectin pathway. The alternative pathway is responsible for the majority of terminal pathway activation; treatment attempts in disease have focused on blocking it. Components of the complement cascade, which are part of the innate immune system, have recently been discovered in protostome horseshoe crab species, putting the system's origins back further than previously assumed. As the complement system has the potential to cause significant damage to host tissues, its activation must be carefully controlled. Complement control proteins are found in larger concentrations in the plasma than complement proteins themselves, and they govern the complement system. On the membranes of self-cells, some complement control proteins are present, preventing complement from attacking them.

Hepatocytes produce the majority of the proteins and glycoproteins that make up the complement system. Tissue macrophages, blood monocytes, and epithelial cells of the genitourinary system and gastrointestinal tract also produce substantial amounts. The three activation routes all produce homologous C3-convertase protease variants. C3 hydrolysis or antigens can activate the mannose-binding lectin pathway without the presence of antibodies. C3-convertase cleaves and activates component C3 in all three pathways, resulting in C3a and C3b and a cascade of additional cleavage and activation processes.

Many diseases with an immune component are thought to involve the complement system, including Barraquer–Simons syndrome, asthma, lupus erythematous, glomerulonephritis, and various forms of arthritis, autoimmune heart disease, multiple sclerosis, inflammatory bowel disease, paroxysmal nocturnal hemoglobinuria, atypical hemolytic uremic syndrome, ischemia-reperfusion injuries, and transplanted organ rejection.

CONCLUSION

The complement system is increasingly being linked to central nervous system illnesses including Alzheimer's disease and other neurodegenerative ailments like spinal cord injury. Atypical hemolytic uremic syndrome and C3 glomerulopathy have been linked to mutations in complement regulator genes, particularly factor H. Furthermore, various single nucleotide polymorphisms and mutations in the complement factor H gene have been linked to age-related macular degeneration, a prevalent eye condition. A person's chance of developing age-related macular degeneration is also affected by polymorphisms in complement component, complement factor B, and complement factor I, as well as deletion of complement factor H-related and complement factor H-related. Both illnesses are assumed to be caused by complement over activation on the surface of host cells or in plasma, with the molecular location of genetic variation in complement proteins offering insights to the disease mechanisms.

Correspondence to: Andrew K Godwin, Department of Pathology and Laboratory Medicine, University of Kansas Medical Center, Kansas City, USA, E-mail: agodwin@1kumc.edu

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