

Catalogue# MCA-5F2: Mouse Monoclonal Antibody to complement C3

The Immunogen: Complement component 3, often simply called C3, is the third protein in complement system. The complement system is a part of the immune system to complement the ability of antibodies and phagocytic cells to clear pathogens such as bacteria and viruses from organism, trigger inflammation and remove debris from cells and tissues (1). C3 plays a central role in complement activation, and is involved in both the [classical](#) and [alternative pathway](#). C3 is synthesized as an intracellular precursor (pro-C3) of 185 kDa which is processed by proteolytic cleavage into two mature chains: alpha subunit (115 kDa) and beta subunit (70 kDa), linked by a disulfide bond (2). C3 activation involves cleavage by C3 convertase into C3a (9 kDa) and C3b (176 kDa). C3a is released into the surrounding fluids. It can bind to receptors on basophils and mast cells triggering them to release their vasoactive contents (e.g., histamine). Because of the role of these materials in anaphylaxis, C3a is called an anaphylatoxin. In blood, C3a is cleaved by carboxypeptidase to produce C3adesArg or ASP (acylation-stimulating protein), which acts as a paracrine signal to increase triglyceride synthesis in adipocytes (3). C3adesArg have been demonstrated to be present at increased levels in patients with obesity, diabetes mellitus type 2 and coronary artery disease (4, 5, 6). C3b is the main effector molecule of the complement system. C3b expresses multiple binding sites for other complement components (C5, properdin, factor B, H), and some membrane proteins (MCP). Binding these proteins to C3b leads either to amplification of C3 convertase, or initiation of Membrane attack Complex (MAC). C3b also serves as an opsonizing agent to bind to the pathogen and target it for destruction by phagocytes. On the other hand, binding of C3b to complement component, factor I and a co-factor, inactivates C3b to iC3b and release C3f (2 kDa). iC3b can further be cleaved to form C3c, C3dg which further produces C3d and C3g (3). Overall, C3 promotes phagocytosis, supports local inflammatory responses against pathogens, and instructs the adaptive immune response to select the appropriate antigens for a humoral response (6). More recently, C3 has been suggested to have a pathophysiological role in Alzheimer's and other neurodegenerative disorders (7). In clinical, the level of (C3) in serum and CSF can be used to help identify immunological disorders, especially those associated with deficiencies of complement components.

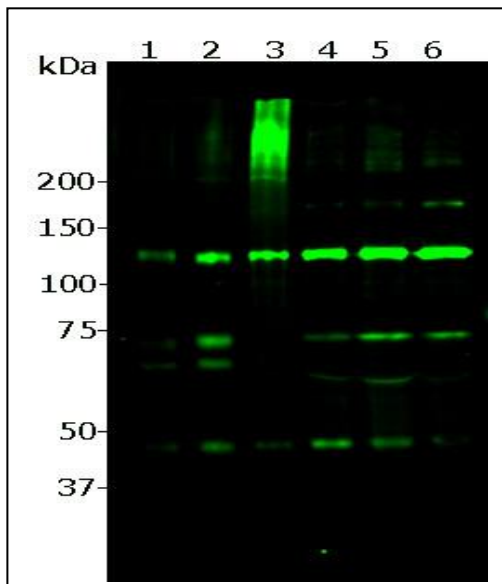


Figure: Western blot analysis of MCA-5F2 on human serum samples. IgG depleted-human serum samples (lane 1, 2), 0.1 µg of purified human C3 protein (lane 3), 10 µg of normal human serum proteins (lanes 4-6) were all blotted with MCA-5F2 at a concentration 1:10,000. The MCA-5F2 monoclonal binds strongly and cleanly to a band at about 115 kDa which represents the intact α subunit of C3 and various proteolytic bands at approximately 68, 65 and 45 kDa. Bands at 190 kDa and above are likely the pro-C3 and its glycosylated form. This antibody does not recognize our recombinant C3 anaphylatoxin domain construct, corresponding to the N-terminus of the α subunit, or our recombinant netrin-like C3 domain construct, corresponding to the C-terminus of the α subunit. As a result we tentatively conclude that the epitope for this antibody is within the α subunit between these two domains.

Antibody characteristics: Antibody is generated from mouse immunized with human C3 protein isolated from human serum. This antibody is mouse IgG1 class antibody and supplied as an aliquot of purified preparation at 1 mg/mL in PBS with 5 mM sodium azide as a preservative (Link to <http://www.encorbio.com/MSDS/azide.htm> for Material Safety Data Sheet).

Suggestions for use: Try at dilutions of 1:10,000 for western blot.

Omim link: <http://omim.org/entry/300005>

References:

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Limitations: This product is for research use only and is not approved for use in humans or in clinical diagnosis.

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