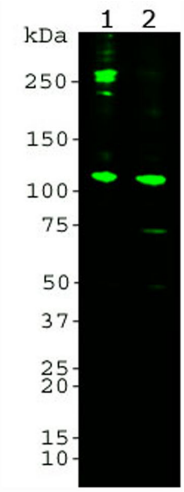
Mouse mAb to Complement C3 MCA-6B1

Ordering Information
Web www.encorbio.com
Email admin@encorbio.com
Phone 352-372-7022
Fax 352-372-7066

HGNC name: C3
RRID: AB_2572254
Immunogen: Recombinant human C3 N-terminal anaphylatoxin construct, amino acids 668-741 of NP_000055.2
Format: Purified antibody at 1mg/mL in 50% PBS, 50% glycerol plus 5mM NaN_3
Storage: Shipped on ice. Store at 4°C for short term, for longer term at -20°C. Avoid freeze / thaw cycles
Recommended dilutions:
Western blot: 1:5,000-1:10,000

Applications	Host	Isotype	Molecular Wt.	Species Cross-Reactivity
Western blot, Elisa	**Mouse**	**IgM**	**185kDa pro-C3, intact α chain at 115kDa and lower molecular weight α chain fragments.**	**Hu**



Western blot analysis of MCA-6B1 on human serum samples. Blot of 0.1µg purified human C3 protein (lane 1), 10µg normal human serum proteins (lane2) was probed with MCA-6B1 at 1:5,000 dilution. The MCA-6B1 monoclonal binds strongly and cleanly to a band at about 115kDa which represents the intact α subunit of C3 and various proteolytic bands at approximately 68 and 48kDa. Bands at 190kDa and above are likely the pro-C3 and its glycosylated form.

Background: Complement component 3, often simply called C3, is the third protein in the 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 the 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 large chains called the α subunit (115 kDa) and the β sunbunit (70 kDa) which are however linked by a disulfide bond (2).

C3 activation involves further cleavage by C3 convertase to produce C3a (9 kDa) and C3b (the remaining 176 kDa of the β subunit and truncated α subunits). C3a is released into the surrounding fluids and 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, and the N-terminal region of the α subunit is referred to as the anaphyloxin domain. In blood, C3a may be further 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

References:

1. Janeway CA J, Travers P, Walport M et al. The complement system and innate immunity. ImmunobiologyThe Immune System in Health and Disease. 5th edition (2001).
2. Brade V, Hall RE, and Colten HR. Biosynthesis of pro-C3, a precursor of the third component of complement.J. Exp. Med. 146:759-765 (1977).
3. Xia Z, Stanhope KL, Digitale E, Simion OM, Chen L, Havel P, Cianflone K. Acylation-stimulating Protein (ASP)/Complement C3adesArg Deficiency Results in Increased Energy Expenditure in MiceJ Biol Chem. 279:4051-7 (2004).
4. Maslowska M, Vu H, Phelis S, Sniderman AD, Rhode BM, Blank D, Cianflone K. Plasma acylation stimulating protein, adipsin and lipids in non-obese and obese populations. European Journal of Clinical Investigation 29:679–86 (1999).
5. Koistinen HA, Vidal H, Karonen SL, Dusserre E, Vallier P, Koivisto VA, Ebeling P. Plasma Acylation Stimulating Protein Concentration and Subcutaneous Adipose Tissue C3 Type 2 Diabetic Men. Atherosclerosis, Thrombosis, and Vascular Biology 21(6):1034–9 (2001).
6. Cianflone K, Zhang XJ, Genest J Jr, Sniderman A. Plasma acylation-

FOR RESEARCH USE ONLY. NOT INTENDED FOR DIAGNOSTIC OR THERAPEUTIC USE.

Abbreviations:
mAb—Monoclonal Antibody **pAb**—Polyclonal Antibody **WB**—Western Blot **IF**—Immunofluorescence **ICC**—Immunocytochemistry **IHC**—Immunohistochemistry **E**—ELISA **Hu**—Human **Mo**—Monkey **Do**—Dog **Rt**—Rat **Ms**—Mouse **Bo**—Cow **Po**—Pig **Ho**—Horse **Ch**—Chicken **Dr**—*D. rerio* **Dm**—*D. melanogaster* **Ce**—*C. elegans* **Sc**—*S. cerevisiae* **Sa**—*S. aureus* **Ec**—*E. coli.*

(4,5,6). C3b is the main effector molecule of the complement system, expressing multiple binding sites for other complement components such as C5, properdin, factor B, factor H and certain membrane proteins such as 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 practice the level of (C3) in serum and CSF can be used to help identify immunological disorders, especially those associated with deficiencies of complement components.