

Complement

Monoclonal Antibodies: Murine Monoclonal Anti-Human iC3b (neo)

For Research Use Only. Not for use in Diagnostic Procedures.

Background

Under normal conditions, activation of either of the complement pathways leads to the formation of C3 convertase enzymes which cleave C3 into two fragments C3a, an anaphylatoxin, and C3b. The C3b fragment has many biologic functions¹ including promotion of phagocytosis and participation as a structural component in both the C3 and C5 convertase enzymes. These processes are under stringent control in vivo. One control mechanism involves a two-site cleavage of C3b by Factor I with the cooperation of Factor H or CR1 as cofactors. When cleaved in this way the biologic functions of C3b are lost. The resulting protein is termed iC3b. This fragment, in turn, has a host of new biologic activities.

iC3b fragments, either in fluid phase or bound to biological surfaces, can interact with a variety of cell types expressing complement receptors (either CR2 or CR3). iC3b levels in fluid phase are elevated in a variety of disease states including Systemic Lupus Erythematosus, Rheumatoid Arthritis as well as in a variety of pathologic conditions including sepsis and Myocardial Infarct.²

Quidel's monoclonal antibodies to complement antigens were prepared using standard techniques. They are purified from mouse ascites fluid via protein A affinity chromatography.³

The specificity of the monoclonal antibody was established via a series of immunological techniques including ELISA, hemagglutination and RIA. Firstly, the antibody was shown by ELISA to bind to C3 antigens using highly pure, immobilized C3. Subsequent studies showed that this antibody agglutinates EC3bi but not EC3b or EC3d cells in an indirect hemagglutination assay. Further experiments showed that this antibody bound to radio-labeled purified iC3b but not to similarly labeled C3, C3b, C3d, or C3c.

Applications

Please contact Quidel Specialty Products Technical Services for application specific information.

EIA ⁴	RIA	IHC ⁵	WB ⁶	FACS ^{3,7,8}
>1:5000	N/T	>1:1000	>1:5000	>1:25

N/T = Not tested.

Specifications

Catalog Number: A209
 Concentration: ≥1.0 mg/ml
 Purity: > 95% by SDS PAGE
 Volume/Vial: 100 µl
 Storage: Short term (30 days) 4 °C
 Long term at or below -20 °C
 Buffer: Borate Buffered Saline
 (pH 8.4 ± 0.2)
 Isotype: 1gG2bK

References

- Buyon, J.P., Tamerius J., Belmont H.M., Abramson S.B., Assessment of disease activity and impending lupus flare in patients with SLE. *Arthritis and Rheumatism* 35:1028-37, 1992.
- Rogers, J., Cooper, N., et al. Complement Activation by β-amyloid in Alzheimer disease. *PNAS* 89:10016-10020, 1992.
- Gemmell, C. A flow cytometric immunoassay to quantify adsorption of complement activation products on artificial surfaces. *J Biomed Mater Res* 37, 474-480, 1997.
- Grehan J.F., Levay-Young B.K., Fogelson J.L., Francois-Bongarcron V, Benson B.A., Dalmaso A.P., "IL-4 and IL-13 Induce Protection of Porcine Endothelial Cells From Killing by Human Complement and From Apoptosis Through Activation of a Phosphatidylinositide 3-Kinase/Akt Pathway" *J Immun* 175:1903-10, 2005.
- Salerno C.T., Kulick D.M., Yeh C.G., Guzman-Paz M, Higgins P.J., Benson B.A., Park S.J., Shumway S.J., Bolman R.M., Dalmaso A.P., "A Soluble Chimeric Inhibitor of C3 an C5 Convertases, Complement Activation Blocker-2, Prolongs Graft Survival in Pig-To-Rhesus Monkey Heart Transplantation" *Xenotransplantation* 9:125-34, 2002

6 Ferguson J.S., Weis J.J., Martin J.L., Schlesinger L.S., "Complement Protein C3 binding to *Mycobacterium tuberculosis* is Initiated by the Classical Pathway in Human Brochoalveolar Lavage Fluid" *Infect and Immun* 72(5):2564-73 2004.

7 Mevorach D, Mascarenhas J.O., Gershov D, Elkon K.B., "Complement-dependent Clearance of Apoptotic Cells by Human Macrophages" *J Exp Med* 188(12):2313-20, 1998.

8 Gershov D, Kim S, Brot N, Elkon K.B., "C-Reactive Protein Binds to Apoptotic Cells, Protects the Cells from Assembly of the Terminal Complement Components, and Sustains an Antiinflammatory Innate Immune Response: Implications for Systemic Autoimmunity" *J Exp Med* 192(9):1353-63, 2000.

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