3050 Spruce Street, St. Louis, MO 63103 USA
Tel: (800) 521-8956 (314) 771-5765 Fax: (800) 325-5052 (314) 771-5757
email: techservice@sial.com sigma-aldrich.com

Product Information

Complement Serum Standard
human

Catalog Number **C9473**
Storage Temperature –70 °C

Product Description

The complement system is a complex cascade involving proteolytic cleavage of serum glycoproteins. The cascade is often activated by cell receptors and ultimately results in induction of the inflammatory response, phagocyte chemotaxis and opsonization, and cell lysis.

Complement factors C3a, C5a, and C4 can induce vasodilatation, increased capillary permeability, and expression of leukocyte adhesion molecules. Complements C3a and C4b are opsonins that bridge phagocytes to microorganisms. Complements C3a and C4a promote phagocyte chemotaxis. Complement C3b may be an opsonin for antgen-antibody complexes which helps prevent damage from the formation of large, insoluble immune aggregates. Complement C5a, like C3a, is an anaphylatoxin and is a chemotactic attractant for induction of neutrophilic release of antimicrobial proteases and oxygen radicals. A complex of complements C5b, C6, C7, and C8 mediates the polymerization of up to eighteen C9 molecules into a tube-like membrane attack complex that is inserted into the plasma membrane of an unwanted organism such as Gram-negative bacteria and viral infected cells. This channel through the lipid bilayer results in lysis of the cell. Ischaemic infarction may also cause initiation of the complement cascade.

Other deleterious effects of complement activation include degranulation of neutrophils, basophils, and mast cells; unwanted release of the neutrophil products elastase and oxygen radicals; and extracorporeal blood circulation. Complement inhibitors are being studied as potential therapeutics for immune diseases and Alzheimer's.

Three pathways have been elucidated through which the complement cascade can be initiated; Classical, Alternate, and Lectin. All three pathways merge through at a common intersection, complement C3.

- The Classical Pathway mediates specific antibody responses. It is initiated by the binding of antibodies to cell surface antigens. Subsequent binding of the antibody to complement C1q subunits of C1 results in catalytically active C1s subunits. The two activated C1s subunits are then able to catalyze the assembly of the C3 convertase (complement C4b2a) from complements C2 and C4.

- In the Alternate Pathway the cascade is initiated by foreign cell surface components rather than the action of antibodies. In the alternate pathway complement C3 undergoes spontaneous cleavage resulting in complement B binding to C3b. Diffusion of the Ba subunit results in an active alternate pathway C3 convertase (C3bBb). C3bBb is stabilized by binding to properdin prior to merging into the common pathway and conversion of C3.

- The Lectin Pathway is similar to the classical pathway. C1q is not involved in the lectin pathway. Instead an opsonin, mannan-binding protein (MBP), is involved in the initiation process.

Component

Complement Serum Standard is supplied as a 0.22 μm filtered solution containing ~18 mM citrate, ~22 mM dextrose, ~3 mM phosphate and ~13 mM calcium.

Storage/Stability

The product ships on dry ice and storage at –70 °C is recommended. Repeated freezing and thawing is **not** recommended.

Precautions and Disclaimer
This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.
Note: Potential Biohazard – Handle as if capable of transmitting infectious agents.

References
1. Kolb, W. P., et al., J. Immunol., **122**, 2103 (1979).
2. Kabat, E. A., and Mayer, M. M., Experimental Immunochemistry, 2nd edition, p. 149, (Charles C. Thomas, Springfield, IL, 1961).
3. Platts-Mills, T. A., and Ishizaka, K., J., Immunol, **113**, 348 (1974).

CS,RBG,JWM,MAM 03/10-1