

Calbiochem®

The Complement System

Complement Reagents of the Highest Quality

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The complement system provides innate defense against microbial infection and is a “complement” to antibody mediated immunity. The complement system consists of thirty five interacting plasma and membrane associated proteins which contribute to host-defense and initiate and amplify inflammation, even in the preimmune state where specific antibodies and lymphocytes are not available. Contained in this system are several soluble factors that prevent spontaneous complement activation from occurring in solution, as well as, several regulatory membrane associated proteins that protect host cells from accidental complement attack.

Activation of the complement system is achieved through three major pathways:

the classical pathway, which is activated by certain isotypes of antibodies bound to antigens (immune complexes); the alternative pathway, which is activated on microbial cell surfaces in the absence of antibody; and the lectin pathway, which is activated by a plasma lectin (mannose binding lectin - MBL) that binds to mannose residues on microbes. Following the activation of complement pathways, several peptides are generated that help to increase the number of circulating leukocytes, promote leukocyte adhesion to vascular endothelium, and attract phagocytes to the site of infection.

The classical pathway is activated mainly when certain antibody molecules (IgM, IgG₁, IgG₂, and IgG₃) bind to a foreign particle. C1 binding to the Fc region of the antibody molecule initiates this pathway. C1, present in serum as a proenzyme, tends to undergo auto-activation, a process strictly controlled by C1 inhibitor. The C1 complex is formed by the calcium dependent interaction of C1q with C1r₂C1s₂. Initially, a conformational change in C1r occurs, followed by proteolytic activation, which results in the cleavage of all four polypeptide chains of C1r₂C1s₂. The two activated C1s subunits are then able to catalyze the assembly of the C3 convertase (C4b2a), which is formed from C2 and C4 (see figure).

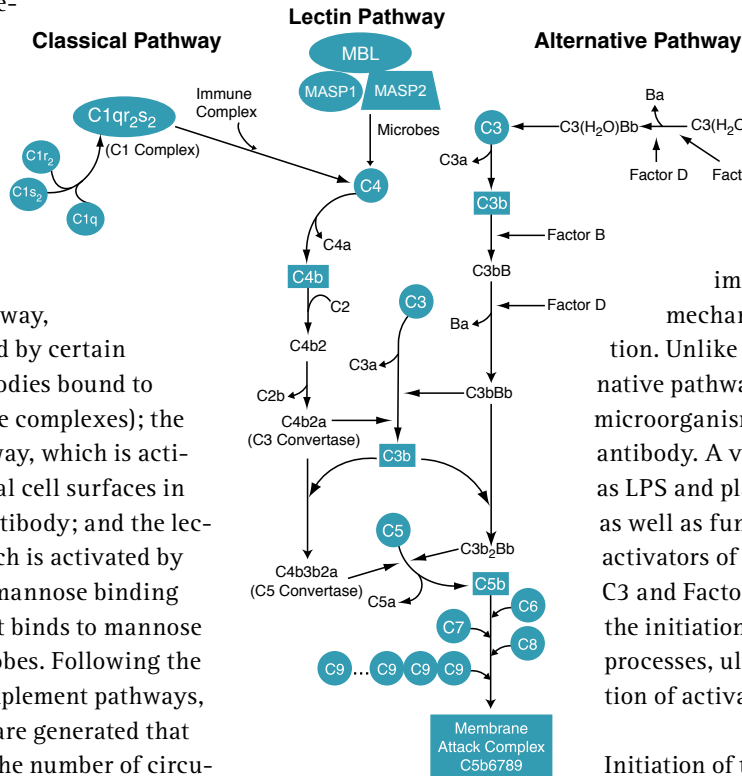
The C4b2a complex cleaves the C3 component. Its enzymatic site is located in the C2a fragment which has substrate specificity for C3. The C3 convertase is an unstable enzyme that undergoes a time and temperature-dependent decay. The C2a fragment of C4b2a is released, leaving the C4b site to take up new native C2 to form a new C4b2a enzyme.

Cleavage of the C3 component releases C3a and C3b. C3a appears to be important in many inflammatory responses. The C3b fragment covalently binds to the cell or

bacterial surface and plays a role in opsonisation. Binding of C3b to the C4b component of the C3 convertase, results in C5 convertase (C4b3b2a) formation.

The alternative pathway is important in host innate defense mechanisms against bacterial infection. Unlike the classical pathway, the alternative pathway can be activated by invading microorganisms in the absence of a specific antibody. A variety of polysaccharides, such as LPS and plant (inulin) polysaccharides, as well as fungi, bacteria, and viruses are activators of this pathway. In this pathway, C3 and Factors B, D, H, I, and P play a role in the initiation, recognition, and amplification processes, ultimately leading to the formation of activator-bound C3/C5 convertase.

Initiation of the alternative pathway occurs by spontaneous low-rate hydrolysis of the thioester in C3 and the resultant continuous supply of C3(H₂O) in solution. Binding of Factor B to C3(H₂O) in the presence of Factor D leads to the formation of the alternative pathway initiation C3 convertase (C3(H₂O)Bb) (see figure). The activity of the initiation C3 convertase is modulated by Factors H, I, and P. This initiation fluid phase C3 convertase can cleave C3 to release C3a and C3b, the same C3 fragments produced by the C3 convertase of the classical pathway. The C3b fragment covalently binds to cellular and microbial surfaces. If the cell or microbe surface is an activator, alternative pathway activation will proceed. If the C3b fragment covalently binds to a cell or microbe surface that is not an activator, alternative pathway activation will stop. Binding of Factor B to a C3b fragment covalently bound to an activator, in the presence of Factor D, will lead to the formation of an alter-



native pathway C3 convertase (C3bBb) on the activator surface. The enzymatic site that mediates C3 cleavage is located in the Bb molecule. Binding of an additional C3b to the C3 convertase leads to the formation of the C5 convertase (C3b₂Bb). Factor P (properdin) stabilizes both the C3 and C5 alternative pathway convertases from decay dissociation.

The lectin pathway begins when mannose-binding lectin (MBL) binds to membrane carbohydrates on microorganisms. Two proteins, MASP1 and MASP2 (equivalent to C1r and C1s of the classical pathway), are associated with MBL to form a complex enzyme similar to C1 of the classical pathway. Like C1, once activated, the MBL- MASP1- MASP2 complex is able to cleave C4 and C2 to form C4bC2a, the C3 convertase that is able to enzymatically split hundreds of molecules of C3 into C3a and C3b.

In all three activation pathways, the cleavage of C5 leads to the assembly of the C5b-9 membrane attack complex (MAC). The membrane attack complex is responsible for the membrane lytic events associated with complement activation. The C5 convertase cleaves the C5 component to generate C5a and C5b fragments. C5a serves as an inflammatory mediator that can activate a variety of cells through G-protein-linked receptors. The MAC is composed of one molecule each of C5b, C6, C7, and C8 and as many as 10 to 12 molecules of C9. When C5 is cleaved by C5 convertase, nascent C5b binds to C6 to

form a stable bimolecular complex that binds C7. Membrane-bound C5b67 commits MAC assembly to a membrane site. The binding of one C8 molecule to each C5b67 complex gives rise to small transmembrane channels that perturb the target membrane. Each membrane-bound C5b678 complex acts as a receptor for multiple numbers of C9 molecules. Binding of one molecule of C9 initiates a process of C9 oligomerization at the site of membrane attack. The MAC, once assembled on the cell membrane, creates transmembrane channels leading to osmotic lysis of the cell. The transmembrane channels formed vary in size depending on the number of C9 molecules incorporated into the channel structure. The formation of the MAC is controlled by S protein in serum, which prevents C9 polymerization and blocks the attachment of C5b67 to the cell surface. This protects cells adjacent to sites of complement activation from accidental attack.

References:

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 Cooper, N.R., and Muller-Eberhard, H.J. 1970. *J. Exp. Med.* 132, 775.
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Complement C1, Human

C1 is the first component of the classical complement pathway. It is a calcium-dependent complex of C1q, C1r, and C1s subcomponents which are present in the C1 complex at molar ratios of 1:2:2, respectively. Binding of C1 to classical pathway activators results in the conversion of the proenzyme C1s subcomponent to an active C1s enzyme. C1s enzyme cleaves both C4 and C2, resulting in formation of the C3 cleaving enzyme, C4bC2a, of the classical complement pathway.

Ref.: Cooper, N.R. 1985. *Adv. Immunol.* 37, 151; Ziccardi, R.J., and Cooper, N.R. 1977. *J. Immunol.* 118, 2047.

Cat. No. 204873 200 µg £ 151

Complement C1-Esterase Inhibitor, Human

Single-chain glycoprotein present in normal human serum at 180 µg/ml. Involved in regulation of complement, coagulation, fibrinolytic and contact (kinin-forming) systems of circulating blood plasma. The only known inhibitor of activated C1r and C1s complement enzymes. The presence of either hereditary or acquired deficiencies of C1 inhibitor produces susceptibility to recurrent attacks of angioedema. *Purity: >90% by SDS-PAGE.*

Ref.: Davis, A.E. 1988. *Annu. Rev. Immunol.* 6, 595.

Cat. No. 204883 1 mg £ 216

Complement C1q, Human

Glycoprotein composed of 18 polypeptide chains consisting of three non-identical subunits, A, B, and C, of M.W. 29,000, 26,000 and 19,000, respectively. Present in normal human serum at 70 µg/ml. Found in circulating blood plasma complexed with two C1r and two C1s molecules to form the first component of complement (C1). Activation of complement via the classical pathway is triggered by the binding of C1q to immune complexes containing IgG or IgM or to a variety of other activating substances, including C-reactive protein, retroviruses, and mitochondria. Subsequent to C1q binding, C1r and C1s are converted to proteolytic enzymes that are responsible for continuation of activation via the classical pathway. *Purity: ≥95% by SDS-PAGE.* M.W. 410,000.

Ref.: Loos, M., et al. 1980. *J. Immunol.* 124, 59; Kolb, W.P., et al. 1979. *J. Immunol.* 122, 2103.

Cat. No. 204876 1 mg £ 185

Complement C1r, Human, Activated, Two-Chain Form

C1r is present in normal human serum at 34 µg/ml. Non-activated C1r is found in circulating blood plasma as a dimer that associates with one C1q molecule and two C1s molecules to form the first component of complement (C1). Following C1q binding to the classical complement pathway activators, each C1r proenzyme monomer is activated by cleavage into two disulfide-linked fragments of M.W. 60,000 and 35,000. The 35,000 peptide contains the C1r enzymatic active site. Activated C1r continues activation via classical pathway by cleaving, and thus activating C1s. *Purity: ≥90% by SDS-PAGE.* M.W. 95,000.

Ref.: Villiers, C.L., et al. 1983. *Biochem. J.* 215, 369; Ziccardi, R.J., and Cooper, N.R. 1976. *J. Immunol.* 116, 496.

Cat. No. 204878 250 µg £ 184

Complement C1s, Human, Activated, Two-Chain Form

C1s is present in normal human serum at 30 µg/ml. Non-activated C1s is found in circulating blood plasma as a Ca²⁺-dependent dimer in association with one C1q molecule and two C1r molecules to form the first component of complement (C1). Following C1q binding to the classical complement pathway activators, each C1r protein is cleaved to form activated C1r enzyme. Dimeric C1r enzyme cleaves, and thus activates, each C1s molecule into two disulfide-linked fragments of M.W. 59,000 and 28,000. The 28 kDa peptide contains the C1s enzymatic active site. Activated C1s continues complement activation via the classical pathway by cleaving, and thus activating, C2 and C4. *Purity: single band by non-reducing SDS-PAGE; two bands by reducing SDS-PAGE.* M.W. 87,000.

Ref.: Cooper, N.R. 1985. *Adv. Immunol.* 37, 151; Sim, R.B. 1981. *Methods Enzymol.* 80, 6.

Cat. No. 204879 250 µg £ 184

Complement C2, Human

Single chain glycoprotein present in normal human serum at 20 µg/ml. Classical pathway activation results in assembly of the C1,C4b complex on target surfaces. Subsequent to binding of the C4b component to this complex, C2 is cleaved by the activated C1s subcomponent of C1 into C2a, the 63 kDa C-terminal fragment, and C2b, the 30 kDa N-terminal fragment. The smaller C2b fragment is released while the larger C2a fragment remains associated with the C1,C4b complex to form the C4b,C2a classical pathway C3 convertase enzyme. M.W. 93,000.

Cat. No. 204882 50 µg £ 206

Complement C3, Human

Glycoprotein composed of two non-identical disulfide-bonded subunits with M.W. of 115,000 (α) and 75,000 (β). Present in normal human serum at 1.25 mg/ml. Classical and alternative activation pathways of complement converge at the C3 step. Activation via either pathway can result in assembly of C3-cleaving enzymes (C3 convertases) on target surfaces. Both C3 convertases cleave the C3 α-chain at peptide bond 77 resulting in the production of C3a (M.W. 9083) and C3b fragments (M.W. 180,000). The C3a peptide, which is released, is one of the three complement derived anaphylatoxins. The nascent C3b fragment can form a covalent ester bond with a target surface. This covalent attachment of C3b to target acceptors is required for continuation of complement activation. *Purity: >95% by SDS-PAGE.* M.W. 190,000.

Ref.: Lambris, J.D., and Muller-Eberhard, H.J. 1986. *Mol. Immunol.* 23, 1237; Tack, B.F., et al. 1980. *Methods Enzymol.* 80, 64.

Cat. No. 204885 250 µg £ 141

Complement C3a, Human

Single chain, 77-amino acid peptide. Activation of either complement pathway can result in formation of C3-cleaving enzymes (C3 convertases) on target surfaces. The C3 convertase enzymes assembled by either pathway cleaves the C3 α-chain at peptide bond 77 resulting in the production and release of the C3a peptide. C3a, one of the three complement-derived anaphylatoxins, expresses a wide variety of biological activities including smooth muscle contraction, platelet and neutrophil activation and aggregation, skin wheal and flare, and immunoregulatory reactions. *Purity: ≥95% by SDS-PAGE.* M.W. 9,083.

Ref.: Hugli, T.E. 1989. *Curr. Top. Micro. Immunol.* 153, 181; Hugli, T.E. 1986. *Complement* 3, 111.

Cat. No. 204881 50 µg £ 166

Complement C3a des-Arg, Human

Single chain, 76-amino acid peptide. Once the C3a peptide has been produced in human plasma or serum, it is rapidly converted to C3a des-Arg by removal of the C-terminal arginine by endogenous serum carboxypeptidase N. This enzymatic process is considered to be a major mechanism for controlling C3a function in vivo because C3a des-Arg has <1% of the biological activity expressed by the C3a peptide. Therefore, C3a des-Arg can serve as a negative control molecule in experiments involved with the biological activities of the intact C3a anaphylatoxin peptide. *Purity: ≥95% by SDS-PAGE.* M.W. 9,000.

Ref.: Kajita, T., and Hugli, T.E. 1991. *Am. J. Pathol.* 138, 1359; Bokish, V.A., and Muller-Eberhard, H.J. 1970. *J. Clin. Invest.* 49, 2427.

Cat. No. 204884 50 µg £ 146

Complement C3b, Human

Cleavage of the C3 α-chain at peptide bond 77 by either of the complement C3 convertase enzymes results in production of C3a (M.W. 9083) and C3b (M.W. 180,000) fragments. The C3b fragment is a glycoprotein composed of the modified C3 α-chain (α') (M.W. 105,000) and the intact C3 β-chain (M.W. 75,000). Nascent C3b has the transient ability to form a covalent ester bond with a variety of target surfaces. Once bound to target surfaces, C3b becomes an essential subunit of both the classical and alternative pathway C5-cleaving enzymes. In addition, surface-bound C3b has opsonic and immune adherence activities which are mediated via binding to CR1 (CD35) complement receptors. *Purity: ≥90% by SDS-PAGE.*

Ref.: Wong, W.W., and Fearon, D.T. 1987. *Methods Enzymol.* 150, 579; Arnout, M.A., et al. 1981. *J. Immunol.* 127, 1348.

Cat. No. 204860 250 µg £ 193

Complement iC3b, Human

iC3b is formed by the cleavage of C3b by Factor I in the presence of Factor H, CR1, or membrane cofactor protein. Factor I cleavage of C3b to iC3b inactivates and prevents C3b from functioning in the C3 or C5 convertase enzymes. The iC3b fragment thus produced is a glycoprotein composed of two C3α' polypeptides of M.W. 43,000 and M.W. 63,000 which are disulfide bonded to the intact C3 β-chain (M.W. 75,000). iC3b interaction with CR3 (CD11b/CD18) receptors present on a variety of white blood cells greatly enhances phagocytosis of iC3b-coated target cells or particles. *Purity: ≥90% by SDS-PAGE.*

Ref.: Rosen, H., and Law, S.K.A. 1989. *Curr. Top. Microbiol. Immunol.* 153, 99; Ross, G.S., and Medof, M.E. 1985. *Adv. Immunol.* 37, 217.

Cat. No. 204863 250 µg £ 227

Complement C3c, Human

C3c is formed by the cleavage of iC3b by Factor I in the presence of CR1 (CD35), or by limited digestion with trypsin or elastin. The C3c fragment is a glycoprotein composed of two C3α' polypeptides of M.W. 43,000 and M.W. 27,000 which are disulfide bonded to the intact C3 β-chain (M.W. 75,000). *Purity: ≥85% by SDS-PAGE.*

Ref.: Yoon, S.E., and Fearon, D.T. 1985. *J. Immunol.* 134, 3332; Davis, A.E., et al. 1984. *J. Immunol.* 132, 1960.

Cat. No. 204866 250 µg £ 227

Complement C3d, Human

C3d is formed by the limited digestion of iC3b by trypsin or elastin. The C3d region of C3 contains the portion of the native C3 molecule which is capable of forming a covalent bond attachment to target surfaces under attack by complement. The binding of C3d to CR2 (CD21) receptors present on B-lymphocytes markedly enhances B cell activation initiated by a wide variety of stimuli. *Purity: ≥85% by SDS-PAGE.* M.W. 30,000.

Ref.: Luxembourg, A.T., and Cooper, N.R. 1994. *J. Immunol.* 153, 4448; Lambris, J.D., et al. 1985. *Proc. Natl. Acad. Sci. USA* 82, 4235.

Cat. No. 204870 100 µg £ 227

Complement C4, Human

Glycoprotein composed of three non-identical subunits of M.W. 93,000 (α), 75,000 (β), and 32,000 (γ) linked by disulfide bonds. Present in normal human serum at 400 µg/ml. On activation of complement via the classical pathway, the C1s subcomponent of the C1 complex is converted to an active serine protease that cleaves the C4 α-chain at peptide bond 77, resulting in production of C4a (M.W. 8758) and C4b fragments (M.W. 193,000). The released C4a peptide is one of the three complement-derived anaphylatoxins. The nascent C4b fragment can form a covalent ester bond with target surfaces. This covalent attachment of C4b to target acceptors is required for continuation of complement activation via classical pathway. *Purity: ≥95% by SDS-PAGE.* M.W. 200,000.

Ref.: Janatova, J. 1983. *Ann. N.Y. Acad. Sci.* 421, 218; Hammer, C.H., et al. 1981. *J. Biol. Chem.* 256, 3995.

Cat. No. 204886 250 µg £ 213

Complement C4a, Human

A single chain peptide composed of 77 amino acids. Activation of the classical complement pathway results in the conversion of the C1 complex to an active enzyme. The activated C1 enzyme cleaves the C4 α-chain at peptide bond 77 resulting in the production and release of the C4a peptide. C4a is one of the three complement-derived anaphylatoxins. *Purity: ≥95% by SDS-PAGE.* M.W. 8758.

Ref.: Murakami, Y., et al. 1993. *Immunol. Lett.* 36, 301; Gorski, J.P., et al. 1981. *J. Biol. Chem.* 256, 2707

Cat. No. 204887 50 µg £ 146

Complement C4b, Human

Cleavage of the C4 α-chain at peptide bond 77 by activated C1s enzyme results in the production of C4a (M.W. 8,758) and C4b (M.W. 193,000) fragments. The C4b fragment is a glycoprotein composed of the modified C4 α-chain (α') and intact β- and γ-chains. Like C3b, C4b has the transient ability to form a covalent ester bond with a variety of target cell surfaces. Once bound to the target surface, C4b becomes an essential non-enzymatic subunit of the classical pathway C3-cleaving enzyme (C4b,C2a). In addition, surface bound C4b has opsonic and immune adherence activities which are mediated via binding to the CR1 (CD35) complement receptor which is found on a variety of inflammatory cells. *Purity: ≥ 95% by SDS-PAGE.*

Ref.: Weisman, H.F., et al. 1990. *Science* 249, 146; Holers, M.V., et al. 1985. *Immunology Today* 6, 188.

Cat. No. 204897 250 µg £ 186

Complement C5, Human

Glycoprotein composed of two non-identical subunits of M.W. 120,000 (α) and 75,000 (β) linked by disulfide bonds. Present in normal human serum at 70 µg/ml. Activation of complement via either the classical or alternative pathway can result in the assembly of C5-cleaving enzymes (C5 convertases) on target surfaces. Both C5 convertases cleave the C5 α-chain at peptide bond 74 resulting in production of C5a (M.W. 11,200) and C5b fragments (M.W. 185,000). Released C5a peptide is one of the three complement-derived anaphylatoxins. C5b fragment combines with C6, C7, C8, and C9 to form lytic C5b-9 complement membrane attack complex (MAC). *Purity: ≥95% by SDS-PAGE.* M.W. 195,000.

Ref.: Janatova, J. 1988. *Methods Enzymol.* 162, 579; Wetsel, R.A., et al. 1980. *J. Immunol. Methods* 35, 319.

Cat. No. 204888 250 µg £ 223

NEW! Complement C5a, Human, Recombinant

Single chain glycopeptide composed of 74 amino acids. Activation of complement by either pathway results in the formation of C5-cleaving enzymes (C5 convertases) on target surfaces. The C5 convertase enzymes cleave the C5 α-chain at peptide bond 74 resulting in the production and release of the C5a glycopeptide. C5a, on a mole/mole basis, is the most biologically active of the three complement-derived anaphylatoxins. C5a expresses a wide variety of biological activities which include: inflammatory cell chemotaxis, smooth muscle contraction, activation and release reactions of neutrophils, mast cells and macrophages. Biological activity: Similar to that of serum derived C5a based on the myeloperoxidase assay. *Purity: ≥95% by SDS-PAGE.*

Ref.: Coney, D.F., and Hugli, T.E. 1993. *Protein Sci.* 2, 1391.

Cat. No. 234397 100 µg £ 298

Complement C5a des-Arg, Human

Single chain glycopeptide composed of 73 amino acids. Once C5a has been produced in human plasma or serum, it is rapidly converted to C5a des-Arg upon removal of the C-terminal arginine by the endogenous serum carboxypeptidase N. Unlike the C3a des-Arg peptide, C5a des-Arg retains significant levels of its biological activities. Thus, C5a des-Arg has been shown to exhibit inflammatory cell chemotaxis, smooth muscle contraction and leukotriene release from guinea pig lung. *Purity: ≥95% by SDS-PAGE.* M.W. 11,100.

Ref.: Webster, R.O., et al. 1980. *Immunopharmacology* 1, 201; McCarthy, K., and Henson, P.M. 1979. *J. Immunol.* 123, 2511.

Cat. No. 204902 30 µg £ 185

Complement C5b,6 Complex, Human

Activation of complement by either pathway results in the formation of C5-cleaving enzymes (C5 convertases) on target surfaces. The C5 convertase enzymes cleave the C5 α-chain at peptide bond 74, resulting in the formation of the C5a anaphylatoxin (M.W. 11,200) and the C5b fragment (M.W. 185,000). The nascent C5b fragment can combine with C6, C7, C8, and C9 to form the lytic C5b-9 membrane attack complex (MAC) on target surfaces. When C5 activation occurs in the presence of C6 only, e.g. in C7-deficient serum, a stable C5b,6 complex is formed. In a process termed Reactive Lysis, the (MAC) can be assembled on target cell surfaces in the absence of any additional complement proteins by incubating target cells with the C5b,6 complex and purified C7, C8, and C9. *Purity: ≥95% by SDS-PAGE.* M.W. 312,000.

Ref.: Podack, E.R., et al. 1978. *J. Immunol.* 120, 1841; Lachmann, P.J., and Thompson, R.A. 1970. *J. Exp. Med.* 131, 643.

Cat. No. 204906 50 µg £ 186

Complement C6, Human

Single-chain glycoprotein present in normal human serum at 64 µg/ml. On activation of complement via either the classical or alternative pathway, C5 is cleaved into C5a and C5b fragments. The C6 binds to the nascent C5b fragment, resulting in formation of the C5b,6 complex. Water-soluble C5b,6 complex combines with C7, C8 and C9 to form lytic C5b-9 complement membrane attack complex (MAC). *Purity:* >85% by SDS-PAGE. M.W. 128,000.

Ref.: Haefliger, J.A., et al. 1989. *J. Biol. Chem.* 264, 18041; Kolb, W.P., et al. 1982. *Biochemistry* 21, 294.

Cat. No. 204890 250 µg £ 206

Complement C7, Human

Single-chain glycoprotein present in normal human serum at 56 µg/ml. On activation of complement via either the classical or alternative pathway, C5 is cleaved into C5a and C5b fragments. The C6 and C7 bind to the nascent C5b fragment, resulting in formation of the C5b-7 complex. Membrane-bound, hydrophobic C5b-7 complex combines with C8 and C9 to form the lytic C5b-9 complement membrane attack complex (MAC). *Purity:* ≥95% by SDS-PAGE. M.W. 121,000.

Ref.: Preissner, K.T., et al. 1985. *J. Immunol.* 135, 445; DiScipio, R.G., and Gagnon, J. 1982. *Mol. Immunol.* 19, 1425.

Cat. No. 204892 250 µg £ 206

Complement C8, Human

Glycoprotein composed of three non-identical subunits of M.W. 64,000 (α), 64,000 (β) and 22,000 (γ). Present in normal human serum at 55 µg/ml. On activation of complement via either the classical or alternative pathway, C5 is cleaved into C5a and C5b fragments. The nascent C5b fragment binds to C6, C7 and C8 resulting in formation of the C5b-8 complex on target surfaces. Each membrane-bound, hydrophobic C5b-8 complex combines with six to twelve C9 molecules to complete the assembly of the lytic C5b-9 complement membrane attack complex (MAC). *Purity:* ≥95% by SDS-PAGE. M.W. 150,000.

Ref.: Steckel, E.W., et al. 1980. *J. Biol. Chem.* 255, 11997; Petersen, B.H., et al. 1976. *J. Clin. Invest.* 57, 283

Cat. No. 204896 250 µg £ 214

Complement C9, Human

Single-chain glycoprotein present in normal human serum at 60 µg/ml. On activation of complement via either the classical or alternative pathway, formation of the C5b fragment initiates assembly of the C5b-9 complement membrane attack complex (MAC) on target surfaces. Full lytic activity of MAC occurs only after binding of six to twelve C9 molecules to each C5b-8 complex. *Purity:* ≥95% by SDS-PAGE. M.W. 71,000.

Ref.: Stanley, K.K., et al. 1985. *EMBO J.* 4, 375; Biesecker, G., and Muller-Eberhard, H.J. 1980. *J. Immunol.* 124, 1291.

Cat. No. 204910 250 µg £ 206

Cobra Venom Factor, *Naja naja kaouthia*

(Cobra venom anticomplementary protein)

Glycoprotein composed of three non-identical disulfide-bonded subunits with M.W. of 68,000 (α), 48,000 (β), and 30,000 (γ). Cobra venom factor (CVF) is a structural and functional analog of cobra, as well as mammalian, C3. Thus, in the presence of Factor B, Factor D and Mg²⁺, CVF can form a stable CVF,Bb complex which is a C3/C5 convertase enzyme, however, the CVF,Bb complex is not susceptible to regulation by Factors H and I. Four to six µg of purified CVF are equal to 1.0 unit of functional activity as measured by the method of Cochrane, C.G., et al. *Purity:* ≥95% by SDS-PAGE. Not available for sale outside of the United States.

Ref.: Fritzing, D.C., et al. 1992. *J. Immunol.* 149, 3554; Vogel, C.W., and Muller-Eberhard, H.J. 1984. *J. Immunol. Methods* 73, 203; Cochrane, C.G., et al. 1970. *J. Immunol.* 105, 55.

Cat. No. 233552 1 mg N/A

Factor B, Human

(C3 Proactivator)

Single-chain glycoprotein present in normal human serum at about 200 µg/ml. During activation of complement via the alternative pathway, Factor B is cleaved and thus activated by Factor D into two fragments of M.W. of 30 kDa (Ba) and 63 kDa (Bb). The Bb fragment contains the serine protease enzymatic active site. The activated Bb fragment continues complement activation via the alternative pathway by cleaving, and thus activating, C3 and C5. *Purity:* ≥90% by SDS-PAGE. M.W. 93,000.

Ref.: Kam, C.M., et al. 1987. *J. Biol. Chem.* 262, 3444; Gotze, O., and Muller-Eberhard, H.J. 1971. *J. Exp. Med.* 134, 90s.

Cat. No. 341262 250 µg £ 181

Factor D, Human

(Adipsin; C3 Proactivator Convertase)

Single-chain glycoprotein composed of 222 amino acids, present in normal human serum at about 1.4 µg/ml. Serine protease that cleaves, and thus activates, Factor B during activation of complement via the alternative pathway. Has recently been shown to be identical to adipsin, a serine protease secreted into the bloodstream by adipocytes. *Purity:* ≥95% by SDS-PAGE. M.W. 24,000.

Ref.: Volankis, J.E., et al. 1985. *N. Engl. J. Med.* 312, 395; Niemann, M.A., et al. 1984. *J. Immunol.* 132, 809.

Cat. No. 341273 25 µg £ 216

Factor H, Human

(β_{1H} Globulin)

Single-chain glycoprotein present in normal human serum at about 500 µg/ml. Regulates formation and function of complement C3 and C5 convertase enzymes. A C3b-binding protein, not an enzyme. Regulatory activity is attributed to its ability to recognize and bind to C3b fragments. *Purity:* ≥95% by SDS-PAGE. M.W. 150,000.

Ref.: Pangburn, M.K., and Muller-Eberhard, H.J. 1984. *Springer Semin. Immunopathol.* 7, 163; Fearon, D.T., and Austen, K.F. 1977. *Proc. Natl. Acad. Sci. USA* 74, 1683

Cat. No. 341274 250 µg £ 206

Factor I, Human

(C3b/C4b Inactivator)

Glycoprotein composed of two non-identical disulfide-bonded subunits with M.W. of 50 kDa (a) and 38 kDa (b). Present in normal human serum at about 34 µg/ml. A serine protease that requires protein cofactor in order to effect substrate cleavage. Thus, either Factor H a complement receptor CR1 confunction of the cofactor required for Factor I-mediated cleavage of C3b, C4-binding protein or complement receptor CR1 can function as the cofactor required for Factor I-mediated cleavage of C4b. *Purity: ≥95% by SDS-PAGE.* M.W. 88,000.

Ref.: Pangburn, M.K., et al. 1977. *J. Exp. Med.* 146, 257; Solal-Celigny, P., et al. 1982. *Clin. Exp. Immunol.* 47, 197.

Cat. No. 341280

250 µg

£ 353

Factor P, Human

(Properdin)

Glycoprotein found in circulating blood plasma in dimeric, trimeric, and tetrameric forms with M.W. of 92 kDa, 138 kDa, and 184 kDa, respectively. Present in normal human serum at about 20 µg/ml. Regulatory protein of the alternative pathway of complement activation. Accelerates complement activation by binding to and stabilizing the alternative pathway C3 and C5 convertase enzymes. *Purity: >90% by SDS-PAGE.* M.W. 220,000.

Ref.: Pangburn, M.K., et al. 1988. *Methods Enzymol.* 162, 639; Smith, C.A., et al. 1984. *J. Biol. Chem.* 259, 4582.

Cat. No. 341283

250 µg

£ 353

Antibodies to Complement Components

Product	Cat. No.	Size	Price £
Anti-Complement C1-Esterase Inhibitor, Human (Goat)	234363	2 ml	115
Anti-Complement C1q, Human (Goat)	234390	1 ml	60
Anti-Complement C2, Human (Goat)	234365	1 ml	149
Anti-Complement C3, Human (Goat)	204869	1 ml	65
Anti-Complement C3a, Human (Rabbit)	204859	1 ml	107
Anti-Complement C4, Human (Goat)	204894	1 ml	65
Anti-Complement C5, Human (Rabbit)	800311	0.5 ml	83
Anti-Complement C5a, Human (Rabbit)	204889	1 ml	176

Product	Cat. No.	Size	Price £
Anti-Complement C5b-9, Human (Rabbit)	204903	1 mg	177
Anti-Complement C6, Human (Goat)	204891	1 ml	65
Anti-Complement C8, Human (Goat)	204904	1 ml	65
Anti-Complement C8, Human (Rabbit)	204898	1 ml	123
Anti-Complement C9, Human (Goat)	204912	1 ml	65
Anti-Factor B, Human (Goat)	341272	2 ml	110
Anti-Factor H, Human (Goat)	341276	1 ml	65

Complement-Depleted Sera

Product	Cat. No.	Size	Price £
Complement C1q-Depleted Human Serum	234401	1 ml	106
Complement C2-Depleted Human Serum	234402	1 ml	101
Complement C3-Depleted Human Serum	234403	1 ml	138
Complement C4-Deficient Guinea Pig Serum	234404	1 ml	109
Complement C5-Depleted Human Serum	234405	1 ml	101

Product	Cat. No.	Size	Price £
Complement C6-Depleted Human Serum	234406	1 ml	101
Complement C7-Depleted Human Serum	234407	1 ml	101
Complement C8-Depleted Human Serum	234408	1 ml	101
Complement C9-Depleted Human Serum	234409	1 ml	101
Factor B-Depleted Human Serum	234410	1 ml	117

All complement compon



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