

COMPLEMENT

The complement system, consisting of about thirty interacting plasma proteins, plays an essential role in host defense mechanisms against infectious agents and in the inflammatory process. Contained in this system are several regulatory membrane proteins that prevent autologous complement activation and protect host cells from accidental complement attack.

The activation of the complement system is achieved through either the classical pathway or the alternative pathway. The classical pathway mediates specific antibody response and is activated mainly when certain antibody molecules (IgM, IgG₁, IgG₂, and IgG₃) bind to a foreign particle. The binding of the Fc region of the antibody molecule to the C1 component initiates this pathway. C1, present in serum as a proenzyme, tends to undergo auto-activation, a process strictly controlled by C1 inhibitor. The C1 component complex is formed by the reversible interaction of C1q and C1r₂s₂. Initially, a conformational change in C1r occurs, followed by proteolytic activation, which results in the cleavage of all four polypeptide chains of C1r₂s₂. 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).

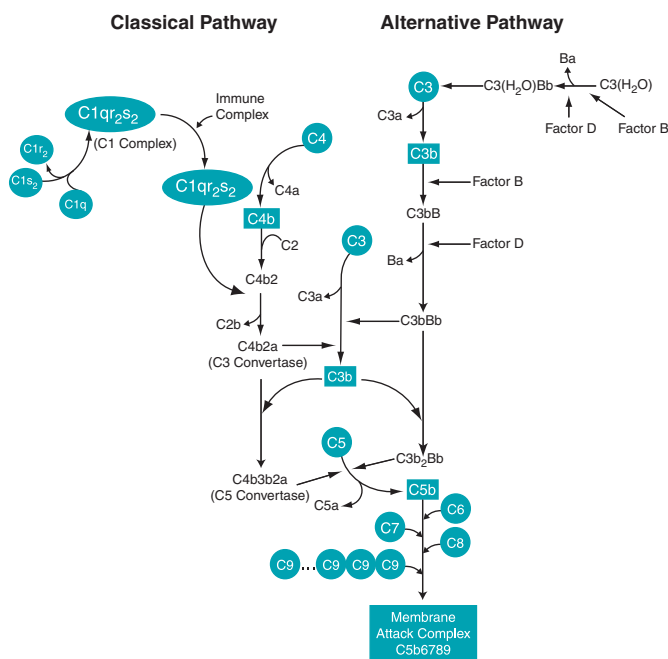
The C4b2a complex cleaves the C3 component. Its enzymatic site is located in the C2a molecule and has substrate specificity for C3. The C3 convertase is an unstable enzyme that undergoes a time and temperature-dependent decay. The C2a fragment of C4b2a is released, leaving the C4b site to take up new 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 subunit covalently binds to the cell or bacterial surface and plays a role in opsonisation. Following the binding of the C3b component to the C4b component, the C3 convertase (C4b2a) becomes C5 convertase (C4b3b2a).

The alternative pathway is important in the host defense mechanism against bacterial infection. Unlike the classical pathway, the alternative pathway is activated by invading microorganisms and is not necessarily an antibody-dependent process. A variety of polysaccharides, such as LPS and plant (inulin) polysaccharides, as well as fungi, bacteria, and viruses can activate 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 the activator-bound C3/C5 convertase.

Initiation of the alternative pathway is shielded by spontaneous low-rate hydrolysis of the thioester in C3 and the resultant continuous supply of C3(H₂O). Binding of Factor B to C3(H₂O) leads to the activation of the proenzyme complex [C3bB(Mg)] by factor D. This results in cleavage of Factor B and release of Ba (30 kDa fragment) and the formation of initial C3 convertase. The activity of initial C3 convertase is modulated by properdin and Factors H and I. Properdin binds to the cell-bound C3b and stabilizes C3/C5 convertase. The activated C3 convertase is covalently linked to the surface of target cells through C3b.

References:

- Polley, M.J., and Muller-Eberhard, H.J. 1966. *Prog. Hematol.* **5**, 2.
 Cooper, N.R., and Muller-Eberhard, H.J. 1970. *J. Exp. Med.* **132**, 775.
 Polley, M.J., et al. 1971. *J. Exp. Med.* **133**, 53.
 Fearon, D.T., and Austen, K.F. 1975. *J. Exp. Med.* **142**, 856.
 Hu, V.W., et al. 1981. *J. Immunol.* **127**, 380.
 Pangburn, M.K., et al. 1981. *J. Exp. Med.* **154**, 856.



In the alternative pathway, C3 convertase can function as a C5 convertase provided that an additional C3b molecule is available in close proximity.

In both activation pathways, the cleavage and activation of C5 leads to the assembly of the membrane attack complex (MAC). The C5 convertase cleaves the C5 component to generate C5a and C5b fragments. C5a serves as an inflammatory mediator that can act on target cells through G-protein-linked receptors. C5b is essential in initiating the lytic sequence of reactions. The MAC is composed of approximately twenty protein molecules with a combined molecular weight of about 1.7 million. It consists of one molecule each of C5b, C6, C7, and C8 and one or more molecules of C9. When C5 is cleaved by C5 convertase, nascent C5b is produced followed by self-assembly of the MAC. Nascent C5b and C6 form a stable bimolecular complex that binds to 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 may perturb the target. 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 membrane attack site. The MAC, once inserted into the cell membranes, creates complete 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.

- Ziccardi, R.J. 1982. *J. Immunol.* **128**, 2500.
 Lachmann, P., et al. 1984. *Springer Semin. Immunopathol.* **7**, 143.
 Muller-Eberhard, H.J. 1988. *Annu. Rev. Biochem.* **57**, 321.
 Niculescu, F., et al. 1994. *J. Biol. Chem.* **269**, 4417.
 Rivas, G., et al. 1994. *Biochemistry* **33**, 2341.

COMPLEMENT COMPONENTS

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, C4b,C2a, 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

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

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

C1r, Human, Activated, Two-Chain Form

Non-activated form composed of a single-chain glycoprotein. 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

C1s, Human, Activated, Two-Chain Form

Non-activated form composed of a single-chain glycoprotein. 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

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

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

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

COMPLEMENT COMPONENTS, CONTINUED

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

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

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

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

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

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

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

C4b, Human

Cleavage of the C4 α-chain at peptide bond 77 by activated C1 enzyme results in the production of C4a (M.W. 8758) 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

COMPLEMENT COMPONENTS, CONTINUED

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 $\mu\text{g}/\text{ml}$. 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: $\geq 95\%$ 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

C5a, Human

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, skin wheel and flare, and immunoregulatory reactions. Purity: $\geq 90\%$ by SDS-PAGE. M.W. 11,200.

Ref.: Hugli, T.E. 1989. *Curr. Top. Micro. Immunol.* **153**, 181; Hugli, T.E. 1981. *Mol. Cell. Biochem.* **41**, 59.

Cat. No. 204900

50 μg

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: $\geq 95\%$ 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

50 μg

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: $\geq 95\%$ 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

C6, Human

Single-chain glycoprotein present in normal human serum at 64 $\mu\text{g}/\text{ml}$. 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

C7, Human

Single-chain glycoprotein present in normal human serum at 56 $\mu\text{g}/\text{ml}$. 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: $\geq 95\%$ 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

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 $\mu\text{g}/\text{ml}$. 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 three to six C9 molecules to complete the assembly of the lytic C5b-9 complement membrane attack complex (MAC). Purity: $\geq 95\%$ 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

100 μg , 250 μg

C9, Human

Single-chain glycoprotein present in normal human serum at 60 $\mu\text{g}/\text{ml}$. On activation of complement via either the classical or alternative pathway, formation of the C5b fragment initiates assembly of the C5b-9m complement membrane attack complex (MAC) on target surfaces. Full lytic activity of MAC occurs only after binding of three to six C9 molecules to each C5b-8 complex. Purity: $\geq 95\%$ 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

COMPLEMENT COMPONENTS, CONTINUED

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 Mg^{2+} , 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 μ g of purified CVF are equal to 1.0 unit of functional activity as measured by the method of Cochrane, C.G., et al. Purity: $\geq 95\%$ by SDS-PAGE. Not available for sale outside of the United States.

Ref.: Fritzingler, 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

Factor B, Human

(C3 Proactivator)

Single-chain glycoprotein present in normal human serum at about 200 μ g/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: $\geq 90\%$ 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 μ g

Factor D, Human

(C3 Proactivator Convertase)

Single-chain glycoprotein composed of 222 amino acids, present in normal human serum at about 1.4 μ g/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 adipisin, a serine protease secreted into the bloodstream by adipocytes. Purity: $\geq 95\%$ 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 μ g

Factor H, Human

($\beta 1_H$ Globulin)

Single-chain glycoprotein present in normal human serum at about 500 μ g/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: $\geq 95\%$ 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 μ g

Factor I, Human

(C3b/C4b Inactivator)

Glycoprotein composed of two non-identical disulfide-bonded subunits of M.W. of 50 kDa (α) and 38 kDa (β). Present in normal human serum at about 34 μ g/ml. A serine protease that requires protein cofactor in order to effect substrate cleavage. Thus, either Factor H or Factor I mediates cleavage of C3b. C4-binding protein or complement receptor CR1 can function as the cofactor required for Factor I-mediated cleavage of C4b. Purity: $\geq 95\%$ 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 μ g

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 μ g/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 μ g

COMPLEMENT COMPONENTS SERA

Complement, Guinea Pig Serum

Lyophilized solid. Suitable for evaluating functional activity of human complement components and for other research requiring a high level of hemolytic activity.

Cat. No. 234395

5 ml

Complement, Rabbit Serum

Lyophilized solid. Tested for suitability for HLA-ABC serology. Total lympholytic complement activity determined under conditions of NIH two-step cytotoxicity test that uses defined HLA isoantisera. Activity present at a titer of at least 1:4. No intrinsic cytotoxicity against human peripheral blood lymphocytes. Intended for micro and macro lymphocyte cytotoxicity testing with human or animal sera. Suitable as an active source of complement for use with hybridoma antibodies, alloantisera and related applications in which a non-cytotoxic source is required.

Cat. No. 234400

5 ml

ANTIBODIES TO COMPLEMENT COMPONENTS

Product	Cat. No.	Size
Anti-C1-Esterase Inhibitor, Human (Goat)	234363	1 ml
Anti-C1q, Human (Goat)	234390	1 ml
Anti-C1s, Human (Goat)	234359	1 ml
Anti-C2, Human (Goat)	234365	1 ml
Anti-C3, Human (Goat)	204869	1 ml
Anti-C3a, Human (Rabbit)	204859	1 ml
Anti-C4, Human (Goat)	204894	1 ml
Anti-C4 Binding Protein, Human (Rabbit)	234380	1 ml
Anti-C5, Human (Rabbit)	800311	1 ml
Anti-C5a, Human (Rabbit)	204889	1 ml

Product	Cat. No.	Size
Anti-C5b-9, Human (Rabbit)	204903	1 mg
Anti-C6, Human (Goat)	204891	1 ml
Anti-C7, Human (Goat)	204893	1 ml
Anti-C8, Human (Goat)	204904	1 ml
Anti-C8, Human (Rabbit)	204898	1 ml
Anti-C9, Human (Goat)	204912	1 ml
Anti-Factor B, Human (Goat)	341272	2 ml
Anti-Factor H, Human (Goat)	341276	1 ml
Anti-Factor I, Human (Goat)	341277	1 ml

COMPLEMENT-DEPLETED SERA

Product	Cat. No.	Size
C1q-Depleted Human Serum	234401	1 ml
C2-Depleted Human Serum	234402	1 ml
C3-Depleted Human Serum	234403	1 ml
C4-Deficient Guinea Pig Serum	234404	1 ml
C5-Depleted Human Serum	234405	1 ml

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

MAJOR PLASMA PROTEINS INVOLVED IN CONTROL OF THE COMPLEMENT SYSTEM

Protein	Specificity	Function
C1 Inhibitor	<i>C1r, C1s</i>	<i>Forms 1:1 covalent complex with both C1r and C1s and removes them from the complex.</i>
Factor H	<i>C3b</i>	<i>Acts as a cofactor in the cleavage of C3b by Factor I.</i>
Factor I	<i>C3b, C4b</i>	<i>Protease that inactivates C3b and C4b.</i>
Factor P	<i>C3bBb</i>	<i>Positive regulator of the alternative pathway. Stabilizes C3/C5 convertases.</i>

Please call our Technical Service Department or your local sales office for more information on these products.

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