

**Antigen** 

Clone



International Blood Group Reference Laboratory

500 North Bristol Park

Northway Filton Bristol

BGRL 16

Complement C3c

BS34 7QH

Product Code 9496

Protein Development and Production Unit Tel: +44 (0)117 921 7500

Immunoglobulin Class Mouse Ig1, kappa light chain

Fax: +44 (0)117 912 5796

**Website:** http://ibgrl.blood.co.uk **Email:** enquiries.IBGRL@nhsbt.nhs.uk

## **Antigen Description and Distribution**

Complement is the name given to a complex series of some 20 proteins which, along with blood clotting, fibrinolysis and kinin formation, forms one of the triggered enzyme systems found in plasma. These systems characteristically produce a rapid, highly amplified response to a trigger stimulus mediated by a cascade phenomenon where the product of one reaction is the enzymic catalyst of the next. The most abundant complement component is C3, which has molecular weight of 195kDa and is present in plasma at a concentration of around 1.2mg/ml. C3 plays a central role in both classical and alternative complement activation pathways. The C3c component is generated over the course of complement activation, where convertase C4b2a (classical pathway) and convertase C3bBb (alternative pathway) cleave C3 to C3b which is further degraded into C3c. When erythrocytes are treated with a complement binding antibody such as anti-Lea C3c, C4 and C3d can be demonstrated on the cell surface. People with C3 deficiency are susceptible to bacterial infection and it can be an indicator for some diseases.

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BGRL 16 (F23b) was made in response to intact human erythrocytes coated with C3/C4¹. In indirect haemagglutination tests, BGRL 16 agglutinates C3c on erythrocytes coated with C3 at low ionic strength by the method of Fruitstone² and with C3/C4 using alternative pathway activation by the method of Freedman and Mollison³. BGRL 16 reacts with epitope 3 on C3 and has an affinity of 0.67 x 10⁻¹ (I/M) for C3c. BGRL 16 does not inhibit the classical activation pathway of complement mediated lysis by using blood group AB red cells sensitized by anti-A or B monoclonal antibodies⁴.

## References

- 1. Dobbie D. Brazier D.M. Gardner B. Holburn A.M. (1987) Transfusion 27, 453-459.
- 2. Fruitstone M.J. (1978) Transfusion 18, 125.
- 3. Freedman J., Mollison P.L. (1976) Vox Sang 31, 241-57.
- 4. Mushens R.E. and Bakacs T. (1992) Transfusion 32, 430-434.