

IBGRL RESEARCH PRODUCTS DATA SHEET

Antigen	Complement C3d
Clone	BGRL 11
Product Code	9445
Immunoglobulin Class	Mouse IgM, kappa light chain

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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 is cleaved by C3 convertase into fragments including C3d. When erythrocytes are treated with a complement binding antibody such as anti-Le^a both C3c, C4 and C3d can be demonstrated on the cell surface, but on the cells of patients with autoimmune haemolytic anaemia of the cold type, only C3d can be demonstrated. It is therefore important that an antiglobulin reagent should contain the appropriate component. However, small amounts of C3d and C4 are to be found on normal red cells so that the anti-C3d component in a general antiglobulin reagent must be at an adequate concentration for detecting C3d without giving a false positive with normal cells.

Clone

BGRL 11 was made in response to intact human erythrocytes coated with C3/C4¹. In indirect haemagglutination tests, BGRL 11 agglutinates C3d 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. Cells coated with C3d were obtained by trypsinization of C3/C4 coated cells. BGRL 11 also agglutinates C3d on erythrocytes of patients with cold haemagglutinin disease¹. BGRL 11 reacts with epitope 3 on C3 and has an affinity of 8.9×10^{-7} (I/M) for C3d. BGRL 11 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.