IBGRL RESEARCH PRODUCTS

NHS Blood and Transplant

International Blood Group Reference Laboratory
500 North Bristol Park
Northway
Filton
Bristol
BS34 7QH

Protein Development and Production Unit
Tel: +44 (0)117 921 7500
Fax: +44 (0)117 912 5796
Website: http://ibgrl.blood.co.uk
Email: enquiries.IBGRL@nhsbt.nhs.uk

Antigen	Complement C3c
Clone	BGRL 13
Product Code	9495
Immunoglobulin Class	Mouse Ig1, kappa light chain

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-Le^a 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.

Clone

BGRL 13 (F44c) was made in response to intact human erythrocytes coated with C3/C4[1]. In indirect haemagglutination tests, BGRL 13 agglutinates C3c on erythrocytes coated with C3 at low ionic strength by the method of Fruitstone[2] and with C3/C4 using alternative pathway activation by the method of Freedman and Mollison[3]. BGRL 13 reacts with epitope 2 on C3 and has an affinity of 0.32 x 10^{-7} (l/M) for C3c. BGRL 13 (Monoclonal Number 181) was submitted to the workshop on monoclonal antibodies against human red blood cells and related antigens, Lund 1990[4]. BGRL 13 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 antibodies[5].

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. Voak D. and Nilsson (1990) J. Immunogenetics 17, 337-342.
5. Mushens R.E. and Bakacs T. (1992) Transfusion 32, 430-434.