

**International Blood Group  
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<b>Antigen</b>	CD58 (LFA-3)
<b>Clone</b>	BRIC 5
<b>Product Code</b>	9405
<b>Immunoglobulin Class</b>	Mouse IgG2a, kappa light chain

**Protein Development  
and Production Unit**

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CD58, or LFA3, is a membrane glycoprotein of 55kDa to 70kDa and is mapped to chromosome 1p13. It occurs in two forms, one transmembrane with a cytoplasmic domain, the other form anchored in the membrane via a glycosylphosphatidylinositol tail. The complete amino acid sequence of both forms has been deduced from cDNA. It is heavily N-glycosylated. CD58 is a cell adhesion molecule which plays a critical role in facilitation of antigen specific recognition through interaction with CD2 on T lymphocytes and natural killer cells<sup>1</sup>. It is a member of the immunoglobulin superfamily of molecules. CD58 has a wide tissue distribution, being present on erythrocytes, platelets, monocytes, a subset of lymphocytes, bone marrow cells, fibroblasts, epithelium and endothelium cells<sup>2</sup>. There are approximately 5000 CD58 molecules on each erythrocyte. There is reduced expression of CD58 on cells of individuals with paroxysmal nocturnal haemoglobinuria.

**Clone**

BRIC 5 was made in response to human erythrocytes. It inhibits human T cell rosette formation and blocks T cell proliferation stimulated by activated T cells. It reacts with epitope cluster 1 defined by the fifth leucocyte workshop<sup>3</sup> and will cross block TS2/9<sup>4</sup>. It has a functional binding affinity to erythrocytes of  $4 \times 10^8 \text{M}^{-1}$ . BRIC 5 binds to a broadly migrating component of 40-65 KDa on an immunoblot of non-reduced erythrocyte membranes. BRIC 5 is an indirect haemagglutinin. The erythrocyte antigen is pronase, trypsin, chymotrypsin and AET sensitive. CD58 was clustered for the first time at the fourth leucocyte workshop<sup>2</sup> by three antibodies including BRIC 5. BRIC 5 was also submitted to the fifth leucocyte workshop<sup>5</sup>. BRIC 5 has been used by immunoblotting of red cell membrane proteins to reveal the membrane alterations in the absence of RHCE<sup>6</sup>.

**References**

1. Makgoba M.W., *et al*, (1989) *Immunology Today* **10** 417-422 (Review)
2. Shaw S. & Johnson J.P., (1989) in *Leucocyte Typing IV; White Cell Differentiation Antigens* Ed. W. Knapp *et al* Oxford University Press pp 714-716
3. Klickstein *et al.*, (1993) *Proceedings of the fifth workshop and conference on white cell differentiation antigens*, Boston, vol. 2 1475-1476.
4. Anstee DJ *et al.* (1991) *Immunology*, **74**: 197-205.
5. Schlossman SF (ed) *et al.*, (1993) *Proceedings of the fifth workshop and conference on white cell differentiation antigens*, Boston, Oxford University Press 1995.
6. Joanna F. Flatt, *et al*, (2012) Study of the D-- phenotype reveals erythrocyte membrane alterations in the absence of RHCE. *BJH* **158**, 262-273.