

DESCRIPTION

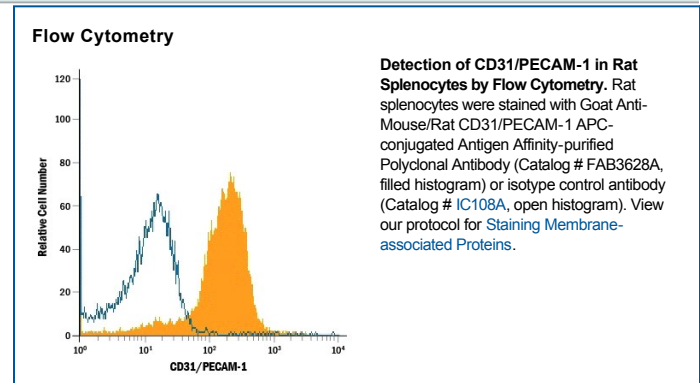
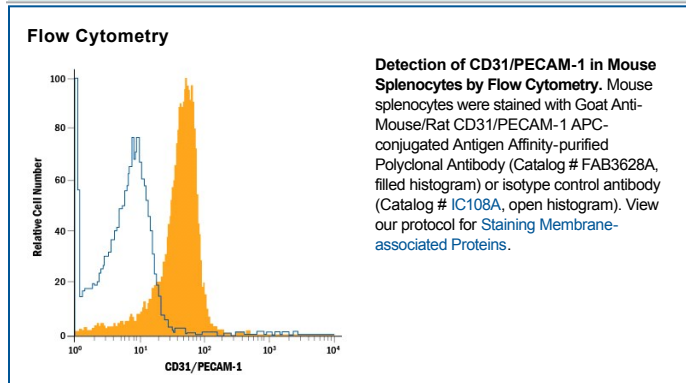
Species Reactivity	Mouse/Rat
Specificity	Detects mouse CD31/PECAM-1 in direct ELISAs and Western blots. In direct ELISAs and Western blots, approximately 10% cross-reactivity with recombinant human CD31 and recombinant porcine CD31 is observed. Detects mouse CD31 and rat CD31 in flow cytometry.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse CD31/PECAM-1 Glu18-Lys590 Accession # Q08481
Conjugate	Allophycocyanin Excitation Wavelength: 620-650 nm Emission Wavelength: 660-670 nm
Formulation	Supplied in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Flow Cytometry	10 μ L/10 ⁶ cells	See Below

DATA



PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. <ul style="list-style-type: none"> ● 12 months from date of receipt, 2 to 8 °C as supplied.

BACKGROUND

PECAM-1 (Platelet-Endothelial Cell Adhesion Molecule-1), also known as CD31, is a 130 kDa type I transmembrane glycoprotein adhesion molecule in the immunoglobulin superfamily (1, 2). Expression is restricted to cells involved in circulation, especially endothelial cells, platelets, monocytes, neutrophils and lymphocyte subsets. PECAM-1 is concentrated at cell-cell junctions and is required for Transendothelial Migration (TEM) (1-3). The Extracellular Domain (ECD) of PECAM-1 has ten potential N-linked glycosylation sites and six C2-type Ig-like domains, the first of which is critical for adhesion and extravasation (3, 4). The cytoplasmic domain contains Immunoregulatory Tyrosine-based Inhibitory and Switch Motifs (ITIM, ITSM) that mediate both inhibition and activation via phosphotyrosine-mediated engagement of SH2-containing signaling molecules (1, 5). Metalloproteinase-mediated ectodomain shedding occurs during apoptosis (6) but increased serum PECAM-1 ectodomain in HIV and active multiple sclerosis occurs independent of apoptosis (7, 8). In humans, expression of six isoforms with exon deletions in the cytoplasmic domain is tissue- and stage-specific, but full-length PECAM-1 is predominant. A form lacking the ITSM predominates in mouse (9). Mouse PECAM-1 ECD shows 77%, 63%, 63%, 63%, and 61% amino acid (aa) identity with rat, human, canine, porcine, and bovine PECAM-1, respectively. PECAM-1 participates with other adhesion molecules in some functions, but is the critical molecule for TEM. Homotypic PECAM-1 adhesion in trans, combined with cycling of PECAM-1 to and from surface-connected endothelial cell vesicles, leads leukocytes across endothelial tight junctions (3, 10). Homotypic adhesion and signaling functions also strongly suppress mitochondria-dependent apoptosis (11). In platelets, PECAM-1 is necessary for limiting thrombus formation (12) and promoting integrin-mediated clot retraction and platelet spreading (13), but mechanisms for these phenomena are unclear. PECAM-1^{-/-} mice are deficient in chemokine-mediated chemotaxis (14).

References:

1. Ilan, N. and J.A. Madri (2003) *Curr. Opin. Cell Biol.* **15**:515.
2. Xie, Y. and W.A. Muller (1993) *Proc. Natl. Acad. Sci. USA* **90**:5569.
3. Liao, F. *et al.* (1997) *J. Exp. Med.* **185**:1349.
4. Nakada, M.T. *et al.* (2000) *J. Immunol.* **164**:452.
5. Chemnitz, J.M. *et al.* (2004) *J. Immunol.* **173**:945.
6. Ilan, N. *et al.* (2001) *FASEB J.* **15**:362.
7. Eugenin, E.A. *et al.* (2006) *J. Leukoc. Biol.* **79**:444.
8. Losy, J. *et al.* (1999) *J. Neuroimmunol.* **99**:169.
9. Wang, Y. *et al.* (2003) *Am. J. Physiol. Heart Circ. Physiol.* **284**:H1008.
10. Mamdough, Z. *et al.* (2003) *Nature* **421**:748.
11. Gao, C. *et al.* (2003) *Blood* **102**:169.
12. Falati, S. *et al.* (2006) *Blood* **107**:535.
13. Wee, J.L. and D.E. Jackson (2005) *Blood* **106**:3816.
14. Wu, Y. *et al.* (2005) *J. Immunol.* **175**:3484.