

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human PEAR1 in direct ELISAs and Western blots. In Western blots, no cross-reactivity with recombinant human Tenascin R or recombinant mouse Tenascin C is observed.
Source	Monoclonal Mouse IgG _{2A} Clone # 492621
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Chinese hamster ovary cell line CHO-derived recombinant human PEAR1 Leu21-Ser754 Accession # Q5VY43
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm
Formulation	Supplied 0.2 mg/mL 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.

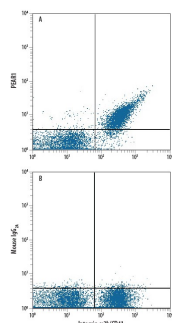
APPLICATIONS

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	0.25-1 µg/10 ⁶ cells	See Below

DATA

Flow Cytometry



Detection of PEAR1 in Human Blood Platelets by Flow Cytometry. Human peripheral blood platelets were stained with Mouse Anti-Human Integrin α2b/CD41 APC-conjugated Monoclonal Antibody (Catalog # [FAB7616A](#)) and either (A) Mouse Anti-Human PEAR1 Alexa Fluor® 488-conjugated Monoclonal Antibody (Catalog # FAB4527G) or (B) Mouse IgG_{2A} Alexa Fluor 488 Isotype Control (Catalog # IC003G). View our protocol for [Staining Membrane-associated Proteins](#).

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

Platelet endothelial aggregation receptor 1 (PEAR1) is a 150 kDa type I transmembrane protein and member of the MEGF family of proteins (1). Human PEAR1 is synthesized as a 1037 amino acid (aa) precursor that contains a 20 aa signal sequence, a 735 aa extracellular domain (ECD), a 21 aa transmembrane region, and a 261 aa cytoplasmic region. The ECD consists of 15 EGF-like repeats that vary in length from 39 to 42 aa and contain a consensus sequence of CX₁₋₂GX₂GX₂-₄CX₃CX₁₋₃CX₁₋₂GX₁₋₂CX₄GX₁CX₁CX₂GX₂GX₂C (1). The consensus repeat contains six conserved glycine residues and eight conserved cysteine residues, suggesting four disulfide-bonded cysteine pairs in each EGF repeat (1). Within the ECD, there are also five potential sites for N-linked glycosylation. The cytoplasmic region contains five potential Src homology 3-binding, proline-rich domains (1). Mature human PEAR1 is 84% aa identical to mature mouse PEAR1. PEAR1 is most highly expressed in platelets and endothelial cells (1). Functionally, PEAR1 is a receptor for a yet undetermined ligand that signals upon the formation of platelet-platelet contacts induced both by platelet aggregations and by platelet centrifugation (1). The signaling enhances and stabilizes platelet thrombi (2). Upon aggregation, the surface-expressed protein is tyrosine-phosphorylated (1). This phosphorylation event is inhibited by the α_{IIb}β₃ antagonist eptifibatide, thus demonstrating that PEAR1 tyrosine phosphorylation is dependent on surface contacts between activated platelets (1). PEAR1 can be phosphorylated in an α_{IIb}β₃ integrin-dependent manner on tyrosine (Tyr925) and serine residues (Ser593 and Ser1029) and, potentially, at Tyr804, Tyr943, and Tyr979 (1). Inherited PEAR1 variations that alter expression or function of the platelet signaling molecule could modify agonist-induced aggregation in native platelets (2). In addition, a genetic variant in PEAR1 could be an important determinant of residual platelet function during aspirin treatment, because the COX1/thromboxane A₂ pathway will be strongly inhibited by aspirin, and maximal aggregation will then be dependent on other secondary signaling pathways (2).

References:

1. Nanda, N. *et al.* (2005) J. Biol. Chem. **280**:24680.
2. Herrera-Galeano, J.E. *et al.* (2008) Arterioscler. Thromb. Vasc. Biol. **28**:1484.

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