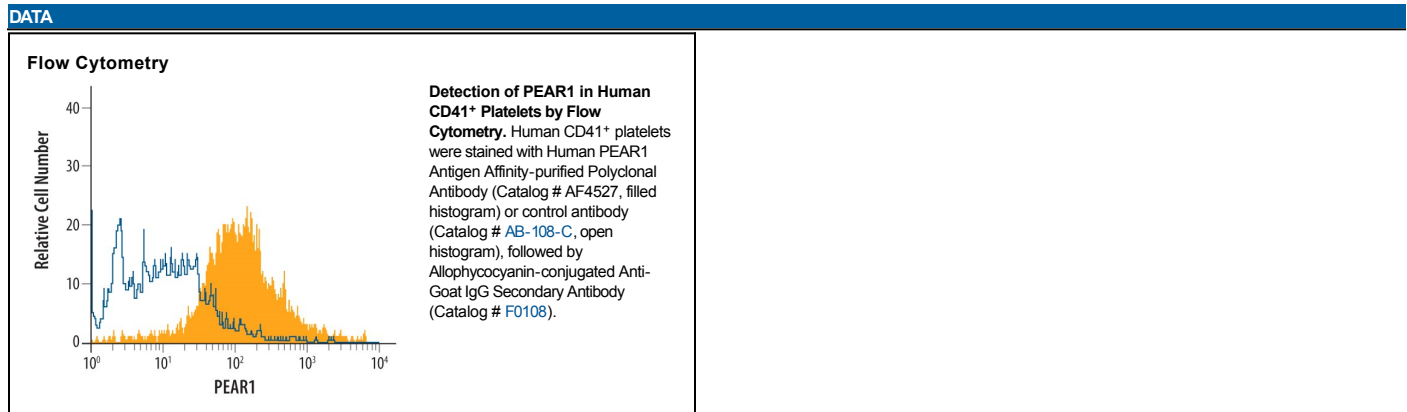


DESCRIPTION	
<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human PEAR1 in direct ELISAs and Western blots.
<b>Source</b>	Polyclonal Goat IgG
<b>Purification</b>	Antigen Affinity-purified
<b>Immunogen</b>	Chinese hamster ovary cell line CHO-derived recombinant human PEAR1 Leu21-Ser754 Accession # Q5VY43
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

APPLICATIONS		
<b>Please Note:</b> Optimal dilutions should be determined by each laboratory for each application. <i>General Protocols</i> are available in the <i>Technical Information</i> section on our website.		
	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Western Blot</b>	0.1 µg/mL	Recombinant Human PEAR1 (Catalog # 4527-PR)
<b>Flow Cytometry</b>	2.5 µg/10 <sup>6</sup> cells	See Below
<b>CyTOF-ready</b>	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	



PREPARATION AND STORAGE	
<b>Reconstitution</b>	Reconstitute at 0.2 mg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
<b>Stability &amp; Storage</b>	<p><b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b></p> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

**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 CX<sub>1-2</sub>GX<sub>2</sub>GX<sub>2-4</sub>CX<sub>3</sub>CX<sub>1-3</sub>CX<sub>1-2</sub>GX<sub>1-2</sub>CX<sub>4</sub>GX<sub>1</sub>CX<sub>1</sub>CX<sub>2</sub>GX<sub>2</sub>GX<sub>2</sub>C (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  $\alpha_{IIb}\beta_3$  antagonist eptifibatid, thus demonstrating that PEAR1 tyrosine phosphorylation is dependent on surface contacts between activated platelets (1). PEAR1 can be phosphorylated in an  $\alpha_{IIb}\beta_3$  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.