## Elabscience Bionovation Inc.



A Reliable Research Partner in Life Science and Medicine

# **GP1BB Polyclonal Antibody**

catalog number: E-AB-90075

Note: Centrifuge before opening to ensure complete recovery of vial contents.

## Description

Reactivity Human; Mouse; Rat

**Immunogen** Recombinant fusion protein of human GP1BB

Host Rabbit Isotype IgG

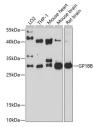
**Purification** Affinity purification

**Buffer** Phosphate buffered solution, pH 7.4, containing 0.05% stabilizer and 50% glycerol.

# Applications Recommended Dilution

**WB** 1:200-1:1000

#### Data



Western blot analysis of extracts of various cell lines using

GP1BB Polyclonal Antibody at 1:1000 dilution.

Observed-MV:30 kDa Calculated-MV:21 kDa/43 kDa

# Preparation & Storage

Storage Storage Store at -20°C Valid for 12 months. Avoid freeze / thaw cycles.

**Shipping** The product is shipped with ice pack, upon receipt, store it immediately at the

temperature recommended.

## Background

Platelet glycoprotein Ib (GPIb) is a heterodimeric transmembrane protein consisting of a disulfide-linked 140 kD alpha chain and 22 kD beta chain. It is part of the GPIb-V-IX system that constitutes the receptor for von Willebrand factor (VWF), and mediates platelet adhesion in the arterial circulation. GPIb alpha chain provides the VWF binding site, and GPIb beta contributes to surface expression of the receptor and participates in transmembrane signaling through phosphorylation of its intracellular domain. Mutations in the GPIb beta subunit have been associated with Bernard-Soulier syndrome, velocardiofacial syndrome and giant platelet disorder. The 206 amino acid precursor of GPIb beta is synthesized from a 1.0 kb mRNA expressed in plateletes and megakaryocytes. A 411 amino acid protein arising from a longer, unspliced transcript in endothelial cells has been described; however, the authenticity of this product has been questioned. Yet another less abundant GPIb beta mRNA species of 3.5 kb, expressed in nonhematopoietic tissues such as endothelium, brain and heart, was shown to result from inefficient usage of a non-consensus polyA signal in the neighboring upstream gene (SEPT5, septin 5). In the absence of polyadenylation from its own imperfect site, the SEPT5 gene produces read-through transcripts that use the consensus polyA signal of this gene.

## For Research Use Only

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