

Product datasheet for **TP724209**

Human GP1BB Protein, hFc Tag

Product data:

Product Type:	Recombinant Proteins
Description:	Human GP1BB Protein, hFc Tag
Expression Host:	HEK293
Tag:	C-Human Fc
Predicted MW:	The protein has a predicted molecular mass of 39.0 kDa after removal of the signal peptide. The apparent molecular mass of GP1BB-hFc is approximately 35-55 kDa due to glycosylation.
Purity:	The purity of the protein is greater than 95% as determined by SDS-PAGE and Coomassie blue staining.
Reconstitution Method:	Lyophilized from sterile PBS, pH 7.4. Normally 5 % - 8% trehalose is added as protectants before lyophilization.
Storage:	Store at -20°C to -80°C for 12 months in lyophilized form. After reconstitution, if not intended for use within a month, aliquot and store at -80°C (Avoid repeated freezing and thawing). Lyophilized proteins are shipped at ambient temperature.
Stability:	12 months from date of despatch
Synonyms:	BDPLT1; BS; CD42C; GPIBB; GPIbbeta



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Summary:

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 platelets 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. [provided by RefSeq, Dec 2010]