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Rabbit Anti-CD42b antibody

SL2347R

Product Name:	CD42b
Chinese Name:	血小板glycoproteinGPIb抗体
Alias:	Antigen CD42b alpha; BSS; CD 42b; CD42b alpha; CD42b antigen; GLYCOCALICIN; Glycoprotein Ib (platelet) alpha polypeptide; Glycoprotein Ibalpha; GP Ib alpha; GP1B; GP1BA; GPIb alpha; MGC34595; Platelet glycoprotein Ib alpha chain; Platelet glycoprotein Ib alpha polypeptide; Platelet membrane glycoprotein 1b alpha subunit.
文献引用 PubMed :	**Specific References(1)**|SL2347R has been referenced in 1 publications. **[IF=13.91]**Ye, Buqing, et al. "Cytosolic carboxypeptidase CCP6 is required for megakaryopoiesis by modulating Mad2 polyglutamylation." The Journal of Experimental Medicine (2014): jem-20141123.**Mouse**. PubMed:25332286
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Mouse,Rat,
Applications:	WB=1:500-2000 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	67kDa
Cellular localization:	The cell membrane
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthetic peptide derived from human GP1B/CD42b:201-300/626<Extracellular>
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.

Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
PubMed:	PubMed
Product Detail:	Glycoprotein Ib (GP Ib) is a platelet surface membrane glycoprotein composed of a heterodimer, an alpha chain and a beta chain, that is linked by disulfide bonds. The Gp Ib functions as a receptor for von Willebrand factor (VWF). The complete receptor complex includes noncovalent association of the alpha and beta subunits with platelet glycoprotein IX and platelet glycoprotein V. The binding of the GP Ib-IX-V complex to VWF facilitates initial platelet adhesion to vascular subendothelium after vascular injury, and also initiates signaling events within the platelet that lead to enhanced platelet activation, thrombosis, and hemostasis. This gene encodes the alpha subunit. Several polymorphisms and mutations have been described in this gene, some of which are the cause of Bernard-Soulier syndromes and platelet-type von Willebrand disease. [provided by RefSeq, Mar 2010]. **Function:** GP-Ib, a surface membrane protein of platelets, participates in the formation of platelet plugs by binding to the A1 domain of vWF, which is already bound to the subendothelium. **Subunit:** Heterodimer composed of GP-Ib alpha and beta; disulfide linked. GP-IX is complexed with the GP-Ib heterodimer via a non covalent linkage. Interacts with FLNB. **Subcellular Location:** Membrane; Single-pass type I membrane protein. **Post-translational modifications:** Glycocalicin, which is approximately coextensive with the extracellular part of the molecule, is cleaved off by calpain during platelet lysis. **DISEASE:** Genetic variations in GP1BA may be a cause of susceptibility to non-arteritic anterior ischemic optic neuropathy (NAION) [MIM:258660]. NAION is an ocular disease due to ischemic injury to the optic nerve. It usually affects the optic disk and leads to visual loss and optic disk swelling of a pallid nature. Visual loss is usually sudden, or over a few days at most and is usually permanent, with some recovery possibly occurring within the first weeks or months. Patients with small disks having smaller or non-existent cups have an anatomical predisposition for non-arteritic anterior ischemic optic neuropathy. As an ischemic episode evolves, the swelling compromises circulation, with a spiral of ischemia resulting in further neuronal damage. Defects in GP1BA are a cause of Bernard-Soulier syndrome (BSS) [MIM:231200]; also known as giant platelet disease (GPD). BSS patients have unusually large platelets and have a clinical bleeding tendency.

	Defects in GP1BA are the cause of benign Mediterranean macrothrombocytopenia (BMM) [MIM:153670]; also known as autosomal dominant benign Bernard-Soulier syndrome. BMM is characterized by mild or no clinical symptoms, normal platelet function, and normal megakaryocyte count. Defects in GP1BA are the cause of von Willebrand disease platelet-type (PVWD) [MIM:177820]; also known as pseudo-von Willebrand disease (pseudo-vWD). This autosomal dominant bleeding disorder is caused by an increased affinity of GP-Ib for soluble vWF resulting in impaired hemostatic function due to the removal of vWF from the circulation. **Similarity:** Contains 7 LRR (leucine-rich) repeats. Contains 1 LRRCT domain. Contains 1 LRRNT domain. **SWISS:** P07359 **Gene ID:** 2811 **Database links:** Entrez Gene: 2811Human Omim: 606672Human SwissProt: P07359Human Unigene: 1472Human **Important Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.