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CD42b Recombinant Mouse mAb

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Catalog # AP94244

Product Information

Host	Rabbit
Clonality	Recombinant
Physical State	Liquid
Isotype	IgG1, Kappa
Purity	affinity purified by Protein G
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SUBCELLULAR LOCATION	Membrane; Single-pass type I membrane protein.
SIMILARITY	Contains 7 LRR (leucine-rich) repeats. Contains 1 LRRCT domain. Contains 1 LRRNT domain.
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.
Post-translational modifications	lycocalicin, 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.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	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].

Additional Information

Dilution	Flow-Cyt=1:50
Format	0.01M TBS(pH7.4) with 1% BSA, 0.09% (W/V) sodium azide and 50% Glyce
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. 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.

Background

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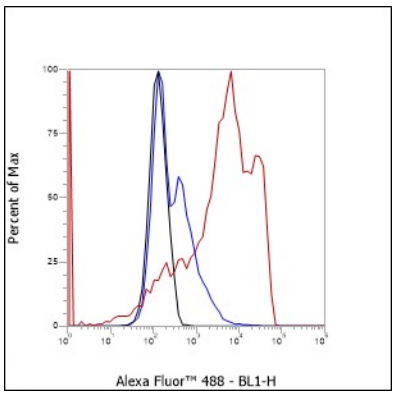
Images



Specimen: Human platelets Fixative: Unfixed Permeabilization: None Primary Ab dilution: 1:50 Secondary Ab: Goat anti Mouse IgG Unlabelled control: The cell without incubation with primary antibody and secondary antibody (Black line). Isotype control: Mouse monoclonal IgG1 (Blue line). Comment: Line red is the positive signal for AP94244