

Integrin allb rabbit pAb

Catalog No: YT7836

Reactivity: Human; Mouse

Applications: WB;ELISA

Target: CD41

Fields: >>Rap1 signaling pathway;>>PI3K-Akt signaling pathway;>>Focal

adhesion;>>ECM-receptor interaction;>>Platelet activation;>>Neutrophil extracellular trap formation;>>Hematopoietic cell lineage;>>Regulation of actin cytoskeleton;>>Human papillomavirus infection;>>Pathways in cancer;>>Small cell lung cancer;>>Hypertrophic cardiomyopathy;>>Arrhythmogenic right

ventricular cardiomyopathy;>>Dilated cardiomyopathy;>>Fluid shear stress and

atherosclerosis

P08514

Q9QUM0

Gene Name: ITGA2B GP2B ITGAB

Protein Name: Integrin allb

Human Gene Id: 3674

Human Swiss Prot

No:

Mouse Gene Id: 16399

Mouse Swiss Prot

No:

Immunogen: Synthesized peptide derived from human Integrin allb AA range: 1-80

Specificity: This antibody detects endogenous levels of Human Integrin allb

Formulation : Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Source: Polyclonal, Rabbit, IgG

Dilution: WB 1:1000-2000 ELISA 1:5000-20000



Purification: The antibody was affinity-purified from rabbit antiserum by affinity-

chromatography using epitope-specific immunogen.

Concentration: 1 mg/ml

Storage Stability: -15°C to -25°C/1 year(Do not lower than -25°C)

Molecularweight: 114kD

Background: This gene encodes a member of the integrin alpha chain family of proteins. The

encoded preproprotein is proteolytically processed to generate light and heavy chains that associate through disulfide linkages to form a subunit of the alpha-IIb/beta-3 integrin cell adhesion receptor. This receptor plays a crucial role in the blood coagulation system, by mediating platelet aggregation. Mutations in this gene are associated with platelet-type bleeding disorders, which are characterized by a failure of platelet aggregation, including Glanzmann

thrombasthenia. [provided by RefSeq, Jan 2016],

Function: disease:Defects in ITGA2B are a cause of Glanzmann thrombasthenia (GT)

[MIM:273800]; also known as thrombasthenia of Glanzmann and Naegeli. This autosomal recessive disorder is the most common inherited disease of platelets. GT is characterized by mucocutaneous bleeding of mild-to-moderate severity and the inability of this integrin to recognize macromolecular or synthetic peptide ligands. GT has been classified clinically into types I and II. In type I, platelets show absence of the glycoprotein IIb/beta-3 complexes at their surface and lack fibrinogen and clot retraction capability. In type II, the platelets express the glycoprotein IIb/beta-3 complex at reduced levels (5-20% controls), have detectable amounts of fibrinogen, and have low or moderate clot retraction capability. The platelets of GT 'variants' have normal or near normal (60-100%)

expression of dysfunctional receptors.,fun

Subcellular Location : Membrane; Single-pass type I membrane protein.

Expression: Isoform 1 and isoform 2 are expressed in platelets and megakaryocytes, but not

in reticulocytes. Not detected in Jurkat, nor in U937 cell lines (PubMed:2351656). Isoform 3 is expressed in prostate adenocarcinoma, as well as in several erythroleukemia, prostate adenocarcinoma and melanoma cell lines, including PC-3, DU-145, HEL, WM983A, WM983B and WM35. Not detected in platelets,

nor in normal prostate (at protein level) (PubMed:9809974).

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