Overview

Product name: Anti-CD41 antibody [MWReg30]
Description: Rat monoclonal [MWReg30] to CD41
Host species: Rat
Specificity: ab33661 recognizes integrin alpha IIb subunit CD41
Tested applications: Suitable for: IHC-P, Flow Cyt, IHC-Fr, IP
Species reactivity: Reacts with: Mouse
Immunogen: The details of the immunogen for this antibody are not available.
General notes: The azide free version is ab94960.

Clone MWReg30 has been reported to inhibit PMA induced aggregation in vitro and to induce hypothermia in vivo.

Properties

Form: Liquid
Storage buffer: Preservative: 0.09% Sodium Azide
Constituents: PBS, pH 7.4
Purity: Protein G purified
Purification notes: Purified IgG prepared by affinity chromatography on Protein G from tissue culture supernatant
Primary antibody notes: Clone MWReg30 has been reported to inhibit PMA induced aggregation in vitro and to induce hypothermia in vivo.
Clonality: Monoclonal
Clone number: MWReg30
Isotype: IgG1

Applications

Our Abpromise guarantee covers the use of ab33661 in the following tested applications.
Integrin alpha-IIb/beta-3 is a receptor for fibronectin, fibrinogen, plasminogen, prothrombin, thrombospondin and vitronectin. It recognizes the sequence R-G-D in a wide array of ligands. It recognizes the sequence H-H-L-G-G-G-A-K-Q-A-G-D-V in fibrinogen gamma chain. Following activation integrin alpha-IIb/beta-3 brings about platelet/platelet interaction through binding of soluble fibrinogen. This step leads to rapid platelet aggregation which physically plugs ruptured endothelial cell surface.

Isoform 1 and isoform 2 were identified in platelets and megakaryocytes, but not in reticulocytes or in Jurkat and U937 white blood cell line. Isoform 3 is expressed by leukemia, prostate adenocarcinoma and melanoma cells but not by platelets or normal prostate or breast epithelial cells.

Defects in ITGA2B are a cause of Glanzmann thrombasthenia (GT) [MIM:273800]; also known as thrombasthenia of Glanzmann and Naegeli. GT is the most common inherited disease of platelets. It is an autosomal recessive disorder 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.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

<table>
<thead>
<tr>
<th>Application</th>
<th>Abreviews</th>
<th>Notes</th>
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<tbody>
<tr>
<td>IHC-P</td>
<td>⭐⭐⭐⭐⭐</td>
<td>Use at an assay dependent concentration. PubMed: 20525356</td>
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<tr>
<td>Flow Cyt</td>
<td>⭐⭐⭐⭐⭐</td>
<td>1/50 - 1/100. Use 10µl of the suggested working dilution to label 106 cells in 100µl.</td>
</tr>
<tr>
<td>IHC-Fr</td>
<td>⭐⭐⭐⭐⭐</td>
<td>1/300.</td>
</tr>
<tr>
<td>IP</td>
<td></td>
<td>Use at an assay dependent concentration.</td>
</tr>
</tbody>
</table>

**Function**
Integrin alpha-IIb/beta-3 is a receptor for fibronectin, fibrinogen, plasminogen, prothrombin, thrombospondin and vitronectin. It recognizes the sequence R-G-D in a wide array of ligands. It recognizes the sequence H-H-L-G-G-G-A-K-Q-A-G-D-V in fibrinogen gamma chain. Following activation integrin alpha-IIb/beta-3 brings about platelet/platelet interaction through binding of soluble fibrinogen. This step leads to rapid platelet aggregation which physically plugs ruptured endothelial cell surface.

**Tissue specificity**
Isoform 1 and isoform 2 were identified in platelets and megakaryocytes, but not in reticulocytes or in Jurkat and U937 white blood cell line. Isoform 3 is expressed by leukemia, prostate adenocarcinoma and melanoma cells but not by platelets or normal prostate or breast epithelial cells.

**Involvement in disease**
Defects in ITGA2B are a cause of Glanzmann thrombasthenia (GT) [MIM:273800]; also known as thrombasthenia of Glanzmann and Naegeli. GT is the most common inherited disease of platelets. It is an autosomal recessive disorder 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.

**Sequence similarities**
Belongs to the integrin alpha chain family. Contains 7 FG-GAP repeats.

**Cellular localization**
Membrane.

**Images**

*ab18407* - Rat monoclonal IgG1, is suitable for use as an isotype control with this antibody.
ab33661 staining CD41 in mouse spleen tissue sections by Immunohistochemistry (IHC-P - paraformaldehyde-fixed, paraffin-embedded sections). Tissue was fixed with formaldehyde and blocked with 1% BSA in PBS for 30 minutes at 20°C. Samples were incubated with primary antibody (1/200 in PBS + 1% BSA) for 9 hours at 4°C. A Biotin-conjugated goat anti-rat IgG polyclonal (1/300) was used as the secondary antibody.

IHC-Fr image of CD41 staining on mouse lung sections using ab33661 (1:300). The sections came from PFA fixed and snap frozen tissue and 10 micron sections were cut using a cryostat. The sections were blocked with 1% BSA at 21°C for 10 mins. The sections were then incubated with ab33661 for 2 hours at 21°C. Goat Polyclonal to Rat IgG conjugated to Biotin was used at 1:200.

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