abcam

Product datasheet

PE Anti-non-muscle Myosin IIA antibody [EPR8965] ab211837

Recombinant RabMAb

1 References 2 Images

Overview

Product name	PE Anti-non-muscle Myosin IIA antibody [EPR8965]
Description	PE Rabbit monoclonal [EPR8965] to non-muscle Myosin IIA
Host species	Rabbit
Conjugation	PE. Ex: 488nm, Em: 575nm
Tested applications	**Suitable for:** Flow Cyt (Intra)
Species reactivity	**Reacts with:** Human
Immunogen	Synthetic peptide. This information is proprietary to Abcam and/or its suppliers.
Positive control	Flow Cyt (intra)ometry: A431 cells
General notes	This product is a recombinant monoclonal antibody, which offers several advantages including: - High batch-to-batch consistency and reproducibility - Improved sensitivity and specificity - Long-term security of supply - Animal-free production For more information **see here**. Our RabMAb® technology is a patented hybridoma-based technology for making rabbit monoclonal antibodies. For details on our patents, please refer to **RabMAb® patents**.

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Upon delivery aliquot. Store at +4°C. Do Not Freeze. Store In the Dark.
Storage buffer	pH: 7.4 Preservative: 0.02% Sodium azide Constituents: PBS, 1% BSA
Purity	Protein A purified
Clonality	Monoclonal
Clone number	EPR8965

Isotype IgG

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab211837 in the following tested applications.

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

Application	Abreviews	Notes
Flow Cyt (Intra)		1/5000. The cellular localisation of this product has been verified in ICC/IF

Target

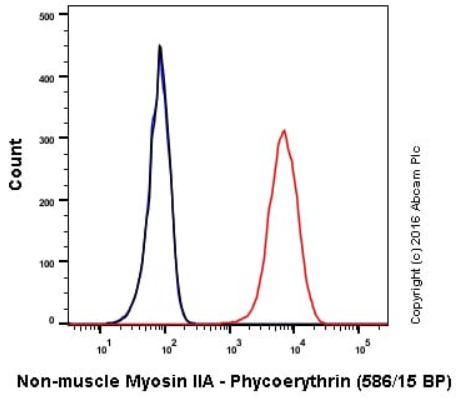
Function Cellular myosin that appears to play a role in cytokinesis, cell shape, and specialized functions such as secretion and capping.

Tissue specificity In the kidney, expressed in the glomeruli. Also expressed in leukocytes.

Involvement in disease Defects in MYH9 are the cause of May-Hegglin anomaly (MHA) [MIM:155100]. MHA is an autosomal dominant macrothrombocytopenia characterized by thrombocytopenia, giant platelets and leukokyte inclusions appearing as highly parallel paracrystalline bodies.
Defects in MYH9 are the cause of Sebastian syndrome (SBS) [MIM:605249]. SBS is an autosomal dominant macrothrombocytopenia characterized by thrombocytopenia, giant platelets and leukocyte inclusions that are smaller and less organized than in May-Hegglin anomaly.
Defects in MYH9 are the cause of Fechtner syndrome (FTNS) [MIM:153640]. FTNS is an autosomal dominant macrothrombocytopenia characterized by thrombocytopenia, giant platelets and leukocyte inclusions that are small and poorly organized. Additionally, FTNS is distinguished by Alport-like clinical features of sensorineural deafness, cataracts and nephritis.
Defects in MYH9 are the cause of Alport syndrome with macrothrombocytopenia (APSM) [MIM:153650]. APSM is an autosomal dominant disorder characterized by the association of ocular lesions, sensorineural hearing loss and nephritis (Alport syndrome) with platelet defects.
Defects in MYH9 are the cause of Epstein syndrome (EPS) [MIM:153650]. EPS is an autosomal dominant disorder characterized by the association of macrothrombocytopathy, sensorineural hearing loss and nephritis.
Defects in MYH9 are the cause of deafness autosomal dominant type 17 (DFNA17) [MIM:603622]. DFNA17 is a form of sensorineural hearing loss. Sensorineural deafness results from damage to the neural receptors of the inner ear, the nerve pathways to the brain, or the area of the brain that receives sound information. DFNA17 is characterized by progressive hearing impairment and cochleosaccular degeneration.
Defects in MYH9 are the cause of macrothrombocytopenia with progressive sensorineural deafness (MPSD) [MIM:600208]. MPSD is an autosomal dominant disorder characterized by the association of macrothrombocytopathy and progressive sensorineural hearing loss without renal dysfunction.
Note=Subjects with mutations in the motor domain of MYH9 present with severe thrombocytopenia and develop nephritis and deafness before the age of 40 years, while those with mutations in the tail domain have a much lower risk of noncongenital complications and significantly higher platelet counts. The clinical course of patients with mutations in the four most frequently affected residues of MYH9 (responsible for 70% of MYH9-related cases) were evaluated. Mutations at residue 1933 do not induce kidney damage or cataracts and cause

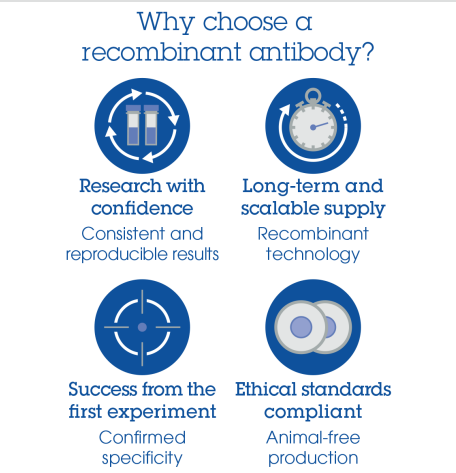
	deafness only in the elderly, those in position 702 result in severe thrombocytopenia and produce nephritis and deafness at a juvenile age, while alterations at residue 1424 or 1841 result in intermediate clinical pictures. Note=Genetic variations in MYH9 are associated with non-diabetic end stage renal disease (ESRD).
Sequence similarities	Contains 1 IQ domain. Contains 1 myosin head-like domain.
Domain	The rodlike tail sequence is highly repetitive, showing cycles of a 28-residue repeat pattern composed of 4 heptapeptides, characteristic for alpha-helical coiled coils.
Post-translational modifications	ISGylated.

Images



Flow Cytometry (Intracellular) - PE Anti-non-muscle Myosin IIA antibody [EPR8965] (ab211837)

Overlay histogram showing A431 cells stained with ab211837 (red line). The cells were fixed with 4% formaldehyde and then permeabilized with 90% methanol at -20°C for 15 min. The cells were then incubated in 1x PBS / 10% normal goat serum to block non-specific protein-protein interactions followed by the antibody (ab211837, 1/5000 dilution) for 30 min at 22°C. Isotype control antibody (black line) was rabbit IgG (monoclonal) Phycoerythrin (**ab209478**) used at the same concentration and conditions as the primary antibody. Unlabelled sample (blue line) was also used as a control. Acquisition of >5,000 events were collected using a 50mW Yellow/Green laser (561nm) and 586/15 bandpass filter.



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