abcam

Product datasheet

HRP Anti-non-muscle Myosin IIA antibody [EPR8965] ab205470

KO VALIDATED Recombinant RabMAb

3 Images

Overview

Product name	HRP Anti-non-muscle Myosin IIA antibody [EPR8965]
Description	HRP Rabbit monoclonal [EPR8965] to non-muscle Myosin IIA
Host species	Rabbit
Conjugation	HRP
Tested applications	**Suitable for:** WB
Species reactivity	**Reacts with:** Human
Immunogen	Synthetic peptide. This information is proprietary to Abcam and/or its suppliers.
Positive control	WB: Jurkat and A431 whole cell and human fetal kidney tissue lysates.
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. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C. Avoid freeze / thaw cycle. Store In the Dark.
Storage buffer	pH: 7.40 Preservative: 0.1% Proclin 300 Solution Constituents: 30% Glycerol (glycerin, glycerine), 1% BSA, PBS
Purity	Protein A purified
Clonality	Monoclonal
Clone number	EPR8965

Isotype IgG

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab205470 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
WB		1/5000. Detects a band of approximately 230 kDa (predicted molecular weight: 227 kDa).

Target

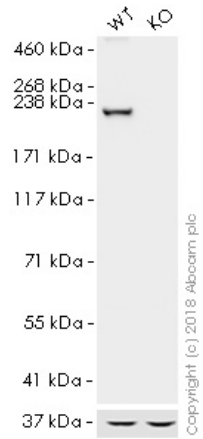
Function Cellular myosin that appears to play a role in cytokinesis, cell shape, and specialized functions such as secretion and capping.

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



Western blot - HRP Anti-non-muscle Myosin IIA antibody [EPR8965] (ab205470)

All lanes : HRP Anti-non-muscle Myosin IIA antibody [EPR8965] (ab205470) at 1/5000 dilution

Lane 1 : Wild-type HAP1 whole cell lysate
Lane 2 : MYH9 (non-muscle Myosin IIA) knockout HAP1 whole cell lysate

Lysates/proteins at 20 µg per lane.

Predicted band size: 227 kDa
Observed band size: 230 kDa

Exposure time: 20 seconds

ab205470 was shown to specifically react with non-muscle Myosin IIA in wild-type HAP1 cells as signal was lost in MYH9 (non-muscle Myosin IIA) knockout cells. Wild-type and MYH9 (non-muscle Myosin IIA) knockout samples were subjected to SDS-PAGE. Ab205470 and **ab184095** (Mouse monoclonal [mAbcam 9484] to GAPDH - Loading Control (Alexa Fluor® 680) loading control) were incubated overnight at 4°C at 1/5000 dilution and 1/20000 dilution respectively. The loading control was imaged using the Licor Odyssey CLx prior to blots being developed with ECL technique.

Western blot - HRP Anti-non-muscle Myosin IIA antibody [EPR8965] (ab205470)

All lanes : HRP Anti-non-muscle Myosin IIA antibody [EPR8965] (ab205470) at 1/5000 dilution

Lane 1 : Jurkat (Human T cell lymphoblast-like cell line) Whole Cell Lysate
Lane 2 : Kidney (Human) Tissue Lysate - fetal normal tissue
Lane 3 : A431 (Human epithelial carcinoma cell line) Whole Cell Lysate

Lysates/proteins at 10 µg per lane.

Developed using the ECL technique.

Performed under reducing conditions.

Predicted band size: 227 kDa
Observed band size: 230 kDa

Exposure time: 15 seconds

This blot was produced using a 3-8% Tris Acetate gel under the TA buffer system. The gel was run at 150V for 60 minutes before being transferred onto a Nitrocellulose membrane at 30V for 70 minutes. The membrane was then blocked for an hour using 3% milk before being incubated with ab205470 overnight at 4°C. Antibody binding was visualised using ECL development solution **ab133406**.

HRP Anti-non-muscle Myosin IIA antibody [EPR8965] (ab205470)

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