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Product datasheet

Anti-non-muscle Myosin IIA antibody ab236085

2 Images

Overview

Product name	Anti-non-muscle Myosin IIA antibody
Description	Rabbit polyclonal to non-muscle Myosin IIA
Host species	Rabbit
Tested applications	**Suitable for:** IHC-P, ICC/IF
Species reactivity	**Reacts with:** Human
Immunogen	Recombinant fragment corresponding to Human non-muscle Myosin IIA aa 2-241. Database link: P35579
Positive control	IHC-P: Human kidney tissue. ICC/IF: HeLa cells.

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 long term. Avoid freeze / thaw cycle.
Storage buffer	pH: 7.4 Preservative: 0.03% Proclin Constituents: PBS, 50% Glycerol
Purity	Protein G purified
Purification notes	Purity >95%
Clonality	Polyclonal
Isotype	IgG

Applications

Our Abpromise guarantee covers the use of **ab236085** 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
IHC-P		1/20 - 1/200.

Application	Abreviews	Notes
ICC/IF		1/50 - 1/200.

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

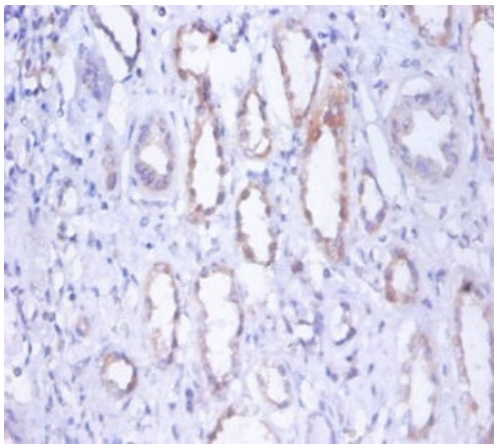
Contains 1 IQ domain.
Contains 1 myosin head-like domain.

Domain

The rodlike tail sequence is highly repetitive, showing cycles of a 28-residue repeat pattern

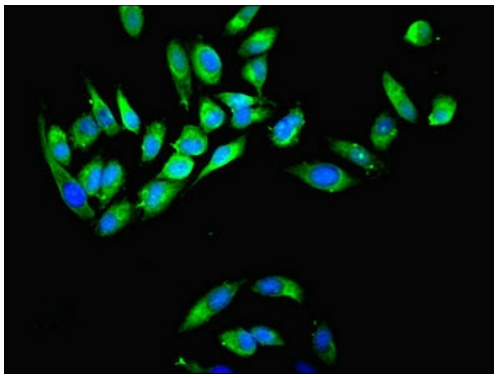
	composed of 4 heptapeptides, characteristic for alpha-helical coiled coils.
Post-translational modifications	ISGylated.

Images



Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-non-muscle Myosin IIA antibody (ab236085)

Paraffin-embedded human kidney tissue stained for non-muscle Myosin IIA using ab236085 at 1/100 dilution in immunohistochemical analysis.



Immunocytochemistry/ Immunofluorescence - Anti-non-muscle Myosin IIA antibody (ab236085)

HeLa (Human epithelial cell line from cervix adenocarcinoma) cells labeling non-muscle Myosin IIA (green) using ab236085 at 1/100 dilution in ICC/IF. An Alexa-Fluor®488-conjugated Goat Anti-Rabbit IgG (H+L) was used as the secondary antibody.

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