

# Myosin Ila (Phospho Ser628) Rabbit pAb

CatalogNo: YP1409

## Key Features

### Host Species

- Rabbit

### Reactivity

- Human, Mouse, Rat

### Applications

- WB, IHC

### MW

- 215kD (Observed)

### Isotype

- IgG

## Storage

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

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

## Recommended Dilution Ratios

**WB 1:500-2000**

**IHC 1:50-300**

## Basic Information

**Clonality** Polyclonal

## Immunogen Information

**Immunogen** Synthesized phospho peptide around human Myosin Ila (Ser1943)

**Specificity** This antibody detects endogenous levels of Human Mouse Rat Myosin Ila (phospho-Ser1943)

## Target Information

**Gene name** MYH9

**Protein Name** Myosin IIa (Ser1943)

Organism	Gene ID	UniProt ID
Human	<a href="#">4627</a> ;	<a href="#">P35579</a> ;
Mouse	<a href="#">17886</a> ;	<a href="#">Q8VDD5</a> ;
Rat	<a href="#">25745</a> ;	<a href="#">Q62812</a> ;

**Cellular Localization**

Cytoplasm, cytoskeleton . Cytoplasm, cell cortex . Cytoplasmic vesicle, secretory vesicle, Cortical granule . Colocalizes with actin filaments at lamellipodia margins and at the leading edge of migrating cells (PubMed:20052411). In retinal pigment epithelial cells, predominantly localized to stress fiber-like structures with some localization to cytoplasmic puncta (PubMed:27331610). .

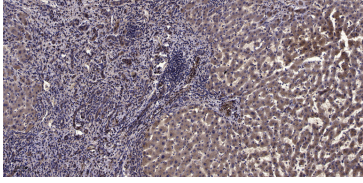
**Tissue specificity** In the kidney, expressed in the glomeruli. Also expressed in leukocytes.

**Function**

Disease: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.,Disease:Defects in MYH9 are the cause of Epstein syndrome (EPS) [MIM:153650]. EPS is an autosomal dominant disorder characterized by the association of macrothrombocytopenia, sensorineural hearing loss and nephritis.,Disease: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.,Disease: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 macrothrombocytopenia and progressive sensorineural hearing loss without renal dysfunction.,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 leukocyte inclusions appearing as highly parallel paracrystalline bodies.,Disease:Defects in MYH9 are the cause of non-syndromic sensorineural 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.,Disease: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.,Disease: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.,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.,Function:Cellular myosin that appears to play a role in cytokinesis, cell shape, and specialized functions such as secretion and capping.,similarity:Contains 1 IQ domain.,similarity:Contains 1 myosin head-like domain.,subunit:Interacts with PDLIM2 (By similarity). Myosin is a hexameric protein that consists of 2 heavy chain subunits (MHC), 2 alkali light chain subunits (MLC) and 2 regulatory light chain subunits (MLC-2).,tissue specificity:In the kidney, expressed in the glomeruli. Also expressed in leukocytes.,

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## Validation Data



Immunohistochemical analysis of paraffin-embedded human liver cancer. 1, Antibody was diluted at 1:200 (4°C overnight). 2, Tris-EDTA, pH 9.0 was used for antigen retrieval. 3, Secondary antibody was diluted at 1:200 (room temperature, 45min).

## Contact information

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Please scan the QR code to access additional product information:  
**Myosin IIa (Phospho Ser628) Rabbit pAb**

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