

Product Name: Myosin VI Rabbit Polyclonal Antibody**Catalog #: APRab14347**

For research use only.

Summary

Description	Rabbit polyclonal Antibody
Host	Rabbit
Application	WB,IHC,ICC/IF,ELISA
Reactivity	Human,Mouse,Rat
Conjugation	Unconjugated
Modification	Unmodified
Isotype	IgG
Clonality	Polyclonal
Form	Liquid
Concentration	1mg/ml
Storage	Aliquot and store at -20°C (valid for 12 months). Avoid freeze/thaw cycles.
Shipping	Ice bags
Buffer	Liquid in PBS containing 50% glycerol, 0.5% protective protein and 0.02% New type preservative N.
Purification	Affinity purification

Application

Dilution Ratio	WB 1:500-1:2000,IHC 1:100-1:300,ICC/IF 1:50-1:200,ELISA 1:5000-1:10000
Molecular Weight	149kDa

Antigen Information

Gene Name	MYO6
Alternative Names	MYO6; KIAA0389; Unconventional myosin-VI; Unconventional myosin-6
Gene ID	4646.0
SwissProt ID	Q9UM54
Immunogen	Synthesized peptide derived from Myosin VI . at AA range: 40-120

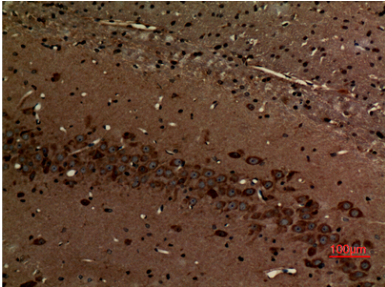
Background

myosin VI(MYO6) Homo sapiens This gene encodes a reverse-direction motor protein that moves toward the minus end of actin filaments and plays a role in intracellular vesicle and organelle transport. The protein consists of a motor domain

containing an ATP- and an actin-binding site and a globular tail which interacts with other proteins. This protein maintains the structural integrity of inner ear hair cells and mutations in this gene cause non-syndromic autosomal dominant and recessive hearing loss. Alternative splicing results in multiple transcript variants encoding distinct isoforms. [provided by RefSeq, Jul 2014],disease:Defects in MYO6 are the cause of non-syndromic sensorineural deafness autosomal dominant type 22 (DFNA22) [MIM:606346]. DFNA22 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. DFNA22 is progressive and postlingual, with onset during childhood. By the age of approximately 50 years, affected individuals invariably have profound sensorineural deafness.,disease:Defects in MYO6 are the cause of non-syndromic sensorineural deafness autosomal recessive type 37 (DFNB37) [MIM:607821],disease:Defects in MYO6 are the cause of sensorineural deafness with hypertrophic cardiomyopathy (DFNHCM) [MIM:606346],domain:Divided into three regions: a N-terminal motor (head) domain, followed by a neck domain consisting of a calmodulin-binding linker domain and a single IQ motif, and a C-terminal tail region with a coiled-coil and a unique globular domain required for interaction with other proteins.,function:Myosins are actin-based motor molecules with ATPase activity. Unconventional myosins serve in intracellular movements. Myosin 6 is a reverse-direction motor protein that moves towards the minus-end of actin filaments. Has slow rate of actin-activated ADP release due to weak ATP binding. Functions in a variety of intracellular processes such as vesicular membrane trafficking and cell migration. Required for the structural integrity of the Golgi apparatus via the p53-dependent pro-survival pathway. Appears to be involved in a very early step of clathrin-mediated endocytosis in polarized epithelial cells. May act as a regulator of F-actin dynamics. May play a role in transporting DAB2 from the plasma membrane to specific cellular targets. Required for structural integrity of inner ear hair cells.,PTM:Phosphorylation in the motor domain, induced by EGF, results in translocation of MYO6 from the cell surface to membrane ruffles and affects F-actin dynamics. Phosphorylated in vitro by p21-activated kinase (PAK),similarity:Contains 1 IQ domain.,similarity:Contains 1 myosin head-like domain.,subcellular location:Also present in endocytic vesicles, and membrane ruffles. Translocates from membrane ruffles, endocytic vesicles and cytoplasm to Golgi apparatus, perinuclear membrane and nucleus through induction by p53 and p53-induced DNA damage. Recruited into membrane ruffles from cell surface by EGF-stimulation. Colocalizes with DAB2 in clathrin-coated pits/vesicles.,subunit:Homodimer. Binding to calmodulin through a unique insert, not found in other myosins, located in the neck region between the motor domain and the IQ domain appears to contribute to the directionality reversal. This interaction occurs only if the C-terminal lobe of calmodulin is occupied by calcium. Interaction with F-actin/ACTN1 occurs only at the apical brush border domain of the proximal tubule cells (By similarity). Interacts with DAB2. In vitro, the C-terminal globular tail binds a C-terminal region of DAB2. Interacts with CFTR. Forms a complex with CFTR and DAB2 in the apical membrane of epithelial cells.,tissue specificity:Expressed in most tissues examined including heart, brain, placenta, pancreas, spleen, thymus, prostate, testis, ovary, small intestine and colon. Highest levels in brain, pancreas, testis and small intestine. Also expressed in fetal brain and cochlea. Isoform 1 and isoform 2, containing the small insert, and isoform 4, containing neither insert, are expressed in unpolarized epithelial cells.,

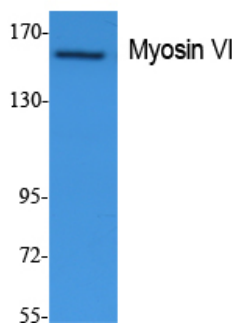
Research Area

Image Data



Immunohistochemical analysis of paraffin-embedded rat-brain, antibody was diluted at 1:100

(kD)



Western Blot analysis of extracts from Jurkat cells, using Myosin VI Polyclonal Antibody. Secondary antibody was diluted at 1:20000