

Product datasheet

Anti-LIS1 antibody ab2607

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Overview

Product name	Anti-LIS1 antibody
Description	Rabbit polyclonal to LIS1
Host species	Rabbit
Tested applications	Suitable for: WB, IHC-P, ICC/IF, IP
Species reactivity	Reacts with: Mouse, Human Predicted to work with: Rat, Rabbit, Chicken, Guinea pig, Cow, Dog, Turkey, Pig, Xenopus laevis, Chimpanzee, Zebrafish, Rhesus monkey, Gorilla, Orangutan, Xenopus tropicalis
Immunogen	Synthetic peptide corresponding to Human LIS1. Represents a portion of human Lissencephaly 1 (Lis 1) protein encoded in part by exons 3 and 4.
Positive control	TF-1 (erythroleukemia cell line) cell lysate.

Properties


Form	Liquid
Storage instructions	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
Storage buffer	Preservative: 0.1% Sodium azide Constituents: 0.021% PBS, 1.764% Sodium citrate, 1.815% Tris
Purification notes	Affinity purified using the immunising peptide immobilized on solid support.
Clonality	Polyclonal
Isotype	IgG

Applications

Our [Abpromise guarantee](#) covers the use of **ab2607** 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		
IHC-P		

Application	Abreviews	Notes
ICC/IF		

IP

Application notes

ICC/IF: Use at an assay dependent dilution (see PMID: 17314247).

IP: Use at a concentration of 2 - 10 µg/ml.

WB: 1/300 - 1/3000. Suggested working dilution of 1/1000 (see PMID: 17314247).

IHC-P: Use at a concentration of 2 µg/ml. Perform heat mediated antigen retrieval before commencing with IHC staining protocol.

Not yet tested in other applications.

Optimal dilutions/concentrations should be determined by the end user.

Target

Function

Required for proper activation of Rho GTPases and actin polymerization at the leading edge of locomoting cerebellar neurons and postmigratory hippocampal neurons in response to calcium influx triggered via NMDA receptors. Non-catalytic subunit of an acetylhydrolase complex which inactivates platelet-activating factor (PAF) by removing the acetyl group at the SN-2 position (By similarity). Positively regulates the activity of the minus-end directed microtubule motor protein dynein. May enhance dynein-mediated microtubule sliding by targeting dynein to the microtubule plus end. Required for several dynein- and microtubule-dependent processes such as the maintenance of Golgi integrity, the peripheral transport of microtubule fragments and the coupling of the nucleus and centrosome. Required during brain development for the proliferation of neuronal precursors and the migration of newly formed neurons from the ventricular/subventricular zone toward the cortical plate. Neuronal migration involves a process called nucleokinesis, whereby migrating cells extend an anterior process into which the nucleus subsequently translocates. During nucleokinesis dynein at the nuclear surface may translocate the nucleus towards the centrosome by exerting force on centrosomal microtubules. May also play a role in other forms of cell locomotion including the migration of fibroblasts during wound healing.

Tissue specificity

Fairly ubiquitous expression in both the frontal and occipital areas of the brain.

Involvement in disease

Defects in PFAFH1B1 are the cause of lissencephaly type 1 (LIS1) [MIM:607432]; also known as classic lissencephaly. LIS1 is characterized by agyria or pachgyria and disorganization of the clear neuronal lamination of normal six-layered cortex. The cortex is abnormally thick and poorly organized with 4 primitive layers. LIS1 is associated with enlarged and dysmorphic ventricles and often hypoplasia of the corpus callosum.

Defects in PFAFH1B1 are the cause of subcortical band heterotopia (SBH) [MIM:607432]. SBH is a mild brain malformation of the lissencephaly spectrum. It is characterized by bilateral and symmetric ribbons of gray matter found in the central white matter between the cortex and the ventricular surface.

Defects in PFAFH1B1 are a cause of Miller-Dieker lissencephaly syndrome (MDLS) [MIM:247200]. MDLS is a contiguous gene deletion syndrome of chromosome 17p13.3, characterized by classical lissencephaly and distinct facial features. Additional congenital malformations can be part of the condition.

Sequence similarities

Belongs to the WD repeat LIS1/nudF family.

Contains 1 LisH domain.

Contains 7 WD repeats.

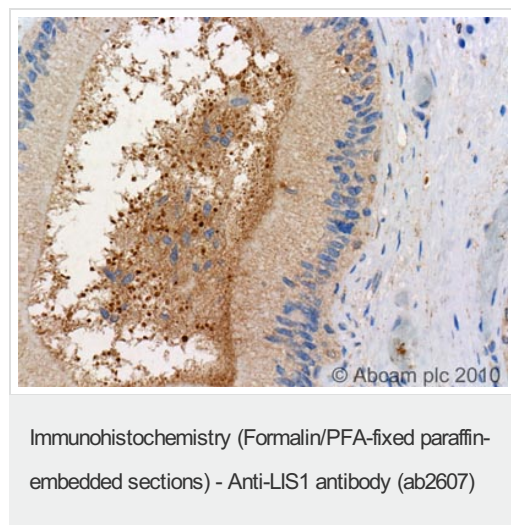
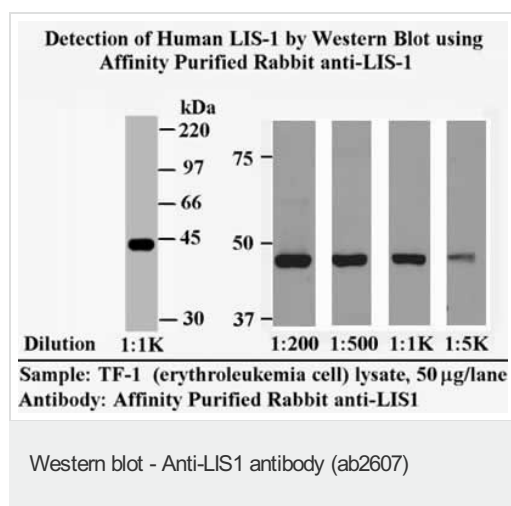
Domain

Dimerization mediated by the LisH domain may be required to activate dynein.

Cellular localization

Cytoplasm > cytoskeleton. Cytoplasm > cytoskeleton > centrosome. Cytoplasm > cytoskeleton > spindle. Nucleus membrane. Redistributes to axons during neuronal development. Also localizes to the microtubules of the manchette in elongating spermatids and to the meiotic spindle in spermatocytes (By similarity). Localizes to the plus end of microtubules and to the centrosome. May localize to the nuclear membrane.

Images



ab2607 (2µg/ml) staining Lis1 in human testis using an automated system (DAKO Autostainer Plus). Using this protocol there is strong staining of cytoplasmic compartment of the mature spermatocyte cells.

Sections were rehydrated and antigen retrieved with the Dako 3 in 1 AR buffer EDTA pH 9.0 in a DAKO PT link. Slides were peroxidase blocked in 3% H₂O₂ in methanol for 10 mins. They were then blocked with Dako Protein block for 10 minutes (containing casein 0.25% in PBS) then incubated with primary antibody for 20 min and detected with Dako envision flex amplification kit for 30 minutes. Colorimetric detection was completed with Diaminobenzidine for 5 minutes. Slides were counterstained with Haematoxylin and coverslipped under DePeX. Please note that, for manual staining, optimization of primary antibody concentration and incubation time is recommended. Signal amplification may be required.

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