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LIS1 Antibody

Catalog # ASC10564

Product Information

Application	WB, IF, ICC, E
Primary Accession	P43034
Other Accession	P43034, 1170794
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	46638
Concentration (mg/ml)	1 mg/mL
Conjugate	Unconjugated
Application Notes	LIS1 antibody can be used for detection of LIS1 by Western blot at 0.5 - 1 g/mL. Antibody can also be used for immunocytochemistry starting at 2.5 g/mL. For immunofluorescence start at 20 g/mL.

Additional Information

Gene ID	5048
Other Names	Platelet-activating factor acetylhydrolase IB subunit alpha {ECO:0000255|HAMAP-Rule:MF_03141}, Lissencephaly-1 protein {ECO:0000255|HAMAP-Rule:MF_03141}, LIS-1 {ECO:0000255|HAMAP-Rule:MF_03141}, PAF acetylhydrolase 45 kDa subunit {ECO:0000255|HAMAP-Rule:MF_03141}, PAF-AH 45 kDa subunit {ECO:0000255|HAMAP-Rule:MF_03141}, PAF-AH alpha {ECO:0000255|HAMAP-Rule:MF_03141}, PAFAH alpha {ECO:0000255|HAMAP-Rule:MF_03141}, PAFAH1B1 {ECO:0000255|HAMAP-Rule:MF_03141}
Target/Specificity	PAFAH1B1;
Reconstitution & Storage	LIS1 antibody can be stored at 4℃ for three months and -20℃, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.
Precautions	LIS1 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	LIS1
Function	Regulatory subunit (beta subunit) of the cytosolic type I platelet-activating factor (PAF) acetylhydrolase (PAF-AH (I)), an enzyme that catalyzes the

	hydrolyze of the acetyl group at the sn-2 position of PAF and its analogs and participates in PAF inactivation. Regulates the PAF-AH (I) activity in a catalytic dimer composition- dependent manner (By similarity). 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 (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. Required for dynein recruitment to microtubule plus ends and BICD2-bound cargos (PubMed:22956769). May modulate the Reelin pathway through interaction of the PAF-AH (I) catalytic dimer with VLDLR (By similarity).
Cellular Location	Cytoplasm, cytoskeleton. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Cytoplasm, cytoskeleton, spindle {ECO:0000255|HAMAP-Rule:MF_03141}. Nucleus membrane {ECO:0000255|HAMAP- Rule:MF_03141}. Note=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.
Tissue Location	Fairly ubiquitous expression in both the frontal and occipital areas of the brain

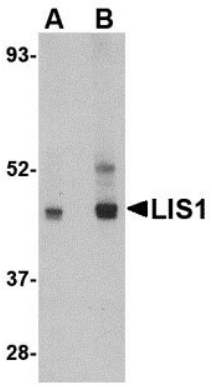
Background

LIS1 Antibody: Lissencephaly is a severe brain developmental disease characterized by the mislocalization of cortical neurons, a smooth cerebral surface, mental retardation, and seizures. Classical lissencephaly is caused by sporadic mutations in the LIS1 gene. While LIS1 is known to act in a pathway deactivating the lipid messenger platelet-activating factor, LIS1 forms a complex with Nudel and 14-3-3epsilon which is then transported from neuronal cell bodies through the actions of DISC1 and KIF5A, a microtubule-dependent directed motor protein kinesin. Decreased expression of LIS1 blocked neural stem cell division, morphogenesis, and motility, suggesting that LIS1 plays an important role in neuronal cell proliferation and localization in the developing brain. At least two isoforms of LIS1 are known to exist.

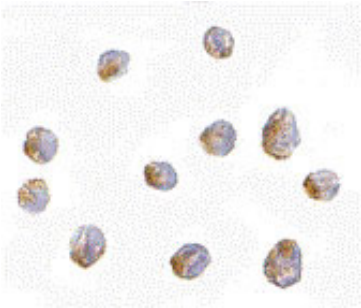
References

McManus MF and Golden JA. Neuronal migration in developmental disorders. J. Child Neurol.2005; 20:280-6.
Reiner O, Carrozzo R, Shen Y, et al. Isolation of a Miller-Dieker lissencephaly gene containing G protein b-subunit-like repeats. Nature1993; 364:717-21.
Hattori M, Adachi H, Tsujimoto M, et al. Miller-Dieker lissencephaly gene encodes a subunit of brain platelet activating factor. Nature1994; 370:216-8.
Taya S, Shinoda T, Tsuboi D, et al. DISC1 regulates the transport of the NUDEL/LIS1/14-3-3e complex through kinesin-1. J. Neurosci.2007; 27:15-26.

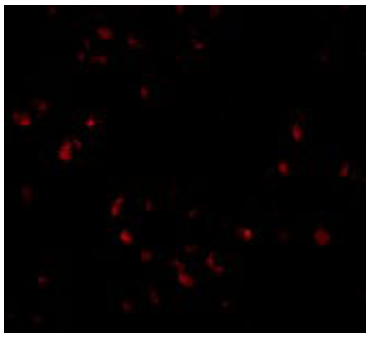
Images



Western blot analysis of LIS1 in HeLa cell lysate with LIS1 antibody at (A) 0.5 and (B) 1 µg/mL.



Immunocytochemistry of LIS1 in Jurkat cells with LIS1 antibody at 2.5 µg/mL.



Immunofluorescence of LIS1 in Jurkat cells with LIS1 antibody at 20 µg/mL.