abcam

Product datasheet

Human L1CAM knockout HeLa cell lysate ab263786

3 Images

Overview

Product name	Human L1CAM knockout HeLa cell lysate	
Product overview		
	Knockout cell lysate achieved by CRISPR/Cas9.	
Parental Cell Line	HeLa	
Organism	Human	
Mutation description	Knockout achieved by using CRISPR/Cas9, 154 bp insertion in exon1.	
Passage number	<20	
Knockout validation	Sanger Sequencing, Western Blot (WB)	
Reconstitution notes	To use as WB control, resuspend the lyophilizate in 50 µL of LDS* Sample Buffer to have a final concentration of 2 mg/ml. For reducing conditions, we recommend a final concentration of 0.1 M DTT. *Usage of SDS sample buffer is not recommended with these lyophilized lysates.	
Notes	Lysate preparation: Our lysates are made using RIPA buffer to which we add a protease inhibitor cocktail and phosphatase inhibitor cocktail (ratio: 300:100:10). <i>This means that the protein of interest is denatured.</i> If you require a native form of the protein please use the live cell version - found <u>here</u> . Please refer to our lysis protocol for further details on how our lysates are prepared.	
	User storage instructions: Lyophilizate may be stored at 4°C. After reconstitution, store at - 20°C for short-term storage or -80°C for long-term storage.	
	Access thousands of knockout cell lysates, generated from commonly used cancer cell lines. See here for more information on knockout cell lysates.	
	Abcam has not and does not intend to apply for the REACH Authorisation of customers' uses of products that contain European Authorisation list (Annex XIV) substances. It is the responsibility of our customers to check the necessity of application of REACH Authorisation, and any other relevant authorisations, for their intended uses.	
	This product is subject to limited use licenses from The Broad Institute, ERS Genomics Limited and Sigma-Aldrich Co. LLC, and is developed with patented technology. For full details of the licenses and patents please refer to our limited use license and patent pages .	
Tested applications	Suitable for: WB	

Properties

Storage instructions	Store at -80°C. Please refer to protocols.	Store at -80°C. Please refer to protocols.		
Components		1 kit		
ab255505 - Human L1CAM knockout HeLa cell lysate		1 x 100µg		
ab255929 - Human wild-type HeLa cell lysate		1 x 100µg		
Cell type	epithelial			
Disease	Adenocarcinoma			
Gender	Female			
STR Analysis	Amelogenin X D5S818: 11, 12 D13S317: 12, 13.3 D7S820: 8, 12 D16S539: 9, 10 vWA: 16, 18 TH01: 7 TPOX: 8,12 CSF1PO: 9, 10			
Target				
Function	Cell adhesion molecule with an important role in the development of the nervous system. Involved in neuron-neuron adhesion, neurite fasciculation, outgrowth of neurites, etc. Binds to axonin on neurons.			
Involvement in disease	 Defects in L1CAM are the cause of hydrocephalus due to stenosis of the (HSAS) [MIM:307000]. Hydrocephalus is a condition in which abnormal is cerebrospinal fluid in the brain causes increased intracranial pressure in usually due to blockage of cerebrospinal fluid outflow in the brain ventricle space at the base of the brain. In children is typically characterized by ere prominence of the forehead, brain atrophy, mental deterioration, and core syndrome includes incontinence, imbalance, and dementia. HSAS is charetardation and enlarged brain ventricles. Defects in L1CAM are the cause of mental retardation-aphasia-shuffling syndrome (MASA) [MIM:303350]; also known as corpus callosum hypop retardation, adducted thumbs, spastic paraparesis, and hydrocephalus of MASA is an X-linked recessive syndrome with a highly variable clinical se features include spasticity and hyperreflexia of lower limbs, shuffling gait aphasia and adducted thumbs. The features of spasticity have been refe spastic paraplegia type 1 (SPG1). Some patients manifest corpus callosum hydrocephalus. Inter- and intrafamilial variability is very wide, such that pa hydrocephalus, MASA, SPG1, and agenesis of corpus callosum can be family. Defects in L1CAM are the cause of spastic paraplegia X-linked type 1 (Spastic paraplegia is a degenerative spinal cord disorder characterized progressive weakness and spasticity of the lower limbs. Note=Defects in L1CAM are the cause of spastic paraplegia X-linked type 1 (Spastic paraplegia is a degenerative spinal cord disorder characterized progressive weakness and spasticity of the lower limbs. Note=Defects in L1CAM are ta cause of partial agenesis of the corpus callosur [MIM:304100]. A syndrome characterized by partial corpus callosur age inferior vermis and cerebellum, mental retardation, seizures and spastici microcephaly, unusual facies, and Hirschsprung disease in some patient 	accumulation of side the skull. This is es or in the subarachno largement of the head, nulsions. In adults the aracterized by mental gait-adducted thumbs lasia, psychomotor or CRASH syndrome. spectrum. Main clinical , mental retardation, erred to as complicated sum hypoplasia and atients with present within the same SPG1) [MIM:303350]. I by a slow, gradual, difying the effects of sis. m (ACCPX) enesis, hypoplasia of ty. Other features inclue		
Sequence similarities	Belongs to the immunoglobulin superfamily. L1/neurofascin/NgCAM family. Contains 5 fibronectin type-III domains.			

Applications

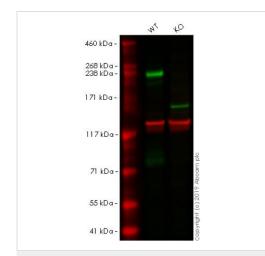
Cellular localization

 The Abpromise guarantee
 Our Abpromise guarantee
 covers the use of ab263786 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		Use at an assay dependent concentration.

Images

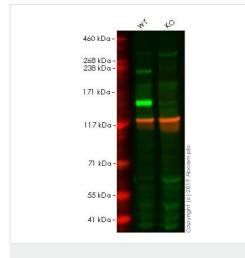


Western blot - Human L1CAM knockout HeLa cell lysate (ab263786) Lane 1: Wild-type HeLa cell lysate (20 µg)

Lane 2: L1CAM knockout HeLa cell lysate (20 µg)

Lanes 1 - 2: Merged signal (red and green). Green - <u>ab182407</u> observed at 220 kDa. Red - loading control, <u>ab130007</u> observed at 125 kDa.

<u>ab182407</u> was shown to react with L1CAM in wild-type HeLa. Loss of signal was observed when knockout cell line <u>ab255401</u> (knockout cell lysate ab263786) was used. Wild-type and L1CAM knockout samples were subjected to SDS-PAGE. <u>ab182407</u> and Anti-Vinculin antibody [VIN-54] (<u>ab130007</u>) were incubated overnight at 4^°C at 1 in 5000 dilution and 1 in 20000 dilution respectively. Blots were developed with Goat anti-Rabbit IgG H&L (IRDye[®] 800CW) preadsorbed (<u>ab216773</u>) and Goat anti-Mouse IgG H&L (IRDye[®] 680RD) preadsorbed (<u>ab216776</u>) secondary antibodies at 1 in 20000 dilution for 1 hour at room temperature before imaging.



Western blot - Human L1CAM knockout HeLa cell Iysate (ab263786) Lane 1: Wild-type HeLa cell lysate (20 µg)

Lane 2: L1CAM knockout HeLa cell lysate (20 µg)

Lanes 1 - 2: Merged signal (red and green). Green - <u>ab208155</u> observed at 220 kDa. Red - loading control, <u>ab130007</u> observed at 125 kDa.

<u>ab208155</u> was shown to react with L1CAM in wild-type HeLa. Loss of signal was observed when knockout cell line <u>ab255401</u> (knockout cell lysate ab263786) was used. Wild-type and L1CAM knockout samples were subjected to SDS-PAGE. <u>ab208155</u> and Anti-Vinculin antibody [VIN-54] (<u>ab130007</u>) were incubated overnight at 4^°C at 1 in 1000 dilution and 1 in 20000 dilution respectively. Blots were developed with Goat anti-Rabbit lgG H&L (IRDye[®] 800CW) preadsorbed (<u>ab216773</u>) and Goat anti-Mouse lgG H&L (IRDye[®] 680RD) preadsorbed (<u>ab216776</u>) secondary antibodies at 1 in 20000 dilution for 1 hour at room temperature before imaging.

Allele-1: 154 bp insertion in exon1

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Sanger Sequencing - Human L1CAM knockout HeLa

cell lysate (ab263786)

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