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Product datasheet

Anti-L1CAM antibody [UJ127.11] ab20148

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Overview

Product name	Anti-L1CAM antibody [UJ127.11]
Description	Mouse monoclonal [UJ127.11] to L1CAM
Host species	Mouse
Specificity	UJ127.11 may be useful in the diagnosis of embryonic tumours (e.g. neuroblastoma).
Tested applications	**Suitable for:** WB
Species reactivity	**Reacts with:** Human
Immunogen	Tissue, cells or virus corresponding to Human L1CAM. Homogenous suspension of 16 week human foetal brain. Database link: **P32004**
General notes	L1CAM can be detected between 200-220 kD. In brain samples it is typically seen at ~ 200 kD. When the protein is overexpressed in vitro it is often detected as a doublet with bands at 200 and 220 kD. The unglycosylated, unprocessed L1CAM is ~ 140-150 kDa. The protein has 21 putative N-glycosylation sites on the extracellular portion of the protein which, when they are all glycosylated, results in a detected MW of 200-220 kD depending upon how many residues are actually glycosylated. L1CAM can be cleaved by the metalloprotease ADAM10 resulting in fragments of 180 kD and 40 kD. L1CAM can also be cleaved by plasmin resulting in fragments of 140 kD and 80 kD. In theory, therefore, one could detect bands at ~220, 200, 180, 140, 80 and 40 kD. The Life Science industry has been in the grips of a reproducibility crisis for a number of years. Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets your needs before purchasing. If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be found below, along with publications, customer reviews and Q&As

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Store at -20°C or -80°C. Avoid freeze / thaw cycle.
Storage buffer	Preservative: 0.02% Sodium azide

	Constituent: 99.98% PBS
Purity	Protein A/G purified
Clonality	Monoclonal
Clone number	UJ127.11
Myeloma	P3x63-Ag8.653
Isotype	IgG1
Light chain type	unknown

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab20148 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	★★★★★ (1)	Use at an assay dependent concentration. Predicted molecular weight: 200-220 kDa. It may also detect smaller cleavage fragments (please see Notes below).

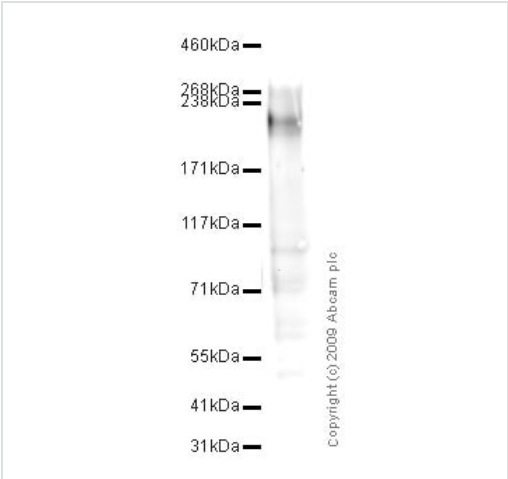
Target

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 aqueduct of Sylvius (HSAS) [MIM:307000]. Hydrocephalus is a condition in which abnormal accumulation of cerebrospinal fluid in the brain causes increased intracranial pressure inside the skull. This is usually due to blockage of cerebrospinal fluid outflow in the brain ventricles or in the subarachnoid space at the base of the brain. In children is typically characterized by enlargement of the head, prominence of the forehead, brain atrophy, mental deterioration, and convulsions. In adults the syndrome includes incontinence, imbalance, and dementia. HSAS is characterized by mental retardation and enlarged brain ventricles.
Defects in L1CAM are the cause of mental retardation-aphasia-shuffling gait-adducted thumbs syndrome (MASA) [MIM:303350]; also known as corpus callosum hypoplasia, psychomotor retardation, adducted thumbs, spastic paraparesis, and hydrocephalus or CRASH syndrome. MASA is an X-linked recessive syndrome with a highly variable clinical spectrum. Main clinical features include spasticity and hyperreflexia of lower limbs, shuffling gait, mental retardation, aphasia and adducted thumbs. The features of spasticity have been referred to as complicated spastic paraplegia type 1 (SPG1). Some patients manifest corpus callosum hypoplasia and hydrocephalus. Inter- and intrafamilial variability is very wide, such that patients with hydrocephalus, MASA, SPG1, and agenesis of corpus callosum can be present within the same family.
Defects in L1CAM are the cause of spastic paraplegia X-linked type 1 (SPG1) [MIM:303350]. Spastic paraplegia is a degenerative spinal cord disorder characterized by a slow, gradual, progressive weakness and spasticity of the lower limbs.
Note=Defects in L1CAM may contribute to Hirschsprung disease by modifying the effects of Hirschsprung disease-associated genes to cause intestinal aganglionosis.

	Defects in L1CAM are a cause of partial agenesis of the corpus callosum (ACCPX) [MIM:304100]. A syndrome characterized by partial corpus callosum agenesis, hypoplasia of inferior vermis and cerebellum, mental retardation, seizures and spasticity. Other features include microcephaly, unusual facies, and Hirschsprung disease in some patients.
Sequence similarities	Belongs to the immunoglobulin superfamily. L1/neurofascin/NgCAM family. Contains 5 fibronectin type-III domains. Contains 6 Ig-like C2-type (immunoglobulin-like) domains.
Cellular localization	Cell membrane.

Images



Western blot - Anti-L1CAM antibody [UJ127.11] (ab20148)

Anti-L1CAM antibody [UJ127.11] (ab20148) at 5 µg/ml + SK N BE (Human neuroblastoma) Whole Cell Lysate at 10 µg

Secondary

Goat polyclonal to Mouse IgG - H&L - Pre-Adsorbed (HRP at 1/3000 dilution

Predicted band size: 200-220 kDa
Observed band size: 200-220 kDa

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