

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse CHL-1/L1CAM-2 in Western blots. In Western blots, approximately 75% cross-reactivity with recombinant human CHL-1.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse CHL-1/L1CAM-2
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	Recombinant Mouse CHL-1/L1CAM-2 (Catalog # 2147-CH)

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <ul style="list-style-type: none"> • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Close homolog of L1 (CHL-1), also known as cell adhesion L1-like (CALL) and L1 cell adhesion molecule 2 (L1CAM-2), belongs to the L1 subfamily of immunoglobulin (Ig) superfamily cell adhesion molecules, which also includes L1, neurofascin and NgCAM-related cell adhesion molecule (NrCAM) (1-3). These molecules are type I transmembrane proteins that have 6 Ig-like domains and 4-5 fibronectin type III-like (FNIII) domains in their extracellular regions. They also share a highly conserved cytoplasmic region of approximately 110 amino acid (aa) residues containing an ankyrin-binding site. CHL-1 is expressed as a highly glycosylated 185 kDa transmembrane protein by subpopulations of neurons and glia of the central and peripheral nervous system (4, 5). Ectodomain shedding via the metalloprotease-disintegrin ADAM8 releases 165 kDa and 125 kDa soluble CHL-1 fragments, which can diffuse away to function at distant sites (6). CHL-1 is not capable of homotypic interactions, but an extracellular binding partner of CHL-1 has not been identified (4). Human *CHL1* has been mapped to chromosome 3p26 and is a candidate gene for 3p⁻ syndrome characterized by mental impairment (7). A missense *CHL1* polymorphism associated increased risk of schizophrenia, has also been reported (8). The functional importance of CHL-1 in the nervous system is also evident in CHL-1 deficient mice, which display behavioral abnormalities and show misguided axons within the hippocampus and olfactory tract (9). Enhanced ectodomain-shedding of CHL-1 is also observed in Wobbler mice, the neurodegenerative mutant mice (6). *In vitro*, soluble or substrate-coated CHL-1 promotes neurite outgrowth and neuronal survival of both cerebellar and hippocampal neurons. Cell surface CHL-1 interacts with integrins in *cis* to potentiate integrin-dependent cell migration toward extracellular matrix proteins (10). For this enhanced cell motility, CHL-1 linkage to the actin cytoskeleton via interaction between ankyrin and the CHL-1 cytoplasmic region is required.

References:

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