

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Reelin in direct ELISAs.
Source	Monoclonal Mouse IgG ₁ Clone # 940209
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Chinese hamster ovary cell line CHO-derived recombinant human Reelin Ser1221-Gln2666 Accession # P78509
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

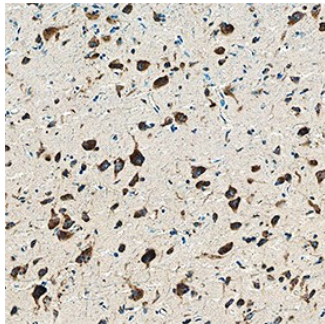
APPLICATIONS

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
Immunohistochemistry	5-15 µg/mL	See Below

DATA

Immunohistochemistry



Reelin in Human Brain. Reelin was detected in immersion fixed paraffin-embedded sections of human brain using Mouse Anti-Human Reelin Monoclonal Antibody (Catalog # MAB8546) at 5 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Mouse HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS002) and counterstained with hematoxylin (blue). Specific staining was localized to neuronal cytoplasm. View our protocol for [Chromogenic IHC Staining of Paraffin-embedded Tissue Sections](#).

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <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

Reelin is a secreted modular glycoprotein that exhibits serine protease activity and is crucial for brain development and function (1-3). It is composed of an N-terminal Reelin domain, eight EGF-like Reelin repeats (RR), and a highly basic C-terminal region (4-6). The N-terminal fragment is suggested to mediate dimerization/oligomerization and receptor recognition, the midpiece receptor binding, and the C-terminal fragment receptor signaling and recognition (1, 5, 7-9). Human Reelin is synthesized as a 3460 amino acid (aa) precursor protein with a molecular weight of approximately 410 kDa (4). During processing, it can be cleaved between RR2 and RR3 or between RR6 and RR7, producing a 180 kDa and a 330 kDa peptide, respectively (1, 6, 10). Within shared regions in the central fragment, human Reelin shares 95% aa sequence identity with mouse and rat Reelin.

Reelin is secreted by Cajal-Retzius cells in the embryo (1, 4, 11). In the adult, it is expressed in the subventricular zone, rostral migratory stream, olfactory bulb, and in the CA1, CA3, and dentate gyrus regions of the hippocampus, as well as in cerebellar granule cells, pyramidal cells of the entorhinal cortex, GABA interneurons, and glial cells (1, 6, 12, 13). Reelin utilizes the receptors VLDLR and ApoE R2, which have been suggested to have divergent roles in Reelin-mediated neuronal migration (1, 2, 6, 12). It has also been shown to interact with Integrin $\alpha\beta 1$ and APP (1, 6, 14, 15). During cortical plate development, Reelin controls cell-cell interactions critical for proper neuronal migration and positioning (1, 2, 4, 5, 11, 12, 16). In the adult, it plays a role in dendrite growth and maturation, and synapse formation (2, 6, 15). Additionally, Reelin has been shown to modulate synaptic transmission and plasticity by regulating the subunit composition and conductivity of NMDA receptors (2, 6, 17). Mutation of the *RELN* gene results in lissencephaly with cerebellar hypoplasia (11, 18). In addition, abnormal Reelin expression in the brain has been associated with a variety of cognitive pathological conditions including autism, schizophrenia, bipolar disorder, major depression, and Alzheimer's disease (1, 6, 11, 13, 19, 20). Peripherally, Reelin is important in the development of neuromuscular junctions. But instead of utilizing the locally expressed ApoE R2 and VLDLR, this function requires the serine protease activity of Reelin (3, 21).

References:

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