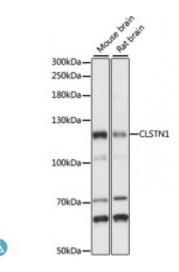
## **Anti-CLSTN1 Antibody**



**Description** 

This gene is a member of the calsyntenin family, a subset of the cadherin superfamily. The encoded transmembrane protein, also known as alcadeinalpha, is thought to bind to kinesin-1 motors to mediate the axonal anterograde transport of certain types of vesicle. Amyloid precursor protein (APP) is trafficked via these vesicles and so this protein is being investigated to see how it might contribute to the mechanisms underlying Alzheimer's disease. Alternative splicing results in multiple transcript variants.

Model STJ117600

**Host** Rabbit

**Reactivity** Mouse, Rat

**Applications** WB

Immunogen Recombinant fusion protein containing a sequence corresponding to amino

acids 29-350 of human CLSTN1 (NP\_055759.3).

**Gene ID** 22883

Gene Symbol <u>CLSTN1</u>

**Dilution range** WB 1:200 - 1:2000

**Tissue Specificity** Expressed in the brain and, a lower level, in the heart, skeletal muscle, kidney

and placenta, Accumulates in dystrophic neurites around the amyloid core of

Alzheimer disease senile plaques (at protein level)

**Purification** Affinity purification

**Note** For Research Use Only (RUO).

Protein Name Calsyntenin-1 Alcadein-alpha Alc-alpha Alzheimer-related cadherin-like

protein Non-classical cadherin XB31alpha

Molecular Weight 109.793 kDa

**Clonality** Polyclonal

**Conjugation** Unconjugated

**Isotype** IgG

**Formulation** PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

**Storage Instruction** Store at -20C. Avoid freeze / thaw cycles.

Database Links HGNC:174470MIM:611321

Alternative Names Calsyntenin-1 Alcadein-alpha Alc-alpha Alzheimer-related cadherin-like

protein Non-classical cadherin XB31alpha

**Function** Induces KLC1 association with vesicles and functions as a cargo in axonal

anterograde transport, Complex formation with APBA2 and APP, stabilizes APP metabolism and enhances APBA2-mediated suppression of beta-APP40 secretion, due to the retardation of intracellular APP maturation, In complex with APBA2 and C99, a C-terminal APP fragment, abolishes C99 interaction with PSEN1 and thus APP C99 cleavage by gamma-secretase, most probably through stabilization of the direct interaction between APBA2 and APP, The intracellular fragment AlcICD suppresses APBB1-dependent transactivation stimulated by APP C-terminal intracellular fragment (AICD), most probably by competing with AICD for APBB1-binding, May modulate calcium-

mediated postsynaptic signals,

**Cellular Localization** Endoplasmic reticulum membrane,

Post-translational Modifications Proteolytically processed under normal cellular conditions, A primary zeta-cleavage generates a large extracellular (soluble) N-terminal domain (sAlc) and a short C-terminal transmembrane fragment (CTF1), A secondary cleavage catalyzed by presenilin gamma-secretase within the transmembrane domain releases the beta-Alc-alpha chain in the extracellular milieu and produces an intracellular fragment (AlcICD), This processing is strongly suppressed in the tripartite complex formed with APBA2 and APP, which seems to prevent the association with PSEN1,

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