

Anti-APP Antibody (8U619)

Product Details

Ig Type:	Rabbit IgG
Reactivity:	Human, Mouse, Rat
Conjugation:	Unconjugated
Clone:	8U619
Purification:	Affinity-chromatography

Applications

Verified Activity:	1. Western Blot -Positive WB detected in: Hela whole cell lysate, A375 whole cell lysate, HEK293 whole cell lysate, Rat Brain whole cell lysate, Mouse Brain whole cell lysate -All lanes: Amyloid beta A4 antibody at 1:1000 -Secondary: Goat polyclonal to rabbit IgG at 1/50000 dilution -Predicted band size: 87, 35, 77, 79, 79, 81, 83, 85, 86, 73, 85 kDa -Observed band size: 100 kDa
	2. Immunofluorescence staining of Hela Cells with TMAH-00074 at 1:50, counter-stained with DAPI. The cells were fixed in 4% formaldehyde, permeated by 0.2% TritonX-100, and blocked in 10% normal Goat Serum. The cells were then incubated with the antibody overnight at 4°C. Nuclear DNA was labeled in blue with DAPI. The secondary antibody was FITC-conjugated AffiniPure Goat Anti-Rabbit IgG (H+L).
Application:	ELISA,IF,WB
Recommended	WB:1:500-1:5000; IF:1:20-1:200.

Properties

Stability & Storage:	Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	A synthetic peptide: Human Amyloid beta A4
Antigen Species:	Human
Gene ID:	351
Uniprot ID:	P05067
Synonyms:	APP695;Amyloid Precursor;Amyloid Precursor Protein 695
Biology Area:	Neuroscience

Research Background

Functions as a cell surface receptor and performs physiological functions on the surface of neurons relevant to neurite growth, neuronal adhesion and axonogenesis. Interaction between APP molecules on neighboring cells promotes synaptogenesis. Involved in cell mobility and transcription regulation through protein-protein

Can promote transcription activation through binding to APBB1-KAT5 and inhibits Notch signaling through interaction with Numb. Couples to apoptosis-inducing pathways such as those mediated by G(O) and JIP. Inhibits G(o) alpha ATPase activity. Acts as a kinesin I membrane receptor, mediating the axonal transport of beta-secretase and presenilin 1. By acting as a kinesin I membrane receptor, plays a role in axonal anterograde transport of cargo towards synapses in axons. Involved in copper homeostasis/oxidative stress through copper ion reduction. In vitro, copper-metallated APP induces neuronal death directly or is potentiated through Cu(2+)-mediated low-density lipoprotein oxidation. Can regulate neurite outgrowth through binding to components of the extracellular matrix such as heparin and collagen I and IV. The splice isoforms that contain the BPTI domain possess protease inhibitor activity. Induces a AGER-dependent pathway that involves activation of p38 MAPK, resulting in internalization of amyloid-beta peptide and leading to mitochondrial dysfunction in cultured cortical neurons. Provides Cu(2+) ions for GPC1 which are required for release of nitric oxide (NO) and subsequent degradation of the heparan sulfate chains on GPC1. Amyloid-beta peptides are lipophilic metal chelators with metal-reducing activity. Bind transient metals such as copper, zinc and iron. In vitro, can reduce Cu(2+) and Fe(3+) to Cu(+) and Fe(2+), respectively. Amyloid-beta protein 42 is a more effective reductant than amyloid-beta protein 40. Amyloid-beta peptides bind to lipoproteins and apolipoproteins E and J in the CSF and to HDL particles in plasma, inhibiting metal-catalyzed oxidation of lipoproteins. APP42-beta may activate mononuclear phagocytes in the brain and elicit inflammatory responses. Promotes both tau aggregation and TPK II-mediated phosphorylation. Interaction with overexpressed HADH2 leads to oxidative stress and neurotoxicity. Also binds GPC1 in lipid rafts. Appicans elicit adhesion of neural cells to the extracellular matrix and may regulate neurite outgrowth in the brain. The gamma-CTF peptides as well as the caspase-cleaved peptides, including C31, are potent enhancers of neuronal apoptosis. N-APP binds TNFRSF21 triggering caspase activation and degeneration of both neuronal cell bodies (via caspase-3) and axons (via caspase-6).

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