Anti-Presenilin 1/PS-1 antibody [APS 18] ab15458

Overview

Product name: Anti-Presenilin 1/PS-1 antibody [APS 18]
Description: Mouse monoclonal [APS 18] to Presenilin 1/PS-1
Host species: Mouse
Tested applications: Suitable for: ICC, IHC-P, IP, ELISA, ICC/IF, WB
Species reactivity: Reacts with: Mouse, Rat, Human, Non human primates
Predicted to work with: Cynomolgus monkey
Immunogen: Synthetic peptide corresponding to Human Presenilin 1/PS-1 aa 313-334.
Sequence: SKYNAESTERESQDTVAENDDG

Properties

Form: Liquid
Storage buffer: Preservative: 0.05% Sodium azide
Constituents: 99% PBS, 0.1% BSA
Purity: Protein G purified
Clonality: Monoclonal
Clone number: APS 18
Isotype: IgG1

Applications

Our Abpromise guarantee covers the use of ab15458 in the following tested applications.
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.
**Function**
Probable catalytic subunit of the gamma-secretase complex, an endoprotease complex that catalyzes the intramembrane cleavage of integral membrane proteins such as Notch receptors and APP (beta-amyloid precursor protein). Requires the other members of the gamma-secretase complex to have a protease activity. May play a role in intracellular signaling and gene expression or in linking chromatin to the nuclear membrane. Stimulates cell-cell adhesion through its association with the E-cadherin/catenin complex. Under conditions of apoptosis or calcium influx, cleaves E-cadherin promoting the disassembly of the E-cadherin/catenin complex and increasing the pool of cytoplasmic beta-catenin, thus negatively regulating Wnt signaling. May also play a role in hematopoiesis.

**Tissue specificity**
Expressed in a wide range of tissues including various regions of the brain, liver, spleen and lymph nodes.

**Involvement in disease**
Defects in PSEN1 are a cause of Alzheimer disease type 3 (AD3) [MIM:607822]. AD3 is a familial early-onset form of Alzheimer disease. Alzheimer disease is a neurodegenerative disorder characterized by progressive dementia, loss of cognitive abilities, and deposition of fibrillar amyloid proteins as intraneuronal neurofibrillary tangles, extracellular amyloid plaques and vascular amyloid deposits. The major constituent of these plaques is the neurotoxic amyloid-beta-APP 40-42 peptide (s), derived proteolytically from the transmembrane precursor protein APP by sequential secretase processing. The cytotoxic C-terminal fragments (CTFs) and the caspase-cleaved products such as C31 derived from APP, are also implicated in neuronal death. Defects in PSEN1 are a cause of frontotemporal dementia [MIM:600274]. Defects in PSEN1 are the cause of cardiomyopathy dilated type 1U (CMD1U) [MIM:613694]. It is a disorder characterized by ventricular dilation and impaired systolic function, resulting in congestive heart failure and arrhythmia. Patients are at risk of premature death. Defects in PSEN1 are the cause of acne inversa familial type 3 (ACNIF3) [MIM:613737]. A chronic relapsing inflammatory disease of the hair follicles characterized by recurrent draining sinuses, painful skin abscesses, and disfiguring scars. Manifestations typically appear after puberty.

**Sequence similarities**
Belongs to the peptidase A22A family.

**Domain**
The PAL motif is required for normal active site conformation.

**Post-translational modifications**
Heterogeneous proteolytic processing generates N-terminal (NTF) and C-terminal (CTF) fragments of approximately 35 and 20 kDa, respectively. During apoptosis, the C-terminal fragment (CTF) is further cleaved by caspase-3 to produce the fragment, PS1-CTF12.

### Application

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After endoproteolysis, the C-terminal fragment (CTF) is phosphorylated on serine residues by PKA and/or PKC. Phosphorylation on Ser-346 inhibits endoproteolysis.

**Cellular localization**

Endoplasmic reticulum membrane. Golgi apparatus membrane. Cell surface. Bound to NOTCH1 also at the cell surface. Colocalizes with CDH1/2 at sites of cell-cell contact. Colocalizes with CTNNB1 in the endoplasmic reticulum and the proximity of the plasma membrane. Also present in azurophil granules of neutrophils.

**Images**

IF showing PS1 using ab15458.

Immunofluorescent analysis of Presenilin 1 / PS-1 using Presenilin 1 / PS-1 Monoclonal antibody (APS 18) ab115458 shows staining in MCF-7 cells. Presenilin 1 / PS-1 staining (green) F-Actin staining with Phalloidin (red) and nuclei with DAPI (blue) is shown. Cells were grown on chamber slides and fixed with formaldehyde prior to staining. Cells were probed without (control) or with an antibody recognizing Presenilin 1 / PS-1 ab115458 at a dilution of 1:20-1:100 over night at 4 °C washed with PBS and incubated with a DyLight-488 conjugated secondary antibody. Images were taken at 60X magnification.

Immunofluorescent analysis of Presenilin 1 / PS-1 using Presenilin 1 / PS-1 Monoclonal antibody (APS 18) ab115458 shows staining in A2058 melanoma cells. Presenilin 1 / PS-1 staining (green) F-Actin staining with Phalloidin (red) and nuclei with DAPI (blue) is shown. Cells were grown on chamber slides and fixed with formaldehyde prior to staining. Cells were probed without (control) or with an antibody recognizing Presenilin 1 / PS-1 ab115458 at a dilution of 1:20-1:100 over night at 4 °C washed with PBS and incubated with a DyLight-488 conjugated secondary antibody. Images were taken at 60X magnification.
Immunofluorescent analysis of Presenilin 1 / PS-1 using Presenilin 1 / PS-1 Monoclonal antibody (APS 18) ab115458 shows staining in HeLa cells. Presenilin 1 / PS-1 staining (green) F-Actin staining with Phalloidin (red) and nuclei with DAPI (blue) is shown. Cells were grown on chamber slides and fixed with formaldehyde prior to staining. Cells were probed without (control) or with or an antibody recognizing Presenilin 1 / PS-1 ab115458 at a dilution of 1:20-1:100 over night at 4 °C washed with PBS and incubated with a DyLight-488 conjugated secondary antibody. Images were taken at 60X magnification.

Immunohistochemistry was performed on normal biopsies of deparaffinized Human liver tissue. To expose target proteins heat induced antigen retrieval was performed using 10mM sodium citrate (pH6.0) buffer microwaved for 8-15 minutes. Following antigen retrieval tissues were blocked in 3% BSA-PBS for 30 minutes at room temperature. Tissues were then probed at a dilution of 1:200 with a mouse monoclonal antibody recognizing Presenilin 1 / PS-1 ab15458 or without primary antibody (negative control) overnight at 4°C in a humidified chamber. Tissues were washed extensively with PBST and endogenous peroxidase activity was quenched with a peroxidase suppressor. Detection was performed using a biotin-conjugated secondary antibody and SA-HRP followed by colorimetric detection using DAB. Tissues were counterstained with hematoxylin and prepped for mounting.

Immunohistochemistry was performed on normal biopsies of deparaffinized Human tonsil tissue. To expose target proteins heat induced antigen retrieval was performed using 10mM sodium citrate (pH6.0) buffer microwaved for 8-15 minutes. Following antigen retrieval tissues were blocked in 3% BSA-PBS for 30 minutes at room temperature. Tissues were then probed at a dilution of 1:20 with a mouse monoclonal antibody recognizing Presenilin 1 / PS-1 ab15458 or without primary antibody (negative control) overnight at 4°C in a humidified chamber. Tissues were washed extensively with PBST and endogenous peroxidase activity was quenched with a peroxidase suppressor. Detection was performed using a biotin-conjugated secondary antibody and SA-HRP followed by colorimetric detection using DAB. Tissues were counterstained with hematoxylin and prepped for mounting.
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