abcam

Product datasheet

Anti-beta Amyloid 1-42 antibody [mOC64] - BSA and Azide free ab271968

Recombinant RabMAb

1 References 5 Images

Overview

Product name	Anti-beta Amyloid 1-42 antibody [mOC64] - BSA and Azide free
Description	Rabbit monoclonal [mOC64] to beta Amyloid 1-42 - BSA and Azide free
Host species	Rabbit
Specificity	This antibody reacts weakly with beta Amyloid 1-40.
Tested applications	**Suitable for:** Dot blot, IHC-P, IHC-FrFl
Species reactivity	**Reacts with:** Mouse, Human
Immunogen	The details of the immunogen for this antibody are not available.
Positive control	Dot Blot: Human beta Amyloid (Aß) 1-42. IHC-P: FFPE Human Brain Alzheimer. IHC-Free floating: Mouse brain
General notes	This antibody was developed as part of a collaboration between Abcam and Professor Charles Glabe, UC Irvine. **ab201060** recognizes a conformation-dependent, aggregation-selective epitope of beta amyloid. mOC64 immunoreactivity maps to residues 3-6 (EFRH) of Aß, and preferentially binds to SDS-resistant oligomers over monomer on Western blots (**Hatami *et al.* 2014**). The epitope is insensitive to thermal denaturation (**Hatami *et al.* 2014**). It does not recognize pyroglytaminylated Aß at position 3 (Aß3(pE)-42) (**Nussbaum *et al.* 2012**). For further information on the immunogen, please refer to **Hatami *et al.* 2014** and **Kayed *et al.* 2007**. ab271968 is the carrier-free version of **ab201060**. Our **carrier-free** antibodies are typically supplied in a PBS-only formulation, purified and free of BSA, sodium azide and glycerol. The carrier-free buffer and high concentration allow for increased conjugation efficiency. This conjugation-ready format is designed for use with fluorochromes, metal isotopes, oligonucleotides, and enzymes, which makes them ideal for antibody labelling, functional and cell-based assays, flow-based assays (e.g. mass cytometry) and Multiplex Imaging applications. Use our **conjugation kits** for antibody conjugates that are ready-to-use in as little as 20 minutes with <1 minute hands-on-time and 100% antibody recovery: available for fluorescent dyes, HRP, biotin and gold. This product is compatible with the Maxpar® Antibody Labeling Kit from Fluidigm, without the need for antibody preparation. Maxpar® is a trademark of Fluidigm Canada Inc.

This product is a recombinant monoclonal antibody, which offers several advantages including:

- High batch-to-batch consistency and reproducibility
- Improved sensitivity and specificity
- Long-term security of supply
- Animal-free production

For more information **see here**.

Our RabMAb® technology is a patented hybridoma-based technology for making rabbit monoclonal antibodies. For details on our patents, please refer to **RabMAb® patents**.

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C. Do Not Freeze.
Storage buffer	pH: 7.2 Constituent: PBS
Carrier free	Yes
Purity	Protein A purified
Clonality	Monoclonal
Clone number	mOC64
Isotype	IgG

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab271968 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
Dot blot		Use at an assay dependent concentration.
IHC-P		Use at an assay dependent concentration. Perform heat mediated antigen retrieval with citrate buffer pH 6 before commencing with IHC staining protocol.
IHC-FrFl		Use at an assay dependent concentration.

Target

Function Functions as a cell surface receptor and performs physiological functions on the surface of neurons relevant to neurite growth, neuronal adhesion and axonogenesis. Involved in cell mobility and transcription regulation through protein-protein interactions. 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 (By similarity). Acts as a kinesin I membrane receptor, mediating the axonal transport of beta-secretase and presenilin 1. 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. Beta-amyloid 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. Beta-amyloid 42 is a more effective reductant than beta-amyloid 40. Beta-amyloid peptides bind to lipoproteins and apolipoproteins E and J in the CSF and to HDL particles in plasma, inhibiting metal-catalyzed oxidation of lipoproteins. Beta-APP42 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. 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).
Tissue specificity	Expressed in all fetal tissues examined with highest levels in brain, kidney, heart and spleen. Weak expression in liver. In adult brain, highest expression found in the frontal lobe of the cortex and in the anterior perisylvian cortex-opercular gyri. Moderate expression in the cerebellar cortex, the posterior perisylvian cortex-opercular gyri and the temporal associated cortex. Weak expression found in the striate, extra-striate and motor cortices. Expressed in cerebrospinal fluid, and plasma. Isoform APP695 is the predominant form in neuronal tissue, isoform APP751 and isoform APP770 are widely expressed in non-neuronal cells. Isoform APP751 is the most abundant form in T-lymphocytes. Appican is expressed in astrocytes.
Involvement in disease	Defects in APP are the cause of Alzheimer disease type 1 (AD1) [MIM:104300]. AD1 is a familial early-onset form of Alzheimer disease. It can be associated with cerebral amyloid angiopathy. 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 APP are the cause of cerebral amyloid angiopathy APP-related (CAA-APP) [MIM:605714]. A hereditary localized amyloidosis due to amyloid-beta A4 peptide(s) deposition in the cerebral vessels. The principal clinical characteristics are recurrent cerebral and cerebellar hemorrhages, recurrent strokes, cerebral ischemia, cerebral infarction, and progressive mental deterioration. Patients develop cerebral hemorrhage because of the severe cerebral amyloid angiopathy. Parenchymal amyloid deposits are rare and largely in the form of pre-amyloid lesions or diffuse plaque-like structures. They are Congo red negative and lack the dense amyloid cores commonly present in Alzheimer disease. Some affected individuals manifest progressive aphasic dementia, leukoencephalopathy, and occipital calcifications.
Sequence similarities	Belongs to the APP family. Contains 1 BPTI/Kunitz inhibitor domain.
Domain	The basolateral sorting signal (BaSS) is required for sorting of membrane proteins to the

basolateral surface of epithelial cells.
The NPXY sequence motif found in many tyrosine-phosphorylated proteins is required for the specific binding of the PID domain. However, additional amino acids either N- or C-terminal to the NPXY motif are often required for complete interaction. The PID domain-containing proteins which bind APP require the YENPTY motif for full interaction. These interactions are independent of phosphorylation on the terminal tyrosine residue. The NPXY site is also involved in clathrin-mediated endocytosis.

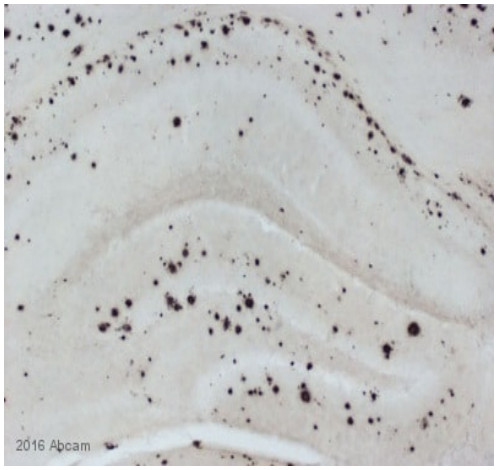
Post-translational modifications

Proteolytically processed under normal cellular conditions. Cleavage either by alpha-secretase, beta-secretase or theta-secretase leads to generation and extracellular release of soluble APP peptides, S-APP-alpha and S-APP-beta, and the retention of corresponding membrane-anchored C-terminal fragments, C80, C83 and C99. Subsequent processing of C80 and C83 by gamma-secretase yields P3 peptides. This is the major secretory pathway and is non-amyloidogenic. Alternatively, presenilin/nicastrin-mediated gamma-secretase processing of C99 releases the amyloid beta proteins, amyloid-beta 40 (Abeta40) and amyloid-beta 42 (Abeta42), major components of amyloid plaques, and the cytotoxic C-terminal fragments, gamma-CTF(50), gamma-CTF(57) and gamma-CTF(59).
Proteolytically cleaved by caspases during neuronal apoptosis. Cleavage at Asp-739 by either caspase-6, -8 or -9 results in the production of the neurotoxic C31 peptide and the increased production of beta-amyloid peptides.
N- and O-glycosylated. O-linkage of chondroitin sulfate to the L-APP isoforms produces the APP proteoglycan core proteins, the appicans. The chondroitin sulfate chain of appicans contains 4-O-sulfated galactose in the linkage region and chondroitin sulfate E in the repeated disaccharide region.
Phosphorylation in the C-terminal on tyrosine, threonine and serine residues is neuron-specific. Phosphorylation can affect APP processing, neuronal differentiation and interaction with other proteins. Phosphorylated on Thr-743 in neuronal cells by Cdc5 kinase and Mapk10, in dividing cells by Cdc2 kinase in a cell-cycle dependent manner with maximal levels at the G2/M phase and, in vitro, by GSK-3-beta. The Thr-743 phosphorylated form causes a conformational change which reduces binding of Fe65 family members. Phosphorylation on Tyr-757 is required for SHC binding. Phosphorylated in the extracellular domain by casein kinases on both soluble and membrane-bound APP. This phosphorylation is inhibited by heparin.
Extracellular binding and reduction of copper, results in a corresponding oxidation of Cys-144 and Cys-158, and the formation of a disulfide bond. In vitro, the APP-Cu(+) complex in the presence of hydrogen peroxide results in an increased production of beta-amyloid-containing peptides.
Trophic-factor deprivation triggers the cleavage of surface APP by beta-secretase to release sAPP-beta which is further cleaved to release an N-terminal fragment of APP (N-APP).
Beta-amyloid peptides are degraded by IDE.

Cellular localization

Membrane. Membrane > clathrin-coated pit. Cell surface protein that rapidly becomes internalized via clathrin-coated pits. During maturation, the immature APP (N-glycosylated in the endoplasmic reticulum) moves to the Golgi complex where complete maturation occurs (O-glycosylated and sulfated). After alpha-secretase cleavage, soluble APP is released into the extracellular space and the C-terminal is internalized to endosomes and lysosomes. Some APP accumulates in secretory transport vesicles leaving the late Golgi compartment and returns to the cell surface. Gamma-CTF(59) peptide is located to both the cytoplasm and nuclei of neurons. It can be translocated to the nucleus through association with APBB1 (Fe65). Beta-APP42 associates with FRPL1 at the cell surface and the complex is then rapidly internalized. APP sorts to the basolateral surface in epithelial cells. During neuronal differentiation, the Thr-743 phosphorylated form is located mainly in growth cones, moderately in neurites and sparingly in the cell body. Casein kinase phosphorylation can occur either at the cell surface or within a post-Golgi compartment.

Images

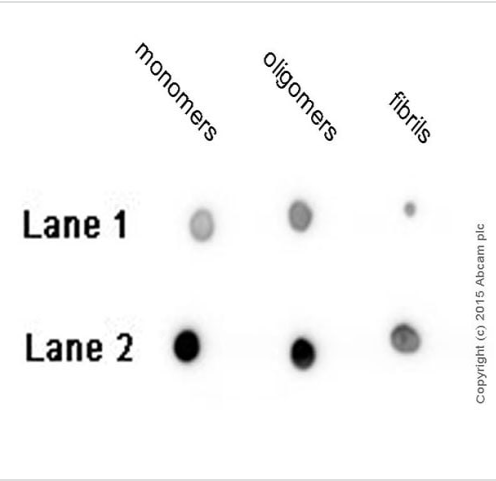


Immunohistochemistry - Free Floating - Anti-beta Amyloid 1-42 antibody [mOC64] - BSA and Azide free (ab271968)

Image courtesy of an anonymous AbReview

Transgenic (APP, PS1) mouse brain stained for beta-Amyloid 1-42 with conformation-specific **ab201060** (1/1000) in immunohistochemical analysis.

This data was developed using the same antibody clone in a different buffer formulation containing PBS, BSA, glycerol, and sodium azide (**ab201060**).



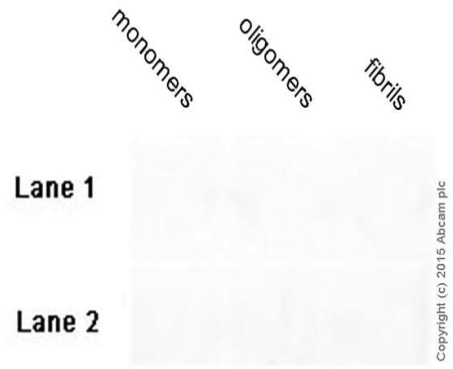
Dot Blot - Anti-beta Amyloid 1-42 antibody [mOC64] - BSA and Azide free (ab271968)

Dot blot analysis of human beta Amyloid 1-42 labeled with **ab201060** at 1/7000 dilution.
Lane 1: beta Amyloid (Aβ) 1-40.
Lane 2: beta Amyloid (Aβ) 1-42.
Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated (**ab97051**) at 1/5000 dilution was used as secondary antibody.
Blocking and diluting buffer: 5% NFDM/TBST.
Exposure time: 30 seconds.

Antibody reactivity was assessed using a dot blot, which is a non-quantitative method that maintains the native conformation of beta Amyloid. Beta Amyloid 1-40 and 1-42 peptides underwent the following aggregation conditions before being spotted onto a nitrocellulose membrane and detected using **ab201060**:
Monomers: 0.3 mg of beta Amyloid peptide was dissolved in 30 µl 100 mM NaOH and incubated at room temperature for 10 minutes. It was then diluted with 970 µl of 1% SDS and boiled for five minutes.
Oligomers: 0.3 mg of beta Amyloid peptide was dissolved in 30 µl 100 mM NaOH and incubated at room temperature for 10 minutes. It was then diluted with 970 µl of 10 mM phosphate buffer pH 7.4 containing 0.02% sodium azide and incubated at room temperature for four days.
Fibrils: 0.3 mg of beta Amyloid peptide was dissolved in 1 ml 50% hexafluoroisopropanol (HFIP) with 0.02% sodium azide. It was then

stirred constantly for nine days; the first seven with a cap on and the final two with the cap removed to allow evaporation of the HFIP. Fibrils were then sedimented at 20,000 rpm in a microcentrifuge for 20 minutes and resuspended in 1 ml of PBS + 0.02% sodium azide.

This data was developed using the same antibody clone in a different buffer formulation containing PBS, BSA, glycerol, and sodium azide (**ab201060**).

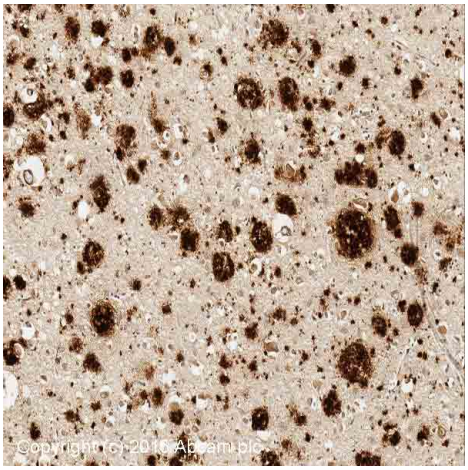


Dot Blot - Anti-beta Amyloid 1-42 antibody [mOC64] - BSA and Azide free (ab271968)

Negative control (secondary ab only):

Lane 1: Human beta Amyloid (Aβ) 1-40.
Lane 2: Human beta Amyloid (Aβ) 1-42.
Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated (**ab97051**) at 1/5000 dilution was used as secondary antibody.
Blocking and diluting buffer: 5% NFDM/TBST.
Exposure time: 30 seconds.

This data was developed using the same antibody clone in a different buffer formulation containing PBS, BSA, glycerol, and sodium azide (**ab201060**).



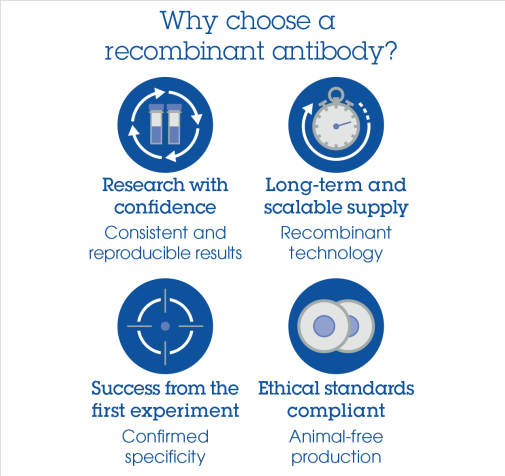
Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-beta Amyloid 1-42 antibody [mOC64] - BSA and Azide free (ab271968)

IHC image of beta Amyloid 1-42 staining in Human Brain Alzheimer formalin fixed paraffin embedded tissue section*, performed on a Leica Bond™ system using the standard protocol F. The section was pre-treated using heat mediated antigen retrieval with sodium citrate buffer (pH6, epitope retrieval solution 1) for 20 mins. The section was then incubated with **ab201060**, 1µg/ml, for 15 mins at room temperature and detected using an HRP conjugated compact polymer system. DAB was used as the chromogen. The section was then counterstained with haematoxylin and mounted with DPX.

For other IHC staining systems (automated and non-automated) customers should optimize variable parameters such as antigen retrieval conditions, primary antibody concentration and antibody incubation times.

*Tissue obtained from the Human Research Tissue Bank, supported by the NIHR Cambridge Biomedical Research Centre

This data was developed using the same antibody clone in a different buffer formulation containing PBS, BSA, glycerol, and sodium azide (**ab201060**).



Anti-beta Amyloid 1-42 antibody [mOC64] - BSA and Azide free (ab271968)

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