

# Anti-beta Amyloid 1-42 antibody [mOC98] - Conformation-Specific ab201061

重组 RabMAb

★★★★★ **6 Abreviews** **8 References** **4 图像**

### 概述

产品名称	Anti-beta Amyloid 1-42抗体[mOC98] - Conformation-Specific
描述	兔单克隆抗体[mOC98] to beta Amyloid 1-42 - Conformation-Specific
宿主	Rabbit
经测试应用	适用于: IHC-P, Dot blot
种属反应性	与反应: Mouse, Human
免疫原	The details of the immunogen for this antibody are not available.
阳性对照	beta Amyloid (A $\beta$ ) 1-42 IHC-P: FFPE human Alzheimer hippocampus tissue sections.
常规说明	<p>This antibody was developed as part of a collaboration between Abcam and Professor Charles Glabe, UC Irvine.</p> <p>ab201061 recognizes a conformation-specific, discontinuous epitope of beta Amyloid that maps to segments AEFRHD and EDVGSNK (<a href="#">Hatami et al. 2014</a>).</p> <p>For further information on the immunogen, please refer to <a href="#">Hatami et al. 2014</a> and <a href="#">Kayed et al. 2007</a>.</p> <p>This product is a recombinant monoclonal antibody, which offers several advantages including:</p> <ul style="list-style-type: none"><li>- High batch-to-batch consistency and reproducibility</li><li>- Improved sensitivity and specificity</li><li>- Long-term security of supply</li><li>- Animal-free production</li></ul> <p>For more information <a href="#">see here</a>.</p> <p>Our RabMAb<sup>®</sup> technology is a patented hybridoma-based technology for making rabbit monoclonal antibodies. For details on our patents, please refer to <a href="#">RabMAb<sup>®</sup> patents</a>.</p>

### 性能

形式	Liquid
存放说明	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C long term. Avoid freeze / thaw cycle.
存储溶液	pH: 7.2

Preservative: 0.01% Sodium azide  
Constituents: 59% PBS, 0.05% BSA, 40% Glycerol

纯度 Protein A purified  
克隆 单克隆  
克隆编号 mOC98  
同种型 IgG

## 应用

**The Abpromise guarantee** Abpromise™ 承诺保证使用 ab201061 于以下的经测试应用

“应用说明”部分 下显示的仅为推荐的起始稀释度;实际最佳的稀释度/浓度应由使用者检定。

应用	Ab评论	说明
IHC-P		Use a concentration of 1 µg/ml. Perform heat mediated antigen retrieval with citrate buffer pH 6 before commencing with IHC staining protocol.
Dot blot		1/10000.

## 靶标

**功能**

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 BPT1 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).

#### 组织特异性

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.

#### 疾病相关

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.

#### 序列相似性

Belongs to the APP family.

Contains 1 BPTI/Kunitz inhibitor domain.

#### 结构域

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.

#### 翻译后修饰

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.

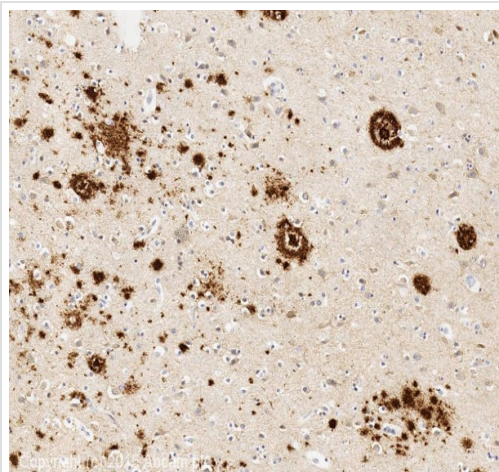
## 细胞定位

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.

## 图片

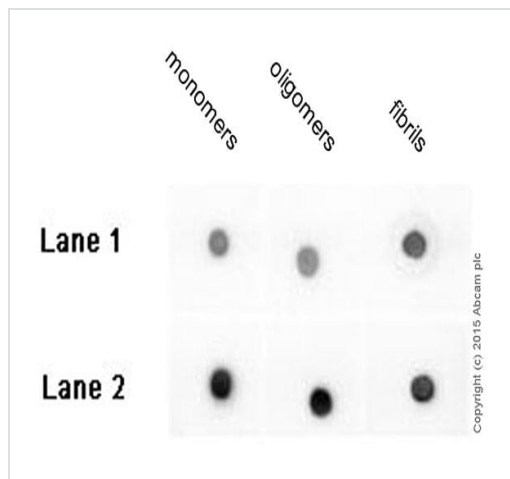


Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-beta Amyloid 1-42 antibody [mOC98] - Conformation-Specific (ab201061)

IHC image of beta Amyloid 1-42 staining in human Alzheimer hippocampus 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 **ab201062**, 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



Dot Blot - Anti-beta Amyloid 1-42 antibody [mOC98] - Conformation-Specific (ab201061)

Dot blot analysis of beta Amyloid 1-42 labeled with ab201061 at 1/10000 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.

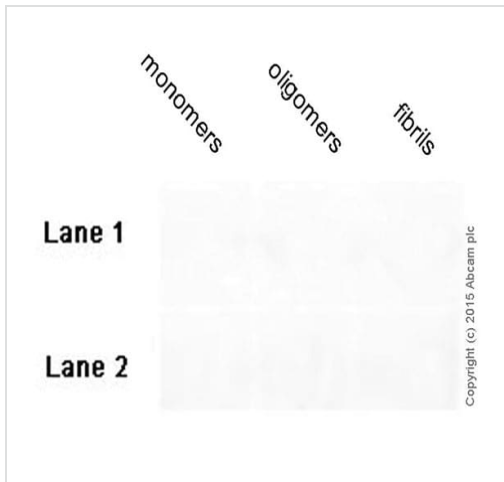
Antibody reactivity was assessed using a dot blot, which is a non-quantitative method that maintains the native conformation of beta Amyloid. Beta Amyloid 1-40 and 1-42 peptides underwent the following aggregation conditions before being spotted onto a nitrocellulose membrane and detected using ab201061:

**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.



Dot Blot - Anti-beta Amyloid 1-42 antibody [mOC98]  
- Conformation-Specific (ab201061)

Negative control (secondary ab only):

Lane 1: beta Amyloid (A $\beta$ ) 1-40.





Lane 2: beta Amyloid (A $\beta$ ) 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.

Why choose a recombinant antibody?

 <p><b>Research with confidence</b> Consistent and reproducible results</p>	 <p><b>Long-term and scalable supply</b> Recombinant technology</p>
 <p><b>Success from the first experiment</b> Confirmed specificity</p>	 <p><b>Ethical standards compliant</b> Animal-free production</p>

Anti-beta Amyloid 1-42 antibody [mOC98] -  
Conformation-Specific (ab201061)

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