

**Anti-Beta Amyloid 1-9 (SHEEP) Antibody**  
**Beta Amyloid 9 Antibody**  
**Catalog # ASR5902****Specification**

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**Anti-Beta Amyloid 1-9 (SHEEP) Antibody - Product Information**

Host	Sheep
Conjugate	Unconjugated
Target Species	Human
Reactivity	Rat, Human, Mouse
Clonality	Polyclonal
Application	WB, E, I, LCI
Application Note	Anti-Beta Amyloid 1-9 antibody is useful for ELISA and Western Blot. Specific conditions for reactivity should be optimized by the end user. Expect a band approximately ~86kDa corresponding to the appropriate cell lysate or extract.
Physical State	Liquid (sterile filtered)
Buffer	0.02 M Potassium Phosphate, 0.15 M Sodium Chloride, pH 7.2
Immunogen	Anti-Beta Amyloid 9 affinity purified antibody was prepared from whole sheep serum produced by repeated immunizations with a synthetic peptide corresponding to the C-terminus region near AB9.
Stabilizer	50% (v/v) Glycerol
Preservative	0.01% (w/v) Sodium Azide

**Anti-Beta Amyloid 1-9 (SHEEP) Antibody - Additional Information****Gene ID** 351**Purity**

Anti-Beta Amyloid 1-9 was affinity purified from monospecific antiserum by immunoaffinity chromatography. A BLAST analysis was used to suggest reactivity with human, sheep, rabbit, canine, bovine, pig, guinea pig, and monkey based on 100% sequence homology. Cross-reactivity with Beta Amyloid from other sources has not been determined.

**Storage Condition**

Store vial at -20° C prior to opening. Aliquot contents and freeze at -20° C or below for extended storage. Avoid cycles of freezing and thawing. Centrifuge product if not completely clear after standing at room temperature. This product is stable for several weeks at 4° C as an undiluted liquid. Dilute only prior to immediate use.

**Precautions Note**

This product is for research use only and is not intended for therapeutic or diagnostic applications.

## Anti-Beta Amyloid 1-9 (SHEEP) Antibody - Protein Information

**Name** APP ([HGNC:620](#))

### Function

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 (PubMed:<a href="http://www.uniprot.org/citations/25122912" target="\_blank">25122912</a>). 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 (By similarity). By acting as a kinesin I membrane receptor, plays a role in axonal anterograde transport of cargo towards synapses in axons (PubMed:<a href="http://www.uniprot.org/citations/17062754" target="\_blank">17062754</a>, PubMed:<a href="http://www.uniprot.org/citations/23011729" target="\_blank">23011729</a>). 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 protein 42]: More effective reductant than amyloid-beta protein 40. May activate mononuclear phagocytes in the brain and elicit inflammatory responses. The gamma-CTF peptides as well as the caspase-cleaved peptides, including C31, are potent enhancers of neuronal apoptosis.

### Cellular Location

Cell membrane; Single-pass type I membrane protein. Membrane; Single-pass type I membrane protein. Perikaryon Cell projection, growth cone. Membrane, clathrin-coated pit. Early endosome. Cytoplasmic vesicle. Note=Cell surface protein that rapidly becomes internalized via clathrin-coated pits. Only a minor proportion is present at the cell membrane; most of the protein is present in intracellular vesicles (PubMed:20580937) 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. 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 (PubMed:10341243). Casein kinase phosphorylation can occur either at the cell surface or within a post-Golgi compartment. Associates with GPC1 in perinuclear compartments. Colocalizes with SORL1 in a vesicular pattern in cytoplasm and perinuclear regions. [C99]: Early endosome [Amyloid-beta protein 40]: Cell surface [Gamma-secretase C-terminal fragment 59]: Nucleus. Cytoplasm Note=Located to both the cytoplasm and nuclei of neurons. It can be translocated to the nucleus through association with APBB1 (Fe65) (PubMed:11544248). In dopaminergic neurons, the phosphorylated Thr-743 form is localized to the nucleus (By similarity) {ECO:0000250|UniProtKB:P12023, ECO:0000269|PubMed:11544248}

### Tissue Location

Expressed in the brain and in cerebrospinal fluid (at protein level) (PubMed:2649245). 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.

### **Anti-Beta Amyloid 1-9 (SHEEP) Antibody - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### **Anti-Beta Amyloid 1-9 (SHEEP) Antibody - Images**

### **Anti-Beta Amyloid 1-9 (SHEEP) Antibody - Background**

Beta Amyloid functions as a cell surface receptor and performs physiological functions on the surface of neurons relevant to neurite growth, neuronal adhesion and axonogenesis. It is expressed in all fetal tissues with the highest levels located in the brain, kidney, heart and spleen tissue. It is involved in cell mobility, transcription regulation via protein-protein interactions and copper homeostasis/oxidative stress through copper ion reduction. The copper-metallated APP induces neuronal death directly in vitro, or is potentiated through Cu<sup>2+</sup> mediated low-density lipoprotein oxidation. It has binding capabilities via its C-terminus for transient metals such as copper, zinc and iron. It binds APBB1-KAT5 to promote transcription activation and inhibits Notch signaling through interaction with Numb. It also promotes tau aggregation and TPK II-mediated phosphorylation. Anti-Beta Amyloid regulates neurite outgrowth by binding components in the cellular matrix such as heparin, collagen I and amyloid-beta peptide, leading to mitochondrial dysfunction in cultured cortical neurons. Defects in APP cause Alzheimer disease type 1 and cerebral amyloid angiopathy. This antibody is ideal for researchers interested in Cancer or Neuroscience research.