

## Amyloid Oligomers (A11) Antibody

Catalog # ASM10487

### Specification

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#### Amyloid Oligomers (A11) Antibody - Product Information

Application	IHC, WB, DB
Primary Accession	<a href="#">P05067</a>
Other Accession	<a href="#">NM_000484.2</a>
Host	Rabbit
Reactivity	Human, Eukaryote, Mouse, Rat
Clonality	Polyclonal

#### Description

Rabbit Anti-Human Amyloid Oligomers (A11) Polyclonal

#### Target/Specificity

Recognizes all types of amyloid oligomers. Appears to recognize a peptide backbone epitope that is common to amyloid oligomers, but is not found in native proteins, amyloidogenic monomer or mature amyloid fibrils.

#### Other Names

Amyloid Oligomer alpha beta Antibody, A11 Antibody, Amyloid Oligomer AlphaBeta Antibody, APP Antibody

#### Immunogen

Synthetic molecular mimic of soluble oligomers

#### Purification

Protein A Purified

Storage -20°C

#### Storage Buffer

PBS, 50% glycerol, 0.09% sodium azide

Shipping Temperature Blue Ice or 4°C

#### Certificate of Analysis

A 1:1000 dilution of SPC-506 was sufficient for detection of amyloid oligomers in 10 µg of mouse brain lysates by colorimetric immunoblot analysis using Goat anti-rabbit IgG:HRP as the secondary antibody.

#### Cellular Localization

Membrane

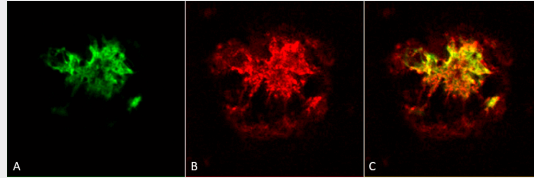
#### Amyloid Oligomers (A11) Antibody - Protocols

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

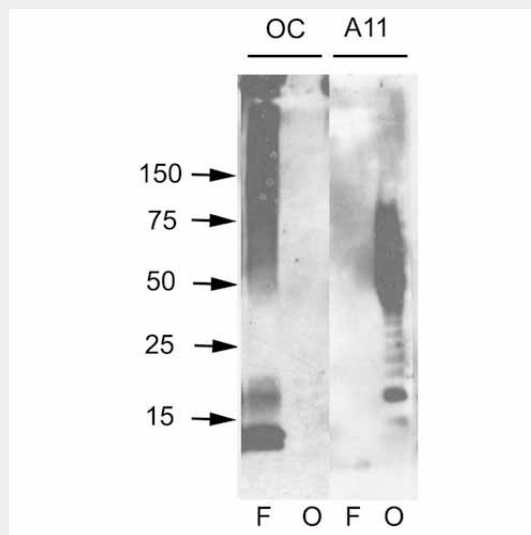
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)

- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

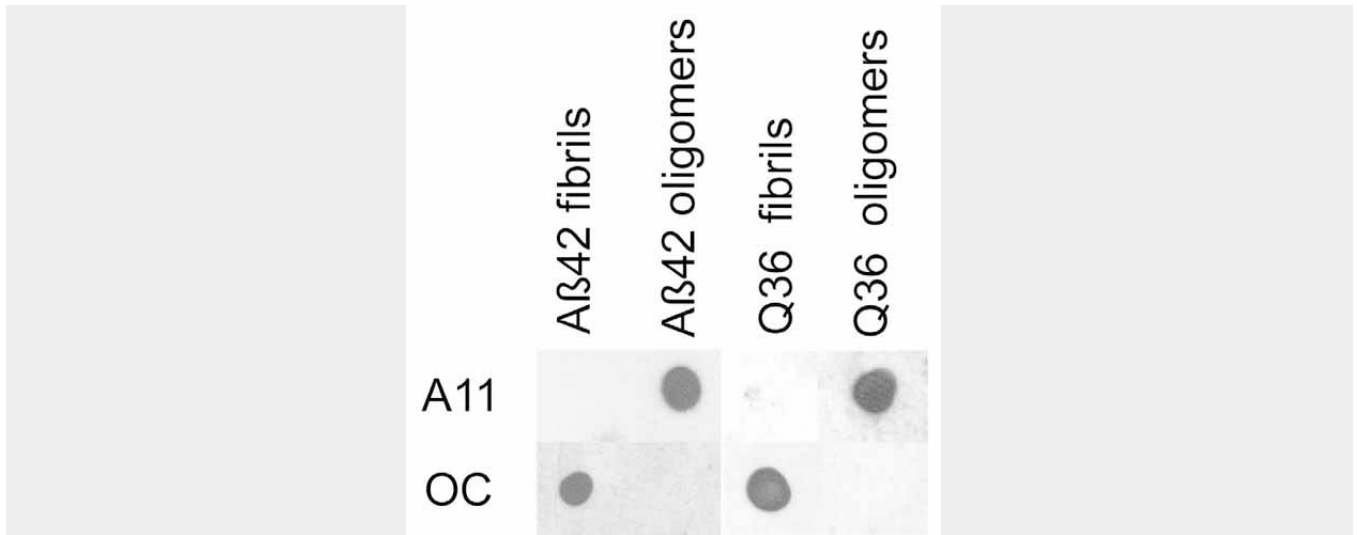
### Amyloid Oligomers (A11) Antibody - Images



Immunohistochemistry analysis using Rabbit Anti-Amyloid Oligomers (A11) Polyclonal Antibody (ASM10487). Tissue: Alzheimer's Disease brain. Species: Human. Fixation: Formalin fixed. Primary Antibody: Rabbit Anti-Amyloid Oligomers (A11) Polyclonal Antibody (ASM10487) at 1:1000. Secondary Antibody: Goat Anti-Rabbit ATTO 594 (red). Localization: Plaque. (A) Amyloid Fibril (OC) Antibody (SPC-507). (B) Amyloid Oligomer (A11) Antibody (ASM10487). (C) Composite. Courtesy of: Dr. Elizabeth Head, University of California, Irvine.



Western blot analysis of Human Abeta42 fibrils and prefibrillar oligomers showing detection of Amyloid Oligomers (A11) protein using Rabbit Anti-Amyloid Oligomers (A11) Polyclonal Antibody (ASM10487). Primary Antibody: Rabbit Anti-Amyloid Oligomers (A11) Polyclonal Antibody (ASM10487) at 1:1000. Courtesy of: Kaye, R., Head, E., Thompson, J. L., McIntire, T. M., Milton, S. C., Cotman, C. W., et al. (2003). Common structure of soluble amyloid oligomers implies common mechanism of pathogenesis. *Science* 300, 486-489. doi: 10.1126/science.1079469.



Dot blot analysis using Rabbit Anti-Amyloid Oligomers (A11) Polyclonal Antibody (ASM10487). Tissue: Aβ42 fibrils and prefibrillar oligomers. Species: Human. Primary Antibody: Rabbit Anti-Amyloid Oligomers (A11) Polyclonal Antibody (ASM10487) at 1:1000. Courtesy of: Kaye, R., Head, E., Thompson, J. L., McIntire, T. M., Milton, S. C., Cotman, C. W., et al. (2003). Common structure of soluble amyloid oligomers implies common mechanism of pathogenesis. *Science* 300, 486-489. doi: 10.1126/science.1079469.

### Amyloid Oligomers (A11) Antibody - Background

Amyloid monomeric proteins can sometimes oligomerize into destructive amyloid fibrils. Amyloidogenic conformations of non-disease related proteins can be created by partial protein misfolding or denaturation. Many degenerative diseases are known to be related to the accumulation of misfolded proteins as amyloid fibres (1, 2). These include the amyloid-β peptide plaques and tau neurofibrillary tangles in senile plaques of Alzheimer's symptomology, the deposition of α-synuclein in the Lewy bodies of Parkinson's disease, and accumulation of polyglutamine-containing aggregates in Huntington's disease (2, 3).

### Amyloid Oligomers (A11) Antibody - References

1. Glabe C.G. (2004) *Trends Biochem Sci.* 29(10): 542-547.
2. Kaye R., et al. (2004) *J Bio. Chem.* 279: 46363-46366.
3. Kaye R., et al. (2003) *Science.* 300(5618): 486-489.