

## DESCRIPTION

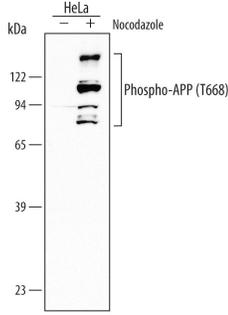
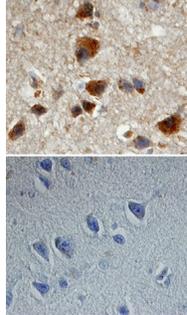
<b>Species Reactivity</b>	Human/Mouse
<b>Specificity</b>	Detects multiple isoforms of human and mouse APP phosphorylated at sites corresponding to T668 of the human APP695 isoform.
<b>Source</b>	Monoclonal Mouse IgG <sub>1</sub> Clone # 304904
<b>Purification</b>	Protein A or G purified from hybridoma culture supernatant
<b>Immunogen</b>	Phosphopeptide containing human APP T668 site
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

## APPLICATIONS

**Please Note:** Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
<b>Western Blot</b>	1 µg/mL	See Below
<b>Immunohistochemistry</b>	8-25 µg/mL	See Below

## DATA

<p><b>Western Blot</b></p>  <p><b>Detection of Human Phospho-APP/Protease Nexin II (T668) by Western Blot.</b> Western blot shows lysates of HeLa human cervical epithelial carcinoma cell line untreated (-) or treated (+) with 1 µg/mL nocodazole for 16 hours. PVDF membrane was probed with 1 µg/mL of Human/Mouse Phospho-APP/Protease Nexin II (T668) Monoclonal Antibody (Catalog # MAB2508), followed by HRP-conjugated Anti-Mouse IgG Secondary Antibody (Catalog # HAF007). This experiment was conducted under reducing conditions and using Immunoblot Buffer Group 1.</p>	<p><b>Immunohistochemistry</b></p>  <p><b>APP/Protease Nexin II in Human Alzheimer's Disease Brain.</b> APP/Protease Nexin II was detected in immersion fixed paraffin-embedded sections of human Alzheimer's disease brain (cortex) using 8 µg/mL Human/Mouse Phospho-APP/Protease Nexin II (T668) Monoclonal Antibody (Catalog # MAB2508) overnight at 4 °C. Tissue was stained with the Anti-Mouse HRP-DAB Cell &amp; Tissue Staining Kit (brown; Catalog # CTS002) and counterstained with hematoxylin (blue). View our protocol for <a href="#">Chromogenic IHC Staining of Paraffin-embedded Tissue Sections</a>.</p>
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## PREPARATION AND STORAGE

<b>Reconstitution</b>	Reconstitute at 0.5 mg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

## BACKGROUND

Amyloid precursor protein (APP) is a type I membrane protein with several human isoforms due to alternative splicing. APP-770, -751, and -733 contain a Kunitz protease inhibitor (KPI) domain (residue 291 - 342) and APP-695 does not. APP is a cell surface molecule with many functions. It can be processed proteolytically in two different pathways. In one pathway, β- and γ-secretase cleave at the β site between residue 670 and 671 and the γ site between residue 711 and 714 to produce β-amyloid peptide (Aβ<sub>40</sub> and Aβ<sub>42</sub>), a major component in plaques found in brains of patients with Alzheimer's disease (1). The other pathway involves α-secretase that cleaves residues between 687 and 688. It is antiamyloidogenic due to its benign character and the prevention of the Aβ peptide formation (2). Soluble APP containing the KPI domain, also referred to as protease nexin II, is a potent inhibitor of serine proteases and may have additional functions. For example, it may regulate the contact face of blood coagulation and limit thrombosis specially in brain due to its localization and coagulation factor XI inhibiting activity (3, 4).

## References:

1. Haass, C. (2004) EMBO J. **23**:483.
2. Lichtenthaler, S. F. and C. Haass (2004) J. Clin. Invest. **113**:1384.
3. Badellino, K.O. and P.N. Walsh (2000) Biochemistry **39**:4769.
4. Xu, F. *et al.* (2005) Proc. Natl. Acad. Sci. USA. **102**:18135.