Product datasheet

Anti-Alpha-synuclein (phospho S129) antibody ab59264

Overview

**Product name**  
Anti-Alpha-synuclein (phospho S129) antibody

**Description**  
Rabbit polyclonal to Alpha-synuclein (phospho S129)

**Host species**  
Rabbit

**Specificity**  
Detects endogenous levels of Synuclein only when phosphorylated at serine 129. Due to 69% sequence homology ab59264 might react with Beta synuclein

**Tested applications**  
Suitable for: ICC/IF, IHC-P, ELISA, IHC-FoFr, WB, IHC-Fr

**Species reactivity**  
Reacts with: Mouse, Human

**Immunogen**  
Synthetic peptide corresponding to Human Alpha-synuclein. Synthetic phosphopeptide derived from Human Synuclein around the phosphorylation site of serine 129 (M-P-SP-E-E).

**Positive control**  
Human brain.

Properties

**Form**  
Liquid

**Storage instructions**  
Shipped at 4°C. Store at -20°C. Stable for 12 months at -20°C.

**Storage buffer**  
pH: 7.40  
Preservative: 0.02% Sodium azide  
 Constituents: PBS, 50% Glycerol, 0.87% Sodium chloride  
 Without Mg+2 and Ca+2

**Purity**  
Immunogen affinity purified

**Clonality**  
Polyclonal

**Isotype**  
IgG

Applications

Our Abpromise guarantee covers the use of ab59264 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.
**Function**
May be involved in the regulation of dopamine release and transport. Induces fibrillization of microtubule-associated protein tau. Reduces neuronal responsiveness to various apoptotic stimuli, leading to a decreased caspase-3 activation.

**Tissue specificity**
Expressed principally in brain but is also expressed in low concentrations in all tissues examined except in liver. Concentrated in presynaptic nerve terminals.

**Involvement in disease**
Genetic alterations of SNCA resulting in aberrant polymerization into fibrils, are associated with several neurodegenerative diseases (synucleinopathies). SNCA fibrillar aggregates represent the major non A-beta component of Alzheimer disease amyloid plaque, and a major component of Lewy body inclusions. They are also found within Lewy body (LB)-like intraneuronal inclusions, glial inclusions and axonal spheroids in neurodegeneration with brain iron accumulation type 1. Parkinson disease 1 Parkinson disease 4 Dementia Lewy body

**Sequence similarities**
Belongs to the synuclein family.

**Domain**
The 'non A-beta component of Alzheimer disease amyloid plaque' domain (NAC domain) is involved in fibrils formation. The middle hydrophobic region forms the core of the filaments. The C-terminus may regulate aggregation and determine the diameter of the filaments.

**Post-translational modifications**
Phosphorylated, predominantly on serine residues. Phosphorylation by CK1 appears to occur on residues distinct from the residue phosphorylated by other kinases. Phosphorylation of Ser-129 is selective and extensive in synucleinopathy lesions. In vitro, phosphorylation at Ser-129 promoted insoluble fibril formation. Phosphorylated on Tyr-125 by a PTK2B-dependent pathway upon osmotic stress. Hallmark lesions of neurodegenerative synucleinopathies contain alpha-synuclein that is modified by nitration of tyrosine residues and possibly by dityrosine cross-linking to generated stable oligomers. Ubiquitinated. The predominant conjugate is the diubiquitinated form. Acetylation at Met-1 seems to be important for proper folding and native oligomeric structure.

**Cellular localization**

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### Target

<table>
<thead>
<tr>
<th>Application</th>
<th>Abreviews</th>
<th>Notes</th>
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<tbody>
<tr>
<td>ICC/IF</td>
<td>🌟🌟🌟🌟🌟</td>
<td>Use at an assay dependent concentration. PubMed: 22513881</td>
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<tr>
<td>IHC-P</td>
<td>1/50 - 1/100.</td>
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<tr>
<td>ELISA</td>
<td>1/5000.</td>
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<td>IHC-FoFr</td>
<td>Use at an assay dependent concentration. PubMed: 20098733</td>
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<tr>
<td>WB</td>
<td>🌟🌟🌟🌟🌟</td>
<td>1/500 - 1/1000.</td>
</tr>
<tr>
<td>IHC-Fr</td>
<td>Use at an assay dependent concentration. PubMed: 23664753</td>
<td></td>
</tr>
</tbody>
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### Images

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Locally administered rotenone induces alpha-synuclein phosphorylation, accumulation and aggregation with gliosis in ENS ganglia

Mouse ganglia tissue stained for Alpha-synuclein (phospho S129) using ab59264 at 1/50 dilution in immunohistochemical analysis.

Section from an untreated mouse is shown in Panel I. Section from a rotenone-treated mouse is shown in Panel J.

(After Figure 1 of Pan-Motijo et al)

All lanes: Anti-Alpha-synuclein (phospho S129) antibody (ab59264) at 1/500 dilution

Lane 1: Mouse brain whole cell lysates

Lane 2: Mouse brain whole cell lysates with immunogen phosphopeptide

Immunohistochemical analysis of paraffin-embedded human brain tissue using ab59264 at a dilution of 1/50-1/100. Left hand image - without immunising peptide; right hand image - with immunising peptide.
All lanes: Anti-Alpha-synuclein (phospho S129) antibody (ab59264) at 1/1000 dilution

Lanes 1-3: Whole tissue lysate prepared from young transgenic mouse overexpressing human alpha-synuclein
Lanes 4-6: Whole tissue lysate prepared from old transgenic mouse overexpressing human alpha-synuclein
Lane 7: Whole tissue lysate prepared from KO mouse

Lysates/proteins at 50 µg per lane.

Secondary All lanes: Goat anti-rabbit Ig (H+L) HRP at 1/1000 dilution

Developed using the ECL technique.

Observed band size: 18,19 kDa

why is the actual band size different from the predicted?
Additional bands at: 60 kDa, 80 kDa. We are unsure as to the identity of these extra bands.

Exposure time: 5 minutes

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