Revision 3

#9784 Store at -20C

Alzheimer's Disease Antibody Sampler Kit

1 Kit (8 x 20 microliters)

Cell Signaling TECHNOLOGY®

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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
β-Amyloid (D54D2) XP® Rabbit mAb	8243	20 µl	5 kDa	Rabbit IgG
Neurofilament-L (C28E10) Rabbit mAb	2837	20 µl	70 kDa	Rabbit IgG
Tau (Tau46) Mouse mAb	4019	20 µl	50-80 kDa	Mouse IgG1
BACE1 (D10E5) Rabbit mAb	5606	20 µl	70 kDa	Rabbit IgG
APP/β-Amyloid (NAB228) Mouse mAb	2450	20 µl	100 to 140 kDa	Mouse IgG2a
α-Synuclein (Syn204) Mouse mAb	2647	20 µl	18 kDa	Mouse IgG2a
GSK-3α/β (D75D3) Rabbit mAb	5676	20 µl	51, 46 kDa	Rabbit IgG
Phospho-GSK-3α (Ser21) (36E9) Rabbit mAb	9316	20 µl	51 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat
Anti-mouse IgG, HRP-linked Antibody	7076	100 µl		Horse

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

The Alzheimer's Disease Antibody Sampler Kit provides an economical means of evaluating Alzheimer's disease-related signaling. The kit contains enough primary and secondary antibodies to perform two western blot experiments per primary antibody.

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Background

Alzheimer's disease (AD) is one of the most common neurodegenerative diseases worldwide. Clinically, it is characterized by the presence of extracellular amyloid plaques and intracellular neurofibrillary tangles, which results in neuronal dysfunction and cell death. Central to this disease is the differential processing of the integral transmembrane glycoprotein Amyloid β (A4) precursor protein (APP) that exists as several isoforms (1). The amino acid sequence of APP contains the amyloid domain, which can be released by a two-step proteolytic cleavage (1). β-secretase (BACE) is an aspartic acid proteinase that catalyses the initial step in APP processing by cleaving and releasing a soluble, extracellular APP-β (sAPPβ) ectodomain and generating a membrane-bound, carboxy-terminal fragment consisting of 99 amino acids (CTF99). Additional processing of CTF99 by γ-secretase generates the amyloid β-peptide (Aβ) that forms aggregates in the brains of AD patients. BACE is an attractive target for inhibitors in AD therapy since it catalyses the first and rate limiting step in amyloidogenic APP processing (2). Pro-BACE-1 is synthesized in the ER before it is transported to the trans-Golgi network to undergo maturation (3). The extracellular deposition and accumulation of the released Aβ fragments and an α-synuclein fragment known as the non- Aβ fragment, form the main components of amyloid plaques in AD. GSK-3α regulates the production of Aβ peptides. Administration of therapeutic concentrations of lithium, a GSK-3 inhibitor, attenuates Aβ production by specifically inhibiting the cleavage of APP by γ-secretase, thereby blocking accumulation of Aβ peptides in the brains of mice that overproduce APP (4). AD is also characterized by the presence of neurofibrillary tangles. These tangles are the result of hyperphosphorylation and oligomerization of the microtubule associated protein Tau and lead to apoptosis of the neuron. In particular, phosphorylation of Tau Ser396 by GSK-3

Background References

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3. Walter, J. et al. (2001) *J Biol Chem* 276, 14634-41.
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7. Al-Chalabi, A. and Miller, C.C. (2003) *Bioessays* 25, 346-55.

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