Product Includes

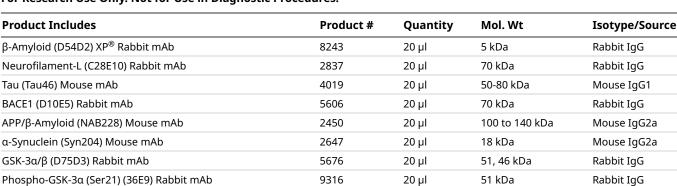
Tau (Tau46) Mouse mAb

BACE1 (D10E5) Rabbit mAb

Anti-rabbit IgG, HRP-linked Antibody

Anti-mouse IgG, HRP-linked Antibody

Alzheimer's Disease Antibody Sampler Kit Store at -20C 1 Kit (8 x 20 microliters) 84 For Research Use Only. Not for Use in Diagnostic Procedures.



100 µl

100 µl

7076 Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

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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 (sAPP β) 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 (A β) 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 A β fragments and an α -synuclein fragment known as the non- A β fragment, form the main components of amyloid plaques in AD. GSK-3 α regulates the production of A β 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	 Selkoe, D.J. (1996) <i>J Biol Chem</i> 271, 18295-8. Hunt, C.E. and Turner, A.J. (2009) <i>FEBS J</i> 276, 1845-59. Walter, J. et al. (2001) <i>J Biol Chem</i> 276, 14634-41. Phiel, C.J. et al. (2003) <i>Nature</i> 423, 435-9. Johnson, G.V. and Stoothoff, W.H. (2004) <i>J Cell Sci</i> 117, 5721-9. Bramblett, G.T. et al. (1993) <i>Neuron</i> 10, 1089-99. Al-Chalabi, A. and Miller, C.C. (2003) <i>Bioessays</i> 25, 346-55.



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Goat

Horse

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