Revision 1

Store at -20C

#85314

β-Amyloid Antibody Sampler Kit

1 Kit (7 x 20 microliters)

Cell Signaling TECHNOLOGY®

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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
APP Antibody	2452	20 µl	100 to 140 kDa	Rabbit
β-Amyloid (1-42) (D3E10) Rabbit mAb	12843	20 µl	4 kDa	Rabbit
β-Amyloid (1-40) (D8Q7I) Rabbit mAb	12990	20 µl	4 kDa	Rabbit IgG
β-Amyloid (1-37) (D2A6H) Rabbit mAb	12467	20 µl	4 kDa	Rabbit IgG
β-Amyloid (1-39) (D5Y9L) Rabbit mAb	12077	20 µl	4 kDa	Rabbit IgG
β-Amyloid (D54D2) XP® Rabbit mAb	8243	20 µl	5 kDa	Rabbit IgG
β-Amyloid (pE3 Peptide) (D5N5H) Rabbit mAb	14975	20 µl	4 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

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

Description

The β-Amyloid Antibody Sampler Kit provides an economical means of detecting APP and APP unmodified/modified fragments using total and fragment-specific antibodies. The kit includes enough antibody to perform two western blot experiments with each 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

Amyloid β (Aβ) precursor protein (APP) is a 100-140 kDa transmembrane glycoprotein that exists as several isoforms (1). The amino acid sequence of APP contains an amyloid domain, which can be processed and released by two-step proteolytic cleavage (1). The extracellular deposition and accumulation of the released Aβ fragments form the main components of amyloid plaques in Alzheimer's disease (1). Several fragments corresponding to progressive APP processing at alternative cleavage sites have been identified (2). These include Aβ (1-37), Aβ (1-39), Aβ (1-40), and Aβ (1-42) (2). These fragments can also be N-terminally modified to generate pyroglutamate-3 Aβ (pE3-peptide) (3). Fragment-specific and pan-Aβ antibodies are used to detect and examine relative levels of individual Aβ fragments.

Background References

1. Selkoe, D.J. (1996) *J Biol Chem* 271, 18295-8.
2. Selkoe, D.J. and Hardy, J. (2016) *EMBO Mol Med* 8, 595-608.
3. Saido, T.C. et al. (1995) *Neuron* 14, 457-66.

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