SY SY Synaptic Systems

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Abeta-pE3

Cat.No. 218 011; Monoclonal mouse antibody, 100 µg purified IgG (lyophilized)

Data Sheet

Reconstitution/ Storage	100 μg purified IgG, lyophilized. Azide was added before lyophilization. For reconstitution add 100 μH_2O to get a 1mg/ml solution in PBS. Then aliquot and store at -20°C until use.
Applications	WB: 1 : 1000 (see remarks) IP: not tested yet ICC: not tested yet IHC: 1 : 400 (see remarks) IHC-P/FFPE: 1 : 100 ELISA: yes (see remarks)
Clone	2-48
Subtype	IgG1 (κ light chain)
Immunogen	Synthetic peptide corresponding to AA 3 to 7 from human Abeta-pE3 (UniProt Id: P05067)
Epitop	Epitop: AA 3 to 7 from human Abeta-pE3 (UniProt Id: P05067)
Reactivity	Reacts with: human (P05067), rat (P08592), mouse (P12023), monkey. Other species not tested yet.
Specificity	Specific for Abeta-pE3.
Remarks	WB : Detects purified Abeta pE3. Complex samples like brain extracts still have to be tested. Boil membrane after blotting for 3min.
	IHC: recommended protocol
	ELISA : The biotinylated antibody (218 011BT) is suitable as detector antibody for sandwich-ELISA with cat. no. 218 111 or 218 511 as capture antibody (protocol for sandwich-ELISA).

TO BE USED IN VITRO / FOR RESEARCH ONLY NOT TOXIC, NOT HAZARDOUS, NOT INFECTIOUS, NOT CONTAGIOUS

Amyloid deposits, also called plaques, of Alzheimer's patients consist of several protein components like the **a**myloid **beta**-peptides (**Abeta**, **A** β) 1-40/42 and additional C- and N-terminally truncated and modified fragments. Very abundant are the isoaspartate (isoAsp)-Abeta and **p**yro**glu**tamyl (**pGlu**)-Abeta peptides. The latter are formed by cyclization of the N-terminal glutamate at position 3 or 11 catalyzed by glutaminyl cyclase (QC) resulting in very amyloidogenic and neurotxic variants of Abeta; **Abeta-pE3** and Abeta pE11.

In contrast to extracellular plaques that do not perfectly correlate with Alzheimer's disease

intraneuronal Abeta accumulation and vascular Abeta deposits have gained more and more evidence to be among the crucial factors responsible for progressive neuron loss.

Selected References SYSY Antibodies

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Pyroglutamate amyloid β (Aβ) aggravates behavioral deficits in transgenic amyloid mouse model for Alzheimer disease. Wittnam JL, Portelius E, Zetterberg H, Gustavsson MK, Schilling S, Koch B, Demuth HU, Blennow K, Wirths O, Bayer TA The Journal of biological chemistry (2012) 287(11): 8154-62. **IP**

Glutaminyl cyclase contributes to the formation of focal and diffuse pyroglutamate (pGlu)-Aβ deposits in hippocampus via distinct cellular mechanisms.

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The presubiculum is preserved from neurodegenerative changes in Alzheimer's disease.

Murray CE, Gami-Patel P, Gkanatsiou E, Brinkmalm G, Portelius E, Wirths O, Heywood W, Blennow K, Ghiso J, Holton JL, Mills K, et al.

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Detection and Quantification of β-Amyloid, Pyroglutamyl Aβ, and Tau in Aged Canines. Schmidt F, Boltze J, Jäger C, Hofmann S, Willems N, Seeger J, Härtig W, Stolzing A Journal of neuropathology and experimental neurology (2015) 74(9): 912-23. **IHC**

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Identification of thyrotropin-releasing hormone as hippocampal glutaminyl cyclase substrate in neurons and reactive astrocytes.

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Journal of Alzheimer's disease : JAD (2014) 39(2): 385-400. IHC; tested species: human

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Acta neuropathologica (2014) 127(6): 787-801. IHC-P; tested species: human

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The Arctic AβPP mutation leads to Alzheimer's disease pathology with highly variable topographic deposition of differentially truncated Aβ.

Kalimo H, Lalowski M, Bogdanovic N, Philipson O, Bird TD, Nochlin D, Schellenberg GD, Brundin R, Olofsson T, Soliymani R, Baumann M, et al.

Acta neuropathologica communications (2013) 1: 60. IHC; tested species: human

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Involvement of perineuronal and perisynaptic extracellular matrix in Alzheimer's disease neuropathology. Morawski M, Brückner G, Jäger C, Seeger G, Matthews RT, Arendt T Brain pathology (Zurich, Switzerland) (2012) 22(4): 547-61. **IHC; tested species: human**