

## Abeta-pE3

Cat.No. 218 311; 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 µl H <sub>2</sub> O to get a 1mg/ml solution in PBS. Then aliquot and store at -20°C until use.
Applications	<b>WB:</b> 1 : 1000 (see remarks) <b>IP:</b> yes <b>ICC:</b> not tested yet <b>IHC:</b> 1 : 100 (see remarks) <b>IHC-P/FFPE:</b> 1 : 1000 up to 1 : 5000
Clone	1-57
Subtype	IgG2b (κ light chain)
Immunogen	Synthetic peptide corresponding to AA 3 to 7 from human Abeta-pE3 (UniProt Id: P05067)
Epitop	Epitop: AA 3 to 5 from human Abeta-pE3 (UniProt Id: P05067)
Reactivity	Reacts with: human (P05067), rat (P08592), mouse (P12023). Other species not tested yet.
Specificity	Specific for Abeta-pE3.
Remarks	<b>WB:</b> Detects purified Abeta pE3. Complex samples like brain extracts still have to be tested. Boil membrane after blotting for 3min.  <b>IHC:</b> recommended protocol

### 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 amyloid beta-peptides (**Abeta**, **Aβ**) 1-40/42 and additional C- and N-terminally truncated and modified fragments. Very abundant are the isoaspartate (isoAsp)-Abeta and pyroglutamyl (**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 neurotoxic 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

N-truncated Abeta starting with position four: early intraneuronal accumulation and rescue of toxicity using NT4X-167, a novel monoclonal antibody.

Antonios G, Saiepour N, Bouter Y, Richard BC, Paetau A, Verkoniemi-Ahola A, Lannfelt L, Ingelsson M, Kovacs GG, Pillot T, Wirths O, et al.

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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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Pyroglutamate amyloid β (Aβ) aggravates behavioral deficits in transgenic amyloid mouse model for Alzheimer disease.

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### Selected General References

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Anti-11[E]-pyroglutamate-modified amyloid β antibodies cross-react with other pathological Aβ species: relevance for immunotherapy.

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Inhibition of glutaminyl cyclase prevents pGlu-Abeta formation after intracortical/hippocampal microinjection in vivo/in situ.

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Physical, morphological and functional differences between pH 5.8 and 7.4 aggregates of the Alzheimer's amyloid peptide Abeta.

Wood SJ, Maleeff B, Hart T, Wetzel R

Journal of molecular biology (1996) 256(5): 870-7.

Water-soluble Abeta (N-40, N-42) oligomers in normal and Alzheimer disease brains.

Kuo YM, Emmerling MR, Vigo-Pelfrey C, Kasunic TC, Kirkpatrick JB, Murdoch GH, Ball MJ, Roher AE

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