

Abeta-pE11

Cat.No. 218 811; 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 ₂ O 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 : 200 (see remarks) IHC-P/FFPE: 1 : 500 (see remarks)
Clone	173D8
Subtype	IgG2b (κ light chain)
Immunogen	Synthetic peptide corresponding to AA 11 to 16 from human Abeta-pE11 (UniProt Id: P05067)
Epitop	Epitop: AA 11 to 16 from human Abeta-pE11 (UniProt Id: P05067)
Reactivity	Reacts with: human (P05067), rat (P08592), mouse (P12023). Other species not tested yet.
Specificity	Specific for Abeta-pE11.
Remarks	WB: Detects purified Abeta-pE11. complex samples like brain extracts not tested yet. Boil membrane after blotting for 3min. IHC: Formic acid treatment required recommended protocol. IHC-P: Formic acid treatment required.

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 glutaminy 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

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. Acta neuropathologica communications (2018) 6(1): 62. **IHC-P; tested species: human**

Selected General References

Pyroglutamate-Aβ 3 and 11 colocalize in amyloid plaques in Alzheimer's disease cerebral cortex with pyroglutamate-Aβ 11 forming the central core.

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Hook V, Schechter I, Demuth HU, Hook G Biological chemistry (2008) 389(8): 993-1006.

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Inhibition of glutaminy cyclase prevents pGlu-Abeta formation after intracortical/hippocampal microinjection in vivo/in situ. Schilling S, Appl T, Hoffmann T, Cynis H, Schulz K, Jagla W, Friedrich D, Wermann M, Buchholz M, Heiser U, von Hörsten S, et al. Journal of neurochemistry (2008) 106(3): 1225-36.

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

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