Synaptic Systems

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Oligo-Abeta-pE3

Cat.No. 218 511; 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_2O to get a 1mg/ml solution in PBS. Then aliquot and store at -20°C until use.
Applications	**WB**: 1 : 500 (see remarks) **IP**: not tested yet **ICC**: not tested yet **IHC**: 1 : 100 **IHC-P/FFPE**: 1 : 100 (see remarks) **ELISA**: yes (see remarks)
Important note for users	The mouse monoclonal antibody clone 9D5, Cat. No. 218 511, is patented (patent application PCT/EP2011/002739). By purchasing this antibody the customer acquires rights to use this product for research purposes only. Any diagnostic and therapeutic in vitro or in vivo use is explicitly excluded.
Clone	9D5
Subtype	IgG2b (κ light chain)
Immunogen	Synthetic peptide corresponding to AA 3 to 38 from human Oligo-Abeta-pE3 (UniProt Id: P05067)
Epitop	Epitop: AA 3 to 38 from human Oligo-Abeta-pE3 (UniProt Id: P05067)
Reactivity	Reacts with: human (P05067), mouse (P12023). Other species not tested yet.
Specificity	Recognizes specific oligomeric structures formed preferentially by Abeta-pE3.
Remarks	**WB**: We recommend the Invitrogen NativePAGE system in combination with PVDF blotting membranes. Boil membrane after blotting for 3min. **Peptide preparation**: Synthetic Abeta peptides were monomerized in 70 % formic acid, and the solvent was evaporated in a speed-vac immediately. Prior to each experiment, peptides were dissolved in 0.3 % ammonia, underwent ultrasonic treatment, and were further diluted to an end concentration of 0.15 % ammonia. **IHC-P**: Formic acid treatment required. **ELISA**: Suitable as capture antibody for sandwich-ELISA with cat. no. 218 011BT as detector 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**, **Aβ**) 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

Identification of low molecular weight pyroglutamate A{beta} oligomers in Alzheimer disease: a novel tool for therapy and diagnosis.
Wirths O, Erck C, Martens H, Harmeier A, Geumann C, Jawhar S, Kumar S, Multhaup G, Walter J, Ingelsson M, Degerman-Gunnarsson M, et al.
The Journal of biological chemistry (2010) 285(53): 41517-24. **WB, IHC, ELISA**

Focusing the amyloid cascade hypothesis on N-truncated Abeta peptides as drug targets against Alzheimer's disease.
Bayer TA, Wirths O
Acta neuropathologica (2014) 127(6): 787-801. **IHC-P; tested species: human**

I716F AβPP mutation associates with the deposition of oligomeric pyroglutamate amyloid-β and α-synucleinopathy with Lewy bodies.
Sieczkowski E, Milenkovic I, Venkataramani V, Giera R, Ströbel T, Höftberger R, Liberski PP, Auff E, Wirths O, Bayer TA, Kovacs GG, et al.
Journal of Alzheimer's disease : JAD (2015) 44(1): 103-14. **IHC; tested species: human**

Oxidative Stress during the Progression of β-Amyloid Pathology in the Neocortex of the Tg2576 Mouse Model of Alzheimer's Disease.
Porcellotti S, Fanelli F, Fracassi A, Sepe S, Cecconi F, Bernardi C, Cimini A, Cerù MP, Moreno S
Oxidative medicine and cellular longevity (2015) 2015: 967203. **IHC**

Oligomeric pyroglutamate amyloid-β is present in microglia and a subfraction of vessels in patients with Alzheimer's disease: implications for immunotherapy.
Wirths O, Hillmann A, Pradier L, Härtig W, Bayer TA
Journal of Alzheimer's disease : JAD (2013) 35(4): 741-9. **IHC**

Antibody 9D5 recognizes oligomeric pyroglutamate amyloid-β in a fraction of amyloid-β deposits in Alzheimer's disease without cross-reactivity with other protein aggregates.
Venkataramani V, Wirths O, Budka H, Härtig W, Kovacs GG, Bayer TA
Journal of Alzheimer's disease : JAD (2012) 29(2): 361-71. **IHC**

Intraneuronal Aβ as a trigger for neuron loss: can this be translated into human pathology?
Bayer TA, Wirths O
Biochemical Society transactions (2011) 39(4): 857-61.

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.
Sullivan CP, Berg EA, Elliott-Bryant R, Fishman JB, McKee AC, Morin PJ, Shia MA, Fine RE
Neuroscience letters (2011) 505(2): 109-12.

Anti-11[E]-pyroglutamate-modified amyloid β antibodies cross-react with other pathological Aβ species: relevance for immunotherapy.
Perez-Garmendia R, Ibarra-Bracamontes V, Vasilevko V, Luna-Muñoz J, Mena R, Govezensky T, Acero G, Manoutcharian K, Cribbs DH, Gevorkian G
Journal of neuroimmunology (2010) 229(1-2): 248-55.

Glutaminyl cyclase inhibition attenuates pyroglutamate Abeta and Alzheimer's disease-like pathology.
Schilling S, Zeitschel U, Hoffmann T, Heiser U, Francke M, Kehlen A, Holzer M, Hutter-Paier B, Prokesch M, Windisch M, Jagla W, et al.
Nature medicine (2008) 14(10): 1106-11.

Alternative pathways for production of beta-amyloid peptides of Alzheimer's disease.
Hook V, Schechter I, Demuth HU, Hook G
Biological chemistry (2008) 389(8): 993-1006.