

Abeta 38/40/42/43

Cat.No. 218 711; 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: yes IP: not tested yet ICC: not tested yet IHC: 1 : 100 up to 1 : 500 (see remarks) IHC-P/FFPE: 1 : 500 (see remarks)
Clone	88B12
Subtype	IgG2b (κ light chain)
Immunogen	Synthetic peptide corresponding to AA 17 to 26 from human Abeta (UniProt Id: P05067)
Epitop	Epitop: AA 17 to 26 from human Abeta (UniProt Id: P05067)
Reactivity	Reacts with: human (P05067), mouse (P12023). Other species not tested yet.
Specificity	Specific for Abeta 38, 40, 42.
Remarks	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**) 1-40/42/43 and additional C- and N-terminally modified fragments of Abeta as for instance Abeta pE3 and Abeta pE11.

An additional Abeta variant, **Abeta 38**, is more soluble compared to other Abeta species and is not found in plaques of sporadic Alzheimer's cases. However, it is detected in the blood-vessel walls of a subset of patients with severe cerebral amyloid angiopathy. It especially accumulates in brains of patients carrying mutations in the Abeta coding region.

Cleavage of amyloid precursor protein APP by β- and γ- secretases results in the generation of the Aβ (βA4)peptide, whereas α-secretase cleaves within the Aβ sequence and prevents the formation of Abeta from APP.

Selected General References

Circulating immune complexes of Abeta and IgM in plasma of patients with Alzheimer's disease. Marcello A, Wirths O, Schneider-Axmann T, Degerman-Gunnarsson M, Lannfelt L, Bayer TA. Journal of neural transmission (Vienna, Austria : 1996) (2009) 116(7): 913-20.

Immune response to Abeta-peptides in peripheral blood from patients with Alzheimer's disease and control subjects. Baril L, Nicolas L, Croisile B, Crozier P, Hessler C, Sassolas A, McCormick JB, Trannoy E. Neuroscience letters (2004) 355(3): 226-30.

Dietary Cu stabilizes brain superoxide dismutase 1 activity and reduces amyloid Abeta production in APP23 transgenic mice. Bayer TA, Schäfer S, Simons A, Kemmling A, Kamer T, Tepest R, Eckert A, Schüssel K, Eikenberg O, Sturchler-Pierrat C, Abramowski D, et al. Proceedings of the National Academy of Sciences of the United States of America (2003) 100(24): 14187-92.

Correlative memory deficits, Abeta elevation, and amyloid plaques in transgenic mice. Hsiao K, Chapman P, Nilsen S, Eckman C, Harigaya Y, Younkin S, Yang F, Cole G. Science (New York, N.Y.) (1996) 274(5284): 99-102.

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. The Journal of biological chemistry (1996) 271(8): 4077-81.