IBL Data Sheet

Code No. 10379

Anti-Human
Amyloidβ E22P (11A1) Mouse IgG MoAb

Volume : 50 µg

Introduction : Alzheimer's disease (AD) is characterized by the presence of extracellular plaques and intracellular neurofibrillary tangles (NFTs) in the brain. Aggregation of the 42-mer amyloid β-protein (Aβ42) plays a critical role in the pathogenesis of AD. Shirasawa and Irie et. al have proposed a toxic conformer with a turn at positions 22 and 23, as well as a nontoxic conformer with a turn at positions 25 and 26, in Aβ42 aggregates from systematic proline scanning and solid-state NMR studies (ref. 1-3). This monoclonal antibody named 11A1 was developed for toxic Aβ42, using E22P-Aβ10-35, a minimum moiety for neurotoxicity containing the turn at positions 22 and 23, for the generation. Immunohistochemical studies showed that not only extracellular but intracellular amyloid was stained in human AD brains (ref. 4), which suggest that 11A1 could detect toxic oligomers of Aβ with the turn at positions 22 and 23.

Antigen : Synthetic peptide of E22P- Amyloidβ10-35 part

Source : Mouse-Mouse hybridoma
(X63 - Ag 8.653 × BALB/c mouse spleen cells)

Clone : 11A1 **Subclass** : IgG_1

Purification : Protein A purified

Form : Lyophilized product from PBS containing 1 % BSA and 0.05 % NaN_3

How to use : 1.0 mL deionized water will be added to the product, then its concentration comes to 50 µg/mL

Stability : Lyophilized product, 5 years at 2 - 8 °C
: Solution, 2 years at –20 °C

Application : This antibody can be used for immunohistochemistry with formalin fixed paraffin embedded tissues after formic acid treatment*[1]. The optimal concentration is 0.5-1.0 µg/mL, however, the concentration should be optimized by each laboratory.
*1: Rinse by running water after formic acid treatment for 5 minutes following de-paraffin.
: This antibody can be used for western blotting (by SDS-PAGE under 2ME(-) condition/ nonreducing condition) at the concentration of 0.5 - 1.0 µg/mL.
: This antibody can be used for immuno-precipitation.

Specificity : Reacts with native human Amyloidβ 1-40, 1-42

Reference :

1. Morimoto A, Irie K, Murakami K, Masuda Y, Ohigashi H, Nagao M, Fukuda H, Shimizu T, Shirasawa T. Analysis of the secondary structure of beta-amyloid (Abeta42) fibrils by systematic proline replacement. J Biol Chem. 2004 Dec 10;279(50):52781-8.
2. Murakami K, Irie K, Ohigashi H, Hara H, Nagao M, Shimizu T, Shirasawa T. Formation and stabilization model of the 42-mer Abeta radical: implications for the long-lasting oxidative stress in Alzheimer's disease. J Am Chem Soc. 2005 Nov 2;127(43):15168-74.
3. Masuda Y, Uemura S, Ohashi R, Nakanishi A, Takegoshi K, Shimizu T, Shirasawa T, Irie K. Identification of physiological and toxic conformations in Abeta42 aggregates. Chembiochem. 2009 Jan 26;10(2):287-95.
4. Murakami K, Horikoshi-Sakuraba Y, Murata N, Noda Y, Masuda Y, Kinoshita N, Hatsuta H, Murayama S, Shirasawa T, Shimizu T, Irie K. Monoclonal Antibody Against the Turn of the 42-Residue Amyloid β-Protein at Positions 22 and 23. ACS Chem. Neurosci. 2010 Sept 28;1(11):747-56.

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