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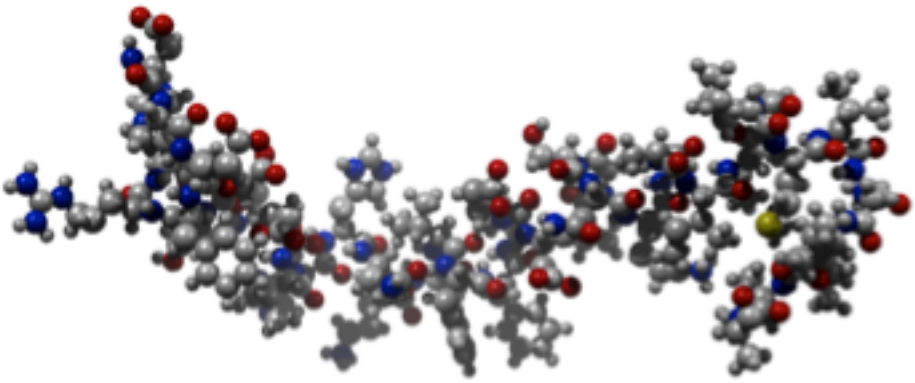
GOT MOAB-2??

MOAB-2: New highly specific amyloid beta (Aβ) monoclonal antibody designed to facilitate and enhance research into Aβ neurotoxicity and association with the neuropathology of Alzheimer's disease.

Alzheimer's disease (AD) is one of the most common forms of dementia (1 http://alzheimers.org.uk/Facts_about_dementia/What_is_dementia/). The disease usually occurs in old age, and is marked by a decline in cognitive reasoning and function. Cognitive memory, reasoning, planning and decision-making are all affected. While the complete cause of AD remains to be determined, amyloid beta protein has long been associated with AD pathology. However the exact nature of the form or forms of amyloid-β peptide (Aβ) associated with the pathology characteristic of AD still remains elusive. In particular, the neurotoxicity of intraneuronal Aβ accumulation is an issue of considerable controversy today, and even the existence of Aβ intraneuronal deposits themselves within neurons has recently been challenged by scientists largely because of the lack of good, highly specific antibodies to Aβ that truly provide the clarity and staining definition that is sorely needed - until now. Biosensis is pleased offer the monoclonal MOAB-2 (catalog number M-1586-100): A new pan-specific, high-titer antibody to Aβ residues 1-4 that provides stronger reactivity, greater sensitivity and unparalleled staining clarity than any other Aβ monoclonal previously available for research. (Image: Amyloid Beta 1-42 peptide http://randombio.com/alz.html).



MOAB-2: Unprecedented features, unparalleled staining & clarity!

MOAB-2 does not cross react with APP or APP C-terminal fragments (unlike other monoclonals such as 6E10 and 4G8) thus allowing the true discrimination and localization of Aβ protein distinct from that of APP.

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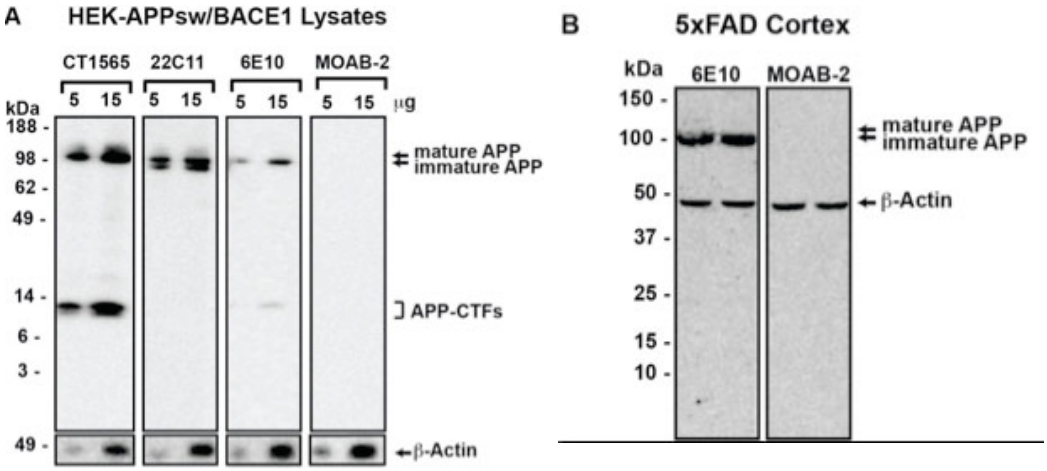

Youmans et al. Molecular Neurodegeneration 2012, 7:8
(Image: MOAB-2 does not detect APP in cell culture media and lysates or cortical brain extracts from 5xFAD mice. (A) Western-blot analysis of 5 µg or 15 µg cell lysates from HEK-APPSwe/BACE1 cells, probed with antibodies against C-terminus of APP (CT1565), N-terminus of APP (2211), Aβ (6E10, MOAB-2) or β-Actin (loading control). Notice all the other clones detect APP or APP-CTFs, but that MOAB-2 does not. (B) Western analysis of 25 µg total protein from detergent-extracted 5xFAD mouse cortex probed with 6E10 or MOAB-2 and β-Actin for loading control demonstrating that MOAB-2 does not detect APP forms, unlike 6E10).

Recent research indicates that the configuration of Aβ protein, particularly Aβ 42 maybe important in the overall neurotoxicity of the protein to neurons. In dot blots, MOAB-2 recognizes all forms of Aβ 42 protein (usually denoted as unaggregated (U), oligomeric (O), and fibrillar (F) forms), and the unaggregated form of AB40. Moreover, MOAB-2 is selective for the more neurotoxic Aβ42 compared to Aβ40. In titration tests, MOAB-2 demonstrated a 25 fold greater sensitivity of detection for the U-, O- and F-Aβ42 forms than for Aβ40, for instance, thus it is superb reagent for focusing in the potential localization and function of Aβ42.

Localization of beta-amyloid protein and in particular the establishment of intraneuronal forms of Aβ protein plays a critical role in our growing understanding of its pathology. MOAB-2 is an exceptionally good immunohistochemical reagent, reacting in both immunohistochemical and immunofluorescent applications, including confocal microscopy, with very low backgrounds and very low signal to noise ratios. Moreover, MOAB-2 reacts in all forms of tissue preparations from fresh frozen to archival formalin fixed paraffin embedded tissues. Antigen retrieval is required for the best staining and multiple methods can be used, however, heat-induced epitope recovery (HEIR) or formic acid pretreatments (88% FA 8 min) provide the most consistent results in our testing. MOAB-2 antibody detects intraneuronal and extracellular beta-amyloid in IHC and does not detect APP {Youmans. KL et al 2012}.

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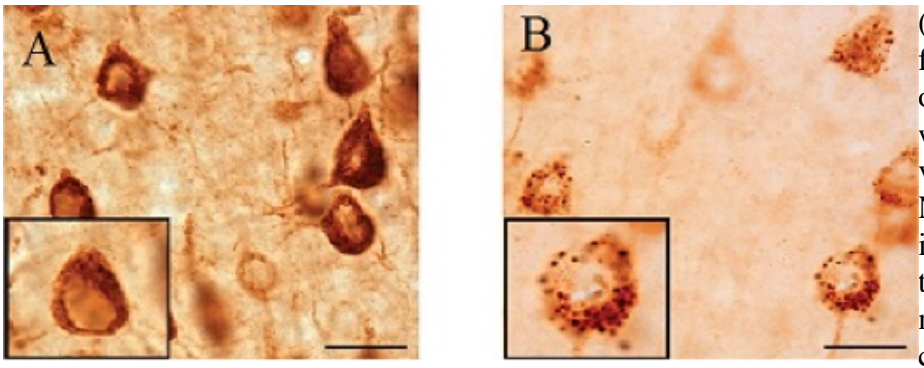
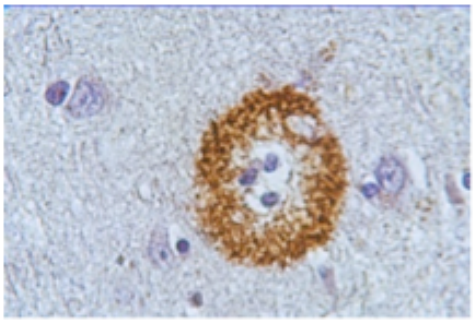


(Image: Coronal sections of the frontal cortex from 1 and 3 month old 5xFAD mice immunostained with 6E10 and MOAB-2 and visualized via DAB staining. Notice how 6E10 is strongly immunoreactive across the field of the cortex, and at higher magnification shows that the cytoplasm is evenly stained with an immunonegative nuclei (**A**) indicating the detection of both APP and Aβ. In contrast, (**B**), MOAB-2 staining of sister sections is substantially less than for 6E10 and the intraneuronal staining is punctate. These results are consistent with MOAB-2 recognizing only Aβ and not APP).

MOAB-2 is also superb for staining the extracellular deposition of Aβ protein such as that seen in plague deposition. The clarity of stain that MOAB-2 can generate is unparalleled by any other reagent. As shown in the accompanying figure, extensive MOAB-2 immunopositive extracellular staining is observed and higher magnification reveals low levels of MOAB-2 intraneuronal immunoreactivity with significant staining of individual plaques as expected for an antibody capable of only detecting Aβ and not APP or APP-CTFs.



MOAB-2 staining of senile plaque in Alzheimer's diseased hippocampus.

Finally with MOAB-2, the discrimination between APP and true intraneuronal Aβ in all its forms can be achieved. Aβ accumulates prior to extracellular plaque deposition and decreases as plaque deposition increases. However, if the Aβ antibodies also detect APP, interpretation of the results can be problematic. In contrast, MOAB-2 discrimination is both possible and precise. Stunning fluorescent results such as these can now be achieved only with MOAB-2, further expanding the experimental research potential of mouse models and Aβ pathology. With the ability to consistently detect Aβ deposits and the ability of MOAB-2 to demonstrate specific, robust intraneuronal staining in vivo, this new Biosensis monoclonal antibody has the potential to expand and truly foster new investigations into the importance of beta amyloid in both mouse model and humans systems.

Biosensis is pleased to bring this new and exciting reagent to the market and we encourage all that have an interest in beta-amyloid or its pathology to try it. (M-1586-100).

To download more information about MOAB-2 click here

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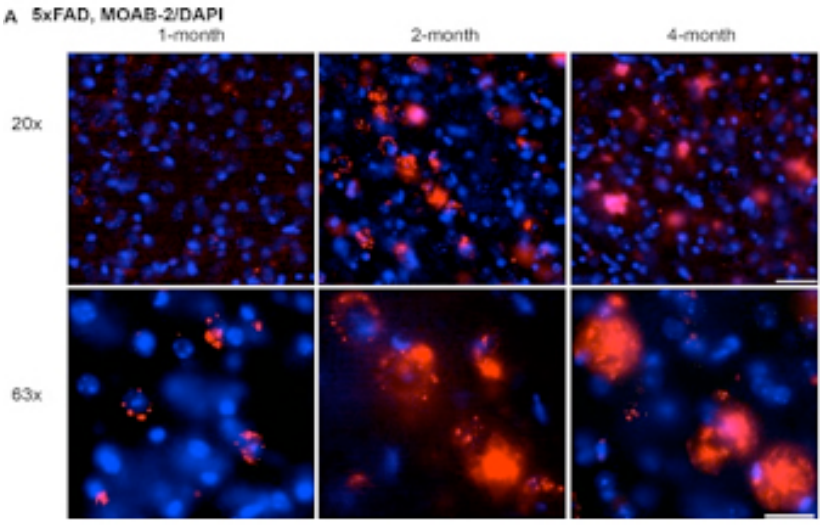
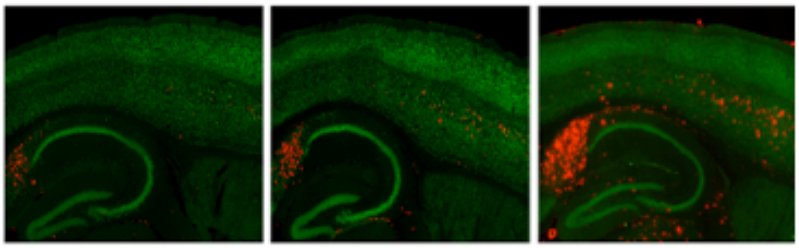


(Image: MOAB-2 detection of intraneuronal Aβ and extracellular plaques in 5xFAD mouse brain tissues. Immunofluorescent detection of Aβ with MOAB-2 in the subiculum of (A) 1, 2 and 4 month old 5xFAD mice. Aβ accumulates extracellularly as the disease progresses making MOAB-2 the ideal reagent for studying both intraneuronal Aβ at early stages and extracellular Aβ at late stages seamlessly).



IHC staining of MOAB-2 on 2, 4, 6 month old 5XFAD hippocampus sections showing increasing Aβ staining over time with MOAB-2 (Red) as the model disease increases; (Green) is a general neuronal marker stain (i.e. Fox3/NeuN).

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