Product Data Sheet

Product Name:	β-Amyloid (12-28)
Catalog Number:	AS-24229 (0.5 mg) Lot Number: See label on vial AS-24230 (1 mg)
Sequence:	H-Val-His-His-Gln-Lys-Leu-Val-Phe-Phe-Ala-Glu-Asp-Val-Gly-Ser-Asn-Lys-OH (3-letter code) VHHQKLVFFAEDVGSNK (1-letter code)
Molecular Weight:	1955.2
Peptide Purity:	>95%
Appearance:	Lyophilized white powder
Peptide Reconstitution:	β-Amyloid (12-28) peptide is freely soluble in water.

Storage: β-Amyloid (12-28) peptide is shipped at ambient temperature. Upon receipt, store lyophilized peptide at –20°C or lower. Reconstituted peptide can be aliquoted and stored at –20 °C or lower.

Description: Aβ (12–28) residues are the binding site for apolipoprotein E (apoE) on Aβ. This sequence encompasses a hydrophobic domain (residues 14–21) and a β-turn (residues 22–28) which place two hydrophobic domains of Aβ 14 to 21 and 29 to 40/42 opposite each other, allowing for the assembly of Aβ peptides into fibrils. The secondary structure of Aβ (12- 28), a neutral peptide, is dominated by a-helix and random coil. The interaction of apoE with residues 12 to 28 of Aβ is not just a non-specific hydrophobic interaction but plays a pivotal role in the mechanism of Aβ pathology in Alzheimer's disease (AD). Aβ (11-28) and five other fragments enhanced aggregation of full length Aβ (1-40). All of the peptides that enhance aggregation contained either residues 17 to 20 or 30 to 35, indicating the importance of these regions for promoting aggregation of full-length Aβ. Ref: Sadowski, M. et al. *Am. J. Pathol.* **165**, 937 (2004); Liu, R. et al. J. *Neurosci. Res.* **75**, 162 (2004).

Additional Information: *Listed below are relevant information that may provide a guideline on how to use this product. End users will have to adapt to their own specific applications.*

$A\beta_{1-11}$, $A\beta_{10-20}$, $A\beta_{15-20}$, $A\beta_{12-28}$, $A\beta_{25-35}$, $A\beta_{37-43}$, $A\beta_{29-40}$, biotinated $A\beta_{1-42}$, and FITC-conjugated $A\beta_{1-42}$ were obtained from AnaSpec. o determine whether exposure to exogenous $A\beta_{42}$ increases $A\beta_{42}$–α7nAChR association and causes $A\beta_{42}$-induced α7nAChR and NMDAR dysfunction, ~20 mg of FCX slices from either control subjects or AD individuals were incubated with 0.1 μM $A\beta_{42}$ at 37°C for 1 h. To test their effects, the following drugs were added immediately after $A\beta_{42}$: S 24795 (1–100 μM), $A\beta_{12-28}$ (10 μM), memantine (30 μM), galantamine (30 μM), PNU 282987 (30 μM), MLA (10 μM), or MLA (10 μM) plus S 24795 (10 μM). Incubation continued for 1 h in the dark to minimize light destruction of the test agents such as S 24795. The incubation mixture in a total incubation volume of 0.5 ml was aerated with 95% O_2/5% CO_2 every 15 min for

1 min during the incubation. Reaction was terminated by the addition of 1.5 ml of ice-cold Ca^{2+}-free K-R. Tissue slices were harvested by brief centrifugation and used as the tissue sources for various assays-Wang, H.Y. et al. *J Neuro* **10**, 10961 (2009).

Published Citations:

Mouedden, M. et al. *J. Neuro.* **145**, 97 (2005).
Osada, Y. et al. *JBC* **280**, 8596 (2005).
Solorzano-Vargas, RS. et al. *Mole. Immunol*. **45**, 881 (2008).
Wang, H.Y. et al. *J Neuro* **10**, 10961 (2009).

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