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Amyloid β -Peptide (1-42)

Amyloid β -Protein (1-42) sodium salt **H-7404**

Solvation of A β 42 with sodium hydroxide followed by lyophilization generates an Abeta peptide with superior solubility and fibrillogenesis characteristics.

Y.Fezoui et al., Amyloid 7, 166 (2000) / R.H.Takahashi et al., PLoS ONE 8, e51965 (2013)

Amyloid β -Protein (1-42) trifluoroacetate salt **H-8146**

(Des-Glu²²)-Amyloid β -Protein (1-42) (E22 Δ Osaka Japanese mutation) **H-7686**

The Osaka (E22delta) mutation of Amyloid β promotes β -sheet transformation, radical production, and synaptotoxicity, but not neurotoxicity.

O.Y.Ovchinnikova et al., J. Mol. Biol. 408, 780 (2011) / T.Suzuki et al., Int. J. Alzheimers Dis., 2010, 431320 (2011) / L.Kulic et al., Transl. Psychiatry, 2, e183 (2012)

FITC- ϵ Ahx-Amyloid β -Protein (1-42) (FITC-LC-Amyloid β -Protein (1-42)) **H-7666**

Fluorophore linked by a flexible spacer.

M. Gold et al., J. Neuroinflammation 10, 5 (2013) / R.S.Jones et al., J. Neuroimmune Pharmacol. 8, 301 (2013)

Amyloid β -Peptide (1-40)

Amyloid β -Protein (1-40) amide **H-7664**

Amyloid β -Protein (40-1) hydrochloride salt **H-7728**

Inactive control.

N.W.Kowall et al., Proc. Natl. Acad. Sci. USA, 88, 7247 (1991) / M.Yaar et al., J. Clin. Invest., 100, 2333 (1997)

(Arg¹³)-Amyloid β -Protein (1-40) (H13R) **H-7662**

H13R, a mutation in the metal-binding region of Abeta reduces its copper-mediated toxicity. The native rodent sequence containing an arginine at this position is more tolerant to metals than the human amyloid peptide.

X.Dai et al., J. Mol. Neurosci., 41, 66 (2010) / J.F.Poduslo et al., PLoS ONE, 5, e8813 (2010) / D.P.Smith et al., J. Biol. Chem., 281, 15145 (2006)

Biotinyl- ϵ Ahx-Amyloid β -Protein (1-40) **H-7456**

The flexible LC spacer increases the accessibility of the biotin moiety.

R.K.Sumbria et al., Bioconjug. Chem., 23, 1318 (2012) / C.L.Samuelsen and M.Meredith, Neuroscience, 180, 96 (2011)

Truncated Amyloid β -Peptides

(Val²)-Amyloid β -Protein (1-6) **H-8296**

G.Di Fede et al., Sci. Rep. 6, 20949 (2016)

Amyloid β -Protein (2-42) **H-7472**

A β 2-42 seems to be a promising biomarker for differentiating AD from other degenerative dementias, such as frontotemporal dementias (FTD). The peptide promotes phagocytosis by macrophages.

J.Wiltfang et al., J. Biol. Chem. 276, 42645 (2001) / A.Olsson et al., Clin. Chem. 51, 336-345 (2005) / M.Bibl et al., J. Alzheimers Dis. 29, 809 (2012) / M.Bibl et al., J. Neural Transm. 119, 805 (2012) / M.Condic et al., Brain. Behav. Immun. 41, 116 (2014)

Amyloid β -Protein (3-40) **H-7672**

Amyloid β -Protein (3-42) **H-7432**

The N-terminally truncated A β 42 may be formed in increased amounts as AD progresses. A β 3-42 is the precursor of the Pyr-peptide. On basis of a mouse model Wirhth et al. propose that (Pyr³)-A β 3-42 (H-8248 /H-4796) positive plaques are resistant to age-dependent degradation due to their high stability and propensity to aggregate.

G.McColl et al., J. Biol. Chem. 284, 22697 (2009) / O.Wirhth et al., J. Neural Transm. 117, 85 (2010) / A.Becker et al., BMC Neurosci. 14, 108 (2013)

(Pyr³)-Amyloid β -Protein (3-42) trifluoroacetate salt **H-8248**



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Amyloid β -Protein (4-42)

H-7434

A β 4-42 could be one of the earliest and most prominent A β species deposited in AD brain. Sequencing of amyloid plaque cores showed that 64% of the isolated A β had a phenylalanine at its N-terminus, and indeed, IP/MS experiments identified A β 4-42 as a major A β species in AD patients. Additionally, A β 4-42 was found to be a component of cotton wool plaques in familial AD patients with the V261I PS1 mutation. A β 4-42 was discovered as well in amyloid deposits from vascular dementia and familial Danish dementia patients. These observations indicate that A β 4-42 may contribute to the development of multiple CNS diseases.

Y.Tomidokoro et al., J. Biol. Chem. 280, 36883 (2005) / E.Portelius et al., Acta Neuropathol. 120, 185 (2010) / G.Antonios et al., Acta Neuropathol. Commun. 1, 56 (2013) / Y.Bouter et al., Acta Neuropathol. 126, 189 (2013)

Amyloid β -Protein (5-42)

H-7436

Abeta 5-42 is produced from amyloid precursor protein by action of caspases. It is deposited in Alzheimer's disease brain as well, but less prone to aggregation.

K.Takeda et al., FASEB J. 18, 755 (2004)

Amyloid β -Protein (11-42)

H-7668

Amyloid β -Protein (16-22)

H-8092

Self-assembling A β sequence.

K.D.Klimov and D. Thirumalai, Structure 11, 295 (2003) / K.Tao et al., Langmuir 27, 2723 (2011)

Acetyl-(N-Me-Leu¹⁷,N-Me-Phe¹⁹)-Amyloid β -Protein (16-20)

H-7658

The backbone-methylated fragment Ac-Lys-MeLeu-Val-MePhe-Phe-NH₂ is a membrane-permeable inhibitor of A β (1-40) fibrillogenesis.

P.Soto et al., Biophys. J., 93, 3015 (2007) / D.J.Gordon and S.C.Meredith, Biochemistry, 42, 475 (2003) /

Amyloid β -Protein (17-40)

H-7532

Cleavage of APP by α - and γ -secretase (i.e. the non-amyloidogenic pathway) yields p3 peptide, a mix of A β 17-40 and A β 17-42. p3 is a major constituent of diffuse

plaques observed in AD brains and pre-amyloid plaques in people affected by Down syndrome.

F.Dulin et al., FEBS Lett. 582, 1865 (2008) / H.Jang et al., Proc. Natl. Acad. Sci. USA 107, 6538 (2010) / W.Han et al., Med. Hypotheses 76, 543 (2011)

APP Mutation

Amyloid Precursor Frameshift Mutant C-Terminal Peptide

H-7674

The sequence RGR^TSSKELA corresponds to human APP frameshift mutant (339-348). RGR^TSSKELA is used for generating antibodies.

F.W.van Leeuwen et al., Science, 279, 242 (1998) / F.W.van Leeuwen et al., Trends Neurosci., 21, 331 (1998)

Scrambled Amyloid- β Peptides

Amyloid β -Protein (1-42) (scrambled)

H-7406

Inactive control for A β 42 H-1368 and H-6466.

S.Soscia et al., PLoS ONE, 5, e9505 (2010) / J.H.Walton et al., Biophys. J. 100, 2302 (2011)

5-FAM-Amyloid β -Protein (1-42) (scrambled)

H-7836

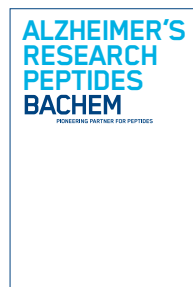
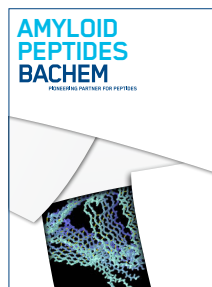
Amyloid β -Protein (1-40) (scrambled)

H-7408

Inactive control for A β 40 H-1194 and H-5568.

Y.Ji et al., J. Alzheimers Dis. 3, 23 (2001)

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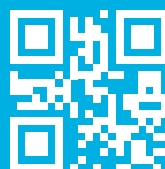
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