ALZHEIMER'S RESEARCH PEPTIDES

BACHEM

PIONEERING PARTNER FOR PEPTIDES

ALZHEIMER'S RESEARCH CATALOG PEPTIDES «TO REMEMBER»

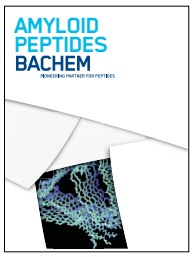
BACHEM offers more than 3600 catalog peptides for different areas of interest and research topics including over 250 Alzheimer's related products available in our inventory. With a record of enduring success and over 45 years of experience in peptide synthesis we continue to be a leader in the peptide industry.

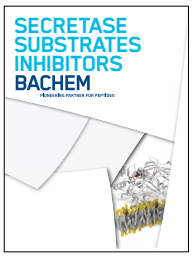
UNLABELED AMYLOID B-PEPTIDES

Different Salts and HFIP-treated Amyloid β-Peptides

For your convenience we have synthesized different salts of Amyloid β-Proteins:

H-6466	Amyloid β-Protein (1-42) HCl	**H-7432**	Amyloid β-Protein (3-42) ammonium **NEW**
H-8146	Amyloid β-Protein (1-42) TFA **NEW**	**H-4796**	(Pyr^3)-Amyloid β-Protein (3-42) ammonium
H-7404	Amyloid β-Protein (1-42) sodium salt **NEW**	**H-8248**	(Pyr^3)-Amyloid β-Protein (3-42) TFA
H-7442	Amyloid β-Protein (1-42) HFIP-treated	**H-7434**	Amyloid β-Protein (4-42) ammonium **NEW**
H-5568	Amyloid β-Protein (1-40) HCl	**H-7436**	Amyloid β-Protein (5-42) ammonium **NEW**
H-1194	Amyloid β-Protein (1-40) TFA	**H-6366**	Amyloid β-Protein (6-20) TFA
H-7438	Amyloid β-Protein (1-40) HFIP-treated	**H-6384**	Amyloid β-Protein (10-35) TFA
H-7664	Amyloid β-Protein (1-40) amide TFA **NEW**	**H-7668**	Amyloid β-Protein (11-42) TFA **NEW**
H-7458	Amyloid β-Protein (1-39) TFA	**H-3682**	Amyloid β-Protein (16-20) TFA
H-2966	Amyloid β-Protein (1-38) TFA	**H-8092**	Amyloid β-Protein (16-22) TFA **NEW**
H-7462	Amyloid β-Protein (1-37) TFA	**H-7532**	Amyloid β-Protein (17-40) TFA **NEW**
H-7865	Amyloid β-Protein (1-28) TFA	**H-3808**	Amyloid β-Protein (20-29) TFA
H-7656	Amyloid β-Protein (1-24) TFA	**H-1192**	Amyloid β-Protein (25-35) TFA
H-2958	Amyloid β-Protein (1-16) TFA	**H-4222**	Amyloid β-Protein (25-35) amide TFA
H-6368	Amyloid β-Protein (1-15) TFA	**H-7728**	Amyloid β-Protein (40-1) HCl **NEW**
H-7372	Amyloid β-Protein (1-14) TFA	**H-2972**	Amyloid β-Protein (40-1) TFA
H-2956	Amyloid β-Protein (1-11) TFA	**H-5572**	Amyloid β-Protein (33-42) TFA
H-7472	Amyloid β-Protein (2-42) TFA **NEW**	**H-2964**	Amyloid β-Protein (35-25) TFA
H-7672	Amyloid β-Protein (3-40) TFA **NEW**		





OUR PRODUCT MONOGRAPHS PROVIDE ADDITIONAL INFORMATION.

UNLABELED AMYLOID β-PEPTIDES

Fragments of Different Lengths

Cleavage of amyloid precursor protein (APP) by β- and γ-secretases yields amyloid β peptides. Aβ (1-40) and the more virulent Aβ (1-42) are the most important APP degradation products. Aβ (1-42), a 42-residue fragment of APP, has been found to be a major constituent of the senile plaques formed in the brains of patients with Alzheimer's disease (AD) and late Down's syndrome. Aβ (1-42) readily forms neurotoxic oligomers at physiological pH. The sequence of H-1368/H-6466/H-7404/H-8146 corresponds to the human, bovine, canine, feline, ovine, guinea pig, and rabbit Aβ42 peptide. The peptide has been used to detect amyloid β-protein multimers in the cerebrospinal fluid of Alzheimer's disease patients through fluorescence correlation spectroscopy.

H-6406	Amyloid β-Protein (1-46)	**H-6366**	Amyloid β-Protein (6-20)
H-1586	Amyloid β-Protein (1-43)	**H-1388**	Amyloid β-Protein (10-20)
H-1368 **H-6466** **H-8146** **H-7404**	Amyloid β-Protein (1-42) Amyloid β-Protein (1-42) hydrochloride salt Amyloid β-Protein (1-42) trifluoroacetate salt **NEW** Amyloid β-Protein (1-42) sodium salt **NEW**	**H-6384**	Amyloid β-Protein (10-35)
H-1194 **H-5568**	Amyloid β-Protein (1-40) trifluoroacetate salt Amyloid β-Protein (1-40) hydrochloride salt	**H-6382**	(Pyr^{11})-Amyloid β-Protein (11-40)
H-7458	Amyloid β-Protein (1-39)	**H-7668**	Amyloid β-Protein (11-42) **NEW**
H-2966	Amyloid β-Protein (1-38)	**H-7910**	Amyloid β-Protein (12-28)
H-7462	Amyloid β-Protein (1-37)	**H-3945**	(Leu^{16})-Amyloid β-Protein (16-19)
H-7865	Amyloid β-Protein (1-28)	**H-3682**	Amyloid β-Protein (16-20)
H-7656	Amyloid β-Protein (1-24)	**H-8092**	Amyloid β-Protein (16-22) **NEW**
H-2958	Amyloid β-Protein (1-16)	**H-7532**	Amyloid β-Protein (17-40) **NEW**
H-6368	Amyloid β-Protein (1-15)	**H-3808**	Amyloid β-Protein (20-29)
H-7372	Amyloid β-Protein (1-14)	**H-1976**	Amyloid β-Protein (22-35)
H-2956	Amyloid β-Protein (1-11)	**H-1192**	Amyloid β-Protein (25-35)
H-7472	Amyloid β-Protein (2-42) **NEW**	**H-4222**	Amyloid β-Protein (25-35) amide
H-7672	Amyloid β-Protein (3-40) **NEW**	**H-3984**	Amyloid β-Protein (29-40)
H-7422	(Pyr^{3})-Amyloid β-Protein (3-40)	**H-5866**	Amyloid β-Protein (31-35)
H-7432	Amyloid β-Protein (3-42) **NEW**	**H-5572**	Amyloid β-Protein (33-42)
H-4796 **H-8248**	(Pyr^{3})-Amyloid β-Protein (3-42) ammonium salt (Pyr^{3})-Amyloid β-Protein (3-42) trifluoroacetate salt **NEW**	**H-5270**	Amyloid β-Protein (36-38)
H-7434	Amyloid β-Protein (4-42) **NEW**	**H-3500**	Amyloid β -Protein (37-39)
H-7436	Amyloid β-Protein (5-42) **NEW**		

Unlabeled Amyloid β-Peptides (mouse, rat)

H-5966	Amyloid β-Protein (1-42) mouse, rat	**H-5638**	Amyloid β-Protein (1-40) mouse, rat

«BACHEM IS OUR NUMBER ONE CHOICE IN PEPTIDE QUALITY AND SECOND TO NONE IN CUSTOMER SERVICE AND COMMUNICATION.»

AMYLOID β-PEPTIDES

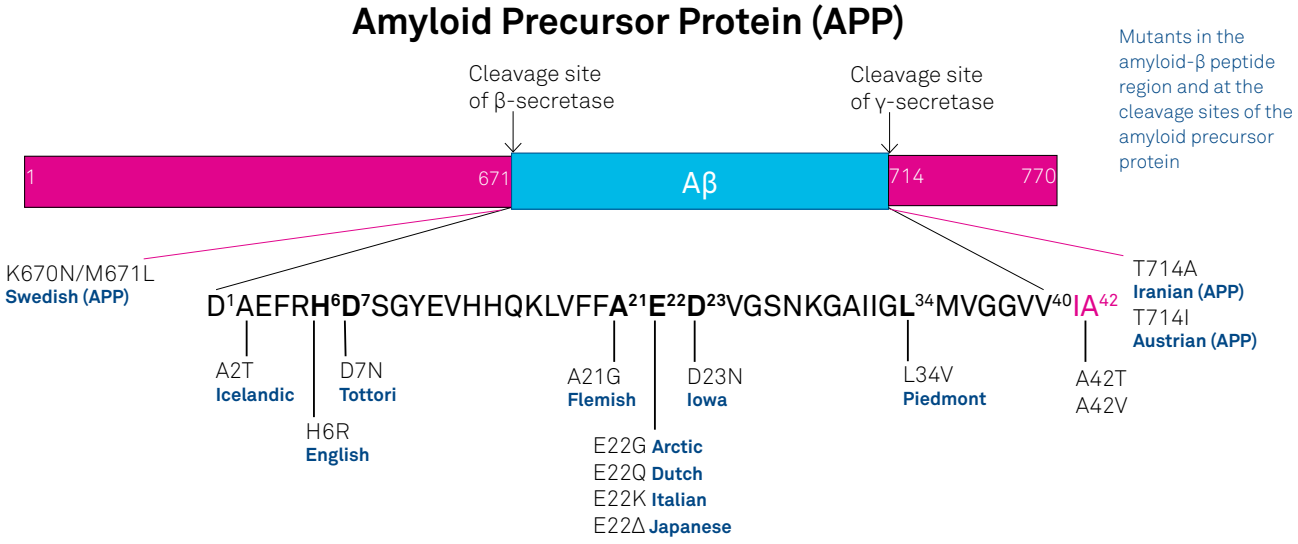
Reverse and Scrambled Sequences, all-D Version

The reverse or scrambled Aβ peptides can be used as a negative control in order to test for toxicity on your favorite cells.

H-3976	Amyloid β-Protein (42-1), reverse sequence of Aβ (1-42) (H-1386, H-6466, H-8146), inactive control for Aβ	**H-2972**	Amyloid β-Protein (40-1), reverse sequence of Aβ (1-40), an inactive control for H-1194 and H-5568
		H-7728	Amyloid β-Protein (40-1) hydrochloride salt **NEW**
H-7406	Amyloid β-Protein (1-42) (scrambled) **NEW**	**H-7408**	Amyloid β-Protein (1-40) (scrambled) **NEW**
H-5566	ent- Amyloid β-Protein (1-42), all-D Aβ (1-42)	**H-2964**	Amyloid β-Protein (35-25) , reverse sequence of Aβ 25-35 (H-1192), inactive control

AMYLOID β-PROTEIN MUTANTS

A number of mutations within the APP gene have been detected in families with an inherited risk for early onset of AD. Usually, they are named after the region, in which they have been detected. A selection of relevant mutations in the Aβ region of APP that we offer is presented below.



Catalog Number	Exchanged in APP	Position in Aβ	Designation
H-7336	H677R	H6R	English
H-7334	D678N	D7N	Tottori
H-6702	A692G	A21G	Flemish
H-6694, H-6124[1]	E693G	E22G	Arctic
H-6696	E693Q	E22Q	Dutch
H-6698	E693K	E22K	Italian
H-7474 NEW, H-7686 NEW [1]	E693Δ	E22Δ	Osaka
H-7412	E693Q/D694N	E22Q/D23N	Dutch/Iowa
H-7332	D694N	D23N	Iowa
H-7414	L705V	L34V	Piedmont
H-6432	E674R	E3R	---
H-6434	G680Q	G9Q	---
H-7662 NEW	H684R	H13R	---
H-6446[1]	F691E	F20E	---

H-7402 NEW, H-7418 NEW	S697C	S26C	---
H-8296[2] NEW	A673V	A2V	---
H-6448[1]	L688R	L17R	---
H-2362[3]	E682Q	E11Q	---
H-6386[4]	K699G/A701C	K28G/A30C	---

All Aβ (1-40) except for: [1]Aβ (1-42),[2]Aβ (1-6), [3]Aβ (1-28), [4]Aβ (1-30) amide. H-7418 is the dimer of H-7402.

The English (H6R) mutation and the Tottori (D7N) mutation of β-amyloid peptides accelerate fibrillation without increasing proto-fibril formation. Ono et al. (J. Biol. Chem. 285, 23186 (2010)) showed that the English and Tottori mutations alter Aβ assembly at its earliest stages, monomer folding and oligomerization, and produce oligomers that are more toxic to cultured neuronal cells than are wild type oligomers. The exchange of His[6] by Arg influences the structure of the Cu(II) complex formed by Aβ peptides.
Contrary to β-amyloid peptides mutated at position 22 (Dutch, Italian, Arctic mutants), the Flemish mutation (A21G) shows a decreased tendency to aggregate and a reduced neurotoxicity. In the studies of Betts and Tsubuki, A21G was degraded significantly more slowly by neprilysin than the wild-type Aβ (1-40) and the E22 mutants. The relative resistance to proteolytic degradation may account for the pathogenicity of the Aβ mutant.

The highly neurotoxic arctic mutant (E22G) of Aβ has been used to study the mechanisms underlying the formation of soluble and insoluble β-amyloid aggregates. As the wild-type Aβ, the arctic mutant preferably assembles in the presence of GM1 ganglioside.
The Dutch mutation (E22Q) of amyloid β-peptide aggregates more readily than the wild-type peptide and the resulting fibrils show increased neurotoxicity. The mutant peptide E22Q induced apoptosis of cerebral endothelial cells at a concentration of 25 µm, whereas wild-type Aβ (1-40) and the Italian mutant E22K showed no effect.
The Italian mutation of β-amyloid (1-40) (E22K) aggregates more rapidly than the wild-type sequence 1-40. It showed increased neurotoxicity, which (according to a solid-phase NMR-study of Masuda et al. (Bioorg. Med. Chem.13, 6803 (2005)) may be due to the salt bridge formed between Lys[22] and Asp[23] in the minor conformer. As the Arctic, Flemish, and Dutch mutants, the Italian mutant is degraded considerably more slowly than wild-type Aβ by neprilysin.

The Iowa (D23N) mutant of Aβ 40 assembles in solution considerably more rapidly to form fibrils than the wild-type Aβ sequence. These fibrils also show a different structure, which could be responsible for their increased toxicity.

MODIFIED AMYLOID β-PEPTIDES

H-5888	(Met(O)^{35})-Amyloid β-Protein (1-42)	**H-4984**	(Asn(4-aminobutyl)^{1,7,23},Gln(4-aminobutyl)^{3,11,22})-Amyloid β-Protein (1-40)
H-7324	(Met(O_2)^{35})-Amyloid β-Protein (1-42)	**H-2962**	(Met(O)^{35})-Amyloid β-Protein (25-35)
H-7308	(Nle^{35})-Amyloid β-Protein (1-42)	**H-7314**	(Nle^{35})-Amyloid β-Protein (25-35)
H-6388	Cys-Gly-Lys-Arg-Amyloid β-Protein (1-42)	**H-6372**	Cys-Gly-Lys-Lys-Gly-Amyloid β-Protein (33-40)
H-4854	(D-Asp^{1})-Amyloid β-Protein (1-42)	**H-6364**	Cys-Gly-His-Gly-Asn-Lys-Ser-Amyloid β-Protein (33-40)
H-7312	(Nle^{35})-Amyloid β-Protein (1-40)	**H-6378**	Cys-Gly-Lys-Lys-Gly-Amyloid β-Protein (35-40)
H-7368	(Cys^{0})-Amyloid β-Protein (1-40)		

MODIFIERS OF Aβ AGGREGATION

H-3684	Acetyl-Amyloid β-Protein (15-20) amide	**H-6074**	ent-[Amyloid β-Protein (20-16)]-β-Ala-D-Lys(ent-[Amyloid β-Protein (16-20)])
H-4062	(Lys^{15})-Amyloid β-Protein (15-21)	**H-7658**	Acetyl-(N-Me-Leu^{17},N-Me-Phe^{19})-Amyloid β-Protein (16-20) amide **NEW**
H-3904	(Arg^{15},Asp^{16,25},Pro^{18,21,23},Val^{22},Ile^{24})-Amyloid β-Protein (15-25)	**H-8092**	Amyloid β-Protein (16-22) **NEW**
H-3978	Gly-Amyloid β-Protein (15-25)-Gly-ε-aminocaproyl(-Lys)$_6$	**H-4876**	(Pro^{18},Asp^{21})-Amyloid β-Protein (17-21)
H-3682	Amyloid β-Protein (16-20)	**H-6138**	Acetyl-(Pro^{18},Asp^{21})-Amyloid β-Protein (17-21) amide

Aβ ANTAGONIST

H-4124	Propionyl-Amyloid β-Protein (31-34) amide

LABELED AMYLOID β-PEPTIDES

The following peptides can be used in Aβ-protein binding studies:

H-5642	Biotin Amyloid β-Protein (1-42)	**H-5914**	Biotin Amyloid β-Protein (1-40)
H-7454	Biotin LC-Amyloid β-Protein (1-42)	**H-7456**	Biotin LC-Amyloid β-Protein (1-40) **NEW**
H-7444	5-FAM-Amyloid β-Protein (1-42)	**H-7446**	5-FAM-Amyloid β-Protein (1-40)
M-2585	FITC β-Ala Amyloid β-Protein (1-42)	**H-6326**	FITC β-Ala Amyloid β-Protein (1-40)
H-7666	FITC LC-Amyloid β-Protein (1-42) **NEW**	**H-7452**	5-TAMRA Amyloid β-Protein (1-40)
H-7448	5-TAMRA Amyloid β-Protein (1-42)		

For your benefit and convenience, we offer the most frequently studied amyloid β-protein fragments, Aβ (1-42) (H-1368/H-6466/H-8146) and Aβ (1-40) (H-1194), in 0.1 mg, 0.5 mg, 1 mg, and 5 mg packages. Additionally, 10 mg and 25 mg packages of all three Aβ (1-42) products are available from stock. Larger quantities can be quoted upon request.

Amyloid β-protein (1-40) that is N-terminally modified with the fluorescent dye (7-diethylaminocoumarin-3-yl)carbonyl (DAC or DEAC) can be utilized to assess the binding properties of amyloid β-protein (1-40) for various membranes since it behaves very similar to the native peptide. In aqueous environments the fluorophore is almost non-fluorescent whereas binding to membranes results in an increase in fluorescence intensity (λ_{ex} = 430 nm, λ_{em} = 470 nm). Increases in the GM1 ganglioside and cholesterol content in the lipid bilayers facilitated the binding of this peptide. For phosphatidylcholine and phosphatidylserine no affinity was observed:

H-6468	(7-Diethylaminocoumarin-3-yl)carbonyl-Amyloid β-Protein (1-40)

FRET SUBSTRATES

M-2565	Abz-(Asn^{670},Leu^{671})-Amyloid β/A4 Protein Precursor$_{770}$ (669-674)-EDDnp	Intramolecularly quenched fluorescent substrate containing the ortho-aminobenzoyl (Abz) / N-(2,4-dinitrophenyl) ethylenediamine (EDDnp) groups as the donor / acceptor pair. It corresponds to the Swedish-mutated (JMV2236) β-amyloid precursor protein (βAPP) sequence targeted by β-secretase BACE (β-site APP-cleaving activity). This FRET substrate is more selectively cleaved by BACE1 and BACE2 than by cathepsin D, a disintegrin and metalloprotease 10 (ADAM10), tumor necrosis α-converting enzyme (TACE), presenilin-1 (PS1), or presenilin-2 (PS2)
M-2560	Abz-Amyloid β/A4 Protein Precursor$_{770}$ (669-674)-EDDnp	This FRET substrate is cleaved by BACE1, BACE2, and cathepsin D
M-2540	Abz-Amyloid β/A4 Protein Precursor$_{770}$ (708-715)-Lys(Dnp)-D-Arg-D-Arg-D-Arg amide	A sensitive fluorogenic (FRET) substrate developed for the analysis of γ-secretase from post mortem non-Alzheimer's and Alzheimer's disease human brain isolates
M-2470	Arg-Glu(EDANS)-(Asn^{670},Leu^{671})-Amyloid β/A4 Protein Precursor$_{770}$ (668-675)-Lys(DABCYL)-Arg	Fluorogenic (FRET) substrate for pro-memapsin-2 containing the β-secretase site EVNLDAEF of the Swedish mutation of APP. The kinetic parameters at pH 4.5 are Km = 5.4 µM and kcat = 0.24 min^{-1}
M-2435	DABCYL-(Asn^{670},Leu^{671})-Amyloid β/A4 Protein Precursor$_{770}$ (667-675)-EDANS	A FRET substrate for β-Secretase corresponding to residues 667 to 674, SEVNLDAEF, of the Swedish double mutation (K670N/M671L) of APP. The sequence can also be cleaved by cathepsin D
M-2570	Lys(Dabsyl)-(Asn^{670},Leu^{671})-Amyloid β/A4 Protein Precursor$_{770}$ (667-676)-Gln-Lucifer Yellow	A highly selective substrate for measuring BACE1 (Km = 9 µM, kcat = 0.02 s^{-1}) and BACE2 activity. In this fluorescence resonance energy transfer (FRET) substrate the fluorescent group Lucifer Yellow is efficiently quenched by Dabsyl (4-(4-Dimethylaminophenylazo)benzenesulfonyl). Enzymatic cleavage can be assayed by detecting the increase in fluorescence emission at 520 nm using an excitation wavelength of 430 nm

M-2420	Mca-(Asn^{670},Leu^{671})-Amyloid β/A4 Protein Precursor$_{770}$ (667-675)-Lys(Dnp)	**M-2460**	Mca-Amyloid β/A4 Protein Precursor$_{770}$ (667-676)-Lys(Dnp)-Arg-Arg amide
M-2485	Mca-(Asn^{670},Leu^{671})-Amyloid β/A4 Protein Precursor$_{770}$ (667-675)-Lys(Dnp) amide	**M-2555**	N-Me-Abz-Amyloid β/A4 Protein Precursor$_{770}$ (708-715)-Lys(Dnp)-D-Arg-D-Arg-D-Arg amide
M-2465	Mca-(Asn^{670},Leu^{671})-Amyloid β/A4 Protein Precursor$_{770}$ (667-676)-Lys(Dnp)-Arg-Arg amide		

AMYLOID β/A4 PROTEIN PRECURSOR770 (APP) FRAGMENTS

H-2232	Acetyl-Amyloid β/A4 Protein Precursor$_{770}$ (96-110) (cyclized)	This cyclized peptide which is homologous to the heparin-binding domain of APP, binds strongly to heparin and inhibits binding of ^{125}I-labeled APP to heparin (IC_{50}= 10^{-7}M). The peptide blocks the heparan sulfate proteoglycan-dependent stimulatory effect of APP on neurite outgrowth
H-3726	Amyloid β/A4 Protein Precursor$_{770}$ (135-155)	The APP fragment FLHQERMDVCETHLHWHTVAK comprises a copper(II)-binding site. Binding of Cu(II) to the peptide induced oxidation of the cysteine and intermolecular dimerization of the peptide. The reduction of Cu(II) to Cu(I) by APP may enhance the production of hydroxyl radicals which in turn may contribute to neurodegeneration in Alzheimer‘s disease
H-2594	Amyloid β/A4 Protein Precursor$_{770}$ (394-410)	The 17-mer peptide AKERLEAKHRERMSQVM is capable of reducing the neurologic damage *in vivo* in a model of central nervous system ischemia, possibly through its effect on synaptic plasticity
H-1608	Amyloid β/A4 Protein Precursor$_{770}$ (403-407)	The growth-promoting activity of the secreted form of amyloid β/A4 protein precursor (sAPP-695) on fibroblasts is represented by the fragment RERMS which seems to be the only site of sAPP-695 involved in the growth stimulation. It has been suggested that RERMS is also involved in the pathogenesis of Alzheimer‘s disease
N-1850	Amyloid β/A4 Protein Precursor$_{770}$ (586-595) (human, mouse, rat)	Amyloid β/A4 Protein Precursor$_{770}$ (586-595) (human, mouse, rat) also known as APP-derived inhibitory peptide (APP-IP), efficiently inhibited the activity of gelatinase A (matrix metalloproteinase-2 (MMP-2)) (IC_{50} = 30 nM), whereas its inhibitory activity towards membrane type 1-matrix metalloproteinase was much weaker (IC_{50} = 2 µM). The decapeptide ISYGNDALMP had poor inhibitory activity towards gelatinase B, matrilysin or stromelysin (IC_{50} > 10 µM). APP-IP is likely an active site-directed inhibitor of gelatinase A. Clarification of its mode of action might provide a lead for the design of novel anti-cancer drugs that block specific processes of tumor invasion and angiogenesis
H-4836	(Asn^{670},Leu^{671})-APP$_{770}$ (667-675), APP$_{770}$ (667-675) Swedish Mutation	SEVNLDAEF corresponds to the mutant junctional sequence of the amyloid precursor protein (APP) found in a Swedish family with early-onset Alzheimer‘s disease, therefore referred to as the ‚Swedish‘ mutation (K670N/M671L). The peptide has been used for assaying cleavage at leucine-aspartate by cathepsin G and chymotrypsin, whereas neither cathepsin B, D nor L generated any products
H-4842	Amyloid β/A4 Protein Precursor$_{770}$ (667-676)	The peptide substrate APP (667-676), SEVKMDAEFR, corresponds to the wild-type amyloid precursor protein (APP) β-secretase cleavage site. SEVKMDAEFR has been used for assaying β-secretase activity
H-4834	(Asn^{670},Leu^{671})-Amyloid β/A4 Protein Precursor$_{770}$ (667-676)	This peptide substrate corresponds to the ‚Swedish‘ Lys-Met/Asn-Leu (K670N/M671L) mutation of the amyloid precursor protein (APP) β-secretase cleavage site. It has been used for assaying β-secretase activity

H-4838	(Val^{671})-Amyloid β/A4 Protein Precursor$_{770}$ (667-676)	The peptide corresponds to the M671V mutation of the amyloid precursor protein (APP) β-secretase cleavage site. It has been used for assaying β-secretase activity but no detectable cleavage by the endogenous β-secretase could be observed
H-6216	Amyloid β/A4 Protein Precursor$_{770}$ (740-770)	Amyloid β/A4 Protein Precursor$_{770}$ (740-770) corresponds to a C-terminal amyloid precursor protein (APP) fragment known as C31. This fragment is intracellularly generated by proteolytic cleavage of APP by caspases-8 and -9. C31 had a proapoptotic and a cytotoxic effect on neuronal cells and was shown to be present in brains of Alzheimer's disease (AD) patients. In cultured cells caspase cleavage of APP was induced by amyloid β-protein and the subsequent generation of C31 contributed to the apoptotic cell death associated with amyloid β-protein. Amyloid precursor binding protein BP1 (APP-BP1) a cell cycle protein which is increased in AD brain was demonstrated to bind to the C31 region of APP and to mediate APP-induced apoptosis
H-7874	Amyloid Precursor Frameshift Mutant C-Terminal Peptide **NEW**	The sequence RGRTSSKELA corresponds to human APP frameshift mutant (339-348). RGRTSSKELA is used for generating antibodies.

β-SECRETASE INHIBITORS

N-1815	Ac-Val-Met-[(2S,4S,5S)-5-amino-4-hydroxy-2-isopropyl-7-methyl-octanoyl]-Ala-Glu-Phe-OH	Ac-VML-psi[CHOH-CH_2]-VAEF, a potent human β-secretase inhibitor with an IC_{50} of 20 nM containing the hydroxyethylene isostere of the Leu-Val motif
H-4848	(Asn^{670},Sta^{671},Val^{672})-Amyloid β/A4 Protein Precursor$_{770}$ (662-675)	Amyloid precursor protein (APP) β-secretase from human brain cleaves full-length APP at the amino terminus of the amyloid β-protein (Aβ) sequence, thus leading to the generation and extracellular release of β-cleaved soluble APP and a corresponding cell-associated carboxy-terminal fragment. The subsequent cleavage of the C-terminal fragment by γ-secretase(s) leads to the formation of Aβ. This new peptide represents a potent substrate analog inhibitor of APP β-secretase with IC_{50} = 30 nM
N-1825	H-Glu-Leu-Asp-[(2R,4S,5S)-5-amino-4-hydroxy-2,7-dimethyl-octanoyl]-Ala-Glu-Phe-OH	Glu-Leu-Asp-Leu-psi[CHOH-CH_2]-Ala-Ala-Glu-Phe (ELDL-psi[CHOH-CH_2]-AAEF), a potent inhibitor for human brain memapsin-2 (β-secretase) with a Ki-value of 0.3 nM. The hydroxyethylene isostere of the Leu-Ala motif functions as a mimic of the transition state

N-1920	H-Glu-Leu-Asp-[(2R,4S,5S)-5-amino-4-hydroxy-2,7-dimethyl-octanoyl]-Val-Glu-Phe-Gly-Gly-D-Arg-D-Arg-D-Arg-D-Arg-D-Arg-D-Arg-D-Arg-D-Arg-D-Arg-OH	**N-1590**	Z-Leu-Leu-4,5-dehydro-Leu-aldehyde
H-5108	OM99-2		

γ-SECRETASE INHIBITORS

H-5106	[(2R,4R,5S)-2-Benzyl-5-(Boc-amino)-4-hydroxy-6-phenyl-hexanoyl]-Leu-Phe-NH_2	**N-1895**	Z-Ile-Leu-aldehyde
N-1890	3,5-Difluorophenylacetyl-Ala-Phg-OMe	**N-1695**	Z-Leu-Leu-Nle-aldehyde

AMYLOID-LIKE PROTEIN 1 (APLP 1)

APLP 1 is a highly sensitive biomarker for the production of Aβ (1-42) derived from the βAPP-like protein APLP1. The peptide is generated by the same proteolytic mechanism as Aβ42. Non-amyloidogenic APL1β28 can be detected in the cerebrospinal fluid and its levels correlate with Aβ42 production. Its ratio to total APL1β (APL1β25, APL1β27, and APL1β28) is significantly increased in familial and sporadic cases of AD:

H-7306	APLP1-derived Aβ-like peptide (1-28)	**H-7302**	APLP1-derived Aβ-like peptide (1-25)
H-7304	APLP1-derived Aβ-like peptide (1-27)		

AMYLOID BRI PEPTIDES

Amyloid Bri Protein (1-34), encoded by the gene BRI located on chromosome 13, is deposited as amyloid fibrils causing neuronal dysfunction and dementia:

H-4728	Amyloid Bri Protein (1-34) (reduced)	**H-5526**	Amyloid Bri Protein (1-34)
H-5052	Amyloid Bri Protein (1-23)	**H-5048**	Amyloid Bri Protein Precursor$_{277}$ (89-106)

AMYLOID DAN PEPTIDES

Amyloid peptide that is associated with dementia:

H-5298	Amyloid Dan Protein (1-34) (reduced)	**H-5528**	Amyloid Dan Protein (1-34)

AMYLOID P-COMPONENT PEPTIDE

A wide variety of cells attach to the surface of polystyrene plastic dishes coated with this peptide, which corresponds to a fragment of the amyloid P component, a glycoprotein found in all types of amyloid deposits:

H-2942	Amyloid P Component (27-38) amide	**H-2944**	Tyr-Amyloid P Component (27-38) amide
H-2946	Amyloid P Component (33-38) amide *		

*this fragment retains 83 % of the cell attachment activity of the dodecapeptide fragment (27-38) (H-2942)

NON-Aβ COMPONENT

The 35 amino acid peptide NAC (α-synuclein (61-95)) has originally been isolated from the insoluble core of Alzheimer‘s disease (AD) amyloid plaque. The sequence corresponds to a fragment of a 140 amino acid precursor known as NACP or α-synuclein and was found to form amyloid fibrils via a nucleation-dependent polymerization mechanism. Accumulation of NAC aggregates in the synapse might be responsible for the neurodegeneration in AD and in the prion diseases.

H-2598	Non-Aβ Component of Alzheimer‘s Disease

HUMANINS AND COLIVELIN

H-5574	Humanin (human)	**H-6336**	Colivelin
H-5576	(Gly^{14})-Humanin (human)		

PRION PEPTIDES

H-1566	Prion Protein (106-126) (human)	**H-4206**	Prion Protein (118-135) (human)
H-4882	Prion Protein (106-126) (human) (scrambled)		

SUBSTRATES

L-1905	H-Lys-Thr-Glu-Glu-Ile-Ser-Glu-Val-Lys-Met-pNA	**I-1625**	Z-Val-Lys-Met-AMC
M-2650	Dansyl-D-Ala-Gly-4-nitro-Phe-Gly-OH		

DIPEPTIDES

G-2420	H-Ile-Phe-OH	**G-2525**	H-Leu-Ile-OH

LIPIDS

0-1270	1-O-Hexadecyl-2-O-acetyl-sn-glycero-3-phosphocholine (PAF (C_{16}))

VARIOUS RELATED PRODUCTS

G-1015	Ac-Asp-Glu-OH (NAAG)	**H-7804**	TRAF-6 Peptide **NEW**
G-1250	L-Carnosine	**H-7806**	TRAF-6 Control Peptide **NEW**
H-3456	CRF (6-33) (human, rat)	**H-2930**	H-Gly-Pro-Arg-OH
H-8230	Galanin (human)	**F-4235**	H-D-Pen-OH
H-7450	Galanin (mouse, rat)	**Q-1860**	Phenserine
G-4430	(Des-Gly)-Glutathione-monoethyl ester (reduced) (GCEE)	**N-1650**	L-trans-Epoxysuccinyl-Leu-3-methylbutyl-amide-ethyl ester (E64d)
H-8430	PACAP-38	**H-4076**	Acetyl-Calpastatin (184-210) (human)
H-3988	Presenilin-1 (331-349)-Cys (human, mouse)	**C-4275**	1,3-Bis-(Z-Leu-Leu)-diaminoacetone **NEW**
H-5512	Secretoneurin (mouse, rat)	**N-1490**	Z-Pro-Pro-aldehyde-dimethyl acetal

RECOMBINANT SOURCE

H-5594	rec Brain-Derived Neurotrophic Factor (human)

BACHEM

PIONEERING PARTNER FOR PEPTIDES

Marketing & Sales Contact

Americas

Bachem Americas, Inc.
Tel. +1 888 422 2436 (toll free in USA & Canada)
+1 310 539 4171
sales.us@bachem.com

Asia Pacific

Bachem Japan K.K.
Tel. +81 3 6661 0774
sales.jp@bachem.com

Europe, Africa, Middle East and India

Bachem AG
Tel. +41 58 595 2020
sales.ch@bachem.com

Visit our website
www.bachem.com
or shop online
shop.bachem.com

www.bachem.com **shop.bachem.com**

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