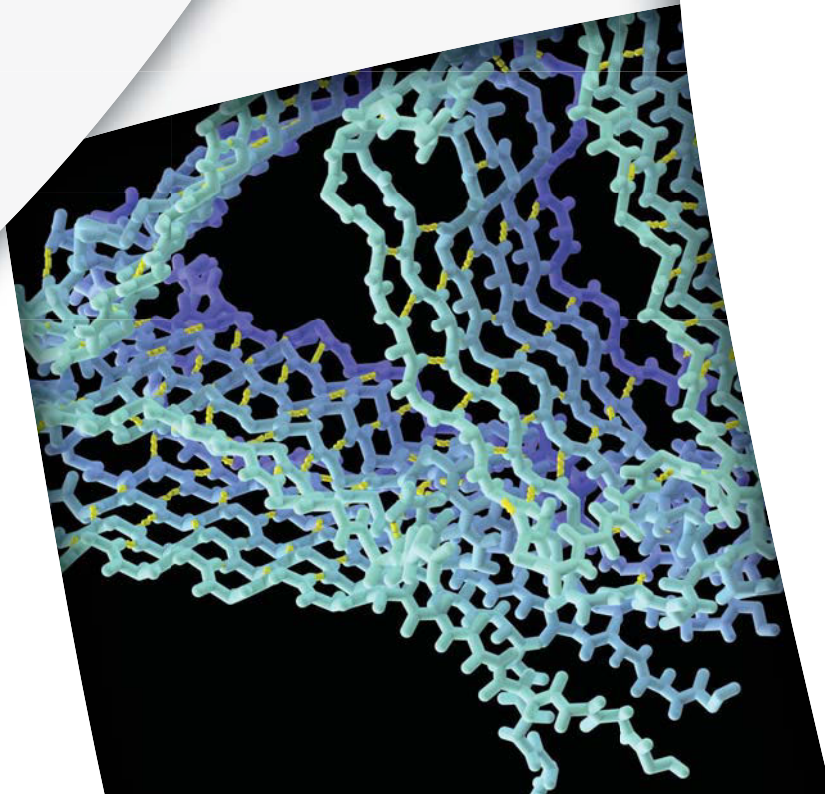


# AMYLOID PEPTIDES BACHEM

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# AMYLOID PEPTIDES OFFERED BY BACHEM

Extracellular amyloid- $\beta$  peptide deposition into cerebellar plaques and formation of intracellular neurofibrillary fibers accompanied by the loss of neurons are characteristic histopathological lesions found in the brains of Alzheimer's disease patients. Individuals suffering from this disease show a gradual loss of cognitive functions and disturbances in behavior. Apart from some rare familial forms of the disease, the onset of Alzheimer's disease is usually above 60 years. Since the risk to develop the disease increases with age, Alzheimer's disease has turned into a major health and social problem in "first world" countries with an increasing proportion of older people, and is going to become one in emerging states. In this brochure we present amyloid peptides and related products for Alzheimer's disease research.

## Alzheimer's Disease

Alzheimer's disease (AD) is the prevalent cause of dementia in elderly people and has become one of the leading causes of death in developed countries together with cardiovascular disorders, cancer, and stroke. It is estimated that more than 46 millions

of people suffer from AD all over the world. As age advances, the risk for developing AD increases. The frequency of AD at the age of 60-64 is about 1% and doubles approximately every five years. By the age of 90 and older, approximately 50% of the population suffers from this disease. AD is an irreversible and progressive neurodegenerative disorder. Symptoms include gradual loss of cognitive functions such as memory, verbal and visuospatial abilities, changes in personality, behavior, and activities of daily living. AD patients in the final stages are completely dependent on the care of others.

The characteristic lesions in the brains of AD patients were first described by the German neuropsychiatrist Alois Alzheimer in 1906 during the post-mortem examination of a mentally ill patient whose deterioration he had observed until her death. The lesions consisted of dense extracellular deposits, now designated as neuritic or senile plaques, and intracellular dense bundles of fibrils, which are now known as neurofibrillary tangles.

## AMYLOID $\beta$ -PROTEIN (1-42)

Cleavage of amyloid precursor protein (APP) by  $\beta$ - and  $\gamma$ -secretases yields amyloid  $\beta$  peptides. A $\beta$  1-40 and the more virulent A $\beta$  1-42 are the most important APP degradation products. A $\beta$ 42 is the main constituent of amyloid plaques.

Currently, diagnosis of AD with adequate testing is approximately 90% accurate. It is based on the exclusion of a variety of diseases causing similar symptoms and a careful neurological and psychiatric examination, as well as neuropsychological testing. Imaging technologies for detecting amyloid plaques and tangles *in vivo* are becoming more precise and thus a valuable additional tool. Numerous potential biomarkers as  $\alpha_1$ -antitrypsin, complement factor H,  $\alpha_2$ -macroglobulin, apolipoprotein J, and apolipoprotein A-I for diagnosing AD are being evaluated. However, post-mortem histopathological examination of the brain is still the only definite diagnosis of this disease.

AD can be either inherited or sporadic. The inherited or familial AD is rare and comprises only 5-10% of all cases. Autosomal dominant mutations in the amyloid  $\beta$ /A4 protein precursor (APP) gene on chromosome 21 and the presenilin-1 or -2 genes on chromosomes 14 and 1, respectively, have been attributed to the early onset (before the age of 65) of this disease. APP belongs to the type-1 integral membrane glycoproteins with at least 10 isoforms generated by alternative splicing of the 19 exons. The predominant transcripts are APP695, APP751, and APP770. 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, e.g. the London APP717 mutations (V717I, V717F, V717G), the Swedish APP670/671 double mutation (K670N/M671L), the Flemish APP692 mutation (A692G), or the Dutch APP693 mutation (E693Q). The Swedish mutation of the  $\beta$ -secretase cleavage site of APP and mutations of positions 692-694 (A $\beta$  21-23), which strongly influence the aggregation behavior of A $\beta$ , have been studied intensively.

A choice of relevant mutations in the A $\beta$  region of APP is assembled in the table on page 3.

The presenilins are another group of proteins involved in the development of AD. Presenilins are integral membrane proteins with eight transmembrane domains localized in the endoplasmic reticulum and the

Golgi apparatus. A multitude of mutations within the presenilin-1 and two within the presenilin-2 gene account for most of the cases of early onset of AD.

Genetic factors may contribute as well to the late onset of AD. Increased susceptibility is associated with the expression of different apolipoprotein E (ApoE) isoforms due to the polymorphism in the APOE gene on chromosome 19. In the central nervous system, ApoE has been implicated in growth and repair during development or after injury. Carriers of the APOE $\epsilon$ 4 allele show a higher risk in developing the disease than carriers of the other two possible alleles APOE $\epsilon$ 2 and APOE $\epsilon$ 3. The ApoE $\epsilon$ 4 effect seems to be dose-dependent since individuals with two of these alleles seem to be at two-fold higher risk to develop the disease than those with one allele. Polymorphisms of the  $\alpha_2$ -macroglobulin gene on chromosome 12 and the gene coding low-density lipoprotein receptor-related protein 1 (LRP-1), LRP1-C/T, have also been suggested to be a risk factor for the late onset of AD. However, further studies in this field are required.

A number of additional, most diverse risk factors have been proposed. These include gender, ethnic group, head trauma, cardiovascular diseases, and educational level.

**Autosomal dominant mutations in the amyloid  $\beta$ /A4 protein precursor (APP) gene on chromosome 21 and the presenilin-1 or -2 genes on chromosomes 14 and 1, respectively, have been attributed to the early onset (before the age of 65) of this disease.**

Exchanged Position		Designation
in APP	in A $\beta$	
A673T	A2T	Icelandic
H677R	H6R	English
D678H	D7H	Taiwanese
D678N	D7N	Tottori
A692G	A21G	Flemish
E693D	E22 $\Delta$	Osaka
E693G	E22G	Arctic
E693Q	E22Q	Dutch
E693K	E22K	Italian
D694N	D23N	Iowa
L705V	L34V	Piedmont

# AD THERAPEUTIC STRATEGIES RELY ON DETAILED KNOWLEDGE OF THE MOLECULES INVOLVED

Women, Hispanics, individuals who have experienced a head trauma earlier in life, and persons who suffer from cardiovascular diseases appear to have a higher risk of developing the disease.

The etiology of AD is still not completely understood. Initial research focused upon determining the molecular structure of the senile plaques and the neurofibrillary tangles originally described by Alois Alzheimer. The main constituents of the senile plaques were identified as cleavage products of APP, designated as amyloid  $\beta$ -peptides ( $A\beta$  peptides). Depending on the composition and the fraction of fibrillar to non-fibrillar forms of these amyloid peptides, several kinds of senile plaques can be distinguished. Three types of proteases,  $\alpha$ -secretase,  $\beta$ -secretase (or  $\beta$ -site APP-cleaving enzyme, BACE), and  $\gamma$ -secretase are involved in APP processing. APP can either be processed by the  $\alpha$ - and  $\gamma$ - or by the  $\beta$ - and  $\gamma$ -secretases. The major two amyloid peptides identified in senile plaques, amyloid  $\beta$ -protein (1-40) ( $A\beta_{40}$ ) and amyloid  $\beta$ -protein (1-42) ( $A\beta_{42}$ ), are generated by successive proteolysis of APP by  $\beta$ - and  $\gamma$ -secretases. Cleavage of APP by  $\beta$ -secretase results in the release of the extracellular N-terminal protein fragment known as soluble APP- $\beta$  molecule (sAPP- $\beta$ ). Then, the membrane-retained APP is further processed within the transmembrane domain by  $\gamma$ -secretase to yield either  $A\beta_{40}$  or  $A\beta_{42}$ . The formation of  $A\beta_{40}$  and  $A\beta_{42}$  is a normal process, and both peptides can be detected in the plasma and cerebrospinal fluid (CSF) of healthy subjects. In most studies, similar concentrations of  $A\beta_{40}$

have been measured in the CSF of both healthy controls and AD patients. On the other hand,  $A\beta_{42}$  concentrations in the CSF of AD patients are significantly lower than in normal controls, probably reflecting an increased deposition as insoluble plaques.

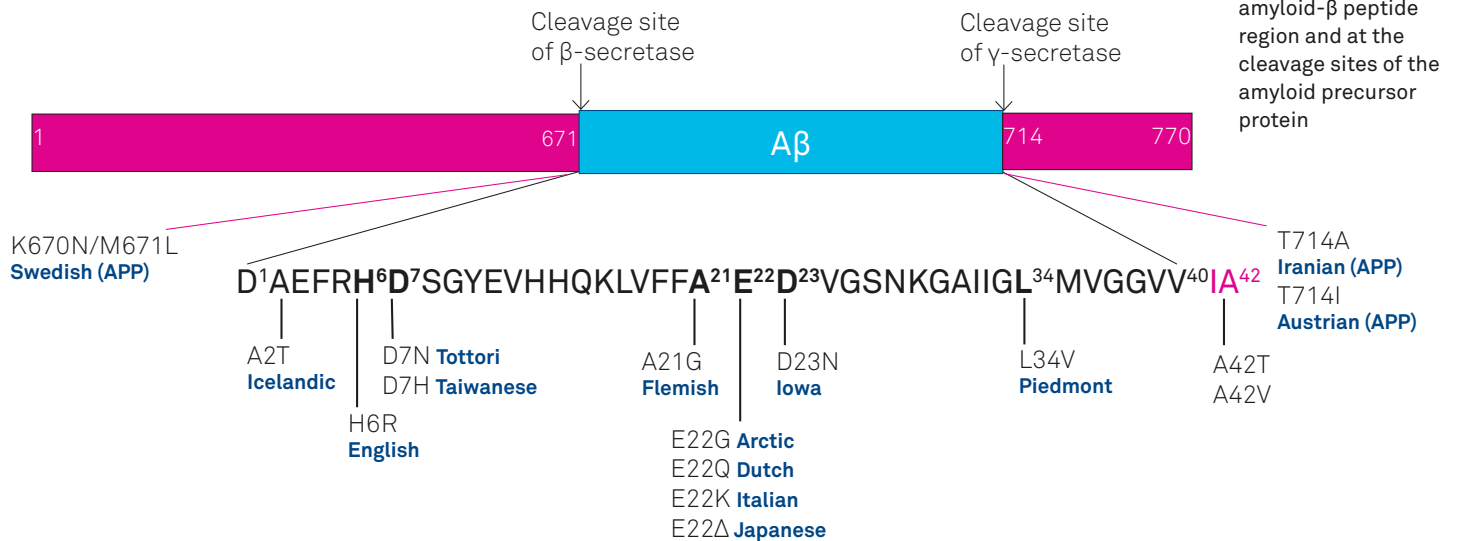
The neurofibrillary tangles found inside neurons of Alzheimer's brains are composed of paired helical filaments whose main components are hyperphosphorylated forms of tau, a microtubule associated protein involved in promoting microtubule assembly and stabilization. Self-assembly into paired helical filaments is believed to be a result of hyperphosphorylation due to either the increased activity of protein kinases or the decreased activity of phosphatases.

Several lines of evidence support the view that the accumulation of  $A\beta_{42}$  in the brain is a primary event in the development of AD. Increased cerebral  $A\beta$  production appears to be characteristic for all the mutations within the APP and the presenilin genes of familial AD. In patients with Down syndrome (trisomy 21), elevated levels of APP and  $A\beta$  due to a third copy of the APP gene result in deposition of  $A\beta$  at an early age between 20 and 30.

Formation of neurofibrillary tangles is considered as a consequence of  $A\beta$  deposition with a further impact on the progression of the disease possibly due to disruption of axonal transport mechanisms in neurons.

The detailed knowledge about the molecules involved in AD has led to the development of several therapeutic strategies.

## Amyloid Precursor Protein (APP)



One strategy aims at the reduction of A $\beta$ <sub>40</sub> and A $\beta$ <sub>42</sub> by inhibition of either  $\beta$ - or  $\gamma$ -secretase activity or by clearance of A $\beta$  in the brain by means of immunization with these peptides. Transition metals as Cu, Fe and Zn play an important role in the pathology of AD. Aggregation and neurotoxicity of A $\beta$  are dependent on the presence of copper, so Cu-chelating agents showed promising effects in animal models. Another approach is the prevention of the cellular inflammatory response in the cerebral cortex elicited by the progressive accumulation of A $\beta$ . Further preventive therapeutic strategies are based on the findings that cholesterol-lowering drugs such as statins and estrogen replacement therapy reduce the risk of developing AD. An additional treatment alternative would be the inhibition of the serine-threonine protein kinases, glycogen synthase kinase 3 (GSK3) and cyclin-dependent kinase 5 (CDK5), which are probably responsible for the phosphorylation of the tau protein. Inhibition of calpain, an enzyme showing increased activity in AD brains, led to promising results in animal studies. Calpain cleaves the CDK5 activator p35 leading to p25 formation and CDK5 overactivation.

Several acetylcholinesterase inhibitors such as tacrine, donepezil, rivastigmine, and galantamine have been approved for the treatment of mild to moderate AD by the FDA and other authorities. They act by reducing the deficits of the neurotransmitter acetylcholine associated with cognitive impairment in AD patients. The amantadine derivative memantine, an NMDA receptor antagonist, which was already used for the treatment of moderate to severe AD in Europe, has gained approval in the United States by the FDA as well.

A promising drug candidate, the  $\beta$ -secretase inhibitor verubecestat (MK-8931) developed for the management of mild to moderate AD, has moved to phase III. Moreover, the BACE inhibitor AZD3293 showed encouraging results in clinical studies. Antibodies as aducanumab and solanezumab, which have been designed to degrade plaques and lower the level of A $\beta$  in the brain, have reached advanced stages of clinical testing for mild cases of AD.

Despite the many promising therapeutic approaches, AD still remains a major burden for the patients, their relatives, and the society.

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# AMYLOID β-PROTEIN (1-42)

## Amyloid β-Protein (1-42)

**H-1368**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGVVIA

## Amyloid β-Protein (1-42) (Hydrochloride salt)

**H-6466**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGVVIA  
(Hydrochloride salt)

## Amyloid β-Protein (1-42) (Sodium salt)

**H-7404 NEW**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGVVIA  
(Sodium salt)

## Amyloid β-Protein (1-42) (Trifluoroacetate salt)

**H-8146 NEW**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGVVIA  
(Trifluoroacetate salt)

## Amyloid β-Protein (1-42) (HFIP-treated)

**H-7442**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGVVIA

## Amyloid β-Protein (1-42) (scrambled)

**H-7406**

AIAEGDSHVLKEGAYMEIFDVQGHVFG-  
GKIFRVVDLGSNVA

## Teplow's Amyloid β-Protein (1-42) (scrambled II)

**H-8282 NEW**

YHAGVDKEVVFDEGAGAEHGLAQKIVRG-  
FGVSDVSMIHINLF

## ent-Amyloid β-Protein (1-42)

**H-5566**

daefrhdsgeyevhhqklvffaedvgsnkgaiiglm-  
vggvvia  
(all-D peptide)

## Amyloid β-Protein (42-1)

**H-3976**

AIVVGGVMLGIIAGKNSGVDEAFFVLKQH-  
HVEYGS DHRFEAD

## Amyloid β-Protein (42-1) (HFIP-treated)

**H-8388 NEW**

AIVVGGVMLGIIAGKNSGVDEAFFVLKQH-  
HVEYGS DHRFEAD

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

DAEFGHDSGFVVRHQKLVFFAEDVGSNK-  
GAIIGLMVGGVVIA

## (Arg<sup>17</sup>)-Amyloid β-Protein (1-42)

**H-6448**

DAEFRHDSGYEVHHQKRVFFAEDVGSNK-  
GAIIGLMVGGVVIA

## (D-Asp<sup>1</sup>)-Amyloid β-Protein (1-42)

**H-4854**

dAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGVVIA

## (Asp<sup>37</sup>)-Amyloid β-Protein (1-42)

**H-7842 NEW**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGVVIA

## Biotinyl-Amyloid β-Protein (1-42)

**H-5642**

Biotinyl-DAEFRHDSGYEVHHQKLVF-  
FAEDVGSNKGAIIGLMVGGVVIA

## Biotinyl-εAhx-Amyloid β-Protein (1-42)

**H-7454 NEW**

Biotinyl-εAhx-DAEFRHDSGYEVHHQKLV-  
FFAEDVGSNKGAIIGLMVGGVVIA

## Cys-Gly-Lys-Arg-Amyloid β-Protein (1-42)

**H-6388**

CGKRD AEF RHDSGYEVHHQKLVFFAED-  
VGSNKGAIIGLMVGGVVIA

## (Des-Glu<sup>1</sup>)-Amyloid β-Protein (1-42)

**H-7686 NEW**

DAEFRHDSGYEVHHQKLVFFADVGSNK-  
GAIIGLMVGGVVIA  
(Osaka Mutation E22Δ)

## 5-FAM-Amyloid β-Protein (1-42)

**H-7444**

Fluorescein-5-carbonyl-DAEFRHDS-  
GYEVHHQKLVFFAEDVGFAMS NK-  
GAIIGLMVGGVVIA

## AMYLOID β-PROTEIN (1-42) (CONTINUED)

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

**H-7836 NEW**

Fluorescein-5-carbonyl-AIAEGDSHV-  
LKEGAYMEIFDVQGHVFGGKIFRVVDLG-  
SHNVA

### FITC-β-Ala-Amyloid β-Protein (1-42)

**M-2585**

FITC-β-Ala-DAEFRHDSGYEVHHQKLVFF  
AEDVGSNKGAIIGLMVGGVIA

### FITC-εAhx-Amyloid β-Protein (1-42)

**H-7666 NEW**

FITC-β-Ala-DAEFRHDSGYEVHHQKLVFF  
AEDVGSNKGAIIGLMVGGVIA

### (Gln<sup>22</sup>)-Amyloid β-Protein (1-42)

**H-7844 NEW**

DAEFRHDSGYEVHHQKLVFFAQDVGSNK-  
GAIIGLMVGGVIA  
(Dutch Mutation E22Q)

### (Glu<sup>20</sup>)-Amyloid β-Protein (1-42)

**H-6446**

DAEFRHDSGYEVHHQKLVFEAEDVGSNK-  
GAIIGLMVGGVIA

### (Gly<sup>21</sup>)-Amyloid β-Protein (1-42)

**H-7846 NEW**

DAEFRHDSGYEVHHQKLVFFGEDVGSNK-  
GAIIGLMVGGVIA  
(Flemish Mutation A21G)

### (Gly<sup>22</sup>)-Amyloid β-Protein (1-42)

**H-6124**

DAEFRHDSGYEVHHQKLVFFAGDVGSNK-  
GAIIGLMVGGVIA  
(Arctic Mutation E22G)

### (Lys<sup>22</sup>)-Amyloid β-Protein (1-42)

**H-7848 NEW**

DAEFRHDSGYEVHHQKLVFFAKDVGSNK-  
GAIIGLMVGGVIA  
(Italian Mutation E22K)

### (Met(O)<sup>35</sup>)-Amyloid β-Protein (1-42)

**H-5888**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK  
GAIIGLM(O)VGGVIA

### (Met(O<sub>2</sub>)<sup>35</sup>)-Amyloid β-Protein (1-42)

**H-7324**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK  
GAIIGLM(O<sub>2</sub>)VGGVIA

### (Nle<sup>35</sup>)-Amyloid β-Protein (1-42)

**H-7308**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGL-Nle-VGGVIA

### 5-TAMRA-Amyloid β-Protein (1-42)

**H-7448**

Fluorescein-5-carbonyl- DAEFRHDS-  
GYEVHHQKLVFFAEDVGFAMSNK-  
GAIIGLMVGGVIA

## AMYLOID β-PROTEIN (1-40)

### Amyloid β-Protein (1-40) (Trifluoroacetate salt)

**H-1194**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGV  
(Trifluoroacetate salt)

### Amyloid β-Protein (1-40) (Hydrochloride salt)

**H-5568**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGV  
(Hydrochloride salt)

### Amyloid β-Protein (1-40) (HFIP-treated)

**H-7438**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGV

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

**H-7408**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGV

# AMYLOID β-PROTEIN (1-40) (CONTINUED)

## Teplow's Amyloid β-Protein (1-40) (scrambled II)

**H-8278 NEW**

YHAGVDKEVVFDEGGAEHGLAQKIVRGF-  
GVSDVSMIHNLF

## Amyloid β-Protein (1-40) amide

**H-7664**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGVV-NH<sub>2</sub>

## Amyloid β-Protein (40-1)

(Hydrochloride salt)

**H-7728 NEW**

VVGGVMLGIIAGKNSGVDEAFFVLKQHH-  
VEYGS DHRFEAD  
(Hydrochloride salt)

## Amyloid β-Protein (40-1)

(Trifluoroacetate salt)

**H-2972**

VVGGVMLGIIAGKNSGVDEAFFVLKQHH-  
VEYGS DHRFEAD  
(Trifluoroacetate salt)

## Amyloid β-Protein (1-40) (mouse, rat)

**H-5638**

DAEFGHDSGFVVRHQKLVFFAEDVGSNK-  
GAIIGLMVGGVV

## (Arg<sup>3</sup>)-Amyloid β-Protein (1-40)

**H-6432**

DARFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGVV

## (Arg<sup>6</sup>)-Amyloid β-Protein (1-40)

**H-7336**

DAEFRDSDSGYEVRRHQKLVFFAEDVGSNK-  
GAIIGLMVGGVV  
(English Mutation H6R)

## (Arg<sup>13</sup>)-Amyloid β-Protein (1-40)

**H-7662 NEW**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGVV

## (Asn<sup>7</sup>)-Amyloid β-Protein (1-40)

**H-7334**

DAEFRHNSGYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGVV  
(Tottori Mutation D7N)

## (Asn<sup>23</sup>)-Amyloid β-Protein (1-40)

**H-7332**

DAEFRHDSGYEVHHQKLVFFAENVGSNK-  
GAIIGLMVGGVV  
(Iowa Mutation D23N)

## (Asn(4-aminobutyl)<sup>1-7-23</sup>,Gln(4-amino- butyl)<sup>3-11-22</sup>)-Amyloid β-Protein (1-40)

**H-4984**

N(4-aminobutyl)AQ(4-aminobutyl)  
FRHN(4-aminobutyl)SGYQ(4-aminobu-  
tyl)VHHQKLVFFAQ(4-aminobutyl)N(4-  
aminobutyl)VGSNKGAIIGLMVGGVV

## Biotinyl-Amyloid β-Protein (1-40)

**H-5914**

Biotinyl-DAEFRHDSGYEVHHQKLVF-  
FAEDVGSNKGAIIGLMVGGVV

## Biotinyl-εAhx-Amyloid β-Protein (1-40)

**H-7456**

Biotinyl-εAhx-DAEFRHDSGYEVHHQKLV  
FFAEDVGSNKGAIIGLMVGGVV

## (Cys<sup>0</sup>)-Amyloid β-Protein (1-40)

**H-7368**

CDAEFRHDSGYEVHHQKLVFFAEDVG-  
SNKGAIIGLMVGGVV

## (Cys<sup>26</sup>)-Amyloid β-Protein (1-40)

**H-7402**

DAEFRHDSGYEVHHQKLVFFAEDVGCNK-  
GAIIGLMVGGVV

## (Cys<sup>26</sup>)-Amyloid β-Protein (1-40)

(Dimer)

**H-7418**

(DAEFRHDSGYEVHHQKLVFFAED-  
VGCNKGAIIGLMVGGVV)<sub>2</sub>

## (Des-Glu<sup>1</sup>)-Amyloid β-Protein (1-40)

**H-7474**

DAEFRHDSGYEVHHQKLVFFADVGSNK-  
GAIIGLMVGGVV  
(Osaka Mutation E22Δ)

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

**H-6468**

Deac-DAEFRHDSGYEVHHQKLVFFAED-  
VGSNKGAIIGLMVGGVV

## AMYLOID β-PROTEIN (1-40) (CONTINUED)

### 5-FAM-Amyloid β-Protein (1-40)

**H-7446**

Fluorescein-5-carbonyl-DAEFRHDS-  
GYEVHHQKLVFFAEDVGFAMSNK-  
GAIIGLMVGGVV

### FITC-β-Ala-Amyloid β-Protein (1-40)

**H-6326**

FITC-β-Ala-DAEFRHDSGYEVHHQKLVFF  
AEDVGSNKGAIIGLMVGGVV

### (Gln<sup>9</sup>)-Amyloid β-Protein (1-40)

**H-6434**

DAEFRHDSQYEVHHQKLVFFAEDVGSNK-  
GAIIGLMVGGVV

### (Gln<sup>22</sup>)-Amyloid β-Protein (1-40)

**H-6696**

DAEFRHDSGYEVHHQKLVFFAQDVGSNK-  
GAIIGLMVGGVV  
(Dutch Mutation E22Q)

### (Gln<sup>22</sup>,Asn<sup>23</sup>)-Amyloid β-Protein (1-40)

**H-7412**

DAEFRHDSGYEVHHQKLVFFAQNVGSNK-  
GAIIGLMVGGVV  
(Dutch/Iowa Mutation E22Q/D23N)

### (Gly<sup>21</sup>)-Amyloid β-Protein (1-40)

**H-6702**

DAEFRHDSGYEVHHQKLVFFGEDVGSNK-  
GAIIGLMVGGVV  
(Flemish Mutation A21G)

### (Gly<sup>22</sup>)-Amyloid β-Protein (1-40)

**H-6694**

DAEFRHDSGYEVHHQKLVFFAGDVGSNK-  
GAIIGLMVGGVV  
(Arctic Mutation E22G)

### (Lys<sup>22</sup>)-Amyloid β-Protein (1-40)

**H-6698**

DAEFRHDSGYEVHHQKLVFFAKDVGSNK-  
GAIIGLMVGGVV  
(Italian Mutation E22K)

### (Met(O)<sup>35</sup>)-Amyloid β-Protein (1-40)

**H-7476**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK  
GAIIGLM(O)VGGVV

### (Nle<sup>35</sup>)-Amyloid β-Protein (1-40)

**H-7312**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGL-Nle-VGGVV

### 5-TAMRA-Amyloid β-Protein (1-40)

**H-7452**

5-TAMRA-DAEFRHDSGYEVHHQKLVF-  
FAEDVGSNKGAIIGLMVGGVV

### Tide Fluor™ 5WS-Amyloid β-Protein (1-40)

**H-8202 NEW**

Tide Fluor™ 5WS-DAEFRHDSGYEVH-  
HQKLVFFAEDVGSNKGAIIGLMVGGVV

### Tide Fluor™ 7WS-Amyloid β-Protein (1-40)

**H-8206 NEW**

Tide Fluor™ 7WS-DAEFRHDSGYEVH-  
HQKLVFFAEDVGSNKGAIIGLMVGGVV

### (Val<sup>34</sup>)-Amyloid β-Protein (1-40)

**H-7414**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-  
GAIIGVMVGGVV  
(Piedmont Mutation L34V)

# AMYLOID β-PROTEIN (25-35)

**Amyloid β-Protein (25-35)**

**H-1192**

GSNKGAIIGLM

**Amyloid β-Protein (35-25)**

**H-2964**

MLGIIAGKNSG

**Amyloid β-Protein (25-35) amide**

**H-4222**

GSNKGAIIGLM-NH<sub>2</sub>

**(Met(O)<sup>35</sup>)-Amyloid β-Protein (25-35)**

**H-2962**

GSNKGAIIGLM(O)

**(Nle<sup>35</sup>)-Amyloid β-Protein (25-35)**

**H-7314**

GSNKGAIIGL-Nle

# AMYLOID β-PROTEIN FRAGMENTS

**Amyloid β-Protein (1-6)**

**H-8362 NEW**

DAEFRH

**(Val<sup>2</sup>)-Amyloid β-Protein (1-6)**

**H-8296 NEW**

DVEFRH

**Amyloid β-Protein (1-6) amide**

**H-8366 NEW**

DAEFRH-NH<sub>2</sub>

**Acetyl-Amyloid β-Protein (1-6) amide**

**H-8368 NEW**

Ac-DAEFRH-NH<sub>2</sub>

**Amyloid β-Protein (1-11)**

**H-2956**

DAEFRHDSGYE

**Amyloid β-Protein (1-12)**

**H-8358 NEW**

DAEFRHDSGYEV

**Amyloid β-Protein (1-14)**

**H-7372**

DAEFRHDSGYEVHH

**Amyloid β-Protein (1-15)**

**H-6368**

DAEFRHDSGYEVHHQ

**Amyloid β-Protein (1-16)**

**H-2958**

DAEFRHDSGYEVHHQK

**Amyloid β-Protein (1-24)**

**H-7656 NEW**

DAEFRHDSGYEVHHQKLVFFAEDV

**Amyloid β-Protein (1-28)**

**H-7865**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK

**(Gln<sup>11</sup>)-Amyloid β-Protein (1-28)**

**H-2362**

DAEFRHDSGYQVHHQKLVFFAEDVGSNK

**(Gly<sup>28</sup>, Cys<sup>30</sup>)-Amyloid β-Protein (1-30) amide**

**H-6386**

DAEFRHDSGYEVHHQKLVFFAEDVGSNG-GC-NH<sub>2</sub>

**Amyloid β-Protein (1-37)**

**H-7462**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVG

**Amyloid β-Protein (1-38)**

**H-2966**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGG

**Amyloid β-Protein (1-39)**

**H-7458**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGV

**Amyloid β-Protein (1-43)**

**H-1586**

DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVVIAT

## AMYLOID β-PROTEIN FRAGMENTS (CONTINUED)

### Amyloid β-Protein (1-46)

**H-6406**

DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVVIV

### Amyloid β-Protein (2-42)

**H-7472 NEW**

AEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVVIA

### Amyloid β-Protein (3-40)

**H-7672 NEW**

EFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV

### (Pyr<sup>3</sup>)-Amyloid β-Protein (3-40)

**H-7422**

<EFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV

### Amyloid β-Protein (3-42)

**H-7432 NEW**

EFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVVIA

### (Pyr<sup>3</sup>)-Amyloid β-Protein (3-42)

(Ammonium salt)

**H-4796**

<EFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVVIA  
(Ammonium salt)

### (Pyr<sup>3</sup>)-Amyloid β-Protein (3-42)

(Trifluoroacetate salt)

**H-8248 NEW**

<EFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVVIA  
(Trifluoroacetate salt)

### Amyloid β-Protein (4-42)

**H-7434 NEW**

FRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVVIA

### Amyloid β-Protein (5-42)

**H-7436 NEW**

RHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVVIA

### Amyloid β-Protein (6-20)

**H-6366**

HDSGYEVHHQKLVFF

### Amyloid β-Protein (10-20)

**H-1388**

YEVHHQKLVFF

### Amyloid β-Protein (10-35)

**H-6384**

YEVHHQKLVFFAEDVGSNKGAIIGLM

### (Pyr<sup>11</sup>)-Amyloid β-Protein (11-40)

**H-6382**

<EVHHQKLVFFAEDVGSNKGAIIGLMVGGVV

### Amyloid β-Protein (11-42)

**H-7668 NEW**

EVHHQKLVFFAEDVGSNKGAIIGLMVGGVVIA

### Amyloid β-Protein (12-28)

**H-7910**

VHHQKLVFFAEDVGSNK

### Acetyl-Amyloid β-Protein (15-20)

amide

**H-3684**

Ac-QKLVFF-NH<sub>2</sub>

### (Lys<sup>15</sup>)-Amyloid β-Protein (15-21)

**H-4062**

KKLVFFA

### Arg<sup>15</sup>, Asp<sup>16-25</sup>, Pro<sup>18-21-23</sup>, Val<sup>22</sup>, Ile<sup>24</sup>-

### Amyloid β-Protein (15-25)

**H-3904**

RDLPPFPVPID

### Gly-Amyloid β-Protein (15-25)-Gly-ε-aminocaproyl(-Lys)<sub>6</sub>

**H-3978**

GQKLVFFAEDVGG-εAhx-KKKKKK

### (Leu<sup>16</sup>)-Amyloid β-Protein (16-19)

**H-3945**

LLVF

### Amyloid β-Protein (16-20)

**H-3682**

KLVFF

### ent-[Amyloid β-Protein (20-16)]-β-Ala-D-Lys(ent-[Amyloid β-Protein (16-20)])

**H-6074**

ffvIk-β-Ala-k(ffvIk)

# AMYLOID β-PROTEIN FRAGMENTS (CONTINUED)

**Acetyl-(N-Me-Leu<sup>17</sup>,N-Me-Phe<sup>19</sup>)-  
Amyloid β-Protein (16-20) amide**

**H-7658 NEW**

Ac-K(Me)LV(Me)FF-NH<sub>2</sub>

**Amyloid β-Protein (16-22)**

**H-8092 NEW**

KLVFFAE

**(Pro<sup>18</sup>,Asp<sup>21</sup>)-Amyloid β-Protein  
(17-21)**

**H-4876**

LPFFD

**Acetyl-(Pro<sup>18</sup>,Asp<sup>21</sup>)-Amyloid β-Protein  
(17-21) amide**

**H-6138**

Ac-LPFFD-NH<sub>2</sub>

**Amyloid β-Protein (17-40)**

**H-7532**

LVFFAEDVGSNKGAIIGLMVGGVV

**Amyloid β-Protein (20-29)**

**H-3808**

FAEDVGSNKG

**Amyloid β-Protein (22-35)**

**H-1976**

EDVGSNKGAIIGLM

**Amyloid β-Protein (29-40)**

**H-3984**

GAIIGLMVGGVV

**Propionyl-Amyloid β-Protein (31-34)  
amide**

**H-4124**

Propionyl-IIGL-NH<sub>2</sub>

**Amyloid β-Protein (31-35)**

**H-5866**

IIGLM

**Cys-Gly-His-Gly-Asn-Lys-Ser-  
Amyloid β-Protein (33-40)**

**H-6364**

CGHGNKSGLMVGGVV

**Cys-Gly-Lys-Lys-Gly-Amyloid  
β-Protein (33-40)**

**H-6372**

CGKKGGLMVGGVV

**Amyloid β-Protein (33-42)**

**H-5572**

GLMVGGVVIA

**Cys-Gly-Lys-Lys-Gly-Amyloid  
β-Protein (35-40)**

**H-6378**

CGKKGMVGGVV

**Amyloid β-Protein (36-38)**

**H-5270**

VGG

**Amyloid β-Protein (37-39)**

**H-3500**

GGV

**Methoxysuccinyl-Val-Val-Ile-Ala-pNA**

(Methoxysuccinyl-Amyloid β-Protein  
(39-42)-p-nitroanilide)

**L-1745**

MeOSuc-VVIA-pNA



# AMYLOID $\beta$ /A4 PROTEIN PRECURSOR (APP) FRAGMENTS

**Acetyl-Amyloid  $\beta$ /A4 Protein  
Precursor<sub>770</sub> (96-110) (cyclized)**  
**H-2232**

Ac-NWCKRGRKQCKTHPH-NH<sub>2</sub>  
(Disulfide bond)

**Amyloid  $\beta$ /A4 Protein  
Precursor<sub>770</sub> (135-155)**  
**H-3726**

FLHQERMDVCETHLHWHTVAK

**Amyloid  $\beta$ /A4 Protein  
Precursor<sub>770</sub> (394-410)**  
**H-2594**

AKERLEAKHRERMSQVM

**Amyloid  $\beta$ /A4 Protein  
Precursor<sub>770</sub> (403-407)**  
**H-1608**

RERMS

**Amyloid  $\beta$ /A4 Protein  
Precursor<sub>770</sub> (586-595)  
(human, mouse, rat)**  
**N-1850**

ISYGN DALMP

**(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid  $\beta$ /A4  
Protein Precursor<sub>770</sub> (667-675)**  
**H-4836**

SEVNLDAEF

(Swedish Double Mutation  
K670N / M671L)

**Amyloid  $\beta$ /A4 Protein  
Precursor<sub>770</sub> (667-676)**  
**H-4842**

SEVKMDAEFR

**(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid  $\beta$ /A4  
Protein Precursor<sub>770</sub> (667-676)**  
**H-4834**

SEVNLDAEFR

(Swedish Double Mutation  
K670N / M671L)

**(Val<sup>671</sup>)-Amyloid  $\beta$ /A4 Protein  
Precursor<sub>770</sub> (667-676)**  
**H-4838**

SEVKVDAEFR

**Amyloid  $\beta$ /A4 Protein  
Precursor<sub>770</sub> (740-770)**  
**H-6216**

AAVTP EERHLSKMQQNGY-  
ENPTYKFFEQMQN

**Amyloid Precursor Frameshift  
Mutant C-Terminal Peptide**  
**H-7674 NEW**  
RGR TSSKELA

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# AMYLOID-LIKE PROTEIN

**APL1 $\beta$ 25**  
**H-7302**

DELAPAGTGVSREAVSGLLIMGAGG

**APL1 $\beta$ 27**  
**H-7304**

DELAPAGTGVSREAVSGLLIMGAGGGS

**APL1 $\beta$ 28**  
**H-7306**

DELAPAGTGVSREAVSGLLIMGAGGG-  
SL

## AMYLOID BRI PEPTIDES

### Amyloid Bri Protein (1-23)

**H-5052**

EASNCFAIRHFENKFAVETLICS  
(Disulfide bond)

### Amyloid Bri Protein (1-34)

**H-5526**

<EASNCFAIRHFENKFAVETLICSRT-  
VKKNIEEN  
(Disulfide bond)

### Amyloid Bri Protein (1-34) (reduced)

**H-4728**

<EASNCFAIRHFENKFAVETLICSRT-  
VKKNIEEN

### Amyloid Bri Protein Precursor<sub>277</sub> (89-106)

**H-5048**

CGIKYIKDDVILNEPSAD

## AMYLOID DAN PEPTIDES

### Amyloid Dan Protein (1-34)

**H-5528**

<EASNCFAIRHFENKFAVETLICFNL-  
FLNSQEKHY  
(Disulfide bond)

### Amyloid Dan Protein (1-34) (reduced)

**H-5298**

<EASNCFAIRHFENKFAVETLICFNL-  
FLNSQEKHY

## AMYLOID P-COMPONENT PEPTIDES

### Amyloid P Component (27-38) amide

**H-2942**

EKPLQNFTLCFR-NH<sub>2</sub>

### Amyloid P Component (33-38) amide

**H-2946**

FTLCFR-NH<sub>2</sub>

### Tyr-Amyloid P Component (27-38) amide

**H-2944**

YEKPLQNFTLCFR-NH<sub>2</sub>

## NON-A $\beta$ COMPONENT ( $\alpha$ -SYNUCLEIN)

### $\alpha$ -Synuclein (34-45) (human)

**H-8382 NEW**

KEGVLYVGSKTK

### $\alpha$ -Synuclein (67-78) (human)

**H-8384 NEW**

GGAVVTGVTAVA

### $\alpha$ -Synuclein (45-54) (human)

**H-8376 NEW**

KEGVVHGVAT

### $\alpha$ -Synuclein (71-82) (human)

**H-8378 NEW**

VTGVTAVAQKTV

### $\alpha$ -Synuclein (61-95) (human)

(Non- $\beta$ -Amyloid Component of  
Alzheimer's Disease, NAC)

**H-2598**

EQVTNVGGAVVTGVTAVAQKTVEGAG-  
SIAAATGFV

### $\alpha$ -Synuclein Binding Peptide

**H-8374 NEW**

Ac-KDGIVNGVKA-NH<sub>2</sub>

# RELATED AD PRODUCTS

Tau protein fragments, inhibitors and substrates for  $\beta$ - and  $\gamma$ -secretase, and further peptides and biochemicals for Alzheimer's research are available on our online shop at [shop.bachem.com](http://shop.bachem.com):

- ↳ Areas of Interest
  - ↳ Alzheimer's Disease
  - ↳ Tau Peptides

## $\beta$ -SECRETASE SUBSTRATES

**DABCYL-(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid  $\beta$ /A4 Protein Precursor<sub>770</sub> (667-675)-EDANS**

**M-2435**

DABCYL-SEVNLDAEF-EDANS

**Mca-(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid  $\beta$ /A4 Protein Precursor<sub>770</sub> (667-675)-Lys(Dnp)**

**M-2420**

Mca-SEVNLDAEFK(Dnp)

**Mca-(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid  $\beta$ /A4 Protein Precursor<sub>770</sub> (667-675)-Lys(Dnp) amide**

**M-2485**

Mca-SEVNLDAEFK(Dnp)-NH<sub>2</sub>

**Lys(Dabsyl)-(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid  $\beta$ /A4 Protein Precursor<sub>770</sub> (667-676)-Gln-Lucifer Yellow**

**M-2570**

K(Dabsyl)SEVNLDAEFRQ-Lucifer Yellow

**Mca-Amyloid  $\beta$ /A4 Protein Precursor<sub>770</sub> (667-676)-Lys(Dnp)-Arg-Arg amide**

**M-2460**

Mca-SEVKMDAEFRK(Dnp)RR-NH<sub>2</sub>

**Mca-(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid  $\beta$ /A4 Protein Precursor<sub>770</sub> (667-676)-Lys(Dnp)-Arg-Arg amide**

**M-2465**

Mca-SEVNLDAEFRK(Dnp)RR-NH<sub>2</sub>

**Arg-Glu(EDANS)-(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid  $\beta$ /A4 Protein Precursor<sub>770</sub> (668-675)-Lys(DABCYL)-Arg**

**M-2470**

RE(EDANS)VNLDAEFK(DABCYL)R

**Abz-Amyloid  $\beta$ /A4 Protein Precursor<sub>770</sub> (669-674)-EDDnp**

**M-2560**

Abz-VKMDAE-EDDnp

**Abz-(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid  $\beta$ /A4 Protein Precursor<sub>770</sub> (669-674)-EDDnp**

**M-2565**

Abz-VNLDAE-EDDnp

**Z-Val-Lys-Met-AMC I-1625**

Z-VKM-AMC

## β-SECRETASE INHIBITORS

Ac-Val-Met-[(2S,4S,5S)-5-amino-4-hydroxy-2-isopropyl-7-methyl-octanoyl]-Ala-Glu-Phe-OH

**N-1815**

Ac-VML-psi[CHOHCH<sub>2</sub>]VAEF

(Asn<sup>670</sup>,Sta<sup>671</sup>,Val<sup>672</sup>)-Amyloid β/A4 Protein Precursor<sub>770</sub> (662-675)

**H-4848**

KTEEISEVN-Sta-VAEF

H-Glu-Leu-Asp-[(2R,4S,5S)-5-amino-4-hydroxy-2,7-dimethyl-octanoyl]-Ala-Glu-Phe-OH

**N-1825**

ELDL-psi[CHOHCH<sub>2</sub>]AAEF

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-OH

**N-1920**

ELDL-psi[CHOHCH<sub>2</sub>]AVEFGGrrrrrrrr

**OM99-2**

**H-5108**

EVNL-psi[CHOHCH<sub>2</sub>]AAEF

Z-Leu-Leu-4,5-dehydro-Leu-aldehyde

**N-1590**

Z-LLΔL-CHO

## γ-SECRETASE SUBSTRATES

Abz-Amyloid β/A4 Protein Precursor<sub>770</sub> (708-715)-Lys(Dnp)-D-Arg-D-Arg-D-Arg amide

**M-2540**

Abz-GGVVIATVK(Dnp)rrr-NH<sub>2</sub>

N-Me-Abz-Amyloid β/A4 Protein Precursor<sub>770</sub> (708-715)-Lys(Dnp)-D-Arg-D-Arg-D-Arg amide

**M-2555**

N-Me-Abz-GGVVIATVK(Dnp)rrr-NH<sub>2</sub>

## γ-SECRETASE INHIBITORS

**L-685,458**

**H-5106**

Boc-F-psi[CHOHCH<sub>2</sub>]FLF-NH<sub>2</sub>

3,5-Difluorophenylacetyl-Ala-Phg-OMe

**N-1890**

Z-Ile-Leu-aldehyde

**N-1895**

Z-IL-CHO

Z-Leu-Leu-Nle-aldehyde

**N-1695**

Z-LL-Nle-CHO

## HUMANIN

**Colivelin**

**H-6336**

SALLRSIPAPAGASRLLLLTGEIDL P

**Humanin (human)**

**H-5574**

MAPRGFSCLLLLTSEIDL PVKRR A

**(Gly<sup>14</sup>)-Humanin (human)**

(S14G-Humanin (human))

**H-5576**

MAPRGFSCLLLLTGEIDL PVKRR A

## PRION PEPTIDES

**Prion Protein (106-126) (human)**

**H-1566**

KTNMKHMAGAAAAGAVV GGLG

**Prion Protein (106-126) (human) (scrambled))**

**H-4882**

NGAKALMGGHGATKVMV GAAA

**Prion Protein (118-135) (human)**

**H-4206**

AGAVV GGLGGYMLGSAMS

## FURTHER PEPTIDES FOR ALZHEIMER RESEARCH

**Ac-Asp-Glu-OH**

(NAAG)

**G-1015**

Ac-DE

**rec Brain-Derived Neurotrophic Factor (human)**

(rec BDNF (human))

**H-5594**

**L-Carnosine**

**G-1250**

**CRF (6-33) (human, rat)**

**H-3456**

ISLDLTFHLLREVLEMARAEQLA QQA-HS

**Galanin (human) (Acetate salt)**

**H-7762 NEW**

GWTLNSAGYLLGPHAVGNHRSFSD-KNGLTS (Acetate salt)

**Galanin (human) (Trifluoroacetate salt)**

**H-8230**

GWTLNSAGYLLGPHAVGNHRSFSD-KNGLTS (Trifluoroacetate salt)

**Galanin (mouse, rat)**

**H-7450**

GWTLNSAGYLLGPHAIDNHRSFSD-KHGLT-NH<sub>2</sub>

**Galanin (porcine)**

**H-1365**

GWTLNSAGYLLGPHAIDNHRSFHD-KYGLA-NH<sub>2</sub>

**Galanin (1-13)-Pro-Pro-(Ala-Leu)<sub>2</sub>Ala amide )**

(M40)

**H-2576**

GWTLNSAGYLLGPPPALALA-NH<sub>2</sub>

**(Des-Gly)-Glutathione-monoethyl ester (reduced)**

(GCEE, γ-GCE)

**G-4430**

E(C-OEt)

**H-Gly-Pro-Arg-OH**

**H-2930**

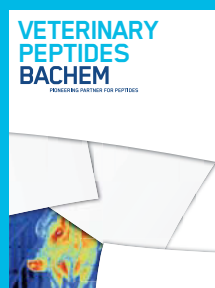
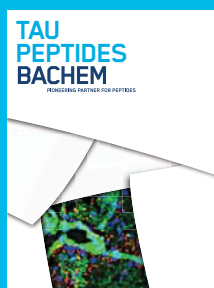
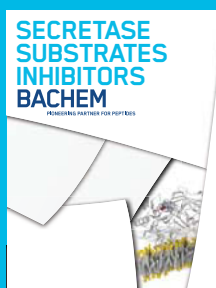
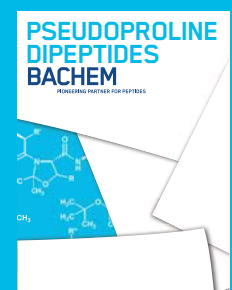
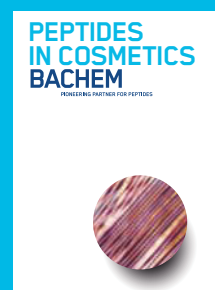
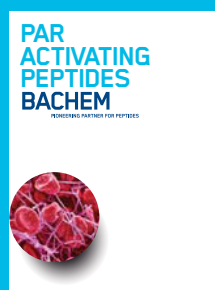
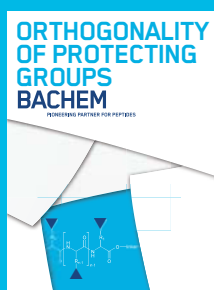
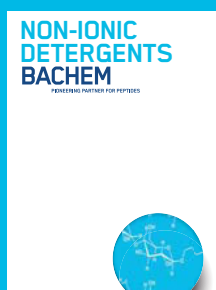
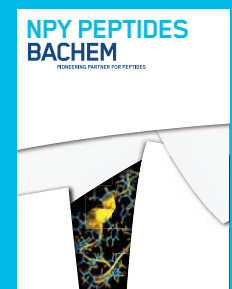
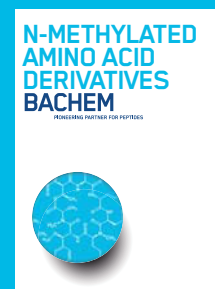
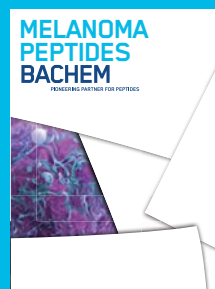
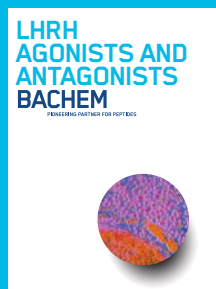
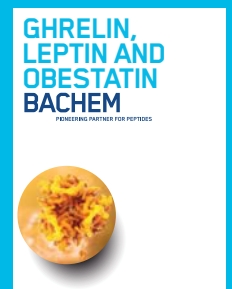
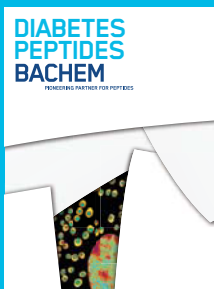
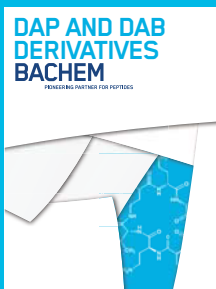
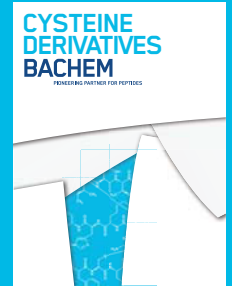
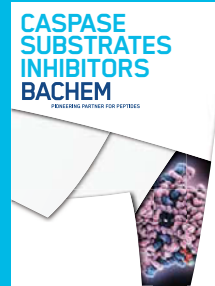
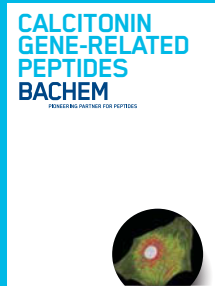
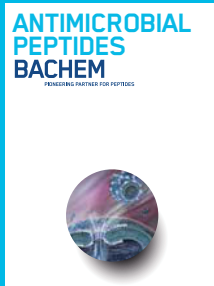
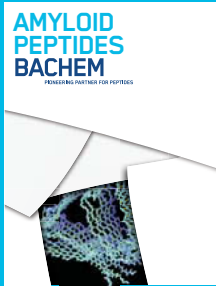
GPR

**H-Ile-Phe-OH**

**G-2420**

IF

# PRODUCT BROCHURES



# FURTHER PEPTIDES FOR ALZHEIMER RESEARCH (CONTINUED)

**rec Leptin (human)**

**H-5578**

**rec Leptin (mouse)**

**H-5582**

**Leptin (116-130) amide (mouse)  
(Acetate salt)**

**H-8244 NEW**

SCSLPQTSGLQKPES-NH<sub>2</sub>  
(Acetate salt)

**Leptin (116-130) amide (mouse)  
(Trifluoroacetate salt)**

**H-3966**

SCSLPQTSGLQKPES-NH<sub>2</sub>  
(Trifluoroacetate salt)

**H-Leu-Ile-OH**

**G-2525**

LI

**PACAP-38 (human, mouse,  
ovine, porcine, rat)**

**H-8430**

HSDGIFTDSYSRYRKQMAVKKYLA AV-  
LGKRYKQRVKNK-NH<sub>2</sub>

**Presenilin-1 (331-349)-Cys  
(human, mouse)**

**H-3988**

NDDGGFSEWEAQRD SHLGC

**Secretoneurin (mouse, rat)**

**H-5512**

TNEIVEEQYTPQSLATLESVFQELG-  
KLTGPSNQ

**TRAF6 Peptide**

**H-7604 NEW**

AAVALLPAVLLALLAPESAS-  
GPSEDPSVNFLK

**TRAF6 Control Peptide**

**H-7606 NEW**

AAVALLPAVLLALLAPESASGASA-  
DASVNFLK

**WRW4**

**H-7596 NEW**

WRWWWW-NH<sub>2</sub>

**Abz-Gly-Ala-Lys(Ac)-Ala-Ala-  
Dap(Dnp)-NH<sub>2</sub>**

**M-2700**

Abz-GAK(Ac)AA-Dpa-NH<sub>2</sub>

**Dansyl-D-Ala-Gly-4-nitro-Phe-  
Gly-OH**

**M-2650**

Dns-aGF(NO<sub>2</sub>)G

**H-Glu(EDANS)-Pro-Leu-Phe-Ala-  
Glu-Arg-Lys(DABCYL)-OH**

**M-2655**

E(EDANS)PLFAERK(DABCYL)

**Acetyl-Calpastatin (184-210)  
(human)**

**H-4076**

Ac-DPMSSTYIEELGKREVTIP-  
PKYRELLA-NH<sub>2</sub>

**1,3-Bis-(Z-Leu-Leu)-  
diaminoacetone**

((Z-LL)<sub>2</sub> Ketone)

**C-4275 NEW**

(Z-LL-CH<sub>2</sub>)<sub>2</sub>CO

**Z-Pro-Pro-aldehyde-  
dimethyl acetal**

**N-1490**

Z-PP-CH(OMe)<sub>2</sub>



## BIOCHEMICALS FOR ALZHEIMER RESEARCH

**Ac-DL-Asp-OH**

**F-4070**

**N-Me-D-Asp-OH**

(NMDA)

**F-2415**

**Ac-Cys-OH**

(NAC)

**E-3710**

**H-D-Pen-OH**

(D-Penicillamine)

**F-4235**

**H-Ser(PO<sub>3</sub>H<sub>2</sub>)-OH**

(L-Phosphoserine, Dexfosfoserine)

**F-2030**

**D-Cycloserine**

**F-1480**

**L-trans-Epoxy succinyl-Leu-  
3-methylbutylamide-ethyl ester**

(E-64d, Aloxiastatin, Loxistatin, EP453)

**N-1650**

**sn-Glycero-3-phosphocholine**

(Choline alfoscerate, L- $\alpha$ -GPC,

L- $\alpha$ -Lecithin)

**O-1590**

**1-O-Hexadecyl-2-O-acetyl-sn-  
glycero-3-phosphocholine**

(PAF (C<sub>16</sub>))

**O-1270**

**Melatonin**

**Q-1300**

**Phenserine**

**Q-1860**

A detailed molecular model of human brain-derived beta-amyloid fibrils. The structure is a complex, interconnected network of light blue and purple polypeptide chains, with yellow highlights indicating specific interactions or structural features. The model is set against a dark background, emphasizing its intricate, fibrous nature.

## ALZHEIMER'S BETA-AMYLOID FIBRILS

Molecular model of human brain-derived beta-amyloid fibrils isolated from a patient with Alzheimer's disease. The fibrils are made up of  $\beta$ -amyloid peptides. These insoluble fibres resist degradation and so build-up in brain tissue, forming the amyloid plaques found in the brains of Alzheimer's disease patients.

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ALFRED PASIEKA

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• <b>Quality</b>	GMP and non-GMP quality State of the art analytical capabilities
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• <b>Capacity</b>	Largest production facilities in the market (Europe and the USA) Up-to-date technology Short to complex peptides from mg to multi-kg and beyond
• <b>Modifications</b>	Acylation, acetylation, amidation, etc. Cyclizations Stabilizing modifications
• <b>Support</b>	Highly motivated and experienced support team Documentation Confidentiality

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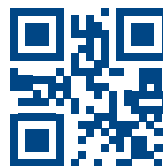
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