AMYLOID PEPTIDES OFFERED BY BACHEM

Extracellular amyloid-β 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.
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 α1-antitrypsin, complement factor H, α2-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 β/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 β-secretase cleavage site of APP and mutations of positions 692-694 (Aβ 21-23), which strongly influence the aggregation behavior of Aβ, have been studied intensively.

A choice of relevant mutations in the Aβ 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 APOEε4 allele show a higher risk in developing the disease than carriers of the other two possible alleles APOEε2 and APOEε3. The ApoEε4 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 α2-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.

<table>
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<td>D23N</td>
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<td>L705V</td>
<td>L34V</td>
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</table>
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 β-peptides (Aβ 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, α-secretase, β-secretase (or β-site APP-cleaving enzyme, BACE), and γ-secretase are involved in APP processing. APP can either be processed by the α- and γ- or by the β- and γ-secretases. The major two amyloid peptides identified in senile plaques, amyloid β-protein (1-40) (Aβ40) and amyloid β-protein (1-42) (Aβ42), are generated by successive proteolysis of APP by β- and γ-secretases. Cleavage of APP by β-secretase results in the release of the extracellular N-terminal protein fragment known as soluble APP-β molecule (sAPP-β). Then, the membrane-retained APP is further processed within the transmembrane domain by γ-secretase to yield either Aβ40 or Aβ42. The formation of Aβ40 and Aβ42 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 Aβ40 have been measured in the CSF of both healthy controls and AD patients. On the other hand, Aβ42 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 Aβ42 in the brain is a primary event in the development of AD. Increased cerebral Aβ 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 Aβ due to a third copy of the APP gene result in deposition of Aβ at an early age between 20 and 30. Formation of neurofibrillary tangles is considered as a consequence of Aβ 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.
One strategy aims at the reduction of Aβ40 and Aβ42 by inhibition of either β- or γ-secretase activity or by clearance of Aβ 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 Aβ 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 Aβ. 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 β-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 Aβ 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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Bachem’s offer for Alzheimer’s research comprises a broad choice of amyloid peptide fragments including Aβ mutant peptides.

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**AMYLOID β-PROTEIN (1-42)**

- **Amyloid β-Protein (1-42)**
  - **H-1368**
  - DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVIA

- **Amyloid β-Protein (1-42) (Hydrochloride salt)**
  - **H-6466**
  - DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVIA

- **Amyloid β-Protein (1-42) (Sodium salt)**
  - **H-7404 NEW**
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- **Amyloid β-Protein (1-42) (Trifluoroacetate salt)**
  - **H-8146 NEW**
  - DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVIA

- **Amyloid β-Protein (1-42) (HFIP-treated)**
  - **H-7442**
  - DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVIA

- **Amyloid β-Protein (1-42) (scrambled)**
  - **H-7406**
  - AIAEGDSHVLKEGAYMEIFDVQGHVF-GKIFRVDLGSHNVA

- **Teplow’s Amyloid β-Protein (1-42) (scrambled II)**
  - **H-8282 NEW**
  - YHAGYDKEVFDEGAGAEHGLAQIVRG-FGVSDVMIHINLF

- **ent-Amyloid β-Protein (1-42)**
  - **H-5566**
  - daefrhdsgyvhkqlffadvgsnkgaiiglmv-ggvia

- **Amyloid β-Protein (42-1)**
  - **H-3976**
  - AIIVGVMIGIAKNSGVDEAFFVLKQH-HVEYGDHRFEAD

- **Amyloid β-Protein (42-1) (HFIP-treated)**
  - **H-8388 NEW**
  - AIIVGVMIGIAKNSGVDEAFFVLKQH-HVEYGDHRFEAD

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

- **(Arg¹⁷)-Amyloid β-Protein (1-42)**
  - **H-6448**
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- **(D-Asp¹)-Amyloid β-Protein (1-42)**
  - **H-4854**
  - dAEFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVIA

- **(Asp³⁷)-Amyloid β-Protein (1-42)**
  - **H-7842 NEW**
  - DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVIA

- **Biotinyl-Amyloid β-Protein (1-42)**
  - **H-5642**
  - Biotinyl-DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVIA

- **Biotinyl-εAhx-Amyloid β-Protein (1-42)**
  - **H-7454 NEW**
  - Biotinyl-εAhx-DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVIA

- **Cys-Gly-Lys-Arg-Amyloid β-Protein (1-42)**
  - **H-6388**
  - CGKRDAEFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVIA

- **(Des-Glu¹)-Amyloid β-Protein (1-42)**
  - **H-7686 NEW**
  - DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVIA

- **5-FAM-Amyloid β-Protein (1-42)**
  - **H-7444**
  - Fluorescein-5-carbonyl-DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVIA
AMYLOID β-PROTEIN (1-42) (CONTINUED)

5-FAM-Amyloid β-Protein (1-42) (scrambled)
H-7836 NEW
Fluorescein-5-carbonyl-AIAEGDSHV-LKEGAYMEIFDVQGHVGKGIFRVDLG-SHNVA

FITC-β-Ala-Amyloid β-Protein (1-42)
M-2585
FITC-β-Ala-DAEFRHDSGYEVHQKLVFF AEDVSNKGAIGLMVGGVVA

FITC-εAhx-Amyloid β-Protein (1-42)
H-7666 NEW
FITC-β-Ala-DAEFRHDSGYEVHQKLVFF AEDVSNKGAIGLMVGGVVA (Dutch Mutation E22Q)

(Gln22)-Amyloid β-Protein (1-42)
H-7844 NEW
DAEFRHDSGYEVHQKLVFFAQDVGSNK-GAIIGLMV

(Dutch Mutation E22K)

(Glu20)-Amyloid β-Protein (1-42)
H-6446
DAEFRHDSGYEVHQKLVEAEDVGSNK-GAIIGLMV

(Arctic Mutation E22G)

5-TAMRA-Amyloid β-Protein (1-42)
H-7448
Fluorescein-5-carbonyl-DAEFRHDS-GYEVHQKLVFFAEDVGFSNKGAIIGLMV

AMYLOID β-PROTEIN (1-40)

Amyloid β-Protein (1-40) (Trifluoroacetate salt)
H-1194
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(Amsterdam Mutation A21G)

5-TAMRA-Amyloid β-Protein (1-42) (scrambled)
H-7408
DAEFRHDSGYEVHQKLVFFAEDVGSNK-GAIIGLMV

Amyloid β-Protein (1-40) (HFIP-treated) (scrambled)
H-7438
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Amyloid β-Protein (1-40) (scrambled)
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Amyloid β-Protein (1-40) (Trifluoroacetate salt)
H-1194
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(Amsterdam Mutation A21G)

Amyloid β-Protein (1-40) (Hydrochloride salt)
H-5558
DAEFRHDSGYEVHQKLVFFAEDVGSNK-GAIIGLMV

(Amsterdam Mutation A21G)
AMYLOID β-PROTEIN (1-40) (CONTINUED)

Teplow's Amyloid β-Protein (1-40) (scrambled II)
H-8278 NEW
YHAGVDKEVFDEGGAEHQKIVRGFGVSDVSMHLNLF

Amyloid β-Protein (1-40) amide
H-7664
DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV-NH$_2$

Amyloid β-Protein (40-1) (Hydrochloride salt)
H-7728 NEW
VVGGVMLGIIAGKNVSDEAFFVLKQHHVEYGDHRFEAD
(Hydrochloride salt)

Amyloid β-Protein (40-1) (Trifluoroacetate salt)
H-2972
VVGGVMLGIIAGKNSVDEAFFVKLQHHVEYGDHRFEAD
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Amyloid β-Protein (1-40) (mouse, rat)
H-5638
DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV

(Arg$_3$)-Amyloid β-Protein (1-40)
H-6432
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(English Mutation H6R)

(Arg$_6$)-Amyloid β-Protein (1-40)
H-7336
DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV

(Asn$_{23}$)-Amyloid β-Protein (1-40)
H-7332
DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV
(Iowa Mutation D23N)

(Asn(4-aminobuty1)$_{1-23}$,Gln(4-aminobuty1)$_{3-11,22}$)-Amyloid β-Protein (1-40)
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Biotinyl-Amyloid β-Protein (1-40)
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Biotinyl-εAhx-Amyloid β-Protein (1-40)
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Biotinyl-εAhx-DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV

(Cys$_0$)-Amyloid β-Protein (1-40)
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CDAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV

(Cys$_{26}$)-Amyloid β-Protein (1-40)
H-7402
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H-7474
DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV
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<td>DAEFRHDSGYEVHHQKLVFFAGDVGSNK-GAIIGLMVGGVV</td>
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<td>(Gly21)-Amyloid β-Protein (1-40)</td>
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<td>(Gly22)-Amyloid β-Protein (1-40)</td>
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<td>(Lys23)-Amyloid β-Protein (1-40)</td>
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<td>(Met(O)35)-Amyloid β-Protein (1-40)</td>
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<td>(Nle35)-Amyloid β-Protein (1-40)</td>
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<td>DAEFRHDSGYEVHHQKLVFFAGDVGSNK-GAIIGLMVGGVV</td>
<td>(Val34)-Amyloid β-Protein (1-40)</td>
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<td>DAEFRHDSGYEVHHQKLVFFAGDVGSNK-GAIIGLMVGGVV</td>
<td>(Piedmont Mutation L34V)</td>
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<tr>
<td>Tide Fluor™ 5WS-Amyloid β-Protein (1-40)</td>
<td>Tide Fluor™ 5WS-Amyloid β-Protein (1-40)</td>
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<td>H-8202 NEW</td>
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<td>Tide Fluor™ 5WS-DAEFRHDSGYEVHHQKLVFFAGDVGSNK-GAIIGLMVGGVV</td>
<td>Tide Fluor™ 7WS-Amyloid β-Protein (1-40)</td>
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AMYLOID β-PROTEIN (25-35)

Amyloid β-Protein (25-35)
H-1192
GSNKGAIIGLM

Amyloid β-Protein (35-25)
H-2964
MLGIAGKNSG

Amyloid β-Protein (25-35) amide
H-4222
GSNKGAIIGLM-NH₂

(Met(O)³⁵)-Amyloid β-Protein (25-35)
H-2962
GSNKGAIIGLM(O)

(Nle⁴⁵)-Amyloid β-Protein (25-35)
H-7314
GSNKGAIIGL-Nle

AMYLOID β-PROTEIN FRAGMENTS

Amyloid β-Protein (1-6)
H-8362 NEW
DAEFRH

(Val³)-Amyloid β-Protein (1-6)
H-8296 NEW
DVEFRH

Amyloid β-Protein (1-6) amide
H-8366 NEW
DAEFRH-NH₂

Acetyl-Amyloid β-Protein (1-6) amide
H-8368 NEW
Ac-DAEFRH-NH₂

Amyloid β-Protein (1-11)
H-2956
DAEFRHDGYE

Amyloid β-Protein (1-12)
H-8358 NEW
DAEFRHDGYEV

Amyloid β-Protein (1-14)
H-7372
DAEFRHDGYEVHH

Amyloid β-Protein (1-15)
H-6368
DAEFRHDGYEVHHQ

Amyloid β-Protein (1-16)
H-2958
DAEFRHDGYEVHHQK

Amyloid β-Protein (1-24)
H-7656 NEW
DAEFRHDGYSVHHQKLVFAEDV

Amyloid β-Protein (1-28)
H-7865
DAEFRHDGYSVHHQKLVFAEDVGSNK

(Gln¹¹)-Amyloid β-Protein (1-28)
H-2362
DAEFRHDGYSVHHQKLVFAEDVGSNK

(Gly²⁸, Cys³⁰)-Amyloid β-Protein (1-30) amide
H-6386
DAEFRHDGYSVHHQKLVFAEDVGSNG-GC-NH₂

Amyloid β-Protein (1-37)
H-7462
DAEFRHDGYSVHHQKLVFAEDVGSNK-GAIIGLMV

Amyloid β-Protein (1-38)
H-2966
DAEFRHDGYSVHHQKLVFAEDVGSNK-GAIIGLMVGG

Amyloid β-Protein (1-39)
H-7458
DAEFRHDGYSVHHQKLVFAEDVGSNK-GAIIGLMVGGV

Amyloid β-Protein (1-43)
H-1586
DAEFRHDGYSVHHQKLVFAEDV-GSNKGAIIGLMVGGVVIAT
Amyloid β-Protein (1-46)
H-6406
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIIGLMVGGVVIA

Amyloid β-Protein (2-42)
H-7472 NEW
AEFRHDSGYEVHHQKLVFFAEDVGGSNK-GAIIIGLMVGGVVIA

Amyloid β-Protein (3-40)
H-7672 NEW
EFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIIGLMV

(Pyr³)-Amyloid β-Protein (3-40)
H-7422
<EFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIIGLMVGGVVIA

Amyloid β-Protein (3-42)
H-7432 NEW
EFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIIGLMVGGVVIA

(Pyr³)-Amyloid β-Protein (3-42) (Ammonium salt)
H-4796
<EFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIIGLMVGGVVIA

(Pyr³)-Amyloid β-Protein (3-42) (Trifluoroacetate salt)
H-8248 NEW
<EFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIIGLMVGGVVIA

Amyloid β-Protein (4-42)
H-7434 NEW
FRHDSGYEVHHQKLVFFAEDVGSNK-GAIIIGLMVGGVVIA

Amyloid β-Protein (5-42)
H-7436 NEW
RHDSGYEVHHQKLVFFAEDVGSNK-GAIIIGLMVGGVVIA

Amyloid β-Protein (6-20)
H-6366
HDSGYEVHHQKLVFF

Amyloid β-Protein (10-20)
H-1388
YEVHHQKLVFF

Amyloid β-Protein (10-35)
H-6384
YEVHHQKLVFFAEDVGSNK-GAIIIGLMV

(Pyr³³)-Amyloid β-Protein (11-40)
H-6382
<EVHQQKLVFFAEDVGSNK-GAIIGLMV-GVV

Amyloid β-Protein (11-42)
H-7668 NEW
EVHQQKLVFFAEDVGSNK-GAIIGLMV-GVVIA

Amyloid β-Protein (12-28)
H-7910
VHHQKLVFFAEDVGSNK

Acetyl-Amyloid β-Protein (15-20) amide
H-3684
Ac-QKLVFF-NH₂

(Lys¹⁵)-Amyloid β-Protein (15-21)
H-4062
KKLVFFA

Arg¹⁶,Asp¹⁶-²⁰,Pro¹⁶-²¹,²²,Val²²,²³,Ile²⁴)-Amyloid β-Protein (15-25)
H-3904
RDLPFFPVPIID

Gly-Amyloid β-Protein (15-25)-Gly-c-aminocaproyl(-Lys)₆
H-3978
GQKLVFFAEDVGG-cAhx-KKKKKK

(Leu¹⁹)-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
ffvlk-β-Ala-k(ffvlk)
AMYLOID β-PROTEIN FRAGMENTS (CONTINUED)

Acetyl-(N-Me-Leu\textsuperscript{17},N-Me-Phe\textsuperscript{19})-Amyloid β-Protein (16-20) amide
H-7658 NEW
Ac-(K(Me)LV(Me)FF)-NH\textsubscript{2}

Amyloid β-Protein (16-22)
H-8092 NEW
KLVFFAE

(Pro\textsuperscript{18},Asp\textsuperscript{21})-Amyloid β-Protein (17-21)
H-4876
LPFFD

Acetyl-(Pro\textsuperscript{18},Asp\textsuperscript{21})-Amyloid β-Protein (17-21) amide
H-6138
Ac-LPFFD-NH\textsubscript{2}

Amyloid β-Protein (17-40)
H-7532
LVFFAEDVSNKGAIIGLMVGGVV

Amyloid β-Protein (20-29)
H-3808
FAEDVSNKK

Amyloid β-Protein (22-35)
H-1976
EDVSNKGAIIGLM

Amyloid β-Protein (29-40)
H-3984
GAIIGLMVGGVV

Propionyl-Amyloid β-Protein (31-34) amide
H-4124
Propionyl-IIGL-NH\textsubscript{2}

Amyloid β-Protein (31-35)
H-5866
IIGLM

Cys-Gly-His-Gly-Asn-Lys-Ser-Amyloid β-Protein (33-40)
H-6364
CGHGNKSLMVGGVV

Cys-Gly-Lys-Lys-Gly-Amyloid β-Protein (33-40)
H-6372
CGKKGLMVGGVV

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 β/A4 PROTEIN PRECURSOR (APP) FRAGMENTS

Acetyl-Amyloid β/A4 Protein Precursor\textsubscript{770} (96-110) (cyclized)
H-2232
Ac-NWCKRGRKQCKTHPH-NH\textsubscript{2} (Disulfide bond)

Amyloid β/A4 Protein Precursor\textsubscript{770} (135-155)
H-3726
FLHQERMDVETHLHWHTVAK

Amyloid β/A4 Protein Precursor\textsubscript{770} (394-410)
H-2594
AKERLEAKHRERMSQVM

Amyloid β/A4 Protein Precursor\textsubscript{770} (403-407)
H-1608
RERMS

Amyloid β/A4 Protein Precursor\textsubscript{770} (586-595) (human, mouse, rat)
N-1850
ISYGNDALMP

(Asn\textsuperscript{670}, Leu\textsuperscript{671})-Amyloid β/A4 Protein Precursor\textsubscript{770} (667-675)
H-4836
SEVNLDAEFR
(Asn\textsuperscript{670}, Leu\textsuperscript{671})-Amyloid β/A4 Protein Precursor\textsubscript{770} (667-676)
H-4842
SEVNLDAEFR

(Val\textsuperscript{671})-Amyloid β/A4 Protein Precursor\textsubscript{770} (667-676)
H-4838
SEVKVDAEFR

Amyloid β/A4 Protein Precursor\textsubscript{770} (740-770)
H-6216
AAVTPEERHLSKMQQNGY-ENPTYKFFEQMQN

Amyloid Precursor Frameshift Mutant C-Terminal Peptide
H-7674 NEW
RGRTSSKELA

AMYLOID-LIKE PROTEIN

APL\textsuperscript{1825}
H-7302
DELAPAGTGVSREAVSGLLIMGAGG

APL\textsuperscript{1827}
H-7304
DELAPAGTGVSREAVSGLLIMGAGGG

APL\textsuperscript{1828}
H-7306
DELAPAGTGVSREAVSGLLIMGAGGGS

BACHEM
PIONEERING PARTNER FOR PEPTIDES
### Amyloid Bri Peptides

<table>
<thead>
<tr>
<th>Peptide Name</th>
<th>Sequence</th>
<th>Note</th>
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<tbody>
<tr>
<td>Amyloid Bri Protein (1-23)</td>
<td>H-5052</td>
<td>EASNCFAIRHFENKFAVETLICS (Disulfide bond)</td>
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<td>Amyloid Bri Protein (1-34)</td>
<td>H-5526</td>
<td>&lt;EASNCFAIRHFENKFAVETLICSRT-VKKNIIEEN (Disulfide bond)</td>
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<td>Amyloid Bri Protein (1-34) (reduced)</td>
<td>H-4728</td>
<td>&lt;EASNCFAIRHFENKFAVETLICSRT-VKKNIIEEN</td>
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### Amyloid Dan Peptides

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<td>Amyloid Dan Protein (1-34)</td>
<td>H-5528</td>
<td>&lt;EASNCFAIRHFENKFAVETLICFNL-FLNSQEKHY (Disulfide bond)</td>
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<tr>
<td>Amyloid Dan Protein (1-34) (reduced)</td>
<td>H-5298</td>
<td>&lt;EASNCFAIRHFENKFAVETLICFNL-FLNSQEKHY</td>
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### Amyloid P-Component Peptides

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<tr>
<td>Amyloid P Component (27-38) amide</td>
<td>H-2942</td>
<td>EKPLQNFTLCFR-NH₂</td>
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<td>Tyr-Amyloid P Component (27-38) amide</td>
<td>H-2944</td>
<td>YEKPLQNFTLCFR-NH₂</td>
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<tr>
<td>Amyloid P Component (33-38) amide</td>
<td>H-2946</td>
<td>FTLCFR-NH₂</td>
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### Non-Aβ Component (α-Synuclein)

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<tr>
<td>α-Synuclein (34-45) (human)</td>
<td>H-8382 NEW</td>
<td>KEGVLYVGSKTK</td>
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<tr>
<td>α-Synuclein (45-54) (human)</td>
<td>H-8376 NEW</td>
<td>KEGVVHGVAT</td>
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<tr>
<td>α-Synuclein (61-95) (human)</td>
<td>H-2598</td>
<td>EQVTNVGGAVGTGVTAVAQKTVEGAG-SIAAATGFV</td>
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<tr>
<td>α-Synuclein (67-78) (human)</td>
<td>H-8384 NEW</td>
<td>GGAVTGVTAVA</td>
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<tr>
<td>α-Synuclein (71-82) (human)</td>
<td>H-8378 NEW</td>
<td>VTGVTAVAQKTV</td>
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<td>α-Synuclein Binding Peptide</td>
<td>H-8374 NEW</td>
<td>Ac-KDGIVNGVKA-NH₂</td>
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Tau protein fragments, inhibitors and substrates for β- and γ-secretase, and further peptides and biochemicals for Alzheimer’s research are available on our online shop at shop.bachem.com:

Areas of Interest
  Alzheimer’s Disease
    Tau Peptides
**β-SECRETASE SUBSTRATES**

DABCYL-(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{670}$ (667-675)-EDANS
M-2435
DABCYL-SEVNLDAEF-EDANS

Mca-(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{670}$ (667-675)-Lys(Dnp)
M-2420
Mca-SEVNLDAEFK(Dnp)

Mca-(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{670}$ (667-675)-Lys(Dnp) amide
M-2485
Mca-SEVNLDAEFK(Dnp)-NH$_2$

Lys(Dabsyl)-(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{670}$ (667-676)-Gln-Lucifer Yellow
M-2570
K(Dabsyl)-SEVNLDAEFRQ-Lucifer Yellow

Mca-Amyloid β/A4 Protein Precursor$_{670}$ (666-676)-Lys(Dnp)-Arg-Arg amide
M-2460
Mca-SEVNLDAEFRK(Dnp)RR-NH$_2$

Mca-(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{670}$ (667-676)-Lys(Dnp)-Arg-Arg amide
M-2465
Mca-SEVNLDAEFRK(Dnp)RR-NH$_2$

Arg-Glu(EDANS)-(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{670}$ (668-675)-Lys(DABCYL)-Arg
M-2470
RE(EDANS)VNLDAEFK(DABCYL)R

Abz-Amyloid β/A4 Protein Precursor$_{770}$ (669-674)-EDDnp
M-2560
Abz-VKMDAE-EDDnp

Abz-(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{770}$ (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$_2$]VAEF  

(Asn$^{670}$,Sta$^{671}$,Val$^{672}$)-Amyloid β/A4 Protein Precursor$_{770}$ (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$_2$]JAAEF

**N-1920**  
ELDL-psi[CHOHCH$_2$]AVEFGGrrrrrrrrr

OM99-2  
**H-5108**  
EVNL-psi[CHOHCH$_2$]AAEF

Z-Leu-Leu-4,5-dehydro-Leu-aldehyde  
**N-1590**  
Z-LLΔL-CHO

**γ-SECRETASE SUBSTRATES**

Abz-Amyloid β/A4 Protein Precursor$_{770}$ (708-715)-Lys(Dnp)-D-Arg-D-Arg-D-Arg amide  
**M-2540**  
Abz-GGVVIATVK(Dnp)rrr-NH$_2$

N-Me-Abz-Amyloid β/A4 Protein Precursor$_{770}$ (708-715)-Lys(Dnp)-D-Arg-D-Arg-D-Arg amide  
**M-2555**  
N-Me-Abz-GGVVIATVK(Dnp)rrr-NH$_2$

**γ-SECRETASE INHIBITORS**

L-685,458  
**H-5106**  
Boc-F-psi[CHOHCH$_2$]FLF-NH$_2$

3,5-Difluorophenylacetyl-Ala-Phg-OMe  
**N-1890**  
Z-Ile-Leu-aldehyde  
**N-1895**  
Z-II-CHO

Z-Leu-Leu-Nle-aldehyde  
**N-1695**  
Z-LL-Nle-CHO
**HUMANIN**

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<td>Colivelin</td>
<td>H-6336</td>
<td>SALLRSIPAPAGASRLLLTGEIDLP</td>
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<td>Humanin (human)</td>
<td>H-5574</td>
<td>MAPRGFSCLLILTSEIDLPVKRRA</td>
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<td>(Gly14)-Humanin (human)</td>
<td>H-5576</td>
<td>MAPRGFSCLLILTSEIDLPVKRRA</td>
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**PRION PEPTIDES**

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<td>H-1566</td>
<td>KTNMKHMAGAAAAGAVVGGGLG</td>
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<td>Prion Protein (106-126) (human)</td>
<td>H-4882</td>
<td>NGAKALMGHGATKVMGAAA</td>
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<tr>
<td>Prion Protein (118-135) (human)</td>
<td>H-4206</td>
<td>AGAVVGGGLGMYLMGSAMS</td>
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**FURTHER PEPTIDES FOR ALZHEIMER RESEARCH**

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<th>Peptide</th>
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<tr>
<td>Ac-Asp-Glu-OH (NAAG)</td>
<td>G-1015</td>
<td>Ac-DE</td>
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<tr>
<td>rec Brain-Derived Neurotrophic Factor (human)</td>
<td>H-5594</td>
<td>(rec BDNF (human))</td>
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<tr>
<td>L-Carnosine</td>
<td>G-1250</td>
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<tr>
<td>CRF (6-33) (human, rat)</td>
<td>H-3456</td>
<td>ISLDLTFFHLLREVLEMARAEQLAQQA-HS</td>
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<tr>
<td>Galanin (human) (Acetate salt)</td>
<td>H-7762 NEW</td>
<td>GWTLNSAGYLLGPHAVGNHRSFSD-KNGLTS (Acetate salt)</td>
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<tr>
<td>Galanin (human) (Trifluoroacetate salt)</td>
<td>H-8230</td>
<td>GWTLNSAGYLLGPHAVGNHRSFSD-KNGLTS (Trifluoroacetate salt)</td>
</tr>
<tr>
<td>Galanin (mouse, rat)</td>
<td>H-7450</td>
<td>GWTLNSAGYLLGPHAI5DNHRSFSD-KHGLT-NH₂</td>
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<tr>
<td>Galanin (porcine)</td>
<td>H-1365</td>
<td>GWTLNSAGYLLGPHAI5DNHRSFHD-KYGLA-NH₂</td>
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<td>Galanin (1-13)-Pro-Pro-(Ala-Leu)₂Ala amide</td>
<td>H-2576</td>
<td>GWTLNSAGYLLGPPPALALA-NH₂</td>
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<tr>
<td>Galanin (1-13)-Pro-Pro-(Ala-Leu)₂Ala amide</td>
<td>H-2576</td>
<td>GWTLNSAGYLLGPPPALALA-NH₂</td>
</tr>
<tr>
<td>(Des-Gly)-Glutathione-monoethyl ester (reduced)</td>
<td>G-4430</td>
<td>E(C-OEt)</td>
</tr>
<tr>
<td>H-Gly-Pro-Arg-OH</td>
<td>H-2930</td>
<td>GPR</td>
</tr>
<tr>
<td>H-Ile-Phe-OH</td>
<td>G-2420</td>
<td>IF</td>
</tr>
</tbody>
</table>
PRODUCT BROCHURES

- Amyloid Peptides
- Antimicrobial Peptides
- Calcitonin Gene-Related Peptides
- Caspase Substrates Inhibitors
- Cysteine Derivatives
- Dap and Dab Derivatives
- Diabetes Peptides
- Endothelins
- Fret Substrates
- Ghrelin, Leptin and Obestatin
- Lhrh Agonists and Antagonists
- Matrix Metallo-Proteinases
- Melanoma Peptides
- N-Methylated Amino Acid Derivatives
- Npy Peptides
- Orthogonality of Protecting Groups
- Par Activating Peptides
- Peptides in Cosmetics
- Pseudoprolline Dipeptides
- Secretase Substrates Inhibitors
- Tau Peptides
- Veterinary Peptides
- Veterinary Peptides
- Veterinary Peptides
rec Leptin (human)  
H-5578

rec Leptin (mouse)  
H-5582

Leptin (116-130) amide (mouse)  
(Acetate salt)  
H-8244 NEW  
SCSLPQTSGLOKPKES-NH₂  
(Acetate salt)

Leptin (116-130) amide (mouse)  
(Trifluoroacetate salt)  
H-3966  
SCSLPQTSGLOKPKES-NH₂  
(Trifluoroacetate salt)

H-Leu-Ile-OH  
G-2525

PACAP-38 (human, mouse, ovine, porcine, rat)  
H-8430  
HSDGIFT_DSYSRYRKQMAVKKYLAAV- 
LGKRYKQRVKKNK-NH₂

Presenilin-1 (331-349)-Cys  
(human, mouse)  
H-3988  
NDDGGFSEEEWAEQRDSDLGC

Secretoneurin (mouse, rat)  
H-5512  
TNEVEEQYTPQSLATLESVFQELG- 
KLTGPSNQ

TRAF6 Peptide  
H-7604 NEW  
AAVALLPAVLALLAPESAS- 
GPSEDPSVNLK

TRAF6 Control Peptide  
H-7606 NEW  
AAVALLPAVLALLAPESASGASA- 
DASVNLK

WRW4  
H-7596 NEW  
WRWWWW-NH₂

Abz-Gly-Ala-Lys(Ac)-Ala-Ala- 
Dap(Dnp)-NH₂  
M-2700  
Abz-GAK(Ac)AA-Dpa-NH₂

Dansyl-D-Ala-Gly-4-nitro-Phe- 
Gly-OH  
M-2650  
Dns-aGF(NO₂)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-DPMSSTYIEELGTKREVTP- 
PKYRELLA-NH₂

1,3-Bis-(Z-Leu-Leu)- 
diaminoacetone  
((Z-LL)₂ Ketone)  
C-4275 NEW  
(Z-LL-CH₂)₂CO

Z-Pro-Pro-aldehyde- 
dimethyl acetal  
N-1490  
Z-PP-CH(OMe)₂
<table>
<thead>
<tr>
<th>Compound</th>
<th>Code</th>
</tr>
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<tbody>
<tr>
<td>Ac-DL-Asp-OH</td>
<td>F-4070</td>
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<tr>
<td>N-Me-D-Asp-OH (NMDA)</td>
<td>F-2415</td>
</tr>
<tr>
<td>Ac-Cys-OH (NAC)</td>
<td>E-3710</td>
</tr>
<tr>
<td>H-D-Pen-OH (D-Penicillamine)</td>
<td>F-4235</td>
</tr>
<tr>
<td>H-Ser(PO₃H₂)-OH (L-Phosphoserine, Dexfosferine)</td>
<td>F-2030</td>
</tr>
<tr>
<td>D-Cycloserine</td>
<td>F-1480</td>
</tr>
<tr>
<td>L-trans-Epoxyysuccinyl-Leu-3-methylbutylamide-ethyl ester (E-64d, Aloxistatin, Loxistatin, EP453)</td>
<td>N-1650</td>
</tr>
<tr>
<td>sn-Glycero-3-phosphocholine (Choline alfoscerate, L-α-GPC, L-α-Lecithin)</td>
<td>O-1590</td>
</tr>
<tr>
<td>1-O-Hexadecyl-2-O-acetyl-sn-glycero-3-phosphocholine (PAF (C₁₆))</td>
<td>O-1270</td>
</tr>
<tr>
<td>Melatonin</td>
<td>Q-1300</td>
</tr>
<tr>
<td>Phenserine</td>
<td>Q-1860</td>
</tr>
</tbody>
</table>
Molecular model of human brain-derived beta-amyloid fibrils isolated from a patient with Alzheimer’s disease. The fibrils are made up of β-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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## Custom Synthesis at Bachem

| **Quality** | GMP and non-GMP quality  
State of the art analytical capabilities |
|-------------|--------------------------------------------------|
| **Chemistry** | Fmoc-, Boc-, Z- and other synthetic strategies  
Synthesis of complex peptides |
| **Capacity** | 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 |
| **Modifications** | Acylation, acetylation, amidation, etc.  
Cyclizations  
Stabilizing modifications |
| **Support** | Highly motivated and experienced support team  
Documentation  
Confidentiality |
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