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.

AMYLOID β-PROTEIN (1-42)

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. Aβ42 is the main constituent of amyloid plaques.

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, a₂-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 a₂-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.
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.

For more details on our Alzheimer’s disease peptides, please go to: shop.bachem.com
AMYLOID β-PROTEIN (1-42)

Amyloid β-Protein (1-42)
H-1368
DAEFRHDSGYEVHHQKLVVFAEDVGSNK-GAIILMVGVGVIA

Amyloid β-Protein (1-42) (Hydrochloride salt)
H-6466
DAEFRHDSGYEVHHQKLVVFAEDVGSNK-GAIILMVGVGVIA (Hydrochloride salt)

Amyloid β-Protein (1-42) (Sodium salt)
H-7404 NEW
DAEFRHDSGYEVHHQKLVVFAEDVGSNK-GAIILMVGVGVIA (Sodium salt)

Amyloid β-Protein (1-42) (Trifluoroacetate salt)
H-8146 NEW
DAEFRHDSGYEVHHQKLVVFAEDVGSNK-GAIILMVGVGVIA (Trifluoroacetate salt)

Amyloid β-Protein (1-42) (HFIP-treated)
H-7442
DAEFRHDSGYEVHHQKLVVFAEDVGSNK-GAIILMVGVGVIA

Teplow’s Amyloid β-Protein (1-42) (scrambled)
H-7406
AIAEGDSHVLKEGAYMEIFDVQGHVFG-KIFRVDLGSNVA

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H-8282 NEW
YHAGVDKEVFDEGAGAEHGLAQKIVRG-FGVSDVSMIHINLF

Amyloid β-Protein (42-1) (HFIP-treated)
H-8388 NEW
AIVVGGVMLGIAGKNSGVDEAFFVLKQH-HVEYGSDFRFEAD

Amyloid β-Protein (1-42) (mouse, rat)
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(Arg¹⁷)-Amyloid β-Protein (1-42)
H-6448
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(D-Asp¹)-Amyloid β-Protein (1-42)
H-4854
daEFRHDSGYEVHHQKLVVFAEDVGSNK-GAIILMVGVGVIA

(Asp³⁷)-Amyloid β-Protein (1-42)
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Biotinyl-Amyloid β-Protein (1-42)
H-5642
Biotinyl-DAEFRHDSGYEVHHQKLVF-FAEDVGSNKGAIIGLMVGVGVIA

Biotinyl-εAhx-Amyloid β-Protein (1-42)
H-7454 NEW
Biotinyl-εAhx-DAEFRHDSGYEVHHQKLVF-FAEDVGSNKGAIIGLMVGVGVIA

Cys-Gly-Lys-Arg-Amyloid β-Protein (1-42)
H-6388
CGKRDAEFRHDSGYEVHHQKLVF-FAEDVGSNKGAIIGLMVGVGVIA

(Des-Glu¹)-Amyloid β-Protein (1-42)
H-7686 NEW
DAEFRHDSGYEVHHQKLVVFAEDVGSNK-GAIILMVGVGVIA (Osaka Mutation E22Δ)

5-FAM-Amyloid β-Protein (1-42)
H-7444
Fluorescein-5-carbonyl-DAEFRHDSGYEVHHQKLVF-FAEDVGSNK-GAIILMVGVGVIA
**AMYLOID \(\beta\)-PROTEIN (1-42) (CONTINUED)**

- **5-FAM-Amyloid \(\beta\)-Protein (1-42)** (scrambled)
  - H-7836 NEW
  - Fluorescein-5-carbonyl-\(\text{AIAEGD}&^\text{SHVLKEGAYMEIFDVQGHVFGKIFR}
  \text{VVDGLGSHNVA}

- **FITC-\(\alpha\)-Ala-Amyloid \(\beta\)-Protein (1-42)**
  - M-2585
  - FITC-\(\alpha\)-Ala-DAEFRHDSGYEVHHQKLVF
  \text{AEDVGSNKGAIIGLMVGGVVIA}

- **FITC-\(\epsilon\)Ahx-Amyloid \(\beta\)-Protein (1-42)**
  - H-7666 NEW
  - FITC-\(\epsilon\)Ahx-DAEFRHDSGYEVHHQKLFFAEDV
  \text{GSNKGAIIGLMVGGVVIA}

- **(Gln\text{22})-Amyloid \(\beta\)-Protein (1-42)**
  - H-7844 NEW
  - DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAII
  \text{GLMVGGVVIA}

- **(Dutch Mutation E22Q)-Amyloid \(\beta\)-Protein (1-42)**
  - H-7848 NEW
  - DAEFRHDSGYEVHHQKLFFAQDVGSNK-GAI
  \text{IGLMVGGVVIA}

- **(Gly\text{22})-Amyloid \(\beta\)-Protein (1-42)**
  - H-6124
  - DAEFRHDSGYEVHHQKLFFAGDVGSNK-GAI
  \text{IGLMVGGVVIA}

- **(Arctic Mutation E22G)-Amyloid \(\beta\)-Protein (1-42)**
  - H-7866 NEW
  - DAEFRHDSGYEVHHQKLFFAKDVGSNK-GAI
  \text{IGLMVGGVVIA}

- **(Glu\text{20})-Amyloid \(\beta\)-Protein (1-42)**
  - H-6446
  - DAEFRHDSGYEVHHQKLFFEAEDVGSNK-GAI
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- **(Flemish Mutation A21G)-Amyloid \(\beta\)-Protein (1-42)**
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- **(Italian Mutation E22K)-Amyloid \(\beta\)-Protein (1-42)**
  - H-7848 NEW
  - DAEFRHDSGYEVHHQKLFFAKDVGSNK-GAI
  \text{IGLMVGGVVIA}

- **(Met(O\text{35})-Amyloid \(\beta\)-Protein (1-42)**
  - H-5888
  - DAEFRHDSGYEVHHQKLFFAEDVG
  \text{SNKGAIIGLMVGGVVIA}

- **(Met(O\text{235})-Amyloid \(\beta\)-Protein (1-42)**
  - H-7324
  - DAEFRHDSGYEVHHQKLFFAEDVG
  \text{SNKGAIIGLMVGGVVIA}

- **(Nle\text{35})-Amyloid \(\beta\)-Protein (1-42)**
  - H-7308
  - DAEFRHDSGYEVHHQKLFFAEDVG
  \text{NSNKGAIIGLMVGGVVIA}

- **5-TAMRA-Amyloid \(\beta\)-Protein (1-42)**
  - H-7448
  - Fluorescein-5-carbonyl-DAEFRHDSGYEVHHQKLFFAEDVG
  \text{FAMSNKGAIIGLMVGGVVIA}

**AMYLOID \(\beta\)-PROTEIN (1-40)**

- **Amyloid \(\beta\)-Protein (1-40)** (Trifluoroacetate salt)
  - H-1194
  - DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAII
  \text{GLMVGGVV}

- **Amyloid \(\beta\)-Protein (1-40)** (Hydrochloride salt)
  - H-5568
  - DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAII
  \text{GLMVGGVV}

- **Amyloid \(\beta\)-Protein (1-40)** (HFiP-treated)
  - H-7438
  - DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAII
  \text{GLMVGGVV}

- **Amyloid \(\beta\)-Protein (1-40)** (scrambled)
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AMYLOID β-PROTEIN (1-40) (CONTINUED)

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FITC-β-Ala-Amyloid β-Protein (1-40)  
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(Gln9)-Amyloid β-Protein (1-40)  
H-6434  
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(Dutch Mutation E22Q)  

(Gln22)-Amyloid β-Protein (1-40)  
H-6696  
DAEFRHDSGYEVHQKLVFFAEDVGFAMSNK-GAILGLMVGGVV  
(Dutch Mutation E22Q)

(Gln22, Asn23)-Amyloid β-Protein (1-40)  
H-7412  
DAEFRHDSGYEVHQKLVFFAEDVGFAMSNK-GAILGLMVGGVV  
(Dutch/Iowa Mutation E22Q/D23N)

(Gly21)-Amyloid β-Protein (1-40)  
H-6702  
DAEFRHDSGYEVHQKLVFFAEDVGFAMSNK-GAILGLMVGGVV  
(Flemish Mutation A21G)

(Gly22)-Amyloid β-Protein (1-40)  
H-6694  
DAEFRHDSGYEVHQKLVFFAEDVGFAMSNK-GAILGLMVGGVV  
(Arctic Mutation E22G)

(Lys23)-Amyloid β-Protein (1-40)  
H-6698  
DAEFRHDSGYEVHQKLVFFAEDVGFAMSNK-GAILGLMVGGVV  
(Italian Mutation E22K)

(Met(O)35)-Amyloid β-Protein (1-40)  
H-7476  
DAEFRHDSGYEVHQKLVFFAEDVGFAMSNK-GAILGLM(O)VGGVV

(Nle35)-Amyloid β-Protein (1-40)  
H-7312  
DAEFRHDSGYEVHQKLVFFAEDVGFAMSNK-GAILGL-Nle-VGGVV

5-TAMRA-Amyloid β-Protein (1-40)  
H-7452  
5-TAMRA-DAEFRHDSGYEVHQKLVFFAEDVGFAMSNK-GAILGLMVGGVV

Tide Fluor™ 5WS-Amyloid β-Protein (1-40)  
H-8202 NEW  
Tide Fluor™ 5WS-DAEFRHDSGYEVHQKLVFFAEDVGFAMSNK-GAILGLMVGGVV

Tide Fluor™ 7WS-Amyloid β-Protein (1-40)  
H-8206 NEW  
Tide Fluor™ 7WS-DAEFRHDSGYEVHQKLVFFAEDVGFAMSNK-GAILGLMVGGVV

(Val34)-Amyloid β-Protein (1-40)  
H-7414  
DAEFRHDSGYEVHQKLVFFAEDVGFAMSNK-GAILGLMVGGVV  
(Piedmont Mutation L34V)
### AMYLOID β-PROTEIN (25-35)

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<th>Peptide</th>
<th>Hexapeptide Number</th>
<th>Sequence</th>
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<tr>
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<td>GSNKGAIIGLM</td>
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<td>(Met(O)35)-Amyloid β-Protein (25-35)</td>
<td>H-2962</td>
<td>GSNKGAIIGLM(O)</td>
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<td>Amyloid β-Protein (35-25)</td>
<td>H-2964</td>
<td>MLGIAGKNSG</td>
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<td>Amyloid β-Protein (25-35) amide</td>
<td>H-4222</td>
<td>GSNKGAIIGLM-NH₂</td>
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### AMYLOID β-PROTEIN FRAGMENTS

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<tr>
<td>Amyloid β-Protein (1-6)</td>
<td>H-8362 NEW</td>
<td>DAEFRH</td>
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<td>(Val³⁵)-Amyloid β-Protein (1-6)</td>
<td>H-8296 NEW</td>
<td>DVEFRH</td>
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<td>Amyloid β-Protein (1-6) amide</td>
<td>H-8366 NEW</td>
<td>DAEFRH-NH₂</td>
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<td>Acetyl-Amyloid β-Protein (1-6) amide</td>
<td>H-8368 NEW</td>
<td>Ac-DAEFRH-NH₂</td>
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<td>Amyloid β-Protein (1-11)</td>
<td>H-2956</td>
<td>DAEFRHDSGYE</td>
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<td>Amyloid β-Protein (1-12)</td>
<td>H-8358 NEW</td>
<td>DAEFRHDSGYEV</td>
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<td>Amyloid β-Protein (1-14)</td>
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<td>Amyloid β-Protein (1-15)</td>
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<td>Amyloid β-Protein (1-24)</td>
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<td>(Gly²⁸, Cys³⁰)-Amyloid β-Protein (1-30) amide</td>
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AMYLOID β-PROTEIN FRAGMENTS (CONTINUED)

Amyloid β-Protein (1-46)
H-6406
DAEFRHDSGYEVHHQKLFFAEDVGSNKGAIIGLMVGGVVIATVIV

Amyloid β-Protein (2-42)
H-7472 NEW
AEFRHDSGYEVHHQKLFFAEDVGSNKGAIIGLMVGGVVI

Amyloid β-Protein (3-40)
H-7672 NEW
EFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVV

(Pyr³)-Amyloid β-Protein (3-40)
H-7422
<EFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVV

Amyloid β-Protein (3-42)
H-7432 NEW
EFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVVI

(Pyr³)-Amyloid β-Protein (3-42)
(Ammonium salt)
H-4796
<EFRHDSGYEVHHQKLFFAEDVGSNK-GAIIGLMVGGVVI

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

Lys¹⁹)-Amyloid β-Protein (15-21)
H-4062
KKLVFFA

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

Gly–Amyloid β-Protein (15-25)–Gly-c-aminocaproyl(-Lys)₆
H-3978
GQKLVFFAEDVGG-εAhx-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-Leu¹⁷,N-Me-Phe¹⁹)-Amyloid β-Protein (16-20) amide
H-7658 NEW
Ac-K(Me)LV(Me)FF-NH₂

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

(Pro¹⁸,Asp²¹)-Amyloid β-Protein (17-21)
H-4876
LPFFD

Acetyl-(Pro¹⁸,Asp²¹)-Amyloid β-Protein (17-21) amide
H-6138
Ac-LPFFD-NH₂

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

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

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

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

Propionyl-Amyloid β-Protein (31-34) amide
H-4124
Propionyl-IIGL-NH₂

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
CGKKGLMVGGVV

Cys-Gly-Lys-Lys-Gly-Amyloid β-Protein (35-40)
H-6378
CGKKGMVGGVV

Amyloid β-Protein (36-38)
H-5270
VG

Amyloid β-Protein (37-39)
H-3500
GG

Methoxysuccinyl-Val-Val-Ile-Ala-pNA
(MeOHsuc-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\(_{770}\) (96-110) (cyclized)
H-2232
Ac-NWCKRGRKQCKTHPH-NH\(_2\)
(Disulfide bond)

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

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

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

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

(Asn\(^{670}\),Leu\(^{671}\))-Amyloid β/A4 Protein Precursor\(_{770}\) (667-675)
H-4836
SEVNDAEFR
(Swedish Double Mutation K670N / M671L)

Amyloid β/A4 Protein Precursor\(_{770}\) (667-676)
H-6842
SEVKMDAEFR

(Asn\(^{670}\),Leu\(^{671}\))-Amyloid β/A4 Protein Precursor\(_{770}\) (667-676)
H-4834
SEVNDAEFR
(Val\(^{671}\))-Amyloid β/A4 Protein Precursor\(_{770}\) (667-676)
H-4838
SEVKVDAEFR

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

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

AMYLOID-LIKE PROTEIN

APL\(_{1825}\)
H-7302
DELAPAGTGVSREAVSGLIMGAGG

APL\(_{1827}\)
H-7304
DELAPAGTGVSREAVSGLIMGAGGS

APL\(_{1828}\)
H-7306
DELAPAGTGVSREAVSGLIMGAGG-GGSL
# Amyloid Bri Peptides

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<tr>
<th>Peptide Name</th>
<th>Sequence</th>
<th>References</th>
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<tbody>
<tr>
<td>Amyloid Bri Protein (1-23)</td>
<td>EASNCFAIRHFENKFAVELICS</td>
<td>H-5052</td>
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<td>EASNCFAIRHFENKFAVELICSRTVKKNIEEEN</td>
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<td>Amyloid Bri Protein Precursor</td>
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# Amyloid Dan Peptides

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<tr>
<td>Amyloid Dan Protein (1-34)</td>
<td>EASNCFAIRHFENKFAVELICFNLFLNSQEKHY</td>
<td>H-5528</td>
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<td>Amyloid Dan Protein (1-34)</td>
<td>EASNCFAIRHFENKFAVELICFNLFLNSQEKHY</td>
<td>H-5298 (reduced)</td>
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# Amyloid P-Component Peptides

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<tr>
<td>Amyloid P Component (27-38) amide</td>
<td>EKPLQNF TCFR-NH₂</td>
<td>H-2942</td>
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<td>Amyloid P Component (33-38) amide</td>
<td>FTCFR-NH₂</td>
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# Tyr-Amyloid P Component (27-38) amide

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<tr>
<td>Tyr-Amyloid P Component (27-38) amide</td>
<td>YEKPLQNF TCFR-NH₂</td>
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# Non-αβ Component (α-Synuclein)

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<tr>
<td>α-Synuclein (34-45) (human)</td>
<td>KEGVLYVGSKTK</td>
<td>H-8382 NEW</td>
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<tr>
<td>α-Synuclein (45-54) (human)</td>
<td>KEGVVHGVTAT</td>
<td>H-8376 NEW</td>
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<tr>
<td>α-Synuclein (61-95) (human)</td>
<td>EQVTNVGGAVVTAVAG- SIAATGFV</td>
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<tr>
<td>α-Synuclein Binding Peptide</td>
<td>Ac-KDGIVNGVKA-NH₂</td>
<td>H-8374 NEW</td>
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<tr>
<td>α-Synuclein (67-78) (human)</td>
<td>GGAVVTVTA</td>
<td>H-8384 NEW</td>
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<td>α-Synuclein (71-82) (human)</td>
<td>VTAVAKTV</td>
<td>H-8378 NEW</td>
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<th>Peptide Name</th>
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<tr>
<td>α-Synuclein (67-78) (human)</td>
<td>GGAVVTVTA</td>
<td>H-8384 NEW</td>
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<tr>
<td>α-Synuclein (71-82) (human)</td>
<td>VTAVAKTV</td>
<td>H-8378 NEW</td>
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RELATED AD PRODUCTS

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**

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<th>Substrate</th>
<th>Code</th>
<th>Description</th>
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<td>DABCYL-(Asn&lt;sup&gt;670&lt;/sup&gt;,Leu&lt;sup&gt;671&lt;/sup&gt;)-Amyloid β/A4 Protein Precursor&lt;sub&gt;770&lt;/sub&gt; (667-675)-EDANS</td>
<td>M-2435</td>
<td><strong>Arg-Glu(EDANS)-(Asn&lt;sup&gt;670&lt;/sup&gt;,Leu&lt;sup&gt;671&lt;/sup&gt;)-Amyloid β/A4 Protein Precursor&lt;sub&gt;770&lt;/sub&gt; (668-675)-Lys(DABCYL)-Arg</strong></td>
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<td>RE(EDANS)VNLDÆEÆF(K(DABCYL))R</td>
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<td>Mca-(Asn&lt;sup&gt;670&lt;/sup&gt;,Leu&lt;sup&gt;671&lt;/sup&gt;)-Amyloid β/A4 Protein Precursor&lt;sub&gt;770&lt;/sub&gt; (667-675)-Lys(Dnp)</td>
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<td>Abz-Amyloid β/A4 Protein Precursor&lt;sub&gt;770&lt;/sub&gt; (669-674)-EDDnp</td>
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<td>Abz-VKMDAE-EDDnp</td>
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<td>M-2485</td>
<td>Abz-(Asn&lt;sup&gt;670&lt;/sup&gt;,Leu&lt;sup&gt;671&lt;/sup&gt;)-Amyloid β/A4 Protein Precursor&lt;sub&gt;770&lt;/sub&gt; (669-674)-EDDnp</td>
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<td>Lys(Dabsyl)-(Asn&lt;sup&gt;670&lt;/sup&gt;,Leu&lt;sup&gt;671&lt;/sup&gt;)-Amyloid β/A4 Protein Precursor&lt;sub&gt;770&lt;/sub&gt; (667-676)-Gln-Lucifer Yellow</td>
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### β-Secretase Inhibitors

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<th>Formula</th>
<th>Modification</th>
<th>Reference</th>
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<tr>
<td>Ac-Val-Met-[(2S,4S,5S)-5-amino-4-hydroxy-2-isopropyl-7-methyl-octanoyl]-Ala-Glu-Phe-OH</td>
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<tr>
<td>Ac-VML-psi[CHOHCH₂]VAEF</td>
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<td>(Asn⁶⁷⁰,Sta⁶⁷¹,Val⁶⁷²)-Amyloid β/A4 Protein Precursor狲 (662-675)</td>
<td>H-4848</td>
<td>KTEEISEVN-Sta-VAEF</td>
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<td>H-Glu-Leu-Asp-[(2R,4S,5S)-5-amino-4-hydroxy-2,7-dimethyl-octanoyl]-Ala-Glu-Phe-OH</td>
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<td>ELDL-psi[CHOHCH₂]AAEF</td>
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<td>OM99-2</td>
<td>H-5108</td>
<td>EVNL-psi[CHOHCH₂]AAEF</td>
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<tr>
<td>Z-Leu-Leu-4,5-dehydro-Leu-aldehyde</td>
<td>N-1590</td>
<td>Z-LLΔL-CHO</td>
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### γ-Secretase Substrates

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<td>Abz-Amyloid β/A4 Protein Precursor狲 (708-715)-Lys(Dnp)-D-Arg-D-Arg-D-Arg amide</td>
<td>M-2540</td>
<td>Abz-GGVVIATVK(Dnp)rrr-NH₂</td>
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<td>N-Me-Abz-GGVVIATVK(Dnp)rrr-NH₂</td>
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<td>Z.Ile-Leu-aldehyde</td>
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### γ-Secretase Inhibitors

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<td>H-5106</td>
<td>Boc-F-psi[CHOHCH₂]FLF-NH₂</td>
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<td>3,5-Difluorophenylacetyl-Ala-Phg-OMe</td>
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<td>Z-Leu-Leu-aldehyde</td>
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**HUMANIN**

Colivelin  
H-6336  
SALLRSIPAPAGSRLLLTLTGEIDLP

Humanin (human)  
H-5574  
MAPRGSCLLLLTGEIDLPVKRRA

**PRION PEPTIDES**

Prion Protein (106-126) (human)  
H-1566  
KTNMKHMAGAAAGAVVGGGLG

Prion Protein (106-126) (human) (scrambled)  
H-4882  
NGAKALMGHGATKVMGAAA

**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  
ISLDTFHLLEVLEMARAEQLAQQA- HS

Galanin (human) (Acetate salt)  
H-7762 NEW  
GWTLNSAGYLGPAHVNHRSFSD-KNGLTS (Acetate salt)

Galanin (human) (Trifluoroacetate salt)  
H-8230  
GWTLNSAGYLGPAHVNHRSFSD-KNGLTS (Trifluoroacetate salt)

Galanin (mouse, rat)  
H-7450  
GWTLNSAGYLGPAHIDNHRSFSD-KHGLT-NH₂

Galanin (porcine)  
H-1365  
GWTLNSAGYLGPAHIDNHRSFHD-KYGLA-NH₂

Galanin (1-13)-Pro-Pro-(Ala-Leu)₂Ala amide (M40)  
H-2576  
GWTLNSAGYLPPALALANH₂

(Des-Gly)-Glutathione-monoethyl ester (reduced) (GCEE, γ-GCE)  
G-4430  
EIC-OEt

H-Gly-Pro-Arg-OH  
H-2930  
GPR

H-Ile-Phe-OH  
G-2420  
IF
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
SCSLPQTSGLOKQES-NH₂
(Acetate salt)

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

H-Leu-Ile-OH
G-2525
LI

PACAP-38 (human, mouse, ovine, porcine, rat)
H-8430
HSDGIFTDSYRSYRKMAMVKKYLAVALGKRKYKVKNKNH₂

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

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

TRAF6 Peptide
H-7604 NEW
AAVALLPAVVALLAPESAS-GPSEDPSVNFKL

TRAF6 Control Peptide
H-7606 NEW
AAVALLPAVVALLAPESASGASA-DASVNFKL

WRW4
H-7596 NEW
WRWWWWW-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-DPMSSTYIEELGKREVTP-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>
</thead>
<tbody>
<tr>
<td>Ac-DL-Asp-OH</td>
<td>F-4070</td>
</tr>
<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-Epoxysuccinyl-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>Phenerserine</td>
<td>Q-1860</td>
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</tbody>
</table>
ALZHEIMER’S BETA-AMYLOID FIBRILS

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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ALFRED PASIEKA
## Custom Synthesis at Bachem

<table>
<thead>
<tr>
<th>Category</th>
<th>Details</th>
</tr>
</thead>
</table>
| • 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                                                                                                                         |
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

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