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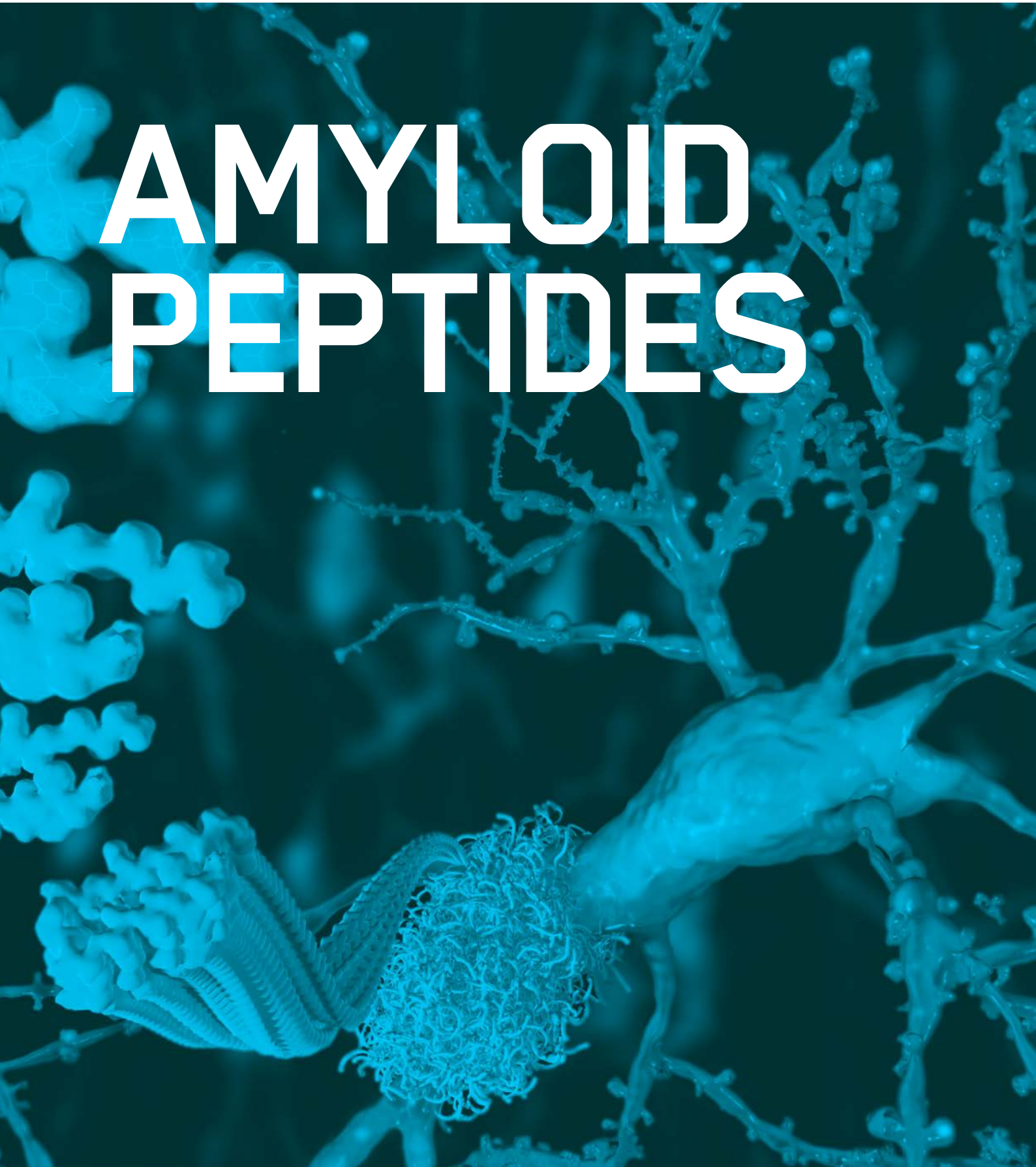
# AMYLOID PEPTIDES



# 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.

## 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.

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 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 $\alpha_2$-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 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.

| Exchanged Position | | Designation |
|---|---|---|
| in APP | in Aβ | |
| A673T | A2T | Icelandic |
| H677R | H6R | English |
| D678H | D7H | Taiwanese |
| D678N | D7N | Tottori |
| A692G | A21G | Flemish |
| E693D | E22Δ | 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.

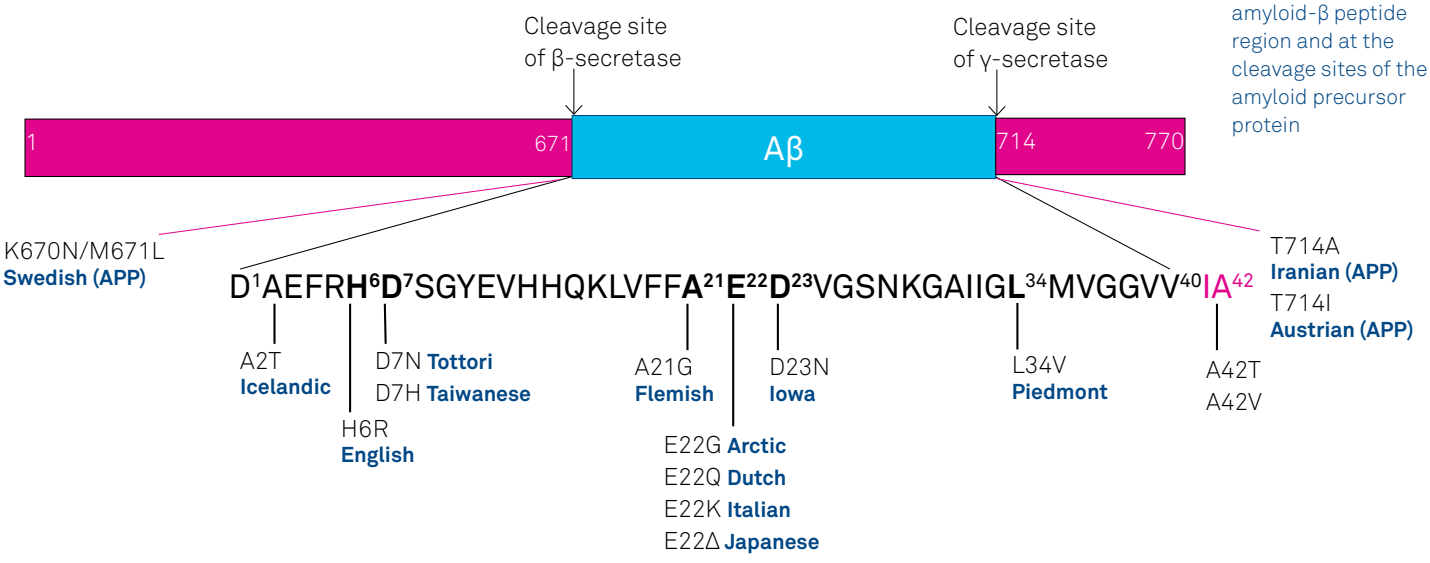
The etiology of AD is still not completely understood. Initial research focused upon determining the molecular structure of the senile plaques and the neurofibrillary tangles originally described by Alois Alzheimer. The main constituents of the senile plaques were identified as cleavage products of APP, designated as amyloid 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.

## Amyloid Precursor Protein (APP)




Mutants in the amyloid-β peptide region and at the cleavage sites of the amyloid precursor protein

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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# PEPTIDES FOR ALZHEIMER'S RESEARCH

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)**
4014447
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVVIA

**Amyloid β-Protein (1-42) (Hydrochloride salt)**
4045866
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVVIA
(Hydrochloride salt)

**Amyloid β-Protein (1-42) (Sodium salt)**
4089802 NEW
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVVIA
(Sodium salt)

**Amyloid β-Protein (1-42) (Trifluoroacetate salt)**
4061966 NEW
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVVIA
(Trifluoroacetate salt)

**Amyloid β-Protein (1-42) (HFIP-treated)**
4090148
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVVIA

**Amyloid β-Protein (1-42) (scrambled)**
4064853
AIAEGDSHVLKEGAYMEIFDVQGHVFG-GKIFRVVDLGSHNVA

**Teplow's Amyloid ß-Protein (1-42) (scrambled II)**
4104168 NEW
YHAGVDKEVVFDEGAGAEHGLAQKIVRG-FGVSDVSMIHINLF

**ent-Amyloid β-Protein (1-42)**
4037836
daefrhdsgyevhhqklvffaedvgsnkgaiiglm-vggvvia
(all-D peptide)

**Amyloid β-Protein (42-1)**
4027991
AIVVGGVMLGIIAGKNSGVDEAFFVLKQH-HVEYGSDHRFEAD

**Amyloid β-Protein (42-1) (HFIP-treated)**
4107743 NEW
AIVVGGVMLGIIAGKNSGVDEAFFVLKQH-HVEYGSDHRFEAD

**Amyloid β-Protein (1-42) (mouse, rat)**
4035885
DAEFGHDSGFEVRHQKLVFFAEDVGSNK-GAIIGLMVGGVVIA

**(Asp$^{37}$)-Amyloid β-Protein (1-42)**
4099631 NEW
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVDGVVIA

**Biotinyl-Amyloid β-Protein (1-42)**
4038795
Biotinyl-DAEFRHDSGYEVHHQKLVF-FAEDVGSNKGAIIGLMVGGVVIA

**Biotinyl-εAhx-Amyloid β-Protein (1-42)**
4090155 NEW
Biotinyl-εAhx-DAEFRHDSGYEVHHQKLV FFAEDVGSNKGAIIGLMVGGVVIA

**Cys-Gly-Lys-Arg-Amyloid β-Protein (1-42)**
4037472
CGKRDAEFRHDSGYEVHHQKLVFFAED-VGSNKGAIIGLMVGGVVIA

**(Des-Glu$^{1}$)-Amyloid β-Protein (1-42)**
4074277 NEW
DAEFRHDSGYEVHHQKLVFFADVGSNK-GAIIGLMVGGVVIA
(Osaka Mutation E22Δ)

**5-FAM-Amyloid β-Protein (1-42)**
4090151
Fluorescein-5-carbonyl-DAEFRHDS-GYEVHHQKLVFFAEDVGFAMSNK-GAIIGLMVGGVVIA

**5-FAM-Amyloid β-Protein (1-42) (scrambled)**
4099694 NEW
Fluorescein-5-carbonyl-AIAEGDSHV-LKEGAYMEIFDVQGHVFGGKIFRVVDLG-SHNVA

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

**FITC-β-Ala-Amyloid β-Protein (1-42)**
4033502
FITC-β-Ala-DAEFRHDSGYEVHHQKLVFF AEDVGSNKGAIIGLMVGGVVIA

**FITC-εAhx-Amyloid β-Protein (1-42)**
4095738 NEW
FITC-β-Ala-DAEFRHDSGYEVHHQKLVFF AEDVGSNKGAIIGLMVGGVVIA

**(Gln$^{22}$)-Amyloid β-Protein (1-42)**
4050945 NEW
DAEFRHDSGYEVHHQKLVFFAQDVGSNK-GAIIGLMVGGVVIA
(Dutch Mutation E22Q)

**(Gly$^{21}$)-Amyloid β-Protein (1-42)**
4039930 NEW
DAEFRHDSGYEVHHQKLVFFGEDVGSNK-GAIIGLMVGGVVIA
(Flemish Mutation A21G)

**(Gly$^{22}$)-Amyloid β-Protein (1-42)**
4035371
DAEFRHDSGYEVHHQKLVFFAGDVGSNK-GAIIGLMVGGVVIA
(Arctic Mutation E22G)

**(Lys$^{22}$)-Amyloid β-Protein (1-42)**
4064438 NEW
DAEFRHDSGYEVHHQKLVFFAKDVGSNK-GAIIGLMVGGVVIA
(Italian Mutation E22K)

**(Met(O)$^{35}$)-Amyloid β-Protein (1-42)**
4041297
DAEFRHDSGYEVHHQKLVFFAEDVGSNK GAIIGLM(O)VGGVVIA

**(Met(O$_2$)$^{35}$)-Amyloid β-Protein (1-42)**
4075678
DAEFRHDSGYEVHHQKLVFFAEDVGSNK GAIIGLM($O_2$)VGGVVIA

**(Nle$^{35}$)-Amyloid β-Protein (1-42)**
4053225
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGL-Nle-VGGVVIA

**5-TAMRA-Amyloid β-Protein (1-42)**
4090153
Fluorescein-5-carbonyl- DAEFRHDS-GYEVHHQKLVFFAEDVGFAMSNK-GAIIGLMVGGVVIA

# AMYLOID β-PROTEIN (1-40)

**Amyloid β-Protein (1-40) (Trifluoroacetate salt)**
4014442
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVV
(Trifluoroacetate salt)

**Amyloid β-Protein (1-40) (Hydrochloride salt)**
4038267
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVV
(Hydrochloride salt)

**Amyloid β-Protein (1-40) (HFIP-treated)**
4090147
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVV

**Amyloid β-Protein (1-40) (scrambled)**
4089803
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVV

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

**Teplow's Amyloid β-Protein (1-40) (scrambled II)**
4104167 NEW
YHAGVDKEVVFDEGGAEHGLAQKIVRGF-GVSDVSMIHNLF

**Amyloid β-Protein (1-40) amide**
4095737
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVV-$NH_2$

**Amyloid β-Protein (40-1) (Hydrochloride salt)**
4095853 NEW
VVGGVMLGIIAGKNSGVDEAFFVLKQHH-VEYGSDHRFEAD
(Hydrochloride salt)

**Amyloid β-Protein (40-1) (Trifluoroacetate salt)**
4030203
VVGGVMLGIIAGKNSGVDEAFFVLKQHH-VEYGSDHRFEAD
(Trifluoroacetate salt)

**Amyloid β-Protein (1-40) (mouse, rat)**
4035886
DAEFGHDSGFEVRHQKLVFFAEDVGSNK-GAIIGLMVGGVV

**(Arg$^{6}$)-Amyloid β-Protein (1-40)**
4075964
DAEFRRDSGYEVRHQKLVFFAEDVGSNK-GAIIGLMVGGVV
(English Mutation H6R)

**(Arg$^{13}$)-Amyloid β-Protein (1-40)**
4095736 NEW
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVV

**(Asn$^{7}$)-Amyloid β-Protein (1-40)**
4075963
DAEFRHNSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVV
(Tottori Mutation D7N)

**(Asn$^{23}$)-Amyloid β-Protein (1-40)**
4039929
DAEFRHDSGYEVHHQKLVFFAENVGSNK-GAIIGLMVGGVV
(Iowa Mutation D23N)

**Biotinyl-Amyloid β-Protein (1-40)**
4039814
Biotinyl-DAEFRHDSGYEVHHQKLVF-FAEDVGSNKGAIIGLMVGGVV

**Biotinyl-εAhx-Amyloid β-Protein (1-40)**
4090156
Biotinyl-εAhx-DAEFRHDSGYEVHHQKLV FFAEDVGSNKGAIIGLMVGGVV

**(Cys$^{0}$)-Amyloid β-Protein (1-40)**
4034867
CDAEFRHDSGYEVHHQKLVFFAEDVG-SNKGAIIGLMVGGVV

**(Cys$^{26}$)-Amyloid β-Protein (1-40)**
4060051
DAEFRHDSGYEVHHQKLVFFAEDVGCNK-GAIIGLMVGGVV

**(Cys$^{26}$)-Amyloid β-Protein (1-40) (Dimer)**
4089807
(DAEFRHDSGYEVHHQKLVFFAED-VGCNKGAIIGLMVGGVV)$_2$

**(Des-Glu$^{1}$)-Amyloid β-Protein (1-40)**
4091431
DAEFRHDSGYEVHHQKLVFFADVGSNK-GAIIGLMVGGVV
(Osaka Mutation E22Δ)

**(7-Diethylaminocoumarin-3-yl) carbonyl-Amyloid β-Protein (1-40)**
4048251
Deac-DAEFRHDSGYEVHHQKLVFFAED-VGSNKGAIIGLMVGGVV

**5-FAM-Amyloid β-Protein (1-40)**
4090152
Fluorescein-5-carbonyl-DAEFRHDS-GYEVHHQKLVFFAEDVGFAMSNK-GAIIGLMVGGVV

**FITC-β-Ala-Amyloid β-Protein (1-40)**
4049306
FITC-β-Ala-DAEFRHDSGYEVHHQKLVFF AEDVGSNKGAIIGLMVGGVV

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

**(Gln$^{9}$)-Amyloid β-Protein (1-40)**
4050901
DAEFRHDSQYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVV

**(Gln$^{22}$)-Amyloid β-Protein (1-40)**
4039928
DAEFRHDSGYEVHHQKLVFFAQDVGSNK-GAIIGLMVGGVV
(Dutch Mutation E22Q)

**(Gln$^{22}$,Asn$^{23}$)-Amyloid β-Protein (1-40)**
4089804
DAEFRHDSGYEVHHQKLVFFAQNVGSNK-GAIIGLMVGGVV
(Dutch/Iowa Mutation E22Q/D23N)

**(Gly$^{21}$)-Amyloid β-Protein (1-40)**
4039927
DAEFRHDSGYEVHHQKLVFFGEDVGSNK-GAIIGLMVGGVV
(Flemish Mutation A21G)

**(Gly$^{22}$)-Amyloid β-Protein (1-40)**
4035372
DAEFRHDSGYEVHHQKLVFFAGDVGSNK-GAIIGLMVGGVV
(Arctic Mutation E22G)

**(Lys$^{22}$)-Amyloid β-Protein (1-40)**
4049003
DAEFRHDSGYEVHHQKLVFFAKDVGSNK-GAIIGLMVGGVV
(Italian Mutation E22K)

**(Met(O)$^{35}$)-Amyloid β-Protein (1-40)**
4018327
DAEFRHDSGYEVHHQKLVFFAEDVGSNK GAIIGLM(O)VGGVV

**(Nle$^{35}$)-Amyloid β-Protein (1-40)**
4041778
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGL-Nle-VGGVV

**5-TAMRA-Amyloid β-Protein (1-40)**
4090154
5-TAMRA-DAEFRHDSGYEVHHQKLVF-FAEDVGSNKGAIIGLMVGGVV

**Tide Fluor™ 5WS-Amyloid β-Protein (1-40)**
4101189 NEW
Tide Fluor™ 5WS-DAEFRHDSGYEVH-HQKLVFFAEDVGSNKGAIIGLMVGGVV

**Tide Fluor™ 7WS-Amyloid β-Protein (1-40)**
4101191 NEW
Tide Fluor™ 7WS-DAEFRHDSGYEVH-HQKLVFFAEDVGSNKGAIIGLMVGGVV

**(Val$^{34}$)-Amyloid β-Protein (1-40)**
4089805
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGVMVGGVV
(Piedmont Mutation L34V)

# AMYLOID β-PROTEIN (25-35)

**Amyloid β-Protein (25-35)**
4030205
GSNKGAIIGLM

**Amyloid β-Protein (35-25)**
4030076
MLGIIAGKNSG

**Amyloid β-Protein (25-35) amide**
4028357
GSNKGAIIGLM-$NH_2$

**(Met(O)$^{35}$)-Amyloid β-Protein (25-35)**
4030351
GSNKGAIIGLM(O)

# AMYLOID β-PROTEIN FRAGMENTS

**Amyloid β-Protein (1-6)**
4052027 NEW
DAEFRH

**(Val$^{2}$)-Amyloid β-Protein (1-6)**
4104331 NEW
DVEFRH

**Amyloid β-Protein (1-6) amide**
4107157 NEW
DAEFRH-$NH_2$

**Acetyl-Amyloid β-Protein (1-6) amide**
4107158 NEW
Ac-DAEFRH-$NH_2$

**Amyloid β-Protein (1-11)**
4030331
DAEFRHDSGYE

**Amyloid β-Protein (1-12)**
4052029 NEW
DAEFRHDSGYEV

**Amyloid β-Protein (1-14)**
4082406
DAEFRHDSGYEVHH

**Amyloid β-Protein (1-15)**
4044506
DAEFRHDSGYEVHHQ

**Amyloid β-Protein (1-16)**
4030330
DAEFRHDSGYEVHHQK

**Amyloid β-Protein (1-24)**
4095720 NEW
DAEFRHDSGYEVHHQKLVFFAEDV

**Amyloid β-Protein (1-28)**
4025511
DAEFRHDSGYEVHHQKLVFFAEDVGSNK

**(Gln$^{11}$)-Amyloid β-Protein (1-28)**
4014779
DAEFRHDSGYQVHHQKLVFFAEDVGSNK

**(Gly$^{28}$, Cys$^{30}$)-Amyloid β-Protein (1-30) amide**
4037473
DAEFRHDSGYEVHHQKLVFFAEDVGSNG-GC-$NH_2$

**Amyloid β-Protein (1-37)**
4062195
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVG

**Amyloid β-Protein (1-38)**
4030204
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGG

**Amyloid β-Protein (1-39)**
4073896
DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGV

**Amyloid β-Protein (1-43)**
4018358
DAEFRHDSGYEVHHQKLVFFAEDVG-SNKGAIIGLMVGGVVIAT

# AMYLOID β-PROTEIN FRAGMENTS (CONTINUED)

**Amyloid β-Protein (1-46)**
4050409
DAEFRHDSGYEVHHQKLVFFAEDVG-SNKGAIIGLMVGGVVIATVIV

**Amyloid β-Protein (2-42)**
4036028 NEW
AEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVVIA

**Amyloid β-Protein (3-40)**
4046796 NEW
EFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVV

**(Pyr$^{3}$)-Amyloid β-Protein (3-40)**
4050043
<EFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVV

**Amyloid β-Protein (3-42)**
4090137 NEW
EFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVVIA

**(Pyr$^{3}$)-Amyloid β-Protein (3-42) (Ammonium salt)**
4029424
<EFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVVIA
(Ammonium salt)

**(Pyr$^{3}$)-Amyloid β-Protein (3-42) (Trifluoroacetate salt)**
4102120 NEW
<EFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVVIA
(Trifluoroacetate salt)

**Amyloid β-Protein (4-42)**
4090138 NEW
FRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVVIA

**Amyloid β-Protein (5-42)**
4041241 NEW
RHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVVIA

**Amyloid β-Protein (6-20)**
4044507
HDSGYEVHHQKLVFF

**Amyloid β-Protein (10-20)**
4030332
YEVHHQKLVFF

**Amyloid β-Protein (10-35)**
4037660
YEVHHQKLVFFAEDVGSNKGAIIGLM

**(Pyr$^{11}$)-Amyloid β-Protein (11-40)**
4037661
<EVHHQKLVFFAEDVGSNKGAIIGLMVG-GVV

**Amyloid β-Protein (11-42)**
4073393 NEW
EVHHQKLVFFAEDVGSNKGAIIGLMVG-GVVIA

**Amyloid β-Protein (12-28)**
4014778
VHHQKLVFFAEDVGSNK

**Acetyl-Amyloid β-Protein (15-20) amide**
4027103
Ac-QKLVFF-$NH_2$

**(Lys$^{15}$)-Amyloid β-Protein (15-21)**
4027870
KKLVFFA

**Amyloid β-Protein (16-20)**
4027102
KLVFF

**ent-[Amyloid β-Protein (20-16)]-β-Ala-D-Lys(ent-[Amyloid β-Protein (16-20)])**
4044533
ffvlk-β-Ala-k(ffvlk)

**Acetyl-(N-Me-Leu$^{17}$,N-Me-Phe$^{19}$)-Amyloid β-Protein (16-20) amide**
4095735 NEW
Ac-K(Me)LV(Me)FF-$NH_2$

**Amyloid β-Protein (16-22)**
4054257 NEW
KLVFFAE

**(Pro$^{18}$,Asp$^{21}$)-Amyloid β-Protein (17-21)**
4033200
LPFFD

# AMYLOID β-PROTEIN FRAGMENTS (CONTINUED)

**Acetyl-(Pro$^{18}$,Asp$^{21}$)-Amyloid β-Protein (17-21) amide**
4045708
Ac-LPFFD-$NH_2$

**Amyloid β-Protein (17-40)**
4034233
LVFFAEDVGSNKGAIIGLMVGGVV

**Amyloid β-Protein (20-29)**
4028923
FAEDVGSNKG

**Amyloid β-Protein (22-35)**
4025352
EDVGSNKGAIIGLM

**Amyloid β-Protein (29-40)**
4027727
GAIIGLMVGGVV

**Amyloid β-Protein (31-35)**
4041418
IIGLM

**Cys-Gly-His-Gly-Asn-Lys-Ser-Amyloid β-Protein (33-40)**
4045204
CGHGNKSGLMVGGVV

**Amyloid β-Protein (33-42)**
4037474
GLMVGGVVIA

**Cys-Gly-Lys-Lys-Gly-Amyloid β-Protein (35-40)**
4039438
CGKKGMVGGVV

**Amyloid β-Protein (36-38)**
4001973
VGG

**Amyloid β-Protein (37-39)**
4008279
GGV

# AMYLOID β/A4 PROTEIN PRECURSOR (APP) FRAGMENTS

**Acetyl-Amyloid β/A4 Protein Precursor$_{770}$ (96-110) (cyclized)**
4025760
Ac-NWCKRGRKQCKTHPH-$NH_2$
(Disulfide bond)

**Amyloid β/A4 Protein Precursor$_{770}$ (135-155)**
4027155
FLHQERMDVCETHLHWHTVAK

**(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{770}$ (667-675)**
4029477
SEVNLDAEF
(Swedish Double Mutation K670N / M671L)

**Amyloid β/A4 Protein Precursor$_{770}$ (667-676)**
4029479
SEVKMDAEFR

**(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{770}$ (667-676)**
4029475
SEVNLDAEFR
(Swedish Double Mutation K670N / M671L)

**Amyloid β/A4 Protein Precursor$_{770}$ (740-770)**
4048249
AAVTPEERHLSKMQQNGY-ENPTYKFFEQMQN

**Amyloid Precursor Frameshift Mutant C-Terminal Peptide**
4095739 NEW
RGRTSSKELA

# AMYLOID-LIKE PROTEIN

**APL1β28**
4074448
DELAPAGTGVSREAVSGLLIMGAGGG-SL

## AMYLOID BRI PEPTIDES

**Amyloid Bri Protein (1-23)**
4034208
EASNCFAIRHFENKFAVETLICS
(Disulfide bond)

**Amyloid Bri Protein (1-34)**
4037515
<EASNCFAIRHFENKFAVETLICSRT-VKKNIIEEN
(Disulfide bond)

**Amyloid Bri Protein (1-34) (reduced)**
4029311
<EASNCFAIRHFENKFAVETLICSRT-VKKNIIEEN

**Amyloid Bri Protein Precursor$_{277}$ (89-106)**
4034207
CGIKYIKDDVILNEPSAD

## AMYLOID DAN PEPTIDES

**Amyloid Dan Protein (1-34) (reduced)**
4035806
<EASNCFAIRHFENKFAVETLICFNL-FLNSQEKHY

## AMYLOID P-COMPONENT PEPTIDES

**Amyloid P Component (27-38) amide**
4030078
EKPLQNFTLCFR-NH$_2$

**Tyr-Amyloid P Component (27-38) amide**
4030347
YEKPLQNFTLCFR-NH$_2$

## NON-Aβ COMPONENT (α-SYNUCLEIN)

**α-Synuclein (34-45) (human)**
4107367 NEW
KEGVLYVGSKTK

**α-Synuclein (45-54) (human)**
4107365 NEW
KEGVVHGVAT

**α-Synuclein (61-95) (human)**
(Non-β-Amyloid Component of Alzheimer's Disease, NAC)
4026207
EQVTNVGGAVVTGVTAVAQKTVEGAG-SIAAATGFV

**α-Synuclein (67-78) (human)**
4107368 NEW
GGAVVTGVTAVA

**α-Synuclein (71-82) (human)**
4107366 NEW
VTGVTAVAQKTV

**α-Synuclein Binding Peptide**
4107364 NEW
Ac-KDGIVNGVKA-NH$_2$

# 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

**Mca-(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{770}$ (667-675)-Lys(Dnp)**
**4029476**
Mca-SEVNLDAEFK(Dnp)

**Mca-(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{770}$ (667-675)-Lys(Dnp) amide**
**4034744**
Mca-SEVNLDAEFK(Dnp)-$NH_2$

**Mca-Amyloid β/A4 Protein Precursor$_{770}$ (667-676)-Lys(Dnp)-Arg-Arg amide**
**4033759**
Mca-SEVKMDAEFRK(Dnp)RR-$NH_2$

**Mca-(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{770}$ (667-676)-Lys(Dnp)-Arg-Arg amide**
**4033760**
Mca-SEVNLDAEFRK(Dnp)RR-$NH_2$

**Arg-Glu(EDANS)-(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{770}$ (668-675)-Lys(DABCYL)-Arg**
**4033536**
RE(EDANS)VNLDAEFK(DABCYL)R

**Abz-Amyloid β/A4 Protein Precursor$_{770}$ (669-674)-EDDnp**
**4045325**
Abz-VKMDAE-EDDnp

**Abz-(Asn$^{670}$,Leu$^{671}$)-Amyloid β/A4 Protein Precursor$_{770}$ (669-674)-EDDnp**
**4045326**
Abz-VNLDAE-EDDnp

**Z-Val-Lys-Met-AMC**
**4017732**
Z-VKM-AMC

## β-SECRETASE INHIBITORS

**(Asn$^{670}$,Sta$^{671}$,Val$^{672}$)-Amyloid β/A4 Protein Precursor$_{770}$ (662-675)**
4029489
KTEEISEVN-Sta-VAEF

**OM99-2**
4034503
EVNL-psi[CHOHCH$_2$]AAEF

**Z-Leu-Leu-4,5-dehydro-Leu-aldehyde**
4027531
Z-LLΔL-CHO

## γ-SECRETASE SUBSTRATES

**Abz-Amyloid β/A4 Protein Precursor$_{770}$ (708-715)-Lys(Dnp)-D-Arg-D-Arg-D-Arg amide**
4043077
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**
4043236
N-Me-Abz-GGVVIATVK(Dnp)rrr-NH$_2$

## γ-SECRETASE INHIBITORS

**Z-Ile-Leu-aldehyde**
4048245
Z-IL-CHO

# HUMANIN

**Colivelin**
4050404
SALLRSIPAPAGASRLLLLTGEIDLP

**Humanin (human)**
4038276
MAPRGFSCLLLLTSEIDLPVKRRA

**(Gly$^{14}$)-Humanin (human)**
(S14G-Humanin (human))
4038277
MAPRGFSCLLLLTGEIDLPVKRRA

# PRION PEPTIDES

**Prion Protein (106-126) (human)**
4025090
KTNMKHMAGAAAAGAVVGGLG

**Prion Protein (106-126) (human) (scrambled))**
4033124
NGAKALMGGHGATKVMVGAAA

# FURTHER PEPTIDES FOR ALZHEIMER RESEARCH

**Ac-Asp-Glu-OH**
(NAAG)
4006232
Ac-DE

**rec Brain-Derived Neurotrophic Factor (human)**
(rec BDNF (human))
4038290

**L-Carnosine**
4030364

**CRF (6-33) (human, rat)**
4026679
ISLDLTFHLLREVLEMARAEQLAQQA-HS

**Galanin (human) (Acetate salt)**
4095896 NEW
GWTLNSAGYLLGPHAVGNHRSFSD-KNGLTS (Acetate salt)

**Galanin (human) (Trifluoroacetate salt)**
4013896
GWTLNSAGYLLGPHAVGNHRSFSD-KNGLTS (Trifluoroacetate salt)

**Galanin (mouse, rat)**
4030645
GWTLNSAGYLLGPHAIDNHRSFSD-KHGLT-$NH_2$

**Galanin (porcine)**
4009988
GWTLNSAGYLLGPHAIDNHRSFHD-KYGLA-$NH_2$

**Galanin (1-13)-Pro-Pro-(Ala-Leu-)$_2$Ala amide )**
(M40)
4030654
GWTLNSAGYLLGPPPALALA-$NH_2$

**(Des-Gly)-Glutathione-monoethyl ester (reduced)**
(GCEE, γ-GCE)
4026208
E(C-OEt)

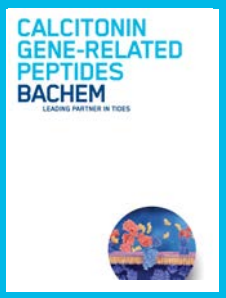
**H-Gly-Pro-Arg-OH**
4005002
GPR

**H-Ile-Phe-OH**
4001668
IF

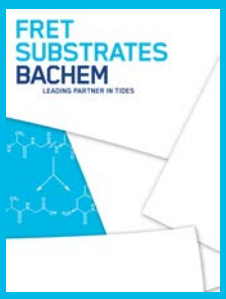
BACHEM

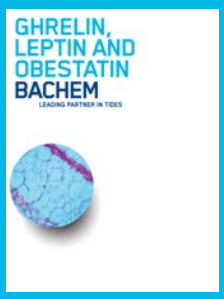
# PRODUCT BROCHURES

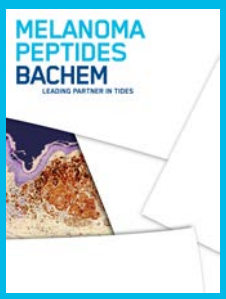


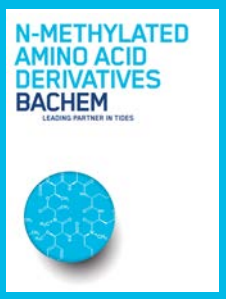




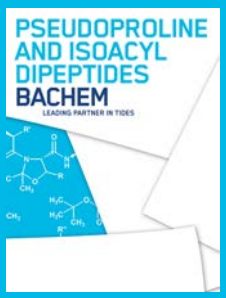










# FURTHER PEPTIDES FOR ALZHEIMER RESEARCH (CONTINUED)

**rec Leptin (human)**
4038283

**rec Leptin (mouse)**
4038284

**Leptin (116-130) amide (mouse) (Acetate salt)**
4100832 NEW
SCSLPQTSGLQKPES-$NH_2$
(Acetate salt)

**Leptin (116-130) amide (mouse) (Trifluoroacetate salt)**
4027666
SCSLPQTSGLQKPES-$NH_2$
(Trifluoroacetate salt)

**H-Leu-Ile-OH**
4002987
LI

**PACAP-38 (human, mouse, ovine, porcine, rat)**
4031157
HSDGIFTDSYSRYRKQMAVKKYLAAV-LGKRYKQRVKNK-$NH_2$

**Secretoneurin (mouse, rat)**
4037186
TNEIVEEQYTPQSLATLESVFQELG-KLTGPSNQ

**TRAF6 Peptide**
4095548 NEW
AAVALLPAVLLALLAPESAS-GPSEDPSVNFLK

**TRAF6 Control Peptide**
4095549 NEW
AAVALLPAVLLALLAPESASGASA-DASVNFLK

**WRW4**
4095537 NEW
WRWWWW-$NH_2$

**Dansyl-D-Ala-Gly-4-nitro-Phe-Gly-OH**
4050412
Dns-aGF($NO_2$)G

**H-Glu(EDANS)-Pro-Leu-Phe-Ala-Glu-Arg-Lys(DABCYL)-OH**
4050532
E(EDANS)PLFAERK(DABCYL)

**Acetyl-Calpastatin (184-210) (human)**
4027881
Ac-DPMSSTYIEELGKREVTIP-PKYRELLA-$NH_2$

**1,3-Bis-(Z-Leu-Leu)-diaminoacetone**
((Z-LL)$_2$ Ketone)
4095624 NEW
(Z-LL-$CH_2$)$_2$CO

**Z-Pro-Pro-aldehyde-dimethyl acetal**
4026718
Z-PP-CH(OMe)$_2$

# BIOCHEMICALS FOR ALZHEIMER RESEARCH

**Ac-DL-Asp-OH**
4036371

**N-Me-D-Asp-OH**
(NMDA)
4011485

**Ac-Cys-OH**
(NAC)
4031426

**H-D-Pen-OH**
(D-Penicillamine)
4032629

**H-Ser($PO_3H_2$)-OH**
(L-Phosphoserine, Dexfosfoserine)
4002875

**D-Cycloserine**
4030155

**L-trans-Epoxysuccinyl-Leu-3-methylbutylamide-ethyl ester**
(E-64d, Aloxistatin, Loxistatin, EP453
4027911

**sn-Glycero-3-phosphocholine**
(Choline alfoscerate, L-α-GPC, L-α-Lecithin)
4030680

**1-O-Hexadecyl-2-O-acetyl-sn-glycero-3-phosphocholine**
(PAF ($C_{16}$))
4006552

**Melatonin**
4008335

## AMYLOID BETA PEPTIDE

Amyloid beta peptide, computer illustration. This protein is the primary component of amyloid plagues in the brains of Alzheimer's patients.

(KEYSTONE/SCIENCE PHOTO LIBRARY)

| Custom Synthesis at Bachem | |
|---|---|
| • Quality | GMP and non-GMP quality<br>State of the art analytical capabilities |
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