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, 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 (LRP1), 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>
<thead>
<tr>
<th>Exchanged Position in APP</th>
<th>Exchanged Position in Aβ</th>
<th>Designation</th>
</tr>
</thead>
<tbody>
<tr>
<td>A673T</td>
<td>A2T</td>
<td>Icelandic</td>
</tr>
<tr>
<td>H677R</td>
<td>H6R</td>
<td>English</td>
</tr>
<tr>
<td>D678H</td>
<td>D7H</td>
<td>Taiwanese</td>
</tr>
<tr>
<td>D678N</td>
<td>D7N</td>
<td>Tottori</td>
</tr>
<tr>
<td>A692G</td>
<td>A21G</td>
<td>Flemish</td>
</tr>
<tr>
<td>E693D</td>
<td>E22Δ</td>
<td>Osaka</td>
</tr>
<tr>
<td>E693G</td>
<td>E22G</td>
<td>Arctic</td>
</tr>
<tr>
<td>E693Q</td>
<td>E22Q</td>
<td>Dutch</td>
</tr>
<tr>
<td>E693K</td>
<td>E22K</td>
<td>Italian</td>
</tr>
<tr>
<td>D694N</td>
<td>D23N</td>
<td>Iowa</td>
</tr>
<tr>
<td>L705V</td>
<td>L34V</td>
<td>Piedmont</td>
</tr>
</tbody>
</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.

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

P.M. Gorman and A. Chakrabartty

T. Hartmann

P.L. McGeer and E.G. McGeer

D.J. Selkoe

R. Cacabelos

J. Hardy and D.J. Selkoe

A.M. Palmer

B.A. Vicioso

D.A. Butterfield and C.B. Pocernich

I. Churcher and D. Beher

E. Gazit

K. Irie et al.

M.R. Nichols et al.

E.M. Sigurdsson

A.K. Tickler et al.

P. Westermark

E. Levy et al.

K. Takano et al.

T. Tomita and T. Iwatsubo

Y. J. Wang et al.

J.X. Chen and S.D. Yan

M.A. Findeis

V.H. Finder and R. Glockshuber

M. Li et al.

M. Tabaton and E. Tamagno

B. Van Broeck et al.

L.B. Hersh and D.W. Rogers
Y. Ohyagi
Intracellular amyloid beta-protein as a therapeutic target for treating Alzheimer’s disease.  

K.A. Bates et al.
Clearance mechanisms of Alzheimer’s amyloid-beta peptide: implications for therapeutic design and diagnostic tests.  

R. Deane et al.
Clearance of amyloid-beta peptide across the blood-brain barrier: implication for therapies in Alzheimer’s disease.  

F. Song et al.
Plasma biomarkers for mild cognitive impairment and Alzheimer’s disease.  

E. Bruno et al.
Lack of interaction between LRP1 and A2M polymorphisms for the risk of Alzheimer disease.  

H.J. Garringer et al.
Modeling familial British and Danish dementia.  

A. Kitamura and H. Kubota
Amyloid oligomers: dynamics and toxicity in the cytosol and nucleus.  

B. Liang et al.
Calpain activation promotes BACE1 expression, amyloid precursor protein processing, and amyloid plaque formation in a transgenic mouse model of Alzheimer disease.  

C.J. Lin et al.
Cu(ll) interaction with amyloid-beta peptide: a review of neuroactive mechanisms in AD brains.  

M.L. Moro et al.
Alzheimer’s disease and amyloid beta-peptide deposition in the brain: a matter of ‘aging’?  

K. Murakami et al.
The turn formation at positions 22 and 23 in the 42-mer amyloid beta peptide: the emerging role in the pathogenesis of Alzheimer’s disease.  

M.P. Murphy and H. LeVine 3rd
Alzheimer’s disease and the amyloid-beta peptide.  

J.F. Quinn et al.
A copper-lowering strategy attenuates amyloid pathology in a transgenic mouse model of Alzheimer’s disease.  

D.R. Thal et al.
Capillary cerebral amyloid angiopathy identifies a distinct APOE epsilon4-associated subtype of sporadic Alzheimer’s disease.  

N. Venketasubramanian et al.
Intergenetic differences in dementia epidemiology: global and Asia-Pacific perspectives.  

B. Zapala et al.
Humanins, the neuroprotective and cytoprotective peptides with anti-apoptotic and anti-inflammatory properties.  

C. Humpel
Identifying and validating biomarkers for Alzheimer’s disease.  
*Trends Biotechnol.* 29, 26-32 (2011)

S. Jawhar et al.
*J. Biol. Chem.* 286, 38825-38832 (2011)

R. Mayeux and N. Schupf
*Neurol. Aging* 32 Suppl 1, S10-S19 (2011)

B. Vincent and P. Govitrapong
Activation of the alpha-secretase processing of AbetaPP as a therapeutic approach in Alzheimer’s disease.  
*J. Alzheimers Dis.* 24 Suppl 2, 75-94 (2011)

W.T. Chen et al.

W. Danysz and C.G. Parsons
Alzheimer’s disease, beta-amyloid, glutamate, NMDA receptors and memantine - searching for the connections.  

R. Epis et al.
Alpha, beta- and gamma-secretases in Alzheimer’s disease.  

B.L. Kagan et al.
Antimicrobial properties of amyloid peptides.  

C.B. Pocernich and D.A. Butterfield
Elevation of glutathione as a therapeutic strategy in Alzheimer disease.  
REFERENCES

N.E. Pryor et al.
Unraveling the early events of amyloid-beta protein (Abeta) aggregation: Techniques for the determination of Abeta aggregate size.

N. Sun et al.
A survey of peptides with effective therapeutic potential in Alzheimer’s disease rodent models or in human clinical studies.

L.N. Zhao et al.
The toxicity of amyloid beta oligomers.

J.L. Crimins et al.
The intersection of amyloid beta and tau in glutamatergic synaptic dysfunction and collapse in Alzheimer’s disease.

R. Perez-Garmendia and G. Gevorkian

E. Drolle et al.
Atomic force microscopy to study molecular mechanisms of amyloid fibril formation and toxicity in Alzheimer’s disease.
Drug. Metab. Rev. 46, 207-223 (2014)

R.D. Johnson et al.
Structural evolution and membrane interactions of Alzheimer’s amyloid-beta peptide oligomers: new knowledge from single-molecule fluorescence studies.
Protein Sci. 23, 869-883 (2014)

M.P. Kummer and M.T. Heneka
Truncated and modified amyloid-beta species.
Alzheimers Res. Ther. 6, 28 (2014)

S. Schedin-Weiss et al.
The role of protein glycosylation in Alzheimer disease.

A. Martorana et al.
Cerebrospinal Fluid Aβ42 Levels: When Physiological Become Pathological State.
CNS Neurosci. Ther. 21, 921-925 (2015)

L. Montoliu-Gaya and S. Villegas
Protein structures in Alzheimer’s disease: The basis for rationale therapeutic design.

M.R. Nichols et al.
Biophysical comparison of soluble amyloid-beta(1-42) protofibrils, oligomers, and protofilaments.

D. Puzzo et al.
The keystone of Alzheimer pathogenesis might be sought in Abeta physiology.
Neuroscience 307, 26-36 (2015)

H.H. Jarosz-Griffiths et al.
Amyloid-beta Receptors: The Good, the Bad, and the Prion Protein.
J. Biol. Chem. 291, 3174-3183 (2016)

F.Z. Javaid et al.
Visual and Ocular Manifestations of Alzheimer’s Disease and Their Use as Biomarkers for Diagnosis and Progression.

T. Mohamed et al.
Amyloid cascade in Alzheimer’s disease: Recent advances in medicinal chemistry.

Z.X. Wang et al.
The Essential Role of Soluble Abeta Oligomers in Alzheimer’s Disease.

X. Zhou et al.
An overview on therapeutics attenuating amyloid beta level in Alzheimer’s disease: targeting neurotransmission, inflammation, oxidative stress and enhanced cholesterol levels.

Y. Zhou et al.
Detection of Abeta Monomers and Oligomers: Early Diagnosis of Alzheimer’s Disease.
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)**

4014447  
DAEFRHDSGYEVHHQKLFFFAEDVGSNK-GAIGLMVGGVIA

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

4045866  
DAEFRHDSGYEVHHQKLFFFAEDVGSNK-GAIGLMVGGVIA  
(Hydrochloride salt)

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

4089802 NEW  
DAEFRHDSGYEVHHQKLFFFAEDVGSNK-GAIGLMVGGVIA  
(Sodium salt)

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

4061966 NEW  
DAEFRHDSGYEVHHQKLFFFAEDVGSNK-GAIGLMVGGVIA  
(Trifluoroacetate salt)

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

4090148  
DAEFRHDSGYEVHHQKLFFFAEDVGSNK-GAIGLMVGGVIA

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

4064853  
AIAEGDSHVLKEGAYMEIFDVQGHVFG-KIFRVVDLGSNHNA

**Teplow’s Amyloid β-Protein (1-42) (scrambled II)**

4104168 NEW  
YHAGVDYKEFDEGAGAEHLAQKVIRG-GFVSDVSMIHINLF

**ent-Amyloid β-Protein (1-42)**

4037836  
daefrhdsgyehkvlffaaedvgsnkgaiiglmv-vggv (all-D peptide)

**Amyloid β-Protein (42-1)**

4027991  
AIVVGVMLGIAGKNSGVDEAFFVLKQH-HVEYGDHRFEAD

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

4107743 NEW  
AIVVGVMLGIAGKNSGVDEAFFVLKQH-HVEYGDHRFEAD

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

4035885  
DAEFRHDSGYEVHHQKLFFFAEDVGSNK-GAIGLMVGGVIA

**Teplow’s Amyloid β-Protein (1-42)**

4099631 NEW  
DAEFRHDSGYEVHHQKLFFFAEDVGSNK-GAIGLMVGGVIA

**Biotinyl-Amyloid β-Protein (1-42)**

4038795  
Biotinyl-DAEFRHDSGYEVHHQKLFFFAEDVGSNK-GAIGLMVGGVIA

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

4090155 NEW  
Biotinyl-εAhx-DAEFRHDSGYEVHHQKLFFFAEDVGSNK-GAIGLMVGGVIA

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

4037472  
CGKRDAEFRHDSGYEVHHQKLFFFAEDVGSNK-GAIGLMVGGVIA

**(Des-Glu)1-Amyloid β-Protein (1-42)**

4074277 NEW  
DAEFRHDSGYEVHHQKLFFFAEDVGSNK-GAIGLMVGGVIA  
(Des-Glu1)-Amyloid β-Protein (1-42)

**5-FAM-Amyloid β-Protein (1-42)**

4090151  
Fluorescein-5-carbonyl-DAEFRHDSGYEVHHQKLFFFAEDVGSNK-GAIGLMVGGVIA

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

4099694 NEW  
Fluorescein-5-carbonyl-AIAEGDSHV-LKEGAYMEIFDVQGHVFGKIFRVVDLGSNHNA
## Amyloid β-Protein (1-42)

### (Continued)

<table>
<thead>
<tr>
<th>Peptide Name</th>
<th>Catalog Number</th>
<th>Sequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>FITC-β-Ala-Amyloid β-Protein (1-42)</td>
<td>4033502</td>
<td>FITC-β-Ala-DAEFRHDSGYEVHHQKLVFF AEDVGSNKGAIIGLMVGVVIA</td>
</tr>
<tr>
<td>FITC-εAhx-Amyloid β-Protein (1-42)</td>
<td>4095738 NEW</td>
<td>FITC-β-Ala-DAEFRHDSGYEVHHQKLVFF AEDVGSNKGAIIGLMVGVVIA</td>
</tr>
<tr>
<td>(Gln22)-Amyloid β-Protein (1-42)</td>
<td>4050945 NEW</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGVVIA (Dutch Mutation E22Q)</td>
</tr>
<tr>
<td>(Gly21)-Amyloid β-Protein (1-42)</td>
<td>4039930 NEW</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDGVDGSNK-GAIIGLMVGVVIA (Flemish Mutation A21G)</td>
</tr>
<tr>
<td>(Gly23)-Amyloid β-Protein (1-42)</td>
<td>4035371</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGVVIA (Arctic Mutation E22G)</td>
</tr>
<tr>
<td>(Lys22)-Amyloid β-Protein (1-42)</td>
<td>4064438 NEW</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDGVDGSNK-GAIIGLMVGVVIA (Italian Mutation E22K)</td>
</tr>
<tr>
<td>(Met(O)35)-Amyloid β-Protein (1-42)</td>
<td>4041297</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGVVIA (Met(O2)35)</td>
</tr>
<tr>
<td>(Nleα)-Amyloid β-Protein (1-42)</td>
<td>4053225</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGL-Nle-VGVVIA</td>
</tr>
<tr>
<td>5-TAMRA-Amyloid β-Protein (1-42)</td>
<td>4090153</td>
<td>Fluorescein-5-carbonyl-DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGVVIA</td>
</tr>
</tbody>
</table>

## Amyloid β-Protein (1-40)

<table>
<thead>
<tr>
<th>Peptide Name</th>
<th>Catalog Number</th>
<th>Sequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amyloid β-Protein (1-40) (Trifluoroacetate salt)</td>
<td>4014442</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGVV (Trifluoroacetate salt)</td>
</tr>
<tr>
<td>Amyloid β-Protein (1-40) (Hydrochloride salt)</td>
<td>4038267</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGVV (Hydrochloride salt)</td>
</tr>
<tr>
<td>Amyloid β-Protein (1-40) (HFIP-treated)</td>
<td>4090147</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGVV (HFIP-treated)</td>
</tr>
<tr>
<td>Amyloid β-Protein (1-40) (scrambled)</td>
<td>4089803</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGVV (scrambled)</td>
</tr>
</tbody>
</table>
AMYLOID β-PROTEIN (1-40) (CONTINUED)

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

Amyloid β-Protein (1-40) amide  
**4095737**  
DAEFRHRNGEVHHQKLVFAEDVGSNK-GAIIGLMVGGVV-NH₂

Amyloid β-Protein (40-1) (Hydrochloride salt)  
**4095853 NEW**  
VVGGVMLGIIAGKNSGVDEAFFVLQKH-VEYSGDHREAD  
(Hydrochloride salt)

Amyloid β-Protein (40-1) (Trifluoroacetate salt)  
**4030203**  
VVGGVMLGIIAGKNSGVDEAFFVLQKH-VEYSGDHREAD  
(Trifluoroacetate salt)

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

(Arg⁶)-Amyloid β-Protein (1-40)  
**4075964**  
DAEFRHRNGEVHHQKLVFAEDVGSNK-GAIIGLMVGGVV  
(English Mutation H6R)

(Arg¹³)-Amyloid β-Protein (1-40)  
**4095736 NEW**  
DAEFRHRNGEVHHQKLVFAEDVGSNK-GAIIGLMVGGVV

(Asn⁷)-Amyloid β-Protein (1-40)  
**4075963**  
DAEFRHRNSGEEVHHQKLVFAEDVGSNK-GAIIGLMVGGVV  
(Tottori Mutation D7N)

(Asn²³)-Amyloid β-Protein (1-40)  
**4039929**  
DAEFRHRSGEEVHHQKLVFAENVGSNK-GAIIGLMVGGVV  
(Iowa Mutation D23N)

Biotinyl-Amyloid β-Protein (1-40)  
**4039814**  
Biotinyl-DAEFRHRNGEVHHQKLVFAEDVGSNK-GAIIGLMVGGVV

Biotinyl-εAhx-Amyloid β-Protein (1-40)  
**4090156**  
Biotinyl-εAhx-DAEFRHRNGEVHHQKLVFAEDVGSNK-GAIIGLMVGGVV

(Amyloid β-Protein (1-40)  
**4034867**  
CDAEFRHRNGEVHHQKLVFAEDVGSNK-GAIIGLMVGGVV

(Cys²⁸)-Amyloid β-Protein (1-40) (Dimer)  
**4089807**  
(DAEFRHRNGEVHHQKLVFAEDVGSNK-GAIIGLMVGGVV)²

(Des-Glu¹)-Amyloid β-Protein (1-40)  
**4091431**  
DAEFRHRNGEVHHQKLVFAEDVGSNK-GAIIGLMVGGVV  
(Osaka Mutation E22Δ)

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

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

FITC-β-Ala-Amyloid β-Protein (1-40)  
**4049306**  
FITC-β-Ala-DAEFRHRNGEVHHQKLVFAEDVGSNK-GAIIGLMVGGVV
<table>
<thead>
<tr>
<th>Mutation/Glyceylation</th>
<th>Amyloid β-Protein (1-40)</th>
<th>Catalog Number</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Gln&lt;sup&gt;9&lt;/sup&gt;)</td>
<td>Amyloid β-Protein (1-40)</td>
<td>4050901</td>
<td>DAEFRHDSQYEVHQQKLVFEDVSNK-GAIQLMVGVV</td>
</tr>
<tr>
<td>(Gln&lt;sup&gt;22&lt;/sup&gt;)</td>
<td>Amyloid β-Protein (1-40)</td>
<td>4039928</td>
<td>DAEFRHDSQYEVHQQKLVFQDVGNNK-GAIQLMVGVV (Dutch Mutation E22Q)</td>
</tr>
<tr>
<td>(Gln&lt;sup&gt;22&lt;/sup&gt;, Asn&lt;sup&gt;23&lt;/sup&gt;)</td>
<td>Amyloid β-Protein (1-40)</td>
<td>4089804</td>
<td>DAEFRHDSQYEVHQQKLVFQHNVGNK-GAIQLMVGVV (Dutch/Iowa Mutation E22Q/D23N)</td>
</tr>
<tr>
<td>(Gly&lt;sup&gt;21&lt;/sup&gt;)</td>
<td>Amyloid β-Protein (1-40)</td>
<td>4039927</td>
<td>DAEFRHDSQYEVHQQKLVFQGEDVSNK-GAIQLMVGVV (Flemish Mutation A21G)</td>
</tr>
<tr>
<td>(Gly&lt;sup&gt;22&lt;/sup&gt;)</td>
<td>Amyloid β-Protein (1-40)</td>
<td>4035372</td>
<td>DAEFRHDSQYEVHQQKLVFQAGDVGNNK-GAIQLMVGVV (Arctic Mutation E22G)</td>
</tr>
<tr>
<td>(Lys&lt;sup&gt;22&lt;/sup&gt;)</td>
<td>Amyloid β-Protein (1-40)</td>
<td>4049003</td>
<td>DAEFRHDSQYEVHQKLVFQAKDVGNNK-GAIQLMVGVV (Italian Mutation E22K)</td>
</tr>
<tr>
<td>(Met(O)&lt;sup&gt;39&lt;/sup&gt;)</td>
<td>Amyloid β-Protein (1-40)</td>
<td>4018327</td>
<td>DAEFRHDSQYEVHQQKLVFEDVSNK-GAIQLM(O)VGVV</td>
</tr>
<tr>
<td>(Nle&lt;sup&gt;35&lt;/sup&gt;)</td>
<td>Amyloid β-Protein (1-40)</td>
<td>4041778</td>
<td>DAEFRHDSQYEVHQKLVFEDVSNK-GAIQL-Nle-VGVV</td>
</tr>
<tr>
<td>5-TAMRA</td>
<td>Amyloid β-Protein (1-40)</td>
<td>4090154</td>
<td>5-TAMRA-DAEFRHDSQYEVHQQKLVF-FAEDVSNK-GAIQLMVGVV</td>
</tr>
<tr>
<td>Tide Fluor&lt;sup&gt;™&lt;/sup&gt; 5WS</td>
<td>Amyloid β-Protein (1-40)</td>
<td>4101189</td>
<td>Tide Fluor&lt;sup&gt;™&lt;/sup&gt; 5WS-DAEFRHDSQYEVHQQKLVFEDVSNK-GAIQLMVGVV</td>
</tr>
<tr>
<td>Tide Fluor&lt;sup&gt;™&lt;/sup&gt; 7WS</td>
<td>Amyloid β-Protein (1-40)</td>
<td>4101191</td>
<td>Tide Fluor&lt;sup&gt;™&lt;/sup&gt; 7WS-DAEFRHDSQYEVHQQKLVFEDVSNK-GAIQLMVGVV</td>
</tr>
<tr>
<td>(Val&lt;sup&gt;34&lt;/sup&gt;)</td>
<td>Amyloid β-Protein (1-40)</td>
<td>4089805</td>
<td>DAEFRHDSQYEVHQKLVFEDVSNK-GAIQLGVMVGVV (Piedmont Mutation L34V)</td>
</tr>
<tr>
<td>Peptide Name</td>
<td>CAS Number</td>
<td>Sequence</td>
<td></td>
</tr>
<tr>
<td>--------------</td>
<td>-------------</td>
<td>----------</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (25-35)</td>
<td>4030205</td>
<td>GSNKGAIIGLM</td>
<td></td>
</tr>
<tr>
<td>(Met(O)35)-Amyloid β-Protein (25-35)</td>
<td>4030351</td>
<td>GSNKGAIIGLM(0)</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (35-25)</td>
<td>4030076</td>
<td>MLGIIAGKNSG</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (25-35) amide</td>
<td>4028357</td>
<td>GSNKGAIIGLM-NH₂</td>
<td></td>
</tr>
<tr>
<td>(Val²⁵)-Amyloid β-Protein (1-6)</td>
<td>4104331</td>
<td>DAEFRH</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (1-6) amide</td>
<td>4107157</td>
<td>DAEFRH-NH₂</td>
<td></td>
</tr>
<tr>
<td>Acetyl-Amyloid β-Protein (1-6) amide</td>
<td>4107158</td>
<td>Ac-DAEFRH-NH₂</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (1-11)</td>
<td>4030331</td>
<td>DAEFRHDSGYE</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (1-12)</td>
<td>4052029</td>
<td>DAEFRHDSGYEV</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (1-14)</td>
<td>4082406</td>
<td>DAEFRHDSGYEVHH</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (1-15)</td>
<td>4044506</td>
<td>DAEFRHDSGYEVHHQ</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (1-16)</td>
<td>4030330</td>
<td>DAEFRHDSGYEVHHQK</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (1-24)</td>
<td>4095720</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDV</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (1-28)</td>
<td>4025511</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK</td>
<td></td>
</tr>
<tr>
<td>(Gln¹⁷)-Amyloid β-Protein (1-28)</td>
<td>4014779</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK</td>
<td></td>
</tr>
<tr>
<td>(Gly²⁸, Cys³⁰)-Amyloid β-Protein (1-30) amide</td>
<td>4037473</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNG-GC-NH₂</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (1-37)</td>
<td>4062195</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVG</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (1-38)</td>
<td>4030204</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGG</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (1-39)</td>
<td>4073896</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGV</td>
<td></td>
</tr>
<tr>
<td>Amyloid β-Protein (1-43)</td>
<td>4018358</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIIGLMVGGVVIAT</td>
<td></td>
</tr>
</tbody>
</table>
AMYLOID β-PROTEIN FRAGMENTS (CONTINUED)

Amyloid β-Protein (1-46)  
4050409  
DAEFRHDSGYEVHHQKLFFAEDVGSNK-GAIILMVGGVVIA

Amyloid β-Protein (2-42)  
4036028 NEW  
AEFRHDSGYEVHHQKLFFAEDVGSNK-GAIILMVGGVVIA

Amyloid β-Protein (3-40)  
4046798 NEW  
EFRHDGVEHQQKLFFAEDVGSNK-GAIILMVGGVV

Amyloid β-Protein (3-42)  
4090137 NEW  
EFRHDGVEHQQKLFFAEDVGSNK-GAIILMVGGVVIA

Amyloid β-Protein (3-42) (Pyr⁷)-Amyloid β-Protein (3-40)  
4050043  
<EFRHDGVEHQQKLFFAEDVGSNK-GAIILMVGGVV

Amyloid β-Protein (3-42) (Pyr⁷)-Amyloid β-Protein (3-42) (Ammonium salt)  
4029424  
<EFRHDGVEHQQKLFFAEDVGSNK-GAIILMVGGVVIA (Ammonium salt)

Amyloid β-Protein (3-42) (Pyr⁷)-Amyloid β-Protein (3-42) (Trifluoroacetate salt)  
4102120 NEW  
<EFRHDGVEHQQKLFFAEDVGSNK-GAIILMVGGVVIA (Trifluoroacetate salt)

Amyloid β-Protein (4-42)  
4090138 NEW  
FRHDSGYEVHHQKLFFAEDVGSNK-GAIILMVGGVVIA

Amyloid β-Protein (5-42)  
4041241 NEW  
RHDSGYEVHHQKLFFAEDVGSNK-GAIILMVGGVVIA

Amyloid β-Protein (5-42) (Pro¹⁸,Asp²¹)-Amyloid β-Protein (17-21)  
40033200  
LPFFD

Amyloid β-Protein (6-20)  
4044507  
HDGGYEHHQKLFF

Amyloid β-Protein (10-20)  
4030332  
YEVHHQKLFF

Amyloid β-Protein (10-35)  
4037660  
YEVHHQKLFFAEDVGSNK-GAIILM

Amyloid β-Protein (11-40)  
4037661  
<EVHQQKLFFAEDVGSNK-GAIILMVG-GVV

Amyloid β-Protein (11-42)  
4073393 NEW  
EVHQQKLFFAEDVGSNK-GAIILMVG-GVVIA

Amyloid β-Protein (12-28)  
4014778  
VHHQKLFFAEDVGSNK

Acetyl-Amyloid β-Protein (15-20) amide  
4027103  
Ac-QKLFF-NH₂

(Amm)Amyloid β-Protein (15-21)  
4027870  
KQLFF

Amyloid β-Protein (16-20)  
4027102  
KLFF

Amyloid β-Protein (16-22)  
4054257 NEW  
KLVVFAE

Acetyl-(N-Me-Leu¹⁷,N-Me-Phe¹⁹)-Amyloid β-Protein (16-20) amide  
4095735 NEW  
Ac-K(Me)LV(Me)FF-NH₂

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

Acetyl-(N-Me-Leu¹⁷,N-Me-Phe¹⁹)-Amyloid β-Protein (16-20) amide  
4095735 NEW  
Ac-K(Me)LV(Me)FF-NH₂

Amyloid β-Protein (17-21)  
40033200  
LPFFD
AMYLOID β-PROTEIN FRAGMENTS (CONTINUED)

**Acetyl-(Pro\textsuperscript{18},Asp\textsuperscript{21})-Amyloid β-Protein (17-21) amide**

4045708
Ac-LPFFD-NH\textsubscript{2}

**Amyloid β-Protein (17-40)**

4034233
LVFAEDVGSNKGAIIGLMVGGVV

**Amyloid β-Protein (20-29)**

4028923
FAEDVGSNK

**Amyloid β-Protein (22-35)**

4025352
EDVGSNKGAIIGLM

**Amyloid β-Protein (29-40)**

4027727
GAIIGLMVG

**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
GLMVG

**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,\textsubscript{770} (96-110) (cyclized)
4025760
Ac-NWCKRGRKQCKTHPH-NH\textsubscript{2}
(Disulfide bond)

Amyloid β/A4 Protein Precursor,\textsubscript{770} (135-155)
4027155
FLHQERMDVCETHLHWHTVAK

(Asn\textsuperscript{670},Leu\textsuperscript{671})-Amyloid β/A4 Protein Precursor,\textsubscript{770} (667-675)
4029477
SEVNLDAEF
(Swedish Double Mutation K670N / M671L)

Amyloid β/A4 Protein Precursor,\textsubscript{770} (667-676)
4029479
SEVKMDAEFR

(Asn\textsuperscript{670},Leu\textsuperscript{671})-Amyloid β/A4 Protein Precursor,\textsubscript{770} (667-676)
4029475
SEVNLDAEFR
(Swedish Double Mutation K670N / M671L)

Amyloid β/A4 Protein Precursor,\textsubscript{770} (740-770)
4048249
AAVTPEERHLSKMQQNGY-ENPTYKFFEQMQN

Amyloid Precursor Frameshift Mutant C-Terminal Peptide
4095739 NEW
RGRTSSKELA

AMYLOID-LIKE PROTEIN

APL1β28
4074448
DELAPAGTVSREAVSGLLIMGAGGG-SL
## Amyloid Bri Peptides

<table>
<thead>
<tr>
<th>Protein Name</th>
<th>Formula</th>
<th>MW (Da)</th>
<th>CAS Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amyloid Bri Protein (1-23)</td>
<td>EASNCFAIRHFENKFAVETLICS (Disulfide bond)</td>
<td>4034208</td>
<td></td>
</tr>
<tr>
<td>Amyloid Bri Protein (1-34)</td>
<td>&lt;EASNCFAIRHFENKFAVETLICSRT-VKKNIEEN (Disulfide bond)</td>
<td>4037515</td>
<td></td>
</tr>
<tr>
<td>Amyloid Bri Protein Precursor277</td>
<td>&lt;EASNCFAIRHFENKFAVETLICSRT-VKKNIEEN</td>
<td>4029311</td>
<td></td>
</tr>
</tbody>
</table>

## Amyloid Dan Peptides

<table>
<thead>
<tr>
<th>Protein Name</th>
<th>Formula</th>
<th>MW (Da)</th>
<th>CAS Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amyloid Dan Protein (1-34)</td>
<td>amide</td>
<td>4035806</td>
<td></td>
</tr>
<tr>
<td>Tyr-Amyloid P Component (27-38)</td>
<td>amide</td>
<td>4033047</td>
<td></td>
</tr>
</tbody>
</table>

## Amyloid P-Component Peptides

<table>
<thead>
<tr>
<th>Protein Name</th>
<th>Formula</th>
<th>MW (Da)</th>
<th>CAS Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amyloid P Component (27-38) amide</td>
<td>EKPLQNFTLCFR-NH$_2$</td>
<td>4030078</td>
<td></td>
</tr>
<tr>
<td>Tyr-Amyloid P Component (27-38) amide</td>
<td>YEKPLQNFTLCFR-NH$_2$</td>
<td>4030347</td>
<td></td>
</tr>
</tbody>
</table>

## Non-αβ Component (α-Synuclein)

<table>
<thead>
<tr>
<th>Protein Name</th>
<th>Formula</th>
<th>MW (Da)</th>
<th>CAS Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>α-Synuclein (34-45) (human)</td>
<td>KEGVLYVGSKTK</td>
<td>4107367 NEW</td>
<td></td>
</tr>
<tr>
<td>α-Synuclein (45-54) (human)</td>
<td>KEGVVHGVAT</td>
<td>4107365 NEW</td>
<td></td>
</tr>
<tr>
<td>α-Synuclein (61-95) (human)</td>
<td>EQVTNVGGAVVTGVTAVAQKTVEGAG-SIAAATGFV</td>
<td>4026207</td>
<td></td>
</tr>
<tr>
<td>α-Synuclein (67-78) (human)</td>
<td>GGAVVGTGVTAVA</td>
<td>4107368 NEW</td>
<td></td>
</tr>
<tr>
<td>α-Synuclein (71-82) (human)</td>
<td>VTGVTAVAQKTVEGAG</td>
<td>4107366 NEW</td>
<td></td>
</tr>
<tr>
<td>α-Synuclein Binding Peptide</td>
<td>Ac-KDGIVNGVKA-NH$_2$</td>
<td>4107364 NEW</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Protein Name</th>
<th>Formula</th>
<th>MW (Da)</th>
<th>CAS Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>α-Synuclein (67-78) (human)</td>
<td>GGAVVGTGVTAVA</td>
<td>4107368 NEW</td>
<td></td>
</tr>
<tr>
<td>α-Synuclein (71-82) (human)</td>
<td>VTGVTAVAQKTVEGAG</td>
<td>4107366 NEW</td>
<td></td>
</tr>
<tr>
<td>α-Synuclein Binding Peptide</td>
<td>Ac-KDGIVNGVKA-NH$_2$</td>
<td>4107364 NEW</td>
<td></td>
</tr>
</tbody>
</table>
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

<table>
<thead>
<tr>
<th>Peptide</th>
<th>CAS Number</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<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>
<td>4029476</td>
<td></td>
</tr>
<tr>
<td>Mca-SEVNLDAEFK(Dnp)</td>
<td></td>
<td></td>
</tr>
<tr>
<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) amide</td>
<td>4034744</td>
<td></td>
</tr>
<tr>
<td>Mca-SEVNLDAEFK(Dnp)-NH₂</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mca-Amyloid β/A4 Protein Precursor&lt;sub&gt;770&lt;/sub&gt; (669-676)-Lys(Dnp)-Arg-Arg amide</td>
<td>4033759</td>
<td></td>
</tr>
<tr>
<td>Mca-SEVKMDAEFRK(Dnp)RR-NH₂</td>
<td></td>
<td></td>
</tr>
<tr>
<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; (667-676)-Lys(Dnp)-Arg-Arg amide</td>
<td>4033760</td>
<td></td>
</tr>
<tr>
<td>Mca-SEVNLDAEFK(Dnp)-Arg-Arg amide</td>
<td></td>
<td></td>
</tr>
<tr>
<td>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</td>
<td>4033536</td>
<td></td>
</tr>
<tr>
<td>RE(EDANS)VNLDAEFK(DABCYL)R</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abz-Amyloid β/A4 Protein Precursor&lt;sub&gt;770&lt;/sub&gt; (669-674)-EDDnp</td>
<td>4045325</td>
<td></td>
</tr>
<tr>
<td>Abz-VNLDAE-EDDnp</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Z-Val-Lys-Met-AMC</td>
<td>4017732</td>
<td></td>
</tr>
<tr>
<td>Z-VKM-AMC</td>
<td></td>
<td></td>
</tr>
<tr>
<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>
<td>4045326</td>
<td></td>
</tr>
<tr>
<td>Abz-VNLDAE-EDDnp</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Z-Val-Lys-Met-AMC</td>
<td>4017732</td>
<td></td>
</tr>
<tr>
<td>Z-VKM-AMC</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**β-SECRETASE INHIBITORS**

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

4029489
KTEEISEVN-Sta-VAEF

OM99-2
4034503
EVNL-psi[CHOHCH<sub>2</sub>]AAEF

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

**γ-SECRETASE SUBSTRATES**

Abz-Amyloid β/A4 Protein Precursor<sub>770</sub> (708–715)-Lys(Dnp)-D-Arg-D-Arg-D-Arg amide
4043077
Abz-GGVVIATVK(Dnp)rrr-NH<sub>2</sub>

N-Me-Abz-Amyloid β/A4 Protein Precursor<sub>770</sub> (708–715)-Lys(Dnp)-D-Arg-D-Arg-D-Arg amide
4043236
N-Me-Abz-GGVVIATVK(Dnp)rrr-NH<sub>2</sub>

**γ-SECRETASE INHIBITORS**

Z-Ile-Leu-aldehyde
4048245
Z-IL-CHO
## HUMANIN

<table>
<thead>
<tr>
<th>Peptide</th>
<th>CAS Number</th>
<th>Sequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colivelin</td>
<td>4050404</td>
<td>SALLRSIPAPASRLLLLTGEIDLP</td>
</tr>
<tr>
<td>Humanin (human)</td>
<td>4038276</td>
<td>MAPRGFSCLLLLTGEIDLPVKRRA</td>
</tr>
<tr>
<td>(Gly&lt;sup&gt;14&lt;/sup&gt;)-Humanin (human)</td>
<td>4038277</td>
<td>MAPRGFSCLLLLTGEIDLPVKRRA</td>
</tr>
</tbody>
</table>

## PRION PEPTIDES

<table>
<thead>
<tr>
<th>Peptide</th>
<th>CAS Number</th>
<th>Sequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prion Protein (106-126) (human)</td>
<td>4025090</td>
<td>KTNMKHMAGAAAGAVVGLG</td>
</tr>
<tr>
<td>Prion Protein (106-126) (human) (scrambled)</td>
<td>4033124</td>
<td>NGAKALMGHHGATKVMGAAA</td>
</tr>
</tbody>
</table>

## FURTHER PEPTIDES FOR ALZHEIMER RESEARCH

<table>
<thead>
<tr>
<th>Peptide</th>
<th>CAS Number</th>
<th>Sequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ac-Asp-Glu-OH (NAAG)</td>
<td>4006232</td>
<td>Ac-DE</td>
</tr>
<tr>
<td>rec Brain-Derived Neurotrophic Factor (human) (rec BDNF (human))</td>
<td>4038290</td>
<td></td>
</tr>
<tr>
<td>L-Carnosine</td>
<td>4030364</td>
<td></td>
</tr>
<tr>
<td>CRF (6-33) (human, rat)</td>
<td>4026679</td>
<td>ISLDTLTFRHLEVARARAEQQQA-HS</td>
</tr>
<tr>
<td>Galanin (human) (Acetate salt)</td>
<td>4095896 NEW</td>
<td>GWTLNSAGYLLGPHAVGNHRSFSD-KNGLT-NH&lt;sub&gt;2&lt;/sub&gt;</td>
</tr>
<tr>
<td>Galanin (mouse, rat)</td>
<td>4030645</td>
<td>GWTLNSAGYLLGPHAVGNHRSFSD-KHGLT-NH&lt;sub&gt;2&lt;/sub&gt;</td>
</tr>
<tr>
<td>Galanin (porcine)</td>
<td>4009988</td>
<td>GWTLNSAGYLLGPHAVGNHRSFHD-KYGLA-NH&lt;sub&gt;2&lt;/sub&gt;</td>
</tr>
<tr>
<td>Galanin (1-13)-Pro-Pro-(Ala-Leu)-&lt;sub&gt;2&lt;/sub&gt;Ala amide) (M40)</td>
<td>4030654</td>
<td>GWTLNSAGYLLGPPPALAL-NH&lt;sub&gt;2&lt;/sub&gt;</td>
</tr>
<tr>
<td>(Des-Gly)-Glutathione-monoethyl ester (reduced) (GCEE, γ-GCE)</td>
<td>4026208</td>
<td>E(C-OEt)</td>
</tr>
<tr>
<td>H-Gly-Pro-Arg-OH</td>
<td>4005002</td>
<td>GPR</td>
</tr>
<tr>
<td>H-Ile-Phe-OH</td>
<td>4001668</td>
<td>IF</td>
</tr>
</tbody>
</table>
FURTHER PEPTIDES FOR ALZHEIMER RESEARCH (CONTINUED)

rec Leptin (human) 4038283
rec Leptin (mouse) 4038284

Leptin (116-130) amide (mouse) (Acetate salt)
4100832 NEW
SCSLPQTSGLQKPESEQ-NH₂
(Acetate salt)

Leptin (116-130) amide (mouse) (Trifluoroacetate salt)
4027666
SCSLPQTSGLQKPESEQ-NH₂
(Trifluoroacetate salt)

H-Leu-Ile-OH 4002987
LI

PACAP-38 (human, mouse, ovine, porcine, rat) 4031157
HSDGIFTQSYRKYOMAVKKYLAAV-LGKRYKQRVKNK-NH₂

Secretoneurin (mouse, rat) 4037186
TNEIIVEEQYTPQSLATLESVFQELG-KLTGPSNQ

TRAF6 Peptide 4095548 NEW
AAVALLPAVLLALLAPESAGPSEDPSVNFLK

TRAF6 Control Peptide 4095549 NEW
AAVALLPAVLLALLAPESAGASA-DASVNFLK

WRW4 4095537 NEW
WRWWW-NH₂

Dansyl-D-Ala-Gly-4-nitro-Phe-Gly-OH 4050412
Dns-aGF(NO₂)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-DPMSSTYIEELKRVETP-PKYRELLA-NH₂

1,3-Bis-(Z-Leu-Leu)-diaminoacetone ([Z-LL]₂ Ketone) 4095624 NEW
(Z-LL-CH₃)₂CO

Z-Pro-Pro-aldehyde-dimethyl acetal 4026718
Z-PP-CH(OMe)₂
<table>
<thead>
<tr>
<th>Biochemical</th>
<th>Code</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ac-DL-Asp-OH</td>
<td>4036371</td>
</tr>
<tr>
<td>N-Me-D-Asp-OH (NMDA)</td>
<td>4011485</td>
</tr>
<tr>
<td>Ac-Cys-OH (NAC)</td>
<td>4031426</td>
</tr>
<tr>
<td>H-D-Pen-OH (D-Penicillamine)</td>
<td>4032629</td>
</tr>
<tr>
<td>H-Ser(PO₃H₂)-OH (L-Phosphoserine, Dextosfoserine)</td>
<td>4002875</td>
</tr>
<tr>
<td>D-Cycloserine</td>
<td>4030155</td>
</tr>
<tr>
<td>L-trans-Epoxysuccinyl-Leu-3-methylbutylamide-ethyl ester (E-64d, Aloixistatin, Loxistatin, EP453)</td>
<td>4027911</td>
</tr>
<tr>
<td>sn-Glycero-3-phosphocholine (Choline alfoscerate, L-α-GPC, L-α-Lecithin)</td>
<td>4030680</td>
</tr>
<tr>
<td>1-O-Hexadecyl-2-O-acetyl-sn-glycero-3-phosphocholine (PAF (C₁₆))</td>
<td>4006552</td>
</tr>
<tr>
<td>Melatonin</td>
<td>4008335</td>
</tr>
</tbody>
</table>
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)
<table>
<thead>
<tr>
<th>Custom Synthesis at Bachem</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 |