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 become a major health and social problem in the developed countries with an increasing proportion of older people. 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 26 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

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.

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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.

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Genetic factors may contribute as well to the late onset of AD. Increased susceptibility is associated with the expression of different apolipoprotein E (ApoE) isoforms due to the polymorphism in the APOE gene on chromosome 19. In the central nervous system, ApoE has been implicated in growth and repair during development or after injury. Carriers of the APOEε4 allele show a higher risk in developing the disease than carriers of the other two possible alleles APOEε2 and APOEε3. The ApoEε4 effect seems to be dose-dependent since individuals with two of these alleles seem to be at two-fold higher risk to develop the disease than those with one allele. Polymorphisms of the α2-macroglobulin gene on chromosome 12 and the gene coding low-density lipoprotein receptor-related protein 1 (LRP-
Amyloid Peptides

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 (GSK-3) 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. 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 also gained approval in the United States by the FDA. 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)

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<td>DAEFRHDSGYQVHHQKLVFFAEDVGSNK</td>
<td>95%</td>
</tr>
<tr>
<td>(Gly¹⁸, Cys³⁵)-Amyloid β-Protein (1-30) amide</td>
<td>H-6386</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GC-NH₂</td>
<td>95%</td>
</tr>
<tr>
<td>Amyloid β-Protein (1-38)</td>
<td>H-2966</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GAIILMVGG</td>
<td>95%</td>
</tr>
<tr>
<td>Amyloid β-Protein (1-43)</td>
<td>H-1586</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GSNKGAIIGLMVGGVVIAT</td>
<td>95%</td>
</tr>
<tr>
<td>Amyloid β-Protein (1-46)</td>
<td>H-6406</td>
<td>DAEFRHDSGYEVHHQKLVFFAEDVGSNK-GSNKGAIIGLMVGGVVIATVIV</td>
<td>95%</td>
</tr>
<tr>
<td>(Pyr³)-Amyloid β-Protein (3-42)</td>
<td>H-4796</td>
<td>&lt;EFRHDSGYEVHHQKLVFFAEDVGSNK-GAIILMVGGVIA</td>
<td>95%</td>
</tr>
<tr>
<td>(Gly²⁸, Cys³⁰)-Amyloid β-Protein (10-35) amide</td>
<td>H-4796</td>
<td>&lt;EFRHDSGYEVHHQKLVFFAEDVGSNK-GAIILMVGGVIA</td>
<td>95%</td>
</tr>
<tr>
<td>(Gly²⁸, Cys³⁰)-Amyloid β-Protein (10-35) amide</td>
<td>H-6384</td>
<td>YEVHHQKLVFFAEDVGSNK-GAIILMVGGVIA</td>
<td>95%</td>
</tr>
<tr>
<td>Amyloid β-Protein (10-20)</td>
<td>H-1388</td>
<td>YEVHHQKLVFFAEDVGSNK-GAIILMVGGVIA</td>
<td>95%</td>
</tr>
<tr>
<td>(Pyr¹¹)-Amyloid β-Protein (11-40)</td>
<td>H-6382</td>
<td>&lt;EVHHQKLVFFAEDVGSNK-GAIILMVGGVIA</td>
<td>95%</td>
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<tr>
<td>Amyloid β-Protein (12-28)</td>
<td>H-7910</td>
<td>VHHQKLVFFAEDVGSNK-GAIILMVGGVIA</td>
<td>95%</td>
</tr>
<tr>
<td>Acetyl-Amyloid β-Protein (15-20) amide</td>
<td>H-3684</td>
<td>Ac-QKLVFF-NH₂</td>
<td>95%</td>
</tr>
<tr>
<td>(Lys¹³⁵)-Amyloid β-Protein (15-21)</td>
<td>H-4062</td>
<td>KKLVFFA</td>
<td>95%</td>
</tr>
</tbody>
</table>
AMYLOID β-PROTEIN FRAGMENTS (CONTINUED)

<table>
<thead>
<tr>
<th>Fragment Description</th>
<th>Peptide Sequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gly-Amyloid β-Protein (15-25)-Gly-ε-aminocaproyl(-Lys)₆</td>
<td>GQKLVFFAEDVGG-εAhx-KKKKK</td>
</tr>
<tr>
<td>H-Leu-Leu-Val-Phe-OH (Leu¹⁶)-Amyloid β-Protein (16-19)</td>
<td>H-3945</td>
</tr>
<tr>
<td>ent-[Amyloid β-Protein (20-16)]-β-Ala-D-Lys(ent-[Amyloid β-Protein (16-20)])</td>
<td>H-6074</td>
</tr>
<tr>
<td>Acetyl-(Pro¹⁸,Asp²¹)-Amyloid β-Protein (17-21) amide</td>
<td>H-6138</td>
</tr>
<tr>
<td>Acetyl-(Pro¹⁸,Asp²¹)-Amyloid β-Protein (17-21) amide</td>
<td>Ac-LPFFD-NH₂</td>
</tr>
<tr>
<td>Amyloid β-Protein (20-29)</td>
<td>H-3808</td>
</tr>
<tr>
<td>FAEDVGSNKG</td>
<td></td>
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<tr>
<td>Amyloid β-Protein (22-35)</td>
<td>H-1976</td>
</tr>
<tr>
<td>EDVGSNKGAIIGLM</td>
<td></td>
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<tr>
<td>Amyloid β-Protein (29-40)</td>
<td>H-3984</td>
</tr>
<tr>
<td>GAIIGLMVGGVV</td>
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</tr>
<tr>
<td>Propionyl-Amyloid β-Protein (31-34) amide</td>
<td>H-4124</td>
</tr>
<tr>
<td>Propionyl-IIILMVGGVV</td>
<td></td>
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<tr>
<td>Amyloid β-Protein (31-35)</td>
<td>H-5866</td>
</tr>
<tr>
<td>IIGLM</td>
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<tr>
<td>Cys-Gly-His-Gly-Asn-Lys-Ser-Amyloid β-Protein (33-40)</td>
<td>H-6364</td>
</tr>
<tr>
<td>CGHNKSLMVGGVV</td>
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<tr>
<td>Cys-Gly-Lys-Lys-Gly-Amyloid β-Protein (33-40)</td>
<td>H-6372</td>
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<tr>
<td>CGKKGLMVGGVV</td>
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<tr>
<td>Amyloid β-Protein (33-42)</td>
<td>H-5572</td>
</tr>
<tr>
<td>GLMVGGVIA</td>
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<tr>
<td>Cys-Gly-Lys-Lys-Gly-Amyloid β-Protein (35-60)</td>
<td>H-6376</td>
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<tr>
<td>CGKKGMVGGVV</td>
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<tr>
<td>Cys-Gly-Lys-Lys-Gly-Amyloid β-Protein (36-42)</td>
<td>H-6378</td>
</tr>
<tr>
<td>CGKKGVMVGVV</td>
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<tr>
<td>H-Val-Gly-Gly-OH</td>
<td>H-5270</td>
</tr>
<tr>
<td>VGG</td>
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</tr>
<tr>
<td>H-Gly-Gly-Val-OH</td>
<td>H-3500</td>
</tr>
<tr>
<td>GGV</td>
<td></td>
</tr>
<tr>
<td>Methoxysuccinyl-Val-Val-Ile-Ala-pNA</td>
<td>L-1745</td>
</tr>
<tr>
<td>(Methoxysuccinyl-Amyloid β-Protein (39-42)-p-nitroanilide)</td>
<td></td>
</tr>
</tbody>
</table>
AMYLOID β/A4 PROTEIN PRECURSOR (APP) FRAGMENTS

**Acetyl-Amyloid β/A4 Protein Precursor**

- **H-2232**
  - Ac-NWCKRGRKQCKTHPH-NH₂
  - (Disulfide bond)

- **Amyloid β/A4 Protein Precursor**
  - **H-3726**
    - FLHQERMDVCETHLHWHTVAK
  - (human, mouse, rat)

- **Amyloid β/A4 Protein Precursor**
  - **H-1608**
    - RERMS
  - (human, mouse, rat)

- **Amyloid β/A4 Protein Precursor**
  - **H-1850**
    - ISYGNDALMP
  - (Asn670, Leu671)-Amyloid β/A4 Protein Precursor
  - **H-4834**
    - SEVNLDAEFR
    - (Swedish Double Mutation K670N / M671L)

- **Amyloid β/A4 Protein Precursor**
  - **H-4838**
    - SEVKVDAEFR
  - (Swedish Double Mutation K670N / M671L)

- **Amyloid β/A4 Protein Precursor**
  - **H-6216**
    - AAVTPEERHLSKMQNGY-ENPTYKFFEQMQN

**Amyloid β/A4 Protein Precursor**

- **H-6842**
  - SEVKMDAEFR
  - (Asn670-Leu671)-Amyloid β/A4 Protein Precursor

**Amyloid-like Protein**

- **APLβ25**
  - **H-7302**
    - DELAPAGTGVSREAVSGLIMGAGG

- **APLβ27**
  - **H-7304**
    - DELAPAGTGVSREAVSGLIMGAGGS

- **APLβ28**
  - **H-7306**
    - DELAPAGTGVSREAVSGLIMGAGG-GL
## Amyloid Bri Peptides

<table>
<thead>
<tr>
<th>Peptide</th>
<th>Sequence</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amyloid Bri Protein (1-23)</td>
<td>H-5052</td>
<td>EASNCFARHFNKFAVETLICS (Disulfide bond)</td>
</tr>
<tr>
<td>Amyloid Bri Protein (1-34)</td>
<td>H-5526</td>
<td>&lt;EASNCFARHFNKFAVETLICSRT-VKKNIEEN (Disulfide bond)</td>
</tr>
<tr>
<td>Amyloid Bri Protein (1-34) (reduced)</td>
<td>H-5728</td>
<td>&lt;EASNCFARHFNKFAVETLICSRT-VKKNIEEN</td>
</tr>
<tr>
<td>Amyloid Bri Protein Precursor (89-106)</td>
<td>H-5048</td>
<td>CGIKYIKDDVLNEPSAD</td>
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</tbody>
</table>

## Amyloid Dan Peptides

<table>
<thead>
<tr>
<th>Peptide</th>
<th>Sequence</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amyloid Dan Protein (1-34)</td>
<td>H-5528</td>
<td>&lt;EASNCFARHFNKFAVETLICFNL-FLNSQEKHY (Disulfide bond)</td>
</tr>
<tr>
<td>Amyloid Dan Protein (1-34) (reduced)</td>
<td>H-5298</td>
<td>&lt;EASNCFARHFNKFAVETLICFNL-FLNSQEKHY</td>
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</tbody>
</table>

## Amyloid P-Component Peptides

<table>
<thead>
<tr>
<th>Peptide</th>
<th>Sequence</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amyloid P Component (27-38) amide</td>
<td>H-2942</td>
<td>EKPLQNFTLCFR-NH₂</td>
</tr>
<tr>
<td>Tyr-Amyloid P Component (27-38) amide</td>
<td>H-2944</td>
<td>YEKPLQNFTLCFR-NH₂</td>
</tr>
<tr>
<td>Amyloid P Component (33-38) amide</td>
<td>H-2946</td>
<td>FTLCFR-NH₂</td>
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</tbody>
</table>

## Non-αβ Component

<table>
<thead>
<tr>
<th>Peptide</th>
<th>Sequence</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-αβ Component of Alzheimer’s Disease (α-Synuclein (61-95) (human))</td>
<td>H-2598</td>
<td>EQVTNVGGAVVTAVATVAQKTEGAG-SIAATGFV</td>
</tr>
</tbody>
</table>
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
Amyloid Peptides

**β-SECRETASE SUBSTRATES**

- **DABCYL-(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid β/A4 Protein Precursor<sub>770</sub> (661-675)-EDANS**
  - M-2445
  - DABCYL-IKTEEISEVNLDAEF-EDANS

- **H-Lys-Thr-Glu-Glu-Ile-Ser-Glu-Val-Lys-Met-pNA**
  - (APP<sub>770</sub> (662-671)-pNA)
  - L-1905
  - KTEEISEVKM-pNA

- **Mca-(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid β/A4 Protein Precursor<sub>770</sub> (667-674)-Dap(Dnp)**
  - M-2425
  - Mca-SEVNLADE-Dpa

- **DABCYL-(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid β/A4 Protein Precursor<sub>770</sub> (667-675)-EDANS**
  - M-2435
  - DABCYL-SEVNLADEAF-EDANS

- **Mca-(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid β/A4 Protein Precursor<sub>770</sub> (667-675)-Lys(Dnp)**
  - M-2420
  - Mca-SEVNLADEFK(Dnp)

- **Mca-(Asn<sup>670</sup>,Leu<sup>671</sup>)-Amyloid β/A4 Protein Precursor<sub>770</sub> (667-675)-Lys(Dnp) amide**
  - M-2485
  - Mca-SEVNLADEFK(Dnp)-NH<sub>2</sub>

- **Lys(Dabsyl)-(Asn<sup>670</sup> Leu<sup>671</sup>)-Amyloid β/A4 Protein Precursor<sub>770</sub> (667-676)-Gln-Lucifer Yellow**
  - M-2570
  - K(Dabsyl)SEVNLADEFRQ-Lucifer Yellow

- **Mca-Amyloid β/A4 Protein Precursor<sub>770</sub> (667-676)-Lys(Dnp)-Arg-Arg amide**
  - M-2460
  - Mca-SEVKMDAEFK(Dnp)RR-NH<sub>2</sub>
**β-SECRETASE INHIBITORS**

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

N-1815

Ac-VML-psi[CHOHCH$_2$]VAEF

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

H-4848

KTEEISEVN-Sta-VAEF

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

N-1825

ELDL-psi[CHOHCH$_2$]AAEF


N-1920

ELDL-psi[CHOHCH$_2$]AVEFGGrrrrrrrr

OM99-2

H-5108

EVNL-psi[CHOHCH$_2$]AAEF

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

N-1590

Z-LLΔL-CHO

**γ-SECRETASE SUBSTRATES**

Abz-Amyloid β/A4 Protein Precursor$_{770}$ (708-715)-Lys(Dnp)-D-Arg-D-Arg-D-Arg amide

M-2540

Abz-GGVVIATVK(Dnp)rrr-NH$_2$

N-Me-Abz-Amyloid β/A4 Protein Precursor$_{770}$ (708-715)-Lys(Dnp)-D-Arg-D-Arg-D-Arg amide

M-2555

N-Me-Abz-GGVVIATVK(Dnp)rrr-NH$_2$

**γ-SECRETASE INHIBITORS**

L-685,458

H-5106

Boc-F-psi[CHOHCH$_2$]FLF-NH$_2$

3,5-Difluorophenylacetyl-Ala-Phg-OMe

N-1890

Z-Ile-Leu-aldehyde

N-1895

Z-IL-CHO

Z-Leu-Leu-Nle-aldehyde

N-1695

Z-LL-Nle-CHO
HUMANIN

Colivelin
H-6336
SALLRSIPAPAGSRLLLTTGEIDLP

Humanin (human)
H-5574
MAPRGFSCLLLLTSEIDLPVKRRA

(Gly14)-Humanin (human)
H-5576
MAPRGFSCLLLTTGEIDLPVKRRA

VARIABLE RELATED PRODUCTS

(trans, trans)-1-Bromo-2,5-bis-(3-carboxy-4-hydroxy)styrilbenzene
(BSB)
Q-2690

L-Carnosine
G-1250

CRF (6-33) (human, rat)
H-3456
ISLDLTFLHLLREVLEMAEQLAQQA-HS

H-D-Pen-OH
(D-Penicillamine)
F-4235

Phenserine
Q-1860

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

Z-Val-Lys-Met-AMC
I-1625
Z-VKM-AMC
ALZHEIMER CELL CULTURE, SEM

Coloured SEM of Alzheimer’s disease culture cells.
Alzheimer’s disease culture cells. Coloured scanning electron micrograph (SEM) of cells used in Alzheimer’s disease research. These cells have been genetically engineered to produce amyloid precursor protein (APP), which in turn forms the protein amyloid. Plaques of amyloid in the brain are a major pathological feature of Alzheimer’s disease. These cells are cultured from a nerve cancer (neuroblastoma), and have shorter and more numerous processes (dendrites and axons) than healthy nerve cells. Alzheimer’s is a brain-wasting disease common in the elderly. It causes confusion, memory loss, personality changes and eventually death.

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