

PRODUCT INFORMATION



Amyloid- β (17-40) Peptide (human) (trifluoroacetate salt)

Item No. 27414

Formal Name:	L-leucyl-L-valyl-L-phenylalanyl-L-phenylalanyl-L-alanyl-L- α -glutamyl-L- α -aspartyl-L-valylglycyl-L-seryl-L-asparaginyll-L-lysylglycyl-L-alanyl-L-isoleucyl-L-L-isoleucylglycyl-L-leucyl-L-methionyl-L-valylglycylglycyl-L-valyl-L-valine	H—Leu—Val—Phe—Phe—Ala—Glu—Asp—Val—Gly—Ser—Asn—Lys—Gly—Ala—Ile—Ile—Gly—Leu—Met—Val—Gly—Gly—Val—Val—OH
Synonym:	A β (17-40)	
MF:	C ₁₁₀ H ₁₇₈ N ₂₆ O ₃₁ S • XCF ₃ COOH	
FW:	2,392.8	
Purity:	≥95%	
Supplied as:	A solid	
Storage:	-20°C	
Stability:	≥4 years	

Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Amyloid- β (A β) (17-40) peptide (human) (trifluoroacetate salt) is supplied as a solid. A stock solution may be made by dissolving the A β (17-40) peptide (human) (trifluoroacetate salt) in the solvent of choice, which should be purged with an inert gas. A β (17-40) peptide (human) (trifluoroacetate salt) is soluble in the organic solvent formic acid at a concentration of approximately 1 mg/ml.

Description

A β (17-40) is a 24-residue fragment of the A β protein that is formed *via* post-translational processing of amyloid precursor protein (APP) by α - and γ -secretases.¹ *In vitro*, A β (17-40) polymerizes into lattice-like structures that do not bind thioflavin T. It inhibits interactions between fibrinogen and biotinylated A β (1-42) (Item No. 27410) as well as between cholesterol and apolipoprotein E (ApoE) with IC₅₀ values of 13.4 and 25 μ M, respectively.^{2,3} A β (17-40) increases the rate of A β (1-40) (Item No. 21617) aggregation *in vitro* when used at an equimolar concentration.⁴ A β (17-40) (25 μ M) induces cell death in cultured rat hippocampal neurons.⁵

References

1. Näslund, J., Jensen, M., Tjernberg, L.O., *et al.* The metabolic pathway generating p3, an A β -peptide fragment, is probably non-amyloidogenic. *Biochem. Biophys. Res. Commun.* **204**(2), 780-787 (1994).
2. Zamolodchikov, D., Berk-Rauch, H.E., Oren, D.A., *et al.* Biochemical and structural analysis of the interaction between β -amyloid and fibrinogen. *Blood* **128**(8), 1144-1151 (2016).
3. Yao, Z.X. and Papadopoulos, V. Function of β -amyloid in cholesterol transport: A lead to neurotoxicity. *FASEB J.* **16**(12), 1677-1679 (2002).
4. Liu, R., McAllister, C., Lyubchenko, Y., *et al.* Residues 17-20 and 30-35 of beta-amyloid play critical roles in aggregation. *J. Neurosci. Res.* **75**(2), 162-171 (2004).
5. Pike, C.J., Overman, M.J., and Cotman, C.W. Amino-terminal deletions enhance aggregation of β -amyloid peptides *in vitro*. *J. Biol. Chem.* **270**(41), 23895-23898 (1995).

WARNING

THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

SAFETY DATA

This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the complete Safety Data Sheet, which has been sent via email to your institution.

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CAYMAN CHEMICAL

1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA

PHONE: [800] 364-9897
[734] 971-3335

FAX: [734] 971-3640

CUSTSERV@CAYMANCHEM.COM
WWW.CAYMANCHEM.COM