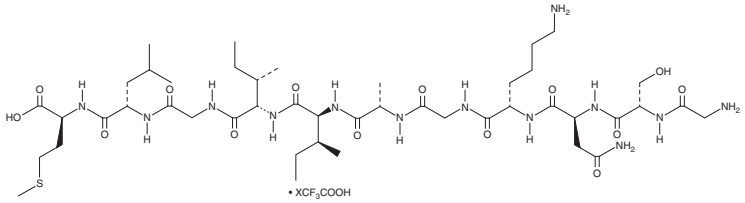
PRODUCT INFORMATION

Amyloid-β (25-35) Peptide (human) (trifluoroacetate salt)

Item No. 24155

Formal Name:	glycyl-L-seryl-L-asparaginyl-L-lysylglycyl-L-alanyl-L-isoleucyl-L-isoleucylglycyl-L-leucyl-L-methionine, trifluoroacetate salt
Synonym:	Aβ (25-35)
MF:	$C_{45}H_{81}N_{13}O_{14}S \bullet XCF_3COOH$
FW:	1,060.3
Purity:	≥95%
Supplied as:	A lyophilized powder
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 (25-35) (Aβ (25-35)) peptide (human) (trifluoroacetate salt) is supplied as a lyophilized powder. A stock solution may be made by dissolving the Aβ (25-35) peptide (human) (trifluoroacetate salt) in water. The solubility of Aβ (25-35) peptide (human) (trifluoroacetate salt) in water is approximately 1 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Aβ (25-35) is an 11-residue fragment of the Aβ protein that retains the physical and biological characteristics of the full length peptide.[1] It forms fibrils that react to thioflavin T and Congo red and are organized in a cross-β arrangement of β-strands similar to Aβ (1-40) (Item No. 21617) and Aβ (1-42) (Item No. 20574) fibrils.[2,3] Aggregated Aβ (25-35) decreases the viability of rat adrenal PC12 cells.[2] It also decreases the viability of primary rat cortical neurons at concentrations ranging from 1 nM to 30 µM.[4] *In vivo*, intracerebral injection of Aβ (25-35) (20 nmol) in rats induces lesions of neuronal and tissue loss.[5] Aggregated Aβ (25-35) administered intracerebroventricularly to rats induces learning and memory impairments in the Y-maze, novel object recognition, and contextual fear conditioning tests.[6]

References

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2. Hughes, E., Burke, R.M., and Doig, A.J. Inhibition of toxicity in the β-amyloid peptide fragment β-(25–35) using N-methylated derivatives. *J. Biol. Chem.* **275(33)**, 25109-25115 (2000).
3. Wei, G., Jewett, A.I., and Shea, J.E. Structural diversity of dimers of the Alzheimer amyloid-β(25-35) peptide and polymorphism of the resulting fibrils. *Phys. Chem. Chem. Phys.* **12(14)**, 3622-3629 (2010).
4. Wang, Y., Liu, L., Hu, W., *et al.* Mechanism of soluble beta-amyloid 25-35 neurotoxicity in primary cultured rat cortical neurons. *Neurosci. Lett.* **618**, 72-76 (2016).
5. Rush, D.K., Aschmies, S., and Merriman, M.C. Intracerebral β-amyloid(25-35) produces tissue damage: Is it neurotoxic? *Neurobiol. Aging* **13(5)**, 591-594 (1992).
6. Tsunekawa, H., Noda, Y., Mouri, A., *et al.* Synergistic effects of selegiline and donepezil on cognitive impairment induced by amyloid beta (25-35). *Behav. Brain Res.* **190(2)**, 224-232 (2008).

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