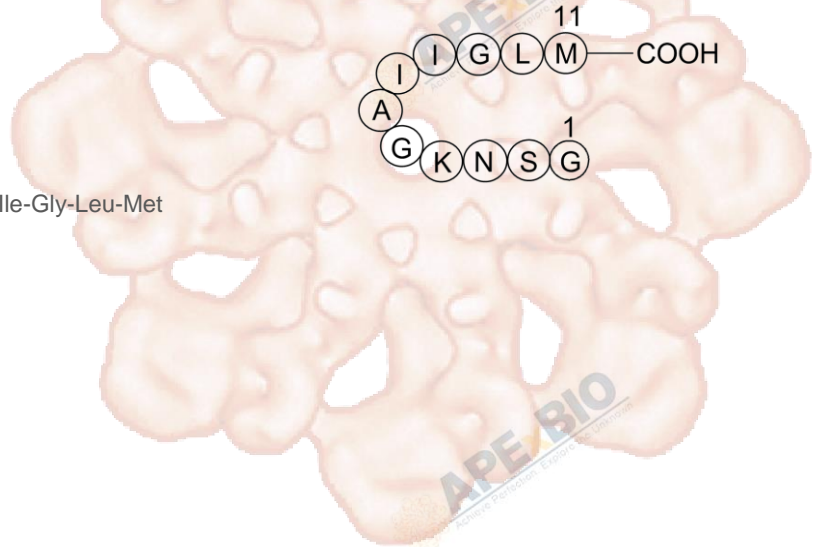


Product Data Sheet

Amyloid Beta-peptide (25-35) (human)

Cat. No.:	A1039
CAS No.:	131602-53-4
Formula:	C45H81N13O14S
M.Wt:	1060.27
Synonyms:	Gly-Ser-Asn-Lys-Gly-Ala-Ile-Ile-Gly-Leu-Met
Target:	Neuroscience
Pathway:	Amyloid β
Storage:	Desiccate at -20°C



Solvent & Solubility

insoluble in EtOH; insoluble in H₂O; ≥ 106 mg/mL in DMSO

In Vitro

Preparing Stock Solutions	Solvent	Mass		
		1mg	5mg	10mg
	Concentration			
	1 mM	0.9432 mL	4.7158 mL	9.4316 mL
	5 mM	0.1886 mL	0.9432 mL	1.8863 mL
	10 mM	0.0943 mL	0.4716 mL	0.9432 mL

Please refer to the solubility information to select the appropriate solvent.

Biological Activity

Shortsummary

Functional domain of A β

IC₅₀ & Target

In Vitro

Cell Viability Assay

Cell Line:	Embryonic rat hippocampal cells
Preparation method:	The solubility of this peptide in sterile water is >0.5mg/ml. Stock solution should be split and stored at -80°C for several months.
Reacting conditions:	20 μ M, 6 hours
Applications:	To investigate the involvement of the tau phosphorylation kinases in A β (25–

35)-induced tau phosphorylation, the level of each kinase was determined after A β (25–35) (20 μ M) exposure for various periods. GSK-3 α did not show a significant change in response to A β (25–35), whereas MAP kinase decreased to ~ 60% of the control after 6h A β (25–35) exposure, when tau was phosphorylated maximally. TPK I/GSK-3 β rapidly increased in response to A β (25–35), reaching a maximum (2.2-fold the control) at 6 h.

Animal experiment

Animal models:	Male Charles River Wistar rats
Dosage form:	Intraperitoneal injection, 400 mg/kg
Applications:	A statistically significant decrease in basal ACh release (-28%) was detected one week after the injection of A β (25–35). The effect persisted for only two weeks. K ⁺ -stimulated ACh release was similarly affected by the treatment. A β (25–35) treatment induced a statistically significant decrease in the stimulated release on day 14 after lesioning (-45%).
Other notes:	Please test the solubility of all compounds indoor, and the actual solubility may slightly differ with the theoretical value. This is caused by an experimental system error and it is normal.

In Vivo

Product Citations

1. Guo CC, Jiao CH, et al. "Silencing of LncRNA BDNF-AS attenuates A β (25-35)-induced neurotoxicity in PC12 cells by suppressing cell apoptosis and oxidative stress." *Neurol Res.* 2018 Jun 14;1-10.PMID:29902125

See more customer validations on www.apexbt.com.

References

- [1] Takashima A, Honda T, Yasutake K, et al. Activation of tau protein kinase I/glycogen synthase kinase-3 β by amyloid β peptide (25–35) enhances phosphorylation of tau in hippocampal neurons. *Neuroscience research*, 1998, 31(4): 317-323.
- [2] Giovannelli L, Casamenti F, Scali C, et al. Differential effects of amyloid peptides β -(1–40) and β -(25–35) injections into the rat nucleus basalis. *Neuroscience*, 1995, 66(4): 781-792.

Caution

FOR RESEARCH PURPOSES ONLY.

NOT FOR HUMAN, VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

Specific storage and handling information for each product is indicated on the product datasheet. Most APEX[®]BIO products are stable under the recommended conditions. Products are sometimes shipped at a temperature that differs from the recommended storage temperature. Short-term storage of many products are stable in the short-term at temperatures that differ from that required for long-term storage. We ensure that the product is shipped under conditions that will maintain the quality of the reagents. Upon receipt of the product, follow the storage recommendations on the product data sheet.



APExBIO Technology

www.apexbt.com

7505 Fannin street, Suite 410, Houston, TX 77054.

Tel: +1-832-696-8203 | Fax: +1-832-641-3177 | Email: info@apexbt.com

