β-Amyloid (1-16)

Cat. No.:	HY-P1466
CAS No.:	131580-10-4
Molecular Formula:	$C_{84}H_{119}N_{27}O_{28}$
Molecular Weight:	1955.01
Sequence:	Asp-Ala-Glu-Phe-Arg-His-Asp-Ser-Gly-Tyr-Glu-Val-His-His-Gln-Lys
Sequence Shortening:	DAEFRHDSGYEVHHQK
Target:	Amyloid-β
Pathway:	Neuronal Signaling
Storage:	Sealed storage, away from moisture Powder -80°C 2 years -20°C 1 year * The compound is unstable in solutions, freshly prepared is recommended.

BIOLOGICAL ACTIVITY

Description	β-Amyloid (1-16) is a β-Amyloid protein fragment involved in metal binding. Beta-amyloid is a peptide that forms amyloid plaques in the brains of Alzheimer's disease (AD) patients.
IC_{50} & Target	Amyloid-β[1]
In Vivo	β-amyloid (1-16) fragment is considered as valid models to examine the contribution of the key histidine residues (His , His in mouse and His , His , His in human fragments) to the Ab–Cu^{2+} interaction. Oxidation targets for β-Amyloid (1-16) are the histidine residues coordinated to the metal ions. Copper is bound to Aβ in senile plaque of Alzheimer's disease with β-Amyloid (1-16) taking part in the coordination of the Cu^{2+} ions. Cu^{2+} and Zn^{2+} are linked with the neurotoxicity of -Amyloid and free radical damage[1]. β-amyloid (1-16) is the minimal amino acidic sequence display a Cu coordination mode which involves three Histidines (His6, His13 and His14). β-amyloid (1-16) is supposed to be involved in metal binding[2]. Human β-amyloid interacts with zinc ions through its metal-binding domain 1-16. The C-tails of the two polypeptide chains of the rat A β(1-16) dimer are oriented in opposite directions to each other, which hinders the assembly of rat Aβ dimers into oligomeric aggregates. Thus, the differences in the structure of zinc-binding sites of human and rat β-Amyloid (1-16), their ability to form regular cross-monomer bonds, and the orientation of their hydrophobic C-tails could be responsible for the resistance of rats to Alzheimer's disease[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Kowalik-Jankowska T, et al. Coordination abilities of the 1-16 and 1-28 fragments of beta-amyloid peptide towards copper(II) ions: a combined potentiometric and spectroscopic study. J Inorg Biochem. 2003 Jul 1;95(4):270-82.

[2]. Minicozzi V, et al. Identifying the minimal copper- and zinc-binding site sequence in amyloid-beta peptides. J Biol Chem. 2008 Apr 18;283(16):10784-92.

[3]. Istrate AN, et al. NMR solution structure of rat aβ(1-16): toward understanding the mechanism of rats' resistance to Alzheimer's disease. Biophys J. 2012 Jan 4;102(1):136-43.

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA