Atabecestat

Cat. No.: HY-109052

CAS No.: 1200493-78-2

Molecular Formula: $C_{18}H_{14}FN_5OS$

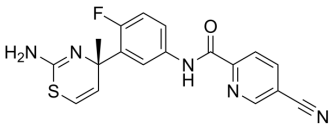
Molecular Weight: 367.4

Target: Beta-secretase

Pathway: Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (680.46 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.7218 mL	13.6091 mL	27.2183 mL
5 mM	0.5444 mL	2.7218 mL	5.4437 mL
10 mM	0.2722 mL	1.3609 mL	2.7218 mL

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

BIOLOGICAL ACTIVITY

Description

Atabecestat (JNJ-54861911) is a potent brain-penetrant and orally active β-site amyloid precursor protein cleaving enzyme 1 (BACE1) inhibitor, achieves robust and high CSF Aβ reduction. Atabecestat s tolerated and displays a sustained pharmacokinetic (PK) and pharmacodynamic (PD) characteristics. Atabecestat has the potential for Alzheimer's Disease treatment[1].

IC_{50} & Target

BACE1

In Vivo

Atabecestat (100 and 300 mg/kg; p.o. once daily for 3 days) reduces the human Aβ levels in mice[2].

Atabecestat (300 mg/kg; p.o. once) inhibits the exacerbation of vascular abnormalities in APPPS1 mice with 3D6 treatment[2].

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MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	5-week-old APPPS1 mice[2]

Dosage:	100 and 300 mg/kg
Administration:	Oral gavage; 100 and 300 mg/kg; once daily for 3 days
Result:	Reduced the level of human $A\beta_{1-40}$ and $A\beta_{1-42}$ levels in the brain of APPPS1 mice at a dose of 300 mg/kg and resulted in less reduction of human Aβ levels at 24 h with a dose of 100 mg/kg.

REFERENCES

[1]. Janssens J, et al. Passive immunotherapy with a novel antibody against 3pE-modified Aβ demonstrates potential for enhanced efficacy and favorable safety in combination with BACE inhibitor treatment in plaque-depositing mice. Neurobiol Dis. 2021 Jul;154:105365.

[2]. Timmers M, et al. Profiling the dynamics of CSF and plasma Aβ reduction after treatment with JNJ-54861911, a potent oral BACE inhibitor.Alzheimers Dement (N Y). 2016 Aug 24;2(3):202-212.

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