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BMS 299897

Catalog No: tcsc1339

Available Sizes

Size: 10mg

Size: 50mg

Specifications

CAS No:
290315-45-6

Formula:
$C_{24}H_{21}ClF_3NO_4S$

Pathway:
Stem Cell/Wnt;Neuronal Signaling

Target:
γ-secretase;γ-secretase

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 30 mg/mL (58.60 mM)

Observed Molecular Weight:
511.94

Product Description

BMS 299897 is a sulfonamide **γ-secretase** inhibitor with an **IC_{50}** of 7 nM for Aβ production inhibition in HEK293 cells stably overexpressing amyloid precursor protein (APP).

IC50 & Target: IC50: 7 nM (Aβ, in HEK293 cells)[1]

In Vitro: BMS-299897 reduces the levels of each of the Aβ peptides. At 1 μM, BMS-299897 decreases these peptides to levels

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ranging from 20 to 50% of the vehicle control. BMS-299897 treatment reduces the portion of QD-BDNF signals moving in the retrograde direction (p=0.0198) with a concomitant increase in the portion of signals moving in the anterograde direction (p=0.0147) [2].

In Vivo: BMS-299897 shows dose- and time-dependent reductions of amyloid β-peptide (Aβ) in brain, cerebrospinal fluid (CSF), and plasma in young transgenic mice, with a correlation between brain and CSF Aβ levels. BMS-299897 reduces both brain and plasma $Aβ_{1-40}$ in APP-YAC mice and increases brain concentrations of APPcarboxy-terminal fragments, consistent with γ-secretase inhibition. BMS-299897, attenuates this $Aβ_{25-35}$-induced $Aβ_{1-42}$ seeding and toxicity. BMS-299897 is administered at 0.1-1 nmol/mouse, concomittantly with $Aβ_{25-35}$ (9 nmol) in male Swiss mice. After one week, the contents in $Aβ_{1-42}$ and $Aβ_{1-40}$, and the levels in lipid peroxidation are analyzed in the mouse hippocampus. Mice are submitted to spontaneous alternation, passive avoidance and object recognition to analyze their short- and long-term memory abilities. $Aβ_{25-35}$ increases $Aβ_{1-42}$ content (+240%) but fails to affect $Aβ_{1-40}$. BMS-299897 blocks the increase in $Aβ_{1-42}$ content and decreased $Aβ_{1-40}$ levels significantly. The compound does not affect $Aβ_{25-35}$-induced increase in hippocampal lipid peroxidation. Behaviorally, BMS-299897 blocks the $Aβ_{25-35}$-induced deficits in spontaneous alternation or novel object recognition, using a 1 h intertrial time interval. The co-administration of the γ-secretase inhibitor BMS-299897, in the 0.1-1 μmol/mouse dose-range, completely blocks the $Aβ_{25-35}$-induced increase in $Aβ_{1-42}$ content[1].