

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

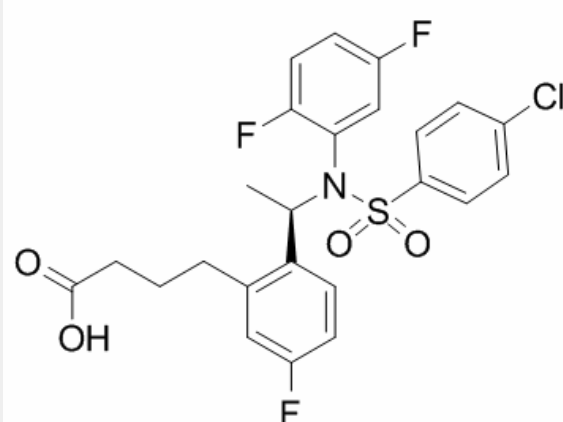
BMS 299897 is a sulfonamide **γ -secretase** inhibitor with an **IC₅₀** of 7 nM for A β production inhibition in HEK293 cells stably overexpressing amyloid precursor protein (APP).

IC50 & Target: IC50: 7 nM (A β , in HEK293 cells)^[1]

In Vitro: BMS-299897 reduces the levels of each of the A β peptides. At 1 μ M, BMS-299897 decreases these peptides to levels

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 ($A\beta$) in brain, cerebrospinal fluid (CSF), and plasma in young transgenic mice, with a correlation between brain and CSF $A\beta$ levels. BMS-299897 reduces both brain and plasma $A\beta_{1-40}$ in APP-YAC mice and increases brain concentrations of APPcarboxy-terminal fragments, consistent with γ -secretase inhibition. BMS-299897, attenuates this $A\beta_{25-35}$ -induced $A\beta_{1-42}$ seeding and toxicity. BMS-299897 is administered at 0.1-1 nmol/mouse, concomittantly with $A\beta_{25-35}$ (9 nmol) in male Swiss mice. After one week, the contents in $A\beta_{1-42}$ and $A\beta_{1-40}$, 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. $A\beta_{25-35}$ increases $A\beta_{1-42}$ content (+240%) but fails to affect $A\beta_{1-40}$. BMS-299897 blocks the increase in $A\beta_{1-42}$ content and decreased $A\beta_{1-40}$ levels significantly. The compound does not affect $A\beta_{25-35}$ -induced increase in hippocampal lipid peroxidation. Behaviorally, BMS-299897 blocks the $A\beta_{25-35}$ -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 μ mol/mouse dose-range, completely blocks the $A\beta_{25-35}$ -induced increase in $A\beta_{1-42}$ content^[1].



All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!