Web: www.taiclone.com
Tel: +886-2-2735-9682
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Fingolimod

Catalog No: tcsc2900

Available Sizes

Size: 5mg

Size: 10mg

Specifications

CAS No:
162359-55-9

Formula:
$C_{19}H_{33}NO_2$

Pathway:
GPCR/G Protein

Target:
LPL Receptor

Purity / Grade:
>98%

Solubility:
Ethanol : 7.69 mg/mL (25.01 mM; Need ultrasonic)

Alternative Names:
FTY720 free base

Observed Molecular Weight:
307.47

Product Description

Fingolimod is a **sphingosine 1-phosphate** (**S1P**) antagonist with **IC_{50}** of 0.033 nM in K562 and NK cells.

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IC50 & Target: IC50: 0.033 nM (S1P, in K562 and NK cells)[1]

In Vitro: The monocyte-derived immature dendritic cells (iDCs) are pretreated with various concentrations of S1P for various periods of time prior to their incubation with NK cells. Four hours incubation of autologous or allogeneic iDCs with 0.2-20 μM of S1P significantly protectes these cells from NK cell lysis. The IC_{50} values of S1P are calculated at 160 nM for autologous iDCs, and 34 nM for allogeneic iDCs. Next, the inhibitory effect of S1P is revered by various concentrations of Fingolimod or SEW2871, with an IC_{50} effect of 173 or 15 nM, respectively[1]. Fingolimod has been reported to reduce LPA synthesis via inhibition of the lysophospholipase autotaxin. Fingolimod treatment correlates with a significant elevation of axonal cAMP, a crucial factor for axonal outgrowth. Additionally, Fingolimod significantly reduces LPA levels in the injured nerve. PF-8380 treatment correlates with improved myelin thickness[2].

In Vivo: Fingolimod treatment results in significantly increased nerve conduction at 14 days post-crush in wildtype C57BL/6 mice. However, *Foxn1*$^{-/-}$ mice, which are devoid of T- but not B-lymphocytes, show an improvement of nerve regeneration under fingolimod treatment. Although the mean increase in nerve conduction velocity in both fingolimod-treated and control*Foxn1*$^{-/-}$ mice implies a potentially positive role of T-lymphocyte deficiency on nerve regeneration, only fingolimod-treated *Foxn1*$^{-/-}$ mice show a significant improvement compared to C57BL/6 controls and performed better in the functional analysis[2]. Treatment of the animals with Fingolimod for 28 d results in a clear reduction in the binding of ^{18}F-GE180 when compare with vehicle-treated animals and evaluated by ex vivo autoradiography. Quantification of the binding of the radiotracer revealed a significant reduction in the binding potential of ^{18}F-GE180 (P[3].