M-110

Cat. No.: HY-12830

CAS No.: 1395048-49-3

Molecular Formula: $C_{22}H_{28}ClN_5O_3$

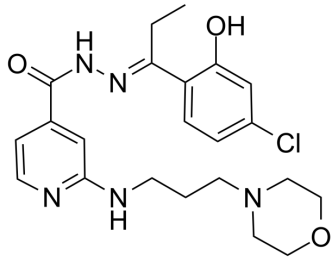
Molecular Weight: 445.94

Target: Pim

Pathway: JAK/STAT Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 33.33 mg/mL (74.74 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2425 mL	11.2123 mL	22.4245 mL
	5 mM	0.4485 mL	2.2425 mL	4.4849 mL
	10 mM	0.2242 mL	1.1212 mL	2.2425 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (5.61 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.66 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

M-110 is a highly selective, ATP-competitive inhibitor of PIM kinases with a preference for PIM-3 (IC_{50}=47 nM). M-110 inhibits PIM-1 and PIM-2 with similar IC_{50}s of 2.5 μM. M-110 inhibits the proliferation of prostate cancer cell lines with IC_{50}s of 0.6 to 0.9 μM[1].

In Vitro

M-110 (0.01-10 μM; 72 hours) inhibiting the growth of DU-145 cells with an IC_{50} value of 0.9 μM[1]. M-110 has no activity on normal human peripheral blood mononuclear cells up to 40 μM[1].
M-110 (10 μM; 18 hours) inhibits STAT3 Tyr705 phosphorylation[1].
M-110 inhibits the expression of active STAT3 through inhibition of PIM-3. M-110 also inhibits the proliferation of 22Rv1, PC3, and SW480 cells, with IC50 values of 0.6 to 0.8 μM[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]	
Cell Line:	DU-145 cells
Concentration:	0.01, 0.1, 1, 10 μM
Incubation Time:	72 hours
Result:	Inhibiting the growth of DU-145 cells with an IC_{50} value of 0.9 μM.

Western Blot Analysis[1]	
Cell Line:	DU-145 cells
Concentration:	10 μM
Incubation Time:	18 hours
Result:	Reduced the expression of p-STAT3 Tyr705 to 23.5%, compared with untreated cells without affecting the expression of STAT3.

REFERENCES

[1]. He Y, et al. Schisantherin A suppresses osteoclast formation and wear particle-induced osteolysis via modulating RANKL signaling pathways. Biochem Biophys Res Commun. 2014 Jul 4;449(3):344-50.

[2]. Zhou E, et al. Schisantherin A protects lipopolysaccharide-induced acute respiratory distress syndrome in mice through inhibiting NF-κB and MAPKs signaling pathways. Int Immunopharmacol. 2014 Sep;22(1):133-40.

[3]. Sa F, et al. Discovery of novel anti-parkinsonian effect of schisantherin A in in vitro and in vivo. Neurosci Lett. 2015 Apr 23;593:7-12.

[4]. Zhang LQ, et al. Schisantherin A protects against 6-OHDA-induced dopaminergic neuron damage in zebrafish and cytotoxicity in SH-SY5Y cells through the ROS/NO and AKT/GSK3β pathways. J Ethnopharmacol. 2015 Apr 29. pii: S0378-8741(15)00306-2.

[5]. Chang M, et al.PIM kinase inhibitors downregulate STAT3(Tyr705) phosphorylation.Mol Cancer Ther. 2010 Sep;9(9):2478-87.

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