OD36 hydrochloride

Cat. No.: HY-19628A

CAS No.: 2387510-88-3

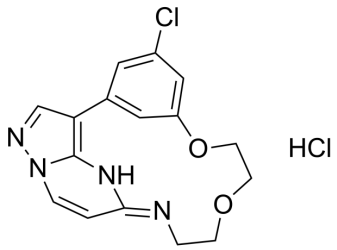
Molecular Formula: $C_{16}H_{16}Cl_2N_4O_2$

Molecular Weight: 367.23

Target: RIP kinase; TGF-β Receptor

Pathway: Apoptosis; TGF-beta/Smad

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

OD36hydrochloride is a RIPK2 inhibitor with an IC_{50} of 5.3 nM. OD36 hydrochloride is a macrocyclic inhibitor with potent binding to the ALK2 kinase ATP pocket. OD36 hydrochloride shows ALK2-directed activity with K_Ds of 37 nM[1][2].

IC_{50} & Target

RIPK2	ACVR1	ACVR1	ALK2 R206H
5.3 nM (IC_{50})	37 nM (Kd)	47 nM (IC_{50})	22 nM (IC_{50})

In Vitro

OD36 also inhibits ALK2 and ALK2 R206H with IC_{50}s of 47 and 22 nM, respevtively[1].
OD36 shows activity against ALK1 with a K_D of 90 nM[2].
OD36 potently antagonize mutant ALK2 signaling and osteogenic differentiation[2].
OD36 (0.1-1 μM; 24 h) efficiently inhibits BMP-6 (50 ng/mL)-induced p-Smad1/5 in KS483 cells[2].
Preincubation of fibrodysplasia ossificans progressiva (FOP) endothelial colony-forming cells (ECFCs) with OD36 (0.5 μM) completely prevents the activation of Smad1/5 and gene targets ID-1 and ID-3 in response to activin A[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[2]

Cell Line:	KS483 cells
Concentration:	0.1, 0.2, and 1 μM
Incubation Time:	24 h
Result:	Inhibited BMP-6 induced p-Smad1/5.

In Vivo

OD36 (6.25 mg/kg; i.p.; once) alleviates inflammation in an acute peritonitis mice model[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57BL/6 mice, muramyl dipeptide (MDP)-induced model of peritonitis[3]
Dosage:	6.25 mg/kg
Administration:	Intraperitoneal injection, 30 min prior to MDP
Result:	Inhibited the recruitment of inflammatory cells to the peritoneum, specifically that of

	neutrophils, and, to a lesser extent, lymphocytes. Decreased RIPK2-specific genes as well as inflammatory cytokine and chemokine gene expression.

REFERENCES

[1]. Justine T Tigno-Aranjuez, et al. In vivo inhibition of RIPK2 kinase alleviates inflammatory disease. J Biol Chem. 2014 Oct 24;289(43):29651-64.

[2]. Gonzalo Sánchez-Duffhues, et al. Development of Macrocycle Kinase Inhibitors for ALK2 Using Fibrodysplasia Ossificans Progressiva-Derived Endothelial Cells. JBMR Plus. 2019 Oct 7;3(11):e10230.

[3]. Tigno-Aranjuez JT, et al. In vivo inhibition of RIPK2 kinase alleviates inflammatory disease. J Biol Chem. 2014 Oct 24;289(43):29651-64.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA