Akt/ROCK-IN-1

Cat. No.: HY-156796

CAS No.: 2983889-44-5

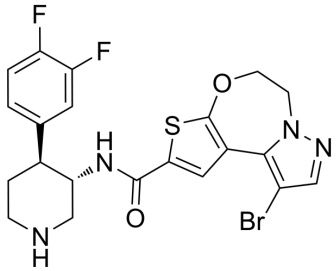
Molecular Formula: $C_{21}H_{19}BrF_2N_4O_2S$

Molecular Weight: 509.37

Target: ADC Cytotoxin

Pathway: Antibody-drug Conjugate/ADC Related

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



BIOLOGICAL ACTIVITY

Description

Akt/ROCK-IN-1 (B12) is a dual inhibitor for Akt and ROCK, with the IC_{50}s of 0.023 nM and 1.47 nM, respectively. Akt/ROCK-IN-1 has antitumor activity for neuroblastoma[1].

In Vitro

Akt/ROCK-IN-1 (0.5 μM; 0-72 h), shows potent antiproliferative effects and excellent differentiation-inducing activity in Neuro2a cells.[1].
Akt/ROCK-IN-1 (800 nM) can lead to a significant increase in the proportion of Neuro2a cells in G0/G1 phase[1].
Akt/ROCK-IN-1 (0.5, 2 μM) can lead to a significant decrease in the phosphorylation levels of mTOR, GSK-3β, and PRAS40, whereas Akt showed a feedback increase in phosphorylation levels[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Akt/ROCK-IN-1 (20, 40 mg/kg; i.p.; everyday for 14d) can inhibit tumor growth in mice are implanted with Neuro2a cells[1].
Akt/ROCK-IN-1 (10 mg/kg; i.p., p.o.; Single Dose) with intraperitoneal administration results in better pharmacokinetic parameters compared to oral administration in rats[1].

Pharmacokinetic Analysis in ICR mice Model[1]

□□□□□□□□[1]

Route	Dose (mg/kg)	T_{max} (h)	$T_{1/2}$ (h)	C_{max} (ng/mL)	Cl (mL·h/kg)	AUC_{0-t} (ng·h/mL)	$AUC_{0-\infty}$ (ng·h/mL)	MRT (h)	V_{ss}/Vd (L/kg)
p.o.	10	2.0	4.3	96.3	15374.1	481.8	650.4	6.2	96
i.p.	10	0.3	2.2	561.3	5934.9	1548.9	1685.0	3.2	19.0

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	mice were implanted with Neuro2a cells[1]
Dosage:	20 mg/kg and 40 mg/kg; everyday for 14d
Administration:	i.p

Result:	Inhibited tumor growth compared to the vehicle control.Mice maintained stable body weights throughout the treatment, indicating a favorable safety profile. Post-treatment analysis showed significant differentiation in tumor cells, demonstrated by increased expression of differentiation markers.

Animal Model:	SD rats or ICR mice[1]
Dosage:	10 mg/kg; Single Dose
Administration:	i.p.; p.o.
Result:	Showed low oral bioavailability and plasma stability, particularly in rat liver microsomes with only 18.0% remaining after 1 hour. Intraperitoneal administration resulted in better pharmacokinetic parameters compared to oral administration, suggesting that this route could be more suitable for achieving therapeutic plasma concentrations.

REFERENCES

[1]. Jinxin Che, et al. Discovery of Novel Oxazepine Derivatives as Akt/ROCK Inhibitors for Growth Arrest and Differentiation Induction in Neuroblastoma Treatment. J Med Chem. 2023 Oct 12;66(19):13530-13555.

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