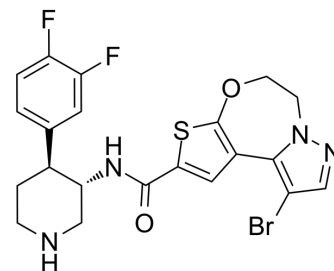


Akt/ROCK-IN-1

Cat. No.:	HY-156796
CAS No.:	2983889-44-5
Molecular Formula:	C ₂₁ H ₁₉ BrF ₂ N ₄ O ₂ S
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	<p>Akt/ROCK-IN-1 (0.5 μM; 0-72 h), shows potent antiproliferative effects and excellent differentiation-inducing activity in Neuro2a cells.^[1]</p> <p>Akt/ROCK-IN-1 (800 nM) can lead to a significant increase in the proportion of Neuro2a cells in G₀/G₁ phase^[1].</p> <p>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].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>																																				
In Vivo	<p>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].</p> <p>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].</p> <p>Pharmacokinetic Analysis in ICR mice Model^[1]</p> <p>☒☒☒☒☒☒^[1]</p> <table border="1"> <thead> <tr> <th>Route</th> <th>Dose (mg/kg)</th> <th>T_{max} (h)</th> <th>T_{1/2} (h)</th> <th>C_{max} (ng/mL)</th> <th>Cl (mL·h/kg)</th> <th>AUC_{0-t} (ng·h/mL)</th> <th>AUC_{0-∞} (ng·h/mL)</th> <th>MRT (h)</th> <th>V_{ss}/V_d (L/kg)</th> </tr> </thead> <tbody> <tr> <td>p.o.</td> <td>10</td> <td>2.0</td> <td>4.3</td> <td>96.3</td> <td>15374.1</td> <td>481.8</td> <td>650.4</td> <td>6.2</td> <td>96</td> </tr> <tr> <td>i.p.</td> <td>10</td> <td>0.3</td> <td>2.2</td> <td>561.3</td> <td>5934.9</td> <td>1548.9</td> <td>1685.0</td> <td>3.2</td> <td>19.0</td> </tr> </tbody> </table> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1"> <tr> <td>Animal Model:</td> <td>mice were implanted with Neuro2a cells^[1]</td> </tr> <tr> <td>Dosage:</td> <td>20 mg/kg and 40 mg/kg; everyday for 14d</td> </tr> <tr> <td>Administration:</td> <td>i.p</td> </tr> </table>	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-∞} (ng·h/mL)	MRT (h)	V _{ss} /V _d (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	Animal Model:	mice were implanted with Neuro2a cells ^[1]	Dosage:	20 mg/kg and 40 mg/kg; everyday for 14d	Administration:	i.p
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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:	Shown 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.

Caution: Product has not been fully validated for medical applications. For research use only.

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