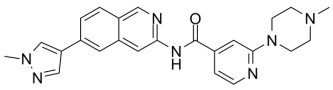
Cirtuvivint

Cat. No.:	HY-137435
CAS No.:	2143917-62-6
Molecular Formula:	$C_{24}H_{25}N_7O$
Molecular Weight:	427.5
Target:	CDK
Pathway:	Cell Cycle/DNA Damage
Storage:	4°C, sealed storage, away from moisture and light * In solvent : -80°C, 1 years; -20°C, 6 months (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 12.5 mg/mL (29.24 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3392 mL	11.6959 mL	23.3918 mL
	5 mM	0.4678 mL	2.3392 mL	4.6784 mL
	10 mM	0.2339 mL	1.1696 mL	2.3392 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: ≥ 1.25 mg/mL (2.92 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.25 mg/mL (2.92 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Cirtuvivint (SM08502) is a potent and orally active CDC-like kinase (CLK) inhibitor. Cirtuvivint can be used for solid tumors research[1].

In Vitro

Cirtuvivint (SM08502) inhibits serine and arginine rich splicing factor (SRSF) phosphorylation and disrupted spliceosome activity, which is associated with inhibition of Wnt pathway-related gene and protein expression. Cirtuvivint induces the generation of splicing variants of Wnt pathway genes, suggesting that its mechanism for inhibition of gene expression includes effects on alternative splicing[1].

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

In Vivo

Orally administered Cirtuvivint (SM08502) significantly inhibits growth of gastrointestinal tumors and decreased SRSF phosphorylation and Wnt pathway gene expression in xenograft mouse models[1].

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REFERENCES

[1]. Betty Y Tam, et al. The CLK inhibitor SM08502 induces anti-tumor activity and reduces Wnt pathway gene expression in gastrointestinal cancer models. Cancer Lett. 2020 Mar 31;473:186-197.

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