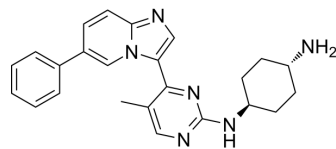


## CDDD11-8

<b>Cat. No.:</b>	HY-162037
<b>CAS No.:</b>	2241659-94-7
<b>Molecular Formula:</b>	C <sub>24</sub> H <sub>26</sub> N <sub>6</sub>
<b>Molecular Weight:</b>	398.5
<b>Target:</b>	CDK; FLT3
<b>Pathway:</b>	Cell Cycle/DNA Damage; Protein Tyrosine Kinase/RTK
<b>Storage:</b>	Powder    -20°C    3 years In solvent   -80°C    6 months -20°C    1 month



### SOLVENT & SOLUBILITY

<b>In Vitro</b>	DMSO : 100 mg/mL (250.94 mM; Need ultrasonic)					
	<b>Preparing Stock Solutions</b>	<b>Solvent</b>	<b>Mass</b>	<b>1 mg</b>	<b>5 mg</b>	<b>10 mg</b>
		<b>Concentration</b>				
		<b>1 mM</b>		2.5094 mL	12.5471 mL	25.0941 mL
		<b>5 mM</b>		0.5019 mL	2.5094 mL	5.0188 mL
<b>10 mM</b>		0.2509 mL	1.2547 mL	2.5094 mL		
Please refer to the solubility information to select the appropriate solvent.						
<b>In Vivo</b>	1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.5 mg/mL (6.27 mM); Clear solution					

### BIOLOGICAL ACTIVITY

<b>Description</b>	CDDD11-8 is an orally active, potent and selective inhibitor of CDK9 and FLT3-ITD, with K <sub>i</sub> values of 8 and 13 nM, respectively. CDDD11-8 reduces the proliferation of leukemia cell lines and was particularly effective against those harboring FLT3-ITD mutation <sup>[1][2]</sup> .
<b>IC<sub>50</sub> &amp; Target</b>	CDK9 8 nM (K <sub>i</sub> )
<b>In Vitro</b>	CDDD11-8 (0-3 μM, 24 h) suppresses the expressions of c-MYC, MCL-1, and XIAP in MV4-11 and MOLM-13 cells <sup>[1]</sup> . CDDD11-8 dose-dependently inhibits proliferation (IC <sub>50</sub> range: 281-734 nM), induces cell cycle arrest, and increases apoptosis of cell lines <sup>[2]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
<b>In Vivo</b>	CDDD11-8 (0-125 mg/kg, PO, daily) induces tumor regression <sup>[1]</sup> .

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Animal Model:	MV4-11 tumor-bearing mice <sup>[1]</sup>
Dosage:	75 or 125 mg/kg
Administration:	PO, daily for 5 or 28 days
Result:	Induced tumor regression. Potently inhibited the phosphorylation of RNAPII at Ser2 and Ser5, confirming the inhibition of CDK9 in vivo. Reduced phosphorylation of FLT3 at Tyr591 and STAT5 at Tyr 694.

## REFERENCES

[1]. Anshabo AT, et al. An Orally Bioavailable and Highly Efficacious Inhibitor of CDK9/FLT3 for the Treatment of Acute Myeloid Leukemia. *Cancers (Basel)*. 2022 Feb 22;14(5):1113.

[2]. Mustafa EH, et al. Selective inhibition of CDK9 in triple negative breast cancer. *Oncogene*. 2023 Nov 24.

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

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