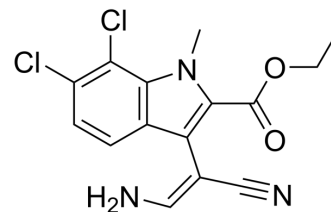


## KH-CB19

<b>Cat. No.:</b>	HY-12828		
<b>CAS No.:</b>	1354037-26-5		
<b>Molecular Formula:</b>	C <sub>15</sub> H <sub>13</sub> Cl <sub>2</sub> N <sub>3</sub> O <sub>2</sub>		
<b>Molecular Weight:</b>	338.19		
<b>Target:</b>	CDK		
<b>Pathway:</b>	Cell Cycle/DNA Damage		
<b>Storage:</b>	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



## SOLVENT & SOLUBILITY

### In Vitro

DMSO : ≥ 50 mg/mL (147.85 mM)  
 \* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent		1 mg	5 mg	10 mg
	Concentration	Mass			
	1 mM		2.9569 mL	14.7846 mL	29.5692 mL
	5 mM		0.5914 mL	2.9569 mL	5.9138 mL
	10 mM		0.2957 mL	1.4785 mL	2.9569 mL

Please refer to the solubility information to select the appropriate solvent.

### In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline  
 Solubility: ≥ 2.5 mg/mL (7.39 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil  
 Solubility: ≥ 2.5 mg/mL (7.39 mM); Clear solution

## BIOLOGICAL ACTIVITY

### Description

KH-CB19 is a potent CLK (cdc2-like kinase) inhibitor (CLK1 IC<sub>50</sub>=19.7 nM; CLK3 IC<sub>50</sub>=530 nM). KH-CB19 shows antiviral activity and inhibits influenza virus replication (IC<sub>50</sub>=13.6 μM)<sup>[1][2]</sup>.

### IC<sub>50</sub> & Target

CLK1 19.7 nM (IC <sub>50</sub> )	CLK3 530 nM (IC <sub>50</sub> )	DYRK1A 55.2 nM (IC <sub>50</sub> )
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### In Vitro

KH-CB19 (10 μM; 1 h) shows CLK inhibition on SR protein phosphorylation in human microvascular endothelial cells<sup>[1]</sup>.  
 KH-CB19 (50 μM; 6 h) inhibits the disruption by CLK4 in A549 cells<sup>[2]</sup>.  
 MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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#### Western Blot Analysis<sup>[1]</sup>

Cell Line:	Human microvascular endothelial cells (HMEC-1)
Concentration:	10 $\mu$ M
Incubation Time:	1 hour
Result:	Led to a reduced phosphorylation of SRp75, SRp55, and SRp20 compared with non TNF- $\alpha$ -stimulated controls. Led to a reduction of the TNF- $\alpha$ -induced increase in phosphorylation of all analyzed SR proteins compared with TNF- $\alpha$ -stimulated controls.

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#### REFERENCES

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- [1]. Fedorov O, et al. Specific CLK inhibitors from a novel chemotype for regulation of alternative splicing. *Chem Biol.* 2011 Jan 28;18(1):67-76.
- [2]. Artarini A, et al. Regulation of influenza A virus mRNA splicing by CLK1. *Antiviral Res.* 2019 Aug;168:187-196.
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**Caution: Product has not been fully validated for medical applications. For research use only.**

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