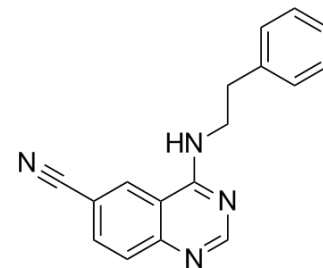


Senexin A

Cat. No.:	HY-15681		
CAS No.:	1366002-50-7		
Molecular Formula:	C ₁₇ H ₁₄ N ₄		
Molecular Weight:	274.32		
Target:	CDK		
Pathway:	Cell Cycle/DNA Damage		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (364.54 mM)
 * "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent		1 mg	5 mg	10 mg
	Concentration	Mass			
1 mM			3.6454 mL	18.2269 mL	36.4538 mL
5 mM			0.7291 mL	3.6454 mL	7.2908 mL
10 mM			0.3645 mL	1.8227 mL	3.6454 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 (9.11 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
 Solubility: ≥ 2.5 mg/mL (9.11 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Senexin A is a CDK8 inhibitor with an IC₅₀ of 280 nM.

IC₅₀ & Target

CDK19 0.31 μM (Kd)	CDK8 0.83 μM (Kd)
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In Vitro

Senexin A inhibits CDK8 and CDK19 ATP site binding with K_d50 of 0.83 μM and 0.31 μM, respectively and CDK8 kinase activity with IC₅₀ of 0.28 μM. Senexin A inhibits β-catenin-dependent transcription in HCT116 colon carcinoma cells. The induction of transcription factor EGR1 upon serum starvation, followed by readdition of serum, is strongly inhibited by Senexin A in HT1080 cells. Senexin A inhibits only p21-induced transcription but not other biological effects of p21. Senexin A also

decreases the expression of many secreted tumor-promoting factors in doxorubicin-treated wild-type HCT116 cells^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Five daily treatment of Senexin A fully reverses tumor-promoting effect of chemotherapy. Senexin A shows no detectable toxicity and no significant effects on body weight, organ weights, or blood cell counts in C57BL/6 mice during the treatment. This effect of doxorubicin treatment is completely abolished, however, when doxorubicin injection is followed by administration of Senexin A. Senexin A treatment strongly improves the response of A549/MEF tumors to doxorubicin^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration ^[1]

Mice: Senexin A toxicity study is conducted by Taconic in C57BL/6 mice, using five mice per group treated with 20 mg/kg Senexin A or carrier (80% propylene glycol), with five daily i.p. injections. Mice are weighed on days 3 and 6, and killed on day 6. Organ weights are determined for brain, kidney, thymus, spleen, lung, and liver. Terminal blood samples are analyzed to determine the numbers of total white blood cells, neutrophils, lymphocytes, monocytes, eosinophils, and basophils^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nucleic Acids Res. 2021 Jan 14;gkaa1289.
- Cell Death Dis. 2020 Sep 15;11(9):754.
- J Cell Biochem. 2019 Aug;120(8):14095-14106.
- Viruses. 2020 Jun 17;12(6):654.

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REFERENCES

[1]. Porter DC, et al. Cyclin-dependent kinase 8 mediates chemotherapy-induced tumor-promoting paracrine activities. Proc Natl Acad Sci U S A. 2012 Aug 21;109(34):13799-804.

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

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