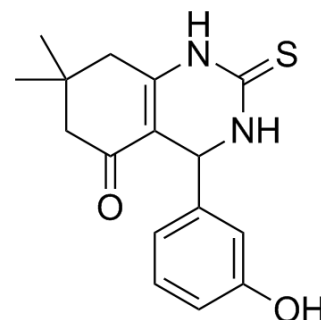


Dimethylenastron

Cat. No.:	HY-19944		
CAS No.:	863774-58-7		
Molecular Formula:	C ₁₆ H ₁₈ N ₂ O ₂ S		
Molecular Weight:	302.39		
Target:	Kinesin		
Pathway:	Cell Cycle/DNA Damage; Cytoskeleton		
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 (330.70 mM; Need ultrasonic)				
		Solvent Concentration	Mass 1 mg	5 mg	10 mg
	Preparing Stock Solutions	1 mM	3.3070 mL	16.5349 mL	33.0699 mL
		5 mM	0.6614 mL	3.3070 mL	6.6140 mL
10 mM		0.3307 mL	1.6535 mL	3.3070 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: ≥ 2.5 mg/mL (8.27 mM); Clear solution 2. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (8.27 mM); Clear solution				

BIOLOGICAL ACTIVITY

Description	Dimethylenastron is a potent kinesin Eg5 inhibitor, with an IC ₅₀ of 200 nM.
IC₅₀ & Target	Eg5 200 nM (IC ₅₀)
In Vitro	Dimethylenastron is a potent Eg5 inhibitor, with an IC ₅₀ of 200 nM. Dimethylenastron exhibits no inhibition of five other kinesin subfamilies (kinesin 1/4/7/10 and one ungrouped-originating from 4 different organisms). Dimethylenastron (0.5, 1 μM) causes accumulation of cells in G2/M in HeLa cells ^[1] . Dimethylenastron (3 and 10 μM) concentration-dependently suppresses the migratory ability of the cancer cells in PANC1 pancreatic cancer cells after treatment for 24 h, but does not inhibit the proliferation of cancer cells at 24 h until 72 h. Dimethylenastron also reduces invasion ability of the cancer cells ^[2] .

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	<p>Dimethylenastron (1.0 μmol) induces a milder scarring but the length of bleb survival is not significantly prolonged compared with the control group. Dimethylenastron (1.0 μmol) reveals a markedly reduced ratio of intraocular pressure and a milder, but not obviously reduced, subconjunctival fibrotic reaction in the rabbits treated with glaucoma filtration surgery [3].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

PROTOCOL

Cell Assay [2]	<p>Cell invasion in response to Dimethylenastron is carried out by transwell assays. The upper surface of the transwell filters is coated with matrigel or fibronectin. Cells suspended in 200 μL serum-free media are added to the chamber, and the chamber is placed in a 24-well plate containing complete medium. After 24 h of incubation at 37°C, the filters are gently taken out and matrigel on the upper surface of the filters is removed by cotton swabs. Cells on the underside of transwell filters are fixed with 4% paraformaldehyde for 30 min, stained with 0.1% crystal violet for 10 min, and then photographed. For quantitative assessment, the number of invading cells is counted in five random fields per filter. The extent of cell invasion is quantified as the number of invading cells in the drug-treatment group divided by the number of invading cells in the control group [2].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
Animal Administration [3]	<p>Just after the conjunctival suture is closed, a metallic needle (30 G) is inserted into the subconjunctival space at the nasal margin of the superior rectus muscle and injection of one of the following agents is delivered: The rabbits receive either no adjuvant after the surgery in the control group, one unilateral subconjunctival injection of Dimethylenastron (1.0 μmol, 3.0 μmol) or of the vehicle (DMSO, 99.9%, 10 mg/mL) alone at baseline, which means an injection directly after surgery and in two further groups additionally at days 3 and 7 thereafter (1.0 μmol, 3.0 μmol) [3].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

REFERENCES

- [1]. Gartner M, et al. Development and biological evaluation of potent and specific inhibitors of mitotic Kinesin Eg5. *Chembiochem*. 2005 Jul;6(7):1173-7.
- [2]. Sun XD, et al. Dimethylenastron suppresses human pancreatic cancer cell migration and invasion in vitro via allosteric inhibition of mitotic kinesin Eg5. *Acta Pharmacol Sin*. 2011 Dec;32(12):1543-8.
- [3]. Lüke J, et al. The effect of adjuvant dimethylenastron, a mitotic Kinesin Eg5 inhibitor, in experimental glaucoma filtration surgery. *Curr Eye Res*. 2010 Dec;35(12):1090-8.

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

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