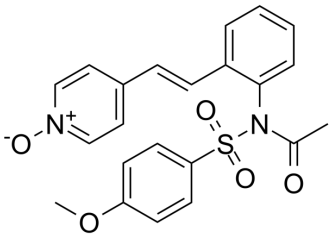
HMN-214

Cat. No.:	HY-12045
CAS No.:	173529-46-9
Molecular Formula:	$C_{22}H_{20}N_2O_5S$
Molecular Weight:	424.47
Target:	Polo-like Kinase (PLK)
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 : 25 mg/mL (58.90 mM; Need ultrasonic)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	2.3559 mL	11.7794 mL	23.5588 mL
5 mM	0.4712 mL	2.3559 mL	4.7118 mL
10 mM	0.2356 mL	1.1779 mL	2.3559 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 (5.89 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.89 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.89 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	HMN-214, an orally bioavailable prodrug of HMN-176, is an inhibitor of polo-like kinase-1 (plk1), with antitumor activity.
IC_{50} & Target	PLK1
In Vitro	HMN-214 is a prodrug of HMN-176. HMN-176 shows potent activities against 22 human tumor cell lines, with a mean IC_{50}s of 118 nM[1]. HMN-176 (3-300 nM) inhibits luciferase expression driven by the MDR1 promoter in a dose dependent manner in HeLa cells. HMN-176 (30-3000 nM) also dose-dependently suppresses complex formation on the Y-box[3]. HMN-214 (3.3 μM)

	enhances luciferase expression relative to vehicle control with the 1,4C-1,4Bis polymer (11-fold) and PEI (37-fold) in PC3-PSMA cells. HMN-214 (≥ 3.3 μM) significantly reduces cell proliferation, causes considerable changes in cell morphology in MB49 cells[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	HMN-214 (33 mg/kg, p.o.) converts to HMN-176 in rats. HMN-214 has no effect on the conduction velocity and the amplitude of action potentials in the aciatic and tibial nerves. HMN-214 (20 mg/kg, p.o.) exhibits antitumor activity in mice[1]. HMN-214 (10, 20 mg/kg, p.o.) decreases MDR1 mRNA expression in nude mice bearing KB- and KB-A.1.-derived tumors[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [4]	Cell proliferation in case of different treatment conditions, relative to untreated control cells (treated as 100% viable, or a live control), is quantified using 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT), a yellow colored reagent which is converted to formazan (a purple dye) by living cells. For screening experiments, transfections are carried out in 96-well cell culture plates, that are seeded with 50,000 cells per well. Following 48 h of transfection, 10 μL of MTT reagent is added to the cells and incubated at 37°C for 2-4 h, and the cells are then lysed by adding 20 μL of MTT detergent and incubated for an additional 2 h at room temperature. Inhibitor dose-optimization transfections are carried out in 24-well plates that are seeded with 50,000 cells per well. After 48 h, 20 μL MTT reagent is added, followed by 100 μL of MTT detergent for lysis for 2 h[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	The ground HMN-214 is suspended with an agate pestle by gradually adding 0.5% methylcellulose 4000 solution to make a 3 mg/mL suspension. This is additionally diluted with methylcellulose 4000 solution to obtain suspensions of the appropriate concentration. Tumor tissue is grown in advance by s.c. transplantation into nude mice. The resulting tumors are removed, cut into cubic fragments of 8 mm^3, and transplanted s.c. into the right axillary region of nude mice with a trocar. When the theoretical volume of the tumor had reached about 145 mm^3, oral administration of HMN-214 is started (day 1)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- PLoS Negl Trop Dis. 2016 Jan 11;10(1):e0004356.
- Department of Pathology. University of California. 2016.
- Harvard Medical School LINCS LIBRARY

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REFERENCES

[1]. Takagi M, et al. In vivo antitumor activity of a novel sulfonamide, HMN-214, against human tumor xenografts in mice and the spectrum of cytotoxicity of its active metabolite, HMN-176. Invest New Drugs. 2003 Nov;21(4):387-99.

[2]. Garland LL, et al. A phase I pharmacokinetic study of HMN-214, a novel oral stilbene derivative with polo-like kinase-1-interacting properties, in patients with advanced solid tumors. Clin Cancer Res. 2006 Sep 1;12(17):5182-9.

[3]. Tanaka H, et al. HMN-176, an active metabolite of the synthetic antitumor agent HMN-214, restores chemosensitivity to multidrug-resistant cells by targeting the transcription factor NF-Y. Cancer Res. 2003 Oct 15;63(20):6942-7.

[4]. Christensen MD, et al. Kinome-level screening identifies inhibition of polo-like kinase-1 (PLK1) as a target for enhancing non-viral transgene expression. J Control

Release. 2015 Apr 28;204:20-9.

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