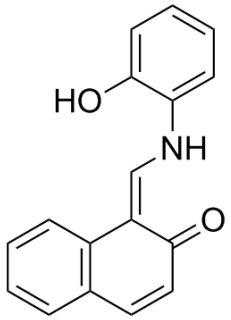
HAMNO

Cat. No.:	HY-111285
CAS No.:	138736-73-9
Molecular Formula:	$C_{17}H_{13}NO_2$
Molecular Weight:	263.29
Target:	Others
Pathway:	Others

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 : 50 mg/mL (189.90 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.7981 mL	18.9905 mL	37.9809 mL
5 mM	0.7596 mL	3.7981 mL	7.5962 mL
10 mM	0.3798 mL	1.8990 mL	3.7981 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: 2.5 mg/mL (9.50 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

HAMNO is a novel protein interaction inhibitor of replication protein A (RPA).

IC_{50} & Target

RPA[1]

In Vitro

HAMNO is a novel protein interaction inhibitor of replication protein A (RPA). RPA is involved in the ATR/Chk1 pathway. HAMNO alone inhibits colony formation in both HNSCC cell lines in the low micromolar range. HAMNO combined with etoposide significantly inhibits colony formation to a greater degree than HAMNO alone. After UMSCC38 cells are exposed to HAMNO, increased pan-nuclear γ-H2AX staining occurs in a dose dependent manner. Cancer derived UMSCC38 cells, as well as another cancer cell line, UMSCC11B, have prominent γ-H2AX staining, particularly after incubation with 20 μM HAMNO. Both UMSCC38 and OKF4 cells present increased γ-H2AX staining after addition of HAMNO, with the greatest increase in signal occurring in S-phase[1].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	In mice, HAMNO slows the progression of UMSCC11B tumors. Ser33 of RPA32, an ATR substrate, is highly phosphorylated after two hours of treatment with 20 μM of etoposide, which is reduced with the addition of 2 μM HAMNO, and is nearly absent at higher concentrations, demonstrating an in vivo effect of HAMNO as an inhibitor of RPA32 phosphorylation by ATR [1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Cell cycle assessment and γ-H2AX staining are monitored in UMSCC38 and OKF4 cells after 2 h incubation with HAMNO (2, 20, 50 μM) and fixed in 70% ethanol overnight. Cells are washed with PBS and incubated overnight in PBS containing 1% BSA, 10% goat serum and PS139-H2AX antibodies, washed and incubated in goat anti-mouse Alexa Fluor 647 antibody for 30 min at room temperature. Cells are incubated in 50 μg/mL propidium iodide and 100 μg/mL RNase A for 30 min, and 10,000 cells per sample are analyzed[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Athymic nude mice are used in this study. UMSCC38 and UMSCC11B cells are implanted into 6-week-old female mice by a single subcutaneous injection of tumor cells (2 to 6×10^5 cells in 100 mL of sterile PBS). The growth rates of tumors are determined by daily monitoring of tumor volume with vernier calipers [tumor volume=1/2(length×width^2)]. Once the tumor size reaches 50 mm^3, etoposide (10 mg/kg mouse) and HAMNO (2 mg/kg) are administered intraperitoneally every day for 3 days. Tumor size is monitored daily and the volume of the tumor is compared among all experimental groups. At least three mice are used per group[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Glanzer JG, et al. RPA inhibition increases replication stress and suppresses tumor growth. Cancer Res. 2014 Sep 15;74(18):5165-72.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA