AGI-24512

Cat. No.: HY-112130

CAS No.: 2201066-53-5

Molecular Formula: $C_{24}H_{24}N_4O_2$

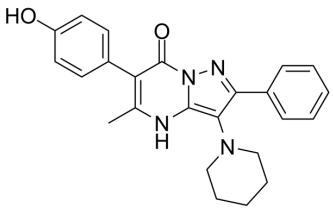
Molecular Weight: 400.47

Target: Methionine Adenosyltransferase (MAT)

Pathway: Epigenetics; Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (249.71 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4971 mL	12.4853 mL	24.9707 mL
5 mM	0.4994 mL	2.4971 mL	4.9941 mL
10 mM	0.2497 mL	1.2485 mL	2.4971 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: ≥ 2.08 mg/mL (5.19 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.08 mg/mL (5.19 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.19 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

AGI-24512 is a potent methionine adenosyltransferase 2α (MAT2A) inhibitor, with an IC_{50} of 8 nM. AGI-24512 triggers DNA damage response. AGI-24512 can block proliferation of MTAP-deleted cancer cells in vitro. AGI-24512 can be used for researching anticancer[1].

IC_{50} & Target

IC50: 8 nM (MAT2A)[1]

In Vitro

AGI-24512 (0-1 μM; 96 hours) blocks proliferation of MTAP (methylthioadenosine phosphorylase)-deleted HCT116 cancer

	cells with an IC_{50} of 100 nM[1]. AGI-24512 significantly increases in γH2AX-positive cells in $MTAP^{-/-}$ HCT116 cells[1]. AGI-24512 inhibits PRMT5-mediated SDMA marks with an IC_{50} of 95 nM in $MTAP^{-/-}$ cells[1]. AGI-24512 leads to a dose-dependent decrease in SAM (S-adenosylmethionine) levels in the HCT116 MTAP-null cell, with an IC_{50} of 100 nM[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	AGI-24512 shows poor oral absorption and a short half-life in rats[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Cancer. 2022 May;3(5):629-648.

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REFERENCES

[1]. Kalev P, et al. MAT2A Inhibition Blocks the Growth of MTAP-Deleted Cancer Cells by Reducing PRMT5-Dependent mRNA Splicing and Inducing DNA Damage. Cancer Cell. 2021 Feb 8;39(2):209-224.e11.

[2]. Konteatis Z, et al. Discovery of AG-270, a First-in-Class Oral MAT2A Inhibitor for the Treatment of Tumors with Homozygous MTAP Deletion. J Med Chem. 2021 Apr 22;64(8):4430-4449.

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