6-Mercaptopurine hydrate

Cat. No.: HY-13677A

CAS No.: 6112-76-1

Molecular Formula: $C_5H_6N_4OS$

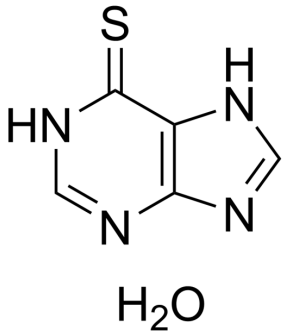
Molecular Weight: 170.19

Target: Nucleoside Antimetabolite/Analog

Pathway: Cell Cycle/DNA Damage

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (293.79 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.8758 mL	29.3789 mL	58.7579 mL
5 mM	1.1752 mL	5.8758 mL	11.7516 mL
10 mM	0.5876 mL	2.9379 mL	5.8758 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 (14.69 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

6-Mercaptopurine hydrate (Mercaptopurine hydrate; 6-MP hydrate) is a purine analogue which acts as an antagonist of the endogenous purines and has been widely used as antileukemic agent and immunosuppressive agent.

IC_{50} & Target

endogenous purines[1]

In Vitro

6-Mercaptopurine hydrate (Mercaptopurine hydrate; 6-MP hydrate) induces NR4A3 transcriptional activity 1.6- to 11-fold (P<0.01) in a dose-responsive manner. It is found that 6-Mercaptopurine hydrate leads to a dose-dependent increase in NR4A3 protein levels.

6-Mercaptopurine hydrate treatment increases cell surface GLUT4 in both basal cells 1.8- to 3.6-fold (P<0.01) and insulin-stimulated cells 2.9- to 4.4-fold (P<0.01) over that in controls. It is also found that 6-Mercaptopurine hydrate increases phospho-AS160 significantly in a dose-responsive manner under both basal and insulin-stimulated conditions[2].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	In the fetal telencephalons of the 6-Mercaptopurine hydrate (6-MP)-treated group, the S phase cell population increases at 36 and 48 h and returns to the control level at 72 h after treatment. The G2/M phase cell population begins to increase at 24 h, peaks at 36 h, decreases at 48 h, and finally returnes to the control level at 72 h. On the other hand, the sub-G1 phase cell population (apoptotic cells) begins to increase at 36 h, peaks at 48 h, and then decreases at 72 h[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [2]	L6 myotubes are treated with DMSO control or 6-Mercaptopurine hydrate (6-MP) for 24 h, with the final 3 h of incubation including treatments in serum-free DMEM, and further incubated in the absence or presence of 100 nM insulin for 60 min at 37°C. Then, protein lysates (50 μg) are collected and subjected to SDS-PAGE. The proteins are finally quantified by densitometric analysis of scanned films using Image J software[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [2]	Cell viability is measured using Cell Viability Assay. L6 skeletal muscle cells are seeded in 96-well plates at a density of 10,000 cells/well and differentiated into myotubes within 7 days. Cells are treated with different doses of 6-Mercaptopurine hydrate (6-MP) for 24 h before the assay. For analysis of cell viability, plates are equilibrated at room temperature for 30 min; 50 μL of Cell Titer-Glo reagent is added to each well, and plates are mixed for 12 min on an orbital shaker. Luminescence is quantified using a luminometer[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Around thirteen-week-old pregnant rats are used in this study. The animals are housed individually in wire-mesh cages in an air-conditioned room (temperature, 23±3°C; humidity, 50±20%; ventilation, 10 times/hour; lighting, 12 h light to12 h dark cycle) and are given pelleted diet and water ad libitum. In the experiment, fifteen pregnant rats are injected i.p. with 50 mg/kg 6-Mercaptopurine hydrate (6-MP) on E13, and three dams each are sacrificed by exsanguination from the abdominal aorta under ether anesthesia at 12, 24, 36, 48, and 72 h. Fetuses are collected from each dam by Caesarean section. As controls, fifteen pregnant rats are injected i.p. with 2.0% methylcellulose solution in distilled water at E13, and three dams are sacrificed at each of the same time-points[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2022 Nov 17;13(1):7031.
- Pharmacol Res. 2024 Jan 26:200:107082.
- Cancers (Basel). 2022 Oct 19;14(20):5127.
- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.
- PLoS Negl Trop Dis. 2019 Aug 20;13(8):e0007681.

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REFERENCES

[1]. Sahasranaman S, et al. Clinical pharmacology and pharmacogenetics of thiopurines. Eur J Clin Pharmacol. 2008 Aug;64(8):753-67.

[2]. Liu Q, et al. 6-Mercaptopurine augments glucose transport activity in skeletal muscle cells in part via a mechanism dependent upon orphan nuclear receptor NR4A3. Am J Physiol Endocrinol Metab. 2013 Nov 1;305(9):E1081-92.

[3]. Kanemitsu H, et al. 6-Mercaptopurine (6-MP) induces cell cycle arrest and apoptosis of neural progenitor cells in the developing fetal rat brain. Neurotoxicol Teratol. 2009 Mar-Apr;31(2):104-9.

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