Aurothioglucose

Cat. No.: HY-A0068

CAS No.: 12192-57-3

Molecular Formula: $C_6H_{11}AuO_5S$

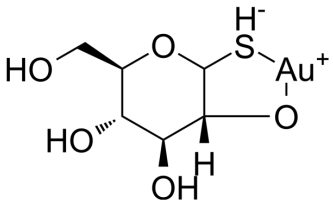
Molecular Weight: 392.18

Target: NF-κB; HIV; Reactive Oxygen Species

Pathway: NF-κB; Anti-infection; Immunology/Inflammation; Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

H_2O : 125 mg/mL (318.73 mM; Need ultrasonic)
DMSO : 6 mg/mL (15.30 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.5498 mL	12.7492 mL	25.4985 mL
5 mM	0.5100 mL	2.5498 mL	5.0997 mL
10 mM	0.2550 mL	1.2749 mL	2.5498 mL

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

BIOLOGICAL ACTIVITY

Description

Aurothioglucose (Gold thioglucose), containing monovalent gold ion, is a potent active-site inhibitor of TrxR1 (thioredoxin reductase 1), with an IC_{50} of 65 nM. Aurothioglucose inhibits the DNA binding of NF-κB in vitro. Aurothioglucose shows anti-HIV and anti-rheumatic activities[1][2].

IC_{50} & Target

IC_{50}: 65 nM (TrxR1)[1]

In Vitro

Aurothioglucose (0-100 μM, 24-72 h) inhibits TrxR1 activity in HeLa cell cytosol but has no effect on the viability of the cells[1].
Aurothioglucose (0-30 μM, 6 h) exhibits very low cytotoxicity on cells[1].
Aurothioglucose (0-20 μM, 24 h) combined with Ebselen (HY-13750) shows a strong synergistic effect, leading to Trx1 (thioredoxin 1) oxidation, reactive oxygen species (ROS) accumulation, and cell death[1].
Aurothioglucose (0-100 μM, 3-12 days) inhibits p24 levels in OM10.1 and Ach2 cells, and inhibits HIV-1 replication in vitro[2].
Aurothioglucose (0-25 μM, 12 days) increases the accumulation of metal gold in a dose-dependent manner[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	HeLa cells
Concentration:	0, 5, 10, 50, 100 μM
Incubation Time:	24, 48, 72 h
Result:	Inhibited TrxR activity by more than 90% at 100 μM in HeLa cells. Cell viability was unaffected by ATG treatment, even at 100 μM after 72 h.

Western Blot Analysis[1]

Cell Line:	HeLa cells
Concentration:	0, 5, 10, and 100 μM
Incubation Time:	24 h
Result:	Showed no significant oxidation of Trx1 or Trx2 in HeLa cells.

Western Blot Analysis[2]

Cell Line:	OM10.1, Ach2 cells
Concentration:	0, 4, 10, 25 and 100 μM
Incubation Time:	3, 6 or 12 days
Result:	Significantly inhibited p24 levels. After 12 days of incubation, the viability of cells treated with 10, 25 and 100 μM Aurothioglucose had decreased to 60% of the control.

In Vivo

Aurothioglucose (25 mg/kg, i.p., single) significantly attenuates lung injury and enhances survival in a clinically relevant murine model of ARDS. The protective effects of Aurothioglucose are GSH dependent[3].
Aurothioglucose (300 mg/kg, i.p., single) induces hypothalamic obesity in mice[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Adult male C3H/HeN mice (8-12 week, LPS/hyperoxia-exposed mice, inflammation/hyperoxia ARDS model)[3]
Dosage:	25 mg/kg
Administration:	IP, single, at 12 h after intratracheal LPS administration
Result:	Significantly attenuated lung injury, increased lung GCLM expression and GSH levels, and decreased mortality.

CUSTOMER VALIDATION

- J Ethnopharmacol. 2020 Mar 1;249:112433.

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REFERENCES

[1]. Du Y, et al. Glutathione and glutaredoxin act as a backup of human thioredoxin reductase 1 to reduce thioredoxin 1 preventing cell death by aurothioglucose. J Biol Chem. 2012 Nov 2;287(45):38210-9.

[2]. Traber KE, et al. Anti-rheumatic compound aurothioglucose inhibits tumor necrosis factor-alpha-induced HIV-1 replication in latently infected OM10.1 and Ach2 cells. Int Immunol. 1999 Feb;11(2):143-50.

[3]. Britt RD Jr, et al. The thioredoxin reductase-1 inhibitor aurothioglucose attenuates lung injury and improves survival in a murine model of acute respiratory distress syndrome. Antioxid Redox Signal. 2014 Jun 10;20(17):2681-91.

[4]. Naruta E, et al. Hypolipidemic effect of pantothenic acid derivatives in mice with hypothalamic obesity induced by aurothioglucose. Exp Toxicol Pathol. 2001 Oct;53(5):393-8.

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