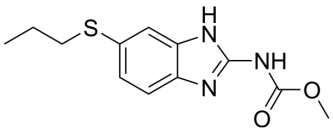
Albendazole

Cat. No.: HY-B0223

CAS No.: 54965-21-8

Molecular Formula: $C_{12}H_{15}N_3O_2S$

Molecular Weight: 265.33



Target: Parasite; Microtubule/Tubulin; Autophagy; Apoptosis; Reactive Oxygen Species; VEGFR; HIF/HIF Prolyl-Hydroxylase; Bacterial; Antibiotic

Pathway: Anti-infection; Cell Cycle/DNA Damage; Cytoskeleton; Autophagy; Apoptosis; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Protein Tyrosine Kinase/RTK

Storage:

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

SOLVENT & SOLUBILITY

In Vitro

DMSO : 20 mg/mL (75.38 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.7689 mL	18.8445 mL	37.6889 mL
5 mM	0.7538 mL	3.7689 mL	7.5378 mL
10 mM	0.3769 mL	1.8844 mL	3.7689 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 mg/mL (7.54 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2 mg/mL (7.54 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Albendazole (SKF-62979) is an orally active and broad-spectrum parasiticide with high effectiveness and low host toxicity, is used for the research of gastrointestinal parasites in humans and animals. Albendazole induces apoptosis and autophagy in cancer cells. Albendazole also inhibits tubulin polymerization and HIF-1α, VEGF expression, has antioxidant activity, and inhibits the glycolytic process in cancer cells[1][2][3][4][5].

IC_{50} & Target

parasites, tubulin[1][2].

	HIF-1α, VEGF[3].
In Vitro	Albendazole (100, 500, 1000 nM; 1, 3, or 5 days) inhibits cell proliferation in a dose-dependent manner[1]. ?Albendazole (100, 250, 500, 1000 nM; 3 days) arrests SKHEP-1 cells at both G0–G1 (250, 500 nM) and G2-M (1000 nM) phases of the cycle[1]. ?Albendazole (5 μM; 24, 36 h) mainly induces early apoptosis in HCT-15 cells and late apoptosis in HCT-1 16, HT29, SW480 cells accompanied with cleavage of PARP and caspase-3 in a time-dependent manner[2]. ?Albendazole (5 μM; 24, 36 h) induces autophagy via increasing autophagy-related protein (such as LC3, Atg7, p-beclin-1, and beclin-1) expression level in HCT-15, HCT-1 16, HT29, and SW480 cells[2]. ?Albendazole (500 nM, 24 h) inhibits hypoxia-induced HIF-1α expression and VEGF expression in A549 cells[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	SKHEP-1 cells
Concentration:	100, 500, 1000 nM
Incubation Time:	1, 3, or 5 days
Result:	Inhibited cell proliferation in a dose-dependent manner.

Cell Cycle Analysis[1]

Cell Line:	SKHEP-1 HCC cells
Concentration:	100, 250, 500, 1000 nM
Incubation Time:	3 days
Result:	Showed dose-dependent effect on the cell cycle kinetics.

Apoptosis Analysis[2]

Cell Line:	HCT-15, HCT-1 16, HT29, SW480 cells
Concentration:	5 μM
Incubation Time:	24, 36 h
Result:	Promoted apoptosis in colon cancer cells.

Cell Autophagy Assay[2]

Cell Line:	HCT-15, HCT-1 16, HT29, SW480 cells
Concentration:	5 μM
Incubation Time:	24, 36 h
Result:	Induced autophagy in colon cancer cells.

Western Blot Analysis[2]

Cell Line:	HCT-15, HCT-1 16, HT29, SW480 cells
Concentration:	5 μM
Incubation Time:	12, 24, 36 h

Result:	Induced apoptosis-related protein (PARP, caspase-3) and autophagy-related protein (such as LC3, Atg7, p-beclin-1, and beclin-1) expression level in a time-dependent manner. Increased phosphorylation of different MAPKs (AMPK, ERK, JNK, p38) and ULK1 protein in a time dependent manner, and up-regulated the activation of different MAPKs. Caused the activation of both MAPK and AMPK pathways.

Western Blot Analysis[3]

Cell Line:	A549 cells
Concentration:	500 nM
Incubation Time:	24 h
Result:	Inhibited hypoxia-induced HIF-1α expression and VEGF expression in A549 cells.

In Vivo

Albendazole (10 mg/kg; i.g.; once a day for 30 days) reduces Echinococcus granulosus cyst weights in mice[4].
?Albendazole (300 mg/kg; p.o.; per day in two divided dose for 20 days) profoundly suppresses tumor growth in vivo[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female BALB/c mice (10-week-age; Echinococcus granulosus infection model)[4].
Dosage:	10 mg/kg
Administration:	Oral gavage; once a day for 30 days.
Result:	Reduced Echinococcus granulosus cyst weights.

Animal Model:	Male BALB/c Nu/Nu mice (6 to 10-week-old; inoculated subcutaneously with SKHEP-1)[1].
Dosage:	50, 150, 300 mg/kg
Administration:	Oral administration; per day in two divided dose for 20 days.
Result:	Profoundly suppressed tumor growth in vivo.

CUSTOMER VALIDATION

- Commun Biol. 2024 Jan 24;7(1):123.
- Toxicol Appl Pharmacol. 2022 Aug 30;116214.
- J Oncol. 2021 Dec 20;2021:4475192.

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REFERENCES

[1]. Vural G, et al. Efficacy of novel albendazole salt formulations against secondary cystic echinococcosis in experimentally infected mice. Parasitology. 2020 Nov;147(13):1425-1432.

[2]. Li L, et al. Determination of albendazole and metabolites in silkworm Bombyx mori hemolymph by ultrafast liquid chromatography tandem triple quadrupole mass spectrometry. PLoS One. 2014 Sep 25;9(9):e105637.

[3]. Pourgholami MH, et al. In vitro and in vivo suppression of growth of hepatocellular carcinoma cells by albendazole. Cancer Lett. 2001 Apr 10;165(1):43-9.

[4]. Jung YY, et al. Regulation of apoptosis and autophagy by albendazole in human colon adenocarcinoma cells. Biochimie. 2022 Jul;198:155-166.

[5]. Zhou F, et al. Albendazole inhibits HIF-1α-dependent glycolysis and VEGF expression in non-small cell lung cancer cells. Mol Cell Biochem. 2017 Apr;428(1-2):171-178.

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