Fenbendazole

Cat. No.: HY-B0413

CAS No.: 43210-67-9

Molecular Formula: $C_{15}H_{13}N_3O_2S$

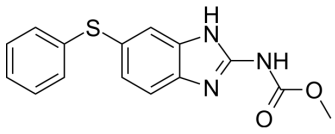
Molecular Weight: 299.35

Target: Parasite; Antibiotic; HIF/HIF Prolyl-Hydroxylase; Microtubule/Tubulin

Pathway: Anti-infection; Metabolic Enzyme/Protease; Cell Cycle/DNA Damage; Cytoskeleton

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 : 10 mg/mL (33.41 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.3406 mL	16.7029 mL	33.4057 mL
5 mM	0.6681 mL	3.3406 mL	6.6811 mL
10 mM	0.3341 mL	1.6703 mL	3.3406 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: ≥ 1 mg/mL (3.34 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1 mg/mL (3.34 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Fenbendazole is an orally active benzimidazole anthelmintic agent, with a broad antiparasitic range. Fenbendazole is a microtubule destabilizing agent and acts on helminthes primarily by binding to tubulin and disrupting the tubulin microtubule equilibrium. Fenbendazole stabilizes the transcriptional activator HIF-1α. Fenbendazole possesses an efficient anti-proliferative activity and induces apoptosis. Fenbendazole causes cell-cycle arrest and mitotic cell death, and has antitumor activity in mice xenografted with wild-type p53[1].

In Vitro

Fenbendazole (1 uM; for 24 h) significantly reduces cell growth in tumour cell lines with wild-type p53, H460 and A549 human NSCLC cell lines[1].

Fenbendazole (1 uM; for 24 h) induces apoptosis and causes an increased level of p53 protein in the mitochondrial fraction[1]

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Fenbendazole (1 uM; for 24 h) causes cell cycle arrest in the mitotic phase in human NSCLC cells[1].

Fenbendazole (1 uM; for 24 h) causes a partial alteration of the microtubule network in human non small cell lung carcinoma (NSCLC) A549 cells[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cycle Analysis[1]

Cell Line:	A549 cells
Concentration:	1 uM
Incubation Time:	For 24 h
Result:	Caused an early elevation of cyclin B1/CDK1 levels (8 h as compared to 16 h in case of control untreated cells). p-histone H3 (Ser10) was found to be up-regulated at 12 and 24 h.

Apoptosis Analysis[1]

Cell Line:	A549 cells
Concentration:	1 uM
Incubation Time:	8, 16, 24, 32, 40, 48 h
Result:	The number of apoptotic cells increased in a time dependent manner with simultaneous decrease in cyclin B1 levels, and ~30% cells had undergone apoptosis after 32 h.

Western Blot Analysis[1]

Cell Line:	H460 cells
Concentration:	1 uM
Incubation Time:	For 24 h
Result:	Caused an increased level of p53 protein in the mitochondrial fraction.

In Vivo

Fenbendazole (1 mg; orally; every second day for 12 day) leads to a marked reduction in tumour size and weight[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female athymic nu/nu mice were xenografted with A549 cells[1]
Dosage:	1 mg/mouse
Administration:	Orally; every second day for 12 day
Result:	Led to a marked reduction in tumour size and weight. Led to a reduction in hemoglobin content in tumors signifying reduced tumor vascularity.

CUSTOMER VALIDATION

- Acta Pharmacol Sin. 2021 Aug 25.
- J Pathol. 2023 Feb 24.

- Commun Biol. 2024 Jan 24;7(1):123.
- J Vet Sci. 2020 Sep;21(5):e72.
- Research Square Preprint. 2021 Aug.

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REFERENCES

[1]. Nilambra Dogra, et al. Fenbendazole acts as a moderate microtubule destabilizing agent and causes cancer cell death by modulating multiple cellular pathways. Sci Rep. 2018 Aug 9;8(1):11926.

[2]. Qiwen Duan, et al. Fenbendazole as a potential anticancer drug. Anticancer Res. 2013 Feb;33(2):355-62.

[3]. Hossein Aleyasin, et al. Antihelminthic benzimidazoles are novel HIF activators that prevent oxidative neuronal death via binding to tubulin. Antioxid Redox Signal. 2015 Jan 10;22(2):121-34.

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