Flubendazole

Cat. No.: HY-B0294

CAS No.: 31430-15-6

Molecular Formula: $C_{16}H_{12}FN_3O_3$

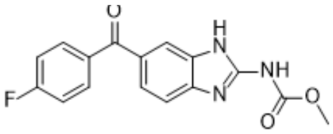
Molecular Weight: 313.28

Target: Parasite; Microtubule/Tubulin; Apoptosis; Autophagy; STAT; MDM-2/p53

Pathway: Anti-infection; Cell Cycle/DNA Damage; Cytoskeleton; Apoptosis; Autophagy; JAK/STAT Signaling; Stem Cell/Wnt

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 : 4.17 mg/mL (13.31 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.1920 mL	15.9602 mL	31.9203 mL
5 mM	0.6384 mL	3.1920 mL	6.3841 mL
10 mM	0.3192 mL	1.5960 mL	3.1920 mL

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

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 5 mg/mL (15.96 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Flubendazole is an anthelmintic drug based on altering microtubule structure, inhibition of tubulin polymerization and disruption of microtubule function. Flubendazole induces apoptosis in human colorectal cancer (CRC) by blocking the STAT3 signaling axis and activation of autophagy. Flubendazole induces P53 expression and reduced Cyclin B1 and p-cdc2 expression. Flubendazole is an antitumor agent. Flubendazole can be used for worm and intestinal parasites[1][2].

IC_{50} & Target

STAT3

In Vitro

Flubendazole (0-400 μM; 48 h) inhibits human colorectal cancer (CRC) cells proliferation[1].
Flubendazole (0.3-1.2 μM; 48 h) induces apoptosis in CRC cells[1].
Flubendazole (0.3-1.2 μM; 24 h) induces autophagy initiation by inactivating mTOR and P62, and upregulating LC3-I/II in CRC cells[1].

Flubendazole (0.3-1.2 μM; 24 h) strongly reduces the expression of P-STAT3 in a dose and time-dependent manner[1].

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

Cell Viability Assay[1]

Cell Line:	CRC cells (HCT116, RKO and SW480)
Concentration:	0-400 μM
Incubation Time:	48 h
Result:	Effectively reduces the viability of CRC cells (HCT116, RKO and SW480) in a concentration-dependent manner, with an IC_{50} of 2-5 μM.

Apoptosis Analysis[1]

Cell Line:	CRC cells (HCT116, RKO and SW480)
Concentration:	0.3, 0.6, 1.2 μM
Incubation Time:	48 h
Result:	Increased the proportion of apoptotic cells in a dose-dependent manner. Dose-dependently effectively increases caspase-3 activity.

Cell Autophagy Assay[1]

Cell Line:	CRC cells (HCT116, RKO and SW480)
Concentration:	0.3, 0.6, 1.2 μM
Incubation Time:	24 h
Result:	Induced autophagy initiation by inactivating mTOR and P62, and upregulating LC3-I/II, which are classical marker of autophagy.

Western Blot Analysis[1]

Cell Line:	CRC cells (HCT116, RKO and SW480)
Concentration:	0.3, 0.6, 1.2 μM
Incubation Time:	24 h
Result:	Strongly reduced the expression of phosphorylated STAT3 (P-STAT3) in a dose and time-dependent manner. No obvious change in total STAT3 expression. Decreased expression of MCL1 and survivin in a dose-dependent manner.

In Vivo

Flubendazole (10, 30 mg/kg; i.p; every other day; 14 days) inhibits growth of CRC tumor xenografts[1].

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

Animal Model:	Female BALB/c athymic nude mice (6-8 weeks) with HCT116 cells[1]
Dosage:	10 or 30 mg/kg
Administration:	Intraperitoneal injection; every other day; 14 days
Result:	Markedly reduced the tumor volume.

	Significantly reduces the protein level of P-STAT3, promotes autophagy and induces apoptosis in vivo.

CUSTOMER VALIDATION

- Theranostics. 2021 Jun 1;11(15):7491-7506.
- Int J Biol Sci. 2023 Apr 23; 19(7): 2270-2288.
- Acta Pharmacol Sin. 2021 Aug 25.
- Commun Biol. 2024 Jan 24;7(1):123.
- ACS Omega. 2020 Nov 15;5(46):29935-29942.

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REFERENCES

[1]. Shichong Lin, et al. Flubendazole demonstrates valid antitumor effects by inhibiting STAT3 and activating autophagy. J Exp Clin Cancer Res. 2019 Jul 8;38(1):293.

[2]. Zhou X, et al. Flubendazole inhibits glioma proliferation by G2/M cell cycle arrest and pro-apoptosis. Cell Death Discov. 2018 Feb 14;4:18.

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