Buprofezin

Cat. No.: HY-B0831

CAS No.: 69327-76-0

Molecular Formula: $C_{16}H_{23}N_3OS$

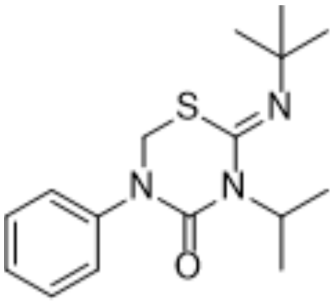
Molecular Weight: 305.44

Target: Reactive Oxygen Species

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

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (327.40 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.2740 mL	16.3698 mL	32.7397 mL
	5 mM	0.6548 mL	3.2740 mL	6.5479 mL
	10 mM	0.3274 mL	1.6370 mL	3.2740 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 (8.18 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.18 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.18 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Buprofezin is a broad-spectrum insecticide and chitin synthesis inhibitor that targets developmental stage coleopteran pests.Buprofezin promotes the conversion of energy metabolism from the aerobic tricarboxylic acid (TCA) cycle and oxidative phosphorylation to anaerobic glycolysis. Buprofezin also promotes the production of reactive oxygen species (ROS) by inhibiting cytochrome c oxidase[1][2].

In Vitro

Buprofezin (100, 300, 1000 μM) significantly reduces viability of HepG2 cells[1].
Buprofezin (3, 10, 30 μM; 24 h) promotes the conversion of energy metabolism from the aerobic tricarboxylic acid (TCA) cycle

and oxidative phosphorylation to anaerobic glycolysis in HepG2 cells[1].
Buprofezin (3, 10, 30 μM; 24 h) inhibits the activity of Complex IV in HepG2 cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

RT-PCR[1]

Cell Line:	HepG2 cells
Concentration:	3, 10, 30 μM
Incubation Time:	24 h
Result:	Significantly increased lactate dehydrogenase B (LDHB) levels when at 30 μM, and slightly increased 6-phosphofructo-2-kinase/fructose-2,6-biphosphatase 3 (PFKFB3). Decreased ATP levels in a concentration-dependent manner to 91.3, 87.9 and 67.2% of the levels in the vehicle control under treatment with 3, 10 and 30 μM buprofezin, respectively. Significantly increased the lactate levels.

Immunofluorescence[1]

Cell Line:	HepG2 cells
Concentration:	3, 10, 30 μM
Incubation Time:	24 h
Result:	Significantly inhibited the activity of Complex IV to 82.2, 69.2 and 63.4% of the vehicle control levels following buprofezin treatment at 3, 10 and 30 μM, respectively.

Cell Viability Assay[1]

Cell Line:	HepG2 cells
Concentration:	3, 10, 30 μM
Incubation Time:	24 h
Result:	Significantly increased the intracellular ROS levels in a concentrate-independent manner, and decreased mtDNA contents.

In Vivo

Buprofezin (46.3, 139, 417 mg/kg; p.o.; single) accumulates in the liver (35.84%), brain (23.58%), stomach (21.94%) and kidney (18.64%)[1].
Buprofezin (46.3, 139, 417 mg/kg; p.o.; single) elevates the MDA level in all organs (especially in the liver and brain) in mice[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male C57BL/6 mice (6 to 8-week-old)[1].
Dosage:	46.3, 139, 417 mg/kg
Administration:	Oral administration; single
Result:	Tended to elevate the MDA level in all organs, and the most significant concentration-dependent increases were observed in the liver and brain. Exhibited the highest concentrations in the liver (35.84%) followed by the brain (23.58%), stomach (21.94%) and kidney (18.64%), while the levels in the mouse spleen and heart were below the limit of detection.

REFERENCES

[1]. Ji X, et al. Potential hepatic toxicity of buprofezin at sublethal concentrations: ROS-mediated conversion of energy metabolism. J Hazard Mater. 2016 Dec 15;320:176-186.

[2]. YoshioIzawa, et al. Inhibition of chitin biosynthesis by buprofezin analogs in relation to their activity controlling Nilaparvata lugens Stål. Pestic Biochem Physiol, 1985, 24(3): 343-347.

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