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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
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- Gefahrgutzuschlag
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Pyraclostrobin

Cat. No.: HY-N6626

CAS No.: 175013-18-0

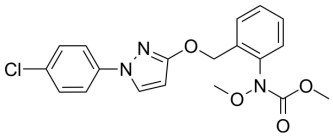
Molecular Formula: $C_{19}H_{18}ClN_3O_4$

Molecular Weight: 387.82

Target: Fungal; Bacterial; Bcl-2 Family; Autophagy; Beclin1; AMPK; mTOR

Pathway: Anti-infection; Apoptosis; Autophagy; Epigenetics; PI3K/Akt/mTOR

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (257.85 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5785 mL	12.8926 mL	25.7852 mL
	5 mM	0.5157 mL	2.5785 mL	5.1570 mL
	10 mM	0.2579 mL	1.2893 mL	2.5785 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: ≥ 2.08 mg/mL (5.36 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.36 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Pyraclostrobin is a highly effective and broad-spectrum strobilurin fungicide. Pyraclostrobin can induce oxidative DNA damage, mitochondrial dysfunction and autophagy through the activation of AMPK/mTOR signaling. Pyraclostrobin can be used to control crop diseases[1][2][3].

IC_{50} & Target	Bax	Bcl-2

In Vitro

Pyraclostrobin (10-80 μmol/L, 24 h) exhibits toxic effects in HepG2 cells[1].
Pyraclostrobin (10-80 μmol/L, 6 h) induced DNA damage and mitochondrial dysfunction in HepG2 cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	HepG2 cells
Concentration:	10 μmol/L, 20 μmol/L, 40 μmol/L, 80 μmol/L
Incubation Time:	24 h
Result:	Inhibited cell survival in concentration-dependent manner with IC_{50} value of 30.22 μmol/L.

Western Blot Analysis[1]

Cell Line:	HepG2 cells
Concentration:	10 μmol/L, 20 μmol/L, 40 μmol/L, 80 μmol/L
Incubation Time:	6 h
Result:	Increased the content of cytochrome c (Cyt c) in the cytoplasm in a concentration-dependent way. Increased pro-apoptotic protein Bax expression and down-regulated anti-apoptosis protein Bcl-2 expression.

In Vivo

Pyraclostrobin (0.01-0.08 μmol/L, Zebrafish larvae were subjected to pyraclostrobin exposure persisting until 72 h post fertilization) results in hepatomegaly in zebrafish larvae[1].
Pyraclostrobin (0.001-0.02 mg/L, Fish were assigned to a vessel containing 20 L of water dissolved Pyraclostrobin for 7-28 days) has the toxic effects on DNA damage and antioxidant enzymatic activities in the zebrafish liver[2].
Pyraclostrobin (33-48 μg/L, Zebrafish larvae at 4 days post fertilization (dpf) were transferred into 24-well plates and subjected to pyraclostrobin until 8 dpf) has toxic effects on zebrafish larvae[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Zebrafish [2]
Dosage:	0.001 mg/L, 0.01 mg/L, 0.02 mg/L
Administration:	Fish were assigned to a vessel containing 20 L of water dissolved Pyraclostrobin
Result:	Showed higher ROS levels in zebrafish livers. Decreased SOD levels and increased MDA levels. Increased the olive tail moments as the dose increased on days 7, 14, 21, and 28.

Animal Model:	Zebrafish larvae [3]
Dosage:	33 mg/L, 36 mg/L, 40 mg/L, 44 mg/L, 48 mg/L
Administration:	Zebrafish larvae at 4 days post fertilization (dpf) were transferred into 24-well plates and subjected to pyraclostrobin until 8 dpf
Result:	Damaged histological and subcellular structure of larval heart and brain. Changed the expression level of cardiac muscle contraction pathway- and neural-related genes and proteins. Impaired larval cardiac function and locomotor behavior.

REFERENCES

[1]. Wu M, et al. Characterization of hepatotoxic effects induced by pyraclostrobin in human HepG2 cells and zebrafish larvae [J]. Chemosphere, 2023, 340: 139732.

[2]. Zhang C, et al. Acute and subchronic toxicity of pyraclostrobin in zebrafish (Danio rerio) [J]. Chemosphere, 2017, 188: 510-516.

[3]. Li H, et al. Mitochondrial dysfunction-based cardiotoxicity and neurotoxicity induced by pyraclostrobin in zebrafish larvae [J]. Environmental Pollution, 2019, 251: 203-211.

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