Tebuconazole

Cat. No.: HY-B0852

CAS No.: 107534-96-3

Molecular Formula: $C_{16}H_{22}ClN_3O$

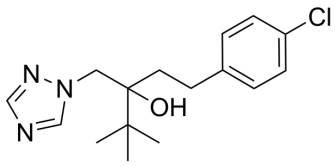
Molecular Weight: 307.82

Target: Cytochrome P450; Fungal; Apoptosis

Pathway: Metabolic Enzyme/Protease; Anti-infection; Apoptosis

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 : ≥ 50 mg/mL (162.43 mM)

H_2O : < 0.1 mg/mL (insoluble)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.2487 mL	16.2433 mL	32.4865 mL
5 mM	0.6497 mL	3.2487 mL	6.4973 mL
10 mM	0.3249 mL	1.6243 mL	3.2487 mL

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

In Vivo

1. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 20 mg/mL (64.97 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (8.12 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.12 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.12 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Tebuconazole is an orally active agricultural azole fungicide which can also inhibit CYP51 with IC_{50}s of 0.9 and 1.3 μM for Candida albicans CYP51 (CaCYP51) and truncated Homo sapiens CYP51 (Δ60HsCYP51), respectively. Tebuconazole induces lipid accumulation and oxidative stress in HepG2 Cells. Tebuconazole decreases MAC-T cells viability and proliferation,

	induces ER-stress-mediated apoptosis and increases oxidative stress levels in MAC-T cells[1][2][3][4][5][6].
IC_{50} & Target	CYP51
In Vitro	Tebuconazole (TEB) (20–80 μM, 24 h) shows lipid accumulation in HepG2 cells[2]. Tebuconazole (20–80 μM, 12 h) increases the nuclear translocation of peroxisome proliferator-activated receptors and the expression of lipid uptake and oxidation-related markers in HepG2 cells[2]. Tebuconazole (20–80 μM, 24 h) increases oxidative stress levels, induces the loss of mitochondrial membrane potential and lower levels of microsomal triglyceride transfer protein in the HepG2 cells[2]. Tebuconazole (0-750 μM, 24 hours) decreases MAC-T cells viability and proliferation and induced mitochondria-mediated apoptotic MAC-T cell death by activating ER stress[3]. Tebuconazole (0-100 μM, 24 hours) induces dose-dependent cell death in H9c2 cardiomyoblasts and in adult rat ventricular myocytes (ARVM)[4]. Tebuconazole (30-60 μM, 24 hours) induces DNA damage and ROS generation and lipid peroxidation in H9c2 cells[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[2]

Cell Line:	HepG2 cells
Concentration:	20,40,80 μM
Incubation Time:	1–12 hours
Result:	Increased the nuclear translocation of peroxisome proliferator-activated receptors and the expression of cluster of differentiation 36, fatty acid transport protein (FATP) 2, FATP5, and carnitine palmitoyltransferase 1.

Apoptosis Analysis[3]

Cell Line:	Bovine mammary gland epithelial cells (MAC-T cells)
Concentration:	100,150,200,250,500,750 μM
Incubation Time:	24 hours
Result:	Decreased cells viability and proliferation and activates apoptotic cell death via the upregulation of pro-apoptotic proteins, such as cleaved caspases 3 and 8 and BAX. Induced loss of mitochondrial membrane potential in MAC-T cells. Induced mitochondria-mediated apoptotic MAC-T cell death by activating ER stress. Induced endoplasmic reticulum (ER) stress via the upregulation of Bip/GRP78; PDI; ATF4; CHOP; and ERO1-Lα.

In Vivo	Tebuconazole (TEB) (10-50 mg/kg, p.o., once daily for 28 days) induces a multiplicity of CYPs and oxidative stress in liver; inhibits testicular P450 and glutathione S-transferase activities; and produces anti-androgenic effects in male rats[5]. Tebuconazole (25-100 mg/kg, p.o., daily for 10 days) causes the proliferation of fetal Leydig cells and increases fetal serum testosterone and progesterone levels in gestational rat[6]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Wistar rats[5]
Dosage:	10, 25, and 50 mg/kg
Administration:	p. o. once daily for 28 days
Result:	Induced CYP1A1/2, CYP2B1/2, CYP2E1, and CYP3A proteins in liver.

	Decreased glutathione content and increased glutathione S-transferase, superoxide dismutase, catalase, and glutathione peroxidase activities in liver . Increased superoxide dismutase activities in kidney and testis. Decreased glutathione S-transferase activity in testis . Decreased serum testosterone concentration and cauda epididymal sperm count .

Animal Model:	Male and female Sprague-Dawley rats[6]
Dosage:	25, 50, and 100 mg/kg
Administration:	Oral gavage (p.o.), for 10 days
Result:	Increased fetal serum testosterone and progesterone levels. Increased the number of fetal Leydig cells per testis without inducing cell aggregation. Up-regulated the expression levels of Star, Cyp11a1, Hsd17b3, and Fshr. Increased phosphorylation of AKT1, ERK1/2, and mTOR, the level of BCL2, as well as the decrease of Beclin1, LC3B, and BAX.

REFERENCES

[1]. Kwon HC, et.al. Tebuconazole Fungicide Induces Lipid Accumulation and Oxidative Stress in HepG2 Cells. Foods. 2021 Sep 22;10(10):2242.

[2]. Lee WY, et.al. Tebuconazole Induces ER-Stress-Mediated Cell Death in Bovine Mammary Epithelial Cell Lines. Toxics. 2023 Apr 21;11(4):397.

[3]. Ben Othmène Y,et.al. Tebuconazole induces ROS-dependent cardiac cell toxicity by activating DNA damage and mitochondrial apoptotic pathway. Ecotoxicol Environ Saf. 2020 Nov;204:111040.

[4]. Yang JD, et.al. Effects of tebuconazole on cytochrome P450 enzymes, oxidative stress, and endocrine disruption in male rats. Environ Toxicol. 2018 Jun 19.

[5]. Ma F, et.al. Gestational exposure to tebuconazole affects the development of rat fetal Leydig cells. Chemosphere. 2021 Jan;262:127792.

[6]. Warrilow AG, et al. Azole affinity of sterol 14α-demethylase (CYP51) enzymes from Candida albicans and Homo sapiens. Antimicrob Agents Chemother. 2013 Mar;57(3):1352-60.

Caution: Product has not been fully validated for medical applications. For research use only.

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