Terfenadine

Cat. No.: HY-B1193

CAS No.: 50679-08-8

Molecular Formula: $C_{32}H_{41}NO_2$

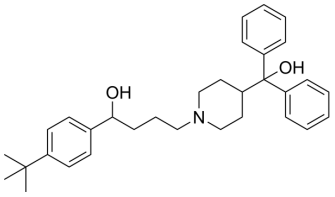
Molecular Weight: 471.67

Target: Potassium Channel; Histamine Receptor; Na+/Ca2+ Exchanger; Caspase; Apoptosis

Pathway: Membrane Transporter/Ion Channel; GPCR/G Protein; Immunology/Inflammation; Neuronal Signaling; 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 (106.01 mM)

H_2O : 0.67 mg/mL (1.42 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.1201 mL	10.6006 mL	21.2013 mL
5 mM	0.4240 mL	2.1201 mL	4.2403 mL
10 mM	0.2120 mL	1.0601 mL	2.1201 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 (5.30 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.30 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.30 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Terfenadine ((±)-Terfenadine) is a potent open-channel blocker of hERG with an IC_{50} of 204 nM[1]. Terfenadine, an H1 histamine receptor antagonist, acts as a potent apoptosis inducer in melanoma cells through modulation of Ca^{2+} homeostasis. Terfenadine induces ROS-dependent apoptosis, simultaneously activates Caspase-4, -2, -9[2].

IC_{50} & Target	H_1 Receptor	Caspase-4	Caspase-2	Caspase-9

In Vitro

Terfenadine ((±)-Terfenadine) (4-20 μM; 24 hours) induces dose and time-dependent apoptosis on A375 melanoma cells. The IC_{50} after 24 h of TEF treatment in complete medium was 10.4 μM for A375 cells, 9.9 μM for Hs294T cells and 9.6 for HT144 cells[2].

?Terfenadine (2-10 μM; 8 hours) induces dose-dependent cytotoxicity[2].

?Terfenadine (10 μM; 8 hours) causes a massive vacuolization of the cytoplasm and autophagic vacuoles of both double and multiple membranes and at various stages. Terfenadine induces autophagy by ROS-dependent and -independent mechanisms[2].

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

Apoptosis Analysis[2]

Cell Line:	A375, HT144 and Hs294T cells
Concentration:	4, 8, 12, 16, 20 μM
Incubation Time:	24 hours
Result:	Induced dose and time-dependent apoptosis.

Cell Cytotoxicity Assay[2]

Cell Line:	A375 melanoma cells
Concentration:	2, 4, 6, 8, 10 μM
Incubation Time:	8 hours
Result:	Induces dose-dependent cytotoxicity.

Cell Autophagy Assay[2]

Cell Line:	A375 cells
Concentration:	10 μM
Incubation Time:	8 hours
Result:	Caused a massive vacuolization of the cytoplasm and autophagic vacuoles of both double and multiple membranes and at various stages.

In Vivo

Terfenadine (p.o.; 40 mg/kg; for 16 days) produces a significant inhibition of tumour growth rate and enhances the anti-cancer effect of EPI in chemo-resistant NSCLC xenograft models[3].

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

Animal Model:	6-week-old male BALB/cA-nu mice[3]
Dosage:	40 mg/kg
Administration:	P.o.; for 16 days
Result:	Produced a significant inhibition of tumour growth rate.

CUSTOMER VALIDATION

- Front Immunol. 2023 Nov 23:14:1282710.
- Front Immunol. 2023 Nov 23;14:1282710.

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REFERENCES

[1]. Kamiya K, et al. Molecular determinants of hERG channel block by terfenadine and cisapride. J Pharmacol Sci. 2008 Nov;108(3):301-307.

[2]. Nicolau-Galmés F, et al. Terfenadine induces apoptosis and autophagy in melanoma cells through ROS-dependent and -independent mechanisms. Apoptosis. 2011 Dec;16(12):1253-67.

[3]. An L, et al. Terfenadine combined with epirubicin impedes the chemo-resistant human non-small cell lung cancer both in vitro and in vivo through EMT and Notch reversal. Pharmacol Res. 2017 Oct;124:105-115.

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