E3330

Cat. No.: HY-19357

CAS No.: 136164-66-4

Molecular Formula: $C_{21}H_{30}O_6$

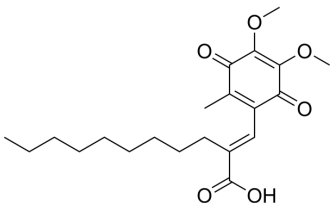
Molecular Weight: 378.46

Target: DNA/RNA Synthesis; NF-κB; AP-1; HIF/HIF Prolyl-Hydroxylase; VEGFR; Reactive Oxygen Species

Pathway: Cell Cycle/DNA Damage; NF-κB; Immunology/Inflammation; Metabolic Enzyme/Protease; Protein Tyrosine Kinase/RTK

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 : 120 mg/mL (317.07 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6423 mL	13.2114 mL	26.4229 mL
	5 mM	0.5285 mL	2.6423 mL	5.2846 mL
	10 mM	0.2642 mL	1.3211 mL	2.6423 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 (5.50 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.50 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.50 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

E3330 (APX-3330) is a direct, orally active and selective inhibitor of Ape-1 (apurinic/apyrimidinic endonuclease 1)/Ref-1 (redox factor-1) redox. E3330 is able to impair tumor growth and blocks the activity of NF-κB, AP-1, and HIF-1α in pancreatic cancer. E3330 shows anticancer activities[1][2][3][4][5].

IC_{50} & Target

Ape-1, Ref-1[1]

In Vitro

E3330 (0-50 μM, 48 h) inhibits the growth of HUVECs, PCECs and EPCs[1].
E3330 (0-5 μM) reduces secreted and intracellular VEGF (vascular endothelial growth factor) expression by pancreatic cancer cells, while concomitantly downregulating the cognate receptor Flk-1/KDR on PCECs[1].
E3330 (0-1 μM) inhibits the differentiation of bone marrow mesenchymal stem cells (BM-MSCs) into $CD31^+$ cells of endothelial lineage[1].
E3330 (0-50 μM, 72 h) decreases cell viability in H1975 cells about 45% at 50 μM[2].
E3330 (0-30 μM) inhibits the growth and migration of pancreatic cancer cells[3].
E3330 (0-30 μM) significantly enhances intracellular ROS level and inhibits CD44 expression in PANC1 cells[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	Human umbilical vein endothelial cells (HUVECs), murine pancreatic cancer associated endothelial cells (mPCECs), human endothelial progenitor cells (hEPCs)
Concentration:	1, 5, and 10 μM (HUVECs); 1. 5, 10. 20. 30, 40, and 50 μM (mPCECs); 1, 5, 10, 15, 20, 25, and 30 μM (hEPCs)
Incubation Time:	48 h
Result:	Inhibited the growth of HUVECs, PCECs and EPCs.

Western Blot Analysis[1]

Cell Line:	mPCECs
Concentration:	1, 5, and 10 μM
Incubation Time:	48 h
Result:	Inhibited the growth of HUVECs, PCECs and EPCs.

Cell Viability Assay[2]

Cell Line:	H1975 cells
Concentration:	0, 5, 10, 20, 30, 40, and 50 μM
Incubation Time:	72 h
Result:	Showed decreased cell viability in about 45% at 50 μM.

In Vivo

E3330 (25 mg/kg, Orally, 5 daily, five days each week for three weeks) is neuroprotective against cisplatin-induced alterations in capsaicin-induced vasodilation[4].
E3330 (0-100 mg/kg, Orally, once) attenuates the liver injury when given at 1 h, 6 h or 12 h after galactosamine challenge[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Sprague-Dawley rats (adult male, 150-175 g)[4]
Dosage:	25 mg/kg
Administration:	Orally, 5 daily, five days each week for three weeks
Result:	Attenuated the cisplatin-induced decrease in capsaicin-induced vasodilatation in the rat hindpaw.

Animal Model:	Male Fischer (F344/DuCrj) rats (160-190 g)[5]
Dosage:	0, 10, 30, and 100 mg/kg
Administration:	Orally, 1 h, 6 h or 12 h after galactosamine challenge
Result:	Attenuated the liver injury when given at 1 h, 6 h or 12 h after galactosamine challenge.

CUSTOMER VALIDATION

- Brain Behav Immun. 2023 Feb 6;S0889-1591(23)00029-6.
- Cell Death Dis. 2021 May 14;12(5):490.

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REFERENCES

[1]. Zou GM, et al. The Ape-1/Ref-1 redox antagonist E3330 inhibits the growth of tumor endothelium and endothelial progenitor cells: therapeutic implications in tumor angiogenesis. J Cell Physiol. 2009 Apr;219(1):209-18.

[2]. Manguinhas R, et al. Impact of the APE1 Redox Function Inhibitor E3330 in Non-small Cell Lung Cancer Cells Exposed to Cisplatin: Increased Cytotoxicity and Impairment of Cell Migration and Invasion. Antioxidants (Basel). 2020 Jun 24;9(6):550.

[3]. Zou GM, et al. Small-molecule inhibitor of the AP endonuclease 1/REF-1 E3330 inhibits pancreatic cancer cell growth and migration. Mol Cancer Ther. 2008 Jul;7(7):2012-21.

[4]. Kelley MR, et al. Role of the DNA base excision repair protein, APE1 in cisplatin, oxaliplatin, or carboplatin induced sensory neuropathy. PLoS One. 2014 Sep 4;9(9):e106485.

[5]. Nagakawa J, et al. Protective effect of E3330, a novel quinone derivative, in galactosamine-induced hepatitis in rats. J Pharmacol Exp Ther. 1993 Jan;264(1):496-500.

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