5,6-Dichlorobenzimidazole riboside

Cat. No.: HY-14392

CAS No.: 53-85-0

Molecular Formula: $C_{12}H_{12}Cl_2N_2O_4$

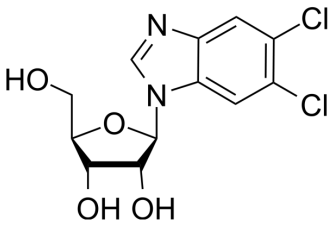
Molecular Weight: 319.14

Target: CDK; Apoptosis

Pathway: Cell Cycle/DNA Damage; Apoptosis

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMF : 100 mg/mL (313.34 mM; Need ultrasonic)
DMSO : 100 mg/mL (313.34 mM; Need ultrasonic)
Ethanol : 7.69 mg/mL (24.10 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.1334 mL	15.6671 mL	31.3342 mL
5 mM	0.6267 mL	3.1334 mL	6.2668 mL
10 mM	0.3133 mL	1.5667 mL	3.1334 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 (7.83 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.83 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.83 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

5,6-Dichlorobenzimidazole riboside (DRB) is a nucleoside analog that inhibits several carboxyl-terminal domain kinases, including casein kinase II and cell cycle-dependent kinases (CDK). 5, 6-dichlorobenzimidazole riboside has antitumor activity. 5, 6-dichlorobenzimidazole riboside can induce apoptosis[1][2][3][4][5][6][7].

In Vitro

5,6-Dichlorobenzimidazole riboside (10-80 μg/mL, 72 h) induces p53-dependent apoptosis of human colon cancer cells by blocking RNA synthesis[5].

5,6-Dichlorobenzimidazole riboside (10-100 μM, 72 h) induces apoptosis of human MCF-7 breast cancer cells by regulating Mcl-1 and BclxL. And activates members of the caspase family in a time - and dose-dependent manner[6].

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

Western Blot Analysis[5]

Cell Line:	LS174T, HT29, SW48
Concentration:	80 μg/mL
Incubation Time:	24 h
Result:	Decreased incorporation of [5,6-3H] uridine and increased level of p53 protein.

Cell Viability Assay[6]

Cell Line:	MCF-7, T-47D
Concentration:	10, 50, 75,100 μM
Incubation Time:	72 h
Result:	Inhibited cell-growth in a dose-dependent manner. Resulted in a higher early apoptotic population (5.7 ± 1.1 vs. 2 ± 0.4%) and late apoptotic population (15.9 ± 2.4 vs. 7.7 ± 0.9%) at a concentration of 75 μM.

Western Blot Analysis[6]

Cell Line:	MCF-7
Concentration:	75 μM
Incubation Time:	0.5, 2, 6, 10 h
Result:	Reduced Mcl-1 protein levels in a time-dependent manner and increased the level of p53 after 6 h.

In Vivo

5,6-Dichlorobenzimidazole riboside (2.0-3.0 mg/0.25 mL water, intraperitoneally injected twice a day for 5 days) does not inhibit the multiplication of the PR8 strain of influenza virus in mice[7].

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

CUSTOMER VALIDATION

- Cell Rep. 2022 Oct 25;41(4):111546.

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REFERENCES

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[6]. Schang LM. Cyclin-dependent kinases as cellular targets for antiviral drugs. J Antimicrob Chemother. 2002 Dec;50(6):779-92.

[7]. te Poele RH, et al. RNA synthesis block by 5, 6-dichloro-1-beta-D-ribofuranosylbenzimidazole (DRB) triggers p53-dependent apoptosis in human colon carcinoma cells. Oncogene. 1999 Oct 14;18(42):5765-72.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA