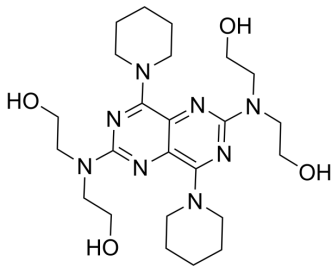
Dipyridamole

Cat. No.:	HY-B0312
CAS No.:	58-32-2
Molecular Formula:	$C_{24}H_{40}N_8O_4$
Molecular Weight:	504.63
Target:	Phosphodiesterase (PDE); Apoptosis
Pathway:	Metabolic Enzyme/Protease; Apoptosis
Storage:	4°C, protect from light * In solvent : -80°C, 1 year; -20°C, 6 months (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (99.08 mM)
H_2O : < 0.1 mg/mL (insoluble)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.9816 mL	9.9082 mL	19.8165 mL
	5 mM	0.3963 mL	1.9816 mL	3.9633 mL
	10 mM	0.1982 mL	0.9908 mL	1.9816 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 (4.95 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.95 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.95 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Dipyridamole is an orally active phosphodiesterase (PDE) inhibitor. Dipyridamole also is an antiplatelet agent used in secondary prophylaxis against stroke. Dipyridamole can induce cancer cell-specific apoptosis[1][2][3].

IC_{50} & Target

PDE

In Vitro

Dipyridamole (5 μM; 15 min) results in a 2.5-fold increase in intracellular cAMP levels in OCI-AML-3 cells[2].
?Dipyridamole (5 μM; 48 h) with the statin combination induces apoptosis in primary AML cells[2].

?Dipyridamole (5 μM; 48 h) possesse cAMP/PKA-independent activity against statininduced SREBP2 activation[2].

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

Apoptosis Analysis[2]

Cell Line:	AML (OCI-AML-2, OCI-AML-3) cell line
Concentration:	5 μM
Incubation Time:	48 h
Result:	Induced apoptosis with the combination of fluvastatin and dipyridamole, cilostazol, forskolin, or dbcAMP in OCI-AML-2 and OCI-AML-3 cells.

RT-PCR[2]

Cell Line:	LP1 cell line
Concentration:	5 μM
Incubation Time:	16 h
Result:	Increased the sensibility of cancer cells to statin-induced apoptosis.

In Vivo

Dipyridamole (10 mg/kg; p.o. once daily for 18 d) mitigates tumor growth, ameliorated concurrent alterations in blood circulation and tumor tissues, and platelet infiltration in tumor tissues[3].

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

Animal Model:	C57BL/6-LLC tumor-bearing mice models[3]
Dosage:	10 mg/kg
Administration:	Oral gavage; 10 mg/kg; once daily for 18 days
Result:	Mitigated tumor growth in tumor-bearing mice.

CUSTOMER VALIDATION

- Nat Cancer. 2022 Aug;3(8):945-960.
- Mediators Inflamm. 2023 Jul 19.

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REFERENCES

[1]. Kerndt CC, Nagalli S. Dipyridamole. 2021 Nov 25. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan–. PMID: 32119342.

[2]. Longo, Joseph, etal. Cyclic AMP-hydrolyzing phosphodiesterase inhibitors potentiate statin-induced cancer cell death. Molecular oncology vol. 14,10 (2020): 2533-2545

[3]. Wang, Jiaan-Der, etal. Exosomal HMGB1 Promoted Cancer Malignancy. Cancers vol. 13,4 877. 19 Feb. 2021.

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

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