## Dip

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)	

### DMSO : ≥ 50 mg/mL (99.08 mM) In Vitro $H_2O: < 0.1 \text{ mg/mL}$ (insoluble) \* "≥" means soluble, but saturation unknown. Mass Solvent 10 mg 1 mg 5 mg Concentration Preparing 9.9082 mL 1 mM 1.9816 mL 19.8165 mL **Stock Solutions** 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- $\beta$ -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

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 <sup>[1][2][3]</sup> .			
IC <sub>50</sub> & Target	PDE			
In Vitro	Dipyridamole (5 $\mu$ M; 15 min) results in a 2.5-fold increase in intracellular cAMP levels in OCI-AML-3 cells <sup>[2]</sup> . ?Dipyridamole (5 $\mu$ M; 48 h) with the statin combination induces apoptosis in primary AML cells <sup>[2]</sup> .			

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oyridamole			
No.:	HY-B0312		
Not	58-32-2		

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# Product Data Sheet

HO

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	?Dipyridamole (5 μM; 48 h) possesse cAMP/PKA-independent activity against statininduced SREBP2 activation <sup>[2]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only. Apoptosis Analysis <sup>[2]</sup>		
	Cell Line:	AML (OCI-AML-2, OCI-AML-3) cell line	
	Concentration:	5 μΜ	
	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 <sup>[2]</sup>		
	Cell Line:	LP1 cell line	
	Concentration:	5 μΜ	
	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 <sup>[3]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.		
	Animal Model:	C57BL/6-LLC tumor-bearing mice models <sup>[3]</sup>	
	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.
- Research Square Preprint. 2024 Apr 9.
- 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.

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