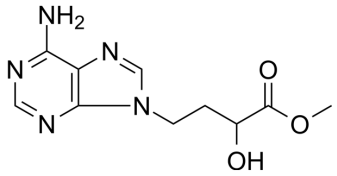
DZ2002

Cat. No.:	HY-18620
CAS No.:	33231-14-0
Molecular Formula:	$C_{10}H_{13}N_5O_3$
Molecular Weight:	251.24
Target:	Others
Pathway:	Others
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 : ≥ 61 mg/mL (242.80 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.9803 mL	19.9013 mL	39.8026 mL
	5 mM	0.7961 mL	3.9803 mL	7.9605 mL
	10 mM	0.3980 mL	1.9901 mL	3.9803 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.95 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.95 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

DZ2002 is an orally active, reversible and low-cytotoxic type III SAHH inhibitor (K_i=17.9 nM), with good immunosuppressive activity. DZ2002 prevents the development of experimental dermal fibrosis by reversing the profibrotic phenotype of various cell types. DZ2002 can be used in studies of autoimmune diseases such as lupus syndrome and systemic sclerosis[1][2].

In Vitro

DZ2002 (0.1, 1, 10 μM; 96 h) inhibits the mixed lymphocyte reaction (MLR) response[1].

DZ2002 (0.1, 1, 10 μM; 24 h) inhibits IL-12 and TNF-α production from both mouse peritoneal exudate cells and humanTHP-1 Cells[1].

DZ2002 (0.1, 1, 10 μM; 64 h) inhibits expression of B7 (CD80/CD86) on differentiated THP-1 cells[1].

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

Cell Proliferation Assay[1]

Cell Line:	BALB/c and C57BL/6 splenocytes (Mitomycin C-pretreated; mixed lymphocyte)
Concentration:	0.1, 1, 10 μM
Incubation Time:	96 h
Result:	Suppressed the MLR by 24.5, 42.3, and 46.0% at dosages of 0.1, 1, and 10 μM, respectively.

Cell Viability Assay[1]

Cell Line:	TG-stimulated mouse peritoneal macrophages and human THP-1 cells
Concentration:	0.1, 1, 10 μM
Incubation Time:	24 h
Result:	Significantly blocked IL-12 p40 production from ~1800 pg/mL in untreated cells to ~850 pg/ml at 10 μM, and drastically reduced the active p70 form from ~1200 pg/mL in untreated cells to ~50 pg/mL. Reduced TNF-α level by 45%.

Cell Viability Assay[1]

Cell Line:	THP-1 cells
Concentration:	0.1, 1, 10 μM
Incubation Time:	64 h
Result:	Dramatically down-regulated CD80 and, in particular, CD86 expression in a dose-dependent manner.

In Vivo

DZ2002 (2, 10, 50 mg/kg; i.p.; twice) blocks the DNFB-induced DTH response. (DNFB-induced DTH is a Th1 cell-mediated immune response, in which IL-12 is highly expressed and macrophages have been shown to play an important role)[1].
DZ2002 (0.08, 2 mg/kg; i.p.; single daily for 7 days) significantly suppresses a delayed-type hypersensitivity reaction as well as antibody secretion[1].
DZ2002 (50, 100 mg/kg; p.o.; single daily for 4 weeks) exerts a potent anti-fibrotic effect on dermal fibrosis by reducing the production of collagen, facilitating its degradation and regulating expression of various soluble factors in SSc mice model[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male and female BALB/c and C57BL/6 mice (6 to 8-week-old; DNFB-induced ear swelling model)[1].
Dosage:	2, 10, 50 mg/kg
Administration:	Intraperitoneal injection; twice (1 h before and 24 h after challenge)
Result:	Suppressed ear swelling by 19.1, 28.7, and 33.1%, respectively and in a dose-dependent manner.

Animal Model:	Male and female BALB/c and C57BL/6 mice (6 to 8-week-old; DNFB-induced ear swelling model)[1].

Dosage:	0.08, 2 mg/kg
Administration:	Intraperitoneal injection; single daily for 7 days.
Result:	Inhibited hemolysis by 24.5 and 18.4% at doses of 0.08 and 2 mg/kg, respectively, thus decreasing anti-SRBC antibody production in vivo.

Animal Model:	Wild-type C57BL/6 mice (8 to 12-week-old; BLM-induced mice model of SSc)[2].
Dosage:	50, 100 mg/kg
Administration:	Oral gavage; single daily for 4 weeks.
Result:	Significantly decreased skin thickness and dermal thickness in BLM-induced mice. Significantly reduced collagen accumulation and α-SMA expression in the dermis of mice and suppressed the mRNA expression of vascular endothelial growth factor (VEGF) in mice skin tissue. Notably reduced collagen content and mRNA expression of the Col1a1 and Col1a2 while promoting that of the matrix metalloproteinase-13 (MMP-13) in the lesional skin of BLM-induced mice.

CUSTOMER VALIDATION

- Ann Transl Med. 2020 Dec;8(23):1582.

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REFERENCES

[1]. Wu QL, et al. Inhibition of S-adenosyl-L-homocysteine hydrolase induces immunosuppression. J Pharmacol Exp Ther. 2005 May;313(2):705-11.

[2]. Zhang Z, et al. DZ2002 ameliorates fibrosis, inflammation, and vasculopathy in experimental systemic sclerosis models. Arthritis Res Ther. 2019 Dec 16;21(1):290.

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