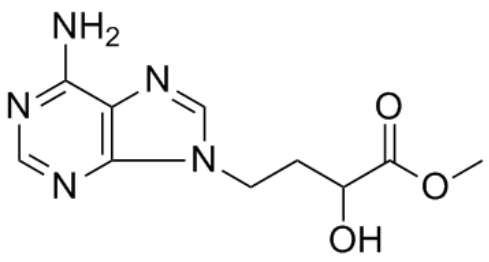
Product Data Sheet

Product Name: DZ2002
Cat. No.: GC35920

Chemical Properties

Cas. No.	33231-14-0		
SMILES	OC(C(OC)=O)CCN1C(N=CN=C2N)=C2N=C1		
Formula	$C_{10}H_{13}N_5O_3$	M.Wt	251.24
Solubility	DMSO: ≥ 61 mg/mL (242.80 mM)	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure			



Background

DZ2002 is a potent and reversible S-Adenosyl-L-homocysteine Hydrolase(SAHH; AdoHcy Hydrolase) inhibitor with Ki of 17.9 nM.IC50 value: 17.9 nM(Ki) [1]Target: AdoHcy Hydrolase inhibitorin vitro: The cytotoxicity of DZ2002 is significantly less than DHCaA with an IC50 of 100 to 600 μM compared with 6 to 14 μM and shows very little cytotoxicity up to 100 μM. DZ2002 had little effects on lymphocyte proliferation (0.1 μM = 150,604 ± 13,862, 1 μM = 159,894 ± 11,152, and 10 μM = 136,157 ± 21,943 cpm) versus untreated Con A-stimulated cells (168,725 ± 8025 cpm). Similarly, little effect was seen in regards to IL-2 production from DZ2002-treated cells (0.1 μM =

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1,838 ± 88, 1 μM = 1,793 ± 58, and 10 μM = 1,731 ± 36 pg/ml) versus untreated Con A-stimulated cells (1,806 ± 43 pg/ml). Although DZ2002 had little effect when T cells were stimulated with Con A, DZ2002 suppressed the MLR by 24.5, 42.3, and 46.0% at dosages of 0.1, 1, and 10 μM, respectively [1]. DZ2002 (500 μmol/L) significantly suppressed TLR agonists-stimulated up-regulation in IL-6, IL-12p40, TNF-α, and IgG and IgM secretion as well as in HLA-DR and CD40 expression of dendritic cells among human PBMCs in vitro. DZ2002 (100 μmol/L) also significantly suppressed TLR agonists-stimulated up-regulation in IL-6 and IL-23p19 production in murine BMDCs, and prevented Th17 differentiation and suppressed IL-17 secretion by the T cells in a BMDC-T cell co-culture system [3].in vivo: As compared with controls, consecutive 7-day i.p. injections of DZ2002 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 [1]. Male C57BL/6 mice immunized with ovalbumin (OVA) were treated with DZ2002 (1, 5, and 25 mg/kg/day) after which lymphocyte proliferation, cytokine production, and IgG responses to OVA were monitored. Administration of DZ2002 dose dependently suppressed OVA-specific lymphocyte proliferation and anti-OVA IgG production compared with controls [2]. Treatment of the mice with DZ2002 significantly attenuated the progression of glomerulonephritis and improved the overall health. In ex vivo studies, treatment of the mice with DZ2002 suppressed the development of pathogenic Th17 cells, significantly decreased IL-17, TGF-β, IL-6, and IL-23p19 production and impeded activation of the STAT3 protein and JNK/NF-κB signaling in splenocytes [3].

[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]. Fu YF, et al. S-adenosyl-L-homocysteine hydrolase inactivation curtails ovalbumin-induced immune responses. J Pharmacol Exp Ther. 2006 Mar;316(3):1229-37. [3]. He SJ, et al. Therapeutic effects of DZ2002, a reversible SAHH inhibitor, on lupus-prone NZB×NZW F1 mice via interference with TLR-mediated APC response. Acta Pharmacol Sin. 2014 Feb;35(2):219-29.

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