

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

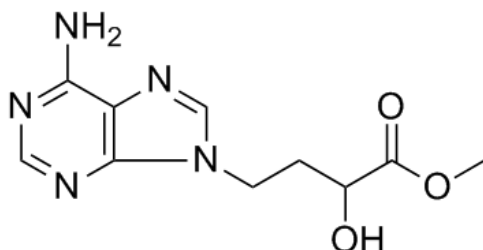
Formula $C_{10}H_{13}N_5O_3$ M.Wt 251.24

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 K_i of 17.9 nM. IC_{50} value: 17.9 nM (K_i) [1] Target: AdoHcy Hydrolase inhibitor in vitro: The cytotoxicity of DZ2002 is significantly less than DHCaA with an IC_{50} 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 = $150,604 \pm 13,862$, 1 μM = $159,894 \pm 11,152$, and 10 μM = $136,157 \pm 21,943$ cpm) versus untreated Con A-stimulated cells ($168,725 \pm 8025$ cpm). Similarly, little effect was seen in regards to IL-2 production from DZ2002-treated cells (0.1 μM =

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

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1,838 $\pm$ 88, 1 μ M = 1,793 $\pm$ 58, and 10 μ M = 1,731 $\pm$ 36 pg/ml) versus untreated Con A-stimulated cells (1,806 $\pm$ 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 μ M, respectively [1]. DZ2002 (500 μ mol/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 μ mol/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- κ B 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 $\times$ NZW F1 mice via interference with TLR-mediated APC response. *Acta Pharmacol Sin*. 2014 Feb;35(2):219-29.

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