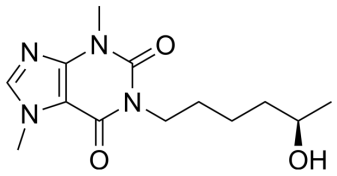
(R)-Lisofylline

Cat. No.:	HY-109854A
CAS No.:	100324-81-0
Molecular Formula:	$C_{13}H_{20}N_4O_3$
Molecular Weight:	280.32
Target:	STAT
Pathway:	JAK/STAT Signaling; Stem Cell/Wnt
Storage:	-20°C, stored under nitrogen * In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (356.74 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.5674 mL	17.8368 mL	35.6735 mL
	5 mM	0.7135 mL	3.5674 mL	7.1347 mL
	10 mM	0.3567 mL	1.7837 mL	3.5674 mL

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

BIOLOGICAL ACTIVITY

Description

(R)-Lisofylline ((R)-Lisophylline) is a (R)-enantiomer of the metabolite of Pentoxifylline with anti-inflammatory properties. (R)-Lisofylline is a lysophosphatidic acid acyltransferase inhibitor with an IC_{50} of 0.6 μM and interrupts IL-12 signaling-mediated STAT4 activation. (R)-Lisofylline has the potential for type 1 diabetes, autoimmune disorders research[1][2].

IC_{50} & Target

IC50: 0.6 μM (Lysophosphatidic acid acyltransferase)[1]
STAT4[1]

In Vitro

(R)-Lisofylline blocks IL-12-driven Th1 differentiation and T cell proliferation in vitro, yet has no effect on IL-12 secretion from APCs ex vivo or in vitro[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

(R)-Lisofylline reduces the impairment of insulin secretion induced by IL-1β in cultured rat islet cells, suppresses IFN-γ production, the onset of diabetes, and macrophage infiltration into islets from NOD mice, as well as Lisofylline improves insulin response and lowers glucose levels in Streptozotocin-treated rats after the oral glucose tolerance test[1].
(R)-Lisofylline prevents β cell dysfunction in NOD mice by inhibition of STAT4 phosphorylation which interrupts IL-12 signaling. (R)-Lisofylline ameliorates experimental allergic encephalomyelitis in mice[1].

(R)-Lisofylline also improves survival in mice injected with a lethal dose of LPS and ameliorates sepsis-induced lung injury in minipigs. In rats given IL-1 intratracheally (R)-Lisofylline pretreatment reduces lung leak but does not decrease neutrophil accumulation in lungs[1].

(R)-Lisofylline also suppresses release of TNF-α in vivo in mice and ex vivo in human blood stimulated with endotoxin derived from Salmonella or Escherichia coli[1].

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

REFERENCES

[1]. Elżbieta Wyska, et al. Physiologically Based Modeling of Lisofylline Pharmacokinetics Following Intravenous Administration in Mice. Eur J Drug Metab Pharmacokinet. 2016 Aug;41(4):403-12.

[2]. B M Hybertson, et al. Lisofylline Prevents Leak, but Not Neutrophil Accumulation, in Lungs of Rats Given IL-1 Intratracheally. J Appl Physiol (1985). 1997 Jan;82(1):226-32.

[3]. J J Bright, et al. Prevention of Experimental Allergic Encephalomyelitis via Inhibition of IL-12 Signaling and IL-12-mediated Th1 Differentiation: An Effect of the Novel Anti-Inflammatory Drug Lisofylline. J Immunol. 1998 Dec 15;161(12):7015-22.

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