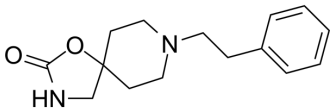
Fenspiride

Cat. No.:	HY-A0027A
CAS No.:	5053-06-5
Molecular Formula:	$C_{15}H_{20}N_2O_2$
Molecular Weight:	260.33
Target:	Histamine Receptor; Phosphodiesterase (PDE)
Pathway:	GPCR/G Protein; Immunology/Inflammation; Neuronal Signaling; Metabolic Enzyme/Protease
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Fenspiride, an orally active non-steroidal antiinflammatory agent, is an antagonist of H1-histamine receptor. Fenspiride inhibits phosphodiesterase 3 (PDE3), phosphodiesterase 4 (PDE4) and phosphodiesterase 5 (PDE5) activities with -log IC_{50} values of 3.44, 4.16 and approximately 3.8, respectively. Fenspiride can be used for the research of respiratory diseases[1][2][3].			
IC_{50} & Target	H_1 Receptor	PDE3	PDE4	PDE5
In Vitro	Fenspiride (around 100 μM) inhibits histamine-induced contraction of isolated guinea pig trachea[2]. Fenspiride (≤1000 μM) produces less than 25% inhibition of phosphodiesterase 1 and phosphodiesterase 2 activities[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.			

In Vivo

Fenspiride (60 mg/kg; p.o. for 3 days) reduces the lipopolysaccharide-induced early rise of tumor necrosis factor concentrations in serum and in the bronchoalveolar lavage fluid (BALF) of the model of endotoxemia[3].

Fenspiride (60 mg/kg; p.o. for 3 days) reduces the lipopolysaccharide-induced primed stimulation of alveolar macrophages [3].

Fenspiride (60 mg/kg; p.o. for 3 days) reduces the increased serum concentrations of extracellular type II phospholipase A 2, the intensity of the neutrophilic alveolar invasion and the lethality due to the lipopolysaccharide[3].

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Animal Model:	Lipopolysaccharide-treated Male Dunkin-Hartley guinea-pigs weighing 400-600 g[3]
Dosage:	60 mg/kg
Administration:	orally for 3 days; pretreated
Result:	Reduced the lipopolysaccharide-induced early rise of tumor necrosis factor concentrations in serum (4.2 vs. 2.3 ng/ml) and in the BALF (55.7 vs. 19.7 ng/ml). Reduced the lipopolysaccharide-induced primed stimulation of alveolar macrophages, (1551.5 vs 771.5 pg/μg protein, P<0.05 for thromboxane B_2 and 12.6 vs. 3.6 pg/μg protein, P<0.05 for leukotriene C4). Reduced the increased serum concentrations of extracellular type II phospholipase A 2 (3.9 vs. 1.2 nmol/ml per min), the intensity of the neutrophilic alveolar invasion and the

	lethality due to the lipopolysaccharide.

REFERENCES

[1]. Matuszewska A, et al. Long-term administration of fenspiride has no negative impact on bone mineral density and bone turnover in young growing rats. Adv Clin Exp Med. 2019 Jun;28(6):771-776.

[2]. Cortijo J, et al. Effects of fenspiride on human bronchial cyclic nucleotide phosphodiesterase isoenzymes: functional and biochemical study. Eur J Pharmacol. 1998 Jan 2;341(1):79-86.

[3]. De Castro CM, et al. Fenspiride: an anti-inflammatory drug with potential benefits in the treatment of endotoxemia. Eur J Pharmacol. 1995 Dec 29;294(2-3):669-76.

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