Veliflapon

Cat. No.: HY-14165

CAS No.: 128253-31-6

Molecular Formula: $C_{23}H_{23}NO_3$

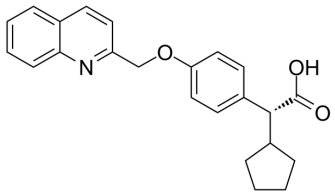
Molecular Weight: 361.43

Target: Leukotriene Receptor; FLAP

Pathway: GPCR/G Protein; Immunology/Inflammation

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (276.68 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.7668 mL	13.8339 mL	27.6679 mL
	5 mM	0.5534 mL	2.7668 mL	5.5336 mL
	10 mM	0.2767 mL	1.3834 mL	2.7668 mL

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

BIOLOGICAL ACTIVITY

Description

Veliflapon (BAY X 1005; DG-031) is an orally active and selective 5-lipoxygenase activating protein (FLAP) inhibitor[1]. Veliflapon inhibits the synthesis of the leukotrienes B4 and C4[2].

IC_{50} & Target

LTB_4 | LTC_4

In Vitro

Veliflapon (BAY X 1005; DG-031) effectively inhibits the synthesis of LTB4 in A23187-stimulated leukocytes from rats, mice and humans (IC_{50}s of 0.026, 0.039 and 0.22 μM, respectively) as well as the formation of LTC4 (IC_{50} of 0.021 μM) in mouse peritoneal macrophages stimulated with opsonized zymosan[3].

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

In Vivo

Veliflapon (BAY X 1005; DG-031; diet; 18.8 mg/kg/day for 16 weeks) inhibits atherogenesis[4].

Veliflapon after topical (18 μg/ear) and oral (48.7 mg/kg) administration has antiedematous effects in the arachidonic acid-induced mouse ear inflammation test[4].

Veliflapon is potent (11.8 and 6.7 mg/kg p.o. at 1 and 5 hours, respectively) and has a long duration of action (ED_{40} of 16 hours, 70 mg/kg p.o.) in the rat whole blood ex vivo leukotriene B4 inhibition assay[4].

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

Animal Model:	Female apoE/LDLR-DKO mouse model[4]
Dosage:	18.8 mg/kg
Administration:	Diet; per day during 16 weeks
Result:	Inhibited atherogenesis.

REFERENCES

[1]. Müller-Peddinghaus R, et al. BAY X1005, a new inhibitor of leukotriene synthesis: in vivo inflammation pharmacology and pharmacokinetics. J Pharmacol Exp Ther. 1993 Oct;267(1):51-7.

[2]. Fruchtmann R, et al. In vitro pharmacology of BAY X1005, a new inhibitor of leukotriene synthesis. Agents Actions. 1993 Mar;38(3-4):188-95.

[3]. Jawień J, et al. BAY x 1005 attenuates atherosclerosis in apoE/LDLR - double knockout mice. J Physiol Pharmacol. 2007 Sep;58(3):583-8.

[4]. Hatzelmann A, et al. Mode of action of the leukotriene synthesis (FLAP) inhibitor BAY X 1005: implications for biological regulation of 5-lipoxygenase. Agents Actions. 1994 Nov;43(1-2):64-8.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA