Quiflapon

Cat. No.: HY-10037

CAS No.: 136668-42-3

Molecular Formula: $C_{34}H_{35}ClN_2O_3S$

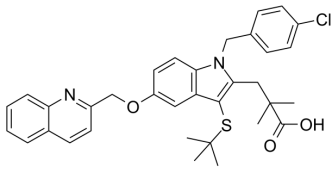
Molecular Weight: 587.17

Target: FLAP; Apoptosis

Pathway: Immunology/Inflammation; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (85.15 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.7031 mL	8.5154 mL	17.0308 mL
5 mM	0.3406 mL	1.7031 mL	3.4062 mL
10 mM	0.1703 mL	0.8515 mL	1.7031 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 2.5 mg/mL (4.26 mM); Suspended solution; Need ultrasonic and warming
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.26 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Quiflapon (MK-591) is a selective and specific 5-lipoxygenase-activating protein (FLAP) inhibitor with an IC_{50} of 1.6 nM in a FLAP binding assay. Quiflapon is also a potent and orally active Leukotriene biosynthesis (LT) inhibitor, shows IC_{50} values of 3.1 and 6.1 nM in intact human and elicited rat PMNLs, respectively. Quiflapon induces cell apoptosis[1][2].

IC_{50} & Target

IC50: 1.6 nM (FLAP)[1].

In Vitro

Quiflapon is a potent inhibitor of leukotriene (LT) biosynthesis in intact human and elicited rat polymorphonuclear leukocytes (PMNLs) (IC_{50} values 3.1 and 6.1 nM, respectively) and in human, squirrel monkey, and rat whole blood (IC_{50} values 510, 69, and 9 nM, respectively). Quiflapon has no effect on rat 5-lipoxygenase. Quiflapon has a high affinity for 5-

	lipoxygenase activating protein (FLAP) as evidenced by an IC_{50} value of 1.6 nM in a FLAP binding assay and inhibition of the photoaffinity labelling of FLAP by two different photoaffinity ligands. Inhibition of activation of 5-lipoxygenase was shown through inhibition of the translocation of the enzyme from the cytosol to the membrane in human PMNLs[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Quiflapon is a potent inhibitor of LT biosynthesis in vivo, first, following ex vivo challenge of blood obtained from treated rats and squirrel monkeys, second, in a rat pleurisy model, and, third, as monitored by inhibition of the urinary excretion of LTE4 in antigen-challenged allergic sheep. Inhibition of antigen-induced bronchoconstriction by Quiflapon is observed in inbred rats pretreated with methysergide, Ascaris-challenged squirrel monkeys, and Ascaris-challenged sheep (early and late phase response) [1]. Pups were treated with either vehicle or Quiflapon 10, 20, or 40 mg/kg subcutaneously daily for days 1-4, 5-9, or 10-14. On day 14, the lungs were inflated, fixed, and stained for histopathological and morphometric analyses. Hyperoxia groups treated with Quiflapon untreated hyperoxia groups showed definite evidence of aberrant alveolarization but no inflammation[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Brideau C, et al. Pharmacology of MK-0591 (3-[1-(4-chlorobenzyl)-3-(t-butylthio)-5-(quinolin-2-yl-methoxy)- indol-2-yl]-2,2-dimethyl propanoic acid), a potent, orally active leukotriene biosynthesis inhibitor. Can J Physiol Pharmacol. 1992 Jun;70(6):799-8

[2]. Park MS, et al. 5-Lipoxygenase-activating protein (FLAP) inhibitor MK-0591 prevents aberrant alveolarization in newborn mice exposed to 85% oxygen in a dose- and time-dependent manner. Lung. 2011 Feb;189(1):43-50.

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