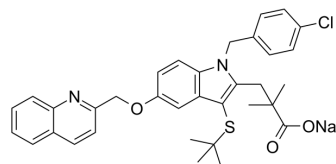


Quiflapon sodium

Cat. No.:	HY-50714
CAS No.:	147030-01-1
Molecular Formula:	C ₃₄ H ₃₄ ClN ₂ NaO ₃ S
Molecular Weight:	609.15
Target:	FLAP; Apoptosis
Pathway:	Immunology/Inflammation; Apoptosis
Storage:	4°C, sealed storage, away from moisture * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro	DMSO : 50 mg/mL (82.08 mM; Need ultrasonic)					
	H ₂ O : < 0.1 mg/mL (insoluble)					
	Preparing Stock Solutions	Solvent	Mass	1 mg	5 mg	10 mg
		Concentration				
		1 mM		1.6416 mL	8.2082 mL	16.4163 mL
5 mM			0.3283 mL	1.6416 mL	3.2833 mL	
10 mM		0.1642 mL	0.8208 mL	1.6416 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: ≥ 2.75 mg/mL (4.51 mM); Clear solution					
	2. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.75 mg/mL (4.51 mM); Clear solution					

BIOLOGICAL ACTIVITY

Description	Quiflapon sodium (MK-591 sodium) is a selective and specific 5-Lipoxygenase-activating protein (FLAP) inhibitor. Quiflapon sodium is an orally active Leukotriene biosynthesis inhibitor. Induces apoptosis.
In Vitro	Quiflapon sodium (MK591) and SB203580 are able to block SEB-induced human PBMC cell proliferation. Quiflapon sodium (MK591) down regulates three genes [for cathepsin L, IL-17 and guanylate binding protein (GBP)-2] that are up regulated by SEB ^[1] . Quiflapon sodium (MK591) undergoes apoptosis within hours of treatment. Quiflapon sodium also induces rapid activation of the stress kinase, c-Jun N-terminal kinase (JNK), which plays an important role in the apoptosis process. Quiflapon sodium triggers apoptosis in prostate cancer cells without inhibition of PI3K-Akt, or ERK. Moreover, Quiflapon sodium and LY294002 exert synergistic effect in inducing apoptosis in prostate cancer cells ^[2] . Quiflapon sodium (MK591) influences cAMP response element-binding protein but not Sp1 ^[4] .

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

In Vivo

Hyperoxia groups of mice treated with Quiflapon sodium (MK591) (20, 40 mg/kg) show alveolarization that resembles that of room air controls while untreated hyperoxia groups show definite evidence of aberrant alveolarization but no inflammation [3]. Comparison of the A β -immunopositive areas between the placebo and Quiflapon sodium (MK591) (320 mg/kg)-treated group reveals a statistically significant reduction of the amyloid burden in the treated mice. Quiflapon sodium also has a significant reduction in brain levels of IL-1 β . Mice treated with Quiflapon sodium show a statistically significant decrease in the steady-state levels of total CREB and its phosphorylated form at Ser133^[4].

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

PROTOCOL

Animal Administration ^[4]

The Tg2576 transgenic mice expressing human APP with the Swedish mutation (K670N/M671L) are used in these studies. They are genotyped by PCR analysis using tail DNA and kept in a pathogen-free environment, on a 12-hour light/dark cycle and have access to food and water ad libitum. All the experiments presented in this paper are performed with female mice. Starting at 7 months of age, mice are randomized to receive Quiflapon sodium (40 mg/kg weight) (n=11) or vehicle (n=9) in their chow diet for 8 months until they are 15 months old. Considering that each mouse eats on average 5 g/day of chow diet and the diet is formulated for 320 mg Quiflapon sodium per kg diet, the final dose of the active drug is approximately 40 mg/kg weight/day. During the study, mice in both groups gain weight regularly, and no significant difference in weight is detected between the two groups. No macroscopic effect on the overall general health is observed in the animals receiving the active treatment. Post-mortem examination shows no sign of macroscopic pathology in any of the organs considered (spleen, liver, thymus, ileum).

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

REFERENCES

- [1]. Mendis C, et al. Effect of 5-lipoxygenase inhibitor MK591 on early molecular and signaling events induced by staphylococcal enterotoxin B in human peripheral blood mononuclear cells. *FEBS J.* 2008 Jun;275(12):3088-98.
- [2]. Sarveswaran S, et al. MK591, a leukotriene biosynthesis inhibitor, induces apoptosis in prostate cancer cells: synergistic action with LY294002, an inhibitor of phosphatidylinositol 3'-kinase. *Cancer Lett.* 2010 May 28;291(2):167-76.
- [3]. 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.
- [4]. Chu J, et al. Involvement of 5-lipoxygenase activating protein in the amyloidotic phenotype of an Alzheimer's disease mouse model. *J Neuroinflammation.* 2012 Jun 14;9:127.

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

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