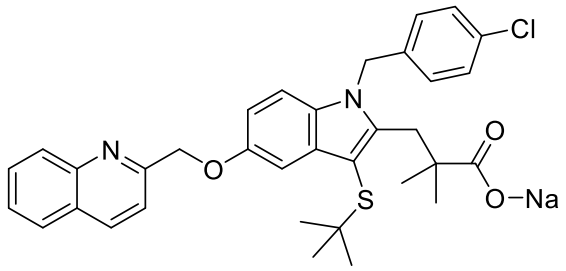
Product data sheet

MedKoo Cat#: 326735 Name: Quiflapon sodium CAS#: 147030-01-1 (sodium) Chemical Formula: $C_{34}H_{34}ClN_2NaO_3S$ Exact Mass: 608.1876 Molecular Weight: 609.1568	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C > 4 years
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product 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.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	50	82.08

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	1.64 mL	8.21 mL	16.42 mL
5 mM	0.33 mL	1.64 mL	3.28 mL
10 mM	0.16 mL	0.82 mL	1.64 mL
50 mM	0.03 mL	0.16 mL	0.33 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Flamand N, Lefebvre J, Surette ME, Picard S, Borgeat P. Arachidonic acid regulates the translocation of 5-lipoxygenase to the nuclear membranes in human neutrophils. J Biol Chem. 2006 Jan 6;281(1):129-36. doi: 10.1074/jbc.M506513200. Epub 2005 Nov 7. PMID: 16275640.
2. Surette ME, Krump E, Picard S, Borgeat P. Activation of leukotriene synthesis in human neutrophils by exogenous arachidonic acid: inhibition by adenosine A(2a) receptor agonists and crucial role of autocrine activation by leukotriene B(4). Mol Pharmacol. 1999 Nov;56(5):1055-62. doi: 10.1124/mol.56.5.1055. PMID: 10531413.

In vivo study

1. Cao RY, Adams MA, Habenicht AJ, Funk CD. Angiotensin II-induced abdominal aortic aneurysm occurs independently of the 5-lipoxygenase pathway in apolipoprotein E-deficient mice. Prostaglandins Other Lipid Mediat. 2007 Aug;84(1-2):34-42. doi: 10.1016/j.prostaglandins.2007.03.005. Epub 2007 Mar 24. PMID: 17643886.
2. Marleau S, Fruteau de Laclos B, Sanchez AB, Poubelle PE, Borgeat P. Role of 5-lipoxygenase products in the local accumulation of neutrophils in dermal inflammation in the rabbit. J Immunol. 1999 Sep 15;163(6):3449-58. PMID: 10477617.

7. Bioactivity
Biological target:

Quiflapon sodium is a potent and selective 5-Lipoxygenase-activating protein (FLAP) inhibitor. MK-0591 is also a potent inhibitor of leukotriene (LT) biosynthesis in intact human and elicited rat polymorphonuclear leukocytes (PMNLs) (IC50 values 3.1 and 6.1 nM,

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respectively) and in human, squirrel monkey, and rat whole blood (IC50 values 510, 69, and 9 nM, respectively). MK-0591 has a high affinity for 5-lipoxygenase activating protein (FLAP) as evidenced by an IC50 value of 1.6 nM in a FLAP binding assay.

In vitro activity

Experiments with the 5-LO-activating protein inhibitor MK-0591 and the intracellular Ca2+ chelator BAPTA-AM demonstrated that the arachidonic acid (AA)-regulated 5-LO translocation is FLAP- and Ca2+-dependent. The redox and competitive 5-lipoxygenase inhibitors L-685,015, L-739,010, and L-702,539 efficiently substituted for AA to reverse the pyrrophenone inhibition of 5-LO translocation, indicating that the site of regulation of 5-LO translocation by AA is at or in the vicinity of the catalytic site. This report demonstrates that AA regulates the translocation of 5-LO in human PMN and unravels a novel mechanism of the cAMP-mediated inhibition of LT biosynthesis.

Reference: J Biol Chem. 2006 Jan 6;281(1):129-36. https://pubmed.ncbi.nlm.nih.gov/16275640/

In vivo activity

Mice with either genetic (5-LO(-/-)) or pharmacological (MK-0591) inhibition of the 5-LO pathway on an apolipoprotein E-deficient (apoE(-/-)). Ang II-induced marked aortic wall remodeling with an incidence of 32, 29 and 40% AAA formation in 5-LO(-/-) apoE(-/-), 5-LO(+/+)apoE(-/-) and 5-LO(+/+)apoE(-/-) mice treated with FLAP inhibitor MK-0591, respectively, with no statistically significant differences in incidence or severity between groups. Abrogation of the 5-LO pathway in mice indicates a lack of role of leukotrienes in Ang II-induced AAA pathogenesis stressing the need for additional non-rodent AAA pre-clinical models to be tested.

Reference: Prostaglandins Other Lipid Mediat. 2007 Aug;84(1-2):34-42. https://pubmed.ncbi.nlm.nih.gov/17643886/