

Product Data Sheet

Product Name: MK 886
Cat. No.: GC12277

Chemical Properties

Cas. No. 118414-82-7

Chemical Name 3-(3-(tert-butylthio)-1-(4-chlorobenzyl)-5-isopropyl-1H-indol-2-yl)-2,2-dimethylpropanoic acid

SMILES C1C=CC=C(C=C1)CN(C2=CC=C(C(C)C)C=C32)C(CC(C)(C(O)=O)C)=C3SC(C)(C)C

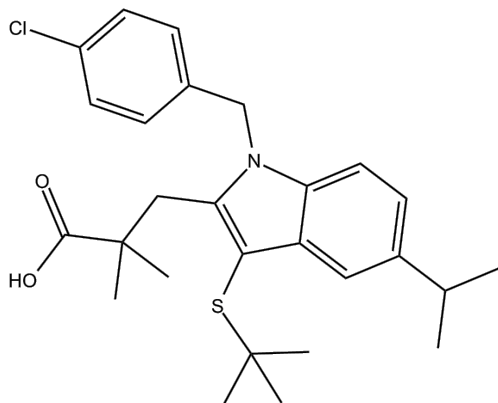
Formula $C_{27}H_{34}ClNO_2S$ M.Wt 472.08

Solubility DMF: 15 mg/ml, DMF:PBS (pH 7.2) (1:3): 0.25 mg/ml, DMSO: 5 mg/ml, Ethanol: 0.14 mg/ml Storage Store at 2-8°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines MM55.K mouse kidney tubular cell line

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

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MM55.K cells were maintained in DMEM supplemented with 10% fetal bovine Preparation Method serum (FBS) at 37°C, 5% CO₂ atmosphere. Cells were treated with MK 886 (25µM) for 24 hours.

Reaction
Conditions 25µM; 24h

Applications MK 886 abrogated the metformin-induced upregulation of OCT-2 and PPARα and downregulated the expression of MATE-1.

Animal experiment [2]:

Animal models Wild-type mice and 5-LOX-deficient mice (B6.129S2-Alox5tmlFun/J)

Preparation Method MK 886 was administered intraperitoneally (i.p.) once a day for 6 days. Mice were tested 30 minutes after the last injection in an automated forced swim test apparatus.

Dosage form 3mg/kg; i.p.; once daily for 6 days

Applications MK 886 slightly increased climbing and significantly reduced rest time in wild-type mice but not in 5-LOX-deficient mice.

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References:

- [1] Arruda AC, Perilhão MS, Santos WA, et al. PPAR α -Dependent Modulation by Metformin of the Expression of OCT-2 and MATE-1 in the Kidney of Mice. *Molecules*. 2020 Jan 17;25(2):392.
- [2] Uz T, Dimitrijevic N, Imbesi M, et al. Effects of MK-886, a 5-lipoxygenase activating protein (FLAP) inhibitor, and 5-lipoxygenase deficiency on the forced swimming behavior of mice. *Neurosci Lett*. 2008 May 9;436(2):269-72.

Background

MK 886 is a 5-lipoxygenase-activating protein (FLAP) inhibitor and PPAR α antagonist (IC₅₀=30nM). MK 886 blocks leukotriene biosynthesis in intact leukocytes and human whole blood (IC₅₀=3nM or 1.1 μ M). MK 886 can be used in research related to inflammatory diseases (such as asthma, arthritis) and cancers (such as gastric cancer, lung cancer)^[1-4].

In vitro, under normoxic or hypoxic conditions, MK 886 (0.5 μ M) was used to treat human pulmonary artery endothelial cells for 24-72 hours. MK 886 significantly reduced endothelial cell proliferation^[5]. MK 886 (25 μ M) was used to treat MM55.K mouse kidney tubular cells for 24 hours. MK 886 abrogated the metformin-induced upregulation of organic cation transporter-2 (OCT-2) and peroxisome proliferator-activated receptor α , and downregulated the expression of multidrug and toxin extrusion protein-1 (MATE-1)^[6].

In vivo, MK 886 (3mg/kg; once daily) was intraperitoneally injected into mice for six consecutive days. MK 886 slightly increased climbing behavior and significantly reduced rest time in mice^[7]. One hour before behavioral testing, MK 886 (10mg/kg) was intraperitoneally injected into Swiss mice. MK 886 significantly reduced the number of open arm entries in the elevated plus maze test in mice^[8].

References:

- [1] Kehrer JP, Biswal SS, La E, et al. Inhibition of peroxisome-proliferator-activated receptor (PPAR) α by MK886. *Biochem J*. 2001 Jun 15;356(Pt 3):899-906.
- [2] Gillard J, Ford-Hutchinson AW, Chan C, et al. L-663,536 (MK-886) (3-[1-(4-chlorobenzyl)-3-t-butylthio-5-isopropylindol-2-yl]-2,2 - dimethylpropanoic acid), a novel, orally active leukotriene biosynthesis inhibitor. *Can J Physiol Pharmacol*. 1989 May;67(5):456-64.
- [3] Shi KN, Li PB, Su HX, et al. MK-886 protects against cardiac ischaemia/reperfusion injury by

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activating proteasome-Keap1-NRF2 signalling. Redox Biol. 2023 Jun;62:102706.

[4] Peres-Buzalaf C, de Paula L, Frantz FG, et al. Control of experimental pulmonary tuberculosis depends more on immunostimulatory leukotrienes than on the absence of immunosuppressive prostaglandins. Prostaglandins Leukot Essent Fatty Acids. 2011 Aug;85(2):75-81.

[5] Porter KM, Kang BY, Adesina SE, et al. Chronic hypoxia promotes pulmonary artery endothelial cell proliferation through H₂O₂-induced 5-lipoxygenase. PLoS One. 2014 Jun 6;9(6):e98532.

[6] Arruda AC, Perilhão MS, Santos WA, et al. PPAR α -Dependent Modulation by Metformin of the Expression of OCT-2 and MATE-1 in the Kidney of Mice. Molecules. 2020 Jan 17;25(2):392.

[7] Uz T, Dimitrijevic N, Imbesi M, et al. Effects of MK-886, a 5-lipoxygenase activating protein (FLAP) inhibitor, and 5-lipoxygenase deficiency on the forced swimming behavior of mice. Neurosci Lett. 2008 May 9;436(2):269-72.

[8] Leo LM, Almeida-Corrêa S, Canetti CA, et al. Age-dependent relevance of endogenous 5-lipoxygenase derivatives in anxiety-like behavior in mice. PLoS One. 2014 Jan 8;9(1):e85009.

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