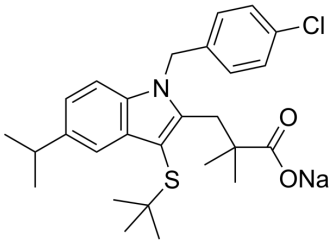
MK-886 sodium salt

Cat. No.:	HY-14166A
CAS No.:	118427-55-7
Molecular Formula:	$C_{27}H_{33}ClNNaO_2S$
Molecular Weight:	494.06
Target:	PPAR; Apoptosis; Leukotriene Receptor; FLAP
Pathway:	Cell Cycle/DNA Damage; Vitamin D Related/Nuclear Receptor; Apoptosis; GPCR/G Protein; Immunology/Inflammation
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	MK-886 (L 663536) sodium salt is a potent, cell-permeable and orally active FLAP (IC_{50} of 30 nM) and leukotriene biosynthesis (IC_{50}s of 3 nM and 1.1 μM in intact leukocytes and human whole blood, respectively) inhibitor. MK-886 sodium salt is also a non-competitive PPARα antagonist and can induce apoptosis[1][2][3].
IC_{50} & Target	IC50: 30 nM (FLAP)[3] IC50: 3 nM (Leukotriene biosynthesis in intact leukocytes) and 1.1 μM (Leukotriene biosynthesis in human whole blood)[2] PPARα[1]
In Vitro	MK-886 sodium salt (0.5-2 μM; 15?hours; primary keratinocytes) treatment reduces keratin-1 expression in a culture of mouse primary keratinocytes[1]. ?Using a transient transfection system in monkey kidney fibroblast CV-1 cells, mouse keratinocyte 308 cells and human lung adenocarcinoma A549 cells, 10 μM MK-886 sodium salt is able to inhibit Wy-14643 activation of PPARα by ~80%. MK-886 sodium salt also decreases PPARα activation by fatty acids in the stable transfection system[1]. ?Although Jurkat cells express all PPAR isoforms, various PPARα and PPARγ agonists are unable to prevent MK-886 sodium salt-induced apoptosis[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	MK-886 sodium salt (L 663536; 5 mg/kg; oral administration; male Sprague-Dawley rats) treatment potently inhibits the antigen-induced dyspnea in inbred rats pretreated with methysergide[2]. ?MK-886 sodium salt (L 663536) inhibits leukotriene biosynthesis in vivo in a rat pleurisy model (ED_{50}, 0.2 mg/kg p.o.), an inflamed rat paw model (ED_{50}, 0.8 mg/kg), a model of leukotriene excretion in rat bile following antigen provocation[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Redox Biol. 2023 Apr 20;62:102706.
- Genes Dev. 2023 Mar 15.
- Chem Biol Interact. 2019 Feb 25;300:123-130.
- J Immunol Res. 2022 May 20;2022:4086710.

- Hum Exp Toxicol. 2021 Feb 4;960327121991901.

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REFERENCES

[1]. Kehrer JP et al. Inhibition of peroxisome-proliferator-activated receptor (PPAR)alpha by MK886. Biochem J. 2001 Jun 15.

[2]. Gillard J et al. L-663,536 (MK-886) (3-[1-(4-chlorobenzyl)-3-t-butyl-thio-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]. Mancini JA, et al. 5-Lipoxygenase-activating protein is the target of a novel hybrid of two classes of leukotriene biosynthesis inhibitors. Mol Pharmacol. 1992 Feb;41(2):267-72.

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