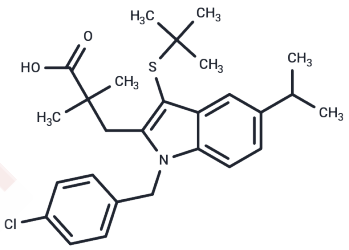
Data Sheet (Cat.No.T6893)

TargetMol

MK-886

Chemical Properties



CAS No. : 118414-82-7

Formula: C27H34ClNO2S

Molecular Weight: 472.08

Appearance: no data available

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description

Description	MK-886 (L 663536) is an inhibitor of leukotriene biosynthesis, acting by inhibiting the 5-lipoxygenase-activating protein (FLAP), and serves as a moderately potent PPARα antagonist.
Targets(IC50)	Apoptosis,FLAP,COX,Leukotriene Receptor,PPAR
In vitro	MK-886, an inhibitor of the 5-lipoxygenase-activating protein (FLAP), potently suppresses leukotriene biosynthesis in intact cells and is frequently used to define a role of the 5-lipoxygenase (EC 1.13.11.34) pathway in cellular or animal models of inflammation, allergy, cancer, and cardiovascular disease. MK-886 inhibits isolated COX-1 (IC50=8 μM) and blocks the formation of the COX-1-derived products 12(S)-hydroxy-5-cis-8,10-trans-heptadecatrienoic acid (12-HHT) and thromboxane B2 in washed human platelets in response to collagen as well as from exogenous arachidonic acid (IC50=13–15 μM).Isolated COX-2 was less affected (IC50=58 μM), and in A549 cells, MK-886 (33 μM) failed to suppress COX-2-dependent 6-ketoprostaglandin (PG)F1α formation. MK-886 (10 μM) inhibits COX-1-mediated platelet aggregation induced by collagen or arachidonic acid whereas thrombin- or U-46619-induced (COX-independent) aggregation is not affected[1].
In vivo	Repeated daily i.p. injections of MK-886 results in increased GluR1 phosphorylation in brain samples obtained from the prefrontal cortex. In contrast, a single injection of MK-886 does not alter cortical GluR1 phosphorylation[2].
Kinase Assay	Enzyme assay is conducted in buffer containing 25 mM Tris, pH 8.0, 1 mM DTT, 1 mM spermine, 50 mM KCl, 0.01% Nonidet P-40, and 1 mM MgCl2. PARP reaction contains 0.1 μCi [3H]NAD+ (200 000 DPM), 1.5 μM NAD+, 150 nM biotinylated NAD+, 1 μg/mL activated calf thymus, and 1?5 nM PARP-1. Autoreactions utilizing SPA bead-based detection are carried out in 50 μL volumes in white 96-well plates. Compounds (e.g., MK-4827) are prepared in 11-point serial dilution in 96-well plate, 5 μL/well in 5% DMSO/Water (10× concentrated). Reactions are initiated by adding first 35 μL of PARP-1 enzyme in buffer and incubating for 5 min at room temperature and then 10 μL of NAD+ and DNA substrate mixture. After 3 h at room temperature, these reactions are terminated by the addition of 50 μL of streptavidin-SPA beads (2.5 mg/mL in 200 mM EDTA, pH 8). After 5 min, they are counted using a TopCount microplate scintillation counter. IC50 data is determined from inhibition curves at various substrate concentrations[1].
Cell Research	IL-1β-stimulated A549 cells (5×106/ml) are pre-incubated with MK-886 (MK, 33 μM), indomethacin (Indo, 10 μM), celecoxib (Cele, 5 μM) or vehicle (DMSO) for 15 min prior to

the addition of 30 μM arachidonic acid. After 15 min at 37 °C, the amount of released 6-keto PGF1α was assessed by ELISA as described in the Materials and methods section. (Only for Reference)

Solubility Information

Solubility	Ethanol: 2.4 mg/mL (5.08 mM),Sonication is recommended. DMSO: 55 mg/mL (116.51 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.1183 mL	10.5914 mL	21.1829 mL
5 mM	0.4237 mL	2.1183 mL	4.2366 mL
10 mM	0.2118 mL	1.0591 mL	2.1183 mL
50 mM	0.0424 mL	0.2118 mL	0.4237 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Reference

Koeberle A, et al. Eur J Pharmacol. 2009, 608(1-3):84-90.
Wang M, Luo W, Yu T, et al. Corynoline protects ang II-induced hypertensive heart failure by increasing PPARα and Inhibiting NF-κB pathway. Biomedicine & Pharmacotherapy. 2022, 150: 113075
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Imbesi M, et al. Brain Res. 2007, 1147:148-53.
Kehrer JP, et al. Biochem J. 2001, 356(Pt 3):899-906.
Gao S, Zhang X, Xu H, et al. Promoting the hippocampal PPARα expression participates in the antidepressant mechanism of reboxetine, a selective norepinephrine reuptake inhibitor. Behavioural Brain Research. 2021: 113535.
Liang X, Liang J, Zhang S, et al.Di-2-ethylhexyl phthalate disrupts hepatic lipid metabolism in obese mice by activating the LXR/SREBP-1c and PPAR-α signaling pathways.Science of The Total Environment.2024: 169919.
Jin L, Zhu W, Hu X, et al.USP25 directly interacts with and deubiquitinates PPARα to increase PPARα stability in hepatocytes and attenuate high-fat diet-induced MASLD in mice.Cell Death & Differentiation.2025: 1-16.