

Product Data Sheet

Product Name: TLC-2716

Cat. No.: GC79763

Chemical Properties

Cas. No. 2408041-54-1

Formula $C_{38}H_{22}ClF_3N_4O_5S$

M.Wt 739.12

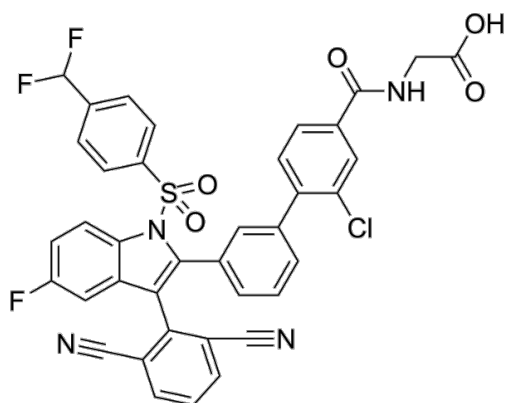
Solubility

Storage Store at -20°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



Background

TLC-2716 is an orally available, gut- and liver-restricted inhibitor against LXR α and LXR β , with EC 50 values of 7 nM and 15 nM, respectively. TLC-2716 represses LXR α / β transcriptional activity, downregulates genes involved in lipogenesis, lipid absorption and lipoprotein metabolism, and preserves peripheral reverse cholesterol transport. TLC-2716 reduces lipid accumulation, suppresses inflammation and fibrotic gene expression, enhances triglyceride-rich lipoprotein clearance, and improves glucose homeostasis and insulin sensitivity. TLC-2716 lowers serum and hepatic triglycerides, plasma cholesterol and other atherogenic lipid profiles in experimental models and humanized liver mice. TLC-2716 can be used for the research of dyslipidemia and related

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

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cardiometabolic disorders [1].

In Vivo, TLC-2716 (0.3-1 mg/kg; oral gavage; once daily; 3 weeks) dose-dependently improves dyslipidemia in high-fat diet-induced obese mice by reducing hepatic de novo lipogenesis, liver TG content, and plasma TG and TC levels without impairing peripheral reverse cholesterol transport^[1]. TLC-2716 (0.1-15 mg/kg; p.o.; daily) reduces dyslipidemia-related endpoints and improves glucose homeostasis in ZDF rats without impairing liver function^[1]. TLC-2716 (0.1-1 mg/kg; p.o.; daily; 21 days) reduces hepatic DNL gene expression, liver TG, and plasma TG and TC in high-fat diet-fed SD rats without impairing liver function^[1]. TLC-2716 (1 mg/kg; p.o.; daily; 8 days) reduces hepatic expression of lipid metabolism genes and trends to reduce liver TG in human liver chimeric mice^[1]. TLC-2716 (15-120 mg/kg; oral gavage; once daily; 26 weeks) reduces plasma TG and TC in lean CD-1 mice without causing observable adverse effects^[1]. TLC-2716 (1-15 mg/kg; oral gavage; once daily; 28 days) reduces plasma TG and TC in cynomolgus monkeys without causing observable adverse effects^[1].

In Vitro, TLC-2716 (1 h at 4 °C) binds to recombinant human LXR α (EC₅₀ = 7 nM) and LXR β (EC₅₀ = 15 nM) in a biochemical binding assay^[1]. TLC-2716 (16 h) induces NCOR recruitment to LXR α and LXR β with EC₅₀ values of 8 nM and 11 nM in HEK293 mammalian two-hybrid assays, and inhibits ABCA1 and SREBP1c reporter activities with IC₅₀ values of 15 nM and 7 nM in HT-29 and HepG2 cells, respectively^[1]. TLC-2716 (5 days) dose-dependently reduces intracellular triglyceride accumulation in primary human Upcyte hepatocytes with an EC₅₀ of 289 nM^[1]. In steatotic human liver organoids, TLC-2716 (500 nM-5 μ M; 3 days) dose-dependently reduces intracellular lipid content and suppresses expression of LXR-associated, lipid metabolism, inflammation, and fibrosis-related genes, with enhanced effects in GCKRTT organoids^[1].

References:

[1]. Li X, et al. An oral, liver-restricted LXR inverse agonist for dyslipidemia: preclinical development and phase 1 trial. Nat Med. 2026;32(3):883-893.

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