Chemical Properties

Product Name: LCQ-908
Cas No.: 956136-95-1
M.Wt: 455.47
Formula: C25H24F3N3O2
Synonyms: Pradigastat; LCQ908; LCQ 908
Chemical Name: 2-\-[4-\-[4-\-[6-(trifluoromethyl)pyridin-3-yl]amino]pyridin-2-yl]phenyl
cyclohexyl]acetic acid
Canonical SMILES: C1CC(CCC1CC(=O)O)C2=CC=C(C=C2)C3=NC=C(C=C3)NC4=CN=C(C=C4)C(F)(F)F
Solubility: Soluble in DMSO
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.
Shopping Condition: Evaluation sample solution: ship with blue ice
All other available size: ship with RT, or blue ice upon request

Biological Activity

Targets: Metabolism
Pathways: DGAT
Description:
IC50: 5 µM for BCRP-mediated efflux activity
LCQ908 is a diacylglycerol acyltransferase-1 (DGAT-1) inhibitor. DGAT-1 has been recognized to catalyze the final committed step of processing dietary fatty acids into triglycerides carried on chylomicrons for transport around the body. Thus, inhibition of DGAT-1 represents a novel
approach to treat metabolic disease.

In vitro: In vitro studies suggest that glucuronidation is the predominant metabolism pathway for elimination of LCQ908 in humans. LCQ908 inhibited BCRP-mediated efflux activity in a dose-dependent fashion with an IC50 value of 5 μM. LCQ908 also inhibited OATP1B1, OATP1B3, and OAT3 activity in a concentration-dependent manner with estimated IC50 values of 1.66 ± 0.95 μM, 3.34 ± 0.64 μM, and 0.973 ± 0.11 μM, respectively [1].

In vivo: LCQ908 was found to suppress the postprandial triglyceride levels in rats, dogs as well as monkeys. In rats whose LPL activity had been abolished, LCQ908 reduced the postprandial accumulation of plasma triglyceride. Additionally, LCQ908 decreased the postprandial rate of CM-TG secretion into the lymphatic duct and reduced the size of CMs [2].

Clinical trial: In a clinical trial, LCQ908 was found to be able to lower fasting triglyceride levels in familial chylomicronemia syndrome patients maintained on a very low-fat diet, and represents a potential drug treatment for this orphan disease [3].

Reference:

