

TUG-770

Chemical Properties

CAS No.:	1402601-82-4
Formula:	C ₁₉ H ₁₄ FNO ₂
Molecular Weight:	307.32
Appearance:	N/A
Storage:	0-4°C for short term (days to weeks), or -20°C for long term (months).

Biological Description

Description	TUG-770 is a highly potent agonist of free fatty acid receptor 1 (FFA1/GPR40, EC ₅₀ : 6 nM for hFFA1).
In vitro	TUG-770 (Compound 22) displayed excellent physicochemical and in vitro ADME properties, with good aqueous solubility, good chemical stability, low lipophilicity, and decreased plasma protein binding (PPB). TUG-770 furthermore showed excellent stability toward human liver microsomes, no inhibition of selected CYP-enzymes implicated in drug-drug interactions, no P-glycoprotein inhibition, and good permeability in the Caco-2 cell assay.
In vivo	Examination of TUG-770 in an acute intraperitoneal glucose tolerance test (IPGTT) in normal mice revealed a good dose-dependent response with maximal reduction in glucose level reached at 50 mg/kg. The effect of TUG-770 was fully sustained after 29 days of daily oral treatment. Additional evaluation of TUG-770 in rats confirmed a significant glucose-lowering effect for the high doses already after 10 min and for all doses after 30 min.

Solubility Information

Solubility	DMSO: 100 mg/mL (325.39 mM) Water: Insoluble (< 1 mg/ml refers to the product slightly soluble or insoluble)
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Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.254 mL	16.27 mL	32.539 mL
5 mM	0.651 mL	3.254 mL	6.508 mL
10 mM	0.325 mL	1.627 mL	3.254 mL
50 mM	0.065 mL	0.325 mL	0.651 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. The storage conditions and period of the stock solution: - 80 °C for 6 months; - 20 °C for 1 month. Please use it as soon as possible.

Reference

1. Christiansen E, Hansen SV, Urban C, Discovery of TUG-770: A Highly Potent Free Fatty Acid Receptor 1 (FFA1/GPR40) Agonist for Treatment of Type 2 Diabetes. ACS Med Chem Lett. 2013 May 9;4(5):441-445.
2. Urano Y, et al. Comparative hepatic transcriptome analyses revealed possible pathogenic mechanisms of fasiglifam (TAK-875)-induced acute liver injury in mice. Chem Biol Interact. 2018 Sep 20;296:185-197.

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