# Saroglitazar magnesium

**Cat. No.:** HY-19937A

**CAS No.:** 1639792-20-3

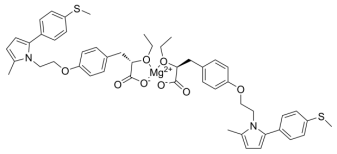
**Molecular Formula:** $C_{50}H_{56}MgN_2O_8S_2$

**Molecular Weight:** 901.42

**Target:** PPAR

**Pathway:** Cell Cycle/DNA Damage; Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor

**Storage:** 4°C, sealed storage, away from moisture
* In solvent : -80°C, 2 years; -20°C, 1 year (sealed storage, away from moisture)



## SOLVENT & SOLUBILITY

**In Vitro**

DMSO : 125 mg/mL (138.67 mM; Need ultrasonic)

| Preparing Stock Solutions | Mass / Solvent / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 1.1094 mL | 5.5468 mL | 11.0936 mL |
| | 5 mM | 0.2219 mL | 1.1094 mL | 2.2187 mL |
| | 10 mM | 0.1109 mL | 0.5547 mL | 1.1094 mL |

Please refer to the solubility information to select the appropriate solvent.

**In Vivo**

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.75 mg/mL (3.05 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.75 mg/mL (3.05 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.75 mg/mL (3.05 mM); Clear solution

## BIOLOGICAL ACTIVITY

**Description**

Saroglitazar magnesium is a novel peroxisome proliferator-activated receptor (PPAR) agonist with predominant PPARα and moderate PPARγ activity with $EC_{50}$ values of 0.65 pM and 3 nM in HepG2 cells, respectively.

**$IC_{50}$ & Target**

| PPARα | PPARγ |
|---|---|
| 0.65 pM (EC50, HepG2 cell) | 3 nM (EC50, HepG2 cell) |

**In Vivo**

In db/db mice, 12-day treatment with Saroglitazar (0.01-3 mg/kg per day, orally) causes dose-dependent reductions in serum triglycerides (TG), free fatty acids (FFA), and glucose. The $ED_{50}$ for these effects is found to be 0.05, 0.19, and 0.19

mg/kg, respectively with AUC-glucose following oral glucose administration (59%) at 1 mg/kg dose. A 90-day repeated dose comparative study in Wistar rats and marmosets confirms efficacy (TG lowering) potential of Saroglitazar and has indicated low risk of PPAR-associated side effects in humans. Based on efficacy and safety profile, Saroglitazar appears to have good potential as novel therapeutic agent for treatment of dyslipidemia and diabetes[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## PROTOCOL

**Animal Administration** [1]

Rats: Rats randomize based on body weights and are divided into three equal groups and receives the daily administration of vehicle (50% w/v honey for marmoset and 0.1% carboxymethylcellulose for Wistar rats) or Saroglitazar (1.5 and 15 mg/kg per day) for 90 days by oral gavage[1].

Mice: Male C57BL/6J-db/db mice are bled on day 0 to determine pretreatment serum glucose and TG. During next 12 days, each animal is dosed (by oral gavage) with vehicle (0.5% sodium carboxymethyl cellulose) or Saroglitazar (0.01, 0.03, 0.1, 0.3,1, and 3 mg/kg per day) or U 72107 (60 mg/kg per day) and on day 12 of the treatment, blood samples are collected (1 h after dosing) from orbital sinus under light ether anesthesia. The serum is isolated and analyzed for glucose, TG, and free fatty acid (FFA)[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## CUSTOMER VALIDATION

- Cell Biol Toxicol. 2020 Jul 1.
- BMC Complement Med Ther. 2021 Apr 10;21(1):118.
- Patent. US20210275504A1.
- Patent. US20190388398A1.

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## REFERENCES

[1]. Jain MR, et al. Saroglitazar, a novel PPARα/γ agonist with predominant PPARα activity, shows lipid-lowering effects in preclinical models. Pharmacol Res Perspect. 2015 Jun;3(3):e00136.



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