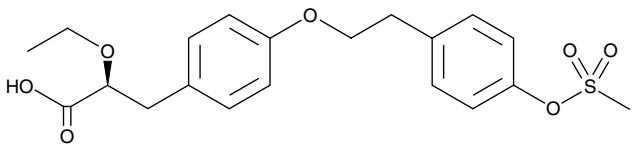
Product Information

Tesaglitazar

Item No. 16791

CAS Registry No.: 251565-85-2
Formal Name: (αS)-ethoxy-4-[2-[4-[(methylsulfonyl)oxy]phenyl]ethoxy]-benzenepropanoic acid
Synonyms: AZ 242, Galida™
MF: $C_{20}H_{24}O_7S$
FW: 408.5
Purity: ≥98%
Stability: ≥2 years at -20°C
Supplied as: A crystalline solid
UV/Vis.: λ_{max}: 225, 275 nm



Laboratory Procedures

For long term storage, we suggest that tesaglitazar be stored as supplied at -20°C. It should be stable for at least two years.

Tesaglitazar is supplied as a crystalline solid. A stock solution may be made by dissolving the tesaglitazar in the solvent of choice. Tesaglitazar is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of tesaglitazar in ethanol is approximately 10 mg/ml and approximately 50 mg/ml in DMSO and DMF.

Tesaglitazar is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, tesaglitazar should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Tesaglitazar has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Peroxisome proliferator-activated receptors (PPARs) are activated by fatty acids and eicosanoids as well as antidyslipidemic agents. Among the receptor isotypes, PPARα demonstrates a particular role in fatty acid oxidation whereas PPARγ is known to be involved in adipocyte differentiation and lipid storage. Tesaglitazar, a dihydro cinnamate derivative, is a dual agonist of PPARα and γ that demonstrates IC_{50} values of 1 and 0.2 µM, respectively in ligand binding assays.[1] At 3 µM/kg/day for three weeks, tesaglitazar has been used to reduce insulin resistance in obese Zucker rats.[2] Furthermore, it has been investigated clinically for its potential to address disorders in glucose and lipid metabolism in patients with type 2 diabetes.[3]

References

1. Cronet, P., Petersen, J.F.W., Folmer, R., *et al.* Structure of the PPARα and -γ ligand binding domain in complex with AZ 242; ligand selectivity and agonist activation in the PPAR family. *Structure* **9(8)**, 699-706 (2001).
2. Wallenius, K., Kjellstedt, A., Thalén, P., *et al.* The PPARα/γ agonist, tesaglitazar, improves insulin mediated switching of tissue glucose and free fatty acid utilization *in vivo* in the obese zucker rat. *PPAR Res.* **2013**, 1-14 (2013).
3. Wilding, J.P.H., Gause-Nilsson, I., Persson, A., *et al.* Tesaglitazar, as add-on therapy to sulphonylurea, dose-dependently improves glucose and lipid abnormalities in patients with type 2 diabetes. *Diab. Vasc. Dis. Res.* **4(3)**, 194-203 (2007).

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