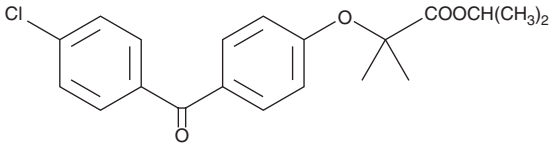
PRODUCT INFORMATION

Fenofibrate

Item No. 10005368

CAS Registry No.: 49562-28-9
Formal Name: 2-[4-(4-chlorobenzoyl)phenoxy]-2-methyl-propanoic acid, 1-methylethyl ester
MF: $C_{20}H_{21}ClO_4$
FW: 360.8
Purity: ≥98%
UV/Vis.: λ_{max}: 287 nm
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Fenofibrate is supplied as a crystalline solid. A stock solution may be made by dissolving the fenofibrate in the solvent of choice, which should be purged with an inert gas. Fenofibrate is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of fenofibrate in these solvents is 1, 15, and 30 mg/ml, respectively.

Fenofibrate is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, fenofibrate should first be dissolved in DMF and then diluted with the aqueous buffer of choice. Fenofibrate has a solubility of 0.25 mg/ml in a 1:3 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Fenofibrate is an agonist of peroxisome proliferator-activated receptor α (PPARα) with EC_{50} values of 18 and 30 µM for mouse and human receptors, respectively, in a transactivation assay.[1] It is selective for PPARα over PPARγ (EC_{50}s = 300 and 200 µM for mouse and human receptors, respectively) and lacks activity at mouse and human PPARδ at a concentration of 100 µM. *In vivo*, fenofibrate (50-100 mg/kg) reduces plasma levels of triglycerides, C-reactive protein, and malondialdehyde (MDA) in mice with fructose-induced hypertriglycemia in a dose-dependent manner.[2] It decreases glomerular and tubular atrophy and necrosis induced by cisplatin (Item No. 13119) in rat kidney when administered at a dose of 100 mg/kg.[3] Fenofibrate also reduces the number of pulmonary lesions induced by 4-nitroquinoline 1-oxide (4-NQO) in lung in Tsumura Suzuki obese diabetic (TSOD) mice.[4].

References

1. Willson, T.M., Brown, P.J., Sternbach, D.D., *et al.* The PPARs: From orphan receptors to drug discovery. *J. Med. Chem.* **43(4)**, 528-550 (2000).
2. Sun, B., Xie, Y., Jiang, J., *et al.* Pleiotropic effects of fenofibrate therapy on rats with hypertriglycemia. *Lipids Health Dis.* **14:27**, (2015).
3. Helmy, M.M., Helmy, M.W., and El-Mas, M.M. Additive renoprotection by pioglitazone and fenofibrate against inflammatory, oxidative and apoptotic manifestations of cisplatin nephrotoxicity: Modulation by PPARs. *PLoS One* **10(11)**, e0142303 (2015).
4. Kuno, T., Hata, K., Takamatsu, M., *et al.* The peroxisome proliferator-activated receptor (PPAR) α agonist fenofibrate suppresses chemically induced lung alveolar proliferative lesions in male obese hyperlipidemic mice. *Int. J. Mol. Sci.* **15(5)**, 9160-9172 (2014).

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.**CAYMANCHEM**.COM