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- Trockeneiszuschlag
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- Expressversand

SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

mail@szabo-scandic.com

www.szabo-scandic.com

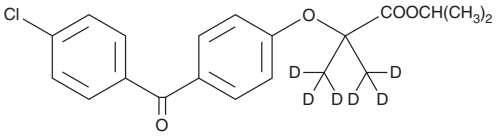
linkedin.com/company/szaboscandic

PRODUCT INFORMATION

Fenofibrate-d_6

Item No. 25710

CAS Registry No.: 1092484-56-4
Formal Name: 2-[4-(4-chlorobenzoyl)phenoxy]-2-(methyl-d_3)-propanoic-3,3,3-d_3 acid, 1-methylethyl ester
MF: $C_{20}H_{15}ClD_6O_4$
FW: 366.9
Chemical Purity: ≥98% (Fenofibrate)
Deuterium Incorporation: ≥99% deuterated forms (d_1-d_6); ≤1% d_0
Supplied as: A 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-d_6 is intended for use as an internal standard for the quantification of fenofibrate (Item No. 10005368) by GC- or LC-MS. The accuracy of the sample weight in this vial is between 5% over and 2% under the amount shown on the vial. If better precision is required, the deuterated standard should be quantitated against a more precisely weighed unlabeled standard by constructing a standard curve of peak intensity ratios (deuterated *versus* unlabeled).

Fenofibrate-d_6 is supplied as a solid. A stock solution may be made by dissolving the fenofibrate-d_6 in the solvent of choice, which should be purged with an inert gas. Fenofibrate-d_6 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of fenofibrate-d_6 in these solvents is approximately 1, 15, and 30 mg/ml, respectively.

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 of 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