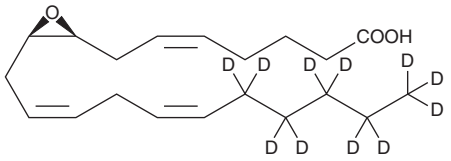
PRODUCT INFORMATION

(±)8(9)-EET-d_{11}

Item No. 10009532

Formal Name: (±)8(9)-epoxy-5Z,8Z,14Z-eicosatrienoic-16,16,17,17,18,18,19,19,20,20,20-d_{11} acid
Synonyms: (±)8,9-EET-d_{11}, (±)8,9-EpETrE-d_{11}
MF: $C_{20}H_{21}D_{11}O_3$
FW: 331.5
Chemical Purity: ≥98% ((±)8(9)-EET)
Deuterium Incorporation: ≥99% deuterated forms (d_1-d_{11}); ≤1% d_0
Supplied as: A solution in ethanol
Storage: -20°C
Stability: ≥1 year

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



NOTE: Relative stereochemistry shown in chemical structure

Laboratory Procedures

(±)8(9)-EET-d_{11} is intended for use as an internal standard for the quantification of 8(9)-EET 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).

(±)8(9)-EET-d_{11} is supplied as a solution in ethanol. To change the solvent, simply evaporate the ethanol under a gentle stream of nitrogen and immediately add the solvent of choice. Solvents such as DMSO and dimethyl formamide purged with an inert gas can be used. The solubility of (±)8(9)-EET-d_{11} in these solvents is approximately 50 mg/ml.

Description

(±)8(9)-EET is a racemic mixture of the R/S enantiomeric forms biosynthesized from arachidonic acid (Item Nos. 90010 | 90010.1 | 10006607).[1,2] (±)8(9)-EET is a major cytochrome P450 (CYP) metabolite in the renal cortex.[3] It activates PPARα in HEK293 cells when used at a concentration of 1 µM but inhibits NF-κB activity induced by IL-1β in a PPARα-dependent and -independent fashion.[4] It is also a substrate of COX-1 and COX-2, as measured by oxygen consumption and product formation assays when used at a concentration of 50 µM.[5] (8S,9R)-EET (1 µg/kg), but not (8R,9S)-EET, reduces the glomerular filtration rate (GFR) through COX-dependent pre-glomerular vasoconstriction in rats.[6]

References

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2. Oliw, E.H., Guengerich, F.P., and Oates, J.A. Oxygenation of arachidonic acid by hepatic monooxygenases. Isolation and metabolism of four epoxide intermediates. *J. Biol. Chem*. **257(7)**, 3771-3781 (1982).
3. Zhang, J.Y., Prakash, C., Yamashita, K., *et al*. Regiospecific and enantioselective metabolism of 8,9-epoxyeicosatrienoic acid by cyclooxygenase. *Biochem. Biophys. Res. Commun*. **183(1)**, 138-143 (1992).
4. Bystrom, J., Wray, J.A., Sugden, M.C., *et al*. Endogenous epoxygenases are modulators of monocyte/macrophage activity. *PloS One* **6(10)**, e26591 (2011).
5. Rand, A.A., Barnych, B., Morisseau, C., *et al*. Cyclooxygenase-derived proangiogenic metabolites of epoxyeicosatrienoic acids. *Proc. Natl. Acad. Sci. USA* **114(17)**, 4370-4375 (2017).
6. Katoh, T., Takahashi, K., Capdevila, J., *et al*. Glomerular stereospecific synthesis and hemodynamic actions of 8,9-epoxyeicosatrienoic acid in rat kidney. *Am. J. Physiol*. **261(4 Pt 2)**, F578-F586 (1991).

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