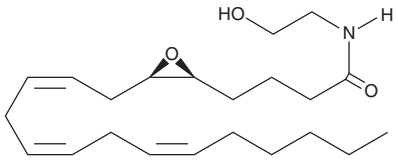
PRODUCT INFORMATION

(±)5(6)-EET Ethanolamide

Item No. 10008596

Formal Name:	N-(2-hydroxyethyl)-(±)5,6-epoxy-8Z,11Z,14Z-eicosatrienamide
Synonym:	(±)5,6-EpETrE Ethanolamide
MF:	$C_{22}H_{37}NO_3$
FW:	363.5
Purity:	≥95%
Supplied as:	A solution in ethanol
Storage:	-80°C
Stability:	≥2 years



NOTE: Relative stereochemistry shown in chemical structure

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

Laboratory Procedures

(±)5(6)-EET ethanolamide 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 (±)5(6)-EET ethanolamide in these solvents is approximately 20 mg/ml.

(±)5(6)-EET ethanolamide is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, the ethanolic solution of (±)5(6)-EET ethanolamide should be diluted with the aqueous buffer of choice. (±)5(6)-EET ethanolamide has a solubility of approximately 0.5 mg/ml in a 1:6 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Arachidonyl ethanolamide (AEA; Item No. 90050) is an endogenous lipid neurotransmitter with cannabinergic activity, binding to both the central cannabinoid (CB_1) and peripheral cannabinoid (CB_2) receptors.[1,2] Fatty acid amide hydrolase (FAAH) is the enzyme responsible for the hydrolysis and inactivation of AEA.[3] Metabolism of AEA by cyclooxygenase-2, leading to formation of prostaglandin ethanolamides, and by lipoxygenases has also been documented.[4] (±)5(6)-EET ethanolamide is a cytochrome P450 (CYP450) metabolite of AEA, although specific stereochemistry rather than a racemic mixture ensues from enzymatic metabolism. (±)5(6)-EET ethanolamidee is a potent and selective agonist of CB_2 (K_i = 8.9 nM for CB_2, 1.4 µM for CB_1), effectively suppressing forskolin-stimulated accumulation of cAMP through this G_i-coupled receptor (IC_{50} = 9.8 nM).[5]

References

1. Felder, C.C., Briley, E.M., Axelrod, J., *et al. Proc. Natl. Acad. Sci. USA* **90**, 7656-7660 (1993).
2. Lambert, D.M. and Fowler, C.J. *J. Med. Chem.* **48(16)**, 5059-5087 (2005).
3. Deutsch, D.G., Ueda, N., and Yamamoto, S. *Prostaglandins Leukot. Essent. Fatty Acids* **66(2&3)**, 201-210 (2002).
4. Kozak, K.R. and Marnett, L.J. *Prostaglandins Leukot. Essent. Fatty Acids* **66(2&3)**, 211-220 (2002).
5. Snider, N.T., Nast, J.A., Tesmer, L.A., *et al. Mol. Pharmacol.* **75(4)**, 965-972 (2009).

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