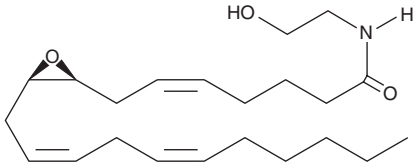
PRODUCT INFORMATION

(±)8(9)-EET Ethanolamide

Item No. 10008597

Formal Name:	N-(2-hydroxyethyl)-(±)8(9)-epoxy-5Z,11Z,14Z-eicosatrienoic amide
Synonym:	(±)8,9-EET Ethanolamide
MF:	$C_{22}H_{37}NO_3$
FW:	363.5
Purity:	≥98%
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

(±)8(9)-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 (±)8(9)-EET ethanolamide in these solvents is approximately 50 mg/ml.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. If an organic solvent-free solution of (±)8(9)-EET ethanolamide is needed, it can be prepared by evaporating the ethanol and directly dissolving the neat oil in aqueous buffers. The solubility of (±)8(9)-EET ethanolamide in PBS, pH 7.2, is approximately 1 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Arachidonoyl ethanolamide (AEA; anandamide) 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 COX-2, lipoxygenases, and cytochrome P450 (CYP450) enzymes has also been documented.[4,5] (±)8(9)-EET ethanolamide is a CYP450 metabolite of AEA, although specific stereochemistry rather than a racemic mixture would likely ensue from enzymatic metabolism.[5] Human liver microsomes metabolize AEA to 5,6-, 8,9-, 11,12-, and 14,15-EET ethanolamides in a time and protein concentration dependent manner.[5] (±)8(9)-EET reduces glomerular filtration rate through cyclooxygenase dependent preglomerular vasoconstriction.[6] The physiological actions of (±)8(9)-EET ethanolamide have not been examined.

References

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