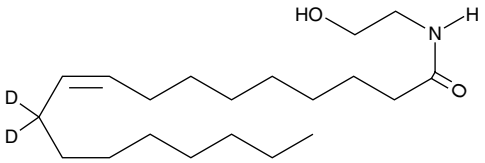
Product Information

Oleoyl Ethanolamide-d_2

Item No. 10007823

CAS Registry No.:	1245477-09-1
Formal Name:	N-(2-hydroxyethyl)-9Z-octadecenamide-11,11-d_2
Synonyms:	OEA-d_2, Oleic Acid Ethanolamide-d_2
MF:	$C_{20}H_{37}D_2NO_2$
FW:	327.5
Chemical Purity:	≥98%
Deuterium Incorporation:	≥99% deuterated forms (d_1-d_2); ≤1% d_0
Stability:	≥1 year at -20°C
Supplied as:	A solution in ethanol



Laboratory Procedures

Oleoyl ethanolamide-d_2 (OEA-d_2) contains two deuterium atoms at the 11 position. It is intended for use as an internal standard for the quantification of OEA by GC- or LC-mass spectrometry (MS). For long term storage, we suggest that OEA-d_2 be stored as supplied at -20°C. It will be stable for at least one year.

OEA-d_2 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 OEA-d_2 in these solvents is approximately 50 mg/ml.

OEA-d_2 is used as an internal standard for the quantification of OEA by stable isotope dilution 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).

OEA is an analogue of the endocannabinoid arachidonoyl ethanolamide (AEA) found in brain tissue and in chocolate.[1] It is one of the long chain fatty acid ethanolamides that accumulates rapidly in infarcted tissue,[2] but its biosynthesis is reduced in the intestine of rats following food deprivation.[3] OEA is an endogenous, potent agonist for peroxisome proliferator-activated receptor α (PPARα), exhibiting an EC_{50} value of 120 nM in a transactivation assay.[4] Systemic administration of OEA suppresses food intake and reduces weight gain in rats (10 mg/kg intraperitoneally) and PPARα wild-type mice, but not in PPARα knockout mice.[3,4] These data indicate that OEA regulates food intake by a PPARα-mediated mechanism.

References

1. di Tomaso, E., Beltramo, M., and Piomelli, D. Brain cannabinoids in chocolate. *Nature* **382**, 677-678 (1996).
2. Epps, D.E., Palmer, J.W., Schmid, H.H.O., *et al.* Inhibition of permeability-dependent Ca^{2+} release from mitochondria by N-acelethanolamines, a class of lipids synthesized in ischemic heart tissue. *J. Biol. Chem.* **257**, 1383-1392 (1982).
3. de Fonseca, F.R., Navarro, M., Gómez, R., *et al.* An anorexic lipid mediator regulated by feeding. *Nature* **414**, 209-212 (2001).
4. Fu, J., Gaetani, S., Oveisi, F., *et al.* Oleylethanolamide regulates feeding and body weight through activation of the nuclear receptor PPAR-α. *Nature* **425**, 90-93 (2003).

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