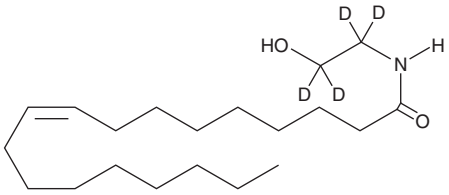
# PRODUCT INFORMATION

## Oleoyl Ethanolamide-$d_4$

*Item No. 9000552*

**CAS Registry No.:** 946524-36-3
**Formal Name:** N-(2-hydroxyethyl-1,1,2,2-$d_4$)-9Z-octadecenamide
**Synonyms:** OEA-$d_4$, Oleic Acid Ethanolamide-$d_4$
**MF:** $C_{20}H_{35}D_4NO_2$
**FW:** 329.6
**Chemical Purity:** ≥98% (Oleoyl Ethanolamide)
**Deuterium Incorporation:** ≥99% deuterated forms ($d_1$-$d_4$); ≤1% $d_0$
**Supplied as:** A 1 mg/ml solution in ethanol
**Storage:** -20°C
**Stability:** ≥4 years




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

### Laboratory Procedures

OEA-$d_4$ is intended for use as an internal standard for the quantification of OEA 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).

OEA-$d_4$ 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-$d_4$ in these solvents is approximately 100 mg/ml.

### Description

OEA is an analog 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 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).



**WARNING**
THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

**SAFETY DATA**
This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the complete Safety Data Sheet, which has been sent *via* email to your institution.

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