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Produktinformation

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- Diagnostik & molekulare Diagnostik
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siehe unsere Liefer- und Versandbedingungen

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- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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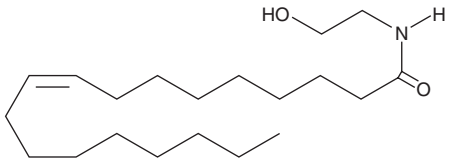
PRODUCT INFORMATION

Oleoyl Ethanolamide

Item No. 90265

CAS Registry No.:	111-58-0
Formal Name:	N-(2-hydroxyethyl)-9Z-octadecenamide
Synonyms:	Oleic Acid Ethanolamide, OEA
MF:	$C_{20}H_{39}NO_2$
FW:	325.5
Purity:	≥98%
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥1 year



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

Laboratory Procedures

OEA is supplied as a crystalline solid. A stock solution may be made by dissolving the OEA in an organic solvent purged with an inert gas. OEA is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of OEA in these solvents is at least 100 mg/ml.

OEA is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, OEA should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. Therefore, further dilutions of the organic solvent 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. We do not recommend storing the aqueous solution for more than one day.

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

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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(3)**, 1383-1392 (1982).
3. de Fonseca, F.R., Navarro, M., Gómez, R., *et al.* An anorexic lipid mediator regulated by feeding. *Nature* **414(6860)**, 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(6953)**, 90-93 (2003).

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