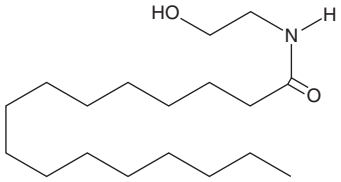
PRODUCT INFORMATION

Palmitoyl Ethanolamide

Item No. 90350

CAS Registry No.: 544-31-0
Formal Name: N-(2-hydroxyethyl)-hexadecanamide
Synonyms: Palmidrol, Palmityl Ethanolamide, PEA
MF: $C_{18}H_{37}NO_2$
FW: 299.5
Purity: ≥98%
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥4 years



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

Laboratory Procedures

Palmitoyl ethanolamide (PEA) is supplied as a crystalline solid. A stock solution may be made by dissolving the PEA in the solvent of choice. PEA is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of PEA in these solvents is approximately 2, 5, and 10 mg/ml, respectively.

PEA is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, PEA should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. PEA has a solubility of approximately 10 µg/ml in a 1:10 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

PEA is an endogenous fatty N-acyl ethanolamine and a derivative of the endocannabinoid arachidonoyl ethanolamide (AEA; Item No. 90050).[1,2] It selectively activates peroxisome proliferator-activated receptor α (PPARα; EC_{50} = 3.1 µM) over PPARβ/δ and PPARγ in HeLa cells expressing the human receptors.[3] PEA binds to RBL-2H3 basophil membranes (IC_{50} = 1 nM), which endogenously express cannabinoid 2 (CB_2), but not CB_1, receptors, and inhibits antigen-induced serotonin release from RBL-2H3 cells (EC_{50} = 0.27 µM).[2] It prevents decreases in paw withdrawal latency in a radiant heat hypersensitivity test, an effect that can be reversed by the CB_1 receptor antagonist SR141716 (rimonabant; Item No. 9000484), PPARγ antagonist GW 9662 (Item No. 70785), and transient receptor potential vanilloid 1 (TRPV1) antagonist capsazepine (Item No. 10007518), in a mouse model of neuropathic pain induced by chronic constriction injury of the sciatic nerve.[4] PEA (10 mg/kg) decreases carrageenan-induced paw edema in wild-type, but not *Ppara*$^{-/-}$, mice.[3] It inhibits tonic convulsions induced by pentylenetetrazole (PTZ; Item No. 18682) in rats when administered at a dose of 40 mg/kg.[5] Formulations containing palmitoyl ethanolamide have been used as dietary supplements.

References

1. Rheault, T.R., Caferro, T.R., Dickerson, S.H., *et al. Bioorg. Med. Chem. Lett.* **19(3)**, 817-820 (2009).
2. Zhong, L., Liao, D., Zhang, M., *et al. Cancer Lett.* **442**, 252-261 (2019).
3. Lo Verme, J., Fu, J., Astarita, G., *et al. Mol. Pharmacol.* **67(1)**, 15-19 (2005).
4. Costa, B., Comelli, F., Bettoni, I., *et al. Pain* **139(3)**, 541-550 (2008).
5. Sheerin, A.H., Zhang, X., Saucier, D.M., *et al. Epilepsia* **45(10)**, 1184-1188 (2004).

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