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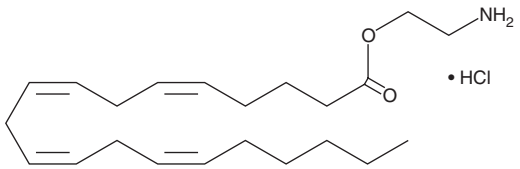
PRODUCT INFORMATION

O-Arachidonoyl Ethanolamine (hydrochloride)

Item No. 91050

CAS Registry No.: 443129-35-9
Formal Name: 5Z,8Z,11Z,14Z-eicosatetraenoic acid, 2-aminoethyl ester, monohydrochloride
Synonyms: Arachidonic Acid-(2-aminoethyl)-ester, O-AEA, Virodhamine
MF: $C_{22}H_{37}NO_2 \bullet HCl$
FW: 384.0
Purity: ≥98%
Supplied as: A neat oil
Storage: -80°C
Stability: As supplied, 6 months from the QC date provided on the Certificate of Analysis, when stored properly



Laboratory Procedures

O-Arachidonoyl ethanolamine (O-AEA) (hydrochloride) is supplied as a neat oil. A stock solution may be made by dissolving the O-AEA (hydrochloride) in the solvent of choice. O-AEA (hydrochloride) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of O-AEA (hydrochloride) in these solvents is approximately 20 mg/ml. O-AEA (hydrochloride) is not stable in solution, dilute samples and use immediately.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Organic solvent-free aqueous solutions of O-AEA (hydrochloride) can be prepared by directly dissolving the neat oil in aqueous buffers. The solubility of O-AEA (hydrochloride) in PBS (pH 7.2) is approximately 100 µg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Arachidonoyl ethanolamide (AEA) was the first endogenous cannabinoid to be isolated and characterized as an agonist acting on the same receptors (CB_1 and CB_2) as tetrahydrocannabinols (THC).[1,2] Since that time, a number of related endocannabinoids have been isolated, most notably 2-arachidonoyl glycerol (2-AG).[3] O-AEA is a recently isolated constituent of human and rat brain wherein the ethanolamine moiety is attached "backwards", as an ester instead of an amide, as in AEA.[1,2,4] O-AEA has mixed agonist/antagonist activity at the CB_1 receptor and does not appear to be the native endogenous cannabinoid agonist at this receptor. This is in keeping with other observations that 2-AG is the primary endogenous CB_1 receptor ligand.[5]

References

1. Devane, W.A., Hanus, L., Breuer, A., *et al. Science* **258**, 1946-1949 (1992).
2. Felder, C.C., Briley, E.M., Axelrod, J., *et al. Proc. Natl. Acad. Sci. USA* **90**, 7656-7660 (1993).
3. Sugiura, T., Kodaka, T., Kondo, S., *et al. J. Biochem.* **122**, 890-895 (1997).
4. Porter, A.C., Sauer, J.-M., Knierman, M.D., *et al. J. Phar. Exp. Ther.* **301**(**3**), 1020-1024 (2002).
5. Sugiura, T., Kodaka, T., Nakane, S., *et al. J. Biol. Chem.* **274**, 2794-2801 (1999).

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