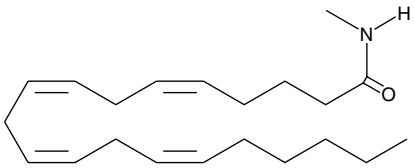
Product Information

Arachidonoyl-N-methyl amide

Item No. 10007294

CAS Registry No.:	156910-29-1
Formal Name:	N-methyl-5Z,8Z,11Z,14Z-eicosatetraenamide
MF:	$C_{21}H_{35}NO$
FW:	317.5
Purity:	≥98%
Stability:	≥1 year at -20°C
Supplied as:	A solution in methyl acetate



Laboratory Procedures

For long term storage, we suggest that arachidonoyl-N-methyl amide be stored as supplied at -20°C. It will be stable for at least one year.

Arachidonoyl-N-methyl amide is supplied as a solution in methyl acetate. To change the solvent, simply evaporate the methyl acetate under a gentle stream of nitrogen and immediately add the solvent of choice. Solvents such as ethanol, DMSO, and dimethyl formamide purged with an inert gas can be used. The solubility of arachidonoyl-N-methyl amide in these solvents is approximately 10 mg/ml.

Further dilutions of the stock 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. For greater aqueous solubility, arachidonic acid N-methyl amide can be directly dissolved in 0.1 M Na_2CO_3 (1 mg/ml) and then diluted with PBS (pH 7.2) to achieve the desired concentration or pH. We do not recommend storing the aqueous solution for more than one day.

Anandamide (AEA) is an endogenous cannabinoid that binds to both central cannabinoid (CB_1) and peripheral cannabinoid (CB_2) receptors. The biological actions of AEA are terminated by cellular uptake and hydrolysis of the amide bond by the enzyme fatty acid amide hydrolase. Arachidonoyl-N-methyl amide is an analog of AEA that binds to the human CB_1 receptor with a K_i of 60 nM.[1] It inhibits rat glial gap junction cell-cell communication 100% at a concentration of 50 µM.[2]

References

1. Sheskin, T., Hanus, L., Slager, J., *et al.* Structural requirements for binding of anandamide-type compounds to the brain cannabinoid receptor. *J. Med. Chem.* **40**, 659-667 (1997).
2. Boger, D.L., Sato, H., Lerner, A.E., *et al.* Arachidonic acid amide inhibitors of gap junction cell-cell communication. *Bioorg. Medicinal Chem. Letters* **9**, 1151-1154 (1999).

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