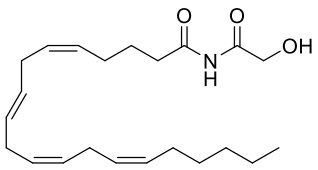
Product data sheet

MedKoo Cat#: 525001 Name: Anandamide CAS#: 94421-68-8 Chemical Formula: $C_{22}H_{35}NO_3$ Exact Mass: 361.2617 Molecular Weight: 361.526	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
Anandamide, also known as N-arachidonoylethanolamine or AEA, is a fatty acid neurotransmitter derived from the non-oxidative metabolism of eicosatetraenoic acid (arachidonic acid) an essential ω-6 polyunsaturated fatty acid. The name is taken from the Sanskrit word ananda, which means "joy, bliss, delight", and amide. It is synthesized from N-arachidonoyl phosphatidylethanolamine by multiple pathways. It is degraded primarily by the fatty acid amide hydrolase (FAAH) enzyme, which converts anandamid.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under “QC And Documents” section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
Ethanol:PBS (1:2)	8.5	23.51

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	**1 mg**	**5 mg**	**10 mg**
1 mM	2.77 mL	13.83 mL	27.66 mL
5 mM	0.55 mL	2.77 mL	5.53 mL
10 mM	0.28 mL	1.38 mL	2.77 mL
50 mM	0.06 mL	0.28 mL	0.55 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of “Calculator”

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Costa L, Moreia-Pinto B, Felgueira E, Ribeiro A, Rebelo I, Fonseca BM. The major endocannabinoid anandamide (AEA) induces apoptosis of human granulosa cells. Prostaglandins Leukot Essent Fatty Acids. 2021 Jun 8;171:102311. doi: 10.1016/j.plefa.2021.102311. Epub ahead of print. PMID: 34126378.
2. Chiurchiù V, Rapino C, Talamonti E, Leuti A, Lanuti M, Gueniche A, Jourdain R, Breton L, Maccarrone M. Anandamide Suppresses Proinflammatory T Cell Responses In Vitro through Type-1 Cannabinoid Receptor-Mediated mTOR Inhibition in Human Keratinocytes. J Immunol. 2016 Nov 1;197(9):3545-3553. doi: 10.4049/jimmunol.1500546. Epub 2016 Sep 30. PMID: 27694494.

In vivo study

1. Çengelli Ünel Ç, Dönertaş B, Aydin Ş, Ulupinar E, Özatik O, Kaygisiz B, Yildirim E, Erol K. Protective effects of anandamide against cisplatin-induced peripheral neuropathy in rats. Turk J Med Sci. 2021 Jun 13. doi: 10.3906/sag-2101-224. Epub ahead of print. PMID: 34118805.
2. Sultan M, Alghetaa H, Mohammed A, Abdulla OA, Wisniewski PJ, Singh N, Nagarkatti P, Nagarkatti M. The Endocannabinoid Anandamide Attenuates Acute Respiratory Distress Syndrome by Downregulating miRNA that Target Inflammatory Pathways. Front Pharmacol. 2021 Apr 27;12:644281. doi: 10.3389/fphar.2021.644281. PMID: 33995054; PMCID: PMC8113864.

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7. Bioactivity

Biological target:

Endogenous and non-selective CB agonist.

In vitro activity

AEA significantly inhibited TNF-α production from 2.5 μM up to 10 μM, being ineffective at lower concentrations (Supplemental Fig. 1A). Furthermore, 2.5 μM AEA did not affect cell viability, which decreased to ∼70% at 10 μM (data not shown). Upon activation with IFN-γ, HaCaT cells also produced significantly higher levels of several proinflammatory cytokines (IL-6, IL-8, TNF-α, IL-12 p40), as compared with untreated controls. When cells were pretreated with 2.5 μM AEA, the production of TNF-α and IL-12 p40 was significantly suppressed, whereas no significant effect was observed on IL-6 and IL-8 levels (Fig. 1A).

Reference: J Immunol. 2016 Nov 1;197(9):3545-3553. https://pubmed.ncbi.nlm.nih.gov/27694494/

In vivo activity

This study observed that the clinical parameters for lung function including specific airways resistance, specific airway conductance, and minute per volume to be significantly improved in SEB + AEA groups and similar to the naïve group, in comparison to SEB + VEH mice (Figure 1A). These data demonstrated that AEA was able to rescue impairments to the lung function caused by SEB exposure. Further, the total number of mononuclear cells (MNCs) in the lung was significantly decreased in SEB + AEA mice when compared to SEB + VEH mice (Figure 1B). Histological sections of the lungs are shown in Figure 1C that are representative pictures of the data presented in Figure 1B.

Reference: Front Pharmacol. 2021; 12: 644281. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8113864/