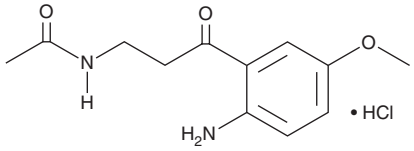
PRODUCT INFORMATION

AMK (hydrochloride)

Item No. 34688

CAS Registry No.: 1215711-91-3
Formal Name: N-[3-(2-amino-5-methoxyphenyl)-3-oxopropyl]-acetamide, monohydrochloride
Synonyms: N-γ-acetyl-5-Methoxykynurenamine, N^1-acetyl-5-Methoxykynuramine
MF: $C_{12}H_{16}N_2O_3$ • HCl
FW: 272.7
Purity: ≥95%
Supplied as: A 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

AMK (hydrochloride) is supplied as a solid. A stock solution may be made by dissolving the AMK (hydrochloride) in the solvent of choice, which should be purged with an inert gas. AMK (hydrochloride) is slightly soluble in methanol.

AMK (hydrochloride) is slightly soluble in aqueous solutions. To enhance aqueous solubility, dilute the organic solvent solution into aqueous buffers or isotonic saline. If performing biological experiments, ensure 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

AMK is an active metabolite of the neurohormone melatonin (Item No. 14427).[1-4] It is formed from melatonin *via* the metabolic intermediate AFMK (Item No. 10005254) that is then deformylated by catalase or formamidase.[5,6] AMK scavenges singlet oxygen *in vitro* when used at a concentration of 200 µM.[1] It inhibits the epinephrine- and arachidonic acid-induced production of prostaglandin E_2 (PGE_2; Item No. 14010) and PGD_2 (Item No. 12010) in ovine seminal vesicle microsomes in a concentration- and time-dependent manner, as well as LPS-induced increases in COX-2 levels in RAW 264.7 macrophages when used at a concentration of 500 µM.[2,3] AMK (20 mg/kg) decreases MPTP-induced increases in lipid peroxidation in the cytosol and mitochondria from substantia nigra and striatum in a mouse model of MPTP-induced Parkinson's disease.[4]

References

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4. Tapias, V., Escames, G., López, L.C., *et al. J. Neurosci. Res.* **87(13)**, 3002-3010 (2009).
5. Tan, D.-X., Manchester, L.C., Reiter, R.J., *et al. Free Radic. Biol. Med.* **29(11)**, 1177-1185 (2000).
6. Hirata, F., Hayaishi, O., Tokuyama, T., *et al. J. Biol. Chem.* **249(4)**, 1311-1313 (1974).

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.**CAYMANCHEM**.COM