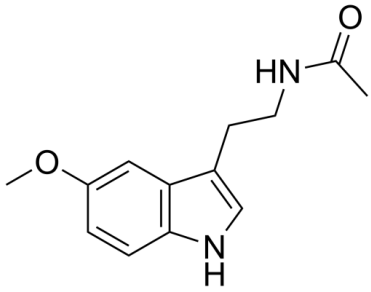
Data Sheet

Product Name:	Melatonin
Cat. No.:	CS-1769
CAS No.:	73-31-4
Molecular Formula:	$C_{13}H_{16}N_2O_2$
Molecular Weight:	232.28
Target:	Apoptosis; Autophagy; Endogenous Metabolite; Melatonin Receptor; Mitophagy
Pathway:	Apoptosis; Autophagy; GPCR/G Protein; Metabolic Enzyme/Protease; Neuronal Signaling
Solubility:	Ethanol : 12.5 mg/mL (ultrasonic);H_2O : 1.1 mg/mL (ultrasonic);DMSO : 100 mg/mL (ultrasonic)



BIOLOGICAL ACTIVITY:

Melatonin is a hormone made by the pineal gland that can activates **melatonin receptor**. Melatonin plays a role in sleep and possesses important antioxidative and anti-inflammatory properties[1][2][3]. Melatonin is a novel selective **ATF-6** inhibitor and induces human hepatoma cell apoptosis through COX-2 downregulation[4]. Melatonin attenuates palmitic acid-induced (HY-N0830) mouse granulosa cells apoptosis via endoplasmic reticulum stress[5]. ***In Vivo:***Melatonin increases the levels of activated PTEN, RSK-1, mTOR and AMPKα kinases, mildly inhibits ERK-1/2 phosphorylation and Bad phosphorylation, significantly inhibits phosphorylations of S6 Ribosomal Protein, 4E-BP1, GSK-3α and GSK-3β, and slightly increases PRAS40 phosphorylation in animals[1]. Melatonin ameliorates the neurotoxiciy and astrocyte activation induced by $A\beta_{1-42}$ in the cerebral cortex. Melatonin also blocks the reduction in Reelin and Dab1 expression induced by $A\beta_{1-42}$[2]. Melatonin treatment and lack of $NLRP3^{-/-}$ share similar inhibition of NF-κB and NLRP3 signaling pathway in mice. Melatonin treatment and lack of $NLRP3^{-/-}$ share some patterns of clock genes expression, and improve cardiomyocytes morphology in mice[3].

PROTOCOL (Extracted from published papers and Only for reference)

Animal Administration: Melatonin is dissolved in 0.9% isotonic saline/5% ethanol at a concentration of 4 mg/kg.[1]A total of two sets of adult male C57BL/6j mice weighing 21-26 g are randomly assigned to one of four groups and treated with intraperitoneal (i.p.) delivery of (i) vehicle (50 μL isotonic saline/5% ethanol), (ii) melatonin (4 mg/kg, dissolved in 0.9% isotonic saline/5% ethanol), (iii) Wortmannin, and (iv) melatonin/Wortmannin immediately after reperfusion. In the first set, mice are exposed to 30 min of focal cerebral ischemia (FCI) and 72 h reperfusion for the evaluation of disseminate ischemic injury in the striatum, and signaling pathway analysis (n=7 per group). The second group of mice is exposed to 90 min of FCI and 24 h reperfusion for the analysis of infarct development, brain swelling and IgG extravasation (n=7 per group).

References:

[1]. Kilic U, et al. Particular phosphorylation of PI3K/Akt on Thr308 via PDK-1 and PTEN mediates melatonin's neuroprotective activity after focal cerebral ischemia in mice. Redox Biol. 2017 Apr 5;12:657-665

[2]. Hu C, et al. Neuroprotective effect of melatonin on soluble Aβ1-42-induced cortical neurodegeneration via Reelin-Dab1 signaling pathway. Neurol Res. 2017 Apr 7:1-1

[3]. Rahim I, et al. Melatonin administration to wild-type mice and non-treated NLRP3 mutant mice share similar inhibition of the inflammatory response

during sepsis. J Pineal Res. 2017 Mar 31

[4]. Bu LJ, et al. Melatonin, a novel selective ATF-6 inhibitor, induces human hepatoma cell apoptosis through COX-2 downregulation. World J Gastroenterol. 2017 Feb 14;23(6):986-998.

[5]. Zhi Chen, et al. Melatonin attenuates palmitic acid-induced mouse granulosa cells apoptosis via endoplasmic reticulum stress. J Ovarian Res. 2019 May 10;12(1):43.

CAIndexNames:

Acetamide, N-[2-(5-methoxy-1H-indol-3-yl)ethyl]-

SMILES:

CC(NCCC1=CNC2=C1C=C(OC)C=C2)=O

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