

Product Data Sheet

Product Name: N-Acetylserotonin

Cat. No.: GC15365

Chemical Properties

Cas. No. 1210-83-9

Chemical Name N-[2-(5-hydroxy-1H-indol-3-yl)ethyl]-acetamide

SMILES CC(=O)NCCc1c[nH]c2cc(O)ccc12

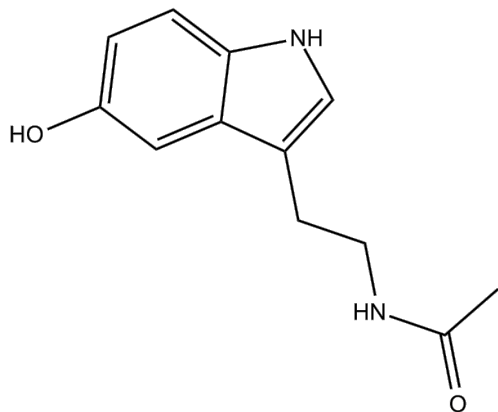
Formula $C_{12}H_{14}N_2O_2$ M.Wt 218.3

Solubility $\geq 11.5\text{mg/mL}$ in ethanol Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines primary cerebrocortical neurons (PCNs)

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Preparation Method PCNs, isolated from E14 to E16 mice, were preincubated with N-Acetylserotonin for 2h. PCNs were subjected to oxygen-glucose deprivation (OGD) or H₂O₂ (1000μmol/L) for 3 or 18h with or without a series of concentrations of N-Acetylserotonin. Then PCNs were stained with 20μM monodansylcadaverine (MDC) in PBS at 37°C for 1h. After washing with PBS, cells were immediately analyzed by fluorescence microscopy. Cell death of PCNs was quantitatively evaluated by the lactate dehydrogenase (LDH) assay.

Reaction Conditions 0.01nM, 0.1nM, 1nM, 10nM, 100nM, 1μM, 10μM, 100μM; 2h

Applications Incubation of PCNs with N-Acetylserotonin (0.01nM to 100μM) resulted in statistically significant inhibition of OGD-mediated PCN cell death. Exposing PCNs to H₂O₂ with N-Acetylserotonin resulted in better dose-dependent inhibition of cell death than the OGD-mediated one.

Animal experiment [2]:

Animal models TrkB F616A knock-in mouse model of neuron injury

Preparation Method Two- to three-month-old Brain-derived neurotrophic factor (BDNF) forebrain conditional knockout mice, which lack cortical, hippocampal, and retinal BDNF, were injected i.p. with N-Acetylserotonin (20mg/kg) or melatonin (1mg/kg). Mice were killed at 0, 0.5, 1, or 2h following drug administration. Brain lysates were prepared and analyzed by immunoblotting with anti-phospho-TrkB Y816.

Dosage form 20mg/kg; i.p.

Applications Both hippocampal and retinal TrkB phosphorylation was evident after 0.5h and peaked 1h following N-Acetylserotonin administration. In contrast, melatonin had no effect.

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References:

- [1] Zhou H, Wang J, Jiang J, et al. N-acetyl-serotonin offers neuroprotection through inhibiting mitochondrial death pathways and autophagic activation in experimental models of ischemic injury. *J Neurosci*. 2014;34(8):2967-2978.
- [2] Jang SW, Liu X, Pradoldej S, et al. N-acetylserotonin activates TrkB receptor in a circadian rhythm. *Proc Natl Acad Sci U S A*. 2010;107(8):3876-3881.

Background

N-Acetylserotonin, a chemical intermediate in melatonin biosynthesis, activates tyrosine kinase receptor B (TrkB) in a circadian rhythm manner. TrkB is a major neurotrophic factor receptor involved in cell survival, differentiation, and synaptic plasticity^[1-2]. N-Acetylserotonin is mainly synthesized in the pineal gland and is subsequently methylated by hydroxyindole-O-methyltransferase to synthesize melatonin^[3]. N-Acetylserotonin has neuroprotective properties and can exert neuroprotective effects by inhibiting oxidative stress, anti-apoptosis, regulating autophagy dysfunction, and anti-inflammation^[1].

In vitro, incubation of primary cerebrocortical neurons (PCNs) with N-Acetylserotonin (0.01nM-100µM; 2h) resulted in statistically significant inhibition of oxygen-glucose deprivation (OGD)-mediated PCN cell death, and exposing PCNs to H₂O₂ (1000µmol/L) with N-Acetylserotonin resulted in better dose-dependent inhibition of cell death^[4]. Pre-incubation neural progenitor cells (NPCs) with N-Acetylserotonin (0, 20, 50, 100, 200, and 400µM; 6h) not only ameliorates H₂O₂-induced cell viability loss, lactate dehydrogenase (LDH) release, and proliferative and migratory capacity impairments, but counteracts H₂O₂-triggered production of nitric oxide (NO), reactive oxygen species (ROS), malondialdehyde (MDA), and 8-hydroxy-deoxyguanosine (8-OHdG) in a dose-dependent manner^[5].

In vivo, the Brain-derived neurotrophic factor (BDNF) forebrain conditional knockout mice treated with N-Acetylserotonin (20mg/kg; i.p.), both hippocampal and retinal TrkB phosphorylation became evident after 0.5h and peaked 1h following N-Acetylserotonin administration^[3]. In the rat striatal injury model induced by 6-hydroxydopamine, treatment with N-Acetylserotonin (2, 5, 10mg/kg-d; i.p.) resulted in a decrease in the number of apomorphine-induced rotations, an increase in dopamine

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levels, and a decrease in both 3,4-dihydroxyphenylacetic acid and homovanillic acid levels^[6]. After treating the traumatic brain injury mouse with N-Acetylserotonin (10mg/kg; i.p.), a decrease in matrix metalloproteinase-9 (MMP-9) and increases in Claudin-5 and Bcl-2 were observed^[7].

References:

- [1] Kang JH, Guo XD, Wang YD, Kang XW. Neuroprotective Effects of N-acetylserotonin and Its Derivative. *Neuroscience*. 2023;517:18-25.
- [2] Boulle F, Kenis G, Cazorla M, et al. TrkB inhibition as a therapeutic target for CNS-related disorders. *Prog Neurobiol*. 2012;98(2):197-206.
- [3] Jang SW, Liu X, Pradoldej S, et al. N-acetylserotonin activates TrkB receptor in a circadian rhythm. *Proc Natl Acad Sci U S A*. 2010;107(8):3876-3881.
- [4] Zhou H, Wang J, Jiang J, et al. N-acetyl-serotonin offers neuroprotection through inhibiting mitochondrial death pathways and autophagic activation in experimental models of ischemic injury. *J Neurosci*. 2014;34(8):2967-2978.
- [5] Li Q, Wang P, Huang C, et al. N-Acetyl Serotonin Protects Neural Progenitor Cells Against Oxidative Stress-Induced Apoptosis and Improves Neurogenesis in Adult Mouse Hippocampus Following Traumatic Brain Injury. *J Mol Neurosci*. 2019;67(4):574-588.
- [6] Aguiar LM, Macedo DS, de Freitas RM, et al. Protective effects of N-acetylserotonin against 6-hydroxydopamine-induced neurotoxicity. *Life Sci*. 2005;76(19):2193-2202.
- [7] Rui T, Wang Z, Li Q, et al. A TrkB receptor agonist N-acetyl serotonin provides cerebral protection after traumatic brain injury by mitigating apoptotic activation and autophagic dysfunction. *Neurochem Int*. 2020;132:104606.

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