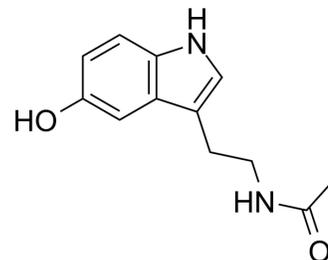


N-Acetyl-5-hydroxytryptamine

Cat. No.:	HY-107854		
CAS No.:	1210-83-9		
Molecular Formula:	C ₁₂ H ₁₄ N ₂ O ₂		
Molecular Weight:	218.25		
Target:	Endogenous Metabolite; Trk Receptor		
Pathway:	Metabolic Enzyme/Protease; Neuronal Signaling; Protein Tyrosine Kinase/RTK		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro	DMSO : 100 mg/mL (458.19 mM; Need ultrasonic)					
		Solvent Concentration	Mass	1 mg	5 mg	10 mg
	Preparing Stock Solutions	1 mM		4.5819 mL	22.9095 mL	45.8190 mL
		5 mM		0.9164 mL	4.5819 mL	9.1638 mL
10 mM			0.4582 mL	2.2910 mL	4.5819 mL	
Please refer to the solubility information to select the appropriate solvent.						
In Vivo	<ol style="list-style-type: none"> Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.5 mg/mL (11.45 mM); Clear solution Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.08 mg/mL (9.53 mM); Clear solution Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.08 mg/mL (9.53 mM); Clear solution 					

BIOLOGICAL ACTIVITY

Description	N-Acetyl-5-hydroxytryptamine is a Melatonin precursor, and that it can potently activate TrkB receptor.	
IC₅₀ & Target	Human Endogenous Metabolite	TrkB
In Vitro	N-Acetyl-5-hydroxytryptamine (NAS), a precursor of Melatonin, is acetylated from serotonin by AANAT (arylalkylamine N-acetyltransferase). N-acetylserotonin activates TrkB receptor in a circadian rhythm. N-Acetyl-5-hydroxytryptamine swiftly	

activates TrkB in a circadian manner and exhibits antidepressant effect in a TrkB-dependent manner. N-Acetyl-5-hydroxytryptamine rapidly activates TrkB, but not TrkA or TrkC, in a neurotrophin- and MT3 receptor-independent manner [1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

To explore whether N-Acetyl-5-hydroxytryptamine, can trigger TrkB activation in vivo, TrkB F616A knockin mice are employed, where it has been shown that TrkB F616A activation can be selectively blocked by 1NMPP1, a derivative of kinase inhibitor PP1, leading to TrkB-null phenotypes. To assess whether N-Acetyl-5-hydroxytryptamine can mimic BDNF, cortical neurons from TrkB F616A knockin mice are prepared. In alignment with a previous report, BDNF- and NAS-mediated TrkB phosphorylation are selectively reduced by 1NMPP1 but not by K252a, whereas serotonin or Melatonin had no effect. These findings suggest that NAS strongly provokes both wild-type TrkB and TrkB F616A tyrosine phosphorylation and activation[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [1]

Mice^[1]

Two-month-old TrkB F616A mice are pretreated with 1NMPP1 in drinking water (50 μ M) 1 day before the experiment, followed by administration of N-Acetyl-5-hydroxytryptamine (20 mg/kg, i.p.) or Melatonin (1 mg/kg, i.p.). Mice are killed at 1 h. The brain homogenates are analyzed by immunoblotting with anti-p-TrkB. Two- to three-month-old BDNF forebrain conditional knockout mice are injected i.p. with N-Acetyl-5-hydroxytryptamine or Melatonin. Mice are killed at 0, 0.5, 1, or 2 h following drug administration. Brain lysates are prepared and analyzed by immunoblotting with anti-phospho-TrkB Y816 [1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Immunother Cancer. 2021 Jul;9(7):e002383.
- Diabetes. 2024 Mar 12;db230553.

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REFERENCES

[1]. Jang SW, et al. N-acetylserotonin activates TrkB receptor in a circadian rhythm. Proc Natl Acad Sci U S A. 2010 Feb 23;107(8):3876-81.

Caution: Product has not been fully validated for medical applications. For research use only.

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