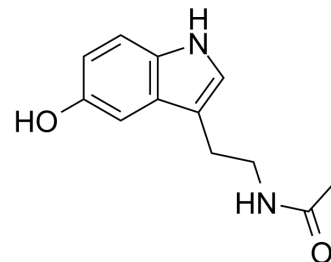


## N-Acetyl-5-hydroxytryptamine

<b>Cat. No.:</b>	HY-107854		
<b>CAS No.:</b>	1210-83-9		
<b>Molecular Formula:</b>	C <sub>12</sub> H <sub>14</sub> N <sub>2</sub> O <sub>2</sub>		
<b>Molecular Weight:</b>	218.25		
<b>Target:</b>	Endogenous Metabolite; Trk Receptor		
<b>Pathway:</b>	Metabolic Enzyme/Protease; Neuronal Signaling; Protein Tyrosine Kinase/RTK		
<b>Storage:</b>	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



### SOLVENT & SOLUBILITY

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

### BIOLOGICAL ACTIVITY

<b>Description</b>	N-Acetyl-5-hydroxytryptamine is a Melatonin precursor, and that it can potently activate TrkB receptor.	
<b>IC<sub>50</sub> &amp; Target</b>	Human Endogenous Metabolite	TrkB
<b>In Vitro</b>	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<sup>[1]</sup>

Two-month-old TrkB F616A mice are pretreated with 1NMPP1 in drinking water (50  $\mu$ 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.

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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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