## N-(p-amylcinnamoyl) Anthranilic Acid

Cat. No.:	IY-118628			
CAS No.:	10683-10-8			
Molecular Formula:	C <sub>21</sub> H <sub>23</sub> NO <sub>3</sub>			
Molecular Weight:	37.41			
Target:	Phospholipase; TRP Channel			
Pathway:	letabolic Enzyme/Protease; Membrane Transporter/Ion Channel; Neuronal Signaling			
Storage:	Powder -20°C 3 years			
	4°C 2 years			
	n solvent -80°C 2 years			
	-20°C 1 year			

## SOLVENT & SOLUBILITY

	Preparing Stock Solutions	Solvent Mass Concentration	1 mg	5 mg	10 mg		
		1 mM	2.9638 mL	14.8188 mL	29.6375 mL		
		5 mM	0.5928 mL	2.9638 mL	5.9275 mL		
		10 mM	0.2964 mL	1.4819 mL	2.9638 mL		
	Please refer to the so	Please refer to the solubility information to select the appropriate solvent.					
In Vivo		1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.08 mg/mL (6.16 mM); Clear solution					
		<ol> <li>Add each solvent one by one: 10% DMSO &gt;&gt; 90% corn oil</li> <li>Solubility: ≥ 2.08 mg/mL (6.16 mM); Clear solution</li> </ol>					

BIOLOGICAL ACTIVITY				
Description	N-(p-amylcinnamoyl) Anthranilic Acid (ACA) is a broad spectrum Phospholipase A <sub>2</sub> (PLA <sub>2</sub> ) inhibitor and TRP channel blocker <sup>[1][2]</sup> . N-(p-amylcinnamoyl) Anthranilic Acid (ACA) is also an effective reversible inhibitor of calcium-activated chloride channels, has potential to treat arrhythmia <sup>[3]</sup> .			
IC <sub>50</sub> & Target	PLA <sub>2</sub> <sup>[1][2]</sup> . TRP channel <sup>[1][2]</sup> .Calcium-activated chloride channels <sup>[3]</sup> .			
In Vitro	N-(p-amylcinnamoyl) Anthranilic Acid (ACA; 20 μM) completely blocks ADPR-induced whole-cell currents and H <sub>2</sub> O <sub>2</sub> -induced Ca <sup>2+</sup> signals (IC <sub>50</sub> =1.7 μM) in HEK293cells transfected with human TRPM2 <sup>[1]</sup> . N-(p-amylcinnamoyl) Anthranilic Acid (ACA; 20 μM) also blocks currents through human TRPM8 and TRPC6 expressed in			

**Product** Data Sheet



HEK293 cells<sup>[1]</sup>.

N-(p-amylcinnamoyl) Anthranilic Acid (ACA) modulates the activity of different TRP channels independent of PLA2<sub>2</sub> inhibition<sup>[1]</sup>.

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

## **CUSTOMER VALIDATION**

- Int J Clin Exp Med. 2023 Jun 1.
- Oxid Med Cell Longev. 2021 Jul 27;2021:7356266.

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## REFERENCES

[1]. Kraft R, et al. Inhibition of TRPM2 cation channels by N-(p-amylcinnamoyl)anthranilic acid. Br J Pharmacol. 2006 Jun;148(3):264-73.

[2]. Harteneck C, et al. N-(p-amylcinnamoyl)anthranilic acid (ACA): a phospholipase A(2) inhibitor and TRP channel blocker. Cardiovasc Drug Rev. 2007 Spring;25(1):61-75.

[3]. Gwanyanya A, et al. Inhibition of the calcium-activated chloride current in cardiac ventricular myocytes by N-(p-amylcinnamoyl)anthranilic acid (ACA). Biochem Biophys Res Commun. 2010 Nov 19;402(3):531-6.

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

 Tel: 609-228-6898
 Fax: 609-228-5909
 E-mail: tech@MedChemExpress.com

 Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA