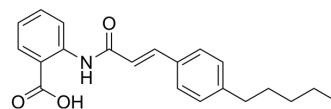


## N-(p-aminocinnamoyl) Anthranilic Acid

<b>Cat. No.:</b>	HY-118628		
<b>CAS No.:</b>	110683-10-8		
<b>Molecular Formula:</b>	C <sub>21</sub> H <sub>23</sub> NO <sub>3</sub>		
<b>Molecular Weight:</b>	337.41		
<b>Target:</b>	Phospholipase; TRP Channel		
<b>Pathway:</b>	Metabolic Enzyme/Protease; Membrane Transporter/Ion Channel; Neuronal Signaling		
<b>Storage:</b>	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



### SOLVENT & SOLUBILITY

#### In Vitro

DMSO : ≥ 125 mg/mL (370.47 mM)  
 \* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		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 solubility information to select the appropriate solvent.

#### In Vivo

- 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
- Add each solvent one by one: 10% DMSO >> 90% corn oil  
 Solubility: ≥ 2.08 mg/mL (6.16 mM); Clear solution

### BIOLOGICAL ACTIVITY

#### Description

N-(p-aminocinnamoyl) Anthranilic Acid (ACA) is a broad spectrum Phospholipase A<sub>2</sub> (PLA<sub>2</sub>) inhibitor and TRP channel blocker [1][2]. N-(p-aminocinnamoyl) Anthranilic Acid (ACA) is also an effective reversible inhibitor of calcium-activated chloride channels, has potential to treat arrhythmia [3].

#### IC<sub>50</sub> & Target

PLA<sub>2</sub> [1][2].  
 TRP channel [1][2]. Calcium-activated chloride channels [3].

#### In Vitro

N-(p-aminocinnamoyl) 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 HEK293 cells transfected with human TRPM2 [1].

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N-(p-aminocinnamoyl) Anthranilic Acid (ACA; 20  $\mu$ M) also blocks currents through human TRPM8 and TRPC6 expressed in HEK293 cells<sup>[1]</sup>.

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

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

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## CUSTOMER VALIDATION

- 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-aminocinnamoyl)anthranilic acid. Br J Pharmacol. 2006 Jun;148(3):264-73.

[2]. Harteneck C, et al. N-(p-aminocinnamoyl)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-aminocinnamoyl)anthranilic acid (ACA). Biochem Biophys Res Commun. 2010 Nov 19;402(3):531-6.

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

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