N-(p-amylcinnamoyl) Anthranilic Acid

Cat. No.: HY-118628

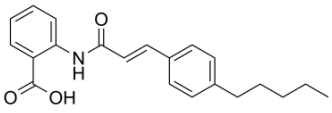
CAS No.: 110683-10-8

Molecular Formula: $C_{21}H_{23}NO_3$

Molecular Weight: 337.41

Target: Phospholipase; TRP Channel

Pathway: Metabolic Enzyme/Protease; Membrane Transporter/Ion Channel; Neuronal Signaling



Storage:

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)

H_2O : < 0.1 mg/mL (insoluble)

* "≥" means soluble, but saturation unknown.

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 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
2. 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-amylcinnamoyl) Anthranilic Acid (ACA) is a broad spectrum Phospholipase A_2 (PLA_2) inhibitor and TRP channel blocker [1][2]. N-(p-amylcinnamoyl) Anthranilic Acid (ACA) is also an effective reversible inhibitor of calcium-activated chloride channels, has potential to treat arrhythmia[3].

IC_{50} & Target

PLA_2[1][2].

TRP channel[1][2].Calcium-activated chloride channels[3].

In Vitro

N-(p-amylcinnamoyl) Anthranilic Acid (ACA; 20 μM) completely blocks ADPR-induced whole-cell currents and H_2O_2-induced

Ca^{2+} signals (IC_{50}=1.7 μM) in HEK293cells transfected with human TRPM2[1].

N-(p-amylcinnamoyl) Anthranilic Acid (ACA; 20 μM) also blocks currents through human TRPM8 and TRPC6 expressed in HEK293 cells[1].

N-(p-amylcinnamoyl) Anthranilic Acid (ACA) modulates the activity of different TRP channels independent of $PLA2_2$ inhibition[1].

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

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.

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