(E)-Cardamonin

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Cat. No.:	HY-N1378			
CAS No.:	19309-14-9			
Molecular Formula:	$C_{16}H_{14}O_4$			
Molecular Weight:	270.28			
Target:	TRP Channel; Apoptosis			
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling; Apoptosis			
Storage:	Powder	-20°C	3 years	
		4°C	2 years	
	In solvent	-80°C	2 years	
		-20°C	1 year	

SOLVENT & SOLUBILITY

		Solvent Mass Concentration	1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM	3.6999 mL	18.4993 mL	36.9987 mL	
	5 mM	0.7400 mL	3.6999 mL	7.3997 mL	
	10 mM	0.3700 mL	1.8499 mL	3.6999 mL	
	Please refer to the so	lubility information to select the app	propriate solvent.		

BIOLOGICAL ACTIVITY			
Description	(E)-Cardamonin ((E)-Cardamomin) is a novel antagonist of hTRPA1 cation channel with an IC ₅₀ of 454 nM.		
IC ₅₀ & Target	IC50: 454 nM (hTRPA1 cation channel) ^[1]		
In Vitro	 (E)-Cardamonin ((E)-Cardamomin) selectively blocksTRPA1 activation (IC₅₀=454 nM) while does not interact with TRPV1 nor TRPV4 channel. Docking analysis of cardamonin demonstrates a compatible interaction with A-967079-binding site of TRPA1. (E)-Cardamonin ((E)-Cardamomin) does not significantly reduce HEK293 cell viability, nor does it impair cardiomyocyte constriction^[1]. (E)-Cardamonin ((E)-Cardamomin) suppresses the expression of Tgase-2, cyclooxygenase-2 (COX-2), and p65 (nuclear factor-κB) in a concentration-dependent manner, and restores the expression of IkB in MG63 and Raw264.7 cells^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only. 		

Product Data Sheet

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In Vivo (E)-Cardamonin ((E)-Cardamomin) (3-30 mg/kg, orally administered) significantly inhibits PBQ-induced writhing. CDN also produces a significant, dose-dependent increase in the withdrawal response latencies in carrageenan-induced hyperalgesia ^[2].

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PROTOCOL	
Cell Assay ^[1]	HEK293 cells are treated with (E)-Cardamonin ((E)-Cardamomin) (0-90 μM). The cells treated in the absence of the test compound are the negative control. After incubated for 24 h, Cell Titer-Glo reagent is added to the cells and Luminescence is acquired on the plate reader ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration ^{[1][2]}	Rats: The rats are divided into groups of six according to their nociceptive pressure thresholds, after which carrageenan (0.1 mL, 1%) is injected into the plantar surface of the left hind paw. The rats received vehicle or (E)-Cardamonin ((E)-Cardamomin) (3-30 mg/kg) or indomethacin (3 mg/kg) orally 2 h after carrageenan injection and are evaluated for paw hyperalgesia 0, 1 and 2 h after administration of compounds. Indomethacin is used as a positive control ^[2] . Mice: Acute pain is induced by an intraperitoneal injection of 0.2 mL of 0.02% PBQ 54 min after oral administration of (E)-Cardamonin ((E)-Cardamonin). Six minutes after the PBQ injection, the total number of writhes is counted for 6 min. The control animals received an appropriate volume of dosing vehicle (80% saline, 10% ethanol and 10% Tween 80). Indomethacin is used as a positive control ^[2] .

CUSTOMER VALIDATION

• Nutrients. 2021, 13(10), 3382.

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REFERENCES

[1]. Wang S, et al. Cardamonin, a Novel Antagonist of hTRPA1 Cation Channel, Reveals Therapeutic Mechanism of Pathological Pain. Molecules. 2016 Aug 29;21(9). pii: E1145.

[2]. Park MK, et al. Novel anti-nociceptive effects of cardamonin via blocking expression of cyclooxygenase-2 andtransglutaminase-2. Pharmacol Biochem Behav. 2014 Mar;118:10-5.

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

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