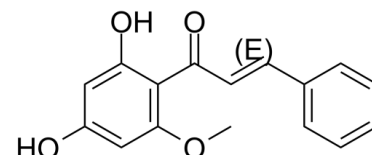


## Data Sheet

<b>Product Name:</b>	(E)-Cardamonin
<b>Cat. No.:</b>	CS-6403
<b>CAS No.:</b>	19309-14-9
<b>Molecular Formula:</b>	C <sub>16</sub> H <sub>14</sub> O <sub>4</sub>
<b>Molecular Weight:</b>	270.28
<b>Target:</b>	Apoptosis; TRP Channel
<b>Pathway:</b>	Apoptosis; Membrane Transporter/Ion Channel; Neuronal Signaling
<b>Solubility:</b>	DMSO : ≥ 28 mg/mL (103.60 mM); H <sub>2</sub> O : < 0.1 mg/mL (insoluble)



### BIOLOGICAL ACTIVITY:

(E)-Cardamonin ((E)-Cardamomin) is a novel antagonist of **hTRPA1** cation channel with an **IC<sub>50</sub>** of 454 nM. IC<sub>50</sub> & Target: IC<sub>50</sub>: 454 nM (hTRPA1 cation channel)<sup>[1]</sup> **In Vitro:** (E)-Cardamonin ((E)-Cardamomin) selectively blocks TRPA1 activation (IC<sub>50</sub>=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<sup>[1]</sup>. (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 IκB in MG63 and Raw264.7 cells<sup>[2]</sup>. **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<sup>[2]</sup>.

### PROTOCOL (Extracted from published papers and Only for reference)

**Cell Assay:** <sup>[1]</sup>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<sup>[1]</sup>. **Animal Administration:** Cardamonin is prepared in 80% saline, 10% ethanol and 10% Tween 80.<sup>[1][2]</sup> **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<sup>[2]</sup>.

**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)-Cardamomin). 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<sup>[2]</sup>.

### 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 and transglutaminase-2. *Pharmacol Biochem Behav*. 2014 Mar;118:10-5.

**CAIndexNames:**

2-Propen-1-one, 1-(2,4-dihydroxy-6-methoxyphenyl)-3-phenyl-, (2E)-

**SMILES:**

O=C(C1=C(OC)C=C(O)C=C1O)/C=C/C2=CC=CC=C2.[(E)]

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

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