Paradol

Cat. No.: HY-14617

CAS No.: 27113-22-0

Molecular Formula: $C_{17}H_{26}O_3$

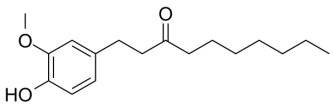
Molecular Weight: 278.39

Target: COX

Pathway: Immunology/Inflammation

Storage:

Pure form	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 140 mg/mL (502.89 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.5921 mL	17.9604 mL	35.9208 mL
5 mM	0.7184 mL	3.5921 mL	7.1842 mL
10 mM	0.3592 mL	1.7960 mL	3.5921 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: ≥ 2.5 mg/mL (8.98 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.98 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.98 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Paradol is a pungent phenolic substance found in ginger and other Zingiberaceae plants. Paradol is an effective inhibitor of tumor promotion in mouse skin carcinogenesis, binds to cyclooxygenase (COX)-2 active site.

IC_{50} & Target

COX-2

In Vitro

Paradol ([6]-paradol) induces apoptosis in an oral squamous carcinoma cell line, KB, in a dose-dependent manner. Paradol

	induces apoptosis through a caspase-3-dependent mechanism[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Administration of Paradol (6-paradol) (10 mg/kg) clearly reduces the number of Iba1-positive cells 1 and 3 days after the challenge. Moreover, Paradol dramatically reduces the number of Iba1-postive cells in periischemic regions even after 3 days following M/R challenge[3]. Paradol (6-paradol) exhibits the strongest anti-inflammatory effect of several paradol compounds in lipopolysaccharide-stimulated BV2 microglia derived from a mouse brain, including 2-, 4-, 6-, 8-, and 10-paradol. Furthermore, Paradol shows the strongest pungency of all of the known paradol analogues. Paradol also shows the highest contact time at the antiobesity site of action on the basis of the results shown for the absorption of the metabolites in this study[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	KB, human oral epidermoid carcinoma cell lines (ATCC CCL-17) are plated at a density of 5×10^3 cells/200 μL/well into 96-well plate. After an overnight growth, the cells are treated with a series of paradol derivatives. All of the derivatives of paradol tested are dissolved in DMSO. The final concentration of DMSO in the culture medium is kept below 0.1% and the controls are treated with DMSO alone. Cell viability is assessed using MTT assay. In brief, after the cells are grown in the media in the absence or presence of the test compounds (e.g., Paradol, 10, 50, 100, 150, and 200 μM) for 48 h, they are then replaced to a 200 μL culture medium containing 0.5 mg/mL MTT for 3 h. The resulting MTT-formazan product is dissolved by an addition of the same volume of DMSO. The amount of formazan is determined by measuring the absorbance at 570 nm[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3][4]	Mice[3] Male ICR mice (7 weeks old, 36±2 g) challenged with middle cerebral artery occlusion (MCAO)/reperfusion (M/R) are randomly divided into vehicle (10% Tween80)- or Paradol-administered groups (n=6~7 per group). Paradol dissolved in 10% Tween80 is orally administered (10 mg/kg) into mice at 1, 5, or 10 mg/kg immediately after reperfusion. Rats[4] Five-week-old Sprague-Dawley rats (male) are used. At 8 weeks of age, the rats are fasted for 14 h prior to the oral administration of olive oil (1 mL) containing zingerone or 6-, 8-, or 12-paradol (10 mg/kg). Three rats in each group are anesthetized with isoflurane, and samples (0.3 mL) of their blood are collected from their jugular vein using a heparinized needle and syringe at 0 (i.e., prior to the oral administration), 0.25, 0.5, 1, 3, 6, and 24 h after the oral administration of the olive oil containing test compounds. The $AUC_{0\text{-}24h}$ values determined using this time schedule are very similar compared with $AUC_{0\text{-}24h}$ that sampled the time points more minutely with other materials in our laboratory. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Sci Adv Mater. 2019 Oct;11(10):1467-1473.
- Research Square Preprint. 2021 May.
- Engineering Journal. 2017 21 (4), ISSN 0125-8281.

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REFERENCES

[1]. van Breemen RB, et al. Cyclooxygenase-2 inhibitors in ginger (Zingiber officinale). Fitoterapia. 2011 Jan;82(1):38-43.

[2]. Keum YS, et al. Induction of apoptosis and caspase-3 activation by chemopreventive [6]-paradol and structurally related compounds in KB cells. Cancer Lett. 2002 Mar 8;177(1):41-7.

[3]. Gaire BP, et al. Neuroprotective effect of 6-paradol in focal cerebral ischemia involves the attenuation of neuroinflammatory responses in activated microglia. PLoS One. 2015 Mar 19;10(3):e0120203.

[4]. Setoguchi S, et al. Pharmacokinetics of Paradol Analogues Orally Administered to Rats. J Agric Food Chem. 2016 Mar 9;64(9):1932-7.

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