Citric acid

Cat. No.: HY-N1428

CAS No.: 77-92-9

Molecular Formula: $C_6H_8O_7$

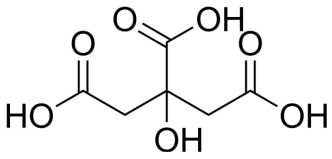
Molecular Weight: 192.12

Target: Apoptosis; Endogenous Metabolite; Antibiotic

Pathway: Apoptosis; Metabolic Enzyme/Protease; Anti-infection

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (520.51 mM; Need ultrasonic)

H_2O : 100 mg/mL (520.51 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.2051 mL	26.0254 mL	52.0508 mL
5 mM	1.0410 mL	5.2051 mL	10.4102 mL
10 mM	0.5205 mL	2.6025 mL	5.2051 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (520.51 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (13.01 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (13.01 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (13.01 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Citric acid is a natural preservative and food tartness enhancer. Citric acid induces apoptosis and cell cycle arrest at G2/M phase and S phase in HaCaT cells. Citric acid cause oxidative damage of the liver by means of the decrease of antioxidative enzyme activities. Citric acid causes renal toxicity in mice[1][2][3].

IC_{50} & Target	Human Endogenous Metabolite

In Vitro

Citric acid (0-12.5 mM; 24 h) shows antiproliferative activity in a dose dependent manner[3].
?Citric acid (12.5 mM; 72 h) induces apoptosis and cell cycle arrest at G2/M phase and S phase in a dosedependent manner[3].
?Citric acid (12.5 mM; 48 h) increases the expression of FAS, BAX, BID, AIF, EndoG, cytochrome c, PARP, GADD153, GRP78 and caspase-3, -8, -9, and decreases of BCL-2 and BCL-Xl[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[3]

Cell Line:	HaCaT cells
Concentration:	0, 2.5, 5, 7.5, 10, 12.5 mM
Incubation Time:	24 h
Result:	Inhibited the cell viability in a dose dependent manner.

Cell Cytotoxicity Assay[3]

Cell Line:	HaCaT cells
Concentration:	12.5 mM
Incubation Time:	0, 12, 24, 48, 72 h
Result:	Induced apoptosis and cell cycle arrest at G2/M phase and S phase in a dosedependent manner.

Western Blot Analysis[3]

Cell Line:	HaCaT cells
Concentration:	12.5 mM
Incubation Time:	12, 24, 48 h
Result:	Increased the expression of FAS, BAX, BID, AIF, EndoG, cytochrome c, PARP, GADD153, GRP78 and caspase-3, -8, -9, and decreased of BCL-2 and BCL-Xl.

In Vivo

Citric acid (120, 240, and 480 mg/kg; i.p.) significantly decreases GSH-Px activity and induces an increase in the MDA (malonyldialdehyde) levels in mouse liver[1].
Citric acid (120, 240, and 480 mg/kg; i.p.) induces apoptosis by increases caspase-3 activity in a dose-dependent manner in mouse hepatocytes[1].
?Citric acid (120, 240, and 480 mg/kg; i.p.; weekly for 3 weeks) causes renal toxicity in mice[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	20 g male Kunming mice[2]
Dosage:	120, 240, 480 mg/kg
Administration:	I.p.; weekly for 3 weeks
Result:	T-SOD and GSH-Px activities in the treated groups decreased with increasing doses of citric acid, NOS activity tended to increase, and H2O2 and MDA contents gradually decreased.

CUSTOMER VALIDATION

- Food Chem. 2022: 134807.
- Insect Biochem Mol Biol. 2023 May 12;103958.
- New J Chem. 03 Aug 2022.

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REFERENCES

[1]. Chen X, et al. Study on injury effect of food additive citric acid on liver tissue in mice. Cytotechnology. 2014 Mar;66(2):275-82.

[2]. Chen X, Lv Q, Liu Y, Deng W. Effects of the food additive, citric acid, on kidney cells of mice. Biotech Histochem. 2015 Jan;90(1):38-44.

[3]. Ying TH, et al. Citric acid induces cell-cycle arrest and apoptosis of human immortalized keratinocyte cell line (HaCaT) via caspase- and mitochondrial-dependent signaling pathways. Anticancer Res. 2013 Oct;33(10):4411-20.

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