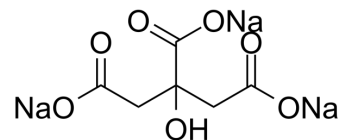


## Citric acid trisodium

<b>Cat. No.:</b>	HY-B2201
<b>CAS No.:</b>	68-04-2
<b>Molecular Formula:</b>	C <sub>6</sub> H <sub>5</sub> Na <sub>3</sub> O <sub>7</sub>
<b>Molecular Weight:</b>	258.07
<b>Target:</b>	Apoptosis; Endogenous Metabolite
<b>Pathway:</b>	Apoptosis; Metabolic Enzyme/Protease
<b>Storage:</b>	4°C, sealed storage, away from moisture * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



### SOLVENT & SOLUBILITY

#### In Vitro

H<sub>2</sub>O : 50 mg/mL (193.75 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	3.8749 mL	19.3746 mL	38.7492 mL
	5 mM	0.7750 mL	3.8749 mL	7.7498 mL
	10 mM	0.3875 mL	1.9375 mL	3.8749 mL

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

### BIOLOGICAL ACTIVITY

#### Description

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

#### IC<sub>50</sub> & Target

Human Endogenous Metabolite

#### In Vitro

Citric acid trisodium (0-12.5 mM; 24 h) shows antiproliferative activity in a dose dependent manner<sup>[3]</sup>.  
 Citric acid trisodium (12.5 mM; 72 h) induces apoptosis and cell cycle arrest at G2/M phase and S phase in a dosedependent manner<sup>[3]</sup>.  
 Citric acid trisodium (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<sup>[3]</sup>.  
 MCE has not independently confirmed the accuracy of these methods. They are for reference only.  
 Cell Viability Assay<sup>[3]</sup>

Cell Line:	HaCaT cells
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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 Cycle Analysis <sup>[3]</sup>	
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 <sup>[3]</sup>	
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 trisodium (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<sup>[1]</sup>.  
 Citric acid trisodium (120, 240, and 480 mg/kg; i.p.) induces apoptosis by increases caspase-3 activity in a dose-dependent manner in mouse hepatocytes<sup>[1]</sup>.  
 Citric acid trisodium (120, 240, and 480 mg/kg; i.p.; weekly for 3 weeks) causes renal toxicity in mice<sup>[2]</sup>.  
 MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	20 g male Kunming mice <sup>[2]</sup>
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.

See more customer validations on [www.MedChemExpress.com](http://www.MedChemExpress.com)

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

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