Tiron

Cat. No.: HY-D0261

CAS No.: 149-45-1

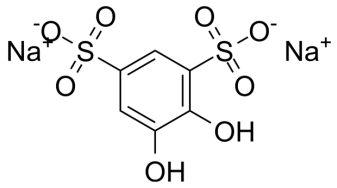
Molecular Formula: $C_6H_4Na_2O_8S_2$

Molecular Weight: 314.2

Target: Biochemical Assay Reagents; Apoptosis

Pathway: Others; Apoptosis

Storage: 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

DMSO : 83.33 mg/mL (265.21 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.1827 mL	15.9134 mL	31.8269 mL
	5 mM	0.6365 mL	3.1827 mL	6.3654 mL
	10 mM	0.3183 mL	1.5913 mL	3.1827 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: ≥ 2.08 mg/mL (6.62 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (6.62 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Tiron is a non-toxic chelator of a variety of metals. Tiron is cell permeable analog of vitamin E and function as hydroxyl radical and superoxide scavenger. Tiron is an orally active antioxidant. Tiron can be used to alleviate acute metal overload in animals[1][2][3].

In Vitro

Tiron (10 mM) protects Chinese hamster V79 cells against H_2O_2-induced cytotoxicity[1].
Tiron (0-20 mM) protects supercoiled DNA from metal-mediated superoxide-dependent strand breaks[1].
Tiron (50 nM-200 nM, 48 h) inhibits HG-induced neonatal rat cardiomyocytes apoptosis[3].
Tiron (50 nM-200 nM, 48 h) reduces intracellular osteopontin in neonatal rat cardiomyocytes[3].
Tiron (0.2 mM, 2 h) inhibits UVB-induced up-regulation of MMP-1 and MMP-3 in HDFs[4].
Tiron (0.7 mM, 48 h) increases the percentage of PT4 cells in both the S and G2/M phases[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cycle Analysis[5]

Cell Line:	PT4 cells
Concentration:	0.7 mM
Incubation Time:	48 h
Result:	Increased the percentage of PT4 cells in S and G2/M phases, along with a reduction of cells in the G0/G1 phase.

In Vivo

Tiron (200 mg/kg, oral gavage) ameliorates oxidative stress and inflammation in a murine model of airway remodeling[2].
Tiron (300 mg/kg, i.p., daily for two weeks) alleviated apoptosis of the left ventricular cardiomyocytes in STZ-induced diabetic mice[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	BALB/c mice challenged with Ovalbumin (OVA) aerosol for 8 weeks[2]
Dosage:	200 mg/kg
Administration:	Oral gavage.
Result:	Inhibited levels of NOx, IL-13 and TGF-β1, and immunoreactivity of NF-κB.

Animal Model:	STZ-induced diabetic mice[3]
Dosage:	300 mg/kg
Administration:	i.p., daily for two weeks.
Result:	Reduced the levels of total oxidized proteins and advanced glycation end products (AGEs)-related proteins. Inhibited cardiac myocyte apoptosis. Decreased PKCδ localization on plasma membrane.

REFERENCES

[1]. Krishna CM, et al. The catecholic metal sequestering agent 1,2-dihydroxybenzene-3,5-disulfonate confers protection against oxidative cell damage. Arch Biochem Biophys. 1992 Apr;294(1):98-106. 2.

[2]. El-Sherbeeny NA, et al. Tiron ameliorates oxidative stress and inflammation in a murine model of airway remodeling. Int Immunopharmacol. 2016 Oct;39:172-180.

[3]. Jiang P, et al. Tiron ameliorates high glucose-induced cardiac myocyte apoptosis by PKCδ-dependent inhibition of osteopontin. Clin Exp Pharmacol Physiol. 2017 Jul;44(7):760-770.

[4]. Lu J, et al. Tiron Inhibits UVB-Induced AP-1 Binding Sites Transcriptional Activation on MMP-1 and MMP-3 Promoters by MAPK Signaling Pathway in Human Dermal Fibroblasts. PLoS One. 2016 Aug 3;11(8):e0159998.

[5]. Monticone M, et al. NAC, tiron and trolox impair survival of cell cultures containing glioblastoma tumorigenic initiating cells by inhibition of cell cycle progression. PLoS One. 2014 Feb 28;9(2):e90085.

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

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