L-Ascorbic acid

Cat. No.: HY-B0166

CAS No.: 50-81-7

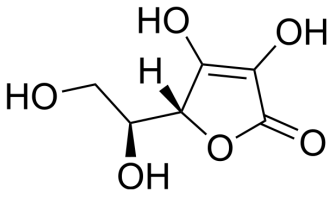
Molecular Formula: $C_6H_8O_6$

Molecular Weight: 176.12

Target: Reactive Oxygen Species; Apoptosis; Calcium Channel; Endogenous Metabolite

Pathway: Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Apoptosis; Membrane Transporter/Ion Channel; Neuronal Signaling

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 100 mg/mL (567.79 mM)
DMSO : 100 mg/mL (567.79 mM; Need ultrasonic)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.6779 mL	28.3897 mL	56.7795 mL
5 mM	1.1356 mL	5.6779 mL	11.3559 mL
10 mM	0.5678 mL	2.8390 mL	5.6779 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 (567.79 mM); Clear solution; Need ultrasonic and warming and heat to 60°C

BIOLOGICAL ACTIVITY

Description

L-Ascorbic acid (L-Ascorbate), an electron donor, is an endogenous antioxidant agent. L-Ascorbic acid inhibits selectively Ca_v 3.2 channels with an IC_{50} of 6.5 μM. L-Ascorbic acid is also a collagen deposition enhancer and an elastogenesis inhibitor[1][2][3]. L-Ascorbic acid exhibits anti-cancer effects through the generation of reactive oxygen species (ROS) and selective damage to cancer cells[4].

IC_{50} & Target			
	T-type calcium channel	Microbial Metabolite	Human Endogenous Metabolite

In Vitro

The anti-cancer effects of L-Ascorbic acid are determined by sodium-dependent vitamin C transporter 2 (SVCT-2), a transporter of L-ascorbic acid. L-Ascorbic acid (0.1 μM-2 mM) exhibits anti-cancer effects according to SVCT-2 expression and L-ascorbic acid uptake. Human colorectal cancer cell lines displays differential responses to L-ascorbic acid, primarily depending on the expression level of SVCT-2[4].

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

Cell Viability Assay[4]

Cell Line:	High SVCT-2 expressing cell lines Sw620, Sw480, LoVo, SNU-C4; Low SVCT-2 expressing cell lines HCT15, HCT116, DLD-1, CoLo-205
Concentration:	0, 0.1 μM, 1 μM, 10 μM, 0.1 mM, 0.5 mM, 1 mM, and 2 mM
Incubation Time:	24 hours
Result:	Some high SVCT-2 expressing cancer cells demonstrated a dramatic cell-autonomous inhibitory effect of L-ascorbic acid. Low SVCT-2 expressing cell lines showed biphasic responses to L-ascorbic acid.

Western Blot Analysis[4]

Cell Line:	Sw620, Sw480, LoVo, SNU-C4, HCT15, HCT116, DLD-1, CoLo-205 cell lines
Concentration:	1 mM
Incubation Time:	
Result:	The cell lines showed different levels of SVCT-2 expression in western blot analyses: Sw620, Sw480, and Lovo expressed high levels of SVCT-2 whereas HCT116, HCT15, and DLD-1 expressed low levels.

In Vivo

L-Ascorbic acid/Tolbutamide produces hypoglycaemic activity in a dose dependant manner in normal (60 mg/kg) and diabetic (40 mg/kg) condition. In the presence of L-ascorbic acid, Tolbuatmide (20 mg/kg) produces early onset of action and maintained for longer period compared to Tolbutamide matching control[5].

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

Animal Model:	Normal rats:Albino rats of either sex weighing between 125-175 g[5]
Dosage:	Group I received L-ascorbic acid 60 mg/kg, Group II received Tolbutamide 20 mg/kg and Group III was given L-ascorbic acid (60 mg/kg) prior to the administration of tolbutamide 20 mg/kg
Administration:	Administered orally
Result:	L-ascorbic acid at the dose of 60 mg/kg produced 50.91% blood glucose reduction at 0.5 h and 20 mg/kg body weight of Tolbutamide produced 33% at 4 h as peak effects. In the presence of L-ascorbic acid (60 mg/kg), the action of Tolbutamide was early in onset and maintained for 6 h.

Animal Model:	Diabetic rats:Albino rats of either sex weighing between 125 to 175 g were fasted overnight before injection with Alloxan[5]
Dosage:	Group I received L-ascorbic acid 40 mg/kg and Group II received Tolbutamide 20 mg/kg while Group III was given L-ascorbic acid 40 mg/kg prior to Tolbutamide administration (20 mg/kg).
Administration:	Oral administration
Result:	L-ascorbic acid (40 mg/kg alone) produced 42.53% blood glucose reduction at 1.5 h and Tolbutamide 20 mg/kg produced 45.09 at 4 h. Administration of L-ascorbic acid 40 mg/kg

	body weight prior to Tolbutamide produced antidiabetic activity at 0.5 h and was maintained for 6 h.

CUSTOMER VALIDATION

- Cancer Cell. 2024 Feb 23:S1535-6108(24)00046-1.
- Nat Immunol. 2022 Dec 21.
- Mil Med Res. 2020 Nov 1;7(1):52.
- Redox Biol. 2022 Aug;54:102392.
- Sci China Life Sci. 2018 Oct;61(10):1151-1167.

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REFERENCES

[1]. Sebastian J Padayatty, et al. Vitamin C as an antioxidant: evaluation of its role in disease prevention. J Am Coll Nutr. 2003 Feb;22(1):18-35.

[2]. Michael T Nelson, et al. Molecular mechanisms of subtype-specific inhibition of neuronal T-type calcium channels by ascorbate. J Neurosci. 2007 Nov 14;27(46):12577-83.

[3]. Aleksander Hinek, et al. Sodium L-ascorbate enhances elastic fibers deposition by fibroblasts from normal and pathologic human skin. J Dermatol Sci. 2014 Sep;75(3):173-82.

[4]. Sungrae Cho, et al. Hormetic dose response to L-ascorbic acid as an anti-cancer drug in colorectal cancer cell lines according to SVCT-2 expression. Sci Rep. 2018 Jul 27;8(1):11372.

[5]. Satyanarayana Sreemantula, et al. Influence of antioxidant (L- ascorbic acid) on tolbutamide induced hypoglycaemia/antihyperglycaemia in normal and diabetic rats. BMC Endocr Disord. 2005 Mar 3;5(1):2.

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