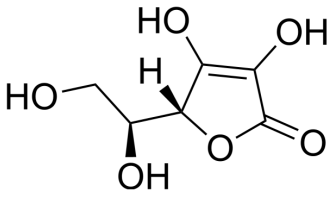
L-Ascorbic acid (GMP Like)

Cat. No.:	HY-B0166GL
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)



BIOLOGICAL ACTIVITY

Description	L-Ascorbic acid (GMP Like) is the GMP Like class L-Ascorbic acid (HY-B0166). L-Ascorbic acid (L-Ascorbate, Vitamin C), an electron donor, is an endogenous antioxidant agent. L-Ascorbic acid inhibits selectively $Ca_V3.2$ 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].
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]. L-Ascorbic acid (10 μg/ml, 5 days) enhances the reprogramming of mouse fibroblasts into IPSCs[5]. L-Ascorbic acid (50 μg/ml, 9 days) promotes fibroblasts conversion into cardiomyocytes[6]. L-Ascorbic acid (50 ng/ml, 4-6 days) facilitates generation of all-iPS cell mice from terminally differentiated B cells[7]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
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.

CUSTOMER VALIDATION

- 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.
- Biomed Pharmacother. September 2022, 113558.

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[2]. 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.

[3]. 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.

[4]. 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.

[5]. 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.

[6]. Esteban MA, Wang T, Qin B, et al. Vitamin C enhances the generation of mouse and human induced pluripotent stem cells. Cell Stem Cell. 2010;6(1):71-79. doi:10.1016/j.stem.2009.12.001

[7]. Talkhabi M, Pahlavan S, Aghdami N, Baharvand H. Ascorbic acid promotes the direct conversion of mouse fibroblasts into beating cardiomyocytes. Biochem Biophys Res Commun. 2015;463(4):699-705.

[8]. Stadtfeld M, Apostolou E, Ferrari F, et al. Ascorbic acid prevents loss of Dlk1-Dio3 imprinting and facilitates generation of all-iPS cell mice from terminally differentiated B cells. Nat Genet. 2012;44(4):398-S2.

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