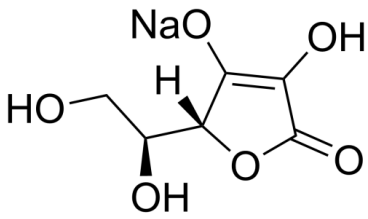
Data Sheet

Product Name:	L-Ascorbic acid (sodium salt)
Cat. No.:	CS-6063
CAS No.:	134-03-2
Molecular Formula:	$C_6H_7NaO_6$
Molecular Weight:	198.11
Target:	Apoptosis; Calcium Channel; Endogenous Metabolite; Reactive Oxygen Species
Pathway:	Apoptosis; Immunology/Inflammation; Membrane Transporter/Ion Channel; Metabolic Enzyme/Protease; Neuronal Signaling; NF-κB
Solubility:	H_2O : 100 mg/mL (ultrasonic);DMSO : 1 mg/mL (ultrasonic)



BIOLOGICAL ACTIVITY:

L-Ascorbic acid sodium salt (Sodium ascorbate), an electron donor, is an endogenous antioxidant agent. L-Ascorbic acid sodium salt selectively inhibits **$Ca_v3.2$ channels** with an **IC_{50}** of 6.5 μM. L-Ascorbic acid sodium salt is also a collagen deposition enhancer and an elastogenesis inhibitor[1][2][3]. ***In Vitro:***The conditioned medium for B16F10 cells significantly inhibits cell apoptosis induced by L-Ascorbic acid sodium salt (Sodium L-ascorbate) (10 mM), and the effective ingredients in the medium show a relative molecular mass below 5,000[4]. ***In Vivo:***Tg rats treated with L-Ascorbic acid sodium salt (Sodium L-ascorbate) show a higher incidence of carcinoma (29.6%), compared to those without L-Ascorbic acid sodium salt (15.4%). Independent of the L-Ascorbic acid sodium salt treatment, transgenic rats exhibit various kinds of malignant tumors in various organs[5].

After 12 weeks of PEITC-treatment, both simple hyperplasia and papillary or nodular (PN) hyperplasia have developed in all animals, but the majority of these lesions have disappeared at week 48, irrespective of the L-Ascorbic acid sodium salt-treatment. The same lesions after 24 weeks of PEITC-treatment have progressed to dysplasia and carcinoma, in a small number of cases by week 48, but enhancement by the L-Ascorbic acid sodium salt-treatment is evident only with simple hyperplasias and PN hyperplasias in rats[6].

PROTOCOL (Extracted from published papers and Only for reference)

Animal Administration:[3]A total of 40 7-week-old male Tg rats are divided into 2 groups. Twenty-seven (group 1) and 13 (group 2) rats are given a powdered MF diet with or without 5% sodium L-ascorbate, respectively. Similarly, a total of 42 7-week-old male Non-tg rats are divided into 2 groups, and 30 (group 3) and 12 (group 4) animals are given a diet with or without 5% sodium L-ascorbate, respectively.

References:

[1]. Hinek A, 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.

[2]. Yang X, et al. Mouse melanoma cell line B16F10-derived conditioned medium inhibits sodium L-ascorbate-induced B16F10 cell apoptosis. Nan Fang Yi Ke Da Xue Xue Bao. 2012 Feb;32(2):146-50.

[3]. Morimura K, et al. Lack of urinary bladder carcinogenicity of sodium L-ascorbate in human c-Ha-ras proto-oncogene transgenic rats. Toxicol Pathol. 2005;33(7):764-7.

[4]. Takagi H, et al. Limited tumor-initiating activity of phenylethyl isothiocyanate by promotion with sodium L-ascorbate in a rat two-stage urinary bladder carcinogenesis model. Cancer Lett. 2005 Mar 10;219(2):147-53.

[5]. 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.

[6]. 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.

CAIndexNames:

L-Ascorbic acid, sodium salt (1:1)

SMILES:

O[C@@H](CO)[C@]1([H])C(O[Na])=C(O)C(O1)=O

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