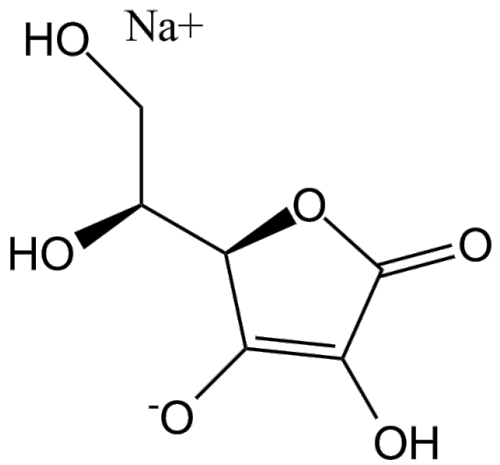
Product Data Sheet

Product Name: Sodium ascorbate
Cat. No.: GC13616

Chemical Properties

Cas. No.	134-03-2		
Chemical Name	sodium (R)-2-((S)-1,2-dihydroxyethyl)-4-hydroxy-5-oxo-2,5-dihydrofuran-3-olate		
SMILES	O=C1C(O)=C([O-])[C@@H]([C@@H](O)CO)O1.[Na+]		
Formula	$C_6H_7NaO_6$	M.Wt	198.11
Solubility	≥ 9.1mg/mL in Water	Storage	4°C, protect from light
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:

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Cell lines	Human neuroblastoma cell lines (HTLA-230, IMR-32, LAN-5, GI-LI-N, SH-SY5Y)
Preparation Method	Cells were maintained in Dulbecco's modified Eagle medium (DMEM) supplemented with 10% fetal bovine serum (FBS), 2mM L-glutamine, and antibiotics at 37°C, 5% CO_2. Cells were treated with Sodium Ascorbate at concentrations ranging from 0.5mM to 3mM for 6–24 hours.
Reaction Conditions	0.5–3mM; 6–24h
Applications	Sodium Ascorbate induced dose- and time-dependent apoptosis in all neuroblastoma cell lines, characterized by an increase in sub-G1 phase cells and caspase activation. Sodium Ascorbate significantly downregulated transferrin receptor (TfR/CD71) expression, leading to reduced intracellular iron levels. Treatment with Sodium Ascorbate also caused early loss of mitochondrial membrane potential (ΔΨm), and these effects were reversible by the iron donor ferric ammonium citrate (FAC), confirming iron depletion as the key mechanism of apoptosis.

Animal experiment [2]:

Animal models	C57BL/6 mice with collagen-induced arthritis (CIA) or lupus-like autoimmune disease (bm12-induced model)

Caution: Product has not been fully validated for medical applications. For research use only.
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Preparation Method	Mice were intraperitoneally injected with Sodium Ascorbate (5mg/mouse, dissolved in PBS) every other day for 7–21 days. For CIA, immunization with chicken type II collagen (CII) was performed 1 day after the first Sodium Ascorbate injection. For antibody response studies, mice were immunized with NP-Ficoll (T-independent) or NP-KLH (T-dependent) antigens.
Dosage form	5mg/mouse; i.p.; intermittent injection (every 48h).
Applications	Sodium Ascorbate significantly delayed arthritis onset and reduced disease severity in CIA mice, as evidenced by decreased clinical scores, synovial inflammation, cartilage/bone damage, and osteoclast activity. Sodium Ascorbate also suppressed autoantibody production (e.g., collagen-specific IgG and rheumatoid factor) in both arthritis and lupus models. Mechanistically, ascorbic acid inhibited B cell differentiation into plasma cells, disrupted germinal center formation, and impaired antibody affinity maturation by blocking STAT3 signaling and promoting B cell apoptosis.

References:
[1] Carosio R, Zuccari G, Orienti I, et al. Sodium ascorbate induces apoptosis in neuroblastoma cell lines by interfering with iron uptake. Mol Cancer. 2007 Aug 30;6:55.
[2] Yin Y, Wu S. Ascorbic acid alleviates rheumatoid arthritis by inhibiting the production of autoantibodies. Cell Commun Signal. 2024 Jul 24;22(1):373.

Background

Sodium ascorbate is an endogenous antioxidant that participates in hydroxylation reactions in the body, promotes the formation of intercellular substances, assists in corticosteroid synthesis, and influences blood cell maturation by promoting folic acid reduction and iron ion absorption. Sodium ascorbate also possesses antihistaminic effects and inhibits the formation of nitrosamines[1-2]. Sodium ascorbate can be used for the prevention and treatment of scurvy, infectious diseases, acute attacks of Keshan disease, liver diseases, as well as adjuvant therapy for various anemias, allergic

skin diseases, and cancer [3-4].

In vitro, Sodium ascorbate (10μM) treatment of B16 murine melanoma cells for 4 hours, Sodium ascorbate significantly induced apoptosis and downregulated transferrin receptor (TfR) expression, thereby reducing intracellular iron levels dependent on iron uptake[5]. Sodium ascorbate (0.5–3mM) treatment of human neuroblastoma cells (e.g., HTLA-230, SH-SY5Y) for 24 hours, Sodium ascorbate significantly induced caspase-dependent apoptosis, accompanied by loss of mitochondrial membrane potential, reduced cell proliferation, and decreased glycolytic capacity[6].

In vivo, Sodium ascorbate (60mg/kg) subcutaneously administered to tumor-bearing mice (P388 leukemia cells) (daily dosing starting from the first day after tumor implantation and continuing for 16 days), Sodium ascorbate significantly reduced tumor volume and weight, and improved mouse survival[7]. Sodium ascorbate (5mg/mouse) intraperitoneally administered to autoimmune arthritis mice (every other day for 21 days starting from the first day after immunization), Sodium ascorbate significantly delayed arthritis onset, reduced disease severity, and decreased autoantibody production[8].

References:
[1] Carr AC, Maggini S. Vitamin C and Immune Function. Nutrients. 2017 Nov 3;9(11):1211. doi: 10.3390/nu9111211.
[2] Padayatty SJ, Levine M. Vitamin C: the known and the unknown and Goldilocks. Oral Dis. 2016 Sep;22(6):463-93.
[3] Gęgotek A, Skrzydlewska E. Ascorbic acid as antioxidant. Vitam Horm. 2023;121:247-270.
[4] Moores J. Vitamin C: a wound healing perspective. Br J Community Nurs. 2013 Dec;Suppl:S6, S8-11.
[5] Kang JS, Cho D, Kim YI, et al. Sodium ascorbate (vitamin C) induces apoptosis in melanoma cells via the down-regulation of transferrin receptor dependent iron uptake. J Cell Physiol. 2005 Jul;204(1):192-7.
[6] Carosio R, Zuccari G, Orienti I, et al. Sodium ascorbate induces apoptosis in neuroblastoma cell lines by interfering with iron uptake. Mol Cancer. 2007 Aug 30;6:55.
[7] Osswald H, Herrmann R, Youssef M. The influence of sodium ascorbate, menadione sodium bisulfite or pyridoxal hydrochloride on the toxic and antineoplastic action of N-methylformamide in P 388 leukemia or M 5076 sarcoma in mice. Toxicology. 1987 Feb;43(2):183-91.
[8] Yin Y, Wu S. Ascorbic acid alleviates rheumatoid arthritis by inhibiting the production of

autoantibodies. Cell Commun Signal. 2024 Jul 24;22(1):373.