Sodium ascorbate

Catalog Number: T6674
CAS Number: 134-03-2
Molecular Formula: C6H8O6·Na
Molecular Weight: 201.13

**Description:** Sodium Ascorbate is a more bioavailable form of vitamin C that is an alternative to taking ascorbic acid as a supplement.

**Storage:** 2 years -80°C in solvent; 3 years -20°C powder;

**Solubility**

<table>
<thead>
<tr>
<th>Solvent</th>
<th>Solubility</th>
</tr>
</thead>
<tbody>
<tr>
<td>DMSO</td>
<td>9.9 mM</td>
</tr>
<tr>
<td>Water</td>
<td>198.9 mM</td>
</tr>
</tbody>
</table>

( < 1 mg/ml refers to the product slightly soluble or insoluble )

**Receptor (IC50)**

Others

**In vitro Activity**

Sodium ascorbate has a growth inhibiting action only at high concentrations in cultured human neoplastic cell lines MCF-7 (breast carcinoma), KB (oral epidermoid carcinoma), and AN3-CA (endometrial adenocarcinoma). Sodium ascorbate combined with vitamin K3 demonstrates a synergistic inhibition of cell growth at 10 to 50 times lower concentrations in cultured human neoplastic cell lines MCF-7, KB, and AN3-CA, at this level separately given vitamins are not toxic. This tumor cell growth inhibitory effect is completely suppressed by the addition of catalase to the culture medium containing vitamins C and K3, suggesting an excessive production of hydrogen peroxide as being implied in mechanisms responsible for the above-mentioned effects. [1] Sodium ascorbate combined with vitamin K3 results in a synergistic effect on growth inhibition in cultured human endometrial adenocarcinoma (AN3CA) cells. [2] Sodium ascorbate results in a rapid increase in the intracellular concentration of Ca2+ ions and subsequent apoptotic cell death in HL-60 cells, characterized by cell shrinkage, nuclear fragmentation and cleavage of internucleosomal DNA to yield fragments that are multiples of 180-200 base pairs, are induced. [3] Sodium ascorbate (100 μM) induces DNA single-strand breaks in human cells, Fibroblasts and Molt-4 cells are significantly more sensitive than lymphocytes. Sodium ascorbate (50 μM) results in significant cell loss in Molt-4 cells, but not in lymphocyte and fibroblast cultures. [4]

**In vivo Activity**

Tg rats treated with sodium L-ascorbate show a higher incidence of carcinoma (29.6%), compared to those without sodium L-ascorbate (15.4%). Independent of the sodium L-ascorbate 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 sodium L-ascorbate-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 sodium L-ascorbate-treatment is evident only with simple hyperplasias and PN hyperplasias in rats[6].

**Animal Experiment**

Animal Model: Tg rats

**Reference**


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