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The multidirectional impact of vitamin C on ageing processes and the health of the geriatric population - a literature review

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Abstract

Introduction

The geriatric population is prone to micronutrient deficiencies, including vitamin C. It is found in plant food, and deficiencies can be treated with over-the-counter supplements. This work aims to expand knowledge about the beneficial effects of vitamin C on diseases in the elderly.

Materials and Methods

A thorough analysis has been conducted following PRISMA recommendations. The research data have been derived from scientific databases of Embase, PubMed, Cochrane Library and Google Scholar. Key word choice was based on their relevance to the review subject beforehand.

The Stage of Knowledge

Modern science has confirmed that vitamin C is essential for health. Through advanced technology, we clearly understand how it supports collagen synthesis, reduces oxidative stress, and helps maintain strong organs and blood vessels. These properties are important for keeping organs functioning properly as the body ages.

However, modern medicine does not rely on biological theory alone. We now use Evidence-Based Medicine (EBM) to prove if a treatment works in real-world scenarios. Clinical trials are the most important tool we have because they separate theoretical benefits from proven results.

Summary

The wide range of effects of vitamin C, from supporting immunity and collagen synthesis to protecting the nervous system, is very important for maintaining the health of seniors. Thanks to its antioxidant properties, ascorbic acid delays the ageing process and supports cognitive function. Ensuring an adequate supply of this vitamin is an essential part of maintaining a good quality of life for the elderly. Regular intake of vitamin C should therefore be standard practice in the daily health care of seniors.

Key words: vitamin C, oxidative stress, cytokine production, ageing, geriatric population, collagen synthesis

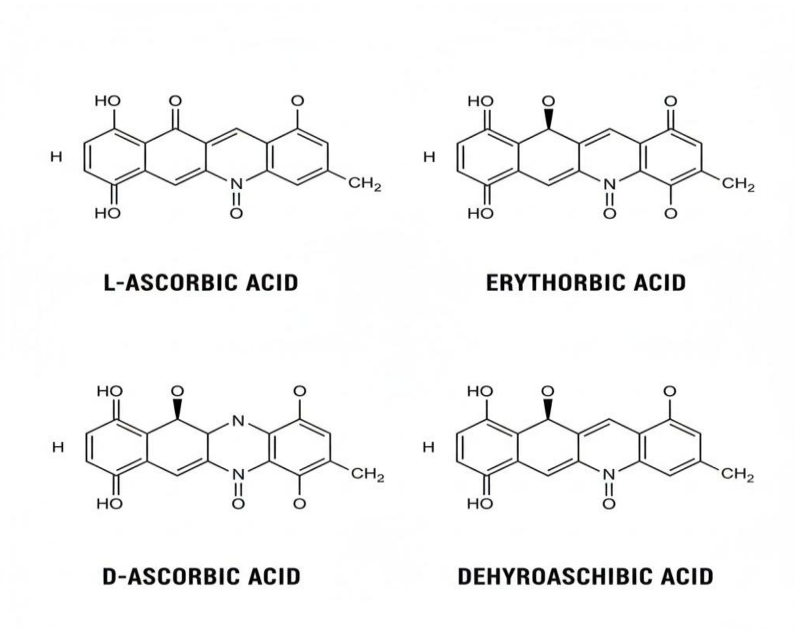
1. **Intruduction:**

According to the World Health Organisation (WHO), population ageing accelerated rapidly in the past few decades. The proportion of people over 60 years old is supposed to increase from 12% to 22% between 2015 and 2050. In 2020, the population over 60 had already outnumbered children under 5 years (1). Along with this comes a rise in the number of geriatric patients with sequelae of health problems ranging from comorbidity of old age diseases, through changes in physiology to certain behaviours, including varying dietary habits. Unfolding issues, taken holistically, require upholding the good health of the elderly as long as it is possible in each case. Prophylaxis, screening and patient education should come beforehand to upkeep the homeostasis.

As vitamin C plays role in above introduced subject, this review aims to inspect the impact of L-ascorbic acid on ageing and following changes in elderly population.

2.1 Vitamin C – Structure and Function within the Human Body

L-ascorbic acid (LAA) is a micronutrient widely known as vitamin C (VIC). It is a 2,3-enediol-L-gluconic acid-β-lactone ($C_6H_8O_6$) with two ionisable hydroxyl groups, which provide its acidic properties and high-water solubility. LAA molecule can donate electrons from enediol group carbons 2 and 3 (2,3). It mainly occurs as ascorbic acid (AA) or dehydroascorbic acid (DHA). LAA isomers: erythrobic acid and D-ascorbic acid are metabolised and eliminated comparably faster, although they can also act as active antioxidants (2,3).



As a reducing agent, VIC functions as a coenzyme, maintaining the reduced form of iron (Fe^{2+}) in hydroxylase enzymes. This role involves VIC in many biological processes, such as immune system activity, collagen biosynthesis, endothelium regulation, intestinal iron absorption, amine metabolism, recycling of α-tocopherol (vitamin E) and oxidative stress management (2,4).

Due to mutations in the L-gluconolactone oxidase gene, the human body is unable to synthesise VIC on its own and requires supplementation through the diet (2). Human cells acquire provided VIC as DHA through the glucose facilitative transporters (GLUT) or the sodium-dependent VIC transporters 1 & 2 (SVCT) (3). VIC's absorption is also possible through passive diffusion, especially at low pH, in non-ionized form (2). VIC distribution varies, achieving different levels in different organs, with the highest in brain and adrenal glands (10 mM) and lowest in muscle-type tissues (0,2 mM). This relationship stems from VIC's involvement in synthesis of hormones and neurotransmitters in respective tissues (2).

About 99% of AA is eliminated through kidneys with less than 1% leaving human organism through faeces. In urinary excretion 44% is eliminated as AA oxalate, 20 % as unmetabolized AA, other 20% as 2,3-diketo-1-gulonic acid and 2% as DHA. Renal VIC reabsorption takes place mainly through SVCT1, which allows to responsibly maintain plasma levels with saturable intestinal absorption (5).

According to current European Food Safety Authority (EFSA) Guidelines, the population reference intake (PRI) of VIC for women ≥ 18 years is 95 mg/day and 110 mg/day for men ≥ 18 years, respectively. Plasma VIC concentration of 50 µmol/L has been determined as adequate (6). However, as of now, France is the only European nation that has set a separate value of 120 mg/day for the population over 75 years. Findings from NHANES 2017-18 indicate that circulating VIC levels were significantly lower in the middle and older age groups (7).

Concentrations ≤23 µmol/L indicate hypovitaminosis C and concentrations ≤11 µmol/L signify deficiency occurring in development of scurvy, which is linked to deficient collagen synthesis (2,8). Scurvy sets off with fatigue and lethargy, the later shifts into connective tissue symptoms: gingivitis, teeth loss, petechiae, spontaneous, ecchymoses, haemorrhaging, anaemia, impaired wound healing, hyperkeratosis, myalgia, arthralgia, dyspnoea and weight loss (5). Nowadays, scurvy prevalence varies, ranging from 7,1% in the United States to occurrence of 73,9% in northern India (9).

In case of toxicity, VIC is well tolerated. Single oral dose of 5-10g alone yields in consequence diarrhoea, abdominal pain and bloating due to osmosis. However, administration with food decreases the adverse effects of the high dose. Chronic VIC overdosing leads to manageable polyuria and kidney stone formation, which may be much more dangerous in risk groups. Therefore, a threshold of 1 g/day oral intake should not be exceeded (5).

2.2 Free Radical Theory of Aging in terms of Vitamic C

Free Radical Theory of Aging (FRTA) is one of the nine suggested integrants of the ageing. It was first proposed in 1956 by Denham Harman as a cumulative damage on cellular level caused by free radicals, like Reactive Oxygen Species (ROS). In nowadays premise it is described as Mitochondrial Free Radical Theory of Aging (MFRTA) as its main objective is focused on mitochondrial DNA (mtDNA) damage due to oxidative stress through inducement of mutations and deletions within ontogenic mitochondrial genome (10).

Published research data have already unravelled that lower VIC levels are associated with ageing and diseases more prevalent in the elderly patients, e.g. chronic kidney disease, pulmonary obstructive disease, coronary disease, Alzheimer’s disease, osteoporosis, as well as respiratory infections (4,7). What enumerated cases have in common in their pathogenesis is the inflammation process linked to oxidative stress and the production of ROS (11). However, the role the of ROS and inflammation is required to adequate function of the immune system, control of the oxidative stress through proper diet and intake of antioxidant micronutrients like VIC should be viewed as beneficial in form of prophylaxis (4).

2.3 Vitamin C and immunity

With the onset of old age, which is conventionally classified as reaching the age of 65, there is a significant increase in the incidence of infectious diseases and their severe course (15, 16). The immune system undergoes profound negative changes as a result of the ageing process, which poses a challenge for modern medicine in terms of providing proper care for such patients (14).

Studies on vitamin C have revealed that it influences prostaglandin production, inhibits histamine activity and stabilises the activity of the enzyme 5-lipoxygenase. In addition, it stimulates interferon production and influences the activity of T and B lymphocytes (12,23) Several studies indicate that it modulates cytokine production, which also has an impact on better management of inflammation (12,18,23). Some of these are so-called pro-inflammatory cytokines, such as tumour necrosis factor TNFα, interleukin 1, 6, 12, 17 and 18, the secretion of which enhances local inflammation, increasing the sensitivity of immune cells to pathogens (15).

On the other hand, their excessively long action contributes to the development of chronic inflammatory diseases (15). The latter phenomenon often occurs in people geriatric diseases and is called inflammaging, which results, among other things, from the so-called training of the immune memory by ma The latter phenomenon often occurs in geriatric patients and is called inflammaging, which results, among other things, from the so-called training of the immune memory by macrophages, due to which the level of pro-inflammatory cytokines is constantly slightly elevated, and anti-inflammatory cytokines are reduced (15). In addition, vitamin C has been shown to have detoxifying properties, as it protects against the harmful effects of heavy metals and radiation (12). Therefore, it is recommended to consume fruits and vegetables such as peppers (12,13), which are rich sources of vitamin C. It is also useful in the prevention and treatment of sepsis, which is a major and frequent health risk in geriatric patients. Studies have shown that, due to its antioxidant properties, the administration of vitamin C to people with sepsis accelerates their recovery (12,14).

Numerous scientific reports indicate the validity of including vitamin C in the treatment protocol for lower respiratory tract infections. In the context of coronavirus infections, supplementation with this compound appears to be one of the most effective interventions supporting therapy. A study conducted in 2021/22 found that the average age of Covid infection is 63 and 66 for men and women, respectively, i.e. in older people. It is worth noting that at the age of 85 and above, the incidence rate reaches 50% (16). It has been shown that intravenous administration of vitamin C reduced mortality in acute respiratory distress syndrome (ARDS), which is often a complication of Covid (17,18).

It has been shown that patients receiving vitamin C therapy had a mortality rate half that of patients receiving only a placebo. Patients in the study group also had significantly better blood oxygenation. This suggests more effective oxygen diffusion, which indirectly indicates a reduction in lung tissue inflammation as a result of better regulation of inflammatory factors such as interleukins (12,18).

2.4 The importance of vitamin C in collagen synthesis

Mobility problems are common among older people. The results of a study conducted among patients over 60 years of age with multiple diseases receiving continuous and long-term care indicate a high level of mobility dependence. Only 13% of seniors declared their ability to change position in bed independently, while nearly 40% (38%) required full support from a carer in this regard. As older people are more likely to develop cancer, diabetes or obesity, the risk of bedsores increases significantly with the deterioration of mobility (22).

A good solution to counteract these negative effects is vitamin supplementation, as it supports the proper functioning of connective tissue, of which collagen is a component (12,20,21). This is because it participates in the enzymatic reaction that converts proline to hydroxyproline and lysine to hydroxylysine during the formation of collagen cross-links. Therefore, a number of studies and articles indicate that it has an impact on the proper regeneration of the skin and its resistance to mechanical damage (12,19,20,21). In vitro analyses using fibroblast cell lines have confirmed that ascorbic acid deficiency results in impaired collagen synthesis and maturation processes, in particular the formation of stable cross-links (19,21).

An appropriate dose of UV radiation stimulates the production of vitamin C, which accumulates in high concentrations in skin keratinocytes (20). In the face of excessive exposure to ultraviolet (UV) radiation, which is a key factor inducing photoaging, vitamin C exhibits strong anti-inflammatory properties. This mechanism is based on the effective neutralisation of reactive oxygen species, which limits the cascade of inflammatory processes within the skin tissue (20). It is worth noting that vitamin C is transported to chondrocytes by a sodium-dependent vitamin C transporter, where it also regulates the proper synthesis of type II collagen. For this reason, ascorbic acid is used in A nutritive mixture solution (NMS) to prevent and regenerate cartilage in joints already damaged as a result of osteoarthritis (24).

2.5 Revisiting the role of vitamin C in iron supplementation

Anaemia constitutes a critical clinical challenge in geriatric medicine, with epidemiological data indicating a prevalence of in approximately 11.0% in men and 10.2% in women aged 65 years and older (25). While iron deficiency anemia (IDA) remains the most prevalent form of anemia globally accounting for approximately 50% (26) of all clinical cases, its relative contribution in the geriatric population is lower, representing roughly one-third (33%) of all anaemia (25). The etiology of iron deficiency is inadequate exogenous intake due to dietary insufficiencies or reduction in absorbtion. A critical element in the differential diagnosis is the anaemia of chronic disease (ACD), in which iron homeostasis is fundamentally disrupted. In such cases, the pathophysiology shifts from a simple lack of iron to a complex state of functional deficiency, where systemic inflammation leads to the sequestration of iron within the reticuloendothelial system, rendering it unavailable for erythropoiesis despite sufficient total body stores (27).

There key pathophysiological mechanisms in iron deficiency anaemia is a result from impaired intestinal absorption due to structural changes and inflammation of the gastrointestinal mucosa, which is characteristic of conditions such as Crohn's disease, ulcerative colitis, and celiac disease. (26,28). Another significant factor is insufficient dietary intake, resulting from a nutritionally imbalanced diet that fails to meet essential micronutrient requirements.

The basic treatment paradigm in iron deficient anemia remains iron supplementation to stimulate erythropoiesis and normalize red blood cell parameters. For decades, clinical practice has been dominated by the hypothesis that ascorbic acid (vitamin C), by reducing iron to its divalent form ($Fe2+$) (29), significantly increases its bioavailability. This concept was reflected in one of the guidelines of The British Society of Gastroenterology from the last decade. They suggested a potential increase in iron bioavailability under the influence of ascorbic acid.

However, this paradigm has been revised thanks to high-quality scientific evidence. A large, prospective randomized clinical trial conducted by Nianyi Li and colleagues published in JAMA Network Open in 2020 (30), was of key importance. This study clearly showed that in patients with iron deficiency anaemia, oral iron supplementation with vitamin C does not show higher efficacy in terms of increasing hemoglobin or ferritin concentrations compared to iron monotherapy.

Consequently, in its updated 2021 recommendations, the British Society of Gastroenterology officially advises against routine co-supplementation with ascorbic acid, pointing to the lack of clinically significant benefits from such intervention.

2.6 The role of vitamin C in vascular homeostasis

In the geriatric population, increased vascular fragility and the resulting formation of petechiae or bruising from minor mechanical injuries often signal deeper structural degradation of the circulatory system. This frailty is frequently compounded by the development of atherosclerotic plaques and impaired tissue perfusion, significantly elevating the risk of cardiovascular diseases and limb pain in elderly patients.

The key importance of vitamin C for vascular homeostasis stems from its ability to increase the bioavailability of nitric oxide (NO) (31), the main vasodilator. Under physiological conditions, ascorbate acts as an antioxidant protector, neutralizing reactive oxygen species (ROS) and preventing the oxidative degradation of NO. Equally important is the role of vitamin C in stabilizing tetrahydrobiopterin (BH4) (32), an essential cofactor of endothelial nitric oxide synthase (eNOS). This prevents enzyme uncoupling, which, in conditions of deficiency, leads to the pathological reduction of NO. That is why clinical data confirm the high efficacy of co-supplementation of BH4, L-arginine, and vitamin C in acute peripheral ischemia. The synergistic action of these compounds induces rapid vasodilation, reduces inflammatory responses, and limits the area of reperfusion necrosis (33). This suggests that it is highly appropriate to include the supplementation protocol as an adjunctive therapy in the treatment of peripheral thromboembolic events in order to optimize tissue perfusion.

Another pillar of the protective action of L-ascorbic acid is its direct effect on the architecture of the extracellular matrix in which endothelial cells are embedded. As mentioned in an earlier chapter, vitamin C is essential for the hydroxylation of proline and lysine residues in collagen molecules, which determines the formation of stable cross-links. That is why geriatric patients, in whom natural collagen synthesis is impaired, supplementation or a diet rich in ascorbate becomes a key element in the prevention of vascular fragility (34).

The last but equally important aspect is the immunomodulatory and anti-apoptotic function of vitamin C within the vessel wall. Endothelium subjected to chronic oxidative stress caused, for example, by hyperglycemia or hyperlipidemia. This process initiates the adhesion of monocytes to the vessel wall, which is the first step in the pathogenesis of atherosclerotic plaque. Vitamin C, thanks to its antioxidant, anti-inflammatory, and cholesterol-lowering properties, is responsible for reducing the likelihood of cardiovascular events in patients with type 2 diabetes (35).

2.7 The role of vitamin C in the nervous system

Ascorbate is suggested as a neuromodulator of glutamatergic, dopaminergic, cholinergic and GABAergic transmission and the behaviors associated with them. Neurodegenerative diseases are usually related to high levels of oxidative stress, which is why ascorbic acid has been recognized as a substance with potential therapeutic effects in ischemic stroke, Alzheimer's disease, Parkinson's disease and Huntington's disease (36). Vitamin C is essential for the proper development and maintenance of the nervous system. The highest accumulation of vitamin C in the human brain occurs in the cerebral cortex, hippocampus, and amygdala. At the same time, higher concentrations of this compound are found in cerebrospinal fluid than in blood plasma (37). In nerve tissue, ascorbate acts as a cofactor for dopamine β-hydroxylase in the conversion of dopamine to norepinephrine (38). Ascorbic acid plays a fundamental role in the porcesses of myelination and remyelination of peripheral nerves, directly affecting of peripheral nerves, directly afeecting the functioning of Schwann cells. Its promyelinating effect is based on two mechanisms. Firstly, it promotes DNA demethylation, which activates the expression of genes responsible for myelin formation. Secondly, it stabilizes collagen triple helices, enabling the formation of the basement membrane, which is an essential foundation for the myelination process (39).

Vitamin C in Alzheimer's disease

The relation of vitamin C with Alzheimer's disease (AD) is equivocal (40). People with Alzheimer's disease have a significant decrease in vitamin C. A deficiency of this vitamin may contribute to the progression of the disease (41). Mounting evidence indicates a role for L-ascorbic acid in ameliorating specific factors linked to AD pathogenesis (42). Namely, the main mechanisms associated with AA neuroprotection involve the scavenging activity against ROS, the modulation of neuroinflammation, the suppression of the fibrillation of amyloid-beta peptide (Aβ), and the chelation of iron, copper and zinc (43). AA is an important antioxidant in the central nervous system; it is released from glial cells into the synaptic cleft, where it is then taken up by neurons for antioxidant protection, which is essential for maintaining their metabolism and proper synaptic function. The interaction between astrocytes and neurons has been found to serve as an essential AA recycling mechanism, contributing to the antioxidant protection of the brain (44).

3. Conclusions

The progressive aging of the population is one of the key challenges facing modern medicine and public health. Ascorbic acid affects many systems. The human body is unable to synthesize this vitamin, so adequate supplementation is necessary. In older age, serum concentrations of this component decrease. By reducing free radicals, vitamin C lowers the incidence of oxidative stress-related diseases, which are common in geriatric patients. It helps treat chronic conditions, but also life-threatening conditions such as sepsis and ARDS. As a key cofactor in collagen synthesis, it affects the condition of connective tissue, which translates into the regeneration of skin and joint cartilage. Vitamin C, as an important neuromodulator, plays a major role in the context of neurodegenerative diseases. This vitamin is an important element of prevention for geriatric patients, especially in cases of oxidative stress.

Disclosure:

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