Review article
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Genetic Regulation of Vitamin C Transport: The Role of SLC23A1 in Nutrient Metabolism, Antioxidant Defense, and Human Health

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Abstract
Vitamin C is an essential micronutrient responsible for antioxidant defense, collagen synthesis, and immune regulation. It enters cells through Sodium-Dependent Vitamin C Transporter 1 (SVCT1) and Sodium-Dependent Vitamin C Transporter 2 (SVCT2), which are produced from Solute Carrier Family 23 Member 1 (SLC23A1) and Solute Carrier Family 23 Member 2 (SLC23A2) genes. Among these, SLC23A1 plays a key role in the absorption and reabsorption of vitamin C in epithelial tissues. Genetic variations can affect vitamin C status, potentially contributing to chronic disease risk. SLC23A1 is closely linked to vitamin C homeostasis, as changes in its expression can affect the efficiency of vitamin C absorption and utilization. This review examines the molecular mechanisms of SLC23A1-mediated vitamin C transport, its role in nutrient metabolism, and the implications of genetic variation for human health, while providing critical insights into food and nutritional science based on SLC23A1 gene expression. SLC23A1 and vitamin C work together in health processes, antioxidant defenses, and metabolism to support the body's function. Understanding the regulation of SLC23A1 may improve nutritional approaches to modulate antioxidant levels and prevent chronic disease.

Keywords: Vitamin C, SLC23A1, Diet, Nutrigenomic, Antioxidant

Highlights
- Solute Carrier Family 23 Member 1 (SLC23A1) produces Sodium-Dependent Vitamin C Transporter 1 (SVCT1), a major transporter responsible for vitamin C uptake.
- Genetic variants in SLC23A1 affect vitamin C absorption and blood vitamin C levels.
- Vitamin C functions as an antioxidant and enzymatic cofactor in multiple metabolic pathways.
- Nutrigenomic interactions between diet and SLC23A1 may influence disease risk.

1. Introduction
Micronutrients play essential roles in human health, maintaining processes such as cellular metabolism and physiological homeostasis (Morris & Mohiuddin, 2023). Among these nutrients, Vitamin C (ascorbic acid) functions as an antioxidant and enzymatic cofactor in multiple biochemical processes, including collagen synthesis, neurotransmitter production, and immune regulation (Alberts et al., 2025). Despite the critical need for vitamin C, humans lack the enzyme required to synthesize it endogenously, so dietary intake is required to maintain adequate physiological levels (Logan et al., 2007).

To ensure proper transport of vitamin C, specialized membrane proteins produced by the Solute Carrier Family 23 Member 1 (SLC23A1) and Solute Carrier Family 23 Member 2 (SLC23A2) genes facilitate the transfer into cells. These proteins belong to the sodium-dependent vitamin C transporter family, which mediates active uptake of ascorbic acid across cellular membranes. SLC23A1 is primarily expressed in epithelial tissues such as the intestine and kidney, where it regulates vitamin C absorption and reabsorption, thereby maintaining systemic nutrient balance (Eck et al., 2013).

Genetic variation within SLC23A1 has been associated with differences in plasma vitamin C concentrations and may contribute to variability in antioxidant capacity among individuals (Timpson et al., 2010). Understanding how genetic factors regulate nutrient transport is a key area of research in nutrigenomics. In parallel, advancements in biotechnology have enabled increased vitamin C content in major crops. Combining these approaches highlights the importance of investigatin-g the bioavailability of vitamin C and its uptake mechanisms, which are under genetic control, when determining overall nutrient status (Carr & Rowe, 2020).

While vitamin C is known to regulate various physiological functions and systems, the necessity of its role may be overestimated in certain populations. In populations with nutrient-rich diets, vitamin C's benefits may be limited by saturation effects, variability in transporter-mediated uptake, and variation in genetic expression (Candeloro et al., 2026). This review examines the biological functions of SLC23A1 and explores its role in nutrient metabolism and human health.

1.1. Molecular Function of the SLC23A1 Transporter
The SLC23A1 gene produces a sodium-dependent transporter that facilitates active uptake of ascorbic acid across epithelial cell membranes. This transporter operates through a sodium gradient that drives vitamin C into cells against its concentration gradient (May, 2011). High expression of SLC23A1 in intestinal epithelial cells allows efficient dietary vitamin C absorption (Eck et al., 2013), while expression in renal tubular cells enables reabsorption of vitamin C from the

the filtrate, preventing nutrient loss through urine (Corpe et al., 2010).

1.2. Role in Nutrient Metabolism

Vitamin C is a cofactor for several enzymes involved in biosynthetic reactions. These include enzymes required for collagen maturation, catecholamine synthesis, and carnitine production (Abdullah et al., 2023). SLC23A1 regulates the cellular availability of vitamin C, thereby indirectly influencing these metabolic pathways (Table 1). Adequate transporter activity ensures that cells maintain sufficient intracellular vitamin C concentrations to support enzymatic function and antioxidant defense (Carr and Maggini, 2017).

1.3. Genetic Variation and Human Health

Variations, called single-nucleotide polymorphisms, within SLC23A1 have been associated with differences in circulating vitamin C levels (Duell et al., 2013). These genetic variations may alter transporter activity or expression levels, thereby affecting vitamin C uptake efficiency (Timpson et al., 2010) (Figure 1). Reduced transporter function may contribute to increased oxidative stress, which has been linked to chronic diseases such as cardiovascular disease and metabolic disorders (Shaghaghi et al., 2016).

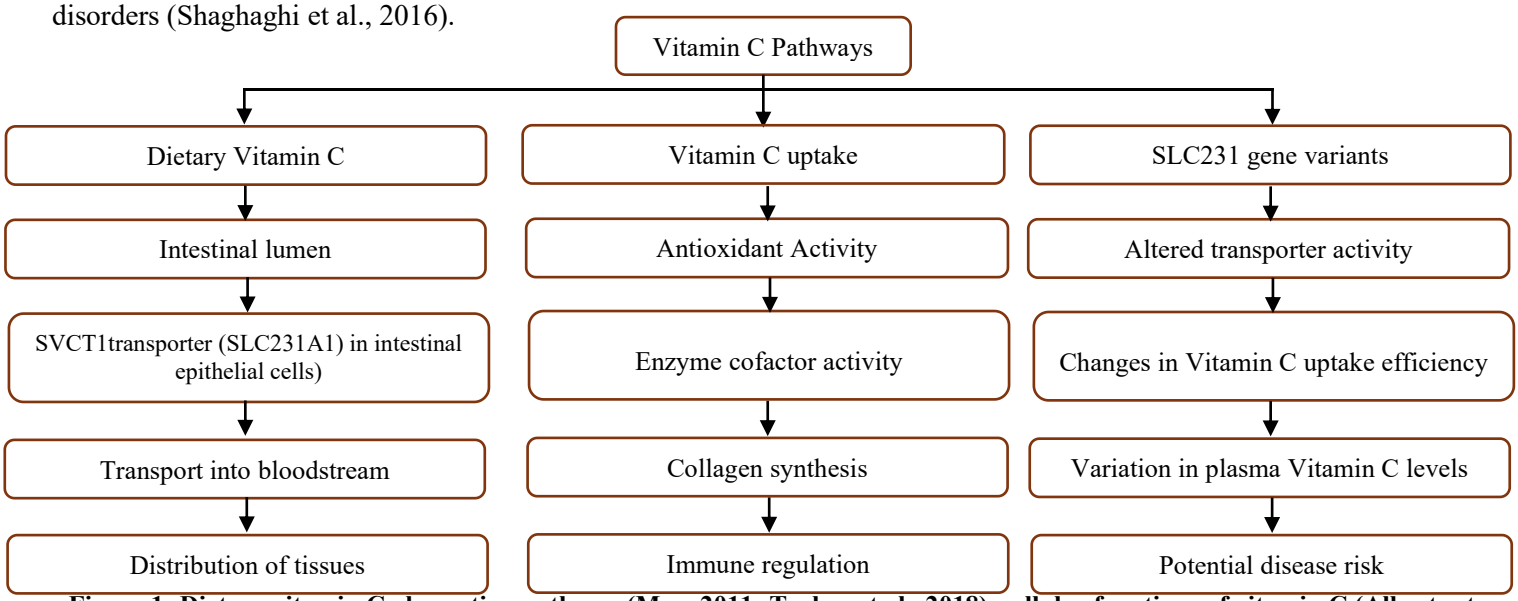

Figure 1: Dietary vitamin C absorption pathway (May, 2011; Traber et al., 2018), cellular functions of vitamin C (Alberts et al., 2025), and genetic regulation of vitamin C homeostasis (Michels et al., 2013).

Efficient transport of vitamin C into cells depends on the activity of the sodium-dependent transporter produced by the SLC23A1, which maintains intracellular ascorbate concentrations necessary for these biochemical reactions (Savini et al., 2008).

1.4. SLC23A1 Links Nutrient Intake & Cellular Metabolism

SLC23A1 controls how vitamin C is transported into the body, which affects antioxidant defense and many metabolic processes. Differences in the SLC23A1 gene can affect the efficiency with which vitamin C is absorbed and used (Shaghaghi et al., 2013) (Figure 1). This shows how nutrigenomics helps explain why people respond differently to the same nutrient intake. Although vitamin C deficiency is relatively rare in developed countries, suboptimal intake may still affect antioxidant capacity and immune function (Rowe and Carr, 2020). Genetic variation in SLC23A1 may further affect vitamin C levels in the body, suggesting that personalized nutritional recommendations may be beneficial in certain populations.

1.5. Nutrigenomic Interactions Between Diet and SLC23A1

Genetic differences in the SLC23A1 gene can affect the amount of Vitamin C in the blood (Figure 1). This shows how one's genome can influence the way nutrients are absorbed and utilized. Variations in this gene, called single-nucleotide polymorphisms, can change how well Vitamin C is transported in the body, affecting how much is absorbed in the intestines and reabsorbed by the kidneys (Corpe et al., 2010) (Figure 1). People with certain genetic variations may have lower Vitamin C levels in their blood, even if they eat the same amount of Vitamin C-rich foods (Michels et al., 2013). These results suggest that variations in the SLC23A1 gene could help explain why some people have different antioxidant levels and could lead to more tailored nutrition plans to improve Vitamin C intake.

1.6. Role of Vitamin C in Cellular Metabolism

Vitamin C is involved in hydroxylation reactions, including collagen synthesis, carnitine production, and catecholamine biosynthesis, all of which require sufficient intracellular vitamin C concentrations (Carr & Maggini, 2017) (Table 1).

Vitamin C is transported into cells primarily via the SLC23A2 transporter, whereas SLC23A1 plays a key role in intestinal absorption and renal reabsorption to maintain systemic vitamin C levels (May, 2012) (Figure 1). Disruptions or mutations in these transporters can indirectly impair these reactions (Yuan & Chen, 2025), affecting tissue health, energy metabolism, and cellular signaling.

Table 1: Major Biological Functions of Vitamin C in Human Metabolism.

Biological Role	Molecular Mechanism	Physiological Importance
Antioxidant defense	Scavenges reactive oxygen species and regenerates other antioxidants (Liu et al., 2023)	Protects cells from oxidative damage (Chandimali et al., 2025)
Collagen synthesis	Cofactor for prolyl and lysyl hydroxylase enzymes (Pinnell, 1985)	Maintains connective tissue, skin, and blood vessel health (Kanniyappan, 2025)
Neurotransmitter synthesis	Required for dopamine β-hydroxylase activity (Rahman et al., 2009)	Supports nervous system signaling (Kocot et al., 2017)
Carnitine biosynthesis	Cofactor in carnitine production pathways (Hao et al., 2025)	Supports fatty acid metabolism and energy production (Broderick et al., 2018)
Immune regulation	Supports leukocyte function and inflammatory responses (Moore and Khanna, 2023)	Enhances immune defense mechanisms (Moore and Khanna, 2023)
Catecholamine Biosynthesis	Produces dopamine, norepinephrine, and epinephrine (Khalil et al., 2024)	Supports the "flight-or-fight" response, mood, and cardiovascular function (Khalil et al., 2024)

1.7. Vitamin C Transport and Oxidative Stress

Oxidative stress occurs when there is an imbalance between reactive oxygen species (ROS) and the body's antioxidant defenses, leading to damage of cellular components such as DNA, proteins, and lipids. Reactive oxygen species, including hydrogen peroxide and hydroxyl radicals, can induce oxidative DNA damage through the formation of 8-oxo-deoxyguanosine (8-oxo-dG). 8-oxo-dG is a modified form of the DNA base guanine that is produced when ROS oxidize DNA (Lutsenko et al., 2002), as illustrated in Figure 2. This figure highlights the formation of 8-oxo-dG and how this modification can lead to incorrect base pairing and subsequent G to T mutations during DNA replication. It is one of the most common forms of oxidative DNA damage found in human cells and can be generated by both endogenous metabolic processes and exogenous factors such as radiation and chemical exposure (Hirano, 2011; Lutsenko et al., 2002). The modification occurs when guanine reacts with oxidative agents such as hydrogen peroxide and hydroxyl radicals, specifically because it is linked to mispair with adenine during DNA replication. This would lead to G to T mutations (Figure 2). Therefore, this type of DNA damage is commonly used as a marker of oxidative stress and genomic instability (Lutsenko et al., 2002). If not properly repaired, the accumulation of 8-oxo-dG can increase mutation frequency and contribute to disease development discussed earlier on in the paper (Hirano, 2011). Vitamin C acts as an antioxidant by neutralizing ROS and reducing oxidative damage in cells. Studies have shown that increased intracellular vitamin C levels reduce mutation frequency and decrease the formation of oxidative DNA damage markers such as 8-oxo-dG under oxidative stress conditions. Since vitamin C plays a key role in reducing oxidative stress, its availability in the body is important for limiting oxidative DNA damage (Lutsenko et al. 2002). The SLC23A1 transporter regulates vitamin C uptake, additionally the genetic variation in this gene can reduce intracellular vitamin C levels even when dietary intake is sufficient (Michels et al. 2013). This reduction in vitamin C availability can weaken antioxidant defense leading to an increase in the formation of oxidative DNA damage such as 8-oxo-dG (Lutsenko et al., 2002; Hirano, 2011). However, even though genetic variation in SLC23A1 has been shown to influence circulating vitamin C levels (Timpson et al., 2010; Michels et al., 2013), there is limited research directly linking these variations to markers of oxidative DNA damage such as 8-oxo-dG in human populations. Therefore, future studies are needed to better understand how SLC23A1 polymorphisms affect intracellular vitamin C availability and susceptibility to oxidative DNA damage.

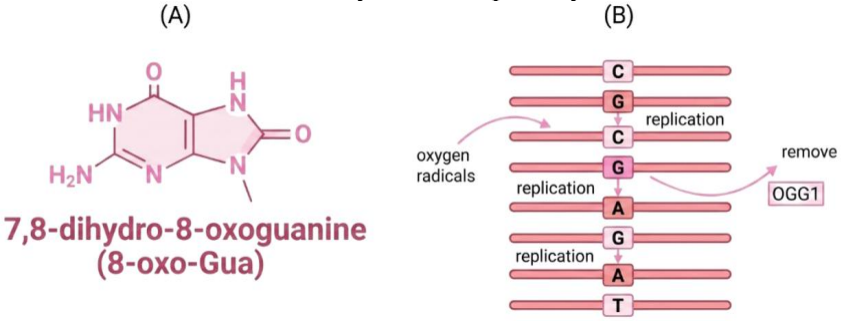

Figure 2: M-echanism of oxidative DNA damage caused by reactive oxygen species (ROS), showing the oxidation of guanine to 8-oxo-deoxyguanosine (8-oxo-dG) and resulting G to T mutations during DNA replication (Hirano, 2011).

Though studies support a relationship between vitamin C, oxidative stress, and DNA damage, there are some limitations to available evidence. Lutsenko et al. (2002) provides strong evidence that intracellular vitamin C can reduce mutation frequency and 8-oxo-dG formation; however, this study was carried out in cultured human cells under controlled conditions, which may not fully reflect what occurs in the human body. Similarly, Hirano (2011) supports the role of 8-oxo-dG as a marker of oxidative DNA damage, but much of the evidence is based on specific models of oxidative stress, which may limit its wider application. In addition, not all human studies show a clear protective effect of vitamin C. Wade et al. (2015) found that although genetic variation in SLC23A1 influences circulating vitamin C levels, it does not strongly affect health outcomes, suggesting that other factors may be involved. Therefore, there is a gap in research, as few studies directly link SLC23A1 variation, intracellular vitamin C levels, and oxidative DNA damage markers such as 8-oxo-dG in human populations.

1.8. Vitamin C Link to Increased Disease Risk

Since vitamin C plays a key role in reducing oxidative stress, its availability in the body is important for limiting oxidative DNA damage (Lutsenko et al., 2002). The SLC23A1 transporter regulates vitamin C absorption in the intestines and reabsorption in the kidney, helping maintain intracellular vitamin C levels (Eck et al., 2013; Corpe et al., 2010). Insufficient vitamin C can impair immune function and reduce the body's ability to respond to infections (Carr and Maggini, 2017) (Figure 1). Additionally, elevated oxidative stress resulting from low vitamin C levels has been linked to the development of chronic diseases such as cardiovascular disease and metabolic disorders (Traber et al., 2019). Studies showed that individuals with low vitamin C tend to have increased oxidative stress and reduced physical performance, which can be improved via vitamin supplements (Table 2) (Paschalis et al., 2014). These findings highlight the importance of maintaining vitamin C levels to support cellular function and reduce disease risk. Furthermore, genetic variation in the SLC23A1 transporter may influence vitamin C availability (Figure 1). This suggests that differences in nutrient absorption could contribute to variability in disease risk (Michels et al., 2013).

Table 2: Technological and Scientific Advancements for Vitamin C.

Category	Current Understanding	Examples	Future Directions
Nutritional Assessment	Measures vitamin C status (Reber et al., 2019)	Testing blood plasma (Jacob and Sotoudeh, 2002) and leukocytes (Bates, 1997; Jacob & Sotoudeh, 2002; Stephen & Utecht, 2001)	Creating wearable biosensors or mobile applications tracking micronutrients in real-time (Sempionatto et al., 2021) that also takes in account of genetics
Food Composition	Identifies natural vitamin C sources (Pehrsson and Haytowitz, 2015)	Mostly found in fruits and vegetables, decreases with heat and cooking, and is primarily present in oranges, guavas, and red and yellow peppers (Pehrsson and Haytowitz, 2015)	Increasing vitamin C content using gene editing techniques in crops (e.g., CRISPR) (Bu et al., 2025) and increased focus on preserving vitamin C via proper storage conditions (Ponder et al., 2022)
Diet and Health Relationship	Antioxidant, acts as a cofactor for biochemical processes (Li & Schellhorn, 2007), and supports immunity (Hu et al., 2020)	Reduces oxidative stress, builds collagen (Traber and Stevens, 2011)	Personalizing diets based on lifestyle and genetics
Functional Foods	Foods fortified with vitamin C (Hasler, 2002)	Fortified juices, cereals (Temple, 2022)	Creating vitamin C rich foods based on a surveyed population
Nutraceuticals	Supplements for vitamin C deficiency (Puri et al., 2022)	Tablets, powders, capsules (Hasler, 2002)	Liposomal nanomedicines (Izadiyan et al., 2025), nano-encapsulation, and other advanced delivery systems (Hao et al., 2026) for better absorption and targeted effects

1.9. SLC23A1 and Disease Risk

Variation in the SLC23A1 gene comes with risk of instability in vitamin C levels, inhibiting the flow of ascorbic acid into the blood (Timpson et al., 2010). This inhibition can lead to overall complications in health and increase of disease, studies linking the lack of vitamin C in blood with various aggressive forms of cancer and neurodegenerative disorders (Chen et al., 2021). A study by Chen et al. (2021) suggests that SLC23A1 specific vitamin C deficiency had a slight decrease in risk of Alzheimer's disease and cardioembolic stroke. An additional study focused on testing the association of vitamin C levels in plasma of patients with aggressive digestive system cancers (Table 2), the findings of which suggest that genetically associated vitamin C imbalance was not a direct cause of these cancers, however healthy levels of vitamin C could potentially prevent colorectal and small intestine cancers (Fu et al., 2021).

1.10. Vitamin C Status and Connections with Physiological Pathways and Morality Rates

While studies indicate there is no direct causation of physiological issues due to improper levels of vitamin C, there are indications of vitamin C imbalance causing underlying issues with processes involved with regulation of the body (Chandimali et al., 2025). A long-term study from Wang et al. (2018) observed levels of ascorbic acid (vitamin C) in the plasma of middle aged to elderly aged women, showing an overall decrease in mortality rate in association with high levels of ascorbic acid present. The study found that the highest levels of plasma ascorbic acid in the cohort of women had the lowest rates of mortality from cardiovascular disease, their risk lowered to 36% and overall mortality rate lowered to 23% (Wang et al., 2018) (Table 2). Antioxidants, such as vitamin C, are crucial in prevention of excess free radicals, an imbalance of which leads to oxidative stress (Chandimali et al., 2025). The repercussions of oxidative stress can lead to health issues including poor cardiovascular health, metabolic disorders like type 2 diabetes, inflammation, and severe cases such as ischemic stroke and various cancers (Tang et al., 2022).

1.11. Biotechnology for Major Vitamin C Crops

There are well-established methods of increasing vitamin C content in crops such as tomatoes (*Solanum Lycopersicon L.*), one of the most easily accessible sources of vitamin C for millions of people year-round. Medyouni et al. (2021) found that subjecting tomato plants to water stress via a 60% reduction in water, also called deficit irrigation, increased the vitamin C content by 20%. (Table 2). Additionally, Javeed et al. (2021) found that irrigation with diluted seawater increased vitamin C content significantly. These methods are not without their disadvantages as they decrease the overall fruit yield and can change the morphology of the fruit, which reduces the ability of the fruit to be sold, leading to significant financial losses for farmers. There are emerging techniques to optimize vitamin C content in tomatoes, such as foliar application of zinc nanoparticles. Application with 100 ppm zinc and zinc oxide nanoparticles to the leaves of the plant significantly increases the ascorbic acid content of tomatoes (Ahmed et al., 2023). Overexpression of GDP-L-galactose phosphorylase under the fruit-specific polygalacturonase promoter, which together effectively optimize the ascorbate biosynthesis pathway, is shown to induce a threefold increase in ascorbic acid content in ripe tomatoes. This was shown to occur through an upregulation of vitamin c associated recycling genes, biosynthetic genes, and transcription factors (Koukounaras et al., 2022). Another significant crop contributing to vitamin C intake is apples (*Malus domestica*) because they are often eaten raw, which bypasses the steps of cooking and processing that cause degradation of vitamin C (Fenech et al., 2006). Even modest increases in vitamin C content of apples would improve both the nutritional value and the shelf life (Mieszczakowska-Frąc et al., 2021). Application of calcium foliar fertilizer was shown to increase the vitamin C content by 32.39% when compared to a clear water control (Xu et al., 2025). The conditions the apples are stored in after harvesting have significant effects on vitamin C content, with freshly picked apples showing the highest content (Ponder et al., 2022). Temperature after harvesting is one of the most impactful variables on vitamin C degradation; while the content of vitamin C will continue to decrease over time, the rate of degradation is slowed if stored at cooler temperatures (Ferreira et al., 2024). This degradation process can be seen in the ratio of L-ascorbic acid (AsA) to L-dehydroascorbic acid (DHA), its oxidized counterpart (Figure 3).

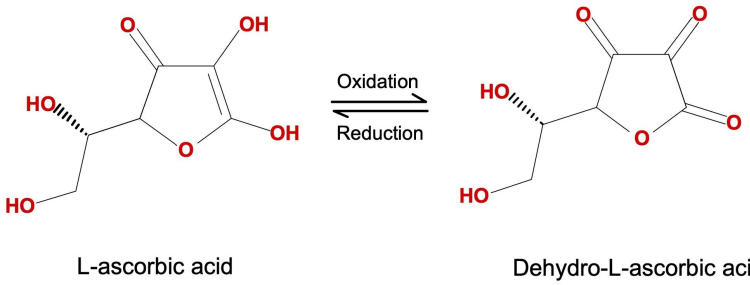

Figure 3: First step in the degradation pathway of vitamin C (Nemet & Monnier, 2011).

Before harvesting, the fruit of an apple has an AsA/DHA ratio shifted towards the reduced form and during storage the ratio shifts towards the oxidized form, which is an indication of oxidative stress (Lemmens et al., 2020). While additional methods for increasing vitamin C content exist, such as CRISPR-Cas9, these methods are better used when enhancing components of the crops with greater longevity than a rapidly degrading molecule such as ascorbic acid. For example, CRISPR-Cas9 has been used to increase the concentration of the inhibitory neurotransmitter gamma aminobutyric acid (GABA), in tomatoes (Nonaka et al., 2017). As a result, these GABA enhanced tomatoes are a putative functional food that may be used to combat lifestyle diseases such as hypertension (Minako et al., 2023). Regarding vitamin C, efforts are likely best focused on maintaining proper conditions in storage because despite potential increases in vitamin C through advancements in biotechnology, any improvement in vitamin C content is likely to be negated by its rapid degradation if

storage conditions are not properly maintained. When put into the context of SLC23A1, this becomes even more significant because of the inability for the Sodium-Dependent Vitamin C Transporter 1 intestinal transporter to uptake the oxidized form of ascorbic acid (Traber et al., 2018). The rapid degradation of vitamin C is a key characteristic of the molecule which introduces a type of bottleneck for vitamin C content in produce: no matter how much vitamin C is able to be packed into the fruit, significant amounts are likely to be unavailable to transporters such as SVCT1 by the time they are ingested. This should widen the scope of future research to focus not only on increasing vitamin C content but also on preserving it.

1.12. Nutritional Informatics & Artificial Intelligence for Vitamin C

Nutritional informatics, increasingly powered by artificial intelligence (AI), is changing how foods and diets are analyzed, manufactured, sustained, and personalized (Agrawal et al., 2025). AI-driven systems can identify patterns in vitamin C intake and account for individual genetic differences, particularly variations in SLC23A1, to personalize diets in mobile applications or biosensors (Table 2). If users provide consent for data collection, it can be used to create personalized nutraceuticals or smart foods with enhanced vitamin C content for specific populations. Many AI-driven innovations are increasingly using personal health data, such as blood markers, genetic information, and wearable biosensors, to create more personalized supplement recommendations (Agrawal et al., 2025; Pokushalov et al., 2024). These platforms identify deficiencies and generate targeted supplement recommendations, sometimes updating them over time. However, these claims often exceed the available evidence. Biomarker interpretation is complex, and there is limited proof that such AI-guided interventions consistently improve health outcomes (Pokushalov et al., 2024). Evidence suggests that variants in the SLC23A1 gene can influence blood vitamin C levels, although findings across studies are not always consistent, indicating that gene and diet interactions are still not fully understood. Some coding-region variants in SLC23A1 have been linked to reduced vitamin C transport activity, but less is known about the effects of non-coding variants, and the functional impact of many single nucleotide polymorphisms remains unclear. This limits how precisely SLC23A1 can currently be used as a target for tailoring vitamin C intake (Timpson et al., 2010; Michels et al., 2013). In addition, accuracy, transparency, and interpretablity in AI models (Cunningham et al., 2024; Pokushalov et al., 2024) are still major challenges. Many studies are based on limited datasets and controlled experimental settings, raising concerns about how well these results translate to real clinical environments (Twala, 2025). More large-scale and clinically validated research is needed before AI-based personalized nutrition and supplement systems can be confidently applied in practice.

1.13. Nanotechnology & Enhanced Vitamin C Delivery

Liposomal nanomedicines (Izadiyan et al., 2025), nano-encapsulation, and other advanced delivery systems for better absorption is a possible future direction for nutraceuticals (Hao et al., 2026) (Table 2). Applications from advancements in these technologies would be especially useful for vitamin C fortified shelf stable foods such as fruit juices and cereals which retain only 20-60% of vitamin C depending on storage conditions (Stešková et al., 2006). Implementation of nanoencapsulation by polymeric nanoparticles or nanoemulsions can increase the stability of L-ascorbic acid, shielding it from potential oxidants that would render it inert to SVCTs (Hao et al., 2026). Another aim of nanotechnologies in increasing vitamin C bioavailability is shielding the molecule from potential imbalances in the stomach that would lead to oxidation of L-ascorbic acid. In a healthy individual with an appropriate stomach pH, L-ascorbic acid would remain in the reduced state and eventually reach the intestine where transporters produced by SLC23A1 would uptake the molecule. However, in individuals with a low stomach pH, also called hypochlorhydria, vitamin C levels are significantly lower due to increased vitamin C oxidation (O'Connor et al., 1989). An increase in stomach pH can be caused by numerous different conditions such as duodenal ulcers, *H. pylori* infections, gastric cancers, chronic stress, and pernicious anemia (Sobala et al., 1989). Furthermore, an increase in stomach pH is the mechanism of action of many commonly used medications, such as proton pump inhibitors which treat gastrointestinal problems such as chronic heartburn and gastroesophageal reflux disorder (Herdiana, 2023). The increase in stomach pH caused by proton pump inhibitors is shown to decrease the amount of vitamin C in the stomach (Henry et al., 2005). By encapsulating vitamin C and allowing it to bypass unfavorable conditions in the stomach, even patients with severe dysregulation in stomach acid could feasibly absorb dietary vitamin C in its reduced state by SVCTs. Future research should focus on encapsulation of L-ascorbic acid to keep it in its reduced state for enhanced dietary absorption by SVCTs even in patients with gastrointestinal dysregulation.

1.14. Limitations & Future Direction

This review does not discuss nutrition informatics tools like existing mobile applications that track vitamin C, fortified foods or nutraceuticals on the market, and scurvy, which is one of the primary diseases caused by severe vitamin C deficiency (Gandhi et al., 2025). Future research can examine how the vitamin C transporter is regulated and expressed under different nutritional conditions to understand how the body adapts metabolically. Testing or examining literature on fortified foods, vitamin C deficiency in different populations, and supplements created using AI can be explored as well.

Conclusion

The SLC23A1 gene plays a central role in regulating vitamin C transport and maintaining intracellular antioxidant capacity. Through its involvement in intestinal absorption and renal reabsorption of vitamin C, SLC23A1 contributes to nutrient homeostasis and supports metabolic processes dependent on this essential micronutrient. Other factors, such dietary intake, supplementation, and bioavailability contribute to the individual's vitamin C status, which highlights the importance of integrating genetic variation into nutrition plans. Concurrently, advancements in agricultural biotechnology, such as bioengineering and gene editing to enhance vitamin C biosynthesis in crops, offer potential to increase dietary intake. However, the effectiveness of these approaches is limited by the degradation of vitamin C during storage and exposure to environmental conditions such as heat, light, and oxidation. There are some contradictions in the research, however, because of the drastic decrease in vitamin C that occurs with exposure to sun, increased temperatures, and oxidizing conditions over time. Future research in biotechnology advancements would be best focused on preserving existing vitamin C content in crops to allow for the greatest intake of L-ascorbic acid, the form of vitamin C which SVCT1 has an exceedingly high affinity for. Together, these developments highlight a comprehensive framework in which genetic factors and biotechnological innovations converge to improve nutritional status, optimize health outcomes, and reduce disease risk. Additionally, this is important in the context of reducing oxidative stress and limiting DNA damage, reinforcing the significance of vitamin C availability in maintaining genomic stability.

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Conflict of Interest

Authors declare no conflict of interest.

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