Review *Clin Ter 2023; 174 Suppl. 2 (6):173-182* *doi: 10.7417/CT.2023.2485*

Nutrigenomics: SNPs correlated to vitamins' deficiencies

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Abstract

Nutrients can influence the physiological processes in the body by interacting with molecular systems. Including nutrigenetics and nutrigenomics, nutritional genomics focuses on how bio-active food components interact with the genome. The purpose of this study is to clarify how nutrigenomics and vitamin dietary deficits relate to one another. Food tolerances among human sub-populations are known to vary due to genetic variation, which may also affect dietary needs. This raises the prospect of tailoring a person's nutritional intake for optimum health and illness prevention, based on their unique genome. To better understand the interplay between genes and nutrients and to plan tailored weight loss, nutrigenetic testing may soon become a key approach. ***Clin Ter 2023; 174 Suppl. 2 (6):173-182 doi: 10.7417/CT.2023.2485***

Key words: *Nutrients, SNPs, nutrigenomics, biomarkers, genes, vitamin A, vitamin B6, vitamin B9, vitamin B12, vitamin C, vitamin D, vitamin E, choline*

Introduction

Diet has significantly altered human metabolic capabilities throughout human development, which has facilitated the rise of contemporary disorders. From an evolutionary perspective, nutrition is a limiting element that puts selective strain on a population, just like other variables in the environment. Selection will be made against certain genotypes when nutritional needs in an individual are not met (1). A population's genotypes vary in their nutrient requirements. However, until these requirements—such as the need for additional calories from carbs and dietary fat—are addressed, the gene that transmits the high dietary requirement will persist in the population. This might apply to genes linked to diabetes and obesity (2).

In the developing discipline of nutrigenomics, cutting-edge genomics methods are used to examine how nutrients affect the genome and gene expression, as well as how genetic variations affect nutrient consumption. To characterize the relationship between nutrients and genes, the term "nutrigenomics" was coined. Physiology, biochemistry, metabolomics, proteomics, transcriptomics, and bioinformatics are all connected to genetics through nutrigenomics (3). Numerous genetic variations have been shown to affect the way proteins are built and function. The study of how dietary factors differently affect people, depending on their genetic makeup, is known as nutrigenetics, and it is a subfield of dietary genome research. Nutrigenomics investigates how dietary factors interact with the genome to regulate modifications to proteins and other metabolic functions (4).

A variety of dietary components serve as cofactors or substrates in metabolic pathways and are essential for DNA metabolism and repair, but there is much less information available about the effects of cofactor on the accuracy of DNA replication and repair. The genotype-dependent response to a particular nutrient must also be taken into consideration, even if certain nutrients can influence how a phenotype develops (5). The importance of genetic coding for assessing genome durability and related health impacts like cancer, degenerative diseases, and postnatal anomalies is widely acknowledged. It is clear that complicated chronic illnesses can emerge due to both ecological and hereditary factors (6). The "fetal basis of adult disease" or "early origins hypothesis" postulates that nutrition and other environmental factors during pregnancy and the early postnatal period have an impact on gene activity and cellular flexibility, which can alter vulnerability to mature illnesses (7).

Vitamins, their role, and effect of vitamin deficiency

Vitamins are necessary chemical substances for the human body to ensure appropriate physiological function and overall health. They operate as coenzymes or as precursors for enzymes that assist reactions within the body, playing important roles in a variety of metabolic processes (8). Thiamine is necessary for the metabolism of food into energy, optimal neuronal function, and preservation of the cardiovascular system. The immune system, the creation of neurotransmitters, and the metabolism of proteins all require

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ISSN 1972-6007

pyridoxine (9). B12 is essential for synthesizing DNA, producing red blood cells, and maintaining healthy nerves: its deficiency might lead to pernicious anemia, nerve damage, and cognitive impairment. The immune system, eyesight, and cell differentiation all depend on vitamin A; among the symptoms of its deficiency are night blindness, dry skin, and an increased risk of infections (10). Antioxidants like vitamin E shield cells from harm, and its absence, although uncommon, can cause neurological problems and muscle weakness. Bone health and blood clotting depend on vitamin K, whose deficiency can decrease blood coagulation and thus raise the risk of bleeding. The immune system, bone health, and calcium absorption all depend on vitamin D. Vitamin deficiency can be avoided by eating a balanced diet that is high in fruits, vegetables, whole grains, and a variety of foods, while malnutrition, restrictive diets, digestive issues, and a few medical diseases can all raise the risk of deficiency, though. Consult a healthcare provider as soon as you suspect a deficiency for accurate diagnosis and effective therapy (11).

Nutrigenetics

Nutrigenetics studies the impact of genetic diversity on nutrient responses and function. Although they are closely linked, it is not the same thing as nutrigenomics (5). Numerous chronic diseases can be predicted using nutrigenetic research, and these conditions may be avoided or treated more effectively by using individualized nutritional management. The majority of nutrigenetic experiments investigate how various polymorphisms influence alterations in eating patterns (5).

Nutrigenomics

The goal of the emerging area of nutrigenomics, which makes use of the most cutting-edge genomics technology available, is to investigate how vitamins affect the genome and gene expression, as well as how genetic variations affect nutrient consumption (12). The area of nutritional research known as "nutrigenomics" focuses on using molecular methods to examine, evaluate, and comprehend the physiological responses of certain populations or individuals to different diets (2). These gene-nutrient interactions depend on the ability of specific nutrients to interact with transcription factors, which in turn regulates RNA polymerase's attraction to gene promoter areas and the quantity of transcripts produced (3).

Single nucleotide polymorphisms (SNPs)

The most prevalent sort of variation in human DNA is single nucleotide polymorphisms (SNPs). In the last two decades, nutrigenomics studies identified phenotypes of SNPs between healthy and micronutrient-deficient populations (13). This correlations usually refer only to nutritional deficiencies. Thus, more research and human studies are needed to determine what function, if any, these SNPs have in developing specific nutrient deficits or other physiological responses (14).

Choline

An important vitamin, called choline, is crucial for several bodily physiological processes. Many foods, both from animal and plant sources, contain choline, but among its main sources are egg yolk, chicken, fish, and dairy products (15). In comparison to other vitamin deficiencies, choline insufficiency is less researched and understood. For people who struggle to get enough choline from their food alone or have particular health concerns that call for higher choline consumption, dietary supplements can be used to have an extra source of choline (16). However, it's crucial to remember that the majority of people can satisfy their choline requirements by eating a well-balanced diet. The undesirable consequences of excessive choline intake may include a fishy body odor or digestive problems (17). (Table 1)

The phosphatidylethanolamine N-methyl transferase (PEMT) gene's particular genetic variations, or single nucleotide polymorphisms (SNPs), are described in Table 1, which includes information on two SNPs (rs12325817 and rs7946) as well as the associated genes, polymorphism functions, and alleles (23). The methylation pathway, which is essential for

Table 1. SNPs related to choline and their effects.

Choline					
RsID	**Gene**	**Polymorphism function**	**Alleles**	**wt/mt**	**Reference**
rs12325817	PEMT	Increased risk of organ dysfunction with low choline diet; lower betaine levels with inadequate choline intake	G/G	mt/mt	(18-20)
		Increased risk of organ dysfunction with low choline diet	C/G	wt/mt	
		Typical	C/C	wt/wt	
rs7946	PEMT	Decreased enzyme activity	T/T	mt/mt	(21-23)
		Somewhat decreased enzyme activity	C/T	wt/mt	
		Typical	C/C	wt/wt	

the manufacture of phosphatidylcholine, is catalyzed by the PEMT gene. The structure of cell membranes is crucially maintained by phosphatidylcholine. People who consume a choline-deficient diet are at an increased risk of developing organ malfunction due to the specific polymorphism indicated by the rs12325817 SNP (24).

Vitamin B6

One of the B-complex vitamins, vitamin B6, often referred to as pyridoxine, is essential for several bodily metabolic activities. As a coenzyme, it speeds up numerous enzymatic processes that are necessary for the metabolism of proteins and the creation of neurotransmitters like serotonin and dopamine. Many foods, both plant-based and animal-based, contain vitamin B6 (25). The finest food sources of vitamin B6 include vegetables, whole grains, legumes, chicken, fish, organ meats, nuts, seeds, and legume-based products. A lack of vitamin B6 can cause neurological issues, skin disorders, irritability and sadness, anemia, weakness, and exhaustion (26). There are vitamin B6 supplements available for people who might have trouble getting enough of it through diet alone, or have particular medical conditions that call for higher B6 intake. The most common forms of these supplements are pyridoxine hydrochloride and pyridoxal-5'-phosphate (27).

SNPs that affect how vitamin B6 is metabolized are included in Table 2, which contains information on two SNPs (rs4654748 and rs5742905) as well as the genes, gene functions, polymorphism functions, and alleles that are connected to them (31). Alkaline phosphatase, an enzyme involved in the metabolism of several phosphate compounds, is represented by the gene ALPL in Table 2. It is essential for adaptive thermogenesis and skeletal mineralization. Lower levels of vitamin B6 are linked to the particular polymorphism indicated by rs4654748 (32). The enzyme known as cystathionine beta-synthase, which is involved in the metabolism and detoxifying processes of cysteine, is encoded for by CBS. An increased risk of excessive homocysteine levels is linked to the particular polymorphism indicated by rs5742905; however, taking extra vitamin B6 can lessen or mitigate this risk.

Vitamin B9

The vital water-soluble vitamin B9, sometimes referred to as folate or folic acid, is involved in a number of key bodily processes. Its main function is to function as a coenzyme in DNA synthesis, cell division, and red blood cell production. Numerous foods naturally contain folate, and many nations also fortify some meals with folic acid to help prevent deficiencies (33). Leafy green vegetables, legumes, citrus fruits, avocados, fortified grains, and liver are a few examples of foods high in folate (34). Due to its critical function in cell division and DNA synthesis, vitamin B9 insufficiency, also known as folate deficiency, can cause a number of health issues. Anemia, neural tube abnormalities, digestive problems, and mood disorders are typical signs of folate insufficiency. Supplements containing folic acid are frequently used to prevent and treat folate deficiency, particularly in pregnant women and others who may have problems getting enough folate through their diet. Prenatal vitamins and over-the-counter folic acid supplements are both readily available (35).

Table 3 offers details on a particular genetic variant (SNP) connected to folate (vitamin B9) metabolism; it also contains information about the MTHFR gene, the SNP rs1801133, the gene's related polymorphism, and the rele-

Table 2. SNPs related to Vitamin B6 and their effects.

Vitamin B6					
RsID	**Gene**	**Polymorphism function**	**Alleles**	**wt/mt**	**Reference**
rs4654748	ALPL	Lower vitamin B6 concentrations	C/C	mt/mt	(28)
		Slightly lower vitamin B6	C/T	wt/mt	
		Typical	T/T	wt/wt	
rs5742905	CBS	Risk of increased homocysteine, responsive to vitamin B6	G/G	mt/mt	(29, 30)
		Risk of increased homocysteine, responsive to vitamin B6	A/G	wt/mt	
		Typical	A/A	wt/wt	

Table 3. SNPs related to Vitamin B9 and their effects.

Vitamin B9					
RsID	**Gene**	**Polymorphism function**	**Alleles**	**wt/mt**	**Reference**
rs1801133	MTHFR	Enzyme function decreased by 70-80%	A/A	mt/mt	(36)
		Enzyme function decreased by 40%	A/G	wt/mt	
		Typical	G/G	wt/wt	

vant alleles. the MTHFR gene codes for the enzyme called methylenetetrahydrofolate reductase (37). It is important to note that the decreased enzyme activity brought on by this polymorphism might result in higher homocysteine levels and may be linked to a number of health issues, including cardiovascular disease and neural tube abnormalities (38).

Vitamin B12

Cobalamin, generally known as vitamin B12, is a water-soluble vitamin that is essential for several bodily physiological processes. It plays a major role in DNA synthesis, neuron function, and red blood cells' production. Foods derived from animals—such as meat, fish, shellfish, dairy products, and eggs—naturally contain vitamin B12 (39). A lack of this vitamin can cause a variety of health issues, with mild to severe symptoms. Common signs include weakness and exhaustion, anemia, difficulty walking, painful lips, memory loss and cognitive impairments, pale complexion, and stomach issues (40). Vitamin B12 deficiency can be treated or prevented with the help of dietary supplements, especially in people who may have trouble absorbing the vitamin through food sources. There are several ways to get vitamin B12 supplements, including oral tablets, lozenges, injections, and sublingual.

The genetic variations (SNPs) that affect how vitamin B12 is metabolized are listed in **Table 4**, which contains information on the SNPs rs602662, rs492602, rs1801222, and rs162036, as well as information on the genes, polymorphisms, and linked alleles that each SNP is associated with. The gene FUT2 codes for an enzyme that alters the glycan chains of glycolipids and glycoproteins present on cell surfaces. The specific polymorphism rs602662 is only related to the risk of low serum vitamin B12 levels when the diet is insufficient in bioavailable sources of vitamin B12. The particular polymorphism represented by rs492602, namely the G allele, is associated with lower levels of vitamin B12 in the body. The gene CUBN encodes for an endocytic receptor, specifically one that facilitates vitamin absorption. The G allele of the specific polymorphism denoted by rs1801222 is associated with elevated B12 levels in the body. MTRR regulates insulin secretion and has a relationship to ion channel genes. The amount of vitamin B12 that is absorbed or used by the body may vary depending on these genetic differences. Certain genotypes may increase the risk of low levels of vitamin B12 or alter the vitamin's blood level (45).

Vitamin E

The body needs vitamin E, a fat-soluble antioxidant, to function properly. Its main function is to shield cells from free radical damage. Free radicals are extremely reactive chemicals that can destroy cells and cause a number of health problems. Many foods contain vitamin E, although plant-based foods tend to be the best sources. Fortified cereals, avocado, kiwifruit, broccoli, and fortified cereals are some of the top food sources of vitamin E (46). Muscle weakness, nerve damage, eye issues, trouble walking and coordinating motions, and anemia are all signs of vitamin E insufficiency. Getting enough vitamin E from dietary sources should be sufficient for most healthy people who follow a balanced diet. There are several types of vitamin E supplements, including capsules, soft gels, and oils (47).

Table 4. SNPs related to vitamin B12 and their effects

Vitamin B12					
RsID	**Gene**	**Polymorphism function**	**Alleles**	**wt/mt**	**Reference**
rs602662	FUT2	Greatest risk for low serum vitamin B12 levels, but only when the diet is low in bioavailable sources of vitamin B12	G/G	mt/mt	(41)
		Greater risk for low serum vitamin B12 levels, but only when the diet is low in bioavailable sources of vitamin B12	G/A	wt/mt	
		Typical	A/A	wt/wt	
rs492602	FUT2	Lower vitamin B12 levels	G/G	mt/mt	(42)
		Lower vitamin B12 levels	G/A	wt/mt	
		Typical	A/A	wt/wt	
rs1801222	CUBN	Lower vitamin B12 concentrations	A/A	mt/mt	(43,44)
		Somewhat lower vitamin B12 concentrations	A/G	wt/mt	
		Typical	G/G	wt/wt	
rs162036	MTRR	Decrease in enzyme activity with potential negative impact on vitamin B12 concentration	G/G	mt/mt	(43,44)
		Partial decrease in enzyme activity with potential negative impact on vitamin B12 concentration	A/G	wt/mt	
		Typical	A/A	wt/wt	

Table 5. SNPs related to vitamin E and their effects.

Vitamin E					
RsID	**Gene**	**Polymorphism function**	**Alleles**	**wt/mt**	**Reference**
rs11057830	SCARB1	Lower plasma vitamin E concentration	A/A	mt/mt	(47, 48)
		Somewhat lower plasma vitamin E concentration	G/A	wt/mt	
		Typical	G/G	wt/wt	
rs1527479	CD36	Lower plasma vitamin E concentration	A/A	mt/mt	(49)
		Somewhat lower plasma vitamin E concentration	G/A	wt/mt	
		Typical	G/G	wt/wt	
rs2108622	CYP4F2	Lower plasma vitamin E concentration	T/T	mt/mt	(49,50)
		Somewhat lower plasma vitamin E concentration	C/T	wt/mt	
		Typical	C/C	wt/wt	

The information about three genetic variations (rsID) and their relation to plasma vitamin E content is presented in Table 5. Each variant has a unique allelic combination and is linked to a particular gene. It serves as a high-density lipoprotein receptor. Lower levels of plasma vitamin E are linked to the A allele. This genotype results in reduced plasma vitamin E concentrations in the individuals. It makes it easier for fatty acids to pass through cell membranes. Lower levels of plasma vitamin E are linked to the A allele. This genotype results in reduced plasma vitamin E concentrations in the individuals. When compared to genotypes with normal plasma vitamin E content, individuals presenting this genotype had lower levels of vitamin E. It is involved in the metabolism of xenobiotics (foreign chemicals) and fatty acids. Variations in the amount of vitamin E in the blood are linked to the T allele. Individuals with this genotype have varying blood levels of vitamin E. The amounts of vitamin E in the blood may vary depending on genotype, with some alleles being linked to higher or lower levels (51).

Vitamin A

A vital component of sustaining vision, bolstering the immune system, and encouraging healthy skin and mucous membranes is the fat-soluble vitamin known as vitamin A. Both animal-based foods—such as liver and organ meats, fish, eggs, dairy products—and plant-based ones—like carrots, sweet potatoes, mangoes, and apricots—contain preformed vitamin A. Night blindness is among the signs of vitamin A insufficiency, along with dry skin, increased susceptibility to infections, and wound healing problems (52). Vitamin A supplements can be helpful for people who have trouble getting enough vitamin A through their meals or who are at risk of insufficiency. Several types of vitamin A supplements are available, including retinyl palmitate and beta-carotene (53).

Table 6 gives details on three genetic variations (rsID) and how they affect how beta-carotene is metabolized and transformed into vitamin A. Each variant has a unique allelic combination and is linked to a particular gene. Encodes a significant enzyme, necessary for the conversion of beta-carotene to vitamin A. In order to create two retinal molecules, it catalyzes the oxidative cleavage of beta-carotene. The G allele is linked to decreased beta-carotene conversion, which raises the amounts of beta-carotene in the blood. This genotype results in decreased beta-carotene conversion, which raises the amounts of beta-carotene in the blood. Individuals with this genotype and those with a normal genotype have somewhat decreased beta-carotene conversion. This gene encodes for a crucial enzyme, necessary for the conversion of beta-carotene to vitamin A. The T allele is linked to lower beta-carotene conversion and a higher risk of developing atherosclerosis when eating poorly. The metabolism of lycopene may also be impacted This gene encodes for a crucial enzyme, necessary for the conversion of beta-carotene to vitamin A. The T allele is linked to lower levels of lutein (another carotenoid) and impaired beta-carotene conversion. The efficiency of beta-carotene conversion may vary depending on genotype, which could alter levels of circulating beta-carotene and possibly other carotenoid levels, like lutein and lycopene (56).

Vitamin D

A vital nutrient, vitamin D performs a number of vital functions in the body. One of its main tasks is helping to control the absorption of calcium and phosphorus, which are essential for keeping strong bones and teeth. Fatty fish, cod liver oil, egg yolks, fortified dairy products or plant-based milk alternatives, and fortified morning cereals are a few food sources of vitamin D. Bone discomfort, soft bones, an increased risk of fractures, delayed wound healing, and diminished immunological function are some frequent signs of vitamin D deficiency [32]. If dietary consumption and sun exposure are insufficient to maintain optimal vitamin D levels, it may be advised to take dietary supplements. Supplemental vitamin D is frequently used to treat or prevent vitamin D insufficiency. They are frequently prescribed to people who don't get much sun exposure, like those who live in northern latitudes, elderly people who spend less time outside, people with darker skin, and people with illnesses that make it difficult for the body to absorb fat (57,58).

Table 6. SNPs related to vitamin A and their effects.

Vitamin A					
RsID	**Gene**	**Polymorphism function**	**Alleles**	**wt/mt**	**Reference**
rs6564851	BCO1	Decreased beta-carotene conversion	G/G	mt/mt	(54)
		Decreased beta-carotene conversion	G/T	wt/mt	
		Typical	T/T	wt/wt	
rs12934922	BCO1	Decreased beta-carotene conversion; may affect lycopene also	T/T	mt/mt	(1,55)
		Decreased beta-carotene conversion	A/T	wt/mt	
		Typical	A/A	wt/wt	
rs7501331	BCO1	Decreased beta-carotene conversion; lower lutein levels; may affect lycopene	T/T	mt/mt	(1,56)
		Decreased beta-carotene conversion	C/T	wt/mt	
		Typical	C/C	wt/wt	

Table 7. SNPs related to vitamin D and their effects.

Vitamin D					
RsID	**Gene**	**Polymorphism function**	**Alleles**	**wt/mt**	**Reference**
rs4588	GC	Lower 25-hydroxyvitamin D (main circulating form) levels	A/A	mt/mt	(59, 60)
		Somewhat lower 25-hydroxyvitamin D (main circulating form) levels	A/C	wt/mt	
		Typical	C/C	wt/wt	
rs2282679	GC	Decreased vitamin D levels	G/G	mt/mt	(61,62)
		Somewhat decreased vitamin D levels	G/T	wt/mt	
		Typical	T/T	wt/wt	
rs7041	GC	Decreased vitamin D levels	A/A	mt/mt	(63)
		Decreased vitamin D levels	A/C	wt/mt	
		Typical	C/C	wt/wt	
rs12794714	CYP2R1	Lower vitamin D levels	A/A	mt/mt	(64)
		Somewhat lower vitamin D levels	A/G	wt/mt	
		Typical	G/G	wt/wt	
rs10741657	CYP2R1	Possible vitamin D insufficiency or deficiency	G/G	mt/mt	(65)
		Possible vitamin D insufficiency or deficiency	A/G	wt/mt	
		Typical	A/A	wt/wt	
rs2228570	VDR	Carrier of Fok1 variants; possibly decreased vitamin D levels	G/G	mt/mt	(66, 67, 68)
		Typical	A/G	wt/mt	
		Typical	G/G	wt/wt	

The information regarding various genetic variations (rsID) and their relationship to vitamin D levels is included in Table 7. Each variant has a unique allelic combination and is linked to a particular gene. Target tissues receive the vitamin D and its plasma metabolites once it binds to them. Lower 25(OH) D levels, which are a measure of vitamin D status, are linked to the A allele. Therefore, those who have this genotype have lower levels of 25(OH) D. In other words, those with the A/A genotype for the rs4588 variant of the GC gene have lower levels of 25(OH) D, a measure of their vitamin D status. This shows that this genetic variation may affect vitamin D transport or metabolism, resulting in reduced amounts of the active form of vitamin D in the blood (66).

Variant rs2282679; Gene: GC

Target tissues receive vitamin D and its plasma metabolites once it binds to them. Serum vitamin D levels are known to be lower in people with the G allele genotype. The levels of total serum vitamin D are a little bit lower in all of the people with this genotype. As a result, those with the G/G genotype for the rs2282679 variant of the GC gene have decreased serum vitamin D levels. Similar to this, people with the G/T genotype have somewhat lower amounts of total serum vitamin D. This implies that this genetic variation may impact vitamin D binding or transport, resulting in lower levels of vitamin D circulating in the blood (67).

Variant rs7041; Gene: GC

Lower levels of serum vitamin D are found in people with the A/A genotype for the rs7041 variant of the GC gene. Similar to this, those with the A/C genotype have somewhat lower levels of vitamin D. This shows that this genetic variation may affect how vitamin D is bound or transported, resulting in lower quantities of vitamin D circulating in the blood (68).

Variant rs12794714; Gene: CYP2R1

Lower amounts of vitamin D are found in people with the rs12794714 variant of the CYP2R1 gene, which has an A/A genotype. A/G genotype carriers have an intermediate phenotype, which means that their levels of vitamin D are in between those of A/A and G/G genotype carriers. This shows that this genetic variation may have an impact on the CYP2R1 enzyme's function or efficiency, altering how vitamin D is converted into its active form and ultimately affecting vitamin D levels (69).

Variant rs10741657; Gene: CYP2R1

Individuals with the G/G genotype for the rs10741657 variant of the CYP2R1 gene are more likely to suffer from vitamin D deficiency or insufficiency, similarly to those with the A/G genotype. This shows that this genetic variation may affect the CYP2R1 enzyme's performance or activity, which could lower the conversion of vitamin D into its active form and increase the risk of low vitamin D levels.

Variant rs2228570; Gene: VDR

Individuals who carry FokI mutation have the G/G genotype for the rs2228570 variant of the VDR gene; as a result, the levels of vitamin D may decline. These people might also be more vulnerable to fractures, malignant melanoma, and dengue fever. Normal vitamin D levels are present in people with the A/G genotype and in people with the A/A genotype. This shows that the VDR protein's activity or function, which can affect vitamin D levels and potentially contribute to some health problems associated with vitamin D shortage, may be impacted by this genetic variant (70).

Vitamin C

Ascorbic acid, another name for vitamin C, is a water-soluble vitamin that is essential for many biological processes, such as the development, growth, and repair of body tissues. For example, the creation of collagen—a protein that serves as the building block of connective tissues in the skin, bones, and blood vessels—depends on vitamin C. Among the foods that are high in vitamin C are citrus fruits, berries, kiwis, mangoes, red and green bell peppers, tomatoes, spinach, and guavas (71). A lack of vitamin C can cause scurvy, a disorder with the following symptoms: anemia; weariness; bleeding gums and loose teeth; slow wound healing; dry, rough, and scaly skin; swelling and coloring of the skin; and depression. Vitamin C insufficiency can be prevented or treated with dietary supplementation. Supplements might be a helpful choice for people who cannot get enough vitamin C from their diet or have certain medical problems that prevent optimal absorption (72).

The information about two genetic variations (rsID) and their relationship to vitamin C levels is provided in Table 8. Each variation is linked to a unique gene that is involved in the transportation of vitamin C.

Variant rs33972313; Gene: SLC23A1

In comparison to those with the C/T or C/C genotypes, plasma vitamin C concentrations are lower in people with the T/T genotype for the rs33972313 variation of the SLC23A1 gene. This implies that this genetic variation may affect the effectiveness or uptake of vitamin C, resulting in reduced amounts of this vitamin in the blood (73).

Table 8.SNPs related to Vitamin C and their effects.

Vitamin C					
RsID	**Gene**	**Polymorphism function**	**Alleles**	**wt/mt**	**Reference**
rs33972313	SLC23A1	9%-11% lower plasma vitamin C concentrations	T/T	mt/mt	(73,74)
		Lower plasma vitamin C	C/T	wt/mt	
		Typical	C/C	wt/wt	
rs6053005	SLC23A2	24% higher (on average) plasma vitamin C concentrations	T/T	mt/mt	(75,76)
		Typical vitamin C levels	C/T	wt/mt	
		Typical vitamin C levels	C/C	wt/wt	

Variant rs6053005; Gene: SLC23A2

In comparison to people with the C/T or C/C genotypes, those with the T/T genotype for the rs6053005 variant of the SLC23A2 gene typically have greater plasma vitamin C concentrations. This shows that this genetic variation may improve vitamin C efficiency or uptake, resulting in higher amounts of this vitamin in the blood (74).

Conclusion

The population-wide prevention and treatment of vitamin deficiency has been recognized as a key public health goal. Every person has a unique nutritional blueprint stored in their DNA. The scientific field of nutrigenomics studies the relationships between genes and nutrients, enabling the creation of individualized nutrition strategies to promote health and fend off disease. To better understand the interplay between genes and nutrients and to plan tailored weight loss, nutrigenetic testing may soon become a key approach.

Acknowledgements
This research was funded by the Provincia Autonoma di Trento in the framework of LP 6/99.

Conflicts of interest statement
Authors declare no conflict of interest.

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