

VitaGenic

Naturally Bioavailable Multivitamin

Alimentum Labs

alimentumlabs.com
1.800.445.4647

Last Revision:
March 10, 2024

VitaGenic

Naturally Bioavailable Multivitamin

A completely natural, organic, and plant-based source of vitamins in their most bioavailable forms to elevate vitality and address nutritional gaps.



Whole Body



Immunity



Metabolism



Detox

Health Indications

- Support Nutritional Balance & Micronutrient Sufficiency
- Enhance Cellular Energy and Metabolism
- Promote Immune Function and Antioxidant Defense
- Support Bone Strength and Joint Health
- Improve Cognitive Function and Mental Clarity
- Regulate Blood Sugar and Insulin Sensitivity
- Maintain Cardiovascular Health & Circulatory Function
- Promote Skin, Hair, and Nail Health

Instructions For Use

Take 1 capsule daily, or as directed by your healthcare provider.

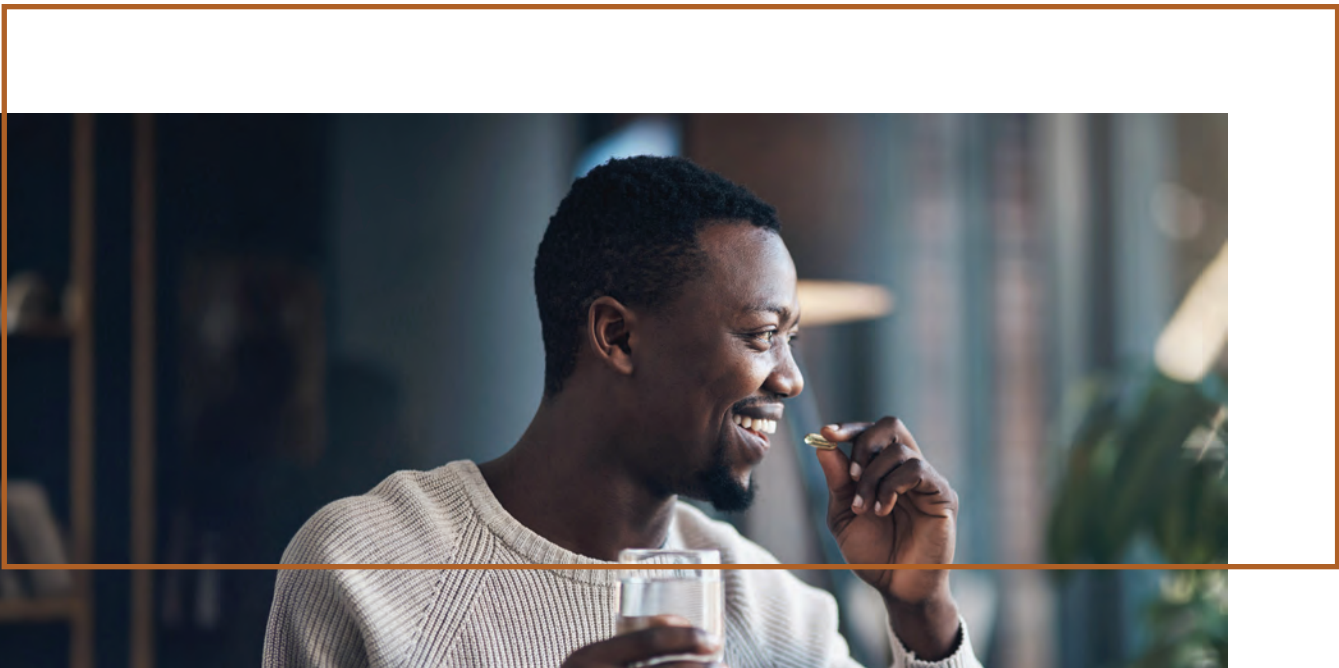
**Individual needs may vary; please consult your practitioner before altering the prescribed doses or protocols.

Product Description

VitaGenic is a 100% organic, plant-based multivitamin designed to deliver essential nutrients in their most bioavailable and natural forms. Unlike synthetic alternatives, VitaGenic sources its vitamins and minerals directly from whole-food plants like guava, lemon, amla, sesbania, holy basil, and annatto, ensuring maximum absorption, cellular nourishment, and bioactivity. With nutrients in their naturally occurring, synergistic forms, VitaGenic helps fill nutritional gaps, enhance vitality, and support overall well-being.

This formula provides a full-spectrum of essential vitamins and minerals without synthetic additives or artificial isolates, offering optimal cellular support and metabolic efficiency. The vitamin B12 in VitaGenic is naturally synthesized, mimicking the way probiotics function in the human gut to ensure maximum absorption and effectiveness.

Designed for cellular health, longevity, and daily vitality, VitaGenic provides a complete and balanced nutritional foundation to support energy, immunity, and overall well-being. Whether you're looking to optimize your health, enhance nutrient intake, or ensure your body receives vitamins in their most natural form, VitaGenic is the cleanest, most effective solution for whole-food-based nutrition.



Key Elements and Features of VitaGenic

100% Plant-Based & Organic

VitaGenic is formulated using only organic, whole-food sources, ensuring that all vitamins and minerals are in their most natural and bioavailable forms. Unlike synthetic multivitamins, this formula delivers nutrients as they occur in nature, maximizing absorption and effectiveness.

Provide a Comprehensive Nutrient Profile

Packed with essential vitamins, minerals, and phytonutrients, VitaGenic provides a full-spectrum nutritional foundation. It includes B-complex vitamins for energy, antioxidants for cellular protection, and essential minerals for metabolic balance, supporting overall vitality and well-being.

Optimize Bioavailability

With naturally occurring cofactors and bioavailability enhancers, VitaGenic ensures that nutrients are easily absorbed and effectively utilized by the body. For example, the inclusion of black pepper extract further enhances absorption, helping you get the most out of every dose.

Support Cellular Health & Longevity

By providing nutrients that fuel mitochondrial function, neurotransmitter balance, and immune defense, VitaGenic promotes long-term health and resilience. Key ingredients like sesbania, amla, and holy basil contribute to antioxidant protection and cellular regeneration.

A Pure, Clean, & Additive Free Option

VitaGenic is free from synthetic ingredients, artificial fillers, and harmful additives, making it a clean and safe choice for daily supplementation. It delivers only what your body needs—nothing extra, nothing synthetic—just pure, plant-powered nutrition.



Gene Spotlight

The vitamins and minerals in multivitamins, including VitaGenic's plant-based formula, interact with key genes that influence metabolism, cognitive function, bone health, immune response, and chronic disease prevention. Genetic variations in genes like *MTHFR*, *VDR*, *TCN2*, *COMT*, *SOD2*, *COL1A1*, *GPX1*, and others affect how individuals absorb and utilize nutrients, shaping nutritional needs and overall health outcomes. Understanding these interactions enables individuals to utilize personalized nutrition strategies designed to optimize wellness.

Genetic Interactions

***MTHFR* (Methylene-Tetrahydrofolate Reductase)**

The *MTHFR* gene regulates folate metabolism, which is essential for DNA synthesis, methylation, and neurotransmitter production. Research shows that variants, such as *MTHFR C677T*, can impair folate metabolism which may increase the risk of neural tube defects, cardiovascular disease, and cognitive decline. Furthermore, individuals with this mutation may benefit more from consuming the active form of vitamin B9, methylfolate (5-MTHF), rather than synthetic folic acid.¹

***VDR* (Vitamin D Receptor)**

VDR gene polymorphisms can affect vitamin D metabolism, impacting bone density, immune function, and inflammation regulation. Variants such as *VDR FokI* and *BsmI* influence how effectively vitamin D supports bone mineralization and calcium absorption, which is critical for individuals at risk of osteoporosis or autoimmune conditions.²

***TCN2* (Transcobalamin 2)**

The *TCN2* gene encodes the transport protein needed for vitamin B12 absorption and utilization. Mutations in *TCN2* can lead to reduced vitamin B12 transport, increasing the risk of neuropathy, anemia, and cognitive impairment. Individuals with these mutations may require higher vitamin B12 intake in its active methylcobalamin form.³

COMT (Catechol-O-Methyltransferase)

COMT regulates dopamine metabolism, influencing mood, cognitive function, and stress response. The *COMT Val158Met* polymorphism affects enzyme activity, thus impacting dopamine breakdown. Magnesium is essential for *COMT* enzyme function, and supplementation can help support cognitive flexibility and emotional regulation, especially in individuals with *COMT* variants.⁴

SOD2 (Superoxide Dismutase 2)

SOD2 encodes an antioxidant enzyme that protects cells from oxidative stress. Zinc plays a crucial role in supporting *SOD2* activity and reducing oxidative damage which is linked to aging, neurodegenerative diseases, and immune function. Variations in *SOD2* may impact antioxidant efficiency, making adequate zinc intake essential for cellular protection.⁵

COL1A1 (Collagen Type 1 Alpha 1)

COL1A1 is essential for bone formation, therefore, genetic variations in this gene can influence bone density and fracture risk. Calcium supplementation, particularly when paired with vitamin D, can be beneficial for individuals with *COL1A1* polymorphisms, as it helps support bone strength and lower the risk of osteoporosis.⁶

GPX1 (Glutathione Peroxidase 1)

GPX1 encodes an antioxidant enzyme that requires selenium for proper function to help to protect against oxidative damage, inflammation, and cellular aging. Variations in *GPX1* can affect antioxidant efficiency, making selenium intake critical for individuals with higher oxidative stress risks.⁷

How VitaGenic Works

VitaGenic works by combining a full-spectrum of essential vitamins, antioxidants, and minerals from organic, plant-based sources to support immune function, cellular repair, and daily vitality. Its B-vitamin complex promotes energy production, nervous system function, and cognitive support by aiding in the conversion of food into energy, regulating neurotransmitters, and maintaining metabolic balance. Essential minerals, delivered in natural whole-food forms, fortify bone strength, nerve signaling, muscle function, and metabolic health, while also regulating electrolyte balance, enzyme function, and antioxidant defense to promote hydration, cardiovascular well-being, and immune resilience. In addition, VitaGenic provides phytonutrients, flavonoids, and absorption boosters that enhance nutrient uptake and efficacy, ensuring protection against oxidative stress and inflammation. By harnessing the synergistic benefits of plant-based vitamins, essential minerals, and targeted bioavailability enhancers, VitaGenic delivers a comprehensive multivitamin designed to promote energy, longevity, and overall wellness.



Key Ingredients

Vitamin C (Ascorbate)

Vitamin C (ascorbate) plays a crucial role in immune function, collagen synthesis, and antioxidant defense. It enhances white blood cell activity, supports epithelial barrier integrity, and promotes faster wound healing. As a cofactor in collagen production, it is essential for skin, joint, and connective tissue health. Additionally, its antioxidant properties help neutralize free radicals, reducing oxidative stress and inflammation, both of which are key contributors to aging and chronic diseases.^{5,8,9}

Vitamin D3 (Cholecalciferol)

Vitamin D3 (cholecalciferol) plays a crucial role in bone health, immune function, and metabolic regulation. It enhances calcium and phosphorus absorption, ensuring proper bone mineralization and skeletal integrity, reducing the risk of osteoporosis and fractures. Beyond its effects on bones, vitamin D3 acts as an immune modulator, influencing T-cell activation, inflammatory response, and antimicrobial defense mechanisms. Genetic interactions with *VDR* (Vitamin D Receptor) polymorphisms can affect vitamin D metabolism and efficiency, potentially impacting bone density, immune responses, and susceptibility to metabolic disorders. Additionally, vitamin D3 has been linked to hormonal regulation, cardiovascular health, and neuroprotection, making it essential for overall wellness.¹⁰⁻¹⁵

Vitamin E (Mixed Tocopherols)

Vitamin E is a potent lipid-soluble antioxidant that plays a crucial role in cell membrane protection, immune function, and cardiovascular health. It neutralizes free radicals, preventing oxidative damage that can contribute to aging, inflammation, and chronic diseases. Vitamin E also influences gene regulation and cellular signaling, with research showing interactions with the *TTPA* gene that regulates vitamin E distribution in the body. Genetic variations in *TTPA* can impact vitamin E levels and its effectiveness in protecting neuronal and cardiovascular health. Additionally, vitamin E has been linked to immune modulation, inflammation control, and lipid metabolism, making it essential for overall cellular function and longevity.¹⁶⁻²⁰

Vitamin K2 (MK7)

Vitamin K2 (MK7) plays a crucial role in bone health, cardiovascular function, and calcium metabolism. It activates osteocalcin and matrix Gla protein (MGP), ensuring that calcium is properly deposited in bones rather than accumulating in arteries. This regulation helps prevent osteoporosis and vascular calcification, reducing the risk of bone fractures and heart disease. Genetic interactions with *VDR* (vitamin D receptor) and *MGP* may influence how effectively vitamin K2 supports bone mineralization and cardiovascular protection. Research suggests that deficiencies in vitamin K2 may lead to increased arterial stiffness and reduced bone density, making it essential for maintaining structural integrity and circulatory health.²¹⁻²⁴

Mixed Carotenoids (Vitamin A)

Mixed carotenoids (vitamin A), which act as precursors to retinoic acid, are essential for vision, immune function, and skin health. Retinoic acid plays a crucial role in regulating gene expression and cellular differentiation, supporting overall cellular function and development. They contribute to healthy vision by forming 11-cis-retinal, a key component of rhodopsin in the retina. Vitamin A also modulates immune responses by influencing *RBP4* (retinol-binding protein 4) expression, which is involved in regulating vitamin A transport. Additionally, it supports skin integrity by activating RAR (retinoic acid receptors), which play a role in keratinocyte differentiation and wound healing. Genetic mutations in the *BCO1* (β -carotene 15,15'-oxygenase 1) gene can impair the conversion of provitamin A carotenoids into retinoids, potentially leading to vitamin A deficiency despite sufficient dietary intake. These variations are particularly relevant in populations relying on plant-based sources of vitamin A, making bioavailable carotenoid intake crucial for maintaining optimal health.²⁵⁻²⁹

Thiamin (Vitamin B1)

Thiamin is essential for energy production and nervous system function, as it acts as a coenzyme in carbohydrate metabolism and ATP synthesis. It plays a key role in the Krebs cycle and pentose phosphate pathway, supporting efficient energy transfer within cells. Thiamin is also crucial for nerve signal transmission and myelin sheath maintenance, making it vital for cognitive and neuromuscular function. Genetic interactions with the *SLC19A2* and *SLC19A3* genes that both encode thiamin transporters, may influence cellular uptake and distribution, thus potentially affecting neurological and metabolic health. Mutations in these genes may lead to thiamin-responsive conditions such as biotin-thiamin-responsive basal ganglia disease, highlighting the importance of adequate B1 intake for maintaining neurological resilience and energy metabolism.³⁰⁻³⁴

Riboflavin (Vitamin B2)

Riboflavin is a key component of energy metabolism, cellular function, and antioxidant defense. It acts as a precursor to flavin mononucleotide and flavin adenine dinucleotide, both of which are essential cofactors in redox reactions. It plays a crucial role in ATP production, fatty acid oxidation, and amino acid metabolism, supporting mitochondrial efficiency and reducing oxidative stress. Riboflavin also contributes to neurological health and red blood cell production, ensuring proper oxygen transport and nervous system function. Genetic interactions with *SLC52A1*, *SLC52A2*, and *SLC52A3*—which encode riboflavin transporters—regulate its absorption and cellular uptake. Mutations in these genes can lead to riboflavin transporter deficiency, a rare neuromuscular disorder that impairs energy metabolism and nerve function. Deficiencies in riboflavin have been linked to neurological disorders, anemia, and metabolic dysfunctions, underscoring its critical role in maintaining overall health.^{35–39}

Niacin (Vitamin B3)

Niacin is essential for metabolism and cognitive health, serving as a precursor for nicotinamide adenine dinucleotide (NAD⁺) and nicotinamide adenine dinucleotide phosphate (NADP⁺), which drive cellular energy production and redox reactions. It supports ATP synthesis, DNA repair, and neuroprotection, playing a role in cognitive function, brain aging, and neurological resilience. Niacin is also involved in ADP-ribosylation reactions, impacting gene expression, calcium signaling, and inflammatory responses. Genetic interactions with *PNPLA3*, which has been linked to lipid metabolism and neurodegenerative disorders, and *SIRT1*, which is involved in cellular stress response and longevity, influence niacin's role in brain health, energy metabolism, and neuroinflammation. Deficiencies in niacin can result in cognitive decline, fatigue, and metabolic imbalances, underscoring its critical role in maintaining neurological and metabolic function.^{40–44}

Vitamin B6 (Pyridoxine)

Vitamin B6 (pyridoxine) is essential for neurotransmitter production and brain function, serving as a cofactor in the synthesis of dopamine, serotonin, GABA, and norepinephrine. It plays a crucial role in amino acid metabolism, neurotransmission, and cognitive development, impacting mood regulation, memory, and neuronal health. Pyridoxal 5'-phosphate (PLP), the active form of vitamin B6, is necessary for synaptic plasticity and neuroprotection, with genetic interactions involving *PROSC*, which regulates PLP homeostasis, and *SLC25A39*, which influences mitochondrial metabolism. Mutations in *PROSC* are linked to vitamin B6-dependent epilepsy, highlighting its importance in neurological function. Adequate B6 levels support brain health, nervous system function, and overall cognitive resilience.⁴⁵⁻⁴⁹

Folate (Vitamin B9)

Folate (vitamin B9) is essential for DNA synthesis, red blood cell formation, and cellular methylation processes, playing a critical role in cell division and genetic stability. It is required for purine and pyrimidine biosynthesis, ensuring the proper replication and repair of DNA. Folate also supports erythropoiesis, preventing megaloblastic anemia by enabling the maturation of red blood cells. Genetic interactions with *MTHFR* (methylenetetrahydrofolate reductase) influence folate metabolism and homocysteine regulation, impacting cardiovascular and neurological health. Variants in *MTHFR* can lead to reduced folate utilization, increasing risks of neural tube defects, cognitive decline, and vascular disorders. Maintaining adequate folate levels is crucial for genome integrity, prenatal development, and overall metabolic function.⁵⁰⁻⁵⁴

Vitamin B12 (Methylcobalamin)

Vitamin B12 (methylcobalamin) is crucial for nerve health, energy production, and red blood cell formation, playing a key role in DNA synthesis, myelin sheath maintenance, and cellular metabolism. It supports neurological function by aiding neurotransmitter regulation and reducing homocysteine levels, which, if elevated, can increase the risk of neurodegenerative diseases. Vitamin B12 is also essential for red blood cell production, preventing megaloblastic anemia and ensuring proper oxygen transport. Genetic interactions with *MTHFR* (methylene tetrahydrofolate reductase) and *TCN2* (transcobalamin 2) influence vitamin B12 metabolism and transport, affecting neurological resilience and cardiovascular health. Deficiencies can lead to fatigue, cognitive decline, and nerve damage, underscoring the necessity of maintaining optimal vitamin B12 levels.⁵⁵⁻⁵⁹

Biotin

Biotin (vitamin B7) is essential for skin, hair, and metabolic function, playing a crucial role in fatty acid synthesis, amino acid metabolism, and energy production. It serves as a cofactor for biotin-dependent carboxylases, which regulate gluconeogenesis, lipid metabolism, and mitochondrial function. Biotin supports keratin production, promoting healthy hair growth and skin regeneration. Genetic interactions with *BTD* (biotinidase) influence biotin recycling and availability, while mutations in *SLC5A6* impact biotin absorption, leading to deficiency-related conditions like dermatitis, brittle nails, and alopecia. Adequate biotin levels contribute to metabolic efficiency, cellular health, and structural integrity of skin and hair.⁶⁰⁻⁶⁴

Pantothenic Acid (Vitamin B5)

Pantothenic acid (vitamin B5) is essential for energy metabolism and hormone synthesis, playing a crucial role as a precursor to coenzyme A (CoA), which supports the citric acid cycle, fatty acid oxidation, and neurotransmitter production. It regulates cholesterol, steroid hormone, and hemoglobin biosynthesis, influencing metabolic pathways that maintain hormonal balance and cellular energy production. Genetic interactions with *PANK1* and *PANK2* (pantothenate kinases) affect CoA biosynthesis and lipid metabolism, with *PANK2* mutations linked to neurodegenerative disorders such as pantothenate kinase-associated neurodegeneration (PKAN). Maintaining optimal vitamin B5 levels is crucial for hormonal health, cognitive function, and metabolic efficiency.^{65–69}

Calcium

Calcium is essential for bone health and muscle function, serving as a fundamental mineral in skeletal development, neuromuscular signaling, and cellular metabolism. Over 99% of the body's calcium is stored in bones and teeth, where it supports bone density and structural integrity. Calcium also plays a key role in muscle contraction and relaxation, acting as a signal for excitation-contraction coupling. Genetic interactions with *CASR* (calcium-sensing receptor) regulate calcium homeostasis and parathyroid hormone secretion, while variations in *SLC8A1* influence calcium transport and cardiac function. Deficiencies in calcium can lead to osteoporosis, muscle cramps, and impaired cellular signaling, emphasizing its importance in maintaining skeletal and neuromuscular health.^{70–73}

Magnesium

Magnesium is vital for nerve transmission, muscle relaxation, and heart health, serving as a cofactor in over 300 enzymatic reactions that regulate neuromuscular function, cardiovascular stability, and cellular metabolism. It acts as a calcium antagonist, preventing excessive excitation in nerves and muscles while supporting proper cardiac rhythm and blood vessel dilation. Magnesium also plays a role in ATP production, making it essential for energy metabolism and muscle function. Genetic interactions with *CASR* (calcium-sensing receptor) and *TRPM6* (transient receptor potential cation channel) regulate magnesium absorption and homeostasis, impacting cardiovascular and neuromuscular health. Deficiencies in magnesium can lead to muscle cramps, arrhythmias, and impaired nerve signaling, highlighting its role in maintaining optimal physiological function.⁷⁴⁻⁷⁷

Zinc

Zinc is essential for immune function, wound healing, and enzymatic activity, serving as a cofactor for over 300 enzymes involved in DNA repair, protein synthesis, and cellular metabolism. It plays a crucial role in T-cell activation, cytokine regulation, and antioxidant defense, supporting immune resilience and inflammation control. Zinc is also critical for collagen formation and tissue regeneration, making it essential for wound healing and skin repair. Genetic interactions with *SLC39A1* (ZIP1) and *SLC30A1* (ZnT1) regulate zinc homeostasis and transport, influencing immune response and inflammatory pathways. Variants in *MT1A* (metallothionein 1A) affect zinc metabolism and oxidative stress regulation, impacting susceptibility to immune dysfunction and wound healing impairments. Zinc deficiency can lead to weakened immunity, delayed wound healing, and metabolic imbalances, highlighting its role in maintaining cellular health and immune efficiency.⁷⁸⁻⁸¹

Selenium

Selenium is essential for antioxidant defense and thyroid function, acting as a key component of selenoproteins, including glutathione peroxidases (*GPX1*, *GPX3*) and thioredoxin reductases (*TXNRD1*, *TXNRD2*), which protect against oxidative stress and inflammation. In the thyroid, selenium plays a critical role in thyroid hormone metabolism, with *DIO1* (deiodinase 1) and *DIO2* (deiodinase 2) facilitating the activation of thyroid hormones. Selenium deficiency has been linked to Hashimoto's thyroiditis, Graves' disease, and thyroid dysfunction due to increased oxidative stress in thyroid tissue. Proper selenium levels help reduce inflammation, modulate immune response, and support metabolic homeostasis, making it crucial for thyroid health and cellular protection.⁸²⁻⁸⁵

Copper

Copper is essential for red blood cell formation and iron metabolism, acting as a key cofactor in heme synthesis and iron transport. It plays a crucial role in erythropoiesis, supporting hemoglobin production and ensuring efficient oxygen transport. Copper-dependent enzymes, such as ceruloplasmin and hephaestin, facilitate iron mobilization and oxidation, regulating iron absorption and preventing anemia. Genetic interactions with *ATP7A* and *ATP7B* influence copper homeostasis, while variations in *SLC40A1* (ferroportin) affect iron export from cells. Copper deficiency can lead to microcytic anemia and impaired iron utilization, underscoring its importance in maintaining blood health and metabolic balance.⁸⁶⁻⁸⁹

Manganese

Manganese is essential for bone health and antioxidant activity, playing a critical role in bone mineralization, skeletal development, and oxidative stress regulation. It is required for osteoblast activity and collagen synthesis, supporting bone density and structural integrity. Manganese also serves as a cofactor for manganese superoxide dismutase (MnSOD, encoded by *SOD2*), a key mitochondrial antioxidant enzyme that protects cells from oxidative damage. Genetic interactions with *SLC39A8* influence manganese transport and homeostasis, impacting both bone metabolism and cellular defense mechanisms. Deficiencies in manganese can contribute to osteoporosis, impaired antioxidant function, and increased susceptibility to oxidative stress, underscoring its importance in maintaining bone strength and cellular resilience.^{90–93}

Chromium

Chromium is essential for blood sugar regulation and insulin function, acting as a cofactor that enhances insulin signaling and glucose metabolism. It plays a role in carbohydrate and lipid metabolism, improving insulin sensitivity and facilitating glucose uptake in cells. Chromium is involved in the function of *SLC11A2* (solute carrier family 11 member 2), which affects glucose transport, and *INSR* (insulin receptor), which modulates insulin binding and action. Studies suggest that chromium deficiency is associated with impaired glucose tolerance and increased risk of type 2 diabetes, emphasizing its role in metabolic homeostasis and endocrine function.^{94–97}

Potassium

Potassium is essential for heart health and electrolyte balance, playing a critical role in regulating blood pressure, nerve signaling, and muscle contractions. It helps maintain cellular fluid balance and generates the electrical impulses needed for heart rhythm and neuromuscular function. Potassium is transported by *KCNJ2* (inwardly rectifying potassium channel 2), which regulates cardiac excitability, and *SLC12A3*, which influences renal potassium handling and blood pressure stability. Deficiencies or imbalances in potassium levels can lead to hypertension, arrhythmias, and neuromuscular dysfunction, underscoring its importance in maintaining cardiovascular and metabolic homeostasis.^{98–101}

Sesbania

Sesbania (*Sesbania grandiflora*) is a nutrient-rich plant containing a variety of essential vitamins, minerals, and phytonutrients. It is particularly high in vitamin A, B-complex vitamins, and vitamin C, contributing to its diverse nutritional profile. The plant is also a good source of calcium, phosphorus, iron, and potassium, supporting its role as a natural mineral source. Sesbania contains flavonoids, tannins, and saponins, which contribute to its bioactive properties. Additionally, its leaves, flowers, and seeds contain triterpenoids and essential amino acids, enhancing its value as a plant-based nutritional supplement.^{102–105}

Guava

Guava (*Psidium guajava*) is rich in vitamins, minerals, and phytonutrients, making it a valuable whole-food source of essential nutrients. It is particularly high in vitamin C, carotenoids, and B-complex vitamins, which contribute to its nutritional profile. Guava also contains dietary fiber, polyphenols, and flavonoids, such as quercetin and catechins, which enhance its antioxidant potential. Additionally, it provides potassium, calcium, magnesium, and iron, supporting its role as a plant-based mineral source. These natural compounds make guava a highly nutritious ingredient in whole-food-based supplementation.^{106–109}

Amla

Amla (*Phyllanthus emblica*), also known as Indian gooseberry, is a nutrient-dense fruit rich in vitamins, minerals, and bioactive compounds. It is one of the most concentrated natural sources of vitamin C, alongside a variety of polyphenols, flavonoids, and tannins such as gallic acid, ellagic acid, emblicanin A & B, and quercetin. Amla also provides essential B-complex vitamins, calcium, phosphorus, iron, and magnesium, making it a valuable whole-food source of nutrients. Additionally, its high antioxidant content and diverse phytochemicals contribute to its broad nutritional profile.^{110–113}

Holy Basil

Holy Basil (*Ocimum sanctum*), also known as Tulsi, is a rich source of vitamins, minerals, and phytonutrients. It contains vitamin C, vitamin A, and essential B-complex vitamins, along with key minerals such as calcium, iron, magnesium, and potassium. Holy Basil is abundant in flavonoids, polyphenols, and terpenoids, including eugenol, rosmarinic acid, luteolin, and apigenin, which contribute to its diverse bioactive properties. Additionally, it contains essential oils and tannins, further enhancing its nutritional profile as a plant-based supplement.^{114–117}

Annatto

Annatto (*Bixa orellana*) is a rich source of carotenoids, tocotrienols, and essential minerals, making it a valuable plant-derived nutritional ingredient. The seeds contain bixin and norbixin, two potent carotenoid compounds responsible for its vibrant color and antioxidant properties. It is also high in vitamin E (tocotrienols and tocopherols), which contribute to its bioactive potential. Annatto provides essential minerals such as calcium, phosphorus, and magnesium, supporting its role as a natural nutrient source. Additionally, the plant contains polyphenols and flavonoids, further enhancing its diverse phytochemical profile.^{118–121}

Lemon Extract

Lemon extract is rich in vitamin C, flavonoids, polyphenols, and carotenoids, providing a diverse range of plant-based nutrients. It contains hesperidin, naringenin, and quercetin, known for their antioxidant properties, as well as trace minerals like potassium, magnesium, and calcium. The presence of essential oils and organic acids further enhances its nutritional profile, making it a natural source of bioactive compounds.^{122–125}

Leucine

Leucine is an essential branched-chain amino acid (BCAA) that plays a crucial role in protein synthesis, muscle metabolism, and energy regulation. It activates the mTOR (mechanistic target of rapamycin) signaling pathway, stimulating muscle protein synthesis and reducing muscle degradation. Leucine also influences glucose metabolism and mitochondrial function, enhancing energy production and cellular respiration. Approximately 80% of leucine is used for protein biosynthesis, while the remainder is metabolized into α -ketoisocaproate (α -KIC) and β -hydroxy β -methylbutyrate (HMB), which further support muscle preservation and metabolic regulation.^{126–129}

Black Pepper Extract (95%)

Black pepper extract (95%) enhances nutrient absorption and bioavailability primarily through its active compound piperine, which modulates intestinal membrane permeability, enzyme activity, and transporter function. Piperine inhibits *CYP3A4*, a key enzyme involved in drug metabolism, and *ABCB1* (P-glycoprotein), a transporter that limits the absorption of various compounds, thereby increasing systemic bioavailability. It also interacts with *UGT1A1* (UDP-glucuronosyltransferase 1A1), reducing the glucuronidation of certain nutrients and prolonging their circulation. Variants in these genes may influence individual responses to piperine, affecting the degree of nutrient enhancement and metabolic processing. These interactions make piperine a powerful natural bioenhancer for vitamins, polyphenols, and coenzymes in whole-food supplementation.^{130–133}

Warnings/Contraindications

When used as directed there are no known contraindications for VitaGenic.

It is always recommended that you consult your practitioner prior to adding any new supplement to your regimen if you are pregnant, breastfeeding, experiencing renal failure, undergoing an organ transplant(s), managing diabetes with insulin, or are taking medication(s) for any pre-existing conditions.

Safety

All ingredients are tested before use for:

- Pathogenic microbial contaminants
- Heavy metals and/or chemical contaminants
- Purity

Additional Information

- Gluten Free
- Dairy Free
- Vegan
- No Sugar
- Non-GMO
- cGMP Facility



References

1. Molina-López, J.; Ricalde, M. A. Q.; Hernández, B. V.; Planells, A.; Otero, R.; Planells, E. Effect of 8-Week of Dietary Micronutrient Supplementation on Gene Expression in Elite Handball Athletes. *PloS One* **2020**, *15* (5), e0232237. <https://doi.org/10.1371/journal.pone.0232237>.
2. Huang, H.-Y.; Caballero, B.; Chang, S.; Alberg, A. J.; Semba, R. D.; Schneyer, C. R.; Wilson, R. F.; Cheng, T.-Y.; Vassy, J.; Prokopowicz, G.; Barnes, G. J.; Bass, E. B. The Efficacy and Safety of Multivitamin and Mineral Supplement Use to Prevent Cancer and Chronic Disease in Adults: A Systematic Review for a National Institutes of Health State-of-the-Science Conference. *Ann. Intern. Med.* **2006**, *145* (5), 372–385. <https://doi.org/10.7326/0003-4819-145-5-200609050-00135>.
3. Kondo, H.; Binder, M. J.; Kolhouse, J. F.; Smythe, W. R.; Podell, E. R.; Allen, R. H. Presence and Formation of Cobalamin Analogues in Multivitamin–Mineral Pills. *J. Clin. Invest.* **1982**, *70* (4), 889–898. <https://doi.org/10.1172/jci110685>.
4. Papenberg, G.; Salami, A.; Persson, J.; Lindenberger, U.; Bäckman, L. Genetics and Functional Imaging: Effects of APOE, BDNF, COMT, and KIBRA in Aging. *Neuropsychol. Rev.* **2015**, *25* (1), 47–62. <https://doi.org/10.1007/s11065-015-9279-8>.
5. Yetley, E. A. Multivitamin and Multimineral Dietary Supplements: Definitions, Characterization, Bioavailability, and Drug Interactions. *Am. J. Clin. Nutr.* **2007**, *85* (1), 269S–276S. <https://doi.org/10.1093/ajcn/85.1.269S>.
6. Wang, S.-M.; Yin, L.-Y.; Zhang, Y.; Fan, J.-H.; Chang, I. J.; Dawsey, S. M.; Taylor, P. R.; Abnet, C. C.; Qiao, Y.-L. Multivitamin and Mineral Supplementation Is Associated with the Reduction of Fracture Risk and Hospitalisation Rate in Chinese Adult Males: A Randomised Controlled Study. *J. Bone Miner. Metab.* **2015**, *33* (3), 294–302. <https://doi.org/10.1007/s00774-014-0589-3>.

7. Handy, D. E.; Loscalzo, J. The Role of Glutathione Peroxidase-1 in Health and Disease. *Free Radic. Biol. Med.* **2022**, *188*, 146–161. <https://doi.org/10.1016/j.freeradbiomed.2022.06.004>.
8. Sánchez, M. C.; Herráiz, A.; Ciudad, M. J.; Arias, M.; Alonso, R.; Doblas, C.; Llama-Palacios, A.; Collado, L. Metabolomics and Biochemical Benefits of Multivitamin and Multiminerall Supplementation in Healthy Individuals: A Pilot Study. *Foods* **2024**, *13* (14), 2207. <https://doi.org/10.3390/foods13142207>.
9. Comerford, K. B. Recent Developments in Multivitamin/Mineral Research. *Adv. Nutr. Bethesda Md* **2013**, *4* (6), 644–656. <https://doi.org/10.3945/an.113.004523>.
10. Wiliński, B.; Wiliński, J.; Somogyi, E.; Piotrowska, J.; Opoka, W. Vitamin D3 (Cholecalciferol) Boosts Hydrogen Sulfide Tissue Concentrations in Heart and Other Mouse Organs. *Folia Biol. (Praha)* **2012**, *60* (3–4), 243–247. https://doi.org/10.3409/fb60_3-4.243-247.
11. Falchetti, A.; Rossi, E.; Cosso, R.; Buffa, A.; Corvaglia, S.; Malavolta, N. Vitamin D and Bone Health. *Food Nutr. Sci.* **2016**, *7* (11), 1033–1051. <https://doi.org/10.4236/fns.2016.711100>.
12. Querales, M. I.; Cruces, M. E.; Rojas, S.; Sánchez, L. [Association between vitamin D deficiency and metabolic syndrome]. *Rev. Med. Chil.* **2010**, *138* (10), 1312–1318.
13. R, S.; Arjunkumar, R. Vitamin D Deficiency in Periodontal Health. *Res. J. Pharm. Technol.* **2014**, *7* (2), 248–252.
14. Amano, Y.; Komiyama, K.; Makishima, M. Vitamin D and Periodontal Disease. *J. Oral Sci.* **2009**, *51* (1), 11–20. <https://doi.org/10.2334/josnusd.51.11>.

15. Giacomet, V.; Vigano, A.; Manfredini, V.; Cerini, C.; Bedogni, G.; Mora, S.; Borelli, M.; Trabattoni, D.; Zuccotti, G. V. Cholecalciferol Supplementation in HIV-Infected Youth with Vitamin D Insufficiency: Effects on Vitamin D Status and T-Cell Phenotype: A Randomized Controlled Trial. *HIV Clin. Trials* **2013**, *14* (2), 51–60. <https://doi.org/10.1310/hct1402-51>.
16. Miller, G. W.; Ulatowski, L.; Labut, E. M.; Lebold, K. M.; Manor, D.; Atkinson, J.; Barton, C. L.; Tanguay, R. L.; Traber, M. G. The α -Tocopherol Transfer Protein Is Essential for Vertebrate Embryogenesis. *PLoS One* **2012**, *7* (10), e47402. <https://doi.org/10.1371/journal.pone.0047402>.
17. Naguib, Y.; Hari, S. P.; Passwater, R.; Huang, D. Antioxidant Activities of Natural Vitamin E Formulations. *J. Nutr. Sci. Vitaminol. (Tokyo)* **2003**, *49* (4), 217–220. <https://doi.org/10.3177/jnsv.49.217>.
18. Karladee, D.; Boonsit, P.; Suriyong, S.; Korawan; Sringarm. Antioxidant Capacities of Vitamin E (Tocopherols) in Purple Rice (*Oryza Sativa* L. Indica), Perilla (*Perilla Frutescens* L.) and Sesame (*Sesamum Indicum*). *Int. J. Agric. Technol.* **2013**.
19. Fischer, A.; Rimbach, G. Gene Regulatory Activity of Vitamin E. In *Vitamin E in Human Health*; Weber, P., Birringer, M., Blumberg, J. B., Eggersdorfer, M., Frank, J., Eds.; Springer International Publishing: Cham, 2019; pp 81–98. https://doi.org/10.1007/978-3-030-05315-4_7.
20. Traber, M. G.; Packer, L. Vitamin E: Beyond Antioxidant Function. *Am. J. Clin. Nutr.* **1995**, *62* (6 Suppl), 1501S–1509S. <https://doi.org/10.1093/ajcn/62.6.1501S>.
21. Jadhav, N.; Ajgaonkar, S.; Saha, P.; Gurav, P.; Pandey, A.; Basudkar, V.; Gada, Y.; Panda, S.; Jadhav, S.; Mehta, D.; Nair, S. Molecular Pathways and Roles for Vitamin K2–7 as a Health-Beneficial Nutraceutical: Challenges and Opportunities. *Front. Pharmacol.* **2022**, *13*, 896920. <https://doi.org/10.3389/fphar.2022.896920>.

22. Kaesler, N.; Schurgers, L. J.; Floege, J. Vitamin K and Cardiovascular Complications in Chronic Kidney Disease Patients. *Kidney Int.* **2021**, *100* (5), 1023–1036. <https://doi.org/10.1016/j.kint.2021.06.037>.
23. Capozzi, A.; Scambia, G.; Migliaccio, S.; Lello, S. Role of Vitamin K2 in Bone Metabolism: A Point of View and a Short Reappraisal of the Literature. *Gynecol. Endocrinol. Off. J. Int. Soc. Gynecol. Endocrinol.* **2020**, *36* (4), 285–288. <https://doi.org/10.1080/09513590.2019.1689554>.
24. Friedman, T. The Role of Vitamin K2 in Bone and Cardiovascular Health. *J. Restor. Med.* **2016**, *5* (1), 14. <https://doi.org/10.14200/jrm.2016.5.0101>.
25. Graßmann, S.; Pivovarova–Ramich, O.; Henze, A.; Raila, J.; Ampem Amoako, Y.; King Nyamekye, R.; Bedu–Addo, G.; Mockenhaupt, F. P.; Schulze, M. B.; Danquah, I. SNP Rs6564851 in the BCO1 Gene Is Associated with Varying Provitamin a Plasma Concentrations but Not with Retinol Concentrations among Adolescents from Rural Ghana. *Nutrients* **2020**, *12* (6), 1786. <https://doi.org/10.3390/nu12061786>.
26. Spiegler, E.; Kim, Y.–K.; Hoyos, B.; Narayanasamy, S.; Jiang, H.; Savio, N.; Curley, R. W.; Harrison, E. H.; Hammerling, U.; Quadro, L. β -Apo-10'-Carotenoids Support Normal Embryonic Development during Vitamin A Deficiency. *Sci. Rep.* **2018**, *8* (1), 8834. <https://doi.org/10.1038/s41598-018-27071-3>.
27. Lietz, G.; Lange, J.; Rimbach, G. Molecular and Dietary Regulation of Beta,Beta-Carotene 15,15'-Monooxygenase 1 (BCMO1). *Arch. Biochem. Biophys.* **2010**, *502* (1), 8–16. <https://doi.org/10.1016/j.abb.2010.06.032>.
28. Zumaraga, M. P. P.; Arquiza, J. M. R. A.; Concepcion, M. A.; Perlas, L.; Alcudia–Catalma, M. N.; Rodriguez, M. Genotype Effects on β -Carotene Conversion to Vitamin A: Implications on Reducing Vitamin A Deficiency in the Philippines. *Food Nutr. Bull.* **2022**, *43* (1), 25–34. <https://doi.org/10.1177/03795721211060229>.

29. Widjaja-Adhi, M. A. K.; Lobo, G. P.; Golczak, M.; Von Lintig, J. A Genetic Dissection of Intestinal Fat-Soluble Vitamin and Carotenoid Absorption. *Hum. Mol. Genet.* **2015**, *24* (11), 3206–3219. <https://doi.org/10.1093/hmg/ddv072>.
30. Bettendorff, L. CHAPTER 5. The Chemistry, Biochemistry and Metabolism of Thiamin (Vitamin B1); Preedy, V. R., Ed.; Royal Society of Chemistry: Cambridge, 2012; pp 71–92. <https://doi.org/10.1039/9781849734714-00071>.
31. Guillard, J.-C. [Vitamin B1 (thiamine)]. *Rev. Prat.* **2013**, *63* (8), 1074–1075, 1077–1078.
32. Wiley, K.; Gupta, M. Vitamin B1 Thiamine Deficiency; 2020.
33. Mrowicka, M.; Mrowicki, J.; Dragan, G.; Majsterek, I. The Importance of Thiamine (Vitamin B1) in Humans. *Biosci. Rep.* **2023**, *43* (10), BSR20230374. <https://doi.org/10.1042/BSR20230374>.
34. Bettendorff, L.; Wins, P. Biochemistry of Thiamine and Thiamine Phosphate Compounds. In *Encyclopedia of Biological Chemistry (Second Edition)*; Lennarz, W. J., Lane, M. D., Eds.; Academic Press: Waltham, 2013; pp 202–209. <https://doi.org/10.1016/B978-0-12-378630-2.00102-X>.
35. Henriques, B. J.; Lucas, T. G.; Gomes, C. M. Therapeutic Approaches Using Riboflavin in Mitochondrial Energy Metabolism Disorders. *Curr. Drug Targets* **2016**, *17* (13), 1527–1534. <https://doi.org/10.2174/1389450117666160813180812>.
36. Powers, H. J.; Corfe, B. M.; Nakano, E. Riboflavin in Development and Cell Fate. *Subcell. Biochem.* **2012**, *56*, 229–245. https://doi.org/10.1007/978-94-007-2199-9_12.
37. Mosegaard, S.; Bruun, G. H.; Flyvbjerg, K. F.; Blikrud, Y. T.; Gregersen, N.; Dembic, M.; Annexstad, E.; Tangeraas, T.; Olsen, R. K. J.; Andresen, B. S. An Intronic Variation in SLC52A1 Causes Exon Skipping and Transient Riboflavin-Responsive Multiple Acyl-CoA Dehydrogenation Deficiency. *Mol. Genet. Metab.* **2017**, *122* (4), 182–188. <https://doi.org/10.1016/j.ymgme.2017.10.014>.

38. McNulty, H.; Pentieva, K.; Ward, M. Causes and Clinical Sequelae of Riboflavin Deficiency. *Annu. Rev. Nutr.* **2023**, *43*, 101–122. <https://doi.org/10.1146/annurev-nutr-061121-084407>.
39. da Silva–Araújo, E. R.; Toscano, A. E.; Silva, P. B. P.; Pereira Dos Santos Junior, J.; Gouveia, H. J. C. B.; da Silva, M. M.; Souza, V. da S.; de Freitas Silva, S. R.; Manhães–de–Castro, R. Effects of Deficiency or Supplementation of Riboflavin on Energy Metabolism: A Systematic Review with Preclinical Studies. *Nutr. Rev.* **2025**, *83* (2), e332–e342. <https://doi.org/10.1093/nutrit/nuae041>.
40. Aguilera–Méndez, A.; Fernández–Lainez, C.; Ibarra–González, I.; Fernandez–Mejia, C. The Chemistry and Biochemistry of Niacin (B3).
41. Kirkland, J. B. Niacin Status, NAD Distribution and ADP–Ribose Metabolism. *Curr. Pharm. Des.* **2009**, *15* (1), 3–11. <https://doi.org/10.2174/138161209787185823>.
42. Paolini, E.; Longo, M.; Meroni, M.; Tria, G.; Cespiati, A.; Lombardi, R.; Badiali, S.; Maggioni, M.; Fracanzani, A. L.; Dongiovanni, P. The I148M PNPLA3 Variant Mitigates Niacin Beneficial Effects: How the Genetic Screening in Non–Alcoholic Fatty Liver Disease Patients Gains Value. *Front. Nutr.* **2023**, *10*, 1101341. <https://doi.org/10.3389/fnut.2023.1101341>.
43. Qin, B.; Xun, P.; Jacobs, D. R.; Zhu, N.; Daviglius, M. L.; Reis, J. P.; Steffen, L. M.; Van Horn, L.; Sidney, S.; He, K. Intake of Niacin, Folate, Vitamin B–6, and Vitamin B–12 through Young Adulthood and Cognitive Function in Midlife: The Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Am. J. Clin. Nutr.* **2017**, *106* (4), 1032–1040. <https://doi.org/10.3945/ajcn.117.157834>.
44. *Niacin in the Central Nervous System: An Update of Biological Aspects and Clinical Applications*. <https://www.mdpi.com/1422-0067/20/4/974> (accessed 2025–02–26).

45. Darin, N.; Reid, E.; Prunetti, L.; Samuelsson, L.; Husain, R. A.; Wilson, M.; El Yacoubi, B.; Footitt, E.; Chong, W. K.; Wilson, L. C.; Prunty, H.; Pope, S.; Heales, S.; Lascelles, K.; Champion, M.; Wassmer, E.; Veggiotti, P.; de Crécy-Lagard, V.; Mills, P. B.; Clayton, P. T. Mutations in PROSC Disrupt Cellular Pyridoxal Phosphate Homeostasis and Cause Vitamin-B6-Dependent Epilepsy. *Am. J. Hum. Genet.* **2016**, *99* (6), 1325–1337. <https://doi.org/10.1016/j.ajhg.2016.10.011>.
46. Brown, M.; Ameer, M. A.; Beier, K. Vitamin B6 Deficiency (Pyridoxine); 2019.
47. Plecko, B.; Struys, E. A.; Jakobs, C. Vitamin B6-Dependent and Responsive Disorders. In *Physician's Guide to the Diagnosis, Treatment, and Follow-Up of Inherited Metabolic Diseases*; Blau, N., Duran, M., Gibson, K. M., Dionisi Vici, C., Eds.; Springer: Berlin, Heidelberg, 2014; pp 179–190. https://doi.org/10.1007/978-3-642-40337-8_11.
48. Dakshinamurti, S.; Dakshinamurti, K. Antihypertensive and Neuroprotective Actions of Pyridoxine and Its Derivatives. *Can. J. Physiol. Pharmacol.* **2015**, *93* (12), 1083–1090. <https://doi.org/10.1139/cjpp-2015-0098>.
49. *The Alkaline Phosphatase (ALPL) Locus Is Associated with B6 Vitamer Levels in CSF and Plasma.* <https://www.mdpi.com/2073-4425/10/1/8> (accessed 2025-02-26).
50. Fenech, M. Folate (Vitamin B9) and Vitamin B12 and Their Function in the Maintenance of Nuclear and Mitochondrial Genome Integrity. *Mutat. Res.* **2012**, *733* (1–2), 21–33. <https://doi.org/10.1016/j.mrfmmm.2011.11.003>.
51. Frye, R. E.; Sequeira, J. M.; Quadros, E. V.; James, S. J.; Rossignol, D. A. Cerebral Folate Receptor Autoantibodies in Autism Spectrum Disorder. *Mol. Psychiatry* **2013**, *18* (3), 369–381. <https://doi.org/10.1038/mp.2011.175>.
52. Freha, G.; Fatma, M.; Cherifa, H. Folates: Key Nutrients to Remember. *Asian J. Pharm. Res. Health Care* **2017**, *9* (2), 75. <https://doi.org/10.18311/ajprhc/2017/10828>.

53. Altmäe, S.; Laanpere, M.; Campoy, C.; Salumets, A. 27. Folate and Female Infertility: Folate–Metabolizing Pathway in Folliculogenesis, Infertility Treatment, and Implantation; Brill, 2014. https://doi.org/10.3920/978-90-8686-767-7_27.
54. Powers, H. J. Interaction among Folate, Riboflavin, Genotype, and Cancer, with Reference to Colorectal and Cervical Cancer. *J. Nutr.* **2005**, *135* (12 Suppl), 2960S–2966S. <https://doi.org/10.1093/jn/135.12.2960S>.
55. Guéant, J.; Coelho, D.; Nicolas, J.–P. [Vitamin B12 and related genetic disorders]. *Bull. Acad. Natl. Med.* **2014**, *198* (6), 1141–1156.
56. Das, D. Vitamin B12 Gene Polymorphisms and Chronic Diseases. *J. Nutr. Disord. Ther.* **2014**, *04* (02). <https://doi.org/10.4172/2161-0509.1000149>.
57. Green, R.; Allen, L. H.; Bjørke–Monsen, A.–L.; Brito, A.; Guéant, J.–L.; Miller, J. W.; Molloy, A. M.; Nexø, E.; Stabler, S.; Toh, B.–H.; Ueland, P. M.; Yajnik, C. Vitamin B12 Deficiency. *Nat. Rev. Dis. Primer* **2017**, *3* (1), 1–20. <https://doi.org/10.1038/nrdp.2017.40>.
58. Agyemang–Yeboah, F.; Oppong, S. B–Vitamins Role in Cellular Metabolism and Clinical Nutrition; 2013.
59. Rennert, O. M. Genetic Defects of Cobalamin Metabolism. *Ann. Clin. Lab. Sci.* **1980**, *10* (4), 356–360.
60. Torshin, I. Yu.; Gromova, O. A. The Effectiveness of Biotin in Therapy Alopecia of Various Origins, Pathologies of the Skin and Nails. *Meditinskiy Sov. Med. Counc.* **2024**, No. 14, 135–144. <https://doi.org/10.21518/ms2024-296>.
61. Dasgupta, A. Chapter 3 – Biotin: From Supplement to Therapy. In *Biotin and Other Interferences in Immunoassays*; Dasgupta, A., Ed.; Elsevier, 2019; pp 37–49. <https://doi.org/10.1016/B978-0-12-816429-7.00003-4>.
62. Scott, W. Literature Review of Both Classic and Novel Roles of Biotin (Vitamin B7) in Cellular Processes; 2020.

63. Belda, E.; Volland, L.; Tremaroli, V.; Falony, G.; Adriouch, S.; Assmann, K. E.; Prifti, E.; Aron-Wisnewsky, J.; Debédat, J.; Le Roy, T.; Nielsen, T.; Amouyal, C.; André, S.; Andreelli, F.; Blüher, M.; Chakaroun, R.; Chilloux, J.; Coelho, L. P.; Dao, M. C.; Das, P.; Fellahi, S.; Forslund, S.; Galleron, N.; Hansen, T. H.; Holmes, B.; Ji, B.; Krogh Pedersen, H.; Le, P.; Le Chatelier, E.; Lewinter, C.; Mannerås-Holm, L.; Marquet, F.; Myridakis, A.; Pelloux, V.; Pons, N.; Quinquis, B.; Rouault, C.; Roume, H.; Salem, J.-E.; Sokolovska, N.; Søndertoft, N. B.; Touch, S.; Vieira-Silva, S.; The MetaCardis Consortium; Galan, P.; Holst, J.; Gøtzte, J. P.; Køber, L.; Vestergaard, H.; Hansen, T.; Hercberg, S.; Oppert, J.-M.; Nielsen, J.; Letunic, I.; Dumas, M.-E.; Stumvoll, M.; Pedersen, O. B.; Bork, P.; Ehrlich, S. D.; Zucker, J.-D.; Bäckhed, F.; Raes, J.; Clément, K. Impairment of Gut Microbial Biotin Metabolism and Host Biotin Status in Severe Obesity: Effect of Biotin and Prebiotic Supplementation on Improved Metabolism. *Gut* **2022**, *71* (12), 2463–2480. <https://doi.org/10.1136/gutjnl-2021-325753>.
64. Carling, R. S.; Turner, C. Methods for Assessment of Biotin (Vitamin B7). **2019**, 193–217. <https://doi.org/10.1016/B978-0-12-813050-6.00010-3>.
65. Garcia, M.; Leonardi, R.; Zhang, Y.-M.; Rehg, J. E.; Jackowski, S. Germline Deletion of Pantothenate Kinases 1 and 2 Reveals the Key Roles for CoA in Postnatal Metabolism. *PLoS ONE* **2012**, *7* (7), e40871. <https://doi.org/10.1371/journal.pone.0040871>.
66. Coxon, K. M.; Chakauya, E.; Ottenhof, H. H.; Whitney, H. M.; Blundell, T. L.; Abell, C.; Smith, A. G. Pantothenate Biosynthesis in Higher Plants. *Biochem. Soc. Trans.* **2005**, *33* (Pt 4), 743–746. <https://doi.org/10.1042/BSTO330743>.
67. Jansen, P. A. M.; van Diepen, J. A.; Ritzen, B.; Zeeuwen, P. L. J. M.; Cacciatore, I.; Cornacchia, C.; van Vlijmen-Willems, I. M. J. J.; de Heuvel, E.; Botman, P. N. M.; Blaauw, R. H.; Hermkens, P. H. H.; Rutjes, F. P. J. T.; Schalkwijk, J. Discovery of Small Molecule Vanin Inhibitors: New Tools to Study Metabolism and Disease. *ACS Chem. Biol.* **2013**, *8* (3), 530–534. <https://doi.org/10.1021/cb3006424>.
68. Ma, Q.; Liang, M.; Tang, X.; Luo, F.; Dou, C. Vitamin B5 Inhibit RANKL Induced Osteoclastogenesis and Ovariectomy Induced Osteoporosis by Scavenging ROS Generation. *Am. J. Transl. Res.* **2019**, *11* (8), 5008–5018.

69. Sampedro, A.; Rodríguez-Granger, J.; Ceballos, J.; Aliaga, L. PANTOTHENIC ACID: AN OVERVIEW FOCUSED ON MEDICAL ASPECTS. *Eur. Sci. J. ESJ* **2015**.
70. Terrell, K.; Choi, S.; Choi, S. Calcium's Role and Signaling in Aging Muscle, Cellular Senescence, and Mineral Interactions. *Int. J. Mol. Sci.* **2023**, *24* (23), 17034. <https://doi.org/10.3390/ijms242317034>.
71. Rajagopal, S.; Ponnusamy, M.; Rajagopal, S.; Ponnusamy, M. Regulation of Calcium in Muscle Physiology; Springer Singapore: Singapore, 2017; pp 15–30. https://doi.org/10.1007/978-981-10-5160-9_2.
72. Cashman, K. D. Calcium Intake, Calcium Bioavailability and Bone Health. *Br. J. Nutr.* **2002**, *87* (S2), S169–S177. <https://doi.org/10.1079/BJN/2002534>.
73. Li, G. H.-Y.; Robinson-Cohen, C.; Sahni, S.; Au, P. C.-M.; Tan, K. C.-B.; Kung, A. W.-C.; Cheung, C.-L. Association of Genetic Variants Related to Serum Calcium Levels with Reduced Bone Mineral Density. *J. Clin. Endocrinol. Metab.* **2020**, *105* (3), e328–336. <https://doi.org/10.1210/clinem/dgz088>.
74. Polimeni, P. I.; Page, E. Brief Reviews: Magnesium in Heart Muscle. *Circ. Res.* **1973**, *33* (4), 367–374. <https://doi.org/10.1161/01.RES.33.4.367>.
75. Larsson, S. C. Urinary Magnesium Excretion as a Marker of Heart Disease Risk. *Am. J. Clin. Nutr.* **2013**, *97* (6), 1159–1160. <https://doi.org/10.3945/ajcn.113.063354>.
76. Kirkland, A. E.; Sarlo, G. L.; Holton, K. F. The Role of Magnesium in Neurological Disorders. *Nutrients* **2018**, *10* (6), 730. <https://doi.org/10.3390/nu10060730>.
77. Kolte, D.; Vijayaraghavan, K.; Khera, S.; Sica, D. A.; Frishman, W. H. Role of Magnesium in Cardiovascular Diseases. *Cardiol. Rev.* **2014**, *22* (4), 182–192. <https://doi.org/10.1097/CRD.0000000000000003>.

78. Reinhold, D.; Ansorge, S.; Grüngreiff, K. Immunobiology of Zinc and Zinc Therapy. *Immunol. Today* **1999**, *20* (2), 102–103. [https://doi.org/10.1016/s0167-5699\(98\)01400-5](https://doi.org/10.1016/s0167-5699(98)01400-5).
79. Prasad, A. S. The Role of Zinc in Gastrointestinal and Liver Disease. *Clin. Gastroenterol.* **1983**, *12* (3), 713–741.
80. Lin, P.-H.; Sermersheim, M.; Li, H.; Lee, P. H. U.; Steinberg, S. M.; Ma, J. Zinc in Wound Healing Modulation. *Nutrients* **2017**, *10* (1), 16. <https://doi.org/10.3390/nu10010016>.
81. Patil, R.; Sontakke, T.; Biradar, A.; Nalage, D. Zinc: An Essential Trace Element for Human Health and Beyond. *Food Health* **2023**, *5* (3), 13. <https://doi.org/10.53388/FH2023013>.
82. Gheorghiu, M. L.; Badiu, C. Selenium Involvement in Mitochondrial Function in Thyroid Disorders. *Hormones* **2020**, *19* (1), 25–30. <https://doi.org/10.1007/s42000-020-00173-2>.
83. Rostami, R.; Nourooz-Zadeh, S.; Mohammadi, A.; Khalkhali, H. R.; Ferns, G.; Nourooz-Zadeh, J. Serum Selenium Status and Its Interrelationship with Serum Biomarkers of Thyroid Function and Antioxidant Defense in Hashimoto's Thyroiditis. *Antioxidants* **2020**, *9* (11), 1070. <https://doi.org/10.3390/antiox9111070>.
84. Kryczyk, J.; Zagrodzki, P. [Selenium in Graves' disease]. *Postepy Hig. Med. Doswiadczalnej Online* **2013**, *67*, 491–498. <https://doi.org/10.5604/17322693.1051000>.
85. Duntas, L. H. Selenium and At-Risk Pregnancy: Challenges and Controversies. *Thyroid Res.* **2020**, *13* (1), 16. <https://doi.org/10.1186/s13044-020-00090-x>.
86. Fox, P. L. The Copper-Iron Chronicles: The Story of an Intimate Relationship. *Biometals* **2003**, *16* (1), 9–40. <https://doi.org/10.1023/A:1020799512190>.
87. Collins, J. F.; Prohaska, J. R.; Knutson, M. D. Metabolic Crossroads of Iron and Copper. *Nutr. Rev.* **2010**, *68* (3), 133–147. <https://doi.org/10.1111/j.1753-4887.2010.00271.x>.

88. Jończy, A.; Mazgaj, R.; Starzyński, R. R.; Poznański, P.; Szudzik, M.; Smuda, E.; Kamyczek, M.; Lipiński, P. Relationship between Down-Regulation of Copper-Related Genes and Decreased Ferroportin Protein Level in the Duodenum of Iron-Deficient Piglets. *Nutrients* **2020**, *13* (1), 104. <https://doi.org/10.3390/nu13010104>.
89. Myint, Z. W.; Oo, T. H.; Thein, K. Z.; Tun, A. M.; Saeed, H. Copper Deficiency Anemia: Review Article. *Ann. Hematol.* **2018**, *97* (9), 1527–1534. <https://doi.org/10.1007/s00277-018-3407-5>.
90. Taskozhina, G.; Batyrova, G.; Umarova, G.; Issanguzhina, Z.; Kereyeva, N. The Manganese-Bone Connection: Investigating the Role of Manganese in Bone Health. *J. Clin. Med.* **2024**, *13* (16), 4679. <https://doi.org/10.3390/jcm13164679>.
91. Rondanelli, M.; Faliva, M. A.; Peroni, G.; Infantino, V.; Gasparri, C.; Iannello, G.; Perna, S.; Riva, A.; Petrangolini, G.; Tartara, A. Essentiality of Manganese for Bone Health: An Overview and Update. *Nat. Prod. Commun.* **2021**, *16* (5), 1934578X211016649. <https://doi.org/10.1177/1934578X211016649>.
92. Chakraborty, S.; Martinez-Finley, E.; Caito, S.; Chen, P.; Aschner, M. Manganese; Maret, W., Wedd, A., Eds.; The Royal Society of Chemistry, 2014; pp 260–281. <https://doi.org/10.1039/9781849739979-00260>.
93. Tong, S.-Y.; Lee, J.-M.; Song, E.-S.; Lee, K.-B.; Kim, M.-K.; Lee, J.-K.; Son, S.-K.; Lee, J.-P.; Kim, J.-H.; Kwon, Y.-I. Functional Polymorphism in Manganese Superoxide Dismutase and Antioxidant Status: Their Interactions on the Risk of Cervical Intraepithelial Neoplasia and Cervical Cancer. *Gynecol. Oncol.* **2009**, *115* (2), 272–276. <https://doi.org/10.1016/j.ygyno.2009.07.032>.
94. Hemmati, A. Assessment of the Serum Chromium Level in Patients with Type 2 Diabetes Mellitus; 2011.
95. Iskra, R.; Antonyak, H. Chromium in Health and Longevity; Malavolta, M., Mocchegiani, E., Eds.; Healthy Ageing and Longevity; Springer International Publishing: Cham, 2018; Vol. 8, pp 133–162. https://doi.org/10.1007/978-3-030-03742-0_5.

96. Rajendran, K. Serum Chromium Levels in Type 2 Diabetic Patients and Its Association with Glycaemic Control. *J. Clin. Diagn. Res.* **2015**.
<https://doi.org/10.7860/JCDR/2015/16062.6753>.
97. Oberleas, D.; Harland, B. The Physiological Role of Chromium in Diabetes, Type II. *FASEB J.* **2011**.
98. Kowey, P. R. The Role of Potassium; Lobo, R. A., Crosignani, P. G., Paoletti, R., Bruschi, F., Eds.; Medical Science Symposia Series; Springer US: Boston, MA, 2002; Vol. 17, pp 151–157. https://doi.org/10.1007/978-1-4615-1061-1_18.
99. Dursun, I.; Sahin, M. Difficulties in Maintaining Potassium Homeostasis in Patients with Heart Failure. *Clin. Cardiol.* **2007**, 29 (9), 388–392.
<https://doi.org/10.1002/clc.4960290904>.
100. Hager, N. A.; McAtee, C. K.; Lesko, M. A.; O'Donnell, A. F. Inwardly Rectifying Potassium Channel Kir2.1 and Its “Kir-lous” Regulation by Protein Trafficking and Roles in Development and Disease. *Front. Cell Dev. Biol.* **2021**, 9, 796136.
<https://doi.org/10.3389/fcell.2021.796136>.
101. Manville, R. W. Potassium -- The Essence of Life. *Sch. Sci. Rev.* **2019**.
102. Veeralakshmi, S. A Study on Nutrient Evaluation of Sesbania Grandiflora Flower Powder Incorporated in Soup Varieties. *ComFin Res.* **2024**.
103. Gomase, P. Sesbania Sesban Linn: A Review on Its Ethnobotany, Phytochemical and Pharmacological Profile. *Asian J. Biomed. Pharm. Sci.* **2012**.
104. Parab, N.; Wight, W. Determination of Some Trace Elements and Macro Minerals of Sesbania Bispinosa (Jacq.); 2016.
105. Bunma, S.; Balslev, H. A Review of the Economic Botany of Sesbania (Leguminosae). *Bot. Rev.* **2019**, 85 (3), 185–251. <https://doi.org/10.1007/s12229-019-09205-y>.

106. E, O. C.; Japhet, N. J.; Azuka, A. B.; Emmanuel, O. O. Phytochemical and Mineral Content in Flesh of Psidium Guajava Fruit. *Res. J. Phytochem.* **2022**, *16* (2), 88–96. <https://doi.org/10.3923/rjphyto.2022.88.96>.
107. Angulo–López, J. E.; Flores–Gallegos, A. C.; Torres–León, C.; Ramírez–Guzmán, K. N.; Martínez, G. A.; Aguilar, C. N. Guava (*Psidium Guajava* L.) Fruit and Valorization of Industrialization By–Products. *Processes* **2021**, *9* (6), 1075. <https://doi.org/10.3390/pr9061075>.
108. Tousif, M. I.; Nazir, M.; Saleem, M.; Tauseef, S.; Shafiq, N.; Hassan, L.; Hussian, H.; Montesano, D.; Naviglio, D.; Zengin, G.; Ahmad, I. *Psidium Guajava* L. An Incalculable but Underexplored Food Crop: Its Phytochemistry, Ethnopharmacology, and Industrial Applications. *Mol. Basel Switz.* **2022**, *27* (20), 7016. <https://doi.org/10.3390/molecules27207016>.
109. Jamieson, S.; Wallace, C. E.; Das, N.; Bhattacharyya, P.; Bishayee, A. Guava (*Psidium Guajava* L.): A Glorious Plant with Cancer Preventive and Therapeutic Potential. *Crit. Rev. Food Sci. Nutr.* **2023**, *63* (2), 192–223. <https://doi.org/10.1080/10408398.2021.1945531>.
110. Prananda, A. T.; Dalimunthe, A.; Harahap, U.; Simanjuntak, Y.; Peronika, E.; Karosekali, N. E.; Hasibuan, P. A. Z.; Syahputra, R. A.; Situmorang, P. C.; Nurkolis, F. *Phyllanthus Emblica*: A Comprehensive Review of Its Phytochemical Composition and Pharmacological Properties. *Front. Pharmacol.* **2023**, *14*, 1288618. <https://doi.org/10.3389/fphar.2023.1288618>.
111. Mahajan, S.; Bisht, M. S.; Chakraborty, A.; Sharma, V. K. Genome of *Phyllanthus Emblica*: The Medicinal Plant Amla with Super Antioxidant Properties. *Front. Plant Sci.* **2023**, *14*, 1210078. <https://doi.org/10.3389/fpls.2023.1210078>.
112. S, T. S.; Suryawanshi, P. P.; Mane, P.; Dappadwad, P.; Sangle, R.; Lahade, R.; Tandle, R.; Bedre, R. Pharmacological and Medicinal Important of Plant *Phyllanthus Emblica* Linn. (*Syn. Emblica Officinalis*), Indian Gooseberry. *Int. J. Res. Appl. Sci. Eng. Technol.* **2023**, *11* (10), 1473–1480. <https://doi.org/10.22214/ijraset.2023.56206>.

113. Singh, E.; Sharma, S.; Pareek, A.; Dwivedi, J.; Yadav, S.; Sharma, S. Phytochemistry, Traditional Uses and Cancer Chemopreventive Activity of Amla (*Phyllanthus Emblica*): The Sustainer. *J. Appl. Pharm. Sci.* **2012**, : (Issue), 176–183.
114. Mahajan, N.; Singh, J.; Sinha, S. Research Article COMPARISON OF TOTAL FLAVONOID, PHENOLIC CONTENT AND ANTIOXIDANT CAPACITY IN LEAF AND SEED EXTRACTS FROM WHITE HOLY BASIL (*Ocimum Sanctum*); 2014.
115. Singh, D.; Chaudhuri, P. K. A Review on Phytochemical and Pharmacological Properties of Holy Basil (*Ocimum Sanctum* L.). *Ind. Crops Prod.* **2018**, *118*, 367–382. <https://doi.org/10.1016/j.indcrop.2018.03.048>.
116. Vidhani, S. I.; G.Vyas, V.; Parmar, H.; Bhalani, V. M.; Hassan, M.; Gaber, A.; A.Golakiya, B. Evaluation of Some Chemical Composition, Minerals Fatty Acid Profiles, Antioxidant and Antimicrobial Activities of Tulsi (*Ocimum Sanctum*) from India. *Am. J. Food Sci. Technol.* **2016**.
117. Baliga, M. S.; Jimmy, R.; Thilakchand, K. R.; Sunitha, V.; Bhat, N. R.; Saldanha, E.; Rao, S.; Rao, P.; Arora, R.; Palatty, P. L. *Ocimum Sanctum* L (Holy Basil or Tulsi) and Its Phytochemicals in the Prevention and Treatment of Cancer. *Nutr. Cancer* **2013**, *65* (sup1), 26–35. <https://doi.org/10.1080/01635581.2013.785010>.
118. Raddatz-Mota, D.; Pérez-Flores, L. J.; Carrari, F.; Mendoza-Espinoza, J. A.; de León-Sánchez, F. D.; Pinzón-López, L. L.; Godoy-Hernández, G.; Rivera-Cabrera, F. Achiote (*Bixa Orellana* L.): A Natural Source of Pigment and Vitamin E. *J. Food Sci. Technol.* **2017**, *54* (6), 1729–1741. <https://doi.org/10.1007/s13197-017-2579-7>.
119. Zarza-García, A. L.; Moo-Huchín, V. M.; Toledo-López, V. M.; Godoy-Hernández, G.; Rivera-Cabrera, F.; Aarland, R. C.; Sauri-Duch, E.; Mendoza-Espinoza, J. A. Chemical, Nutritional, and Biological Composition of Three Seed Morphotypes of *Bixa Orellana* L. Bixaceae (Achiote) in the Yucatan Peninsula, Mexico. *Pak. J. Bot.* **2021**, *53* (6). [https://doi.org/10.30848/PJB2021-6\(12\)](https://doi.org/10.30848/PJB2021-6(12)).

120. Wurts, M.; Torreblanca, R. [Analysis of the Seed Bixa Orellana, L. (Annatto) and the Waste Generated in the Extraction of Its Pigments]. *Arch. Latinoam. Nutr.* **1983**.
121. Elias, M. E. A.; Schroth, G.; Macêdo, J. L. V.; Mota, M. S. S.; D'Angelo, S. A. MINERAL NUTRITION, GROWTH AND YIELDS OF ANNATTO TREES (BIXA ORELLANA) IN AGROFORESTRY ON AN AMAZONIAN FERRALSOL. *Exp. Agric.* **2002**, *38* (3), 277–289. <https://doi.org/10.1017/S0014479702003034>.
122. González–Molina, E.; Moreno, D. A.; García–Viguera, C. Genotype and Harvest Time Influence the Phytochemical Quality of Fino Lemon Juice (Citrus Limon (L.) Burm. F.) for Industrial Use. *J. Agric. Food Chem.* **2008**, *56* (5), 1669–1675. <https://doi.org/10.1021/jf073282w>.
123. Hashemipour, M.; Kargar, M.; Ghannadi, A.; Kelishadi, R. The Effect of Citrus Aurantifolia (Lemon) Peels on Cardiometabolic Risk Factors and Markers of Endothelial Function in Adolescents with Excess Weight: A Triple–Masked Randomized Controlled Trial. *Med. J. Islam. Repub. Iran* **2016**.
124. Qurban, F.; Hussain, S.; Waqas, M.; Hadiya Shahzad, H.; Rukhsar, A.; Javed, A. Phytochemistry, Nutritional, and Pharmacological Potential of Citrus Limonum. *Sci. Inq. Rev.* **2024**, *8* (3), 1–23. <https://doi.org/10.32350/sir.83.01>.
125. Valgimigli, L.; Gabbanini, S.; Berlini, E.; Lucchi, E.; Beltramini, C.; Bertarelli, Y. L. Lemon (*Citrus Limon*, Burm.f.) Essential Oil Enhances the Trans-epidermal Release of Lipid- (A, E) and Water- (B6 , C) Soluble Vitamins from Topical Emulsions in Reconstructed Human Epidermis. *Int. J. Cosmet. Sci.* **2012**, *34* (4), 347–356. <https://doi.org/10.1111/j.1468-2494.2012.00725.x>.
126. Xia, Z.; Shang, H.–Y.; Wang, Q.–J.; Zhao, Y.; Ding, X.–M. [The role and mechanism of leucyl–tRNA synthetase in the regulation of protein synthesis in aging skeletal muscle]. *Sheng Li Xue Bao* **2020**, *72* (4), 523–531.

127. Zhao, Y.; Cholewa, J.; Shang, H.; Yang, Y.; Ding, X.; Wang, Q.; Su, Q.; Zanchi, N. E.; Xia, Z. Advances in the Role of Leucine–Sensing in the Regulation of Protein Synthesis in Aging Skeletal Muscle. *Front. Cell Dev. Biol.* **2021**, *9*, 646482. <https://doi.org/10.3389/fcell.2021.646482>.
128. Duan, Y.; Li, F.; Liu, H.; Li, Y.; Liu, Y.; Kong, X.; Zhang, Y.; Deng, D.; Tang, Y.; Feng, Z.; Wu, G.; Yin, Y. Nutritional and Regulatory Roles of Leucine in Muscle Growth and Fat Reduction. *Front. Biosci. Landmark Ed.* **2015**, *20* (4), 796–813. <https://doi.org/10.2741/4338>.
129. Garlick, P. J. The Role of Leucine in the Regulation of Protein Metabolism. *J. Nutr.* **2005**, *135* (6 Suppl), 1553S–6S. <https://doi.org/10.1093/jn/135.6.1553S>.
130. Patil, U.; Singh, A.; Chakraborty, A. Role of Piperine As A Bioavailability Enhancer; 2011.
131. Sarangi, B.; Jana, U.; Mohanta, G. P.; Manna, P. K. PIPERINE: A NATURAL BIOENHANCER. *INDIAN DRUGS* **2017**, *54* (06), 5–19. <https://doi.org/10.53879/id.54.06.10905>.
132. Singh, A.; Deep, A. Piperine : A Bioenhancer. *Int. J. Pharm. Res. Technol.* **2019**. <https://doi.org/10.31838/ijprt/01.01.01>.
133. Han, H.-K. The Effects of Black Pepper on the Intestinal Absorption and Hepatic Metabolism of Drugs. *Expert Opin. Drug Metab. Toxicol.* **2011**, *7* (6), 721–729. <https://doi.org/10.1517/17425255.2011.570332>.