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Review Article

Evaluating Vitamins & Minerals & Their Effect on Your Biological System When Taken in Multivitamin

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Abstract

This thesis aims to explore the intricate interactions, reactions, and counteractions of vitamins within the human body. Vitamins are essential organic compounds required in small quantities for the proper functioning of various physiological processes. While each vitamin plays a unique role, their interdependencies and potential for interactions are crucial to understand. This thesis examines the mechanisms behind vitamin interactions, including absorption, metabolism, and potential antagonistic or synergistic effects. By delving into these complexities, this research seeks to contribute to a comprehensive understanding of how vitamins interact, react, and counteract with each other, and their implications for human health.

Keywords: Vitamin Interactions, Absorption, Metabolism, Synergistic Effects, Antagonistic Effects.

INTRODUCTION

Vitamins are essential micronutrients required for various biological processes, including metabolism, growth, and immune function. While they are often studied and consumed individually, vitamins can also influence each other's absorption, metabolism, and overall efficacy. The interactions between vitamins can be complex and multifaceted, leading to synergistic, antagonistic, or counteractive effects. This thesis aims to shed light on the interrelationships of vitamins and explore the mechanisms behind their interactions, reactions, and counteractions.

Absorption and Metabolism:

Vitamin absorption and metabolism are fundamental processes that determine their availability and utilization within the body. Several vitamins share similar absorption pathways, leading to potential competition for uptake. For instance, vitamins A, D, E, and K are fat-soluble and require dietary fats for optimal absorption. Consequently, a deficiency in dietary fats may impair the absorption of these vitamins. Similarly, vitamin C can enhance the absorption of non-heme iron, thereby increasing iron bioavailability.

Interactions:

Vitamin interactions can occur at various stages, including absorption, transport, metabolism, and cellular utilization. For example, vitamin D enhances the absorption of calcium, facilitating bone health. Conversely, excessive vitamin D intake can lead to increased calcium levels, potentially resulting in adverse effects such as kidney stones. Vitamin E is known for its antioxidant properties, which can protect other vitamins, such as vitamin A, from oxidation. Additionally, vitamins C and E work synergistically, regenerating each other and maximizing their antioxidant potential.

Reactions:

Vitamin reactions refer to chemical changes that occur when vitamins interact, potentially altering their individual properties or functions. Some vitamins, such as vitamin C, act as reducing agents, donating electrons to maintain the stability of other vitamins. Vitamin C can react with vitamin E radicals, regenerating the antioxidant form of vitamin E. Similarly, vitamins B2, B3, and B6 participate in redox reactions, contributing to energy metabolism and supporting the antioxidant function of other vitamins.

Antagonistic Effects:

Certain vitamin interactions can lead to antagonistic effects, where one vitamin interferes with the absorption, metabolism, or function of another. For example, excessive vitamin C intake may impair the absorption of vitamin B12, leading to a deficiency. High doses of vitamin A can antagonize vitamin D activity, reducing its effectiveness in regulating calcium metabolism. Understanding these antagonistic effects is crucial to avoid unintended consequences of excessive vitamin supplementation.

Synergistic Effects:

In contrast to antagonistic effects, vitamins can also exhibit synergistic interactions, where the combined effect is greater than the sum of their individual effects. Vitamin D and calcium exemplify such synergy, working together to maintain bone health. Vitamin C enhances the absorption of non-heme iron, while iron, in turn, improves the absorption of vitamin C. These synergistic interactions underscore the importance of a well-balanced diet to optimize vitamin utilization and overall health.

Counteractions:

Counteractions refer to the ability of one vitamin to counteract or mitigate the adverse effects of another. For instance, vitamin E acts as an antioxidant, neutralizing free radicals and protecting against oxidative damage. This counter extends to vitamin A, as vitamin E can prevent the oxidation of retinol, the active form of vitamin A. Additionally, vitamin C can counteract the potential pro-oxidant effects of high-dose vitamin E supplementation by regenerating its antioxidant form. These counteractions emphasize the intricate balance required for optimal vitamin interactions within the body.

Implications for Human Health:

Understanding the interactions, reactions, and counteractions of vitamins is essential for promoting human health and preventing nutrient imbalances. Imbalances or deficiencies in one vitamin can affect the absorption, metabolism, or function of others, leading to adverse health outcomes. For example, vitamin D deficiency can impair calcium absorption and contribute to bone disorders such as osteoporosis. Similarly, a deficiency in vitamin B12 can result in reduced folate activation, leading to anemia and neural tube defects. By considering the complex relationships between vitamins, healthcare professionals can develop targeted interventions to address specific nutrient deficiencies and optimize overall health. Multivitamins are popular dietary supplements that typically contain a combination of vitamins and minerals. While the idea of getting a variety of nutrients in a single supplement may seem appealing, research suggests that mixing vitamins and minerals together in a multivitamin can actually be counterproductive.

The reason for this is that some vitamins and minerals compete with each other for absorption in the body. For example, calcium and iron can interfere with each other's absorption, as can zinc and copper. Additionally, some vitamins and minerals can interact with each other in ways that may be harmful. For example, high levels of vitamin A can interfere with vitamin D, which is essential for bone health.

Vitamins and minerals are essential elements that the human biological system needs for proper development & normal functionality. Vitamins include substances such as A, C, D, E, K, and the B complex: thiamin (B1), riboflavin (B2), niacin (B3), pantothenic acid (B5), pyridoxal (B6), cobalamin (B12), biotin and folate. Essential minerals include calcium, phosphorus, potassium, sodium, chloride, magnesium, iron, zinc, iodine, sulfur, cobalt, copper, fluoride, manganese, and selenium.

Despite the fact that our biological system needs these essential nutrients for optimal health, education, especially within the integrative & alternative medicine fields, regarding how these nutrients affect the biological system, particularly when they interact with each other, or when used in excess or in deficient amounts, is lacking & sometimes deceptive & irresponsible.

In my professional opinion, most(not all) over the counter(OTC), store bought and internet sold vitamins and minerals(as well as herbal supplements should be restricted to compliant dispensaries that only release such supplements to the patient after they are prescribed by qualified board certified and/or licensed healthcare professionals who have a solid background in "Medical Nutrition, Orthomolecular Medicine or is a Naturopathic or Homeopathic Physician, Midwife or Doctor of Integrative Medicine, and only after mandatory vitamin & nutrient CLIA certified lab testing have been performed.

Vitamins and minerals fall under "Chemistry and Biochemistry" because they are dealing with metals (i.e. Alchemy), and unless one understands advanced Chemistry and how metals interact with each other & how they interact with our biological system, then even recommend or use any vitamin or mineral supplement can have long-term consequences on our biological system.

Another concern is that multivitamins often contain high doses of nutrients, which can be problematic for some people. Excessive intake of certain vitamins and minerals can lead to toxicity and other health problems. For example, excessive intake of vitamin A can cause liver damage, while excessive intake of iron can lead to gastrointestinal distress and even organ damage.

For these reasons, it is generally recommended that individuals obtain their nutrients from a balanced and varied diet, rather than relying on multivitamin supplements. In cases where nutrient deficiencies are suspected, it may be more appropriate to take individual supplements in targeted doses, rather than relying on a multivitamin.

My conclusion is intended to show that while the idea of a one-stop-shop for all your daily nutritional needs may be appealing, mixing vitamins and minerals together in a multivitamin can lead to interactions and other problems. As with any dietary supplement, it is important to discuss the use of multivitamins with a healthcare provider to determine if they are appropriate for your individual need this review will give further detail of how some of the common vitamins and minerals interact with each other and what kind of impact these nutrients have on the biological system and overall health of individual when taken improperly, such as in multivitamin form.

Vitamins & Minerals-Benefits, Interactions and Risks

The B-plexes, comprising eight essential micronutrients (B1, B2, B3, B5, B6, B7, B9, and B12), play critical roles in energy metabolism, neurological function, and overall health. Deficiencies in these vitamins are associated with severe health and psychological conditions, including anemia, neuropathy, and cognitive impairment. Over-the-counter (OTC) dietary supplements are widely used to prevent or treat such deficiencies, yet concerns persist regarding the quality, safety, and efficacy of these products

B12- Vitamin B12 is a water-soluble red crystalline vitamin of the "B" group. It is not a specific compound, rather a group of related compounds. The 'B' vitamins are a group of Water-Soluble Vitamins so-named because they are commonly found together in foods and often share similar functions as coenzymes for various processes within the body.

Cobalamin:

Cobalt is an integral component of the Vitamin B12 molecule - many of the biological functions of Vitamin B12 are due to its Cobalt content. It is the Cobalt content of the Vitamin B12 molecule that facilitates the synthesis of endogenous Deoxyribonucleic Acid (DNA)

Calcium may enhance the absorption of Vitamin B12:

- Supplemental Calcium prevents Metformin-induced Vitamin B12 deficiency.
- Excessively large dosages of supplemental Vitamin B2 may cause the excretion of other B Vitamins via the Urine.
- Folate (as opposed to Folic Acid) may be synergistic with Vitamin B12.
- Vitamin B1 may enhance the function of Vitamin B12.
- Vitamin B3 may be synergistic with Vitamin B12.
- Vitamin B6 may enhance the function of Vitamin B12.
- Vitamin C may enhance the function of Vitamin B12.
- Vitamin E may be essential for the conversion of Vitamin B12 to its most active form.
- Methionine may be essential for the utilization of Vitamin B12
- High-dose Vitamin C (>2000 mg/day) may reduce B12 bioavailability (Simon *et al*., 1999).

Risks:

Synthetic cyanocobalamin may accumulate as a heavy metal if not converted to active forms. B12 deficiency, common in vegans and older adults, can cause anemia and neuropathy.

Dosage: 2.4–1000 mcg/day; monitor levels in high-dose supplementation (>500 mcg/day).

The synthetic/fermented derived B12 is known as cyanocobalamin. This is usually found in most lower quality OTC multi-vitamins sold in local stores & online. It also has a chemistry cyanide molecule attached to a cobalamin molecule (cobalt). While it must be converted to either of the active Coenzyme forms within the body in order to exert therapeutic effects, it in fact is not the most compatible with the human biological system and does not assimilate into the bodies cells(digested) and thus can accumulates in the body as a heavy metal.

- The Liver converts approximately 1% of Cyanocobalamin to the Methylcobalamin form of Vitamin B12. Most supplemental Cyanocobalamin is converted within the Intestines to Adenosylcobalamin. Thus it has no therapeutic effect.
- Cobalamin (Cbl) is a general term for all Vitamin B12-like compounds (i.e. compounds that contain the Dimethyl benzimidazolyl cobamide nucleus that characterizes Vitamin B12).
- Adenosylcobalamin, Methylcobalamin & Hydroxocobalamin forms of supplemental Vitamin B12 are compatible with & therapeutic to our biological system.
- Adenosylcobalamin (also known as ado-Cbl; Cobamamide; Coenzyme B12; Deoxyadenosylcobalamin; Dibencozide; Dimethyl Benzimidazole-cobamide Coenzyme) is an active endogenous coenzyme form of Vitamin B12 that helps to maintain the correct Fatty Acids in Myelin, the fatty sheath surrounding Nerves. Adenosylcobalamin is formed endogenously and its impaired biosynthesis can lead to a condition known as Methylmalonic Academia. Approximately 70% of the Liver's Vitamin B12 reserves are in the

form of Adenosylcobalamin. Although some manufacturers produce this form of Vitamin B12 in supplemental form, it is more expensive than Cyanocobalamin. Most supplemental Cyanocobalamin is converted within the Intestines to Adenosylcobalamin.

- Hydroxocobalamin (also known as Hydroxocobemine; Hydroxycobalamin; Vitamin B12b) is the ideal form of supplemental Vitamin B12 (due to it remaining in circulation for longer than other forms of Vitamin B12). This form is only administered via intramuscular (IM) injection. Hydroxocobalamin differs from Cyanocobalamin by the presence of a Hydroxyl Ion in place of the Cyanide Ion.
- Mecobalamin is a form of Vitamin B12 that is suitable for intravenous injection (by a physician).
- Methylcobalamin (also known as met-Cbl; MeCbl; Methyl-B12) is an active endogenous Coenzyme form of Vitamin B12 that transfers a Methyl group from an inactive form of Folic Acid to Homocysteine forming Methionine. This is the only active endogenous coenzyme form of Vitamin B12 that is available as a supplement (in tablet form). Methylcobalamin accounts for approximately 70% of the total Blood Plasma Vitamin B12 reserves. Clinical trials have shown that supplemental Methylcobalamin is greatly superior to other forms of Vitamin B12 supplements in terms of its ability to enhance human health.

Folic Acid(B9) is a water-soluble Vitamin of the "B" Group that is actually a Vitamin within a Vitamin because its chemical structure contains Pteridine, PABA and Glutamic Acid. Chemically, Folic Acid is pteroylglutamic acid, composed of a Pterin, Para-Aminobenzoic Acid (PABA), and Glutamic Acid moieties. Folic acid, a synthetic man-made version of Folate, used in B-plexes, are in fact contra-indicated in its negative interactions with B12.

- Folic acid, particularly in doses of 500mcg or higher can mask vitamin B12 deficiency by completely correcting hematological abnormalities. In vitamin B12 deficiency, folic acid can produce complete resolution of the characteristic megaloblastic anemia, while allowing potentially irreversible neurological damage (from continued inactivity of methylmalonyl mutase) to progress.
- Thus, vitamin B12 status should be determined before folic acid is given as monotherapy.
- Folinic Acid, the best form, and Folic Acid are NOT the same. Synthetic Folic Acid is not as healthy, and yet this is one of the most common prescribed B vitamins given to people with Sickle Cell Disease. Yet Heart Disease and Heart Attacks remain as one of the leading causes of death in sickle cell patients....
- Folic acid may reduce the bodies ability to absorb and retain Zinc in some studies.

Folic Acid in Sickle Cell and Cardiovascular Disease

There is some concern that taking too much folic acid for a long period of time might cause serious side effects. Some research suggests that taking folic acid in doses of 800-1200 mcg might increase the risk of heart attack in people who have heart problems. Other research suggests that taking these high doses might also increase the risk of cancer such as lung or prostate cancer. Yet the Sickle Cell Foundation and most Hematologists recommend sickle cell patients take 800-1000mcg of Folic acid daily. This could be the explanation of the heart attacks that is common with sickle cell deaths.

There is some concern that green tea might keep folic acid/Folate from working the way it should in the body. This might lead to a condition that is similar to folic acid deficiency. Daily intake of tea is known to cause "anemia".

Folic acid increases homocysteine because folic acid is not the body's primary form of folate – l-methylfolate is folic acid isn't active at all in the human body until it is transformed by a gene called DHFR. the folic acid transforming gene, DHFR, is extremely slow. Since this DHFR gene is very slow, folic acid just sits in your blood stream standing in the way of l-methylfolate so l-methylfolate which then cannot do its job. Thus, if l-methylfolate cannot do its job, homocysteine goes up.

High doses of folic acid might cause abdominal cramps, diarrhea, rash, sleep disorders, irritability, confusion, nausea, stomach upset, behavior changes, skin reactions, seizures, gas, excitability, and other side effects.

Homocysteine:

Is a toxic endogenous, sulphydryl-containing Amino Acid - a homologue of Cysteine. Studies show that elevated Homocysteine levels may be an underlying cause of Sickle- Cell Anemia (Sickle-Cell Anemia patients have been found to have significantly higher Homocysteine levels compared with normal, healthy persons). Thus, allopathic doctors prescribing folic acid, as opposed to folate, are contributing to increasing the risk of detrimental or even fatal health consequences of sickle cell patients.

Excessive Homocysteine may cause abnormal Blood Clotting (by inhibiting the production of Tissue Plasminogen Activator).

- Excessive Homocysteine levels may cause many forms of Cardiovascular Disease (excess Homocysteine correlates with the occurrence of

Cardiovascular Diseases more closely than Cholesterol):

- Elevated Homocysteine levels may cause Aneurysm.
- Elevated Homocysteine may cause Atherosclerosis.
- Homocysteine may facilitate the deposition of Cholesterol around the Heart Muscle.
- Intermittent Claudication
- Elevated Homocysteine levels may increase the risk of Ischemic Heart Disease including:
- Angina
- Excessive Homocysteine levels may increase the risk of Heart Attack - it has been estimated that approximately 40% of all Heart Attacks are caused by elevated Homocysteine levels.
- Thrombosis (including Deep Vein Thrombosis)
- Excessive Homocysteine levels may be a primary underlying cause of many

Cerebrovascular Diseases:

- Excessive Homocysteine levels may cause Stroke - it has been estimated that approximately 40% of all Strokes are caused by elevated Homocysteine levels references.
- Elevated Homocysteine levels may increase the risk of Preeclampsia.

Folate, L-Methylfolate Calcium = Metafolin = Levomefolic Acid & Folinic acid = Calcium folinate has the opposite effect than Folic acid and is beneficial to the biological system. It reduces Homocysteine levels & protects the cardiovascular system. Recent research has shown that Folinic Acid may be the optimal form of supplemental Folic Acid. It bypasses the deconjugation and reduction steps required for standard Folic Acid and is more metabolically active - it is capable of boosting levels of Folic Acid in circumstances where regular Folic Acid has little effect.

B1 (Thiamine)- water-soluble Vitamin of the "B" Group of Vitamins

- Vitamin B1 is an essential cofactor for the conversion of dietary Tryptophan to Picolinic Acid (an isomer of the Nicotinic Acid form of Vitamin B3).
- Vitamin B1 enhances the function of Vitamin B12.
- Excessive levels of Copper may cause a reduction in the body's Vitamin B1 levels

Forms of B1:

- Benfotiamine is a pharmaceutical, fat-soluble analogue of Vitamin B1. Its bioavailability is 3.6 times higher than that of Thiamine Hydrochloride.
- Fursultiamine (also known as: Allithiamin; Allithiamine; Thiamine Tetrahydrofurfuryl Disulfide; TTFD) is an analogue of Vitamin B1 that is highly fat- soluble and is therefore not subject to rate-limiting active transport systems associated with water-soluble forms of Vitamin B1. This means that larger quantities of this form can be absorbed by the body compared to water-soluble forms of Vitamin B1.
- Sulbutiamine (also known as Arcalion; Enerion; Surmenalit; Sulbuthiamine) is an analogue of Vitamin B1 that is highly fat-soluble and is therefore not subject to rate-limiting active transport systems associated with water-soluble forms of Vitamin B1.
- Thiamine Diphosphate (also known as Cocarboxylase; Thiamin Diphosphate; Thiamin Pyrophosphate; Thiamine Pyrophosphate; TDP; TPP) is a biologically active endogenous Coenzyme form of Vitamin B1.
- Thiamine Hydrochloride (Thiamine HCl) consists of 89% Thiamine and 11% Hydrochloric Acid (HCl) and is the most common form of Vitamin B1 used in Vitamin B1 supplements.
- Thiamine Monophosphate (also known as Thiamine Phosphate; TMP) is an endogenous form of Vitamin B1.
- Thiamine Nitrate (also known as Thiamine Mononitrate) is a form of Vitamin B1 used in some Vitamin B1 supplements.
- Thiamine Propyl Disulfide is an easily absorbed form of Vitamin B1.

B2(Riboflavin)- a water-soluble orange-yellow crystal of the "B" Vitamin group.

- Vitamin B2 may enhance the body's absorption of Iron.
- Vitamin B2 may reduce the body's urinary excretion of Selenium and may thereby facilitate the retention of Selenium.
- Vitamin B2 is required for the synthesis of active forms of Folic Acid.
- Vitamin B2 may enhance the absorption of Vitamin B6: Vitamin B2 is an essential cofactor for the conversion of dietary Vitamin B6 (e.g. Pyridoxine) to its active endogenous form (Pyridoxal-5-Phosphate).
- Vitamin B2 may protect against the neurotoxic effects of Glutamic Acid.
- Phosphorus is involved in the conversion of exogenous Vitamin B2 to its active endogenous coenzyme form.
- Biotin may enhance the production of Vitamin B2 within the body.
- Vitamin C may protect Vitamin B2 from oxidation.
- Vitamin B2 may play a role in lowering Homocysteine levels (it is a cofactor for the enzyme that catalyzes the production of a specific form of Folic Acid required for the

remethylation of Homocysteine to form Methionine).

- Excessive consumption of Boron may cause excessive excretion of Vitamin B2.
- Boric Acid may bind to and prevent the absorption of Vitamin B2 and may increase the urinary excretion of Vitamin B2.

B3 (Niacin):

Acidic water-soluble Vitamin from the "B" group of Vitamins. In the USA the term Niacin usually refers specifically to the Nicotinic Acid form of Vitamin B3. Nicotinic Acid is a specific form of Vitamin B3. Nicotinic Acid supplements are manufactured by oxidizing Nicotine

- This is not a cause for alarm as Nicotinic Acid is not associated with any of the toxic effects attributable to Nicotine

Phosphorus is involved in the conversion of exogenous Vitamin B3 to its active endogenous coenzyme form.

- Biotin may enhance the function of Vitamin B3.
- Inositol may stimulate Niacinamide to bind to the Benzodiazepine Receptors in the Brain.
- Vitamin B12 may enhance the function of Vitamin B3.
- The Niacinamide form of Vitamin B3 may increase Choline levels in the Brain.
- A Vitamin B3 derivative (NADPH) is a cofactor for the conversion of dietary Folate to its active form - Tetrahydrofolic Acid.
- Niacinamide (the other B3) can be converted within the body to Nicotinic Acid (however Nicotinic Acid cannot be reconverted back to Niacinamide).
- If Nicotinic Acid is continually consumed on an empty stomach, it may cause the release of excessive Hydrochloric Acid within the Stomach - which may increase the risk of Peptic
- Ulcers
- Nicotinic Acid may cause Erythema (red flushing of the Skin) - especially when it is consumed on an empty Stomach (this side effect is generally harmless - it occurs as a result of dilation of the Capillaries that service the Skin and usually only persists for a period of one hour maximum). The Erythema caused by Nicotinic Acid consumption occurs as a result of Prostaglandin D2 release stimulated by Nicotinic Acid.
- Arrhythmias patients should consult a doctor before using the Nicotinic Acid form of Vitamin B3
- Vitamin B3 may occasionally worsen some Skin Allergies
- Excessive consumption of Nicotinic Acid may increase Homocysteine levels

Forms of B3

- Niacytin is a bound and unavailable form of Vitamin B3 present in some foods, especially Cereals and Grains.
- Niacinamide (also known as Nicotinamide) is the main form of Vitamin B3 present in dietary sources. Chemically, it is the Amide of Nicotinic Acid.
- Nicotinic Acid (Niacin) is the acid form of Vitamin B3.
- Xanthinol Nicotinate is a synthetic form of the Nicotinic Acid form of Vitamin B3 that can pass easily through Cell Membranes into Cells much more readily than Niacin

Forms of Nicotinic Acid:

Inositol Hexanicotinate (also known as Hexanicotinoyl Inositol, IHN, Inositol Hexaniacinate or Inositol Niacinate; Inositol Nicotinate) consists of one molecule of Inositol bound to six molecules of Nicotinic Acid (by weight, 10% Inositol +

90% Nicotinic Acid). Chemically, it is the hexanicotinic acid ester of Inositol. Inositol Hexanicotinate is absorbed intact and then hydrolzyed within the body to release "free" Nicotinic Acid and Inositol. Inositol Hexanicotinate is metabolized more slowly than "free" Nicotinic Acid - it does not reach maximum Blood Serum levels until approximately 10 hours after its oral ingestion:

- This form of Nicotinic Acid is primarily used as a non-flushing, sustained- release form of Nicotinic Acid and is regarded by some experts as the optimal form of Nicotinic Acid. The side effects associated with regular Nicotinic Acid do not occur with Inositol Hexanicotinate.
- Inositol Hexanicotinate is regarded as the safest form of supplemental Nicotinic Acid and can be safely used in doses of up to 4,000 mg per day.
- Picolinic Acid is an isomer of Nicotinic Acid (but which possesses different therapeutic properties to Nicotinic Acid).
- Xanthinol Nicotinate is a synthetic form of Nicotinic Acid consisting of Nicotinic Acid bound to Xanthinol. It is classed as a Pharmaceutical Drug and as a Smart Drug.

B5 (Pantothenic Acid)- a water soluble Organic Acid that is present in all living Cells.

- Vitamin B5 is an essential cofactor for the conversion of Choline to Acetylcholine.
- PABA (Para Aminobenzoic Acid) may improve the utilization of Vitamin B5.
- Vitamin C may protect Vitamin B5 from oxidation.
- Vitamin B5 is an enemy of Taurine(Taurine is a non-essential Sulfur-Containing Amino Acid. It functions independently within the body (i.e. it is not incorporated into Proteins). It is

manufactured endogenously within the Liver from Cysteine and Methionin

Forms of Vitamin B5

- Calcium Pantothenate is the most prevalent form of supplemental Vitamin B5. It is comprised of 91.5% Pantothenic Acid + 7.5% Calcium.
- d-Pantothenic Acid is a natural form of Vitamin B5 and the most common form of Vitamin B5 that is available in supplements (usually as Calcium Pantothenate).
- Dexpanthenol is an Alcohol form of Pantothenic Acid. It is the active ingredient in some (topically-applied) cosmetics and toiletries.
- Pantetheine is the condensation product of Pantothenic Acid and Aminoethanethiol - it is an intermediate product in the endogenous biosynthesis of Coenzyme A.
- Pantethine is a natural form of Vitamin B5 that is regarded by some experts as the superior form of this Vitamin (it possesses superior Cell Respiration qualities above Pantothenic Acid).
- Panthothenyl is the Acyl Radical of Pantothenic Acid.
- Pantoyl is the Acyl Radical of Pantoic Acid.
- Pantoyltaurine (also known as: Thiopanic Acid) is Pantothenic Acid in which the Carboxyl Group is replaced by a Sulfonic Acid Group. It is similar to Pantothenic Acid in structure except that Taurine replaces Beta-Alanine in the molecule.

B6 (Pyridoxine)- a collective term for several compounds that are water-soluble Vitamin of the "B" group.

- Vitamin B6 enhances the function of Calcium.
- Vitamin B6 facilitates the incorporation of Iron into Hemoglobin - many people who have used Iron supplements for a long period of time to alleviate Anemia without success are rapidly cured when they incorporate Vitamin B6 into their regime.
- Vitamin B6 maintains normal intracellular Magnesium levels.
- Vitamin B6 regulates the body's Sodium:Potassium balance.
- Supplemental Vitamin B6 increases the intracellular concentration of Zinc (Vitamin B6 enhances the conversion of Tryptophan to Picolinic Acid; Picolinic Acid, in turn, greatly enhances the absorption of Zinc into the body's Cells).
- Vitamin B6 may enhance the function of Folate - it is an essential cofactor for the conversion of the Tetrahydrofolate (THF) form of Folic Acid to the Methyl Tetrahydrofolate form of Folic Acid (as required for the SAM Cycle).
- Vitamin B6 is an essential cofactor for the conversion of Tryptophan to Picolinic Acid (the isomer of the Nicotinic Acid form of Vitamin B3).
- Vitamin B6 may enhance the function of Vitamin B12.

Forms of Vitamin B6

- Pyridoxal is a form of Vitamin B6 obtained via the diet from Animal Food sources of Vitamin B6. Pyridoxine is very efficiently and rapidly converted to the Pyridoxal form by the Liver.
- Pyridoxal-5-Phosphate is the active endogenous coenzyme form of Vitamin B6.
- Pyridoxamine is found in Animal Food sources of Vitamine B6.
- Pyridoxine is the most common supplemental form of Vitamin B6 (in the form of Pyridoxine Hydrochloride). Pyridoxine is very efficiently and rapidly converted to the Pyridoxal form by the Liver.

Choline: water-soluble, lipotropic "B" Group Vitamin

- Choline assists the body to conserve Folic Acid(Folate).
- Alpha-Glycerylphosphorylcholine (a specific form of Choline) causes a rapid increase in plasma Choline levels.
- Folic Acid may enhance the function of Choline (Folic Acid "spares" Choline from being used for methylation, permitting more Choline to be used by the body for other purposes).
- The Niacinamide form of Vitamin B3 increases Brain Choline levels.
- Vitamin B5 is a cofactor for the conversion of Choline to Acetylcholine production.
- Vitamin B12 counteracts the impairment in Learning ability associated with insufficient

Acetylcholine production caused by Choline deficiency.

Forms of Choline

- Alpha-Glycerylphosphorylcholine (Alpha-GPC) is a form of Choline that consists of Choline bound to Glycerophosphate. It is the most bioavailable form of Choline. It is more potent than other forms of Choline (including Cytidine Diphosphate Choline (CDP-Choline)).
- Choline Bitartrate consists of 42% Choline bound to Tartaric Acid. This is by far the most common form of Choline present in commercially-available supplements. The main disadvantage of Choline Bitartrate is that it causes Diarrhea in some people.
- Choline Chloride consists of 33.3% Choline bound to the Chloride form of Chlorine. This form of Choline is present in some liquid Choline supplements. The main disadvantage of

Choline Chloride is that it possesses a very strong taste that some people find unpleasant.

- Choline Citrate (Choline Dihydrogen Citrate) consists of 38% Choline bound to Citric Acid (Citrate). This form of Choline is included in some Choline supplements. It is less capable of causing Gastrointestinal discomfort compared to Choline Bitartate.
- Choline Pantothenate consists of Choline bound to the Pantothenic Acid form of Vitamin B5. This form of Choline is included in some liquid Choline supplements.
- Cytidine Diphosphate Choline (CDP-Choline) is one of the active endogenous forms of Choline and is the form that is responsible for Choline's status as a lipotrope. It has only recently become available commercially as a supplement.
- Phosphatidylcholine (PC) is a type of Phosholipid that includes Choline within its chemical structure. Phosphatidylcholine provides Choline more efficiently to the Brain than the free form Choline in Choline itself.

Copper:

An essential acidic trace mineral, may be an enemy of Folic acid. Copper is involved in the production of Red Blood Cells (via its involvement in the production of Hemoglobin)

- Copper may increase the absorption of Iron.
- Boron may enhance the function of Copper and may increase serum Copper levels.
- Cobalt(Cobalamim) enhances the function of Copper.
- Sodium facilitates the absorption of Copper.
- Homocysteine, which is increased via the use of Folic acid interferes with Copper - it may reduce tissue levels of Copper and may reduce the activity of Copper-dependent Antioxidant enzymes.
- Calcium may reduce Copper absorption.
- Manganese may interfere with the body's absorption of Copper.
- Molybdenum competes with Copper for absorption - 500 mcg or more results in significant losses of Copper from the body.
- Zinc competes with Copper for absorption.
- Vitamin C may interfere with the absorption of Copper.
- Copper causes the oxidation of Vitamin C to Hydrogen Peroxide (and for this reason
- Copper should not be consumed simultaneously with Vitamin C)
- Copper may interfere with Magnesium.

With each meal (1,500 mg/day) for 64 days. Blood samples were obtained at 0, 28, 52, and 64 days in order to determine serum copper and serum ceruloplasmin. Each subject thus served as his own control. Analyses were repeated 20 days after the ascorbic acid supplement was terminated. Serum ceruloplasmin activity was significantly reduced (p less than 0.01) at every data point throughout the ascorbic acid supplementation period. A similar but nonsignificant trend was observed for serum copper. Furthermore there was a significant increase (p less than 0.01) in serum copper concentration 20 days after the supplementation period. Although observed effects occurred within physiological ranges of normal values, this study confirms that a high ascorbic acid intake is antagonistic to copper status of men as has been demonstrated in laboratory animals.

Summary:

- Human study demonstrated that vitamin C (1,500 mg per day for 64 days) causes significant reductions in serum ceruloplasmin (copper's transporting protein) and non-significant reductions in serum copper levels.
- Animal study (rats) demonstrated that vitamin C lowers copper absorption by decreasing the concentration of soluble copper in the small intestine after 42 days of vitamin C supplementation.
- Animal studies indicate that vitamin C may inhibit copper-induced oxidative damage

Zinc: a type of essential Micromineral. Zinc is involved in the structure and function of Cell Membranes

- Zinc is required for vitamin A transport.

Zinc may interfere with the absorption of Folic Acid.

Supplemental folate and folic acid influences zinc homeostasis, perhaps through formation of an insoluble chelate and impairment of absorption. If you take any type of aspirin or willow bark, then you are risking having proper amounts of folate depleted from your body.

- Excessive Zinc levels may reduce Chromium levels
- Zinc competes with Copper for absorption (it is noteworthy that the Zinc

Monomethionine form of Zinc does NOT compete with Copper for absorption).

- Zinc competes with Iron for absorption and excessive Zinc consumption may cause the depletion of Iron.
- Zinc may reduce the absorption of Magnesium.
- Excessive consumption of Zinc may inhibit the absorption of Manganese.
- Zinc is an enemy of Molybdenum.
- Zinc may compete with Phosphorus for absorption into the body.
- Excessive Zinc intake may cause (Copper deficiency-induced) Anemia

Zinc(and iodine) is a main nutrient in the spinal fluid of men that is released during ejaculation. Excessive sexual activity resulting in ejaculation results in depletion of zinc, iodine & other nutrients in the spinal fluid and prostate of men, resulting in prostatitis, lower back pain, arthritis and eventual impotence.

Ejaculation every day and multiple times a day are damaging to the male reproductive system because of the body lacking the ability to adequately replenish zinc, iodine & other nutrients to keep up with sexual activity & balance the biological system, especially when males are taking low quality vitamins or have poor nutritional & dietary habits that violate Edenic law(i.e. meat eating).

The zinc, iodine & other nutrients in the male sexual fluids & sperm serve more than just for healthy fertilization of female eggs. The fluids also serve to transfer the genetic soul & personality of the man to the woman for bonding (in addition to sweat), as well as to help promote healthy vaginal & uterine health, and to balance the sexual hormones (which in turns balances all other biological hormones, including the regulation of mental & emotional health & wellbeing) by functioning as a vitamin/mineral supplement to the vagina. This vital biological process is cut off with the use of condoms, smoking, alcohol, drugs & marijuana usage where THC (as well as nicotine) destroys the b-vitamins, zinc & nutrient quality of the male sperm and sexual fluids, as well as reduce such nutrients required for cervical health, thus increasing the risk for HPV, cervical dysplasia, cervical cancer, and the proliferation of candida & other detrimental bacteria in the vaginal system...

Forms of Zinc

- Chelated Zinc is a general term for forms of Zinc where Zinc is combined with Amino Acids. The Amino Acids in Chelated Zinc facilitate the absorption of Zinc. Chelated Zinc consists of 80% - 90% Amino Acids bound to 10 - 20% Zinc.
- Krebs Cycle Zinc is a form of Chelated Zinc that consists of elemental Zinc bound to a combination of the chemicals involved in the Krebs Cycle (Citric Acid, Furmaric Acid, Malic Acid, Succinic Acid and Aspartic Acid).
- Zinc Acetate (also known as Zinc Aceticum) consists of 35.5% Zinc bound to 64.5% Acetic Acid. Zinc Acetate Dihydrate consists of 29.78% Zinc.
- Zinc Ascorbate consists of 10% elemental Zinc bound to 90% Ascorbic Acid (Vitamin C).
- Zinc Aspartate consists of 32% elemental Zinc bound to 68% Aspartic Acid.
- Zinc Aspartate Citrate is a form of Chelated Zinc that consists of 25% elemental Zinc bound to Aspartic Acid and Citric Acid.
- Zinc Citrate consists of 30.3% elemental Zinc bound to 69.7% Citric Acid. It is a very easily absorbed form of Zinc.
- Zinc Gluconate consists of 14% elemental Zinc bound to 86% Gluconic Acid. This form of Zinc is used in the manufacture of Zinc lozenges and Zinc Nasal Gel Pumps for the treatment of the Common Cold.
- Zinc Glycinate is a form of Chelated Zinc that consists of 26% elemental Zinc bound to 74% Glycine.
- Zinc Histidinate is a form of Chelated Zinc that consists of Zinc bound to Histidine.
- Zinc Lysinate is a form of Chelated Zinc that consists of 14% elemental Zinc bound to 86% Lysine.
- Zinc Monomethionine (also known as Zinc Monomethionate or OptiZinc) consists of Zinc bound to Methionine. It is a desirable form of Zinc supplementation as it is easily absorbed. Zinc Monomethionine (unlike other forms of Zinc) does not interfere with the absorption of Copper.
- Zinc Orotate consists of 83% Orotic Acid (Orotate) bound to 17% Zinc. It is easily absorbed.
- Zinc Picolinate consists of 20% Zinc bound to 80% Picolinic Acid (Picolinate).
- Zinc Picolinate is easily absorbed and this form of Zinc may be the optimal form of Zinc supplementation as some clinical studies have shown Zinc Picolinate to be absorbed more effectively than other forms of Zinc.
- Zinc Pyrithione (also known as Zinc Omadine) is a Zinc-Sulfur compound that is NOT consumed orally but applied topically (usually to the Scalp).
- Zinc Succinate is a form of Zinc that consists of 35% elemental Zinc bound to 65% Succinic Acid.
- Zinc Sulfate ($ZnSO_4$) (also known as Zinc Sulphate) consists of 23% Zinc bound to 77% Sulfur. This inorganic form of Zinc is less well absorbed compared to other forms of Zinc and has been known to cause Gastric Irritation.
- Zinc-65 is a highly toxic Radioactive Isotope of Zinc.

Potassium: is an alkaline forming Electrolyte Macromineral. Potassium is essential for healthy cardiovascular functions.

- Potassium enhances calcium reabsorption
- Potassium excretion is positively related to bone mineral density
- Magnesium is required for potassium uptake in cells.
- Combination of magnesium, calcium, and potassium reduces the risk of stroke.

Potassium supplements can reduce absorption of vitamin B12. This effect has been reported with potassium chloride and, to a lesser extent, with potassium citrate.

- Potassium may also antagonize Iron & Manganese & visa versa

Forms of Potassium

- Chelated Potassium is a generic term for all forms of Potassium consisting of Potassium bound to an Amino Acid:
- Potassium Aspartate (C4H5KNO4) consists of 22% Potassium chelated to 78% Aspartic Acid (an Amino Acid). It is easily absorbed when consumed orally. Its molecular weight is 171.19.
- Potassium Ascorbate is a buffered form of Vitamin C that consists of 63% Ascorbic Acid bound to 37% Potassium.
- Potassium Bicarbonate (KHCO3) (also known as Carbonic Acid; Potassium Hydrogen Carbonate; Potassium Acid Carbonate) is believed to be the best form of Potassium for lowering elevated Blood Pressure and for alleviating Osteoporosis. This form of Potassium is the most common form of Potassium found in Fruits and Vegetables. It contains 39% elemental Potassium.
- Potassium Carbonate (also known as Salt of Tartar; Pearl Ash) is an inorganic Form of Potassium comprised of Potassium bound to Carbon.
- Potassium Chloride (KCl) consists of 52% Potassium bound to 48% Chlorine (Chloride).

It is an inorganic form of Potassium that enhances the production of endogenous Hydrochloric Acid. This form of Potassium is used in commercially available Salt Substitutes.

- Potassium Citrate (also known as Citrate Tripotassique; Potassio Citrato; Kaliumcitrat) is an organic form of Potassium that consists of 20.9% Potassium bound to 79.1% Citric Acid. Potassium Citrate is the form of Potassium present in many Potassium supplements. It is easily absorbed when consumed orally.
- Potassium Fumarate is an organic form of Potassium that consists of Potassium bound to Fumaric Acid.
- Potassium Gluconate is an organic form of Potassium that consists of 16.6% Potassium bound to 83.4% Gluconic Acid.
- Potassium Hydroxide (also known as Caustic Potash) is NEVER used as a nutritional supplement (it is highly toxic if consumed orally), however it IS a component of some topically applied Cosmaceuticals and Toiletries in which it functions as an emulsifier.
- Potassium Nitrate is a toxic Nitrate.
- Potassium Nitrite is a toxic Nitrite.
- Potassium Orotate consists of 20% Potassium bound to 80% Orotic Acid.
- Potassium Oxalate consists of Potassium bound to Oxalic Acid.
- Potassium Oxide is an inorganic form of Potassium.
- Potassium Phosphate consists of Potassium bound to Phosphorus.
- Potassium Sorbate is a form of Potassium combined with Sorbic Acid that prevents the growth of detrimental Moulds and Bacteria.
- Potassium Sulfate is an inorganic form of Potassium that consists of Potassium bound to Sulfur.

Iron (Ferrous Sulfate): Iron is an alkaline forming Micromineral. Iron is essential for the formation of Red Blood Cells (due to its incorporation into Hemoglobin).

- Cobalt may increase the body's absorption of Iron.
- Copper may increase the body's absorption of Iron.
- Molybdenum may enhance Iron metabolism.
- Vitamin A may facilitate the absorption of Iron.
- Vitamin B2 may facilitate the absorption of Iron.
- Vitamin B6 may facilitate the incorporation of Iron into Hemoglobin.
- Vitamin C is essential for absorption of Iron:
  - Vitamin C increases the absorption of Iron by such a large extent that if Vitamin Cand Iron are consumed simultaneously there is a risk of increasing the body's Iron status to dangerous levels.
  - Vitamin C converts the Ferric Iron that interferes with Hemoglobin back to
  - Ferrous Iron.
  - Vitamin C converts the hard to absorb Ferric (inorganic) form of Iron to the more easily absorbed Ferrous (organic) form of Iron.
  - Some authors claim that Vitamin C only increases Iron absorption if existing Iron stores are low.
- Vitamin C may counteract the toxic effects of Iron
- Folate may counteract the toxic effects of Iron.
- Lipoic Acid may counteract the toxic effects of Iron.
- Excessive consumption of Calcium may reduce the absorption of Iron.
- Chromium may interfere with the absorption of Iron.

Phosphorus may bind with Iron and may decrease its absorption.

- Potassium may interfere with Iron.
- Zinc may compete with Iron for absorption.

- Lipoic Acid may bind to (chelate) Iron and as a result of this binding may cause Iron deficiency.
- Vitamin C reacts with Ferric Iron (oxidized Iron) to form Ferrous Iron (reduced Iron) - Ferrous Iron, in turn reacts with Hydrogen Peroxide to form Hydroxyl Free Radicals.
- The wrong type of Iron contributes to rusting of bones which shows up later as arthritis & rheumatism. Most arthritis problems are directly related to "IRON"....
- The wrong type of iron cannot be absorbed in the body and thus doesn't become digested and simply accumulates in the organs and joints leading to premature aging....
- Excessive Iron may reduce life expectancy (i.e. Iron may have anti-Life Extension properties). Excessive Iron may accelerate the Aging Process (due to Iron's role in the generation of Hydroxyl Free Radicals).
- Reducing iron is one of the most important aspects of Anti-aging medicine...
- Iron interferes with Amino acid such as Glutathione.
- People who are slightly anemic are healthier than people with normal levels of Iron-Hemoglobin in their system... Yet those who take Iron supplements for Anemia almost never see such Anemia reversed, especially iron/ferrous sulfate.
- Ferrous Phosphate and Ferrous Gluconate can reverse anemia within about 1 month compared to sulfate patients who may have been using it for years.

Forms of Iron

- Chelated Iron is a general term for forms of Iron where Iron is bound (chelated) with Amino Acids. The average absorption rate of orally-ingested Chelated Iron is 10%. Iron chelates most effectively with the Amino Acid Cysteine.
- Ferric Iron (also known as Fe+3) is a generic term for all oxidized forms of Iron (as opposed to reduced (Ferrous) forms of Iron.
- Ferritin is the form in which Iron (as Ferric Oxide) is stored within the body bound to the Globulin - Transferrin. Ferritin contains 23% Iron.
- Ferrous Aspartate (Iron Aspartate) is a form of Chelated Iron that consists of Iron bound to Aspartic Acid.
- Ferrous Carbonate consists of Iron bound to Carbonic Acid. 24% of orally- ingested Ferrous Carbonate is absorbed by the body.
- Ferrous Chloride consists of Iron bound to the Chloride form of Chlorine.
- Ferrous Citrate (Iron Citrate) consists of Iron bound to Citric Acid. 24% of orally- ingested Ferrous Citrate is absorbed by the body.
- Ferrous Fumarate (Iron Fumarate) (FeC4H204) consists of 31.9% to 33.2% Iron bound to Fumaric Acid. 31% of orally-ingested Ferrous Fumarate is absorbed by the body. Ferrous Fumarate has been shown to inhibit Vitamin E absorption. Its molecular weight is 169.9.
- Ferrous Gluconate (Iron Gluconate) (C12H22FeO14) consists of 11.9% - 12.8% Iron bound to Gluconic Acid. 12% of orally-ingested Ferrous Gluconate is absorbed by the body.
- Ferrous Iron (also known as Fe+2) is a generic term for all reduced (as opposed to oxidized (Ferric) forms of Iron.
- Ferrous Oxide is a form of Iron into which other forms of Iron are converted within the body for further incorporation into Ferritin.
- Ferrous Succinate (Iron Succinate) consists of Iron bound to Succinic Acid.
- Ferrous Sulfate (Iron Sulfate) is an inorganic form of Iron that consists of 20% Iron bound to 80% Sulfur (Sulfate). It is a less desirable form of Iron supplement as it destroys Vitamin E and causes Constipation and Nausea. This is the iron that is most prescribed by allopathic MDs, as is the case with other toxic sulfates.

Ferric Iron is the oxidized form of Ferrous Iron. It is recommended that Ferric Iron be avoided as it destroys Vitamin E and is responsible for the conversion of Hemoglobin to Methemoglobin.

- Heme Iron (Haem Iron) is Iron conjugated with a Protein - e.g. Hemoglobin or Myoglobin. Heme Iron can be obtained from the diet from animal sources - e.g. Meats. 20 - 30% of orally-ingested Heme Iron is absorbed by the human body. It is regarded as a relatively safe form of Iron.
- Iron Bisglycinate (also known as Ferrochel; Ferrous Bisglycinate) is a form of Chelated Iron that is claimed by its manufacturers to be highly bioavailable. Its manufacturers also claim that Iron Bisglycinate does not cause the gastrointestinal irritation or Constipation that some forms of supplemental Iron cause. references
- Iron Citrate consists of Iron bound to Citric Acid. This form of Iron is well tolerated by sensitive Gastrointestinal Tracts.
- Iron Lactate consists of Iron bound to Lactic Acid.
- Iron Phosphate consists of 16% Iron bound to 84% Phosphorus.
- Iron Picolinate consists of Iron bound to Picolinic Acid. Some researchers regard Iron Picolinate as the optimal form of Iron supplementation. The bioavailability of this form of Iron is very high.

Manganese: Manganese is a type of Essential Micromineral.

- Manganese may enhance the utilization of Biotin.
- Manganese may convert Vitamin B1 into its biologically active form - Thiamine

Pyrophosphate (TPP).

- Manganese may enhance the function of Vitamin C
- Manganese may enhance the function of Vitamin E.
- Manganese may improve the absorption of dietary Iron.
- Manganese may improve the absorption of Silicon.
- Manganese may improve the absorption of Zinc
- Excessive consumption of Calcium may interfere with Manganese & vice versa.
- Excessive consumption of Copper may inhibit the absorption of Manganese & vice versa.
- Excessive consumption of Iron may inhibit the absorption of Manganese & vice versa.
- Excessive consumption of Magnesium may reduce the absorption of Manganese.
- Phosphorus may interfere with the absorption of Manganese.
- Potassium may interfere with the absorption of Manganese & vice versa.
- Excessive consumption of Zinc may inhibit the absorption of Manganese
- Dairy Products may reduce the body's absorption of Manganese (due to the high Calcium content of most Dairy Products). Thus, consumption of cows milk for example, can lead to Osteoporosis & other metabolic bone disorders, especially due to the DNA/RNA structure of dairy milk designed specifically to nurture & produce large, healthy baby calf(cows), which leads to leeching of calcium from human bones.

Forms of Manganese

- Manganese Ascorbate consists of 13% Manganese bound to 87% Ascorbic Acid (Vitamin C).
- Manganese Aspartate is a supplemental form of Manganese that consists of 17% Manganese bound to 83% Aspartic Acid.
- Manganese Chelate is a general term for Manganese bound to Amino Acids. On average, Manganese Chelate is 10% Manganese. Its absorption is generally excellent. The most effective Amino Acid for chelation with Manganese is Arginine.
- Manganese Chloride consists of 27.7% Manganese bound to the Chloride form of Chlorine.
- Manganese Citrate ($Mn_3(C_6H_5O_7)_2$) is a supplemental form of Manganese that consists of 30% Manganese bound to 70% Citric Acid. Its molecular weight is 723.17.
- Manganese Gluconate ($MnC_{12}H_{22}O_{14}$) is a supplemental form of Manganese that consists of Manganese bound to Gluconic Acid. It consists of 11.0% - 12.2% Manganese. Its molecular weight is 445.24 (for Anhydrous Manganese Gluconate) or 481.27 (for Manganese Gluconate Dihydrate).
- Manganese Orotate is a supplemental form of Manganese that consists of 31.5% Manganese bound to Orotic Acid.
- Manganese Picolinate is a supplemental form of Manganese that consists of 20% Manganese bound to 80% Picolinic Acid.
- Manganese Sulfate ($MnSO_4$) is an inorganic supplemental form of Manganese that consists of 31.8% - 37.2% Manganese bound to Sulfur. It is equal in terms of its bioavailability to Manganese Gluconate. Its molecular weight is 169.01.

Vitamin C: Vitamin C is a water-soluble acidic Vitamin that occurs physically as a white, crystalline powder. It is usually found in nature in its ***L -Isomer*** form but its ***D-Isomer*** is named ***Isoascorbic Acid***.

Vitamin C facilitates the normal transport of Calcium through the body's membranes and Vitamin C inhibits the increased excretion of Calcium caused by Chitosan.

- Vitamin C converts the dangerous Hexavalent form of Chromium to its' beneficial Trivalent form.
- Vitamin C is essential for absorption of Iron:
  - Vitamin C increases the absorption of Iron by such a large extent that if Vitamin C and Iron are consumed simultaneously there is a risk of increasing the body's Iron status to dangerous levels
  - Vitamin C converts the non-bioavailable Ferric (inorganic) form of Iron to the more easily absorbed Ferrous (organic) form of Iron.
- Vitamin C may enhance the absorption of Magnesium.
- Vitamin C may enhance the function of Phosphorus.
- Vitamin C may facilitate the conversion of inorganic forms of Selenium (Selenates and Selenites) into active organic forms.
- Vitamin C is a cofactor for the conversion of Folic Acid to its active form (Tetrahydrafolic Acid (THFA)) and also protects Folic Acid from oxidation: references
  - Vitamin C protects Tetrahydrobiopterin from degradation references

- Vitamin C enhances the Antioxidant properties of Vitamin A, protects Vitamin A from oxidation, protects against Vitamin A toxicity and facilitates its absorption.
- Vitamin C may prevent the oxidation of Vitamin B1.
- Vitamin C may prevent the oxidation of Vitamin B2.
- Vitamin C may prevent the oxidation of Vitamin B5.
- Vitamin C may facilitate the retention of Vitamin B12.
- Vitamin C is synergistic with Vitamin E and may prevent the oxidation of Vitamin E:
  - Vitamin C may increase plasma Vitamin E levels (by retarding the degradation/oxidation of Vitamin E).
  - Vitamin C regenerates Vitamin E's degraded by-product - Tocopheryl Quinone.
  - Vitamin C quenches Tocopheroxyl Free Radicals (the Free Radicals generated by Vitamin E).
- Copper may decrease the absorption of Vitamin C and stimulates the oxidation of Vitamin C to Hydrogen Peroxide.
- Iron may decrease the absorption of Vitamin C (despite the fact that Vitamin C increases Iron absorption):
  - Iron may cause the oxidation of Vitamin C, resulting in the formation of Hydrogen Peroxide.

Forms of Vitamin C

The term Ascorbic Acid is a generic term for all Vitamin C forms, however it also refers to pure Vitamin C (also known as single entity Ascorbic Acid or L-Ascorbic Acid) that is not bound to any other compound. Some researchers regard pure Ascorbic Acid as the best form of oral Vitamin C supplementation (with the exception of people prone to Gastrointestinal distress from highly acidic compounds). Pure Ascorbic Acid is water-soluble (i.e. not fat-soluble) and is therefore unable to “reach” fatty regions of the human body.

Ascorbin Stearate is a fat-soluble, ester form of Vitamin C that is sometimes utilized in the commercial processing of foods as an Antioxidant. It consists of Ascorbic Acid bound to Stearic Acid.

Ascorbyl Palmitate (also known as 6-Palmitoyl-L-Ascorbic Acid; Palmitoyl Ascorbate; Vitamin C Ester) is a fat-soluble ester form of Vitamin C that consists of 42.8% Ascorbic Acid bound to 57.2% Palmitic Acid. Structurally, it is Ascorbic Acid with a Palmitic Acid chain attached at the sixth position. Ascorbyl Palmitate is retained within the body for a longer period of time than water-soluble forms of Vitamin C:

- Ascorbyl Palmitate is more effective than water-soluble Vitamin C for preventing lipid peroxidation and for transporting Ascorbic Acid to the Heart, Brain and Central Nervous System.
- Ascorbyl Palmitate is claimed to be thirty times more effective for preventing some forms of Cancer compared to water-soluble forms of Vitamin C. Clinical studies support this claim.
- Ascorbyl Palmitate is utilized commercially in some Processed Foods as an Anti-Staling Agent (due to its Antioxidant properties).
- The pH of Ascorbyl Palmitate is neutral.
- Ascorbyl Palmitate is an ingredient in some topically applied Skin care products at a concentration of up to 20%. It is believed that Ascorbyl Palmitate is the ideal form of Vitamin C for topical application to the Skin. Skin Ascorbic Acid levels are eight times higher after topical application of Ascorbyl Palmitate compared to topical application of Ascorbic Acid.
- Oral Ascorbyl Palmitate works best when used in conjunction with water- soluble Vitamin C leading some researchers to recommend Vitamin C supplementation in the form of 50% Ascorbyl Palmitate combined with 50% pure Ascorbic Acid.
- Ascorbyl Tetraisopalmitate is a modified form of Ascorbyl Palmitate used in topical Skin care products.

Ascorbic Acid 2-Phosphate (also known as Asc2P; Asc-2-P; Ascorbate-2-O- Phosphate; Ascorbyl-2-Polyphosphate; Asc-2-O-Phosphate) is a stable (Oxidation-resistant) form of Vitamin C used in some topically applied Skin Care products. It is more stable than other forms of Vitamin C when in an aqueous solution exposed to air. This form of Vitamin C has been demonstrated in laboratory studies to inhibit the shortening of Telomeres.

Calcium Ascorbate is a buffered form of Vitamin C. It consists of 82% Ascorbic Acid bound to 18% Calcium. This form of Vitamin C minimizes the Gastrointestinal discomfort (acidic irritation) that sometimes occurs in people who use pure Ascorbic Acid.

Ester-C is a patented form of Vitamin C that consists of several molecules of Ascorbic Acid linked together to form one large molecule (it is an ester of Vitamin C molecules). Ester-C also contains metabolites of Vitamin C such as Threonic Acid that are claimed by its manufacturer to increase its bioavailability:

- Ester-C is claimed to be the most bioavailable form of Vitamin C by those commercial ventures who market it. Some scientific studies have concluded that Ester-C is NOT absorbed any more efficiently than other (less-expensive) forms of Vitamin C. references

- Isoascorbic Acid (also known as D-Ascorbic Acid or Erythroascorbic Acid) is the "d" isomer of Ascorbic Acid. It is a component of some Vitamin C supplements.
- Liposomal Vitamin C is Ascorbic Acid encapsulated in Liposomes in order to maximize the absorption of Vitamin C.
- Magnesium Ascorbate is a buffered form of Vitamin C. It consists of 64% Ascorbic Acid bound to 36% Magnesium.
- Magnesium Ascorbyl Phosphate consists of Ascorbic Acid bound to Magnesium and Phosphorus. It is a stable form of Vitamin C that is used in many topically- applied Vitamin C products (such as Skin Creams and Skin-Bleaching Creams).
- Potassium Ascorbate is a buffered form of Vitamin C that consists of 63% Ascorbic Acid bound to 37% Potassium.
- Sodium Ascorbate is a buffered form of Vitamin C that consists of 90% Ascorbic Acid bound to 10% Sodium. This is the optimal form of Vitamin C for intravenous injection.
- Sodium Ascorbyl Phosphate is a water-soluble form of Vitamin C that consists of Ascorbic Acid bound to Sodium and Phosphorus. It is used in topical skin care products.

Best Form?

Pure Ascorbic Acid is the least expensive form of oral Vitamin C supplementation and is the form used in many clinical studies that have demonstrated the therapeutic effectiveness of Vitamin C. Some authors claim that pure Ascorbic Acid (orally) is more effective than Calcium Ascorbate or Sodium Ascorbate orally.

Recent studies also provide scientific evidence for the use of the Ascorbyl Palmitate form. A logical strategy may be to use a combination of 50% pure Ascorbic Acid (which is water-soluble) + 50% Ascorbyl Palmitate (which is fat- soluble). This would ensure delivery of Vitamin C to both the water-soluble and fat-soluble regions of the body.

The Sodium Ascorbate form of Vitamin C is the optimal form for intravenous injection. Other forms of Vitamin C are too acidic for injecting.

Vitamin D:

Vitamin D is not one singular compound, but a group of related Fat-Soluble Vitamins. Vitamin D Analogs are a group of Pharmaceutical Drugs that mimic the effects of Vitamin D (specifically, they mimic the effects of Vitamin D3 (Cholecalciferol)). Vitamin D Analogs are claimed to possess an advantage over natural Vitamin D in that they are more effective and less toxic than natural Vitamin D

- Boron may be essential for the conversion of Vitamin D to its active form.
- Vitamin A may counteract the toxicity of very high doses of Vitamin D.
- Most African Americans vegan, vegetarian or meat eater is deficient in this very essential Vitamin (the source of more diseases that you can ever imagine when deficient in Vitamin D).
- Excessive consumption of Phosphorus may interfere with the conversion of Vitamin D to its active endogenous form.
- Vitamin A may interfere with one of the functions of Vitamin D - specifically, Vitamin A impairs the ability of Vitamin D2 to increase Bone Calcium levels
- Vitamin D2 is associated with increased risk of Heart Disease and is not healthy for humans. It's the most commonly prescribed by Allopathic MDs and found in Dairy Milk.
- Excessive intake of Vitamin D may increase the urinary excretion of Magnesium:
- On the other hand, Vitamin D in non-excessive doses increases the body's absorption of Magnesium and Vitamin D deficiency impairs the absorption of Magnesium.
- Vitamin D3 is the only healthy form of Vitamin D. Yet any supplement of Vitamin D above 1000IU is dangerous when taken without constant blood labs during such intake. To self medicate with Vitamin D at 1000 or higher is asking for disease!

Forms of Vitamin D:

Calcidiol- Calcidiol is one of the several forms of Vitamin D - in terms of Vitamin D activity it is regarded as partially-active. It is the major circulating form of Vitamin D and is the first intermediate form of Vitamin D synthesized during the conversion of Vitamin D3 to Calcitriol.

Calcitriol is one of the several forms of Vitamin D. It is ten times more potent than Vitamin D3. Calcitriol is the active form of Vitamin D in regulating Bone growth and Calcium metabolism.

Secacalciferol is one of the several forms of Vitamin D. Plasma concentrations of Secacalciferol are believed to be 100 times higher than those of the Calcitriol form of Vitamin D.

- Vitamin D1
- Vitamin D2- is NOT equivalent (in terms of therapeutic effects and safety) to Vitamin D3
- Vitamin D3- can be classed as a Hormone.

CONCLUSION

Vitamins play vital roles in maintaining optimal health and supporting various physiological processes within the human body. Understanding the interactions, reactions, and counteractions of vitamins provides valuable insights into their complex dynamics. The interdependencies between vitamins highlight the

importance of a well-balanced diet to ensure adequate intake of all essential nutrients. By recognizing potential synergistic, antagonistic, and counteractive effects, healthcare professionals can design effective strategies for preventing nutrient imbalances and promoting optimal health outcomes. Further research in this field is warranted to uncover additional mechanisms and explore the potential interactions between vitamins and other nutrients. Ultimately, a comprehensive understanding of vitamin interactions will contribute to personalized nutrition interventions and improved health outcomes for individuals.

Peer-Reviewed Professional Journals

1. Green, T. J., et al. Effect of folic acid supplementation on plasma zinc concentrations of young women. Nutrition 19(6):522-523, 2003.

Women of reproductive age are advised to consume supplements or fortified foods containing at least 400 g/d folic acid for the prevention of neural tubes defects. Concerns exist about the adverse effects of folic acid on zinc status. Seventy-eight women (18 to 49 y) were assigned for 12-wk to receive either a placebo or a 400 g/d folic acid supplement. At 12-wk mean (95% CI) red cell folate increased by 431 (350–511) nmol/L in the supplemented group relative to placebo ($P < 0.001$) but there was no change in plasma zinc in either group ($P = 0.213$). Folic acid supplementation does not reduce plasma zinc concentrations in women of childbearing age.

2. Hansen, M., et al. Folic acid enrichment of bread does not appear to affect zinc absorption in young women. American Journal of Clinical Nutrition. 74(1):125-129, 2001.

Research Department of Human Nutrition, LMC Centre for Advanced Food Studies, the Royal Veterinary and Agricultural University, Frederiksberg, Denmark. In several countries cereals are now enriched with folic acid to reduce the risk of neural tube defects. Human studies suggest that folic acid interferes with zinc absorption. This raises concerns about the zinc status of high-risk groups such as infants, pregnant women, and older persons. The authors sought to determine the effect of added folic acid on zinc absorption from white bread with high and low zinc contents. Zinc absorption was measured in 15 healthy women (22-33 y), each of whom consumed 4 single meals spaced 2 wk apart in a randomized crossover design. The servings of bread (100 g) differed in zinc and folic acid contents as follows: A, 1.2 mg Zn and 17 microg folic acid; B, 1.2 mg Zn and 144 microg folic acid; C, 3.0 mg Zn and 17 microg folic acid; and D, 2.9 mg Zn and 144 microg folic acid. Meals were extrinsically labeled with 65Zn and absorption was estimated from whole-body retention measurements. Folate status was assessed by measuring plasma and erythrocyte folate and plasma homocysteine concentrations. Mean (+/-SD) zinc absorption did not differ significantly in relation to the folate content of the breads at either the low zinc content (38.8 +/- 13.5% and 40.6 +/- 16.5% for A and B, respectively; $P = 0.74$) or the high zinc content (26.7 +/- 9.3% and 22.7 +/- 6.6% for C and D, respectively; $P = 0.16$). There was no significant correlation between folate status and zinc absorption ($r < 0.3$, $P > 0.1$). Fortification of white bread with a commonly used amount of folic acid did not appear to influence zinc absorption at either a high or a low zinc content.

3. Kauwell, G. P., et al. Zinc status is not adversely affected by folic acid supplementation and zinc intake does not impair folate utilization in human subjects. Journal of utrition 125(1):66-72, 1995.

4. Food Science and Human Nutrition Department, University of Florida, Gainesville, USA.

Changes in zinc status in response to folic acid supplementation and the effect of zincintake on folate utilization were evaluated in 12 men (20-34 y old) consuming a diet containing 3.5 or 14.5 mg zinc/d for two 25-d intervals. Deuterium-labeled folic acid (800 micrograms/d) or a placebo was administered orally during each phase. No differences in plasma zinc, erythrocyte zinc, urinary zinc, erythrocyte metallothionein or serum alkaline phosphatase, due to supplemental folic acid, were detected at either level of zinc intake. Differences in the response to folic acid supplementation, due to the level of zinc intake, were not detected for serum, erythrocyte or urinary (labeled and unlabeled) folate. Within the constraints of this short-term folic acid supplementation study, adverse effects on zinc status were not observed and our data suggest that folic acid utilization was not influenced by level of zinc intake.

5. Lowenthal, E. A., et al. Homocysteine elevation in sickle cell disease. Journal of the American College of Nutrition. 19(5):608-612, 2000.

Plasma concentrations of homocysteine, vitamin B12 and folate were measured in 49 adults with sickle cell disease and 16 normotensive Black controls. All subjects with sickle cell disease had been prescribed folic acid 1 mg by mouth daily. The median plasma concentration of homocysteine of subjects with sickle cell disease was approximately 1.5 fold higher than that of controls ($p=0.0008$). This difference persisted, even when subjects with renal insufficiency were excluded. Plasma folate levels were 1.5 fold higher in subjects with sickle cell disease than in controls ($p=0.0498$). There was no significant difference in plasma vitamin B12 concentrations between the two groups. There was no difference in plasma homocysteine concentrations between transfused and non-transfused sickle cell subjects. Patients with sickle cell disease have elevated plasma concentrations of homocysteine in spite of elevated plasma folate levels and vitamin B12 concentrations similar to those observed in controls.

The concentration of folate required to normalize plasma homocysteine levels in patients with sickle cell disease may be higher than that of normal

controls. Patients with sickle cell disease are likely to have a higher nutritional requirement for folic acid than the general population.

Summary: *Human study demonstrated that sickle cell anemia patients have plasma homocysteine levels that are (on average) 1.5-fold higher than normal, healthy persons.*

6. Graham, I. M., et al. Plasma homocysteine as risk factor for vascular disease. Journal of the American Medical Association. 277(22):1775-1781, 1997

Department of Cardiology, Adelaide Hospital, Trinity College, Dublin, Ireland. Elevated plasma homocysteine is a known risk factor for atherosclerotic vascular disease, but the strength of the relationship and the interaction of plasma homocysteine with other risk factors are unclear. The objective was to establish the magnitude of the vascular disease risk associated with an increased plasma homocysteine level and to examine interaction effects between elevated plasma homocysteine level and conventional risk factors. This was a case-control study. A total of 750 cases of atherosclerotic vascular disease (cardiac, cerebral, and peripheral) and 800 controls of both sexes younger than 60 years were studied. Plasma total homocysteine was measured while subjects were fasting and after a standardized methionine-loading test, which involves the administration of 100 mg of methionine per kilogram and stresses the metabolic pathway responsible for the irreversible degradation of homocysteine. Plasma cobalamin, pyridoxal 5'-phosphate, red blood cell folate, serum cholesterol, smoking, and blood pressure were also measured. The relative risk for vascular disease in the top fifth compared with the bottom four fifths of the control fasting total homocysteine distribution was 2.2 (95% confidence interval, 1.6-2.9). Methionine loading identified an additional 27% of at-risk cases. A dose-response effect was noted between total homocysteine level and risk. The risk was similar to and independent of that of other risk factors, but interaction effects were noted between homocysteine and these risk factors; for both sexes combined, an increased fasting homocysteine level showed a more than multiplicative effect on risk in smokers and in hypertensive subjects. Red blood cell folate, cobalamin, and pyridoxal phosphate, all of which modulate homocysteine metabolism, were inversely related to total homocysteine levels. Compared with nonusers of vitamin supplements, the small number of subjects taking such vitamins appeared to have a substantially lower risk of vascular disease, a proportion of which was attributable to lower plasma homocysteine levels. An increased plasma total homocysteine level confers an independent risk of vascular disease similar to that of smoking or hyperlipidemia. It powerfully increases the risk associated with smoking and hypertension. It is time to undertake randomized controlled trials of the effect of vitamins that reduce plasma homocysteine levels on vascular disease risk.

7. Nygard, O., et al. Total plasma homocysteine and cardiovascular risk profile. Journal of the American Medical Association. 274(19):1526-1533,1995.

Section for Medical Informatics and Statistics, University of Bergen, Norway. The objective of this study was to estimate the relations between established cardiovascular risk factors and total homocysteine (tHcy) in plasma. Health examination survey by the Norwegian Health Screening Service in 1992 and 1993. SETTING--General community, Hordaland County of Western Norway. A total of 7591 men and 8585 women, 40 to 67 years of age, with no history of hypertension, diabetes, coronary heart disease, or cerebrovascular disease were included. Plasma tHcy level was measured. The level of plasma tHcy was higher in men than in women and increased with age. In subjects 40 to 42 years old, geometric means were 10.8 mumol/L for 5918 men and 9.1 mumol/L for 6348 women. At age 65 to 67 years, the corresponding tHcy values were 12.3 mumol/L (1386 men) and 11.0 mumol/L (1932 women). Plasma tHcy level increased markedly with the daily number of cigarettes smoked in all age groups. Its relation to smoking was particularly strong in women. The combined effect of age, sex, and smoking was striking. Heavy-smoking men aged 65 to 67 years had a mean tHcy level 4.8 mumol/L higher than never-smoking women aged 40 to 42 years. Plasma tHcy level also was positively related to total cholesterol level, blood pressure, and heart rate and inversely related to physical activity. The relations were not substantially changed by multivariate adjustment, including intake of vitamin supplements, fruits, and vegetables. Elevated plasma tHcy level was associated with major components of the cardiovascular risk profile, ie, male sex, old age, smoking, high blood pressure, elevated cholesterol level, and lack of exercise. These findings should influence future studies on the etiology and pathogenesis of cardiovascular disease.

8. Stampfer, M. J., et al. Can lowering homocysteine levels reduce cardiovascular risk? New England Journal of Medicine. 332(5):328-329,1995.

9. Firshein, R. The Nutraceutical Revolution. Riverhead Books via Penguin Putnam Inc., New York, USA. 1998:147.

10. Segala, M. (editor). Disease Prevention and Treatment 3rd Edition. Life

Extension Media. Florida, USA. 2000:348-350. Homocysteine levels above 6.3 micromoles per liter of blood cause a steep increase in the risk of cardiovascular diseases such as heart attacks. Each 3 micromole increase in homocysteine is related to a 35% increase in the risk of heart attack.

11. Kelly, G. S. Folates: supplemental forms and therapeutic applications. Alt Med Rev. 3(3):208-220, 1998.

12. Pinto, J., et al. Increased urinary riboflavin excretion resulting from boric acid ingestion. J Lab Clin Med. 92(1):126-34, 1978.

The urinary excretion of riboflavin (vitamin B2) was determined in 14 patients, both children and adults, after the ingestion of boric acid. These patients reported to the New York City Poison Control Center during a 2 1/2 year period. Riboflavin was determined by a modification of a previously described method which is based upon competitive protein binding. Boric acid ingestion was associated with greatly increased urinary riboflavin excretion in approximately two thirds of the patients. Most of the riboflavin appeared to be excreted within the first 24 hr after ingestion of boric acid. These data provide evidence of a previously unrecognized hazard of boric acid ingestion in patients.

13. Hustad, S., et al. Riboflavin as a determinant of plasma total homocysteine: effect modification by the methylenetetrahydrofolate reductase C677T polymorphism. Clinical Chemistry. 46(8):1065-1071, 2000.

Plasma total homocysteine (tHcy) is a risk factor for cardiovascular disease. tHcy concentrations are partly determined by folate, cobalamin, and vitamin B6 status, and methylenetetrahydrofolate reductase (MTHFR) and other flavoenzymes are important for the biotransformation of these vitamins. This motivates the investigation of the possible relationship between riboflavin status and tHcy. The study had a cross-sectional design and included 423 healthy blood donors, ages 19–69 years. The authors determined plasma tHcy, serum folate, serum cobalamin, serum creatinine, and MTHFR C677T genotype. In addition, they measured riboflavin and its two coenzyme forms, flavin mononucleotide and flavin adenine dinucleotide, in EDTA plasma by capillary electrophoresis and laser-induced fluorescence detection. Riboflavin determined tHcy independently in a multiple linear regression model with adjustment for sex, age, folate, cobalamin, creatinine, and MTHFR genotype (P 5 0.008). tHcy was 1.4 mmol/L higher in the lowest compared with the highest riboflavin quartile. The riboflavin-tHcy relationship was modified by genotype (P 5 0.004) and was essentially confined to subjects with the C677T transition of the MTHFR gene. Plasma riboflavin is an independent determinant of plasma tHcy. Studies on deficient populations are needed to evaluate the utility of riboflavin supplementation in hyperhomocysteinemia.

14. Morrow, J. D., et al. Identification of skin as a major site of prostaglandin D2 release following oral administration of niacin in humans. J Invest Dermatol.98(5):812-815, 1992.

Oral administration of niacin (nicotinic acid) at pharmacologic doses that reduce serum cholesterol levels induces intense flushing in humans. The authors have previously shown that the vasodilation following ingestion of niacin is due to the release of prostaglandin D2. However, the site from which prostaglandin D2 is released is not known. It has previously been shown that topical application of methylnicotinate causes local cutaneous erythema. The authors investigated whether topical methylnicotinate causes a release of prostaglandin D2 locally from skin and the possibility that skin may be a major contributor to the release of prostaglandin D2 when niacin is administered orally. Topical administration of methylnicotinate (10(-1) M) to the forearms of human volunteers resulted in 58- to 122-times increases in levels of prostaglandin D2 and 25- to 33-times increases in levels of the metabolite of prostaglandin D2, 9 alpha,11 beta-prostaglandin F2, in blood drawn from the antecubital vein draining the treated sites. Increased levels of prostaglandin D2 and 9 alpha,11 beta-prostaglandin F2 were not found in blood drawn simultaneously from veins in the contralateral arm, indicating that the prostaglandin D2 was released from the site of methylnicotinate application. The release of prostaglandin D2 in response to topically applied methylnicotinate occurred in a dose-dependent manner over the concentration range of 10(-3) to 10(-1) M. The release of prostaglandin D2 was not accompanied by a release of histamine, suggesting that the release of prostaglandin D2 was not from the mast cell. Following oral ingestion of niacin, levels of prostaglandin D2 in superficial venous blood draining the skin were 14 to 1200 times higher than the level in arterial blood supplying the skin of the same arm. This finding indicates that the skin is a major site from which prostaglandin D2 is released following oral ingestion of niacin. These studies thus indicate that the cutaneous vasodilation that occurs following oral administration of niacin is primarily due to a release of PGD2 from a niacin responsive cell that resides in the skin.

15. Garg, R., et al. Niacin treatment increases plasma homocyst(e)ine levels. Am Heart J. 138(6 Part 1):1082-1087, 1999. Lilly Research Laboratories, Eli Lilly and Company, Indianapolis, IN, USA.

Studies have reported high levels of plasma homocyst(e)ine as an independent risk factor for arterial occlusive disease. The Cholesterol Lowering Atherosclerosis Study reported an increase in plasma homocyst(e)ine levels in patients receiving both colestipol and niacin compared with placebo. Thus the objective of this study was to examine the effect of niacin treatment on plasma homocyst(e)ine levels. The Arterial Disease Multiple Intervention Trial, a multicenter randomized, placebo-controlled trial, examined the effect of niacin compared with placebo on homocyst(e)ine in a subset of 52 participants with peripheral arterial disease. During the screening phase, titration of niacin dose from 100 mg to 1000 mg daily resulted in a 17% increase in mean plasma homocyst(e)ine level from 13.1 +/- 4.4 micromol/L to 15.3 +/- 5.6 micromol/L (P <.0001). At 18 weeks after randomization, there was an absolute 55% increase from baseline in mean plasma homocyst(e)ine levels in the niacin group and a 7% decrease in the placebo group (P =.0001). This difference remained statistically significant at the end of follow-up at 48 weeks. Niacin substantially increased plasma homocyst(e)ine levels,

which could potentially reduce the expected benefits of niacin associated with lipoprotein modification. However, plasma homocyst(e)ine levels can be decreased by folic acid supplementation. Thus further studies are needed to determine whether B vitamin supplementation to patients undergoing long-term niacin treatment would be beneficial.

16. Dean, W. & Morgenthaler, J. Smart Drugs & Nutrients. B & J Publications, Santa Cruz, California, USA. 1990:155.
17. Greenwell, I. Enhancing cognitive function. Life Extension. 6(5), 2000. The brain utilizes vitamin B5 for the conversion of choline into the neurotransmitter, acetylcholine.
18. Turnlaud, J. R., et al. Vitamin B6 depletion followed by repletion with animal- or plant-source diets in calcium and magnesium metabolism in young women. American Journal of Clinical Nutrition. 56(5):905-10, 1992.

An 84-98-day study was conducted in young women to determine the effect of vitamin B6-deficient diets on calcium and magnesium metabolism. A vitamin B6- deficient formula diet fed initially was followed by either animal- or plant-source protein food diets containing four increasing amounts of vitamin B6. Calcium balance was negative during vitamin B6 depletion. Serum calcium was higher and calcium balance negative with the plant protein diets. Magnesium balance was negative during vitamin B6 depletion due to increased urinary magnesium excretion. Urinary calcium decreased during vitamin B6 depletion and increased as dietary vitamin B6 increased. Urinary oxalate was significantly higher at the end than at the beginning of vitamin B6 depletion and was higher with plant than animal protein diets. Vitamin B6 depletion may alter calcium and magnesium metabolism and dietary components associated with the protein source may influence calcium retention.

19. Abraham, G. E., et al. Effect of vitamin B6 on plasma red blood cell levels in premenopausal women. Ann Clin Lab Sci. 11(4):333-336, 1981.
After four weeks of vitamin B6 supplementation (200 mg per day), red blood cell magnesium levels returned to normal.

20. Holman, P. Pyridoxine – vitamin B-6. Journal of the Australasian College ofNutritional and Environmental Medicine. 14(1), 1995.
Vitamin B6 appears to facilitate the transport of magnesium across cell membranes.

Oral consumption of 200 mg of vitamin B6 per day has been demonstrated to significantly improve red blood cell and plasma magnesium levels.

21. Turnlaud, J. R., et al. Vitamin B6 depletion followed by repletion with animal- or plant-source diets in calcium and magnesium metabolism in young women. The American Journal of Clinical Nutrition. 56(5):905-10, 1992.

An 84-98-d study was conducted in young women to determine the effect of vitamin B6-deficient diets on calcium and magnesium metabolism. A vitamin B6-deficient formula diet fed initially was followed by either animal- or plant-source protein food diets containing four increasing amounts of vitamin B-6. Calcium balance was negative during vitamin B6 depletion. Serum calcium was higher and calcium balance negative with the plant protein diets. Magnesium balance was negative during vitamin B6 depletion due to increased urinary magnesium excretion. Urinary calcium decreased during vitamin B-6 depletion and increased as dietary vitamin B6 increased. Urinary oxalate was significantly higher at the end than at the beginning of vitamin B6 depletion and was higher with plant than animal protein diets. The results suggest that vitamin B6 depletion may alter calcium and magnesium metabolism and that dietary components associated with the protein source may influence calcium retention.

22. Varela-Mreiras, G., et al. Effect of chronic choline deficiency on liver folate content and distribution. J Nutr Biochem. 3:519-522, 1992.
23. Jacob, R. A., et al. Folate nutriture alters choline status of women and men fed low choline diets. Journal of Nutrition. 129(3):712-717, 1999.

Choline and folate share methylation pathways and, in studies of rats, were shown to be metabolically inter-related. To determine whether choline status is related to folate intake in humans, we measured the effect of controlled folate depletion and repletion on the plasma choline and phosphatidylcholine concentrations of 11 healthy men (33-46 y) and 10 healthy women (49-63 y) fed low-choline diets in two separate metabolic unit studies. Total folate intake was varied by supplementing low folate (25 and 56 microg/d for men and women, respectively) and low choline (238 and 147 mg/d for men and women, respectively) diets with pteroylglutamic acid for 2-6 wk following folate- depletion periods of 4-5 wk. The low folate/choline intakes resulted in subclinical folate deficiencies; mean plasma choline decreases of 28 and 25% in the men and women, respectively; and a plasma phosphatidylcholine decrease of 26% in the men ($P < 0.05$). No functional choline deficiency occurred, as measured by serum transaminase and lipid concentrations. The decreases in choline status measures returned to baseline or higher upon moderate folate repletion and were more responsive to folate repletion than plasma folate and homocysteine. Feeding methionine supplements to the men did not prevent plasma choline depletion, indicating that folate is a more limiting nutrient for these methylation pathways. The results indicate that 1) choline is utilized as a methyl donor when folate intake is low, 2) the de novo synthesis of phosphatidylcholine is insufficient to maintain choline status when intakes of folate and choline are low, and 3) dietary choline is required by adults in an amount > 250

mg/d to maintain plasma choline and phosphatidylcholine when folate intake is low.

24. Dean, W. & Morgenthaler, J. Smart Drugs & Nutrients. B & J Publications, Santa Cruz, California, USA. 1990:155.

25. Greenwell, I. Enhancing cognitive function. Life Extension. 6(5), 2000. The brain utilizes vitamin B5 for the conversion of choline into the neurotransmitter, acetylcholine.

26. Erb, C., et al. Enhancement of brain choline levels by nicotinamide: mechanism of action. Neuroscience Letters. 249(2-3):11

27. Brewer, G. J., et al. Initial therapy of patients with Wilson's disease with tetrathiomolybdate. Arch Neurol. 48:42-47, 1991.

28. Wapnir, R. A. Copper absorption and bioavailability. American Journal of Clinical Nutrition. 67(Supplement):1054S-1060S, 1998.

29. Molybdenum [monograph]. Alternative Medicine Review. 11(2):156-161, 2006.

Molybdenum in the form of tetrathiomolybdate complexes with copper and protein, resulting in decreased copper levels. High molybdenum intake (dietary or supplemental) may interfere with copper absorption and likewise, high copper intake may interfere with molybdenum absorption.

30. Fischer, P. W. F., et al. The effect of dietary zinc on intestinal copper absorption. American Journal of Clinical Nutrition. 34(9):1670-1675, 1981.

Everted duodenal segments, tied into sacs, taken from animals fed different amounts of zinc were used to investigate the antagonistic effect of dietary zinc on copper absorption. The intestinal segments taken from animals fed low amounts of zinc transferred more copper from a nutrient medium across the mucosal cells than did intestines from rats fed high levels of zinc. The mucosal cells from animals fed low amounts of zinc retained less copper than the cells from animals fed high amounts of the element. This retained copper was bound to a protein fraction having a molecular weight similar to that of metallothionein. The data suggest that zinc exerts its antagonistic effect by inducing the synthesis of a copper-binding ligand, probably a thionein, in the mucosal cells which sequesters copper from the nutrient medium, making it unavailable for serosal transfer. This may be a possible mechanism by which dietary zinc decreases copper absorption and leads to a decreased copper status.

31. Finley, E. G., et al. Influence of ascorbic acid supplementation on copper status in young adult men. American Journal of Clinical Nutrition. 37(4):553-556, 1983.

The influence of ascorbic acid supplementation on the copper status of young adult men was investigated. Subjects consuming self-selected diets took 500 mg of ascorbic acid

32. Kelleher, S. L., et al. Zinc supplementation reduces iron absorption through age- dependent changes in small intestine iron transporter expression in suckling rat pups. Journal of Nutrition. 136(5):1185-1191, 2006.Department of Nutritional Biology, University of California, Davis, CA, USA.

Zinc (Zn) supplementation negatively affects iron (Fe) absorption; however, the molecular mechanisms are not understood. The authors determined effects of Zn supplementation during mid- and late infancy on intestinal Fe transport mechanisms using a suckling rat model. Suckled rat pups were supplemented with 0 (control), 300 (low), or 750 (high) mug Zn/d until weaning at postnatal day (PN) 20. At mid- (PN10) and late (PN20) infancy, tissue Fe distribution, Fe absorption, intestine DMT1, ferroportin-1 (FPN) and hephaestin expression, and localization and liver hepcidin expression were measured. During early infancy, DMT1 and FPN were localized intracellularly. Negative effects of Zn supplementation on Fe absorption were associated with increased small intestine Fe retention, decreased hephaestin, and increased FPN expression. During late infancy, both DMT1 and FPN were appropriately localized to the apical and basolateral membrane, respectively, and negative effects of Zn supplementation on Fe absorption were absent. Although FPN protein level was lower in Zn-supplemented pups, hephaestin protein level was increased, which may have facilitated enhanced Fe efflux. These results indicate that Zn supplementation reduced Fe absorption during early infancy as a consequence of increased intestinal Fe retention due to reduced hephaestin levels. These effects were age-dependent, further demonstrating that Fe transport regulation is not fully developed until weaning, which may have important implications regarding the safety and efficacy of Zn supplementation programs for infants.

33. Olivares, M., et al. Acute inhibition of iron absorption by zinc. Nutrition Research. 27(5):279-282, 2007.

Iron and zinc deficiencies are the most common nutritional deficiencies worldwide. Combined zinc and iron supplementation is one of the strategies used to prevent these deficiencies. Zinc has an inhibitory effect on iron absorption. The objective of the study was to determine the duration of the inhibitory effect of zinc on iron absorption. Fifteen healthy subjects were selected to participate in the study. Subjects received a water solution with 0.5 mg of elemental iron, as ferrous sulfate, given alone or with zinc (11.71 mg), as zinc sulfate, in a molar ratio 20:1 zinc to iron, provided simultaneously with iron or 30 and 60 minutes before iron administration. The double radioisotopic technique was used to measure iron absorption. An inhibitory effect of zinc on iron absorption was observed when both mineral were given simultaneously; however, this inhibitory effect was not observed when zinc was administered 30 or 60 minutes before (analysis of variance for repeated measures, $F = 5.96$, $P < .002$; Scheffé post hoc test, $P <$

.006). In conclusion, zinc administration with iron in aqueous solution leads to the inhibition of iron bioavailability. However, this inhibitory effect lasts less than 30 minutes. The timing of this negative interaction should be considered for supplementation programs with both minerals.

34. Lemann, J., Jr., et al. Potassium administration reduces and potassium deprivation increases urinary calcium excretion in healthy adults. Kidney Int.39(5):973-983, 1991. Department of Medicine, Medical College of Wisconsin, Milwaukee, USA.

This study was undertaken to evaluate the effects of dietary potassium (K) intake, independent of whether the accompanying anion is Cl- or HCO3-, on urinary Ca excretion in healthy adults. The effects of KCl, KHCO3, NaCl and NaHCO3 supplements, 90 mmol/day for four days, were compared in ten subjects fed normal constant diets. Using synthetic diets, the effects of dietary KCl-deprivation for five days followed by recovery were assessed in four subjects and of KHCO3-deprivation for five days followed by recovery were assessed in four subjects. On the fourth day of salt administration, daily urinary Ca excretion and fasting UCa V/GFR were lower during the administration of KCl than during NaCl supplements (delta = -1.11 +/- 0.28SEM mmol/day; P less than 0.005 and -0.0077 +/- 0.0022 mmol/liter GFR; P less than0.01), and lower during KHCO3 than during control (-1.26 +/- 0.29 mmol/day; P lessthan 0.005 and -0.0069 +/- 0.0019 mmol/liter GFR; P = 0.005). Both dietary KCl and KHCO3 deprivation (mean reduction in dietary K intake -67 +/- 8 mmol/day) were accompanied by an increase in daily urinary Ca excretion and fasting UCaV/GFR that averaged on the fifth day +1.31 +/- 0.25 mmol/day (P less than 0.005) and +0.0069 +/- 0.0012 mmol/liter GFR (P less than 0.005) above control. Both daily urinary Ca excretion and fasting UCaV/GFR returned toward or to control at the end of recovery. These observations indicate that: 1) KHCO3 decreases fasting and 24-hour urinary Ca excretion; 2) KCl nor NaHCO3, unlike NaCl, do not increase fasting or 24-hour Ca excretion and 3) K deprivation increases both fasting and 24-hour urinary Ca excretion whether the accompanying anion is Cl- or HCO3-. The mechanisms for this effect of K may be mediated by: 1) alterations in ECF volume, since transient increases in urinary Na and Cl excretion and weight loss accompanied KCl or KHCO3 administration, while persistent reductions in urinary Na and Cl excretion and a trend for weight gain accompanied K deprivation; 2) K mediated alterations in renal tubular phosphate transport and renal synthesis of 1.25-(OH)2-vitamin D, since KCl or KHCO3 administration tended to be accompanied by a rise in fasting serum PO4 and TmPO4 and a fall in fasting UPO4 V/GFR, a all in serum 1,25-(OH)2-D and a decrease in fasting UCa V/GFR, while dietary KCl or KHCO3 deprivation were accompanied by a reverse sequence.

35. Lemann, J., Jr., et al. Potassium causes calcium retention in healthy adults.Journal of Nutrition. 123(9):1623-1626, 1993. Department of Medicine, Medical College of Wisconsin, Froedtert Memorial Lutheran Hospital, Milwaukee, USA.

The administration of 60 mmol/d of KHCO3 to healthy adults reduced urinary calcium excretion by 0.9 mmol/d and caused calcium balance to become equivalently more positive. Other studies showed that 90 mmol/d of KHCO3 reduced both daily and fasting urinary calcium excretion rates, whereas deprivation of either KCl or KHCO3, using synthetic diets, was accompanied by increased daily and fasting urinary calcium excretion rates. A significant inverse relationship between the changes in urinary calcium and the changes in urinary potassium was observed: delta urinary Ca (mmol/d) = 0.29-0.015 delta urinary K (mmol/d); r = -0.65. Correlative evaluation of additional data suggested that the fall in urinary calcium during potassium administration may be related to the natriuretic effects of potassium, resulting in ECF-volume contraction or to potassium-induced phosphate retention and suppression of calcitriol synthesis, or to both mechanisms.

36. Cook, J. D., et al. Calcium supplementation: Effect on iron absorption.American Journal of Clinical Nutrition. 53(1):106-111, 1991.

The influence of calcium supplements on the absorption of dietary nonheme iron and of iron supplements was evaluated in 61 normal volunteer subjects by use of a double- radioisotope technique. When taken without food, calcium carbonate did not inhibit the absorption of ferrous sulphate with doses of either 300 mg Ca and 37 mg Fe or 600 mg Ca and 18 mg Fe. However, at the latter levels, calcium citrate and calcium phosphate reduced iron absorption significantly by 49% and 62%, respectively. All calcium supplements inhibited absorption of the iron supplement when taken with food. The absorption of dietary nonheme iron was also inhibited by all three supplements. This inhibition was less pronounced from a meal of high iron availability and low calcium content (28%) than from a breakfast meal of low iron availability and high calcium content (55%). These results suggest that taking regular calcium supplements with meals makes it more difficult for women to meet their daily iron requirement.

37. Linder, M. C. Nutrition and metabolism of vitamins. In: Nutritional Biochemistry and Metabolism, 2nd Edition. Maria C. Linder (editor). Simon& Schuster, Connecticut, USA, 1991:146.

Vitamin C plays an important role in iron metabolism. It enhances intestinal iron absorption and transfer into the blood. It may also be involved in the mobilization of stored iron, especially from hemosiderin in the spleen.

38. Monsen, E. R. Ascorbic acid: An enhancing factor in iron absorption. In: Nutritional Bioavailability of Iron. American Chemical Society, 1982:85-95.

39. Fishman, S. M., et al. The role of vitamins in the prevention and control of anemia. Pub Health Nutr. 3(2):125-150, 2000.Division of Human Nutrition, Johns Hopkins School of Hygiene and Public Health, Baltimore, MD, USA.

While iron deficiency is regarded as the major cause of nutritional anaemia, changes in vitamins A, B12, C and E, folic acid and riboflavin status have also been linked to its development and control. Riboflavin enhances the haematological response to iron, and its deficiency may account for a significant proportion of anaemia in many populations.

40. Powers, H. J. Riboflavin (vitamin B-2) and health. American Journal of Clinical Nutrition. 77(6):1352-1360, 2003. Centre for Human Nutrition, The University of Sheffield, Sheffield, United Kingdom.

There is reasonably good evidence that poor riboflavin status interferes with iron handling and contributes to the etiology of anemia when iron intakes are low. Various mechanisms for this have been proposed, including effects on the gastrointestinal tract that might compromise the handling of other nutrients.

41. Polla, A. Therapy by taking away: the case of iron. Biochem Pharmacol. 57(12):1345-1349, 1999.

The recent finding of the beneficial effects of iron deprivation in the outcome of muscle necrosis in an animal model of genetic myopathy served as the basis of this commentary. Here, "taking away" iron by controlled dietary deprivation is proposed as a reasonable, feasible, cheap, and efficient clinical approach to many diverse diseases, all of which have a free radical component. Indeed, iron potentiates the generation of the highly reactive and toxic hydroxyl radical, and, thus, of oxidative damage. Iron deprivation may represent the first really efficient antioxidant, preventing oxidative stress in all subcellular compartments, tissues, and organs. Iron/iron deprivation also modulates programmed cell death (apoptosis), which should be the subject of further studies to better define the mechanisms mediating these complex effects. Related to its antioxidant effects, iron deprivation may find applications in the anti-aging field, whether programmed or premature aging, and whether in cosmetics or in gerontology.

42. Polla, A., et al. Iron as the malignant spirit in successful ageing. Ageing Research Reviews. 2:25-37, 2002.

Decreased iron stores contribute to increased life expectancy.

43. Wilgus, H. S., et al. Factors affecting manganese utilization in the chicken. Journal of Nutrition. 18:35, 1939.

Calcium impairs manganese utilization.

44. Freeland-Graves, J. H. Manganese: an essential nutrient for humans. Nutrition Today. 23:13-19, 1989.

45. Homson, A. B. R., et al. Interrelation of intestinal transport system for manganese and iron. J Labs Clin Med. 73:6422, 1971.

Iron impairs the utilization of manganese.

46. Wilgus, H. S., et al. Factors affecting manganese utilization in the chicken. Journal of Nutrition. 18:35, 1939.

Phosphorus impairs manganese utilization.

47. Finley, E. G., et al. Influence of ascorbic acid supplementation on copper status in young adult men. American Journal of Clinical Nutrition. 37(4):553-556, 1983.

The influence of ascorbic acid supplementation on the copper status of young adult men was investigated. Subjects consuming self-selected diets took 500 mg of ascorbic acid with each meal (1,500 mg/day) for 64 days. Blood samples were obtained at 0, 28, 52, and 64 days in order to determine serum copper and serum ceruloplasmin. Each subject thus served as his own control. Analyses were repeated 20 days after the ascorbic acid supplement was terminated. Serum ceruloplasmin activity was significantly reduced (p less than 0.01) at every data point throughout the ascorbic acid supplementation period. A similar but nonsignificant trend was observed for serum copper. Furthermore there was a significant increase (p less than 0.01) in serum copper concentration 20 days after the supplementation period. Although observed effects occurred within physiological ranges of normal values, this study confirms that a high ascorbic acid intake is antagonistic to copper status of men as has been demonstrated in laboratory animals.

48. Simon, J. A., et al. Relation of serum ascorbic acid to serum vitamin B12, serum ferritin, and kidney stones in US adults. Arch Intern Med.159(6):19-24, 1999.

Concern has been raised that high levels of ascorbic acid consumption may lead to potential adverse effects, such as vitamin B12 deficiency, iron overload, and kidney stones. This study examined the relation of serum ascorbic acid level, which reflects intake, to serum vitamin B12 level, serum ferritin level, and kidney stones. Data collected on a random sample of the US population enrolled in the Second National Health and Nutrition Examination Survey, 1976-1980 was analyzed using linear and logistic regression models. Serum ascorbic acid, serum vitamin B12, hemoglobin, red blood cell mean corpuscular volume (MCV), and serum ferritin levels were measured using standardized protocols. History of kidney stones was determined by self-report. After multivariate adjustment, serum ascorbic acid level was associated with higher serum vitamin B12 levels among women in regression models that assumed a linear relationship; each 57-pmol/L (1.0-mg/dL) increase in serum ascorbic acid level was independently

associated with a serum vitamin B12 level increase of 60 pmol/L. Among men, serum ascorbic acid levelwas marginally associated with higher serum vitamin B12 levels: each 57- micromol/L (1.0-mg/dL) increase in serum ascorbic acid level was associated with a serum vitamin B12 level increase of 27 pmol/L (36 pg/mL). In addition, serum ascorbic acid level was not associated with correlates of vitamin B12 deficiency, such as higher MCV levels, macrocytosis or lower hemoglobin concentrations.

49. Hegsted, M., et al. Effect of boron on vitamin D deficient rats. Biol Trace Elem Res. 28(3):243-255, 1991. School of Human Ecology, Department of Experimental Statistics, Louisiana Agricultural Experiment Station, Louisiana State University Agricultural Center, Baton Rouge, USA.

The effects of different levels of dietary boron were determined in vitamin D deficient rats. Vitamin D deficient diets containing either 0.158 ppm or 2.72 ppm of boron were fed to rats for 11 w, and calcium, magnesium, and phosphorus apparent absorption and balance were measured in the twelfth week. Higher apparent absorption and balance values for calcium and phosphorus were observed in the rats with higher dietary boron, but very few differences were seen in body wt, organ wt, and bone parameters. Balance measurements represented the present status of the rats after 12 w on the diets, but other measurements represented an accumulation over the lifetime of the rat, including a suckling period with ample vitamin D and boron. The data demonstrated that when rats are vitamin D deficient, as indicated by hypocalcemia, the level of boron in the diet affects mineral balance.

50. Nielsen, F. H. How should dietary guidance be given for mineral elements with beneficial actions or suspected of being essential? Journal of Nutrition.126(9 Supplement):2377S-2385S, 1996.

Boron deficiency in chickens increases the requirement for vitamin D

51. Johansson, S., et al. Vitamin A antagonizes calcium response to vitamin D in man. J Bone Miner Res. 16(10):1899-1905, 2001. Department of Medical Sciences, University Hospital, Uppsala, Sweden.

For unknown reasons, the highest incidence of osteoporosis is found in northernEurope. In these populations, the sunlight exposure is limited and the vitamin A intake is high. The interaction between vitamin A and D has been the subject of several in vitro and animal studies. The authors have studied the acute effects of vitamin A and D on calcium homeostasis in 9 healthy human subjects. They compared the effect of (i) 15 mg of retinyl palmitate, (ii) 2 microg of 1,25-dihydroxyvitamin D3 [1,25(OH)2D3], (iii) 15 mg of retinyl palmitate plus 2 microg of 1,25(OH)2D3, and (iv) placebo in a double-blind crossover study. The subjects took vitamin preparations at 10:00 p.m. and the following day blood samples were collected five times from 8:00 a.m. to 4:00 p.m. Serum levels of1,25(OH)2D3 and retinyl esters increased (1.7-fold and 8.3-fold, respectively; $p < 0.01$). As expected, serum calcium (S-calcium) increased (2.3%; $p < 0.01$) and S-parathyroid hormone (PTH) decreased (-32%; $p < 0.05$) after 1,25(OH)2D3 intake. In contrast, retinyl palmitate intake resulted in a significant decrease in S- calcium when taken alone (-1.0%; $p < 0.05$) and diminished the calcium response to 1,25(OH)2D3 after the combined intake (1.4%; $p < 0.01$). S-PTH was unaffected by retinyl palmitate. No significant changes in serum levels of the degradation product of C-telopeptide of type I collagen (CrossLaps), or U-calcium/creatinine levels were found. An intake of vitamin A corresponding to about one serving of liver antagonizes the rapid intestinal calcium response to physiological levels of vitamin D in man.

52. Cannell, J. J., et al. Use of vitamin D in clinical practice. Alternative Medicine Review. 13(1):6-20, 2008.

Vitamin A antagonizes the action of vitamin D, and high retinol intake thwarts vitamin D's protective effect on distal colorectal adenoma.

53. Meintzer, R., et al. Vitamin D and magnesium absorption. Journal of Nutrition. 56:285-294, 1955.

Vitamin D increases the absorption of dietary magnesium. During vitamin D deficiency, magnesium requirements increase. On the other hand, excessive intake of vitamin D results in increased urinary losses of magnesium, thereby increasing magnesium requirement.