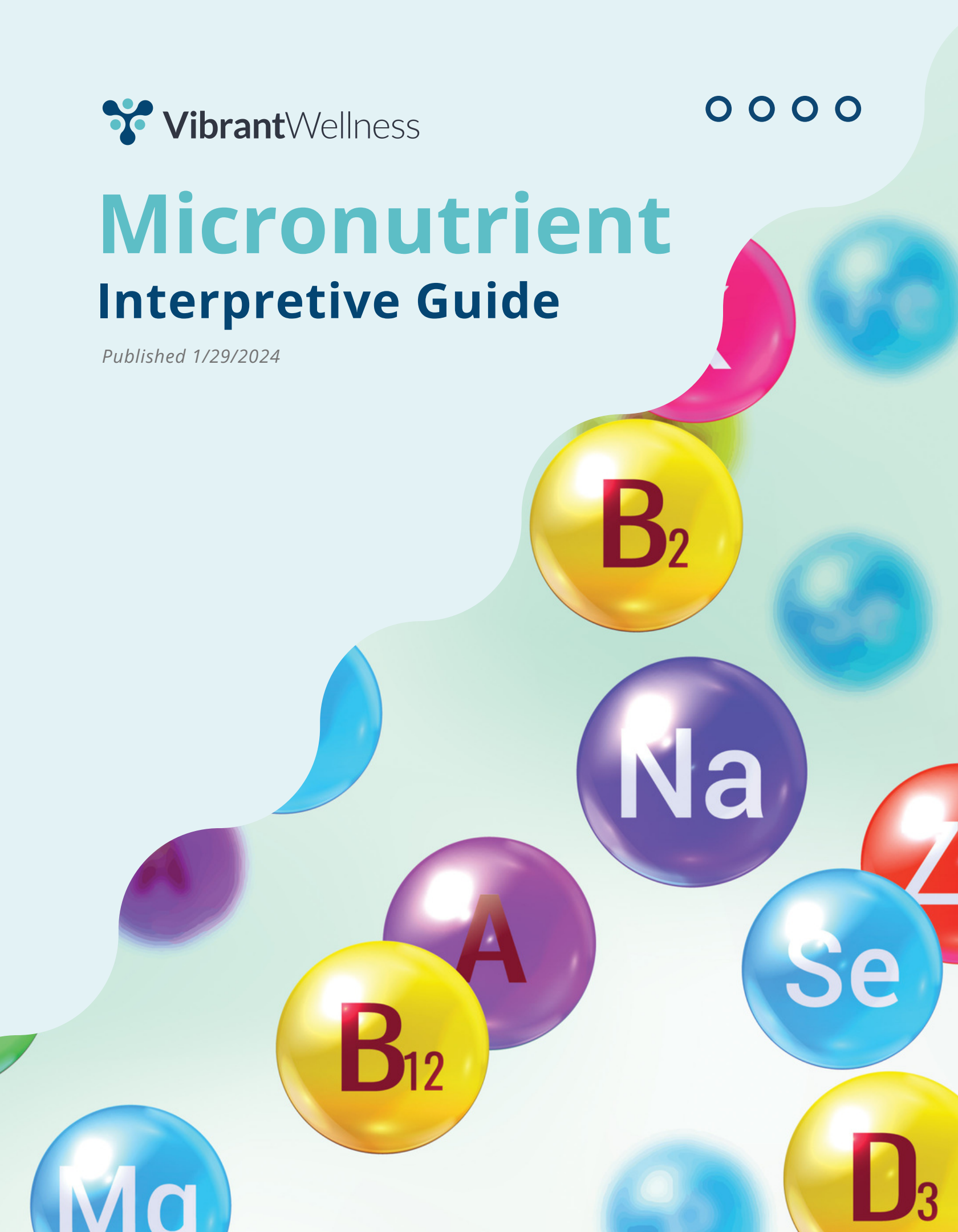
VibrantWellness

Micronutrient

Interpretive Guide

Published 1/29/2024



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The Micronutrient Panel is a comprehensive assessment of extra- and intracellular levels of vitamins, minerals, co-factors, amino acids, metabolites, antioxidants, and essential fatty acids. It provides the most complete and accurate picture of a patient's micronutrient status.

Interpreting the Micronutrient Panel

Serum results reflect nutrient status within the past week.
Cellular results reflect nutrient status within the past **3-4 *weeks* for White Blood Cells (WBC)** and **3-4 *months* for Red Blood Cells (RBC).**

Factors that can affect nutrient status:

- A change in diet or exercise regime.
- Initiation, discontinuation, or change in dose of medication(s).
- Initiation, discontinuation, or change in dose of supplement(s).
- Experiencing a major life event, trauma, or change in stress level.

Common scenarios and how to address them:

1. Low Serum + Normal Cellular
   - Long-term (cellular) nutrient status is optimal, but short-term (serum) needs improvement.
   - Try:
     i. Increasing dietary intake of the nutrient.
     ii. Changing supplement dose, form, or function.
     iii. Checking for drug-nutrient depletions.
2. Low Serum + Low Cellular
   - Short and long-term nutrient status is not optimal, suggesting both low dietary intake and intestinal and cellular malabsorption as possible causes.
   - Try:
     i. Increasing dietary intake of the nutrient.
     ii. Increasing supplement dosage.
     iii. Checking for drug-nutrient depletion.
     iv. Conducting follow-up testing to identify the root cause(s) of maldigestion and malabsorption.
3. Normal/High Serum + Low Cellular
   - Short-term status of micronutrients is optimal, but cellular absorption may be an issue.
   - Try:
     i. Increasing dietary intake of the nutrient.
     ii. Increasing supplement dosage.
     iii. Considering synergistic nutrients for cellular absorption.
     iv. Considering levels of oxidative stress on nutrient depletion.
     v. Conducting follow-up testing to identify the source of maldigestion and malabsorption.

TIP: Check out the WBC Count to assess if the values are skewed!

- LOW WBC counts may falsely "concentrate" the analytes, resulting in false highs.
- HIGH WBC counts may falsely "dilute" the analytes, resulting in false low.

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess/ Toxicities
Vitamin A ***Vitamins*** ***Serum WBC*** Vitamin A is an essential, fat-soluble vitamin necessary for vision, immune function, and cell growth. Other vital roles that vitamin A plays in the body include gene transcription, hematopoiesis, and antioxidant activity.	Adult males: 900 mcg RAE/day Adult females: 700 mcg RAE/day RDAs are the amount needed to prevent chronic deficiency, but individuals may need more for optimal health. These measurements are the equivalent of 2500 IU/day for women and 3000 IU/day for men of pre-formed vitamin A sources (animal sources). The upper intake level (UL) for adult vitamin A is 3,000 mcg RAE/day.	• Beef liver • Sweet potato • Spinach • Pumpkin • Carrots • Herring	**Inadequate intake** **Malabsorption Disorders:** Conditions affecting the digestive tract can hinder vitamin A absorption, including: • Celiac disease • IBD • Pancreatic insufficiency • Liver disorders (e.g., cirrhosis, NAFLD) • Gallbladder disorders (e.g., holecystitis, gallstones) • Short bowel syndrome • Giardiasis **Alcoholism:** Alcohol abuse can interfere with vitamin A utilization. **Zinc deficiency:** Zinc helps convert inactive vitamin A (retinol) into its active form (retinoic acid) within the retina. **Hypothyroidism:** retinoids seem to play a role in the development and maturation of the thyroid cell phenotype.[2]	Excess vitamin A levels, known as hypervitaminosis A, can occur when the body accumulates 10,000 IU or more daily over a prolonged period.[3] Vitamin A is a fat-soluble vitamin, and excessive amounts are stored in the body rather than excreted. **High Dietary Intake:** Consistently consuming large quantities of vitamin A-rich foods like fish liver, eggs, dairy products can lead to an accumulation of vitamin A. **Chronic Overconsumption of Fortified Foods:** Regularly consuming excessive amounts of highly fortified foods, such as certain breakfast cereals, can contribute to elevated vitamin A levels. **Consumption of Animal Liver:** Consuming large amounts of animal liver can result in elevated vitamin A levels as the liver is rich in vitamin A. **High-Dose Supplements:** Consuming vitamin A supplements at doses significantly higher than the recommended daily allowance (RDA) over a prolonged period can lead to excess vitamin A levels. **Consumption of High-Dose Vitamin A Medications:** Certain prescription medications, like isotretinoin (used to treat severe acne), contain high doses of vitamin A and can lead to elevated levels when used improperly. **Liver Dysfunction:** Liver disorders, such as hepatitis or cirrhosis, can impair the liver's ability to metabolize and store vitamin A properly, resulting in elevated levels.
Vitamin B1 **Vitamins** **Serum WBC** Vitamin B1 (Thiamine) is a water-soluble vitamin essential for energy metabolism, nerve function, and maintaining proper heart and muscle function. It acts as a coenzyme in the breakdown and transformation of carbohydrates, fats, and proteins to produce energy in the form of ATP.	Adult males: 1.2 mg /day Adult females: 1.1 mg/day Pregnancy: 1.4 mg Therapeutic intake of thiamin is commonly 25-100 mg/day. No UL for thiamin has been set.	• Fortified breakfast cereals • Enriched food products • Pork • Trout • Black beans • Mussels	**Inadequate intake** **Alcohol Abuse:** Alcohol interferes with thiamine absorption and utilization. **Malabsorption:** Conditions affecting the digestive system, such as gastric bypass surgery, can hinder thiamine absorption. Thiamin can become depleted or deficient from frequent consumption of thiaminases present in higher amounts in raw fish and tannins/tannic acid (tea and coffee). Thiamin is vulnerable to loss during cooking.	**Excessive Supplementation** The human body excretes excess thiamin in the urine. There is a lack of evidence of toxicity from high thiamin intake from food or supplements.

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess/ Toxicities
Vitamin B2 ***Vitamins Serum WBC*** Vitamin B2 (Riboflavin) Two critical coenzymes involved in energy metabolism are derived from riboflavin to participate in oxidation/reduction reactions. Riboflavin is also essential for the enzymes nitric oxide synthase (NOS) and glutathione reductase, the latter of which regenerates glutathione and is crucial for antioxidation/ detoxification.	Adult males: 1.1 to 1.3 mg Adult females 0.9 to 1.1 mg Pregnant: 1.4 mg Lactating 1.6 mg Typical levels of therapeutic riboflavin intake are 25-50 mg/day. No UL for riboflavin has been set.	• Beef liver • Fortified breakfast cereals, oats • Yogurt • Milk • Clams • Almonds • Swiss cheese	**Inadequate intake** **Malabsorption Disorders (e.g., celiac disease, Crohn's):** intestinal inflammation and damage can hinder absorption **Alcoholism** can lead to poor dietary intake and impaired absorption of riboflavin, contributing to deficiency. **Medications** such as some antidepressants and antipsychotics, can interfere with riboflavin absorption and utilization.	Most riboflavin is used immediately and not stored in the body, so excess amounts are excreted in the urine.[4] There are no adverse effects from high riboflavin intakes from foods or supplements up to 400 mg daily for at least three months.[5] **Overconsumption of Fortified Foods:** Consuming large quantities of foods fortified with riboflavin, such as breakfast cereals, can contribute to elevated riboflavin levels. **Excessive Riboflavin Supplements:** Taking high doses of riboflavin supplements above the recommended levels can lead to elevated riboflavin levels in the body. Excess dietary riboflavin, usually from supplements, can cause urine to become bright yellow.
Vitamin B3 ***Vitamins Serum WBC*** Vitamin B3 (Niacin) is extensively involved in metabolic reduction reactions through NAD-NADPH pathways. Over 200 enzymes in the human body require niacin. It is also involved in fatty acid synthesis, ATP synthesis, DNA repair, lowers cholesterol/LDL, and aids in circulation.	Adults: 13 to 20 mg Pregnant: 17 mg Lactating: 20 mg Niacin is often recommended therapeutically for lipid management. Niacin has been shown to lower LDL cholesterol, lipoprotein(a), triglyceride, and fibrinogen levels while raising HDL levels. Flushing can occur at high doses. Aspirin may help reduce flushing. Time-release niacin or no-flush niacin is not recommended for therapeutic treatment. Monitor liver function carefully with high dose niacin supplementation.	• Beef liver • Chicken breast • Marinara • Turkey breast • Salmon • Tuna • Pork • Brown rice • Peanuts • Fortified breakfast cereal	**Inadequate Intake** **Malabsorption Disorders (e.g., celiac disease, Crohn's):** intestinal inflammation and damage can hinder absorption **Medications** such as isoniazid (used for tuberculosis treatment) can inhibit the conversion of tryptophan to niacin, potentially leading to lower niacin levels. **Alcoholism** can lead to poor dietary intake and impaired niacin absorption, contributing to a deficiency. Long-term inadequate niacin intake leads to pellagra, a condition characterized by niacin deficiency. It often occurs in regions with limited dietary diversity and reliance on corn-based diets.	The tolerable upper intake level (UL) for niacin for adults is 35 mg/day, based on flushing as the critical adverse effect.[6] **Excessive Supplementation:** Taking high doses of niacin supplements, often beyond the recommended levels, can lead to elevated niacin levels in the body. **Niacin Therapy:** High doses of niacin may be prescribed for certain medical conditions, such as high cholesterol, which can result in elevated niacin levels. **Energy drinks** can contain large quantities of vitamins, including niacin[7]

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess/ Toxicities
Vitamin B5 ***Vitamins Serum WBC*** Vitamin B5 (Pantothenic Acid) is part of the structural component of coenzyme A. It is also important for synthesis of red blood cells, sex hormones, adrenal hormones, and vitamin D. A significant function of B5 is to work with carnitine and CoQ10 for fatty acid oxidation/metabolism.	Take with food if administered orally Adults: 5 mg/day Pregnant: 6 mg/day Lactating: 7 mg/day Because the breakdown of B5 is metabolically slow and deficiency is rare, there is likely no need for supplementation.	• Beef liver • Fortified breakfast cereal • Shiitake mushrooms • Sunflower seeds • Chicken breast • Tuna • Avocado	**Inadequate intake:** Deficiency of B5 is very rare, however, in a diet that is high in biotin, or if high dose biotin supplementation occurs, B5 may become conditionally deficient due to competition for the same uptake receptor in the intestine.[8] **Malabsorption Disorders (e.g., celiac disease, Crohn's):** intestinal inflammation and damage can hinder absorption **Alcoholism** can lead to poor dietary intake and impaired B5 absorption, contributing to a deficiency.	There are currently no upper limits established since there have been no reports of vitamin B5 toxicity in humans with high intake.[9] Some individuals taking large doses of pantothenic acid supplements (e.g., 10 to 20 g/day) develop mild diarrhea and gastrointestinal distress, but the mechanism for this effect is not known.[1 8] **Overconsumption of Fortified Foods:** Consuming excessive amounts of foods fortified with pantothenic acid or pantothenic acid-containing supplements can lead to elevated pantothenic acid levels. **Excessive Supplements:** Taking high doses of pantothenic acid supplements, often beyond the recommended levels, can lead to elevated pantothenic acid levels in the body. **Pantothenic Acid Therapy:** High doses of pantothenic acid may be used in medical settings for specific conditions, which can result in elevated pantothenic acid levels. **Metabolic Disorders:** Certain rare genetic conditions (e.g., Pantothenate Kinase-Associated Neurodegeneration) can disrupt pantothenic acid metabolism, potentially leading to elevated pantothenic acid levels.
Vitamin B6 **Vitamins SerumWBC** Vitamin B6 (Pyridoxine) There are 117 known vitamin B6-dependent enzymes important for metabolic function. Most are aminotransferase reactions (transfer of amino groups for protein metabolism). Vitamin B6 also aids in the absorption of Vitamin B12. Vitamin B6 converts tryptophan to serotonin and can modulate steroid hormone activity. Vitamin B6 is involved in the methylation cycle and clearance of homocysteine, along with folate and B12.	Adults: 1.3 mg Adults over age 51: • Males: 1.7 mg • Females: 1.5 mg Pregnant: 1.9 mg Lactating: 2.0 mg	• Chickpeas • Beef liver • Yellowfin tuna • Sockeye salmon • Chicken breast • Fortified breakfast cereal • Potatoes • Turkey • Banana	**Inadequate intake** **Malabsorption Disorders (e.g., celiac disease, Crohn's):** intestinal inflammation and damage can hinder absorption **Certain medications** such as isoniazid (used for tuberculosis treatment) and penicillamine (used for Wilson's disease), can interfere with vitamin B6 metabolism and lead to low levels. **Renal impairment:** Chronic renal failure patients, especially those on hemodialysis or peritoneal dialysis, often have low serum pyridoxal-5′-phosphate (PLP) concentrations and may exhibit signs and symptoms of vitamin B6 deficiency. **Autoimmune disorders:** Patients with autoimmune diseases such as rheumatoid arthritis have increased catabolism of vitamin B6, indicating increased metabolic needs for B6.	Board of the Institute of Medicine set the tolerable upper intake level (UL) for pyridoxine at 100 mg/day for adults.[8] **Elevated B6 may be a result of therapies for health/medical conditions.[10]** • Pyridoxine/vitamin B6-dependency syndromes • Isoniazid (INH)-induced B6 deficiency • INH-induced neuropathy prophylaxis • INH poisoning • Premenstrual syndrome (PMS)

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess/ Toxicities
Folate ***Vitamins Serum*** Folate (Vitamin B9) is the naturally occurring form found in foods; folic acid is the supplement/synthetic form of B9. Folate is more bioavailable than folic acid. Once in circulation, folate gets methylated. Using the 5-Methyl-tetrahydrofolate (5-MTHF) form of folate reduces masking of B12 deficiency and reduces metabolic defects caused by the MTHFR snp.[11]	As dietary folate equivalents (DFE), 1 DFE = 1 mcg food folate = 0.6 mcg of folic acid from fortified food or as a supplement consumed with food = 0.5 mcg of a supplement taken on an empty stomach. Adults: 400 mcg DFE Pregnant: 600 mcg DFE Lactating: 500 mcg DFE Doses of folate ranging from 400 mcg to 10 mg have been used clinically. A more common therapeutic range is 400 to 1000 mcg per day.	• Beef liver • Green leafy vegetables (esp. spinach and mustard greens) • Black-eyed peas • Breakfast cereals • White rice • Asparagus • Brussels sprouts	**Inadequate Intake** **Malabsorption** **Digestive Disorders:** Conditions affecting the digestive tract, such as celiac disease, Crohn's disease, and certain gastrointestinal surgeries, can impair folate absorption. **Alcoholism:** Chronic alcohol abuse can damage the lining of the intestines, reducing the absorption of folate **Increased Requirements** **Pregnancy and Breastfeeding:** The demand for folate increases during pregnancy and breastfeeding, and if dietary intake is insufficient, it can lead to a deficiency. **Growth Phases:** Rapid growth periods, such as adolescence, may require higher folate intake. **Medications and Medical Treatments:** Some medications like methotrexate, anticonvulsants, antacids, and oral contraceptives can interfere with folate metabolism and cause deficiency. **Hemodialysis:** People undergoing hemodialysis for kidney disease can experience increased folate loss and may require supplementation. **Medical Conditions** **Anemia:** Individuals with certain types of anemia, such as megaloblastic anemia, may have an increased need for folate and can develop a deficiency. **Hemolytic Anemias:** Conditions where red blood cells are destroyed faster than the body can replace them may require additional folate. **Excessive Stress or Infection:** Certain medical conditions or severe stress can increase the body's folate requirements. **Genetic Factors** **MTHFR Gene Mutations:** Variants in the MTHFR gene can affect folate metabolism, potentially leading to reduced folate levels. **Aging:** Older adults may be at higher risk of folate deficiency due to reduced dietary intake, reduced absorption, and increased medication use.	Excess folate levels, though rare, can occur and may be attributed to various causes. Folate is a water-soluble B vitamin, and excess amounts are typically excreted by the body through urine. No adverse effects have been associated with the consumption of excess folate from food. Concerns regarding safety are limited to synthetic folic acid intake[8]. **High Dietary Intake:** Consuming very high amounts of folate-rich foods consistently over an extended period can result in elevated folate levels. **Overconsumption of Fortified Foods:** Consuming large quantities of fortified foods containing added folic acid can contribute to excess folate levels. **High-Dose Supplements:** Taking high doses of folate supplements, often more than the recommended daily allowance (RDA), can lead to elevated folate levels in the body. **Kidney Dysfunction** **Impaired Excretion:** Kidney dysfunction or impaired renal function can result in reduced excretion of folate, leading to its accumulation in the body. **Certain Medical Conditions** **Cancer:** Some types of cancer, particularly leukemias and cancers of the digestive tract, can lead to elevated folate levels, likely due to altered folate metabolism. **Epilepsy:** Long-term use of antiepileptic medications, such as phenytoin and phenobarbital, can interfere with folate metabolism and result in elevated folate levels. **Vitamin B12 Deficiency or Anemia** **Masking B12 Deficiency:** Excessive folate supplementation can mask symptoms of vitamin B12 deficiency or pernicious anemia, as it can correct the anemia without treating the underlying B12 deficiency. **Intestinal Microbiota:** Certain individuals may have gut bacteria that produce excessive folate, contributing to elevated levels.

Nutrient	RDA *Adults = 19+*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess/ Toxicities
Vitamin B12 ***Vitamins*** ***Serum WBC*** Vitamin B12 (cobalamin) is an important coenzyme in its active forms: methylcobalamin and adenosyl cobalamin. B12 facilitates the metabolism of folic acid through its primary role as a methyl donor. B12 requires intrinsic factor for absorption, which is calcium dependent. The role of vitamin B12 in the production of some neurotransmitters may also be evidenced by mood imbalance in susceptible individuals	Adults: 2.4 mcg Pregnant: 2.6 mcg Lactating: 2.8 mcg	• Beef liver • Clams • Nutritional yeast • Atlantic salmon • Chunk light tuna, canned • Ground beef • Milk • Yogurt, plain • Fortified breakfast cereals	**Malabsoprtion** • **Pernicious Anemia:** Pernicious anemia is an autoimmune condition where the body's immune system attacks cells in the stomach that produce a protein needed for B12 absorption. • **Celiac Disease:** Celiac disease can damage the intestines and hinder the absorption of B12 and other nutrients. • **Atrophic Gastritis:** Chronic inflammation and thinning of the stomach lining can impair B12 absorption. • Hypochlorhydria[12] • Oral contraceptive pill[13] • Chronic alcohol dependence • Obesity, pregnancy, preeclampsia, and eclampsia may also predispose someone to vitamin B12 deficiency[14] **Bariatric Surgery:** Certain weight loss surgeries, such as gastric bypass, can lead to reduced B12 absorption due to alterations in the digestive tract. **Medication** • **Proton Pump Inhibitors (PPIs) and H2 Blockers:** Long-term use of these medications for reducing stomach acid can hinder B12 absorption. • **Metformin:** A medication used to manage diabetes may reduce B12 levels in some individuals.	No toxic or adverse effects have been associated with large intakes of vitamin B12 from food or supplements in healthy people. When high doses of vitamin B12 are given orally, only a small percentage can be absorbed, which may explain the low toxicity. Because of the low toxicity of vitamin B12, no tolerable upper intake level (UL) has been set by the US Food and Nutrition Board.[8] Although cobalamin toxicity can be unexpected and unusual, one case report reminds us that the administration of any drug is not entirely safe.[15] **Excessive Supplements:** Supraphysiological serum B12 levels are more often a result of high-dose supplementation or IV injections. Per this paper[16], high or supraphysiological serum B12 levels without supplementation have been associated with many pathological conditions, including renal failure, hematological disorders, cancer, and hepatic or autoimmune diseases. All conditions may show elevated concentrations of B12 transport proteins. In addition, there may be an increased release of B12 in liver disease due to hepatic cytolysis and/or reduced B12 clearance. Thus, a high or supraphysiological serum B12 concentration without supplementation could be a diagnostic marker for severe underlying disease. Furthermore, very high serum B12 levels may be of prognostic significance. High levels have been frequently reported in critically ill patients. **Polycythemia Vera:** A rare blood cancer that causes the bone marrow to produce too many red blood cells can result in elevated B12 levels. Elevated serum B12 levels may also be associated with a functional deficiency of the vitamin due to a failure of cellular uptake or intracellular processing, trafficking, or utilization.
Vitamin C **Vitamins** **Serum WBC** Vitamin C (Ascorbic Acid) has a significant function as an antioxidant. It boosts immunity by increasing white blood cells, in addition to supporting regeneration of vitamin E. Vitamin C can also reduce atherosclerosis, stroke, and high blood pressure, and inflammation. It is necessary for optimal collagen production because of its role in the generation of connective tissue. Vitamin C is also an essential component of L-carnitine, which is necessary to metabolize fats into energy.	Adult males: 90 mg Adult Females: 75 mg Pregnant: 85 mg Lactating: 120 mg	• Red and green sweet peppers • Oranges • Grapefruit • Kiwifruit • Broccoli • Strawberries • Brussels sprouts • Tomatoes • Cantaloupe • Cabbage • Cauliflower	**Inadequate intake:** Vitamin C is most commonly depleted without sufficient dietary intake. The vitamin C pool in the body is usually depleted in 4 to 12. Ascorbic acid is affected by many factors that can impair absorption and its functions. Consuming fruits and vegetables regularly is the best way to prevent vitamin C deficiency.[17] Vitamin C levels can be depleted during times of severe oxidative stress since it is a cofactor required both in catecholamine biosynthesis and in adrenal steroidogenesis.[18]	A tolerable upper intake level (UL) for vitamin C was set at 2 g (2,000 mg) daily in order to prevent generally healthy adults from experiencing diarrhea and gastrointestinal disturbances. Such symptoms are not generally serious, especially if they resolve with temporary discontinuation of vitamin C supplementation. Several possible adverse health effects of very large doses of vitamin C have been identified, mainly based on in vitro experiments or isolated case reports, and include: • Genetic mutations • Congenital disabilities • Cancer • Atherosclerosis • Kidney stones • "Rebound scurvy" • Increased oxidative stress • Excess iron absorption • Vitamin B12 deficiency • Erosion of dental enamel However, none of these alleged adverse health effects have been confirmed in subsequent studies, and there is no reliable scientific evidence that doses of vitamin C up to 10 g/day in adults are toxic or detrimental to health.[8]

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess/ Toxicities
Vitamin D3 ***Vitamins*** ***SerumWBC*** Vitamin D3 (Cholecalciferol) is a fat-soluble vitamin obtained through sunlight, food, and supplement intake. It is then converted into 25-OH-D3 (which is what most standard labs run and is most commonly used to assess total vitamin D status). In the kidneys, 25-OH-D3 is converted into 1,25-OH-D3, aka calcitriol, the active form of vitamin D3. Vitamin D3 regulates the function of hundreds of genes, supports the immune system, supports the production and function of endocrine hormones, is essential for normal growth and development of bones and teeth, tightly regulates the levels of calcium and phosphorus being absorbed intestinally as well as released from bone, regulates cell differentiation and growth, and may play an important role in regulating mood. Patients who present with hypercalcemia, hyperphosphatemia, and low PTH may suffer from unregulated conversion of 25-OH-D3 to 1,25-OH-D3.	Adults: 600 IU Adults over age 70: 800 IU Pregnancy: 600-800 IU Lactating: 600-800 IU	• Cod liver oil • Rainbow trout • Sockeye salmon • White mushrooms • Milk, fortified	Vitamin D deficiency is very common in the U.S. **Inadequate intake** **Malabsorption Disorders:** Certain malabsorption syndromes such as celiac disease, short bowel syndrome, gastric bypass, inflammatory bowel disease, chronic pancreatic insufficiency, and cystic fibrosis may lead to vitamin D deficiency. Lower oral intake of vitamin D is more prevalent in the elderly population. **Decreased sun exposure:** About 50% to 90% of vitamin D is photochemically converted to vitamin D3 from 7-dehydrocholesterol in the skin via sunlight, while the rest comes from the diet. Twenty minutes of sunshine daily with over 40% of skin exposed is required to prevent vitamin D deficiency. Cutaneous synthesis of vitamin D declines with aging. Dark-skinned people have less cutaneous vitamin D synthesis. Decreased exposure to the sun, as seen in institutionalized individuals or prolonged hospitalizations, can also lead to vitamin D deficiency. Adequate sun exposure is reduced in individuals who use sunscreens consistently. **Decreased endogenous synthesis:** Individuals with chronic liver disease such as cirrhosis can have defective 25-hydroxylation, leading to deficiency of active vitamin D. Defects in 1-alpha 25-hydroxylation can be seen in hyperparathyroidism, renal failure, and 1-alpha hydroxylase deficiency. **Increased hepatic catabolism:** Medications such as phenobarbital, carbamazepine, dexamethasone, nifedipine, spironolactone, clotrimazole, and rifampin induce hepatic cytochrome P450 enzymes, which activate the degradation of vitamin D. End-organ resistance to vitamin D can be seen in hereditary vitamin D-resistant rickets.[19]	**Excessive Supplement Intake:** Hypervitaminosis D is rare and usually caused by excessive doses of vitamin D due to misuse of over-the-counter supplements or erroneous prescriptions. Intake levels less than 10,000 IU/day are unlikely to cause toxicity. However, Vitamin D toxicity has been observed in individuals taking over 50,000 IU/day for a few months.[8 20] Vitamin D toxicity induces abnormally high serum calcium concentration (hypercalcemia), which could result in bone loss, kidney stones, and calcification of organs like the heart and kidneys if untreated over a long period.[8] Supplementing vitamin K2 alongside vitamin D is recommended to keep blood calcium levels in homeostasis and prevent excess bone demineralization of calcium with higher vitamin D intake. Less commonly, **poisoning from exposure to rodenticides** containing cholecalciferol can also lead to vitamin D toxicity.[18] Vitamin D toxicity (hypervitaminosis D) has not been observed to result from sun exposure.[8]
Vitamin D, 25-OH ***Vitamins*** ***Serum*** 25-OH-D3 is a standard lab test that measures the inactive precursor to 1,25-OH-D3, a combination of two forms of vitamin D in the body: vitamin D2 and D3. 25-OH-D3 has a longer half-life in the blood than 1,25-OH-D3. Therefore, levels may differ from levels of active 1,25-OH-D3. Because 25-OH-D3 is a precursor to active forms of vitamin D (calcitriol), it is essential to note that it does not reflect overall active D3 levels but rather what is available for conversion if cofactors are sufficient.	n/a	See Vitamin D3 sources	**See Vitamin D3**	**See Vitamin D3**

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess/ Toxicities
Vitamin E ***Vitamins*** ***Serum WBC*** Vitamin E (Tocopherol or alpha-tocopherol) is a fat-soluble vitamin and is an important antioxidant that reduces the formation of reactive oxygen species (ROS) that result from fat oxidation. Vitamin E also regulates cell signaling, influences immune function, and inhibits coagulation, which can play a role in reducing atherosclerosis and lowering rates of ischemic heart disease.	Adults: 15 mg (22.4 IU) Pregnant: 15 mg Lactating: 19 mg The UL is set at 1000 mg/day in order to prevent interference in vitamin K clotting pathways. The only supplementary form of vitamin E that reverses deficiency symptoms is α-tocopherol. Alpha lipoic acid is an important cofactor that can aid in restoring vitamin E levels when depleted.	• Wheat germ oil • Sunflower seeds • Almonds • Sunflower oil • Safflower oil • Hazelnuts	In developed countries, it is unlikely that vitamin E deficiency occurs due to diet intake insufficiency. More common causes are: • **Premature low birth weight** infants weighing less than 1500 grams (3.3 pounds). **Malabsorption Disorders:** • **Disrupted fat malabsorption** as the small intestine requires fat to absorb vitamin E. • Patients with **cystic fibrosis** fail to secrete pancreatic enzymes to absorb vitamins A, D, E, and K. • **Short-bowel syndrome** patients may take years to develop symptoms. Surgical resection, mesenteric vascular thrombosis, and pseudo-obstruction are a few examples of this issue. • **Chronic cholestatic hepatobiliary disease** leads to decreased bile flow and micelle formation needed for vitamin E absorption. • **Crohn's disease,** exocrine pancreatic insufficiency, and liver disease may not absorb fat. **Genetics:** • **Mutations** in the tocopherol transfer protein, causing impaired fat metabolism. • **Abetalipoproteinemia**, an autosomal-recessive disease, causes lipoprotein production and transportation errors. • **Isolated vitamin E deficiency** syndrome, an autosomal recessive disorder of the chromosome arm 8q. In developing countries, the most common cause is inadequate vitamin E intake.[21]	Few side effects have been noted in adults taking supplements of less than 2,000 mg of α-tocopherol daily (either natural or synthetic vitamin E). The most worrisome possibility is that of impaired blood clotting, which increases the likelihood of hemorrhage in some individuals. The UL of 1,000 mg/day of α-tocopherol in any supplemental form (equivalent to 1,500 IU/day of RRR-α-tocopherol or 1,100 IU/day of all-rac-α-tocopherol) corresponds to the highest dose unlikely to result in hemorrhage in almost all adults.[8]
Vitamin K1 ***Vitamins*** ***Serum WBC*** Vitamin K1 (phylloquinone) is a fat-soluble vitamin and is present primarily in green leafy vegetables and is the main dietary form of vitamin K1. Vitamin K assists with blood clotting, supports the K1 formation of bone and bone matrix, and aids in glucose to glycogen conversion for storage in the liver.	Adult males: 120 mcg Adult females: 90 mcg Pregnant + Lactating: 90 mcg	• Natto • Collards • Turnip greens • Spinach • Kale • Broccoli • Soybeans • Carrot juice	Dietary deficiency of vitamin K1 is extremely rare unless there has been significant damage to the intestinal lining, such as in inflammatory bowel disorders (Crohn's, ulcerative colitis, etc.), liver disease, cystic fibrosis, and fat malabsorption disorders. Taking broad-spectrum antibiotics can reduce vitamin K1 production in the gut. Individuals with chronic kidney disease are at risk for vitamin K1 deficiency. Individuals with ApoE4 genotype may be at greater risk for low vitamin K1. Since vitamin K1 is a fat-soluble vitamin, following a chronically low-fat diet can inhibit absorption.	Although allergic reactions are possible, there is no known toxicity associated with high doses (dietary or supplemental) of the phylloquinone (vitamin K1) or menaquinone (vitamin K2) forms of vitamin K). No tolerable upper intake level (UL) has been established for vitamin K1.

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess/ Toxicities
Vitamin K2 ***Vitamins*** ***Serum WBC*** Vitamin K2 (a series of menaquinones) are predominantly of bacterial origin and are present in modest amounts in various animal-based and fermented foods. Vitamin K2 is the primary storage form of vitamin K in animals. Bacteria, e.g., Bacillus subtilis, in the colon, can convert K1 (from K plant-based foods) into vitamin K2. Vitamin K2 is necessary to prevent arterial calcification, which is done by activating matrix GLA protein (MGP). MGP in blood vessels inhibits soft tissue calcification. MGP needs to be carboxylated to work properly, and vitamin K2-MK7 plays a significant role in this carboxylation.	Studies suggest daily therapeutic doses of about 360-500 micrograms (mcg) of vitamin K2.	See Vitamin K1 sources	Since vitamin K is a fat-soluble vitamin, following a **chronically low-fat diet** can inhibit absorption. **Malabsorption disorders:** Dietary vitamin K deficiency is extremely rare unless there has been significant damage to the intestinal lining, such as in inflammatory bowel disorders (Crohn's, ulcerative colitis, etc.), liver disease, cystic fibrosis, and fat malabsorption disorders. **Broad-spectrum antibiotics** can reduce vitamin K production in the gut. Individuals with **chronic kidney disease** are at risk for vitamin K deficiency. Individuals with **ApoE4** genotype may be at greater risk for low vitamin K.	Although allergic reactions are possible, there is no known toxicity associated with high doses (dietary or supplemental) of the phylloquinone (vitamin K1) or menaquinone (vitamin K2) forms of vitamin K). No tolerable upper intake level (UL) has been established for vitamin K
Calcium ***Minerals*** ***Serum WBC*** Calcium is a mineral that is a major component of bones and teeth, is required for muscle contraction, nerve transmission, cellular metabolism, and aids in blood clotting.	Adults: 1,000 mg Adults age 51-70: 1,000 mg (Males), 1,200 (Females) Adults over age 70: 1,200 mg Pregnant & Lactating: 1,300 mg	• Plain yogurt • Fortified orange juice • Part-skim mozzarella • Sardines, oil-canned • Milk • Fortified soy milk • Whole milk	**Inadequate intake** **Disease** • **Metabolic alkalosis** can cause decreased serum ionized calcium due to calcium-binding albumin more readily during alkalotic states. • **Chronic Diseases:** hypoparathyroidism, chronic kidney disease, liver disease, and vitamin D deficiency • **Acute illnesses:** sepsis, pancreatitis (due to fat saponification), and acute kidney injury can also result in hypocalcemia. • **Severe hypomagnesemia** can secondarily cause hypocalcemia, as is sometimes seen in proton pump inhibitor (PPI) therapy. PPI therapy also decreases stomach acidity, decreasing calcium absorption via the intestinal tract. **Other medications** • Long-term use of corticosteroids, antiepileptics, aminoglycosides, cisplatin, and bisphosphonates. **Transfusions** • Acute hypocalcemia is common among patients receiving large transfusions, such as during treatment of traumatic hemorrhage, due to citrate and chelation products. • As calcium is vital for proper coagulation, trauma patients receiving blood transfusions should be monitored closely to avoid severe hypocalcemic events.[22]	Hypercalcemia has not been associated with the overconsumption of calcium occurring naturally in food. **Malignancy and primary hyperparathyroidism** are the most common causes of elevated calcium concentrations in the blood (hypercalcemia) **Vitamin D toxicity** can alsoinduce hypercalcemia, which could result in bone loss, kidney stones, and calcification of organs like the heart and kidneys if untreated over a long period. **Over-the-counter calcium supplements in combination with antacids:** Those at risk of hypercalcemia from these supplements are postmenopausal women, pregnant women, transplant recipients, patients with bulimia, and patients on dialysis.[8]

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess/ Toxicities
Chromium ***Minerals*** ***Serum*** Chromium is an essential nutrient used in trace amounts in humans that acts as a cofactor for chromodulin. This peptide enhances the effect of insulin on target tissues, which aids in regulating blood sugar and lipid metabolism.	Adult males: 35 mcg Adult females: 25 mcg Pregnant: 30 mcg Lactating: 45 mcg	• Grape juice • Ham • English muffin • Brewer's yeast • Orange juice • Beef	Deficiency is very rare but can occur in patients receiving IV parenteral nutrition without supplemental chromium added and individuals who regularly participate in endurance exercise.	Supplemental chromium is generally not needed as dietary consumption easily meets physiological needs. **Excessive supplementation:** Many studies have demonstrated the safety of daily doses of up to 1,000 mcg of chromium for several months). Most of the concerns regarding the long-term safety of trivalent chromium supplementation arise from several studies in cell culture, suggesting trivalent chromium, especially in the form of chromium picolinate, may increase DNA damage. A study of 10 women taking 400 mcg/day of chromium as chromium picolinate found no evidence of increased oxidative damage to DNA as measured by antibodies to an oxidized DNA base. However, there have been a few isolated reports of serious adverse reactions to chromium picolinate. Kidney failure was reported five months after a six-week course of 600 mcg/day of chromium in the form of chromium picolinate (83). In comparison, kidney failure and impaired liver function were reported after the use of 1,200 to 2,400 mcg/day of chromium in the form of chromium picolinate over four to five months.[8]
Copper ***Minerals*** ***Serum WBC*** Copper assists with the transport of iron, supports energy production within cells, supports methylation and gene transcription that affects cellular detoxification mechanisms and neurotransmitter generation, supports the myelin sheath around nerves, and aids in connective tissue development. Copper is important for redox reactions and is a potent antioxidant for this reason. Copper also supports melanin production in hair, skin, and nail cells.	Adults: 890 mcg Pregnant: 1,000 mcg Lactating: 1,300 mcg	• Beef liver • Oysters • Baking chocolate • Potatoes • Shiitake mushrooms • Cashew nuts • Dungeness crab • Sunflower seed kernels • Turkey giblets • Chocolate, dark 70-85% • Tofu	Deficiencies or excesses of copper are rare in healthy people. However, copper deficiency can occur in the following populations: • Infants or children fed only cow's milk formula. • Premature infants and infants or children with recurring diarrhea. • Individuals with malabsorption syndromes such as celiac disease, bowel resections, Crohn's disease, and ulcerative colitis. • Individuals with cystic fibrosis. • Individuals with high supplemental zinc intake for prolonged periods. Consider testing for the presence of celiac disease or neurological indications of demyelination to assess if copper deficiency may be associated. **Nutrient interaction:** Copper may become deficient or depleted in the presence of supplemental zinc intake at or above 60 mg/day for prolonged periods.	Most serum copper is found in ceruloplasmin; elevated levels may indicate increased inflammation and oxidative stress rather than excess copper in the blood. **Excessive supplementation:** Copper supplementation of 2 mg/day is usually sufficient to correct deficiencies of copper. The UL for copper is 10 mg/day, which has been shown not to produce liver damage in healthy individuals. Some research suggests elevated blood levels of free unbound copper, which depletes zinc levels, may have an association with the onset of Alzheimer's disease, and supplementation of copper in this population is not recommended if zinc deficiency is suspected. Elevated copper levels have also been associated with Wilson's disease, a rare, autosomal recessive disorder caused by abnormal copper accumulation in the body, particularly involving the brain, liver, and cornea.
Copper/Zinc Ratio ***Minerals*** ***Serum WBC***	N/A	N/A	A low ratio indicates higher levels of zinc relative to copper. A low copper-to-zinc ratio may be associated with a copper deficiency or zinc toxicity.	A high ratio indicates higher levels of copper relative to zinc. Systemic inflammation can increase the Cu/Zn ratio.[23]

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess/ Toxicities
Iron ***Minerals*** ***Serum RBC*** Iron is required to produce red blood cells (hematopoiesis) and is part of hemoglobin (the pigment that gives red blood cells their color). Hemoglobin binds to oxygen and facilitates transport from the lungs via the arteries to all cells throughout the body. Once the oxygen is delivered, the iron (as part of hemoglobin) binds the carbon dioxide, which is then transported back to the lung, where it gets exhaled. Iron is also involved in the conversion of blood sugar to energy. The production of enzymes (which play a vital role in producing new cells, amino acids, hormones, and neurotransmitters) also depends on iron. This aspect becomes crucial during recovery from illnesses or strenuous exercise. The immune system is dependent on iron for its efficient functioning. Physical and mental growth require sufficient iron levels, particularly important in childhood and pregnancy, where the developing baby solely depends on its mother's iron supply.	Adult males: 8 mg Adult females 18 mg Pregnant: 27 mg Lactating: 9 mg	• Fortified breakfast cereals • Oysters • White beans • Beef liver • Lentils • Spinach • Tofu	**Inadequate intake:** Since the body can't produce iron, it's essential to consume sufficient iron through dietary intake. **Body Loss:** Iron is lost by the body in various ways, including urination, defecation, sweating, and exfoliation of old skin cells. **Bleeding** contributes to further loss of iron, which is why women have a higher demand for iron than men. If iron stores are low, normal hemoglobin production slows down, which means the transport of oxygen is diminished, resulting in symptoms such as fatigue, dizziness, lowered immunity, or reduced ability for athletes to keep up with their training programs.	Iron levels are typically evaluated in conjunction with other iron tests or a full anemia panel. High levels of serum iron can occur as a result of: • Multiple blood transfusions. • Excessive iron supplementation or injection. ◦ High iron levels from dietary or supplementation are more likely in men and post-menopausal women because they do not lose iron through menstruation. • Lead poisoning. • Liver or kidney disease. • Genetic disease, e.g., hemochromatosis, when too much iron accumulates in the body and can damage organs.

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess/ Toxicities
Magnesium ***Minerals*** ***RBC*** Magnesium assists enzymes in more than 300 chemical reactions in the body, supporting cellular activity, participating in muscle contraction, aiding in blood clotting, and as a critical component of bone/skeletal tissue.	Adult males: 400 mg Adult females 310 mg **Pregnancy:** Age 19-30: 350 mg Age 31-50: 360 mg **Lactating:** Age 19-30: 310 mg Age 31-50: 320 mg	• Pumpkin seeds • Chia seeds • Almonds • Spinach • Cashews	**Inadequate intake:** Hypomagnesemia can be secondary to decreased intake, as seen in the following: • Starvation • Alcohol use disorder (with a reported prevalence of 30%) • Anorexia nervosa • Terminal cancer • Critically ill patients who are receiving total parenteral nutrition **Medications:** • Loop and thiazide diuretics • Proton pump inhibitors • Aminoglycoside antibiotics • Amphotericin B • Pentamidine • Digitalis • Chemotherapeutic drugs, such as cisplatin, cyclosporine • Antibodies that bind to epidermal growth factor (EGF) receptors (cetuximab, matuzumab, panitumumab) • Laxative abuse **Redistribution from the extracellular to the intracellular compartment:** • Treatment of diabetic ketoacidosis by insulin • Refeeding syndrome • Correction of metabolic acidosis • Acute pancreatitis • Ethanol withdrawal syndrome **Gastrointestinal and renal losses:** • Acute diarrhea • Chronic diarrhea (Crohn's disease, ulcerative colitis) • Hungry bone syndrome (an increased magnesium uptake by renewing bone following parathyroidectomy or thyroidectomy, causing a decrease in serum magnesium) • Acute pancreatitis • Gastric bypass surgery • Genetic disorders • Gitelman syndrome • Bartter syndrome • Familial hypomagnesemia with hypercalciuria and nephrocalcinosis • Renal malformations and early-onset diabetes mellitus caused by HNF1-beta mutation • Autosomal recessive isolated hypomagnesemia caused by EGF mutation • Autosomal dominant isolated hypomagnesemia caused by Na-K-ATPase gamma subunit, Kv1.1, and cyclin M2 mutations. • Intestinal hypomagnesemia with secondary hypocalcemia **Acquired tubular dysfunction:** • Post-kidney transplant • Recovery from acute tubular necrosis • Post obstructive diuresis[24]	Adverse effects have not been identified from magnesium occurring naturally in food. However, adverse effects from excessive magnesium have been observed with intakes of various magnesium salts (i.e., supplemental magnesium). The initial symptom of excess magnesium supplementation is diarrhea.[8]

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess/ Toxicities
Manganese ***Minerals*** ***Serum WBC*** Manganese is important in many enzyme-mediated chemical reactions, including enzymes involved in antioxidant actions in mitochondria and enzymes in synthesizing cartilage in skin and bone. Manganese also activates enzymes that participate in carbohydrates, amino acids, and cholesterol metabolism. In addition, enzymes that incorporate manganese convert the neuroexcitatory glutamate into glutamine.	Adult males: 2.3 mg Adult females 1.8 mg Pregnant: 2.0 mg Lactating: 2.6 mg Upper Limit: 11 mg	• Blue mussels • Hazelnuts • Pecans • Brown rice • Oysters • Calms • Chickpeas • Spinach • Pineapple • Soybean • Whole wheat bread • Oatmeal	Manganese deficiency is quite rare, and there is more concern for toxicity related to manganese overexposure. **Inadequate intake** **Medication:** Magnesium-containing antacids, laxatives, and the antibiotic medication, tetracycline, may decrease manganese absorption if taken together with manganese-containing foods or supplements.[8]	Chronic total parenteral nutrition (TPN) use in critically ill patients: The metal is readily absorbed through the intestinal tract, and absorption is variable based on the dietary intake level, with biliary and pancreatic metabolism affecting excretion. However, these mechanisms are bypassed with IV administration, owing to the potentiality for manganism with IV preparations containing the essential metal. **Environmental exposure** • Consumption of contaminated well water. • Inhalation in welding, smelting, and mining industries. • Methylcyclopentadienyl manganese tricarbonyl (MMT) is a manganese-containing compound used in gasoline as an anti-knock additive. **Inherited manganese overload disorders:** Autosomal recessive mutations in the SLC30A10 gene, which encodes a manganese transporter expressed in the liver and brain, causes a manganese overload syndrome.[8 25]
Potassium ***Minerals*** ***Serum WBC*** Potassium is one of the main bodily electrolytes (a substance that carries an electrical charge). Potassium helps regulate blood pressure and heart contractions and is needed for muscle contractions. It also helps control intracellular and extracellular fluid balance in an appropriate ratio with sodium.	Adult males: 3,400 mg Adult females: 2,600 Pregnant: 2,900 mg Lactating: 2,800 mg	• Apricots, dried • Lentils • Squash, acorn • Prunes • Raisins • Potato • Kidney beans • Orange juice	Blood electrolyte levels are not typically regarded as markers of nutritional status. Deviations from the normal range generally are not caused by dietary factors. Decreased potassium intake, in isolation, rarely results in hypokalemia due to the ability of the kidneys to effectively minimize potassium excretion. However, reduced intake can contribute to hypokalemia in the presence of other causes, such as malnutrition or diuretic therapy. **Increased potassium loss (skin, gastrointestinal, and renal losses):** In general, body fluid losses should be considered before dietary intake if potassium levels are low because most cases of hypokalemia result from gastrointestinal (GI) or renal losses. • Magnesium deficiency can contribute to potassium loss, and potassium repletion is more difficult with inadequate magnesium levels. • Diabetes can cause excess potassium loss through urine. • Excessive alcohol use, excessive laxative use, excessive sweating, diabetic ketoacidosis, folic acid deficiency, and some antibiotic use can cause potassium deficiency. • Potassium-wasting diuretics are a drug category known to cause potassium depletion. **Transcellular shifts (increased intracellular uptake):** Cellular uptake of potassium is promoted by alkalemia, insulin, beta-adrenergic stimulation, aldosterone, and xanthines, such as caffeine.[26]	Abnormally elevated serum potassium concentrations are referred to as hyperkalemia. Hyperkalemia occurs when potassium intake exceeds the capacity of the kidneys to eliminate it. **Excess supplementation** • Oral doses of potassium >18 g taken at one time in individuals not accustomed to high intakes may lead to severe hyperkalemia, even in those with normal kidney function. • Potassium supplements should not be used by anyone with diabetes, insulin resistance, impaired kidney function, or anyone using ACE inhibitors or NSAIDs. • There is a risk for refeeding syndrome if severe potassium depletion is corrected rapidly with supplementation. **Medication:** Potassium-sparing diuretics may increase potassium levels. **Medical Conditions** • Acute or chronic kidney failure. • Insufficient aldosterone secretion (hypoaldosteronism). • Hyperkalemia may also result from a shift of intracellular potassium into the circulation, which may occur with the rupture of red blood cells (hemolysis) or tissue damage (e.g., trauma or severe burns.[8]

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess/ Toxicities
Sodium ***Minerals*** ***Serum RBC*** Sodium is one of the main bodily electrolytes (a substance that carries an electrical charge). Sodium helps maintain extracellular and intracellular fluid balance, is needed for muscle contractions, and helps with nerve signaling.	Limiting sodium intake to less than 2,300 mg per day ~ 1 teaspoon of table salt is recommended.	• Deli meat sandwiches • Pizza • Burritos • Tacos • Soups • Savory snacks (chips, crackers, popcorn) • Poultry • Pasta mixed dishes • Burgers	**Inadequate intake:** Changes in the diet that remove or reduce processed foods will dramatically reduce sodium intake and sometimes cause acute depletion of sodium levels and/or loss of stored water from liver and muscle glycogen stores. **Medical Conditions** • Sodium is excreted through the body by any significant loss of body fluid, such as diarrhea, vomiting, or excessive sweating. • Addison's disease (lack of production of mineralocorticoids from the adrenal glands) may also result in excessive loss of electrolytes, including sodium. **Medications:** diuretics, non-steroidal anti-inflammatory drugs (NSAIDs), opiate derivatives, phenothiazines, serotonin-reuptake inhibitors (SSRIs), and tricyclic antidepressants increase the risk of hyponatremia.	Hypernatremia generally develops from excess water loss (e.g., burns, respiratory infections, renal loss, osmotic diarrhea, hypothalamic disorders) or reduced water intake, frequently accompanied by an impaired thirst mechanism.
Zinc ***Minerals*** ***Serum*** Zinc is critical for normal growth and sexual maturation. It plays a role in the immune system and is important to the proper function of at least 300 enzymes. Zinc plays a critical role in the structure of proteins and cell membranes. Zinc also regulates gene function and influences cell signaling, hormone release, and nerve signaling.	Adult males: 11 mg Adult females: 8 mg Pregnant: 11 mg Lactating: 12 mg	• Oysters • Beef • Blue crab • Fortified breakfast cereal • Pumpkin seeds	**Inadequate intake:** especially in diets that lack meat intake, have excess phytates (present in legumes, seeds, soy products, and whole grains), or oxalates (found in spinach, okra, nuts, and tea) **Chronic illnesses:** the presence of chronic illnesses (chronic gastrointestinal diseases, diabetes, liver disease, sickle cell disease, kidney disease, excess alcohol consumption, HIV infection) or chronic infections.[27] **Nutrient interaction:** Supplementation of elemental iron may decrease the absorption of zinc. For this reason, pregnant women and individuals with anemia who supplement iron may need to take supplemental zinc, separate from iron supplementation. While higher doses of supplementary zinc uptake impair the uptake of copper, intake of copper does not impair the uptake of zinc except when zinc status is already at least marginally deficient.	**Contamination:** • Isolated outbreaks of acute zinc toxicity have occurred due to the consumption of **food or beverages contaminated with zinc** released from galvanized containers. • Metal fume fever has been reported after the inhalation of zinc oxide fumes. **Excess supplementation:** Milder gastrointestinal distress has been reported at doses of 50 to 150 mg/day of supplemental zinc. **Zinc and Copper:** The major consequence of long-term excessive zinc consumption is copper deficiency. Total zinc intakes of 60 mg/day (50 mg supplemental and 10 mg dietary zinc) for up to 10 weeks have been found to result in signs of copper deficiency. To prevent copper deficiency, the US Food and Nutrition Board set the tolerable upper intake level (UL) for adults at 40 mg/day, including dietary and supplemental zinc.[8]
Arginine ***Amino Acids*** ***Serum*** Arginine is important during periods of illness and chronic conditions like hypertension and type II diabetes, as these states tend to be characterized by an increase in arginase (the enzyme that degrades L-arginine), resulting in a transient deficiency; this precedes an increase in blood pressure in these states and can be partially remedied by an increase in L-arginine intake or resolution of the illness/disease state.	N/A	• Red meat • Poultry • Fish • Dairy • Soy • Quinoa • Buckwheat • Nuts • Seeds • Beans	Nutrient interaction: Low arginine may indicate too much lysine or histidine supplementation competing for absorption or consumption of too many lysine-containing foods, including meat and dairy products.[28] Low arginine is also associated with elevated ammonia.[29] Increased utilization: Arginine is important during periods of illness and chronic conditions like hypertension and type II diabetes, as these states tend to be characterized by an increase in arginase (the enzyme that degrades L-arginine), resulting in a transient deficiency; this precedes an increase in blood pressure in these states, and can be partially remedied by an increase in L-arginine intake or resolution of the illness/disease state.	**Excessive supplementation:** • To maintain elevated arginine levels throughout the day, arginine can be taken up to three times a day, with a combined dose total of 15 to 18 g. • Note: L-citrulline supplementation is more effective at maintaining elevated arginine levels for long periods. • Taking more than 10 g of arginine at once can result in gastrointestinal distress and diarrhea. • Individuals who have had cold sores or genital herpes can potentially trigger the virus that causes those conditions with excess L-arginine. • Additionally, L-arginine isn't recommended for people who have had a recent heart attack due to concerns that the supplement might increase the risk of death.[30]

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess / Toxicities
Asparagine ***Amino Acids*** ***Serum WBC*** Asparagine is a non-essential amino acid (can be synthesized in the body) and is required for the development and function of the brain. Asparagine can be synthesized from glutamine and aspartate. Asparagine is also required for DNA and RNA synthesis and removal of the cellular waste product ammonia.	N/A	• Red meat • Poultry • Fish • Dairy • Soy • Quinoa • Buckwheat • Nuts • Seeds • Beans	Asparagine deficiency is unlikely due to its endogenous synthesis and ubiquitous presence in plant and animal foods.	There is currently no established RDA, AI, or UL for asparagine. Asparagine is rarely supplemented directly due to its endogenous production in the body, but it could be indirectly supplemented through glutamine.
Citrulline ***Amino Acids*** ***Serum*** Citrulline can be produced by arginine by one of two mechanisms: either directly via arginine giving off a nitric oxide molecule (which is the path involved in the nitric oxide cycle), or indirectly via arginine's conversion into ornithine (which is involved in the urea cycle), sequestering ammonia.	N/A	• Red meat • Poultry • Fish • Dairy • Soy • Quinoa • Buckwheat • Nuts • Seeds • Beans	**Low arginine intake:** The majority of L-citrulline either floats in the blood passively or gets transported to the kidneys for convection into arginine Citrulline can be produced by arginine by one of two mechanisms: either directly via arginine giving off a nitric oxide molecule (which is the path involved in the nitric oxide cycle), or indirectly via arginine's conversion into ornithine (which is involved in the urea cycle), sequestering ammonia.	**Genetics:** • Citrullinemia type I (CTLN1) is a rare autosomal recessive genetic disorder. CTLN1 is caused by a deficiency of the enzyme argininosuccinate synthase, which converts citrulline and aspartate into argininosuccinate. Complete defects in this enzyme result in extremely elevated levels of citrulline.[31] • Citrullinemia type II (CTLN2) occurs in adults as recurrent hyperammonemia with neuropsychiatric symptoms due to impairment of the argininosuccinate synthase 1 step of the urea cycle.[32] Low protein diets are intended to minimize production of ammonia. Arginine helps to remove ammonia from the blood.
Glutamine ***Amino Acids*** ***Serum WBC*** Glutamine is a conditionally essential amino acid (conditional mainly during times of disease or muscle wasting, such HIV/AIDS, cancer, or severe infections). In the intestinal lining, glutamine is the preferred source of fuel for intestinal epithelial cells and the main energy source for leukocytes (immune cells). Other important functions of glutamine include: transporting nitrogen between cells, acting as a precursor to glutathione production, acting as a precursor to nucleotides (for DNA and RNA synthesis), participating in gluconeogenesis in the absence of adequate carbohydrate intake, blunting the rise of blood glucose after consuming carbohydrate-rich meals, and regulating intestinal tight junctions.	N/A	• Red meat • Poultry • Fish • Dairy • Soy • Quinoa • Buckwheat • Nuts • Seeds • Beans	Glutamine depletion or deficiency is rare, as glutamine can be made endogenously and is ubiquitous in the food supply from both plant and animal sources. **Health conditions:** • Some studies suggest an increase in intestinal permeability when intestinal epithelial cells lack sufficient glutamine, as well as insufficient availability for leukocyte function. • Glutamine is known to be depleted in certain types of physiological stress such as burns, major trauma, and cancers that consume available intracellular glutamine stores more rapidly than skeletal muscle can generate it, leading to increased muscle wasting. **Exercise:** During physical activity, serum glutamine is consumed for longer endurance events (2+ hours); some evidence exists that chronic endurance exercise reduces glutamine levels to affect immune cell function and proliferation.	There is currently no established RDA, AI, or UL for glutamine. Glutamine is typically sold as L-glutamine, and doses have been studied in humans ranging from 500 mg/day to 50 g/day. Higher doses (>10 g/day) are commonly used in the treatment of intestinal barrier permeability. • In some individuals, glutamine is converted more efficiently to glutamate, which can lead to a neuron-excitatory state, increased anxiety, tension headaches/migraines, and even tachycardia. If any of these symptoms occur after consuming glutamine, supplementation should be discontinued.

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess / Toxicities
Isoleucine ***Amino Acids*** ***Serum*** Essential amino acid (needs to be obtained via diet) and 1 of 3 branched chain amino acids (alongside leucine and valine). Isoleucine is necessary for hemoglobin formation and regulating blood sugar and energy levels. Isoleucine is concentrated in muscle tissues in humans.	19 mg per 2.2 pounds of body weight.[33]	• Red meat • Poultry • Fish • Dairy • Soy • Quinoa • Buckwheat • Nuts • Seeds • Beans	**Inadequate Intake:** A diet that lacks sufficient protein or is deficient in sources of BCAAs (e.g., meat, dairy, eggs, legumes, and certain grains) can lead to deficiencies in isoleucine, leucine, and valine. **Vegetarian and Vegan Diets:** Individuals following strict vegetarian or vegan diets may have a higher risk of BCAA deficiency as plant-based diets generally provide lower amounts of BCAAs. **Medical Conditions** • **Malnutrition:** General malnutrition or starvation can result in BCAA deficiencies along with other nutrient deficiencies. • **Malabsorption Disorders:** Gastrointestinal conditions like celiac disease or Crohn's disease can hinder the absorption of amino acids, potentially leading to BCAA deficiencies. • Conditions that increase protein requirements, such as growth, pregnancy, or certain medical conditions, can lead to an increased demand for BCAAs. In such cases, a diet that does not meet these increased requirements can result in deficiency. **Medications** such as valproic acid (used for epilepsy and mood disorders), can interfere with BCAA metabolism and lead to deficiency.	**High Protein Diet** especially from animal sources. **Excess Supplementation of Branched Chain Amino Acids** often used by athletes and bodybuilders. **Liver dysfunction:** non-alcoholic fatty liver diseases (NAFLD), cirrhosis, and hepatocellular carcinoma (HCC) have been associated with elevated BCAA.[34] **Genetics** • **Maple Syrup Urine Disease (MSUD):** MSUD is a rare inherited disorder that affects the body's ability to metabolize BCAAs. It leads to the accumulation of isoleucine, leucine, and valine in the blood and tissues, potentially causing neurological problems and other health issues.
Leucine ***Amino Acids*** ***Serum*** Leucine is one of nine essential amino acids (it must be obtained via diet) and one of three branched-chain amino acids (alongside isoleucine and valine). Leucine is important for protein synthesis and many metabolic functions. It contributes to regulating blood sugar levels, growing and repairing muscle and bone tissue, producing growth hormones, and healing wounds. Leucine also prevents the breakdown of muscle proteins after trauma or severe stress and may be beneficial for individuals with phenylketonuria.	42 mg per 2.2 pounds is recommended.[33]	• Red meat • Poultry • Fish • Dairy • Soy • Quinoa • Buckwheat • Nuts • Seeds	Leucine is available in many foods, and deficiency is rare. **Inadequate Dietary Intake:** A diet that lacks sufficient protein or is deficient in sources of BCAAs (e.g., meat, dairy, eggs, legumes, and certain grains) can lead to isoleucine, leucine, and valine deficiencies. **Vegetarian and Vegan Diets:** Individuals following strict vegetarian or vegan diets may have a higher risk of BCAA deficiency as plant-based diets generally provide lower amounts of BCAAs. **Medical Conditions** • **Malnutrition:** General malnutrition or starvation can result in BCAA and other nutrient deficiencies. • **Malabsorption Disorders:** Gastrointestinal conditions like celiac disease or Crohn's disease can hinder the absorption of amino acids, potentially leading to BCAA deficiencies. • Conditions that increase protein requirements, such as growth, pregnancy, or certain medical conditions, can increase demand for BCAAs. In such cases, a diet that does not meet these increased requirements can result in deficiency. **Medications** such as valproic acid (used for epilepsy and mood disorders) can interfere with BCAA metabolism and lead to deficiency.	**High Protein Diet,** especially from animal sources. **Excess Supplementation of Branched Chain Amino Acids,** often used by athletes and bodybuilders. **Liver dysfunction:** non-alcoholic fatty liver diseases (NAFLD), cirrhosis, and hepatocellular carcinoma (HCC) have been associated with elevated BCAA.[34] **Genetics:** Maple Syrup Urine Disease (MSUD): MSUD is a rare inherited disorder that affects the body's ability to metabolize BCAAs. It leads to the accumulation of isoleucine, leucine, and valine in the blood and tissues, potentially causing neurological problems and other health issues.

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess / Toxicities
Serine ***Amino Acids*** ***Serum WBC*** Serine is a non-essential amino acid (it can be synthesized in the body to some extent). It can be synthesized endogenously from dietary glycine, which is not considered an essential amino acid. D-serine is a neuromodulator produced in the glial cells of the brain and modulates the functions of neurons. Serine can be considered a nootropic nutrient. Serine enhances the binding of other compounds at NMDA (N-methyl-D-aspartate) receptors.	N/A	• Red meat • Poultry • Fish • Dairy • Soy • Quinoa • Buckwheat • Nuts • Seeds • Beans	Serine deficiency would be rare. However, supraphysiological doses may be necessary to confer benefit over standard dietary intake. **Inadequate Dietary Intake:** A diet lacking sufficient protein or primarily plant-based, such as vegetarian and vegan diets, may increase the risk of serine deficiency. **Medical Conditions** • **Malnutrition:** General malnutrition or starvation can result in BCAA and other nutrient deficiencies. • **Malabsorption Disorders:** Gastrointestinal conditions like celiac disease or Crohn's disease can hinder the absorption of amino acids, potentially leading to BCAA deficiencies. • Conditions that increase protein requirements, such as growth, pregnancy, or certain medical conditions, can increase demand for BCAAs. In such cases, a diet that does not meet these increased requirements can result in deficiency.	There is currently no established RDA, AI, or UL for serine supplementation or intake. **Excessive supplementation:** • Serine can be supplemented to reduce symptoms of cognitive decline and reduce symptoms of cocaine dependence and schizophrenia. • Phosphatidylserine is a common supplemental phospholipid that contains serine. • Doses of 30 mg/kg of body weight are commonly used in cognitive decline patients.
Valine ***Amino Acids*** ***Serum*** Essential amino acid (must be obtained via diet). Valine is a branched-chain essential amino acid that has stimulant activity. It promotes muscle growth and tissue repair. It is a precursor in the penicillin biosynthetic pathway. Valine maintains mental vigor, muscle coordination, and emotional calm as a glycogenic amino acid.	24 mg per 2.2 pounds.[33]	• Red meat • Poultry • Fish • Dairy • Soy • Quinoa • Buckwheat • Nuts • Seeds • Beans	**Inadequate Dietary Intake:** A diet that lacks sufficient protein or is deficient in sources of BCAAs (e.g., meat, dairy, eggs, legumes, and certain grains) can lead to isoleucine, leucine, and valine deficiencies. **Vegetarian and Vegan Diets:** Individuals following strict vegetarian or vegan diets may have a higher risk of BCAA deficiency as plant-based diets generally provide lower amounts of BCAAs. **Medical Conditions** • **Malnutrition:** General malnutrition or starvation can result in BCAA and other nutrient deficiencies. • **Malabsorption Disorders:** Gastrointestinal conditions like celiac disease or Crohn's disease can hinder the absorption of amino acids, potentially leading to BCAA deficiencies • Conditions that increase protein requirements, such as growth, pregnancy, or certain medical conditions, can increase demand for BCAAs. In such cases, a diet that does not meet these increased requirements can result in a deficiency. **Medications** such as valproic acid (used for epilepsy and mood disorders) can interfere with BCAA metabolism and lead to deficiency.	High protein diet, especially from animal sources. Excess Supplementation of branched chain amino acids, often used by athletes and bodybuilders. **Liver dysfunction:** non-alcoholic fatty liver diseases (NAFLD), cirrhosis, and hepatocellular carcinoma (HCC) have been associated with elevated BCAA.[34] **Genetics** **Maple Syrup Urine Disease (MSUD):** MSUD is a rare inherited disorder that affects the body's ability to metabolize BCAAs. It leads to the accumulation of isoleucine, leucine, and valine in the blood and tissues, potentially causing neurological problems and other health issues.

Nutrient	RDA *Adults 19+*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess / Toxicities
Carnitine ***Metabolites*** ***Serum WBC*** Carnitine is an essential cofactor in the metabolism of lipids and cellular energy production. It promotes neuroprotection through antioxidant properties and modulates and promotes synaptic neurotransmission. Carnitine can be rate-limiting in ketone body uptake by brain astrocytes and also reduces oxidative stress.	Healthy children and adults do not need to consume carnitine from food or supplements because the liver and kidneys synthesize sufficient amounts to meet daily needs.[1]	• Beef steak • Ground beef • Milk, whole • Codfish • Chicken breast	Nutritional carnitine deficiencies have not been identified in healthy people without metabolic disorders, suggesting that most people can synthesize enough L-carnitine.[8] **Inadequate Dietary Intake:** • Dietary intake is the primary source of carnitine and accounts for almost three-fourths of the total body stores. The remaining one-fourth of the carnitine pool can be produced endogenously from lysine and methionine, mainly by the liver and kidneys.[35] • Carnitine is primarily obtained from the diet, particularly animal products like meat, fish, and dairy. A diet that lacks these sources can lead to carnitine deficiency, although this is relatively rare. • Individuals following strict vegan or vegetarian diets may have a higher risk of carnitine deficiency since plant-based diets typically provide lower amounts of carnitine. • Iron and vitamin C are required for endogenous carnitine synthesis, and severe liver disorders can also impair endogenous synthesis. • There may be an increased demand for carnitine during ketosis. **Medical Conditions** • Chronic kidney disease and hemodialysis.[36] • Malabsorption disorders like celiac disease, Crohn's disease, and other gastrointestinal disorders can impair the absorption of nutrients, including carnitine, leading to deficiencies. Medications like antibiotics may interfere with carnitine metabolism or excretion, potentially leading to deficiency. Long-term treatment with pivalic acid antibiotics may decrease serum carnitine concentration.[37] **Genetics** • Primary carnitine deficiency is a genetic disorder of the cellular carnitine transporter system that causes a shortage of carnitine within cells. • Secondary carnitine deficiency results from certain disorders (such as chronic renal failure) that reduce endogenous carnitine synthesis or increase its excretion or from chronic use of pivalate-containing medications that reduce carnitine absorption or increase its excretion.[1]	Carnitine does not have an established tolerable upper intake level. However, doses of approximately three g/day of carnitine supplements can cause nausea, vomiting, abdominal cramps, diarrhea, and a fishy body odor.[1] L-carnitine generally appears well tolerated; no toxic effects have been reported concerning intakes of high doses of L-carnitine. L-carnitine supplementation may cause mild gastrointestinal symptoms, including nausea, vomiting, abdominal cramps, and diarrhea. Supplements providing more than three g/day may cause a "fishy" body odor.[8]
Choline ***Metabolites*** ***Serum WBC*** Choline is an essential nutrient required for many metabolic functions. The body requires choline to synthesize phosphatidylcholine and sphingomyelin, two vital cell membrane phospholipids. Additionally, choline is needed for the production of acetylcholine, an important neurotransmitter for memory, mood, muscle control, and other nervous system functions. It also plays an important role in modulating gene expression, cell membrane signaling, lipid transport, and metabolism. Meat, poultry, fish, dairy products, eggs, whole grains, and cruciferous vegetables are rich in choline. A deficiency of choline can cause muscle damage, liver damage, and nonalcoholic fatty liver disease (NAFLD or hepatosteatosis).	Adult males: 550 mg Adult females: 425 mg Pregnant: 450 mg Lactating: 550 mg[1]	• Beef liver • Egg • Soybeans • Chicken breast • Codfish • Potatoes, red • Wheat Germ • Beans, kidney • Quinoa • Milk, 1% fat	Choline depletion is typically not a concern, and limited information exists on how it would occur. However, low intake of choline may lead to inefficient methylation. Deficiency in dietary choline is known to increase hepatic triglyceride accumulation. This results in lower blood triglycerides but increases the accumulation of triglycerides in the liver. **Inadequate intake:** Individuals following strict vegan or vegetarian diets may be at a higher risk of choline deficiency, as plant-based diets typically provide lower amounts of choline. **Excessive alcohol consumption** can interfere with choline metabolism and increase the risk of choline deficiency. **Medical Conditions** • **Malabsorption disorders:** Gastrointestinal conditions like celiac disease, Crohn's disease, and other disorders that affect nutrient absorption can lead to choline deficiency. • **Increased Choline Requirements:** Specific medical conditions, growth, pregnancy, and breastfeeding may increase the body's demand for choline. If dietary intake does not meet these increased requirements, it can result in deficiency. **Genetics:** A single nucleotide polymorphism (SNP; rs12325817) of the PEMT gene is thought to increase the susceptibility to choline deficiency-induced organ dysfunction, which may alter the dietary requirement for choline and thus increase the likelihood of developing signs of deficiency when choline intake is inadequate.[8] **Medication:** anticholinergic drugs may interfere with choline utilization or metabolism.	The AI for choline is 425 mg/day for women and 550 mg/day for men. **Excess supplementation:** The tolerable upper intake level (UL) for choline for adults 19 years and older is 3,500 mg daily based on the amount shown to produce side effects. Reaching this high amount would most likely be caused by taking very high dose supplements rather than from diet alone. Very high intakes of choline can lead to low blood pressure (hypotension) and liver toxicity. It may also lead to the excess production of TMAO, which is associated with a higher risk of cardiovascular disease. Other symptoms include excessive sweating, fishy body odor, or nausea/vomiting.[38]

Nutrient	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess / Toxicities
Inositol ***Metabolites*** ***Serum WBC*** Inositol derivatives are used in the cellular signaling process after the insulin receptor is activated. They are crucial for the development of peripheral nerves, help move fats out of the liver, promote the production of lecithin, and are anti-arteriosclerotic and anti-atherogenic. Inositol can be released from phytate compounds via intestinal bacteria-breaking phytate-degrading enzymes (Lactobacillus plantarum, Lactobacillus brevis, Lactobacillus curvatus, L. gasseri B. subtilis, and Saccharomyces cerevisiae). Inositol is also stored in the liver, spinal cord nerves, brain, and cerebral spinal fluid.	• Meat • Fruits • Corn • Beans • Grains • Legumes	**Inadequate intake** **Medical Conditions** • Metabolic syndrome • Type 2 diabetes • Polycystic ovary syndrome (PCOS) • Insulin resistance • Obesity **Medication:** If many courses of antibiotics are used, inositol depletion may occur from microbiome conversion.	There is currently no established RDA, AI, or UL for inositol.
Methylmalonic Acid (MMA) ***Metabolites*** ***Serum*** MMA is a vitamin B12-associated metabolite and the most sensitive marker of vitamin B12 status.			
Coenzyme Q10 (CoQ10) ***Antioxidants*** ***Serum WBC*** CoQ10 is a fat-soluble compound primarily synthesized by the body and consumed in the diet. It is found in virtually all cell membranes and participates in the mitochondria to convert carbohydrates and fatty acids into ATP. CoQ10 also supports cell signaling, gene expression, stimulation of cell growth, inhibition of apoptosis, control of thiol groups, formation of hydrogen peroxide, and control of membrane channels.	Foods are considered poor sources of the CoQ10. Foods that contain more CoQ10 than others include organ meats from red meat sources. Nuts are considered a moderate source but must be eaten in extreme amounts to meet the daily requirement.	**Inadequate intake:** Endogenous supplies are generated in the liver, but they can also be found in organ meat, soy oil, sardines, and peanuts. **Aging:** Aging leads to a natural decrease in levels of CoQ10 due to decreased synthesis and increased degradation, and this deficit cannot be compensated for by diet. **Medication:** CoQ10 is most commonly depleted through cholesterol-lowering medications, such as statins. **Genetics:** • Primary deficiency is associated with defects in genes directly involved in the biosynthesis of CoQ10. To date, CoQ10 and idebenone supplementation is the only treatment for deficiencies, with early detection leading to a better prognosis. • Causes of secondary deficiency include mutations of genes not directly involved in the biosynthesis of CoQ10 (APTX, ETFDH, BRAF, ANO10), impaired CoQ10 synthesis, insufficient dietary intake, and excessive cellular usage of CoQ10.[39]	There is currently no established RDA, AI, or UL for CoQ10. There have been no reports of significant adverse side effects of oral coenzyme Q10 supplementation at doses as high as 3,000 mg/day for up to eight months, 1,200 mg/day for up to 16 months), and 600 mg/day for up to 30 months. According to the observed safe level (OSL) risk assessment method, evidence of safety is strong with doses up to 1,200 mg/day of coenzyme Q10. Because reliable data on lactating women are unavailable, supplementation should be avoided during breastfeeding.[8]
Cysteine ***Antioxidants*** ***Serum WBC*** Cysteine has antioxidant properties and is a precursor molecule to glutathione production, the master antioxidant. Cysteine is also an essential source of sulfide for iron-sulfide metabolism. Cysteine will easily bind metals, such as iron, nickel, copper, zinc, and heavy metals, such as mercury and lead, to its thiol group, which may confer some chelation benefits. Cysteine counteracts acetaldehyde effects from the consumption of alcohol and can reduce hangovers.	• Meat • Poultry • Eggs • Dairy • Red peppers • Garlic • Onions • Broccoli • Brussel sprouts • Oats • Granola • Wheat germ • Lentils	Cysteine can be synthesized endogenously if sufficient methionine is available in the diet. Depletion is extremely rare. **Inadequate Intake:** Individuals following strict vegan or vegetarian diets may have a higher risk of cysteine deficiency since plant-based diets typically provide lower amounts of cysteine. **Malabsorption Disorders:** Gastrointestinal conditions like celiac disease or Crohn's disease can impair the absorption of nutrients, including cysteine, leading to deficiencies. **Increased Cysteine Requirements:** Children and adolescents, especially during rapid growth and development periods, may have increased cysteine requirements to support tissue development. **Medical Conditions** • **Cystinuria:** Cystinuria is a genetic disorder that affects the transport of cystine, a form of cysteine, in the kidneys. Individuals with cystinuria have a higher risk of forming cystine stones in the urinary tract, and this condition leads to increased cysteine loss in the urine, resulting in lower levels in the body. • **Chronic Illness:** Some chronic medical conditions, particularly those involving oxidative stress, such as chronic inflammatory diseases or chronic kidney disease, may increase the body's demand for cysteine. • **Burn Injuries:** Severe burn injuries can lead to an increased requirement for cysteine to support wound healing and tissue repair.	There is currently no established RDA, AI, or UL for cysteine. **Excess supplementation:** • Cysteine is typically purchased in supplement form as N-acetylcysteine (NAC). • For general antioxidant support, doses start at 500 mg/day and can increase depending upon direction from the medical provider. • **AVOID:** D-cysteine or D-cystine, which are toxic.

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess / Toxicities
Glutathione ***Antioxidants*** ***WBC*** Glutathione (GSH) is the master intracellular antioxidant. The only cells in the body that have been found to absorb intact GSH are hepatocytes, intestinal mucosal cells, and retinal cells.	N/A	Dietary glutathione does not correlate with systemic levels of glutathione. Still, sources of glutathione are fruits and vegetables such as: • Asparagus • Avocado • Spinach • Broccoli • Cantaloupe • Tomato • Carrot • Grapefruit • Orange • Zucchini • Strawberry • Watermelon • Papaya • Red bell peppers • Peaches • Lemons • Mangoes • Cauliflower • Cabbage	Glutathione levels deplete naturally during **aging.** However, this could be related to decreased protein intake in aging individuals. **Pro-inflammatory states and elevated oxidative stress** will drain GSH stores and require a conditionally greater intake of either high cysteine foods or NAC as a supplement to increase endogenous GSH production. Depleted glutathione is converted back to its active state through an NADPH-dependent enzyme, so it stands to reason that **low levels of NAD (or niacin, nicotinic acid)** could further limit this conversion back to the active GSH form. **Genetics:** • Glutathione synthetase deficiency. • Consider mutations in the GSHPx gene to determine if deficiency or depletion is genetically influenced.	There currently is no RDA, AI, or UL established for glutathione intake. **Excess supplementation:** • High-dose supplementation is sometimes used for various purposes, including skin lightening and antioxidant support. • Glutathione cannot enter cells intact and must be synthesized inside the cell to be effective. Therefore, supplementation usually has negligible benefit. • Supplementing the building blocks such as NAC, glutamic acid, and glycine may increase cellular production of glutathione; however, NAC may be most impactful as the amino acid cysteine is known to be rate-limiting for the synthesis of glutathione. • Direct glutathione supplementation has only been shown to benefit slowing the breakdown of nitric oxide in the bloodstream.
Selenium ***Antioxidants*** ***Serum WBC*** Selenium is an essential trace element required for immune function and thyroid hormone synthesis through its actions in selenoproteins such as iodothyronine deiodinase and the direct conversion of thyroxine (T4) to triiodothyronine (T3). Additionally, this mineral assists enzymes in protecting cell membranes from damage, and selenium is a critical component of antioxidant reactions by supporting the production of selenoproteins, such as glutathione peroxidase. Selenium helps regenerate vitamins C and E from their oxidized forms, supporting their antioxidant action.	Adults: 55 mcg Pregnant: 60 mcg Lactating: 70 mcg Protein-based food sources of selenium appear to be the most effective at increasing circulating levels of glutathione peroxidase.	• Brazil nuts • Tellowfin tuna • Halibut • Sardines • Ham • Shrimp • Beef • Turkey • Chicken	**Inadequate intake:** Selenium deficiency occurs when there is inadequate dietary intake of selenium, typically due to a scarcity of selenium sources in a given region. Interestingly, many selenium-deficiency diseases are linked with concurrent vitamin E deficiency.[40] Individuals at risk for low levels of selenium or selenium depletion are patients who have had bariatric surgery, celiac patients, and Crohn's disease patients.	The tolerable upper intake level (UL) for selenium at 400 µg/day in adults is based on preventing hair and nail brittleness and loss and early signs of chronic selenium toxicity (52). The UL of 400 mcg/day for adults includes both selenium obtained from food and supplements.[8] Although selenium is required for health, high doses of selenium can be toxic. Acute and fatal toxicities have occurred with accidental or suicidal ingestion of gram quantities of selenium. Chronic selenium toxicity (selenosis) may occur with smaller doses of selenium over long periods. The most frequently reported symptoms of selenosis are hair and nail brittleness and loss. Other symptoms may include gastrointestinal disturbances, skin rashes, a garlic breath odor, fatigue, irritability, and neurologic disorders.

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess / Toxicities
Eicosapentaenoic acid (EPA), n-3 ***Omega Fatty Acids*** ***RBC*** Eicosapentaenoic acid (EPA) is an omega-3 fatty acid that participates in the health of cellular membranes, mediates lipid actions, and reduces inflammatory responses in the body. EPA and docosahexaenoic acid (DHA) influence the types of inflammatory response mediators made in favor of anti-inflammatory eicosanoids such as leukotrienes, prostaglandins, and thromboxanes. EPA and DHA are also noted for moderate to strong anti-depressant effects. Specific to EPA, it has been shown to suppress the signaling of TNF-α in adipocytes. EPA also increases cerebral oxygenation. EPA appears to have some beneficial influence on regulating leptin levels and increasing adiponectin. EPA may enhance adaptive immunity by stimulating B cell responsiveness.	N/A - The Food and Nutrition Board of the National Academy of Medicine did not establish specific intake recommendations for EPA, DHA, or other long-chain omega-3s. Adequate Intakes (AI) apply only to alpha-linolenic acid (ALA) because ALA is the only omega-3 that is essential. ALA is the precursor to EPA and DHA. **ALA AI:** Adults males: 1.6 g Adult females: 1.1 g Pregnant: 1.4 g Lactating: 1.3 g[1]	• Atlantic salmon • Atlantic herring • Sardines • Atlantic mackerel • Rainbow trout • Eastern oysters ALA is the precursor to EPA and DHA and can be found in high concentrations in chia and flax seeds.	**Inadequate intake** of omega-3 fatty acids is the primary reason for deficiency of EPA, or low levels of EPA. **Malabsorption disorders:** Essential fatty acid deficiency has been found in patients with chronic fat malabsorption and cystic fibrosis.[8] **Genetics:** Certain genetic polymorphisms, such as reduced activity of the FADS1 and FADS2 genes, may lead to reduced conversion of ALA into EPA and DHA.	• Currently, no official dietary intake recommendations have been established. • Several official health organizations have proposed a minimum dietary intake level of 500 mg/day of EPA+DHA. • Because the efficiency of conversion of ALA to EPA is so low, supplementing EPA is generally recommended to meet therapeutic doses. • High-dose supplementation of omega-3 fatty acids (including EPA) has been shown to reduce the need for non-steroidal anti-inflammatory drugs (NSAIDs). • People with ulcerative colitis have been shown to need fewer corticosteroids when supplementing with high-dose omega-3 fatty acids. • Adverse side effects observed with high doses of omega-3 fatty acids from supplement form include gastrointestinal upset and loose stools. • Omega-3 supplements, including EPA and DHA, should be used with caution in persons with clotting disorders or on anti-clotting medication as omega-3s may prolong clotting times.
Docosapentaenoic acid (DPA), n-3 ***Omega Fatty Acids*** ***RBC*** Docosapentaenoic acid (DPA) is an omega-3 fatty acid that is structurally similar EPA. DPA is an intermediary omega-3 fatty acid between the conversion of EPA and DHA. DPA supports the production of healthy blood vessels and reduces clotting.	N/A	• Salmon • Atlantic Mackerel • Pompano • Herring • Rainbow trout • Sablefish • Whitefish • Bluefin tuna • Grass-fed beef • Lamb	**Inadequate intake:** Deficiency of DHA is typically due to low dietary intake of high-DPA foods. **Malabsorption disorders:** Essential fatty acid deficiency has been found to occur in patients with chronic fat malabsorption and in patients with cystic fibrosis.[8]	Adverse side effects observed with high doses of omega-3 fatty acids from supplement form include gastrointestinal upset and loose stools.
Docosahexaenoic acid (DHA), n-3 ***Omega Fatty Acids*** ***RBC*** Docosahexaenoic acid (DHA) is an omega-3 fatty acid that can be synthesized from alpha-linolenic acid (ALA) in the human body. Still, conversion is inefficient and may not meet physiological needs for some. Essential fatty acids, which are converted to omega-3 and omega-6 fatty acids in the body, play a critical role in cell membrane structure and function. Eicosapentaenoic acid (EPA) and DHA influence the types of inflammatory response mediators in favor of less inflammatory eicosanoids such as leukotrienes, prostaglandins, and thromboxanes. EPA and DHA are also noted for moderate to strong anti-depressant effects. Specific to DHA, it makes up an important component of retinal and neuronal cell membranes and, therefore, may play a role in neurological development and function, as well as visual function development. DHA, along with EPA, may help to lower lipids in individuals with Type 2 diabetes mellitus. The third trimester of pregnancy and the first three months of life after birth are the most critical times for the accumulation of DHA in the infant brain.	N/A - The Food and Nutrition Board of the National Academy of Medicine did not establish specific intake recommendations for EPA, DHA, or other long-chain omega-3s. Adequate Intakes apply only to alpha-linolenic acid (ALA) because ALA is the only omega-3 that is essential. ALA is the precursor to EPA and DHA. **ALA AI:** Adults males: 1.6 g Adult females: 1.1 g Pregnant: 1.4 g Lactating: 1.3 g[1]	• Atlantic salmon • Atlantic herring • Sardines • Atlantic mackerel • Rainbow trout • Eastern oysters ALA is the precursor to EPA and DHA and can be found in high concentrations in chia and flax seeds.	**Inadequate intake:** Low dietary intake of omega-3 fatty acids from fatty fish, fish oil, or krill oil is the primary reason for deficiency of DHA. Only about 0-4% of dietary ALA is converted to DHA in men, and up to 9% of dietary ALA is converted to DHA in women due to estrogenic effects on the conversion of fatty acids. **Malabsorption disorders:** Essential fatty acid deficiency has been found to occur in patients with chronic fat malabsorption and in patients with cystic fibrosis.[8] **Genetics** • Specific genetic polymorphisms, such as reduced activity of the FADS1 and FADS2 genes, may lead to reduced conversion of ALA into EPA and DHA. Individuals with APOE4 genetic SNPs may experience lower serum levels of DHA as DHA oxidation is increased in these individuals. • In addition to assessing DHA status relative to risk for Alzheimer's disease and dementia, consider evaluating for the presence of APOE4 genetic SNPs.	• Currently, no official dietary intake recommendations have been established. • High-dose supplementation of omega-3 fatty acids (including DHA) has been shown to reduce the need for non-steroidal anti-inflammatory drugs (NSAIDs). • Adverse side effects observed with high doses of omega-3 fatty acids from supplement form include gastrointestinal upset and loose stools. • Omega-3 supplements, including EPA and DHA, should be used with caution in persons with clotting disorders or on anti-clotting medication as omega-3s may prolong clotting times.

Nutrient	RDA (Recommended Daily Allowance) *Adults = 19+ y/o*	Rich Dietary Sources[1]	Low / Suboptimal / Deficiencies	High / Excess / Toxicities
Linoleic Acid, n-6 ***Omega Fatty Acids*** ***RBC*** Linoleic Acid (LA) is considered the "parent" omega-6 fatty acid. All other omega-6 fatty acids are synthesized from linoleic acid by desaturation and elongation reactions. Thus, LA must be consumed in the diet for all omega-6 fatty acids to be used and incorporated into the body's cells and cell membranes. LA stimulates cell division and repair.	N/A	• Vegetable oils (corn, soybean, sunflower, safflower, canola, peanut) • Avocado • Walnuts • Seeds	**Inadequate intake:** Given the large quantities of vegetable oils in the typical Western diet, depletion or low levels of LA are usually only seen on a fat-free diet, a diet very restricted in dietary fat, or in situations of fat malabsorption. **Malabsorption disorders:** Essential fatty acid deficiency has been found to occur in patients with chronic fat malabsorption and in patients with cystic fibrosis.[8]	Linoleic acid and omega-6 fatty acids are plentiful in the Western diet, so supplementation is typically unnecessary. The best way to lower LA levels is to decrease the intake of refined vegetable oil (corn, soy, peanut, canola, safflower oil) and replace it with an alternative such as olive oil, coconut oil, avocado oil, or fat from animal sources.
Arachidonic Acid, n-6 ***Omega Fatty Acids*** ***RBC*** Arachidonic Acid (AA) is an essential polyunsaturated fatty Acid (PUFA). It is a precursor in the biosynthesis of important molecules like prostaglandins, thromboxanes, and leukotrienes. It is also an integral constituent of the cell membrane, providing it with fluidity and flexibility. Thus, it is important for the function of most cells, especially in the nervous system, skeletal muscles, and immune system. Metabolites obtained from the oxidation of arachidonic acid contribute to inflammation and wound healing processes.	N/A	• Meat (pork, chicken, beef) • Fish (farmed salmon, flatfish, flounder, sardines) • Dairy (cream cheese)	Fish oil supplementation has been shown to lower AA, likely through decreasing activity of the delta-6-desaturase enzyme. **Malabsorption disorders:** Essential fatty acid deficiency has been found to occur in patients with chronic fat malabsorption and in patients with cystic fibrosis.[8]	Arachidonic acid can have two origins: the diet or endogenous synthesis from a precursor, particularly linoleic acid, which is consumed in fairly high amounts in most diets. The rate of conversion is largely dependent on the activity of the delta-6-desaturase. **Excessive Intake:** Dietary sources of preformed arachidonic acid are eggs and meat; fish also contain arachidonic acid.[42]
AA/EPA Ratio ***Omega Fatty Acids*** ***RBC*** Eicosapentaenoic acid (EPA) is a key anti-inflammatory/anti-aggregatory long-chain polyunsaturated omega-3 fatty acid. Conversely, the omega-6 fatty acid arachidonic acid (AA) is a precursor to several pro-inflammatory/pro-aggregatory mediators. EPA acts competitively with AA for the key cyclooxygenase and lipoxygenase enzymes to form less inflammatory products.	N/A	N/A	A low AA:EPA ratio reflects a low AA relative to EPA, which might shift the balance towards an overly anti-inflammatory/anti-aggregatory state, which may not be beneficial as inflammation is a natural and necessary part of the immune response, and platelet aggregation is a natural and necessary part of blood clotting and wound healing. Additionally, as AA is a crucial component of cell membranes, a low AA:EPA ratio may negatively impact cellular membrane integrity, permeability, and function. Furthermore, as AA is important to brain development and cognitive function, a low AA:EPA ratio may negatively impact neurological and cognitive function.	A high AA:EPA ratio corresponds with higher levels of inflammation. Epidemiological studies have shown that a higher AA:EPA ratio is associated with an increased risk of coronary artery disease, acute coronary syndrome, myocardial infarction, stroke, chronic heart failure, peripheral artery disease, and vascular disease.[41] Decreasing the AA:EPA ratio through treatment with purified EPA has been shown in clinical studies to be effective in primary and secondary prevention of coronary artery disease and reduces the risk of cardiovascular events following percutaneous coronary intervention. The AA:EPA ratio is a valuable predictor of cardiovascular risk.[41]
Omega 3-Index ***Omega Fatty Acids*** ***RBC*** The omega-3 index is the amount of EPA plus DHA in red blood cell membranes expressed as the percent of total red blood cell membrane fatty acids. Please note this value is a percentage, with the denominator being the sum of all fatty acids measured in the red blood cells; thus, the index can vary based on the fatty acid composition of the diet.	N/A	N/A	The EPA + DHA content of red blood cell membranes correlates with that of cardiac muscle cells, and several observational studies indicate that a lower omega-3 index is associated with an increased risk of coronary heart disease mortality. It is therefore proposed that the omega-3 index be used as a biomarker for cardiovascular disease risk, with suggested cutoffs as follows: high risk, <4%; intermediate risk, 4%-8%; and low risk, >8%.[8]	Supplementation with EPA + DHA from fish oil capsules for approximately five months dose-dependently increased the omega-3 index in 115 healthy young adults (ages 20-45 years), validating the use of the omega-3 index as a biomarker of EPA + DHA intake.[8]

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