DEMO DEMO

Name: DEMO DEMO
Date of Birth: 00-00-0000
Biological Sex: Female
Age: 50
Height:
Weight:
Fasting:

Telephone: 000-000-0000
Street Address:
Email:

FINAL REPORT

Accession ID: 2322874238

Provider Information

Practice Name: DEMO CLIENT, MD
Provider Name: DEMO CLIENT, MD
Phlebotomist: 0

Telephone: 000-000-0000
Address: 3521 Leonard Ct, Santa Clara, CA 95054

Specimen Information

Sample Type	Collection Time	Received Time	Report	Final Report Date
Serum	Local 2026-06-19 18:30	2026-06-19 14:02 (PDT)	Nutrient Baseline - P2	2026-06-24 10:18 (PDT)
TES	Local 2026-06-19 18:30	2026-06-19 14:02 (PDT)	Nutrient Baseline - P2	2026-06-24 10:18 (PDT)
EDTA	Local 2026-06-19 18:30	2026-06-19 14:02 (PDT)	Nutrient Baseline - P2	2026-06-24 10:18 (PDT)

SAMPLE

Disclaimer: Vibrant provides and makes available this report and any related services pursuant to the Terms of Use Agreement (the "Terms") on its website at www.vibrant-wellness.com. By accessing or otherwise using the report or any services, you acknowledge that you have read, understood, and agree to be bound by these terms. If you do not agree to these terms, you shall not access or use the report .

VibrantWellness
3521 Leonard Ct, Santa Clara, CA 95054
1-866-364-0963 | support@vibrant-america.com | www.vibrant-wellness.com

TNP Test not performed R&L Refer to risks and limitations at the end of report Notes Refer to Lab notes at the end of the table

Nutrient Baseline

INTRODUCTION

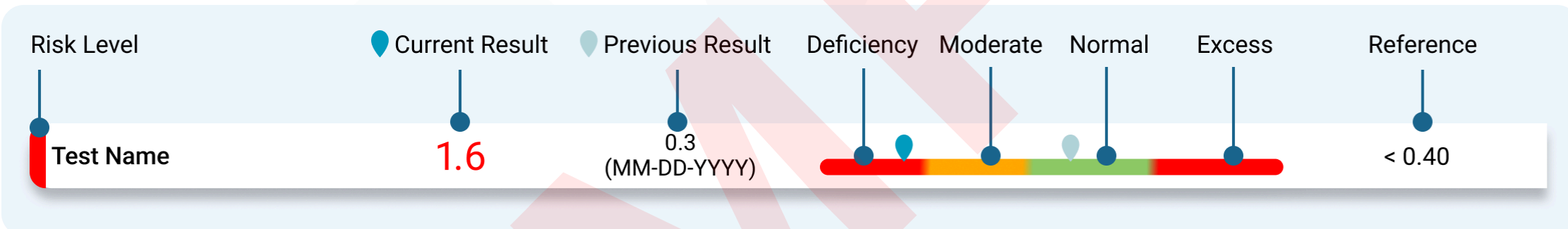
Vibrant Wellness is pleased to present Nutrient Zoomer Baseline testing to support healthy lifestyle choices in consultation with your healthcare provider. Nutrient Baseline measures circulating nutrient levels to evaluate nutrient availability in the extracellular space, reflecting recent intake and short-term nutritional status. Results are intended to be interpreted by healthcare providers to help inform personalized nutrition and wellness recommendations related to energy production and metabolic function.

Methodology

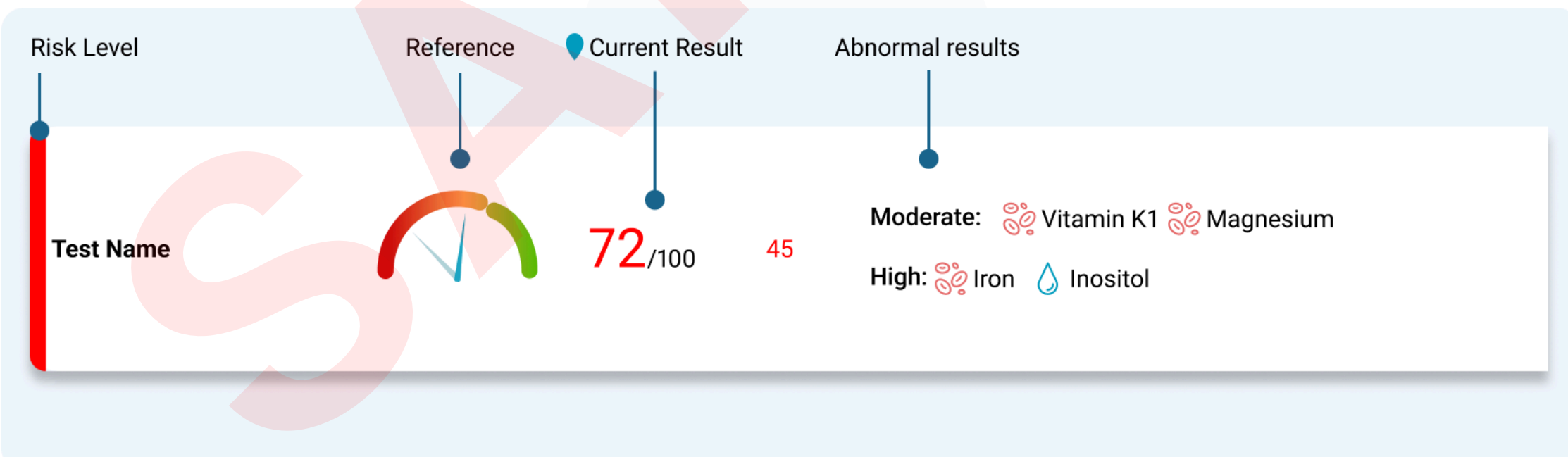
The Nutrient Baseline panel uses tandem liquid chromatography mass spectrometry methodology (LC-MS/MS) for quantitative detection of the Intracellular (RBC) and Extracellular (Serum) Micronutrients markers and uses inductively coupled plasma mass spectrometry (ICP-MS) for quantitative detection metals in serum (extracellular), and RBC (intracellular).

Interpretation of Report

The Vibrant Nutrient Zoomer report begins with the Summary which provides concise information on the abnormal serum and cellular analytes along with corresponding results from previous testing (if applicable). This is followed by a complete list of all analytes tested with quantitative results to enable a full overview along with the corresponding reference ranges. Reference ranges have been established using a cohort of 1000 apparently healthy adults over 18 years of age, and pediatric reference ranges are not available. The classification of Red indicates a result that is outside the reference range, and the classification of Green denotes a result that is within the reference range. Values are adjusted for white blood cell (WBC) counts where applicable. The patient's value is considered deficient if it falls on the far left (red) side of the reference range. The patient's value is considered in excess if it is on the far right (red) side of the reference range. The patient is considered moderately deficient if their result falls within the yellow (moderate) area of the reference range. The patient's value is considered normal if it falls within the green area of the reference range. The current result and previous result are listed to the left of the reference range. The reference metric, used to establish the reference range, is listed to the right of the reference range illustration (see image below)



A weightage is assigned to each nutrient in each category, based on its clinical relevance, structure, and function specific to that category. A score dial is also used to illustrate the patients' nutrient scores. A green score is ≥ 80 and is considered optimal. A yellow score is ≥ 70 and <80 and indicates a moderate risk of deficiency. A red score is any result below 70 and is considered deficient. (see image below).



Please note: Consider all supplements in relation to medical history and symptoms. Not all recommended supplements are appropriate in all individual cases. It is important that you discuss any modifications to your diet, exercise, drug, and/or nutritional supplementation with your healthcare provider before making any changes. Vitamin and mineral suggestions are based on age-matched and gender-matched RDAs and ULs.

Regulatory Disclaimer: This test was performed by Vibrant America Clinical Laboratory at 3521 Leonard Ct, Santa Clara, CA 95054 (CLIA No. 05D2078809, CAP No. 8970308). This test was developed, and its performance characteristics determined, by Vibrant America Clinical Laboratory. This test has not been cleared or approved by the U.S. Food and Drug Administration (FDA).

Nutrient Baseline - Summary

Nutrient Baseline - Summary

Serum RBC

Test Name	Reference	Current	Previous	Abnormal

Bone, Joint and Muscle Health

30.0/100

High: Copper (Cu), Iron (Fe), AA/EPA Ratio

Low: Vitamin D3 (Cholecalciferol), Vitamin D (25-Hydroxy Vitamin D), Calcium (Ca), Vitamin K2 (Menaquinone-4), Magnesium (Mg), Vitamin C (Ascorbic Acid), Vitamin K1 (Phylloquinone), Zinc (Zn), Magnesium (Mg), EPA (Eicosapentaenoic Acid), DHA (Docosahexaenoic Acid)

Cardiovascular Health

22.0/100

High: AA/EPA Ratio, Iron (Fe)

Moderate: Omega 3 Index

Low: Magnesium (Mg), Coenzyme Q10 (Co Q10), Vitamin B12 (Cobalamin), Vitamin K2 (Menaquinone-4), Vitamin B6 (Pyridoxal 5'-Phosphate), L-Arginine, Vitamin E (Alpha Tocopherol), Selenium (Se), Free Carnitine, L-Citrulline, EPA (Eicosapentaenoic Acid), DHA (Docosahexaenoic Acid), Magnesium (Mg), DPA (Docosapentaenoic Acid)

Gastrointestinal Barrier

38.0/100

High: Iron (Fe), Linoleic Acid (LA), AA/EPA Ratio

Low: L-Glutamine, Zinc (Zn), Vitamin A (Retinol), Vitamin D (25-Hydroxy Vitamin D), Selenium (Se), L-Arginine, DHA (Docosahexaenoic Acid), EPA (Eicosapentaenoic Acid), Magnesium (Mg)

SAMPLE

Nutrient Baseline - Summary

Nutrient Baseline - Summary

Serum RBC

Test Name	Reference	Current	Previous	Abnormal
Liver Detoxification	≥80	34.0/100		High: Iron (Fe), AA/EPA Ratio, Linoleic Acid (LA) Moderate: Omega 3 Index Low: Choline, L-Glutamine, Selenium (Se), Vitamin B12 (Cobalamin), Vitamin B2 (Riboflavin), Vitamin B6 (Pyridoxal 5'-Phosphate), Vitamin B3 (Niacin), Zinc (Zn), Magnesium (Mg), Magnesium (Mg), EPA (Eicosapentaenoic Acid), DHA (Docosahexaenoic Acid)
Mitochondrial Function	≥80	28.0/100		High: Iron (Fe), Linoleic Acid (LA), AA/EPA Ratio Low: Coenzyme Q10 (Co Q10), Free Carnitine, Magnesium (Mg), Vitamin B1 (Thiamine), Vitamin B2 (Riboflavin), Vitamin B3 (Niacin), Vitamin B5 (Pantothenic Acid), Vitamin C (Ascorbic Acid), Vitamin E (Alpha Tocopherol), L-Arginine, Magnesium (Mg), DHA (Docosahexaenoic Acid), EPA (Eicosapentaenoic Acid), DPA (Docosapentaenoic Acid)
Skin and Anti-Aging	≥80	24.0/100		High: Copper (Cu), Linoleic Acid (LA), Iron (Fe), AA/EPA Ratio Moderate: Omega 3 Index Low: Vitamin C (Ascorbic Acid), Vitamin E (Alpha Tocopherol), Vitamin A (Retinol), Zinc (Zn), Selenium (Se), Coenzyme Q10 (Co Q10), Vitamin B3 (Niacin), Vitamin B5 (Pantothenic Acid), DHA (Docosahexaenoic Acid), EPA (Eicosapentaenoic Acid), Magnesium (Mg)

SAMPLE

Nutrient Baseline - Summary

Nutrient Baseline - Summary

Serum RBC

Test Name	Reference	Current	Previous	Abnormal

Neurological, Cognitive Function and Mood

32.0/100

High: Iron (Fe) AA/EPA Ratio

Moderate: Omega 3 Index

Low: Vitamin B12 (Cobalamin) Choline
Magnesium (Mg)
Vitamin B6 (Pyridoxal 5'-Phosphate)
Zinc (Zn) Iodine (I)
Vitamin D (25-Hydroxy Vitamin D)
Vitamin B1 (Thiamine) Selenium (Se)
Myo-Inositol (Inositol)
Vitamin E (Alpha Tocopherol)
DHA (Docosahexaenoic Acid)
Magnesium (Mg)
EPA (Eicosapentaenoic Acid)
DPA (Docosapentaenoic Acid)

SAMPLE

Patient Name: DEMO DEMO
Date of Birth: 00-00-0000 Accession ID: 2322874238
Service Date: 2026-06-19 11:30 (PDT)

Nutrient Baseline - All Markers

VITAMINS

FAT-SOLUBLE		Current	Previous	Result	Reference
Vitamin A (Retinol)	Serum	38.9		0 40.7 154	40.8-154.5 (mcg/dL)
Vitamin D3 (Cholecalciferol)	Serum	0.3		0 0.3 1.8	0.4-1.8 (ng/mL)
Vitamin D (25-Hydroxy Vitamin D)	Serum	16.4		0 29.9 108	30.0-108.0 (ng/mL)
Vitamin E (Alpha Tocopherol)	Serum	6.8		0 7.3 30.6	7.4-30.6 (mg/L)
Vitamin K1 (Phylloquinone)	Serum	0.09		0 0.09 8.1	0.1-8.1 (ng/mL)
Vitamin K2 (Menaquinone-4)	Serum	0.08		0 0.09 5.19	0.1-5.19 (ng/mL)
Coenzyme Q10 (Co Q10)	Serum	0.52		0 0.55 2.78	0.56-2.78 (µg/mL)

WATER-SOLUBLE		Current	Previous	Result	Reference
Vitamin B1 (Thiamine)	Serum	1.1		0 1.3 71.3	1.4-71.3 (nmol/L)
Vitamin B2 (Riboflavin)	Serum	5.2		0 5.5 126	5.6-126.1 (mcg/L)
Vitamin B3 (Niacin)	Serum	2.3		0 2.5 36.1	2.6-36.1 (ng/mL)
Vitamin B5 (Pantothenic Acid)	Serum	21.4		0 22.6 429	22.7-429.2 (mcg/L)
Vitamin B6 (Pyridoxal 5'-Phosphate)	Serum	2.5		0 2.7 76.2	2.8-76.2 (ng/mL)
Vitamin B9 (Folate)	Serum	5.8		0 4.5	≥4.6 (ng/mL)
	RBC	101.0		0 95.4	≥95.5 (ng/mL)
Vitamin B12 (Cobalamin)	Serum	205.0		0 231 1245	232.0-1245.0 (pg/mL)
MMA (Methylmalonic Acid)	Serum	0.62		0 0.09 0.5	0.1-0.5 (nmol/mL)
Vitamin C (Ascorbic Acid)	Serum	0.1		0 0.1 1.1	0.2-1.1 (mg/dL)
Myo-Inositol (Inositol)	Serum	19.6		0 20.4 60.7	20.5-60.7 (nmol/mL)
Choline	Serum	5.9		0 6.7 31	6.8-31.0 (nmol/mL)

Patient Name: **DEMO DEMO**
Date of Birth: **00-00-0000** Accession ID: **2322874238**
Service Date: **2026-06-19 11:30 (PDT)**

Nutrient Baseline - All Markers

MINERALS

Test Name		Current	Previous	Result	Reference
Iron (Fe)	Serum	92.6		0 36 145	37.0-145.0 (ug/dL)
	RBC	121.0		0 88.8 117	88.9-117.0 (mg/dL)
Magnesium (Mg)	Serum	1.3		1 1.5 2.6	1.6-2.6 (mg/dL)
	RBC	3.2		0 3.5 7.7	3.6-7.7 (mg/dL)
Manganese (Mn)	Serum	0.3		0 0.2 2	0.3-2.0 (ng/mL)
Calcium (Ca)	Serum	8.5		6.5 8.8 10.6	8.9-10.6 (mg/dL)
Potassium (K)	Serum	4.5		3 3.4 5.1	3.5-5.1 (mmol/L)
	RBC	377.1		0 360 466	360.9-466.3 (mg/dL)
Sodium (Na)	Serum	140.0		125 135 145	136.0-145.0 (mmol/L)
Chromium (Cr)	Serum	0.08		0 0.09 0.7	0.1-0.7 (ng/mL)
Selenium (Se)	Serum	104.0		0 109 218	109.8-218.4 (ng/mL)
Iodine (I)	Serum	40.0		0 42.6 91.8	42.7-91.8 (ng/mL)
Zinc (Zn)	Serum	0.4		0 0.4 1	0.5-1.0 (mcg/mL)
Copper (Cu)	Serum	2.0		0 0.5 1.8	0.6-1.8 (mcg/mL)
Copper to Zinc Ratio (Cu:Zn)	Serum	5.0		0 0.8 2.6	0.9-2.6

AMINO ACIDS

Test Name		Current	Previous	Result	Reference
L-Arginine	Serum	74.2		0 81.5 249	81.6-249.0 (nmol/mL)
L-Citrulline	Serum	16.5		0 18.6 47.5	18.7-47.5 (nmol/mL)
L-Cysteine	Serum	14.2		0 3.3 37	3.4-37.0 (nmol/mL)
L-Glutamine	Serum	382.0		0 393 699	393.5-699.3 (nmol/mL)
L-Serine	Serum	121.7		0 94.1 246	94.2-246.8 (nmol/mL)
L-Asparagine	Serum	48.3		0 39.1 89.8	39.2-89.8 (nmol/mL)

Patient Name: **DEMO DEMO**
Date of Birth: **00-00-0000** Accession ID: **2322874238**
Service Date: **2026-06-19 11:30 (PDT)**

Nutrient Baseline - All Markers

AMINO ACIDS

Test Name		Current	Previous	Result	Reference
Free Carnitine	Serum	10.8		0 / 11.5 / 43.4	11.6-43.4 (nmol/mL)
Branched Chain AAs		**Current**	**Previous**	**Result**	**Reference**
L-Isoleucine	Serum	96.4		0 / 25.4 / 158	25.5-158.9 (nmol/mL)
L-Leucine	Serum	178.2		0 / 101 / 249	101.2-249.3 (nmol/mL)
L-Valine	Serum	221.6		0 / 155 / 368	155.9-368.0 (nmol/mL)

FATTY ACIDS

AA/EPA		Current	Previous	Result	Reference
AA/EPA Ratio	RBC	>127		0 / 2.4 / 10.9	2.5-10.9
OMEGA-3		**Current**	**Previous**	**Result**	**Reference**
DHA (Docosahexaenoic Acid)	RBC	2.10		0 / 2.41 / 10.5	2.42-10.52 (%)
EPA (Eicosapentaenoic Acid)	RBC	0.12		0 / 0.14 / 2.26	0.15-2.26 (%)
DPA (Docosapentaenoic Acid)	RBC	0.39		0 / 0.44 / 1.8	0.45-1.8 (%)
Total Omega-3	RBC	2.90		0 / 3.24 / 13.9	3.25-13.99 (%)
Omega 3 Index	RBC	3.10		0 / 2.65 / 7.99 / 12.6	8.0-12.65 (%)
OMEGA-6		**Current**	**Previous**	**Result**	**Reference**
Arachidonic Acid (AA)	RBC	17.60		0 / 5.49 / 19.0	5.5-19.01 (%)
Linoleic Acid (LA)	RBC	11.40		0 / 3.21 / 10.4	3.22-10.49 (%)
Total Omega-6	RBC	36.50		0 / 11.0 / 34.9	11.03-34.96 (%)

SAMPLE

Nutrient Baseline - Summary

VITAMINS

FAT-SOLUBLE		Current	Previous	Result	Reference
Vitamin A (Retinol)	Serum	38.9		0 / 40.7 / 154	40.8-154.5 (mcg/dL)

PHYSIOLOGICAL FUNCTION

Vitamin A is a group of fat-soluble vitamins which includes retinol, retinal, retinoic acid, and several provitamin A carotenoids, among which beta-carotene is the most important. Vitamin A has multiple functions including: growth and development in infants, children and adolescents, maintenance of the immune system, and healthy vision. Vitamin A is needed by the retina of the eye for both low-light and color vision. Vitamin A also functions as retinoic acid, an important hormone-like growth factor for epithelial and other cells. Other important roles that vitamin A plays in the body include: gene transcription, haematopoiesis, and antioxidant activity.

HOW IT GETS DEPLETED

Vitamin A deficiency may occur with chronic alcoholism, zinc deficiency, hypothyroidism, and use of laxatives.

CLINICAL MANIFESTATIONS OF DEPLETION

Vitamin A deficiency may result in night blindness, impaired immunity, impaired healing, increased risk of infection, thyroid disorders, leukoplakia or keratosis. Assess zinc status to assess if zinc deficiency has led to secondary functional deficiency of vitamin A release from liver stores.

FOOD SOURCES

Food sources of vitamin A include: cod liver oil, liver (turkey, beef, pork, fish and chicken), dandelion greens, fortified cereals and milk, butter, eggs, sweet potato, pumpkin, carrot, cantaloupe, mango, spinach, broccoli leaf (broccoli florets have much less), kale, and butternut.

SUPPLEMENT CONSIDERATION

The RDA for vitamin A is 700mcg RAE/day for women and 900 mcg RAE/day for men. This is the amount needed to prevent chronic deficiency, but more may be needed 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 vitamin A in adults is set at 3,000 µg RAE/day. Vitamin A toxicity can occur from taking pre-formed vitamin A from sources other than plant sources.

SAMPLE

VITAMINS

FAT-SOLUBLE		Current	Previous	Result	Reference
Vitamin D3 (Cholecalciferol)	Serum	0.3		0 0.3 1.8	0.4-1.8 (ng/mL)

PHYSIOLOGICAL FUNCTION

Vitamin D3 is also referred to as 1,25-hydroxyvitamin D3 (1,25-OHD3). 1,25-OHD3 has a shorter half-life in the blood than 25-OHD (what most standard labs run, and what is most commonly used to assess total vitamin D status), and, therefore, levels may differ from what is measured as 25-OHD. The conversion of 25-OHD to 1,25-OHD is performed in the kidneys and regulated by parathyroid hormone (PTH). When blood calcium levels fall, PTH signals the kidneys to convert more 25-OHD to 1,25-OHD, which increases intestinal absorption of calcium, and reduces bone demineralization of calcium. Vitamin D3 also regulates the function of hundreds of genes, supports the immune system, supports production and function of endocrine hormones, is important 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-OHD to 1,25-OHD.

HOW IT GETS DEPLETED

Vitamin D deficiency is very common in the U.S. The most common reasons for vitamin D deficiency include: lack of sun exposure and regular use of sunscreen. Individuals with darker pigmented skin are at greater risk for vitamin D deficiency. Chronic liver disease and kidney failure are risk factors for vitamin D deficiency. Patients who present with hypercalcemia, hyperphosphatemia, and low PTH may suffer from unregulated conversion of 25-OHD to 1,25-OHD. Some medications can deplete vitamin D: anti-inflammatory medications, antibiotics, anticonvulsant medications, cholesterol lowering medications, laxatives and anti-ulcer medications.

CLINICAL MANIFESTATIONS OF DEPLETION

Conditions associated with low vitamin D status include: Alzheimer's disease, asthma, autism, cancer, cavities, colds and flus, cystic fibrosis, dementia, depression, diabetes 1 and 2, eczema and psoriasis, hearing loss, heart disease, hypertension, infertility, inflammatory bowel disease, insomnia, macular degeneration, migraines, multiple sclerosis, Crohn's disease, muscle pain, obesity, osteomalacia, osteoporosis, periodontal disease, preeclampsia, rheumatoid arthritis, schizophrenia, seizures, septicemia, and tuberculosis. Reasons for suboptimal vitamin D3 levels include lack of sun exposure (particularly in northern latitudes and during the winter season), malabsorption (due to Celiac disease, or other inflammatory digestive disorders), inadequate hepatic vitamin D 25-hydroxylase enzyme activity, and some prescription medications such as antiepileptic drugs, including phenytoin, phenobarbital, and carbamazepine, that increase 25-OHD metabolism. Levels of PTH may be high-normal or elevated in sub-clinical and frank vitamin D deficiency.

FOOD SOURCES

Food sources of vitamin D include: dairy products, such as fortified milk and yogurt, fortified orange juice, egg yolks, liver, fatty fish, such as salmon, tuna, mackerel, sardines, shrimp, mushrooms grown in adequate sunlight, baker's yeast. Naturally occurring sources will contain vitamin D3, whereas fortified sources (baker's yeast) will contain D2.

SUPPLEMENT CONSIDERATION

The previously established RDA of 400IU/day has been found to be insufficient for therapeutic needs. Common doses are used between 1000 and 10,000 IU/day. Vitamin D comes in two forms: D2 (ergocalciferol) and D3 (cholecalciferol); both forms can be used as active vitamin D3 (cholecalciferol) in the body, as D2 can be converted to D3 when needed, however, this is a tightly controlled metabolic process in order to prevent excess D2 to D3 conversion. Vitamin D is produced endogenously when skin is exposed to ultraviolet light from the sun. Supplementation with Vitamin D is almost always necessary, as it is extremely difficult to meet needs through diet and sun exposure alone. Consult with your practitioner for supplement recommendations and target goal for serum levels. Because vitamin D can be stored or trapped in adipose tissue (fat cells), obese individuals and pregnant women have higher vitamin D requirements. Obtaining too much vitamin D from sun exposure is not possible, but it is possible to obtain too much from supplementation. Taking too much vitamin D in supplement form can also cause an increase in blood levels of calcium, or hypercalcemia, due to increased intestinal absorption of calcium when serum vitamin D levels are high. Supplementing vitamin K2 alongside vitamin D is recommended in order to keep blood levels of calcium in homeostasis and prevent excess bone demineralization of calcium with higher vitamin D intake. Vitamin D toxicity has been observed in individuals taking greater than 50,000 IU/day, but intake levels less than 10,000 IU/day are unlikely to cause toxicity.

SAMPLE

Nutrient Baseline - Summary

VITAMINS

FAT-SOLUBLE		Current	Previous	Result	Reference
Vitamin D (25-Hydroxy Vitamin D)	Serum	16.4		0 29.9 108	30.0-108.0 (ng/mL)

PHYSIOLOGICAL FUNCTION

25-hydroxyvitamin D is a standard lab test which measures the inactive precursor to 1,25-OHD, which is a combination of two forms of vitamin D in the body: vitamin D2 and vitamin D3. 25-OHD has a longer half-life in the blood than 1,25-OHD, and, therefore, levels may differ from levels of active 1,25-OHD3. Because 25-OHD is a precursor to active forms of vitamin D, it is important to note that it is not reflective of overall active D3 levels, but rather what is available for conversion if cofactors are sufficient. The conversion of 25-OHD to 1,25-OHD is performed in the kidneys and regulated by parathyroid hormone (PTH). When blood calcium levels fall, PTH signals the kidneys to convert more 25-OHD to 1,25-OHD, which increases intestinal absorption of calcium, and reduces bone demineralization of calcium. Upon conversion to 1,25-OHD, it also regulates the function of hundreds of genes, supports the immune system, supports production and function of endocrine hormones, is important 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.

HOW IT GETS DEPLETED

Vitamin D deficiency is very common in the U.S. The most common reasons for vitamin D deficiency include: lack of sun exposure and regular use of sunscreen. Individuals with darker pigmented skin are at greater risk for vitamin D deficiency. Chronic liver disease and kidney failure are risk factors for vitamin D deficiency. Patients who present with hypercalcemia, hyperphosphatemia, and low PTH may suffer from unregulated conversion of 25-OH-VitD to 1,25-OHD. Some medications can deplete vitamin D: anti-inflammatory medications, antibiotics, anticonvulsant medications, cholesterol lowering medications, laxatives and anti-ulcer medications.

CLINICAL MANIFESTATIONS OF DEPLETION

Conditions that have been associated with low vitamin D status include: Alzheimer's disease, asthma, autism, cancer, cavities, colds and flus, cystic fibrosis, dementia, depression, diabetes 1 and 2, eczema and psoriasis, hearing loss, heart disease, hypertension, infertility, inflammatory bowel disease, insomnia, macular degeneration, migraines, multiple sclerosis, Crohn's disease, muscle pain, obesity, osteomalacia, osteoporosis, periodontal disease, preeclampsia, rheumatoid arthritis, schizophrenia, seizures, septicemia, and tuberculosis. Reasons for suboptimal 25-OHD levels, specifically, include lack of sun exposure (particularly in northern latitudes and during the winter season), malabsorption (due to Celiac disease, or other inflammatory digestive disorders), inadequate hepatic vitamin D 25-hydroxylase enzyme activity, and some prescription medications such as antiepileptic drugs, including phenytoin, phenobarbital, and carbamazepine, that increase 25-OHD metabolism. Levels of PTH may be high-normal or elevated in sub-clinical and frank vitamin D deficiency.

FOOD SOURCES

Food sources of vitamin D include: dairy products, such as fortified milk and yogurt, fortified orange juice, egg yolks, liver, fatty fish, such as salmon, tuna, mackerel, sardines, shrimp, mushrooms grown in adequate sunlight, baker's yeast. Naturally occurring sources will contain vitamin D3, whereas fortified sources (baker's yeast) will contain D2.

SUPPLEMENT CONSIDERATION

The previously established RDA of 400IU/day has been found to be insufficient for therapeutic needs. Common doses are used between 1000 and 10,000 IU/day. Vitamin D comes in two forms: D2 (ergocalciferol) and D3 (cholecalciferol); both forms can be converted to active vitamin D in the body (25-hydroxyvitamin D). Vitamin D is produced when skin is exposed to ultraviolet light from the sun. Supplementation with Vitamin D is almost always necessary, as it is extremely difficult to meet needs though diet and sun exposure alone. Consult with your practitioner for supplement recommendations and target goal for serum levels. Because vitamin D can be stored or trapped in adipose tissue (fat cells) obese individuals and pregnant women have higher vitamin D requirements. Obtaining too much vitamin D from sun exposure is not possible, but it is possible to obtain too much from supplementation. Taking too much vitamin D in supplement form can also cause in increase in blood levels of calcium, or hypercalcemia, due to increased intestinal absorption of calcium when serum vitamin D levels are high. Vitamin D toxicity has been observed in individuals taking greater than 50,000 IU/day, but intake levels less than 10,000 IU/day are unlikely to cause toxicity.

SAMPLE

Nutrient Baseline - Summary

VITAMINS

FAT-SOLUBLE		Current	Previous	Result	Reference
Vitamin E (Alpha Tocopherol)	Serum	6.8		0 / 7.3 / 30.6	7.4-30.6 (mg/L)

PHYSIOLOGICAL FUNCTION

Vitamin E 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.

HOW IT GETS DEPLETED

Vitamin E may become depleted or deficient due to intestinal malabsorption. Smoking also depletes the body's vitamin E stores.

CLINICAL MANIFESTATIONS OF DEPLETION

Vitamin E deficiency may result in peripheral neuropathy, ataxia, muscle weakness, skeletal myopathy, retinopathy, and increased risk of CVD, red blood cell destruction, prostate cancer, and cataracts.

FOOD SOURCES

Dietary sources of vitamin E include: plant seeds (such as sunflower seeds), walnuts, hazelnuts, olive oil, canola oil, wheat germ oil, sunflower oil, safflower oil, tomato, avocado, spinach, and Swiss chard.

SUPPLEMENT CONSIDERATION

The RDA for vitamin E is 15mg/day. The UL for vitamin E 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. In addition, α-Lipoic acid is an important co-factor that can aid in restoring vitamin E levels when depleted.

Vitamin K1 (Phylloquinone)	Serum	0.09		0 / 0.09 / 8.1	0.1-8.1 (ng/mL)

PHYSIOLOGICAL FUNCTION

Vitamin K is a group of fat-soluble vitamins. This group of vitamins includes two natural vitamins: vitamin K1 and vitamin K2. These Vitamins are structurally similar and their name comes from the German word 'klotting'. Vitamin K1, also known as phylloquinone, assists with blood clotting, supports the formation of bone and bone matrix, and aids in glucose to glycogen conversion for storage in the liver.

HOW IT GETS DEPLETED

Dietary deficiency of vitamin K 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 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. Since Vitamin K is a fat-soluble vitamin, following a chronically low-fat diet can inhibit absorption.

CLINICAL MANIFESTATIONS OF DEPLETION

Symptoms of vitamin K depletion or deficiency include: excessive bleeding, menorrhagia, bruises that form easily, or appearance of ruptured capillaries.

FOOD SOURCES

The best sources of Vitamin K1 are plant foods, especially dark green leafy vegetables. Note: the absorption of vitamin K1 from food is extremely low. Only 10 percent of the vitamin K, which is found in green leafy vegetables, is absorbed in your body. There's no variable or modification of the consumption that will significantly increase the absorption.

SUPPLEMENT CONSIDERATION

The AI for vitamin K is set at 90 µg/day for women and 120 µg/day for men. Individuals who are on certain anti-clotting medications should consult with their medical provider about their dietary vitamin K intake. Individuals suffering from blood clotting disorders, osteoporosis, coronary artery disease, cancer, liver disease, celiac disease, Crohn's disease, ulcerative colitis or cystic fibrosis should discuss dietary intake of vitamin K with their healthcare provider.

SAMPLE

Nutrient Baseline - Summary

VITAMINS

FAT-SOLUBLE		Current	Previous	Result	Reference
Vitamin K2 (Menaquinone-4)	Serum	0.08		0 / 0.09 / 5.19	0.1-5.19 (ng/mL)

PHYSIOLOGICAL FUNCTION

Vitamin K is a group of fat-soluble vitamins. This group of vitamins includes two natural vitamins: vitamin K1 and vitamin K2. Vitamin K2 is the main storage form of Vitamin K in animals. It has several forms, referred to as menaquinones. The nomenclature denoting vitamin K2 types will include an 'MK' to specify this is a menaquinone, and the number following this denotes how many isoprenyl units are on the side chain of the molecule. The most common forms are MK-4 and MK-7. Bacteria in the colon can convert K1 (from plant-based foods) into vitamin K2. Vitamin K2 is necessary to prevent arterial calcification, which it does by activating matrix GLA protein (MGP). This matrix GLA protein is present in blood vessels and inhibits soft tissue calcification. Matrix GLA protein needs to be carboxylated to work properly, and Vitamin K2-MK7 plays a major role in this carboxylation.

HOW IT GETS DEPLETED

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. In addition, the use of oral blood-thinning medications and some antibiotics can interfere with vitamin K. 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. Since Vitamin K is a fat-soluble vitamin, following a chronically low-fat diet can inhibit absorption.

CLINICAL MANIFESTATIONS OF DEPLETION

Inadequate levels of both Vitamin K1 and K2 will radically increase risk for heart disease and stroke. Chronically low vitamin K levels can lead to uncontrolled bleeding and chronic marginally low vitamin K levels are correlated in some studies with osteoporosis. Because vitamin K2 also assists in calcium homeostasis, low or deficient levels of vitamin K2 can lead to unregulated calcium release from bone tissue sources in the presence of vitamin D3 supplementation. Supplementation of vitamin D2 does not tend to lead to this, however. It is recommended that vitamin K2 be supplemented when vitamin D3 is supplemented. Levels of K2 are inversely related to cardiovascular disease and coronary calcification.

FOOD SOURCES

The best sources of vitamin K2 include some fermented foods predominantly natto and some rare fermented cheeses, and liver. There are minor amounts present in egg yolk and butter.

SUPPLEMENT CONSIDERATION

Studies suggest daily therapeutic doses of about 360-500 micrograms (mcg) of vitamin K2. Fermented foods contain a wide variety of different bacteria, and only certain ones—such as Bacillus subtilis—actually make vitamin K2. Dietary vitamin K2 intake is enhanced with regular consumption of fermented foods. You can make fermented foods yourself, by using a starter culture specifically designed to optimize K2. Vitamin K2 supplements come in 'MK' varieties, and MK-4 is what all forms of vitamin K2 are converted in vivo. If one takes an MK-7 variety, the body will convert it to MK-4; however, MK-4 supplements can be found commercially to bypass activation after absorption.

SAMPLE

Nutrient Baseline - Summary

VITAMINS

FAT-SOLUBLE		Current	Previous	Result	Reference
Coenzyme Q10 (Co Q10)	Serum	0.52		0 / 0.55 / 2.78	0.56-2.78 (µg/mL)

PHYSIOLOGICAL FUNCTION

CoQ10 is a fat-soluble compound primarily synthesized by the body and also 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.

HOW IT GETS DEPLETED

CoQ10 is most commonly depleted through use of cholesterol-lowering medication, such as statins. Other causes of CoQ10 deficiency include genetic mutations that limit biosynthesis, unknown reasons in the aging process, cancer, and smoking.

CLINICAL MANIFESTATIONS OF DEPLETION

Signs of CoQ10 deficiency include muscle weakness and fatigue, high blood pressure, and slowed thinking; more extreme symptoms of CoQ10 deficiency include chest pain, heart failure, and seizures.

FOOD SOURCES

Food sources of CoQ10 are considered poor sources of the nutrient. Foods that contain more CoQ10 than others include organ meats from red meat sources. Nuts are considered a moderate source but would have to be eaten in extreme amounts to get the daily requirement.

SUPPLEMENT CONSIDERATION

There is currently no established RDA, AI, or UL for CoQ10. CoQ10 comes in both ubiquinone and ubiquinol forms; ubiquinol is considered the active form, however, the body uses both forms as needed. Typical doses required to restore minimum CoQ10 levels while using statin drugs are 100-200 mg/day. Intestinal absorption of CoQ10 is limited, but optimized if consumed with a meal containing fat. There are really no adverse symptoms with high dose CoQ10 supplementation; however, supplementation is typically not recommended for pregnant or lactating women due to lack of controlled studies.

SAMPLE

VITAMINS

WATER-SOLUBLE		Current	Previous	Result	Reference
Vitamin B1 (Thiamine)	Serum	1.1		0 / 1.3 / 71.3	1.4-71.3 (nmol/L)

PHYSIOLOGICAL FUNCTION

Vitamin B1 aids in energy transformation and production of ATP. It acts as a coenzyme in the breakdown of carbohydrates, fats and proteins to produce energy.

HOW IT GETS DEPLETED

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. Can be depleted with excessive or chronic alcohol intake. There may be higher risk of depletion with gastric bypass surgery.

CLINICAL MANIFESTATIONS OF DEPLETION

Thiamin deficiency can lead to nervous system and cardiac abnormalities. The most severe form of thiamin deficiency is called beri beri, a condition commonly resulting in weakness, fatigue, confusion, irritability, weight loss, muscle wasting, and peripheral neuropathy.

FOOD SOURCES

Food sources of thiamin include: pork, organ meats, legumes, sweet potato, brown rice, brewer's yeast, pine nuts, sunflower seeds, enriched grains.

SUPPLEMENT CONSIDERATION

The RDA for thiamin is 1.0 mg/day for females and 1.2 mg/day for males. The RDA for thiamin during pregnancy/lactation is 1.4 mg/day. Therapeutic intake of thiamin is commonly 25-100mg/day. No UL for thiamin has been set. Thiamin is commonly found in multi-B vitamin products.

Vitamin B2 (Riboflavin)	Serum	5.2		0 / 5.5 / 126	5.6-126.1 (mcg/L)

PHYSIOLOGICAL FUNCTION

Two very important coenzymes involved in energy metabolism are derived from riboflavin to participate in oxidation/reduction reactions. Riboflavin is also essential for NOS enzyme (nitric oxide synthase) and glutathione reductase which regenerates glutathione, and which is very important for antioxidation/detoxification.

HOW IT GETS DEPLETED

Riboflavin is commonly depleted by excessive or chronic alcohol consumption. Need for riboflavin is increased in the elderly.

CLINICAL MANIFESTATIONS OF DEPLETION

Frank deficiency of riboflavin is rare, however, marginal deficiency is common. Deficiency of riboflavin is associated with fatigue/weakness.

FOOD SOURCES

Food sources high in riboflavin include: organ meats, dairy foods, eggs, leafy greens (spinach), broccoli, and liver. *Enriched grains include riboflavin.

SUPPLEMENT CONSIDERATION

The RDA for riboflavin is 1.7 mg/day. Common levels of therapeutic intake of riboflavin are 25-50 mg/day. No UL for riboflavin has been set.

SAMPLE

Nutrient Baseline - Summary

VITAMINS

WATER-SOLUBLE		Current	Previous	Result	Reference
Vitamin B3 (Niacin)	Serum	2.3		0 / 2.5 / 36.1	2.6-36.1 (ng/mL)

PHYSIOLOGICAL FUNCTION

Niacin is extensively involved in metabolic reduction reactions through NAD-NADPH pathways. Over 200 enzymes in the human body require niacin. Other important major functions of niacin include: fatty acid synthesis, ATP synthesis, DNA repair, lower cholesterol/LDL, and aids in circulation.

HOW IT GETS DEPLETED

Synthesized from tryptophan and uses iron, B6, and riboflavin as cofactors; deficiencies of these companion nutrients may be underlying causes. Can be depleted by oral contraceptives and statin drugs.

CLINICAL MANIFESTATIONS OF DEPLETION

Symptoms of niacin deficiency include: vomiting, constipation, red tongue, headache, fatigue, and depression. Severe deficiency of niacin is called pellagra. Pellagra is commonly accompanied by the following 4Ds: dermatitis, diarrhea, dementia, death.

FOOD SOURCES

The most concentrated sources of niacin are in animal products (pork), peanuts/peanut butter, tofu, and eggs. Also consider food sources high in tryptophan. Enriched grains provide supplemental niacin.

SUPPLEMENT CONSIDERATION

The RDA for niacin is 20 mg/day. The UL for niacin is 35 mg/day, but oral administration up to 6g per day has been used without side effects. 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.

Vitamin B5 (Pantothenic Acid)	Serum	21.4		0 / 22.6 / 429	22.7-429.2 (mcg/L)

PHYSIOLOGICAL FUNCTION

Vitamin B5 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. Another significant function of B5 is to work with carnitine and CoQ10 for fatty acid oxidation/metabolism.

HOW IT GETS DEPLETED

It is possible to block absorption of B5 in the intestines by taking high doses of supplemental biotin.

CLINICAL MANIFESTATIONS OF DEPLETION

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.

FOOD SOURCES

Food sources of B5 include: beef, pork, chicken, fish, egg yolks, whole grains, legumes, lentils.

SUPPLEMENT CONSIDERATION

There is currently no RDA established for B5. The AI for B5 is 5 mg/day in adults, 6 mg/day during pregnancy, and 7 mg/day during lactation. Because breakdown of B5 is metabolically slow, and deficiency is rare, there is probably no need for supplementation.

SAMPLE

Nutrient Baseline - Summary

VITAMINS

WATER-SOLUBLE		Current	Previous	Result	Reference
Vitamin B6 (Pyridoxal 5'-Phosphate)	Serum	2.5		0 — 2.7 — 76.2	2.8-76.2 (ng/mL)

PHYSIOLOGICAL FUNCTION

There are 117 known 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. B6 converts tryptophan to serotonin and can modulate steroid hormone activity. B6 is involved in the methylation cycle and clearance of homocysteine along with folate and B12.

HOW IT GETS DEPLETED

Antibiotics can reduce B6 levels. Oral contraceptives can interfere with B6 metabolism. Food additives such as FD & C yellow #5 may interfere with B6. Drugs such as isoniazid and dopamine may interfere with vitamin B6. Alcoholics are thought to be most at risk for Vitamin B6 deficiency due to low dietary intakes and impaired metabolism.

CLINICAL MANIFESTATIONS OF DEPLETION

Depletion of vitamin B6 can manifest as impaired protein synthesis, growth failure, immune dysfunction, microcytic anemia (small RBC's), elevated homocysteine, depression/fatigue, or anxiety. B6 insufficiency should be considered in the instance of mood disorders, nervous system dysfunction, pregnancy, the use of oral contraceptives or amphetamines, and cigarette smoking.

FOOD SOURCES

Food sources of B6 include: beef, liver, poultry, and fish. There is a high abundance of B6 in plant foods such as: legumes, whole grains, lentils, soybeans, nuts, seeds, and non-citrus fruits. Vitamin B6 is better absorbed from animal sources.

SUPPLEMENT CONSIDERATION

The RDA for B6 is 2 mg/day. The UL for B6 is 100 mg/day. High dose supplements are sometimes used to relieve PMS and carpal tunnel syndrome. High dose B6 supplementation can cause neuropathies (nerve damage). Levels greater than 2 g/day have been shown to induce neuropathy or sensory neuropathy. Doses of greater than 150 mg may suppress lactation. Therapeutic range for vitamin B6 is considered to be 30 to 500 mg/day.

SAMPLE

VITAMINS

WATER-SOLUBLE		Current	Previous	Result	Reference
Vitamin B12 (Cobalamin)	Serum	205.0		0 / 231 / 1245	232.0-1245.0 (pg/mL)

PHYSIOLOGICAL FUNCTION

Vitamin B12 is an important coenzyme when in its active form of methylcobalamin. 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.

HOW IT GETS DEPLETED

Age is a risk factor for deficiency of B12 due to a natural decline in intrinsic factor. Chronic use of PPIs may reduce HCl and lead to sub-clinical deficiencies. Some genetic SNPs (such as MTHFR) may lead to deficiencies in active B12 (methylcobalamin).

CLINICAL MANIFESTATIONS OF DEPLETION

Deficiency of B12 can appear as pernicious anemia, usually due to lack of intrinsic factor. Another form of anemia associated with B12 deficiency is megoblastic anemia, when folate is in excess and insufficient B12 is present, which creates a 'folate trap.' Another symptom of B12 deficiency is dementia due to degeneration of myelin. In B12 deficiency, methylmalonyl CoA will be metabolized to methylmalonic acid (MMA), which is why MMA is considered the definitive marker for B12 deficiency. Achlorhydria (insufficient stomach acid) can lead to B12 deficiency because HCl is required to cleave B12 from intrinsic factor.

FOOD SOURCES

Vitamin B12 is synthesized by bacteria and exists in all animal foods. Vitamin B12 is only available from animal sources. The B12 synthesized by gut bacteria may not be a significant source for humans, as it is not absorbed in the colon.

SUPPLEMENT CONSIDERATION

The RDA for B12 is 6 mcg/day. Consider the upper limit of folate supplementation as a factor for the supplementation of B12, due to potential for folate trap. Vitamin B12 is extremely safe. No toxicity from high doses of vitamin B12 has ever been reported. Intramuscular injections are often used, particularly in the elderly to bypass intrinsic factor. Humans store large amounts of B12 in the liver so larger doses can be given at 6 month intervals. Supplementation is highly encouraged on a vegan diet. Due to high storage capacity in the liver, it may take years to deplete the body of B12 after adopting a vegan diet. Consider MTHFR genetic, and methyl cobalamin supplementation, particularly with hyperhomocysteinemia. Methylcobalamin is the recommended form of supplementation, but may be poorly absorbed in people taking antacids or those with very poor absorption (celiac, intestinal permeability, etc). Cyanocobalamin is not recommended for patients with MTHFR mutations. Hydroxocobalamin is recommended for patients with autoimmune diseases and elevated nitric oxide levels. Glutathione is also required for methylcobalamin to be bound for transport adequately. Vitamin B12 supplementation may help manage anemia, asthma, fatigue, hepatitis, dementia, epilepsy, depression, psychosis, irritability, ataxia, numbness, tingling, neuropathy, AIDS, multiple sclerosis, tinnitus, and infertility. Supplemental B12 is commonly given in 1000 to 5000 mcg doses.

MMA (Methylmalonic Acid)	Serum	0.62		0 / 0.09 / 0.5	0.1-0.5 (nmol/mL)

PHYSIOLOGICAL FUNCTION

Methylmalonyl-CoA is converted into succinyl-CoA in a reaction that requires B12 as a cofactor; succinyl-CoA then enters the Krebs cycle where it functions to produce energy.

CLINICAL MANIFESTATIONS OF EXCESS / RISK FOR TOXICITY

Excess MMA is not directly toxic but signals metabolic stress or vitamin B12 deficiency that requires correction.

FOOD SOURCES

There are no food sources of MMA. Food sources of B12 include: animal proteins, including dairy. *Fortified breakfast cereals may also contain B12.

SUPPLEMENT CONSIDERATION

Consider supplement recommendations for B12.

SAMPLE

Nutrient Baseline - Summary

VITAMINS

WATER-SOLUBLE		Current	Previous	Result	Reference
Vitamin C (Ascorbic Acid)	Serum	0.1		0 0.1 1.1	0.2-1.1 (mg/dL)

PHYSIOLOGICAL FUNCTION

Vitamin C has a major function of being an antioxidant. It boosts immunity through 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. Because of its role in the generation of connective tissue, it is necessary for optimal collagen production. Vitamin C is also an important component of l-carnitine, which is necessary for breakdown of fats into energy.

HOW IT GETS DEPLETED

Vitamin C is most commonly depleted in the absence of sufficient dietary intake. Vitamin C levels can be depleted during times of severe oxidative stress.

CLINICAL MANIFESTATIONS OF DEPLETION

Low levels of vitamin C have been associated with reduced bone density. Signs of deficiency include: bleeding gums, easy bruising, anemia, fatigue, weakness and joint pain. These symptoms are the result of weakened or deficient connective tissues throughout the body. Severe cases of vitamin C deficiency are called scurvy.

FOOD SOURCES

Food sources of vitamin C include: oysters, tropical fruits such as guava, papaya, pineapple, oranges, kiwi, and cantaloupe; leafy greens such as kale and spinach; cruciferous vegetables such as broccoli, brussel sprouts, cauliflower, and cabbage; berries, such as strawberries, raspberries, blueberries, blackberries, bell peppers, and amaranth.

SUPPLEMENT CONSIDERATION

The RDA for vitamin C is 75 mg/day for women and 90 mg/day for men. 120 mg/day is recommended during pregnancy and lactation. The half-life of vitamin C in circulation after supplementation is about 30 minutes, therefore, large singular doses of vitamin C may not be as therapeutic as smaller, more frequent doses of vitamin C. In addition to taking supplemental vitamin C, α-Lipoic acid helps restore vitamin C levels when depleted.

SAMPLE

Nutrient Baseline - Summary

VITAMINS

WATER-SOLUBLE		Current	Previous	Result	Reference
Myo-Inositol (Inositol)	Serum	19.6		0 / 20.4 / 60.7	20.5-60.7 (nmol/mL)

PHYSIOLOGICAL FUNCTION

Inositol derivatives are used in the cellular signaling process after the insulin receptor is activated; it is crucial for the development of peripheral nerves, helps move fats out of the liver, promotes the production of lecithin, and is anti-arteriosclerotic, and anti-atherogenic.

HOW IT GETS DEPLETED

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). If many courses of antibiotics are used, there may be some depletion of inositol from microbiome conversion. Inositol is also stored in the liver, spinal cord nerves, and in the brain and cerebral spinal fluid.

CLINICAL MANIFESTATIONS OF DEPLETION

There do not appear to be any clinical manifestations of depletion of inositol. Inositol can be synthesized in the human body from glucose-6-phosphate, a derivative of glucose, therefore, deficiency would be rare. Urinary levels of inositol derivatives (D-chiro-inositols and myo-inositols) are seen as a biomarker for insulin resistance. Conditions associated with depletion of inositol, however, are depression, anxiety, PCOS, diabetes, CVD, and obesity.

FOOD SOURCES

Good dietary sources of inositol include: oranges, cantaloupe, prunes, navy beans, grapefruit, limes, blackberries, kiwis, rutabagas, fresh green beans, unrefined molasses, stone ground wheat, bran flakes, and pumperknickel.

SUPPLEMENT CONSIDERATION

There is currently no established RDA, AI, or UL for inositol. Myo-inositol is noted for its benefits to female fertility and insulin sensitivity, and is used often in treatment for PCOS in dosages of 2-4g/day. Higher doses of inositol are used to treat psychiatric conditions like depression and anxiety/OCD in much higher doses of 12-18 g/day; some mild gastrointestinal distress is noted with the higher doses and may need to be consumed in split doses. Lowering blood glucose can be seen with doses of inositol around 2-4 g/day. Currently supplementation of inositol has shown some promise in treating Alzheimer's to reduce progression of fibril formation. Inositol may decrease LDL-C and ApoB in persons with metabolic syndrome with doses of 5-10 g/day. Doses of inositol of 4 g/day have been associated with improvement of all markers of glycemic control and insulin resistance in gestational diabetes.

SAMPLE

Nutrient Baseline - Summary

VITAMINS

WATER-SOLUBLE		Current	Previous	Result	Reference
Choline	Serum	5.9		0 / 6.7 / 31	6.8-31.0 (nmol/mL)

PHYSIOLOGICAL FUNCTION

Choline is metabolized within cellular mitochondria resulting in production of trimethylglycine; TMG plays a role in supporting methyl donation processes either directly (methylating homocysteine) or indirectly through supporting production of S-adenosyl methionine (SAMe). Choline is converted into acetylcholine (ACh).

HOW IT GETS DEPLETED

Depletion of choline is typically not a concern, and limited information exists on how depletion would happen primarily. However, lower intake of choline may lead to inefficient methylation.

CLINICAL MANIFESTATIONS OF DEPLETION

Deficiency in dietary choline is known to increase hepatic triglyceride accumulation. This results in lower blood triglycerides, but increased accumulation of triglycerides in the liver. Subjects with a mutation in the MTHFR enzyme seem to place more burden on choline in methylation cycles. Depletion of choline can also lead to muscle damage.

FOOD SOURCES

Eggs, liver, and peanuts are the best sources of choline. Poultry, fish, and cruciferous veggies are good sources of choline. *Dietary choline sources, including lecithin (phosphatidylcholine), may increase serum TMAO in humans, although the evidence is mixed.

SUPPLEMENT CONSIDERATION

The AI for choline is 425 mg/day for women and 550 mg/day for men. The UL is 3,500 mg/day. Choline bitartrate is the most common supplemental form of choline for most general purposes, such as liver health. CDP-Choline and Alpha-GPC are commonly used for nootropic purposes. Supplemental choline can enhance systemic methylation. Excessive consumption of choline ≥7,500 mg has been associated with low blood pressure, excessive sweating, fishy body odor, and gastrointestinal side effects.

SAMPLE

Nutrient Baseline - Summary

MINERALS

Test Name		Current	Previous	Result	Reference
Magnesium (Mg)	Serum	1.3		1 / 1.5 / 2.6	1.6-2.6 (mg/dL)
	RBC	3.2		0 / 3.5 / 7.7	3.6-7.7 (mg/dL)

PHYSIOLOGICAL FUNCTION

Important functions of magnesium include: assisting 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.

HOW IT GETS DEPLETED

Alcohol will lead to increased excretion in urine. Prolonged use of diuretics will lead to increased urinary excretion. Excessive sweating/long bouts of endurance exercise. Hyperparathyroidism, Chronic renal failure, malabsorptive conditions (celiac disease, Crohn's disease, partial bowel resection), diabetes (30% show signs of depletion). Age is a risk factor for magnesium depletion because intestinal absorption of magnesium declines with age. High doses of zinc in supplemental form can interfere with absorption of Magnesium.

CLINICAL MANIFESTATIONS OF DEPLETION

Primary magnesium deficiency is rare. Deficiency is usually secondary to another condition. Signs and symptoms of deficiency include weakness, heart irregularities, muscle cramps/twitches, insomnia, mental confusion, fatigue, irritability. Magnesium deficiency can impede Vitamin D and calcium absorption; increasing risk for bone mineral density disorders. Magnesium depletion is commonly associated with other disease states including both type 1 and type 2 diabetes, hypertension, endothelial dysfunction, asthma, and migraine headaches.

FOOD SOURCES

Magnesium is part of chlorophyll so leafy greens are rich in magnesium. Best food sources include: oats, brown rice, spinach, swiss chard, almonds, cashews, hazelnuts, potatoes, bananas, milk, raisins, halibut, avocado, black strap molasses, and chocolate.

SUPPLEMENT CONSIDERATION

The UL for magnesium is 350 milligrams from supplements or medicines because it may cause diarrhea. Severe toxicity may cause confusion, loss of kidney function, difficulty breathing and cardiac arrest; individuals with kidney disease are at higher risk for magnesium toxicity. The use of supraphysiological doses of magnesium can be used therapeutically. Supplemental magnesium is available in several different salts/chelations including: magnesium oxide, magnesium glycinate, magnesium chloride, magnesium citrate, and magnesium threonate. These compounds have different absorption, bioavailability and therapeutic values. Magnesium oxide and magnesium citrate are typically recommended for their ability to draw water into the gastrointestinal tract and have a laxative effect to produce a bowel movement. Also, it can help alleviate acid-reflux. Magnesium citrate has better bioavailability and is typically preferred over magnesium oxide. Citrates have also been shown to bind oxalates and may be the best form for those following a low oxalate diet. Magnesium glycinate has good bioavailability and is recommended to help increase magnesium levels without the bowel side effects. Magnesium malate is a form of magnesium that has been studied for its positive effects on depression, chronic fatigue, diabetes, and cardiovascular disease. Magnesium Threonate has recently been studied to cross the blood-brain barrier and improve memory and brain function, and potentially relieve headaches and migraines. Many studies have shown that supplemental Mg at doses around 400 mg/day reduces blood pressure in mildly hypertensive patients and pregnant women with preeclampsia.

SAMPLE

Nutrient Baseline - Summary

MINERALS

Test Name		Current	Previous	Result	Reference
Calcium (Ca)	Serum	8.5		6.5 / 8.8 / 10.6	8.9-10.6 (mg/dL)

PHYSIOLOGICAL FUNCTION

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.

HOW IT GETS DEPLETED

Calcium stores in the blood are not depleted metabolically, however, calcium stores elsewhere in the body may become depleted, conditionally, due to increased demand. Low dietary intake of calcium during times of growth or stress may result in low stores of calcium. Evaluate vitamin D and magnesium levels alongside calcium status. Iron supplementation may interfere with calcium absorption, and it is recommended to take iron supplements at least 2 hours apart from a meal containing calcium-rich foods.

CLINICAL MANIFESTATIONS OF DEPLETION

A deficiency of calcium causes osteoporosis. Some research connects low calcium intake to increased risks of high blood pressure, colon cancer, and preeclampsia (high blood pressure and excess protein in the urine of a woman more than 20 weeks pregnant).

FOOD SOURCES

Good sources of calcium are: dairy foods, salmon, turnip greens, Chinese cabbage, kale, bok choy, and broccoli. Sardines and other canned fish with bones are additional sources. Some foods such as orange juice and bread are fortified with calcium. Chinese cabbage, kale, and turnip greens contain absorbable calcium. Spinach and some other vegetables contain calcium that is poorly absorbed.

SUPPLEMENT CONSIDERATION

The AI for adults aged 19 to 50 is 1000 mg/day. Because calcium is so critical to preventing bone disease later in life, the AI is higher for adolescents. The AI for males and females aged nine to 18 is 1300 mg/day. For those aged 51 and older, the AI is 1200mg/day. The UL for calcium is 2,500 milligrams. Excess calcium may cause mineral imbalances because it interferes with the absorption of iron, magnesium, zinc, and other minerals. Forms of calcium supplementation available include calcium carbonate, calcium citrate, calcium citrate malate, calcium gluconate, and calcium lactate. Calcium citrate is the preferred form of calcium for individuals with hypo- or achlorhydria (low or insufficient stomach acid). In order to maximize absorption of calcium supplements, limit doses to no more than 500mg/dose. Supplementation of calcium should be accompanied by concurrent adequate vitamin D supplementation due to insufficient vitamin D levels impairing cellular calcium absorption, which can lead to atopic calcium deposits in epidermal tissue. Iron supplementation may interfere with calcium absorption, and it is recommended to take iron supplements at least 2 hours apart from a meal containing calcium-rich foods.

SAMPLE

Nutrient Baseline - Summary

MINERALS

Test Name		Current	Previous	Result	Reference
Chromium (Cr)	Serum	0.08		0 0.09 0.7	0.1-0.7 (ng/mL)

PHYSIOLOGICAL FUNCTION

Chromium is an essential nutrient used in trace amounts in humans that acts as a cofactor for chromodulin, a peptide that enhances the effect of insulin on target tissues, which aids in regulation of blood sugar and lipid metabolism.

HOW IT GETS DEPLETED

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.

CLINICAL MANIFESTATIONS OF DEPLETION

Chromium deficiency can contribute to the development of diabetes and metabolic syndrome. Even mild deficiencies of chromium can produce problems in blood sugar metabolism, and contribute to other symptoms such as anxiety or fatigue.

FOOD SOURCES

Food sources of chromium include: brewer's yeast, especially beer, broccoli, grape juice, meat and whole-grain products. Some fruits, vegetables, and spices provide chromium. Romaine lettuce, raw onions and ripe tomatoes are all good sources.

SUPPLEMENT CONSIDERATION

The AI for chromium is 35 µg/day for men and 25 µg/day for women. Increased needs may be present during pregnancy and lactation. Supplemental chromium is generally not needed as dietary consumption easily meets physiological needs. Supplementation is poorly studied and insufficient evidence exists to provide recommendations, but chromium picolinate is a form commonly used in treatment of insulin resistance and diabetes.

SAMPLE

Nutrient Baseline - Summary

MINERALS

Test Name		Current	Previous	Result	Reference
Selenium (Se)	Serum	104.0		0 109 218	109.8-218.4 (ng/mL)

PHYSIOLOGICAL FUNCTION

Selenium is an essential trace element required for immune function and for the synthesis of thyroid hormones, 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 to regenerate vitamin C and vitamin E from their oxidized forms, supporting antioxidant action of these vitamins.

HOW IT GETS DEPLETED

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.

CLINICAL MANIFESTATIONS OF DEPLETION

Selenium deficiency is very rare in developed countries. Low selenium intake may decrease an individual's ability to fight viral infections. Some research also links low intakes to some cancers. Toxicity causes brittle hair and nails and is most likely to occur with supplements. Selenium deficiency may not always produce overt symptoms of disease, but may manifest as increased oxidative stress in deficient individuals, due to decreased action of glutathione peroxidase, decreased antioxidant regeneration, decreased conversion of thyroid hormones, and reduced methionine metabolism. Severe selenium deficiency can result in Keshan disease, in which the heart becomes enlarged alongside cardiac insufficiency. Selenium supplementation prevents further progression of the condition, but does not reverse damage that has already occurred.

FOOD SOURCES

Food sources of selenium include: Brazil nuts, tuna, cod, turkey, chicken breast, beef roast, sunflower seeds, and ground beef. Organ meats, seafood, other meats, and whole grains are additional sources. Depending upon the soil in which they are grown, Brazil nuts are one of the richest sources of selenium.

SUPPLEMENT CONSIDERATION

The RDA for selenium is 55 µg/day. The UL for selenium is 400 µg/day, including food sources. Protein-based food sources of selenium appear to be the most effective at increasing circulating levels of glutathione peroxidase. Selenium supplementation in individuals with autoimmune thyroiditis has been shown to reduce circulating autoantibody levels. Selenium supplementation has been found to reduce viral load progression in individuals with HIV. Selenium supplementation in persons with sepsis and septic shock has been shown to reduce mortality. Selenium comes in the following supplemental forms: sodium, selenite, sodium selenate, selenomethionine. Selenomethionine has been shown to increase blood levels of selenium more effectively than the inorganic forms of selenium (selenite and selenate). Sodium selenate is absorbed to a lesser extent than sodium selenite, but sodium selenate is retained in greater amounts. Selenium supplements are not recommended for individuals with or at risk for diabetes mellitus.

SAMPLE

Nutrient Baseline - Summary

MINERALS

Test Name		Current	Previous	Result	Reference
Iodine (I)	Serum	40.0		0 — 42.6 — 91.8	42.7-91.8 (ng/mL)

PHYSIOLOGICAL FUNCTION

Iodine plays vital role in synthesis of thyroid hormones, which regulate basal metabolic rate, thermogenesis, growth, and neurocognitive development. Iodine supports normal fetal brain development, energy metabolism, cardiovascular function, and skin and hair integrity by ensuring adequate production of thyroxine (T4) and triiodothyronine (T3).

HOW IT GETS DEPLETED

Deficiency occurs from chronically low dietary intake (iodine-poor soil/food, non-iodized salt, restrictive diets), increased requirements during pregnancy and lactation, malabsorption, or exposure to goitrogens that inhibit thyroidal iodine uptake.

CLINICAL MANIFESTATIONS OF DEPLETION

Deficiency causes fatigue, cold intolerance, weight gain, constipation, bradycardia, dry coarse skin, hair loss, and menstrual irregularities, which are the clinical manifestations of goiter and hypothyroidism.

FOOD SOURCES

Prunes, Lima beans, Nori, Kelp, Kombu, Wakame, Cod, Tuna, Scallops, Shrimp, Oysters, Crab, Beef liver, Chicken, Eggs, Milk, Cheese, Yogurt

SAMPLE

Nutrient Baseline - Summary

MINERALS

Test Name		Current	Previous	Result	Reference
Zinc (Zn)	Serum	0.4		0 / 0.4 / 1	0.5-1.0 (mcg/mL)

PHYSIOLOGICAL FUNCTION

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, influences cell signaling, hormone release, and nerve signaling.

HOW IT GETS DEPLETED

Insufficient dietary intake, especially in populations that rely heavily on cereal grains for caloric intake, due to high levels of phytic acid impairing uptake of zinc. 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. Supplementation of elemental iron may decrease absorption of zinc. For this reason, pregnant women and individuals with anemia that are supplementing iron may need to take supplemental zinc, separate from iron supplement.

CLINICAL MANIFESTATIONS OF DEPLETION

Zinc deficiency causes delayed growth and sexual development, decreased immune function, altered sense of taste, hair loss, pregnancy complications, and gastrointestinal distress. Loss of zinc from cell membranes impairs their function and increases the susceptibility of the membrane to oxidative damage. Loss of zinc through malabsorption increases susceptibility to infections through depressed immune function. Increased urination in individuals with diabetes mellitus may lead to marginal zinc deficiency. Individuals that may have higher risk of zinc deficiency include: pregnant and lactating women; patients receiving total parenteral nutrition (TPN); malnourished individuals; individuals with eating disorders such as anorexia nervosa; individuals with impaired intestinal absorption and/or persistent diarrhea such as celiac disease, Crohn's disease, and ulcerative colitis; alcoholics and individuals with liver disease; individuals over 65 years of age; strict vegetarians.

FOOD SOURCES

Oysters, beef and clams are rich sources of absorbable zinc. Whole grains also contain zinc, but it is less available for absorption due to high phytic acid content of grains.

SUPPLEMENT CONSIDERATION

The RDA for zinc is 8mg/day for women and 11mg/day for men. The UL for zinc is 40mg/day. Long-term supplementation of zinc at levels of 60mg/day or greater interfere with absorption of copper. Zinc lozenges are commonly used to shorten the duration of the common cold. Zinc comes in the forms of: zinc acetate, zinc gluconate, zinc picolinate, zinc sulfate, zinc carnosine. Zinc carnosine has been used in combination therapies to aid in supporting the health of the epithelial linings of both the stomach and the intestines during times of physiological stress such as stomach ulcers and impaired intestinal barrier function. Zinc can be toxic when consumed in large doses, or when taken for prolonged periods of time in the absence of a zinc deficiency. Single doses of 200-450mg of zinc may induce vomiting and gastrointestinal distress.

SAMPLE

Nutrient Baseline - Summary

MINERALS

Test Name		Current	Previous	Result	Reference
Copper (Cu)	Serum	2.0		0 0.5 1.8	0.6-1.8 (mcg/mL)

PHYSIOLOGICAL FUNCTION

Copper assists with the transport of iron, supports energy production within cells, supports methylation and gene transcription that affects cellular detoxification mechanisms, 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 the cells of hair, skin, and nails. Most serum copper is found in ceruloplasmin and elevated levels may be an indicator of increased inflammation and oxidative stress, rather than specifically excess copper in the blood.

CLINICAL MANIFESTATIONS OF EXCESS / RISK FOR TOXICITY

Excess copper can cause nausea, liver damage, abdominal pain, and oxidative stress. Chronic toxicity may lead to neurological symptoms.

FOOD SOURCES

Rich sources of copper include liver, shellfish, cashews, hazelnuts, almonds, peanut butter, lentils, mushrooms, and sunflower seeds.

SUPPLEMENT CONSIDERATION

The RDA for copper in adults is 900 µg/day. The UL for copper is 10 mg/day, which has been shown not to produce liver damage in healthy individuals. Copper supplementation of 2 mg/day is usually sufficient to correct deficiencies of copper. Copper is most commonly available in the following supplemental forms: cupric oxide, copper gluconate, copper sulfate, copper amino acid chelates. 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.

Copper to Zinc Ratio (Cu:Zn)	Serum	5.0		0 0.8 2.6	0.9-2.6

PHYSIOLOGICAL FUNCTION

The Copper to Zinc Ratio in serum is an important indicator of the balance between copper and zinc levels in the body. This ratio is crucial for various biochemical processes, including immune function, antioxidant activity, and enzyme function. An optimal copper to zinc ratio is essential for maintaining overall health and preventing imbalances that can lead to health issues.

FOOD SOURCES

Copper is found in organ meats, shellfish, nuts, and seeds, while zinc is abundant in meat, dairy, nuts, and legumes. The ratio can be managed by adjusting dietary intake of copper- and zinc-rich foods to achieve a balance that supports health.

SUPPLEMENT CONSIDERATION

Supplementation should be considered based on individual needs and under medical supervision. Zinc supplements can be used to correct high copper levels, and copper supplements can address zinc excess. Regular monitoring of serum levels is crucial to ensure a healthy copper to zinc ratio.

SAMPLE

Nutrient Baseline - Summary

MINERALS

Test Name		Current	Previous	Result	Reference
Iron (Fe)	RBC	121.0		0 / 88.8 / 117	88.9-117.0 (mg/dL)

PHYSIOLOGICAL FUNCTION

Iron is required for the production of red blood cells (a process known as hematopoiesis), but it's also part of hemoglobin (that is the pigment of the red blood cells) binding to the oxygen and thus facilitating its 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, from 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 the production of new cells, amino acids, hormones and neurotransmitters) also depends on iron, this aspect becomes crucial during the recovery process from illnesses or following 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 supplies.

CLINICAL MANIFESTATIONS OF EXCESS / RISK FOR TOXICITY

Excess iron can cause iron overload, leading to fatigue, liver damage, and joint pain. Chronic high levels increase oxidative stress and organ toxicity.

FOOD SOURCES

Symptoms of iron deficiency can occur even before the condition has progressed to iron deficiency anemia. Symptoms of iron deficiency are not unique to iron deficiency. Iron is needed for many enzymes to function normally, so a wide range of symptoms may eventually emerge, either as the secondary result of the anemia, or as other primary results of iron deficiency. Symptoms of iron deficiency include: fatigue, dizziness, pallor, hair loss, twitches, irritability, weakness, pica, brittle or grooved nails.

SUPPLEMENT CONSIDERATION

Frequently used forms of iron in supplements include ferrous and ferric iron salts, such as ferrous sulfate, ferrous gluconate, ferric citrate, and ferric sulfate. Because of its higher solubility, ferrous iron in dietary supplements is more bioavailable than ferric iron. High doses of supplemental iron (45 mg/day or more) may cause gastrointestinal side effects, such as nausea and constipation. Other forms of supplemental iron, such as heme iron polypeptides, carbonyl iron, iron amino-acid chelates, and polysaccharide-iron complexes, might have fewer gastrointestinal side effects than ferrous or ferric salts. Many medicinal herbs can offer iron boosting properties to those who suffer from iron deficiency. These medicinal properties can easily be assimilated into the bloodstream as a hot water infusion (tea). Iron enhancing herbs include yellow dock, red raspberry leaf, gentian, yellow root, turmeric, mullein, nettle, parsley, ginseng, watercress, and dandelion.

SAMPLE

AMINO ACIDS

Test Name		Current	Previous	Result	Reference
L-Arginine	Serum	74.2		0 / 81.5 / 249	81.6-249.0 (nmol/mL)

PHYSIOLOGICAL FUNCTION

L-Arginine is a conditionally essential amino acid found in the diet. It is a dietary supplement used mostly by athletes because it is the amino acid that directly produces nitric oxide via the nitric oxide synthase enzymes. Arginine helps heal injuries, aids kidneys in removing waste, and boosts immune system function.

HOW IT GETS DEPLETED

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 the enzyme that degrades L-arginine (known as arginase) 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.

CLINICAL MANIFESTATIONS OF DEPLETION

Arginine is one of the three substrates to form creatine which is a vital nutrient (deficiency induces mental retardation) and is also used to form agmatine, a signalling molecule in the body. Arginine is an intermediate in both the urea cyle (with L-ornithine, L-citrulline, and arginosuccinate) and the nitric oxide cycle (with ornithine and arginosuccinate), and vicariously through ornithine it produces polyamine structures which can regulate cellular function. In some individuals with viral infections such as shingles, arginine supplementation may exacerbate symptoms and consultation with a healthcare provider is recommended.

FOOD SOURCES

Dietary arginine accounts for 40-60% of serum arginine. Food sources include: turkey, pork, chicken, pumpkin seeds, soybeans, peanuts, spirulina, dairy, chickpeas, lentils.

SUPPLEMENT CONSIDERATION

To maintain elevated arginine levels throughout the day, arginine can be taken up to three times a day, with a combined dose total of 15-18g. Note: L-citrulline supplementation is more effective at maintaining elevated arginine levels for long periods of time. Taking more than 10g of arginine at once can result in gastrointestinal distress and diarrhea.

L-Citrulline	Serum	16.5		0 / 18.6 / 47.5	18.7-47.5 (nmol/mL)

PHYSIOLOGICAL FUNCTION

Citrulline is an amino acid that is not one of the essential amino acids nor a common dietary amino acid (named after its only good natural source, watermelons), but is interconverted in the body and serves roles similar to L-arginine and L-ornithine.

HOW IT GETS DEPLETED

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.

CLINICAL MANIFESTATIONS OF DEPLETION

The majority of L-citrulline either floats in the blood passively or gets transported to the kidneys for conversion into arginine.

FOOD SOURCES

Food sources of L-citrulline include watermelon, muskmelons, bitter melons, squashes, gourds, cucumbers, and pumpkins.

SUPPLEMENT CONSIDERATION

There is mixed evidence as to whether citrulline supplementation can help with muscle protein synthesis.

SAMPLE

Nutrient Baseline - Summary

AMINO ACIDS

Test Name		Current	Previous	Result	Reference
L-Glutamine	Serum	382.0		0 393 699	393.5-699.3 (nmol/mL)

PHYSIOLOGICAL FUNCTION

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.

HOW IT GETS DEPLETED

Glutamine is known to be depleted in certain types of physiological stress such as burns, major trauma, and cancers that consume available intra-cellular glutamine stores more rapidly than skeletal muscle can generate it, leading to increased muscle wasting. During physical activity, serum glutamine is consumed for longer endurance events (2+ hours); some evidence exists that chronic endurance exercise reduced glutamine levels to affect immune cell function and proliferation.

CLINICAL MANIFESTATIONS OF DEPLETION

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. Some studies suggest an increase in intestinal permeability when intestinal epithelial cells lack sufficient glutamine, as well as insufficient availability for leukocyte function.

FOOD SOURCES

Very good sources of glutamine include: whey, casein, milk, white rice, corn, and tofu. Good sources of glutamine include: meat and eggs.

SUPPLEMENT CONSIDERATION

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 – 50g/day. Higher doses (>10 g/day) are commonly used in the treatment of intestinal barrier permeability. Supplementation of glutamine has not been shown to enhance muscle growth in healthy individuals. Typically an increase in serum insulin results after consumption of glutamine due to increased conversion to glucose. This may impact individuals with insulin resistance. Glutamine supplementation is also potentially beneficial to improve mental focus and concentration, as well as curbing cravings for sugar and alcohol. In some individuals, glutamine is converted more efficiently to glutamate, which can lead to a neuro-excitatory state, increased anxiety, tension headaches/migraines, and even tachycardia. If any of these symptoms occur after consuming glutamine, discontinue supplementation and discuss with your provider.

SAMPLE

Nutrient Baseline - Summary

AMINO ACIDS

Test Name		Current	Previous	Result	Reference
Free Carnitine	Serum	10.8		0 / 11.5 / 43.4	11.6-43.4 (nmol/mL)

PHYSIOLOGICAL FUNCTION

Carnitine is an essential co-factor in the metabolism of lipids and the production of cellular energy. It promotes neuroprotection through antioxidant properties and modulation and promotion of synaptic neurotransmission. Carnitine can be rate-limiting in ketone body uptake by brain astrocytes, and also reduces oxidative stress.

HOW IT GETS DEPLETED

Carnitine deficiency is rare in individuals who consume an omnivorous diet. Iron and Vitamin C are required for endogenous carnitine synthesis, and endogenous synthesis can also be impaired by severe liver disorders. Deficiency may be found in vegetarians and vegans, and individuals with impaired absorption of nutrients, such as in inflammatory bowel disorders and celiac disease. There may be increased demand for carnitine during ketosis.

CLINICAL MANIFESTATIONS OF DEPLETION

Symptoms of carnitine depletion or deficiency include: male infertility, impaired muscle metabolism of energy, which can cause myopathy, hypoglycemia, muscle necrosis, myoglobinuria, lipid-storage myopathy, hypoglycemia, fatty liver, and hyperammonemia with muscle aches, fatigue, confusion, and cardiomyopathy.

FOOD SOURCES

Carnitine is found naturally occurring in high amounts in red meat, eggs, and dairy products.

SUPPLEMENT CONSIDERATION

There is currently no established RDA, AI, or UL for carnitine intake. Supplementation of carnitine has been used in treatment of chronic degenerative diseases of the brain and for slowing down the progression of Alzheimer's disease. Carnitine supplementation in athletes has not been shown to consistently produce performance benefits. Carnitine comes in supplement form as L-carnitine. Some research suggests that diets high in carnitine may have associations with cardiovascular inflammation due to the over-abundance of opportunistic microbes that convert dietary carnitine to trimethylamine oxidase (TMAO). Consider testing levels of these bacteria to assess if TMAO levels are due to intestinal dysbiosis.

No markers are outside the normal reference range

FATTY ACIDS

AA/EPA		Current	Previous	Result	Reference
AA/EPA Ratio	RBC	>127		0 / 2.4 / 10.9	2.5-10.9

SAMPLE

Nutrient Baseline - Summary

FATTY ACIDS

OMEGA-3		Current	Previous	Result	Reference
DHA (Docosahexaenoic Acid)	RBC	2.10		0 2.41 10.5	2.42-10.52 (%)

PHYSIOLOGICAL FUNCTION

Docosapentanoic acid (DPA) is a structurally similar omega-3 fatty acid to 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.

HOW IT GETS DEPLETED

Deficiency of DPA is typically due to low dietary intake of high DPA foods.

CLINICAL MANIFESTATIONS OF DEPLETION

Low levels of DPA are associated with increased risk of thrombosis and stroke death.

FOOD SOURCES

Good sources of DPA include: fish oil, fatty fish such as salmon, and grass-fed beef.

SUPPLEMENT CONSIDERATION

Adverse side effects observed with high dose omega-3 fatty acids from supplement form include gastrointestinal upset and loose stools.

SAMPLE

Nutrient Baseline - Summary

FATTY ACIDS

OMEGA-3		Current	Previous	Result	Reference
EPA (Eicosapentaenoic Acid)	RBC	0.12		0 0.14 2.26	0.15-2.26 (%)

PHYSIOLOGICAL FUNCTION

Eicosapentanoic 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 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 signaling of TNF-α in adipocytes. EPA also increases cerebral oxygenation. EPA appears to have some beneficial influence on regulating levels of leptin and increasing adiponectin. EPA may enhance adaptive immunity by stimulating B cell responsiveness.

HOW IT GETS DEPLETED

Lower dietary intake of omega-3 fatty acids is the primary reason for deficiency of EPA, or low levels of EPA. Certain genetic polymorphisms such as reduced activity of the FADS1 and FADS2 genes may lead to reduced conversion of ALA into EPA and DHA.

CLINICAL MANIFESTATIONS OF DEPLETION

EPA can be manufactured in the body from ALA, as well as retroconverted from DHA. However, relying solely on intake of ALA to provide adequate levels of EPA is not recommended due to poor or inefficient conversion from ALA to EPA. Lower levels of EPA or deficient intake of EPA have been linked to increased risk for cardiovascular disease, arrhythmia, blood clots, heart attacks, stroke, elevated triglyceride levels, increased growth of atherosclerotic plaque, reduced vascular endothelial function, skin cancer, and increased inflammation. Lower levels of EPA are also associated with lower brain mass in older adults.

FOOD SOURCES

Good sources of EPA include: fatty fish such as Pacific herring, salmon, oysters, tuna, and omega-3 enriched eggs. Food sources of ALA, the essential fatty acid EPA precursor include: flaxseeds and flaxseed oil, chia seeds, walnuts, and canola oil.

SUPPLEMENT CONSIDERATION

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). Persons suffering from ulcerative colitis have been shown to need fewer corticosteroids when supplementing with high dose omega-3 fatty acids. Adverse side effects observed with high dose 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.

SAMPLE

Nutrient Baseline - Summary

FATTY ACIDS

OMEGA-3		Current	Previous	Result	Reference
DPA (Docosapentaenoic Acid)	RBC	0.39		0 / 0.44 / 1.8	0.45-1.8 (%)

PHYSIOLOGICAL FUNCTION

Docosapentanoic acid (DPA) is a structurally similar omega-3 fatty acid to 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.

HOW IT GETS DEPLETED

Deficiency of DPA is typically due to low dietary intake of high DPA foods.

CLINICAL MANIFESTATIONS OF DEPLETION

Low levels of DPA are associated with increased risk of thrombosis and stroke death.

FOOD SOURCES

Good sources of DPA include: fish oil, fatty fish such as salmon, and grass-fed beef.

SUPPLEMENT CONSIDERATION

Adverse side effects observed with high dose omega-3 fatty acids from supplement form include gastrointestinal upset and loose stools.

Total Omega-3	RBC	2.90		0 / 3.24 / 13.9	3.25-13.99 (%)

PHYSIOLOGICAL FUNCTION

The Omega-3 Index measures the sum of EPA and DHA fatty acids in whole blood. This index is calculated to reflect the same fatty acids in red blood cell membranes. The index serves as an indicator for various health outcomes, including the risk of sudden cardiac death and nonfatal cardiovascular events. It can also assess the effectiveness of omega-3 therapy and has a positive impact on heart rate, blood pressure, triglyceride levels, inflammation, endothelial function, and cognitive function.

HOW IT GETS DEPLETED

The Omega-3 Index can be depleted due to a low intake of omega-3 rich foods, or a high intake of other types of fatty acids like saturated, mono-unsaturated, and omega-6 fatty acids. The index is calculated as a percentage, with the denominator being the sum of all fatty acids in the blood, making it sensitive to the overall fatty acid composition of the diet.

CLINICAL MANIFESTATIONS OF DEPLETION

Low levels of the Omega-3 Index are associated with an increased risk of cardiac death, as well as potential negative impacts on inflammation, blood pressure, and cognitive function.

FOOD SOURCES

To improve a low Omega-3 Index (<8.0%), consider increasing the intake of both plant-based (like flaxseeds and walnuts) and animal-based (like fatty fish) sources of EPA and DHA. Also, assess and potentially adjust the intake of other types of fatty acids, including saturated fats, mono-unsaturated fats, and omega-6 polyunsaturated fats.

SUPPLEMENT CONSIDERATION

While there's no officially established RDA for omega-3s, some health organizations suggest a minimum intake of 500 mg/day of EPA and DHA combined. ALA, another type of omega-3, has a low conversion rate to DHA, so direct DHA supplementation is often recommended. Adult doses generally start at 250 mg/day of DHA. For diabetic individuals and those with certain inflammatory conditions, higher doses may offer additional benefits. However, consult your healthcare provider for personalized advice, especially if you are pregnant, lactating, or have a clotting disorder. Note that high-dose supplementation may cause gastrointestinal upset and should be taken cautiously.

SAMPLE

Nutrient Baseline - Summary

FATTY ACIDS

OMEGA-3		Current	Previous	Result	Reference
Omega 3 Index	RBC	3.10		0 2.65 7.99 12.6	8.0-12.65 (%)

PHYSIOLOGICAL FUNCTION

Omega-3 Index is the sum of EPA % and DHA % as measured in whole blood, and derived by validated calculations to yield the equivalent sum of EPA % and DHA % in red blood cell membranes. Please note this value is a percentage, with the denominator being the sum of all Fatty Acids measured in the blood and thus the index can vary based on fatty acid composition of the diet. The index can be used as an indicator of risk for sudden cardiac death and nonfatal cardiovascular events and as a therapeutic target. It can also be used to assess adherence to omega-3 therapy and/or success or failure of such therapy. Optimal omega-3 index positively impacts heart rate, blood pressure, triglyceride levels, myocardial efficiency, inflammatory responses, and endothelial function while also improving cognitive function.

HOW IT GETS DEPLETED

The Omega-3 Index is a validated biomarker of tissue membrane omega-3 (n-3) polyunsaturated fatty acid (PUFA) status. The ratio is expressed as a percentage where the denominator is the sum of all fatty acids measured in the blood. Thus, a decrease in the ratio can be caused by a low intake of omega-3 fatty acids and incorporation of those fatty acids into cell membranes; or due to a proportionally high intake of other dietary fatty acids (saturated fatty acids, mono-unsaturated fatty acids and omega-6's polyunsaturated fatty acids).

CLINICAL MANIFESTATIONS OF DEPLETION

Low levels of omega-3 index are associated with increased risk for cardiac death.

FOOD SOURCES

If omega-3 index is <8.0% it is advised to increase dietary sources of omega-3's (EPA and DHA) from both plant and animal sources. Because the omega-3 index is a relative ratio of omega-3 compared to all other fatty acids in the blood, it is also important to evaluate intake of all other dietary fatty acids (saturated fatty acids, mono-unsaturated fatty acids and omega-6's polyunsaturated fatty acids).

SUPPLEMENT CONSIDERATION

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 DHA is so low, supplementing DHA is generally recommended to meet therapeutic doses. The recommended minimum level of DHA supplementation in adults is 250 mg per day. Pregnant and lactating women are recommended to consume at least 200 mg DHA per day. Diabetic individuals may benefit from supplementing DHA (along with EPA) due to its triglyceride-lowering effects. High dose supplementation of omega-3 fatty acids (including DHA) has been shown to reduce the need for non-steroidal anti-inflammatory drugs (NSAIDS). Persons suffering from ulcerative colitis have been shown to need fewer corticosteroids when supplementing with high dose omega-3 fatty acids. Adverse side effects observed with high dose 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.

SAMPLE

Nutrient Baseline - Summary

FATTY ACIDS

OMEGA-6		Current	Previous	Result	Reference
Linoleic Acid (LA)	RBC	11.40		0 / 3.21 / 10.4	3.22-10.49 (%)

PHYSIOLOGICAL FUNCTION

Linoleic Acid 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, linoleic acid must be consumed in the diet in order 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.

FOOD SOURCES

Linoleic acid is found in high concentrations in vegetable oils. Corn, soybean, sunflower, safflower, canola, and peanut oil provide the greatest amount of dietary linoleic acid. Other dietary sources include avocados, nuts, and seeds.

SUPPLEMENT CONSIDERATION

There is typically no need for supplementation of linoleic acid or any omega-6 fatty acid, as these fats are plentiful in the western diet. Decreasing intake of refined vegetable oil (corn, soy, peanut, canola, safflower oil) and replacing with an alternative choice such as olive oil, coconut oil, avocado oil, or fat from animal sources is the best way to balance LA levels.

Total Omega-6	RBC	36.50		0 / 11.0 / 34.9	11.03-34.96 (%)

PHYSIOLOGICAL FUNCTION

The Total Omega-6 Index measures the sum of various omega-6 fatty acids as found in whole blood, expressed as a percentage of total fatty acids. This index serves as an indicator of inflammatory responses but also has other functions like maintaining skin health and aiding brain function. Additionally, the index can be used to assess the balance between omega-3 and omega-6 fatty acids in the body, which is crucial for various physiological functions including cell membrane integrity and inflammatory pathways.

FOOD SOURCES

If the Total Omega-6 Index is not within the desired range, consider adjusting the dietary sources of omega-6, which are primarily found in vegetable oils, nuts, and seeds. It is also essential to balance the intake of other types of fatty acids, including omega-3s, saturated fats, and mono-unsaturated fats.

SUPPLEMENT CONSIDERATION

There is no established RDA for omega-6 fatty acids. Supplemental forms are available but should be used cautiously, as excessive omega-6 intake can promote inflammation. Balance with omega-3 is often considered important in reducing inflammation. If you're considering supplements, consult your healthcare provider, especially if you have inflammatory diseases or other medical conditions. Note that while gamma-linolenic acid (GLA), a type of omega-6, has anti-inflammatory properties, excessive supplementation can have pro-inflammatory effects.

SAMPLE

Risk and Limitations

Test results reflect biological and analytical findings at the time of specimen collection and may vary between individuals. Reference ranges for most of laboratory-developed tests (LDT) were established using a healthy adult population and may not be representative of other specific populations (e.g. pediatric, pregnant, individuals with chronic conditions or from all ethnic backgrounds). They do not provide absolute levels at which the symptoms may occur and hence clinical correlation by the provider is recommended.

Results may be affected by pre-analytical variables related to specimen collection, handling, transport, storage, and inherent biological variability. Specimens including urine, saliva, stool, and blood-based samples (serum, plasma, EDTA whole blood, TES, and dried blood spots) may be impacted by improper collection technique, contamination, insufficient sample volume, delayed shipment or processing, temperature excursions, or improper storage conditions. Additional factors such as hemolysis; anticoagulant effects; clotting, centrifugation, or mixing parameters; incomplete mixing with transport media; and variability in dried blood spot application or saturation may further affect analyte stability or result accuracy. Specimen-specific factors, including urine dilution or concentration, variability in saliva composition or flow rate, and intermittent microbial shedding in stool, may also contribute to result variability. These factors may impact result accuracy and, in some cases, lead to a Test Not Performed (TNP). When clinically appropriate, repeat testing may be recommended; however, repeat testing may still fail to produce a reportable result if the underlying limitations persist.

All laboratory testing methodologies are subject to inherent analytical limitations related to instrument performance, assay design, methodological variability, and the specifications of FDA-approved and laboratory-developed analytes included in a test panel. As with all clinical laboratory testing, there is a small possibility of incorrect results due to technical errors, sample misidentification, contamination, rare genetic variants, or software-related issues.

Genetic testing is helpful in analyzing risks to various diseases. However, it is important to note that genetic risk determinants are neither necessary nor sufficient for the development of disease. Environmental and lifestyle risk factors could also affect the risk of disease development. Genetic risk does not indicate how common a health condition or variant is within the population; a risk-associated variant may be common or uncommon. Interpretation of genetic results should consider individual health context, as population-based reference frameworks may not fully represent all age groups, ethnic backgrounds, or health profiles. Genetic testing evaluates only the genotypes indicated and does not assess other genetic abnormalities found elsewhere in the genome. Different laboratories may test different variants when evaluating genetic risk for a given condition; therefore, genetic risk results may not be directly comparable between laboratories.

Some individuals may experience anxiety related to their genetic test results. Vibrant encourages any concerned individual to consult with a qualified healthcare professional prior to sample collection for a genetic test. Users of the test are encouraged to discuss their test results with a genetic counselor, board-certified clinical molecular geneticist, or equivalent health care professional. In some cases, the identification of risk-associated genetic variants may prompt discussion with a healthcare provider about additional testing or follow-up.

The reported analytes, SNPs, and associated informational content are informed by scientific knowledge at the time of reporting, including peer-reviewed scientific publications, publicly available research, and guidance from recognized scientific and public health organizations. Interpretive content may be updated as scientific knowledge continues to evolve. The informational content included in this report is derived from publicly available scientific literature and is provided for educational and informational purposes only. This content does not replace medical advice from a qualified healthcare professional. Any wellness, nutritional, or dietary recommendations, diagnoses of medical conditions, or treatment decisions based on these results are made at the discretion and responsibility of the ordering healthcare professional.

Vibrant does not diagnose, treat, or cure medical conditions and does not replace the care of a licensed medical practitioner or counselor, nor does Vibrant recommend self-diagnosis or self-medication. Depending on the nature of testing, individuals who receive moderate- or high-risk results may be advised to pursue confirmatory testing and appropriate medical follow-up. Vibrant assumes no liability for any loss, injury, or damages arising from the procurement, compilation, interpretation, delivery, or reporting of information contained in this report, nor from any decisions made or actions taken based on these results.

The supplement recommendations and dosage guidelines provided are intended for general informational purposes only and should not replace professional medical advice; final dosage decisions must be made in consultation with your healthcare provider. Vibrant disclaims any liability for adverse effects, outcomes, or consequences arising from the use of these suggestions.