

**FUNCTIONAL  
BLOOD CHEMISTRY  
ANALYSIS**

**By  
Nikolas R. Hedberg, D.C., D.A.B.C.I.**

## **Functional Blood Chemistry Analysis**

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# ABOUT THE AUTHOR

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He is the founder of the Immune Restoration Center in Asheville, North Carolina where he helps people worldwide overcome chronic illnesses using functional medicine approaches to health. He is the author of the book *The Thyroid Alternative*, a comprehensive guide to understanding the causes of thyroid disorders and how to address them.

Dr. Hedberg is a member of the ACA's Council on Diagnosis & Internal Disorders, the North Carolina Integrative Medical Society, the North Carolina Chiropractic Association and the American Chiropractic Association.

Dr. Hedberg has a special interest in the connection between chronic stealth infections and chronic illnesses. This passion led him to create an online educational program for practitioners in the diagnosis and management of infection-related illness at [www.infectionconnection.net](http://www.infectionconnection.net).

Dr. Hedberg lectures at integrative medicine conferences and he is the author of many peer-reviewed articles on functional medicine.



# DEDICATION

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This book is dedicated to Dr. Bill Beakey for his contribution to blood chemistry analysis, genuine nature and generosity. Dr. Beakey should also be recognized for his ethical business practices and exceptional professionalism. Dr. Beakey is a rare seed in the functional medicine world today.



# ACKNOWLEDGMENTS

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I would like to recognize the following individuals for their contribution to functional blood chemistry analysis:

Dr. Bill Beakey

Dr. Dicken Weatherby

Dr. Jeffrey Moss

Dr. David Brady

These individuals should be recognized for their deep understand of biochemistry and physiology as it relates to blood chemistry analysis. Thank you for helping me see the bigger picture in this often misunderstood aspect of functional medicine.

I would also like to recognize Dr. Bill Beakey for his thorough review and editing of the material.



# FOREWORD

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When I first received a preliminary copy of *Functional Blood Chemistry Analysis*, I had a reaction that many of you might have had when first learning about it. There are so many books on blood chemistry interpretation available now that range from standard medical fare to those geared specifically for nutritionally oriented practitioners looking for “optimal” ranges, why do I need another one? However, upon reading some sections of the book, I realized that Dr. Hedberg was going in a very different direction compared to all the other blood chemistry books in my library – a direction that I immediately realized was going to make this book an important and often used reference.

As we all know, the practice of clinical nutrition has dramatically changed since I first entered the field in 1979. At that time, our focus was patients with acute ailments or single, readily identifiable “diseases.” Because of this, the blood chemistry textbooks of the time that we tended to use were standard, medical blood chemistry texts or similar nutritionally oriented texts, often written by authors affiliated with supplement companies, which, like the standard medical texts, only revolved around classic disease entities. Unfortunately, these types of books now often fall short in terms of serving our needs. Why? Our practices now tend to be populated with aging patients experiencing a whole range of signs and symptoms that are often difficult to categorize into single and even multiple classic disease categories. Because of this, many of us, me included, have, for the last several years, taken a functional medicine approach that focuses on metabolic imbalances such as chronic inflammation and insulin resistance.

This evolution towards a functional medicine approach with chronically ill patients, as I suggested above, created a void in my reference library. Now, while I still need a standard blood chemistry reference book that contains information on classic disease entities, I also need a book that includes diagnostic information on functionally oriented metabolic imbalances. Why? A very large body of research is making it clear that, if we have a better understanding of key metabolic factors such as low grade, chronic metabolic acidosis, chronic inflammation, insulin resistance, loss of muscle mass (sarcopenia), and several others, our task of time and cost effectively addressing patient chief complaints will be ever so much easier.

*Functional Blood Chemistry Analysis* fills this void. As you will see, Dr. Hedberg has created the standard blood chemistry text that is needed for today's busy, nutritionally oriented practice – an easy to read, easy to use, well referenced, “just the facts” book that contains the standard disease oriented information that is nice to know plus the metabolically oriented, functional information that is essential to know if we are going to continue to address the needs of an aging patient population whose needs are becoming increasingly more varied and complicated.

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# BLOOD CHEMISTRY ANALYSIS: THE BIG PICTURE APPROACH

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The purpose of this book is to provide the functional medicine practitioner with an understanding of the physiology and biochemistry behind imbalances on a blood chemistry and clinical recommendations to address those imbalances. No other laboratory diagnostic tool can provide the physician with the amount of data about a patient's biochemical status.

It is my goal to help practitioners see the “bigger picture” when looking at a blood chemistry so they can adequately understand the results and implement the correct strategies to get the patient well as quickly as possible with the fewest number of interventions. It is also extremely important to understand the pathological possibilities when interpreting blood chemistries.

A blood chemistry should not be viewed as a report of multiple individual numbers in isolation but rather a picture of the patient's biochemistry. This approach will result in more rapid patient improvements when foundational aspects of metabolic dysfunction are addressed from the beginning.

The antiquated model of functional blood chemistry analysis involves looking at individual markers and prescribing supplements to address the imbalances. One example is prescribing liver supportive nutrients when liver enzymes come back elevated. That model lacks a deeper understanding of biochemistry and actually abandons a true functional medicine model which focuses on cause rather than treating symptoms. Supplementation can however be extremely beneficial when there is a true deficiency of a micronutrient for example. Significant clinical outcomes can be achieved when foundational aspects are evaluated and addressed.

## THE FOLLOWING FOUNDATIONAL ASPECTS SHOULD BE EVALUATED AND ADDRESSED IF FOUND TO BE OUT OF BALANCE:

- **Inflammation:** CRP, ESR, CBC, Fibrinogen, Homocysteine
- **Protein deficiency:** Dynamometer grip strength, 3-day diet diary, body composition, amino acids, plasma proteins, creatinine, BUN, uric acid

- **Metabolic acidosis:** 1st morning urinary pH, CO<sub>2</sub>, sodium, potassium, BUN, uric acid, plasma proteins
- **Identify insulin resistance:** Fasting glucose, A1c, insulin
- **Infection:** CBC, hs-CRP, ESR, urinalysis and infection-specific testing
- **Food sensitivities:** 3-day diet diary, IgG/IgA/IgE food sensitivity testing
- **Environmental toxin:** Environmental history, toxin-specific lab testing ie. DMPS urinary challenge
- **Stress:** History, Cortisol, DHEA, Zinc taste test
- **Gastrointestinal dysfunction:** History & physical exam, stool analysis
- **Abnormal body composition (increased adipose tissue & decreased lean body mass):**  
Bodyfat %
- **Sleep disorders**
- **Lack of exercise**
- **Current emotional state**
- **Social isolation**

## ADDRESS OVERT ENDOCRINE ISSUES:

Patients will not get well if there is a true endocrine imbalance. One example is hypothyroidism. Thyroid hormone must be restored to normal levels in order for the patient to get well and back into balance. Other examples are adrenal gland and sex hormone imbalances.

## IDENTIFY AND CORRECT ANEMIAS AND NUTRIENT DEFICIENCIES:

Examples include iron-deficiency anemia, vitamin D deficiency, Zinc, vitamin B12, B6 and selenium.

This approach will correct many biochemical imbalances and begin to restore the patient's health. A detailed explanation of how to correct each imbalance goes beyond the scope of this text and assumes a basic knowledge of functional medicine approaches to health.

“Functional” or “optimal” reference ranges are not included in this text. Standard laboratory ranges are based on sick populations and will vary from lab to lab and city to city. This indicates that standard laboratory ranges may not be based on good health and are mainly used to rule out pathology. However, “functional” and “optimal” ranges also lack hard science and are based on anecdotal and observational

information. One advantage of using these ranges may be due to the fact that they are not as broad as standard ranges thus narrowing the window. It is therefore recommended to follow standard reference ranges in regards to pathology and “functional” or “optimal” ranges as a general guide but not as an absolute parameter. It is not recommended to tightly follow any ranges without regard for how the patient is feeling. If the patient reports no health issues and is symptom-free, it may be against the patient’s best interest to begin extensive dietary and supplement regimens based on some laboratory markers that are out of range. Blood chemistry analysis software is available which can be extremely useful for patient education and to help the patient better understand their results. The more educated the patient is about their results, the more likely they are to comply with treatment recommendations. A blood chemistry analysis software recommendation is made in the resources section of this text.

There is no substitute for a thorough patient history which can provide the diagnosis the vast majority of the time. Practitioners should enhance their history-taking skills and not rely mainly on blood chemistry reports. “Treat the patient, not the labs” should be at the forefront of the practitioners level of thinking which removes any hint of an agenda or “cookie-cutter” approach. Tests should only be ordered if there is a strong suspicion that the results will achieve a diagnosis or actually change the way the patient will be treated.

As you become more skilled in seeing the “bigger picture” of each blood chemistry combined with exceptional clinical diagnostic skills, your patients will get better much quicker with fewer interventions.

### **A STANDARD BLOOD CHEMISTRY WHICH GIVES THE CLINICIAN A GOOD OVERALL PICTURE INCLUDES:**

- CBC w/differential w/platelets
- Comprehensive metabolic panel
- Lipid panel
- Magnesium
- Phosphorous
- Uric Acid
- LDH
- GGT
- TSH
- Thyroxine (T4)
- T3 uptake

- Iron
- Iron saturation
- Total Iron Binding Capacity (TIBC)
- Ferritin
- Erythrocyte Sedimentation Rate
- Highly Sensitive C-reactive protein (Cardiac)
- Hemoglobin A1C

Additional helpful markers include homocysteine, fibrinogen, vitamin D, and a more detailed thyroid analysis including total T3, Free T4 & T3, reverse T3. A urinalysis with microscopy is recommended as part of the standard profile to rule out a variety of pathological and metabolic imbalances.

These markers will help the clinician identify the vast majority of biochemical imbalances as well as signs of pathology. Additional markers can be added if the practitioner deems them necessary or if more information is needed.

Each marker will now be covered in detail including reference ranges, biochemistry, physiology, signs and symptoms, related tests, clinical indications and nutraceuticals.

# GLUCOSE

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**Reference Range:** 70-99 mg/dL

Glucose is an extremely important fuel for metabolism under the control of multiple mechanisms. Glucose enters the body via ingestion of carbohydrates and is absorbed through the small intestine under the influence of thyroid hormone, cortisol and B-complex. Once absorbed into the bloodstream, it travels through the liver and then into muscle tissue and cells throughout the body. Approximately 300-500 grams of glucose are stored in the liver, muscle and cells in the form of glycogen. Glucose can also be converted to triglyceride and stored as adipose tissue. Fasting blood glucose levels are maintained at approximately 70-120 mg depending on the health of the individual.

The liver is the main site of glucose production via gluconeogenesis from fat and protein by way of the Krebs's cycle (Citric Acid Cycle). Under the influence of epinephrine, glycogen is converted into glucose. Glucose is metabolized into water and carbon dioxide as it supplies energy for metabolism. Proper utilization of glucose is dependent on vitamins B1 and B5. The vast majority of blood glucose is reabsorbed in the kidney but when levels reach 170 mg, renal threshold is exceeded and glucose will appear in the urine. After a carbohydrate meal, blood glucose levels rise and the beta cells of the pancreas release insulin which drives glucose across the cell membrane for metabolism in the cytosol and mitochondria of the cell.

The liver is the main organ of fasting glucose regulation. When blood glucose levels drop, the liver increases blood glucose levels via glucagon production by the pancreas. When blood glucose levels rise, islet cells are inhibited from releasing glucose from the liver. This mechanism is influenced by thyroid hormone, cortisol, insulin and epinephrine.

Insulin facilitates the uptake of glucose across cell membranes from the blood. Insulin also inhibits gluconeogenesis from amino acids and it influences the deposition of glycogen in the liver.

Thyroid hormone increases intestinal and renal absorption of glucose. Thyroid hormone can deplete muscle glycogen stores and increase glycogenolysis in the liver. Since thyroid hormone increases metabolic rate, it increases the peripheral utilization of glucose.

Cortisol mobilizes amino acids from body tissues such as skeletal muscle, gut lining and skin for gluconeogenesis in the liver. Excess cortisol levels also inhibit peripheral glucose utilization leading to insulin resistance.

Epinephrine increases glycogenolysis in the liver and skeletal muscle.

Glucagon is a hormone made by the alpha cells in the pancreas which stimulates glycogenolysis in the liver.

Growth hormone is made by the anterior pituitary and excessive levels result in insulin resistance due to inhibition of glucose phosphorylation in the cell.

A glucose tolerance can be ordered which requires a fasting glucose and then the patient drinks a glucose solution to spike blood sugar and glucose levels are tested ever two hours for up to 6 hours to monitor the body's response to a glucose surge.

### **SYMPTOMS OF ELEVATED GLUCOSE:**

- Fatigue
- Increased thirst
- Increased urination
- Blurry vision
- Slow wound healing

### **SYMPTOMS OF LOW GLUCOSE:**

- Fatigue
- Shaking
- Trembling
- Anxiety
- Irritability
- Confusion
- Blurred vision
- Hunger

GLUCOSE LEVEL	INDICATION
70-99 mg/dL	Normal Fasting Glucose
100-125 mg/dL	Impaired Fasting Glucose (pre-diabetes)
126 mg/dL and above on more than one test.	Diabetes

The main mechanisms affecting blood glucose levels are exercise, diet and the body's stress response.

### RELATED TESTS:

Hemoglobin A1C, C-peptide, Urinalysis, CMP, Insulin, Microalbumin, glucose tolerance test, thyroid panel, IGF-1, Amylase, Celiac panel, Food sensitivities, Amino acids, Organic acids, Adrenal stress index, First morning urinary pH

### ELEVATED LEVELS MAY INDICATE:

- Metabolic acidosis
- Insulin Resistance
- Diabetes
- Elevated catecholamines
- Hyperthyroidism
- Graves' disease
- Early-onset Hashimoto's thyroiditis
- Cushing's (elevated cortisol levels)
- Anterior Pituitary Tumor (elevated growth hormone)
- Acromegaly (elevated growth hormone)
- Malnutrition
- Pheochromocytoma
- Pancreatitis
- Food sensitivities
- Low-sodium diet

- Estrogen replacement
- Corticosteroids
- Lithium
- Anti-depressants
- Diuretics
- Salicylates
- Trauma (heart attack, stroke, extreme stress)

**DECREASED LEVELS MAY INDICATE:**

- Hypoglycemia
- Cirrhosis of the liver
- Low carbohydrate diet
- Glycogen-Storage Disease (von Gierke's Disease)
- Addison's disease (low cortisol levels)
- Adrenal fatigue
- Anterior pituitary insufficiency secondary to hypothyroidism, low growth hormone, low cortisol
- Insulinoma
- Malabsorption secondary to Celiac disease
- Excessive alcohol
- Hypothyroidism
- Starvation
- Acetaminophen
- Anabolic steroids

## CLINICAL INDICATIONS:

Patients with elevated glucose should have their body composition evaluated looking specifically for elevated bodyfat % (bioimpedance scale, skin calipers). Ideal bodyfat:

22-28% for women

12-18% for men

Grip strength can be measured via a dynamometer to evaluate for protein malnutrition.

Look for decreased gluteal mass and/or sagging/floppy triceps as well as decreased gait speed. Increasing muscle mass and reducing bodyfat can bring down A1c levels by improving insulin resistance. Patients should be instructed to commence a resistance training program to increase lean body mass and aerobic exercise. The combination of resistance training and aerobic exercise has been shown to be more effective than either type of exercise alone. In addition, if protein malnutrition is present, a minimum of 1.2 grams of protein per kilogram bodyweight may be necessary to restore levels and build lean body mass. Depending on the patient's level of physical activity protein requirements can increase to an upper limit of 2.0 grams of protein per kilogram bodyweight.

A moderate to low-carbohydrate diet (40/30/30) or consumption of carbohydrates only after exercise can be beneficial. A low glycemic index diet can also be beneficial in controlling blood glucose. Increasing fiber in the diet and/or fiber supplementation can reduce post-prandial glucose loads, improve insulin sensitivity and control appetite. A "Mediterranean" style diet that is high in fiber can be an excellent choice for many patients with insulin resistance. Consumption of saturated and trans fats increase post-prandial insulinemia compared to monounsaturated fats which improve beta cell function and insulin sensitivity.

pH evaluation by 1st morning urinary pH via Hydrion pH paper should be done 5 days in a row, discard the high and the low, and average the middle three readings. Metabolic acidosis has been linked to insulin resistance. Ideal pH is 6.4-7.4 preferably measured after at least 6 hours of sleep. A diet rich in the buffering minerals magnesium, calcium, potassium and zinc can improve metabolic acidosis. Supplementation with magnesium glycinate and potassium bicarbonate taken before bed can reverse metabolic acidosis and shift pH into the alkaline range.

## NUTRACEUTICALS THAT MAY BE BENEFICIAL FOR INSULIN RESISTANCE:

- Berberine (Goldenseal, Chinese Coptis, Oregon Grape Root, Barberry)
- Inositol
- Adrenal adaptogens: Eleutherococcus, Rhodiola rosea, Ashwagandha, Panax Ginseng, American Ginseng
- Japanese Knotweed (Resveratrol)

- Olive Leaf Extract
- Reishi Mushroom (*Ganoderma lucidum*)
- *Gymnema sylvestre*
- Acetyl-L-carnitine
- Green Tea (EGCG)
- Fenugreek
- Whey Protein (high in leucine)
- Inositol
- L-carnitine
- L-carnosine
- Caffeine
- Vanadium
- Alpha Lipoic Acid
- Chromium
- Magnesium
- Zinc
- Vitamin D (cholecalciferol, D3)
- Omega-3 Fatty Acids
- Branched Chain Amino Acids
- Cinnamon bark
- B-vitamins

# HEMOGLOBIN A1C

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**Reference Range:** 4.8-5.6%

A1c is a measure of the concentration of glycated hemoglobin A1c in the blood. Blood glucose binds to hemoglobin A which makes up about 95-98% of the hemoglobin in the body. As blood glucose levels rise, more binding occurs to hemoglobin A inside the red blood cell. Glucose bound to hemoglobin is considered glycated hemoglobin thus the name HbA1c or A1c. A1c is mainly used in conventional medicine to monitor diabetics over time. This test provides a picture of the amount of glucose in the blood over the previous 2-3 months. This helps the clinician decide if the treatment has been working effectively to manage blood glucose levels. The American Diabetes Association recommends A1c testing at least twice a year but it is sometimes tested up to four times a year. A1c does not reflect glucose control over the last 3-4 weeks.

A1c is not a sensitive marker so fasting blood glucose should be utilized for these purposes. A1c is more of a general marker of blood glucose and does not reflect acute or recent shifts. A1c levels may not be accurate in individuals with pregnancy, iron-deficiency anemia, vitamin B12 anemia, sickle cell, thalassemia, kidney or liver disease, recent blood transfusion, and recent severe bleeding.

As long as the patient is inflamed, it will be difficult to reduce A1c levels. Identification of the source of inflammation such as infection, food sensitivity, stress, abnormal body composition etc. is paramount to successful clinical outcomes.

Non-diabetic individuals should have an A1c level of 4%-6%.

A diabetic will have A1c levels of 6.5% or higher.

Individuals with A1c levels of 5.7%-6.4% are at an increased risk for developing diabetes.

## SIGNS AND SYMPTOMS OF INSULIN RESISTANCE:

- Acanthosis nigricans
- Increased abdominal obesity
- Fatigue

- Weight-loss resistance
- Post-prandial fatigue and brain fog
- Hair loss (PCOS)
- Decreased lean body mass
- Sugar cravings
- Metabolic acidosis

## **RELATED TESTS:**

Fasting glucose, C-peptide, Urinalysis, CMP, Insulin, Microalbumin, Glucose tolerance test, Thyroid panel, IGF-1, Amylase, Celiac panel, Cortisol, DHEA, Food sensitivities, vitamin D, RBC magnesium, Gait speed, Dynamometer grip strength evaluation, Body composition

## **ELEVATED LEVELS MAY INDICATE:**

- Diabetes
- Insulin Resistance
- Iron-deficiency anemia
- Low-sodium diet
- Inflammation
- Metabolic acidosis
- Multiple Food Sensitivities
- Excessive cortisol levels/decreased DHEA
- Abnormal body composition (decreased muscle mass/increased bodyfat %)
- High carbohydrate diet
- Sedentary lifestyle
- Obesity

## **DECREASED LEVELS MAY INDICATE:**

- Hemolysis
- Macrocytic anemia (B12 deficiency)

- Heavy Bleeding
- Sickle Cell
- Thalassemia

### CLINICAL INDICATIONS:

Patients with elevated A1c should have their body composition evaluated looking specifically for elevated bodyfat % (bioimpedance scale, skin calipers). Ideal bodyfat:

22-28% for women

12-18% for men

Grip strength can be measured via a dynamometer to evaluate for protein malnutrition.

Look for decreased gluteal mass and/or sagging/floppy triceps as well as decreased gait speed. Increasing muscle mass and reducing bodyfat can bring down A1c levels by improving insulin resistance. Patients should be instructed to commence a resistance training program to increase lean body mass and aerobic exercise. The combination of resistance training and aerobic exercise has been shown to be more effective than either type of exercise alone. In addition, if protein malnutrition is present, a minimum of 1.2 grams of protein per kilogram bodyweight may be necessary to restore levels and build lean body mass. Depending on the patient's level of physical activity protein requirements can increase to an upper limit of 2.0 grams of protein per kilogram bodyweight.

A moderate to low-carbohydrate diet (40/30/30) or consumption of carbohydrates only after exercise can be beneficial. A low glycemic index diet can also be beneficial in controlling blood glucose. Increasing fiber in the diet and/or fiber supplementation can reduce post-prandial glucose loads, improve insulin sensitivity and control appetite. A "Mediterranean" style diet that is high in fiber can be an excellent choice for many patients with insulin resistance. Consumption of saturated and trans fats increase post-prandial insulinemia compared to monounsaturated fats which improve beta cell function and insulin sensitivity.

pH evaluation by 1st morning urinary pH via Hydrion pH paper should be done 5 days in a row, discard the high and the low, and average the middle three readings. Metabolic acidosis has been linked to insulin resistance. Ideal pH is 6.4-7.4 preferably measured after at least 6 hours of sleep. A diet rich in the buffering minerals magnesium, calcium, potassium and zinc can improve metabolic acidosis. Supplementation with magnesium glycinate and potassium bicarbonate taken before bed can reverse metabolic acidosis and shift pH into the alkaline range.

Nutraceuticals that may be beneficial for insulin resistance:

See glucose.



# URIC ACID

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**Reference Range:** 3.7-8.6 mg/dL

Uric acid is formed by the metabolism of nucleic acids and is the end-product of purine (adenine & guanine) metabolism. Foods high in purines include organ meats, sardines and anchovies. Alcohol from beer and wine slow the urinary excretion of uric acid. Uric acid is mainly secreted in the urine but small amounts also pass through the gastrointestinal lumen. Uric acid is mainly metabolized in organs that have high metabolic capacity such as the liver, bone marrow and muscle tissue. Purine metabolism is utilized by the body for anabolic processes such as building muscle and other tissues of the body. Imbalances in uric acid can thus be viewed as changes in anabolic vs. catabolic physiological patterns. Gender differences are apparent with females making approximately 20% less uric acid than males. Increased consumption of purine-rich foods will of course increase serum uric acid concentrations. Uric acid is not completely metabolized and excreted on a daily basis and thus contributes to the body's "uric acid pool" which can build over time. Serum and urine levels of uric acid indicate that the amount excreted is equal to the amount filtered.

Gout occurs when there is increased production of uric acid indicated by elevated serum and urinary uric acid levels. This increases the uric pool and uric acid crystals are then deposited in joints resulting in pain and inflammation. The most commonly affected joint is the big toe. Excessive uric acid can be a result of increase production and/or decreased clearance from the body.

## **RELATED TESTS:**

Kidney stone analysis, X-ray of suspected joints, Synovial fluid analysis, Ferritin, Iron/TIBC, CBC, Ceruloplasmin, CMP, Urinalysis, Rheumatoid Factor, Amino acids, Organic acids, 1st morning urinary pH

## **ELEVATED LEVELS MAY INDICATE:**

- Gout
- Inflammation
- Insulin resistance

- Catabolic physiology
- Rheumatoid arthritis
- Atherosclerosis
- Liver failure
- Hyperhomocysteinemia
- Leukemia
- Multiple Myeloma
- Polycythemia vera
- Lymphoblastomas
- Diuretics
- Glomerulonephritis (Check BUN and creatinine levels)
- Collagen disease
- Toxic nephritis
- Diabetic glomerulosclerosis
- Toxemia of pregnancy
- Metastatic cancer
- Chemotherapy
- Fasting
- Stress
- Excessive exercise

**DECREASED LEVELS MAY INDICATE:**

- Metabolic acidosis
- Alcoholism
- Kidney disease
- Toxemia of pregnancy
- Iron deficiency

- Copper deficiency
- Low hematopoiesis
- Fanconi syndrome (check for glycosuria, amino aciduria, hyperphosphaturia)
- Wilson's disease

### **CLINICAL INDICATIONS:**

Dietary evaluation of purine-rich foods is the first priority as well as looking at the overall acidic load of the patient's diet. An alkaline-forming diet can reduce purine exposure and shift the pH to a more alkaline range. 1<sup>st</sup> morning urinary pH can be performed for a general analysis of body pH. If uric acid levels are elevated due to a chronic inflammatory process or insulin resistance then this must be identified and addressed.

Vitamin C has been shown to increase the urinary excretion of uric acid.



# BLOOD UREA NITROGEN (BUN)

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**Reference Range:** 6-20 mg/dL

BUN is mainly used to identify kidney dysfunction along with other tests such as creatinine. BUN is a measurement of the amount of urea in the blood which is produced by the metabolism of protein in the liver. Amino acids are metabolized in the liver to ammonia and then to urea for urinary excretion. Since BUN is made in the liver and excreted by the kidney, disorders of either organ will affect serum levels. A diseased liver will not be able to adequately metabolize amino acids and thus may result in low BUN levels. Diseased kidneys may not be able to efficiently excrete urea and may thus result in elevated BUN serum levels. BUN is manufactured from protein so amino acid deficiencies from the inability to digest and absorb, may result in lower BUN levels. Blood levels increase with age and males have slightly higher levels than females.

BUN is also ordered to monitor patients with diabetes, congestive heart failure and myocardial infarction. Dialysis patients will also be tested regularly to monitor kidney function.

## SIGNS AND SYMPTOMS OF KIDNEY DYSFUNCTION INCLUDE:

- Puffiness around the eyes
- High blood pressure
- Fatigue
- Edema
- Poor appetite
- Decreased urine volume
- Problems with urination
- Mid-back/flank pain
- Foamy, bloody or coffee-colored urine

## RELATED TESTS:

Sodium, Potassium, Creatinine, Creatinine clearance, Calcium, CMP, Microalbumin, eGFR, Urinalysis, Amino acids, Organic acids, Zinc taste test, Dynamometer grip strength

## A BUN/CREATININE RATIO MAY ALSO BE ORDERED FOR DIAGNOSTIC PURPOSES. AN ELEVATED BUN/CREATININE RATIO MAY INDICATE:

- Dehydration
- Congestive heart failure
- High protein diet
- GI bleeding

## A DECREASED BUN/CREATININE MAY INDICATE:

- Liver disease
- Malnutrition

## NORMAL CREATININE/HIGH BUN OF 24-26 MAY BE INDICATIVE OF:

- Catabolic physiology
- Branched-chain amino acid deficiency
- Gluconeogenesis
- Insulin resistance
- Inflammation
- GI atrophy

## ELEVATED BUN LEVELS MAY INDICATE:

- Catabolism
- Kidney Failure
- Congestive heart failure
- Shock
- Stress
- Severe burns

- Dehydration
- Excessive protein intake
- Gastrointestinal bleeding
- Pregnancy

**DECREASED BUN LEVELS MAY INDICATE:**

- Hypochlorhydria
- Protein Deficiency
- Maldigestion
- Malabsorption
- Liver Disease
- Malnutrition
- Overhydration
- Pregnancy
- Chloramphenicol
- Streptomycin

**CLINICAL INDICATIONS:**

After ruling out pathology, imbalances in BUN levels are mainly caused by issues of abnormal protein intake, assimilation and utilization. A dietary review of protein intake with body composition, grip strength testing, Zinc taste test and possibly an amino acid/organic acid evaluation may be in order.



# CREATININE

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**Reference Range:** .76-1.27 mg/dL

Serum creatinine is mainly used to identify and monitor kidney disease. It's use is closely related to BUN and is often connected as a ratio to make a diagnosis. Creatinine is a waste product of creatine breakdown in muscle tissue. The vast majority of creatinine is excreted in the urine and is thus an excellent indicator of kidney function. Creatinine levels are also affected by the amount of lean body mass. Since males normally have more muscle mass than females, their creatinine levels are usually higher.

Blood creatinine levels can be combined with a 24-hour urinary creatinine clearance test to see how the kidneys are functioning. Serum creatinine levels are used to measure eGFR (estimated glomerular filtration rate)

## **RELATED TESTS:**

Creatinine clearance test, Microalbumin, BUN, BUN/creatinine ratio, Urinalysis, CMP, Amino acids, Organic acids, Dynamometer grip strength, Body composition

## **ELEVATED LEVELS MAY INDICATE:**

- Excessive exercise resulting in increased muscle tissue breakdown
- Kidney disease
- Pyelonephritis
- Glomerulonephritis
- Increased muscle mass
- Kidney stone
- Prostate disease
- Diabetes
- Congestive heart failure

- Atherosclerosis
- Dehydration
- Autoimmune disease (Lupus)
- High meat diet

**DECREASED LEVELS MAY INDICATE:**

- Decreased muscle mass
- Small stature
- Pregnancy

**CLINICAL INDICATIONS:**

Low creatinine levels may indicate decreased lean body mass which is a valuable indicator of overall health. Lean body mass and grip strength can give a good indication if the patient needs to increase protein calories and/or commence a resistance training program to build muscle mass. A minimum of 1.2 grams of protein per kilogram body weight may be necessary to increase lean body mass. In addition to food intake, a protein powder such as whey can be added to the diet for extra protein. Whey protein should be high in leucine for the most anabolic effect.

# BLOOD ELECTROLYTES

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## **Sodium, Potassium, Chloride and Carbon Dioxide (bicarbonate)**

**Reference Range Sodium:** 134-144 mmol/L

**Reference Range Potassium:** 3.5-5.2 mmol/L

**Reference Range Chloride:** 97-108 mmol/L

**Reference Range CO<sub>2</sub>:** 20-32 mmol/L

The blood electrolytes are combined due to the delicate interaction among them and because they are reported together on blood chemistry reports. Electrolytes are transported through the blood and lymphatics driven primarily by the heart and skeletal muscle. Carbon dioxide is a product of the metabolism of protein, carbohydrates and fats and is therefore a good indicator of metabolic acidosis.

Potassium along with water, is released from cellular storage during increased catabolic activity. Sodium and chloride mainly reside outside of the cell in equivalent concentrations. Sodium can be stored in the bone and is utilized during times of metabolic acidosis. Potassium on the other hand has an equal concentration in the cell and blood. Potassium is transported into the cell along with glucose and therefore insulin resistance may result in elevated potassium in the blood.

Alterations in sodium concentration can affect the thyroid gland, adrenal gland, brain, heart, lungs, liver and kidneys.

## **SYMPTOMS OF LOW SODIUM (HYPONATREMIA) INCLUDE:**

- Weakness
- Lethargy
- Confusion

**SYMPTOMS OF ELEVATED SODIUM (HYPERNATREMIA) INCLUDE:**

- Muscle twitching
- Agitation
- Thirst
- Decreased urinary output

**HYPONATREMIA USUALLY OCCURS DUE TO:**

- Diuretics
- Diarrhea
- Addison's disease
- Cirrhosis
- Excessive fluid intake
- Heart failure
- Excessive ADH production
- Nephrotic syndrome and other kidney diseases
- Medications such as diuretics, anti-depressants, heparin and ACE inhibitors can cause hyponatremia

**HYPERNATREMIA IS USUALLY CAUSED BY:**

- Insulin resistance
- Metabolic acidosis
- Dehydration
- Hypothyroidism
- Cushing's disease
- Diabetes insipidus which is due to decreased ADH production
- Medications such as anabolic steroids, oral contraceptives, antibiotics, cough medicine, corticosteroids and laxatives can cause hypernatremia

**ELEVATED POTASSIUM LEVELS (HYPERKALEMIA) IS MAINLY SEEN IN:**

- Kidney disease
- Diuretics
- Infections
- Addison's disease
- Diabetes
- Dehydration
- Hyperaldosteronism
- Tissue injury
- NSAIDS, ACE inhibitors, beta blockers

**DECREASED POTASSIUM LEVELS (HYPOKALEMIA) MAY BE CAUSED BY:**

- Insulin resistance
- Metabolic acidosis
- Low dietary intake
- Vomiting
- Diarrhea
- Excessive sweating
- Hyperaldosteronism
- Diabetes
- Excessive acetaminophen intake
- Antibiotics, antifungals, corticosteroids

Foods high in potassium include fruits, vegetables, nuts and seeds.

Chloride works with sodium, potassium and bicarbonate to regulate fluid-electrolyte balance and pH. Most chloride is found outside of the cell and it tends to follow sodium levels. However, chloride can shift independently of sodium during shifts in acid-base balance due to its buffering abilities. Chloride is used to make hydrochloric acid in the stomach which can result in low chloride levels after a meal.

**INCREASED CHLORIDE (HYPERCHLOREMIA) LEVELS MAY BE CAUSED BY:**

- Insulin resistance
- Hyperventilation (respiratory alkalosis)
- Cushing's disease
- Excessive stress
- Dehydration
- Metabolic acidosis
- Kidney disease

**DECREASED CHLORIDE (HYPOCHLOREMIA) LEVELS MAY BE CAUSED BY:**

- Addison's disease
- Adrenal fatigue
- Vomiting
- Respiratory acidosis
- Metabolic alkalosis
- Congestive heart failure
- Emphysema

CO<sub>2</sub> measurements in the blood are mainly bicarbonate which works with sodium, potassium and chloride to regulate acid-base balance. Blood gases may be ordered to differentiate pH imbalances due to respiratory pathology or metabolic imbalances of acids and bases.

**INCREASED CO<sub>2</sub> LEVELS MAY BE CAUSED BY:**

- Metabolic alkalosis
- Vomiting
- Cushing's disease
- Excessive stress
- Lung disease
- Conn syndrome

- Lung diseases such as COPD
- Hydrocortisone, steroids, diuretics

### DECREASED CO<sub>2</sub> LEVELS MAY BE CAUSED BY:

- Metabolic acidosis
- Inflammation
- Aspirin overdose
- Addison's disease
- Adrenal fatigue
- Diarrhea
- Diabetic ketoacidosis
- Kidney disease
- Methanol poisoning
- Tetracycline, methicillin, diuretics

It is important to discuss water in the body when covering blood electrolytes. The majority of water is stored inside the cell accounting for about 40% of body weight and water stored extracellularly accounts for 15-20% of body weight. All of the secretions of the body including gastric, biliary, pancreatic and small intestine contain the blood electrolytes.

The kidney is the major secretory organ controlling blood electrolyte equilibrium with the remaining excretion occurring in the sweat and stool. Carbon dioxide is excreted by the lungs. The kidney and lung are thus the major regulators of blood electrolytes, acid-base balance and body fluid. The kidney is under control of aldosterone from the adrenal cortex and antidiuretic hormone from the posterior pituitary. Thyroid hormone also influences water and electrolyte concentration because of its control of cellular metabolism.

Edema, thirst and changes in blood pressure are signs of electrolyte imbalances. Edema mainly occurs in the lower legs in such cases.

## **RELATED TESTS:**

Aldosterone, ADH, Comprehensive Metabolic Profile, Cortisol, 1<sup>st</sup> morning urinary pH, DHEA, Thyroid panel, Glucose, A1C, CBC, Urinalysis

An excellent example of blood electrolyte balance can be seen when looking at dehydration. This example indicates how the body regulates electrolytes, fluid and acid-base balance.

## **DEHYDRATION RESULTS IN THE FOLLOWING:**

- Increased sodium
- Normal potassium
- Normal or decreased CO<sub>2</sub>
- Increased chloride
- Decreased blood volume
- Increased urinary sodium
- Increased urinary potassium
- Decreased urine pH
- Decreased urine volume

## **MALABSORPTION PATTERNS:**

- Decreased sodium
- Decreased potassium
- Normal or decreased CO<sub>2</sub>
- Normal chloride
- A serum carotene, lactulose/manitol and urinary 5-HIAA can confirm the diagnosis.

## **CONGESTIVE HEART FAILURE:**

- Normal or decreased sodium
- Normal potassium
- Normal CO<sub>2</sub>
- Decreased chloride

**PYLORIC OBSTRUCTION:**

- Decreased sodium
- Decreased potassium
- Increased CO<sub>2</sub>
- Decreased chloride

**DIARRHEA:**

- Decreased sodium
- Decreased potassium
- Decreased CO<sub>2</sub>
- Decreased chloride

**DIAPHORESIS:**

- Decreased sodium
- Normal potassium
- Normal CO<sub>2</sub>
- Decreased chloride

**KIDNEY FAILURE:**

- Decreased sodium
- Increased potassium
- Decreased CO<sub>2</sub>
- Increased chloride

**EMPHYSEMA:**

- Normal sodium
- Normal potassium
- Increased CO<sub>2</sub>
- Decreased chloride

**SALICYLATE TOXICITY:**

- Normal sodium
- Normal or decreased potassium
- Decreased CO<sub>2</sub>
- Increased chloride

**ADRENAL CORTICAL INSUFFICIENCY AKA “ADRENAL FATIGUE”:**

- Decreased sodium
- Increased potassium
- Normal or decreased CO<sub>2</sub>
- Decreased chloride

**DIABETES INSIPIDUS:**

- Normal or increased sodium
- Normal potassium
- Normal CO<sub>2</sub>
- Increased chloride

**PRIMARY ALDOSTERONISM:**

- Increased sodium
- Decreased potassium
- Increased CO<sub>2</sub>
- Decreased chloride

**RENAL TUBULAR ACIDOSIS:**

- Decreased sodium
- Decreased potassium
- Decreased CO<sub>2</sub>
- Increased chloride

### DIABETIC ACIDOSIS:

- Decreased sodium
- Normal or increased potassium
- Decreased CO<sub>2</sub>
- Decreased chloride

### **CLINICAL INDICATIONS:**

pH evaluation by 1<sup>st</sup> morning urinary pH via Hydrion pH paper should be done 5 days in a row, discard the high and the low, and average the middle three readings. Metabolic acidosis has been linked to insulin resistance and electrolyte imbalances. Ideal pH is 6.4-7.4 preferably measured after at least 6 hours of sleep. A diet rich in the buffering minerals magnesium, calcium, potassium and zinc can improve metabolic acidosis. Supplementation with magnesium glycinate and potassium bicarbonate taken before bed can reverse metabolic acidosis and shift pH into the alkaline range.

Adrenal gland imbalances can result in fluid-electrolyte imbalances which can be evaluated via blood, urine or salivary cortisol and DHEA levels. The source of the adrenal stress must be identified for optimal clinical outcomes.

### CAUSES OF ADRENAL STRESS INCLUDE:

- Infections
- Insulin resistance
- Food sensitivities
- Anemia
- Pain
- Emotional stress
- Metabolic acidosis
- Gastrointestinal hyperpermeability
- Inflammation
- Insufficient protein/caloric intake
- Excessive exercise
- Sleep deprivation

- Increased environmental toxic load
- Hyper and hypothyroidism
- Autoimmune diseases
- Social isolation

**ADRENAL IMBALANCES CAN BE SUPPORTED WITH THE FOLLOWING ADRENAL ADAPTOGENS:**

- Eleutherococcus (Siberian Ginseng)
- Ashwagandha
- Rhodiola rosea
- Panax ginseng
- American ginseng

Licorice root can increase the half-life of cortisol which may be useful for decreased cortisol levels.

Phosphorylated and/or phosphatidyl serine can help balance cortisol by inhibiting the stress response of the hypothalamic/pituitary/adrenal axis therefore reducing cortisol levels.

Pregnenelone in a time-released capsule taken in the AM can be useful in patients with adrenal fatigue.

DHEA supplementation may be necessary when DHEA levels are extremely low and also to reduce excessive cortisol levels. Topical magnesium may increase DHEA levels.

It is extremely important to reduce insulin resistance in patients with electrolyte and adrenal gland imbalances. See A1c and glucose for more details on addressing insulin resistance.

# CALCIUM

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**Reference Range:** 8.7-10.2 mg/dL

Calcium is important for the function of nerves, the heart, muscles, and in blood clotting and bone formation. 99% of calcium is found in bone and 1% in the blood. The daily requirement of calcium is approximately 500-800 mg with only 50% of ingested calcium actually absorbed. Calcium is absorbed in the small intestine under the influence of vitamin D and parathyroid hormone. Calcium absorption is optimal in an acidic pH in the intestine. Calcium absorption is inhibited by increased amounts of fat, phytic acids and phosphorous in the diet. Once calcium is absorbed, it is transported either bound to phosphate in an ionized state (free-form) or in an un-ionized protein-bound form in about a 50:50 ratio. There is a delicate balance between calcium and phosphorous in the blood that is always adapting to a state of equilibrium. Calcium is mainly found in the extracellular fluid whereas large quantities of phosphate are found inside the cell. Calcium accounts for 2-3% of total body weight with an average of 1,100 grams stored in the bone. Vitamin D and parathyroid hormone not only affect calcium absorption in the bone, but also in the kidney where about 99% of calcium is reabsorbed.

Ionized (free) calcium can be tested to help differentiate where the calcium imbalance is coming from. Blood protein levels can affect total calcium levels and must therefore be taken into account when interpreting blood tests.

A drop in serum calcium levels results in increased parathyroid hormone secretion which pulls calcium from bone into the bloodstream. Parathyroid hormone also increases calcium absorption through the intestine, kidney and increases urinary excretion of phosphate.

When calcium levels rise, calcitonin is released from the thyroid gland which reduces serum calcium levels.

Blood pH also affects serum calcium levels. When the pH is more acidic, calcium levels increase and when the pH is more alkaline, calcium levels decrease.

**THE MAIN CLINICAL INDICATORS OF CALCIUM DYSREGULATION INCLUDE:**

- Kidney stones
- Bone pathology
- Muscle spasms
- Thyroid disease
- Malnutrition
- Convulsions

Calcium levels should be monitored in patients with cancer, especially of the kidney, breast, lung, head & neck, and in multiple myeloma.

**SYMPTOMS OF DECREASED CALCIUM INCLUDE:**

- Tingling in the fingers
- Abdominal cramps
- Muscle cramps

**SYMPTOMS OF ELEVATED CALCIUM LEVELS INCLUDE:**

- Increased thirst
- Nausea
- Vomiting
- Fatigue
- Weakness
- Urinary frequency
- Loss of appetite
- Constipation
- Abdominal pain

## **RELATED TESTS:**

Parathyroid hormone, Calcitonin, CMP, Vitamin D, Magnesium, Phosphorous, Thyroid panel, Amylase, Celiac panel, Urinary metals challenge test, Amino acids, Comprehensive digestive stool analysis, Dynamometer grip strength, Cortisol, DHEA-sulfate, Progesterone, Estrogens, Testosterone, First morning urinary pH

## **ELEVATED CALCIUM LEVELS MAY INDICATE:**

- Hyperparathyroidism
- Chemical or heavy metal toxicity
- Excess Vitamin D Supplementation
- Sarcoidosis
- Metastatic Carcinoma of Bone
- Hyperthyroidism
- Tuberculosis
- Kidney transplant
- Thiazide diuretics

## **DECREASED CALCIUM LEVELS MAY INDICATE:**

- Hypoproteinemia (most common cause, ionized calcium stays normal)
- Vitamin D deficiency
- Magnesium deficiency
- Insufficient dietary calcium
- Osteoporosis
- Hypoparathyroidism
- Heavy Metal Toxicity
- Excessive dietary phytate intake
- Malabsorption
- HCL Deficiency
- Pancreatitis

- Kidney failure
- Malnutrition
- Nephrotic Syndrome
- Cirrhosis of the liver
- Nephritis
- Renal Tubular Acidosis
- Hypothyroidism

### **CLINICAL INDICATIONS FOR CALCIUM IMBALANCES:**

Since calcium levels are affected by pH, evaluation by 1<sup>st</sup> morning urinary pH via Hydrion pH paper should be done 5 days in a row, discard the high and the low, and average the middle three readings. Metabolic acidosis has been linked to insulin resistance. Ideal pH is 6.4-7.4 preferably measured after at least 6 hours of sleep. A diet rich in the buffering minerals magnesium, calcium, potassium and zinc can improve metabolic acidosis. Supplementation with magnesium glycinate and potassium bicarbonate taken before bed can reverse metabolic acidosis and shift pH into the alkaline range.

Calcium supplementation is not necessary in men and can actually increase the risk of a cardiovascular event. Calcium supplementation in women is controversial due to some scientific evidence that it can increase the risk of a cardiovascular event. The higher the dose of calcium supplementation combined with high dietary intake of calcium, the greater the risk factors outlined above. It is recommended that calcium be obtained mainly from dietary sources rich in calcium. If calcium supplementation is utilized then 500-1,000mg may be the safest dose. In addition, calcium supplementation has not been shown to prevent bone fracture or improve osteoporosis. Dairy products are often recommended for calcium but due to the high phosphorous content it has been postulated that dairy may not be a good source of calcium once digestion, absorption and assimilation are taken into account.

### **FOOD HIGH IN CALCIUM INCLUDE:**

- Green-leafy vegetables such as kale, collards, chard and spinach
- Broccoli
- Green beans
- Okra
- Almonds
- Sesame seeds

- Brazil nuts
- Figs
- Beans
- Quinoa
- Blackstrap molasses
- Canned sardines

A 3-day diet diary will provide data regarding dietary calcium intake.



# PHOSPHORUS

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**Reference Range:** 2.5-4.5 mg/dL

Phosphorous is absorbed in the small intestine under the influence of parathyroid hormone and vitamin D with a daily requirement of approximately 1.0-1.5 grams. Phosphorous is important in bone growth, muscle and nerve function, and energy production. Phosphorous also plays an important role in acid-base balance acting as a buffering agent. Phosphorus differs from calcium in that it is present in large quantities inside the cell. Phosphorus and calcium are constantly maintaining a state of equilibrium in the blood. Approximately 80-90% of stored phosphate is in the bone and teeth with the remainder in intracellular and extracellular fluid. 10% is found in muscle and 1% in the nervous system. 60% of phosphate excretion occurs in the kidney with the rest being excreted in the feces. The amount excreted in the kidney is under the control of vitamin D and parathyroid hormone. Parathyroid hormone increases phosphate secretion in the kidney thus inhibiting reabsorption by the renal tubules. Since calcium and phosphorous are so intimately connected, an understanding of calcium physiology clearly explains the balance between the two. Hyperparathyroidism is an example of this relationship which results in elevated serum calcium levels and decreased serum phosphorous levels. Hypoparathyroidism results in the exact opposite presentation.

## SYMPTOMS OF LOW PHOSPHOROUS INCLUDE:

- Muscle weakness
- Confusion

## SYMPTOMS OF HIGH PHOSPHOROUS INCLUDE:

- Seizures
- Muscle cramps
- Confusion

## RELATED TESTS:

Magnesium, Parathyroid Hormone, Vitamin D, Calcium, Electrolyte panel, Thyroid panel,

1st morning urinary pH, Amino acids, Zinc taste test

## ELEVATED LEVELS MAY INDICATE:

- Excessive Phosphate Intake
- Chronic Nephritis
- Kidney failure
- Hypoparathyroidism
- Hypocalcemia
- Diabetic ketoacidosis
- Soft drinks

## DECREASED LEVELS MAY INDICATE:

- Metabolic acidosis
- Hypercalcemia
- Hypochlorhydria
- Insufficient protein assimilation
- Malnutrition
- Hypovitaminosis D
- Malabsorption
- Renal Tubular Acidosis
- Hyperparathyroidism
- Rickets
- Osteomalacia
- Hypokalemia
- Hypothyroidism

- Chronic antacid use
- Alcoholism
- Diuretics
- Burns

### **CLINICAL INDICATIONS:**

Addressing metabolic acidosis along with pathological or dietary influences can balance abnormal phosphorous levels. A dietary review looking specifically at protein intake and acid versus alkaline-forming foods is important.

Meat, eggs, dairy, nuts, legumes and grains are all excellent sources of phosphorous.



# MAGNESIUM

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**Reference Range:** 1.6-2.6 mg/dL

Magnesium is involved in approximately 350 enzymatic reactions in the body mainly related to energy production, muscle contraction, nerve function, strong bone formation and phosphorylation. Average dietary intake is 360mg a day however it has been reported that up to 90% of Americans are deficient in magnesium. Magnesium is similar to calcium once it is in the bloodstream in that 50% is bound to protein and the rest is unbound. Magnesium mainly resides intracellularly with only 1% of total body magnesium in the blood and 50-75% found in the bone. Magnesium is influenced by parathyroid hormone which decreases excretion in the kidney. Growth hormone increases the intestinal absorption of magnesium. Addison's disease and hyperthyroidism will result in elevation of serum magnesium. Magnesium deficiency can lead to potassium and calcium deficiency due to impaired metabolism.

## RELATED TESTS:

Calcium, Potassium, Vitamin D, Parathyroid Hormone, Phosphorous, CO<sub>2</sub>, Sodium, Chloride, Thyroid panel, RBC magnesium

## SYMPTOMS OF MAGNESIUM DEFICIENCY:

- Cardiac Arrhythmia
- Loss of appetite
- Confusion
- Diarrhea
- Fatigue
- Muscle Weakness/Cramps
- Irritability
- Nausea

- Numbness/Tingling
- Seizures

**ELEVATED LEVELS MAY INDICATE:**

- Addison's disease
- Adrenal Fatigue
- Hypothyroidism
- Dehydration
- Hyperparathyroidism
- Kidney failure
- Excessive antacid intake
- Diabetic Acidosis
- Thyroid medication

**DECREASED LEVELS MAY INDICATE:**

- Laennec's Cirrhosis
- Hypoparathyroidism
- Ulcerative Colitis
- Malnutrition
- Malabsorption
- Alcoholism
- Toxemia of pregnancy
- Severe Burns
- Diarrhea
- Diuretics
- Crohn's disease
- Diabetes

## CLINICAL INDICATIONS:

Serum magnesium may not be a good indicator of magnesium deficiency and therefore the more specific test red blood cell magnesium may be indicated. Magnesium is depleted during metabolic acidosis, stress, inflammation and a high carbohydrate diet. 1<sup>st</sup> morning urinary pH can be useful to diagnose a deficiency in buffering minerals. If the pH is found to be acidic then magnesium supplementation along with potassium bicarbonate can be utilized to increase buffering mineral reserves and shift the pH into a healthier alkaline range.

Have the patient measure their 1st morning urinary pH for 5 days in a row, discard the high and the low, and average the middle three for an estimate of pH. Measurements should be taken preferably after approximately 6 hours of sleep/fast.

Magnesium glycinate is the preferred form of magnesium due to its excellent level of absorption. Combine magnesium glycinate with potassium bicarbonate taken before bed and incrementally increase the dose each night until the 1st morning urinary pH is in the alkaline range. Taking magnesium at night can also improve sleep due to its calming effect on muscle tissue and the nervous system.

Foods rich in magnesium include green leafy vegetables, nuts, seeds, coffee, chocolate, dairy, beans and whole grains. Generally, foods rich in fiber are rich in magnesium.

## MAGNESIUM SUPPLEMENTATION MAY BE USEFUL FOR THE FOLLOWING CONDITIONS:

- Constipation
- Works as an antacid for GERD
- Cardiovascular issues: Chest pain, irregular heartbeat, high blood pressure, mitral valve prolapse
- ADHD
- Anxiety
- Chronic fatigue syndrome
- Lyme disease
- Fibromyalgia
- Leg cramps
- Diabetes
- Kidney stones
- Migraine headaches

- Osteoporosis
- PMS
- Altitude sickness
- Urinary incontinence
- Restless leg syndrome
- Asthma
- Hay fever
- Multiple sclerosis
- Hearing loss prevention
- Stroke prevention
- Eclampsia and pre-eclampsia
- Magnesium can be applied to the skin to treat strep infections, ulcers, boils, carbuncles and to speed up wound healing

# PLASMA PROTEIN

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**Reference Range Total Protein:** 6.0-8.5 g/dL

**Reference Range Albumin:** 3.5-5.5 g/dL

**Reference Range Globulin:** 1.5-4.5 g/dL

**Reference Range Albumin/Globulin Ratio:** 1.1-2.5

Proteins are broken down into amino acids in the stomach and small intestine by hydrochloric acid and pepsin in the stomach and then pancreatic enzymes such as trypsin and chymotrypsin in the duodenum. When digestion is functioning optimally, about 95% of the protein ingested is properly broken down into amino acids and absorbed in the small intestine. Amino acids are transported to the liver where albumin, globulin, prothrombin and fibrinogen are manufactured. On average, a 70kg man produces about 15-20 grams of plasma protein per day where a constant equilibrium is maintained in the blood. The absolute minimum daily requirement of protein is 0.5 grams per kilogram bodyweight but in chronically ill patients the range is .8-2.0 grams per kilogram bodyweight.

Blood proteins are utilized for tissue growth, as buffers in acid-base balance, maintaining water balance through oncotic pressure, antibody formation, coagulation precursors, and in the transport of sex hormones, thyroid hormone, lipids, vitamins and metals. Stored protein in the liver, kidney, gut lining, skin and muscle can all be catabolized during times of starvation or excessive need for repair when under stress to meet the caloric needs of the individual. This process occurs in the liver where amino acids can be transformed into glucose for energy, hormones and purines and pyrimidines for new cell production. Amino acids can also be converted into pyruvic acid, alpha-ketoglutaric acid and aspartic acid for use in the Citric Acid Cycle (Kreb's Cycle). The by-products of amino acids metabolism are creatinine, urea, CO<sub>2</sub>, uric acid, phosphates and water. Only a minimal amount of protein and amino acids are ever excreted from the body via feces and urine.

Plasma proteins are under regulation by the hormones cortisol, thyroid hormone, androgens, growth hormone and insulin. The quality and quantity of the protein ingested is a major factor in protein metabolism. Insulin, androgens and growth hormone create an anabolic state which is a net increase of protein synthesis. Excessive cortisol and thyroid hormone create a catabolic state which is a net increase in

the breakdown of amino acids. However, physiological amounts of cortisol and thyroid hormone have an opposite effect creating an anabolic state when there is a deficiency. Growth hormone and insulin can act synergistically to create an anabolic state.

Albumin makes up 60% of plasma proteins and globulin makes up the other 40%. Albumin is involved in maintaining osmotic pressure and as a carrier of hormones, drugs, vitamins small molecules and ions such as calcium. Globulin is a heterogenous group involved in hormones, enzymes and antibodies. A ratio of the two is known as the Albumin/Globulin ratio which is utilized to make a diagnosis along with the other plasma protein values.

Elevated globulin can be indicative of food sensitivities and/or inflammation.

### **SYMPTOMS FROM ABNORMAL PLASMA PROTEINS INCLUDE:**

- Fatigue
- Periorbital edema
- Jaundice
- Weight loss
- Edema around the mid-section and legs

### **RELATED TESTS:**

CMP, Albumin, Globulin, A/G Ratio, BUN, Creatinine, Liver Panel, Protein electrophoresis, Urinalysis, Dynamometer grip strength, Body composition, Amino acids

### **ELEVATED TOTAL PROTEIN MAY INDICATE:**

- Any Inflammatory Process
- Multiple myeloma
- Dehydration
- Viral Infection

### **DECREASED TOTAL PROTEIN MAY INDICATE:**

- Catabolic state
- Sarcopenia
- Insufficient dietary protein

- Congestive heart failure
- Kidney disease
- Malnutrition
- Liver disease
- Decreased Hydrochloric Acid
- Malnutrition
- Malabsorption
- Celiac Disease
- Inflammatory Bowel

**ELEVATED ALBUMIN MAY INDICATE:**

- Dehydration

**DECREASED ALBUMIN MAY INDICATE:**

- Hypothyroidism
- Diabetes
- Congestive Heart Failure
- Cancer
- Malnutrition
- Malabsorption
- Liver Cirrhosis
- Nephrotic Syndrome
- Infections
- Chronic Inflammation
- Burns
- Surgery
- Shock

**HIGH ALBUMIN/GLOBULIN RATIO MAY INDICATE:**

- Leukemia

**LOW A/G RATIO MAY INDICATE:**

- Autoimmune disease
- Multiple myeloma
- Liver Cirrhosis
- Kidney disease (nephrotic syndrome)

**STARVATION:**

- Decreased albumin and globulins

**IDIOPATHIC STEATORRHEA:**

- Decreased albumin and globulins

**PORTAL CIRRHOSIS:**

- Decreased Albumin
- Increased Globulin

**ACUTE AND CHRONIC BACTERIAL INFECTIONS:**

- Decreased or normal albumin
- Increased globulin

**HYPOGAMMAGLOBULINEMIA:**

- Normal Albumin
- Decreased Globulins

**VIRAL HEPATITIS:**

- Decreased Albumin
- Increased Globulins

**MULTIPLE MYELOMA:**

- Decreased Albumin
- Increased Gamma Globulin
- Increased Total Protein

**NEPHROTIC SYNDROME:**

- Decreased Albumin
- Increased Alpha-2 globulin
- Increased Beta globulin
- Decreased gamma globulin

**CLINICAL INDICATIONS:**

A dietary review of protein intake is important in imbalanced plasma protein levels. Testing grip strength by dynamometer can be useful for identifying protein malnutrition. Body composition testing looking at bodyfat % can be used to identify decreases in lean body mass and increases in adipose tissue. .8-2.0 grams of protein per kilogram bodyweight may be needed to restore amino acid balance and build lean body mass.

Patients may need to commence a resistance training program to build muscle and reduce body fat. In addition to dietary protein intake, whey protein or other protein powder source such as pea, hemp or rice protein can be added to the diet to increase lean body mass. Whey protein supplements high in leucine will provide the most anabolic effect. Branched chain amino acids and/or free-form amino acid supplements can be useful in certain cases as well.



# BILIRUBIN

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**Reference Range:** 0-1.2 mg/dL

Bilirubin is a by-product of hemoglobin metabolism but approximately 30% is derived from other compounds including myoglobin, catalases and cytochromes. This blood test mainly used to assess liver function (cirrhosis, hepatitis, gall stones) and increased red blood cell destruction such as in hemolytic anemia. As red blood cells are destroyed each day, about 7-8 grams of hemoglobin is produced. This process occurs in the liver, spleen and bone marrow (reticuloendothelial system). Globin is cleaved and the heme portion loses its iron resulting in a protoporphyrin which yields the golden-hued pigment bilirubin. In order for bilirubin to remain in the blood it must stay bound to albumin. Bilirubin is transported to the liver where it is conjugated by glucuronyl transferase and uridine triphosphate. Sulfates are also involved in conjugation but to a lesser degree. Conjugation creates a water-soluble compound so it can be excreted into the bile. Jaundice (yellow pigmentation) occurs when the excretory mechanisms of bilirubin are compromised resulting in a build-up of bilirubin in the liver and blood stream. The kidneys can excrete water-soluble bilirubin in cases of accumulation. Jaundice (yellow skin and eyes) can occur when conjugation is normal but the excretory process is not.

Clinical signs of obstructive jaundice where bilirubin excretion into the feces is compromised include light, gray or colorless stool. The fecal compound urobilinogen is reabsorbed into portal circulation where it is almost completely cleared and then re-excreted as bilirubin. Small amounts pass through hepatic clearance and end up in the urine as urobilinogen. Hepatic parenchymal disease results in decreased bilirubin clearance and increased urobilinogen in the urine. If there is complete obstruction of the bile ducts, urobilinogen will be absent from the urine.

Direct bilirubin is conjugated and indirect bilirubin is unconjugated.

## SIGNS & SYMPTOMS OF ABNORMAL BILIRUBIN LEVELS INCLUDE:

- Fatigue/malaise
- Amber, dark-colored urine
- Nausea/vomiting

- Abdominal pain
- Swelling

## **RELATED TESTS:**

Urinalysis, CBC, Liver panel, GGT, Hepatitis panel, Alkaline phosphatase, ALT, AST, Reticulocyte count, Haptoglobin, Babesia, Intrinsic factor antibodies, Methylmalonic acid, Porphyrin profile, Heavy metal urine challenge test, Hepatitis screen

## **INCREASED UNCONJUGATED (INDIRECT) BILIRUBIN MAY INDICATE:**

- Cirrhosis
- Hemolytic anemia
- Pernicious anemia
- Gilbert Syndrome
- Chemical or heavy metal toxicity

## **INCREASED CONJUGATED (DIRECT) BILIRUBIN DUE TO DECREASED EXCRETION MAY INDICATE:**

- Alcoholic liver disease
- Viral hepatitis
- Drug reactions

## **INCREASED CONJUGATED BILIRUBIN DUE TO BLOCKAGE OF EXCRETORY PATHWAYS MAY INDICATE:**

- Gall stones
- Scarring of the bile ducts
- Tumors

Low levels of bilirubin are not considered significant.

Strenuous exercise can increase serum bilirubin levels.

Males tend to have higher levels than females and African-Americans tend to have lower levels than Caucasians.

**HEREDITARY SPHEROCYTOSIS:**

- Increased indirect serum bilirubin
- Normal direct serum bilirubin
- Normal urine bilirubin
- Increased urine urobilinogen
- Increased stool urobilinogen

**VIRAL HEPATITIS:**

- Increased indirect serum bilirubin
- Increased direct serum bilirubin
- Increased urine bilirubin
- Normal or increased urine urobilinogen
- Normal or decreased stool urobilinogen

**LAENNEC'S CIRRHOSIS:**

- Increased indirect serum bilirubin
- Increased direct serum bilirubin
- Increased urine bilirubin
- Normal or increased urine urobilinogen
- Normal stool urobilinogen

**CARCINOMA OF THE HEAD OF THE PANCREAS:**

- Normal indirect serum bilirubin
- Increased direct serum bilirubin
- Increased urine bilirubin
- Decreased urine urobilinogen
- Decreased stool urobilinogen

**CHOLEDOCHOLITHIASIS:**

- Normal indirect serum bilirubin
- Increased intermittently direct serum bilirubin
- Increased urine bilirubin
- Decreased urine urobilinogen
- Decreased stool urobilinogen

# ALKALINE PHOSPHATASE (ALP)

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**Reference Range:** 25-150 IU/L

The phosphatases are enzymes classified by their maximum activity at specific pH levels. Alkaline phosphatase is mainly found in the hepatobiliary system, bone, intestinal mucosa, kidney, and thyroid gland. ALP isoenzymes are found in each tissue which can be tested to identify the tissue of highest ALP activity for diagnosis of pathology. ALP is important for digestion and mucosal absorption due to its role in the hydrolysis of organic phosphates. ALP is also involved in osteoblastic tissue activity and regeneration and proliferation of liver tissue. ALP is utilized to distinguish obstructive and hepatocellular jaundice. ALP is elevated in obstructive jaundice but it is infrequently elevated in hepatocellular jaundice. An elevated ALP in hepatomegaly without jaundice may indicate metastatic liver disease. ALP can be used to differentiate hyperparathyroidism from various bone diseases when tested along with phosphorous, calcium and x-ray.

Elevations in ALP, liver enzymes and bilirubin usually indicate liver pathology. If ALP, calcium and phosphorous are elevated, then the issue usually involves bone. In hepatitis, ALP will not be as elevated as AST and ALT. In bile duct obstruction, ALP and bilirubin will be elevated much higher than AST and ALT.

## SIGNS AND SYMPTOMS OF ABNORMAL ALP INCLUDE:

- Dark urine/light-colored stool
- Fatigue
- Weakness
- Vomiting
- Nausea
- Jaundice
- Pruritis
- Loss of appetite
- Abdominal pain and swelling

Bone and/or joint pain, deformed bones or frequent fractures can indicate a bone disorder.

### **RELATED TESTS:**

Liver panel, AST, ALT, GGT, Bilirubin, Bone markers, ALP isoenzymes, Zinc taste test, Thyroid panel, Amylase, Intrinsic factor antibodies, CBC, methylmalonic acid, Vitamin D, ALP isoenzymes

### **ELEVATED LEVELS MAY INDICATE:**

- Elevated bone turnover/loss such as a healing fracture (normal in growing children)
- Bile duct obstruction (gall stones, scarring, cancer)
- Cirrhosis of the Liver
- Hepatitis
- Rheumatoid Arthritis
- Hypothyroidism
- Paget's disease
- Rickets
- Bile acid deficiency
- Excessive dietary fat or protein
- Metastatic Bone Tumors
- Metastatic Prostatic Carcinoma
- Osteogenic Sarcoma
- Osteomalacia
- Carcinoma of the Head of the Pancreas
- Metastatic Carcinoma of the Liver
- Pregnancy

### **DECREASED LEVELS MAY INDICATE:**

- Hypothyroidism
- Pernicious anemia

- Scurvy
- Low fat or low protein diet
- Zinc deficiency
- Excessive vitamin D intake
- Oral contraception

## CLINICAL INDICATIONS:

After ruling out any serious pathological causes of abnormal alkaline phosphatase via blood testing or diagnostic imaging, a simple and inexpensive Zinc taste test can be done to evaluate Zinc status. Simply purchase a liquid Zinc solution and follow these instructions:

Put about 2 tablespoons of the Zinc solution in the patient's mouth and swish it around for 30 seconds noting any specific tastes. Spit out the solution and do not swallow because Zinc can cause nausea on an empty stomach.

- If the patient doesn't taste anything then the patient may be Zinc deficient.
- If the patient notices a "dry", "furry", "sweet" or "mineral" taste then the patient may be Zinc deficient.
- If the patient notices a strong unpleasant taste that gets worse over time then the patient probably has a mild Zinc deficiency.
- If the patient immediately notices a strong unpleasant or "metallic" taste then Zinc levels are probably sufficient.

Zinc supplementation of 30mg b.i.d. with food from a product that contains 1mg of copper for every 30mg of Zinc. Severe Zinc deficiency may require 60-90 days of supplementation and mild Zinc deficiency may require 30-60 days of supplementation. Recheck every 30 days.



# LACTATE DEHYDROGENASE (LDH)

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**Reference Range:** 0-225 IU/L

The LDH test is mainly used to identify acute or chronic tissue damage and has been traditionally used to diagnose damage to heart tissue related to myocardial infarction. LDH is an enzyme found in every tissue in the body so it can be utilized as a broad and specific diagnostic marker. Specificity is attained by ordering the LDH isoenzymes test which identifies what tissue is actually being damaged. The total LDH is the sum of all five enzymes with LDH-2 making up the highest percentage. These are broken down into five categories:

**LDH-1:** kidney, heart, and red cells

**LDH-2:** red blood cells, heart, and kidney (not as specific as LDH-1)

**LDH-3:** lungs & other tissues

**LDH-4:** lymph nodes, white blood cells, muscle, and liver (not as specific as LDH-5)

**LDH-5:** skeletal muscle, liver

LDH levels will rise 24-48 hours after a heart attack, then peak at 48-72 hours, and the return to normal after 10-14 days.

## RELATED TESTS:

Troponin, Myoglobin, Liver panel, Creatine kinase, Tumor markers, Cerebrospinal fluid analysis

## ELEVATED LDH LEVELS MAY INDICATE:

- Liver disease
- Kidney disease
- Pancreatitis
- Hemolytic anemia

- Mononucleosis
- Stroke
- Pernicious anemia
- Testicular cancer
- Lymphoma
- Multiple types of cancer
- Pneumonia
- Muscular dystrophy
- Lung or intestinal infarction
- Aspirin
- Narcotics
- Alcohol
- Strenuous exercise

Elevated platelets can present with a false elevation of LDH

**DECREASED LDH LEVELS MAY INDICATE:**

- Excessive vitamin C consumption

# ASPARTATE AMINOTRANSFERASE, AST (SGOT)

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**Reference Range:** 0-40 IU/L

AST is an enzyme found in many cells throughout the body with the majority of them found in the liver and heart and the minority found in the skeletal muscle and kidney. AST is released into the blood when there is damage to any of these tissues. AST along with ALT are mainly used to detect damage to liver cells but they can be useful for determining damage to other tissues as well. AST levels may rise to ten times their normal level during acute hepatitis and normally return to normal after approximately 2 months but may stay elevated for up to 6 months. In chronic hepatitis AST is elevated about four times the normal level but will fluctuate from normal to elevated. ALT will be higher than AST in the majority of liver pathologies with the exception of alcoholic hepatitis, the first 24-48 hours of acute hepatitis, cirrhosis and injury from bile duct obstruction.

## SIGN AND SYMPTOMS OF LIVER DISEASE MAY INCLUDE:

- Jaundice
- Fatigue
- Weakness
- Abdominal pain/swelling
- Nausea
- Vomiting
- Pruritis
- Dark-colored urine
- Light-colored stool
- Loss of appetite

## **RELATED TESTS:**

Total protein, ALT, GGT, Alkaline phosphatase, Bilirubin, Liver panel, Albumin, Hepatitis A,B,C screen, Copper, Ceruloplasmin, Creatine kinase, Amylase, Ferritin, Iron, Amino acid profile, Organic acids, Celiac disease/gluten-intolerance evaluation

## **ELEVATED LEVELS MAY INDICATE:**

- Systemic Inflammation
- Acute myocardial infarction
- Liver disease
- Skeletal Muscle breakdown/strenuous exercise
- Metastatic cancer
- Obesity
- Diabetes
- Hepatitis
- Alcoholism
- Medications
- Cirrhosis
- Skeletal muscle injections
- Acute pancreatitis
- Wilson's disease
- Hemochromatosis
- Glutathione depletion
- Celiac disease/gluten-intolerance

## **DECREASED LEVELS MAY INDICATE:**

- Pregnancy
- Vitamin B6 deficiency
- Protein deficiency
- Alcoholism
- Liver disease

## CLINICAL INDICATIONS:

The liver requires adequate amounts of amino acids to function properly so a dietary review of protein intake is paramount when there is dysfunction. A minimum of 1.2 grams of protein per kg bodyweight up to 2.0 grams per kilogram may be necessary to replenish amino acids levels. Protein supplementation in the form of protein powder, free-form amino acids and custom blended amino acid preparations can be useful in ensuring adequate protein and amino acid assimilation. A grip strength test via dynamometer is a good clinical indicator of protein malnutrition.

Glutathione is a key factor in optimal liver and detoxification function. Glutathione is a tripeptide comprised of the three amino acids l-cysteine, glycine and l-glutamic acid. Glutathione levels may be increased with the following interventions and nutraceuticals:

- Increase dietary protein intake
- Whey protein
- N-acetyl cysteine
- Selenomethionine
- Alpha Lipoic Acid
- Reduced Glutathione
- Vitamin C
- Quercetin
- Milk thistle
- Free-form amino acids
- S-AdoMet
- Melatonin
- D-ribose-l-cysteine
- 5-methyltetrahydrofolate
- Pyridoxal-5-phosphate
- Methylcobalamin
- 30 minutes of daily exercise
- Increasing sulfur-rich foods (see GGT)



# ALT (SGPT) ALANINE AMINOTRANSFERASE

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**Reference Range:** 0-44 IU/L

This is a test mainly used to identify types of liver disease. The information on AST is virtually identical to ALT with a few exceptions. ALT is more specific to the liver than AST. In most types of liver disease ALT is going to be higher than AST. The AST will be higher in cirrhosis, alcoholic hepatitis and muscle injury.

See AST for pertinent information.



# GGT

## (GAMMA GLUTAMYL-TRANSFERASE)

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**Reference Range:** 0-65 IU/L

GGT is used to diagnose liver disease or damaged bile ducts and to help differentiate liver from bone disease when the alkaline phosphatase is elevated. GGT is mainly found in the liver but also present in the pancreas, kidneys and spleen. GGT and ALP will be elevated in liver disease but only ALP will be elevated in bone disease. GGT alone is a controversial test because it is non-specific thereby limiting its diagnostic usefulness. Symptoms of elevated GGT are the same as under ALT and AST. GGT levels are higher in men but levels in women increase with age. GGT levels are very sensitive and may increase with only a minor amount of alcohol when other liver markers are normal.

### RELATED TESTS:

Liver panel, Bilirubin, AST, ALT, ALP, Hepatitis screen, Amino acids

### ELEVATED LEVELS MAY INDICATE:

- Liver disease
- Alcohol consumption
- Bile duct obstruction/damage
- Congestive heart failure
- Testosterone replacement
- Smoking
- NSAIDS and other drugs
- Increased Glutathione Demand

### DECREASED LEVELS MAY INDICATE:

- Low GGT with elevated ALP is most likely bone disease
- Oral contraceptives

### CLINICAL INDICATIONS:

Elevated GGT may indicate the need for more glutathione which is the body's most abundant antioxidant. Glutathione is necessary for proper detoxification but it can be depleted from chronic infections, protein deficiency, metal/environmental toxicity, chronic inflammation, medications, stress and autoimmune diseases. The goal is to identify the underlying cause of the depletion and supporting glutathione levels as well. Increasing dietary protein intake and 30 minutes a day of exercise can increase glutathione levels. The patient can be instructed to increase the consumption of sulfur-rich foods including:

- Wild seafood
- Red meats (buffalo, grass-fed beef, lamb)
- Turkey & chicken (pasture raised)
- Eggs
- Oats
- Garlic, onions, and all of the allium family
- Corn
- Chocolate
- Legumes (lentils, peas, beans)
- Nuts & seeds
- Broccoli and all brassica vegetables (kale, Brussels sprouts, cabbage)
- Asparagus
- Avocado (high in glutathione, which breaks down during digestion, yielding cysteine)
- Watermelon (also high in glutathione)
- Swiss Chard
- Parsley
- Sweet potatoes and "yams" (American yams, Genus Ipomoea, not Dioscorea, which the rest of the world calls "yams")

- Bananas
- Coconut
- Tomatoes
- Dairy products ( raw goat & cow)

**NUTRACEUTICALS THAT MAY RAISE GLUTATHIONE:**

- Whey protein
- Selenomethionine
- Alpha lipoic acid
- Milk thistle
- N-acetyl cysteine
- 5-methyltetrahydrofolate
- Methylcobalamin
- Pyridoxal-5-phosphate
- Ascorbic acid
- Quercetin
- Mixed tocopherols
- Free-form amino acids
- SAMe
- Melatonin
- D-ribose-l-cysteine

Glutathione can be taken in a reduced form orally to increase levels and improve immune system function.



# TOTAL IRON BINDING CAPACITY (TIBC)

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**Reference Range:** 250-450 ug/dL

This test measures the total amount of iron bound to protein in the blood. Transferrin is the primary iron-binding protein in the body thus TIBC is an excellent indirect measurement of this protein. The UIBC is often tested along with TIBC which is a marker of how much transferrin is not yet saturated with iron. Serum iron is also run along side TIBC which is used to calculate the transferrin saturation. These markers give the best indicator of iron status in the body along with ferritin.

When iron levels are low, the serum iron will be low, the TIBC will increase in an attempt to bind more available iron, and the % saturation will be decreased. When iron levels are high, such as in hemochromatosis, serum iron will be elevated, the TIBC will be low because the iron-binding proteins are saturated and there is no room left for binding, and the % saturation will be high for obvious reasons.

Transferrin can be ordered instead of TIBC and UIBC as an indicator of liver function and nutritional status. Transferrin will be low in liver disease and protein malnutrition. Chronic inflammatory processes such as infection and malignancy can cause elevations in transferrin.

See serum iron for more detailed information on iron evaluation.



# SERUM IRON

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**Reference Range:** 40-155 ug/dL

Iron is a trace element important for the formation of hemoglobin, myoglobin and cellular enzymes. Hemoglobin carries oxygen to all body tissues thus iron is an extremely essential nutrient. The average American ingests approximately 6-15 mg of iron each day from food. 2 mg is required on average to replace iron loss from menstruation, excretion and pregnancy. The most highly absorbable form of iron is ferrous iron mainly found in animal food sources. The plant-based ferric iron can be reduced to the ferrous form if there is adequate hydrochloric acid levels in the stomach. Iron absorption is enhanced by ascorbic acid whereas phytic acid from grains and phosphorous inhibits the absorption. In pyridoxine(B6) deficiency, the rate of iron absorption increases to the point of excess. Iron is transported by beta globulins called transferrin made by the liver to the bone marrow, liver and other body tissues. Approximately 20-25 mg of iron is released into the blood daily from the degradation of hemoglobin which the body recycles and uses over and over. 20% of total body iron is stored as ferritin and hemosiderin in the bone marrow, liver and spleen. The body can draw iron from this pool when needed for hemoglobin synthesis. Another 20% of total body iron is in the form of myoglobin and the remaining 50-60% is in the form of hemoglobin. Approximately 1mg of iron is lost daily in the feces, urine and sweat. Menstruation can result in 16-32 mg of iron loss each month. Anemic patients will have decreased oxygen concentrations in the intestine which leads to increased iron absorption.

Hemochromatosis is a condition of increased iron absorption resulting in elevated serum iron levels, ferritin and saturation of plasma transferrin. The excess iron levels result in spillage into various body tissues such as the brain, heart, liver, pancreas, skin and endocrine glands. Liver biopsy can confirm iron deposits in the tissue above normal. Hemosiderosis will present with iron deposits found only in the reticuloendothelial tissue.

In iron-deficiency anemia, serum iron levels will drop and iron-binding capacity will increase in an attempt to make hemoglobin. Gastrointestinal bleeding, pregnancy, the growing child or when the intake does not match excretion of iron can result in anemia.

**SYMPTOMS OF IRON-DEFICIENCY:**

- Fatigue
- Weakness
- Dizziness
- Headache
- Shortness of breath
- Chest pain
- Leg pain
- Pica: Cravings for chalk, dirt, clay, licorice
- Burning tongue
- Spoon nails
- Mouth sores
- Cognitive deficits

**SYMPTOMS OF EXCESS IRON:**

- Fatigue
- Abdominal pain
- Cognitive deficits
- Low sex drive
- Heart problems
- Weakness
- Joint pain

**RELATED TESTS:**

Ferritin, TIBC, UIBC, Transferrin, Zinc, Ceruloplasmin, CBC

**CAUSES OF ELEVATED SERUM IRON INCLUDE:**

- Lead poisoning
- Iron injections
- Blood transfusions
- Liver & Kidney disease
- Hemochromatosis
- Oral contraceptives
- Alcohol
- Iron-rich foods
- Testosterone
- Excessive vitamin C intake

**CAUSES OF DECREASED SERUM IRON INCLUDE:**

- Testosterone
- Metformin
- Aspirin
- ACTH
- Stress
- Sleep deprivation
- Celiac disease/malabsorption
- Hydrochloric acid deficiency (Hypothyroidism, H. Pylori, Zinc and B-vitamin deficiency)
- Tea
- Coffee

**Reference Range TIBC:** 250-450 ug/dL

**Reference Range UIBC:** 150-375 ug/dL

**Reference Range % Saturation:** 15-55%

DISEASE	IRON	TIBC/ TRANSFERRIN	UIBC	%TRANSFERRIN SATURATION	FERRITIN
<b>Iron deficiency</b>	Low	High	High	Low	Low
<b>Hemo- chromatosis</b>	High	Low	Low	High	High
<b>Chronic Illness</b>	Low	Low	Low/Normal	Low	Normal/High
<b>Hemolytic Anemia</b>	High	Normal/Low	Low/Normal	High	High
<b>Sideroblastic Anemia</b>	Normal/ High	Normal/Low	Low/Normal	High	High
<b>Iron Poisoning</b>	High	Normal	Low	High	Normal

## CLINICAL INDICATIONS:

Decreased iron levels may indicate a serious pathology causing internal bleeding and must be thoroughly evaluated immediately. Referral to a gastroenterologist for evaluation is often warranted.

Elevated iron levels require referral to a hematologist or gastroenterologist for evaluation as well. Therapeutic phlebotomies and blood donations can bring down iron stores effectively. Once iron and ferritin levels are balanced, careful monitoring is required. If iron and ferritin levels are extremely low, referral to a hematologist for intravenous iron infusions can quickly and effectively return ferritin levels to a healthy range. Iron supplementation may be required when ferritin levels are below normal.

Curcumin and green tea have been shown to be excellent iron chelators.

The thyroid is dependent on iron for manufacturing thyroid hormone and cases of hypothyroidism are often seen in patients with low ferritin levels. Iron restoration can in some cases return thyroid function to normal.

Meat and eggs provide the most bioavailable (ferrous) form of iron.

Plant-based sources (ferric) include green leafy vegetables, whole grains, wheat germ, molasses, raisins.

Iron supplementation may be necessary to increase iron stores. It is recommended to take iron with food and vitamin C to enhance absorption. Alcohol, beta-carotene, HCL and sugar all increase iron absorption. Phytates from food can inhibit absorption and should be avoided during supplementation unless they are soaked or sprouted.

The clinician must carefully decide if iron supplementation will exacerbate the patient's current condition due to the increase in oxidative stress that iron can create. If the patient is severely inflamed then it may be more prudent to address other issues for a period of time until they are more likely to assimilate and utilize the iron more effectively. Patients can tell immediately if the iron is doing more harm than good when they report brain fog, fatigue, blurry vision and headache upon commencing iron supplementation.

## SUBSTANCES THAT IMPAIR IRON ABSORPTION:

**Medications** that reduce the amount of acid in the stomach such as antacids or proton pump inhibitors can lead to hypochlorhydria (low stomach acid) or achlorhydria which is the complete absence of stomach acid.

**Calcium** (like iron) is an essential mineral, which means the body gets this nutrient from diet. Calcium is found in foods such as milk, yogurt, cheese, sardines, canned salmon, tofu, broccoli, almonds, figs, turnip greens and rhubarb and is the only known substance to inhibit absorption of both non-heme and heme iron. Where 50 milligrams or less of calcium has little if any effect on iron absorption, calcium in amounts 300-600 milligrams inhibit the absorption of heme iron similarly to nonheme iron. One cup of skimmed milk contains about 300 milligrams of calcium. When calcium is recommended by a healthcare provider, as is often the case for women trying to prevent bone loss, these supplements can be taken at bedtime. Calcium supplements are best taken with vitamin D and in a citrate rather than carbonate form.

**Eggs** contain a compound that impairs absorption of iron. Phosphoprotein called phosvitin is a protein with a iron binding capacity that may be responsible for the low bioavailability of iron from eggs. This iron inhibiting characteristic of eggs is called the "egg factor". The egg factor has been observed in several separate studies. One boiled egg can reduce absorption of iron in a meal by as much as 28%

**Oxalates** impair the absorption of nonheme iron. Oxalates are compounds derived from oxalic acid and found in foods such as spinach, kale, beets, nuts, chocolate, tea, wheat bran, rhubarb, strawberries and herbs such as oregano, basil, and parsley. The presence of oxalates in spinach explains why the iron in spinach is not absorbed. In fact, it is reported that the iron from spinach that does get absorbed is probably from the minute particles of sand or dirt clinging to the plant rather than the iron contained in the plant.

**Polyphenols** are major inhibitors of iron absorption. Polyphenols or phenolic compounds include chlorogenic acid found in cocoa, coffee and some herbs. Phenolic acid found in apples, peppermint and some herbal teas, and tannins found in black teas, coffee, cocoa, spices, walnuts, fruits such as apples, blackberries, raspberries and blueberries all have the ability to inhibit iron absorption. Of the polyphenols, Swedish cocoa and certain teas demonstrate the most powerful iron absorption inhibiting capabilities, in some cases up to 90%. Coffee is high in tannin and chlorogenic acid; one cup of certain types of coffee can inhibit iron absorption by as much as 60%. These foods or substance should not be consumed within two hours prior to and following your main iron-rich meal

**Phytate** is a compound contained in soy protein and fiber. Even low levels of phytate (about 5 percent of the amounts in cereal whole flours) have a strong inhibitory effect on iron bioavailability. Phytate is found in walnuts, almonds, sesame, dried beans, lentils and peas, and cereals and whole grains. Phytate compounds can reduce iron absorption by 50 to 65 percent.

# FERRITIN

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**Reference Range:** 30-400 ng/mL

This test measures the amount of iron stored in the body. Ferritin is the primary iron-containing protein in the body stored inside cells. Approximately 30% of the iron in the body is stored as ferritin or hemosiderin, and 70% is found in hemoglobin in red blood cells. Ferritin and hemosiderin are mainly found in the liver, bone marrow, skeletal muscle and spleen. The iron in red blood cells is normally sufficient to carry on normal metabolic processes, but when the need increases, iron is pulled from storage. Excessive bleeding, malabsorption, blood donations and inflammation can lead to depletion of iron. Too much iron intake from supplements or food can increase iron levels to an abnormal state resulting in iron deposits in various tissues throughout the body. This can occur in the genetic condition of iron-overload known as hemochromatosis. Ferritin can also increase in cases of malignancy, autoimmune disease, inflammation, excessive alcohol intake, hepatitis, liver disease, and infection. Any form of damage to the bone marrow, liver or spleen can lead to increases of ferritin in the blood.

## SYMPTOMS OF IRON-DEFICIENCY:

- Fatigue
- Weakness
- Dizziness
- Headache
- Shortness of breath
- Chest pain
- Leg pain
- Pica: Cravings for chalk, dirt, clay, licorice
- Burning tongue
- Spoon nails

- Mouth sores
- Cognitive deficits

### **SYMPTOMS OF EXCESS IRON:**

- Fatigue
- Abdominal pain
- Cognitive deficits
- Low sex drive
- Heart problems
- Weakness
- Joint pain
- Jaundice

### **RELATED TESTS:**

Serum Iron, TIBC, UIBC, Transferrin, Zinc, Ceruloplasmin, CBC, CMP, CRP, ESR

### **CAUSES OF ELEVATED FERRITIN:**

- Infection/inflammation
- Lead poisoning
- Iron injections
- Blood transfusions
- Liver & Kidney disease
- Hemochromatosis
- Oral contraceptives
- Alcohol
- Iron-rich foods
- Testosterone
- Excessive vitamin C intake

**CAUSES OF DECREASED FERRITIN:**

- Internal bleeding
- Blood donations
- Inflammation
- Testosterone replacement
- Metformin
- Aspirin
- ACTH
- Stress
- Sleep deprivation
- Celiac disease/malabsorption
- Hydrochloric acid deficiency (hypothyroidism, H. Pylori, Zinc and B-vitamin deficiency)
- Tea
- Coffee
- Excessive intake of phytates

DISEASE	IRON	TIBC/ TRANSFERRIN	UIBC	%TRANSFERRIN SATURATION	FERRITIN
<b>Iron deficiency</b>	Low	High	High	Low	Low
<b>Hemo- chromatosis</b>	High	Low	Low	High	High
<b>Chronic Illness</b>	Low	Low	Low/Normal	Low	Normal/High
<b>Hemolytic Anemia</b>	High	Normal/Low	Low/Normal	High	High
<b>Sideroblastic Anemia</b>	Normal/ High	Normal/Low	Low/Normal	High	High
<b>Iron Poisoning</b>	High	Normal	Low	High	Normal

## CLINICAL INDICATIONS:

Decreased ferritin levels may indicate a serious pathology causing internal bleeding and must be thoroughly evaluated immediately. Referral to a gastroenterologist for evaluation is often warranted.

Elevated ferritin levels require referral to a hematologist or gastroenterologist for evaluation as well. Therapeutic phlebotomies and blood donations can bring down iron stores effectively. Once iron and ferritin levels are balanced, careful monitoring is required. If iron and ferritin levels are extremely low, referral to a hematologist for intravenous iron infusions can quickly and effectively return ferritin levels to a healthy range. Iron supplementation may be required when ferritin levels are below normal.

The thyroid is dependent on iron for manufacturing thyroid hormone and cases of hypothyroidism are often seen in patients with low ferritin levels. Iron restoration can in some cases return thyroid function to normal.

Meat and eggs provide the most bioavailable (ferrous) form of iron.

Plant-based sources (ferric) include green leafy vegetables, whole grains, wheat germ, molasses, raisins.

Iron supplementation may be necessary to increase iron stores. It is recommended to take iron with food and vitamin C to enhance absorption. Phytates from grains can inhibit absorption and should be avoided during supplementation unless they are soaked or sprouted.

The clinician must carefully decide if iron supplementation will exacerbate the patient's current condition due to the increase in oxidative stress that iron can create. If the patient is severely inflamed then it may be more prudent to address other issues for a period of time until they are more likely to assimilate and utilize the iron more effectively. Patients can tell immediately if the iron is doing more harm than good when they report brain fog, fatigue, blurry vision and headache upon commencing iron supplementation.

# CHOLESTEROL

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**Reference Range:** 100-199 mg/dL

Cholesterol is an antioxidant, steroid and precursor to sex & adrenal hormones. Cholesterol has a similar molecular profile to vitamin D, bile acids and corticosteroids. Cholesterol makes up the cell membranes of every tissue and cell in the body. Bile acids are made from cholesterol which are important for fat emulsification and absorption of nutrients. Various lipoproteins transport cholesterol in the blood such as HDL and LDL. HDL was once thought to be “good” cholesterol and LDL was once thought to be “bad” cholesterol. Elevations in cholesterol and lipoproteins indicate a physiological response to something thus naming one “bad” or “good” is erroneous. Total cholesterol is a total measurement of the lipoproteins combined. Approximately 90% of this measurement indicates the amount of cholesterol made by the liver and 10% is from dietary intake. Foods high in saturated fat, cholesterol and trans fats can increase total cholesterol levels. Cholesterol can be laid down in blood vessels as plaques which may eventually occlude blood flow resulting in ischemia, myocardial infarction, stroke, and embolism. Atherosclerosis is hardening of the arteries as a result of excessive plaque and can be the precursor to heart disease. In addition to cholesterol absorption into the lymphatic system, it is also found in the adrenal cortex, intestinal mucosa, liver and genital organs.

The cholesterol test is mainly used as a risk factor for heart disease in conventional medicine. Integrative medicine clinicians view cholesterol as an indicator of various inflammatory and endocrine imbalances. Cholesterol levels and thyroid function are inversely proportional as an example.

Cholesterol below 150 is considered an ominous sign and may indicate a serious pathology such as carcinoma, liver disease and severe malnutrition.

The debate continues around cholesterol and its role in heart disease. Some studies show that higher cholesterol levels are actually protective and can increase lifespan. Alternatively, lower cholesterol levels have been linked to increased risk of heart murmur and heart disease. Due to the multitude of factors related to heart disease, isolation of cholesterol and its lipoproteins does not adequately explain cause and effect.

Cholesterol can be looked at in combination with triglycerides with an ideal ratio of 2:1.

## RELATED TESTS:

HDL, LDL, Lipid Profile, Cardiac C-reactive protein, Homocysteine, Triglycerides, Thyroid panel, VAP cholesterol test, Lipoprotein a, A1c, Glucose, Amylase, Pregnenelone, Cortisol, DHEA-sulfate, Sex hormones

## ELEVATED LEVELS MAY INDICATE:

- Insulin resistance
- Diabetes
- Metabolic Syndrome
- Fatty liver
- Hypothyroidism
- Acute biliary obstruction
- Pancreatitis
- Hypertension
- Oxidative Stress
- Genetics
- Cigarette Smoking
- Obesity
- Excessive Vitamin D Intake
- Oral Contraceptives
- Beta Blockers
- Epinephrine
- Anabolic Steroids
- Early Starvation
- Nephrotic Syndrome
- Obstructive Jaundice

**DECREASED LEVELS MAY INDICATE:**

- Chemical/metal toxicity
- Liver Disease (Portal Cirrhosis)
- Malnutrition
- Low dietary carbohydrates
- Viral hepatitis
- Hyperthyroidism
- Carcinoma
- Niacin supplementation
- Red yeast rice supplementation
- Idiopathic Steatorrhea
- Acanthocytosis
- Statin medications

**CLINICAL INDICATIONS FOR CHOLESTEROL IMBALANCES:**

Cholesterol levels can be lowered through dietary changes, exercise and supplementation with red yeast rice, niacin, green tea extract and inositol. Lowering cholesterol however does not address the underlying causes of the cholesterol imbalance. Thyroid function should be thoroughly evaluated through blood chemistries and basal body temperature. Basal body temperature can be done first thing in the morning before moving around and plotted on a graph for about 7-10 days. Minimum ideal temperature can be around 97.8 degrees Fahrenheit. A consistently low temperature may indicate a thyroid-specific issue whereas peaks and valleys may indicate adrenal dysfunction or autoimmunity influencing thyroid function.

Decreasing simple sugars and carbohydrates and/or high-fat foods may reduce cholesterol levels. When exercise is implemented and proper dietary changes are made but cholesterol levels stay elevated, investigation for a chronic inflammatory process and/or insulin resistance may be required. Genetic influences may result in cholesterol levels that will not change no matter what treatment plan is implemented. Since cholesterol is an antioxidant the question must be asked why the liver is producing large amounts of antioxidants and therefore the investigation ensues.

All of the sex hormones including testosterone, estrogens, and progesterone as well as cortisol and DHEA are manufactured from cholesterol. Deficiencies and imbalances in these levels may result in cholesterol level shifts and must therefore be investigated as a related factor.



# TRIGLYCERIDES

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**Reference Range:** 0-149 mg/dL

Triglycerides are part of the lipid profile used to assess cardiovascular risk function. They are a form of fat stored mainly in adipose tissue and used by the body to fuel metabolic activity in muscle tissue. Triglycerides can be released from adipose tissue in between meals for energy. They are transported mainly by VLDL. Triglyceride levels can increase up to 10 times fasting levels after a meal depending on its macronutrient content. Conventional advice to lower triglycerides is the same as that for elevated cholesterol. Triglycerides are emulsified by bile acids and hydrolyzed by pancreatic lipase. Triglycerides are mainly manufactured from carbohydrates, but protein and fats can also be a source. Insulin increases the production of triglycerides in the liver and adipose tissue.

## RELATED TESTS:

Lipid profile, Cholesterol, HDL, LDL, Cardiovascular risk assessment markers, Thyroid panel

## ELEVATED LEVELS MAY INDICATE:

- Insulin resistance
- Diabetes
- Fatty liver
- Hypothyroidism
- Kidney Disease (Nephrotic Syndrome)
- Cigarette Smoking
- Alcohol
- Lack of Exercise
- Obesity
- Genetics

- Pancreatitis can occur when levels are >1,000
- Estrogen replacement
- Corticosteroids
- Beta blockers
- Early Starvation
- Obstructive Jaundice
- Low-sodium diet

**DECREASED LEVELS MAY INDICATE:**

- Oxidative Stress
- Chemical/metal toxicity
- Liver dysfunction (Portal Cirrhosis)
- Low dietary carbohydrates
- Malabsorption
- Idiopathic Steatorrhea
- Malnutrition
- Acanthocytosis

**CLINICAL INDICATIONS:**

The most common cause of elevated triglycerides is insulin resistance. Exercise of any form is the simplest and most effective way to reduce triglyceride levels. See the clinical recommendations under glucose for more details regarding dietary recommendations.

# HDL CHOLESTEROL

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**Reference Range:** >39 mg/dL

HDL is a lipoprotein comprised mainly of protein but also made of cholesterol and triglyceride. HDL scavenges cholesterol from body tissues and carries it back to the liver for disposal. This has earned it the title of “good cholesterol.” Higher levels of HDL have been purported to decrease risk of heart disease but recent findings suggest that this is too simplistic and that many other factors come into play. In fact, in some cases high levels of HDL actually increase the risk of heart disease due to various genetic factors.

A cholesterol to HDL ratio is sometimes reported with an ideal number of 3.5:1.

## RELATED TESTS:

Same as cholesterol

## ELEVATED LEVELS MAY INDICATE:

- Exercise
- Moderate Alcohol Consumption
- Fish oil consumption

## DECREASED LEVELS MAY INDICATE:

- Oxidative stress
- Chemical/metal toxicity
- Sedentary lifestyle
- Obesity
- Insulin resistance
- Fatty liver

- Starvation
- Diabetes
- Hypothyroidism
- Uremia
- Cigarette Smoking

# LDL

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**Reference Range:** 0-99 mg/dL

LDL (low density lipoprotein) is a lipoprotein comprised of protein, triglyceride and mainly cholesterol. LDL, in contrast to HDL, transports cholesterol and deposits it on blood vessel linings. It has been found to be connected with increased rates of heart disease and has thus earned the title of “bad cholesterol.” LDL acts as an antioxidant to quench endotoxins from a dysfunctional GI tract.

## RELATED TESTS:

Same as cholesterol

## ELEVATED LEVELS MAY INDICATE:

- Leaky gut
- Gut atrophy
- Diabetes
- Genetics
- Cigarette Smoking
- Low HDL
- Abnormal Weight Gain
- Oxidative Stress
- Insulin Resistance
- Hypothyroidism
- Hypertension
- Previous Coronary Artery Disease

**DECREASED LEVELS MAY INDICATE:**

- Infection
- Inflammation
- Hyperthyroidism
- Cirrhosis of the Liver

See clinical indications and nutraceuticals for cholesterol.

# VLDL

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**Reference Range:** 5-40 mg/dL

VLDL (very low density lipoprotein) is a lipoprotein comprised of protein, cholesterol and mainly triglyceride. VLDL eventually becomes LDL when it loses its triglyceride content. Like LDL, VLDL is said to have a connection with atherosclerosis and increase risk for heart disease. As VLDL levels increase, conversion to LDL actually slows down resulting in a build up of these particles.

Diagnostic data on VLDL is similar to that of LDL.

## VERTICAL AUTO PROFILE (VAP) CHOLESTEROL TEST

The VAP test provides a more detailed analysis of cholesterol which can be helpful in the prevention of cardiovascular disease. The VAP test includes the following:

More accurate, direct measurement of LDL.

Measurement of LDL pattern density. This is important because small, dense LDL (“Pattern B”) triples the likelihood of developing coronary plaque and suffering a heart attack.

Measurement of lipoprotein subclasses, which include HDL2 and HDL3, intermediate-density lipoprotein (IDL), very-low-density lipoproteins (VLDL1, VLDL2, VLDL3), and lipoprotein(a) [Lp(a)], a particularly dangerous lipoprotein that can lead to heart attacks and strokes.

**LDL:** Low-density lipoprotein; elevated levels are considered a primary cause of heart disease. LDL is the primary cholesterol target in heart disease risk management.

**HDL:** High-density lipoprotein; considered protective to the cardiovascular system. Low levels are associated with increased risk for coronary heart disease.

**VLDL:** Very-low-density lipoprotein; the main carrier for triglycerides. Elevated levels can be an independent risk factor for heart disease.

**Total Cholesterol:** The total amount of cholesterol circulating throughout your body.

**Triglycerides:** Energy-rich molecules needed for normal functions throughout the body. Elevated levels are associated with diabetes and cardiovascular disease.

**Non-HDL Cholesterol:** The sum of LDL and VLDL; elevated levels are a better predictor of heart disease risk than LDL alone.

**Lp(a):** Lipoprotein(a); an inherited risk factor for heart disease. It is more dangerous than other types of cholesterol, and does not respond to traditional LDL-lowering drugs.

**IDL:** Intermediate-density lipoprotein; an inherited, independent risk factor for heart disease. It is often elevated in patients with a family history of diabetes.

**Real LDL:** The “real” cholesterol circulating in your body, it is a component of LDL. Real LDL is calculated by subtracting Lp(a) and IDL from LDL.

**LDL Size Pattern:** LDL particles vary in size, ranging from small, dense “Pattern B” particles to large, buoyant “Pattern A” particles. Smaller LDL particles are associated with an increased risk for heart disease. Small, dense LDL (“Pattern B”) is associated with insulin resistance or diabetes.

**Metabolic Syndrome:** A condition characterized by a combination of several metabolic risk factors—including elevated triglycerides, low HDL, and small, dense “Pattern B” LDL particles—that increase the overall risk for heart disease.

**HDL2\ HDL3:** HDL subfractions are used to predict cardiovascular risk. HDL2 is large and buoyant, and is the most protective form of HDL. Low HDL2 with normal LDL is associated with cardiovascular risk. HDL3 is not as protective as HDL 2.

**VLDL3:** VLDL3 is the densest VLDL sub-fraction, and confers a greater risk factor for heart disease than both VLDL1 and VLDL2.

# C-REACTIVE PROTEIN

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**Reference Range:** 0.00-3.00 mg/L

C-reactive protein or CRP is used to measure inflammation in the body. CRP is an acute phase protein made in the liver in response to an inflammatory process such as an infection, trauma, autoimmune disease, inflammatory bowel, food sensitivity etc. It's release occurs about 2 hours after onset of the pathological process. This test can be used for both acute and chronic inflammatory processes. CRP is non-specific and therefore is not diagnostic for any particular disease process.

A standard CRP can be ordered or a highly sensitive CRP (hs-CRP) to measure inflammation. The difference between the two is in the amount of the protein actually measured in the blood. The hs-CRP is more sensitive by definition because it measures much smaller amounts of the acute phase protein than the standard test which measures gross amounts. The hs-CRP may be a better test for healthy individuals as it will pick up more subtle levels of chronic inflammation as a preventive measure.

## RELATED TESTS:

Urinalysis, Erythrocyte Sedimentation Rate, Rheumatoid Factor, ANA, Lipid panel and cardiovascular risk markers, CBC, Estrogens, Food sensitivities, Celiac panel

## ELEVATED LEVELS MAY INDICATE:

- Infection
- Inflammation
- Physical Trauma
- Autoimmune Disease: Lupus, Rheumatoid Arthritis etc.
- Vasculitis
- Pelvic Inflammatory Disease
- Arthritis

- Birth Control Pills
- Late-stage pregnancy
- Estrogen replacement
- Obesity

Decreased levels are not significant.

## **CLINICAL INDICATIONS:**

The most important aspect of elevated CRP is to identify the underlying cause of the inflammation. In the meantime, certain nutraceuticals have been shown to reduce inflammation. Due to the fact that all chronic illnesses have an inflammatory component, improvement in symptomatology can be rapid when inflammation is reduced.

## **THE FOLLOWING NUTRACEUTICALS MAY REDUCE INFLAMMATION:**

- Omega-3 fatty acids
- Curcumin
- Quercetin
- Resveratrol
- EGCG (Green tea extract)
- Boswellia
- Ginger
- Celery seed extract
- Bromelain
- Niacinamide
- MSM
- N-acetyl cysteine
- Rutin
- Rosemary
- Proteolytic enzymes

# THYROID STIMULATING HORMONE (TSH)

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**Reference Range:** 0.450-4.500 uIU/mL

TSH (Thyrotropin) is a hormone produced by the anterior pituitary which stimulates the production of thyroid hormone from the thyroid gland. The hypothalamus monitors levels of thyroid hormones and releases thyrotropin releasing hormone (TRH) to stimulate the anterior pituitary to release TSH. As thyroid function decreases, TSH levels will increase. If thyroid function is excessive, TSH levels will decrease. The TSH test is used as a diagnostic and screening test for thyroid function as well as in cases of infertility. The TSH test is highly controversial regarding its accuracy alone for evaluating thyroid function due to the many factors that can affect its levels. TSH levels can be “normal” on conventional laboratory tests but the patient can still have signs and symptoms of hypothyroidism.

## SIGNS & SYMPTOMS OF HYPERTHYROIDISM INCLUDE:

- Rapid heart rate
- Insomnia
- Eye irritation
- Weight loss
- Restlessness
- Increased body temperature
- Sweating
- Hand tremors
- Nervousness
- Weakness
- Diarrhea

- Light sensitivity
- Bulging eyes

**SIGNS & SYMPTOMS OF HYPOTHYROIDISM INCLUDE:**

- Fatigue
- Weight gain
- Depression
- Constipation
- Dry skin
- Dry and brittle nails
- Cold intolerance
- Cold hands and feet,
- Thinning/loss of the lateral 1/3 of the eyebrow
- Slow Achilles reflex
- Puffy skin
- Hair loss
- Menstrual irregularities

**RELATED TESTS:**

Thyroxine, Total T4, Total T3, Free T4, Free T3, Cortisol, Progesterone, Estrogen, Testosterone, DHEA, Thyroid peroxidase, Anti-thyroglobulin, Thyroglobulin

**ELEVATED LEVELS MAY INDICATE:**

- Hashimoto's Thyroiditis
- Hypothyroidism
- Iodine deficiency
- Mercury poisoning
- Addison's/Low Cortisol
- Hypothalamic dysfunction

- Pituitary tumor
- Elevated Reverse T3
- Extreme stress
- Excess estrogen
- Insulin resistance
- Inflammation
- Excess progesterone

**DECREASED LEVELS MAY INDICATE:**

- Graves' disease
- Hyperthyroidism
- Cushing's/Elevated Cortisol
- Hypothalamic dysfunction
- Excessive Thyroid Medication
- 1st Trimester of pregnancy
- Excessive testosterone
- Thyroid Nodule

TSH	T4	T3	INTERPRETATION
High	Normal	Normal	Mild (subclinical) hypothyroidism
High	Low	Low or normal	Hypothyroidism
Low	Normal	Normal	Mild (subclinical) hyperthyroidism
Low	High or normal	High or normal	Hyperthyroidism
Low	Low or normal	Low or normal	Non-thyroidal illness, pituitary hypothyroidism

## CLINICAL INDICATIONS:

When evaluating thyroid function it is first important to rule out autoimmunity by testing thyroid antibody levels. If the patient has hypothyroidism without autoimmunity then evaluation of the following should be performed based on history and clinical indications:

- Dietary protein intake evaluation through diet diary, body composition and grip strength
- Is the diet deficient in iodine?
- Adrenal gland evaluation of Cortisol and DHEA
- Blood sugar/insulin resistance by fasting glucose and A1c
- Gastrointestinal evaluation for dysbiosis
- Zinc taste test
- Ferritin
- Toxicological evaluation through history, direct lab testing and/or DMPS urine challenge test
- Markers of inflammation eg. Hs-CRP, Homocysteine, Fibrinogen, ESR
- Sex hormone evaluation
- Reverse T3
- Total T3
- Free T3
- T4
- Free T4
- TSH
- Thyroid ultrasound

## IF THYROID ANTIBODIES ARE PRESENT THEN THE FOLLOWING SHOULD BE EVALUATED BASED ON HISTORY AND CLINICAL INDICATIONS:

- History of excessive iodine intake
- Gastrointestinal pathogen screen for H. Pylori, Fungus, Yersinia, Dysbiosis, Bacterial infections
- Epstein-Barr Virus profile
- Yersinia enterocolitica blood test

- H. Pylori blood and/or breathe test
- MELISA metals test if indicated for mercury sensitivity
- Food sensitivities
- Lyme disease evaluation if indicated
- Hepatitis C
- Parvovirus B-19
- Rickettsia
- Toxicological history
- Vitamin D levels and receptor polymorphism
- Estrogen evaluation
- Celiac panel/gluten intolerance testing
- Gastrointestinal hyperpermeability
- Thyroid ultrasound

Basal body temperature testing can be done first thing in the morning without moving around and plotted on a graph. A consistently low temperature may indicate a true thyroid issue whereas an erratic pattern of many peaks and valleys may indicate adrenal gland dysfunction affecting the thyroid and/or autoimmunity. Basal body temperature is ideally 97.8 degrees when measuring axillary temperature and 98.2 degrees when measuring oral temperature as a minimum.

Some patients may benefit from T3 alone to “reset” the HPT axis and bypass any chance of reverse T3 elevations. Some of these patients may only need to take T3 for a short period of time such as 30-60 days to achieve this re-balancing procedure.

If TSH levels are suppressed without autoimmunity then an evaluation of adrenal function and causes of inflammation may be necessary.

### NUTRACEUTICALS FOR AUTOIMMUNITY:

- Selenomethionine
- N-acetyl cysteine
- Vitamin D
- Reishi mushroom extract

- Antioxidants C, E, CoQ10, A
- Bugleweed and Lemon balm can be helpful in Graves' disease
- L-carnitine can reduce symptoms in Graves' disease
- Zinc
- Probiotics

NUTRACEUTICALS FOR NON-AUTOIMMUNE HYPOTHYROIDISM:

- Iodine
- Zinc
- Magnesium
- Iron
- Ashwagandha
- Eleutherococcus
- B-complex
- Vitamin A
- Vitamin D
- Vitamin C
- Selenomethionine

# THYROXINE (T4)

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**Reference Range Total T4:** 4.5-12.0 ug/dL

**Reference Range Free T4:** 0.82-1.77 ng/dL

T4 is the major hormone (~90%) produced by the thyroid gland in response to TSH. It is manufactured from iodine and the amino acid l-tyrosine. When T4 levels drop, the hypothalamus signals the anterior pituitary via TRH to increase TSH production. When T4 levels increase, the opposite happens. T4 is inactive until it is converted into the active thyroid hormone T3 (triiodothyronine) mainly in the liver. T4 is protein-bound until it becomes free T4 which is the unbound form of T4 making up only .1% of total T4. Free T4 is the active form of T4 but free T3 is 5 times more active.

See TSH for clinical information and related tests.

## ELEVATED LEVELS MAY INDICATE:

- Hyperthyroidism
- Graves' disease
- Early stage Hashimoto's
- Pregnancy
- Levothyroxine intake

## DECREASED LEVELS MAY INDICATE:

- Hypothyroidism
- Hashimoto's Thyroiditis
- Iodine deficiency
- Cushing's/High Cortisol
- Mercury poisoning
- Zinc deficiency
- Iron-deficiency anemia
- Protein malnutrition



# TRIODOTHYRONINE (T3)

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**Reference Range Total T3:** 71-180 ng/dL

**Reference Range Free T3:** 2.0-4.4 pg/mL

**Reference Range Reverse T3:** 9.2-24.1 ng/dL

T3 is the active form of thyroid hormone comprising about 10% of thyroid hormone produced by the thyroid gland. T4 is converted into T3 mainly in the liver. Total T3 is the protein-bound portion of T3 comprising 99.7% of T3 and free T3 is the active unbound form. Free T3 is 5 times more active than free T4.

The T3 uptake test measures the amount of available thyroid hormone binding proteins in the blood. Excessive testosterone levels may increase the T3 uptake and excessive estrogen levels will decrease the T3 uptake.

Reverse T3 is an inactive form of T3 made from T4. Reverse T3 will elevate when the body is under any type of biochemical, physical or emotional stress. Reverse T3 can cause the symptoms of hypothyroidism when all other thyroid markers appear to be normal.

One method of thyroid analysis is evaluating the Total T3 to reverse T3 ratio. Total T3 is divided by reverse T3 and the ideal ratio is said to be between 10-14. In many chronically ill patients the ratio will be below 10 which may be an indicator for the use of T3 administration alone. T3 administration will bring down the reverse T3 and “reset” the hypothalamic-pituitary-thyroid axis. Administration of T4 when the reverse T3 is already high may increase the reverse T3 even more.

Patients can also measure their basal body temperature every morning plotted on a graph. If the measurements appear to be low but consistent, then this is most likely a true thyroid issue. If the measurements fluctuate up and down resulting in an erratic, unstable pattern, then the thyroid issue may be mainly caused by autoimmunity, stress and adrenal gland dysfunction.

See TSH for clinical signs, symptoms and related tests.

**ELEVATED LEVELS MAY INDICATE:**

- Hyperthyroidism
- Grave's disease

**DECREASED LEVELS MAY INDICATE:**

- Cushing's/High Cortisol
- Mercury poisoning
- Inflammation
- Insulin resistance
- Iodine deficiency
- Selenium deficiency
- Addison's/Low Cortisol
- Extreme stress/illness
- Sleep deprivation
- Pregnancy

# THYROID ANTIBODIES

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Thyroid Peroxidase (TPO), Antithyroglobulin (TGAb), Thyroid Stimulating Hormone Receptor Antibodies (TSHRAb).

Thyroid antibodies are performed to diagnose the autoimmune thyroid diseases Hashimoto's thyroiditis and Graves' disease. The immune system creates antibodies against thyroid tissue and enzymes which result in inflammation and destruction of the thyroid gland.

TPO antibodies are present in Hashimoto's and Graves' disease.

TGAb is present in Hashimoto's and thyroid carcinoma.

Thyroid Stimulating Immunoglobulin (TSI) is present in Grave's disease. It binds to receptors and promotes thyroid hormone production.

Thyroid binding inhibitory immunoglobulin (TBII) binds to TSH receptors and prevents thyroid hormone production resulting in hypothyroidism. This test is rarely utilized.

A small percentage of patients with autoimmune thyroiditis do not make antibodies so a negative test does not rule out autoimmunity. A negative test may also simply mean that the antibodies were not present in the blood at the time of the test. Thyroid antibodies may also be elevated in autoimmune collagen vascular disease, Type 1 diabetes, rheumatoid arthritis, lupus, Sjogren's, thyroid cancer and pernicious anemia. Certain individuals will have elevated antibody levels but no thyroid issues. These tests are still not as accurate and reliable as they should be thus a good history and clinical signs and symptoms are extremely important.



# COMPLETE BLOOD COUNT/CBC WITH DIFFERENTIAL

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The CBC is used to diagnose blood disorders such as cancer, infection, inflammation, and anemia. White blood cells, red blood cells and platelets are the three types of cells in blood plasma and are measured in the CBC. These cells are produced and mature in the bone marrow. The CBC includes the white blood cell count preferably with differential, red blood cell count, hemoglobin, hematocrit, red cell distribution width, mean corpuscular volume, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, reticulocyte count and platelets.

White blood cells fight infection and repair damaged tissue and include neutrophils, lymphocytes, monocytes, eosinophils and basophils. Shifts in these cells indicate infection from virus, bacteria, fungus or parasite as well as allergies and blood cancers like leukemia.

Red blood cells carry oxygen through the blood via hemoglobin and have a lifespan of 120 days. Shifts in the number of RBC's and their size can indicate anemia and polycythemia. Anemia can result from thyroid disorders, inflammation, vitamin B12 and folate deficiencies, iron deficiency and a variety of other conditions.

Platelets are involved in blood clotting and too little platelet counts can result in excessive bleeding and bruising.

## **White Blood Cell Count (WBC)**

**Reference Range WBC:** 4.0-10.5 x10E3/uL

**Reference Range Neutrophils:** 40-74%

**Reference Range Lymphocytes:** 14-46%

**Reference Range Monocytes:** 4-13%

**Reference Range Eosinophils:** 0-7%

**Reference Range Basophils:** 0-3%

The WBC is part of the CBC and is used to diagnose infection, inflammation, allergies and cancer. WBC's also known as leukocytes are found in the blood, lymphatic system and body tissues. The WBC count is the total number of neutrophils, lymphocytes, monocytes, eosinophils and basophils. Neutrophils, eosinophils and basophils are classified as granulocytes whereas lymphocytes and monocytes are classified as agranulocytes. Lymphocytes are further broken down into B lymphocytes (antibody-producing), T lymphocytes and natural killer cells. When there is an infection for example, the bone marrow releases extra white blood cells to combat the infectious agent.

## **RELATED TESTS:**

Blood smear, ANA, Specific autoimmune disease markers, Food sensitivities, Stool analysis, Cortisol, DHEA-sulfate, Creatinine, Creatine kinase, Methylmalonic acid, Ferritin, Serum iron, TIBC, DMPS urine challenge, Specific infectious disease tests, MTHFR

## **ELEVATED WHITE BLOOD CELL COUNT (LEUKOCYTOSIS) MAY INDICATE:**

- Infection
- Inflammation
- Allergies
- Autoimmune disease
- Pregnancy
- Severe stress
- Excessive exercise
- Asthma
- Trauma
- Burns
- Surgery
- Heart Attack
- Leukemia
- Vasculitis
- Inflammatory bowel
- Myeloproliferative neoplasm

**DECREASED WHITE BLOOD CELL COUNT (LEUKOPENIA) MAY INDICATE:**

- HIV
- Bone Marrow Disorders
- Vitamin B12 deficiency
- Folate deficiency
- Chronic infection
- Heavy metal toxicity
- Autoimmune disease
- Lymphoma
- Myelodysplastic syndrome
- Chemotherapy
- Radiation
- Certain medications
- Sepsis

**WHITE BLOOD CELL EVALUATION:**

<b>Neutrophils</b>	Low Neutrophils (Neutropenia)	Elevated Neutrophils (Neutrophilia)
	<ul style="list-style-type: none"> <li>• Cancer of the bone marrow</li> <li>• Immunodeficiency</li> <li>• Autoimmune disease</li> <li>• Overwhelming infection (sepsis)</li> <li>• Chemotherapy</li> <li>• Viral Infection</li> <li>• Myelodysplasia</li> <li>• Radiation</li> <li>• Medications</li> </ul>	<ul style="list-style-type: none"> <li>• Acute bacterial infections</li> <li>• Chronic myeloid leukemia</li> <li>• Inflammation</li> <li>• Trauma</li> <li>• Heart attack</li> <li>• Burns</li> <li>• Stress</li> <li>• Excessive exercise</li> </ul>

<b>Lymphocytes</b>	<p>Low Lymphocytes (Lymphocytopenia)</p> <ul style="list-style-type: none"> <li>• Bacterial infections</li> <li>• Autoimmune disease</li> <li>• HIV</li> <li>• Viral hepatitis</li> <li>• Influenza</li> <li>• Chemotherapy</li> <li>• Radiation</li> <li>• Corticosteroids</li> </ul>	<p>Elevated Lymphocytes (Lymphocytosis)</p> <ul style="list-style-type: none"> <li>• Viral infections</li> <li>• Stress</li> <li>• Toxoplasmosis</li> <li>• Chronic inflammation</li> <li>• Lymphocytic leukemia</li> <li>• Lymphoma</li> <li>• Pertussis</li> <li>• Tuberculosis</li> </ul>
<b>Monocytes</b>	<p>Low Monocytes</p> <ul style="list-style-type: none"> <li>• Bone marrow damage</li> <li>• Hairy cell leukemia</li> </ul>	<p>Elevated Monocytes</p> <ul style="list-style-type: none"> <li>• Chronic infections</li> <li>• Monocytic leukemia</li> <li>• Myelomonocytic leukemia</li> <li>• Collagen vascular disease: rheumatoid arthritis, vasculitis, lupus, scleroderma</li> <li>• Parasitic infections</li> </ul>
<b>Eosinophils</b>	<p>Low Eosinophils is not clinically significant</p>	<p>Elevated Eosinophils</p> <ul style="list-style-type: none"> <li>• Allergies</li> <li>• Parasitic infections</li> <li>• Fungal infections</li> <li>• Leukemia</li> <li>• Lymphoma</li> <li>• Celiac disease</li> <li>• Inflammatory bowel</li> <li>• Chronic inflammation</li> <li>• Drug reactions</li> <li>• Asthma</li> </ul>
<b>Basophils</b>	<p>Low Basophils is not clinically significant</p>	<p>Elevated Basophils</p> <ul style="list-style-type: none"> <li>• Leukemia</li> <li>• Inflammation</li> <li>• Allergies</li> </ul>

## RED BLOOD CELL EVALUATION

**Reference Range RBC:** 4.14-5.8 x10E6/uL

**Reference Range Hemoglobin:** 12.6-17.7 g/dL

**Reference Range Hematocrit:** 37.5-51%

**Reference Range MCV:** 79-97 fL

**Reference Range MCH:** 26.6-33 pg

**Reference Range MCHC:** 31.5-35.7 g/dL

**Reference Range RDW:** 12.3-15.4%

<b>Red Blood Cell Count</b>	Low RBC's (Anemia) <ul style="list-style-type: none"> <li>• Anemia</li> <li>• Kidney failure</li> <li>• Chronic inflammation</li> <li>• Bleeding</li> <li>• RBC destruction (Babesia)</li> <li>• Iron deficiency</li> <li>• B12 &amp; Folate deficiency</li> <li>• Bone marrow disorder</li> </ul>	Elevated RBC's (polycythemia) <ul style="list-style-type: none"> <li>• Dehydration</li> <li>• Polycythemia vera</li> <li>• Lung disease</li> <li>• Smoking</li> <li>• Kidney tumor (excess erythropoietin)</li> <li>• Genetic causes</li> </ul>
<b>Hemoglobin</b>	Low Hemoglobin same as RBC	Same as RBC
<b>Hematocrit</b>	Low Hematocrit same as RBC	Same as RBC
<b>Mean Corpuscular Volume (MCV)</b>	Low MCV <ul style="list-style-type: none"> <li>• Small red blood cells (microcytic)</li> <li>• Iron deficiency</li> <li>• Thalassemias</li> </ul>	Elevated MCV <ul style="list-style-type: none"> <li>• RBC's are larger than normal (macrocytic)</li> <li>• B12 or Folate deficiency</li> <li>• Hypothyroidism</li> </ul>
<b>Mean Corpuscular Hemoglobin (MCH)</b>	Same as MCV	Same as MCV

<p><b>Mean Corpuscular Hemoglobin Concentration (MCHC)</b></p>	<p>Same as MCV</p>	<p>Elevated MCHC</p> <ul style="list-style-type: none"> <li>• Autoimmune hemolytic anemia</li> <li>• Burns</li> <li>• Spherocytosis</li> </ul>
<p><b>RBD Distribution Width (RDW)</b></p> <ul style="list-style-type: none"> <li>• RDW helps differentiate mixed anemias such as Iron and Bvitamin deficiencies when MCV is normal.</li> </ul>	<p>Low RDW indicates uniformity in size of RBCs</p>	<p>Elevated RDW</p> <ul style="list-style-type: none"> <li>• Selenium deficiency</li> <li>• Inflammation</li> <li>• Iron deficiency anemia</li> <li>• Pernicious anemia</li> <li>• Poikilocytosis</li> </ul>
<p><b>Reticulocyte Count</b></p>	<p>Low Reticulocyte Count</p> <ul style="list-style-type: none"> <li>• Bone marrow disorder</li> <li>• Iron, B12 or Folate deficiency</li> </ul>	<p>Elevated Reticulocyte Count</p> <ul style="list-style-type: none"> <li>• Iron supplementation</li> <li>• Hemolysis</li> <li>• Bleeding</li> </ul>

## PLATELET EVALUATION

**Reference Range Platelet Count:** 140-415 x10E3/uL

<b>Platelet Count</b>	<b>Low Platelets (Thrombocytopenia)</b> <ul style="list-style-type: none"> <li>• Viral infections</li> <li>• Chemotherapy</li> <li>• Radiation</li> <li>• Rickettsial infections</li> <li>• Platelet autoantibodies</li> <li>• Drugs</li> <li>• Myelodysplasia</li> <li>• Cirrhosis</li> <li>• Autoimmune disease</li> <li>• Sepsis</li> <li>• Leukemia</li> <li>• Lymphoma</li> </ul>	<b>Elevated Platelets (Thrombocytosis)</b> <ul style="list-style-type: none"> <li>• Iron deficiency anemia</li> <li>• Hemolytic anemia</li> <li>• Cancers</li> <li>• Rheumatoid arthritis</li> <li>• Lupus</li> <li>• Inflammatory bowel</li> <li>• Myeloproliferative disorders</li> </ul>
<b>Mean Platelet Volume (MPV)</b>	<b>Low MPV indicates small platelet size</b> <ul style="list-style-type: none"> <li>• High number of old platelets</li> <li>• Bone marrow disorders</li> </ul>	<b>Elevated MPV indicates large platelet size</b> <ul style="list-style-type: none"> <li>• High number of young platelets</li> <li>• Bone marrow disorder</li> </ul>
<b>Platelet Distribution Width (PDW)</b>	<b>Low PDW indicates uniformity in size of platelets</b>	<b>Elevated PDW</b> <ul style="list-style-type: none"> <li>• Variable platelet size indicates conditions affecting production</li> </ul>



# VITAMIN D

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**Reference Range:** 30-100 ng/mL

The vitamin D test is used to identify vitamin D deficiency, monitor vitamin D therapy and identify causes of imbalanced calcium, magnesium, phosphorous and/or parathyroid hormone. Vitamin D was misnamed a vitamin and is actually a hormone. Two types of vitamin D tests exist including 25-hydroxyvitamin D and 1,25-hydroxyvitamin D. 25-hydroxyvitamin D is inactive and the precursor to the active form 1,25-hydroxyvitamin D (calcitriol). The 25-hydroxyvitamin D is most commonly ordered because it is the most abundant of the two and has the longest half-life of 12-19 days compared to only a few hours for 1,25-hydroxyvitamin D. Vitamin D is a fat-soluble vitamin and can therefore be used as a diagnostic measure for cystic fibrosis, Crohn's disease, Celiac disease and other forms of malabsorption. Inflammation and obesity can impact the absorption and metabolism of vitamin D.

The vitamin D receptor is mainly found in cells of the immune system, endocrine system, neuromusculoskeletal system, and cardiovascular system.

Vitamin D can be attained by sunlight (ultraviolet B radiation) exposure to the skin and then activated to 1,25-hydroxyvitamin D in the liver and kidneys. Lighter-skinned individuals require less time in the sun for vitamin D production compared to darker-skinned individuals who require much longer periods of time in the sun. D3 or cholecalciferol is the form of vitamin D manufactured through this mechanism and D2 or ergocalciferol can be attained from plants. Currently, the literature supports both forms as being equally effective however D3 is more efficiently metabolized than D2. Ergocalciferol is the form of vitamin D used to fortify foods such as milk. D3 can be found naturally in egg yolks and fatty fish.

Vitamin D has been traditionally viewed as a "bone supplement" due to its connection with healthy bone formation and teeth. Vitamin D deficiency can lead to softening and malformation of bones in children known as rickets and osteomalacia in adults. The non-classical actions of vitamin D include regulation of hormone secretion, immune function and cell proliferation and differentiation. More and more research however is pointing out the importance of vitamin D for a variety of health issues such as autoimmune disease, diabetes/insulin resistance, PCOS, mood, respiratory disease, cardiovascular disease and a significant number of cancers. It has also been linked to increases in all-cause mortality.

According to the CDC approximately 1/3 of Americans are deficient in vitamin D however these statistics are based on the standard laboratory ranges for 25-hydroxyvitamin D through commercial laboratories. In addition, vitamin D receptor polymorphisms are not taken into account and neither is vitamin D receptor deactivation via microbial activity such as the Epstein-Barr virus and *Borrelia burgdorferi*. Both scenarios may require much higher levels of vitamin D supplementation and strategies to resensitize the receptors.

Low serum calcium levels can be an indication of vitamin D deficiency. The 1,25-hydroxyvitamin D test can be utilized when serum calcium levels are high which can be connected to lymphoma, hyperparathyroidism or sarcoidosis. Enzyme abnormalities involved in the conversion of 25-hydroxyvitamin D to 1,25-hydroxyvitamin D can also cause imbalances in both levels. Low 1,25-hydroxyvitamin D levels can be associated with kidney disease.

According to the Endocrine Society a vitamin D deficiency is a level below 20 ng/ml and an insufficiency is 21-29 ng/ml. 30 ng/ml or higher indicates a sufficiency however this is defined as a level to basically maintain healthy bones and does not take into account benefits from higher levels in certain individuals.

Magnesium deficiency can result in an unresponsive serum calcium deficiency despite vitamin D supplementation which requires magnesium sufficiency to correct this imbalance.

Although vitamin D toxicity is extremely rare, excess levels can cause calcification mainly of the blood vessels and kidneys.

Vitamin D supplementation can be done via oral capsules and drops as well as through topical creams which are used for psoriasis. Minimum recommendations are 800-1200IU/day with higher doses of 10,000IU/day to 50,000IU/week in specific cases. Vitamin D supplements that contain vitamin K can be beneficial for proper utilization of vitamin D. Since vitamin D is a fat-soluble vitamin it is recommended to be taken with food that contains an adequate macronutrient percentage of fat calories.

Currently, “optimal” vitamin D levels may be in the range of 30-50 ng/ml since some research points to toxicity at a level greater than 50 ng/ml. Interestingly, equatorial inhabitants with traditional lifestyles have serum 25-hydroxyvitamin D levels around 46 ng/ml which may provide a clue as to what is optimal for human beings. D2 and D3 can be measured together for a total vitamin D which may reflect the most accurate measurement at this time.

The main foods fortified with vitamin D include milk, cereal grains and fruit juices. Fortification has reduced the rate of rickets successfully.

## RELATED TESTS:

Parathyroid hormone, Calcium, Phosphorous, Magnesium, RBC magnesium, Bone markers.

**ELEVATED LEVELS OF 25-HYDROXYVITAMIN D MAY INDICATE:**

- Excessive supplementation

**DECREASED LEVELS OF 25-HYDROXYVITAMIN D MAY INDICATE:**

- Malabsorption
- Celiac disease
- Lack of sunlight
- Inflammation
- Obesity/abnormal body composition
- Magnesium deficiency
- Excessive sunscreen application
- Liver and kidney disease
- Autoimmune disease
- Infection
- Parathyroid tumor (primary hyperparathyroidism)

**CLINICAL INDICATIONS:**

The 25-hydroxyvitamin D test may not be an accurate representation of vitamin D status once supplementation has begun due to a variety of factors such as inflammation, abnormal body composition and gastrointestinal health. Increasing vitamin D intake when 25-hydroxyvitamin levels do not increase after supplementation, may not be appropriate. 1,25-hydroxyvitamin D levels may be increasing significantly while 25-hydroxyvitamin D levels do not after supplementation. It is therefore more prudent to maintain a consistent dose while addressing other abnormal metabolic imbalances.



# FIBRINOGEN

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**Reference Range:** 193-507 mg/dL

Fibrinogen, made in the liver, is a protein required for blood clot formation but it is also an excellent marker of inflammation. Fibrinogen can be used to assess cardiovascular risk and chronic inflammation. Fibrinogen falls into the category of acute phase reactants which are produced during acute tissue injury and inflammation. Fibrinogen can also predict blood clot formation such as a risk marker for thromboembolism. Elevated fibrinogen levels have been correlated with increased mortality. Coagulation testing is used in chronic illnesses such as in Lyme disease and chronic fatigue syndrome due to the connection between chronic infections and increased coagulation. Coagulation is upregulated by certain stealth microbes to reduce the immune system's ability to target and remove the infection. Focusing on reducing the fibrinolytic system and coagulation mechanisms can aid in successful patient outcomes.

Fibrinogen activity is a separate test that can be ordered to assess the time it takes for a fibrin clot to form. A fibrinogen antigen test can also be run which uses a fibrinogen antibody that binds to fibrinogen to measure the quantity of fibrinogen in the blood rather than activity.

## **RELATED TESTS:**

C-reactive protein, Homocysteine, Prothrombin Time, D-dimer, Partial Thromboplastin Time, Thrombin Time, Coagulation Factors, ESR

## **ELEVATED LEVELS MAY INDICATE:**

- Inflammation
- Infections
- Rheumatoid arthritis
- Glomerulonephritis
- Peripheral arterial disease
- Myocardial infarction

- Stroke
- Cancer
- Coronary heart disease
- Trauma
- Pregnancy
- Estrogen replacement
- Oral contraceptives
- Cigarette smoking

**DECREASED LEVELS MAY INDICATE:**

- Afibrinogenemia
- Hypofibrinogenemia
- Malnutrition
- Liver disease
- Kinase enzymes

**CLINICAL INDICATIONS:**

Chronic infections can upregulate coagulation mechanisms as an attempt by the microbe to enhance its stealth abilities and reduce the immune system's potential to eradicate and remove. A full infectious disease evaluation for bacterial, viral, parasitic and fungal infections may be warranted. As with elevated CRP levels, identification of the underlying cause of inflammation is paramount.

**NUTRACEUTICALS THAT MAY REDUCE BLOOD COAGULATION:**

- Omega-3 fatty acids
- Garlic
- Quercetin
- Lumbrokinase
- Serrapeptase
- Nattokinase

- CoQ10
- Bromelain
- Ginger
- N-Acetyl Cysteine
- Capsaicin

NUTRACEUTICALS THAT MAY REDUCE INFLAMMATION:

- Omega-3 fatty acids
- Curcumin
- Quercetin
- Resveratrol
- EGCG (Green tea extract)
- Boswellia
- Ginger
- Celery seed extract
- Bromelain
- Niacinamide
- MSM
- N-acetyl cysteine
- Rutin
- Rosemary
- Proteolytic enzymes



# HOMOCYSTEINE

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**Reference Range:** 0.0-15.0 umol/L

Homocysteine is used to diagnose the rare genetic disorder homocystinuria, vitamin B12 and folate deficiencies, and to assess cardiovascular risk. Homocysteine is a sulfur-containing amino acid manufactured from the metabolism of methionine. Vitamin B12, B6 and folate are required for homocysteine metabolism thus supplementation can reduce homocysteine levels. Homocysteine in excess can damage blood vessels, cause bone loss and increased fracture risk, macular degeneration, gall stones and reduce cognitive function. It is currently controversial if reducing homocysteine levels actually reduces the risk of cardiovascular disease due to conflicting evidence.

In homocystinuria, homocysteine and methionine levels build up in the system resulting in detached lens, long slender fingers, osteoporosis, skeletal deformities, atherosclerosis and increased risk of thromboembolism.

Females produce less homocysteine than males.

## RELATED TESTS:

Vitamin B12, Folic acid, Intrinsic factor antibodies, Amino acid profile, MTHFR, Organic acids, Methylmalonic acid, CBC, CRP, Fibrinogen

## ELEVATED LEVELS MAY INDICATE:

- B12 deficiency
- Folate deficiency
- B6 deficiency
- Inflammation
- Menopause
- Cigarette smoking

**DECREASED LEVELS MAY INDICATE:**

- ▶ Malnutrition

**CLINICAL INDICATIONS:**

Supplementation can certainly lower homocysteine but this has not shown to provide a reduction in the risk of a cardiovascular event. It is more important to identify the cause of the elevation which is usually chronic inflammation. Chronic inflammation can result in abnormal methionine metabolism and disruptions in methylation. Focusing on lowering homocysteine with supplements is akin to lowering cholesterol with red yeast rice which may or may not have any clinical relevance.

# INSULIN

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**Reference Range:** 2.6-24.9 uIU/mL

Insulin is a hormone made in the beta cells of the pancreas which controls blood glucose metabolism. It is released in response to increases in blood glucose levels to transport glucose into mainly muscle and adipose tissue cells for metabolism. Insulin also signals the liver to store glucose as glycogen for future need or to convert it to fatty acids. The insulin test is used to monitor insulin resistance, diabetes, insulin medication, acute and chronic hypoglycemia, and to diagnose insulinoma.

Type 1 diabetes is a condition in which the pancreas cannot produce adequate insulin requiring insulin injections. Type 2 diabetes is severe insulin resistance requiring careful monitoring of food choices and medications that resensitize insulin receptors.

## SYMPTOMS OF ELEVATED INSULIN INCLUDE:

- Confusion
- Seizure
- Irritability
- Nausea
- Fatigue
- Shakiness
- Blurry vision
- Sweating
- Palpitations
- Hunger
- Fainting

Insulin resistance is found in polycystic ovarian syndrome, cardiovascular disease, pre-diabetes, metabolic syndrome, and disorders of the pituitary and adrenal glands. Consequences of insulin resistance include hypertension, hyperlipidemia, and heart disease. Obesity, especially around the abdomen and a high carbohydrate diet with lack of exercise can lead to insulin resistance. The liver can become highly insulin resistant resulting in elevated blood glucose and triglycerides.

## **RELATED TESTS:**

Glucose, Glucose Tolerance Test, C-Peptide, Hemoglobin A1C

## **ELEVATED LEVELS MAY INDICATE:**

- Growth Hormone Injections
- Cushing's disease
- Insulin resistance
- PCOS
- Insulinoma
- Acromegaly
- Obesity
- Metabolic syndrome
- Type 2 diabetes
- Corticosteroids
- Oral contraceptives
- Levodopa
- Fructose & galactose intolerance

## **DECREASED LEVELS MAY INDICATE:**

- Pancreatitis
- Pancreatic cancer
- Diabetes
- Hypopituitarism

## Insulin

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DISORDER	FASTING INSULIN LEVEL	FASTING GLUCOSE LEVEL
<b>Insulin Resistance</b>	High	Normal or High
<b>Insufficient Insulin Production</b>	Low	High
<b>Hypoglycemia</b>	Normal or High	Low

### CLINICAL INDICATIONS:

See A1c and Glucose

# RECOMMENDED RESOURCES

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## **Dr. Hedberg's office:**

Immune Restoration Center  
141 Asheland Ave. Suite 301  
Asheville, NC 28801  
Phone: 828-254-4024

<http://www.drhedberg.com>

[info@drhedberg.com](mailto:info@drhedberg.com)

Facebook: [www.facebook.com/DrNikolasHedberg](http://www.facebook.com/DrNikolasHedberg)

Twitter: [www.twitter.com/drhedberg](http://www.twitter.com/drhedberg)

LinkedIn: [www.linkedin.com/in/drnikolashedberg](http://www.linkedin.com/in/drnikolashedberg)

## **ONLINE TRAINING IN FUNCTIONAL MEDICINE APPROACHES TO INFECTION-RELATED CHRONIC ILLNESSES:**

Go to <http://www.infectionconnection.net> for more information and for free infectious disease reports.

Dr. Hedberg's book *The Thyroid Alternative* can be purchased in paperback or for Kindle at <http://www.amazon.com>.

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Phone: 866-999-4041

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