Testing Nutritional Status: The Ultimate Cheat Sheet

by Chris Masterjohn, PhD

This is a “cheat sheet” in two ways:

- All of the lab testing required for comprehensive nutritional screening is reduced to a single page, with hyperlinks making ordering any of the tests just one click away.
- In just five pages, I provide full instructions for lab testing, blood pressure, and dietary analysis, as well as an algorithm for quick decisions on what to do next for each marker that may be off.

This “cheat sheet” is *ultimate* because of what comes next:

- Over 70 pages list the signs and symptoms associated with all the possible nutrient imbalances, the potential causes of nutrient imbalances, and an action plan for correcting each imbalance.

While you can read the more than 75 pages assembled here straight through if it tickles your fancy, this guide is not meant to be used that way. It retains its “cheat sheet” status by making only the parts that are relevant to you one click away as you move through the findings of your initial nutritional screening.

To make the best use of this guide, please read the disclaimer and the instructions for use before beginning.

Important Disclaimer

This cheat sheet is meant for educational purposes only, and is not a substitute for or a component of a comprehensive training in medicine or dietetics, nor does it constitute medical or nutritional advice or act as a substitute for seeking such advice from a qualified health professional. If you are a health care practitioner, always do your due diligence to research alternative explanations for the information herein and ensure that any actions you take are consistent with the legal and ethical frameworks governing your practice. If you are an individual seeking to improve your own health, always ask your doctor about taking any health-related measures and never ignore professional medical advice on the basis of anything contained herein. As the author of this educational product I am not responsible or liable for the results of taking any actions on the basis of this information.

In order to make the cheat sheet easier to read, I have used a conversational tone in many places with personal pronouns, such as “I” and “you.” This is meant *only* to make it more pleasant to read, and is not meant to imply that the guide constitutes any form of advice, whether personal or general.

How to Use This Cheat Sheet

If you are a health care practitioner, you can use this cheat sheet to more quickly find information related to nutritional testing that could help your patients and clients. It is important to always cross-reference information in this guide with your own knowledge from your training, current research and treatment guidelines, and information about tests provided by the laboratories that offer those tests.

If you are an individual reading this for your own private benefit, it will help you better understand the tests your doctor may order for you, and provide ideas for testing or nutritional strategies that you could discuss with your doctor. Depending on where you live, you may be able to legally order tests through a direct-to-consumer service such as directlabs.com. If you do so, it is important to discuss all results with your doctor, because these tests might provide evidence of medical conditions that are not discussed in this guide, and your doctor can provide important input on the safety of any nutritional strategies you choose to use.

There are three ways you can use this cheat sheet, depending on your resources and priorities:

- **Option 1: The Comprehensive Approach**. This is the right option if money is not an issue for you, or if your medical insurance will cover all of the testing. In option 1, you order all of the lab tests listed in the comprehensive screening. While waiting for the test results, you conduct your own dietary analysis and measure your blood pressure. You then search through the index of the signs and symptoms of nutritional imbalances for any that apply to you. When all of the data is in, you read the full sections of the guide that are flagged as relevant to determine which nutritional imbalances are most likely to be affecting you and what the right course of action is.

- **Option 2: The Time-Saving Approach.** This is the right option if your time is severely constrained, making the dietary analysis infeasible. In this case, complete all aspects of option 1, but skip the initial dietary analysis. Only resort to dietary analysis when troubleshooting a specific nutritional imbalance proves difficult.

- **Option 3: The Cost-Saving Approach.** This is the right option if you want to be conservative about lab testing for any reason, including financial costs. Skip the lab tests and begin with the dietary analysis and blood pressure measurement. Browse through the index of the signs and symptoms of nutritional imbalances. Read through the full sections of the guide that are flagged as relevant by any of your data. Use the suggestions for lab testing only when needed to help clarify the appropriate nutritional strategy to implement.

If at any point you need help, head to the ask me for help section.

Ready? Choose your approach, and dig right in!

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Vitamin A

Signs and Symptoms of Deficiency: Well established signs and symptoms of vitamin A deficiency include the following: poor night vision; dry eyes; hyperkeratosis around hair follicles, or appearing as bumps on the skin that can be mistaken for goosebumps or acne, or on the surface of the conjunctiva (Bitot's spots); poor immunity to infectious diseases. Less well established but plausible signs and symptoms of deficiency include the following: kidney stones; disrupted circadian rhythm and an inability to use light therapy to entrain a healthy circadian rhythm; autoimmune disorders; asthma and allergies; food intolerances; low sex hormones; and delayed puberty.

Risk Factors for Deficiency: Diets that do not contain at least one of the following: a weekly serving of liver; regular use of cod liver oil, a multivitamin, or another supplement providing 100% of the US RDA for vitamin A as retinol. If the diet is also poor in dairy products and eggs, and does not contain several servings per day of red, orange, yellow, or green vegetables, vitamin A deficiency becomes very plausible. Diets where fats come from polyunsaturated vegetable oils are more likely to produce vitamin A deficiency than diets where the fat is mostly saturated or monounsaturated. A low-fat diet will not intrinsically produce vitamin A deficiency, but it will increase its likelihood by leading to lower absorption of vitamin A from food. Long-term

use of glucocorticoids, high-protein diets, and high-dose vitamin D may contribute to vitamin A deficiency in combination with poor dietary intake.

Signs and Symptoms of Toxicity: Most commonly, nausea, vomiting, and headache. In extremes, anorexia, blurred vision, scaling skin, hair loss (alopecia), organ damage, death. Osteopenia and osteoporosis can be worsened by vitamin A at non-toxic levels when vitamin D and calcium are deficient. It is prudent to keep vitamin A below 10,000 IU per day during the first eight weeks of pregnancy due to a possible risk of birth defects unless blood measurements, signs, and symptoms justify higher intakes to prevent deficiency.

Risk Factors for Toxicity: Months or years of consistently taking at least 165 IU per kilogram body weight per day, and in the majority of cases greater than 2300 IU per kilogram body weight per day. For a person weighing 70 kilograms (154 pounds), this is a minimum of 11,550 IU and higher than 161,000 IU per day in the majority of cases. These figures apply to cases where vitamin D was not supplemented alongside it. When vitamin D is taken alongside vitamin A, the majority of vitamin A toxicity cases involve months or years of consistently taking more than 4620 IU vitamin A per kilogram body weight per day, which for a person weighing 70 kilograms is 323,400 IU per day. Almost all vitamin A is prepared in oil; however, vitamin A preparations that are water-soluble, emulsified, or solid may cause toxicity in weeks rather than months and at ten times lower doses.

Testing for Vitamin A Deficiency:

- **Serum vitamin A** (individually: LabCorp, Quest, in panels: Genova ION Profile + 40 amino acids, Genova's fat-soluble vitamin panel). This should be kept toward the middle of the reference range (third quintile) and low-normal results do not necessarily rule out a problem.
- **Retinol-binding protein** (LabCorp, Quest) can be measured alongside serum vitamin A, but may be affected by a greater number of variables unrelated to vitamin A status (it is increased in insulin resistance and type 2 diabetes, and decreased in type 1 diabetes, systemic inflammation, and a variety of liver and kidney diseases).

Testing for Vitamin A Toxicity:

- **Serum vitamin A** (LabCorp, Quest) will be high in most cases.
- In descending order of likelihood, the following tests may show elevations: **γ-glutamyltransferase** (LabCorp, Quest), **triglycerides** (LabCorp, Quest), **alkaline phosphatase** (LabCorp, Quest), **prothrombin time** (LabCorp, Quest), **cholesterol** (LabCorp, Quest), **aspartate aminotransferase** (LabCorp, Quest), **bilirubin** (LabCorp, Quest), and **calcium** (LabCorp, Quest).

Testing Caveats: Zinc is necessary for virtually every step in vitamin A metabolism, including its transport in the blood. Zinc deficiency should *always* be considered as an explanation for an apparent case of vitamin A deficiency that does not respond well to dietary and supplemental strategies, regardless of whether serum vitamin A is altered. Adiposity may cause cellular vitamin A deficiency without lowering serum levels. Fatty liver disease compromises the liver's ability to store vitamin A and may raise serum levels. Drugs that are vitamin A derivatives (known as retinoids; e.g., isotretinoin, marketed as Accutane) may cause vitamin A deficiency

signs by hurting the body's utilization of natural vitamin A. Chronic alcohol abuse and protein deficiency also hurt vitamin A utilization.

Correcting Vitamin A Deficiency: The strategy to fix the deficiency should be determined by the cause. If the cause is a dietary deficiency, the first step is to reverse the dietary risk factors listed above. Supplements providing 25,000-50,000 IU per day appear to be well within the margin of safety for short-term use (several weeks) in an adult, and may help resolve a deficiency more quickly, but should not be used without close monitoring of serum vitamin A to ensure it stays within the normal range. Someone who develops vitamin A deficiency on an apparently adequate diet may need higher doses long-term (months, years, or indefinitely), but again, you should only use high doses if you consistently monitor serum levels. You may often need trial and error to find the right dose. You should rule out deficiencies of other nutrients, especially vitamin D, before initiating high-dose vitamin A. If malabsorption is the cause of deficiency, the solutions listed under "General Considerations for Fat-Soluble Vitamins" should be followed. If factors listed under "testing caveats" are responsible for deficiency signs, these should be resolved independently and vitamin A supplementation should not be used to compensate for them.

Correcting Vitamin A Toxicity: The only well established treatment for vitamin A toxicity is the removal of the toxic dose of vitamin A. Many of the secondary effects of vitamin A toxicity, such as organ damage and hypercalcemia, require medical care that is beyond the scope of this guide. I recommend testing for deficiencies of the other fat-soluble vitamins and correcting any that exist.

Molybdenum and Sulfur Catabolism

The transsulfuration pathway is closely connected to the methylation pathway and represents the intersection between the methylation pathway and the antioxidant system by providing the amino acid cysteine for the synthesis of glutathione, the master antioxidant of the cell. The pathway is activated by high methionine inputs, serving to get rid of the excess (as would occur after a high-protein meal), and by oxidative stress, serving to increase the synthesis of glutathione when it is most needed. If the sole need is to get rid of excess methionine, the cysteine is catabolized to taurine and sulfate. If the sole need is to increase glutathione synthesis, it is directed into that pathway. Most of the time, cysteine meets a mix of these fates.

Regardless of the fate of cysteine, the amino acid serine and vitamin B6 are needed for its production from homocysteine. The conversion always generates alpha-ketobutyrate as a byproduct, which is the major source of methylmalonic acid, discussed above as a marker of vitamin B12 status. The methylmalonic acid requires biotin to be produced, and vitamin B12 to be eliminated. The synthesis of glutathione from cysteine requires glycine, magnesium, and ATP. On its way to sulf**a**te, cysteine first generates sulf**i**te. Sulfite is toxic and its accumulation can cause deficiencies of thiamin and vitamin B6. The conversion of sulfite to sulfate requires **molybdenum**, an essential mineral.

Of the many nutrients that make an appearance in sulfur catabolism, only molybdenum is unique to the pathway. Therefore, this section is devoted specifically to molybdenum.

Signs and Symptoms of Molybdenum Deficiency: Molybdenum deficiency is not well characterized and is conventionally thought to be uncommon except as a rare genetic disorder in its utilization, sulfite oxidase deficiency. This causes severe defects such as seizures, mental retardation, and dislocated lenses within the eye, all occuring in the newborn, and is unlikely to be informative for what moderate deficits in molybdenum might look like. More moderate sulfite accumulation may impair thiamin and B6 status and cause the signs and symptoms discussed in those sections. Presumably, molybdenum deficits could contribute to sulfite sensitivity as well, which results in in allergy-like reactions (dermatitis, hives, flushing, low blood pressure, abdominal pain, diarrhea, anaphylaxis, asthma) to the sulfites used as food additives.

Risk Factors for Molybdenum Deficiency: Molybdenum is very rich in legumes. The need for molybdenum increases with high intakes protein, especially of animal proteins, as a result of their high methionine content. Thus, a diet rich in animal proteins and low in legumes is likely to lead to significantly lower molybdenum status than the opposite pattern. The need for molybdenum appears to also increase during pregnancy, where the production of hydrogen sulfide, essential to the growth of the placenta and embryo, provides an additional source of sulfite. The morning sickness of pregnancy may result from sulfite-induced B6 deficiency that occurs on the background of low molybdenum intakes.

Molybdenum Excess: There is no evidence for molybdenum toxicity in humans. Excess molybdenum induces copper deficiency in ruminants, but this has no relevance to humans because it results from byproducts produced in the rumen. The Institute of Medicine set the tolerable upper intake level (TUIL) for molybdenum on the basis of reproductive defects induced in female rats by taking the lowest level that caused no harm, dividing it by 30, and adjusting it for bodyweight. The upper limit is set at 30 micrograms per day per kilogram bodyweight, which in a 70 kilogram individual is 2100 micrograms per day. There is no reason to use doses higher than this, and supplements often contain as little as 150 micrograms and rarely contain more than one milligram. Thus, molybdenum supplements according to the labeled use should be overwhelmingly safe.

Testing for Molybdenum Status

- **Serum or Whole Blood Molybdenum** (HDRI) Many blood tests of molybdenum levels are designed to only look for excess and only report whether molybdenum is below the range of excess. HDRI, by contrast, reports an exact value within a normal range. Either test will work, but ideally it should be tested along with one or more of the functional markers listed below.
- **Uric Acid** (LabCorp, Quest) In addition to converting sulfite to sulfate, molybdenum is also necessary to make uric acid. Uric acid is low in molybdenum deficiency.
- **Urinary Sulfite and Sulfate** (available from HDRI as part of the “sulphur panel” on their requisition form) High urinary sulfite and low urinary sulfate indicates a molybdenum deficiency.
- **Serum Sulfate and the Cysteine-to-Sulfate Ratio** (Genova Oxidative Stress 2.0 Blood Panel) Although not a direct measure of sulfite accumulation, low serum sulfate and a high cysteine-to-sulfate ratio may indicate molybdenum deficiency.

Correcting a Molybdenum Deficiency: Increasing legumes and decreasing animal protein may help correct a molybdenum deficit, but animal protein is nutritionally important and some

individuals do not tolerate legumes well. If dietary measures are unfeasible or insufficient, 500-1000 micrograms per day of molybdenum supplementation should be more than adequate, and this can likely be dropped to 100-200 micrograms per day as a maintenance dose once markers and signs and symptoms resolve.

Vitamin D, Calcium, and Phosphorus

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Magnesium

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Vitamin K

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B Vitamins Involved in Energy Metabolism

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Thiamin (Vitamin B1)

This section is not included in the preview.

Riboflavin (Vitamin B2)

This section is not included in the preview.

Niacin (Vitamin B3)

This section is not included in the preview.

Pantothenic Acid (Vitamin B5)

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Vitamin B6

This section is not included in the preview.

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B Vitamins Involved in Methylation

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Vitamin C

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Manganese

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Zinc

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Copper

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Selenium

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Iron

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Glutathione

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Iodine

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How to Ask for My Help With This Cheat Sheet

If you would like to ask me specific questions by email and receive a private response, please use this link. The questions should be precise, specific, and should not contain links, attachments, or details of individual cases that I would have to consider. I charge a small fee for this service.

If you would like to discuss your own case with me or the cases of others you are helping, you may sign up for a single consultation or a consulting package using this link.