Test requisition # Physician:

MOSAIC DIAGNOSTICS
Formerly Great Plains Laboratory

CLIENT #:

ORDER:
TEST:
CLIENT REF:
PATIENT:
ID:
SEX:
AGE: **DOB:**

Toxic Metals; urine

TOXIC METALS				
		RESULT µg/g Creat	REFERENCE INTERVAL	WITHIN REFERENCE / OUTSIDE REFERENCE
Aluminum	(Al)	4.4	< 25	
Antimony	(Sb)	0.025	< 0.18	
Arsenic	(As)	7.9	< 50	
Barium	(Ba)	1.0	< 5	
Beryllium	(Be)	<dl	< 0.01	
Bismuth	(Bi)	<dl	< 1	
Cadmium	(Cd)	0.13	< 0.9	
Cesium	(Cs)	15	< 10	
Gadolinium	(Gd)	<dl	< 0.8	
Lead	(Pb)	0.28	< 1.2	
Mercury	(Hg)	0.75	< 1.3	
Nickel	(Ni)	1.1	< 5	
Palladium	(Pd)	<dl	< 0.3	
Platinum	(Pt)	<dl	< 0.1	
Tellurium	(Te)	<dl	< 0.5	
Thallium	(Tl)	0.21	< 0.5	
Thorium	(Th)	<dl	< 0.02	
Tin	(Sn)	0.21	< 5	
Tungsten	(W)	0.088	< 0.4	
Uranium	(U)	0.005	< 0.03	

URINE CREATININE			
	RESULT	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	117	30 – 225	

SPECIMEN DATA

Comments:
Date Collected: 04/12/2023
Date Received: 04/22/2023
Date Reported: 04/25/2023
Methodology: ICP-MS QQQ, Creatinine by Jaffe Reaction

Collection Period: Random
pH upon receipt: Acceptable

< dl: less than detection limit
Results are creatinine corrected to account for urine dilution variations. **Reference intervals are based upon NHANES (cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions.** Chelation (provocation) agents can increase urinary excretion of metals/elements.

MOSAIC DIAGNOSTICS
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CLIENT #:

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Essential Elements; urine

ESSENTIAL ELEMENTS				
		RESULT mEq/g Creat	REFERENCE INTERVAL	PERCENTILE 2.5th 16th 50th 84th 97.5th
Sodium	(Na)	95.3	45 – 200	
Potassium	(K)	33.5	20 – 110	
		RESULT µg/mg Creat		
Phosphorus	(P)	487	180 – 1100	
Calcium	(Ca)	22	30 – 350	
Magnesium	(Mg)	48.9	25 – 230	
Zinc	(Zn)	0.10	0.1 – 1.5	
Copper	(Cu)	0.0066	0.006 – 0.026	
Sulfur	(S)	378	250 – 1050	
Molybdenum	(Mo)	0.0186	0.013 – 0.13	
Boron	(B)	0.98	0.6 – 4	
Lithium	(Li)	0.0382	0.009 – 0.2	
Selenium	(Se)	0.021	0.03 – 0.25	
Strontium	(Sr)	0.063	0.045 – 0.3	

		RESULT µg/g Creat	REFERENCE INTERVAL	68th 95th
Cobalt	(Co)	0.69	< 1.7	
Iron	(Fe)	4	< 50	
Manganese	(Mn)	0.05	< 0.6	
Chromium	(Cr)	0.12	< 2	
Vanadium	(V)	0.01	< 0.8	

URINE CREATININE			
	RESULT	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	117	30 – 225	

SPECIMEN DATA

Comments:
Date Collected: 04/12/2023
Date Received: 04/22/2023
Date Reported: 04/25/2023
Collection Period: Random
pH upon receipt: Acceptable
Methodology: ISE, Spectrophotometry, ICP-MS QQQ, Creatinine by Jaffe Reaction

< dl: less than detection limit
Results are creatinine corrected to account for urine dilution variations. **Reference intervals are based upon NHANES (cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions.** Chelation (provocation) agents can increase urinary excretion of metals/elements.

Introduction

This analysis of urinary elements was performed by ICP-Mass Spectroscopy following acid digestion of the specimen. Urine element analysis is intended primarily for: diagnostic assessment of toxic element status, monitoring detoxification therapy, and identifying or quantifying renal wasting conditions. It is difficult and problematic to use urinary elements analysis to assess nutritional status or adequacy for essential elements. Blood, cell, and other elemental assimilation and retention parameters are better indicators of nutritional status.

- 24 Hour Collections
  "Essential and other" elements are reported as mg/24 h; mg element/urine volume (L) is equivalent to ppm. "Potentially Toxic Elements" are reported as µg/24 h; µg element/urine volume (L) is equivalent to ppb.
- Timed Samples (< 24 hour collections)
  All "Potentially Toxic Elements" are reported as µg/g creatinine; all other elements are reported as µg/mg creatinine. Normalization per creatinine reduces the potentially great margin of error which can be introduced by variation in the sample volume. It should be noted, however, that creatinine excretion can vary significantly within an individual over the course of a day.

If one intends to utilize urinary elements analysis to assess nutritional status or renal wasting of essential elements, it is recommended that unprovoked urine samples be collected for a complete 24 hour period. For provocation (challenge) tests for potentially toxic elements, shorter timed collections can be utilized, based upon the pharmacokinetics of the specific chelating agent. When using EDTA, DMPS or DMSA, urine collections up to 12 hours are sufficient to recover greater than 90% of the mobilized metals. Specifically, we recommend collection times of: 9 - 12 hours post intravenous EDTA, 6 hours post intravenous or oral DMPS and, 6 hours post oral bolus administration of DMSA. What ever collection time is selected by the physician, it is important to maintain consistency for subsequent testing for a given patient.

If an essential element is sufficiently abnormal per urine measurement, a descriptive text is included with the report. Because renal excretion is a minor route of excretion for some elements, (Cu, Fe, Mn Zn), urinary excretion may not influence or reflect body stores. Also, renal excretion for many elements reflects homeostasis and the loss of quantities that may be at higher dietary levels than is needed temporarily. For these reasons, descriptive texts are provided for specific elements when deviations are clinically significant. For potentially toxic elements, a descriptive text is provided whenever levels are measured to be higher than expected. If no descriptive texts follow this introduction, then all essential element levels are within acceptable range and all potentially toxic elements are within expected limits.

Reference intervals and corresponding graphs shown in this report are representative of a healthy population under non-provoked conditions. Descriptive texts appear in this report on the basis of measured results and correspond to non-challenge, non-provoked conditions.

Chelation (provocation) agents can increase urinary excretion of metals/elements. Provoked reference intervals have not been established therefore non-provoked reference intervals shown are not recommended for comparison purposes with provoked test results. Provoked results can be compared with non-provoked results (not reference intervals) to assess body burden of metals and to distinguish between transient exposure and net retention of metals. Provoked results can also be compared to previous provoked results to monitor therapies implemented by the treating physician. Additionally, Ca-EDTA provoked results can be used to calculate the EDTA/Lead Excretion Ratio (LER) in patients with elevated blood levels.

CAUTION: Even the most sensitive instruments have some detection limit below which a measurement cannot be made reliably. Any value below the method detection limit is simply reported as "< dl." If an individual excretes an abnormally high volume of urine, urinary components are likely to be extremely dilute. It is possible for an individual to excrete a relatively large amount of an element per day that is so diluted by the large urine volume that the value measured is near the dl. This cannot automatically be assumed to be within the reference range.

This analysis of urinary essential elements was performed by ICP-Mass Spectroscopy. Analysis of essential and other elements in urine is used primarily to identify and characterize renal wasting conditions. Analysis of essential elements in urine is not a direct approach for assessing nutritional status or adequacy. Blood, cell, and other assimilation and retention parameters are optimal direct indicators of essential element status.

If one intends to utilize urinary elements analysis to assess nutritional status or renal wasting of essential elements, it is recommended that unprovoked urine samples be collected for a complete 24 hour period. For 24 hour urine collections essential elements are reported as mg/24 h. For timed or first morning urine collections, elements are normalized per gram creatinine to avoid the potentially great margin of error which can be introduced by variation in the sample volume (concentration). It should be noted that creatinine excretion for an individual may vary to some extent over the course of a day, and from day to day.

If an essential element is sufficiently abnormal per urine measurement, a descriptive text is included with the report. If there are no descriptive texts following this introduction, all essential element levels are within acceptable range. All reference ranges are age and sex specific.

This analysis of urinary toxic metals and essential elements was performed by ICP-Mass Spectroscopy. Analysis of metals in urine is traditionally used for evaluation of very recent or ongoing exposure to potentially toxic metals. The urinary excretion of certain metals is known to be increased (provoked) to a variable extent after administration of specific chelating agents. Reference values and corresponding graphs are representative of a healthy population under non-provoked conditions; reference values have not been established for provoked urine samples.

Analysis of essential and other elements in urine is used primarily to identify and characterize renal wasting conditions. Analysis of essential elements in urine is not a direct approach for assessing nutritional status or adequacy. Blood, cell, and other assimilation and retention parameters are optimal direct indicators of essential element status.

If one intends to utilize urinary elements analysis to assess nutritional status or renal wasting of essential elements, it is recommended that unprovoked urine samples be collected for a complete 24 hour period. For 24 hour urine collections essential elements are reported as mg/24 h, and toxic metals are reported as µg/24 h. For timed, random or first morning urine collections, elements and metals are normalized per gram creatinine to avoid the potentially great margin of error that can be introduced by variation in the sample volume (concentration). It should be noted that creatinine excretion for an individual may vary to some extent over the course of a day, and from day to day.

If an essential element is sufficiently abnormal per urine measurement, a descriptive text is included with the report. For potentially toxic elements, a descriptive text is provided whenever levels are measured to be higher than the unprovoked reference values. If there are no descriptive texts following this introduction, all essential element levels are within acceptable range and all potentially toxic metals are at levels below the unprovoked reference values. All reference ranges and reference values are age and sex specific.

Boron Low

Boron (B) is introduced to the body mainly through food (noncitrus fruits, leafy vegetables, nuts, legumes, wine, cider, beer) and drinking water but is also found in food preservatives (sodium borate), and insecticides (boric acid). Although there is an absolute requirement for B in vascular plants, evidence for biological essentiality in animals (including man) is limited. It has been proposed that boron contributes to living systems by acting indirectly as a proton donor and that it exerts a particular influence on cell membrane and structure and function. Boron is rapidly absorbed and excreted largely in the urine. Based on urinary recovery findings, more than 90% of ingested B is usually absorbed. Boron is distributed throughout the tissues and organs of animals and humans at concentrations mostly between 4.6 and 55.5 nmol (0.05 and 0.6 µg)/g fresh weight. Among the organs that contain the highest amounts of B are bone, spleen, and thyroid.

Boron influences macromineral metabolism and steroid hormone metabolism (testosterone, estrogen, DHEA, and 1,25 dihydroxycholecalciferol). A B deficient diet may also affect calcium metabolism and thus affects the composition, structure, and strength of bone. Signs of B deficiency in animals vary in nature and severity as the diet varies in its content of calcium, phosphorus, magnesium, potassium, cholecalciferol, aluminum, and methionine. Boron is also thought to have an estrogenic effect. In post-menopausal women consuming a very low B diet, B supplementation reduced the total plasma concentration of calcium and the urinary excretions of calcium and magnesium, and elevated the serum concentrations of 17ß-estradiol, testosterone, and ionized calcium, mimicking the effects of estrogen ingestion in postmenopausal women. In another study of magnesium and B deprivation among 13 healthy postmenopausal women (aged 50-78 years), it was found that marginal magnesium and B deprivation may also affect brain function as measured by EEG. It seems there may be increased CNS activity following boron deprivation. In long term hemodialysis patients serum boron levels may be excessively decreased.

No B requirements have been set as of 1998. Estimates are that between 1-2 mg/day may be required. Average intake in the U.S. has been estimated at between 1.7-4.3 mg/day.

Calcium Low

This individual's urine calcium is lower than one standard deviation below the mean of the reference population and corresponds to the lower 17% (approximately) of that population.

Low urinary calcium may be the result of: insufficient dietary intake, insufficient gastricacidification, inadequate vitamin D (or vitamin D function), or excessive phosphates, oxalates (spinach) or phytates (cereal grains) which may form insoluble calcium salts in the intestine. Intestinal absorption of calcium also is hindered in cases of lipid malabsorption; undigested fats can form insoluble calcium compounds. A very low protein diet or an overly alkaline intestine (pH > 7.5 approx.) can result in poor calcium uptake. Insufficient acidophilic flora, such as Lactobacilli, can impair calcium uptake (Harper, Rev. Phys.Biochem. 17th ed.p.576). Correction of dietary imbalances typically normalizes calcium uptake within several days; urine levels may take longer to normalize if there is need for calcium deposition in body issues.

Use of thiazide diuretics decreases calcium concentration in urine.

Pathological conditions that may feature subnormal urine calcium include: hypoparathyroidism, gastric hypochlorhydria, gastrointestinal malabsorption featuring impaired vitamin D uptake, lack of sunlight for vitamin D activation, steatorrhea, fatty acid metabo- lism disorder, some types of hypertension, tetany (serum calcium ion concentration also low), pre-eclampsia, genetic hypocalciuric hypercalcemia (elevated blood Ca), renal osteodystrophy, and vitamin D-resistant rickets.

Dietary deficiency or poor absorption of calcium increases the absorption of lead, increases blood and tissue levels of lead and, enhances the adverse effects of lead on cognitive function and behavior.

Urine analysis is not a preferred way to assess body calcium stores, and nutritional sufficiency of calcium should be assessed through dietary analysis. Whole blood calcium level, serum calcium level, serum vitamin D level (1,25-dihydroxy), parathyroid hormone determinations, and bone density measurement are tests that are more indicative of calcium status.

Cesium High

This individuals urine Cesium (Cs) level is higher than expected, reflecting exposure to Cs but symptoms may not be evident. Very high levels of Cs in urine are often associated with the use of cesium chloride as a questionable anti-cancer treatment. Cesium is a naturally-occurring element found in rocks, soil and dust at low concentrations. It is present in the environment only in the stable form of Cs133; the radioactive isotopes 134Cs and 137Cs are not measured or reported by Doctor's Data. Natural deposits of Cs ores occur in Main, South Dakota and Manitoba (Bernic Lake), Canada. Cesium may bio-accumulate in aquatic food chains; higher levels of cesium have been found in Pacific deep-sea fish and local shellfish since the 2011 Fukoshima reactor accident. Cesium may be used in high-density drilling fluids (oil and gas industry) and may contaminate local water and vegetation; Cs has been found in cow's milk. Cesium may occur naturally in mineral waters; one study analyzed the Cs concentration in 163 mineral and thermal waters and found the level ranged from 4.5 to 148 µg per liter.

Cesium can be absorbed after oral ingestion, upon breathing contaminated air and through contact with the skin. Cesium is readily absorbed across the brush border of the intestines in a manner similar to potassium and most is eventually excreted through the urine and feces. The biological half-life of Cs in humans ranges from 15 days in infants to 100-150 days in adults.

The cesium-137 isotope is used in cancer treatments, for ventricular function and pulmonary imaging studies, industrial radiology, and for food and instrument sterilization; Cs137 agents may contain small amounts of Cs133. Non-radioactive cesium chloride may be advertised on the internet as "high pH therapy." Currently there is no support in the scientific literature for that purpose as advertised. Radioactive Cs isotopes may contaminate soil at nuclear waste sites. Cesium may be used in industry for the production of photoelectric cells, vacuum tubes, spectrographic instruments, scintillation counters, DNA biochemistry, in various optical or detecting devices.

Target organs of potential toxic effects of Cs are the liver, intestine, heart, and kidneys. Physiological effects of excessive Cs include ventricular arrhythmias and displacement of potassium from muscle cells and erythrocytes. Cesium can have significant effects on both the central and peripheral nervous systems. Cesium may cause epileptic seizures because it can share the same receptor as the excitatory bioamine glycine. Cesium can interfere with active ion transport by blocking potassium channels and also can interfere with lipid metabolism. Excessive Cs may modify plasma membrane integrity, alter cytoplasmic components and cause cytogenetic damage.

It is unlikely that children or adults would be exposed to enough Cs133 to experience any health effects that could be related to the stable Cs itself. Animals given very large doses of Cs compounds have shown changes in behavior, such as increased activity or decreased activity, but it is unlikely that a human would be exposed to enough stable Cs to cause similar effects.

The isotope Cs137 is used in radiation therapy for certain types of cancer. Other medical uses of Cs are monitoring left ventricular function with Cs137 iodide probes and monitoring pulmonary endothelial permeability with Cs137 iodide crystal mini-detectors. Again, it is emphasized that Cs measured at Doctor's Data is Cs133, not Cs137. Environmental contamination by Cs137 as a result of radioactive fallout could be a concern. Exposure to Cs may be assessed by hair elemental analysis.

Commonly used chelating agents are not effective binders of Cs.

Copper Low

Low urinary copper may or may not correspond to subnormal copper levels in body tissues, and other laboratory tests are more indicative of copper status. Such tests include measurement of: whole blood or blood cell copper, hair copper, erythrocyte superoxide dismutase activity, and serum ceruloplasmin.Because the major route of copper excretion is via bile and feces, urinary levels may fluctuate without reflecting or influencing body stores.

Lower than normal excretion of copper (and other elements) can occur in renal insufficiency; in which case blood levels may be normal or elevated. Inadequate levels of molybdenum or zinc allow increased retention of copper, and transient hypocuprinuria may occur during periods when copper stores are accumulating.

Low urinary copper may also correspond to copper deficiency of nutritional or gastrointestinal origins.The richest dietary sources of copper are: nuts, shellfish, liver, raisins and legumes. Dairy products generally are low in copper content. Gastric hypochlorhydria, sprue, and pancreatic dysfunction may inhibit copper uptake.

Magnesium Low

This individual's magnesium level is lower than one standard deviation below the mean of the reference population which means that this individual's urine magnesium level corresponds to the lowest 17% (approximately) of that population.

In renal insufficiency, magnesium (along with other elements) can be low in urine but elevated in blood. Creatinine clearance and blood metabolite levels should be measured if a renal transport disorder is suspected.

24-hour urine levels of magnesium are considered by some authors to be a sensitive indicator of magnesium status (Galland, Magnesium 8 no.2, 1988, pp 78-83). Less than 24 mg/hr urinary Mg excretion suggests deficiency (Lauler, Am. J. Cardiology 63 no 14, 1989, 16).

Homeostatic regulation of blood magnesium levels is normally maintained within close limits. There are, however, many possible nutritional, metabolic, and hormonal factors which can result in subnormal urine levels of magnesium. These are listed below.

- Junk food diet, consumption of magnesium-deficient foods
- Malabsorption syndromes resulting in magnesium deficiency
  - Gluten enteropathy, sprue
  - Immune dysregulation, food reactivities with villous atrophy in the small intestine
  - Intestinal dysbiosis
  - Intestinal fistulas, bypass or resection surgery
  - Radiation enteritis
  - Gastric hypochlorhydria
  - Pancreatic insufficiency
  - Biliary insufficiency, steatorrhea
- Hypocalcemia with increased retention of Mg
- Hypothyroidism
- Alkalosis
- Alcoholic withdrawal
- Prolonged diarrhea

Magnesium status can be difficult to assess; whole blood and blood cell levels are more indicative than serum/plasma levels. The magnesium challenge method may be most indicative: baseline 24-hour urine mg measurement, followed by 0.2 mEq/Kg intravenous Mg, followed by 24-hour Mg measurement. A deficiency is judged to be present if less than 80% of Mg challenge is excreted. Ref. Jones et al. "Magnesium Requirements in Adults", Med. Journal Clin. Nutr., 20 (1967) pp. 632-35.

Molybdenum Low

This individual's molybdenum level is lower than one standard deviation below the mean of the reference population which means that this individual's urine molybdenum level corresponds to the lowest 17% (approximately) of that population.

Molybdenum is an essential activator of some important enzymes in the body: sulfite oxidase (catalyzes formation of sulfate from sulfite), xanthine oxidase (formation of uric acid and superoxide ion from xanthine), and aldehyde oxidase (processes aldehydes). Over 50% of absorbed Mo is normally excreted in urine; the remainder is excreted via bile to the feces or is excreted in sweat.

The level of molybdenum in urine may be a transient finding and may not reflect body tissue or liver levels. In copper deficiency, retention of molybdenum is increased (tissue levels could be normal or high), while urine levels might be subnormal. In renal insufficiency, molybdenum (and other elements) can be low in urine. Creatinine clearance and blood metabolite levels should be measured if a renal transport disorder is suspected.

Individuals receiving prolonged total parenteral nutrition ("TPN") may have low body tissue and urine levels of molybdenum because it is occasionally omitted from TPN formulations.

Molybdenum in foods is mostly in soluble complexes, and only a small amount is required daily (100 to 200 micrograms, adults). Therefore, frank molybdenum deficiency is uncommon. However, GI dysfunctions, poor-quality diet, and stressors can combine to produce inadequate molybdenum. Tungsten is a powerful antagonist of molybdenum retention, copper less so. Episodic exposures to high levels of either may result in periods of low Mo excretion that follow prior periods of high Mo excretion. Sulfites, aldehydes and high amounts of purines in the diet may increase need for and retention of molybdenum. Prolonged use of dithiol chelators (DMPS,DMSA) or MSM can result in poor molybdenum status as indicated by low levels in red blood cells(DDI observations).

A multielement hair analysis plus analyses for serum and urine uric acid can be used to confirm or rule out molybdenum insufficiency.

Potassium Low

The level of potassium (K) is lower than expected in this sample. K is an electrolyte and a potentiator of enzyme functions in cells. K can be low in the body as the result of gastrointestinal or renal dysfunction, or as a side effect of some diuretics. In adrenocortical hyperactivity, blood levels of K are depressed, while urinary K is increased. Diabetic acidosis and other medical conditions may result in severe K loss. Symptoms of true K deficiency include: muscle weakness, fatigue, and tachycardia. An electrocardiogram may show abnormalities when K is low in serum/plasma or whole blood.

Appropriate tests to confirm low K in body tissues may include measurements of packed red blood cell K; serum or whole blood K and sodium/K ratio.

Selenium Low

Urine accounts for about one-half of the total body excretion of dietary selenium when normal amounts are ingested. Seafood, organ meats, cereal grains, and seleniferous vegetables (garlic, onions) are good dietary sources. Selenium is also excreted in sweat, and lesser amounts are present in fecal matter. Because diets are highly variable in selenium content, urine is not a reliable indicator of selenium adequacy or function.

Low urinary selenium may be a consequence of: junk food diet or highly-processed food diet, gastrointestinal dysfunctions, renal insufficiency (in which case other elements will be subnormal in urine but possibly elevated in blood), and long-term parenteral nutrition or special diets that are low in selenium.

Selenium is a necessary element for proper activity of two enzymes in human metabolism: glutathione peroxidase (GPx) and iodothyronine deiodinase (ITD). Selenium deficiency may cause weakness or rate limitation for one or both of these enzymes. GPx oxidizes glutathione while reducing oxidized lipids. Weak GPx activity may allow excessive inflammation to occur. ITD deiodinates thyroxine prohormone and catalyzes T4 _ T3. Selenium deficiency may be a cause of insufficient T3 and thyroid dysfunction (Berry J.M. Nature 349, 1991 pp.438-40).

Symptoms consistent with selenium deficiency include: myalgia, increased inflammatory responses, hypothyroidism with low T3. Cardiomyopathy and Keshan disease can occur in cases of severe, chronic Se deficiency. Subnormal selenium may accentuate the effects of cadmium, mercury or arsenic overload. Confirmatory tests for selenium status include packed red bloodcell elements, and hair elemental analysis (provided that antidandruff shampoos have not been used.

Zinc Low

Low urinary zinc is not likely to correspond to global zinc deficiency because the major route for zinc excretion is via the bile, intestinal transport and feces. Typically, from two to ten percent of total zinc excretion occurs via urine; a similar amount occurs in sweat; the remainder (about 80 to 95%) occurs via biliary secretion to the intestine and is excreted in feces. Urine levels may fluctuate without reflecting or influencing body stores.

Zinc can be low (along with other elements) in urine in renal insufficiency. Creatinine clearance and blood metabolite levels should be measured if a renal transport disorder is suspected.

Low urine zinc may be a consequence of: junk food diet or highly-processed food diet, gastrointestinal dysfunctions, excess dietary copper or iron (impairing intestinal uptake of zinc), excess dietary fiber or folate (particularly if dietary zinc level is low to begin with), and inadequate levels of binding ligands for Zn uptake. Binding ligands are: citrate, histidine, cysteine, other organic and amino acids. Excessive loss of zinc to sweating during prolonged physical exertion can cause temporary, low levels of urinary zinc. It has been demonstrated experimentally that lead and nickel increase zinc excretion and that zinc deficiency enhances absorption of lead.

Symptoms and manifestations of zinc deficiency include: impaired sense of taste and/or smell, poor visual adaptation to darkness, slowed hair growth or hair loss, slowed wound healing, subnormal sperm count, loss of libido, immune suppression (poor neutrophil function, low phagocytic activity), poor appetite, anorexia in severely Zn-deficient individuals, and cessation of growth.

Other laboratory tests that are useful in assessing zinc adequacy include: measurement of zinc in whole blood or erythrocytes. RBC Zn is low in chronic Zn deficiency but is not low in short term depletion conditions. Hair zinc analysis can be confirmatory: low means low, but elevated hair zinc often appears to reflect maldistribution and need for zinc. Due to the high affinities of EDTA, DMPS and DMSA, low urinary zinc after administration of these chelating agents is suggestive of zinc deficiency. Other clinical findings that would be consistent with zinc insufficiency include: enlarged prostate, myalgia, (mild) lactic acidosis, history of alcoholism, failure to thrive (infants, children), protein malabsorption, acrodermatitis enteropathica, and subnormal plasma/serum protein levels.