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1).

POTASSIUM:

 The Tolerable Upper Intake Level (UL) specifies the highest average daily intake level of a nutrient, consumed on a habitual basis, that is likely to pose no risk of adverse health effects for nearly all apparently healthy individuals in a given Dietary Reference Intake (DRI) age, sex, and life-stage group. The potential for adverse health effects increases as intakes increase above the UL. The UL is intended to provide guidance on intake levels that are safe; it is not intended to serve as an intake goal. The Guiding Principles for Developing Dietary Reference Intakes Based on Chronic Disease (Guiding Principles Report) recommended that the UL be retained in the expanded DRI model, but that it should characterize toxicological risk (NASEM, 2017). Although this conceptual revision narrows the scope of the UL, it allows for a more nuanced characterization of the different types of risk that can exist with intake of a nutrient or other food substance. This chapter presents the committee's review of the evidence on the toxicological effects of excessive potassium intake and its conclusion regarding establishing a potassium UL. For context, the committee's findings are preceded by a brief summary of the decision made regarding the potassium UL in the Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate (2005 DRI Report) (IOM, 2005).

Potential indicators reviewed included gastrointestinal discomfort from certain forms of potassium supplements and arrhythmia from hyperkalemia. Available evidence indicated that, in generally healthy individuals, excess potassium is excreted in the urine. Because they may have impaired potassium excretion, individuals with certain conditions (e.g., chronic kidney disease, end-stage renal disease, diabetes, severe heart failure, adrenal insufficiency) and individuals who use certain medications (e.g., angiotensin-converting enzyme inhibitors [ACE-Is] and angiotensin-receptor blockers [ARBs]) were identified as potentially vulnerable subpopulations in which potassium intakes at the AI may not be appropriate.

Although dietary potassium intake can be increased through behavioral change, there is a self-limiting aspect to such changes that makes toxic adverse effects from increases in dietary potassium intake unlikely. Reports and studies evaluating potassium supplements were therefore considered most useful to determine whether a potassium intake level that could lead to toxicity could be quantified. For ethical reasons, trials cannot be designed to evaluate whether an intervention will increase the incidence of adverse effects. Consequently, adverse effect data in trials are almost always secondary outcomes. These data, particularly if systematically and carefully reported, can provide useful information for evaluating the likelihood of adverse effects. However, as secondary outcomes, these trials may not be adequately powered to identify a statistically significant occurrence of an adverse effect. These strengths and limitations need to be taken into account when using data from trials for evaluating the potential for adverse effects.

Guided by the first step of the DRI organizing framework, the committee sought to identify potential indicators of toxicological adverse effects from excessive potassium intake. The section that follows describes the evidence the committee reviewed to identify indicators that could potentially inform the derivation of the potassium UL.

Meta-analyses of randomized controlled trial data found that increasing potassium intake did not increase blood lipids, plasma adrenaline, or plasma noradrenaline concentrations among adults (Aburto et al., 2013). No other potential indicator of potassium toxicity was identified from the committee's literature scan.

Additional exploration of systematic reviews and case reports on toxicity, adverse effects, and poisonings from potassium intake were undertaken in an effort to identify potential toxicological adverse effects. From these efforts, the committee identified a collection of case reports on deaths and sublethal symptomology attributed to high levels of potassium intake. The committee also compiled reported adverse effects of the potassium trials included in the Agency for Healthcare Research and Quality systematic review, Sodium and Potassium Intake: Effects on Chronic Disease Outcomes and Risks (AHRQ Systematic Review) (Newberry et al., 2018), and the committee's supplemental literature searches.

2).

CALCIUM:

Besides your teeth, there's no harder structure in your body than your bones. Your bones form the sturdy framework of your body, just like the boards in the walls of your home make up the sturdy framework of your house. But what makes your bones so hard?

Well, your bones are hardened by minerals. Minerals are inorganic compounds needed by your body to regulate chemical reactions and maintain structures. There are many different minerals needed for optimum health, but when we're talking about bone strength, calcium is at the top of the list. You get calcium into your body through food sources such as dairy products, legumes, almonds and some dark green vegetables. In this lesson, you will learn more about calcium and what happens when you have too little or too much of this mineral in your body.

Deficiency

If you were to look inside your body, you would see that 99% of calcium is found in your bones and teeth. The amount that's left over might be small, but that doesn't mean it's unimportant. Calcium is a mineral needed not only for strengthening bones and teeth, but also for proper muscle contractions, the transmission of nervous system messages, and other important functions dealing with the regulation of heart function and blood clotting.

It's hard to believe that your body only requires 1% of the available calcium to carry out all of these important non-bone-related functions. Yet this system works because your body uses your bones as somewhat of a storage locker for calcium. You see, the level of calcium floating around in your blood is very closely monitored by your body.

If you're consuming too little calcium or calcium is not making it into your body due to a problem with calcium absorption, your blood calcium level drops, alerting your body to the calcium deficiency. When your body detects this, it allows bone resorption, which is the process of breaking down bone to release minerals. So if the blood needs more calcium, it goes to the bone storage locker and reabsorbs as much as it needs. If this process continues over time, the prolonged deficiency of calcium can lead to the reduced bone mass associated with osteoporosis.

We learned that calcium has many functions in your body, so a calcium deficiency will not only affect your bones.

3).

MAGNESSIUM:

Magnesium

Did you ever get one of those eyebrow twitches where your eyebrow or eyelid will not stop moving? This can be very distracting and so annoying that it drives you to the point where you would pull out your hair if it would just make it stop! So, what causes your eyebrow to twitch like that? Well, one possible culprit is a magnesium deficiency.

Magnesium is a major mineral that helps with many processes in your body, including nerve function, energy production, bone strength and normal muscle contraction. When your body is low, or deficient, in magnesium, it can disrupt the ion balance in your muscles and nerves and leave you feeling twitchy. In this lesson, we will learn more about magnesium and the factors that contribute to magnesium deficiency. We will also consider any effects that can arise if your body has too much magnesium.

Sources

It's not hard to find sources of magnesium since it's found in a variety of foods. A good way to remember magnesium food sources is to think of foods that are high in fiber. Fiber is part of plants, so plant-based foods, such as green leafy vegetables, beans, nuts and seeds, are good sources. There are also some animal products that contain magnesium, such as milk, yogurt and eggs, and magnesium is often added to breakfast cereals to boost their nutrient content.

Deficiency

With all these sources to choose from you would think that magnesium deficiency would be rare. While this is true in an otherwise healthy individual, there are some conditions and circumstances that can rob your body of proper magnesium levels. These factors that lead to deficiency include a prolonged low intake of magnesium due to a poor diet, digestive disorders that interfere with magnesium absorption, chronic stress and diuretic use.

Diuretics are substances that make you have to urinate more often, such as caffeine, alcohol and blood pressure medication. Their use can cause you to lose magnesium through urine when you visit the restroom. These factors either directly lower your level of magnesium or they interfere with your body's ability to absorb or retain enough magnesium.

Now, if you think back to our definition of magnesium, we see that it helps out with many functions in your body. So, it stands to reason that if you don't have the magnesium needed to carry out these functions, you would develop related symptoms. For example, we learned that magnesium helps with nerve function and energy production, so a deficiency could show up as depression, fatigue or numbness and tingling.

4).

CHLORIDE:

Chlorine deficiency, condition in which chlorine is insufficient or is not utilized properly. Chlorine is a component of all body secretions and excretions resulting from processes of building (anabolism) and breaking down (catabolism) body tissues. Levels of chlorine closely parallel levels of sodium intake and output, since a primary source of both is sodium chloride, or common table salt. Chlorine is stored to a limited extent in the skin, subcutaneous tissues, and skeleton and constitutes two-thirds of the negatively charged ions (anions) in the blood. Chlorides (chlorine compounds) play an essential role in the electrical neutrality and pressure of extracellular fluids and in the acid-base balance of the body. Gastric secretion is composed of chlorides in the form of hydrochloric acid and salts. Chlorine is readily absorbed during digestion, and similarly its rate of excretion through sweat, kidney excretion, and intestinal expulsion is high. The body’s supplies of chlorine are rapidly depleted during hot weather, when excessive perspiration reduces the fluid content of the body. Also, stored chlorides may become dangerously low in periods of severe vomiting and diarrhea and in diseases that produce severe alkalosis, an accumulation of base or loss of acid in the body. Treatment of chlorine deficiency is directed towards the underlying cause.

Chloride helps the body maintain its fluid balance. It also helps make the digestive enzymes that help the body metabolize food. Changes in chloride levels can harm these functions.

When chloride levels are moderately high, a person may not notice any symptoms. Long-term hyperchloremia, however, can cause a range of symptoms.

Those include:

fluid retention

high blood pressure

muscle weakness, spasms, or twitches

irregular heart rate

confusion, difficulty concentrating, and personality changes

numbness or tingling

seizures and convulsions

The severity of symptoms depends on how high chloride levels are, how long they have remained that high, and individual factors such as:

health

nutritional status

use of various medications

The symptoms of hyperchloremia and electrolyte imbalances are so general that it is impossible to diagnose this syndrome based on symptoms alone. People should not self-diagnose.

A simple blood test can detect hyperchloremia.

In children and adults, causes of hyperchloremia include:

Gastrointestinal problems, such as vomiting or diarrhea. These issues can cause dehydration.

A high fever that causes sweating and dehydration.

Dehydration due to medications, intense exercise, heat exposure, or not drinking enough fluids.

High sodium levels in the blood. Chloride tends to rise when sodium does.

Too much salt intake. Chloride is an ingredient in sodium chloride, which is table salt.

Diabetes insipidus, which causes the kidneys to pass large amounts of fluid.

Diabetic coma.

Some medications, particularly hormones, diuretics, and corticosteroids, such as hydrocortisone.

Starvation due to eating disorders, severe malnourishment, or problems absorbing nutrients from food.

Addison’s disease, a disorder that occurs when the adrenal glands cannot produce enough hormones.

5).

IRON:

Iron deficiency:

If absorption cannot compensate for losses or low dietary intakes, and body stores are used up, then iron deficiency sets in. Because so much of the body’s iron is in the blood, iron losses are greatest whenever blood is lost. Bleeding from any site incurs iron losses. Active bleeding ulcers, menstruation and injury result in iron losses.

Women are especially prone to iron deficiency during their reproductive years because of repeated blood losses during menstruation. Pregnancy places iron demands on women as well because iron is needed to support the added blood volume, the growth of the fetus and blood loss during childbirth. Infants and young children receive little iron from their high milk diets, yet extra iron is needed to support their rapid growth. The rapid growth of adolescence, especially for males, and the menstrual losses of teen females demand extra iron that a typical teen diet may not provide.

Assessment of iron deficiency:

Iron deficiency develops in stages. In the first stage of iron deficiency, iron stores diminish. Measures of serum ferritin reflect iron stores and are most voluble in assessing iron status.

The second stage of iron deficiency is characterized by a decrease in iron being transported within the body. Serum iron falls, and the iron carrying protein transferrin increases (an adaptation that enhances iron absorption). Together, these two measures can determine the severity of iron deficiency; the more transferrin and the less iron in the blood, the more advanced the deficiency.

The third stage of iron deficiency occurs when the supply of transport iron diminishes to the point that it limits hemoglobin production. Now the hemoglobin precursor, erythrocyte protoporphyrin, begins to accumulate as hemoglobin and hematocrit values decline.

Overview of iron deficiency symptoms:

Eyes: Blue sclera (sclera is a tough fibrous tissue that covers the white of the eye, blue sclera has an abnormal degree of blueness).

Immune system: Reduced resistance to infection.

Nervous/muscular systems: Reduced work productivity, reduced physical fitness, weakness, fatigue, impaired cognitive function, reduced learning ability, increased distractibility, impaired reactivity and coordination.

Skin: Itching, pale nail beds and palm creases, concave nails, hair loss, impaired wound healing.

General: Reduced resistance to cold, inability to regulate body temperature, pica (clay eating and ice eating).

Iron toxicity:

The body normally absorbs less iron if its stores are full, but some individuals are poorly defended against iron toxicity. Once considered rare, iron overload has emerged as an important disorder of iron metabolism.

Iron overload is known as hemochromatosis and usually is caused by a gene that enhances iron absorption. Other causes of iron overload include repeated blood transfusions, massive doses of dietary iron and rare metabolic disorders. Additionally, long-term overconsumption of iron may cause hemosiderosis, a condition characterized by large deposits of the iron storage protein hemosiderin in the liver and other tissues.

Iron overload is most often diagnosed when tissue damage occurs, especially in iron-storing organs such as the liver. Infections are likely to develop because bacteria thrive on iron-rich blood. Ironically, some of the signs of iron overload are analogous to those of iron deficiency: fatigue, headache, irritability and lowered work performance. Therefore, taking supplements before measuring iron status is clearly unwise.

Other common symptoms of iron overload include enlarged liver, skin pigmentation, lethargy, joint diseases, loss of body hair, amenorrhea and impotence. Untreated hemochromatosis aggravates the risks of diabetes, liver cancer, heart disease and arthritis.

In the United States, an estimated 10 percent of the population is in positive iron balance, with 1 percent having iron overload. Iron overload is more common in men than women and is twice as prevalent in men as iron deficiency. Some researchers have expressed concern about the widespread iron fortification of foods. Such fortification does make it hard for people with hemochromatosis to follow a low-iron diet but equal dangers lie in indiscriminate use of iron supplements.

Blood letting is the best treatment for hemochromatosis along with following a low-iron diet designed by a certified nutritionist containing substances that interfere with iron absorption. Some examples of substances that block iron absorption in such a diet include black tea, phytic acid found in whole grains, taking calcium with meals containing iron, and reducing vitamin C intake.

Iron recommendations and intakes:

Infants up until 6 months require 6 mg per day. From 6 months to 1 year, 10 mg is required.

Children age 1 to 10 require 10 mg per day.

Males age 11 to 18 require 12 mg per day.

Males age 19 to 51+ require 10 mg per day.

Females age 11 to 50 require 15 mg per day.

Females older than age 51 require 10 mg per day.

Pregnant women require 30 mg per day.

Lactating women require 15 mg per day.

Iron in selected foods:

Meat, poultry and fish will contribute highly absorbable iron. Legumes, dark green leafy vegetables, green beans, tomato juice, parsley, artichoke, dried fruits and corn flour contain respectable amounts of non-heme iron.

Iron is an essential nutrient that is vital to the processes by which cells generate energy. Iron also can be damaging when it accumulates in the body. In fact, iron is a problem nutrient for millions of people. Some people simply don’t eat enough iron-containing foods to support their health optimally while others have so much iron that it threatens their well-being. The principle that too little or too much of a nutrient is harmful seems particularly apropos for iron.

Iron has a knack of switching back and forth between two ionic states. In the reduced state, iron has lost two electrons and therefore has a net positive charge of two. Iron in the reduced state is known as ferrous iron. In the oxidized state, iron has lost a third electron, has a net positive charge of three and is known as ferric iron. Because iron can exist in different ionic states, iron can serve as a co-factor to enzymes involved in oxidation-reduction reactions. In every cell, iron works with several of the electron-transport chain proteins that perform the final steps of the energy yielding pathways. These proteins transfer hydrogens and electrons from energy- yielding nutrients to oxygen, forming water and, in the process, make ATP for the cells’ use. If you recall from my previous article on this website, ATP is adenosine triphosphate, the cellular energy currency of the body. A direct precursor to this substance is nicotinamide adenine dinucleotide (NADH).

Most of the body’s iron is found in two proteins: hemoglobin in the red blood cells and myoglobin in the muscle cells. In both, iron helps accept, carry and then release oxygen. Iron also is found in many enzymes that oxidize compound reactions so widespread in metabolism that they occur in all cells. Enzymes involved in the making of amino acids, hormones and neurotransmitters require iron.