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POTASSIUM

Toxicity Value

Potassium chloride and sodium chloride were infused into the reticulorumen of male Holstein calves approximately 6 mo of age and 260 kg, at .29, .58, 1.15, 1.73, 2.31, or 2.88 g potassium per kilogram body weight or 1.35, 2.12, or 2.16 g sodium per kilogram in equal volumes of water. Paired controls were infused with water. Calves were monitored for physiological changes for 6 h at 15, 30, or 60-min intervals. Potassium and total solids of plasma and packed cell volume were increased at potassium doses greater than .29 g of potassium per kilogram body weight within 1 h after dosing. At the higher doses of potassium, sodium content of plasma increased about 1 h after the increase in plasma potassium. Respiration rates within a potassium treatment varied with respect to time after dosing, but generally they increased, and associated variables of carbon dioxide pressure, pH, and bicarbonate in blood were decreased accordingly.

Clinical toxicity signs, including excess salivation, muscular tremors of legs, and excitability were observed with potassium doses greater than .58 g of potassium per kilogram body weight. Three of five calves given 1.73 g of potassium per kilogram, three of four calves given 2.31 g of potassium per kilogram, and one calf given 2.88 g of potassium per kilogram body weight died.

With a small number of calve, oral sodium infusions increased plasma sodium in proportion to the dose, but plasma potassium remained relatively constant. Sodium infusions of 2.12 and 2.16 g of sodium per kilogram body weight were fatal.

Deficiency manifestation

Potassium deficiency alters the function of the heart and blood vessels, nerves, muscles, gut, and kidneys. Overall, children and young adults tolerate hypokalemia better than elderly individuals do. Prompt correction is warranted in the presence of ischemic heart disease or in patients receiving digitalis.

Cardiovascular

Epidemiologic studies link a low-potassium diet with an increased prevalence of hypertension. Hypokalemia has been shown experimentally to increase blood pressure modestly (5 to 10 mm Hg), and similarly, potassium supplementation can lower blood pressure.Potassium deficiency probably increases blood pressure by stimulating sodium retention, with resultant intravascular volume expansion, and by sensitizing the vasculature to endogenous vasoconstrictors.7 In part, sodium retention is related to decreased expression of the kidney-specific isoform of WNK1, which leads to increased NCC- and ENaC-mediated sodium reabsorption in the distal convoluted tubule and cortical collecting duct, respectively.

Hypokalemia increases the risk for a variety of ventricular arrhythmias, including ventricular fibrillation.9 Diuretic-induced hypokalemia is of particular concern, as sudden cardiac death may occur more commonly in those treated with thiazide diuretics.Ventricular arrhythmias are also more common in hypokalemic patients receiving digoxin.

Hormonal

Hypokalemia impairs insulin release and also induces insulin resistance, resulting in worsened glucose control in diabetic patients.Experimental studies have demonstrated that the insulin resistance observed with thiazide diuretics is due to endothelial dysfunction mediated by thiazide-induced hypokalemia and hyperuricemia.

Muscular

Hypokalemia hyperpolarizes skeletal muscle cells, thereby impairing muscle contraction. Hypokalemia also reduces skeletal muscle blood flow, possibly by impairing local nitric oxide release, which can predispose patients to rhabdomyolysis during vigorous exercise.

Renal

Hypokalemia leads to several important disturbances of renal function. These include reduced medullary blood flow and increased renal vascular resistance that may predispose to hypertension, tubulointerstitial and cystic changes, alterations in acid-base balance, and impairment of renal concentrating mechanisms.

Tubulointerstitial and Cystic Changes

Potassium depletion causes tubulointerstitial fibrosis that is generally greatest in the outer medulla. Although usually reversible, it may result in renal failure. Experimental studies suggest that there is increased risk for irreversible renal injury in the neonatal period.Potassium depletion also causes renal hypertrophy and predisposes to renal cyst formation, particularly when there is increased mineralocorticoid activity.

Acid-Base

Metabolic alkalosis is a common acid-base consequence of potassium depletion and is due to increased renal net acid excretion.Conversely, metabolic alkalosis may increase renal potassium excretion, resulting in potassium depletion. Severe hypokalemia can lead to respiratory muscle weakness and the development of respiratory acidosis.

Polyuria

Severe hypokalemia also impairs concentrating ability, causing mild polyuria, typically 2 to 3 liters per day. Both increased thirst and mild nephrogenic diabetes insipidus contribute to the polyuria.

Hepatic Encephalopathy

Hypokalemia increases renal ammonia production, approximately half of which returns to the systemic circulation through the renal veins and may worsen hepatic encephalopathy.

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CALCIUM

Toxicity value

Voluminous literature data show that great interdependence exists between the nutrition status of the organism and the degree of accumulation and toxicity of heavy metals. In this work, the connection between dietary calcium and cadmium toxicity is discussed from the toxicological point of view. Cadmium is one of the most dangerous occupational and environmental poisons. The intake of diet containing an inappropriate amount of calcium causes increased absorption of cadmium from the gastrointestinal tract and increased accumulation of this metal in the organism, finally leading to enhancement of cadmium toxic action. The large intake of calcium protects against absorption, cumulation and toxicity of this heavy metal. Interactions between calcium and cadmium may take place at different stages of their metabolism (absorption, distribution in the organism, elimination) and cadmium may interfere with the biological functions of Ca2+ ions.

Deficiency Manifestation

Hypocalcemia, commonly known as calcium deficiency disease, occurs when calcium levels in the blood are low. A long-term deficiency can lead to dental changes, cataracts, alterations in the brain, and osteoporosis, which causes the bones to become brittle.

Complications of hypocalcemia can be life-threatening, and if the condition goes untreated, it could eventually lead to death.

A calcium deficiency may have no early symptoms. To avoid complications, a person should seek prompt diagnosis and treatment if they experience any of the symptoms listed below.

In this article, we also describe the prevalence of calcium deficiency disease, how to prevent it, and how it is treated.

Calcium is a vital mineral. Your body uses it to build strong bones and teeth. Calcium is also needed for your heart and other muscles to function properly. When you don’t get enough calcium, you increase your risk of developing disorders like:

osteoporosis

osteopenia

calcium deficiency disease (hypocalcemia)

Children who don’t get enough calcium may not grow to their full potential height as adults.

You should consume the recommended amount of calcium per day through the food you eat, supplements, or vitamins.

MAGNISIUM

TOXICITY VALUE

The toxicity of magnesium sulfate (MgSO(4)), and the influence of calcium (Ca), were assessed in very soft freshwater (natural Magela Creek water [NMCW]) using six freshwater species (Chlorella sp., Lemna aequinoctialis, Amerianna cumingi, Moinodaphnia macleayi, Hydra viridissima, and Mogurnda mogurnda). The study involved five stages: toxicity of MgSO(4) in NMCW, determination of the toxic ion, influence of Ca on Mg toxicity, toxicity of MgSO(4) at an Mg:Ca mass ratio of 9:1, and derivation of water quality guideline values for Mg. The toxicity of MgSO(4) was higher than previously reported, with chronic median inhibition concentration (IC50)/acute median lethal concentration (LC50) values ranging from 4 to 1,215 mg/L, as Mg. Experiments exposing the 3 most sensitive species (L. aequinoctialis, H. viridissima, and A. cumingi) to Na(2)SO(4) and MgCl(2) confirmed that Mg was the toxic ion. Additionally, Ca was shown to have an ameliorative effect on Mg toxicity. For L. aequinoctialis and H. viridissima, Mg toxicity at the IC50 concentration was eliminated at Mg:Ca (mass) ratios of < or =10:1 and < or =9:1, respectively. For A. cumingi, a 10 to 30% effect persisted at the IC50 concentration at Mg:Ca ratios <9:1. The toxicity of MgSO(4) in NMCW at a constant Mg:Ca ratio of 9:1 was lower than at background Ca, with chronic IC50/acute LC50 values from 96 to 4,054 mg/L, as Mg. Water quality guideline values for Mg (to protect 99% of species) at Mg:Ca mass ratios of >9:1 and < or =9:1 were 0.8 and 2.5 mg/L, respectively. Magnesium can be toxic at concentrations approaching natural background levels, but toxicity is dependent on Ca concentrations, with exposure in very low ionic concentration, Ca-deficient waters posing the greatest risk to aquatic life.

Deficiency manifestation

Magnesium deficiency is an electrolyte disturbance in which there is a low level of magnesium in the body. It can result in multiple symptoms.Symptoms include tremor, poor coordination, muscle spasms, loss of appetite, personality changes, and nystagmus.Complications may include seizures or cardiac arrest such as from torsade de pointes.Those with low magnesium often have low potassium.

Causes include low dietary intake, alcoholism, diarrhea, increased urinary loss, poor absorption from the intestines, and diabetes mellitus.A number of medications may also cause low magnesium, including proton pump inhibitors (PPIs) and furosemide.The diagnosis is typically based on finding low blood magnesium levels (hypomagnesemia).Normal magnesium levels are between 0.6-1.1 mmol/L (1.46–2.68 mg/dL) with levels less than 0.6 mmol/L (1.46 mg/dL) defining hypomagnesemia.[1] Specific electrocardiogram (ECG) changes may be seen.

Treatment is with magnesium either by mouth or intravenously.For those with severe symptoms, intravenous magnesium sulfate may be used.Associated low potassium or low calcium should also be treated.] The condition is relatively common among people in hospital.

CHLORIDE

Toxicity value

Freshwater mussels (order Unionoida) are one of the most imperiled groups of animals in the world. However, many ambient water quality criteria and other environmental guideline values do not include data for freshwater mussels, in part because mussel toxicity test methods are comparatively new and data may not have been available when criteria and guidelines were derived. The objectives of the present study were to evaluate the acute toxicity of sodium chloride (NaCl) and potassium chloride (KCl) to larvae (glochidia) and/or juveniles of a unionid mussel (fatmucket, Lampsilis siliquoidea), and to determine the potential influences of water hardness (50, 100, 200, and 300 mg/L as CaCO3) and other major ions (Ca, K, SO4, or HCO3) on the acute toxicity of NaCl to the mussels. From the KCl test, the 50% effect concentration (EC50) for fatmucket glochidia was 30 mg K/L, similar to or slightly lower than EC50s for juvenile fatmucket (37-46 mg K/L) tested previously in our laboratory. From the NaCl tests, the EC50s for glochidia increased from 441 to 1,597 mg Cl/L and the EC50s for juvenile mussels increased from 911 to 3,092 mg Cl/L with increasing water hardness from 50 to 300 mg/L. Increasing K from 0.4 to 1.9 mg/L, SO4 from 13 to 40 mg/L, or HCO3 from 44 to 200 mg/L in the 50 mg/L hardness water did not substantially change the NaCl EC50s for juvenile mussels, whereas increasing Ca from 9.9 to 42 mg/L increased the EC50s by a factor of 2. The overall results indicate that glochidia were equally or more sensitive to NaCl and KCl compared to juvenile mussels, and the increased water hardness ameliorated the acute toxicity of NaCl to glochidia and juveniles. These responses rank fatmucket among the most acutely sensitive freshwater organisms to NaCl and KCl.

Deficiency Manifestation

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.

IRON

TOXICITY VALUE

Iron poisoning is an iron overload caused by a large excess of iron intake and usually refers to an acute overload rather than a gradual one. The term has been primarily associated with young childrenwho consumed large quantities of iron supplement pills, which resemble sweets and are widely used, including by pregnant women; approximately 3 grams is lethal for a two-year-old. Targeted packaging restrictions in the US for supplement containers with over 250 mg elemental iron have existed since 1978, and recommendations for unit packaging have reduced the several iron poisoning fatalities per year to almost zero since 1998. No known cases of iron poisoning have been identified that are associated with iron mining.

DEFICIENCY MANIFESTATION

Iron deficiency anemia is a common type of anemia — a condition in which blood lacks adequate healthy red blood cells. Red blood cells carry oxygen to the body's tissues.

As the name implies, iron deficiency anemia is due to insufficient iron. Without enough iron, your body can't produce enough of a substance in red blood cells that enables them to carry oxygen (hemoglobin). As a result, iron deficiency anemia may leave you tired and short of breath.

You can usually correct iron deficiency anemia with iron supplementation. Sometimes additional tests or treatments for iron deficiency anemia are necessary, especially if your doctor suspects that you're bleeding internally.

Symptoms

Initially, iron deficiency anemia can be so mild that it goes unnoticed. But as the body becomes more deficient in iron and anemia worsens, the signs and symptoms intensify.

Iron deficiency anemia signs and symptoms may include:

Extreme fatigue

Weakness

Pale skin

Chest pain, fast heartbeat or shortness of breath

Headache, dizziness or lightheadedness

Cold hands and feet

Inflammation or soreness of your tongue

Brittle nails

Unusual cravings for non-nutritive substances, such as ice, dirt or starch

Poor appetite, especially in infants and children with iron deficiency anemia