Magnesium in Dogs

Magnesium sulfate at 50–150 mg/kg/day high dose

Regular dose 5-10mg per pound once per day.

Low magnesium: Lack of intake, lack of absorption from the gut, or failure to resorb from kidney. Common in ill patients and chronic inanition.

Magnesium (Mg) homeostasis is not under direct hormonal control but is mainly determined by absorption from the GI tract; excretion by the kidneys; and the varying requirements of the body for pregnancy, lactation, and growth. Magnesium is the second most common intracellular cation after potassium, with 50%–60% of total body Mg distributed in bone, 40%–50% in soft tissues, and <1% in the extracellular fluid. Therefore, plasma Mg does not provide an indication of intracellular or bone Mg stores. Intracellular Mg is required for activation of enzymes involving phosphate compounds such as ATPases, kinases, and phosphatases; and for synthesis of RNA, DNA, and protein. Magnesium is a cofactor for >300 enzymatic reactions involving ATP, including glycolysis and oxidative phosphorylation. It is also important in the function of the Na+/K+-ATPase pump, membrane stabilization, nerve conduction, ion transportation, and calcium channel activity. Magnesium also regulates the movement of calcium into smooth muscle cells, giving it a pivotal role in cardiac contractile strength and peripheral vascular tone. Low ionized Mg concentrations accelerate the transmission of nerve impulses. Clinical manifestations of severe hypomagnesemia include muscle weakness, muscle fasciculation, ventricular arrhythmias, seizures, ataxia, and coma.

Ruminants are more prone to hypomagnesemia than monogastric animals. The variation in Mg metabolism among species is mainly because of anatomic and physiologic differences in digestive tracts. Ruminants absorb Mg less efficiently than nonruminants (35% vs 70% of intake). The rumen is the main site of absorption, and there are active transport mechanisms. Absorption from the large intestine occurs with high Mg intakes. In nonruminants, the small intestine is the main site of absorption. Species differences in Mg metabolism are attributable to variation in both absorption efficiency of Mg from the gut and reabsorption of Mg by the kidney tubules.

Magnesium is cleared by glomerular filtration and, in the absence of renal disease, renal homeostatic mechanisms will attempt to maintain Mg balance. When the diet contains excessive Mg, renal tubular resorption decreases, maintaining serum concentrations of Mg within narrow physiologic limits. Renal excretion of Mg may be used to evaluate Mg balance.

IV administration of large doses of Mg is safely used to concurrently diagnose and treat hypomagnesemia. The IV Mg retention test, which involves the determination of urinary Mg retention, has become the gold standard to determine Mg status in human medicine and has been validated in horses, dogs, and cattle. Animals deficient in Mg will retain a large proportion of the administered Mg, whereas animals with sufficient Mg will excrete most of it.

Subclinical hypomagnesemia is common in critically ill horses and small animals and can increase the severity of the systemic inflammatory response syndrome; worsen the systemic response to endotoxin; and lead to ileus, cardiac arrhythmias, refractory hypokalemia, and hypocalcemia.

Low serum Mg concentrations have been reported to occur in 65% of critically ill people, 39%–46% of dogs and cats in the intensive care unit (ICU), 49% of hospitalized horses, 54% of equine surgical colic patients, and 78% of horses with enterocolitis. In human and canine ICU populations, hypomagnesemic patients had higher rates of concurrent hypokalemia and hyponatremia and a longer length of hospitalization. In another study, 54% of equine surgical colic patients had low iMg levels, and these horses had a significantly greater prevalence of postoperative ileus.

Although diets for horses and small animals are rarely deficient in Mg, subclinical acute hypomagnesemia is very common in critically ill animals. Serum Mg concentration may be low as a result of altered Mg homeostasis, cellular or third-space redistribution, GI loss of Mg, or diuresis secondary to aggressive fluid therapy with IV fluids unsupplemented with Mg.

Mild hypocalcemia and hypomagnesemia stimulate PTH release, but severe Mg depletion and acute hypermagnesemia decrease PTH release. Consequently, parallel determination of calcium and PTH concentrations is important in the investigation of Mg homeostasis.

If long-term fluid therapy is required to support an inappetent animal in the ICU, Mg should be supplemented. A constant rate infusion of Mg sulfate at 50–150 mg/kg/day, IV (0.1–0.3 mL/kg/day of the 50% solution), provides daily requirements.

Magnesium. It is thought that magnesium can help reduce allergic inflammation. While this may or may not be true, magnesium's real benefit is calming the nervous system and reducing anxiety. Many dogs with chronic allergies suffer secondarily from anxiety. I generally recommend magnesium citrate. However, magnesium glycinate may also be used.

Dose 10 mg/kg given once daily. (Alternate dose was 12.5mg/kg) and another dose as high as 50mg/kg

Recommended Brand: While I personally use Pure Encapsulations, there is an abundance of options for this particular supplement.