



FIGURE 72-10 The magnesium physiologic black box. See [Figure 72-1](#) for nomenclature and the text for details. Magnesium values are provided in milligrams (mg) and milliequivalents (mEq). GI, Gastrointestinal; ICF, intracellular fluid.

Many instances of magnesium deficiency are caused by excessive renal losses. Examples include the magnesuria that accompanies saline infusions, diuretic use, alcohol use, and secondary hyperaldosteronism states such as cirrhosis and ascites. As with calcium and phosphorus, the fractional excretion of magnesium (FE_{Mg}) can be calculated, and it should be used as an index of whether the kidney is appropriately conserving magnesium in states of hypomagnesaemia or whether renal magnesium wasting is the cause of the hypomagnesaemia. The normal FE_{Mg} is 2% to 4%. Hypomagnesemic individuals have FE_{Mg} values below 1% to 2%.

With regard to homeostatic regulation, magnesium homeostasis can best be viewed as a renal T_m -regulated process (see Renal Phosphate Handling), with the renal T_m for magnesium set at a fixed level of about 2.2 mg/dL. In this scenario, abundant dietary magnesium exists, and excessive magnesium intake is managed by spillage of excess magnesium over the T_m set at 2.2 mg/dL and into the urine. Conversely, in settings of dietary magnesium deficiency, which equate evolutionarily with caloric deficiency, short-term deficiency is prevented when serum levels fall below the renal T_m of 2.0 mg/dL. No known independent hormonal regulatory system for magnesium exists.

PROSPECTUS FOR THE FUTURE

Although it may seem that calcium, PTH, vitamin D, magnesium, and phosphorus homeostasis and skeletal biology are well understood, it should be clear that many of the physiologic details described in this chapter have been elucidated only during the past 10 to 15 years, and new regulatory proteins (e.g., fibroblast growth factor 23) and diseases continue to be identified. This area of research is dynamic, with many unanswered questions remaining.

SUGGESTED READINGS

- Christov M, Juppner H: Insights from genetic disorders of phosphate homeostasis, *Semin Nephrol* 33:143–157, 2013.
- Lentz RD, Brown DM, Kjellstrand CM: Treatment of severe hypophosphatemia, *Ann Intern Med* 89:941–944, 1978.
- Melmed S, Polonsky KS, Larsen PR, et al, editors: *Williams textbook of endocrinology*, ed 12, Philadelphia, 2012, Saunders.
- Rosen CJ, editor: *The American Society for Bone and Mineral Research primer on metabolic bone diseases and disorders of mineral metabolism*, ed 8, Washington, D.C., 2013, American Society for Bone and Mineral Research.