Magnesium is essential for the optimal function of a diversity of life-sustaining processes. It is cofactor of more than 300 enzymes, participating in the metabolism of carbohydrates, lipids, proteins, and nucleic acids, in the synthesis of hydrogen transporters, and particularly in all reactions involving the formation and use of adenosine triphosphate (ATP). Magnesium also serves as a regulator of many physiological functions, including neuromuscular, cardiovascular, immunological, and hormonal functions, as well as the maintenance of membrane stability.¹,²

Magnesium can participate in the reactions involving the formation and use of ATP by two different mechanisms. Kinetic studies of several enzymes requiring both magnesium and ATP showed that enzyme activity depends on the ratio, as well as on the absolute concentrations, of the two cofactors and that magnesium chelates strongly with ATP, forming a Mg(ATP) complex, the active substrate for enzyme action. Similarly, a magnesium complex with adenosine diphosphate (ADP), Mg(ADP), appears to be the active substrate for some enzymes. The second general mechanism of magnesium action is the direct binding of free magnesium (Mg²⁺) to the enzyme protein and resultant allosteric activation. With some enzymes magnesium has a dual function, not only forming part of the reactive substrate but also activating the enzyme allosterically. However, in some cases Mg(AMP) and Mg²⁺ may have opposite actions. For example, the Mg(AMP) complex stimulates the type-L calcium channels and Mg²⁺ inhibits them.

Magnesium is also known to alter both receptor sites and ion movements across the cell membrane. By making complexes with phospholipids, magnesium stabilizes the membranes, reducing their fluidity and permeability. Thus, magnesium is an important modulator of intracellular ion concentrations. In magnesium deficit, intracellular concentrations of calcium and sodium increase, and concentrations of potassium and phosphorus decrease. Simultaneously, the membrane depolarizes. These alterations may be the result of magnesium’s direct effect on sodium, calcium, or potassium channels or the indirect result of its effect on enzymes in the cell membrane that are involved in active transport, for example,
(Na⁺K⁺)—ATPase. Magnesium also regulates lipid and phosphoinositide-derived second messengers.

Within the cell, magnesium affects the function of organelles such as sarcoplasmatic reticulum, primarily by its ability to alter calcium flux, or mitochondria, by altering their membrane's permeability to protons, which leads to alterations in the coupling of oxidative phosphorylation and electron transport chains, thus affecting the efficiency of ATP production. Magnesium also acts as a calcium antagonist. In the neuromuscular system it reduces the electric excitability of the neurons and inhibits the release of acetylcholine by the nerve endings at the neuromuscular junction, and blocks the effect of N-methyl-D-aspartate, an excitatory neurotransmitter of the central nervous system. In muscle contraction, both stimulation and the activity of the calcium transport system in the sarcoplasmatic reticulum membranes depend on the presence of Mg²⁺. Troponin contains four calcium-binding sites, two of which have a high affinity for calcium and bind Mg²⁺ competitively. These calcium-magnesium type sites do not seem directly involved in any rapid twitching mechanism, but play a structural role in muscle. Magnesium bound to these sites may maintain the protein permanently in a particular conformational state regardless of the fluctuation in calcium (assuming that both magnesium and calcium-induced structural changes are essentially the same). This conformation may be a prerequisite for calcium activation via binding at the calcium-specific sites.

These and many other functions make it easy to understand why performing exercise is highly dependent on the regulation of magnesium homeostasis. Additionally, there is evidence that exercise performance seems to be impaired under conditions of magnesium deficiency. This is why individuals performing exercise should pay extra attention to magnesium status.

**Athletes and Magnesium Status**

Frequently, physically active individuals fail to consume a diet that contains adequate amounts of minerals, including magnesium, which leads to marginal nutrient deficiency and results in substandard training and impaired performance. Additionally, mineral losses in urine and sweat are more important during exercise than at rest. These conditions may contribute to frequent mineral deficits among athletes.

During exercise, compartmental shifts of magnesium have been observed, but data to demonstrate magnesium variations induced by exercise are inconsistent. Such heterogeneity can be partially attributed to differences in experimental designs and work intensity and duration. Moreover, the timing of blood sample and the different analytical protocols have to be taken into account.

With respect to blood extracellular magnesium, various authors have indicated that high-intensity exercise leads to hypermagnesemia as a consequence of the decrease in plasma volume. These changes may depend on the relative contribution of anaerobic metabolism to the total energy expended during exercise.