Possibly, magnesium deficiency contributes to diminished host defenses in the elderly (1). Elin (5) showed profound immunosuppressive capability in magnesium-deficient mice that had low numbers of antibody-synthesizing cells and low serum immunoglobulin concentrations. He cited studies in magnesium-depleted rats with decreased serum γ globulin and immunoglobulin G concentrations.

I suggest that magnesium deficiency in elderly patients contributes to cachexia. Magnesium deficiency puts the patient at risk of increased activity of damaging mediators that contribute to cachexia (eg, inflammatory cytokines, PGE2, and oxygen free radicals) and it reduces the tissues’ antioxidant capacity. Magnesium deficiency may increase the risk for cognitive and psychiatric disorders, which may be amenable to magnesium therapy (10).

It may be prudent to evaluate the magnesium status of elderly patients with cachexia. If these subjects are magnesium deficient, magnesium supplementation may be beneficial because magnesium supplementation was shown to rapidly reverse the clinical symptoms (anorexia, apathy, weakness, and weight loss) of severely malnourished, magnesium-deficient children (11). However, numerous factors must be considered in geriatric cachexia. The chronicity of marginal or inadequate intakes of iron, calcium, magnesium, and zinc; the prolonged use of medications; and the reduced absorption or metabolism of nutrients may adversely affect the nutritional status of the elderly (9). The effects of magnesium supplementation late in the course of magnesium deficiency in the elderly are not known, but are worthy of investigation.

Joan L Caddell

Department of Pediatrics
Thomas Jefferson University
706A Jefferson Medical College Building
1025 Walnut Street
Philadelphia, PA 19107-5083
E-mail: joan.caddell@mail.tju.edu

REFERENCES


Reply to JL Caddell

Dear Sir:

We thank Caddell for pointing out the important role of magnesium deficiency in cachexia. Although the focus of our review was on the cytokines themselves, we agree that there are numerous factors to be considered in geriatric cachexia (1). Inadequate food intake, reduced absorption, cytokine production, and medications can all affect the nutritional status of the elderly. Thi- azide diuretics and loop diuretics are among the most common medications taken by the elderly and they clearly can cause a loss of magnesium. This loss may be overlooked because of apparently normal serum magnesium concentrations. However, when skeletal muscle biopsies are performed, subnormal magnesium and potassium concentrations are found (2). Insufficient dietary supplies of magnesium may inhibit protein synthesis by decreasing serum insulin-like growth factor I (2). Therefore, minerals like magnesium and vitamins are important supplements in the treatment of cachexia.

We agree that magnesium deficiency may exacerbate the elevation of inflammatory cytokines caused by other etiologies. Furthermore, magnesium deficiency may decrease endogenous antioxidant capacity and diminish host defenses. Magnesium deficiency may play an essential role in cellular reactions and in immunoinflammatory processes (3). Magnesium deficiency can also affect mineral homeostasis, induce membrane damage, increase lipid peroxidation, and increase cytokine concentrations, thus reducing immunocompetence (4). Weglicki et al (5–7) found that dramatic elevations in interleukin 6, interleukin 1, and tumor necrosis factor α may promote cardiac lesions in magnesium-deficient rodents. This activation of immune cells probably occurs early in magnesium deficiency because magnesium-deficient rats that received magnesium-replacement therapy before endotoxin challenge had significantly lower tumor necrosis factor α production than controls (3). It was also noted that vitamin E supplements can prevent the occurrence of myocardium reperfusion injury, possibly through the restoration of endogenous antioxidant defenses in the hypomagnesemic state (7).

Stress and chronic inflammation, under conditions of mineral or antioxidant deficiency, probably further stimulate the secretion of catecholamines and cortisol, which then stimulate the release of cytokines. As we emphasized in our review, cytokines rarely act alone because they stimulate a variety of cell types to produce and secrete a cascade of other cytokines (1). All of these interactions point to the complex roles of cytokines in causing cachexia (1). The effects of magnesium supplementation, the use of antioxidants, as well as the use
of cytokine inhibitors in the treatment of geriatric cachexia require further study.

Shing-Shing Yeh

VA Medical Center
Geriatric Division
Department of Medicine
79 Middleville Road
Northport, NY 11768-2290
E-mail: shingshing.yeh@med.va.gov

Michael W Schuster

Weill Medical College
Cornell University
New York Presbyterian Hospital
520 E 70th St
Starr 341
New York, NY 10021

REFERENCES

Adolphe Quetelet

Dear Sir:

I read with interest the supplement to the July 1999 issue of The American Journal of Clinical Nutrition, entitled “Assessment of Childhood and Adolescent Obesity.” Most of the authors used the body mass index as an assessment tool; Franklin used the Benn index. When Adolphe Quetelet proposed what is now known as the body mass index in his Physique Sociale in 1869, he indicated that it applied to adults (1, 2). During the developmental period of both sexes, the index was wt2/ht5.

It might be interesting to recrunch the numbers and see what results this produces. And, because what is now called the body mass index is rarely called Quetelet’s index, let’s honor the man who proposed both by naming the one for children and adolescents for Quetelet.

Emma S Weigley

327 South Smedley Street
Philadelphia, PA 19103-6717

REFERENCES