Taking a BITE Out of Weight Loss

“A great-aunt of mine died at the age of 93. Although she had been confined to bed for some time her faculties were still well preserved, and the only evidence of her condition was the decrease in appetite…and in half an hour she fell into her last sleep.” — Brillat-Savanin

As described by Brillat-Savanin, for so many older persons it is the loss of the desire to eat that leads to a loss of enjoyment of life, weight loss and eventually death. The “anorexia-cachexia” syndrome is characterized by poor appetite and a progressive depletion of caloric stores, body fat, and muscle. Older persons often develop this “wasting syndrome” which is associated with an extremely poor quality of life. The GAIN registry demonstrated that in a thousand persons living in nursing homes around the United States, from Washington State to Florida, over 30% of those who continued to lose weight died, whereas over 90% of those who reversed their weight loss survived (Figure 1). Numerous studies have shown that weight loss in older persons leads to increased morbidity and mortality. Older persons with protein-energy malnutrition are more likely to have pressure ulcers. (continued on page 2)

Nutrition remains an extremely understudied area of medicine and one in which physicians often have a paucity of knowledge and practical advice. Much of what passes for modern knowledge was already entrenched in the literature of medicine at the start of the modern era. Thus Aretaeus the Cappadocian described the anorexia of aging thus, “…for in old men, even without any disease, owing to their being near the close of life, the appetite is nearly gone” and correctly attributes its pathogenesis (continued on page 20)
cers, immune dysfunction, infections, hip fracture, cognitive abnormalities, anemia, muscle weakness, fatigue and edema. In particular caloric depletion due to anorexia leads to very low $\text{CD}_4^+/\text{CD}_8^+$ T-cell ratio reminiscent of the levels seen in patients with acquired immuno-deficiency syndrome (AIDS). The Cochrane Database (2002) examined 2,464 persons in 31 trials and found that, when calories are ingested as a supplement, mortality decreased and length of hospitalization declined. Progressive wasting due to insufficient caloric ingestion appears to be a hallmark of impending death. Over the last decade much evidence has emerged that both anorexia seen in older persons and the excessive muscle wasting (cachexia syndrome) are due to the excess production of cytokines.

**Cytokines and Aging**

Cytokines are peptides which are produced by every nucleated cell in the body and act as intercellular messengers marshalling the ability of the host to defend itself and repair damaged tissues. The family of cytokines consists of at least 27 interleukins, the interferons, tumor necrosis factors, transforming growth factor B and the cellular stimulating factors.

When cytokines were first discovered in the 1960s, they were thought to only serve as chemical messengers within the immune system. Subsequently it has become clear that they also have direct effects on the central nervous system, muscle, bone and other tissues.

Excess cytokines not only produce anorexia and sickness behavior but also pull protein off muscle and calcium off bone, decrease red blood cell production, inhibit memory retention, inhibit albumin production and cause extravasation of albumin from blood vessels (Figure 2). This last effect is the predominant reason why cytokine excess is associated with hypoalbuminemia.

With aging, stress leads to a shift from the Th-1 to Th-2 cytokine response. Classically interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF$\alpha$) tend to be increased during the physiological aging process.

**Cytokines, Sarcopenia and Function**

Irving Rosenberg from Tufts University coined the term sarcopenia (sarc = flesh; penia = loss) for the excessive muscle loss that occurs in aging.
It is now well documented that many older persons have a physiological anorexia associated with aging that puts them at major risk for developing severe protein energy malnutrition when disease occurs. According to the National Health and Nutrition Examination Survey (NHANES), up to 16% of Americans over the age of 65 ingest less than 1000 calories per day.

This small caloric intake is incompatible with maintaining adequate vitamin and trace mineral intake. The prevalence of protein energy malnutrition and its associated vitamin deficiencies is extremely high in hospitalized older persons (ranging from 12–65%) and in 10 to 85% of nursing home residents. Malnutrition is particularly common in older persons in transitional or subacute care units.

In older persons attending an outpatient clinic, approximately 10% have some degree of malnutrition. While most studies have concentrated on the prevalence of protein energy malnutrition, a few have specifically concentrated on vitamin deficiency. In one study of noninstitutionalized older men, the prevalence of vitamin deficiency was directly related to the individuals having a low caloric intake. In those ingesting less than 21.5 kcal/kg body weight per day, more than 20% had vitamin A, folate, vitamin B₂, and vitamin B₆ deficiency. Those ingesting greater amounts of calories were only at risk for folate and vitamin B₆ deficiency. As might be expected, the highest level of deficiencies occurs in hospitalized and institutionalized older persons. Not surprisingly, the old old tend to have higher levels of vitamin deficiency than the young old. Studies that have looked at vitamin deficiency in independent older persons include the Survey in Europe on Nutrition in the Elderly (SEN-ECA), which included persons 70 to 75 years, the Boston Nutritional Status Survey with 686 persons over age 60, the New Mexico Longitudinal Process Study with 304 healthy elderly persons over age 65 and the German VERA study of 2006 persons aged 75 to 97 years. In these studies, the frequency of vitamin B deficiency varies from 0% for niacin in one study to 56% for pyridoxine in another study. Vitamin A deficiency occurs in 1% or less of the population, even though low intakes have been found in 4 to 17% of the population. While vitamin A deficiency is a rare occurrence, low beta-carotene levels were found in 10.3% of the population. Vitamin D deficiency occurred in 2.6 to 6.8% of the population. In general, hospitalized old persons and those in institutions tend to have lower vitamin levels and deficiencies.

### Table 1. PREVALENCE OF VITAMIN DEFICIENCIES IN OLDER PERSONS

<table>
<thead>
<tr>
<th>Vitamin</th>
<th>Independent</th>
<th>Hospitalized</th>
<th>Nursing Home</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A</td>
<td>1%</td>
<td>?</td>
<td>?</td>
</tr>
<tr>
<td>B₁ (thiamine)</td>
<td>13-43%</td>
<td>40%</td>
<td>2-5%</td>
</tr>
<tr>
<td>B₂ (riboflavin)</td>
<td>3-42%</td>
<td>12%</td>
<td>1-34%</td>
</tr>
<tr>
<td>B₆ (Pyridoxine)</td>
<td>5-56%</td>
<td>19%</td>
<td>21-93%</td>
</tr>
<tr>
<td>Folate</td>
<td>2.5-34%</td>
<td>24%</td>
<td>4-24%</td>
</tr>
<tr>
<td>Vitamin B₁₂ (cyanocobalamin)</td>
<td>4-43%</td>
<td>?</td>
<td>4-29%</td>
</tr>
<tr>
<td>Vitamin C (ascorbic acid)</td>
<td>?</td>
<td>?</td>
<td>0-5%</td>
</tr>
<tr>
<td>Vitamin D³</td>
<td>2-5%</td>
<td>22%</td>
<td>35%</td>
</tr>
</tbody>
</table>

(?)=unknown

Based on measured levels as intake interacts with sunlight.
greater degrees of deficiencies. Table 1 summarizes the presence of vitamin deficiencies in these populations. In persons reporting the ingestion of vitamin supplements there are fewer vitamin deficiencies. However, levels of deficiency as high as 6% for thiamine, 13% for folate, 18% for vitamin B₁₂, 33% for niacin, and 39% for pyridoxine have been found in some of these populations (presumably related to only occasional use).

**DIETARY REFERENCE INTAKES**

Dietary reference intakes (DRIs) were developed by the Food and Nutrition Board of the Institute of Medicine of the National Academy of Sciences and published in 1998. For the first time, this group published data for individuals aged 70 years and older. The committee utilized recent research on requirements for older persons in making their determinations.

They described 4 kinds of reference values: recommended dietary allowance (RDA), adequate intake (AI), tolerable upper intake level (TUL) and estimated average requirement (EAR). RDAs represent the daily dietary intake necessary to meet the nutrient requirements of 97 to 98% of healthy people of a certain age. An AI, which is based on the judgment of an expert panel, is established when there is insufficient evidence to determine an RDA. EARs represent the amount of nutrient intake that will meet the requirements of 50% of healthy individuals. The RDA is set at 2 standard deviations above the EAR. The TUL represents the nutrient intake value for chronic daily use that is unlikely to produce risks of adverse effects in the vast majority of individuals. The DRIs for vitamins in persons over age 70 are given in Table 2.

**VITAMIN DEFICIENCIES: THEIR ROLE IN OLDER PERSONS**

Table 3 (see page 5) provides an overview of the effects of vitamin deficiencies in older persons. It should be remembered that in most elderly persons, severe deficiency of any single vitamin is unusual and it is the synergistic effects of multiple subclinical vitamin deficiencies that result in deleterious effects.

**Thiamine.** Thiamine is a coenzyme necessary for the transfer of energy in the oxidative decarboxylation of alpha-keto acid and transketolase degradation. A study in New Zealand found that 43.4% of 222 older persons had levels of thiamine pyrophosphate below the fifth percentile of younger subjects, and that, three years later, the thiamine pyrophosphate levels had fallen another 20%. The researchers concluded that aging per se played a major role in the reduction of thiamine levels. Other causes of thiamine deficiency include alcoholism and diuretic use. Cardiac failure has been associated with thiamine deficiency in one study but not in another. Thiamine levels are normal in type 2 diabetic patients. Risks of acute thiamine deficiency are increased during hospitalization,

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**Table 2. Dietary Reference Intakes for Vitamins in Persons Over Age 70 and Major Functions of the Vitamins**

<table>
<thead>
<tr>
<th>VITAMIN</th>
<th>RDA*</th>
<th>AI*</th>
<th>TUL*</th>
<th>FUNCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiamine</td>
<td>1.3</td>
<td>1.1</td>
<td>-</td>
<td>Carbohydrate and branched chain amino acid metabolism</td>
</tr>
<tr>
<td>(µg/d)</td>
<td>(males)</td>
<td>(females)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Riboflavin</td>
<td>1.3</td>
<td>1.1</td>
<td>-</td>
<td>Oxidation/Reduction reactions</td>
</tr>
<tr>
<td>(µg/d)</td>
<td>(males)</td>
<td>(females)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Niacin</td>
<td>16</td>
<td>14</td>
<td>35</td>
<td>Dehydrogenase reactions</td>
</tr>
<tr>
<td>(µg/d)</td>
<td>(males)</td>
<td>(females)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin B₆</td>
<td>1.7</td>
<td>1.5</td>
<td>-</td>
<td>Metabolism of amino acids glycogen and sphingoid bases</td>
</tr>
<tr>
<td>(mg/d)</td>
<td>(males)</td>
<td>(females)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Folate</td>
<td>400</td>
<td>-</td>
<td>1000</td>
<td>Metabolism of nucleic and amino acids</td>
</tr>
<tr>
<td>(mg/d)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin B₁₂</td>
<td>2.4</td>
<td>-</td>
<td>-</td>
<td>Metabolism of odd chain fatty acids and methyl transfer</td>
</tr>
<tr>
<td>(µg/d)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pantothentic Acid</td>
<td>-</td>
<td>5</td>
<td>-</td>
<td>Fatty acid metabolism</td>
</tr>
<tr>
<td>(mg/d)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Biotin</td>
<td>-</td>
<td>30</td>
<td>-</td>
<td>Bicarbonate-dependent carboxylase reactions</td>
</tr>
<tr>
<td>(µg/d)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin D³</td>
<td>-</td>
<td>15</td>
<td>50</td>
<td>Regulation of calcium and phosphorous</td>
</tr>
<tr>
<td>(µg/d)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*RDA = Recommended dietary allowance which is the average daily dietary intake level sufficient to meet the nutrient requirements of 97 to 98% of healthy individuals.  
AI = Adequate Intake is a proxy for the RDA when insufficient evidence is available to determine the RDA.  
TUL = Tolerable Upper Intake Level which is the highest safe intake level for almost all of the population.  
†Applies only to supplements or forms obtained from fortified foods.  
‡30 to 50% of older persons malabsorb food-bound vitamin B₁₂ and thus should meet the RDA by eating food fortified with vitamin B₁₂ or by utilizing supplements.

(continued from page 3)
since the provision of intravenous glucose solutions can result in increased glucose utilization for energy metabolism. This results in the increased utilization of thiamine stores and can precipitate thiamine deficiency in the older person with marginal thiamine status. This may be particularly true in older persons who are using alcohol or taking diuretics.

The major signs of mild thiamine deficiency are delirium and peripheral neuropathy (particularly loss of vibration and position senses), which may lead to falls and possibly aggravation of underlying heart failure. A severe thiamine deficiency eventually leads to Wernicke’s encephalopathy with a sixth nerve palsy and coma. Thiamine deficiency is also a cause of high output heart failure (beriberi). Long-term thiamine deficiency in alcoholics has been associated with Korsakoff’s psychosis, which involves destruction of the mamillary bodies and is characterized by cognitive impairment and confabulation.

Two studies in older persons have suggested that thiamine supplementation improved cognitive function and overall quality of life in older persons. The role of subclinical thiamine deficiency in delirium, heart failure, and falls in older persons remains to be determined; but available evidence suggests that this is not unlikely, particularly in the hospitalized older person. The availability of a direct erythrocyte thiamine pyrophosphate assay to replace the old transketolase activity coefficient should make it possible to determine more accurately the role of thiamine deficiency in older persons.

Riboflavin (vitamin B2). Riboflavin acts with its coenzymes, flavin mononucleotide and flavin adenine dinucleotide in many oxidation-reduction reactions. There is some evidence that riboflavin requirements may increase with aging and that erythrocyte glutathione reductase activity declines with aging.

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**Table 3. CLINICAL FEATURES OF VITAMIN DEFICIENCY**

<table>
<thead>
<tr>
<th>VITAMIN</th>
<th>CLINICAL FEATURES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fat Soluble</td>
<td></td>
</tr>
<tr>
<td>Vitamin A</td>
<td>Keratinization of lung, GI tract, and urinary tract epithelia, night blindness,</td>
</tr>
<tr>
<td></td>
<td>keratomalacia, xerophthalmia, increased susceptibility to infections</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>Osteomalacia, osteoporosis, muscle weakness, bone and muscle tenderness,</td>
</tr>
<tr>
<td></td>
<td>stooped posture, loss of height</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>Peripheral neuropathy, cerebellar ataxia, hemolytic anemia</td>
</tr>
<tr>
<td>Water Soluble</td>
<td></td>
</tr>
<tr>
<td>Thiamine</td>
<td>Fatigue, irritation, sleep disturbance, delirium, coma, sixth nerve palsy,</td>
</tr>
<tr>
<td></td>
<td>cardiac failure, loss of vibration and position senses</td>
</tr>
<tr>
<td>Riboflavin</td>
<td>Cheilosis, oral, ocular, cutaneous, and genital lesions, conjunctival inflammation,</td>
</tr>
<tr>
<td></td>
<td>dyssebacea, anemia</td>
</tr>
<tr>
<td>Niacin</td>
<td>Photosensitive dermatitis, dementia, peripheral neuropathy, glossitis, diarrhea,</td>
</tr>
<tr>
<td></td>
<td>constipation, organic psychosis</td>
</tr>
<tr>
<td>Pyridoxine</td>
<td>Glossitis, peripheral neuropathy, lymphopenia, seborrheic dermatosis, cheilosis</td>
</tr>
<tr>
<td>Folate</td>
<td>Malabsorption, delirium, megaloblastic anemia, diarrhea</td>
</tr>
<tr>
<td>Vitamin B12</td>
<td>Loss of position and vibration senses, ataxia, optic atrophy, anemia, diarrhea,</td>
</tr>
<tr>
<td></td>
<td>cognitive impairment, anorexia, constipation, glossitis</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>Bruising, poor wound healing, hemorrhosis, bleeding gums, petachiae</td>
</tr>
<tr>
<td>Vitamin K</td>
<td>Hypoprothrombinemia (increased bleeding tendency)</td>
</tr>
</tbody>
</table>

Persons with riboflavin deficiency may have a magenta tongue, mucocutaneous fissure (cheilosis), conjunctival inflammation, and anemia. Riboflavin may play a role in the pathogenesis of age-related cataract development. Riboflavin deficiency usually occurs in combination with other vitamin deficiencies. The major cause of riboflavin deficiency in older persons is poor dietary intake.

Niacin. Niacin plays a role in dehydrogenase reactions as a coenzyme for nicotinamide adenine dinucleotide and nicotinamide adenine dinucleotide. **(continued on page 9)**
some older persons. Sarcopenia is associated with a decline in muscle strength and in resting metabolic rate as well as physical inactivity and is the calling card of the frailty syndrome in older persons. Persons with sarcopenia are likely to have more impaired function than those without sarcopenia.

The causes of sarcopenia are multifactorial and include physical inactivity, anorexia, low testosterone, low growth hormone and insulin-growth factor-1, peripheral vascular disease, mitochondrial DNA deletion mutations (resulting in increased oxidative damage leading to intrafiber atrophy and fiber breakage), and cytokine excess. It has been suggested that myostatin, a hormone that inhibits myoblast proliferation, may act excessively in older persons, resulting in sarcopenia. Testosterone causes the mesenchymal pluripotential cells to differentiate as muscle satellite (repair) cells rather than into adipocytes. Cytokines, particularly interleukin-6 (IL-6) and tumor necrosis factor α (TNFα), result in protein loss from muscle. Thus, it is a combination of factors that lead to sarcopenia. The pathophysiology of sarcopenia and its consequences are reviewed in depth in a series of articles appearing in The Journals of Gerontology Series A: Medical Sciences in the October and November issues of 2003.

Elevated levels of inflammatory markers have been associated with increased morbidity and mortality in older persons. For example, persons with elevated levels of IL-6 have been found to be at greater risk of development of disability. In patients with rheumatoid arthritis, chronic obstructive pulmonary disease and heart failure, both TNFα and IL-6 are associated with reduced lean body mass. The Health ABC Study found that older persons with high levels of TNFα or IL-6 had smaller muscle area, less appendicular muscle mass, lower knee extensor strength and less grip strength compared to those with normal cytokine levels. Overall, these results strongly suggest a key role for cytokines in the pathogenesis of sarcopenia.

**Anorexia of Aging**

In ancient Roman times, Cicero pointed out that “I am grateful to old age because it has increased my desire for good conversation and decreased my interest in good food.” The concept of a physiological anorexia of aging, as first hypothesized by Morley in 1988, is now well established. The causes of this physiological anorexia of aging are multifactorial and include altered taste and smell, increased stretch of the stomach antrum secondary to decreased fundal compliance, increased release of the satiety hormone (cholecystokinin) and decreased release of the orexigenic hormone (ghrelin), a decline in testosterone, and an increase in circulating leptin levels. In addition to this, as most older persons have some level of chronic inflammatory disease (e.g., arthritis, sinusitis), the release of anorectic cytokines further leads to anorexia.

Thus, when older persons develop a disease process such as cancer, cardiac failure, and chronic obstructive pulmonary disease, they are already set up to develop severe anorexia and cachexia. However, there are many conditions more amenable to cure that also produce anorexia in older persons.

Many of these treatable conditions are also associated with cytokine production, and thus, while awaiting the effect of treating the primary disease, it is also useful to lower the excess cytokine burden.
Guidelines for Good Health

By Natalie Moretz, MS, RD

Eat several times a day.
Try not to eat a large amount of food at one meal. Eat more calories in the beginning of the day rather than at night.

Eat 3-5 vegetables every day.
Choose colorful, fresh or frozen vegetables when cooking or snacking – such as spinach, tomatoes, broccoli, squash, carrots, cabbage, etc.

Stay at a good weight.
Both weight loss and weight gain are dangerous for the elderly.

Eat 2 to 3 fruits every day.
Use fresh or frozen fruits, which are a good source of fiber. Juice does not have the fiber you need, but may count toward your liquid intake.

Make good choices of fat.
Stay away from saturated fats or animal fats. Good fat choices are vegetable oil, olive oil, or liquid margarine. Use liquid or tub margarine instead of stick. Your goal should be only 1-2 fat choices per meal.

Increase fiber-rich foods in your diet.
Fiber has been shown to lower blood sugar levels and cholesterol. Good sources include berries, dried beans, prunes, whole wheat bread, brown rice, bran, raisins, fresh fruits, vegetables, and more.

Drink plenty of liquids.
This is very important. Try to drink at least 6 - 8 cups of liquid each day.

Keep avoiding simple sugars.
Limit refined starches like white flour, low fiber cereals, and sweets like cakes, cookies, candy, sodas, and fruit punches.

Include lean protein 2 to 3 times each day.
Portion size should approximate the size of the palm of your hand. Limit high-fat meats like bologna, sausage, and bacon. Eat fish four times weekly.

Use spices as desired.
Black pepper, thyme, garlic, cumin, onion, turmeric, oregano, basil, sage, ginger, etc.

Exercise.
It’s good to exercise at least four times a week. Start out slow until you reach 30 minutes or more per day.

Adapted from a handout by Sandy Long, RD. VA Medical Center, Marion, IL
Orexigenic Agents

Cyproheptadine acetate was the original orexigenic (appetite-enhancing) drug used in nursing homes. Its effects were disappointing and it was often associated with delirium. Anabolic agents, such as growth hormone, do reverse weight loss and promote nitrogen retention but have a number of side effects such as arthralgias and carpal tunnel syndrome. In a large study in intensive care units, growth hormone resulted in increased mortality in malnourished patients. Anabolic steroid hormones, such as testosterone may have a role in the treatment of sarcopenia or, when used in combination with drugs that block cytokine production, in the treatment of anorexia-cachexia “wasting” syndromes. Megestrol acetate lowers testosterone in males and thus consideration should be given to providing testosterone injections together with megestrol acetate in males. Anecdotally, this combination has been successful in the management of some patients with AIDS. Oral anabolic agents have the propensity to produce hepatotoxicity and increased creatinine levels. Before these agents are widely used, there is a need for large studies of their use in older persons with the anorexia-cachexia syndrome.

Dronabinol is a terahydrocannabinol (active cannabis extract) drug. It has been shown to stimulate appetite to a small degree in patients with AIDS and cancer. It appears to produce its effects by activating the endogenous cannabis (CB-1 or anandamide) receptor. A single study showed a small, nonsignificant weight gain in older nursing home residents. In addition, it has an antinausea effect, produces a mild decrease in pain and can enhance general well-being. In our experience, this drug can be very useful in end-of-life care.

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At present the only available

(continued from page 6)

(continued on page 14)
VITAMIN DEFICIENCY

(continued from page 5)

otide phosphate. Garry and colleagues found that 54% of community-living older persons had intakes of less than three fourths of the RDA. The assays to determine niacin nutritive status are of limited value. Niacin deficiency in older persons was found to be between 1% and 50% when using the urinary excretion of N-methyl nicotinamide as a proxy.

In developed countries niacin deficiency occurs predominantly in association with alcoholism. Isoniazid, used for the treatment of tuberculosis, can also induce niacin deficiency. As niacin can be obtained from tryptophan conversion, persons with the carcinoid syndrome can develop niacin deficiency.

Full-blown niacin deficiency presents as pellagra. This is characterized by the three D’s: dementia, dermatitis, and diarrhea, although constipation is more common. Dermatitis most often presents as hyperpigmented, cracked skin in sun-exposed areas. Niacin deficiency is also associated with a peripheral neuropathy. Low niacin levels are associated with increased mortality in older persons.

Pyridoxine (vitamin B₆): Pyridoxine is a cofactor in numerous reactions involved in intermediary metabolism. In a healthy, upper middle class population in New Mexico, 61% of older women ingested less than 1 mg of pyridoxine a day and 54% of the men ingested less than 1.1 mg. In Boston, low dietary intake was found in 56% of subjects. At least 5% of the healthy elderly population has low pyridoxal-5-phosphate levels and in a nursing home study, 93% were found to have low levels. Pyridoxal phosphate levels decline with age. Pyridoxal-5-phosphate levels are reduced in diabetes mellitus with up to one-third of diabetics having a vitamin B₆ deficiency.

Pyridoxine deficiency is associated with glossitis, peripheral neuropathy, cheilosis, lymphopenia, and seborrheic dermatosis. Depletion studies of vitamin B₆ resulted in a deterioration of immune function in older persons. Pyridoxine is a cofactor for cystathionine synthease and thus low pyridoxine levels can lead to elevated homocysteine. Elevated homocysteine levels have been associated with accelerated atherosclerosis. Low intake of pyridoxine is associated with increased risk of coronary artery disease and myocardial infarction. Vitamin B₆ deficiency has also been associated with carbohydrate intolerance. Deijen and colleagues found that vitamin B₆ supplementation in older persons resulted in improvement of long-term memory by improving storage of information. Overall subclinical pyridoxine deficiency appears to have the potential to produce a variety of deleterious effects in the frail and/or demented older persons.

Folate: Folate plays a role in single carbon atom transport in intermediary metabolism. The 1998 DRIs increased the RDA for folate to 400 mg for both men and women from the 1989 RDAs of 200 mg for men and 180 mg for women. The RDA for folate is the same for both young and old adults.

Folate absorption is not age-dependent but it appears to decline in persons with atrophic gastritis. High alcohol intake severely blocks folic and metabolic pathways and represents one of the major reasons for folate deficiency. Numerous drugs including cotrimazole, phenytoin, phenobarbital, methotrexate, and triamterene interfere with folate metabolism.

In the United States, older men take in an average of 300 mg of folate a day, which is less than the RDA. In most studies of elderly persons, folate deficiency is about 2.5%, but other studies have found levels of 7.8% to 34%.

Folate deficiency can result in megaloblastic anemia, malabsorption, leukopenia, thrombocytopenia, glossitis, anorexia, fatigue, diarrhea, delirium, and/or de-
mentia. Folate is the major vitamin determinant of elevated homocysteine levels which are considered a major risk factor for the development of atherosclerosis. Homocysteine inhibits collagen cross-linking, activates protoagulants, is cytotoxic to blood vessels, and plays a role in oxidation damage produced by the metabolism of LDL cholesterol. In the Framingham study, hyperhomocysteinemia was present in 14% of subjects aged 67 to 96. Elevated homocysteine levels are strongly correlated with coronary artery disease as well as dementia.

Some studies suggest that folate deficiency is linked to depression and other neuropsychiatric disorders. Overall, the evidence for the involvement of folate deficiency in a variety of disorders is very strong. Serum and/or red-cell folate levels along with homocysteine levels should be assessed in older persons with delirium, dementia and atherosclerosis and vigorously treated if found to be low.

**Table 4. STUDIES ON EFFECTS OF VITAMIN ADMINISTRATION IN OLDER SUBJECTS**

<table>
<thead>
<tr>
<th>AUTHOR, DATE</th>
<th>TREATMENT</th>
<th>OUTCOME</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>COGNITION</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eastley et al, 2000</td>
<td>Vitamin B₁₂</td>
<td>Improvement in mid cognitive impairment patients on verbal fluency test</td>
</tr>
<tr>
<td>Hassing et al, 1999</td>
<td>Folic acid</td>
<td>Improvement in impairment of word recall and object recall</td>
</tr>
<tr>
<td>Deijen et al, 1992</td>
<td>Vitamin B₆</td>
<td>Improvement in long-term memory</td>
</tr>
<tr>
<td>Smidt et al, 1991</td>
<td>Thiamine</td>
<td>Improvement in cognitive function</td>
</tr>
<tr>
<td>Wilkinson et al, 1997</td>
<td>Thiamine</td>
<td>Improved cognition</td>
</tr>
<tr>
<td><strong>IMMUNE FUNCTION</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Buzina-Suboticanec, 1988</td>
<td>Multivitamin</td>
<td>Improvement in delayed cutaneous hypersensitivity</td>
</tr>
<tr>
<td>Chandra, 1993</td>
<td>Vitamin-trace element supplement</td>
<td>Increased natural killer cells, proliferation of white cells following mitogen exposure, increased interleukin-2 production, and decreased infections</td>
</tr>
<tr>
<td>Pike and Chandra, 1995</td>
<td>Vitamin-trace</td>
<td>Increase in CD57 and no decrease in CD4 T cells compared to placebo</td>
</tr>
<tr>
<td>Bogden et al, 1994</td>
<td>Vitamin-trace element supplement</td>
<td>Improved delayed cutaneous hypersensitivity</td>
</tr>
<tr>
<td>Chavance et al, 1993</td>
<td>Multivitamin</td>
<td>No difference in infections</td>
</tr>
<tr>
<td><strong>BIOCHEMICAL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mann et al, 1987</td>
<td>Multivitamin</td>
<td>Increase in vitamins C, B₁₂, B₂, and folate, but no effect on vitamins A and E</td>
</tr>
<tr>
<td>Kauwell et al, 2000</td>
<td>Folate</td>
<td>415 mg/d of folate, but not 200 mg/d</td>
</tr>
<tr>
<td>van der Wielen et al, 1995</td>
<td>Fortified fruit juice</td>
<td>Reduced serum homocysteine levels</td>
</tr>
<tr>
<td><strong>HIP FRACTURE</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chapuy et al, 1994</td>
<td>Vitamin D and calcium</td>
<td>Decreased hip fracture and decreased mortality rate</td>
</tr>
</tbody>
</table>

**Vitamin B₁₂** (cyanocobalamin): Vitamin B₁₂ plays a role in the metabolism of odd-chain fatty acids and methyl transfer. Vitamin B₁₂ deficiency results in an elevation of both homocysteine and methylmalonic acid. Low serum vitamin B₁₂ levels were found in 2.7% of older persons in the SENECA study and in 5% in the Boston Nutritional Survey. Vitamin B₁₂ levels have been observed to decline at the rate of about 3.4 pmol/L annually in the elderly. Food-bound vitamin B₁₂ is poorly absorbed with aging, making deficiency more common in those older individuals with atrophic gastritis. Bacterial overgrowth has also been demonstrated to inhibit vitamin B₁₂ absorption. However, following treatment with antibiotics, absorption is enhanced. Pernicious anemia (intrinsic factor deficiency) can occur in up to 5% of 80 year olds in some populations. Regional ileitis or other forms of ileal disease can also decrease vitamin B₁₂ absorption. Drugs, including chronic use of H₂ antagonists, colchicine, aminosalicylic acid, and a variety of anesthetic agents, can interfere with vitamin B₁₂ metabolism.

Vitamin B₁₂ deficiency can result in megaloblastic anemia. More commonly, a vitamin B₁₂ deficiency produces cognitive impairment in older persons, which may occur without any evidence of megaloblastic anemia. Vitamin B₁₂ deficiency also produces subacute combined degeneration of the spinal cord with a loss of both position and vibration senses. Optic atrophy, ataxia, and elevated homocysteine levels can also be seen in persons with vitamin B₁₂ deficiency.

Many older persons with borderline normal vitamin B₁₂ levels have subclinical signs of deficiency. Thus, it is recommended that the presence or absence of elevated vitamin B₁₂ levels be monitored in these patients. Vitamin B₁₂ replacement can be done by injection or nasal spray in those persons lacking intrinsic factor and possibly by high-dose oral therapy.

(continued on page 11)
Vitamin C. Vitamin C’s major function is to protect cells against oxidants. Vitamin C levels decline with age in whole blood, serum, and white cells. Low vitamin C levels have been associated with cancer, cataracts and atherosclerosis. Scurvy is rarely seen in older persons. However, bruising (sometimes known as “senile” purpura) often responds to vitamin C treatment.

Vitamin K. Vitamin K plays an important role in coagulation and in the formation of δ-carboxyglutamate. The major sources of vitamin K are green vegetables, grain products, meats, and fats. Coagulation defects suggestive of vitamin K deficiency have been found in up to one half of older persons. Vitamin K deficiency may also result in more fragile bones.

Vitamin A. Vitamin A is involved in vision, epithelial cell differentiation, and growth. Decline in lycopene has been associated with a decline in functional status. The association between vitamin A and beta-carotene and cancer remains controversial.

Vitamin D. Vitamin D not only plays an important role in maintenance of bone metabolism but also in muscle metabolism and immune function. A longitudinal study has demonstrated that vitamin D levels decline with age even in healthy older persons. In community-living older persons very low vitamin D levels can be found especially during the colder months. Hospitalized and institutionalized older persons commonly have vitamin D deficiency.

The reasons for vitamin D deficiency in older persons are multifactorial (see figure on page 9). They include a decrease in sunlight exposure, use of sunblock, decreased skin synthesis of cholecalciferol, decreased vitamin D intake, decreased 1α-hydroxylation of vitamin D in the kidney, and altered binding capacity for vitamin D metabolites in the serum. There is also evidence for vitamin D receptor resistance in the gastrointestinal tract. Medications, such as phenytoin, accelerate metabolism of 25(OH) vitamin D in the liver. Glucocorticoids and chronic renal failure inhibit the activity of 1α-hydroxylase in the kidney.

The combination of vitamin D and calcium represents the first approach to the prevention of osteopenia in older persons. Chapuy and her colleagues showed that vitamin D and calcium supplementation reduces hip fractures in nursing home residents. Vitamin D and calcium increase bone mineral density in community dwelling elders.

Vitamin E. Vitamin E is a major endogenous antioxidant and also alters prostaglandin metabolism, thus modulating the immune system. Vitamin E deficiency is very uncommon in older persons. Vitamin E, at least in pharmacological doses, has been considered protective against a number of age-related diseases. Vitamin E inhibits platelet aggregation and limits the damage produced by oxidized LDL cholesterol, suggesting a possible role in the treatment of atherosclerosis-associated diseases.

<table>
<thead>
<tr>
<th>TABLE 5. DRUGS THAT ALTER VITAMIN FUNCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A - Tetracycline, mineral oil, neomycin</td>
</tr>
<tr>
<td>Vitamin D - Phenytoin, mineral oil, steroids, cholestyramine</td>
</tr>
<tr>
<td>Vitamin E - Iron, vitamin A, vitamin K</td>
</tr>
<tr>
<td>Thiamine - Alcohol</td>
</tr>
<tr>
<td>Riboflavin - Alcohol, phenothiazines, tricyclic antidepressants</td>
</tr>
<tr>
<td>Pyridoxine - Isoniazid, phenobarbital, phenytoin, hydralazine</td>
</tr>
<tr>
<td>Niacin - HMG-CoA reductase inhibitors</td>
</tr>
<tr>
<td>Folate - Sulfasalazine, alcohol, phenytoin, phenobarbital</td>
</tr>
<tr>
<td>Vitamin B₁₂ - H₂ antagonists, colchicine, antibiotics</td>
</tr>
</tbody>
</table>

VITAMIN REPLACEMENT STUDIES IN OLDER PERSONS

A number of studies have examined the effects of vitamin replacement in older persons (See Table 4 on page 10). Overall, these studies have demonstrated positive effects on cognition, immune function, prevalence of infection, decreased hip fractures, and a decline in homocysteine levels. The studies that examine immune function have classically involved either a multivitamin or a vitamin-trace mineral supplement. The studies on cognition have involved a single vitamin and include positive effects of vitamin B₁₂, folic acid, vitamin B₆ and thiamine. Chapuy and her colleagues demonstrated that vitamin D and calcium supplementation decreased hip fractures and mortality. High levels of folate decreased homocysteine levels. Mann and his colleagues showed that a multivitamin supplement could improve vitamin nutrition in older persons.
WEIGHT LOSS SHOULD BE A

**Pathogenesis of Anorexia**

-albumin
- cytokines
- leptin
- cholecystokinin
- testosterone
- ghrelin
- nitric oxide release
- fundal relaxation
- early antral filling
- early satiation

**Pathogenesis of Sarcopenia**

- anorexia
- creatine
- testosterone
- IGF-1
- vascular disease
- cytokines
- muscle isoform

**CHANGES IN METABOLIC RATE WITH AGING**

Effects of aging on energy expenditure. BAT = brown adipose tissue; RMR = resting metabolic rate

![Graph showing changes in metabolic rate with aging](image)
### Treatable Causes of Weight Loss

<table>
<thead>
<tr>
<th>Medications</th>
<th>Emotional (depression)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anorexia tardive, alcoholism, abuse</td>
<td></td>
</tr>
<tr>
<td>Late-life paranoia</td>
<td></td>
</tr>
<tr>
<td>Wallowing problems</td>
<td></td>
</tr>
<tr>
<td>Oral problems</td>
<td></td>
</tr>
<tr>
<td>Nosocomial infections, no money (poverty)</td>
<td></td>
</tr>
<tr>
<td>Wandering and other dementia-related behaviors</td>
<td></td>
</tr>
<tr>
<td>Hyperthyroidism, hypoadrenalism, hyperglycemia, hypercalcemia, hypertension (pheochromocytoma)</td>
<td></td>
</tr>
<tr>
<td>Enteral problems</td>
<td></td>
</tr>
<tr>
<td>Eating problems</td>
<td></td>
</tr>
<tr>
<td>Low salt, low cholesterol diet</td>
<td></td>
</tr>
<tr>
<td>Stones, shopping problems</td>
<td></td>
</tr>
</tbody>
</table>

### Management of Weight Loss

1. Treat underlying disease
2. Caloric supplements between meals
3. Orexigenic agents
   - Mirtazapine (Remeron®)
     - 15 to 30 mg daily for persons with depression
   - Dronabinol (Marinol®)
     - 2.5 mg once or twice daily predominantly for palliative care
   - Megestrol acetate (Megace®)
     - 800 mg liquid daily; females respond better than males
4. Consider enteral feeding, peripheral parenteral nutrition, or total parenteral nutrition in selected cases
drug that has consistently produced weight gain is megestrol acetate.

**Megestrol Acetate**

Megestrol acetate is a semi-synthetic progestational steroid. In 1987, megestrol acetate was suggested as a treatment for cachexia in cancer patients after observations of weight gain in women with metastatic breast cancer. In 1993, it was approved by the Federal Drug Administration (FDA) for treating weight loss in patients with AIDS. Megestrol acetate is now well established as a treatment for cachexia in patients with cancer and AIDS. It accounts for the vast majority of orexigenic prescriptions in nursing homes.

Megestrol acetate increases appetite rapidly, promotes significant weight gain within the first few weeks, and improves quality of life. The outcomes of some of the larger studies utilizing megestrol acetate for the treatment of protein energy malnutrition are summarized in Table 1 (see page 8). A systematic review of the effects of high dose progestins in 15 trials comprised of over 2000 patients showed significant weight gain.

In malnourished nursing home patients, Yeh and her colleagues showed that a daily dose of 800 mg of megestrol acetate improved appetite and well being and significantly increased weight. Two other retrospective studies in nursing home patients 71 to 100 years of age showed significant weight gain. In the Saint Louis University study, the patients had significant increase in food intake, body mass index, serum albumin, prealbumin, hemoglobin, and absolute lymphocyte count (Figure 4).

Side effects of megestrol acetate use have been rare, with edema being the single most commonly reported effect. Occasionally patients on megestrol acetate for prolonged periods develop adrenocortical crisis. In patients with cancer or AIDS this also can occur as a complication of the disease process. Megestrol acetate is a progestational agent and, as such, may be expected to increase the occurrence of deep vein thrombosis. While two retrospective studies have suggested this might be the case, the control groups have had an inappropriately low incidence of deep vein thrombosis. Deep vein thrombosis is extraordinarily common in nursing home residents and an indepth analysis of these studies by David Thomas found that if these studies were compared to the true incidence of deep vein thrombosis in nursing homes, the incidence in the patients receiving megestrol acetate would have, in fact, been lower! Megestrol acetate in males produces an increase in adipose tissue compared to lean tissue mass. This appears to be due to the lowering of testosterone and suggests that depot testosterone (200 mg/ml every 2 weeks intramuscularly) could be given in males receiving megestrol acetate. However no longterm controlled trials exist and megestrol acetate has increased weight in males in the absence of concomitant testosterone therapy. Megestrol acetate also occasionally causes hypoglycemia, but we have not found this to be a problem in nursing home residents.

It is now well established that megestrol acetate decreases cytokine production and that this represents the major mechanism by which it produces appetite and weight gain. Studies in older persons have suggested (continued on page 22)
The Division of Geriatrics at Saint Louis University hosted the inaugural class of the first United States-based Geriatric Academy where practicing geriatricians received enhanced post-graduate education in geriatrics and leadership on January 5-9, 2004.

These Scholars traveled through snowstorms and rain from Massachusetts, Connecticut, South Carolina, Pennsylvania, Illinois, Texas, Tennessee, West Virginia, and Missouri to attend the Academy.

In all, 30 geriatricians received instruction in (1) Leadership and Management, (2) General Geriatrics, (3) Education Methodology, (4) Geriatric Systems and Continuous Quality Improvement (CQI), (5) Patient-Oriented Research, and (6) Health Systems and Community Networking.

Guest lecturers included Dr. Eric Tangalos, of the Mayo Clinic, who spoke on leadership and presented case studies in ethics in the nursing home. Dr. Larry Lawhorne, of Michigan State University, spoke about nursing home medical direction. Dr. Lawrence Rubenstein, of the University of California - Los Angeles, provided case studies on Gait and Balance and a lecture on Falls Prevention. Saint Louis University faculty in finance, business, medicine, nursing, and public health also provided instruction through plenary presentations, workshops, (continued on page 19)
Leptin is a hormone produced by fat cells that reduces food intake and increases metabolic activity. When it was discovered, it created much excitement as an agent that would cure the modern obesity epidemic. Unfortunately, obese persons were found to be resistant to the effects of leptin.

William Banks and John Morley at the St. Louis VA Medical Center GRECC have recently shown that the leptin resistance is due to the elevated triglycerides that are present in obese persons. Lowering triglycerides allows leptin to pass across the blood-brain barrier and thus to effectively overcome leptin resistance.

ZINC DEFICIENCY SIMPLIFIED

**CAUSES**

Decreased intake  
Lung cancer  
Cirrhosis of liver  
Diabetes  
Alcoholism  
Diuretics

**EFFECTS**

Hypogonadism  
Decreased immune function  
Diarrhea  
Impared night vision  
Impared taste  
Anorexia  
Poor wound healing  
Impared immune function  
Irritability/Apathy  
Low ZINC
Berlin Hosts Second Cachexia Conference in December, 2003

The three-day conference began on December 4 with Robert Schwartz pointing out that with aging, weight loss is more closely associated with mortality than is weight gain. Similarly, a low body mass index in older persons is a very poor prognostic factor. A number of presentations highlighted the factors involved in the pathogenesis of anorexia. Cytokines were considered the major cause of disease-associated anorexia and it was pointed out that progestagens, corticosteroids, and n-3 fatty acids inhibit cytokines and enhance food intake. Of newer agents under development, interest focused on the melanocortin system and the stomach hormone, ghrelin.

A number of presentations focused on disease specific muscle wasting. There was a focus on the devastating effects of weight loss and cachexia in chronic heart failure. Professor Stefan Anker pointed out that the presence of more than six percent weight loss most closely relates to subsequently impaired survival and should be used to diagnose cardiac cachexia. He also pointed out that low cholesterol and elevated uric acid were associated with high mortality in cardiac failure. Other cachexia-associated diseases are AIDS wasting, rheumatoid arthritis, chronic liver failure, renal failure, and chronic lung disease. Dr. Berry said that in the presence of cachexia, there is an increase in length of hospitalization and in over-all health care costs.

John Morley reviewed the changes of energy metabolism with aging. The decline in resting metabolic rate with aging is predominantly due to age-related loss of muscle mass. However, decrease in energy metabolism is also due to a decline in the activity of the Na⁺K⁺ATPase energy pump, decreased protein turnover and changes in mitochondrial membrane protein permeability. Physical energy expenditure also declines with aging. There are also small declines in meal-induced thermogenesis and in adaptive thermogenesis. William Evans reported that megestrol acetate, a synthetic progestin, stimulated appetite and resulted in weight gain in malnourished older persons. He has also found that albuterol, a β2 adrenergic agonist, increased muscle mass in Parkinson’s patients.

Sarcopenia, an excess of muscle loss in aging humans, has multiple causes including anorexia, hypogonadism in men, atherosclerosis, decreased creatine intake, low growth hormone and muscle IGF-1, free radical damage, and cytokine excess. Professor Rosenthal has elegantly demonstrated that the muscle isoform of IGF-1 plays a key role in preventing the age-related decline in muscle loss. Cytokines have multiple effects on muscle as demonstrated in the figure on page 18.

A number of presentations examined the presently available therapies for cachexia. Appropriate treatment of the underlying disease was stressed. Professor Tisdale pointed out that the lipid mobilizing factor and the proteolysis-inducing factor which are produced by human cancers, can have their effects inhibited by eicosapentaenoic acid. Erythropoetin enhances exercise capacity in anemic persons with cancer cachexia. Dr. Jatoi related that two ongoing trials using TNFα inhibitor...
hibitors (infliximab and etanercept) are examining their role in the treatment of cancer cachexia.

Professor Von Roenn reviewed the available appetite stimulants. Cyproheptadine, hydrazine sulfate, and melatonin were considered ineffective. Eicosapentaenoic acid and thalidomide (a cytokine antagonist) may have some effects, but minimal data are available at present. Corticosteroids increase appetite but fail to produce weight gain and the development of proximal muscle weakness limits its usefulness. Dronabinol is an effective appetite stimulant but it appears to have less ability to produce weight gain than megestrol acetate. Megestrol acetate increases food intake, produces weight gain, and enhances quality of life. Side effects include testosterone deficiency, adrenal insufficiency, and thrombosis. A poster presentation by Dr. Yeh suggested that in older males, megestrol acetate may enhance survival over the first year, but not over four years.

Growth hormone has been shown to increase muscle mass and function in one study in AIDS patients. While an open label study showed weight gain with oxandrolone, a placebo-controlled study failed to show a significant effect of the drug on weight gain.

Overall, this was an exciting conference organized by Professor Anker. It certainly highlighted the need to actively treat weight loss associated with disease and also offered hope of exciting new therapeutic possibilities on the horizon.
Joseph Flaherty, MD, of the Division of Geriatric Medicine at SLU, talks about the development of CLINICAL GLIDEPATH™ TOOLS for robust, frail, demented, and end-of-life patients.

After class each day, the Scholars braved the cold January weather and joined faculty members for dinner. This group joined Dr. Flaherty for a tour of Mari de Villa, a planned community with independent, assisted-living, and nursing home facilities, which was followed by dinner at the site.

composed the poem below to thank the SLU faculty for their week-long efforts.

To apply for the next session of the Geriatrics Academy which will be held in St. Louis on July 12-16, 2004, please send a letter of interest and your curriculum vita to:

tumosan@slu.edu or
Saint Louis University Geriatrics Academy
Division of Geriatric Medicine
Attn: Nina Tumosa, PhD
1402 S Grand Blvd, Room M238
St. Louis MO 63104

Ode to Our Mentors

St. Louis is known for its beer, But we have now discovered SLU
The teaching was incredible and we appreciate all the edibles.
We can’t forget the food, and after Dr. Thomas’s talks, we won’t get sued.

Dr. Morley, we can’t keep up with your pace,
But to stay on scheduled time, we will need Foley, PEG, and IV line.
We give special thanks to Nina for keeping us from all getting angina.
Let’s not forget Dr. Wilson’s teaching flare and her special tour of Life Care.
To Rafi and your mother, We wouldn’t have one without the other.

Dr. Flaherty taught us about delirium and let’s not forget the Glidepath eponym.
In your case discussions, Dr. Philpot, you emphasized what was really hot.
Dr. Morley, your teaching was fantastic, but on day 5, our brains have turned to plastic.
In sum, we thank you, great faculty of SLU for imparting to us the best of the geriatric clues.
to the stomach, “…the stomach is the president of pleasure and digest.” Maimonides, the Hebrew physician, clearly distinguished between sarcopenia and cachexia, “… for wasting which resembles old age (sarcopenia), and wasting which should be curtailed. The epidemic of obesity in the United States is occurring at middle age when most individuals are relatively anorectic and thus the solution is not to continue to decrease food intake but rather to increase exercise! Many years ago, Willett demonstrated that the more we eat the less likely we are to get cardiovascular disease.

Magical potions (vitamins and minerals in ridiculous doses in pill form) have become the modern alchemists’ tools to prolong life and to defraud the aging public. Yet few good studies exist on the effects of these nutrients taken over prolonged periods of time in healthy elderly.

Certainly epidemiological studies suggest that eating fish four or more times per week may improve health and longevity. However we are now being warned that toxins are accumulating in unsafe proportions in fish due to the failure to appropriately regulate the discharge of toxic effluent from our industries’ factories.

Fortunately, as pointed out by Maimonides, “wine…to the elderly is extremely beneficial…the most salutary of these wines are those which are particularly warming…and which have a red appearance.” With the rediscovery of the French paradox, there is an emerging belief that alcohol in moderation slows the appearance of atherosclerosis, strengthens the bones, and perhaps even enhances cognition. I have always enjoyed visiting nursing homes in Europe where wine is served with the meals and in one case, was even produced on the land around the nursing home specifically for the enjoyment of its occupants.

With these thoughts in mind, we are particularly happy to produce this issue of Aging Successfully which is dedicated to nutrition in older persons. We are grateful to PAR Pharmaceuticals for their unrestricted educational grant that allowed the production of this issue. Finally, we would like to dedicate this issue to the consultant dietitians who have been the leaders in enhancing nutritional care of older persons in nursing homes.

John E. Mosley
CONCLUSION

The data reviewed here clearly suggest that vitamin deficiency is not rare in community-living elderly persons. When older persons are admitted to the hospital there is a further increase in vitamin deficiency. While classic vitamin deficiency states are unusual in older persons in the United States, lesser levels of combined hypovitaminosis are common and can present subtly or with atypical manifestations. The causes of vitamin deficiency in older persons include the chronic causes of protein energy malnutrition or because of increased needs, such as those that occur with hypermetabolic states (infection, malignancy, thyroid disease and diabetes mellitus), alcohol use, and liver disease, and increased vitamin excretion associated with proteinuria, diuretics, or laxative abuse. In addition, a variety of drugs interact with vitamins leading to vitamin deficiencies or toxicities (see Table 5 on page 11).

Based on these findings, it would seem that many older persons would benefit from vitamin supplementation. While most healthy elderly have little difficulty absorbing oral vitamin supplements, there is some evidence suggesting that ill older persons might absorb vitamins poorly and require parenteral administration. This raises the question of whether or not the majority of older persons admitted to the hospital should receive intravenous vitamin supplementation. The recent study by Sullivan and his colleagues suggesting extraordinary poor oral intakes would further support this viewpoint. Whether or not such vitamin supplementation would decrease the incidence of delirium or nosocomial infections is deserving of further study.

Geriatric Medicine Introduces New Faculty

Dr. Julie Gammack joined the Division of Geriatric Medicine at Saint Louis University as an Assistant Professor in late 2003. A graduate of the University of Minnesota Medical School, she did her Internship/Residency and a Fellowship in Geriatric Medicine at the University of Washington School of Medicine in Seattle, Washington. Following a two-year stint as an Assistant Professor at Baylor College of Medicine, she has returned to the Midwest just in time for winter.

Dr. Akeeb Adadokum brings his expertise in geriatric rehabilitation to the Division of Geriatric Medicine at Saint Louis University. He received his medical training at the Ahmadu Bello University School of Medicine in Zaria, Nigeria. He completed his residency in Family Medicine at Meharry Medical College in Tennessee, and a Fellowship in Geriatric Medicine at the University of Louisville, Kentucky.

Dr. Ramzi Hajjar returns to the Division of Geriatric Medicine from the sunny state of Florida. Dr. Hajjar first completed his residency and subsequently his geriatric fellowship at SLU. Dr. Hajjar worked as an Assistant Professor at the University of South Florida and a geriatric primary care physician at the Bay Pines VA Medical Center in Bay Pines, Florida, where he received the Instructor of the Year teaching award. Welcome back, Dr. Hajjar!
that megestrol acetate decreases IL-6 levels. In addition, in older patients weight gain correlated with a fall in sIL-2R and TNFR-p75 levels.

For weight gain, the appropriate dose is 800 mg daily of the liquid form of megestrol acetate. This dosing should be continued for at least 3 to 4 months. Caution should be taken in its administration to bed-bound patients at increased risk of deep vein thrombosis.

**Conclusion**

Weight loss in a nursing home resident is a sentinel event utilized by state and federal oversight agencies to determine problems with quality of care.

Much evidence has now accumulated to suggest that cytokine excess is a major cause of the anorexia-cachexia syndrome. Megestrol acetate has been shown to inhibit cytokine production, enhance appetite and produce weight gain. In some studies it has also been shown to improve quality of life. Based on the Saint Louis University Nursing Home study and a review of the literature, it was concluded that “…megestrol acetate is an efficient and safe means for improving the treatment-resistant malnutrition of older persons in long-term care facilities.”

---

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Crossword Puzzle Book
Challenges and Choices
Aging Successfully Newsletter
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13th Annual Multi-Disciplinary Certificate Program in Geriatrics for Non- Physicians

Fridays, March 12, 26, April 16, 30, May 14, 28, 2004 at the Holiday Inn Brandywine, Peoria, Illinois

Fridays, March 19, April 2, 9, 23, May 7, 21, 2004 at the Stonegate Conference Center, Hoffman Estates, Illinois

Fridays, February 27, March 12, 26, April 9, 23, 30, 2004 at the Holliday Inn, Collinsville, Illinois

Saint Louis University Geriatric Academy (SLUGA)
July 12-16, 2004
(see page 15)

Meet Me in St. Louis
15th Annual Saint Louis University Summer Geriatric Institute
June 1-3, 2004

Improve Chronic Care Quality
September 10-11, 2004 in Columbia, Missouri

All of the conferences will be held at Saint Louis University, except as noted. For more information about any of these conferences, please call 314-977-8848.

Been Here? Done This?
Offering regular updates on geriatrics, Cyberounds, an internet-based educational program for physicians and other health providers, is edited by Dr. John E. Morley. The internet address for Cyberounds is: www.cyberounds.com

A cybersite for seniors has been developed in collaboration with Saint Louis University and the Gateway Geriatric Education Center. Besides articles written by geriatric experts, this site provides health updates and an interactive question and answer section. The address for this site is www.thedoctorwillseeyounow. See you in cyberspace!
This newsletter is a publication of:
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Department of Internal Medicine
Saint Louis University School of Medicine
Geriatric Research, Education, and Clinical Center (GRECC)
St. Louis Veterans Affairs Medical Center
Gateway Geriatric Education Center of Missouri and Illinois (Gateway GEC)
(supported by a grant from the Bureau of Health Professions, Health Resources and Services Administration)

With special thanks to PAR Pharmaceuticals

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