Seventeen

Status and Functional Impact of Nutrition in Older Adults

Jeffrey B. Blumberg

1. Introduction

Nutrition has been recognized as an important factor influencing the functional outcome of aging. The adherence to particular dietary patterns by older as well as younger adults can affect theterminal years of the life span. Conversely, aging is accompanied by a variety of physiological, psychological, economic, and social changes that compromise nutritional status and/or affect nutritional requirements (Munro & Danford, 1989). Nutritional status surveys of the elderly have shown a relatively low prevalence of frank nutrient deficiencies, but a marked increase in the risk of malnutrition and evidence of subclinical deficiencies with a direct impact on function (Rosenberg, 1995). Nutrition is also an important factor in the progressive changes in body composition associated with aging, such as the loss of bone and lean body mass (Cumlea & Baumberger, 1989). Moreover, the evidence is now undisputed that diet and nutrition are directly linked to many of the chronic diseases afflicting older and elderly adults (Nutrition Policy Board, 1988; Committee on Diet and Health, 1989). Thus, nutrition now represents an important part of the solution to the demographic challenge posed by the growing population of older adults to the public health policy need for aging that affects the morbidity and mortality of older adults (Schneider & Brod, 1990).

Aging produces physiological changes that affect the need for several essential nutrients (Munro & Schierer, 1989). Although most standards of nutritional requirements for older adults are based upon extrapolation from the recommendations for younger adults, accumulating evidence that indicates dietary goals are necessary to achieve optimal health in later life (Blumberg, 1989). Some of the apparent age-related changes in body composition and physiological function that appear to influence nutritional requirements in older adults are presented in Table I. In addition, decrements in senses of taste and smell can have significant impact on food selection and nutrient intake. Sedentary lifestyles, drug use, social isolation, physical disability, and chronic diseases can also substantially affect food choices, interfere with food preparation, and impair nutritional status.

The absence of validated age-specific values for anthropometric, biochem-
Table 1
Examples of Age-Related Changes in Body Composition and Physiological Function
That Influence Nutrient Requirements

<table>
<thead>
<tr>
<th>Change in body composition or physiological function</th>
<th>Impact on nutrient requirement</th>
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<tbody>
<tr>
<td>Decreased muscle mass (sarcopenia)</td>
<td>Decreased need for calories</td>
</tr>
<tr>
<td>Decreased bone density (osteopenia)</td>
<td>Increased need for calcium and vitamin D</td>
</tr>
<tr>
<td>Decreased immune function</td>
<td>Increased need for vitamins B, and E and zinc</td>
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<tr>
<td>Increased gastric pH (atrophic gastritis)</td>
<td>Increased need for vitamin B₁₂, folic acid, calcium, iron, and zinc</td>
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<tr>
<td>Decreased skin capacity for cholecalciferol synthesis</td>
<td>Increased need for vitamin D</td>
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<tr>
<td>Increased vitamin D production</td>
<td>Increased need for vitamin D</td>
</tr>
<tr>
<td>Decreased calcium bioavailability</td>
<td>Increased need for calcium and vitamin D</td>
</tr>
<tr>
<td>Decreased hepatic uptake of retinol</td>
<td>Decreased need for vitamin A</td>
</tr>
<tr>
<td>Decreased efficiency in metabolic utilization of pyridoxal</td>
<td>Increased need for vitamin B₆</td>
</tr>
<tr>
<td>Increased oxidative stress status</td>
<td>Increased need for β-carotene, vitamin C, and vitamin E</td>
</tr>
<tr>
<td>Increased level of homocysteine</td>
<td>Increased need for folate, vitamin B₁₂, and vitamin B₁₅</td>
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and clinical standards has always con-
founded an adequate nutritional assessment of elderly individuals and the older adult population. Indeed, many standards of interpretation have been based on studies of younger populations. Research approaches in nutrition have begun to focus on measures of nutrient intake and status not merely as normative means but as part of an effort to identify and predict the functional and health consequences of these assessments. Thus, while the chapter on nutrition and aging in the previous edition of this series appropriately emphasized the nutritional status of the elderly (Ausman & Russell, 1990), attention here is devoted to the impact of selected nutrients on physiological function and the risk of chronic disease.

II. Body Composition

A. Bone Density and Fractures

The major determinants of bone volume include genetics and the status of sex hormones, exercise, and calcium and vitamin D nutrition (Nelson et al., 1991; Nelson et al., 1988). Peak bone mass is achieved at about age 23, but as much as 40% of skeletal calcium can be lost thereafter; in women, almost half of this loss occurs within 5 years after menopause. The age-related decline in the capacity to absorb calcium (Bullamore, Wilkinson, Gallagher, Nordin, & Marshall, 1970) appears to be due to a gut resistance to the action of 1,25-dihydroxyvitamin D₃, resulting from a loss of vitamin D receptors in the duodenal mucosa (Ebeling et al., 1992). A decrease in the renal production of 1,25-dihydroxyvitamin D₃ with age has also been reported (Tsai, Heath, Kumar, &iggs, 1984). Important, et. al. also list situations common to older people compromise their calcium nutrition and help to explain the striking deficiency of dietary calcium among persons aged 65 years and older. For example, lactose intolerance leads to the avoidance of dairy products and increased fiber intake for laxation impairs calcium bioavailability (Knox et al., 1991), as does atrophic gastritis (Recker, 1985).

Oral tests of physiological [but not pharmacological] doses of cholecalciferol indi-
I. Introduction

Aging has been recognized as an important factor influencing the functional and cognitive aspects of aging. The adherence to particular patterns by older adults can affect the terminal points of the life span. Conversely, aging is influenced by a variety of physiological, psychological, economic, and social factors that compromise nutritional status and influence nutritional requirements. For example, the prevalence of frank nutritional deficiencies, such as a low prevalence of protein-energy malnutrition, is increasing in the elderly. Although most standards of nutritional requirements for older adults continue to be based upon extrapolation from the recommendations for younger adults, evidence is accumulating that indicates that different dietary goals are necessary to achieve optimal health in later life. Aging produces physiological changes that affect the need for several essential nutrients (Munro & Schiller, 1992). Although most standards of nutritional requirements for older adults continue to be based upon extrapolation from the recommendations for younger adults, evidence is accumulating that indicates different dietary goals are necessary to achieve optimal health in later life (Blumberg, 1991). Some of the apparent age-related changes in body composition and physiological function that appear to influence nutrient requirements in older adults are listed in Table 1. In addition, decrements in the senses of taste and smell can have a significant impact on food selection and nutrient intake. Sedentary lifestyles, drug therapies, social isolation, physical disabilities, and chronic diseases can also substantially affect food choices, interfere with shopping and cooking, and impair nutritional status.

The absence of validated age-adjusted values for anthropometric, biochemical, and body mass and muscle strength in older populations is important to appreciate that it is as important a role in the role of skeletal integrity. As weak factor for falls, it is worth noting that associated loss of skeletal muscle is related to the reduction in bone mass in the elderly (Cohn et al. 1985 and 1987). The loss of muscle mass is influenced by creatinine excretion rate, which is from about 2000 mg/hr at age 30, 1500 mg/hr at age 70 (Tzankoff & Smolin, 1979). Direct regional assessment by dual energy x-ray absorptiometry indicates that the bone mass is more than 80% of area in men, but only 30% in women (Fiatarone et al., 1991). In lean body mass, termed LBM, is accelerated after menopause (Rosenberg, 1989). In contrast, the loss of muscle mass and muscle mass is associated with an increase in muscle fat (Borkan, Hults, and Silbert, 1983).

For example, differentiation of muscle mass via an assessment of muscle mass and muscle mass loss can be achieved at any age, while protein intake is maintained. Flynn, N. A., and Krause (1989) at there may also be a gender difference in the rate of body composition with age, as the most rapid loss of body mass and muscle mass on nutritional status is substantial. Measurements have been suggested...
diminish by about 100 cal per decade after age 45 as basal metabolism strongly reflects the lean, metabolizing component of body mass (Tzankoff & Norris, 1977). As energy intake is reduced with age due to a lower basal metabolic rate and a more sedentary lifestyle, it becomes increasingly difficult for an older person to satisfy his or her micronutrient requirements through diet alone. With regard to micronutrients, Fiatarone et al. (1990) observed that inadequate intakes of vitamin D, magnesium, calcium, and zinc are strongly associated with sarcopenia.

Requirements for dietary protein are based largely on nitrogen balance studies rather than the maintenance of muscle mass. Protein provides about 10–20% of total energy intake in both young and older adults, despite the smaller energy intake characteristic of the latter group. While a number of reports have identified protein–calorie malnutrition as a major problem among the elderly, this status appears to be almost exclusive to elderly individuals with concomitant diseases associated with wasting. Among free-living older adults, median protein intakes tend to exceed RDA standards (Sahyoun, 1992), although biochemical markers for protein status, e.g., transferrin, total protein, and transthyretin in serum and plasma albumin, are generally noted to decline with advancing age after age 60 (Munro, 1992). Campbell, Crim, Dallas, Young, and Evans (1994a), by employing a new nitrogen balance study in elderly subjects and a recalculation of data from previous studies, have estimated the dietary protein requirement of healthy older men and women to be 0.91 = 0.043 g kg⁻¹ day⁻¹, a value that is higher than the intakes of 0.8 and 0.6 g kg⁻¹ day⁻¹ recommended by the RDA and the 1985 joint FDA/WHO/UNU Expert Consultation, respectively. They recommend that a safe protein allowance for the elderly requires an intake of 1.0–1.25 g kg⁻¹ day⁻¹ from a diet containing high-quality protein; similarly, Young (1990) has suggested that protein requirements for older adults should be increased. That this higher requirement for protein in older adults occurs despite their decreased muscle mass appears to suggest a lower efficiency of dietary protein utilization. Interestingly, Meredith, Frontera, O'Reilly, and Evans (1992) found that providing elderly volunteers with a daily protein-energy supplement increased the size and mass of the midthigh muscle more than in nonsupplemented controls after a regimen of resistance training.

The functional significance of sarcopenia is also substantial, as preservation of the fat-free compartment is highly predictive of muscle function and mobility in the elderly (Fiatarone et al., 1991). Frontera, Hughes, Lutz, and Evans (1991) examined the isokinetic strength of the elbow and knee extensors and flexors in healthy older adults and observed that strength was significantly lower in those aged 65–78 years than in those aged 45–54 years. Adjusting the data for body composition revealed muscle mass as the major determinant of the age- and gender-related differences in muscle strength, independent of muscle location (upper vs lower extremities) and function (extension vs flexion). Thus, if muscle mass can be preserved into old age, it would be reasonable to expect that muscle strength could also be preserved. While the reduction in muscle mass with age appears to account for the quantitative loss of strength, Bruce, Newton, and Woledge (1989) note that qualitative changes in maximal force production may result from decrements in neural recruitment capacity. Fiatarone et al. (1990) have reported that resistance training can significantly restore muscle mass and strength even in very old people who have lost substantial amounts of both; the observation of inadequate intakes of several micronutrients associated with sarcopenia suggests that appropriate nutritional intervention may be synergistic with the resistance training. However,
very modest micronutrient supplementation at approximately one-third of the RDA levels during such training does not appear to significantly enhance the response to training (Fiatarone et al., 1994). Moreover, in addition to a potentially greater requirement for protein with exercise, Campbell, Crim, Dallas, Young, and Evans (1994b) found that weight maintenance during resistance training by older adults was associated with an increased caloric requirement. Interestingly, Roberts et al. (1992) have suggested that the current RDA levels for those aged 50+ years not only may significantly underestimate usual energy requirements but, further, that the low levels of energy expenditure suggested by the RDA may favor increases in body fat mass.

The impact of sarcopenia extends beyond its contribution to weakness, risk of falls, and the inability to perform activities of daily living. The importance of muscle mass to the maintenance of basal metabolic rate, insulin sensitivity, and physical activity implicates sarcopenia as a risk factor in many of the leading chronic diseases common to older adults, including Type II diabetes, coronary artery disease, and hypertension.

III. Immune Function

A. Aging and Immunity

The age-related decline in lean tissue mass is not insignificantly composed of the losses in the immune system, which comprises 8% of total lean mass (Makinodan & Kay, 1980). Involution of the thymus, beginning at puberty and complete by middle age, may represent one of the earliest age-related decrements in the immune system, which correlates in part with other impairments in immunity through the life span (Hirokawa, 1992). Defects as diverse as dysfunction of T and B lymphocytes, elevated levels of circulating immune complexes, an increase in antibodies, and monoclonal gammopathy have been recognized as common aging process (Meydani & Blumberg, 1991). Alterations in intracellular calcium, decreases in interleukin (IL) production and IL-2 mRNA expression, and poor proliferative response to mitogens are some of the age-associated changes observed in lymphoid cells that are linked to the failure of the immune system with aging (Miller, 1995).

Both T and B cells from older adults demonstrate defects in activation, particularly the cell cycle events for differentiation (Thoman & Weigle, 1989). Shifts in the distribution of functionally distinct T-cell subsets with age, particularly increased accumulation of memory cells, by chronic antigenic stimulation have been reported; these memory T-cells appear to be resistant to intracellular calcium, a condition that may lead to decreased signal transduction (Grossmann & Rabinovitch, 1990). In addition, alterations in T lymphocytes of phosphatidylinositol 3-kinase signaling pathways, cytokine production, and the number and function of membrane-bound receptors of T-cells have been closely associated with the decline in T-cell functions during aging (Binovitch, June, Grossmann, & Ledet, 1986; Fernandes & Venkatraman, 1990). Further, decreased lymphocyte reactivity in older adults is negatively correlated with an increased membrane phospholipid viscosity which may reflect a dysfunction of lipid homeostasis (Huber et al., 1991). Age-related T-cell defects may increase susceptibility to malignancies, infections, and the development of autoimmune diseases in the elderly (Schwab & Weksler, 1987).

B. Nutrition and Immune Responsiveness

Due to the dependence of immune responsiveness upon both macro-
micronutrient adequacy, general assessments of nutritional status often include measures of total lymphocyte count, delayed-hypersensitivity skin test (DHST), mitogen-stimulated lymphocyte proliferation, and the stimulated elaboration of lymphocytic cytokines. Severe malnutrition or any one of several nutrient deficiencies can compromise immune functions, which can be restored by provision of the appropriate nutrients (McMurray, 1984; Chandra, 1981; Cunningham-Rundles, 1993). However, as these severe nutritional states are relatively rare even among the older population, research has focused on whether enhancement of micronutrient status can prevent or retard the typical age-related declines in immune function.

Chandra, Joshi, Au, Woodford, and Chandra (1982) first examined the nutritional and immunological status of a group of apparently healthy elderly subjects without presenting underlying systemic disease, among those with clinical, hematological, and/or biochemical evidence of nutrient inadequacies, DHST and T-cell numbers (including CD4⁺ subset) and response to mitogens were significantly reduced. Nutritional advice and supplementation sufficient to increase energy intake by approximately 500 kcal/day and to provide at least the RDA levels of micronutrients for 8 weeks improved each of these immune parameters and nutritional status as assessed by serum levels of albumin, prealbumin, transferrin, retinol-binding protein, zinc, and iron. Targeting nutritional advice and supplementation to the specific needs of his older patients, Chandra (1989) observed improvements in natural killer cell activity, mitogen-stimulated lymphocyte proliferation, and DHST, as well as higher antibody-forming cell responses and IL-2 production.

More general and longer term nutritional supplementation to free-living older adults has also resulted in improved immunological stasis. Bogden et al. (1990, 1994) administered One-a-Day type multivitamin–mineral supplements for 1 year in double-blind, placebo-controlled trials and found that the treatment significantly enhanced lymphocyte proliferative responses and/or DHST. Similarly, Chandra (1992), employing a multivitamin–mineral supplement containing moderately higher doses of vitamin E and β-carotene for 1 year, reported that the treatment significantly enhanced lymphocyte proliferative responses, IL-2 production, natural killer cell activity, and antibody responses to influenza vaccine. Importantly, Chandra (1992) also noted that the subjects using the supplement had 48% fewer days of infectious disease episodes and required 56% fewer days of antibiotic drug treatment from the placebo group.

The well-documented effects of various nutrients on maintenance of optimum immunity has led to clinical studies of single nutrients, particularly the antioxidants (Bendich & Chandra, 1990). For example, the administration of supplemental vitamin C to healthy older subjects has been found to enhance lymphocyte proliferative responses, DHST, serum IgG, IgM, and complement C3 levels (Kennes, Dumont, Brohee, Hubert, & Neve, 1983; Ziemienski, Wartanowicz, & Kios, 1986). In their community-based survey, Goodwin and Garry (1983) found that older adults within the top 10% for plasma ascorbate concentration had significantly fewer anergic subjects as defined by DHST.

Several molecular and biochemical mechanisms of ascorbate-mediated immunostimulation have been proposed, including (a) modulation of intracellular cyclic nucleotide levels, (b) modulation of prostaglandin (PG) synthesis, (c) protection of 5'-lipoxigenase, (d) enhancement of cytokine production, (e) antagonism of the immunosuppressive interactions of histamine and leukocytes, and (f) neutralization of phagocyte-derived autoreactive and immunosuppressive oxidants (Anderson, Smit, Joone, & Van Staden, 1990). Vi-
Vitamin C appears to serve as the first-line plasma antioxidant in the defense against phagocyte-derived reactive oxidants; only when ascorbate is depleted does detectable free radical damage, measured by the appearance of lipid peroxides, ensue (Frei, Stocker, & Ames, 1988).

In placebo-controlled, double-blind trials with vitamin E in healthy older adults, S. N. Meydani et al. (1990) and M. Meydani et al. (1993b) have demonstrated significant improvements in DHST and/or lymphocyte proliferation, as well reductions in plasma lipid peroxides and the production of PGE2 by peripheral blood mononuclear cells. Cannon et al. (1990) found that, in older men, vitamin E supplementation effectively restored severe blunted acute phase immune responses, including increases in circulating neutrophils and creatine kinase, following an intense bout of eccentric exercise to levels comparable to those of young men; this enhancement in immune responsiveness, together with an inhibition of lipid peroxidation and pro-inflammatory cytokines IL-1 and IL-6, appears to be consistent with the concept that vitamin E provides protection against exercise-induced oxidative injury (Cannon et al., 1991; M. Meydani et al., 1993a).

Ziemlanski, Wartanowicz, and Kios (1986) reported that vitamin E supplementation of institutionalized elderly women increased total serum protein, with the principal effect on α1- and β2-globulin fractions, significant increases in IgG and complement C3 levels were noted when vitamin C was combined with vitamin E. Penn et al. (1991) administered a combination of vitamins A, C, and E to hospitalized geriatric patients and reported improvements in cell-mediated immune function, as assessed by significant increases in the absolute number of T cells, T4 subtypes, T4:T8 ratio, and mitogen-stimulated lymphocyte proliferation in the treated vs placebo group.

Vitamin E may exert its immunostimulatory effect in older adults by inhibiting PG synthesis and/or decreasing radical formation. Vitamin E can both the lipoxygenase and cyclooxygen pathways of arachidonic acid metabolic. Oxygen metabolites, especially hydroperoxide, produced by actuated phases depress lymphocyte proliferation α-tocopherol has been shown to decrease hydrogen peroxide formation by morphonuclear leukocytes (Blum, 1993).

After supplementing older subjects β-carotene, Watson, Prabhala, Plezia, and Alberts (1991) found a dose-dependent decrease in T-helper cells, natural cells, and peripheral blood mononuclear cells with IL-2 and transferrin receptor. Talbott, Miller, and Kerkvliet (1987) reported that supplementation of healthily individuals with vitamin B6 significantly increased lymphocyte proliferative responses to several mitogens and the percentage of T-helper cells. S. N. Meydani et al. (1991b), by employing a vitamin depletion-repletion protocol, showed IL-2 production and mitogenic response T- and B-cell mitogens are affected changes in dietary vitamin B6; supplementation with high doses of pyridoxine proved these responses beyond their line values in these healthy older adults.

The occurrence of low serum thymulin and other indirect evidence of a pre-mild zinc deficiency in the elderly population have prompted some trials with supplementation (Chandra et al., 1984a; Bogden et al., 1984). Duchateau, Delepess, Vrijens, and C. (1981) examined the effect of zinc supplementation on immune response healthy institutionalized subjects over years of age and observed an increase in DHST and the percent of circulating cells, as well as improved serum IgG body formation against tetanus. However, Chandra (1984b) has reported that excessive zinc supplementation pairs the immune responses.

Essential fatty acids and dietary