Undernutrition in the Elderly: A Physiological or Pathological Process?

Elliot M. Berry

Department of Medicine, Hadassah University Hospital, Jerusalem, Israel

Many studies have shown that there is a progressive decline in both energy intake and energy expenditure with increasing age, with alterations in body composition such that the proportion of adipose tissue increases at the expense of the lean body mass. Nutritional recommendations for the elderly are based on the assumption that these changes are detrimental. Thus, the recommendations have been designed in general to reverse the changes and restore the body composition and nutritional requirements to that of the younger age groups.

Other considerations suggest that this is not necessarily correct. Following the concept of Cannon that there is inherent “wisdom” in the body’s metabolism and the comment of Shock that “aging is not a disease,” the changes found in the elderly may in fact reflect their different physiological status and be an adaptive response designed to promote longevity. The aim of this chapter is to suggest that the “undernutrition” of the elderly—far from being a pathological process—may rather be an attempt to slow the progress of various disease processes, in particular atherogenesis and tumorigenesis.

ENERGY BALANCE AND BODY WEIGHT IN THE ELDERLY

The physiological changes in energy metabolism with increasing age have been studied by a number of workers. After the age of 30 years, energy intake decreases by approximately 12 kcal/day/year, and the basal metabolic rate (BMR) by 5 kcal/day/year (1). Height decreases by approximately 1 mm/year, and since the lean body mass (LBM) decreases by about 2% to 3% per decade, the BMR/metabolic mass remains relatively stable. There is also a decline of about 15% in total body water, together with a redistribution of body fat toward the trunk. Average body weight may not change over the decades and in fact decreases by 10% between the ages of 70 and 80 years. Body weight is considered to be a regulated variable and in view of the decrease in energy intake, there must be a corresponding decrease in energy expenditure (metabolic rate and physical activity). However, it is not clear which
is the cause and which the effect. Is the reduced LBM or metabolically active tissue responsible for the reduction in activity of the elderly, or is the reduction in LBM an adaptation to reduced activity and both consequent on a decline in energy intake? In other words, is the decline in food intake or the decline in energy expenditure the initiating factor? Measurements of energy expenditure in the elderly are not well documented (2), but activity rhythms in healthy elderly people are often well preserved (3). The RDA 1989 do not assume that a decline in activity is either desirable or inevitable. Rather, physical training, including strength training, is effective in the elderly and may blunt many of the physiological declines associated with aging (4). At all events body metabolism appears to become more “efficient” with increasing age, which would lead if unchecked to an increase in body weight that does not normally occur. Table 1 summarizes the function of various systems of the body at the age of 70 years as a percentage of that at the age of 30. In general, in the absence of disease, physical function continues to be appropriate but reserve capacity, the ability to respond to stress, diminishes linearly with time.

The decrease in appetite and weight loss with advancing age has led to a study of the neurophysiology of feeding behavior and has given rise to the term “anorexia in the elderly” (5), although it has absolutely no pathopsychological relationship to the disorder of adolescent girls. Brain levels of neuropeptide Y and norepinephrine, both stimulators of feeding, are reduced in Alzheimer’s disease. The causes of a reduced body weight in the elderly are multiple and are detailed in Table 2. Most are related to organic, socio-economic, or iatrogenic (drug) causes. A percentage, not as yet known, may also be related to the physiological changes in metabolism detailed above and may thus be regarded as a normal response. The situation concerning fluid intake in the elderly is different and it appears that elderly people experience less thirst in response to water deprivation and must therefore drink more frequently (6). If the major causes of mortality in Western developed countries are heart disease (41%), cancer (22%), and strokes (8%), the principal causes of morbidity due to aging in the elderly are immobility and decreased cognitive function. For example, hip fractures start to rise in frequency after the age of 40 years, increasing exponentially, doubling every 6 years. We know virtually nothing about the causes

### TABLE 1. Changes due to aging: physiologic function at 70 years as a percentage of that at age 30

<table>
<thead>
<tr>
<th>System</th>
<th>Function</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular system</td>
<td>Cardiac output</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td>Maximum heart rate</td>
<td>75</td>
</tr>
<tr>
<td>Respiratory system</td>
<td>Vital capacity</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td>Residual volume</td>
<td>130-150</td>
</tr>
<tr>
<td></td>
<td>Maximum O₂ uptake</td>
<td>40</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>Muscle mass</td>
<td>70</td>
</tr>
<tr>
<td></td>
<td>Hand grip, flexibility</td>
<td>70</td>
</tr>
<tr>
<td></td>
<td>Bone mineralization</td>
<td>70-80</td>
</tr>
<tr>
<td>Renal function</td>
<td>Conduction velocity</td>
<td>85</td>
</tr>
<tr>
<td>Nervous system</td>
<td>Resting glucose uptake</td>
<td>100</td>
</tr>
<tr>
<td>Metabolism</td>
<td>Taste and smell</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>Fasting blood glucose</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>Basal metabolic rate</td>
<td>85</td>
</tr>
</tbody>
</table>
and prevention of the senile dementias that afflict 20% of people over the age of 80 years (7). However, all these processes may be influenced by the nutritional status of the patient.

**THE EFFECT OF ENERGY RESTRICTION ON ATHEROGENESIS**

Atheromatous plaques are multifactorial in origin, and their formation depends on genetic, metabolic, and environmental variables. With increasing age serum cholesterol increases as a result of increased production and diminished fractional clearance of low density lipoprotein (LDL). A wealth of epidemiologic evidence shows that atheroma increases with obesity, hypertension, diabetes, and hypercholesterolemia and that these diseases are both interactive and additive. Weight reduction, especially if coupled with physical activity, which increases high density lipoprotein (HDL) (8), is a primary (but not very successful) treatment for all these risk factors responsible for myocardial infarction and cerebrovascular accidents. Higher cerebral function depends on a steady supply of nutrients and oxygen. Brain organization is plastic, such that gradual ischemia of most regions may be accommodated without loss of function; in other words, if the same blood vessel was occluded over a number of months, instead of instantaneously as in a stroke, there would not be many neurologic sequelae. The problem of senile dementia is one of promoting and maintaining cerebral blood flow. Such changes may contribute to pathology in Alzheimer's disease, although the primary process is not vascular.

**THE EFFECT OF ENERGY RESTRICTION ON LONGEVITY AND TUMORIGENESIS**

More than 50 years ago McCay first documented that energy restriction in rodents increased longevity. It was also shown that underfed animals were less susceptible to experimental tumors. The findings are robust but there appear to be strain...
TABLE 3. Effects of nutrition on the incidence of mammary carcinoma in rats

<table>
<thead>
<tr>
<th>Item</th>
<th>Dietary regimen</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High fat ad libitum</td>
</tr>
<tr>
<td>Energy intake (kcal/day)</td>
<td>41</td>
</tr>
<tr>
<td>Fat (g/day)</td>
<td>2.7</td>
</tr>
<tr>
<td>Linoleic acid (g)</td>
<td>1.5</td>
</tr>
<tr>
<td>Body weight (g)</td>
<td>217</td>
</tr>
<tr>
<td>Body composition</td>
<td></td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>24</td>
</tr>
<tr>
<td>% protein</td>
<td>20</td>
</tr>
<tr>
<td>Tumor incidence (%)</td>
<td>73</td>
</tr>
</tbody>
</table>

*Adapted from Pariza, ref. 13.

variations—rats are more susceptible than mice. The putative mechanisms concern a number of systems, including the pituitary-adrenal axis, decreased gonadotrophins and enzyme induction, slowing primary aging processes (9), and protecting cellular homeostasis (10). The effects on the immune system are thought to involve decreased cell turnover and increased natural killer cell (NK) activity. Good and Lorenz (11) have examined these phenomena in genetically short-lived mice and in models of autoimmune disease. They have pointed out that the energy restriction is effective only if micronutrients (minerals, especially zinc, and vitamins) are present in adequate amounts, and its effect is enhanced by physical exercise (12).

The nutritional factors involve interaction between energy intake, energy content, and the dose of the carcinogen used. Total energy intake appears to be more important than the source of energy (13). The nature of dietary fat has been a major point of interest, since polyunsaturated fats may promote carcinogenesis while at the same time decrease the risk of atheroma. Pariza and coworkers have attempted to dissect the various interactions in a rat model, as shown in Table 3. It appears that the incidence of tumors depended on the total energy intake, the efficiency of utilization, and the circadian rhythm. It did not depend on the energy source, the percent of fat in the diet, the amount of linoleic acid ingested, body weight, or even on the age of introduction of the energy restriction. There may be an effect on free radicals, inhibiting an age-related increase in membrane 22:5 fatty acids, and thus causing a reduction in the peroxidation of membrane lipids (14). Peroxidized LDL lipids are more atherogenic than the native lipoproteins. Another possible mechanism is through modulating gene expression of metabolic enzymes and oncogenes (15). In animals, a restriction of 15% to 20% in food intake is effective in prolonging longevity and promoting resistance to tumorigenesis.

These findings have been confirmed in animals but have not yet been systematically applied to human geriatrics or oncology. Epidemiologic evidence in man is, however, supportive of a relationship between energy metabolism and carcinogenesis (13). Human obesity correlates with the incidence of carcinoma of the breast and
undernutrition

Carcinoma of the colon is associated with reduced physical activity, whereas increased exercise during adolescence lowers the subsequent risk of developing breast and uterine cancer. These findings suggest that research in the field of diet–cancer should concentrate more on the effects of total energy intake than on those of single nutrients, for example, fats, where neither breast nor colon cancer appears to be related to the quality of dietary fat ingested (16,17). In light of this argument, the appearance of cachectin or tumor necrosis factor (TNF) may be seen as an adaptive response to the presence of the tumor (18). TNF is secreted by cells of the immune system; it is cytotoxic/static to tumor cells in vitro, induces interleukin-1, and suppresses the principal enzyme responsible for storage of adipose tissue triglyceride, lipoprotein lipase. The other name for TNF, cachectin, emphasizes the anorectic potency of the protein. Thus, TNF aids the immune response and decreases caloric intake, which may help in tumor suppression.

**THE EFFECTS OF ZINC ON THE PHYSIOLOGY OF AGING, BRAIN DEVELOPMENT AND FUNCTION, AND ON THE IMMUNE SYSTEM**

Although we have considered the effects of total nutrition on longevity and tumorigenesis, the micronutrient zinc has a peculiar role in linking the different aspects of the physiology of aging, immunocompetence, and brain function. Zinc deficiency may lead to anorexia and a decrease in taste (hypogeusia). In the immune system there is decreased wound healing and T-cell dysfunction. Zinc supplementation alleviates the immune deficiency associated with acrodermatitis enteropathica (11). Zinc may also be involved in cerebral function in animals (19) and in man (20). Volunteer subjects given large doses of histidine develop "zincuria," cerebellar dysfunction, mental changes, and alterations in taste and smell that were all reversed by zinc replacement. Zinc is a cofactor in enzymes involved with myelination and in the formation of catecholamine and glutamate neurotransmitters. Thus, adequate zinc supplementation is a simple, practical recommendation for the elderly.

**CHANGES IN BRAIN STRUCTURE AND FUNCTION WITH AGING**

The debate over whether there is loss of neurons associated with the aging process has been summarized by Coleman and Flood (21). There appears to be decreased density of large neurons in the cerebral cortex and a decrease in cortical volume. Computed tomography (CT) studies on 64 healthy men aged between 31 and 87 years suggested that several brain regions are selectively altered with age—there is symmetrical atrophy of the cingulate gyrus and sulcus, with asymmetrical widening of the central and postcentral sulcus on the left and the intraparietal sulcus on the right (22). The functional correlates of these changes are speculative, but could relate to decreased verbal fluency and auditory tasks as well as to an age-related decline in the left-hand performance relative to the right. Positron emission tomography (PET)
studies have not revealed any absolute changes in glucose utilization (23), despite the presence of structural atrophic changes, as shown by hypofrontality and the strong relationship between age and ventricular size as measured by CT. If structure–function relationships in the brain are analogous to other organs, then continued mental activity should be encouraged to counter the effects of brain atrophy.

Another attempt to assess brain function with aging used power spectrum analysis of electroencephalogram (EEG) waveforms from different brain areas during the performance of various neuropsychological–cognitive tasks (24). These were then correlated with a number of biochemical indices of nutrition. The principal findings from this study on 28 healthy subjects over the age of 60 years was a decrement in alpha wave activity in subjects with low thiamine levels. There were no correlates with zinc, perhaps because none was malnourished, but of interest was the finding that the EEG frequency responses of older subjects with high iron stores were similar to those of younger subjects; however, these studies are difficult to interpret because of the lack of consistency in the correlations between plasma iron, transferrin, and ferritin. It should also be noted that neuropsychiatric disorders due to cobalamin deficiency may occur in the absence of anemia or macrocytosis (25). Other studies in normal subjects over the age of 60 years showed a correlation between cognitive function and levels of vitamin C, B_{12}, folate, and riboflavin (26). Undernutrition may also suppress age-related changes in dendritic spines (27). Necropsy studies of human brains have shown an age-related loss of dopamine uptake sites in the putamen (28), which may explain the increased sensitivity to drug-induced Parkinson's disease with advancing age.

The effects of nutrition on brain function have not been fully studied in the elderly. The topic that was first investigated by Wurtman and coworkers at the Massachusetts Institute of Technology relates to the nutritional precursors of neurotransmitter synthesis, and to the competition between amino acids to cross the blood–brain barrier. Tryptophan competes with the other large neutral amino acids (LNAA), the concentration of which is in turn affected by the balance between protein (raises) and carbohydrate (lowers) in the diet. Older patients (over 40 years) perform poorly after carbohydrate meals as compared to protein, with lapses in sustained attention (29). Together with the Wurtman team and John Growdon at the Massachusetts General Hospital, we have recently investigated the effects of different diets on the action of L-dopa (another amino acid) in patients with Parkinson's disease. By using a suitable ratio of carbohydrate to protein, we were able to abolish postprandial alterations in the levels of the LNAA and thus provide more predictable dose-response effects (30). Such simple dietary manipulations may be applicable to other neurotransmitter precursors and await evaluation for a possible role in improving cerebral function in the elderly.

CONCLUSION

It follows from the above discussion that active thin people should live longer and be less susceptible to cancer than age-matched subjects with the opposite habitus
and lifestyle, but this remains to be proven. If trials are performed on energy restriction in the elderly, then vitamins, minerals, and trace elements (with particular attention to calcium and zinc) must be included in adequate amounts.

REFERENCES


**DISCUSSION**

*Dr. Kritchevsky:* The Lipid Research Clinics data show that in men, but not in women, a cholesterol below 170 to 180 mg/dl increases the risk of colon cancer. This is an area that has been bothering people for a long time and views range from dismissing the finding on the grounds that the affected individuals probably had cancer on entry to the program to saying it simply proves you should not worry about your cholesterol level. I think both views are wrong. If you have a high cholesterol you should lower it, but in some cases a low cholesterol may put people at risk of other diseases. Data are now emerging on body type and previous diet that may be relevant to this argument.

*Dr. Berry:* I have looked at adipose tissue biopsies in patients routinely coming for colonoscopy and have found no difference in the polyunsaturated/saturated ratio between cancer cases and controls, so I have been unable to confirm any effect of quality of dietary fat on development of colon carcinoma.

*Dr. Harris:* I want to make a comment about low cholesterol as well. There are data that show that people who have had prolonged weight loss drop their serum cholesterol and seem to be at increased risk of poor outcome. The issue to be considered is how much do we know about these individuals before they entered the studies? In some of the data we have examined from the National Center for Health Statistics there seem to be two groups of individuals with low cholesterol. One group has a very low risk of poor outcomes such as heart disease; the other has low cholesterol associated with poor health, and this group is at high risk of poor outcome. We need to segregate these two populations and look at them longitudinally to be able to identify any health risks associated with low cholesterol per se.

*Dr. Schlierf:* You recommended a low salt intake. Could you define this more precisely? We have already learned that the aging kidney is not able to conserve sodium, so sodium excretion will continue even if sodium intake is low. Orthostatic hypotension is one of the most common identifiable causes of falls in elderly people and this may be related to depletion of circulating blood volume due to sodium depletion. In addition, elderly people are often given diuretics that will aggravate the situation even more.

*Dr. Berry:* Salt intake will depend on the ambient temperature. If there is sweating, salt should not be restricted. My feeling is that, in view of the risks of fluid overload, hypertension, and heart failure in the elderly, salt intake should be moderate—only enough to give a little taste to the food. In my view, the problem of orthostatic hypotension commonly stems from overtreatment of hypertension in the elderly.

*Dr. Chen:* What is the possible mechanism to explain the change in tumor incidence induced by physical exercise?

*Dr. Berry:* I would guess that this is related to blood flow or to sympathetic activity.

*Dr. Glick:* Perhaps it could work through its effect on reducing obesity?

*Dr. Kritchevsky:* Exercise affects insulinemia, and insulin is a tumor growth factor. If you induce mammary tumors in rats and then make them diabetic, the tumor growth rate decreases significantly (1).

*Dr. Schiffman:* I should like to comment on food intake in the elderly. We did a large study (2) in a retirement home in North Carolina where we measured every ounce of food...
consumed and also collected 24-h recall data. The actual intakes were between 250 and 300 kcal greater than the recalled intakes, so recall is clearly inaccurate. The question still remains to be resolved as to how much these elderly people are really eating. I believe it is more likely that the weight loss occurring in old age is the result of absorption problems rather than undereating.

Dr. Berry: There is not much evidence for malabsorption in the elderly. If negative energy balance occurs, then they are either doing more exercise or eating less.

Dr. Bowman: Underreporting is an increasingly recognized problem in the use of 24-h recall. However, it is probable that such underestimates of food intake span the age ranges, so that even though the absolute numbers may not be correct, there may still be a relative change in old age.

Dr. Meredith: If you look at energy intake required for constant body weight in a metabolic ward, which is a fairly good measure of energy needs in the short term, there does not seem to be much change with age. From age 50 years to 80 it remains around 30 kcal/kg per day, although we know that during this period there is a reduction in lean mass. It thus seems that energy needs per unit body weight do not change much, although clearly per unit of lean body mass they may actually be increasing.

Dr. Berry: I am suspicious of metabolic experiments of this type in a free-living population. They are often done over weekends and who knows what happens then?

Dr. Mertz: There are many trace elements that are important for immune function and general health. The danger of recommending supplements for fortification is that we create imbalances. I hope I have interpreted your recommendation correctly in assuming that the nutrients you emphasize—zinc, calcium, and so on—should come from a balanced diet, not necessarily from individual supplements.

Dr. Berry: That is correct. However, I don’t think there is any harm in giving vitamin supplements to the elderly since in most cases the excess will be excreted. Zinc may be involved in cerebral function in a number of ways. It is an endogenous modulator of glutamate neurotransmission, it is a cofactor of enzymes forming myelin and catecholamines, and it is involved in DNA repair. It is also involved in T-cell function.

Dr. Schiffman: One difficulty experienced by elderly people taking zinc supplements is that it is excreted in the saliva and causes an unpleasant taste. This leads to anorexia and poor eating.

Dr. Mertz: That is correct. There are other disadvantages such as the well known antagonism between zinc and copper.

Dr. Chandra: I agree that we should be cautious of giving zinc in excess. In studies that we published 7 years ago (3), we showed that if zinc intake exceeded 150 mg per day, there was a definite deleterious effect on a variety of immunological functions including neutrophil function and T-cell function. This may in part be because of its effect on copper absorption, but we also showed that serum and cell-bound lipoproteins increased, particularly the low density lipoproteins that are immunosuppressive. I think that when we refer to supplements as opposed to foods that are rich in zinc we have to be careful about the amounts, so once again the key word is moderation.

REFERENCES