Energy Balance in the Elderly

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Although the cut-off point is not clearly defined, and has great interindividual variability, age-related changes in energy balance of adults appear to be divided into two distinct, non-symmetric phases: the first phase is typically associated with a positive energy balance and an increase in weight and adiposity (but with a decrease in muscle mass), with more than 20% of the US adult population being overweight (1,2). This phase usually occurs between 20 and 65 years of age. The second phase, usually beginning after the age of 65 to 70 years, is associated with a loss of weight, mostly accountable for by a loss of lean body mass (3) (Fig. 1), and with a high prevalence of malnutrition (4).

The metabolic origins of the age-dependent compositional changes have not been clearly identified. Activity of growth hormone and testosterone, which promote lean tissue growth, is reduced with aging (5); this may contribute to the shift in balance from lean to adipose tissue. Indeed, growth hormone therapy can in part correct age-associated compositional changes (6). A decreased trophic effect of the autonomic nervous system on muscle and a decreased capacity for muscle fiber regeneration have also been implicated (7). Interestingly, with advancing age above 60

![FIG. 1. Age-associated changes in weight and composition. A: Between 20 and 65 years. From Shepard JW (1). B: Between 70 and 81 years. From Steen B (3).](image-url)
years, mortality risk of being underweight becomes greater, whereas being mildly overweight is associated with the least mortality (8).

With aging a redistribution of body fat occurs, with gains in fat depots in the central and intraabdominal space, and away from peripheral subcutaneous fat. Such a redistribution is associated with increased risks of hypertension, diabetes, stroke, gallbladder disease, and coronary artery disease (9).

Understanding the age-related changes in energy balance requires a separate discussion of its two components: energy output and energy intake. Age-related changes that may reduce energy intake and output are listed in Table 1.

### ENERGY OUTPUT: CONTROL AND AGE-RELATED CHANGES

Energy expenditure has been divided by nutritionists into the following components: (a) basal metabolic rate (BMR), (b) the thermic effect of meals (TEM), (c) physical activity, and (d) adaptive (diet-induced) thermogenesis (DIT).

#### Basal Metabolic Rate

BMR amounts to about 1400 to 1600 kcal per day, or 60% to 65% of total energy expenditure in a moderately active young adult (5). It originates primarily from, and therefore is highly correlated with, lean body mass. The metabolic origins of the BMR, especially the quantitative contribution of its individual components, are not clearly established. Protein turnover is thought to constitute about 15% to 25% of the BMR (10). The energy cost of the "sodium pump," needed to keep sodium extracellular and potassium intracellular against their concentration gradients, is estimated at 20% to 40% (11), and the cost of "futile" cycles in the metabolism of carbohydrate and fat are estimated at less than 5% (12). The energy costs of the other components of BMR—the maintenance of muscle tone and activity of involuntary muscles—have not been determined experimentally.

The BMR is governed by thyroid hormones that stimulate activity of the sodium pump (5), as well as protein turnover (5). In addition to its direct effect, T₃ can influence metabolic rate by enhancing sympathetic activity (13).
With advanced age there is a gradual 10% to 20% decline in BMR (14), which is typically accompanied by a reduced thyroid hormone activity (5) and a reduced responsiveness to norepinephrine (5). This decline also parallels the loss of muscle mass (Fig. 1). Of the known biochemical contributors to the BMR, only protein turnover is consistently reduced with aging (15), apparently reflecting the decline in lean body mass.

**Thermic Effect of Meals**

The TEM is the increment in energy expenditure that is observed upon meal taking and that lasts for several hours thereafter. The total energy of this thermic effect amounts to about 5% to 10% of intake. It has an "obligatory" component that reflects the metabolic cost of converting the ingested macronutrients into body protein, fat, and glycogen, and an adaptive component, which is stimulated by norepinephrine, and which apparently amounts to less than 30% of the total TEM (5).

The thermic responses to a glucose meal (16) and to a protein meal (17) are decreased with advancing age (but see ref. 18). One would not expect an effect of aging on the obligatory component of the TEM, but based on a reduced capacity for adaptive thermogenesis observed in old rodents (5), a reduced adaptive component of TEM in the elderly person is feasible.

**Physical Activity**

The amount of energy spent in physical activity varies greatly, with very highly active individuals expending more than 2000 kcal, and those moderately active expending 700 to 800 kcal, or about 30% of their total energy expenditure (5). Aging is usually associated with a significant decline in physical activity. In young subjects, as physical activity increases, there is a compensatory rise in spontaneous food intake, but data from animals suggest that older subjects do not adequately compensate intake for an increased expenditure, displaying a negative balance (5).

Aging is associated with a decline in physical working capacity (VO₂max), amounting to about 5% to 10% per decade between the ages of 25 and 65 years (1,19). This decline is due to the smaller muscle mass, to changes in cardiac performance, decreased responsiveness to catecholamines, and other causes (19,20). A decline in physical working capacity means that a greater effort is necessary to carry out the same physical tasks. This will tend to cause a reduction in spontaneous physical activity, which will lead to a further decline in the capacity for work. Exacerbated by age-related diseases, such as cardiovascular disease, musculoskeletal disease, osteopenia, obesity, and others, a vicious cycle is created, which tends to limit physical activity and to reduce the physical working capacity. Physical training will correct age-related deterioration in physical working capacity by as much as 50% (21).
Physical training apparently potentiates the thermic effect of meals in both young and old individuals (18).

**Diet-Induced Thermogenesis**

Chronically overfed rats accrue only a fraction of the excess energy intake, while the rest is dissipated as heat. Clearly, a component of this heat originates from the additional metabolic costs incurred in depositing excess energy as fat (obligatory component), but in rodents, the major portion of DIT originates in the metabolically active brown adipose tissue (BAT) (22). Due to the small amount of BAT in man (5), the ability to dissipate of excess energy intake is apparently limited and difficult to demonstrate clearly (23). DIT in elderly persons has not been studied, but in rats the capacity for DIT (and for cold-induced thermogenesis) may decline with age to about 10% to 20% of the level found in younger animals (5). A decline in the capacity for DIT is compatible with lower tissue norepinephrine turnover (5) and a lower responsiveness to exogenous norepinephrine.

**ENERGY INTAKE: CONTROL AND AGE-RELATED CHANGES**

With age, there is a gradual decline in food intake that corresponds to the smaller energy expenditure. During the early decades of adult life, total energy balance is positive, whereas in the late years, especially among the institutionalized elderly (24), energy (and protein) deficiency becomes more common, as food intake often falls short of expenditure. However, whether a true "anorexia of aging" exists, independently of disease, medications, poverty, and psychosocial conditions that tend to suppress appetite, is not clear. Although as many as 30% and up to 60% or more of institutionalized elderly people display signs of energy deficiency, such signs were observed in only 3% of healthy free-living elderly persons (24). Moreover, no significant fat loss occurred in healthy elderly people between the ages of 70 and 81 years, only a small trend (3) (Fig. 1). I shall now present a brief overview of the control of feeding, highlighting age-associated changes that could influence feeding.

**Central Control**

The central control mechanism of feeding is a complex network of interconnected brain structures (5). The most studied anatomic sites are the ventromedial nucleus (VMH) and the paraventricular nucleus (PVN), the ablation of both of which leads to hyperphagia, and the lateral area (LH) of the hypothalamus, the ablation of which leads to aphagia. In addition to its proposed role as the center for the control of feeding, the hypothalamus is also a prime activator of the autonomic nervous system: the VMH activates the sympathetic branch and exerts an inhibitory effect on the parasympathetic branch of the autonomic nervous system (5). Accordingly, VMH
lesions stimulate vagal activity, hyperinsulinemia, and depletion of circulating substrates, leading to a state of overeating, whereas LH lesions stimulate sympathetic activity and substrate mobilization from fat and glycogen stores (autocannibalism) and induce satiety. The relative importance of a “feeding center” imbalance versus an “autonomic” imbalance in determining feeding behavior of hypothalamic lesioned animals has not been clearly defined.

Aging is associated with a significant decline in hypothalamic neurons in both the VMH and the LH regions (25). This could possibly influence the acuity of the sensations of hunger and satiety. Aging is also associated with a decline in activity of both the parasympathetic and the sympathetic branches of the autonomic nervous system (26), but their proportionate declines as pertaining to regulation of energy balance are not clear.

Neurochemical Mechanisms

The central control mechanism of feeding responds to cues from the periphery reflecting the nutritional state, as well as to sensual stimulation. Whatever effect a peripheral stimulus has on feeding, whether transmitted in the form of a metabolite, a hormone, or by a sensory input, is encoded into the brain neurotransmitter system to elicit a feeding response. These neurotransmitters include monoamines and neuropeptides.

Catecholamines

Direct injection of norepinephrine into the VMH or PVN will stimulate feeding, whereas its administration into the LH will inhibit feeding. This effect of norepinephrine is mediated through α-adrenergic receptors in the VMH and through β-receptors in the LH (5).

Aging is associated with a significant decline in catecholamine synthesis and activity in the hypothalamus, in both man and animals (25). In addition, brain tissue (studied in the cortex and cerebellum only) from aged rats has an impaired capacity for synthesis and regulation of α- and β-adrenergic receptors (27). A decline in catecholamine activity in the hypothalamus will also cause a suppressed release of hypothalamic gonadotropin-releasing hormone (GnRH), growth hormone-releasing hormone (GHRH), and thyroid-releasing hormone (TRH), leading respectively to impairments in reproductive functions, a decline in lean body mass, and a fall in BMR.

Neuropeptides

In the rat the administration into the hypothalamus of either opioid peptides or neuropeptide Y (28) will stimulate feeding, whereas the administration of corticotropin releasing factor (CRF) will suppress it.
It was reported that older rats respond less to opioid agonists or antagonists than younger rats (4), and that they have a lower concentration of opioid peptides in the hypothalamus (4). Aging may thus be associated with a decreased opiate-based feeding drive. CRF appears to play a role in the loss of appetite associated with depression (29), which is not an uncommon occurrence in the elderly. The orexigenic effect of neuropeptide Y is apparently not age-dependent (4).

Peripheral Control

Senses

Clearly, the extent of sensual pleasure that we derive from food influences food intake. With aging there is a decreased acuity of taste in association with a significant atrophy of the taste buds, and a reduction in the ability to detect odors and to identify the foods eaten (5).

Peptide Hormones

Peripheral injections of a variety of gastrointestinal hormones (and other peptides) into rats reduce food intake. These peptides include cholecystokinin (CCK), bombesin, gastrin-releasing peptide, glucagon, somatostatin, substance P, and neuropeptide Y (28). However, the physiological significance of these hormones in producing normal satiety is not clear. A role for CCK in producing anorexia of aging was proposed recently on the basis that higher than normal serum levels of CCK were observed in elderly men, and that exogenous CCK-8 was more effective in decreasing feeding in older as compared to younger mice (4).

A continuous administration of insulin will suppress (unlike single injections that stimulate) feeding, perhaps by a direct effect in the brain (5). The importance of hyperinsulinemia in elderly feeding behavior is not known.

ANOREXIA OF AGING

Highlighted above are data on normal physiological changes in some aspects of the control mechanism of feeding that hold a potential for causing a downward trend in feeding during old age. However, the most common causes of anorexia found in elderly individuals (mostly institutionalized) are induced by disease states or they are psychosocial or poverty related (30,31). Common causes of anorexia in elderly people are given in Table 2.

Disease-Related Anorexia

Cancer: cachectin, interleukin, and prostaglandins produced in cancer patients are potent anorexic agents. Radiation therapy will impair feeding as well. Chronic ob-
**TABLE 2. Common causes for anorexia in the elderly**

<table>
<thead>
<tr>
<th>Disease states associated with reduced food intake</th>
<th>Psychosocial causes</th>
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<tbody>
<tr>
<td>Cancer</td>
<td>Chronic constipation</td>
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<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>Impaired mobility</td>
</tr>
<tr>
<td>Swallowing difficulties</td>
<td>Dementia</td>
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<tr>
<td>Abdominal angina</td>
<td>Depression</td>
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**Structive pulmonary disease:** patients with chronic obstructive pulmonary disease may have difficulties in breathing and eating at the same time. **Swallowing problems:** elderly persons often develop swallowing difficulties related to cerebrovascular accidents, medications, and other disease states. **Abdominal angina:** stomach distension is accompanied by severe pain, leading to early termination of meals. **Chronic constipation:** leads to a sensation of fullness. **Medications:** digoxin and psychotropic and analgesic/anti-inflammatory drugs may cause anorexia in a large number of patients. **Dementia:** may produce an indifference to foods. **Depression:** will often produce anorexia, perhaps induced by an increased hypothalamic CRF. **Reduced mobility:** can result in an impaired capacity to obtain food and self-feed.

**Socioeconomic Status**

Social isolation and poverty will affect appetite and the ability to purchase desirable food.

**CONCLUSION**

Although there appear to be age-related changes in some components of the control mechanism of feeding, their significance for producing "anorexia of aging" remains to be determined.

**REFERENCES**

DISCUSSION

Dr. Schiffman: The role of palatability in energy expenditure seems to be of increasing importance. Two recent studies from England (1,2) show that thermogenesis increases when more palatable food is consumed; thus, if your food tastes good you may be less likely to get fat. Could you comment on this?

Dr. Glick: These are very interesting data. Jacques Leblanc and coworkers (3,4) found that the cephalic phase of eating involves the activation of a noradrenergic and insulin-mediated mechanism for thermogenesis in both man and animals that is independent of
nutrient absorption. I think we have the start of promising research developments in this area.

Dr. Durnin: You said that a high proportion of institutionalized elderly people suffer from protein-energy malnutrition. How did you define this?

Dr. Glick: The data are from Rudman Feller and are composite, representing the compilation of results from about 20 studies. Malnutrition was defined in two ways: first, the percentage of elderly people consuming less than their RDA; and second, indicators of poor nutrition obtained from measurements of weight for height and other anthropometric data, together with data on serum albumin and other blood indicators of nutritional status. The figures I gave referring to energy malnutrition in between 30% and 66% of institutionalized elderly were based on the physical indicators.

Dr. Durnin: None of these indicators is really satisfactory. You can have extremely well nourished people who fit all these definitions of protein-energy malnutrition. I'm not sure how much reliability we should attach to this very high percentage.

Dr. Glick: I agree that without precise standards it is difficult to produce 100% reliable estimates for the level of protein and/or energy malnutrition. However, using the same criteria the prevalence of malnutrition among healthy free-living elderly people is much lower, around 3%, so there is no doubt that there is a high prevalence of malnutrition among the institutionalized.

Dr. Mertz: People certainly eat less with increasing age, but I should like to caution against interpreting data from surveys and drawing conclusions about the adequacy of the diet. We have just reported in abstract a study done over 15 years in which we compared food intake records of 266 well trained people with food energy requirements for weight maintenance, which we determined in long-term feeding studies. We found that about 80% of our subjects underreported their usual energy intake—in other words, if we put them on their reported diet they lost weight. The average underreporting among these individuals was 25%. The intakes of essential nutrients estimated from recall surveys may therefore be substantially better than they seem.

Dr. Glick: This appears to be the consensus. However, I have focused on objective indicators rather than comparisons between reported intakes and the RDA.

Dr. Steen: I think there may be a selective underreporting of different components of the diet. For instance, when we compare urinary nitrogen excretion with dietary history data on protein intake, the correlation is surprisingly good. On the other hand, we have suspicions that there is underreporting of fat and sugar. I think we can rely on the dietary history of protein intake, however.

Dr. Hallfrisch: I have a comment about "successful aging." Normal or average aging is not necessarily particularly healthy aging. The "normal" aging person may have one or more chronic diseases and may be taking several long-term prescriptions. However, there are people who age without developing chronic diseases and without having to take drugs, and these are the successful agers. We did a longitudinal study of successful agers in which we compared men who exercised until late life with those who were sedentary but healthy and not overweight. Not only do the exercisers consume more energy, but they eat a diet of different composition. I thought this was because they were more health conscious until I heard a report of Judith Stern (5) in which she described exercising and nonexercising rats given a choice of carbohydrate, protein, and fat. The exercising rats ate more carbohydrate and the sedentary rats more fat.

Dr. Glick: I pointed out the importance of having a clear picture of changes in adiposity with advancing age. In Dr. Steen's longitudinal study, published in 1988, we see a trend to
decreasing fatness in the elderly that does not reach a statistical significance. I should like to ask Dr. Steen whether his data now, after three additional years of observations, show a statistically significant decline in body fat content in older people, with advancing age.

Dr. Steen: There is a numerical trend to decreasing body fat in late age but it does not reach statistical significance. However, the proportion of the energy intake taken as fat is significantly lower in our subjects than in the Swedish population as a whole.

Dr. Nestel: Are these elderly people more health conscious? Elderly people tend to be more interested in survival than younger persons, so maybe there is a deliberate change in fat intake in a direction they think is beneficial.

Dr. Steen: We have asked this question. The answer is nearly equally divided between those who say they have reduced their fat intake for health reasons and those who say they no longer like taking much fat since it makes them feel sated.

Dr. Hallfrisch: In our data on 105 men, examining longitudinal changes in their diet over 25 years, we have shown a decline in fat intake from 42% of diet energy to 35%. Most of this decline was a secular trend seen between the 1960s and the 1980s and shared by the whole population; however, there was also a smaller, but significant, age effect such that older men ate less fat during all three decades.

REFERENCES