CONSENSUS STATEMENT

Energy balance and its components: implications for body weight regulation[1–3]

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A fundamental principle of nutrition and metabolism is that body weight change is associated with an imbalance between the energy content of food eaten and energy expended by the body to maintain life and to perform physical work. Such an energy balance framework is a potentially powerful tool for investigating the regulation of body weight. However, we need a better understanding of the components of energy balance and their interactions over various time scales to explain the natural history of conditions such as obesity and to estimate the magnitude and potential success of therapeutic interventions. Therefore, the ASN and the International Life Sciences Institute convened a panel composed of members with expertise in weight management, energy metabolism, physical activity, and behavior to review the published scientific literature and to hear presentations from other experts in these fields. The Consensus Panel met 9–12 May 2011 in Chicago, IL, and was charged to provide answers to the following 5 questions:

1. Explain energy balance and imbalance in terms of a biological system in which energy intake and energy expenditure change over time in response to the environment.
2. What are the interactions between the components of energy balance and how are they regulated?
3. What is the veracity of some of the popular beliefs related to energy balance?
4. What limitations do we face in the study of energy balance and its components?
5. What research would better inform our knowledge of energy balance and its components?

Question 1: Explain energy balance and imbalance in terms of a biological system in which energy intake and energy expenditure change over time in response to the environment

Human physiology complies with the first law of thermodynamics, which states that energy can be transformed from one form to another but cannot be created or destroyed. This law is usually formulated as follows: the rate of change in body E_S[10] is equal to the difference between the rates of E_I and E_O. All of these terms are expressed as energy per unit of time.

E_I primarily consists of the chemical energy from the food and fluids we consume. E_O includes the radiant, conductive, and convective heat lost; any work performed; and the latent heat of evaporation. E_S is the rate of change in the body's macronutrient stores. The energy balance equation ($E_S = E_I - E_O$) is a statement of the principle of energy conservation.

Components of intake

Energy intake includes 3 major macronutrient groups—carbohydrate, protein, and fat—and a smaller component from alcohol. Once ingested, the net absorption of the major macronutrient groups is variable and incomplete, with fecal losses accounting for ~2–10% of gross E_I. The net absorption of dietary energy components varies among individuals and is dependent on the specific foods eaten, how they are prepared, and intestinal factors.

The metabolizable energy (hereafter referred to as E_I) of a diet represents the difference between the absolute energy of ingested substrates and the energy losses found in feces and urine. Commonly used energy densities for carbohydrate (4 kcal/g, 17 kJ/g), protein (4 kcal/g, 17 kJ/g), and fat (9 kcal/g, 38 kJ/g) represent population

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[10] Abbreviations used: AEE, activity energy expenditure; E_I, energy intake; E_O, energy output; E_S, energy storage; REE, resting energy expenditure; TEF, thermic effect of food.

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averages for metabolizable energy, which is the amount of fuel actually available to cells for conducting biological processes.

Digestibility depends on the composition of the food item and on its content of fiber and other indigestible components. Such components can mechanically limit the access of digestive enzymes to food that would potentially be digestible. For example, nuts and other plant materials have cell walls that cannot be digested by gut enzymes, and they thereby protect the cell contents from digestion if not masticated sufficiently to disrupt the cell structure. These effects can have a large impact on the absorption of ingested macronutrients. The variability in absorptive efficiency depends on many additional factors (eg, gut flora, food preparation, diet composition), which may explain the individual differences in metabolizable E_I.

Components of expenditure (E_O)

Absorbed carbohydrates, proteins, and fats are transformed in vivo to substrates that can ultimately either be oxidized to produce metabolically useful energy that drives biological processes or they may be stored. The rate of whole-body energy expenditure, or E_O, varies within a 24-h period and across the life span. Expended energy reflects fuels metabolized for growth, body maintenance needs, physical activity, pregnancy and lactation, and many other processes.

The main energy expenditure terms are REE, TEF, and AEE. REE is the rate of energy expenditure at rest and comprises approximately two-thirds of E_O. REE varies between and within individuals depending on body size, body composition, and recent energy imbalance. Greater total tissue mass increases REE, and the contribution of lean tissue is greater than fat tissue. Moreover, within lean tissue, high metabolic organs such as the brain, heart, kidney, and liver contribute disproportionately to REE. There is also a large variability in REE (~250 kcal/d, ~1000 kJ/d) that is not explained by differences in body composition (1).

The TEF is the obligatory energy expenditure that is associated with digestion and processing of ingested foods. Diet composition has a strong effect on TEF. There is a hierarchy of macronutrient effects on the magnitude of TEF, with isocaloric amounts of protein > carbohydrate > fat. Normally, TEF is assumed to be a fixed percentage of E_I, but variation between and within individuals occurs. AEE is the energy expenditure rate during activity and can be further partitioned into exercise energy expenditure and nonexercise activity thermogenesis.

Components of storage

Triglycerides, which are present within adipose tissue, are the body's major fuel reserve. A lean adult has ~35 billion adipocytes, each containing ~0.4–0.6 μg triglyceride and totaling 130,000 kcal stored energy. An extremely obese adult can have 4 times as many adipocytes (140 billion), each containing twice as much lipid (0.8–1.2 μg triglyceride) and totaling ~1 million kcal stored energy (2).

E_S reflects net changes in the body mass of carbohydrate, protein, and fat. Carbohydrate is stored mainly in the form of intracellular glycogen in skeletal muscle and liver. The total mass of glycogen is relatively small, several hundred grams, and turnover is rapid; maximal amounts are observed in the postmeal state. Water is weakly bonded to glycogen so that glycogen's synthesis and catabolism also involve alterations in fluid balance. Body protein takes many specific forms and, as with glycogen, is associated with water but at a lower value per gram. Lipid in the form of triglyceride is the largest source of stored energy in most adults and has no water associated with it.

Any imbalance between the intake and utilization of these macronutrients will lead to an alteration in body composition. The energy stored per unit body weight of carbohydrate, fat, and protein varies considerably, especially when accounting for the associated intracellular water. Furthermore, dietary carbohydrate intake has an impact on renal sodium excretion, which results in changes in extracellular fluid. Therefore, changes in body weight are expected when the macronutrient composition of the diet is altered, even when the energy content of the diet is held constant.

The long-term stability of body weight is often considered a marker of zero E_S, and thus energy balance. However, as described above, changes in body weight also include changes in body water, which may be variable, and therefore weight change may not directly represent energy imbalances, particularly over the short term.

Question 2: What are the interactions between the components of energy balance and how are they regulated?

The 3 main terms of the energy balance equation continuously change over time. Beginning at conception, E_S remains positive, on average, throughout growth and development. This positive energy imbalance is reflected by increasing body weight. If adult weight is then maintained over the long term, average E_S approaches zero, and an approximate average state of energy balance is present. However, most adults gain fat throughout their lives and in later life lose skeletal muscle; the energy content of body fat change is much higher than that of lean tissue change. Thus, even with weight stability, "perfect" energy balance over the long term does not occur in most older adults.

Over a 24-h period, a typical person eats several meals during the day, and energy balance is strongly positive during and soon after each meal. Energy output is continuous but with increases due to episodic physical activity and reduction during sleep. Energy balance is thus highly variable over a 1-d period, and this variability is shown in dynamic changes in E_S. Most adults also vary their daily eating and activity patterns; thus, E_S also varies from day to day, with energy balance achieved only when averaged over longer time periods.

The development of obesity by necessity requires positive energy imbalance over and above that required for normal growth and development. As in lean individuals, a state of energy balance over the long term with similar short-term fluctuations in intake and expenditure is also approximated in obese individuals, but in obese individuals this is achieved with a higher amount of body fat.

The counterpart of excess weight gain is a negative energy balance leading to weight loss over time. For example, if an acute reduction in E_I is maintained over time, then, assuming patterns of behavior remain unchanged, changes in the 3 processes—reduced REE, AEE, and TEF—will gradually also lower E_O as weight is lost. Eventually, these passive compensatory effects will lead to a diminishing energy imbalance with ultimate restoration of a steady state at a lower body weight.

Although it is clear that E_I and E_O are part of a biologically regulated system, the exact nature of how this system works in humans has not been fully established. Two different system designs have been generally discussed, a "set point" and a "settling point."

The idea of a set point is borrowed from the field of engineering in which feedback control systems are designed to regulate a particular variable to match a specified target. In contrast, a settling point has traditionally been used to describe a system without active feedback control of food intake and energy expenditure. Models that do not directly specify a set-point value but that include active feedback control have also been called settling-point models. These 2 systems do, in fact, overlap, and there are insufficient data to decide whether one or both are valid. What is clear, however, is that perturbations in the components of energy intake or expenditure result in compensatory changes in these components. These include passive compensatory changes such as an increase in energy expenditure with an increase in body size and active compensation such as changes in food intake after exercise.

The following is a brief review of the interactions among the energy balance components.

Food intake on subsequent food intake

Food intake is temporally variable. We eat meals that reflect the satiation that develops during a meal and satiety between meals. The energy content of a given meal is highly variable between individuals and highly variable between meals in an individual. However, the variation in total caloric intake summed across all meals over a day is far less variable. This suggests that there is meal-to-meal compensation of intake, which is confirmed by a negative correlation between successive meal energy content. If we over- or underconsume energy in one meal, we partially compensate for that intake in subsequent meals during the same day. In addition to variation in intake between meals on a given day, we also vary the amount of food eaten each day. Energy expenditure rarely shows the same degree of variation across days. Hence, we are almost perpetually in energy imbalance on the time scale of hours or days. When a given day's intake and expenditure are plotted against each other, there is little association. It is only when they are averaged over much longer periods (weeks) that there begins to be a balance struck between intake and expenditure (3). The panel emphasized that this is a key point that is sometimes overlooked: energy balance as a concept depends on the time domain over which it is considered. We are always in energy imbalance, but the relative imbalance is greater over the short term than over the long term.

Food composition has been suggested to have a large impact on satiety and satiation. It is generally believed that the major macronutrients differ in their effects, with protein having a greater effect than carbohydrate, which has a greater effect than fat. However, the data are not consistent among all studies. In addition, many environmental factors such as social context, as well as liking and wanting food, play an important role in the energy consumed at a meal.

Satiety and satiation depend on several physiologic and molecular mechanisms. Satiation mechanisms include distension of the gastrointestinal tract communicated to the brain and the secretion of a number of gut peptides that interact with receptors principally in the hind-brain. A factor potentially linked to satiety is the hormone ghrelin, which is produced by the stomach. Ghrelin is unique among known gut peptides in that it is orexegenic. Its production increases with time since the last meal, and injections of ghrelin promote food intake. The hormonal regulation of food intake has been discussed in greater detail elsewhere (4).

In addition, there are a large number of sensory and cognitive stimuli that affect food intake and physiology. For example, liking and wanting food can overcome feelings of satiation and satiety and lead to food intake despite feeling full or not being hungry. Also, sensory-specific satiation can affect food intake—although people may feel full after a large main course of savory food, they are still able to eat a sweet dessert.

Food intake on energy expenditure

After the overconsumption of energy there is an increase in body size leading to a passive increase in E_O. This is due to the following factors: an increase in REE, mainly as a result of an increase in lean tissue mass and to a lesser extent an increase in fat mass; an increase in AEE associated with the increased cost of moving a larger body mass; and an increased TEF due to greater E_I. Finally, there is an additional energy cost for tissue deposition and increased protein turnover.

There has been a long-standing debate about whether, in addition to these passive effects on E_O, there is an active stimulation of expenditure during overfeeding that opposes weight gain; however, there is little evidence for an active effect on REE during overfeeding when one accounts for the additional energy cost of tissue deposition. It has also been suggested that nonexercise activity thermogenesis may increase to partially offset the effect of overfeeding (5). This effect was reported to be $\leq$500 kcal/d (2100 kJ/d), which would be a major compensatory factor for opposing weight gain when caloric consumption is increased, but other studies have failed to find effects of a similar magnitude (6, 7).

During restriction of food intake there is a reduction in whole-body E_O, due in part to the reduction in body mass that follows the lowered calorie intake. This can be accounted for by reductions in REE secondary to loss of lean and fat mass, reductions in AEE due to reduced amounts and costs of activity, a decrease in TEF due to lower E_I, mostly caused by reductions in protein turnover and its associated energy cost. In addition to the passive compensation described above, there is evidence for an active reduction in REE during calorie restriction whose magnitude is dependent on the degree of calorie restriction (8).

Many studies have addressed the effect of meal patterning on REE during weight stability. On average, almost tripling the number of daily meals but providing the same total amount of energy had a barely detectable effect on REE, which suggests that meal patterning does not elicit a greater or lower compensation in expenditure.

The effect of exercise on E_I

If demands for energy are met from food intake then it is often assumed that there must be some mechanism that provides a link between expenditure and intake. However, studies of short duration in which E_O is increased by exercise showed no compensatory change in E_I over 1 or 2 d. As the duration of the studies increased, evidence for compensation emerged with longer-duration studies showing greater but incomplete compensation.

Data from several studies showed no relation between AEE and subsequent weight change. Therefore, low AEE as measured by doubly labeled water at a single time point was not a predictor of weight gain over a protracted period (9–11). Cross-sectional data on AEE that span the recent increase in the prevalence of obesity showed that during this long period of time, levels of AEE have not declined (12). However, recent modeling work has suggested that declines in occupational activity over the past 5 decades could explain the observed increases in body weight over

the same period (13) but only if such activity changes were not compensated for by nonoccupational activity changes in or modulations of food intake.

Exercise interventions result in great individual variation in body weight response. Part of the variation may be due to adherence. However, even when exercise sessions are closely supervised, and hence the adherence issue is eliminated, there is still a tremendous variation in response, with some individuals losing significant amounts of weight and some actually gaining weight (14). Measurements of food intake before and after exercise suggest part of the variability in weight change due to exercise lies in how completely individuals compensate for their exercise prescription with elevated food intake, which corresponds to their hunger after exercise.

The effect of exercise on E_O

A popular idea is that a major benefit of physical activity comes not only from the actual energy that is expended during the exercise itself but also from an after-effect of physical activity on REE. There are data showing a positive effect of vigorous or moderate physical activity on REE. This follows 2 separate phases: a large effect that lasts ~2 h and a smaller but more prolonged effect that could take up to 48 h to return to baseline (15). This is called excess postexercise oxygen consumption and accounts for ~6–15% of the energy expended during an exercise session (16), which adds little to TEE.

Another popular belief is that exercise training results in body-composition changes that generate an additional energy benefit of exercise mediated through REE. But such potential effects of exercise training on REE may have been confounded because the post–exercise training REE was measured too soon after the final exercise bout, contaminating it due to excess postexercise oxygen consumption (15). Measurements that are not so confounded suggest that the impact of exercise training on REE is negligible. Whether habitual exercise produces long-term changes in other components of E_O is unclear.

Exercise interventions may be counteracted by compensatory reductions in physical activity at other times of the day, although the data on this point are mixed. Some studies found that exercise had no overall effect on daily E_O because the individuals reduced their normal activities. Other studies reported that there was no activity compensation from the addition of an exercise intervention and thus an increase in E_O was observed. Indeed, in some studies there was an increase in E_O beyond that accounted for by the exercise alone.

These data emphasize a major point that we would like to reinforce. All of the components of energy balance interact with each other. Consequently, it is absolutely necessary to take all of these interactions into consideration when conducting intervention research in the field of obesity. To take a simple example, it may not be very useful to enhance physical activity but to allow subjects to eat what they wish (and thus compensate for their elevated expenditure).

Question 3: What is the veracity of some of the popular beliefs related to energy balance?

A. "The typically observed weight-loss plateau at 6 to 8 mo after a weight-loss intervention is primarily due to a reduction in energy expenditure, ie, slowed metabolism."

Although the measurement of E_O at the plateau is decreased, it does not decrease to the amount of the prescribed or self-reported energy intake. Thus, the plateau may well be attributed to failure to comply with the diet (17). Modeling studies support this interpretation and suggest that if subjects had complied with the prescribed diet, the plateau due to metabolic change would not have occurred for several years, which would have led to much greater weight loss than that observed (18). These data also emphasize that, whereas it is possible to cognitively intervene in our food intake amounts, such interventions are extremely difficult to sustain because of the biological and psychological drives to eat.

B. "Obesity is due to low energy expenditure, ie, low metabolism."

The existence of a low metabolic rate in obesity was erroneously reported in early studies in which the REE was inappropriately normalized by dividing it by body weight. A simple division of REE by total weight leads to a lower estimate of the mass-specific metabolic rate because obese people have an increased relative amount of body fat, which has a lower metabolic rate than does lean tissue. This normalization error led to the notion that low metabolism was the cause of the obesity. The error was compounded by a misuse of the energy balance concept, which is properly applied only at the level of the entire organism. Thus, it is invalid to consider metabolism per kilogram of body weight, or even per kilogram of fat-free mass, as a component of this system. A balance is not struck between total food intake per individual and expenditure per kilogram but rather between *energy intake per individual* and *energy expenditure per individual*. Lower REE per kilogram of body weight therefore cannot be a "cause" of obesity.

In absolute terms, obese people expend more energy than do their lean counterparts. However, this observation should not be overinterpreted to infer that low REE is not a risk factor for obesity. This is because obese people might have had a lower REE than that predicted for their body size and composition before gaining their excess weight. Therefore, it is unclear the extent to which obesity results from reduced energy expenditure, but it is clear that the maintenance of obesity is not due to reduced energy expenditure.

C. "It takes a reduction of 3500 kcal (15,000 kJ) of energy intake to lose 1 lb of body weight."

The origin of the "3500 kcal per pound" rule is based on the calculated energy content of body weight change and is often misapplied to predict the weight-change time course after a given intervention (19). This is a fundamental error because no time period is specified for that intervention. The impression is given that even a temporary intervention will therefore result in a permanent body weight change. Furthermore, the erroneous application of the rule to predict the impact of a permanent intervention gives the impression that a linear change in body weight is expected over protracted periods of time, which is known to be untrue. Rather, even when perfect adherence to an intervention with no active compensation is assumed, it is generally acknowledged that weight change will slow over time due to passive compensatory changes in energy expenditure that occur with the weight change. Therefore, the panel recommended that the 3500 kcal per pound rule should no longer be used.

With the use of a model that accounts for the passive compensatory effects on E_O, a new rule of thumb representing a best-case scenario has been proposed for the average overweight person: every permanent 10-kcal change in energy intake/d will lead

to an eventual weight change of 1 lb when the body weight reaches a new steady state (~100 kJ/d per kg of weight change). It will take nearly 1 y to achieve 50% and ~3 y to achieve 95% of this weight loss (20).

Whereas the above rule of thumb may be useful for approximate estimations and represents a significant theoretical improvement over the 3500 kcal per pound rule, a more accurate assessment of the amount and time course of predicted weight change for a given reduction in E_I may be very valuable and informative for an individual patient. Newly developed dynamic energy balance models for weight loss require complex calculations that are simplified for users in web-based programs (http://bwsimulator.niddk.nih.gov; http://www.pbrc.edu/the-research/tools/weight-loss-predictor). Model predictions such as these provide a more realistic guide as to what patients can expect with changes in energy balance.

D. "Small changes in lifestyle can prevent or reverse obesity."

Small lifestyle changes in either intake or expenditure (activity) are being increasingly promoted as viable interventions. It is important not to have unreasonable expectations about the impact of such interventions on body weight. Because the 3500 kcal per pound rule has often been used to model the effects of such interventions, unrealistic predictions are frequently made about the likely weight-loss benefits of exercise and dietary interventions that make only minor adjustments to lifestyle. As noted above, it is inappropriate to use the 3500 kcal per pound rule to model the effects of interventions. To illustrate this problem, a 40-kcal/d (170-kJ/d) permanent reduction in energy intake resulting from taxing sweetened beverages has been predicted to result in ~20 lb (9 kg) of weight loss in 5 y according to the 3500 kcal per pound rule, whereas only 4 lb (2 kg) of weight loss is predicted using the new rule of thumb (20).

The recommendation that an overweight or obese person should expend an additional daily 100 kcal (420 kJ) in walking (ie, walking one mile a day), given the new rule of thumb discussed above, would result in a weight loss of ~10 lb (4.5 kg) over 5 y, as opposed to a loss of 50 lb (23 kg) if the 3500 kcal per pound rule is used. Although a 10-lb weight loss can often produce major health gains, which points to a potentially significant benefit of small lifestyle changes, it is not nearly the amount of weight loss from this physical activity regimen that the 3500 kcal per pound rule suggests. Moreover, even the revised rule is an optimistic assessment of weight change because it does not account for the potential active compensation of E_I.

Question 4: What limitations do we face in the study of energy balance and its components?

Our ability to measure precisely individual components of energy expenditure or energy intake is relatively poor in light of the potential impact of small changes described above on body weight, especially over extended time scales in free-living individuals. For example, the doubly labeled water method has a precision of ~5%, which translates to an uncertainty of energy expenditure of >100 kcal/d (420 kJ/d). In addition, the accuracy and precision of energy intake measurements by self-report in free-living individuals are much worse. Thus, the combined error of assessing energy imbalance can easily reach 1000 kcal/d (4200 kJ/d) (21). This potential error prevents evaluation of the benefits of interventions that have a small benefit on weight change over time. New technologies currently in development may be more accurate and precise, but that remains to be seen.

Another limitation that we face is that body weight over a day, and between days, fluctuates unrelated to changes in energy stores because of changes in hydration and alimentary tract content, which are the primary contributors to the typical 1–2-lb day-to-day fluctuations in weight. Yet another limitation we face is that the calculation of the energy deficit generated by a given diet requires knowing the energy requirement to maintain the baseline body weight. As stated above, the imprecision is >100 kcal/d when the most precise methods currently available are used. The uncertainty of baseline energy requirements translates to a considerable interindividual variability of weight loss, even if adherence to the prescribed diet is perfect. For example, if the baseline energy requirement of an overweight or obese person is 100–200 kcal/d higher or lower than measured, then perfect adherence to a diet will result in an error of ~5–10 lb (2.3–4.5 kg) in predicted weight change over a year because of measurement error alone. This limitation is less of a concern in studies designed to measure average differences between groups.

In inpatient studies, more precise measurement techniques are available, which thereby decreases measurement error. For example, whole-room calorimeters can measure E_O with 1–2% precision (22) and weighed, supervised food intake with measured excreta can provide very accurate and precise measurements of E_I. However, such studies do not represent free-living conditions.

Finally, the characteristically long time scale (~1 y half-time) for human body weight and composition changes to occur make it difficult to study comprehensively the dynamics of energy balance because we cannot generally keep humans in metabolic wards for such extended periods. Even in a free-living situation we cannot track E_I or E_O for prolonged periods using current technologies. We are thus limited to "snapshots" of periods of ~2 wk.

Question 5: What research would better inform our knowledge of energy balance and its components?

It is important to recognize that the energy balance system is interactive and complex: a change in one component can affect one or more other components. The panel identified the following important gaps in our knowledge that deserve future investigation:

1. Although we know much from short-term studies about the major components of energy balance, our knowledge is still deficient regarding their interaction over the long term. Therefore, we need long-term, longitudinal studies to learn the details of the relations between components of energy balance and changes in body composition and weight among children and adults.
2. It has been shown that biological and psychological factors affect the components of energy balance. But generally, these have been studied independently of one another and an integrative approach is required. We need to know the relative importance of preingestive factors (cognitive and sensory effects of food/meals) on energy intake, energy balance, and the physiologic response to a meal.
3. Although our knowledge of the broader implications of physical activity and exercise have been investigated, we need to understand the effects of different doses (volume, intensity, pattern, timing) and types (endurance, resistance)

of exercise on *1*) total daily energy expenditure and its components (REE, TEF, AEE), *2*) E_I and food preferences, and *3*) body composition and body weight in children and adults.

4. The individual variation in weight-loss response to energy balance interventions is striking, and therefore we need to know the mechanism or mechanisms responsible for the underlying active compensatory differences in energy intake, food preferences, and body weight in children and adults. In particular, we have almost no information from energy balance studies subsequent to weight loss during the difficult period of weight maintenance. How can we identify population subgroups or even individuals who will respond or not respond to a dietary or exercise intervention?
5. Measurements of energy input and output are neither precise nor accurate enough to allow the calculation of energy balance over the appropriate timeframe needed to understand the mechanisms responsible for excess weight gain. Accordingly, we need to develop new methods that can reliably measure energy balance over extended time periods in free-living people.

The 1-d Consensus Conference included presentations from the following speakers: David Allison (University of Alabama at Birmingham), John Blundell (University of Leeds), Myles Faith (University of North Carolina), James Hill (University of Colorado at Denver), John Jakicic (University of Pittsburgh), Richard Mattes (Purdue University), John Peters (University of Colorado at Denver), Eric Ravussin (Pennington Biomedical Research Center), and Susan Roberts (Jean Mayer USDA Human Nutrition Center on Aging). All authors read and approved the final manuscript. All authors participated equally in the development of the statement.

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