With a world defined in terms of diversity of its people, only 36% of the conference participants were women. On the basis of birth origin, only 0.9% of the participants present were born in developing countries. Edmonton, Canada, and Brussels, Belgium, vied for the most northerly latitude of representation in the conference, whereas the southern limit was defined by Tucson, AZ, and San Antonio, TX. The percentage of the world’s population represented by the combined populations of Belgium, Germany, Canada, and the United States, the nations represented by the participants, is < 7%. So, the chair’s claim edged into hyperbole.

Several hypotheses about this lack of geographic representation occurred to me. Is San Diego really that much further from Bangkok, Rio de Janeiro, or Mexico City than it is from Brussels or Stuttgart? Perhaps there is no interest in or experience with functional foods outside of the Western world, although the International Life Sciences Institute sponsored and published results from a functional food seminar held in Southeast Asia (2). Perhaps there is not the same degree of research expertise and contribution from developing countries that there is from the northern temperate zone. If this is the explanation, it constitutes a serious wake-up call for those of us working in Latin America, Asia, and Africa to invest research resources to earn our place on the platform of upcoming meetings on this growing topic. We represent, after all, the majority of the world’s potential consumers of functional foods. The topic (and such products) impinges on our health, given that commercial globalization has already brought outlets for so-called physiologically active food components to Guatemala and many other countries of Latin America. So even if researchers into this topic are yet to be found in the academic institutions of developing countries, at least the roster of the discussants could have gone beyond Arizona, Texas, and 3 sites within the Washington, DC, Beltway.

A few other observations are worthy of mention. Despite its title, relatively little evidence or experience was shared on aging save for the associations of vitamin E and immune function (3) and some passing references to Alzheimer disease (4). When reading the supplement, one should focus on the words of John Fernstrom, eg, “I am concerned at this moment more about safety than efficacy,” because he alone cried out to orient the priorities to the logical axis in any paradigm for interventions for human health amid a wilderness of enthusiastic advocates.

The lessons I derived from receiving my Ross Research Conference proceedings as an AJCN supplement were profound but not all of them were of an academic nature.

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REFERENCES


Metabolic response to weight loss

Dear Sir:

Several years ago Leibl et al (1) reported data suggesting that altered body weight produces changes in energy expenditure that favor a return to original body weight. Additional data from this group suggest that the compensatory changes in energy expenditure might be related to changes in thyroid or catecholamine status, or both (2). In this recent report, Rosenbaum et al (2) mentioned that “previous studies did not achieve the degree of weight stability and control of nutrient intake of this study and did not examine the same subjects during dynamic weight change as well as during static weight maintenance at altered body weights.” In fact, we reported the metabolic responses of subjects during dynamic and energy balance phases of weight loss under tightly controlled metabolic ward conditions (3, 4). Our findings provide a potentially important contrast in results.

We measured resting metabolic rate (RMR), thyroid hormones, and plasma and urinary catecholamines in 24 overweight postmenopausal women (3). The metabolic variables were first assessed in the static phase of energy balance in the overweight state after 10 d in the General Clinical Research Center (GCRC). Subjects were then provided an energy-restricted diet containing 3347 kJ/d (800 kcal/d) while they were outpatients of the GCRC until they reached a normal body weight. They returned to the GCRC while weight-stable or both (2). In this recent report, Rosenbaum et al (2) mentioned that “previous studies did not achieve the degree of weight stability and control of nutrient intake of this study and did not examine the same subjects during dynamic weight change as well as during static weight maintenance at altered body weights.” In fact, we reported the metabolic responses of subjects during dynamic and energy balance phases of weight loss under tightly controlled metabolic ward conditions (3, 4). Our findings provide a potentially important contrast in results.

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More recently, we studied 32 overweight premenopausal women under weight-stable conditions in the GCRC before and after weight loss and found that RMR decreased after normalization of body weight (6). Again, however, the new RMR was appropriate for the reduced FFM and fat mass. Urinary norepinephrine and dopamine also decreased after weight loss, but the changes were not significantly different after adjustment for changes in FFM (R Weinsier et al, unpublished observations, 2000).

It is of more than academic interest to point out the similarities and differences between the results of our studies and those of Rosenbaum et al because both were conducted under tightly controlled metabolic ward conditions with use of identical energy restrictions (3347 kJ/d). Arguably, the outcomes should have been similar. Both studies suggest that during negative energy balance, RMR is reduced disproportionately to the decreases in FFM and fat mass and that thyroid hormones change in association with (and may possibly explain) the changes in energy requirements (5). However, our data suggest that on adaptation to a weight-reduced steady state, RMR and thermogenic hormones are appropriate for the subjects’ new body composition and do not predispose the subjects to regain the lost weight.

Differences in the 2 studies might have contributed to the conflicting outcomes. The subjects studied by Rosenbaum et al were severely obese, with an average BMI of ≈48, whereas our subjects were overweight, with a BMI of 28. This weight difference could account for the subjects’ different metabolic responses. However, we believe that more plausible explanations exist. First, Rosenbaum et al’s more obese subjects had an absolute RMR value ~50% higher than that in our subjects. Thus, the fixed 3347-kJ/d energy-restricted diet would have produced an energy deficit almost twice that in our subjects. We confirmed in a subset of our subjects that 10 d of stabilization in the weight-reduced state was sufficient to produce a metabolic steady state (4). However, the considerably greater energy deficit in Rosenbaum et al’s subjects could have necessitated a period longer than the 14 days they used to establish a steady state. Second, different statistical approaches were used to adjust RMR for changes in body composition after weight loss. We adjusted RMR for FFM and fat mass by using analysis of covariance (5, 7). Rosenbaum et al’s findings in the earlier report of reduced RMR after weight loss (1) appear to have been based on the ratio of RMR to FFM, which may sometimes lead to spurious results (8, 9). When they adjusted RMR for changes in FFM and fat mass by using regression analysis, RMR values after weight loss were apparently not significantly different from baseline values (1).

In summary, both studies indicate that during the dynamic phase of energy restriction, thyroid hormones change in conjunction with reductions in RMR, placing subjects in an energy conservation mode. The findings of Rosenbaum et al suggest that on return to energy balance conditions, weight loss is associated with changes in thermogenic hormones which might, by virtue of effects on energy expenditure, favor a return to usual body weight. By contrast, our findings suggest that the energy-conserving metabolic changes persist only during the period of energy restriction. Once energy balance is restored, metabolic variables appear to normalize and become appropriate for the new reduced body mass and do not explain the weight-regain tendency of these subjects (7).

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REFERENCES


Reply to R Weinsier et al

Dear Sir:

Weinsier et al raise important issues regarding our studies of the effects of weight loss on systems of energy homeostasis, including carbohydrate metabolism, catecholamine excretion, and thyroid function (1). The nature and magnitude of the changes in energy metabolism that accompany weight reduction are critical issues from both physiologic and therapeutic perspectives. In their letter, Weinsier et al describe points of similarity and distinction between our respective studies of the effects on energy homeostasis of maintenance of a reduced body weight. We found that both the process of weight loss and the maintenance of a reduced body weight are associated with significant declines in total 24-h energy expenditure (TEE), resting energy expenditure (REE), and non–resting energy expenditure (NREE) beyond those expected from the changes in metabolic mass (1–3). In our recent article (1), we reported that both weight loss and the maintenance of a reduced body weight are associated with significant declines in urinary norepinephrine and dopamine excretion and circulating concentrations of triiodothyronine (T3). In earlier articles, we described decreases in REE in some subjects under these circumstances (2, 3). Weinsier et al (4, 5) noted similar decreases in REE, circulating T3 concentrations, and urinary catecholamine excretion during weight loss, but found that these decreases did not persist during sustained maintenance of a reduced body weight.

The differences in our results may have been due to differences in our subject populations and study designs. In our previous studies (2, 3), we found that maintenance of a reduced body weight (≥10% below initial body weight) is associated with a significant decline in REE adjusted for fat-free mass (FFM) in obese premenopausal subjects but not in subjects who have never been obese. Weinsier et al point out that the obese subjects we studied were significantly fatter than the obese subjects they studied: the mean (±SEM) fat mass in our subjects was 67 ± 3 kg compared with 30–31 ± 1 kg in Weinsier et al’s subjects (4, 5). Our obese subjects had significantly (≈10%) higher REE (adjusted for FFM) at their usual body weights than did our never-obese subjects. We concluded that this relative increase in REE was due to increased cardiopulmonary work in the more obese subjects. After weight loss, the REE (adjusted for FFM) of our reduced-obese subjects was significantly lower than it had been in the same subjects before weight loss, but was similar to that of our never-obese subjects at their usual body weight. Thus, the adjusted REE of our reduced-obese subjects was not significantly lower than that of the never-obese subjects. The persistent decline in REE in our obese subjects during weight maintenance at a reduced body weight may reflect their higher REEs at usual body weight compared with Weinsier et al’s “leaner” obese subjects.

The subjects in our inpatient studies were intentionally restricted to an amount of physical activity designed to maintain a degree of physical fitness equal to that on admission to the study (1–3). In contrast, Weinsier et al’s subjects were not restricted with regard to physical activity (4, 5). The weight loss in Weinsier et al’s subjects was apparently due to both a hypocaloric diet and physical activity [which increased by 33% in the weight-reduced subjects (4)], whereas the weight loss in our subjects was intentionally achieved solely through a reduction in energy intake. Several studies showed that the addition of exercise to a weight-loss regimen will significantly blunt the decline in REE that occurs during and after weight loss (6–12). Thus, the lack of association of maintenance of a reduced body weight with lowered REE in Weinsier et al’s subjects may also reflect effects of increased physical activity during their outpatient weight loss.

We showed previously that maintenance of a reduced body weight is associated with a significant decline in sympathetic nervous system tone as measured by effects on heart rate variability of sequential pharmacologic blockade of the sympathetic and parasympathetic branches of the autonomic nervous system (13). Schwartz et al (14) reported that weight loss induced by diet alone results in a 17% decline in urinary norepinephrine excretion (similar to the values in our studies), but that the addition of exercise to the weight-loss regimen abolishes this decline. Although we are aware of no studies that examined the effects of exercise training on the thyroid axis during maintenance of a reduced body weight, the observation that exercise increases sympathetic nervous system output (as reflected in blunting of the weight-loss-associated decline in urinary norepinephrine excretion) suggests that the decline in T3 might also be mitigated with exercise through sympathetic nervous system–mediated effects on the thyroid axis (1).

The observations by Weinsier et al (4, 5) indicate that the decreases in catecholamine excretion and in circulating concentrations of thyroid hormones that accompany weight loss by diet alone may be ameliorated by increased physical activity during weight loss or maintenance of a reduced body weight. However, despite the prevention of these endocrine adaptations to body weight reduction, the weight-reduced subjects studied by Weinsier et al still needed to increase their physical activity by ≈33% to maintain a reduced body weight without decreasing energy intake (4). Thus, Weinsier et al’s subjects apparently experienced the same decreases in total energy expenditure experienced by our subjects and compensated for this decline by their increase in physical activity. Weinsier et al’s observation that TEE does not change significantly after weight loss in weight-reduced subjects who significantly increase their physical activity agrees with our observation that TEE declines significantly in weight-reduced subjects who do not change their physical activity from baseline after