Special Article

A *Festschrift* for Roland L. Weinsier: Nutrition Scientist, Educator, and Clinician[1]

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Abstract

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Roland L. Weinsier, M.D., Dr.P.H., devoted himself to the fields of nutrition and obesity for more than 35 years. He contributed outstanding work related to the treatment of obesity through dietary and lifestyle change; metabolic/energetic influences on obesity, weight loss, and weight regain; body composition changes accompanying weight loss and regain; the health benefits and risks of weight loss; nutrition education for physicians; and nutrition support of sick patients. He served on the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) National Task Force on Prevention and Treatment of Obesity, as Chair of the University of Alabama at Birmingham's Department of Nutrition Sciences, and as Founder and Director of its NIDDK-funded Clinical Nutrition Research Center. He was a long-time and active member of NAASO, serving in the roles of Councilor, Publications Committee Chair, Continuing Medical Education Course Director, Public Relations Committee Chair, and Membership Committee Co-Chair, to name just a few. He was well respected as a staunch defender of NAASO's scientific integrity in these roles. Sadly, Roland Weinsier died on November 27, 2002. He will be missed and remembered by many as a revered and beloved teacher, mentor, healer, and scholar.

Key words: nutrition science, medical nutrition education, medical nutrition practice

Pathophysiology of the Metabolic Syndrome

The metabolic syndrome, also known as the insulin resistance syndrome and syndrome X, represents a specific body phenotype in conjunction with a group of metabolic abnormalities that are risk factors for coronary heart disease. The notion of a metabolic syndrome was first considered more than 50 years ago by Vague, who noticed that an increase in upper body fat, which he called masculine or android obesity, was observed commonly in obese men and was associated with diabetes, coronary heart disease, and gout (1). Since then, the concept of a metabolic syndrome has advanced and was formally defined in 2001 (2). Roland Weinsier contributed to our recognition of the features of the metabolic syndrome and described the relationships between upper-body obesity and hypertension (3) and hypertriglyceridemia (4).

The characteristics of the metabolic syndrome include excess abdominal fat, insulin-resistant glucose metabolism (hyperinsulinemia, impaired glucose tolerance, impaired insulin-mediated glucose disposal, type 2 diabetes mellitus), dyslipidemia (hypertriglyceridemia, low serum high-density lipoprotein-cholesterol concentration), and hypertension. Additional metabolic abnormalities associated with abdominal obesity that are also risk factors for coronary heart disease have been identified (5,6): increased serum concentrations of apolipoprotein B; small, dense low-density-lipoprotein particles; and plasminogen activator inhibitor 1

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Table 1. Defining criteria for the metabolic syndrome

Risk factor	Defining level
Increased waist circumference	
Men	>102 cm (>40 inches)
Women	>88 cm (>35 inches)
Triglycerides	≥150 mg/dL (1.69 mM)
HDL cholesterol	
Men	<40 mg/dL (1.04 mM)
Women	<50 mg/dL (1.29 mM)
Blood pressure	≥130 mm Hg systolic or ≥85 mm Hg diastolic
Fasting blood glucose	110 to 125 mg/dL (≥6.1 mM)

Three or more of these criteria are needed to diagnose metabolic syndrome.

with impaired fibrinolysis. Obesity itself is not a requirement for the metabolic syndrome; metabolically obese, normal-weight persons, presumably with increased abdominal fat mass, have been identified (7). Recently, the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) established specific clinical criteria for diagnosing the metabolic syndrome, shown in Table 1 (2). The prevalence of the metabolic syndrome in adults increases linearly with increasing age. In the United States, the prevalence of the metabolic syndrome in men and women is ~7% in those who are 20 to 29 years old and increases to ~45% in those who are age 60 years and older (8).

An excessive release of free fatty acids into plasma may be responsible for the insulin resistance and dyslipidemia associated with the metabolic syndrome, by impairing the ability of insulin to stimulate muscle glucose uptake (9) and suppress hepatic glucose production (9) and by stimulating hepatic very-low-density lipoprotein triglyceride production (10). Roland Weinsier made important contributions to our understanding of the interrelationships between obesity and weight gain, abdominal fat, and race. In a series of elegant and difficult-to-perform 2- to 4-year longitudinal studies (11,12), he demonstrated that: 1) visceral fat gain is associated with total body fat gain and is not a consequence of aging; 2) white women have more visceral fat than black women; and 3) weight gain and weight loss affect visceral fat mass and the ratio of visceral fat to subcutaneous abdominal fat differently in white than in black women.

Because even modest losses of 5% to 10% of body weight in obese patients who have the metabolic syndrome improve all aspects of the syndrome simultaneously (13), weight loss is the treatment of choice in this patient population. It is not known whether weight loss in lean patients with the metabolic syndrome also has beneficial effects; this issue deserves further study. The mechanism(s) responsible for the beneficial effect of weight loss is not completely known, but the mechanism(s) could relate to weight-loss-induced effects on fatty acid and lipoprotein metabolism and hepatic and intramuscular fat content (14,15).

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The Role of Physical Inactivity in Obesity

An inverse relationship between current activity-related energy expenditure (AEE)[2] and obesity has been reported. This suggests that physical activity (PA) may influence weight gain and obesity; however, it does not rule out the possibility that inactivity is caused by weight gain and obesity, rather than the reverse. If increased PA helps to prevent weight gain, high PA must be predictive of low weight gain. With Roland Weinsier, we have recently shown this using doubly-labeled water. Using a human post-obese model under tightly controlled energy balance conditions, we found that previously obese women who maintained a lower body weight had AEE over 42% higher and PA over 40% higher than women who gained weight ($>$ 6 kg/year) (1). The gainers gained 8.9 kg more fat and 1.6 kg more lean mass than the maintainers during the average of 1.1 years of follow-up. Using equations reported by Forbes (2), we calculated that the difference in energy balance required to produce this difference in body composition was 274 kcal/d. AEE accounted for 212 kcal/d, or 77%, of this difference, suggesting that PA had a strong influence on the resistance to weight gain found in the maintainers, even stronger than diet (1).

For the weight gainers to have the same AEE as the maintainers, the gainers would have to expend an additional 4 kcal/kg body weight per day, or 78 min of moderate intensity exercise. This estimate is very similar to the estimate of 80 min/d of moderate intensity exercise by Schoeller et al. (3). It would be even greater (101 minutes) if no dietary restriction were present.

We believe that some combination of PA and dietary restriction must be used by most Americans to prevent weight gain. These data indicate that being physically active is helpful in maintaining weight. Therefore, increasing PA is important for maintaining weight.

[2] Nonstandard abbreviations: AEE, activity-related energy expenditure; NIDDK, National Institute of Diabetes and Digestive and Kidney Diseases; PA, physical activity; UAB, University of Alabama at Birmingham; REE, resting energy expenditure; FFM, fat-free mass; PAL, PA level; TPN, total parenteral nutrition; ABN, American Board of Nutrition; ASCN, American Society for Clinical Nutrition.

$$\downarrow \text{fitness} \rightarrow \downarrow \text{ease of PA} \rightarrow \downarrow \text{PA} \rightarrow \uparrow \text{wt} \;\Rightarrow\; \downarrow\downarrow \text{fitness} \rightarrow \downarrow\downarrow \text{ease of PA} \rightarrow \downarrow\downarrow \text{PA} \rightarrow \uparrow\uparrow \text{wt}$$

$$\text{resistance training} \rightarrow \uparrow\uparrow \text{fitness} \rightarrow \uparrow\uparrow \text{ease of PA} \rightarrow \uparrow\uparrow \text{PA} \rightarrow \text{no wt} \uparrow$$

Figure 1: Top line depicts model for progressive decrease in PA and increased weight gain. Bottom line depicts proposed effects of resistance training. wt, weight.

It is possible that muscle function may be related to the ease of activity and the likelihood of being active. We have recently shown that more participation in free-living PA is related to greater $V\text{O}_{2\text{max}}$ and less difficulty in being active (4). In addition, we found that strength-training-induced increases in muscle function are associated with increases in free-living total energy expenditure in older adults (5). Quadricep strength is also independently related to endurance (4). One explanation for a relationship between strength and endurance could be that less muscle activation is needed to perform a task by a stronger muscle, hence delaying fatigue. Muscle metabolic economy (measured with ^{31}P magnetic resonance spectroscopy), $V\text{O}_{2\text{max}}$, and quadricep strength are all independently related to weight gain. We have found that 9 weeks of resistance training improves exercise economy (5). We have also found that ease of performing daily tasks, as measured by normalized integrated electromyography and heart rate, is improved after resistance training (6).

Overall, these findings suggest that low fitness leads to reduced PA and reduced PA leads to weight gain. The cycle continues with weight gain and low PA leading to further decreases in fitness. Improved muscle metabolic economy and strength may make it easier to be physically active. It is suggested that physical training may break the cycle by improving strength, making PA less demanding, increasing PA, and decreasing weight gain (Figure 1).

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Genetic and Environmental Influences on Obesity

Roland Weinsier's chosen lifestyle and scientific research were both affirmations of the importance of the environment and behavior to body-weight regulation. However, a substantial body of research has documented the profound effect of genetic variations on inter-individual differences in adiposity (1). This research may seem to contradict the importance of the influence of environment and behavior, but Roland Weinsier's recent work, which embraced both genetic and environmental factors, illustrates that there is no inherent contradiction and that both factors are involved in the determination of adiposity. The nature of the environmental and genetic influences is complex and can be best understood from multiple perspectives using multiple scientific approaches.

The increasing trend in obesity levels that has occurred over the past several decades supports the strong environmental influence on obesity (2). Although genes may influence the degree of susceptibility to environmental changes, the gene pool is unlikely to have changed substantially in this time (3). This strongly suggests that the recent increase is due to changes in the environment. In searching for environmental factors, most investigators look to relatively recent events and focus on changes in the commercial food supply and technological developments that putatively lead to lesser PA. Before we accept that the recent increase in obesity represents a sharp acceleration we should carefully review long-term data.

Several reports suggest that the increase in obesity has actually been occurring for 100 years or more and may not be accelerating at all. Helmchen (4) studied data from U.S. veterans 50 to 59 years of age who were examined in 1905 and 1909 and found that the veterans were more than three times as likely to be obese than their counterparts examined 25 years earlier, an annual average increase of 4.5% in obesity rate. Data from the National Health and Nutrition Examination Surveys conducted from the 1970s to the 1990s showed an annual increase in obesity rates of roughly 4%. Helmchen concluded that obesity was increasing at least as rapidly at the beginning as it was at the end of the 20th century. Similar conclusions were reached by other investigators [e.g., Okasha et al. (5)]. Environmental factors, such as industrialization, central heating, vaccinations, reductions in infectious diseases, increased availability of food, and changing attitudes, all of which may have both near-term and trans-generational (perhaps epigenetic) effects, should be considered as causes for the increase in obesity.

Calorie availability is one obvious environmental factor to consider. The amount of calories available per person per day has increased over the past several decades (6). It is unclear whether this is a major cause of the increase in obesity rates or simply a reflection of increased demand for calories due to increased obesity rates or an enabling, but not truly driving, factor. The proportion of energy consumed as fat can also be correlated with rates of obesity in different populations (7). Similarly, a tight correlation can be drawn between obesity rates at any time in recent years and the percentage of households with televisions and cars (8). Other factors may be less obvious. Bluestone and Rose (9) showed that in recent years the number of hours worked per person has substantially increased on average. Thus, just as obesity levels have gone up, free time has gone down. Could increased obesity levels result, in part, from decreased free time available for healthier food preparation, PA, planning of stress-reducing events, etc.? From an econometric perspective, Komlos et al. (10) have hypothesized that the increasing trend in obesity is part of a general pattern of worrying less about the future and living for today. Nevertheless, these ecological correlations can only suggest hypotheses, not confirm them. We could find correlations between obesity rates and virtually any factor that has progressively changed over time or that varies with economic wealth across countries.

The evidence for a genetic influence on obesity is equally compelling (11). Animal breeders have known for years that animals can be selectively bred for increased or decreased body weights or adiposity. Many scientists recognize that natural selection has bred us to be relatively good at seeking out and storing energy. This is critical because throughout history periodic shortages of energy have been a major threat to survival (12). As a species we are predisposed to obesity in our current rich environment. However, some data strongly indicate that approximately two-thirds of the inter-individual differences in relative adiposity are due to differences in genotype (13). Genetic studies have also shown the transience of the environmental influence. Factors that influence body weight at one point in time tend to have little influence many years later. This indicates that, to influence obesity, putative environmental factors should be in place throughout the life span and not simply at one point (14,15).

Specific genes influence body weight or body composition. For example, knocking out the perilipin gene results in dramatically leaner-than-usual mice by interfering with the storage of lipid in adipocytes without reducing food intake. The *ob/ob* mouse is a well-known genetic model of obesity that is homozygous for a mutation in the leptin gene, which causes the nonfunctional form of a protein to be produced; mice become dramatically obese by increasing food intake, showing the genetic effect working through behavior. Koteja et al. (16) showed that mice can be selected for increased spontaneous activity, producing strains that have dramatically increased PA and, at least for males, lower body weights. Substantial evidence from other species shows that it is possible to selectively breed for increased or decreased food intake (1), showing the strong genetic influence on behaviors involving obesity. Other genetic effects may influence response to the environment or diet. For example, the PTP-1B knockout mouse appears to be phenotypically normal when fed a usual diet, but is obesity resistant when fed a high-fat diet, showing the complexity of the gene–environment interaction (17). Mayeux-Portas et al. (18) found that mice homozygous null for the *Thy-1* gene (a cell-adhesion molecule) did not adjust their food intake in response to environmental cues for eating as do normal mice, suggesting that sensitivity to the social environment is, in part, genetically determined. Our group showed genetic influences on objectively measured food intake behavior among humans (19).

Albert Einstein said, "Make things as simple as possible but no simpler." Consideration of either genes or environmental factors in isolation makes things too simple. Reality is more complex. Roland Weinsier has provided us with an example of considering both factors; it is the job of future researchers to further clarify this complex set of influences.

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The Birmingham–Lausanne Collaboration: A Decade of Joint Research on Energy Metabolism

International, national, or regional collaborations among investigators start with an exchange of ideas and may simply stop there. It is always a challenging task for both parties. On the two occasions in which Dr. Weinsier came to Lausanne University for a sabbatical, several fruitful and synergistic interactions were developed, primarily in the field of energy metabolism and PA, an area of great scientific interest for both parties.

A general outline of the diversity of clinical and nonclinical studies performed by Dr. Weinsier gives evidence of a logical progression of his research throughout his career (Figure 2).

Fat Oxidation and Resting Energy Expenditure (REE)

The collaboration was initiated with the exploration of factors controlling substrate oxidation in women of different body fat, from normal to overweight to obese (1). This constitutes the initial vision of the research, during which Dr. A. Tremblay (Laval University, Canada) was also present in Lausanne. The study outlined a new model suggesting that a change in body fat mass engenders a change of fat oxidation in the same direction: the higher the body fat mass, the greater the resting fat oxidation. It was concluded that the rise in fat oxidation accompanying the body fat gain in the dynamic phase of obesity constitution contributes to the long-term control of fat balance in overweight and obese individuals (1). A subsequent study showed evidence that in post-obese women who have lost body fat, fat oxidation decreases, as evidenced by a significant rise in respiratory quotient (2), a finding that, in a longitudinal manner, corroborated the previous study. Although the concept was challenged in a subsequent analysis a few years later (3), it remains essentially intact, especially after the discovery of leptin, a hormone synthesized in adipose tissue with lipolytic properties.

Measurement of fat oxidation requires a very accurate and precise indirect calorimeter. However, in a clinical setting, it is virtually impossible to assess on a routine basis either the REE or substrate oxidation of an obese patient. Consequently, using a rather heterogeneous sample of Swiss women ($n = 600$), it was decided to assess whether simple anthropometric measurements (easily determined by the clinician) could predict REE in women classified into five groups of increasing BMI (averaging from 18.9 kg/m^2 to 44.6 kg/m^2). A prediction equation for REE was derived from the whole sample of women using body weight, height, and age as factors, a sort of "Harris–Benedict-like" equation, but hopefully valid for overweight and obese individuals. The predictive equation was:

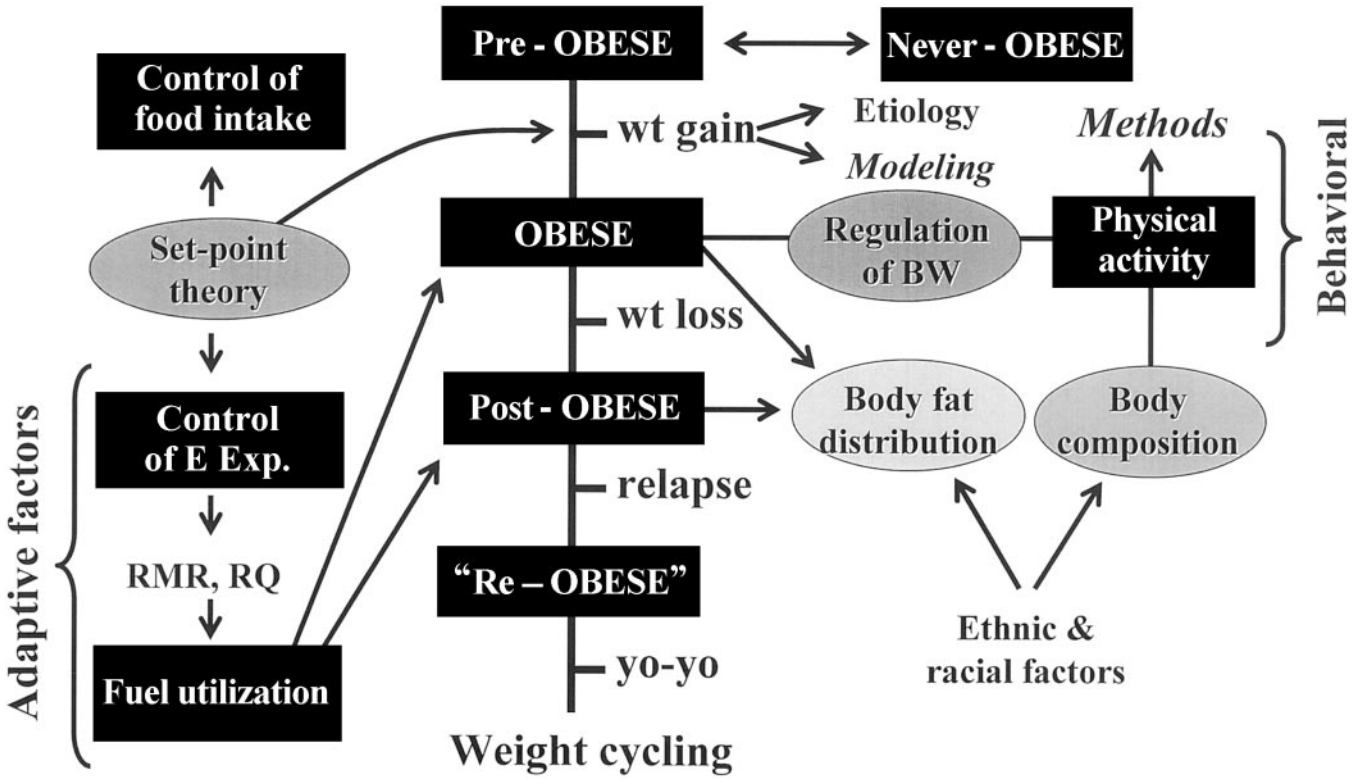

Figure 2: Comprehensive research overview directed by Dr. Weinsier: a logical progression.

$$REE(kcal/d) = 394 + 10.2 \times weight(kg) + 2.85 \times height(cm) - 3.8 \times age(years)$$

Compared with the standard Harris–Benedict, this equation had a lower *Y* intercept. However, a similar weighted factor for body weight was observed. The effect of body height on REE was amplified, whereas the effect of age was slightly reduced as compared with the Harris–Benedict equation.

At extremes of BMI (<20 and >40 kg/m^2), we must admit that the accuracy of most anthropometric equations to predict REE becomes uncertain. Predictive equations seem to be reasonably accurate for groups of subjects, but have an SD of ~8%. Therefore, at BMI extremes, one should measure REE by indirect calorimetry, rather than attempting to predict it from anthropometry.

The legitimate superiority of body composition measurements over anthropometry to predict REE (4) has been confirmed, but, at the population level, anthropometric based equations are still useful for clinicians not familiar with body composition, and, interestingly, both standard equations (e.g., Harris–Benedict type) and recent ones (such as that derived here with a much larger number of subjects) are rather consistent in their predictions of REE.

Predictive Models of Body-Weight Gain

Another challenging area of research relates to the time course of weight gain in humans (5), when small energy imbalance is produced by a net decrease in energy expenditure. The historical basis of this analysis was a classic, somehow disturbing, statement which has occasionally been seen in textbooks on obesity: for example, "a small daily increase of energy intake, will, over time, result in substantial weight gain." This assertion does not take into account the fact that there is a dynamic interaction between energy intake and energy expenditure that tends to maintain long-term energy equilibrium. A more correct statement would be "a small but persistent excess of energy intake relative to energy expenditure will, over time, result in substantial weight gain." If the net excess energy intake is not persistent, due to adaptive metabolic mechanisms and the variant response of energy expenditure, enormous weight gain is neither predicted nor observed with small, chronic, positive-energy imbalances.

Modeling of body-weight change when there is a small decrease in energy expenditure, based on previous analysis on the metabolic activity of fat-free mass (FFM) (6), led us to the following conclusions: 1) the time course of weight gain to achieve energy balance (after an initial energy dislocation) is longer for obese subjects; 2) the REE response rises more slowly with increasing degrees of obesity; 3) the absolute amount of weight gain is also greater in the obese: it was calculated that the magnitude of weight gain in response to a reduced energy expenditure ranging from 50 to 200 kcal/d is ~6 to 13 kg in the lean individuals, but much higher (by ~50%) in the obese individuals ranging from 9 to 19 kg. This indicates that reduced energy expenditure has a greater negative impact on obese individuals, who are more prone to gaining further weight with fewer counteractive adaptive processes, than lean individuals.

Table 2. Comparison of physical activity indices

	Nonobese	Borderline obese	Percent variance
BMI (kg/m^2)	25	30	
REE (kcal/d)	1480	1550	
24 hour EE (kcal/d)	2150	2200	
Activity EE (kcal/d)	450	450	
PAL_{24h}	1.45	1.42	−2%
PAL_{day}	1.65	1.58	−4%
PAR	4.0	4.5	+12%
METs	3.4	3.4	0
ARTE (minutes)	152	110	−28%

The table shows the variances among different estimates of physical activity when an obese woman is compared to a lean woman having the same daily energy expenditure due to PA (AEE).
PAR, PA ratio; MET, metabolic equivalent; ARTE, activity-related time-equivalent.

Physical Activity

PA remains the cornerstone for controlling body weight with aging, for maintaining FFM during weight loss, and for preventing relapse after diet-induced weight loss. There are several ways to assess PA, and a number of excellent reviews have been published on this topic. However, one issue rarely discussed is the different modes of expression of PA modalities. For example, PA can be expressed in energy terms (i.e., kilocalories or kiloJoules per day), which is useful for nutritionists to calculate total energy requirement. Alternative indices, such as duration of PA (in minutes per day, or minutes per week), intensity of activity expressed as metabolic equivalents, PA ratio, and PA level (PAL) are not always suitable for investigations. Proxies of PA, such as pedometers, can also be used to assess the number of steps taken per day. Accelerometers are of particular interest because they are able to quantify both the level and duration of PA (7).

More recently, a new index called the activity-related time-equivalent, proposed by Dr. Weinsier's group (8), is of great interest for comparing the activities of groups of individuals with different physical characteristics (e.g., obese vs. controls). It represents an indication of the amount of time spent at an energy expenditure level equivalent to that of a composite reference of standardized activities.

To show the disparity of results with different PA indices, a comparison of a nonobese person (BMI of 25 kg/m^2) and a borderline-obese person (BMI of 30 kg/m^2) is presented in Table 2.

The development of PAL, not only applied over 24 hours (as currently used by the Food and Agriculture Organization of the United Nations and the World Health Organization), but at a level that takes into account only daytime activity (PAL_{day}), may be useful in future work because it excludes the confounding effect of sleep duration.

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Thermogenesis and Human Obesity

Thermogenesis, or, more specifically, the lack of thermogenesis, has often been hypothesized to be a cause of human obesity. Reports that obese individuals consume less dietary energy than lean controls support the hypothesis, as do a large number of animal studies that demonstrate a deficient thermogenic response of brown adipose tissue in genetically obese rodents (1). Cross-sectional measurements of the thermic effects of meals on humans have provided mixed results, but they frequently find that obese individuals do display a lower rate of thermogenesis than lean controls (2). The difference, however, is much smaller than the dramatic 50% increases in energy expenditure observed in rodents, and it usually amounts to $<$100 kcal/d lower energy expenditure in humans (3).

In an effort to manipulate thermogenesis, many investigators have performed studies in which human subjects were overfed $\geq$1000 kcal/d. Only seven of these studies included a measure of total daily energy expenditure using doubly-labeled water; these constitute the best available evidence for the investigation of changes in energy expenditure. Six of these studies found no increase in expenditure beyond the $\sim$10% to 15% associated with increases in body size and meal size (3). The seventh found an increase in expenditure that accounted for 50% of excess caloric intake, that is, of the magnitude of the thermic effects of feeding in rodents. Surprisingly, the increase was not due to thermogenesis per se, but rather to an increase in nonexercise PA (4). Therefore, these human overfeeding studies do not support the thermogenesis hypothesis, but one of the studies suggests that PA may be the important factor.

Few longitudinal studies focusing on the role of energy expenditure in weight gain have been performed. Although one study did find that a low resting metabolic rate adjusted for FFM, fat mass, gender, and age did predict weight gain (5), that finding was not confirmed by Weinsier et al. (6). Indeed, in the latter study, neither resting metabolic rate nor the thermic effect of meals predicted weight gain. Rather, two recent studies (one of Weinsier's and one of ours) did find that the portion of total energy expenditure used for PA predicted weight gain (7,8). Active women maintained weight over a 1-year follow-up, whereas sedentary women gained weight. Both studies found evidence of a threshold of activity associated with the prevention of weight gain. Specifically, it was found that subjects who were active enough to raise their ratio of total energy expenditure to REE above 1.7 were better able to maintain their weights than were subjects who were less active. This corresponded to the addition to a sedentary lifestyle of 80 to 90 min/d of moderate activity, such as a brisk walk, or 30 to 40 minutes of vigorous activity, such as aerobics. Of note, this is approximately the same level of PA that successfully maintains weight as reported in a retrospective study of the National Weight Loss Registry database (9).

An important aspect of both of these studies is that most of the subjects were individuals who had recently undergone weight loss. In one study, all of the subjects were previously obese women, whereas the other included both previously obese women and never-obese women. It is possible that the level of PA required to prevent weight gain in previously obese persons may differ from that for never-obese individuals, as the previously obese individuals had, by definition, demonstrated a propensity toward weight

gain. Additional longitudinal studies in other subject populations are needed to further address the role of PA in weight maintenance. These studies, however, strongly indicate that the development of obesity in humans is not related to deficient dietary thermogenesis, but to insufficient PA. This is a significant paradigm shift in obesity research.

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Debunking Old Hypotheses in the Regulation of Energy Metabolism and Creating New Ones: The Influence of Roland Weinsier and the Alabama Studies

In reviewing the accomplishments and scientific progress from the 10-year period that I worked with Dr. Weinsier, I became surprised with how this work challenged fundamental beliefs and led to new theories and paradigms in our general understanding of the regulation of energy metabolism in humans, the relationship between obesity and insulin resistance and increased risk for type 2 diabetes, and, in particular, ethnic differences in these factors. Dr. Weinsier's contribution to this work, briefly reviewed here, ranged from his invaluable critiques as a colleague to co-authorship of papers.

During this time period, we completed two longitudinal studies in children that examined the natural history of the development of obesity in growing children (1,2). These studies did not support the notion that low energy expenditure predicted the development of obesity. However, we did find evidence that low levels of PA, as indicated by low fitness, predicted the development of obesity (2). On a similar note, we also examined whether a lower energy expenditure in African Americans may have contributed to the higher levels of obesity in this subgroup of the population. Our studies in children, however, failed to support this notion (3–5), and, again, there was more evidence to suggest lower fitness among African-American children (6).

The work in Alabama also allowed a focused effort on the study of why African Americans have a greater risk of developing type 2 diabetes. We showed that visceral fat is present in children (7), develops during growth (8), and contributes to disease risk (9). In addition, we showed that visceral fat was lower in African-American children (7) and that greater insulin resistance was independent of both total adiposity and visceral fat (9). Finally, we showed that the increased insulin response to glucose infusion among African Americans could not be explained simply by an increase in secretion. Instead, we showed that the increased insulin was due, in part, to an increase in secretion, as well as a decrease in clearance in the liver (10).

Collectively this work tends to support the idea that fat mass is not necessarily the predominant factor explaining increased insulin resistance among African Americans. Rather, the obesity-related risk for developing type 2 diabetes is due to a constellation of risk factors, probably acting in an additive fashion affecting insulin resistance, and the eventual development of type 2 diabetes probably depends on β-cell function and the ability or inability to compensate for the degree of insulin resistance.

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Role of Energy Density in Food Intake Regulation

In the early 1980s, Roland Weinsier and his colleagues published several papers on the dietary management of obesity that have had a significant impact on our understanding of eating behavior and satiety (1–3). Rather than following the traditional emphasis on varying the macronutrient composition of the diet, Dr. Weinsier proposed that consumption of a diet low in energy density (calories per gram) would enable patients to consume a satisfying volume of food even when restricting calories (1).

With his typical intellectual curiosity and broad interests, Weinsier did not stop at investigating the clinical applications of a dietary strategy based on the energy density of foods. He wanted to know why such a diet worked. In one of his most highly cited articles (2), he and his colleagues hypothesized that consumption of a diet low in energy density affects intake by slowing eating and enhancing satiety. They tested this theory over two 5-day periods, during which both obese and lean subjects ate as much as they wanted of either a high-energy-density diet (high in fat, meat, dessert) or a low-energy-density diet (high in fresh fruits, vegetables, whole grains, and low in fat). Energy intakes and ratings of hunger and fullness were measured daily. The results showed that spontaneous energy intake over 5 days was nearly halved during consumption of the low-energy-density diet (1570 vs. 3000 kcal/day). When eating the high-energy-density diet, both obese and lean subjects ate more quickly and were more likely to eat until they were unpleasantly full than when eating the diet lower in energy density. Furthermore, although the participants rated themselves as slightly less hungry before meals high in energy density, they continued to eat more calories than when eating meals lower in energy density.

This study not only shed new light on satiety, but it was also extraordinarily advanced in the methods employed. Twenty years ago, few studies tested both obese and lean individuals, few studies measured intake for more than a day, and long-term studies often relied on self-reported intakes rather than accurately weighed intake measurements. Hunger and satiety were rarely assessed. To this day, only a few such thorough and well-controlled studies of satiety have been conducted.

A major conclusion of Dr. Weinsier's study was that the amount of energy consumed is not the sole, or even a major, determinant of satiety, at least not on a short-term basis. The study laid the foundations for a growing body of literature on the influence of the energy density of foods on energy intake (4,5). Surprisingly, although the study participants ate nearly twice as many calories on the high-energy-density diet, there was little difference in the amount of food consumed on the two diets. On average, subjects consumed 2243 grams per day on the low-energy-density diet and 2000 grams on the high-energy-density diet. This finding that individuals in *ad libitum* feeding studies consume a consistent weight of food, even when the energy density is varied, has been confirmed in a number of subsequent studies (4,5).

Weinsier and his colleagues (2) concluded that a diet low in energy density "might be advantageous for the prevention as well as the treatment of obesity." The unlimited consumption of vegetables and fruits, combined with some reduction in dietary fat, allowed participants to eat a satisfying volume of food while restricting calories. Always concerned for the health of his patients, Dr. Weinsier also determined that a diet low in energy density was adequate to meet nutritional needs (3). He left us with the following wise thoughts about weight management, thoughts that are often forgotten in our current quest for a solution to the obesity epidemic (1): "Weight loss can be achieved with any energy-restricted diet, regardless of its composition, and can, thus, be promoted as an effective dietary regimen. The end result, however, does not necessarily justify the means. Weight control should be considered as only one way in

which diet can improve health; i.e., the diet should be directed at overall good health, one aspect of which is weight control."

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Applying Energy Density: The *EatRight* Weight Management Program

Roland Weinsier established the University of Alabama at Birmingham (UAB) Nutrition Clinic in 1976 to treat nutrition-related disorders. He developed and published the *Time-Calorie Displacement Approach to Weight Control* for use in this clinic to treat obesity (1). His approach was based on the concept of energy density: ingesting large volumes of low-energy-dense foods requires more time and produces satiety at a relatively low energy intake, thus leading to weight loss (2). In 1990, the name of the weight-management program was changed to *EatRight*.

In this program, an initial calorie level is prescribed with a specified number of servings from five food groups: fats/oils, meat/dairy, starches, fruits, and vegetables. In the fats/oils and meat/dairy groups, only a maximum number of servings is prescribed; for starches, a range; and for the fruits and vegetables groups, a minimum number of servings is prescribed.

In 1983, Dr. Weinsier participated in a committee convened by the International Congress of Obesity to develop therapeutic guidelines for the three components that should be included in obesity-treatment programs: diet, exercise, and behavior modification. The committee recommended that any dietary program used to treat obesity should have a sound scientific rationale, be safe and nutritionally adequate, and be practical and effective for long-term weight management (3). Not surprisingly, the *EatRight* program met these guidelines. Weinsier's initial work, discussed above by Dr. Rolls, established a sound scientific rationale for the program (4). With regard to nutritional adequacy, calculated intakes of vitamins and macronutrients in the *EatRight* program compare favorably with Recommended Dietary Guidelines (2). Further, vitamin and mineral levels measured at baseline and after 10 weeks and 20 weeks in 26 obese individuals who lost 0.7 kg/wk in the *EatRight* program showed no significant changes except for vitamin C, which increased (5).

To examine its long-term efficacy, subjects in the *EatRight* Weight Management Program were evaluated in a retrospective study (6). Of 213 obese subjects who participated in the program over a six-year period, follow-up weights were determined in 147 subjects who could be contacted. Subjects lost, on average, 6.3 kg during 7 months of treatment. Importantly, after a mean follow-up period of 2 years, over one-half of the patients were at or below their post-treatment weight, and the mean net weight loss had not diminished. This contrasts with weight-loss programs in general, where almost all weight that is lost is regained within 5 years.

Limited additional data support the efficacy of a low-energy-density dietary program on body weight. A 14-day crossover study found that energy density, independent of diet composition, influenced energy intake and body weight (7). A 3-month study at Mayo Clinic demonstrated that a low-energy-density diet with unlimited amounts of vegetables and fruits led to greater weight loss than did an energy-restricted diet (D.D. Hensrud, unpublished data). A review that evaluated studies of the dietary components that affect energy density, including fat, fiber, and water, reported that consumption of lower-energy-density foods leads to decreases in energy intake and body weight (8).

In summary, Roland Weinsier's accomplishments came full-circle with this work, from clinical research to clinical practice. The *EatRight* program continues today and has treated over 2000 individuals since 1997. It has produced three books (1,9,10). This work has also inspired other programs to use the principles of energy density to help people manage their weight and, most importantly, to improve their health.

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Treating Obesity in Primary Care Practice: A Feedback Gym Program

As pointed out above, scientists have been showing that moderate physical exercise has a determining role in weight control. Recently, Weinsier et al. delivered precise measures confirming this hypothesis and identifying muscular strength as one of the responsible parameters (1). The positive impact of strength training on energy expenditure has been repeatedly shown in other studies. In addition, recent prospective data show the importance of cardiorespiratory fitness on health outcomes (e.g., improved cardiovascular survival) (2,3). In the therapeutic approach to the obese patient, behavioral imbalance often limits adherence to diet and exercise programs.

Based on these considerations, I developed a structured weight-control program with medical and dietary management in combination with a coached training and wellness center, all located in the same facility. The center provides guided, progressive aerobic and strength training with documentation of training frequency and periodic interactions among training coach, physician nutrition specialist, and dietitian.

Preliminary results from the program's initial 6-month period are convincing, showing, as expected, loss primarily of fat mass, while maintaining lean body mass, and improved physical condition. However, the most notable observation is that the training coach acquired a substantial and central role in the behavioral management of the obese patients. Through ongoing training supervision, the trainer becomes spontaneously aware of behavioral patterns and emotional aspects of the patients and communicates these to the physician and dietitian. This dialogue provides a spirit of a "caring team" that has, by itself, a reassuring effect on the patient. As a result, subjective self-assessment of well-being (using a visual analogue scale) showed favorable changes in all patients.

We propose to call this new concept of interactive training a "feed-back gym." In this setting the following goals are achieved:

- An efficient impact on body composition and, therefore, on weight management.
- An improvement of cardiorespiratory fitness and, therefore, a basis for a decreased cardiovascular risk.
- A favorable effect on functional capacity (back, joints, etc.) by improved muscular strength and better mobility.
- Successful behavioral change by accomplishing the training program.
- An opportunity for the therapists (training coach, physician nutrition specialist, and dietitian) to approach further behavioral disorders through their interactive network (feedback).

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Fine Tuning Our Understanding of Ethnic Differences in Obesity

The prevalence of obesity in African-American women, already observed for several decades to be substantially

above average (1), has now reached truly alarming proportions, even when viewed in the context of the overall epidemic of obesity in the U.S. population. As of the 1999–2000 National Health and Nutrition Examination Survey data, the prevalence of obesity (BMI $\geq$30 kg/m^2) was 50% among black women, compared with 28% in black men, 30% in non-Hispanic white women, and 27% in non-Hispanic white men (2). Fifteen percent of black women have class 3 obesity (BMI $\geq$40 kg/m^2, equivalent to $\sim$100 lb or more overweight). Excess obesity in African-American females, although once observed primarily in adulthood, is now also observed during childhood and adolescence (3).

Roland Weinsier and his colleagues have brought much needed objectivity and systematic reasoning to the effort to understand black/white differences in female obesity, particularly by tackling the issue of whether ethnic differences in energy metabolism are the primary cause. The hypothesis that reduced metabolic rate leads to obesity stems from an observed association of low REE with weight gain in Pima Indians (4). This hypothesis was extended to black/white differences based on numerous reports of poorer outcomes in weight-loss programs (i.e., less weight loss) among black compared with white participants (5–9), together with several observations of lower REE in blacks compared with whites (10), and one report that suggested a greater reduction in REE in response to weight loss in blacks than whites (11). The underlying question is whether ethnic differences in weight gain and obesity development are due to factors that are modifiable vs. innate and not readily modifiable. The answer to this question has obvious implications for how prevention and treatment should be approached.

Luke et al. (12) reported no association of REE (adjusted for body composition) with differences in body fat between Nigerians and African Americans. In an accompanying commentary, Goran and Weinsier (13) questioned the biological plausibility of the purported causal relationship of REE with obesity development. They also suggested that the impression that REE is inappropriately low in African Americans may stem from the inability to adjust completely for ethnic differences in the composition of lean tissue, specifically organ size. Hunter and Weinsier et al. (14) subsequently reported data supporting this line of reasoning. Many other relevant findings come from studies in which matched samples of overweight black and white women were compared with regard to energy expenditure, fitness, and body composition variables before and after weight loss to within the normal weight range (BMI $<$25 kg/m^2) (15,16). Taken together, these studies suggest that lower activity-related energy expenditure rather than REE may predispose black women to excess weight gain.

Fitting together all the pieces in this complex puzzle will require further exploration in at least the following directions, in both children and adults: establishing whether our current measures of REE are equally reflective of true metabolic size across ethnicity; understanding which, if any, specific components of energy expenditure differ by ethnicity once metabolic size has been considered; determining the trajectories of weight loss and regain in black and white women over time; clarifying ethnic differences in the energy cost of PA and in total energy expenditure after weight loss; evaluating changes in body composition, including analyses for subtypes of body fat, with weight loss in ethnic groups (17); and determining the implications of these changes in body fat for energy balance. Closer attention is needed to the assessment of PA in both observational studies of weight change and weight reduction interventions. Finally, given the probable dependency of individual and average ethnic group responses to weight loss on the dose of PA or exercise in the program, studies of ethnic differences in response to interventions involving different doses of exercise may be informative.

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Nutrition Support

The mid-1970s and early 1980s were exciting times for physicians and other healthcare providers interested in new challenges and opportunities in clinical nutrition. Although Roland Weinsier published only three first-author articles that directly pertain to hospital nutrition support, they addressed critical issues in the field that are as pertinent today as they were in 1979 to 1982: assessment of the malnourished patient and judicious use of specialized nutrition support. His observations helped lay the foundation for policies and procedures commonly employed today.

Charles E. Butterworth, Jr., the founding Director of the Nutrition Program and the Department of Nutrition Sciences at the UAB, published a provocative landmark article in 1974, entitled "The Skeleton in the Hospital Closet," that drew attention to iatrogenic malnutrition at a time when miraculous advances were occurring in other areas of medicine (1). After joining the department in 1975, Dr. Weinsier published one of the first systematic studies documenting a high prevalence of malnutrition among hospitalized general medical patients (by far his most-cited article; Ref. 2). Of 134 patients, 48% had a high likelihood of malnutrition at the time of hospital admission. Not only did this condition correlate with a longer hospital stay, but nutrition status worsened during hospitalization in 69% of follow-up patients. Other articles at the time showed similar findings, with hospital malnutrition prevalence rates ranging from 44% to 50%. These important early observations prompted the establishment of nutrition screening and assessment processes that are currently mandated by the Joint Commission for the Accreditation of Healthcare Organizations (3) and prompted the establishment of nutrition care guidelines such as those of the American Society for Parenteral and Enteral Nutrition's 2002 Guidelines for the Use of Parenteral and Enteral Nutrition in Adult and Pediatric Patients (4). All patients admitted to U.S. hospitals are now screened for risk of malnutrition.

An equally significant contribution to the early field of nutrition support was Weinsier and Krumdieck's 1981 article warning of the danger of overzealous use of total parenteral nutrition (TPN). The authors reported the biochemical responses, pathophysiology, and deaths of two extremely cachectic patients who received high-dextrose infusions of TPN (5). In the title of the article, they referred to "the refeeding syndrome revisited," recalling the unfortunate deaths of concentration camp victims who, after surviving the horrors and starvation of internment, succumbed to the refeeding that occurred on liberation from the camps. The refeeding syndrome is now well defined (6) for all practitioners of nutrition support, and guidelines for judicious refeeding are standard-of-care procedures (7).

As an additional warning to the potential complications of TPN, Weinsier et al. conducted a prospective study of the frequency of metabolic abnormalities among 100 medical and surgical patients receiving TPN (8). High prevalence rates of serious hyperglycemia, hypophosphatemia, and hypokalemia were observed. The authors suggested that "greater attention to the prevention and management of the metabolic complications of [TPN] by physicians would improve patient care and extend the usefulness of this form of nutritional support." These critical observations, among others, led to the creation of the American Society for Parenteral and Enteral Nutrition, whose goal is the promulgation of safe and effective patient care by nutrition support practitioners.

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Teaching, Training, and Certifying Physicians

Individuals who knew Roland Weinsier only for his accomplishments in obesity research may be surprised at the substantial contributions he made to medical nutrition education. For most of his career, he placed a high priority on teaching nutrition to medical students, residents, subspecialty nutrition fellows, and physicians in practice. Charles E. Butterworth, Jr., and Carlos L. Krumdieck, who recruited Roland to UAB in 1975, had inserted a few hours of nutrition lectures into the first-year medical school curriculum, but it was Dr. Weinsier's vision that expanded those lectures into a 50-contact-hour freestanding course, Introduction to Clinical Nutrition, in 1976. It was, and probably still is, the most intensive such course in any U.S. medical school. Dr. Weinsier was the first to introduce smaller-group clinical correlations into the UAB School of Medicine curriculum before 1980; these have now become a standard teaching method in most medical schools. He always taught a major portion of the course, and although he recruited others to teach in their areas of expertise, he attended every lecture.

In the late 1970s, Dr. Weinsier also started an elective clinical rotation for fourth-year medical students and internal medicine residents. As he modeled clinical nutrition practice at the bedside and in the clinic, he gained their respect as an expert physician who cared for patients and believed that all healthcare practitioners should integrate nutrition into the care of their patients. In 1981, Dr. Weinsier initiated UAB's Fellowship in Clinical Nutrition, making it one of the earliest and longest-standing nutrition fellowship programs in the U.S. He also made contributions to continuing medical education in an annual UAB Nutrition Symposium between 1984 and 1994 and through many lectures at continuing medical education conferences around the country.

On the conviction that nutrition education was needed at all medical schools, Dr. Weinsier broadened his activities to a national scale. He received grants to develop and evaluate educational programs from the American Board of Nutrition (ABN), the Donner Foundation, the Health Resources and Service Administration, the National Dairy Council, the National Live Stock and Meat Board, and the Ruth Mott Fund. The latter grant funded a national workshop in 1983 that led to a National Nutrition Test-Item Bank, a computer bank of nearly 4000 test items that was intended to serve as a national resource for nutrition educators. In 1984, Dr. Weinsier was appointed to the American Society for Clinical Nutrition (ASCN) Committee on Medical-Nutrition Education, and he chaired it from 1987 to 1990. Under the committee's auspices, he spearheaded the Southeast Regional Medical Nutrition Education Network, a network of 11 medical schools that documented wide variations in the nutrition knowledge of medical students and correlated it with the quantity and quality of nutrition content in the schools' curricula. In 1988, he convened a consensus conference of ~100 medical school deans and nutrition educators that developed and published content priorities for teaching nutrition to medical students. Under his leadership, the committee also conducted and published a nationwide study that identified critical components of effective nutrition training for medical residents. In 1991, Dr. Weinsier proposed, established, and obtained initial funding for the ASCN′s Award for Excellence in Medical/Dental Nutrition Education; this is now one of ASCN′s important annual awards.

Dr. Weinsier was also committed to maintaining a high-quality certification process for physician nutrition specialists. He served on the Board of Directors of the ABN for 11 years and took responsibility for developing its examination from 1984 to 2000. He moved the ABN′s national secretariat office to UAB in 1994 and hosted it until it was superceded by the Intersociety Professional Nutrition Education Consortium. He served as the NAASO's representative to that consortium from its inception in 1997 and as a member of the Board of Directors of its newly established American Board of Physician Nutrition Specialists until his death.

Dr. Weinsier was also a prolific contributor to nutrition education texts and articles. With Charles E. Butterworth, Jr., and Douglas C. Heimburger, he conceived and co-authored the *Handbook of Clinical Nutrition*, which has sold more than 50,000 copies in three editions (1981, 1990, and 1997, Mosby, St. Louis, MO). With Sarah L. Morgan, he co-authored two editions of *Fundamentals of Clinical Nutrition* (1993 and 1998, Mosby, St. Louis, MO). He wrote chapters for *Medicine for the Practicing Physician*, *Cecil's Textbook of Medicine*, and *Conn's Current Therapy*, and was to have served as a co-editor of the 10th edition of Shils et al., *Modern Nutrition in Health and Disease* (2004). He

authored or co-authored 19 peer-reviewed medical nutrition education articles, examples of which involved the following: describing the findings of his research on factors critical to medical nutrition education (1); helping establish the National Nutrition Test-Item Bank (2); developing a national status report on medical nutrition education (3); comparing nutrition knowledge and attitudes of medical students in different institutions (4); co-authoring national consensus priorities for nutrition content in medical school curricula (5); developing components of effective clinical nutrition training in residency programs (6); and co-authoring materials for training and certifying physician nutrition specialists (Intersociety Professional Nutrition Education Consortium and the American Board of Physician Nutrition Specialists). Dr. Weinsier also helped found the Southeast Regional Medical Nutrition Education Network.

Dr. Weinsier also served in a number of national educational advisory positions including the National Academy of Sciences Food and Nutrition Board's Committee on Nutrition in Medical Education in 1984; a U.S. House of Representatives hearing on nutrition education in 1983; and the University of North Carolina's Nutrition in Medicine project's Advisory Committee from 1993 to 2001. He was an invited visiting professor at Fu Jen University, Taipei, Taiwan, in 1988; the University of London Medical Schools, England, in 1990; the University of Lausanne School of Medicine, Switzerland, in 1991 and 2000 (while on sabbatical); and the Mayo Clinic, Rochester, MN, in 1996. His medical education awards included Best Freshman Medical School Professor Award at UAB in 1981; Best Medical Basic Sciences Professor Award at UAB in 1983 and 1987; the National Dairy Council Award for Excellence in Nutrition Education, ASCN, 1995; and the UAB President's Award for Excellence in Teaching in 2000.

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Vale

Roland Weinsier had an extremely productive career, encompassing research, teaching, patient care, administration, and national and international consultations and policy making. He authored more than 150 peer-reviewed journal articles and 16 books or book chapters. He collaborated with almost 300 co-authors.

His 10 most cited articles are listed below. However, some of his most important work has surely been published in the last several years (and perhaps has yet to be published by his collaborators), so it will be interesting to revisit his citation record 20 years from now.

Dr. Weinsier held a broad range of national advisory positions, serving on 18 committees for federal organizations (Federal Trade Commission, Health Resources and Services Administration, National Research Council/National Academy of Sciences, NIH, U.S. Congress, and U.S. Department of Agriculture/Department of Health and Human Services) and on 23 committees for professional organizations (ASCN, American Society for Nutritional Sciences, NAASO, ABN, American Heart Association, American Society for Parenteral and Enteral Nutrition, International Congress on Obesity, Intersociety Professional Nutrition Education Consortium, American Board of Physician Nutrition Specialists). Among other awards, he received the C.E. Butterworth, Jr., Professorship in Nutrition Sciences from UAB, 1996–1999; the Joseph F. Volker Distinguished Faculty Award from the UAB School of Health Related Professions in 2000; and the Jonathan E. Rhoads Honorary Research Lectureship from the American Society for Parenteral and Enteral Nutrition in 2001. He chaired the UAB Department of Nutrition Sciences from 1988 through 1999. He is missed by all who knew and worked with him.

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Roland Weinsier's 10 Most-Cited Articles

1. **Weinsier RL, Hunker EM, Krumdieck CL, et al.** Hospital malnutrition—prospective evaluation of general medical patients during the course of hospitalization. *Am J Clin Nutr*. 1979;32:418–26 (244 citations).
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