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RESEARCH ISSUES: THE FOOD ENVIRONMENT AND OBESITY

Energy Density, Energy Intake, and Body Weight Regulation in Adults[1–5]

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ABSTRACT

The role of dietary energy density (ED) in the regulation of energy intake (EI) is controversial. Methodologically, there is also debate about whether beverages should be included in dietary ED calculations. To address these issues, studies examining the effects of ED on EI or body weight in nonelderly adults were reviewed. Different approaches to calculating dietary ED do not appear to alter the direction of reported relations between ED and body weight. Evidence that lowering dietary ED reduces EI in short-term studies is convincing, but there are currently insufficient data to determine long-term effectiveness for weight loss. The review also identified key barriers to progress in understanding the role of ED in energy regulation, in particular the absence of a standard definition of ED, and the lack of data from multiple long-term clinical trials examining the effectiveness of low-ED diet recommendations for preventing both primary weight gain and weight regain in nonobese individuals. Long-term clinical trials designed to examine the impact of dietary ED on energy regulation, and including multiple ED calculation methods within the same study, are still needed to determine the importance of ED in the regulation of EI and body weight. *Adv Nutr 2014;5:835–850.*

Introduction

Obesity is one of the major health crises of our time. The majority of adult Americans are now either overweight or obese (1), and recent research indicates that obesity is approaching smoking as the major cause of disability and premature death (2,3). National improvements in dietary intake, and in particular a reversal of the documented increase in energy intake (EI)[6] (4–6), are clearly an important key to preventing unwanted weight gain and associated comorbidities. However, there is no general consensus on how to achieve this important goal.

Of the many dietary factors suggested to play an important role in the regulation of EI, energy density (ED) has received particular attention (7–13) because small changes in the ED of the diet, if uncompensated for by alterations in the quantity of food consumed, could lead to large cumulative changes in EI. The ED of a food can be defined as the metabolizable energy content per unit weight of a food (kJ/g or kcal/g) (11) and is determined by the macronutrient and moisture content of the food. As the most- and least-energy-dense nutrients, fat [2.15 kJ/g (9 kcal/g)] and water (0 kJ/g), are the primary determinants of ED.

Dietary ED can be defined as the ED of the total diet. At present, no consensus has been reached on the appropriate method for calculating dietary ED, with debate centering on the inclusion of beverages in the calculation (8). Studies have used different definitions of dietary ED that vary predominantly by whether some or all beverages are included in the calculation and, if so, what types. For example, studies have used ED values based only on food, whereas others have included both food and energy-containing beverages, and some have included food and all beverages (8,14,15).

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[6] Abbreviations used: DASH, Dietary Approaches to Stop Hypertension; ED, energy density; EI, energy intake; TDEI, total daily energy intake.

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The goals of this review are to summarize information relevant to standardizing a definition of dietary ED, provide a review of primary research publications examining the effects of ED on EI and body weight in adults, and suggest future research directions for elucidating the role of ED in body weight regulation.

Critical Review of the Literature

Literature search, selection of studies for inclusion, and data extraction. A literature search conducted by using PubMed identified English-language clinical and observational studies examining relations between ED and EI, appetite, and body weight and/or BMI. Studies in which ED was an explicit independent variable as well as studies in which ED was not an explicit variable or outcome but could be calculated from reported results were considered. Reference lists of these publications and relevant review articles were searched to identify additional germane studies.

The eligibility criteria for the studies reviewed herein are outlined in **Table 1**. In particular, we focused on studies in nonelderly adults (age 18–60 y) because regulation of EI is impaired in the elderly (16–18). We also generally included studies regardless of the method used to alter ED. As a result, resolving independent effects of ED on EI and body weight from effects of dietary factor(s) that also change when ED is altered (e.g., dietary fat and fiber content, water content of foods, palatability) becomes difficult. This is especially true in studies long enough to demonstrate changes in body weight. However, independent effects of ED are less relevant when one considers that, in free-living individuals, changing dietary ED inevitably alters multiple dietary components. Therefore, for this review we chose to consider ED as 1 dietary factor among many rather than as an independent dietary determinant of EI and body weight. The 1 exception is that studies aiming to determine the effects of adding fiber to meals were not selected for review. Although interventions using added fiber may reduce dietary ED, the reduction is small and any effects on appetite are likely outweighed by the established physiologic effects of fiber (19,20). Readers are referred to a recent comprehensive review summarizing the evidence for effects of fiber on EI and body weight (21).

A total of 92 relevant studies were identified that met all of the eligibility criteria (15,22–112). These studies were then classified by study design. Observational studies were recognized to be potentially confounded by bias in reported EI and dietary ED but, with this qualification, were included to explore the effect of inclusion or exclusion of beverages on relations between BMI and ED. Shorter-duration clinical studies (duration of <1 mo) were deemed relevant for providing mechanistic evidence of a role of ED in energy balance regulation. Only those trials in which all or most food was provided to and consumed by subjects in a laboratory setting were selected rather than studies that used self-reported EI as the key outcome because of well-recognized inaccuracies in self-reported EI (113,114). Longer-duration clinical studies (duration of ≥1 mo) reporting change in body weight, BMI, or body fat as a primary outcome were deemed to provide the most conclusive evidence on relations between ED and body weight.

To facilitate interpretation of the evidence, clinical studies in which ED was the independent variable and EI or body weight the dependent variable were categorized according to study duration as follows: *1*) preload and single-meal studies, *2*) interventions of 1–3 d in duration, *3*) interventions of 3 d to 4 wk in duration in which change in EI from the provided food was the outcome, and *4*) interventions ≥1 mo in duration in which change in body weight

TABLE 1 Criteria and rationale for study exclusion[1]

Exclusion criteria	Rationale
Applied across all study designs	
Mean age of study population <18 or >60 y	Energy regulation dysregulated in older adults; physiologic differences between children/adolescents and adults
Studies in clinical or unique populations (e.g., pregnant, binge-eating disorder, etc.) (51,122,123)	Not reflective of general population or typical physiologic conditions
Studies reporting data that had been presented as part of an earlier report (124–128)	Likely to report similar findings
Observational studies	
Studies using an FFQ to measure dietary intake and not excluding nonplausible reporters (129)	Measurement error associated with FFQ may confound associations
Small sample size (130)	High probability of type I error
Clinical studies	
No comparison group (131)	Effect of ED modification cannot be determined
ED not reported and cannot be determined with confidence if change in ED differed between groups (includes studies that did not measure EI) (132–140)	Unclear if ED was altered
Duration of ED manipulation differed between subjects (141)	Unable to be grouped with other studies
Studies <1 mo duration in which the majority of the diet was not provided by study investigators (e.g., EI was measured by food record or diet recall) (132,142–148)	High measurement error

[1] ED, energy density; EI, energy intake.

was the outcome. Together, these divisions permitted an examination of the evidence for acute, short-, and longer-term effects of ED modification on appetite and energy regulation.

In addition to information on study population and design, the following information was extracted from each study to facilitate comparisons of clinical studies: *1*) preload and single-meal studies [ED of the manipulated meal or preload and EI during the manipulated meal (single-meal studies) or the sum of preload EI and EI at the subsequent meal (preload studies)], *2*) interventions of 1 d to 4 wk in duration [dietary ED and total daily EI (TDEI)], and *3*) long-term studies (dietary ED and body weight change). Only main effects of ED were considered, although several studies examined interactions between ED and an additional factor (e.g., portion size, fat proportion, sex, dietary restraint, eating rate). Any relevant data reported in graphical rather than text format was estimated from figures.

Observational studies. Twenty cross-sectional studies (15,22–37,91,92,94) and 7 prospective cohort studies with follow-up ranging from 6 mo to 8 y (26,38–42,90) examining associations between dietary ED and either body weight or BMI were identified (**Tables 2** and **3**). Twelve of 17 cross-sectional studies reported a positive association between ED and BMI or overweight/obesity (24,27–29,32–36,91,92,94). An additional 3 studies noted an association between ED and BMI that was modified by sex: 2 studies reported a positive relation in women and no relation in men (25,30) and the third reported a positive relation in certain age groups of men but no relation in women (31). Eight studies compared mean dietary ED between obese (BMI $\geq$30 kg/m^2) and normal-weight (BMI <25 kg/m^2) subjects. Mean dietary ED was significantly higher in the obese individuals in only 3 studies (30,33,34), with all studies observing an estimated difference of <0.06 kJ/g (0.25 kcal/g).

Differences in dietary intake assessment methods may underlie inconsistent findings. Food diaries were used in 5 studies, FFQs in 3, and 24-h food recalls in the other 12 (Table 2). Of the studies that used food diaries, 4 (80%) failed to observe a positive association between EI and BMI, whereas only 1 study (8%) that used 24-h recalls and no studies that used FFQs failed to find any evidence of a positive association.

Results from prospective cohort studies appeared to support a positive association between dietary ED and BMI or overweight/obesity but suggested that relations between ED and prospective weight change may be modified by weight status. Six studies explored whether associations between dietary ED and prospective weight change differed between normal-weight and overweight subjects. A positive association between ED and weight gain (41,90) or change in waist circumference (38,40) was reported in 4 studies. One study confirmed this association in a normal-weight cohort but also reported that higher dietary ED at baseline was associated with weight loss in an overweight cohort (38). In contrast, 2 studies documented positive relations between ED and weight gain in overweight cohorts but no association (42) or an inverse association (26) in a normal-weight cohort. Finally, 2 randomized trials examined associations between dietary ED and weight change during intentional weight loss by combining data from all subjects participating in the respective trials (43,44). Lindström et al. (44) reported greater weight loss in individuals in the lowest compared with highest dietary ED quartile during the intervention. Similarly, Ledikwe et al. (43) reported that individuals having the largest reduction in dietary ED while enrolled in the PREMIER trial achieved a 3.5-kg greater weight loss over 6 mo compared with individuals with a slight increase in dietary ED during the trial.

Taken together, findings from observational studies are somewhat inconsistent but generally support a positive association between self-reported ED and both BMI status and weight gain. Nonetheless, causality cannot be determined from observational studies, and given the known biases in dietary reporting and the inconsistencies noted in cross-sectional studies, the possibility that the results were confounded by limitations in dietary assessment methodology cannot be ruled out.

Inclusion of beverages in ED calculations. Several observational studies used multiple methods of calculating dietary ED by including or not including beverages and/or varying the types of beverages in the calculation. Although numerous methods have been examined [e.g., (14,15)], the most common methods used for calculating dietary ED included food only, food and energy-containing beverages, food and all beverages except for water, and food and all beverages in the calculation. Associations between ED calculated by using any of these 4 methods and indicators of weight status were extracted from study reports allowing an examination of whether including beverages in the calculation of ED alters conclusions regarding associations between dietary ED and body weight.

As summarized in Tables 2 and 3, 5 cross-sectional (15,23,27,28,33) and 2 cohort (42,90) studies reported associations between dietary ED and body weight when ED was calculated both with and without the inclusion of beverages. The direction and statistical significance of the associations reported in these studies were generally not altered by the method used to calculate ED.

Single-meal crossover studies examining the effect of including noncaloric beverages with a meal on ad libitum EI are also relevant (56,115–117). As recently reviewed by Daniels and Popkin (118), and summarized in **Figure 1**, these studies demonstrated that, relative to a no-beverage control condition, lowering the ED of an ad libitum meal by including noncaloric beverages had no effect on EI. Furthermore, decreasing the ED of a meal by including caloric beverages with the meal resulted in increases in EI that approximated

TABLE 2 Cross-sectional studies examining associations between dietary ED calculated with and without the inclusion of beverages and weight status[1]

Authors, year (reference)	Population and age	Diet assessment method	Dietary ED		Main findings	
			Normal-weight (BMI <25 kg/m^2)	Obese (BMI ≥30 kg/m^2)	Food only	With beverages
			kcal/g			
Cox et al., 2000 (15)	75 M/F; 43 y	FD	1.36 0.76[2] 1.17[3]	1.40 0.83[2] 1.14[3]	No differences in dietary ED between normal-weight and OB	No differences in dietary ED between normal-weight and OB[2,3]
Cuco et al., 2001 (22)	572 M/F; 25–65 y	FR	—	—	—	Dietary ED not associated with BMI[3]
de Castro, 2004 (23)	952 M/F; 35 y	FD	—	—	Dietary ED not associated with BMI	Dietary ED not associated with BMI[2]
Esmaillzadeh et al., 2011 (91)	486 F; 40–60 y	FFQ	—	—	BMI 2.5 kg/m^2 higher and odds of high WC 4 times greater in highest vs. lowest ED tertile (>2.1 vs. < 1.6 kcal/g)	—
Hartline-Grafton et al., 2009 (24)	348 F; 47 y	FR	1.80	1.93	Dietary ED (kJ/g) associated with BMI (β = 0.39 kg/m^2)	—
Howarth et al., 2005 (25)	1932 M/F; 20–60 y	FR	—	—	—	Dietary ED (kJ/g) associated with BMI in women (β = 0.33 kg/m^2); no association in men[4]
Howarth et al., 2006 (92)	191,023 M/F; 45–75	FFQ	—	—	—	Dietary ED [kJ/g] associated with BMI (β ~ 1 kg/m^2) and 4–34% increased risk of overweight depending on ethnicity
Kant and Graubard, 2006 (29)	37,530 M/F; 25–74 y	FR	—	—	—	Dietary ED (kcal/g) associated with increased risk of obesity (β = 0.24)[2,3]
Kant and Graubard, 2005 (28)	13,017 M/F; ≥20 y	FR	1.88 0.93[2] 1.28[3]	1.97 0.91[2] 1.34[3]	Dietary ED (kJ/g) associated with BMI in men (β = 0.40 kg/m^2) and women (β = 0.37 kg/m^2)	Dietary ED not associated with BMI[2] Dietary ED (kJ/g) associated with BMI in men (β = 0.42 kg/m^2) and women (β = 0.47 kg/m^2)[3]
Kant et al., 2008 (27)	8265 M/F; ≥20 y	FR	—	—	Nonbreakfast dietary ED (kcal/g) associated with BMI in women (β = 0.95 kg/m^2); trend for association in men (β = 0.30 kg/m^2)	Nonbreakfast dietary ED (kcal/g) associated with BMI in women (β = 1.14 kg/m^2); trend for association in men (β = 0.41 kg/m^2)[3]
Ledikwe et al., 2006 (30)	7356 M/F; ≥20 y	FR	1.87	1.95*	BMI 0.8 kg/m^2 higher in highest vs. lowest dietary ED tertile (<1.6 vs. >2.0 kcal/g) in women; no association in men	—
Martí-Henneberg et al., 1999 (31)	649 M/F; 21–65 y	FR	—	—	—	Positive correlation between BMI and dietary ED within some age groups in men (r = 0.29–0.44); no association in women[2]
Mendoza et al., 2007 (32)	9688 M/F; ≥20 y	FR	—	—	Standardized dietary ED (kcal/g) associated with BMI in men (β = 0.37 kg/m^2) and women (β = 0.44 kg/m^2)	—

(Continued)

TABLE 2 (*Continued*)

Authors, year (reference)	Population and age	Diet assessment method	Dietary ED		Main findings	
			Normal-weight (BMI <25 kg/m^2)	Obese (BMI ≥30 kg/m^2)	Food only	With beverages
			kcal/g			
Murakami et al., 2007 (94)	1136 F; 18–22 y	FFQ	—	—	BMI 0.6 kg/m^2 higher and WC 1.7 cm greater in highest vs. lowest ED tertile (1.2 vs. 1.7 kcal/g)	—
Raynor et al., 2011 (33)	287 M/F; 18–65 y	FR	1.60 0.91[2] 1.15[3]	1.83* 1.01[2]* 1.29[3]*	Dietary ED 0.2 kcal/g lower in weight-loss maintainers compared with individuals who had never been overweight and 0.4 kcal/g lower compared with overweight individuals	Dietary ED 0.1 kcal/g lower in weight-loss maintainers compared with individuals who had never been overweight and ~0.2 kcal/g lower compared with overweight individuals[2,3]
Stookey, 2001 (35)	5783 M/F; 20–59 y	FR	2.55[3,5]	2.57[3,5,6]	—	Dietary ED (kJ/g) associated with increased odds of overweight (OR = 1.06)[3,5]
Westerterp-Plantenga et al., 1996 (36)	68 F; 33 y	FD	—	—	—	OB women consumed a greater proportion of total energy from high-ED foods (>3.6 kcal/g) (24% vs. 13%) and a lower proportion of energy from low-ED foods (<1.8 kcal/g) (24 vs. 38%) compared with NO women[2]
Yao et al., 2003 (37)	130 M/F; 35–49 y	FD	—	—	Dietary ED not associated with body fat percentage	—
Iqbal et al., 2006 (26)	1762 (862 M/900 F); 45 y	FD	0.93[2]	0.87[2]*	—	Dietary ED 0.06 kcal/g higher in nor mal-weight compared with OB men and women[2]
Saquib et al., 2008 (34)	2718 F; 53 y	FR	1.41	1.57*	Dietary ED 0.16 kcal/g lower in nor mal-weight compared to over weight and OB women	—

[1] *Significantly different from normal-weight, $P \leq 0.05$. ED, energy density; FD, food diary; FR, food recall; NO, nonobese (BMI <30 kg/m^2); OB, obese; WC, waist circumference.
[2] All foods and beverages were included.
[3] All foods and energy-containing beverages were included.
[4] All foods and beverages except water were included.
[5] Only limited energy-containing beverages were included.
[6] BMI ≥25 kg/m^2.

TABLE 3 Longitudinal associations between dietary ED calculated with and without the inclusion of beverages and weight change[1]

Authors, year (reference)	Population, age, and follow-up	Diet assessment method	Normal-weight (BMI <25 kg/m^2)		Overweight and obese (BMI ≥25 kg/m^2)	
			Food only	Beverages included	Food only	Beverages included
Bes-Rastrollo et al., 2008 (90)	50,026 F 24–44 y 8 y	FFQ	Weight gain associated with increase in dietary ED; 1.0 kg greater weight gain in quintile of largest ED in crease compared with ED decrease (1.0 vs. −0.1 kcal/g)	Weight gain associated with increase in dietary ED; 1.6 kg^2 and 0.7 kg^3 greater weight gain in quintile of largest ED increase compared with ED decrease (1.0 vs. −0.1 kcal/g) in nonobese and obese subjects	Weight gain associated with increase in dietary ED; 3.7 kg (overweight) and 3.9 kg (obese) greater weight gain in quintile of largest ED increase compared with no change in ED (~1.0 vs. ~0 kcal/g)	—
Du et al., 2009 (38)	89,432 M/F 20–78 y 6.5 y	FFQ	BL dietary ED (kcal/g) associated with Δweight (β = 0.03 kg/y)	—	BL dietary ED (kcal/g) associated with Δweight (β = −0.1 kg/y)	—
Greene et al., 2006 (39)	74 M/F 50 y 2 y	FD	—	—	BL dietary ED correlated with Δweight (r = 0.24)	—
Iqbal et al., 2006 (26)	1762 M/F 30–60 y 5 y	FD	—	BL dietary ED (kJ/g) associated with Δweight in women (β = −0.12 kg/y); no association in men[2]	—	BL dietary ED (kJ/g) associated with Δweight in overweight (β = 0.14 kg/y) and obese women (β = 0.28 kg/y); no effect modification by BMI in men[2]
Romaguera et al., 2010 (40)	48,631 M/F 50 y 5.5 y	FFQ	BL dietary ED (kcal/g) associated with Δwaist circumference for a given BMI in nonobese and obese adults (β = 0.12 cm)	—	No effect modification by BMI	—
Savage et al., 2008 (41)	168 F 24–47 y 6 y	FD	ΔWeight 0.65 kg/y greater in highest compared with lowest tertile of dietary ED (≥1.85 vs. ≤1.5 kcal/g) in nonobese and obese adults	—	No effect modification by BMI	—
Vergnaud et al., 2009 (42)	2707 M/F 35–60 y 6 y	FD	ΔWeight not associated with BL dietary ED or ΔED	ΔWeight not associated with BL dietary ED or ΔED[2]	ΔWeight 0.16 kg/y greater in highest vs. lowest tertile of BL dietary ED (1.6 vs. 1.2 kcal/g) ΔWeight 0.43 kg/y lower in tertile of largest decrease in dietary ED compared with tertile of increased dietary ED (−0.26 vs. 0.11 kcal/g)	ΔWeight not associated with BL dietary ED[2] ΔWeight 0.42 kg/y lower in tertile of largest decrease in dietary ED compared with tertile of increased dietary ED[2]

(Continued)

TABLE 3 (*Continued*)

Authors, year (reference)	Population, age, and follow-up	Diet assessment method	Normal-weight (BMI <25 kg/m^2)		Overweight and obese (BMI ≥25 kg/m^2)	
			Food only	Beverages included	Food only	Beverages included
Lindström et al., 2006 (44)[5]	500 M/F 40–64 y 3 y	FD	—	—	—	Weight loss 0.73 kg/y greater in lowest vs. highest quartile of mean dietary ED during follow-up (<0.8 vs. >1.0 kcal/g)[4]
Ledikwe et al., 2007 (43)[5]	657 M/F ≥25 y 6 mo	FR	Weight loss associated with decrease in dietary ED (kcal/g) (β = 2.2 kg); 3.5 kg greater weight loss in tertile of largest ED decrease compared with tertile of greatest ED increase (−0.9 vs. 0.2 kcal/g) in nonobese and obese subjects	—	—	—

[1] BL, baseline; ED, energy density; FD, food diary; FR, food recall; ΔED, $ED_{post} - ED_{pre}$; ΔWeight, $weight_{post} - weight_{pre}$.
[2] All foods and beverages were included.
[3] All foods and energy-containing beverages were included.
[4] All foods and beverages except water were included.
[5] Longitudinal analysis combining all groups within a randomized trial.

the energy content of the beverage consumed (115,117), suggesting that individuals do not eat less food to compensate for energy added by beverages. These findings are in contrast to clinical studies in which ED is manipulated by altering food composition (discussed below) and suggest that any influence of ED on EI may only occur when the change is within the nonbeverage components of a meal. Therefore, including beverage consumption in ED calculations may potentially bias associations between ED and weight status toward the null, and based on evidence to date, the effects of ED on EI (discussed below) are most readily seen in studies in which ED is calculated without the inclusion of beverages. An approach that was recently suggested is to analyze beverages separately or as a covariate for effects on EI (8).

Effects of ED manipulation on EI in clinical studies. Twenty-eight preload and single-meal studies were included in this review (45–65,95,97–102). These studies typically provided volume, mass, or energy-matched preloads or meals and measured ad libitum EI at subsequent meals or EI during a single ad libitum meal. Nine clinical studies ranging in duration from 1 to 3 d (66–73,103) and 14 studies ranging from 3 d to 3 wk (74–82,107,108,110–112) in duration were also identified and are summarized in **Figure 2**. The 1-d to 3-wk studies typically followed a crossover design, most implemented a washout period between interventions, and dietary ED was manipulated by altering the ED of a portion of the diet or of all foods provided to participants. Whereas preload and single-meal designs were deemed useful for evaluating effects of ED on satiation and satiety, provided-food short-term studies were deemed useful for determining the efficacy of manipulating ED to alter TDEI. The methods used to vary ED included manipulation of fat proportion, incorporation of water into food products, addition of water-rich foods, and/or use of artificial sweeteners or fat mimetics. Most, but not all, studies controlled for food palatability.

In 23 of the 28 included preload and single-meal studies, EI was less in the lowest-ED condition relative to the highest-ED condition irrespective of whether the preloads were volume-matched (i.e., energy content differed between treatments) or were isoenergetic (i.e., energy content was the same between treatments). There was no apparent difference in the responses of nonobese and obese subjects to ED manipulation. In no study did EI during the low-ED condition exceed EI during the high-ED condition.

There was notable consistency in the results from the 1- to 3-d and the 3-d to3-wk studies (Fig. 2). Lower ED interventions consistently resulted in decreased ad libitum TDEI in both nonobese and obese individuals. It is noteworthy that linear relations between the percentage difference in ED (%ΔED) and the percentage difference in TDEI (%ΔTDEI) were observed. Moreover, the magnitude of the change in TDEI was also substantial. For example, results from the 3-d to 3-wk studies indicate that a 25% reduction in dietary ED can be expected to result in ~20% reduction in TDEI

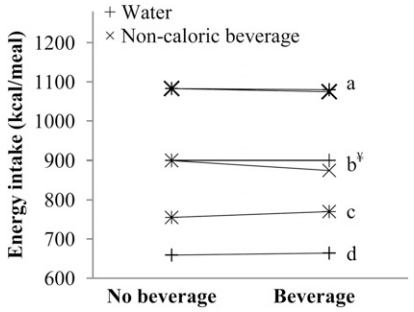

FIGURE 1 Plot of crossover studies examining the effect of reducing energy density through the addition of noncaloric beverages to meals on ad libitum energy intake. ¥Small beverage portion compared with large beverage portion. Data from references [a]115, [b]116, [c]117, and [d]56.

(Fig. 2*B*), an amount that would have a substantial impact on body weight if sustained over time (119).

A number of conclusions can be drawn on the basis of the body of evidence from the short-term clinical trials reviewed. First, in controlled laboratory environments, low-ED foods are more satiating than comparable high-ED foods. In other words, per unit of energy, lower-ED foods acutely suppress appetite to a greater extent than higher-ED foods. Second, when provided in controlled environments, the appetite-suppressing effects of lower-ED relative to higher-ED foods persist for at least 3 wk. This effect results in lower TDEI when lower-ED foods are substituted for higher-ED foods. Third, the consistent magnitude and direction of the relation between ED and TDEI (Fig. 2) suggest that these effects are independent of the methods used to alter ED.

The relevance of the short-term controlled studies reviewed herein to long-term energy balance regulation has been questioned (10,11). It has been argued that, over time, individuals learn to restrict intake of higher-ED foods, thereby mitigating any impact of ED on body weight (10,11). Providing covertly manipulated and unfamiliar foods to study participants is hypothesized to uncouple learned sensory cues from the nutritional properties of foods and thereby increase the influence of food weight and volume on EI (120). Over time, individuals may learn to compensate for reductions in ED by eating more food and/or seeking higher ED foods. The time period of the studies reviewed above may have been insufficient for individuals to learn to compensate for ED manipulation. Furthermore, in free-living environments, individuals can adjust EI by selecting from foods varying widely in ED. However, the range of the ED of foods in provided-food studies is commonly less than what is available to free-living individuals, which may prevent study participants from fully compensating for reduced dietary ED (120). As such, the controlled clinical trials reviewed herein establish the efficacy of ED manipulation for altering EI. However, these studies do not themselves address the sustainability of changes in dietary ED in free-living subjects, and therefore cannot demonstrate the effectiveness of low ED diets for weight control.

Long-term clinical studies of dietary ED reduction and body weight. A third and most important category of studies examined effects of interventions that altered dietary ED on body weight change (**Table 4**). Thirteen clinical studies of >1 mo duration were identified in which a dietary intervention was conducted, a change in body weight measured, and the intervention was determined to result in a quantifiable change in dietary ED (34,43,83,85–89,93,96,104–106).

Study designs and interventions were heterogeneous. Few interventions focused specifically on dietary ED (83,89,93). Rather, the majority of interventions were designed to decrease (e.g., dietary fat) or increase (e.g., fruits

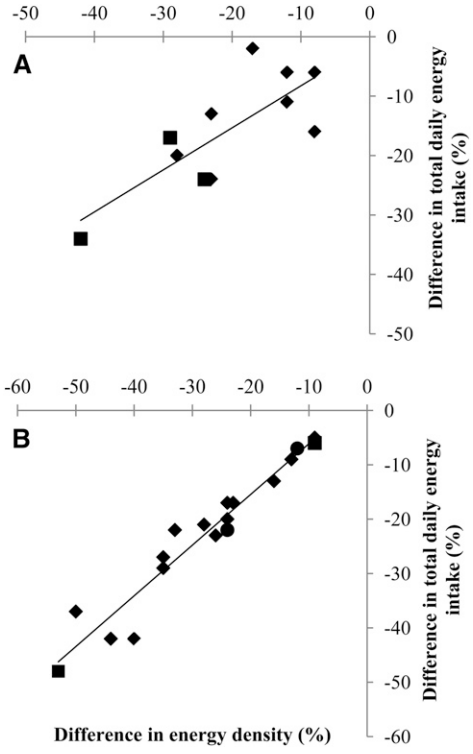

FIGURE 2 Plots of crossover studies examining the effect of reducing dietary ED on TDEI over 1–3 d (*A*) and 3 d to 3 wk (*B*). TDEI and dietary ED were extracted for each study. ΔTDEI were calculated by subtracting TDEI during $TDEI_{HED}$ from TDEI during the lowest ED condition or from TDEI during an intermediate ED condition. The percentage difference in TDEI was calculated as $\Delta TDEI/TDEI_{HED} \times 100$. Differences in dietary ED were calculated by subtracting the ED of the total diet during ED_{HED} from the ED of the total diet during the lowest or intermediate ED condition. The percentage difference in ED was calculated as $\Delta ED/ED_{HED} \times 100$. ◆, nonobese; ●, obese; ■, nonobese and obese. ED, energy density; ΔED, difference in energy density; ED_{HED}, ED of the highest ED condition; TDEI, total daily energy intake; ΔTDEI, differences in total daily energy intake; $TDEI_{HED}$, TDEI of the highest energy density condition.

TABLE 4 Randomized controlled trials of ≥1 mo in duration examining effects of reducing dietary ED on body weight change in adults[1]

Authors, year (reference)	Population	Duration	Intervention	ΔED	ΔWeight
				kcal/g	*kg*
Energy restriction recommended					
de Oliveira et al., 2008 (96)	*n* = 49 nonobese and obese F	7 wk	Random assignment to 1 of 3 groups:		
			Apple: Add 3 apples/d to usual diet	Apple: −0.2[a]	Apple: −1.3[a]
			Pear: Add 3 pears/d to usual diet	Pear: −0.3[a]	Pear: −2.2[a]
			Oat: Add 3 oat cookies/d to usual diet	Oat: +1.0[b]	Oat: −0.7[b]
Ledikwe et al., 2007 (43)	*n* = 658 nonobese and obese M/F	6 mo	Random assignment to 1 of 2 weight-loss interventions:		
			Control: One weight-loss education session	Control: −0.2[a]	Control: −1.1[a]
			RF: Extensive reduced-fat diet counseling	RF: −0.3[a]	RF: −5.1[b]
			RF+D: Extensive reduced-fat diet and DASH diet counseling	RF+D: −0.6[b]	RF+D: −6.1[b]
Raynor et al., 2012 (89)	*n* = 29 nonobese & obese M/F	3 mo	Random assignment to 1 of 2 interventions:		
			LE+LF: Low-kcal + low-fat diet education	LE+LF: −0.7	LE+LF: −7.7[a]
			LED+LE+LF: Low-ED + low-kcal + low-fat diet intervention	LED+LE+LF: −0.7*	LED+LE+LF: −5.7[b]
Rolls et al., 2005 (86)	*n* = 200 nonobese and obese M/F	12 mo	Random assignment to 1 of 4 interventions during weight loss (6 mo) and weight-loss maintenance (6 mo)	6 mo	6 mo
			Control: No additional intervention	C: −0.5[a]	C: −9.0[a]
			S1: Eat 1 serving/d of low-ED soup	S1: −0.6[b]	S1: −7.9[ab]
			S2: Eat 2 servings/d of low-ED soup	S2: −0.8[c]	S2: −7.6[ab]
			HS: Eat 2 servings/d of high-ED snacks	HS: −0.2[d]	HS: −6.1[b]
				12 mo	12 mo
				C: −0.3[a]	C: −8.1[a]
				S1: −0.5[b]	S1: −6.1[ab]
				S2: −0.6[b]	S2: −7.2[a]
				HS: −0.1[c]	HS: −4.8[b]
Weight-loss maintenance					
Due et al., 2008 (104)	*n* = 106 nonobese and obese M/F	6 mo	Random assignment to 1 of 3 weight-loss maintenance interventions:		
			Control: 35% of energy from fat	Control: 0[2,a]	Control: +3.8
			LF: 20–30% of energy from fat	LF: −0.7[b]	LF: +2.2
			HF: 35–45% of energy from fat	HF: +0.3[a]	HF: +2.5
Lowe et al., 2008 (93)	*n* = 103 nonobese and obese M/F	14 wk	Random assignment to 1 of 3 weight-loss maintenance interventions:		
			CB: received cognitive behavior therapy	CB: −0.1[3,a]	CB: −2.9%
			SM: received enhanced self-monitoring training	SM: −0.1[a]	SM: −2.4%
			SM+ED: received ESM + low-ED diet education	SM+ED: −0.4[b,*]	SM+ED: −2.7%
No recommendation to restrict energy intake					
Bray et al., 2002 (106)	*n* = 36; nonobese and obese M	9 mo	Random assignment to 1 of 3 interventions; all food provided throughout:		
			Control: 33% of energy from fat	Control: 0	Control: −3.8
			FR: Fat-reduced diet, 25% of energy from fat	FR: −0.1	FR: −1.8
			FS: Fat-substituted diet, 25% of energy from fat + 33 g/d olestra	FS: −0.2	FS: −6.3
Ello-Martin et al., 2007 (83)	*n* = 71 obese F	12 mo	Random assignment to 1 of 2 groups counseled to reduce dietary ED by:		
			LF: Reducing dietary fat intake	LF: −0.36[a]	LF: −6.4[a]
			LF+FV: Reducing dietary fat intake + increasing fruit and vegetable intake	LF+FV: −0.41[b]	LF+FV: −7.9[b]

(Continued)

TABLE 4 (*Continued*)

Authors, year (reference)	Population	Duration	Intervention	ΔED	ΔWeight
				kcal/g	*kg*
Raben et al., 2002 (85)	*n* = 41 nonobese M/F	10 wk	Random assignment to 1 of 2 groups incorporating, into habitual diets, beverages and foods containing:		
			Asp: Aspartame	Asp: −0.2[3,a]	Asp: −1.0[a]
			Suc: Sucrose	Suc: 0.0[b]	Suc: +1.6[b]
Raynor et al., 2012 (89)	*n* = 29; nonobese and obese M/F	3 mo	Random assignment to 1 of 2 interventions:		
			LED: Low-ED education (no energy restriction recommendation)	LED: −0.9	LED: −9.3[a]
			LE+LF: Low kcal + low fat diet education	LE+LF: −0.7*	LE+LF: −7.7[a]
Roy et al., 2002 (105)	*n* = 15 nonobese and obese F	10–12 wk	Random assignment to 1 of 3 interventions; all food provided throughout:		
			Control: 40% of energy from fat	Control: 0[a]	Control: −2.5[a]
			FR: Fat-reduced diet, 31% of energy from fat	FR: −0.3[b]	FR: −3.0[a]
			FS: Fat-substituted diet, 31% of energy from fat + 30 g/d olestra	FS: −0.7[b]	FS: −5.0[b]
Saquib et al., 2008 (34)	*n* = 2718 nonobese and obese F	4 y	Random assignment to 1 of 2 groups:		
			Control: Received print education materials	C: +0.05[a]	C: +1.4
			FV: Counseled to follow a high-fiber, fruit and vegetable, and low-fat diet	FV: −0.2[b]	FV: +1.8
Saris et al., 2000 (87)	*n* = 398 obese M/F	6 mo	Random assignment to 1 of 3 groups:		
			Control: Diet of typical macronutrient content	C: −0.06[a]	C: +0.8[a]
			LF+SC: Low-fat + high simple-CHO diet	LF+SC: −0.10[b]	LF+SC: −0.9[b]
			LF+CC: Low-fat + high complex-CHO diet	LF+CC: −0.18[c]	LF+CC: −1.8[b]
Westerterp- Plantenga et al., 1998 (88)	*n* = 40 nonobese M/F	6 mo	Restrained (R) and unrestrained (UR) eaters randomly assigned to:	RF-R: −0.1	RF-R: −1.5
			RF: Reduced-fat diet	RF-UR: −0.1	RF-UR: −0.2
			FF: Full-fat diet	FF-R: +0.1	FF-R: +0.2
				FF-UR: +0.1	FF-UR: +1.8

[1] CHO, carbohydrate; DASH, Dietary Approaches to Stop Hypertension; ED, energy density; Δ, change. Within a column, means without a common superscript letter are significantly different. *Significance not altered by inclusion or exclusion of energy-containing beverages in ED calculation.
[2] Baseline ED not provided.
[3] Energy-containing beverages included in ED calculation.

and vegetables) consumption of diet components that influence ED. Nine studies explicitly recommended dietary fat reduction (34,43,83,87–89,104–106), with 3 of those also emphasizing increased consumption of dietary fiber and low-ED fruits and vegetables (34,43,83). Eight studies provided food to participants: 2 used fat mimetics and provided all food to study participants (105,106), 3 provided a selection of reduced-fat or full-fat foods (87,88,104), 2 provided single low-ED or high-ED foods (86,96,104), and 1 provided sucrose-containing or artificially sweetened foods and beverages (85). Three studies included recommendations to restrict EI (43,86,104), 2 were designed to examine maintenance of weight loss (93,104), 7 made no EI recommendations (34,83,85,87,88,105,106), and 1 included groups receiving separate instructions with respect to EI (89). Self-reported dietary intake was used to calculate ED in all but the 2 studies in which all food was provided.

Study results were inconsistent, with those that implemented interventions focused specifically on reducing dietary ED reporting favorable (83,89), attenuated (89), or no (93) effects on body weight. Furthermore, findings from long-term studies appeared to be associated less with the method used to alter dietary ED and more with recommendations regarding EI restriction. When reductions in dietary ED were not coupled with recommendations to restrict EI, a modest reduction in body weight of ~2 kg more than the comparison group over 10 wk to 1 y was observed in 6 of 8 studies (83,85,87,88,105,106), although differences reached statistical significance in only 4 studies (83,87,88,105). Findings from studies in which recommendations to reduce ED were coupled with recommendations to restrict EI were less consistent (43,86,96). Ledikwe et al. (43) documented a 1.7-kJ/g (0.4-kcal/g) greater reduction in dietary ED and 5-kg greater weight loss over 6 mo in individuals receiving extensive counseling on both weight loss and the Dietary Approaches to Stop Hypertension (DASH) diet compared with a control group who received only 1 diet education session. However, when compared with a third group who received the weight-loss counseling but not the DASH diet counseling, the weight loss + DASH group did not lose more weight despite a 1.3-kJ/g (0.3-kcal/g) greater reduction in dietary ED (43). The absence of an effect suggests that the more involved counseling rather than the reduction in dietary ED underpinned differences in weight loss between the DASH and control groups. In separate studies, de Oliveira et al. (96) and Rolls et al. (86) provided evidence that adding high-ED foods to energy-restricted diets may attenuate weight loss. However, Rolls et al. failed to demonstrate an added weight-loss benefit of reducing dietary ED (by adding soups to the daily diet) relative to a control condition (86). In a pilot study, Raynor et al. (89) reported that adding low-ED diet education to recommendations to restrict energy and reduce fat intake attenuated weight loss and did not impact dietary ED. Two studies examining diet modification for maintaining weight loss did not show any effect of altering dietary ED on weight-loss maintenance (93,104).

Taken together, the long-term trials reviewed herein provide some evidence that lowering dietary ED may promote small spontaneous reductions in body weight when ad libitum consumption is recommended. The studies do not provide consistent support for the hypothesis that lower-ED diets are more effective for weight loss than EI restriction alone but suggest that adding high-ED foods to energy-restricted diets could attenuate weight loss. None of the studies suggest that reducing dietary ED promotes weight gain. However, the number of studies is small, their interventions heterogeneous, and further work in this area is needed.

Summary of the evidence.

1. Different approaches to calculating dietary ED that vary according to beverage inclusion criteria do not appear to alter the direction of reported relations between ED and body weight status. Nevertheless, given that EI is influenced by both food and beverage consumption, but that beverages appear to have little effect on appetite (121), it is recommended that future studies of ED–energy balance interrelations routinely analyze results with calculations of ED both for food-only and food + all beverages (caloric and noncaloric) so that the effects of beverages in the calculation of dietary ED can be further evaluated. Standardization of semisolid products such as milkshakes and drinkable yogurts as a food or beverage is needed to ensure consistency across studies.
2. A substantial number of short-term studies providing food and manipulating ED have been conducted. A considerable portion of this work (56–62,66–68,71,72) was produced by a single research laboratory. Nonetheless, similar studies have been completed by other groups, and when the total body of evidence is considered, a strong positive association between ED and ad libitum EI is consistently observed. This association provides evidence that lowering dietary ED is efficacious for reducing TDEI and provides indirect evidence that lower-ED diets may be efficacious for weight management. This relation appears to be independent of the method used to alter ED.
3. Prospective cohort studies suggest a positive association between ED and weight change. However, there are relatively few long-term interventions that have implemented interventions focused specifically on reducing dietary ED. Studies reporting changes in dietary ED and body weight resulting from interventions that aim to increase or decrease consumption of dietary determinants of ED have provided some evidence relevant to determining the effectiveness of reducing dietary ED for healthy weight management. In long-term studies that recommend ad

libitum consumption of low-ED diets, a modest reduction in body weight is generally observed. In long-term studies that recommend energy restriction for weight loss, no consistent benefit of consuming a lower-ED diet beyond energy restriction alone is observed. These modest effects of dietary ED reduction on body weight are surprising considering the consistent, robust effects of ED manipulation on EI observed in shorter trials. One potential interpretation of these data are that less-energy-dense diets consumed ad libitum may be effective for prevention of weight gain but may not confer advantage for weight loss. Others have suggested that, over time, individuals learn to eat smaller portions of high-ED foods or a greater total amount of food in response to dietary ED reduction, thereby mitigating long-term effects of ED manipulation on body weight (10,120). Possibly, more substantial reductions in dietary ED need to be achieved to demonstrate weight-loss benefit. However, the lack of an adequate number of long-term interventions specifically focused on ED manipulation and the inconsistencies between prospective and short-term studies relative to longer-term studies suggest that more research is needed before definitive conclusions regarding the effectiveness of low-ED diets can be made.

Future Directions

Substantial effort has been devoted to evaluating the short-term effects of ED on EI. However, a key barrier to progress in understanding the role of ED in weight regulation is the lack of data from multiple, long-term clinical trials examining the effectiveness of low-ED diet education for the prevention of weight gain and weight regain in nonobese individuals (a separate topic) and for promoting weight loss in obese and overweight individuals. Although not included in this review, a lack of long-term trials conducted in children and adolescents is also evident (13). Moreover, there are few data from long-term randomized controlled trials providing food so that the results are not confounded by inaccurate self-reports of EI and so that the efficacy of ED manipulation for long-term weight management can be definitively determined. Prioritization of funding for clinical trials that provide food is needed to understand the efficacy of ED for healthy weight management. Such studies should monitor all beverage consumption by study participants so that the question of whether beverage intakes should be included in calculations of ED can be resolved. In lieu of such trials, interventions targeting ED specifically rather than dietary components related to ED (e.g., reducing fat intake) would inform on the effectiveness of low-ED diet education for weight management. It would also be useful for all long-term dietary intervention trials examining weight loss or weight maintenance to report dietary ED. Ideally, these trials should assess both food and total beverage intake.

Studies are also needed to examine psychological and physiologic mechanisms underpinning relations between ED and EI, and how these relations change when attempting to reduce dietary ED, to provide support for or against the postulated effectiveness of low-ED diets for healthy weight management (13). In addition, studies examining barriers to adoption of low-ED diets in free-living subjects would help provide necessary information for adapting low-ED eating plans to community interventions should randomized controlled trials confirm that such diets are efficacious for healthy weight management.

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