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Increasing low-energy-dense foods and decreasing high-energy-dense foods differently influence weight loss trial outcomes

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Abstract

BACKGROUND/OBJECTIVE—Although reducing energy density (ED) enhances weight loss, it is unclear whether all dietary strategies that reduce ED are comparable, hindering effective ED guidelines for obesity treatment. This study examined how changes in number of low-energy-dense (LED) (<4.186 kJ/1.0 kcal g⁻¹) and high-energy-dense (HED) (>12.56 kJ/3.0 kcal g⁻¹) foods consumed affected dietary ED and weight loss within an 18-month weight loss trial.

METHODS—This secondary analysis examined data from participants randomized to an energy-restricted lifestyle intervention or lifestyle intervention plus limited non-nutrient dense, energy-dense food variety (*n* = 183). Number of daily LED and HED foods consumed was calculated from three, 24-h dietary recalls and anthropometrics were measured at 0, 6 and 18 months. Multivariable-adjusted generalized linear models and repeated-measures mixed linear models examined associations between 6-month changes in number of LED and HED foods and changes in ED, body mass index (BMI), and percent weight loss at 6 and 18 months.

RESULTS—Among mostly female (58%), White (92%) participants aged 51.9 years following an energy-restricted diet, increasing number of LED foods or decreasing number of HED foods consumed was associated with 6- and 18-month reductions in ED ($\beta = -0.25$ to -0.38 kJ g⁻¹ (-0.06 to -0.09 kcal g⁻¹), *P* < 0.001). Only increasing number of LED foods consumed was associated with 6- and 18-month reductions in BMI ($\beta = -0.16$ to -0.2 kg m⁻², *P* < 0.05) and 6-month reductions in percent weight loss ($\beta = -0.5\%$, *P* < 0.05). Participants consuming 2 HED foods per day and 6.6 LED foods per day experienced better weight loss outcomes at 6- and 18-month than participants only consuming 2 HED foods per day.

CONCLUSION—Despite similar reductions in ED from reducing number of HED foods or increasing number of LED foods consumed, only increasing number of LED foods related to weight loss. This provides preliminary evidence that methods used to reduce dietary ED may differentially influence weight loss trajectories. Randomized controlled trials are needed to inform ED recommendations for weight loss.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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INTRODUCTION

Obesity is a modifiable chronic disease, but novel treatment strategies that are viable and sustainable are needed.^{1,2} Diet remains the leading risk factor for obesity,³ and as such, is a compelling target. Although any strategy that promotes negative energy balance can promote weight loss,⁴ sustained energy restriction is difficult to maintain and long-term dietary adherence remains challenging. As a result, more than half of the weight lost during weight loss interventions is regained over time,^{5–7} underscoring the importance of understanding predictors of weight loss success.

A meta-analysis comparing the effectiveness of behavioral obesity treatment programs (versus control) suggested that behavioral programs, on average, resulted in 2.8 kg greater weight loss at 12 months than control, although there was substantial variability between programs.⁶ Although there were generally few differences between behavioral weight loss programs that predicted greater success,⁶ some have speculated that the dietary goals and method for achieving the dietary goal may influence an individual's ability to adhere to an energy-restricted diet over time.⁸ Energy-restricted diets often lead to reduced satiety, which can invoke feelings of deprivation, increased consumption, and may contribute to the lack of long-term success from behavioral interventions.⁹ Strategies that enhance satiety and reduce food desirability on energy-restricted diets may increase the efficacy of these interventions.

One promising strategy to decrease energy intake without compromising satiety on energy-restricted diets is to reduce dietary energy density (ED), which is defined as the amount of energy in food/diet relative to its weight (kcal g⁻¹).¹⁰ Research suggests that the homeostatic regulation of food intake is more sensitive to changes in food weight and volume than to the amount of energy consumed.^{11,12} Consequently, reducing dietary ED enhances satiety by preserving food volume on energy-restricted diets^{13,14} and may be an effective strategy to promote weight loss.^{13,15–18}

Despite the consistent, protective association between decreased dietary ED and sustained weight loss in multiple studies,^{13,19} it is unclear how to develop ED prescriptions as a strategy to improve weight management. For example, it is possible to reduce dietary ED by (1) increasing the portions/number of low-energy-dense (LED) foods consumed, (2) decreasing the portions/number of high-energy-dense (HED) foods consumed and/or (3) both, but the extent to which these strategies influence weight loss have not been compared.

From a behavioral standpoint, it is worthwhile to examine whether changing the number of LED and HED foods consumed reduces dietary ED and improves weight loss.²⁰ Having goals for the number of different LED and HED foods to consume may be easier to implement than shifting the proportions of LED and HED foods used in recipes to reduce the ED of a meal.²¹ In addition, reducing dietary ED by reducing the number of HED foods consumed may reduce the HED variety and reduce the frequency of exposure to HED foods, potentially limiting food cravings.^{8,21}

Akin to concern that promoting fruit and vegetable consumption without concurrent guidance to restrict other food groups has limited influence on energy intake and weight control,^{22,23} it is important to evaluate the effectiveness of a behaviorally aligned strategy to

reduce dietary ED during weight loss. A recent review suggests that substituting less healthy foods with healthier choices is more effective than pure promotional strategies to change nutritional intake,²⁴ but it is unclear if this applies to LED and HED foods. Therefore, to inform the development of appropriate dietary ED guidelines, the purpose of this secondary analysis of an existing weight loss trial was to determine whether the method of reducing dietary ED (that is, increasing number of LED foods, decreasing number of HED foods or both) similarly influenced total ED and short- and long-term weight loss for participants following an energy-restricted diet.

MATERIALS AND METHODS

Study population and measures

This secondary analysis examined 183 adults with overweight and obesity recruited from Knoxville, TN, USA and Providence, RI, USA to participate in an 18-month randomized controlled weight loss trial between July 2006 and August 2008. Eligible participants were between the ages of 21 and 65 years with a body mass index (BMI) between 27 and 45 kg m⁻². The original 18-month behavioral weight loss intervention randomized 204 participants to one of two groups: (1) a standardized lifestyle intervention (energy-restricted, low-fat diet; physical activity prescription, and a cognitive behavioral intervention), or (2) a standardized lifestyle intervention with a limited variety prescription (consume 2 foods that are non-nutrient-dense, energy-dense-foods (that is, chips, cookies, etc.)) for the intervention period. Daily calorie goals ranged from 5023 to 6279 kJ (1200–1500 kcal) per day depending on the participant's entry weight. Details about the original study have been previously published.²⁵ Informed consent was obtained from all participants and the Institutional Review Boards at the Miriam Hospital in Providence, RI, USA and at the University of Tennessee, Knoxville, TN, USA approved all study procedures.

Dietary data collection

Dietary data were collected over a 1-week period using three random 24-h recalls (2 weekdays and 1 weekend day) by trained interviewers over the phone using the Nutrition Data System Software for Research. Participants were provided with two-dimensional food shapes to help estimate portion size.

Exposure variables

The total number of LED and HED foods mentioned per day irrespective of serving size were estimated from the mean of three 24-h recalls. LED foods were defined as any food contributing 4.186 kJ (1 kcal) g⁻¹ and HED foods were defined as any food contributing 12.56 kJ (3 kcal) g⁻¹.¹⁷ Beverages were not included in the classifications. Items that counted as HED or LED foods included items that can be commonly consumed alone and occur naturally as one food (that is, fruits, vegetables, etc.), condiments (that is, butter, mayonnaise, etc.) and processed foods that contain ingredients, but are eaten as one food (that is, bread, crackers, etc.). Mixed dishes (that is, sandwich) were broken into their food components and the individual components that met the definition of a LED or HED food were counted toward daily intake irrespective of serving size. The same food eaten 2 times per day was counted toward the daily total each time it was consumed at a different eating

occasion (that is, if tomato was consumed as part of a sandwich at lunch, and then as part of a pasta dish at dinner, it would be counted as contributing twice to the LED goal). This technique differs from assessing the overall ED of the meal to allow for better testing of the hypothesis that the number (versus proportion) of LED and HED foods in a meal have differential implications for weight control (Supplementary Table 1). Change in number of LED or HED foods was computed by subtracting the number of LED or HED foods consumed at baseline from the number of foods consumed at 6 months; thus if participants decreased their consumption of HED foods between baseline and 6 months, the change in HED foods would be a negative value. In order to control for the variance in change related to the baseline value, we examined the residualized changes in LED and HED foods between baseline and 6 months by regressing baseline values on the 6-month change values. For example, the 6-month residualized change in number of HED foods was calculated by regressing the reported baseline number of HED foods on the change score (baseline number of HED foods subtracted from 6-month number of HED foods). Thus, the effect of the beta coefficients can be interpreted as a one-unit increase or a one-unit decrease in number of LED or HED foods on the outcomes of interest.

In addition, we further explored the influence of changing number of LED and HED foods consumed using clinically meaningful groupings. First, we examined participants who either reported decreased intake of number of HED or LED foods by >1 food, maintained a relatively stable intake (a change of -1 to 1 food), or increased intake by >1 food. Based on preliminary evidence that ED prescriptions may be effective weight loss tools,¹⁷ we explored the extent to which participants who consumed ≥ 2 HED foods and consumed \geq to the 75th percentile in LED foods (≥ 6.6 foods per day) compared with participants who did not meet at least one of those dimensions. Exact sample sizes for each group are reported in the tables.

Outcome variables

We examined the 6- and 18-month changes in total ED, changes in BMI and percent weight loss. Dietary ED calculated from the weight and energy from foods only (kcal g^{-1})¹⁹ was computed as the mean ED from all 24-h recalls at each time point (baseline, 6 and 18 months). Height (measured to the nearest millimeter) and weight (measured to the nearest 0.05 kg) were measured at 0, 6 and 18 months using a stadiometer and calibrated digital scale, respectively. BMI (kg m^{-2}) was calculated at each time point and changes in BMI were computed by subtracting baseline values from 6- and 18-month values. Percentage weight loss was computed by dividing absolute weight change between 0 and 6 months or 0 and 18 months by baseline weight and multiplying by 100.

Covariates

Demographic information including age, sex, race and education were measured via self-report questionnaire at baseline.

Statistical analyses

Among participants with complete dietary and anthropometric data at 6 ($n = 183$) and 18 ($n = 178$) months, we examined the associations between 6-month changes and number of LED

and HED foods in three ways. First, we examined the continuous associations between changes in number of LED or HED foods and our outcome variables. We then created multiple categorical variables to examine group-level differences in our outcomes of interest. For both number of LED and HED foods, we examined the effect of decreased (< -1 unit), stable (between -1 and 1 unit) or increased (>1 unit) intake over 6 months. We also examined differences in 6- and 18-month outcomes among participants who ate ≤ 2 HED foods per day, ≤ 6.6 LED foods per day (the 75th percentile of intake), neither or both to understand whether there were clinically meaningful differences among subgroups that may exist in free-living settings.

We examined the data to ensure it met model assumptions and used generalized linear models to assess whether 6-month changes in number of LED and HED foods influenced 6-month changes in dietary ED, BMI and percent weight loss and we used mixed linear models with minimum variance quadratic estimation of the covariance parameters to assess 18-month changes. We tested for interactions between our exposure variables (6-month changes in number of LED and HED foods) and intervention condition for all analyses, using a P -value of 0.05 to determine statistical significance. Tukey adjustment was used for all post-hoc comparisons between subgroups.

Initial models were adjusted for age, sex and intervention condition, and multivariable models were further adjusted for race, educational attainment, and change in either number of LED or HED foods. When both change in number of LED foods and HED foods were entered into the same model, we also tested for interactions between those two variables. All analyses were conducted using SAS v. 9.4 (Cary, NC, USA).

Code availability

Statistical coding is available upon reasonable request.

RESULTS

Participants were predominately female (58%) college-educated (68%) and White (92%) with a mean age of 51.9 ± 8.8 years. The mean BMI at baseline was 34.8 ± 4.2 kg m⁻² and energy intake was 8087 ± 3098 kJ (1932 ± 740 kcal) per day. The mean dietary and body weight changes over the 2-year period are detailed in Table 1. In general, overall ED decreased between baseline (5.23 kJ g⁻¹ or 1.25 kcal g⁻¹) and 6 months (3.43 kJ g⁻¹ or 0.82 kcal g⁻¹), as well as baseline and 18 months (3.64 kJ g⁻¹ or 0.87 kcal g⁻¹; $P < 0.0001$). Similarly, the mean number of LED foods consumed per day significantly increased by approximately one food at both time points. Conversely, the mean number of HED foods consumed only significantly decreased at 6 months (4.02 ± 0.13 versus 2.98 ± 0.14 , $P < 0.0001$); no significant decrease in the number of HED foods consumed was observed at 18 months. With the exception of percent energy from carbohydrate and protein, which increased over time, energy from dietary fats and added sugars decreased over the 2-year period.²⁵

The continuous association between 6-month changes in number of LED and HED foods with 6- and 18-month changes in dietary ED, BMI and percent weight loss are presented in

Table 2. No significant interactions between number of LED or HED foods consumed and the original intervention treatment group were detected, so treatment group was included as a covariate in all analyses. In both the base and covariate-adjusted models, increasing the number of LED foods consumed between baseline and 6 months was associated with a significant 0.07–0.08 unit decrease in dietary ED at 6 months, and a 0.06 unit decrease in dietary ED at 18 months. Similarly, decreasing the number of HED foods consumed between baseline and 6 months was significantly associated with a 0.05–0.09 decrease in dietary ED at both 6 and 18 months. Despite similar reductions in ED, only increasing the number of LED foods consumed between baseline and 6 months was significantly associated with 6- and 18-month decreases in BMI and percent weight loss (6 months only). In final models adjusting for sociodemographic covariates and the change in number of HED foods, an increase in number of LED foods was associated with a -0.18 ± 0.06 unit decrease in BMI ($P=0.007$) and a $-0.5 \text{ unit} \pm 0.18$ reduction in percent weight loss at 6 months ($P=0.007$) and a $-0.16 \text{ unit} \pm 0.07$ decrease in BMI ($P=0.03$) and a marginal $-0.39 \text{ unit} \pm 0.2$ ($P=0.05$) reduction in percent weight loss at 18 months. Changes in number of HED foods at 6 months were nonsignificantly associated with changes in BMI or percent weight loss at 6 and 18 months. We also adjusted for residualized 6-month changes in energy intake in exploratory models for BMI change and percent weight loss (data not shown) but no changes in the effect of number of LED or HED foods were observed and model fit was not improved.

Table 3 presents the 6- and 18-month changes in ED, BMI and percent weight loss stratified by participants with decreased (< -1), stable (-1 to 1), or increased (>1) number of LED and HED consumed during the 6-month weight loss phase. In all models, participants who increased number of LED food consumed had greater reductions in total ED compared with those who decreased number of LED foods consumed at 6 and 18 months ($P<0.05$). Similarly, participants who decreased the number of HED foods had significantly greater reductions in total ED at 6 and 18 months compared with participants who either increased or maintained number of HED consumed in the first 6 months. Participants who increased number of LED foods lost -3.5 ± 0.4 BMI units on average at 6 months, which was significantly greater than participants who decreased the number of LED foods (-2.4 ± 0.5). However, no significant differences in BMI among the three LED change groups were observed at 18 months and no significant differences in percent weight loss were observed at either time point. BMI and percent weight loss were similar among participants who decreased, maintained a stable intake or increased number of HED foods at both 6 and 18 months. In exploratory models, we adjusted for residualized 6-month changes in energy intake (data not shown) but no changes in the effect of number of LED or HED foods was observed and model fit was not improved at either time point.

The 6- and 18-month changes in ED, BMI and percent weight loss among participants who simultaneously decreased number of HED foods (-2 per day) and increased number of LED foods ($+6.6$ per day) or who achieved one of these goals or neither are presented in Table 4. In adjusted models, participants in Group 4 (high LED/low HED) had significantly greater 6-month changes in BMI ($-4.99 \text{ kg m}^{-2} \pm 0.61$) as compared with participants in Group 1 (low LED/high HED) ($-3.21 \text{ kg m}^{-2} \pm 0.38$) or participants in Group 2 (low LED/low HED) ($-2.82 \text{ kg m}^{-2} \pm 0.40$). Participants in Group 4 (high LED/low HED) also achieved

significantly greater percent weight loss at 6 months ($-13.5\% \pm 2.01$) compared with participants in Group 1 (low LED/high HED) ($-9.2\% \pm 1.07$) or in Group 2 (low LED/low HED) ($-8.3\% \pm 1.12$). No differences were observed between participants in Group 3 (high LED/high HED) versus Group 4 (high LED/low HED). In addition, participants in Group 4 (high LED/low HED) had a significantly greater change in 18-month BMI ($-5.00 \text{ kg m}^{-2} \pm 0.73$) as compared with participants in Group 2 (low LED/low HED) ($-2.42 \text{ kg m}^{-2} \pm 0.48$) in adjusted models. Participants Group 1 (low LED/high HED) also had a borderline lower change in BMI ($-3.26 \text{ kg m}^{-2} \pm 0.45$) than participants in Group 4 (high LED/low HED) ($P = 0.07$). Similarly percent weight loss was higher among participants at 18 months in Group 4 (high LED/low HED) ($-13.5\% \pm 2.01$) compared with participants in Group 2 (low LED/low HED) ($-7.1\% \pm 1.31$).

DISCUSSION

The present study found that the method of reducing dietary ED (that is, increasing number of LED foods versus decreasing number of HED foods) between baseline and 6 months similarly decreased overall dietary ED at 6 months and 18 months; however, only increasing number of LED foods was associated with reductions in BMI (at 6 and 18 months) and percent weight loss (at 6 months). In addition, individuals who increased number of LED foods from baseline to 6 months (>1 food) experienced significantly greater BMI reduction at 6 months than those who decreased number of LED foods. Although it is important to cautiously interpret these findings, exploratory analyses revealed that individuals who consumed both a high number of LED foods (6.6) and a low number of HED foods (2) experienced greater reductions in BMI and percent weight loss than individuals who did not meet either target (at 6 months only) or individuals who only met the HED target (at 6 and 18 months). Together, these findings suggest that increasing the number of LED foods consumed is an essential aspect of reducing dietary ED in order to produce short and long-term weight loss success.

In comparison with existing research, the present study observed a modest effect on ED reduction associated with a one-unit increase in LED foods. Consistent with the classification described by Ledikwe *et al.*,¹⁸ participants modestly reduced their overall dietary ED by 0.43 kcal g^{-1} . Thus, the 0.07 – 0.08 decrease in ED associated with consuming one additional LED food or one fewer HED food represents a 17% change in overall ED, which is meaningful for a small dietary change. Furthermore, the observation that a one-unit increase (versus decrease) in LED foods corresponded with a nearly one unit greater reduction in BMI, suggests that it is clinically meaningful.

The most prominent reason that increasing the number of LED foods consumed is helpful for short- and long-term weight loss success is through its ability to assist with appetite regulation. Increasing the number of LED foods consumed is more likely to increase food volume, which enhances satiation.^{13,26} Enhanced satiation should assist with enhanced long-term adherence to an energy-restricted diet. Beyond enhancing satiety, increasing the number of LED foods, which are often high in fiber, may increase fecal fat excretion, reduce eating speed, and favorably influence lipolysis and thermogenesis in adipose tissue, which promote weight loss.^{27–29}

Our exploratory results also suggest that both increasing the number of LED foods and decreasing the number of HED foods consumed may assist with weight management. Increasing number of LED foods with concomitant restriction of number of HED foods (either through natural displacement or conscious restriction) may assist with long-term dietary adherence to energy restriction through several mechanisms. Reducing number of HED foods consumed reduces dietary variety of these foods, which reduces food cravings, and increases habituation to these foods.²¹ Moreover, this strategy helps consumers develop food environments that align with healthier decisions, requiring them to exert less willpower to reduce dietary ED.³⁰ Thus, these goals, increasing number of LED and decreasing number of HED foods consumed, may assist with satiation, while reducing problematic factors (food cravings, environmental cues) for dietary adherence.³¹ Finally, increasing number of LED foods and decreasing number of HED foods has implications for total dietary quality. Many LED foods like fruits and vegetables improve dietary quality, which has been shown to favorably influence the gut microbiome, and improve outcomes associated with obesity and other chronic diseases.³²

Although no significant interactions between changes in number of LED and changes in number of HED foods were detected, simultaneously increasing number of LED foods and decreasing number of HED foods should theoretically lead to the largest decreases in body weight. This is consistent with what we observed and with national data, which found that individuals with normal weight versus obesity consume a higher proportion of energy from very low and low ED foods and a lower proportion of energy from HED foods.³³ Similarly, we would anticipate that participants with low LED/high HED intake and the least reduction in dietary ED would have the least favorable changes in body weight. However, our results suggest that the group with the least favorable changes in body weight were those who exclusively decreased number of HED foods, despite achieving reductions in overall dietary ED comparable to those with high LED/low HED consumption and significantly less than those with low LED/high HED consumption.

There are various possible explanations for this finding. First, it is possible that restricting number of HED foods when not accompanied by an increase in number of LED foods is associated with greater hunger, cravings and diet attrition. This is consistent with literature suggesting that restrictive diets are difficult to follow long-term and align with the findings we observe.³⁴ For example, participants with low LED/high HED food intake consumed a similar number of LED foods as participants who exclusively decreased HED foods (~4 LED foods per day at baseline and 6 months). However, participants with low LED/high HED food intake consumed a mean of approximately 4 HED foods per day at both time points, whereas participants with low LED/low HED food intake decreased HED food intake by more than >2 foods to a mean of 1.4 HED foods per day (data not shown). Similarly, energy intake decreased from 7799 to 5940 kJ (1863 to 1419 kcal) in the group with low LED/high HED foods and from 7878 to 4379 kJ (1882 to 1046 kcal) in the group with low LED/low HED foods, which may have not been sustainable (data not shown). There is some suggestion that energy restriction, particularly when extreme, induces changes in the neuroendocrine regulation of appetite, promotes marked reductions in energy expenditure, and encourages other adaptive changes in the body that promote weight regain.³⁵ Taken together, this study provides promising preliminary evidence that increasing the number of

LED foods consumed aids in achieving sustained weight loss, and that purely restrictive regimens may be less effective long term.

Some limitations of the present analysis must be noted. Importantly, because this was a secondary analysis rather than an intervention aimed at altering the number of LED and HED foods consumed, it is possible that individuals who increased LED foods differed in other important ways from individuals who did not. However, the original intervention assignments did not modify associations between the change in HED and LED foods and any study outcomes, and we adjusted for intervention condition in our analyses increasing our confidence in these conclusions. In addition, previous analysis of this data suggests that this sample was racially homogenous with similar dietary intake patterns.²⁵ Finally, dietary data were self-reported and may be prone to the underreporting bias prevalent among individuals trying to lose weight. However, because this bias was non-differential, it is likely that it attenuated observed associations.

This study also had a number of strengths. Three-day dietary recalls were used to calculate nutrient data, which more accurately reflects the variability in the different number of LED and HED foods consumed each day than fewer days of recall.^{25,36} In addition, there was limited loss to follow-up, and participant's weights were measured by trained interventionists for 18 months. The sample also had a relatively balanced gender distribution, making these results more generalizable to both male and female weight loss participants.

CONCLUSION

In conclusion, reducing dietary ED is consistently associated with reductions in total energy intake and weight loss. To date, little research has explored whether the manner in which people reduce ED differentially affects weight loss outcomes. The present study provides strong preliminary evidence from an existing weight loss trial that the manner in which people reduce total ED is important for weight loss. Increasing the number of LED foods consumed on energy-restricted diets may be necessary in order to induce weight loss despite evidence that either decreasing HED foods or increasing LED foods promotes reductions in ED. Further research using long-term randomized controlled clinical trials that are larger and more diverse examining the independent effectiveness of ED prescriptions (that is, to increase number of LED foods versus decrease number of HED foods or both) are needed to directly compare ED reduction methods and elucidate the mechanism through which reducing ED promotes weight loss.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

References

1. Flegal KM, Carroll MD, Kit BK, Ogden CL. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999–2010. *JAMA*. 2012; 307:491–497. [PubMed: 22253363]

2. NCD Risk Factor Collaboration. Trends in adult body-mass index in 200 countries from 1975 to 2014: a pooled analysis of 1698 population-based measurement studies with 19. 2 million participants. *Lancet*. 2016; 387:1377–1396. [PubMed: 27115820]
3. Marczak L, O'Rourke K, Shepard D. When and why people die in the United States, 1990–2013. *JAMA*. 2016; 315:241.
4. Sacks FM, Bray GA, Carey VJ, Smith SR, Ryan DH, Anton SD, et al. Comparison of weight-loss diets with different compositions of fat, protein, and carbohydrates. *N Engl J Med*. 2009; 360:859–873. [PubMed: 19246357]
5. Barte JC, ter Bogt NC, Bogers RP, Teixeira PJ, Blissmer B, Mori TA, et al. Maintenance of weight loss after lifestyle interventions for overweight and obesity, a systematic review. *Obes Rev*. 2010; 11:899–906. [PubMed: 20345430]
6. Hartmann-Boyce J, Johns DJ, Jebb SA, Aveyard P. Behavioural Weight Management Review Group. Effect of behavioural techniques and delivery mode on effectiveness of weight management: systematic review, meta-analysis and meta-regression. *Obes Rev*. 2014; 15:598–609. [PubMed: 24636238]
7. Loveman E, Frampton GK, Shepherd J, Picot J, Cooper K, Bryant J, et al. The clinical effectiveness and cost-effectiveness of long-term weight management schemes for adults: a systematic review. *Health Technol Assess*. 2011; 15:1–182.
8. Looney SM, Raynor HA. Behavioral lifestyle intervention in the treatment of obesity. *Health Serv Insights*. 2013; 6:15–31. [PubMed: 25114557]
9. Geiselman PJ. Control of food intake. A physiologically complex, motivated behavioral system. *Endocrinol Metab Clin North Am*. 1996; 25:815–829. [PubMed: 8977047]
10. Stubbs J, Ferres S, Horgan G. Energy density of foods: effects on energy intake. *Crit Rev Food Sci Nutr*. 2000; 40:481–515. [PubMed: 11186237]
11. Poppitt SD, Prentice AM. Energy density and its role in the control of food intake: evidence from metabolic and community studies. *Appetite*. 1996; 26:153–174. [PubMed: 8737167]
12. Rolls BJ, Bell EA. Dietary approaches to the treatment of obesity. *Med Clin North Am*. 2000; 84:401–418. [PubMed: 10793649]
13. Karl JP, Roberts SB. Energy density, energy intake, and body weight regulation in adults. *Adv Nutr*. 2014; 5:835–850. [PubMed: 25398750]
14. Rolls BJ. The relationship between dietary energy density and energy intake. *Physiol Behav*. 2009; 97:609–615. [PubMed: 19303887]
15. Ello-Martin JA, Roe LS, Ledikwe JH, Beach AM, Rolls BJ. Dietary energy density in the treatment of obesity: a year-long trial comparing 2 weight-loss diets. *Am J Clin Nutr*. 2007; 85:1465–1477. [PubMed: 17556681]
16. Lowe MR, Butryn ML, Thomas JG, Coletta M. Meal replacements, reduced energy density eating, and weight loss maintenance in primary care patients: a randomized controlled trial. *Obesity*. 2014; 22:94–100. [PubMed: 23894101]
17. Raynor HA, Looney SM, Steeves EA, Spence M, Gorin AA. The effects of an energy density prescription on diet quality and weight loss: a pilot randomized controlled trial. *J Acad Nutr Diet*. 2012; 112:1397–1402. [PubMed: 22575072]
18. Ledikwe JH, Rolls BJ, Smiciklas-Wright H, Mitchell DC, Ard JD, Champagne C, et al. Reductions in dietary energy density are associated with weight loss in overweight and obese participants in the PREMIER trial. *Am J Clin Nutr*. 2007; 85:1212–1221. [PubMed: 17490955]
19. Ledikwe JH, Blanck HM, Khan LK, Serdula MK, Seymour JD, Tohill BC, et al. Dietary energy density determined by eight calculation methods in a nationally representative United States population. *J Nutr*. 2005; 135:273–278. [PubMed: 15671225]
20. Levitsky DA, Iyer S, Pacanowski CR. Number of foods available at a meal determines the amount consumed. *Eat Behav*. 2012; 13:183–187. [PubMed: 22664394]
21. Apolzan JW, Myers CA, Champagne CM, Beyl RA, Raynor HA, Anton SA, et al. Frequency of consuming foods predicts changes in cravings for those foods during weight loss: the POUNDS lost study. *Obesity*. 2017; 25:1343–1348. [PubMed: 28618170]

22. Kaiser KA, Brown AW, Bohan Brown MM, Shikany JM, Mattes RD, Allison DB. Increased fruit and vegetable intake has no discernible effect on weight loss: a systematic review and meta-analysis. *Am J Clin Nutr.* 2014; 100:567–576. [PubMed: 24965308]
23. Mytton OT, Nnoaham K, Eyles H, Scarborough P, Ni Mhurchu C. Systematic review and meta-analysis of the effect of increased vegetable and fruit consumption on body weight and energy intake. *BMC Public Health.* 2014; 14:886. [PubMed: 25168465]
24. Grieger JA, Johnson BJ, Wycherley TP, Golley RK. Evaluation of simulation models that estimate the effect of dietary strategies on nutritional intake: a systematic review. *J Nutr.* 2017; 147:908–931. [PubMed: 28404833]
25. Raynor HA, Steeves EA, Hecht J, Fava JL, Wing RR. Limiting variety in non-nutrient-dense, energy-dense foods during a lifestyle intervention: a randomized controlled trial. *Am J Clin Nutr.* 2012; 95:1305–1314. [PubMed: 22552025]
26. Stelmach-Mardas M, Rodacki T, Dobrowolska-Iwanek J, Brzozowska A, Walkowiak J, Wojtanowska-Krosniak A, et al. Link between food energy density and body weight changes in obese adults. *Nutrients.* 2016; 8:229. [PubMed: 27104562]
27. Han SF, Jiao J, Zhang W, Xu JY, Zhang W, Chun-Ling F, et al. Lipolysis and thermogenesis in adipose tissues as new potential mechanisms for metabolic benefits of dietary fiber. *Nutrition.* 2017; 33:118–124. [PubMed: 27461561]
28. Slavin JL. Dietary fiber and body weight. *Nutrition.* 2005; 21:411–418. [PubMed: 15797686]
29. Yao M, Roberts SB. Dietary energy density and weight regulation. *Nutr Rev.* 2001; 59:247–258. [PubMed: 11518179]
30. Ammerman AS, Hartman T, DeMarco MM. Behavioral economics and the supplemental nutrition assistance program. *Am J Prev Med.* 2017; 52:S145–S150. [PubMed: 28109416]
31. Raynor HA, Niemeier HM, Wing RR. Effect of limiting snack food variety on long-term sensory-specific satiety and monotony during obesity treatment. *Eat Behav.* 2006; 7:1–14. [PubMed: 16360618]
32. Anderson JJ, Nieman DC. Diet quality-the Greeks had it right! *Nutrients.* 2016; 8:636.
33. Vernarelli, JA., Mitchell, DC., Rolls, BJ., Hartman, TJ. Dietary energy density and obesity: how consumption patterns differ by body weight status. *Eur J Nutr.* 2016. e-pub ahead of print 13 October 2016; e-pub ahead of print 13 October 2016; <https://doi.org/10.1007/s00394-016-1324-8>
34. Hill AJ. The psychology of food craving. *Proc Nutr Soc.* 2007; 66:277–285. [PubMed: 17466108]
35. Sainsbury A, Zhang L. Role of the hypothalamus in the neuroendocrine regulation of body weight and composition during energy deficit. *Obes Rev.* 2012; 13:234–257. [PubMed: 22070225]
36. Drewnowski A, Henderson SA, Driscoll A, Rolls BJ. The dietary variety score: assessing diet quality in healthy young and older adults. *J Am Diet Assoc.* 1997; 97:266–271. [PubMed: 9060943]

Energy density and adiposity measurements at 0, 6 and 18 months (mean \pm s.e.; $n=183$)

Table 1

	0 mo	6 mo	18 mo	Comparison (<i>P</i> -values)	
				0-6	0-18
Energy density kJ g ⁻¹ (kcal g ⁻¹)	5.23 \pm 0.17 (1.25 \pm 0.04)	2.61 \pm 0.13 (0.82 \pm 0.03)	3.64 \pm 0.17 (0.87 \pm 0.04)	<0.0001	<0.0001
Number of low-energy-dense foods (1.0 kcal g ⁻¹)	4.46 \pm 0.14	5.64 \pm 0.18	5.54 \pm 0.22	<0.0001	<0.0001
Number of high-energy-dense foods (3.0 kcal g ⁻¹)	4.02 \pm 0.13	2.98 \pm 0.14	3.55 \pm 0.18	<0.0001	0.072
Body mass index (kg m ⁻²)	34.8 \pm 0.29	31.1 \pm 0.29	31.6 \pm 0.34	<0.0001	<0.0001
Percent weight loss (%)	—	- 10.5 \pm 0.42	- 9.3 \pm 0.61	—	—
Total energy (kcal)	1934 \pm 51	1338 \pm 36	1467 \pm 51	<0.0001	<0.0001

Table 2

Continuous associations between 6-month changes in number of LED and HED foods and 6- and 18-month changes in dietary energy density, body mass index and percent weight loss ($n=183$)

	6-month changes		18-month changes	
	β (s.e.)	P-value	β (s.e.)	P-value
<i>Dietary energy density kJ g⁻¹ [kcal g⁻¹]</i>				
<i>LED₀₋₆</i>				
Model 1	-0.29 (0.08) [-0.07(0.02)]	<0.0001	-0.25 (0.08) [-0.06 (0.02)]	0.0003
Model 2	-0.29 (0.08) [-0.07 (0.02)]	0.0001	-0.25 (0.08) [-0.06 (0.02)]	0.0003
Model 3	-0.33 (0.08) [-0.08 (0.02)]	<0.0001	-0.25 (0.08) [-0.06 (0.01)]	<0.0001
<i>HED₀₋₆</i>				
Model 1	0.29 (0.08) [0.07 (0.02)]	0.0009	0.21 (0.08) [0.05 (0.02)]	0.004
Model 2	0.29 (0.08) [0.07 (0.02)]	0.003	0.25 (0.08) [0.06 (0.02)]	0.005
Model 3	0.38 (0.08) [0.09 (0.02)]	<0.0001	0.29 (0.08) [0.07 (0.02)]	0.0005
<i>Body mass index (kg m⁻²)</i>				
<i>LED₀₋₆</i>				
Model 1	-0.18 (0.06)	0.005	-0.16 (0.07)	0.03
Model 2	-0.17 (0.06)	0.008	-0.16 (0.07)	0.03
Model 3	-0.18 (0.06)	0.007	-0.16 (0.07)	0.03
<i>HED₀₋₆</i>				
Model 1	-0.03 (0.08)	0.77	-0.01 (0.09)	0.88
Model 2	0.0003 (0.09)	1.0	0.01 (0.09)	0.90
Model 3	0.05 (0.09)	0.59	0.03 (0.09)	0.71
<i>Percent weight loss (%)</i>				
<i>LED₀₋₆</i>				
Model 1	-0.50 (0.17)	0.005	-0.40 (0.19)	0.04
Model 2	-0.47 (0.18)	0.009	-0.39 (0.20)	0.0499
Model 3	-0.50 (0.18)	0.007	-0.39 (0.20)	0.05
<i>HED₀₋₆</i>				
Model 1	0.01 (0.24)	0.96	-0.03 (0.24)	0.89
Model 2	0.07 (0.24)	0.77	0.03 (0.25)	0.90
Model 3	0.20 (0.24)	0.41	0.09 (0.25)	0.72

Abbreviations: HED, high-energy-dense foods; LED, low-energy-dense foods. Model 1 adjusts for group, age and sex. Model 2 adjusts for the covariates in Model 1, as well as race (white versus non-white) and education (some college education, college graduate or post-graduate). Model 3 adjusts for the covariates in previous models and change in number of LED and HED foods.

Table 3
Mean (s.e.) changes in energy density, BMI and percent weight loss among participants with decreased, stable or increased LED and HED food intake ($n=183$)

6-month changes	6-month outcomes						18-month outcomes					
	LED			HED			LED			HED		
	Decreased (<-1 unit)	Stable (between -1 and 1 unit)	Increased (>1 unit)	Decreased (<-1 unit)	Stable (between -1 and 1 unit)	Increased (>1 unit)	Decreased (<-1 unit)	Stable (between -1 and 1 unit)	Increased (>1 unit)	Decreased (<-1 unit)	Stable (between -1 and 1 unit)	Increased (>1 unit)
<i>ED change (kcal g⁻¹)</i>												
Model 1	-0.24 (0.09) ^a	-0.29 (0.07) ^a	-0.62 (0.06) ^b	-0.62 (0.06) ^a	-0.29 (0.06) ^b	-0.26 (0.12) ^b	-0.24 (0.09) ^a	-0.25 (0.07) ^a	-0.59 (0.05) ^b	-0.57 (0.06) ^b	-0.29 (0.06) ^a	-0.25 (0.11) ^a
Model 2	-0.36 (0.10) ^a	-0.40 ± (0.10) ^a	-0.74 (0.10) ^b	-0.70 (0.10) ^a	-0.36 (0.10) ^b	-0.37 (0.20) ^b	-0.31 (0.12) ^a	-0.31 (0.10) ^a	-0.65 (0.10) ^b	-0.59 (0.09) ^b	-0.30 (0.09) ^a	-0.29 (0.15) ^a
Model 3	-0.21 (0.13) ^a	-0.32 ± (0.10) ^a	-0.66 (0.10) ^b	-0.65 (0.09) ^a	-0.31 (0.09) ^b	-0.22 (0.15) ^b	-0.17 (0.12) ^a	-0.24 (0.10) ^a	-0.57 (0.09) ^b	-0.55 (0.09) ^b	-0.26 (0.09) ^a	-0.16 (0.14) ^a
<i>Changes in BMI (kg m⁻²)</i>												
Model 1	-3.1 (0.4) ^a	-4.0 (0.3) ^{a,b}	-4.2 (0.2) ^b	-3.8 (0.2)	-4.0 (0.2)	-4.1 (0.5)	-2.9 (0.4)	-3.8 (0.3)	-3.9 (0.3)	-3.6 (0.3)	-3.8 (0.3)	-3.9 (0.5)
Model 2	-2.4 (0.5) ^a	-3.4 (0.4) ^{a,b}	-3.5 (0.4) ^b	-3.2 (0.4)	-3.4 (0.4)	-3.4 (0.4)	-2.2 (0.5)	-3.2 (0.4)	-3.2 (0.4)	-2.9 (0.4)	-3.1 (0.4)	-3.1 (0.7)
Model 3	-2.4 (0.5) ^a	-3.3 (0.4) ^{a,b}	-3.5 (0.4) ^b	-3.0 (0.4)	-3.2 (0.4)	-3.0 (0.6)	-2.2 (0.6)	-3.2 (0.5)	-3.2 (0.4)	-2.8 (0.4)	-3.0 (0.4)	-2.7 (0.7)
<i>Percent weight loss (%)</i>												
Model 1	-9.2 (1.0)	-11.1 (0.7)	-11.9 (0.6)	-10.9 (0.6)	-11.4 (0.7)	-11.3 (1.3)	-8.8 (1.2)	-10.8 (0.9)	-11.6 (0.7)	-10.4 (0.7)	-10.9 (0.8)	-10.8 (1.5)
Model 2	-7.4 (1.3)	-9.5 (1.1)	-9.9 (1.1)	-9.1 (1.0)	-9.5 (1.1)	-9.2 (1.6)	-7.0 (1.4)	-9.0 (1.2)	-9.2 (1.1)	-8.6 (1.1)	-9.0 (1.1)	-8.7 (1.8)
Model 3	-7.1 (1.4)	-9.3 (1.2)	-9.8 (1.1)	-8.8 (1.0)	-9.0 (1.1)	-8.3 (1.7)	-6.9 (1.5)	-9.0 (1.2)	-9.2 (1.1)	-8.2 (1.1)	-8.5 (1.1)	-7.8 (1.8)

Abbreviations: BMI, body mass index; ED, energy density; HED, high-energy-dense foods; LED, low-energy-dense foods. Changes in number of LED or HED foods were categorized into three categories: decreased (< -1), stable (-1 to 1) and increased (>1). Tukey adjustment was used for all post-hoc comparisons. Labeled means in a row under the same change period and independent variable without a common superscript differ ($P<0.05$). Because the interactions between group and LED and HED and the interactions between LED and HED were not significant, there was no interaction terms in all models. Model 1 adjusts for group, age and sex. Model 2 adjusts for the covariates in Model 1, as well as race (white versus non-white) and education (some college education, college graduate or post-graduate). Model 3 adjusts for the covariates in previous models and change in number of HED or LED foods. There are 33 participants in the decreased LED group, 60 in the stable LED group and 90 in the increased LED group. There are 85 participants in the decreased HED group, 76 in the stable HED group and 22 in the increased HED group.

Table 4

Mean (s.e.) changes in body mass index, weight, and percent weight loss for individuals with either high (> 6.6 per day) intake of LED foods, low intake of HED foods (< 2 per day), neither or both ($n=183$)

	6-month outcomes				18-month outcomes			
	Group 1 low LED/ high HED	Group 2 low LED/low HED	Group 3 high LED/ high HED	Group 4 high LED/low HED	Group 1 low LED/ high HED	Group 2 low LED/low HED	Group 3 high LED/ high HED	Group 4 high LED/low HED
<i>Energy density change (one unit of food) (kJ g⁻¹) [kcal g⁻¹]</i>								
Model 1	-1.17 (0.25) ^a	-2.64 (0.33) ^b	-1.93 (0.33) ^{ab}	-2.47 (0.59) ^{ab}	-1.26 (0.25) ^a	-2.47 (0.33) ^b	-1.63 (0.33) ^{ab}	-1.93 (0.59) ^{ab}
	[-0.28 (0.06) ^a]	[-0.63 (0.08) ^b]	[-0.46 (0.08) ^{ab}]	[-0.59 (0.14) ^{ab}]	[-0.30 (0.06) ^a]	[-0.59 (0.08) ^b]	[-0.39 (0.08) ^{ab}]	[-0.46 (0.14) ^{ab}]
Model 2	-1.51 (0.42) ^a	-2.97 (0.46) ^b	-2.26 (0.50) ^{ab}	-2.80 (0.71) ^{ab}	-1.26 (0.25) ^a	-2.47 (0.46) ^b	-1.59 (0.46) ^{ab}	-1.88 (0.68) ^{ab}
	[-0.36 (0.10) ^a]	[-0.71 (0.11) ^b]	[-0.54 (0.12) ^{ab}]	[-0.67 (0.17) ^{ab}]	[-0.30 (0.10) ^a]	[-0.59 (0.11) ^b]	[-0.38 (0.11) ^{ab}]	[-0.45 (0.16) ^{ab}]
<i>Changes in body mass index (kg m⁻²)</i>								
Model 1	-3.85 (0.23) ^a	-3.45 (0.29) ^a	-4.10 (0.31) ^{ab}	-5.58 (0.54) ^b	-3.76 (0.28) ^{ab}	-2.89 (0.34) ^a	-4.02 (0.37) ^{ab}	-5.41 (0.64) ^b
Model 2	-3.21 (0.38) ^a	-2.82 (0.40) ^a	-3.41 (0.43) ^{ab}	-4.99 (0.61) ^b	-3.26 (0.45) ^{ab}	-2.42 (0.48) ^a	-3.55 (0.52) ^{ab}	-5.00 (0.73) ^b
<i>Percent weight loss (%)</i>								
Model 1	-11.0 (0.66) ^{ab}	-10.1 (0.81) ^a	-11.4 (0.87) ^{ab}	-15.2 (1.53) ^b	-10.8 (0.76) ^{ab}	-8.71 (0.94) ^a	-11.1 (1.01) ^{ab}	-14.9 (1.77) ^b
Model 2	-9.17 (1.07) ^a	-8.26 (1.12) ^a	-9.32 (1.23) ^{ab}	-13.5 (1.72) ^b	-9.10 (1.24) ^{ab}	-7.11 (1.31) ^a	-9.35 (1.43) ^{ab}	-13.5 (2.01) ^b

Abbreviations: HED, high-energy-dense foods; LED, low-energy-dense foods. Group 1 is <6.6 LED foods and >2 HED foods per day at 6 months ($n=75$), Group 2 is <6.6 LED foods but > 2 HED foods at 6 months ($n=50$), Group 3 is > 6.6 LED foods at 6 months but <2 HED foods ($n=44$), and Group 4 is > 6.6 LED foods and < 2 HED foods at 6 months ($n=14$). Model 1 adjusts for age, sex and group. Model 2 adjusts for covariates in model 1 and education and race. Groups with different superscript letters under the same change period denote statistically significant differences in Tukey adjusted means ($P<0.05$).