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Review

Associations between dietary energy density and obesity: A systematic review and meta-analysis of observational studies

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ABSTRACT

Objective: Although many studies have shown an association between dietary energy density (DED) and obesity, there has been no systematic review and meta-analysis on this topic. Therefore, the objective of this study was to qualitatively and quantitatively review and summarize the literature on association between DED and obesity.
Methods: We searched titles, abstracts, and keywords of articles indexed in ScienceDirect, ProQuest, MEDLINE, and Google Scholar databases until January 2015 to identify eligible studies. We excluded studies that did not examine DED for whole diet and studies that included patients with cancer, pregnant women, the elderly (>60 y old), and children (<2 y old). There were no language or publication date restrictions.
Results: Of the 37 studies included in this review, most articles reported a direct association between DED and obesity. We performed a meta-analysis on 23 of these studies. In comparison with the lowest NTILE of DED, subjects in the highest NTILE of DED had significant weight gain (2.26 kg, 95% confidence interval [CI]: 1.00–3.53), greater adjusted mean body mass index (BMI) (0.50 kg/m^2, 95% CI: 0.02–0.98 for males and 0.85 kg/m^2, 95% CI: 0.51–1.19 for females), and risk of excess adiposity (odds ratio [OR]: 1.27, 95% CI: 1.04–1.55). We did not observe significant associations between DED and risk of elevated BMI (OR: 1.13, 95% CI: 1.00–1.27) and abdominal obesity (OR: 1.17, 95% CI: 0.19–7.38). We found no evidence of publication bias.
Conclusion: The present review showed that DED was directly associated with risk of excess adiposity, higher weight change, and BMI. Lower DED should be considered a prevention strategy for obesity.

Introduction

Between 1980 and 2010, the global prevalence of obesity doubled, becoming a leading global health problem [1]. The worldwide prevalence of obesity in men (body mass index [BMI] ≥30 kg/m^2) increased from 4.8% in 1980 to 9.8% in 2008. Corresponding values for women rose from 7.9% in 1980 to 13.8% in 2008 [1]. Globally, obesity and overweight are contributors to at least 2.8 million deaths each year [2]. Additionally, increased risk for various chronic diseases as a consequence of obesity has been well established [3]. Given the high prevalence of obesity in both developed and developing countries, dietary factors that are responsible for this epidemic are highly relevant.

Evaluation of the overall effect of diet is frequently preferred to assessment of individual dietary components. Among dietary factors, dietary energy density (DED), as a measure of overall diet, has been at the focus of many recent studies. The Western dietary pattern is mainly loaded by high-energy-dense foods [4] and is known as an "obesity-inducing dietary pattern" [5]. High-energy-dense diets are rich in fat and energy, but low in fiber, fruits, and vegetables [6,7]. Moreover, higher DED is inversely related to diet

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quality [8], which may encourage weight gain. Research has shown that higher DED increases the risk of metabolic syndrome as well as its components [9,10] and diabetes [11].

The link between DED and weight status has been assessed in several epidemiologic and intervention studies in different age groups. However, results have been inconsistent, and it has not been possible to establish a conclusive relation between DED and obesity. While some epidemiologic data has revealed a positive link between DED and body weight in both adults and children [12–15], others have failed to find significant associations [16–18]. Among some longitudinal studies, direct associations between weight gain and DED were observed only in subjects who were initially overweight or obese [19,20]. Conversely, some investigators have found an inverse relationship between weight gain and DED in normal-weight women, while no significant association has been observed in men [20]. Furthermore, although not a primary aim, several studies assessing the association of DED with various chronic diseases have also presented data on anthropometric measures in secondary analyses or as general participant characteristics. As far as we know, there has been one metaanalysis on the topic of DED and obesity summarizing six observational studies published before September 1, 2008 [21]. This study, by Wilks et al., limited its search to human studies in the English language. Their findings revealed no association between DED and subsequent change in adiposity in children. It is not clear if the inconsistent findings in the literature to date are attributable to differences in methods of calculating DED, in assessment of anthropometric measures, or in variation among study populations (e.g., sex, BMI, age) or physical activity levels. Our goals were to perform a systematic review and a metaanalysis to examine whether evidence from observational studies overall show a direct link between DED and obesity, and to calculate an estimate of the risk. We included both studies that evaluated this directly as a primary aim and those that provided data on these relationships either in baseline measurements or as general characteristics or covariates.

Materials and methods

Search strategy and inclusion criteria

We searched for articles that were published before January 2015 in ScienceDirect, ProQuest, MEDLINE, and Google Scholar databases. Articles were included that used *energy density*, *energy-density*, *energy dense*, *calorie density*, *caloric density*, *calorie dense*, *energy concentration*, or *calorie concentration* in the title, abstract, or keywords (a MeSH term was not defined for *Energy density*).

Exclusion criteria

We excluded studies that did not consider DED for the whole diet and that included patients with cancer or those who were pregnant, elderly (>60 y old), or children <2 y old. No language or publication-date restriction was set. Two authors (M.H.R. and F.H.) independently determined inclusion and exclusion of articles through the titles, abstracts, and, finally, the full article text. Additional articles were obtained by hand-searching references of the eligible articles already identified.

Data extraction

The first author's name, publication year, sample size, age of subjects, study design, and confounders were extracted and then tabulated. The following values were extracted from the first and the last NTILEs of DED: mean and standard deviation (SD) or standard error (SE) of obesity-related indices, the frequency of obesity and overweight, and the risk of obesity. We also extracted baseline values of obesity-related indices from cohort studies. Some studies reported data stratified by sex. When this occurred, we extracted several effect sizes from one paper.

We used scores determined by the Newcastle-Ottawa Scale [22] to run a subgroup analysis.

Statistical analyses

Reported SEs were converted to SDs. For cross-sectional studies in which the risk of obesity was not reported, we calculated odds ratios (OR) by using the frequency of obesity and overweight across NTILEs of DED. The overall risk of obesity was estimated by pooling the reported and calculated ORs. Most studies reported the following data: mean ± SD or SE of weight, BMI, waist circumference (WC), waist-to-hip ratio (WHR), and obesity risk. The analysis was performed separately for means and obesity risk. The overall risk of obesity was calculated using log-transformed ORs. Stata 11 (StataCorp., College Station, TX, USA) software was used to perform the meta-analysis. Pooled effect sizes were estimated by random effects models. Between-study heterogeneity and between-subgroup heterogeneity was evaluated by I square (I^2) and fixed-effect models, respectively. We performed sensitivity analyses to evaluate the contribution of each study on the overall effect. To investigate publication bias, we ran Begg's adjusted rank correlation test.

Results

We reviewed 5876 articles according to inclusion and exclusion criteria and, finally, 37 [8–15,19,20,23–49] studies were included in the present systematic review (Fig. 1). Characteristics of the eligible studies are reported in Table 1. Fourteen studies were conducted on subjects <18 y old [15,23–25,27,32,34,36,37,39,44–47]. Studies used either cross-sectional [8–15,23,28,29,31,33,35,37–39,42,43,45,48,49] or cohort designs [19,20,24–27,30,32,34,36,40,41,44,46,47]. The following three dietary assessment tools were frequently used in these studies: food-frequency questionnaires [8,9,11,13,26,30,39,40,42,44,49], dietary records [20,23–25,28,29,31,32,34,36,46], and dietary recalls [10,12,14,15,27,33,35,41,43,47,48]. Although 18 studies included beverages in the calculation of DED [8,9,11–13,20,23–25,28,30,31,42–44,47,49], 19 studies [10,14,15,19,26,27,29,32–35,37,38,40,41,45,46,48] calculated DED by using solid foods only. Eighteen articles reported a direct association between DED and obesity [8–10,12–15,19,20,25,26,33,34,37,38,40,41,48]. On the other hand, 15 studies did not observe any relationship between DED and obesity [23,27–32,35,39,42–47]. Wang et al. and Alexy et al. reported inverse associations between DED and BMI [11,24]. Other studies did not perform statistical analyses [49] or reported inconsistent results [36].

Our meta-analysis was performed on 23 studies [8–14,19,25,26,29,30,33–39,41,42,48,49]. We extracted 77 effect sizes for different obesity indices. The overall effect of an association between DED and mean body weight is illustrated in Figure 2. The results have been stratified by different study designs. In the cross-sectional subgroup, the mean body weight of subjects in the lowest NTILE is compared with the mean body weight of those in the highest NTILE of DED. The results show that mean body weight did not differ between these NTILEs (mean difference: 1.88 kg, 95% CI: −2.03 to 5.79; $P = 0.72$). In the cohort subgroup, change in body weight was compared between individuals with low-energy-density diets and those who consumed high-energy-density diets. The results showed that consumption of a high-energy-dense diet resulted in more weight gain (2.26 kg, 95% CI: 1.00–3.53; $P < 0.01$). Although heterogeneity was high the in the cross-sectional subgroup ($I^2 = 97.0\%$, $P < 0.01$), there was no heterogeneity in the cohort subgroup ($I^2 = 25.7\%$, $P = 0.26$) and there was significant between-subgroup heterogeneity ($P < 0.001$). We also ran a complementary subgroup analysis on cross-sectional data. As reported in Table 2, heterogeneity was significant in most cases.

The pooled effect of the association between DED and mean BMI was also calculated. As presented in Table 2, the primary subgroup analysis did not attenuate between-study heterogeneity. Therefore, further subgroup analyses were run. Both crude