Review

Dietary Factors and Eating Behaviors Affecting Diet-Induced Thermogenesis in Obese Individuals: A Systematic Review

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Summary

Obese individuals are considered to have lower energy expenditure (EE) rates than non-obese individuals. We aimed to investigate the effects of various factors related to food intake on diet-induced thermogenesis (DIT) in the EE of obese individuals. Using Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines, we reviewed relevant literature from PubMed, Embase, and Medline databases from study inception till the end of July 2019. Studies on dietary factors affecting DIT in obese individuals were included. Fifteen studies were included; these studies assessed macronutrient, single-nutrient, or supplement meal compositions, as well as dietary patterns and behaviors. The effect of obesity on DIT was not constant in each study. Differences in DIT pertained to the protein ratio being higher than the fat ratio or the carbohydrate ratio being higher than the fat ratio. High intake of calcium and vitamin D as well as high-oleic peanut supplements increased DIT in obese people. In addition, ascorbic acid intake, fatty acid saturation, and the chain length of various fatty acids had no effects on DIT. In conclusion, the findings suggest that in obese individuals, DIT is associated with various factors related to meal, nutrient, and dietary habits. However, because of the complexity of the relationship between DIT and obesity, it is difficult to determine the critical element underlying this association.

Key Words energy expenditure, food consumption, diet-induced thermogenesis, vitamin D, calcium

Obesity is a highly prevalent chronic disease worldwide (Global status report on noncommunicable diseases. https://apps.who.int/iris/handle/10665/148114).

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Abbreviations: AUC, area under the curve; BED, binge eating disorder; BMI, body mass index; BMR, basal metabolic rate; CHO, carbohydrate; CI, confidence interval; CVP, conventional peanuts; DIT, diet-induced thermogenesis; EE, energy expenditure; ER, energy restriction; FORs, fat oxidation rates; HCT, high-calcium trial; HF, high-fat; HOP, high-oleic peanuts; LCT, low-calcium trial; MIT, meal-induced thermogenesis; MUFA, monounsaturated fat; NEFA, non-esterified fatty acid; PAT, physical activity thermogenesis; PUFA, polyunsaturated fat; REE, resting energy expenditure; RQ, respiratory quotient; SFA, saturated fat; SNS, sympathetic nervous system; TDEE, total daily energy expenditure; TEF, thermic effect of food; TEM, thermic effect of a meal; UCP1, uncoupling protein 1; WL, weight loss; WM, weight maintenance.

Moreover, it is an independent risk factor for the onset of other chronic diseases such as metabolic syndrome, type 2 diabetes, dyslipidemia, hypertension, and cardiovascular disease (1). Obesity—a state of energy imbalance—is characterized by excessive fat storage because of the difference between energy intake and energy expenditure (EE) (2). Daily total EE comprises three main components: basal metabolic rate (BMR), physical activity thermogenesis (PAT), and diet-induced thermogenesis (DIT) (3). Basal EE accounts for the largest component of daily total EE, whereas PAT is the variable most dependent on personal lifestyle. Over the past few decades, the understanding of the regulation of body weight and energy balance has greatly advanced, but data regarding the effect of EE changes on positive energy balance are controversial. The presence of a low EE provides positive energy balance for obesity development owing to low resting EE (REE), active EE, DIT, or a combination of all these components (4). In addition,
the term “DIT” is represented varyingly as the thermic effect of food (TEF), meal-induced thermogenesis (MIT), and thermic effect of a meal (TEM). In this review, while DIT was used as the basis, the original word used in each reference paper was used as is.

DIT accounts for approximately 10–15% of TEE and is related to the digestion, absorption, and storage of food (5). Furthermore, DIT is known to increase to take preceding energy intake (5). Regardless, most previous studies were conducted on the general population; thus, the effect of DIT on obese individuals remains unclear. In addition, previous research suggested the theory that DIT is reduced in obesity; although this theory is plausible, discrepant findings persist in the literature (6). Therefore, this study aimed to examine the differences in the results of studies on obese adults in terms of EE (and its components); furthermore, these differences were discussed from the viewpoint of whether they may contribute to sustaining obesity.

Materials and Methods

This review was conducted according to Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines (7).

Literature search strategy. The literature search for this systematic review was conducted using three databases (PubMed; Embase; and Medline) from study inception until the end of July 2019. This search used the following keywords in free text and medical subject headings (MeSH): obesity, diet induced thermogenesis, meal induced thermogenesis, thermic effect of food, and postprandial thermogenesis. Then, a search was conducted using a combination of words such as: “obesity AND ***” ; “obesity AND diet-induced thermogenesis/DIT,” “obesity AND meal induced thermogenesis/MIT,” “obesity AND postprandial thermogenesis,” and “obesity AND thermic effect of food/TEF.” For this review, limits were applied so as to include only human studies, those in English, those conducted in adults, and those conducted from July 2009 to July 2019. The search results were exported from the database in the “*.enl” format to be included in a specific library in the reference manager Endnote version X8 (Thomson Reuters, New York, USA).

Inclusion and exclusion criteria. The titles and abstracts of all the retrieved publications were imported into EndNote, and duplicate and triplicate studies were excluded. When the available information on the titles and abstracts was not sufficient, full-text articles were read. The inclusion criteria were as follows: interventional studies, randomized control trials, studies on individuals with overweight or obesity, and studies with DIT data as outcomes.

The exclusion criteria were (1) papers that had not undergone peer review such as monographs and conference proceedings, (2) novels written in languages other than English, (3) papers that did not provide original text, (4) papers not focusing on humans, and (5) papers conducted using qualitative research methods.

Systematic review process. Title and abstracts were assessed for full-text retrieval. Full-text articles were assessed considering the inclusion and exclusion criteria by two independent reviewers (PMY and CNN) to allow for adequate data extraction. The following data were extracted: type of intervention, classification of participants, sample size, and main results.

Results

A total of 2,709 papers were identified from the three databases searched: 573 papers remained after applying the following filters: studies conducted from July 2009 to July 2019 and studies with obese adults. Next, the titles and abstracts of all papers were examined, and duplicates were removed. Twenty-eight full-text articles were reviewed (Fig. 1). Only 15 papers answered the review questions targeted in this study. Table 1 summarizes the data on the participants and study protocol of the 15 studies. Three studies focused on meal composition, such as carbohydrate-rich or protein-rich meals; seven focused on single-nutrient interventions; and five focused on dietary behaviors such as eating slowly, binge eating, meal-timing, and whether or not breakfast was skipped. Another study assessed the relationship between energy restriction (ER) and DIT among obese individuals.

Study selection process

The study selection process is summarized in Fig. 1. A total of 2,709 articles were initially identified, and their titles and abstracts were examined by two authors. Of the 2,709 studies, 573 remained after applying the following filters: studies conducted from July 2009 to July 2019, studies on humans, and studies focusing on obesity. On screening the titles and abstracts, 38 studies were selected for full-text reading. Of these studies, nine were excluded, as they were reviewed, and included 15 small sample sizes.

Effect of macronutrient meal composition on DIT

Three studies examined differences in DIT and the degree of macronutrient oxidation of meals. One (8) study included 15 lean and 15 obese women. These participants received two different isocaloric (~2,026 kJ each) protein-rich and fat-rich meals over two visits that were 1 wk apart; the meals were equal in volume and provided in random order. The degree of DIT was not significantly different between the lean and obese participants after the two meals. However, the degree of DIT associated with the protein-rich meal was significantly higher (by almost three times) than that associated with the fat-rich meal in both study groups. Furthermore, the macronutrient oxidation rates were not significantly different between the lean and obese women after the test meals. These results indicate that the level of DIT is higher after protein intake than after fat intake in both lean and obese individuals.

Another study (9) focused on 19 lean and 22 obese women after the consumption of two different isocaloric meals—one rich in carbohydrate (CHO) and one rich in fat—on two occasions 1 wk apart. This study found that the MIT level was not different between the lean and obese women after the intake of the CHO-rich or fat-rich
## Table 1. Summary of included studies that investigated outcomes on DIT in obese individuals.

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Author, year (reference)</th>
<th>Subjects</th>
<th>Group (n; BMI (kg/m²); Age</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Meal composition</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Protein-rich &amp; fat-rich meal</td>
<td>Tentolouris et al., 2016 (8)</td>
<td>lean (15): 22.9±2.1</td>
<td>leptos (15): 37.2±4.3</td>
<td>• DIT is higher after a protein-rich in comparison with a fat-rich meal. Macro-nutrient oxidation rate was no difference.</td>
</tr>
<tr>
<td>CHO-rich food</td>
<td>Gepner et al., 2016 (10)</td>
<td>obese men (140): 31.1±3.8</td>
<td>45±8</td>
<td>• Higher early thermic effect of high-CHO food, early absorption and/or sympathetic tone, associates with visceral adiposity.</td>
</tr>
<tr>
<td><strong>Nutrient/supplement</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ca &amp; vit D</td>
<td>Ping-Delfos and Soares, 2011 (11)</td>
<td>men (4), women (7): 31.0±2.4</td>
<td>54±1.2</td>
<td>• Higher Ca and vit D at a breakfast acutely increased postprandial FOR and DIT, and reduced spontaneous EI in the subsequent 24 h period.</td>
</tr>
<tr>
<td>Dietary fatty acid (SFA, MUFA, PUFA)</td>
<td>Cleveenger et al., 2015 (13)</td>
<td>women (16): 32.6±1.1</td>
<td>23.6±6.0</td>
<td>• Premenopausal obese women, high-fat rich in either MUFAs, PUFAs, or SFAs did not differentially affect TEF or postprandial substrate oxidation.</td>
</tr>
<tr>
<td>SFA chain length</td>
<td>Ngo et al., 2018 (14)</td>
<td>overweight men (13): 29.3±0.6</td>
<td>23.8±1.4</td>
<td>• Premenopausal obese women, high-fat rich in either MUFAs, PUFAs, or SFAs did not differentially affect TEF or postprandial substrate oxidation.</td>
</tr>
<tr>
<td>High-oleic peanuts</td>
<td>Duarte Moreira Alves et al., 2014 (15)</td>
<td>control (124): 29.8±3.6</td>
<td>18–50</td>
<td>• High oleic peanuts increased DIT and appetite sensation.</td>
</tr>
<tr>
<td>Protein supplements</td>
<td>Kjolbæk et al., 2017 (17)</td>
<td>whey protein and Ca (38): 28.8±3.24</td>
<td>18–60</td>
<td>• Protein supplementation does not result in improved WM success, or blood biochemistry after WL compared with the effects of normal dietary protein intake.</td>
</tr>
<tr>
<td>Milk protein (whey drink, casein drink, skim milk)</td>
<td>Lorenzen et al., 2012 (18)</td>
<td>overweight men (17): 29±4</td>
<td>31±9</td>
<td>• Milk reduced EI more than isocaloric drinks containing only whey or casein.</td>
</tr>
<tr>
<td><strong>Dietary pattern &amp; behavior</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Energy restriction</td>
<td>Antoni et al., 2016 (19)</td>
<td>males (7): 29.1±0.8</td>
<td>42±5.2</td>
<td>• Ability of substantial ER to acutely alter postprandial glucose–lipid metabolism as well as incomplete EI compensation among overweight/obese.</td>
</tr>
<tr>
<td>Breakfast</td>
<td>Chowdhury et al., 2016 (20)</td>
<td>breakfast group (11): 35.4±6.1</td>
<td>44±10</td>
<td>• No difference in weight, insulin sensitivity increase in breakfast group.</td>
</tr>
<tr>
<td>Eat slow</td>
<td>Reddy et al., 2015 (21)</td>
<td>obese (10): 4.21±1.4</td>
<td>41.8±6.7</td>
<td>• Eating a meal slowly appears to confer beneficial metabolic effects in terms of energy expenditure, elevated serum adiponectin and suppressed NEFA.</td>
</tr>
<tr>
<td>Eating mostly CHO &amp; pro at lunch or dinner</td>
<td>Alves et al., 2014 (22)</td>
<td>diet (18): 31.0±3.2</td>
<td>CT: 31.4±7.6</td>
<td>• CHO at dinner/protein at lunch within a hypocaloric balanced diet had similar effect on body composition and biochemical markers, but higher effect on DIT compared with control diet. Eating CHO at lunch/protein at dinner had a deleterious impact on glucose homeostasis.</td>
</tr>
<tr>
<td>With/without BED</td>
<td>Raymond et al., 2012 (23)</td>
<td>no disorder women (17): 34.8±6.0</td>
<td>18–45</td>
<td>• Increased EI reported by BED is due to increased food consumption, not metabolic or underreporting differences.</td>
</tr>
</tbody>
</table>

CHO, carbohydrate; DIT, diet induced thermogenesis; MIT, meal induced thermogenesis; FOR, fat oxidation rates; TEF, thermic effect of food; SFA, saturated fatty acid; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid; TG, triglyceride; WM, weight maintenance; WL, weight loss; EI, energy intake; ER, energy restriction; NEFA, non esterified fatty acid; CT, control; DCNP, carbohydrate/diurnal protein; NCDP, carbohydrate/nocturnal protein; BED, binge eating disorder.
meal but was significantly higher after the intake of the CHO-rich meal than after the intake of the fat-rich meal ($p<0.005$).

Gepner et al. (10) studied the dynamics of TEF of specific food items, such as high-carbohydrate foods, including walnuts or whole-grain bread, and showed that the relationship between TEF and visceral adiposity was limited. They measured the fasting and 40-min postprandial REE, which reflects the TEF, prior to the utilization (oxidation) of the ingested food (as suggested by a lack of change in the respiratory quotient [RQ]). The results were as follows: (1) the intake of a high-carbohydrate food item (whole-grain bread) induced a greater TEF response in individuals with an increased absolute visceral fat content and (2) the presence of a higher percentage of visceral fat and higher body mass index (BMI) was associated with a lower REE per kg. These findings suggest that visceral adiposity is associated with the more efficient processing of food in the first ingestion phase and/or with a greater early sympathetic response.

Fig. 1. PRISMA flow chart in this review.
Diet-Induced Thermogenesis and Obesity

to carbohydrate-rich foods.

Effect of single nutrients or supplements on the DIT of obese individuals

Seven studies compared differences in the DIT level associated with single-nutrient non-meals such as protein supplements, calcium and vitamin D, ascorbic acid, high-oleic peanuts, and various fatty acids.

The findings of one study (11) investigating the mechanism associating dietary calcium and vitamin D with body weight regulation require confirmation. Eleven participants aged 54±1.2 y with a BMI of 31±2.4 kg/m² were within-subject randomized, and their values were compared after a low-calcium trial (LCT) and a high-calcium trial (HCT) with an isocaloric meal. DIT, fat oxidation rates (FORs), serum leptin levels, and subjective feelings of hunger/satiety were measured at the time of fasting and hourly over 8 h. HCT resulted in a lower suppression of ΔFOR (p=0.02) and a significantly greater DIT (p=0.01). Furthermore, Δleptin following HCT but not LCT was negatively related to 24-h fat intake (r=-0.81, p=0.016). In conclusion, higher calcium and vitamin D intakes increased postprandial FORs and DIT over two successive meals.

Newsom et al. (12) studied the effect of the TEF of ascorbic acid on β-adrenergic stimulation in sedentary overweight and obese adults. Ascorbic acid supplementation prevented increases in the plasma concentrations of oxidized low-density lipoprotein in the postprandial state (p=0.04); however, this did not affect the REE (1,668±107 kcal/d and 1,684±84 kcal/d; p=0.91) or the area under the TEF response curve (33.4±2.4 kcal and 30.5±3.6 kcal; p=0.52) (control and ascorbic acid, respectively). Furthermore, acute ascorbic acid administration had no effect on the respiratory exchange ratio, heart rate, or arterial blood pressure in the pre- and post-absorptive states (all p>0.64).

In addition to the abovementioned articles on vitamins and minerals, three studies focused on the effects of various dietary fatty acids. One study (13) demonstrated the effects of three high-fat (HF) meals with different dietary fatty acid compositions on TEM. Sixteen obese premenopausal women (18–39 y) consumed isocaloric HF meals (70% of energy from fat) rich in either saturated fat (SFA), monounsaturated fat (MUFA), or polyunsaturated fat (PUFA) for the assessment of substrate oxidation and EE for the determination of TEM. A significant time effect on both substrate oxidation and EE was observed (p<0.05). No treatment difference was observed in terms of postprandial substrate utilization or TEM between the test meals.

In addition, Nguo et al. (14) compared the acute metabolic response of two HF meals rich in SFA differing in chain length and one rich in MUFA in overweight healthy men aged 18–40 y with a BMI of 27–35 kg/m² and waist circumference ≥94 cm (Caucasians) or BMI of 25–30 kg/m² and waist circumference ≥90 cm (Asian and Indians). There were no significant differences in MIT, fat oxidation degree, triglyceride level, or subjective appetite responses between the two SFA (short/medium versus long) meals or between the MUFA and SFA meals.

Another study (15) explored the effects of the acute ingestion of high-oleic and conventional peanuts on appetite, food intake, and energy metabolism in 71 overweight and obese men. Participants were assigned to the following groups: control (CT, n=24); conventional peanuts (CVP, n=23); and high-oleic peanuts (HOP, n=24). Participants consumed 56 g of peanuts (CVP and HOP) or control biscuits (CT) after an overnight fast. The postprandial EE and DIT level were significantly higher in the HOP group than the CVP group. The degree of substrate oxidation did not differ between the groups. The intake of high-oleic peanuts contributed to a higher DIT, higher sensation of fullness, and an incomplete compensation for energy intake compared to conventional peanuts; this finding may aid in the formulation of weight-loss dietary interventions. Few additional studies have evaluated the effects of peanuts on energy expenditure. In one such study, Alper and Mattes designed a crossover trial with 15 lean subjects who consumed conventional peanuts (~505 kcal/d). This study found an incremental increase of 11% (p>0.01) in REE without changing the DIT (p>0.05) after 19 wk (16).

Two studies focused on the effect of protein intake on the DIT of obese people. One study (17) investigated the effect of the intake of protein supplements from either whey or without calcium or soy on weight maintenance (WM) success after weight loss (WL) compared to that of a control. Two-hundred-and-twenty participants aged 18–60 y with a BMI ranging from 27.6 to 40.4 kg/m² were included. The study was initiated with an 8-wk WL period followed by a 24-wk WM period. During the WM period, participants consumed the following isocaloric supplements (45–48 g/d): whey and calcium (whey +), and whey, soy, or maltodextrin (control). A total of 151 participants completed the WM period. The intake of the control and three protein supplements did not result in differences in weight regain, fat mass regain, or improvements in lean body mass during the WM period. Compared to the control group, protein supplementation resulted in a higher DIT (~30 kJ/2.5 h) and REE (243 kJ/d).

Another study (18) compared the effect of whey, casein, and milk, consumed as part of a mixed meal, on satiety, DIT, and substrate oxidation. Seventeen slightly overweight (29±4 kg/m²) men were served three isocaloric test meals containing either a whey drink, casein drink, or skim milk for a subsequent ad libitum lunch. We observed no significant effect on the subjective appetite sensation, but the energy intake at lunch was lower after the milk test meal than after the casein (9%; p=0.0260) and whey (9%; p=0.0258) test meals. The postprandial lipid oxidation level was significantly higher after the casein test meal intake than after the whey test meal intake (p=0.0147), after adjustment for baseline values. There was no significant difference in the effect on EE, protein oxidation, or carbohydrate oxidation.
Association between dietary behavior and DIT in obese subjects

Five studies focused on dietary patterns and behavior, while some examined the effect of meal compositions or single-nutrient meals on DIT in obese people. The subject of the articles was ER, breakfast, eating slowly, eating more carbohydrates and protein for breakfast or lunch, and presence of binge eating disorder (BED).

One study (19) was conducted on 10 overweight or obese participants who completed three dietary interventions in a random order with a 1-wk washout period: isocaloric intake, partial 75% ER and total 100% ER. There was a delay in the time for the glucose level to peak after total ER only ($p=0.024$). Both total and partial ER interventions produced a decrease in the postprandial triacylglycerol responses ($−75$ and $−59\%$, respectively; both $p<0.05$) and 3-d energy intake deficits of about 30% (both $p=0.015$). The resting and MIT degrees were not significantly affected by either ER intervention. This study showed the ability of substantial ER to acutely alter postprandial glucose and lipid metabolism as well as incomplete energy-intake compensation among overweight/obese participants.

Chowdhury et al. (20) conducted a randomized controlled trial to examine the causal links between breakfast habits and the components of energy balance in free-living obese adults aged 21–60 y, comprising 15 women and 9 men. Their energy balance (resting metabolic rate, PAT, DIT, and energy intake [EI]) under free-living conditions was measured, and they were randomly allocated to eat breakfast daily ($≥700$ kcal before $1100$ h) or undergo extended fasting (0 kcal until 1200 h) for 6 wk; their baseline and follow-up health marker measures (e.g., hematology/adipose biopsies) were recorded. Interestingly, the intake of breakfast resulted in a greater PAT degree in the morning than when individuals fasted during that period (difference: $188$ kcal/d; $95\%$ confidence interval [CI]: 40, 335), but had no consistent effect on 24-h PAT (difference: $272$ kcal/d; $95\%$ CI: 225, 798). The energy intake was not significantly greater with breakfast intake than fasting. Daily breakfast intake led to greater physical activity levels during the morning, whereas morning fasting resulted in partial dietary compensation (i.e., greater energy intake) later in the day in obese adults. There were no differences between the groups in terms of weight change and most health outcomes, but insulin sensitivity was increased with breakfast intake relative to fasting.

Another research study (21) explored the postprandial effects of meal duration on human metabolism and appetite following a standard meal eaten slowly over 40 min (D40) and the same meal eaten quickly over 10 min (D10) on a different day. All participants ($n=10$) were obese (BMI $>30$ kg/m$^2$) premenopausal, white, Caucasian women, with normoglycemia. The postprandial TEF (over 240 min) was significantly greater for D40 than for D10 (mean [standard error of the mean]: 80.9 kcal [3.8] vs 29.9 kcal [3.4]; $10.6\%$ vs $3.9\%$, $p=0.006$; area under the curve [AUC] 71.7 kcal/h vs. 22.4 kcal/h, $p=0.02$). The postprandial plasma non-esterified fatty acid (NEFA) levels were significantly lower, and adiponectin levels were significantly higher for D40 than for D10. The other postprandial analyses and appetite measures were equivalent. Therefore, this study concluded that eating slowly was associated with an enhanced TEF, elevated serum adiponectin levels, and suppressed NEFA levels in obese women.

Yet another study (22) evaluated the effects of two dietary patterns in which carbohydrates and proteins were eaten mostly at lunch or dinner on body weight and composition, energy metabolism, and biochemical markers in overweight/obese men. Fifty-eight men ($30.0\pm 7.4$ y; $30.8\pm 2.4$ kg/m$^2$) followed a covert hypocaloric balanced diet ($−10\%$ of daily energy requirements) for 8 wk. Participants were randomly categorized into three groups: the control diet (CT); diurnal carbohydrate/nocturnal protein (DCNP); and nocturnal carbohydrate/diurnal protein (NCDP) groups. The main analyzed outcomes were weight loss, body composition, DIT, and glucose/lipid profile. After adjustment, the NCDP group presented a significantly higher DIT level and EE after lunch, compared to the DCNP group, but after dinner, there were no differences between the groups.

Finally, Raymond et al. (23) determined the differences in energy intake or EE to distinguish overweight/obese women with and without BED. Seventeen overweight/obese women with BED and 17 overweight/obese controls completed random 24-h dietary recall interviews and had their total daily EE (TDEE) assessed using the doubly labeled water method. Participants underwent two baseline dual energy X-ray absorptiometry scans and had BMR and TEF. The results indicated no between-group differences with respect to TDEE, BMR, and TEF. There was no difference in the prevalence of BED between the non-binge day intake and control groups (2.233 and 2.140 kcal, respectively).

Discussion

Since the early 1900s, obesity has been known to be caused by an imbalance between energy intake and expenditure. This view is a prerequisite for the study of the causes of obesity today, and over the past few decades, it has contributed to a dramatic increase in the understanding of the physiology that promotes the adjustment of energy balance. Decades of research have shown progress in this area, but results concerning different degrees of DIT on total EEs in obese adults are inconsistent. It is still unclear which components of DIT change in obese individuals.

The aim of this review was to appraise published studies that dietary factors and eating behaviors affecting DIT in obese individuals. Here, potential differences that could contribute to sustaining obesity were discussed. We searched for these systematic review articles and examined the results for differences in the DIT of obese individuals, based on the composition of energy nutrients in meals (meal composition), single nutrients or supplements, and dietary patterns or behaviors.
Many studies reported that the main determinant of DIT is the energy content of food, followed by the protein fraction of food. The thermic effect of alcohol is similar to that of protein (6). Therefore, the main determinants of DIT are the energy content and protein fraction of the diet. Protein plays a key role in body weight regulation through satiety related to DIT (24).

The extent to which the metabolic rate increases after meal ingestion is macronutrient-dependent. In mixed-meal studies, convincing evidence indicates that meals with a higher protein content potentiate greater thermogenesis than do carbohydrate and fat-rich diets (9). This could be attributed to the increased EE associated with intestinal food-stuff absorption, metabolic effects of hepatic metabolism, the body having an inadequate storage capacity for protein, energy required for protein synthesis, high adenosine triphosphate cost of peptide synthesis, high energy cost of urea, creatinine and other nitrogenous end-products, and gluconeogenesis. The results of research, including this review, show that there is no difference between obese and non-obese individuals, although there is a difference in the degree of DIT due to meal composition, as high-protein or high-carbohydrate than equivalent fat.

De Jonge and Bray (25) evaluated 49 studies that compared DIT in subjects who were obese with those who were lean. Of the 29 studies measuring DIT in subjects who were age-matched and sufficiently obese, 22 reported a reduced DIT in obesity. The reported reason for this is that the reduction of DIT in obesity is related to the degree of insulin resistance, which may be affected by a low level of sympathetic activity.

Seven papers (11–15, 17, 18) compared differences in the DIT of obese individuals in terms of specific components such as ascorbic acid, minerals such as calcium and vitamin D, the type of fatty acids, and the length of the chains or protein supplements. In the case of ascorbic acid (13), its acute administration was not effective in increasing the TEF and had no effect on the respiratory exchange ratio, heart rate, or arterial blood pressure. However, this powerful antioxidant prevented increases in the plasma concentration of oxidized low-density lipoprotein in the postprandial state \( p = 0.04 \). Therefore, it may still have a clinical application based on its ability to preserve postprandial endothelial function in at-risk populations. Dairy calcium and vitamin D intake (12) acutely increased whole-body fat oxidation and DIT in a sequential meal and reduced spontaneous energy intake in the subsequent 24-h period. Additionally, high-oleic peanut intake contributes to a greater increment in DIT and cumulative EE (AUC) compared to that of conventional peanut intake. The fatty acid profile associated with high-oleic peanut intake seems to be the main factor responsible for these effects not being attributed to protein content, as both high-oleic and conventional peanuts have similar amounts of protein (30.0% and 29.1%, respectively). In a study of premenopausal obese women (13), HF meals rich in either MUFAs, PUEAs, or SFAs did not differentially affect TEM or postprandial substrate oxidation. In another similar study, the SF A chain length and fatty acid saturation status has no acute differential effect on MIT, fat oxidation, triglyceride level, or subjective appetite responses in healthy overweight men.

There is accumulating evidence that a high-protein diet intake (17) may promote weight loss and prevent weight (re)gain better than a low-protein diet intake, and that this may be because a high-protein diet could induce higher DIT levels, increase satiety, and decrease hunger levels. However, the effect of different protein sources was limited. Lorenzen et al. (18) showed that whey, casein, and milk intakes had similar effects on acute appetite sensation, but ad libitum energy intake at the subsequent lunch was significantly decreased by milk intake compared to casein and whey beverage intakes. Thus, these results do not support the theory that whey is more satiating than casein and has no significant effect on postprandial EE. However, a small-but-significant increased lipid oxidation level was observed after casein intake compared to whey intake. Similarly, Kjolbaek et al. (17) showed that protein supplement intake (whey and calcium, whey, soy, or maltodextrin) did not improve body composition over a 24-wk WM period despite resulting in higher DIT and EE compared to that of the control.

Five papers examined differences in the DIT of obese people according to eating behavior or dietary pattern. A study focusing on meal times and the type of macronutrient (8–10) demonstrated that for 8 wk, the negative effect of eating carbohydrates mostly at lunch and protein mostly at dinner on glucose homeostasis. In addition, eating carbohydrates mostly at dinner and protein mostly at lunch within a hypocaloric balanced diet had a similar effect on body composition and biochemical markers, but a higher effect on DIT compared to that of the control diet.

A study (19) that examined the effect of total and partial ER found that MIT was not significantly affected by either ER intervention. There were no differences between the groups that had breakfast or fasted in the morning in terms of DIT. This suggests that there are no differences in overall energy intake or physical activity between obese individuals who fast during the morning or those who consume breakfast daily for 6 wk. In addition, the resting metabolic rate and blood lipid profiles were not differently affected by eating breakfast or fasting, although there was some evidence on skipping breakfast reducing insulin sensitivity. Another interesting study (21) demonstrated that eating slowly resulted in an enhanced TEF, elevated serum adiponectin level, and suppressed NEFA level. This was thought to confer a beneficial metabolic effect in terms of EE, metabolic profile, and lipid control. Finally, there were no differences in TEF between overweight/obese women with and without BED.

The issue of whether obesity is associated with a lower DIT level is controversial. Several studies have shown that DIT values of obese people (26, 27) are low, but other studies demonstrated no differences in DIT values between obese and non-obese people (8, 9). The energy
balance of humans is affected by several factors, not just energy intake and EE. In the case of EE measurement, the DIT level can affect the content of the meal as well as various activities occurring simultaneously (non-exercise activity thermogenesis and exercise). EE has been measured using both indirect and direct methods so far. This makes the comparison of findings and estimation and reporting of DIT difficult. Energy intake is a confounder in the determination of DIT because obesity may lead to a prolonged absorptive state. Many variables involved in DIT, such as digestion, absorption, food preference, and metabolic factors, vary among individuals; notwithstanding, even if they are assumed to be the same, they may affect the absorptive state. In particular, it is difficult to maintain the same percentage of CHO: protein: fat according to the appropriate dietary reference intake in the regular meals of all individuals.

Additionally, many of these factors can affect the DIT response. Meanwhile, the molecular mechanisms are not fully understood. High-fat feeding sensitizes animals to the thermogenic effects of norepinephrine (28, 29), strongly suggesting that thermogenic fat is a critical regulator of DIT. An obvious molecular candidate for driving DIT is uncoupling protein 1 (UCP1), the principal effector protein controlling cold-mediated, non-shivering thermogenesis in fat (30). However, UCP1-knockout mice do not exhibit suppression of metabolic rate in the context of DIT, as the increase in oxygen consumption between wild-type and knockout mice is identical in response to acute high-fat feeding (31). Therefore, it was recently reported that UCP1-independent mechanisms play a role in DIT in obese individuals (31, 32). Another mechanism may be due to the low sympathetic activity. The sympathetic nervous system (SNS) is important in virtually all components of daily EE, including RMR, physical activity, and DIT (33). SNS activation results in the release of norepinephrine and the subsequent stimulation of adrenergic receptors (34). Sympathetic underactivity and blunted sympathetic responses such as these could contribute to deficient thermogenesis, positive energy balance, and weight gain (35). However, a recent report noted that in interpreting differences in SNS activity between obese and non-obese subjects, it is not always clear whether the observed differences are causal or secondary to the changed nutritional state. It is also important to distinguish between established steady-state alterations and acute changes during weight loss or weight gain (36). Overall, the relationship between SNS activity and obesity is complex, with evidence of low SNS activity in some, but not all, studies (37).

In conclusion, the present findings suggest that in obese individuals, DIT is associated with various factors related to meal, nutrient, and dietary habits. Furthermore, it is difficult to determine the critical element because of the associated complexity. Energy intake and EE are not separate entities. In daily life, people do not occur energy-expending activity separately during 3–5 h after meals as they do under experimental conditions. Irrespective of the type of activity, physical activities differ in terms of EE in all people. In addition, it is also important to measure the sum of the DIT values generated by the continuous intake of more than 2–3 meals daily, rather than by adjusting the ratio of the energy nutrients in the composition of a fractional meal or a supplement. Therefore, it is important to measure DIT using a metabolic chamber capable of measuring in freely ambulatory individuals for at least 24 h.

Disclosure of state of COI

The authors declare that they have no competing interests.

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Author contribution

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