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Diet induced thermogenesis, older and newer data with emphasis on obesity and diabetes mellitus - A narrative review

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ABSTRACT

Obesity is a major public health problem with a prevalence increasing at an alarming rate worldwide. There is an urgent need for efficient approaches to weight management. Diet induced thermogenesis (DIT) is the process by which the body increases its energy expenditure in response to a meal. It is estimated to account for approximately 10 % of total energy expenditure and is considered a potentially modifiable component of energy expenditure. The palatability of food, meal's composition in macronutrients, the circadian rhythm and sleep, as well as individual's characteristics such as age, the presence of obesity or diabetes mellitus, and the proportion of physical activity are the main factors that affect DIT. However, studies examining DIT are mostly characterized by small sample size and the methodology varies considerably between studies. It seems that even today there is a lot of contradiction between the relative studies. Inspite of that, future research might lead to the modification of DIT in order to achieve some weight loss in obese people.

1. Introduction

The global prevalence of obesity has increased dramatically over the last two decades and nearly a third of the world population is now classified as overweight or obese [1,2]. Obesity is associated with type 2 diabetes mellitus (T2DM), cardiovascular disease, cancer, and gastrointestinal, respiratory, and musculoskeletal complications; hence, efficient prevention and treatment methods are urgently required [3–6].

Obesity progresses gradually as calorie intake surpasses expenditure, emphasizing the potential clinical significance of minor changes in energy expenditure over time [7]. The total energy expenditure comprises various components. Basal metabolism, the energy utilized at rest, comprises approximately 60 % of the total daily energy expenditure [7,8]. The thermic effect of food, also known as dietary-induced thermogenesis (DIT), contributes to approximately 10 % of total energy expenditure by increasing metabolism following a meal. This includes the energy expended in processing and storing food, as well as the metabolic effects of nutrient intake. Intentional activities such as sports-related exercises contribute between 0 % and 10 % of the total energy expenditure. Non-exercise activity thermogenesis, which includes daily activities, fidgeting and maintaining posture, accounts for approximately the remaining 20 % of total energy expenditure [7,9].

Research and clinical evidence indicates the potential of modifying the thermic effect of food as a strategy for weight loss. This narrative review aimed to describe the factors that affect the thermic effect of food and highlight potential areas for further research.

2. Measuring methods of the thermic effect of food

There are several methods for measuring the thermic effects of food [7,8]. Each method has different precision and cost-effectiveness [7,8]. Energy expenditure can be measured using indirect calorimetry, direct calorimetry, and several non-calorimetric techniques [8,10]. Indirect calorimetry is the most widely used technique and involves the measurement of oxygen consumption and carbon dioxide production [7,8]. Direct calorimetry quantifies body heat loss through an isothermal system, a heat sink (adiabatic) system, or a convection system [7,8]. This method is accurate, extremely expensive to build and run, and requiring at least one full-time technician [7,8]. Non-calorimetric methods estimate energy expenditure by extrapolating variables related to energy expenditure [7,8]. The methods for measuring energy expenditure and the thermic effect of food have been thoroughly described in a previous review by Levine [8] and are beyond the scope of this review. Herein,

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Abbreviations

T2DM	type 2 diabetes mellitus
DIT	dietary-induced thermogenesis
PUFA	high-polysaturated fatty acids
MUFA	monosaturated fatty acids
SAFA	saturated fatty acids, RMR: resting metabolic rate
BMR	basal metabolic rate
GIT	glucose induced thermogenesis
BAT	brown adipose tissue
^{18}F-FDG-PET/CT	^{18}F-fluoro-2-deoxy$_D$ -glucose-positron emission tomography
GDM	gestational diabetes mellitus
PCOS	polycystic ovary syndrome
HOMA-IR	Homeostatic Model Assessment for Insulin Resistance

when the measuring method of DIT is not mentioned, this is indirect calorimetry; otherwise, it is specified.

3. Factors that affect DIT

3.1. Palatability, chewing and the cephalic phase of DIT

Diet-induced thermogenesis can be divided into two phases. The first phase is the cephalic phase, which corresponds to the energy lost as heat while the subject sees, smells, and tastes food, and is estimated to be 30–53 % of the total DIT [11,12]. The second phase is gastrointestinal, which refers to thermogenesis produced from digestion, absorption, metabolism, and storage of meal components [11,12].

Food palatability could be expected to influence postprandial thermogenesis, at least in its early phase. In a small study with eight healthy, non-obese participants, the DIT 90 min postprandially was significantly higher after tasty than after non-palatable meal ($p < 0.05$). In addition, the increase in insulin and norepinephrine levels was significantly higher after palatable meal, indicating possible participation of the sympathetic nervous system in meal-induced thermogenesis [13]. In accordance with these findings, Hashkes et al. reported a significant difference in 90min DIT between meals of different palatability in non-obese subjects. However, the differences were ameliorated in people with obesity [14].

Studies examining whether adding sweet flavor to food affects DIT have conflicting findings. One study examined whether the sweet flavor obtained with a low-energy sweetener (aspartame) or with sucrose affects differently DIT [15]. The study showed that adding aspartame or sucrose did not affect 2 h DIT. However, DIT was higher with sucrose 30–60min postprandially, compared to maltodextrins and maltodextrins plus aspartame. This finding may reflect the effect of taste on the cephalic phase of thermogenesis [15]. On the other hand, Weststrate et al. compared two isocaloric meals, one normal liquid meal and one with kinin added in order to be unpleasant; no difference was observed in 2 h postprandial thermogenesis [16]. In another study, the use of spices or mustard did not seem to alter the thermic effects of food [17]. Similarly, extended measurement of DIT up to 6 h postprandially did not reveal significant differences between tasty and control meals, despite that respiratory quotient and glucose levels were higher after palatable food intake [18].

Of interest, Tittelbach and Mattes gave 16 healthy volunteers a test meal in capsule to avoid triggering of taste, followed by palatable foods, in which subjects were allowed to chew but not to shallow; no difference was observed in DIT after oral exposure to palatable food compared with no oral exposure [19]. Opposing were the results of a previous study, in which four groups were examined: first simply eating, second seeing, smelling and tasting without swallowing, third seeing and smelling without ingesting, and fourth tube feeding of the tested meal to the participants [20]. DIT was measured up to 110 min postprandially and was significantly higher with ordinary meal digestion than with tube feeding (12 % and 5.7 % of resting values, respectively, $p < 0.01$). In the case of viewing/smelling/tasting and viewing/smelling, the change in energy expenditure postprandially was +3.2 % and-2.6 %, respectively, both lower than meal eating ($p < 0.01$), as expected [20]. On the top of the above findings, Hamada et al. in a cross over trial, compared drinking rapidly with either tasting and drinking or chewing/tasting/-drinking the same liquid meal; DIT 90 min post ingestion was significantly higher with chewing, followed by tasting without chewing and finally drinking alone [chewing trial DIT 7.4 ± 0.7 (3.6–10.7)kcal, taste trial DIT 5.6 ± 0.5 (3.2–7.8) kcal and drinking trial DIT 3.4 ± 0.4 (1.5–5.9) kcal, all $p < 0.05$] [21]. This is in accordance with the results of other studies that also reported higher DIT with oral feeding than tube feeding [11,22], as well as with prolonged chewing than fast eating of the same meal [23,24].

Overall, even though orosensory stimulation and chewing may play an important role in DIT further studies are needed.

3.2. Meal' s composition

3.2.1. Energy content

The amount of energy in the ingested food may have an important role in the postprandial rise in the metabolic rate. In one study, the effect of meal composition and energy content on DIT was examined in sixteen adult, female subjects without obesity [25]. DIT 5 h postprandially correlated positively with calories included in the digested meal ($p < 0.001$), but not with the composition in macronutrients [25]. Two more studies supported the above findings [26,27], while others were inconclusive [28,29]. In a recent review and meta-analysis, Quatela et al. used a mixed model meta-regression analysis and estimated that DIT rise by 1.1 kJ/h per 100 kJ increase in energy content of the meal consumed ($p < 0.001$) [30].

3.2.2. Composition in macronutrients

Regarding the macronutrient content in food, it is established that the thermic effect of proteins is greater (20–30 %) than that of carbohydrates (5–10 %), while the lowest values of DIT are observed in fatty foods (0–3%) [31]. In a study by Aita et al. DIT measured 2 h postprandially was 6.44 ± 2.01 % of the total meal' s energy for a protein, 3.49 ± 2.00 % for a carbohydrate rich and 2.32 ± 0.90 % for a fatty meal [32]. Similarly, another study examined 15 lean and 15 individuals with obesity and observed higher DIT up to 3 h postprandially after a protein rich compared to fat rich food in both groups (mean difference [Δ] = 205.6 ± 78.9 kJ/h, $P < 0.001$ for lean and [Δ] = 169.8 ± 114.3 kJ/h, $P < 0.001$ for obese individuals) [33]. In agreement were the results of other studies, there was however heterogeneity in meal composition, as well as in the duration of metabolic rate measurements [34–40].

On the other hand, Riggs et al. found higher thermogenic effect of high protein/high fat versus high protein/low fat meals among normal weight subjects, attributed to higher content in protein in the first meal (37 g versus 30.8 g of protein respectively), but the differences were ameliorated in over or under-weighted participants [41]. Other authors have also failed to report significant differences between foods with different compositions [42–44].

Few studies have assessed the thermic effects of alcohol consumption. According to Raben et al. the DIT of alcohol was 27 %, which was significantly higher compared with protein, carbohydrate, and fat meal ingestion (9 %, 8.3 %, 7.1 %, and 7.1 % energy intake, respectively; all $p < 0.01$) [43]. Protein was also followed by a higher DIT (17 %) than fat or carbohydrates, but the differences were not significant [43]. Suter et al. also observed that alcohol ingestion, while in fasting state, resulted in a rise in metabolic rate of 7 % over baseline, which was equal to 17 % of energy consumed as ethanol [45]. However, in a previous study,

alcohol-induced thermogenesis was estimated at only 15 % of the energy content of alcohol; hence, authors concluded that alcohol could act as a carbohydrate because of its thermic effects [46].

3.2.3. The fatty acids content

The structure of fatty acids contained in food may also affect DIT. Kasai et al. reported that medium chain triglycerides have a higher thermogenetic effect than long chain ones [47]. Similar were the results of another group of investigators, with DIT 6 h postprandially being 47.9 ± 4.6 kcal for medium vs. 34.4 ± 3.6 kcal for long chain fatty acids ($p < 0.05$) [48]. Suzuki et al. also reported greater postprandial thermogenesis in foods containing medium-chain fatty acids than in those containing long-chain fatty acids, regardless of the food structure (liquid or solid) [49]. These observations are in line with previous studies [50,51]. Bendixen et al. also observed that chemically structured fats induced greater increases in DIT than conventional fats [52].

In addition, in a study by Casas-Agustench et al., DIT was assessed up to 5 h postprandially after three isocaloric meals with the same composition in macronutrients but different degrees of fatty acid saturation [53]. DIT was 28 % higher after a meal reach in polysaturated fatty acids (PUFA) and 23 % after a meal with a high content in monosaturated fatty acids (MUFA) compared with a meal rich in saturated fatty acids (SAFA) (DIT 12.3 %, 11.8 % and 9.6 % above resting metabolic rate (RMR) respectively, all $p < 0.05$) [53]. On the other hand, in another study no significant difference was observed between MUFA and SAFA thermogenic effects; however, in subgroup analysis, higher DIT was observed with MUFA than with SAFA-rich breakfast in participants with high waist circumference (≥99 cm) ($p = 0.043$) [54]. When SAFA containing only short- and medium-chain fatty acids was compared to SAFA with long-chain fatty acids alone and MUFA, no difference was observed among the three groups [55]. Two other studies with female participants also failed to detect any difference between MUFA-, PUFA-, and SAFA-containing meals [56,57].

In summary, although the results of current research are inconclusive, according to the above, we could assume that the greater the meal's content is in energy, proteins, medium chain fatty acids, PUFA, MUFA and perhaps alcohol, the higher is expected the DIT.

3.3. The circadian rhythm, sleep and DIT

Several studies have shown that circadian variations during the day may affect DIT. Romon et al. reported higher DIT in the morning compared with afternoon ($p = 0.04$), as well as night ($p = 0.02$), for the same meal (DIT 15.9 ± 1.6, 13.5 ± 1.8, 10.9 ± 2.2 % of meal energy, respectively), along with a non-significant trend favoring afternoon postprandial thermogenesis over nocturnal one ($p = 0.06$) [58]. Similarly, a recent trial, also reported higher DIT in the morning than in the evening ($p = 0.03$) [59]. In both of the aforementioned studies, the fasting metabolic rate did not differ significantly between the different time points examined [58,59]. Therefore, the diurnal variation in post-meal thermogenesis cannot be attributed to variations in fasting metabolic rate; it is more likely that slower gastric emptying [60] accompanied by increased insulin resistance [61] in the evening, as well as higher epinephrine and norepinephrine levels in the morning [62], play a key role in the decline of DIT throughout the day.

However, other studies failed to show a circadian rhythm of postprandial thermogenesis [63–65]. Further studies are needed to examine whether time of food consumption affects energy expenditure, and further assess whether avoid eating late in the day could help in weight reduction.

In addition, disturbances in normal sleep break circadian rhythms and may influence DIT. Indeed, in a study, acute sleep deprivation was followed by a reduction of 20 % ($p < 0.001$) in post-breakfast thermogenesis in healthy, normal-weight individuals and a 5 % reduction in RMR [66]. Interestingly, free food intake did not differ between days of sleep and sleep deprivation [66]. This seems to be an effort by the body to replace the energy lost during wakefulness. Two other studies on the other hand showed no significant effect of sleep deprivation on DIT; however, sleep was reduced and not fully missed [67,68].

3.4. Individual' s characteristics

Apart from meal composition and eating habits, subjects' characteristics, such as age, as well as the presence of certain diseases, seem to alter the thermic effect of food.

3.4.1. Age

Several studies have proposed that DIT declines with age. In a retrospective study from the Mayo Clinic, data from 136 older (age 60–88 years) and 141 younger (age 18–35 years) adults were analyzed. A significantly lower DIT was observed in the older group (6.4 % vs. 7.3 % for young participants, $p = 0.02$), and this difference was not diminished after adjustment for body composition or blood insulin levels [69]. Although the basal metabolic rate (BMR) did not initially differ between the two groups, after adjusting for fat-free mass, BMR was significantly lower in older participants than in young participants ($p = 0.01$) [69]. Both basal and postprandial metabolic rates were lower among older adults than among middle-aged and young adults, according to Thörne and Wahren [70]. A more striking decline of 48%–50 % in postmeal thermogenesis was also reported with age in two smaller trials [71,72]. In line were as well, the findings of Goley et al. and Bloesch et al. that both used oral glucose load to assess postprandial thermogenesis in young and older individuals [73,74]. Nevertheless, in Bloesch's study, the reported changes in both BMR and DIT with age disappeared after considering free fat mass [73]. Likewise, Visser et al. observed lower meal-induced thermogenesis in older men than in younger participants, but the difference was not significant after adjusting for body composition [75]. In contrast, Vaughan et al. reported no differences at all [76].

The reasons for the attenuation of the thermic effect of food with advanced age are not very clear, and the hypothesis that the observed differences in DIT between young and older individuals could be attributed to an impairment of the postprandial sympathetic nervous system response with aging, although supported by some data [71], was not confirmed by others [72]. Alterations in body composition, with a reduction in lean mass as a person gets older, may partly explain the decline in DIT [73,75], but there is objecting evidence that reports differences independent of free fat mass [69]. Finally, changes in humoral responses, such as higher insulin levels measured in older people due to insulin resistance, have been proposed as a possible link between aging and meal-induced thermogenesis reduction [77], but this is also not well established [69].

3.4.2. Body weight

Several studies have shown that obesity is associated with reduced postmeal thermogenesis In a large study from Northern Europe that included 701 subjects with obesity and 113 subjects without obesity showed that DIT 3 h after a fat-rich meal was negatively associated with body weight [78]. Similarly, another study that consisted of 10 participants with obesity class III and 10 lean matched controls, the DIT measured up to 180 min after ingestion of a mixed meal was significantly lower in the obese group ($p < 0.001$) [79]. Other studies have also reported diminished postprandial thermogenesis after mixed meal ingestion in subjects with obesity compared with lean subjects [29,80,81]. Swaminathan et al. likewise observed lower thermogenic response to fatty and mixed meals with obesity; however differences were ameliorated when high carbohydrate or protein meals were tested [82]. In addition Tounian et al. also reported a similar thermic effect of sucrose in children with obesity or normal weight; however, in subgroup analysis, children with both obesity and family history of obesity had lower DIT values than children with a negative family history [83]. In addition, no difference in the thermic effect of a carbohydrate-rich meal

was found between overweight and lean subjects; on the contrary, DIT correlated positively with body fat mass [84]. According to the above findings, obesity may interfere only with the processing of fat- or protein-rich meals. However some other trials, which examined high protein, high fat, or mixed meals, also failed to reveal a significant reduction in the thermic effect of food among participants with obesity compared with normal-weight subjects [33,85,86]. On the other hand, some authors have shown that even formerly obese individuals may experience lower post-meal thermogenesis than never-obese individuals [87,88]; nevertheless, other studies have reported amelioration of these differences in DIT with weight loss [79,80,89]. In another study, very lean healthy participants experienced lower postprandial thermogenesis than normal-weight controls, while subjects with anorexia nervosa had a similar DIT with controls [90]. Reverse results were observed with regard to RMR [90]. Taken together, it seems that individuals in extremes of body weight show impairment in mechanisms that regulate energy expenditure. Therefore, efforts have been made to clarify the mechanisms underlying the possible relationship between obesity and DIT. A blurred postprandial response of sympathetic nervous system [88,91], as well as insulin resistance [92,93] among individuals with obesity have been proposed as key factors, but these observations were not confirmed by other authors [84,94]. Interestingly, Hibi et al. examined 21 men with normal body mass index but different body composition in brown adipose tissue (BAT), as assessed by ^{18}F-fluoro-2-deoxy$_D$-glucose-positron emission tomography (^{18}F-FDG-PET/CT) [95]. Participants underwent 24- hour whole room indirect calorimetry under normal temperature conditions and DIT was expressed as a percentage of 24-h total energy expenditure; BAT-positive participants had higher DIT than BAT-negative ones (9.7 ± 2.5 % and 6.5 ± 4.0 % respectively, $P = 0.03$), as well as lower respiratory quotient ($p = 0.03$) [95]. Authors concluded that BAT plays an important role in meal induced thermogenesis even in warm temperatures, and BAT activation could therefore have therapeutic implications for patients with obesity. In summary, the available evidence regarding the impact of obesity on DIT is inconclusive. Methodological variations in study design may be a reason for this, and a former review has highlighted this issue [96]. Furthermore, in cases where a reverse relationship truly exists, impairment in meal-induced thermogenesis could be either a result or a cause of obesity [97]. Therefore, further investigation is required to examine causality.

3.4.3. Physical activity

Physically active individuals appear to experience higher postprandial thermogenesis than inactive individuals. Jones et al. reported an almost 25 % higher DIT in men with regular aerobic exercise than sedentary matched controls ($p < 0.05$) [72]. Similar was the impact of resistance exercise on DIT [98]. A slightly wider difference was also observed in a previous study; DIT was 45 % higher in the young active group and 31 % higher in the older active group than in the corresponding age-matched sedentary group ($p < 0.01$) [99].

In contrast, in a trial by Burke et al. although a trend for higher DIT was observed in young, highly trained women, the results did not reach statistical significance [100]. Similarly, in a study by Bowden and McMurray, training status was not found to affect DIT overall or after a fat-rich meal; however, post hoc analysis revealed that trained participants had a greater peak response when a carbohydrate-rich meal was used [44]. Some authors failed to document any increase in DIT with habitual exercise [101–103], whereas others paradoxically reported lower food-induced thermogenesis in well-trained subjects [104,105].

The effect of acute or short-term exercise on postprandial thermogenesis has also been addressed by some authors; a 12 week course of cycling led to an increase in meal-induced thermogenesis in lean participants, as well as in those with obesity or diabetes [106]. However, in another study no difference was observed in DIT measured in resting conditions and after 1 h of exercise [107].

3.4.4. Diabetes mellitus and insulin resistance

People with diabetes mellitus as well as those predisposed to diabetes mellitus may have impaired DIT. According to a small trial, subjects with obesity with T2DM or insulin resistance had lower GIT than obese, insulin-sensitive controls; however the results were reverse in terms of resting and total metabolic rate [108]. Nevertheless, insulin resistance is more frequent in obese individuals; therefore, body weight may be a confounding factor. To examine this hypothesis, Gumbiner et al. enrolled 9 non-obese subjects with insulin independent T2DM and 16 non-obese healthy controls and participants underwent hyperinsulinemic clamps; GIT was significantly lower in patients with T2DM than the control group [109]. Lower thermogenesis has also been reported 3 h after a mixed meal among subjects with both obesity and mild T2DM compared with participants with obese without diabetes, while the lean group presented the highest DIT of all [87]. In a study by Segal et al. insulin resistance, assessed by euglycemic-hyperinsulinemic clamp as well, was associated with impaired DIT, independently of the presence of obesity [94]. In line were the results of Camastra et al. who analyzed data from 322 studies with participants without diabetes; they reported that the GIT correlated positively with insulin sensitivity, irrespectively of the body mass index [110]. However, the waist-to-hip ratio correlated negatively with the GIT [110].

Several studies have focused on women with previous gestational diabetes mellitus (GDM), a condition known to increase the risk of T2DM [111]. Robinson et al. reported significantly lower thermogenesis 2 h after a mixed meal among women with a history of GDM, compared with matched controls ($p < 0.05$) [112]. This difference could be at least partly attributed to the higher insulin resistance observed in participants with GDM [112]. Nevertheless, in a more recent trial, only early (0–30 min) postprandial thermogenesis was impaired in women with previous GDM, and this defect was in line with the thermogenic response to adrenaline [113]. The total DIT was similar between women with and without a GDM history, in accordance with similar insulin sensitivity [113]. In a study by Kousta et al. which included 29 women with a GDM history and 37 controls, the overall postprandial response did not differ between the two groups; however, a delay in meal-induced thermogenesis was observed among women with previous GDM [114]. This was in parallel with a delay in postprandial insulin and noradrenaline responses [114]. According to the above findings therefore, it is most likely that women with GDM experience a different pattern of DIT, with a more dilated pick than matched controls.

Insulin resistance plays a pivotal role in the pathogenesis of polycystic ovary syndrome (PCOS) [115]. Robinson et al. examined 14 women with PCOS and 14 controls; half of the participants in each group were obese [116]. Insulin resistance was measured using an intravenous insulin tolerance test [116]. Investigators observed that thermogenesis 2 h after a mixed meal was reduced in the PCOS group, for both lean and obese subgroups, compared with healthy controls [116]. This impairment in DIT has been attributed to reduced insulin sensitivity in women with PCOS [116]. The RMR did not differ between groups [116]. Segal et al. on the other hand, reported a slightly higher DIT in subjects with PCOS compared with controls, difference did not however reach statistical significance [117]. Unfortunately, the most recent and larger-sample studies on the effect of PCOS on DIT are lacking.

Frailty, on the other hand, has also been associated with chronic inflammation and insulin resistance and could therefore affect postprandial thermogenesis [118]. This hypothesis was examined by Goulet et al.; no difference was observed in the DIT between frail women and matched controls, although postprandial insulin resistance was impaired in the frail group. However, this is to our knowledge the only available study in this field and no conclusions can be drawn [119].

Taken together, individuals in the spectrum of insulin resistance, either with overt diabetes mellitus or at risk for diabetes, seem to have diminished meal-induced thermogenesis compared with insulin-sensitive controls. In addition, a small, heterogeneous group of studies has addressed the effect of antidiabetic agents on DIT. Treatment with

acarbose, an alpha-glucosidase inhibitor, in 12 patients with poorly controlled T2DM resulted in an increase in 120 min post-meal thermogenesis; this was accompanied by a change in the DIT pattern due to a delay in carbohydrate absorption by acarbose [120]. In addition, in a study of 19 healthy, non-diabetic, normal weight men, intranasal insulin administration significantly increased DIT, probably by acting on brain signaling pathways [121]. On the other hand, the achievement of normoglycemia with a 72 h intravenous insulin infusion in 10 patients with T2DM, although improved carbohydrate oxidation, had no effect on DIT after a mixed meal, compared with the post-infusion state [122]. No difference was observed in DIT between insulin detemir and NPH in patients with type 1 diabetes mellitus [123]. Notably, glucagon-like peptide 1 and glucagon-like peptide 1 receptor agonists had a negative effect on meal-induced thermogenesis according to a recent review [124]. In summary, the effects of different categories of antidiabetic drugs on postprandial thermogenesis are heterogeneous.

3.4.5. Thyroid disease

Both hypothyroidism and hyperthyroidism have been linked to increased insulin resistance [125,126]. We could therefore hypothesize that thyroid disease may influence thermogenesis induced by food as well. Few studies however have addressed the impact of thyroid dysfunction on glucose induced thermogenesis, while studies about the effect of thyroid diseases on post-mixed meal thermogenesis are meager. Kozacz et al. reported lower GIT in patients with hypothyroidism compared to healthy controls (GIT 19.68 $\pm$ 3.90 versus 55.40 $\pm$ 7.32 kJ, respectively, $p < 0.0001$), which was accompanied by significantly lower RMR, as well as higher catecholamine levels in the hypothyroid group [127]. Nevertheless, insulin resistance, as estimated by Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) did not differ between groups [127]. Randin et al. observed higher RMR values among patients with Grave's disease, GIT was however similar between subjects with hyperthyroidism and controls [128]. Likewise, Acheson et al. found no difference in the thermic effect of a liquid meal between pre and post exogenous thyroid hormone administration in healthy volunteers, although RMR was increased with treatment induced hyperthyroidism [129].

4. Conclusion

DIT contributes to approximately 10 % of total energy expenditure by increasing metabolism following a meal. Studies examining DIT are limited, have small sample sizes, and methodology varies considerably between studies. Emerging data suggest that the main factors that affect DIT are palatability of food, meal' s composition in macronutrients, the circadian rhythm and sleep, as well as individual' s characteristics such as age, the presence of obesity or diabetes mellitus, and the proportion of physical activity. However the existing data are in many cases conflicting. This is most probably due to different methodology among studies (composition of test meals, duration, different types and degrees of obesity and other factors).

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CRediT authorship contribution statement

Evangelia Tzeravini: Writing – review & editing, Data curation. **Anastasios Tentolouris:** Writing – review & editing, Data curation. **Alexander Kokkinos:** Writing – review & editing, Supervision. **Nikolaos Tentolouris:** Writing – review & editing, Supervision, Methodology. **Nikolaos Katsilambros:** Writing – review & editing, Supervision.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] Hruby A, Hu FB. The epidemiology of obesity: a big picture. Pharmacoeconomics 2015;33:673–89. https://doi.org/10.1007/s40273-014-0243-x.
[2] Chooi YC, Ding C, Magkos F. The epidemiology of obesity. Metabolism 2019;92:6–10. https://doi.org/10.1016/j.metabol.2018.09.005.
[3] Ansari S, Haboubi H, Haboubi N. Adult obesity complications: challenges and clinical impact. Ther Adv Endocrinol Metab 2020;11:2042018820934955. https://doi.org/10.1177/2042018820934955.
[4] Bliddal H, Leeds AR, Christensen R. Osteoarthritis, obesity and weight loss: evidence, hypotheses and horizons - a scoping review. Obes Rev 2014;15:578–86. https://doi.org/10.1111/obr.12173.
[5] Pati S, Irfan W, Jameel A, Ahmed S, Shahid RK. Obesity and cancer: a current overview of epidemiology, pathogenesis, outcomes, and management. Cancers 2023;15. https://doi.org/10.3390/cancers15020485.
[6] Tentolouris A, Ntanasis-Stathopoulos I, Terpos E. Obesity and multiple myeloma: emerging mechanisms and perspectives. Semin Cancer Biol 2023;92:45–60. https://doi.org/10.1016/j.semcancer.2023.04.003.
[7] Calcagno M, Kahleova H, Alwarith J, Burgess NN, Flores RA, Busta ML, et al. The thermic effect of food: a review. J Am Coll Nutr 2019;38:547–51. https://doi.org/10.1080/07315724.2018.1552544.
[8] Levine JA. Measurement of energy expenditure. Publ Health Nutr 2005;8:1123–32. https://doi.org/10.1079/phn2005800.
[9] Levine JA. Non-exercise activity thermogenesis (NEAT). Best Pract Res Clin Endocrinol Metabol 2002;16:679–702. https://doi.org/10.1053/beem.2002.0227.
[10] Cunningham JJ. Calculation of energy expenditure from indirect calorimetry: assessment of the Weir equation. Nutrition 1990;6:222–3. https://www.ncbi.nlm.nih.gov/pubmed/2136001.
[11] LeBlanc J, Cabanac M, Samson P. Reduced postprandial heat production with gavage as compared with meal feeding in human subjects. Am J Physiol 1984;246:E95–101. https://doi.org/10.1152/ajpendo.1984.246.1.E95.
[12] Garrel DR, de Jonge L. Intragastric vs oral feeding: effect on the thermogenic response to feeding in lean and obese subjects. Am J Clin Nutr 1994;59:971–4. https://doi.org/10.1093/ajcn/59.5.971.
[13] LeBlanc J, Brondel L. Role of palatability on meal-induced thermogenesis in human subjects. Am J Physiol 1985;248:E333–6. https://doi.org/10.1152/ajpendo.1985.248.3.E333.
[14] Hashkes PJ, Gartside PS, Blondheim SH. Effect of food palatability on early (cephalic) phase of diet-induced thermogenesis in nonobese and obese man. Int J Obes Relat Metab Disord 1997;21:608–13. https://doi.org/10.1038/sj.ijo.0800449.
[15] Prat-Larquemin L, Oppert JM, Bellisle F, Guy-Grand B. Sweet taste of aspartame and sucrose: effects on diet-induced thermogenesis. Appetite 2000;34:245–51. https://doi.org/10.1006/appe.1999.0310.
[16] Weststrate JA, Dopheide T, Robroch L, Deurenberg P, Hautvast JG. Does variation in palatability affect the postprandial response in energy expenditure? Appetite 1990;15:209–19. https://doi.org/10.1016/0195-6663(90)90021-y.
[17] Gregersen NT, Belza A, Jensen MG, Ritz C, Bitz C, Hels O, et al. Acute effects of mustard, horseradish, black pepper and ginger on energy expenditure, appetite, ad libitum energy intake and energy balance in human subjects. Br J Nutr 2013;109:556–63. https://doi.org/10.1017/S0007114512001201.
[18] Sawaya AL, Fuss PJ, Dallal GE, Tsay R, McCrory MA, Young V, et al. Meal palatability, substrate oxidation and blood glucose in young and older men. Physiol Behav 2001;72:5–12. https://doi.org/10.1016/s0031-9384(00)00292-4.
[19] Tittelbach TJ, Mattes RD. Effect of orosensory stimulation on postprandial thermogenesis in humans. Physiol Behav 2002;75:71–81. https://doi.org/10.1016/s0031-9384(01)00644-8.
[20] Brondel L, Fricker J, Fantino M. Postprandial thermogenesis and alimentary sensory stimulation in human subjects. Int J Obes Relat Metab Disord 1999;23:34–40. https://doi.org/10.1038/sj.ijo.0800754.
[21] Hamada Y, Hayashi N. Chewing increases postprandial diet-induced thermogenesis. Sci Rep 2021;11:23714. https://doi.org/10.1038/s41598-021-03109-x.
[22] De Jonge L, Agoues I, Garrel DR. Decreased thermogenic response to food with intragastric vs. oral feeding. Am J Physiol 1991;260:E238–42. https://doi.org/10.1152/ajpendo.1991.260.2.E238.
[23] Hamada Y, Miyaji A, Hayashi N. Effect of postprandial gum chewing on diet-induced thermogenesis. Obesity 2016;24:878–85. https://doi.org/10.1002/oby.21421.
[24] Hamada Y, Kashima H, Hayashi N. The number of chews and meal duration affect diet-induced thermogenesis and splanchnic circulation. Obesity 2014;22:E62–9. https://doi.org/10.1002/oby.20715.
[25] Kinabo JL, Durnin JV. Thermic effect of food in man: effect of meal composition, and energy content. Br J Nutr 1990;64:37–44. https://doi.org/10.1079/bjn19900007.
[26] Martin A, Normand S, Sothier M, Peyrat J, Louche-Pelissier C, Laville M. Is advice for breakfast consumption justified? Results from a short-term dietary and

metabolic experiment in young healthy men. Br J Nutr 2000;84:337–44. https://doi.org/10.1017/s0007114500001616.
[27] Hill JO, Heymsfield SB, McMannus 3rd C, DiGirolamo M. Meal size and thermic response to food in male subjects as a function of maximum aerobic capacity. Metabolism 1984;33:743–9. https://doi.org/10.1016/0026-0495(84)90216-6.
[28] Bennett C, Reed GW, Peters JC, Abumrad NN, Sun M, Hill JO. Short-term effects of dietary-fat ingestion on energy expenditure and nutrient balance. Am J Clin Nutr 1992;55:1071–7. https://doi.org/10.1093/ajcn/55.6.1071.
[29] Segal KR, Edano A, Blando L, Pi-Sunyer FX. Comparison of thermic effects of constant and relative caloric loads in lean and obese men. Am J Clin Nutr 1990;51:14–21. https://doi.org/10.1093/ajcn/51.1.14.
[30] Quatela A, Callister R, Patterson A, MacDonald-Wicks L. The energy content and composition of meals consumed after an overnight fast and their effects on diet induced thermogenesis: a systematic review, meta-analyses and meta-regressions. Nutrients 2016;8. https://doi.org/10.3390/nu8110670.
[31] Tappy L. Thermic effect of food and sympathetic nervous system activity in humans. Reprod Nutr Dev 1996;36:391–7. https://doi.org/10.1051/rnd:19960405.
[32] Aita S, Matsushita M, Yoneshiro T, Hatano T, Kameya T, Ohkubo I, et al. Brown fat-associated postprandial thermogenesis in humans: different effects of isocaloric meals rich in carbohydrate, fat, and protein. Front Nutr 2022;9:1040444. https://doi.org/10.3389/fnut.2022.1040444.
[33] Tentolouris N, Pavlatos S, Kokkinos A, Perrea D, Pagoni S, Katsilambros N. Diet-induced thermogenesis and substrate oxidation are not different between lean and obese women after two different isocaloric meals, one rich in protein and one rich in fat. Metabolism 2008;57:313–20. https://doi.org/10.1016/j.metabol.2007.10.004.
[34] Kassis A, Godin JP, Moille SE, Nielsen-Moennoz C, Groulx K, Oguey-Araymon S, et al. Effects of protein quantity and type on diet induced thermogenesis in overweight adults: a randomized controlled trial. Clin Nutr 2019;38:1570–80. https://doi.org/10.1016/j.clnu.2018.08.004.
[35] Scott CB, Devore R. Diet-induced thermogenesis: variations among three isocaloric meal-replacement shakes. Nutrition 2005;21:874–7. https://doi.org/10.1016/j.nut.2004.12.010.
[36] Westerterp-Plantenga MS, Rolland V, Wilson SA, Westerterp KR. Satiety related to 24 h diet-induced thermogenesis during high protein/carbohydrate vs high fat diets measured in a respiration chamber. Eur J Clin Nutr 1999;53:495–502. https://doi.org/10.1038/sj.ejcn.1600782.
[37] Johnston CS, Day CS, Swan PD. Postprandial thermogenesis is increased 100% on a high-protein, low-fat diet versus a high-carbohydrate, low-fat diet in healthy, young women. J Am Coll Nutr 2002;21:55–61. https://doi.org/10.1080/07315724.2002.10719194.
[38] Karst H, Steiniger J, Noack R, Steglich HD. Diet-induced thermogenesis in man: thermic effects of single proteins, carbohydrates and fats depending on their energy amount. Ann Nutr Metab 1984;28:245–52. https://doi.org/10.1159/000176811.
[39] Petzke KJ, Klaus S. Reduced postprandial energy expenditure and increased exogenous fat oxidation in young woman after ingestion of test meals with a low protein content. Nutr Metab 2008;5:25. https://doi.org/10.1186/1743-7075-5-25.
[40] Westerterp KR, Wilson SA, Rolland V. Diet induced thermogenesis measured over 24h in a respiration chamber: effect of diet composition. Int J Obes Relat Metab Disord 1999;23:287–92. https://doi.org/10.1038/sj.ijo.0800810.
[41] Riggs AJ, White BD, Gropper SS. Changes in energy expenditure associated with ingestion of high protein, high fat versus high protein, low fat meals among underweight, normal weight, and overweight females. Nutr J 2007;6:40. https://doi.org/10.1186/1475-2891-6-40.
[42] Lorenzen J, Frederiksen R, Hoppe C, Hvid R, Astrup A. The effect of milk proteins on appetite regulation and diet-induced thermogenesis. Eur J Clin Nutr 2012;66:622–7. https://doi.org/10.1038/ejcn.2011.221.
[43] Raben A, Agerholm-Larsen L, Flint A, Holst JJ, Astrup A. Meals with similar energy densities but rich in protein, fat, carbohydrate, or alcohol have different effects on energy expenditure and substrate metabolism but not on appetite and energy intake. Am J Clin Nutr 2003;77:91–100. https://doi.org/10.1093/ajcn/77.1.91.
[44] Bowden VL, McMurray RG. Effects of training status on the metabolic responses to high carbohydrate and high fat meals. Int J Sport Nutr Exerc Metabol 2000;10:16–27. https://doi.org/10.1123/ijsnem.10.1.16.
[45] Suter PM, Jequier E, Schutz Y. Effect of ethanol on energy expenditure. Am J Physiol 1994;266:R1204–12. https://doi.org/10.1152/ajpregu.1994.266.4.R1204.
[46] Murgatroyd PR, Van De Ven ML, Goldberg GR, Prentice AM. Alcohol and the regulation of energy balance: overnight effects on diet-induced thermogenesis and fuel storage. Br J Nutr 1996;75:33–45. https://doi.org/10.1079/bjn19960108.
[47] Kasai M, Nosaka N, Maki H, Suzuki Y, Takeuchi H, Aoyama T, et al. Comparison of diet-induced thermogenesis of foods containing medium- versus long-chain triacylglycerols. J Nutr Sci Vitaminol 2002;48:536–40. https://doi.org/10.3177/jnsv.48.536.
[48] Ogawa A, Nosaka N, Kasai M, Aoyama T, Okazaki M, Igarashi O, et al. Dietary medium- and long-chain triacylglycerols accelerate diet-induced thermogenesis in humans. J Oleo Sci 2007;56:283–7. https://doi.org/10.5650/jos.56.283.
[49] Suzuki Y, Nosaka N, Maki H, Kasai M, Aoyama T, Haruna H, et al. Effects of margarine containing medium-chain triglycerides on diet-induced thermogenesis. J Oleo Sci 2005;54:299–304. https://doi.org/10.5650/jos.54.299.
[50] Hill JO, Peters JC, Yang D, Sharp T, Kaler M, Abumrad NN, et al. Thermogenesis in humans during overfeeding with medium-chain triglycerides. Metabolism 1989;38:641–8. https://doi.org/10.1016/0026-0495(89)90101-7.
[51] Dulloo AG, Fathi M, Mensi N, Girardier L. Twenty-four-hour energy expenditure and urinary catecholamines of humans consuming low-to-moderate amounts of medium-chain triglycerides: a dose-response study in a human respiratory chamber. Eur J Clin Nutr 1996;50:152–8. http://www.ncbi.nlm.nih.gov/pubmed/8654328.
[52] Bendixen H, Flint A, Raben A, Hoy CE, Mu H, Xu X, et al. Effect of 3 modified fats and a conventional fat on appetite, energy intake, energy expenditure, and substrate oxidation in healthy men. Am J Clin Nutr 2002;75:47–56. https://doi.org/10.1093/ajcn/75.1.47.
[53] Casas-Agustench P, Lopez-Uriarte P, Bullo M, Ros E, Gomez-Flores A, Salas-Salvado J. Acute effects of three high-fat meals with different fat saturations on energy expenditure, substrate oxidation and satiety. Clin Nutr 2009;28:39–45. https://doi.org/10.1016/j.clnu.2008.10.008.
[54] Piers LS, Walker KZ, Stoney RM, Soares MJ, O'Dea K. The influence of the type of dietary fat on postprandial fat oxidation rates: monounsaturated (olive oil) vs saturated fat (cream). Int J Obes Relat Metab Disord 2002;26:814–21. https://doi.org/10.1038/sj.ijo.0801993.
[55] Nguo K, Huggins CE, Truby H, Sinclair AJ, Clarke RE, Bonham MP. No effect of saturated fatty acid chain length on meal-induced thermogenesis in overweight men. Nutr Res 2018;51:102–10. https://doi.org/10.1016/j.nutres.2018.01.003.
[56] Clevenger HC, Kozimor AL, Paton CM, Cooper JA. Acute effect of dietary fatty acid composition on postprandial metabolism in women. Exp Physiol 2014;99:1182–90. https://doi.org/10.1113/expphysiol.2013.077222.
[57] Clevenger HC, Stevenson JL, Cooper JA. Metabolic responses to dietary fatty acids in obese women. Physiol Behav 2015;139:73–9. https://doi.org/10.1016/j.physbeh.2014.11.022.
[58] Romon M, Edme JL, Boulenguez C, Lescroart JL, Frimat P. Circadian variation of diet-induced thermogenesis. Am J Clin Nutr 1993;57:476–80. https://doi.org/10.1093/ajcn/57.4.476.
[59] Bo S, Fadda M, Castiglione A, Ciccone G, De Francesco A, Fedele D, et al. Is the timing of caloric intake associated with variation in diet-induced thermogenesis and in the metabolic pattern? A randomized cross-over study. Int J Obes 2015;39:1689–95. https://doi.org/10.1038/ijo.2015.138.
[60] Goo RH, Moore JG, Greenberg E, Alazraki NP. Circadian variation in gastric emptying of meals in humans. Gastroenterology 1987;93:515–8. https://doi.org/10.1016/0016-5085(87)90913-9.
[61] Van Cauter E, Desir D, Decoster C, Fery F, Balasse EO. Nocturnal decrease in glucose tolerance during constant glucose infusion. J Clin Endocrinol Metab 1989;69:604–11. https://doi.org/10.1210/jcem-69-3-604.
[62] Linsell CR, Lightman SL, Mullen PE, Brown MJ, Causon RC. Circadian rhythms of epinephrine and norepinephrine in man. J Clin Endocrinol Metab 1985;60:1210–5. https://doi.org/10.1210/jcem-60-6-1210.
[63] Weststrate JA, Weys PJ, Poortvliet EJ, Deurenberg P, Hautvast JG. Diurnal variation in postabsorptive resting metabolic rate and diet-induced thermogenesis. Am J Clin Nutr 1989;50:908–14. https://doi.org/10.1093/ajcn/50.5.908.
[64] Sato M, Nakamura K, Ogata H, Miyashita A, Nagasaka S, Omi N, et al. Acute effect of late evening meal on diurnal variation of blood glucose and energy metabolism. Obes Res Clin Pract 2011;5:e169–266. https://doi.org/10.1016/j.orcp.2011.02.001.
[65] Bandin C, Scheer FA, Luque AJ, Avila-Gandia V, Zamora S, Madrid JA, et al. Meal timing affects glucose tolerance, substrate oxidation and circadian-related variables: a randomized, crossover trial. Int J Obes 2015;39:828–33. https://doi.org/10.1038/ijo.2014.182.
[66] Benedict C, Hallschmid M, Lassen A, Mahnke C, Schultes B, Schioth HB, et al. Acute sleep deprivation reduces energy expenditure in healthy men. Am J Clin Nutr 2011;93:1229–36. https://doi.org/10.3945/ajcn.110.006460.
[67] Nedeltcheva AV, Kilkus JM, Imperial J, Kasza K, Schoeller DA, Penev PD. Sleep curtailment is accompanied by increased intake of calories from snacks. Am J Clin Nutr 2009;89:126–33. https://doi.org/10.3945/ajcn.2008.26574.
[68] St-Onge MP, Roberts AL, Chen J, Kelleman M, O'Keeffe M, RoyChoudhury A, et al. Short sleep duration increases energy intakes but does not change energy expenditure in normal-weight individuals. Am J Clin Nutr 2011;94:410–6. https://doi.org/10.3945/ajcn.111.013904.
[69] Du S, Rajjo T, Santosa S, Jensen MD. The thermic effect of food is reduced in older adults. Horm Metab Res 2014;46:365–9. https://doi.org/10.1055/s-0033-1357205.
[70] Thorne A, Wahren J. Diminished meal-induced thermogenesis in elderly man. Clin Physiol 1990;10:427–37. https://doi.org/10.1111/j.1475-097x.1990.tb00823.x.
[71] Schwartz RS, Jaeger LF, Veith RC. The thermic effect of feeding in older men: the importance of the sympathetic nervous system. Metabolism 1990;39:733–7. https://doi.org/10.1016/0026-0495(90)90109-p.
[72] Jones PP, Van Pelt RE, Johnson DG, Seals DR. Role of sympathetic neural activation in age- and habitual exercise-related differences in the thermic effect of food. J Clin Endocrinol Metab 2004;89:5138–44. https://doi.org/10.1210/jc.2004-0101.
[73] Bloesch D, Schutz Y, Breitenstein E, Jequier E, Felber JP. Thermogenic response to an oral glucose load in man: comparison between young and elderly subjects. J Am Coll Nutr 1988;7:471–83. https://doi.org/10.1080/07315724.1988.10720263.

[74] Golay A, Schutz Y, Broquet C, Moeri R, Felber JP, Jequier E. Decreased thermogenic response to an oral glucose load in older subjects. J Am Geriatr Soc 1983;31:144–8. https://doi.org/10.1111/j.1532-5415.1983.tb04853.x.

[75] Visser M, Deurenberg P, van Staveren WA, Hautvast JG. Resting metabolic rate and diet-induced thermogenesis in young and elderly subjects: relationship with body composition, fat distribution, and physical activity level. Am J Clin Nutr 1995;61:772–8. https://doi.org/10.1093/ajcn/61.4.772.

[76] Vaughan L, Zurlo F, Ravussin E. Aging and energy expenditure. Am J Clin Nutr 1991;53:821–5. https://doi.org/10.1093/ajcn/53.4.821.

[77] Leenen R, van der Kooy K, Deurenberg P, Seidell JC, Weststrate JA, Schouten FJ, et al. Visceral fat accumulation in obese subjects: relation to energy expenditure and response to weight loss. Am J Physiol 1992;263:E913–9. https://doi.org/10.1152/ajpendo.1992.263.5.E913.

[78] Blaak EE, Hul G, Verdich C, Stich V, Martinez JA, Petersen M, et al. Impaired fat-induced thermogenesis in obese subjects: the NUGENOB study. Obesity 2007;15:653–63. https://doi.org/10.1038/oby.2007.606.

[79] Thorne A, Hallberg D, Wahren J. Meal-induced thermogenesis in obese patients before and after weight reduction. Clin Physiol 1989;9:481–98. https://doi.org/10.1111/j.1475-097x.1989.tb01002.x.

[80] Bukkens SG, McNeill G, Smith JS, Morrison DC. Postprandial thermogenesis in post-obese women and weight-matched controls. Int J Obes 1991;15:147–54. http://www.ncbi.nlm.nih.gov/pubmed/2040551.

[81] Segal KR, Edano A, Tomas MB. Thermic effect of a meal over 3 and 6 hours in lean and obese men. Metabolism 1990;39:985–92. https://doi.org/10.1016/0026-0495(90)90312-z.

[82] Swaminathan R, King RF, Holmfield J, Siwek RA, Baker M, Wales JK. Thermic effect of feeding carbohydrate, fat, protein and mixed meal in lean and obese subjects. Am J Clin Nutr 1985;42:177–81. https://doi.org/10.1093/ajcn/42.2.177.

[83] Tounian P, Girardet JP, Carlier L, Frelut ML, Veinberg F, Fontaine JL. Resting energy expenditure and food-induced thermogenesis in obese children. J Pediatr Gastroenterol Nutr 1993;16:451–7. https://doi.org/10.1097/00005176-199305000-00019.

[84] Marques-Lopes I, Forga L, Martinez JA. Thermogenesis induced by a high-carbohydrate meal in fasted lean and overweight young men: insulin, body fat, and sympathetic nervous system involvement. Nutrition 2003;19:25–9. https://doi.org/10.1016/s0899-9007(02)00950-4.

[85] Maffeis C, Schutz Y, Grezzani A, Provera S, Piacentini G, Tato L. Meal-induced thermogenesis and obesity: is a fat meal a risk factor for fat gain in children? J Clin Endocrinol Metab 2001;86:214–9. https://doi.org/10.1210/jcem.86.1.7132.

[86] D'Alessio DA, Kavle EC, Mozzoli MA, Smalley KJ, Polansky M, Kendrick ZV, et al. Thermic effect of food in lean and obese men. J Clin Invest 1988;81:1781–9. https://doi.org/10.1172/JCI113520.

[87] Shetty PS, Jung RT, James WP, Barrand MA, Callingham BA. Postprandial thermogenesis in obesity. Clin Sci (Lond) 1981;60:519–25. https://doi.org/10.1042/cs0600519.

[88] Bessard T, Schutz Y, Jequier E. Energy expenditure and postprandial thermogenesis in obese women before and after weight loss. Am J Clin Nutr 1983;38:680–93. https://doi.org/10.1093/ajcn/38.5.680.

[89] Thorne A, Naslund I, Wahren J. Meal-induced thermogenesis in previously obese patients. Clin Physiol 1990;10:99–109. https://doi.org/10.1111/j.1475-097x.1990.tb00087.x.

[90] Scalfi L, Coltorti A, Borrelli R, Contaldo F. Postprandial thermogenesis in leanness and anorexia nervosa. Ann Nutr Metab 1992;36:48–54. https://doi.org/10.1159/000177698.

[91] Schutz Y, Bessard T, Jequier E. Diet-induced thermogenesis measured over a whole day in obese and nonobese women. Am J Clin Nutr 1984;40:542–52. https://doi.org/10.1093/ajcn/40.3.542.

[92] Golay A, Schutz Y, Meyer HU, Thiebaud D, Curchod B, Maeder E, et al. Glucose-induced thermogenesis in nondiabetic and diabetic obese subjects. Diabetes 1982;31:1023–8. https://doi.org/10.2337/diacare.31.11.1023.

[93] Ravussin E, Bogardus C, Schwartz RS, Robbins DC, Wolfe RR, Horton ES, et al. Thermic effect of infused glucose and insulin in man. Decreased response with increased insulin resistance in obesity and noninsulin-dependent diabetes mellitus. J Clin Invest 1983;72:893–902. https://doi.org/10.1172/JCI111060.

[94] Segal KR, Albu J, Chun A, Edano A, Legaspi B, Pi-Sunyer FX. Independent effects of obesity and insulin resistance on postprandial thermogenesis in men. J Clin Invest 1992;89:824–33. https://doi.org/10.1172/JCI115661.

[95] Hibi M, Oishi S, Matsushita M, Yoneshiro T, Yamaguchi T, Usui C, et al. Brown adipose tissue is involved in diet-induced thermogenesis and whole-body fat utilization in healthy humans. Int J Obes 2016;40:1655–61. https://doi.org/10.1038/ijo.2016.124.

[96] Granata GP, Brandon LJ. The thermic effect of food and obesity: discrepant results and methodological variations. Nutr Rev 2002;60:223–33. https://doi.org/10.1301/002966402320289359.

[97] Carneiro IP, Elliott SA, Siervo M, Padwal R, Bertoli S, Battezzati A, et al. Is obesity associated with altered energy expenditure? Adv Nutr 2016;7:476–87. https://doi.org/10.3945/an.115.008755.

[98] Thyfault JP, Richmond SR, Carper MJ, Potteiger JA, Hulver MW. Postprandial metabolism in resistance-trained versus sedentary males. Med Sci Sports Exerc 2004;36:709–16. https://doi.org/10.1249/01.mss.0000121946.98885.f5.

[99] Poehlman ET, Melby CL, Badylak SF. Relation of age and physical exercise status on metabolic rate in younger and older healthy men. J Gerontol 1991;46:B54–8. https://doi.org/10.1093/geronj/46.2.b54.

[100] Burke CM, Bullough RC, Melby CL. Resting metabolic rate and postprandial thermogenesis by level of aerobic fitness in young women. Eur J Clin Nutr 1993;47:575–85. http://www.ncbi.nlm.nih.gov/pubmed/8404794.

[101] Thorne A. Diet-induced thermogenesis. An experimental study in healthy and obese individuals. Acta Chir Scand Suppl 1990;558:6–59. http://www.ncbi.nlm.nih.gov/pubmed/2178297.

[102] Ratcliff L, Gropper SS, White BD, Shannon DM, Huggins KW. The influence of habitual exercise training and meal form on diet-induced thermogenesis in college-age men. Int J Sport Nutr Exerc Metabol 2011;21:11–8. https://doi.org/10.1123/ijsnem.21.1.11.

[103] Thorne A, Wahren J. Diet-induced thermogenesis in well-trained subjects. Clin Physiol 1989;9:295–305. https://doi.org/10.1111/j.1475-097x.1989.tb00982.x.

[104] Tremblay A, Cote J, LeBlanc J. Diminished dietary thermogenesis in exercise-trained human subjects. Eur J Appl Physiol Occup Physiol 1983;52:1–4. https://doi.org/10.1007/BF00429016.

[105] LeBlanc J, Mercier P, Samson P. Diet-induced thermogenesis with relation to training state in female subjects. Can J Physiol Pharmacol 1984;62:334–7. https://doi.org/10.1139/y84-052.

[106] Segal KR, Blando L, Ginsberg-Fellner F, Edano A. Postprandial thermogenesis at rest and postexercise before and after physical training in lean, obese, and mildly diabetic men. Metabolism 1992;41:868–78. https://doi.org/10.1016/0026-0495(92)90169-b.

[107] Ohnaka M, Iwamoto M, Sakamoto S, Niwa Y, Matoba H, Nakayasu K, et al. Does prolonged exercise alter diet-induced thermogenesis? Ann Nutr Metab 1998;42:311–8. https://doi.org/10.1159/000012750.

[108] Nair KS, Webster J, Garrow JS. Effect of impaired glucose tolerance and type II diabetes on resting metabolic rate and thermic response to a glucose meal in obese women. Metabolism 1986;35:640–4. https://doi.org/10.1016/0026-0495(86)90171-x.

[109] Gumbiner B, Thorburn AW, Henry RR. Reduced glucose-induced thermogenesis is present in noninsulin-dependent diabetes mellitus without obesity. J Clin Endocrinol Metab 1991;72:801–7. https://doi.org/10.1210/jcem-72-4-801.

[110] Camastra S, Bonora E, Del Prato S, Rett K, Weck M, Ferrannini E. Effect of obesity and insulin resistance on resting and glucose-induced thermogenesis in man. EGIR (European Group for the Study of Insulin Resistance). Int J Obes Relat Metab Disord 1999;23:1307–13. https://doi.org/10.1038/sj.ijo.0801072.

[111] Bellamy L, Casas JP, Hingorani AD, Williams D. Type 2 diabetes mellitus after gestational diabetes: a systematic review and meta-analysis. Lancet 2009;373:1773–9. https://doi.org/10.1016/S0140-6736(09)60731-5.

[112] Robinson S, Niththyananthan R, Anyaoku V, Elkeles RS, Beard RW, Johnston DG. Reduced postprandial energy expenditure in women predisposed to type 2 diabetes. Diabet Med 1994;11:545–50. https://doi.org/10.1111/j.1464-5491.1994.tb02033.x.

[113] Forbes S, Robinson S, Parker KH, Macdonald IA, McCarthy MI, Johnston DG. The thermic response to food is related to sensitivity to adrenaline in a group at risk for the development of type II diabetes. Eur J Clin Nutr 2009;63:1360–7. https://doi.org/10.1038/ejcn.2009.91.

[114] Kousta E, Parker KH, Lawrence NJ, Penny A, Millauer BA, Anyaoku V, et al. Delayed metabolic and thermogenic response to a mixed meal in normoglycemic European women with previous gestational diabetes. J Clin Endocrinol Metab 2002;87:3407–12. https://doi.org/10.1210/jcem.87.7.8698.

[115] Sadeghi HM, Adeli I, Calina D, Docea AO, Mousavi T, Daniali M, et al. Polycystic ovary syndrome: a comprehensive review of pathogenesis, management, and drug repurposing. Int J Mol Sci 2022;23. https://doi.org/10.3390/ijms23020583.

[116] Robinson S, Chan SP, Spacey S, Anyaoku V, Johnston DG, Franks S. Postprandial thermogenesis is reduced in polycystic ovary syndrome and is associated with increased insulin resistance. Clin Endocrinol 1992;36:537–43. https://doi.org/10.1111/j.1365-2265.1992.tb02262.x.

[117] Segal KR, Dunaif A. Resting metabolic rate and postprandial thermogenesis in polycystic ovarian syndrome. Int J Obes 1990;14:559–67. http://www.ncbi.nlm.nih.gov/pubmed/2228390.

[118] Abdelhafiz AH, Koay L, Sinclair AJ. The emergence of frailty may lead to a state of burnt out type 2 diabetes. J Frailty Aging 2016;5:162–7. http://www.ncbi.nlm.nih.gov/pubmed/29239594.

[119] Goulet ED, Khursigara Z, Gougeon R, Morais JA. Postprandial insulin sensitivity and thermogenesis in frail elderly women. Appl Physiol Nutr Metabol 2010;35:526–33. https://doi.org/10.1139/H10-041.

[120] Brooks B, Molyneaux L, Zilkens R, Ross G, Yue DK. The use of Acarbose in Type 2 diabetic patients in secondary failure: effects on glycaemic control and diet induced thermogenesis. Diabetes Res Clin Pract 1998;42:175–80. https://doi.org/10.1016/s0168-8227(98)00114-4.

[121] Benedict C, Brede S, Schioth HB, Lehnert H, Schultes B, Born J, et al. Intranasal insulin enhances postprandial thermogenesis and lowers postprandial serum insulin levels in healthy men. Diabetes 2011;60:114–8. https://doi.org/10.2337/db10-0329.

[122] Rigalleau V, Rabemanantsoa C, Gin H. A three-day insulin-induced normoglycemia improves carbohydrate oxidation in type 2 diabetic subjects. Metabolism 2002;51:1484–8. https://doi.org/10.1053/meta.2002.35593.

[123] Zachariah S, Sheldon B, Shojaee-Moradie F, Jackson NC, Backhouse K, Johnsen S, et al. Insulin detemir reduces weight gain as a result of reduced food intake in patients with type 1 diabetes. Diabetes Care 2011;34:1487–91. https://doi.org/10.2337/dc11-0098.

[124] Maciel MG, Beserra BTS, Oliveira FCB, Ribeiro CM, Coelho MS, Neves FAR, et al. The effect of glucagon-like peptide 1 and glucagon-like peptide 1 receptor agonists on energy expenditure: a systematic review and meta-analysis. Diabetes Res Clin Pract 2018;142:222–35. https://doi.org/10.1016/j.diabres.2018.05.034.

[125] Maratou E, Hadjidakis DJ, Kollias A, Tsegka K, Peppa M, Alevizaki M, et al. Studies of insulin resistance in patients with clinical and subclinical hypothyroidism. Eur J Endocrinol 2009;160:785–90. https://doi.org/10.1530/EJE-08-0797.

[126] Biondi B, Kahaly GJ, Robertson RP. Thyroid dysfunction and diabetes mellitus: two closely associated disorders. Endocr Rev 2019;40:789–824. https://doi.org/10.1210/er.2018-00163.

[127] Kozacz A, Grunt P, Steczkowska M, Mikulski T, Dabrowski J, Gorecka M, et al. Thermogenic effect of glucose in hypothyroid subjects. Int J Endocrinol 2014; 2014:308017. https://doi.org/10.1155/2014/308017.

[128] Randin JP, Schutz Y, Scazziga B, Lemarchand-Beraud T, Felber JP, Jequier E. Unaltered glucose-induced thermogenesis in Graves' disease. Am J Clin Nutr 1986;43:738–44. https://doi.org/10.1093/ajcn/43.5.738.

[129] Acheson K, Jequier E, Burger A, Danforth Jr E. Thyroid hormones and thermogenesis: the metabolic cost of food and exercise. Metabolism 1984;33: 262–5. https://doi.org/10.1016/0026-0495(84)90048-9.