



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. In spite 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 $^{\text{D}}$ -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 swallow; 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 $[\Delta] = 205.6 \pm 78.9$ kJ/h, $P < 0.001$ for lean and $[\Delta] = 169.8 \pm 114.3$ 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 thermogenic 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 rich in polyunsaturated fatty acids (PUFA) and 23 % after a meal with a high content in monounsaturated 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 ($9.7 \pm 2.5\%$ and $6.5 \pm 4.0\%$ 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 peak 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 ± 3.90 versus 55.40 ± 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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Evangelia Tzeravini: Writing – review & editing, Data curation.
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Tentolouris Nikolaos: Writing – review & editing, Supervision, Methodology.
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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.

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