International Journal of Food Sciences and Nutrition

ISSN: (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/iijf20

Effects of acute and chronic nuts consumption on energy metabolism: a systematic review of randomised clinical trials

Yuliana María Franco Estrada, Ana Paula Silva Caldas, Alessandra da Silva & Josefina Bressan

To cite this article: Yuliana María Franco Estrada, Ana Paula Silva Caldas, Alessandra da Silva & Josefina Bressan (2021): Effects of acute and chronic nuts consumption on energy metabolism: a systematic review of randomised clinical trials, International Journal of Food Sciences and Nutrition, DOI: 10.1080/09637486.2021.1984401

To link to this article: https://doi.org/10.1080/09637486.2021.1984401

Published online: 04 Oct 2021.

INTERNATIONAL JOURNAL OF FOOD SCIENCES AND NUTRITION
https://doi.org/10.1080/09637486.2021.1984401

Taylor & Francis
Taylor & Francis Group

RESEARCH ARTICLE

Effects of acute and chronic nuts consumption on energy metabolism: a systematic review of randomised clinical trials

Yuliana María Franco Estrada, Ana Paula Silva Caldas, Alessandra da Silva and Josefina Bressan

Departamento de Nutrição e Saúde, Universidade Federal de Viçosa, Minas Gerais, Brazil

ABSTRACT

Nuts are high-energy density foods and are associated with beneficial effects on health, including weight control. Effects on resting energy expenditure, respiratory quotient, and diet-induced thermogenesis are suggested mechanisms behind the effects of nuts consumption on weight control. Thus, we revised the randomised clinical trials that assessed acute and chronic nuts consumption effects on energy metabolism. Walnuts (22.1 g to 56 g) consumption appears to modulate energy metabolism markers differently depending on the dose and profile of the evaluated subject. In its turn, 56 g of high-oleic peanuts increased postprandial energy expenditure and thermic effect of food after three hours postprandial compared to consumption of conventional peanuts. Almonds, hazelnuts, peanuts, and a mix of nuts were the nuts studies in the chronic studies, which does not seem to influence energy metabolism markers. Further studies are needed to elucidate the effects of other types of nuts consumption on energy metabolism.

ARTICLE HISTORY
Received 8 June 2021
Revised 14 September 2021
Accepted 20 September 2021

KEYWORDS
Nutrition; nuts; postprandial energy expenditure; resting energy expenditure; thermic effect of food

Introduction

Obesity is currently considered a global epidemic and one of the main public health concerns in the world. The World Health Organisation (WHO) projections showed that more than 1.9 billion adults were overweight in the last decade, of which 650 million were obese (WHO 2020a). Furthermore, overweight and obesity are independent risk factors for non-communicable chronic diseases, leading to around 2.8 million deaths annually (WHO 2020b).

In the literature, a modest weight loss between 5% and 10% has been shown to promote favourable health effects (Bray et al. 2018). Among the most effective weight control strategies, reducing energy consumption and increasing energy expenditure at a negative energy balance are highlighted (Jéquier and Tappy 1999; Bray 2012; Valeria et al. 2014). Of the three principal components of energy expenditure, resting energy expenditure (REE) is responsible for 60–75% of total energy expenditure (TEE). Additionally, the contribution of diet-induced thermogenesis (DIT) and physical activity to TEE is 10–15% and 10–30%, respectively. Thus, increasing REE and DIT can be potential nutritional strategies for body weight management, positively impacting obesity control and prevention (Dulloo et al. 1999).

Besides caloric restriction, the adoption of healthy eating patterns, which include foods with functional claims, is a powerful approach to prevent or control obesity and its comorbidities (Mancini et al. 2016; Soltani et al. 2016). From this perspective, nuts are a group of oilseeds including almonds, pistachios, pine nuts, macadamia, Brazil nuts, cashew nuts, walnuts, hazelnuts, pecans, and peanuts (Souza et al. 2017). They are high in unsaturated fatty acids such as monounsaturated (MUFA) and polyunsaturated (PUFA) fatty acids, protein, fibre, vitamins, minerals, and phytochemicals (Coates et al. 2018). The consumption of nuts is associated with body weight control despite being calorie dense (Martínez-González and Bes-Rastrollo 2011; Flores-Mateo et al. 2013). Among several proposed mechanisms behind this effect, improvement in energy metabolism mediated by an increase in REE, DIT, and fat oxidation has been suggested (Alper and Mattes 2002; Tan et al. 2014; Tindall et al. 2018). However, no review critically and systematically evaluated the findings of energy metabolism reported by randomised clinical trials focussed on acute and chronic nuts consumption.

Thus, we aimed to revise the studies that evaluate acute and chronic nuts consumption effects on energy

CONTACT Yuliana María Franco Estrada yulises58@gmail.com Department of Nutrition and Health, Universidade Federal de Viçosa, Av. P.H. Rolfs s/n, Campus Universitário, Viçosa - Minas Gerais 36570-900, Brazil

metabolism. Furthermore, the physiological mechanism involved is discussed.

Material and methods

Protocol and registration

This systematic review was carried out according to the PRISMA statement (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) (Moher et al. 2009). The review was registered on PROSPERO: registration number CRD42020141910.

Eligibility criteria

Type of study: Randomised clinical trials (RCTs).

Type of subject: Adults $\geq$ 18 years old, healthy, normal weight, overweight, or obese were included. Studies conducted with athletes, pregnant or lactating women, and individuals with specific pathologies that may interfere with energy metabolism were excluded (Spies et al. 2012; Ajith and Jayakumar 2016).

Type of intervention: Studies that evaluated the effects of acute (postprandial studies) and chronic (regular consumption of nuts) consumption of several varieties of nuts (almond, Brazil nut, cashew nut, hazelnut, macadamia, pistachio, walnut, tree nuts, peanuts), in different forms (roasted, raw, paste, or crushed) and quantities on energy metabolism. Uncontrolled or controlled studies where nuts were combined with meals or other foods were also included. However, studies conducted with nut products such as nut oil were not considered.

Primary results: Changes in resting energy expenditure (REE), respiratory quotient (RQ), and nutrient oxidation ratio.

Literature search

Studies were identified by searching electronic databases: PubMed/MEDLINE, Scopus, The Cochrane Library, and Web of Science (Science and Social Science Citation Index). To improve the search terms, the Medical Subject Headings (MeSH) were used. The following search terms were used: title and abstract ("nuts" OR "almonds" OR "brazil nuts" OR "cashews" OR "hazelnuts" OR "macadamia" OR "pecans" OR "pine nuts" OR "pistachios" OR "walnuts" OR "peanuts" OR "Baru nut" OR "tree nuts" OR "groundnut") and for all text (thermogenesis OR substrate oxidation OR resting metabolic rate OR resting energy expenditure OR basal metabolic rate OR thermic effect of food OR indirect calorimetry OR indirect calorimetric OR respiration calorimetric OR respiration calorimetry OR energy expenditure OR doubly labelled water OR respiratory quotient OR macronutrient oxidation OR energy metabolism OR fat oxidation OR carbohydrate oxidation OR protein oxidation). Filters for human studies were used when available. The search strategy had no date restrictions. The literature search was conducted on July 17th, 2019, but an updated search was performed on July 30, 2021.

Study selection and data extraction

Eligibility was separately evaluated by two reviewers (YMFE and APSC), and any discrepancy was resolved by a third reviewer (AS). The first stage of the screening process involved the assessment of titles and abstracts of the search results against the inclusion and exclusion criteria. Then, the pre-selected studies were read in full for eligibility. For studies that meet the inclusion criteria, the following relevant information was extracted: authors, publication year, study design, subject characteristics (sample size, mean age, gender, BMI), intervention design (type of nut, control group, and study duration), and outcome measures of interest (REE, DIT, RQ, and nutrient oxidation ratio). When important information was lacking, an email was sent to the authors. All the extracted information was summarised in a standardised data extraction table (Table 1).

Risk of bias assessment

The risk of bias of the selected studies was checked using the Cochrane collaboration tool (Higgins et al. 2011). Two authors (YMFE and APSC) independently classified the studies as having a high, low, or unclear, risk of bias according to the following domains: (1) selection, (2) performance, (3) detection, (4) attrition, and (5) reporting bias. Regarding study selection, discrepancies were solved by a third author (AS). For assessing some items, the outcomes reported in the published reports were compared to study protocols, when available. Risk of bias assessment was carried out using Review Manager v. 5.3 (Copenhagen: The Nordic Cochrane Centre, 2014). Risk of bias was classified according to the percentage of "low risk" questions, where $>$ 80%, 50 to 79%, $<$ 50% was classified as low, moderate, and high risk of bias, respectively (Gomes et al. 2017).

Table 1. Characteristics of clinical trials investigating the effects of acute and chronic nuts consumption on energy metabolism.

Author, year, country	Study Design (Follow-up)	Characteristics of Subjects	Characteristics of Intervention	Results
Acute studies				
Casas-Agustench et al. 2009 Spain	Randomised, Crossover (Acute, 5 h pp)	n: 29 men Eutrophic and overweight Age: 22 ± 4 y BMI: 24.1 ± 4.5 kg/m^2	G1: isocaloric meal rich in PUFA, walnuts: 22.1 g; 33.3 %E G2: isocaloric meal rich in MUFA (virgin olive oil): 20.4 g; 30.8 %E G3: isocaloric meal rich in SFA (dairy products): 20.8 g; 31.9 %E	G1 *vs.* G3: ↑ PEE and ↑ TEF ↔RQ, CO, FO, PO
Tapsell et al. 2009 Australia	Randomised, Crossover, Controlled (Acute, 8 h pp)	n: 16 (4 with type 2 diabetes) Overweight and obesity F/M: 9/7 Age: 52.8 ± 10 y BMI: 31.2 ± 2.9 kg/m^2	G1: 25–35 g of walnuts within an isocaloric diet (PUFA: 8.5 % E; MUFA: 13.5 % E) G2: olive oil within an isocaloric diet (PUFA: 8.9 %E; MUFA: 13.4 %E)	↔ PEE, ↓ RQ, CO, ↑ FO, ↔PO
Alves, Moreira, Macedo, Costa et al. 2014 Brazil	Randomised, Controlled (Acute, 3 h pp)	n: 71 men Overweight Age: 27.1 ± 0.9 y BMI: 29.8 ± 0.3 kg/m^2	G1: 56 g of unpeeled roasted conventional peanuts (51% of oleic fatty acid from total fat) G2: 56 g of unpeeled roasted high-oleic peanuts (81.5% of oleic fatty acid from total fat) G3: 56 g of biscuits (35.6% of oleic fatty acid from total fat)	G2 *vs.* G1: ↑ PEE and ↑ TEF ↔ RQ, CO, FO, PO
Gepner et al. 2016 Israel	Randomised, Crossover, Controlled (Acute, 40 min pp)	N: 40 men Overweight and obesity Age: 45 ± 8 y BMI: 31.1 ± 3.8 kg/m^2	G1: 56 g of walnuts (8% carbohydrate; 84% fat, of which 72% PUFA) G2: 150 g of whole-grain bread (48% carbohydrate; 32% fat)	↓ TEF ↔ RQ
Chronic studies				
Fraser et al. 2002 USA	Randomised, controlled, crossover (24 weeks weeks)	n: 81[§] (41) Euthophic, overweight, and obese F/M: 38/43 Age: 49.5 ± 13.5* y BMI (kg/m^2): 26.3 ± 6.33*	G1: 54,3 g of raw or dry-roasted almonds, without food guidance G2: Usual diet, without nuts or dietary advice	↔REE, RQ
Hollis and Mattes 2007 USA	Randomised, controlled, crossover (10 weeks)	n: 20 women Overweight Age: 24,0 ± 9,0 y BMI (kg/m^2): 25.9 ± 3.1	G1: ∼58 g○ of raw, unsalted almonds G2: Usual diet, without nuts	↔ REE, TEF
Tey et al. 2011 New Zealand	Randomised, controlled, parallel arm (12 weeks)	n: 61[§] (27) Eutrophic and overweight F/M: 63/55 Age 37.5 ± 14.8 y BMI (kg/m^2): 23.8 ± 2.8	G1: 42 g hazelnuts. G2: Usual diet, without nuts	↔REE, RQ
Alves, Moreira, Macedo, Alfenas et al. 2014 Brazil	Randomised, controlled, parallel arm (4 weeks)	n: 65 men Overweight Age: 27.4 ± 1,7* y BMI (kg/m^2): 29.7 ± 0.5*	G1: Calorie restriction diet (-250 kcal) + 56 g unpeeled roasted conventional peanuts (51% of oleic fatty acid from total fat). G2: Calorie restriction diet (-250 kcal) + 56 g unpeeled roasted high-oleic peanuts (81.5% of oleic fatty acid from total fat). G3: Calorie restriction diet (-250 kcal)	↔ REE, RQ, CO, FO
Chronic studies (uncontrolled)				
Claesson et al. 2009 Sweden	Randomised, parallel (2 weeks)	n: 25 (13 REE) F/M: 14/11 Eutrophic Age: 23.4 ± 2.7 y BMI (kg/m^2): 22.2 ± 1.7	G1: ∼259 g○ roasted and salted peanuts within a habitual diet G2: Candy, 20 kcal/kg/day within a habitual diet	G1: ↑REE compared to baseline G2: ↔ REE compared to baseline
Agebratt et al. 2016 Sweden	Randomised, parallel (8 weeks)	n: 30 F/M: 18/12 Eutrophic and overweight Age: 23.5 ± 3.7 y BMI (kg/m^2): 22.3 ± 1.9	G1: Habitual diet with 81-91 g of a mix of nuts (cashews, peanuts, walnuts, almonds, pistachios, hazelnuts, Brazil nuts, macadamias, pecan nuts) G2: Habitual diet with 7 kcal per kg bodyweight per day (637 g) of bananas, apples, citrus fruits, pears, melons, grapes, mangos, kiwis, persimmons, pineapples or plums.	G1: ↑REE compared to baseline G2: ↔ REE compared to baseline

G, Grupo; pp, postprandial; F, female; M, male; %E, percentage of energy; PEE, postprandial energy expenditure; TEF, Thermic effect of food; REE, resting energy expenditure; RQ, respiratory quotient; FO, Fat oxidation; CO, carbohydrate oxidation; PUFA, polyunsaturated fat; SFA, saturated fat, MUFA, monounsaturated fat; *Standard error; § Studies that performed the analyzes on subsamples, highlighted in parentheses next to the total number of individuals. ^ Grams estimated by the USDA.

Results

Study selection

Six hundred and twelve studies were identified by the electronic database search. After excluding duplicates, 477 studies remained. Based on the title and abstract, 463 studies were excluded. The most common exclusion criteria were: studies carried out with animals, pregnant women, children, adolescents, or individuals with cardiometabolic diseases; interventions with nut by-products, such as oil; and in vitro studies. The remaining fourteen studies were read in full for eligibility. Ten studies met all the criteria for the systematic review and were included in the review (Figure 1).

Study characteristics

Study design

Clinical trials evaluating the acute and chronic effects of nuts consumption on energy metabolism were included in this systematic review. Among the ten selected studies, four were randomised, postprandial studies (Casas-Agustench et al. 2009; Tapsell et al. 2009; Alves, Moreira, Macedo, Costa et al. 2014; Gepner et al. 2016), two were chronic, randomised parallel clinical trials (Alves, Moreira, Macedo, Alfenas et al. 2014; Tey et al. 2011), two were chronic, randomised crossover clinical trials (Fraser et al. 2002; Hollis and Mattes 2007), two were chronic, randomised uncontrolled clinical trials (Claesson et al. 2009;

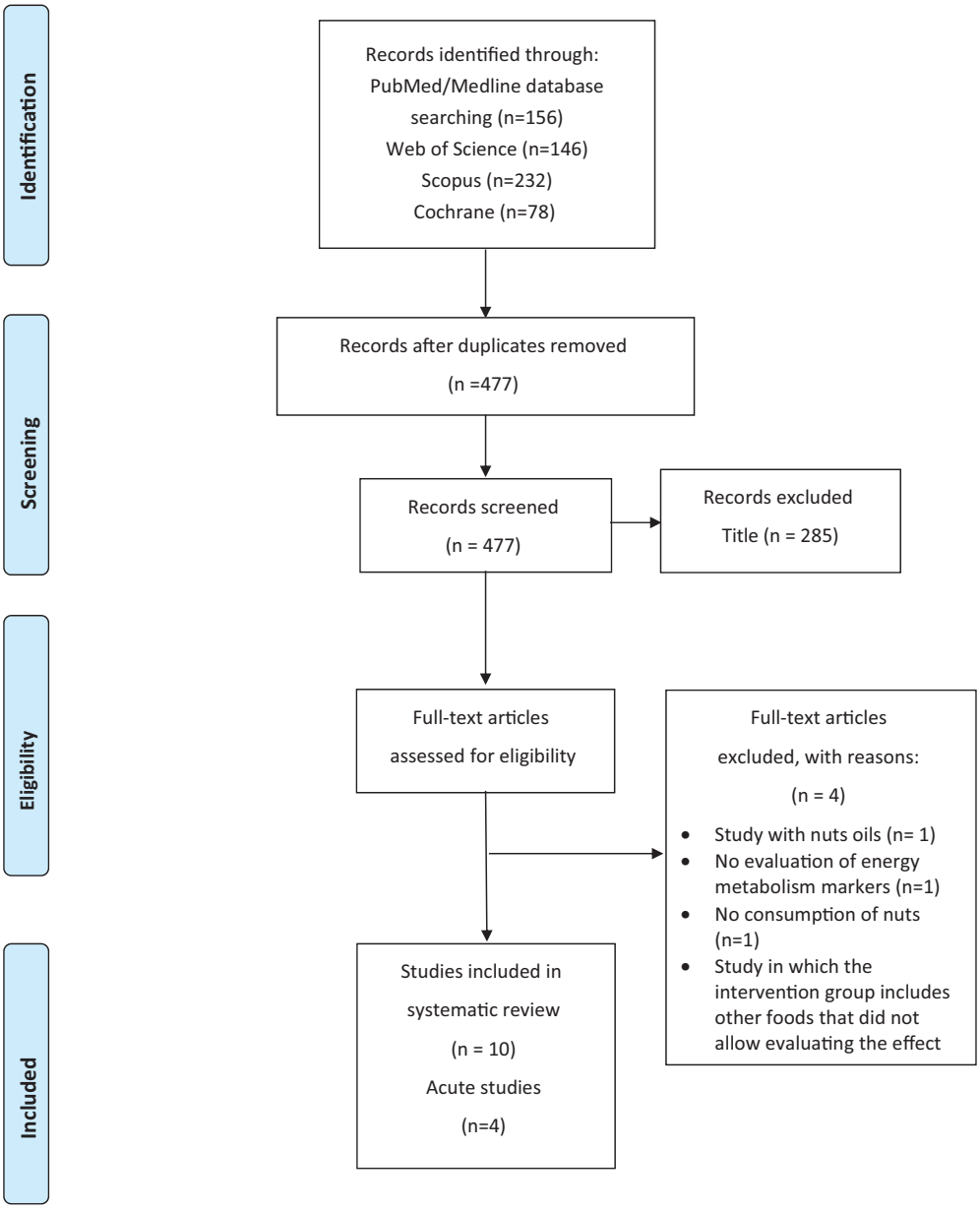

Figure 1. Flowchart of selected studies.

Agebratt et al. 2016). The duration of the chronic studies ranged from 2 to 24 weeks.

Eligible trials included a total of 453 normal healthy weight or overweight subjects. The mean age of the participants ranged from 22 ± 4 to 52.8 ± 10 years. Four studies were carried out with men (Casas-Agustench et al. 2009; Alves, Moreira, Macedo, Costa et al. 2014; Alves, Moreira, Macedo, Alfenas et al. 2014; Gepner et al. 2016), one with women (Hollis and Mattes 2007); and five studies involved both genders (Fraser et al. 2002; Claesson et al. 2009; Tapsell et al. 2009; Tey et al. 2011; Agebratt et al. 2016). Regarding study location, two were conducted in the United States (Fraser et al. 2002; Hollis and Mattes 2007), one in New Zealand (Tey et al. 2011), two in Brazil (Alves, Moreira, Macedo, Costa et al. 2014; Alves, Moreira, Macedo, Alfenas et al. 2014), one in Israel (Gepner et al. 2016), one in Spain (Casas-Agustench et al. 2009), one in Australia (Tapsell et al. 2009), and two in Sweden (Claesson et al. 2009; Agebratt et al. 2016) (Table 1).

The studies included in the systematic review differ as to the type and dose of nuts studied. Furthermore, they differ regarding the energy metabolism markers assessed and regarding the profile of the assessed subjects. Due to this lack of homogeneity between studies, a meta-analysis was not performed.

Intervention

Ten different nuts were evaluated, including almonds (Fraser et al. 2002; Hollis and Mattes 2007), conventional and high-oleic peanuts (Claesson et al. 2009; Alves, Moreira, Macedo, Costa et al. 2014; Alves, Moreira, Macedo, Alfenas et al. 2014), walnuts (Casas-Agustench et al. 2009; Gepner et al. 2016; Tapsell et al. 2009), hazelnuts (Tey et al. 2011), and a mix of nuts (cashews, peanuts, walnuts, almonds, pistachios, hazelnuts, Brazil nuts, macadamias, pecan nuts) (Agebratt et al. 2016). The nuts were raw or toasted, with or without shell, and salted or unsalted (Table 1). In turn, in the chronic studies, the doses of nuts ranged approximately 42–259 g, and in the acute studies, between 22–56 g. In some studies, the quantity of nuts consumed was exhibited as a percentage of total diet energy and converted into g/day to compare studies (Claesson et al. 2009; Hollis and Mattes 2007). Two studies evaluated nut consumption on a calorie restriction diet (-200 kcal) (Alves, Moreira, Macedo, Costa et al. 2014; Alves, Moreira, Macedo, Alfenas et al. 2014). For the other studies, nuts were consumed as part of a habitual diet (Fraser et al. 2002; Hollis and Mattes, 2007; Claesson et al. 2009; Tey et al. 2011; Agebratt et al. 2016;). In three studies (Fraser et al. 2002; Hollis and Mattes 2007; Tey et al. 2011), a habitual diet free of nuts was considered the control group; in two studies, the control group was a calorie restriction diet (-250 kcal) free of nuts (Alves, Moreira, Macedo, Costa et al. 2014; Alves, Moreira, Macedo, Alfenas et al. 2014) (Table 1).

Outcomes evaluated

All studies included in this review evaluated REE, DIT, RQ, and nutrient oxidation ratios by indirect calorimetry. Despite the use of different protocols, all the studies followed the recommendations proposed by Compher et al. (2006). REE was measured in six studies (Claesson et al. 2009; Fraser et al. 2002; Hollis and Mattes 2007; Tey et al. 2011; Alves, Moreira, Macedo, Alfenas et al. 2014; Agebratt et al. 2016), DIT was evaluated in four studies (Casas-Agustench et al. 2009; Tapsell et al. 2009; Alves, Moreira, Macedo, Costa et al. 2014; Gepner et al. 2016), respiratory quotient was measured in seven studies (Fraser et al. 2002; Casas-Agustench et al. 2009;Tapsell et al. 2009; Alves, Moreira, Macedo, Costa et al. 2014; Alves, Moreira, Macedo, Alfenas et al. 2014; Gepner et al. 2016; Tey et al. 2011), while nutrient oxidation ratio was assessed in four studies (Casas-Agustench et al. 2009; Tapsell et al. 2009; Alves, Moreira, Macedo, Costa et al. 2014; Alves, Moreira, Macedo, Alfenas et al. 2014).

Risk of bias within studies

The risk of bias of the included studies was graphically represented in Figure 2. Most studies did not provide enough information about each domain for suitable judgement and were classified as "unclear". One study was assessed as "high risk of bias" regarding selective reporting of outcome (Tey et al. 2011). According to the classification proposed by Gomes et al. (2017) for the evaluation of study quality, all studies in the present review were classification as having "high risk of bias" because <50% of each item was classified as "low risk of bias".

Effects of acute nuts consumption on energy metabolism: Results of randomised clinical trials

Of the four acute studies, three evaluated the effects of walnut consumption on markers of energy metabolism. One study showed that the consumption of 22.1 g of walnuts within an isocaloric meal increased postprandial energy expenditure (PEE) and thermic effect of food (TEF) after five hours postprandial

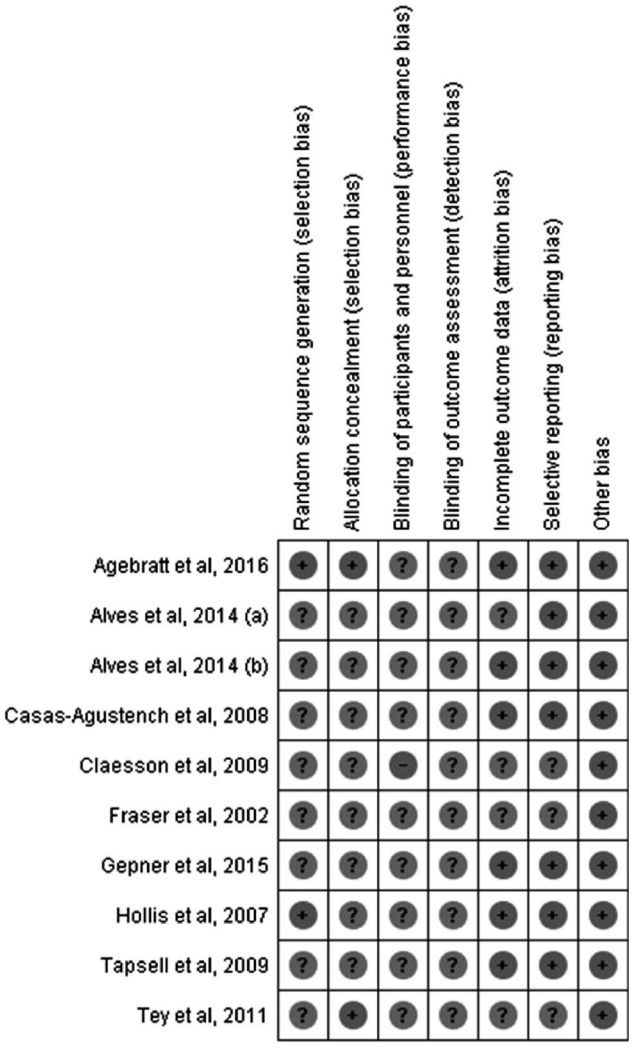

Figure 2. Risk of bias summary: review authors' judgments about each risk of bias item for each included study in the systematic review.? = unclear; + = low risk of bias; - = high risk of bias.

compared to consumption of an isocaloric diet rich in SFA in eutrophic men (Casas-Agustench et al. 2009). Furthermore, the consumption of 25–35 g of walnuts in an isocaloric diet decreased the RQ, carbohydrate oxidation, and increased fatty oxidation after eight hours postprandial compared to the consumption of olive oil in women and men with obesity (Tapsell et al. 2009). In contrast, one study reported that consumption of 56 g walnuts decreased TEF after 40 minutes postprandial compared to the consumption of 156 g of whole-grain bread in men with obesity (Gepner et al. 2016). Finally, one study observed that consumption of 56 g of high-oleic peanuts increased PEE and TEF after three hours postprandial compared to consumption of the same amount of conventional peanuts in overweight men (Alves, Moreira, Macedo, Costa et al. 2014).

Effects of chronic nuts consumption on energy metabolism: Results of controlled, randomised clinical trials

In all evaluated chronic studies, REE, RQ, and nutrient oxidation rates remained unchanged after daily nut intake for at least four weeks compared to the control group (Fraser et al. 2002; Hollis and Mattes 2007; Tey et al. 2011; Alves, Moreira, Macedo, Alfenas et al. 2014). In addition, all studies controlled possible bias that influences energy metabolism, such as food intake and physical activity practice throughout the follow-up.

In overweight men, the intake of 56 g of peanut irrespective of the cultivar (conventional or high-oleic peanut), for four weeks and within an energy-restricted diet (-250 kcal), did not change REE, RQ, or rate of nutrient oxidation compared to the control group (Alves, Moreira, Macedo, Alfenas et al. 2014). However, the intra-group comparison showed that peanut intake increased fat oxidation after follow-up for both groups compared to the baseline. Moreover, for the final intervention, a significant decrease in RQ was observed following conventional peanut intake. After 12 weeks, hazelnut intake (approximately 42 g/day) combined with a habitual diet did not change REE and RQ of overweight subjects compared to the consumption of a habitual diet free of nuts (Tey et al. 2011).

In another study with a crossover design (three weeks of washout), healthy women that consumed approximately 344.1 kcal of almonds combined with a habitual diet of ten weeks demonstrated no increase in REE compared to control (Hollis and Mattes 2007). Fraser et al. (2002) verified that REE and RQ remained unchanged after a daily intake of approximately 54,3 g of almonds for 24 weeks in overweight subjects. In this study, there was no washout period. For this reason, all participants started the study with a control period (24 weeks of a habitual nut free diet) followed by an intervention period (24 weeks of 54.3 g/day of almonds combined with a habitual diet).

Effects of chronic nuts consumption on energy metabolism: Results of uncontrolled, randomised clinical trials

The consumption of ~259 g of peanuts within a regular caloric diet increased REE compared to baseline after two weeks of intervention in eutrophic subjects (Claesson et al. 2009). Furthermore, the consumption of 81–91 g of a mix of nuts in the context of a habitual diet increased REE compared to baseline after

eight weeks of intervention also in eutrophics (Agebratt et al. 2016).

Discussion

As far as we know, the present systematic review is the first to gather the available evidence on the effect of acute and chronic consumption of nuts on energy metabolism markers. Studies suggest that acute walnut consumption appears to modulate energy metabolism markers differently. The consumption of 22.1 g of walnuts increased the postprandial energy expenditure and the thermic effect of the food, whereas the consumption of 25 to 35 g of walnuts decreased the respiratory quotient, carbohydrate oxidation, and increased fat oxidation. In contrast, the consumption of higher doses of walnuts (56 g) decreased the thermic effect of the food. In addition to the postprandial evaluation time, these studies also differed in terms of the comparison group and the profile of the evaluated subjects. One study also observed that the acute consumption of 56 g of high-oleic peanuts increased the postprandial energy expenditure and the thermic effect of the food compared to the consumption of conventional peanuts in line with the observed effects of the consumption of 22.1 g of walnuts.

Despite its high energy content, nut intake is associated with thermogenic effects based on nutritional characteristics such as high MUFA, PUFA, and protein (Agebratt et al. 2016; De Souza et al. 2017). According to the literature, incorporating foods with a high content of MUFA and PUFA into diet can increase energy metabolism. On the other hand, compared to saturated fatty acids, unsaturated fatty acids are more sensitive to oxidation due to chain length and degree of unsaturation (Alper and Mattes 2002; DeLany et al. 2000). Additionally, they require extra enzymatic steps (the two auxiliary enzymes, enoyl-CoA isomerase, and 2,4-dienoyl-CoA reductase) for the complete oxidation, which could contribute to the differences in the diet induced thermogenesis in comparison to saturated fats (Clevenger et al. 2014).

Furthermore, studies have shown that molecular mechanisms associated with energy metabolism can be modulated by MUFA and PUFA intake through stimulation of receptors activated by peroxisome proliferators (PPAR), mainly PPAR-α, and mitochondrial uncoupling protein (UCP). Once activated, PPAR-α and UCP can promote lipid oxidation and increase thermogenic response (Rosado et al. 2010; Castrejón-Tellez et al. 2016). The PPAR are nuclear receptors that regulate the expression of genes associated with fatty acid oxidation (PPARα and PPARδ), thermogenesis (PPARα and PPARδ) and fat synthesis and storage (PPARγ). Some preclinical evidence demonstrates PUFA has the greatest potential for binding to and activating PPAR compared with MUFA and SFA. Also, the type of PUFA can be relevant for energy metabolism modulation. In a study conducted by DeLany et al. (2000), a meal with a high content of α-linolenic acid (n-3 PUFA) resulted in the greatest fat oxidation compared with both oleic and linoleic acid high meals. As mentioned earlier, these mechanisms can at least partially support the results of the present study, once the highest impact on energy metabolism was observed after acute intake of walnut, which has a high content of PUFA, especially α-linolenic acid.

The potential effect observed between high-oleic peanuts and conventional peanuts could be strictly due to oleic acid content. Studies show that the consumption of foods with high content of this type of fat raises ethanolamide levels, a metabolite produced from oleic fatty acids in the intestine. After absorption, this metabolite modulates endogenous lipid metabolism by acting as a PPAR-α agonist, regulating fat absorption, lipolysis, β-oxidation, and lipid concentrations in the tissues and blood (Fu et al. 2003; Bowen et al. 2017). As previously reported, meals with high content of unsaturated fat appear to be metabolically beneficial to promote higher DIT and fat oxidation than meals high in saturated fat. This thermal effect seems to be influenced by oleic acid, suggesting that diets enriched with oleic acid can influence energy expenditure (Piers et al. 2002; Soares et al. 2004). Similarly, the consumption of foods with high-protein content can also increase REE and DIT due to mechanisms inherent to the physiological process of protein oxidation. This nutrient requires more energy for metabolic processes such as deamination, gluconeogenesis, and urea formation (Hermsdorff et al. 2007; Gilbert et al. 2011).

The evidence cited above supports the hypothesis that acute nut consumption could increase DIT and help control body weight. However, these effects were not observed after long-term nuts consumption such as almonds, hazelnuts, and peanuts of four to 24 weeks. Available evidence suggests no effect of chronic nuts consumption on REE and RQ compared to the habitual nut-free diet. Despite the absence of effects after chronic nuts consumption of energy metabolism markers in all randomised, controlled clinical trials, differences in methodology must be considered. Some studies analysed normal weight and overweight

Table 2. Nuts composition (100 g serving).

Nuts	Calories (kcal)	Total Fat (g)	SFA (g)	MUFA (g)	PUFA (g)	Protein (g)
Almonds	579	49.9	3.8	31.6	12.3	21.2
Brazil nuts	659	67.1	16.1	23.9	24.4	14.3
Cashews	553	43.8	7.78	23.8	7.84	18.2
Hazelnuts	628	60.8	4.46	45.7	7.92	15.0
Macadamia nut	718	75.8	12.1	58.9	1.5	7.91
Peanuts	570	49.6	7.1	22.3	17.2	26.2
Pecan	691	72.0	6.18	40.8	21.6	9.17
Pistachios	560	45.3	5.91	23.3	14.4	20.2
Walnuts	654	65.2	6.13	8.93	47.2	15.2

Composition of whole raw nuts based on USDA database (USDA Food Data Central 2021). SFA: Saturated fatty acids; MUFA: monounsaturated fatty acids; PUFA: polyunsaturated fatty acids.

subjects within the same experimental group (Hollis and Mattes 2007; Tey et al. 2011). Scientific evidence suggests a positive association between body mass index and energy metabolism (Carneiro et al. 2016; Thielecke et al. 1997). Thus, more elevated body weight consequently results in a more prominent REE (Westerterp 2017).

Likewise, the studies evaluated REE in different sexes within the same intervention groups without adjusting body composition (Fraser et al. 2002; Tey et al. 2011). It is well established that body composition differs between men and women. Physiologically, men and women have more lean and fat mass, respectively (Schorr et al. 2018). Also, subjects with low fat-free mass and high-fat mass may present different thermogenic responses than eutrophic or obese individuals with a higher proportion of fat free mass (Prado et al. 2014; Soares and Müller 2018). Therefore, differences in nutritional status and sex of individuals potentially influence the different responses in energy metabolism after nutritional interventions.

Interestingly, though with controversial results, most acute studies have investigated the effects of walnuts. At the same time, none of the chronic studies evaluated the isolated effect of walnut consumption, which makes us question whether chronic walnut consumption could modulate energy metabolism markers. Among all nuts, walnuts contain the highest amounts of PUFA (Ros 2010; USDA 2021) (Table 2). Another point that caught our attention is the different forms (raw or roasted, peeled or unpeeled, salted or not) of the nuts used in the studies and whether this could influence energy metabolism markers. As far as we know, no study has investigated the different forms of nuts in these markers. Studies have investigated the bioaccessibility of lipids present in different forms of almonds, but the results seem controversial (Mandalari et al. 2014, 2018). Despite this, almonds seem to confer potential cardioprotective effects and high satiety (Grundy et al. 2016).

Regarding the risk of bias, the studies were classified as "unclear" for almost all the assessed domains (Alves, Moreira, Macedo, Costa et al. 2014; Alves, Moreira, Macedo, Alfenas et al. 2014; Fraser et al. 2002; Hollis and Mattes 2007; Tey et al. 2011). Therefore, to better comprehend the effects of nut consumption on energy metabolism and possible mechanisms involved, we recommend the conduction of RCTs with greater control over biases.

We emphasise that this is the first review to critically evaluate the effects of chronic nut consumption on energy metabolism. Future studies should adjust methodologies to consider biases such as sex and body composition to help understand the effect of nut intake on energy metabolism and elucidate the physiological mechanisms involved.

The systematic review shows that chronic nuts consumption does not affect energy metabolism. Nevertheless, based on the proven health benefits attributed to nut intake, we highlight their inclusion in diets to promote a healthy and balanced dietary pattern. Previous systematic reviews have shown the benefit of nut consumption on lipid profile (Del Gobbo et al. 2015), insulin resistance (Tindall et al. 2019), inflammation and endothelial function (Neale et al. 2017), intestinal microbiota (Lamuel-Raventos and Onge 2017), and also in the reduced risk of cardiovascular disease, total cancer, and all-cause mortality, and mortality from respiratory disease, diabetes, and infections (Aune et al. 2016). Additionally, a systematic review of fifteen meta-analyses of observational studies showed a decreased risk of cardiovascular and cancer mortality, colon cancer, hypertension, and ischaemic stroke for higher nut consumption (Martini et al. 2021). Besides, nuts consumption is associated with increased satiety and reduced hunger, which can assist in body weight control (Akhlaghi et al. 2020). Beneficial effects of nuts consumption have also been described when in the

context of balanced diets (Chiavaroli et al. 2018; Del Bo' et al. 2019).

Conclusion

Walnuts and peanuts were the nuts studied in the acute studies, with different results in energy metabolism markers, which seem to depend on the dose, postprandial time, profile of the evaluated subjects, and control group. Acute consumption of 22.1 g of walnuts increased postprandial energy expenditure and thermic effect of food after five hours postprandial in eutrophic men, while higher doses (56 g walnuts) decreased thermic effect of food after forty minutes postprandial in men with obesity. On the other hand, moderate amounts (25–35 g of walnuts) decreased the respiratory quotient, carbohydrate oxidation, and increased fatty oxidation after eight hours postprandial in subjects with obesity. For peanuts, 56 g of high-oleic peanuts increased postprandial energy expenditure and thermic effect of food after three hours postprandial compared to consumption of the same amount of conventional peanuts in overweight men. On the other hand, almonds, hazelnuts, peanuts, and a mix of nuts were the nuts studies in the chronic studies. Thus, the chronic consumption of nuts does not seem to influence energy metabolism markers. However, due to different population characteristics, types, and doses of nuts, further studies are needed to elucidate the effects of nuts consumption on energy metabolism.

Acknowledgments

The authors acknowledge the Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES) for scholarship conceded to YMFE, APSC, and AS.

Disclosure statement

No potential competing interest was reported by the authors.

Funding

This work was supported by the Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES) – Finance Code 001 and the Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) Process 428038/2018-2. Brazil. JB is CNPq fellows. The funders had no role in the study design, data collection and analysis, decision to publish, or preparation of the article.

ORCID

Yuliana María Franco Estrada http://orcid.org/0000-0002-9765-0151
Ana Paula Silva Caldas http://orcid.org/0000-0002-7517-3323
Alessandra da Silva http://orcid.org/0000-0002-4188-2586
Josefina Bressan http://orcid.org/0000-0002-4993-9436

References

Agebratt C, Ström E, Romu T, Dahlqvist-Leinhard O, Borga M, Leandersson P, Nystrom FH. 2016. A randomized study of the effects of additional fruit and nuts consumption on hepatic fat content, cardiovascular risk factors and basal metabolic rate. PLoS One. 11(1):e0147149–13.

Ajith TA, Jayakumar TG. 2016. Peroxisome proliferator-activated receptors in cardiac energy metabolism and cardiovascular disease. Clin Exp Pharmacol Physiol. 43(7): 649–658.

Akhlaghi M, Ghobadi S, Zare M, Foshati S. 2020. Effect of nuts on energy intake, hunger, and fullness, a systematic review and meta-analysis of randomized clinical trials. Crit Rev Food Sci Nutr. 60(1):84–93.

Alper CM, Mattes RD. 2002. Effects of chronic peanut consumption on energy balance and hedonics. Int J Obes. 26(8):1129–1137.

Alves RDM, Moreira APB, Macedo VS, Costa NMB, Alfenas RCG, Bressan J. 2014. High-oleic peanuts increase diet-induced thermogenesis in overweight and obese men. Nutr Hosp. 29(5):1024–1032.

Alves RDM, Moreira APB, Macedo VS, Alfenas RCG, Bressan J, Mattes R, Costa NMB. 2014. Regular intake of high-oleic peanuts improves fat oxidation and body composition in overweight/obese men pursuing a energy-restricted diet. Obesity (Silver Spring)). 22(6):1422–1429.

Aune D, Keum N, Giovannucci E, Fadnes LT, Boffetta P, Greenwood DC, Tonstad S, Vatten LJ, Riboli E, Norat T. 2016. Nut consumption and risk of cardiovascular disease, total cancer, all-cause and cause-specific mortality: a systematic review and dose-response meta-analysis of prospective studies. BMC Med. 14(1):207.

Bowen KJ, Kris-Etherton PM, Shearer GC, West SG, Reddivari L, Jones PJH. 2017. Oleic acid-derived oleoylethanolamide: a nutritional science perspective. Prog Lipid Res. 67(8):1–15.

Bray G. 2012. Identification, evaluation, and treatment of overweight and obesity. In Obesity. pp. 47–64. CRC Press.

Bray GA, Heisel WE, Afshin A, Jensen MD, Dietz WH, Long M, Kushner RF, Daniels SR, Wadden TA, Tsai AG, et al. 2018. The science of obesity management: an endocrine society scientific statement. Endocr Rev. 39(2): 79–132.

Carneiro IP, Elliott SA, Siervo M, Padwal R, Bertoli S, Battezzati A, Prado CM. 2016. Is obesity associated with altered energy expenditure? Adv Nutr. 7(3):476–487.

Casas-Agustench P, López-Uriarte P, Bulló M, Ros E, Gómez-Flores A, Salas-Salvadó J. 2009. Acute effects of three high-fat meals with different fat saturations on

energy expenditure, substrate oxidation and satiety. Clin Nutr. 28(1):39–45.
Castrejón-Tellez V, Rodríguez-Pérez JM, Pérez-Torres I, Pérez-Hernández N, Cruz-Lagunas A, Guarner-Lans V, Vargas-Alarcón G, Rubio-Ruiz ME. 2016. The effect of resveratrol and quercetin treatment on PPAR mediated uncoupling protein (UCP-) 1, 2, and 3 expression in visceral white adipose tissue from metabolic syndrome rats. Int J Mol Sci. 17(7):1–13.
Chiavaroli L, Nishi SK, Khan TA, Braunstein CR, Glenn AJ, Mejia SB, Rahelić D, Kahleová H, Salas-Salvadó J, Jenkins DJA, et al. 2018. Portfolio dietary pattern and cardiovascular disease: a systematic review and meta-analysis of controlled trials. Prog Cardiovasc Dis. 61(1):43–53.
Claesson A-L, Holm G, Ernersson A, Lindström T, Nystrom FH. 2009. Two weeks of overfeeding with candy, but not peanuts, increases insulin levels and body weight. Scand J Clin Lab Invest. 69(5):598–605.
Clevenger HC, Kozimor AL, Paton CM, Cooper JA. 2014. Acute effect of dietary fatty acid composition on postprandial metabolism in women. Exp. Physiol. 99(9): 1182–1190.
Coates A, Hill A, Tan S. 2018. Nuts and cardiovascular disease prevention. Curr Atheroscler Rep. 20(10):48–49.
Compher C, Frankenfield D, Keim N, Roth-Yousey L, Evidence Analysis Working Group. 2006. Best practice methods to apply to measurement of resting metabolic rate in adults: a systematic review. J Am Diet Assoc. 106(6):881–903.
Del Bo' C, Marino M, Martine D, Tucci M, Ciappellano S, Riso P, Porrini M. 2019. Overview of human intervention studies evaluating the impact of the Mediterranean diet on markers of DNA damage. Nutrients. 11(2):391.
Del Gobbo LC, Falk MC, Feldman R, Lewis K, Mozaffarian D. 2015. Effects of tree nuts on blood lipids, apolipoproteins, and blood pressure: Systematic review, meta-analysis, and dose-response of 61 controlled intervention trials. Am J Clin Nutr. 102(6):1347–1356.
DeLany JP, Windhauser MM, Champagne CM, Bray GA. 2000. Differential oxidation of individual dietary fatty acids in humans. Am J Clin Nutr. 72(4):905–911.
Dulloo AG, Duret C, Rohrer D, Girardier L, Mensi N, Fathi M, Chantre P, Vandermander J. 1999. Efficacy of a green tea extract rich in catechin polyphenols and caffeine in increasing 24-h energy expenditure and fat oxidation in humans. Am J Clin Nutr. 70(6):1040–1045.
Flores-Mateo G, Rojas-Rueda D, Basora J, Ros E, Salas-Salvadó J. 2013. Nut intake and adiposity: meta-analysis of clinical trials. Am J Clin Nutr. 97(6):1346–1355.
Fraser GE, Bennett HW, Jaceldo KB, Sabaté J. 2002. Effect on body weight of a free 76 kilojoule (320 calorie) daily supplement of almonds for six months. J Am Coll Nutr. 21(3):275–283.
Fu J, Gaetani S, Oveisi F, Lo Verme J, Serrano A, Rodríguez De Fonseca F, Rosengarth A, Luecke H, Di Giacomo B, Tarzia G, et al. 2003. Oleylethanolamide regulates feeding and body weight through activation of the nuclear receptor PPAR-α. Nature. 425(6953):90–93.
Gepner Y, Bril N, Shelef I, Schwarzfuchs D, Serfaty D, Rein M, Cohen N, Shemesh E, Tangi-Rosental O, Sarusi B, et al. 2016. Higher visceral adiposity is associated with an enhanced early thermogenic response to carbohydrate-rich food. Clin Nutr. 35(2):422–427.
Gilbert JA, Bendsen NT, Tremblay A, Astrup A. 2011. Effect of proteins from different sources on body composition. Nutr Metab Cardiovasc Dis. 21 Suppl 2(10): B16–B31.
Gomes JMG, Costa JA, Alfenas RCG. 2017. Metabolic endotoxemia and diabetes mellitus: a systematic review. Metabolism. 68(10):133–144.
Grundy MML, Lapsley K, Ellis PR. 2016. **A** review of the impact of processing on nutrient bioaccessibility and digestion of almonds. Int J Food Sci Technol. 51(9): 1937–1946.
Hermsdorff HHM, Volp ACP, Bressan J. 2007. Macronutrient profile affects diet-induced thermogenesis and energy intake. Arch Latinoam Nutr. 57(1):33–42.
Higgins JPT, Altman DG, Gotzsche PC, Juni P, Moher D, Oxman AD, Savović J, Schulz KF, Weeks L, Sterne JAC. Cochrane Statistical Methods Group 2011. The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. BMJ. 343(2):d5928 (oct18
Hollis J, Mattes R. 2007. Effect of chronic consumption of almonds on body weight in healthy humans. Br J Nutr. 98(3):651–656.
Jéquier E, Tappy L. 1999. Regulation of body weight in humans. Physiol Rev. 79(2):451–480.
Lamuel-Raventos RM, Onge MPS. 2017. Prebiotic nut compounds and human microbiota. Crit Rev Food Sci Nutr. 57(14):3154–3163.
Mancini JG, Filion KB, Atallah R, Eisenberg MJ. 2016. Systematic review of the Mediterranean diet for long-term weight loss. Am J Med. 129(4):407–415.e4.
Mandalari G, Grundy MML, Grassby T, Parker ML, Cross KL, Chessa S, Bisignano C, Barreca D, Bellocco E, Laganà G, et al. 2014. **T**he effects of processing and mastication on almond lipid bioaccessibility using novel methods of *in vitro* digestion modelling and micro-structural analysis. Br J Nutr. 112(9):1521–1529.
Mandalari G, Parker ML, Grundy MML, Grassby T, Smeriglio A, Bisignano C, Raciti R, Trombetta D, Baer DJ, Wilde PJ. 2018. Understanding the effect of particle size and processing on almond lipid bioaccessibility through microstructural analysis: from mastication to faecal collection. Nutrients. 10(2):213.
Martini D, Godos J, Marventano S, Tieri M, Ghelfi F, Titta L, Lafranconi A, Trigueiro H, Gambera A, Alonzo E, et al. 2021. Nut and legume consumption and human health: an umbrella review of observational studies. Int J Food Sci Nutr. 1–8.
Martínez-González MA, Bes-Rastrollo M. 2011. Nut consumption, weight gain and obesity: epidemiological evidence. Nutr Metab Cardiovasc Dis. 21 Suppl 1:S40–S45.
Moher D, The PRISMA Group, Liberati A, Tetzlaff J, Altman DG, Group TP. 2009. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. PLoS Med. 6(7):e1000097–6.
Neale EP, Tapsell LC, Guan V, Batterham MJ. 2017. The effect of nut consumption on markers of inflammation and endothelial function: a systematic review and meta-analysis of randomised controlled trials. BMJ Open. 7(11):e016863–14.

Piers LS, Walker KZ, Stoney RM, Soares MJ, O'Dea K. 2002. The influence of the type of dietary fat on postprandial fat oxidation rates: Monounsaturated (olive oil) vs saturated fat (cream). Int J Obes. 26(6):814–821.

Prado CMM, Siervo M, Mire E, Heymsfield SB, Stephan BCM, Broyles S, Smith SR, Wells JCK, Katzmarzyk PT. 2014. A population-based approach to define body-composition phenotypes. Am J Clin Nutr. 99(6):1369–1377.

Ros E. 2010. Health benefits of nut consumption. Nutrients. 2(7):652–682.

Rosado EL, Bressan J, Martínez JA, Marques-Lopes I. 2010. Interactions of the PPARγ2 polymorphism with fat intake affecting energy metabolism and nutritional outcomes in obese women. Ann Nutr Metab. 57(3-4):242–250.

Schorr M, Dichtel LE, Gerweck AV, Valera RD, Torriani M, Miller KK, Bredella MA. 2018. Sex differences in body composition and association with cardiometabolic risk. Biol Sex Differ. 9(1):28–10.

Soares MJ, Cummings SJ, Mamo JCL, Kenrick M, Piers LS. 2004. The acute effects of olive oil v. cream on postprandial thermogenesis and substrate oxidation in postmenopausal women. Br J Nutr. 91(2):245–252.

Soares MJ, Müller MJ. 2018. Resting energy expenditure and body composition: critical aspects for clinical nutrition. Eur J Clin Nutr. 72(9):1208–1214.

Soltani S, Shirani F, Chitsazi MJ, Salehi-Abargouei A. 2016. The effect of dietary approaches to stop hypertension (DASH) diet on weight and body composition in adults: a systematic review and meta-analysis of randomized controlled clinical trials. Obes Rev. 17(5):442–454.

Souza RGM, Schincaglia RM, Pimentel GD, Mota JF. 2017. Nuts and human health outcomes: a systematic review. Nutrients. 9(12):1–23.

Spies CM, Straub RH, Buttgereit F. 2012. Energy metabolism and rheumatic diseases: from cell to organism. Arthritis Res Ther. 14(3):216–210

Tan SY, Dhillon J, Mattes RD. 2014. A review of the effects of nuts on appetite, food intake, metabolism, and body weight. Am J Clin Nutr. 100 Suppl 1(Suppl_1): 412S–422S.

Tapsell L, Batterham M, Tan SY, Warensjö E. 2009. The effect of a calorie controlled diet containing walnuts on substrate oxidation during 8-hours in a room calorimeter. J Am Coll Nutr. 28(5):611–617.

Tey SL, Brown R, Gray A, Chisholm A, Delahunty C. 2011. Nuts improve diet quality compared to other energy-dense snacks while maintaining body weight. J Nutr Metab. 2011(suppl_1):357350–357311.

Thielecke F, Moseneder J, Kroke A, Klipstein-Grobusch K, Boeing H, Noack R. 1997. Determination of total energy expenditure, resting metabolic rate and physical activity in lean and overweight people. Z Ernahrungswiss. 36(4): 310–312.

Tindall AM, Johnston EA, Kris-Etherton PM, Petersen KS. 2019. The effect of nuts on markers of glycemic control: a systematic review and meta-analysis of randomized controlled trials. Am J Clin Nutr. 109(2):297–314.

Tindall AM, Petersen KS, Lamendella R, Shearer GC, Murray-Kolb LE, Proctor DN, Kris-Etherton PM. 2018. Tree nut consumption and adipose tissue mass: mechanisms of action. Curr Dev Nutr. 2(11):nzy069–11.

USDA Food Data Central 2021. https://www.usda.gov/.

Valeria A, Castro B, Kolka CM, Kim SP, Bergman RN. 2014. Obesity, insulin resistance and comorbidities? Mechanisms of association. Arq Bras Endocrinol Metabol. 58(6):600–609.

Westerterp KR. 2017. Control of energy expenditure in humans. Eur J Clin Nutr. 71(3):340–344.

WHO. 2020a. Obesity and overweight. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight.

WHO 2020b. Obesity. https://www.who.int/health-topics/obesity#tab=tab_1.