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Effects of energy-restricted diets with or without nuts on weight, body composition and glycaemic control in adults: a scoping review

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Abstract

Energy-restricted (ER) diets promote weight loss and improve body composition and glycaemic control. Nut consumption also improves these parameters. However, less is known about the combined benefit of these two strategies. This scoping review implemented a systematic search of Medline, Embase and Scopus to identify randomised controlled trials evaluating the effect of ER diets with or without nuts on body mass, body composition and glycaemic control in adults. After reviewing titles and abstracts, twenty-nine full-text articles were screened, resulting in seven studies reported in eight papers that met the inclusion criteria. Energy restriction was achieved by prescribing a set energy target or reducing intake by 1000–4200 kJ from daily energy requirements. Interventions ranged from 4 to 52 weeks in duration and contained 42–84 g/d of almonds, peanuts, pistachios or walnuts. While all studies reported that energy restriction resulted in significant weight loss, the addition of nuts to ER diets demonstrated significantly greater weight loss in only approximately half of the included studies (4/7 studies). There was limited evidence to support additional benefits from nuts for body composition measures or glycaemic control. Although improvements in weight loss and glycaemia were not consistent when nuts were included in ER diets, no study revealed an adverse effect of nut consumption on health outcomes. Future studies could explore the effect of consuming different types and amounts of nuts, combined with various levels of energy restriction on weight, body composition and glycaemic control.

Key words: **energy restriction: glucose regulation: nuts: scoping review: weight loss**

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Introduction

Energy restriction is an effective tool for promoting weight loss[1]. Various guidelines for management of overweight and obesity recommend lifestyle intervention, including dietary modification to achieve energy restriction, for loss of weight and fat mass[2]. Multiple interventions have reported improved glycaemic control when following an energy-restricted (ER) diet and achieving 5% weight loss[3,4]. A 2017 review of fifty-eight weight loss trials involving patients with type 2 diabetes reported that for every 1 kg of body weight lost estimated haemoglobin A1c reduced by 0·1 percentage points[5,6]

Maintaining high diet quality in ER diets can be challenging as additional care must be taken to ensure the diet meets the recommended amount of essential nutrients and the optimal macronutrient composition of ER diets remains unclear[7,8] The inclusion of nutrient-rich foods, such as nuts, in ER diets can help achieve recommended intakes of essential nutrients. Nuts contain a broad range of health-promoting nutrients including protein, fibre, unsaturated fats, B group vitamins and magnesium[9]. However, nuts are an energy-dense food[10] and thus there may be some concern about including nuts when trying to achieve weight loss[11,12]. Encouragingly, several epidemiological studies have reported that the inclusion of nuts into a diet is associated with lower body weight[13,14] and less weight gain[15–17]. A systematic review and meta-analysis of nut-feeding trials with or without dietary isocaloric substitution instructions found no change in weight with nut consumption, although it excluded studies that used energy-restricted diets with or without nuts[18], and thus it is unclear whether the inclusion of nuts to ER diets alters weight loss success.

Many of the nutrients found in nuts are suggested to play a key role in promoting weight loss[19,20]. The rich source of protein and fibre promotes satiety, assisting with reducing the overconsumption of food[21]. Protein in nuts may also aid with maintaining muscle mass, which can be lost during weight loss, and limit the reduction in resting energy expenditure commonly associated with weight loss[21]. Compared with saturated fats, unsaturated fats in nuts may also promote weight loss by increasing fat oxidation and diet-induced thermogenesis[9,18]. Additionally, complex plant cell wall matrices in nuts have been proposed to limit the enzymatic degradation of nuts within the gastrointestinal tract, which leads to encapsulation of fat and thus

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a reduction in fat absorption, resulting in reduced energy availability[6,22].

Furthermore, regular nut consumption is proposed to promote glycaemic control due to their high fibre content[23] and fatty acid profile that been linked to better insulin sensitivity[24]. High amounts of monounsaturated fats (MUFA), protein and fibre in nuts delay gastric emptying which lowers postprandial glucose[25]. When nuts are consumed with individual foods[26,27] or meals with a high carbohydrate content[28], they reduce the glucose response and, over time, may aid in reducing insulin resistance. However, there is inconsistency in the literature on effects of nuts on glycaemic indices (glucose, insulin and HbA1c) from randomised controlled trials in populations described as generally healthy, or diagnosed with dyslipidaemia, metabolic syndrome or pre-diabetes[29]. Clarity is needed whether additional improvements in glycaemic control can be achieved through the addition of nuts to ER diets beyond benefits achieved due to energy restriction alone.

No review to date has evaluated the effect of ER diets with or without nuts on weight, body composition or glycaemic control. Thus, a scoping review with a systematic search was used to identify randomised controlled trials (RCTs) evaluating the effect of a nut-enriched ER diet on weight, body composition and glycaemic control in adults with overweight or obesity.

Review question

The main question of this review is:

Does the inclusion of nuts in an ER diet impact anthro pometric measures and glycaemic control in adults with overweight or obesity?

Method and materials

The scoping review was registered with Open Science Framework (registration DOI 10·17605/OSF.IO/HETDJ) and performed according to the JBI methodology for scoping reviews[30,31]. The completed Preferred Reporting Items for Systematic Reviews and Meta Analysis (PRISMA) extension for Scoping Reviews checklist can be found in Supplementary Material A[32]. The review involved five stages: (1) review questions were established; (2) relevant studies were identified; (3) eligible studies were included in the review; (4) information from eligible studies were collated; (5) study information was summarised to answer the review question.

Eligibility criteria

This review considered RCTs that included adults (>18 years), with overweight (BMI 25·0–29·9 kg/m^2) or obesity (≥30·0 kg/m^2) with or without chronic diseases (including type 1 and type 2 diabetes) who had participated in a weight loss dietary intervention.

Studies were evaluated if the intervention included an ER diet with nuts and assessed body mass and glucose via one of the following measures: venous glucose, fasting capillary blood glucose, or interstitial glucose obtained via a venous blood draw, finger prick, flash glucose monitor (FGM) or continuous glucose monitor (CGM). To be included in the review, studies had to report on both body mass and fasting glucose; however, we also extracted data on body mass index (BMI), body composition (fat mass, per cent fat mass, fat-free mass, per cent fat-free mass, lean mass and per cent lean mass), fasting insulin, estimated haemoglobin A1c (HbA1c) and insulin resistance (homeostatic model of assessment of insulin resistance (HOMA-IR) and homeostatic model of assessment of pancreatic β-cell function (HOMA-β)). The methods/technology used to assess body mass, body composition and glycaemic control (capturing glucose, insulin and insulin resistance) were also extracted.

All ER diets contained at least one type of tree nut or peanut in a whole, chopped or paste form. No restrictions were set for the time to achieve weight loss, or the amount of tree nuts/peanuts consumed. Studies were excluded if ER diets contained nut oil or nuts combined with another food or as part of a dietary pattern, where the exclusive effect of the nut consumption could not be determined.

Types of studies

This scoping review considered only RCTs; observational studies, non-intervention studies, opinion pieces, conference abstracts and non-human cell culture studies excluded. No date restrictions were used in the database search. Studies published in languages other than English or not in a peer-reviewed journal were excluded from the review.

Search strategy

An initial search limited to Medline and Embase was conducted to identify articles on the topic. Keywords and subject headings (MeSH and Emtree) found in the titles and abstracts of relevant studies were used to develop a search strategy. Search terms and subject headings were adapted for specific databases and reviewed by an academic librarian at the University of South Australia. The full search strategies are shown in Supplementary Material B. The search was conducted on 12 April 2021 and re-run on 25 January 2022 in online databases Medline, Scopus and EMBASE.

Study selection

Following the search, duplicates were removed before screening by title and abstract in Covidence[33] by two investigators (L.C.M. and A.M.H., A.M.C. or S.C. who evenly divided the role of the second investigator). Full-text screening was performed by L.C.M. and A.M.H. Any disagreements in title/abstract and full-text screening were resolved by a third investigator (A.M.C or S.C).

Data extraction

A data extraction table was developed and piloted on two studies by L.C.M.; data were double checked by A.M.C., A.M.H. and S.C. Data for all studies were then extracted by L.C.M. The data extracted included author(s), year of publication, country, population characteristics (the number of participants randomised to the intervention/control, age, gender, health status), intervention and control characteristics (duration, amount and

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type of nut or comparator food, level of energy restriction and description of diet composition, physical activity/behaviour education, compliance monitoring) and method/technology used to assess body mass, body composition and glycaemic control. Extracted outcome data included pre–post intervention and change in body mass, BMI, body composition (fat mass, per cent fat mass, fat-free mass, per cent fat-free mass, lean mass and per cent lean mass) and glycaemic control (fasting glucose, fasting insulin, interstitial glucose, capillary blood glucose, HbA1c, HOMA-IR, HOMA-β).

Data analysis and presentation

Effect sizes (Cohen's *d*) were calculated for each study. In instances where an effect size could not be calculated as pre, post or change values were not provided, authors were contacted to provide data. Effect sizes were not calculated if values were reported as least-squares mean. A narrative summary accompanies the tabulated results and describes how the results relate to the review objectives.

Results

Study inclusion

The review process was conducted as shown in Fig. 1. The initial search retrieved 859 papers. After 166 duplicates were removed, 693 studies were title/abstract screened (666 studies excluded). A total of twenty-seven studies were full text screened to determine eligibility; twenty studies were excluded, resulting in six studies reported in seven papers. An additional study was identified after re-searching Medline, Embase and Scopus in January 2022. The final number of studies eligible for inclusion in the review was seven, reported in eight papers.

The review includes two papers that reported data from the same study[34,35]; both papers reported body mass, BMI, fat mass and lean mass, whilst per cent fat mass and per cent lean mass were reported in Alves *et al.*[34] and glycaemic control (glucose, insulin and HOMA-IR) was reported in Moreira Alves *et al.*[35]. Of the eight papers included in this review, one paper reported an intention-to-treat and a compliers analysis[36], three papers reported only an intention-to-treat analysis[34,37,38] and four papers reported a completers analysis[35,39–41].

Characteristics of included studies

Population. Characteristics of the seven studies included in this review are presented in Table 1. Study publication dates ranged from 2003 to 2022. Studies were conducted in North America[36–38,40], Brazil[35,41,42] and Iran[39]. Across all studies, 676 participants aged between 18 and 79 years were included. Male and female participants were enrolled in three studies[36,38,40] whilst four studies (five papers) included males[35,42] or females[37,39,41] only. All studies included participants with overweight or obesity. Most studies enrolled healthy participants without established chronic disease except for one study that included participants with type 2 diabetes and hypertension[38]. Rock *et al.* reported body mass data from the intervention and control groups for insulin-sensitive (IS) and insulin-resistant (IR) participants separately[37].

Intervention design. All studies used parallel designs with four studies[36,38–40] structured as a two-arm design and three studies (four papers) as a three-arm design[34,35,37,41]. Study duration ranged from 4 to 52 weeks with three studies of 12 weeks[36,39,40]. The prescribed diets provided participants with energy deficits (to achieve weight loss) that ranged from 1046 to 4184 kJ less than the participants' total daily energy requirements[34–37,39,40]. For one study[41] energy restriction was prescribed as a reduction of 1046 kJ less than the group mean energy intake. Wien *et al.*[38] reported the absolute caloric amount provided by an almond-enriched and a carbohydrate enriched low-calorie diet (4234 kJ and 4246 kJ, respectively) but did not report the level of caloric restriction that corresponded with these amounts compared with habitual diets. All ER diets were designed to achieve the desired energy levels either by including nuts for intervention arms or being devoid of nuts for control arms, and most studies used a healthy whole food diet apart from one study that instructed participants to consume a meal replacement (protein formula) daily along with a healthy whole food diet[38]. Daily nut consumption ranged from 38 to 84 g (providing ~908–2004 kJ/217–479 kcal) in the nut-enriched diets. The type of nuts included in each study was almonds[36,38,39], peanuts (whole peanuts with skins left on (WP), whole peanuts with skins removed (SP)[41], conventional peanuts (CVP) and high-oleic peanuts (HOP))[34,35], pistachio nuts[40] and walnuts[37]. Nut form was reported in only five studies where nuts were salted[36,40], unblanched[38] or dry-roasted[36,41]. All participants were unblinded to the treatment they received as whole nuts were used.

The planned macronutrient composition of nut-enriched and nut-free diets was reported in all but one study[36] (Table 1). Three studies (four papers) matched the overall macronutrient composition of intervention and control diets[34,35,39,41], one study matched on energy from protein but had different amounts of energy from fat and carbohydrate between diets (the nut-enriched diet had higher energy from fat)[40], one study used two comparator diets ((1) a low-carbohydrate diet that matched the macronutrient energy distribution of the nut-enriched diet, and (2) a low-fat diet with higher carbohydrate, lower protein and lower fat energy contributions than the nut-enriched diet)[37] and the final study used an extremely low-fat comparator diet[38].

Methods to assess weight, BMI, body composition and glycaemic control

Body mass (reported as weight) was measured using electronic scales[36,39,40] and bioelectrical impedance analysis (BIA)[38,41] (Table 1). Rock *et al.*[37] mentioned scales were used to record body mass, but it was unclear what type and brand these were. The technology used to measure body mass was not mentioned in two studies (three papers)[34,35,41]. Body mass index was calculated in five studies (six papers)[34,35,38–41] using measures of height and weight (kg/m^2). Fat mass (kg and %) was measured in three studies (four papers) using BIA[34,35] or dual-energy X-ray absorptiometry (DEXA)[36,38]. De Oliveira Fialho *et al.*[41]

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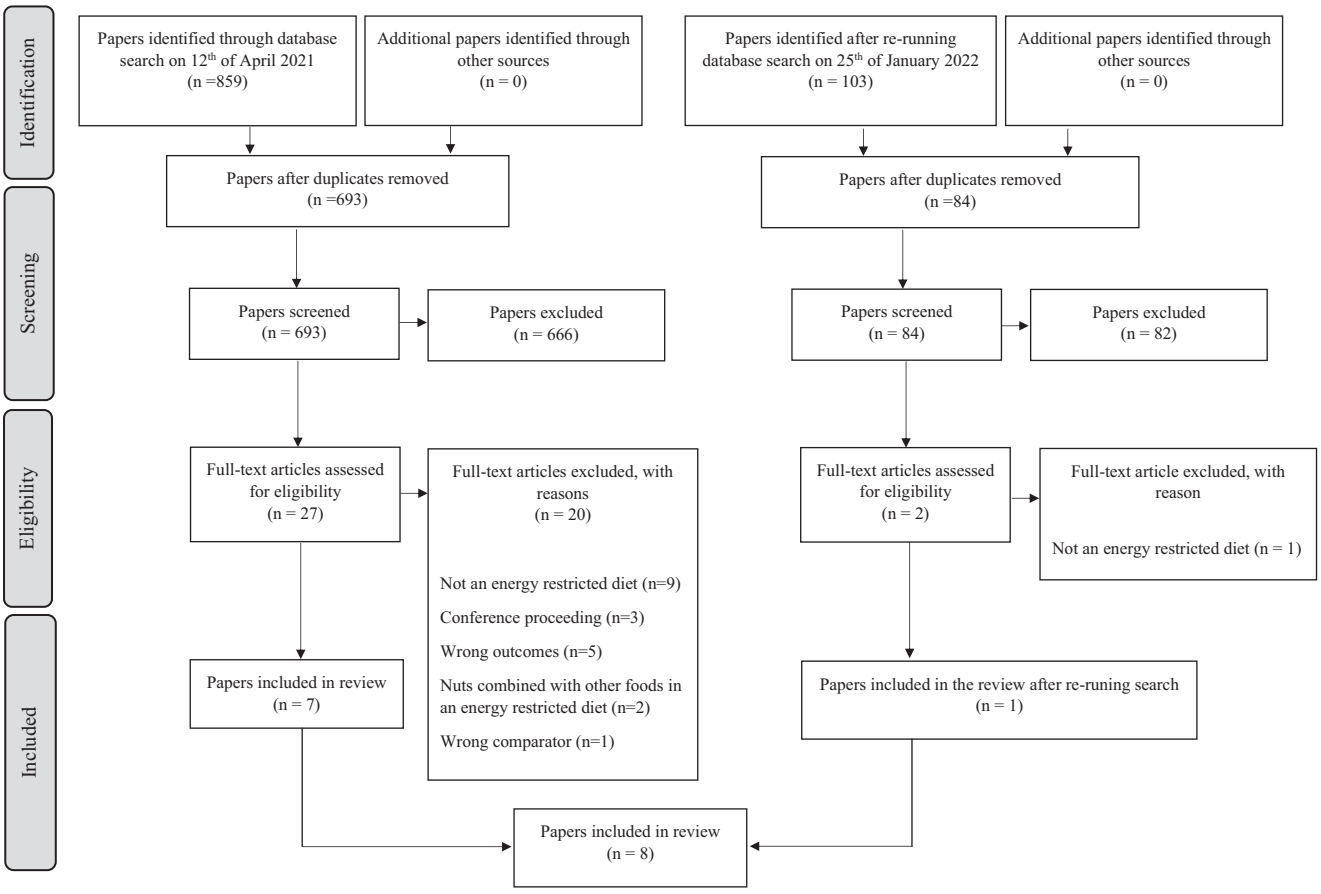

Fig. 1. PRISMA flowchart.

also measured total fat mass using BIA and reported per cent fat mass. Lean mass (kg and %) was reported in one study (two papers)[34,35] using DEXA, and fat-free mass (kg and %) was reported in two studies using BIA[38] or DEXA[36]. De Oliveira Fialho *et al.*[41] similarly measured lean mass using BIA which was reported as per cent lean mass.

Venous blood samples were obtained in all studies to measure fasting glucose and insulin[35–41]. Fasting serum glucose was the most commonly reported assessment of fasting glucose[35,36,40] followed by fasting plasma glucose[39] (three studies did not state the type of fasting blood sample collected)[37,38,41]. No studies measured HbA1c or glucose with techniques such as interstitial glucose or capillary blood glucose via continual glucose monitoring, flash glucose monitoring or finger pricks.

Insulin resistance was determined using the HOMA-IR calculator formulas ((fasting glucose, mmol/l) × (fasting insulin, mIU/l))/22·5[37] and ((fasting insulin, pM × fasting glucose, mM /22·5)[38]. Moreira Alves *et al.*[35] and de Oliveira Fialho *et al.*[41] determined insulin resistance using the formula (insulin (mU/l)/22·5$e^{-\ln \text{ glucose (mmol/l)}}$) as mentioned in Mathews *et al.*[43]. HOMA-β was calculated in one study[37] from measures of glucose and insulin (no formula mentioned).

Effect of intervention on anthropometry

Weight and BMI. Anthropometric outcomes are presented in Table 2 and Supplementary Material C. Four studies found significantly greater weight loss after a nut-enriched ER diet compared with an ER diet without nuts[37–39,41]. In two of these studies, these differences were only observed for certain comparisons; insulin-sensitive but not insulin-resistant participants in the study by Rock *et al.*[37] and for participants consuming whole peanuts but not skinned peanuts in the study by de Oliveira Fialho *et al.*[41]. All studies found significant weight loss over time, with the magnitude of weight loss achieved between ~1·5 and 3·7 kg for studies up to 12 weeks[34–36,39–41], ~19·5 kg for a 24-week study[38] and ~8·1 kg for a year-long study[37].

There were five studies (six papers) that reported BMI[34,35,38–41]. Of these five studies, all reported significant reductions in BMI over time. However, four reported a significantly greater reduction in BMI after an ER diet with nuts compared with an ER diet without nuts[38–41].

Total fat mass and per cent total fat mass. Fat mass was reported in four studies (five papers)[34–36,38,41]: two studies

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Table 1. Summary of study characteristics

Author, year, country	Study design, (length of intervention)	Number, (age), sex, health characteristics	Intervention: • Number randomised, energy restriction (kJ + kcal), diet composition, test food (g/d), PA recommendation, education provided (nutrition/behaviour), compliance monitoring	Control:	Outcomes
Abazarfard *et al.*[39], 2014, Iran	Randomised, non-blinded, two-arm parallel study, (12 weeks)	*N* = 108, (20–55 years), 0 M/108 F, People with overweight or obesity	○ *n* = 54 • 4184 kJ (1000 kcal) reduction from TDER • 54% carbohydrate, 16% protein, 30% fat • 50 g/d raw almonds as (2 × 25 g snacks) • Encouraged to walk (medium speed) 30 min/d • Healthy nutrition, stimulus control and self-monitoring • Diet compliance via 24-h recall every 15 d	○ *n* = 54 • 4184 kJ (1000 kcal) reduction from TDER • 54% carbohydrate, 16% protein, 30% fat • Nut-free, fat (sunflower + corn oil) + meat replaced nuts • Encouraged to walk (medium speed) 30 min/d • Healthy nutrition, stimulus control and self-monitoring • Diet compliance via 24-h recall every 15 d	Anthropometry - Weight (kg) - BMI (kg/m^2) Glycaemic control - Glucose*
Alves *et al.*[34] and Moreira Alves *et al.*[35] 2014, Brazil	Randomised, non-blinded, three-arm parallel study (4 weeks)	*N* = 76, (18–50 years), 76 M/0 F, People with overweight or obesity	○ CVP *n* = 24, HOP *n* = 27 • 1046 kJ (250 kcal) reduction from TDRE • 55% carbohydrate, 15% protein, 30% fat • 56 g/d CVP or HOP • Encouraged to maintain usual PA level • Diet counselling weekly at clinic appointments • Diet compliance via 3-d food records at baseline and 4 weeks	○ *n* = 25 • 1046 kJ (250 kcal) reduction from TDRE • 55% carbohydrate, 15% protein, 30% fat • No control test food; nut-free with energy from nuts offset in the balance of the diet • Encouraged to maintain usual PA level • Diet counselling weekly at clinic appointments • Diet compliance via 3-d food records at baseline and 4 weeks	Anthropometry - Weight (kg) - BMI (kg/m^2) - FM (kg, %) - LM (kg, %) Glycaemic control - Glucose* - Insulin† - HOMA-IR
de Oliveira Fialho *et al.*[41], 2022 Brazil	Randomised, non-blinded, three-arm parallel study (8 weeks)	*N* = 24, (25–41 years), 0 M/26 F, People with obesity	○ WP *n* = 8, SP *n* = 8 • 1046 kJ (250 kcal) reduction from customary average caloric intake of the WP or SP group • 45–65% carbohydrate, 10–35% protein, 20–35% fat • 56 g/d roasted WP or SP • No PA recommendations mentioned • Provided with self-selected exchange food list • Peanut portions + compliance questions provided weekly • Daily peanut intake captured in diet records weeks 4 + 8	○ *n* = 8 • 1046 kJ (250 kcal) reduction from customary average caloric intake of the control group • 45–65% carbohydrate, 10–35% protein, 20–35% fat • No peanuts or other nuts, energy from nuts offset in the balance of the diet • No PA recommendations mentioned • Provided with self-selected exchange food list • No compliance monitoring mentioned	Anthropometry - Weight (kg) - BMI (kg/m^2) - FM (%) - LM (%) Glycaemic control - Glucose* - Insulin† - HOMA-IR
Dhillon *et al.*[36], 2016 United States	Randomised, non-blinded, two-arm parallel study (12 weeks)	*N* = 86, (18–60 years), 21 M/65 F, People with overweight or obesity	○ *n* = 43 • 2092 kJ (500 kcal) reduction from TDRE • Target diet composition not reported • 15% E from almonds, (mean 38 g/d) dry-roasted, salted almonds • Encouraged to maintain usual PA level • Diet counselling using the MyPlate food guidance system; weekly for the first five weeks followed by every second week • Weekly 24-h recalls used to monitor compliance	○ *n* = 43 • 2092 kJ (500 kcal) reduction from TDRE • Target diet composition not reported • No control test food, no nuts, seeds or nut-containing products • Encouraged to maintain usual PA level • Diet counselling using the MyPlate food guidance system; weekly for the first five weeks followed by every second week • Weekly 24-h recalls used to monitor compliance	Anthropometry - Weight (kg) - FM (kg, %) - FFM (kg, %) Glycaemic control - Glucose* - Insulin‡
Li *et al.*[40], 2010 United States	Randomised, non-blinded, two-arm parallel study (12 weeks)	*N* = 70, (20–65 years), 13 M/57 F, People with overweight or obesity	○ *n* = 36 • 2092 kJ (500 kcal) reduction from TDER • 55% carbohydrate, 15% protein, 30% fat • 53 g/d salted pistachios (84 g with shell) as afternoon snack • No PA recommendations mentioned • Provided meal plans meeting required energy plus dietetic counselling every 2 weeks • ER assessed against a daily food intake checklist	○ *n* = 34 • 2092 kJ (500 kcal) reduction from TDRE • 65% carbohydrate, 15% protein, 20% fat • 56 g/d salted pretzels as afternoon snack • No PA recommendations mentioned • Provided meal plans meeting required energy plus dietetic counselling every 2 weeks • ER assessed against a daily food intake checklist	Anthropometry - Weight (kg) - BMI (kg/m^2) Glycaemic control - Glucose* - Insulin†

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Table 1. (*Continued*)

Author, year, country	Study design, (length of intervention)	Number, (age), sex, health characteristics	Intervention: • Number randomised, energy restriction (kJ + kcal), diet composition, test food (g/d), PA recommendation, education provided (nutrition/behaviour), compliance monitoring	Control:	Outcomes
Rock *et al.*[37], 2016 United States	Randomised, non-blinded, three-arm parallel study (52 weeks)	*N* = 245, (22–72 years), 0 M/245 F, People with overweight or obesity	○ *n* = 82 • 2092–4184 kJ (500–1000 kcal) reduction from TDRE • 45% carbohydrate, 20% protein, 35% fat • Mean 42 g/d walnuts (18% E from walnuts prescribed) • Aim for at least 60 min/d of moderate-intensity exercise • Detailed diet description, meal plans plus regular dietetic email/phone call follow-up • Group-based behavioural education (self-monitoring goal setting, preventing relapse, modifying problematic thoughts about weight, food +PA) • Red blood cell α-linolenic acid	○ LF *n* = 82, LC *n* = 81 • Both diets: 2092–4184 kJ (500–1000 kcal) reduction from TDRE • LF diet: 65% carbohydrates, 15% protein. 20% fat • LC diet: 45% carbohydrates, 20% protein, 35% fat • No nuts and no comparator snack • Aim for at least 60 min/d of moderate-intensity exercise • Detailed diet description, meal plans plus regular dietetic email/phone call follow-up • Group-based behavioural education (self-monitoring goal setting, preventing relapse, modifying problematic thoughts about weight, food + PA) • Red blood cell α-linolenic acid	Anthropometry - Weight (kg) Intervention and control groups reported as responses in people who were IS and IR Glycaemic control - Glucose* - Insulin† - HOMA-IR - HOMA-β
Wien *et al.*[38], 2003 United States	Randomised, non-blinded, two-arm parallel study (24 weeks)	*N* = 65, (27–79 years) 28M/37 F, People with overweight or obesity with/ without type 2 diabetes or hypertension	○ *n* = 32 • Total energy provided was 4234 kJ (1012 kcal) but degree of restriction not stated • 32% carbohydrate, 29% protein, 39% fat • Low-calorie meal replacement • 84 g/d unsalted, unblanched almonds • No exercise during weeks 1–4 then walk for 20–30 min 3–5 times per week • Weekly dietetic counselling, nutrition and behaviour modification classes • Compliance monitored through daily food and exercise logs reviewed weekly by dietitian	○ *n* = 33 • Total energy provided was 4246 kJ (1015 kcal) but degree of restriction not stated • 53% carbohydrate, 29% protein, 18% fat • Low-calorie meal replacements • No control test food; energy from almonds replaced with complex carbohydrates +2 tsp safflower oil to meet essential fatty acid requirements • No exercise during weeks 1–4 then walk for 20–30 minutes 3–5 times per week • Weekly dietetic counselling, nutrition and behaviour modification classes • Compliance monitored through daily food and exercise logs reviewed weekly by dietitian	Anthropometry - Weight§ - BMI (kg/m^2) - Fat mass§ - Fat-free mass§ Glycaemic control - Glucose* - Insulin† - HOMA-IR

kJ, kilojoules; g/d, grams per day; PA, physical activity, *N*, number of completed participants; M, number of males; F, number of females; kg, kilograms; BMI, body mass index; kg/m^2, kilogram per square metre; TDRE, total daily energy requirement; %, per cent; CVP, conventional peanut; HOP, high-oleic peanut; FM, fat mass; LM, lean mass; HOMA-IR, homeostatic model of assessment for insulin resistance; WP, whole peanut; SP, skinned peanut; FFM, fat-free mass; WR, walnut-rich diet; LF, low-fat/high-carbohydrate diet; LC, low-carbohydrate/high-fat diet; IS, insulin-sensitive; IR, insulin-resistant; HOMA-β, homeostatic model of assessment of β cell function; ER, energy restriction.
* Data were reported as milligram per decilitre but converted to millimole per litre.
† Data were reported as micro-unit per millilitre but converted to picomole per litre.
‡ Data were reported as milligram per decilitre but converted to picomole per litre.
§ Data were reported as pound but converted to kilogram.

(three papers) reported both absolute fat mass and per cent fat mass[34–36] whilst the other two studies reported only absolute fat mass[38] or only per cent fat mass[41].Only one study[38] found a significantly greater reduction in total fat mass with nuts compared with the nut-free ER diet. The majority of these studies found significant reductions in total fat mass over time in all groups, with the exception of the study reported by Alves *et al.*[34] and Moreira Alves *et al.*[35], where the reduction observed in the nut-free group did not reach significance, but this was not sufficient to generate a group by time difference.

From the three studies that reported per cent total fat mass, two studies[36,41] found a significant difference between groups, with Dhillon *et al.* reporting a greater reduction in the almond-enriched ER diet group compared with the nut-free ER diet group[36]. Comparatively, de Oliveira Fialho *et al.* found a greater reduction after an ER diet without nuts compared with a skinned peanut-enriched ER diet, noting that the skinned peanut group had a higher per cent fat mass at baseline[41]. All three studies that reported per cent total fat mass found a significant reduction over time[34,36,41].

Fat-free mass and per cent fat-free mass. There were two studies that reported fat-free mass, and both found small but significant reductions following weight loss; however, there were no differences between those consuming an ER diet with or without nuts[36,38]. Dhillon *et al.*[36] was the only study to record

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Table 2. Changes in body weight and body composition over time and between groups

Author, year	Technology/ method used to assess weight or body composition	Results: Within-group effect (time) Intervention:	Control:	Between-group effect (intervention-control)
Abazarfard *et al.*[39], 2014	Weight: SECA electronic scales BMI: Weight/height (kg/m^2)	Weight −3·7 kg reduction (p<0·001), (mean [SD], pre 76·4 [2·7] kg, post 72·7 [4·2] kg) BMI −1·5 kg/m^2 reduction ($p < 0·001$), (mean [SD], pre 29·9 [1·2] kg/m^2, post 28·5 [1·4] kg/m^2)	Weight −1·3 kg reduction ($p = 0·016$), (mean [SD], pre 75·6 [2·4] kg, post 74·3 [4·3] kg) BMI −0·5 kg/m^2 reduction ($p = 0·018$), (mean [SD], pre 29·4 [1·7] kg/m^2, post 28·9 [2·1] kg/m^2)	Weight Greater weight loss following intervention compared with control (difference in weight loss: −2·4 kg, $p < 0·001$, $d = 0·63$) BMI Greater reduction in BMI following intervention compared with control (difference in BMI: −1·0 kg/m^2), ($p < 0·001$), ($d = 0·63$)
Alves *et al.*[34] and Moreira Alves *et al.*[35] 2014	Weight: Brand not reported BMI: Weight/height (kg/m^2) Total fat mass (kg, %) and lean mass (kg, %): GE-Luna Prodigy DEXA	Weight CVP: −1·5 kg reduction ($p < 0·05$)* HOP: −1·7 kg reduction ($p < 0·05$)* BMI CVP: −0·5 kg/m^2 reduction ($p < 0·05$)* HOP: −0·6 kg/m^2 reduction ($p < 0·05$)*	Weight −2·2 kg reduction ($p < 0·05$)* BMI −0·7 kg/m^2 reduction ($p < 0·05$)*	Weight No significant difference ($p > 0·05$). (CVP and control: difference in weight loss: 0·7 kg), ($d = −0·52$) (HOP and control: difference in weight loss: 0·5 kg) ($d = −0·34$) BMI No significant difference ($p > 0·05$). (CVP and control: difference in BMI: 0·2 kg/m^2) ($d = −0·49$). (HOP and control: difference in BMI: 0·1 kg/m^2) ($d = −0·31$)
		Total fat mass CVP: −1 kg reduction ($p < 0·05$)* HOP: −1·4 kg reduction ($p < 0·05$)* Total lean mass CVP: −0·4 kg no significant change ($p > 0·05$)* HOP: −1·9 kg no significant change ($p > 0·05$)* Percent total fat mass CVP: −0·6% no significant change ($p > 0·05$)* HOP: −1% reduction ($p < 0·05$)* Percent total lean mass CVP: −0·6% no significant change ($p > 0·05$)* HOP: 0·9% increase ($p < 0·05$)*	Total fat mass −0·8 kg no significant change ($p > 0·05$)* Total lean mass −1·3 kg reduced ($p < 0·05$)* Percent total fat mass −0·1% no significant change ($p > 0·05$)* Percent total lean mass −0·03% no significant change ($p > 0·05$)*	Total fat mass No significant difference ($p > 0·05$). (CVP and control: difference in TFM: −0·2 kg) ($d = 0·16$) (HOP and control: difference in TFM: −0·6 kg) ($d = 0·43$) Total lean mass No significant difference ($p > 0·05$). (CVP and control: difference in TLM: 0·9 kg) ($d = −0·56$) (HOP and control: difference in TLM: −0·6 kg) (HOP and control: $d = −0·78$) Percent total fat mass No significant difference ($p > 0·05$). (CVP and control: difference in TFM %: −0·5%) ($d = 0·40$) (HOP and control: difference in TFM%: −0·9%) ($d = 0·67$) Percent total lean mass No significant difference ($p > 0·05$) (CVP and control: difference in TLM%: −0·57% ($d = 0·36$) (HOP and control: difference in TLM%: 0·93%) ($d = 0·62$)
de Oliveira Fialho *et al.*[41], 2022	Weight: Brand not reported BMI: Weight/height (kg/m^2) Total fat mass (%) and lean mass (%): BIA, Biodynamics model 310	Weight WP: −3·2 kg reduction ($p = 0·005$), (mean [SD], pre 84·2 [7·7] kg, post 81·0 [8·5] kg) SP: −2·6 kg reduction ($p = 0·005$), (mean [SD], pre 87·0 [14·0] kg, post 84·4 [14·2] kg) BMI WP: −1·1 kg/m^2 reduction ($p = 0·002$), (mean [SD], pre 32·3 [2·0] kg/m^2, post 31·2 [2·4] kg/m^2) SP: -1·2 kg/m^2 reduction ($p = 0·002$), (mean [SD],	Weight −1·8 kg reduction ($p = 0·004$), (mean [SD], pre 83·7 [8·9] kg, post 81·9 [9·9] kg) BMI −0·7 kg/m^2 no significant change ($p = 0·074$), (mean [SD], pre 32·8 [2·5] kg/m^2, post 32·1 [2·5] kg/m^2) Percent total fat mass −2·9% reduction ($p = 0·018$), (mean [SD], pre 38·4 [2·6] %, post 35·5 [3·1] %)	Weight Greater weight loss WP and control ($p > 0·05$), (difference in weight loss: −1·4 kg) ($d = 0·41$) No difference between following SP compared with control ($p < 0·05$), (difference in weight loss: −0·8 kg), ($d = 0·28$) BMI Greater reduction in BMI following WP compared with control (p<0·05), (difference in BMI: -0·4 kg/m^2), ($d = 0·39$) No difference between SP and control ($p > 0·05$), (difference in BMI: −0·5 kg/m^2) ($d = 0·33$)

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Table 2. (*Continued*)

Author, year	Technology/ method used to assess weight or body composition	Results: Within-group effect (time) Intervention:	Control:	Between-group effect (intervention-control)
		pre 33·6 [3·8] kg/m^2, post 32·4 [3·0] kg/m^2) Percent total fat mass WP: −1·3% reduction ($p = 0·018$), (mean [SD], pre 38·0 [3·8] %, post 36·7 [4·1] %) SP: −2·40% reduction ($p = 0·008$), (mean [SD], pre 43·2 [3·3] %, post 40·8 [3·8] %) Percent total lean mass WP: 2·9% increase ($p = 0·001$), (mean [SD], pre 61·8 [1·6] %, post 64·7 [2·2] %) SP: 1·9% increase ($p = 0·001$), (mean [SD], pre 57·8 [2·1] %, post 59·7 [2·6] %)	Percent total lean mass 2·2% increase ($p = 0·000$), (mean [SD], pre 61·5 [1·5] %, post 63·7 [2·1] %)	Percent total fat mass No difference between WP and control ($p > 0·05$), (difference in TFM%: 1·6%), ($d = −0·66$) Greater reduction in fat mass following control compared with SP ($p < 0·05$), (difference in TFM%: 0·5%), ($d = −0·16$) Percent total lean mass No difference between WP and control ($p > 0·05$), (difference in TLM%: 0·70%), ($d = 0·21$) Greater increase in lean mass following control compared with SP ($p < 0·05$), (difference in TLM%: −0·3%), ($d = −0·12$)
Dhillon *et al.*[36], 2016	Weight: Tanita; model ABC Total fat mass (kg, %) and fat-free mass (%): GE-Lunar iDXA	Weight ITT analysis: −2·2 kg reduction ($p < 0·05$)* Compliers analysis: Reduction ($p < 0·05$)* Total fat mass ITT analysis: Reduction†, ($p < 0·05$)* Compliers analysis: Reduction†, ($p < 0·05$)* Percent total fat mass ITT analysis: Reduction†, ($p < 0·05$)* Compliers analysis: Reduction†, ($p < 0·05$)* Total fat-free mass ITT analysis: Reduction, no significant change, ($p > 0·05$)* Compliers analysis: Reduction†, ($p < 0·05$)* Percent total fat-free mass ITT analysis: Increase†, ($p < 0·05$)* Compliers analysis: Increase†, ($p < 0·05$)*	Weight ITT analysis: −1·1 kg reduction ($p < 0·05$)* Compliers analysis: Reduction ($p < 0·05$)* Total fat mass ITT analysis: Reduction†, ($p < 0·05$)* Compliers analysis: Reduction†, ($p < 0·05$)* Percent total fat mass ITT analysis: Reduction†, ($p < 0·05$)* Compliers analysis: Reduction†, ($p < 0·05$)* Total fat-free mass ITT analysis: Reduction, no significant change, ($p > 0·05$)* Compliers analysis: Reduction†, ($p < 0·05$)* Percent total fat-free mass ITT analysis: Increase†, ($p < 0·05$)* Compliers analysis: Increase†, ($p < 0·05$)*	Weight ITT analysis: No significant difference, ($p = 0·2$), (difference in weight loss: −1·1 kg)‡ Compliers analysis: No significant difference, ($p = 0·10$)‡ Total fat mass ITT analysis: No significant difference, ($p > 0·05$)‡ Compliers analysis: No significant difference, ($p > 0·05$)‡ Percent total fat mass ITT analysis: No significant difference, ($p > 0·05$)‡ Compliers analysis: Greater reduction in fat mass following intervention compared with control, ($p < 0·05$)‡ Total fat-free mass ITT analysis: No significant difference, ($p > 0·05$)‡ Compliers analysis: No significant difference, ($p > 0·05$)‡ Percent total fat-free mass ITT analysis: No significant difference, ($p > 0·05$)‡ Compliers analysis: Greater increase in percent total fat-free mass following intervention compared with control, ($p < 0·05$)‡
Li *et al.*[40], 2010	Weight: Detecto-Medic, Deteco-scale BMI: Weight/height (kg/m^2)	Weight −3·7 kg reduction ($p < 0·01$), (least-squares mean [SE]: pre 86·0 [1·4] kg, post 82·3 [1·6] kg) BMI −1·3% reduction ($p < 0·05$), (least-squares mean [SE]: pre 30·1 [0·4] kg/m^2, post 28·8 [0·4] kg/m^2)	Weight −2·7 kg reduction ($p < 0·01$), (least-squares mean [SE]: pre 85·5 [2·2] kg, post 82·8 [2·5] kg) BMI −0·6% reduction ($p < 0·05$), (least-squares mean [SE], pre 30·9 [0·4] kg/m^2, post 30·3 [0·5] kg/m^2)	Weight No significant difference (difference in weight loss: −1·0 kg), ($p = 0·09$)‡ BMI Greater reduction in BMI following intervention compared with control (difference in BMI: −0·7 kg/m^2), ($p < 0·05$)‡

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Table 2. (*Continued*)

Author, year	Technology/ method used to assess weight or body composition	Results: Within-group effect (time) Intervention:	Control:	Between-group effect (intervention-control)
Rock *et al.*[37], 2016	Weight: Scales (type and brand not mentioned)	Weight IS WR: −8·1 kg reduction ($p < 0·001$)* IR WR: −6·8 kg reduction ($p < 0·001$)*	Weight IS LF: −7·5 kg reduction ($p < 0·001$)* IR LF: −8·8 kg reduction ($p < 0·001$)* IS LC: −4·3 kg reduction ($p < 0·001$)* IR LC: −7·0 kg reduction ($p < 0·001$)*	Weight Greater weight loss following IS WR compared with IS LC ($p = 0·04$), (difference in weight loss: −3·8 kg), ($d = 0·88$) No difference between all other groups (all $p > 0·05$), (IS WR and IS LF: difference in weight loss: −0·6 kg) ($d = 0·08$), (IR WR and IR LF: difference in weight loss: 2 kg) ($d = -0·30$), (IR WR and IR LC: difference in weight loss: 0·2 kg) ($d = -0·04$)
Wien *et al.*[38], 2003	Weight, total fat mass and fat-free mass: BIA, Tanita, TBF-300 BMI: Weight/height (kg/m^2)	Weight −19·5 kg significance not reported§, (least-squares mean [SE], pre 111·0 [1·8] kg, post 91·5 [1·0] kg) BMI −6·7 kg/m^2 significance not reported, (least-squares mean [SE], pre 38·3 [0·3] kg/m^2, post 31·6 [0·3] kg/m^2) Total fat mass −14·1 kg significance not reported§, (least-squares mean [SE], pre 46·5 [1·0] kg, post 32·4 [1·1] kg) Total fat-free mass −5·1 kg reduction†, ($p < 0·0001$)§, (least-squares mean [SE], pre 62·6 [0·6] kg, post 57·5 [0·7] kg)	Weight −12·1 kg significance not reported§, (least-squares mean [SE], pre 111·0 [1·8] kg, post 98·9 [1·0] kg) BMI −4·2 kg/m^2 significance not reported, (least-squares mean [SE], pre 38·4 [0·3] kg/m^2, post 34·2 [0·3] kg/m^2) Total fat mass −9·1 kg significance not reported§, (least-squares mean [SE], pre 46·3 [1·0] kg, post 37·2 [1·1] kg) Total fat-free mass −2·5 kg reduction†, ($p < 0·0001$)§, (least-squares mean [SE], pre 62·3 [0·6] kg, post 59·9 [0·7] kg)	Weight Greater weight loss following intervention compared with control, (difference in weight loss: −7·4 kg), ($p = 0·0001$)‡ BMI Greater reduction in BMI following intervention compared with control, (difference in BMI: −2·5 kg/m^2), ($p = 0·0001$)‡ Total fat mass Greater reduction in fat mass following intervention compared with control (difference in TFM: −5·0 kg), ($p < 0·05$)‡ Total fat-free mass No significant difference (difference in TFFM: −2·6 kg), ($p > 0·05$)‡

BMI, body mass index; kg/m^2, kilogram per square metre; kg, kilogram; *p*, statistical significance ($p < 0.05$); SD, standard deviation; *d*, Cohen's *d* effect size; DEXA, dual-energy X-ray absorptiometry; CVP, conventional peanut; HOP, high-oleic peanut; BIA, bioelectrical impedance analysis; WP, whole peanut; SP, skinned peanut; ITT, intention to treat; SE, standard error; IS, insulin-sensitive; WR, walnut-rich diet; IR, insulin-resistant; LF, low-fat/high-carbohydrate diet; LC, low-carbohydrate/high-fat diet.
* No pre/post values reported in paper.
† No change value reported in paper.
‡ No Cohen's *d* effect size calculated.
§ Data were reported as pounds but converted to kilograms.

per cent fat-free mass and found that, while both groups had significant increases over time, there was a significantly greater increase after an ER diet with almonds compared with a nut-free ER diet.

Lean mass and per cent lean mass. Measures of lean mass were reported in two studies (three papers)[34,35,41]. One study evaluated absolute amount of lean mass[35] and per cent lean mass[34] which they reported separately across two publications, whilst de Oliveira Fialho *et al.* reported only per cent lean mass[41]. Total lean mass was significantly reduced after a 4-week ER diet without peanuts; however, this reduction was not statistically different to that observed in the intervention group[34,35].

Per cent lean mass increased over time in all groups in the study by de Oliveira Fialho[41], but there was a significantly greater increase in per cent lean mass in the nut-free group after an 8-week ER diet compared with the group consuming 56 g/d of skinned peanuts (noting the skinned peanut group had a lower per cent lean mass at baseline), but no difference when compared with the group consuming 56 g/d of whole peanuts. This was different to Alves *et al.*[34], who found a small but significant increase in per cent lean mass over time in the high-oleic peanut group.

Effect of intervention on glycaemic control

Fasting glucose. Glycaemic control outcomes are presented in Table 3 and Supplementary Material C. Abazarfard *et al.*[39] was

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Table 3. Changes in measures of glycaemic control over time and between groups

Author, year	Technology/ method used to assess glycaemic control	Results: Within-group effect (time) Intervention:	Control:	Between-group effect (intervention-control)
Abazarfard *et al.*[39], 2014	Fasting plasma glucose: Venous blood draw	Fasting glucose −0·7 mmol/l reduction, ($p < 0·001$)*, (mean [SD]: pre 5·7 [0·3] mmol/l, post 5·0 [0·3] mmol/l)	Fasting glucose −0·4 mmol/l reduction ($p < 0·001$)*, (mean [SD]: pre 5·6 [0·3] mmol/l, post 5·2 [0·3] mmol/l)	Fasting glucose Greater reduction in glucose following intervention compared with control (difference in glucose: −0·3 mmol/l), ($p < 0·001$), ($d = 1·41$)
Moreira Alves *et al.*[35], 2014	Fasting serum glucose and insulin: Venous blood draw HOMA-IR: (insulin (mU/l)/$22·5e^{-\ln \text{glucose (mmol/l)}}$)	Fasting glucose CVP: 0·2 mmol/l increase, no significant change ($p > 0·05$)*,† HOP: 0·3 mmol/l increase ($p < 0·05$)*,† Fasting insulin CVP: 3·40 pmol/l no significant change ($p > 0·05$)‡,† HOP: −5·63 pmol/l no significant change ($p > 0·05$)‡,† HOMA-IR CVP: 0·18 no significant change ($p > 0·05$)† HOP: −0·02 no significant change ($p > 0·05$)†	Fasting glucose 0·2 mmol/l increase, ($p < 0·05$)*,† Fasting insulin 5·14 pmol/l no significant change ($p > 0·05$)‡,† HOMA-IR 0·26 no significant change ($p > 0·05$)†	Fasting glucose No significant difference ($p > 0·05$). (CVP and control: difference in glucose: 0·0 mmol/l), ($d = 0·00$). (HOP and control: difference in glucose: 0·1 mmol/l), ($d = 0·22$) Fasting insulin No significant difference ($p > 0·05$). (CVP and control: difference in insulin: −1·74 pmol/l), ($d = −0·05$), (HOP and control: difference in insulin: −10·76 pmol/l), ($d = 0·01$) HOMA-IR No significant difference ($p > 0·05$), (CVP and control: difference in HOMA: −0·08), ($d = −0·06$), (HOP and control: difference in HOMA: −0·28), ($d = −0·18$)
de Oliveira Fialho *et al.*[41], 2022	Fasting glucose and insulin (unclear if serum or plasma): Venous blood draw HOMA-IR: (insulin (mU/l)/$22·5e^{-\ln}$ glucose (mmol/l))	Fasting glucose WP: −0·1 mmol/l no significant change ($p = 0·509$)*, (mean [SD]: pre 4·9 [0·7] mmol/l, post 4·8 [0·3] mmol/l) SP: 0·2 mmol/l no significant change (p=0·768)*, (mean [SD]: pre 5·0 [1·0] mmol/l, post 5·2 [1·3] mmol/l) Fasting insulin WP: 10·42 pmol/l no significant change ($p = 0·580$)‡, (mean [SD]: pre 69·45 [26·39] pmol/l, post 79·87 [26·39] pmol/l) SP: 10·42 pmol/l no significant change ($p = 0·631$)‡, (mean [SD]: pre 83·34 [50·00] pmol/l, post 93·76 [44·45] pmol/l) HOMA-IR WP: −0·2 no significant change ($p = 0·687$), (mean [SD]: pre 2·5 [1·8], post 2·3 [1·1]) SP: 0·7 no significant change ($p = 0·417$), (mean [SD]: pre 2·2 [5·1], post 2·9 [5·8])	Fasting glucose −0·2 mmol/l no significant change ($p = 0·834$)*, (mean [SD]: pre 4·8 [0·7] mmol/l, post 4·6 [0·6] mmol/l) Fasting insulin 12·50 pmol/l no significant change ($p = 0·259$)‡, (mean [SD]: pre 59·73 [34·73] pmol/l, post 72·23 [34·03] pmol/l) HOMA-IR 0·6 no significant change ($p = 0·180$), (mean [SD]: pre 1·4 [1·9], post 2·0 [1·6])	Fasting glucose No significant difference ($p > 0·05$), (WP and control: difference in glucose: 0·1 mmol/l), ($d = −0·58$). (SP and control: difference in glucose: 0·1 mmol/l), ($d = 0·00$) Fasting insulin No significant difference ($p > 0·05$), (WP and control: difference in insulin: −2·08 pmol/l), ($d = −0·15$). (SP and control: difference in insulin: −2·08 pmol/l), ($d = −0·15$) HOMA-IR No significant difference ($p > 0·05$). (WP and control: difference in HOMA: −0·8), ($d = −0·85$). (SP and control: difference in HOMA: 0·1), ($d = 0·12$)
Dhillon *et al.*[36], 2016	Fasting serum glucose and insulin: Venous blood draw	Fasting glucose ITT analysis: 0·2 mmol/l increase ($p < 0·05$)*,† Compliers analysis: No significant change ($p > 0·05$)*,† Fasting insulin ITT analysis: No significant change ($p > 0·05$)§,† Compliers analysis: No significant change ($p > 0·05$)§,†	Fasting glucose ITT analysis: 0·1 mmol/l increase ($p < 0·05$)*,† Compliers analysis: No significant change ($p > 0·05$)*,† Fasting insulin ITT analysis: No significant change ($p > 0·05$)§,† Compliers analysis: No significant change ($p > 0·05$)§,†	Fasting glucose ITT analysis: No significant difference, (difference in glucose: 0.1) ($p = 0.75$)¶ Compliers analysis: No significant difference ($p = 0.62$)¶ Fasting insulin ITT analysis: No significant difference ($p = 0.55$)¶ Compliers analysis: No significant difference ($p = 0.95$)¶

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Table 3. (*Continued*)

Author, year	Technology/ method used to assess glycaemic control	Results: Within-group effect (time) Intervention:	Control:	Between-group effect (intervention-control)
Li *et al.*[40], 2010	Fasting serum glucose and insulin: Venous blood draw	Fasting glucose −0·2 mmol/l no significant change ($p > 0·05$)*, (least-squares mean [SE], pre 4·9 [0·2] mmol/l, post 4·7 [0·1] mmol/l) Fasting insulin −18·75 pmol/l reduction ($p < 0·05$)‡, (least-squares mean [SE], pre 79·17 [12·50] pmol/l, post 60·42 [6·95] pmol/l)	Fasting glucose −0·5 mmol/l no significant change ($p > 0·05$)*, (least-squares mean [SE], pre 5·4 [0·5] mmol/l, post 4·9 [0·1] mmol/l) Fasting insulin 9·72 pmol/l no significant change ($p > 0·05$)‡, (least-squares mean [SE], pre 102·79 [20·84] pmol/l, post 112·51 [27·09] pmol/l)	Fasting glucose No significant difference (difference in glucose: 0.3 mmol/l) ($p > 0.05$)¶ Fasting insulin No significant difference (difference in insulin:-28.50 pmol/l) ($p > 0.05$)¶
Rock *et al.*[37], 2016	Fasting serum glucose and insulin: Venous blood draw HOMA-β: Not reported HOMA-IR: ((fasting glucose, mmol/l) × (fasting insulin, mIU/l))/22·5	Fasting glucose WR: −0·2 reduction¶, ($p < 0·05$)*, (mean [SEM], pre: 5·4 [0·1] mmol/l, post 5·2 [0·1] mmol/l) Fasting insulin WR: −1·39 no significant change ($p > 0·05$)‡, (mean [SEM], pre: 97·23 [6·95] pmol/l, post 95·84 [8·33] pmol/l) HOMA-IR WR: −1 no significant change, ($p > 0·05$), (mean [SEM], pre: 4 [0·2], post 3 [0·3]) HOMA-β WR: no significant change, ($p > 0·05$)†	Fasting glucose LF: −0·3 reduction¶, ($p < 0·05$)*, (mean [SEM], pre: 5·4 [0·1] mmol/l, post 5·1 [0·1] mmol/l) LC: −0·1 no significant change ($p > 0·05$)*, (mean [SEM], pre: 5·3 [0·1] mmol/l, post 5·2 [0·1] mmol/l) Fasting insulin LF: −12·50 reduction¶, ($p < 0·05$)‡, (mean [SEM], pre: 97·23 [6·95] pmol/l, post 84·73 [5·56] pmol/l) LC: −13·89 no significant change ($p > 0·05$)‡, (mean [SEM], pre: 104·18 [6·95] pmol/l, post 90·29 [6·95] pmol/l) HOMA-IR LF: −1 reduction¶, ($p < 0·05$), (mean [SEM], pre: 4 [0·2], post 3 [0·2]) LC: −1 reduction¶, ($p < 0·05$), (mean [SEM], pre: 4 [0·3], post 3 [0·3]) HOMA-β LF: no significant change ($p > 0·05$)† LC: no significant change ($p > 0·05$)†	Fasting glucose No significant difference (all $p > 0·05$). (WR and LF: difference in glucose: 0·1 mmol/l), ($d = −0·17$). (WR and LC: difference in glucose: −0·1 mmol/l), ($d = 6·13$) Fasting insulin No significant difference (all $p > 0·05$). (WR and LF: difference in insulin: 11·11 pmol/l), ($d = −0·23$). (WR and LC: difference in inulin: 12·50 pmol/l), ($d = −0·26$) HOMA-IR No significant difference (all $p > 0·05$). (WR and LF: difference in HOMA: 0), ($d = 0·00$). (WR and LC: difference in HOMA: 0), ($d = 0·00$) HOMA-β No significant difference (all $p > 0.05$)¶
Wien *et al.*[38], 2003	Fasting serum glucose and insulin: Venous blood draw HOMA-IR: ((fasting insulin, pM × fasting glucose, mM/ 22·5)	Fasting glucose −1·3 mmol/l reduction¶, ($p < 0·001$)*, (least-squares mean [SE], pre 8·4 [0·6] mmol/l, post 7·1 [0·6] mmol/l) Fasting insulin −173·62 pmol/l reduction¶, ($p < 0·0001$)‡, (least-squares mean [SE], pre 319·47 [34·73] pmol/l, post 145·85 [34·73] pmol/l) HOMA-IR −13 reduction¶, ($p < 0·0001$), (least-squares mean [SE], pre 20 [4], post 7 [2])	Fasting glucose −1·3 mmol/l reduction¶, ($p < 0·001$)*, (least-squares mean [SE], pre 8·4 [0·6] mmol/l, post 7·1 [0·6] mmol/l) Fasting insulin −104·18 pmol/l reduction¶, ($p < 0·0001$)‡, (least-squares mean [SE], pre 326·42 [34·73] pmol/l, post 222·24 [34·73] pmol/l) HOMA-IR −6 reduction¶, ($p < 0·0001$), (least-squares mean [SE], pre 17 [4], post 11 [2])	Fasting glucose No significant difference (difference in glucose: 0.0 mmol/l), ($p > 0.05$)¶ Fasting insulin No significant difference (difference in insulin: −69.44 pmol/l), ($p > 0.05$)¶ HOMA-IR No significant difference (difference in HOMA: −7), ($p > 0.05$)¶

mmol/l, millimole per litre; *p*, statistical significance ($p < 0.05$); SD, standard deviation; *d*, Cohen's *d* effect size; HOMA-IR, homeostatic model of assessment for insulin resistance; mU/l, milli-units per litre; CVP, conventional peanut group; HOP, high-oleic peanut group; WP, whole peanut with skins; SP: whole peanut without skin; pmol/l, picomole per litre; ITT, intention-to-treat analysis; SE, standard error; HOMA-β, homeostatic model of assessment of β cell function; mIU/l, milli international units per litre; WR, walnut-rich diet; SEM, standard error or mean; LF, low-fat/high-carbohydrate diet; LC, low-carbohydrate/high-fat diet; pM, picomolar; mM, millimolar.

* Data were reported as milligram per decilitre but converted to millimole per litre.

† No pre/post values reported in paper.

‡ Data were reported as micro-unit per millilitre but converted to picomole per litre.

§ Data were reported as milligram per decilitre.

¶ No Cohen's d effect size calculated.

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the only study that found a significantly greater reduction in fasting glucose with nut consumption (12-week ER diet with 50 g/d of almonds) compared with an ER diet without nuts. Energy restriction did not produce consistent changes in fasting glucose between studies; three studies reported significant reductions in fasting glucose[37–39], while two studies reported a significant increase in fasting glucose[35,36]. More specifically, in the study by Moreira Alves *et al.*[35] both the high-oleic peanuts and control groups had higher fasting glucose levels at the end of the study compared with baseline levels but there were no changes over time for the conventional peanut group. In the study by Dhillon *et al.*, there were significant increases in fasting glucose levels at 12 weeks compared with baseline (time effect only), but this could only be detected when an intention-to-treat analysis approach was used, but this change did not reach significance in the compliers analysis[36].

Fasting insulin. Six studies reported on fasting insulin[35–38,40,41]. Of these, three studies[37,38,40] found a significant reduction in insulin over time; however, there were no differences between those consuming an ER diet with or without nuts.

Insulin resistance and pancreatic β-cell function (HOMA-IR and HOMA-β). One study reported both HOMA-IR and HOMA-β[37], and three studies reported HOMA-IR only[35,38,41]. From the four studies that reported HOMA-IR, two studies[37,38] found a significant reduction in HOMA-IR over time; however, no difference between the ER diets with or without nuts was observed. Rock *et al.*[37] found no differences in HOMA-β over time (after 52 weeks) or between ER nut-free and nut-containing (42 g/d of walnuts) groups.

Discussion

This scoping review reports on changes in measures of anthropometry and glycaemic control from interventions testing nut-enriched ER diets compared with nut-free ER diets. More than half (four out of seven) of the studies included in this review[37–39,41] found individuals consuming a nut-enriched ER diet achieved significantly more weight loss compared with individuals in the nut-free ER diet. In those studies, weight loss from a nut-enriched ER diet ranged from −2·6 to −19·5 kg (per cent weight loss: −3·0% to −18·0%), which equates to ∼−0·1 to −0·2 kg each week[37–39,41]. In those same studies, the added benefit from the nut-enriched ER diets ranged from −1·4 to −7·4 kg[37–39,41]. In these same studies, the extent of energy restriction in studies varied considerably (approximately four-fold), the amount of nuts consumed varied two-fold (42 g to 84 g), and two studies tightly matched intervention and control diets for macronutrient composition[37,39], while the other two studies had nut and control diets with extremely different macronutrient compositions[38,41]. Two studies exclusively recruited women with obesity but without existing chronic disease[39,41], one recruited women with overweight or obesity who were non-diabetic but were then categorised as either being insulin sensitive or insulin resistant[37], and one recruited both men and women with type 2 diabetes and hypertension[38].

However, three studies (reported in four papers)[34–36,40] found no difference in weight loss between the nut-enriched and nut-free ER diets. These studies[34–36,40] typically included a smaller amount of nuts in an ER diet (38–56 g/d) compared with studies that found a significantly greater weight loss when nuts were consumed (50–84 g/d)[37–39,41]. Increasing the quantity of nuts in the diet may enhance satiety more effectively and contribute to greater weight loss. This notion is supported by three papers outside the scope of this review (as they did not included glucose as an outcome measure[44–46]) that all reported no additional improvements in measures of anthropometry when nuts were added (42 g/d of mixed nuts[46], 56 g/d of almonds[44] or 38 g peanuts + 35 g peanut butter[45]) to ER diets, compared with nut-free ER diets. The potential dose-response effects of nuts have been evaluated in other studies of cardiovascular risk factors. A previous meta-analysis found the dose–response between nut intake and total cholesterol and LDL cholesterol was non-linear (P-nonlinearity <0·001 each), but stronger effects were observed for ≥60 g nuts/d[47]. As evidence builds for effects of nuts in ER diets on weight loss, it would be helpful to determine whether similar dose–response relationships exist for weight, body composition and glucose.

The degree of energy restriction varied widely in the included studies, and there was little consistency in how the inclusion of nuts affected weight loss between studies with similar levels of restriction. For example, the inclusion of 56 g of peanuts to diets with a modest average energy restriction (−1046 kJ) resulted in greater weight loss compared with no nut consumption in young to middle-aged women[41] while similarly aged men (18–50 years) did not have this benefit[34,35]. Key differences between these studies includes the length of supplementation, 8 weeks for women[41] and 4 weeks for men[34,35], raising the possibility that a longer duration of supplementation with nuts may be required to see a greater weight loss. Another possibility is that, whilst the degree of energy restriction and amount of nuts consumed were constant between studies in women[41] and men[34,35], the proportion of energy from nuts would have been higher for women. Future studies may find it beneficial to prescribe the dose of nuts on the basis of the initial body weight of participants or as a per cent of total energy rather than the same absolute dose for all participants.

The type of anthropometric measures reported in included studies varied. Less than half of the studies in this review reported on fat mass, with three[36,38,41] demonstrating a significant difference between the control and intervention groups. Only one study (two papers) found a reduction over time in the nut-enriched group but no difference between groups[34,35].

The two studies reporting fat-free mass found reductions over time associated with the consumption of ER diets, but nuts had no additional effect[36,38]. Mixed findings were observed in the two studies (three papers) reporting lean mass[34,35,41]. In one study, while there was an increase in per cent lean mass in all groups over time, there was a greater increase in per cent total lean mass in the nut-free ER diet compared with the skinned peanut ER diet, although groups differed at baseline, likely explaining the difference[41]. In the second study, a small but significant increase in per cent lean mass was reported over time

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in the high-oleic peanut group only, with no change in the conventional peanut group or control group and no between-group differences[34,35]. These two studies were both relatively short interventions (4–8 weeks), prescribed the same amount of energy restriction (1046 kJ reduction) and included the same type and amount of nuts in the diet (56 g of peanuts)[34,35,41]. It should be noted that the study by de Oliveira Fialho *et al.*[41] was conducted only in women while the study reported by Alves *et al.* and Moreira Alves *et al.*[34,35] was conducted only in men, which may have contributed to the different findings. Future studies considering the effects of nuts in combination with energy restriction should continue to assess changes in body composition to add to this small body of literature.

Papers included in this scoping review have highlighted that weight, BMI and fat mass were the most common measures reported, with far fewer papers reporting the effect of a nut-rich diet on fat-free mass (three studies)[34,35,41] or lean mass (two studies)[36,38]. Fat-free mass, which includes lean mass and bone, is an important measure used to observe changes in muscle mass[48]. This review has identified that future studies of nut-enriched ER diets should report all components of body composition to observe changes in fat-free mass and the maintenance of muscle mass[49].

Various mechanisms have been proposed to explain why a nut-rich ER diet may facilitate weight loss and improve body composition beyond that of an ER diet alone[6]. Nuts are rich in protein, unsaturated fats and fibre, which may prevent over-consumption by increasing hormones that assist with satiety (e.g. cholecystokinin and peptide YY)[21]. High amounts of unsaturated fats in nuts could also increase fat oxidation and diet-induced thermogenesis, further assisting with weight loss[21]. Nuts also provide less energy than what is predicted by the Atwater factor due to incomplete lipid release[21].

There is inconsistency in the literature on effects of nuts on glycaemic control. In this review, the majority of studies (six out of seven) did not find a significant difference in fasting glucose, fasting insulin, HOMA-IR and HOMA-β between the ER diets with or without nuts[35–38,40,41]. Only Abazarfard *et al.*[39] found a significantly greater reduction in fasting glucose in the group eating an almond-enriched ER diet (50 g/d) for 12 weeks compared with a nut-free ER diet. In this study, adults consuming the nut-enriched diet lost a larger amount of weight over 12 weeks compared with adults on the control diet (−3·7 kg compared with −1·3 kg), and this greater weight loss may explain the greater reduction in fasting glucose compared with the nut-free diet. However, the clinical significance of these measures should be considered. Individuals in the nut-enriched ER diet had an average reduction in fasting glucose of 0·7 mmol/l compared with 0·4 mmol/l in the nut-free group[39], and it is unlikely that a difference of 0·3 mmol/l in glucose would be clinically meaningful. While Abazarfard *et al.*[39] recruited participants on the basis that they did not have a diagnosis of diabetes, baseline fasting glucose levels were on average within the range for prediabetes and those in the almond enriched group had significantly higher levels, which may also have influenced the findings from this study.

This scoping review has highlighted a gap in the literature where no studies with a nut-rich ER diet have recorded interstitial glucose or HbA1c. HbA1c is a common biomarker used to measure long-term glycaemic variability over the previous 2–3 months [13]. As the majority of studies in this review intervened for 4–12 weeks, short intervention length may have been one reason why no studies in this review reported on this parameter. Additionally, HbA1c is regularly used by clinicians to monitor hyperglycaemia[50] in persons with diabetes[51]; few studies in this review evaluated the effects of nuts in this clinical population.

When considering data from studies with tree nuts and peanuts without energy restriction, there is consistent evidence that the addition of nuts to meals or nuts consumed as snacks results in lower postprandial glycaemia among healthy participants, those with impaired glucose tolerance and those with type 2 diabetes[24]. However, the effects of consuming peanuts or tree nuts without energy restriction on fasting values of glucose and insulin are not consistent. A review of twelve studies published in 2014 reported no difference in fasting insulin and HOMA-IR (although the direction of effect favoured tree nuts) between diets (without ER) with or without tree nuts, but a significantly greater reduction in fasting glucose (weighted mean difference (WMD): −0·15 mmol/l; 95% CI: −0·27, −0·02 mmol/l) and HbA1c (WMD: −0·07%, 95% CI: −0·10%, −0·03%) with nuts compared with a nut-free diet[52]. In contrast, a more recent review of forty studies published in 2019 reported the opposite with significantly lower fasting insulin (WMD: −0·40 μIU/mL; 95% CI: −0·73, −0·07 μIU/mL; and HOMA-IR (calculated from fasting glucose and insulin) (WMD: −0·23; 95% CI: −0·40, −0·06) with nuts. Compared with the previous review, there was no longer a significant effect of nut consumption on fasting glucose (WMD: −0·52 mg/dL; 95% CI: −1·43, 0·38 mg/dL) or HbA1c (WMD: 0·02%; 95% CI: −0·01%, 0·04%), likely due to a greater variability in the response[25].

There is growing evidence from several epidemiologic studies that postprandial hyperglycaemia commonly precedes fasting hyperglycaemia in the development of metabolic syndrome[53] and type 2 diabetes[54]. Thus, future studies should test the glycaemic response to nut-enriched energy restricted diets with both fasting and postprandial blood samples. Multiple mechanisms have been proposed to explain why a nut-rich ER diet improves glycaemic control[24]. Weight loss from energy restriction reduces the expression of multiple proinflammatory cytokines, such as tumour necrosis factor-α (TNF-α)[55]. Dampening the expression of TNF-α reduces insulin resistance which allows insulin to effectively regulate glucose[55]. An ER diet can also reduce the size of adipocytes, which improves insulin sensitivity[56]. Low amounts of carbohydrates and high amounts of unsaturated fat, fibre and protein in nuts also delay gastric emptying, which lowers postprandial glucose[57]. The rich source of MUFA in nuts can also improve β cell efficiency through enhanced intestinal section of glucagon-like-peptide-1[24,58]. We have demonstrated a larger postprandial response when almonds are consumed compared with carbohydrate-rich snacks in some pancreatic peptides (glucose-dependent insulinotropic polypeptide (GIP), and pancreatic polypeptide (PP)) as well as in glucagon, but no difference in GLP-1 concentrations[59]. These findings are similar to those reported with walnuts and pistachios[27,60], but others

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have reported an attenuation in postprandial GLP-1 and insulin following walnuts compared with a nut-free comparator matched on total fat but with a different fatty acid composition[61]. Testing postprandial glucose, insulin and GLP-1 responses after consuming an energy-restricted diet with nuts may allow these mechanisms to be explored in further detail[62].

Additional consideration should be given to the macronutrient composition of the diet when interpreting glycaemic responses. A meta-analysis of feeding trials has provided evidence that dietary macronutrients have diverse effects on glucose–insulin homeostasis[63], with fatty acid composition being a key driver of the different glycaemic responses with isocaloric exchanges between carbohydrate and fat[64]. Only one study included in the review detected a significantly different effect on glucose with the inclusion of almonds, and while these authors did match the control and intervention diets on total fat and carbohydrate, the inclusion of almonds would have increased the monounsaturated fat content compared with the nut-free diet[39]. It should be noted there were some studies that did not detect a significant effect on glucose with nut consumption that also ensured the intervention and control diets were matched on per cent energy from macronutrients[34,35,41], although the extent of energy restriction used in these studies was four-fold lower, and the recruited participants had better glucose control compared with people in the study by Abazarfard *et al.*[39]. Future studies should continue to explore if there is an optimal degree of energy restriction and nut consumption that, when combined, results in beneficial effects on glycaemic control, and whether there is a requirement for participants to have some metabolic impairment before an improvement can be seen.

Limitations of the review

This scoping review excluded studies of ER diets that contained nuts combined with another food to isolate the effect of nuts alone. This is a common approach when teasing out specific food effects, but it did highlight the paucity of available literature that could be included in this review. While other studies have tested the effects of ER diets that include nuts as part of an overall dietary pattern (such as an ER-Mediterranean diet either compared with habitual diets or with an ER-high-carbohydrate/low-fat diet) and found benefits for both weight loss[65,66] and metabolic complications associated with obesity[67], these studies do not allow the benefit to be attributed to a specific food.

Studies all relied on self-report intake, and few studies reported on dietary compliance to energy restriction in the intervention or control groups, whether the prescribed amount of nuts was consistently consumed or whether the control groups maintained a nut-free diet for the duration of the study. Furthermore, the nutrient composition of diets and comparator foods were not consistent across studies, which may have influenced the findings, particularly for glycaemic control.

This review excluded studies published in languages other than English, which may have restricted the amount of literature included in this review. Most studies identified in this review were from North or South America, and it cannot be assumed that the effects of nut-enriched ER diets on weight, body composition and glycaemic control found in this review are the same for other populations. A final limitation was the relative lack of long-term outcomes addressed by the studies identified in the review.

Conclusion

Studies varied considerably in the extent of energy restriction and the type and quantity of nuts incorporated into the diet. While all studies found improvements in body mass following an ER diet, there were inconsistent effects on glucose and insulin. The inclusion of nuts to ER diets also generated inconsistent effects on measures of adiposity and glycaemic control, but importantly, no study revealed an adverse effect of nut consumption on health outcomes. These outcomes may be due to variable intervention periods, the way nuts were incorporated into the diet or testing of populations that predominantly did not have impaired glucose control. Despite these mixed findings, nuts are a nutrient-rich snack that can help achieve recommended intakes of essential nutrients during energy restriction and therefore should be included in future ER weight loss diets.

Recommendations for research

This review has identified scope for future studies testing ER diets with and without nuts to investigate temporal patterns of glucose regulation, using CGM or FGM enabling investigation of both fasting and postprandial responses. Future studies should also consider whether there is a minimum length of time that nuts are consumed before a benefit is observed, directly compare diets with and without nuts that include different levels of ER, and directly compare different types and/or amounts of nuts within ER diets to help clarify how nuts can effectively support weight management.

Most of the studies identified in this review were conducted in healthy populations, highlighting that there is limited information about the potential of nut-enriched ER diets to benefit population with chronic cardiometabolic conditions. There is also a need to explore whether ER diets with nuts improve weight and glycaemic control in a greater diversity of people from different ethnicities. As there are high rates of obesity[68] and associated metabolic complications[69] in many countries around the world, it is imperative that high-quality, sustainable dietary solutions are offered[70]. Inclusion of nuts into a low-energy diet provides a shelf-stable, sustainable source of protein and essential nutrients[71] that have the potential to help people manage satiety as they lose weight[46].

Ideally, future studies would consider either providing all study foods to reduce the requirement for self-reporting energy intake and/or conduct studies within a metabolic ward. Whilst the practicality of this latter suggestion may be challenging, this would provide greater certainty that the prescribed amount of nuts was being consumed, and that the nut-free diet was truly devoid of nuts. Future studies could also consider newer approaches to monitoring food intake[72], noting that this field is rapidly advancing[73]. Additionally, the development and utilisation of specific biomarkers that reflect nut intake[74,75]

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may, in time, limit burden on participants and enhance confidence with adherence to prescribed diets.

Supplementary material

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Authorship

Conceptualisation and methodology: L.C.M., A.M.H., S.C. and A.M.C. Established the search strategy and identified eligible papers: L.C.M., A.M.H, S.C. and A.M.C. Data extraction, analysis and interpretation of data: L.C.M., A.M.H., S.C. and A.M.C. Preparation of the first manuscript draft: L.C.M. Drafting and editing: L.C.M., A.M.H., S.C. and A.M.C.

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