Public Health Nutrition: 20(12), 2079–2095 doi:10.1017/S1368980017000994

Review Article

Eating frequency and weight and body composition: a systematic review of observational studies

Raquel Canuto[1], Anderson da Silva Garcez[2], Gilberto Kac[3], Pedro Israel Cabral de Lira[4] and Maria Teresa Anselmo Olinto[2,5,*]

[1]Department of Nutrition, Federal University of Rio Grande do Sul, Porto Alegre, Brazil: [2]Graduate Program in Collective Health, University of Vale do Rio dos Sinos, Av. Unisinos 950, CP 275, São Leopoldo, RS 93022-000, Brazil: [3]Graduate Program in Nutrition, Federal University of Rio de Janeiro, Rio de Janeiro, Brazil: [4]Graduate Program in Nutrition, Department of Nutrition, Federal University of Pernambuco, Recife, Brazil: [5]Department of Nutrition, Federal University of Health Science of Porto Alegre, Porto Alegre, Brazil

Submitted 19 October 2016: Final revision received 13 April 2017: Accepted 18 April 2017: First published online 5 June 2017

Abstract

Objective: The present review aimed to examine the association of eating frequency with body weight or body composition in adults of both sexes.

Design: PubMed, EMBASE and Scopus databases were searched. PRISMA and MOOSE protocols were followed. Observational studies published up to August 2016 were included. The methodological quality of the studies was assessed with the Downs and Black checklist.

Setting: A systematic review of the literature.

Subjects: Adults (*n* 136 052); the majority of studies were developed in the USA and Europe.

Results: Thirty-one articles were included in the review: two prospective and twenty-nine cross-sectional studies. Thirteen per cent of the studies received quality scores above 80 %. The assessment of eating frequency and body composition or body weight varied widely across the studies. Potential confounders were included in 73 % of the studies. Fourteen studies reported an inverse association between eating frequency and body weight or body composition, and seven studies found a positive association. The majority of studies applied multiple analyses adjusted for potential confounders, such as sex, age, education, income, smoking, physical activity and alcohol intake. Six studies took into account under-reporting of eating frequency and/or energy intake in the analysis, and one investigated the mediation effect of energy intake.

Conclusions: There is not sufficient evidence confirming the association between eating frequency and body weight or body composition when misreporting bias is taken into account. However, in men, a potential protective effect of high eating frequency was observed on BMI and visceral obesity.

Keywords
Systematic literature review
Obesity
Eating frequency
Body weight
Meal pattern
Body composition

Obesity is increasing at alarming rates worldwide[1]. The media, health professionals and guidelines for health and weight management have postulated that higher eating frequency may be good for weight management[2], but such a recommendation lacks solid evidence to justify it.

Since the 1960s, some scientists have suggested an inverse association between the consumption of a greater number of small meals per day and body weight maintenance[3]. According to them, the consumption of more meals per day might lead to greater thermogenesis, higher insulin sensitivity and lower total energy intake[4,5].

Since then, studies that have attempted to determine the effects of eating frequency on weight have reached different conclusions. Some experimental and observational studies of eating patterns and body weight status conducted in the 1960s and 1970s found an inverse relationship between eating frequency and adiposity, supporting the claim for an association between lower body weight and higher eating frequency[3,6]. More recently, mainly in the

*Corresponding author: Email mtolinto@gmail.com

Table 1 Search strategy for Pubmed, EMBASE and Scopus

Exposure	
EMBASE	'meal frequency' OR 'meal frequencies' OR 'meals'/exp OR 'meals' OR 'meal time'/exp OR 'meal time' OR 'mealtime' OR 'mealtimes' OR 'meal times' OR 'eating frequency' OR 'eating frequencies' OR 'eating episodes' OR 'meal pattern' OR 'meal patterns' OR 'eating pattern' OR 'eating patterns' OR 'eating behaviors' OR 'dietary pattern' OR 'dietary patterns' OR 'dietary habits' OR 'diet habit' OR' diet habits'
Scopus	'meal frequency' OR 'meal frequencies' OR 'meals' OR 'meal time' OR 'mealtime' OR 'mealtimes' OR 'meal times' OR 'eating frequency' OR 'eating frequencies' OR 'eating episodes' OR 'meal pattern' OR 'meal patterns' OR 'eating pattern' OR 'eating patterns' OR 'eating behaviors' OR 'dietary pattern' OR 'dietary patterns' OR 'dietary habits' OR 'diet habit' OR 'diet habits'
Outcome	
PubMed	'weight' [all fields] OR 'overweight' [all fields] OR 'obesity' [all fields] OR 'adiposity' [all fields] OR 'waist circumference' [all fields] OR 'BMI' [all fields] OR 'waist-to-hip ratio' [all fields] OR 'abdominal obesity '[all fields] OR 'change body weight' [all fields]
EMBASE	'weight'/exp OR 'weight' OR 'overweight'/exp OR 'overweight' OR 'obesity'/exp OR 'obesity' OR 'adiposity'/exp OR 'adiposity' OR 'waist circumference'/exp OR 'waist circumference' OR 'BMI'/exp OR 'BMI' OR 'waist-to-hip ratio'/exp OR 'waist-to-hip ratio' OR 'abdominal obesity'/exp OR 'abdominal obesity' OR 'change body weight'
Scopus	'weight' OR 'overweight' OR 'obesity' OR 'adiposity' OR 'waist circumference' OR 'BMI' OR 'waist-to-hip ratio' OR 'abdominal obesity' OR 'change body weight'
Design	
PubMed	Case-Control Study [all fields] OR Case Control Study [all fields] OR Epidemiological Studies [all fields] OR Retrospective Studies [all fields] OR Cohort Study [all fields] OR Incidence Study [all fields] OR Cross-Sectional Study [all fields] OR Cross Sectional Study [all fields] OR Prevalence Study [all fields] OR Longitudinal Study [all fields] OR Follow-Up Study [all fields] OR Prospective Study [all fields]
EMBASE	'case control study'/de OR 'cohort analysis'/de OR 'cross-sectional study'/de OR 'longitudinal study'/de OR 'observational study'/de OR 'prospective study'/de OR 'retrospective study'/de
Scopus	case-control study OR case control study OR epidemiological studies OR retrospective studies OR cohort study OR incidence study OR cross-sectional study OR cross sectional study OR prevalence study OR longitudinal study OR follow-up study OR prospective study
Limits	
PubMed	('adult' [all fields] OR 'adults' [all fields]) AND ('humans' [MeSH terms] AND 'adult' [MeSH terms])
EMBASE	([article]/lim OR [article in press]/lim) AND [adult]/lim AND [humans]/lim AND [embase]/lim
Scopus	(LIMIT-TO (DOCTYPE, 'ar')) AND (LIMIT-TO (SRCTYPE, 'j')) AND (EXCLUDE (EXACTKEYWORD, 'Adolescent') OR EXCLUDE (EXACTKEYWORD, 'Child'))

MeSH, medical subject heading.

2000s, studies have shown mixed conclusions. A meta-analysis on meal frequency with respect to changes in fat and lean mass based on experimental research, published in 2015, found only a small potential benefit of increased feeding frequency for fat mass and body fat percentage[7]. Two observational studies showed results in the same direction[8,9]; while others have also reported a sex difference[10,11]. On the other hand, some studies found a positive association between eating frequency and body weight status[12,13] or did not find any relationship[14,15].

Overall energy intake also has a relevant role in the causal pathway that links meal frequency and weight maintenance, although the results of studies evaluating the effect of eating frequency on energy intake were inconclusive. Edelstein *et al.*[16] and Howarth *et al.*[17] showed that total energy intake increased with increasing frequency of meals or snacks, in both men and women. Meanwhile, Westerterp-Plantenga *et al.*[18] described that healthy young men with a high habitual meal frequency had lower total energy intake. Additionally, a study reported that meal frequency and a period of fasting have no major impact on energy intake[19].

Considering the need to organize these divergent evidences, the aim of the present systematic literature review (SLR) was to examine the association between eating frequency and body weight and body composition in adults of both sexes.

Methods

An SLR was conducted aiming to find original articles on the association between eating frequency and body composition or body weight. The PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses)[20] and MOOSE (Guidelines for Meta-Analyses and Systematic Reviews of Observational Studies)[21] protocols were followed. Thus, the research protocol was identified using the PICO (patient, intervention, comparison, outcome) strategy. The articles retrieved from the literature met the following inclusion criteria: (i) the study design was observational; (ii) the article was derived from original research; (iii) the measured outcome included at least one of weight, change in body weight, overweight, obesity, BMI, adiposity, waist circumference, waist-to-hip ratio or abdominal obesity; (iv) the exposure measurement included eating frequency as number of meals per day; and (v) the samples comprised adults (aged >18 years).

PubMed, EMBASE and Scopus databases were searched. Articles published from 1960 to August 2016 were included. The search strategies are shown in Table 1. Terms relative to eating frequency and body weight were used. Additional papers were identified in the reference lists of selected articles that met the inclusion criteria. All records identified were uploaded or manually entered into EndNote X4.

The searches were conducted by two independent investigators (R.C. and A.S.G.) and their results were compared.

The articles that met all the established criteria were included. Two reviewers (R.C. and A.S.G.) independently read all titles and abstracts. At a second stage, the reviewers read in full all manuscripts that had consensus about their inclusion. If consensus between the two reviewers could not be reached, a third reviewer (M.T.A.O.) was called upon to make a final decision. In four instances the full text of the article was not available. In theses cases we contacted the authors by email up to three times.

The data were extracted and summarized according to the following variables: first author, date of publication, study design, sample size, subjects' age, follow-up duration (prospective studies), outcomes and exposure assessment, statistical analysis including confounders and mediators used in the adjusted analysis, and numerical results.

Guidelines for SLR and meta-analysis have drawn attention to the importance of evaluating the possible bias in the key methodology domains of the primary studies[22]. In the present SLR, a validated checklist originally proposed by Downs and Black was used in order to assess the quality of the selected articles, especially regarding possible bias. This checklist, originally proposed to rate the quality of clinical trials, consists of twenty-seven items that evaluate the risk of bias in five domains: reporting, external validity, internal validity, confounding and power. Subsequently, this checklist was adapted for observational studies[23], and items 8, 13, 23 and 24 were eliminated for longitudinal studies, while items 8, 9, 13, 17, 23 and 24 were excluded for the assessment of cross-sectional studies. In the present SLR, items 14 and 15 were also eliminated for both designs because they evaluate the blinding process and most observational studies do not take blinding into consideration. The final scale ranged from 0 (poorest quality) to 21 points (best quality) for longitudinal studies and 19 points (best quality) for cross-sectional studies. All items received scores of 0 or 1 (1 if the item was contemplated in the study and 0 if the item was not contemplated or was not able to be determined), with the exception of item 5. Item 5 evaluates if a list of main confounders was provided, ranging from 0 to 2 (0 = no; 1 = partially; 2 = yes). In item 27, the score (0 or 1) depended on whether the statistical power of the survey was explicitly stated in the article.

A score of quality was created as follows: the number given by the total sum of the questions was then divided by the number of total applicable items in the study and finally multiplied by 100.

In the second stage of the quality assessment, in the same way, a general assessment of the quality of the articles was performed for each item of the evaluation instrument. The studies with questions that had scores of 1 or 2 were classified as having a 'low risk of bias', whereas scores of 0 reflected a 'risk of bias'.

The two reviewers (R.C. and A.S.G.) independently made use of the checklist to assess the quality of the retrieved articles. When a consensus could not be reached between them, the third reviewer (M.T.A.O.) was called upon to make a final decision.

Results

The search strategies resulted in 6357 articles (2503 from PubMed; 2380 from EMBASE; 1474 from Scopus). After excluding duplications, 5789 titles and abstracts were examined; 209 full texts were selected for reading. One hundred and eighty-two articles were excluded for the following reasons: outcome and exposure measurements did not meet the inclusion criteria (n 172); the study population was not adult (n 1); and the article did not show the statistical results for the analysis of the relationship between exposure and outcome (n 1). Twenty-seven articles met all of the inclusion criteria. The references of these articles were checked, resulting in four additional articles. As a result, a total of thirty-one articles were included in the present SLR (Fig. 1).

The studies had different sample characteristics. The majority of the studies were conducted in the USA[6,9,12,13,15,17,24–28] and European countries[8,10,24,29–38]. Two studies included only men[13,36], five included only women[32,35,37,39,40] and twenty-four included both sexes[6,8–12,14,15,17,24–31,33,34,37,38,41–43]. The sample sizes of the studies ranged between eighty-two[37] and 34 974 individuals[33], and the age of the participants was between 18 and 90 years old. Two prospective[13,15] and twenty-nine cross-sectional studies were retrieved. The follow-up of prospective studies was 8 and 10 years[13,15] (Table 2).

In the reporting items, most articles were classified as having a 'low risk of bias'. On the other hand, in the external validity domain, several of the articles were not clear about how their participants were selected (42 %) or/and did not rely on representative samples (70 %). As regards internal validity, 34·8 % of the studies did not use an accurate method (valid and reliable) to measure the outcomes, using self-reported measures. In the confounding domain, 54·8 % of the articles did not describe characteristics of participants lost between the initial selection process and the final sample, and 29·0 % of the studies did not perform any adjustment for confounding in the analysis. Finally, almost all studies (96·7 %) did not report a power calculation for their sample size and were classified as having a 'risk of bias' in the power domain (Fig. 2).

Considering the fourteen studies that found inverse associations between eating frequency and the outcomes, the following was observed: all studies were cross-sectional; eleven had scores of quality above 70 %[8–11,24,25,30,33,36,43,44]; and eight studies found an association between eating frequency and outcomes measured as body weight, BMI, overweight or obesity[8–10,24,25,30], five as waist circumference

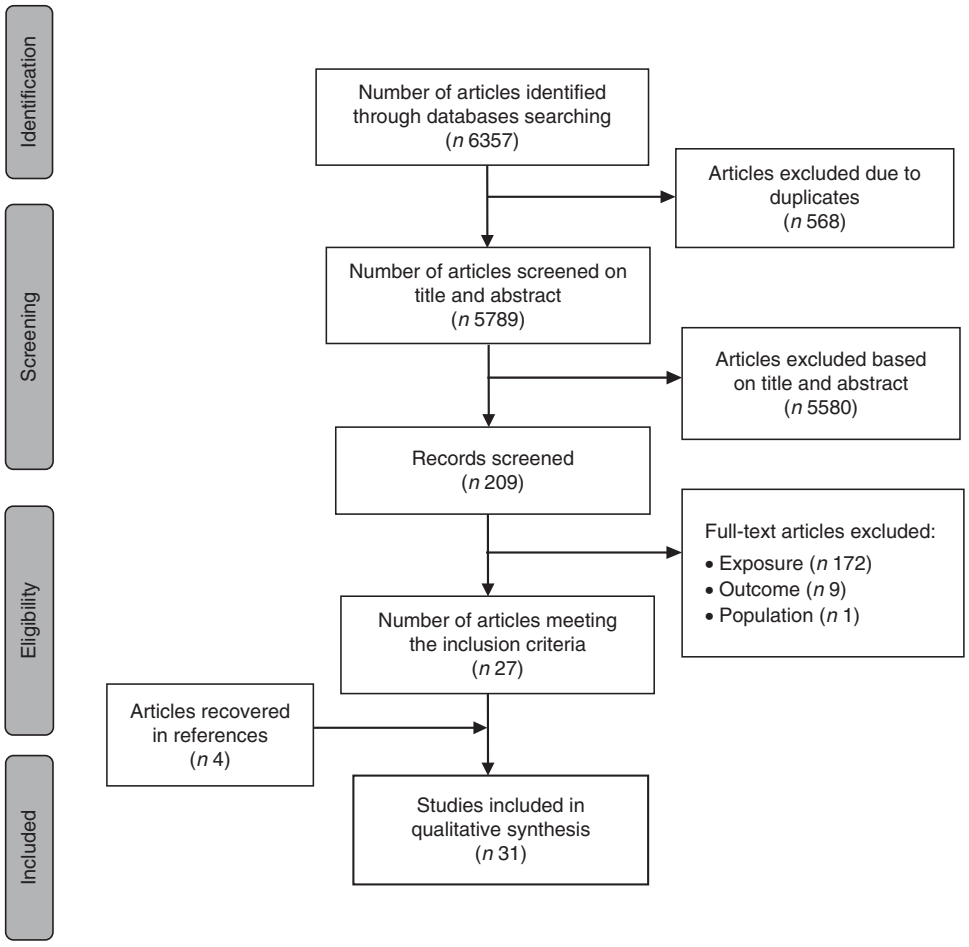

Fig. 1 The search and selection process in the present systematic literature review according to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement

or waist-to-hip ratio[10,11,25,36,44] and one as adiposity index[6]. Only one study used self-reported measurements[28]. In most studies (*n* 11) multiple recalls or meal pattern questionnaires were used in order to assess the eating frequency[6,8–11,24–26,28,33,36]. Five studies classified the exposure as a continuous variable[8,11,25,26,30]; seven according to the three major meals (breakfast, lunch and dinner) and compared the intake of three meals with a greater or lower number of meals[9,10,24,33,36,43,44]. Most studies (*n* 10) performed multiple analyses or other statistical methods to adjust for possible confounding, including sociodemographic variables such as sex[8–10,24,25,30,33,36,43,44], age[9–11,24,25,30,33,36,43,44], education[10,11,24,33,36,44,45], income[10,43,44] and race/ethnicity[25,44]; and behavioural variables such as smoking[24,25,30,33,36,43,44], physical activity[9–11,16,24,30,33,36,43,44], alcohol intake[10,11,30,33,43,44] and dietary characteristics[9–11,24,25,30,36,43,44]. Seven studies took into account energy intake as a confounder[6,9,10,24,25,30,36] and in three of them misreporters of energy were excluded[10,24,36] (Table 3).

Eating frequency was positively associated with the outcomes in seven studies and one of them presented a prospective design[13]. Five studies received quality scores above 70 %[12,13,17,32,38]. The outcomes were measured as weight, BMI, overweight/obesity in six studies[12,13,17,34,35,38]; two studies used self-reported measurements[13,17], including the prospective study. In the majority of studies (*n* 5), eating frequency was assessed through multiple recalls or meal pattern questionnaires. In four studies, the exposure was classified as a continuous variable[32,34,35,38], and in two according to the three major meals and compared with a greater number of meals[12,17]. Four studies that provided a complete list of sociodemographic (age, race/ethnicity, education and income) and behavioural (smoking, physical activity and dietary characteristics) confounders were included in the analysis[12,13,17,38]. In five studies, energy intake was investigated as a confounder[12,17,32,34,38], and one investigated the mediation effect of energy intake[17]. In three of them its measurement took into account misreporting[12,32,38] (Table 4).

Ten studies did not show associations between eating frequency and outcomes, one being prospective[15]. Seven received quality scores above 70 %[14,15,29,31,39,40,42]. Six studies used a simple question or meal pattern questionnaire to access eating frequency[14,15,31,39,41,42] and three used multiple recalls[27,29,37]. Five studies provided a complete list of

Table 2 Summary of population and design characteristics of the studies sorted according to quality scores

Study	Year(s)	Population	Sample size	Age (years)	Study design
Aljuraiban *et al.* (2015)[24]	1996–1999	Women and men from INTERMAP	2696	40–59	Cross-sectional
Murakami & Livingstone (2015)[12]	2003–2012	Women and men from NHANES 2003–2012	18 696	≥60	Cross-sectional
Gigante *et al.* (1997)[42]	1994	Brazilian women and men	1035	20–69	Cross-sectional
Karatzi *et al.* (2015)[29]	NA	Greek women and men	164	Mean 46·8 (SD 9·3)	Cross-sectional
Oliveira *et al.* (2009)[44]	NA	Brazilian women and men	570	19–59	Cross-sectional
Kant *et al.* (1995)[15]	1971–1975 to 1982–1984	Women and men from NHANES I and NHEFS	7147	25–74	Prospective: 8–10-year follow-up
Holmback *et al.* (2010)[10]	1991–1995	Women and men from Sweden Diet and Cancer cohort	3009	47–68	Cross-sectional
Kim *et al.* (2014)[14]	2005	Women and men from Third Korean National Health and Nutrition Examination Survey	4625	≥19	Cross-sectional
Mohindra *et al.* (2009)[25]	1998–1991	US adults from Louisiana Bogalusa Heart Study	504	19–28	Cross-sectional
Howarth *et al.* (2005)[17]	1994–1996	Women and men from US Continuing Survey of Food Intake	2685	Younger: 20–59 Older: 60–90	Cross-sectional
Ma *et al.* (2003)[9]	NA	American women and men	299	20–70	Cross-sectional
Mills *et al.* (2011)[40]	2008	American women	1099	40–60	Cross-sectional
Smith *et al.* (2012)[11]	2004–2006	Australian women and men	2775	26–36	Cross-sectional
Titan *et al.* (2011)[30]	1993 and 1997	Women and men from Norfolk cohort of EPIC	14 666	45–75	Cross-sectional
Yannakoulia *et al.* (2007)[32]	NA	Greek women	64 pre- and 50 postmenopausal	24–74	Cross-sectional
Murakami and Livingstone (2014)[38]	2000–2001	British women and men	1487	19–64	Cross-sectional
Berg *et al.* (2009)[31]	2001–2004	Swiss women and men	3610	25–74	Cross-sectional
Drummond *et al.* (1998)[8]	NA	Women and men workers of Scotland	95	Mean 20 (SD 55)	Cross-sectional
Marín-Guerrero *et al.* (2008)[33]	1999	Spanish women and men	34 974	25–64	Cross-sectional
Peixoto *et al.* (2007)[43]	2001	Brazilian women and men	1252	20–64	Cross-sectional
Ruidavets *et al.* (2002)[36]	1996–1997	French men	330	45–64	Cross-sectional
Teichmann *et al.* (2006)[39]	NA	Brazilian women	981	20–60	Cross-sectional
van der Heijden *et al.* (2007)[13]	1992–2002	Men from HPSF	20 064	46–81	Prospective: 10-year follow-up
Bachman *et al.* (2011)[26]	2006–2007	American women and men	257	18–65	Cross-sectional
Bertéus Forslund *et al.* (2005)[34]	Reference: 1994–1999 Obese: 1997–2001	Swiss women and men	Obese: 4470 Reference: 1092	Obese: 30–60 Reference: 37–60	Cross-sectional
Bertéus-Forslund *et al.* (2002)[35]	Obese: 1994–1999 Reference: NA	Swiss obese women	Obese: 83 Reference: 94	37–60	Cross-sectional
Metzner *et al.* (1997)[6]	1967–1969	American women and men	2028	35–69	Cross-sectional
Pearcey and de Castro (2002)[27]	NA	Women and men from research pool at Georgia State University	19 weight-gaining men and women and 19 weight-stable	NA	Cross-sectional
Reicks *et al.* (2014)[28]	2013	American women and men	2702	18–80	Cross-sectional
Amosa *et al.* (2001)[37]	1994	Polynesian and European women	82	18–27	Cross-sectional
Al-Isa (1999)[41]	1997–1998	Kuwait women and men university students	842	18–23	Cross-sectional

NA, not available; INTERMAP, International Study on Macro/Micronutrients and Blood Pressure; NHANES, National Health and Nutrition Examination Survey; NHEFS, NHANES Epidemiologic Follow-up Study; EPIC, European Prospective Investigation into Cancer and Nutrition; HPSF, Health Professionals Follow-up Study.

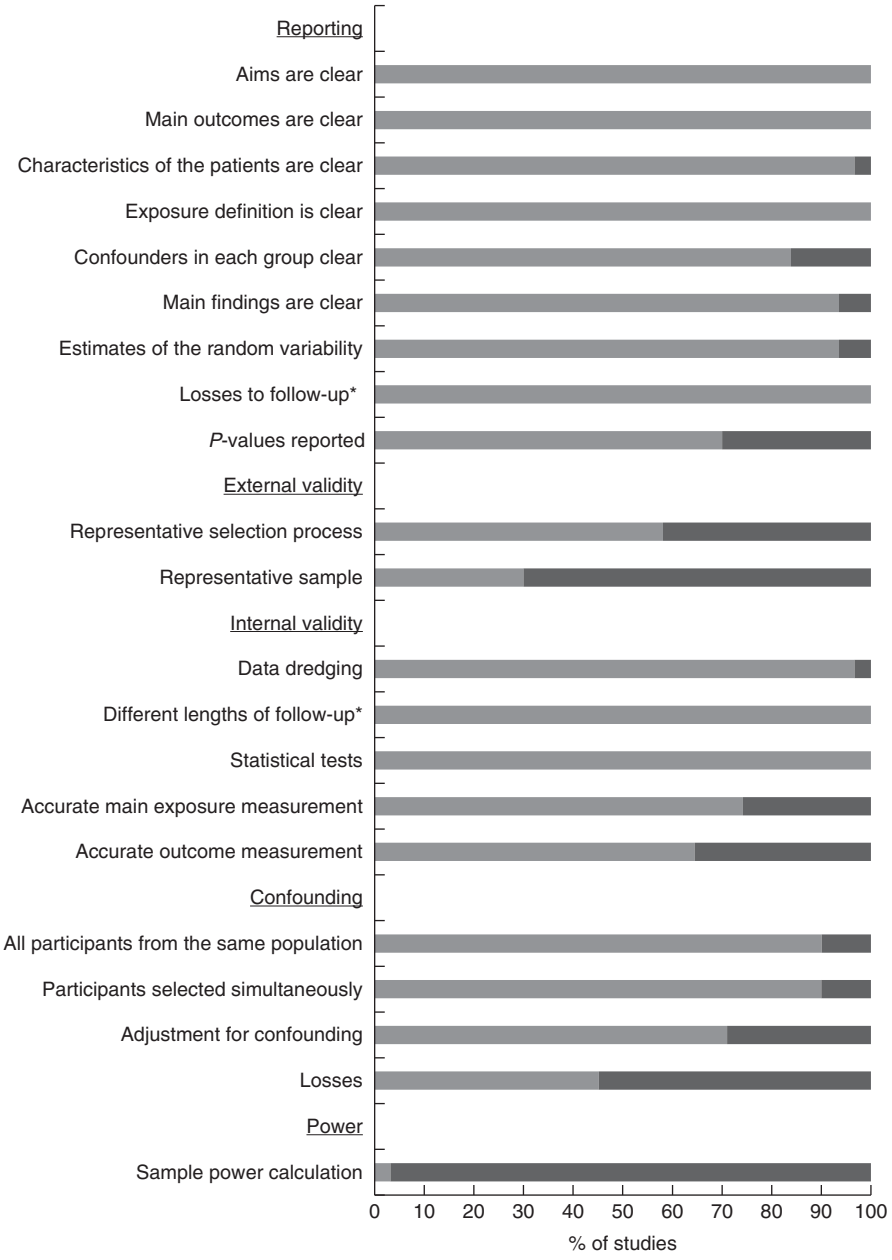

Fig. 2 Summary of quality assessment (■, low risk of bias; ■, risk of bias) of the studies (*n* 31) included in the present systematic literature review. *Items 'different lengths of follow-up' and 'losses to follow-up' were evaluated only in prospective studies

possible sociodemographic confounders (age, race/ethnicity, education and income)[15,39–42] and four of behavioural confounders (smoking, physical activity and alcohol intake)[15,39,41,42]. Investigation of energy intake as confounder was included in the analyses of only two studies[15,40] (Table 5).

Among the ten studies that showed analyses in men separately, five found inverse associations between high eating frequency and waist circumference[10,11,44] or waist-to-hip ratio[30,36] and seven found inverse associations for body weight[8,10,11,30,33,36,43,46]. All of these studies adjusted for physical activity and dietary characteristics in multiple analyses, but only two of them took into account dietary intake misreporting[10,36]. On the other hand, when the results were analysed only in women, no pattern was observed in the results.

Finally, when the results were analysed according to exposure and outcomes, no association pattern was observed.

Discussion

Our SLR focused on the association of eating frequency with body composition or body weight. We concluded that, to date, there is not sufficient evidence for establishing a clear association between eating frequency and

Table 3 Summary of the main results of studies that found an inverse association between eating frequency and body weight or body composition (*n* 14)

Study	Quality score (%)	Outcomes	Exposure assessment	Exposure classification	Statistical analysis	Results
Aljuraiban *et al.* (2015)[24]	94·7	BMI: continuous variable	No. of meals: main meals and snacks Assessment: 4 × 24 h recall	<4 *v.* 4 *v.* 5 *v.* ≥6 meals and continuous variable	BMI: generalized linear model adjusted for sex, age, population sample, educational level, PA, smoking, diet, dietary supplement and EI BMI difference: multiple linear regression model adjusted for sex, age, population sample and EI Energy misreporting was defined based on EI:EER and <2092 kJ/24 h (<500 kcal/24 h) or >20 920 kJ/24 h (>5000 kcal/24 h) for women and >33 472 kJ/24 h (>8000 kcal/24 h) for men	Men and women BMI (kg/m^2), mean (95 % CI): >4 meals: 29·0 (28·8, 29·5) 4 meals: 28·4 (28·0, 28·5) 5 meals: 28·1 (27·7, 28·4) ≥6 meals: 27·3 (26·8, 27·8) $P < 0·01$ BMI difference (kg/m^2): $\beta = -1·1$ (95 % CI −1·6, −0·7) $P < 0·0001$
Oliveira *et al.* (2009)[44]	89·5	Overweight: BMI ≥ 25·0 kg/m^2 WC risk category: men > 94 cm, women > 80 cm	Total no. of meals: main meals and snacks Assessment: simple question	≤3 *v.* ≥4 meals	Logistic regression model adjusted for sex, age, race, marital status, education, income, occupational status, self-rated health, PA, smoking, alcohol intake, parity, morbidities, dietary practices, frequency of fruits, vegetables, meats, sausages and derivatives intake	Men WC, OR (95 % CI): ≥4 *v.* ≤3 meals: 3·5 (1·3, 9·3) BMI: NS Women WC and BMI: NS
Holmback *et al.* (2010)[10]	84·2	Underweight: BMI < 18·5 kg/m^2 Normal weight: BMI = 18·5–25·0 kg/m^2 Overweight: BMI = 25·0–29·9 kg/m^2 Obesity: BMI ≥ 30 kg/m^2 WC increased risk: 80 cm (women), 94 cm (men) WC greatly increased risk: 88 cm (women), 102 cm (men)	No. of meals: main meals and snacks Assessment: meal pattern questionnaire	≤3 *v.* ≥6 meals 4–5 *v.* ≥6 meals	Logistic regression model adjusted for age, education, SES, smoking, alcohol intake, PA activity and EI Energy misreporting was defined based on EI:BMR	Men Obese, OR (95 % CI): ≥6 *v.* ≤3 meals: 2·4 (1·02, 5·7) ≥6 *v.* 4–5 meals: 1·1 (0·6, 2·2) WC greatly increased risk, OR (95 % CI): ≥6 *v.* ≤3 meals: 2·1 (1·0, 4·3) ≥6 *v.* 4–5 meals: 1·6 (0·9, 2·8) Women Obese: $P = 0·12$ WC greatly increased risk: $P = 0·07$
Mohindra *et al.* (2009)[25]	84·2	Overweight: BMI = ≥25·0–29·0 kg/m^2 Obesity: BMI ≥ 30·0 kg/m^2	Total no. of meals: main meals and snacks Assessment: 1 × 24 h dietary recall	Continuous variable	Logistic regression model adjusted for sex, age, ethnicity and EI	Men and women Normal *v.* overweight, OR (95 % CI): 0·9 (0·8, 1·1) Normal *v.* obese, OR (95 % CI): 0·8 (0·7, 1·0)
Titan *et al.* (2001)[30]	78·9	BMI (kg/m^2) and WHR: continuous variables	Total no. of meals: main meals, snacks, biscuits with coffee breaks Assessment: simple question	Continuous variable	Multiple linear regression adjusted for age, obesity, smoking, PA, intakes of alcohol, fat, protein and carbohydrate, and EI	Men BMI (kg/m^2): $\beta = -0·08$ (SE 0·03) $P = 0·02$ WHR: $\beta = -0·01$ (SE 0·01) $P = 0·42$ Women BMI (kg/m^2): $\beta = 0·05$ (SE 0·04) $P = 0·27$ WHR: $\beta = -0·01$ (SE 0·01) $P = 0·02$

Table 3 *Continued*

Study	Quality score (%)	Outcomes	Exposure assessment	Exposure classification	Statistical analysis	Results	
Ma *et al.* (2003)[9]	78·9	Obesity: BMI ≥ 30·0 kg/m^2	Total no. of meals: main meals and snacks Assessment: 10–15 × 24 h dietary recall	≤3 *v.* ≥4 meals	Logistic regression model adjusted for sex, age, PA and EI	Men and women ≤3*v.* ≥4 meals, OR (95 % CI): 0·5 (0·3, 0·9)	
Smith *et al.* (2012)[11]	78·9	BMI (kg/m^2) and WC (cm): continuous variables	Total no. of meals: main meals and snacks Assessment: meal pattern questionnaire	Continuous variable	Multiple linear regression adjusted for age, education, PA, alcohol intake, diet quality and overall dietary quality	Men WC (cm): $\beta = -0·7$ (95 % CI −1·1, −0·3) BMI (kg/m^2): $\beta = -0·3$ (95 % CI −0·4, −0·1)	Women WC (cm): $\beta = -0·1$ (95 % CI −0·5, 0·3) BMI (kg/m^2): $\beta = -0·1$ (95 % CI −0·2, 0·9)
Drummond *et al.* (1998)[8]	73·7	Body weight (kg), BMI (kg/m^2) and body fat %: continuous variables	Total no. of meals: any occasion when food was taken Assessment: 7 d food record	Continuous variable	Pearson's correlation without adjustments	Men Body weight: $r = -0·34$, $P = 0·03$ BMI: $P = 0·09$ Body fat %: $P = 0·17$	Women Body weight: $P = 0·41$ BMI: $P = 0·35$ Body fat %: $P = 0·43$
Marín-Guerrero *et al.* (2008)[33]	73·7	Obesity: BMI ≥30·0 kg/m^2	3–4 meals: 3 main meals and afternoon tea 2 meals: 2 main meals 1 meal: 1 main meal Snack: small amounts of food many times over the course of the day Assessment: meal pattern questionnaire	1 *v.* ≥3–4 meals ≤2 *v.* ≥3–4 meals 3–4 *v.* ≥5 meals	Logistic regression model adjusted for age, education, size of town of residence, marital status, PA, smoking, alcohol intake and health status	Men 3–4 *v.* 2 meals, OR (95 % CI): 1·6 (CI 1·4, 1·9) 3–4 *v.* 1 meals, OR (95 % CI): 1·4 (0·9, 2·1) 3–4 *v.* ≥5 meals, OR (95 % CI): 1·4 (1·0, 2·0)	Women 3–4 *v.* 2 meals, OR (95 % CI): 1·3 (1·0, 1·6) 3–4 *v.* 1 meals, OR (95 % CI): 1·1 (0·7, 1·8) 3–4 *v.* ≥5 meals, OR (95 % CI): 1·5 (1·2, 1·9)
Peixoto *et al.* (2007)[43]	73·7	BMI (kg/m^2): continuous variables	No. of meals Assessment: simple question	≤3 *v.* ≥4 meals	Multiple linear regression adjusted for age, income, smoking, alcohol intake, PA, frequency of meat and vegetable intake	Men <3 *v.* ≥4 meals: $\beta = -0·8$ (95 % CI −1·5, −2·1)	Women NS
Ruidavets *et al.* (2002)[36]	73·7	BMI (kg/m^2) and WHR: continuous variables	Total no. of meals: any food or drink intake providing energy Assessment: 3 d food record	1–2 *v.* ≥5 meals 3 *v.* ≥5 meals 3 *v.* ≥5 meals	Multiple linear regression adjusted for age, education, PA, smoking, habits of dieting and EI Energy misreporting was defined based on EI:BMR	Men BMI (kg/m^2): ≥5 meals: reference 4 meals: $\beta = 1·7$ (SE 0·7) 3 meals: $\beta = 1·7$ (SE 0·7) 1–2 meals: $\beta = 3·7$ (SE 1·1) $P = 0·05$ WHR: ≥5 meals: reference 4 meals: $\beta = 0·01$ (SE 0·01) 3 meals: $\beta = 0·01$ (SE 0·01) 1–2 meals: $\beta = 0·04$ (SE 0·02) $P < 0·01$	

Table 3 *Continued*

Study	Quality score (%)	Outcomes	Exposure assessment	Exposure classification	Statistical analysis	Results
Bachman *et al.* (2011)[26]	68·4	Overweight/obese (OW): BMI = 27·0–45·0 kg/m^2 Weight-loss maintainers (WLM): BMI > 25·0 kg/m^2 at some point in life, lost > 10 % of maximum body weight and maintained that for at least 5 years, and were weight-stable within the previous 2 years Normal weight (NW): BMI = 19·0–24·9 kg/m^2 at entry into the trial, never overweight or obese and were weight-stable within the previous 2 years	Main meals: breakfast, lunch and dinner Snack: any food eaten outside habitual meal times Assessment: 3 ×24 h dietary recalls	Continuous variable	Covariance analyses adjusted for sex, age, PA and Pearson correlation without adjustment.	Men and women Snacks, mean (SD): WLM: 1·9 (1·1) NW: 2·3 (1·1) OW: 1·5 (1·3) $P < 0·05$ Meals, mean (SD): WLM: 2·7 (0·4) NW: 2·7 (0·4) OW: 2·7 (0·5) $P > 0·05$ All participants BMI *v.* snacks: $r = -0·20$ $P < 0·01$
Metzner *et al.* (1977)[6]	68·4	Adiposity index: continuous variable	Total no. of meals: main meals and snacks Assessment: 1 × 24 h dietary recall	2 *v.* 6 meals	ANCOVA with energy (calories) per kilogram of ideal weight as the covariate	Men and women Mean adiposity index gets smaller as the number of meals increases from 2 to 6 (numerical results not shown)
Reicks *et al.* (2014)[28]	63·2	BMI (kg/m^2): continuous variable Self-reported	No. of meals: main meals and snacks Assessment: self-administered meal pattern questionnaire and meal pattern history	1–5 *v.* 6–10 *v.* ≥11 meals	Quantile regression of the median without adjustment	Men and women BMI (kg/m^2), median: 1–5 meals: 27·0 6–10 meals: 26·5 ≥11 meals: 26·2 $P = 0·008$

WC, waist circumference; WHR, waist-to-hip ratio; PA, physical activity; EI, energy intake; EER, estimated energy requirement; SES, socio-economic status; β, linear regression coefficient; NS, not statistically significant and *P* value not available; *r*, correlation coefficient.

Table 4 Summary of the main results of studies that found a positive association between eating frequency and body weight or body composition (*n* 6)

Study	Quality score (%)	Outcome	Exposure assessment	Exposure classification	Statistical methods and confounders	Results	
Murakami and Livingstone (2015)[12]	94·7	Overweight: BMI ≥ 25·0 kg/m^2 Central obesity: Men, WC >102 cm; women, WC > 88 cm	EFall: all eating occasions EFenergy: all eating occasions except for those providing no energy EF ≥ 50 kcal: all eating occasions providing ≥50 kcal (≥209 kJ) Assessment: 2 × 24 h dietary recalls	EFall and EFenergy: ≤3·5 *v.* 4 *v.* 4·5 *v.* 5 *v.* ≥5·5 meals EF ≥ 50 kcal: ≤3 *v.* 3·5 *v.* 5 *v.* 4·5 *v.* ≥5 meals	Logistic regression model adjusted for age, race/ethnicity, education, income, smoking, PA, intakes of alcohol, protein, fat, sugar and dietary fibre, EI: EER Energy misreporting was defined based on EI:EER	Overweight, Men EFall, OR (95 % CI): ≤3·5 meals: 1·0 (ref.) 4 meals: 1·2 (0·9, 1·6) 4·5 meals: 1·1 (0·9, 1·3) 5 meals: 1·2 (0·9, 1·5) ≥5·5: 1·4 (1·2, 1·7) *P* = 0·0006 EFenergy, OR (95 % CI): ≤3·5meals: 1·0 (ref.) 4 meals: 1·3 (1·0, 1·6) 4·5 meals: 1·1 (0·8, 1·3) 5 meals: 1·1 (0·9, 1·5) ≥5·5 meals: 1·4 (1·2, 1·7) *P* = 0·002 EF ≥ 50 kcal, OR (95 % CI): ≤3 meals: 1·0 (ref.) 3·5 meals: 1·4 (1·1, 1·7) 4 meals: 1·3 (1·0, 1·7) 4·5 meals: 1·4 (1·15, 1·8) ≥5meals: 1·5 (1·2, 1·9) *P* = 0·003	Overweight, Women EFall: *P* = 0·40 EFenergy: *P* = 0·56 EF ≥ 50 kcal, OR (95 % CI): ≤3 meals: 1·0 (ref.) 3·5 meals: 1·2 (0·9, 1·5) 4 meals: 1·3 (1·0, 1·7) 4·5 meals: 1·3 (1·0, 1·6) ≥ 5 meals: 1·4(1·2, 1·8) *P* = 0·001
						Central obesity, Men EFall, OR (95 % CI): ≤3·5 meals: 1·0 (ref.) 4 meals: 14 (0·9, 1·4) 4·5 meals: 1·1 (0·9, 1·4) 5 meals: 1·4 (1·1, 1·7) ≥5·5 meals: 1·4 (1·2, 1·7) *P* = 0·0001 EFenergy, OR (95 % CI): ≤3·5 meals: 1·0 (ref.) 4 meals: 1·1 (0·9, 1·4) 4·5 meals: 1·1 (0·9, 1·4) 5 meals: 1·4 (1·1, 1·7) ≥5·5 meals: 1·4 (1·1, 1·7) *P* = 0·0001 EF ≥ 50 kcal, OR (95 % CI): ≤3 meals: 1·0 (ref.) 3·5 meals: 1·0 (0·9, 1·2) 4 meals: 1·3 (1·0, 1·6) 4·5 meals: 1·4 (1·1, 1·8) ≥5 meals: 1·4 (1·1, 1·7) *P* = 0·002	Central obesity, Women EFall: *P* = 0·31 EFenergy: *P* = 0·52 EF ≥ 50 kcal, OR (95 % CI): ≤3 meals: 1·0 (ref.) 3·5 meals: 1·1 (0·9, 1·4) 4 meals: 1·4 (1·1, 1·7) 4·5 meals: 1·3 (1·0, 1·6) ≥ 5 meals: 1·3 (1·0, 1·6) *P* = 0·03
Howarth *et al.* (2007)[17]	78·9	BMI (kg/m^2): continuous variable Self-reported	No. of meals: breakfast, brunch, lunch, dinner, supper or snack Assessment: self-administered meal pattern questionnaire	≤3 *v.* 3·5–6 meals ≤3 *v.* >6 meals	Multiple linear regression adjusted for sex, age, ethnicity, education, income urbanity, geographic region, smoking, self-reported chronic disease, TV viewing, fibre and fat	Younger men and women ≤3 *v.* 3·5–6 meals: *β* = 0·37 *P* = 0·19 ≤3 *v.* >6 meals: *β* = 1·28 *P* = 0·006	Older men and women ≤3 *v.* 3·5–6 meals: *β* = 0·87 *P* = 0·022 ≤3 *v.* >6 meals: *β* = 2·32 *P* = 0·004

Table 4 *Continued*

Study	Quality score (%)	Outcome	Exposure assessment	Exposure classification	Statistical methods and confounders	Results	
Yannakoulia *et al.* (2007)[32]	78·9	BMI (kg/m^2), WC (cm), WHR and body fat %: continuous variables	Total no. of meals: any eating occasion when food or drink was taken Assessment: 3 d food record	Continuous variable	Multiple linear regression adjusted for age, PA and EI Energy misreporting was defined based on EI:BMR	Postmenopausal Body fat %: $\beta = 0{\cdot}41$ $P = 0{\cdot}01$ BMI, WC, WHR: $P < 0{\cdot}05$	Premenopausal Body fat %, BMI, WC, WHR: $P < 0{\cdot}05$
Murakami & Livingstone (2014)[38]	73·7	BMI (kg/m^2) and WC (cm): continuous variables	EFall: all eating occasions EFenergy: all eating occasions except for those providing no energy EF ≥ 210 kJ: all eating occasions providing ≥210 kJ Assessment: 7 d weighed food record	Continuous variable	Multiple linear regression adjusted for age, social class, smoking, PA, intakes of protein, fat, total sugar, alcohol, dietary fibre and EI:EER Energy misreporting was defined based on EI:EER	Men EFall BMI (kg/m^2): $\beta = 0{\cdot}19$ (SE 0·08) $P = 0{\cdot}03$ WC (cm): $\beta = 0{\cdot}35$ (SE 0·23) $P = 0{\cdot}12$ EFenergy BMI (kg/m^2): $\beta = 0{\cdot}23$ (SE 0·09) $P \leq 0{\cdot}01$ WC (cm): $\beta = 0{\cdot}47$ (SE 0·25) $P = 0{\cdot}07$ EF ≥ 210 kJ BMI (kg/m^2): $\beta = 0{\cdot}37$ (SE 0·13) $P = 0{\cdot}004$ WC (cm): $\beta = 0{\cdot}80$ (SE 0·35) $P = 0{\cdot}02$	Women EFall BMI (kg/m^2): $\beta = 0{\cdot}05$ (SE 0·11) $P = 0{\cdot}67$ WC (cm): $\beta = 0{\cdot}12$ (SE 0·25) $P = 0{\cdot}63$ EFenergy BMI (kg/m^2): $\beta = 0{\cdot}06$ (SE 0·13) $P = 0{\cdot}63$ WC (cm): $\beta = 0{\cdot}14$ (SE 0·29) $P = 0{\cdot}63$ EF ≥ 210 kJ BMI (kg/m^2): $\beta = 0{\cdot}61$ (SE 0·21) $P = 0{\cdot}004$ WC (cm): $\beta = 1{\cdot}22$ (SE 0·47) $P = 0{\cdot}01$
Bertéus Forslund *et al.* (2005)[34]	68·4	Reference: BMI < 30·0 kg/m^2 Obesity: BMI ≥ 30·0 kg/m^2	Total no. of meals: main meal, light meal/breakfast, snacks or drink-only Assessment: self-administered meal pattern questionnaire	Continuous variable	Logistic regression model adjusted for age and EI	Men and women Reference *v.* obesity, OR (95 % CI): 1·2 (1·1, 1·3)	
Bertéus Forslund *et al.* (2002)[35]	68·4	Obesity: BMI ≥ 30·0 kg/m^2	Total no. of meals: main meals, light meal/breakfast, snacks or drink-only Assessment: self-administered meal pattern questionnaire	Continuous variable	Two-sample *t* test without adjustment	Women Obese *v.* Reference, meals (mean): 6·1 *v.* 5·2 $P < 0{\cdot}0001$	

WC, waist circumference; WHR, waist-to-hip ratio; PA, physical activity; EI, energy intake; EER, estimated energy requirement; TV, television; ref., reference category; β, linear regression coefficient.

Table 5 Summary of the main results of studies that did not find an association between eating frequency and body weight or body composition (*n* 10)

Study	Quality score (%)	Outcome	Exposure assessment	Exposure classification	Confounders	Results
Gigante *et al.* (1997)[42]	89·5	Obesity: BMI ≥ 30·0 kg/m^2	No. of meals: breakfast, lunch, dinner and snacks. Assessment: meal pattern questionnaire	≤3 *v.* 4–6 meals	Logistic regression model adjusted for sex, age, race, marital status, education, income, occupational status, self-rated health, PA, smoking, alcohol intake, parental overweight status, parity and morbidities	Men and women <3 *v.* 4–6 meals, OR (95 % CI): 0·8 (0·4, 1·7)
Karatzi *et al.* (2015)[29]	89·5	BMI (kg/m^2) and WC (cm): continuous variables	No. of meals: food or drink Assessment: 3 d food record	Continuous variable	Multiple linear regression model adjusted for age, intakes of carbohydrates and fat, and EI	BMI (kg/m^2), one-meal increase: $\beta = -0{\cdot}01$ (95 % CI −0·01, +0·01) WC (cm), one-meal increase: $\beta = -0{\cdot}01$ (95 % CI −0·01, +0·01)
Kant *et al.* (1995)[15]	85·7	Weight (kg): continuous variable	Total no. of meals: main meals and snacks Assessment: 1971: 1 × 24 h dietary recall 1982: simple question	Continuous variable	Multiple linear regression model adjusted for age, education, race, baseline BMI, length of follow-up, smoking, alcohol intake, PA, parity, morbidity and EI	Men: $\beta = 0{\cdot}08$ (SE 0·13) $P = 0{\cdot}52$ Women: $\beta = 0{\cdot}2299$ (SE 0·15) $P = 0{\cdot}13$
Kim *et al.* (2014)[14]	84·2	BMI (kg/m^2) and WC (cm): continuous variables	No. of meals Assessment: simple question	≤2 *v.* 3 *v.* 4 *v.* ≥5 meals	General linear model without adjustments	WC (cm), mean (SD): ≤2 meals: 79·9 (0·6) 3 meals: 81·2 (0·3) 4 meals: 80·7 (0·3) ≥5 meals: 79·5 (0·4) $P = 0{\cdot}007$ BMI (kg/m^2), mean (SD): ≤2 meals: 23·4 (0·2) 3 meals: 23·6 (0·1) 4 meals: 23·6 (0·1) ≥5 meals 23·3 (0·1) $P = 0{\cdot}212$
Mills *et al.* (2011)[40]	78·9	Overweight: BMI ≥ 25·0–29·9 kg/m^2 Obesity: BMI ≥ 30·0 kg/m^2 Self-reported	Total no. of meals: breakfast, lunch, dinner and snack Assessment: 1 d food record	Continuous variable	Logistic regression model adjusted for age, income, marital status, race, education, menopausal status and EI Energy misreporting was defined based on EI:BMR	Men and women Meals Normal *v.* overweight/obesity, OR (95 % CI): 0·9 (0·7, 1·2) Snacking Normal *v.* overweight/obesity, OR (95 % CI): 1·0 (0·7, 1·2)
Berg *et al.* (2009)[31]	73·7	Reference: BMI < 30·0 kg/m^2 Obesity: BMI ≥ 30·0 kg/m^2	Total no. of meals (1–8): morning coffee, breakfast, between-meal snack, lunch, between-meal snack, dinner, supper and night meal Assessment: self-administered meal pattern questionnaire	Continuous variable	Logistic regression model adjusted for sex, age, smoking and PA	Men and women Reference *v.* obesity, OR (95 % CI): 0·9 (0·9, 1·0)
Teichmann *et al.* (2006)[39]	73·7	Obesity: BMI ≥ 30·0 kg/m^2	Total no. of meals: main meals and snacks Assessment: simple question	1–2 *v.* 3 meals 1–2 *v.* 4 meals 1–2 *v.* ≤5 meals	Poisson regression model adjusted for sex, age, race, marital status, education, income, occupational status, self-rated health, PA, smoking, alcohol intake, parental overweight status, parity and morbidities	Women Obesity, PR (95 % CI): 1–2 meals: 1·0 (ref.) 3 meals: 1·7 (0·9, 3·2) 4 meals: 1·3 (CI 0·7, 2·5) ≤5 meals: 0·9 (0·5, 1·8)

Table 5 *Continued*

Study	Quality score (%)	Outcome	Exposure assessment	Exposure classification	Confounders	Results
Pearcey and de Castro (2002)[27]	63·2	Weight-gaining: a weight gain of >5 % during the previous 6 months Weight-stable: a weight gain of <5 % during the previous 6 months	Total no. of meals: main meals and snacks Assessment: 7 d food record	Continuous variable	Student's *t* test without adjustments	Men and women Weight-stable group, mean (SE): 3·30 (0·14) $P < 0·05$ Weight-gaining group, mean (SE): 3·52 (0·13) $P < 0·05$
Amosa *et al.* (2001)[37]	57·9	Obesity: BMI $\geq$30·0 kg/m^2	Total no. of meals in the seven records: breakfast, lunch, dinner and snacks Assessment: 7 d food record	Continuous variable	Mann–Whitney test without adjustments	European women: Meals, median (range): Non-obese: 31 (20–40) Obese: 29 (13–39) $P > 0·05$ Polynesian women: Meals, median (range): Non-obese: 31 (17–58) Obese: 28 (17–53) $P > 0·05$
Al-Isa *et al.* (1999)[41]	42·1	Non-obesity: BMI $\leq$ 25·0 kg/m^2 Obesity I: BMI = 25·0–30·0 kg/m^2 Obesity II: BMI $\geq$ 30·0 kg/m^2	No. of the main meals: 1, 2 or 3 Assessment: simple question	1 *v.* 2 *v.* 3 meals	Logistic regression model adjusted for sex, age, marital status, obesity among parents, parents' education, parents' occupation, dieting, last dental or physical check-up, education, number of male, female and total siblings, college major, number of obese relatives, number of people residing at home, number of servants, birth order, countries prefer visiting, family income, chronic disease, PA and eating between meals	Men and women $P > 0·05$

WC, waist circumference; PA, physical activity; EI, energy intake; β, linear regression coefficient; PR: prevalence ratio; ref., reference category.

body composition or body weight. However, among men, a potential protective effect of high eating frequency on these outcomes was observed.

The findings should be interpreted in light of the methodological characteristics of the articles included. First, the outcome and exposure measurement might not be accurate in some studies. Moreover, the outcome measurement varied among the studies, thereby limiting the comparability among them. For example, the role of eating frequency on body weight might be different from the one it has on central adiposity. With respect to exposure, different methods were used for data collection. Some studies used methods such as multiple recalls or food diaries and meal pattern questionnaires, and may be more accurate than others, such as simple questions, for eating frequency assessment. Although dietary records and 24 h dietary recalls are subject to misreporting, particularly under-reporting[47], there is no information about the validity of most of the meal pattern questionnaires and simple questions used in these studies. Furthermore, different cut-offs were used to determine high or low eating frequency. Only six authors classified eating frequency according to the three major meals (breakfast, lunch and dinner) and compared intake of three meals with intake of a greater number of meals. Other studies assessed the exposure as a continuous variable or compared the extremes of eating frequency (e.g. two *v.* six meals per day).

Second, the results are based mostly on cross-sectional studies, with only two studies selected having a longitudinal design, which should be considered a limitation in the current available literature. Longitudinal studies are well known for being a better study design to investigate the temporal relationship between the exposure and change in outcome status. The issue of reverse causality is especially important in this case, because people skip meals, thus reducing eating frequency, when they become overweight in an attempt to lose weight or to prevent further gain[47].

Finally, obesity is a multifactorial disorder arising from genetic, environmental, socio-economic and behavioural factors. These differ in their respective contributions to the obesity epidemic[48]. In this sense, another methodological issue that is very important in this type of epidemiological investigation is the inclusion of main confounders and mediators in the analysis. A confounding variable is an extraneous variable in a statistical model that correlates (positively or negatively) with both the exposure and the outcome variable; meanwhile, a mediator factor is a variable that occurs in the causal pathway between the exposure and the outcome[49]. The majority of studies compiled in the present SLR applied multiple analyses adjusted for potential confounders, such as sex, age, education, income, smoking, physical activity and alcohol intake.

The effect of dietary characteristics (energy intake and quality of diet) in the causal pathway linking eating frequency to body weight and body composition was adjusted for in the multiple analyses of most studies. However, in order to understand the role of a variable in a causal chain, sometimes it is more informative to stratify the analyses according to this variable, rather than adjusting for it in multiple analyses. In this sense, only one study investigated the mediation effect of energy intake by stratifying the analysis according to it[17]. However, several studies included in the present SLR showed that higher eating frequency is positively associated with energy intake[10,17,30,34,40]. In Holmback *et al.*[10], eating frequency and carbohydrate energy percentage, as well as relative fibre intake, increased with higher eating frequency; while the energy percentage from fat, protein and alcohol decreased. Bertéus Forslund *et al.*[34] found that sweet and fatty food groups were associated with snacking and contributed considerably to energy intake. Regarding quality of diet, Mills *et al.*[40] showed that intakes of fruit and vegetables, whole grains, dietary fibre, dairy and added sugars also increased as eating frequency increased. Other aspects of diet, such as meal timing, are also important in the control and reduction of body weight, total body fat and visceral fat[50].

In our SLR, fourteen studies reported an inverse association between eating frequency and body weight or body composition. Murakami and Livingstone and Bellisle *et al.*[12,51] have called attention to this apparent inverse relationship between eating frequency and adiposity measures, suggesting it is an artifact that in part can be attributed to the under-reporting of eating frequency concomitant with the under-reporting of energy intake by overweight or obese subjects. In this regard, Murakami and Livingstone's[12] study showed the importance of evaluating energy intake misreporting when examining the association between eating frequency and overweight/obesity and central obesity. In their study, energy intake misreporting was evaluated based on ratio of energy intake to estimated energy requirement (EI:EER). In the multiple analyses, without taking into account energy intake or EI:EER, eating frequency showed an inverse or null association with the outcomes. However, after full adjustment including EI:EER, a completely different picture emerged: eating frequency was positively associated with overweight/obesity and central obesity. Only three studies that reported an inverse association between eating frequency and body weight or body composition in the present SLR took into account under-reporting of energy intake and/or eating frequency; and three of the six studies that found a positive association between eating frequency and outcomes took into account misreporting of energy intake.

Physical activity also plays a role in the association that links eating frequency with body weight and body composition[52]. Physical activity might be a potential confounder in this association, since physical activity practice may improve diet quality[53,54]; although physical

activity has been well described in the literature as an independent factor in the control and reduction of body weight, total body fat and visceral fat[55,56].

Even though it was not the objective of our SLR to investigate differences between sexes, when analysing the results of the articles included, a potential protective effect of high eating frequency on the outcomes was observed among men. In general, increased eating frequency has been postulated to increase metabolism[4], appetite control and food intake[46,57], and to improve glucose and insulin control[58,59]. However, this difference could be due to the fact that men who have high eating frequency also have a healthier lifestyle, including practice of physical activity and healthier eating habits, which results in reduced body fat and waist circumference. In Holmback *et al.*[10], a high fibre intake was the clearest diet-quality indicator associated with a high eating frequency among men. In two other studies, men were more physically active than women, which may help to explain the protective effect found only in men[11,43]. However, only in Smith *et al.*'s study[11] was physical activity measured by a direct method (pedometer). In the other two studies[10,43], self-reported measurements were used, which can be inaccurate. In addition, there is evidence that energy intake compensation is poor in women, a factor normally associated with obesity[60]. Drummond *et al.*[8] showed that men compensated for extra eating occasions by reducing the mean energy per eating episode. Although all studies adjusted for physical activity and dietary characteristics in the multiple analyses, it is possible that residual confounding of both lifestyle factors could contribute to the results.

The present review is the first SLR of observational studies that examines specifically the association of eating frequency with body weight and body composition in adults with a systematic approach. Several narrative reviews have been conducted[2,52,61–63] and these concluded that the evidence available to suggest the presence of an association between eating frequency and weight, BMI and body fatness is limited. In addition, a meta-analysis evaluating experimental research suggested that eating frequency is positively associated with reductions in fat mass and body fat percentage, as well as an increase in fat-free mass; however, the positive findings were the product of a single study and need to be interpreted with circumspection[7].

Considering there is contradictory evidence about the association between eating frequency and body weight, it is important to assess the whole body of evidence about this topic and in particular to do so systematically. In addition, researchers have suggested that nutrition policy decisions will have to be made using the totality of the available evidence[64]. It is almost inevitable that causal chains in nutrition outcomes involve long periods of latency, complex individual variability in the biological response, and cultural, economic and geographic influences; aspects that observational studies may help to understand, since observational studies for food habits indicate what happens over a lifetime of consumption[45,64,65].

In this respect, at the same time as we encourage the conduct of more clinical trials to help to examine and potentially determine this causal relationship, future high-quality observational studies are needed to understand the role of eating frequency on loss and maintenance of weight and body composition and to guide clinical recommendations. However, evaluating eating behaviour is also a complex task and we demonstrated substantial heterogeneity in the methodological quality of studies. Thus, in Table 6, we call attention to some important methodological issues that

Table 6 Methodological recommendations for future observational studies

Design	Long-term longitudinal studies
Sample characteristics	Population-based studies with representative samples and sample size to perform analysis by sex
Exposure assessment	Accurate methods should be used for data collection, such as recalls and food diaries, or standardized pattern of meal questionnaires, which can measure food habits; and misreporting should be taken into account in the analyses
	It is necessary to standardize the method of classifying the eating occasion (main meal and snacks) in order to use a reproducible method between studies
	The analysis should compare the intake of the three major meals (breakfast, lunch and dinner) with a greater number of meals, in order to propose health recommendations based on results
Outcome assessment	Anthropometric measurements of weight, height to determine BMI, as well as abdominal adiposity measurements (waist, abdominal and hip circumferences), should be obtained using standard techniques, avoiding using self-reported measures, especially in cross-sectional studies
	In addition to BMI and abdominal adiposity, measures of body composition should be included, such as fat mass and fat-free mass, in order to understand the role of eating frequency on body composition. Body composition should be evaluated by objective measurements, such as dual-energy X-ray absorptiometry, impedance or densitometry, when it is possible
Confounders and mediators	The analyses should be stratified by sex, if the sample size allows for this, in order to explore whether there are differences in the association between eating frequency and adiposity according to sex
	Should apply multivariate analyses or other statistical methods to control for possible confounding, such as by age, education, race/ethnicity, marital status, income, physical activity, smoking and sleep duration
	When associations between eating frequency and body weight or body composition are found, it is very important to investigate the role of food habits, with special attention to energy intake, quality of diet, meal composition (macro- and micronutrients) and meal timing as mediator factors. Furthermore, they should be appropriately measured and under-reporting bias should be taken in account

should be considered in future observational studies. It is necessary to conduct studies with long-term longitudinal design and representative samples. Outcome and exposure should be measured with accurate methods and classified based on clinically relevant aspects. Moreover, it is important that statistical analyses should be stratified by sex, if the sample size allows, and to include the potentially relevant confounders and mediators, with special attention to nutrients and energy intake, and these should be appropriately measured.

Acknowledgements

Financial support: This study was supported by the Foundation for Research Support of the State of Rio Grande do Sul (grant number 1220-2551/13-3). M.T.A.O. and G.K. received research productivity grants from the Brazilian National Council for Scientific and Technological Development (grant numbers 307257/2013-4 and 304182/2013-3). *Conflict of interest:* none. *Authorship:* R.C. and M.T.A.O. conceptualized the study. R.C. and A.S.G. completed the searches, abstract/title screening, data extraction and quality assessment. R.C. and M.T.A.O. drafted the manuscript. G.K. and P.I.C.L. assisted in drafting and revision of the manuscript. All authors read and approved the final manuscript. *Ethics of human subject participation:* Not applicable.

References

1. Finucane MM, Stevens GA, Cowan MJ *et al.* (2011) National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. *Lancet* **377**, 557–567.
2. Seagle HM, Strain GW, Makris A *et al.* (2009) Position of the American Dietetic Association: weight management. *J Am Diet Assoc* **109**, 330–346.
3. Fabry P, Hejl Z, Fodor J *et al.* (1964) The frequency of meals. Its relation to overweight, hypercholesterolaemia, and decreased glucose-tolerance. *Lancet* **2**, 614–615.
4. Jenkins DJ, Wolever TM, Vuksan V *et al.* (1989) Nibbling versus gorging: metabolic advantages of increased meal frequency. *N Engl J Med* **321**, 929–934.
5. Verboeket-van de Venne WP & Westerterp KR (1991) Influence of the feeding frequency on nutrient utilization in man: consequences for energy metabolism. *Eur J Clin Nutr* **45**, 161–169.
6. Metzner HL, Lamphiear DE, Wheeler NC *et al.* (1977) The relationship between frequency of eating and adiposity in adult men and women in the Tecumseh Community Health Study. *Am J Clin Nutr* **30**, 712–715.
7. Schoenfeld JB, Aragon AA & Krieger JW (2015) Effects of meal frequency on weight loss and body composition: a meta-analysis. *Nutr Rev* **73**, 69–82.
8. Drummond SE, Crombie NE, Cursiter MC *et al.* (1998) Evidence that eating frequency is inversely related to body weight status in male, but not female, non-obese adults reporting valid dietary intakes. *Int J Obes Relat Metab Disord* **22**, 105–112.
9. Ma Y, Bertone ER, Stanek EJ 3rd *et al.* (2003) Association between eating patterns and obesity in a free-living US adult population. *Am J Epidemiol* **158**, 85–92.
10. Holmback I, Ericson U, Gullberg B *et al.* (2010) A high eating frequency is associated with an overall healthy lifestyle in middle-aged men and women and reduced likelihood of general and central obesity in men. *Br J Nutr* **104**, 1065–1073.
11. Smith KJ, Blizzard L, McNaughton SA *et al.* (2012) Daily eating frequency and cardiometabolic risk factors in young Australian adults: cross-sectional analyses. *Br J Nutr* **108**, 1086–1094.
12. Murakami K & Livingstone MB (2015) Eating frequency is positively associated with overweight and central obesity in US adults. *J Nutr* **145**, 2715–2724.
13. van der Heijden AA, Hu FB, Rimm EB *et al.* (2007) A prospective study of breakfast consumption and weight gain among US men. *Obesity (Silver Spring)* **15**, 2463–2469.
14. Kim S, Park GH, Yang JH *et al.* (2014) Eating frequency is inversely associated with blood pressure and hypertension in Korean adults: analysis of the Third Korean National Health and Nutrition Examination Survey. *Eur J Clin Nutr* **68**, 481–489.
15. Kant AK, Schatzkin A, Graubard BI *et al.* (1995) Frequency of eating occasions and weight change in the NHANES I Epidemiologic Follow-up Study. *Int J Obes Relat Metab Disord* **19**, 468–474.
16. Edelstein SL, Barrett-Connor EL, Wingard DL *et al.* (1992) Increased meal frequency associated with decreased cholesterol concentrations; Rancho Bernardo, CA, 1984–1987. *Am J Clin Nutr* **55**, 664–669.
17. Howarth NC, Huang TT, Roberts SB *et al.* (2007) Eating patterns and dietary composition in relation to BMI in younger and older adults. *Int J Obes (Lond)* **31**, 675–684.
18. Westerterp-Plantenga MS, Kovacs EM & Melanson KJ (2002) Habitual meal frequency and energy intake regulation in partially temporally isolated men. *Int J Obes Relat Metab Disord* **26**, 102–110.
19. Taylor MA & Garrow JS (2001) Compared with nibbling, neither gorging nor a morning fast affect short-term energy balance in obese patients in a chamber calorimeter. *Int J Obes Relat Metab Disord* **25**, 519–528.
20. Moher D, Shamseer L, Clarke M *et al.* (2015) Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement. *Syst Rev* **4**, 1.
21. Stroup DF, Berlin JA, Morton SC *et al.* (2000) Meta-analysis of observational studies in epidemiology: a proposal for reporting. Meta-analysis Of Observational Studies in Epidemiology (MOOSE) group. *JAMA* **283**, 2008–2012.
22. Higgins JPT & Green S (editors) (2011) Cochrane Handbook for Systematic Reviews of Interventions Version 5.1.0 [updated March 2011]. http://handbook.cochrane.org/ (accessed February 2015).
23. Monteiro PO & Victora CG (2005) Rapid growth in infancy and childhood and obesity in later life – a systematic review. *Obes Rev* **6**, 143–154.
24. Aljuraiban GS, Chan Q, Oude Griep LM *et al.* (2015) The impact of eating frequency and time of intake on nutrient quality and body mass index: the INTERMAP Study, a population-based study. *J Acad Nutr Diet* **115**, 528–536.e1.
25. Mohindra NA, Nicklas TA, O'Neil C E *et al.* (2009) Eating patterns and overweight status in young adults: the Bogalusa Heart Study. *Int J Food Sci Nutr* **60**, Suppl. 3, 14–25.
26. Bachman JL, Phelan S, Wing RR *et al.* (2011) Eating frequency is higher in weight loss maintainers and normal-weight individuals than in overweight individuals. *J Am Diet Assoc* **111**, 1730–1734.
27. Pearcey SM & de Castro JM (2002) Food intake and meal patterns of weight-stable and weight-gaining persons. *Am J Clin Nutr* **76**, 107–112.

28. Reicks M, Degeneffe D, Rendahl A *et al.* (2014) Associations between eating occasion characteristics and age, gender, presence of children and BMI among US adults. *J Am Coll Nutr* **33**, 315–327.
29. Karatzi K, Yannakoulia M, Psaltopoulou T *et al.* (2015) Meal patterns in healthy adults: inverse association of eating frequency with subclinical atherosclerosis indexes. *Clin Nutr* **34**, 302–308.
30. Titan SM, Bingham S, Welch A *et al.* (2001) Frequency of eating and concentrations of serum cholesterol in the Norfolk population of the European prospective investigation into cancer (EPIC-Norfolk): cross sectional study. *BMJ* **323**, 1286–1288.
31. Berg C, Lappas G, Wolk A *et al.* (2009) Eating patterns and portion size associated with obesity in a Swedish population. *Appetite* **52**, 21–26.
32. Yannakoulia M, Melistas L, Solomou E *et al.* (2007) Association of eating frequency with body fatness in pre- and postmenopausal women. *Obesity (Silver Spring)* **15**, 100–106.
33. Marín-Guerrero AC, Gutierrez-Fisac JL, Guallar-Castillon P *et al.* (2008) Eating behaviours and obesity in the adult population of Spain. *Br J Nutr* **100**, 1142–1148.
34. Bertéus Forslund BH, Torgerson JS, Sjostrom L *et al.* (2005) Snacking frequency in relation to energy intake and food choices in obese men and women compared to a reference population. *Int J Obes (Lond)* **29**, 711–719.
35. Bertéus-Forslund H, Lindroos AK, Sjostrom L *et al.* (2002) Meal patterns and obesity in Swedish women – a simple instrument describing usual meal types, frequency and temporal distribution. *Eur J Clin Nutr* **56**, 740–747.
36. Ruidavets JB, Bongard V, Bataille V *et al.* (2002) Eating frequency and body fatness in middle-aged men. *Int J Obes Relat Metab Disord* **26**, 1476–1483.
37. Amosa T, Rush E & Plank L (2001) Frequency of eating occasions reported by young New Zealand Polynesian and European women. *Pac Health Dialog* **8**, 59–65.
38. Murakami K & Livingstone MB (2014) Eating frequency in relation to body mass index and waist circumference in British adults. *Int J Obes (Lond)* **38**, 1200–1206.
39. Teichmann L, Olinto MTA, Costa JSD *et al.* (2006) Risk factors associated with overweight and obesity in women living in São Leopoldo, RG. *Rev Bras Epidemiol* **9**, 360–373.
40. Mills JP, Perry CD & Reicks M (2011) Eating frequency is associated with energy intake but not obesity in midlife women. *Obesity (Silver Spring)* **19**, 552–559.
41. Al-Isa AN (1999) Obesity among Kuwait University students: an explorative study. *J R Soc Promot Health* **119**, 223–227.
42. Gigante DP, Barros FC, Post CLA *et al.* (1997) Prevalence and risk factors of obesity in adults. *Rev Saude Publica* **31**, 236–246.
43. Peixoto Mdo R, Benicio MH & Jardim PC (2007) The relationship between body mass index and lifestyle in a Brazilian adult population: a cross-sectional survey. *Cad Saude Publica* **23**, 2694–2740.
44. Oliveira LP, Assis AM, Silva Mda C *et al.* (2009) Factors associated with overweight and abdominal fat in adults in Salvador, Bahia State, Brazil. *Cad Saude Publica* **25**, 570–582.
45. Blumberg J, Heaney RP, Huncharek M *et al.* (2010) Evidence-based criteria in the nutritional context. *Nutr Rev* **68**, 478–484.
46. Leidy HJ & Campbell WW (2011) The effect of eating frequency on appetite control and food intake: brief synopsis of controlled feeding studies. *J Nutr* **141**, 154–157.
47. Summerbell CD, Moody RC, Shanks J *et al.* (1996) Relationship between feeding pattern and body mass index in 220 free-living people in four age groups. *Eur J Clin Nutr* **50**, 513–519.
48. Cohen DA (2008) Obesity and the built environment: changes in environmental cues cause energy imbalances. *Int J Obes (Lond)* **32**, Suppl. 7, S137–S142.
49. Rothman K & Lash T (2008) *Modern Epidemiology*, 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins.
50. Jakubowicz D, Froy O, Wainstein J *et al.* (2012) Meal timing and composition influence ghrelin levels, appetite scores and weight loss maintenance in overweight and obese adults. *Steroids* **77**, 323–331.
51. Bellisle F, McDevitt R & Prentice AM (1997) Meal frequency and energy balance. *Br J Nutr* **77**, Suppl. 1, S57–S70.
52. Cohen DA (2008) Obesity and the built environment: changes in environmental cues cause energy imbalances. *Int J Obes (Lond)* **32**, Suppl. 7, S137–S142.
53. Holmes MD, Chen WY, Hankinson SE *et al.* (2009) Physical activity's impact on the association of fat and fiber intake with survival after breast cancer. *Am J Epidemiol* **170**, 1250–1256.
54. Pearson N & Biddle SJ (2011) Sedentary behavior and dietary intake in children, adolescents, and adults. A systematic review. *Am J Prev Med* **41**, 178–188.
55. Ross R & Janssen I (2001) Physical activity, total and regional obesity: dose–response considerations. *Med Sci Sports Exerc* **33**, 6 Suppl., S521–S527.
56. Slentz CA, Houmard JA & Kraus WE (2009) Exercise, abdominal obesity, skeletal muscle, and metabolic risk: evidence for a dose response. *Obesity (Silver Spring)* **17**, Suppl. 3, S27–S33.
57. Leidy HJ, Armstrong CL, Tang M *et al.* (2010) The influence of higher protein intake and greater eating frequency on appetite control in overweight and obese men. *Obesity (Silver Spring)* **18**, 1725–1732.
58. Schwarz NA, Rigby BR, La Bounty P *et al.* (2011) A review of weight control strategies and their effects on the regulation of hormonal balance. *J Nutr Metab* **2011**, 15.
59. Farshchi HR, Taylor MA & Macdonald IA (2005) Beneficial metabolic effects of regular meal frequency on dietary thermogenesis, insulin sensitivity, and fasting lipid profiles in healthy obese women. *Am J Clin Nutr* **81**, 16–24.
60. Lissner L, Levitsky DA, Strupp BJ *et al.* (1987) Dietary fat and the regulation of energy intake in human subjects. *Am J Clin Nutr* **46**, 886–892.
61. Kulovitz MG, Kravitz LR, Mermier C *et al.* (2014) Potential role of meal frequency as a strategy for weight loss and health in overweight or obese adults. *Nutrition* **30**, 386–392.
62. La Bounty PM, Campbell BI, Wilson J *et al.* (2011) International Society of Sports Nutrition position stand: meal frequency. *J Int Soc Sports Nutr* **8**, 4.
63. Hutfless S, Gudzune KA, Maruthur N *et al.* (2013) Strategies to prevent weight gain in adults: a systematic review. *Am J Prev Med* **45**, e41–e51.
64. Mitchell HL, Aggett PJ, Richardson DP *et al.* (2011) Food & health forum meeting: evidence-based nutrition. *Br J Nutr* **105**, 322–328.
65. Mann JI (2010) Evidence-based nutrition: does it differ from evidence-based medicine? *Ann Med* **42**, 475–486.