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The frequency of family meals and nutritional health in children: a meta-analysis

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Summary

Findings on the relationship between family meal frequency and children's nutritional health are inconsistent. The reasons for these mixed results have to date remained largely unexplored. This systematic review and meta-analysis of 57 studies (203,706 participants) examines (i) the relationship between family meal frequency and various nutritional health outcomes and (ii) two potential explanations for the inconsistent findings: sociodemographic characteristics and mealtime characteristics. Separate meta-analyses revealed significant associations between higher family meal frequency and better overall diet quality ($r = 0.13$), more healthy diet ($r = 0.10$), less unhealthy diet ($r = -0.04$) and lower body mass index, BMI ($r = -0.05$). Child's age, country, number of family members present at meals and meal type (i.e. breakfast, lunch or dinner) did not moderate the relationship of meal frequency with healthy diet, unhealthy diet or BMI. Socioeconomic status only moderated the relationship with BMI. The findings show a significant relationship between frequent family meals and better nutritional health – in younger and older children, across countries and socioeconomic groups, and for meals taken with the whole family vs. one parent. Building on these findings, research can now target the causal direction of the relationship between family meal frequency and nutritional health.

Keywords: Body mass index, children, diet, family meal.

Introduction

Childhood obesity is a serious health condition with short-term and long-term risks to both psychological and physical health, such as low self-esteem and a higher risk of developing asthma, diabetes and cardiovascular disease at a young age. Childhood obesity also strongly predicts obesity in adulthood (1–3). Obesity rates are high and rising around the globe, with serious consequences for people's quality of life and even life expectancy. For example, it is anticipated that the current generation of children in the United States may – as a result of the obesity epidemic – be the first with a lower average life expectancy than their parents (4).

The home food environment as a gateway for early obesity prevention

Current weight loss interventions have limited, if any, success (5). Consequently, researchers have begun to focus on *preventing* weight gain. The childhood years represent a unique window of opportunity to pre-empt the formation of detrimental health habits. But which prevention approaches are effective? One promising approach is the promotion of healthy eating habits and competences. Such competences are crucial in today's obesogenic food environment. The obesity epidemic is increasingly understood as a consequence of a food environment that promotes excessive energy intake through inexpensive, calorie-dense and nutrient-poor foods, available in large portion sizes everywhere at any time (6). Children, however, especially in early childhood, do not generally interact with this food environment autonomously. Rather, their nutritional gatekeepers – parents, grandparents and other caretakers – shape their nutritional ecosystem (7–9).

In the United States, about two-thirds of children's daily calories have been found to stem from food prepared at home (10). The size of the 'home bias' differs depending

on the age of the child: as children grow older, they increasingly consume calories outside the home environment (10). Furthermore, as a consequence of different school systems and school meal programmes, the influence of the home food environment differs between countries. In Germany, e.g. lunch is generally the most caloric meal and most – especially younger – students eat lunch at home with their families, also on school days (11). Importantly, parents as nutritional gatekeepers influence their child's eating behaviour both directly, through the food they prepare at home, and indirectly, through their behaviour, attitudes and the nutritional environments they choose for their children outside the home. Thus, parents shape their child's eating behaviour both inside and outside the home environment (12). Our focus is on the home food environment and how it can be harnessed in the service of preventing childhood obesity. Specifically, what measures can be taken to improve this food environment?

One entry point for interventions may be the family meal. Assuming that children eat three meals a day, a large proportion of them in the family context, family meals offer a rich opportunity to expose children to healthy foods. Moreover, communal meals present a learning opportunity: children can potentially learn about nutrition in theory and practice, and parents can model healthy eating. Family meals thus constitute a social setting with the potential to shape children's eating routines and behaviours from an early stage. Family meals have recently become a buzzing focus of scientific attention: according to the *Web of Science*, the average number of publications on family meals increased from some 5–8 per year between 1970 and 1995 to, on average, 45 publications per year between 2010 and 2015.

Relationship between family meals and nutritional health

Findings on the relationship between the frequency of family meals (henceforth 'meal frequency') and overweight and obesity are mixed. Some studies have found that regular family meals are associated with a lower risk of overweight and obesity (e.g. (13,14); others have found no link (e.g. (15,16). Furthermore, frequent family meals have been observed to be associated with several positive dietary outcomes, including higher average fruit and vegetable intake (e.g. (17), lower fast food and soft drink consumption (e.g. (18,19) and better overall diet quality (e.g. (20). However, effect sizes differ, and some studies failed to find significant links (e.g. (21) for fruits and vegetables, (22) for soft drinks).

Let us make important clarification at this point. As a result of the correlational nature of the studies on family meals synthesized in this meta-analysis, we will not be able to draw causal inferences. In principle, four possible types of association between meal frequency and nutritional health are conceivable: first, family meals might be a causal factor in improving children's eating behaviour and nutritional health. Findings of longitudinal and intervention studies support this possibility. For instance, one randomized control intervention study showed that promoting family meals resulted in a significant decrease in children's weight gain after 1 year (23). Second, the opposite may hold, and health-conscious families may eat together more frequently. To our knowledge, there is no study – longitudinal, intervention or otherwise – that supports this direction of causality. Third, the link between meal frequency and nutritional health might be explained by third variables, such as socioeconomic status (SES) or family functioning. Indeed, a link has been observed between a *chaotic* family environment and overweight in children (24). Key features of a chaotic family environment are stress and lack of structure and routines (25). Thus, a more positive family environment and/or higher SES might explain part of the relationship between higher meal frequency and better nutritional health. Fourth, and most likely, a reciprocal relationship might underlie the link between meal frequency and nutritional health. In other words, both causal directions might be operative, with healthy families having more regular family meals and, at the same time, more regular family meals promoting family members' nutritional health.

Definitions of nutritional health

In this meta-analysis, we consider four nutritional health outcomes: body mass index (BMI), healthy diet, unhealthy diet and overall diet quality. *BMI* relates body weight to height and is often employed to describe whether a person is underweight, normal weight, overweight or obese (26). However, BMI is only an approximate indicator of health. For example, it does not differentiate between fat and muscle mass. Moreover, obesity does not increase mortality in all circumstances (e.g. in old age; (27); in some, it may even have survival benefits (e.g. after surgery; (28). *Healthy diet* is often operationalized as the number of portions of fruit and vegetables consumed per day (29). Evidence shows that eating five or more portions per day reduces the risk of cancer and cardiovascular disease (30). *Unhealthy diet* is generally operationalized in terms of consumption of sugar-sweetened beverages, fast food or unhealthy sweet or salty snacks (29,31). A higher intake of sugar, fat and energy is known to be associated with a greater risk of developing diabetes, high blood pressure, coronary heart disease and obesity (32–34). Some studies do not differentiate between healthy and unhealthy diet but report *overall diet quality*. One gold standard measure is the Healthy Eating Index, which assesses compliance with dietary guidelines on, e.g. consumption of fruits, whole grains, fatty acids and sodium (35).

The role of sociodemographic and mealtime characteristics

Studies on family meal frequency differ in the properties of the populations targeted, including SES, children's age and country. Further, there is not yet a standard definition of what exactly constitutes a *family meal*. Both factors may impair reliable measurement and thus contribute to inconsistent findings. In the following, we summarize findings on the role of sociodemographics and methodological approaches in family meal studies and suggest how they may affect the link between meal frequency and nutritional health.

Socioeconomic status

Family meals are most frequent in high-SES homes. At the same time, higher SES predicts healthier diet and body weight (36). But to what extent does higher SES explain the positive link between family meals and nutritional health? If SES is a significant driver of the link between meal frequency and nutritional health, lower effect sizes should be observed in studies controlling for SES.

Age

As children grow up, they become more independent from their family and the influence of peers increases (37). If family meals have a lesser influence on the nutritional health of adolescents, studies with samples of children should, ceteris paribus, report larger effect sizes than studies with samples of adolescents.

Country

The large majority of family meal studies originate in the United States, followed by European countries, South America, Australia, New Zealand and Asian countries. Meta-analytic techniques afford the opportunity to investigate the effect of country differences on family meals.

Meal type

Another source of heterogeneity between family meal studies may be differences in the meal types considered. Whereas most studies have investigated either 'family dinner' or simply 'family meals', some have looked specifically at 'family breakfast' or 'family lunch'.

Family members at the table

Studies differ with regard to who must be present at the table for a meal to be considered a 'family meal'. Definitions range from 'at least one parent' to 'the whole family'. One study with a sample of 160 parent–child pairs investigated variations in terms of group size (i.e. who was present at the table) and found small differences in weight outcomes. More specifically, measures asking about 'sitting and eating together' revealed stronger effects than measures that addressed either sitting together *or* eating together (38).

The present investigation

To the best of our knowledge, only one previous meta-analysis has examined the relationship between family meal frequency and children's nutritional health (39). It found that regular family meals were associated with better nutritional health in children. However, due to the small number of studies analysed (k = 17), the authors were not able to investigate potential reasons for the heterogeneity in results across studies. The present meta-analysis aims to fill this gap. Taking advantage of the surge in studies on family meals, it investigates potential sources of heterogeneity and examines potential moderators. By examining moderating factors, we aim to bring the field one step closer to fully understanding the nature of the association between meal frequency and nutritional health.

This meta-analysis has the following objectives:

1 To identify and quantify the nutritional health correlates of family meals; these correlates are measured in terms of the child's BMI, healthy diet, unhealthy diet and overall diet quality;
2 To determine the impact of demographic characteristics (age, gender, SES, country) and mealtime characteristics (meal type, family members present at the table) on the association between meal frequency and nutritional health.

Method

Literature search and study selection

The search strategy and keywords were developed in collaboration with a professional librarian. The literature search consisted of the following three steps: first, we conducted systematic literature searches in *Web of Science* (search terms: ['family meal' OR 'mealtime*' OR 'shared meal' OR 'dinner'] AND ['BMI' OR 'body mass index' OR 'overweight' OR 'obesity' OR 'food intake' OR 'eat*' OR 'diet' OR 'nutrition'], refined by topic 'child*' OR 'adolescent*' OR 'young adult*'); *PubMed* (Medical Subject Headings (search terms: ['diet' OR 'feeding behavior'] AND 'family', filter: 'preschool child', 'child', 'adolescent') and in *PsycInfo* (search terms: ['body mass index' (Thesaurus) OR 'body weight' OR 'obesity' OR 'overweight' OR 'diets' OR 'eating behavior' (Thesaurus) OR 'food' (Thesaurus) OR 'food preferences' OR 'nutrition' (Thesaurus)] AND ['mealtimes' (Thesaurus) OR 'meal*' OR 'dinner' OR 'lunch']). The search terms used differ between databases because we used both free and controlled vocabulary (i.e. Medical Subject Heading terms in Pubmed; Thesaurus terms in PsycInfo). The literature search was conducted in January 2017. It covered both published and unpublished studies (e.g. conference

abstracts, dissertations) in English or German. Second, we performed forward searches. Using *Web of Science*, we systematically searched for studies that cited key studies identified in the literature search. Third, we conducted backward searches, i.e. we manually examined the reference lists of reviews on family meals.

These searches identified a total of 3,906 articles (see Fig. 1, for a PRISMA flow chart illustrating the study selection process). Inclusion/exclusion of the first 500 studies was determined independently by the first author and a second trained rater. Because the agreement rate (94%) was high (40), the remaining studies were screened by only one rater each. The eligibility criteria for inclusion in the meta-analysis were as follows: (i) a measure of family meal frequency; (ii) at least one indicator of nutritional health and (iii) a statistical association between family meal frequency and nutritional health. Measures of nutritional health considered were (i) BMI, (ii) healthy diet, (iii) unhealthy diet and (iv) overall diet quality. Studies were excluded if (i) children were not the target population; (ii) the study examined children with feeding problems (e.g. children with autism) or with diseases requiring a special diet (e.g. children with cystic fibrosis or diabetes) and (iii) the study reported insufficient statistics to calculate an effect size. Using these criteria, we arrived at a total of 57 studies (13–22,38,41–86).

Coding of studies

In accordance with existing guidelines (87), we extracted the following information from each study:

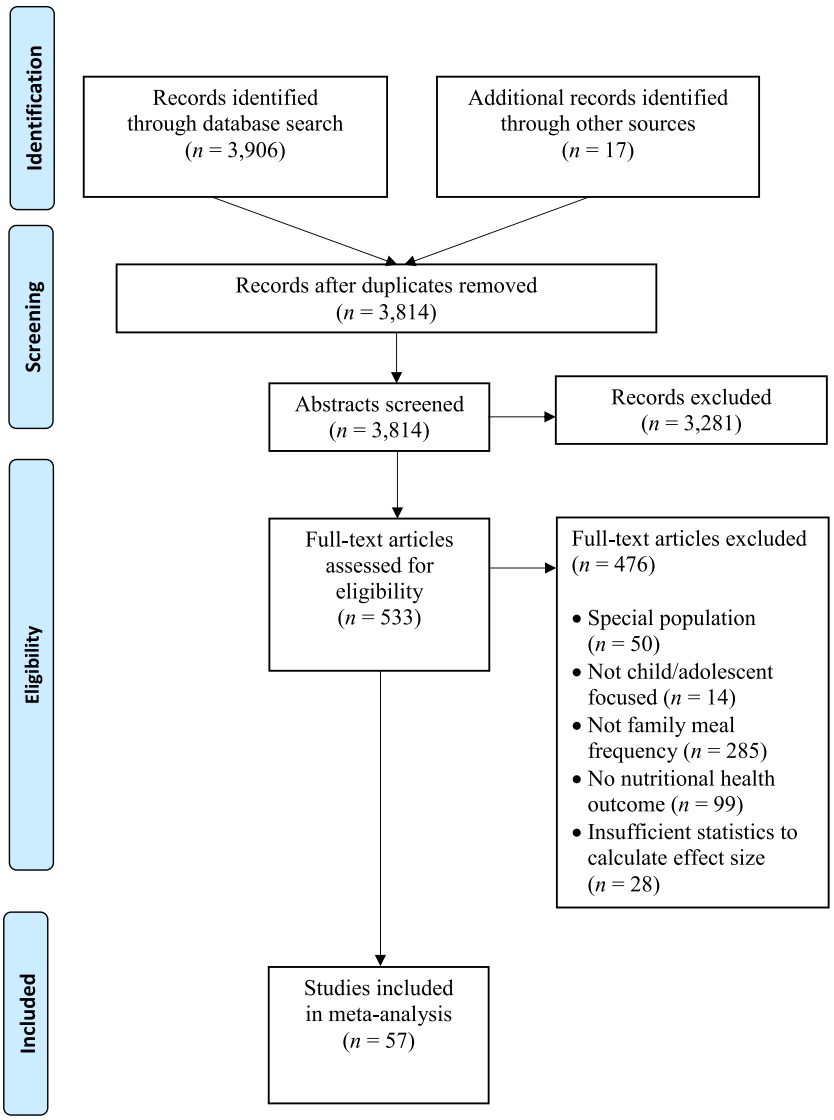

Figure 1 PRISMA flow chart showing the study selection process. [Colour figure can be viewed at wileyonlinelibrary.com]

- Source characteristics: author, year of publication
- Sample characteristics: sample size, ethnic composition, age
- Measure characteristics: outcome type (BMI, healthy diet, unhealthy diet, overall diet quality)
- Design characteristics: longitudinal, cross-sectional
- Study quality: external validity (subpopulation, sampling procedures); construct validity (measurement characteristics, including reliability and validity)
- Moderators: age group, country, SES, family members present, meal type

Table 1 summarizes selected study characteristics relevant for the meta-analysis.

Data synthesis

Calculation of effect sizes

The primary studies reported multiple levels of variables and statistics. For each study, we calculated the correlation coefficient r as an effect size that quantifies the magnitude of the association between family meal frequency and children's nutritional health. We chose r as the effect size because both the frequency of family meals and the frequency of food consumption are naturally continuous. Consequently, most studies measured meal frequency and nutritional health on a continuous scale, and many reported correlation coefficients. Additionally, r is easy to interpret and can be extracted from several statistical parameters. Where statistics from group comparisons were reported (e.g. means, t values, odds ratios or frequencies), Cohen's d was calculated and converted to r (87,88). Where only standardized regression coefficients were available, we used those (89,90). Correlation coefficients were transformed to a z score metric using Fisher's z-transformation. In all analyses, we used r-to-z transformed values. For figures including funnel and forest plots, the pooled effect sizes were back-transformed to r values.

Meta-analyses: estimating effect sizes for nutritional health outcomes

We applied random-effects models, because we expected systematic heterogeneity between studies due to differences in study samples, measurements and quality. Random-effects models do not assume one true effect size but a distribution of effect sizes. The pooled effect size represents an estimate of the mean of this distribution. Heterogeneity was quantified by the I^2 statistic, specifying the degree of systematic variation between studies (91): an I^2 value of 0 means that variation in effect sizes between studies results from random error; values above 0 indicate the proportion of systematic between-study variation.

In order to investigate the associations between family meal frequency and nutritional health, we analysed the following nutritional health outcomes in separate meta-analyses: (ii) children's BMI (reported or measured), (ii) healthy diet (consumption of healthy foods, e.g. fruit and vegetable intake), (iii) unhealthy diet (consumption of unhealthy foods, e.g. intake of sugar-sweetened beverages, fast food, sweet and salty snacks), (iv) overall diet quality (dietary index combining healthy and unhealthy diet, e.g. the Healthy Eating Index as a measure of compliance to the Dietary Guidelines for Americans). We performed separate meta-analyses for each of our four outcome types because, first, they represent qualitatively different aspects of nutritional health, and the strength of their association with meal frequency may thus differ. Second, we observed large non-random variability, not only across but also within outcome categories. To control for differences between outcome categories, we explored the effect of demographic and meal characteristics within outcome categories.

Combining subgroups and outcomes

Some studies reported statistics for multiple subgroups (e.g. separate results for boys and girls, or for younger and older children). In these cases, we computed separate effect sizes for each subgroup as well as a pooled effect size across subgroups (88). Other studies reported several nonindependent outcomes (e.g. separate results for fruit and vegetable consumption). In order to adjust for dependencies in effect sizes, we calculated a pooled effect size but took the correlation between the outcomes into account. We applied the same procedure to studies reporting different outcomes but otherwise sharing the same data and, consequently, the same sample.

Moderator analyses

Within each meta-analysis of more than 10 studies, we investigated the following sociodemographic and mealtime characteristics as potential moderators: (i) age (children <11 years/adolescents ≥ 11 years); (ii) SES (controlled/not controlled; note that the group of studies controlled for SES includes both studies that adjusted their effect sizes for indicators of SES and studies where the target population was homogenous with respect to SES); (iii) country (North America/Europe/South America/Asia/Australia or New Zealand); (iv) type of meal (breakfast/lunch/dinner/unspecified); (v) family members present at the table (all or most family members/one parent or some family members/unspecified). In order to examine differences in the strength of meal frequency effects and variability between effect sizes, we first calculated separate effect sizes and the heterogeneity index I^2 for each category of the potential moderator. In a second step, we tested for

Table 1 Selected characteristics of studies included in the meta-analysis

First author	Year	Country	Outcome	*N*	Age	SES	Meal type	Family members
Andaya	2011	US	H, U	794	children	yes	B, L, D	unsp.
Appelhans	2014	US	H, U	103	children	yes	unsp.	most/all
Ayala	2007	US	U	167	adolescents	yes	B, L, D	unsp.
Bauer	2011	US	BMI, H, U	253	adolescents	yes	unsp.	unsp.
Befort	2006	US	H	228	adolescents	no	unsp.	unsp.
BeLue	2009	US	BMI	35,184	adolescents	no	unsp.	most/all
Berge	2014	US	BMI, H, U	2,682	adolescents	yes	unsp.	most/all
Chan	2011	US	BMI	141	adolescents	no	unsp.	most/all
Christian	2013	England	H	1,516	children	yes	D	unsp.
Crombie	2009	Scotland	O	300	children	yes	unsp.	unsp.
Cutler	2011	US	H, U	4,746	adolescents	yes	unsp.	most/all
de Wit	2015	Europe[a]	H, U	2,764	adolescents	no	B, D	most/all
Fink	2014	US	H, U	1,992	adolescents	yes	unsp.	unsp.
Fulkerson	2008	US	BMI	2,516	adolescents	yes	unsp.	most/all
Fulkerson	2009	US	BMI, H, U	139	adolescents	yes	D	most/all
Gable	2007	US	BMI	8,000	children	yes	B, D	unsp.
Gillman	2000	US	H, U	16,202	adolescents	no	D	unsp.
Goldman	2012	US	H	229	children	yes	unsp.	unsp.
Granner	2011	US	H	736	adolescents	no	D	unsp.
Horning	2016	US	BMI, H, U, O	160	children	no	D	most/all
Jaballas	2011	US	BMI	339	children	no	unsp.	one/some
Koszewski	2011	US	H	108	children	yes	B, L, D	unsp.
Larson, MacLehose	2013	US	H, U	2,507	adolescents	yes	B	most/all
Larson, Wall	2013	US	BMI	2,793	adolescents	yes	unsp.	most/all
Larson	2016	US	H, O	827	adolescents	no	B	most/all
Laurson	2008	US	BMI	268	adolescents	no	D	unsp.
Lee	2014	Korea	BMI	3,435	children	no	D	unsp.
Leech	2014	Australia	U	155	adolescents	no	D	one/some
Lehto	2011	Finland	BMI	604	children	no	D	unsp.
Lillico	2014	Canada	BMI	3,341	adolescents	no	unsp.	one/some
Liu	2014	US	BMI	1,000	children	no	D	one/some
Mamun	2005	Australia	BMI, U	3,757	adolescents	no	unsp.	unsp.
Moon	2014	Korea	H	2,588	children	no	B, unsp.	one/some
Ness	2012	US	BMI	5,342	adolescents	no	unsp.	most/all
Ntalla	2016	Greece	BMI	1,929	adolescents	yes	unsp.	unsp.
Peters	2013	Australia	H	269	children	no	D	unsp.
Price	2009	US	BMI	4,688	adolescents	no	D	unsp.
Prior	2013	UK	H, U	76	adolescents	no	unsp.	unsp.
Pyper	2016	Canada	H	3,206	children	yes	unsp.	unsp.
Ranjit	2015	US	O, H, U	2,502	adolescents	no	unsp.	unsp.
Reed	2013	US	BMI	43	adolescents	yes	B, D	unsp.
Roos	2001	Finland	H	65,059	adolescents	no	D	unsp.
Roos	2014	Europe	BMI	2,586	adolescents	yes	B, D	one/some
Santiago-T.	2014	US	O	187	adolescents	yes	unsp.	unsp.
Sen	2006	US	BMI	2,524	adolescents	yes	D	unsp.
Serrano	2014	Puerto Rico	BMI, O	112	adolescents	no	unsp.	unsp.
Skafida	2013	Scotland	O	2,190	children	no	unsp.	most/all
Spurrier	2008	Australia	U	280	children	no	unsp.	one/some
Sweetman	2011	UK	H	434	children	yes	unsp.	one/some
Taveras	2005	US	BMI	3,088	adolescents	yes	D	unsp.
Utter	2008	New Zealand	H, U	3,119	adolescents	yes	D	most/all
V. Lippevelde	2012	Europe[b]	BMI	6374	adolescents	yes	B	one/some
Verzeletti	2010	Europe[c]	H	1,4407	adolescents	yes	D	one/some
Videon	2003	US	H	18,177	adolescents	yes	unsp.	one/some
Woodruff	2009	Canada	BMI, U	3,025	adolescents	no	D	one/some
Woodruff	2010	Canada	O	985	adolescents	no	D	one/some
Wyse	2011	Australia	H	396	children	no	D	unsp.

[1]Netherlands, Poland, Portugal, Poland and UK.

[2]Belgium, Greece, Hungary, Netherlands, Norway, Slovenia, Spain and Switzerland.

[3]Belgium and Italy.

B, breakfast; BMI, body mass index; D, dinner; H, healthy eating; L, lunch; most/all, most or all family members; *N*, sample size on which effect size calculations were based; O, overall diet quality; one/some, one parent or some family members; U, unhealthy eating; unsp., unspecified.

moderator effects using the *QM* test for moderators with *c* – 1 degrees of freedom, where *c* is the number of categories in the moderator variable.

Publication bias

We used funnel plots and trim and fill methods to investigate the likelihood of publication bias due to the file drawer problem (studies with null findings are less likely to be published and therefore included in meta-analyses). Funnel plots depict effect sizes and the corresponding standard errors. An asymmetric funnel plot indicates a higher probability of publication bias. Funnel plot asymmetry was tested using Egger's linear regression method. Trim and fill methods add missing studies until the funnel plot shows a symmetric distribution. Next, we computed an adjusted pooled effect size, taking into account effect sizes added by the trim and fill method (92).

Results

Study quality

Only 8 of 75 studies had a longitudinal design. Almost all studies used convenience samples; 14 studies analysed a specific subgroup of the population (e.g. low-income families). However, an important strength of the meta-analytic approach is that it calculates an overall effect size that covers diverse samples. Most studies (59%) used a validated scale to measure healthy diet, unhealthy diet, and overall diet quality or reported a reliability index for the measure used. BMI was objectively measured in about half of the studies; the other half used self-reported BMI. A large majority of the studies (74%) did not use a validated scale or report a reliability index for their family meal frequency measure. More details of the quality coding analysis are reported in the Supporting Information (Table S1).

Meta-analyses

Across all studies, having frequent family meals was significantly associated with a lower BMI (r = −0.05, 95% CI [−0.06,–0.03]), a more healthy diet (r = 0.10, 95% CI [0.09, 0.12]), a less unhealthy diet (r = −0.04, 95% CI [−0.07, −0.03]) and better overall diet quality (r = 0.13, 95% CI [0.06, 0.20]). We found large heterogeneity across studies, as indicated by high I^2 values (see Table 2 for statistical details, and Figs 2–5 for forest plots).

Moderator analyses

Demographic characteristics

We tested whether the demographic characteristics of age, country and SES moderated the association between family meal frequency and BMI, healthy diet or unhealthy diet. Moderator effects for age and country were not significant for any of the outcomes (see *QM* statistics in Table 3). SES was a significant moderator only in studies investigating BMI as the outcome. Subgroup analyses revealed larger effect sizes of family meal frequency on BMI in studies not controlling for SES than in studies controlling for SES.

Table 2 Results of meta-analyses on the effects of family meal frequency by outcome type

Outcome	*r*	[95% CI]	*k*	I^2 (%)
BMI	−0.05**	[−0.06, −0.03]	25	78
Healthy diet	0.10**	[0.09, 0.12]	27	87
Unhealthy diet	−0.04**	[−0.07, −0.03]	19	78
Overall diet quality	0.13*	[0.06, 0.20]	9	85

Results from random-effects models. CI, confidence interval; I^2, heterogeneity index; *k*, number of samples; *r*, correlation coefficient.

Mealtime characteristics

We next examined the number of family members present at the table and meal type as potential moderators. No significant moderator effects were observed (Table 3).

Publication bias

The funnel plot for overall diet quality showed a roughly symmetrical distribution. The plots for BMI, healthy diet and unhealthy diet were slightly skewed to the right. Egger's tests for funnel plot asymmetry were significant for BMI (p = 0.001) and healthy diet (p = 0.046) but not for unhealthy diet (p = 0.103). Trim and fill analyses imputed five hypothetically missing studies for BMI, four studies for healthy diet and three studies for unhealthy diet (Fig. 6a–6d). Importantly, though, the adjusted effect sizes, taking publication bias into account, remained the same or were only slightly lower but still significant (BMI: r = −0.042; 95% CI [−0.06, −0.03]; healthy diet: r = 0.10; 95% CI [0.08, 0.12]; unhealthy diet: r = −0.037; 95% CI [−0.06, −0.02]; overall diet quality: r = 0.16; 95% CI [0.09, 0.23]).

Discussion

Our meta-analyses found evidence of small and significant associations between family meal frequency and children's nutritional health. The associations with healthy diet and overall diet quality were stronger than those with BMI and unhealthy diet. The results from our sample of 57 studies, more than three times the size of the sample used by Hammons and Fiese (39), were thus consistent with their findings of small associations between frequent family meals and lower risk for overweight, more healthy diet and less unhealthy diet. Our findings make an important contribution to the research and discussion on family meals, as they show

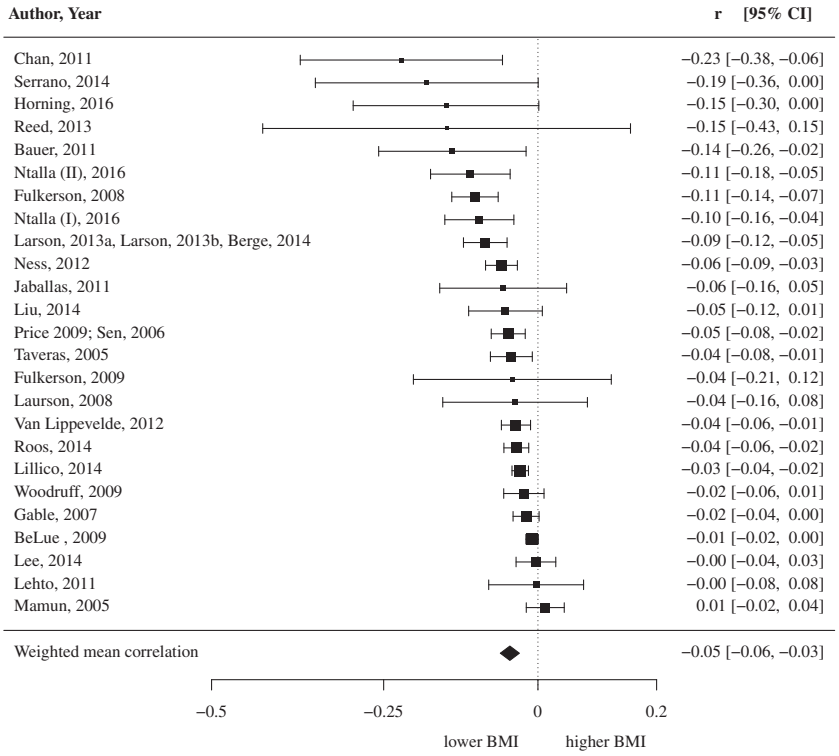

Figure 2 Forest plot showing the distribution of effect sizes for body mass index.

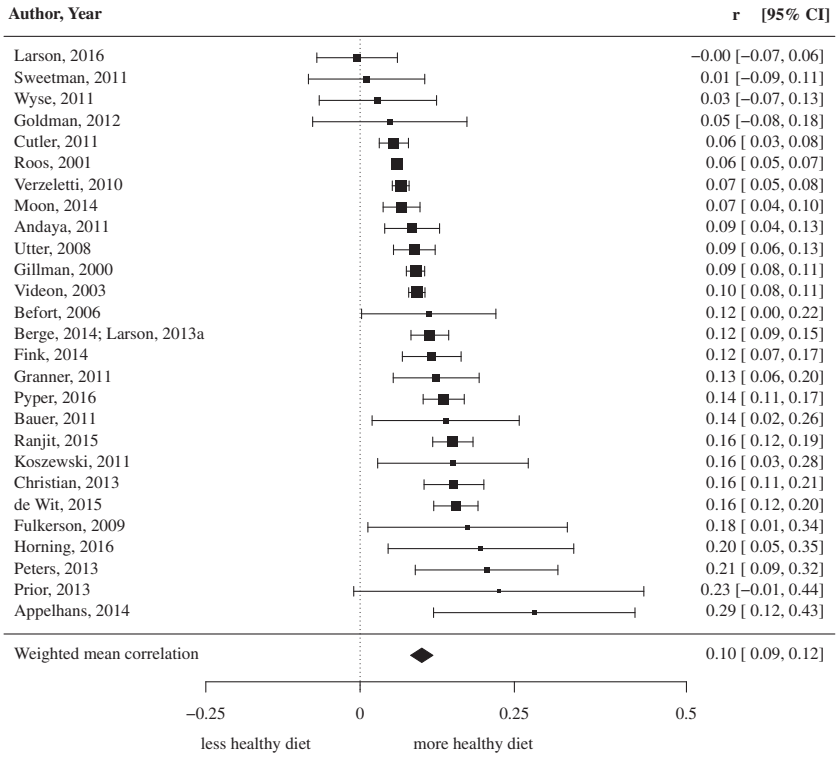

Figure 3 Forest plot showing the distribution of effect sizes for healthy diet.

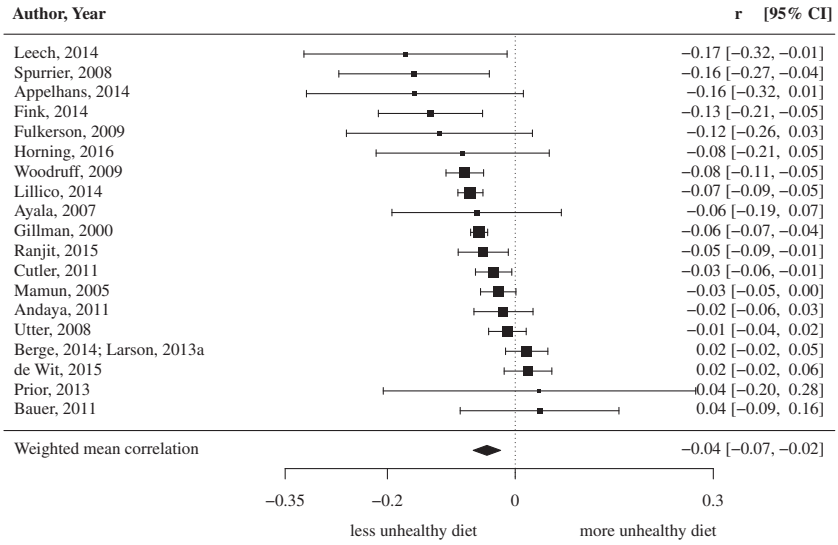

Figure 4 Forest plot showing the distribution of effect sizes for unhealthy diet.

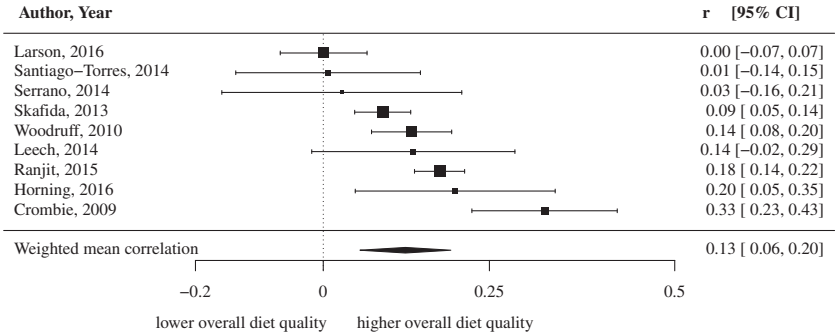

Figure 5 Forest plot showing the distribution of effect sizes for overall diet quality.

that the association between family meals and nutritional health is robust above and beyond the influence of potential moderators, such as country, age, family members present at the table and type of meal. SES was a significant moderator only in studies investigating BMI: the negative association of family meal frequency with BMI was smaller in studies that controlled for SES than in studies that did not.

In what follows, we discuss qualitative aspects of family meals, i.e. nutritional and social factors that have the potential to explain the association between family meal frequency and nutritional health.

Potential mechanisms underlying the association between meal frequency and nutritional health

The studies examining the association between meal frequency and children's nutritional health are correlational in nature. As such, we can only speculate as to the causal mechanisms underlying the link between meal frequency and nutritional health. In this section, we focus on how family meals may improve nutritional health in children. We chose to focus on this causal direction because all studies included in this meta-analysis proposed this causality. Also, first results from longitudinal and randomized control trials support the idea that a high frequency of family meals is conducive to nutritional health (e.g. (23). It thus seems reasonable to assume that at least part of the effect found in correlational studies is due to a causal pathway from meal frequency to nutritional health.

Our meta-analytic findings suggest that frequent family meals are associated with better diet quality, higher consumption of healthy foods and lower consumption of unhealthy foods. One explanation for this pattern is nutritional: family meals impact the composition of the food to which children are exposed (93). Specifically, the number of family dinners is negatively correlated with the number of ready-made dinners (54,94); in contrast, meals eaten alone or with friends are more likely to include fast food or ready-made food (95). Social factors may also contribute to the link between the social institution and activity 'family meals' and nutritional health. Shared meals with the family offer a recurrent and rich learning environment. Parental

Table 3 Results of moderator analyses with subgroups

Outcome	Moderator	Subgroups of moderator	*r*	[95% CI]	*k*	I^2 (%)	*QM*
BMI	Age	Child	−0.02*	[−0.04, −0.01]	6	0	1.63
		Adolescent	−0.05**	[−0.07, −0.04]	19	82	
	Country	Europe	−0.05**	[−0.09, −0.02]	5	67	7.94
		South America	−0.19	[−0.37, −0.01]	1		
		North America	−0.05**	[−0.07, −0.03]	17	76	
		Asia	−0.03	[−0.04, −0.03]	1		
		Australia or New Zealand	−0.01	[−0.02, −0.04]	1		
	SES	Not controlled for SES	−0.06**	[−0.09, −0.04]	12	72	4.30*
		Controlled for SES	−0.03**	[−0.05, −0.01]	13	66	
	Family members	One parent, some members	−0.03**	[−0.04, −0.02]	4	0	0.44
		All or most members	−0.06**	[−0.09, −0.03]	10	84	
		Unspecified	−0.05**	[−0.09, −0.03]	12	71	
	Meal type	Breakfast	−0.07	[−0.16, 0.00]	3	96	1.47
		Dinner	−0.04**	[−0.05, −0.02]	10	7	
		Meal (unspecified)	−0.06*	[−0.09, −0.03]	14	89	
Healthy diet	Age	Child	0.11**	[0.08, 0.15]	12	64	0.61
		Adolescent	0.10**	[0.08, 0.12]	15	91	
	Country	Europe	0.10**	[0.05, 0.15]	6	95	0.88
		North America	0.11**	[0.09, 0.13]	17	76	
		Asia	0.07**	[0.04, 0.10]	1		
		Australia or New Zealand	0.10*	[0.02, 0.19]	3	70	
	SES	Controlled for SES	0.11**	[0.09, 0.13]	14	66	0.23
		Not controlled for SES	0.10**	[0.07, 0.13]	13	93	
	Family members	One parent, some members	0.08**	[0.06, 0.10]	3	72	0.80
		All or most members	0.11**	[0.06, 0.17]	8	88	
		Unspecified	0.11**	[0.09, 0.14]	16	79	
	Meal type	Breakfast	0.10**	[0.03, 0.16]	5	82	0.70
		Lunch	0.12	[−0.02, 0.26]	2	53	
		Dinner	0.09**	[0.07, 0.12]	12	83	
		Meal (unspecified)	0.11**	[0.09, 0.14]	14	77	
Unhealthy diet	Age	Child	−0.09*	[−0.17, −0.01]	4	54	0.89
		Adolescent	−0.04**	[−0.06, −0.02]	15	81	
	Country	Europe	0.02	[−0.02, 0.02]	2	0	3.60
		North America	−0.05**	[−0.07, −0.03]	13	75	
		Australia or New Zealand	−0.07	[0.14, −0.01]	4	87	
	SES	Controlled for SES	−0.03	[−0.06, 0.00]	9	66	0.01
		Not controlled for SES	−0.03**	[−0.08, −0.03]	10	80	
	Family members	One parent, some members	−0.08**	[−0.09, −0.06]	3	2	9.3
		All or most members	−0.02	[−0.05, 0.01]	8	65	
		Unspecified	−0.05**	[−0.06, −0.03]	8	33	
	Meal type	Breakfast	−0.01	[−0.06, −0.06]	2	60	4.91
		Lunch	0.04	[0.03, 0.11]	1		
		Dinner	–0.06**	[−0.09, −0.03]	7	63	
		Meal (unspecified)	−0.04*	[−0.07, −0.01]	12	78	

*$p < 0.05$.
**$p < 0.01$.
Results from mixed effects models. CI, confidence interval; I^2, heterogeneity index; *k*, number of samples; *QM*, *QM* test of moderators with c – 1 degrees of freedom, where c is the number of categories in the moderator variable; *r* = correlation coefficient; SES, socioeconomic status.

feedings styles, such as role modelling or encouragement, may positively influence children's dietary behaviour (e.g. (96,97) inside and outside the family home.

Moderator analyses

Socioeconomic status

Children with lower SES are more likely to experience poorer health, including obesity (98). With respect to BMI, we found lower effect sizes in studies controlling for SES than in studies not controlling for SES. This observation indicates that the positive effect of family meals can be partly attributed to differences in SES. However, the pooled effect size for studies controlling for SES was still significant, suggesting that the association between family meal frequency and nutritional health is likely to exist above and beyond differences in SES.

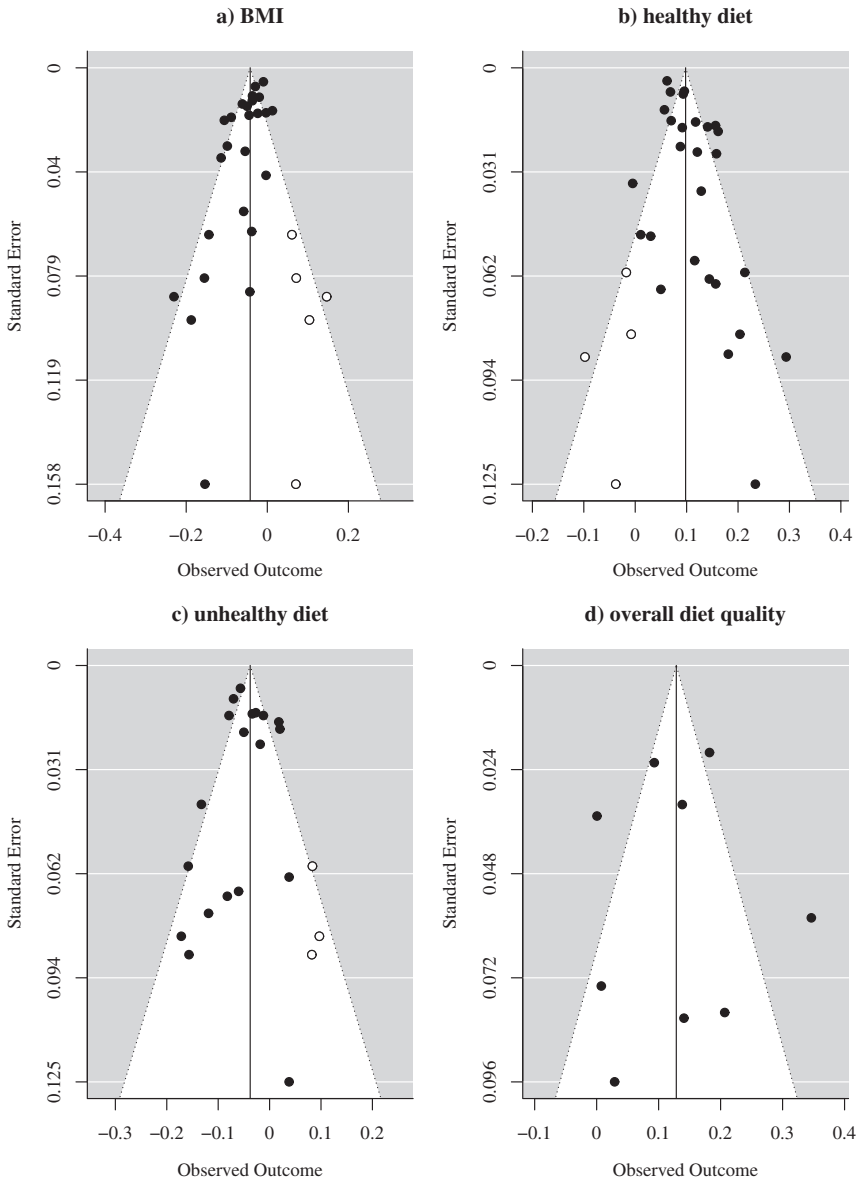

Figure 6 Funnel plots with trimmed and filled effect sizes for (a) BMI, (b) healthy diet, (c) unhealthy diet and (d) overall diet quality.

Age

Our findings suggest that the association between meal frequency and nutritional health is independent of children's age. This result is somewhat unexpected; past studies have shown that influence of the family decreases as children grow older and, as adolescents, participate in fewer family meals (99). Importantly though, our findings point to the possibility of a reciprocal causal relationship: having frequent family meals at an early age may start a causal upward spiral, in which family meals promote nutritional health, and nutritional health promotes family meals (100). Such a mechanism results in a stronger association between family meals and health outcomes that remains stable, or may even increase, with age.

Country

On a descriptive level, we found small differences in pooled effect sizes between countries; however, these differences were not significant. It is important to bear in mind that few studies have been conducted outside the United States, meaning that the number of studies in the other moderator subcategories was small. Nevertheless, our moderator analyses suggest that the association between family meal frequency and nutritional health does not differ substantially between countries.

Meal type

We found no significant differences in effect sizes across meal types, suggesting that the association between meal

frequency and nutritional health holds no matter whether families eat breakfast, lunch or dinner together. Again, these results should be interpreted with caution, because the majority of studies examined family dinners or did not specify the meal type. Only a few studies investigated family breakfast and lunch (in many countries, children's school schedules prohibit them from having lunch at home).

Family members present at the table

Effect sizes did not differ across different definitions of a 'family meal' for any of the outcome measures. These findings suggest that the association between meal frequency and nutritional health does not depend on whether a meal takes place with one parent or with the whole family.

Limitations

As emphasized before, because the studies entered in this meta-analysis were observational, we cannot draw causal conclusions. Importantly, the opposite causal direction – namely, that health-conscious families eat together more often – is also conceivable. To our knowledge, one randomized control intervention study has demonstrated a preventive effect of family meal frequency on weight gain in children (23). Despite this first promising result, we cannot rule out the possibility that third variables may play a role. For example, a more positive family environment is a predictor of better health in children and is also associated with more frequent family meals (101). Regular family meals might thus be a manifestation of a more positive family environment. Importantly, however, the frequency of family meals seems easier to modify than less tangible dimensions of the family environment; it thus offers a more practical lever for promoting nutritional health in children. Our analysis also suggests that SES does not account for the association between family meal frequency and nutritional health – or at least not fully. More research in this area is needed, however, because the simple statistical control for SES does not suffice to exclude the possibility that part of the effect of meal frequency can be explained by SES (102).

The effect sizes identified in this meta-analysis are small. One explanation could be the considerable variation in how families practice daily routines such as family meals. As reviewed above, analysing nutritional and social factors (e.g. serving healthy foods and parental feeding styles) in combination may ultimately help to understand the association between family meal frequency and children's nutritional health. The complexity of the nutritional health outcomes investigated may also account for the weak associations. BMI and dietary behaviour are complex constructs influenced by a number of factors.

We found large heterogeneity in results across all nutritional health outcomes. The main reason is likely to be that studies differ in how they define and measure both family meal frequency and nutritional health outcomes. Where possible, we used moderator analyses to investigate potential sources of heterogeneity (i.e. differences in meal type and family members present). Although the inclusion of moderators resulted in a reduction of heterogeneity in most subgroups (e.g. an I^2 index of 7% in studies investigating family dinners with BMI as the outcome), no single moderator was able to explain a large part of heterogeneity between studies. This might be due to the large variation in family meal measures implemented across studies. For example, some studies used the average number of family meals per week, others dichotomized the frequency using median splits, and some compared extreme poles, such as having family meals every day vs. once a week. The same applies to the food consumption scales and BMI measures.

Implications and future directions

Our findings show an association between higher family meal frequency and better nutritional health across a broad range of study and sample characteristics. However, the effect sizes are small and should be interpreted with caution, not least because the direction of causality is not yet settled. For all these reasons, healthcare professionals are well advised to be cautious in simply prescribing a higher frequency of family meals. Furthermore, family meals are not of one kind. A first experimental study by Fiese and colleagues (103) investigated the effects of a noisy distraction on family mealtime dynamics and found increased unhealthy eating in children and negative communication patterns in adults. More experimental research is needed to investigate such less obvious qualities of family mealtimes.

One insight from this meta-analysis is that the quality of the primary studies synthesized is relatively low. A large majority of studies did not use a validated scale to measure family meal frequency and about half of the studies used a BMI measure based on self-reported weight and height. Only about one in 10 studies was longitudinal in design. Future research – ideally longitudinal in nature – would greatly benefit from a widely accepted definition of meal frequency and from using validated scales.

Our moderator analyses did not find significant differences between countries. This is important because it suggests that findings stemming predominantly from one country can inform research and policy in similar countries as well. Likewise, we did not observe substantial differences between different types and definitions of family meals.

Finally, our results raise the possibility that other communal meals (e.g. at kindergarten or school) may also be associated with children's nutritional habits. Relatedly, the growing attention to family meals coincides with an increasing number of mothers entering the workforce, making the provision of regular family meals more of a challenge. Interventions should take into account the lifestyle of modern

families. There is already evidence suggesting that 'family-style meals' at school could have beneficial effects on children's nutritional health. For example, Hendy and Raudenbush (104) showed experimentally that teachers' role modelling of novel food consumption increased the likelihood of children consuming these foods in the future.

Conclusion

Our study demonstrates an association between family meal frequency and several nutritional health outcomes in children. Importantly, our findings of small effect sizes from correlational studies suggest that other factors beyond mere frequency are also in operation. In light of the growing interest among scientists, public health officials and the general public in how family meal routines can impact children's nutritional health, the next frontier for the research community is to design and implement randomized control trials. This approach will help to reveal the causal direction of the association between meal frequency and nutritional health as well as the mechanisms that make family meals more or less healthy and that could potentially be harnessed in future obesity-prevention interventions.

Conflict of interest statement

The authors report no conflicts of interest.

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Supporting information

Additional Supporting Information may be found online in the supporting information tab for this article. https://doi.org/10.1111/obr.12659

Table S1: Selected characteristics of studies included in the meta-analysis

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