Methodological Challenges in Studies Examining the Effects of Breakfast on Cognitive Performance and Appetite in Children and Adolescents[1–3]

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ABSTRACT

Breakfast is purported to confer a number of benefits on diet quality, health, appetite regulation, and cognitive performance. However, new evidence has challenged the long-held belief that breakfast is the most important meal of the day. This review aims to provide a comprehensive discussion of the key methodological challenges and considerations in studies assessing the effect of breakfast on cognitive performance and appetite control, along with recommendations for future research. This review focuses on the myriad challenges involved in studying children and adolescents specifically. Key methodological challenges and considerations include study design and location, sampling and sample section, choice of objective cognitive tests, choice of objective and subjective appetite measures, merits of providing a fixed breakfast compared with ad libitum, assessment and definition of habitual breakfast consumption, transparency of treatment condition, difficulty of isolating the direct effects of breakfast consumption, untangling acute and chronic effects, and influence of confounding variables. These methodological challenges have hampered a clear substantiation of the potential positive effects of breakfast on cognition and appetite control and contributed to the debate questioning the notion that breakfast is the most important meal of the day. *Adv Nutr* 2017;8(Suppl):184S–96S.

Keywords: breakfast, cognitive performance, appetite, methodological considerations, methods

Introduction

Breakfast is often considered to be the most important meal of the day (1). Observational studies have associated regular consumption of breakfast with a lower BMI, lower risk of chronic disease, and higher diet quality (2–6). Moreover, consuming breakfast relative to no breakfast confers benefits on appetite regulation and cognition (7, 8). However, new evidence has contradicted prior research, questioning whether breakfast is, indeed, the most important meal of the day. Some intervention studies in children have shown no distinct advantage of breakfast consumption on weight loss and cognition compared with breakfast skipping (9, 10). This review aims to provide a discussion of the key methodological challenges in studies assessing the effect of breakfast on cognition and appetite control in children and adolescents that have contributed to the debate, along with recommendations for future research.

Breakfast and Cognitive Performance

A good deal of research has considered the effects of breakfast on objective cognitive performance outcomes. Cognition is an umbrella term that describes complex mental functions, such as memory, attention, reaction time, and executive function (11). Previous studies on breakfast and cognitive performance considered the acute effects of a single breakfast meal (12–15). Acute effects are temporary effects that occur shortly after breakfast consumption on the same

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morning of consumption, e.g., from 10 min to 4 h postingestion (16). These studies used breakfast and no-breakfast comparisons (12, 15, 17), comparisons of breakfast type (18–20), or both (13, 21). The effect of chronic breakfast interventions on cognition was also examined (22–25). Chronic effects induced by chronic breakfast interventions are long-term effects that occur after the repeated consumption of breakfast over time, e.g., from 1 mo to 3 y (16). Furthermore, the association between habitual breakfast consumption (HBC)[8] (i.e., usual breakfast consumption) and cognitive performance has been examined in cross-sectional studies (26–29). Recently, Adolphus et al. (7) systematically reviewed the evidence in children and adolescents. Although somewhat mixed, the data from acute intervention studies comparing breakfast with no breakfast suggested that consuming breakfast has a transient beneficial effect on cognitive function measured within 4 h postingestion (7). However, conclusions about the acute effects of breakfast composition and the effects of chronic breakfast interventions were limited by the paucity of studies with consistent findings because of various methodological limitations.

Acute intervention studies

Study design: parallel groups or crossover? The use of a parallel-group or crossover design can influence the findings when assessing the effects of breakfast on cognitive performance. There are advantages and disadvantages of both approaches, with no clear advantage of the use of one design over another. Most acute intervention studies favor crossover over parallel-group designs. A parallel-group design introduces additional variation between treatment conditions. The detection of a breakfast-induced cognitive effect must compete with other sources of variation in cognitive performance between conditions, such as intelligence quotient (IQ) (30), age (30), sex (31, 32), and socioeconomic status (SES) (33). Random assignment to treatment and statistical testing for differences in participant characteristics between groups assigned to different treatment conditions attempt to control for this. However, participants assigned to different treatment conditions are likely to differ on many unmeasured characteristics and could contribute interindividual variation (34). Much of this interindividual variation is eliminated with the use of a crossover design. Hence, crossover designs are potentially more sensitive for detecting measurable changes in cognition, provided that there are no carry-over effects from one treatment arm to the next.

However, exposing participants to all breakfast conditions introduces different limitations. It increases the number of visits required, leading to greater participant burden and, consequently, the likelihood of attrition. This is particularly important in field studies that are conducted in the school environment, where the researcher needs to minimize burden and disruption of the research to schools and also attempt to align a research protocol within a defined school schedule. It also increases the potential practice effects on administered cognitive tests and necessitates the use of truly equivalent parallel forms of these tests. Furthermore, the design could increase expectancy effects from increased familiarity with the study procedures, guessing the true aim and bias because of the inability to blind the breakfast and no-breakfast conditions.

Study location: laboratory or field? Acute studies in both the laboratory (14, 35–37) and field (12, 13, 15, 21) have been conducted to examine the effect of breakfast on cognitive performance in children and adolescents. More research has been conducted in controlled laboratory environments than in more realistic field settings. Laboratory-based studies allow the researchers to exert greater control and ensure compliance with study procedures. Most importantly, they allow control over food intake during the test day. Furthermore, some laboratory-based studies include a standardized evening meal and overnight stay and, hence, a monitored overnight fasting period. However, there is always a tradeoff between experimental control and ecological validity. A difficulty with well-controlled laboratory-based studies is the potential for behavior change because of the novel environment and the lack of ecological validity, which is the extent to which the results can be generalized to natural situations. The Hawthorne effect may be present, such that participants may be motivated to perform well on the cognitive tasks simply because they are under investigation (38). The act of arriving at a research unit in a novel environment might inflate cognitive performance by increasing arousal, motivation, or effort. Therefore, this renders the findings from laboratory-based studies less generalizable to real-life situations, such as in the classroom or at work.

A more realistic indication of the cognitive response to breakfast is achieved in real-life environments. Most of the published acute field-based studies are conducted in school settings (12, 13, 15, 21). Field-based studies have the advantage of aligning with the participants' normal, familiar environment and daily routine, providing ecologically valid evidence (39). Although it may be more informative to conduct testing in the field, this will cause a substantial loss of control over the study procedures and extraneous variables that exist in participants' normal daily routines. For example, in school-based studies, there may be differences in how cognitively demanding the participants' lessons are between test sessions, which could affect performance during the cognitive test sessions. Most importantly, compliance with the fasting requirements may be compromised, which will render the findings of these studies inaccurate.

Sampling and sample selection. The published acute studies on breakfast and cognitive performance have 2 main limitations with regard to the study samples: small sample size and recruitment bias. Both of these limitations have hampered a clear substantiation of the effects of breakfast on cognitive performance. Many published acute studies lack large representative samples and do not report power calculations (13, 14, 37, 40, 41). Therefore, it is unlikely that the studies are sufficiently powered to detect measurable changes

[8] Abbreviations used: HBC, habitual breakfast consumption; IQ, intelligence quotient; SBP, school breakfast program; SES, socioeconomic status; VAS, visual analog scale.

because of the small sample sizes. A true effect of breakfast on cognition may fail to be detected because of inadequate power rather than a true lack of effect. Furthermore, in the few studies that have included a power calculation, the calculations are problematic because they are based on effect size conventions rather than effect sizes demonstrated in previous research (42). Consequently, the sample size may still be insufficient to detect a statistically significant effect.

Many studies on breakfast and cognitive performance also suffer unintended recruitment bias. This causes 2 problems. First, the findings cannot be generalized across different demographic groups. Second, the recruitment bias may reduce the sensitivity of the intervention on cognitive function. Most studies are conducted in children, precluding firm conclusions about the effects in adolescents (10, 13, 20, 35, 36, 40, 43). Cognitive responses may differ within this older, pubertal age group. The rate of glucose utilization in the brain gradually declines from age 10 and usually reaches adult levels by the age of 16–18 y (44). Furthermore, adolescence involves a period of rapid development, growth, and lifestyle change, which is also coupled with changing eating habits and increased control over eating habits (45). It therefore is likely that children and adolescents differ in their response to breakfast relative to fasting.

Studies also have tended to comprise samples of children of a mid- to high SES (13, 15, 41, 46, 47). However, there are some acute studies that have examined children of a low SES (12, 43, 48). Beneficial effects of breakfast consumption are inconsistently reported in well-nourished children of a mid- to high SES (13, 15, 41, 46, 47). This may because they have better cognitive ability or greater cognitive reserve (33). Indeed, there have been some indications that baseline IQ modifies the relation between breakfast and cognitive performance, such that the effects of breakfast are greater in, or specific to, school children with lower IQs (36, 49). Therefore, samples of children who are mostly of a mid- to high SES may be fairly well protected against any negative effects of breakfast omission, resulting in no detection of any effect of breakfast skipping.

Cognitive testing. Another key methodological limitation of acute breakfast studies lies in the choice of cognitive tests used. Cognitive function typically is assessed by researcher-administered objective cognitive performance tasks that measure a component of cognitive function. A variety of cognitive domains (e.g., attention, memory, executive function, or psychomotor function) have been examined with the use of an extensive assortment of both computerized (13, 14, 17, 20, 21, 40) and pen-and-paper tasks (47, 50). A key issue with the cognitive tests used in the acute intervention studies is that the majority of studies do not state that the cognitive test choice was driven by previous evidence showing the task to be sensitive to nutritional manipulations (13, 46). Therefore, some cognitive tests (e.g., Peabody Picture Vocabulary) may not be sensitive enough to detect differences in performance induced by breakfast manipulations, which are likely to be small. Many cognitive tests have the capacity to discriminate between groups or populations [e.g., for diagnostic purposes, such as the Mini Mental State Examination (51)], but this does not mean that these tests are suitable for repeated administration to detect small differences that occur between treatments groups over time (52). The failure to detect an effect in some studies but not others may be a result of true lack of effect. However, it may be because the tasks are insensitive to small changes in performance (11). The latter will mask real effects of breakfast consumption on cognitive performance. Cognitive performance tasks suitable for administration in acute breakfast manipulations include serial sevens, free word recall, and cued word recall, based on their relatively consistent sensitivity to acute macronutrient manipulations (53).

Many studies administer a large number of cognitive tasks as part of a battery (12, 13, 17–19, 21). This scattergun approach allows for a comprehensive assessment of multiple cognitive domains. However, this approach may give spurious results. If many independent tests are performed on the same sample, the probability of obtaining significant results will increase merely because of the number of comparisons and the definition of a significant result. Alternatively, some studies combine the individual cognitive test outcomes into composite scores (14, 20). The advantage of using composite scores is that this provides a focused number of outcomes and limits the number of statistical tests needed compared with analyzing each outcome separately. However, this approach makes it difficult to partial out the effects of breakfast on specific tasks and outcomes. The systematic review by Adolphus et al. (7) found that tasks requiring attention, executive function, and memory were facilitated more reliably when breakfast was consumed than when fasting. These findings may be useful to formulate more focused testing batteries.

The temporal distribution of the cognitive tasks across the morning is also an important methodological consideration. However, in previous studies, the temporal positioning of the tasks is not typically driven by an evidence-based rationale. Within the published acute studies, administration of cognitive tests across the morning is highly variable and ranges from immediately postbreakfast (e.g., 10 min postingestion) (20, 46) to the late morning (e.g., 200 min postingestion) (14, 18). Some studies track postbreakfast performance across the morning at various time points usually shortly after breakfast (e.g., 60 min postingestion), in the midmorning (e.g., 120–180 min postingestion), and in the late-morning (e.g., 210 min postingestion) (14, 18–20). Conversely, many acute studies include only one post-intervention testing period (37, 46, 54). Some studies include no baseline (prebreakfast) measures (17, 21, 46). Some studies do not include appropriate post hoc comparisons to confirm when statistical differences in performance occurred across conditions (14). However, effects of breakfast consumption relative to fasting appear most commonly in the mid-late morning (~180 min post breakfast) (36, 40, 49, 55–57). This may be when performance decrements under fasting conditions become apparent, allowing for greater discrimination between conditions. Therefore, the positioning of multiple cognitive test sessions, especially during the late-morning time frame, may be important in elucidating the effects of breakfast on cognition.

There are other key methodological considerations in relation to the cognitive test battery in studies on breakfast and cognitive performance. These include validity, reliability, and utility in the study sample. The selection of appropriate cognitive tests for dietary interventions has been reviewed exhaustively (11, 52, 58). Therefore, these methodological considerations will not be discussed in the current review.

Breakfast manipulation: fixed or ad libitum? The mode of administration of the breakfast meal is a fundamental methodological consideration. There are 2 methods of providing breakfast in acute intervention studies. The first is the administration of a standardized breakfast meal that provides the same amount and type of food to all participants, instructing participants to consume the entirety of the breakfast meal (fixed breakfast) (12). The second is the provision of a choice of breakfast foods, instructing participants to self-serve their chosen breakfast foods and to eat until they are comfortably full (ad libitum breakfast) (17). Acute studies comparing different breakfast types always require fixed breakfast manipulations to ensure that the treatment conditions only differ in terms of the nutrient component under test (20, 21). In the published acute intervention studies comparing breakfast with no breakfast on cognition, the breakfast manipulations are typically fixed rather than ad libitum (12, 35, 36, 40, 49, 57). Fixed breakfast interventions reduce intake variability but often assume that a prescribed portion size and type of breakfast is suitable for all participants. Fixed breakfast interventions are questionable in studies that include samples of children across a wide age range (e.g., 5–16 y of age), and it is likely that the selected portion size is not suitable for all participants. This approach assumes that one size fits all in terms of portion size and food type. The chosen breakfast foods also may not be palatable to some study participants, which will reduce compliance and also negatively affect mood (59) and, in turn, cognitive function (60–62). Furthermore, a fixed breakfast manipulation may not be representative of participants' normal breakfast size and type and therefore has low ecological validity (7).

Ad libitum breakfast manipulations allow participants to choose a breakfast that is palatable and suitable for them in terms of portion size (17). This approach, therefore, better reflects the participants' usual eating habits. This is also particularly important, because previous studies have shown that consuming meals dissimilar in composition to those eaten habitually results in poorer mood and cognitive performance (63, 64). Consequently, benefits to cognitive performance from consuming breakfast may be most apparent with test meals that resemble habitual meals. Therefore, the use of a fixed breakfast manipulation in some studies may account for the observed null findings, because performance may have declined under both breakfast and no-breakfast conditions.

Transparency of treatment condition. A major limitation of acute studies comparing breakfast with no breakfast is the inherent inability to blind participants to the study conditions. This is less problematic for studies comparing different breakfast types, because it is easier to conceal treatment conditions (21). In acute studies of breakfast compared with no breakfast, the control (no breakfast) usually is under fasting conditions, but some studies attempt to include placebo controls, such as very low energy conditions (e.g., with sugar-free jelly) (41, 46, 50). These are not true placebos, but may control for the extra attention given to participants during breakfast. It is likely that participants hold preconceptions about the effects of breakfast (65–67). There is a general consensus, widely communicated, that breakfast has a role in aiding concentration (65, 67).These preconceptions could lead to an expectation of poorer cognitive performance in the absence of breakfast. In turn, this could cause participants under the no-breakfast condition to engage in compensatory efforts on cognitive tasks to attempt to counteract the expected poorer performance caused by skipping breakfast. Consequently, the unblinded treatment conditions may account for some of the inconsistencies in findings and null results observed in studies comparing breakfast with no breakfast on cognition (10, 68).

Chronic intervention studies

Study design. The chronic intervention studies examining breakfast effects on cognitive performance published to date are all evaluations of breakfast provision at school as school breakfast programs (SBPs). Breakfast typically is provided in a breakfast club environment, such as in a supervised classroom or canteen at school providing a breakfast meal and activities to support learning (54). Chronic intervention studies all include comparisons of SBP with no SBP. Evaluations of SBPs are particularly difficult to conduct in a controlled and scientifically robust manner, because they can be logistically challenging in applied research settings. It is likely that these studies require considerable cooperation from schools, parents, and even local educational authorities. Generally, SBP studies are opportunistic evaluations of government-funded school breakfast provision already in existence (25, 69). Therefore, researchers do not have sufficient control over the design because the evaluation is planned after implementation (25, 69). As a consequence, many are not randomized, use quasi-experimental designs, and have no baseline measurements (22, 25, 69, 70). Furthermore, compliance with the intervention in terms of whether or not the SBP breakfast was consumed is rarely stated (69). Rather, studies usually describe compliance in terms of attendance rates; therefore, it is not clear whether the intervention was successful in changing breakfast eating habits (70). A previous systematic review of the efficacy of interventions aimed at increasing breakfast eating frequency suggested that chronic breakfast interventions have particularly poor efficacy (71). Clearly, changes in cognitive performance reported in chronic intervention studies that do not affect breakfast eating behavior or have low participation rates are an artifact of other factors.

Randomized controlled trials are the best approach to attempt to determine with certainty the effects of school breakfast provision on cognitive performance, but, to our knowledge, there are few published to date. The published randomized controlled trials provided weak support for a beneficial effect of breakfast provision at school. However, these studies are not without limitations, which may account for the lack of effects on cognitive function. Previous trials suffered substantial contamination between treatment arms, such that more pupils in the control arm than in the intervention arm had a breakfast club operating at their school (23).

Isolating direct effects of breakfast consumption. Chronic SBP intervention studies also present difficulties in attributing the direct effects of the breakfast meal or the regime of providing a free school breakfast in a breakfast club environment to cognitive outcomes (54). In the published chronic intervention studies, food consumed before school is not recorded under both conditions, and fasting requirements are not prescribed. Hence, participants under the non-SBP conditions often consume breakfast at home, so these studies might be comparing the effects of consuming breakfast at home with consuming breakfast at school, rather than consuming no breakfast. Given that some studies describe compliance in terms of attendance rates, it is possible that participants attended the SBP but did not eat breakfast (25, 70). Consequently, these studies are not a true test of breakfast per se but are more strictly a test of the SBP regimen. In addition, it is difficult to isolate any advantageous effects from the impact of concomitant activity taking place in the schools at the same time. SBPs often are associated with increased attendance (72), punctuality (73), and readiness to learn (74), all of which are likely to affect cognitive performance concurrently.

Biased comparisons. In chronic SBP intervention studies, researchers often compare children who regularly participate in an SBP with nonparticipants. Consequently, condition is self-selected. This is likely to impose bias, because children who chose to participate in a SBP may differ systematically in ways that also affect cognitive performance from those who chose not to participate. Other studies use matched schools or classes as controls, but this approach is often unsatisfactory, because children often are only matched on the basis of age or school year group. Thus, these comparisons threaten the validity of the findings, unless comprehensive control over confounders is applied in the analysis. For example, control over SES is imperative, because more children who are of a mid- to high SES may choose to attend an SBP.

Untangling acute and chronic effects. The effects of breakfast consumption on cognitive function can be acute or chronic. For example, consumption of breakfast may facilitate cognitive performance shortly after consumption on the same morning by the provision of glucose as an energy substrate for the brain (75). Alternatively, consuming breakfast chronically may cause long-term changes in cognition by the correction of nutritional deficiencies, such as those of iron and iodine (76, 77). Chronic SBP intervention studies commonly assess performance at one follow-up period after an intervention duration ranging from 1 mo (78) to 3 y (25). The temporal positioning of the cognitive tasks on a particular day at follow-up often is not reported (23) or occurred shortly after the consumption of breakfast at school (e.g., at 1000) (24, 25). The consequence of conducting cognitive testing shortly after breakfast consumption within a chronic intervention context is that any observed breakfast-induced changes to cognitive performance will be a result of both acute and chronic consumption. As a result, it is impossible to untangle the acute effects from the chronic effects of consuming breakfast on cognitive performance.

Cross-sectional studies

Confounding variables. The validity of the findings from cross-sectional studies examining the association between HBC and cognitive performance may be threatened by both residual and unmeasured confounding (26, 27). Residual confounding is due to measurement error in the confounders included in an analysis, whereas unmeasured confounding is due to omission of a confounder from an analysis (79). Confounding can be caused by variables that are associated with both cognitive performance and breakfast consumption and are not on the causal pathway between these variables (79). Adequacy of control for confounders varies within cross-sectional studies, and some studies fail to adjust for important confounders, such as SES, sex, age, and other healthy lifestyle behaviors such as physical activity. Therefore, it is probable that there is some unmeasured confounding in the results. Moreover, it is probable that there is some residual confounding in the results from measurement error. For example, some studies relied on children's reports of parental educational level as a proxy measure of SES, which may have introduced measurement error.

Assessment and definition of HBC. The methods for assessing and defining HBC can have a profound impact on the findings in cross-sectional studies. Measurements of HBC are normally unvalidated brief dietary assessments, such as questionnaires with one item to assess breakfast intake frequency (e.g., How often do you consume breakfast per week?) (28), which may yield an inadequate assessment of habitual intake. Often, participants are not given a clear definition of breakfast, meaning that breakfast was subjectively interpreted by the individual (26, 28). What is considered to be breakfast is likely to vary between participants. The use of a questionnaire with a single item to measure HBC frequency does not allow for the assessment of the type and amount of food consumed, and the time of day it is consumed. Therefore, the data will not allow the researchers to consider breakfast composition in the analysis, nor will it allow for the use of a standardized definition of breakfast post hoc (e.g., threshold amount of food or energy or time of day) to reduce inconsistencies between participants. With regard to defining breakfast, researchers may benefit from using the definition proposed in recent commentary discussing in detail the issues surrounding how to define breakfast (80).

The classification of HBC is also an important methodological consideration. Previous studies have used various methods to define HBC (26–29, 81). Typically, participants are classified into HBC groups on a frequency basis, in which a specific number of days of breakfast intake per week are used to define, e.g., rare, occasional, or frequent HBC (27, 28). However, there is large variation in the frequency of breakfast intake to indicate the various consumption categories. For example, some studies use a 3-category classification system to define HBC (e.g., regular: ≥4 d/wk, irregular: 2–3 d/wk, and never: 0–1 d/wk) (27). Dichotomous classification systems are also used to define HBC as regular (≥5 d/wk) or irregular (<5 d/wk) (82). These differences may account for the conflicting results.

Breakfast and Appetite

Breakfast consumption is often recommended as a strategy to help individuals maintain or achieve a healthy BMI (83, 84). It has been suggested that breakfast confers positive effects on body weight via appetite control (85). However, it is not clear whether the association of breakfast with healthier body weight is mediated by appetite control or other lifestyle factors also associated with breakfast consumption, such as increased physical activity (86–88). Previous studies examining the effect of breakfast on appetite control mostly considered the acute effect of a single breakfast meal on subjective ratings of satiety or subsequent energy intake (89, 90). Recently, Leidy et al. (8) systematically reviewed this evidence in both children and adults. Despite some inconsistencies, the findings suggest that breakfast consumption confers benefits to appetite regulation compared with breakfast skipping. Furthermore, high-protein and high-fiber breakfasts lead to greater satiety and/or decreased energy intake. In addition, there is some evidence that low–glycemic index and low–glycemic load breakfasts enhance feelings of satiety (8).

Neural and hormonal signals from food ingestion originating in the gastrointestinal tract and peripheral organs and interacting with receptors in the central nervous system contribute to the cessation of eating. Satiety and regulatory hormones released from the gastrointestinal tract are relatively short-term signals that require interaction with long-term regulators (i.e., insulin and leptin) to maintain energy homeostasis (91). Food intake control mechanisms are sensitive to both the energy content and the macronutrient composition of food. Metabolic energy ultimately is derived from the 3 macronutrients (i.e., carbohydrate, fat, and protein), but they have different abilities to suppress food intake (92).

Terms such as hunger, appetite, satiety, and satiation are used widely within the field of food intake regulation. Hunger refers to the biological drive for individuals to search for food. It determines what, how much, and when to eat (93). Satiation, also termed intrameal satiety (94), refers to the processes that leads to a reduction or termination of eating within a meal and determines meal size. Satiation typically is assessed by the amount of energy consumed from a test meal. Satiety, often referred to as postingestive or intermeal satiety (94), is defined as the state of inhibition over further food intake once a period of eating has ended, and is due to the consequences of food ingestion (i.e., physiologic signals from food ingestion). Satiety is typically assessed by the duration of appetite suppression, and is influenced by the decrease in the rewarding proprieties of food, referred to as sensory-specific satiety (95). The satiety cascade collectively describes the processes that intervene between the biological drive to eat and the satiating efficiency of food (96). It embodies the important distinction between satiation and satiety and provides a useful framework for understanding the processes involved in food intake regulation.

A hierarchical satiating effect of macronutrients on short-term food intake has been proposed in adults, with protein suppressing food intake more than carbohydrate and fat (calorie for calorie) (92, 97). However, little is known about the role of macronutrient composition in the regulation of food intake in children. The purpose of this section is 2-fold: *1*) to highlight methodological issues that must be considered in studies of appetite and food intake in children and adolescents, and *2*) to summarize what little is known about short-term food intake in an effort to inform both the design of future work in this area, and the development of food guidelines to promote optimal health in this age group.

Measurement of subjective appetite. The most widely used approach to assess subjective appetite is to ask participants a series of questions relating to motivation to eat with the use of a visual analog scale (VAS) (98). The original method, developed by Hill and Blundell (1982) (98), is composed of 6 questions anchored at each end with extreme statements [e.g., "How strong is your desire to eat?" (very weak to very strong)]. The scales are completed before and after intake of the food or meal under test (commonly referred to as a preload) and at regular intervals throughout the study measurement period. VASs in preload studies are reliable, evidenced by relatively low coefficients of reproducibility and unaffected by prior diet standardization, and they can discriminate between test foods when the sample size approaches 20 participants (99).

The predictive validity of the VAS on food intake remains a matter of debate in the literature (100, 101). In one of the original investigations assessing the validity of subjective hunger in free-living men and women for 7 consecutive days, a positive group correlation was found (r = 0.5, P < 0.02) between subjective hunger and reported energy intake from food records on weekdays, but not weekend days (100). However, there was no significant association between hunger ratings and energy content of eating occurrences within individual subjects, suggesting that the VAS is not a valid predictor of subsequent energy intake. In addition, it has been reported that subjects ate when hunger ratings were low or had not changed. This may be taken to suggest that subjective measures of appetite are not a good proxy indicator of forthcoming food intake or that, in a natural setting, people often eat when they are not hungry because of interactions with environmental food cues (102, 103).

It has been reported that children lack the conceptual ability to operationalize and separate their feelings of appetite, as is required when motivation-to-eat VASs originally designed for adults are used (104). Until recently, there were no published studies reporting the reproducibility and predictive validity of the VAS in older children. In what was, to our knowledge, the only reproducibility study in boys aged 9–14 y, the change in appetite after a glucose solution on 2 separate days was similar, although there was day-to-day variation in baseline appetite (105). A criticism of the abovementioned study is that appetite scores increased rather than decreasing after the consumption of energy in solution, supporting earlier work that children are unable to complete VASs in a quantitative manner that is reflective of their actual feeling of appetite. However, the greater energy content in the test meals resulted in decreases in appetite sensations, and the VAS strongly predicted food intake at the next meal. These associations were more variable in obese boys. The sensory experience and eating frequency of liquids may account for the failure of the motivation-to-eat VAS scores to decrease in response to liquid calories. Postingestive satiety scores were weaker after fruit consumption in liquid form than after consumption in solid form in adults (106, 107). These findings could be due to the perception that liquids produce weaker satiety than solid foods (108), slower gastric emptying after consumption of solid foods (109), or the higher eating rate of liquids and semisolids, although eating more slowly may not increase subjective satiety (110). There is some evidence that solids may elicit a greater postingestive satiety response in young children (111). However, comparative studies of solid and liquid calories on subjective appetite and food intake in children are needed to inform dietary guidance for promoting healthier body weights.

Pictorial- and silhouette-based satiety scales may be a solution to the shortcomings and weaker predictive validity of the VAS on food intake in pre- and peripubertal children. For example, Faith et al. (112) developed sex-based silhouettes with varying degrees of stomach filling in response to hypothetical eating conditions in young children aged 4–6 y. Although not widely tested, there is support for the use of the new scales at least in younger children. Questions remain regarding the sensitivity of existing and newer scales to assess motivation-to-eat levels in children.

Objective measures of appetite: short-term food intake. With the exception of observational studies, the most basic and common experimental design to measure appetite objectively in a controlled setting is the preload–test meal paradigm. Studies that use this design have been conducted to measure the acute (i.e., short-term) effects of a wide range of dietary manipulations (given as a preload) on both subjective appetite (see previous section) and objective appetite (energy intake from a subsequent ad libitum test meal). One common method of assessing satiation and satiety involves having participants consume preloads of either fixed or ad libitum energy as a meal (or snack) compared with a calorie-free control or meal (or snack) skipping, followed a short time later by an ad libitum test meal (113). Comparing intake at the ad libitum test meal across preload conditions assesses the effect of the preload (or snack) on satiety. Caloric compensation can be used to express the extent to which an individual reduces his energy intake at the test meal after a caloric preload relative to the control: caloric compensation (percentage) = [food intake after control (kilocalories) – food intake after preload (kilocalories)]/kilocalories in preload. For example, a caloric compensation score of 100% indicates that an individual reduces his food intake at a subsequent test meal by an amount equal to the energy in the preload. It has been reported that children innately alter their energy intake to account for both energy content and macronutrient composition (114–117). The ability of an individual to suppress energy intake in response to macronutrient composition may guide meal planning to aid in the regulation of daily food intake and limit excursions in overeating.

The preload–test meal design has been used to describe the effects of macronutrient preloads on short-term food intake suppression in children, but, to our knowledge, it is limited to a few reports. Studies using this design vary greatly in terms of preload energy content and dose (i.e., fixed, ad libitum, or corrected for body weight and composition), physical state and form (i.e., solid, semisolid, or liquid), volume, weight, type of test meal, time to the next meal (i.e., <30 min to >5 h), and time of day (i.e., morning, midmorning, or early afternoon). Pure macronutrient preloads consistently decrease subsequent food intake in children when the measurement interval is short (30–60 min) (116, 118, 119). Longer delay intervals are associated with more variable outcomes (120), possibly because of diminishing returns from any potential satiety-enhancing effects as the time to the next meal increases.

There are several criticisms of these short-term food intake studies. First, few studies provide a physiologic rationale for the time delay between preload consumption and the next meal, making comparisons across studies difficult and potentially leading to erroneous conclusions on intake suppression. For example, children who consumed a glucose and whey protein preload on 2 separate days had similar food intake suppression at 30 min, but greater food intake suppression from the whey protein preload at 60 min (116), highlighting the importance of time delay selection in study design. What complicates interpretation of preload studies is that often both the treatment dose and time to the next meal are modified with little to no justification.

Second, participants are often unblinded to the delay between first and second meal availability, which may affect

TABLE 1 Summary of the research recommendations for studies examining the effect of breakfast on cognition[1]

Limitation	Recommendations	Implications
Acute intervention studies		
Sampling and sample selection		
Overrepresentation of children	Research is required in adolescent samples.	Improves the generalizability of the findings to adolescents.
Small unrepresentative samples; lack of power calculations	Research is required from sufficiently powered studies that use larger samples. Power calculations based on effect sizes demonstrated in previous research vs. effect size conventions are required.	Improves the generalizability of the findings. Improves confidence that null findings on CF are due to true lack of effect vs. lack of power.
Overrepresentation of children of a mid- to high SES	Research is required in more at-risk populations, such as populations of a low SES and those with poorer cognitive ability.	Improves the generalizability of the findings. Increases the sensitivity of the intervention on CF.
Study location		
Lack of field-based studies	More research is required from field-based studies, e.g., in schools.	Improves the ecological validity of the findings.
Breakfast manipulation		
Lack of realistic breakfast manipulations	Research is required that uses ad libitum breakfast manipulations.	Improves the ecological validity of the findings. Benefits to CF may be more demonstrable with test meals that resemble habitual meals.
CF tests		
Lack of studies that use sensitive CF tests	Research is required that uses tests with proven sensitivity to similar acute nutritional manipulations (e.g., serial sevens, free word recall, and cued word recall). Research required with more focused testing batteries that examine domains facilitated more reliably by breakfast consumption (attention, executive function, memory).	Improves confidence that null findings are due to true lack of effect vs. test insensitivity.
Chronic intervention studies (SBPs)		
Study design		
Poor-quality study design	Research is required from RCTs. Policy-makers responsible for SBPs should collaborate with researchers in the early design stages, before roll-out, to allow for a robust evaluation.	Improves the internal validity of the findings.
Poor or unreported effectiveness in increasing breakfast consumption	Research is required that measures breakfast consumption and attendance at SBPs. Interventions should adopt an evidence-based theoretical framework to produce change in breakfast eating.	Improves confidence that the null findings are due to true lack of effect vs. lack of effectiveness in increasing breakfast consumption.
Isolation of acute vs. chronic effects		
Temporal positioning of CF tasks unreported or administered postbreakfast	Effects should be measurable under fasting conditions after repeated consumption of the breakfast over time.	Allows for the isolation of acute effects from chronic effects.
Cross-sectional studies		
Assessment and definition of HBC		
Unvalidated brief dietary assessments	Research is required with the use of validated dietary measures that assess both frequency and composition of breakfast intake (e.g., food diary).	Improves the internal validity of the findings.
		Allows for data on both the frequency and composition of breakfast to be considered in the analysis.
Breakfast-eating occasion not defined	Research is required that adopts a standardized definition of breakfast: the breakfast-eating occasion should be defined for participants, or researchers should use a definition of breakfast post hoc (e.g., threshold amount of food and/or time of day).	Reduces inconsistencies between participants.
Confounding		
Lack of control for confounders	Research-driven selection and accurate measurement of a range of confounders to include in the analysis are required.	Reduces residual and unmeasured confounding. Improves the internal validity of the findings.

[1] CF, cognitive function; HBC, habitual breakfast consumption; RCT, randomized controlled trial; SBP, school breakfast program; SES, socioeconomic status.

TABLE 2 Summary of the research recommendations for studies examining the effect of breakfast on appetite

Limitation	Recommendations	Implications
Subjective motivation-to-eat visual analog scale was developed for adults, not children	Develop and adopt a standardized, reliable, and valid scale for assessing subjective feelings of appetite in children (i.e., pictorial- or silhouette-based satiety scales).	Provides an opportunity to apply satiety health claims on optimized child-directed products that may help promote healthier body weights and attenuate the risk of developing chronic disease.
Inconsistences in study designs as to when food intake is assessed (i.e., varying times between breakfast consumption and subsequent meal)	Consider using delay intervals that are reflective of typical snack consumption patterns, and consider the appropriateness of the test food and/or meal in relation to the time of day.	More securely assesses the effects of a preload or snack on food intake suppression and satiety in a real world–relevant manner.
Inconsistences in study designs as to how food intake is assessed (i.e., single vs. buffet meal can influence feelings of hunger)	Use study designs that are ecologically relevant (i.e., fixed meal sizes and lower food variety are more reflective of real-world eating environments).	
Lack of accounting for differences in physiology (e.g., excess adipose tissue may influence satiety signals; effect of sex hormones on food intake regulation is unknown)	Use study designs to explore the effects of physiology and sex on food intake control.	Provides researchers and clinicians with a targeted understanding of the physiological factors regulating food intake. Furthermore, it may provide relevant information to assist participants and patients in weight-loss strategies.

satiety signals inherent in the preload or test meal as a result of anticipation of a forthcoming palatable test meal. Adult participants, for example, consumed more calories at an ad libitum test meal when they were informed that access to food would be restricted for 90 min compared with 15 min later (121). Furthermore, time-blinded participants were more likely to eat or initiate a meal request more closely related to declines in blood glucose (122), suggesting that physiologic regulation of eating is more likely preserved in the presence of time-blinding, although it may lack relevance to real-life eating behaviors.

Third, the nature of the test meal can vary greatly in terms of palatability and number of foods (variety) (123, 124). Single (or homogeneous) foods and buffet-style meals are the 2 most commonly reported test meal approaches in studies of short-term food intake. In the single-food paradigm, there is the possibility that satiety signals arising from the previously consumed food may be diminished because of sensory-specific satiety induced by the test meal. The advantage of the buffet-style meal is that many foods are offered, typically in excess of energy requirements, thus allowing for measurement of both satiation and macronutrient selection between experimental conditions. The disadvantage of the buffet-style approach is that it provides a less realistic representation of what individuals might be exposed to within their normal mealtime environment; further, an increased variety of foods has been associated with body fatness in adults (125, 126), supporting the problematic nature of an excess of food variety and choice among adults at mealtime.

In children, there is no evidence that variety of food in test meals is a factor in overriding signals from previously consumed foods. Sugar preloads in the form of drinks decreased subsequent cookie consumption in children aged 9–10 y, suggesting sensory-specific satiety for sweet foods (127). Others found that preschool children, when given food choices, accomplished caloric compensation in test meals after a preload by selectively reducing the intake of nonpreferred foods and maintaining consumption of highly preferred foods (128, 129). Whether or not obese children overeat when presented with a large variety of food in a meal, or if macronutrient composition of the preload is a determinant of the outcome, is unknown. In both instances, providing energy in excess of requirements is in contrast to typical meal patterns in which individuals generally consume a meal of a fixed size; these factors may account for the lack of external validity of laboratory studies and those occurring in the real world.

In addition, the effect of childhood obesity on the regulation of short-term food intake was only recently reported. Several cross-sectional studies in children showed a positive relation between dietary fat intake and body fat mass (130, 131). It was suggested that the accumulation of adipose tissue may be a negative factor affecting food intake control (132), such that excess adipose tissue may reduce satiety signals derived from dietary fat contributing to further weight gain. Body fatness, which is based on skinfold measurement, was inversely associated with intake reduction at a later meal in girls aged 3–5 y, but not in boys, after they consumed sucrose and low-glucose maltodextrin in beverages (132). Because the boys compensated (55%) for a significantly greater percentage of energy in the preloads than did the girls (35%), the authors suggested that the difference in compensation was likely attributable in part to a greater proportion of body fat in the girls.

Furthermore, in comparative studies of appetite and food intake in normal-weight compared with overweight and obese children, preloads or meals should be provided on the basis of body weight or composition. It has been reported that obese children fail to suppress energy intake to the same extent as normal weight children when snacks or preloads are provided in a fixed rather than on a body weight basis (116).

These observations may be macronutrient specific: it was reported that food intake suppression at a test meal 30 min after ingestion of a 50-g glucose drink was decreased to a similar extent in normal-weight and obese boys, whereas obese boys did not respond as well to a preload consisting of protein (116). These findings suggest that the effect of protein, but not carbohydrate, may be body-weight or composition dependent.

Finally, hyperinsulinemia has been proposed as one metabolic explanation for the reduced precision of intake regulation in obesity. It is probable that ~30% of overweight children between 10 and 13 y of age are insulin resistant (133). How hyperinsulinemia affects satiety signals in children arising from each of the macronutrients is unknown. When compared with normal-weight subjects, obese boys showed passive overconsumption of high-fat diets; the authors proposed hyperinsulinemia as the explanation (134). There is support for the hypothesis that hyperinsulinemia may be the "price to pay for body weight stability," as proposed by Tremblay et al. (135). Hyperinsulinemic men compensated better than normo-insulinemic men at a test meal after a glucose preload (136). After the glucose drink, plasma insulin, cholecystokinin, and leptin were higher, and ghrelin and adiponectin were lower in hyperinsulinemic men than they were in normo-insulinemic men (136). It remains unknown how body fat and associated hormones in the presence or absence of insulin resistance affects food intake in children. Perhaps hyperinsulinemia during puberty, a normal transition during Tanner stages 1–5 (137–139), is also a mechanism by which prepubertal body fat is reduced. Although insulin resistance seems to oppose further weight gain in adults, opposite results have been found in children (135). The gut produces ≥1 orexigenic peptide; a preprandial rise in ghrelin concentrations is associated with meal initiation (140, 141). Mean ghrelin concentrations are higher in children in Tanner stage 1 than in children in Tanner stages 4–5 (142); thus, Tanner stage may need to be included as a covariate in future studies. Therefore, it is of interest to understand the effect of hyperinsulinemia and the interaction of insulin and other satiety hormones on food intake control not only in obese children, but also in normal-weight children.

Research Recommendations

The research recommendations for studies examining the effect of breakfast on cognition and appetite are summarized in **Table 1** and **Table 2**, respectively.

Conclusions

In conclusion, there is a general recognition and understanding of the methodological limitations and issues encountered when conducting studies on the benefits of breakfast, particularly in children. Given the inconsistent findings and the potential importance of clarifying the role of breakfast and breakfast composition on postmeal behaviors (i.e., appetite and cognitive performance), future work controlling for these methodological shortcomings is warranted.

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References

1. Rampersaud GC, Pereira MA, Girard BL, Adams J, Metzl JD. Breakfast habits, nutritional status, body weight, and academic performance in children and adolescents. J Am Diet Assoc 2005;105:743–60.
2. Deshmukh-Taskar PR, Nicklas TA, O'Neil CE, Keast DR, Radcliffe JD, Cho S. The relationship of breakfast skipping and type of breakfast consumption with nutrient intake and weight status in children and adolescents: the National Health and Nutrition Examination Survey 1999–2006. J Am Diet Assoc 2010;110:869–78.
3. O'Neil CE, Nicklas TA, Fulgoni, 3rd VL. Nutrient intake, diet quality, and weight/adiposity parameters in breakfast patterns compared with no breakfast in adults: National Health and Nutrition Examination Survey 2001–2008. J Acad Nutr Diet 2014;114(12, Suppl)S27–43.
4. Szajewska H, Ruszczynski M. Systematic review demonstrating that breakfast consumption influences body weight outcomes in children and adolescents in Europe. Crit Rev Food Sci Nutr 2010;50:113–9.
5. De La Hunty A, Ashwell M. Are people who regularly eat breakfast cereals slimmer than those who don't? A systematic review of the evidence. Nutr Bull 2007;32:118–28.
6. Mesas AE, Munoz-Pareja M, Lopez-Garcia E, Rodriguez-Artalejo F. Selected eating behaviours and excess body weight: a systematic review. Obes Rev 2012;13:106–35.
7. Adolphus K, Lawton CL, Champ CL, Dye L. The effects of breakfast and breakfast composition on cognition in children and adolescents: a systematic review. Adv Nutr 2016;7:590S–612S.
8. Leidy HJ, Gwin JA, Roenfeldt CA, Zino AZ, Shafer RS. Evaluating the intervention-based evidence surrounding the causal role of breakfast on markers of weight management, with specific focus on breakfast composition and size. Adv Nutr 2016;7:563S–75S.
9. Rosado JL, del R Arellano M, Montemayor K, García OP, Caamaño M del C. An increase of cereal intake as an approach to weight reduction in children is effective only when accompanied by nutrition education: a randomized controlled trial. Nutr J 2008;7:28.
10. Kral TV, Heo M, Whiteford LM, Faith MS. Effects on cognitive performance of eating compared with omitting breakfast in elementary schoolchildren. J Dev Behav Pediatr 2012;33:9–16.
11. de Jager CA, Dye L, de Bruin EA, Butler L, Fletcher J, Lamport DJ, Latulippe ME, Spencer JPE, Wesnes K. Criteria for validation and selection of cognitive tests for investigating the effects of foods and nutrients. Nutr Rev 2014;72:162–79.
12. Defeyter MA, Russo R. The effect of breakfast cereal consumption on adolescents' cognitive performance and mood. Front Hum Neurosci 2013;7:789.
13. Mahoney CR, Taylor HA, Kanarek RB, Samuel P. Effect of breakfast composition on cognitive processes in elementary school children. Physiol Behav 2005;85:635–45.
14. Wesnes KA, Pincock C, Richardson D, Helm G, Hails S. Breakfast reduces declines in attention and memory over the morning in schoolchildren. Appetite 2003;41:329–31.
15. Widenhorn-Müller K, Hille K, Klenk J, Weiland U. Influence of having breakfast on cognitive performance and mood in 13- to 20-year-old high school students: results of a crossover trial. Pediatrics 2008;122:279–84.
16. EFSA. Guidance on the scientific requirements for health claims related to functions of the nervous system, including psychological functions. EFSA Journal 2012;10:2816.
17. Cooper SB, Bandelow S, Nevill ME. Breakfast consumption and cognitive function in adolescent schoolchildren. Physiol Behav 2011;103:431–9.
18. Brindal E, Baird D, Danthiir V, Wilson C, Bowen J, Slater A, Noakes M. Ingesting breakfast meals of different glycaemic load does not alter cognition and satiety in children. Eur J Clin Nutr 2012;66(10):1166–71.

19. Brindal E, Baird D, Slater A, Danthiir V, Wilson C, Bowen J, Noakes M. The effect of beverages varying in glycaemic load on postprandial glucose responses, appetite and cognition in 10–12-year-old school children. Br J Nutr 2013;110:529–37.
20. Ingwersen J, Defeyter MA, Kennedy DO, Wesnes K, Scholey AB. A low glycaemic index breakfast cereal preferentially prevents children's cognitive performance from declining throughout the morning. Appetite 2007;49:240–4.
21. Cooper SB, Bandelow S, Nute ML, Morris JG, Nevill ME. Breakfast glycaemic index and cognitive function in adolescent school children. Br J Nutr 2012;107:1823–32.
22. Richter LM, Rose C, Griesel RD. Cognitive and behavioural effects of a school breakfast. S Afr Med J 1997;87(1, Suppl)93–100.
23. Shemilt I, Harvey I, Shepstone L, Swift L, Reading R, Mugford M, Belderson P, Norris N, Thoburn J, Robinson J. A national evaluation of school breakfast clubs: evidence from a cluster randomized controlled trial and an observational analysis. Child Care Health Dev 2004;30:413–27.
24. Murphy S, Moore GF, Tapper K, Lynch R, Clarke R, Raisanen L, Desousa C, Moore L. Free healthy breakfasts in primary schools: a cluster randomised controlled trial of a policy intervention in Wales, United Kingdom. Public Health Nutr 2011;14:219–26.
25. Cueto S, Chinen M. Educational impact of a school breakfast programme in rural Peru. Int J Educ Dev 2008;28:132–48.
26. Taki Y, Hashizume H, Sassa Y, Takeuchi H, Asano M, Asano K, Kawashima R. Breakfast staple types affect brain gray matter volume and cognitive function in healthy children. PLoS One 2010;5:e15213.
27. Gajre NS, Fernandez S, Balakrishna N, Vazir S. Breakfast eating habit and its influence on attention-concentration, immediate memory and school achievement. Indian Pediatr 2008;45:824–8.
28. Liu J, Hwang W-T, Dickerman B, Compher C. Regular breakfast consumption is associated with increased IQ in kindergarten children. Early Hum Dev 2013;89(4):257–62.
29. Mohd Nasir MT, Norimah AK, Hazizi AS, Nurliyana AR, Loh SH, Suraya I. Child feeding practices, food habits, anthropometric indicators and cognitive performance among preschoolers in Peninsular Malaysia. Appetite 2012;58:525–30.
30. Luciana M, Nelson CA. Assessment of neuropsychological function through use of the Cambridge Neuropsychological Testing Automated Battery: performance in 4- to 12-year-old children. Dev Neuropsychol 2002;22:595–624.
31. Calvin CM, Fernandes C, Smith P, Visscher PM, Deary IJ. Sex, intelligence and educational achievement in a national cohort of over 175,000 11-year-old schoolchildren in England. Intelligence 2010;38:424–32.
32. Strand S, Deary IJ, Smith P. Sex differences in Cognitive Abilities Test scores: A UK national picture. Br J Educ Psychol 2006;76:463–80.
33. McCulloch A, Joshi HE. Neighbourhood and family influences on the cognitive ability of children in the British National Child Development Study. Soc Sci Med 2001;53:579–91.
34. Elbourne DR, Altman DG, Higgins JP, Curtin F, Worthington HV, Vail A. Meta-analyses involving cross-over trials: methodological issues. Int J Epidemiol 2002;31:140–9.
35. Pivik RT, Tennal KB, Chapman SD, Gu Y. Eating breakfast enhances the efficiency of neural networks engaged during mental arithmetic in school-aged children. Physiol Behav 2012;106:548–55.
36. Pollitt E, Leibel R, Greenfield D. Brief fasting, stress, and cognition in children. Am J Clin Nutr 1981;34:1526–33.
37. Fulford J, Varley-Campbell JL, Williams CA. The effect of breakfast versus no breakfast on brain activity in adolescents when performing cognitive tasks, as assessed by fMRI. Nutr Neurosci 2016;19:110–5.
38. McCarney R, Warner J, Iliffe S, van Haselen R, Griffin M, Fisher P. The Hawthorne Effect: a randomised, controlled trial. BMC Med Res Methodol 2007;7:30.
39. Schmuckler MA. What is ecological validity? A dimensional analysis. Infancy 2001;2:419–36.
40. Maffeis C, Fornari E, Surano MG, Comencini E, Corradi M, Tommasi M, Fasan I, Cortese S. Breakfast skipping in prepubertal obese children: hormonal, metabolic and cognitive consequences. Eur J Clin Nutr 2012;66:314–21.
41. Cromer BA, Tarnowski KJ, Stein AM, Harton P, Thornton DJ. The School Breakfast Program and cognition in adolescents. J Dev Behav Pediatr 1990;11:295–300.
42. di Stephano J. How much power is enough? Against the development of an arbitrary convention for statistical power calculations. Funct Ecol 2003;17:707–9.
43. Muthayya S, Thomas T, Srinivasan K, Rao K, Kurpad AV, van Klinken J-W, Owen G, de Bruin EA. Consumption of a mid-morning snack improves memory but not attention in school children. Physiol Behav 2007;90:142–50.
44. Chugani HT. A critical period of brain development: studies of cerebral glucose utilization with PET. Prev Med 1998;27:184–8.
45. Livingstone MB, Robson PJ, Wallace JM. Issues in dietary intake assessment of children and adolescents. Br J Nutr 2004;92 Suppl 2: S213–22.
46. Busch CR, Taylor HA, Kanarek RB, Holcomb PJ. The effects of a confectionery snack on attention in young boys. Physiol Behav 2002;77: 333–40.
47. Dickie NH, Bender AE. Breakfast and performance in schoolchildren. Br J Nutr 1982;48:483–96.
48. Benton D, Maconie A, Williams C. The influence of the glycaemic load of breakfast on the behaviour of children in school. Physiol Behav 2007;92:717–24.
49. Pollitt E, Cueto S, Jacoby E. Fasting and cognition in well- and undernourished schoolchildren: a review of three experimental studies. Am J Clin Nutr 1998;67:779S–84S.
50. Chandler AM, Walker SP, Connolly K, Grantham-McGregor SM. School breakfast improves verbal fluency in undernourished Jamaican children. J Nutr 1995;125:894–900.
51. Crum RM, Anthony JC, Bassett SS, Folstein MF. Population-based norms for the mini-mental state examination by age and educational level. JAMA 1993;269:2386–91.
52. Wesnes KA. Evaluation of techniques to identify beneficial effects of nutrition and natural products on cognitive function. Nutr Rev 2010;68:S22–8.
53. Hoyland A, Lawton CL, Dye L. Acute effects of macronutrient manipulations on cognitive test performance in healthy young adults: a systematic research review. Neurosci Biobehav Rev 2008;32:72–85.
54. Defeyter MA, Graham PL, Walton J, Apicella T. News and views: breakfast clubs: availability for British schoolchildren and the nutritional, social and academic benefits. Nutr Bull 2010;35:245–53.
55. Cueto S, Jacoby E, Pollitt E. Breakfast prevents delays of attention and memory functions among nutritionally at-risk boys. J Appl Dev Psychol 1998;19:219–33.
56. Simeon DT, Grantham-Mcgregor S. Effects of missing breakfast on the cognitive functions of school-children of differing nutritional status. Am J Clin Nutr 1989;49:646–53.
57. Pollitt E, Lewis NL, Garza C, Shulman RJ. Fasting and cognitive function. J Psychiatr Res 1982–1983;17:169–74.
58. Isaacs E, Oates J. Nutrition and cognition: assessing cognitive abilities in children and young people. Eur J Nutr 2008;47:4–24.
59. Macht M, Mueller J. Immediate effects of chocolate on experimentally induced mood states. Appetite 2007;49:667–74.
60. Dye L, Blundell J. Functional foods: psychological and behavioural functions. Br J Nutr 2002;88:S187–211.
61. Hetherington MM, Cunningham K, Dye L, Gibson EL, Gregersen NT, Halford JC, Lawton CL, Lluch A, Mela DJ, Van Trijp HC. Potential benefits of satiety to the consumer: scientific considerations. Nutr Res Rev 2013;26:22–38.
62. Schmitt JA, Benton D, Kallus KW. General methodological considerations for the assessment of nutritional influences on human cognitive functions. Eur J Nutr 2005;44:459–64.
63. Lloyd HM, Green MW, Rogers PJ. Mood and cognitive performance effects of isocaloric lunches differing in fat and carbohydrate content. Physiol Behav 1994;56:51–7.
64. Wyon DP, Abrahamsson L, Jartelius M, Fletcher RJ. An experimental study of the effects of energy intake at breakfast on the test performance of 10-year-old children in school. Int J Food Sci Nutr 1997;48:5–12.

65. Chapman GE, Melton CL, Hammond GK. College and university students' breakfast consumption patterns: behaviours, beliefs, motivations and personal and environmental influences. Can J Diet Pract Res 1998;59:176–82.
66. Eilat-Adar S, Koren-Morag N, Siman-Tov M, Livne I, Altmen H. School-based intervention to promote eating daily and healthy breakfast: a survey and a case-control study. Eur J Clin Nutr 2011;65:203–9.
67. Reddan J, Wahlstrom K, Reicks M. Children's perceived benefits and barriers in relation to eating breakfast in schools with or without Universal School Breakfast. J Nutr Educ Behav 2002;34:47–52.
68. López I, de Andraca I, Perales CG, Heresi E, Castillo M, Colombo M. Breakfast omission and cognitive performance of normal, wasted, and stunted school children. Eur J Clin Nutr 1993;47:533–42.
69. Nkhoma OWW, Duffy ME, Cory-Slechta DA, Davidson PW, McSorley EM, Strain JJ, O'Brien GM. Early-stage primary school children attending a school in the Malawian School Feeding Program (SFP) have better reversal learning and lean muscle mass growth than those attending a non-SFP school. J Nutr 2013;143:1324–30.
70. Lieberman HM, Hunt IF, Coulson AH, Clark VA, Swendseid ME, Ho L. Evaluation of a ghetto school breakfast program. J Am Diet Assoc 1976;68:132–8.
71. Kothe EJ, Mullan B. Increasing the frequency of breakfast consumption. Br Food J 2011;113:784–96.
72. Kleinman RE, Hall S, Green H, Korzec-Ramirez D, Patton K, Pagano ME, Murphy JM. Diet, breakfast, and academic performance in children. Ann Nutr Metab 2002;46 Suppl 1:24–30.
73. Murphy JM, Pagano ME, Nachmani J, Sperling P, Kane S, Kleinman RE. The relationship of school breakfast to psychosocial and academic functioning: cross-sectional and longitudinal observations in an inner-city school sample. Arch Pediatr Adolesc Med 1998;152:899–907.
74. Wahlstrom KL, Begalle MS. More than test scores: results of the Universal School Breakfast Pilot in Minnesota. Topics Clin Nutr 1999;15: 17–29.
75. Messier C. Glucose improvement of memory: a review. Eur J Pharmacol 2004;490:33–57.
76. Falkingham M, Abdelhamid A, Curtis P, Fairweather-Tait S, Dye L, Hooper L. The effects of oral iron supplementation on cognition in older children and adults: a systematic review and meta-analysis. Nutr J 2010;9:4.
77. Jáuregui-Lobera I. Iron deficiency and cognitive functions. Neuropsychiatr Dis Treat 2014;10:2087–95.
78. Jacoby E, Cueto S, Pollitt E. Benefits of a school breakfast programme among Andean children in Huaraz, Peru. Food Nutr Bull 1996;17:54–64.
79. McNamee R. Confounding and confounders. Occup Environ Med 2003;60:227–34.
80. O'Neil CE, Byrd-Bredbenner C, Hayes D, Jana L, Klinger SE, Stephenson-Martin S. The role of breakfast in health: definition and criteria for a quality breakfast. J Acad Nutr Diet 2014;114(12, Suppl)S8–S26.
81. Baldinger N, Krebs A, Muller R, Aeberli I. Swiss children consuming breakfast regularly have better motor functional skills and are less overweight than breakfast skippers. J Am Coll Nutr 2012;31:87–93.
82. Ghazi HF, Isa ZM, Aljunid S, Tamil AM, Abdalqader MA. Nutritional status, nutritional habit and breakfast intake in relation to IQ among primary school children in Baghdad city, Iraq. Pak J Nutr 2012;11:379–82.
83. Cho S, Dietrich M, Brown CJ, Clark CA, Block G. The effect of breakfast type on total daily energy intake and body mass index: results from the Third National Health and Nutrition Examination Survey (NHANES III). J Am Coll Nutr 2003;22:296–302.
84. van der Heijden AA, Hu FB, Rimm EB, van Dam RM. A prospective study of breakfast consumption and weight gain among U.S. men. Obesity (Silver Spring) 2007;15:2463–9.
85. Geliebter A, Astbury NM, Aviram-Friedman R, Yahav E, Hashim S. Skipping breakfast leads to weight loss but also elevated cholesterol compared with consuming daily breakfasts of oat porridge or frosted cornflakes in overweight individuals: a randomised controlled trial. J Nutr Sci 2014;3:e56.
86. Corder K, van Sluijs EM, Ridgway CL, Steele RM, Prynne CJ, Stephen AM, Bamber DJ, Dunn VJ, Goodyer IM, Ekelund U. Breakfast consumption and physical activity in adolescents: daily associations and hourly patterns. Am J Clin Nutr 2014;99(2):361–8.
87. Papoutsou S, Briassoulis G, Wolters M, Peplies J, Iacoviello L, Eiben G, Veidebaum T, Molnar D, Russo P, Michels N, et al. No breakfast at home: association with cardiovascular disease risk factors in childhood. Eur J Clin Nutr 2014;68:829–34.
88. Albertson AM, Thompson D, Franko DL, Kleinman RE, Barton BA, Crockett SJ. Consumption of breakfast cereal is associated with positive health outcomes: evidence from the National Heart, Lung, and Blood Institute Growth and Health Study. Nutr Res 2008;28:744–52.
89. Astbury NM, Taylor MA, Macdonald IA. Breakfast consumption affects appetite, energy intake, and the metabolic and endocrine responses to foods consumed later in the day in male habitual breakfast eaters. J Nutr 2011;141:1381–9.
90. Levitsky DA, Pacanowski CR. Effect of skipping breakfast on subsequent energy intake. Physiol Behav 2013;119:9–16.
91. Woods SC. Gastrointestinal satiety signals I. An overview of gastrointestinal signals that influence food intake. Am J Physiol Gastrointest Liver Physiol 2004;286:G7–13.
92. Astrup A. Carbohydrates as macronutrients in relation to protein and fat for body weight control. Int J Obes 2006;30 S3:S4–9.
93. Blundell JE, Lawton CL, Cotton JR, Macdiarmid JI. Control of human appetite: implications for the intake of dietary fat. Annu Rev Nutr 1996;16:285–319.
94. Gerstein DE, Woodward-Lopez G, Evans AE, Kelsey K, Drewnowski A. Clarifying concepts about macronutrients' effects on satiation and satiety. J Am Diet Assoc 2004;104:1151–3.
95. Van Kleef E, Van Trijp JC, Van Den Borne JJ, Zondervan C. Successful development of satiety enhancing food products: towards a multidisciplinary agenda of research challenges. Crit Rev Food Sci Nutr 2012; 52:611–28.
96. Blundell JE, Rogers PJ, Hill AJ. Evaluating the satiating power of foods: Implications for acceptance and consumption. In: Solms J, Booth DA, Pangbourne RM, Raunhardt O, editors. Food acceptance and nutrition. London: Academic Press; 1987. p. 205–19.
97. Mikkelsen PB, Toubro S, Astrup A. Effect of fat-reduced diets on 24-h energy expenditure: comparisons between animal protein, vegetable protein, and carbohydrate. Am J Clin Nutr 2000;72:1135–41.
98. Hill AJ, Blundell JE. Nutrients and behaviour: research strategies for the investigation of taste characteristics, food preferences, hunger sensations and eating patterns in man. J Psychiatr Res 1982–1983;17:203–12.
99. Flint A, Raben A, Blundell JE, Astrup A. Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. Int J Obes Relat Metab Disord 2000;24:38–48.
100. Mattes R. Hunger ratings are not a valid proxy measure of reported food intake in humans. Appetite 1990;15:103–13.
101. Stubbs RJ, Hughes DA, Johnstone AM, Rowley E, Reid C, Elia M, Stratton R, Delargy H, King N, Blundell JE. The use of visual analogue scales to assess motivation to eat in human subjects: a review of their reliability and validity with an evaluation of new hand-held computerized systems for temporal tracking of appetite ratings. Br J Nutr 2000;84:405–15.
102. Spence C, Okajima K, Cheok AD, Petit O, Michel C. Eating with our eyes: from visual hunger to digital satiation. Brain Cogn.
103. Rodin J, Marcus J. Psychological factors in human feeding. Pharmacol Ther 1982;16:447–68.
104. Shields BJ, Palermo TM, Powers JD, Grewe SD, Smith GA. Predictors of a child's ability to use a visual analogue scale. Child Care Health Dev 2003;29:281–90.
105. Bellissimo N, Thomas SG, Pencharz PB, Goode RC, Anderson GH. Reproducibility of short-term food intake and subjective appetite scores after a glucose preload, ventilation threshold, and body composition in boys. Appl Physiol Nutr Metab 2008;33:326–37.

106. Houchins JA, Tan SY, Campbell WW, Mattes RD. Effects of fruit and vegetable, consumed in solid vs beverage forms, on acute and chronic appetitive responses in lean and obese adults. Int J Obes (Lond) 2013; 37:1109–15.
107. Haber GB, Heaton KW, Murphy D, Burroughs LF. Depletion and disruption of dietary fibre. Effects on satiety, plasma-glucose, and serum-insulin. Lancet 1977;2:679–82.
108. Cassady BA, Considine RV, Mattes RD. Beverage consumption, appetite, and energy intake: what did you expect? Am J Clin Nutr 2012;95:587–93.
109. Zhu Y, Hsu WH, Hollis JH. The impact of food viscosity on eating rate, subjective appetite, glycemic response and gastric emptying rate. PLoS One 2013;8:e67482.
110. Martin CK, Anton SD, Walden H, Arnett C, Greenway FL, Williamson DA. Slower eating rate reduces the food intake of men, but not women: implications for behavioral weight control. Behav Res Ther 2007;45:2349–59.
111. Patel BP, Bellissimo N, Luhovyy B, Bennett LJ, Hurton E, Painter JE, Anderson GH. An after-school snack of raisins lowers cumulative food intake in young children. J Food Sci 2013;78 Suppl 1:A5–10.
112. Faith MS, Kermanshah M, Kissileff HR. Development and preliminary validation of a silhouette satiety scale for children. Physiol Behav 2002; 76:173–8.
113. Anderson GH, Woodend D. Consumption of sugars and the regulation of short-term satiety and food intake. Am J Clin Nutr 2003;78:843S–9S.
114. Cecil JE, Palmer CN, Wrieden W, Murrie I, Bolton-Smith C, Watt P, Wallis DJ, Hetherington MM. Energy intakes of children after preloads: adjustment, not compensation. Am J Clin Nutr 2005;82:302–8.
115. Bellissimo N, Pencharz PB, Thomas SG, Anderson GH. Effect of television viewing at mealtime on food intake after a glucose preload in boys. Pediatr Res 2007;61:745–9.
116. Bellissimo N, Desantadina MV, Pencharz PB, Berall GB, Thomas SG, Anderson GH. A comparison of short-term appetite and energy intakes in normal weight and obese boys following glucose and whey-protein drinks. Int J Obes (Lond) 2008;32:362–71.
117. Branton A, Akhavan T, Gladanac B, Pollard D, Welch J, Rossiter M, Bellissimo N. Pre-meal video game playing and a glucose preload suppress food intake in normal weight boys. Appetite 2014;83:256–62.
118. Patel BP, Bellissimo N, Thomas SG, Hamilton JK, Anderson GH. Television viewing at mealtime reduces caloric compensation in peripubertal, but not postpubertal, girls. Pediatr Res 2011;70:513–7.
119. Bellissimo N, Thomas SG, Goode RC, Anderson GH. Effect of short-duration physical activity and ventilation threshold on subjective appetite and short-term energy intake in boys. Appetite 2007;49:644–51.
120. Zandstra EH, Mathey MF, Graaf C, van Staveren WA. Short-term regulation of food intake in children, young adults and the elderly. Eur J Clin Nutr 2000;54:239–46.
121. De Graaf C, De Jong LS, Lambers AC. Palatability affects satiation but not satiety. Physiol Behav 1999;66:681–8.
122. Melanson KJ, Westerterp-Plantenga MS, Saris WH, Smith FJ, Campfield LA. Blood glucose patterns and appetite in time-blinded humans: carbohydrate versus fat. Am J Physiol 1999;277:R337–45.
123. Thivel D, Genin PM, Mathieu M-E, Pereira B, Metz L. Reproducibility of an in-laboratory test meal to assess ad libitum energy intake in adolescents with obesity. Appetite 2016;105:129–33.
124. Wisniewski L, Epstein LH, Caggiula AR. Effect of food change on consumption, hedonics, and salivation. Physiol Behav 1992;52:21–6.
125. McCrory MA, Fuss PJ, McCallum JE, Yao M, Vinken AG, Hays NP, Roberts SB. Dietary variety within food groups: association with energy intake and body fatness in men and women. Am J Clin Nutr 1999;69:440–7.
126. Raynor HA, Epstein LH. Dietary variety, energy regulation, and obesity. Psychol Bull 2001;127:325–41.
127. Anderson GH, Saravis S, Schacher R, Zlotkin S, Leiter LA. Aspartame: effect on lunch-time food intake, appetite and hedonic response in children. Appetite 1989;13:93–103.
128. Birch LL, McPhee L, Sullivan S. Children's food intake following drinks sweetened with sucrose or aspartame: time course effects. Physiol Behav 1989;45:387–95.
129. Birch LL, McPhee LS, Bryant JL, Johnson SL. Children's lunch intake: effects of midmorning snacks varying in energy density and fat content. Appetite 1993;20:83–94.
130. Gazzaniga JM, Burns TL. Relationship between diet composition and body fatness, with adjustment for resting energy expenditure and physical activity, in preadolescent children. Am J Clin Nutr 1993;58: 21–8.
131. Nguyen VT, Larson DE, Johnson RK, Goran MI. Fat intake and adiposity in children of lean and obese parents. Am J Clin Nutr 1996;63: 507–13.
132. Johnson SL, Birch LL. Parents' and children's adiposity and eating style. Pediatrics 1994;94:653–61.
133. Shaibi GQ, Ball GD, Cruz ML, Weigensberg MJ, Salem GJ, Goran MI. Cardiovascular fitness and physical activity in children with and without impaired glucose tolerance. Int J Obes (Lond) 2006; 30:45–9.
134. Lawton CL, Burley VJ, Wales JK, Blundell JE. Dietary fat and appetite control in obese subjects: weak effects on satiation and satiety. Int J Obes Relat Metab Disord 1993;17:409–16.
135. Tremblay A, Boule N, Doucet E, Woods SC. Is the insulin resistance syndrome the price to be paid to achieve body weight stability? Int J Obes (Lond) 2005;29:1295–8.
136. Samra RA, Wolever TM, Anderson GH. Enhanced food intake regulatory responses after a glucose drink in hyperinsulinemic men. Int J Obes (Lond) 2007;31:1222–31.
137. Moran A, Jacobs DR, Jr., Steinberger J, Hong CP, Prineas R, Luepker R, Sinaiko AR. Insulin resistance during puberty: results from clamp studies in 357 children. Diabetes 1999;48:2039–44.
138. Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. Arch Dis Child 1969;44:291–303.
139. Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. Arch Dis Child 1970;45:13–23.
140. Cummings DE, Purnell JQ, Frayo RS, Schmidova K, Wisse BE, Weigle DS. A preprandial rise in plasma ghrelin levels suggests a role in meal initiation in humans. Diabetes 2001;50:1714–9.
141. Shintani M, Ogawa Y, Ebihara K, Aizawa-Abe M, Miyanaga F, Takaya K, Hayashi T, Inoue G, Hosoda K, Kojima M, et al. Ghrelin, an endogenous growth hormone secretagogue, is a novel orexigenic peptide that antagonizes leptin action through the activation of hypothalamic neuropeptide Y/Y1 receptor pathway. Diabetes 2001;50:227–32.
142. Pomerants T, Tillmann V, Jurimae J, Jurimae T. Relationship between ghrelin and anthropometrical, body composition parameters and testosterone levels in boys at different stages of puberty. J Endocrinol Invest 2006;29:962–7.