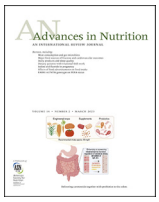
Advances in Nutrition 14 (2023) 339–351

American Society for Nutrition
Excellence in Nutrition Research and Practice

Advances in Nutrition
AN INTERNATIONAL REVIEW JOURNAL
journal homepage: www.journals.elsevier.com/advances-in-nutrition



Review

The Effects of Food Advertisements on Food Intake and Neural Activity: A Systematic Review and Meta-Analysis of Recent Experimental Studies

Pablo Arrona-Cardoza [1,†], Katherine Labonté [1,†], José Miguel Cisneros-Franco [1,2], Daiva E. Nielsen [1,*]

[1] *School of Human Nutrition, McGill University, Sainte-Anne-de-Bellevue, Canada;* [2] *Desautels Faculty of Management, McGill University, Montreal, Canada*

A B S T R A C T

Food advertisements are ubiquitous in our daily environment. However, the relationships between exposure to food advertising and outcomes related to ingestive behavior require further investigation. The objective was to conduct a systematic review and meta-analysis of behavioral and neural responses to food advertising in experimental studies. PubMed, Web of Science, and Scopus were searched for articles published from January 2014 to November 2021 using a search strategy following PRISMA guidelines. Experimental studies conducted with human participants were included. A random-effects inverse-variance meta-analysis was performed on standardized mean differences (SMD) of food intake (behavioral outcome) between the food advertisement and nonfood advertisement conditions of each study. Subgroup analyses were performed by age, BMI group, study design, and advertising media type. A seed-based d mapping meta-analysis of neuroimaging studies was performed to evaluate neural activity between experimental conditions. Nineteen articles were eligible for inclusion, 13 for food intake ($n = 1303$) and 6 for neural activity ($n = 303$). The pooled analysis of food intake revealed small, but statistically significant, effects of increased intake after viewing food advertising compared with the control condition among adults and children (adult SMD: 0.16; 95% CI: 0.03, 0.28; $P = 0.01$; $I^2 = 0$; 95% CI: 0, 95.0%; Children SMD: 0.25; 95% CI: 0.14, 0.37; $P < 0.0001$; $I^2 = 60.4\%$; 95% CI: 25.6%, 79.0%). The neuroimaging studies involved children only, and the pooled analysis corrected for multiple comparisons identified one significant cluster, the middle occipital gyrus, with increased activity after food advertising exposure compared with the control condition (peak coordinates: 30, −86, 12; *z*-value: 6.301, size: 226 voxels; $P < 0.001$). These findings suggest that acute exposure to food advertising increases food intake among children and adults and that the middle occipital gyrus is an implicated brain region among children. (PROSPERO registration: CRD42022311357)

Keywords: food advertising, food intake, neural activity, neuroimaging, meta-analysis

Statements of Significance

This systematic review and meta-analysis of neurobehavioral responses to food advertising indicated a statistically significant overall pooled effect of food advertising on food intake among both children and adults. This is a novel and relevant finding because previous work has only detected an effect of food advertising on food intake among children.

Abbreviations: RoB 2, Cochrane Risk of Bias tool; SDM, Seed-based d mapping; SMD, standardized mean difference; TFCE, threshold-free-cluster enhancement.

* Corresponding author. 21,111 Lakeshore Road, School of Human Nutrition, McGill University, Sainte-Anne-de-Bellevue, QC H9X 3V9, Canada. *E-mail address:* daiva.nielsen@mcgill.ca (D.E. Nielsen).

† PAC and KL contributed equally to this work.

https://doi.org/10.1016/j.advnut.2022.12.003
Received 3 August 2022; Received in revised form 1 December 2022; Accepted 22 December 2022; Available online 31 December 2022

Introduction

It is estimated that 2.1 billion individuals (37% of men and 38% of women) worldwide are overweight (BMI, 25–29.9 kg/m^2), while a third of these (~680 million) are classified as living with obesity (BMI ≥30 kg/m^2) [1]. Overweight and obesity are among the top risk factors for diabetes, hypertension, stroke, and heart disease [1, 2]. Moreover, among 84 risk factors evaluated by the 2017 Global Burden of Disease Study [3], excess weight had the greatest increase in prevalence since 1990 and was among the top 5 risk factors in terms of attributable deaths and disability-adjusted life-years. The increase in obesity rates worldwide is likely the result of multiple factors, including changes in eating behavior [4], genetic susceptibility [5], reduction in physical activity [6], and agricultural innovation and business practices that have resulted in calorie-dense foods being easily accessible and affordable [7].

Evidence from genetics and brain imaging studies suggests that obesity is inherited via brain function [8, 9]. Notably, 75% of obesity-related genes are preferentially expressed in the brain [8] and there is considerable genetic overlap among obesity, cognitive test scores, and brain imaging findings [9–12]. Indeed, depending on considerations of eating behaviors and or/BMI groups, obesity has been associated with differences in activity in the orbitofrontal cortex, amygdala, and hippocampus; brain areas involved in decision-making, impulse control, and learning [13], and a recent study found the associations between cortical thickness and BMI to be mediated by impulsivity and uncontrolled eating [14]. Research using fMRI further shows that these responses can be significantly modulated by food-related marketing exposures, such as food advertising in children [15] and adolescents [16], or more general exposures to food images among adults [17]. Taken together, these findings support the notion of obesity as a neurobehavioral condition [18] and underscore the need to integrate neuroimaging techniques alongside other measures in the study of the effects of food-related stimuli, such as food advertising, on ingestive behavior.

An intriguing aspect of ingestive behavior that warrants investigation in the context of exposure to food advertising is food cue reactivity. According to the "cued overeating model," food cue reactivity is a set of physiologically conditioned responses to food cues, such as increased salivation, heart rate, and neural activity, that may lead to increased eating [19]. Research has suggested that different forms of food advertising, such as commercials, advergames (online-based videogames used to promote a product or brand), and logos, could act as cues that influence ingestive behaviors, specifically toward consuming highly processed hyperpalatable foods [20–22]. A previous meta-analysis found that short-term exposure to unhealthy food advertising, including television food commercials and advergames, increased immediate calorie consumption in children, but not in adults [23]. Likewise, exposure to food commercials and subsequent striatum activation was associated with increased BMI over 1-y follow-up in adolescents [24]. Further, a separate meta-analysis of brain responses to watching commercials found that food commercials caused more significant brain responses in the cuneus on both the hemispheres and right middle occipital gyrus than nonfood commercials [25], highlighting a crucial role of the brain in the relationship between food advertising exposure, food intake, and obesity outcomes.

Although food cue reactivity may be an important contributor to the behavioral and neural responses to food advertising, there is interindividual variability in food cue reactivity [26]. These interindividual differences may, in part, account for the differences in ingestive behaviors that are implicated in overweight and obesity. Moreover, forms of food advertising have changed in recent years with increased exposure to advertising through online media, including social media platforms and new forms of marketing practices, such as advergames [27, 28]. Therefore, a comprehensive review of recent studies on the effects of food advertising on behavioral and neural responses is required. We conducted a systematic review and meta-analysis of behavioral and neural responses to food advertising in recent experimental studies. Subgroup analysis examined whether the effects of food advertising on the outcomes of interest differed by participant age (children compared with adults), BMI group (people with normal weight compared with overweight/obesity), study design (within-subjects compared with between-subjects), and advertising media type (television commercials compared with advergames).

Methods

Literature search and screening

The systematic review protocol was registered with the International Prospective Register of Systematic Reviews Database (PROSPERO registration number: CRD42022311357). A search strategy was developed per the PRISMA guidelines in collaboration with an academic librarian. The Population, Intervention, Comparison, Outcome, Study design (PICOS) criteria used to identify the research question are shown in Supplemental Table 1. The search strategy was targeted to identify experiments that investigated the effects of food advertising on behavioral and neural outcomes. PubMed, Web of Science, and Scopus were searched for articles published in the past 7 y (from January 1, 2014 to November 1, 2021). This time range was selected to capture articles published after a previous meta-analysis on this topic [23]. Studies were restricted to English language articles only. Reviews that appeared within the search were manually checked for additional citations. Peer-reviewed studies were included; grey literature and self-published studies were excluded. The search terms aimed to identify experiments that evaluated behavioral and neural responses to food advertising and included: "food images," "food cues," "food advertising," "food commercials," "food brands," "food commercial," "commercial of food," "food ad," "food ads" AND "functional MRI," "functional magnetic resonance," "fMRI," "brain imaging," "reactivity," "food intake," "EEG," "MEG" AND "experiment," "study," "intervention" (Supplemental Table 2).

Studies eligible for inclusion were those that examined the impact of food commercials, food advertising, food brands, or food advergames on the outcomes of interest among children or adults. Prespecified outcomes of interest included food intake during or after the experimental condition (behavioral outcome) and brain activity (neural outcome) evaluated via fMRI, electroencephalography, or magnetoencephalography. Physiologic outcomes, including salivation and assessment of appetite hormones, were documented during the literature screening for possible consideration in meta-analysis if the retrieved studies evaluated those outcomes. Studies that employed an experimental design were included, including pilot studies. Exclusion criteria were observational studies and animal

studies, and studies with special populations (eating disorders, weight loss surgery, and neuropsychiatry disorders), studies that did not use a food advertising condition, studies that did not include a nonfood control group, articles with fMRI results not coming from whole brain analysis (i.e., regions of interest), experiments looking at food cues outside of the context of food advertising, secondary analysis of published data used in another publication included in the meta-analysis, and reviews (Supplemental Table 3). Results were independently screened in 2 stages (title/abstract and full-text) by 2 reviewers (PA and JMC). A 3rd reviewer (DN) was involved to resolve any disagreements.

Data extraction

Information extracted included the following: *1)* article identification (e.g., title, digital object identifier, authors, and date); *2)* characteristics of the study (e.g., methodology, exclusion criteria, and type of advertising media); *3)* types of outcomes and measurement (behavioral, physiologic, and neural whole brain or regions of interest analysis); and *4)* results (e.g., important findings, group differences, study highlights, and effect sizes). Extractions were done by a single reviewer (KL) with input from a second reviewer (PA) where necessary.

Owing to missing values from the original publications, to request clarification of reported values, or to request data according to the BMI group of participants, authors were contacted via email to obtain the data necessary for the meta-analysis. An email for data requests was sent to the corresponding author of 19 out of the 20 articles selected (one article included all required information). Over a 12-wk period, up to 3 email contacts were attempted to clarify and/or obtain required data. Required data were obtained for 8 studies (representing 8 publications). Some authors were unresponsive or unable to provide the required data. In some cases, the information provided in the article enabled us to proceed with the meta-analysis, but with raw (unadjusted) means instead of means adjusted for the covariates mentioned in the publications [22, 29–31], or by estimating a correlation coefficient based on the available studies [32–34]. Ultimately, only one of the selected articles could not be included in the meta-analysis due to an inability to obtain or estimate the required information [11].

Statistical analyses

Random-effects meta-analysis using standardized mean differences.

Behavioral, physiologic, and neural outcomes using non-fMRI techniques were planned to be considered in separate analyses. Meta-analysis of fMRI studies used a specific analytical technique described below (seed-based d Mapping [SDM]). Because all outcome measures were continuous in nature, standardized mean differences (SMDs) were calculated between food and nonfood advertising conditions [35]. SMDs were estimated using Hedges' g based on the mean, SD (calculated from the SEM, when necessary), and sample size reported for each condition [35]. The SE of each SMD was also calculated. For studies using a within-subjects design, the correlation between both conditions was taken into account in the calculation of the SE of the SMD to get a more reliable estimate that considers the nonindependence of the observations [35]. When it was not possible to obtain the correlation coefficient from the authors (3 publications reporting on 4 studies [32–34]), it was estimated to be $r = 0.8$, which represents the average correlation coefficient observed in the retrieved studies in which this information was provided.

The overall meta-analysis aimed to examine the pooled differences in outcomes between food and nonfood advertising conditions, regardless of participant or study characteristics. For one study that compared the effect of advertisements promoting high- compared with low-calorie foods [31], we considered the high-calorie advertisement condition alone to maximize the consistency with the other studies (which used advertisements for highly palatable foods). When individual articles exclusively reported study results for 2 separate groups [i.e., participants with normal weight compared with with overweight/obesity [32], and participants with or without self-reported episodes of binge eating [34]], the groups were merged using Cochrane's formula for combining summary statistics [36]. In one study [34], the SD of food intake in the nonfood advertising condition was unavailable, but could be calculated based on other statistics provided in the text (*P* value and sample size).

When a study included an experimental manipulation that was irrelevant to the aim of the current meta-analysis (e.g., use of a protective message [30]), only data from the control condition were used in the analysis (e.g., no protective message). Although all studies offered highly palatable, energy-dense food options to participants during or after exposure to advertising (e.g., chocolate, candy, or pizza), 4 studies also offered lower energy-density food options (e.g., fruits and vegetables [31, 33, 37, 38]), and one study also offered a nonwater beverage (i.e., orange juice [33]). Therefore, the food intake outcome in the present analysis refers to overall intake, regardless of the energy-density of food or beverage consumed (except water).

After calculating SMDs and SEs, the inverse-variance method was used in the Review Manager version 5.4 software to ensure that the weight given to each study in the calculation of the meta-analytic overall effect size was based on the level of precision of each study. A random-effects model was used to calculate this overall effect because differences in sample and methodology across studies were expected. Cochran's Q statistic, as well as the I2 statistic and its 95% CI [39] were computed to assess heterogeneity across studies. Interpretation of effect sizes [40] and heterogeneity [41] was based on convention. Publication bias was visually assessed with a funnel plot and quantitatively assessed with Egger's regression test.

Prespecified subgroup analyses by age (children compared with adults) and BMI group (participants with normal weight compared with participants with overweight/obesity) were conducted to further examine the effects of food advertising on the outcomes of interest according to these variables. Owing to the availability of a sufficient number of studies with use of different study designs and different advertisement media types, 2 exploratory subgroup analyses were added to the analysis plan: study design (within-subjects compared with between-subjects) and advertising media type (television commercials compared with advergames).

Meta-analysis of fMRI studies

A meta-analysis using SDM (https://www.sdmproject.com/) was conducted. SDM is a weighted, voxel-based meta-analytic method that combines neuroimaging studies using cluster peak coordinates and effect sizes to find common patterns of activation in the brain in response to a specific stimulation [42]. Voxels

are small rectangular cuboids that are composed of scanner 3D images of the brain, similar to what pixels are to digital photographs [42]. SDM is advantageous over other meta-analytical methods because it allows weighting results from individual studies according to their sample size [42]. Furthermore, SDM is able to consider both positive (hyperactivation) and negative (hypoactivation) differences in neural activity in one analysis, enabling an overall assessment of the neural response of the exposures under study [42]. On the contrary, other methods require a separate meta-analysis to be performed for positive peaks and negative peaks [42]. The SDM method has been used in previous neuroimaging meta-analyses of food cue reactivity specifically comparing food cue reactivity between individuals with normal weight and obesity [43].

Using SDM, individual results for changes in neural activity between the experimental (food advertising) and control (nonfood advertising) conditions of each study were aggregated to identify common areas in the brain that were activated in response to food advertising. The standard coordinates of the brain regions with activation and their corresponding statistics (t or z-values) were extracted. For one study that compared the effect of advertisements promoting unhealthy compared with healthier fast foods [16], only the unhealthy condition was considered to maximize the consistency with the other studies included.

Sdm-Psi 6.22 was used to conduct the SDM meta-analysis [44]. The main analysis used a voxel-wise $P < 0.001$ threshold of significance with a cluster extent >10 voxels. Subsequently, a threshold-free-cluster enhancement (TFCE) multiple comparison correction of $P < 0.05$ was applied (*threshold-free* in TFCE refers to the cluster-forming threshold [45]) to obtain peak coordinates and clusters breakdowns [46]. The thresholded SDM maps were overlaid onto the Colin brain template in Montreal Neurological Institute space and visualized with Mango 4.0 (freely available from http://ric.uthscsa.edu/mango/mango.html). Publication bias was visually assessed with a funnel plot comparing the residual effect sizes of fMRI study results against their SEs. A test for excess statistical significance was conducted to quantitatively examine the potential for publication bias. The SDM method conducts multiple imputations and meta-regression to estimate nonstatistically significant unreported effects [47, 48].

Sensitivity analyses

Sensitivity analyses were conducted to determine whether the pattern of results would change when either using a range of correlation coefficients for the 4 within-subjects studies that did not provide this value between experimental conditions (food and nonfood advertising) or removing these studies (from 3 publications [32–34]). Given that no estimates were made for data in the neural activity meta-analysis, sensitivity analyses were not performed for this outcome.

Bias assessment

The potential for bias among the included studies was evaluated using relevant criteria from the Cochrane Risk of Bias tool (RoB 2) [49] and the risk of bias assessment tools for nonrandomized studies [50], which was applied in a previous systematic review on a related topic [51]. We considered 5 domains: allocation to the exposure condition and confounding variables (random allocation and consideration of BMI, age, sex, or hunger), blinding of participants (whether participants knew the aims of the study), measurement of outcome (quality/objectiveness of the outcome measures), incomplete outcome data (whether there were missing data or participants excluded from analyses), and selective outcome reporting (detailed preregistration of study). Three authors (PA, KL, and DN) independently assessed the methodological quality of each included study and any discrepancies were discussed to reach consensus.

Results

Articles retrieved for the systematic review

A total of 219 articles were retrieved during the initial screening (Figure 1). On removal of duplicates, 151 articles were assessed for eligibility. Following title/abstract screening, 46 articles were eligible for full-text screening, of which 20 articles were deemed eligible for meta-analysis. However, one of these articles was removed because of the inability to obtain the required information from the corresponding author, yielding a total of 19 articles available for meta-analyses. From the 19 articles, 13 publications reported on food intake [20, 22, 29–34, 37, 38, 52–54] and 6 studies reported on neural activity (using only fMRI) [15, 16, 21, 55–57] (Table 1). No eligible article retrieved from the search strategy reported on appetite hormones, and no article used electroencephalography or magnetoencephalography to measure neural activity. Only one article reported on salivation in addition to food intake and so was included in the review, but ultimately a meta-analysis of physiologic outcomes could not be performed [20]. Thus, on identification of eligible articles, a meta-analysis was conducted for food intake, and the neural activity meta-analysis consisted of only fMRI studies.

Summary of results: food intake and salivation

A total of 1303 participants were included in the 13 articles that reported on food intake. Of the 13 articles, 10 publications reported on experiments with children [22, 29–33, 37, 38, 52, 53] and 3 were experiments with adults (aged ≥18 y) only [20, 34, 54]. Six articles used a within-subjects design [20, 32–34, 37, 38] and 7 a between-subjects design [22, 29–31, 52–54]. A random assignment to the condition was carried out in all between-subjects design experiments. All articles based on within-subjects designs reported the randomization or counterbalancing of the condition order. Seven articles measured the impact of television commercials on consumption [20, 32–34, 38, 53, 54], 4 examined exposure to advergames [22, 29–31], one used both television commercials and advergames [37], and one studied exposure to YouTube vlogs from social media influencers presenting food products [52]. Most studies measured participants' consumption of snack items that were provided as part of the experiment (e.g., chocolate and jelly candy); except for 2 studies that used meal items (e.g., pizza) [20, 32] and one that used both [38]. Two publications each conducted 2 separate studies within an article [30, 32], totaling 15 individual studies that were used to calculate the overall effect size for the meta-analysis. Six studies assessed food intake while the participants were being exposed to the condition [22, 30, 32, 53], whereas the remaining 9 evaluated food intake after the exposure [20, 29, 31, 33, 34, 37, 38, 52, 54]. Values for

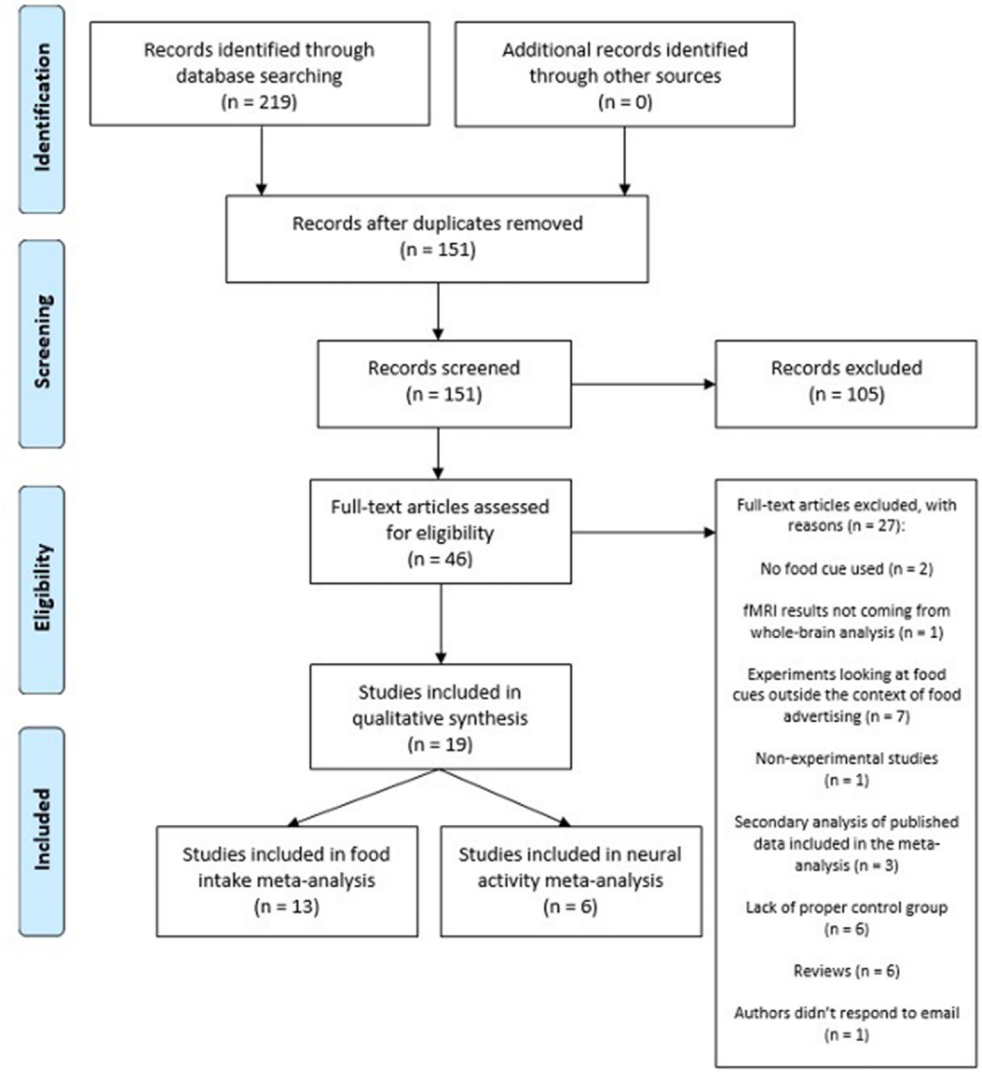


FIGURE 1. PRISMA flowchart of the study selection process.

means, SDs, sample sizes, and correlation coefficients for the food intake outcome from the individual studies can be found in Supplemental Table 4.

Studies with children were mostly in a school setting, except for 3 conducted in a laboratory [32, 38, 53] and one during school camp [37]. All 3 studies with adults were conducted in a lab setting. Most studies described assessing hunger with a visual analog scale (VAS), although there was variation in the characteristics of the VAS [20, 22, 29–31, 38, 52]. Five other studies evaluated hunger with a Likert scale [33, 34, 37, 53, 54]. Four studies offered a meal before the experiment, with variability between the studies in the type of meal [32, 34, 37, 53]. Regarding the publication that reported on salivation [20], it was found that in participants with normal weight there was no significant difference in salivation during the food commercial exposure compared with baseline salivation. However, their salivation in response to in vivo food exposure increased compared with baseline, regardless of previous commercial exposure [20]. Moreover, in participants with overweight/obesity, salivation increased after in vivo food exposure only when previously exposed to food commercials [20].

Meta-analysis of food intake: Overall pooled outcome

With all 15 possible comparisons included, a SMD of 0.23 ($P < 0.00001$; 95% CI: 0.14, 0.32) was observed, which constituted a small, but significant effect size that indicates participants consumed more food in the food advertising condition compared with those under control condition (Figure 2). There was moderate to substantial heterogeneity across studies (Cochran's Q = 29.40; $P = 0.009$; $I^2 = 52.4\%$; 95% CI: 14.2%, 73.6%). A visual inspection of the funnel plot showed evidence of asymmetry (Figure 3), which was consistent with the result from Egger's test ($P = 0.022$ for the intercept).

Subgroup analysis by age group (children compared with adults)

A subgroup analysis that was based on the age of participants (children compared with adults) was conducted. The subgroup difference was not significant ($\chi^2 = 1.20$; $P = 0.27$; $I^2 = 16.7\%$). For the 12 experiments that included children only, there was evidence of a significant small effect of food advertisement

TABLE 1
Characteristics of the studies included in the food intake and neural activity meta-analyses

Study	Outcome	Final number of participants	Sex of participants	Age (y)	Design	Relevant advertising medium	Outcome (food intake)	Outcome (brain)
Anderson et al. 2015a[1] [32]	Behavioral	27	27M	Mean and SD not provided Range: 9–14	Within-subjects	Nonfood vs. food TV commercials	Ad-libitum pizza intake (kcal)	N/A
Anderson et al. 2015b [32]	Behavioral	23	23F	Mean and SD not provided Range: 9–14	Within-subjects	Nonfood vs. food TV commercials	Ad-libitum pizza intake (kcal)	N/A
Boyland et al. 2017 [20]	Behavioral	55	55F	Mean (± SD): 32.4 ± 9.8 Range: 20–62	Within-subjects	Nonfood vs. food TV commercials	Ad-libitum pizza intake (kcal)	N/A
Bruce et al. 2014 [21]	fMRI	17	10M, 7F	Mean (± SD): 11.8 ± 1.4 Range: 10–14	Within-subjects	Nonfood vs. food logos	N/A	Difference in brain activity when seeing logos
Bruce et al. 2016 [55]	fMRI	23	11M, 12F	Mean: 10.5 (SD not provided) Range: 8–14	Within-subjects	Nonfood vs. food TV commercials	N/A	Difference in brain activity when seeing commercials
Coates et al. 2019 [52]	Behavioral	151	71M, 80F	Mean (± SD): 10.3 ± 0.6 Range: 9–11	Between-subjects	YouTube vlogs from social media influencers presenting nonfood and food products	Ad-libitum marketed snack intake (kcal)	N/A
Egbert et al. 2020 [34]	Behavioral	38	38F	Mean (± SD): 18.8 ± 1.0 Range: 18–22	Within-subjects	Nonfood vs. food TV commercials	Ad-libitum candy intake (kcal)	N/A
Emond et al. 2016 [53]	Behavioral	60	33M, 27F	Mean (± SD): 4.1 ± 0.9 Range: 2–5	Between-subjects	Nonfood vs. food TV commercials	Advertised snack intake (kcal)	N/A
Folkvord et al. 2014 [22]	Behavioral	261	131M, 130F	Mean (± SD): 7.7 ± 0.7 Range: 7–10	Between-subjects	Nonfood vs. food advergame	Ad-libitum jelly candy and milk chocolate candy shells intake (kcal)	N/A
Folkvord et al. 2015 [29]	Behavioral	92	42M, 50F	Mean (± SD): 8.4 ± 1.1 Range not provided	Between-subjects	Nonfood vs. food advergame	Ad-libitum jelly candy and milk chocolate candy shells intake (kcal)	N/A
Folkvord et al. 2016 [31]	Behavioral	218	112M, 106F	Mean (± SD): 11.1 ± 0.8 Range not provided	Between-subjects	Nonfood vs. energy-dense food advergame	Ad-libitum snack intake (kcal)	N/A
Folkvord et al. 2017a [30]	Behavioral	211	107M, 104F	Mean (± SD): 9.0 ± 1.2 Range: 6–11	Between-subjects	Nonfood vs. food advergame	Ad-libitum jelly candy and milk chocolate candy shells intake (kcal)	N/A
Folkvord et al. 2017b [30]	Behavioral	351	165M, 186F	Mean (± SD): 8.9 ± 1.7 Range: 6–12	Between-subjects	Nonfood vs. food advergame	Ad-libitum jelly candy and milk chocolate candy shells intake (kcal)	N/A
Gearhardt et al. 2014 [56]	fMRI	30	13M, 17F	Mean (± SD): 15.2 ± 1.1 Range: 14–17	Within-subjects	Nonfood vs. food TV commercials	N/A	Difference in brain activity when seeing commercials
Gearhardt et al. 2020 [16]	fMRI	171	82M, 89F	Mean (± SD): 14.3 ± 1.0 Range: 13–16	Within-subjects	Nonfood vs. unhealthy food TV commercials	N/A	Difference in brain activity

(*continued on next page*)

TABLE 1 (*continued*)

Study	Outcome	Final number of participants	Sex of participants	Age (y)	Design	Relevant advertising medium	Outcome (food intake)	Outcome (brain)
								when seeing commercials
Kearney et al. 2021 [33]	Behavioral	101	40M, 61F	Mean (± SD): 9.9 ± 0.5 Range: 8–10	Within-subjects	Nonfood vs. food TV commercials	Ad-libitum snack foods and beverages intake (kcal)	N/A
Kidd and Loxton 2018 [54]	Behavioral	98[2]	35M, 63F	Mean (± SD): 25.5 ± 8.4 Range: 18–53	Between-subjects	Nonfood vs. food TV commercials	Ad-libitum milk chocolate candy shells intake (g)	N/A
Masterson et al. 2019a [38]	Behavioral	41	19M, 22F	Mean (± SD): 7.9 ± 0.7 Range: 7–9	Within-subjects	Nonfood vs. food TV commercials	Ad-libitum intake of high- and low-energy dense foods (kcal)	N/A
Masterson et al. 2019b [15]	fMRI	25	12M, 13F	Mean (± SD): 8.6 ± 1.1 Range: 7–10	Within-subjects	Nonfood vs. food brand images	N/A	Difference in brain activity when seeing brand images
Norman et al. 2018 [37]	Behavioral	154	77M, 77F	Mean (± SD): 9.3 ± 1.6 Range: 7–12	Within-subjects	Nonfood vs. food advergame and TV commercials	Ad-libitum snack intake (kJ)	N/A
Rapuano et al. 2016 [57]	fMRI	37	17M, 20F	Mean (± SD): 14.4 ± 1.3 Range: 12–16	Within-subjects	Nonfood vs. food TV commercials	N/A	Difference in brain activity when seeing commercials

Because some studies included experimental conditions that were not relevant to the aim of the current meta-analysis (e.g., use of a protective message), the final number of participants indicated in this Table sometimes amounts to more than the sum of participants from the food and nonfood advertising conditions indicated in the forest plots. For 2 studies that compared intake of the specific snack that was marketed in the food advertisement to intake of an alternative snack [52, 53], only data from the marketed snack condition was considered.

[1] Letters "a" and "b" indicate either a single publication that reported on results from two different studies or two distinct publications in the same year with the same first author surname.

[2] One of the 98 participants was identified as an outlier and excluded from the analysis, but the sex of this participant was not specified.

exposure on food intake (SMD: 0.25; $P < 0.0001$; 95% CI: 0.14, 0.37; $I^2 = 60.4\%$; 95% CI: 25.6%, 79.0%). From the remaining 3 studies involving adult participants, food advertising exposure also had a small, but significant effect on food intake (SMD: 0.16; 95% CI: 0.03, 0.28; $P = 0.01$; $I^2 = 0$, 95% CI: 0, 95.0%) (Supplemental Figure 1).

Subgroup analysis by BMI group (participants with normal weight compared with those with overweight or obesity)

The subgroup analysis according to BMI group revealed no significant difference ($\chi^2 = 0.31$, $P = 0.58$, $I^2 = 0$). For the 6 experiments from which effect sizes were able to be calculated from participants with normal weight, there was no evidence of an effect of food advertisement exposure on food intake (SMD: 0.11; 95% CI: −0.06, 0.28; $P = 0.19$; $I^2 = 56.4\%$; 95% CI: 0, 82.4%). However, for the same 6 experiments from which effect sizes were able to be obtained from participants with overweight and obesity, food advertising exposure had a significant small effect size on food intake (SMD: 0.17; 95% CI: 0.04, 0.30, $P = 0.01$; $I^2 = 0$; 95% CI: 0, 26.2%) (Supplemental Figure 2).

Subgroup analysis by study design (within-subjects compared with between-subjects design)

The subgroup analysis showed a significant subgroup difference, with the effect size being larger for between-subjects than within-subjects designs ($\chi^2 = 8.64$, $P = 0.003$, $I^2 = 88.4\%$, 95% CI: 55.9%, 97.0%). For the 7 experiments that used a within-subjects design, food advertising exposure showed a significant small effect toward food intake (SMD: 0.14; 95% CI: 0.08, 0.20; $P < 0.00001$; $I^2 = 0$, 95% CI: 0, 84.3%). For the 8 experiments that were between-subjects, advertising exposure had a significant medium effect size on food intake with participants consuming a greater amount of food in the food advertisement condition than the control condition (SMD: 0.43; 95% CI: 0.25–0.61; $P < 0.00001$; $I^2 = 43.3\%$; 95% CI: 0, 74.9%) (Supplemental Figure 3).

Subgroup analysis by advertising media type (television commercials compared with advergames)

Out of the 15 studies on advertising and food intake, 2 were omitted because one used a YouTuber video blog [52], and we only had data for the condition that combined television commercials and advergames for the other study [37]. The subgroup analysis revealed a borderline significant difference ($\chi^2 = 3.80$; $P = 0.051$; $I^2 = 73.7\%$; 95% CI: 0, 94.1%) between exposure conditions (television commercials or advergames). For the 8 experiments that used television commercials as exposure, food advertising exposure showed a significant small effect toward food intake (SMD: 0.16; 95% CI: 0.06, 0.25; $P = 0.001$; $I^2 = 30.5\%$; 95% CI: 0, 69.0%). From the remaining 5 experiments that had advergames as the exposure, advertising exposure had a significant medium effect on food intake with participants consuming a greater amount of food in the food advertisement condition than those under control

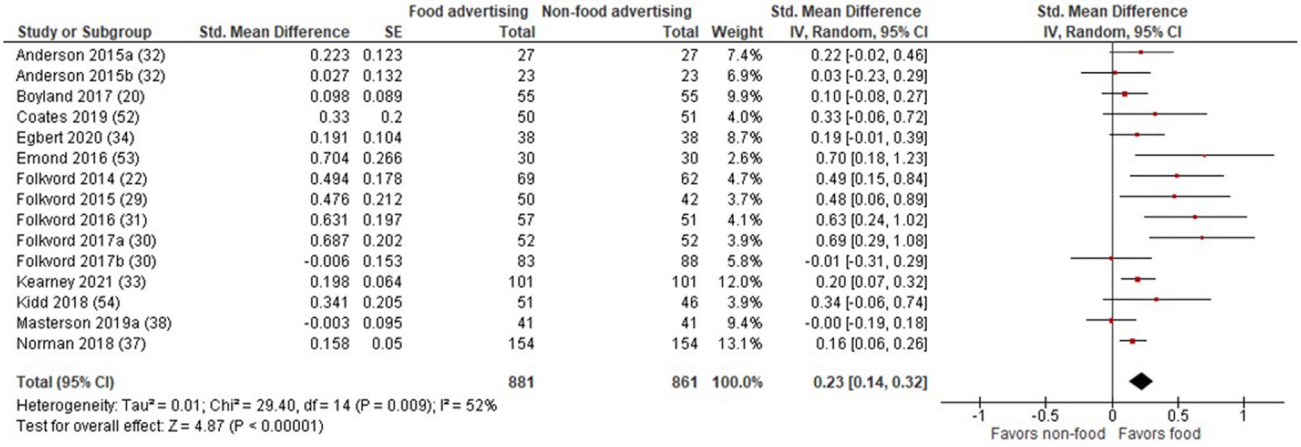


FIGURE 2. Forest plot of the effect sizes for the food intake meta-analysis.

condition (SMD: 0.44; 95% CI: 0.17, 0.71; $P = 0.001$; $I^2 = 63.1\%$; 95% CI: 2.5%, 86.0%) (Supplemental Figure 4).

Results from sensitivity analyses

Detailed results of the sensitivity analyses for overall food intake as well as subgroup analyses by age, BMI, study design, and media type are presented in Supplemental Tables 5 and 6. Three of the studies involved children participants [32, 33]. All 4 studies employed a within-subjects design and were conducted using television commercials. The pattern of results for the overall food intake meta-analysis and for the tests of subgroup differences did not change when using a range of correlation coefficients (Supplemental Table 5). When excluding the 4 studies, the subgroup comparison results remained unchanged, although certain SMDs became attenuated among some individual subgroups (Supplemental Table 6).

Meta-analysis of neural activity

A total of 303 participants were included in the 6 studies. All 6 studies that reported on neural activity outcomes were performed on children (<18 y) and used a within-subjects design. Four articles measured the impact of television advertising (commercials) on neural activity, one examined exposure to food brands, and the remaining studied exposure to food logos. The standard coordinates of the brain regions with activation and their corresponding statistics can be found in Supplemental Table 7.

We found that there was one significant cluster, which was the right middle occipital gyrus (peak Montreal Neurological Institute coordinates: 30, −86, 12; *z*-value: 6.301, size: 226 voxels; TFCE corrected $P < 0.001$) (Figure 4). Given the small number of studies, further subgroup analyses could not be performed. A visual inspection of the funnel plot showed evidence of asymmetry, suggesting presence of publication bias (Supplemental Figure 5). This was consistent with the result for the test of excess statistical significance ($P < 0.001$). Additional clusters were identified before correcting for multiple comparisons (voxel-wise $P < 0.001$). These results are presented in Supplemental Table S8 and Supplemental Figure 6.

Bias assessment

The bias assessment of the included studies is summarized in Supplemental Table 9. No study included in both meta-analyses ranked with low RoB for all 5 criteria. Most studies had a low

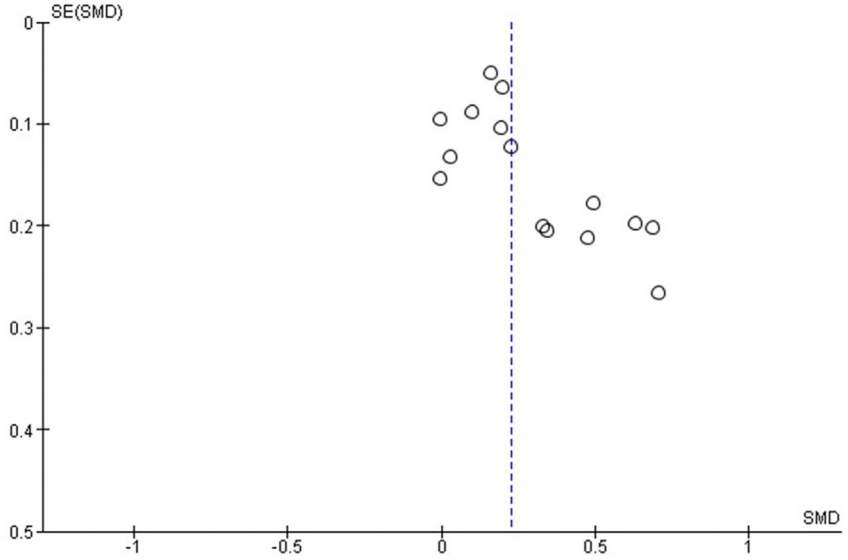


FIGURE 3. Funnel plot for the food intake meta-analysis.

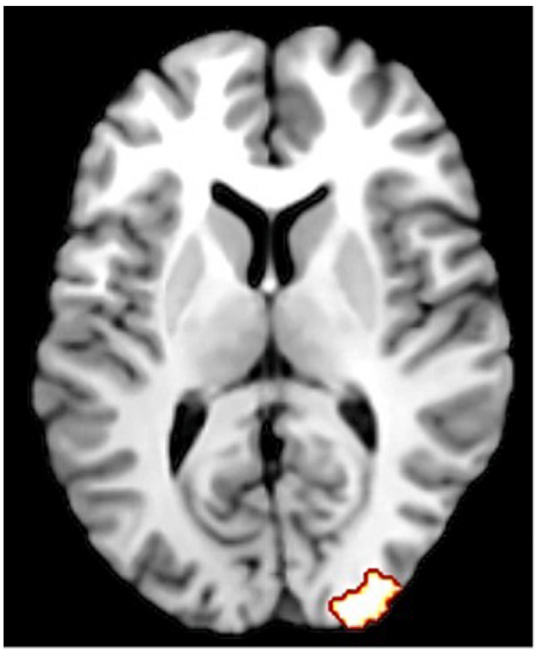


FIGURE 4. Illustration of the significant cluster with greater activation in the food compared with the nonfood advertising condition (right middle occipital gyrus)

RoB through allocation to exposure condition and confounding variables (86%), whereas the remaining had an unclear RoB. Most studies had an unclear RoB related to the blinding of participants (62%), whereas the rest had a low RoB. All studies had a low RoB for measurement of the outcome because all studies used an objective measurement of the outcome (weighed food intake or brain activity through fMRI). When evaluating incomplete outcome data, most studies had low RoB (66%), whereas only one study had a high RoB, and the remaining studies had an unclear RoB. Finally, regarding selective outcome reporting, all studies had an unclear RoB, mostly related to the lack of information about preregistration, or to the fact that the preregistration was retrospective and lacking in detail.

Discussion

The present investigation was a systematic review and meta-analysis of experimental studies that examined the effects of exposure to food advertising on food intake and neural activity (15 studies for food intake and 6 studies for fMRI data). The food intake meta-analysis showed that the difference in food consumption was significant, with a small effect in the direction of increased intake after exposure to food advertising than to nonfood advertising. A previous meta-analysis that evaluated experimental studies conducted before 2014 reported a similar effect of increased food intake after exposure to food commercials (SMD: 0.37; $P = 0.01$; 95% CI: 0.09, 0.65; $I^2 = 98\%$) [23]. However, the significant effect was only observed among children and not adults. To our knowledge, the present meta-analysis is the first to demonstrate an overall effect of increased food intake after food advertising exposure in adults, which could be because of differences in study design/protocol or types of food advertising media since the time of the previous meta-analysis.

It is widely accepted that food advertising elicits a modification of behavior in children toward increased consumption of hyperpalatable, ultraprocessed food [28, 58]. As a result, exposure to food advertising is considered a risk factor for childhood obesity because children may be unable to understand the selling or persuasive intent of advertising [28]. Further, food advertisements that target children are uniquely designed to influence children's food brand awareness and create brand attachment by tailoring the brand attributes to exploit hedonic and emotional responses in children [59]. It should be noted that most studies from our meta-analysis did not look at postexperimental food consumption or future unhealthy weight gain. Thus, it is unclear whether participants could have reduced their intake after the experiment to compensate for the overeating. However, a study included in our meta-analysis by Norman et al. [37], measured intake on the subsequent lunch after the postexperimental consumption of a snack. They found that all children ate more after exposure to food advertising compared with nonfood advertising, suggesting that unhealthy food advertising exposure contributes to increased energy intake even after some time has elapsed since the exposure [37]. Thus, more research on the effects of food advertising exposure on later (nonacute) food intake is warranted.

Previous research has produced inconsistent results on whether food advertising confers the same risks in adults [23, 60]. Given their more advanced cognitive development, adults are considered more critical viewers of advertising and hence may be less susceptible to their influence [28]. Nevertheless, a small, but overall significant, effect toward increased food consumption among adults exposed to the food advertising condition was detected in this meta-analysis despite the fact that the 3 individual studies included reported null results [20, 34, 54]. It is important to note that in these studies (and in fact most studies included in our meta-analyses), a description of a sample size calculation was not provided. Therefore, it is unclear whether the individual studies we retrieved were sufficiently powered for their analysis. Although we estimated correlation coefficients for a few studies, results from our sensitivity analyses support our conclusions as results were highly consistent across a range of correlation coefficients or on exclusion of these studies (though exclusion impacts statistical power).

The subgroup analysis by BMI group revealed a significant effect among participants with overweight and obesity toward increased food intake after exposure to food advertising (although there was no significant difference in the effect size between BMI groups). Indeed, exposure to food commercials has been associated with an increased risk of developing obesity [61]. The present results align with previous suggestions that individuals with already existing obesity may become sensitized to the visual and auditory cues found in food advertising and become more reactive to it, thus increasing their food intake compared with their lean counterparts [62]. Furthermore, in the present meta-analysis, there was a positive effect toward increased food consumption in the food advertising condition irrespective of the study design, although the effect was larger in the between-subjects design. This finding also differs from Boyland and colleagues' meta-analysis, which reported larger effect sizes for studies that used within-subjects rather than between-subjects designs [23]. One of the main strengths of the between-subjects design is the minimization of learning and

transferring across conditions, whereas strengths of the within-subjects design include controlling for interindividual variation and minimization of random noise [63]. Although either design appears suitable for experimental studies of responses to food-related stimuli, given the opposite findings from the present study compared with those of Boyland and colleagues, additional research is warranted to better understand factors related to the study design that may impact experimental results (e.g., length of washout period in a within-subjects design or participant matching in a between-subjects design).

Both advertising media types influenced intake in the food advertising condition, however, the effect tended to be larger in advergames and the subgroup comparison was borderline significant. Advergames may involve greater engagement by the viewer because of the need for interaction, and so this may have contributed to the larger effect of this media format. This result is in contrast to a previous meta-analysis that reported that the effect tended to be larger for television commercials compared with advergames [23]. Given that time spent online has increased since the time of the previously reviewed studies, it is possible that the formats and engagements with digital forms of food advertising have also evolved [64]. Therefore, additional investigation into the effects of different media formats is warranted.

Finally, there was substantial variability in the methods used in the individual studies included in the present meta-analysis. For instance, hunger was controlled in different ways across studies. While some assessed baseline hunger with either a VAS [20, 22, 29–32, 38, 52] or a Likert scale [33, 34, 37, 53, 54], there were differences in how hunger was controlled for, with some using the scale response as a covariate, whereas others offered food in the presence of hunger before conducting the experiment. There was also variability in the types of foods that were described as offered before the experiment, with studies offering either a granola bar [34], bananas, cheese and crackers [53], a breakfast, including cereals, fruits and toast [37], or an unspecified breakfast [32]. Similarly, there was heterogeneity in the length of time between consumption of the study snack/meal and the time of the experiment, as well as the time of day in which the experiment was conducted (morning compared with afternoon). These considerations are important, as the type of meal [65], time of day [66], and interindividual variability in appetite [67] can impact hunger and satiety in varying ways. Other relevant factors known to affect appetite, such as sleep and caffeine consumption [68, 69], were not discussed nor assessed in the retrieved studies. Guidelines for experimental designs and tools to assess the quality of these considerations in experimental studies evaluating outcomes related to ingestive behavior currently do not exist, highlighting the need for the development of such tools.

Results from the SDM meta-analysis on brain activity found that the food commercials condition produced larger brain responses in the right middle occipital gyrus than the nonfood conditions (after correcting for multiple comparisons). The middle occipital gyrus is part of the occipital gyri complex, which is mainly responsible for object recognition [70]. Previous research has shown an involvement of the middle occipital gyrus in response to food cues. For instance, in a study evaluating brain responses to unhealthy and healthy foods, children had a stronger reaction in the middle occipital gyrus in response to unhealthy foods than with healthy foods [71]. In line with our results, 3 separate meta-analyses have also found the middle occipital gyrus to be involved in responses to food cues [25, 72, 73]. Van der Laan et al. [72] performed a meta-analysis of 17 studies to determine the concurrence in the brain regions activated in response to viewing pictures of food. The middle occipital gyrus was in one of the 16 significant clusters that showed a stronger response to pictures of food than nonfood pictures [72]. Furthermore, they assessed the modulation effect of the energy content of the foods by performing a meta-analysis contrasting high-calorie foods compared with low-calorie foods, and found that middle occipital gyrus was one of the 5 clusters where neural activation was higher during viewing of high- compared with low-calorie foods [72]. More recently, Yeung [25] conducted a meta-analysis of 7 studies that recruited only children and adolescents, to evaluate differences in brain activation in response to food commercials compared with nonfood commercials. Food commercials were reported to cause larger brain responses than nonfood commercials in different areas of the brain, including the cuneus and the middle occipital gyrus [25]. Lastly, Yang et al. [73] performed a meta-analysis of 59 studies that evaluated brain responses to high-calorie food cues (including food images and commercials) in individuals with normal weight or obesity. They found that viewing high-calorie food cues consistently activated many areas of the brain, including the middle occipital gyrus [73].

From the available evidence, the activation of the middle occipital gyrus appears to be greater in response to food compared with nonfood visual stimuli, and in particularly, high-calorie compared with low-calorie food stimuli. Indeed, the occipital cortex has been shown to consistently respond to drug-related cues (e.g., alcohol, cocaine, marijuana, tobacco), compared with nondrug cues [74]. It is plausible that high-calorie food images modulate neural activity in the visual areas because of their highly rewarding properties, similar to how drug-related cues do. Furthermore, before correcting our analysis for multiple comparisons, additional brain areas were identified as being significantly activated when watching food advertisements. Some of these areas have also been implicated in previous food cue reactivity research. In a meta-analysis by van der Laan et al. [72], the fusiform gyrus was found to be one of the main brain regions activated in response to viewing food pictures. The left superior occipital gyrus was identified in our uncorrected analysis, and indeed the whole occipital lobe plays a role in visual processing of food cues [72]. Finally, the posterior cingulate gyrus was identified, which has been associated with heightened activation in response to high-calorie food cues among those with higher BMI [75]. Thus, although our most robust result pertained to the middle occipital gyrus (which survived TFCE correction), these other areas warrant additional investigation.

To our knowledge, the present systematic review and meta-analysis is the first to evaluate the effect of food advertising exposure on both behavioral and neural outcomes as part of the same investigation. This work updates the evidence obtained from meta-analysis by Boyland et al. [23] and performs a further important subgroup analysis by BMI group. In addition, a separate meta-analysis evaluated neural responses to food advertising exposures using the activation likelihood estimation method [25] rather than SDM as conducted presently. A limitation of some versions of the former method is that a single study can drive the findings of the pooled analysis because the total number of peaks are counted regardless of whether they are part of the same or

different studies [42]. The SDM method resolves this limitation by separating the peaks of each study and weighting the individual studies by sample size. Further, SDM accounts for both hypo- and hyperactivation in response to a given stimulation by recreating positive and negative maps in the same image [42]. We identified evidence of publication bias in both the behavioral and neural outcomes of interest. Therefore, the present findings should be interpreted cautiously and additional experimental studies are needed.

The present investigation is not without limitations. The number of studies that evaluated food intake is smaller than the previous work, likely because the year range in the literature search was shorter than previous work. This was because the objective of this present investigation was to update the evidence according to the most recent studies on the topic, which is warranted given that the platforms for food advertising have evolved since previous work (e.g., advertising on YouTube and social media platforms in addition to television) [52]. Moreover, society has increased time spent online [76] and so engagement with digital forms of food advertising has likely changed since previous work. Although we performed a bias assessment using a selection of RoB and related criteria, we could not rate the overall risk of bias or grade the strength of evidence because the present tools available for these activities are not aligned with laboratory-based experimental studies [23]. The high level of control and objective outcome assessment appears to indicate low RoB in the present group of studies. Nevertheless, experimental researchers have an opportunity to develop quality assessment tools that are tailored to these study designs.

The limited number of fMRI studies and overall sample size in the SDM meta-analysis precluded subgroup analyses for the neural response outcome. We nevertheless proceeded with the SDM approach given its advantages over other methods [42], but we may have been underpowered to detect additional brain regions that are activated during exposure to food-related marketing [77]. Hence, we included results before correcting for multiple comparisons to facilitate broader dissemination of potentially relevant areas. Indeed, several brain regions were identified in some individual fMRI studies included in this review [16, 56, 57], and so consideration of additional areas is warranted. Lastly, studies retrieved from the literature search were limited to fMRI only, where participants are restricted in a supine position and unnaturalistic setting. Uptake of other neuroimaging tools that allow for less restricted conditions may be advantageous for future work. Moreover, there are several additional methodological factors and participants' characteristics not able to be considered that could be related to the outcomes of interest (e.g., use of popular children's media characters in food advertising [78], participant sex [79], individual eating behaviors [80], race/ethnicity [81], and genetic risk of obesity [11]). Further research is also warranted to improve the scientific understanding of neural responses to differing types of food stimuli. In particular, future work could benefit from considering individual food preferences when evaluating neural responses to food stimuli. It is well known that personal food preferences exist because of individual and cultural factors [82], but most experimental protocols have lacked consideration of participants' food preferences.

In conclusion, this systematic review and meta-analysis provides evidence that acute exposure to food advertisements increases food intake among both children and adults, although the effect tends to be larger among children. Among children, the right middle occipital gyrus had higher activation in response to food advertising exposure, suggesting a role of this brain area in the processing of food-related marketing stimuli. These findings should be considered when developing and reviewing policies related to the food environment and food marketing. Further research should investigate the role of specific food cues tailored to the individual preferences of the participants, which may produce brain responses unique to the individual. Finally, further investigation of modern forms of food advertising prevalent on social media is warranted, such as those involving influencers and sponsored ads.

Author disclosures

PA is a recipient of a Consejo Nacional de Ciencia y Tecnología (CONACYT) graduate scholarship. KL received support from the Fonds de recherche du Québec–Nature et technologies (FRQNT) in the form of a postdoctoral scholarship. DEN is a member of ASN's Statistical Review Board. The other author reports no conflicts of interest.

Acknowledgments

We thank Filip Morys for his valuable assistance with proofreading and feedback. PA and DN: designed research; PA, KL, and JMC: conducted the systematic review and data extraction; PA and KL: analyzed data; PA and KL: drafted the manuscript; JMC and DN: critically revised the manuscript; DN: has primary responsibility for final content. All authors read and approved the final manuscript.

Funding

Supported by the Natural Sciences and Engineering Research Council of Canada (NSERC) and the Institut sur la nutrition et les aliments fonctionnels (INAF).

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.advnut.2022.12.003.

References

[1] M. Ng, T. Fleming, M. Robinson, B. Thomson, N. Graetz, C. Margono, et al., Global, regional, and national prevalence of overweight and obesity in children and adults during 1980-2013: a systematic analysis for the Global Burden of Disease Study 2013, Lancet 384 (2014) 766–781.
[2] L.M. Jaacks, S. Vandevijvere, A. Pan, C.J. McGowan, C. Wallace, F. Imamura, et al., The obesity transition: stages of the global epidemic, Lancet Diabetes Endocrinol 7 (2019) 231–240. Epub 20190128.
[3] GBD 2017 Risk Factor Collaborators, Global, regional, and National comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017, Lancet 392 (2018) 1923–1994.
[4] S. Donofry, C.M Stillman, K.I. Erickson, A review of the relationship between eating behavior, obesity and functional brain network organization, Soc Cogn Affect Neurosci 15 (10) (2020) 1157–1181.

[5] M. Brandkvist, J.H. Bjørngaard, R.A. Ødegård, B.O. Åsvold, E.R. Sund, G.Å. Vie, Quantifying the impact of genes on body mass index during the obesity epidemic: longitudinal findings from the HUNT Study, BMJ 366 (2019) l4067.
[6] R. Guthold, G.A. Stevens, L.M. Riley, F.C. Bull, Worldwide trends in insufficient physical activity from 2001 to 2016: a pooled analysis of 358 population-based surveys with 1.9 million participants, Lancet Glob Health 6 (2018) e1077–e1086.
[7] R. Dalle Molle, H. Fatemi, A. Dagher, R.D. Levitan, P.P. Silveira, L. Dubé, Gene and environment interaction: is the differential susceptibility hypothesis relevant for obesity? Neurosci Biobehav Rev 73 (2017) 326–339.
[8] A.E. Locke, B. Kahali, S.I. Berndt, A.E. Justice, T.H. Pers, F.R. Day, et al., Genetic studies of body mass index yield new insights for obesity biology, Nature 518 (2015) 197–206.
[9] U. Vainik, T.E. Baker, M. Dadar, Y. Zeighami, A. Michaud, Y. Zhang, et al., Neurobehavioral correlates of obesity are largely heritable, Proc Natl Acad Sci USA 115 (2018) 9312–9317.
[10] R.E. Marioni, J. Yang, D. Dykiert, R. Mõttus, A. Campbell, G. Davies, et al., Assessing the genetic overlap between BMI and cognitive function, Mol Psychiatry 21 (2016) 1477–1482.
[11] K.M. Rapuano, A.L. Zieselman, W.M. Kelley, J.D. Sargent, T.F. Heatherton, D. Gilbert-Diamond, Genetic risk for obesity predicts nucleus accumbens size and responsivity to real-world food cues, Proc Natl Acad Sci U S A 114 (2017) 160–165.
[12] S. Sanchez-Roige, P. Fontanillas, S.L. Elson, , 23andMe Research Team, A. Pandit, E.M. Schmidt, et al., Genome-wide association study of delay discounting in 23,217 adult research participants of European ancestry, Nat Neurosci 21 (2018) 16–18.
[13] U. Vainik, A. Dagher, L. Dubé, L.K. Fellows, Neurobehavioural correlates of body mass index and eating behaviours in adults: a systematic review, Neurosci Biobehav Rev 37 (2013) 279–299.
[14] I. Garcia-Garcia, S. Neseliler, F. Morys, M. Dadar, Y.H.C. Yau, S.G. Scala, et al., Relationship between impulsivity, uncontrolled eating and body mass index: a hierarchical model, Int J Obes (Lond). 46 (2022) 129–136.
[15] T.D. Masterson, W.M. Stein, E. Beidler, M. Bermudez, L.K. English, K.L. Keller, Brain response to food brands correlates with increased intake from branded meals in children: an fMRI study, Brain Imaging Behav 13 (2019) 1035–1048.
[16] A.N. Gearhardt, S. Yokum, J.L. Harris, L.H. Epstein, J.C. Lumeng, Neural response to fast food commercials in adolescents predicts intake, Am J Clin Nutr 111 (2020) 493–502.
[17] M. Grosshans, C. Vollmert, S. Vollstädt-Klein, H. Tost, S. Leber, P. Bach, et al., Association of leptin with food cue-induced activation in human reward pathways, Arch Gen Psychiatry 69 (2012) 529–537.
[18] S. O'Rahilly, I.S. Farooqi, Human obesity: a heritable neurobehavioral disorder that is highly sensitive to environmental conditions, Diabetes 57 (2008) 2905–2910.
[19] A. Jansen, R.C. Havermans, C. Nederkoorn, in: V. Preedy, R. Watson, C. Martin (Eds.), Handbook of Behavior, Food and Nutrition, Springer, New York (NY), 2011, pp. 1431–1443.
[20] E.J. Boyland, R.H. Burgon, C.A. Hardman, Reactivity to television food commercials in overweight and lean adults: physiological, cognitive and behavioural responses, Physiol Behav 177 (2017) 182–188.
[21] A.S. Bruce, J.M. Bruce, W.R. Black, R.J. Lepping, J.M. Henry, J.B.C. Cherry, et al., Branding and a child's brain: an fMRI study of neural responses to logos, Soc Cogn Affect Neurosci 9 (2014) 118–122.
[22] F. Folkvord, D.J. Anschütz, C. Nederkoorn, H. Westerik, M. Buijzen, Impulsivity, "advergames," and food intake, Pediatrics 133 (2014) 1007–1012.
[23] E.J. Boyland, S. Nolan, B. Kelly, C. Tudur-Smith, A. Jones, J.C. Halford, et al., Advertising as a cue to consume: a systematic review and meta-analysis of the effects of acute exposure to unhealthy food and nonalcoholic beverage advertising on intake in children and adults, Am J Clin Nutr 103 (2016) 519–533.
[24] S. Yokum, A.N. Gearhardt, J.L. Harris, K.D. Brownell, E. Stice, Individual differences in striatum activity to food commercials predict weight gain in adolescents, Obesity (Silver Spring) 22 (2014) 2544–2551.
[25] A.W.K. Yeung, Brain responses to watching food commercials compared with nonfood commercials: a meta-analysis on neuroimaging studies, Public Health Nutr 24 (2021) 2153–2160.
[26] S.E. Kanoski, K.N. Boutelle, Food cue reactivity: neurobiological and behavioral underpinnings, Rev Endocr Metab Disord 23 (2022) 683–696.
[27] F. Folkvord, Riet J. van't, The persuasive effect of advergames promoting unhealthy foods among children: a meta-analysis, Appetite 129 (2018) 245–251.
[28] M. Story, S. French, Food advertising and marketing directed at children and adolescents in the US, Int J Behav Nutr Phys Act 1 (2004) 3.
[29] F. Folkvord, D.J. Anschütz, R.W. Wiers, M. Buijzen, The role of attentional bias in the effect of food advertising on actual food intake among children, Appetite 84 (2015) 251–258.
[30] F. Folkvord, F. Lupiáñez-Villanueva, C. Codagnone, F. Bogliacino, G. Veltri, G. Gaskell, Does a 'protective' message reduce the impact of an advergame promoting unhealthy foods to children? An experimental study in Spain and the Netherlands, Appetite 112 (2017) 117–123.
[31] F. Folkvord, D.J. Anschütz, M. Buijzen, The association between BMI development among young children and (un)healthy food choices in response to food advertisements: a longitudinal study, Int J Behav Nutr Phys Act 13 (2016) 16.
[32] G.H. Anderson, S. Khodabandeh, B. Patel, B.L. Luhovyy, N. Bellissimo, R.C. Mollard, Mealtime exposure to food advertisements while watching television increases food intake in overweight and obese girls but has a paradoxical effect in boys, Appl Physiol Nutr Metab 40 (2015) 162–167.
[33] J. Kearney, R. Fitzgerald, G. Burnside, S. Higham, N. Flannigan, J.C.G. Halford, et al., Television advertisements for high-sugar foods and beverages: effect on children's snack food intake, Br J Nutr 125 (2021) 591–597.
[34] A.H. Egbert, L. Nicholson, A. Sroka, R. Silton, A.M. Bohnert, Binge eating, but not dietary restraint, moderates the association between unhealthy food marketing exposure and sugary food consumption, Eat Behav 38 (2020), 101401.
[35] H. Cooper, L.V. Hedges, J.C. Valentine (Eds.), The Handbook of Research Synthesis and Meta-Analysis, Second ed., Russell Sage Foundation, New York, 2009.
[36] J.P.T. Higgins, T. Li, J.J. Deeks, Cochrane Handbook for Systematic Reviews of Interventions, Chapter 6: Choosing effect measures and computing estimates of effect (2022). Available from: https://training.cochrane.org/handbook/current/chapter-06.
[37] J. Norman, B. Kelly, A.-T. McMahon, E. Boyland, L.A. Baur, K. Chapman, et al., Sustained impact of energy-dense TV and online food advertising on children's dietary intake: a within-subject, randomised, crossover, counter-balanced trial, Int J Behav Nutr Phys Act 15 (2018) 37.
[38] T.D. Masterson, M.A. Bermudez, M. Austen, E. Lundquist, A.L. Pearce, A.S. Bruce, et al., Food commercials do not affect energy intake in a laboratory meal but do alter brain responses to visual food cues in children, Appetite 132 (2019) 154–165.
[39] M. Borenstein, L.V. Hedges, J.P.T. Higgins, H.R. Rothstein, Introduction to meta-analysis, Chapter 16:Identifying and Quantifying Heterogeneity, John Wiley & Sons, Ltd, Chichester, 2009, pp. 107–125.
[40] J. Cohen, Statistical Power Analysis for the Behavioral Sciences, Second ed., Routledge, New York, 1988.
[41] J.J. Deeks, J.P.T. Higgins, D.G. Altman, Cochrane Handbook for Systematic Reviews of Interventions, Chapter 10: Analysing data and undertaking meta-analyses (2022). Available from: https://training.cochrane.org/handbook/current/chapter-10.
[42] J. Radua, D. Mataix-Cols, Meta-analytic methods for neuroimaging data explained, Biol Mood Anxiety Disord 2 (2012) 6.
[43] F. Morys, I. García-García, A. Dagher, Is obesity related to enhanced neural reactivity to visual food cues? A review and meta-analysis, Soc Cogn Affect Neurosci (2020) nsaa113.
[44] J. Radua, D. Mataix-Cols, Voxel-wise meta-analysis of grey matter changes in obsessive-compulsive disorder, Br J Psychiatry 195 (2009) 393–402.
[45] S.M. Smith, T.E. Nichols, Threshold-free cluster enhancement: addressing problems of smoothing, threshold dependence and localisation in cluster inference, NeuroImage 44 (2009) 83–98.
[46] A. Albajes-Eizagirre, A. Solanes, M.A. Fullana, J.P.A. Ioannidis, P. Fusar-Poli, C. Torrent, et al., Meta-analysis of voxel-based neuroimaging studies using seed-based d mapping with permutation of subject images (SDM-PSI), J Vis Exp 153 (2019).
[47] A. Albajes-Eizagirre, A. Solanes, J. Radua, Meta-analysis of non-statistically significant unreported effects, Stat Methods Med Res 28 (2019) 3741–3754.
[48] J. Radua, Package 'metansue', CRAN, 2020 [cited 2022]. Available from: https://cran.r-project.org/web/packages/metansue/metansue.pdf.

[49] J.A.C. Sterne, J. Savović, M.J. Page, R.G. Elbers, N.S. Blencowe, I. Boutron, et al., RoB 2: a revised tool for assessing risk of bias in randomised trials, BMJ 366 (2019) l4898.
[50] S.Y. Kim, J.E. Park, Y.J. Lee, H.J. Seo, S.S. Sheen, S. Hahn, et al., Testing a tool for assessing the risk of bias for nonrandomized studies showed moderate reliability and promising validity, J Clin Epidemiol 66 (2013) 408–414.
[51] G. Weydmann, F.B. Souzedo, P. Tavares, L. Corrêa, H. Heidrich, H. Holland, et al., Parsing the link between reinforcement sensitivity theory and eating behavior: a systematic review, Neurosci Biobehav Rev 134 (2022), 104525.
[52] A.E. Coates, C.A. Hardman, J.C.G. Halford, P. Christiansen, E.J. Boyland, The effect of influencer marketing of food and a "protective" advertising disclosure on children's food intake, Pediatr Obes 14 (2019), e12540.
[53] J.A. Emond, R.K. Lansigan, A. Ramanujam, D. Gilbert-Diamond, Randomized exposure to food advertisements and eating in the absence of hunger among preschoolers, Pediatrics 138 (2016), e20162361.
[54] C. Kidd, N.J. Loxton, Junk food advertising moderates the indirect effect of reward sensitivity and food consumption via the urge to eat, Physiol Behav 188 (2018) 276–282.
[55] A.S. Bruce, S.W. Pruitt, O.R. Ha, J.B.C. Cherry, T.R. Smith, J.M. Bruce, et al., The influence of televised food commercials on children's food choices: evidence from ventromedial prefrontal cortex activations, J Pediatr 177 (2016) 27–32.e1.
[56] A.N. Gearhardt, S. Yokum, E. Stice, J.L. Harris, K.D. Brownell, Relation of obesity to neural activation in response to food commercials, Soc Cogn Affect Neurosci 9 (2014) 932–938.
[57] K.M. Rapuano, J.F. Huckins, J.D. Sargent, T.F. Heatherton, W.M. Kelley, Individual differences in reward and somatosensory-motor brain regions correlate with adiposity in adolescents, Cereb Cortex 26 (2016) 2602–2611.
[58] B. Kelly, J.C. Halford, E.J. Boyland, K. Chapman, I. Bautista-Castaño, C. Berg, et al., Television food advertising to children: a global perspective, Am J Public Health 100 (2010) 1730–1736.
[59] B. Kelly, E. Boyland, L. King, A. Bauman, K. Chapman, C. Hughes, Children's exposure to television food advertising contributes to strong brand attachments, Int J Environ Res Public Health 16 (2019) 2358.
[60] S.D.H. Mills, L.M. Tanner, J. Adams, Systematic literature review of the effects of food and drink advertising on food and drink-related behaviour, attitudes and beliefs in adult populations, Obes Rev 14 (2013) 303–314.
[61] J. Norman, B. Kelly, E. Boyland, A.-T. McMahon, The impact of marketing and advertising on food behaviours: evaluating the evidence for a causal relationship, Curr Nutr Rep 5 (2016) 139–149.
[62] R. Belfort-DeAguiar, D. Seo, Food cues and obesity: overpowering hormones and energy balance regulation, Curr Obes Rep 7 (2018) 122–129.
[63] G. Charness, U. Gneezy, M.A. Kuhn, Experimental methods: between-subject and within-subject design, J Econ Behav Organ 81 (2012) 1–8.
[64] E. Boyland, D. Thivel, A. Mazur, S. Ring-Dimitriou, M.L. Frelut, D. Weghuber, et al., Digital food marketing to young people: a substantial public health challenge, Ann Nutr Metab 76 (2020) 6–9.
[65] H.J. Leidy, J.W. Apolzan, R.D. Mattes, W.W. Campbell, Food form and portion size affect postprandial appetite sensations and hormonal responses in healthy, nonobese, older adults, Obesity (Silver Spring) 18 (2010) 293–299.
[66] F.A.J.L. Scheer, C.J. Morris, S.A. Shea, The internal circadian clock increases hunger and appetite in the evening independent of food intake and other behaviors, Obesity (Silver Spring) 21 (2013) 421–423.
[67] J. Blundell, C. de Graaf, T. Hulshof, S. Jebb, B. Livingstone, A. Lluch, et al., Appetite control: methodological aspects of the evaluation of foods, Obes Rev 11 (2010) 251–270.
[68] S.M. Greer, A.N. Goldstein, M.P. Walker, The impact of sleep deprivation on food desire in the human brain, Nat Commun 4 (2013) 2259.
[69] M.M. Schubert, C. Irwin, R.F. Seay, H.E. Clarke, D. Allegro, B. Desbrow, Caffeine, coffee, and appetite control: a review, Int J Food Sci Nutr 68 (2017) 901–912.
[70] K. Grill-Spector, Z. Kourtzi, N. Kanwisher, The lateral occipital complex and its role in object recognition, Vision Res 41 (2001) 1409–1422.
[71] F. van Meer, L.N. van der Laan, L. Charbonnier, M.A. Viergever, R.A.H. Adan, P.A.M. Smeets, et al., Developmental differences in the brain response to unhealthy food cues: an fMRI study of children and adults, Am J Clin Nutr 104 (2016) 1515–1522.
[72] L.N. van der Laan, D.T.D. de Ridder, M.A. Viergever, P.A.M. Smeets, The first taste is always with the eyes: a meta-analysis on the neural correlates of processing visual food cues, NeuroImage 55 (2011) 296–303.
[73] Y. Yang, Q. Wu, F. Morys, Brain responses to high-calorie visual food cues in individuals with normal-weight or obesity: an activation likelihood estimation meta-analysis, Brain Sci (2021) 11.
[74] C.A. Hanlon, L.T. Dowdle, T. Naselaris, M. Canterberry, B.M. Cortese, Visual cortex activation to drug cues: a meta-analysis of functional neuroimaging papers in addiction and substance abuse literature, Drug Alcohol Depend 143 (2014) 206–212.
[75] S.D. Donofry, J.M. Jakicic, R.J. Rogers, J.C. Watt, K.A. Roecklein, K.I. Erickson, Comparison of food cue-evoked and resting-state functional connectivity in obesity, Psychosom Med 82 (2020) 261–271.
[76] Statista, Daily time spent on social networking by internet users worldwide from 2012 to 2022, Statista (2022). Available from: https://www.statista.com/statistics/433871/daily-social-media-usage-worldwide/.
[77] V.I. Müller, E.C. Cieslik, A.R. Laird, P.T. Fox, J. Radua, D. Mataix-Cols, et al., Ten simple rules for neuroimaging meta-analysis, Neurosci Biobehav Rev 84 (2018) 151–161.
[78] L.M. Powell, G. Szczypka, F.J. Chaloupka, Exposure to food advertising on television among US children, Arch Pediatr Adolesc Med 161 (2007) 553–560.
[79] J. Strahler, A. Hermann, N.M. Schmidt, R. Stark, J. Hennig, A.J. Munk, Food cue-elicited brain potentials change throughout menstrual cycle: modulation by eating styles, negative affect, and premenstrual complaints, Horm Behav 124 (2020), 104811.
[80] J.S. Coelho, A. Jansen, A. Roefs, C. Nederkoorn, Eating behavior in response to food-cue exposure: examining the cue-reactivity and counteractive-control models, Psychol Addict Behav 23 (2009) 131–139.
[81] F. Fleming-Milici, J.L. Harris, Television food advertising viewed by preschoolers, children and adolescents: contributors to differences in exposure for black and white youth in the United States, Pediatr Obes 13 (2018) 103–110.
[82] P.J. Chen, M. Antonelli, Conceptual models of food choice: influential factors related to foods, individual differences, and society, Foods 9 (2020) 1898.