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REVIEW

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Food and non-alcoholic beverage marketing in children and adults: A systematic review and activation likelihood estimation meta-analysis of functional magnetic resonance imaging studies

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Summary

Food marketing impacts the food behaviors of children and adults, but the underpinning neural mechanisms are poorly understood. This systematic review and meta-analysis pooled evidence from neuroimaging studies of exposure to food marketing stimuli (vs. control) on brain activations in children and adults to clarify regions associated with responding. Databases were searched for articles published to March 2022. Inclusion criteria included human functional magnetic resonance imaging (fMRI) studies employing a contrast between a food marketing stimulus and a non-food/non-exposure control, published in English in a peer-reviewed journal, reporting whole brain (not Region of Interest [ROI] only) co-ordinates. Eleven studies met inclusion criteria, of which eight were included in the quantitative synthesis (Activation Likelihood Estimation [ALE] meta-analysis). Food marketing exposures (vs. controls) produced greater activation in two clusters lying across the middle occipital gyrus, lingual gyrus, and cuneus (cluster 1), and the postcentral gyrus, precentral gyrus, and the inferior parietal lobule/supramarginal gyrus (cluster 2). Brain responses to food marketing are most consistently observed in areas relating to visual processing, attention, sensorimotor activity, and emotional processing. Subgroup analyses (e.g., adults vs. children) were not possible because of the paucity of data, and sensitivity analyses highlighted some instability in the clusters; therefore, conclusions remain tentative pending further research.

KEYWORDS
brain, fMRI, food marketing, meta-analysis

1 | INTRODUCTION

The extensive marketing of unhealthy foods and non-alcoholic beverages (hereafter referred to as food) is a critical characteristic of the current obesogenic food environment[1] and has been strongly implicated in rising levels of obesity globally, particularly in children.[2,3] Numerous systematic reviews and meta-analyses have demonstrated the significant detrimental impacts of unhealthy food marketing exposure on eating and health-related outcomes in children,[4–6] with some evidence that minority and socioeconomically disadvantaged groups are disproportionately exposed.[7,8] Some mandatory government policies have been shown to be effective in reducing children's exposure to food marketing, the persuasive power of that marketing, and purchasing of unhealthy foods by or on behalf of children.[9,10] However, many countries continue to rely on ineffective industry self-regulatory policies[9] despite the numerous best-practice recommendations including greater restrictions that have been issued by United Nations organizations and other authoritative bodies.[11]

Evidence of effects of food marketing on adult eating behaviors is less clear than the research conducted with youth. Acute effects of food marketing exposure on intake in adults have not been consistently observed in experimental settings,[12,13] though there are cross-sectional associations between unhealthy food advert exposure and diet-related outcomes in adults,[14] and parents' perceptions of food products are influenced by the presence of marketing features such as celebrity endorsements on packaging.[15] Evidence from parallel literatures on alcohol and smoking also suggests that marketing impacts adults' use of promoted products.[14,16] There is additional evidence to suggest that food marketing has broader sociocultural impacts, including influencing dietary norms, driving population-level shifts in food and drink category preferences, and affecting the cultural values underpinning food behaviors.[17]

One potential explanation for the lack of observed effects of food marketing on acute consumption in adults may be awareness that they are being observed, creating social desirability bias,[12] so some studies have sought to use alternative outcome measures, such as physiological effects, to try to overcome this limitation.[18] While the importance of physiological influences as a contextual factor influencing food behaviors has been noted in models of food marketing impacts, their specific role is yet to be characterized.[19] This reflects the need for research that helps elucidate the specific mechanism(s) through which food marketing exerts its effects[20] and that uses outcomes less susceptible to behavioral bias, such as neuroimaging. Neuroimaging studies can non-invasively identify priming effects of food marketing on subconscious, automatic physiological and psychological processes[21] that might not be captured by self-report measures and may also be used as a meaningful predictor of eating behavior.[22]

The evidence of food marketing's impact on food behaviors appears to be consistent with food cue reactivity theory, whereby exposure to visual food cues (e.g., images and videos) triggers cue-induced craving in both children and adults.[23,24] Craving has been shown to systematically and prospectively predict food-related outcomes with effect sizes similar to real food exposure and greater than those for olfactory cues.[24] The sight of food also elicits many other physiological, emotional, and cognitive responses.[25] Alongside salient food imagery, visual branding is also a key component of food marketing. Branding, such as logos, acts as a representation of a brand and is frequently presented to consumers on products, in media marketing and on signage, as well as being integrated into sports events and promotions,[26] as brands seek to develop and nurture emotional connections that will influence consumers' behavior.[27,28] Children as young as three years of age can recognize brand logos and associate them with products[29] and brand imagery has been shown to significantly impact children's taste preference and food choices.[30] Greater recognition of food brands has previously been found to be associated with higher body mass index in pre-school children.[31] Evidence of impacts from even brief or subliminal exposures to brand imagery is indicative of the power of food marketing exposure in the real world, which can often operate below conscious awareness,[20,32] particularly via digital media.[28]

Neural mechanisms are believed to play an important role in mediating eating behavior through regulation of food motivation and behavioral control.[33] The ability of food marketing to activate key neural systems, such as reward-related pathways, could be critical to their effectiveness.[21] Understanding the neural mechanisms underpinning food marketing influences could have implications for identifying particularly vulnerable populations (e.g., those with developmentally linked heightened reward sensitivity[34] or genetic susceptibility to real world food cues such as marketing[35,36]). It could also inform policy development to protect those groups, and have the potential to inform individual interventions (e.g., those that target the relevant neurobiological systems). Brain responses to both food[25] and food marketing cues[37] have been observed, but studies are small and heterogeneous in participants and methodology and therefore, evidence synthesis is warranted.

This study systematically reviewed and meta-analyzed neuroimaging studies of exposure to any form of commercial food marketing stimulus on brain activation in children and adults relative to a non-food or non-exposure control. The primary objective was to use these pooled analyses to clarify the brain regions associated with responding to food marketing exposure to improve understanding of the potential mechanisms through which such marketing exerts its effects.

2 | METHODS

This systematic review and Activation Likelihood Estimation (ALE) meta-analysis was pre-registered with PROSPERO (registration number: CRD42020190176, available from https://www.crd.york.ac.uk/prospero/display_record.php?RecordID=190176) and is reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines.[38]

2.1 | Search methods

The comprehensive search strategy (see supplement) was developed and executed by an experienced information specialist (MM). Searches were conducted in Scopus, MEDLINE, CINAHL, EMBASE, PsycINFO, CENTRAL (via The Cochrane Library), Business Source Complete, EconLit, Academic Search Complete, TRIP, Google, and Google Scholar (targeted searches for both Google sources), using the key concepts ('fMRI' terms combined with OR) AND ('food' terms combined with OR) AND ('marketing' terms combined with OR). Both thesaurus and free-text terms were combined. Databases were initially searched for articles published up to June 10, 2020, this was later updated to March 16, 2022. These searches were supplemented by (i) hand searching reference lists of retrieved systematic reviews, (ii) contact with topic experts, and (iii) forward and backward citation searching of included studies.

2.2 | Eligibility criteria

The criteria for inclusion were the following: a) human functional magnetic resonance imaging (fMRI) studies; b) published in English in a peer-reviewed journal; c) studies of healthy (systemic disease-free) child (0–18 years) and/or adult (18 y+) populations; d) employed a contrast between a commercial food marketing stimulus (as defined by the World Health Organization[11] e.g., TV commercial, brand logo, product placement image), and a control stimulus (e.g., non-food images such as stationery items or non-food marketing images such as an advertisement for a toy or car) or baseline activity; and e) coordinates were reported in the article or supplementary material in either Montreal Neurological Institute (MNI)[39] or Talairach space.[40] Studies that reported Region of Interest (ROI) results only (i.e., did not report results from whole-brain analyses) were excluded (e.g.[41]). This is because inclusion of ROI studies is understood to introduce bias into ALE meta-analyses.[42,43]

2.3 | Study selection and data extraction

Two reviewers from a pool of five (EB, CR, MM, MA, and TM) independently screened studies against the inclusion criteria; assessing titles and abstracts to identify potentially relevant studies then reviewing full texts. One reviewer (AC) extracted the relevant data, and these were cross-checked by a second reviewer (CR). The reviewers extracted the following information: study information (e.g., authors, year, study country, funding, and conflicts of interest); population (e.g., number of participants, age, gender, and body weight), study design (e.g., description of control and food marketing stimuli, contrast(s)), and outcome measures (e.g., XYZ coordinates, statistical corrections). For both study selection and data extraction, disagreement was resolved through consensus, and, if necessary, consulting a third reviewer.

2.4 | Quality assessment

No appropriate tool exists for assessing risk of bias in neuroimaging studies specifically, so quality assessment was undertaken using the Newcastle–Ottawa Scale (NOS) for experimental and non-randomized study designs.[44] The quality of included studies was assessed by one reviewer (EB) and independently checked for agreement by a second (MA). In addition, because of results from ALE analyses being susceptible to dominance from individual large cohort studies, "leave one out" sensitivity analyses were conducted (detailed below).

2.5 | Additional handling of data

Where necessary, authors of eligible studies were contacted by email to provide missing or additional data. Studies that reported coordinates in the Talairach space[45,46] were converted into MNI coordinates using GingerALE (Brainmap GingerALE version 2.3.6 Research Imaging Institute: http://brainmap.org).

In cases where a food marketing > non-food/control image or commercial was presented in addition to a food marketing > baseline activation, we used data from the former contrast only.[45,46] In cases where unhealthy food marketing > non-food marketing, and healthy food marketing > non-food marketing were presented, we used data from the former contrast only.[47] Where there were two publications from the same cohort,[48,49] only data from the article published first[49] was included in the meta-analysis.

2.6 | ALE meta-analysis

One primary ALE meta-analysis was conducted, followed by a series of "leave-one-out" sensitivity analyses in which each effect size was removed in turn, and the pooled effect was recalculated. These analyses were conducted to assess stability of results following exclusion of individual studies. To have been included in the primary meta-analysis, the article must have reported the results of a direct contrast between activity while viewing food marketing and activity while viewing non-food marketing, control images, or baseline activity (experimental condition minus control condition activation). Given that vulnerability to food marketing is thought to vary by age,[20] we intended to conduct subgroup analyses based on age of participants (adults vs. children including adolescents) but this was not possible because of an insufficient amount of data. We did not consider data from between groups contrasts (e.g., effects of food marketing in participants with overweight > healthy weight in the meta-analysis) as this was not our primary research question.

To determine consistency in reported regions of neural activation during exposure to food marketing stimuli, we performed coordinate-based (x,y,z) ALE meta-analyses (single dataset analysis). Analyses were performed in Brainmap GingerALE version 2.3.6. This approach assesses the spatial convergence of foci across studies using the reported coordinates of activation peaks from the individual studies (rather than peak height/signal intensity). GingerALE software algorithms use kernel techniques to assess spatial uncertainty around reported peaks.[50] Overlap between kernels is used to assess spatial location convergence that is greater than expected by chance.

We adhered to the ALE method (http://www.brainmap.org/ale/) of Eickhoff et al.[42,51] with the correction devised by Turkeltaub et al.[43] for minimizing within-experiment and within group effects. The correction uses a random effects model, and minimizes within-experiment effects (differences in number of reported foci that are in close proximity, which affects an individual experiment's contribution to an ALE map) and within-group effects (multiple contributions from the same sample, with the same contrast within the same article). Therefore, reported ALE coordinates represent the degree of concordance in activation across independent studies. This method assigns an ALE value to each voxel (1 mm^3 volumes of brain tissue): ALE values increase with the number of studies that report activated peaks at a voxel or in close proximity. Thus, consistency of voxel activation across studies can be assessed. The standardized procedures for performing ALE using GingerALE are described in the GingerALE user manual (Research Imaging Institute, 2013).

In GingerALE, Modeled Activation (MA) maps are produced for each experiment using the reported coordinates in MNI space. Each voxel within the MA map has an MA score that reflects the likelihood of that location having fMRI activation (based on a 3D normal probability distribution centered on entered coordinates). Individual MA maps are then combined to form an experimental ALE map, with ALE values for each voxel. True convergence of activation foci can then be distinguished from random clustering (noise) by testing against the null hypothesis (by creating a null distribution map) that there is a random spatial association between experiments.[52]

A *p* value is then calculated for each voxel based on the probability of attaining an ALE value that differs from that of the corresponding voxel on a null-distribution map, via random permutation. In our analysis, *p* values were generated by 1,000 permutations.

We adhered to the recommendations on methodology reported by Eickhoff et al.[52] As such, a cluster-level family-wise error (FWE) correction at $p < 0.05$ was employed to control for multiple comparisons. Our initial cluster-forming threshold was set at $p < 0.01$ (rather than $p < 0.001$) because of the relatively small number of studies in our analysis, meaning a less conservative initial cluster-forming threshold was more appropriate.

Multi-image Analysis GUI (MANGO http://ric.uthscsa.edu/mango) was used to overlay ALE maps onto an anatomical image using MNI coordinates.

3 | RESULTS

3.1 | Description of included studies

See Figure 1 for a PRISMA indicating the study selection steps. A total of 446 articles were returned from the database searches, removal of duplicates left 305 articles for screening. Of these, 212 were excluded following review of titles and abstracts. Full text reviews excluded 83 articles (see Figure 1 for reasons). An additional article was identified via supplementary searches, for a total of 11 eligible studies (all "good" quality; see Table 1) of which eight were included in the meta-analysis. Three studies could not be included in the quantitative synthesis because of the required data being unavailable (n = 2)[53,54] or because there was duplication of data from the same cohort with another included study (n = 1),[48] but are included in the narrative synthesis.

Of the 11 eligible studies, two featured adult participants,[54,55] five were conducted in adolescents (13 years and over),[47–49,56,57] and four with children (12 years and under).[45,46,53,58] The numbers of participants ranged from 17 to 171 with mean age ranging from 8.56 years to 37.09 years (Table 1). The stimuli types used were TV commercials in five studies,[45,47–49,57] brand logo images in four studies,[46,53,54,58] images from TV commercials in one study,[55] and multiple marketing images (e.g., of print ads, store displays, websites) in one study.[56]

3.2 | Primary ALE meta-analysis: food marketing exposure − control contrast

The food marketing exposure minus control contrast ALE meta-analysis pooled the data from eight eligible experiments (from eight articles, with a total of 371 participants and 73 reported foci).

The results (Table 2, Figure 2) revealed two significant clusters. The largest of these clusters has three peaks that lie in the middle occipital gyrus and the cuneus (Table 2). The cluster is situated across the cuneus (49.4%), middle occipital gyrus (45.9%), and the lingual gyrus (4.3%). The second cluster has three peaks that lie in the postcentral gyrus, and the cluster is situated across the postcentral gyrus (79.7%), precentral gyrus (16.9%), and inferior parietal lobule/supramarginal gyrus (3.4%).

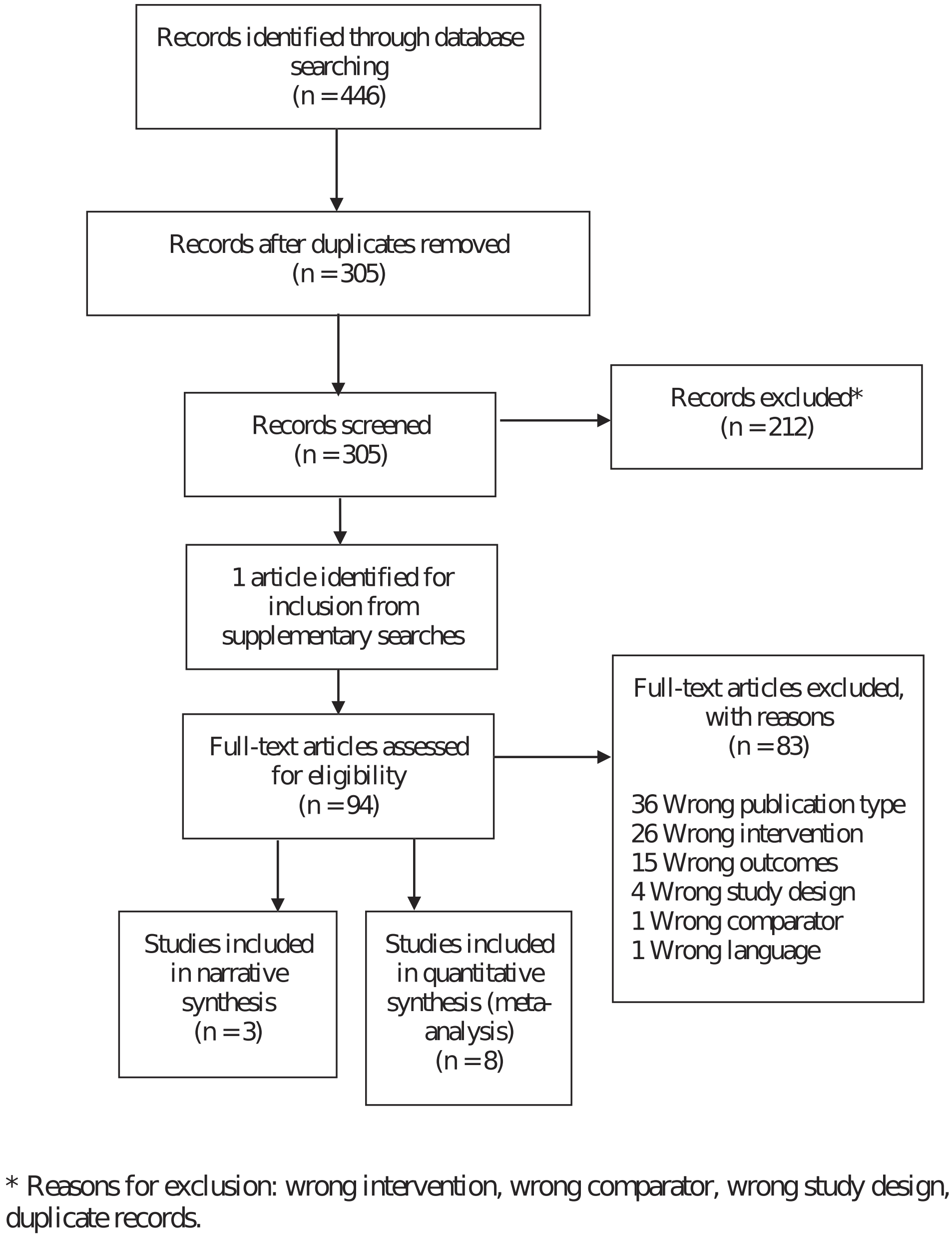
3.3 | Sensitivity analyses

The primary analysis was supplemented by an additional eight "leave one out" analyses. The result files from each of these analyses are presented in the supplementary materials; however they are briefly summarized here. The results from the main analysis (i.e., two clusters; 1 = right middle occipital gyrus/cuneus, 2 = postcentral gyrus) remained stable following the removal of Bruce et al.,[45] Courtney et al.,[55] or Gearhardt et al.[47] However, the cluster in the right middle occipital gyrus/cuneus was no longer present (although the postcentral gyrus cluster remained) following removal of either Bruce et al.[46] or Masterson et al.[58] In addition, the cluster in the postcentral gyrus was no longer present (although the middle right occipital gyrus/cuneus cluster remained) in cases where either Burger et al.[56] or Rapuano et al.[57] were removed. Following the removal of Gearhardt et al.,[49] neither cluster from the primary analysis remained significant, instead a cluster appeared that was centered in the left insular cortex.

3.4 | Narrative synthesis of studies not included in the meta-analysis

Two studies of three that could not be included in the meta-analysis-reported significant differences in activation between food marketing and control stimuli. Bruce et al.[53] reported on a group (healthy weight, obese) by stimulus type (food logo, nonfood logo) interaction. Here, the healthy weight children showed greater brain activation to food (vs. nonfood) logos in the middle and inferior frontal gyrus, the superior temporal gyrus, the parahippocampal gyrus, and the insula as well as greater bilateral activation in Brodmann's area 10 extending to the inferior frontal gyrus. The children with obesity did not show any significantly greater brain activation in any area relative to the children with healthy weight. Yokum et al.[48] report the same baseline data as Gearhardt et al.[49] whereby adolescents showed greater activation in the orbitofrontal cortex, anterior cingulate cortex, postcentral gyrus, and occipital gyrus in response to food commercials compared with non-food commercials. Fehse et al.[54] compared the contrast of

FIGURE 1 Study selection.



popular (defined as representing popular conventional food brands) > organic brands (alternative brands of organic origin) in adults, reporting activation in the ventromedial prefrontal cortex, whereas the contrast of organic > popular brands found activations in the left dorsolateral prefrontal cortex. However, the contrast of either food stimulus with the control exposures (colorful rectangles) was not reported.

4 | DISCUSSION

The current study explored brain activations in response to commercial food marketing exposures relative to a control stimulus using ALE meta-analysis. This is the first meta-analysis to include all forms of food marketing exposure stimuli (logos, static commercial images, TV commercials, and multiple marketing images) and both child and adult participants. Results show that food marketing exposures, compared with controls, produced greater activation in two clusters that lie across: the middle occipital gyrus, lingual gyrus, and cuneus (cluster 1), and the postcentral gyrus, precentral gyrus, and the inferior parietal lobule/supramarginal gyrus (cluster 2). This illustrates that the totality of the data so far suggests that the most consistently observed brain responses to food marketing exposure involve visual processing (cuneus, middle occipital gyrus, and lingual gyrus), somatosensory processing (post-central gyrus), and interpretation of sensory stimuli and perception of emotions (supramarginal gyrus).

Visual systems are often implicated in food marketing research.[59] Heightened brain responses to food marketing in the visual system, relative to control stimuli, are understood to reflect a heightened representation of food marketing at a pre-conscious level that may

TABLE 1 Description of included studies.

Publication	NOS score and rating	N (male)	Age in years, mean ± SD	Control stimulus	Food marketing stimulus	Contrast	Statistical threshold and correction for multiple comparisons
Included in ALE meta-analysis							
Bruce et al. (2014)[46]	7 – good	17 (10)	11.8 ± 1.4	Images of non-food brand logos	Images of food brand logos	Food brand logos > Non-food logos	Cluster level $p < 0.01$ corrected with FWE
Bruce et al. (2016)[45]	7 – good	23 (11)	10.5 (SD not reported)	Non-food television commercials	Food television commercials	Food commercials > Non-food commercials	Cluster level $p < 0.05$ corrected by imposing a $p < 0.005$ statistical threshold and a minimum cluster extent of 48 voxels
Burger & Stice (2014)[56]	7 – good	25 (13)	15.2 ± 0.8	Non-food/beverage marketing images	Coca-Cola marketing images	Coca-Cola marketing images > Non-food marketing images	Cluster level $p < 0.05$ corrected with FWE
Courtney et al. (2018)[55]	7 – good	43 (19)	19.83 ± 0.49	Images of non-food commercials	Images of fast food commercials	Food commercials > Non-food commercials	Cluster level $p < 0.001$ (false positives controlled for using AFNI 3dClustSim with the spatial autocorrelation function)
Gearhardt et al. (2014)[49]	7 – good	30 (13)	15.20 ± 1.06	Non-food television commercials	Food television commercials	Food commercials > Non-food commercials	Cluster level $p < 0.05$ corrected with Monte Carlo simulations
Gearhardt et al. (2020)[47]	8 – good	171 (72)	14.18 ± 1.03	Non-food television commercials	Unhealthy fast-food television commercials	Unhealthy fast-food > Non-food commercials	Cluster level $p < 0.05$ corrected using AFNI 3dClustSim with the spatial autocorrelation function
Masterson et al. (2019)[58]	7 – good	25 (12)	8.56 ± 1.12	Images of non-food brands	Images of food brands	Food brand images > Non-food brand images	Cluster level $p < 0.05$ corrected with Monte Carlo simulations
Rapuano et al. (2016)[57]	7 – good	37 (17)	14.4 ± 1.3	Non-food television commercials	Food television commercials	Food commercials > Non-food commercials	Cluster level $p < 0.005$ corrected with Monte Carlo simulations
Included in narrative synthesis only				Results as reported in abstract			
Bruce et al. (2013)[53]	7 – good	20 (9)	11.85 ± 1.23	Compared with the healthy-weight children, children with obesity showed significantly less brain activation to food logo images in the bilateral middle/inferior prefrontal cortex, an area involved in cognitive control.			
Fehse et al. (2017)[54]	7 – good	23 (14)	37.09 ± 7.6	The results show higher activations in medial prefrontal cortex for popular brand logos, as expected with respect to the existing literature on decision-making and self-control. For organic brands, we found relatively higher activations in dorsolateral parts of the prefrontal cortex.			
Yokum et al. (2014)[48]	7 – good	30 (13)	15.2 ± 1.1	Activation in the striatum, but not OFC, in response to television food commercials relative to non-food commercials and in response to food commercials relative to the television show was positively associated with change in BMI over a 1-year follow-up.			

Abbreviations: AFNI, Analysis of Functional NeuroImages; ALE, Activation Likelihood Estimation; BMI, body mass index; FWE, family wise error, NOS, Newcastle–Ottawa Scale; OFC, orbitofrontal cortex; SD, standard deviation.

TABLE 2 Locations (MNI) of significant clusters from the contrast food marketing exposure minus the control condition.

		Peak voxel coordinates					No of contributing experiments	
Cluster	Brain region	x	y	z	Cluster size (mm^3)	ALE value	N	%
Primary analysis								
1	Middle Occipital Gyrus R	32	−78	10	2040	0.0161	3 [Bruce et al., 2014[46];	37.5
	Middle Occipital Gyrus R	30	−88	14		0.0115	Gearhardt et al, 2014[49];	
	Cuneus	24	−94	12		0.0114	Gearhardt et al, 2020[47]]	
2	Postcentral gyrus R	60	−12	28	1,656	0.0212	4 [Burger et al., 2014[46];	50.0
	Postcentral gyrus R	56	−20	36		0.0198	Gearhardt et al, 2014[49];	
	Postcentral gyrus R	52	−24	36		0.0145	Gearhardt et al, 2020[47];	
							Rapuano et al, 2016[57]]	

Total number of experiments for primary analysis = 8, Cluster 1 lies 49.4% in cuneus, 45.9% in mid occipital gyrus and 4.3% lingual gyrus, Cluster 2 79.7% in post central gyrus, 16.9% precentral gyrus, 3.4% inferior parietal lobule (supramarginal gyrus).
Abbreviations: ALE, Activation Likelihood Estimation; MNI, Montreal Neurological Institute.

influence decision-making on food choices.[60] In addition, greater activity in visual areas may be indicative of greater attention allocation to food-branded images relative to control images. For example, the cuneus is understood to have a role in attention.[61] Previous research has also shown that attentional bias is a key moderator of the impact of food advertising exposure on children's food intake.[59] As such, the results from the current analysis are salient and concerning from a public health perspective, if they indicate that greater attention is commanded by branded-food images given that the majority of foods that are marketed are unhealthy.[62,63] The increased activity to food marketing in brain areas involved in visual processing observed in the current analysis may also reflect greater salience of brands and branded foods. This is of particular interest given that branding and brand building are key elements of food marketing strategies,[64] including in the contemporary digital era,[65] and visual cues are known to be potent triggers for approach motivation and consumption behaviors in both children and adults.[24] Indeed, brain activation to branded food cues has been shown to be associated with food intake in children.[58] Further, previous research has observed that reduced activity in brain regions associated with visual salience (precuneus and superior parietal lobe) can lead to healthier food intake in adults.[47]

The second cluster in the current analysis identified somatosensory areas (postcentral gyrus), motor areas (precentral gyrus), and areas involved in interpretation of sensory stimuli and perception of emotions (supramarginal gyrus). The somatosensory-postcentral gyrus has been observed to have a role in taste perception, as well as activation relating to food cues,[49,66] and motor activation in response to viewing food marketing may reflect neural circuitry engaged in expected consumption of viewed foods.[49] Taken together, these regions comprise sensorimotor activation relating to the approach of foods. Previous research has demonstrated associations in adults, but not children, between approach bias and greater consumption of snack food[67] as well as greater responsivity to television advertising for soft drinks.[68]

It is noteworthy that food marketing increased activation in the supramarginal gyrus, as this activation not only reflects interpreting sensory stimuli (i.e., foods) but is also involved in emotional processing. This suggests that branded foods produce an emotional response, which likely reflects how food marketing works — by conditioning an emotional attachment to brands.[28,69] Effective marketers draw on the power of emotion to drive impulsive behavior (quick choices) in consumers and loyalty to particular brands.[70] Food marketing rarely presents rational, information-based content, intending to persuade consumers at a conscious level, rather it infers that there are elaborate emotional benefits to consumption (e.g., feeling good).[71] The emotional attachment, combined with the increased attention and salience, as well as a sensorimotor approach response may undermine an individuals' ability to control their eating behavior, particularly when confronted with branded foods.[72]

Given the relatively modest number of included studies, additional fMRI studies using relevant food marketing contrasts would significantly help our understanding of these observed neurological phenomena and their potential relation to the well-documented behavioral effects of advertising.[4] Studies that explore whether ethnicity or socioeconomic position influences responding would be particularly useful[7] as would those that would facilitate comparisons of adult and child populations, given children's and adolescents' vulnerability to food marketing is thought to be driven by immature cognitive development, limited self-regulatory competence, and hypersensitivity to reward and appetitive cues.[71] Further research is also needed to fully elucidate the extent to which these observed effects are specific to food marketing, as some studies have found similar activations in visual areas for non-food stimuli in comparison to control that suggest this may be more of a generalized marketing effect.[58] Given the rapid growth in digital food marketing in recent years,[73,74] it would also be beneficial for studies to explore whether the brain regions responsive to digital marketing are consistent with those for other stimuli types (such as TV commercials and brand logos).

4.1 | Strengths and limitations

One of the strengths of the current paper was the transparent and clearly defined inclusion and exclusion criteria. This approach has

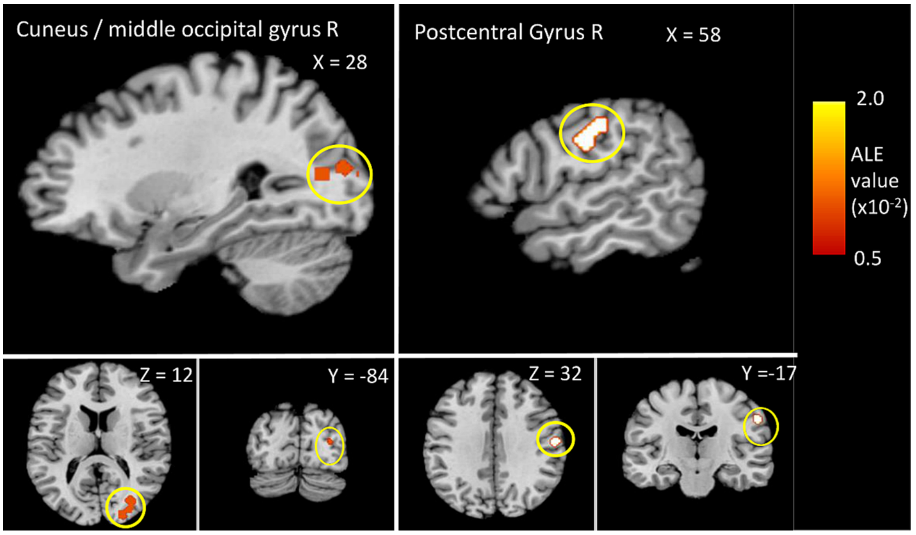


FIGURE 2 Localization of significant ALE clusters from the food marketing exposure – control contrast (main analysis). GingerALE output overlaid onto a standard template (Colin27_T1_seg_MNI.nii) in Montreal Neurological Institute (MNI) space.

afforded an unbiased assessment of brain regions that are activated when viewing food marketing images relative to controls based on the totality of the directly relevant evidence. Given that the literature shows several small and heterogeneous studies, the current quantitative synthesis is valuable to provide clarity of the overall picture. Better understanding of neural responses to food marketing may help to develop better interventions for overconsumption resulting from such marketing exposure.[75] These findings can also help to better direct ROI analyses for future studies in food marketing, which currently often rely on the food image literature to determine ROI.

However, as our systematic searches have revealed, the research in this area is still in its infancy, and there are a limited number of studies that contribute to our meta-analysis (albeit all deemed to be of "good" overall quality). This meant that the important planned subgroup analysis to compare effects in adult versus child participants, or other potentially relevant analyses (e.g., comparing results by stimulus type) were not possible in the present study. Given that there are known neurobiological differences between children and adults that are likely to affect response to food marketing exposure[20,34] and indeed behavioral differences in responding have been demonstrated,[12] it is a limitation of the present analysis that we are not meaningfully able to disentangle findings by age of participants. Nevertheless, our findings can be considered a launching point for consideration of the neural mechanisms that may be affected by food marketing (i.e., where there is consistency in reported regions of neural activation during exposure to food marketing). Identifying limitations in the evidence base (e.g., lack of relevant studies with adults, small sample sizes with a paucity of power calculations) may also be useful for informing future research activity in this field. Developing standardized protocols for food marketing neuroimaging studies (as has been undertaken for food marketing monitoring, where such protocols have been used to facilitate the collection of comparable data internationally[62,76]) may also be useful to address this issue.

In addition, because of the small number of contributing experiments in the current analysis, a more lenient cluster-forming threshold of $p < 0.01$ (rather than $p < 0.001$) was employed. This is an acceptable threshold to use in instances such as this where there are relatively few contributing experiments to the ALE; however, we would suggest treating the results with some caution, and suggest that the analysis be updated once a greater number of contributing experiments have been published. Eickhoff et al.[52] suggest a critical threshold of 17 experiments contributing to an ALE affords confidence that significant clusters are robust from being biased by one dominant (large sample) study. To mitigate the potential bias in the results from one dominant study, we conducted a series of leave-one-out sensitivity analyses. However, the sensitivity analysis does highlight some instability in the reported clusters that illustrate the need for cautious interpretation of findings. This instability may be, at least in part, explained by differences in responding by the type of marketing stimulus presented given the variability of stimuli used in the included studies (e.g., from brand logos to full TV commercials) as well as the inclusion of studies with both adults and children in the same analysis. Future research should seek to determine whether unique brain response patterns are elicited by different marketing media forms. The data so far does suggest that the greatest consistency of activation to food marketing exposure is observed in areas relating to visual processing, attention, sensorimotor activity, and emotional processing, which may underlie food choice decision-making.

5 | CONCLUSION

The findings of this systematic review and meta-analysis, although tentative, add strength to the notion that neurological responding (visual processing, somatosensory processing, interpretation of sensory stimuli, and perception of emotions) is part of the mechanism that drives observed effects of food marketing on eating behavior.[20] Results are consistent with those showing impacts of food marketing exposure on food intake and its behavioral antecedents,[4,77] and that effective restriction of food marketing exposure and its powerful persuasive strategies would support countries' obesity prevention efforts.[9]

AUTHOR CONTRIBUTIONS

EB was responsible for the systematic review, wrote the manuscript, and was involved in the interpretation of results. CR was responsible for the statistical analyses and was involved in interpretation of results. MM, AC, MA, TM, and AB were involved with the systematic review and the interpretation of results. EB, AC, and CR accessed and verified the data. All authors were involved in devising and agreeing the final protocol for this work, had full access to all the data in the study, had final responsibility for the decision to submit for publication, reviewed and commented on the draft manuscript, and approved the submission of the final manuscript.

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CONFLICT OF INTEREST STATEMENT

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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