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CARDIOVASCULAR DISEASE (JHY WU, SECTION EDITOR)

The Impact of Marketing and Advertising on Food Behaviours: Evaluating the Evidence for a Causal Relationship

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Abstract The prevention of overweight in childhood is paramount to long-term heart health. Food marketing predominately promotes unhealthy products which, if over-consumed, will lead to overweight. International health expert calls for further restriction of children's exposure to food marketing remain relatively unheeded, with a lack of evidence showing a causal link between food marketing and children's dietary behaviours and obesity an oft-cited reason for this policy inertia. This direct link is difficult to measure and quantify with a multiplicity of determinants contributing to dietary intake and the development of overweight. The Bradford Hill Criteria provide a credible framework by which epidemiological studies may be examined to consider whether a causal interpretation of an observed association is valid. This paper draws upon current evidence that examines the relationship between food marketing, across a range of different media, and children's food behaviours, and appraises these studies against Bradford Hill's causality framework.

Keywords Food · Beverage · Child · Marketing · Advertising · Obesity · Causation · Bradford Hill

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Introduction

Cardiovascular disease (CVD) is largely preventable, yet, globally, it contributes to the greatest burden of premature mortality [1]. Modifiable, diet-related CVD risk factors include overweight and obesity and high consumption of saturated and trans fatty acids, refined carbohydrates and sodium [2, 3]. Establishing healthy dietary behaviours and maintaining a healthy weight in childhood is paramount for maximising heart health [4]; however, within our current food environment, this has never been more challenging.

Overweight is a natural response to today's obesogenic environment [5]. Our food supply is dominated by inexpensive, highly processed, yet highly palatable, energy-dense nutrient-poor food products [6]. This food environment is all our current generation of young people have ever known; they are high soft drink, snack and fast food consumers [7, 8] and are more susceptible to overweight than ever before [9]. A risk that is highlighted by the fact that a positive energy gap of approximately 200–300 kJ a day is all that is needed for the development of overweight in children [10, 11].

The public face of this toxic food environment is food marketing. The ubiquitous promotion of unhealthy food establishes societal norms around acceptable and desirable foods [12, 13] and adverts serve as conditioned stimuli that trigger food cravings and cue an increase in food consumption [14•], particularly in children [15•]. The disparity between

food advertising expenditure for different food groups is extreme [13]: government campaigns promoting fruit and vegetables are dwarfed by the billions of dollars spent on fast food and 'junk' food marketing each year [16, 17]. The majority of these advertised foods and drinks are high in added fat, sugar and salt; are contrary to dietary recommendations; and, if eaten in excess, can contribute to overweight and the risk of developing CVD [18–21].

Restricting food marketing to children has been identified at the highest levels of international policy setting as a priority public health nutrition intervention [22, 23]. It is one of 25 targets set by the World Health Organisation to reduce non-communicable disease premature mortality by 2025 [24]. Some countries have shown leadership in policy action reform; however, the global response as a whole remains limited [25], stymied by the food and advertising industries actively contesting and undermining public health policies and programmes [12, 26, 27].

A large number of reviews over the past decade have assessed the relationship between different aspects of food marketing and its effect on children [15•, 28•, 29•, 30•, 31–34]. The most recent comprehensive systematic review concluded that there is strong evidence that food marketing affects children's food purchases both at a food category and brand level and found modest evidence that it influences their food knowledge, preferences and choices; consumption behaviours; and diet-related health [32].

Despite this evidence, the difficulty in quantifying the relative contribution of food marketing on childhood obesity, and the establishment of a causal relationship between the two, are oft-cited reasons, by both governments [35, 36] and the food industry [37], for the limited action to restrict children's exposure to unhealthy food marketing. The development of obesity is multi-factorial [5] and the pathway linking food marketing exposures to children's weight is complex; most likely operating through sequenced and cumulative impacts over time, ultimately influencing the consumption of unhealthy foods [30•]. As such, the direct link to obesity is difficult to measure and quantify [30•]. Examining the evidence in relation to children's food behaviours, in particularly food consumption and intake, is, therefore, an appropriate way to investigate a causal link between unhealthy food marketing and children's weight [31].

Ascribing cause and effect in many areas of epidemiology is difficult, where observational studies that identify associations between exposure and outcomes may be a result of reverse causation, chance, bias or confounding [38]. Conducting experimental studies with high ecological validity in this field of research is also difficult. Given the prolific and integrated exposure to food marketing in everyday life, isolating its effect in an experimental setting is challenging and, in the longer-term, expensive and methodologically difficult [39]. The 'Bradford Hill Criteria', first published 50 years ago, are a recognised and widely used framework against which epidemiological studies may be examined to consider whether a causal interpretation of an observed association is valid [40, 41]. In this vein, these guidelines have been widely used in the public health arena to explore whether causal links exist between an exposure of interest and a behaviour or health outcome [42–45].

This paper draws upon current evidence from meta-analyses, reviews and empirical studies that examine the relationship between food marketing, across a range of different media, and children's food behaviours, including food preferences and choices, short-term food consumption and usual dietary intake. Included data are those published since the last systematic review in 2009, plus seminal papers prior to this date. Each study was reviewed and appraised according to the relevant Bradford Hill Criteria (Table 1). In this way, we categorise the evidence and examine whether there is a case to be made that a causal relationship exists between children's exposure to food promotion and their subsequent food behaviours.

Evidence Review of a Causal Relationship Between Food Promotion Exposure and Food Behaviours

We considered the following Bradford Hill Criteria in our appraisal of the evidence: strength of association, experimental evidence, dose-response relationship, consistency, temporality, biological plausibility and coherence. As noted by Bradford Hill himself, these are not pre-requisites that must be satisfied before an association can be judged as causal but rather serve as prompts for considering the weight of the evidence to assess if cause and effect is a realistic and credible deduction [40].

Bradford Hill Criteria: Strength of Association

Observational Evidence

Observational studies have found statistically significant positive associations between children's (3–18 years) exposure to food marketing and their food choice, consumption and usual intake [46–51], with effect sizes ranging from small (odds ratio (OR) <2) to moderate-strong (OR ≥3 or <4) [52]. Marketing exposure was assessed through either parental or self-report or using data on advertising patterns, including Gross Ratings Points (GRPs). GRPs give the proportion of the target audience reached by advertising for the category of interest in a specific geographic area during a certain time period (using this measure, an advertisement that reached 80 % of the specified audience and was shown 100 times during the year would have a GRP of 8000 (80 % × 100)) [50].

Longitudinal studies are considered to be the strongest non-randomised study design as these eliminate the possibility of

Table 1 Summary of evidence relating to specific Bradford Hill Criteria

Bradford Hill Criteria	Relation to causality	Study type/evidence base	Examples of specific reviews and studies
Strength of association	Hill suggests that strong associations are more likely to be causal than weak ones. However, he noted that relatively weak associations are common in epidemiology and should not be dismissed in representing a causal relationship [40].	Meta-analyses, reviews, longitudinal studies, cross-sectional studies, experimental studies	*Meta-analyses*: Boyland et al. 2016 [15•] *Longitudinal studies*: Olafsdottir et al. 2014 [46] *Cross-sectional studies*: *TV advertising*: Kelly et al. 2016 [47]; Giese et al. 2015 [48]; Scully et al. 2012 [49]; Andreyeva et al. 2011 [50] *Premium offers*: Longacre et al. 2016 [51] *Experimental studies:* *TV advertising*: Uribe and Fuentes-García 2015 [56]; Boyland et al. 2011 [57]; Dhar and Baylis 2011 [58]; Dovey et al. 2011 [59]; Anschutz et al. 2010 [60] and 2009 [61]; Harris et al. 2009 [62]; Halford et al. 2008, 2007 and 2004 [63–65]; Gorn and Goldberg, 1982 [66] *Advergames*: Folkvord et al. 2015, 2014 and 2013 [67–69]; Harris et al. 2012 [70] *Product placement*: Matthes et al. 2015 [71]; Auty and Lewis 2004 [72] *Brand endorsements*: *Reviews:* Smits et al. 2015 [28•]; Kraak and Story 2015 [29•]; *Experimental studies (not included in the above reviews):* Dixon et al. 2014 [73]; Elliott et al. 2013 [74]; Boyland et al. 2013 [75]; Robinson et al. 2007 [78] *Premium offers:* McAlister and Cornwell, 2012 [76]; Hobin et al. 2012 [77]
Experimental evidence	In Hill's opinion, experimental evidence offers the strongest support of a causal interpretation [40].	Reviews, experimental studies	*Experimental studies*: *TV advertising*: Uribe and Fuentes-García 2015 [56]; Boyland et al. 2015 [79]; Boyland et al. 2011 [57]; Dhar and Baylis 2011 [58]; Dovey et al. 2011 [59]; Anschutz et al. 2010 [60] and 2009 [61]; Harris et al. 2009 [62]; Halford et al. 2008, 2007 and 2004 [63–65]; Gorn and Goldberg 1982 [66] *Advergames*: Folkvord et al. 2015, 2014 and 2013 [67–69]; Harris et al. 2012 [70] *Product placement*: Matthes et al. 2015 [71]; Auty and Lewis 2004 [72] *Brand endorsements*: *Reviews*: Smits et al. 2015 [28•]; Kraak and Story 2015 [29•]; *Experimental studies (not included in the above reviews)*: Dixon et al. 2014 [73]; Elliott et al. 2013 [74]; Boyland et al. 2013 [75] *Premium offers*: McAlister and Cornwell 2012 [76]; Hobin et al. 2012 [77]
Dose-response	Dose-response relationships strengthen the likelihood of their being a causal relationship between an exposure and subsequent behaviour or health outcome [40].	Longitudinal studies, cross-sectional studies, experimental studies	*Longitudinal studies*: Olafsdottir et al. 2014 [46]; *Cross-sectional studies*: *TV advertising*: Kelly et al. 2016 [47]; Ng et al. 2015 [80]; Giese et al. 2015 [48]; Scully et al. 2012 [49]; Andreyeva et al. 2011 [50] *Experimental studies*: *TV advertising*: Uribe and Fuentes-García 2015 [56]; *Advergames*: Harris et al. 2012 [70] *Product placement*: Matthes et al. 2015 [71]
Consistency of evidence	A causal interpretation is strengthened when consistent findings are observed repeatedly across different study designs, populations and settings [40].	Reviews, longitudinal studies, cross-sectional studies, experimental studies	*Longitudinal studies*: Olafsdottir et al. 2014 [46]; *Cross-sectional studies*: *TV advertising*: Kelly et al. 2016 [47]; Ng et al. 2015 [80]; Giese et al. 2015 [48]; Lioutas and Tzimitra-Kalogianni 2014 [81]; Lee et al. 2014 [83]; Scully et al. 2012 [49]; Andreyeva et al. 2011 [50] *Premium offers*: Longacre et al. 2016 [51]

Table 1 (continued)

Bradford Hill Criteria	Relation to causality	Study type/evidence base	Examples of specific reviews and studies
			Experimental studies: *TV advertising*: Uribe and Fuentes-García 2015 [56]; Boyland et al. 2011 [57]; Dhar and Baylis 2011 [58]; Dovey et al. 2011 [59]; Anschutz et al. 2010 [60] and 2009 [61]; Harris et al. 2009 [62]; Halford et al. 2008, 2007 and 2004 [63–65]; Gorn and Goldberg, 1982 [66] *Advergames*: Folkvord et al. 2015, 2014 and 2013 [67–69]; Harris et al. 2012 [70] *Product placement:* Matthes et al. 2015 [71]; Auty and Lewis 2004 [72] *Brand endorsements*: *Reviews:* Smits et al. 2015 [28•]; Kraak and Story 2015 [29•] *Experimental studies (not included in the above reviews):* Dixon et al. 2014 [73]; Elliott et al. 2013 [74]; Boyland et al. 2013 [75] *Premium offers:* Gregori et al. 2014 [85]; Gregori et al. 2013 [84]; McAlister and Cornwell 2012 [76]; Hobin et al. 2012 [77] *Internet pop-up ad:* Tarabashkina et al. 2015 [82]
Temporality	Temporality is the one necessary criterion that must be met, that is, the exposure must precede the outcome [40].	Longitudinal studies, experimental studies	*Longitudinal studies*: Olafsdottir et al. 2014 [46]; *Experimental studies*: *TV advertising*: Boyland et al. 2015 [79]; Boyland et al. 2011 [57]; Dovey et al. 2011 [59]; Halford et al. 2008, 2007 and 2004 [63–65]; Gorn and Goldberg 1982 [66] *Advergames*: Folkvord et al. 2015 and 2013 [67, 69]; Harris et al. 2012 [70] *Product placement*: Matthes et al. 2015 [71]; Auty and Lewis 2004 [72] *Brand endorsements*: Dixon et al. 2014 [73]; Boyland et al. 2013 [75]
Plausibility and Coherence	The presumptive causal relationship is strengthened if the suspected connection is biologically plausible and does not seriously conflict with currently recognised theory or knowledge [40].	Meta-analyses, experimental studies	*Meta-analyses*: Boswell and Kober 2016 [14•] *Experimental studies*: Gearhardt et al. 2014 [97]; Bruce et al. 2013 and 2014 [98, 99]
Specificity	Absence of specificity is not sufficient to reject causality—Hill notes that one-to-one relationships are not frequent [40].	This criterion was not evaluated in this review.	

reverse causality. However, we found limited evidence in this regard, identifying only one prospective cohort study from Sweden (baseline ($n = 1733$, mean age 5.7 years) and 2-year follow-up ($n = 1333$)) [46]. Consuming sweetened beverages at least weekly at follow-up was predicted by exposure to commercial TV at baseline (OR 1.4, 95 % CI 1.1–1.9 ($p < 0.001$)), independent of parental sweetened beverage consumption norms. In this study, cross-sectional analyses of baseline data found stronger effects, with exposure to commercial TV associated with drinking sweetened beverages at least weekly (OR 1.6, 95 % CI 1.3–2.1 ($p < 0.001$)), compared with no commercial TV exposure. Where parents did not fully limit their children's exposure to commercials, the likelihood of drinking sweetened beverages at least weekly was more than double compared with children whose parents restricted their exposure completely (OR 1.9, 95 % CI 1.4–2.6 ($p < 0.001$)). Parent proxy reports may have been subject to social desirability bias with reported screen time and sweetened drink intakes lower than national comparative data. As such, the strength of the association would have been attenuated.

Similar trends were seen in the following studies when children self-reported exposure to food marketing (either on TV, print, on public transport, at school or digitally) and their usual food intake and/or purchase requests for promoted products [47–49].

An Australian study ($n = 12{,}188$, 12–17 years) found that compared with children who did not watch commercial TV, average viewers (≤2 h/day) were more likely to report asking for a food product they had seen advertised in the last month

(OR 1.25, 95 % CI 1.10–1.42 ($p < 0.01$)), whilst more frequent viewers (>2 h/day) were more likely to have both asked for (OR 1.61, 95 % CI 1.38–1.88 ($p < 0.001$)) and tried a new food product they had seen advertised (OR 1.48, 95 % CI 1.27–1.71 ($p < 0.001$)) [49]. Stronger associations were seen between exposure to all forms of non-broadcast (non-TV) marketing and children's food choices, particularly in children with greater than two exposures in the last month (ORs 2.48–3.74 ($p < 0.001$)). The stronger association with digital marketing could be as a result of the personalised nature of these messages and the interactive content [53].

Another Australian study ($n = 417$, 10–16 years) showed a significant positive association between watching commercial TV when children did not skip through advertisements, and unhealthy dietary intake ($F [3, 307] = 5.44$, $p = 0.001$). In contrast, watching commercial TV without advertisements was not linked to poor diet and non-commercial TV was only weakly associated with unhealthy food and drink intake [47]. This pattern was also observed in a study of young people from three European countries ($n = 2851$, age 8–21 years) with those exposed to all types of unhealthy TV food advertising having a higher consumption of fast food, snacks and soft drinks of up to 1 unit per week compared to unexposed participants ($p < 0.001$) across all countries [48].

The reliance of these studies on self-report and recall gives potential for measurement error with underreporting of dietary intake [54] and for true exposure to marketing to be underestimated [55], likely diminishing the strength of associations and attenuating results towards the null hypothesis. Conversely, there is also the possibility of some recall bias, with children who consume more unhealthy foods potentially being more likely to remember advertisements for these products.

A US study ($n = 9760$, mean age 11.2 years) took a different approach [50]. Dietary intake data from a national cohort study was compared with annual GRPs for spot advertising for sugary drinks and fast food restaurants purchased for the survey year and the preceding 2 years. For soft drink intake, an increase in exposure to advertisements for soft drink by 10,000 GRPs over the 3 years (equivalent to 100 advertisements over 3 years) was associated with a 9.4 % increase in children's consumption of soft drinks ($p < 0.01$). Advertisements for fast food were also associated with increased soft drink intake.

There is a risk that observed associations may have been due to confounding by other correlated dietary and lifestyle factors; however, factors such as such as age, gender, weight, socio-demographic characteristics and total TV viewing were typically adjusted for in multivariate analyses across the studies described.

Experimental Evidence

Associations seen in observational studies are supported by a strong, rigorous experimental evidence base. A recent meta-analysis reviewed the effects of acute exposure to unhealthy food advertising on food and drink consumption in children and adults [15•]. Sixteen experimental studies reported outcomes in children: 12 on TV and 4 on Internet advergames (an advergame is an industry designed online game with the brand embedded as a central component, such as a game piece). Twelve of these studies found unhealthy food advertising had a significant effect on food consumption, with children consuming a greater amount of food after seeing food advertising compared with controls (standardised mean difference 0.56; $p = 0.003$; 95 % CI 0.18, 0.94; $I^2 = 98$ %). Publication bias was ruled out with no signs of missing studies. Food advertising on TV and advergames was seen to have a significant effect on children's food consumption with a moderate magnitude.

Similarly, our review of experimental evidence, presented below, uncovered that exposure to marketing across multiple platforms strongly influenced children's food preferences, choices or food consumption. This included studies that manipulated exposures to TV advertising [56–66], Internet advergames [67–70], product placement in movies [71, 72], brand endorsers [28•, 29•, 73–75] and premium offers [76, 77].

Experimental Studies Using TV Advertising A series of studies by a UK research group consistently showed significant increases in children's immediate food consumption ($p < 0.001$) following exposure to unhealthy food advertising embedded in cartoons, across two different age ranges (5–7 and 8–11 years) [63–65]. In these studies, children, on average, consumed 16 % more kilojoules after exposure to food adverts compared with control toy adverts, in within-person crossover design trials. An increased effect was commonly observed amongst overweight and obese children, although this difference in intake by weight status was only seen in unhealthy food advertising conditions.

A more recent UK study examined TV advertising effects amongst children who had higher than usual exposures to marketing, as determined by volume of TV watched [57]. This study ($n = 281$, 6–13 years) demonstrated that food advertisements increased the preference for branded, energy-dense foods particularly in children who watched more TV (>21 h per week) ($p < 0.001$) [57].

Similarly in the USA, children ($n = 108$, 7–11 years) were shown cartoons embedded with either food or non-food advertisements and were given a bowl of crackers, which they could eat whilst watching [62]. Children ate 45 % more during the food advertising condition ($p = 0.01$), regardless of weight status.

Experimental Studies Using Internet Advergames The significant effect of unhealthy food Internet advergames on children's subsequent food intake has been demonstrated [67–70].

A US study ($n = 121$, 7–12 years) found that children who played a branded unhealthy food game ate over 50 % more energy-dense snack foods (322 kJ) than those who played a similar healthy food game ($p < 0.05$) [70]. Likewise, children ($n = 270$, 8–10 years) in a Dutch study ate more ($p < 0.001$) after playing a game promoting energy-dense foods compared with a non-food game (284 kJ (53 %)) and with the no-game control group (316 kJ (57 %)) [67].

Experimental Studies Using Product Placement Two European studies have shown the effect of product placement in movies on subsequent soft drink selection [72] and food choice and consumption [71]. After watching a two minute movie, children ($n = 57$, 11–12 years; $n = 48$, 6–7 years) who were exposed to a Pepsi product placement were more likely to choose Pepsi as a drink ($p = 0.04$) [72]. The second study showed children ($n = 121$, 6–14 years) a 7-min excerpt from a popular children's movie that contained a product placement for the savoury snack, Utz Cheese Balls. Exposure to the product placement exerted a significant effect on snack consumption ($p < 0.05$).

Experimental Studies Using Brand Endorsers The strength of the effect of branding, in the form of brand mascots and characters, cartoon media characters, and celebrity or sports endorsers, on children's food behaviours has been reported in two recent systematic reviews [28•, 29•]. The reviews examined 18 experimental studies (for children aged between 3 and 12 years) with a wide heterogeneity of design. They concluded that the evidence clearly demonstrates that brand endorsers have the persuasive capability of increasing children's liking of, and preference for, foods they endorse. Particularly strong effects were seen when familiar media characters were paired with energy-dense, nutrient-poor foods.

The power of branding was demonstrated in a study of American pre-schoolers ($n = 63$, 3–5 years), where they were asked to rate the taste of identical foods, with or without the McDonalds logo [78]. After tasting the foods, children were significantly more likely to state that the McDonalds branded foods tasted better than the matched plain-packaged pair ($p < 0.001$) for both healthy and non-healthy foods. This study was replicated in Canadian children ($n = 65$, 3–5 years) with similar findings, but noting highly colourful packaging, as typically found in non-healthy foods targeting children, was also influential [74].

A UK study ($n = 181$, 8–11 years) demonstrated the strength of the effect of exposure on TV to a sports celebrity endorser and found that significant consumption effects ($p < 0.05$) were not only seen after exposure to the endorsed commercial but also after TV footage of the endorser in his role as a presenter [75].

Experimental Studies Using Premium Offers The strength of toy premiums on children's food choices has been demonstrated in two different age ranges. When presented with 'meal-deal' images, young children ($n = 56$, 3–5 years) were more likely to favour both healthy ($p < 0.01$) and unhealthy ($p < 0.001$) meals when they were paired with a collectible toy, compared with foods paired with a non-collectible toy or no toy [76]. At a holiday camp, older children ($n = 337$, 6–12 years) were offered a choice from two unhealthy and two 'healthier' McDonalds Happy Meals [77]. When all meals were paired with a toy, 19 % of children chose the 'healthier meals' compared with 40 % when the toy was only offered with the 'healthier' meals (OR = 3.19, 95 % CI 1.89–5.40) ($p < 0.0001$).

Bradford Hill Criteria: Experimental Evidence

A vast body of robust experimental research has been conducted, largely by research groups in the UK [57, 59, 63–65, 72, 75, 79] and also in the USA [62] [70, 76], the Netherlands [60, 61] [67–69], Australia [73], Austria [71], Canada [58, 66, 74] [77] and Chile [56] amongst a range of different ethnic populations [28•, 29•, 62, 70]. All these studies demonstrated significant effects on children's food behaviours from exposure to a wide range of advertising media and promotions. As reported above, the majority of these studies examined the acute, short-term effects of marketing on children's food behaviours (3–12 years). Many utilised between-subject study designs with children randomised to conditions ($n = 63$–1302) [56, 60–62, 66–77]; the remainder within-subject, counterbalanced designs with randomisation to condition order with a washout period of more than 2 weeks ($n = 42$–281) [57, 59, 63–65, 79]. In the main, studies have been conducted in familiar settings such as schools, childcare centres or school camps [57, 59–65, 68, 69, 71–73, 75, 79].

As previously mentioned, conducting experimental studies in this domain over longer periods is methodologically challenging and expensive [39] and, as such, research of this nature is limited. Two studies, however, give insight to the longer effects of food advertising exposure. The first study conducted at a Canadian children's camp over a 14-day period in 1982 ($n = 288$, 5–8 years) saw children who were exposed to 5 min of candy advertisements daily select significantly less fruit as a snack, compared with children in other advertising conditions (healthy food and non-food ads) (33–36 %, $p < 0.001$) [66]. A second Canadian ecological study compared household food purchases in the predominantly French-speaking province of Quebec ($n = 5024$) (which has a ban on food advertising to children) with the neighbouring, predominantly English-speaking province of Ontario ($n = 9177$) (without a ban) [58]. French-speaking households with children in Quebec had a 13 % ($p < 0.05$) lower propensity for purchasing fast food compared with equivalent

French-speaking households in Ontario: this equated to 40,691 fewer households in Quebec purchasing fast food per week in 1992, translating to an estimated reduction of fast food purchases of $88 million per year.

Bradford Hill Criteria: Dose-Response

Dose-response relationships were identified in a number of cross-sectional studies. An Australian study ($n = 417$, 10–16 years) [47] demonstrated that every hour of commercial TV viewing per week was associated with a 0.067 unit increase in unhealthy diet score; with food and drink scores calculated from reported frequencies for commonly advertised unhealthy products.

This trend was also observed amongst young Swedish children ($n = 1733$, mean age 5.7 years) with the odds ratio for at least weekly consumption of sweetened beverages being 1.5 (1.2–1.9) for each hour of TV watched per day. Further, exposure to commercial channels was independently associated with sweetened beverage consumption, regardless of the amount watched [46].

Another US study ($n = 9760$, mean age 11.2 years), which used purchased advertising data [50], found an increase in exposure to fast food ads by 100 advertisements over 3 years was associated with a 1.1 % increase in children's consumption of fast food ($p < 0.1$). The same increase in exposure to adverts for soft drinks increased fast food consumption by 7.4 % ($p < 0.05$).

This pattern was further shown in another Australian study ($n = 12{,}188$, 12–17 years) [49]. As children's exposure to both commercial TV and non-broadcast advertising increased, so too did their intakes of commonly advertised foods or likelihood of requesting or trying advertised foods (all $p < 0.001$). For example, with an increase from one to two digital marketing exposures, the odds ratios increased from 1.34 to 3.19 for children being likely to ask for an advertised food and from 1.47 to 2.54 for children being likely to try them. Likewise, this trend was also observed in a recently published Malaysian study ($n = 402$, 7–12 years), for every additional hour of TV viewing there was a 6 % increase (OR 1.06 (1.04–1.08) ($p < 0.05$) in the likelihood of children liking and asking their parents for advertised non-core food products [80].

Experimental studies have also demonstrated dose-response relationships across different media. The effects from playing branded advergames were increased for children who had played them previously: these children consumed 577 kJ more from unhealthy snack foods than those who played healthy or non-food advergames [70]. The authors suggest that familiarity makes game play more automatic and, potentially, the advertising message becomes more noticeable strengthening the effect.

High-frequency product placement in a movie had an increased effect on consumption. Given the choice of three similar snacks, 45 % of children consumed the advertised product after high-frequency exposure compared with 31 % after a lower-frequency product placement and 18 % in the control condition ($p < 0.05$) [71]. Similarly, combined exposure to McDonald's food product placement plus McDonalds TV advertising saw children's ($n = 483$, 9–15 years) intention to consume fast food increase from 18 % (control condition) to 47 % (single exposure) to 54 % ($p < 0.05$) [56], and for their intention to consume McDonalds increase from 38 % (control) to 45 % (single exposure) to 57 % (combined exposure) ($p < 0.05$).

Bradford Hill Criteria: Consistency of Evidence

Evidence from observational and experimental studies on the association between food marketing exposure and food consumption behaviours is highly consistent. Significant positive associations were seen in observational studies across a range of populations and countries, using a variety of instruments and methods to measure exposures to marketing and food behaviours in children aged 3–18 years [47–51, 80, 81]. Similarly, experimental studies have shown significant effects from exposure to TV advertising, Internet advergames, product placement, branding and premium offers on children's food preferences, choices and short-term food consumption in children aged 3–12 in a variety of different populations [28•, 29•, 56–78, 82]. These results have been demonstrated consistently across heterogeneity of study designs: within- and between-subjects, varying lengths of advertising exposure, and in a large variety of conditions and settings.

A small percentage of studies were inconsistent with other findings. A Korean cross-sectional study ($n = 2419$, 11–13 years) found that all associations between food advertising and reported intake disappeared after adjusting for the amount of television watched [83]. Two identical experimental studies in Latin America ($n = 600$, 3–10 years) [84] and India ($n = 1680$, 3–11 years) [85] found no significant association between TV food advertising exposure and children's subsequent snack food consumption. In these studies, children were exposed to varying amounts of food adverts embedded within a cartoon programme, presumably to assess if snack intake increased with increasing exposures. However, the absolute exposure to food advertisements was minimal (between 0 and 3 ads). Half of the children were also given a toy with their snack and this did not increase intake of the food. However, toy premiums are known to encourage purchase and choice rather than stimulate consumption once the product is obtained [33, 76]. Further, an Australian study ($n = 354$, 7–13 years) which explored the effect of a pop-up Internet advertisement on children's subsequent snack choice found that although exposed children chose the advertised food more frequently than the control group, differences did not reach significance [82]. Further analysis revealed a significant result ($p < 0.001$) in a subset of children who had low nutrition

knowledge and were hedonism-oriented, with obese children more likely to belong to this group ($p = 0.037$).

Bradford Hill Criteria: Temporality

The temporal relationship between food advertising and subsequent food behaviours is clearly established. A number of randomised controlled studies, discussed in this paper, have shown significant effects on children's food behaviours *after* showing them unhealthy food advertising on TV [57, 59, 63–66, 79], in advergames [67, 69, 70], as product placements [71, 72] and as branding in the form of celebrity endorsements [73, 75]. The one longitudinal study included in this paper also confirms this relationship [46].

Bradford Hill Criteria: Biological Plausibility and Coherence

Evidence suggests that young children are predisposed to prefer foods that are sweet and salty, particularly those that are high in energy density (e.g. high fat) [86]. These food preferences, however, are malleable and can be shaped through experiential learning from exposure to different foods [87]. Food and beverage advertising is predominately for foods that are high in added fat, sugar and salt [18–21], and exposure promotes these foods as being a normal part of daily intake and potentially undermines healthy nutrition messages [13]. Eating patterns are established early in life, generally extending into adulthood, and there is evidence that food marketing negatively influences the food environment and has a bearing on how children's dietary patterns evolve [33]. It is a hedonistic, not a homeostatic need, that is the main driver to consume these types of foods, with highly processed foods of this nature more likely to prompt overeating than healthier, less processed foods [88].

Food promotion is typically characterised by mouthwatering images of food, catchy music, humour, positive imagery and celebrity endorsements: content likely to promote positive, emotional associations, with both brands and products [89]. Contemporary social cognitive theories suggest that repeated exposure to this type of promotion can lead to changes in attitudes, beliefs and behaviours without a conscious, deliberate processing of the information presented [90–92]. Children are more likely to process food advertisements through this implicit route and so less likely to be able to defend themselves against its effects [93, 94].

Cue-Reactivity Theory proposes that food-related cues prompt cravings for food and induce subsequent food intake via previously conditioned responses [95, 96]. The strength of the influence of cue-reactivity and how it can explain behavioural responses to food advertising has been demonstrated in a recently published quantitative meta-analysis that assessed the predictive effects of food cue reactivity and craving on eating and weight-related outcomes [14•]. Results found that cue-condition and cue-reactivity paradigms had medium to large effects on eating ($r = 0.32$, 95 % CI 0.26–0.39, $z = 9.08$, $p < 0.001$) and weight outcomes ($r = 0.51$, 95 % CI 0.26–0.69, $z = 3.69$, $p < 0.001$). Visual food cues (e.g. images and videos) were as strongly related to food behavioural outcomes as reactivity to real-food exposure.

These theories are reinforced by recent functional magnetic resonance imaging (FMRI) studies. Areas of the brain related to both reward and cognitive control have been shown to be activated in children in response to food commercials [97] and food logos [98, 99], with obese children showing more pronounced responses to food logos ($p < 0.01$) [98].

Bradford Hill Criteria: Specificity

This criterion was not evaluated in this review. Food marketing is one of many intra-, inter- and environmental determinants which have the potential to affect dietary behaviours [5, 100]. Bradford Hill notes that the absence of specificity is not sufficient to reject causality with one-to-one relationships not frequently observed [40].

Conclusions

The current evidence on exposure to food marketing and children's food behaviours, when examined together, satisfies all key criteria commonly used to evaluate causal relationships in epidemiology. As such, there is compelling evidence that the two are causally related. The experimental evidence base is particularly strong for children aged 3–12 years, with exposure to marketing across all media platforms consistently demonstrating significant, negative effects on food preferences, choices and short-term food consumption. Observational evidence for children aged 3–18 years, in addition to confirming these findings, also shows the presence of significant positive associations between marketing exposure and poorer usual dietary intakes.

This review also highlights where gaps in the evidence base exist. Specifically, there is a lack of longitudinal evidence and experimental studies that investigate outcomes in adolescents and in the longer term, particularly whether the demonstrated acute increases in food consumption are not compensated for at later eating occasions leading to net energy imbalance. However, as previously noted, studies of this nature would be methodologically challenging and, in the case of longer studies, expensive. There is also a scope for further studies to assess the impact of other forms of online marketing. Despite these evidence limitations, the sum of the evidence appraised against the Bradford Hill criteria, and the particularly strong evidence base for children aged 3–12 years, substantiates the call at the highest levels for tighter

restrictions on all forms of food marketing to children. We concur with other public health advocates that it is time to shift the locus of responsibility for childhood obesity away from the individual and towards those that control the food system and resultant obesogenic environment [27].

Compliance with Ethical Standards

Conflict of Interest Jennifer Norman declares that she has no conflict of interest.

Bridget Kelly declares that she has no conflict of interest.

Emma Boyland declares that she has no conflict of interest.

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Human and Animal Rights and Informed Consent This review contains some studies with human subjects performed by Dr Kelly and Dr Boyland. There are no animal studies included in this article.

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