**Title: The relationship between food and non-alcoholic beverage marketing and children and adolescents’ eating behaviors and health: a systematic review and series of meta-analyses**

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**Key Points**

**Question:** What is the relationship between food marketing (compared to less or no food marketing) and eating behavior and health in children and adolescents across the extant literature?

**Findings:** In this systematic review and meta-analyses of ninety-six studies (64 randomized controlled trials, 32 non-randomized studies), food marketing was associated with significant increases in food intake, choice, preference, and purchase requests. There was no clear evidence of relationships with purchasing, and little evidence on dental health or body weight outcomes.

**Meaning:** Results support theimplementation of policies to restrict children’s exposure to food marketing.

**Abstract**

**Importance**

There is widespread interest in the impact of food marketing on children, however, the comprehensive global evidence reviews are now dated.

**Objective**

To quantify the relationship between food and non-alcoholic beverage marketing and behavioral and health outcomes in children and adolescents to inform updated World Health Organization guidelines.

**Data sources**

Twenty-two databases were searched (including MEDLINE, CINAHL, Web of Science, EMBASE, and The Cochrane Library) with a publication date limit from 2008 through March 2020. This review was pre-registered (Prospero CRD42019137993).

**Study selection**

Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines were followed. Inclusion criteria were primary studies assessing the relationship between food marketing and specified outcomes in children and adolescents (0-19 years). Exclusion criteria were qualitative studies or those on advertising of infant formula. Of 31,063 articles identified, 96 were eligible for inclusion in the systematic review and 80 in the meta-analyses (19,372 participants).

**Data extraction and synthesis**

Two reviewers independently extracted data. Random effects models were used for meta-analyses; meta-regressions, sensitivity analyses and p curve analyses were also performed. Where appropriate, pooling was conducted using combining p values and vote counting by direction of effect. GRADE was used to judge certainty of evidence.

**Main outcomes and measures**

Critical outcomes were intake, choice, preference, and purchasing. Important outcomes were purchase requests, dental caries, body weight, and diet-related non-communicable diseases.

**Results**

Food marketing was associated with significant increases in intake (SMD 0.25, 95% CI 0.15-0.35; p<.001), choice (OR 1.77, 95% CI 1.26-2.50; p<.001), and preference (SMD 0.30, 95% CI 0.12-0.49; p=.001). Substantial heterogeneity (all >76%) was unexplained by sensitivity or moderator analyses. The combination of p-values for purchase requests was significant (p<.001) but no clear evidence was found for marketing effects on purchasing. Data on dental health and body weight outcomes were scarce. The certainty of evidence was graded as very low to moderate for intake and choice, and very low for preference and purchasing.

**Conclusions and relevance**

Food marketing is associated with increased intake, choice, preference, and purchase requests in children and adolescents. Implementation of policies to restrict children’s exposure is expected to benefit child health.

**Introduction**

Global trends show substantial increases in obesity amongst children in recent decades.1 This has serious implications for morbidity and mortality given that childhood obesity tracks into adulthood2 and excess weight is an important risk factor for non-communicable disease.3 Changes in global systems are key drivers of rising obesity, specifically growth in the production of affordable, highly processed foods which are effectively marketed.4

Food and/or non-alcoholic beverage (hereafter: food) marketing that largely promotes products high in fat, sugar and/or salt (HFSS) is prevalent across television,5 digital media,6 outdoor spaces,7 and sport8 . Children and adolescents are particularly vulnerable to the effects of food marketing given their immature cognitive and emotional development, peer-group influence, and high exposure.9,10 The pathway linking exposure to HFSS food marketing with behavioral and health impacts is complex11 but associations meet the criteria for a causal relationship.12 HFSS food marketing also negatively affects numerous child rights including the right to the enjoyment of the highest attainable standard of health, the right to adequate food, and the right to privacy.13

Implementation of the WHO Set of Recommendations on the Marketing of Foods and Non-alcoholic Beverages to Children14 has been inconsistent13. The underpinning evidence review15 largely predated the internet as a major marketing platform16 and there is over a decade of new research to consider. Although its conclusions are corroborated by more recent reviews and meta-analyses,17-21 these are also limited to television advertising and dated digital marketing forms (e.g., advergaming), include selective outcomes such as intake, and lack assessment of evidential value or certainty. Therefore, WHO commissioned the current research to inform the development of updated recommendations to restrict food marketing to children.

**Methods**

We conducted a systematic review and a series of meta-analyses following Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines.22 The WHO Nutrition Guidance Expert Advisory Group Subgroup (NUGAG) on Policy Actions formulated the research question and identified the critical and important outcomes to be captured (Appendix A in the supplement). The terms marketing, exposure, and power were used as defined by WHO.23 The protocol was pre-registered in May 2019 (CRD42019137993).

**Search strategy and selection criteria**

We considered primary studies (randomized controlled trials (RCT) or non-randomized studies (NRS)) for inclusion if they assessed the relationship between food marketing and specified outcomes in children (0-19 years). Exclusion criteria comprised qualitative designs and studies assessing the impact of advertising for infant formula or of marketing strategies outside WHO’s definition. Critical outcomes comprised food intake, choice, preference, and purchasing (by, or on behalf of, children). Important outcomes were purchase requests (by children to a caregiver), dental caries/erosion, body weight/body mass index/obesity, diet-related non-communicable diseases (NCDs, including validated surrogate indicators). Outcomes are defined in the supplement (Appendix A).

Searches were conducted in April 2019 and updated in March 2020 by an information specialist. Searches were limited to studies added to databases from 1st January 2009 (the previous global review included evidence to December 2008)15. We searched MEDLINE, CINAHL, Web of Science, EMBASE, ERIC, The Cochrane Library (CDSR, CENTRAL), Business Source Complete, EconLit, Emerald, JSTOR, HMIC, Advertising Education Forum, The Campbell Library, Database of Promoting Health Effectiveness Reviews (DoPHER), Healthevidence.org, TRIP, IRIS, Global Index Medicus, KOREAMED, Communication & Mass Media Complete, Academic Search Complete, Index to Legal Periodicals & Books Full Text (H.W. Wilson). Targeted searches of Google and Google Scholar were undertaken. The search strategy is provided in the supplement (Appendix A). All searches were peer reviewed (checked for accuracy by three researchers and a WHO librarian).

These searches were supplemented by (i) hand searching reference lists of retrieved systematic reviews and eligible studies, (ii) contact with topic experts, (iii) forward and backward citation searching of included studies and (iv) a WHO evidence call for data24. No language restrictions were applied.

Two reviewers independently screened studies against the inclusion criteria; assessing titles and abstracts to identify potentially relevant studies then reviewing full texts. Titles and abstracts of articles not in English were screened using Google Translate, then researchers proficient in both languages translated full texts for review. For multiple publications from the same cohort, we used data from the main contrast (food marketing versus no, less, or less powerful marketing) or biggest sample. Disagreement was resolved through consensus, and, if necessary, consulting a third reviewer. The search and screening processes were combined for this and a parallel review on the effectiveness of food marketing policies (Prospero CRD42019132506).

**Quality assessment**

We used Risk of Bias 2 (RoB2) to assess bias in RCTs and the Newcastle-Ottawa Scale (NOS) to assess quality of NRS. Bias assessments were conducted by one reviewer and independently checked by a second.

**Data analysis**

Two reviewers independently extracted data using pre-piloted forms. Study authors were contacted if necessary to provide data. Where data were only available in a figure, we used WebPlotDigitizer for extraction.25

For studies with multiple interventions, we extracted data from all relevant interventions and the control group or most relevant comparator intervention. For studies with interventions comprising different levels of the same marketing exposures, we selected the largest (e.g., most advertisements) as the exposure arm to maximize identification of effects. Relevant outcome measures and effect estimates were extracted. Where more than one eligible effect measure was available, we extracted the most comprehensive measure (e.g., overall intake rather than of a single item) or prioritised the unhealthy categories.

Cochrane recommendations were followed for the synthesis.26 Meta-analysis was used where studies were sufficiently homogenous. Where meta-analysis was not possible, we selected the most appropriate synthesis method available: combining p values using Fisher’s method or vote counting by direction of effect (Appendix A in the supplement).

For meta-analyses, random effects restricted maximum likelihood estimator analyses were conducted using the ‘metafor’ package in R.27 The I2 (inconsistency) statistic was used to assess heterogeneity, with a value of I2 > 50% indicating substantial heterogeneity. We undertook leave-one-out, Trim and Fill28 analyses, Graphical Displays of Heterogeneity (GOSH) and Egger’s regression test to examine bias.29 We examined any influential cases with a DFBETA score >1.30

When appropriate, we conducted subgroup (moderation) analyses by study design (RCT vs NRS), marketing manipulation type (exposure vs power) and marketing channel (television vs digital vs packaging). Within RCTs we examined whether risk of bias scores (low vs medium) moderated the effect (not possible for the preference outcome due to the small number of data points), and within NRS we conducted meta-regressions to examine if scores on the NOS were associated with the effect. For diet and choice outcomes we examined whether mean age of children in the sample or body mass index (BMI) Z score of the sample was associated with effect size using meta-regression (not possible for the preference outcome due to the small number of data points).To examine evidential value we conducted p curve analyses using the ‘dmetar’ function in R.31

We used GRADE32 to judge the certainty of evidence as high, moderate, low, or very low (Appendix A in the supplement). Research team certainty assessments were revised where necessary following discussion with the WHO NUGAG Subgroup.

**Results**

31063 titles were assessed for eligibility and 28682 were ineligible (figure 1).

Of 2381 full text articles assessed, 96 studies were included in the systematic review and 80 in the meta-analyses. Study characteristics are in the supplement (Appendix B). Pooled critical outcome data for food intake, choice, and preference are summarised in Table 1. Overall forest plots are shown in figures 2, 3, and 4. Forest plots for subgroup analyses, GOSH and p curve plots are in the supplement (Appendix D).

Data relating to other outcomes, bias assessments and all GRADE tables are in the supplement (Appendices C, E, and F). No relevant studies were identified with the diet related NCDs outcome.

For food intake, 46 studies (in 43 articles) were identified (31 RCTs33-61, eight observational NRS62-69, and seven experimental NRS70-75). Pooled analyses of data from 41 studies (42 effect sizes) found that food marketing was associated with a significant increase in intake (SMD 0.25 95% CI 0.15-0.35, Z=4.77, I2=77.2%, p<.001; figure 2). The effect was robust to sensitivity analyses and GOSH analyses demonstrated that across 100000 iterations of the analyses the pooled effect was SMD~.24(see Appendix D in the supplement). . There was no statistical evidence that study design (X2(1)=1.75, p=.19), marketing manipulation type (X2(1)=0.39, p=.53) or marketing channel (X2(2)=0.71, p=.70) significantly moderated the effect sizes. A meta-regression of average age of children in the studies (mean=8.6 years, range: 4.1–13.6) on the effect size was not significant (B=-.02, p=.35). There was no association between BMI Z scores (mean=1.01, range: .01–2.30) and the effect size (B=.20, p=.24). The p curve continuous test for evidential value was significant (Z=8.226, p<.01), indicating a true effect, as the distributions of p values were more frequent at p<.01 compared to p~.05. Of the five studies not included in the pooled analyses, three found effects of food marketing on intake53,62,68 and two found no effect.64,75 The certainty of evidence for RCTs was moderate (affected by unexplained high heterogeneity), and for NRS was very low (observational studies have a lower starting position within the GRADE assessment and certainty was downgraded due to the imprecision of the effect size estimates from these studies).

For food choice, 37 studies (in 36 articles) were identified (27 RCTs59,76-96 and 10 experimental NRS72,97-100). Pooled analyses of data from 27 studies found that food marketing was significantly associated with food choice (OR 1.77, 95% CI 1.26-2.50; Z=3.27, I2=77.5%, p<.001; figure 3). Specifically, food marketing exposure was associated with increased odds of 1.78 times greater choice of the test item(s), irrespective of whether the test item was unhealthy or healthy. However, we note that only 385,87,99 of 27 effect sizes reported on choice of healthier items specifically and only one of those did so within a study design in which the marketing exposure itself was for healthier food85. The effect was robust to sensitivity analyses and GOSH analyses demonstrated that across 100000 iterations of the analyses the pooled effect was OR~1.702 (see Appendix D in the supplement). There was no statistical evidence that study design (X2(1)=3.01, p=.08), marketing manipulation type (X2(1)=0.012, p=.91) or marketing channel (X2(2)=0.02, p=.99) significantly moderated the effect sizes. A meta-regression of average age of children in the studies (mean=8.76y, range: 4.0–11.8) on the effect size was not significant (B=-.08, p=.53). The continuous test for evidential value was significant (Z=8.287, p<.01). Ten studies were not included in the pooled analysis, of these eight found an effect of food marketing on food choice (of which seven were in the direction of greater choice of test items with food marketing exposure74,101-106 while one found greater choice of test items in the control condition107) and two found no effect.104,108 Supplementary analysis of three of these studies103,104 that used a cross-over design with binary outcomes showed a non-significant pooled odds ratio (OR 3.45 95% CI 0.97-12.43). The certainty of evidence for RCTs was moderate (unexplained high heterogeneity), and for NRS was very low (observational studies, risk of bias, and imprecision of the effect size estimates).

For food preference, 20 studies (in 19 articles) were identified (12 RCTs53,77,79,94,104,109-114 and eight experimental NRS97,103,106,115-119). Pooled analyses of data from 12 studies found that food marketing was significantly associated with increased food preference (SMD 0.30, 95% CI 0.12-0.49, Z=3.21, I2=90.0%, p=0.001; figure 4). The effect was robust to sensitivity analyses and GOSH analyses demonstrated that across 100000 iterations of the analyses the pooled effect was SMD~ .53 (see Appendix D in the supplement). There was no statistical evidence that study design (X2(1)=0.19, p=.67), marketing manipulation type (X2(1)=0.44, p=.51) or marketing channel (X2(2)=1.29, p=.53) significantly moderated the effect sizes. The continuous test for evidential value was significant (Z=5.504, p<.01). Eight studies were not able to be included in the pooled analysis, of which six found an effect of food marketing on preference53,103,104,106,112 and two found no effect.117,118 When studies with cross-over designs and binary outcomes103,104,106,112 were analysed separately there was a significant effect of marketing on preference (OR=3.49 [95% CI 2.03 to 6.22], z=4.40, p<.001). The certainty of evidence for both RCTs and NRS was very low (inconsistency, imprecision).

For food purchasing, five studies (one RCT120, one experimental NRS121, and three observational NRS66,122,123) were identified. All three observational NRS (moderate-high quality) found an association between food marketing and purchasing (two effects of public health harm66,122, one of public health benefit123). The RCT (with some concerns of bias)120 and moderate quality experimental NRS121 found no effect. The proportion of studies finding clear effects of potential public heath harm (1 of 4) was 25% (95% CI 1.3%-78.1%). The proportion of studies finding unclear effects of potential public health harm (1 of 4) was 25% (95% CI 1.3%-78.1%). The proportion of studies that showed any effect (clear or unclear) of public health harm (2 of 5) was 40% (95% CI 7.3-83.0%). The certainty of evidence for both RCTs and NRS was very low (risk of bias, inconsistency, imprecision).

For purchase requests, six studies (five RCTs60,81,110,111,114 and one observational NRS68) were identified. The combination of p-values was statistically significant (ps<.001) in both model iterations (see Appendix A in the supplement) suggesting evidence of food marketing associations with this outcome. The certainty of evidence for RCTs was moderate (risk of bias), and for NRS was very low (observational studies, risk of bias).

For dental caries, two observational NRS were identified. A moderate quality study found a clear effect of public health harm124 and a high quality study found no effect.69 The proportion of studies that showed any effect (clear or unclear) of public health harm (1 of 2) was 50% (95% CI: 9%-90.5%). The certainty of evidence was very low (risk of bias, inconsistency, indirectness).

Very little evidence was available on the relationship between food marketing and body weight or BMI. Our review identified a single, moderate quality observational NRS with no significant effects66. The certainty of evidence was very low (risk of bias, indirectness).

No studies were found with relevant data on diet related NCDs or validated surrogate indicators.

**Discussion**

Food marketing exposure is associated with increases in children’s food intake, choice of and preference towards test items, and purchase requests. There was little evidence to support associations with food purchasing by or on behalf of children, while data relating to dental health and body weight outcomes were scarce. No studies were found for the diet related NCDs or validated surrogate indicators outcome.

The effect sizes from the pooled analyses were small for intake and preference, and moderate to large for choice, and were robust to sensitivity analyses. P curve analyses demonstrated significant evidential value, indicative of a lack of selective reporting or ‘p hacking’. These findings are largely consistent with, and build on, previous findings15,17,18,20,21 although there are some discrepancies. For example, Russell et al.20 identified a moderating effect of BMI, such that children with overweight or obesity consumed an average of 45.6 kcal more than children with healthy weight following exposure to food advertisements. That type of subgroup analysis was not possible here due to a lack of appropriate data reported in the studies (of the five effect sizes included in each group20, two came from pre-2009 so were excluded here).

A strength of the present review is that it has linked diverse formats of food marketing exposure (including newer digital forms such as influencer marketing) to a range of behavioural and health outcomes. Other analyses have reported on a single format of marketing exposure (e.g., screen-based) and fewer than three outcomes. The certainty of evidence for critical outcomes was most frequently rated as very low or moderate, which could be regarded as a limitation. However, as has been described previously125, this reflects the nature of the GRADE criteria. GRADE prioritises RCT data with clinical outcomes and requires certainty to be downgraded where there is unexplained heterogeneity, even where results are consistent between RCTs and NRS and show similar findings to previous reviews, as here. The substantial observed heterogeneity, also consistent with previous meta-analyses17,18,20, was unexplained by sensitivity analyses, or subgroup analyses on overall study design (although for intake and choice outcomes variability was reduced when only RCTs were included), marketing manipulation, marketing format, study quality, participant age or BMI. Therefore, this heterogeneity is likely a consequence of the large number of studies and more nuanced differences in study design (e.g., stimulus types and outcome measurement). Substantial variability in outcome measurement is acceptable in meta-analysis but has implications for heterogeneity and therefore GRADE assessments.126

As with the previous WHO review,15 much of the evidence lies at the proximal end of the spectrum (relative to a hierarchy of food marketing effects11) with data available on food intake, choice and preference outcomes, but far less for the more distal outcomes (body weight and NCDs). Intake studies tend to measure immediate or short-term intake (directly following exposure to the marketing stimulus), rather than assessing diet across the day or longer term. Research gaps at the distal end likely reflect the substantial methodological challenge of conducting such studies, given that weight gain (or development of diet-related NCDs) typically occurs gradually and there is limited variability in the marketing exposure children experience within any given country or culture.11

The evidence is almost exclusively from higher income countries, with only six studies conducted in lower-middle income countries.64,69,95,100,103,124 The representativeness of the data for those populations may be limited and there was no opportunity to examine potential differences by income. Although we could explore the impact of BMI and age on some outcomes through meta-regression, we could not conduct formal subgroup analyses by age (e.g., child versus adolescent), socioeconomic status, gender, or rural/urban residential status due to inadequate reporting (i.e., insufficient studies with data segregated by these characteristics) and a lack of studies of adolescents. Future research should address this.

This review provides a comprehensive update and quantitative synthesis of evidence of food marketing associations with critical behavioural outcomes and demonstrates the evidential value of these studies. WHO has previously recommended that Member States enact policies to restrict children’s exposure to unhealthy food marketing14 and the review findings support this position.

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EB was responsible for the systematic review, wrote the manuscript, was involved in the interpretation of results. AJ was responsible for the data analysis, wrote the manuscript, and was involved in interpretation of results. LM, MM, JH, AB, and KA were involved with the systematic reviews and the interpretation of results. EB had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. All authors were involved in devising and agreeing the final protocol for this work, reviewed and commented on the draft manuscript, and approved the submission of the final manuscript.

**Declaration of interests**

We declare no competing interests.

**Supplementary material** – see uploaded file.

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***Figure Titles and Legends:***

***Figure 1*: Study selection**

Legend: PRISMA flow diagram detailing the study selection process

Footnote: \* Reasons for exclusion: incorrect intervention, comparator, population, or date, duplicate records.

***Figure 2*: Forest plot of intake data from eligible studies**

Legend: Forest plot of the effect sizes for each study

***Figure 3*: Forest plot of choice data from eligible studies**

Legend: Forest plot of the effect sizes for each study

***Figure 4*: Forest plot of preference data from eligible studies**

Legend: Forest plot of the effect sizes for each study

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
|  | **Number of studies** | **Number of subjects in experimental arm**  **(% of total sample)** | **Number of subjects in control arm**  **(% of total sample)** | **Effect size**  **(95% CI)** | **GRADE certainty of evidence** |
| **Non-randomized studies** |  |  |  |  |  |
| Intake | 11 | 4245/8436  (50.3) | 4191/8436  (49.7) | SMD 0.34\*  (0.12 - 0.57) | Very low |
| Choice | 5 | 261/416  (62.7) | 155/416  (37.3) | OR 0.56\*  (0.05 - 5.99) | Very low |
| Preference | 4 | 1010/1972  (51.2) | 962/1972  (48.8) | SMD 0.21†  (0.07 - 0.35) | Very low |
| **Randomized trials** |  |  |  |  |  |
| Intake | 30 | 1456/2908  (50.1) | 1452/2908  (49.9) | SMD 0.20\*  (0.10 - 0.30) | Moderate |
| Choice | 22 | 1916/3838  (49.9) | 1922/3838  (50.1) | OR 1.97\*  (1.46 - 2.66) | Moderate |
| Preference | 8 | 894/1802  (49.6) | 908/1802  (50.4) | SMD 0.38†  (0.03 - 0.72) | Very low |

SMD = standardized mean difference. OR = odds ratio.

\*The high heterogeneity of the pooled effect size (≥50%) is unexplained by sensitivity analyses (although the effect did not change direction or significance).

†High heterogeneity (although the effect did not change direction or significance).

Sensitivity analyses demonstrated that there was variability in the effect when individual studies were removed but these analyses do not provide a public health relevant explanation for the heterogeneity so downgrading for heterogeneity is still appropriate within the GRADE assessment.

Definitions of all outcomes and detailed justification for the GRADE quality of evidence is given in the supplement (Appendix F).

***Table 1*: Pooled effects of food marketing compared with no marketing, less food marketing or less powerful food marketing on critical outcomes**