

Technical Appendix

Children, Commerce, and Obesity: What Role Does Marketing Play?

Obesity is only one of many aspects of children's and young people's wellbeing which may be affected by what businesses do, and the Government already has policies in place to address it. But because it has generated so much debate, media coverage, and research, because it was raised by many of the submissions to our consultation (see Appendix D) and because it is such a complex and contested topic, we here examine the issues and evidence, specifically about the role of marketing, in more detail than would be possible in the space available in the main report.

This appendix is not a comprehensive or fully up-to-date review. Instead, we draw mainly on the most comprehensive review to-date, the US Institute of Medicine's *Food Marketing to Children and Youth: Threat or Opportunity?* (McGinnis et al, 2006), supplemented by other sources and our own knowledge of marketing and of research on media effects. We have not attempted a systematic review of research published since the IoM review but, as far as we know, there is none that would affect our conclusions.

This appendix is structured as follows:

- First, as background (Section 1), we briefly review the definition and measurement of obesity and the UK policy context.
- Section 2 summarises the various influences on children's diets and related health outcomes including obesity. We discuss the complex nature of the problem; the challenge of establishing cause and effect; biological factors; lifestyle factors; food preferences and food availability; and the distinction between brands, product categories, and diets.
- Section 3 introduces the Institute of Medicine (IoM) study, including its definitions, framework, method and scope.
- Section 4 reviews the IoM's evidence on the influence of marketing on the precursors of children's diet, the diets themselves, and diet-related health.
- Section 5 then addresses the important issue of effect size: how big are the effects of marketing (specifically, television advertising) relative to other potential influences?
- Finally, Section 6 lists our conclusions.

Questions of 'impact' raise many issues of theory, methodology and the interpretation of findings, especially as these may inform policy deliberations. In what follows, we try to highlight the strengths and weaknesses of the various approaches that have been taken.

Our overall purpose is to examine in some detail the extent to which the evidence supports the claim that food marketing plays a material role in explaining childhood obesity. In doing so, we also aim to discuss some broader issues of research methodology that could not, for reasons of space, be elaborated in the main body of the report although they underpin many of its conclusions on other topics as well as on obesity.

Throughout, we emphasise that food marketing is one of many factors that, through a series of complex processes and mutual interactions, contribute to children's diet and obesity. Although food marketing does play a role in explaining childhood obesity, such claims need to be examined with considerable care. In particular, we hope that more studies will consider effect size (and, therefore, practical significance) rather than treating the role of marketing as only an either/or question.

1 Background

1.1 Obesity: Definitions and Measurement

There are many diet-related health issues, but we here focus only on obesity. The simplest definition of obesity is that it is the excessive accumulation of fat in the body. The immediate cause is equally simple: fat accumulates when more calories are consumed than are expended; this imbalance between calorie input and energy expenditure results, over time, in an accumulation of fat as adipose tissue.

To ascertain the prevalence of obesity world-wide, an agreed measure is necessary. The most widely used appears to be the body mass index (BMI) which is universally recognised as the appropriate measure for population surveillance, although this does not distinguish between weight associated with muscle and weight associated with fat at an individual level.¹

It is more difficult to obtain reliable and generally agreed indicators of obesity in children, though there are moves toward WHO-approved measures, and BMI measures with age-related reference curves are available for a number of countries for both adults and children.

Analyses based on BMI trends may have underestimated the scale of the obesity problem. BMI fell slightly during the 1980s before rising steeply in the 1990s. This probably reflected an initial shift of body mass from muscle to fat, with the loss of muscle masking the increase in fat. Waist circumference,

¹ The formula for BMI is the individual's weight (in kilograms) divided by the square of his or her height (in metres). 'Normal' weight is in the BMI range from 18.5 to 25 with BMI 25-30 often labelled 'overweight'. We can set cut-off points of >30 as obesity, with 30-35 as 'moderate' obesity, 'severe' obesity as in the range 35-40, with anything above 40 classified as 'very severe' obesity (WHO Consultation on Obesity, 2000; p9). Actuarial tables used by life assurance firms that relate mortality rates of men and women of different weights and heights are also used as a measure of being overweight or obese. These tables report 'typical' weight ranges for individuals of different heights, genders and frame sizes.

which measures abdominal fat and is a strong predictor of health problems as it informs clinicians about fat distribution, has increased much more rapidly than BMI over the same period, suggesting that obesity is also a function of where as well as how much adiposity is present in any individual (WHO Consultation on Obesity, 2000; section 2.4).

In short, measures of obesity are available and used extensively to obtain statistics on this problem worldwide. The measures are not immune to criticism, however, and results should be interpreted carefully.

1.2 The UK Policy Context

The Government sees obesity as one of the biggest health challenges the country faces and has made a public commitment to take action that will prevent more serious illness and reduce the costs to the health service and society in the future.

One view of the potential future scale of the problem was set out by the Foresight report *“Tackling Obesities: Future Choices”*, published in October 2007 (Government Office for Science, 2007). The report’s own summary of its conclusions is as follows (page 2):

“By 2050, Foresight modelling indicates that 60% of adult men, 50% of adult women and about 25% of all children under 16 could be obese....The NHS costs attributable to overweight and obesity are projected to double to £10 billion by 2050. The wider costs to society and business are estimated to reach £49.9 billion per year (at today’s prices)”.

We note that these projections are based on extrapolating 12 years’ data (from 1993 to 2004) a further 46 years to 2050, with apparently no theoretical justification for the particular extrapolation model. In our view, it would have been preferable to employ a range of complementary models, including a ‘causal’ model using an analysis and projection of explanatory variables (Armstrong, 1978), especially given the limited historical data and the very long-term nature of the forecasts.

Such projections should also take into account the likelihood that, even without government intervention, over a 46-year interval, a significant number of people will modify their diet and lifestyle to limit their weight gain and that companies will develop increasingly palatable lower-calorie food products, and other products and services, to exploit the resulting market opportunities. Alternatively, it might be better simply to acknowledge that we currently have no scientific basis for making such long-term projections.

Despite these concerns about the specific Foresight projections, we wholly agree that, based on the increases in obesity which have already occurred, this represents a major and urgent health challenge and requires a vigorous policy response. Further, the nature of obesity-related diseases, such as diabetes and heart disease, is that the main negative health consequences of

an increase in children's obesity today will take several decades to present (Kopelman, 2008). Action therefore needs to be taken well before the full consequences of inaction are clear.

In January 2008 the Government published "*Healthy Weight, Healthy Lives; A Cross-Government Strategy for England*" (Department of Health, 2008). Supported by £372 million for implementation, the aim is to enable everyone in society to maintain a healthy weight. The framework for action has five key themes:

- Children: healthy growth and healthy weight
- Promoting healthier food choices
- Building physical activity into our lives
- Creating incentives for better health
- Personalised advice and support

The strategy acknowledges that advertising is one of the many factors that can influence children's diets. The Government has therefore worked with the regulator Ofcom to restrict broadcast advertising to children of foods high in fat, salt and sugar (HFSS) and the Committee on Advertising Practice has updated the content rules for advertising food to children in non-broadcast media.

2 What Determines the Incidence of Obesity Among Children?

2.1 Obesity – a Multi-Factorial Problem

There is a general consensus that many factors operate in determining body weight, including obesity, and that they interact and mutually influence each other. In multi-factorial situations, the influence of two or more factors on an outcome (eg obesity) may result from their interaction with each other or they may each have a separate, additive effect. For example, any genetic contribution to obesity will likely interact with environmental factors in determining a person's weight, rather than each playing an entirely separate role.

As we shall see, it is problematic that, in relation to research on food promotion, few researchers have examined the complex interactions that operate, tending instead to treat food promotion as a simple, additive factor impacting on diet preferences or habits. Yet food marketing must be understood within the larger web of causality underlying children's food choice, health and obesity.

For example, research suggests that exercise levels (of both parents and

children), meal habits (of both parents and children) and exposure to advertising each make an independent contribution to accounting for variation in children's food choice, health and obesity, and further that they interact with each other, indirectly affecting children's health. Food knowledge also matters, though it does not translate straightforwardly into food behaviour. Declining levels of exercise are an important part of the explanation for rising obesity levels.

These complexities provide a difficult challenge to researchers seeking to establish cause and effect.

2.2 The Challenge of Establishing Cause and Effect

As argued in Livingstone (2004), most research in this field, as in other investigations of media effects, broadly follows Lasswell's original model (1948), asking '*who says what to whom on what channel and with what effect?*' The point is that each element of this question makes a difference, and so evidence must be evaluated in relation to the specific research question asked:

Question	Typical Methods	Main Factors Considered
Who	Analysis of range of message sources	Advertisers. Broadcasters. Health education bodies. Governments. Etc.
Says What	Message (content) analysis	Persuasive strategies. Balance of un/healthy messages. Food labelling. Etc.
To Whom	Range of sampling strategies	Children (variously defined). Young people. Parents. Peers. By age, gender, ethnicity, socio-economic status, etc.
On What Channel	Mapping of range of promotional channels (extent, expenditure)	Television advertising, public service messages, signs and packaging, merchandising, cross-promotions, etc.
With What Effect	Experiments, quasi-experiments, observations, interviews, surveys	Short/long term effects, direct/indirect effects, cognitive/behavioural/emotional effects. Effects on food knowledge, purchase intention, preference, attitudes, liking, purchase behaviour, pester power, memory for ads, products, etc.

The body of available research on food marketing to children is highly imperfect, neither comprehensive nor even in coverage, and containing many gaps, especially for marketing other than television advertising and for research conducted outside the USA. Many studies are designed to identify correlations not causes. Possible confounding factors tend to be examined where convenient to measure (e.g. age, gender) rather than appropriate (e.g. parental diet, peers' exposure to media).

To provide definitive proof of the effects of food marketing on obesity, it would be necessary firstly to isolate this variable from the range of other potential

factors involved, and secondly to demonstrate a direct causal relationship. However, there are several reasons why this would be difficult to achieve.

Only an experiment can demonstrate causality, as only an experiment controls for the many confounding factors that, in everyday life, distinguish children exposed to many, from those exposed to few, promotional messages.² However, in practice all experiments are vulnerable to the charge that they do not realistically reflect the conditions of everyday life – in other words, that their findings are not generalisable.

To make an experiment generalisable, one must conduct an experiment under realistic conditions – exposing children to controlled messages in ordinary rather than laboratory circumstances, randomly assigning children to, say, ‘high promotion’ and ‘low promotion’ conditions (i.e. experimental and control groups) in a convincingly naturalistic fashion, and taking measures over the long-term rather than short-term.

Attempts to conduct naturalistic or field experiments typically encounter two difficulties. First, it is more difficult than in laboratory experiments to eliminate extraneous or confounding factors, reducing certainty when drawing conclusions that the observed effects are due to variation in the independent measure (message exposure). Second, if one seeks to expose children over the longer-term to hypothesised harmful exposure, one encounters serious ethical difficulties which make it unlikely that such an experiment would be permitted by a human subjects/ethics committee (although this is less of an issue in the context of marketing messages similar to, or replicated from, the real world).

In the inevitable absence of such a ‘perfect experiment’, it has been argued that the convergence of findings between correlational and experimental studies strengthens the case for effects. This is fair, for both are central to the claim of effects. With purely correlational evidence, the direction of causality, and the question of third causes, cannot be resolved. With purely experimental evidence, the claim that findings can be generalised to the everyday lives of children cannot be sustained. Or, to put the same point more positively, with a correlational study, one can demonstrate the existence of an association between exposure and behaviour under naturalistic conditions. With an experiment one can demonstrate the existence of a causal effect of exposure on behaviour under controlled conditions. Still, an inference will always be required to link the two.

2.3 Biological Factors

There are various factors that can be generally categorised as dispositional

² This is achieved through the elimination of the influence of confounding variables, where possible, and through random allocation of participants to experimental or control conditions in order to balance out any influence of remaining confounds. Additionally, the use of blind or double blind administration to prevent the introduction of further confounds is standard in experimental studies.

based on genetic and physiological characteristics. For example it is known that people differ in the extent to which they are able to gain and lose weight, and there a heritability component in these individual differences. In a classic study Sims (1989) reported on the so-called 'Vermont prisoner study' in which prison inmates were paid to gain weight. Prisoners who had no family history of obesity were unable to gain enough weight to become overweight, even if they consumed up to 6,000 calories a day. The little weight they gained was, further, quickly lost when the study ended. On the other hand, prisoners with a family history of obesity gained weight readily and did not lose it again right away.

However, a genetic contribution does not mean that one's fate is predetermined. Other factors must also be in place before the genetic contribution is expressed. The World Health Organisation considers "...*that the genes involved in weight gain increase the risk or susceptibility of an individual to the development of obesity when exposed to an adverse environment*" (WHO Consultation on Obesity, 2000; p134). There are also individual and group differences in the hormonal and neural regulation of appetite, energy expenditure, early life growth and development patterns which we recognise but extended comment would be beyond our remit here.

2.4 Lifestyle Factors

Turning now to lifestyle factors, exercise has an important role to play. Many everyday activities involve little expenditure of energy - watching TV, reading, working or playing at a computer, talking with friends, eating, and driving a car. Modern societies have increased the importance of these, and they have reduced time spent on energy-intensive activities.

There is also a significant relation between inactivity and eating among both adolescents (Lytle et al., 1995) and adults (Simoes et al., 1995). So more sedentary lifestyles are associated with increased food intake as well as reduced energy expenditure. Physical exercise can help the expenditure of energy but it also needs to be both regular and sustained.

The World Health Organisation (WHO, op. cit.; p116) states that moderate physical activity is "*of profound significance*" because there is evidence of an interaction between levels of physical activity and the proportion of dietary fat intake, which determines whether energy balance can be sustained. Specifically, people who sustain moderate or high levels of physical activity throughout life can tolerate diets with a relatively high fat content (e.g. 35-40% of energy) whereas lower fat intakes (20-25% of energy) are needed to minimise energy imbalance and weight gain in sedentary individuals and societies.

Contrary to popular imagination, the evidence suggests that there has been a decline in energy consumption in diet between 1970 and 1990, although there has also been an increase in dietary fat intake as a proportion of overall diet during that period (Prentice and Jebb, 1995).

Short-term dietary interventions are the most effective weight loss strategy to initially lose weight, followed by drug interventions, with exercise the least effective strategy (Curioni and Lourenço, 2005). However weight can be quickly put on again and exercise seems to have a key role in maintaining weight loss (Prentice and Jebb, op. cit, p259). It should be noted however that reliable measures of diet and activity levels are difficult to obtain in practice.

2.5 Food preference and food availability

Children's (and adults') food consumption is determined by a combination of their *food preferences* – which of two or more alternative foods they select if given the choice – and *product availability* – the range of foods available to them.

Food preferences are largely determined by taste preferences developed in the early years, although as people mature, they also try to take health effects into account when making food choices (Conner and Armitage, 2002).

For small children, the range of foods available to them is determined almost entirely by their parents and other carers inside and outside the home. Later, schools too become a strong influence. In all these contexts, cost is a significant factor, especially for low-income families and school meals. Most families also cite 'lack of time' as a major barrier to healthy meal preparation. Cost and lack of time remain important as children get older and increasingly buy their own meals and snacks. For instance, the distance between a school and the nearest fast food outlet may well have significant health implications.

Food preferences and product availability also interact:

- Parents, carers, schools, and food businesses (shops, cafes, etc) tend to offer children what they will eat without a fuss, subject to cost and (except for food businesses) health considerations.
- As food preferences develop over time in the early years, they are strongly influenced by familiarity as well as by other influences in the home (eg for boys, what they see older males eating). In other words, what children are given to eat in the early years is an important determinant of their lifelong food preferences.

The latter point has important policy implications. It provides support for the fact that the primary targets of the Government's current *Change4Life* strategy are the parents – especially mothers – of young children. It also suggests that attempts to "wean" children away from the less healthy diet they are used to (as in Jamie Oliver's approach in *Jamie's School Dinners*) would be more effective if applied gradually, rather than expecting children to change overnight.

A further implication of the distinction between food preferences and product

availability is that it suggests that teenagers may tend to eat a less healthy diet than younger children. Typically, teenagers still have limited impulse control as well as limited money (or willingness to spend it on healthier meals) so that their food preferences are often still similar to those of younger children, while their freedom to choose what they eat is much greater.

2.6 Brands, Product Categories, and Diets

In considering the influence of marketing, especially advertising, on obesity, it is important to distinguish between brands, product categories, and diets (Ambler, 2006). In this context, a 'brand' is a specific named product or service such as a can of regular Coca-Cola.³ In marketing, brands compete against other brands within broader 'product categories', although there is often some ambiguity about how broadly these should be defined.

For instance, regular Coca-Cola clearly competes against regular Pepsi-Cola, so the category could be defined narrowly as regular colas. But it also competes against other regular carbonated soft drinks (CSDs), low-calorie CSDs (including Diet Coke, which is also part of the broader Coca-Cola brand), and other soft drinks including water, and possibly against other low-ticket items including crisps and sweets. Choosing the most appropriate category depends on the aim of the exercise and the research evidence on consumer attitudes and behaviour (essentially, which alternatives the consumer considers when making a purchase).

The reason why this distinction matters is that many commentators, having correctly noted that most food advertising is of HFSS foods, then limit their analysis of the impact of advertising to something along the following lines: *"Of course it increases children's consumption of these foods, otherwise companies wouldn't do it"*.

In reality, the reason companies advertise is to increase the sales (and, in some cases, relative prices) of their brands, not to increase category sales. Obviously, if the advertising also increases the size of the category as well as the brand's market share, that is an added bonus (despite also helping the competition), but unless the category is new or very narrowly defined, or the brand has an extremely high market share, the effect of brand advertising on total category sales is small – in fact, usually too small to measure.

This also explains the failure of earlier generic category-level campaigns such as *"An apple a day keeps the doctor away"* from the 1920s to *"Go to work on an egg"* and *"Drink a pint a milka day"* in the 1950s and 60s (Fletcher, 2008, page 44).

This is not to say that brand advertising has *no* effect on category sales, only that the effect on the category is typically much smaller – eg by a factor of five

³ The other main meanings of 'brand', as a noun, are (i) trademark and (ii) 'brand equity' (Barwise, 2003, page xii).

or ten – than the effect on the advertised brand. From a policy perspective, in which almost all policies have costs as well as benefits, effect sizes matter.

Further, neither brands nor categories are the same as diets. A lab-based advertising experiment that, for instance, increases children's preference for Coke versus Pepsi tells us literally nothing about the extent to which, in the real world, Coca-Cola advertising increases children's total caloric intake and likelihood of obesity.

The potential links between advertising and obesity are therefore as follows:

- Advertising exposure
- Advertising/brand awareness leading to recall or at least recognition (e.g. whether a child remembers or recognises the Coke advertising, brand name/logo, etc.)
- Brand preference (e.g. Coke versus Pepsi)
- Brand consumption (i.e. the child actually consuming the product, which depends on many factors, of which brand preference is only one; as already noted, for young children, these other factors are dominant)
- Category consumption (e.g. all regular CSDs)
- Total caloric intake (i.e. all calories consumed)
- Net caloric intake (after allowing for physical activity)
- The impact on obesity (allowing for genetic and other intervening variables).

Finally, even at the brand level, advertising is rarely the most important or powerful part of marketing. In the long term, the appeal of the product itself is usually the most important factor, supplemented by other factors such as price and distribution as well as advertising.

In conclusion, marketing aims to increase the sales of specific brands by increasing both children's preference for them (mainly through advertising, packaging, and product development) and by increasing their availability (through price, promotion and distribution, as well as the influence of other marketing variables on the adults who control the food available to children).

Against this background, we now turn to the findings of the Institute of Medicine (IoM) report.

3. The 2006 US Institute of Medicine (IoM) Report

Before examining the IoM report, some justification for choosing it over other available reviews on the impact of marketing on food choice should be made. The first major review was done for a UK government department over 10 years ago (Young et al, 1996). More recently, four reviews appeared within four years of each other: Hastings et al (2003), Kaiser Family Foundation (2004), Kunkel et al (2004), Brand (2007) and the IoM review (McGinnis et al, 2006).

Much of the literature is common to all of these reports although the coverage differs slightly. Both the Kaiser Family Foundation (KFF) and IoM reports deal specifically with obesity although the former widens the scope by considering the role of media more generally (not just TV advertising) in obesity. Hastings et al (2003) worked with a brief from the UK Food Standards Agency that asked them to look at the current extent and nature of food marketing to children and the effect, if any, that this marketing has on their food knowledge, preferences and behaviour. Young et al (1996) covered similar ground. Kunkel et al (2004) and Brand (2007) looked at the more general issue of advertising to children.

There is also a narrative research review by Ofcom in the UK (Ofcom, 2004); a collaborative analysis of research from 20 European countries sponsored by the European Commission and administered through the European Heart Network (Matthews et al., 2005); and a series of World Health Organization (WHO) reports examining the possible linkages between marketing and childhood obesity (WHO and FAO, 2003) as well as the international regulatory environment for addressing such concerns (Hawkes, 2004).

Two of the reports (Hastings et al., IoM) used systematic review methods which in our opinion make them prime candidates for consideration. A systematic review strives for transparency at every stage of evidence gathering and evaluation. So the databases used, how 'grey' or commercially confidential material is obtained, and the search strategies employed are all fully described. In addition the ways in which each study or the results from each study considered separately are evaluated on criteria such as sample size, validity and reliability of procedures are discussed and described.

We chose the IoM study as our main evidence base because it is more recent and comprehensive and covers more literature than the review by Hastings et al (2003).⁴ In the IoM study, obesity as a health-related outcome is incorporated into a model where all aspects of marketing are considered. The criteria for evaluating studies include assessing their ecological validity (IoM, 5-13)⁵. Although it was a review produced in the USA, the catchment for research was worldwide (although the authors limited their review to English language research) and there is no reason to believe that their findings were specific only to the US.

3.1 Definitions and Framework

The IoM study uses a broad definition of marketing that embraces the '4 Ps' familiar to marketing managers worldwide:

- Product (ie features, quality, quantity, packaging)

⁴ Although Hastings et al. comes in as two volumes, the second volume is an appendix that provides a detailed summary of the relevant studies

⁵ Although both reviews describe clearly how the systematic review process was done, IoM explicitly mention that studies were evaluated on the extent to which the results obtained are likely to generalize to the naturally occurring world of marketing and young people's diets.

- Place (ie location, outlets and distribution points used to reach the target market)
- Price (ie strategy, determinants, levels)
- Promotion (ie advertising, sales promotion, public relations, trade promotions)

Advertising is thus located as one part of the marketing mix targeting children and young people (from 0-18 years), although the vast bulk of existing research is concentrated on television advertising rather than other aspects of marketing.

The report takes an ecological perspective to the relationships between marketing and the diets and health of children and youth. This recognises multiple factors at work, each of which may interact with the others, as shown in Figure 1 (McGinnis et al, 2006, page 3).

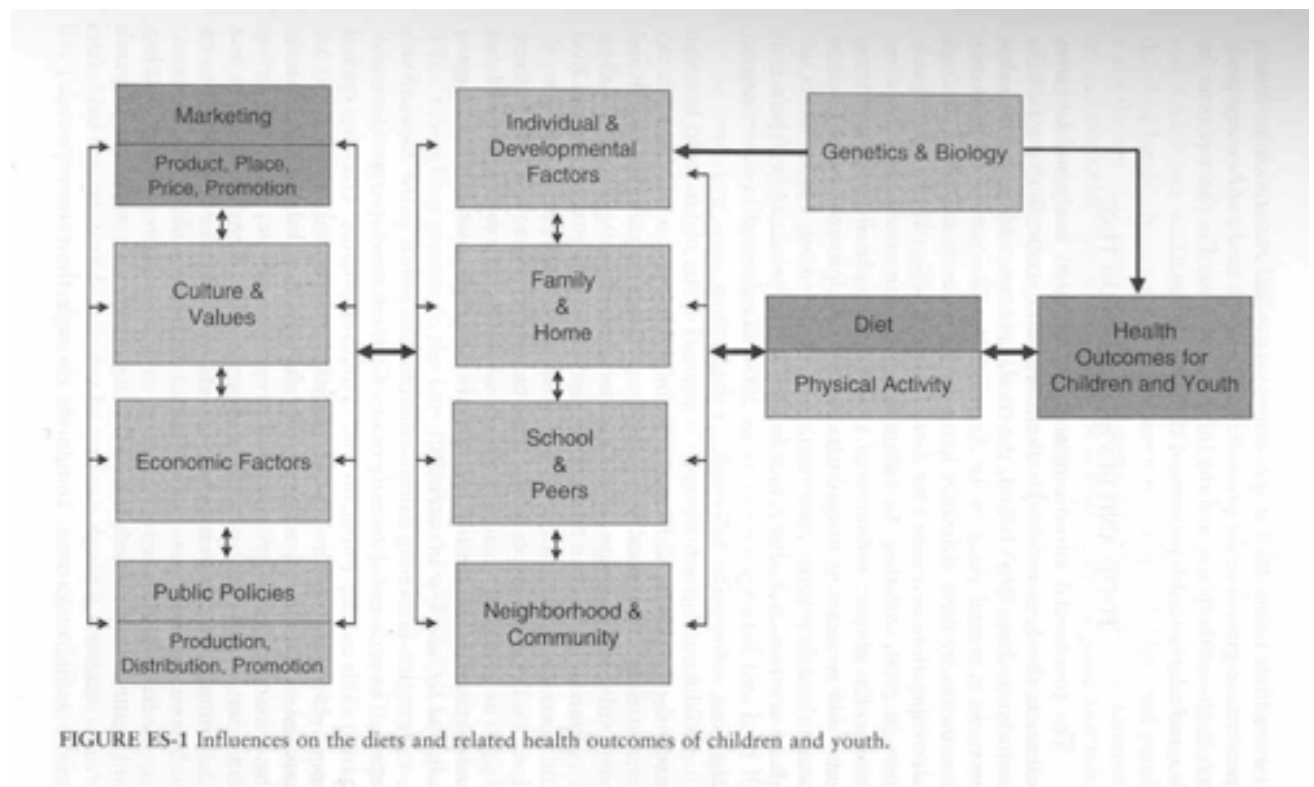


Figure 1: The US Institute of Medicine Framework

From Figure 1 we can see that children function within several layers of influence ranging from individual and developmental factors, through family and home, school and peers to neighbourhood and community (see the child development theory of Bronfenbrenner, 2005, and its application to diet in Story, Neumark-Sztainer, & French, 2002).

Marketing influences (as defined by the 4 Ps) are grouped together with culture and values, economic factors, and public policies. The main outcome

is health, which is mediated by genetics and biology on the one hand, and diet and physical activity on the other. The interrelationships among these various factors are indicated by arrows.

To understand these interrelationships, we must consider the elements of this model in turn.

1. The initiating factor is *marketing*, described by the 4Ps. For the population of children and young people, this might include a supermarket display at eye-level, a vending machine in school, a TV ad for sugared cereal on after school or price information on a billboard.
2. Second, one must consider the *precursors of diet*. A precursor factor is a factor through which causal influence passes. Examples include the TV ad for sugared cereal above if there is evidence that exposure to such marketing causes the child to make purchase requests. Food or beverage preferences, beliefs and purchase requests are specifically mentioned by the authors.
3. *Diet* itself is the third factor. It is defined in the report as the distribution and amount of food consumed on a regular basis. Not all studies measure diet in this way: the authors refer to the type of study which measures, say “the number of pieces of fruit or candy consumed in a child care setting during an afternoon following an exposure to television advertising for fruit or candy that morning” (5-4) as really only measuring short-term dietary behaviour.
4. Finally, *diet-related health*, including obesity and potential dependent variables such as the metabolic syndrome or type 2 diabetes. The authors use the term adiposity to cover this constellation of problems. Only physical health, not psycho-social health, is considered.

There are several moderators that can change the nature of the causal relationships. Most important here are age, socio-economic status (SES), gender, race or ethnicity, and family genetics. So, for example, for those children with low SES, the relatively high price and lack of availability of fresh vegetables (two of the Ps in a marketing mix) might stimulate demand for foodstuffs such as burgers, chips, or kebabs which are more available and cheaper per calorie.

The well-known literature on age-related differences in comprehending advertising intent would suggest that, for those children who do not understand the role of advertising (part of one of the Ps), there is a more powerful relationship between each of the three pairs: marketing and precursors of diet, marketing and diet, and marketing and diet-related health. These three pairs were therefore used to structure the IoM review of the literature which therefore fell into three sections looking at the relationships between each of the pairs of factors.

Such complexity does justice to the multi-factor nature of the influences on children’s diets, including the extent to which obesity is mediated by genetic,

biological and behavioural (ie exercise or physical activity) factors. The child is conceptualised as a constantly changing entity affecting and being affected by a variety of interdependent factors.

Inevitably, empirical researchers tend to examine the role of a limited number of factors operative at any one time, it being left to those who review the literature to put together the bigger picture. While it is, further, not difficult to be sceptical of the sample or methods employed in any one research study, it is important to seek this broader picture based on an assessment of the cumulative evidence.

3.2 The Process of Evaluating the Evidence

Chapter 5 of the IoM report examines the influence of marketing on the diets and diet-related health of children and youth. The evidence base consisted of 123 published empirical studies, providing 155 sets of results in all, obtained from a search that found nearly 200 studies in the published literature.⁶

After a description of the procedures involved in their systematic evidence review, the authors compare their collection of evidence with that of Hastings et al (2003), who had identified 55 articles or entries describing 51 relevant studies for systematic review. To quote the IoM report:

“Building on the base of that work, and applying even more stringent criteria for the publication quality of the studies reviewed, we have been able to assess and identify an even larger body of evidence. Hence, this committee’s review of the evidence represents the most comprehensive and rigorous assessment to date of food and beverage marketing’s influence on children and youth” (5-9)

The findings of each study were coded in terms of the cause variable (some aspect of marketing) and the effect variable (some aspect of factor 2, 3, or 4). Notes were then made on the measures used to operationalise each variable, the statistical significance level obtained, and the relevance of the results (assessed as high, medium, or low in terms of evidence for a causal relationship and extent to which one can generalise the results to everyday life).⁷

⁶ This process of distillation is characteristic of a systematic review where a sweep of the literature identifies a short list according to the criterion of relevance which is then reduced by a process of evaluation to the remaining pool that satisfy various criteria of acceptability and quality.

⁷ Competing concerns with internal and external validity often have to be traded off in social research and it is vital to assess the strength of each. In the IoM report, results were classified in terms of usefulness. A useful finding would be one that showed a relationship between marketing and diet-related health without being certain that one causes the other. A more useful relationship would reveal the direction of the relationship i.e. which was likely to be the cause and which the effect but without making any claims about the extent to which this would generalise to the everyday lives of children and young people. The strongest relationship would be one where a causal relationship has been established and one is confident that it has ecological validity and can apply in the world outside the laboratory.

Each of the 155 results was classified as belonging to either pair 1 & 2 (marketing and precursors of diet - 45 results), 1 & 3 (marketing and diet – 36 results), or 1 & 4 (marketing and diet-related health – 74 results⁸).

Unfortunately for the purposes of our present inquiry, most of the research identified (all but 6 of the 155 results included) concerned television advertising, even though this represents just one form of advertising targeting children in today's changed media landscape and advertising represents only a small part of marketing. As the authors say, with some understatement, "...considerable work is still needed to develop a full understanding of marketing's current role" (5-6).

However, the authors comment that:

"Overall, the research results included in the systematic evidence review were of sufficient quality, diversity, and scope to support certain findings about the influence of marketing, including the overall finding that food and beverage marketing influences the preferences and purchase requests of children, influences consumption at least in the short term, is a likely contributor to less healthful diets, and may contribute to negative diet-related health outcomes and risk" (5-20).

In the following section, we consider the results in each of these three areas in turn.

4 The Influence of Marketing on Children's Diet

4.1 Marketing and Precursors of Diet

There were 45 results in this section, mostly focussed on the marketing of high calorie, low nutrient food and beverages. Dependent variables included food preferences, purchase requests and beliefs.

The food preference literature used mostly experimental methods or cross-sectional designs and about 70% of the results were statistically significant (in the expected direction). Research into purchase request behaviour and how that was influenced by marketing involved a combination of cross-sectional designs and experiments and the vast majority produced significant results. The literature on how beliefs were influenced by marketing is usually explored using experimental methods and results show a majority producing significant results.

The IoM authors argue that one can generalise from these studies because

Finally, results were assessed on the strength of the research support for a finding using a 3-point scale with labels "strong", "moderate", and "weak".

⁸ Of the 74 results in the last category, 55 had low causal (internal) inference validity but 63 had high ecological (external) validity.

the ecological validity is good. An analysis of the extent to which different degrees of causal inference validity and ecological validity affect the probability of getting a significant result (the ratio of significant to non-significant results) suggests that the ratio or probability remains steady irrespective of the strength of research support. So if we compare high or medium ratings with low, the proportion of significant results remained high, leading the authors to conclude that this analysis is “...*providing further confidence in evidence-based findings for the influence of marketing on young people’s preferences for, purchase requests of, and beliefs about foods and beverages*” (5-24).

When four studies were removed that did not deal with television advertising, a similar pattern of results emerged. The authors identify three key findings as follows:

- There is strong evidence that television advertising influences the food and beverage preferences of children aged 2-11 years. There is insufficient evidence about its influence on the preferences of teens aged 12-18 years (5-26).
- There is strong evidence that television advertising influences the food and beverage purchase requests of children aged 2-11 years. There is insufficient evidence about its influence on the purchase requests of teens aged 12-18 years (5-27).
- There is moderate evidence that television advertising influences the food and beverage beliefs of children aged 2-11 years. There is insufficient evidence about its influence on the beliefs of teens aged 12-18 years (5-28).

Overall, the findings were summarised, as fairly supported by the evidence, thus:

“...[there is] a strong case for concluding that television advertising influences children’s food and beverage preferences, purchase requests and beliefs” (5-29).

Since content analyses of TV advertising of food and beverages generally show that children are exposed to advertising for high calorie and low nutrient products (Gamble and Cotugna, 1999; Kunkel and McIlrath, 2003), it seems plausible that each brand that is advertised results in a heightened probability that the child will prefer it, request it, get it and that it will enter the household and become part of the diet of children.

However, one must be wary of the simple assumption in this account that children’s beliefs and brand preferences, having been influenced by advertising, then significantly influence their purchase requests and actual diet (or food choices), partly through the child’s influence over the food choices of the family. Direct evidence on processes within the family is scarce, and the distinctions between brands, categories, and diets are important (see Section

2.6 above). To a remarkable extent, lobbyists campaigning for more restrictions on advertising have failed to address these complexities.

Further, this account evokes a distorted image of the child as merely a passive recipient of advertising, which elicits behaviours in an inevitable sequence from heightened preference through purchase requests to consumption.

The subtleties of children's active interpretations and actions within a multilayered social ecology of influencing and being influenced by parents, schools, peers and media must frame the process of food decision making, especially as this unfolds over time.

Nevertheless, as the IoM authors suggest, tracing a link from advertising to food preferences enables us to fill in at least one part of this bigger picture.

4.2 Marketing and Diet

There were 36 results in this section, which examined the relation between marketing (in fact, television advertising in all but two cases⁹) as the independent variable and actual dietary behaviour as the dependent variable.

The IoM authors classified results into two categories – effects on short-term consumption (e.g. within the experimental situation), and effects on usual dietary intake. An example of the first would be an experimental design in which some children are assigned to a treatment group that watched only sugared food and beverage ads in a TV programme, while others watched ads for fruit or milk, say, and then both groups are allowed free choice of a snack afterwards. An example of the second is provided by Bolton (1983), who used self-report diaries on television exposure in order to compute exposure to TV advertising combined with dietary intake diaries to calculate snacking frequency and nutrient information.

The IoM authors conclude, having noting that causal inference is weak in most of these studies, that: *“There is moderate evidence that television advertising influences the usual dietary intake of younger children ages 2-5 years and that there is weak evidence that it influences the dietary intake of older children ages 6-11 years”* (5-38). When it comes to teens, *“There is also weak evidence that it does **not** [emphasis added] influence the usual dietary intake of teens ages 12-18 years”* (5-38).

The Impact of Price (French, et al, 2001)

⁹ As there were only two studies that did not use TV advertising as the measure of marketing influence, one by French et al. (2001) on the effect of vending machine prices on the consumption of low-fat snacks and the other by Auty and Lewis (2004) on the effects of product placement, the authors couch their conclusions with reference to TV advertising

One high-quality study¹⁰ examines another aspect of the marketing mix, namely the relative price and purchase of low-fat snacks (French et al, 2001). Vending machines that sold both regular and low-fat snacks were placed in both workplaces and schools. The low-fat snacks were systematically varied on two dimensions of the marketing mix – ‘promotion’ (strictly, point-of-purchase advertising) and pricing.

There were three levels of ‘promotion’: no signs, signs labelling low-fat snacks, and signs labelling low-fat snacks with signs placed on the vending machine encouraging a low-fat snack choice. There were four levels of pricing: price equal to other items, 10% reduction, 25% reduction, 50% reduction. All possible combinations were used in a balanced design. The dependent variable was sales which were recorded separately for low-fat and regular snacks.

It was found that reducing the prices of the low-fat snacks had a strong effect on the sales of low-fat snacks in both the workplace and the secondary school. Further, the greater the reduction in the price of the low-fat snacks, the greater were their sales, both in absolute terms and as a percentage of total sales. In contrast, only the strongest promotional condition (labels plus signage) had a small but statistically significant effect on low fat snack sales. The implication is that, for both teens and adults, the effect of changing prices has a much stronger effect on consumption than the type of ‘promotion’ (point-of-purchase advertising) tested by these researchers¹¹.

Perhaps surprisingly, our further analysis of French et al’s results suggests that, although cutting the price of low-fat snacks did significantly increase the sales of low-fat snacks, it did not lead to any reduction in the sales of regular snacks.¹² The purchasers continued buying, and presumably eating, roughly the same number of regular snacks under all four price conditions for the low-fat snacks. The only difference was that, when the price of the low-fat snacks was reduced, purchases of low-fat snacks (and therefore total snack purchases) significantly increased. French et al did not report this result.

4.3 Marketing and Diet-Related Health

In the IoM report, 65 articles with 74 results were identified in this area. Practically all of them used a correlational design where data on the amount of TV viewing and some measure of adiposity (usually BMI or, for some,

¹⁰ In this study, measurement is good, the setting is naturalistic, the design is a classic experiment and thus both internal and external validity are high. The only provisos are that the experiment was fairly short-term and that it looked only at purchases from the vending machine, not total consumption.

¹¹ The different effect on sales of pricing versus advertising is well-known. For example the advertising elasticity (percentage change in sales for a 1% change in advertising) has been estimated as averaging only 0.1. Average price elasticity, however (percentage change in sales for a 1% change in price) is estimated as -1.7. (Tellis, 2004, pages 16-17).

¹² We used the results for the percentage and number of low-fat snacks sold (French et al, 2001, page 114) to derive the total number of snacks sold. Subtracting the number of low-fat snacks then gives the number of regular snacks sold.

skinfold thickness) were obtained¹³. For the most part, the methods used are large scale health surveys, generally based on well-drawn and representative national samples and often with careful measures applied to the outcome variable (health).

However, when it comes to examining the specific effects of marketing in this research, even though it includes direct measures of health, this field encounters a difficulty. The marketing (independent) variable is generally compromised, being measured simply as the self-reported amount of overall television viewing rather than the actual exposure to advertising. Although it seems likely that the more one watches television, the more advertising one is exposed to, this point is not itself supported by evidence.¹⁴

Moreover, there are many other likely correlates of self-reported television viewing that may also have consequences for, or be correlated with, health. These cannot be ruled out when examining the conclusions of such research, as the IoM authors themselves note. Specifically, as well as indicating exposure to advertising, television viewing...

1. ...takes up time that otherwise might be given to greater physical activity, leading to lower calorie expenditure, leading to greater adiposity
2. ...indicates an underlying preference (individual or family) for sedentary activities (including video gaming, reading, etc), leading to a lower energy expenditure and hence greater adiposity
3. ...provides a context for snacking, leading to higher calorie intake, leading to greater adiposity
4. ...blunts one's sensitivity to satiety cues, leading to greater calorie intake when eating during viewing, leading to greater adiposity
5. ...results in a reduced metabolic level, leading to less efficient processing of calorie intake, leading to greater adiposity
6. ...indicates exposure to food and beverage consumption within programmes, resulting in increased preferences, purchase requests, and other precursors of diet which then increase calorie intake, leading to greater adiposity¹⁵
7. ...if self-reported, reflects the respondent's attitude to the social desirability or otherwise of reporting oneself as watching a lot of television, which may correlate with her/his attitude towards food and/or health, including calorie intake.¹⁶

¹³ We are not suggesting that all these studies were the same. Our interest lies in the sort of moderating variables used and statistical analysis used to disentangle the cluster of variables that are unfortunately confounded in the behaviour known as 'television viewing'

¹⁴ The situation is even 'noisier' in the UK. Because a large percentage of TV viewing can be done on BBC channels (and thus avoiding advertising as the BBC does not carry advertising) then some heavy TV viewers might watch less advertising than other viewers who watch equally much but less BBC. Also – and this applies to both countries – the new media platforms that carry advertising like the Internet will not be measured. So errors of commission, in the former case, and errors of omission, in the latter, will both occur

¹⁵ The extent to which diet is represented in television programmes is a neglected area with only one source i.e. Dickenson (2005)

¹⁶ This point is especially problematic in studies (the majority) in which both the dependent

Also problematic for these large scale survey studies are the standard problems with inferring cause from a correlation – first, reverse causation (e.g. heavier young people choose a sedentary activity such as watching TV) and, second, mediating variables (e.g. adiposity and TV viewing are mediated by, or both separately explained by, SES).

With these appropriate caveats the authors draw the following conclusions. Of the 74 results, 69% were statistically significant, and the authors concluded that there is strong statistical evidence that exposure to television advertising is associated with adiposity in children ages 2-11 years and teens ages 12-18 years (5-47). Note that, given the methodological difficulties already noted, the claim is, appropriately, one of association rather than causal influence.

Can these findings shed any light on causality? Included in the literature were 17 studies which used a longitudinal design. They were all panel studies and these, taken with one experimental study (Robinson, 1999), do provide evidence that the direction of causality is at least partly in the predicted direction – from TV viewing to obesity, rather than the reverse.

The Robinson (1999) Advertising Exposure Experiment

Nonetheless, there remain grounds for scepticism. To consider one carefully conducted study in detail, the Robinson study was a randomised controlled trial intervention. Children participated over a 6 month period in a school-based curriculum designed to reduce TV, video and video game use. After two months of concentrated intervention, parents were also brought into the process.

It seemed to work. After six months, the children in the experimental group (as compared with the control group) had

- A significantly lower increase in BMI and 3 out of 4 other measures of adiposity,
- A highly significant reduction (4-6 hours per week) in television viewing and
- A significant reduction (about one a week) in meals eaten in front of the television set.

However, there were no significant differences between control and intervention groups on (i) reductions in servings of high fat foods, (ii) frequency of snacking while viewing, (iii) servings of highly advertised foods, (iv) sedentary behaviours and (v) various measures of physical activity.

This mixed set of findings, apparently successful but posing a considerable puzzle as to how the study achieved its effect, has not been satisfactorily

and the independent variables are self reported, creating so-called 'common-methods bias', ie spurious correlations purely due to individual response-style differences.

explained. Nor can it determine whether the effect was achieved by reducing exposure to advertising in particular or television in general.

The evidence reviewed in the IoM report from the longitudinal studies is also strong concerning directionality, with 80% of results finding a significant relationship in the expected direction – greater exposure to television results in greater adiposity. Again, however, one cannot separate the effect of viewing television from the effect of viewing advertisements.

When results are examined by age, most of the studies on younger children are strong on causal inference and show significant trends leading the IoM authors to claim that *“there is strong evidence that television advertising influences the short-term consumption of children ages 2-11 years. There is insufficient evidence about its influence on the short-term consumption of teens ages 12-18 years”* (5-35).

Alternative Explanations

Taking a different approach, the IoM authors then examined whether alternative explanations could account for the positive association between exposure to television advertising/TV and adiposity. Identifying 17 correlational results with medium causal inference validity, they examined evidence for each of the six possible pathways between television viewing and adiposity (as listed above), in addition to the hypothesised one of food and beverage advertising leading, via the various precursors of diet, to increased dietary intake and to increased adiposity.

After suggesting, plausibly enough, that several of these pathways may operate simultaneously to influence adiposity, they observed that:

“The question for the committee is whether exposure to television advertising is among them, not whether exposure to television advertising is the sole influence or the most important influence. None of the 17 results that tested enough variables to receive a medium causal inference rating covered all 7 plausible explanations using measures other than television viewing. Not one included direct measures of satiety cues, metabolic rate, consumption depictions, or television advertising” (5-51)

Finally, they conclude:

*“The association between adiposity and exposure to television advertising remains after taking alternative explanations into account, but the research does not convincingly rule out other possible explanations for the association; therefore **current evidence is not sufficient to arrive at any finding about a causal relationship from television advertising to adiposity among children and youth.** It is important to note that even a small influence, aggregated over the entire population of American children and youth, would be consequential in impact”* (our emphasis, 5-57).

5. Effect Size: How Big Are the Effects of Marketing?

Although few of the studies reported in this literature report the size of the effect (ie how much of the variation in the outcome measure – food preference, diet or obesity - is explained by marketing/advertising exposure),¹⁷ available comparisons with other factors suggest that the direct effect of food marketing (in the main, television advertising) on children is, though generally statistically significant, nonetheless small. It must be remembered that findings that are statistically significant may or may not be significant in policy terms.¹⁸

The IoM report briefly considers the size of the effect in the research it reviews, noting that “*The research examined typically explained a small rather than large amount of the overall variability in adiposity*” (page 5-91). It cites one national survey in the USA which found that, even when diet, age, gender, race/ethnicity, socioeconomic status, and television viewing are all taken into account, only 8.5% of the variability in children’s adiposity, and 11.4% of the variability in teens’ adiposity, can be explained (Storey et al., 2003). Storey et al estimated that, for every additional hour of *daily* television viewing, BMI could increase by 0.2. For this large increase in viewing, Dietz and Gortmaker (1985) estimated that the prevalence of teenage obesity could increase by just 2% (and again, it should be noted that this study considers the effects of television viewing rather than specifically the exposure to advertising).

Hancox and Poulton (2005), analysing a longitudinal panel survey of 1037 children from birth to 15 years in New Zealand, note that “*Correlation coefficients of this magnitude [between 0.05 and 0.12] are generally regarded as indicating a small effect size and suggest that television viewing [NB not advertising exposure] explains little of the variance in BMI*” (p.3-4).¹⁹ They add, however, that the correlations between viewing and BMI are greater than those observed between measured levels of physical exercise and BMI, and between measured dietary intake and BMI (though the relation between

¹⁷ Effect size is a statistical term that here refers to the proportion of observed variance in children’s food choice or obesity that can be explained by the direct and indirect effects of advertising exposure, when all other relevant factors (especially those known to correlate with both advertising exposure and food choice, such as socioeconomic status) have been statistically controlled for.

¹⁸ Requiring statistical significance is a means of ensuring that a *qualitative* result is highly unlikely to have been obtained by chance. This depends on the relationship between the underlying effect size, if any, and the sample size. Socially significant findings, on the other hand, are those where the *quantitative* effect size is big enough to make a difference that matters – which is, of course, partly a matter of judgment. With a small sample, few results are statistically significant. With a large enough sample, however, a result may be statistically significant even if the effect size is too small to be of any practical significance. See Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences* (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum Associates.

¹⁹ They also point to a range of measurement difficulties affecting surveys, especially longitudinal surveys, which reduces the strength of the association; the ‘true’ association between viewing and overweight, they therefore propose to be greater.

energy consumed and expended must, in theory, account for BMI). Hence, they still recommend interventions aimed at reducing television viewing.

A longitudinal study (with 548 11-12 year olds over 19 months) found that, for each additional hour of television viewing per day, fruit and vegetable consumption decreased by only 0.14 servings per day (Boynton-Jarrett et al., 2003). In the US Youth Risk Behavior Survey (of 15,000 high school students), *“Boys and girls were about 20% to 25% less likely to be classified as overweight if they reported 2 to 3 hours of TV per day and about 40% less likely to be classified as overweight if they reported less than 1 hour of TV per day, compared with those who watched [ie reported] 4 or more hours of TV”* (Eisenmann et al., 2002, p.379). Again, note that both these studies relate to total television viewing.

Several studies suggest that the influence of advertising exposure is small, especially in comparison with findings of greater influence of parental diet, product price, family meal habits or exercise (Ashton, 2004; French, 2003), though this is consistent with findings in other areas of media effect (Emmers-Sommer & Allen, 1999; Hearold, 1986; Kline, 2003; Livingstone, 1996) In particular, Bolton (1983) found that, among the broad array of factors within the home that influence children’s eating habits, the impact of exposure to food advertising was very small. As this study was one of the few listed by the IoM authors where effect size is discussed, it is worth examining it in more detail.

The Bolton (1983) Study of Influences within the Home

Bolton (1983) developed and tested a causal model of influences within the home on children’s dietary behaviour and their interrelationships. The extent to which the child has been exposed to food commercials, the kind of supervision and behaviour of the parents, and various behaviour patterns and other characteristics of the child all have a role to play in influencing the child’s eventual diet and the nutritional status of that diet.

These variables were operationalised using questionnaires and diaries and given to a sample of 262 children aged 2-11 years in 2-parent families in Cleveland, Ohio, in November 1977. The TV viewing diaries (far more accurate than self-reported estimates of the amount of viewing) were combined with detailed data from local TV stations to produce reliable estimates of the number of food commercials to which each child was exposed.

Using multivariate statistical analysis, Bolton found that children’s exposure to television food advertising did increase the number of snacks they consumed and that such viewing had a subsequent and independent effect on the child’s dietary efficiency and caloric intake. Exposure to food commercials had no statistically significant direct effect on caloric intake and only a very small indirect effect (accounting for 1% of the variance) through the effect on snacking. The effect on ‘nutritional efficiency’ (a measure of the balance

between nutrients and calories), while statistically significant, was also extremely small, accounting for 2% of variance directly and a further 1% indirectly. Bolton concluded that *“It is unlikely that effects of this magnitude could seriously affect their nutritional and physical well-being”* (page 194).

Specifically, she estimated that an additional 25 minutes a week of exposure to food commercials (equivalent to an extra 50 30-second commercials per week or seven per day, every day) would result in the consumption of one (0.97) additional snack a week, with the consequent result of an increased caloric intake of 1.39 per cent and a decrease in nutrient efficiency of 1.41 per cent. She also calculated that the child would have to increase her viewing by a third to access an additional 25 minutes of food commercials. The equivalent in the UK today would be an even greater increase in total viewing.

Variations in parental snacking behaviour accounted for 15 times as much of the variation in children’s snacking behaviour as did variations in exposure to television advertising. This gives us an indication of the order of magnitude involved when considering the relative effects of advertising on diet as compared with other models such as parents. Note that this was in the USA in 1977 (before pay TV and with commercial-free public TV having a much smaller share of both adults’ and children’s viewing than commercial-free pay-TV and the BBC have in the UK today) so the amount of advertising per hour will have been much higher than for the UK even before the recent restrictions.

The one proviso is that, because this was a cross-sectional study, part of the relatively high correlation between parents’ and children’s snacking behaviour is likely to have genetic causes: adults with a genetic predisposition to snack and watch TV are likely to have children who share that predisposition.

Effect Size: Summary

In sum, the indications are that the influence on children’s diets and obesity of television viewing and, especially, exposure to TV advertising is small. This is the case both for experiments (generally, on television advertising) and for surveys (generally, on overall television exposure).

However, although this is generally acknowledged (e.g. Ashton, 2004) some researchers argue that small effects in statistical terms add up to a large number of children in absolute terms, and that the cumulative effects over the period of a child’s development may be more sizeable, as some recent longitudinal research claims to show (e.g., Hancox and Poulton, 2005).

6. Conclusions

1. The influences on children’s diet and related health outcomes are wide-ranging and complex. Consequently, in order to provide reliable, policy-relevant evidence, research needs to be carefully designed and executed in

order to address the difficult issues of complexity and causality.

2. At the individual level, the determinants of adiposity are diet, physical activity, and genetics. At the UK population level, we assume that any changes to the gene pool over the last few years are likely to have been so small as to account for a negligible proportion of the increase in obesity. Therefore, the only significant drivers of the increase are changes in diet and changes in physical activity.

3. Further, our understanding is that the main change over the last 30-50 years has been a substantial reduction in physical activity including among children. There seems to be limited detailed research into how and why this has happened, how the trend could be halted or reversed, and the actual and potential role of the commercial world in both. In contrast, there has been a substantial amount of research into the determinants of children's diet although less into the longer-term trends in calorie consumption.

4. What children eat is determined by a combination of their food preferences and the food products available to them. Children's food preferences are almost entirely based on what they enjoy eating (i.e. taste). Taste preferences are initially innate and then develop in the early years, when they are determined by various factors. Familiarity with foods is important, as well as the influence of role models, especially same-sex role models, in the home. Once developed, taste preferences change little and only gradually.

5. The choice of food products available to children reflects a combination of decisions made by parents and other carers, schools, and food businesses. Within the home, by far the strongest determinant is what the rest of the family eats. Other influences are what the child will eat without a fuss, cost, preparation time, and nutrition. Food choice is often based on negotiation within the family, sometimes (especially with older children) involving reverse socialisation where parents are socialised by their children into healthier food choices.

6. Marketing is thus only one factor in this much broader range of influences. Marketing aims to increase the sales of specific brands by increasing both consumers' preference for them (eg through product development and advertising) and their availability (through price and distribution). In the case of children, especially younger children, product availability operates mainly through the adults who control the food provided to them.

7. There is an extensive research literature on the impact of marketing on children's diet. Unfortunately, most of this is of low quality in terms of relevance and/or reliability. A further limitation of the present evidence base is that almost all the research to date on the possible impact of the commercial world on children's obesity focuses on TV advertising. Advertising represents only part of the 'marketing mix' for food manufacturers and retailers. Furthermore, advertisers increasingly use other media such as the internet, partly because of tighter restrictions on the advertising of HFSS foods on

television.

8. Even within this narrow area, much of the cited research tells us little about the extent to which TV advertising is a material factor in children's obesity. Most researchers believe that it is one of many individual, social, environmental, and cultural factors but its significance relative to the other factors remains unclear and highly contested. However, we are not aware of any serious studies that suggest that advertising is a *major* influence on children's diet.

9. Most previous research in this area has important limitations:

- Many studies are cross-sectional but omit most of the relevant other factors and fail to address the question of causality. For instance, many have found a weak correlation between hours of TV viewing and obesity, but there are numerous causal mechanisms (eg more snacking, less exercise, a common underlying factor which might be genetic, the influence of programmes rather than advertisements) that could account for this weak correlation even if exposure to TV commercials had no effect whatsoever.
- Similarly, many studies demonstrate a weak effect of advertising on brand preference without giving any evidence that such brand preference perceptibly impacts category consumption, never mind overall diet.
- Most experimental studies lack 'ecological validity', that is, the experimental conditions are too far removed from the real world to be reliable.
- Other studies suffer from other weaknesses in design or execution, eg the sample is small and/or unrepresentative or the measures were of claimed rather than actual behaviour.

10. Despite the difficulty of doing valid research in this area, there have been several very convincing studies, discussed in some detail in Sections 4 and 5 of this appendix. Based on these and on the wider research base, our interpretation of the evidence is that extensive exposure to TV advertising does tend to increase children's consumption of HFSS foods, but the effect size is very small. The figure of 2% has been quoted but should be regarded as only indicative. It should also be noted that this is a measure of the effect on food consumption, which is only one of several factors in obesity. The influence on obesity is therefore likely to be even smaller than the effect on HFSS food consumption.

11. Other marketing factors such as price and distribution are likely to be at least as important as advertising. In the long run, another key factor will be the food industry's ability to develop healthier products which taste as good, or almost as good, as HFSS ones and cost the same, or almost the same.

12. For the future, little would be achieved by conducting more studies of limited relevance and/or reliability. What we need is a smaller number of better designed studies which come closer to the standards of randomised controlled trials.

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