

Assessing a child's appetite could help to prevent obesity

Childhood obesity: prevalence and diagnosis

Obesity is a condition of excess body fat accumulation that affects health. Body-mass index (BMI), a ratio of weight to the square of height (calculated using the equation: weight (kg)/height (m)²), is the most commonly used index of body fat in both adults and children. There are internationally recognised cut-off values to categorise adults as overweight (BMI>25kg/m²) and obese (BMI>30kg/m²). For children, defining overweight and obesity is more complex because BMI varies with development (age), sex and ethnicity. This means that the adult cut-offs cannot be used. Instead, age- sex- and population-specific cut-offs are applied, using reference data. In the UK, children's BMI values are compared to population reference data for children of the same age and sex from 1990 (UK90).¹ A child is usually defined as overweight if they have a BMI at or above the 91st centile relative to the population reference data; and obese if their BMI is at or above the 98th centile.² Other countries, such as the United States,³ have their own BMI-for-age reference standards. An international reference, produced by the International Obesity Task Force (IOTF), has been developed using data from children in the US, UK, Hong Kong, the Netherlands, Singapore and Brazil.⁴ This can be used to compare the prevalence of childhood overweight and obesity across different countries.

Childhood obesity affects 10% of children worldwide and the prevalence has over the past thirty years has doubled or trebled in almost all industrialised countries.^{5,6} In the UK, rates appear to be levelling off, but the prevalence remains too high. Data from the UK National Child Measurement Programme show that 1 in 5 children aged 4-5 years are overweight or obese when they start Primary School; and the proportion rises to 1 in 3 children aged 10-11 years at the end of Primary School.⁷

Health consequences of obesity in childhood

Obesity in childhood is associated with a wide-range of health complications. Children are at increased risk of cardiovascular conditions such as hypertension during childhood, as well as psychological ill health.⁸ Obesity has also been associated with asthma in childhood, with overweight and obese children at a 40-50% increased risk than normal weight children.^{9,10} Obesity in childhood tracks into adulthood, with obese children and adolescents approximately five times more likely to be obese in adulthood than those who are not obese.¹¹ Childhood obesity also predicts obesity-related morbidities in adulthood, including coronary heart disease, type II diabetes, and a range of cancers.^{12,13} In addition to the personal costs of obesity, there are huge economic costs to society. Obesity and obesity-related conditions cost the NHS over £5 billion per year.¹⁴

Risk factors for childhood obesity

Differences in child BMI are strongly influenced by genetic variation, with heritability estimates from twin studies averaging more than 50%.¹⁵⁻¹⁷ Environmental changes over the past 30 years, such as the increased availability of cheap, palatable food, and advances in technology which have reduced physical activity demands, have also played an important role in the large increase in obesity prevalence.¹⁸ Interestingly though, not everyone in the population has gained weight in response to the changing environment. In fact, individuals with the highest BMI have shown the largest increases in weight, so whereas thinner people

have tended to remain thin, fatter people have become considerably fatter.¹⁹ This observation has led researchers to hypothesise that those individuals who are at the highest genetic risk of obesity have gained the most weight, suggesting gene-environment interactions in the determination of weight. The implications of which being that more personalised intervention may be required.

The role of appetite in paediatric weight gain

The Behavioural Susceptibility Model of obesity proposes that the basis for a gene-environment interaction is that 'obesity genes' are influencing weight, at least partly through an appetitive pathway. In particular, individuals that inherit a set of genes that confer greater responsiveness to external food cues (wanting to eat when you see, smell or taste palatable food), and lower sensitivity to satiety ('fullness') are more likely to overeat in response to the current 'obesogenic' environment, and to become obese.²⁰ It is now well-established that children who react more strongly to appetising food than other children are more likely to be overweight, and prospective data have shown that questionnaire based measures of higher food responsiveness in infancy drives weight gain over time, suggesting a causal role for this trait in the development of overweight.^{21,22} A large body of research also indicates that overweight children tend to show blunted sensitivity to 'fullness' compared with normal weight children, and prospective data have also shown that taking longer to feel full predicts weight gain over time; again, suggesting a causal role for lower satiety sensitivity in the development of overweight.^{21,22} In support of the Behavioural Susceptibility Model, these appetitive traits have been shown to have a strong genetic basis in both infancy²³ and childhood²⁴ with heritability estimates as high as 62%; supporting an appetitive pathway for 'obesity genes'.

Although it is now well-established that greater food cue responsiveness and lower satiety sensitivity confer greater risk of obesity, until recently it has not been clear *how* these appetitive traits lead to overeating, in an everyday context. However, a recent study characterised the everyday eating patterns of a large sample (n=2203) of British toddlers (21 months), from the Gemini twin birth cohort. Parents completed a questionnaire about how food responsive and satiety sensitive their children were. Questions such as "My child is always asking for food" assessed food responsiveness, with scores ranging from 1 (least food responsive) to 5 (most food responsive). Questions such as "My child gets full up easily" assessed satiety sensitivity and scores ranged from 1 (least satiety sensitive) to 5 (most satiety sensitive). Parents also completed food and drink diaries over three days for each child. Information from the food diaries was used to calculate each child's average number of eating occasions (meals and snacks) in order to determine how often they ate, and the average amount of calories eaten at each eating occasion, per day, to determine how much they ate. The study found that children who are less sensitive to fullness consume more each time they eat (Figure 1), and children who are more responsive to food cues eat more frequently i.e. more times per week (Figure 2).

The findings suggest that both food responsiveness and satiety sensitivity may lead to overeating, but importantly, they appear to do it in different ways - one via *how often* children eat, and the other via *how much* they eat. It therefore seems that different aspects of appetite drive eating patterns in early life and these eating patterns potentially explain

how some children end up gaining weight via different pathways. This has important implications for approaches to prevention and treatment, and highlights the potential of targeted interventions based on the behaviours that characterise these appetitive traits.

In the modern environment food is abundant, cheap, easily accessible and widely advertised; so children who are highly responsive to food have many opportunities to act on their urges to eat. At the same time, if a child takes longer to feel full, or is less sensitive to fullness signals, they are likely to eat more at a meal in order to feel satisfied. This is contrary to the widely held belief that young children have an innate ability to regulate their appetite – i.e. that a large meal would be compensated for by a smaller meal at the next meal time, or that a day with many meals would be followed by a day with fewer meals. In fact, some children seem to be better at regulating their food intake than others. Food responsive children eat more frequently but do not reduce the amount of food they consume each time, and children with poor satiety responsiveness consume more food each time they eat, but do not eat less often to compensate for it.

Figure 1 illustrates the difference in energy intake per meal/snack for children scoring high (5/5) and low (1/5) on satiety sensitivity; children with poorer satiety sensitivity consumed more calories each time they ate. It is therefore possible to see how children with less sensitivity to fullness might end up overeating and gaining excess weight; they do not compensate for eating large amounts by eating less often.

Figure 2 illustrates the difference in eating frequency for children scoring high (5) and low (1) on food responsiveness. Children with high food responsiveness eat more frequently and as this increase in eating frequency is not compensated for by eating less each time, they could potentially end up overeating and gaining excess weight.



Figure 1. Average kJs consumed per eating occasion for children with the lowest satiety sensitivity score, as measured with the Child Eating Behaviour Questionnaire (score of 1) compared to those with the highest score (score of 5). The food images have been obtained from the Preschool Food Atlas.²⁵ The Atlas provides a range of age appropriate portion sizes to estimate food served and food leftover to children aged 1.5 to 4 years of age. The photo for the lowest satiety sensitivity score represents 18g of cheese (306 kJ), and the photo for the highest satiety sensitivity score represents 9g of cheese (149kJ), thus the difference in kJ between the two photos is 157kJ.

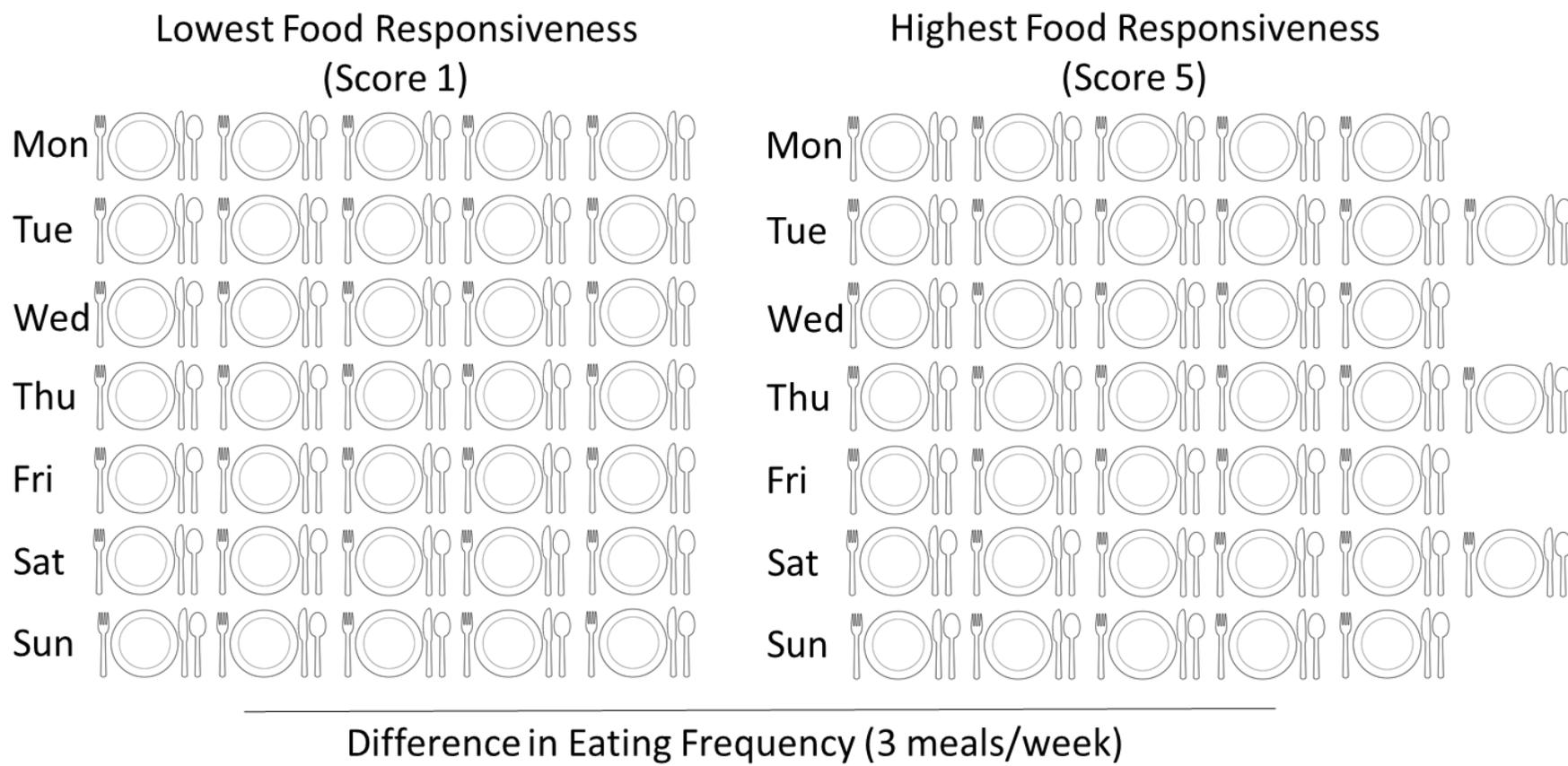


Figure 2. Average number of eating occasions per week for children with the lowest food responsiveness score, as measured with the Child Eating Behaviour Questionnaire (score of 1) compared to those with the lowest score (score of 5). Those with the highest food responsiveness scores consumed on average 3 meals more per week.

Preventing childhood obesity

Once weight is gained it is difficult to lose; the likelihood of an obese individual ever achieving a healthy weight is very low.²⁶ Given that 1 in 5 children are already overweight by the time they enter primary school at 4-5 years old, prevention in early life is crucial. In the current environment, a higher proportion of children are now overweight or obese, compared with previous years.^{27,28} This may be one of the reasons why parents do not recognise overweight or obesity in their children, even when they are provided with information about their child's weight status.²⁹

There are a number of government initiatives specifically targeting schools and school children. The Healthy Schools Programme³⁰ aims to increase awareness of the opportunities that exist in schools for improving health. A fundamental part of the Programme is the National Healthy School Standard, a national guidance and accreditation process to support the development of healthy schools through local educational and health partnerships.³¹ The UK National Child Measurement Programme measures the heights and weights of all children in the first (4-5 years) and last (10-11 years) years of primary school, and provides feedback to parents of children classified as overweight or obese as a means of promoting change. However, many parents are unconvinced, and maintain that their child is 'healthy'.³² It is crucial for parents to recognise overweight and obesity as a first step towards behaviour change.³³ Primary and community health professionals, including GPs, practice nurses, dieticians, health visitors and school nurses also have an important role in helping parents in recognising and managing childhood obesity.

What can GPs do?

Health professionals, and in particular, General Practitioners (GPs) have a key role to play in recognising and managing childhood obesity; but research has shown that they have difficulty assessing a child's weight status by sight.³⁴ It is therefore important that there is a structure in place within primary care teams to weigh and measure children, and use growth charts to determine if a child is overweight or obese (and not simply rely on judgement by sight). There are guidelines available to health professionals on the weight management of children and adolescents in primary care³⁵. However, whilst many GPs feel it is their role to raise the issue of a child's weight, they are often reluctant to weigh children or speak to parents about child weight, for fear of causing distress to parents and children.³⁶ It is important that GPs feel confident in broaching the subject with parents and children. One way in which this could be achieved is if it became routine for all children to be weighed during GP appointments, however an important consideration is that GPs' time is limited. Other health professionals such as school nurses could potentially take on some of the responsibility for monitoring children's weight. It might also be useful to ensure health professionals have access to advice on how to talk to parents about childhood weight, for example rather than focusing on weight and encouraging dieting or weight loss, framing it to be about health and the benefits of healthy eating and exercise.

Targeting eating behaviours

Evidence that genetic risk for obesity operates through appetitive mechanisms suggests that behavioural interventions that target over-responsiveness to food cues and low sensitivity to satiety might provide effective means for preventing and managing childhood

overweight. Strategies such as careful portion control and slow eating are already used to circumvent poor satiety sensitivity, and there may be other opportunities to blunt responsiveness to food cues, such as attention control or self-regulation training.

However, determining appropriate portion sizes for young children is not straightforward. Currently there is very little guidance on appropriate portion sizes for toddlers, and feeding frequency. The Infant and Toddler Forum³⁷ has developed recommendations on eating frequency and portion sizes for children aged one to four years, but these recommendations are averages and are based on the idea that that individual children have the ability to adjust their intake according to their energy needs. However, our research³⁸ suggests that children with poorer satiety sensitivity and increased food responsiveness are unable to self-regulate their intake, and are at risk of overeating. Parents may benefit from information about the behaviours that predispose children to overweight, so that they might put strategies in place to manage them.

Child feeding guidance for parents

Reducing young children's portion sizes or the number of meals and snacks that are offered might provide a means for limiting excessive weight gain for children at greater risk of obesity. If a child is food responsive, parents might benefit from advice and guidance from health professionals on how to reduce the number of times per day their child eats; or be provided with suggestions for 'healthier' snacks that are low in energy density and unlikely to contribute to excessive energy intake. Whereas a parent whose child does not seem to have good sensitivity to satiety (no 'off switch' once they have started eating), might need specific advice on limiting portion sizes, and having a 'no second helpings' policy. It has been suggested that tackling portion sizes at a policy level, for example reducing portion sizes in restaurants, is key to reducing obesity levels.³⁹ Indeed, these changes are important for tackling obesity at a population level, but they do not target individuals or address the gene-environment interactions at play. There are individual differences in eating behaviour; personalised guidance is therefore needed for families. For example, discussions around appropriate portion sizes and snacking behaviour might benefit parents of young children who are susceptible to overeating.

Reference List

1. Cole TJ. Growth monitoring with the British 1990 growth reference. *Arch Dis Child*. 1997;76:47–49.
2. Reilly JJ, Dorosty AR, Emmett PM, Alspac T, Team S. Identification of the obese child : adequacy of the body mass index for clinical practice and epidemiology. *Int J Obes*. 2000;24:1623–1627.
3. Kuczmarski, Robert J., Cynthia L. Ogden, Laurence M. Grummer-Strawn, Katherine M. Flegal, Shumei S. Guo, Rong Wei, Zuguo Mei, Lester R. Curtin, Alex F. Roche and CLJ. CDC growth charts: United States. *Adv Data*. 2000;314(1-27).
4. Cole, T. J., Bellizzi, M. C., Flegal, K. M., & Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ*. 2000;320 (7244):1240.
5. Han JC, Lawlor D a, Kimm SYS. Childhood obesity. *Lancet*. 2010;375(9727):1737–48. doi:10.1016/S0140-6736(10)60171-7.
6. Wang Y, Lobstein T. Worldwide trends in childhood overweight and obesity. *Int J Pediatr Obes*. 2006;1(1):11–25. doi:10.1080/17477160600586747.
7. <https://www.noo.org.uk/NCMP>.
8. Pulgarón ER. Childhood obesity: a review of increased risk for physical and psychological comorbidities. *Clin Ther*. 2013;35(1):A18–32. doi:10.1016/j.clinthera.2012.12.014.
9. Reilly JJ. Descriptive epidemiology and health consequences of childhood obesity. *Best Pract Res Clin Endocrinol Metab*. 2005;19(3):327–41. doi:10.1016/j.beem.2005.04.002.
10. Egan KB, Ettinger AS, Bracken MB. Childhood body mass index and subsequent physician-diagnosed asthma: a systematic review and meta-analysis of prospective cohort studies. *BMC Pediatr*. 2013;13(1):121. doi:10.1186/1471-2431-13-121.
11. Simmonds M, Llewellyn A, Owen CG, Woolacott N. Predicting adult obesity from childhood obesity : a systematic review and meta-analysis. *Obes Rev*. 2016;17(February):95–107. doi:10.1111/obr.12334.
12. Llewellyn A, Simmonds M, Owen CG, Woolacott N. Childhood obesity as a predictor of morbidity in adulthood a systematic review and meta-analysis. *Obes Rev*. 2016;17:56–67. doi:10.1111/obr.12316.
13. Reilly JJ, Kelly J. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: systematic review. *Int J Obes (Lond)*. 2011;35(7):891–8. doi:10.1038/ijo.2010.222.
14. Scarborough P, Bhatnagar P, Wickramasinghe KK, Allender S, Foster C, Rayner M. The economic burden of ill health due to diet , physical inactivity , smoking , alcohol and obesity in the UK : an update to 2006 – 07 NHS costs. *J Public Health (Bangkok)*. 2011;33(4):527–535. doi:10.1093/pubmed/fdr033.
15. Llewellyn CH, Jaarsveld CHM Van, Plomin R, Fisher A, Wardle J. Inherited behavioral susceptibility to adiposity in infancy : a multivariate genetic analysis of appetite and

- weight in the Gemini birth cohort 1 – 3. *Am J Clin Nutr.* 2012;95(3):633–639. doi:10.3945/ajcn.111.023671.Am.
16. Silventoinen K, Rokholm B, Kaprio J, Sørensen TI a. The genetic and environmental influences on childhood obesity: a systematic review of twin and adoption studies. *Int J Obes (Lond).* 2010;34(1):29–40. doi:10.1038/ijo.2009.177.
 17. Wardle J, Carnell S, Haworth CM, Plomin R. Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment. *Am J Clin Nutr.* 2008;87(2):398–404. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/18258631>.
 18. Swinburn BA, Sacks G, Lo SK, et al. Estimating the changes in energy flux that characterize the rise in obesity prevalence. *Am J Clin Nutr.* 2009;89:1723–1728. doi:10.3945/ajcn.2008.27061.1.
 19. Wardle J, Boniface D. Changes in the distributions of body mass index and waist circumference in English adults, 1993/1994 to 2002/2003. *Int J Obes (Lond).* 2008;32(3):527–32. doi:10.1038/sj.ijo.0803740.
 20. Carnell S, Wardle J. Appetite and adiposity in children: evidence for a behavioral susceptibility theory of obesity. *Am J Clin Nutr.* 2008;88(1):22–9. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/18614720>.
 21. van Jaarsveld, C. H., Llewellyn, C. H., Johnson, L., & Wardle J. Prospective associations between appetitive traits and weight gain in infancy. *Am J Clin Nutr.* 2011;94(6):1562–1567. doi:10.3945/ajcn.111.015818.Individual.
 22. van Jaarsveld CHM, Boniface D, Llewellyn CH, Wardle J. Appetite and growth: a longitudinal sibling analysis. *JAMA Pediatr.* 2014;168(4):345–50. doi:10.1001/jamapediatrics.2013.4951.
 23. Llewellyn CH, Jaarsveld CHM Van, Johnson L, Carnell S, Wardle J. Nature and nurture in infant appetite : analysis of the Gemini twin birth. *Am J.* 2010;91(12):1172–1179. doi:10.3945/ajcn.2009.28868.INTRODUCTION.
 24. Llewellyn, C. H., Van Jaarsveld, C. H., Boniface, D., Carnell, S., & Wardle J. Eating rate is a heritable phenotype related to weight in children. *Am J Clin Nutr.* 2008;88(6):1560–1566. doi:10.3945/ajcn.2008.26175.1.
 25. Foster, E., Hawkins, A., & Adamson A. *Young person's food atlas—pre-school.* London, UK: Food Standards Agency, 2010.; 2010.
 26. Fildes A, Charlton J, Rudisill C, Littlejohns P, Prevost AT, Gulliford MC. Probability of an Obese Person Attaining Normal Body Weight : Cohort Study Using Electronic Health Records. *Am J Public Health.* 2015;105(9):54–59. doi:10.2105/AJPH.2015.302773.
 27. Jaarsveld CHM Van, Gulliford MC. Childhood obesity trends from primary care electronic health records in England between 1994 and 2013 : population-based cohort study. *Arch.* 2015;0:1–6. doi:10.1136/archdischild-2014-307151.
 28. England PH. *Health Survey for England: Health, social care and lifestyles.*; 2014. Available at: <http://www.hscic.gov.uk/catalogue/PUB19295/HSE2014-Sum-bklet.pdf>.
 29. Falconer CL, Park MH, Croker H, et al. The benefits and harms of providing parents with weight feedback as part of the national child measurement programme : a prospective cohort study. *BMC Public Health.* 2014;14:549–559.

30. Agency HD. Healthy schools programme. Wired for Health. 2002. Available at:
Available from:<http://www.wiredforhealth.gov.uk/healthy/healint.html>.
31. Department of Health. National healthy school standard. Dept of Health. 2002.
Available at: Available from:<http://www.wiredforhealth.gov.uk/healthy/healsch.html>.
32. Syrad H, Falconer C, Cooke L, et al. "Health and happiness is more important than weight": a qualitative investigation of the views of parents receiving written feedback on their child's weight as part of the National Child Measurement Programme. *J Hum Nutr Diet*. 2014;n/a–n/a. doi:10.1111/jhn.12217.
33. Falconer C, Park M, Skow Á, et al. Scoping the impact of the national child measurement programme feedback on the child obesity pathway : study protocol. *BMC Public Health*. 2012;12:783. doi:10.1186/1471-2458-12-783.
34. Ahlers-schmidt CR, Ph D, Kroeker D, et al. Visual Recognition of Child Body Mass Index by Medical Students , Resident Physicians , and Community Physicians. *Kansas J Med*. 2010;3(5):7–14.
35. Gibson P, Edmunds L, Haslam DW, Poskitt E. *An Approach to Weight Management in Children and Adolescents (2-18 years) in Primary Care.*; 2002. Available at:
<http://www.localtransport.dft.gov.uk/schooltravel/respack/index.htm>.
36. van Gerwen M, Franc C, Rosman S, Le Vaillant M, Pelletier-Fleury N. Primary care physicians' knowledge, attitudes, beliefs and practices regarding childhood obesity: A systematic review. *Obes Rev*. 2009;10(2):227–36. doi:10.1111/j.1467-789X.2008.00532.x.
37. <https://www.infantandtoddlerforum.org/>.
38. Syrad H, Johnson L, Wardle J, Llewellyn CH. Appetitive traits and food intake patterns in early life. *Am J Clin Nutr*. 2015;(C):2–6. doi:10.3945/ajcn.115.117382.
39. Marteau TM, Hollands GJ, Shemilt I, Jebb S a. Downsizing: policy options to reduce portion sizes to help tackle obesity. *Bmj*. 2015;5863(December):h5863. doi:10.1136/bmj.h5863.