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PAPER

Food and activity preferences in children of lean and obese parents

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BACKGROUND: Children of obese parents have a substantially higher risk of adult obesity than children of lean parents. Adoption and twin studies have shown that this risk is largely genetic but the proximal mechanisms of the genetic risk are not known. Comparisons of energy intake or expenditure in children of obese and lean parents have produced mixed, but generally negative results. An alternative hypothesis is that the early expression of obesity risk is through food and activity preferences, which provides a basis for later weight gain. The aim of this study was therefore to compare food and activity preferences in a large sample of young children from obese and lean families using parental obesity as a marker of the obesity-risk phenotype. Because the children from the families with obese parents were not yet overweight, differences observed in the two types of families are more likely to be causes than effects of obesity.
METHODS: A total of 428 children aged 4 – 5 y, whose parents were either obese/overweight or normal-weight/lean were selected from a population sample of families with twin births. Food and activity preferences were assessed with a combination of food intake and taste tasks, and questionnaires completed by the mother during a home visit.
FINDINGS: Children from the obese/overweight families had a higher preference for fatty foods in a taste test, a lower liking for vegetables, and a more 'overeating-type' eating style. They also had a stronger preference for sedentary activities, and spent more time in sedentary pastimes. There were no differences in speed of eating or reported frequency of intake of high-fat foods.
CONCLUSION: Part of the process whereby a genetic risk of obesity is transmitted to the next generation could be through differences in diet and activity preferences, which would place susceptible individuals at risk of positive energy balance in the permissive nutritional environment of industrialised countries today.
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Introduction

Children of obese parents have more than 5 times the risk of children of normal-weight parents of becoming obese in adult life.[1] Investigations of children adopted at birth have demonstrated that familial risk for weight is largely genetic, finding little or no correlation between the body weights of adopted children and the weights of their adoptive parents, compared with a correlation with the weights of their biological parents that is as high as the correlation with parents rearing their own children.[2] The heritability of weight has also been consistently confirmed in twin studies, with heritability estimates exceeding 70%.[3] Although there are few genetic studies of overweight or obesity *per se*, these studies also indicate substantial genetic influence, including analyses of both weight and overweight in the present sample of young twins.[4] Parental obesity can therefore be used as a marker of a higher genetic risk of obesity for young children who are not yet overweight, providing the opportunity to characterise the obesity-risk phenotype before the situation is complicated by the multitude of biological, psychological and social consequences of obesity.

Now that the role of genetic factors in obesity is widely accepted, research has turned towards understanding the mechanisms by which genotypes become phenotypes.[5] Several investigators have used comparisons between children of normal-weight and overweight parents to study energy balance, predicting that in high-risk groups, either energy expenditure must be low (and intake not commensurately

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low) or energy intake must be high (without high energy expenditure). Unfortunately the results have been inconsistent. The early observations of lower energy expenditure in infants and children of obese parents[6–8] have not been confirmed in more recent studies with larger samples.[9–12] Results for energy intake have been similarly disappointing, with most studies finding no differences in intake,[10,12] although there have been suggestions that the percentage of energy intake from fat might be higher in high-risk children.[13–15] Why should groups of children who can be reliably predicted to store more fat in the long term seemingly not have a more positive energy balance? Part of the explanation for this apparently paradoxical finding could be that the energy balance differences needed to underpin the differing weight trajectories of high- and low-risk children are extremely small and only have a cumulative effect over many years. Hence they are hard to detect without highly reliable measures, repeated sampling and large sample sizes.

A more sensitive index of the way in which the high-risk genotype might result in the obese phenotype is through preferences for sedentary over vigorous activities, or for higher-fat over lower-fat foods. The behavioural expression of such preferences would almost certainly be moderated by environmental factors, parenting styles, and other psychosocial factors, which would mean that consistent differences in energy intake or energy expenditure might be difficult to detect. Few studies have taken this psychological perspective and examined food or activity preferences in relation to obesity risk, even though these should be easier to detect than differences in energy balance, and might be more informative about the processes leading to the development of obesity. There are some promising indications: children's preference for fat has been found to be correlated with parental body mass index (BMI).[15] Avidity of sucking (indexing a food-responsive eating style) has been found to be higher in infants of overweight than normal-weight parents.[11,16] Other studies have linked fussiness and slowness in eating with underweight.[17] Related concepts have been tested in the context of behavioral-economic studies.[18,19] However, sample sizes have tended to be small and there is a strong likelihood of a 'file drawer' effect in publication of findings of this kind.

The present study was therefore designed to assess food preferences, eating style and activity preferences, in a large sample of young children from families with obese/overweight or normal-weight parents. Because the children from the families with obese/overweight parents were not yet overweight, differences observed in the two types of families are more likely to be causes than effects of obesity. Children from the families with obese/overweight parents (high-risk children) were predicted to prefer higher fat foods, to show stronger responsiveness to food cues, to have lower satiety sensitivity, and to prefer sedentary activities. The participants for this study were selected from a large and representative twin sample so that future analyses can test the hypothesis that the phenotypic associations established in the present study are mediated genetically. In addition, the twin sample is being followed up longitudinally so that eventually it will be possible to add the power of longitudinal analyses to help sort out cause and effect in the development of obesity.

Methods

Study population and procedures

Participants were 428 twin children; 200 from families with overweight/obese parents and 228 from families with normal-weight/lean parents. There is no obvious reason to suppose that twins should be different from other siblings in the way that genetic risk of obesity is expressed. A recent study comparing twins and singletons found little mean difference in BMI.[32] The families were drawn from the Twins Early Development Study (TEDS) which includes 10 000 pairs of twins born in England and Wales in 1994 and 1995, representing more than half of all twins born in those years.[20] The TEDS sample has been shown to be reasonably representative of UK families with young children in relation to parental education and occupation.[20] On the basis of their self-reported weights and heights in 1997, 31% of mothers in the TEDS sample and 48% of fathers had a BMI over 25, with 9% of mothers and 10% of fathers having a BMI over 30. These are slightly lower than the 1997 UK population figures for this age group,[19] which indicates that 14.5% of women and 13.1% of men were obese, but self-reports are known to substantially under-estimate BMI. The overweight/obese families (which we henceforward refer to as the 'obese families') were selected for the present study by identifying families with same-sex twins in which the mother's reported BMI was at least 28.5 (and therefore the true BMI could be expected to be higher) and the father's reported BMI was at least 25. Normal weight/lean families (which we refer to as 'lean families') in which both parents' BMI was less than 25 were selected to come from the same areas of the country, and to provide an approximate match in terms of social class, for which we used paternal occupation as an indicator.

In all, 231 families were contacted by letter and telephone and invited to participate in the study of children's eating habits. Of these, 214 families (with 428 twin children) agreed to take part. When the children were 4 or 5-y-old, families were visited in their home, where mothers and children were weighed and measured, mothers completed questionnaire instruments to assess their children's eating and activity habits and preferences, children did a taste preference task, and children's intake in a test snack was assessed.

Measures

Anthropometric. Maternal and paternal weights were reported at the time that parents completed other questionnaires about themselves and their children, when the children were aged 3. Mother's and children's weights and

heights were measured at the time of the home visit using a Sohnle electronic scale and a tape measure. BMI was calculated in the usual manner for parents (kg/m^2) and for children.[10]

Children's percentage of fat and lean tissue was assessed using bioelectrical impedance analysis (Maltron BF-906 Body Fat Analyser).

Zygosity information was obtained from a parent-report questionnaire,[20] with uncertain zygosity pairs (about 5%) diagnosed using DNA analysis.[22,23]

Children's food preferences. Maternal ratings of children's liking for meats, sweet desserts, fruits and vegetables were derived from a factor analysis of ratings of a list of 95 common foods.[24]

Fat preferences were assessed by asking children to taste and rank order their liking for six foods. Three were high fat and respectively sweet, savoury and bland (chocolate, cheese, butter biscuits) and three were low fat and also sweet, savoury and bland (jelly babies, carrots, ryvita, see Appendix). Average ranked preference for the three higher fat foods was calculated to index fat preference. Details about this method, which has been shown to be reliable in this age group, have been reported previously.[25,26]

Children's eating style. Eating style was assessed with the Children's Eating Behaviour Questionnaire (CEBQ). The CEBQ is a parent-rated instrument incorporating scales for positive reactions to foods (food responsiveness; emotional overeating; desire for drinks (in practice mostly sweetened drinks); enjoyment of food) and negative reactions to foods (satiety sensitivity; fussiness; slowness) with good internal validity and reliability.[27]

Children's food intake. Frequency of intake of high-fat, low-fat, high-fibre and low-fibre foods was assessed from a Food Frequency Questionnaire designed for British children.[28] Meal size was estimated from mothers' ratings of children's preferred portion sizes from sets of photographs of three different portion sizes for each of 12 common foods and these were averaged to estimate typical portion size ($\alpha = 0.73$).

Intake of palatable foods under conditions of satiety was assessed by offering the children three popular varieties of biscuit in a 'test snack', carried out within 1 h of having eaten a meal. Weight of biscuits eaten (weighed with Tanita 1479 scales) was used as a behavioural index of intake under conditions of satiety.

Children's activity preferences. Mothers rated their children's enjoyment of a selection of high and low impact physical activities.[29] Mean enjoyment scores were calculated for each group. Time spent in sedentary activities (computer games, TV watching) was averaged over weekdays and weekends.[30]

Mothers rated their children's overall activity level, identifying each child on a scale from 'much more active' to 'much less active' than other children of their age.[31]

Analysis

Mean differences between the two groups were analysed using *t*-tests. The inclusion of two children from the obese and lean families is unlikely to have much effect on this analysis of mean comparisons between the obese and lean families. Analyses conducted for just one child per family yield a similar pattern of results but in the interest of increased power we present analyses for the entire sample.

Results

Demographic and anthropometric differences

Parental age, occupation and education for the two groups of parents are shown in Table 1. There were no differences between the groups in mother's or father's age. As expected, since the sample was drawn to be matched for social class, there was no difference between the groups in father's socioeconomic status (SES; shown in Table 1 as manual *vs* non-manual occupation). There was also no difference in the educational level of the mothers, which was classified as having no more than the minimum qualifications in the English educational system (no qualifications, GCSEs, CSEs or O-levels) *vs* any higher level of qualification (A-level or higher). Reported BMIs for mothers in the two groups differed by more than 3 standard deviations, and for fathers by more than 2 standard deviations. As expected, mothers' BMI calculated from measured weight and height was substantially higher than their BMI based on reported height and weight (obese group, 36.0 *vs* 33.7; lean group, 23.3 *vs* 22.3), and the difference was greater for obese mothers, although there was a very high correlation between the two values ($r = 0.93$, $P < 0.001$). It is likely that father's true BMI was also under-estimated by self-reports, but fathers were not weighed.

Table 1 Demographic and anthropometric characteristics of the parents (percentages or means and standard deviations)

	'Lean' families (n = 114)	*'Obese' families (n = 100)*	*Difference (P)*
Mother's (reported) BMI	22.30 (1.69)	33.74 (3.72)	0.000
Mother's measured BMI	23.28 (2.33)	36.00 (4.59)	0.000
Father's (reported) BMI	22.93 (1.3)	29.23 (3.17)	0.000
Mother's age	34.88 (3.93)	34.78 (4.67)	0.81
Father's age	38.26 (5.01)	38.56 (5.34)	0.78
Father's occupation:			
Manual	46%	46%	0.97
Non-manual	54%	54%	
Mother's education:			
O-levels or less	65%	71%	0.34
A-levels or more	35%	29%	

On the basis of measured BMI, one of the obese mothers was no longer obese (but was still overweight) and 26 of the normal-weight mothers had a BMI slightly over 25. Group comparisons were carried out on the basis of the original allocations, but also checked after excluding families where the group allocation would be changed. As the differences were minimal, results are reported from the original groupings.

As shown in Table 2, age, gender and zygosity distributions were similar in high-risk and low risk children. Weight and BMI were higher in the children of obese than lean families, but the difference in percentage body fat was not significant. The effect size of the BMI difference was much smaller in the children than in the parents and few children were overweight. The small BMI difference between children in the two groups as compared to the large BMI difference between their parents implies that parent–offspring resemblance for young children is modest even at the extremes of parental weight. Because the parents in this sample are highly selected for weight, it is not appropriate to calculate parent–offspring correlations within these groups. Within the entire TEDS sample, the correlation between reported BMI of mothers and their offspring at 4 y is also modest ($r = 0.16$).

Food preferences

There were small differences in food preferences, both in the taste test and the preference record. As predicted, high-risk children ranked the higher-fat foods more highly (closer to one) in the food preference task ($t = 1.92$, $P = 0.06$). High risk children had lower liking for vegetables on the basis of their mother's reports ($t = 2.3$, $P = 0.02$), but there were no group differences in mothers' ratings of children's liking for protein foods, sweet desserts or fruits (see Table 3).

Table 2 Anthropometric characteristics of the children (percentages or means and standard deviations)

	Children from 'lean' families (n = 228)	*Children from 'obese' families (n = 200)*	*Difference (P)*
Age	4.43 (0.33)	4.42 (0.36)	0.92
Gender			
Girls	49%	55%	0.20
Boys	51%	45%	
Zygosity			
DZ	50%	54%	0.52
MZ	50%	46%	
Weight (kg)	17.53 (2.48)	18.30 (2.81)	0.003
Height (cm)	104.75 (4.78)	104.90 (5.28)	0.75
BMI	15.93 (1.48)	16.56 (1.71)	0.000
Percentage body fat	19.50 (4.55)	20.08 (4.95)	0.22

Eating style (CEBQ)

Children from obese families had slightly higher scores on three of the four positive styles: food responsiveness ($t = 1.97$, $P = 0.05$); overeating in response to emotional cues ($t = 1.77$, $P = 0.08$); and desire for drinks ($t = 2.6$, $P = 0.009$). There were no differences in any of the negative styles, nor any difference in enjoyment of foods (see Table 4).

Food intake

There were no group differences in frequency of intake of high-fat, low-fat, high-fibre or low-fibre foods. High- and low-risk children were rated as selecting similar-sized food portions. Both groups ate around 35 g of biscuits in the test snack, with no differences between high- and low-risk children.

Activity preferences

The children of obese families were rated as enjoying low-impact (sedentary) activities more ($t = 2.06$, $P = 0.04$) and being less active than other children ($t = 2.37$, $P = 0.02$) (see Table 5). They also spent more hours at the computer ($t = 3.2$, $P = 0.001$) and watching TV ($t = 5.9$, $P < 0.001$). There were no differences in enjoyment of high impact activities.

Table 4 Children's eating style — scores on the Children's Eating Behaviour Scale (means and standard deviations)

	Children from 'lean' families (n = 228)	*Children from 'obese' families (n = 200)*	*Difference (P)*
Food responsiveness	2.42 (0.73)	2.57 (0.80)	0.05
Emotional over-eating	1.79 (0.52)	1.88 (0.52)	0.08
Desire for drinks	2.66 (1.03)	2.93 (1.04)	0.009
Enjoyment of foods	3.73 (0.73)	3.70 (0.77)	0.72
Slowness	2.90 (0.74)	2.92 (0.71)	0.82
Satiety sensitivity	2.92 (0.62)	2.98 (0.65)	0.31
Fussiness	2.83 (0.80)	2.94 (0.82)	0.16

Table 3 Children's taste preferences (means and standard deviations)

	Children from 'lean' families (n = 228)	*Children from 'obese' families (n = 200)*	*Difference (P)*
Ranking of fat foods in taste test, (1 = high to 6 = low)	3.31 (0.67)	3.19 (0.66)	0.06
Liking for vegetables (1 = low to 5 = high)	2.98 (0.97)	2.76 (0.99)	0.02
Liking for meat and fish	3.54 (0.90)	3.55 (0.91)	0.87
Liking for desserts	3.80 (0.90)	3.81 (0.81)	0.93
Liking for fruit	4.04 (0.89)	3.97 (0.88)	0.40

Table 5 Children's activity levels (means and standard deviations)

	Children from 'lean' families (n = 228)	*Children from 'obese' families (n = 200)*	*Difference (P)*
Activity rating (1, less active; 5, more active)	3.41 (0.75)	3.24 (0.67)	0.02
Enjoyment of low impact activities (1, low; 4, high)	2.80 (0.47)	2.90 (0.50)	0.04
Time watching TV (hours per week)	11.21 (5.57)	14.49 (5.55)	0.000
Time playing computer (hours per week)	1.19 (2.43)	2.15 (3.51)	0.001
Enjoyment of high impact activities	3.03 (0.58)	2.95 (0.65)	0.16

Discussion

This study is one of the largest to date to investigate appetite and activity preferences in children at risk of obesity. The results suggest that higher-risk children show modestly higher preferences for the taste of fatty foods, like foods in the lowest energy-density group (vegetables) less, and show stronger positive appetitive reactions to food and drink. They also show a much stronger preference for sedentary activities. These results are consistent with existing data on obese children and adults, which indicate that they tend to prefer high-fat, energy-dense foods,[33] like vegetables less,[34,35] are more responsive to food cues,[36] are more likely to overeat in negative emotional states[37] and are more inactive.[38] Hitherto, it has not been possible to be sure if these characteristics pre-dated the obesity problem and therefore might be causally related, or were consequences either of the obesity itself or of the steps that the obese person might take in trying to control their weight (eg strict dieting). The present results are consistent with the hypothesis that they play a causal role, although as discussed later, multivariate genetic analyses and longitudinal analyses will strengthen this hypothesis.

The appetite profile of the higher risk children (greater preference for fat, more responsive to food cues) might be benign, or even advantageous, when food supplies are low, but would increase the risk of overeating when foods are as plentiful and palatable as they are in Western industrialised countries today. It might also be less risky if matched by a desire for high levels of physical activity, but these results suggest the opposite — the high-risk children preferred sedentary activities and spent more time engaged in sedentary pastimes, as indicated both by time spent at the TV and computer, and parent ratings of activity levels.

In the present results, as in most of the recent findings on intake, there were no differences in reported intake of any food group, nor were there differences in intake during the snack task, nor differences in mother's rating of the child's preferred portion size. As discussed above, differences in intake need only be very small, and intake is notoriously hard to assess, so any conclusions about food intake must be cautious, but within the limitations of the methods used to measure food intake (that is, the use of parent reports and a snack task), there was no evidence that children of high-risk parents were actually eating different amounts of food. These results raise the question of why the high-risk children in this sample did not, as a consequence of the differences in preferences or eating style, eat more. One explanation may be that the characteristics measured in this study represent behavioural tendencies, and their expression will depend upon the permissiveness of the environment. If, for example, meals and activities are largely determined by parents and schools, then there might be little difference in intake between high and low risk groups. This hypothesis would predict that the phenotypic correlation between these behavioural tendencies and children's weight gain is mediated environmentally. Environmental mediation of the association between behavioural tendencies and children's weight gain is possible — genetic research shows that environmental as well as genetic factors are involved. If confirmed, this hypothesis could suggest features of the environment that might profitably be modified to prevent weight gain.

Another, not mutually exclusive, hypothesis is that genetic factors mediate the association between these behavioural tendencies and obesity. This genetic hypothesis would predict that these behavioural tendencies are heritable and that their relationship to weight and weight gain is mediated genetically. In line with this hypothesis, recent twin studies indicate that genetic factors affect many aspects of eating such as the number, timing and composition of meals as well as degree of hunger and sense of fullness after eating.[39–41] Multivariate genetic analysis that decomposes the phenotypic covariance between variables into genetic and environmental contributions to covariance can be used to address these issues.[42] We are currently applying multivariate genetic analyses to these data to investigate the genetic and environmental origins of the links between behavioural propensities and weight. The children in the present sample will also be followed up, both to confirm that weight gain is higher in the children from the obese families, and to test the prediction arising from the present results, that children who have higher fat and lower activity preferences will eventually gain more weight.

If behavioural risk factors were found to mediate genetic influences on eating and weight, it might point to new behavioural interventions that could disrupt the pathways between genes and obesity. Finding genetic markers predicting obesity risk more precisely could facilitate environmental and behavioural interventions targeted to children most likely to profit from them.

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References

1 Lake JK, Power C, Cole TJ. Child to adult body mass index in the 1958 British birth cohort: associations with parental obesity. *Arch Dis Child* 1997; **77**: 376–381.
2 Grilo CM, Pogue-Guile MF. The nature of environmental influences on weight and obesity: a behavior genetic analysis. *Psychol Bull* 1991; **110**: 520–537.
3 Maes HH, Neale MC, Eaves LJ. Genetic and environmental factors in relative body weight and human adiposity. *Behav Genet* 1997; **27**: 325–351.
4 Koeppen-Schomerus G, Wardle, J, Plomin R. A genetic analysis of weight and overweight in four year old twin pairs. *Int J Obes Relat Metab Disord* 2001; **25**: 838–844.
5 Faith MS, Johnson SL, Allison DB. Putting the behavior into the behavior genetics of obesity. *Behav Genet* 1997; **27**: 423–439.
6 Griffiths M, Payne PR. Energy expenditure in small children of obese and non-obese parents. *Nature* 1976; **260**: 698–700.
7 Roberts SB, Savage J, Coward WA, Chew B, Lucas A. Energy expenditure and intake in infants born to lean and overweight mothers. *New Engl J Med* 1988; **318**: 461–466.
8 Freymond D, Larson K, Bogardus C, Ravussin E. Energy expenditure during normo- and overfeeding in peripubertal children of lean and obese Pima Indians. *Am J Physiol* 1989; **257**: E647–653.
9 Davies PS, Wells JC, Fieldhouse CA, Day JM, Lucas A. Parental body composition and infant energy expenditure. *Am J Clin Nutr* 1995; **61**: 1026–1029.
10 Goran MI, Carpenter WH, McGloin A, Johnson R, Hardin JM, Weinsier RL. Energy expenditure in children of lean and obese parents. *Am J Physiol* 1995; **268**: E917–924.
11 Stunkard AJ, Berkowitz RI, Stallings VA, Schoeller DA. Energy intake, not energy output, is a determinant of body size in infants. *Am J Clin Nutr* 1999; **69**: 524–530.
12 Treuth MS, Butte NF, Wong WW. Effects of familial predisposition to obesity on energy expenditure in multiethnic prepubertal girls. *Am J Clin Nutr* 2000; **71**: 893–900.
13 Eck LH, Klesges RC, Hanson CL, Slawson D. Children at familial risk for obesity: an examination of dietary intake, physical activity and weight status. *Int J Obes Relat Metab Disord* 1992; **16**: 71–78.
14 Nguyen VT, Larson DE, Johnson RK, Goran MI. Fat intake and adiposity in children of lean and obese parents. *Am J Clin Nutr* 1996; **63**: 507–513.
15 Fisher JO, Birch LL. Fat preferences and fat consumption of 3- to 5-year-old children are related to parental adiposity. *J Am Diet Assoc* 1995; **95**: 759–764.
16 Milstein RM. Responsiveness in newborn infants of overweight and normal weight parents. *Appetite* 1980; **1**: 65–74.
17 Douglas JE, Bryon M. Interview data on severe behavioural eating difficulties in young children. *Arch Dis Child* 1996; **75**: 304–308.
18 Epstein LH. Application of behavioral economic principles to treatment of childhood obesity. In: Allison DB (ed). *Obesity treatment: establishing goals, improving outcomes, and reviewing the research agenda*. Plenum Press: New York; 1995. pp 113–119.
19 Epstein LH, Saelens BE. Behavioral economics of food intake: food intake and energy expenditure. In: Bickel WK, Vuchinich RE (eds). *Reframing health behavior change with health economics*. Lawrence Erlbaum: Mahwah, NJ; 2000. pp 113–119.
20 Dale PS, Simonoff E, Bishop DV, Eley TC, Oliver B, Price TS *et al*. Genetic influence on language delay in two-year-old children. *Nature Neurosci* 1998; **1**: 324–328.
21 Department of Health. *Health survey for England 1998: cardiovascular disease*. Stationery Office: London; 1999.
22 Goldsmith, HH. A zygosity questionnaire for young twins: a research note. *Behav Genet* 1991; **21**: 257–269.
23 Freeman B, Powell J, Ball D, Hill L, Craig I, Plomin R. DNA by mail: an inexpensive and noninvasive method for collecting DNA samples from widely dispersed populations. *Behav Genet* 1997; **27**: 251–257.
24 Wardle J, Sanderson S. Food preferences in children. *Appetite* 2000; **35**: 213 (abstract).
25 Birch LL, Sullivan SA. Measuring children's food preferences. *J Sch Health* 1991; **61**: 212–214.
26 Guthrie CA, Rapoport L, Wardle, J. Assessing young children's food preferences: a comparison of three methods. *Appetite* 2000; **35**: 73–77.
27 Wardle J, Guthrie CA, Sanderson S, Rapoport L. Development of the children's eating behaviour questionnaire. *J Child Psychol Psychiat* (in press).
28 Hammond J, Nelson M, Chinn S, Rona RJ. Validation of a food frequency questionnaire for assessing dietary intake in a study of coronary heart disease risk factors in children. *Eur J Clin Nutr* 1993; **47**: 242–250.
29 Epstein LH, Valoski A, Wing RR, Perkins KA, Fernstrom M, Marks B *et al*. Perception of eating and exercise in children as a function of child and parent weight status. *Appetite* 1989; **12**: 105–118.
30 Taras HL, Sallis JF, Patterson TL, Nader PR, Nelson JA. Television's influence on children's diet and physical activity. *J Dev Behav Pediatr* 1989; **10**: 176–180.
31 Whincup P, Cook D, Papacosta O, Walker M, Perry I. Maternal factors and development of cardiovascular risk: evidence from a study of blood pressure in children. *J Hum Hypertens* 1994; **8**: 337–343.
32 Pietilainen KH, Kaprio J, Rissanen A, Winter T, Rimpela A, Viken RJ *et al*. Distribution and heritability of BMI in Finnish adolescents aged 16 y and 17 y: a study of 4884 twins and 2509 singletons. *Int J Obes Relat Metab Disord* 1999; **23**: 107–115.
33 Mela DJ, Sacchetti DA. Sensory preferences for fats: relationships with diet and body composition. *Am J Clin Nutr* 1991; **53**: 908–915.
34 Vereecken C, Maes L. Food consumption and physical activity in overweight children. [abstract]. *Int J Obes Relat Metab Disord* 2000; **24**(Suppl 1): S127.
35 Muller MJ, Koertringer I, Mast M, Languix K, Frunch A. Physical activity and diet in 5 to 7 year old children. *Public Health Nutr* 1999; **2**: 443–444.
36 Braet C, van Strien T. Assessment of emotional, externally induced and restrained eating behaviour in nine to twelve-year-old obese and non-obese children. *Behav Res Ther* 1997; **35**: 863–873.
37 Ganley RM. Emotion and eating in obesity: A review of the literature. *Int J Eat Disord* 1989; **8**: 343–361.
38 Jebb SA, Moore MS. Contribution of a sedentary lifestyle and inactivity to the etiology of overweight and obesity: current evidence and research issues. *Med Sci Sports Exerc* 1999; **31**(11 Suppl): S534–541.
39 Faith MS, Rha SS, Neale MC, Allison DB. Evidence for genetic influence on human energy intake: results from a twin study using measured observations. *Behav Genet* 1999; **29**: 145–154.
40 De Castro JM. Behavioral genetics of food intake regulation in free-living humans. *Nutrition* 1999; **15**: 550–554.
41 De Castro JM. Heritability of hunger relationships with food intake in free-living humans. *Physiol Behav* 1999; **67**: 249–258.
42 Plomin R, DeFries JC, McClearn GE, McGuffin P. *Behavioral genetics*, 4th edn. Worth: New York; 2001.

Appendix

Table A1 Composition of foods used in the taste test (g/100 g)

Food	*Fat*	*Carbohydrate*	*Protein*
Ryvita (lower fat, savoury, salty)	1.6	63.3	9.4
Carrot (lower fat, savoury, bland)	0.5	6.0	0.7
Jelly baby (lower fat, sweet)	0.0	79.5	4.0
Cheese (higher fat, savoury, salty)	34.4	0.1	25.0
Butter puff (higher fat, savoury, bland)	26.5	60.7	10.4
Chocolate (higher fat, sweet)	29.4	56.8	7.8