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Symposium on 'Treatment of obesity'

Understanding the aetiology of childhood obesity: implications for treatment

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Childhood obesity poses one of the greatest challenges to paediatric health in the 21st century. Developing effective strategies for treatment and prevention is therefore a priority for clinical medicine and public health. This process is taking place at a time of unprecedented change in the understanding of the role of genetic factors in human health and disease, and genetic research into obesity has challenged assumptions about causal processes. In the spirit of the conference theme, the present paper will discuss the implications of the understanding of genetic and environmental influences on obesity for the development of effective behavioural treatments.

Childhood obesity: Genetic factors: Environment: Twin studies: Eating behaviour

Prevalence and health risks of obesity in childhood

Clinical research into childhood obesity is uncovering more and more evidence of the threat posed by excess body fat stores to health and well-being. Obese children have higher blood pressure, serum cholesterol and insulin resistance, pointing to a raised risk of CVD (Goran *et al.* 2003). They are also more likely to have sleep difficulties, asthma and a range of other medical difficulties (Wing *et al.* 2003; Bibi *et al.* 2004). Adverse psycho-social consequences pose an additional burden. Clinical case studies show markedly lower self esteem and higher risk of depression (McElroy *et al.* 2004), and although data from community samples indicate that this outcome is partly a self-selection bias (Lamertz *et al.* 2002), almost all obese children have experiences of teasing, social exclusion, discrimination and prejudice (Puhl & Brownell, 2001; Strauss & Pollack, 2003). Long-term follow-up studies also show poorer educational and social outcomes for obese children compared with normal-weight children, even after matching for childhood socio-economic status and intelligence (Gortmaker *et al.* 1993). The more that is learned about the consequences for health and quality of life of childhood obesity, the stronger becomes the case for investment in clinical research and service provision.

The fact that children tend to maintain their relative BMI position as they mature means that the heaviest children are likely to become obese adults. Analyses of cohort data from the 1958 British Birth Cohort (Lake *et al.* 1997), the Framingham Study population (Fuentes *et al.* 2003) or the National Longitudinal Study of Youth (McTigue *et al.* 2002) confirm that the risk of adult obesity is raised many times by obesity in childhood. Obese children therefore stand to develop, in adulthood if not earlier, all the diseases that are associated with long-standing obesity. Obesity beginning in childhood may even confer additional risks to health over obesity beginning in adulthood (Must *et al.* 1992; Power *et al.* 1997), and early-onset obesity is associated with a worse body image and a higher risk of binge eating (Wardle *et al.* 2002*b*). In terms of limiting future health costs, investment in clinical research is certain to repay the investment.

Alarming findings are coming from analyses of trends in obesity prevalence in childhood, which indicate that an obesity epidemic is now apparent even in the youngest groups. If at-risk levels of childhood adiposity are defined

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in terms of exceeding the age-matched BMI thresholds associated with adult obesity (Cole *et al.* 2000), then currently approximately 6% of 6–9-year-olds in the UK should be defined as 'obese', which is almost double the 1990 value. Moreover, assuming that UK trends follow those in North America, this percentage is likely to rise further (Hedley *et al.* 2004). Most analyses suggest that there are two distinct processes behind the population trends. Overall, adiposity is rising, and therefore obesity rates will inevitably increase as higher proportions of the population exceed the BMI threshold for obesity. There also appears to be a greater-than-expected increase in the highest levels of BMI, suggesting that susceptible sub-groups are reacting more strongly to the environmental changes (Thomsen *et al.* 1999; Heude *et al.* 2003; Sturm, 2003).

There is a reasonable consensus on the likely suspects for the rising adiposity: environmental changes that have made food more palatable, accessible and plentiful, combined with technological changes that have reduced the need for physical activity. Together, these factors produce ever-stronger pressures towards positive energy balance (Jeffery & Utter, 2003; Prentice & Jebb, 2003). There is less agreement on how to modify these aspects of the environment in order to decrease the impact on human health, and it may prove hard to achieve the political, industrial and public support to make the substantial changes that will be needed (Peters *et al.* 2002; Brownell, 2004). However, the likely economic burden, both in terms of loss of productivity and direct health costs, of a continuing increase in obesity is so serious that the tide of opinion is starting to change (Thompson & Wolf, 2001; Sturm, 2002).

The clinical problem of obesity is rapidly moving from a rare to a common paediatric condition. Millions of children worldwide are having their life chances compromised by excess body fat and the numbers will increase unless the political will can be found to modify the environment. For those children who are already overweight and obese there is an urgent need to develop effective treatments, and to achieve this objective it is necessary to have a better understanding of the processes that lead to a persistent positive energy balance.

The causes of obesity (why are some children fatter than others?)

Gaining weight is a deceptively simple process; it merely involves taking in more energy than is expended. However, underlying this apparent simplicity is the much more challenging question of why some individuals find themselves consistently in this state of positive energy balance, while others balance their intake against their expenditure to maintain a stable weight.

The best predictor of obesity in a child is obesity in family members; it is well-known that obesity tends to run in families. Quantitative genetics has made great strides in unravelling the extent to which familial resemblance is a result of shared genes or shared environments. Family studies reveal that BMI similarities largely parallel genetic relatedness (Grilo & Pogue-Geile, 1991; Allison *et al.* 1996). Sharing genes, more than sharing homes, is what promotes within-family similarity in adiposity (Stunkard *et al.* 1990; Sorensen & Stunkard, 1993). Twin studies are particularly informative because monozygotic twins are genetically identical, while dizygotic twins have the genetic relatedness of siblings (50% shared genes on average), but are similar to monozygotic twins in growing up at the same time and being the same age as one another. The results of twin studies indicate that the heritability for BMI is approximately 0·70, approaching that for height. Most studies have been carried out with adults, but in a large twin study on young children's weights heritability estimates have been reported to be fairly similar to those found in adults (Koeppen-Schomerus *et al.* 2001). These results indicate that approximately 70% of the variability in BMI within the population is attributable to heritable genetic differences. Of course, this finding does not address the issue of increasing population weights, which are, without doubt, a consequence of environmental factors. However, the message from the genetic studies is that whatever the overall environmentally set level, there are always likely to be thinner and fatter individuals, and this variability is largely explained by genetic differences.

If genetic differences explain about 70% of the variation in weight between individuals, then environmental differences must explain about 30%. The key question is what aspects of the environment are responsible for variability between individuals. Fortunately, there are research designs that help to clarify whether the environmental effects derive from the shared environment of the home or from individually-specific aspects of the environment (Hewitt, 1997). To the extent that individuals who grow up in the same family are even more alike than would be expected from their genetic similarity, it can be concluded that aspects of the shared family environment, such as the food available in the home, must impact on weight. Surprisingly, studies of adult twin pairs tend to find shared environmental effects to be extremely small (Grilo & Pogue-Geile, 1991), indicating that most of the environmental influences on weight are unique to the individual. Adoption studies provide the strongest evidence, because children grow up sharing a home environment with family members to whom they are genetically unrelated. They find similar results: adults who were adopted at birth do not resemble either their adoptive siblings or their adoptive parents in adiposity (Stunkard *et al.* 1986; Sorensen & Stunkard, 1993). However, most of these studies have been carried out in adults, and by adulthood family members' lives will have diverged considerably, so the effects of sharing an environment in childhood is likely to have diminished. Studies of adiposity in childhood could be expected to show stronger shared environment effects. In line with this prediction, shared environment effects have been found to be substantially higher in a study of 4–5-year-old twins (Koeppen-Schomerus *et al.* 2001). This result suggests that children's adiposity is partly determined by their home environment, even though effects wane as children mature and move away from the family home.

The family studies described in this section partition causal factors broadly into genes, shared environmental characteristics and non-shared environmental characteristics. The results point to genetically-determined individual characteristics as the main causal influence, followed in the case of young children by a mixture of shared family environments and individual environments. These conclusions raise two important questions: (1) if genetic effects dominate, does it follow that obesity cannot be effectively treated; (2) if family environments have an impact on children's adiposity, what are the active characteristics of the environment and is it possible to change them.

Genetic influences on adiposity

In the past the expectations were that 'the' obesity gene would be discovered, mutations of which would confer a massive risk of obesity. It was also assumed that new obesity treatments would follow from this greater understanding of the biological pathways. Finding the gene for leptin deficiency was a dramatic start (Zhang *et al.* 1994). Shortly afterwards two massively-obese children with a mutation in the homologous gene were identified (Montague *et al.* 1997). They showed the same characteristic hyperphagia and, more importantly, were successfully treated by injections of leptin. This advance provided an early success story in going from biology to clinical medicine. However, single gene causes of obesity are probably exceptional, and few others have been discovered. Current thinking is that there are many genes, possibly hundreds, each contributing a very small amount to variation in weight (Barsh *et al.* 2000). As has been the case for other complex traits, there is increasing recognition that the genetic pathways influencing weight are likely to be very difficult to unravel when individual genes have such small effects on the phenotype (Bougneres, 2002).

Most of the emphasis in genetic research has been on the biological processes related to weight regulation, and there has been steady progress in mapping out the physiological pathways underlying appetite and weight (Cummings & Schwartz, 2003), although this research has not yet yielded any major new approaches to treatment or prevention. There is another potential route through which genes might affect adiposity, and that is through behaviour. Behaviour genetic research has shown that many behavioural, emotional and cognitive traits, from shyness to mathematical ability, are highly heritable (Plomin *et al.* 1994). Given that two behaviours, eating and activity, are the fundamental determinants of energy balance, it is plausible that they could constitute steps on the pathway from genes to adiposity (Faith *et al.* 1997; Wardle *et al.* 2001).

There has, in the past, been a good deal of interest in eating behaviour in obesity. An impressive field of studies on animals and humans from Stanley Schachter and his colleagues (Schachter, 1968; Kozlowski & Schachter, 1975; Rodin & Slochower, 1976) has suggested that obese individuals have greater responsiveness to food cues in the external environment and lower sensitivity to internal signals of hunger and satiety. Together these eating behaviour 'styles' were hypothesised to determine an individual's likelihood of overeating when environmental cues and opportunities are available. Schachter's ideas met great acclaim when they were first produced, but interest faded after it appeared that restraint, not obesity, was the root cause of any eating abnormalities in the obese (Herman & Polivy, 1975; Rodin, 1981). However, since this time there have been such dramatic changes in the relative prevalence of obesity and eating disorders, along with less certainty that all abnormalities of eating behaviour are caused by restraint (Johnson & Wardle, 2005), that it could be timely to revisit the idea that eating behaviours are part of the phenotype that influence obesity risk.

Research into the eating behaviour styles associated with adiposity has continued in paediatric samples and the results have been broadly consistent with Schachter's (Schachter, 1968; Kozlowski & Schachter, 1975; Rodin & Slochower, 1976) original ideas. Obese children do not show the normal deceleration in eating speed during a meal, which is assumed to reflect their lower sensitivity to satiation (Barkeling *et al.* 1992). Obese children are more responsive to food palatability cues in a test eating situation (Jansen *et al.* 2003) and score more highly on a parent-completed version of the External Eating subscale of the Dutch Eating Behaviour Questionnaire (Braet & Van Strien, 1997). Obese children (and adults) work relatively harder for palatable food rewards than for non-food reinforcers than non-obese groups (Smith & Epstein, 1991; Saelens & Epstein, 1996), suggesting that food cues are more enticing for them. Obese children also work relatively less hard for physical activity reinforcers (Epstein *et al.* 1991), suggesting that energy expenditure behaviours may be less reinforcing. As these studies have been carried out on children who are already obese and are usually recruited from a clinical environment, care must be taken in extrapolating to non-clinical samples. Furthermore, it cannot be assumed that behavioural differences precede adiposity. However, the results are consistent with the idea that individual's eating behaviour styles play a part in determining the impact on them of the obesogenic environment.

Environmental influences on adiposity

There has been enormous interest among scientists, public policy analysts and the media in determining which of the many environmental changes that have taken place have driven the increasing levels of adiposity in the population. Less attention has been paid to identifying the environmental factors that might contribute to individual differences in adiposity, even though they are vital for individuals who are trying to achieve weight control. Parsimony would suggest that the environmental causes for individual differences should be similar to those that cause secular trends: transport options in the individual's local environment; day-to-day energy expenditure demands (design of homes and workplaces; types of job, opportunities for leisure activity); food options (availability, cost and type of food available locally); sub-cultural norms

for eating and leisure activity. If these ideas are correct, obesity clustering might be expected to be seen in neighbourhoods with fewer options for healthy eating and activity, in schools that provide more fast foods and less physical education and in occupations in which there is less opportunity for activity. There is an emerging literature examining associations between BMI and environmental characteristics (Frank *et al.* 2004). This research has not yet yielded strong associations with childhood obesity (Burdette & Whitaker, 2004), but there is undoubtedly considerable scope for further work on the impact of neighbourhood effects. Natural experiments in which a change in environmental characteristics (e.g. a fast food store opening in the neighbourhood; access to new forms of transport) can be linked with BMI changes will make an even stronger case (Bell *et al.* 2002).

In relation to early childhood obesity the key environments are likely to be the home and family, since young children spend most of their time in their home and parents have more influence over the child's behaviour at this age. No study, to the author's knowledge, has related the food and activity characteristics of the home environment to children's BMI, although one study (Strauss & Knight, 1999) has shown associations with family structure. There also appear to be no intervention studies that address the impact of changes in the home environment on children's weight.

Implications of research into causal factors for obesity treatment

On the model presented above two factors conspire to cause the development of obesity: (1) the child has inherited a bio-behavioural system that increases the risk of positive energy balance behaviours; (2) the child has grown up in an environment that allows the expression of these behavioural tendencies. The implications for treatment are, therefore, that modification of the environment will contribute to weight control in young children, and that teaching older children the skills to resist any inclinations towards overeating and under-activity will help them to control their weight as they become independent decision makers in relation to food and activity.

Modifying the environment to reduce adiposity

In designing strategies for the management of childhood obesity, the only environment over which parents have direct control is the home. Parents can try to make the micro-environment of the home as 'non-obesogenic' as possible for the time that the child is likely to be influenced by the domestic setting, i.e. in the earlier years. They can ensure that high-energy-dense foods and drinks are excluded from the home and they can serve a choice of healthy foods so that the child learns about choice, without being expected to select the less-desirable option. They can ensure that TV-watching and eating are separated and that food is eaten only at meal times, which should ideally be shared eating occasions where the parents can model

healthier choices. They can keep sweet and fatty snacks and sugary drinks for special occasions, and ensure that their own behaviour provides appropriate norms and modelling experiences for healthy controlled eating. In relation to activity, they can model and promote active leisure pastimes, limit TV and computer games, and use walking or cycling as means of transport whenever possible.

There may also be aspects of feeding style that influence weight gain (Wardle, 2004). None of the existing studies are definitive, but questions have been raised about the role of parental control in regulating children's intake. Birch and colleagues (Fisher & Birch, 1999) have suggested that high levels of parental control might impair children's learning to regulate their energy intake and consequently increase their risk of excess weight gain. However, other researchers have found either no association between control and children's weight, or a reverse association (Robinson *et al.* 2001; Wardle *et al.* 2002*a*). One possible explanation for these varied results is the type of control that is exerted. Where parents control the food coming into the home, then this situation is likely to cause much less friction than when the food is in the home, but denied to the child.

How much effect are changes in the home environment likely to have on children's weights? The short answer is that it is not known, because no studies have assessed the impact on weight of modifications of the home environment, except as an integral part of the family-based behavioural treatment, which is discussed in the following section. However, given that about 30 % of the variation in young children's BMI is explained by shared environment factors (Koeppen-Schomerus *et al.* 2001), of which the children's home is a plausible candidate, it is likely that keeping the home as free as possible of the 'obesogenic' features could make an important contribution to weight control. The effects of the home environment on weight are likely to wane with maturation, as the child's unique environment expands to encompass the neighbourhood, friends' homes, schools and shops, which provide many more choices about food and activity. However, even if family effects are transient, they are no less valuable while they endure, and every extra year without the marginalisation and stigmatisation that many obese children endure is surely a benefit (Strauss & Pollack, 2003).

Changes in the broader environment (schools, local facilities, etc.) are part of public health developments over which parents have little control. For the foreseeable future, children who are at risk of obesity will have to learn the appropriate skills to choose and use their environments in ways that diminish their risk of excess consumption. However, some parents might welcome encouragement and support to become more 'politically' active in relation to the provision of healthy foods in school and even to challenge local shops and supermarkets to adopt more responsible marketing practices. This process is likely to be slow and frustrating, but 'parent power' will be a force for change in the future, and therefore parents whose children are visibly suffering from the obesogenic environment might usefully be an advance guard.

Modifying eating and activity behaviours to reduce adiposity

The greatest leverage for long-term weight control will lie in individuals learning new patterns of behaviour that control the tendency to drift into positive energy balance. Behavioural treatments have, either explicitly or implicitly, addressed many of these behavioural traits through the strategies of self-monitoring, stimulus control, goal setting and reinforcement that are central to the behavioural approach. For example, if high-risk individuals are more responsive to food cues (Jansen *et al.* 2003), then learning to limit cue exposure (stimulus control in the language of behaviour therapy) is critical. If foods are relatively more rewarding than other stimuli for the individual at risk of obesity, then additional reinforcement can maintain alternative choices. If satiety sensitivity is low, then specific intake goals should be helpful. If activity is inherently less rewarding for the higher-risk child, then providing additional external rewards could redress the balance, and likewise targeting reduction of sedentary activities can make a contribution. Both in connection with eating and activity, the higher-risk individual may need to become more observant and vigilant. Self-monitoring is central to the behavioural approach; regular weighing and keeping food and activity diaries are included in all behavioural programmes.

There is general agreement that the best results in childhood obesity treatment have come from family-based behavioural treatment. This approach was developed in the 1980s by Epstein (see Epstein *et al.* 1990*a*,*b*, 1994, 2000, 2001; Wrotniak *et al.* 2004) and represented an attempt to apply the treatment approaches that had been found to be effective in adults to the paediatric context. Parents aided and supported their child as she or he learned to keep food records and set goals for change of diet, activity and weight, and helped to develop a programme of rewards to apply when the child achieved the goals that had been set. Parents were also encouraged to follow the same programme themselves. In addition, changes in the whole family environment (types of foods at meals, activity patterns) were recommended. Excellent results have been reported over a whole series of studies in which successive variations and improvements of the programme have been shown to produce 15–20 % reductions in percentage overweight in the active treatment condition (Epstein *et al.* 1990*a*). Treatments targeting parents and children have been shown to be better than those targeting children alone (Epstein *et al.* 2001; Wrotniak *et al.* 2004), and treatments that incorporate a reduction in sedentary activity (Epstein *et al.* 2000) are better than those that promote activity. Long-term follow-up results have also been reported, indicating that 10 years later a substantial proportion of the losses are maintained (Epstein *et al.* 1990*b*, 1994). The conceptual background for this work has been the proven efficacy of behavioural treatments. The present paper argues that there may be additional reasons to believe that learning to eat differently, combined with provision of a favourable environment, is the best option for children who are obese or at risk of becoming obese.

Implications for research into the treatment of childhood obesity

At present relatively little is known about the proximal causes of weight variation across the population. There is good evidence that inherited genetic characteristics play an important role, but this does not indicate how genes have their effect, merely that the causal factors lie partly within the individual. It is crucial to understand more about the pathways through which genetic variation modifies weight. Research designs that investigate biological and behavioural responses in individuals who are not yet markedly obese, but vary in relation to their risk of becoming obese, would help to shed light on the aetiological factors.

Behavioural treatments have had good success, and might be even more effective if conceptualised in terms of helping children to choose and use their environments in more discriminating ways. Some additional treatment strategies might be indicated for older children to promote long-term behaviour change.

For younger children, research into aspects of the home environment that promote obesity risk is also likely to provide clinically-useful results. This area of work will be novel, with few proven ways to assess the critical aspects of home environment. If obese children are conceptualised as more vulnerable to obesogenic environments, it highlights the need to find more ways to help parents to create less obesogenic microenvironments for their young children, and to help the children to learn how to resist the external environments themselves as they become more independent.

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