

## CHILDHOOD OBESITY: NATURE OR NURTURE?

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### ABSTRACT

With a growing proportion of children now classified as obese, the causes of this condition urgently need to be established. This paper suggests that an interaction between nature and nurture causes this condition. Evidence from family, twin and adoption studies suggests that inheritance has an impact in genetic susceptibility and, although exact mechanisms have not been found to explain common obesity, genome-wide scans have had promising results. Environmental influences, including exercise and particularly diet impact on weight gain, with parents playing a crucial role in the provision of fatty foods, inappropriate portion sizes and eating patterns. These environmental influences could be regarded as more noteworthy as they are amenable to intervention. Future research should investigate how exactly nature and nurture interact to cause childhood obesity. Recommendations are made for intervention and treatment.

### INTRODUCTION

Hippocrates wrote “corpulence is not only a disease itself but a harbinger of others” (Haslam & James, 2005), marking obesity as a serious condition associated with physical problems such as hypertension, diabetes and psychological problems such as low self-esteem (Veugelers & Fitzgerald, 2005). Although body mass index (BMI) - assessing weight in relation to height - is an accepted way of defining obesity in adults, effects of age and sex on growth in children make classification using this index difficult (Haslam & James, 2005). However, the World Health Organisation (WHO) suggests that a child could be classified as obese if their weight is greater than 95% of their peers of a similar age and height (Anderson & Butcher, 2006). With over 7% of Irish children now obese (Williams et al., 2009), it is critical to establish causal factors with a view to prevention and treatment (Bouchard, 2009).

In this paper, both sides of the nature-nurture debate will be addressed, however, rather than claiming that one plays the dominant role in childhood obesity, it will be suggested that how they interact is key. Firstly, genetic factors and their interaction with the environment will be reviewed. Then environmental influences and the mediating effects of genetics will be discussed in more detail.

## **NATURE**

Research suggests that susceptibility to weight gain is influenced by genetic components (O’Rahilly & Farooqi, 2006). Family, twin and adoption studies assess the influence of genetics on weight, and consistently report that 40-70% of the variation between children regarding their weight could be attributed to genes (Willer et al., 2009). These estimates are only slightly less than those for height, a trait almost unquestioningly regarded as heritable by the average person (Farooqi & O’Rahilly, 2006). Family studies have shown that children with obese parents or siblings are five times more likely to be obese (Bouchard, 2009). However, it is unclear from these studies whether this is a result of unhealthy family eating habits or genetic susceptibility.

## **TWIN AND ADOPTION STUDIES**

Supporting the importance of genes, one twin study comparing monozygotic (MZ) and dizygotic (DZ) twins showed that 90% of variance in weight was associated with genetic factors (Dubois, Girard, Girard, Tremblay, Boivin, & Pérusse, 2007). Additionally, as the genetic influence on weight declines with age, full environmental influence may not be detected in this study as it only follows children up to age 5 (Dubois et al., 2007). Addressing this limitation, another study following twins up to age 18 supports the assertion that there is a strong genetic component to weight (Silventoinen et al., 2007). While these studies do not rule out the effects of the environment, they do imply that environment does not influence weight independently of genetic influences (Dubois et al., 2007).

Adoption studies are a useful way of separating environmental and genetic effects (O’Rahilly & Farooqi, 2006). One study investigating the weight of Danish children adopted over 23 years showed a greater correlation between adoptees and birth parents than adoptees and adoptive parents, reinforcing the gene-obesity association (Sorensen, Holst

& Stunkard, 1998). However, most correlations were small, so the rest of the variation in weight must be influenced by factors such as environment. Also, the influence of the prenatal environment and postnatal environment before adoption cannot be ruled out (O’Rahilly & Farooqi, 2006).

From reviewing the evidence presented, it could be suggested that genetic inheritance has an influence on obesity. Since one of the important implications for research is prevention and treatment, knowledge of the exact genes and mechanisms by which genes affect childhood obesity is essential.

## **GENES**

In recent years, research has associated a mutation in the gene controlling leptin production with obesity (Farooqi & O’Rahilly, 2006). Leptin is a hormone produced by fatty tissues in the body. It crosses the blood-brain barrier and interacts with receptors in the hypothalamus, stimulating the release of hormones known to influence weight by decreasing appetite and increasing energy expenditure (Farooqi et al., 2006). A mutation in the gene encoding leptin can result in hyperphagia (increased appetite) and so excessive food seeking and obesity (O’Rahilly & Farooqi, 2006).

A mutation in the gene encoding proopiomelanocortin (POMC) has also been associated with obesity (Farooqi & O’Rahilly, 2006). POMC is associated with production of melanocortins in the hypothalamus which have a role in appetite regulation (O’Rahilly & Farooqi, 2006). The hyperphagia resulting from a lack of melanocortin increases food intake and is related to obesity (O’Rahilly & Farooqi, 2006). Notably, the environment plays a role whether these genes lead to obesity. Controlled experiments have shown that factors such as lack of exercise and a high fat diet increase the risk of obesity. Mice with a mutation on the POMC gene became hyperphagic and obese on a high fat diet but not on a standard balanced diet, and studies on people have supported this (Farooqi & O’Rahilly, 2006). This shows the importance of environmental influences on obesity, which will be further discussed later.

However, it must be noted that mutations in genes encoding leptin and POMC together only account for about 5% of cases of childhood obesity (Bouchard, 2009). Therefore the value of this research in terms of prevention and treatment is limited, as the results are only applicable to,

at best, 5% of obese people, and these are likely to be at the extreme end of the spectrum (Farooqi & O'Rahilly, 2006).

More research needs to be done on genes that may explain a greater amount of the heritability of childhood obesity. Several genome wide scans carried out in the last few years have identified connections between commonly occurring genes and obesity. Herbert and colleagues (2006) showed that a common genetic variant near the INSIG2 gene, occurring in 10% of individuals is associated with obesity. The San Antonio Family Heart Study, an ongoing project started in 1991, produced evidence suggesting that chromosome 2, located near the POMC gene, is associated with leptin production (O'Rahilly & Farooqi, 2006). More research needs to be carried out to confirm these results, elucidate their exact mechanisms and clarify how they interact with environmental influences.

From the evidence presented, it could be suggested that although genetics affects childhood obesity, the environment influences expression of genes. This paper will now review key aspects of the environment, with reference to how genes mediate their effects. Obesity is often conceptualized as an energy imbalance in susceptible subjects, and so the two main environmental factors that may cause this imbalance, diet and physical inactivity, will be reviewed.

### **NURTURE- FOOD INTAKE AND FOOD QUALITY**

Several factors surrounding diet have been associated with obesity such as total food intake, type of food ingested and eating patterns. If obesity is seen to be a result of an imbalance between energy intake and expenditure, the amount of food ingested is an important factor (Moreno & Rodriguez, 2007). Although some studies found no association between food intake and obesity, this may be due to under-reporting of food consumed (Newby, 2007). Two cross-sectional studies show that there is a positive association between energy intake and childhood obesity, even controlling for physical inactivity and parental body weight (Tucker, Seljaas & Hager, 1997). The role of parents in food intake is critical; overweight mothers have been found to serve their children larger portions and more fatty foods, contributing to weight gain (Nguyen, Larson, Johnson & Goran, 1996). Social learning theory posits that children learn from the actions of significant others such as parents (Bandura, 1999). If correct, it could be that unhealthy eating

demonstrated by parents in these formative years results in heightened fat intake during adolescence and beyond (Bandura, 1999).

In addition, research suggests that the type of food consumed may influence obesity levels. Some studies have shown that obese children eat more fatty foods and fewer carbohydrates than their lean counterparts, even taking physical fitness and parental weight into account (Moreno & Rodriguez, 2007). Fatty foods are energy dense and are associated with obesity because they are most likely to be stored in the body, do not provide strong satiety signals in contrast to carbohydrates and are generally considered to be palatable, thereby encouraging their consumption (Newby, 2007). Fast food contains high levels of fat and their frequent consumption may lead to elevated energy intake and reduced consumption of more low fat nutritious foods such as fruit and vegetables (Moreno & Rodriguez, 2007). Other types of food such as soft drinks have also been associated with obesity (Newby, 2007). Children fail to reduce solid food consumption to compensate for the extra calories ingested from soft drinks, leading to weight gain (Moreno & Rodriguez, 2007). Again, parental influence plays a role as children are more likely to eat excessive amounts of fatty foods if their mother is obese (Nguyen et al., 1996).

### **EATING PATTERNS**

Eating patterns may also influence childhood obesity. An uneven energy distribution throughout the day, where less is eaten at breakfast and lunch, and more is eaten at dinner may influence weight gain. Children are more likely to be inactive in the evening, usually watching TV or studying, and so excess energy is not burned off (Maffeis et al., 2000). Recurrent TV dinners are associated with increased fried food consumption which heightens chances of obesity (Moreno & Rodriguez, 2007). This may be because parents are more likely to provide food of greater nutritional value than children and adolescents would prepare themselves (Gillman et al., 2000). Also, family meals prevent TV dinners and potential “mindless eating” that may lead to increased calorie intake (Veuglers & Fitzgerald, 2005).

In addition, a pattern of serving large food portions is associated with obesity as children’s energy intake increases (Newby, 2007). Over time, they become worse at regulating intake according to feelings of

satiety, leading to further increases in intake (Newby, 2007). Parental influence is again important in this area, as obese parents have been shown to serve larger portions to their children (Nguyen et al., 1996).

It must be noted that diet and eating patterns do not operate independent of genetics. Some children overeat to cope with negative emotions, and this pattern is associated with inherited characteristics (Tholin, Rasmussen, Tynelius & Karlsson, 2005). A genome-wide linkage analysis showed that an uninhibited eating pattern is associated with a gene that encodes peroxisome proliferator activated receptor  $\gamma$  (PPAR-  $\gamma$ ). This receptor is mainly found in adipose tissue and is involved in leptin action, which influences eating and appetite (Steinle et al., 2002).

Additionally, genetics can mediate the effects of systematic increased food intake. Bouchard and colleagues (1990) overfed MZ twin pairs and the amount gained by the twins varied from 3 kg to 12kg. The strongest predictor of weight gain for the identical twin was the amount of weight gained by other, suggesting that heredity has an important impact on weight gain in response to diet (Bouchard et al., 1990).

So far, aspects of diet have been shown to impact on childhood obesity, although not independent of genetic influences on both eating behaviours and susceptibility to weight gain. The energy surplus caused by consumption of high energy food and large portion sizes can be combated by physical activity which utilizes excess energy (Hill & Wyatt, 2005). Collecting data on more than 130,000 youth from 34 countries, one study suggested that lower physical activity was associated with obesity (Janssen et al., 2005).

## **PHYSICAL ACTIVITY**

Although many factors affect children's participation in physical activities, such as gender, availability of parks and psychological factors such as perceived competence (Sallis, Prochaska & Taylor, 2000) this discussion will focus on TV viewing and playing video games. Janssen and his colleagues (2005) found that TV viewing was associated with being overweight. However, there is inconsistent evidence to support the hypothesis that the cause of this link is a decreased amount of time spent engaging in physical activity (Han, Lawlor & Kimm, 2010). Exposure to advertisements on TV for high fat, low nutritional foods may provide an

explanation. Advertising increases children's desire for this food, as measured by increased requests for snacks, and fosters the belief that snack food consumption will not lead to excess weight gain, as the models in the advertisements are usually of average weight or thinner (Vandewater, Shim & Caplovitz, 2004).

Vandewater and colleagues also (2004) found that time spent using video games was associated with weight gain. However, as this data was correlational cause and effect could not be established. In contrast to the standard interpretation – that video game use decreases the time spent in physical activities and so contributes to obesity – it could be the case that those who are overweight are less inclined to engage in physical activity and so use entertainment media more frequently (Vandewater et al., 2004).

Although results supporting the idea that TV and video game use lead to obesity because of a reduced activity level are inconsistent, it cannot be concluded from this that exercise is unimportant. TV and video game use may just be a poor indicator of activity level (Han et al., 2010). Beneficial effects of exercise can clearly be seen in the obesity interventions discussed later.

### **BROADER ENVIRONMENTAL INFLUENCES**

Although this discussion focused on more proximal factors that influence childhood obesity, broader environmental factors may also have an effect. For example, access to an outdoor play area can influence the amount of physical activity (Spurrier, Magarey, Golley, Curnow, & Sawyer, 2008). Also, changes to the built environment in the shape of urban sprawl increases motorised transport and so decreases the amount of energy children use during the course of their day (Anderson & Butcher, 2006). The amount of energy used for everyday living has fallen as people have adopted increasingly sedentary lifestyles (Prentice & Jebb, 1995). This is particularly problematic taking into account our evolutionary heritage which favoured individuals with parsimonious energy metabolism, and which stores energy as excess fats (Han et al., 2010).

Some research has shown that genetics mediates the relationship between weight and physical activity (Esparza et al., 2000). One twin study showed that MZ twins were more similar in activity level than DZ twins, taking into account effects of the shared environment. This

suggests heredity has an effect on activity level (Eriksson, Rasmussen, & Tynelius, 2006).

In summary, evidence from family, twin and adoption studies suggests that inheritance has an impact in genetic susceptibility and although exact mechanisms have not been found to explain common obesity, genome-wide scans have generated promising results. Proximal environmental influences, including exercise and particularly diet impact on weight gain, with parents playing a crucial role in the provision of fatty foods, inappropriate portion sizes and eating patterns. Although these environmental influences can also be affected by inherited characteristics, it could be suggested that the environment is slightly more important as it is amenable to change and intervention. It is, however, important to keep in mind research about the genetic component to weight in order to identify groups that may be most at risk.

### **CONCLUSION: PREVENTION AND TREATMENT**

Measures regarding the prevention and treatment of obesity can be targeted at the individual, family and institutional levels (Han et al., 2010). Prevention is regarded as critical by the World Health Organisation in order to shield children from the difficulties of weight loss and co-morbid disorders (World Health Organisation [WHO], 2004). At the individual level, treatments targeting children's diet and activity patterns tend to have high efficacy rates, as children's behaviour is quite malleable (McCambridge et al., 2006). Strength training seems to be of particular benefit, because results are apparent over a shorter time and overweight children are likely to be stronger than their peers, giving them an advantage (McCambridge et al., 2006). Walking with a pedometer (which has gadget appeal) instead of using transport has been found to aid weight loss (McCambridge et al., 2006). As parental influence on diet is critical, encouragement of parents to offer appropriate portions, nutritious foods and foster physical activity has made a difference to children's weight (Hawkins et al., 2009).

At an institutional level, government intervention is warranted as the estimated yearly cost of obesity is over €30 million. The Irish government has recommended an increase in the amount of physical activity in the school curriculum to two hours per week in order to decrease rates of obesity (Department of Health and Children [DOHC],



2009). This is particularly important because according to the Growing Up in Ireland study, 75% of children did not partake in the recommended 60 minutes of exercise per day, one main reason being the lack of opportunity (Williams et al., 2009).

In conclusion, it could be suggested that asking whether nature or nurture cause childhood obesity is not the right question. As this paper has argued, these factors are both at play and the real question that needs to be answered is how they interact to cause obesity. Further research is needed but we should note that because environmental factors can be altered to prevent or treat excessive weight gain, it could be suggested that they slightly outweigh the importance of genetic inheritance.

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