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Food preferences

Genetic and environmental influences both play a role in food preferences in adolescence

The formation of food preferences in children and adolescents is a complex process with both genetic and environmental factors at play. This edition of *The Global Fruit and Veg Newsletter* offers three viewpoints examining the role of genetics in shaping food preferences and obesity in adolescents and children.

Joanne Cecil provides a summary of the connections between genotype and behavioural phenotype in the maintenance of child energy balance and obesity. She reports that individual genetic differences exist in susceptibility and resistance towards weight gain and obesity. Individual genetic variations include common gene polymorphisms (such as variance to the fat mass and obesity-associated (FTO) gene), and the rarer, single gene mutations that lead to monogenic obesities. The former predisposes children towards obesogenic eating behaviours via changes to the appetite pathways. While the latter lead to extreme obesity in children, and appear to disrupt appetite regulating mechanisms that control food intake, thus changing the energy balance.

Andrea Smith and Claire Llewellyn discuss the how preferences for different foods are shaped by both genetic and environmental factors, providing data showing that even twins have differing food preferences. One example, a 2016 twin study, showed that 54% of variance of preferences for vegetable consumption could be explained by genetics at 18-19 years. This is the largest amount of preferences explained by genetic influences, with other foods having lower genetic influences in preference formation – for example starch preferences are only 32% explained by genetic influences in 18-19 year olds. This variance in preferences may not only be due to differences in genetics. The food environment of various products also differs considerably, with different foods being advertised differently, having different consumption opportunities, and different cultural consumption norms.

The role of the parent in creating a positive environment for healthy eating preferences in children and adolescences is also well established. Marion Hetherington in her article discusses the role of the parent in promoting vegetables to children via complementary feeding. She gives the example of adding vegetable flavours to milk or baby rice. This resulted in greater liking and intake of vegetables, thus setting up healthy eating preferences at an earlier age.

The evidence suggests that when it comes to obesity and food preferences in children and adolescents, genetics does play a role. However environmental factors are also vital, and - to a larger extent - within our control. We can alter many environmental factors to create healthier consumption preferences, such as changes in food advertising, food consumption opportunities and norms. Altering the food environment is a positive not just for children and adolescents, but for the wider population. Currently, being overweight is "normal" in England with 61.7% of adults either overweight or obese. Changes to environmental factors – including increased fruit and vegetable consumption – will help reduce this worrying statistic, and keep it lower.

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A genetic approach to understanding obesity: genotype and behavioural phenotype in the maintenance of child obesity

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Relationship between genotype and behavioural phenotype for obesity

Child obesity has become one of the most significant complex public health problems of this century. Globally, an estimated 42 million children below five years of age present with excess adipose tissue. Yet, not all children become overweight or obese in an 'obesogenic environment', suggesting that individual differences exist in susceptibility and resistance towards weight gain and obesity[1]. Genetic variation, environmental differences and variation in the response to the environment determine susceptibility and the expressed phenotype.

Evidence for a genetic role in obesity and eating behaviour?

The evidence supporting a genetic contribution to obesity is considerable. Twin, familial aggregation and family studies have been consistent in characterising a heritable association between the influence of genes and obesity. Notably, twin studies have shown heritability estimates as high as 75% for child BMI[2] and similarly high estimates for body fat[3]. Heritability estimates for appetite and eating behavioural traits associated with susceptibility towards obesity have also been documented in children. For example, the genetic influence towards macronutrient preference, food preference, satiety and food enjoyment, eating in the absence of hunger, food fussiness and preference for fruit and vegetables have been shown to be highly heritable[4].

Functional studies, which link candidate genes to an obese phenotype, provide additional robust evidence to support a genetic role in obesity and eating behaviour. Single gene mutations, for example in the leptin (LEP), leptin receptor (LEPR), melanocortin 4 receptor (MC4R), pro-opiomelanocortin (POMC) genes lead to extreme obesity in children and appear to disrupt appetite regulating mechanisms that control food intake leading to hyperphagia[5]. These monogenic obesities have yielded important insights into key pathways underlying the control of energy balance, but are population rare (5-7%) and don't reflect the obesities commonly observed today. Instead, most obese phenotypes are thought to be polygenic and involve complex gene-gene and gene-environment interactions operating frequently with small effects.

Common gene polymorphisms: a role in obesity and eating behaviour

Extensive 'genome-wide associations studies' (GWAS), powered to detect small effects by testing in large populations, have transformed progress in identifying new obesity gene variants or single nucleotide polymorphisms (SNPS) for hypothesis driven research. To date, evidence for over 90 susceptibility loci regulating body weight have been identified[6]. Candidate common gene variants that predispose to polygenic obesity and contribute to variance in eating behaviours associated with child obesity include the fat mass and obesity-associated (FTO) gene, peroxisome proliferator-activated receptor (PPARG), melanocortin 4 receptor (MC4R), adrenergic receptors and reward related variants such as D2 dopamine receptor (DRD2) gene polymorphisms[7].

Currently, the most robustly characterized gene model for polygenic obesity is the FTO gene, discovered from a multi-centre GWAS for type 2 diabetes[8]. FTO, residing on chromosome 16, encodes a protein with 2-oxoglutarate-dependent nucleic acid demethylase activity[9] and is expressed in greatest proportion in brain tissue, and elsewhere in pancreatic islet, adipose tissue and adrenal gland. FTO SNP rs9939609, located in the first intron and present at a high allelic frequency (approx. 39%), has been associated with increased BMI in children and adults[8]. In children, one copy of the minor (A) allele (risk allele) was associated with an increase in BMI of approx. 0.2 kg/m2 from ages 7-10 years, increasing to approx. 0.4 kg/m2 at age 11 years[8]. Multiple studies have since confirmed a role for FTO gene variants in influencing BMI and fat mass in children[10,11]. It is possible that SNPs in intron 1 of FTO are involved in the expression of other genes nearby (e.g. RPGRIP1 and IRX3) and might therefore mediate an influence on obesity via these neighbouring genes, illustrating a more complex understanding FTO than originally envisaged[12].

FTO related susceptibility to polygenic obesity is thought to be primarily mediated via a central role in the control of food via alterations in appetitive pathways, rather than via energy expenditure. Experimental studies in rodents have shown that expression of the gene is concentrated in brain regions known to be responsible for regulating feeding[9,13]. In support of functional evidence, studies conducted in children have shown that FTO variants predispose to obesity risk though increased energy intake[10,14] a preference for energy dense foods[10,14] reduced satiety responsiveness[15] and loss of control over eating[16]. Thus, FTO appears to represent a gene model that predisposes towards obesogenic eating behaviours.

Molecular genetics has contributed valuable information about the genetic architecture of common complex disease such as obesity. The knowledge that polygenic obesity, like monogenic obesity, appears to be driven principally by disruption of appetite regulation can potentially be used in development of novel therapeutic targets and behavioural strategies which may have implications for diagnosis, prevention and management of obesity.

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Genetic and environmental influences on fruit and vegetable liking in adolescence

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It is estimated that around 10% of the world's entire health burden is attributable to sub-optimal dietary intake, with 5.7% of disability-adjusted life-years (DALY's) lost due to diets low in fruit and vegetables[1]. Our food and drink preferences influence strongly what we choose to eat or drink, evidenced by the fact that preferences predict actual food intake[2]. Understanding the factors that shape our liking of fruit and vegetables is therefore important for public health initiatives that aim to increase intake.

Twins can establish genetic and environmental influence on fruit and vegetable liking

Twin studies are a powerful method for understanding the extent to which liking for fruit and vegetables (or any trait) is driven by genetic and environmental influence. The basis of the method is to compare resemblance in liking between identical twin pairs who share 100% of their genes, with liking between non-identical twins who share about 50% of their genes. Because both types of twins share their environments to a very similar extent, the only real difference between the two types of twins is the fact that identical twins are twice as similar genetically. Greater resemblance between liking for identical versus non-identical twins therefore indicates a genetic contribution to preferences; similar liking for both identical and non-identical pairs indicates that environmental factors shared completely by twin pairs are important (e.g. maternal gestational diet); and low similarity in liking between twin pairs suggests that factors unique to each individual twin are key (e.g. illness).

Environmental influence on liking for fruit and vegetables changes from early childhood to late adolescence

Previous studies have shown that aspects of the environment shared entirely by twin pairs play an important role in shaping liking for fruit (51% to 53%) and vegetables (35% to 51%) among young children[3,4], alongside modest genetic influence (fruit: 53% to 54%; vegetables: 37% to 54%). This is not unexpected given the importance of the family environment for the eating behaviour of young children. However, the relative influence of genes and the environment can change dramatically with age, and the drivers of older teenagers' preferences were unknown until very recently. In 2016, we explored this for the first time in a large sample of 18-19 year old twins (n=2865) from the Twins Early Development Study (TEDS), a population based British cohort of twins born in 1994-96[5]. Preferences for 62 individual foods were self-reported, and categorised into six food groups: fruit, vegetables, meat/fish, dairy, starch food and snacks. In contrast to the study of younger children we found no influence of the shared environment on preferences for any type of food. Instead, aspects of the environment that are not shared by twin pairs (experiences unique to each individual twin, such as having different friends) exerted an important influence on liking for all of the foods (46% to 68%), alongside modest genetic influence (32% to 54%; in keeping with estimates observed for young children) (see Figure 1).

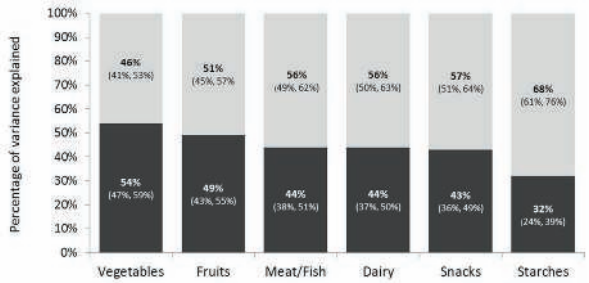

Figure 1. Proportions of variation in food preferences explained by genetic (black portion of bars) and unique environmental (grey portion of bars) influences at 18-19 years of age (n=2685).

How could genes influence preferences for fruit and vegetables?

Food preferences can vary considerably, even among people from the same cultural background, evidenced by the fact that the food preferences of twin pairs can differ. Genes play a part in explaining some of these differences, and specific genes and their pathways have been proposed. Variants in the TASR gene family (a family of bitter taste receptors) affect sensitivity towards bitter compounds[6], and carriers of a variant in the TAS2R8 gene are especially sensitive to bitter tastes, and have lower liking of cruciferous vegetables[7,8]. Other proposed mechanisms relate more to cognitive aspects of food preferences[9], such as food neophobia[10] or reward circuitry[11].

What environmental influences might be important?

Much more is known about the environmental influences that shape food preferences, especially for children. When it comes to vegetable liking, exposure is key. In short, we like what we know, and we eat what we like. Repeatedly offering (15 times or more) vegetables to children can increase both liking and intake over the short term[12]. However, research is needed to establish which strategies are effective for modifying preferences for fruit and vegetables on a wider scale (e.g. for public health initiatives).

Conclusions

Our findings indicate that the effects of family upbringing on liking for fruit and vegetables (and other foods) have entirely disappeared by late adolescence; replaced instead by environmental influences that are unique to each individual twin. This suggests that efforts to improve adolescent nutrition may be best targeted at the wider environment rather than the home. However, the substantial influence of the non-shared environment suggests considerable scope for modification of food preferences by environmental means. Research is needed to establish the most effective strategies for increasing liking of fruit and vegetables in this age group on a large scale.

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Adopting a vegetables first approach to complementary feeding

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Parents want to do the best for their children and guidance suggests introducing solids at six months (6m) after exclusive breastfeeding. In the early stages of complementary feeding (CF), parents vary in the approaches they use to encourage acceptance of solid foods. For example, in some cultures it is customary to pre-chew foods before offering to infants and in other cultures, foods such as vegetables are pureed then offered by spoon at the time of weaning. Recently baby-led weaning has become a popular strategy where parents provide finger foods so that autonomy in eating is encouraged from the beginning. Weaning guidelines tend to focus on when to introduce solids however the Guiding Principles for Complementary Feeding published by the WHO[1] contains specific guidance on how to feed as well as how much (See figure 1). Feeding responsively means being in touch with the ways in which infants communicate their needs, likes and dislikes. In order to achieve these parents must be able to identify, interpret and respond to signals of hunger, appetite and satiety as well as food preferences (see Hetherington, 2017).

Weaning with vegetables

Given that children struggle to consume sufficient vegetables due to their bitter taste or unusual texture, attempts to introduce vegetables in school age children is challenging[2]. In contrast, offering a variety of vegetables during CF promotes vegetable acceptance since this period presents a unique window of opportunity to experience new foods, specifically vegetables. At the age of 6m infants willing to try novel foods and are setting the foundations for healthy eating. Taste preferences established early on-track into later life. For example, offering a high variety of vegetables, compared to no or low vegetable variety during the first 10 days of CF increased acceptance and intake of vegetables which lasted up to six years later[3].

Most mothers in the UK offer baby rice or fruit as a first food, most likely because they hope that this first experience will be enjoyable. Baby rice made with breastmilk or formula may form a bridge between milk feeding and solids, as fruit is naturally sweet. However, adopting a vegetables first approach is more likely to facilitate acceptance of these foods when it matters most – at the beginning of the flavour journey[4]. In France, mothers report adding vegetable flavours to milk around the time of weaning. We tested this practice in a small sample of UK mothers by systematically adding vegetable flavours (purees of cooked vegetable) to milk for 12 days and then baby rice for 12 days. The result was an enhanced liking for vegetables and an increased intake compared to the control group. Infants given vegetables in this way demonstrated fewer negative facial expressions, more positive overt behaviours and a faster rate of eating vegetables during filmed lab visits[5]. Therefore, although mothers may be reluctant to use vegetables as a first food[6], they might do this using a step-by-step introduction towards acceptance of the pure and distinctive flavour of vegetables.

In conclusion, after the milk feeding phase, the period of complementary feeding provides the optimal opportunity to introduce a variety of vegetable flavours first and often to promote liking and intake to set the foundations of healthy eating.

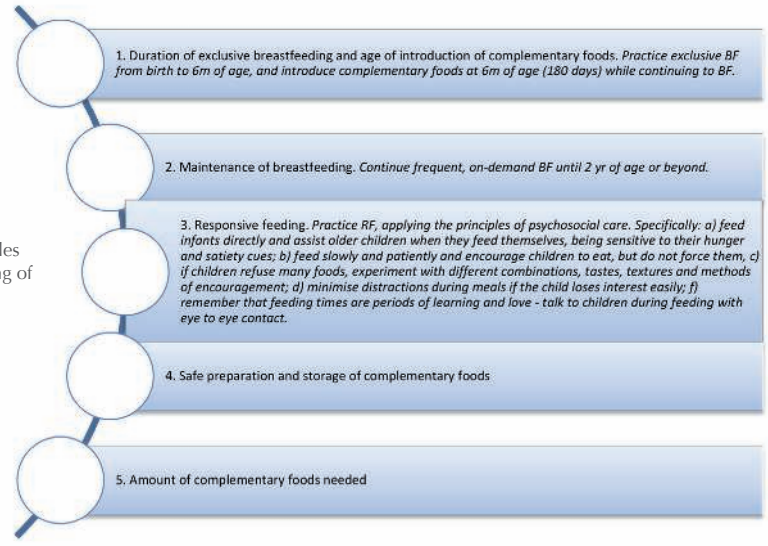

Figure 1: Guiding principles for complementary feeding of the breastfed child

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