Understanding food fussiness in young children: implications for food choice, health, weight and interventions

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Abstract

Purpose of review

This review examines the concepts of fussy eating and food neophobia in the context of key determinants of the development of children’s food preferences. We discuss the evidence for genetic versus parental and other environmental influences on the ontogeny of these behavioural traits, and the implications of current knowledge for interventions that attempt to lessen the impact of these traits on children’s diets. Finally, we consider whether these traits increase the risk of a child becoming obese, or alternatively, underweight and malnourished.

Recent findings

Fussy eating and neophobia are related concepts with both genetic and environmental aetiologies. Parent-child correlations and heritability estimates are moderate to high for both traits, but aspects of the family environment remain influential in young children, although no longer in young adults. Parental strategies based around repeat tasting opportunities can improve acceptance of disliked foods in even the fussiest children. Fussy eating and neophobia are not risk factors for obesity, but could limit growth in severe cases.

Summary

Fussy eating and food neophobia are common concerns for parents, though health risks are low. Dissemination of evidence-based strategies to parents that can encourage a more varied diet in young children would be helpful.

Keywords: Children; picky/fussy eating; food neophobia; fruit; vegetables; body weight
Introduction

Both parents and health professionals will be familiar with the observation that children appear to vary substantially in their eating behaviour and food preferences, even within families. On the one hand, parents may struggle to persuade one child to eat ‘healthily’ or even to eat enough, whereas for another child, perhaps a sibling, the concern may instead be how to stop the child eating too much of almost anything. This review will address how these differences might arise, as well as highlighting fundamental patterns and consistencies in the development of food preferences during childhood. Two particular restrictive eating concepts, ‘food neophobia’ and ‘fussy (or picky) eating’ could each contribute to a child rejecting a large range of foods; however, despite this overlap in outcome, they are normally defined differently, and this review will consider these definitions as well as the time course of their expression, possible shared aetiology, and impact on children’s eating behaviour. A key question is whether children’s different eating styles and preferences have implications for their development and later health, including risk of becoming obese, or conversely, malnourished. If these risks exist, then there is a need for effective interventions, including advice to parents on strategies that they might use to modify their child’s eating behaviour. Even without evidence for broad health risks, dissemination of accurate advice to caregivers is likely to benefit family cohesion and may improve the nutrition of some children [1]. Recent findings on these issues, as well as on the timing of development of these behaviours, and evidence for their heritability, will be reviewed here. The implications for risk of obesity, or malnutrition, and the modifiability of children’s food fussiness, will be discussed.

Defining food fussiness and food neophobia

Picky or fussy eating is a concept that describes a person’s tendency to reject a large range of foods, including familiar foods eaten regularly by other family members, so self-selecting a rather restricted diet. Food neophobia is usually defined in relation to avoidance of trying new or unfamiliar foods [2]. Food fussiness and neophobia are quite common, particularly in young children (see below), and can be a source of anxiety for parents (or caregiver) around meal times. They are both consistent behavioural traits with strong genetic components (see below), and associations with other traits such as anxiety and shyness [3]. Most parents are aware of basic healthy eating messages [4] and so their child’s refusal to eat vegetables, for example, becomes a cause of parent-child conflict that can be stressful for both parties. Whilst there is no clear consensus on
how food fussiness should be defined, most definitions overlap in the concept being described. Definitions have recently been reviewed [5] and there is a consistent theme of both strong but limited likes and more extensive dislikes, and reluctance to try new foods, to the extent that family meals are often modified, or separate meals provided, to satisfy the need to feed the fussy child sufficient food. It can be seen that there is overlap of these descriptions of fussy eating with food neophobia, though the latter is normally defined in terms of specific refusal to try new foods Given that at some point in their development, all foods are novel to a child, it is likely that the primary carer will find it harder to distinguish a fussy eater from a neophobic child than researchers may wish, even if a clear distinction does exist. Indeed, the distinction may simply arise from concepts imposed by researchers who designed questionnaires to measure one concept or another: limitations of measurement are discussed further below.

Determinants of children’s food preferences

Children’s food preferences are determined by a complex interaction of genetic and environmental factors which can be categorised into the following domains: (a) genetic (influences of behavioural traits, as well as other innate neurobiological and physiological effects) (b) prenatal (sensory experience in utero; maternal nutrition) (c) early postnatal experience (breast or formula feeding; weaning practices) (d) parental feeding practices (food choice, portion size, reinforcing strategies, modelling) (e) family environment (social, economic, presence of siblings). Each of these will be summarised in turn, and any relevance to fussy eating discussed.

Genetic influences

The relative contribution of genes and environment to behavioural trait phenotypes is usually expressed in terms of heritability, i.e. the ratio of total variance of a trait due to genetic influences to total phenotypic variance, including environmental (acquired) factors. Heritability is usually measured by either genome wide association studies (GWAS) or twin (including adoption) studies. However, whilst GWAS have been able statistically to link genetic loci to eating related traits, especially body mass index (BMI), they have had only limited success in explaining the phenotypic variance [6]. For example, a recent GWAS in 339, 224 individuals identified 97 BMI-associated loci, but the total BMI variance explained was just 2.7% [7].

The low phenotypic variance explained by GWAS is in marked contrast to the high heritability estimates reported from twin and adoption studies, for example ranging from 50% to 78% for adiposity [8, 9]. Of relevance to this review, some
behavioural phenotypes likely contributing to expression of appetite and obesity risk have shown high heritability in twin studies. For example, heritability of satiety responsiveness averaged 63%, and 75% for food cue responsiveness/enjoyment [10]. Children's food preferences also show selective heritability: in one twin study of 4-5 year-olds, heritability of food preferences ranged from 20% for dessert foods to 78% for liking for meat and fish, with intermediate levels for fruits and vegetables, with shared family environment explaining most of the remaining variance [11]. In a much larger twin study (Gemini; n=2686), genetic contributions to these food group preferences in 3-year olds were qualitatively similar, with highest heritability for nutrient-dense foods (protein, vegetables and fruit: 48-54%) compared to starchy foods, snacks and dairy (respectively, 32%, 29% and 27%) [12]. Again, shared family environment explained most of the other variance. This order of heritability of food likes may reflect the relative biological importance of the food groups (e.g. nutritional benefit vs. risk of harm). Moreover, this food group order is seen in the susceptibility of foods to fussiness and neophobia [13] or, in reverse, to food enjoyment: thus, it is not surprising that there is evidence for genetic influence on these eating tendencies (see below) [14-16]. By comparison, a recent study of 2865 twins aged 18-19 years [17] found that, as children grow into young adults and experience increasing independence in eating and food choice, the unique environment experienced by each individual becomes more influential on preference patterns within food groups, although heritability still has a moderate influence, ranging from 32% to 54% across food groups. At this age, the influence of shared family environment appeared to be minimal.

In addition, gene polymorphisms in taste receptors could differentially affect children's food preferences [18]: for example, polymorphisms in the TAS2R38 gene affect sensitivity to bitter tastes [19]. In children, this genetic bitter taste sensitivity is associated with preferring sweeter tasting foods and drinks [20, 21], perhaps counterintuitively because of greater detection of sweetness [22], and dislike of bitter vegetables [23, 24]. Despite this evidence, few studies have reported links between these genetic taste differences and fussy eating or neophobia, although in one large sample, being more sensitive to bitter taste at age 10 years was weakly associated with being a fussy eater in early childhood [25]. By contrast, children with lower detection thresholds for bitterness have been reported to be more emotionally reactive to food [26]. It also needs to be born in mind that food texture, as well as taste and olfaction, has a strong influence on young children's acceptance of foods [27].

**Prenatal influences**
In humans and other animals, there is evidence that maternal diet during pregnancy can produce a nutrient-dependent intrauterine ‘programming’ that can influence the offspring’s appetites and food choices [28]. In one study, maternal macronutrient intakes during pregnancy predicted the children’s macronutrient intakes at 10 years of age, especially protein and fat, more strongly than either paternal intakes or mothers’ postnatal intakes [29]. Another study found that enhanced liking for salty tastes in children was linked to severe ‘morning sickness’ in the pregnant mothers [30]. One mechanism that has been suggested to link maternal diet to at least infant preferences is the ability of the foetus to learn from exposure to flavours in utero. Neonates exposed in utero to vegetable flavours in the amniotic fluid via their mothers’ diet show greater acceptance of those flavours in their first solid meals [31].

**Postnatal early feeding influences**

It is well established that children are born liking sweetness, and disliking bitterness; innate dispositions that are thought to reflect adaptive advantages, i.e. a liking for sweetness encourages consumption of good energy sources including breastmilk, whereas a dislike of bitterness may protect against poisoning by plant alkaloids [32, 33]. Nevertheless, innate tendencies are mutable by experience, and even the breastfed neonate is susceptible to learned influences on likes and dislikes: several key studies by Mennella and colleagues have shown that maternal diet affected the flavour of the mother’s milk, which in turn altered flavour acceptance in their babies [34, 35]. In an early study, infants recently exposed to garlic flavour in their mothers’ breastmilk spent less time breastfeeding but consumed equal amounts, when garlic flavour was present compared to infants who had not had recent exposure to garlic-flavoured breastmilk. That is, the unexposed infants drank at half the overall intake rate of the exposed infants, suggesting that the novelty of the flavour may have slowed their drinking rate [34]. Conversely, in a subsequent study, infants whose mothers had been consuming carrot juice, which flavoured their breast milk, ate less of a cereal mixed with carrot juice than did infants not exposed to carrot-flavoured breastmilk [35]. The authors speculated that this might represent a form of sensory-specific satiety to carrot flavour in previously exposed infants, though alternatively, in a semi-solid familiar and bland food, the novelty of the carrot flavour to unexposed infants might have stimulated intake. However, this effect on intake contrasts with a subsequent study where infants’ facial expressions were recorded: infants showed fewer negative responses to carrot-flavoured cereal when their mothers had regularly drunk carrot juice during either pregnancy or lactation [31]. Furthermore, when weaning, breastfed infants have been shown to accept a novel vegetable or fruit more readily than formula-fed babies [36, 37].
These effects were attributed to early exposure to vegetable or fruit flavours in breastmilk, though this was not demonstrated at the time. Such early exposure to flavour variety could reduce later neophobia or fussiness, and regular breastfeeding has been associated with lower occurrence of food neophobia or fussiness in some studies [3, 38], but not all [39]. Consistent with a benefit to fussiness, as well as exposure to flavours, recent analysis of data from four large cohort studies (approx. final n = 13,700) revealed that being regularly breastfed predicted greater variety of healthy foods in preschool children’s diets [40].

**Parental feeding practices**

Once weaning has occurred, children are faced with an array of novel sensory experiences, including texture as well as flavour, so rapid learning is likely to occur, influencing development of likes and dislikes. At the same time, a child’s innate temperament and eating dispositions (including enjoyment, fussiness or neophobia; see above) will also come to the fore, and parents determined that their child eats the healthiest diet can find this a stressful phase when their infant fails to comply. Birch and colleagues have shown that parents that manage to introduce fruits and vegetables in the first few months of weaning will likely encourage acceptance of other similar foods quite rapidly [41]. In older preschool infants, it may take 5-15 ‘taste exposures’ before acceptance of a novel vegetable is achieved [42-46], although there is evidence that earlier introduction to fruit has a lasting benefit on fruit consumption in preschool children too [47]. Conversely, parents may notice that most children have little difficulty in accepting sweet, fatty or starchy foods: this reflects both a facility to learn to like foods providing rapid energy delivery [48] and an inherent tendency to accept such foods as non-threatening, i.e. even neophobic children rarely avoid such foods [13]. The parental struggle to encourage fussy eating children to eat sufficient food almost inevitably leads their parents to apply pressure to eat [49-51].

**Family environment**

For young children, parents are of course responsible for supplying their food and drink, and by and large for feeding them. Therefore, it is not surprising that the strongest predictor of a child’s diet is that of their parents [47, 4, 52]. Moreover, parental control over their children’s eating need not involve just overt pressure or restriction, but can include covert strategies involving manipulation of the family food and mealtime environments [53]. These subtler strategies may be more usual in better educated families, and it may be relevant that a population-based survey of 4914 4-year-olds found parent-reported fussy eaters to be more common in families with lower
household incomes [54]. However, this could reflect greater concerns about wasting food in poorer families.

Aside from pragmatic issues of food availability, parents can provide crucial role models to encourage - or discourage - their children’s food choice. Children are very susceptible to modelling, or observational learning, particularly of parental behaviour (no doubt conferring survival advantages), but also of respected peers [55-57]. These peers can be siblings, and some intervention results support the idea that parents could use older siblings as suitable role models [58]. However, as well as modelling eating, having siblings can also provide more stimulus for physical activity [59], and this may underlie evidence that having fewer than two siblings increases risk of obesity [60, 61]. Moreover, recent evidence suggests that having more siblings and being later born is associated with choosing to eat smaller portions of both vegetables and confectionery even in adults [62]. This might indicate pressure to accept a smaller share as the norm when brought up in larger families. Oddly, it seems that the influence of sibling number on picky eating and neophobia has not been formally reported.

**Ontogeny of food fussiness and neophobia**

**Prevalence and timing**

Estimates of the prevalence of food fussiness vary widely from study to study and also vary with the age of the children studied. For example, in a sample of Dutch 4 year olds, prevalence was 5.6% [63]. In a similar aged sample from the USA, however, prevalence was reported as 21% [64], and a study of Chinese 7-12 year olds found that 59% were identified as ‘picky’ [65]. Researchers who have investigated prevalence in different age groups typically find the highest rates of pickiness in 2-3 year old children. For example, in one study 25% of 7-8 month old infants, 35% of 12-14 month olds, and 50% of North American 2 year olds were identified as picky eaters [66]. The Generation R study of a large population-based cohort in the Netherlands has recently reported prevalence rates of 27% at 18 months, 28% at age 3 years dropping to 13% at 6 years [67]. On the other hand, another study found a steady increase in pickiness from 2 years to a peak at 6 years, when prevalence plateaued [1].

A major contributor to the inconsistency of these estimates is the lack of a consensus as to the definition of fussy or picky eating, and the absence of a reliable and validated instrument with which to measure it. This difficulty is well illustrated by a survey conducted in Singapore by Goh and Jacob [68]. When parents or grandparents of
1-10 year old children were asked whether the child was a ‘picky eater’, 25% said ‘All the time’ and a further 24%, said ‘Some of the time’. However, when given examples of typical picky eating behaviour, the percentage responding ‘All the time’ increased to 50%.

Many studies of picky eating in childhood have used a similar single item measure of pickiness [e.g., 64]. Others use the Food Fussiness scale from the well-established and psychometrically sound Child Eating Behaviour Questionnaire [69, 70], although two items could be argued to relate more to neophobia (e.g., "My child refuses new foods at first") than to general fussiness.

Neophobia is generally measured using Pliner’s Child Food Neophobia Scale (CFNS) [71] but like the CEBQ Food Fussiness scale, it appears to measure both traits, including items such as “My child is very particular about the foods s/he will eat”. Whilst many believe the two traits to be behaviourally distinct [3], it is clear that there is considerable overlap between them. The alternative and perhaps more valid means of measurement is by behavioural tests in which children are asked to try a number of unfamiliar foods and the degree of their willingness to do so is used as a measure of the trait.

It has been suggested that food neophobia represents an innate predisposition which functions to protect toddlers from the possibility of accidental poisoning [72, 13], and is present in all children to a greater or lesser extent [73]. There is broad agreement amongst researchers that from a very low level in the first year of life, there is a steep increase in neophobia in the second year reaching a peak between the ages of 2 and 6 years [74, 13] and declining thereafter [75] – a very similar pattern to the data for food fussiness. In a rare longitudinal study of the stability of neophobia over time, mother-child dyads were recruited when infants were six months of age, and followed up at 12 months and 4.5 years [76]. Neophobia was assessed using behavioural tests at 6 and 12 months, and at 4.5 years using both behavioural tests and parent report (CFNS). The extent to which rejection of novel foods at 6 months predicted neophobia at 4.5 years depended upon mothers’ own levels of neophobia: if maternal neophobia was high then children were rated as neophobic at 4.5, but not if it was low. In contrast, children who rejected novel foods at 12 months tended to have high levels of neophobia at 4.5 years regardless of their mothers’ levels. This study highlights the importance of familial influences on eating behaviour traits, and may be related to the finding (Generation R study) that mothers’ anxiety levels predict food fussiness in 4-year-old children [77]. It might help the child’s eating behaviour if mothers are aware of the influence of their own neophobic and anxious tendencies.
**Heritability versus environment**

**Family studies**

Family studies examine the resemblance in food neophobia between parents and children or between siblings. Most of these studies have found low to moderate resemblance between family members in their reactions to novel foods. In an early study of 81 sibling pairs aged 5-11 years and their mothers [78] and administered both child and adult versions of the Food Neophobia Scale [79]. The resemblance between mothers and children for food neophobia (was moderate to small (r=0.23) and no other significant correlations were observed. This is consistent with the results of other studies in this area with a variety of different ages of children. Galloway and colleagues [3] reported correlations of 0.2 between one hundred and ninety two 7 year old girls and their mothers on the food neophobia scale and similarly, a recent study found correlations of 0.22 for neophobia and 0.31 for pickiness between mothers and 2-6 year old child pairs [39]. Larger correlations between parents and children’s neophobia have been reported in three studies: in the first of these, all members of 57 families completed the FNS and the only significant correlation was 0.52 between mothers and children, but with no such association between fathers and children [80]. The second study of 1593 children reported correlations of 0.14-0.43 between mothers and children and 0.15-0.25 between fathers and children, the magnitude being dependent on the age of the child [80]. More recently, levels of neophobia in 63 parents and their 5 year-old children were correlated 0.48 [81]. Correlations of this magnitude are consistent with moderate to strong heritability but could equally be the result of shared home environments, especially as one study found that the correlation between levels of neophobia in mothers and fathers (who are not genetically related) was of a similar magnitude [80].

**Twin studies**

Twin studies can provide more robust estimates of the relative contribution of genes and environment to a given trait. This is achieved by comparing the similarity between monozygotic (identical) twins who share 100% of their genes with that of dizygotic (non-identical) twins, who share on average 50% of their segregating genes. If the former are more similar than the latter, the trait in question is assumed to be heritable to some extent. Model-fitting analyses provide an estimate of heritability and
provide an indication of the relative contribution of environmental factors, both shared (the same for both twins) and non-shared (particular to each twin).

In the earliest study of its kind, 3 year-old MZ and DZ twins were compared using the Colorado Children's Temperament Inventory “Reactions to food” subscale (5 items assessing both neophobic and picky eating behaviours), but there was no evidence of heritability [82], although the sample was small for these types of analyses (n=91 pairs of twins). Nevertheless, in an even smaller sample of 4-7 year-old twins (n=66 pairs), food neophobia (CFNS) was estimated to be 72% heritable [83]. The results of a recent study involving a far larger sample of 3 year-old twins from the Gemini cohort (n=1330 pairs) are consistent with this, reporting heritability estimates of 78% for food fussiness [16]. Interestingly, this study also observed strong phenotypic associations between fussiness and acceptance of fruits and vegetables, which were largely explained by common genes. In other words, the genes that influence food fussiness also drive liking for fruits and vegetables. The same group of researchers has recently published data on the aetiology of food fussiness and food neophobia in the Gemini twin cohort (n=1932 pairs) at 15 months of age; only the third study to examine the two traits in the same sample [15]. Heritability estimates were 58% for food neophobia and 46% for fussiness – rather lower than those in previous studies in older children. The authors also examined correlations between the two traits. Two previous studies reported positive correlations of r=0.19 in 7 year-old girls [49, 3] and r=0.53 in 2-6 year olds [39], and the former concluded that the predictors and consequences of the two traits differed. However, the most recent study found a higher correlation of r=0.73 between fussiness and neophobia, and a common aetiology indicated by high genetic and shared environment correlations (0.76 and 0.78 respectively) [15].

Heritability estimates have been forthcoming in studies of older children. In the largest twin study to date (TEDS: n=5390 8-11 year-old twin pairs), approximately 78% of the variance in neophobia (short CFNS) was attributable to genetic factors [84]. Genetic influences on food neophobia appear still to be operating in adulthood albeit slightly less strongly – estimates being between 61-67% in Finnish samples [85, 86].

**Intervention strategies**

Whilst genetic influences on eating behaviour are strong, a substantial proportion of the variance in fussiness and neophobia is determined by environmental factors which are more or less modifiable. The strongest predictors of children’s liking for food are sweetness and familiarity [87] and it therefore makes sense to conclude that increasing children’s familiarity with foods will make them more acceptable. ‘Mere
‘exposure’ – the process of repeatedly offering small tastes of a food on up to 14 occasions – has been the most successful means by which to increase acceptance of previously disliked or unfamiliar foods, and study after study has demonstrated its effectiveness [88, 37, 46]. In one study, parents of 2-6 year olds were asked to offer their child a small taste of a previously disliked vegetable every day for 14 days [45]. Pre- and post-intervention taste tests were conducted in the child’s home by researchers to ascertain the impact of this daily exposure on liking and intake, with statistically significant increases observed in both outcome measures.

This type of technique is highly successful when children are willing to try the foods offered, but a minority of the fussiest or most neophobic children will not entertain the idea of tasting something they do not recognise or believe that they dislike. For these children, some form of incentive may be required in order to achieve the number of taste exposures required to effect change in acceptability. A number of lab-based studies in the 1980s had investigated the impact of offering rewards to children if they ate or drank target foods or drinks [43, 89, 90]. Typical findings were that liking decreased. A parallel literature in the field of social psychology studied the effect of rewards on children’s motivation to perform a task such as drawing or puzzle-solving [91]. Again, it appeared that children’s enthusiasm for a task was reduced if they were rewarded for completing it (means-end devaluing). It seems that children may come to believe that the rewarded food is not as attractive as the food given as a reward. In contrast, a number of school-based studies had used rewards-based interventions to increase children’s acceptance of fruits and vegetables with very positive results [92, 93]. When examining these conflicting literatures, Cooke and colleagues [94] concluded that the undermining effect of rewards only occurred in certain circumstances and depended on two main issues: (i) the type of reward (ii) the initial level of liking for the target food or task.

Nevertheless, parents frequently use rewards to shape their children’s eating behaviour and many believe it to be highly effective [95, 57]. In an attempt to disentangle the effects of exposure from those of rewards, and to compare different types of reward, a pair of studies was carried out, one school-based [96] and one home-based [97]. Both compared the effect of ‘exposure plus rewards’ (both social and tangible) with exposure alone on children’s liking and intake of individually selected vegetables, and examined compliance with the tasting protocol in the different conditions. Results were very positive: increases in children’s liking and intake of target vegetables were greatest in the tangible reward condition and increases were maintained three months after the withdrawal of rewards. Importantly, compliance in
the reward condition was a great deal higher than with exposure alone, and in the school-based study, reached 100%.

In recent work carried out by Fildes and colleagues, ‘Tiny Tastes’, a set of printed materials with instructions on how to carry out the ‘exposure plus reward’ procedure, was mailed directly to parents with no researcher involvement. Again, substantial increases in vegetable acceptance were observed in the intervention group and the techniques proved popular with parents and children alike [98]. One might speculate that these interventions work by reducing food fussiness or neophobia, but none has included pre- and post-intervention measures of these traits.

**Recent intervention findings**

In contrast to the low-intensity exposure-based interventions described above, some recent large-scale programmes to improve children’s dietary habits and eating behaviours have involved considerable amounts of time and resources. In Australia, the MEND 2-4 programme comprises ten weekly 90-minute workshops involving dietary and physical activity elements for parents and their children, and includes a food exposure component [99]. Primarily an obesity prevention programme, secondary outcomes include changes in children’s food fussiness and food neophobia. No effect of the intervention was observed on either trait immediately post-intervention or at 6 months follow-up, although there was a significant reduction in food neophobia at 12-months follow-up. The authors speculated that this finding might be the attributable to the fact that parents reported continuing to use the taste exposure techniques after the intervention ended, suggesting that relatively brief interventions may be insufficient to effect permanent change in children’s attitudes to foods.

Several school-based studies have also targeted a reduction in fussiness and/or neophobia as an aim of their intervention with mixed results. In the UK, 12 months of fortnightly 90-minute kitchen classroom activities including tasting sessions failed to reduce neophobia and fussiness in primary school children aged 7-9 years [100]. In a study in the Netherlands, children with a mean age of 10 participated in five 45-minute ‘Taste Lessons’ either with or without experiential elements [101]. Whilst willingness to try foods was included as an outcome measure, no repeated tasting activity was incorporated into the intervention itself. Perhaps not surprisingly, no intervention effects were observed for neophobia or vegetable consumption. In contrast, a recent implementation of the ‘Food Dudes’ exposure, rewards and peer-modelling intervention in schools in Italy produced significant decreases in food neophobia which were sustained at 6 months follow-up [102]. It appears that straightforward taste exposure
remains the most effective means of changing responses to foods long-term, although with the fussiest children, social rewards in the form of praise or small tangible rewards, e.g. stickers might be necessary to achieve the all-important first taste.

Implications for children’s health and weight

For fussy eating and food neophobic children, the obvious concern is that their avoidance of nutrient-rich foods especially vegetables and meat may lead to nutrient deficiencies [2, 5]. On the other hand, if they restrict themselves largely to highly palatable energy-dense (e.g. sweet fatty) foods instead [66], there is a risk that their long-term eating habits could lead to increased adiposity and associated adverse health consequences [2].

Numerous studies have confirmed that fussy eaters or neophobic children consume fewer vegetables and fruit [reviewed by 5], but also less fish and meat [103, 13, 47, 104, 54]. Surprisingly few studies, however, have found evidence for nutrient-specific deficiencies [5], one exception being that fussy eating girls may have lower vitamin E and folate intake, as determined from 24-hour dietary recalls [49]. Importantly, a recent study of 7-12 year-old Chinese children found lower levels of magnesium, iron and copper in the blood of picky eaters, as well as slower growth [65]; interestingly, these picky eaters actually had higher IQ on average, adjusted for potential confounders. Nevertheless, there is some evidence that energy intake is lower in fussy eating or neophobic children [105, 64], though not necessarily in relation to body weight [106]. This is despite consistent evidence that parents of picky eaters apply greater pressure to eat [49, 50]. Also, in children under 3 years old, picky eating was related to a slight risk of being underweight (weight to length ratio) but only for 21% of 34 picky eaters, vs. 7% of non-picky eaters [107]. Furthermore, there is recent evidence from longitudinal studies that fussy eating can limit growth: where fussy eating was measured in children aged 4, BMI and fat-free mass was lower in fussy eaters when aged 6 years [108]. Similarly, in separate surveys of Canadian (n=1498) [105] and Dutch (n=4914) [54] 4-year-olds, picky/fussy eaters were nearly twice as likely to be underweight than non-picky/fussy eaters. In a large cohort study (ALSPAC), being very sensitive to bitter taste was associated with shorter height aged 10 [25]. However, another longitudinal study in 340 infants from 14 months to 3.7 years did not find any relationship between fussiness and BMI [109]. This is also consistent with an earlier study reporting a lack of relationship between neophobia and child weight [110]. In a longitudinal study tracking weight and appetitive behaviour in infants over the first year
Millennium Infant Study; 111, avoidant behaviour, some of which was apparent in most infants by 12 months, was unrelated to weight gain once differences in appetite were accounted for. However, infants gained less weight if their caregivers responded to food refusal with more pressure to eat; this might simply be caregivers responding to slow infant growth, although it is possible the pressure could actually suppress the young infants’ appetite for some foods.

Two longitudinal studies have been published recently that tracked children's growth from 5 years old, in relation to picky/fussy eating status. In Dutch children measured at age 9, picky eaters (n=403) were shorter, more likely to be underweight and less likely to be overweight than non-picky eaters (n=621) [106]. In an US sample of girls followed from age 5 to 15 years, persistent picky eaters (n=33) had lower BMI than non-fussy children (n=148) [112]. Nevertheless, an important observation was that the fussy eaters’ BMI tracked the 50th percentile, whereas the non-fussy eaters’ BMI tracked the 65th percentile. Thus, at least in girls, fussy eaters actually had on average a normal healthy BMI compared to the slightly overweight non-fussy children. Overall, the somewhat conflicting evidence suggests that fussy eating may reduce the risk of obesity and may not be a serious health concern unless the diet restriction is extreme.

Conclusion

It is clear from the evidence reviewed that fussy eating and food neophobia are very common traits, with strong genetic components but also with important influences from the environment, especially for fussiness in young children. Parents should consider such food-related behaviour in their children to be part of normal development, and avoid imposing rigid strategies that are likely to provoke conflict. In the context of our obesogenic environment and increasing prevalence of obesity, parents may draw some comfort from the observation that fussy and/or neophobic children are unlikely to become obese, and conversely are at small risk of malnutrition, despite a tendency to eat less fruit, vegetables and meats. Nevertheless, parents concerned that their child’s diet appears to be particularly limited should have their growth monitored. The most successful intervention strategies that parents can easily apply will likely combine repeated taste exposure of a slightly disliked food, in the context of positive social and emotional modelling, with the use of non-food rewards for those children whose neophobia resists even a small taste. Fussy eating typically improves gradually over years, and its impact on nutritional status can be ameliorated by such positive parental strategies, especially when applied at an early age:
dissemination of this knowledge to parents via health professionals is likely to be helpful.

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