Autism spectrum disorder and food neophobia: clinical and subclinical links

Gregory L. Wallace, PhD[1], Clare Llewellyn, PhD[2], Alison Fildes, PhD[2,3], & Angelica Ronald, PhD[4]

Affiliations: [1]Department of Speech, Language, and Hearing Sciences, The George Washington University, Washington, DC, USA; [2]Department of Behavioural Science and Health, University College London, London, UK; [3]School of Psychology, University of Leeds, Leeds, UK; [4]Centre for Brain and Cognitive Development, Birkbeck, University of London, London, UK

Authors' Last Names: Wallace, Llewellyn, Fildes, Ronald

Address correspondence to: Gregory L. Wallace, Hall of Government Room 211, 2115 G Street NW, Washington, DC 20052; gwallac1@gwu.edu; 202-994-8285

Funding Source: TEDS is funded by Medical Research Council grant G0901245, and G0500079 to Robert Plomin.

Short running head: Autism and Food Neophobia

Abbreviations: ASD: Autism Spectrum Disorder; BMI: Body Mass Index; FN: Food Neophobia; TEDS: Twin Early Development Study; SES: Socioeconomic Status; CAST: Childhood Autism Spectrum Test; DAWBA: Development and Well-Being Assessment

Abstract

Background: Autism spectrum disorder (ASD) has been linked with eating and feeding related atypicalities, including food neophobia (refusal to try unfamiliar foods), since its earliest description. Nevertheless, whether associations between ASD traits and food neophobia extend subclinically into the broader population of children and their potential additive health impacts remain unexplored. **Objective:** We examined ASD-control group differences in food neophobia and ASD trait-food neophobia trait associations as well as the ability of food neophobia and autistic traits to predict one index of later health-related outcomes (body mass index). **Design:** Participants in the present study were a large community-based sample of 8-11 year olds (n=4,564), including a relatively small group of children diagnosed with ASD (n=37). Parents of these 8-11-year-old children completed assessments of food neophobia and autistic traits, as well as providing height and weight metrics at 12 years of age. **Results:** Children with ASD were rated as more food neophobic than their same-age non-ASD peers (2.67+/-0.83 vs. 2.22 +/-0.73; $p<.001$) and there were subclinical associations between food neophobia and ASD traits (all three of social, communication, and restricted/repetitive behavior) in this community-based sample of children ($ps<.05$). Moreover, while food neophobia alone predicted lower body mass index, the interaction of food neophobia and ASD traits predicted higher body mass index ($ps\leq.01$), suggesting that elevated ASD traits in combination with food neophobia exert opposing influences on weight to food neophobia alone. **Conclusions:** These findings implicate clinical and subclinical connections between ASD traits and feeding behaviors that could impact health outcomes and therefore should be further explored in future studies of shared etiology and intervention strategy.

Keywords: autism, autistic traits, food neophobia, food selectivity, picky eating, body mass index

From its earliest description(1), autism spectrum disorder (ASD), a neurodevelopmental disorder characterized by social-communication deficits and presence of restricted and repetitive behaviors, has been linked with feeding-related problems. Even with changing diagnostic conceptualizations of ASD during the intervening 70+ years, food selectivity or 'picky eating' has remained highly prevalent among individuals with ASD, with reports of as many as 90% of children with ASD exhibiting these atypical feeding behaviors(2,3).

One core component of food selectivity is food neophobia (FN), the propensity to refuse to try unfamiliar foods. Although a normative aspect of early child development, when FN persists beyond later childhood, a dietary and nutritional cost is incurred. FN limits dietary variety with particularly adverse impacts upon consumption of nutrient-rich fruits and vegetables(4), which cascades to influence broader health and development. There is mixed evidence as to whether FN is associated with body mass index (BMI)(5,6). Moreover, efforts to explore obese versus normal weight differences in eating patterns in the laboratory have sometimes been hampered because of difficulty finding a test food that enough obese participants find acceptable(7). Data from early animal studies also have supported this observation insofar as obesity-inducing lesions in the ventromedial hypothalamus in rats produced 'finicky' behavior such that obese rats would not consume bitter-tasting food, but would overeat the highly palatable food(8). This suggests that children who are highly fussy about eating more nutrient dense and less palatable foods (e.g., fruits and vegetables) are actually more at risk of overweight/obesity than those who have a more varied diet. Taken together, this suggests that further research is needed to elucidate the relationship between FN and BMI,

particularly among school-aged children for whom the likelihood of becoming overweight/obese is increasingly common.

FN appears to be increased in ASD not only during childhood(9), but also adolescence and young adulthood(10). The most frequently utilized approach to explore links between FN and ASD is a case-control design. However, another approach involves examining individual differences in these behavioral traits and their relationships to one another, within a community-based, representative sample. There is ample evidence for the dimensionality of autistic behavior, varying from subclinical traits to clinical expression of symptoms(11-14). Thus, in the present study, we not only compared ASD and control groups in their FN but also examined how individual differences in autistic traits are predictive of individual differences in FN in a large and representative community-based sample of children. Trait-based approaches avoid biases inherent to clinical samples, including sidestepping the influences of frequently co-occurring conditions with ASD (e.g., anxiety, depression, medical, metabolic, genetic disorders). Moreover, most studies of children with ASD utilize clinic-based samples with potential concerns over representativeness of the broader population, which can bias findings and interpretation of results. Therefore, the purpose of the present investigation are threefold: 1) Establish whether FN is associated with autistic traits in a large community-based sample. 2) Examine whether FN is atypical in children with ASD in comparison to same-age peers. 3) Investigate associations between BMI and not only FN and autistic traits separately, but also their interaction.

Methods

Participants

Parents and children in the current study participated in the Twins Early Development Study (TEDS), a community-based sample of twins born between 1994-1996 in England and Wales. This sample is representative of the broader population of families with children in the United Kingdom in terms of maternal education (~38% A-levels [i.e., subject-based qualifications needed for matriculation to university] or higher) and race (~93% white). More details about TEDS can be found elsewhere(15). Children were excluded if there were reports of extreme prenatal or perinatal difficulties or severe medical disorders, sex or zygosity was unknown, or they were missing data from initial TEDS contact. Note that individuals with ASD were not excluded in order to include the full spectrum of autistic traits. As is standard for analyses requiring independent individuals, one twin per pair (regardless of whether the twin had an autism spectrum disorder) was chosen at random (based on random number generation with those selected assigned a 1 and those unselected assigned a 0) for statistical analyses described below.

Children in TEDS were screened for possible ASD diagnoses using the Childhood Autism Spectrum Test (CAST)(16) and separate questions concerning prior diagnoses of autism or Asperger Syndrome at ages 7, 8, and 9 years. Also considered were families who spontaneously contacted TEDS to report a suspicion or new diagnosis of ASD. CAST screening scores of 15 or higher and those flagged by parents (upon questioning or spontaneously) as having an ASD diagnosis were re-contacted, and phone interviewed using the ASD module of the Development and Well-Being Assessment (DAWBA)(17). The ASD module of the DAWBA has been shown to be a reliable and valid instrument to establish an ASD diagnosis. It demonstrates high correlations with both one of the gold

standard instruments in the field, the Autism Diagnostic Inventory(18,19), and 'best estimate research diagnosis' which also includes information from the Autism Diagnostic Observation Schedule and other gathered clinical information(20). Furthermore, the ASD module of the DAWBA demonstrates excellent sensitivity and specificity(20). See **Table 1** for sample demographics and supplementary Table 1 for participant flowchart. Note that this study does not prospectively assign participants to an intervention; thus, it is not a clinical trial.

Ethical approval for the study was granted by the King's College London Institute of Psychiatry ethics committee. Parents provided informed consent at each data collection wave.

Measures

Socioeconomic Status (SES)

At first contact (when twins were ~18 months old), parental education (highest qualification) and occupation (highest job status) were obtained. An SES composite score was derived by standardizing the education and occupation ratings (via the rank-based van der Waerden transformation), summing these two weighted scores and then standardizing this sum again(21).

Autistic Traits

The CAST(16) is a parent report autistic traits questionnaire designed to be completed in non-clinical settings(22). The CAST is composed of 31 questions answered in a Yes/No format and demonstrates good internal consistency in the TEDS sample (Cronbach's

alpha=0.73)(14). The CAST provides not only a Total score indicative of overall autistic traits, but also three components: Social, Communication, and Restricted/Repetitive Behavior (RRB) traits(14). The CAST data used in the present study were collected at age 8 years. Because autistic trait scores were skewed, log-transformed CAST scores were used in all analyses.

Internalizing Behavioral Traits

Internalizing traits were quantified at age 7 years using the emotional problems subscale of the Strengths and Difficulties Questionnaire (SDQ)(23), which is composed of five items (two anxiety, two depression and one somatic related behaviors) on a three-point Likert scale (never, sometimes, often) and demonstrates adequate internal consistency (Cronbach's alpha=0.63)(24).

Food Neophobia (FN)

Parent reports of FN (on a four-point-scale ranging from 'strongly agree' to 'strongly disagree') were obtained using the four-item version of the Child Food Neophobia Scale(25) when twins were 8-11 years old. Items constituting this short form of the instrument include the following: "My child is constantly sampling new and different foods" (reversed), "My child doesn't trust new foods," "My child is afraid to eat things s/he has never had before." and "If my child doesn't know what's in a food s/he won't try it." The short form of the Child Food Neophobia Scale demonstrates good reliability and validity, including high internal consistency (Cronbach's alpha=0.88)(26). FN served as

the primary dependent variable for several (i.e., t-test, chi-square, and regression) analyses described below.

Body Mass Index (BMI)

Parents reported their children's height and weight at age 12 years, which were used to calculate BMI (BMI=weight in kilograms/height in meters^2). Using the 1990 British growth reference curves, BMI standard deviation scores (M=0, SD=1 at each age) were calculated using Microsoft Excel Growth Macro software(27). BMI served as a primary dependent variable for one of the regression analyses described below. The International Obesity Task Force criteria, which identify BMI values for each age associated with predicted BMIs of 25 and 30 at 18 years of age, were used to determine underweight (non-ASD n=630; ASD n=6), healthy weight (non-ASD n=2763; ASD n=17), overweight (non-ASD n=492; ASD n=3), and obese (non-ASD n=74; ASD n=0) status(28).

Data Analysis

Analyses were carried out using SPSS 24(29). To this end, an independent samples t-test was used to examine ASD-control group differences in mean FN score. Furthermore, chi-square analysis was used to examine whether children with ASD were more likely to be food neophobic than non-ASD children. For the purposes of these analyses, children were categorized as food neophobic at three different cutoffs of the 80th, 90th, and 95th percentiles on the Child Food Neophobia Scale, given variable estimates of FN across child development.

Hierarchical multiple regressions were completed with FN score serving as the dependent variable. In order to examine more specific links between autistic traits and FN, demographic predictors (age, sex, SES) were entered in the first block, followed by the autistic traits scores in the second block. Separate regression models were run for each of the autistic traits scores (Total, Social, Communication, and Repetitive/Restricted Behavior). To ensure that the potential associations between FN and autistic traits were specific and not a product of elevated behavioral ratings overall, these same regression models were run again, with SDQ internalizing behavioral trait scores (given the link between anxiety/depression and food-related issues in the broader population(30) and those with ASD(31)) added to the first block of demographic predictors described above.

Finally, the association between age- and sex-standardized BMI at age 12 years and FN, autistic traits, and the interaction of FN and autistic traits were examined in a separate hierarchical multiple regression after accounting for the effects of demographic factors. Demographic predictors (age, sex, SES) were again entered in the first block, followed by the overall autistic traits score (using the Total CAST score), FN ratings, and the interaction of autistic traits and FN in the second block. Note that fewer families were contacted at age 12 than age 8 resulting in a smaller sample size (n=3,136) for the regression including BMI data.

Results

Children with ASD (n=37: 33 males and 4 females) were rated as demonstrating significantly more trait-based FN than the non-ASD TEDS sample (n=4564: 2221 males and 2343 females) (ASD $\underline{M}$=2.67, $\underline{SD}$=0.83 vs. non-ASD $\underline{M}$=2.22, $\underline{SD}$=0.73; t=3.73,

$p<0.001$, $d=0.57$; see **Figure 1**), and were significantly more likely to be rated as food neophobic than their non-ASD peers at all three designations of the 80th ($X^2=12.23$, $p<0.001$), 90th ($X^2=11.29$, $p=0.001$), and 95th ($X^2=12.26$, $p<0.001$) percentile scorers (see **Table 2**).

Hierarchical multiple regressions revealed several significant associations with food neophobia ratings (see **Tables 3-6**). Among the demographic factors examined in model 1, age and sex were significantly associated with FN (ps<0.05) such that younger children, and males had higher FN scores. In model 2, significant positive associations of overall autistic trait ratings with FN score, above and beyond the influence of the demographic factors (i.e., age, sex, SES) were found ($p<0.001$), along with the emergence of an association between higher SES and higher FN ratings not observed in model 1 ($p<0.05$). Follow-up hierarchical multiple regressions demonstrated that higher scores for all three components of autistic traits (CAST Social, Communication, and Repetitive/Restricted Behavior scores) were predictive of higher FN scores after taking into account these demographic factors (see Tables 3-6; ps<0.05). After adding SDQ internalizing behavioral trait ratings to the first model, subsequent hierarchical regressions revealed the same pattern of results, except that CAST Repetitive/Restricted Behavior scores were no longer a significant predictor of FN ratings.

Finally, a hierarchical multiple regression showed that even after accounting for associated demographic factors, both FN alone as well as its interaction with autistic traits were predictive of BMI at age 12 years (see **Table 7**). Specifically, higher FN was associated with having a lower BMI, while the interaction of autistic and FN trait ratings was associated with higher BMI. Two of the three demographic factors examined in

model 1 demonstrated significant associations with BMI ($ps \leq 0.01$): sex (males having lower BMI) and SES (negative correlation). In model 2, unlike autistic traits alone, higher FN ratings alone ($p<0.001$) were predictive of lower BMI at 12 years. In contrast, the interaction of FN with autistic traits ($p=0.01$) was predictive of higher BMI at age 12 years.

Discussion

This is the first study to examine links between FN and autistic behavior at the clinical and subclinical levels in a large community-based sample. Based on these findings, not only are children with ASD more likely to be food neophobic than their same-age non-ASD peers, but this relationship extends subclinically. FN was positively associated with overall autistic traits, as well as its three subcomponents (social, communication, and restricted/repetitive behavior), in a community representative sample of school-aged children. Furthermore, while autistic traits were not independently associated with body weight, FN was negatively associated with BMI. However, the interaction of FN and autistic traits was positively associated with BMI, suggesting that neophobic children who also exhibit elevated levels of autistic traits may have mitigated risk of underweight. This is both a novel and potentially clinically informative finding requiring further investigation.

This study joins many others in demonstrating atypical eating-related behaviors in ASD (for review, see(32,33)). While most other ASD-control group comparisons have examined broader concepts like food selectivity and ‘picky’ eating, the current

investigation focused on FN specifically. Thus, this study replicates and extends the few studies to demonstrate empirically increased FN in ASD, which have included samples of children of the same age and younger than those studied here(9) as well as adolescents/young adults(10). Combining the results here with these prior studies suggests that FN is a stable and persistent eating behavioral trait in ASD across child and adolescent development.

The current study also extends the relationship between ASD and FN to subclinical levels in a large and representative community-based sample. Increases in overall autistic trait ratings, as well as its three subcomponents (social, communication, and restricted/repetitive behavior) were associated with increased FN in this large community-based sample of children. The current study joins one other, which examined the relationship between separate, but related food avoidant 'picky' eating behavior (measured using two items asking parents if their child "does not eat well" or "refuses to eat"), and ASD-like behavior in a large community-based sample of young children from the Netherlands (n=3,748). Persistent 'picky' eating from 1.5-6 years was found to be predictive of ASD behavior (unlike behavioral or emotional problems) at age 7(34). Taken together, these studies indicate a broader population-wide linkage between ASD-like behavior and atypical eating patterns characterized by food avoidance. Nevertheless, longitudinal studies are needed to determine the directionality of the relationships of these two early emerging classes of behavior. Furthermore, given the early emergence of FN behaviors(35) and its linkage to ASD, its predictive power as an early marker of ASD should be further investigated.

Although autistic traits alone were not predictive of later BMI in the present study, there was evidence that autistic traits not only mitigate the association between increased FN and decreased BMI but also exert an opposing influence. One possibility is that elevated autistic traits might lessen the impact of FN on food intake. There is emerging evidence that some children with ASD exhibit a greater propensity to overeat (a risk factor for overweight/obesity in the general population) compared to typically developing children(36), in spite of the increased prevalence of co-occurring FN and other food selectivity patterns. Speculatively, within this interactive effect, increasing FN could serve to limit the dietary repertoire while increasing autistic traits could drive this limited diet towards more palatable and calorie-rich foods via sensory-related mechanisms(32), which might then counteract the negative impact of FN on BMI. Regardless, other health-related impacts of FN may be exacerbated by elevated autistic traits. FN presents barriers to adequate consumption of fruits and vegetables(4), and thus to adequate nutritional intake. Inadequate macronutrient and micronutrient intake has been observed among children with ASD(3), suggesting that autistic traits may imbue their own as well as additive risks for poor nutrition in the general population; a possibility that should be investigated in future research.

It is becoming increasingly clear that health outcomes in ASD are poor across the board with elevated rates of risk factors for cardiovascular disease (among many others) during adolescence (e.g., dyslipidemia)(37) and well into adulthood (e.g., diabetes, high blood pressure)(38). One of the most salient and well-replicated health-related risk factors in ASD is elevated rates of obesity during childhood and adolescence(39). It is possible that in the context of ASD, FN alone, and in conjunction with other factors

(behavioral, metabolic, pharmacological, etc.), leads to risk for overeating of desired foods (e.g., high fat, high carbohydrate foods) that cascades to risks of becoming overweight/obese. Learning more about the health implications of FN and related eating atypicalities in ASD and those with elevated ASD traits is critical. Unfortunately, the limited size of the ASD sample in the current study prevented us from examining associations between FN and BMI in this group, but future research, including large studies like those from the Healthy Weight Research Network(40), might endeavor to answer such unresolved questions.

Although the present study relies upon a large representative community-based sample, limitations should be considered, such as generalizability concerns. For example, twins may be more likely to experience feeding difficulties and have lower birth weights than their singleton peers. However, feeding concerns typically associated with twin births as well as lower weights would be largely resolved by the 8-12 year age range investigated here, though the group as a whole remains fairly lean. It is also important to note that autistic trait ratings do not differ for twins and singletons based on findings from at least one large study(41). Another potential limitation is the reliance upon parent report for these data. However, such an approach conveys considerable advantages to in-person testing, including enabling data collection from large samples and facilitating observation of consistently expressed behavioral traits across contexts and time, thus providing an accurate picture of everyday behavior. Finally, inclusion of other body composition indices (e.g., % body fat) and dietary information (e.g., food diaries) would have been helpful to assess potential links with nutritional intake. Future research should endeavor to address these shortcomings.

In conclusion, the current study demonstrated associations between not only an ASD diagnosis and FN but also dimensional autistic traits in the general population and FN (i.e., greater endorsement of autistic traits, more food neophobic behavior). Additionally, increased FN alone was associated with decreased BMI while the combination of increased autistic traits and increased FN was linked with increased BMI. This suggests that FN might not exert similar influences on health-related factors in the context of ASD. Further work is needed to clarify the health implications, both short-term and long-term, of FN and related food selectivity in ASD.

Acknowledgments: The authors have neither financial relationships nor conflicts of interest relevant to this article to disclose. Dr. Wallace conceptualized and designed the study, drafted the initial manuscript, and reviewed and revised the manuscript.
Drs. Ronald, Llewellyn, and Fildes conceptualized and designed the study, and critically reviewed the manuscript. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

References

1. Kanner L. Autistic disturbances of affective contact. *Nerv Child*. 1943;2:217-250.

2. Ahearn WH, Castine T, Nault K, Green G. An assessment of food acceptance in children with autism or pervasive developmental disorder-not otherwise specified. *J Autism Dev Disord*. 2001;31:505-511.

3. Sharp WG, Berry RC, McCracken C, Nuhu NN, Marvel E, Saulnier CA, Klin A, Jones W, Jaquess DL. Feeding problems and nutrient intake in children with autism spectrum disorders: A meta-analysis and comprehensive review of the literature. *J Autism Dev Disord*. 2013;43:2159-2173.

4. Oliveira A, Jones L, de Lauzon-Guillain B, Emmett P, Moreira P, Charles MA, Lopes C. Early problematic eating behaviours are associated with lower fruit and vegetable intake and less dietary variety at 4-5 years of age. A prospective analysis of three European birth cohorts. *Br J Nutr*. 2015;114:763-771.

5. Cole NC, An R, Lee S, Donovan SM. Correlates of picky eating and food neophobia in young children: a systematic review and meta-analysis. *Nutr Rev.* 2017;75:516-532.

6. Webber L, Hill C, Saxton J, Van Jaarsveld CHM, Wardle J. Eating behaviour and weight in children. *Int J Obes*. 2009;33:21-28.

7. Guss J, Kissileff H. Microstructural analyses of human ingestive patterns: From description to mechanistic hypotheses. *Neurosci Biobehav Rev*. 2000;24:261-268.

8. Schachter S. Some extraordinary facts about obese humans and rats. *Am Psychol.* 1971;26:129-144

9. Martins, Y., Young, R. L., & Robson, D. C. (2008). Feeding and eating behaviors in children with autism and typically developing children. *J Autism Dev Disord*. *38*, 1878-1887.

10. Kuschner ES, Eisenberg IW, Orionzi B, Simmons WK, Kenworthy L, Martin A, Wallace GL. A preliminary study of self-reported food selectivity in adolescents and young adults with autism spectrum disorder. *Res Autism Spectr Disord*. 2015;15-16:53-59.

11. Lundström S, Chang Z, Råstam M, Gillberg C, Larsson H, Anckarsäter H, Lichtenstein P. Autism spectrum disorders and autistic like traits: similar etiology in the extreme end and the normal variation. *Arch Gen Psychiatry*. 2012;69:46-52.

12. Robinson EB, Koenen KC, McCormick MC, Munir K, Hallett V, Happé F, Plomin R, Ronald A. Evidence that autistic traits show the same etiology in the general population and at the quantitative extremes (5%, 2.5%, and 1%). *Arch Gen Psychiatry*. 2011;68:1113-1121.

13. Robinson EB, St Pourcain B, Anttila V, Kosmicki JA, Bulik-Sullivan B, Grove J, Maller J, Samocha KE, Sanders SJ, Ripke S, Martin J, Hollegaard MV, Werge T, Hougaard DM, iPSYCH-SSI-Broad Autism Group, Neale BM, Evans DM, Skuse D, Mortensen PB, Børglum AD, Ronald A, Smith GD, Daly MJ. Genetic risk for autism spectrum disorders and neuropsychiatric variation in the general population. *Nat Genet*. 2016;48:552-555.

14. Ronald A, Happé F, Bolton P, Butcher LM, Price TS, Wheelwright S, Baron-Cohen S, Plomin R. Genetic heterogeneity between the three components of the autism spectrum: a twin study. *J Am Acad Child Adolesc Psychiatry*. 2006;45:691-699.

15. Haworth CM, Davis OS, Plomin R. Twins Early Development Study (TEDS): a genetically sensitive investigation of cognitive and behavioral development from childhood to young adulthood. *Twin Res Hum Genet*. 2013;16:117-125.

16. Scott FJ, Baron-Cohen S, Bolton P, Brayne C. The CAST (Childhood Asperger Syndrome Test): preliminary development of a UK screen for mainstream primary-school-age children. *Autism*. 2002;6:9-31.

17. Dworzynski K, Happé F, Bolton P, Ronald A. Relationship between symptom domains in autism spectrum disorders: A population based twin study. *J Autism Dev Disord*. 2009;39:1197-1210.

18. Le Couteur A, Rutter M, Lord C, Rios P, Robertson S, Holdgrafer M, McLennan J. Autism Diagnostic Interview - a standardized investigator-based instrument. *J Autism Dev Disord*. 1989;19:363-387.

19. Lord C, Rutter M, Le Couteur A. Autism Diagnostic Interview-Revised – a revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *J Autism Dev Disord*. 1994;24:659-685.

20. McEwen FS, Stewart CS, Colvert E, Woodhouse E, Curran S, Gillan N, Hallett V, Lietz S, Garnett T, Ronald A, Murphy D, Happé F, Bolton P.
Diagnosing autism spectrum disorder in community settings using the Development and Well-Being Assessment: Validation in a UK population-based twin sample. *J Child Psychol Psychiatry*. 2016;57:161-70.

21. Hanscombe KB, Trzaskowski M, Haworth CMA, Davis OSP, Dale PS, Plomin R. Socioeconomic status (SES) and children's intelligence (IQ): In a UK-representative sample SES moderates the environmental, not genetic, effect on IQ. *PLoS One*. 2012;*7*.

22. Williams J, Scott F, Stott C, Allison C, Bolton P, Baron-Cohen S, Brayne C. The CAST (Childhood Asperger Syndrome Test): Test accuracy. *Autism*. 2005;9:45-68.

23. Goodman R, Ford T, Simmons H, Gatward R, Meltzer H. Using the Strengths and Difficulties Questionnaire (SDQ) to screen for child psychiatric disorders in a community sample. *Br J Psychiatry*. 2000;177:534-539.

24. Hallett V, Ronald A, Rijsdijk F, Happé F. Association of autistic-like and internalizing traits during childhood: A longitudinal twin study. *Am J Psychiatry*. 2010;167:809-817.

25. Pliner P, Hobden K. Development of a scale to measure the trait of food neophobia in humans. *Appetite*. 1992;19:105-120.

26. Cooke L, Haworth CM, Wardle J. Genetic and environmental influences on children's food neophobia. *Am J Clin Nutr*. 2007;86:428-433.

27. Cole TJ, Freeman JV, Preece MA. Body mass index reference curves for the UK, 1990. *Arch Dis Child*. 1995;73:25-29.

28. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: International survey. *BMJ*. 2000;20:1240-1243.

29. IBM Corp. IBM SPSS statistics for windows (Version 24). 2016;Armonk, NY: IBM Corp.

30. American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th Ed.). Washington, DC: Author.

31. Twachtman-Reilly J, Amaral SC, Zebrowski PP. Addressing feeding disorders in children on the autism spectrum in school-based settings: physiological and behavioral issues. *Lang Speech Hear Serv Sch*. 2008;39:261-272.

32. Cermak SA, Curtin C, Bandini LG. Food selectivity and sensory sensitivity in children with autism spectrum disorders. *J Am Diet Assoc*. 2010;110:238-246.

33. Kral TV, Eriksen WT, Souders MC, Pinto-Martin JA. Eating behaviors, diet quality, and gastrointestinal symptoms in children with autism spectrum disorders: a brief review. *J Pediatr Nurs*. 2013;28:548-556.

34. Cardona Cano S, Hoek HW, van Hoeken D, de Barse LM, Jaddoe VW, Verhulst FC, Tiemeier H. Behavioral outcomes of picky eating in childhood: a prospective study in the general population. *J Child Psychol Psychiatry*. 2016;57:1239-1246.

35. Blossfeld I, Collins A, Kiely M, Delahunty C. Texture preferences of 12-month-old infants and the role of early experiences. *Food Qual Prefer*. 2007;18:396-404.

36. Hess JA, Matson JL, Dixon DR. Psychiatric symptom endorsements in children and adolescents diagnosed with autism spectrum disorders: a comparison to typically developing children and adolescents. *J Dev Phys Disabil*. 2010;22:485-496.

37. Davignon MN, Yinge Q, Massolo M, Croen L. Psychiatric and medical conditions in transition-aged individuals with ASD. *Pediatrics*. 2018;141:S335-S345.

38. Croen LA, Zerbo O, Qian Y, Massolo ML, Rich S, Sidney S, Kripke C. The health status of adults on the autism spectrum. *Autism*. 2015;19:814-823.

39. Phillips KL, Schieve LA, Visser S, Boulet S, Sharma AJ, Kogan MD, Boyle CA, Yeargin-Allsopp M. Prevalence and impact of unhealthy weight in a national sample of US adolescents with autism and other learning and behavioral disabilities. *Maternal and Child Health Journal*. 2014;18:1964-1975.

40. Curtin C, Must A, Phillips S, Bandini L. The healthy weight research network: a research agenda to promote healthy weight among youth with autism spectrum disorder and other developmental disabilities. *Pediatr Obes*. 2017;12:e6-e9.

41. Curran S, Dworzynski K, Happé F, Ronald A, Allison C, Baron-Cohen S, Brayne C, Bolton PF. No major effect of twinning on autistic traits. *Autism Res*. 2011;4:377-382.

1

Table 1. Demographic characteristics and summary scores for the study groups: mean (standard deviation) and range.

	Non-ASD Sample (max n=4,564)		ASD Sample (max n=37)	
	Mean (SD)	Range	Mean (SD)	Range
Age	9.88 (0.87)	8.32-11.61	10.05 (0.91)	8.67-11.39
SES	0.26 (0.97)	-2.49-2.65	0.37 (0.99)	-1.54-2.03
Mean Food Neophobia Score	2.22 (0.73)	1-4	2.67 (0.83)	1-4
CAST Total	4.91 (3.25)	0-19	17.08 (4.09)	3-28
CAST Social	1.56 (1.49)	0-11	6.22 (2.58)	1-11
CAST Communication	1.91 (1.76)	0-10	7.06 (2.23)	1-11
CAST RRB	1.45 (1.23)	0-7	3.81 (1.63)	0-7
SDQ Internalizing Behaviors	2.26 (1.88)	0-10	2.14 (2.01)	0-7
BMI	17.77 (3.01)	12.08-39.39	16.84 (2.60)	12.82-22.48

Note: SES=Socioeconomic Status; CAST=Childhood Autism Spectrum Test; RRB=Restricted/Repetitive Behavior; SDQ=Strengths and Difficulties Questionnaire; BMI=Body Mass Index

Table 2. Food neophobia rates at various cutoff scores on the Child Neophobia Scale for the autism spectrum disorder (ASD) and non-ASD general community samples.

	80th Percentile Scorers (Neophobic:Not Neophobic)*	90th Percentile Scorers (Neophobic:Not Neophobic)*	95th Percentile Scorers (Neophobic:Not Neophobic)*
Non-ASD Sample	915:3649	464:4100	314:4250
ASD Sample	16:21	10:27	8:29

Note: Analysis completed using Chi-square. *$ps \leq 0.001$

Table 3. Food neophobia ratings regressed onto age, sex, socioeconomic status, and overall autistic trait ratings.

n=4,245	R^2	*F* Change	*B*	SE *B*	*t*	*p*
Predictor	Food Neophobia					
Model 1	0.007	9.91				
Age			-0.03	0.01	-2.11	0.04
Sex			0.11	0.02	4.74	<0.001
SES			0.02	0.01	1.46	0.15
Model 2	0.015	32.90				
Age			-0.03	0.01	-1.98	0.05
Sex			0.08	0.02	3.43	0.001
SES			0.03	0.01	2.44	0.02
CAST Total			0.26	0.05	5.74	<0.001

Note: Analysis completed using hierarchical multiple regression. SES=Socioeconomic Status; CAST=Childhood Autism Spectrum Test; 1=male, 0=female

Table 4. Food neophobia ratings regressed onto age, sex, socioeconomic status, and autistic social trait ratings.

n=4,246	R^2	*F* Change	*B*	SE *B*	*t*	*p*
Predictor	Food Neophobia					
Model 1	0.007	9.94				
Age			-0.03	0.01	-2.11	0.04
Sex			0.11	0.02	4.75	<0.001
SES			0.02	0.01	1.46	0.14
Model 2	0.011	15.60				
Age			-0.03	0.01	-2.11	0.04
Sex			0.08	0.02	3.56	<0.001
SES			0.02	0.01	1.79	0.07
CAST Social			0.19	0.05	3.95	<0.001

Note: Analysis completed using hierarchical multiple regression. SES=Socioeconomic Status; CAST=Childhood Autism Spectrum Test; 1=male, 0=female

Table 5. Food neophobia ratings regressed onto age, sex, socioeconomic status, and autistic communication trait ratings.

n=4,245	R^2	*F* Change	*B*	SE *B*	*t*	*p*
Predictor	Food Neophobia					
Model 1	0.007	9.90				
Age			-0.03	0.01	-2.12	0.03
Sex			0.11	0.02	4.74	<0.001
SES			0.02	0.01	1.45	0.15
Model 2	0.016	36.99				
Age			-0.03	0.01	-2.02	0.04
Sex			0.09	0.02	4.11	<0.001
SES			0.03	0.01	2.47	0.01
CAST Communication			0.26	0.04	6.08	<0.001

Note: Analysis completed using hierarchical multiple regression. SES=Socioeconomic Status; CAST=Childhood Autism Spectrum Test; 1=male, 0=female

Table 6. Food neophobia ratings regressed onto age, sex, socioeconomic status, and autistic repetitive behavior trait ratings.

n=4,238	R^2	*F* Change	*B*	SE *B*	*t*	*p*
Predictor	Food Neophobia					
Model 1	0.007	9.99				
Age			-0.03	0.01	-2.10	0.04
Sex			0.11	0.02	4.77	<0.001
SES			0.02	0.01	1.47	0.14
Model 2	0.008	4.42				
Age			-0.03	0.01	-2.02	0.04
Sex			0.10	0.02	4.58	<0.001
SES			0.02	0.01	1.66	0.10
CAST RRB			0.10	0.05	2.10	0.04

Note: Analysis completed using hierarchical multiple regression. SES=Socioeconomic Status; CAST=Childhood Autism Spectrum Test; 1=male, 0=female

Table 7. Body mass index standard deviation scores at 12 years regressed onto age, sex, socioeconomic status, overall autistic trait ratings, food neophobia ratings, and the interaction of autistic traits and food neophobia scores.

n=3,136	R^2	*F* Change	*B*	SE *B*	*t*	*p*
Predictor	Body Mass Index Standard Deviation Scores at 12 years					
Model 1	0.012	13.19				
Age			-0.03	0.03	-1.23	0.22
Sex			0.15	0.04	3.31	0.001
SES			-0.12	0.02	-5.30	<0.001
Model 2	0.021	9.05				
Age			-0.03	0.03	-1.37	0.17
Sex			-0.15	0.05	3.26	0.001
SES			-0.11	0.02	-4.90	<0.001
CAST Total			-0.50	0.28	-1.78	0.08
Food Neophobia			-0.35	0.09	-3.75	<0.001
CAST Total x Food Neophobia			0.29	0.12	2.43	0.01

Note: Analysis completed using hierarchical multiple regression. SES=Socioeconomic Status; CAST=Childhood Autism Spectrum Test; 1=male, 0=female

Figure Legend.

Figure 1. Significant differences in food neophobia scores for the autism spectrum disorder (ASD; n=37) and non-autism spectrum disorder (non-ASD; n=4,564) groups.

Note: Analysis was completed using an independent samples t-test.