Appetite 195 (2024) 107230

Contents lists available at ScienceDirect

Appetite

journal homepage: www.elsevier.com/locate/appet

Heritability of adult picky eating in the Netherlands

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ARTICLE INFO

Keywords:
Adult picky eating
Heritability
Twin studies
Food preference questionnaire

ABSTRACT

Adult picky eating (APE), the rejection of familiar and unfamiliar foods leading to a diet with limited variety, is an understudied phenomenon which can have both physical and psychological negative consequences. The aetiology of individual differences in APE is understudied, although there is reason to believe that it is partly heritable. Therefore, we aimed to estimate the heritability of APE with data from the Netherlands Twin Register ($n = 8016$) with classical genetic structural equation modelling. In order to use these data, we firstly investigated whether a Food Preference Questionnaire (FPQ) could measure APE with a pre-registered prestudy. Adult participants ($n = 414$) filled in online questionnaires, including a FPQ and measures related to APE. Spearman's rho correlation quantified the relationship between different elements of the Dutch FPQ and different scores on measures of APE. Results of the prestudy showed that the mean liking score on the FPQ could be used to measure APE ($\rho > .50$). This measure was then used in the main study to estimate the heritability of APE. Results showed that broad-sense heritability for APE is 49 % (additive genetic effects 14 % (95 % CI [00, 38]) + dominance genetic effects 35 % (95 % CI [11, 52]), while the remaining variance is explained by unique environmental factors. Future studies may focus on uncovering the specific genetic and unique environmental factors that play a role in APE.

1. Introduction

Picky eating is generally defined as the rejection of both familiar and unfamiliar foods, leading to a diet with limited variety (Ellis, Galloway, Webb, & Martz, 2017; Thompson, Cummins, Brown, & Kyle, 2015). It is different from food neophobia, which is the fear and rejection of specifically novel foods (Ellis et al., 2017). However, picky eating and food neophobia correlate (Elkins & Zickgraf, 2018), and some scholars argue that food neophobia is a component or subset of picky eating (Dovey, Staples, Gibson, & Halford, 2008). Picky eating might be a subclinical manifestation of the American Psychiatric Association's (2013) *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*) disorder Avoidant/Restrictive Food Intake Disorder (ARFID; Kauer, Pelchat, Rozin, & Zickgraf, 2015). To be diagnosed with ARFID, individuals also need to experience significant interference with psychosocial functioning and health consequences, such as weight loss or nutrient deficiency (American Psychiatric Association, 2013). Although picky eating seems to be a normal phase in development during young childhood (Cardona Cano et al., 2015), in some cases, it persists into adulthood. In addition, picky eating can also have a late onset in adolescence or young adulthood (Van Tine, McNicholas, Safer, & Agras, 2017). The prevalence of picky eating in adulthood is estimated between 18 % and 46 % in different samples from the United States (Dial et al., 2021; Ellis, Zickgraf et al., 2018; Kauer et al., 2015; Van Tine et al., 2017; Zickgraf & Schepps, 2016).

Although not as severe as in individuals diagnosed with ARFID, adult picky eaters can experience both psychological and physical negative consequences because of their eating patterns. Specifically, adult picky eaters have heightened social anxiety (Wildes, Zucker, & Marcus, 2012), especially around social food settings (Dial et al., 2021; Kauer et al., 2015; Thompson et al., 2015). More generally, adult picky eating behavior predicts an impaired quality of life (Fox, Coulthard, Williamson, & Aldridge, 2023). The physical problems adult picky eaters experience mostly relate to low dietary variety, especially with regards

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https://doi.org/10.1016/j.appet.2024.107230
Received 27 October 2023; Received in revised form 21 December 2023; Accepted 18 January 2024
Available online 24 January 2024

to fruit and vegetable intake (Dial et al., 2021; Ellis, Galloway, et al., 2018; Kauer et al., 2015; Zickgraf & Schepps, 2016), which may increase the risk of folate and iron deficiency (especially in females, Nagao-Sato et al., 2023), cardiovascular diseases (Wang et al., 2014) and might lower the life-expectancy (Zhan et al., 2017).

Relatively little is known about the aetiology of individual variation in adult picky eating (APE). In a retrospective study, parental pressure to eat is positively correlated with APE, whilst on the other hand parental positive encouragement to eat is related to lower later picky eating (Ellis, Schenk, et al., 2018; Zohar, 2022). Recollections of early negative experiences with food also are associated with APE (Ellis, Schenk, et al., 2018). At the same time, participants describe feeling a disgust response when encountering "unsafe" foods (Thompson et al., 2015). This is in line with the more biological finding that disgust sensitivity plays a role in APE (Egolf, Siegrist, & Hartmann, 2018; Ellis, Schenk, et al., 2018; Harris et al., 2019; Kauer et al., 2015). Cognitive rigidity is also thought to play a role in picky eating, in both typically and non-typically developing children and young adult college students (Zickgraf, Richard, Zucker, & Wallace, 2022). Adult picky eaters often reject foods based on texture (Fox, Coulthard, Williamson, & Wallis, 2018). More specifically, oral texture sensitivity correlates moderately with selective eating in both clinical and non-clinical samples of children and young adults (Nederkoorn, Houben, & Havermans, 2019; Zickgraf et al., 2022). Lastly, self-identified adult picky eaters rate the taste of bitter and sour foods as more intense, leading authors to conclude that taste sensitivity might also play a role in APE (Kauer et al., 2015). Thus, it seems that sensitivity to sensory properties of food, as well as early food experiences, contribute to the development of APE.

But where do these sensitivities come from? Taste sensitivity is partly influenced by genetic factors, with estimations of the heritability for sensitivity to bitter tastes at around 30 % (Hansen, Reed, Wright, Martin, & Breslin, 2006) and sour tastes at around 50 % (Wise, Hansen, Reed, & Breslin, 2007). The same holds for disgust sensitivity; genetic factors explain approximately 50 % of the variation (Sherlock, Zietsch, Tybur, & Jern, 2016). The heritability of cognitive flexibility, the opposite of cognitive rigidity, is estimated to be around 40 % (Guimaraes et al., 2020). Furthermore, adult food neophobia is highly heritable with heritability estimates of 61–69 % (Knaapila et al., 2007, 2011). More generally, several twin studies show that patterns of food preference or liking (for example for fruit and vegetables, meat or sweet snacks) are moderately heritable indicating that genetic factors influence food liking-disliking (Pallister et al., 2015; Vink et al.,2020; Van Hooijdonk, Willemsen, Feskens, & Boomsma, 2020). Childhood picky eating, which in childhood literature is often called food fussiness, has heritability estimates above 70 % (Fildes, van Jaarsveld, Cooke, Wardle, & Llewellyn, 2016; Nas et al., 2023). Currently, however, it is not clear whether genetic factors also play a role in APE behaviour and if so, to what extent. Therefore, this study aimed to investigate the heritability of APE using existing data from the Netherlands Twin Register (NTR, Boomsma et al., 2006; Willemsen et al., 2013).

APE can be measured by a variety of questionnaires (Cardona Cano et al., 2015). Early research into APE simply asked participants to self-identify as picky eater (e.g., Kauer et al., 2015; Thompson et al., 2015). In 2017, the Adult Picky Eating Questionnaire (APEQ) was developed and validated, which consists of 16 questions divided in four subscales: Meal Presentation, Food Variety, Meal Disengagement, and Taste Aversion (Ellis et al., 2017). In 2018, the Nine-Item ARFID Screen (NIAS) was developed, with three subscales, one of which was a picky eating subscale (Zickgraf & Ellis, 2018). To measure food neophobia, a correlate of APE, the leading, most-used instrument (Rabadán & Bernabéu, 2021) is the Food Neophobia Scale (FNS; Pliner & Hobden, 1992). A Food Preference Questionnaire has also been used to measure APE (Nederkoorn et al., 2019), but without validation or theoretical substantiation. In our twin sample, a Food Preference Questionnaire (FPQ) was completed by the participants. We first did a prestudy to explore whether (subscores) of the FPQ can be used to measure APE by comparing the FPQ scores with the scores on other APE questionnaires in a Dutch sample of adults (comparable to the twin sample).

In conclusion, this study focused on two research questions. First, how do scores derived from an extensive Food Preference Questionnaire relate to other, previously validated measures of adult picky eating (prestudy)? We expected that the sub scores on the FPQ (mean liking score, low proportion score, sour/bitter liking, percentage never tried) and disgust (from another questionnaire) would correlate highly (pre-registered $r > 0.50$) with the following elements of APE: level of adult picky eating, overall disliking of food, taste aversion of bitter and sour tastes, food variety and food neophobia, derived from the validated measures APEQ, NIAS, and mFNS. This was investigated in a sample of Dutch adult participants who completed online questionnaires. Second, what is the heritability of the APE-related sub scores derived from the Food Preference Questionnaire (main study). This was investigated with classical genetic structural equation modelling on existing twin data from the NTR (Boomsma et al., 2006; Willemsen et al., 2013). Based on the heritability of traits similar to APE, such as described above, we expected a significant contribution of genetic factors on APE.

2. Prestudy

2.1. Methods of prestudy

2.1.1. Pre-registration

This study's desired sample size (based on power calculation), measured variables, hypotheses, and planned analyses were pre-registered on Open Science Framework (https://osf.io/wp4zx/) prior to any data being collected.

2.1.2. Participants

The recruitment period was from February 7th until March 30th' 2023. Participants were recruited through the personal and professional networks of the involved researchers, through posts on LinkedIn and Facebook. We used Facebook advertisement to reach a broad audience and additionally posted on two Facebook groups for (adult) picky eaters. Lastly, participants were recruited through the Radboud SONA System, an online platform where students and other people interested in participating in research can sign up for studies. 15 gift vouchers for a well-known Dutch online shop, each worth €25,-, were raffled among participants who completed the whole survey. Students who signed up through Radboud SONA Systems could also receive course credit. In total, we recruited 574 Dutch participants, but 160 participants had to be excluded due to not fulfilling the inclusion criteria. The inclusion criteria were: 1) participants had to give informed consent, both about the questionnaire as a whole as well as specifically consenting to giving out information on weight, height and any previous history of eating disorder(s) ($N = 127$ participants excluded) and 2) participants had to complete the entire questionnaire ($N = 33$ participants excluded). Participants with (history of) an eating disorder were not excluded. Since the questions in Qualtrics were mandatory and participants who were under 18 were automatically led to the end of the questionnaire, all participants who finished the survey had complete data without missing values and were 18 or older, so no additional participants were excluded. The final sample consisted of 414 participants. This sample size is sufficient according to the pre-registered power calculation, which indicated that a minimum of between 247 (power of 80 %) and 386 (power of 95 %) participants was needed (https://osf.io/wp4zx/). The mean age of the participants was 35.0 years old ($SD = 15.5$, range 18–86, median 30.0). The sample consisted of more females (76.8 %) than males. More information on demographics can be found in Supplementary Table S1.

2.1.3. Materials

A survey was composed, consisting of several existing questionnaires that assess (elements of) APE. Questionnaires that were not available in

Dutch (i.e. mFNS, picky eating subscale of the NIAS, APEQ) were translated into Dutch following a standard forward-backward procedure, whereby the scale was first translated from English to Dutch by the main researcher, and afterwards translated back from Dutch to English by an independent researcher who is a native Dutch speaker and has lived in the USA.

2.1.3.1. Self-identification as a picky eater. We asked participants whether they considered themselves a picky eater (yes/no), and why (open answer). This was based on Thompson et al. (2015) and Kauer et al. (2015), who used this self-identification method to determine whether participants in their study were adult picky eaters.

2.1.3.2. Modified Food Neophobia Scale. The modified version of the Food Neophobia scale (mFNS) is used. The original 10-item version is developed by Pliner and Hobden (1992). The modified version developed by Elkins and Zickgraf (2018) includes 5-items ($\alpha = 0.87$) with questions such as "I am afraid to eat things I have never had before", rated on a 5-point scale ranging from *Strongly disagree* to *Strongly agree*. Items 1 and 3 were reverse-coded before the mean score of the questionnaire was calculated. A higher score indicated more food neophobia symptoms.

2.1.3.3. Picky eating subscale of the Nine-Item ARFID Screening. The Nine-Item ARFID Screening (NIAS) measures three aspects of food avoidance: picky eating, poor appetite and fear of negative consequences (Zickgraf & Ellis, 2018). In the current study, only the ***picky eating subscale*** was used ($\omega = 0.87$ in original validation study; Zickgraf & Ellis, 2018). This subscale consists of three questions such as "I dislike most of the foods that other people eat", rated on a 6-point Likert scale ranging from *Strongly disagree (0)* to *Strongly agree (5)*. The sum score (0-15) on the three items was calculated.

2.1.3.4. Adult Picky Eating Questionnaire. The Adult Picky Eating Questionnaire (APEQ) consists of 16 items measuring four different aspects of picky eating: Meal Presentation ($\alpha = 0.79$), Food Variety ($\alpha = 0.77$), Meal Disengagement ($\alpha = 0.81$), and Taste Aversion ($\alpha = 0.84$; Ellis et al., 2017). In the current study, all four aspects were measured, to be able to calculate an overall ***mean APEQ*** score. In addition, the mean scores on the subscales ***Food Variety*** and ***Taste Aversion*** were used. The questions are answered on a 5-point Likert scale ranging from *Never* to *Always*, with questions such as "I prefer foods of a particular color" (subscale Meal Presentation), "My usual diet lacks a variety of food groups" (subscale Food Variety), "I usually feel that I have something better to do than eating" (subscale Meal Disengagement), and "I reject bitter foods, even if they are only slightly bitter" (subscale Taste Aversion).

2.1.3.5. Food preference questionnaire. The Food Preference Questionnaire (FPQ) by Duffy et al. (2007) was used, with modifications to better suit the Dutch population (as described in Vink et al., 2020). The questionnaire consisted of 103 different items, of which 71 food, 14 drinks, and 18 activities. The activities were not included in the current study. For each item, participants had to indicate how much they liked the item on a scale of 0 (*Strong dislike*) to 10 (*Strong like*), or check the option *Never tried/Not applicable*.

From the FPQ, four different scores were composed. First, the mean liking score of all food and drink items per person was used, excluding items where the participant indicated "Never tried". This score was called "**FPQ mean liking**". In addition, a score called "**FPQ Low Proportion**" was used, for which it was counted how often participants indicated that they have a strong aversion (either score 0, 1 or 2) towards a food or drink item (i.e., that they strongly dislike that item), and this amount was divided by the total number of items for which participants gave a liking score, excluding items where the participant indicated "Never tried". Thirdly, out of all items from the FPQ we selected six that were deemed bitter or sour, based on two criteria: either, their sour or bitter taste intensity score in a Dutch taste database (Mars, de Graaf, Teo, & van Langeveld, 2020) was higher than 50 (on a scale of 0–100), and/or they were rated as "definitely sour/bitter" by three researchers who independently rated all items. The mean liking score on these six items (pickles, beer, black coffee, vinegar, lemon, and grapefruit), excluding items where the participant indicated "Never tried", was called "**FPQ Sour/bitter liking**". Fourth, it was counted how often participants indicated they never tried a food/drink item, and this score was called "**FPQ never tried**".

2.1.3.6. Avoidance of Food Groups Questionnaire. This questionnaire was developed by the NTR and also used in research before (e.g., Wesseldijk, Tybur, Boomsma, Willemsen, & Vink, 2023). Participants were asked "Do you eat this food or not?", and "if not, for what reasons". This was called Avoidance of Food Groups Questionnaire (AFG). The 12 food groups were: fish, shellfish, red meat, pork, poultry, gluten, dairy, eggs, sweets, soy, nuts, and alcohol. Participants could choose multiple reasons for avoiding these food groups: allergic, intolerant, sensitive, illness, disgust, health, weight loss, beliefs: vegetarianism/veganism or beliefs: religion (in the original questionnaire, this was one option called "beliefs (eg religion, veganism)", which was split up in the current study), or the option "other reason". We used the AFG to count how often a participant indicated that they did not eat a certain food group because of disgust. This score was called "AFG disgust".

For all questionnaires, the Omega Total (McDonald, 1999) was calculated using the psych package (version 2.2.9; Revelle, 2022) in R Statistical Software (version 4.1.1; R Core Team, 2021). This was done to determine scale reliability instead of using Cronbach's alpha, as advised by McNeish (2018) for congeneric scales. All questionnaires had good to excellent reliability with omega ranging between 0.73 and 0.96, except the subscale Taste Aversion ($\omega^t = 0.61$; see Table 1).

2.1.4. Procedure

This prestudy was carried out in accordance with the guidelines of the Ethics Committee of the Faculty of Social Science of the Radboud University, outlined in protocol [ECSW-LT-2022-11-15-37361]. Data were collected with Qualtrics software. Participants completed the questionnaire online. The median time to complete the survey was a little over 13 min. First, they received an information letter and informed consent form.

After providing active consent, participants received a trigger warning, in which it was explained that the questionnaire would concern behaviour, thoughts and feelings surrounding food and food preferences, and that this could elicit unpleasant feelings or memories. Participants could leave the questionnaire at any time and some Dutch resources/websites were listed where participants could get more information or help regarding their eating behaviour.

First, participants were asked for some demographic characteristics (i.e., age, gender, weight, height, highest completed education). Then, they received the food questionnaires in the order as presented in the Materials section. At the end of the survey, participants were asked to indicate any food allergies, whether they were vegetarian or vegan, and

Table 1
Omega Total Internal Reliability Scores for the Questionnaires in the prestudy.

Scale	ω^t [CI]
Modified Food Neophobia Scale (mFNS)	.86 [.84, .92]
Nine-Item ARFID Screen (NIAS) Picky Eating subscale	.84 [.82, .92]
Adult Picky Eating Questionnaire (APEQ)	.90 [.89, .95]
- APEQ Food Variety	.76 [.66, .80]
- APEQ Taste Aversion	.61 [.14, .70]
Food Preference Questionnaire (FPQ)	.96 [.96, .98]

Note. CI = Confidence Interval at the 95% level, acquired through bootstrapping.

whether they currently had an eating disorder (if yes, which one) or whether they had had an eating disorder in the past (if yes, which one).

2.1.5. Analyses

All data analyses were performed in R Statistical Software (version 4.1.1; R Core Team, 2021). BMI was calculated by dividing the weight in kilograms by the squared height in meters. We checked assumptions of Pearson's correlation test by plotting the computed scores in a density plot, by checking for outliers with a boxplot, and by plotting the relevant scatterplots. The psychometric characteristics of all questionnaire scores can be found in Supplementary Table S3. Visual inspection showed that all relevant scores deviated severely from normality, except the mean liking score of the FPQ, as can be seen in the density plots (Figs. S1–S7). As a consequence, we used different statistical tests than pre-registered (see below).

2.1.5.1. Deviations from preregistration. We performed Spearman's rho correlations instead of Pearson's correlations as most APE-related scores, except the FPQ mean liking, were not normally distributed (See Supplemental File, Figs. S1–S10), had outliers (See Supplemental Table S3), and transformation of the scores did not improve normality. In addition, it was pre-registered that we would perform two independent sample t-tests with the self-identified picky eating score. However, two alternatives were chosen. First, a Welch's *t*-test was chosen to determine whether self-identified picky eaters scored significantly lower on the FPQ mean liking score than participants who did not self-identify as a picky eater, because the two groups differed in size. Second, a Mann Whitney *U* test was chosen to test whether self-identified picky eaters scored significantly higher on the FPQ low proportion score than participants who did not self-identify as picky eater because the FPQ Low Proportion score was not normally distributed.

An overview of the tests performed between the five hypothesized elements of APE and the validated APE measures can be found in Table 2. We only correlated the FPQ and AFG measures with other, validated APE measures if we suspected a high correlation based on similarities regarding content (as preregistered). Using these tests, we assessed 1) whether the mean liking score of the FPQ could be used to measure the level of adult picky eating; 2) whether the low proportion score of the FPQ could be used to measure overall disliking of food; 3) whether the sour/bitter liking score of the FPQ could be used to measure taste aversion of bitter and sour tastes; 4) whether the FPQ 'never tried' score or the AFG disgust score could be used to measure (lack of) food variety; and 5) whether the FPQ 'never tried' score could be used to measure food neophobia.

Table 2
Associations Tested Between the Study Measures in the prestudy.

	FPQ mean liking	FPQ Low Proportion	FPQ sour/ bitter liking	FPQ never tried	AFG disgust
APEQ total mean	Adult picky eating [a]	Overall disliking of food [a]			
NIAS picky eating subscale sum score	Adult picky eating [a]	Overall disliking of food [a]			
APEQ Taste Aversion subscale mean			Taste aversion bitter/sour [a]		
APEQ Food Variety subscale mean				Food variety [a]	Food variety [a]
mFNS total mean				Food neophobia [a]	
Self-identified picky eating (yes/no)	Adult picky eating [b]	Overall disliking of food [c]			

Note. The table shows which elements of adult picky eating we hypothesized to be able to measure with the FPQ and AFG. In columns are the scores derived from the FPQ and AFG, in rows the scores derived from other measures of (elements of) adult picky eating. FPQ = Food Preference Questionnaire; AFG = Avoidance of Food Groups questionnaire; APEQ = Adult Picky Eating Questionnaire; NIAS = Nine-Item ARFID Screen; FNS = Food Neophobia Scale.
[a] Spearman's Rho correlation.
[b] Welch's *t*-test.
[c] Mann Whitney *U* test.

2.2. Results of prestudy

2.2.1. Main outcomes

The scores composed of the FPQ and AFG significantly correlated with the validated APE measures (see Table 3). However, only the Spearman's rho correlations between the FPQ mean liking score and two APE questionnaires (Mean of the total APEQ and Sum score of NIAS picky eating subscale) were above the pre-registered cut-off $\rho = 0.50$. In addition, a one-tailed independent sample Welch's *t*-test showed that the mean liking score of the FPQ differed significantly between self-identified picky eating groups ($t(125.56) = 10.85$, $p < .001$), with adult picky eaters ($M = 5.3$, $SD = 1.0$, $N = 83$) having a lower mean liking score than non-picky eaters ($M = 6.6$, $SD = 1.0$, $N = 331$). The effect size was $d = -1.34$, which indicates a large effect.

Lastly, a one-tailed Mann-Whitney *U* test showed that the low proportion score of the FPQ differed significantly between self-identified picky eating groups ($U = 22611$, $p < .001$), with adult picky eaters ($M = 0.2$, $SD = 0.1$, $N = 83$) more often giving low liking scores than non-picky eaters ($M = 0.1$, $SD = 0.1$, $N = 331$). The effect size Cliff's delta was 0.65, indicating a large effect. Because the Spearman rho correlations for the FPQ low proportion score with the Mean APEQ and with the sum score of the NIAS Picky Eating Subscale were below the cut-off of 0.50, the FPQ low proportion score was not used as element of adult picky eating behaviour in the twin analyses.

2.3. Conclusion of prestudy

The results of the prestudy showed that an extensive food preference questionnaire can be used to measure APE in a Dutch sample. The mean liking score on the FPQ correlated substantially with both the mean score on the APEQ and the sum score on the picky eating subscale of the NIAS. These are both previously validated (Ellis et al., 2017; Zickgraf & Ellis, 2018) and often used questionnaires to measure APE. In addition, self-identified picky eaters had a significantly lower FPQ mean liking score than non-picky eating. However, other elements of the FPQ and the AFG did not correlate strongly enough, leading to the conclusion that the mean liking score of the FPQ is a good indicator of APE. Based on these results, we decided to use the mean liking score of the FPQ in the Main Study to estimate the heritability of APE, and not the low proportion score of the FPQ, the FPQ sour/bitter liking score, the FPQ never tried score, or the AFG disgust score.

3. Main study

3.1. Methods of main study

3.1.1. Dataset

For the analysis of twin data, data from NTR survey 11 (collected in 2015) were included. In this survey, participants were asked to fill out the same FPQ and AFG as in the Pre Study, in addition to questions about demographic factors (date of birth, sex, height, weight, and educational history), and other items not included in the current study. The Omega Total for the FPQ in this sample was high (0.93, 95 % Confidence

Table 3
Spearman's Rho Correlations Between Relevant Study Variables in the prestudy and Mean scores on FPQ Mean liking and FPQ Low proportion for self-identified picky eaters (yes/no).

		FPQ Mean Liking	FPQ Low Proportion	FPQ Sour/Bitter Liking	FPQ Never Tried	AFG Disgust
Mean APEQ		**−.52***	.44*			
Sumscore NIAS		**−.53***	.49*			
Picky Eating Subscale						
Mean APEQ Taste Aversion Subscale				−.40*		
Mean APEQ Food Variety Subscale					.22*	.39*
Mean FNS					.24*	
Self-identified	Yes	***M*=5.3**[a]	***M*=0.2**[b]			
picky eating	No	***M*=6.6**[a]	***M*=0.1**[b]			

Note. All correlations are one-tailed Spearman's ρ correlations with $N = 414$. FPQ = Food Preference Questionnaire; AFG = Avoidance of Food Groups questionnaire; APEQ = Adult Picky Eating Questionnaire; NIAS = Nine-Item ARFID Screen; FNS = Food Neophobia Scale. * = $p < .001$. [a] Mean difference tested with a one-tailed independent sample Welch's *t*-test; [b] Mean difference tested with a one-tailed Mann-Whitney *U* test. **Bold** = correlation above the pre-registered cut-off of $\rho = 0.50$ or significant difference between self-identified picky eaters ($N_{yes} = 83$, $N_{no} = 331$).

Interval [0.92, 0.93]).

Data were available of 8225 twins and multiples. Only the participants of age 18 and above were included ($n = 20$ participants excluded), and all participants with more than 20 % missing items over all the relevant questions were removed from the sample ($n = 157$ participants excluded). In addition, data from participants with unknown zygosity were removed ($n = 32$ participants excluded). This resulted in a final sample of 8016 adult twins, of which 2476 (30.9 %) males and 5540 (69.1 %) females. They had a mean age of 35.3 ($SD = 15.2$, range 18–93, median 29.0). In total, 1085 monozygotic males (MZM; 346 complete pairs), 673 dizygotic males (DZM; 179 complete pairs), 2951 monozygotic females (MZF; 1110 complete pairs), 1490 dizygotic females (DZF; 453 complete pairs) and 1817 opposite sex twins (DOS; 451 complete pairs) were included in the analyses.

3.1.2. Analyses

All analyses were performed in R Statistical Software (R Core Team, 2021; version 4.1.1). Data were loaded into R from an SPSS file. Then, BMI and the sub scores of the FPQ were calculated for each person as described in the Pre Study.

To determine the heritability of APE, data were analysed based on a classical twin design (Boomsma, Busjahn, & Peltonen, 2002), by genetic structural equation modelling, with the package OpenMx (Neale et al., 2016; version 2.20.6). The rationale for the classical twin design is that monozygotic twins (MZ) share 100 %, or nearly 100 %, of their DNA whereas dizygotic twins (DZ) share 50 % on average of their segregating genes. Thus, if the within-twin pair correlations of MZ twins are higher than those of DZ twins, we may infer that genetic factors underlie individual differences in the elements of APE (Boomsma et al., 2002).

The correlations between twins in a monozygotic twin pair and the correlations between twins in a dizygotic twin pair were first calculated in a so-called saturated model, in which means, standard deviations and correlations are estimated freely. Then, we fitted more parsimonious models step-by-step, and compared these with the previously most parsimonious model. Differences between the negative log-likelihoods (-2LL) of more constrained models as compared to less constrained models were tested using χ^2 tests, with degrees of freedom (*df*) reflecting the difference in parameter estimates between the models. If the result was a *p*-value higher than 0.01, the restricted model was not significantly different from the previous model and was therefore chosen as the current most parsimonious model. This model was then used for comparison to the next, even more restricted model. If the χ^2 test resulted in a *p*-value lower than 0.01, the restricted model significantly diminished the model fit, and it was not retained.

In this way, we first tested whether males and females had different means and standard deviations by constraining those values to be equal across the sexes. Second, we imposed equality constraints on the correlations between male-male and female-female twin pairs to test for quantitative sex differences (i.e., if the magnitude of the genetic and environmental factors differs between males and females). Third, we imposed equality constraints on the correlations between same-sex and opposite-sex DZ twins to test for qualitative sex differences (i.e., if different genes play a role in males and females).

Next, genetic structural equation modelling was performed with equality constraints imposed based on the outcome of the most parsimonious model. In such a model, variance in the outcome measure can be explained by a combination of additive genetic effects (A), non-additive or dominance genetic effects (D), shared environmental effects (C), and nonshared environmental effects (E). The effects of D and C factors cannot be estimated simultaneously in data from twins only. Thus, either an ADE or ACE model must be chosen based on the MZ and DZ correlations. If the MZ correlations are more than double the DZ correlations, this suggests the presence of dominance genetic effects, and thus an ADE model will be fitted to the data. If the DZ correlations are more than half the MZ correlations, this suggests the presence of common environmental effects and thus an ACE model will be fitted. In both models, E reflects the unique environment not shared by twin pairs, as well as measurement error. Thus, based on the data of within-twin pair correlations, either narrow-sense heritability (the contribution of A on the variance of a trait) or broad-sense heritability (the contribution of both A and D on the variance of a trait) can be estimated (Knopik, Neiderhiser, DeFries, & Plomin, 2016).

The fitted model was compared on goodness of fit with other, more parsimonious models (i.e., for ACE models: an AE model, an CE model, and an E model. For ADE models: an AE model and an E model. A DE model is theoretically unlikely, because there would be no dominance genetic effects without there also being additive genetic effects). Models were again compared with each other using a χ^2 test, with the same criteria as described above.

3.2. Results of main study

3.2.1. Psychometric properties of the questionnaire

The sample mean of the FPQ mean liking score was 5.8 ($SD = 0.9$), and the score ranged from 1.6 to 9.7. Visual inspection showed that the mean liking score of the FPQ did not deviate severely from normality, which can be seen in the density plot in Supplemental Fig. S11.

3.2.2. Main outcomes

The saturated model showed that the MZ twin correlations were higher (MZM = 0.49 and MZF = 0.50) than the DZ twin correlations (DZM = 0.19, DZF = 0.16 and DOS = 0.15), and that there was little suggestion of sex differences in correlations, see Table 4. The most parsimonious model turned out to be one where the standard deviations were constrained to be equal between males and females, the correlations between monozygotic males and dizygotic males were constrained to be equal to the correlations between monozygotic females and dizygotic females, and the correlations between dizygotic same-sex twins

Table 4
Twin Correlations From the Saturated Model in the main study.

	Zygosity	Correlation [CI]
Full saturated model	MZM	.49 [.41, .56]
	DZM	.19 [.06, .31]
	MZF	.50 [.45, .54]
	DZF	.16 [.07, .25]
	DOS	.15 [.05, .24]
Most parsimonious model	MZ	.50 [.46 - .53]
	DZ	.16 [.10-.22]

Note. MZM = monozygotic male; DZM = dizygotic male; MZF = monozygotic female; DZF = dizygotic female; DOS = dizygotic opposite sex twin; MZ = Monozygotic; DZ = Dizygotic; CI = 95% confidence interval.

were constrained to be equal to the correlations between dizygotic opposite-sex twins. However, constraining the mean scores to be equal between males and females significantly lowered the fit of the model ($p < .001$), because males on average had a higher mean liking score ($M = 6.0$, $SD = 0.02$) than females ($M = 5.7$, $SD = 0.01$). The fit results of the saturated and restricted models can be found in Supplemental Table S4. The MZ correlation in the most parsimonious model was 0.50 (95 % Confidence Interval [0.46, 0.53]), whereas the DZ correlation was 0.16 (95 % Confidence Interval [0.10, 0.22]). Thus, the DZ correlation was less than half of the MZ correlation, and an ADE model was fitted to the data, with the means separately estimated for males and females.

The ADE model revealed that the broad-sense heritability (A + D estimates) for APE was 49 %; with additive genetic effects accounting for 14 % (95 % Confidence Interval [00, 38]) of the variance and dominance genetic effects accounting for 35 % (95 % Confidence Interval [11, 52]). The remaining 51 % (95 % Confidence Interval [47, 54]) came from E, nonshared environmental effects and measurement error. The ADE model was the best fitting model; both an AE ($p = .004$) and E ($p < .001$) model significantly lowered the fit (see Table 5).

4. Discussion

The main aim of the current study was to examine the heritability of adult picky eating. To this end, we investigated which elements of adult picky eating could be measured with a Food Preference Questionnaire in a prestudy, and what the heritability of adult picky eating as measured with the Food Preference Questionnaire was in the main study. The results showed that the overall mean liking score on a validated food preference questionnaire could be used as a proxy measure for Adult Picky Eating. Furthermore, we found that 49 % of the variance in this measure of APE was explained by (additive and dominance) genetic effects, while the remaining variance is explained by unique environmental factors.

In the prestudy, we found that the correlation between the FPQ mean liking score and existing APE questionnaires was high enough to conclude that specifically this score can be used as a proxy measure of APE. Although convergent validity is usually seen as adequate with a correlation of 0.50 or higher (or −0.50 and lower; Abma, Rovers, & van der Wees, 2016), one could still argue that this cut-off is rather arbitrary, since the correlation between the FPQ mean liking score and the existing APE questionnaires was just above cut-off ($\rho = 0.52$ and 0.53), while correlations with the FPQ low proportion score were just below this cut-off ($\rho = 0.44$ and 0.49). So, overall disliking of food probably does play a role in APE, but the specific APE questionnaires (APEQ and NIAS) measure more elements (such as food variety) than only overall disliking. We also expected that different scores of the FPQ could be used to measure taste aversion of bitter and sour tastes, food neophobia, and food variety. However, that was not confirmed by the results since these correlations, although significant, were clearly below the pre-registered cut-off. There are a couple of explanations for these lacking findings. First, it should be noted that the FPQ sour/bitter liking score consisted of only six items: pickles, beer, black coffee, vinegar, lemon, and grapefruit. Almost all foods have a complex flavour palette with more than one dominant flavour, which is also shown in the sensory food database by Mars et al. (2020). Thus, the categories bitter and sour might not have been captured well enough by this small list of food items. Additionally, it is likely that food neophobia as measured with the mFNS does not only consist of rejection of novel foods, but also has a fear or anxiety component that is not captured with the FPQ never tried score. Lastly, food variety as measured by the APEQ is complex, consisting of multiple elements such as variety within and between food groups, which explains a lower correlation with the FPQ and AFG.

In the main study, we found a broad sense heritability of 49 % for APE, which consisted of additive genetic effects (A, 14 %) and dominance genetic effects (D, 35 %). The remaining variance was accounted for by non-shared environmental effects (E), which is defined as those environmental factors that are not shared between twins in a twin pair. The pattern of twin correlations did not suggest an influence of shared environment (which includes the shared family environment in childhood and adolescence, including for example parenting practices) on APE behaviour, but this needs to be formally tested in an extended model (for example, a model with twins and parents allows the inclusion of both Dominant genetic factors (D) and common environmental factors (C) shared by family members). Although this is not in line with previous research stating that parental feeding practices in childhood are related with later levels of APE (Ellis, Schenk, et al., 2018; Zohar, 2022), other twin studies exploring related traits also did not find a significant role of the common environment. Previous research on food preferences in general (Vink et al., 2020), adult food neophobia (Knaapila et al., 2007) and other underlying traits of APE such as cognitive flexibility (Guimaraes et al., 2020), disgust sensitivity (Sherlock et al., 2016) and taste sensitivity (Hansen et al., 2006; Wise et al., 2007) also found either an ADE model or an AE model (with heritability estimates varying between 30 % and 70 %). Indeed, even in children, for whom one would expect parental environment to be more influential in eating behaviour, no considerable common environmental influence on food fussiness was found (C = 5 % in on average 3,5-year-old children (Fildes et al., 2016), and C = 25 % in 16-months-old children only (no C in 3-, 5-, 7-, 13-year-olds; Nas et al., 2023)), while the trait was shown to be highly heritable (A = 78 % in Fildes et al., 2016 and 60–84 % in Nas et al., 2023). The current results thus seem to be in line with other research in the fields of childhood picky eating and adult food preference.

4.1. Limitations and strengths

Since both studies relied completely on self-report measures, standard limitations of that apply. For example, it has been found that self-reported weight, used to calculate BMI, is usually underestimated (Gosse, 2014). Within our two Dutch samples we observed that the mean

Table 5
ADE Model Comparison Fitting Results of the main study.

	Test	-2LL	df	X^2	Δdf	*p*	AIC
0. Saturated ADE model		**20186.65**	**8011**				**20196.65**
1. AE model	1 vs. 0	20195.06	8012	8.41	1	.004	20203.06
2. E model	2 vs. 0	20623.86	8013	437.21	2	.000	20629.86

Note. -2LL = −2 log-likelihood; df = degrees of freedom; AIC = Akaike's Information Criterion. **Bold** = best fitting model.

liking score of the FPQ was lower in the main study ($M = 5.8$, $SD = 0.9$) than in the prestudy ($M = 6.4$, $SD = 1.1$), although not formally tested. This difference might be due to the fact that two questionnaires were distributed 8 years apart (2015 vs. 2023). Another reason could be that participants in the prestudy were recruited explicitly for a questionnaire about eating behavior. It is possible that people who responded to this message are more interested in food/eating behavior, and therefore have a more diverse diet, compared to the twin sample in the main study who were not specifically recruited based on (an interest in) eating behavior. In general, the samples were rather similar in demographic characteristics (Supplementary Table S1). As such, we are confident to state that the findings of the sample of the Pre Study regarding the overlap between the FPQ mean liking score and existing measures of APE may also generalize to the other adult Dutch sample from the Main Study.

In addition, the classical twin modelling used in the main study has some standard limitations (Verweij, Mosing, Zietsch, & Medland, 2012). The first is the equal environment assumption, which asserts that the environmental covariation within MZ twin pairs is the same as within DZ twin pairs (e.g. that a more similar treatment of MZ twins compared to DZ twins does not influence the trait). If this assumption is violated, this could inflate the A and D estimates in the model. Previous research has shown that this inflation bias in food-related traits, such as BMI (Felson, 2014) and disordered eating behaviours (Klump, Holly, Iacono, McGue, & Willson, 2000), is negligible although parents seem to be more likely to use differential restrictive feeding practices when they had differential concern for the weight status of their children (Payne, Galloway, & Webb, 2011). Thus, we expect the same is true for the current study. The second assumption of classical twin modelling is that DZ twins share approximately 50 % of their genes on average, which is only met if the population mates at random (Verweij et al., 2012). If this assumption is violated, it can inflate the C estimate. Another limitation is that with classical twin modelling, C and D estimates cannot be estimated simultaneously. We opted for estimating the influence of D in the current study based on the genetic correlations between twin pairs, but future research might benefit from extending the current design by adding parents or children of twins into the model, making it possible for C and D to be estimated at the same time (Verweij et al., 2012). The classical twin model as we applied it, did not account for possible interaction or correlation between genes and the environment. Any AE interaction could lead to inflation of the E-estimate, while inflation of the A-estimate may occur when there is AE correlation (see Purcell 2002). Research suggests that gene-environment interactions could play a role in eating-related phenomena, such as obesity/BMI (Reddon, Guéant, & Meyre, 2016; Selzam et al., 2018), eating disorders (Steiger & Thaler, 2016), and even childhood picky eating (Patel, Donovan, & Lee, 2020). Therefore, it might be that the E estimate in the current study is inflated. An interesting avenue for future research would therefore be to perform gene-environment interaction studies.

Despite these limitations, the current study is a valuable addition to the existing literature and also has several strengths. Firstly, the prestudy confirmed that a food preference questionnaire can be used to measure APE. Secondly, the APEQ, NIAS picky eating subscale and mFNS were all translated from English into Dutch through forward-backward translation procedures. The Dutch versions of these questionnaires showed very good scale reliability in our prestudy. Thus, these scales may be used in future research into APE in Dutch samples, although they will need to be further validated. The main study makes a contribution because it is, to our knowledge, the first study to investigate the heritability of picky eating in an adult sample. Another strength of this study is its sample size; compared to previous twin studies on the heritability of food-related phenomena with sample sizes between 109 and 1211 (Guimaraes et al., 2020; Hansen et al., 2006; Knaapila et al., 2007, 2011; Sherlock et al., 2016; Wise et al., 2007), the current sample size of more than 8000 twins is large.

4.2. Future research into genetic and environmental factors

The findings of this study provide some direction for future research. The fact that genetic factors play a substantial role in overall food preference as a proxy for APE is interesting, and might explain to people elements of their behaviour for which they may not have a clear understanding. One next step is to identify which genes are responsible for individual differences in APE. If APE is a complex trait, with many different genes making small contributions, a Genome-Wide Association Study (GWAS) can be performed (Duncan, Ostacher, & Ballon, 2019). However, a gene-candidate study could also be useful. Some obvious candidate genes come to mind. For example, a previous candidate gene study identified two genes (TAS2R38 and CA6) that are related to bitter taste perception, to be associated with childhood picky eating (Cole, Wang, Donovan, Lee, & Teran-Garcia, 2017). It is likely that these same genes also play a role in APE. Other possible candidates include genes related to other taste perceptions (Chamoun et al., 2018; Törnwall et al., 2014) or disgust sensitivity (Kang, Kim, Namkoong, & An, 2010). Another way to extend the current findings in future studies is by investigating genetic correlations, either in the classical twin design by multivariate models or by using GWAS summary statistics (Zhang et al., 2021). These can give more insight into the mechanisms underlying APE. For example, APE might be genetically correlated with disgust sensitivity, taste sensitivity, or cognitive rigidity. Lastly, it would be important to explore the genetic overlap between childhood picky eating or food fussiness with adult picky eating behaviour in order to explore whether the same genes influence both picky eating behaviour in childhood and in adulthood or whether there are different mechanisms at play.

Next to the genetic aspects, future research could also aim to deepen the understanding of the unique environment that contributes to the development of APE. This could eventually lead to better prevention and intervention programmes. One possible environmental factor is early negative experiences with food, such as a choking experience, medical problems related to food (intake) or weight problems during childhood (Ellis, Schenk et al., 2018). Furthermore, if a child has weight problems during childhood, this might result in differential parental treatment regarding restrictive feeding practices compared to a child without weight problems (Payne et al., 2011). For future research it is important to explore how strongly child picky eating behavior is related to APE, and which childhood symptoms or factors predict APE. It would also be worthwhile to investigate whether the eating behaviour of spouses can affect picky eating behaviour in adults, since it has been suggested that food preference in adulthood is associated with the food preference of spouses (spouse correlations up to $r = .31$ for vegetables and savory snacks, Vink et al., 2020). Other possibilities should also be explored; potentially first with qualitative studies to discover the factors that have contributed to their APE development, according to picky eaters themselves.

Future studies should explore whether the prevalence of APE varies in different countries and different cultures. In our sample (prestudy), 20 % of participants identified themselves as picky eater, which is on the lower end of the range of previous estimations from the US (18 %–46 %, Dial et al., 2021; Ellis, Zickgraf et al., 2018; Kauer et al., 2015; Van Tine et al., 2017; Zickgraf & Schepps, 2016).

4.3. Conclusions

In conclusion, the current study has shown that APE is partly heritable in a Dutch sample. By gaining more insights into the origins of APE, we may be able to intervene in the future with the result to mitigate some negative consequences (e.g., mortality, cardiovascular disease) associated with it, caused by lower dietary variety (Zickgraf & Schepps, 2016), especially with regards to fruits and vegetables (Dial et al., 2021; Ellis, Galloway, et al., 2018; Kauer et al., 2015; Zickgraf & Schepps, 2016). To get to the bottom of factors that mitigate negative

consequences, genetically informative designs could be used to estimate environmental effects more 'purely'. All in all, this study is the first study investigating the heritability of picky eating in adults, which can help to shed light on this understudied phenomenon.

Funding

Data collection for the prestudy was supported by the Behavioural Science Institute of the Radboud University, the Netherlands. Data collection for the main study was funded by a grant from BBMRI–NL: Enrichment of NTR with information on dietary intake and aspects of eating behavior; BBRMI-CP2011-38; PI Boomsma and Feskens.

Ethical declaration

Research has been performed in accordance with the Declaration of Helsinki. The prestudy was carried out in accordance with the guidelines of the Ethics Committee of the Faculty of Social Science of the Radboud University, outlined in protocol [ECSW-LT-2022-11-15-37361]. For the main study, the Medical Research Ethics Committee of the VU University Medical Centre declared the research was not subject to the WMO (Medical Research involving human subjects act) [METC 2014.487].

CRediT authorship contribution statement

Emma A. Koenders: Conceptualization, Data curation, Formal analysis, Methodology, Project administration, Writing – original draft, Writing – review & editing, Investigation. **Laura W. Wesseldijk:** Formal analysis, Methodology, Supervision, Writing – original draft, Writing – review & editing. **Dorret I. Boomsma:** Data curation, Investigation, Project administration, Writing – original draft, Writing – review & editing. **Junilla K. Larsen:** Conceptualization, Methodology, Supervision, Writing – original draft, Writing – review & editing. **Jacqueline M. Vink:** Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Supervision, Writing – original draft, Writing – review & editing.

Declaration of Competing interest

The authors have no conflicts of interest to declare.

Data availability

Data will be made available on request.

Acknowledgements

For the prestudy, we thank Fenna J.M. Andriessen for her help with the translation of the surveys and we thank Carina de Kort for her advice on the categorization of bitter/sour foods. We thank all participants for their contribution to the study.

For the main study, we thank all members of the Netherlands Twin Register (NTR) who participated in the food preference survey for their contribution.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.appet.2024.107230.

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