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THE HOME ENVIRONMENT SHAPES EMOTIONAL EATING

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Conflict of interest

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Abstract

Emotional overeating (EOE) is the tendency to eat more in response to negative emotions; its etiology in early life is unknown**.** We established the relative genetic and environmental influences on EOE in toddlerhood and early childhood. Data were from Gemini, a population-based cohort of 2402 British twins born in 2007. EOE was measured using the 'emotional overeating' scale of the Child Eating Behavior Questionnaire at 16 months and 5 years. A longitudinal quantitative genetic model established that genetic influences on EOE were minimal; on the other hand, shared environmental influences explained most of the variance. EOE was moderately stable from 16 months to 5 years and continuing environmental factors shared by twin pairs at both ages explained the longitudinal association.

Keywords: Emotional eating, eating behavior, developmental psychology, twins, heritability, weight, obesity

Abbreviations: EOE, emotional overeating; CEBQ, Child Eating Behavior Questionnaire

Emotional overeating (EOE) is the tendency to overeat in response to stress and negative emotions (Macht, 2008). It emerges early (Wardle, Guthrie, Sanderson, & Rapoport, 2001) and tracks moderately from early to late childhood [r=0.29]) (Ashcroft, Semmler, Carnell, van Jaarsveld, & Wardle, 2008). Understanding its etiology is important because it has been associated with excessive weight and weight gain in childhood (Braet & Van Strien, 1997; Parkinson, Drewett, Le Couteur, Adamson, & T, 2010; Steinsbekk & Wichstrom, 2015; Viana, Sinde, & Saxton, 2008), as well as bulimia nervosa and binge eating disorder (Pearson, Riley, Davis, & Smith, 2014).

Two main theories have been formulated to explain the development of EOE. The Psychosomatic Theory (Kaplan & Kaplan, 1957) proposes that obese individuals have not learned to distinguish successfully between the arousal caused by hunger and negative emotion; possibly because of classical conditioning in early life. This leads to increased food intake in response to negative feelings, and predisposes those individuals to weight gain (Bruch, 1964). This theory proposes that EOE is learned, rather than innate.

The Internal/External theory (Schachter, Goldman, & Gordon, 1968) suggests a different basis for EOE. It proposes that healthy weight individuals tend to decrease their food intake in stressful situations, in response to internal physiological stress cues. On the other hand, obese individuals' appetites are abnormal in not being affected by stress. The theory still predicts that obese individuals eat more than normal weight individuals during times of stress, but due to the inability to respond 'normally' to stress cues (van Strien & Ouwens, 2003). Such aberrations in biology could be innate or learned. There has been some support for both theories (Psychosomatic Theory: (Bongers, van den Akker, Havermans, & Jansen, 2015; Bruch, 1975; Heatherton, Striepe, & Wittenberg, 1998; Jansen, Havermans, & Nederkoorn, 2011) ; Internal/External theory: (Herman & Polivy, 1984; Schachter et al., 1968; Willner, Muscat, & Papp, 1992). A potential mechanism through which children might

learn to overeat in response to negative emotions is classical conditioning. Parents who sooth their children's negative emotions with food might inadvertently condition them to associate stress with food consumption or hunger, and in doing so establish maladaptive eating patterns at an early age. A recent experimental study of adults (n=127) supported this notion; pairing negative emotion with highly palatable foods resulted in a greater desire to consume, which in turn might result in overeating (Bongers et al., 2015).

Twin studies provide a powerful method for understanding the extent to which individual differences in a characteristic are determined by genetic and environmental variation. Importantly, twin analyses can also provide insight into the relative importance of two different types of environmental influence – aspects that are shared by two twins in a pair (shared environmental effects), and influences that are unique to each individual twin (unique environmental effects).

Self-report psychometric questionnaires, such as the Three Eating Factor Questionnaire (TFEQ) (Stunkard & Messick, 1985) and the Dutch Eating Behavior Questionnaire (DEBQ) (Vanstrien, Frijters, Bergers, & Defares, 1986), are the most commonly used measures of EOE for adults. Both questionnaires measure a variety of eating behaviors and employ similar items to assess EOE (example items; TFEQ: 'When I feel anxious, I find myself eating"; DEBQ: "Do you get the desire to eat when you are anxious, worried or tense?").

So far three adult studies (two using the Three Factor Eating Questionnaire and one using the Dutch Eating Behavior Questionnaire) have reported moderate additive genetic influences on EOE; explaining 45% of the variance in a UK sample (Sung, Lee, Song, Lee, & Lee, 2010), 31% in a Finnish sample (Keskitalo et al., 2008), and 45% in a Swedish sample (Tholin, Rasmussen, Tynelius, & Karlsson, 2005). In these adult samples the shared

environment did not contribute to variation in EOE; instead, unique aspects of the environment (unshared between twin pairs) explained remaining variance.

A recent study compared adult MZ twins raised together with MZ twins raised apart. Studies of twins reared apart rely on the assumption that MZ twins reared apart share only their genes; while MZ twins reared together share both their genes and many aspects of their environment. This assumption therefore makes it possible to directly estimate the contributions of genes and the shared environment by comparing the similarity of MZ twins reared together versus apart. This study confirmed the moderate influence of additive genetics on individual differences in EOE (55%); and, in keeping with the other twin studies, reported no influence of the shared environment (Elder et al., 2012). A caveat to this design is the potential influence of shared exposures in utero that contribute to twin similarity, prior to birth.

Genetic and environmental sources of influence are known to vary considerably with age (Bergen, Gardner, & Kendler, 2007). EOE emerges early, and its etiology may differ considerably in childhood. The home family environment plays an important role for young children, raising the possibility that shared environmental effects on EOE might be observed in early childhood.

Parents are presumed to be the most powerful socialization agents of young children's eating behavior (Swinburn et al., 2011); but as children grow and mature, their eating is likely to be increasingly shaped by external factors. Longitudinal research suggesting that childhood bullying and peer influence increase the risk of obesity in adulthood, supports the increasing importance of peers in later childhood (Takizawa, Danese, Maughan, & Arseneault, 2015). Earlier in development, previous research has suggested that parents who use food to soothe elicit emotional eating behavior in their children (Braden et al., 2014). Further evidence for the importance of parental feeding comes from an experimental study (n=25 mother-child

dyads) showing that children whose mothers use food to regulate emotions overconsume in the absence of hunger when induced with a negative mood (Blissett, Haycraft, & Farrow, 2010). In addition, these maladaptive feeding practices have also been linked to maternal negative affect, posing a potential direct link between maternal affect, parental feeding behavior and the development of child EOE (Rodgers et al., 2014).

Hence establishing the relative influence of genes and environment in the development of EOE in early childhood and across different developmental stages is important because it can inform interventions aimed to reduce EOE later. If the home environment exerts an important influence on the development of children's emotional eating (as opposed to genetic influence), targeting aspects of the home environment associated with EOE (e.g. emotional feeding) is warranted. So far no studies have established the relative influence of genes and environment in shaping EOE in children.

This study uses prospective data from a large pediatric twin study, Gemini, to quantify the relative importance of genetic and environmental influences on EOE from toddlerhood to early childhood. The longitudinal design also allows us to establish if continuing genetic or environmental influences contribute to stability of EOE from toddlerhood to early childhood. We hypothesize that the shared environment plays a significant role in shaping EOE in childhood, in contrast to adult studies.

Methods

Participants

All participants were drawn from Gemini (http://www.geministudy.co.uk/); a population-based twin birth cohort set up in 2007 to investigate genetic and environmental influences on early growth (van Jaarsveld, Johnson, Llewellyn, & Wardle, 2010). Between March and December 2007 the Office for National Statistics wrote to all eligible families with twins

born that year (N=6754) to ask for their consent to pass their contact details on to the Gemini researchers. 3435 families agreed to be contacted. 2402 families completed the baseline questionnaire; the baseline sample included 749 monozygotic pairs [MZs] and 1616 dizygotic pairs [DZs]; 37 pairs were of unknown zygosity.

Follow-up questionnaires were sent to families when children were 16 months and 5 years old. Gemini is generally representative of UK twins when compared with national twin statistics on zygosity and sex, gestational age at birth and birth weight. Ethical approval was granted by the University College London Committee for the Ethics of non–National Health Service Human Research, and all aspects of data collection and storage were in accordance with the standards stipulated by this body.

Measurement of emotional overeating

EOE was measured using the 'emotional overeating' scale of the parent-reported Child Eating Behavior Questionnaire (CEBQ); (Wardle et al., 2001). The CEBQ is a parent reported questionnaire which consists of 35 items that describe a range of children's eating behaviors. The questionnaire was developed to quantify child eating behaviors hypothesized to relate to weight and weight gain in childhood. Parents rate how much the statements apply to their children on a 5-point Likert scale (ranging from "Never" to "Always").

The EOE (4 items) scale has high internal reliability ($\alpha = 0.72 - 0.79$) and scores correlate moderately over a two-week period ($r = 0.52$) (Wardle et al., 2001).

The CEBQ has been validated using behavioral measures of eating behavior in children aged 4-5 years (Carnell & Wardle, 2007). Parents completed the EOE scale when twins were on average 16 months old and 5 years old. At 5 years, parents responded to four statements from the standard EOE scale about their child's tendency to eat in response to negative emotions

("My child eats more when worried", "My child eats more when annoyed", "My child eats more when anxious", and "My child eats more when s/he has nothing else to do").

Following in-depth qualitative pilot work with a sample of mothers with 18-month old toddlers, the emotion adjectives from the standard EOE scale were modified for the 16-month questionnaire to ensure that they were age-appropriate ('irritable' instead of 'worried'; 'grumpy' instead of 'annoyed'; 'upset' instead of 'anxious'). The fourth item from the standard EOE scale ("My child eats more when s/he has nothing else to do") was not included in the 16-month questionnaire because mothers had not observed this behavior in their children at this young age. This item was therefore omitted from the 5-year scale as well to ensure consistency of items across the two ages. Parents responded to the items using a 5-point Likert scale ('never'; 'rarely'; 'sometimes'; 'often'; 'always'). Means were calculated for each child for EOE at both ages; scores were only included if a minimum of 2 out of 3 items were completed. The internal consistency for the EOE scale in this sample at 16 months was $\alpha = 0.82$; and at 5 years was $\alpha = 0.81$.

Zygosity, age and gestational age

Gestational age was reported by parents. Information on weight at birth was taken from the child's personal health record and reported by parents. Age of the child at EOE measurement was calculated from their date of birth and the date of questionnaire completion. Opposite sex twin pairs were classified as DZ. Parents of same sex twins completed a 20-item questionnaire to establish their zygosity. The questionnaire has been validated against DNA markers, showing agreement for 95% of cases; and the questionnaire is consistent over time, with 96% rating the same zygosity status at 3 years (Price et al., 2000).

Gemini is an inclusive population-based cohort and all families with twins were invited to participate regardless of health problems, disorders or birth complications. Additional sensitivity analyses were conducted excluding twins with a reported chromosomal disorder

(n=20), as well as twins born earlier than 34 weeks gestation (n=262). Children born before 34 weeks are likely to be tube-fed which can affect their feeding development, and has the potential to impact their eating behavior in toddlerhood and early childhood. The results from the full sample did not differ from the reduced sample, so the full sample was therefore used.

Statistical analyses

Because twins share their age and gestational age exactly (and sex for same-sex pairs), regress scores on gestational age, age at the time of measurement, and sex prior to heritability analyses to ensure these factors do not inflate the shared environmental effect. All twin analyses were performed on the regressed EOE scores. Pearson's correlation coefficient was used to establish the association between EOE at 16 months and 5 years.

Twin analyses

The basis of the twin method is to compare resemblance between identical twins (monozygotics, MZ) who share 100% of their genes, with that between non-identical twins (dizygotics, DZs) who share approximately 50% of their segregating genes. Because both types of twins are assumed to share their environments to a similar extent, difference in resemblance between the two types of twins reflects genetic contributions to the trait. If there is little difference in resemblance, common environments shared by twin pairs can be assumed to be largely responsible for individual differences in that trait. The extent to which identical twins differ is due to unique environmental influences not shared by twin pairs (and measurement error) (Rijsdijk & Sham, 2002). Genetic and environmental contributions to variation in EOE are estimated using two methods: comparisons of twin correlations and maximum likelihood structural equation modelling (MLSEM).

Twin correlations

Correlations of twins for EOE were calculated and compared for MZs and DZs at 16 months and at 5 years. Twin correlations were calculated using ratings of EOE for each twin to assess the similarity within twin pairs. The pattern of resemblance provides an indication of the relative importance of genetic and environmental influences on EOE at each age. Cross-twin cross-time (CT-CT) correlations provide an indication of the contribution of continuing genetic and environmental influences to the longitudinal phenotypic association (the stability of EOE from 16 months to 5 years). CT-CT correlations use the same principles as the twin correlations explained above; but instead of correlating the twin pair for EOE at the same age, CT-CT correlations relate twin 1's EOE score at 16 months to twin 2's EOE score at 5 years, and vice versa.

Higher average CT-CT correlations for MZ pairs relative to DZ pairs indicates that common genetic factors at both ages contribute to the phenotypic association; similar CT-CT correlations for both types of twins indicates that common shared environmental effects at both ages are important in driving the phenotypic association. Twin correlations and CT-CT correlations were calculated using OpenMx Software (Boker et al., 2011); a package designed to use in R (R Core Team, 2015). OpenMx provides one average CT-CT correlation for MZ and DZ twin pairs separately, as it deems twin order to be irrelevant.

Maximum likelihood structural equation modelling

Maximum Likelihood structural equation modelling (MLSEM) was used to provide reliable parameter estimates of additive genetic effects (A), shared environmental effects (C) and unique environmental effects (E) with 95% confidence intervals and goodness-of-fit statistics. A bivariate longitudinal model was run providing estimates of A, C and E at 16

months and 5 years as well as information about the extent to which the genetic, shared environmental and unique environmental influences underlying EOE at 16 months were the same as those at 5 years, denoted by the additive genetic [rA], shared environmental [rC], and unique environmental [rE] correlations. A high rA would indicate that the majority of the additive genetic effects at 16 months persist at 5 years, whereas a low rA would indicate that additive genetic factors are largely unique to each age. The longitudinal model also quantifies the extent to which continuing genetic and environmental influences explain the longitudinal phenotypic correlation from 16 months to 5 years (denoted as bivariate A, C and E). That is, the bivariate estimates explain whether stability in EOE from 16 months to 5 years is largely due to the same genes or the same environmental factors influencing the trait at both ages. The bivariate estimates are calculated by dividing the covariance of the latent factors (A, C and E) by the phenotypic correlation of between the two variables. Bivariate estimates and etiological correlations are independent of the univariate A, C, and E contributions at 16 months and 5 years. For example, EOE could be highly heritable at both ages and correlated over time, but with few genetic effects in common at either age (low rA), and the longitudinal association being driven entirely by shared environmental effects (low bivariate A; high bivariate C) (Posthuma et al., 2003). MLSEM was carried out using OpenMx software (Boker et al., 2011). A number of fit statistics were available, including the Likelihood Ratio test (LRT), Aikaike's Information Criterion (AIC) and the Bayesian Information Criterion (BIC). As all models are nested, the LRT can be used for model identification. The BIC statistic was also taken into consideration because it takes account of sample size and number of parameters, and is therefore more appropriate than the overly conservative LRT and AIC in the context of large datasets like Gemini (Posada & Buckley, 2004; Trudeau, Shephard, Bouchard, & Laurencelle, 2003).

First a saturated model was fitted to the data, with no parameter constraints (i.e. estimating only means, variances and covariances for MZs and DZs), to provide fit statistics against which to test the goodness of fit of the ACE model. Then a full ACE model was fitted. For the univariate analyses more parsimonious sub-models were then tested for goodness-of-fit against the full ACE model; sub-models dropped A, C, and A and C together (E is never dropped because it includes measurement error). For longitudinal analyses, a full ACE model was fitted first and compared to a saturated model for goodness-of-fit. Non-significant parameters were then dropped to identify the most parsimonious model. LRT and the lowest BIC value indicate the best fitting model.

Results

EOE scores were available for 3774 children at 16 months and 1986 children at 5 years, with a combined sample for the analysis of 3784 children who had data at either 16 months, 5 years or both ages (MLSEM is able to include participants who have missing data at one time point). The descriptive statistics for the analysis sample are shown in Table 1. The children included in these analyses did not differ from the full sample in terms of zygosity, sex, gestational age or birth weight. Twin pairs with unknown zygosity were excluded from the analyses (n= 37). EOE at 16 months was significantly associated with EOE at 5 years of age ($r=0.25$, 95% CI [0.19, 0.30]; $p<0.001$), such that toddlers who were prone to eating more in response to negative emotions tended to do this as children as well.

There were some differences between families who remained in the study at 5 years and those who did not. Mothers of children in the 5 year sample were more educated, older and had a lower BMI at baseline, than at 16 months. However there were no differences between the samples in relation to the sex and gestational age of the twins.

Twin Analyses

The intraclass correlations for the twin pairs at 16 months and 5 years are shown in Table 2. At both ages the MZ and DZ correlations were high and similar for both types of twins. This suggested a low contribution from genes and a strong contribution from the shared environment to variation in EOE.

Before running the longitudinal twin model, sex limitation models were conducted to test for differences between boys and girls. Sex limitation models allow for separate estimates of A, C and E for males and females. These can then be compared to models equating the estimates across the sexes. The LRT suggested that models assuming separate paths for males and females provided a better fit at 16 months and five years. However, the A, C and E estimates for both and girls were not significantly different. Therefore, for the purposes of simplicity and accessibility, a model that included both boys and girls together is presented in this paper. However, fit statistics and parameter estimates for the sex limitation models can be found in Appendix 1.

Longitudinal analyses

The averaged CT-CT correlations for the MZs and DZs demonstrated similar patterns to the simple ICCs (Table 2). This suggested that continuing shared environmental influences largely explained the correlation from 16 months to 5 years, and that there were few continuing genetic influences that contributed to stability in EOE from toddlerhood to early childhood.

MLSEM was used to calculate the univariate estimates for 16 months and 5 years (Figure 1). Additive genetic effects were significant at 16 months and 5 years (10% and 4% respectively). The majority of variance in EOE was explained by shared environmental effects (87% and 93% at 16 months and 5 years respectively). The variance explained by the

unique environment at each age was small (3% and 3% at 16 months and 5 years respectively).

A path diagram of the full longitudinal ACE model is presented in Figure 1. LRT suggested no deterioration of fit between the saturated model and the full ACE model ($\Delta \chi^2 = 13.218$, p=0.72). A moderate shared environmental correlation (r_C=0.27) between 16 months and 5 years indicated that even though continuing aspects of the shared environment account for the stability of EOE, many new shared environmental influences come into play at five years. There was also a significant negative genetic correlation between the two time points (r_A= -0.26; 95% CI [-0.45, -0.08]). However, because the genetic components of variance at both ages were very small (especially at age 5 years, 4%), the genetic correlation is unreliable and difficult to interpret. The unique environmental correlation was non-significant (r_E = 0.03; 95% CI [-0.11, - 0.17]), indicating that none of the unique environmental effects that influenced EOE continued to influence EOE at 5 years of age.

The bivariate estimates quantified the contribution of common genetic and environmental factors to the longitudinal association between EOE at 16 months and 5 years of age. These suggested that the longitudinal association was completely driven by shared environmental effects (Bivariate C: 1.07; 95% CI [1.03, 1.11]). The bivariate A was very small (Bivariate A: -0.07; 95% CI [-0.12, -0.02]) and bivariate E was non-significant (Bivariate E: 0.00; 95% CI [-0.01, 0.02]). These results made sense in the light of the fact that shared environmental factors were largely driving variation in EOE at both ages.

To find the most parsimonious solution, parameters were constrained to be zero. In submodel 1 bivariate estimates for E were dropped, as they were found to be non-significant (BivE: 0.00; 95% CI [-0.01, 0.02]). Additionally in submodel 2, bivariate estimates for A were also dropped (Bivariate A: -0.07; 95% CI [-0.12, -0.02]). However the paths indicating

variance explained by the latent factor E at 16 month and 5 years individually remained as they cannot be removed because they include random measurement error. When comparing the fit of the submodels with the full ACE model, the LRT suggested a significant deterioration of fit. However, due to the large sample size, the LRT can be oversensitive, detecting small changes in fit as significantly different. The BIC, that takes account of the sample size (as well as the number of parameters), suggested that submodel 2 was the best fitting model, because it produced the lowest BIC score (BIC=-16939.898). However the change in BIC (Δ BIC=1.503) between the models was less than 2, indicating that the difference was too small to discriminate meaningfully between the models. BIC difference guidelines indicate that a difference of > 2 is considered suggestive of a better model fit (Trudeau et al., 2003). The full ACE longitudinal model is therefore presented as the best-fitting model. A full list of all estimates and fit statistics is presented in Table 4.

Discussion

This is the first childhood study to investigate genetic and environmental contributions to the development of EOE, tracking children from toddlerhood (16 months) to early childhood (5 years). The results were in line with the hypothesis of a substantial effect of the shared environment on EOE in early life. However, it was somewhat surprising to observe that additive genetic effects contributed so little to this trait at either age (10 % and 4% respectively at 16 months and 5 years). These findings contrast with the high heritability estimates observed for other eating behaviours – Satiety Responsiveness (SR) (63%) and Enjoyment of Food (EF) (75%) – measured in 10 year-old children (Carnell, Haworth, Plomin, & Wardle, 2008). They also contrast with the high heritability estimates for four eating behaviours measured in Gemini at 3 months of age: Satiety Responsiveness (72%);

Slowness of Eating (84%); Food Responsiveness (59%); Enjoyment of food (54%) (Llewellyn, van Jaarsveld, Johnson, Carnell, & Wardle, 2010).

Evidence for the importance of the shared environment in shaping individual differences in this trait during both toddlerhood (88%) and early childhood (93%) also contrasts with previous studies of EOE in adults. These studies found no role of the shared environment, and a moderate contribution from genetic influences (Keskitalo et al., 2008; Sung et al., 2010; Tholin et al., 2005). However, heritability estimates are known to vary, particularly by age, and previous studies of EOE have only used adult samples. In order for genetic influences to play out, individuals need the agency to make independent choices in order to 'act out' their genetic predispositions. The young age of the sample could therefore explain the high impact of shared environments, as toddlers and children have limited access to food to regulate their emotions as they choose. Future studies could follow children into adolescence to investigate if genetic influences start to emerge as children gain the independence to act in line with their genetically predisposed traits (a phenomenon termed 'active gene-environment correlation') (Bergen et al., 2007).

A 'passive gene-environment correlation' might also explain the high shared environmental effects on variation in EOE; this refers to the 'double whammy' of a child inheriting both genes and environment related to their parents' and their own genetically-determined trait. For example, it seems likely that parents, who emotionally overeat, partly by virtue of their genetic predisposition, create an environment that nurtures this behavior in their children; children therefore inherit from their parents both the genes and the environment that encourage EOE. Passive gene-environment correlations serve to inflate shared environmental effects (Rijsdijk & Sham, 2002). One way to test for passive gene-environment correlation is to use an adoption study design, comparing the correlations between a measure of the family environment (e.g. emotional feeding) and child measures of

EOE in adoptive and non-adoptive families. Higher correlations in non-adoptive families would indicate a passive gene-environment correlation, as biological parents pass on their genetic material as well as create the family environment (Rijsdijk & Sham, 2002).

On the whole, it is perhaps unsurprising that for young children the shared environment plays an important role in shaping the development of this behavior as parents have been shown to be the most important socialization agents of young children's eating behavior (Swinburn et al., 2011), affecting their eating through parenting styles and feeding practices (Zlatevska, Dubelaar, & Holden, 2014), modeling eating behavior (Brownson, Boehmer, & Luke, 2005) and being the main gatekeepers of food (Piernas & Popkin, 2011).

We observed that EOE in toddlerhood correlated positively and moderately with EOE in childhood (r=0.25); and the longitudinal association could be explained largely by continuing shared environmental influences from toddlerhood to early childhood. However the moderate shared environmental correlation (r_C=0.27) indicated that many novel shared environmental factors influence EOE at five years. There were no unique environmental effects that continued from toddlerhood to early childhood. There was a significant genetic correlation (r_A), but due to the very small contribution of additive genetic effects on EOE at either age, this correlation is difficult to interpret. In addition, model fit statistics tentatively suggested a slightly better solution for a model excluding bivariate estimates of additive genetic effects (A) and unique environmental effects (E), highlighting the importance of shared environmental contributions to the longitudinal stability of EOE.

Implications

In comparison to previous cross sectional studies of adults, we report estimates for genetic and environmental influences on EOE at two different assessment points during childhood. The observation that the shared environment played the most important role in shaping EOE

in early childhood suggests that many of the early influences on EOE will be modifiable, in contrast to genetic influence that can be harder to change. This finding provides hope that it will be possible to develop interventions to prevent or reduce the development of EOE in childhood. Because EOE is associated with both obesity and eating disorders such interventions are needed.

While twin studies provide important insights into the relative importance of genetic and environmental influences on given characteristics, no information about the specific factors involved is given. Future research is needed to establish the modifiable shared environmental factors that play a causal role in shaping EOE in early childhood. Some evidence has suggested that parental feeding practices influence children's EOE. A recent study measuring parental feeding practices and children's eating behaviors in a longitudinal cohort of Norwegian families (N=797, age 6 and 8 years) found instrumental feeding practices (i.e. using food as a reward) at age 6 predicted increased emotional overeating at age 8 (Steinsbekk, Belsky, & Wichstrom, 2016). Similarly, further research has established that children whose parents actively control their emotions through feeding engage more in EOE (Braden et al., 2014; Tan & Holub, 2015). In addition, children whose parents highly control their food intake express more EOE behaviors (Farrow, Haycraft, & Blissett, 2015).

However, there is evidence indicating that child emotional eating elicits parental controlling feeding behavior (such as monitoring, restriction and pressure to eat), suggesting a potential bidirectional association between child eating and parental feeding (Haycraft & Blissett, 2012). Although, results from a larger observational study did not confirm that child eating at 6 years predicted parental feeding at 8 years (Steinsbekk et al., 2016).

Because EOE is linked to emotional dysregulation (Elks et al., 2012), focusing on parental efforts to promote emotion regulation (i.e. not using food to sooth negative emotions) in their offspring might be valuable. Lastly, a stressful and chaotic home

environment has been associated with childhood obesity, potentially because it provides the environment in which a child would be more likely to learn to emotionally overeat (Gundersen, Mahatmya, Garasky, & Lohman, 2011; Wardle & Boniface, 2008). Notably though, studies are needed to test the assumption that stressful environments directly increase the risk of developing EOE. In addition to purely environmental studies, future research is needed to understand how environmental exposures potentially modify the expression of genes involved in EOE, through epigenetic processes. Twin studies and epigenetic research have the potential to complement one another and elucidate how genes and environments interact in the development of complex human traits in early life. A particularly powerful design is to compare the epigenetic profiles of identical twins discordant for an environmental exposure (Bell & Spector, 2011); this is because identical twins are genetically the same.

Overall, the high influence of the shared environment supports early theory (Psychosomatic Theory and Internal/External Theory) suggesting that EOE is largely learned in early life.

Limitations

Twin studies assume that MZ and DZ twins share their environments to the same extent (so-called the 'equal environments assumption') (Rijsdijk & Sham, 2002). A recent analysis of past twin research testing for violation of the 'equal environments assumption' concluded that in the majority of studies reviewed no violations of the assumption occurred (Felson, 2014). Another limitation of the study is that due to the longitudinal nature of the study some families drop out over time. There were some differences between the families who did not provide follow up data at five years, and those who did. Mothers of families who remained in the study were more educated, older and had a lower BMI at baseline. However there were no differences regarding sex and gestational age of the twins. Children of mothers who are more

educated and healthier might be less likely to emotionally overeat themselves, or less likely to emotionally feed their children. This could explain the slightly reduced mean and variance of EOE when the children were five years old.

The CEBQ is parent-reported and biases are therefore possible. For example, some of the shared environmental effect may reflect a parent's own tendency to emotionally overeat insofar as parents who tend to do this may assume that both of their children do this as well. On the other hand, parents may find it difficult to observe this behavior with accuracy in young children, and therefore rate two twins the same. However, parents are well placed to report on their children's eating behavior, arguably knowing their children better than other potential respondents. In addition, a range of other parent-reported eating behaviors showed high heritability in this sample during infancy; suggesting that parents are indeed able to observe differences between their twins for a range other eating behaviors, adding confidence to these findings (Llewellyn et al., 2010). Nevertheless, it would be useful to collect information from other raters in future studies (e.g. childcare providers), to compare with parental reports. Additional laboratory-based studies to validate the EOE subscale would be useful. However, exposing children to stress to observe their subsequent consumption behavior poses practical and ethical difficulties. Furthermore, twin analyses require large numbers of participants, and a psychometric questionnaire, like the CEBQ, therefore remains the only option to measure behavior in large cohorts like Gemini.

Conclusion

Variation in EOE during toddlerhood and early childhood is largely influenced by environmental factors shared by both twins in a family. In contrast to most other eating

behaviors that have been explored in infancy and childhood, additive genetic effects play a minor role. Future studies are needed to identify the actual environmental factors influencing the development of EOE during the early years, and to elucidate when genetic influences emerge. Modifiable shared environmental factors that promote EOE need to be identified so that they can be targeted in interventions aimed to reduce the development EOE in childhood.

References

Ashcroft, J., Semmler, C., Carnell, S., van Jaarsveld, C. H., & Wardle, J. (2008). Continuity and stability of eating behaviour traits in children. *Eur J Clin Nutr, 62*(8), 985-990. doi:10.1038/sj.ejcn.1602855

Bell, J. T., & Spector, T. D. (2011). A twin approach to unraveling epigenetics. *Trends in Genetics, 27*(3), 116-125. doi:10.1016/j.tig.2010.12.005

Bergen, S. E., Gardner, C. O., & Kendler, K. S. (2007). Age-related changes in heritability of behavioral phenotypes over adolescence and young adulthood: a meta-analysis. *Twin Res Hum Genet, 10*(3), 423-433. doi:10.1375/twin.10.3.423

Blissett, J., Haycraft, E., & Farrow, C. (2010). Inducing preschool children's emotional eating: relations with parental feeding practices. *Am J Clin Nutr, 92*(2), 359-365. doi:10.3945/ajcn.2010.29375

Boker, S., Neale, M., Maes, H., Wilde, M., Spiegel, M., Brick, T., . . . Fox, J. (2011). OpenMx: An Open Source Extended Structural Equation Modeling Framework. *Psychometrika, 76*(2), 306-317. doi:10.1007/s11336-010-9200-6

Bongers, P., van den Akker, K., Havermans, R., & Jansen, A. (2015). Emotional eating and Pavlovian learning: Does negative mood facilitate appetitive conditioning? *Appetite, 89*, 226-236. doi:10.1016/j.appet.2015.02.018

Braden, A., Rhee, K., Peterson, C. B., Rydell, S. A., Zucker, N., & Boutelle, K. (2014). Associations between child emotional eating and general parenting style, feeding practices, and parent psychopathology. *Appetite, 80*, 35-40. doi:DOI 10.1016/j.appet.2014.04.017

Braet, C., & Van Strien, T. (1997). Assessment of emotional, externally induced and restrained eating behaviour in nine to twelve-year-old obese and non-obese children. *Behav Res Ther, 35*(9), 863-873.

Brownson, R. C., Boehmer, T. K., & Luke, D. A. (2005). Declining rates of physical activity in the United States: What are the contributors? *Annual Review of Public Health, 26*, 421-443. doi:10.1146/annurev.publhealth.26.021304.14437

Bruch, H. (1964). Psychological Aspects of Overeating and Obesity. *Psychosomatics, 5*, 269-274.

Bruch, H. (1975). Obesity and anorexia nervosa: psychosocial aspects. *Aust N Z J Psychiatry, 9*(3), 159-161.

Carnell, S., Haworth, C. M., Plomin, R., & Wardle, J. (2008). Genetic influence on appetite in children. *Int J Obes (Lond), 32*(10), 1468-1473. doi:10.1038/ijo.2008.127

Carnell, S., & Wardle, J. (2007). Measuring behavioural susceptibility to obesity: Validation of the child eating behaviour questionnaire. *Appetite, 48*(1), 104-113. doi:DOI 10.1016/j.appet.2006.07.075

Elks, C. E., den Hoed, M., Zhao, J. H., Sharp, S. J., Wareham, N. J., Loos, R. J., & Ong, K. K. (2012). Variability in the heritability of body mass index: a systematic review and meta-regression. *Front Endocrinol (Lausanne), 3*, 29. doi:10.3389/fendo.2012.00029

Farrow, C. V., Haycraft, E., & Blissett, J. M. (2015). Teaching our children when to eat: how parental feeding practices inform the development of emotional eating-a longitudinal experimental design. *Am J Clin Nutr*. doi:10.3945/ajcn.114.103713

Felson, J. (2014). What can we learn from twin studies? A comprehensive evaluation of the equal environments assumption. *Soc Sci Res, 43*, 184-199. doi:10.1016/j.ssresearch.2013.10.004

Gundersen, C., Mahatmya, D., Garasky, S., & Lohman, B. (2011). Linking psychosocial stressors and childhood obesity. *Obes Rev, 12*(5), e54-63. doi:10.1111/j.1467-789X.2010.00813.x

Haycraft, E., & Blissett, J. (2012). Predictors of Paternal and Maternal Controlling Feeding Practices with 2- to 5-year-old Children. *Journal of Nutrition Education and Behavior, 44*(5), 390-397. doi:10.1016/j.jneb.2010.03.001

Heatherton, T. F., Striepe, M., & Wittenberg, L. (1998). Emotional distress and disinhibited eating: The role of self. *Personality and Social Psychology Bulletin, 24*(3), 301-313. doi:Doi 10.1177/0146167298243007

Herman, C. P., & Polivy, J. (1984). A boundary model for the regulation of eating. *Res Publ Assoc Res Nerv Ment Dis, 62*, 141-156.

Jansen, A., Havermans, R. C., & Nederkoorn, C. (2011). Cued Overeating. *Handbook of Behavior, Food and Nutrition*, 1431-1443. doi:10.1007/978-0-387-92271-3_92

Kaplan, H. I., & Kaplan, H. S. (1957). The psychosomatic concept of obesity. *J Nerv Ment Dis, 125*(2), 181-201.

Keskitalo, K., Tuorila, H., Spector, T. D., Cherkas, L. F., Knaapila, A., Kaprio, J., . . . Perola, M. (2008). The Three-Factor Eating Questionnaire, body mass index, and responses to sweet and salty fatty foods: a twin study of genetic and environmental associations. *Am J Clin Nutr, 88*(2), 263-271.

Llewellyn, C. H., van Jaarsveld, C. H., Johnson, L., Carnell, S., & Wardle, J. (2010). Nature and nurture in infant appetite: analysis of the Gemini twin birth cohort. *Am J Clin Nutr, 91*(5), 1172-1179. doi:10.3945/ajcn.2009.28868

Macht, M. (2008). How emotions affect eating: A five-way model. *Appetite, 50*(1), 1-11. doi:DOI 10.1016/j.appet.2007.07.002

Parkinson, K. N., Drewett, R. F., Le Couteur, A. S., Adamson, A. J., & T, G. M. S. C. (2010). Do maternal ratings of appetite in infants predict later Child Eating Behaviour Questionnaire scores and body mass index? *Appetite, 54*(1), 186-190. doi:DOI 10.1016/j.appet.2009.10.007

Pearson, C. M., Riley, E. N., Davis, H. A., & Smith, G. T. (2014). Research Review: Two pathways toward impulsive action: an integrative risk model for bulimic behavior in youth. *Journal of Child Psychology and Psychiatry, 55*(8), 852-864. doi:10.1111/jcpp.12214

Piernas, C., & Popkin, B. M. (2011). Food Portion Patterns and Trends among U.S. Children and the Relationship to Total Eating Occasion Size, 1977-2006. *Journal of Nutrition, 141*(6), 1159-1164. doi:10.3945/jn.111.138727

Posada, D., & Buckley, T. R. (2004). Model selection and model averaging in phylogenetics: advantages of akaike information criterion and bayesian approaches over likelihood ratio tests. *Syst Biol, 53*(5), 793-808. doi:10.1080/10635150490522304

Posthuma, D., Beem, A. L., de Geus, E. J., van Baal, G. C., von Hjelmborg, J. B., Iachine, I., & Boomsma, D. I. (2003). Theory and practice in quantitative genetics. *Twin Res, 6*(5), 361-376. doi:10.1375/136905203770326367

Price, T. S., Freeman, B., Craig, I., Petrill, S. A., Ebersole, L., & Plomin, R. (2000). Infant zygosity can be assigned by parental report questionnaire data. *Twin Res, 3*(3), 129-133.

Rijsdijk, F. V., & Sham, P. C. (2002). Analytic approaches to twin data using structural equation models. *Brief Bioinform, 3*(2), 119-133.

Rodgers, R. F., Paxton, S. J., McLean, S. A., Campbell, K. J., Wertheim, E. H., Skouteris, H., & Gibbons, K. (2014). Maternal negative affect is associated with emotional feeding practices and emotional eating in young children. *Appetite, 80*, 242-247. doi:DOI 10.1016/j.appet.2014.05.022

Schachter, S., Goldman, R., & Gordon, A. (1968). Effects of fear, food deprivation, and obesity on eating. *J Pers Soc Psychol, 10*(2), 91-97.

Steinsbekk, S., Belsky, J., & Wichstrom, L. (2016). Parental Feeding and Child Eating: An Investigation of Reciprocal Effects. *Child Development*. doi:10.1111/cdev.12546

Steinsbekk, S., & Wichstrom, L. (2015). Predictors of Change in BMI From the Age of 4 to 8. *Journal of Pediatric Psychology*. doi:10.1093/jpepsy/jsv052

Stunkard, A. J., & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *J Psychosom Res, 29*(1), 71-83.

Sung, J., Lee, K., Song, Y. M., Lee, M. K., & Lee, D. H. (2010). Heritability of Eating Behavior Assessed Using the DEBQ (Dutch Eating Behavior Questionnaire) and Weight-related Traits: The Healthy Twin Study. *Obesity (Silver Spring), 18*(5), 1000-1005. doi:Doi 10.1038/Oby.2009.389

Swinburn, B. A., Sacks, G., Hall, K. D., McPherson, K., Finegood, D. T., Moodie, M. L., & Gortmaker, S. L. (2011). The global obesity pandemic: shaped by global drivers and local environments. *Lancet, 378*(9793), 804-814. doi:10.1016/S0140-6736(11)60813-1

Takizawa, R., Danese, A., Maughan, B., & Arseneault, L. (2015). Bullying victimization in childhood predicts inflammation and obesity at mid-life: a five-decade birth cohort study. *Psychological Medicine, 45*(13), 2705-2715. doi:10.1017/S0033291715000653

Tan, C. C., & Holub, S. C. (2015). Emotion Regulation Feeding Practices Link Parents' Emotional Eating to Children's Emotional Eating: A Moderated Mediation Study. *Journal of Pediatric Psychology*. doi:10.1093/jpepsy/jsv015

Tholin, S., Rasmussen, F., Tynelius, P., & Karlsson, J. (2005). Genetic and environmental influences on eating behavior: the Swedish Young Male Twins Study. *Am J Clin Nutr, 81*(3), 564-569.

Trudeau, F., Shephard, R. J., Bouchard, S., & Laurencelle, L. (2003). BMI in the Trois-Rivieres study: child-adult and child-parent relationships. *Am J Hum Biol, 15*(2), 187-191. doi:10.1002/ajhb.10131

van Jaarsveld, C. H., Johnson, L., Llewellyn, C., & Wardle, J. (2010). Gemini: a UK twin birth cohort with a focus on early childhood weight trajectories, appetite and the family environment. *Twin Res Hum Genet, 13*(1), 72-78. doi:10.1375/twin.13.1.72

van Strien, T., & Ouwens, M. A. (2003). Counterregulation in female obese emotional eaters: Schachter, Goldman, and Gordon's (1968) test of psychosomatic theory revisited. *Eat Behav, 3*(4), 329-340.

van Strien, T., Frijters, J. E. R., Bergers, G. P. A., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (Debq) for Assessment of Restrained, Emotional, and External Eating

Behavior. *International Journal of Eating Disorders, 5*(2), 295-315. doi:Doi 10.1002/1098-108x(198602)5:2<295::Aid-Eat2260050209>3.0.Co;2-T

Viana, V., Sinde, S., & Saxton, J. C. (2008). Children's eating behaviour questionnaire: associations with BMI in Portuguese children. *British Journal of Nutrition, 100*(2), 445-450. doi:Doi 10.1017/S0007114508894391

Wardle, J., & Boniface, D. (2008). Changes in the distributions of body mass index and waist circumference in English adults, 1993/1994 to 2002/2003. *Int J Obes (Lond), 32*(3), 527-532. doi:10.1038/sj.ijo.0803740

Wardle, J., Guthrie, C. A., Sanderson, S., & Rapoport, L. (2001). Development of the Children's Eating Behaviour Questionnaire. *J Child Psychol Psychiatry, 42*(7), 963-970.

Willner, P., Muscat, R., & Papp, M. (1992). Chronic mild stress-induced anhedonia: a realistic animal model of depression. *Neurosci Biobehav Rev, 16*(4), 525-534.

Zlatevska, N., Dubelaar, C., & Holden, S. S. (2014). Sizing Up the Effect of Portion Size on Consumption: A Meta-Analytic Review. *Journal of Marketing, 78*(3), 140-154.

Figures and Tables

Table 1 Descriptive statistics for the analysis sample (n=3784; 1892 twin pairs)

	N (%) or Mean (SD)
Total	3784
Zygosity	
MZ pairs	613 (32.4)
DZ pairs	1279 (67.6)
Sex	
Males	1860 (49.2)
Females	1924 (50.8)
Gestational age (weeks)	36.21 (2.47)
Weight at birth (kg)	2.46 (0.54)
Age at 16 months (months)	15.82 (1.15)
Emotional Overeating at 16 months	1.64 (0.59)
Age at 5 years (years)	5.15 (0.13)
Emotional Overeating at 5 years	1.38 (0.48)

Table 2 Twin correlations (95% Confidence Intervals) for emotional overeating scores measured at 16 months and 5 years

	MZ[1]	**DZ[1]**
16 months **Twin correlations (95% CI[1])**	0.97 95% CI [0.97, 0.98]	0.92 95% CI [0.92, 0.93]
5 years **Twin correlations (95% CI)**	0.97 95% CI [0.97, 0.98]	0.95 95% CI [0.94, 0.96]
16 months/5 years **Twin correlations (95% CI)**	0.25 95% CI [0.19, 0.30]	0.25 95% CI [0.20, 0.31]

[1]Abbreviations: MZ: Monozygotic; DZ: Dizygotic, CI: Confidence Intervals, CT/CT, cross-twin cross-time

Table 3: Fit statistics for longitudinal models for Emotional Overeating at 16 months and 5 years

Fit Statistics						
Model	**-2LL[1]**	**df[1]**	**Δ X^2 (df)**	**p-value**	**BIC[1]**	**ΔBIC[1]**
Sat[1]	9448.452	5732			-16900.861	
ACE[1]	9456.383	5749	13.218 (17)	0.72	-16938.395	37.534
Submodel 1[2]	9464.649	5744	8.267 (1)	<0.01	-16938.035	-0.36
Submodel 2[3]	9468.468	5745	12.085 (2)	<0.01	-16939.898	1.503

Table 4 shows fit statistics for the longitudinal analyses of EOE measured at 16 months and 5 years. The BIC and ΔBIC was used to identify the best fitting model. More parsimonious submodels with a lower BIC value compared to the full ACE model, and with a BIC change of at least 2 compared to the full ACE model, are preferred (Trudeau et al., 2003). Submodel 2 had a lower BIC value than the full ACE model, but the change in BIC was not sufficient to select it over the full ACE model (Δ = -1.503).

[1] Abbreviations: 2LL: -2 times log-likelihood of data; Δ-2LL: difference in 2 times log-likelihood; df: degrees of freedom; Δ X^2: change in chi-square; BIC: Bayesian Information Criterion; Δ BIC: change in Bayesian Information; Sat: Saturated model; ACE: Full model including all factors.

[2] Submodel 1: In this submodel genetic covariation between EOE at 16 months and 5 years was constrained to 0 (i.e. Bivariate A and the additive genetic correlation (r_A) were dropped). Submodel 1 is nested in and compared against the full ACE model.

[3] Submodel 2: In this Submodel both genetic covariation and unique environmental covariation between EOE at 16 months and 5 years were constrained to 0 (i.e. Bivariate A, Bivariate E, the additive genetic correlation (r_A) and the non-shared environmental correlation (r_E) were dropped). Submodel 2 is nested in and compared against the full ACE model.

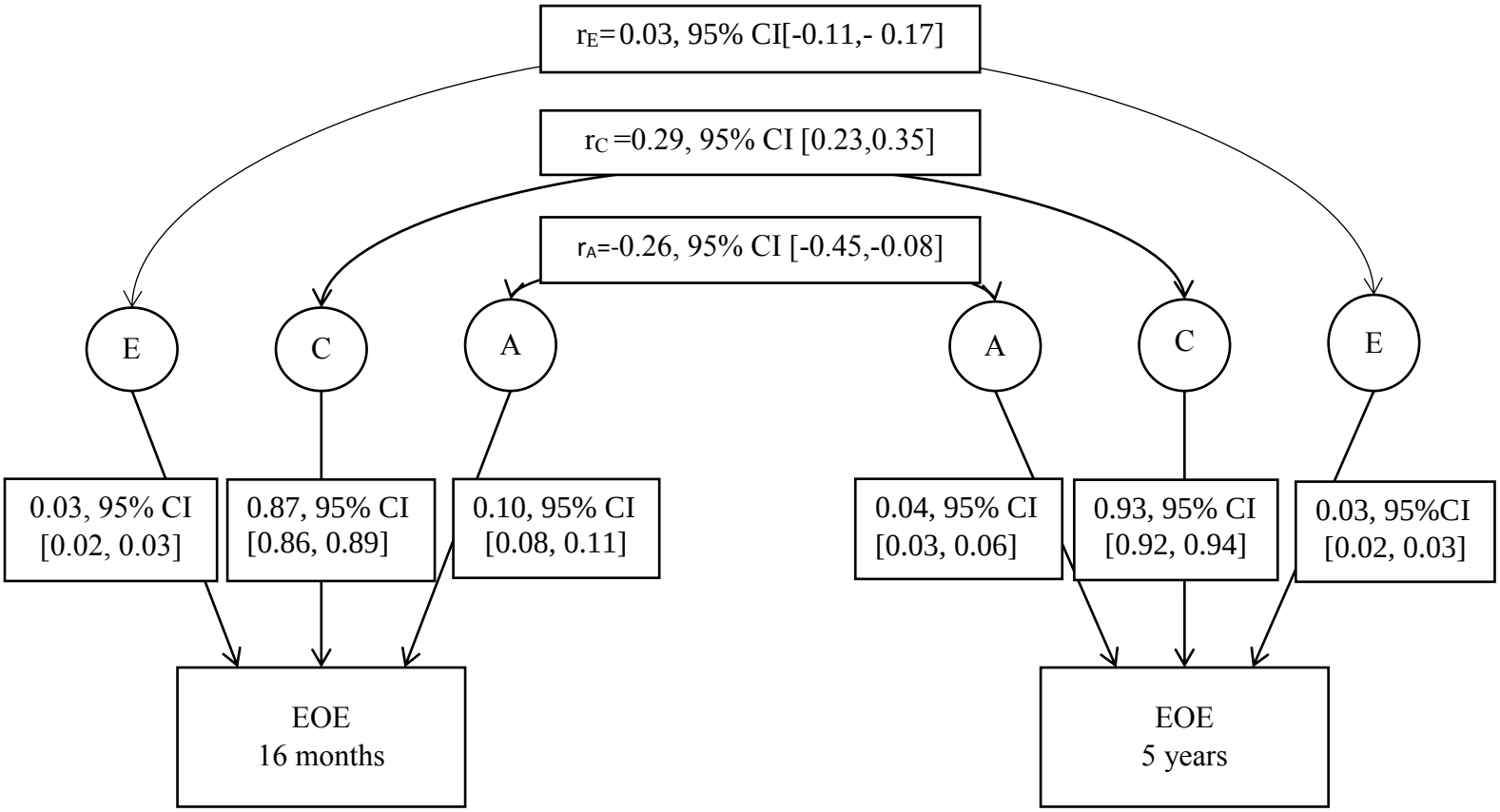

Figure 1 shows the full longitudinal model including all parameters. The rectangular boxes represent the measured phenotype (EOE) using the Child Eating Behavior Questionnaire at 16 months and 5 years. The circles indicate the latent factors of additive genetic effects (A), shared environmental effects (C) and non-shared environmental effects (E). The straight single-headed arrows reflect casual pathways with the variance explained by each latent factor (including 95% confidence intervals). The etiological correlations are shown on the curved double-headed arrows. These indicate the proportion of genetic (r_A), shared environmental (r_C) and unique environmental (r_E) influences that are common across the two ages. The non-significant etiological correlation (r_E), with a 95% Confidence Interval crossing 0, is represented as a dotted line. Bivariate estimates (not shown on the path diagram) quantify the proportion of the longitudinal association ($r=0.25$, $p<0.001$)

attributable to common genetic (bivariate A: -0.07; 95% CI [-0.12, -0.02], shared environmental (bivariate C: 1.07; 95% CI [1.03, 1.11], and unique environmental factors (bivariate E: 0.00; 95% CI [0.01, 0.02] at both 16 months and 5 years.

Appendix 1 Sex limitation models

Table 1.1a Sex Limitation Model for EOE measured at 16 months, ACE estimates for males and females

Model	Male			Female				
	A_m[1]	C_m[1]	E_m[1]	A_f[1]	C_f[1]	E_f[1]	r_A[1]	r_C[1]
Full sex limitation (r_A=free)	0.08 (0.06-0.10)	0.88 (0.85-0.89)	0.04 (0.04-0.05)	0.11 (0.9-0.12)	0.88 (0.86-0.90)	0.01 (0.01-0.01)	0.5 (0.48-0.5)	1
Full sex limitation (r_C=free)	0.08 (0.06-0.10)	0.88 (0.85-0.89)	0.04 (0.04-0.05)	0.11 (0.9-0.12)	0.88 (0.86-0.90)	0.01 (0.01-0.01)	0.5	1 (0.99-1.00)
Common effects model (rA=0.5, rC=1)	0.08 (0.06-0.10)	0.88 (0.85-0.89)	0.04 (0.04-0.05)	0.11 (0.9-0.12)	0.88 (0.86-0.90)	0.01 (0.01-0.01)	0.5	1
	A		**C**		**E**		**scalar**	
Scalar Model	0.09 (0.0.08-0.11)		0.88 (0.86-0.89)		0.03 (0.02-0.03)		0.95 (0.92-0.98)	
	A		**C**		**E**		r_A[1]	r_C[1]
Null model (no sex differences)	0.09 (0.08-0.11)		0.88 (0.86-0.89)		0.03 (0.02-0.03)		0.5	1

[1] Abbreviations: A: additive genetic component of variance; C: shared environmental component of variance; E: unique environmental component of variance; r_A: genetic correlation, r_C: shared environmental correlation, r_E: non-shared environmental correlation.

Table 1.1 b Fit statistics for sex limitation modelling for EOE at 16 months

EOE 16 months Model	Comparison	Ep[1]	-2LL[1]	Df[1]	Δ X^2 (df)[1]	*p*-value	*BIC*
1 Saturated model		23	6052.284	3691			-21756.553
2 Full sex limitation (r_A=free)	1	9	6133.728	3705	81.44 (14)	<0.001	-21780.588
3 Full sex limitation (r_C=free)	1	9	6133.728	3705	81.44 (14)	<0.001	-21780.588
4 Common effects model (r_A=0.5, r_C=1)	2 & 3	8	6133.728	3706	0.00 (1)	1	-21788.122
5 Scalar Model	4	6	6260.272	3708	127.544 (2)	<0.001	-21676.647
6 Null model (no sex differences)	5	5	6274.631	3709	14.3359 (1)	<0.001	-21669.822

[1] Abbreviations: Ep: estimated parameters, -2LL: -2 log-likelihood of data, Df: degrees of freedom, BIC: Bayesian Information Criterion

Table 1.2a Sex Limitation Model for EOE measured at 5 years, ACE estimates for males and females

Model	Male			Female				
	A_m[1]	C_m[1]	E_m[1]	A_f[1]	C_f[1]	E_f[1]	r_A[1]	r_C[1]
Full sex limitation (r_A=free)	0.25 (0.00-0.79)	0.74 (0.18-0.99)	0.00 (0.00-0.35)	0.23 (0.00-0.79)	0.75 (0.22-0.99)	0.02 (0.00-0.33)	0.5 (0.00-0.5)	1
Full sex limitation (r_C=free)	0.25 (0.00-0.79)	0.74 (0.18-0.99)	0.00 (0.00-0.35)	0.23 (0.00-0.79)	0.75 (0.22-0.99)	0.02 (0.00-0.33)	0.5	0.1 (0.19-1)
Common effects model (rA=0.5, rC=1)	0.04 (0.02-0.06)	0.93 (0.91-0.95)	0.03 (0.02-0.03)	0.06 (0.04-0.08)	0.92 (0.90-0.94)	0.02 (0.02-0.03)	0.5	1
	A		**C**		**E**		**scalar**	

Scalar Model	0.05 (0.04-0.06)	0.93 (0.91-0.94)	0.03 (0.02-0.03)	0.099 (0.97-1.00)	
	A	**C**	**E**	**r_A[1]**	**r_C[1]**
Null model (no sex differences)	0.05 (0.04-0.06)	0.93 (0.91-0.94)	0.03 (0.02-0.03)	0.5	1

[1] Abbreviations: A: additive genetic component of variance; C: shared environmental component of variance; E: unique environmental component of variance; r_A: genetic correlation, r_C: shared environmental correlation, r_E: non-shared environmental correlation.

Table 1.2 b Fit statistics for sex limitation modelling for EOE at 5 years

5 years EOE[1] Model	**Comparison**	**Ep[1]**	**-2LL[1]**	**Df[1]**	**Δ X^2 (df)[1]**	***p*-value**	**BIC[1]**
1 Saturated model		23	3027.727	1915			-11400.320
2 Full sex limitation (r_A=free)	1	9	3629.984	1929	602.257 (14)	<0.001	-10903.542
3 Full sex limitation (r_C=free)	1	9	3629.984	1929	602.257 (14)	<0.001	-10903.542
4 Common effects model (r_A=0.5, r_C=1)	2 & 3	8	3043.86	1930	-586.125 (1)	1	-11497.201
5 Scalar Model	4	6	3044.784	1932	0.925 (2)	0.63	-11511.345
6 Null model (no sex differences)	5	5	3044.813	1933	0.029 (1)	0.86	-11518.851

[1] Abbreviations: EOE: Emotional Overeating; Ep: estimated parameters, -2LL: -2 log-likelihood of data, Df: degrees of freedom, BIC: Bayesian Information Criterion