# THE AETIOLOGY OF EMOTIONAL EATING IN CHILDHOOD

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**Declaration**

I, Moritz Herle, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the text.

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# Abstract

Emotional overeating (EOE) and under-eating (EUE) in response to stress are common behaviours which emerge in childhood. However, their aetiology is largely unknown. This thesis analysed data from a UK cohort of 2402 families with twins to investigate the aetiology of EOE and EUE in childhood. Study 1 demonstrated low heritability of EOE at 16 months (9%) and five years (3%). The majority of individual differences were explained by shared environmental factors (16 months: 89%, 5 years: 95%). However, only 8% of these environmental factors were found to influence EOE at both time points. EOE was found to track (r = 0.25) and this association was explained by shared environmental factors. Study 2 established low heritability (7%) for EUE and dominance of shared environmental factors (91%) at five years. EOE and EUE correlated (r = 0.43) and shared environmental factors accounted for this association. However, their aetiologies were partly distinct, with 25% of shared environmental factors affecting both behaviours. Study 3 characterised the child, parent and environmental factors associated with child EOE and EUE. Emotional feeding was found to influence both EOE and EUE, whereas parental pressure to eat was only associated with EUE. Maternal emotional overeating was specifically linked to EOE. Study 4 provided evidence for the causal effect of emotional feeding on child EOE using prospective data. Study 5 found significant gene-environment interactions underlying EOE and EUE whereby a stressful home environment increased their heritability. Study 6 replicated Study 2 in an independent sample. Study 7 showed that parental belief of twins' zygosity did not impact their ratings of child eating behaviours. The thesis showed that EOE and EUE are learned and not inherited in childhood. Their aetiology is complex and due to specific parental behaviours which deem to be promising intervention targets.

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# Chapter 1 Emotional eating

## 1.1 Aims of the literature review

This literature review will introduce the concept of emotional over- and under-eating. The first sections will give an overview of the most commonly used tools to measure emotional eating in adults and children. Furthermore, research on the health outcomes associated with both emotional overeating and emotional under-eating are reviewed. Then, previous research addressing the aetiology of emotional over- and under-eating in childhood will be discussed. First, focussing on psychological theory, as well as evidence from adult twin studies. Following, literature on the childhood factors associated with both behaviours is evaluated, covering parental feeding practices and other external factors.

## 1.2 What is emotional eating?

Negative emotions and stress do not only impact our mood, but can also have a substantial effect on our appetite. This tendency, referred to as emotional eating, can be separated into two different patterns. For some people stress and negative emotions lead to increased craving of highly palatable foods, whereas others lose their appetite altogether. The former - emotional overeating - is a widely recognised behaviour. In fact, traditional psychological theory suggests that emotional overeating is fundamental to the development of obesity (Schachter, 1968; Schachter, Goldman, & Gordon, 1968). Both emotional over- and under-eating are common behaviours. On average one third of adults experience an increase of appetite and about half report a decrease of appetite when exposed to stress and negative emotions (Macht, 2008).

Even though these behaviours are common in the population, surprisingly little is known about their aetiology. The majority of research has focussed on emotional overeating, due to the potential negative health consequences of overconsuming highly palatable foods, for obesity and dietary quality. Emotional under-eating has received even less attention. Both behaviours have been found to emerge in early childhood (Ashcroft, Semmler, Carnell, van Jaarsveld, & Wardle, 2008). However, few studies have investigated how these behaviours develop in early life.

Starting with Schachter's early work on emotional eating in the second half of the previous century (Schachter, 1968; Schachter et al., 1968), behavioural scientists have aimed to measure and study emotional eating behaviour, first in adults and then children. In order to study emotional eating, researchers have used behavioural

observations, as well as psychometric measures to explore emotional eating behaviour in larger samples.

## 1.3 Measuring emotional eating

### 1.3.1 Behavioural observations

Behavioural observations of emotional eating can be conducted with animals or humans. Laboratory based research has distinct advantages as stress exposure can be manipulated and food intake can be measured with precision, but these studies lack the ecological validity of observational research conducted in more naturalistic settings.

#### 1.3.1.1. Animal studies

Early animal studies investigated the impact of induced stress on food intake. Typically, in studies of this type, rodents are exposed to environmental stressors of varying intensity and duration. Brief stressors range from tail pinches to electric shocks, while exposure to continuous noise for an extended period of time induces low level but enduring stress. Researchers monitor and compare food intake of the rodents in the different stress conditions, in order to make inferences about the effect of stress on food intake.

Results from early studies suggested that exposure to intense stressors led to a decrease in food intake in rats (Sterritt, 1962; Sterritt & Shemberg, 1963). On the other hand mild stressors, as well as chronic stress, resulted in increased food consumption in rodents (Alario, Gamallo, Beato, & Trancho, 1987; Dallman et al., 2003; Kupferman, 1964; Pare, 1964; Sampson, Muscat, Phillips, & Willner, 1992; Strongman, Coles, Remington, & Wookey, 1970; Ullman, 1951; Willner, Muscat, & Papp, 1992). Additional research demonstrated substantial individual variation in stress reactivity and stress response strategy, with some rats showing increased grooming behaviour instead of food intake in response to stress (Macht, Krebs, Weyers, & Janke, 2001). These studies highlighted individual differences in how animals respond to stress – not all animals demonstrated the same feeding response.

Animal studies can be advantageous insofar as they enable researchers to fully control the intensity and duration of the environmental stressor. Moreover, dietary intake can be measured with great precision. However, the extent to which animal behaviour in a highly controlled setting can be extrapolated to humans is a matter of much debate. Overall, animal studies suggested large individual differences in appetite changes in response to stress. In addition, different stressors had different

effects - intense stress resulted in a decrease of food intake, whereas chronic low level stress was associated with increased consumption.

#### 1.3.1.2 Behavioural observations in humans

In humans, similar paradigms have been applied to observe changes in appetite and food intake in adults using laboratory-based research methods. Commonly, participants undergo a stress induction, which often consists of being asked to prepare a presentation on a controversial topic with the expectation of being reviewed or graded on their performance. Then participants are presented with an array of foods and told to eat as much as they desire. Researchers compare the food intake of participants exposed to the stress induction with participants performing a neutral task. This paradigm has been used to study the effect of stress on food intake in adults, in order to create situations in which emotional over or under-eating occurs. In one study, participants (stress group, n = 34; control group, n = 34) in the stress condition were asked to prepare a speech on a controversial topic, expecting it to be filmed and assessed. Participants in the control group were asked to read a neutral text. Emotional overeating was measured for both groups and all participants were presented with a buffet of palatable foods and allowed to eat as much as they wanted for 15 minutes. While there were no differences in food intake between the control and the intervention groups, participants scoring high on emotional overeating were found to eat more in the stress condition (Oliver, Wardle, & Gibson, 2000). These results highlighted the existence of individual differences in underlying predisposition to emotionally overeat in response to stressors – some adults have a tendency to do this, while others do not. The origin of this predisposition, however, cannot be deciphered from these experimental studies, and is a question of great interest to researchers.

Similar study designs have been used to explore emotional eating behaviour in children. Of course, the stress induction process in children needs careful consideration in order to safeguard their wellbeing. Studies have used some imaginative tasks, such as asking children to finish a jigsaw puzzle, with the promise of a reward upon completion. In the stress condition, a piece of the puzzle is missing and the children are not allowed to receive the prize initially, while the researcher was looking for the lost puzzle piece. In the control condition children were given all puzzle pieces from the beginning, and are able to complete the puzzle. Following the task, children are presented with different snack foods and informed they could eat as much as they want, and their consumption is measured. This paradigm has been deemed useful and previous studies have been able to successfully quantify the direct

effect of stress on children's food consumption (Blissett, Haycraft, & Farrow, 2010; Farrow, Haycraft, & Blissett, 2015a).

Like animal research, human observational research conducted in the laboratory has many advantages. Stress intensity and exposure can be controlled and food intake can be measured precisely, and analysed by different food groups. However, laboratory-based research is time consuming and expensive, making it prohibitive for large sample sizes. Furthermore, laboratory situations can be seen as artificial, only providing a snapshot of emotional eating in this particular instance. Moreover, experimental stressors created in the laboratory for human participants cannot accurately reflect chronic stress, preventing investigations into the impact of low intensity, enduring stress on food intake. Laboratory-based tasks can also be onerous for the participants involved (and their parents in the case of children).

In order to address some of these limitations, researchers have tracked mood, stress levels and food consumption, in naturalistic settings. Emotional state and motivation to eat were measured in a group of university students (n = 22) over four weeks leading up to an important exam. In comparison to a control group (n = 20) with no exam, students reported a higher tendency to consume more food in order to distract themselves (Macht, Haupt, & Ellgring, 2005). On the other hand, an observational study of soldiers (n = 475) reported a decrease of food intake when in combat. Soldiers reported lack of opportunity and time as the main reason for reduced eating, but fear was given as the second most important reason for decreased appetite. (Popper, Smits, Meiselman, & Hirsch, 1989). These studies suggest the situation and type of stressor may influence a person's emotional eating response. More novel research investigated the effect of witnessing a natural disaster on eating behaviour. A study measured emotional eating in adult women (n = 105) before and after the 2010 Christchurch, New Zealand, earthquake (Kuijer & Boyce, 2012). Results showed that the stress of a natural disaster exacerbated emotional overeating only in those women who rated themselves highly on emotional overeating before the earthquake, resulting in an increased intake of snack foods. These findings suggest that a single highly stressful situation does not elicit new emotional overeating behaviour in previously unaffected individuals, but disproportionately affects those with a predisposition to overeating in response to stress (Kuijer & Boyce, 2012). Again, highlighting that there are individual differences in predisposition to emotionally overeat, but the cause of this predisposition cannot be teased out by these observational and experimental studies.

Observational and laboratory based studies have suggested substantial variation in how stress affects food consumption, finding support for both increased and decreased intake. Stress intensity and duration have been implicated as potential factors explaining the divergent effects of stress on food intake, but underlying predisposition may also determine if an individual is more likely to decrease or increase their appetite in response to stress. A recent review, summarising evidence from animal and human research concluded that for some people, once established, eating in response to stress becomes habitual resulting in repeated overconsumption (Turton, Chami, & Treasure, 2017). Much less is known about the tendency to under-eat in response to stress and negative emotions.

### 1.3.2 Psychometric measures

In order to facilitate research in larger clinical and population based samples, psychometric questionnaires quantifying eating behaviours have been developed. Usually these tools measure emotional eating alongside other eating behaviours deemed to be related to appetite regulation such as sensitivity to internal satiety cues (fullness sensitivity) and responsiveness to food cues in the environment (wanting to eat, or eat more, in response to the sight, smell or taste of highly palatable food). The different questionnaires measuring emotional over- and under-eating in adults and children are discussed below.

#### 1.3.2.1 Emotional overeating

##### 1.3.2.1.1 Adults

The Three Factor Eating Questionnaire (TEFQ) (Stunkard & Messick, 1985) and The Dutch Eating Behaviour Questionnaire (DEBQ) (Van Strien, Frijters, Bergers, & Defares, 1986) are the most commonly used psychometric measures of eating behaviour for adult samples. The original Three-Factor Eating Questionnaire consisted of 51 items capturing three distinct eating traits. For 36 of the items, participants indicate if the statements apply to them by selecting either ‘true’ or ‘false’. For the remaining 15 items, participants rate how often they engage in the behaviours described, using a four point Likert-scale ranging from ‘not at all’ to ‘very much’. The items cluster in three factors: Cognitive Restraint, Disinhibition and Hunger. Cognitive restraint refers to the deliberate attempt to restrict food intake in order control weight (example item: “I deliberately take small helpings as a means of controlling my weight”). Disinhibition, on the one hand refers to eating behaviour in situations where food is available and appealing (example item: “I usually eat too much at social occasions, like parties and picnics”). But the Disinhibition factor also includes items

probing eating in response to stress and negative emotions (example item: “When I feel blue, I often overeat”). The Hunger factor examines the general appetite of the participants (example item: “I am always hungry enough to eat at any time”).

Since the original TEFQ was published in 1985, a revised condensed version has been developed (Karlsson, Persson, Sjostrom, & Sullivan, 2000). This Three-Factor Eating Questionnaire – R18 (TEFQ18) version is shorter, consisting of 18 items. Participants rate how much the statements apply to them using a four point Likert-scale ranging from ‘definitely true’ to ‘definitely false’. In comparison to the previous longer version, the factor Cognitive restraint remained consistent, however two new factors emerged: Uncontrolled eating (Disinhibition) and Emotional eating. Uncontrolled eating was a combination of the previously separate Disinhibition and Hunger subscales. Items tapping into eating in response to negative emotions clustered into its own factor, named Emotional eating (Karlsson et al., 2000). Additional analyses, including food frequency (n = 887) data, implied that high Cognitive restraint was associated with reduced caloric intake as well as increased intake of healthy foods such as green vegetables and reduced intake of unhealthy ‘junk’ foods. Overall, high Uncontrolled eating was found to be associated with a higher intake of energy-dense foods, while Emotional eating was found to correlate with higher intake of snacks, such as cakes and biscuits (de Lauzon et al., 2004).

Around the same time the original TFEQ was developed, a different group of researchers developed the Dutch Eating Questionnaire (DEBQ) (Van Strien et al., 1986). The original version of the DEBQ is shorter than the TEFQ (33 items) and participants use a five point Likert-scale to rate the applicability of the items to their own habitual behaviour. Similar to the TFEQ, items cluster into three factors: Restrained eating, Emotional eating and External eating. Restraint eating refers to dieting behaviours similar to the TFEQ Cognitive restraint scale (example item: “Do you deliberately eat food that are slimming”). External eating is similar to the TFEQ ‘Disinhibition’ or ‘Uncontrolled eating’ scale, and measures eating behaviour when foods are available and appealing (example item: “If food smells or looks good, do you eat more than usual?”). Finally, the Emotional eating scale of the DEBQ is similar to the Emotional eating factor included in the TFEQ-18 (example item: “Do you have the desire to eat more when you are disappointed?”). The Emotional eating scale of DEBQ is extensive, including a total of 12 items covering a wider range of emotions from boredom, to sadness and fear. In comparison, only three items are included in the TFEQ-R18 Emotional eating scale, which probes eating in response to anxiety, sadness and loneliness (Karlsson et al., 2000).

In order to address the potential different effects distinct emotional states might have on eating, the Emotional Eating Scale (EES) was developed (Arnow, Kenardy, & Agras, 1995). The questionnaire consists of a list of 25 emotions and participants are asked to indicate to what degree these emotions change their desire to eat using a five point Likert-scale (from “no desire to eat” to “an overwhelming urge to eat”). Factor analyses suggested three separate clusters of emotions: Anger/Frustration; Anxiety and Depression. Like the TEFQ and DEBQ, the EES does not measure emotional under-eating.

More recently a new comprehensive measure was developed, the Adult Eating Behaviour Questionnaire (AEBQ) (Hunot et al., 2016). This tool consists of 35 items and aims to cover a comprehensive range of adult eating behaviours than the previously established TFEQ, DEBQ and EES. The AEBQ broadly differentiates between ‘food avoidant’ and ‘food approach’ eating behaviours. Four ‘food avoidant’ behaviours characterise a lower interest in food, a less avid appetite, and lower food intake: Satiety responsiveness measures sensitivity to internal fullness (example item: “I get full up easily”), Food fussiness describes the tendency to refuse to try new foods or to be picky about foods an individual is willing to eat based on aspects such as texture (example item: “I refuse new foods at first”), Emotional under-eating measures the tendency to eat less in response to negative emotions (example item: “I eat less when I am upset”) and Slowness of eating describes general eating pace (example item: “I eat slowly”). Conversely, four ‘food approach’ behaviours describe a more avid appetite, a greater interest in food, and a tendency to overeat. Food responsiveness refers to the tendency to want to eat if the food available is particularly appealing and attractive (example item: “When I see or smell food that I like, it makes me want to eat”), Hunger indicates overall appetite size (example: “I often feel hungry”) and Enjoyment of food measures the pleasure and reward experienced from eating (example item: “I look forward to mealtimes”). Finally, the AEBQ includes five items clustering into a subscale of Emotional overeating. Items included are similar to the ones in previous eating behaviour questionnaires and cover different negative emotions (example item: “I eat more when upset”).

The TFEQ (TFEQ-18), DEBQ, EES and the new AEBQ, all only include items relating to negative emotions. However, it is also possible that individuals change their eating behaviour in response to positive emotions. The Emotional Appetite Questionnaire (EAQ) was developed to measure appetite changes in response to a broader range of emotions (Nolan, Halperin, & Geliebter, 2010). Participants are asked to indicate if they eat less, the same, or more (using a 1-9 Likert-scale) in response to 14 different

emotions, ranging from sad, to tired, to enthusiastic. Furthermore, changes in appetite were rated in response to different situations such as 'When under pressure' or 'After receiving good news'.

The TFEQ (TFEQ-18), DEBQ, EES, EAQ, and the AEBQ represent the most common psychometric measures of adult emotional overeating; although the AEBQ is very recent. The tools differ regarding the number of items included in each emotional overeating scale as well as the range of emotions covered. However overall, items tend to be fairly similar across the different questionnaires. A list of all items included in the different questionnaires examining emotional overeating can be found in the **Appendix 1.1**.

#### 1.3.2.1.2 Children

Several psychometric questionnaires are available to measure emotional overeating in children. Some of which were developed by adapting adult eating behaviour questionnaires into parent-rated or child-rated tools. A full list of items measuring emotional overeating in children can be found in **Appendix 1.2**.

The DEBQ-Parent version was developed to measure the same eating behaviours in children, and results confirmed the same three factor structure of eating behaviour in children as is observed in adults (Braet & VanStrien, 1997). The three subscales were: Restraint (example item: "How often does your child try not to eat between meals?"), External eating (example item: "If food tastes good to your child, does he or she eat more than usual?") and Emotional overeating (example item: "Does your child have the desire to eat, when he/she is anxious, worried or tense?"). The total questionnaire consists of 33 items, of which 13 relate to emotional overeating. In addition to the parent rated version of the DEBQ, a child self-rated version was subsequently created (van Strien & Oosterveld, 2008). This version was shortened (20 items) and the wording of some items adapted slightly to ensure that items were age appropriate and easy to understand. The original scale was developed for 7-12 year old children and the same three eating behaviour scales were identified: Restrained eating (example: "I watch what I eat"), External eating (example: "I have desire to eat when I walk past a candy store") and Emotional overeating (seven items, example: I have desire to eat when I am afraid").

In a similar way, the items of the EES, were modified to make them appropriate for children and adolescents (EES-C) (Tanofsky-Kraff, Theim, et al., 2007). The EES-C includes 25 different negative emotions covering a wide range, from nervousness, to sadness and to frustration. When developing this measure, the original sample

included 159 children (USA) of different ages, ranging from eight to 18 years. However this self-reported questionnaire might not be appropriate for young children, due to the length and the complexity of some of the emotions included (e.g. 'resentful' or 'furious').

Another child self-report measure is the Eating Pattern Inventory for Children (EPI-C) (Schacht, Richter-Appelt, Schulte-Markwort, Hebebrand, & Schimmelmann, 2006) which was developed to measure emotional overeating in the context of eating disorders risk and consists of 20 items using a four point Likert-scale. Similar to other questionnaires, the EPI-C includes items relating to dietary restraint and responsiveness to food cues. Emotional overeating was measured with four items tapping into eating more in response to negative emotion (disappointment, loneliness, worry, unhappiness). The original EPI-C was developed in a sample of 8 – 11 year old German children. Just like the EES-C, this self-report measure might be best suited for measuring emotional overeating in late childhood and early adolescence, as older children are more likely to be able to comprehend and respond to the items in a reliable way. However, for younger children these self-reported measures might not be suitable. Parent-rated questionnaires are also available for younger age groups, which offer a more reliable option.

In comparison to the DEBQ-P and EES-C, the Child Eating Behaviour Questionnaire (CEBQ) was specifically designed to measure a variety of eating behaviours in childhood (Wardle, Guthrie, Sanderson, & Rapoport, 2001b). This parent-report questionnaire for children was developed well before the adult version (the AEBQ, described above), which was based on the CEBQ. The CEBQ consists of 35 items and is a widely used parent-report psychometric measure of eight eating behaviours in childhood, hypothesised to play a causal role in the development of under- and overweight. Parents rate the behaviour of their child using a five-point Likert-scale to indicate the frequency with which their child typically demonstrates each behaviour that comprise the 35 items (1 – 5; 'Never', 'Rarely', 'Sometimes', 'Often' and 'Always').

Just like the AEBQ, eating behaviours in the CEBQ are conceptualised as 'food avoidant' and 'food approach'. Food avoidant behaviours refer to eating behaviours regulating eating offset and decrease of intake: Satiety responsiveness describes the sensitivity to internal fullness cues (example item: "My child gets full before his/her meal is finished"); Slowness of eating measures the general eating speed (example item: "My child takes more than 30 minutes to finish a meal"), Food fussiness indicates the tendency of the child to refuse new and unknown foods (example item: "My child

refuses new foods at first") and Emotional under-eating (EUE)[1] refers to the child's tendency to eat less in response to negative emotions (example item: "My child eats less, when she/he is upset"). 'Food approach' behaviours are stipulated to regulate eating onset and increase intake: Food responsiveness describes the child's tendency to eat more if food is tasty (example item: "If allowed my child would eating too much"), Desire to drink measures the tendency of the child wanting to drink (example item: "My child is always asking for a drink") and Enjoyment of food indicates the overall enjoyment a child gets from eating (example item: "My child enjoys eating"). Finally, the questionnaire includes four items asking parents to rate their children's tendency to want to eat more in response to negative emotion; Emotional overeating (EOE)[1]:

- My child eats more when worried
- My child eats more when annoyed
- My child eats more when anxious
- My child eats more when s/he has nothing else to do

The full CEBQ can be found in **Appendix 2.2**.

In comparison to the DEBQ-P, EES-C and EPI-C the CEBQ was specifically developed to measure a large range of eating behaviours in early life. This measure is widely used, has been translated into several different languages (Demir & Bektas, 2017; dos Passos, Gigante, Maciel, & Matijasevich, 2015; E. F. Sleddens, Kremers, & Thijs, 2008; Svensson et al., 2011), and the factor structure has been shown to be robust across many samples and ages (Domoff, Miller, Kaciroti, & Lumeng, 2015; Mallan et al., 2013; Quah et al., 2017). A version has also been developed for infants, to measure feeding behaviour during the first few months of life when infants are still exclusively milk-fed (before any solid food has been introduced) – the Baby Eating Behaviour Questionnaire (BEBQ) (Llewellyn, van Jaarsveld, Johnson, Carnell, & Wardle, 2011). The BEBQ is also a parent-report questionnaire, and it measures four distinct feeding behaviours: Satiety responsiveness, Food responsiveness, Slowness of eating and Enjoyment of food. However, emotional over- and under-eating are not included because it is too difficult to measure these behaviours in early infancy; many

[1] From this point forward emotional overeating and emotional under-eating will be referred to as EOE and EUE if these constructs were measured by the Child Eating Behaviour Questionnaire

mothers who ‘feed on demand’ feed mainly in response to crying, making feeding and crying in infancy inextricably linked (Llewellyn et al., 2011).

#### 1.3.2.2 Emotional under-eating

##### 1.3.2.2.1 Adults

The AEBQ and the EAQ are the only psychometric questionnaires that include items relating to a decrease of appetite in response to negative emotions. The emotional under-eating subscale of the AEBQ consists of five items, covering a range of different negative emotions (example item: “I eat less when I’m upset”) (Hunot et al., 2016). The EAQ allows participants to indicate if they eat less in response to negative and positive situations and is therefore able to measure emotional under-eating (Nolan et al., 2010). None of the other established adult eating behaviour questionnaires - the DEBQ, TFEQ and ESC - include items measuring emotional under-eating.

##### 1.3.2.2.2 Children

For children the CEBQ is the only questionnaire including items on emotional under-eating. The questionnaire includes four items asking parents to rate their children’s tendency to want to eat less in response to negative emotion:

- My child eats less when anxious
- My child eats less when angry
- My child eats less when s/he is tired
- My child eats more when she is happy

The other main child eating behaviour questionnaires - the DEBQ-P, DEBP-C, and EES-C - are adaptations of their respective adult versions and do not include any items relating to emotional under-eating.

#### 1.3.2.3 Emotional over and under-eating in the context of other eating behaviours

In line with the theoretical constructs of ‘food approach’ and ‘food avoidance’ traits, eating behaviours tend to cluster together – ‘food approach’ traits tend to be positively correlated with one another (e.g. Emotional overeating and Food responsiveness), as are the ‘food avoidance traits’ (e.g. Emotional under-eating and Food fussiness). Food avoidance traits and food approach traits also tend to correlate *negatively* (e.g. Food responsiveness and Satiety responsiveness). These associations seem intuitive as children who are very sensitive to satiety cues are presumably less likely

to demonstrate high food responsiveness, as they are less likely to desire food present in their surroundings when not hungry because they are more attuned to their feelings of fullness. Furthermore, children who really enjoy eating food are probably less likely to be very fussy and anxious about trying novel foods, but might be more likely to eat more in response to stress. These patterns of association have repeatedly been described in studies of children using the CEBQ (Cao et al., 2012; Domoff et al., 2015; Mallan et al., 2013; Svensson et al., 2011; Viana, Sinde, & Saxton, 2008).

A similar pattern was found for adult eating behaviours. In line with the CEBQ, the AEBQ subscales also cluster into 'food avoidance' traits which tend to positively correlate with one another, and 'food approach' traits which also positively correlate with one another but tend to negatively correlate with 'food avoidance' traits (Hunot et al., 2016). Furthermore, subscales of other adult eating behaviour questionnaires describe a comparable situation. The TFEQ-18 Emotional eating and Uncontrolled eating subscales correlate positively, whereas there is no association between Emotional eating and Cognitive restraint and negative correlation between Uncontrolled eating and Cognitive restraint (Karlsson et al., 2000).

The exception to this general pattern is the relationship between EOE and EUE. In line with the pattern of interrelationships observed for the other food approach and food avoidance eating behaviours, one would expect EOE and EUE to be negatively correlated, but in fact they are positively correlated. This observation has been made repeatedly across many studies (r = 0.16 - 0.30) (Domoff et al., 2015; Ek et al., 2016; Mallan et al., 2013; Steinsbekk, Belsky, & Wichstrom, 2016; Viana et al., 2008; Wardle, Guthrie, Sanderson, & Rapoport, 2001). This indicates that children who tend to overeat in response to negative emotion, tend also to under-eat in response to emotions. This raises many questions about the nature of their relationship, and raises the possibility that they are, in fact, different expressions of the same underlying trait - the tendency to have one's appetite up- or down-regulated by stress. As outlined previously, observational and experimental studies of animals and humans confirm large variation in stress-related appetite changes, and suggest stress duration and intensity as potential factors influencing down- or up- regulation of food intake, as well as predispositions. The nature of their relationship is currently unknown and needs to be elucidated.

A table summarising all studies that have reported interrelationships between the different eating behaviours of the CEBQ can be found in **Appendix 1.3**.

#### 1.3.2.4 Limitations of psychometric measures

As outlined above, there are several psychometric measures of emotional over and under-eating in adults and children. However the validity of these psychometric measures has been criticised in a recent review (Bongers & Jansen, 2016) which highlighted the large heterogeneity in findings with many studies showing non-significant associations between psychometric measures of emotional overeating and food intake under stress. Furthermore, the authors argued that negative mood states are transient and emotional overeating is only possible in the presence of highly palatable foods. In the light of mixed evidence for an association between high emotional overeating scores and food intake, the authors suggested that psychometric questionnaires of emotional overeating actually assess other related behavioural tendencies and personality traits. Emotional overeating, as measured by psychometric questionnaires, was suggested be an expression of overall impulsivity, not exclusively linked to food intake, as well as a tendency to be overly concerned about eating (Adriaanse, de Ridder, & Evers, 2011; Bongers & Jansen, 2016).

In fact, the validity of psychometric emotional overeating scales and their hypothesised association with obesity has been criticised previously. Research has claimed that social desirability, the tendency of participants to answer questionnaires in a way that conforms to what they believe is expected from them, biases psychometric questionnaires of emotional eating. People with overweight and obesity might score higher on emotional overeating scales as they believe these behaviours might be anticipated from them. Therefore social desirability might be leading to spurious associations between emotional overeating and weight (Allison & Heshka, 1993). In one study, 868 adult participants rated their emotional overeating behaviour using the DEBQ, as well as indicating their tendency to please others and desire to avoid conflicts using The Marlow-Crowne Social Desirability Test (Allison & Heshka, 2007). In addition, 43 extra participants were recruited, of whom half were instructed to answer the DEBQ trying to create the most favourable impression of themselves possible. The other half were asked to answer the DEBQ items trying to paint a negative picture of themselves. Findings confirmed that social desirability might influence DEBQ emotional overeating scores. Participants scoring high on social desirability scored low on emotional eating, indicating that this behaviour was not seen as socially desirable in this sample of normal weight participants. Furthermore, participants aiming to present a very negative image of themselves had emotional eating scores more than twice as high as those instructed to present a very positive image of themselves, indicating this behaviour was seen as socially unfavourable

(Allison & Heshka, 2007). These findings suggest that samples from the general population may underreport their levels of emotional overeating. However, this could differ when studying emotional overeating in participants with overweight or obesity, who might resort to rating high emotional overeating tendency in order to explain their weight problems (Allison & Heshka, 1993).

In addition, psychometrically-measured emotional eating has been proposed to reflect the extent to which individuals attribute overeating behaviour to emotional distress. One previous study asked 43 female participants to come to a laboratory to take part in a taste test in which participants were asked to try to eat exactly 20g of provided snack foods (Adriaanse, Prinsen, Huberts, de Ridder, & Evers, 2016). The next day, participants returned to the laboratory and were randomly informed that they overconsumed or ate an acceptable amount. Moreover, participants were asked to retrospectively indicate their mood on the previous day, prior to the taste test, and to complete the DEBQ to indicate their tendency to emotionally overeat. Results suggested that participants who scored higher on DEBQ-measured emotional overeating were found to indicate high levels of low mood only when they were told to have overconsumed. Findings were interpreted as suggesting that individuals who score highly on psychometric measures of emotional overeating use emotional distress as an explanation for overconsumption (Adriaanse et al., 2016).

In summary, psychometric questionnaires of emotional eating have been criticised due to the lack of evidence showing that individuals who score highly on emotional eating scales consume more in response to stress in laboratory settings. Furthermore, social desirability might influence ratings of emotional overeating tendencies and the direction of these biases have been hypothesised to vary according to the weight status of participants. Finally, emotional overeating traits captured by psychometric scales might be an indicator of the tendency to attribute overeating to negative emotions retrospectively, rather than a true measure of eating in response to emotional stress.

In the defence of psychometric measures, stress inductions in the laboratory could be perceived as artificial and may not reflect the continuous daily stress individuals are exposed to in the real world. Moreover, while social desirability is likely to affect adults' reports of their own eating behaviours, it is less likely to influence child ratings or parental ratings of their children's eating behaviours. Psychometric measures are necessary to collect data in large population based cohorts. They are cheap, easy to administer and often the pragmatic choice when conducting quantitative research in

which larger sample sizes are preferable. Importantly, psychometric questionnaires allow researchers to gain a sense of the *general tendency* to over- or under-eat in response to emotion – i.e. an individual's underlying predisposition. In contrast, behavioural measures of emotional over- and under-eating observed in a laboratory might only reflect specific behaviours in response to the particular laboratory-based scenario, and on that particular day. Psychometric measures are beneficial as they enable a broader sense of the tendency towards emotional over and under-eating to be captured. In essence, the psychometric measure is capturing the underlying enduring trait, rather than a particular state at one time.

## 1.4 The importance of emotional eating for health

Emotional over- and under-eating have been of great interest to researchers because of their potential impact on overweight, underweight and overall mental health. A brief summary of the potential roles of emotional over- and under-eating in health outcomes is described in sections 1.4.1 and 1.4.2 below.

### 1.4.1 Emotional overeating

#### 1.4.1.1 Emotional overeating and mental health

##### 1.4.1.1.1 Adults

There has been a longstanding observation that obesity and depression often co-occur. A review of longitudinal studies has concluded a complex bi-directional relationship exists, by which depression and obesity interplay to influence one another over time (Luppino et al., 2010). The role of emotional overeating is implicit in this relationship in light of the fact that changes in appetite and eating behaviour are one of the symptom criteria for clinical diagnosis for depression (American Psychiatric Association, 2013). More recently, to further interrogate this association, eating in response to stress has received particular attention. Emotional overeating has been hypothesised to be a potential mediator in the complex reciprocal relationship between obesity and depression. A cross-sectional study of 3614 Finish adults found a positive association between emotional overeating and consumption of sweet foods. Furthermore, the positive association between depression and intake of sweet foods become non-significant, after controlling emotional overeating, suggesting that the fact people engage in emotional overeating when depressed explains this relationship (Konttinen, Mannisto, Sarlio-Lahteenkorva, Silventoinen, & Haukkala, 2010). More recently the associations between emotional overeating, depression and weight gain have been investigated using cross-sectional analyses of adult data

collected in Spain (n = 1409) and Denmark (n = 1396). Findings from both samples indicated that emotional overeating acted as a mediator between depression and BMI (van Strien et al., 2016). In summary, these previous studies propose emotional overeating as a key behavioural mechanism explaining the association between depression and obesity. However, the studies were cross-sectional and longitudinal research is needed to examine the direction of causation. For instance, a longitudinal analysis from the Whitehall Study in the UK found evidence that high sugar consumption in men resulted in an increased risk of developing depression five years later. Although, as emotional overeating was not included in these analyses, it is not clear what role this behaviour plays in the observed association (Knuppel, Shipley, Llewellyn, & Brunner, 2017).

Emotional overeating has also been suggested to be a crucial behaviour in Binge Eating Disorder (BED). BED is marked by reoccurring episodes of uncontrolled eating, during which large amounts of food are consumed in a short period of time (American Psychiatric Association, 2013). Previous research has found that obesity and BED often co-occur (de Zwaan, 2001) and that patients with BED tend to show elevated levels of emotional overeating (Tanofsky, Wilfley, Spurrell, Welch, & Brownell, 1997). Furthermore, emotional eating has been found to more prevalent in patients with bulimia nervosa, an eating disorders indicated by alternating periods of binge eating and purging (Wardle, 1987).

Overall previous evidence suggests that emotional overeating is a key behaviour underlying eating disorders and depression. Furthermore, emotional overeating offers a likely link for explaining the comorbidity between obesity and other mental health disorders.

#### 1.4.1.1.2 Children

In comparison to adults, less research has examined the relationship between emotional overeating and mental health in children. Adolescent emotional overeating has been suggested as a precursor of later BED. A prospective study of 231 adolescent girls (mean age = 14.9 years) suggested that emotional overeating at baseline predicted an increased risk for binge eating behaviour two years later (Stice, Presnell, & Spangler, 2002). These findings have been corroborated by reviews of cross-sectional studies suggesting that children who binge eat also have elevated levels of emotional overeating, similar to the results observed in adults (Marcus & Kalarchian, 2003; Tanofsky-Kraff, Goossens, et al., 2007).

It has also been suggested that emotional overeating is more prevalent in children and adolescents with a diagnosis of Attention Deficit Hyperactivity Disorder (ADHD). In a sample of 2414 adolescents (11-17 years), teenagers with an ADHD diagnosis (n = 101) were at increased risk of obesity and reported higher prevalence of worrying about their control over their eating (Erhart et al., 2012). ADHD is marked by lower inhibitory control and lower emotional regulation, which might lead to increased emotional overeating. More recently, a cross-sectional study of 785 Chinese children (mean age = 10.6 years) examined the associations between ADHD, bulimia, depression and emotional overeating. Findings suggested complex relationships, with ADHD related to higher levels of emotional overeating, and emotional overeating linked with increased levels of depression. However, cross-sectional data analyses cannot elucidate the direction of relationships between these different variables (Tong, Shi, & Li, 2017).

In conclusion, previous research implicates emotional overeating in several mental health disorders in childhood. Emotional overeating has been proposed to predict later eating disorders and has been indicated as a potential behavioural mechanism underlying the association between ADHD and obesity. These findings underline the need to better understand emotional eating in early life, before many of these mental health problems emerge

#### 1.4.1.2 Emotional overeating and weight

##### 1.4.1.2.1 Adults

Observational studies have sometimes found that stressful situations lead to increased caloric intake, which raises the possibility that a tendency to overeat predisposes to obesity. As an example, a study of over 45000 adults from the Finnish Public Sector Cohort Study suggested that working in highly demanding jobs, with low job control and lower pay was associated with higher BMI. Findings remained significant after accounting the effect of socio-economic status (Kouvonen, Kivimaki, Cox, Cox, & Vahtera, 2005). Emotional overeating has been suggested to be behavioural link between stress and obesity. The idea being that for some individuals high levels of stress elicit emotional overeating, in turn resulting in excess weight gain (Greeno & Wing, 1994). Evidence for this link comes from longitudinal studies. In a study of 1562 adults, measuring their emotional overeating and BMI over a two-year period, emotional overeating was the biggest predictor of weight gain (Koenders & van Strien, 2011). Similar findings were brought forward by a smaller prospective study (n = 590) of Dutch adults, showing that emotional overeating predicted weight

gain over two years (van Strien, Herman, & Verheijden, 2012). Moreover, a larger study from Switzerland (n = 3425) found that emotional overeating predicted weight gain over a period of one year (Dohle, Hartmann, & Keller, 2014). These studies provide convincing evidence that a tendency to emotionally overeat leads to greater weight gain over time. This is problematic given the current public health concern regarding the high rates of overweight and obesity, and co-morbid diseases in the Western world. Understanding the origin of this behaviour would help public health initiatives concerned with reducing 'obesogenic' behaviours.

#### 1.4.1.2.2 Children

The association between emotional overeating and weight has also been investigated in children. About half of the studies to date have found no association between emotional overeating and weight in children (Caccialanza et al., 2004; Cao et al., 2012; Jahnke & Warschburger, 2008; Jollie-Trottier, Holm, & McDonald, 2009; McCarthy et al., 2015; E. F. Sleddens et al., 2008; E. F. C. Sleddens, Kremers, De Vries, & Thijs, 2010; Snoek, Engels, van Strien, & Otten, 2013; Svensson et al., 2011; van Strien & Oosterveld, 2008; Wardle et al., 1992; Webber, Hill, Saxton, Van Jaarsveld, & Wardle, 2009). However the other half have found significant positive associations between emotional overeating and BMI, indicating that children who tend to emotionally overeat more, also tend to have a higher BMI (Braet et al., 2008; Braet & Van Strien, 1997; Domoff et al., 2015; dos Passos et al., 2015; Hajna et al., 2014; P. W. Jansen et al., 2012; Joyce & Zimmer-Gembeck, 2009; Parkinson, Drewett, Le Couteur, Adamson, & T, 2010; Sanchez, Weisstaub, Santos, Corvalan, & Uauy, 2016; Spence, Carson, Casey, & Boule, 2011; Steinsbekk & Wichstrom, 2015b; Viana et al., 2008; Webber et al., 2009). Importantly, no study has suggested a negative association between EOE and weight.

The majority of the studies of emotional eating and BMI in childhood have used cross-sectional analyses. Of special interest are the three longitudinal studies that examined the likely direction of the relationship between emotional overeating and weight (Parkinson et al., 2010; Snoek et al., 2013; Steinsbekk & Wichstrom, 2015a). As part of the Gateshead Millennium Study (UK), parents rated their children's (n = 344) EOE at 5-6 years and again at 7-8 years. BMI was also measured at both time points. Although there were no significant differences in EOE scores cross-sectionally between the low, middle and high BMI groups, EOE at 5-6 years significantly predicted increases in BMI from 5-6 years to 7-8 years. Analyses controlled for age, sex and birthweight and results were consistent with the hypothesis that EOE plays a causal role in early weight gain, such that children scoring high on emotional

overeating at baseline were found to have a larger increase in BMI over a two-year follow up (Parkinson et al., 2010). On the other hand, a similar sized study of Dutch adolescents (n=328) found no effect of emotional overeating on increases in BMI from 13 to 17 years (Snoek et al., 2013).

The third prospective study of EOE and BMI in a large sample of Norwegian children (n=760) found a significant positive association between EOE at four years and weight gain from four to eight years (Steinsbekk & Wichstrom, 2015a). This study controlled for other eating behaviours (such as Satiety responsiveness and Food responsiveness). After adjusting for the other eating behaviours, the relationship between EOE and weight was non-significant, although the prospective relationship between Food responsiveness and weight remained. This finding suggests that the relationship between EOE and weight might be mediated by Food responsiveness, such that only food responsive children are likely to emotionally overeat, and therefore to gain weight. To date these remain the only longitudinal studies examining the role of emotional eating on the development of weight in children.

In summary, there is tentative evidence that emotional overeating in childhood might lead to increased weight gain. Together, with research highlighting the potential role of childhood emotional overeating in other mental health problems, these findings underline the need to further understand how emotional overeating develops in early life.

### 1.4.2 Emotional under-eating

In comparison to emotional overeating, very few studies have examined associations between emotional under-eating and health outcomes.

#### 1.4.2.1 Emotional under-eating, mental health and weight

##### 1.4.2.1.1 Adults

Due to the lack of validated measures of emotional under-eating in adults, there is a dearth of research investigating the association between emotional under-eating and health outcomes. However, emotional under-eating is potentially an important behaviour in the development and maintenance of eating disorders. Restrictive eating patterns such as dieting have been associated with anorexia nervosa (Culbert, Racine, & Klump, 2015). One hypothetical relationship would be that a tendency to emotionally under-eat promotes the tendency to diet and restrict food intake, increasing the risk of developing anorexia nervosa. Tentative support comes from a retrospective study of 42 women diagnosed with anorexia nervosa who were asked

to recall their childhood experiences (Y. R. Kim, Heo, Kang, Song, & Treasure, 2010). In comparison to healthy controls, women with anorexia nervosa indicated that they had a tendency to emotionally under-eat during childhood. In addition, general under-eating during childhood was found to be a risk factor for adult anorexia nervosa in the 1970 British Cohort Study (Nicholls & Viner, 2009). There is therefore some tentative evidence that emotional under-eating during childhood has long-term effects on the development of adult eating disorders. Furthermore, the tendency to reduce food intake in response to stress might promote dieting behaviours, which have been associated with increased risk of eating disorders. However, in comparison to emotional overeating, very little research has focussed on the consequences of emotional under-eating and more work is needed to understand this behaviour.

#### 1.4.2.1.2 Children

In comparison to adults, the CEBQ provides a validated measure of emotional under-eating, which has enabled research into the outcomes of this behaviour. Some research has aimed to establish the link between emotional under-eating and weight in childhood. A small number of cross-sectional studies have found a significant negative association between EUE and weight (Domoff et al., 2015; P. W. Jansen et al., 2012; Viana et al., 2008) suggesting that children who tend to emotionally under-eat, tend also to be thinner. However many other cross-sectional studies found no significant relationship (Cao et al., 2012; dos Passos et al., 2015; McCarthy et al., 2015; Parkinson et al., 2010; Sanchez et al., 2016; E. F. Sleddens et al., 2008; Spence et al., 2011; Svensson et al., 2011; Webber et al., 2009). Apart from these cross-sectional studies, there has only been one longitudinal study assessing the impact of EUE on weight gain, which did not find a significant association (Parkinson et al., 2010). However, no study has found a positive association between EUE and weight.

### 1.4.3 Summary

The impact of emotional overeating has been studied in adults and children. Results suggest that emotional overeating may play a role in weight gain and obesity. Moreover emotional overeating has been suggested to be a key behaviour in BED, ADHD and depression. Less is known about the health outcomes related to emotional under-eating. Tentative evidence suggests that emotional under-eating may be associated with underweight as well as potentially playing a role in eating disorders marked by excessive dietary restriction such as anorexia nervosa.

Overall, emotional over- and under-eating are key eating behaviours potentially related to many physical and mental health outcomes. Research elucidating the development of these behaviours is essential. Understanding the aetiology of emotional eating in childhood may help identify targets for interventions aiming to prevent emotional over and under-eating early in life, before any negative health consequences emerge.

## 1.5 The aetiology of emotional eating

### 1.5.1 Emotional eating and psychological theory

Emotional overeating is a key behaviour in two prominent theories of the development of obesity. The Psychosomatic Theory (Kaplan and Kaplan 1957) proposes that individuals with obesity have not learned to successfully distinguish between arousal caused by hunger, versus negative emotion; possibly because of classical conditioning in early life. The Psychosomatic theory therefore places emphasis on the influence of early experiences, pointing towards the importance of learning and the environment. For example, parents who use food to induce a positive mood and distract from negative emotions, are proposed to teach children to engage in emotional overeating through conditioning. The hypothesis being that if food consumption regularly follows the onset of negative feelings, a classically conditioned hunger response to stress can develop because negative emotions are always paired with consumption of highly palatable food (Bruch 1964). Studies suggesting that emotional overeating behaviour is higher amongst people with overweight or obesity in comparison to healthy weight controls have been seen as evidence in favour of Psychosomatic Theory. As outlined in section 1.4 many cross-sectional and longitudinal studies have reported associations between emotional overeating and overweight and obesity in both adults and children.

In contrast, the Internal/External theory (Schachter, Goldman et al. 1968) suggests a different role for emotional overeating in the development of obesity. It proposes that the *normal* response to stress – demonstrated by healthy weight individuals – is to *decrease* food intake in stressful situations, in response to internal physiological stress cues. Obese individuals' appetites are hypothesised to be abnormal in that they are *not* affected by stress. The theory still predicts that individuals with obesity eat more than normal weight individuals during times of stress, but due to the inability to respond 'normally' to stress cues insofar as they do not down-regulate their intake (van Strien and Ouwens 2003).

The Psychosomatic Theory suggests that emotional eating might be a learned behaviour, whereas the Internal/External theory infers a more biological basis for this behaviour, although postulates no cause of the aberration (lack of appetite down-regulation) observed among obese individuals. In fact, neither of the two theories specifically address the *aetiology* of emotional eating itself. Rather, the theories focus on the role of emotional eating in the aetiology of obesity. A comprehensive theory of the development of emotional eating is in need of development. Aberrations in appetite responses to stress could be learned or inherited, and research is needed to establish the aetiology of these two different behaviours, as well as to elucidate the nature of their positive relationship commonly observed in chilhood.

### 1.5.2 Twin studies of emotional eating

Twin studies provide a powerful method for understanding the extent to which individual differences in a characteristic such as emotional eating are shaped by genes and environmental influences. Importantly, twin analyses can also provide insight into the relative importance of two different types of environmental influence – aspects of the environment that are completely shared by two twins in a pair (shared environmental effects), and environmental influences that are unique to each individual twin (non-shared environmental effects). Twin studies over the last century have revolutionised our understanding of the aetiology of some of the most important health-related human traits, including Body Mass Index (T. J. C. Polderman et al., 2015).

#### 1.5.2.1 Key assumptions of the twin method

Twin research exploits the natural occurrence of identical (monozygotic, [MZ]) and non-identical twins (dizygotic, [DZ]). MZ twins are natural genetic clones of one another, sharing 100% of their genome; whereas DZ twins share on average 50% of their segregating genes, in keeping with regular siblings. Importantly, both types of twins share their environments to a very similar extent insofar as they are gestated in the same mother at the same time, are exactly the same age, and grow up in the same family. This means that resemblance between MZ and DZ twins can be compared to estimate genetic and environmental contributions to any given measureable trait. If MZ pairs are more similar than DZs, we assume that genetic factors must be contributing to this difference, because the only real difference between the two types of twins is that MZs are twice as similar genetically (because the extent to which environmental factors are shared is equal for both types of twins).

As ‘a rule of thumb’, genetic influences can be estimated broadly by doubling the difference between the MZ and DZ correlations. The statistic derived is commonly referred to as ‘heritability’, which quantifies the proportion of trait variation attributable to genetic variation, and can be thought of as an index of the genetic effect size ranging from 0% (genes do not contribute at all to trait variation) to 100% (genes entirely explain trait variation). Environmental effects are also estimated, and separated out into those that are completely shared between siblings (those factors that contribute to their similarity), and non-shared (those that contribute to sibling differences) (Rijsdijk & Sham, 2002) .

The twin method is based on some key assumptions. In order to extrapolate findings from a twin study to the wider population, twin cohorts must be representative. Importantly as well, the ‘equal environments assumption [EEA]’ must be met. The EEA stipulates that the environmental factors contributing to variation in the trait are shared by, and affect MZ and DZ twins to the same degree. For example, if MZs are treated more similarly than DZs and this contributes to increased similarity between them on a particular trait, the EEA has been violated. Furthermore, the twins themselves or the participants (parents, teacher, doctors) rating the behaviour of the twins, must not be influenced by the twins’ zygosity. For example, if the twins themselves or other raters assume them to be identical, they might be biased in their responses, perhaps reporting the twins to be more similar than they actually are, resulting in unreliable estimates (Rijsdijk & Sham, 2002).

#### 1.5.2.2 Twin studies of adult emotional eating

There have been a few twin studies exploring the aetiology of emotional overeating in adults; an overview of these is presented in **Table 1.1**. Emotional overeating in adult twins has been measured using the adult version of the Dutch Eating behaviour Questionnaire (DEBQ) (Vanstrien, Frijters, Bergers, & Defares, 1986) and the revised version of the Three Factor Eating Questionnaire (R18) (TFEQ) (de Lauzon et al., 2004). The items in the emotional eating scales of each questionnaire are shown in **Appendix 1.1**.

A study of Swedish male twins (MZ: 456 pairs; DZ: 326 pairs) aged 23-29 years, suggested that 60% of the variation in emotional overeating, measured with the TFEQ, was explained by genetic effects. Non-shared environmental effects explained the remaining 40%, with no detectable effect of the shared environment (Tholin, Rasmussen, Tynelius, & Karlsson, 2005). A subsequent study of adult twins from the UK and Finland confirmed that the shared environment did not contribute to variation

in emotional overeating, measured with the TFEQ (Keskitalo et al., 2008). Participants were between 17 and 82 years of age (MZ: 314 pairs, DZ: 327 pairs), and estimates varied between men and women. The majority of variation was explained by non-shared environmental effects in both sexes. In women, a larger proportion of variation in emotional overeating was explained by genetic effects (45% UK, 31% Finland); while they were non-significant for men. However, there were far fewer men (n = 231) than women (n = 1095) included in the study, and estimates derived from smaller sample sizes have larger confidence intervals, making them less reliable. Due to the small sample of male participants, the confidence intervals for the genetic effects were wide, and heritability could have been as high as 47% (UK males = 0% - 47%; Finish males = 0% - 38%; the authors did not report the point estimates, only the 95% confidence intervals) (Keskitalo et al., 2008). More recently a study of adult twins (mean age: 38.1 years) from Korea (MZ: 441 pairs, DZ: 124 pairs) reported moderate genetic effects (32%), but again the majority of variation in emotional overeating was explained by non-shared environmental factors, in keeping with the other adult studies (Sung, Lee, Song, Lee, & Lee, 2010).

A recent study investigated the genetic and shared environmental contributions to variation in emotional overeating using a slightly different twin design that takes advantage of identical twins that have been raised apart (Elder et al., 2012). Comparing MZ twins who are reared together, with MZ twins reared apart provides direct information about the importance of the shared environment. MZ twins reared-apart share only their genes; whereas MZ twins reared together share both their genes and aspects of their environment. This comparison therefore makes it possible to directly estimate the contributions of genes and shared environments. Greater similarity between the MZs reared together reflects the additional shared environmental effects that do not contribute to similarity for those reared apart. Emotional eating was measured with the TFEQ in 22 MZ twin pairs raised apart (MZA) and 38 MZ twins raised together (MZT). MZAs and MZT differed significantly by age, with the MZA group being significantly older (MZA mean age = 50.7; MZT mean age = 28.7). Both types of twins were correlated for emotional overeating, but there was no difference between the two types of twins in their similarity. These results showed that genetics played a moderate role in explaining individual differences in emotional eating (55%), and that the shared environment did not contribute at all to variation in this trait, in line with the other classic twin studies (Elder et al., 2012).

In conclusion, adult twin studies examining the genetic and environmental influences on emotional overeating suggest that individual differences in emotional overeating

are partly explained by genetic variation, but the majority of differences between people are attributable to aspects of the environment unique to each person. Large twin studies have the power to calculate precise estimates, with narrow confidence intervals. However the previous studies were limited in size, producing less reliable estimates, especially when examining sex differences. Furthermore, the large age range of participants adds to the heterogeneity of the findings and limits interpretation. Genetic and environmental influences on individual differences in behavioural traits (as well as BMI) can vary profoundly with development (Bergen, Gardner, & Kendler, 2007). For example, longitudinal twin studies of BMI have shown that heritability estimates are not stable, but are age-dependent (Briley & Tucker-Drob, 2013; Haworth et al., 2008; SilventoinenJelenkovic, et al., 2016). In particular, genetic influence is lowest, and shared environmental influence is highest during early childhood, after which shared environmental influence diminishes and genetic influence increases progressively throughout adolescence and into early adulthood.

Individual differences in a range of appetitive traits, such as satiety sensitivity and responsiveness to food cues have been investigated in children using the twin method (Carnell, Haworth, Plomin, & Wardle, 2008; Llewellyn, van Jaarsveld, Johnson, Carnell, & Wardle, 2010; Smith et al., 2016). Twin studies of emotional overeating in children would help to elucidate its aetiology, but to date there have been none. There have also been no twin studies of emotional *under-eating* in either adults or children. Additionally, longitudinal twin studies of emotional eating starting early in childhood are needed to understand better the aetiology of emotional eating as it emerges and develops over childhood. Studies of other appetitive traits in children may help shed some light on the likely aetiology of emotional over- and under-eating in children. These are discussed in the following section.

**Table 1.1 Twin Studies investigating genetic and environmental contribution to EOE in adults**

| Study | Questionnaire | Sample | Age | Nationality | Estimates (95% CI) | |
|---|---|---|---|---|---|---|
| **Tholin et al (2005)** | TFEQ-R21 | MZ: 456<br>DZ: 326 | 23-29 years | Swedish | A: 60% (24, 67)<br>C: 0% (0, 37)<br>E: 40% (33, 48) | |
| **Keskitalo[1] et al (2008)** | TFEQ-R21 | MZ: 314<br>DZ: 327 | 17-82 years | UK & Finnish | Males (UK)<br>A: 0, 47%<br>C: 0%<br>E: 53, 100%<br>Females (UK)<br>A: 34, 54%<br>C: 0%<br>E: 39, 57% | Males (FL)<br>A: 0, 38%<br>C: 0%<br>E: 62, 100<br>Females (FL)<br>A: 7, 51%<br>C: 0%<br>E:49,93% |
| **Sung et al (2010)** | DEBQ | MZ: 441<br>DZ:124 | 20-65 years | Korean | A: 25%<br>C: 0%<br>E: 75% | |
| **Elder et al (2012)** | TFEQ-R21 | MZA: 22<br>MZT: 38 | 18-72 years | USA | A: 55% (11, 75)<br>C: 0% (0, 49)<br>E: 45% (23, 71) | |

Abbreviations: TFEQ-R21 = Three Factor Eating Questionnaire, MZ = Monozygotic, DZ = Dizygotic, A = latent factor, genetic effects, C = latent factor, shared-environmental effects

[1] This study did not report point estimates. Therefore only upper and lower confidence intervals were presented

#### 1.5.2.3 Twin studies of child eating behaviours

The classic twin method has been employed to estimate the heritability of a range of eating behaviours at different developmental stages in childhood. The majority of research investigating the genetic and environmental aetiology of childhood eating behaviours comes from the Gemini study. This prospective twin cohort is the biggest ever twin study specifically set up to investigate eating behaviours and appetite regulation right from the beginning of life. At baseline, 2402 families with twins born in England and Wales between March and December 2007 joined the cohort (C. H. van Jaarsveld, Johnson, Llewellyn, & Wardle, 2010). Analysing data from the Gemini study, the heritability of the BEBQ traits were assessed in 729 MZ and 1605 DZ infant twin pairs when they were approximately three months of age: heritability estimates were high for Slowness in eating and Satiety responsiveness (85% and 72% respectively), and moderate for Enjoyment of food and Food responsiveness (53% and 59%), with environmental factors *shared between twins* accounting for the majority of the remaining variance (45% and 30%) (Llewellyn, van Jaarsveld, et al., 2010). A subsequent study of Gemini twins investigated the aetiology of fussy eating in toddlerhood (16 months). Researchers divided the CEBQ Food fussiness scale into two components – 'picky eating' (being fussy and picky about familiar foods) and food neophobia (refusal to try new foods). Data from 1921 toddlers (626 MZ, 1306 DZ twins) showed that 'picky eating' was equally explained by genetic and shared environmental factors (46%), whereas food neophobia had a stronger genetic component to its aetiology although with important shared environmental influence as well (heritability = 58%, shared environment = 22%) (Smith et al., 2016). In the same cohort at 3.5 years of age, fussy eating measured using the full CEBQ Food fussiness scale was found to be largely genetically determined (heritability = 78%) (Fildes, van Jaarsveld et al. 2016); suggesting that the heritability of this trait may increase with age during the early years.

The importance of genetic factors underlying the aetiology of eating behaviours in infancy and toddlerhood were in line with previous research investigating the heritability of eating behaviours in later childhood. Data from of over 5400 twin pairs (aged 8 - 11 years) from the Twins Early Development Study (TEDS) were analysed. TEDS is one of the largest twin birth cohorts in the world including over 15000 twin pairs born in the UK in 1994 - 1996. Enjoyment of food and Satiety responsiveness were measured with the CEBQ and results suggested high heritability for both traits (75% and 63%), with shared environmental factors playing only a minor role (16% and 21%) (Carnell et al., 2008).

Overall, previous twin studies have investigated the heritability of many child eating behaviours, but no study has established the heritability of child emotional over- and under-eating. Furthermore, complex twin models can be applied to understand the association between child emotional over- and under-eating. Apart from simple heritability estimates, bivariate twin models enable researchers to dissect the associations between two separate but correlated behaviours and determine whether the association is driven by genetic or environmental influences common to the two behaviours. Previously, this approach was used to understand the correlation between food neophobia and food fussiness analysing data from the Gemini study (Smith et al., 2016). This study found common genetic factors were most important in explaining the strong phenotypic correlation observed between the two behaviours. The same method can be applied to emotional over- and under-eating, which have been found to positively correlate as described in **1.3.2.3**.

Despite the potential insights offered by the twin method, no childhood twin study has aimed to estimate the heritability of emotional over- or under-eating. This gap in the literature is surprising given that emotional eating has been at the centre of obesity research for many years.

While twin studies only produce broad indications of the relative contribution of genetic and environmental factors underlying individual differences, previous studies have aimed to identify specific genetic and environmental factors associated with emotional eating in childhood. Key findings are discussed in the following sections.

### 1.5.3 Specific genetic and environmental influences on emotional eating

#### 1.5.3.1 Specific genetic influences (e.g. BMI-SNPs)

In addition to twin research, recent molecular genetic work with adults has identified some specific common genetic variants associated with emotional overeating. These are the same variants as those influencing BMI, and were examined after they were discovered first in relation to BMI. In fact, studies have shown that emotional overeating mediates some of the well-established association between genetic risk for obesity (indexed using a composite score of the 97 obesity-associated genetic variants) and BMI – i.e. emotional overeating is a behavioural mediator of genetic risk of obesity. These studies are summarised below.

The genetic aetiology of BMI has received substantial attention since the completion of the human genome project in 2000 (Collins & McKusick, 2001). Genome-wide association studies (GWAS) have enabled the detection of common genetic variants

(in the form of single nucleotide polymorphisms, SNPs) associated with a phenotype. For BMI, 97 SNPs have been identified; in accumulation accounting for about 2.7% of the variance in adult BMI (Locke et al., 2015). These genetic variants have been studied in the context of eating behaviours, with the hypothesis that eating behaviours are behavioural expressions of, and mediators of genetic risk of obesity. An earlier study of 3852 US adults included genetic data of 32 obesity risk loci, aggregated to a genetic risk score. Emotional eating, cognitive restraint and uncontrolled eating were measured with the Three Factor Eating Questionnaire (TFEQ-18) (Cornelis et al., 2014). The genetic risk score for obesity was positively associated with Emotional eating and Uncontrolled eating, providing evidence that genetic risk for obesity also predisposes to emotional overeating in adults.

A following study took advantage of newly discovered genetic loci associated with obesity, including 90 associated SNPs that were aggregated into a genetic risk score for obesity. Eating behaviours of 5863 adults were measured with the TFEQ-18. Results confirmed previous findings, showing that the associations between the SNPs and BMI were mediated by Emotional overeating; providing more evidence for the role of Emotional overeating in the development of obesity (Konttinen et al., 2015).

Subsequent studies aimed to investigate the association between genetic risk of obesity and emotional overeating in children. A smaller study (n = 632) of Norwegian children, investigated the effect of genetic risk for obesity, based on 32 associated SNPs aggregated into a genetic risk score, on child weight gain longitudinally when the children were four, six and eight years old (Steinsbekk, Belsky, Guzey, Wardle, & Wichstrom, 2016). Results confirmed that children at greater genetic risk of obesity had elevated levels of EOE, but they did not find significant mediation of EOE on *weight gain* longitudinally. However, large samples are needed to detect the small effects of genetic risk scores, especially if the included number of SNPs is small. In this study the small sample (n = 632) *and* the smaller number of SNPs (32) may have reduced the power to detect an effect. In addition, there is less variation in *weight gain* than in weight at any one time, further limiting power to detect an effect.

In summary, discussed research indicates that emotional overeating mediates the genetic risk for obesity in adults. This effect is less clear in children and more studies with larger sample sizes are needed.

### 1.5.3.2 Specific individual and environmental influences on emotional eating

#### 1.5.3.2.1 Emotion Regulation

It has been suggested that the ability to regulate one's emotions plays a role in the likelihood that an individual will develop a tendency to emotionally overeat. A previous study, compared children experiencing loss of control eating behaviours with children rated to be in control of their eating (n = 60, 8-13 years old) (Czaja, Rief, & Hilbert, 2009). Loss of control eating behaviour was determined by interview. Group comparison highlighted children reporting loss of control eating showed higher use of dysfunctional emotion regulation behaviours such as becoming aggressive or withdrawing from the situation entirely (Czaja et al., 2009).

Recently, a cross-sectional study investigated the relationship between emotion regulation, emotional overeating and intake of energy rich foods in a large sample of Chinese teenagers (n = 4316). Findings suggested that suppression of emotions is associated with greater emotional overeating; and emotional overeating mediated the relationship between emotion regulation and intake of energy dense food (Lu, Tao, Hou, Zhang, & Ren, 2016). However, the cross-sectional nature of the data precludes an inferences about the direction of these relationships.

In addition to cross-sectional research, a small preliminary study investigated the effect of emotion regulation training on Binge Eating Disorder symptoms in adults. As discussed in **Chapter 1.4.1** emotional overeating has been suggested as key behaviour in adults with BED. Throughout 11 sessions, patients with a BED diagnosis practised emotion regulation strategies as well as stress management and relaxation (n = 11). Three months after the intervention participants reported a decrease in binge eating behaviours (Clyne & Blampied, 2004). However, this preliminary intervention was small and replication including a control group is necessary.

In addition, a child's ability to regulate their emotions has also been suggested as a mediating factor between parental feeding behaviour and child emotional overeating – the hypothesis being that a child who struggles to regulate their emotions is more likely to be offered food to soothe them by their parents who are in need of strategies to calm their child down. A study of 254 families with four year old children included measures of child emotional overeating, child emotional regulation and parental use of food as a reward. Cross-sectional mediation analyses suggested that the relationship between parental use of food as a reward and child emotional overeating was partially mediated by child emotion regulation (Powell, Frankel, & Hernandez, 2017).This study indicates a complex relationship between child emotional regulation

ability, parental feeding behaviours and child emotional overeating. However to fully understand the direction of causation longitudinal studies are needed.

In summary these findings suggest that the ability to self–regulate emotions might be a crucial factor in the development of emotional overeating. However, no previous study has investigated the relationship between emotional regulation and emotional under-eating specifically in either children or adults.

#### 1.5.3.2.2 Parent level factors

Parents are deemed to be essential for the shaping of early eating behaviours. Parents might influence their children's eating behaviours and weight development through two main mechanisms; (i) intergenerational transmission whereby children inherit genes from their parents that influence eating behaviours (and weight) and (ii) shaping their child's eating behaviour through their own behaviour. The impact of parental feeding practices on child eating behaviour has received considerable attention in the literature (Mitchell, Farrow, Haycraft, & Meyer, 2013). Broadly speaking, parents or other caregivers act as providers who expose their children to flavours and foods, regulate the timing and amount of food consumed, and encourage them to develop appropriate eating behaviours. Moreover parents and caregivers can also act as role models, modelling eating behaviours themselves which children learn to emulate (Savage, Fisher, & Birch, 2007).

#### 1.5.3.2.3 Parental feeding

Research has investigated the impact of parental feeding practices on the development of childhood emotional overeating.

Many different psychometric questionnaires have been developed to quantify parental feeding behaviour. One of the first and most commonly used tools is the Child Feeding Questionnaire (Birch et al., 2001). Apart from parental concerns about their child's weight, this questionnaire asks parents to indicate their feeding practices which cluster in three separate behaviours: Pressure to eat, Restriction and Monitoring. Subsequent questionnaires, extended these dimensions, adding Instrumental feeding, the tendency to use food as a reward for good behaviours, as well as Emotional feeding, the tendency to use food to soothe and distract from negative emotions. These feeding practices, including the ones proposed by Birch et al (2001) are included in the Parental Feeding Style Questionnaire (PFSQ) (Wardle, Sanderson, Guthrie, Rapoport, & Plomin, 2002) and the Comprehensive Feeding Practices Questionnaire (CFPQ) (Musher-Eizenman & Holub, 2007). In addition others have emphasised the importance of structure and rules during mealtimes (E.

Jansen, Mallan, Nicholson, & Daniels, 2014). While many different questionnaires have been devised, clustering in different and often overlapping feeding practices, parental emotional feeding has received the most attention in relation to emotional overeating.

Emotional feeding has been cross-sectionally associated with child emotional overeating in a sample of 108 (USA) mothers and their children, aged eight to 12 years. After controlling for age and sex, parental emotional feeding remained the strongest predictor associated with child emotional overeating (Braden et al., 2014). Similar results have been reported from another cross-sectional study of parents and their 4 - 9 year old children (n = 95, USA). Results suggested that emotional feeding acts as a mediator between parental emotional overeating and child emotional overeating, indicating that parents who engage in emotional overeating themselves might be more likely to use emotional feeding strategies, thereby eliciting emotional overeating in their children (Tan & Holub, 2015). Similarly, a study of 306 Australian mothers and their two year-old children indicated that the association between maternal emotional overeating and child emotional overeating is mostly explained by emotional feeding practices, with instrumental feeding playing a minor role (Rodgers et al., 2014). More recently a similar positive association between emotional feeding and instrumental feeding, and child EOE was reported in a sample of 1201 primary school children in Turkey (Demir & Bektas, 2017).

These previous studies imply a possible relationship between parental feeding and child emotional eating behaviour. However, cross-sectional studies cannot elucidate the causal direction of this relationship. This is important as it is plausible that parents may adopt feeding practices merely as a response to their children's' eating behaviour. For example, parents may simply be more likely to emotionally feed a child who has a tendency to emotionally overeat in the first place. Therefore longitudinal studies with repeated measures of child eating and parental feeding are essential to disentangle this complex relationship.

***Reciprocity in child eating and parental feeding***

Some of the most important quantitative studies in the area of child eating behaviour and parental feeding have been conducted as part of the Trondheim Early Secure Study (TESS). TESS is a longitudinal cohort in Trondheim, Norway, that has been collecting data from over 800 families on child eating behaviours and parental feeding behaviours from early to late childhood on a biennial basis. Recently two longitudinal

analyses from TESS have contributed greatly to our understanding of the association between child emotional overeating and parental feeding.

The first of these longitudinal studies suggested that parental instrumental feeding (using food as a reward), when children were six years old predicted greater increases in child EOE over a two year period, but the reverse association was not observed. This suggests that parental instrumental feeding results in greater child emotional overeating behaviour (Steinsbekk, Belsky, & Wichstrom, 2016). More recently, the analyses were extended to include data when the children were six, eight and ten years old. Importantly, in addition, these analyses included child negative affect, hypothesising that the child's temperament influences child emotional overeating as well as parental emotional feeding. Results supported the notion that negative affect influences both child emotional eating and parental emotional feeding. Bidirectional associations were found between parental emotional feeding and child EOE, between the ages six and eight, and between eight and 10 years (Steinsbekk, Barker, Llewellyn, Fildes, & Wichstrom, 2017). In summary, these results suggest a complex reciprocal relationship between parental feeding and child EOE, especially in later childhood.

Research from other groups also provide support for a reciprocal relationship between parental emotional feeding and child emotional overeating. A prospective study of 323 Australian mothers with two year-old children found evidence that parental emotional feeding predicted child EOE one year later. Again the relationship was found to potentially be bi-directional, with higher child EOE at two years also predicting increases in parental emotional feeding one year later (Rodgers et al., 2013).

These findings regarding child emotional overeating are in line with other research testing the reciprocal relationship between parental feeding, child eating and child weight. Longitudinal research from the Generation R cohort (n > 4000 families) in the Netherlands has found evidence for bi-directional associations between parental restriction and higher child BMI, and between pressure to eat, and lower child BMI in preschool aged children (two years and six years). Results imply that parents not only shape but also respond to their child's weight by adjusting their feeding behaviour accordingly, although sometimes to the detriment of the child (P. W. Jansen et al., 2014). Analysing the same sample, a study has also reported a similar bi-directional relationship between child fussy eating and greater parental pressure to eat, measured when children were 1.5, three and six years old. Child food fussiness was

found to be the cause as well as the consequence of parental pressure to eat, highlighting the complexity of parent child interactions (P. W. Jansen et al., 2017). Overall results support the idea of complex interplay between children and parents in the development of eating and feeding behaviour in early life.

There has been no longitudinal study of parental feeding and its impact on child emotional under-eating. In one of the only studies to report a cross-sectional association between parent feeding and child EUE, similar to EOE, a positive correlation was found between emotional feeding, instrumental feeding and child EUE in the sample of 1201 Turkish children. Results suggest that just like emotional overeating, parental emotional feeding might be play a role in the development of emotional under-eating (Demir & Bektas, 2017).

To date only one study (Steinsbekk et al., 2017) has tested this specific bi-directional association specifically in regards to emotional feeding and emotional overeating, and more research is needed to enhance understanding of these relationships across other ages and in different samples. Research on the impact of parental feeding on child emotional under-eating is lacking, with only one cross-sectional study specifically investigating the impact of parenting on emotional under-eating in childhood (Demir & Bektas, 2017). Moreover there is only one study examining emotional overeating in very early childhood (toddlerhood) when it first starts to emerge (Rodgers et al., 2013). However the sample size was moderate and the time between first measurement and follow-up was short (one year), precluding any inferences regarding the longer-term effects of emotional feeding in early childhood on later emotional overeating or vice versa.

***Studies using laboratory based measures of emotional eating***

In addition to observational studies, laboratory-based studies have aimed to understand how parental feeding practices influence objectively measured food intake of children in response to experimentally-induced stress. In one such study a sample of 25 children (aged three to five years) were allocated to either a control or negative mood condition (Blissett et al., 2010). In the negative mood condition children were presented with a jigsaw puzzle that could not be solved to induce a negative emotional state. Afterwards children were presented with a variety of snack foods (salted crisps, chocolate chip cookies, chocolate buttons, green grapes, carrot sticks and, bread sticks) and researchers observed the amount eaten by every child. Maternal feeding practices were also measured. Results showed there were no significant differences in the amount of food consumed by the control and negative

emotion group. However across groups, children of parents who reported engaging in emotional feeding practices, ate significantly more snack foods overall (Blissett et al., 2010). The lack of group difference in food consumption might be seen as an indication that the mood manipulation was not sufficient to elicit emotional overeating. Findings certainly suggested that parental emotional feeding might be essential to the development of child emotional overeating.

This sample was followed up, and the experiment repeated two years later (Farrow, Haycraft & Blissett, 2015). In the repeated experiment the mood manipulation was refined to guarantee greater changes in mood in the experimental group. Children were asked to colour in a picture, with different sections of the image numbered to correspond with a colour (e.g. number one stands for red). On completion of the task, children were promised that they would be allowed to play with their chosen toy available in the research laboratory. In the experimental group the final coloured crayon was not available, leaving the children unable to complete the task and receive the reward, whereas in the control group the children were handed all the colours without delay. In line with the previous study (Blissett et al., 2010) children were presented with a variety of highly palatable snack foods (e.g. biscuits, crisps etc.) after the mood manipulation. After four minutes, children in the experimental group were handed the missing crayon and were allowed to finish the task and receive the reward. In contrast to the previous study, children in the experimental group consumed significantly more calories than the control group, highlighting that a negative mood may indeed increase consumption of highly palatable foods. Furthermore this increase might also indicate that the tendency to eat in response to stress increases with age. Parental feeding styles, collected two years prior to this experiment, indicated that children in the experimental condition whose mothers reported using food as a reward and constraining their child's food intake for health reasons, consumed even more when faced with stress and disappointment. In contrast to the first study, no effect of parental emotional feeding on child emotional eating was found two years later (Farrow et al., 2015).

***Summary of parental feeding practices and child emotional eating***

Evidence from observational and laboratory based studies support a complex bi-directional relationship between parental feeding and child eating, underlying the development of emotional overeating in childhood. In particular, parental emotional feeding (using food to soothe) and instrumental feeding (using food as a reward) have been associated with greater emotional overeating in childhood. So far the literature

consists mostly of smaller studies, with only one larger study analysing data from a prospective cohort (Steinsbekk et al., 2017). This study suggested a bi-directional association between child EOE and parental emotional feeding in later childhood, but more research is needed to replicate this finding. Moreover, Steinsbekk and colleagues focus on middle (aged six) to later (aged 10) childhood. However, EOE emerges earlier in life (Ashcroft et al., 2008; Cao et al., 2012; Mallan et al., 2013) and future research would benefit from exploring the relationship between parent feeding and child emotional overeating from as early as possible when these behaviours first start to emerge.

There is a lack of research trying to understand the role of parental feeding styles in emotional under-eating in childhood. Tentative observations come from one cross-sectional study, also linking EUE with emotional and instrumental parental feeding (Demir & Bektas, 2017), but replication is needed, as well as longitudinal research to test the direction of the relationship between parental feeding and child emotional under-eating. Emotional over- and under-eating have been found to correlate but the reasons for this correlation remain unclear (see **Chapter 1.3.2.3**). Therefore research is needed to identify parental feeding practices that are exclusively associated with emotional over and under-eating, or shared between the two.

#### 1.5.3.2.4 Early feeding environment

The early life feeding environment could also be a potential influence on the development of child emotional over- and under-eating. A child's very first experiences of eating and feeding are during the milk-feeding phase. Even during this early period parents have distinct styles and philosophies that often govern how they feed their infant. In particular, two important and different feeding philosophies are schedule feeding or feeding on demand. Schedule feeding refers to the mother enforcing strict times when the child is fed. In contrast, responsive feeding (or 'demand' feeding) is more flexible, involving a child being fed whenever they are deemed hungry, usually signalled by crying (and other behavioural cues). The latter has been suggested as advantageous, as long as the mother truly responds to the baby's hunger and stops feeding when he or she is full (DiSantis, Hodges, Johnson, & Fisher, 2011). In contrast, schedule feeding potentially ignores the baby's hunger and therefore might disrupt the development of appetite regulation, potentially resulting in increased child weight (Hurley, Cross, & Hughes, 2011). This effect could potentially extent to emotional over- and under-eating, as disturbed appetite regulation might lead to greater appetite changes in response to stress.

Apart from the use of responsive feeding versus scheduled feeding, the feeding mode has also been of great interest to researchers. In comparison to bottle feeding, breastfed infants are more 'in charge' of the feeding interaction and are better able to stop of their own accord once they are sated. Studies have also shown that infants consume fewer calories from the breast than from a bottle, and have better appetite regulation in childhood indicated by higher satiety sensitivity (DiSantis, Collins, Fisher, & Davey, 2011; Hassiotou & Geddes, 2014). Therefore it is possible that breastfeeding (or bottle feeding) influences the development of child emotional over and under-eating as well. Breast fed babies might develop stronger appetite regulation which might make them less likely to under or over-eat in response to stress. So far no research has aimed to address these questions.

#### 1.5.3.2.5 Parental eating behaviour

Apart from actively shaping their children's eating behaviours, parents might also influence their children by acting as a role model. A few cross-sectional studies have investigated the associations between parental emotional eating behaviours and child emotional overeating. One study analysed the emotional overeating of adolescents (n = 475) aged 15 - 18 years and their parents. Emotional overeating was positively correlated between fathers and sons, mothers and sons, and mothers and daughters (de Lauzon-Guillain et al., 2009). In younger children, a smaller study (n = 142) confirmed the correlation between maternal emotional overeating and child emotional overeating (3 - 6 years) (Jahnke & Warschburger, 2008). However, both studies were cross-sectional and longitudinal studies are needed to disentangle the direction of the relationship between child and parental emotional overeating. The familial resemblance described in the studies could also be influenced by genetic confounding, as parents not only model the behaviour, but also pass on potentially associated genes. So far no research has been conducted to examine the association between parental eating behaviour and child emotional under-eating.

#### 1.5.3.2.6 General parenting styles and maternal mental health

Other more general parenting styles have also been associated with emotional overeating in childhood. A study of 428 adolescents and their families indicated that teenagers experiencing low maternal support but high psychological control were found to engage more in emotional overeating (Snoek, Engels, Janssens, & van Strien, 2007). Maternal psychopathology, such as anxiety, depression and overall stress, have been associated with maternal emotional feeding, which in turn is hypothesised to encourage child emotional overeating (Rodgers et al., 2014; Vandewalle, Moens, & Braet, 2014). Similarly, a study of 116 UK mothers with

preadolescent children showed that maternal attachment anxiety was associated with maternal emotional feeding behaviour, which predicted child emotional overeating (Hardman, Christiansen, & Wilkinson, 2016).

These studies tentatively suggested that the mother-child relationship, and maternal anxiety and depression are important in the development of child emotional overeating through increasing the tendency for mothers to engage in maladaptive emotional feeding. Moreover, maternal mental health problems might impact a child's ability to develop good self-regulation themselves when faced with negative emotion. However, research has only been cross-sectional, precluding conclusions about the likely direction of relationships; and sample sizes have been small, limiting reliability and generalisability. Additionally, studies have focussed exclusively on school-aged children. Yet variation in eating (or feeding) behaviour is measurable from early life (Llewellyn et al., 2011; Thompson et al., 2009). Future longitudinal studies investigating these relationships from early in childhood and beyond would help to elucidate the relationship between parenting and children's tendency to emotionally eat from the earliest time that these tendencies start to emerge (DiSantis, Hodges, et al., 2011).

#### 1.5.3.2.7 Marital conflict

More recently a study tested the effect of marital conflict on child eating behaviours. 95 families (USA) were included in the study, and parents rated the emotional overeating tendencies of their children (5 to 12 years). In turn children rated the marital conflict they witnessed within their families. Results showed that high marital conflict was associated with maladaptive child eating behaviours, including increased child emotional overeating. Greater child emotional insecurity was also associated with greater child emotional overeating (Bi, Haak, Gilbert, & Keller, 2017).

Again there is limited research focussing on the effect of other parental factors on the development of child emotional under-eating. One observational study investigated the effect of the quality of relationship between two parents in a family on their child's eating behaviour. In a sample of 168 mothers and their 3.5 year old children, researchers found that child emotional under-eating was associated with decreased warmth in the mother-father relationship and increased hostility expressed between the parents (Haycraft & Blissett, 2010). More research is needed to replicate this finding and uncover other parental predictors of child emotional under-eating.

#### 1.5.3.3 Stressful home environments and external factors

Emotional overeating and under-eating by definition can only occur in response to stressful and negative situations. Apart from the potential stressors discussed above, such as maternal mental health, other more general stress factors are likely to impact child emotional over and under-eating. Exposure to stress in childhood has been considered a risk factor for paediatric obesity, and it is possible that emotional overeating might be the mediating causal link between stress and weight gain (S. M. Wilson & Sato, 2014). Therefore, it is important to test for the impact of general family life stressors, such as growing up in a single caregiver household or low household income, on child emotional eating.

As children grow up and spend more and more time outside the family home, stress experienced within peer-groups becomes more important. High levels of childhood stress outside the home, such as problems with friends, have been associated with school-aged children's emotional overeating (n = 437, aged 5 - 12 years), as well as increased consumption of sweet and fatty foods (Michels et al., 2012). An Australian study of 194 pre-schoolers (3 - 4 years), investigated the associations between child BMI and self-reported child peer problems (Mallan, Daniels, & Nicholson, 2017). Results suggested a significant association between child BMI and peer problems. However this association was fully mediated by food approach eating behaviours, namely Food responsiveness and EOE. These results indicate how emotional overeating can result in negative health outcomes, when used as an emotion regulation strategy in daily life (Mallan et al., 2017). These previous studies have focussed on childhood stress outside the family home. More research is needed to investigate how stressful home environments impact on child emotional over- and under-eating.

## 1.6 Summary

Emotional overeating and emotional under-eating are common, with large proportions of the population engaging in one or both behaviours. Emotional overeating has been of particular interest to behavioural scientists, due to its impact on health. Emotional overeating has been implicated in weight gain and obesity, as well as other mental health problems such as BED, ADHD and depression. When it comes to emotional under-eating less is known about effects on health outcomes. There are tentative results proposing that emotional under-eating is associated with underweight in childhood, and it has been hypothesised to be a precursor of restrictive eating and therefore a risk factor for anorexia nervosa. Both emotional over- and under-eating

emerge in childhood and more research is needed to understand their development in early life. Surprisingly, emotional over- and under-eating have been found to positively correlate, proposing a potential shared aetiology. However, the nature of this association is unknown.

Previous research has aimed to investigate why and how emotional eating behaviour develops in childhood, but the majority of the literature has focussed on emotional overeating. Child characteristics such as emotion regulation ability have been suggested to be important. Additionally parental factors, such as feeding practices have been examined. Emotional feeding has emerged as a key parental driver of emotional overeating, and there is tentative evidence that this feeding behaviour relates to emotional under-eating as well. In addition to parental feeding practices, parents engaging in emotional overeating themselves was found to be associated with child emotional overeating, as well as parental emotional feeding. Very little research has specifically investigated the aetiology of emotional under-eating.

Twin studies provide an excellent framework to elucidate the aetiology of individual differences in behavioural traits. Three twin studies have estimated the heritability of adult emotional overeating, suggesting small to moderate effects of genes, with the majority of variance explained by environmental factors specific to the individuals. To date, there have been no twin studies of childhood emotional overeating. There have also been no twin studies of emotional under-eating in either adulthood or childhood. Overall, the aetiology of emotional over- and under-eating in childhood remains relatively unknown.

# Chapter 2 Research aims

## 2.1 Aims and outline of the research in the current thesis

The first chapter of this thesis summarised previous research investigating the aetiology of emotional eating in adulthood and childhood. Twin studies were introduced as a powerful design for examining the genetic and environmental influences on the aetiology of characteristics such as emotional eating. Previous twin research has established that many eating behaviours have a strong genetic basis in infancy and childhood, but no twin studies have investigated the aetiology of emotional over- and under-eating in childhood, leaving an important gap in the literature. Research has also suggested that emotional over- and under-eating co-occur in childhood insofar as they tend to correlate positively, suggesting some shared aetiology. However, the nature of their relationship is not well understood, and no study to date has investigated the extent of their common aetiology, or whether common aetiological factors can explain this association. Some research has aimed to understand the aetiology of emotional overeating, suggesting that child level factors such as emotion regulation might play a key role. In addition, parental feeding practices have been implicated in the development of childhood emotional overeating. However, virtually nothing is known about the childhood correlates of emotional under-eating.

In general, studies of the aetiology of emotional eating have tended to be small, limiting the reliability and generalisability of findings, and there have been few prospective studies of the development of emotional eating. Large prospective studies are needed to establish the likely causal shapers of emotional eating as it emerges in childhood; studies of these behaviours in very early childhood, as soon as they start to emerge, would provide the most insight.

The dearth of research into the aetiology of emotional eating comes as a surprise given that emotional overeating has been at the centre of obesity research since the second half of the 20$^{th}$ Century, and its implication in the development of several mental and physical health problems. This thesis aims to fill this gap, and provide a detailed investigation into the aetiology of emotional over- and under-eating in a large sample of young British twin children, using a range of epidemiological and behavioural genetic approaches. In addition, this PhD includes a replication of some of the findings in an independent sample, and examines the extent of twin-specific parental rating bias in the measure of emotional over- and under-eating used in this

research; the Child Eating Behaviour Questionnaire. Replication and interrogation of measures are the cornerstones of rigorous and thorough scientific practice, and ensure that science is reliable and reproducible; these approaches were therefore undertaken alongside novel research (Munafò et al., 2017).

Specifically, the following five aims were addressed in the thesis, in seven studies:

**Aim 1:** Use a twin design to establish the genetic and environmental contributions to individual differences in emotional over- and under-eating in toddlerhood and middle childhood

*Study 1* (**Chapter 4**) estimated the genetic and environmental contribution to individual differences in emotional overeating in toddlerhood (16 months) and middle childhood (five years). Furthermore, the longitudinal association of emotional overeating was decomposed into genetic and environmental factors.

**Aim 2:** Use a twin design to establish the extent of common genetic and environmental influences underlying both emotional under- and overeating, and the extent to which common influences explain their positive association

*Study 2* (**Chapter 5**) used a bivariate twin design to establish the genetic and environmental contributions to individual differences in emotional under-eating, and established the extent of common aetiology underlying emotional over- and under-eating in middle childhood.

**Aim 3:** Characterise the early life correlates and shapers of childhood emotional over- and under-eating

*Study 3* (**Chapter 6**) consisted of cross-sectional analyses of data collected when the children were five years old and aimed to identify child, parental and home environmental factors associated with *both* emotional over- and under-eating as well discovering factors relating specifically to each.

*Study 4* (**Chapter 7**) used a longitudinal approach investigating the direction of causation between parental emotional feeding and child emotional overeating.

**Aim 4:** Use a twin design to test if the aetiology of emotional over- and under-eating varies by level of environmental stress (gene-environment interaction)

*Study 5* (**Chapter 8**) used a continuous moderator twin design to test if the aetiology of emotional over- and under-eating changes with increasing household stress.

**Aim 5:** Replicate the twin study findings in an independent sample, and test for twin-specific parental bias in parents' reports of child eating behaviours

*Study 6* (**Chapter 9**) was a replication of Study 2, estimating the genetic and environmental contribution to individual differences in child emotional over- and under-eating in an independent sample.

*Study 7* (**Chapter 10**) tested if parent-rated questionnaires of child eating behaviours can be used reliably in twin research. Parents' ratings are potentially influenced by their knowledge of their twins' zygosity. Study 7 examined the presence of such bias by comparing the ratings of twin pairs whose zygosity status was correctly identified or misclassified by their parents.

## 2.2 My contributions to the research included in this thesis

I played a key role in developing the aims of this thesis and the design of the studies, together with my supervisors Professor Jane Wardle (who sadly past away at the end of the first year of my PhD), Dr Clare Llewellyn (my primary supervisor) and Dr Alison Fildes (my second supervisor). Dr Frühling Rijsdijk, based at the Social, Genetic and Developmental Psychiatry Centre, King's College London, is an expert in twin methodology and assisted with twin analyses as an external supervisor.

The majority of studies conducted in this thesis analyse data previously collected as part of the Gemini cohort. The study started in 2007 and I was not part of the initial recruitment process or data collection. However, throughout my PhD I have been heavily involved in running the cohort and have taken over a number of administrative tasks, including answering queries from families, managing and updating the contact database, and collecting and entering height and weight data submitted by the parents every three months.

I designed and ran all analyses conducted for this thesis. To do so, I undertook extensive training in statistics, especially in the analyses of twin data using maximum likelihood structural equation modelling.

# Chapter 3 Methods

## 3.1 The Gemini twin cohort - Overview

The Gemini cohort study was set up by Professor Jane Wardle at the Health Behaviour Research Centre, Department of Epidemiology and Public Health, University College London, in 2007. Its main aims are to: (1) investigate the genetic and environmental influences on weight gain and eating behaviour in childhood, (2) identify modifiable risk factors for excessive early weight gain, and (3) establish a database of early developmental exposures to assess the contributors to long-term health (C. H. van Jaarsveld et al., 2010). All studies presented in this thesis analyse existing Gemini data, including baseline measures and data collected when the twins were 16 months and five years old.

### 3.1.1 Recruitment, description and representativeness of the Gemini sample

In January 2008 all families (N = 6754) with twins born between March and December 2007 in England and Wales were invited to enrol in the study by the Office of National Statistics. Half of the families (n = 3425, 51%) agreed to be contacted by the research team. Between February and April 2008 consent forms and baseline questionnaires were sent out to these families and 2402 (36%) completed and returned the baseline questionnaire. At baseline, one third of twin pairs were male (n = 785, 32.7%), one third were female (n = 801, 33.3%) and one third were of opposite sex (n = 816, 34.0%). The sample included families with twins in England and Wales. Families were fairly equally distributed across the country, as seen in **Figure 3.1**. Response rates to initial contact, ranged slightly by region of residence, with lowest response rates in London in contrast to high response rates in the South East, East of England, East Midlands and South West ($\chi^2 = 241.261$, $p < 0.01$) (C. H. van Jaarsveld et al., 2010).

Since its initiation the Gemini study has collected data on child weight, eating behaviours, parental feeding practices and other home environmental factors at multiple time-points, primarily using parent-report questionnaires. A schematic overview of the Gemini data collection phases is shown in **Table 3.1**. This thesis will focus on questionnaire-based measures of: eating behaviours (the Baby Eating Behaviour Questionnaire (BEBQ); the Child Eating Behaviour Questionnaire (CEBQ), and its version for toddlers, the Child Eating Behaviour Questionnaire Toddler version (CEBQ-T)); child emotion regulation (Strengths and Difficulties Questionnaire (SDQ)), parental feeding practices (Parental Feeding Style Questionnaire (PFSQ), The Feeding Practices and Structure Questionnaire (FPSQ), Child Feeding Questionnaire

(CFQ)); stress in the home environment (Confusion, Hubbub and Order Scale (CHAOS)); and other sociodemographic and family characteristics (maternal education, employment and relationship status, socio-economic status, breastfeeding), as well as anthropometric data of the twins. All measures are described in detail in this chapter.

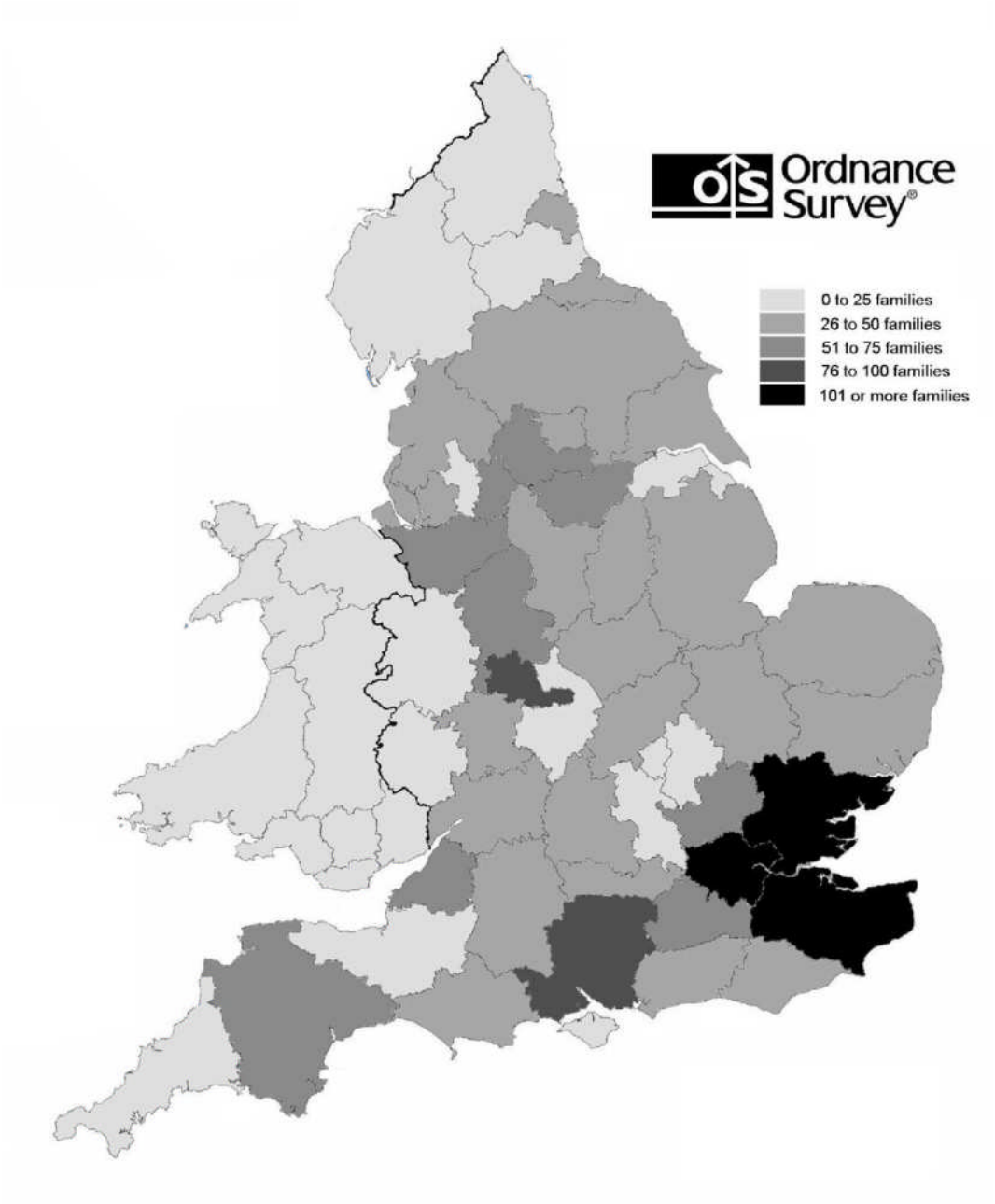


**Figure 3.1 Map of England and Wales showing the distribution of families participating in Gemini (taken from Jaarsveld et al 2010)**

**Table 3.1 Schematic overview of the assessment points and measures collected in Gemini and used in this thesis**

| Assessment | Twin Age<br>Data collection period<br>Response rate of families n [% of baseline] | 0-1 years<br>2007-2008<br>2402 (100%) | 1-2 years<br>2008-2009<br>1930 (80%) | 2-3 years<br>2009-2010<br>1364 (57%) | 5-6 years<br>2012-2013<br>1087 (45%) |
|---|---|---|---|---|---|
| **Child characteristics** | Birth weight | X | | | |
| | Anthropometrics (height and weight[1]) | X | X | X | X |
| | DNA | | X | | |
| | Emotional regulation (SDQ) | | | | |
| **Socio-demographics** | Parental education | X | | | |
| | Parental ethnicity | X | | | |
| | Parental employment | X | X | | |
| | Parental relationship status | X | X | | |
| **Home environment** | Stress in the home (CHAOS) | | | | X |
| **Parental feeding** | Parental feeding practices | X | X | X | X |
| | Milk feeding | X | | | |
| **Eating behaviour** | DEBQ (maternal eating behaviour) | | X | | |
| | BEBQ | X | | | |
| | CEBQ-T | | X | | |
| | CEBQ | | | | X |

SDQ = Strength and Difficulties Questionnaire; CHAOS = Confusion, Hubbub and Order Scale; DEBQ = Dutch Eating Behaviour Questionnaire, BEBQ = Baby Eating Behaviour Questionnaire; CEBQ-T = Child Eating Behaviour Questionnaire-Toddler; CEBQ = Child Eating Behaviour Questionnaire

[1] Height and weight data have been collected every three months since 2009, when the twins were approximately two years old

Compared with national twin statistics, Gemini twins are representative regarding sex, zygosity, gestational age and birth weight (see **Table 3.2**). However, compared to population statistics Gemini mothers were slightly older and healthier insofar as they smoked less (12.7% versus 21%) and had a slightly lower BMI than the population mean. Rates of vegetable and fruit consumption were comparable between Gemini parents and national statistics. White-British families were over-represented. The baseline parental characteristics in comparison to national health statistics are shown in **Table 3.2 and 3.3**.

**Table 3.2 Characteristics of Gemini twins compared to National twin statistics.**

Table adapted from van Jaarsveld et al. (2010).

| | **Gemini Cohort (Baseline)** | **National twin statistics[1]** |
|---|---|---|
| | **N (%)** | **%** |
| **Sex of twin pair** | | |
| Male | 785 (32.7%) | 32.1% |
| Female | 801 (33.3%) | 32.8% |
| Opposite sex | 816 (34.0%) | 35.1% |
| **Pre-term (<37 weeks)** | 1045 (43.5%) | 40% |
| | **Mean (SD)** | **Mean** |
| **Gestational age, mean (SD)** | 36.20 (2.48) | 37 |
| **Birth weight, mean (SD)** | 2.46 (0.54) | 2.50 |

[1] Office for National Statistics (2006). Birth Statistics Series FM1 no.35. Review of the Registrar General on births and patterns of family building in England and Wales. Newport. (Numbers are for twin births in 2006).

**Table 3.3 Characteristics of Gemini families compared to National health statistics.**

Table adapted from van Jaarsveld et al. (2010).

| Families | Gemini Cohort (Baseline) | National health statistics[1] |
|---|---|---|
| | **Mean (SD)** | **Mean** |
| **Age at twins' birth** | | |
| Mother | 33.6 (5.2) | 29.5[1] |
| Father | 36.4 (6.2) | |
| **BMI in kg/m$^2$** | | |
| Mother | 25.1 (4.8) | 26.8[2] |
| Father | 26.4 (3.9) | 27.1[2] |
| | **N (%)** | **%** |
| **Mother's Ethnicity** | | |
| White-British | 2089 (87.8%) | 72.6%[1] |
| Non White British | 311 (12.9%) | 21.9% |
| Not known | 2 (0.1%) | |
| **Current Smoker** | | |
| Mother | 306 (12.7%) | 21.0%[1] |
| Father | 466 (19.4%) | 24.0%[1] |
| **At least 5 portions of fruit per day** | | |
| Mother | 790 (32.9%) | 31.0%[1] |
| Father | 663 (27.6%) | 27.0%[1] |

[1] Health Survey for England 2007 Volume 1. Health lifestyles: knowledge, attitudes and behaviour. Ed R. Craig & N. Shelton. The health and social care Information Centre, 2008.

[2] BMI calculated from self-reported height and weight.

Gemini is a longitudinal study and data are still being collected. **Table 3.4** shows the characteristics of the Gemini sample at baseline, and for families who provided follow up data at 16 months and five years. Just like most longitudinal cohorts, there was participant attrition over time. The cohort started off with 2402 families, with reduced sample sizes at 16 months (1922 families) and five years (1039 families). Descriptive statistics were compared between the different time points to test for significant changes between the participating families. Across the first five years of the cohort, the sample became less representative of the general population. Compared to baseline, mothers contributing data when their twins were five years old had a lower BMI (25.41 versus 24.74, t (2336) = 3.43, $p < 0.05$) and were older (32.22 years versus 33.83 years, t(2394) = -7.62, $p < 0.01$). Mothers remaining in the study were more highly educated insofar as more had studied to degree level (49.5% versus 41.9%, $\chi$ (1) = 53.37, $p < 0.01$), and families were of higher socio economic status (70.8% versus 63.1%, $\chi$ (2) = 57.58, $p$ <0.01).

**Table 3.4 Characteristics of Gemini twins at baseline, 16 months and five years**

| | Gemini (Baseline) | Gemini 16 months | Gemini 5 years | National Statistics[2] |
|---|---|---|---|---|
| | **Mean (SD) or N (%)** | **Mean (SD) or N (%)** | **Mean (SD) or N (%)** | **Mean or %** |
| **N of children** | 4804 | 3844 | 2078 | |
| **Sex** | | | | |
| Male | 2386 (49.7%) | 1902 (49.2%) | 1053 (48.4%) | |
| Female | 2418 (50.3%) | 1942 (50.8%) | 1121 (51.6%) | |
| **Gestational age (weeks)** | 36.20 (2.48) | 36.21 (2.47) | 36.25 (2.44) | 37 |
| **Birth weight** | 2.46 (0.54) | 2.47 (0.54) | 2.46 (0.54) | 2.5 |
| **Twin Age at data collection** | 8.17 (2.18) | 15.82 (1.15) | 5.15 (0.13) | |
| **Maternal age at birth** | 33.6 (5.2) | 33.4 (5.0) | 33.8 (4.7) | 29.5 |
| **Maternal BMI at birth** | 25.1 (4.8) | 24.98 (4.64) | 24.73 (4.56) | 26.8 |
| **Maternal** | | | | |
| No degree | 1396 (58.1) | 1055 (54.9) | 525 (50.5) | |
| University degree | 1006 (41.9) | 867 (45.1) | 544(49.5) | |
| **Socioeconomic status[1]** | | | | |
| High | 1515 (63.1) | 1289 (67.1) | 736 (70.8) | 49% |
| Intermediate | 407 (16.9) | 307 (16.0) | 152 (14.6) | 18% |
| Low | 472 (19.7) | 320 (16.6) | 148 (14.2) | 33% |
| Not known | 8 (3) | 6 (0.3) | 3 (0.3) | |
| **Mother's Ethnicity** | | | | |
| White-British | 2089 (87.8) | 1698 (88.3) | 931 (89.1) | 72.8 |
| Non White British | 311 (12.9) | 224 (11.7) | 108 (10.9) | 27.4 |
| Not known | 2 (0.1) | 0 | 0 | |

[1]Classified using the Office for National Statistics National Statistics Socio-economic Classification (NS-SEC) (Office for National Statistics 2005) and grouped into higher (higher and lower managerial and professional occupations), intermediate (intermediate occupations, small employers and own account workers) and lower SES (lower supervisory and technical occupations, (semi)routine occupations, never worked and long-term unemployed)

[2] Health Survey for England 2007 Volume 1. Health lifestyles: knowledge, attitudes and behaviour. Ed R. Craig & N. Shelton. The health and social care Information Centre, 2008.

### 3.1.2 Zygosity assignment

One prerequisite of twin research is the successful identification of monozygotic (MZ) and di-zygotic (DZ) twin pairs. This can be an issue in large cohorts and especially when infants or very young children are involved. Due to both the high cost and the difficulty of collecting DNA samples using cheek swabs in very young children, questionnaires were the preferred method to assess zygosity status. Several zygosity questionnaires have been shown to be valid and reliable for use in children (Goldsmith, 1991; Price et al., 2000; Rietveld et al., 2000). The best way to test the validity of a zygosity questionnaire is to compare the measure against the results of DNA markers. Having reliable measures of zygosity is crucial for conducting successful twin research, especially in light of evidence suggesting that up to a third of parents misclassify their identical twins as non-identical due to misinformation from health professionals (Ooki, Yokoyama, & Asaka, 2004; C. H. M. van Jaarsveld, Llewellyn, Fildes, Fisher, & Wardle, 2012).

In Gemini, all opposite sex twins (816 pairs) were classified as DZ at baseline. Families with same-sex twins (1586 pairs) were asked to complete a questionnaire to determine the zygosity of their twins (Price et al., 2000) when they were on average eight months old (mean = 8.17, SD = 2.1). The 20-item questionnaire examines general physical resemblance, such as eye and hair colour, timing of teeth coming through, and the ability of others (friends and family members) to distinguish the siblings. All questions relating to twin's zygosity can be seen in **Appendix 2.1**.

934 families (58.9%) completed the questionnaire again when the twins were on average 29 months old (mean = 28.8, SD = 3.3). Mean questionnaire scores were calculated, creating values between 0 and 1 for each twin pair, and scores were used to determine zygosity. In line with Price et al (Price et al., 2000), lower scores indicate greater similarity, whereas higher scores indicate difference. Twin pairs scoring 0.64 and lower were classified as MZ; twin pairs scoring 0.70 and above were classified as DZ; scores between 0.64 and 0.70 were considered 'uncertain'. Of 934 families who answered the questionnaires at both time points, 66 pairs were found to be of uncertain zygosity. Of the remaining 868 pairs, 95.3% (827 pairs) of the zygosity assignment matched across the two time points.

In addition to the questionnaire, DNA was used to ascertain the zygosity of a subset of the twins. The process of zygosity testing with DNA involves detecting multiple tandem-repeat copies of 10-15 base pairs sequences, using hyper-variable minisatellite DNA probes. These tandem repeat copies can be found all over the

genome and are identical for MZ twins, but differ for DZ twins. (Hill & Jeffreys, 1985; Jeffreys, Wilson, & Thein, 1985). 1127 families provided DNA samples for both twins in order for them to be genotyped for obesity-related common genetic variants (single nucleotide polymorphisms, SNPs). Of these, 81 twin pairs were randomly selected for zygosity testing using their DNA in order to validate the zygosity questionnaire. In addition, some families elected to have DNA-based zygosity testing (n = 117) and we tested a further 112 pairs of the 1127 families who could not be classified using questionnaire data but who had provided DNA samples (88 pairs showed a mismatch between the baseline and 29 month zygosity questionnaires, while 24 pairs had unclear scores on the first zygosity questionnaire and were missing the second zygosity questionnaire).

For the 81 randomly selected pairs, genotyping and questionnaire classification of zygosity matched in all cases. Results from the questionnaire (all pairs for whom questionnaire data only was used to allocate zygosity, n=1239) and the DNA testing (all pairs who were zygosity tested using DNA, n=310, including: the random sample, the parent-requested sample, and the additional pairs who couldn't be classified via questionnaire) were combined to provide the most accurate zygosity assignment for the Gemini sample. A total of 749 twin pairs (31.2 %) were classified as MZ and 1616 (67.3%) twin pairs were classified as DZ (including 816 opposite sex DZ twins), based on the questionnaire and DNA results. For a further 37 pairs (1.5%) zygosity could not be established, as questionnaire results were unclear and no DNA was provided. **Table 3.5** shows the number of MZ and DZ pairs at baseline, 16 months and five years. The total number of pairs is declining as the study continues, but importantly the ratio of MZ and DZ twin pairs stays similar.

**Table 3.5 Zygosity at baseline, 16 months and five years**

| Zygosity established from questionnaire and DNA | | | |
|---|---|---|---|
| | **Baseline** | **16 months** | **Five years** |
| | **Frequency (%)** | **Frequency (%)** | **Frequency (%)** |
| **MZM** | 352 (14.7) | 290 (15.0) | 181 (16.7) |
| **DZM** | 409 (17.0) | 325 (16.8) | 172 (15.8) |
| **MZF** | 397 (16.6) | 326 (16.9) | 181 (16.7) |
| **DZF** | 391 (16.3) | 316 (16.4) | 209 (19.2) |
| **DZO** | 816 (34.0) | 644 (33.4) | 335 (30.8) |
| **Unknown** | 37 (1.5) | 30 (1.6) | 9 (0.8) |
| **Total** | 2402 | 1931 | 1087 |

Abbreviations: MZM = male-male monozygotic pairs; DZM = male-male dizygotic pair; MZF = female-female monozygotic pair; DZF = female-female dizygotic pair; DZO = opposite sex dizygotic pair

## 3.2 Measures

### 3.2.1 Emotional over- and under-eating in children

Emotional over- and under-eating were measured at five years using the Child Eating Behaviour Questionnaire (CEBQ), a parent report questionnaire designed to assess a range of eating behaviours in children (Wardle et al., 2001). The questionnaire consists of 35 items and parents use a 5-point Likert-scale to rate their child's behaviour (never', 'rarely', 'sometimes', 'often', or 'always'). The 35 items cluster into eight distinct eating behaviours: Satiety responsiveness, Food responsiveness, Emotional overeating (EOE), Emotional under-eating (EUE), Food fussiness, Desire to drink, Enjoyment of food and Slowness in eating. The original development paper reported high Cronbach's alphas for the EOE and EUE subscales (0.72 - 0.79 and 0.74 - 0.75 respectively) indicating good internal reliability (Wardle et al., 2001). Test-retest reliability has been shown to be moderate for both EOE (0.52) and EUE (0.64) over a two-week period (Wardle et al., 2001). The factor structure of the CEBQ has been replicated in different samples of children across many countries (Cao et al., 2012; Carnell & Wardle, 2007; Domoff et al., 2015; Mallan et al., 2013; Sparks &

Radnitz, 2012; Svensson et al., 2011; Viana et al., 2008; Wardle et al., 2001). The full questionnaire can be found in **Appendix 2.2**.

A principle component analysis (PCA) was conducted to verify the factor structure in this sample when the children were five years old (mean = 5.15, SD = 0.13). To conduct these analyses one twin was randomly selected from each family, leaving between 1036 and 1052 participants per item. This was necessary as data collected from families are not independent from another. Twins from the same family are deemed to be more similar to each other than two random individuals from the population. By selecting one twin per family, clustering of data in families was avoided. An oblique rotation method was used, which allows for correlations between factors, in line with the wealth of research showing interrelationships between the eight scales (see **Chapter 1.3.2.3**). All original 35 items were entered. The PCA supported the original factor structure, with eight components identified, each with an eigenvalue greater than one (range: 1.101, 7.873). The eight components accounted for 66.4% of the total variance. A full list of all items and their factor loadings (structure matrix, which allows for inter-correlations of the components) is presented in **Table 3.6**. Regarding the different components of interest, four items loaded substantially onto the EUE subscale ("My child eats less when tired", "My child eats more when happy", "My child eats less when angry", "My child eats less when upset"; all factor loadings > 0.63) replicating the originally proposed scale. For EOE, the items "My child eats more when worried", "My child east more when anxious" and "My child eats more when annoyed" all loaded on to one component (loadings > 0.79). The factor loading for the item "My child eats more when has nothing else to do" was higher on the Food Responsiveness component (0.59) than on the EOE component (0.42). This pattern was also described in the original development of the CEBQ. As pointed out by Wardle et al (2001), previous measures of child emotional overeating have included an item relating to boredom, such as the Dutch Eating Behaviour Questionnaire – Parent version and the Emotional Eating Scale – Child version. This item was therefore retained as part of the EOE subscale, as this was suggested by the authors who developed the Child Eating Behaviour Questionnaire (Wardle et al., 2001). The Cronbach's alpha (0.71) indicated good internal consistency of the EOE subscale. A more thorough discussion of this item can be found in **Chapter 11**.

Mean scores were calculated for CEBQ subscales EOE and EUE (range: 1 – 5) with higher scores indicating a greater emotional over- or under-eating behaviour. In order to calculate scores complete data was required on three out of the four items loading on EOE and EUE.

The CEBQ for toddlers (CEBQ-T) is a modified version of the Child Eating Behaviour Questionnaire (CEBQ), which is age-appropriate for a much younger sample. It was developed using intensive pilot work with 10 mothers of 15-month old toddlers who were recruited by the Gemini research team to discuss the proposed items. In relation to the EOE scale, the mothers were asked to consider which of the following adjectives were most appropriate to describe their twins' emotional states ('irritable', 'grumpy', 'anxious', 'has nothing else to do', 'tired, 'happy', 'and upset'). The mothers' answers informed the creation of the EOE subscale of the CEBQ-T, leading to the rewording of three items; 'worried', 'annoyed' and 'anxious' were replaced with 'irritable', 'grumpy' and 'upset', respectively. The item referring to eating in response to boredom was removed completely, as mothers felt that boredom was not an emotion that they were able to decipher in toddlers. The other major decision was that the EUE scale was removed entirely from the CEBQ-T. Mothers in the pilot work indicated that they did not recognise this behaviour in their children at this age. Moreover the Desire to drink scale was removed as some toddlers might still drink milk as part of meal. Therefore this scale was considered confusing at this age. A full script outlining all topics in the pilot phone calls can be found in **Appendix 2.3**. The full CEBQ-T can be found in **Appendix 2.4**.

A full PCA was conducted to verify the validity the CEBQ-T in this sample when the twins were 16 months old (mean = 15.82, SD = 1.15). As before, one twin was randomly selected from each family, resulting in a sample size between 1883 – 1927 children per item. As expected, the PCA revealed six components (all CEBQ scales except those not included in the CEBQ-T; EUE and Desire to drink) all with an eigenvalue greater than 1, explaining 61.9% of the variance in the 27 items. The majority of the items loaded on the expected components. The three items theorised to describe EOE loaded on one component, showing high factor loadings (< 0.83). A full list of the factor loadings of all items can be found in **Table 3.7.** The Cronbach's alpha for the EOE subscale was high (0.82) indicating high internal consistency. Just as for the CEBQ, mean scores for the EOE subscales (range 1 – 5) were calculated for children who had data on two out of three items.

**Table 3.6: Component loadings for all items of the Child Eating Behaviour Questionnaire when children were five years old (Structure Matrix)**

| Items[1] | | | Components Determined Through PCA[2] | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Original scale | N | 1 (EF) | 2 (FR) | 3 (FF) | 4 (DD) | 5 (EOE) | 6 (EUE) | 7 (SE) | 8 (SR) |
| **My child eats more when worried** | EOE | 1044 | | | | | -.788 | | | |
| **My child eats more when anxious** | EOE | 1036 | | | | | -.879 | | | |
| **My child eats more when annoyed** | EOE | 1041 | | | | | -.859 | .304 | | |
| **My child eats more when has nothing else to do** | EOE | 1041 | | .587 | | | -.417 | .336 | | |
| **My child eats less when tired** | EUE | 1053 | | | | | | .626 | | .350 |
| **My child eats more when happy** | EUE | 1041 | | | | | -.360 | .769 | | |
| **My child eats less when upset** | EUE | 1038 | | | | | | .848 | | |
| **My child eats less when angry** | EUE | 1036 | | | | | -.368 | .781 | | |
| **My child loves food** | EF | 1048 | -.810 | | .377 | | | | -.346 | -.386 |
| **My child looks forward to mealtimes** | EF | 1047 | -.766 | | .341 | | | | | |
| **My child is interested in food** | EF | 1051 | -.832 | | .406 | | | | -.336 | |
| **My child enjoys eating** | EF | 1047 | -.868 | | .399 | | | | -.312 | |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **My child leaves food on plate at the end of a meal** | SR | 1049 | .338 | | | | .389 | .773 |
| **My child cannot eat a meal if had a snack just before** | SR | 1044 | | | | .306 | | .563 |
| **My child gets full before meal is finished** | SR | 1049 | | | | | .387 | .834 |
| **My child gets full up easily** | SR | 1046 | .415 | | | .300 | .437 | .732 |
| **My child has a big appetite [R]** | SR | 1051 | .643 | -.453 | | | .433 | .445 |
| **My child takes more than 30 minutes to finish a meal** | SE | 1048 | .304 | | | | .805 | .319 |
| **My child finishes meal quickly [R]** | SE | 1048 | .376 | -.337 | | | .783 | .329 |
| **My child eats slowly** | SE | 1051 | | | | | .873 | .365 |
| **My child eats more slowly during the course of a meal** | SE | 1044 | | | | | .616 | .380 |
| **If allowed to my child would eat too much** | FR | 1052 | -.318 | .757 | | | | |
| **My child is always asking for food** | FR | 1053 | -.358 | .719 | | | | |
| **Even if my child is full up finds room to eat favourite food** | FR | 1046 | | .640 | | | | |
| **Given the choice my child would eat most of the time** | FR | 1043 | -.309 | .788 | .319 | | | |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Given the chance my child would always have food in mouth** | FR | 1046 | | .763 | | .349 | -.332 | | |
| **My child refuses new foods at first** | FF | 1052 | | | -.891 | | | | |
| **My child is difficult to please with meals** | FF | 1049 | .549 | | -.677 | | | .300 | .434 |
| **My child decides that does not like a food even without tasting it** | FF | 1049 | .315 | | -.838 | | | | .301 |
| **My child enjoys tasting new foods [R]** | FF | 1053 | .422 | | -.900 | | | | |
| **My child enjoys a wide variety of foods [R]** | FF | 1048 | .614 | | -.725 | | | | |
| **My child is interested in tasting food not tasted before [R]** | FF | 1049 | .430 | | -.880 | | | | |
| **My child is always asking for a drink** | DD | 1053 | | | | .809 | | | |
| **If given the chance my child would drink continuously throughout the day** | DD | 1049 | | | | .907 | | | |
| **If given the chance my child would always be having a drink** | DD | 1047 | | | | .921 | | | |

[1] Items marked with [R] have been reversed for scoring purposes

[2] The *n* is based on half the sample, selecting one twin at random out of each family

Abbreviations: EOE; 'Emotional overeating'; EF, 'Enjoyment of food'; SR, 'Satiety responsiveness'; SE, 'Slowness in eating'; FR, 'Food responsiveness'; FF, 'Food fussiness'

**Table 3.7 Component loading for all items of the Child Eating Behaviour Questionnaire – Toddler (Structure Matrix)**

| Item[1] | Components Determined Through PCA[2] | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Original scale | N | 1 (FR) | 2 (EOE) | 3 (SR) | 4 (FR) | 5 (FF) | 6 (SE) |
| **My child eats more when irritable** | EOE | 1914 | | .829 | | | | |
| **My child eats more when grumpy** | EOE | 1921 | | .888 | | .325 | | |
| **My child eats more when upset** | EOE | 1920 | | .860 | | .342 | | |
| **My child loves food** | EF | 1926 | -.832 | | -.402 | | -.427 | -.351 |
| **My child is interested in food** | EF | 1926 | -.807 | | | | -.410 | |
| **My child enjoys eating** | EF | 1926 | -.827 | | -.329 | | -.473 | |
| **My child looks forward to mealtimes** | EF | 1902 | -.773 | | | | -.358 | |
| **My child has a big appetite [R]** | SR | 1925 | .688 | | | -.477 | | .423 |
| **My child gets full before meal is finished** | SR | 1924 | .337 | | .771 | | | .401 |
| **My child leaves food on plate or in the jar at the end of a meal** | SR | 1927 | .377 | | .716 | | .319 | .469 |
| **My child cannot eat a meal if had a snack just before** | SR | 1883 | .310 | | .562 | | | |
| **My child gets full up easily** | SR | 1923 | .434 | | .689 | | | |
| **My child finishes meal quickly [R]** | SE | 1926 | .421 | | | -.318 | | .796 |
| **My child eats slowly** | SE | 1926 | .310 | | .326 | | | .808 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **My child eats more slowly during the course of a meal** | SE | 1922 | | | .591 | | | .309 |
| **My child takes more than 30 minutes to finish a meal** | SE | 1927 | | | .354 | | | .665 |
| **My child is always asking for food** | FR | 1914 | | .338 | | .628 | | |
| **If allowed to my child would eat too much** | FR | 1920 | | | | .746 | | |
| **Given the choice my child would eat most of the time** | FR | 1926 | -.353 | | | .817 | | |
| **Even when my child has just eaten well is happy to eat again if offered** | FR | 1915 | | | | .771 | | |
| **My child refuses new foods at first** | FF | 1926 | .310 | | | | .808 | |
| **My child enjoys a wide variety of foods R** | FF | 1926 | .615 | | | | .690 | |
| **My child enjoys tasting new foods R** | FF | 1926 | .492 | | | | .802 | |
| **My child refuses to eat certain types of food** | FF | 1927 | | | .316 | | .709 | |
| **My child is difficult to please with meals** | FF | 1925 | .551 | | .423 | | .711 | .319 |
| **My child decides that does not like a food even without tasting it** | FF | 1926 | .331 | | | | .763 | |
| **My child is interested in tasting food not tasted before [R]** | FF | 1926 | .444 | | | | .788 | |

[1] Items marked with [R] have been reversed for scoring purposes ; [2] The *n* is based on half the sample, selecting one twin at random out of each family. Abbreviations: EOE = Emotional overeating; EF = Enjoyment of food; SR = Satiety responsiveness; SE = Slowness in eating; FR = Food responsiveness; FF = Food fussiness

### 3.2.2 Measuring eating behaviours in adults – The Dutch Eating Behaviour Questionnaire

The Dutch Eating Behaviour Questionnaire (DEBQ) was sent to Gemini parents, when their twins were two years old. The original DEBQ consists of 33 items measuring three distinct eating behaviours: External eating, Restraint and Emotional eating. The primary care giver was asked to rate how often they engaged in the 33 behaviours using a 5 point Likert-scale (ranging from 'seldom' to 'often') (Van Strien et al., 1986). The DEBQ has been described in more detail in section **Chapter 1.3.2.1**.The External eating scale describes sensitivity to external food cues, as in the tendency to eat more if food is appealing. This subscale consists of ten items (e.g. "If food smells and looks good, do you eat more than usual?"). The Restraint scale comprises ten questions and covers restrictive behaviours regarding the limitation of food intake (e.g. "Do you try to eat less at mealtimes than you would like to eat?"). The Emotional eating scale has 13 items and questions probe overeating in response to negative emotions and stress (e.g. "Do you have a desire to eating when you are feeling lonely?"). The DEBQ is a widely used psychometric questionnaire and its structure and validity have been supported in multiple samples (Bozan, Bas, & Asci, 2011; Cebolla, Barrada, van Strien, Oliver, & Banos, 2014; Dakanalis et al., 2013; Dutton & Dovey, 2016; Lluch et al., 1996; Wardle, 1987).

A shortened version of the DEBQ was sent to the Gemini families when the twins were two years old (24.89 months, SD = 1.30). A list of all 15 items included is presented in **Table 3.8**. This shortened version has previously been used in other cohorts such as the Twins Early Development Study (TEDS) and extensive pilot work suggested that the shortened scale correlated very well ($r > 0.9$) with the full original scale consisting of 33 items.

Principal component analysis was used to test the factor structure of this shortened version of the DEBQ in this sample of Gemini mothers. Ratings on all items were available for the majority of mothers (n range 1363 - 1367). The results revealed three factors with Eigenvalues greater than 1 explaining 67.7% of the variance. The three factors clustered into External eating, Restriction and Emotional eating, just as proposed in the original scale. The Cronbach's alphas were high for all three subscales: External Eating, alpha = 0.82; Restriction, alpha = 0.89; and Emotional

Eating, alpha = 0.91; indicating high internal consistency. Mean scores for three subscales (range: 1 - 5)

**Table 3.8 Component loading for all items of the shortened Dutch Eating Behaviour Questionnaire in Gemini (Structure Matrix)**

| Dutch Eating Behaviour Questionnaire* | | | | | |
|---|---|---|---|---|---|
| **Items** | **Original scale** | **N** | **1 (EE)** | **2 (R)** | **3 (EX)** |
| **Desire to eat something delicious…** | EX | 1365 | | | 0.639 |
| **Eat more than normal when food is good…** | EX | 1366 | | | 0.830 |
| **Eat more than usual when food is tasty…** | EX | 1367 | | | 0.842 |
| **Desire to eat when others are eating…** | EX | 1367 | 0.315 | | 0.698 |
| **Desire to keep eating when delicious…** | EX | 1367 | | | 0.795 |
| **Reject food or drinks because of worry about weight…** | R | 1367 | | -0.805 | |
| **Take into account weight when eat…** | R | 1367 | | -0.845 | |
| **Eat foods that are slimming…** | R | 1365 | | -0.798 | |
| **Try not to eat in evening because watching weight…** | R | 1364 | | -0.842 | |
| **Not eating because of watching your weight…** | R | 1367 | | -0.858 | |
| **Desire to eat when somebody lets you down…** | EE | 1365 | 0.906 | -0.322 | |
| **Desire to eat when angry…** | EE | 1365 | 0.878 | | |
| **Desire to eat when disappointed…** | EE | 1366 | 0.822 | | |

| | | | |
|---|---|---|---|
| **Eating when you feel lonely…** | EE | 1363 | 0.921 |

Abbreviations: EX = External eating, R = Restriction, EE = Emotional eating

*Exact item wording cannot be shown because of copyright restrictions.

### 3.2.3 Measuring eating behaviour in infants – The Baby Eating Behaviour Questionnaire (BEBQ)

The Baby Eating Behaviour Questionnaire (BEBQ) is a modified version of the CEBQ to assess infant eating behaviours (Llewellyn et al., 2011). The questionnaire consists of 18 items, rated by parents on a 5-point Likert-scale, when twins were eight months old. Principal Component Analyses revealed four distinct eating behaviours: Enjoyment of food (four items, e.g. “My baby enjoyed feeding time”), Food responsiveness (six items, e.g. “If given the chance, my baby would always be feeding”), Slowness in eating (four items, e.g. “My baby fed slowly”), Satiety responsiveness (three items, e.g. “My baby got full up easily”) and one item regarding general hunger (“My baby had a big appetite”). The internal reliability of the constructs was good with Cronbach’s alphas ranging from 0.73-0.81 (Llewellyn et al., 2011). The validity of the questionnaire and the association between infant eating behaviour and weight has been confirmed in an independent Australian sample (Mallan, Daniels, & de Jersey, 2014). The full BEBQ can be found in the **Appendix 2.5**. This questionnaire was used alongside the CEBQ-T in Study 7 (**Chapter 10**) to examine twin-specific parental bias in reporting of twin eating behaviour.

### 3.2.4 Emotion regulation

Child emotion regulation was measured using a subscale of the Strengths and Difficulties Questionnaire (SDQ) (Goodman, 2001) and described how easily upset and emotional a child is. None of the items were adapted for the Gemini cohort. The scale consisted of five items, which were rated on a five point Likert-Scale by the parents choosing from the following options ‘never’, ‘rarely’, ‘sometimes’, ‘often’, or ‘always’. The five items were:

- My child often complains of headaches, stomach-aches etc.
- My child has many worries, and often seems worried
- My child is often unhappy, downhearted, or tearful
- My child is nervous or clingy in new situations
- My child has many fears, and is easily scared

Mean scores (range: 1 – 5) were calculated with higher scores indicating lower emotion regulation ability. At least four of the five items needed to be scored to calculate the mean. The internal consistency of this scale was good (alpha = 0.74).

### 3.2.5 Measuring household stress – The Confusion, Hubbub and Order Scale

The Confusion, Hubbub and Order Scale (CHAOS) is a parent-report psychometric tool devised to quantify the stress experienced in a household. The original scale consists of 15 questions and includes items probing noise, overcrowding and family conflicts (A. P. Matheny, Wachs, Ludwig, & Phillips, 1995). When their twins were five years old Gemini parents answered a shortened version of the CHAOS scale, which was developed previously for other twin cohorts (Hart, Petrill, Deckard, & Thompson, 2007; Petrill, Pike, Tom, & Plomin, 2004). Parents indicated if the statements applied to their household by choosing between "true" or "false". The following six items were included and are listed in **Table 3.9.** A total mean score for each family was calculated by dividing the number of 'true' statements indicative of family chaos by the total number of answered items. Scores ranged from 0 (no chaos) to 1 (highest indication of family chaos). Cronbach's alpha of 0.79 suggested good reliability.

**Table 3.9 Items of the short version of the The Confusion, Hubbub and Order Scale (CHAOS)**

| The Confusion, Hubbub and Order Scale |
|---|
| We almost always seem to be rushed |
| It is a real zoo in our home |
| There is often a fuss going on in our home |
| You cannot hear yourself think in our home |
| Our home is a good place to relax (reversed scored) |
| The atmosphere in our home is calm (reversed scored) |

### 3.2.6 Measuring parental feeding practices

In order to create a rich picture of parental feeding practices in Gemini, mothers were sent an extensive battery of questions about their feeding practices when their twins were 16 months and five years old. Analyses in this thesis only included Emotional feeding measured at *both* 16 months and five years. For all other subscales only data collected at five years were included.

Many questionnaires have been developed to measure a wide variety of parental feeding practices, and most questionnaires include multiple scales. For Gemini,

certain standalone subscales were selected from larger questionnaires, without necessarily including the whole questionnaire in its entirety. The scales chosen to measure different parental feeding styles are described in the following sections. All described scales mothers were asked to indicate how much the states behaviours apply them using a five point Likert-scale ('never', 'rarely', 'sometimes', 'often', or 'always'). Mean scores were calculated (range: 1 – 5) for each parental feeding practice with higher scores indicating higher tendency to engage in the measured behaviour.

#### 3.2.6.1 Emotional feeding

Emotional feeding describes the tendency to offer a child food in order to soothe him or her. Parents rated their emotional feeding behaviour using a subscale of the Parental Feeding Style Questionnaire (PFSQ) (Wardle et al., 2002) twice, once when their twins were 16 months and again when their twins were five years old. Parents used a five point Likert-scale, ranging from 'never' to 'always'. In order to ensure that the phrasing of the items was appropriate for toddlers, extensive pilot work was conducted including telephone interviews with mothers of toddlers. This process led to the rephrasing of four out of five items ("hurt him/herself" changed to "has been hurt", "angry" changed to "grumpy", "worried" changed to "irritable" and "feeling bored" rephrased as "I give my child something to eat to occupy him/her (when in company, travelling etc.)). Data on three out of four items were required to calculate the mean scores at both time points. A full list of all items included at 16 months and five years can be found in **Table 3.10**. The Cronbach's alphas indicated good internal consistency at 16 months (alpha = 0.85) and five years (alpha = 0.79).

**Table 3.10 List of items measuring Emotional feeding at 16 months and five years**

| **Emotional Feeding at 16 months (adapted from PFSQ)** |
|---|
| I give my child something to eat to make him/her feel better when he/she is feeling upset |
| I give my child something to eat to make him/her feel better when he/she has been hurt |
| I give my child something to eat to make him/her feel better when he/she is grumpy |
| I give my child something to eat to make him/her feel better when he/she is feeling irritable |
| I give my child something to eat to occupy him/her (e.g. when in company or travelling) |
| **Emotional Feeding at five years (taken from PFSQ)** |
| I give my child something to eat to make him/her feel better when he/she is feeling upset |
| I give my child something to eat to make him/her feel better when he/she has hurt himself/herself |
| I give my child something to eat to make him/her feel better when he/she is feeling angry |
| I give my child something to eat to make him/her feel better when he/she is worried |
| I give my child something to eat to occupy him/her if he/she is feeling bored |

Abbreviations: PFSQ = Parental Feeding Style Questionnaire

### 3.2.6.2 Instrumental feeding

Parents indicated their tendency to use food as a contingency for a desired outcome such as good behaviour. The instrumental feeding subscale from the PFSQ (Wardle et al., 2002) was included in the questionnaire booklet when twins were five years old. An additional item ("I use foods my child likes as a way to get him/her to eat 'healthy' foods') was added to tap into the use of food to reward the consumption of healthy foods. A list of the five items relating to instrumental feeding can be found in **Table 3.11**. Data on at least four items were required to calculate the mean scores. A Cronbach's alpha of 0.67 was only slightly lower than the acceptable range (> 0.7).

### 3.2.6.3 Encouragement

Encouragement refers to the parent's tendency to motivate their child to eat more. This subscale (five items, data on at least four items required to be included) was taken from the PFSQ (Wardle et al., 2002). Items were amended to refer specifically to fruit and vegetable consumption and a more varied diet, rather than just more food in general. One item "I encourage my child to try foods that s/he hasn't tasted before" was deleted because it was deemed too similar to "I praise my child is s/he eats a new food". A list of the items included can be seen in **Table 3.11.** The Cronbach's alpha of this subscale was lower than desired (alpha = 0.60).

### 3.2.6.4 Control

Also from the PFSQ (Wardle et al., 2002), the subscale Control was adapted and sent to parents. Controlling feeding practices indicate the extent to which parents regulate their children's food intake. This subscale consisted of six items, which was a shortened version of the original ten items. Four items that were deemed too similar to other items in the scale were removed:

- 'I allow my child to decide when s/he has had enough snacks to eat'
- 'I decide when it is time for my child to have a snack'
- 'I decide the times when my child eats his/her meals'
- 'I insist my child eats meals at the table'

A list of all Control items included can be seen in **Table 3.11**. Data on five out of the six items were required to calculate the mean score for this subscale. The internal reliability of the scale was slightly lower than desired (Cronbach's alpha = 0.65).

### 3.2.6.5 Pressure to eat

The Pressure to eat scale was adapted from the Child Feeding Questionnaire (CFQ) (Birch et al., 2001).This feeding practice refers the parents' tendency to coerce their children to eat more, such as demanding them to finish their plates. In addition to the original four items, one item was added to the original scale to capture pushiness with regard to fruit and vegetables specifically: "I insist my child eats some fruit or vegetables, even is s/he doesn't want them". No other changes were made. A full list of all items measuring Pressure to eat can be found in **Table 3.11**. Data on at least four items was needed to calculate the mean score. The Cronbach's alpha of 0.61 was lower than desired.

#### 3.2.6.6 Monitoring

The Monitoring subscale was taken from the CFQ (Birch et al., 2001) and refers to the tendency of the parents to track their children's food intake. The original items were formulated as questions and were rephrased into statements for the Gemini questionnaire in order to fit the other parental feeding items. The three items included in this scale are presented in **Table 3.11**. Data on at least two of three items were required to calculate the mean scores. The internal consistency of this subscale was acceptable (alpha = 0.72).

#### 3.2.6.7 Modelling

Parental modelling describes the degree to which parents aim to act as a role model to influence their children's eating. Parent might actively show their children how much they enjoy eating healthy foods. The scale used was from the Comprehensive Feeding Practices Questionnaire (CFPQ) (Musher-Eizenman & Holub, 2007). It includes four items which can be seen in **Table 3.11**. Mothers needed to have at least three out of the four items for the mean score to be calculated. The internal consistency of this scale was good (alpha = 0.79).

#### 3.2.6.8 Mealtime structure

Mealtime structure refers to the general rules at mealtimes, such as allowing the children to eat in front of the TV. This scale used was from the Preschool Feeding Questionnaire (Baughcum et al., 2001) without any changes, and included three items which can be seen in **Table 3.11**. Data on at least two of the three items were required to calculate the mean score. The Cronbach's alpha indicated that the internal reliable of the scale was low (alpha = 0.43).

**Table 3.11 List of parental feeding practices measured when twins were five years old**

| **Instrumental feeding (adapted from PFSQ)** |
|---|
| I use foods that my child likes as a way to get him/her to eat “healthy” foods |
| If my child misbehaves I withhold his/her favourite food |
| I use puddings as a bribe to get my child to eat his/her main course |
| I reward my child with something to eat when he/she is well-behaved |
| In order to get my child to behave him/herself I promise him/her something to eat |
| **Encouragement (adapted from PFSQ)** |
| I encourage my child to eat a wide variety of foods |
| I praise my child if he/she eats fruit or vegetables |
| I encourage my child to eat fruit or vegetables |
| I present fruit or vegetables in an attractive way to my child |
| I praise my child if he/she eats a new food |
| **Control (adapted from PFSQ)** |
| I allow my child to choose which foods to have for meals |
| I decide how many snacks my child should have |
| I let my child decide when he/she would like to have his/her meal |
| I let my child eat between meals whenever he/she wants |
| I decide what my child eats between meals |
| I allow my child to wander around during a meal |
| **Pressure to eat (adapted from CFQ)** |
| I have to be especially careful to make sure my child eats enough |
| My child should always eat all of the food on his/her plate |
| If I did not guide or regulate my child’s eating, he/she would eat much less than he/she should |
| I insist my child eats some fruit or vegetables, even if he/she doesn’t want them |

| |
|---|
| If my child says “I’m not hungry”, I try to get him/her to eat anyway |
| **Monitoring (adapted from CFQ)** |
| I keep track of the high fat foods that my child eats |
| I keep track of the sugary foods that my child eats |
| I keep track of the foods my child’s been eating when he/she is not with me (e.g. with a childminder or family member) |
| **Modelling (adapted from CFPQ)** |
| I show my child how much I enjoy eating healthy foods |
| I try to eat healthy foods in front of my child, even if they are not my favourite |
| I try to show enthusiasm about eating healthy foods |
| I model healthy eating for my child by eating healthy foods myself |
| **Mealtime structure (adapted from PFQ)** |
| My child watches TV during meals (reversed coded) |
| My child has a set mealtime and snack routine |
| I sit down with my child when he/she eats meals |

Abbreviations: PFSQ = Parental Feeding Style Questionnaire, CFQ = Child Feeding Questionnaire, CFPQ = Comprehensive Feeding Practices Questionnaire, PFQ = Preschool Feeding Questionnaire

### 3.2.7 Milk-feeding method and milk-feeding philosophy

In addition to the questionnaires regarding parental feeding practices, mothers were asked to indicate the milk-feeding method they used during the first three months of their twins' lives. The mother could choose from the following options: 'entirely breastfeeding'; 'mostly breastfeeding with some bottle-feeding'; 'equally breastfeeding and bottle-feeding'; 'mostly bottle-feeding and some breastfeeding'; 'almost entirely bottle-feeding (only tried breastfeeding a few times)'; 'entirely bottle-feeding (never tried breastfeeding)'; and 'other'. Mothers were also asked to indicate whether they followed a strict feeding routine or responded freely to the demands of their twins. Mothers were asked: 'Which of the following best describes each of your twin's eating routine during their first three months?'. The response options were: 'I fed my baby whenever he/she cried, got fussy or seemed hungry', 'My baby was on a flexible feeding schedule (about every 3-4 hours)' and 'My baby on a rigid feeding schedule (e.g. I woke him/her up to eat on time)'. The first two options were collapsed into one category, creating two overall groups ('On demand' and 'On schedule' feeding). These questions and response options were taken from the Infant Feeding Questionnaire (Baughcum et al., 2001)

### 3.2.8 Anthropometric measures

Weight and height are parent-reported every three months in the Gemini cohort. At baseline (eight months), parents were asked to provide all the anthropometric measurements taken by health professionals up to that point (in 'the red book'). In cases where this information was unavailable, parents were asked to report the birth weight (and current weight) of their twins themselves (3.6% of parents). The same procedure was used to measure all anthropometric measures taken prior to the 16-month questionnaire completion. When the twins were about two years old, all parents were sent digital weighing scales and a height chart to measure their twins themselves. Parents are asked to upload the heights and weights of their children every three months on to the Gemini website, providing a rich database of the twins' anthropometrics. Weight and height were also measured in the five-year questionnaire. Birth weight, weight at 16 months, and weight and height at five years were included in this thesis. At five years, height and weight measurements were used to create body mass index (BMI: weight (kg) / height $(m)^2$) Measurements closest to 60 months (five years) were used. If data were missing at 60 months, data collected at 57 or 63 months was used to generate the largest sample size possible.

BMI was not used for children aged 16 months as height cannot be measured reliably until two years of age. Furthermore age-, gestational age-, and sex-adjusted standard deviation scores (SDS) for weight and BMI were calculated using British 1990 growth reference data. SDS scores are used rather than raw scores for children, because children's weights and BMIs vary considerably with development before 18 years of age. SDS provide an indication of how the children's weights and BMI compare to other British children of the same age and sex using reference data (Cole, 1990; Freeman et al., 1995).

### 3.2.9 Socio-demographic information

#### 3.2.9.1 Age

For each data collection phase included in this thesis (16 months and five years), chronological age was calculated using the twins' birthdays and the date when the questionnaires were completed. At baseline, gestational age was recorded by asking mothers to report the number of weeks they had been pregnant at the time of birth. Additionally the age of the parents at the birth of their twins was calculated from the differences between the parents' and twins' dates of birth.

#### 3.2.9.2 Marital status

The baseline questionnaire included an item on the martial status of the main responder. Categories were: 'married or cohabiting', 'divorced', 'widowed', 'separated' or 'single'. The last four categories were combined into one, creating a two category variable, dividing responders in 'with partner' or 'single'.

#### 3.2.9.3 Parental education and employment

At baseline responders were asked to indicate their level of education choosing from one of the following categories, which were then collapsed into two groups indicating if mothers had a university degree including response options ; 'Undergraduate degree' and 'Postgraduate qualification (Masters, PhD) versus 'No qualifications' and 'CSE, GCSE or 'O' level'; 'Vocational qualification (GNVQ, BTEC)' 'A' or 'AS' level' 'Higher National Certificate (HNC) or Diploma (HND).

Also at baseline, mothers were asked to report their employment status from the following options: 'at home', 'maternity leave', 'decided to stay home', 'part time or full time employment'. Responses were then collapsed into two categories: Mothers either 'at home' or some form of 'employment'. This was done to indicate if a mother

was at home with her twins, regardless of what the reason for it was, such as unemployment or deciding to be a stay at home mother.

#### 3.2.9.4 Ethnicity

In the baseline questionnaire, parents were asked to indicate their ethnicity choosing from one of 16 categories: 'White British', 'White Irish', 'Other White background', 'Caribbean', 'African', 'Other Black background', 'Indian', 'Pakistani', 'Bangladeshi', 'Other Asian background', 'White and Black Caribbean', 'White and Black African', 'White and Asian', 'Other Mixed background', 'Chinese', 'Any other'.

Responses were then dichotomised in to 'White-British' and 'Non-White British'. A large proportion of the families rated themselves as 'White-British'. Subgroup analyses of other specific ethnic groups were therefore not possible.

#### 3.2.9.5 Socio-economic status

Parents also described their occupation and that of their partner and this was used to calculate the National Statistics Socioeconomic Class (NS-SEC) index. This tool assigns job descriptions to a corresponding four digit Standard Occupational Classification 2000 code (Office for National Statistics, 2005). These codes are then linked to a reversed eight-category NS-SEC classification; with higher scores representing higher socioeconomic class. The person in the household with the highest score was defined as the household reference and their score represented household NS-SEC. The reference person was the partner for 41% of families, the mother for 29% of families and was equal in 18% of families. In the other 12% of families, data were missing or the mother was single and therefore the mother was automatically assigned as household reference person. NS-SEC scores were grouped into three categories in order to have adequate group sizes for analyses. These categories are: higher (higher and lower managerial and professional occupations), intermediate (intermediate occupations, small employers and own account workers – self-employed with no employees) and lower occupational classifications (lower supervisory and technical occupations, (semi-) routine occupations, never worked and long-term unemployed).

This three category NS-SEC was used as the main indicator of socio-economic status in this thesis as it has been shown to have clear construct validity in identifying social

class differences in validated health outcome measures (Chandola & Jenkinson, 2000)

## 3.3 Conclusion

The Gemini cohort is the largest twin birth cohort focussing on the development of eating behaviours in early life, and the only twin cohort with measures of emotional eating in childhood. It is therefore the ideal sample to address the research questions posed in this thesis (**Chapter 2**). The Gemini cohort is largely representative of twins in the general population. However, as is the case in most cohorts, families of white ethnicity are over represented and parents are of higher socio-economic status, healthier, and have a slightly lower mean BMI than the wider UK population. Gemini includes one of the most detailed measures of emotional over and under-eating at two crucial stages of development, toddlerhood and middle childhood. Furthermore the extensive use of measures capturing the home environment and parental feeding strategies within a twin cohort allows for the investigation of gene-environment interplay.

## 3.4 Analyses

### 3.4.1 Complex samples general linear modelling

Complex samples general linear models (CSGLM) were used to test the associations between parental feeding practices, home environmental factors, socio-economic status, and individual child factors on childhood EOE and EUE (see Study 3, **Chapter 6**). CSGLM enables analysis of data from related individuals, in which variables are deemed to correlate in families (Carlin, Gurrin, Sterne, Morley, & Dwyer, 2005). This method adjusts for clustering of the twins in families by widening the standard errors around the regression coefficient in order to account for the reduced variation in the sample, due to correlation between twins. This way the full dataset can be analysed, maximising the sample size, which increases the power to detect small effects. The effect of each predictor is presented by unstandardized B-values (B). The greater a B-value the larger the effect of the predictor on the outcome variable (child EOE and EUE in this thesis). A positive B indicates that as the predictor increases, the outcome variable also increases; a negative B indicates that as the predictor increases, the outcome variable decreases. The proportion of variance in the outcome variable explained by all the predictors in the model (in the aggregate) is indicated by the $R^2$.

A greater value of $R^2$ implies a greater proportion of variance explained in the given model. $R^2$ values can be directly transferred into percentages (e.g. $R^2 = 0.25$ equals 25% variance explained) easing interpretation.

Data analysed with CSGLMs needs to meet the same assumptions as when using linear regression (Field, 2013). These are: (i) linearity of association; (ii) independence of error, although CSGLM account for the non-independence of family data; (iii) homoscedasticity; (iv) normality of residuals; and (v) no collinearity. The tests to verify that the data met these assumptions are presented in the corresponding study (**Chapter 6**).

Power calculations were conducted using G*Power (version 3.0.10; Softpedia) (Faul, Erdfelder, Lang, & Buchner, 2007). Models including up to ten predictors and a sample of over 800 participants, included in the analyses in Study 3, had 99% power to detect a small $R^2$ of 0.05.

### 3.4.2 Structural equation modelling

Structural Equation Modelling (SEM) was used to test bidirectional prospective associations between parental feeding practices and child EOE (Study 4 **Chapter 7**). In comparison to other methodologies, SEM enables direct comparison of bi-directional regression coefficients within one model, instead of running two separate regression analyses, allowing for more meaningful interpretation. Analyses were conducted in R using the statistical package lavaan (Rosseel, 2012) and its add-on lavaan.survey (Oberski, 2014) which also enables adjustment for clustering of twins in families. Data from both twins in a pair can therefore be included in the analyses, maximising the sample size and statistical power. Previous literature suggest that any SEM model should include more than 200 participants (Weston & Gore, 2006); Study 4 included 821 participants.

## 3.5 The twin method

Over the past century the twin method has been used to investigate genetic and environmental contributions to individual differences in complex human traits. Researchers have been using this methodology to examine a wide spectrum of aspects of human life accumulating in a total of 17,804 investigated traits, spanning from disease, to behaviour to political opinion. Twin research is conducted worldwide

and more than 14 million twins are currently included in a multitude of studies (T. J. C. Polderman et al., 2015).

### 3.5.1 The underlying logic of the twin method

The twin method was formulised at the turn of the last century and its underlying assumptions remain today (Fisher, 1919; Rende, Plomin, & Vandenberg, 1990). The twin method takes advantage of the natural occurrence of identical, monozygotic (MZ) and non-identical, dizygotic (DZ) twins. MZ twins are natural clones, sharing all of their genetic material, whereas DZ twins share on average 50% of their segregating genes, like any other regular siblings. Importantly, however, MZ and DZ twins are assumed to share their environments to a very similar extent (from the prenatal environment to later environmental factors). This being true, if MZ twins are more similar than DZ twins on the trait of interest, researchers assume a genetic contribution to trait variation because the only difference between the two types of twins is that MZs are twice as similar genetically, while both types of twins share their environments equally. Comparing the resemblance between MZ and DZ twins on a measurable trait enables researchers to decompose the variation of the trait into genetic and environmental contributions. Resemblance between MZ twins could reflect both their genetic relatedness as well as aspects of the shared environment, because they share 100% of both; but the extent to which they differ captures only environmental influences unique to each individual twin, as well as measurement error.

Comparison of MZ and DZ pairs allows for the variation (V) of any given trait to be decomposed into three latent factors: (i) heritability or genetic effects[2] (A), (ii) shared environmental effects (all factors that increase similarity between two twins in a pair, above and beyond genetic resemblance) (C), and (iii) non-shared environmental effects (factors that contribute to differences between the pairs), which also includes measurement error (E) (Robert Plomin, DeFries, Knopik, & Neiderhiser, 2013).

[2] From this point forward the terms ‘heritability’ and ‘genetic effects’ are used interchangeably

### 3.5.2 Estimating genetic and environmental variance contributions using correlations

The simplest way to derive indications of genetic and environmental contributions to variation in any given trait is to compare intraclass correlations (ICCs) for MZ and DZ pairs. If similarity of MZ twins is greater than the correlation of DZ twin pairs for the same phenotype, a significant effect of genes can be inferred, as the only assumed difference between the two types of twins is that MZ twins are twice as similar genetically as DZ twins. If correlations for MZ and DZ twin pairs are similar, environmental influences shared between two twins in one family can be assumed to be important in explaining individual differences in the observed phenotype. Falconer's Formula (Falconer & Mackay, 1996) can be used to calculate rough estimates of the effect of the latent factors A, C and E. The total variance (V) is decomposed into the three components: A, C and E (V = A + C + E). The correlation between the MZ pairs (rMZ) includes all genetic effects and all shared environmental effects: rMZ = 1A + 1C. For DZ twins the correlation (rDZ) reflects only half of the genetic effects, but all shared environmental effects: rDZ = 0.5*A + 1C. Using these equations, the contributions of A, C and E to the total variance (V = 1) can be calculated. Genetic effects (A), or heritability ($h^2$), are calculated by doubling the difference between the MZ and DZ correlations: A = 2*(rMZ - rDZ). Non-shared environmental factors are everything that does not contribute to MZ twin similarity: E = 1 - rMZ. Because the three variance components together amount to 1, the shared environmental contribution can be calculated from A and E (C = 1 – A + E) (Rijsdijk & Sham, 2002).

### 3.5.3 Estimating genetic and environmental variance contributions using path analyses and structural equations

Following on from Falconer's Formula the univariate twin model can be illustrated as a path diagram (**Figure 3.2**). The latent factors A, C and E are represented in circles and the measured phenotype (e.g. emotional overeating) in rectangular boxes for two twins in a pair. The double-headed arrows connect the twins, representing their relationship in accordance with their zygosity. MZ twins are genetically identical and so the correlation between the latent factor A is constrained to 1, whereas DZ twins only share on average half of their genes, so their correlation of genetic relatedness is fixed at 0.5. Regardless of their zygosity, both types of twins share their environments to the same extent, so the correlation for the shared environment (C)

is fixed at 1 for both MZs and DZs. Because the non-shared environmental factors (E) contribute to differences between the twins, this latent factor is not correlated between them.

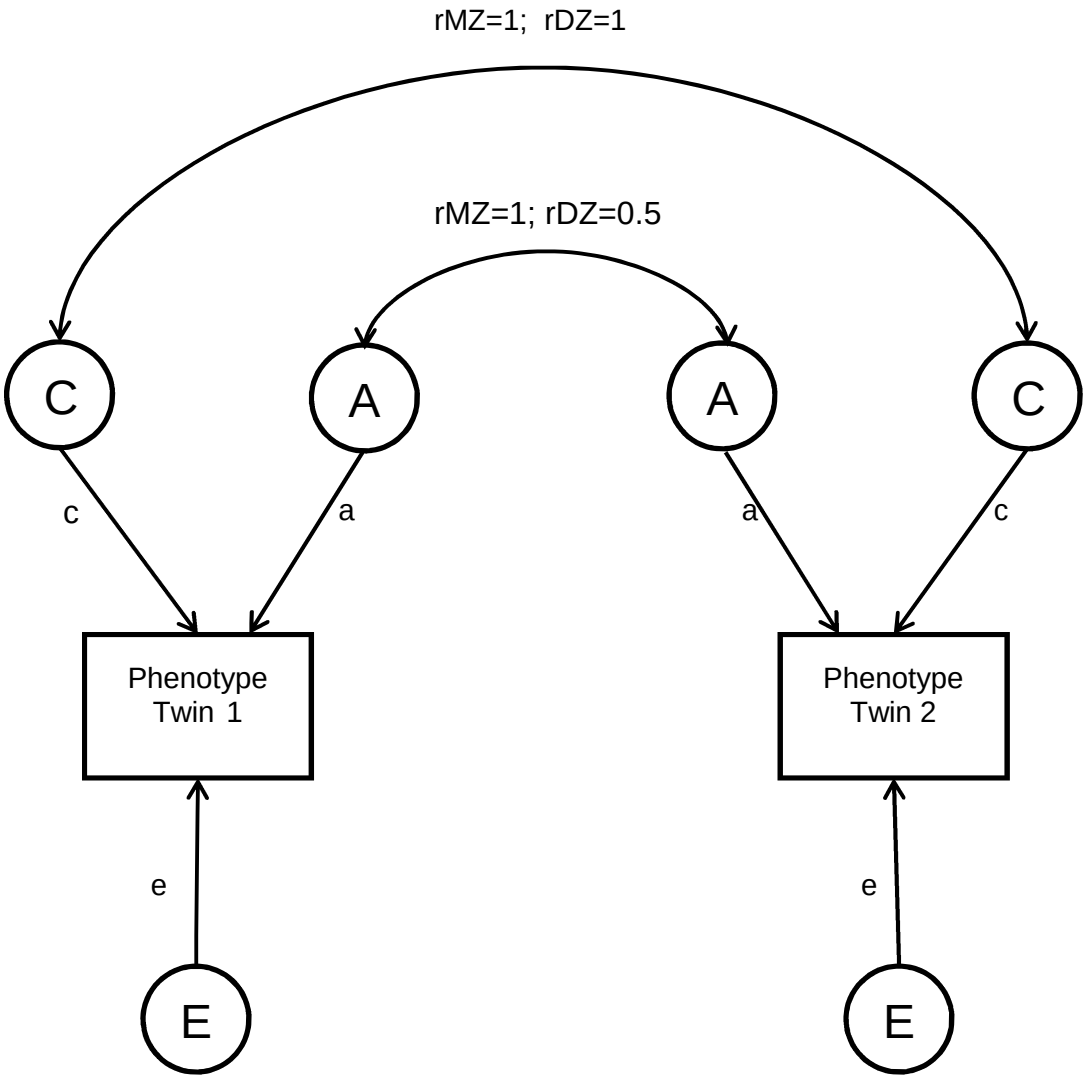


**Figure 3.2 Path diagram representing the relationships between the latent factors A, C, and E for MZ and DZ twins**

*Observed phenotypes for twin 1 and twin 2 are represented in rectangular boxes. Latent variables A (additive genetics effects, heritability), C (shared environmental effects) and E (non-shared environmental effects) are displayed in the circles. The single headed arrows indicate causal pathways from latent factors, denoted as 'a', 'c' and 'e'. Double headed arrows show the correlation between the latent factors between the two twins. These correlations are determined by the zygosity of the twin pair. Identical twins share all of their genetic and shared-environmental factors, whereas non-identical twins only share half of their genetic correlations. Non-shared environmental factors are unique to each twin are therefore not correlated between the two twins.*

Wright's rules of path analyses (Wright, 1920), allow us to estimate the predicted variance and covariance. To derive the direct effect of a latent factor on the variance of an observed phenotype, the coefficients of the latent factors need to be squared. The total variation of a phenotype (V) is therefore accounted for by the sum of the squared effects of the three latent factors ($V = a^2 + c^2 + e^2$). To calculate the covariance between twin 1 and twin 2, all paths connecting the two twins need to be considered. Due to the difference in genetic relatedness, these calculations for the covariances are different for MZ and DZ twin pairs. The path linking A factors in MZ pairs is calculated by following the connecting path between the two twins, ($a * 1 * a = a^2$), whereas for DZ twins the genetic covariance is calculated as follows: ($a * 0.5 * a = 0.5 * a^2$). The total covariance between twin 1 and twin 2 is explained by all connected factors and can be described in the following matrices. Matrices on the diagonal are total variances and matrices on the off-diagonal are covariances.

Variance/Covariance DZ =

$$\begin{bmatrix} a^2 + c^2 + e^2 & 0.5\,a^2 + c^2 \\ 0.5\,a^2 + c^2 & a^2 + c^2 + e^2 \end{bmatrix}$$

Variance/Covariance MZ =

$$\begin{bmatrix} a^2 + c^2 + e^2 & a^2 + c^2 \\ a^2 + c^2 & a^2 + c^2 + e^2 \end{bmatrix}$$

Maximum likelihood structural equation modelling is used to estimate the size of the effects for each of the latent factors A, C and E.

### 3.5.4 Maximum likelihood structural equation modelling

Maximum Likelihood Structural Equation Modelling (MLSEM) is commonly used to analyse twin data as it provides reliable estimates of A, C and E with 95% confidence intervals and goodness-of-fit statistics. In this thesis MLSEM was carried out using OpenMx software version 32 (Virginia Commonwealth University, Richmond, VA), a software package designed for R (Team, 2013). Initially a saturated model is 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, and subsequent submodels. The twin model of interest is fitted and compared against the saturated model, and more parsimonious submodels can also be tested by constraining parameters to zero and examining the goodness-of-fit of the submodel against the full model. It is important to note that non-shared environmental factors cannot be excluded as they include measurement error. The best fitting model is selected using goodness-of-fit statistics provided by OpenMx. Firstly, the change in minus twice the log-likelihood (-2LL) between two models is compared, which is very similar to a $\chi^2$ test, with difference in number of parameters being the number of allowed degrees of freedom. Additionally the Bayesian Information Criterion (BIC) (Raftery, 1995) is considered as an indicator for model fit in this thesis. The BIC is not a statistical test but can be used to aid model selection, as the best-fitting model and most parsimonious model is indicated by the lowest negative BIC score. The BIC is calculated as follows: $BIC = \chi^2 - (df \times ln\ (n))$ (n = total number of pairs). The BIC takes into account the complexity of the proposed models (i.e. the number of parameters in the model) as well as the sample size and can be used to compare models that are not nested within one another. When comparing the BIC of two models, a difference of 2-6 indicates some support for one model over the other. A difference greater than 10 provides very strong support for choosing the more parsimonious model with the lowest negative BIC score (Raftery, 1995).

### 3.5.5 Extensions of the classic twin model

The classic twin model described above has been extended to answer more complex research questions. The following models have been used in this thesis and present some of the extensions that have been added to the classic model since its original inception. Presented models are: a bivariate model; a model that tests for sex differences; a threshold model that uses categorical data; and a model that tests for the presence of gene-environment interaction.

#### 3.5.5.1 Bivariate Cholesky ACE Model

The standard, univariate twin model can be expanded to include two variables measured for each twin. This bivariate approach is used in Study 1 and Study 2, described in **Chapter 4 and Chapter 5**. The bivariate twin model estimates the effects of the latent factors A, C and E to variation in both traits individually, but in addition allows estimation of the extent to which genetic and environmental contributions are shared between the two phenotypes. In keeping with the univariate model, the twin

correlation for MZ and DZ pairs can be considered to give an indication of underlying genetic and environmental effects. For univariate analyses, the rMZ and rDZ for one phenotype are examined; but for bivariate analyses, cross-twin cross-trait correlations are examined instead, whereby trait 1 in twin 1 is correlated with trait 2 in twin 2 (and vice versa) for MZ and DZ pairs (see **Table 3.8)**. Again, if the cross-twin cross-trait correlations are higher for MZ than for DZ twins, genetic effects can be assumed to underlie the covariation between the two phenotypes. On the other hand, if the cross-twin cross-trait correlations are similar for both MZ and DZ pairs, shared environmental effects are contributing to the covariation between the two traits.

**Table 3.12: Variance/Covariance matrix for within-trait and cross-trait correlations within and across twin pairs underlying the bivariate twin model**

| | | **Twin 1** | | **Twin 2** | |
|---|---|---|---|---|---|
| | | **Phenotype 1** | **Phenotype 2** | **Phenotype 1** | **Phenotype 2** |
| **Twin 1** | **Phenotype 1** | 1 | | | |
| | **Phenotype 2** | Cross-trait Within-twin | 1 | | |
| **Twin 2** | **Phenotype 1** | Within-trait Cross-twin Phenotype 1 | Cross-trait Cross-twin | 1 | |
| | **Phenotype 2** | Cross-trait Cross-twin | Within-trait Cross-twin Phenotype 2 | Cross-trait Within-twin | 1 |

In keeping with the univariate ACE path analysis, the bivariate ACE model estimates the extent of common latent factors A, C and E underlying the two phenotypes, and the extent to which these common latent factors contribute towards the phenotypic correlation between them. This bivariate Cholesky ACE model is shown in **Figure 3.3**, representing the expected the variances and covariances for two hypothetical phenotypes.

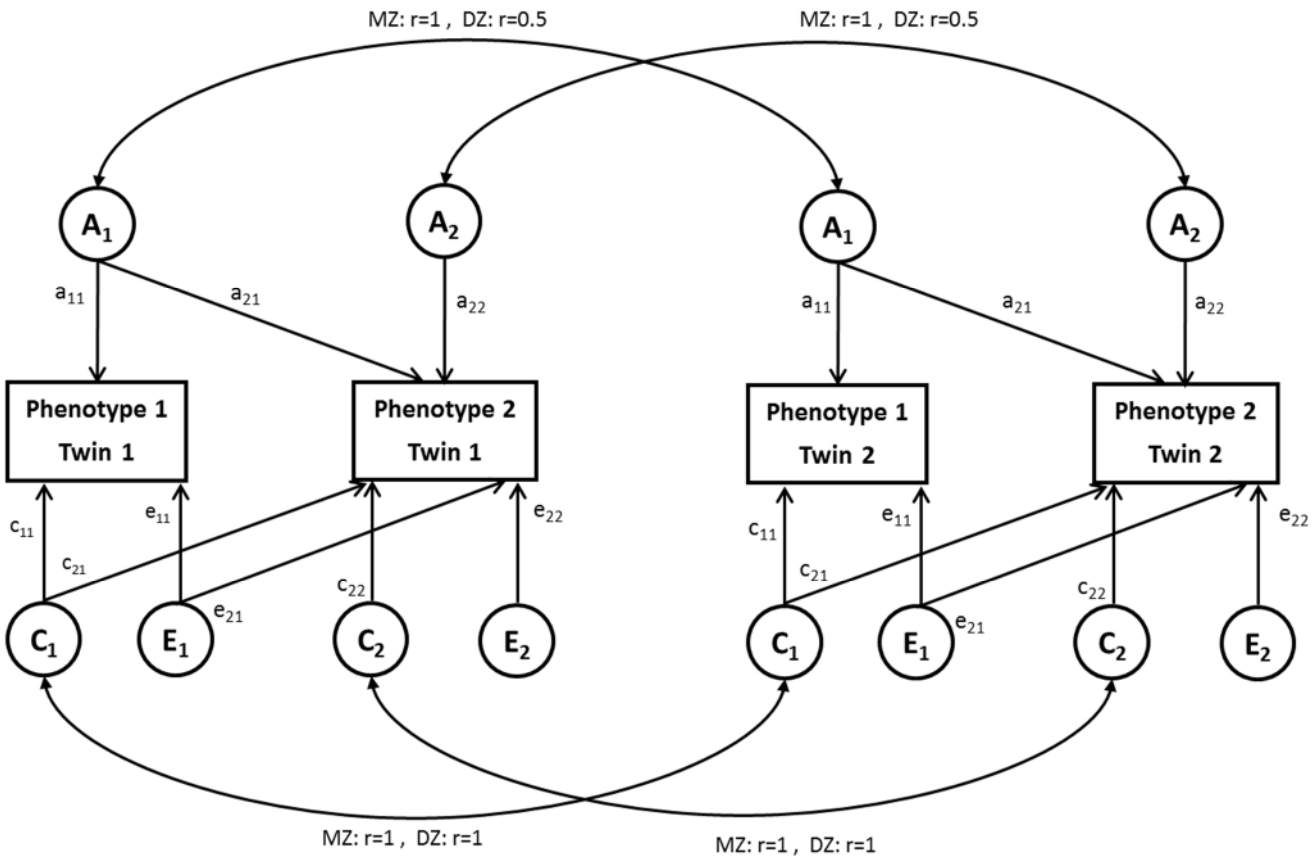


**Figure 3.3 Bivariate Cholesky ACE model of expected variance and covariance of two observed phenotypes**

*Latent factors A, C and E are represented in circles, and are specific to phenotype 1 ($A_1$, $C_1$, $E_1$) and phenotype 2 ($A_2$, $C_2$, $E_2$). Path coefficients (single headed arrows) are either unique to phenotype 1 ($a_{11}$, $c_{11}$, $e_{11}$) and phenotype 2 ($a_{22}$, $c_{22}$, $e_{22}$) or indicate the effect of latent factors contributing to phenotype 1 on phenotype 2 ($a_{21}$, $c_{21}$, $e_{21}$). As before the correlations between A and C are determined by the zygosity of the twin pair. MZ twins share all of their genes and shared environments (r = 1), whereas DZ twins only share half (r = 0.5) of their additive genetic effects but all of their shared environmental effects (r = 1).*

Following path tracing rules, the predicted variance and covariance for the two phenotypes can be estimated. For example, the genetic effects (A) on phenotype 1 are calculated using different coefficients of relatedness for MZs and DZs, as in the univariate model: MZ twins, [a11 × 1 × a11] = $a11^2$; DZ twins [a11 × 0.5 × a11] = $0.5a11^2$. In the Bivariate Cholesky ACE model, the variance in phenotype 2 is explained by residual latent factors (A2, C2, and E2) that are independent of A1, C1 and E1. Following all available paths genetic effects underlying phenotype 2 are also calculated using different coefficients of relatedness for MZs and DZs: MZ twins, [a21 × 1 × a21] + [a22 × 1 × a22] = $a21^2 + a22^2$; DZ twins, [a21 × 0.5 × a21] + [a22 × 0.5 × a22] = $0.5a21^2 + 0.5a22^2$. The same principle is applied to estimate the paths for the latent factors C and E. The paths a21, c21 e12 indicate the effect of A1, C1 and E1 on phenotype 2. In order to estimate their effect a21, c21 and e21 need to be square following path tracing rules [a21 x 1 x a21] = $a21^2$. **Table 3.9** depicts expected variances and covariances for the Bivariate Cholesky ACE model.

**Table 3.13 The bivariate Cholesky ACE Model, expected variance/covariance for MZ and DZ twins**

| **MZ Twins** | | **Twin 1** | | **Twin 2** | |
|---|---|---|---|---|---|
| | | **Phenotype 1** | **Phenotype 2** | **Phenotype 1** | **Phenotype 2** |
| **Twin 1** | **Phenotype 1** | $a_{11}+ c_{11}+ e_{11}$ | | $a_{11}^2+c_{11}^2$ | |
| | **Phenotype 2** | $a_{11}a_{21}+c_{11}c_{21}+e_{11}e_{21}$ | $a_{21}^2+a_{22}^2+c_{21}^2 +c_{22}^2+c_{21}^2+e_{22}^2$ | $a_{11}a_{21}+c_{11}c_{21}$ | $a_{21}^2+a_{22}^2+c_{21}^2+c_{22}^2$ |
| **Twin 2** | **Phenotype 1** | $a_{11}^2+c_{11}^2$ | | $a_{11}+ c_{11}+ e_{11}$ | |
| | **Phenotype 2** | $a_{11}a_{21}+c_{11}c_{21}$ | $a_{21}^2+a_{22}^2+c_{21}^2+c_{22}^2$ | $a_{11}a_{21}+c_{11}c_{21}+e_{11}e_{21}$ | $a_{21}^2+a_{22}^2+c_{21}^2 +c_{22}^2+c_{21}^2+e_{22}^2$ |
| **DZ Twins** | | **Twin 1** | | **Twin 2** | |
| | | **Phenotype 1** | **Phenotype 2** | **Phenotype 1** | **Phenotype 2** |
| **Twin 1** | **Phenotype 1** | $a_{11}+ c_{11}+ e_{11}$ | | $0.5a_{11}^2+c_{11}^2$ | |
| | **Phenotype 2** | $a_{11}a_{21}+c_{11}c_{21}+e_{11}e_{21}$ | $a_{21}^2+a_{22}^2+c_{21}^2 +c_{22}^2+c_{21}^2+e_{22}^2$ | $0.5a_{11}a_{21}+c_{11}c_{21}$ | $0.5a_{21}^2+0.5a_{22}^2+c_{21}^2+c_{22}^2$ |
| **Twin 2** | **Phenotype 1** | $0.5a_{11}^2+c_{11}^2$ | | $a_{11}+ c_{11}+ e_{11}$ | |
| | **Phenotype 2** | $0.5a_{11}a_{21}+c_{11}c_{21}$ | $0.5a_{21}^2+0.5a_{22}^2+c_{21}^2+c_{22}^2$ | $a_{11}a_{21}+c_{11}c_{21}+e_{11}e_{21}$ | $a_{21}^2+a_{22}^2+c_{21}^2 +c_{22}^2+c_{21}^2+e_{22}^2$ |

#### 3.5.5.2 The bivariate correlated factors model

In the bivariate Cholesky ACE model the order of the two measured phenotypes is crucial, as variation in phenotype 2 is always explained by residuals of the latent factors underlying phenotype 1. Often a causal order between two phenotypes cannot be established (e.g. with cross-sectional data), and the bivariate Cholesky ACE model cannot be applied. In the case that a causal relationship of the variables is unknown, the Cholesky ACE model can be transformed into the correlated factors model (Loehlin, 1996). The Correlated Factors Model is in effect a standardised version of the bivariate Cholesky ACE model, where the order of the two variables is irrelevant, and arrows between latent factors are bi-directional. A path diagram of the Correlated Factors Model is displayed in **Figure 3.4**.

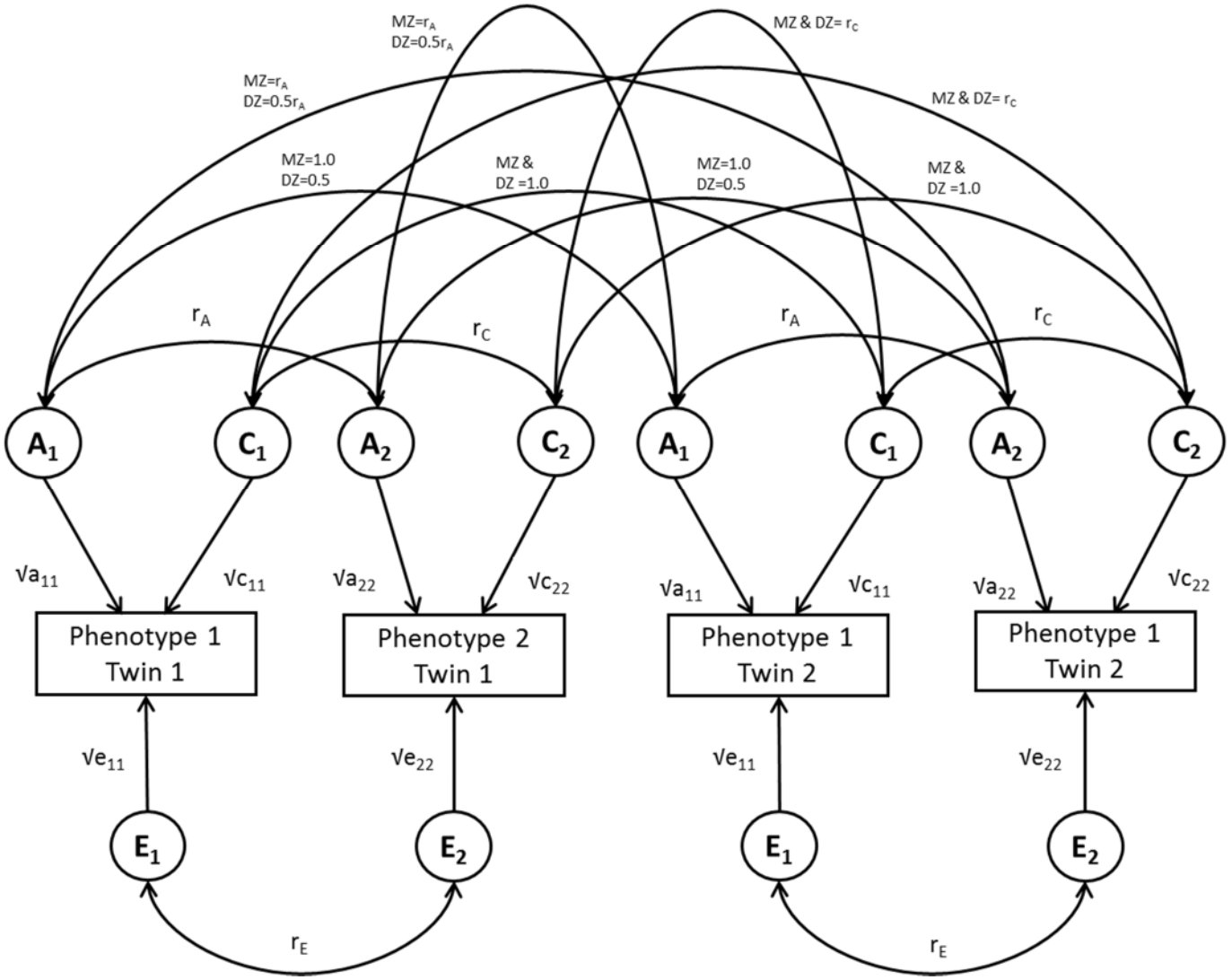


**Figure 3.4: Bivariate Correlated Factors Model for two twins.**

*Observed variables are shown in rectangles, latent factors in circles, separated for the two phenotypes ($A_1$, $C_1$, $E_1$, $a_{11}$, $c_{11}$, $e_{11}$ and $A_2$, $C_2$, $E_2$, $a_{22}$, $c_{22}$, $e_{22}$). Due to its standardised nature, the square root path coefficients (represented by single headed arrows) indicate variance explained by latent factors. Double headed arrows represent correlations that differ for MZ and DZ twins. The aetiological correlations indicate the extent to which the latent factors are shared between the two variables ($r_A$, $r_C$, $r_E$).*

The correlated factors model provides: (i) univariate estimates of the effect of A, C and E on the two phenotypes; and (ii) aetiological correlations ($r_A$, $r_C$, $r_E$) indicating the extent to which the A, C and E for each phenotype are shared. Similar to standard Pearson's correlations, aetiological correlations range from -1 to 1. A high positive aetiological correlation indicates that a large proportion of the latent factors underlying phenotype 1 also affect phenotype 2, whereas a low aetiological correlation would imply that the effects of the latent factors are specific to each phenotype. As with Pearson's correlations, the correlation coefficient needs to be squared to indicate variance explained. For example, a $r_A = 0.5$ indicates that 25% of the latent genetic factors for both phenotypes are the same.

Additionally bivariate estimates (BivA, BivC, BivE) decompose the phenotypic correlation of the two phenotypes into the effects of A, C and E. The bivariate estimate for A is derived by: (i) multiplying the square-root of the heritability of phenotype 1 ($\sqrt{a_1^2}$) (with the heritability of phenotype 2 ($\sqrt{a_2^2}$); and (ii) multiplying this product by the genetic correlation between the two phenotypes ($r_A$). The same procedure is used for BivC and BivE.

$$BivA = \sqrt{a_1^2} \times r_A \times \sqrt{a_2^2}$$

$$BivC = \sqrt{c_1^2} \times r_C \times \sqrt{c_2^2}$$

$$BivE = \sqrt{e_1^2} \times r_E \times \sqrt{e_2^2}$$

If all bivariate estimates are of the same sign (negative or positive), they can be expressed as direct proportions of the phenotypic correlation between the two variables. As an example: Two phenotypes are found to correlate positively r = 0.5. All bivariate estimates were found to be positive; together BivA = 0.25, BivC = 0.125, BivE = 0.125 add up to the overall phenotypic correlations (r = 0.5). In order to calculate the contributions of genetic effects on the phenotypic correlation, the BivA estimate needs to be divided by the phenotypic correlation: BivA / r. In this example that would be 0.25 / 0.5 = 0.5, which infers that 50% of the phenotypic correlation was due to genetic effects. Following this example for BivC and BivE of 0.125, indicate that 25% (0.125 / 0.5) of the phenotypic correlation were due to shared and non-shared environmental factors each. Together these proportion add up to 100%, the same way as BivA + BivC + BivE = phenotypic correlation.

### 3.5.5.3 Univariate sex differences

The classic univariate twin model can be extended to test for differences between males and females. It is theoretically possible that the underlying aetiology of a measured phenotype differs in two distinct ways between males and females. First, 'qualitative' sex differences imply that the genes that influence a phenotype in females are different to those that influence the same phenotype in males. As an example, sex limitation models suggested that there are different genes accounting for variance in BMI in women than in men. These findings were seen as an indicator that there might sex specific genetic factors that influence sex differences in fat distribution and deposition (Schousboe et al., 2003). Secondly, 'quantitative' sex differences imply that even if the genetic and environmental influences on a particular phenotype are the same for males in females, they have a different effect size for males and females. As an example the heritability of depression symptoms was estimated in a population of adult twins in Sri Lanka. Heritability estimates were found to differ between the sexes, but they were not qualitatively different. It was suggested that men and women are exposed to very different environmental exposures, which changes the relative contribution of genetic and environmental influences on depression in this specific population (Ball et al., 2009). Of course, it is also possible for both quantitative and qualitative sex differences to occur at the same time.

Testing for potential sex differences is important, as combining males and female twins in one sample can mask the different effects underlying variation in males and females. To test for the presence of sex differences in genetic and environmental influences, the sample is split into same-sex pairs (MZ males [MZM], DZ males [DZM], MZ females [MZF], DZ females [DZF]) and DZ pairs of opposite sex [DZOS]). A comparison of the twin pair similarity (twin correlations) between the different types of twin pairs can be used to give a first indication of the presence of sex differences. If the opposite-sex DZ twin correlation is much smaller than the correlation of same-sex DZ twins, potential 'qualitative' sex differences can be considered. Qualitative sex differences imply that genetic effects underlying variation are different between males and females. Therefore, opposite-sex DZ twin pairs are bound to show less similarity than same-sex DZ pairs for this phenotype. In comparison, a different MZ:DZ correlation ratio between male same-sex pairs and female same-sex pairs, with DZOS falling in between, indicates the presence of 'quantitative' sex differences.

To test for the presence of sex differences a number of consecutive models are tested against each other. A full sex limitation model for one phenotype is depicted in **Figure 3.5**.

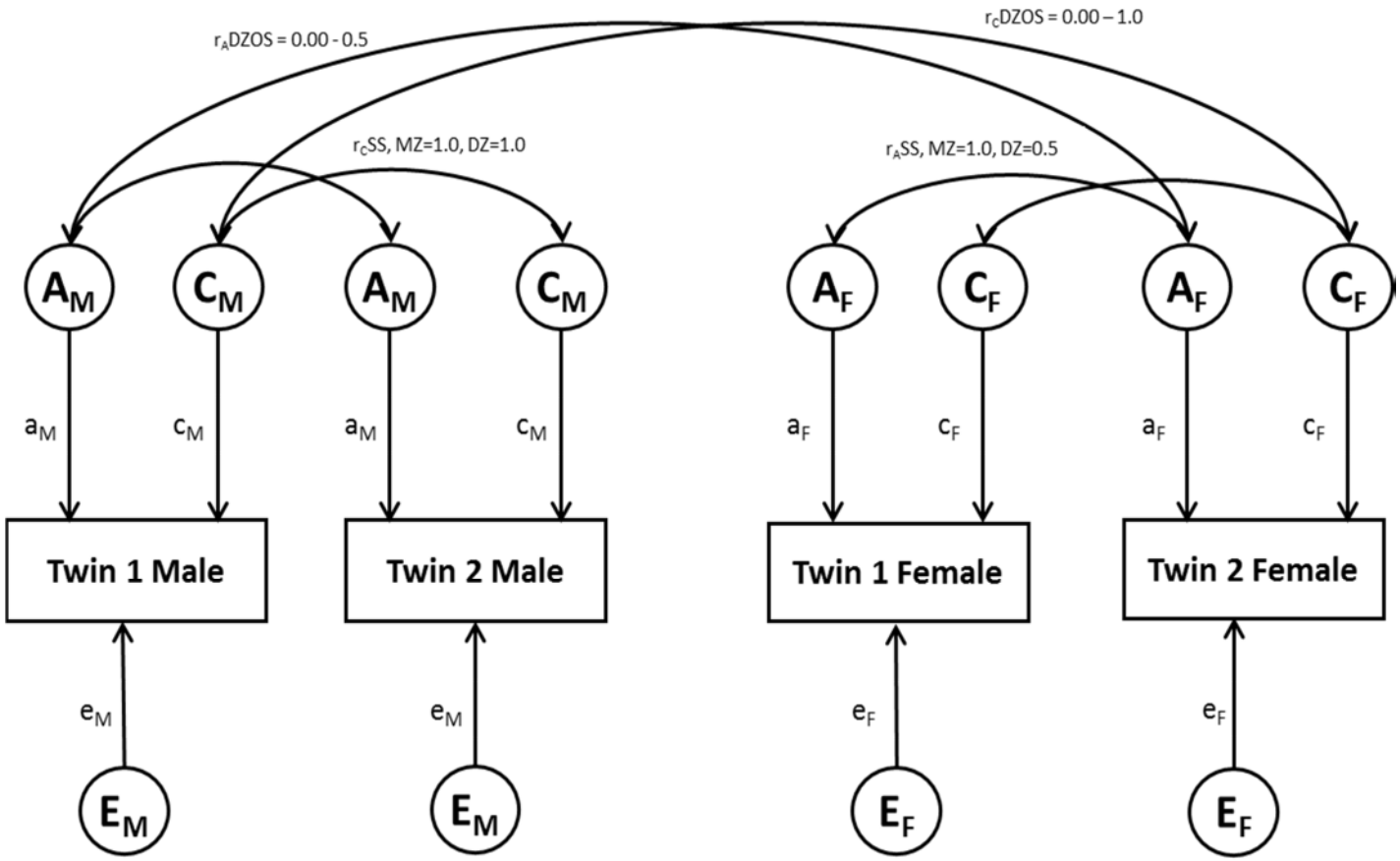


**Figure 3.5: Univariate full sex-limitation model**

*Latent factors in circles and measured phenotypes in rectangular boxes. Single headed arrows represent path coefficients for males ($a_m$, $c_m$, $e_m$) and females ($a_f$, $c_f$, $e_f$) separately. Within same-sex (SS) pairs genetic and shared-environmental correlations are dependent on the zygosity of the twin pair, MZ or DZ. In opposite-sex (OS) twin pairs the genetic ($r_A$) and shared-environmental ($r_C$) correlation may be allowed to vary to test for the presence of sex differences.*

Parameters included in the univariate sex-limitation model, including opposite sex twin pairs, are then constrained to test for the presence of sex differences. Genetic and shared-environmental correlations within opposite sex twin pairs are fixed to their theoretical values ($r_A$ = 0.5, $r_C$ = 1.0) and tested against the full model allowing the parameters to be free. Two separate models are needed, one fixing $r_A$ = 0.5 and leaving $r_C$ free. The second model leaves $r_A$ free to estimate, and fixes $r_C$ to 1. This is necessary as is not possible to leave both parameters free to estimate; it would render the model unidentified. Models estimating a significantly lower $r_A$, than their theoretical 0.5 indicate the presence of qualitative genetic sex differences; and models estimating a significantly lower $r_C$, than their theoretical 1.0 indicate the presence of qualitative shared environmental sex differences. Then, a model which fixes both $r_A$ and $r_C$ to their theoretical values for opposite-sex DZ twins (A, C and E estimates are allowed to vary for males and females, called the 'common effects' model). This common effects model tests for the presence quantitative sex differences.

After the 'common effects model' is tested, a so-called 'scalar model' is fitted to the data. In this model, the presence of underlying variance differences between males and females is tested. The model assumes that the path estimates for females are the same as the estimates for males as a function of the scalar. The value of the scalar indicates how the size of the variance differs between males and females. A scalar estimated to be close to one indicates no significant effects (i.e. no variance differences between the sexes). The final model (null model – 'homogeneity model') tests for the presence of any sex differences by fitting a reduced model equating all parameters estimates for males and females. This model includes fewer parameters and implies that the aetiology of the phenotype is exactly the same for males and females (no qualitative or quantitative sex differences, and no variance differences between males and females). A superior model fit of the null model implies the absence of any differences between males and females (Michael C. Neale & Cardon, 2010).

#### 3.5.5.4 Bivariate sex differences

The univariate sex limitation model can be extended into a bivariate model, including two phenotypes. As an extension to the model in **Chapter 3.5.5.3** this model provides additional information to the univariate estimates for A, C and E. The bivariate sex limitation models allow researchers to test for differences between the phenotypic

correlations for males and females, as well as establish the presence of sex differences in the underlying aetiological correlations between the latent factors.

Extending the univariate sex limitation model to include two phenotypes is not straightforward. Exactly as in the univariate sex limitation model, MZ and same-sex and opposite-sex DZ pairs are included to test for qualitative and quantitative sex differences. However, applying a Cholesky decomposition (see **Figure 3.6**) to model bivariate sex limitation can produce different model fit statistics, depending on the order of the phenotypes entered into the model. In the Cholesky decomposition specification, the effect of latent factors is partitioned into factors shared between phenotype 1 and 2, and factors specific to phenotype 2. The order of the phenotypes should be random when using cross-sectional data and does not pose a problem when using same-sex MZ and DZ twins. However, to test for the presence of scalar sex differences opposite-sex DZ twin pairs are needed. Scalar sex differences indicate that the same genetic, shared and non-shared environmental factors affect variance in males and females, but they do so to a different extent. Using a cholesky decomposition the order of the variables can introduce differences in the model fit and different estimates for A, C and E. Therefore, a correlational approach should always be taken when running bivariate sex limitation models (M. C. Neale, Roysamb, & Jacobson, 2006). In this model, including opposite-sex twin pairs, the aetiological correlations between the two variables across the sexes are constrained to their theoretical values of 1 ($r_C$) and 0.5 ($r_A$). Just in the univariate sex limitation models, this process is conducted in two stages, first constraining $r_C = 1$ and leaving $r_A$ to be estimated freely. Consecutively, in a separate model $r_A$ is constrained to 0.5, with $r_C$ being free to vary. Then in a third stage, both are constrained to their theoretical values ($r_A = 0.5$ and $r_C = 1$) (common effects model). In a fourth model, variances are allowed to differ between males and females. In the final model, the null model, parameter estimates for males and females are equated to be the same indicating no sex differences.

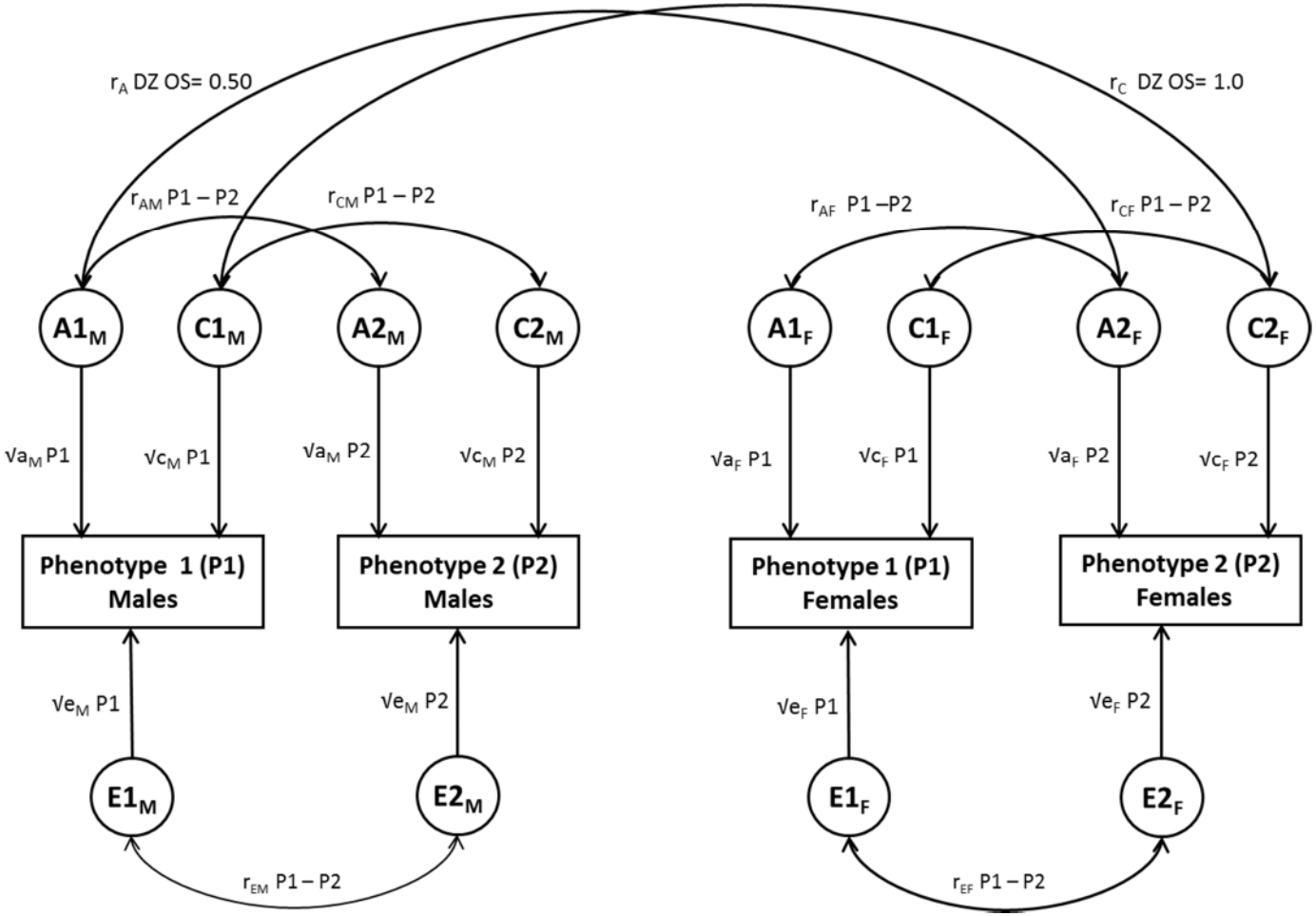


**Figure 3.6 Path diagram illustrating bivariate sex limitation model including opposite-sex twin pairs.**

*Subscript M stands for male, subscript F for female. Observed phenotypes for males and females are displayed in rectangular boxes, latent factors A, C and E for males and females for phenotype 1 and 2 are displayed in circles. Single arrows represent path estimates indicating variance explained of the corresponding latent factor; double headed arrows present aetiological correlations between latent factors, split by same-sex and opposite-sex twin pairs. $r_A$ and $r_C$ for opposite-sex twin pairs are constrained to their theoretical values of 0.5 and 1, respectively, to test for the presence of qualitative sex differences.*

### 3.5.5.5 Liability threshold models

The primary outcome variables of this thesis are childhood EOE and EUE. EOE has been found to be less common in childhood, with some children rated not to engage in this behaviour. Liability threshold models are used to analyse categorical (e.g. presence or absence of a disease) or ordinal data, and can also be applied to data with a skewed distribution, given participants are often categorised instead (e.g. 1 = engage in EOE; 0 = never engage in EOE). In comparison to the models explained above, in Liability threshold models, concordance rates for twin pairs, split by zygosity, can be used to produce a first estimation of the importance of genes and environments. The logic behind this is illustrated in **Table 3.10**. A large MZ:DZ concordance ratio difference implies that genetic factors underlie the liability of the measured behaviour, whereas a similar MZ:DZ ratio implies that shared environmental factors are of importance here.

**Table 3.14 Contingency table underlying Liability threshold models**

| | **MZ/DZ Twins** | |
|---|---|---|
| **MZ/DZ Twins** | **Twin 1** | |
| **Twin 2** | **Affected** | **Unaffected** |
| **Affected** | Concordant affected | Discordant pair |
| **Unaffected** | Discordant pair | Concordant unaffected |

This joint distribution of liability, for twin 1 and twin 2, is assumed to follow a bivariate normal distribution with a mean of 0 and a standard deviation of 1. The proportions of affected and unaffected twins can be used to estimate thresholds and tetrachoric correlations, allowing path estimates to be calculated that indicate the effect of A, C and E.

#### 3.5.5.6 Gene-environment correlation

Gene-environment correlation describes the situation where exposure to an environmental factor is not random but correlated with the individual's genotype. In the literature three specific types of gene-environment correlation have been suggested: active, passive and evocative (Robert Plomin et al., 2013). Active gene-environment correlation describes the situation when an individual seeks out environments matching their genetic propensities. A child with the genetic propensity to develop strong reading skills might actively choose activities that involve reading such as going to the library or joining a book club. The obvious disposition to enjoy and be good at reading might then evoke responses from the environment as well. Described as an evocative gene-environment correlation, teachers might recognise that the child is a strong reader and will encourage their reading by recommending more difficult books, or spending extra time with them to support their accelerated development. Lastly, a passive gene-environment correlation occurs when children inherit not only the genes associated with reading but are also exposed to a family environment which reflects these propensities in the parents. For example, parents with strong reading ability might be passionate readers themselves, creating a home environment that corresponds to their own and their children's genotype – i.e. a house full of books, and much family time spent reading.

All three gene-environment correlations can impact the estimates derived from classic twin models. MZ twins are genetic clones and are therefore more likely to actively seek and evoke more similar exposures from their environments potentially inflating similarity within twin MZ twin pairs. 'Passive' gene-environment correlations are deemed to increase similarity across MZ and DZ twins equally, as both types of twins will inherit genes and home environment factors to the same extent. The classic twin design cannot unpick the presence of these gene-environment correlations; designs that incorporate environmental measures need to be used. Twin models including more than one phenotype (bivariate and multivariate) as well as longitudinal data can help to detect positive gene-environment correlations. In addition, comparisons of associations of family environmental factors and child behaviours between adoptive and non-adoptive families can test for passive gene-environment correlation by controlling for familial relatedness (Rijsdijk & Sham, 2002).

#### 3.5.5.7 Gene-environment interaction

In addition to gene-environment correlations, gene-environment interactions also contribute to the complex interplay between genetic and environmental factors underlying human individual differences. Gene-environment interactions are situations where the effect of a genotype on a phenotype is dependent on an environmental factor, or conversely, that the effect of an environmental factor on a phenotype is dependent on genotype (Plomin et al., 2013). Gene-environment interactions are likely to affect the aetiology of most complex traits, including the development of child emotional eating. The importance of gene–environment interactions have been detected in previous research, providing evidence for the effect of socio-economic status on the heritability of antisocial behaviour, with higher heritability being reported for twins from wealthier families (Tuvblad, Grann, & Lichtenstein, 2006), whereas physical activity has been found to dampen genetic influence on BMI in young adults (Mustelin, Silventoinen, Pietilainen, Rissanen, & Kaprio, 2009).

The classic twin design cannot detect gene-environment interactions and more complex models including measures of the environmental exposures need to be included. If the environmental factor is categorical this process is fairly straight forward as the sample can be divided in to two groups (exposed/unexposed, employed/unemployed, etc.) and estimates of A, C and E compared across the groups (similar to models of sex-differences). This approach allows comparison of the aetiology of one phenotype across two different environmental conditions, giving insight into possible gene-environment interactions. However often environmental factors are not binary, and forcing a continuous variable into artificial categories can be problematic as it assumes equal variances across the two groups, leads to reduced sample sizes in each group, and a great deal of information is lost; reducing power (Briley, Harden, Bates, & Tucker-Drob, 2015). To include continuous environmental exposures, continuous moderator twin models have been developed. Here the measured continuous environmental factor is added to classical twin model to test if estimates of genetic and environmental influences decrease or increase as a linear function of the moderator. Hypothetically, the effect of the environment will not only influence the contribution of A, C and E but also the phenotype itself. This measured environmental factor can therefore be added to the classic twin model,

impacting on the means, variance/covariance structure as well as estimated paths. The continuous moderator model is illustrated in **Figure 3.7**.

This model is used in Study 5 **Chapter 8**, to test the moderating effect of household stress on the aetiology of EOE and EUE.

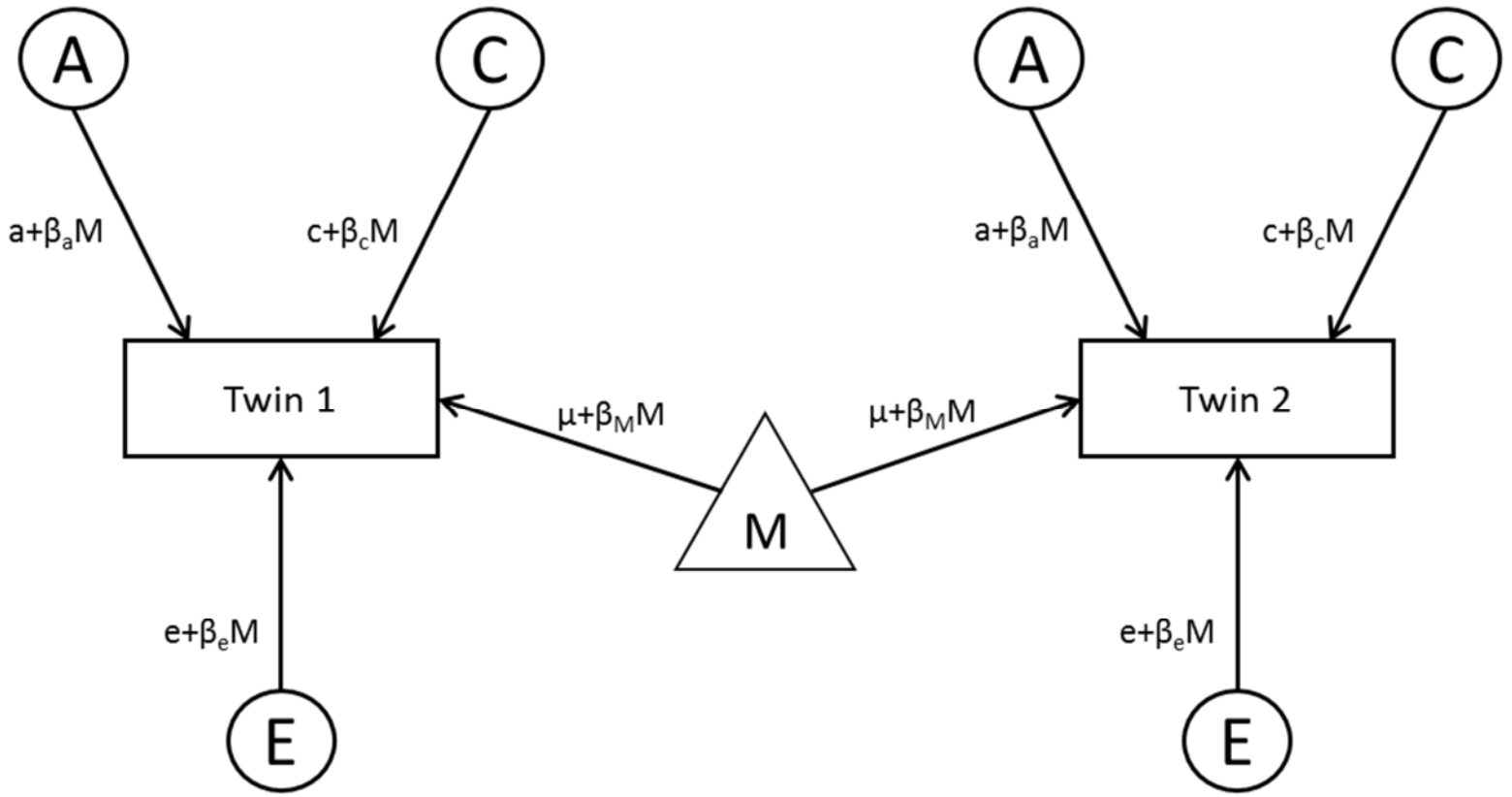


**Figure 3.7 Gene-Environment Interaction Twin Model for one pair of twins.**

*Latent factors are represented in circles (A, C and E) and measured phenotype in rectangular boxes. The moderating factor (M) is presented in a triangle in the centre, affecting the measured phenotype and the paths (single headed arrows) from latent factors to phenotype.*

This model calculates unstandardized and unmoderated path estimates for A, C and E ($a_{Un}$, $c_{Un}$, $e_{Un}$) in the absence of the moderator variable, creating reference estimates. Furthermore, effects of the moderator on the mean ($\beta_M$) and the latent factors ($\beta_a$, $\beta_c$, $\beta_e$) are estimated. Significant moderation is indicated by significant $\beta$s with confidence intervals not crossing zero. The values of the $\beta$s represent the change in the effect of the latent factor (A, C and E) for a one unit increase of the moderator.

In order to illustrate the effect of the moderator, unstandardized variance explained by A, C and E (Va, Vc and Ve) are calculated for each level of the moderator using the following equations:

$$Va = (a_{Un} + \beta a \times \text{Level of Moderator})^2$$

$$Vc = (c_{Un} + \beta c \times \text{Level of Moderator})^2$$

$$Ve = (e_{Un} + \beta e \times \text{Level of Moderator})^2$$

Adding Va, Vc and Ve together equals the total variance at each given level of the moderator (Va + Vc + Ve = Vt).These calculated estimates can then be used to plot the change in variance explained by A, C and E across the different levels of moderation.

In comparison to the previously discussed models, the continuous moderator model testing for gene-environment interaction does not necessarily assume normally distributed data. Data that are negatively or positively skewed might be a consequence of the underlying gene-environment interaction. Therefore, transforming variables in order to adjust for skewness are not recommended in these models. However, the model is not able to distinguish between skew due to gene-environment interaction, or skew due to poor measurement quality resulting in non-normal distribution. This is one of the major downfalls of this model (Purcell, 2002).

More recently, new approaches incorporating Item Response Theory into gene-environment interaction modelling have been proposed in order to address these issues regarding skewed data (Murray, Molenaar, Johnson, & Krueger, 2016). A more detailed discussion of this can be found in **Chapter 8.5** – in context of the data analysed in this study.

### 3.5.6 Statistical power

The statistical power of twin studies is dependent on various factors: the sample size, the ratio of MZ:DZ twin pairs, the expected effect of genetic, shared and non-shared environmental factors, the number of variables included (univariate, bivariate and multivariate) and the type of data that is being analysed (continuous versus categorical data). The Gemini study started with over 2400 twin pairs at baseline and over 1000 pairs were still enrolled when the twins were five years. Verhulst (2017) addressed statistical power of twin studies by modelling statistical power in different scenarios of heritability and effect from the shared environments (Verhulst, 2017). **Figure 3.8** (adapted from Verhulst, 2017), indicated that with a sample greater than 1000 twin pairs provides good power (> 90%) to detect significant parameters under all modelled scenarios. Greatest power was achieved at moderate heritability (A = 33%) and high shared environmental effects (C = 50%) (Verhulst, 2017).

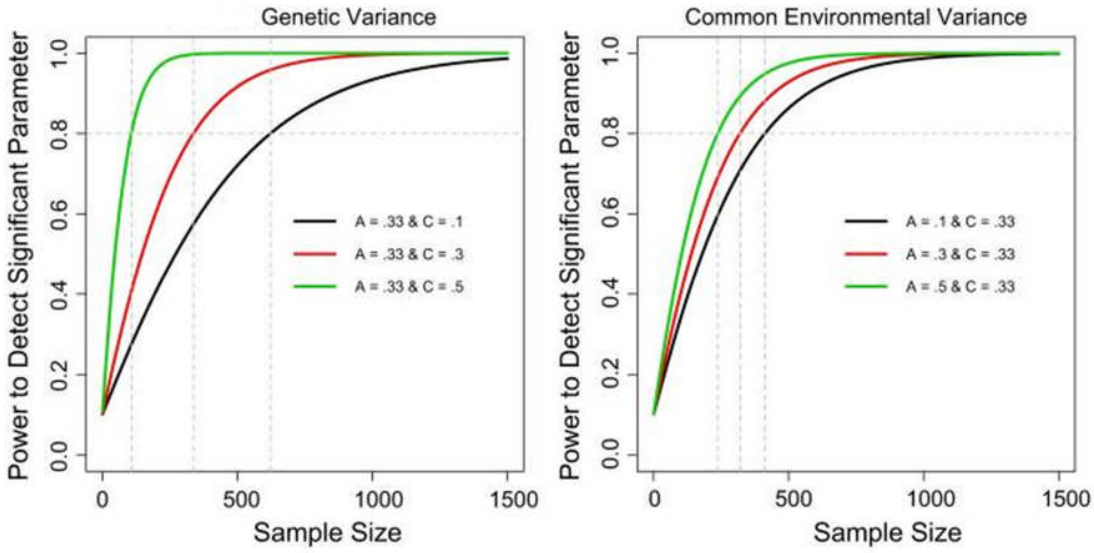


**Figure 3.8 Power to detect significant parameters in twin studies at different levels of heritability and shared environmental effects**

Figure adapted from Verhulst (2017)

Moreover, the effect of the MZ:DZ ratio on the statistical power of twins studies was investigated. At baseline the Gemini sample consisted of 749 MZ twins and 1616 DZ pairs, roughly a 1:2 MZ:DZ ratio. **Figure 3.9** (adapted from Verhulst, 2017) indicated that with a sample greater than 1000 twin pairs, studies are well power (> 90%) to detect significant genetic and shared environmental effects (Verhulst, 2017).

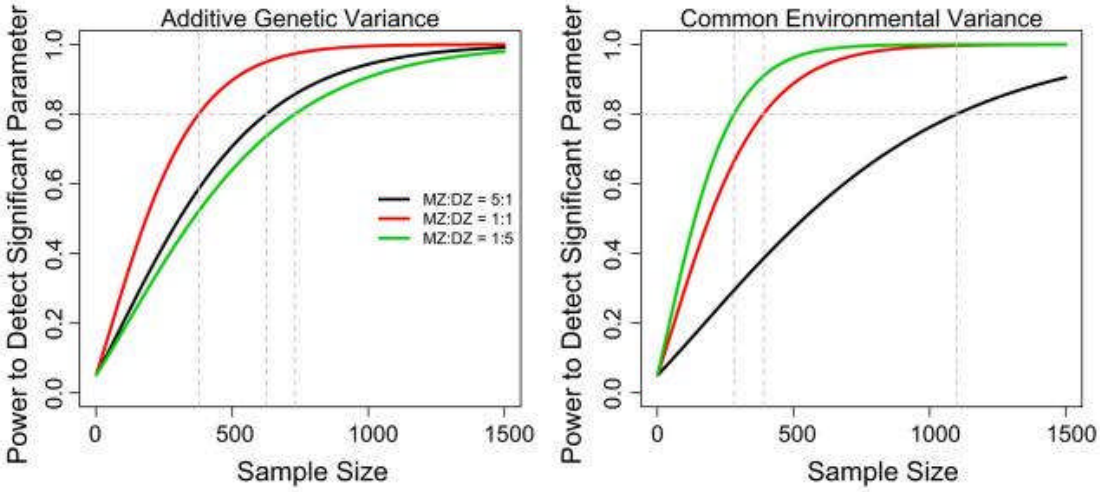


**Figure 3.9 Power to detect significant parameters in twin studies at different levels of MZ:DZ ration (5:1, 1:1, 1:5)**

Figure adapted from Verhulst (2017)

Moreover, it has been pointed out that analysing categorical data, using threshold models, the sample size needs to be increased to gain sufficient power. It has been suggested that roughly three times as many twin pairs are needed to conduct successful threshold models in comparison to continuous data. Power decreases even more if a measured behaviour is rare (M. C. Neale, Eaves, & Kendler, 1994). **Figure 3.10** (adapted from Verhulst, 2017) shows the statistical power to detect a significant parameters contemplating continuous data and binary data with different prevalence rates (50%, 40%, 30%, 20%, 10% and 5%) (Verhulst, 2017). The use to continuous data is of advantage and a sample of over 1000 twin pairs was seen at 99% to detect significant parameter estimates. In summary it can be concluded, that the sample size greater than 1000 twin pairs this thesis was statistically powered to detect significant parameters estimates for the effect of genetic and environmental factors.

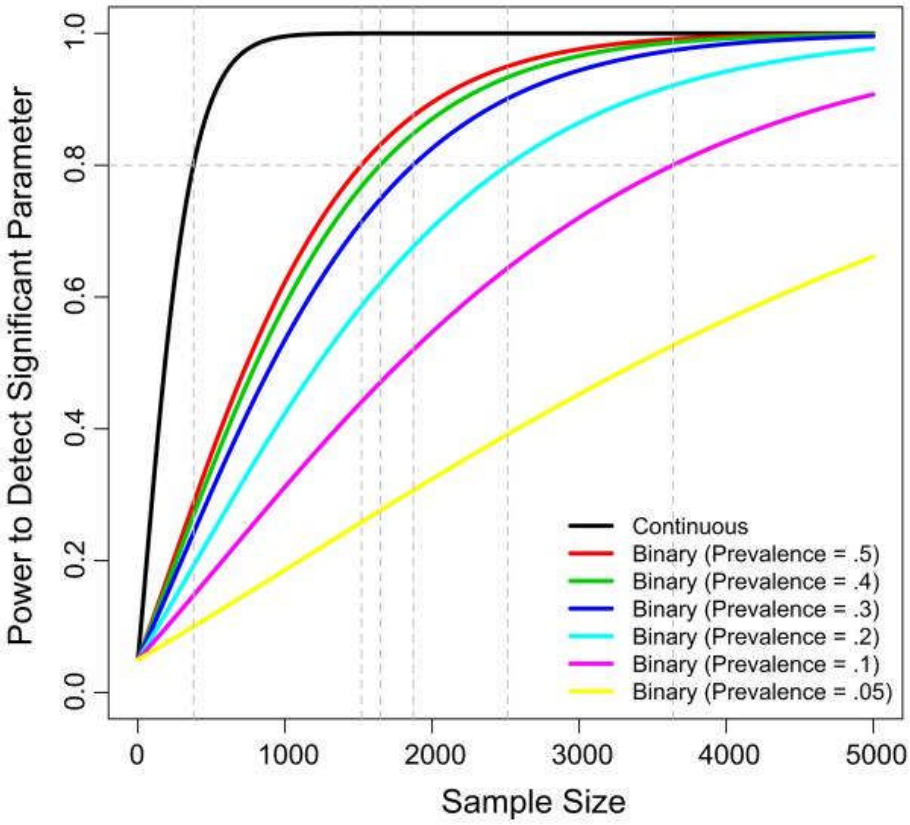


**Figure 3.10 Statistical power to detect significant parameter estimates using continuous versus categorical data with varying prevalence rates**

Figure adapted from Verhulst (2017)

### 3.5.7 Limitations of the twin method

The twin model has been shown to be a consistent and reliable research methodology. However, there are limitations.

#### 3.5.7.1 Representativeness of twins

In order to interpret findings from twin research, twins must be representative of the general population, i.e. singletons. Compared with age-matched singletons twins have been shown not to differ on various physical and behavioural traits, including: bone mineral density, blood pressure, alcohol and tobacco consumption (Andrew et al., 2001), personality traits (Johnson, Krueger, Bouchard, & McGue, 2002), and motor development (R. S. Wilson & Harpring, 1972). However twins are born earlier and have a lower birth weight, even taking into account their size at birth relative to their gestational age (van Dommelen, de Gunst, van der Vaart, van Buuren, & Boomsma, 2008). They experience 'catch up' growth after birth, reaching a similar size to singletons at around 2.5 years of age (Bleker, Breur, & Huidekoper, 1979; van Dommelen et al., 2008; R. S. Wilson, 1979). However, older twins mostly do not differ from singletons, and twin cohorts are therefore deemed representative.

#### 3.5.7.2 Violation of the equal environments assumption (EEA)

The EEA states that environmental exposures influencing the variation of a trait are unrelated to the zygosity of the twin pairs – i.e. that MZs and DZs share their environments to the same extent. A violation of the EEA could lead to an overestimation of the genetic contribution to variation if MZs, in fact, share their environments more closely than DZs. This is because the higher MZ correlation would reflect both increased shared environmental influences, as well as increased similarity in genetic relatedness, compared to the DZ correlation, rather than just increased genetic relatedness. Because heritability is estimated by doubling the difference between the MZ and DZ correlation, a higher MZ correlation due to greater environmental similarity would be masked as heritability.

Before birth MZ and DZ twins share the same prenatal environment and are exposed to the same environmental factors influencing the pregnant mother (Rijsdijk & Sham, 2002). Furthermore, both MZ and DZ pairs tend to grow up in the same family from birth until they leave home. However, the fact MZ twins look identical and are often perceived as more similar (by virtue of the fact that they are more similar on all genetically-determined

traits), has given rise to the claim that they might be treated more similarly by their parents in comparison to DZ twins (Felson, 2014a).

The EEA has been widely debated and still remains controversial (Fosse, Joseph, & Richardson, 2015). It has been pointed out as a fundamental flaw of the twin method, and poses a challenge to the validity of twin research (Joseph, 2013). However multiple attempts to test the potential violation of the EEA have been conducted and are discussed in detail below (Borkenau, Riemann, Angleitner, & Spinath, 2002; Conley, Rauscher, Dawes, Magnusson, & Siegal, 2013; Cronk et al., 2002; Felson, 2014a; Goodman & Stevenson, 1989; Hettema, Neale, & Kendler, 1995; Kendler, Neale, Kessler, Heath, & Eaves, 1993b; LoParo & Waldman, 2014; Morris-Yates, Andrews, Howie, & Henderson, 1990; Xian et al., 2000).

#### 3.5.7.3 Accounting for physical resemblance, environmental exposure and social contact

One way to test the validity of the EEA is to see if MZ twins are treated more similarly due to their physical resemblance. In order to adhere to the EEA these similarities should not be associated with the intraclass correlations of MZ and DZ twins. Multiple studies have investigated this notion, addressing physical similarity. Even though studies found evidence that MZ twins do look more similar, there were no associations found between physical resemblance and correlations on various traits such as eating attitudes, personality traits, intelligence and reading skills. In other words, no violations of the EEA were detected accounting for physical resemblance in MZ twins (Hettema et al., 1995; Klump, Holly, Iacono, McGue, & Willson, 2000; A. P. Matheny, Jr., Wilson, & Dolan, 1976; R. Plomin, Willerman, & Loehlin, 1976).

Other studies have examined if MZ twins are exposed to more similar environmental exposures than DZ twins. To do so twins rated their upbringing and other environmental exposures retrospectively. MZ twins were indeed found to experience more similar environments but no associations between similarity in environmental exposure and correlations on various traits such as anxiety and depression (Morris-Yates et al., 1990), binge eating disorder (Bulik, Sullivan, & Kendler, 1998) or externalizing disorders (LoParo & Waldman, 2014) were found. These findings support the validity of the EEA, however the retrospective nature of the data collection (e.g. childhood memories) could have influenced the accuracy of measurement of environmental exposure.

Another factor that could potentially lead to violation of the EEA, is the idea that adult MZ twins have a stronger personal relationship with their co-twin than adult DZ twins, and might therefore be more similar if increased social contact is maintained. Several studies have investigated if the degree of social contact between the twin pairs is associated with the correlation on behavioural traits. No associations were found between increased social contact and correlations on personality traits such as neuroticism and extraversion (Kaprio, Koskenvuo, & Rose, 1990; Rose, Koskenvuo, Kaprio, Sarna, & Langinvainio, 1988), substance abuse disorder (LaBuda, Svikis, & Pickens, 1997) or rates of physical activity (Eriksson, Rasmussen, & Tynelius, 2006).

Further evidence that a close personal relationship between MZ twins does not contribute to greater similarity than DZ twins comes from studies using the identical twins raised-apart design. Here correlations between identical twins raised together are compared with those of identical twins reared apart. Research has found that MZ twins correlate highly on anthropometrics (e.g. BMI and waist circumference) (Zhou et al., 2015), IQ (Bouchard, Lykken, Mcgue, Segal, & Tellegen, 1990) and personality traits such as impulsiveness (Coccaro, Bergeman, & Mcclearn, 1993), regardless of whether they were raised together or apart.

#### 3.5.7.4 The 'misclassified zygosity' design

Another way to test the EEA is the 'misclassified zygosity' design. Sometimes twins are misinformed about their zygosity, or simply believe they are non-identical even though they are, in fact, identical. The 'misclassified zygosity' design exploits this occurrence to test the EEA by comparing the correlations of a trait for MZ pairs who correctly believe themselves to be MZs, and MZ pairs who have misclassified themselves as DZs. Matching correlations across both types of MZs are seen as support for the EEA. Early research using this design supported the EEA insofar as identical twins were found to correlate to the same extent on personality traits and cognitive ability, regardless of their believed zygosity (Scarr and Carter-Saltzman 1979). Since then the misclassified zygosity design has been used to provide support for the validity of the EEA in relation to a range of other traits, including: hyperactivity, major depression, generalized anxiety disorder, phobia, bulimia, post-traumatic stress disorder, alcohol and nicotine dependence as well as dieting patterns (Conley et al., 2013; Cronk et al., 2002; Goodman & Stevenson, 1989; Gunderson et al., 2006; Kendler, Neale, Kessler, Heath, & Eaves, 1993a, 1994; Xian et al., 2000). A recent review concluded that the EEA is valid for most

traits, and if violated would only result in a minor inflation of heritability, of no more than 10% (Felson, 2014a).

#### 3.5.7.5 Potential for rater bias in relation to zygosity

Twin studies with samples of children often rely on parent-rated measures. One of the criticisms of parent-rated measures in twin research is that parents' ratings of their twins' behaviour might be biased by their perception of their twins' zygosity. For example, parents might be inclined to rate their twins more similarly if they believe them to be identical. On the other hand, parents who believe their twins to be non-identical might rate them more differently. This potential parental bias would result in an inflated difference between MZ and DZ correlations, and therefore an overestimation of genetic effects (which are estimated by doubling the difference between the MZ and DZ correlations).

### 3.5.8 Summary

The twin method is a well-established and validated research methodology, which has been used to investigate the origins of individual differences in complex human traits. Twin research is conducted globally and findings have been crucial to our understanding of human nature (T. J. C. Polderman et al., 2015). At its core, the twin method exploits the natural occurrence of identical and non-identical twins, in order to decompose the variation in human behaviour into genetic and environmental sources. This basic model can be extended to answer more complex research questions, and some of those have been used in this thesis, including the bivariate correlated factors model, the sex limitation model and the continuous moderator model. Due to its longstanding history, and its ability to challenge dominant views in psychology, the twin method has been thoroughly scrutinised in the past. Reviews and meta-analyses of findings of twin research support the methodology. Further twin research stands out due to the use of advanced statistics and analyses of large population-based cohorts (R. Plomin, DeFries, Knopik, & Neiderhiser, 2016). Even in the face of technological advances, driving large-scale molecular genetic research, twin studies remain of high value. They can inform us about the impact of the environment, as well as work in conjunction with novel technologies, by analysing endophenotypes such as neuroimaging data (van Dongen, Slagboom, Draisma, Martin, & Boomsma, 2012), as well as incorporating other behavioural genetic methods such as mendelian randomisation into a twin model framework (Minica, Dolan, Boomsma, De Geus, & Neale, 2017).

Overall, the twin method is the perfect approach to address the research questions posed in this thesis, targeting key gaps in the literature – understanding the aetiology of emotional over and under-eating in childhood.

# Chapter 4 Study 1: Genetic and environmental contributions to individual differences in EOE from toddlerhood to middle childhood

## 4.1 Background

The study described in this chapter has been published[3] in the journal *Child Development*. Emotional overeating is commonly observed in children and has been found to track across development. Of note is that emotional overeating has been suggested to increase with age, shown by a study of 322 children whose eating behaviours were measured with the CEBQ when they were four and 11 years old. EOE increased significantly (mean four years = 1.8; mean 11 years = 2.1) (Ashcroft et al., 2008). However, additional research has shown that EOE is already present and measurable using the CEBQ in toddlers, with evidence coming from three independent samples of children (age range = 12 - 31 months) (Cao et al., 2012; Mallan et al., 2013; McCarthy et al., 2015).

The aetiology of emotional overeating behaviour in early life remains largely unknown. So far, no twin study has investigated the contributions of genes and environments to individual differences in child emotional overeating. Previous twin research has exclusively analysed samples of adult twins and found low to moderate heritability of emotional overeating, with the majority of variance explained by non-shared environmental influences. For more details on these studies see **Chapter 1.5.2**. Further research is needed to understand the aetiology of emotional overeating at different stages of development, especially during early childhood. Analysis of prospective paediatric data gives tremendous advantages over observations at a single age because it can highlight how the underlying aetiology of behaviour changes with development.

Previous twin research has followed a variety of phenotypes through childhood, such as BMI (Haworth et al., 2008) and cognitive ability (Davis, Haworth, & Plomin, 2009); as well

[3] A version of this study has been published (**Appendix 5.2);**
Herle, M, Fildes, A, Rijsdijk, F, Steinsbekk, S and Llewellyn CH. (2017). The home environment shapes emotional eating. *Child Development.* doi: 10.1111/cdev.12799
I hereby acknowledge the contribution made to this study by the diligent peer review process, which influenced how the findings are presented in the published paper and this thesis chapter.

as from adolescence into adulthood, including IQ (Haworth et al., 2010) and disordered eating (Klump, Burt, Mcgue, & Iacono, 2007). Findings suggest substantial changes in aetiology across development, with genetic effects increasing with age. At the same time, for all traits studied, shared environmental factors were found to be more important earlier on with their effect diminishing as the samples became older and genetic influence takes over. It has been hypothesised that these increases in heritability (and decreases in shared environmental effects) are due to gene-environment correlations. As we grow up and gain more independence, we can make choices acting upon our own genetic propensity. Acting in accordance with our genetic dispositions strengthens the genetic effect underlying the individual differences in the measured behaviour, which is reflected in an increased estimate of heritability.

Employing longitudinal twin models allows researchers to compare the genetic and environmental contributions to behaviour across different developmental stages. Furthermore, this approach can quantify the extent to which the genetic and environmental influences at play at a younger age continue to influence the trait over time (contributing to trait stability), and the extent to which new influences come on line as children mature (contributing to change over time). Through identifying substantial changes in aetiology, this approach highlights potential windows of opportunity for intervention; the age at which environmental influence is strongest, might be the age at which environmental modification is likely to be most successful.

## 4.2 Aims

The aims of this study were:

1. To estimate the relative contribution of genetic and environmental factors to individual differences in child emotional overeating in toddlerhood and childhood
2. To investigate how the aetiology of child emotional overeating changes throughout child development

## 4.3 Methods

### 4.3.1 Sample

Data for this study came from the Gemini cohort - a full description of the sample can be found in **Chapter 3.1.**

### 4.3.2 Measures

Emotional overeating was reported by parents using the CEBQ-T and CEBQ when their children were 16 months and five years old respectively. A more detailed description of these two psychometric scales can be found in **Chapter 3.2.1**. This study included EOE scores for 3774 children (1887 twin pairs) at 16 months and 1986 children (993 twin pairs) at five years, with a combined sample for the analysis of 3784 children (1892 twin pairs) who had data at either 16 months, five years or both ages. A detailed description of zygosity measurement is included in **Chapter 3.1.2**. All covariates in these analyses (age, sex and gestational age) were parent reported. **Chapter 3.2** gives a more detailed description of these measures.

### 4.3.3 Analyses

In order to quantify the genetic and environmental contribution to individual differences in EOE across childhood, a longitudinal twin model was used. As discussed in more detail in **Chapter 3.5**, twin research compares the within-pair similarity for MZ and DZ pairs using intraclass correlations. Maximum likelihood structural equation modelling produces reliable estimates of the variance components of latent genetic (A), shared environmental (C) and non-shared environmental factors (E), as well as 95% confidence intervals, and goodness of fit statistics. All analyses were carried out in R using the OpenMx software (Boker et al., 2011).

### 4.3.4 Data preparation

Prior to analyses, scores on EOE measured at 16 months and five years were regressed by age at measurement, gestational age and sex. This is common practice in twin modelling, as age and sex (for same sex twin pairs only) is completely correlated within twin pairs, and might therefore inflate the twin pair similarity (and the shared environmental effect). Raw and regressed EOE scores were positively skewed, and were therefore transformed using log transformation. Comparisons between the raw, regressed and transformed scores are discussed below (**Chapter 4.4.2**).

In addition, families who provided data at both time points were compared with families who did not complete the questionnaire when the twins were five years old. Chi-square tests were conducted to test for differences in percentage of mothers studying to degree level, as well as differences in families classifying as low, middle or high socio-economic

status. Mother's age and BMI at birth, child weight at birth and gestational age were compared between the two groups using t-tests.

### 4.3.5 Longitudinal twin model

#### 4.3.5.1 Intraclass correlations

Correlations between twins for EOE were calculated and compared for MZs and DZs at 16 months and five years. 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 five years).

#### 4.3.5.2 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. In the first instance two separate univariate twin models were conducted for EOE at 16 months and at five years. Then a bivariate Correlated Factors Model was run providing estimates of A, C and E at 16 months and five 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 five years, denoted by the additive genetic [$r_A$], shared environmental [$r_C$], and unique environmental [$r_E$] correlations. The longitudinal model also quantifies the extent to which continuing genetic and environmental influences explain the longitudinal phenotypic correlation from 16 months to five years (denoted as bivariate A, C and E), by decomposing the phenotypic correlation into to proportions of common A, common C and common E. The bivariate estimates therefore explain whether stability in EOE from 16 months to five years is largely due to the same genes or the same environmental factors influencing the trait at both ages.

For univariate and bivariate models 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. Likelihood Ratio Test (LRT) and the lowest Bayesian Information Criterion (BIC)

value indicate the best fitting model. A more detailed explanation of the theory and practicality underlying this method is provided in **Chapter 3.5.5**.

### 4.3.6 Sensitivity analyses

#### 4.3.6.1 Sex limitation Models

Additionally to test for differences in the aetiology of EOE between boys and girls, sex limitation models were conducted. As described in detail in **Chapter 3.5.5.3**, sex limitation models initially separate estimates of A, C and E (as well as means and variances) for males and females. Subsequently, the models equating across males and females test for the presence of qualitative (*different* genes and/or environmental factors influence EOE in males and females) and quantitative (*the same* genetic and environmental factors influence EOE in males and females but the *magnitude of the effects differ*) sex differences in the aetiology of EOE in toddlerhood and middle childhood.

#### 4.3.6.2 Threshold Models

The method described above applies to continuous and normally distributed data. In the case of ordinal, or very negatively or positively skewed data, a threshold model approach can be taken. As outlined in the methods chapter (**Chapter 3.5.5.5**), threshold models estimate the genetic and environmental contribution to the liability of falling into one category over the other. Threshold liability models are often used if analysed variables are different disease statuses, e.g. classifying as normal weight, overweight or obese. Converting continuous variables into artificial categories is not desirable as it reduces variation in the phenotypes and the analyses of ordinal data require greater samples sizes to achieve appropriate statistical power. However, threshold models can be used to further validate findings from positively or negatively skewed data. Because EOE scores at both time points were skewed, EOE scores were divided into three categories. Children rated as ‘never’ engaging in EOE formed one group (0); remaining participants (those scoring > 1) were divided into low (1) and high (2) categories using a median split.

## 4.4 Results

### 4.4.1 Descriptive statistics

The descriptive statistics for the analysis sample are shown in **Table 4.1**. EOE at 16 months was significantly associated with EOE at five years of age (r = 0.25, 95% CI:

0.19, 0.30; $p < 0.01$), such that toddlers who were prone to eating more in response to negative emotions tended also to do this as five year olds.

There were some differences between families who provided data on EOE at five years and those who did not. Mothers of children providing data at five years were more educated (50.5% with university degree versus 39.8% with university degree, $\chi$ (1) = 12.51, $p < 0.01$), more likely to be in the highest social class (71.5% versus 63.1%, $\chi$ (2) = 16.95, $p < 0.01$), older (mean age 33.84 years versus 32.82 years, t (1916) = 4.47 $p <$ 0.01) and had a lower BMI at baseline, than mothers with data at 16 months only (24.67 versus 25.32, t(1878) = -3.08, $p < 0.01$). However there were no statistical differences between the samples in relation to gestational age and birth weight of the twins. See **Table 4.2** for a full description of results.

**Table 4.1 Descriptive statistics for sample analysed in Chapter 4 Study 1**

| **Analyses Sample** | |
|---|---|
| | **N (%) or Mean[1] (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** | 15.82 (1.15) |
| **Emotional Overeating at 16 months[1]** | 1.64 (0.59) |
| **Age at 5 years** | 5.15 (0.13) |
| **Emotional Overeating at 5 years[1]** | 1.38 (0.48) |

Abbreviations: MZ = monozygotic; DZ= dizygotic; age at 5 year questionnaire completion, calculated using the date of birth of the twins and the date when the questionnaire was filled in

[1] Means (SDs) presented are raw scores

**Table 4.2 Comparison of the families who provided at 16 months and 5 years with families who provided data at 16 months only**

| | Data at 16 months and 5 years (n = 1976, 988 families) | Data at 16 months only (n = 1808, 904 families) | |
|---|---|---|---|
| | **N (%) or Mean (SD)** | **N (%) or Mean (SD)** | ***p*-value** |
| **Gestational age (weeks)** | 36.27 (2.44) | 36.15 (2.51) | 0.09 |
| **Maternal age (years)** | 33.84 (4.75) | 32.82 (5.29) | <0.01 |
| **Weight at birth (kg)** | 2.46 (0.54) | 2.47 (0.54) | 0.89 |
| **Maternal BMI at birth of twins** | 24.67 (4.43) | 25.32 (4.83) | <0.01 |
| **Socioeconomic status[1] [2]** | | | |
| High | 704 (71.5) | 569 (63.1) | <0.01 |
| Intermediate | 144 (14.6) | 154 (17.1) | |
| Low | 137 (13.9) | 178 (19.8) | |
| **Education [3]** | | | |
| no degree | 493 (49.9) | 543 (60.1) | <0.01 |
| University degree | 494 (50.1) | 361 (39.9) | |

[1] Classified using the Office for National Statistics National Statistics Socio-economic Classification (NS-SEC) (Office for National Statistics 2005) and grouped into higher (higher and lower managerial and professional occupations), intermediate (intermediate occupations, small employers and own account workers) and lower SES (lower supervisory and technical occupations, (semi)routine occupations, never worked and long-term unemployed).

[2] Three families had missing data for Socio-economic status but for EOE

[3] One family had missing data for maternal education

### 4.4.2 Distribution of EOE at 16 months and five years

Raw scores were regressed by sex, gestational age and age at measurement. Then regressed scores were log transformed and multiplied in an attempt to reduce skew and increase standard deviations. Performance of MLSEM is improved if only positive definite matrices are included. Therefore, negative values were converted to positive by shifting the whole distribution across zero. Means, standard deviations, skew and kurtosis for raw, regressed and transformed scores for EOE at 16 months and five years in this sample are presented in **Table 4.3.** As presented in **Figure 4.1**, transformations were not successful and variables remained non-normally distributed. However indications of skew and kurtosis were all in the acceptable range (all < 1). Overall, multiplied and log-transformed residuals were found have the greatest standard deviations and the lowest skew and were therefore used in these analyses.

**Table 4.3 Raw, regressed and transformed scores of Emotional Overeating at 16 months and five years**

| 16 months | Mean (SD) | Skew | Kurtosis | Range |
|---|---|---|---|---|
| **Raw scores** | 1.64 (0.59) | 0.8 | 0.87 | 1, 5 |
| **Regressed by age, sex and gestational age** | 0 (0.59) | 0.8 | 0.89 | -0.69, 3.37 |
| **Log-Transformed, multiplied by 5 and shifted across zero** | 3.26 (1.43) | 0.2 | -0.91 | 1.37, 8.4 |
| **5 years** | **Mean (SD)** | **Skew** | **Kurtosis** | **Range** |
| **Raw scores** | 1.57 (0.51) | 0.81 | 0.18 | 1, 3.75 |
| **Regressed by age, sex and gestational age** | 0 (0.51) | 0.81 | 0.17 | -0.64, 2.17 |
| **Log-Transformed, multiplied by 5 and shifted across zero** | 3.31 (1.22) | 0.36 | -0.8 | 1.53, 7.14 |

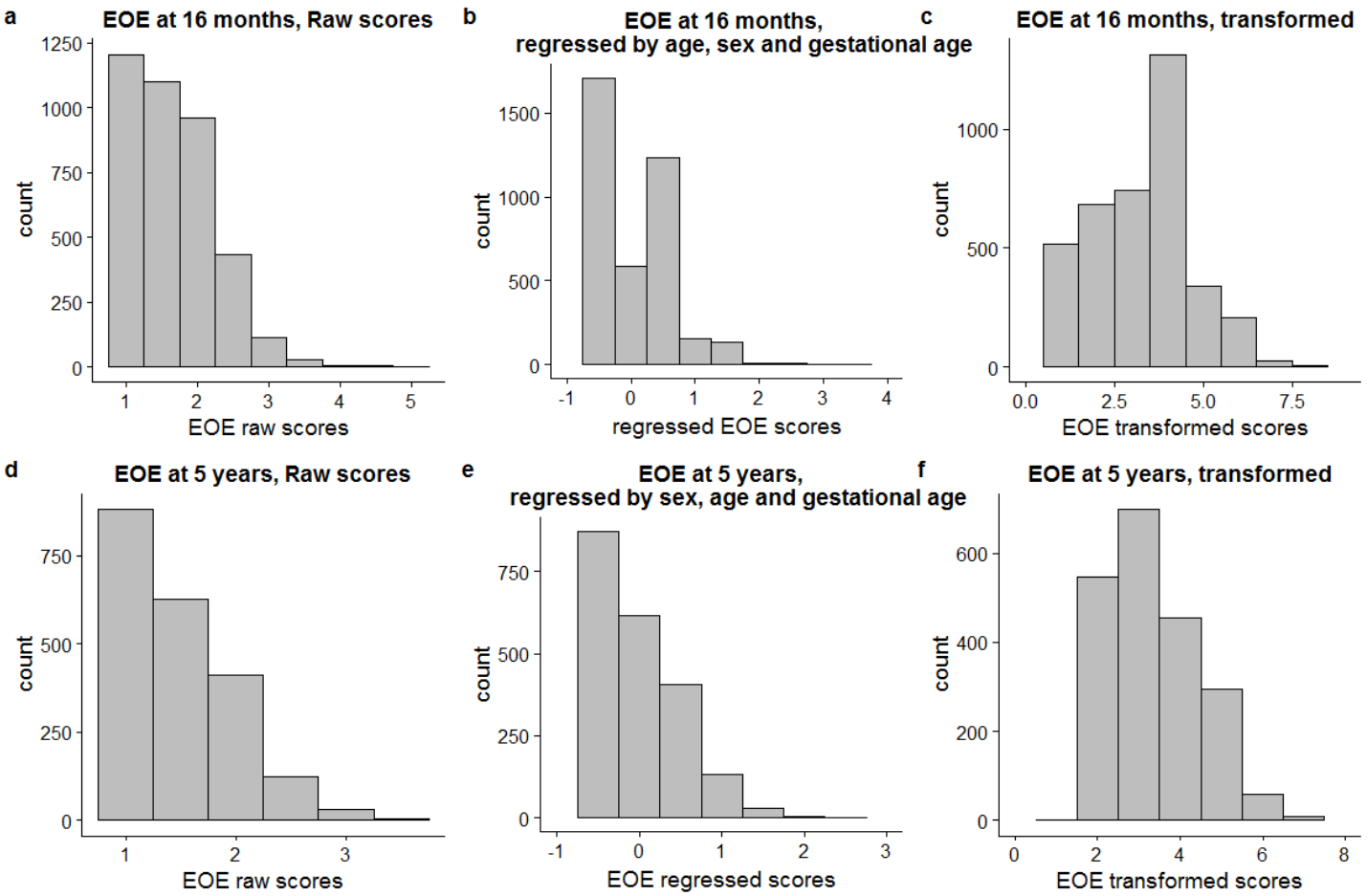


**Figure 4.1 Figures a-c show raw regressed and transformed scores for EOE at 16 months of age; Figures d-f show raw, regressed and transformed scores for EOE at five years of age**

### 4.4.2 Longitudinal twin model

#### 4.4.2.1 Intraclass correlations

The intraclass correlations for the twin pairs at 16 months and five years were stratified by sex and are shown in **Table 4.4**. At both ages the MZ and DZ correlations were high and similar across all types of twins. This pattern suggested a low contribution from genes and a strong contribution from the shared environment to variation in EOE at both ages. Comparing across males and females, estimates were very similar, indicating the absence of any sex specific effects.

**Table 4.4 Twin correlations (95% confidence intervals) for emotional overeating scores measured at 16 months and five years and for the cross-twin cross-time correlation**

| | **MZM** (280 pairs) | **DZM** (311 pairs) | **MZF** (312 pairs) | **DZF** (315 pairs) | **DZOS** (639 pairs) |
|---|---|---|---|---|---|
| **EOE 16 months** | 0.96 (0.95, 0.97) | 0.92 (0.91, 0.93) | 0.99 (0.99, 0.99) | 0.94 (0.93, 0.95) | 0.95 (0.94, 0.95) |
| | **MZM** (159 pairs) | **DZM** (152 pairs) | **MZF** (159 pairs) | **DZF** (195 pairs) | **DZOS** (304 pairs) |
| **EOE 5 years** | 0.97 (0.97, 0.98) | 0.97 (0.96, 0.98) | 0.98 (0.97, 0.98) | 0.97 (0.96, 0.98) | 0.97 (0.96, 0.97) |
| | **MZM** (159 pairs) | **DZM** (152 pairs) | **MZF** (159 pairs) | **DZF** (195 pairs) | **DZOS** (304 pairs) |
| **CT - CT** | 0.25 (0.18, 0.31) | 0.26 (0.20, 0.32) | 0.27 (0.21, 0.33) | 0.28 (0.22, 0.34) | 0.26 (0.20,0.32) |

Abbreviations: MZM = Monozygotic Male-Male; DZM = Di-zygotic Male-Male; MZF = Monozygotic Female-Female; DZF = Di-zygotic Female-Female; DZOS = Di-zygotic Male-Female; EOE = Emotional Overeating; CT-CT = Cross-twin cross-trait

#### 4.4.2.2 Maximum likelihood structural equation modelling

***Estimating the contribution of genes and environments to individual differences in child EOE at 16 months and five years***

MLSEM was used to calculate the univariate A, C and E parameters for EOE at 16 months and five years. Parameter estimates and fit statistics for all models are displayed in **Table 4.5a** and **4.5b**. At first a full ACE model, including parameter estimates for A, C and E, was compared against the saturated model. Three submodels were then fitted, each dropping one latent factor at a time (AE model, CE model and E model). The latent factor E can never be dropped from the model as it includes random measurement error. As all models are nested, the LRT was used to distinguish the model fit between the different models. At both time points the full ACE model was found to fit the data best (16 months: $\Delta \chi^2 = 8.076$, $p = 0.23$; 5 years: $\Delta \chi^2 = 6.444$, $p = 0.38$). The BIC confirmed this by assigning the greatest negative value to the full ACE model at both 16 months (BIC = -30257.561) and five years (BIC = -17109.522).

Genetic effects were significant at 16 months and five years, but contributed little to variation in EOE at either age (9%, 95% CI: 8, 10% and 3%, 95% CI: 2, 4% respectively). Shared environmental effects explained the majority of variance in EOE at both ages (16 months: 89%, 95% CI: 87, 90%; 5 years: 95%, 95% CI: 93, 96%). The variance explained by the unique environment at each age was small (16 months: 2%, 95% CI: 2, 3%; 5 years: 3%, 95% CI: 2, 3%).

**Table 4.5a Parameter estimates (95% CI) and fit statistics for EOE at 16 months**

| EOE 16 months (n = 3832) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Model** | **A** | **C** | **E** | **-2LL** | **df** | **Δ $\chi^2$ (df)** | **p-value** | **BIC** |
| **Sat** | | | | -1801.627 | 3764 | | | -30220.337 |
| **ACE** | 0.09 (0.08, 0.10) | 0.89 (0.87, 0.90) | 0.02 (0.02, 0.03) | -1793.55 | 3770 | 8.076 (6) | 0.23 | -30257.561 |
| **AE** | 0.97 (0.97,0.97) | | 0.03 (0.03, 0.03) | -376.850 | 3771 | 1416.701 (1) | <0.001 | -28848.41 |
| **CE** | | 0.95 (0.94, 0.95) | 0.05 (0.05, 0.06) | -1603.787 | 3771 | 189.765 (1) | < 0.001 | -30075.347 |
| **E** | | | 1 | 2620.748 | 3772 | 4414.299 (2) | < 0.001 | -25858.362 |

Model: AE, CE and E models are nested within the full ACE model. The ACE model dissects the phenotypic variance into A, C and E; the AE model drops the C parameter and assesses the variance explained by the A and E parameters only; the CE model drops the A parameter and assesses the variance explained by the C and E parameters only; the E model drops both the A and C parameters and assesses the variance explained by E only. The best-fitting is bolded.

**Table 4.5b Parameter estimates (95% CI) and fit statistics for EOE at five years**

| EOE at five years (n = 1996) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Model** | **A** | **C** | **E** | **-2LL** | **df** | **Δ $\chi^2$ (df)** | **p-value** | **BIC** |
| **Sat** | | | | -2151.598 | 1976 | | | -17070.666 |
| **ACE** | 0.03 (0.02, 0.04) | 0.95 (0.93, 0.96) | 0.03 (0.02, 0.03) | -2145.154 | 1982 | 6.444 (6) | 0.38 | -17109.522 |
| **AE** | 0.97 (0.96, 0.97) | | 0.03 (0.03, 0.04) | -1086.008 | 1983 | 1059.146 (1) | < 0.01 | -16057.926 |
| **CE** | | 0.96 (0.96, 0.97) | 0.04 (0.03, 0.04) | -2124.917 | 1983 | 20.236 (1) | < 0.01 | -17096.836 |
| **E** | | | 1 | 529.869 | 1984 | 2675.022 (2) | < 0.01 | -14449.600 |

Model: AE, CE and E models are nested within the full ACE model. The ACE model dissects the phenotypic variance into A, C and E; the AE model drops the C parameter and assesses the variance explained by the A and E parameters only; the CE model drops the A parameter and assesses the variance explained by the C and E parameters only; the E model drops both the A and C parameters and assesses the variance explained by E only. The best-fitting is bolded.

***Decomposing the longitudinal association of child EOE from 16 months to five years***

At first a Cholesky decomposition was applied to the data, allowing latent factors A, C and E at 16 months to influence variation in EOE at five years. For ease of interpretation this Cholesky decomposition was transformed into a Correlated Factors Model.

A path diagram of the full Correlated Factors ACE model is presented in **Figure 4.2**. LRT suggested no deterioration of fit between the saturated model and the full ACE model ($\Delta\chi^2 = 14.059$, $p = 0.66$), indicating that the ACE model fitted the data well.

A moderate shared environmental correlation ($r_C = 0.29$, 95% CI 0.24,0.36) between 16 months and five years indicated that, some of the shared environmental influences on EOE at 16 months and five years were the same, but many new shared environmental influences also come into play at five years. There was also a significant negative genetic correlation between the two time points ($r_A = -0.38$; 95% CI -0.84, -0.12). However, because the genetic components of variance at both ages were very small (especially at age five years), 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 five years of age.

The bivariate estimates quantified the proportion of the longitudinal association (between EOE at 16 months and five years) explained by common genetic and environmental factors. These suggested that the longitudinal association was completely driven by the shared environmental effects that were at play at both ages. The bivariate A was very close to zero (BivA: -0.01; 95% CI -0.02, -0.00) and bivariate E was non-significant (BivE: 0.00; 95% CI -0.01, 0.02). Hence shared environmental factors accounted for the total phenotypic correlation (BivC: 0.26; 95% CI 0.22, 0.33). 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 a more parsimonious model, non-significant or small parameters were constrained to be zero. In submodel 1 the aetiological correlation $r_E$ for E was dropped, as it was found to be non-significant ($r_E = 0.03$; 95% CI -0.11, 0.17). Fit statistics indicated that this reduced submodel fit the data equally ($\Delta\chi^2 = 0.21$, $p = 0.64$). On

balance, the full ACE longitudinal model is used for interpretation. A full list of all estimates and fit statistics is presented in **Table 4.6**.

**Table 4.6 Fit statistics for longitudinal models for Emotional Overeating at 16 months and five years**

| **Fit Statistics** | | | | | |
|---|---|---|---|---|---|
| **Model** | **-2LL** | **df** | **Δ $\chi^2$ (df)** | **p-value** | **BIC** |
| **Sat** | 11927.94 | 5732 | | | -31349.438 |
| **ACE** | 11942.00 | 5749 | 14.059 (17) | 0.66 | -31463.731 |
| **Submodel [1]** | 11942.21 | 5744 | 0.21 (1) | 0.65 | -31471.071 |

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

[1] Submodel 1: In this submodel non-significant $r_E$ was constrained to 0. Submodel 1 is nested in and compared against the full ACE model.

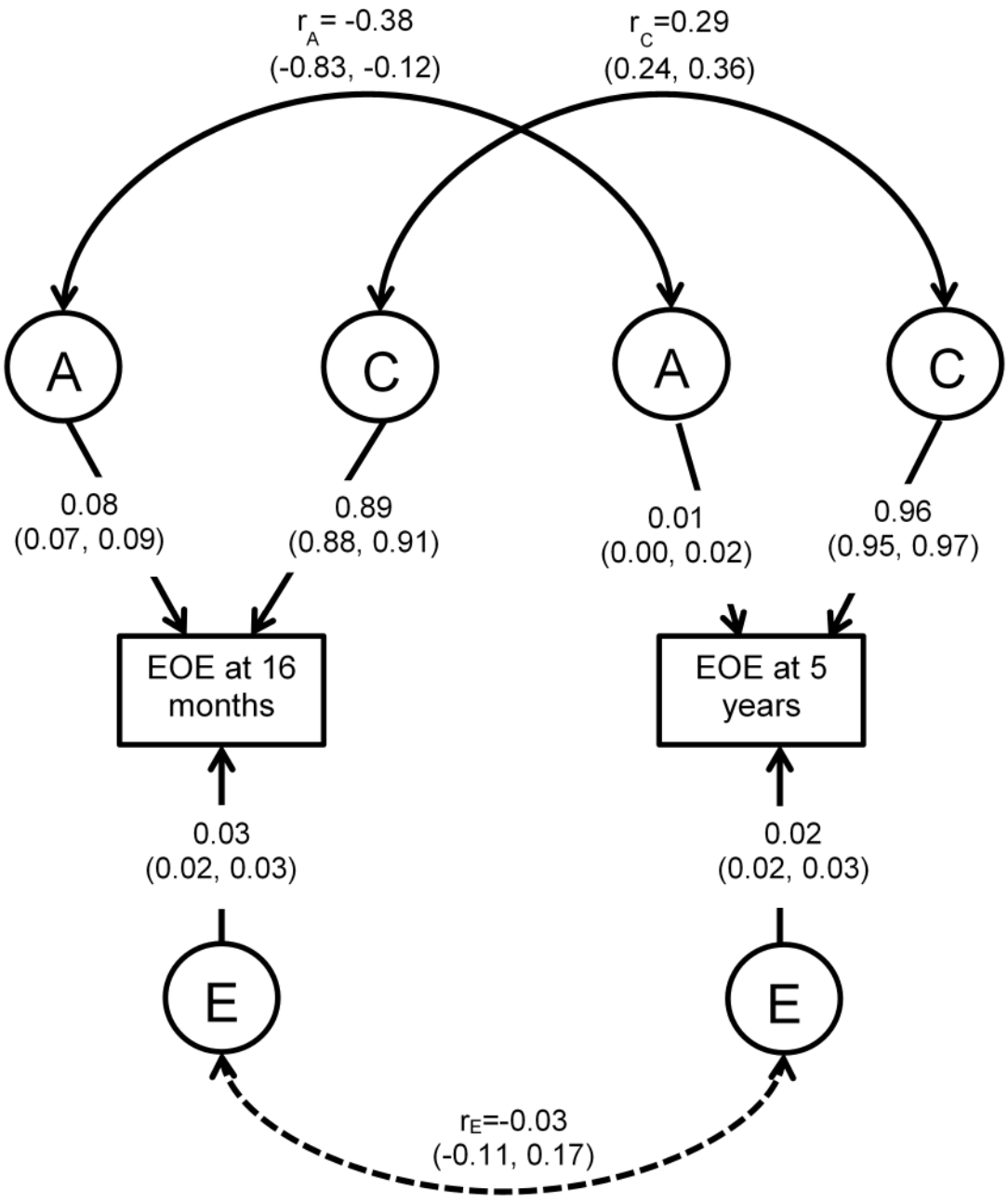


**Figure 4.2: The full longitudinal Correlated Factors ACE model including all parameters.**

*The rectangular boxes represent the measured phenotype (Emotional Overeating, EOE) at 16 months and five 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 Intervals crossing 0, is represented as a dotted line.*

#### 4.4.2.3 Sex differences

Sex limitation models were conducted to test for differences in parameter estimates between boys and girls. A full description of the sex limitation twin models can be found in **Chapter 3.5.5.3**. To reiterate briefly, sex limitation models estimate paths for A, C and E separately for males and females. The first model estimates means and variances separately for twin 1 and twin 2 as well as for males and females (saturated model, Model 1). Subsequently, in two separate models (Model 2 and Model 3) the genetic and shared-environmental correlation within opposite sex twins were fixed to their theoretical values ($r_A$ = 0.5, $r_C$ = 1.0) and tested against the saturated model. Then both $r_A$ and $r_C$ were fixed to their theoretical values of 0.5 and 1.0 in the common effects model (common effects model, Model 4). In a fifth model, variance differences between males and females were tested (scalar model, Model 5). If the estimated scalar is close to 1 no variance differences between males and females are indicated. In a final model (null model, Model 6) all estimates are equated across males and females, allowing for one A, C and E estimate across both sexes, as well as the same variance for males and females. The model fit is compared using the LRT and the BIC.

It is possible to extend a univariate sex limitation model into a bivariate sex limitation model including two variables. However, this was not possible in this situation. As shown in **Table 4.4** intraclass correlations were very high for MZ and DZ twins. Stratifying the sample by sex and zygosity resulted in high intercorrelations within the data structure, which pre-empted successful model fitting of a bivariate sex limitation twin model. Therefore, two separate univariate models for EOE at 16 months and at five years were conducted instead.

***Sex differences at 16 months***

All fit statistics for the separate models at 16 months can be found in **Table 4.7**. The LRT suggested sex differences, indicated by significant differences in -2LL between the different models. However the BIC values, which take the sample size into account when comparing model fit, suggested that the common effects model (Model 4) fitted the data best, as indicated by the greatest negative BMI value (BIC = -21788.122). The common effects model allows A, C and E to differ in effect size for males and females. Parameter estimates for males and females for A, C and E from this model

are presented in the **Appendix 3.1a;** it is clear that they do not differ substantially across males and females. For example, the heritability estimate for EOE at 16 months for males is 8% (95% CI: 6, 11%), and for females it is 11% (95%CI: 9, 12%). When equating across the sexes (in the null model, Model 6), heritability is very similar at 9% (95% CI: 8, 11%). Similar estimates were also observed for C and E for males and females separately, and when equated. These very similar results for males and females indicate that sex differences are of little importance for this behaviour. Therefore, a model combining males and females was preferred.

**Table 4.7 Fit statistics for sex limitation modelling for EOE at 16 months**

| EOE 16 months | Comparison | Ep | -2LL | Df | Δ $\chi^2$ (df) | *p*-value | BIC |
|---|---|---|---|---|---|---|---|
| **1 Saturated model [1]** | | 23 | 6052.284 | 3691 | | | -21756.553 |
| **2 Full sex limitation ($r_A$=free)[2]** | 1 | 9 | 6133.728 | 3705 | 81.44 (14) | <0.01 | -21780.588 |
| **3 Full sex limitation ($r_C$=free)[3]** | 1 | 9 | 6133.728 | 3705 | 81.44 (14) | <0.01 | -21780.588 |
| **4 Common effects model ($r_A$=0.5, $r_C$=1)[4]** | 2 & 3 | 8 | 6133.728 | 3706 | 0.00 (1) | 1 | -21788.122 |
| **5 Scalar Model [5]** | 4 | 6 | 6260.272 | 3708 | 127.544 (2) | <0.01 | -21676.647 |
| **6 Null model (no sex differences) [6]** | 5 | 5 | 6274.631 | 3709 | 14.3359 (1) | <0.01 | -21669.822 |

Abbreviations: Ep = number of estimated parameters, -2LL = -2 log-likelihood of data, Df = degrees of freedom, BIC = Bayesian Information Criterion, $r_A$ = genetic correlation, $r_C$ = shared environmental correlation

[1] Model 1: Saturated Model estimates means and variances separately for twin 1, twin 2 and for males and females

[2] Model 2: Aetiological genetic correlation ($r_A$) between DZ opposite sex pairs is allowed to be free, whereas shared environmental correlation ($r_C$) was fixed to 1.

[3] Model 3 Aetiological genetic correlation ($r_A$) between DZ opposite sex pairs is fixed to 0.5, whereas shared environmental correlation ($r_C$) was allowed to be free.

[4] Model 4, Common effects model, both $r_A$ and $r_C$ between DZ opposite sex twin pairs are fixed to their theoretical values of 0.5 and 1. Parameter estimates for A, C and E were estimated freely for males and females.

[5] Model 5, Scalar Model, variances are allowed to vary between males and females

[6] Model 6, Null Model, A, C and E estimates are equated to be the same for males and females implying no sex differences underlying the aetiology of this phenotype

***Sex differences at five years***

At five years fit statistics indicated that equating parameters across males and females (null model) did not produce a significant drop in model fit ($\Delta \chi^2 = 0.029$, $p = 0.86$) in comparison to the Scalar Model. Furthermore the greatest negative BIC value (BIC = -11518.851) indicated a superior fit of the null model, which was the preferred model. All fit statistics can be seen in **Table 4.8**. Estimated parameters for A, C and E for each model are shown in **Appendix 3.1b.**

**Table 4.8 Fit statistics for sex limitation modelling for EOE at five years**

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

Abbreviations: Ep = number of estimated parameters, -2LL = -2 log-likelihood of data, Df = degrees of freedom, BIC = Bayesian Information Criterion, $r_A$ = genetic correlation, $r_C$ = shared environmental correlation

[1] Model 1: Saturated Model estimates means and variances separately for twin 1, twin 2 and for males and females

[2] Model 2: Aetiological genetic correlation ($r_A$) between DZ opposite sex pairs is allowed to be free, whereas shared environmental correlation ($r_C$) was fixed to 1.

[3] Model 3 Aetiological genetic correlation ($r_A$) between DZ opposite sex pairs is fixed to 0.5, whereas shared environmental correlation ($r_C$) was allowed to be free.

[4] Model 4, Common effects model, both $r_A$ and $r_C$ between DZ opposite sex twin pairs are fixed to their theoretical values of 0.5 and 1. Parameter estimates for A, C and E are estimated freely for males and females

[5] Model 5, Scalar Model, variances are allowed to vary between males and females

[6] Model 6, Null Model, A, C and E estimates are equated to be the same for males and females implying no sex differences underlying the aetiology of this phenotype

#### 4.4.2.4 Alternative threshold models

Concordance rates for twin pairs split by zygosity for EOE at 16 months and five years are presented in **Table 4.9a** and **4.9b**. The majority of twin pairs were concordant for their EOE score indicating high twin pair similarity. Concordance rates were slightly different for MZ and DZ twins, with the DZ twin pairs scoring slightly more different at 16 months (MZ: 97.5% pairs concordant; DZ: 90.9% concordant). A similar pattern was observed at five years (MZ: 98.9% pairs concordant DZ: 95.9% pairs concordant). **Table 4.10** shows the fit statistics and ACE parameters derived from threshold models for EOE at 16 months and five years. When comparing these results with the estimates derived from the previous models treating EOE as a continuous variable, findings yielded very similar results. The estimates for C were slightly higher when using a threshold model, probably due to decreased variation in EOE following categorization of the data. Converting continuous variables into categorical data means more twins are likely to receive the same score, increasing the overall similarity within twin pairs, and therefore inflating the shared environmental effect. As estimated parameters were very similar using continuous or categorical data, on balance, it was decided that using continuous data is more informative in this instance, and estimates derived from continuous data models presented in **Figure 4.2** are taken forward for interpretation in the discussion.

**Table 4.9a Emotional Overeating categories for each twin, split by zygosity at 16 months**

| Emotional Overeating Categories at 16 months | | | | | | | |
|---|---|---|---|---|---|---|---|
| **MZ** | **Twin 1** | | | **DZ** | **Twin 1** | | |
| **612 pairs** | | | | **1275 pairs** | | | |
| **Twin 2** | **EOE=0** | **EOE=1** | **EOE=2** | **Twin 2** | **EOE=0** | **EOE=1** | **EOE=2** |
| **EOE=0** | 196 | 3 | 1 | **EOE=0** | 377 | 10 | 6 |
| **EOE=1** | 6 | 185 | 1 | **EOE=1** | 11 | 318 | 14 |
| **EOE=2** | 0 | 4 | 216 | **EOE=2** | 5 | 16 | 518 |

Abbreviations: MZ = Monozygotic; DZ = Di-zygotic; EOE = Emotional Overeating

**Table 4.9b Emotional Overeating categories for each twin, split by zygosity at five years**

| Emotional Overeating Categories at five years | | | | | | | |
|---|---|---|---|---|---|---|---|
| **MZ** | **Twin 1** | | | **DZ** | **Twin 1** | | |
| **336 pairs** | | | | **663 pairs** | | | |
| **Twin 2** | **EOE=0** | **EOE=1** | **EOE=2** | **Twin 2** | **EOE=0** | **EOE=1** | **EOE=2** |
| **EOE=0** | 189 | 1 | 2 | **EOE=0** | 336 | 2 | 0 |
| **EOE=1** | 0 | 31 | 1 | **EOE=1** | 4 | 73 | 6 |
| **EOE=2** | 0 | 0 | 112 | **EOE=2** | 1 | 4 | 237 |

Abbreviations: MZ = Monozygotic; DZ = Di-zygotic; EOE = Emotional Overeating

**Table 4.10 MLSEM outcomes for variance components A, C and E, 95% Confidence Intervals and fit statistics for Emotional Overeating at 16 months and five years using categorical data**

| **Threshold models** | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Emotional Overeating** | **Model** | **A** | **C** | **E** | **-2LL** | **Δ-2LL** | **df** | **p-value** |
| **16 months** | **Sat** | | | | 5119.556 | | 3767 | |
| (n =3832) | **ACE** | 0.03 (0.02-0.04) | 0.96 (0.95-97) | 0.007 (0.005-0.01) | 5115.795 | -4.15 | 3776 | 1 |
| **5 years** | **Sat** | | | | 2192.29 | | 1979 | |
| (n = 1996) | **ACE** | 0.00 ($8.45 \times 10^{-9}$- $8.48 \times 10^{-9}$) | 0.99 (0.99-0.99) | 0.00 ($1.94 \times 10^{-3}$-$2.17 \times 10^{-3}$) | 2161.083 | -31.207 | 1980 | 1 |

Abbreviations: 2LL = -2 times log-likelihood of data; Δ-2LL = difference in 2 times log-likelihood; df = degrees of freedom; Sat = Saturated model; ACE = Full model including all factors

## 4.5 Discussion

### 4.5.1 Summary of findings

The first aim of this study was:

> To estimate the relative contribution of genetic and environmental factors to individual differences in child emotional overeating in toddlerhood and childhood

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 (five years). Somewhat surprisingly, genetic effects contributed very little to this trait at either age (16 months: 9 %, 95% CI: 8, 10%; and 5 years: 3%, 95% CI: 2, 4%). These findings contrast with the high heritability estimates observed for other eating behaviours measured in 10-year-old children – Satiety responsiveness (63%) and Enjoyment of food (75%) (Carnell et al., 2008). They also contrast with the high heritability estimates for four eating behaviours measured in Gemini in infancy: Satiety responsiveness (72%); Slowness of eating (84%); Food responsiveness (59%); Enjoyment of food (54%) (Llewellyn, van Jaarsveld, et al., 2010).

Evidence for the importance of the shared environment in shaping individual differences in this trait during both toddlerhood (89%) and early childhood (96%) also contrasts with previous studies of emotional overeating in adults. As outlined in **Chapter 1.3** 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 emotional overeating 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 on' 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 (e.g. 'active gene-environment correlation') (Bergen et al., 2007).

Within twin research the focus often lies on establishing the heritability of human behaviours, and the information gained about the influence of the environment

sometimes gets downplayed. However, in keeping with the findings presented here, substantial effects of the shared environment have been reported for other childhood behaviours. An earlier adoption study compared the similarity between parents and their offspring, and adopted versus non-adopted siblings (n = 220), in order to estimate the effects of genes and family environment on individual differences in TV watching. Results suggested equal effects of genetic and environmental factors (R. Plomin, Corley, Defries, & Fulker, 1990). Another example comes from research investigating the aetiology of autism. A study of 202 twin pairs, with at least one sibling clinically diagnosed with autism, showed that the majority of sibling concordance/discordance rates was explained by the shared environment (55%) (Hallmayer et al., 2011). Similarly, objective observations of secure mother-child attachment patterns were not found to be heritable in a small sample of 87 twin pairs (mean age: 4.5 years). However individual differences were substantially shaped by shared environmental factors (52%) (Bokhorst et al., 2003).

One previous twin study of adult emotional overeating suggested aetiological differences between males and females, suggesting a higher heritability for women (Keskitalo et al., 2008). The sex limitation model suggested potential quantitative sex differences in child EOE at 16 months (see **Table 4.7**). However, the A, C and E estimates were very similar for males and females, suggesting a comparable aetiology for males and females at 16 months.

A ‘passive gene-environment correlation’ might also explain the high shared environmental effects on variation in EOE in this study. 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 behaviour in their children; children therefore inherit from their parents both the genes and the environment that encourage emotional overeating. 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 parental and child measures of emotional overeating 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).

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

The second aim of this study was:

> To investigate how the aetiology of child emotional overeating changes throughout child development

Results suggested 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.

Previous longitudinal studies of emotional overeating have found similar and somewhat higher indications of longitudinal tracking. A study of 428 British children measured EOE at four and 11 years. Results suggested a stronger correlation of 0.45 between the two ages. Moreover, EOE was found to significantly increase with age (Ashcroft et al., 2008). These previous findings differ from the results presented in this chapter. In this sample, the longitudinal correlation was smaller, and the mean actually decreased with time. However, the age of the children varied substantially between the two studies. Ashcroft et al focussed on middle (four years) to later childhood (11 years) (Ashcroft et al., 2008), whereas here the period from toddlerhood to middle childhood was the focus of this study. From 16 months to five years, children's abilities, autonomy and environment change drastically, and therefore a lower phenotypic correlation between the two ages is not surprising.

Similarly to Ashcroft et al (2008), a longitudinal Norwegian study that analysed child EOE prospectively from six to eight years of age (n = 623) found strong tracking, indicated by a high positive correlation (r = 0.59) (Steinsbekk, Belsky, & Wichstrom, 2016). Recently, this study was extended to include measures of EOE when the children were ten years old as well (n = 801). Again the earlier child EOE was found to a significant predictor of later EOE, from six to eight (r = 0.53) and eight to ten years (r = 0.6) (Steinsbekk et al.,

2017). These previous studies suggest longitudinal stability of emotional overeating might only be established from middle childhood. This study found a significant but smaller longitudinal association from toddlerhood to middle childhood. More longitudinal research is needed, tracking child EOE from early life into adolescence. This will be possible in the Gemini cohort, as data collection is ongoing.

In contrast to other longitudinal studies, the analyses presented here took advantage of twin data. This enabled an examination of the extent to which genetic and environmental factors underlying EOE correlate across development, and how they contribute to stability and change in EOE from toddlerhood to middle childhood. Results suggested that the entire longitudinal correlation (r = 0.25) between EOE at 16 months and five years could be explained by continuing shared environmental factors that persisted over that period. However, the moderate shared environmental correlation ($r_C = 0.29$) indicated that many novel shared environmental factors come into play to influence EOE at five years as well, reflecting considerable developmental changes occurring between the two ages.

There were no unique environmental effects that continued from toddlerhood to early childhood. The genetic correlation ($r_A$) between child EOE at 16 months and five years was negative but significant. But due to the very small contribution of additive genetic effects on EOE at either age, this correlation is difficult to interpret.

### 4.5.2 Implications

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 can be derived. Future research is needed to establish the modifiable shared environmental factors that play a causal role in shaping emotional overeating in early childhood. Previous studies have suggested that parental feeding practices influence children's emotional overeating. Research has suggested 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 behaviours (Farrow, Haycraft, & Blissett, 2015b).

However, there is also evidence indicating that child emotional eating elicits parental controlling feeding behaviour as well (such as monitoring, restriction and pressure to eat),

suggesting a potential bidirectional association between child eating and parental feeding (Haycraft & Blissett, 2012).

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 emotional overeating

### 4.5.3 Strengths and limitations

This study is the first twin study of emotional overeating focussing on toddlers and no previous study has aimed to investigate the genetic and environmental contributions to emotional overeating stability and change across different developmental stages. These findings are therefore novel, shining light on the aetiology of emotional overeating during an under-researched developmental period. Other strengths of this study include the large sample size, providing fairly precise parameter estimates, as well as the homogeneous age and measurement at the two ages, due to the use of a longitudinal cohort.

However, there are limitations to be considered. 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 than those who dropped out. However there were no significant differences regarding the 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, compared to 16 months. Overall, the Gemini cohort is over-representative of white mothers with higher education and the majority of the families are of higher socio-economic status. Future studies would benefit from studying child emotional overeating in more diverse samples.

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 behaviour with accuracy in young children, and therefore rate two twins the same, increasing the

similarity of twin pairs regardless of zygosity, resulting in artificially inflated estimates of the shared environmental contribution. However, parents are well placed to report on their children's eating behaviour, arguably knowing their children better than other potential respondents. In order to prevent any rater bias, assessments from others such as carers or teachers could be incorporated into future research. Furthermore a comparison of child rated and parent rated emotional overeating would be of interest. Yet, the age from which children can reliably rate their own emotional overeating behaviours is not clear. Nevertheless, studies including older children and young adolescents should consider incorporating both child and parent rated questionnaires of emotional overeating.

A further limitation is that EOE scores were not normally distributed at either 16 months or five years of age, with about one third of 16 month olds, and quarter of five year olds not reported to engage in emotional overeating behaviour at all. However mean scores for EOE reported here were of similar size in other comparable cohorts (dos Passos et al., 2015; Mallan et al., 2013; Tan & Holub, 2015). In order to address the skewness of the data, EOE scores were categorised and reanalysed as a threshold model. Results from these secondary analyses produced very similar results to findings reported when using EOE as continuous variable (see **Table 4.9a, 4.9b** and **4.10**). The low means and variance for EOE in these datasets, questions the validity of the measure. This is also suggested by the low estimates for the non-shared environmental effects, which also captures random measurement error (as well as environmental influences unique to each child, e.g. attending a different school than their co-twin, or illness).This observation suggests that error was correlated across the two twins, and captured by the shared environmental effect. In order to scrutinise this issue in more detail, these twin analyses were repeated in an independent twin sample with EOE, in which I was able to decompose individual differences in EOE in four-year-old twins. This replication is described in detail in **Chapter 9**. Results suggested increased estimates for genetic and non-shared environmental effects. However the big majority of the variation was explained by shared environmental factors, very similar to estimates produced here. Findings from the replication support the validity of the measure, producing the same pattern of results in an independent sample.

## 4.6 Conclusions

These findings are in stark contrast to heritability studies of other childhood eating behaviours, which often find moderate to high contributions of genetic effects (Carnell et al., 2008; Llewellyn, van Jaarsveld, et al., 2010; Smith et al., 2017). Furthermore EOE was found to track across early development, suggesting that early interventions may be useful in curbing the development of this eating behaviour. Longitudinal twin modelling revealed that this trait stability was not influenced by genes but driven by shared environmental effects. However, only a small proportion ($r_C$ = 0.29, 95% CI 0.23, 0.35, the equivalent of ~8.4% of variance explained) of shared environmental factors were common between 16 months and five years, reflecting the vast environmental changes that children experience over this time, that contribute to developmental change in this behaviour from toddlerhood to middle childhood. The development of emotional overeating was found to differ from other childhood eating behaviours, with influences of the shared environment being highlighted as the most important. Environmental influences shared by twin pairs are likely to include family and home factors related to eating, such as parental feeding and eating behaviour. This is the first twin study of emotional overeating in a childhood sample, filling a substantial gap in the literature. However, the aetiology of emotional under-eating remains unknown. In **Chapter 5** a bivariate twin model was applied to examine the aetiology of EUE in middle childhood, as well as establish the extent to which EOE and EUE share their aetiology.

# Chapter 5 Study 2: Genetic and environmental contributions to individual differences in EOE and EUE at 5 years[4]

## 5.1 Background

As shown in **Chapter 4** individual differences in EOE are mainly driven by shared environmental factors in toddlerhood as well as in middle childhood. So far no twin study has investigated the contributions of genes and environments to individual differences in child or adult emotional under-eating. As discussed in more detail in **Chapter 1.5.2**, there have been three twin studies of emotional *overeating* in adults. All three reported low to moderate genetic effects, with the majority of variation being attributed to environmental factors unique (non-shared) to each individual, and no influence at all from the shared environment. All three studies were of small size (585-782 twin pairs included), and the estimates reported therefore had large confidence intervals. In addition, there was large heterogeneity in age – age ranges were 17 - 82, 20 - 65 and 23 - 29 years across the three studies (Keskitalo et al., 2008; Sung et al., 2010; Tholin et al., 2005).

Variation in emotional over- and under-eating has been observed from early childhood (Ashcroft et al., 2008), and interestingly, as outlined in **Chapter 1.3.2.3**, even though they have different associations with weight and weight gain, emotional over- and under-eating tend to be positively correlated (r = 0.16 - 0.30) such that children who score higher on emotional overeating tend also score higher on emotional under-eating (Wardle, Guthrie et al. 2001, Viana, Sinde et al. 2008, Mallan, Liu et al. 2013, Domoff, Miller et al. 2015, Ek, Sorjonen et al. 2016, Steinsbekk, Belsky et al. 2016). This suggests that some children have a tendency to *both* overeat *and* under-eat in response to stress and negative emotions. This positive correlation could indicate that the two behaviours might be different aspects of the same underlying trait, the tendency to experience changes in

[4] A version of this chapter has been published, see **Appendix 5.3;**
Herle, M., Fildes, A., Steinsbekk, S., Rijsdijk, F., & Llewellyn CH (2017). Emotional over- and under-eating in early childhood are learned not inherited. *Scientific Reports*.
I hereby acknowledge the contribution made to this study by the diligent peer review process, which influenced how the findings are presented in the published paper and this thesis chapter.

one's appetite in response to stress. However, no previous study has attempted to understand the nature of the relationship between emotional over- and under-eating.

The twin method is a powerful tool for interrogating the common aetiology underlying different behavioural traits. Twins can be used to quantify the extent of common genetic and environmental influences underlying emotional over- and under-eating, and the extent to which shared genes and shared environments explain the positive association between them.

## 5.2 Aims

The aims of this study were:

1. To establish for the first time the genetic and environmental contribution to individual differences in EUE in childhood
2. To investigate the extent to which emotional over and under-eating share their genetic and environmental aetiology

## 5.3 Methods

### 5.3.1 Sample

Data analysed for this study were drawn from Gemini, described in detail in **Chapter 3.1**. This study included data collected from 1027 twin pairs (346 MZ pairs; 681 DZ pairs) when they were approximately five years old (mean = 5.15 years; SD = 0.13).

### 5.3.2 Measures

EOE and EUE were measured using the Child Eating Behaviour Questionnaire (Wardle et al., 2001). The CEBQ includes subscales for EOE and EUE each containing four items. A detailed description of this measure can be found in **Chapter 3.2.1** and the full questionnaire is attached in **Appendix 2.2**.

### 5.3.3 Analyses

Intraclass correlations, indicating twin pair similarity, were calculated for MZ and DZ pairs to give a first indication of genetic and environmental factors contributing to individual differences in EOE and EUE at five years.

Maximum likelihood structural equation modelling was used to estimate more reliably the proportions of variance in EOE and EUE explained by three latent factors: additive genetic effects (A), shared (C) and non-shared (E) environmental effects.

At first a full ACE model, including parameter estimates for A, C and E, was compared against the saturated model. Then three submodels were fitted, each dropping one latent factor (AE model, CE model and E model). As all models are nested, the Likelihood Ratio Test (LRT) was used to distinguish the model fit between the different models

Following a bivariate Correlated Factors Model was used to quantify the genetic and environmental contributions to covariation between EOE and EUE. The bivariate Correlated Factors Model estimates aetiological correlations which indicate how many genetic ($r_A$), shared environmental ($r_C$) and unique environmental ($r_E$) influences underlying EOE and EUE are the same. In addition, this model decomposes the phenotypic correlation between EOE and EUE into the latent factors A, C and E. These bivariate estimates (denoted: BivA, BivC and BivE) indicate the extent to which common genetic, shared environmental and unique environmental factors explain the phenotypic correlation between the two behaviours (i.e. whether EOE and EUE are positively correlated because the same genetic or environmental factors influence them both). A more detailed description of the twin method and its underlying principles can be found in **Chapter 3.5**. All analyses were carried out using OpenMx software (Boker et al., 2011) a statistical package run in R.

### 5.3.4 Data preparation

Prior to analyses scores on EOE and EUE were regressed by age at EOE and EUE measurement, gestational age and sex. This is common practice in twin research, as age (and sex in same sex pairs) is always the same within twin pairs, and could therefore inflate the twin pair similarity, and increased the shared environmental effect. Regressed scores for EOE were then log transformed, and a positive value greater than the lowest now negative score was added to the whole distribution to avoid negative numbers, as positive values are favoured by Maximum likelihood structural equation modelling.

## 5.4 Results

### 5.4.1 Descriptive statistics

Data for EOE, EUE and the covariates (age at measurement, gestational age and sex) were available for 1027 twin pairs. A summary of data analysed in this study is presented in **Table 5.1**. Mean scores for EOE were low (mean = 1.56, SD = 0.51). Mean scores for EUE were higher (mean = 2.66, SD = 0.84), indicating that on average, children sometimes demonstrated emotional under-eating. EOE and EUE were significantly positively correlated, with a moderate effect size (r = 0.43, 95% CI: 0.37, 0.47), indicating that children who emotionally overeat tend also to emotionally under-eat to some extent.

### 5.4.2 Distributions of EOE and EUE within this sample

EOE was less common than EUE in this sample; 530 (25.0%) children were rated as not engaging in emotional overeating at all, while only 48 children (2.33%) received the lowest possible score for EUE. Regressed EUE scores were found to be close to a normal distribution and were therefore not log-transformed. However EUE residuals were shifted across (2 added to the distribution) to avoid negative values. The raw, regressed and transformed scores for EOE and EUE are shown in **Figure 5.1**. **Figures 5.1 a-c** show scores for EOE, whereas **Figures 5.1 d-f** show the EUE scores. Transformation was successful for EUE, however EOE remained positively skewed after transformation.

**Table 5.1 Descriptive statistics for the sample included in the analyses in Study 2**

| **Twin pairs** | **N (%) or Mean (SD)** |
|---|---|
| **Total** | 1027 pairs |
| | (2054 children) |
| **Zygosity** | |
| MZ pairs | 346 (33.7) |
| DZ pairs | 681 (66.3) |
| **Sex** | |
| Males | 1000 (48.69) |
| Females | 1054 (51.31) |
| **Gestational age (weeks)** | 36.26 (2.43) |
| **Age at measurement of EOE and EUE (years)** | 5.15 (0.13) |
| **Emotional Overeating at 5 years**[1] | 1.57 (0.51) |
| **Emotional Under-eating at 5 years**[1] | 2.68 (0.85) |

Abbreviations: MZ = Monozygotic; DZ = Di-zygotic

[1] Means (SDs) are for raw scores

**Table 5.2 Raw, regressed and transformed scores of emotional overeating and emotional under-eating at five years**

| Emotional Overeating | Mean (SD) | Skew | Kurtosis | Range |
|---|---|---|---|---|
| **Raw scores** | 1.57 (0.51) | 0.81 | 0.18 | 1, 3.75 |
| **Regressed by age, sex and gestational age** | 0 (0.51) | 0.81 | 0.17 | -0.64, 2.17 |
| **Log-Transformed, multiplied by 2 and shifted across zero** | 3.31 (1.22) | 0.36 | -0.8 | 1.53, 7.14 |
| **Emotional Under-eating** | **Mean (SD)** | **Skew** | **Kurtosis** | **Range** |
| **Raw scores** | 2.68 (0.85) | 0.08 | -0.65 | 1 ,5 |
| **Regressed by age, sex and gestational age** | 0 (0.84) | 0.09 | -0.64 | -1.73, 2.34 |
| **Shifted across zero** | 2 (0.84) | 0.09 | -0.64 | 0.27, 4.34 |

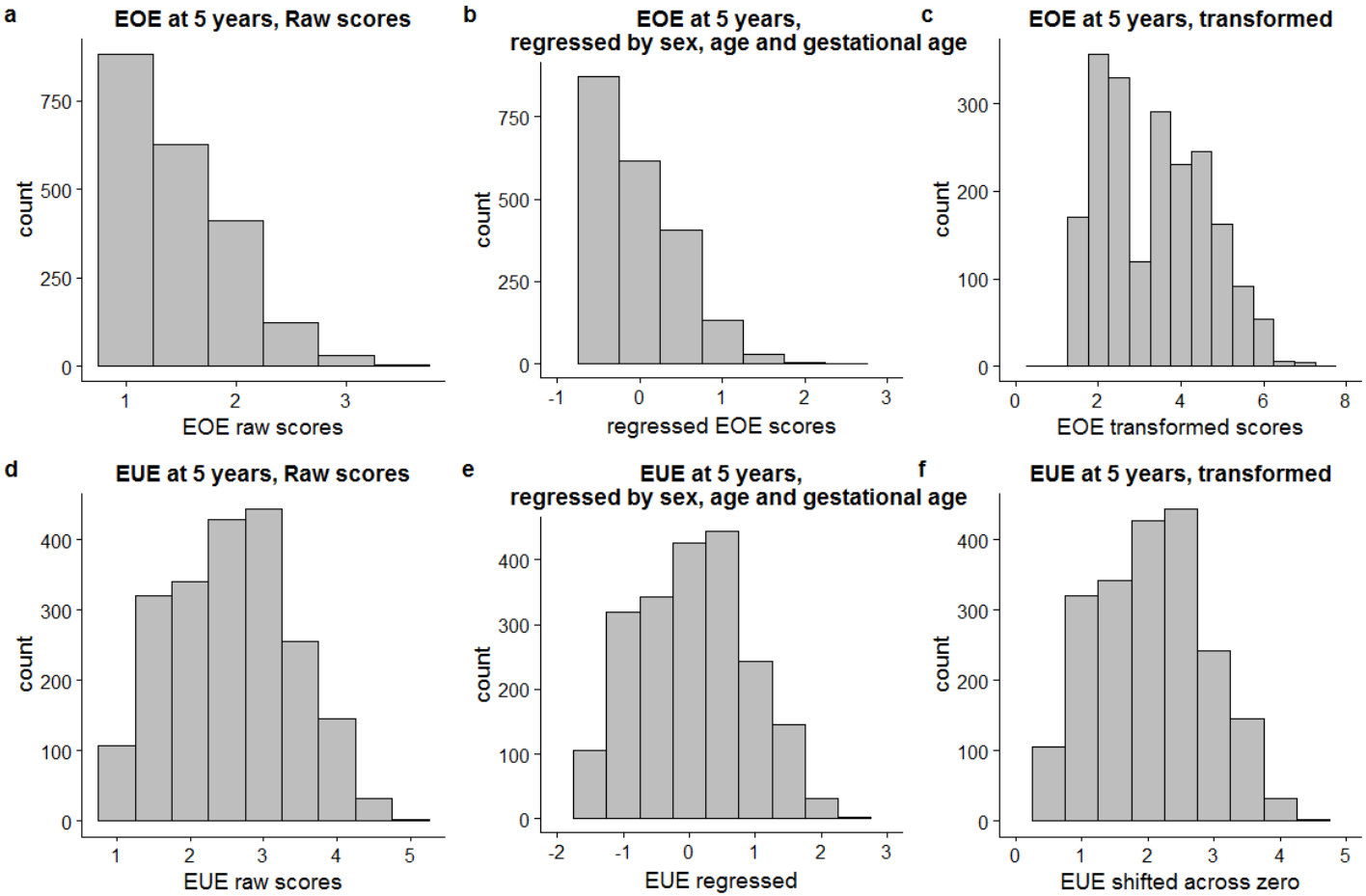


**Figure 5.1 Figures a-c show raw (a), regressed (b), and transformed (c) scores for EOE; Figures d-f show raw (d), regressed (e), and shifted (c) scores for EUE**

### 5.4.3 Twin modelling

#### 5.4.3.1 Intraclass correlations

ICCs for EOE and EUE were calculated for MZ and DZ twin pairs separately and stratified by sex to examine the patterns of resemblance for each behaviour. As shown in **Table 5.3**, the ICCs were high and of similar magnitude for both MZs and DZs, for both EUE and EOE. This pattern of twin correlations suggests strong shared environmental factors underlying variation in both EUE and EOE. The cross-twin cross-trait (CT-CT) correlations showed a similar pattern to the univariate ICCs (also shown in **Table 5.3**). The CT-CT correlations for both MZ and DZ pairs were significant, of similar magnitude, and of a comparable effect size to the phenotypic correlation itself indicating that shared environmental influences are largely driving the observed phenotypic association between EOE and EUE. ICCs were very similar between males and females, indicating no significant sex differences in aetiology.

**Table 5.3 Intraclass correlations (95% Confidence intervals) for EOE and EUE as well as the cross-twin cross-trait correlations for MZ and DZ twin pairs, split by sex**

| | **MZM** (176 pairs) | **DZM** (166 pairs) | **MZF** (170 pairs) | **DZF** (199 pairs) | **DZOS** (316 pairs) |
|---|---|---|---|---|---|
| **EOE** | 0.98 (0.97, 0.98) | 0.95 (0.93, 0.96) | 0.98 (0.97, 0.98) | 0.93 (0.92, 0.95) | 0.96 (0.95, 0.97) |
| **EUE** | 0.98 (0.97, 0.98) | 0.95 (0.93, 0.96) | 0.98 (0.98, 0.99) | 0.96 (0.94, 0.96) | 0.94 (0.93, 0.95) |
| **CT-CT** | 0.46 (0.42, 0.52) | 0.47 (0.42, 0.52) | 0.41 (0.36, 0.46) | 0.43 (0.38, 0.49) | 0.44 (0.39,0.49) |

MZM: Monozygotic Male-Male; DZM: Di-zygotic Male-Male; MZF: Monozygotic Female-Female; DZF: Di-zygotic Female-Female; DZOS: Di-zygotic Male-Female; EOE: Emotional Overeating; CT-CT: Cross-twin cross-trait

#### 5.4.3.2 Maximum likelihood structural equation modelling

***Establishing the genetic and environmental contributions to EOE and EUE***

MLSEM was used to calculate the univariate A, C and E parameters for EOE and EUE at five years. Parameters estimates and fit statistics for all models are displayed in **Table 5.4a** and **5.4b**. For both EOE and EUE, the ACE model was found to be of best fit to the data (EOE: $\Delta \chi^2 = 5.024$, $p = 0.54$; EUE: $\Delta \chi^2 = 11.611$, $p = 0.07$).

Genetic effects were significant for EOE and EUE but were of low importance for both behaviours (EOE: 7%, 95%CI: 6, 9; EUE: 7%, 95%CI: 6, 9). Shared environmental effects explained the majority of variance for both EOE and EUE (EOE: 91%, 95%CI: 89, 92; EUE: 91%, 95%CI: 90, 92,). The variance explained by the unique environment for each behaviour was small (EOE: 2%, 95%CI: 2, 3; EUE: 2%, 95%CI: 1, 2).

**Table 5.4a Parameter estimates (95% CI) and fit statistics for EOE at five years from the univariate analyses**

| EOE 5 years (n = 2052) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Model** | **A** | **C** | **E** | **-2LL** | **df** | **Δ $\chi^2$ (df)** | **p-value** | **BIC** |
| **Sat** | | | | -1687.181 | 2042 | | | -15946.187 |
| **ACE** | 0.07 | 0.91 | 0.02 | -1682.157 | 2048 | 5.024 (6) | 0.54 | -15983.060 |
| | (0.06, 0.09) | (0.89, 0.92) | (0.02, 0.03) | | | | | |
| **AE** | 0.97 | | 0.03 | -835.466 | 2049 | 846.691 (1) | < 0.001 | -15143.352 |
| | (0.97, 0.98) | | (0.02, 0.03) | | | | | |
| **CE** | | 0.95 | 0.05 | -1593.989 | 2049 | 88.168 (1) | < 0.001 | -15901.875 |
| | | (0.95, 0.96) | (0.04, 0.05) | | | | | |
| **E** | | | 1 | 851.947 | 2050 | 2534.105 (2) | < 0.001 | -13462.921 |

Models: Saturated Model, estimates all means and variances freely. The ACE model dissects the phenotypic variance into A, C and E; the AE model drops the C parameter and assesses the variance explained by the A and E parameters only; the CE model drops the A parameter and assesses the variance explained by the C and E parameters only; the E model drops both the A and C parameters and assesses the variance explained by E only. The best-fitting is bolded, indicated by a non-significant change in –2LL.

**Table 5.4b Parameter estimates (95% CI) and fit statistics for EUE at five years from the univariate analyses**

| EUE at five years (n = 2054) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Model** | **A** | **C** | **E** | **-2LL** | **df** | **Δ $\chi^2$ (df)** | **p-value** | **BIC** |
| **Sat** | | | | 410.8875 | 2044 | | | -13862.084 |
| **ACE** | 0.07 | 0.91 | 0.02 | 422.4991 | 2050 | 11.61159 | 0.07 | -13892.370 |
| | (0.06, 0.09) | (0.90, 0.92) | (0.01, 0.02) | | | | | |
| **AE** | 0.98 | | 0.02 | 1316.9515 | 2051 | 894.45248 | < 0.001 | -13004.900 |
| | (0.97, 0.98) | | (0.02, 0.03) | | | | | |
| **CE** | | 0.96 | 0.04 | 539.3630 | 2051 | 116.86389 | < 0.001 | -13782.489 |
| | | (0.95, 0.96) | (0.04, 0.05) | | | | | |
| **E** | | | 1 | 3114.5597 | 2052 | 2692.06064 | < 0.001 | -11214.275 |

Models: Saturated Model, estimates all means and variances freely. The ACE model dissects the phenotypic variance into A, C and E; the AE model drops the C parameter and assesses the variance explained by the A and E parameters only; the CE model drops the A parameter and assesses the variance explained by the C and E parameters only; the E model drops both the A and C parameters and assesses the variance explained by E only. The best-fitting is bolded, indicated by a non-significant change in –2LL.

***Decomposing the correlation between EOE and EUE into genetic and environmental factors***

A bivariate correlated factors model including all parameters (A, C, and E for EOE and EUE; $r_A$, $r_C$, and $r_E$ between EOE and EUE) was tested against the saturated model. The LRT indicated no significant difference in fit between the two models ($\Delta \chi^2 = 21.957$, $p = 0.19$), confirming that the bivariate ACE correlated factors model fitted the data well. In line with the LRT, the BIC favoured the bivariate correlated factors Model over the saturated model, indicated by the lower value. Fit statistics for the saturated and bivariate correlated factors model are shown in **Table 5.5**.

Parameter estimates for A, C and E (and 95% confidence intervals, CIs) indicated the relative importance of genetic, shared environmental and unique environmental influences on variation in EOE and EUE. As suggested by the ICCs and univariate analyses, variation in both EOE and EUE was largely explained by shared environmental influences (EOE: C = 0.92, 95% CI: 0.91, 0.93; EUE: C = 0.91, 95% CI: 0.89, 0.92). In contrast, genetic effects only played a minor role in explaining variation in either of the two behaviours (EOE: A = 0.06, 95% CI: 0.06, 0.09; EUE: A = 0.07, 95% CI: 0.06, 0.09). Contributions from non-shared environmental factors were also small (EOE: E = 0.02, 95% CI: 0.02, 0.03; EUE: E = 0.02, 95% CI: 0.02, 0.02).

**Table 5.5 Model fit statistics for the saturated model and the full bivariate Correlated Factors Model**

| Model | parameters | -2LL[1] | df[1] | Δ $\chi^2$ (df) | p-value | BIC[1] |
|---|---|---|---|---|---|---|
| **Sat[1]** | 28 | 6202.517 | 4078 | | | -22273.598 |
| **ACE[1]** | 11 | 6224.474 | 4095 | 21.957 (17) | 0.19 | -22370.349 |

[1]Abbreviations: 2LL: -2 log-likelihood of data; df: degrees of freedom; Δ $\chi^2$: change in chi-square; BIC: Bayesian Information Criterion; Sat: Saturated model; ACE: Full bivariate Correlation Factors Model

The shared environmental correlation ($r_C$) was significant, positive and moderate in effect size ($r_C$= 0.49, 95% CI: 0.43, 0.54) indicating that a quarter ($0.49^2$ = 24% of variance explained) of the shared environmental influences that underlie variation in EOE are the same as those influencing EUE at five years of age. There was also a significant negative genetic correlation which was moderate in effect size ($r_A$ = -0.37, 95% CI: -0.50, -0.23). However, because the genetic contributions to variation in EUE and EOE were so small (6-7%), the genetic correlation between them is difficult to interpret. The correlation for non-shared environmental effects was non-significant ($r_E$= 0.03, 95% CI: -0.1, 0.08).

The bivariate estimate for A was very small (BivA = -0.03, 95% CI: -0.04, -0.02), and bivariate E was estimated as zero (BivE = 0.00, 95% CI: -0.00, 0.00). Hence the common shared environmental factors underlying both EOE and EUE were driving the observed phenotypic association (r = 0.43) between them (BivC = 0.44, 95% CI: 0.39, 0.49). **Figure 5.2** presents a path diagram of the full correlated factors model, displaying the effect of the latent factors A, C and E on EOE and EUE as well as the aetiological correlations ($r_A$, $r_C$, $r_E$) between them.

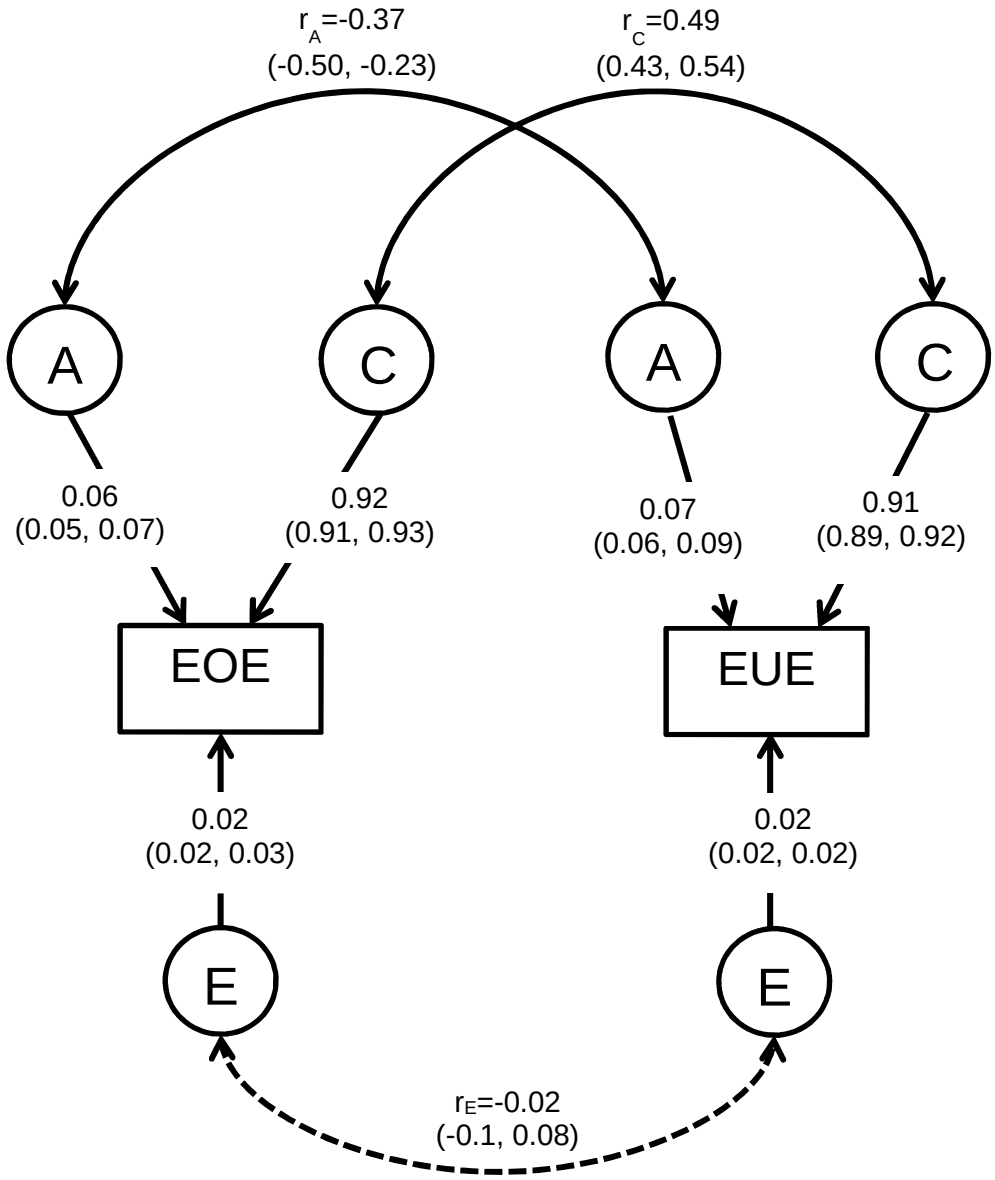


**Figure 5.2 Correlated Factors Model of EOE and EUE in Gemini**

*The rectangular boxes represent the measured phenotype (emotional overeating, EOE and emotional under-eating, EUE) using the Child Eating Behaviour Questionnaire at five years of age. The circles indicate the latent factors: additive genetic effects (A), shared environmental effects (C) and non-shared environmental effects (E). The straight single-headed arrows reflect pathways with the variance explained by each latent factor (including 95% confidence intervals, CI). The etiological correlations are shown on the curved double-headed arrows. These indicate the extent of common genetic ($r_A$), shared environmental ($r_C$) and non-shared environmental ($r_E$) influences across the two phenotypes. The non-significant etiological correlation ($r_E$), with a 95% CI crossing 0, is represented as a dotted line.*

#### 5.4.3.3 Sex differences

As shown in **Table 5.3**, ICCs and cross-trait cross-twin correlations did not differ between male-male and female-female twins. ICCs for opposite sex DZ twins were of similar magnitude. Full sex limitation models were conducted to test for the presence of quantitative and qualitative sex differences. A detailed description of this process can be found in **Chapter 3.5.5.4**. To reiterate, sex limitation models stratify twin pairs by sex estimating paths for males and females separately. Fit statistics indicate which model fits the data best, suggesting qualitative, quantitative, scalar (variance) or no differences between males and females (**Table 5.5**). Fit statistics indicated qualitative and quantitative sex differences. Model 2, constraining $r_A = 0.5$ between DZ opposite sex twin pairs and $r_C$ allowed to vary freely, fitted the data best, indicated by the lowest BIC value (-31682.63). All path estimates (including 95% Confidence Intervals) for the best-fitting model (Model 2) are presented in **Table 5.6**. However, the 95% confidence intervals for the sex-stratified path estimates overlapped between males and females, indicating no meaningful sex differences. Therefore, the model including males and females together was preferred for interpretation.

**Table 5.6 Fit statistics for Bivariate Sex Limitation models for EOE and EUE at five years**

| Model | Comparison | Ep | -2LL | Df | Δ $\chi^2$ (df) | *p*-value | BIC |
|---|---|---|---|---|---|---|---|
| **1 Full sex limitation ($r_A$ = free)[2]** | | 26 | -2715.705 | 4080 | | | -31205.79 |
| **2 Full sex limitation ($r_C$ = free)[3]** | | 26 | -3192.547 | 4080 | | | -31682.63 |
| **3 Common effects model ($r_A$ = 0.5, $r_C$ = 1)[4]** | 2 | 22 | -2715.705 | 4084 | 476.842 (4) | <0.01 | -31233.72 |
| **4 Scalar Model [5]** | 3 | 19 | -2713.747 | 4087 | 1.958 (3) | 0.58 | -31252.71 |
| **5 Null model (no sex differences)[6]** | 4 | 11 | -2702.424 | 4095 | 11.323 (8) | 0.18 | -31297.25 |

Abbreviations: Ep = number of estimated parameters, -2LL = -2 log-likelihood of data, Df = degrees of freedom, BIC = Bayesian Information Criterion, $r_A$ = genetic correlation, $r_C$ = shared environmental correlation

[1] Model 1: Constrained correlation model estimates separate correlations for same-sex and opposite sex twin pairs

[2] Model 2: Aetiological genetic correlation ($r_A$) between DZ opposite sex pairs is allowed to be free; shared environmental correlation ($r_C$) was fixed to 1.

[3] Model 3 Aetiological genetic correlation ($r_A$) between DZ opposite sex pairs is fixed to 0.5; shared environmental correlation ($r_C$) was allowed to be free.

[4] Model 4, Common effects model, both $r_A$ and $r_C$ between DZ opposite sex twin pairs are fixed to their theoretical values of 0.5 and 1. Parameter estimates for A, C and E are estimated freely for males and females

[5] Model 5, Scalar Model, variances are allowed to vary between males and females

[6] Model 6, Null Model, A, C and E estimates are equated to be the same for males and females implying no sex differences underlying the aetiology of this phenotype

**Table 5.7 Path estimates (95% Confidence Intervals) from Model 2**

Parameters are estimated separately for males and females. The genetic correlation between DZ opposite sex differences was fixed to 0.5, whereas the shared environmental correlation was ($r_C$) was allowed to be free

| | **EOE** | | | **EUE** | | |
|---|---|---|---|---|---|---|
| | **A** | **C** | **E** | **A** | **C** | **E** |
| **Males** | 0.05 (0.03, 0.07) | 0.93 (0.91, 0.95) | 0.02 (0.02, 0.03) | 0.06 (0.04, 0.09) | 0.92 (0.89, 0.94) | 0.02 (0.02, 0.03) |
| **Females** | 0.07 (0.05, 0.08) | 0.91 (0.89, 0.93) | 0.02 (0.02, 0.03) | 0.06 (0.04, 0.08) | 0.92 (0.90, 0.94) | 0.02 (0.01, 0.02) |
| | **$r_A$** | | **$r_C$** | | **$r_E$** | |
| **Males** | -0.25 (-0.50, 0.02) | | 0.52 (0.46, 0.57) | | -0.11 (-0.66, 0.03) | |
| **Females** | -0.56 (-0.77, 0.57) | | 0.49 (0.42, 0.54) | | 0.11 (-0.04, 0.26) | |

Abbreviations: A: additive genetic effects; C: shared-environmental effects; E: non-shared environmental correlation; $r_A$: genetic correlation; $r_C$: shared-environmental correlation; $r_E$: non-shared environmental correlation

## 5.5 Discussion

### 5.5.1 Summary of findings

The first aim of this study was:

> To establish for the first time the genetic and environmental contribution to individual differences in EUE in childhood

This was the first twin study of the aetiology of EUE in either adults or children. Results suggested that environmental factors shared between twin pairs are the most important contributors to individual differences in EUE explaining the majority of variance (91%). Genetic effects on EUE were significant but not of great importance at this age (7%). The very low heritability estimates for EOE measured at five years, were already discussed in Study 1 (**Chapter 4**). Here, the low heritability for EUE at five years was equally surprising, and in stark contrast to the much higher heritability estimates observed for a range of other eating behaviours in both infants and children, captured using the CEBQ and the BEBQ. In infancy in this sample, Satiety responsiveness, Food responsiveness, Enjoyment of food, and Slowness in eating were moderately to highly heritable (53-84%) (Llewellyn, van Jaarsveld, et al., 2010). In toddlers (3 years) in this sample, heritability was high for Food fussiness (78%) (Fildes, van Jaarsveld, Cooke, Wardle, & Llewellyn, 2016). High heritability estimates were also found for Satiety and Food responsiveness (53% and 75%) in a sample 10-year-old twins (Carnell et al., 2008). These findings for EUE stand in contrast to all of these other eating behaviours.

The second aim of this study was:

> To investigate the extent to which emotional over- and under-eating share their genetic and environmental aetiology

A high and positive correlation between EOE and EUE was found (r = 0.43), in line with previous studies (Domoff et al., 2015; Sleddens, Kremers, & Thijs, 2008; Wardle, Guthrie, Sanderson, & Rapoport, 2001). However, the correlation found here was higher than previously reported (range 0.16 – 0.30). In comparison to previous studies, the sample size for analyses here was much larger. The most comparable study by Domoff et al (2015) analysed data from 1002 four-year-old children, reporting the highest correlation of all previous studies (r = 0.3) of EOE and EUE (Domoff et al., 2015). However, the sample of this previous differs substantially from the Gemini cohort. Domoff et al (2015)

aimed to validate the structure of the CEBQ in a low income sample of US children. In comparison the Gemini families are mostly of high socio-economic status. Further, cultural differences between US and UK parents regarding emotional feeding strategies might as well have contributed to the difference in correlation between EOE and EUE.

Results from the bivariate twin model suggested that about one quarter ($r_C$ = 0.49; $0.49^2$ = 24%) of shared environmental factors are the same for both EOE and EUE. The genetic correlation was negative and significant ($r_A$= -0.37, 95% CI: -0.51, -0.26). In the context of a positive phenotypic correlation between EOE and EUE, and very low heritability of each, estimate for $r_A$ becomes difficult to interpret. A tentative explanation would be that carrying genes associated with EOE makes individuals less likely to carry genes associated with EUE and vice versa. However, this interpretation is of little use, as genetics effects on both EOE and EUE were very low.

The model also decomposes the phenotypic correlation between EOE and EUE into the latent factors A, C and E. Bivariate estimates for genetic and non-shared environmental effects were non-significant (BivA = -0.03, 95% CI: -0.004, -0.02; BivE = 0.00, 95% CI: -0.00, 0.00) indicating that genes and unique environmental influences common to both EOE and EUE did not contribute to the positive association between these two behaviours. The phenotypic correlation between EUE and EOE was entirely explained by shared environmental effects influencing both (BivC = 0.44, 95% CI: 0.39, 0.48).

### 5.5.2 Implications

Environmental factors shared by two twins within one family were the most important shapers of EOE and EUE. Furthermore, the positive association between these two behaviours was explained entirely by common shared environmental influences. However, EOE and EUE were found to be somewhat aetiologically distinct, as not all shared environmental factors affected both behaviours.

Aetiological correlations indicated that shared environmental factors underlying EOE explained about one quarter of the variance in EUE and vice versa. These findings suggest that under- and overeating in response to negative emotions are behaviours that are learned in early childhood, and that some of the underlying environmental factors that shape them are the same. That is, there are certain shared environmental factors that make a child more likely to both under- and over-eat in response to negative emotion. On the other hand, the majority of the shared environmental factors are specific to EOE

or EUE. This distinction is crucial. Both EOE and EUE were found to have similar aetiological pattern, to the extent that both show low heritability and shared environmental effects explain the majority of variation. However their aetiology differed substantially, as only a minority of the shared environmental factors affect both behaviours, suggesting environmental specificity.

As discussed in more detail in **Chapter 1.5.1**, emotional overeating has been traditionally described as learned in childhood. The Psychosomatic Theory of obesity hypothesises that weight gain is the result of the maladaptive pairing of feelings of stress and sadness with food intake. Participants with obesity were found to consume more in the face of stress and negative emotion, potentially as a result of established emotional overeating patterns in early life (Schachter, 1968). The results of this study support this notion by providing evidence that the majority of individual differences in EOE are explained by factors shared within one family. Potential shared environmental factors affecting both child emotional over- and under-eating could be parental feeding strategies and policies affecting both children. In fact, parental behaviours, mainly parental feeding strategies and their associations with child emotional overeating.

A longitudinal study (n = 801) of Norwegian children (aged, six, eight and ten years) used structural equation modelling to investigate the direction of relationships between parental emotional feeding (the tendency of the parents to use food to sooth an upset child calming him or her down) and child EOE. Findings showed that high parental emotional feeding resulted in increased child EOE later on. However the associations were found to be bidirectional, suggesting that once a child has established a tendency to emotionally overeat, parents then respond with more emotional feeding, and the child's tendency to emotionally overeat is reinforced even further (Steinsbekk et al., 2017). Using observations in the lab Farrow et al (2015) tested if children (n = 41) whose parents emotionally fed them at three to five years of age were more likely to emotionally overeat two years later. Children were exposed to a mild stressor, and their consumption of snacks was recorded. Results suggested that children of parents who reported higher levels of emotional feeding two years prior, consumed more calories when exposed to a mild stressor (Farrow et al., 2015). A cross-sectional study (n = 95 child-parent dyads) also reported a positive association between child EOE and parental emotional feeding (Tan & Holub, 2015).

Although there have been a few studies on the environmental shapers of emotional overeating, studies aiming to understand the risk factors for emotional under-eating are sparse. The current findings indicate that some of the factors that shape emotional overeating are the same as those that shape emotional under-eating. It is therefore reasonable to assume that parents' feeding practices may affect emotional under-eating as well, although more research is needed to confirm this hypothesis. In addition, future research needs to identify shared environmental factors specific to emotional over- and under-eating. One cross-sectional study (n = 156 mother child dyads) pointed towards the quality of the relationship of the parents as specific to EUE. Mothers describing their relationship to their partner as warm and positive correlated negatively with child EUE, whereas a hostile parental relationship was positively associated with EUE. On the other hand, there was no association between EOE and parental relationship (Haycraft & Blissett, 2010). This importance of the parental relationship for the development of EUE indicates that a hostile home environment lacking emotional support might be specific to EUE. However longitudinal research is needed to investigate the direction of this relationship further and more studies of the environmental shapers of emotional under-eating are needed.

There has been no previous twin study of emotional under-eating, but as described in detail in **Chapter 1.5.2**, there have been three twin studies of emotional overeating in adults, none of which observed any shared environmental effects; all of the variance in emotional overeating was explained by genetic and non-shared environmental effects. Overall, these adult studies have also reported larger genetic influences on emotional overeating than were found in the present study, although estimates varied widely (9%-60%) due to small sample sizes and wide age ranges (Keskitalo et al., 2008; Sung et al., 2010; Tholin et al., 2005). Outcomes from twin studies are age- and sample-specific. This is of particular importance for twin studies because previous research has suggested that the influence of genetic effects increase steadily with development for a variety of phenotypes (e.g: IQ,(Haworth et al., 2010); and BMI, (Haworth et al., 2008). Therefore, findings from adult studies cannot be extrapolated to children. Furthermore, estimates can vary between cultures, due to potential differences in the genetic population structure and environmental exposures. For example, heritability estimates for adult depression were found to be much lower in a sample from a low to middle income Asian country (Sri Lanka), when compared to analyses of Western data (Ball et al., 2009).

### 5.5.3 Strengths and limitations

As with all twin research, this study needs to adhere to the assumptions of the twin method. Twin studies assume that MZ and DZ twins are exposed to environmental factors to the same extent – so-called the 'equal environments' assumption. This assumption has been tested and confirmed previously and is therefore deemed valid (Conley et al., 2013; Felson, 2014b). For more details see **Chapter 3.5.7**.

Emotional over- and under-eating were parent reported, and the measures could be subject to bias. Direct observations and laboratory tests could be advantageous, but such methods are time consuming and costly for large samples and pose ethical and practical challenges. Parents are deemed to know their children better than anyone else and are arguably the best informants of their children's eating behaviour. In addition, EOE and EUE scores may be influenced to some extent by the parents' own tendencies to emotionally over- or under-eat, or by their emotional feeding practices. Both might have resulted in parents scoring the two children more similarly, inflating the shared environmental effect (because both twins would be rated similarly regardless of their zygosity). However, this bias should apply equally to other parent-reported child eating behaviours (e.g. food fussiness), yet EOE and EUE show much higher shared environmental influence and much lower genetic influence in comparison to other child eating behaviours (Fildes et al., 2016; Llewellyn, van Jaarsveld, et al., 2010; Smith et al., 2016); suggesting that parents can and do distinguish between their child and their own behaviour. Another way to test the influence of parental emotional eating tendencies on their ratings of child emotional over and under-eating would be to include and compare the maternal ratings with paternal ratings or ratings provided by another carer such as grandparents or teachers. This potential multiple rater twin model would compare the estimates derived from different raters and allow an investigation of potential bias.

As discussed previously in Study 1 and 2 (**Chapter 4**), the prevalence of EOE was fairly low, with about a quarter of the children reported to never engage in this behaviour. On the other hand, EUE was more common and data were close to a normal distribution. For both EOE and EUE, twin pair correlations were very high, resulting in very small estimates of the effect of non-shared environmental factors. This could be seen as an indicator of low validity of the measures in these sample, as mothers might have just scored across their twins, finding it difficult to use the measure appropriately. However previous twin analyses of other subscales of CEBQ have found great differences

between MZ and DZ correlation, e.g. Food fussiness (Fildes et al., 2016) and Satiety and Food responsiveness (Carnell et al., 2008). This variation in MZ and DZ correlation for other eating behaviours on the CEBQ, validates the results presented here, as it seems unlikely that a potential cross-rater bias would only affect one subscale of a longer questionnaire in which items are ordered randomly. In order to examine these issues further, a replication of these analyses in an independent sample is presented in Study 6 (**Chapter 9**). Findings presented there in detail replicate the same pattern of analyses here, however with overall lower estimates of MZ and DZ twin similarity. These slight differences in results suggest that any cross-rater bias is not systemic to the measures, and therefore support their validity.

## 5.6 Conclusions

The study described in this chapter provides evidence that individual differences in EOE and EUE at five years are mainly shaped by environmental factors shared by twin pairs within one family. Heritability was low for both behaviours. EOE and EUE were positively correlated and the correlation was slightly stronger than previously reported. This association was entirely driven by shared environmental influences. However, the aetiology of EOE and EUE was also somewhat distinct, insofar as only some of the shared environmental influences were common to both behaviours. However, although twin research is an excellent method to describe the sources of individual differences in child emotional over and under-eating, it cannot pinpoint specific environmental factors. Research presented in Study 3 (**Chapter 6**), built on the twin studies reported in Study 1 and 2 (**Chapter 4** and **5**), and aimed to identify specific environmental factors associated with childhood emotional over- and under-eating.

# Chapter 6 Study 3: Identifying environmental factors associated with childhood emotional over and under-eating

## 6.1 Introduction

The results described in the first two studies of this thesis suggested that child EOE and EUE are influenced by environmental factors shared within one family. The majority of research into specific environmental shapers of emotional overeating has focussed largely on parental emotional feeding (providing food and snacks to calm a child down and soothe emotions). Evidence for a positive association between parental emotional feeding and child emotional overeating comes from both cross-sectional (Braden et al., 2014) and longitudinal research (Rodgers et al., 2013; Steinsbekk et al., 2017); suggesting that emotional feeding might be the mechanism by which parents 'teach' their children to emotionally overeat.

Parental factors other than feeding practices might also influence child emotional over- and under-eating. In particular, the very early milk-feeding environment could potentially play a role in the development of child emotional overeating and under-eating, but has never been explored. It has been hypothesised that rigid feeding on schedule interrupts an infant's ability to develop good self-regulation skills, which may affect both appetite regulation and emotion regulation. Feeding on a schedule versus feeding in response to an infant's cues for hunger and satiety might therefore play a role in the development of maladaptive emotional over- and under-eating tendencies in early postnatal life. So far no research has investigated this hypothesis. Moreover, breastfeeding has been linked with improved appetite regulation compared to bottle-feeding (DiSantis, Collins, et al., 2011; Hassiotou & Geddes, 2014), and breastfeeding mothers are might be more likely to feed their infant on demand than to keep to a strict schedule; it is possible that breast and bottle feeding are involved in the early development of emotional overeating and under-eating. More research is needed to understand the potential links between the early life feeding environment and child emotional over- and under-eating.

Other parental behaviours have also been linked with child emotional eating. Research has shown that maternal emotional overeating is associated with child emotional overeating, consistent with the hypothesis that parental modelling may be involved in the

inter-generational transmission of emotional overeating (de Lauzon-Guillain et al., 2009; Jahnke & Warschburger, 2008; Snoek et al., 2007). No previous research has tested for the effect of parental emotional eating on child emotional under-eating, but the high shared environmental influence on EUE and EOE established in Study 2, indicate that parental modelling might be of importance to each of these behaviours.

Emotional eating is a response to a stressful environment. Exposure to stress has been associated with increased risk of childhood obesity, and emotional overeating could be one of the behavioural mechanisms that mediates this relationship (S. M. Wilson & Sato, 2014). Experiencing emotional problems and conflict with peers has been associated with emotional overeating in school-aged children (Michels et al., 2012), as well as toddlers (Mallan et al., 2017). Apart from stress experienced outside the home, perceived home stress might be another contributing factor. A straightforward way to investigate this possible link would be the use of established psychometric scales of household chaos, such as the Chaos, Hubbub and Order Scale (CHAOS) (A. P. Matheny et al., 1995). Other factors, in addition to a generally chaotic home life, potentially contribute to a stressful environment within the family home and therefore are also potential risk factors for emotional over- and under-eating. A hostile parental relationship has been associated with child EUE (Haycraft & Blissett, 2010) and witnessing marital conflict was associated with higher emotional overeating in children (Bi et al., 2017). Other factors that contribute to a stressful family life include being of low socio-economic status and growing up in a single-parent household. Furthermore, growing up with a stay-at-home mothers might influence the parent-child relationship which in turn might impact on the child's tendency to emotionally over- and under-eat.

As described in the introduction **Chapter 1.5.3.2.1**, characteristics of the children themselves have also been implicated in the development of emotional eating. Some research has suggested that the child's ability to regulate their emotions is involved in the development of emotional overeating, and this makes intuitive sense – a child who is less adept at regulating his or her own emotions is more likely to engage in maladaptive behaviours such as comfort eating, to soothe their emotions. In particular, as discussed in more detail in **Chapter 1.5.3.2.1** research has shown that children who experience a lot of negative emotions and tend to engage in maladaptive coping strategies such as screaming or withdrawal are more likely to experience changes in appetite in response to stress (Lu, Tao, Hou, Zhang, & Ren, 2016; Powell et al., 2017).

The majority of the literature exploring predictors of emotional eating in childhood consists of cross-sectional studies examining child emotional overeating. There is a dearth of research investigating emotional under-eating. The results presented in Study 2 (**Chapter 5**) indicated that there are both common and trait-specific environmental factors associated with child EOE and EUE. However, previous research has focused on a limited number of potential factors, and no previous studies have aimed to unpick the factors linked to both child emotional over- and under-eating, and those are specific to each.

## 6.2 Aims

This study aimed to identify specific environmental factors associated with emotional over- and under-eating in middle childhood. The following aims were addressed:

1. To identify factors associated with *both* emotional over- *and* under-eating at five years
2. To identify factors associated specifically with emotional overeating and emotional under-eating

## 6.3 Methods

### 6.3.1 Sample

The data analysed in this chapter were from the Gemini cohort, as described in detail in **Chapter 3.1**. The twins were five years old (mean = 5.15 years; SD = 0.13) and the full analysis sample included 1168 individual children from 583 families**.**

### 6.3.2 Measures

*Anthropometric measures*

Age, sex, gestational age and child BMI were parent reported. Standard deviation scores for child BMI (BMI-SDS) were calculated using UK90 reference data (Freeman et al., 1995). In order to achieve the maximum sample size, BMI scores using height and weight data collected closest to the target age five years were used: at 57, 60 or 63 months. If children were missing data for measurements at 57, 60 or 63 months, but had at least three weight measures and two height measures between two to five years, these values were used to impute BMI at five years using interpolation. David Boniface, the departmental statistician conducted the imputation of BMI at five years.

*Emotional overeating and under-eating*

EOE and EUE (four items each) were measured using the Child Eating Behaviour Questionnaire (CEBQ) (Wardle et al., 2001b) when the twins were five years old. A detailed description of this measure can be found in **Chapter 3.2.1**. The full questionnaire can be seen in **Appendix 2.2**.

*Child Emotional Regulation*

The Emotional Regulation subscale of the Strength and Difficulties Questionnaire (SDQ) (Goodman, 2001) was used to measure the child's ability to self-regulate their emotions when they were five years old. The subscale consists of six items (example: "My child is nervous or clingy in new situations"). More details on this measure can be found in **Chapter 3.2.4** including a full list of items.

*Household stress and home environment*

The Confusion, Hubbub And Order Scale (CHAOS) (A. P. Matheny et al., 1995) was used to describe the household stress experienced by a family when the twins were five years old. In this shortened version parents were asked to rate six items (example item: "There is often a fuss going on in our home") when their twins were five years old, choosing between "false" and "true" and the full questionnaire is provided in **Chapter 3.2.5, Table 3.9**.

*Sociodemographic measures*

An in-depth description of the following measures can be found in **Chapter 3.2.9**. Maternal education at baseline was divided into two categories; having a university degree or not. Furthermore, maternal employment at baseline was considered. Mothers were divided into either working (full time or part time) or staying at home (unemployed, maternity leave or decided to be stay-at-home mothers). Additionally, the maternal relationship status at baseline was included; mothers were dichotomised according to whether they reported having a partner ('co-habiting' or 'married') or if they classified themselves as being single ('divorced', 'widowed', 'separated' or 'single').

Socio-economic status of the family at baseline was indexed using the National Statistics Socio-economic Classification (NS-SEC) (Office for National Statistics, 2005), which

codes the occupational status of the highest earner within a family. A reduced version of three categories, low, middle and high, was used to classify the Gemini families.

*Early milk-feeding*

Mothers were asked to indicate their 'feeding philosophy' during the first three months of life, choosing either 'On Demand (e.g. fed baby when he/she cried)' or 'On a schedule (e.g. fed baby at set times)'.

In addition, mothers indicated the extent to which they breastfed their twins choosing from these response options: 'Entirely breastfeeding', 'mostly breastfeeding', 'equally breast- and bottle feeding', 'mostly bottle', 'almost entirely bottle' or 'entirely bottle'. This due to the high number of response option this variable was treated as a continuous measure (range: 1 – 6), whereby a score of 1 implied entirely breastfeeding and 6 indicated entirely bottle feeding. All measures characterising the early feeding environment are described in more detail in **Chapter 3.2.7**.

*Maternal Eating Behaviour*

Mothers of the twins answered the Dutch Eating Behaviour Questionnaire (DEBQ) (Van Strien et al., 1986) when the twins aged two years old. The DEBQ consists of three subscales: Emotional eating, restrained eating and external eating. A detailed description of this questionnaire can be found in **Chapter 3.2.2**. The shortened version that was sent to mothers is attached in **Table 3.8**.

*Parental Feeding Strategies*

As described in detail in **Chapter 3.2.6** a combination from different subscales from different parental feeding questionnaires were used to measure parental feeding strategies of the Gemini parents when the twins were five years old. Parents were asked to rate their own feeding behaviour using a five point Likert-scale ranging from 'disagree' to 'agree' for all scales. Mean scores were created for the different scales (range: 1 – 5). The scales are described again briefly below.

*Pressure to eat*

The Pressure to eat subscale of the Child Feeding Questionnaire (CFQ) (Birch et al., 2001) was included to ascertain the extent to which parents pressure or coerce their child

to eat. The scale consisted of five items (example item: “My child should always eat all of the food on his/her plate”).

*Modelling*

In order to measure how much parents model eating behaviour in order to influence their children, the Modelling subscale was taken from the Comprehensive Feeding Practices Questionnaire (Musher-Eizenman & Holub, 2007). The subscale consisted of four items (example item: “I show my child how much I enjoy eating healthy foods.”).

*Monitoring*

The Monitoring subscale from was taken from the Child Feeding Questionnaire (CFQ) (Birch et al., 2001) Parents used three items to indicate how much they monitor or keep track of their child’s food intake (example item: “I keep track of the foods my child’s been eating when he/she is not with me.”).

*Instrumental Feeding*

All subscales of The Parental Feeding Style Questionnaire (PFSQ) (Wardle et al., 2002) were sent to parents. Instrumental feeding refers to the parents’ tendency to use food in a “means-end” contingency, i.e. offering food as a reward for good behaviour or taking it away to punish poor behaviour (example item: “I reward my child with something to eat when he/she is well-behaved”). The subscale consists of five items.

*Emotional Feeding*

Emotional feeding describes the parent’s tendency to use food to soothe a distressed child. This subscale of the PFSQ consists of five items (example item: “I give my child something to eat to make him/her feel better when he/she is worried.”).

*Encouragement to eat*

This subscale of the PFSQ describes the parent’s tendency to motivate their child to eat healthy food (e.g. fruit and vegetables) and a varied diet. Parents rated this behaviour with four items (example item: “I praise my child if he/she eats a new food.”).

*Control*

The last subscale taken from the PFSQ was parental Control. Parents reported the extent to which they actively attempt to regulate their children's eating on a six-item subscale (example item: "I decide what my child eats between meals.").

*Structured mealtime*

The Structured mealtime subscale was taken from The Pre-schooler Feeding Questionnaire (PFQ) (Baughcum et al., 2001). Parents indicate their implementation of rules around dinner time using three items (example item: "My child watches TV during meals.").

### 6.3.3 Analyses

#### 6.3.3.1 Descriptive analyses: Cross-sectional analyses of emotional over- and under-eating in middle childhood

In the first instance Pearson's correlations were calculated to examine the simple associations between EOE, EUE and the continuous predictors (age, gestational age, child BMI, emotion regulation, maternal eating behaviours, feeding method, chaos in the home, parental feeding strategies). T-tests were used to test for differences in EOE and EUE across dichotomous categorical predictors (sex, feeding routine, maternal education and employment). One-way Analyses of Variance (ANOVA) was used to compare means for variables with more than one category (socio economic status). For these descriptive analyses one twin was randomly selected from each pair to account for the fact that children in this sample are related and therefore not fully independent from another.

#### 6.3.3.2 Aim 1) Identifying factors associated with both emotional over- and under-eating

Factors that were significantly associated with EOE and/or EUE in descriptive analyses were carried forward to examine which factors were associated with *both* EOE and EUE in multiple regression analyses (run as Complex Samples General Linear Models). Separate models were run for EOE and EUE as dependent variables. The following variables were included in each model (model 1): Child emotional regulation, chaos in the home, all maternal eating behaviours, instrumental and emotional feeding, pressure to eat, control and mealtime structure. All analyses controlled for child sex, age, gestational age and 5-year BMI-SDS. Although they were not significantly associated

with EOE and EUE, it is common practice to account for these factors, enabling better comparison with other previous studies. This model allowed me to identify factors associated with *both* EOE and EUE.

#### 6.3.3.3 Aim 2) Identifying factors associated specifically with emotional overeating and emotional under-eating

In order to identify factors associated with EOE and EUE specifically, the other emotional eating behaviour (EOE or EUE) was added in to the model, addition to the variables included in model 1 (model 2). For the model predicting EOE, EUE was added; for the model predicting EUE, EOE was added. This allowed me to identify factors associated uniquely with EOE or EUE, controlling for the correlation between the two behaviours.

All of these analyses were carried out using Complex Samples General Linear Models to account for the clustering of twin pairs. This method enables the whole data set to be used to maximise statistical power to detect significant effects. Due to the large sample size, the alpha level was set to 0.01 in order to reduce the likelihood of Type 2 errors. A lower alpha level ensures that minor and trivial effects do not reach statistical significance; a potential issue in larger samples. A description of this method can be found in **Chapter 3.4.1**.

#### 6.3.3.4 Tests of normality for descriptive analyses

The normality of distributions of continuous variables included in the analyses were investigated by inspecting skew and kurtosis statistics (**Appendix 4.1** displays means, standard deviation, skew and kurtosis). For almost all variables skew and kurtosis were in the acceptable range (between -1 and 1). Age and gestational age were slightly negatively skewed but fairly close to the normal range. Regardless of skew, age, gestational age and BMI-SDS had a more peaked (leptokurtic) distribution (score > 1). However, it has been suggested that with a large sample size higher kurtosis values (<7) are acceptable (H. Y. Kim, 2013).

#### 6.3.3.5 Test of assumptions of complex samples general linear models

As described in **Chapter 3.4.1**, five main assumptions need to be met to justify the use of Complex Samples General Linear Models:

*Linearity*: Visual inspection of scatterplots confirmed the linearity of associations between predictors and dependent variables.

*Independent errors of the residuals*: The Durbin Watson test was used to test for the presence of autocorrelation between the errors of the residuals. The possible test scores range from 0 – 4, with numbers from 1.5 – 2.5 indicating no autocorrelation. The Durbin Watson score was 2.01 meeting this assumption.

*Homoscedasticity*: The variance of the residuals needs to be constant for each level of the dependent variable. Scatterplots were used to judge this assumption.

*Normality of errors*: Visual inspection of the P-P plots of the residuals indicated normality of errors.

*No multicollinearity* (high correlations between variables entered into the regression analyses): The presence of perfect linear correlations between variables included in a regression precludes differentiation between them, violating the assumptions of linear regression analyses. Correlations between predictor variables in these models were all below 0.8, indicating the absence of multicollinearity (Field, 2013). Variance Inflation Factors (Vifs) were < 10 for all variables confirming the absence of multicollinearity.

## 6.4 Results

### 6.4.1 Descriptive statistics: Cross-sectional correlates of emotional over- and under-eating at five years

**Table 6.1** shows the descriptive statistics for all variables and their correlations with EOE and EUE. The table shows the correlation coefficients for the continuous variables and EOE and EUE, and means for EOE and EUE in different groups (sex, maternal education, employment, relationship status and feeding routine, feeding method, socio-economic status).

**Table 6.1 Descriptive statists and associations between child characteristics, maternal behaviours, home environment and parental feeding strategies and child EOE and EUE at five years (n = 573)**

| | Descriptive statistics (n = 573) | | Pearson's Correlation or Mean (SD) | |
|---|---|---|---|---|
| | | **Mean (SD) / N (%)** | **EOE** | **EUE** |
| **Child Characteristics** | **Age (years)** | 5.15 (0.13) | 0.021 | 0.024 |
| | **Gestational age (weeks)** | 36.21 (2.49) | 0.087 | 0.056 |
| | **Sex**<br>Females<br>Males | <br>293 (50.3%)<br>290 (49.7%) | <br>1.54 (0.51)<br>1.57 (0.50) | <br>2.65 (0.84)<br>2.63 (0.82) |
| | **Child BMI-SDS** | -0.247 (1.08) | 0.09 | -0.03 |
| | **Emotion Regulation (SDQ)** | 2.083 (0.785) | **0.135**** | **0.143**** |
| | **Emotional Under-Eating** | 2.64 (0.83) | 0.419** | 1 |
| | **Emotional Overeating** | 1.55 (0.51) | 1 | 0.419** |
| **Home environment** | **CHAOS** | 0.40 (0.33) | **0.187**** | **0.115**** |
| | **Maternal relationship status**<br>With partner<br>Single | <br>583 (97.4%)<br>15 (2.6%) | <br>1.55 (0.51)<br>1.58 (0.54) | <br>2.64 (0.82)<br>2.65 (1.05) |
| | **Maternal Education**<br>University degree<br>No university degree | <br>323 (55.4%)<br>260 (446%) | <br>1.55 (0.52)<br>1.55 (0.49) | <br>2.65 (0.82)<br>2.63 (0.84) |
| | **Maternal Employment**<br>At home[1]<br>Working (full or part time) | <br>485 (83.2%)<br>98 (16.8%) | <br>1.54 (0.49)<br>1.61 (0.57) | <br>2.65 (0.83)<br>2.61 (0.82) |
| | **Socio-Economic Status (NSSEC)[2]**<br>Low<br>Medium<br>High | <br>59 (10.1%)<br>80 (13.8%)<br>442 (76.1%) | <br>1.61 (0.49)<br>1.56 (0.51)<br>1.54 (0.51) | <br>2.57 (0.84)<br>2.69 (0.84)<br>2.63 (0.82) |
| **Maternal Behaviours** | **Maternal Emotional Eating** | 2.14 (0.96) | **0.221**** | **0.132**** |
| | **Maternal Restraint Eating** | 2.70 (0.94) | **0.178**** | 0.101 |
| | **Maternal External Eating** | 3.07 (0.65) | **0.160**** | **0.151**** |
| | **Feeding Method** | 3.21 (1.73) | 0.053 | -0.039 |
| | **Feeding Routine[3]**<br>On demand<br>On schedule | <br>280 (48%)<br>295 (50.6%) | <br>1.57 (0.53)<br>1.53 (0.49) | <br>2.69 (0.85)<br>2.59 (0.80) |

| | | | | |
|---|---|---|---|---|
| **Parental Feeding Strategies** | **Instrumental Feeding** | 2.32 (0.62) | **0.313**** | **0.285**** |
| | **Emotional Feeding** | 1.69 (0.55) | **0.476**** | **0.320**** |
| | **Encouragement** | 4.15 (0.50) | -0.03 | 0.073 |
| | **Pressure to eat** | 2.71 (0.64) | **0.118**** | **0.239**** |
| | **Monitoring** | 3.59 (0.90) | -0.001 | 0.018 |
| | **Modelling** | 3.75 (0.69) | -0.001 | 0.050 |
| | **Control** | 4.15 (0.43) | **-0.159**** | **-0.120**** |
| | **Mealtime Structure** | 4.06 (0.57) | **-.0132**** | -0.072 |

Abbreviations: BMI-SDS, Body Mass Index standard deviation score; SDQ, Strengths and Difficulties Questionnaire; CHAOS, The Confusion, Hubub and Order Scale; NSSEC, National Statistics Socio-economic Classification.

[1] 'At home' category included mothers who were on maternity leave, unemployed, or those who decided to stay at home

[2] Two families with missing data on NSSEC

[3] Eight mothers did not report their feeding routine

** Significance level $p < 0.01$

The patterns of associations were similar for EOE and EUE, suggestive of some common influences. Surprisingly, among the child characteristics emotion regulation ability was found to correlate with both EOE and EUE, indicating that children who are less able to regulate their emotions, display higher levels of emotional over- and under-eating. Most aspects of the early home environment were unrelated to EOE and EUE. However, the CHAOS scale was positively associated with both EOE and EUE, such that greater household stress was linked with more emotional over and under-eating. All maternal eating behaviours were significantly and positively correlated with EOE, and higher maternal Emotional Eating and External Eating were also associated with higher levels of child EUE. No aspect of early milk-feeding was significantly associated with EOE or EUE.

Several of the parental feeding strategies were associated with both EOE and EUE. Strikingly the associations were in the same direction, such that the parental feeding practices that were significantly and positively associated with EOE were also positively associated with EUE (instrumental feeding, emotional feeding and pressure to eat), and parental feeding practices that were negatively associated with EOE were also negatively associated with EUE as well (control and mealtime structure). Mealtime structure was the only parental feeding style that was significantly associated with EOE, but not with EUE. Greater mealtime structure was associated with lower levels of child emotional overeating.

These results are consistent with the hypothesis that children whose parents use food to control their behaviour (higher instrumental feeding) and emotions (higher emotional feeding), exert greater pressure on them to eat, and have less control over mealtimes, are more likely to develop a tendency to both over- and under-eat in response to stress and negative emotions. As described in previous Study 2 (**Chapter 5**)**,** a substantial positive correlation was observed between EOE and EUE.

### 6.4.2 Aim 1) Factors associated with *both* emotional over- *and* under-eating at five years

The complex samples general linear model analyses (model 1) suggested that parental emotional feeding was the only predictor significantly associated with *both* child EOE (B = 0.360, 99% CI: 0.247, 0.474) and EUE (B = 0.292, 99% CI: 0.110, 0.474). Greater emotional feeding was related to both higher EOE and higher EUE in children. A 1-unit

increase in the Emotional feeding scale (e.g. scoring ‘sometimes’ versus ‘rarely’) was associated with an increase of 0.36 units on the EOE scale. The effect was slightly smaller for emotional under-eating – a 1-unit increase in Emotional feeding was associated with a 0.29 unit increase in EUE. Parental pressure to eat was significantly positively associated with child EUE only, and the effect was smaller than that for Emotional Feeding (B = 0.192, 99% CI: 0.043, 0.341).

Parental emotional overeating was positively significantly associated with child EOE, but the effect was much smaller than that for parental emotional feeding (B = 0.062, 99% CI: 0.004, 0.120). A 1-unit increase in parental emotional overeating (e.g. scoring ‘always’ versus ‘often’) was only associated with an increase of 0.06 units on the EOE scale. Parental emotional overeating was not significantly associated with child EUE. No other associations were statistically significant. Model 1 explained more variance in EOE (27%; $R^2 = 0.266$) than in EUE (15%; $R^2 = 0.154$). All outcomes are displayed in **Table 6.2**.

**Table 6.2. Model 1: Results of complex samples general linear modelling predicting *both* EOE and EUE at 5 years (n = 1168)**

| | EOE | | EUE | |
|---|---|---|---|---|
| | **B (99% CI)** | **p-value** | **B (99% CI)** | **p-value** |
| **Child age** | 0.011 (-0.387, 0.408) | 0.944 | -0.052 (-0.677, 0.573) | 0.829 |
| **Gestational age** | 0.011 (-0.008, 0.030) | 0.143 | 0.009 (-0023, 0.041) | 0.457 |
| **Sex** | 0.022 (-0.056, 0.099) | 0.472 | -0.034 (-0.166, 0.099) | 0.510 |
| **Child BMI-SDS** | 0.026 (-0.017, 0.068) | 0.177 | -0.21 (-0.091, 0.050) | 0.453 |
| **Child Emotion Regulation** | 0.046 (-0.017, 0.109) | 0.061 | 0.069 (-0.026, 0.163) | 0.062 |
| **DEBQ Emotion overeating** | **0.062 (0.004, 0.120)** | **< 0.01** | 0.034 (-0.068, 0.137) | 0.390 |
| **DEBQ Restraint** | 0.025 (-0.027, 0.078) | 0.212 | 0.007 (-0.096, 0.109) | 0.864 |
| **DEBQ External Eating** | -0.005 (-0.088, 0.077) | 0.866 | 0.055 (-0.094, 0.204) | 0.342 |
| **Instrumental Feeding** | 0.048 (-0.053, 0.150) | 0.219 | 0.118 (-0.056, 0.292) | 0.080 |
| **Emotional Feeding** | **0.360 (0.247, 0.474)** | **< 0.01** | **0.292 (0.110, 0.474)** | **< 0.01** |
| **Pressure to eat** | -0.019 (-0.101, 0.063) | 0.554 | **0.192 (0.043, 0.341)** | **< 0.01** |
| **Control** | -0.047 (-0.172, 0.082) | 0.344 | -0.104 (-0.330, 0.123) | 0.237 |
| **Mealtime structure** | 0.007 (-0.092, 0.106) | 0.862 | 0.003 (-0.162, 0.167) | 0.968 |
| **CHAOS** | 0.128 (-0.023, 0.280) | 0.029 | 0.094 (-0.176, 0.264) | 0.368 |
| **$R^2$** | 0.266 | | 0.154 | |

Abbreviations: BMI-SDS = Body Mass Index Standard Deviation Score; SDQ = Strength and Difficulties questionnaires DEBQ = Dutch Eating Behaviour Questionnaire; EOE = Emotional Overeating, CHAOS = The Confusion, Hubub and Order Scale $R^2$ = coefficient of variance explained

### 6.4.3 Aim 2) Identifying factors associated *specifically* with emotional overeating and emotional under-eating

After EUE was entered into the model (model 2), the significant predictors for EOE remained the same, but the effect sizes were slightly attenuated. Parental emotional feeding was still significantly positively associated with child EOE (B = 0.308, 99% CI: 0.207, 0.410). A 1-unit increase in the Emotional feeding scale (e.g. scoring 'sometimes' versus 'rarely') was associated with an increase of 0.31 units on the EOE scale. The association between maternal emotional overeating and child EOE remained significant but small (B = 0.054, 99% CI: 0.001, 0.106). A 1-unit increase of maternal emotional eating was associated with a small increase on EOE by 0.05. As expected, EUE itself was significant and positively associated with EOE (B = 0.176, 99% CI: 0.123, 0.228). A 1-unit increase on the EUE scale was associated with a 0.18 unit increase in EOE. Together, all variables explained 34% of the variance in EOE ($R^2$ = 0.337).

Regarding EUE, after controlling for EOE (model 2), parental emotional feeding was no longer associated with EUE. Parental pressure to eat remained uniquely associated with child EUE (B = 0.197, 99% CI: 0.057, 0.337). That meant, that a 1-unit increase (e.g. scoring 'always' versus 'often') on the Parental pressure to eat scale was associated with a 0.20 increase on the EUE scale. EOE was the strongest predictor of EUE (B = 0.549, 99% CI: 0.391, 0.708). A 1-unit increase in EOE was associated with a 0.55 increase of EUE. Together, all variables explained 24% of the variance in EUE ($R^2$ = 0.236). The findings from Model 2 are displayed in **Table 6.3**.

**Table 6.3 Model 2: Results of complex samples general linear modelling predictors *independently associated* with EOE and EUE at 5 years (n = 1168)**

| | EOE | | EUE | |
|---|---|---|---|---|
| | **B (99% CI)** | **p-value** | **B (99% CI)** | **p-value** |
| **Child age** | 0.020 (-0.354, 0.394) | 0.890 | -0.058 (-0.644, 0.528) | 0.798 |
| **Gestational age** | 0.009 (-0.009, 0.028) | 0.195 | 0.003 (-0.027, 0.034) | 0.781 |
| **Sex** | 0.028 (-0.046, 0.101) | 0.331 | -0.046 (-0.171, 0.080) | 0.347 |
| **Child BMI-SDS** | 0.029 (-0.011, 0.070) | 0.059 | -0.035 (-0.1,02, 0.032) | 0.182 |
| **Child Emotion Regulation** | 0.025 (-0.028, 0.078) | 0.229 | 0.048 (-0.043, 0.140) | 0.170 |
| **DEBQ EOE** | **0.054 (0.001, 0.106)** | **< 0.01** | 0.001 (-0.095, 0.098) | 0.971 |
| **DEBQ Restraint** | 0.028 (-0.020, 0.076) | 0.128 | -0.009 (-0.106, 0.087) | 0.803 |
| **DEBQ External Eating** | -0.019 (-0.093, 0.056) | 0.514 | 0.060 (-0.080, 0.200) | 0.268 |
| **Instrumental Feeding** | 0.023 (-0.068, 0.115) | 0.511 | 0.094 (-0.071, 0.259) | 0.142 |
| **Emotional Feeding** | **0.308 (0.207, 0.410)** | **< 0.01** | 0.094 (-0.085, 0.274) | 0.175 |
| **Pressure to eat** | -0.043 (-0.113, 0.028) | 0.118 | **0.197 (0.057, 0.337)** | **< 0.01** |
| **Control** | -0.020 (-0.140, 0.100) | 0.662 | -0.083 (-0.301, 0.136) | 0.329 |
| **Mealtime structure** | -0.007 (-0.096, 0.082) | 0.837 | 0.006 (-0.150, 0.162) | 0.919 |
| **CHAOS** | 0.096 (-0.042, 0.235) | 0.073 | 0.032 (-0.220, 0.284) | 0.742 |
| **CEBQ EUE** | **0.176 (0.123, 0.228)** | **< 0.01** | N/A | |
| **CEBQ EOE** | N/A | | **0.549 (0.391, 0.708)** | **< 0.01** |
| **$R^2$** | 0.337 | | 0.236 | |

Abbreviations: BMI-SDS = Body Mass Index Standard Deviation Score; SDQ = Strength and Difficulties questionnaires DEBQ = Dutch Eating Behaviour Questionnaire; EOE = Emotional Overeating, CHAOS = The Confusion, Hubub and Order Scale $R^2$ = coefficient of variance explained

## 6.5 Discussion

### 6.5.1 Summary of findings

Descriptive univariate analyses showed that both EOE and EUE were both positively and negatively associated with a variety of child, parental and home environmental factors. Most of the significant associations were similar for EOE and EUE. Both behaviour were positively correlated with emotion regulation. Furthermore, positive association were found between EOE, EUE and these parental feeding behaviours: instrumental feeding, emotional feeding, and pressure to eat. There were significant negative correlations between EOE, EUE and parental control. Chaos in the home was positively associated with EOE and EUE. All maternal eating behaviours were positively correlated with EOE and EUE, apart from Restraint which was not associated with EUE. These significant correlates were carried forward into multivariate analyses to address the research aims of this study. The first research aim was:

> To identify factors associated with *both* emotional over- *and* under-eating at five years

Complex samples general linear models were used to establish which of the correlates relate to *both* child EOE and EUE in multivariable analyses. Results revealed parental emotional feeding was associated with *both* child EOE and EUE. Maternal emotional eating was significantly associated with child EOE only, whereas parental pressure to eat only related to child EUE. All other variables were found not be significantly associated, suggesting that many of the discussed univariate associations are explained or attenuated by relationships between the predictor variables.

The second research aim was:

> To identify factors associated specifically with emotional overeating and emotional under-eating

Adjusting for the EOE or vice versa for EUE changed the outcomes. After controlling for EUE, parental emotional feeding remained independently positively associated with EOE but effect size was slightly attenuated. Furthermore, the effect of maternal emotional eating was significant but small.

Before controlling for EOE, emotional feeding *and* parental pressure to eat were both significantly associated with EUE. Pressure to eat remained significantly associated with EUE once EOE was controlled for, indicating that parental pressure to eat was specifically associated with EUE.

### 6.5.2 Implications

Univariate correlations in this study suggested significant positive association between emotional regulation and EOE and EUE. This association has been brought forward by previous research, with cross-sectional studies suggesting that maladaptive emotional regulation, such as suppression of emotions was associated with increased emotional overeating and intake in energy rich foods in Chinese adolescents (n = 4316) (Lu et al., 2016). In contrast, findings presented here indicated that once other factors such as emotional feeding were considered, the association between emotion regulation, EOE and EUE became non-significant, indicating that the significant correlations in the univariate analyses might be spurious.

Similarly, the significant correlation between household stress on EOE and EUE did not survive the adjustment for the other associated variables. This findings can be seen as an indication that a stressful home might be a reflection of maladaptive emotional feeding practices.

In line with previous literature the findings of this study supported the importance of emotional feeding in the development of EOE in childhood proposed in both in longitudinal (Steinsbekk et al., 2017) and cross-sectional studies (Blissett et al., 2010; Braden et al., 2014; Tan & Holub, 2015). The Psychosomatic Theory of obesity (Kaplan & Kaplan, 1957) proposes that obesity is caused by emotional overeating which is learned in early life through a maladaptive pairing of stress cues and food consumption. The importance of emotional feeding in the context of child emotional overeating, provides support for this theory.

In addition, maternal emotional eating remained specifically associated with EOE to a small degree, supporting the hypothesis that maternal modelling might be an important influence on the development of child EOE. Results from Studies 1 and 2 showed that genetic effects play only a minor role in the aetiology of EOE. Therefore, the association between maternal and child emotional overeating is likely to be due to behavioural modelling, rather than intergenerational transmission of genes.

Previous research has also aimed to incorporate both emotional feeding and maternal emotional overeating into one model of child emotional overeating. A cross-sectional analysis of 306 Australian mothers and their two year old children included measures of maternal and child emotional overeating as well as maternal emotional feeding (Rodgers et al., 2014). Results from structural equation modelling supported an association of parental emotional feeding on child emotional eating, as well as a smaller association between maternal emotional overeating and child emotional overeating. Additionally, the model found evidence for a significant association between maternal emotional eating and emotional feeding, suggesting that mothers who are prone to engage emotional overeating might also be more likely to use emotional feeding practices (Rodgers et al., 2014). These results highlight the interrelationships between maternal and child emotional eating and emotional feeding, in line with the results in Study 3.

This is the first study that found a significant associational between parental pressure to eat and EUE. However, parental pressure to eat has been studied previously with the majority of the literature has focused on its association with child BMI, suggesting that parents of children with a low BMI respond with applying more pressure on them to increase their food intake (Gregory, Paxton, & Brozovic, 2010; Spruijt-Metz, Lindquist, Birch, Fisher, & Goran, 2002). Here, child BMI was controlled for in the analyses. Therefore the association between parental pressure to eat and child EUE is independent of the child's weight. Previous studies have also investigated associations between parental pressure to eat and child eating behaviours. As part of the Physical Exercise and Appetite in Children Study (PEACHES), mothers (n = 213) rated their tendency to pressure their child to eat, as well as their child's (eight years) eating behaviours (Webber, Cooke, Hill, & Wardle, 2010). Results showed that, after controlling for child BMI, parental pressure to eat was positively associated with greater food avoidant behaviours (Food fussiness, Satiety responsiveness) and negatively associated with Enjoyment of food. Analyses did not include child EUE, however EUE tends to be positively associated with the other food avoidant behaviours.

Very little research has focussed on the predictors of emotional under-eating in either adults or children. In addition to the association between parental pressure to eat and EUE found in the present study, family relationships have also been linked to EUE in childhood. A study of 156 mothers found that children of parents who had a warm and positive relationship with one another engaged less in EUE than children whose parents

had a more hostile relationship (Haycraft & Blissett, 2010). Maternal warmth and the nature of the parent-child relationship were not included in these analyses, but are potential mechanisms underlying the development of emotional under-eating. More research is needed to explore these hypotheses.

Finally, results in this study propose that emotional feeding was associated with both EOE and EUE. This finding was surprising, as it seemed counter-intuitive that emotional feeding could be related to a child's tendency to *both* over- and under-eat in response to stress. One tentative explanation might be that this association reflects the 'success' of parental emotional feeding practices. Imagining a hypothetical scenario in which a child is upset or unhappy. The mother decides to use her child's favourite snack to change her child's mood. Now, there is two possible outcomes; the child could either respond to the emotional feeding attempt and eat the snack or refuse it. When afterwards the mother is asked to rate her child's tendency to emotionally over- or under-eat, the child's response to the mother's pervious emotional feeding is likely to influence her opinion. If the child responded to the emotional feeding, the mother might be more likely to rate her child as an emotional overeater. If her child refused to respond to the emotional feeding attempt, the mother might rate her child to be an emotional under-eating. This mechanism would be one potential explanation of how one feeding behaviour can be associated with two seemingly opposite eating behaviours. Differences in child's response to emotional feeding might arise from a variety of factors, for example the intensity of the stressor. It is important to keep in mind that these tentative conclusions are based on cross-sectional data and a lot more research is needed to fully understand the causal links between parental feeding, EOE and EUE.

In order to summarise the results of this chapter **Figure 6.1** was created. It illustrates the findings from Models 1 and 2, highlighting that: (i) EOE and EUE are correlated (overlap), (ii) emotional feeding influences EOE as well as the covariance between EOE and EUE, (iii) parental pressure is uniquely associated with EUE, (iv) maternal emotional eating is uniquely associated with EOE.

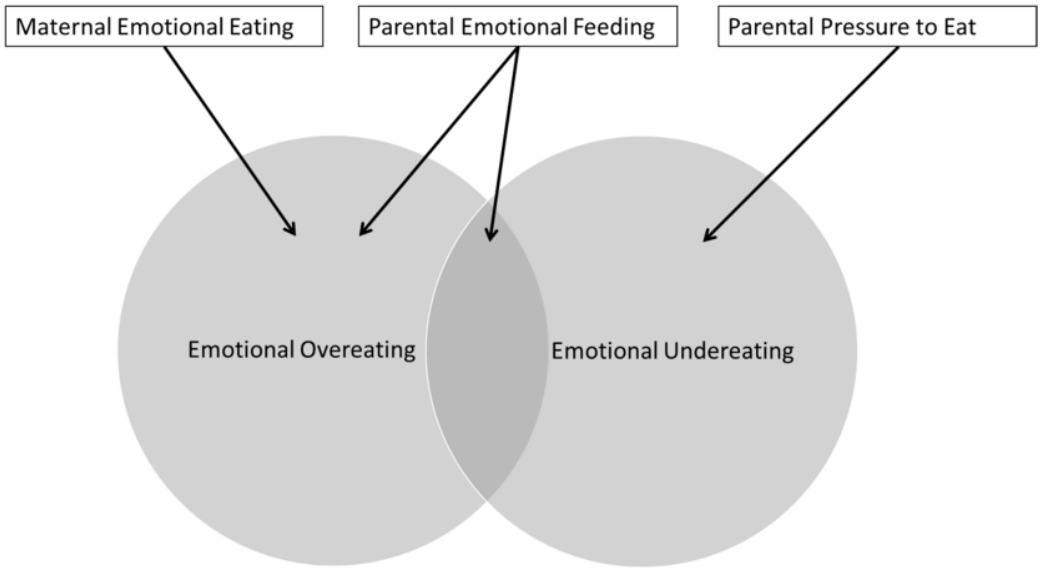


**Figure 6.1 The relationship between parental emotional feeding, parental pressure and maternal emotional eating to eat their influence on child EOE and EUE**

### 6.5.3 Strengths and limitations

In order to make true comparisons between EOE and EUE, only children with complete data on all variables were included. This approach decreased the sample size, which reduced statistical power. Furthermore, almost all measures were parent rated. This might have introduced bias, as mothers rate their child's eating behaviour, their own eating behaviour, as well as their feeding practices. Mothers might have been cross-rating across the different scales, potentially blurring the lines between their own and their twins' behaviour. The fact that the same person rated all three constructs might have contributed to the correlations between these three dimensions. Future research would benefit from collecting measures from multiple raters, and objective measures. However, when collecting data from large cohorts such as Gemini, data collection needs to be pragmatic. Using concise questionnaires guarantees the greatest response rate, and is low cost. Moving forward, as the children grow older, including self-rated questionnaires for the children would be one way to reduce some of the potential bias introduced through parent-report; although child reports are also subject to their own bias.

Even though variables spanned a fairly large spectrum of potential factors, from indicators of socio-economic status, parental eating behaviours and parental feeding behaviours, it is likely that other important factors were not included. For example, parents indicated their perceived household stress within the family. However the twins' experience of the household might be different. Therefore future research would benefit from including data on the child's experience of environmental stressors. Furthermore, by five years of age, children already spend significant parts of their lives outside the family home attending school or in the company of other caregivers or friends. Gemini does not include any information about the stressful situations that a child might experience in these other environments, such as conflicts with peers or teachers. A previous study (age = 5 - 12 years) examined the impact of daily life stress as measured by children themselves with emotional overeating. The results indicated that daily life hassles, problems and stressful life events were significantly correlated with emotional overeating (Michels et al., 2012). Future research in middle childhood should consider a more child-centred approach in addition to parent-rated questionnaires.

Additionally, previous studies (Duke, Bryson, Hammer, & Agras, 2004; Loth, MacLehose, Fulkerson, Crow, & Neumark-Sztainer, 2013) have highlighted that low income and non-white parents engage more in pressuring feeding behaviours. Lower SES families might

also be more likely to experience stressful home environments. The Gemini sample is predominately white and the majority of families are of higher socio-economic status, limiting the generalisability of these findings. This is likely to be due partly to the longitudinal nature of the cohort, with high income families being more likely to continue to participate as the study progresses. Future research should make efforts to include data from more diverse samples.

Many of the socio-demographic variables were only collected at baseline, such as maternal education, maternal relationship status and maternal employment. However many changes could have occurred between then and when the twins were five years old. The fact that the measures were not all collected in parallel may have attenuated the associations, resulting in non-significant findings.

In addition to the maternal rating of the child EOE and EUE, mothers also rated their own feeding practices. As highlighted in **Chapter 3.2.6**, some of the parental feeding measures had low internal reliability, indicated by Cronbach alphas < 0.70. For instrumental feeding, encouragement to eat, control, parental pressure to eat, the Cronbach's alphas were between 0.6 than 0.7. In order to reduce the length of the questionnaire booklet sent to parents, some of the subscales were shortened, and a low number of items can result in a decrease of Cronbach's alphas. Mealtime structure (from the Preschool Feeding Questionnaire, Baughcum et al, 2001) had the lowest reliability (alpha = 0.43), but it was included to create a broader picture of the family eating environment, as the items on this scale are unique and not covered by any of the other scales. Nevertheless, it was not associated with either EOE or EUE in the multivariable models. This finding should be interpreted with caution due to the low reliability of the scale. In contrast, the scale measuring parental emotional feeding showed good reliability: alpha = 0.79. This may be one of the reasons why a significant association was observed between this scale and both EOE and EUE in Model 1.

Finally but most importantly, the analyses in this chapter were cross-sectional, precluding any inferences regarding causality. Longitudinal studies with repeated measures of both the exposures and the outcomes (EOE and EUE) are needed to investigate the causal relationship between child eating behaviours, parental behaviour and household stress. This is crucial as the direction of causation between parental feeding and child eating has been debated. Previous longitudinal studies have suggested that parental feeding

behaviour predicts child eating behaviours (Faith, Scanlon, Birch, Francis, & Sherry, 2004), whereas others have suggested a child-responsive model whereby parents develop their feeding practices in response to their child's emerging eating behaviour (Harris, Fildes, Mallan, & Llewellyn, 2016). Regarding emotional eating, Rodgers et al (2013) suggested a bidirectional link between parental emotional feeding to child emotional eating measured when children were two years old and again one year later. Only one recent longitudinal study has specifically investigated the direction of causation between emotional feeding and child EOE, confirming this reciprocal relationship in middle to later childhood (six, eight and ten years) (Steinsbekk et al., 2017).

## 6.6 Conclusions

Outcomes from this study aimed to identify environmental correlates associated with *both* emotional over *and* under-eating as well as environmental factors *specific* to each. Results indicated that parental emotional feeding is associated with both EOE and EUE, when controlling for other child characteristics and parental and home environmental influences. Maternal emotional eating was related specifically to EOE, and parental pressure to eat was independently related to EUE. However, these analyses were cross-sectional and no causal inference can therefore be made. In order to understand the causal relationship between parental feeding behaviour and child emotional over- and under-eating, longitudinal studies with repeated measures of both are needed. Research described in the following study (Study 4, **Chapter 7**) exploited longitudinal data of both child emotional overeating and parental emotional feeding to investigate the direction of causation between them.

# Chapter 7 Study 4: Testing for reciprocal effects between child emotional overeating and parental emotional feeding using longitudinal prospective data

## 7.1 Background

Study 3 (**Chapter 6**) established that parental emotional feeding was associated with both EOE and EUE cross-sectionally in Gemini, consistent with the hypothesis that it is one of the shared environmental influences on emotional overeating in childhood. However, the cross-sectional nature of the analyses prevent any inferences being made about the causal direction between emotional eating and parental feeding.

Two previous longitudinal cohort studies investigated the relationship between parental emotional feeding and child emotional overeating. A study following 222 Australian parents and their two-year-old children tested the effect of parental feeding on child eating behaviour and vice versa one year later (Rodgers et al., 2013). Results suggested that emotional feeding predicted higher EOE one year later. However, a significant but smaller association was also found between child EOE and later parental emotional feeding, suggesting a potential bi-directional relationship (Rodgers et al., 2013).

More recently, Steinsbekk et al (2017) tested the reciprocity between parental emotional feeding and child emotional eating, using a larger sample (n = 801) of Norwegian families with measures of child EOE and parental emotional feeding at three time-points: aged six, eight and ten years (Steinsbekk et al., 2017). Significant reciprocal paths between parental emotional feeding and child EOE were found across all time-points, suggesting a complex interrelationship between child emotional overeating and parental emotional feeding whereby they influence one another. These two previous longitudinal studies are in need of replication, and only one (Steinsbekk et al, 2017) used a statistical method that enabled the researchers to test for reciprocal effects – structural equation modelling allowed the researchers to directly compare the strength of cross-lagged associations. However, previous studies have either focussed on early childhood (two years) or middle to later childhood (six, eight and ten years), and research is missing investigating the development of the emotional feeding- emotional eating relationship in between: from early into middle childhood, utilising longer follow-up times. In addition, there have been no longitudinal investigations using British children. However, given the many differences

in parenting across cultures, it is not possible to extrapolate findings from non-British studies to British families. A study of British children would provide important information on the likely causal relationship between parental emotional feeding and child emotional eating that is relevant and useful for informing UK guidelines for parents.

Investigating longitudinal bi-directional associations requires that: (i) the data are prospective with repeated measures collected at more than one time point for each variable of interest; and (ii) structural equation modelling (SEM) is used to compare longitudinal paths going in opposite directions (from parent to child, and from child to parent behaviours). SEM allows researchers to directly compare the strength of the association in each direction simultaneously, which offers advantages over running two separate regression analyses. The Gemini study has collected repeated measures for child EOE and parental emotional feeding at 16 months and five years, making it the ideal dataset to address this question using SEM.

## 7.2 Aim

The aim of this study was to explore the longitudinal bi-directional relationship between child EOE and parental emotional feeding measured when children were 16 months and five years old. The following research question was addressed:

> What is the direction of the relationship between parental emotional feeding and child emotional overeating in early life?

## 7.3 Methods

### 7.3.1 Participants

Data from the Gemini cohort were analysed, including 821 children who had data available for EOE (16 months and five years), parental emotional feeding (16 months and five years) and all covariates: sex, gestational age, weight-SDS scores at 16 months, BMI-SDS at five years, and age at measurements. A description of the Gemini cohort can be found in **Chapter 3.1**.

### 7.3.2 Measures

Child EOE was measured with the Child Eating Behaviour Questionnaire and its version adapted for toddlers (Wardle et al., 2001). Parental emotional feeding was measured

using the subscale of Parental Feeding Style Questionnaire (PFSQ) (Wardle et al., 2002). Covariates were parent reported child sex, age at both measurement time points, weight-SDS at 16 months and BMI-SDS at five years. A more detailed description of all these measures can be found in **Chapter 3.2.1**.

### 7.3.3 Analyses

A cross-lagged SEM was used to estimate the effects of early child EOE on later parental emotional feeding and vice versa. Findings were adjusted to account for the clustering of twins within families. Child EOE and parental emotional feeding were allowed to correlate cross-sectionally at each time point of data collection (16 months and five years). The model also estimated the extent to which EOE at 16 months predicted EOE at five years, as well as how parental emotional feeding at 16 months predicted parental emotional feeding at five years (i.e. the within-trait longitudinal associations). The associations of interest were the cross-lagged associations between: child EOE at 16 months and parental emotional feeding at five years; parental emotional feeding at 16 months and child EOE at five years. Age at measurement, gestational age, weight at 16 months and BMI at five years were entered as covariates. All analyses were conducted in R using the statistical package lavaan (Rosseel, 2012). More details regarding this method can be found in **Chapter 3.4.2**. An alpha level of 0.01 was chosen for these analyses with 99% Confidence Intervals. Choosing a more stringent alpha level reduced the likelihood of making a Type 2 error, given the large sample size.

## 7.4 Results

### 7.4.1 Descriptive statistics

Descriptive statistics of the sample included in this analysis can be found in **Table 7.1**.

**Table 7.1 Descriptive statistics for analysis sample for all variables included (n=821)**

| Sample Characteristics | Mean (SD) / N (%) |
|---|---|
| **Sex** | |
| female | 417 (50.8%) |
| **Gestational age (weeks)** | 36.06 (2.65) |
| **Weight-SDS at 16 months** | -0.05 (1.06) |
| **BMI-SDS at 5 years** | -0.23 (1.07) |
| **Age at 16 months (months)** | 15.56 (0.70) |
| **Child EOE at 16 months** | 1.60 (0.61) |
| **Parental emotional feeding 16 months** | 1.96 (0.73) |
| **Age at five years (years)** | 5.15 (0.13) |
| **Child EOE at five years** | 1.54 (0.50) |
| **Parental emotional feeding at five years** | 1.68 (0.55) |

Abbreviations: Weight-SDS = Weight Standard Deviation Score; BMI-SDS = Body Mass Index Standard Deviation Score; EOE = Emotional Overeating

### 7.4.2 Longitudinal analysis

The cross-lagged structural equation model is depicted in **Figure 7.1.** The cross-sectional correlations between EOE and parental emotional feeding were small but significant at both ages (16 months: r = 0.187; five years: r = 0.077). Parental emotional feeding tracked strongly from 16 months to five years (B = 0.381, 99% CI: 0.288, 0.474), and EOE tracked moderately from 16 months to five years (B = 0.175, 99% CI: 0.071, 0.280). Only the cross-lagged path from parental emotional feeding to child EOE (B = 0.146, 99% CI: 0.053, 0.239) was significant, indicating that parental emotional feeding at 16 months predicted child EOE at five years. The path from child EOE at 16 months to later parental emotional feeding (B = 0.076, 99% CI: -0.027, 0.180) was not significant.

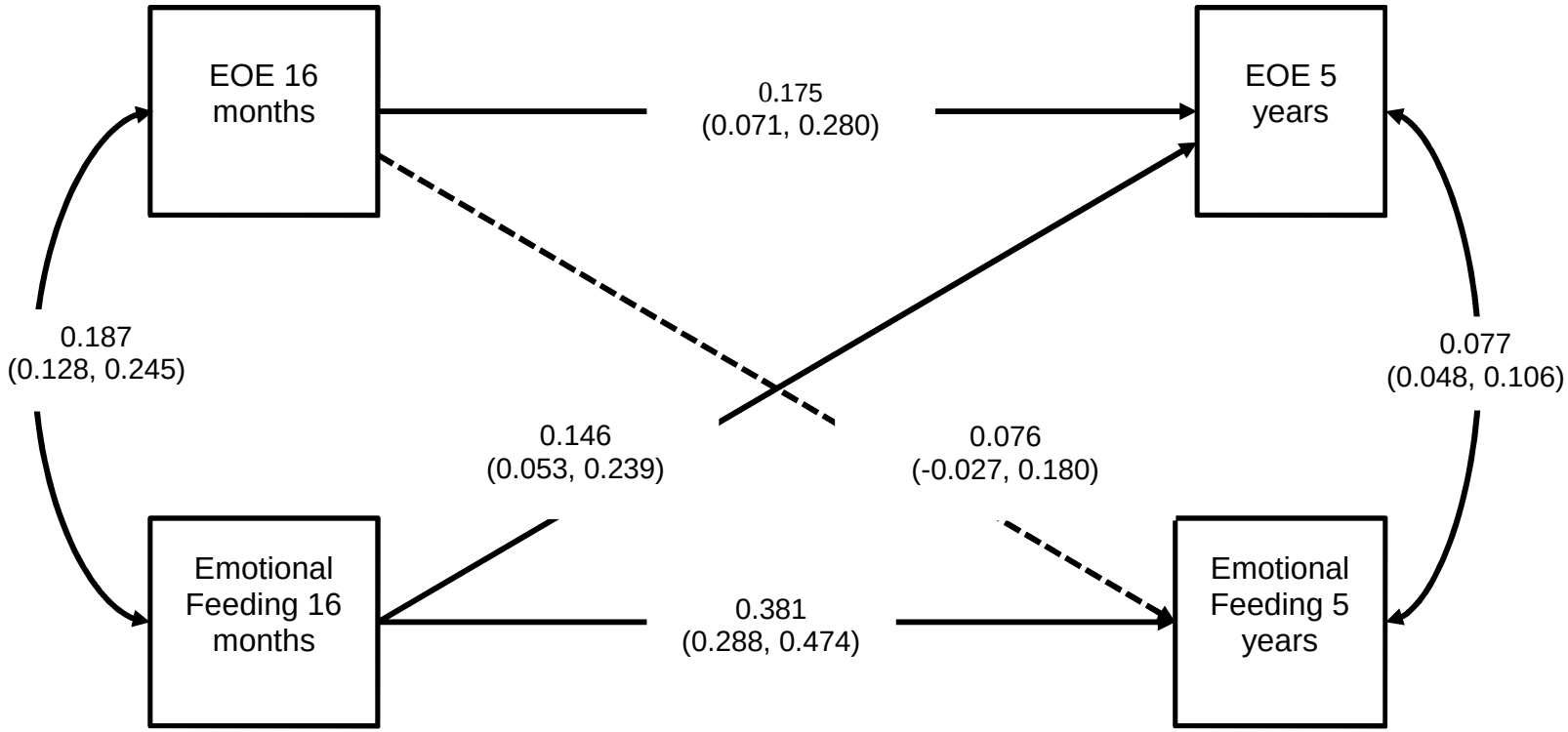


**Figure 7.1 Structural equation model depicting the longitudinal association between child EOE and parental emotional feeding (n = 821).**

*Double ended arrows represent correlations, whereas single headed arrows depict regression coefficients. Dotted lines indicate non-significant paths (p>0.01). Analysis was adjusted for age, sex, and gestational age, weight-SDS at 16 months and BMI-SDS at 5 years, as well as for clustering of twins in families.*

## 7.5 Discussion

### 7.5.1 Summary of findings

This study aimed to answer the following research question:

> What is the direction of the relationship between parental emotional feeding and child emotional overeating?

The findings showed no evidence for a child driven relationship between child EOE and parental emotional feeding from 16 months to five years: child EOE at 16 months did not predict later parental emotional feeding. However, the reverse relationship was significant: greater parental emotional feeding during toddlerhood significantly predicted higher EOE scores four years later. This study provides further evidence of the importance of parental emotional feeding in the development of child EOE.

### 7.5.2 Implications

In contrast to these findings, Steinsbekk et al (2017) reported a reciprocal association from parental emotional feeding to child EOE and vice versa from six to eight, and eight to ten years of age (Steinsbekk et al., 2017). The study presented here (n = 821) and the previous one by Steinsbekk and colleagues were of comparable size (n = 801) and included the same measures of child emotional overeating and parental emotional feeding. The main difference between the studies was the ages of the children. Here the focus was on toddlerhood and middle childhood and analyses spanned a period of significant early life development, whereas Steinsbekk et al (2017) included older primary-school aged children. While their results indicated reciprocal paths between child EOE and parental emotional feeding, the paths from parental feeding to child EOE were strongest and of similar magnitude to those presented here (B range = 0.09 – 0.15). Moreover, their model also included measures of child negative affect measured at four years. Results showed that child temperament influenced both child emotional overeating and parental feeding, such that child negative affect was associated with increased EOE and parental emotional feeding. This finding suggests that other child factors may interact to determine the relationship between child eating and parental feeding.

Child eating behaviours are already emerging in very early childhood (Ashcroft et al., 2008); studies investigating early developmental periods are therefore necessary to understand their aetiology more fully. The results presented here do not support the hypothesis of bi-directional associations between child emotional eating and parental emotional feeding in the first five years. Nevertheless, it is possible that with time EOE

and parental emotional feeding begin to reinforce each other, becoming reciprocal as the child matures. This may happen once the child's tendency to emotional overeat has become more established, and predictable to the parent. A similar study by Rodgers et al (2013) found that parental emotional feeding at two years predicted child emotional overeating one year later. This study was smaller (n = 222) and had a fairly short follow up time. Additionally, a significant bi-directional association was found from child emotional overeating to parental emotional feeding (Rodgers et al., 2013). However, separate multiple regression analyses were used in this study, precluding a direct comparison of the path estimates within one model. The analyses presented here used a structural equation modelling approach, which allows the strength of the opposite paths to be directly compared, while accounting for correlations of predictor and outcome at both baseline and follow-up. This method therefore provides a more stringent approach.

Next to the cross-lagged paths, the longitudinal tracking of child EOE and parental emotional feeding was moderate. In line with previous research indicating the stability of eating behaviours across childhood (Ashcroft et al., 2008), EOE was found to track moderately from 16 months to five years. Nevertheless, this tracking is quite striking given the considerable developmental changes that occur between 16 months and five years of age; the two ages are characterised by distinct food environments, and greatly increased independence from 16 months to five years. At five years of age, children attend school and can verbalise what they would like to eat and make more active choices regarding how much and what type of foods they eat. The even higher association between emotional feeding at 16 months and emotional feeding at five years highlights the persistence of parental feeding strategies across childhood. This finding supports previous studies of the stability of emotional feeding across time-points (Steinsbekk et al., 2017), and implies that once emotional feeding strategies are established parents are likely to continue to use these throughout a child's early development.

The results of this study suggest that interventions targeting parental emotional feeding might be a successful way to address emotional overeating in children, particularly in early life. Parents are in need of clear guidance about the use of food to soothe their children and alternative positive feeding strategies are in need of exploration and promotion. Additional research is needed to understand better when and why parents engage in emotional feeding, in order to design effective interventions and guidelines.

Previous trials have aimed to educate mothers and encourage positive parental feeding styles. Of note is the NOURISH trial in Australia, which included 698 mothers with four months old children (L. A. Daniels et al., 2009). Mothers randomised into the intervention group were enrolled in an extensive training programme which aimed to promote positive parental feeding practices and child food preferences. Modules included group sessions covering the theory underlying child feeding as well as advice regarding responsive feeding, positive parenting and modelling healthy eating patterns. Outcomes suggested that the intervention was successful in promoting positive parental feeding strategies. At 20 months follow-up, mothers in the intervention group were found to engage less in maladaptive feeding practices such as pressure to eat, encouragement, instrumental feeding and emotional feeding in comparison to mothers in the control group. Furthermore, the intervention was found to be successful in reducing the child's EOE two years after the parents participated in the initial programme (L. A. Daniels et al., 2014). Studies like the NOURISH trial provide evidence that parental intervention has the potential to change parental feeding strategies, which in turn can impact the development of child emotional eating. Overall the results from this study confirm the importance of parental emotional feeding in the development of child emotional overeating, supporting the use of trials aiming to modify parental behaviours to encourage healthy development of their children.

### 7.5.3 Strengths and limitations

The SEM approach is a robust research design. It enables exploration of the direction of associations between child eating and parental feeding as it allows direct comparisons between cross-lagged paths within one model. Furthermore, the analysis sample used in this study was large and SEM allows the inclusion of the maximum number of participants by adjusting for clustering of data collected in families.

This study addresses an understudied developmental phase – toddlerhood – adding to two existing longitudinal studies. However, as discussed previously, all measures are rated by the same person, which may have increased the risk of bias, resulting in inflated correlations between child EOE and parental emotional feeding. Objective or multiple-rater measures would be beneficial carrying this work forward, but these are time-consuming, expensive and difficult to collect in the large samples needed to detect significant effects. Furthermore, the gap between 16 months and five years is larger than previous studies, which may have limited the extent to which significant relationships could be detected. The inclusion of more time points in the analyses

would have been beneficial. A cross-lagged model with more than two time points with repeated masseurs of child emotional overeating and parental emotional feeding, would have elucidated if and when the change from a unidirectional to a bi-directional relationship occurs.

Another limitation was the difference in the measures of child EOE at the two time points. As outlined in **Chapter 3.2.1**, the original CEBQ items for EOE were modified to be age appropriate for toddlers. After extensive pilot work, including in-depth telephone interviews with participating mothers, one item was removed ('My child wants to more when he/she has nothing else to do") from the scale at 16 months. For the three remaining items, the wording of the questions was changed slightly. Overall the EOE scales used at 16 months and five years were not identical, potentially influencing the results of this longitudinal analysis. However, it is crucial to ensure that measures are age appropriate and make sense in the context of toddlerhood and despite these differences EOE was found to track significantly across the two time-points.

Steinsbekk et al (2017) also included child negative affectivity in the model, adding another dimension to represent the complexities underlying child emotional overeating. The results suggested that negative affectivity at age four, affects both parental emotional feeding and child EOE, such that higher negative affect was found to be associated with higher EOE and EUE (Steinsbekk et al, 2017). Previous research has also suggested that maternal negative affectivity influences the mother's tendency to engage in emotional feeding. A cross-sectional SEM including 323 Australian mother-child dyads highlighted that maternal experiences of stress, anxiety and depression were associated with maternal emotional eating, which was also associated with their tendency to engage in emotional and instrumental feeding (Rodgers et al., 2014). The results presented in Study 3 (**Chapter 6**) showed that maternal emotional eating is significantly associated in childhood EOE. Therefore, in the future more complex longitudinal models including other child and parental factors, such as child emotion regulation and maternal emotional overeating, should be considered when investigating the aetiology of emotional overeating. However, this requires longitudinal cohorts that have measured all of these factors repeatedly.

As mentioned previously, emotional under-eating was not measured before five years of age in Gemini. It was therefore not possible to apply this same research design to establish the directional relationships between parental pressure to eat and child EUE in Gemini. Emotional under-eating remains an under-researched area and more

longitudinal studies are needed to investigate its aetiology more fully. Due to the lack of appropriate measures in early childhood, future studies should also explore the relationship between parental feeding and child emotional under-eating in later childhood. Given that parental pressure to eat was independently associated with emotional under-eating in Study 3 (**Chapter 6**), this particular feeding behaviour needs to be explored.

## 7.6 Conclusions

In summary, the results presented in Studies 1 and 2 (**Chapters 4 and 5**) indicated the importance of shared environmental factors underlying individual differences in childhood emotional over- and under-eating. Study 3 (**Chapter 6**) attempted to characterise child, parent and environmental factors associated with both EOE and EUE, and those specific to each behaviour. Parental emotional feeding was related to both emotional over- and under-eating. Parental pressure to eat was exclusively associated with emotional under-eating. This study (Study 4) used prospective data to show that parental emotional feeding in toddlerhood influences EOE in middle childhood. The research described in the following study (Study 5, **Chapter 8**) brought genetic and environmental factors together by exploring how the aetiology of childhood emotional over and under-eating varies according to environmental stress; i.e. gene-environment interaction in emotional eating.

# Chapter 8 Study 6: Testing for Gene-environment interaction underlying EOE and EUE

## 8.1 Background

The results of the previous two studies (**Chapters 4** and **5**) highlighted that the majority of variation in emotional over- and under-eating in early childhood was explained by shared environmental effects. Genetics were found to play only a minor role. However it is possible for the aetiology of a behaviour to change in the context of specific environmental factors. This concept is called gene-environment interaction and describes a situation in which genetic and environmental influences on individual differences in a behaviour increase or decrease depending on the level of a specific (environmental) variable (described in **Chapter 3**, Section **3.5.5.7**). For example, a genetic predisposition towards lung cancer may never be expressed unless an individual smokes. Standard twin models, such as those presented in Study 1 and Study 2, would mask any underlying gene-environment interactions as the single estimates for genetic, shared and non-shared environmental effects are averaged across the whole sample and therefore across all conditions of environmental exposure (Purcell, 2002). In the example of the heritability of lung cancer, estimates would be different if a sample included both smokers *and* non-smokers, versus including *only* smoker or only non-smokers.

Gene-environment interactions have been crucial to many theoretical constructs in psychological research. The diathesis-stress model hypothesises that the effect of genes increase in the face of adverse environments (Rende & Plomin, 1992). This framework has been applied to obesity; the Behavioural Susceptibility Theory (BST) of obesity proposes that inherited differences in eating behaviours make some individuals more likely to overeat in response to the opportunities offered by the obesogenic modern food environment (Carnell & Wardle, 2008; Llewellyn & Fildes, 2017). In line with the diathesis-stress framework, genetic risk for obesity is magnified in the face environmental factors associated with increased risk of obesity including low socioeconomic status and living in a country with a more obesogenic environment. For example, a large twin study, analysing data from the Young Netherlands Twin Register of over 33000 twin individuals investigated the effect of socioeconomic status, indicated by parent education, on the heritability of BMI across development (Silventoinen, Huppertz, et al., 2016). Results found evidence for significant gene-environment interaction, whereby the heritability of BMI was much

higher in twins of lower socioeconomic status in comparison to twins with highly educated parents. These differences were especially pronounced at preadolescence. At 12-13 years heritability of BMI was 71% for female twins with parents of low to middle education. In contrast, for female twins with parents with high education the heritability was considerably smaller, explaining only 37% of the variance in BMI. Results were similar for boys (86% versus 25%) (Silventoinen, Huppertz, et al., 2016). Similar effects of socioeconomic status have been suggested in adults as well (Dinescu, Horn, Duncan, & Turkheimer, 2016; Johnson, Kyvik, Skytthe, Deary, & Sorensen, 2011), highlighting how socioeconomic status not only influences the risk of obesity, but also enhances genetic expression on BMI. Likewise, BMI heritability estimates have been found to be higher in countries with a more obesogenic environment, marked by increased food availability, higher GDP and a higher average BMI (Min, Chiu, & Wang, 2013).

These previous studies highlight how heritability estimates from twin studies are not set in stone, but can vary across different environmental contexts. Regarding BMI, research suggests that environmental factors such as low socio-economic status in a highly obesogenic environment increase the heritability. These findings are in line with the BST, proposing that genetic risk for obesity is more prominent in obesogenic environments (Llewellyn & Fildes, 2017).

Gene-environment interactions do not always result in negative outcomes such as heightened disease risk. In contrast to the diathesis-stress model, the bio-ecological framework (Bronfenbrenner & Ceci, 1994) stipulates that positive environments allow individuals to express their genetically influenced potential more freely, leading to increased heritability estimates. This idea has been tested in research investigating the aetiology of cognitive abilities. Here, a gene-environment interaction twin model showed that with increasing family income the heritability of cognitive ability in adolescence increased, whereas shared-environmental influence decreased (n = 839 twin pairs, 17 years old) (Harden, Turkheimer, & Loehlin, 2007).

Gene-environment interactions are likely to affect the development of childhood emotional over- and under-eating as well. One potential factor that could influence the genetic and environmental variation in emotional over- and under-eating is growing up in a stressful home environment. In line with the diathesis-stress model, a highly stressful home environment might result in an increase in heritability of emotional over- and under-eating. So far no previous study has tested for gene-environment interactions underlying emotional over- and under-eating in childhood.

## 8.2 Aim

The aim of this study was:

> To investigate whether the genetic and environmental influence on EOE and EUE is conditional on (moderated by) household chaos levels.

## 8.3 Methods

### 8.3.1 Sample

Data analysed in this study came from the Gemini twin cohort when the twins were five years old. A full description of the sample can be found in **Chapter 3.1**.

### 8.3.2 Measures

Age, sex and gestational age were all parent reported. Emotional over- and under-eating were measured using the Child Eating Behaviour Questionnaire (Wardle et al., 2001). Household stress was rated with a shortened version of the Confusion, Hubbub and Order Scale (CHAOS) (A. P. Matheny et al., 1995). Parents rated their experience of household stress by indicating if five statements applied to their families. Statements indicating household stress were added together creating a sum score ranging from zero (no household stress) to five (all statements applied to their family). Only families who answered all five items of the CHAOS scale were included. All measures are described in detail in **Chapter 3.2**.

### 8.3.3 Analyses

A Gene-environment interaction twin model was fitted to EOE and EUE separately, using the CHAOS score as a continuous moderator. Maximum Likelihood Structural Equation Modelling was conducted to derive estimates (and 95% confidence intervals) for the effects of the latent A, C and E factors in the absence of the moderator ($a_{Un}$, $c_{Un}$ and $e_{Un}$), as well as $\beta$-estimates indicating the change in effect for every increasing unit of the moderator. Significant moderation is indicated by a significant beta estimate ($\beta_a$, $\beta_c$, $\beta_e$) indicating changes in the variance contribution for A, C or E with increasing strength of the moderating variable. A possible gene-environmental correlation between the moderator and EOE and EUE is accounted for (regressed out) by modelling the effects of the moderator on the means (main effect, $\beta_M$). A path diagram illustrating this model and a more detailed discussion is presented in **Chapter 3.5.5.7**. Analyses were carried out in R using the OpenMx package (Boker et al., 2011).

Prior to analyses, raw scores of EOE and EUE were regressed by age at measurement, sex and gestational age. As described previously this is common in twin research as age and sex (same sex twin pairs only) is completely correlated within twin pairs, and might therefore inflate both the MZ and DZ twin pair similarity and estimates of the shared environment. In comparison to analyses presented in Study 1 and Study 2, regressed scores for EOE and EUE were not log transformed as the skew of the data can reflect underlying gene-environment interaction. However due to low variation in EOE, scores were multiplied by 2 to widen the distribution, and ease the optimisation process in the model-fitting analyses.

## 8.4 Results

### 8.4.1 Descriptive statistics

The sample in these analyses included 1814 children who had data on all included variables (zygosity, age at measurement, gestational age, sex, EOE, EUE and one CHAOS score per family). An overview of the descriptive statistics is presented in **Table 8.1**. Some level of household chaos was common, with only 180 families (19.76%) reporting no household stressors at all. **Figure 8.1** shows the distribution of scores for the CHAOS scale in this analysis sample.

The moderator variable – parental ratings of household chaos (CHAOS) – was entered as raw scores in the analyses. This way, the original scale of the moderator variable (ranging from 0 – 5) was retained. This eases interpretation of the produced models, as an increase in one unit reflects one more count of household stress

**Table 8.1 Descriptive statistics for the sample included in the analyses**

| Twin pairs | N (%) or Mean (SD) |
|---|---|
| **Total** | 907 pairs |
| | (1814 children) |
| **Zygosity** | |
| MZ pairs | 297 (33.88) |
| DZ pairs | 610 (66.12) |
| **Sex** | |
| Males | 872 (48.07) |
| Females | 942 (51.93) |
| **Gestational age (weeks)** | 36.26 (2.43) |
| **Age at measurement of EOE and EUE (years)** | 5.15 (0.13) |
| **Emotional Overeating at 5 years (raw score)** | 1.56 (0.51) |
| **Emotional Under-eating at 5 years (raw score)** | 2.68 (0.84) |
| **CHAOS at 5 years** | 1.99 (1.64) |

Abbreviations: EOE = Emotional Overeating; EUE = Emotional Under-eating, CHAOS = Confusion, Hubbub and Order Scale

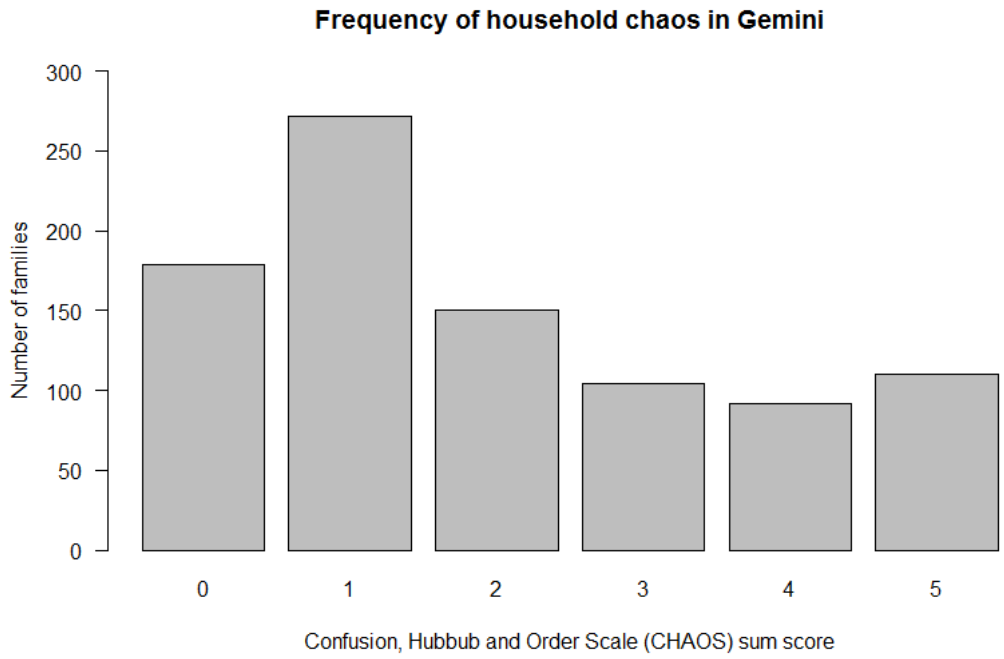


**Figure 8.1 Frequency of sum scores of the Confusion, Hubbub and Order Scale (CHAOS) Gemini**

### 8.4.2 Gene-environment interaction

#### 8.4.2.1 Household stress and child EOE

Results from twin model fitting revealed significant moderation of genetic (A) influences by household chaos. Shared environmental (C) and non-shared environmental (E) effects were not significantly moderated by household CHAOS. **Table 8.2** displays the unstandardized path estimates (95% confidence intervals) for A, C and E in the absence of any indication of household chaos ($a_{Un}$, $c_{Un}$, $e_{Un}$), as well as the unstandardized path estimates (95% Confidence Intervals) for the effect of the moderator on each latent factor ($\beta_a$, $\beta_c$ *and* $\beta_e$); these indicate the change in genetic, shared and non-shared environmental influence per one unit increase in household chaos. Confidence intervals crossing or including zero indicate no significant moderation of a path.

**Table 8.2 Unstandardized path estimates (95% confidence intervals) for unmoderated variance contributions from A, C and E and the effect of the moderator (*βa*, *βc* and *βe*) on individual differences in child EOE**

| Paths | Unstandardized estimates (95% CI) |
|---|---|
| $a_{Un}$ | 0.12 |
| | (0.01, 0.17) |
| $c_{Un}$ | 0.92 |
| | (0.85, 0.99) |
| $e_{Un}$ | 0.14 |
| | (0.13, 0.16) |
| $\beta_a$ | 0.08 |
| | (0.06, 0.10) |
| $\beta_c$ | 0.00 |
| | (-0.03, 0.04) |
| $\beta_e$ | 0.01 |
| | (0.00, 0.02) |

Abbreviations: $a_{Un}$ = Unmoderated path a; $c_{Un}$ = Unmoderated path c; $e_{Un}$ = Unmoderated path e; $\beta_a$ = effect of moderator on path a, $\beta_c$ = effect of moderator on path c, $\beta_e$ = effect of moderator on path e

As described in **Chapter 3.5.5.7** estimates for A, C and E at each level of the moderator can be calculated with the following equations

$$Va = (a_{Un} + \beta a \times \text{Level of Moderator})^2$$

$$Vc = (c_{Un} + \beta c \times \text{Level of Moderator})^2$$

$$Ve = (e_{Un} + \beta e \times \text{Level of Moderator})^2$$

These equations were used to calculate the total unstandardized variance (Vt = Va + Vc + Ve) explained at every level of CHAOS (0 – 5) using the contribution of the unmoderated latent factors A, C and E as well as those as a function of the moderator (*βa*, *βc* and *βe*) (**Table 8.4)**. Derived *β*-value for A (*β*a) was significant. Therefore, changes in Va across the moderator strength reflect a significant change. Results indicate that with increasing household stress the overall variance of EOE increases. With no household stress there is no genetic effect, with low household stress (~1) there is a small genetic effect, and with maximum household stress it is about three times higher. However, while the effects of genes increased significantly as a function of household stress, the overall impact of genes is still small.

To illustrate these findings, values in **Table 8.3** were plotted to create **Figure 8.2**.

**Table 8.3 Unstandardized variance contributions from A, C and E, as well as total variance at each level of the moderator (0 – 5) for child EOE**

| Level of CHAOS | Va | Vc | Ve | Vt |
|---|---|---|---|---|
| **0** | 0.02 | 0.84 | 0.02 | 0.88 |
| **1** | 0.04 | 0.85 | 0.02 | 0.91 |
| **2** | 0.08 | 0.86 | 0.03 | 0.97 |
| **3** | 0.14 | 0.87 | 0.03 | 1.04 |
| **4** | 0.21 | 0.89 | 0.03 | 1.13 |
| **5** | 0.29 | 0.90 | 0.04 | 1.23 |

Abbreviations: CHAOS = Confusion, Hubbub and Order Scale, Va = Unstandardised variance contribution by genetic effects, Vc = Unstandardised variance contribution by shared environmental effects, Ve = Unstandardised variance contribution by non-shared environmental effects, Vt = Total variance

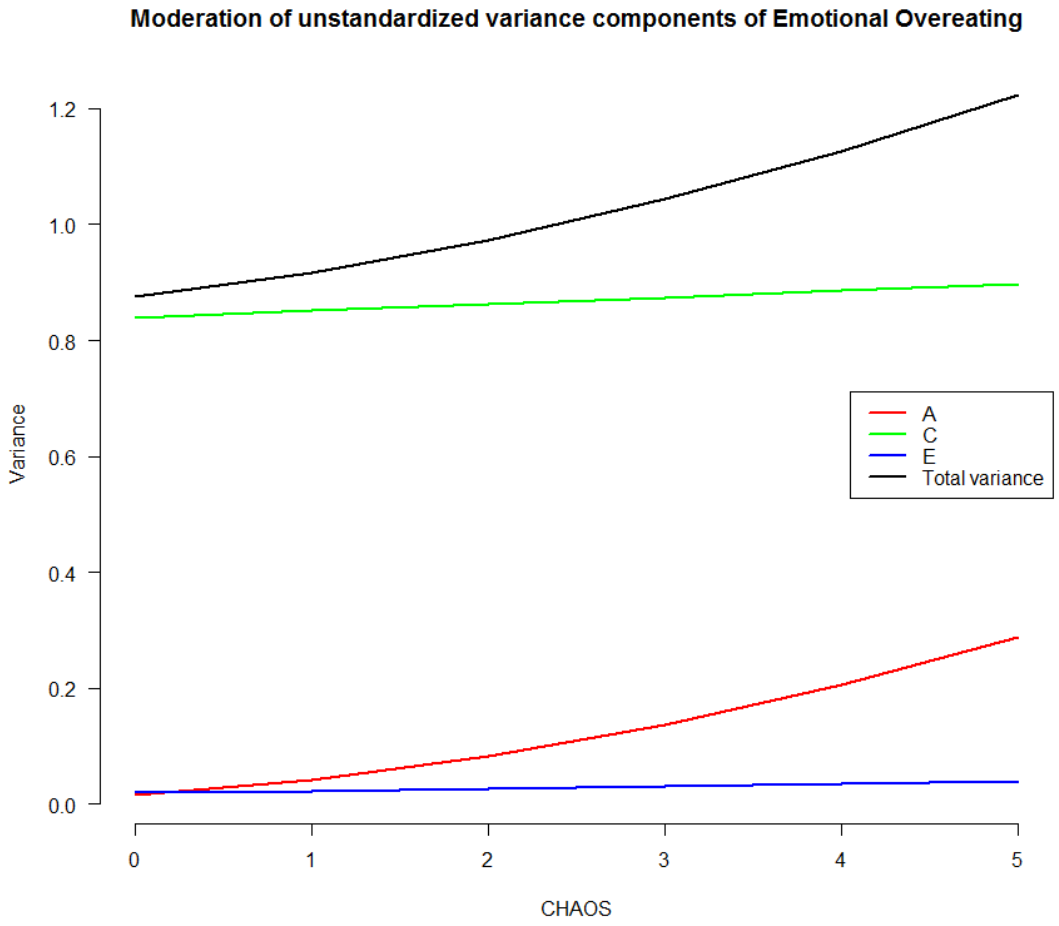


**Figure 8.2 Unstandardized Emotional Overeating variance by Household stress (CHAOS scale)**

*Unstandardized genetic and shared and non-shared environmental components in individual differences in child Emotional Overeating as a function of increasing household stress, as measured by the Confusion, Hubbub and Order Scale (CHAOS). Household stress ranges from 0 (no reported household stress) to 5 (maximum household stress)*

#### 8.4.2.2 Household stress and child EUE

The procedure described above in **Chapter 8.4.2.1** was used to examine the effect of household CHAOS on the aetiology of child EUE. Twin model fitting showed that genetic influences on EUE, like EOE, were significantly moderated by household stress, indicated by a significant estimate for $\beta_a$. In addition, the non-shared environmental influence (E) was also negatively moderated by household stress, indicated by a significant estimate for $\beta_e$. In keeping with the findings for EOE, there was no significant moderation of the shared environmental (C) influence by household stress.

**Table 8.4** presents the unmoderated paths for A, C and E ($a_{Un}$, $c_{Un}$, $e_{Un}$), as well as their change by one increasing unit of the CHAOS scale ($\beta_a$, $\beta_c$ *and* $\beta_e$).

**Table 8.4 Unstandardized path estimates (95% Confidence Intervals) for unmoderated variance contributions from A, C and E and the effect of the moderator ($\beta_a$, $\beta_c$ *and* $\beta_e$) on individual differences in child EUE**

| Paths | Unstandardized estimates (95% CI) |
|---|---|
| $a_{Un}$ | 0.27 |
| | (-0.46, 0.38) |
| $c_{Un}$ | 1.56 |
| | (1.46, 1.71) |
| $e_{Un}$ | 0.31 |
| | (0.26, 0.39) |
| $\beta_a$ | 0.07 |
| | (0.04, 0.28) |
| $\beta_c$ | 0.00 |
| | (-0.05, 0.06) |
| $\beta_e$ | -0.03 |
| | (-0.05, -0.02) |

Abbreviations: $a_{Un}$ = Unmoderated path a; $c_{Un}$ = Unmoderated path c; $e_{Un}$ = Unmoderated path $\beta_a$ = effect of moderator on path a, $\beta_c$ = effect of moderator on path c, $\beta_e$ = = effect of moderator on path e

Again, unstandardized variance components of A, C and E (Va, Vc, Ve) and total variance (Vt) at each level of the moderator (CHAOS, range: 0 – 5) were calculated (see **Table 8.5**). As genetic and non-shared environmental paths were found to be significantly moderated in this model, the changes in Va and Vc with increasing levels of household stress are significant. Genetic effects on EUE were non-significant in the absence of any household stress, but increase significantly as a function of increasing household stress. In turn, the variation contributions of the non-shared environmental factors decreased. Again, while significant, the changes in contributions from genetic and non-shared environmental factors were small. **Figure 8.3** illustrates these changes, by plotting the unstandardized variance contribution to the total variance at each level of household chaos.

**Table 8.5 Unstandardized variance contributions from A, C and E, as well as total variance at each level of the moderator (0 – 5) for child EUE**

| Level of CHAOS | Va | Vc | Ve | Vt |
|---|---|---|---|---|
| **0** | 0.07 | 2.51 | 0.10 | 2.68 |
| **1** | 0.12 | 2.53 | 0.08 | 2.73 |
| **2** | 0.18 | 2.55 | 0.06 | 2.79 |
| **3** | 0.24 | 2.57 | 0.05 | 2.86 |
| **4** | 0.32 | 2.60 | 0.03 | 2.95 |
| **5** | 0.41 | 2.62 | 0.02 | 3.05 |

Abbreviations: CHAOS = Confusion, Hubbub and Order Scale, Va = Unstandardized variance contribution by genetic effects, Vc = Unstandardized variance contribution by shared environmental effects, Ve = Unstandardized variance contribution by non-shared environmental effects, Vt = Total variance

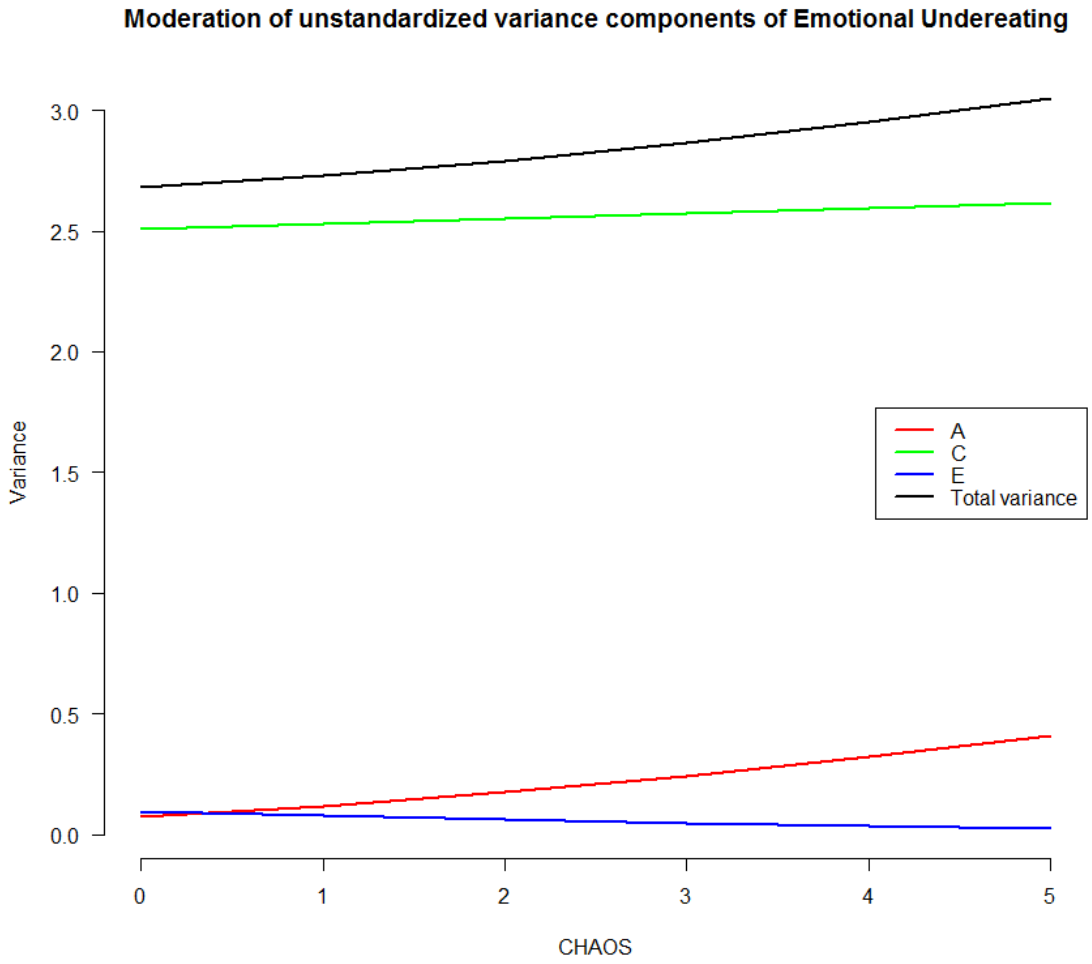


**Figure 8.3 Unstandardized variance in Emotional Under-eating by Household stress** (CHAOS scale)

*Unstandardized genetic and shared and non-shared environmental components in individual difference in child Emotional Under-eating as a function of increasing household stress, as measured by the Confusion, Hubbub and Order Scale (CHAOS). Household stress ranges from 0; no reported household stress, to 5; maximum household stress*

## 8.5 Discussion

### 8.5.1 Summary

The aim of this study was:

> To investigate whether the genetic and environmental influence on EOE and EUE is conditional on (moderated by) household stress levels.

A continuous moderator gene-environment interaction model was fitted to test if genetic, shared and non-shared environmental influence on individual differences in EOE and EUE changed as a function of household stress, when the children were five years old. Regarding EOE, results showed that genetic influence increased significantly with increasing household stress. There was no moderation of the non-shared and shared-environmental components. However, as already discussed in Study 1 and Study 2 (**Chapter 4** and **5**), shared environmental effects explained the majority of variation in child EOE and remained the most important factor underlying this behaviour at all levels of household stress. Even at the highest level of household stress, the shared environment was the largest contributor to individual differences in EOE. With increasing household stress, genetic effects became significantly more important, albeit not to a large extent. This is indicative of gene-environment interaction in which more stress and chaos in the home provides an environment that allows genetic influence on emotional overeating to be fully expressed. Overall variation in child EOE increased in the face of high household chaos, indicating that stressful environments illicit greater individual differences in emotional overeating in children and this effect was mainly due to increases in genetic expression.

The analyses for EUE at five years produced very similar findings to those for EOE. Twin model fitting results suggested significant moderation of the genetic and non-shared environmental contributions underlying individual differences in child EUE. The non-standardised variance contribution from genetic influence rose from 0.07 at zero household stress to 0.41 for maximum household stress. Similarly to EOE, shared environmental effects explained the majority of the variance in the absence of, and at all increasing levels of household stress. Furthermore, the moderation of non-shared environmental effects was significant and negative, but very small; resulting in a minor negative change (0.10 to 0.02) to the variance explained such that there was a slightly smaller effect of the non-shared environment as household stress increased. Overall individual differences in EUE increased as a function of

cumulative household stress and this effect was mainly due to increases in genetic expression.

**Figure 8.5** (adapted from (Llewellyn, Carnell, & Wardle, 2010) gives an illustration of the theoretical underpinnings of emotional eating behaviour in childhood. Emotional overeating and under-eating increase with increased household stress. Individuals at high genetic risk for this behaviour have a greater increase than children with average or low genetic risk.


**Figure 8.4 Theoretical illustration of the interaction between genetic susceptibility to emotional eating and household stress, adapted from Llewellyn et al 2010.**

### 8.5.2 Implications

The results in this chapter highlight the complex and dynamic aetiology of child EOE and EUE. Environmental factors shared by two twins within one pair explained the majority of individual differences in both behaviours. However, with accumulating household stress, genetic influence increased significantly, although not to a large extent.

This indication of gene-environment interaction highlights how negative and stressful environments could elicit stronger genetic contributions to individual differences in childhood emotional over- and under-eating. These findings are in line with the diathesis-stress model, in which negative environmental exposures increase the biological risk for maladaptive behaviour and disease (Rende & Plomin, 1992). In order for emotional eating to be expressed to its fullest potential, a stressful environment is necessary.

Regardless of the increased genetic component, in more chaotic homes shared environmental factors remained the strongest contributor to the variance in emotional eating. As shown in Study 3 and 4 (**Chapter 6** and **7**), parental emotional feeding was found to be cross-sectionally and longitudinally associated with child EOE, whereas parental pressure to eat was specifically linked to child EUE. These parental behaviours might be the main predictors of childhood emotional over under-eating in both low and highly chaotic households.

Previous research has investigated the associations between stress in the home and parenting. A cross-sectional investigation of 106 families with five- to six-year-old children studied the link between household stress (measured with the Confusion, Hubbub and Order Scale) and overall parenting. High household stress was positively associated with increased parenting stress as well as increased child impulsivity, indicating that household stress has direct effects on child and parent behaviour (Dumas et al., 2005). Similarly, a study of 118 families with two children between four and eight years of age, revealed that a chaotic home environment was associated with increased child problem behaviours as well as child reported maternal and paternal hostility and an overall more negative child-parent relationship (Coldwell, Pike, & Dunn, 2006). More recently a longitudinal study followed 100 families over the first 12 months of their infant's life. Along with chaos in the home, negative life events, maternal emotional availability and negative co-parenting were assessed at one month postpartum and then repeatedly every three months (Whitesell, Teti, Crosby, & Kim, 2015). Results suggested that a chaotic home environment has long-term

consequences. High levels of chaos in the home were associated with a higher number of negative life events later on. Parents in highly chaotic homes reported the co-parenting behaviours of their partners more negatively. Additionally, maternal emotional availability, the mothers ability to appropriately respond to her child's emotions, was significantly lower and decreased over time in families rated as highly chaotic (Whitesell et al., 2015). These previous studies suggest that a stressful home environment is predictive of maladaptive parenting. Future research is needed to test how chaos in the family home is associated with parental feeding practices, such as emotional feeding and parental pressure to eat specifically to uncover the relationship between chaotic households and maladaptive feeding strategies affecting child emotional over- and under-eating.

Overall, the findings presented here imply that the aetiology of child EOE and EUE fits into the diathesis-stress framework, in which eating exposure to negative, more stressful environments increases genetic influence. However, even in the absence of any chaos in the home, shared environmental effects explained the majority of variation in child EOE and EUE. This pervasiveness of shared environmental factors is in line with findings discussed in **Chapter 6** and **7**, positing parental emotional feeding and parental pressure to eat as key parental behaviours underlying these traits and thus a major focus of treatment and intervention.

### 8.5.3 Strengths and limitations

#### 8.5.3.1 Measurement of household chaos

Just like in the previous chapters, all variables in these analyses were parent-rated. Parents rated their twins' propensity to emotionally over- or under-eat, as well as their perception of the chaos in their family. Included measures, the Child Eating Behaviour Questionnaire (Wardle et al., 2001b) and the Confusion Hubbub and Order Scale (CHAOS) (A. P. Matheny et al., 1995), are validated and commonly used in research. However, a reduced version of the CHAOS scale consisting of five items, instead of the original tool with 15 items, was used. Including more items would have enabled a more fine grained measurement of the household stress. However the validity of this shortened version has been previously demonstrated (Pike, Iervolino, Eley, Price, & Plomin, 2006).

Furthermore, the household stress rated by the parents might differ from the child's experience, potentially affecting associations with childhood emotional over- and under-eating. A previous study of 348 families with two children between 11 and 17 years of age, investigated the differences between child- and parent-rated household

stress (D. Daniels, Dunn, Furstenberg, & Plomin, 1985). Results suggested that not only do parents and children have a different understanding of household stress, even siblings within the same family experience different levels of chaos in the home. If home chaos is not a family-level but child-specific variable, individual differences in child experiences of household stress can be investigated using a twin design. Household stress was child reported with the CHAOS scale in a sample of 2337 twin pairs aged 9-12 years old (Hanscombe, Haworth, Davis, Jaffee, & Plomin, 2010). Results indicated that twin pairs within the same family experience their household differently. Individual differences in household chaos experiences were mostly explained by shared and non-shared environmental factors. However, genetic effects were substantial with a heritability estimate of 22%. Findings indicate that genetically determined traits, such as personality for example, influence the perception of household chaos.

In the analyses presented here, parental ratings of home stress meant that there was only one score per family, precluding the decomposition of variance into genetic and environmental factors. However this does not mean that household chaos can be conceptualised as a *true* environmental factor not actively created by - or as a response to - the child's behaviour. It is very much possible that this variable is also influenced by many genetically determined traits in the parents, as well as other child factors such as personality (Gene-environment correlation). However, I have accounted for any potential Gene-environment correlation by regressing out the effect of the CHAOS from the dependent variables.

Future studies might benefit from including child reports of household stress to extend these analyses, enabling a more thorough exploration of the association of stress in the home and child emotional over- and under-eating. Although child-rated measures of household stress, such as the children's daily hassles and daily uplifts scales (Kanner, Coyne, Schaefer, & Lazarus, 1981) or the child-completed CHAOS scale are available, they would not have been appropriate for the current study as the children were quite young. Previous research analysing child-rated household chaos focussed on older children and these measures could be included in future data collections within the Gemini cohort.

Even though the results suggested significant moderation of the genetic contribution to individual differences in child EOE and EUE, the effect sizes were fairly modest. One reason could be fairly high socio-economic status of the families included in the Gemini cohort. As outlined in **Chapter 3.1**, the majority of the sample falls into the

highest tertile of socio-economic status, indicated by family income. Wealthy families might be likely to experience lower household chaos overall, which might have dampened the effect detected in this chapter. However, scores on the CHAOS scale in this sample suggested that only around 20% of the families did not experience any household stress at all, with the remaining 80% reporting at least one indicator of household stress. Future research would benefit from repeating these analyses in larger and more socio-economically diverse samples using the original and larger CHAOS scale. This way, more variation in household chaos might be measured including more extreme cases, potentially detecting greater moderating effects.

#### 8.5.3.2 Limitations of Gene-environment interaction models

Apart from the limitations regarding the measures of household stress, the continuous moderator twin model comes with some limitations. As discussed in the method section in **Chapter 8.4.2.1** and more generally in **Chapter 3.5.5.7**, emotional over and under-eating data were analysed without any prior transformations. That meant that, especially for child EOE, the variance distribution was positively skewed. Analysing skewed data was necessary in this instance, as continuous moderator twin models cannot differentiate if the skew in the data is a consequence of the moderating variable or an artefact of poor reliability of the measure introducing error. Therefore analysing transformed dependent variables might artificially result in non-significant findings, by supressing the detection of significant gene-environment interaction. For this reason, researchers have been advised to analyse non-transformed variables when fitting continuous moderator twin models (Purcell, 2002).

However, the skew in the data could in fact be due to effects other than interaction effects, e.g. poor item distribution across the scale. To account for this, Murray et al (2016) proposed that Item Response Theory (IRT) models should be applied (Murray et al., 2016). In comparison to Classical Test Theory, which assumes that every item on a scale is of equal difficulty and can henceforth be combined freely, ITR treats every item individually. In doing so, IRT assumes an unmeasured latent factor, on which the individual questionnaire items load. It has been suggested that incorporating this approach into gene-environment interaction modelling, is advantageous, as (error-free) latent variables are deemed to be normally distributed, with a standard deviation close to one, diminishing skew in the data. Murray et al (2016) compared gene-environment interaction models using Item Response Theory with more traditional transformed and raw sum scores. Results suggested that variables derived from Item Response Theory performed better, producing less

biased estimates, especially when the raw scores were highly skewed (Murray et al., 2016).

The analyses conducted in this chapter used the traditional gene-environment interaction approach proposed by Purcell in 2002 (Purcell, 2002). Future analyses would benefit from adopting this new approach (Murray et al., 2016), and comparing findings. However, it is important to note that IRT is computationally demanding, requiring larger sample sizes and therefore might not always be feasible. As an example, the sample included in the paper by Murray et al (2016) investigating the utility of IRT in gene-environment interaction twin models included over 4000 twin pairs.

## 8.6 Conclusions

Analyses in this chapter aimed to test if household stress moderates the genetic and environmental influence on child emotional over- and under-eating. Results revealed that with increasing household stress, genetic influence on emotional over- and under-eating increased significantly. In addition, small but significant moderation was found for the effects of non-shared environmental factors on emotional under-eating.

These findings place emotional over- and under-eating within a diathesis-stress framework in which exposure to negative environments increases the heritability. Overall, the results highlighted the complex and dynamic effects of genes and environments underlying the development of child emotional over- and under-eating. However, as highlighted in the previous chapters, shared environmental factors were the strongest influences underlying both child emotional over- and under-eating.

The previous five studies described in **Chapters 4 – 8** addressed the first four aims outlined in **Chapter 2**, investigating the aetiology of child emotional over- and under-eating. The following two studies, Study 6 (**Chapter 9**) and Study 7 (**Chapter 10**), were conducted to replicate findings of Study 2 (**Chapter 5**) in an independent sample and to examine twin-specific bias in parental reporting their twin children's eating behaviour measured using the CEBQ and BEBQ (an infant versions).

# Chapter 9 Study 6: Replication of the aetiology of emotional over- and under-eating in an independent sample of four-year-old twins

## 9.1 Background

This study chapter is a replication of the analyses presented in Study 2 (**Chapter 5**), which established the heritability of EOE and EUE in middle childhood (age 5 years) in Gemini. In addition, the analyses examined genetic and environmental contributions to the positive association between EOE and EUE. Results showed low heritability for EOE and EUE (6 and 7%), with the majority of individual differences in these behaviours being explained by shared environmental factors (92% and 91% respectively). The observed association between EOE and EUE was substantial (r = 0.43). About one quarter of the shared environmental effects ($r_C$ = 0.49; $0.49^2$ = 0.24, 24%) underlying EOE and EUE were the same, and it was these common shared environmental effects that entirely explained the positive correlation between these two behaviours.

Replication of results in independent samples is essential to verify previous findings. Results presented in Study 2 (**Chapter 5**) reported very high intraclass correlations for MZ and DZ twins, which might be seen as an indicator of poor reliability of the measure, with mothers giving very similar scores for both twins within one pair. Therefore, it is crucial to confirm if this pattern of aetiology can be replicated in an independent sample. The twins included in the following analyses were selected because they were identified as being at high or low risk of obesity, based on the weight category of their parents (families with two overweight/obese parents or two lean parents were selected); families also had a wider variation in socioeconomic status than Gemini. This selection process increased the variation in parent and child weight within the sample and therefore potentially also increases variation in parental feeding practices and child eating behaviour. Furthermore, twin research addressing the aetiology of EOE and EUE in childhood is very rare, with the studies described in this thesis in Study 1 and 2 (**Chapter 4** and **5**) being the only two twin studies investigating these phenotypes in childhood to date.

## 9.2 Aims

The aims of this study were

1. To estimate the genetic and environmental influence on EOE and EUE at four years of age in an independent sample
2. To investigate the extent to which EOE and EUE share their genetic and environmental aetiology in an independent sample

## 9.3 Methods

### 9.3.1 Sample

Data analysed in this study were a subsample of twin children from the Twins Early Development Study (TEDS). TEDS is one of the largest twin birth cohorts in the world including over 15000 twin pairs born in the UK in 1994 - 1996 (Trouton, Spinath, & Plomin, 2002). When the twins were four years old, 214 families were recruited for an in-depth study of the eating and feeding behaviours of 'obese' and 'lean' families with young children (Wardle et al., 2002). The subsample was deliberately oversampled for parents with obesity, selecting 100 overweight or obese sets of parents. These overweight or obese families were selected by identifying families in which the mother's reported BMI was at least 28.5 and the father's reported BMI was at least 25. Normal weight families (n = 114) in which both parents' BMI was less than 25 were selected to come from the same areas of the country, and to provide an approximate match in terms of social class, for which we used paternal occupation as an indicator. Families were visited at home by research assistants and undertook a series of tests and completed various questionnaires. All DZ twin pairs were same sex.

### 9.3.2 Measures

EOE and EUE were measured using the Child Eating Behaviour Questionnaire (Wardle et al., 2001). The CEBQ includes subscales for EOE and EUE each containing four items. A detailed description of this measure can be found in **Chapter 3.2.1**. Similar to Gemini, TEDS includes in-depth descriptive measures of the families. In order to compare this replication sample with the Gemini sample, the following measures were investigated: maternal age at twin birth, ethnicity, maternal education (percentage of mother with a university degree), and socio-economic status (highest professional occupation per family, clustered in high, intermediate and low). Importantly, the BMI of the twins in the two samples was also compared. BMI-Standard Deviation Scores (BMI-SDS) were calculated using British 1990 growth

reference data. SDS scores were used rather than raw scores, because children's weights and BMIs vary considerably with development before 18 years of age. SDS provide an indication of how the children's weights and BMI compare to other children of the same age and sex (Cole, 1990; Freeman et al., 1995).

### 9.3.3 Analyses

At first intraclass correlations (ICCs) were calculated for MZ and DZ twins. Comparing twin pair similarity across MZ and DZ twins provides an indication of the aetiology of EOE and EUE. More details on the use of ICCs in twin research can be found in **Chapter 3.5.1**.

Two univariate twin models were then run using Maximum Likelihood Structural Equation Modelling (MLSEM) in order to decompose the variance of EOE and EUE into three latent factors additive genetic effects (A), shared (C) and non-shared (E) environmental effects.

A bivariate Correlated Factors Model was applied to estimate aetiological correlations (denoted: $r_A$, $r_C$, $r_E$) between EOE and EUE which indicate how many genetic ($r_A$), shared environmental ($r_C$) and unique environmental ($r_E$) influences underlying EOE and EUE are the same. In addition, this model decomposes the phenotypic correlation between EOE and EUE into the latent factors A, C and E. These bivariate estimates (denoted: BivA, BivC and BivE) indicate the extent to which common genetic, shared environmental and unique environmental factors explain the phenotypic correlation between the two behaviours. A more detailed description of the twin method and its underlying principles can be found in **Chapter 3.5**. All analyses were carried out using OpenMx software (Boker et al., 2011) a statistical package run in R. Results from this chapter will be presented side by side with results from Study 2. This way the results from this study can be directly compared with the previous findings.

### 9.3.4 Data preparation

Prior to analyses, scores on EOE and EUE were regressed by age at measurement, gestational age and sex. This is common practice in twin research, as age (and sex in same sex pairs) is always the same within twin pairs, and could therefore inflate the twin pair similarity and increased the shared environmental effect.

## 9.4 Results

### 9.4.1 Descriptive statistics

A comparison of the descriptive statistics for the available data in this chapter and Study 2 (**Chapter 5**) is presented in **Table 9.1**. In the TEDS subsample, data for EOE and EUE was available for 394 twins (197 pairs) when the children were four years old**.** Mean scores for EOE and EUE were similar, but slightly higher in TEDS (EOE: mean = 1.84; EUE: mean = 2.84) versus Gemini (EOE: mean= 1.56 EUE: mean = 2.66). EOE and EUE were significantly and substantially positively correlated (r = 0.53, 95%CI: 0.44, 0.61), in keeping with the observation in Gemini (r = 0.43, 95%CI: 0.37, 0.47).

There were some crucial differences between the Gemini sample and the TEDS sample analysed here. TEDS twin were on average heavier then Gemini twins (TEDS: BMI-SDS = 0.45, Gemini: BMI-SDS = -0.22). Furthermore, mothers of TEDS twins were older when their twins were born (34.8 versus 33.8). The majority of both samples consisted of white families. However, a smaller proportion of the TEDS families fell into the highest social class in comparison to Gemini (38.6% versus 70.9%).

### 9.4.2 Distribution of EOE and EUE sample

EOE and EUE were more common in this sample than in Gemini. From the 394 children, only 12.2% (48 children) were rated to never engage in EOE. EUE was found to be even more common, as only 1.3% (5 children) were rated to never engage in EUE. Prior to analyses the raw scores of EOE and EUE were regressed by age, sex and gestational age of the twins. After inspection of histograms (see **Figure 9.1**), it was decided that EOE and EUE were close to a normal distribution in this sample so transformations were not necessary. However, to avoid the introduction of negative values into the MLSEM analyses, the smallest value of regressed EOE and EUE was added to their respective distributions, shifting all values across zero without changing their distributions. Raw, regressed and modified EOE and EUE scores are displayed in **Figure 9.1.**

**Table 9.1 Descriptive statistics for the sample included in the analyses in comparison to Chapter 5**

| Sample | Gemini | TEDS |
|---|---|---|
| **Twin pairs** | **N (%) or Mean (SD)** | **N (%) or Mean (SD)** |
| **Total** | 1027 pairs | 197 pairs |
| **Zygosity** | | |
| MZ pairs | 346 (33.7) | 89 (45.2) |
| DZ pairs | 681 (66.3) | 108 (54.8) |
| **Sex** | | |
| Males | 1000 (48.7) | 177 (44.9) |
| Females | 1054 (51.3) | 217 (55.1) |
| **Gestational age (weeks)** | 36.26 (2.43) | 36.63 (2.61) |
| **Age at measurement of EOE and EUE (years)** | 5.15 (0.13) | 4.41 (0.35) |
| **Emotional Overeating (EOE)** | 1.56 (0.51) | 1.84 (0.53) |
| **Emotional Under-eating (EUE)** | 2.66 (0.84) | 2.84 (0.82) |
| **Child BMI-SDS** | -0.22 (1.14) | 0.45 (1.19) |
| **Maternal age at twin birth** | 33.8 (4.7) | 34.8 (4.36) |
| **Highest maternal education** | | |
| University degree | 544 (49.5%) | 50 (12.7%) |
| **Ethnicity** | | |
| white | 981 (96.6%) | 368 (93.4) |
| non-white | 46 (3.4%) | 26 (6.6) |
| **Socio-economic status** | | |
| High | 727 (70.9%) | 76 (38.6%) |
| Intermediate | 151 (14.7%) | 78 (39.6%) |
| Low | 145 (14.1%) | 27 (13.7%) |
| Missing | 4 (0.3%) | 16 (8.1%) |

[1] Families were grouped by the occupation of the highest earner per family: high (higher and lower managerial and professional occupations), intermediate (intermediate occupations, small employers and own account workers – self-employed with no employees) and lower occupational classifications (lower supervisory and technical occupations, (semi-) routine occupations, never worked and long-term unemployed).

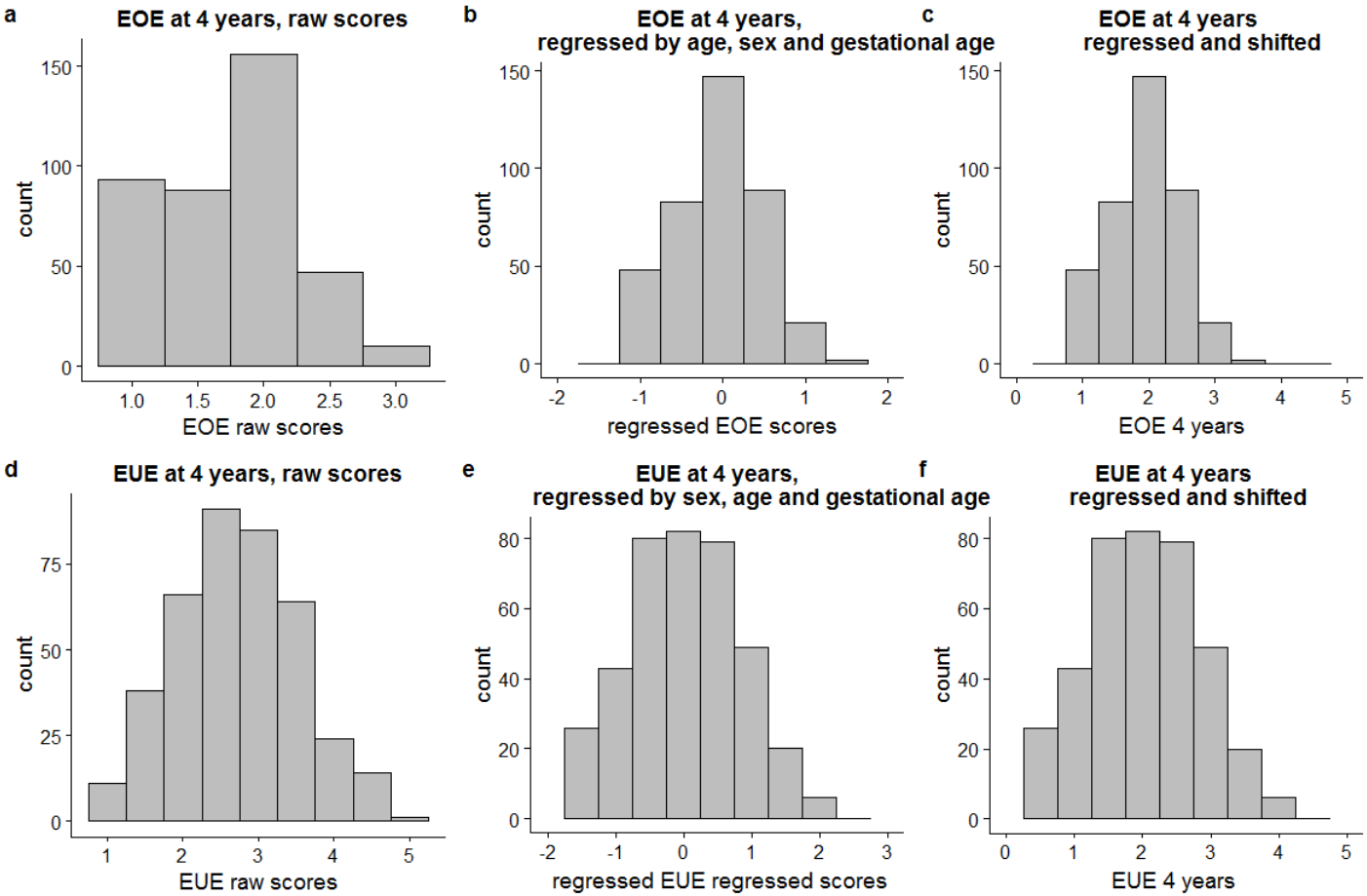


**Figure 9.1 Figures a-c show raw (a), regressed (b), and regressed and shifted (c) scores for EOE; Figures d-f show raw (d), regressed (e), and regressed and shifted (c) scores for EUE**

### 9.4.3 Intraclass correlations

Intraclass correlations (ICCs) were calculated for MZ pairs and DZ pairs separately, with higher ICCs indicating greater twin pair similarity. The pattern of twin resemblance provides an indication of the genetic and environmental contributions to variation in a trait. Generally speaking, greater similarity between MZ than DZ pairs, indicates the presence of genetic influence. Very similar ICCs across zygosity indicate important shared environmental factors. As this is a replication study, the ICCs from the TEDS data are presented alongside the ICCs from the Gemini data calculated in Study 2. The ICCs were similar and high for both MZ and DZ pairs suggesting strong shared environmental influence on individual differences in EOE and EUE. Furthermore, the cross-twin cross-trait (CT-CT) correlations indicated that the phenotypic correlation between EOE and EUE was of a similar magnitude for MZ and DZ pairs. Overall this pattern of resemblance replicated the estimates from Study 2, however, the ICCs in TEDS were generally lower for MZ and DZ pairs, suggesting a more important role of non-shared environmental effects in the TEDS subsample.

**Table 9.2 Intraclass correlations (95% Confidence intervals) for EOE and EUE as well as cross-twin cross-trait correlations for MZ and DZ twin pairs**

| **Sample** | **Gemini** | | **TEDS** | |
|---|---|---|---|---|
| | **MZ** | **DZ** | **MZ** | **DZ** |
| **EOE** | 0.98 | 0.94 | 0.74 | 0.72 |
| **ICCs (95% CI)** | (0.97-0.98) | (0.92-0.94) | (0.64, 0.81) | (0.61, 0.79) |
| **EUE** | 0.98 | 0.95 | 0.81 | 0.80 |
| **ICCs (95% CI)** | (0.97-0.98) | (0.94-0.95) | (0.74, 0.86) | (0.72, 0.85) |
| **CT-CT (95% CI)** | 0.43 | 0.44 | 0.49 | 0.49 |
| | (0.42-0.43) | (0.39-0.49) | (0.38, 0.58) | (0.39, 0.58) |

MZ: Monozygotic; DZ: Dizygotic; EOE: Emotional Overeating; EUE: Emotional Under-eating; ICCs: Intraclass correlations; CI: Confidence intervals; CT-CT: Cross-twin cross-trait correlations

### 9.4.4 Maximum likelihood structural equation modelling

***Establishing the genetic and environmental contributions to EOE and EUE***

Two separate univariate analyses were conducted to obtain A, C and E parameters for EOE and EUE at four years in TEDS. After fitting a saturated model and a full ACE model, three submodels were tested, each dropping one latent factor (AE model, CE model and E model). The LRT was used to distinguish between the different models. Furthermore, the BIC was taken into consideration for model fit. For both EOE and EUE the full ACE model fitted the data well, with no significant loss of model fit, compared to the saturated model (EOE: $\Delta \chi^2 = 1.244$, $p = 0.97$; EUE: $\Delta \chi^2 = 1.117$, $p = 0.98$). Additionally, for EOE and EUE, the LRT and BIC suggested a good model fit for CE decomposition, dropping the genetic component entirely (EOE: $\Delta \chi^2 = 0.041$, $p = 0.84$; EUE: $\Delta \chi^2 = 0.223$, $p = 0.63$). However, due the small number of participants included in this sample, the confidence intervals around the parameter estimates were wide, allowing for latent factors to be dropped more liberally precluding a straight forward interpretation. The full ACE models were therefore considered for interpretation here.

In keeping with the findings from Gemini, the heritability estimates for EOE (2%, 95% CI: 0, 24) and EUE (4%, 95% CI: 0, 20%) were small, and the majority of variance was explained by shared environmental factors (EOE: 72%, 95% CI: 52, 79%; EUE: 77%, 95% CI: 63, 85%). Non-shared environmental influences were considerable for both EOE (25%, 95% CI: 19, 34%) and EUE (19%, 95% CI: 13, 25%). Overall these results corroborate the results reported in Study 2 (**Chapter 5**), however estimates of the non-shared environment were notably larger. Relevant estimates and fit-statistics are listed in **Table 10.3a** and **10.3b**.

**Table 9.3a Parameter estimates (95% CI) and fit statistics for EOE at four years**

| EOE four years (n = 390) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Model[1]** | **A** | **C** | **E** | **-2LL** | **df** | **Δ $\chi^2$ (df)** | **p-value** | **BIC** |
| **Sat** | | | | -46.162 | 380 | | | |
| **ACE** | 0.02 (0.00, 0.24) | 0.72 (0.52, 0.79) | 0.26 (0.19, 0.34) | -44.918 | 386 | 1.244 (6) | 0.97 | -2086.189 |
| **AE** | 0.76 (0.68, 0.82) | | 0.24 (0.18, 0.32) | -14.433 | 387 | 30.485 (1) | <0.001 | -2060.992 |
| **CE** | | 0.73 (0.66, 0.79) | 0.27 (0.21, 0.34) | -44.877 | 387 | 0.041 (1) | 0.84 | -2091.436 |
| **E** | | | 1 | 102.509 | 388 | 147.438 (2) | < 0.001 | -1949.338 |

Abbreviations 2LL: -2 log-likelihood of data; df: degrees of freedom; Δ $\chi^2$: change in chi-square; BIC: Bayesian Information Criterion

Models: AE, CE and E models are nested within the full ACE model. The ACE model dissects the phenotypic variance into A, C and E; the AE model drops the C parameter and assesses the variance explained by the A and E parameters only; the CE model drops the A parameter and assesses the variance explained by the C and E parameters only; the E model drops both the A and C parameters and assesses the variance explained by E only. Best-fitting models are bold

**Table 9.3b Parameter estimates (95% CI) and fit statistics for EUE at four years.**

| EUE at four years (n = 390) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Model[1]** | **A** | **C** | **E** | **-2LL** | **df** | **Δ $\chi^2$ (df)** | **p-value** | **BIC** |
| **Sat** | | | | 749.015 | 380 | | | -1260.526 |
| **ACE** | 0.04 (0.00, 0.20) | 0.77 (0.63, 0.85) | 0.19 (0.13, 0.25) | 750.133 | 386 | 1.117 (6) | 0.98 | -1291.1381 |
| **AE** | 0.82 (0.76, 0.86) | | 0.18 (0.14, 0.24) | 794.056 | 387 | 43.923 (1) | < 0.001 | -1252.5035 |
| **CE** | | 0.80 (0.75, 0.85) | 0.20 (0.15, 0.25) | 750.361 | 387 | 0.223 (1) | 0.63 | -1296.198 |
| **E** | | | 1 | 951.653 | 388 | 201.520 (2) | < 0.001 | -1100.193 |

Abbreviations 2LL: -2 log-likelihood of data; df: degrees of freedom; Δ $\chi^2$: change in chi-square; BIC: Bayesian Information Criterion

Models: AE, CE and E models are nested within the full ACE model. The ACE model dissects the phenotypic variance into A, C and E; the AE model drops the C parameter and assesses the variance explained by the A and E parameters only; the CE model drops the A parameter and assesses the variance explained by the C and E parameters only; the E model drops both the A and C parameters and assesses the variance explained by E only. Best-fitting models are bold

***Decomposing the correlation between EOE and EUE into genetic and environmental factors***

A bivariate Correlated Factors Model was fit to the data to investigate the phenotypic covariation between EOE and EUE (r = 0.53, 95%CI: 0.44, 0.61). In addition to estimating the effects of the latent factors A, C and E, the bivariate Correlated Factors Model estimates the extent to which the genetic and environmental factors are shared between the two phenotypes ($r_A$, $r_C$, and $r_E$). Furthermore, bivariate estimates (BivA, BivC and BivE) decompose the phenotypic correlation between EOE and EUE into proportions of covariation explained by common A, C and E.

The proposed full ACE model fitted the data well compared to the saturated model ($\Delta \chi^2$ =6.134, p=0.99). As suggested from the univariate analyses, genetic effects were non-significant for both EOE (A=0.03, 95% CI: 0.00, 0.25) and EUE (A= 0.04, 95% CI: 0.00, 0.21), but confidence intervals were wide. Environmental factors shared by the twins were dominant in driving individual differences in both EOE (C = 0.71, 95% CI: 0.52, 0.79) and EUE (C = 0.77, 95% CI: 0.62, 0.85), and the contribution from non-shared environmental factors was substantial (EOE: E = 0.26, 95% CI: 0.19, 0.34; EUE: E = 0.19, 95% CI: 0.14, 0.25).

The aetiological correlations indicated that only shared environmental factors ($r_C$ = 0.77, 95% CI: 0.51, 0.85) were common to both behaviours, because the non-shared and genetic aetiological correlations were both non-significant, although moderate in size ($r_E$ = 0.21, 95% CI: -0.00, 0.38; $r_A$ = 0.26, 95% CI: -1.00, 0.99). In line with this observation, when decomposing the phenotypic correlation (r = 0.53) into A, C and E, bivariate estimates indicated that the majority of the association was being driven by shared environmental factors underlying both behaviours (BivC =0.50, 95% CI: 0.35, 0.60). Bivariate estimates for A and E were non-significant (BivA = -0.01, 95% CI: -0.10, 0.12; BivE: 0.05, 95% CI:-0.00, 0.09). **Table 9.4** shows the fit statistics for the saturated and the full bivariate correlated factors ACE model. **Figure 9.1** shows a path diagram illustrating the results.

**Table 9.4 Model fit statistics for the saturated model and the full bivariate Correlated Factors Model**

| Model | parameters | -2LL[1] | df[1] | Δ $\chi^2$ (df) | p-value | BIC |
|---|---|---|---|---|---|---|
| **Sat** | 28 | 612.828 | 752 | | | -3363.948 |
| **ACE** | 11 | 618.962 | 769 | 6.134 (17) | 0.99 | -3447.715 |

Abbreviations: 2LL: -2 log-likelihood of data; df: degrees of freedom; Δ $\chi^2$: change in chi-square; BIC: Bayesian Information Criterion; Sat: Saturated model; ACE: Full bivariate Correlation Factors Model

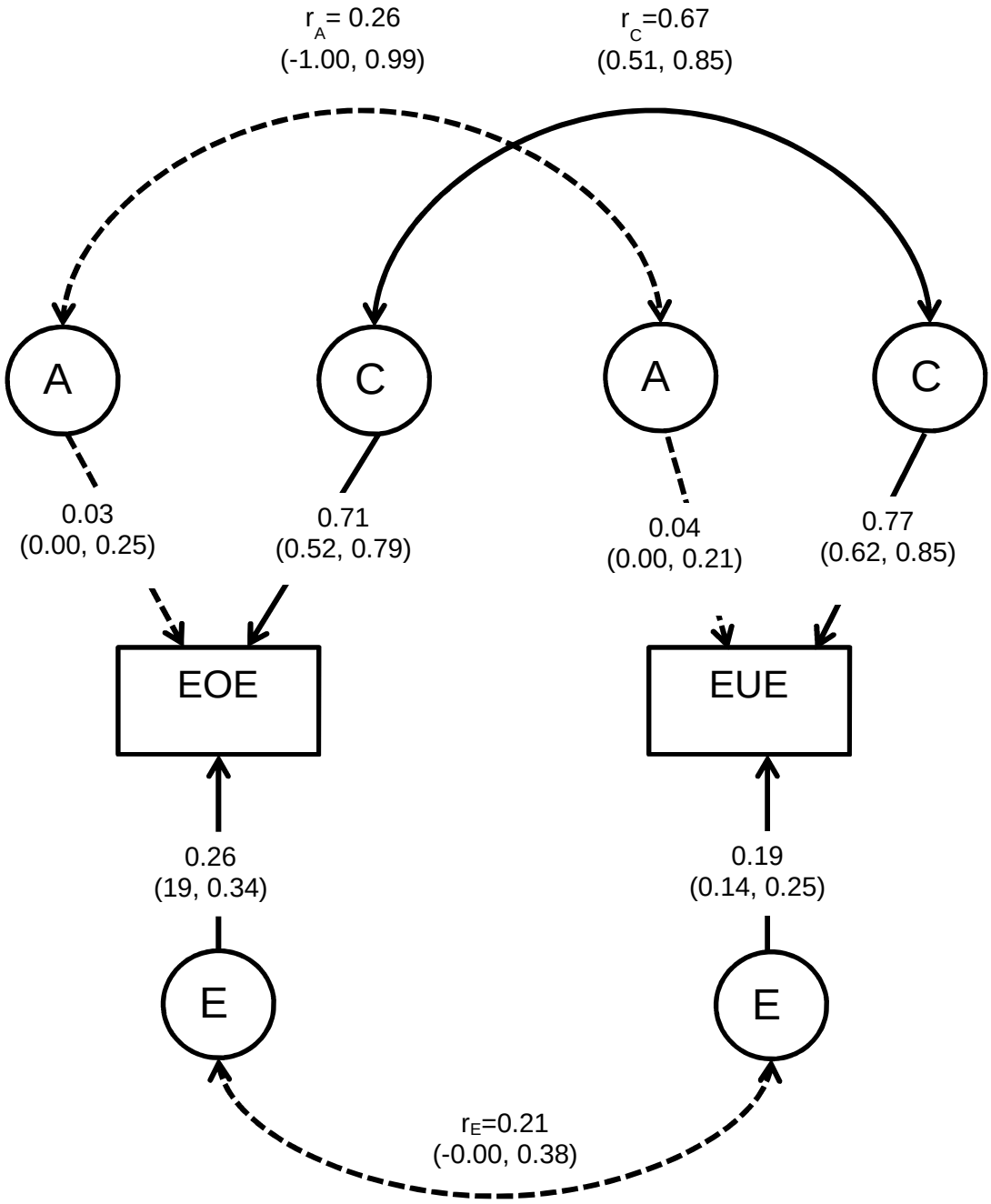


**Figure 9.2 Correlated Factors Model of EOE and EUE in the TEDS subsample**

*The rectangular boxes represent the measured phenotype (emotional overeating, EOE and emotional under-eating, EUE) using the Child Eating Behaviour Questionnaire at four years of age. The circles indicate the latent factors: additive genetic effects (A), shared environmental effects (C) and non-shared environmental effects (E). The straight single-headed arrows reflect pathways with the variance explained by each latent factor (including 95% confidence intervals, CI). The non-significant A paths, with 95% CIs crossing 0, are represented as dotted lines. The aetiological correlations are shown on the curved double-headed arrows. These indicate the extent of common genetic ($r_A$), shared environmental ($r_C$) and non-shared environmental ($r_E$) influences across the two phenotypes. The non-significant aetiological correlations ($r_A$ and $r_E$), with 95% CI crossing 0, are represented as dotted lines.*

**Sex differences**

Study 2 (**Chapter 5**) concluded that there were no significant qualitative and quantitative sex differences underlying the aetiology of EOE and EUE. The TEDS sample in this study only consisted of same-sex twin pairs. Full sex-limitation models were therefore not be conducted. Moreover, the sample size is too small to run sex limitation models, because in order to do so the sample needs to be split by sex *and* zygosity (rather than simply by zygosity), resulting in small group sizes.

## 9.5 Discussion

### 9.5.1 Summary of findings

The analyses presented in this chapter act as a replication of the findings shown in Study 2 (**Chapter 5**). The results corroborated with those in Gemini, highlighting the substantial contribution of environmental factors shared between twin pairs underlying individual differences in EOE and EUE. **Table 9.6** directly compares the estimates for Gemini and TEDS. Just as in Study 2, heritability was low for EOE (3%, 95% CI: 0, 25%) and EUE (4%, 95% CI: 0, 21%), and shared environmental factors explained the majority of individual differences in the two behaviours (EOE: C = 71%, 95% CI: 52, 79%; EUE: C = 77%, 95% CI: 62, 85%). One of the main differences between the two sets of results was the considerable contribution of non-shared environmental factors to both EOE and EUE in TEDS (EOE: E = 26%, 95% CI: 19, 34%; EUE: E = 19%, 95% CI: 14, 25%). However, due to the small sample size in TEDS, the confidence intervals were much wider, making it difficult to directly compare the estimates to those from Gemini.

Again, the phenotypic correlation between EOE and EUE was substantial (r = 0.53), surpassing even the correlation observed in Study 2 (r = 0.43), and those reported by previous other studies (r = 0.16 – 0.30) (Domoff et al., 2015; Ek et al., 2016; E. F. Sleddens et al., 2008; Viana et al., 2008; Wardle et al., 2001). The aetiological correlations were similar, indicating that about 45% ($r_C$ = 0.67; $0.67^2$ = 45%) of the shared environmental effects underlying EOE also influence EUE (compared to 24% in Gemini). Again, the majority of the phenotypic correlation between the two behaviours was being driven entirely by these common shared environmental effects (BivC = 0.50, 95% CI: 0.35, 0.60), with the remaining bivariate estimates being non-significant (BivA = -0.01, 95% CI: -0.10, 0.12; BivE: 0.05, 95% CI:-0.00, 0.09).

### 9.5.2 Implications

In summary these findings - the aetiology of EOE and EUE in an independent sample - confirm the findings already presented in this thesis. Overall, individual differences in EOE and EUE are largely due to environmental factors shared between the twins. Genetic effects played only a very minor role. The phenotypic correlation between the two phenotypes was large, and mainly driven by shared environmental effects, some of which were unique to EOE or EUE, and some of which are shared between them.

### 9.5.3 Strengths and limitations

The aetiology of EOE and EUE in childhood remains an understudied research question. Study 2 described the first study ever undertaken to understand the association between EOE and EUE in childhood using a twin design. The fact that the overall findings were replicated in this study provides support for the results from Gemini.

This replication benefits from using the same measures of EOE and EUE - the CEBQ - as well as from being conducted with a sample of similar age and ethnicity. However there were some distinct differences between the samples. This TEDS subsample was specifically selected to oversample overweight parents. Importantly, the twins themselves were also heavier than the Gemini twins and showed greater variation in BMI. The sample sizes differed substantially (Gemini: n = 2054 versus TEDS: n = 394). The reduced sample size in this study led to some differences in the reliability of the estimates, especially in the univariate analyses. Here, in contrast to Study 2 (**Chapter 5**), the MLSEM suggested that a reduced model only including the latent factors C and E, was preferred over the full ACE model. However, small sample sizes result in imprecise estimates, with large confidence intervals. Hence, it is not recommended that reduced models are interpreted in twin studies of small size. Interpretation of submodels from small samples has the potential of overestimating the effect of one latent factor over another and is not recommended. Therefore the interpretation focuses on the results from the full models including all three latent factors (A, C and E).

In addition, differences might have arisen from discrepancies in the variation in EOE in the two samples. Participating TEDS families were selected for high or low risk of obesity. This selection might have led to a wider distribution of EOE scores amongst the children. As seen in the histograms in **Figure 9.1**, scores seem to be normally distributed for EOE, which differs from EOE scores in Gemini at five years of age. This increased variation in EOE and EUE, which might have influenced the observed ICCs. In comparison to the estimates from Study 2**,** overall the TEDS twin pairs were less similar to each other, resulting in higher estimates of the non-shared environmental factors. The ICCs for MZ pairs include all factors that contribute to twin similarity, namely and genetic and shared-environmental effects. Therefore very high ICCs, as shown in Study 2, can be interpreted as a lack of variation in the phenotype, or a systemic error (correlated error) in the measure, leading to similarity of scores across twin pairs. The fact that the ICCs are lower for twins here in an independent sample, supports the measure, as such continuous measurement errors should be

expected to be present in both samples. Additionally, because TEDS research assistants were present when parents completed the CEBQ, parents were able to ask any questions. Therefore, TEDS parents might have rated their twins more accurately.

This subsample of TEDS only included same-sex twin pairs, and the sample was considerably smaller than Gemini. Full sex limitation models could therefore not be conducted, which meant it was not possible to provide a direct replication of the sex differences models presented in Study 2 (**Chapter 5**). More twin research is needed including the analyses of opposite-sex twin pairs.

## 9.6 Conclusion

The results presented in this chapter generally replicated the findings from Study 2. Both EOE and EUE were found to be of low heritability in middle childhood, with shared environmental effects explaining the majority of individual differences. In line with many previous studies (**Chapter 1.3.2.3)**, EOE and EUE were correlated positively, and in line with findings from Gemini, the association was explained entirely by shared environmental factors. In this sample, EOE scores were more varied resulting in lower twin correlations overall. This differences between the samples supports the validity of the CEBQ subscales of EOE and EUE as it suggests that the high shared environmental effect did not result from correlated error in reporting by parents.

Parent rated questionnaires, like the CEBQ, have been widely used to study the aetiology of eating behaviours in infancy, early and middle childhood. Results in this Study 6 replicated previous findings and therefore support the validity of the measure. However, parental ratings of child eating behaviour might be influenced by the parents' belief of their twins' zygosity. The presence of this potential bias was tested in the final Study 7 in the following **Chapter 10**.

**Table 9.5 Estimates for A, C and E, as well as aetiological correlations ($r_A$, $r_C$, $r_E$) for data analysed in Gemini (Chapter 5) in comparison to estimates in this chapter**

| **Gemini at 5 years (n = 2054)** | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **EOE** | | | **EUE** | | | **Aetiological correlations** | | |
| **A** | **C** | **E** | **A** | **C** | **E** | **$r_A$** | **$r_C$** | **$r_E$** |
| 0.06 (0.05, 0.09) | 0.92 (0.91, 0.93) | 0.02 (0.02, 0.03) | 0.07 (0.06, 0.09) | 0.91 (0.89, 0.92) | 0.02 (0.02, 0.02) | -0.37 (-0.50,-0.23) | 0.49 (0.43, 0.54) | -0.02 (-0.1, 0.08) |
| **TEDS at 4 years (n = 394)** | | | | | | | | |
| **EOE** | | | **EUE** | | | **Aetiological correlations** | | |
| **A** | **C** | **E** | **A** | **C** | **E** | **$r_A$** | **$r_C$** | **$r_E$** |
| 0.03 (0.00, 0.25) | 0.71 (0.52, 0.79) | 0.26 (0.19, 0.34) | 0.04 (0.00, 0.21) | 0.77 (0.62, 0.85) | 0.19 (0.14, 0.25) | 0.26 (-1.00, 0.99) | 0.67 (0.51, 0.85) | 0.21 (-0.00, 0.38) |

Abbreviations: A = additive genetic effects; C = shared-environmental effects; E = non-shared environmental correlation; $r_A$ = genetic correlation; $r_C$ = shared-environmental correlation; $r_E$ = non-shared environmental correlation

# Chapter 10 Study 7: Testing for zygosity-related reporting bias in parents' ratings of their twin children's eating behaviour

## 10.1 Background

The study described in this chapter has been published[5] in the journal *Behavior Genetics*. As discussed in detail in **Chapter 3.5**, the twin method is based on the comparison of resemblance between MZ and DZ twin pairs. One issue is that when analysing infant and child samples, the data collected are often, by necessity, parent rated. This provides the potential for parental bias to occur. In particular, parents might be influenced by their beliefs about their children's zygosity when describing the behaviour of their twins. It is possible that parents who believe their twins to be identical rate them more similarly or conversely if they believe their twins to be non-identical parents may inflate the differences between them. Artificial inflation of similarity within identical twin pairs, or differences between non-identical twin pairs would result in an overestimation of additive genetic effects, and leading to unreliable results. Research described in this thesis uses parent-rated measures of eating behaviours to investigate their aetiology using the twin method. This potential bias would therefore compromise the reliability of the findings. Moving forward, a rigorous examination to rule out this bias is crucial to provide evidence for the reliability of the findings from this thesis.

The presence of this bias can be tested for, by exploiting the fact that some parents hold false beliefs about their twins' zygosity. Researchers can take advantage of misclassifications to test directly for parental rater bias regarding their twins' zygosity. A simple method is to compare the parent-rated similarity of pairs who have been correctly classified as MZ or DZ by their parents, with similarity for pairs who have been misclassified. Previously this 'misclassified zygosity design' has been used in studies of adult twins using self-reported data to test the equal-environments assumption (EEA).

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[5] Herle, M., A. Fildes, C. van Jaarsveld, F. Rijsdijk and C. H. Llewellyn (2016). Parental Reports of Infant and Child Eating Behaviors are not Affected by Their Beliefs About Their Twins' Zygosity. Behavior Genetics. doi: 10.1007/s10519-016-9798-y., See **Appendix 5.4**.
I hereby acknowledge the contribution made to this study by the diligent peer review process, which influenced how the findings are presented in the published paper and this thesis chapter.

The EEA stipulates that environmental factors that contribute to twin pair similarity affect MZ and DZ twins to an equal extent. As outlined in more detail in **Chapter 3.5.7**, research over recent decades has concluded that the EEA is valid (Borkenau et al., 2002; Conley et al., 2013; Felson, 2014b; Morris-Yates et al., 1990; Scarr & Carter-Saltzman, 1979). So far no study has applied this method to samples of young twins and their parents' ratings. A previous study in Gemini showed that about one third of parents were mistaken about the zygosity of their twins, with the majority being MZ twins mistakenly believed to be DZ (C. H. M. van Jaarsveld et al., 2012). This makes it possible to use this design to test for parental rater bias in relation to zygosity, in this sample.

## 10.2 Aim

The aim of this study was to test for zygosity-related parental bias in parent-reported measures of infants' and toddler's eating behaviours in Gemini, using a 'misclassified zygosity' design.

## 10.3 Methods

### 10.3.1 Zygosity classification and parental beliefs

Data analysed in this study were taken from the Gemini cohort (described in **Chapter 3.1**). Only same-sex twin pairs were included as opposite sex twin pairs are always non-identical. In addition to the zygosity questionnaire and DNA genotyping, parents of same-sex twins were asked "Do you think your twins are identical?" at baseline when the children were approximately eight months (n = 1565 parents, mean = 8.17, range = 4.01 - 20.3), and again when they were approximately 29 months old (n = 898 parents; mean = 28.8, range: 22.9 - 47.6). Parents' responses to this question were compared to zygosity classifications derived from the questionnaire and DNA to identify misclassified twin pairs. A more detailed description of the zygosity classification in the Gemini sample is provided in **Chapter 3.1.2**.

### 10.3.2 Eating behaviours

Parents rated their twins' eating behaviours using the BEBQ (Llewellyn et al., 2011) when the twins were eight months old (mean = 8.17, SD = 2.18). When the twins were 16 months old (mean = 15.8, SD = 1.2), parents rated their children's eating behaviour again using the CEBQ-T. A detailed description of these questionnaires can be found in **Chapter 3.2.1** and **3.2.3**. When the twins were three years old parents were informed by the research team about their twins' zygosity. Therefore these analyses could only be

conducted for data collected prior to three years, precluding repetition of these analyses of data collected at five years.

### 10.3.3 Analyses

Four categories were created from the parental classifications of zygosity, and zygosity established using the questionnaire and DNA: (i) MZC, MZ pairs correctly classified as MZ by parents; (ii) MZI, MZ pairs incorrectly classified as DZs by parents; (iii) DZC, DZ pairs correctly classified as DZ by parents; (iv) DZI, DZ pairs incorrectly classified as MZs by parents.

Intraclass correlations ) with 95% confidence intervals were calculated for the four eating behaviours measured by the BEBQ at eight months, and for the six eating behaviours measured by the CEBQ-T at 16 months for each of the correctly classified and misclassified zygosity pairs (MZC, MZI, DZC, DZI). Prior to analyses, scales scores of the BEBQ and CEBQ-T were adjusted for age, sex and gestational age using a regression procedure, because twins are always identical for their age and gestational age (and sex in same-sex pairs). This procedure prevents artificial inflation of similarity. Analyses were conducted using SPSS 22 for windows.

## 10.4 Results

### 10.4.1 Zygosity categories

Researcher-classified and parent-classified zygosity were available for 1528 same-sex pairs of twins at eight months. Overall there was high agreement between the parental and researcher classifications (85.2%). Nevertheless, one third (30.1%, 220/731) of MZ twins were mistakenly believed to be DZ twins by parents. The number of incorrectly classified same-sex DZ twins was small (0.75%, 6/797). At 29 months, data for researcher-classified and parent-classified zygosity were available for 898 twin pairs. Similar to eight months, just under one third of parents (26.3%, 119/453) misclassified their MZ twins as DZ. Again the number of misclassified same-sex DZ twins was low (0.45%, 2/445). **Table 10.1** displays a full breakdown of frequencies and percentages per zygosity category in this sample.

**Table 10.1 Frequencies (n) and percentages (%) of twin pairs in each of the four zygosity categories at eight months and at 29 months**

| Zygosity categories based on parents' beliefs and derived from questionnaire and DNA data | 8 months | | 29 months | |
|---|---|---|---|---|
| | **n** | **%** | **n** | **%** |
| **Total** | 1528[a] | 100 | 898[b] | 100 |
| **MZC*** | 511 | 33.4 | 334 | 37.2 |
| **MZI*** | 220 | 14.4 | 119 | 13.2 |
| **DZC*** | 791 | 51.8 | 443 | 49.3 |
| **DZI*** | 6 | 0.4 | 2 | 0.2 |

Abbreviations: MZ = Monozygotic; DZ = Dizygotic; MZC = MZ pairs correctly classified as MZ by parents; MZI = MZ pairs misclassified as DZs by parents; DZC = DZ pairs correctly classified as DZ by parents; DZI = DZ pairs misclassified as MZs by parents

[a] n is less than the total n for same-sex pairs with researcher-classified zygosity (1549) because it only includes pairs with *both* classified zygosity at 8 months *and* pairs whose parents answered the question "do you think your twins are identical?"

[b] n is less than the total n for same-sex pairs with researcher-classified zygosity (1257) because it only includes pairs with *both* classified zygosity at 29 months (using questionnaire and DNA data) *and* pairs whose parents answered the question "do you think your twins are identical?"

### 10.4.2 Comparison of intraclass correlations for eating behaviours

#### 10.4.2.1 Baby Eating Behaviour Questionnaire

ICCs were calculated for the regressed BEBQ scores for the four zygosity categories: MZC, MZI, DZC and DZI. All ICCs for each of the four eating behaviours by zygosity category are presented in **Table 10.2**. Comparing across the correctly and incorrectly classified twins, results showed no differences in magnitude. Overall MZ twin pairs were rated as much more similar then DZ twin pairs, regardless of whether parents perceived them to be MZ or DZ. For three out of four eating behaviours (Satiety Responsiveness, Enjoyment of Food and Slowness of eating) the 95% CIs overlapped between the correctly and incorrectly classified identical twins, suggesting no statistically significant difference between the two groups. For Food Responsiveness, the ICCs did not overlap. However estimates were very similar and the difference between them was very small (MZC 0.89; MZI, 0.82; ΔICCs= 0.07). Correctly classified DZ twins were consistently rated less similar for all traits in comparison to identical twins. However due to the low number of incorrectly classified DZ pairs (n=6), reliable ICCs could not be calculated for this group, indicated by the large CIs crossing zero.

#### 10.4.2.2 Child Eating Behaviour Questionnaire – Toddler Version

ICCs were also calculated for parent rated eating behaviours at 16 months using the CEBQ-T. Results were similar to the BEBQ; CIs overlapped between correctly and incorrectly classified MZ pairs for SR, FR, FF, EF and SE, suggesting that parental belief about zygosity did not influence parental ratings for these five eating behaviours. For EOE the confidence intervals were virtually the same (MZC, 0.98, MZI, 0.99). Similar to the BEBQ findings, ICCs for non-identical twins were significantly smaller for all CEBQ-T eating behaviours measured. Again, low numbers of incorrectly classified non-identical twins made it impossible to calculate reliable estimates for this group. All ICCs for each of the six eating behaviours by zygosity category at 16 months are also presented in **Table 10.2**

**Table 10.2 Intraclass correlations for eating behaviours measured at eight months (BEBQ) and 16 months (CEBQ-T) by zygosity category**

| **BEBQ 8 months** | **MZC** | **MZI** | **DZC** | **DZI** |
|---|---|---|---|---|
| **SR** | 0.84 | 0.80 | 0.51 | 0.88 |
| **95% CI** | 0.81, 0.86 | 0.75, 0.84 | 0.45, 0.56 | 0.46, 0.98 |
| **n (pairs)** | 502 | 215 | 772 | 6 |
| **FR** | 0.89 | 0.82 | 0.60 | 0.98 |
| **95% CI** | 0.87, 0.91 | 0.77, 0.86 | 0.55, 0.64 | 0.87, 0.99 |
| **n (pairs)** | 500 | 215 | 768 | 6 |
| **EF** | 0.80 | 0.80 | 0.47 | 0.80 |
| **95% CI** | 0.76, 0.83 | 0.75, 0.85 | 0.41, 0.52 | 0.1 0.97 |
| **n (pairs)** | 499 | 212 | 769 | 6 |
| **SE** | 0.82 | 0.82 | 0.40 | 0.28 |
| **95% CI** | 0.79, 0.85 | 0.77, 0.86 | 0.39, 0.46 | -0.45, 0.85 |
| **n (pairs)** | 502 | 216 | 772 | 6 |
| **CEBQ-T 16 months** | **MZC** | **MZI** | **DZC** | **DZI** |
| **SR** | 0.93 | 0.94 | 0.62 | 0.36 |
| **95% CI** | 0.91, 0.94 | 0.92, 0.96 | 0.55, 0.67 | -0.71, 0.99 |
| **n (pairs)** | 308 | 113 | 413 | 2 |
| **FR** | 0.95 | 0.96 | 0.66 | -0.21 |
| **95% CI** | 0.93, 0.96 | 0.94, 0.97 | 0.6, 0.71 | -0.77, 0.99 |
| **n (pairs)** | 308 | 112 | 412 | 2 |
| **EF** | 0.92 | 0.92 | 0.59 | 0.77 |
| **95% CI** | 0.90, 0.94 | 0.88, 0.95 | 0.52, 0.65 | -0.66, 1.00 |
| **n (pairs)** | 308 | 113 | 413 | 2 |
| **FF** | 0.91 | 0.88 | 0.55 | 0.84 |

| | | | | |
|---|---|---|---|---|
| **95% CI** | 0.88, 0.92 | 0.82, 0.92 | 0.48, 0.62 | -0.81, 0.98 |
| **n (pairs)** | 308 | 113 | 413 | 2 |
| **EOE** | 0.98 | 0.99 | 0.90 | -0.83 |
| **95% CI** | 0.97, 0.98 | 0.98, 0.99 | 0.88, 0.92 | -1.00, 0.17 |
| **n (pairs)** | 308 | 113 | 412 | 2 |
| **SE** | 0.88 | 0.88 | 0.43 | 0.99 |
| **95% CI** | 0.85, 0.90 | 0.84, 0.92 | 0.35, 0.50 | 0.99, 1.00 |
| **n (pairs)** | 308 | 113 | 413 | 4 |

Abbreviations; SR = Satiety Responsiveness; FR = Food Responsiveness; EF= Enjoyment of Food; FF = Food Fussiness; EOE = Emotional Overeating; SE = Slowness of Eating; MZC = MZ pairs correctly classified as MZ by parents; MZI = MZ pairs misclassified as DZs by parents; DZC = DZ pairs correctly classified as DZ by parents; CI = Confidence Interval.

## 10.5 Discussion

### 10.5.1 Summary of findings

This is the first study to use the 'misclassified zygosity design' to test for parental bias in reporting infants' and children's eating behaviours. Similar to one previous study (Ooki et al., 2004) a significant proportion of Gemini parents were misinformed about the zygosity of their twins. Approximately 15% of parents of same-sex twins held a false belief about their twins' zygosity. Almost one third (30%) of parents of identical twins believed them to be non-identical, while very few (<1%) parents believed their non-identical twins to be identical. These rates reported here are lower than in Ooki et al (2004), who indicated that of 36.4% of their twin sample was misclassified. Comparisons of similarity in ratings of eating behaviour between correctly classified and misclassified MZ twin pairs, revealed little difference between the two groups at either eight months or 16 months of age. Furthermore, MZ twin pairs were consistently more similar than DZ twin pairs for all eating behaviours at both ages. These results suggest there is no zygosity-related bias in parents' ratings of their infants' and toddler's eating behaviours.

### 10.5.2 Implications

The twin method has been a successful tool for investigating the aetiology of complex human traits. An extensive meta-analysis found that over the past century, twin research has been employed to investigate to the aetiology of over 17000 human traits worldwide, suggesting various estimates of genetic and environmental influences, with well-established high heritability estimates for individual differences in BMI and IQ (T. J. Polderman et al., 2015).

Since its inception, critique of the underlying assumptions of the twin method has been crucial for its continued success and development. As discussed in **Chapter 3.5.7**, multiple previous studies have explored and confirmed the validity of twin research (Conley et al., 2013; Felson, 2014b). In adults, previous research examined the effect of self-reported zygosity on twin similarity in adult eating patterns. Identical twins were much more similar to each other than non-identical twins regardless of their self-reported zygosity (Gunderson et al., 2006). In comparison to this and other previous research focussing on *self-reported* zygosity, this represents one of the first studies to focus on *parent-reported zygosity* and its influence on parent-rated measures of their children's behaviours. Parent measures are often the only way to collect large quantities of data on

children in a reliable and pragmatic manner. Hence parent-rated questionnaires and observations have been crucial for research investigating aspects of child development and have been used in many twin studies investigating the genetic and environmental contribution to individual differences among children, such as depressive symptoms (Thapar & McGuffin, 1994a), temperament (Scott et al., 2016) and attention difficulties (Chang, Lichtenstein, Asherson, & Larsson, 2013). In keeping with the findings from adult studies, this research suggests that parents' ratings are not influenced by their belief about their twins' zygosity, as twin correlations were of the same magnitude regardless of parents' beliefs. Multiple previous twin studies have investigated the genetic and environmental aetiology of infant and child eating behaviours using parent-rated questionnaires (Fildes et al., 2016; Llewellyn, van Jaarsveld, et al., 2010; Llewellyn, van Jaarsveld, Boniface, Carnell, & Wardle, 2008; Smith et al., 2016) and the results of this study validate these previous findings, indicating that parents' ratings of their twin children's eating behaviour were not biased by their beliefs about their zygosity.

This study also highlights that parents are often misinformed about the zygosity of their twins. Previous research suggests that parental misclassification of MZs and DZs often stems from false information given by health professionals (C. H. M. van Jaarsveld et al., 2012). In this study, the majority of parents (n = 1375, 96.4 %) agreed with the health professional's opinion about their twins' zygosity. This overall agreement suggests parents trust health professionals and base their own opinion on the judgements of clinicians. However, many health professionals use prenatal scans to determine zygosity which can be misleading. Scans that show twins are dichorionic (each twin has their own placenta) are often seen as an indicator of di-zygosity, regardless of the fact that approximately one third of MZ twin pairs also develop with separate placentas (Hall, 2003). It is much less likely that parents would be misinformed by a prenatal scan about their DZ twins being MZ; only very rare cases have been reported in which originally dichorionic twins' placentas fuse, resulting in monochorionic presentation at prenatal scans. The rarity of these cases makes it very unlikely that monochorionic DZ twins are incorrectly classified as MZ (Chen, Chmait, Vanderbilt, Wu, & Randolph, 2013; Nylander & Osunkoya, 1970).

A previous study investigated obstetricians' knowledge of twin chronicity and zygosity status. Of the 430 physicians included, only 4 answered all questions correctly, and scores across the whole group averaged at 57% correct answers. These knowledge gaps

among health professionals regarding twin prenatal development have been suggested as the cause of misinformation (Cleary-Goldman, Morgan, Robinson, D'alton, & Schulkin, 2005). Reliable classification of zygosity in same-sex twins is not only crucial for twin research but also for medical reasons, such as prenatal diagnosis of genetic disease or disorders and transplant compatibility, as well as the identity and social development of the children (Hall, 2003; Stewart, 2000).

### 10.5.3 Strengths and limitations

This study has a number of advantages over previous research. It is the first study to examine the potential for bias in parents' ratings of their twin children's behaviour, in relation to their beliefs about their twins' zygosity. It therefore addresses one of the remaining gaps in the critical evaluation of the twin method. Furthermore, repeated measures of parental beliefs at eight months and 29 months enable the investigation of bias at two separate time points, providing robust evidence. Overall the sample size was large, producing estimates with narrow confidence intervals for the majority of the zygosity categories.

Nevertheless, there were also a number of limitations that need to be considered. The number of misclassified DZ twins was very small; correlations for this group were therefore often not significant or had very wide confidence intervals, preventing meaningful interpretation. Larger samples including a greater proportion of misclassified DZ twins would allow for direct comparisons between correctly and incorrectly classified DZ pairs, which would enable a more comprehensive exploration of zygosity-related parental bias. The occurrence of DZ twins misclassified as MZ by a health professional is very rare; it was therefore expected that there would only be a small number of these pairs in Gemini. A previous study investigating parental zygosity misclassification from Japan (1244 families) found a slightly higher number of misclassified DZ twins (10.6%) (Ooki et al., 2004). Importantly this study reported higher rates of misclassifications overall, showing that in total 36.4% of the parents held a false belief about their twins' zygosity status. Data analysed in this study was an aggregation of smaller samples across past decades starting at 1989 till 2002, which might have introduced variation in parental knowledge, hospital practices and technological advances regarding twin zygosity classification. Furthermore, the exact age of the twins at zygosity classification could have influenced the accuracy. Heterogeneity across the decades might be reflected in higher rates of misclassification overall (Ooki et al., 2004). In Gemini all twins

were born within the same year (March to December 2007), and it is reasonable to assume that information and procedures across different hospitals were fairly similar.

In addition to sample size, a further limitation is introduced by the zygosity allocation in Gemini. For the majority of the sample zygosity was ascertained using a zygosity questionnaire sent to parents when the twins were eight and 29 months old. When comparing questionnaire results collected at eight months with all available DNA collected, zygosity allocation matched for 87.5% of the sample. For data collected at 29 months the accuracy of the questionnaire was higher at 96.8 %; indicating that the questionnaire may be slightly more accurate for toddlers than infants. Children are likely to become more distinct as they grow up, so it seems reasonable that parent-rated zygosity is slightly more accurate when the twins are older. For 624 pairs zygosity was allocated using only the questionnaire at eight months, for these pairs zygosity may therefore by slightly less accurate. Regarding rates of overall accuracy, it is also important to acknowledge that DNA was only used to zygosity-test a subset of the sample which included twin pairs who were difficult to classify (pairs for whom there was a mismatch between the zygosity questionnaire results between 8 months and 28 months, and pairs whose parents requested a DNA test, implying that they were uncertain about their twins' zygosity), as well as a random sample of 81 pairs. For the *random* sample only there was a 100% match between questionnaire and DNA zygosity classification. Even though zygosity can be accurately classified using a parental questionnaire for most twin pairs, DNA genotyping remains the gold standard for zygosity ascertainment and should ideally be available for every twin pair. Nevertheless, zygosity testing using DNA is costly and the use of a questionnaire is more feasible for large cohorts like Gemini.

Lastly, this study focuses exclusively on child eating behaviours. Findings cannot necessarily be extrapolated to other child behaviours and more research is needed to test for parental biases related to other parent reported phenotypes, such as childhood temperament (Saudino, 2005), childhood depressive symptoms (Thapar & Mcguffin, 1994b) and childhood anxiety (Eley et al., 2003).

Moreover, the research team informed the parents of their twins' zygosity when they were three years old. Therefore the conducted analyses could not be repeated for the eating behaviour data collected at five years. Nevertheless, findings suggesting no parental bias

regarding zygosity at two prior time points (infancy and toddlerhood) suggest the validity of the parent-reported questionnaires in general.

## 10.6 Conclusion

This study provides evidence that parents' ratings of their children's eating behaviours are not influenced by their beliefs about the twins' zygosity. These findings validate previous twin studies investigating the heritability of eating behaviours in childhood. Twin analyses were the predominant research method in this thesis. Studies 1, 2, 5 and 6 (**Chapters 4, 5, 8 & 9**) used twin methodology to examine the aetiology of children's emotional overeating and emotional under-eating for the very first time, using the CEBQ-T and CEBQ. The outcomes of this final study support this approach providing confidence that the results in this thesis are not affected by zygosity-related parental bias. Together with the previous **Chapter 9**, Study 6, this study was conducted to test for twin-specific parental rating bias in the main measure used in this thesis. Replication studies and tests of measures are an essential aspect of rigorous scientific practice, and findings from these two studies support the findings produced in this thesis.

# Chapter 11 Discussion

## 11.1 Summary of findings

The overall purpose of this thesis was to investigate the aetiology of emotional overeating and under-eating in childhood. Five aims were addressed in seven studies **(Chapters 4-10)**. The findings relating to each aim are summarised below.

### 11.1.1 Aim 1: Use a twin design to establish the genetic and environmental contributions to individual differences in emotional over- and under-eating in toddlerhood and middle childhood

Studies 1 and 2 (**Chapter 4** and **5**) described results from the first twin studies of child emotional over- and under-eating. Overall, individual differences in both behaviours were mainly explained by shared environmental factors. Heritability estimates were low for both EOE and EUE (6% and 7%), and stand in contrast to heritability estimates derived for a number of other child eating behaviours, such as Enjoyment of food (63%), Satiety responsiveness (75%) (Carnell & Wardle, 2008), and Food fussiness (78%) (Fildes et al., 2016). Results suggested that both emotional overeating and under-eating are heavily shaped by environmental factors within a family (EOE, 92%; EUE, 91%), while genetic effects are less important.

Study 1 (**Chapter 4**) exploited repeated measures of EOE in the Gemini cohort. EOE was found to track moderately from toddlerhood (16 months) to middle childhood (five years) (r = 0.25). Twin modelling results revealed that this longitudinal association was driven almost entirely by shared environmental effects, meaning that continuing factors family environmental factors from toddlerhood to middle childhood accounted for the stability in child emotional overeating across this time. However, the shared environmental effects at play also differed substantially between the two time points. Only about 8% ($r_C$ = 0.29, $0.29^2$ = 0.08) of shared environmental factors affecting EOE at 16 months also influenced EOE at five years. These findings reflected the developmental differences between children at 16 months and five years of age, and the difference in influence on this behaviour at five years of age compared with 16 months. This makes sense given the different food environment they are exposed to, as they get older. As children mature, and start school (at approximately 4.5 years of age) they may spend significantly more time outside the family home than they did as young toddlers, and the

gains in independence that they make means that they can also take more active choices regarding how much and what kinds of food they eat.

### 11.1.2 Aim 2: Use a twin design to establish the extent of common genetic and environmental influence underlying emotional over- and under-eating, and the extent to which common influences explain their positive association

Somewhat counter-intuitively, previous studies have consistently described a significant positive correlation between emotional over and under-eating in children (Domoff et al., 2015; Ek et al., 2016; Mallan et al., 2013; Steinsbekk, Belsky, & Wichstrom, 2016; Viana et al., 2008; Wardle et al., 2001). Results from Study 3 (**Chapter 6**) confirmed these findings, revealing a strong positive correlation between EOE and EUE in Gemini ($r = 0.43$) when the twins were five years old. A bivariate twin model showed that even though both behaviours are marked by a similar aetiology - low heritability and a strong effect of the shared environment - their underlying aetiologies are, in fact, fairly distinct. Results suggested some overlap between the shared environmental factors influencing the two behaviours, with about a quarter of shared environmental factors on both EUE and EOE being the same ($r_C = 0.49$, $0.49^2 = 0.24$). However, this means that the majority of the shared environmental factors on EOE and EUE are specific to each behaviour. However, the positive correlation between EOE and EUE was entirely explained by the shared environmental factors that were common across the two behaviours. This suggests that children who emotionally overeat are also likely to engage in emotional under-eating to some extent, and that common aspects of the shared home or family environment explain the positive relationship between these two behaviours. However, the majority of factors within the shared home environment driving child emotional overeating are in fact different from those behind emotional under-eating. Findings imply that emotional over- and under-eating are learned in childhood.

### 11.1.3 Aim 3: Characterising the early life correlates and shapers of emotional over- and under-eating in childhood

Study 3 (**Chapter 6**) aimed to identify child, parental and home environmental factors associated with *both* EOE and EUE, as well as factors *specific* to each behaviour. Results from the twin study (Study 2 (**Chapter 5**)) indicated that some environmental factors are affecting *both* behaviours whereas others are *specific* to either EOE or EUE. However, twin studies provide no indication as to the actual factors involved. In order to identify potential early life factors that may be involved, a large battery of variables were

examined in relation to EOE and EUE, including household stress, indicators of family socio-economic status, early milk-feeding (bottle- versus breastfeeding, and feeding philosophy), maternal eating behaviour, parental feeding practices and child emotion regulation.

Results suggested that parental emotional feeding was significantly associated with *both* EOE *and* EUE in childhood, whereas parental pressure to eat was exclusively associated with child EUE. Moreover, maternal emotional eating had a small but significant association with EOE, but not with child EUE. However, these analyses were cross-sectional precluding causal inference. Research designs that are able to establish causality are needed to provide evidence that these factors are causally involved.

Study 4 (**Chapter 7**) used structural equation modelling to analyse repeated measures of child EOE and parental emotional feeding at 16 months and five years. These bi-directional prospective analyses enabled the examination of the causal direction of the relationship. Results suggested that greater parental emotional feeding at 16 months predicted increases in child EOE from 16 months to five years, with no effect in the other direction (from child EOE at 16 months to parental emotional feeding at five years). This study suggested that parental emotional feeding plays a causal role in the development of child EOE. There was no evidence that parental emotional feeding develops in response to child EOE. These results were in line with a previous longitudinal study in toddlerhood (Rodgers et al., 2013). However, a longitudinal study following children from four to ten years suggested a bi-directional relationship between emotional feeding and child emotional overeating, which may start to emerge in later childhood as emotional eating becomes more established (Steinsbekk et al., 2017).

### 11.1.4 Aim 4: Use the twin design to test if the aetiology of emotional over- and under-eating varies by level of environmental stress (gene-environment interaction)

Study 5 (**Chapter 8**) aimed to test whether the level of household stress, measured with the Chaos, Hubub and Order Scale (CHAOS) in a child's home moderates the genetic influence on EOE and EUE. Results suggested significant gene-environment interactions affecting both behaviours. The heritability of both EOE and EUE increased significantly as a function of increasing household stress. Overall the effects of the interactions were small, but the variance explained by genetic effects at the highest level of household

stress was four times larger than in the absence of any household stress. Findings suggested that with adverse home environments the effect of genetics on emotional eating behaviours increases. This positions EOE and EUE within the diathesis-stress model, in which biological factors increase with growing exposure to environmental stressors. It has been suggested that the diathesis-stress model underlies individual differences in weight, whereby exposure to a highly obesogenic environment increases genetic effects (Min et al., 2013). However, regardless of the gene-environment interaction, shared environmental factors remained the most important factors, accounting for the majority of the variance in emotional eating regardless of household stress levels.

### 11.1.5 Aim 5: Replicate the twin study findings in an independent sample, and test for twin-specific parental bias in parents' reports of child eating behaviours

Replication of findings in independent samples is a crucial aspect of science. Study 6 (**Chapter 9**), was conducted as a replication of Study 2. EOE and EUE were measured in an independent sample of twins, a subsample of the Twins Early Development Study (TEDS), at a comparable age (four years) to Gemini in Study 2. Results confirmed the low heritability of EOE (3%, Gemini: 6%) and EUE (4%, Gemini: 7%), and replicated the high contribution of shared environmental factors for children's emotional eating behaviour (EOE: 71%; EUE: 77%, Gemini: EOE: 92%, EUE: 91%). In contrast to Study 2, the analyses in this sample indicated a greater effect of non-shared environmental factors. In Gemini, in Study 2, non-shared environmental factors accounted for 2% of individual differences in EOE and EUE. In TEDS, non-shared environmental effects were much larger for EOE (26%) and EUE (19%).

Again, EOE and EUE were substantially positively correlated ($r = 0.53$). In comparison with the findings from Gemini, a larger proportion (45% versus 24%) of shared environmental factors were found to influence both EOE and EUE, confirming that the aetiology of EOE and EUE is partially shared. However, there was also substantial environmental specificity underlying individual differences in EOE and EUE. In line with the findings from Study 2, the positive correlation between EOE and EUE was entirely explained by common shared environmental factors. Overall, Study 2 and Study 6 reported similar findings, confirming the importance of shared environmental factors in the aetiology of child EOE and EUE. Furthermore, the findings affirmed that genetics play only small role in variation in these two behaviours.

Importantly, there were some key differences between the two samples: the TEDS subsample included more families classified as of lower and intermediate socio-economic background. Furthermore, the TEDS parents were purposefully selected to increase the genetic risk for obesity in this sample; half of the parents selected were overweight or obese. As a result, there was more variation in EOE and EUE scores and mothers were younger and had a higher BMI at the twins' birth. Therefore, the replication of similar estimates for genetic and environmental contributions to individual differences in EOE and EUE in middle childhood in TEDS increases confidence in the findings from Gemini in Study 2 (**Chapter 5**).

The final study (Study 7 (**Chapter 10**)) tested for the presence of parental bias regarding their twins' zygosity when rating their eating behaviours. Most twin research of young children uses parent-report questionnaires as a means to collect large quantities of data from children who are too young to complete these measures for themselves. However, these data are potentially biased as parents might rate twin siblings to be more similar to one another if they believe them to be identical twins, compared to if they believe them to be non-identical twins. This can be a problem as previous studies have suggested that up to a third of parents hold a false belief about their twins' zygosity, and these false beliefs might lead to unreliable parental ratings (Ooki et al., 2004; C. H. M. van Jaarsveld et al., 2012). Results from Study 7 highlighted that parental beliefs about their twins' zygosity status did not influence the similarity of their ratings of their twins' eating behaviours. Identical twin pairs whose parents believed them to be non-identical, were rated as similarly as correctly classified identical twin pairs. One of the underlying assumptions of the twin method is that perceived zygosity does not influence the ratings of the behaviours under study. Previous studies have investigated the effects of perceived zygosity in adults (Conley et al., 2013; Gunderson et al., 2006). Study 7 was the first research to test for the effect of perceived zygosity on parental ratings in children, adding substantial support to twin research of child eating behaviours.

## 11.2 Implications

The findings have implications for intervention development, theory and future work. These are laid out in the sections below.

### 11.2.1 Implications for intervention development

Overall, the results highlighted that shared environmental factors are the main driver of individual differences in EOE and EUE in childhood, in contrast to other child eating behaviours (e.g. satiety responsiveness, Carnell et al., 2008). Furthermore, Study 3 highlighted parental emotional feeding as an important potential environmental factor linked to both EOE and EUE, whereas parental pressure was exclusively associated with child EUE. Bi-directional longitudinal analyses provided evidence for a causal link from parental emotional feeding to child EOE.

These findings have direct implications for the future development of interventions addressing maladaptive emotional eating behaviours in childhood. Intervention success might be best achieved by targeting parental feeding behaviours and, specifically, parental emotional feeding. Parents might be in need in clear guidance regarding parental feeding and would benefit from interventions equipping them with alternative solutions for soothing their children without providing food. The Child Feeding Guide developed by children's feeding specialists from the University of Loughborough (http://www.childfeedingguide.co.uk) aims to help parents by offering guidance on how to feed their children. Their guide acknowledges that providing food to make a child feel better can have negative effects on a child's health and suggests that a "Kisses not Cookies" approach should be taken. These excellent recommendations are excellent; more information regarding the association between emotional feeding and child emotional overeating could be added to expand these guidelines.

Parental feeding practices have been targeted in some parental interventions aiming to reduce childhood obesity. A review found seven randomised controlled trials that targeted some aspect parental feeding (Redsell et al., 2016). These trials were very heterogeneous, included a wide age range of children, and consisted of different components designed to affect child energy intake through teaching parents about healthy feeding practices.

Of note is the NOURISH Trial started in Australia in 2009 (L. A. Daniels et al., 2009). In this randomised control trial, 820 first time mothers were allocated to a control or intervention group. The intervention consisted of six bi-weekly group sessions, including education on parental feeding practices and healthy child nutrition, as well as providing a space for peer support, with the aim of improving child food intake and parental feeding

practices. Intervention materials covered a wide range of feeding practices, including emotional feeding. In addition to education on feeding practices, participants were asked to monitor their feeding practices in a workbook and received targeted advice on how to improve. In comparison to the control group, attending the intervention resulted in a small but significant decrease of emotional feeding. This outcome suggests that emotional feeding can be altered through intervention with parents (L. A. Daniels, Mallan, Nicholson, Battistutta, & Magarey, 2013). Moreover, the intervention was found to be effective in reducing child emotional overeating at two years of age. In comparison to the control group, children whose parents participated in the intervention were found to engage in lower levels of emotional overeating two years after the intervention had finished (L. A. Daniels et al., 2014).

The outcomes of the NOURISH Trial provide tentative evidence that targeting parental feeding strategies in a group setting has potential positive long-term effects on their children's eating behaviours, including emotional overeating. Emotional feeding practices were targeted by this intervention, embedded in a larger programme covering a variety of feeding practices such as instrumental and responsive feeding. Results from this thesis imply that emotional feeding is associated with both emotional over- and under-eating and therefore might be a pragmatic intervention target for future studies. Trials targeting emotional over- and under-eating specifically would be needed to fully understand the impact of parental emotional feeding on child emotional eating.

### 11.2.2 Working towards a theoretical framework of emotional over and under-eating in childhood

The research conducted in this thesis has further implications, informing the theoretical understanding of childhood emotional over- and under-eating. In summary results suggest that:

1. Childhood emotional over- and under-eating are mainly driven by environmental factors shared within one family.

2. The aetiologies of childhood emotional over and under-eating are partly distinct. However, common shared environmental factors explain the positive correlation between them.

3. Parental emotional feeding plays a key role in the development of emotional over-eating, and is implicated in the development of emotional under-eating as well.

4. Adverse environmental factors moderate the aetiology of emotional over- and under-eating to some degree. In line with the diathesis-stress model, exposure to negative environments escalates the genetic expression of emotional eating in children.

The following model aims to incorporate these findings into one theoretical framework. A stressful home environment elicits emotional over- and under-eating. The impact of the stressful environment is slightly amplified in the presence of individual genetic risk, in line with the diathesis-model. Specific parental behaviours, such as their own eating behaviour and their feeding practices, influence child emotional over- and under-eating. Together these three factors - a stressful home environment, a genetic susceptibility to emotionally over- or under-eat, and having a parent who emotional feeds them (and tends to emotionally over- or under-eat themselves) - influence a child's tendency to emotional over- or under-eat.

**Figure 11.1** illustrates four different scenarios, indicating how the absence or presence of genetic susceptibility and parental behavioural factors result in differing levels of child emotional eating behaviour. In line with the diathesis-stress model, these influences become magnified in response to a stressful environment. In the model, the presence or absence of genetic susceptibility is indicated by 'Genetics (+)' and 'Genetics (-)' respectively. For parental behaviour, presence is indicated by 'Parents (+)' and absence by 'Parents (-)'. Children living in a household with low stress, no genetic susceptibility and whose parents do not engage in emotional feeding or eating (Genetics (-), Parents (-)) are hypothesised to show lower levels of emotional eating. In contrast, children, living in highly stressful environments, who carry genetic susceptibility associated with emotional eating and whose parents engage in maladaptive behaviours (Genetics (+), Parents (+)) are hypothesised to show the greatest levels of emotional eating. The majority of children will be likely to fall somewhere in between these two extremes, with their emotional eating behaviour being determined by a combination of genetic and parental risk factors in moderately stressful home environments.

The research presented in this thesis has suggested that both emotional over- and under-eating are mainly influenced by shared environmental factors. Genetic effects were found to be minor. In order to reflect this, the proposed model suggests that parental factors

have a greater effect on emotional eating than genetic factors. Therefore children in the ‘Genetics (-) and Parents (+)’ group were hypothesised to engage in higher levels of emotional eating than children in the ‘Genetic (+) and Parents (-)’ group.

Results of this research informed the conceptualisation of the model. However, more research is needed to test the different pathways included. For example, complex models are needed to investigate the interaction of stressful home environments, parental emotional feeding and child emotional eating.

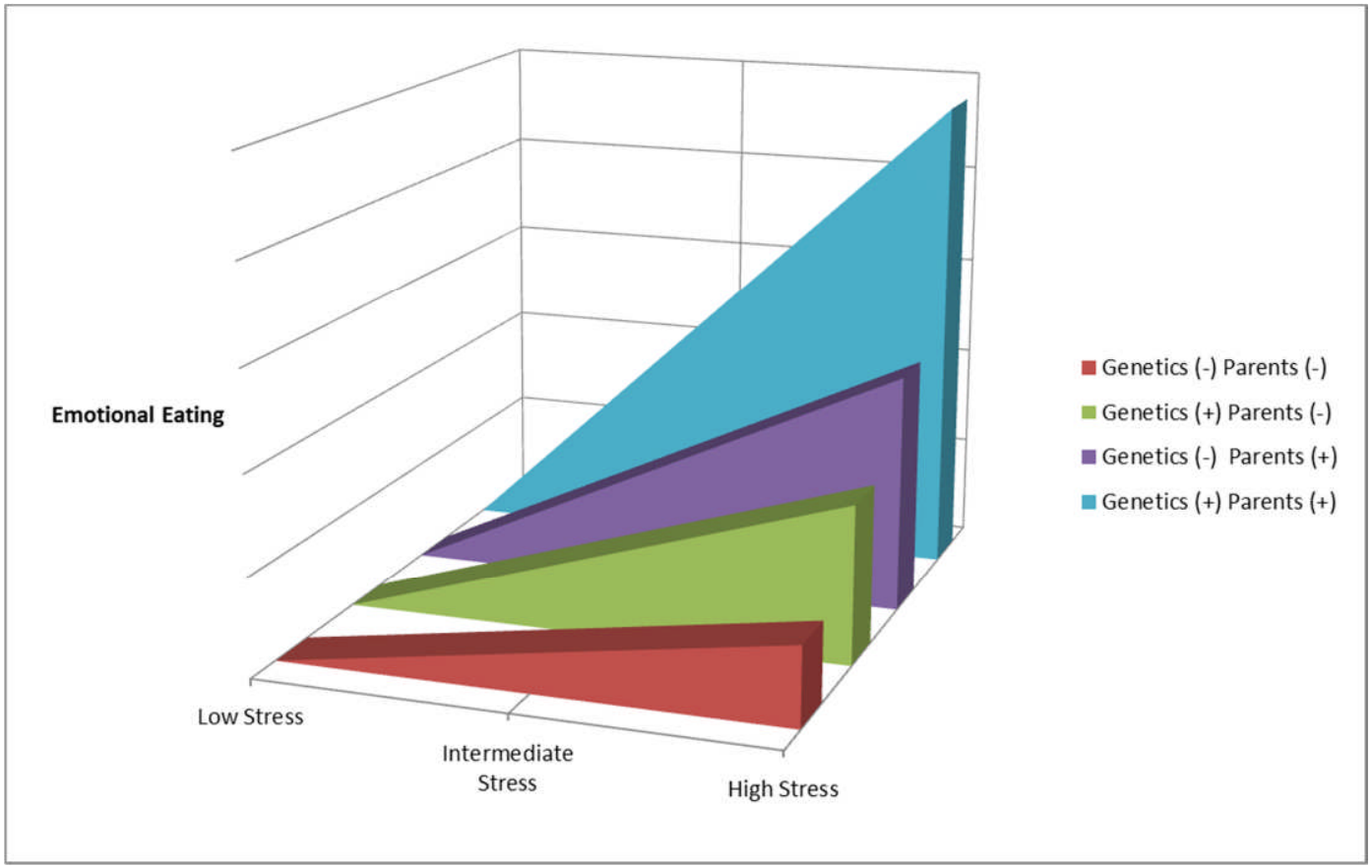


**Figure 11.1 Conceptual model of child emotional eating behaviour incorporating household stress, genetic susceptibility and parental behaviours.**

*Figure displays four different scenarios: absence or presence of genetic risk (Genetics (+), Genetics (-)) and absence or presence of parental behavioural risk (Parents (+), Parents (-)). The effect of genes and parenting is amplified in the face of intermediate and highly stressful home environment.*

## 11.3 Limitations

Various limitations of the individual studies have been described in the accompanying chapters. Additionally, there were a number of *overall limitations* related to much of the research in this thesis. These are discussed in detail below.

### 11.3.1 Representativeness of the sample

The majority of the data analysed in this thesis was drawn from the Gemini birth cohort. As discussed in **Chapter 3**, Gemini is not entirely representative of the population of the United Kingdom (and even less so of non-UK samples). White families are over-represented in the cohort. Furthermore, a large proportion of the sample falls into the highest tertile of the socio-economic classification (NS-SEC). Overall, families of the Gemini cohort are not entirely representative of the wider population. The mothers were slightly older, had a higher level of education, and overall the families were of higher socio-economic status, highlighting the need for replication in different populations, including families of lower socio-economic status. Study 6 (**Chapter 9**) replicated some of the findings (Study 2) in an independent sample (TEDS) of twins of comparable age. Families included in this replication sample differed substantially from the Gemini families insofar as they had a much higher proportion of overweight and obese parents, and children and families were of lower socio-economic status. The findings from Study 7 largely replicated the results from Study 2, supporting the generalisability of the outcomes from Gemini.

Additionally, the representativeness of twin cohorts in general has been questioned, due to the fact that twins sometimes differ from singletons, preventing extrapolation of findings from twins. As described in **Chapter 3.5.7** twins are born earlier and have a lower birth weight than singletons (van Dommelen et al., 2008), but they tend to catch up by the time they are 2.5 years old (Bleker et al., 1979; Luke et al., 1991). Moreover, studies comparing adult twins with age-matched singletons have shown no differences in biological measures such as bone mineral density and blood pressure as well as alcohol consumption or tobacco use (Andrew et al., 2001). In addition to biological measures, there were no differences in personality traits, such as emotionality and aggression (Johnson et al., 2002). Together, these studies support the generalisability of findings from twins to singletons, because they do not differ on several important outcomes studied.

### 11.3.2 Measurement issues

#### 11.3.2.1 Maternal ratings

The majority of measures included in this thesis came from parent–rated questionnaires, which have the potential for bias. Mothers' assessments of their children's eating behaviours might have been influenced by their own eating behaviour and their feeding behaviour, potentially blurring the lines between themselves and their children. This might have resulted in increased correlations between the different psychometric measures, leading to inflated estimates. However, the correlations between child EOE, EUE and maternal eating behaviour derived in Study 3 (**Chapter 6**) were small to moderate, ranging from 0.10 to 0.22. These fairly modest correlation coefficients can be seen as an indication that, if present, the effect of maternal ratings on the correlations between the different variables was small. These correlation may also, of course, reflect genuine relationships between maternal and child eating. Support for this latter explanation comes from a study that investigated parent-offspring correlations in families with adolescent children (n = 639) (de Lauzon-Guillain et al., 2009), using measures of eating behaviours which adolescents and parents rated themselves, removing the possibility of rater bias. Correlations between parents and adolescents were higher, with highest correlation between sons' Uncontrolled eating and fathers' Cognitive restraint eating (r = 0.36) (de Lauzon-Guillain et al., 2009). Overall, the correlations were of a similar or larger magnitude than those observed in this thesis, indicating that with age children and parents may become more similar to one other. This study suggests the maternal rating for both their child's and their own eating behaviours in Gemini did not inflate the association between them.

As well as rating their children's and their own eating behaviours, mothers also rated their feeding practices. Correlations between feeding practices and EOE and EUE varied substantially, ranging from -0.16 to 0.48 (see Study 3, **Chapter 6**). The strongest correlation was between child EOE and parental emotional feeding, which was expected based on previous literature. However, it is possible that this correlation was slightly inflated by the fact that both scales were rated by the mothers, and items probing child behaviour (example item: "My child eats more when worried") and parent behaviour (example item: "I give my child something to eat to make him/her feel better when he/she is worried") were phrased similarly. In order to avoid this potential bias, future research should aim to use child-rated questionnaires to measure child emotional eating

behaviours. However, these are only appropriate for children of sufficient maturity to be able to understand the questions and report on their own behaviour accurately. This simply is not possible with toddlers or young children. In addition, a very large sample was needed for the analyses in this thesis and parent-rated questionnaires were the most pragmatic and feasible option. However, laboratory-based studies are needed to validate these psychometric measures.

Studies 1, 2, 5 and 6 used the twin method to decompose the variation in parent-rated child emotional over- and under-eating into genetic and environmental components. The twin method is based on comparisons of similarity between identical and non-identical twin pairs. The intraclass correlations for EOE and EUE were very high for both types of twins (Studies 1 and 2). These high twin pair similarities formed the basis of the shared environmental effects derived from the maximum likelihood structural equation modelling. At the same time, the identical twin pair differences gave a rough indication of the effect of the non-shared environmental effects, as these are the only factors contributing to differences between identical twin pairs, as genes and shared environmental effects are completely shared. Therefore, the results described in Studies 1, 2 and 5 yielded very low estimates for non-shared environmental effects. This could in fact reflect unreliability of the measures, and be a sign that mothers were not able to fully detect the behaviour in their children, resulting in them scoring the two twins very similarly. This bias would result in increased twin correlations regardless of zygosity, in turn causing an inflation of the shared environmental effects. In comparison to twin studies of the other subscales of the Child Eating Behaviours Questionnaire, the estimates of shared environmental factors were substantially higher for EOE and EUE (Carnell et al., 2008; Fildes et al., 2016; Smith et al., 2017).

However, some support for the measure comes from the replication study discussed in Study 6 (**Chapter 9**). Here the same measures of EOE and EUE were analysed in a separate but smaller and slightly more diverse sample from TEDS. The twin correlations were somewhat lower for both EOE and EUE for both identical and non-identical twins, resulting in higher estimates of the non-shared environment (TEDS: EOE: 26%; EUE: 19%, Gemini: EOE: 2%; EUE: 2%) – of the magnitude typically seen in twin studies of other characteristics such as child BMI (Haworth et al., 2008). Nevertheless, the overall aetiology of EOE and EUE was very similar in the two samples, marked by low heritability EOE and EUE (TEDS: EOE: 3%; EUE: 4%, Gemini: EOE: 6%; EUE: 7%), with the

majority of variance explained by shared environmental effects (TEDS: EOE: 71%; EUE: 77%, Gemini: EOE: 92%, EUE: 91%). This replication study highlights how estimates from twin studies are population specific, as well as supporting the main findings brought forward in this thesis.

In addition, the differences in twin correlations for MZ twin pairs (TEDS; EOE: 0.74, EUE: 0.81; Gemini: EOE: 0.98 and EUE: 0.98) are important and can be seen as a validation of the EOE and EUE measures. If mothers were not able to distinguish between their twins' emotional eating behaviour, this pattern of very high twin resemblance would have been observed in both samples. Therefore, the difference in results between the two samples suggests that mothers can, in fact, distinguish their twins' EOE and EUE.

Considering these two studies together, parental ratings for emotional eating in children are not perfect but are the most pragmatic way to conduct research in very large samples of children. Additionally, psychometric questionnaires provide the advantage of being a standardised measure, whereas laboratory-based measures can vary according to the type of stressor used, the intensity of the stress induced, and the particular emotion induced.

#### 11.3.2.2 Eating in response to boredom and positive emotions

The EOE subscale of the Child Eating Behaviour Questionnaire consists of four items covering different negative emotional states (worried, annoyed, anxious and bored). **Chapter 3.2.1** used Principal Component Analysis to test the factor structure of the scale at 16 months and five years. The factor loadings showed that the item 'My child eats more when s/he has nothing else to do' loaded onto the Food Responsiveness component as well as a component with the other EOE items. This pattern was described in the original development of the scale (Wardle et al., 2001); the boredom was retained in the EOE scale for these analyses in order to facilitate better comparisons with previous research, which has included this item in the EOE scale as well. Similarly, a questionnaire item probing eating in response to boredom was included in the Dutch Eating Behaviour Questionnaire as well ('Do you have the desire to eat when you are bored or restless').

However, previous studies have suggested that eating out of boredom might be a distinct eating behaviour that differs conceptually (and aetiologically) from eating in response to emotions. Koaball et al (2012) extended the Emotional Eating Scale, adding six more items related to eating in response to boredom ("I have the desire to eat more when

feeling: ‘Blah’, ‘Nothing to do’, ‘Unstimulated’, ‘Unexcited’, ‘Restless’ and ‘Disinterested’”) (Koball, Meers, Storfer-Isser, Domoff, & Musher-Eizenman, 2012). The outcomes of the factor analyses confirmed the notion that eating in response to boredom could be considered as a distinct eating behaviour. Research investigating boredom is a growing field, with recent research proposing boredom is distinct from other negative emotions (van Tilburg & Igou, 2017). Boredom is marked by low attention, lack of engagement and low physical arousal, which could be considered substantially different from experiencing sadness, stress or anger (van Tilburg & Igou, 2017).

The research presented in this thesis included measures of emotional overeating using the Child Eating Behaviour Questionnaire and the Child Eating Behaviour Questionnaire – Toddler Version. The latter omitted the item probing eating in response to boredom, because in-depth pilot work with mothers indicated that the item was not appropriate for toddlers. Therefore, the issues around boredom only apply to EOE measured at five years. However, this small difference in EOE items in the CEBQ-T and CEBQ might have influenced the longitudinal studies (Study 1 and Study 4). The absence of the boredom item at 16 months might have weakened the association between EOE at 16 months and five years.

Alongside negative emotions and boredom, positive feelings also have the potential to impact appetite. Eating in response to happiness has been discussed previously and could be considered as a separate eating behaviour altogether. In a previous laboratory-based study (n = 65) participants rated their own emotional eating behaviours using the DEBQ and were allocated to a negative, neutral or positive mood induction using film clips. After watching the films, participants were offered bowls of highly palatable snack foods such as crisps and chocolate. Participants were allowed to eat as much as they wanted and their intake was monitored. When comparing the three mood groups, results revealed that participants rated highly as emotional eaters only consumed more after watching the positive film clip. No effect of the negative or neutral mood induction was found for emotional and non-emotional eaters. These findings highlight that positive emotions can impact food intake as well, and might even be more important than negative emotions (Bongers, Jansen, Havermans, Roefs, & Nederkoorn, 2013).

In addition to laboratory-based studies, natural observations have supported the notion that positive emotions are related to increased food intake. One study asked 43 female

participants (17-25 years) to report all foods consumed over a period of seven days using a diet diary. In addition, participants were asked to indicate their emotional state prior to every eating occasion. Results indicated that unhealthy snack foods were more likely to be consumed following positive emotions (Evers, Adriaanse, de Ridder, & de Witt Huberts, 2013). There is therefore emerging tentative evidence that eating in response to positive emotions might be its own eating behaviour, but little is known about how this behaviour develops. Eating large quantities of highly palatable foods are often part of celebrations and social gatherings, such as birthdays. This pairing of positive mood and eating often starts in childhood and more research is needed to quantify the prevalence and onset of positive emotional eating in childhood. Furthermore, some individuals might be prone to reward themselves with highly palatable foods after achieving a goal or finishing a difficult task.

### 11.3.3 Passive gene-environment correlation

This thesis showed that shared environmental factors explained the majority of individual differences in child EOE and EUE. In turn heritability was low for both behaviours. The high impact of the shared environment could have been inflated due to the presence of a passive gene-environment correlation. Passive gene-environment correlation describes a situation in which the home environment is determined by heritable parental behaviours. Parents pass on the associated genes as well as create a home environment that nurtures the behaviour. Passive gene-environment correlations can lead to inflated correlations between home environmental factors and child outcomes, as well as an overestimation of the effect of the shared environment. In the case of emotional overeating, it has been suggested that parents who emotionally overeat themselves are prone to emotional feeding, which in turn nurtures emotional overeating in their child. In the case of a passive gene-environment correlation, the association between parental emotional feeding and child emotional eating might be explained by the intergenerational transmission of genes associated with emotional overeating instead of the parental behaviour (Rijsdijk & Sham, 2002; Scarr & Mccartney, 1983). However, in the light of the fact that heritability estimates for EOE and EUE are low, the effects of potential gene-environment correlations should be considered small.

Analysing data from families with and without adopted children presents the most straightforward approach to test for the presence of passive gene-environment correlation. In this design the correlation between a family environment (e.g. emotional

feeding practices) and the child behaviour (e.g. emotional overeating) is compared between families with adopted children and families with non-adopted children. A greater correlation in families with non-adopted children, indicates passive gene-environment correlation, as children are exposed to the family environment as well as inheriting associated genes with the home environment and the outcome behaviour (Rijsdijk & Sham, 2002). Adopted families were not included in the Gemini cohort and therefore these analyses were not possible but this would be an interesting direction for future research.

## 11.4 Future directions

The work described in this thesis highlights several potential areas for future research. As already outlined in **Chapter 11.3.2.2** eating in response to boredom has been considered its own independent eating behaviour. Therefore, future research should consider investigating the aetiology of eating in response to boredom separately from other aspects of emotional eating. Furthermore, the impact of positive emotions on food intake and how this behaviour develops needs more investigation.

The Eating Pattern Inventory – Child (EPI-C), as described in more detail in **Chapter 1.3.2.1.2**, measures eating in response to negative and positive emotions. Therefore, this measure would be the ideal tool to compare eating in response to different emotions in childhood. Of course, investigating these different behaviours in a twin sample would have the advantage of enabling the decomposition of variance into genetic and environmental effects.

The main variables of this thesis were child EOE and EUE, measured by the CEBQ and its toddler version CEBQ-T. Both questionnaires were parent reported, and may therefore subject to bias, as discussed in **Chapter 11.3.2.1**. Previous research validated several scales of the CEBQ using observational measures (Carnell & Wardle, 2007). Carnell and Wardle (2007) measured overall energy intake, eating speed and eating in the absence of hunger for 111 five-year-old children, to show that Satiety Responsiveness, Enjoyment of Food and Slowness in Eating relate to real world objective measures of these traits (Carnell & Wardle, 2007). Future research is needed to test the validity of the EOE and EUE subscales as well. Some researchers have developed sensitive and age-appropriate methods for inducing negative mood in children (Blissett et al., 2010; Farrow et al., 2015a). These paradigms would be ideal for testing if child EOE and EUE related

to greater or reduced food intake in response to stress, and offer a method for validating the EOE and EUE subscales of the CEBQ. This work needs to be carried out and would strengthen the findings of this thesis and all other previous research undertaken using these scales.

Study 1 (**Chapter 4**) investigated the genetic and environmental contributions to EOE measured at two time points in early childhood; 16 months and five years. The results suggested that EOE is fairly stable across these four years of life (r = 0.25), despite this period being marked by considerable developmental change. This finding was lower than previous research suggesting that EOE tracks across childhood, from four to 11 years (r = 0.45) (Ashcroft et al., 2008) and from six to eight (r = 0.43) and eight to ten years (r = 0.50) (Steinsbekk et al., 2017). So far only one longitudinal study has investigated the longitudinal stability of EUE, suggesting that this behaviour tracks moderately from four to 11 years (r = 0.29) (Ashcroft et al., 2008). However it is not known how and if emotional over- and under-eating change from childhood to adolescence and later into adulthood. In order to investigate these research questions, longitudinal cohorts with repeated measures of child and adolescent emotional over- and under-eating are necessary. The Gemini cohort will continue to follow the twins, and new data collection is currently underway to measure EOE and EUE at 10 years of age. Furthermore, other bigger and older cohorts would provide the perfect opportunity to follow emotional over- and under-eating through different developmental phases. Emotional eating behaviour has been measured in the TESS (Trondheim Early Secure Study) (Steinsbekk et al., 2017) cohort at ages four, six, eight and ten years and new data collection would be ideal to investigate their development into early adolescence. Similarly, the Generation R Study (Kooijman et al., 2016) has measured eating behaviours, including emotional overeating, at different time points during childhood. Children are about to enter adolescence, and more data on emotional over- and under-eating would enable the longitudinal tracking of these behaviours to be established. Furthermore, these longitudinal studies could investigate the effect of changing from parent-rated to child-rated questionnaires of emotional over- and under-eating, as children grow older and are able to respond for themselves.

## 11.5 Conclusions

In summary, findings from this thesis suggest that childhood emotional over- and under-eating are learned not inherited in early life. The majority of individual differences in both behaviours were explained by shared environmental factors. Genetics only played a

minor role, highlighted by low heritability estimates. In line with previous research EOE and EUE were positively correlated, and although their underlying patterns of genetic and environmental influence was similar, they had fairly distinct aetiologies,. Common shared environmental factors are responsible for the association between EOE and EUE, but the majority of shared environmental influences were in fact specific to each behaviour. Parental emotional feeding was associated with *both* child EOE and EUE, while parental pressure to eat was specifically associated with EUE, and maternal emotional eating was specifically associated with EOE. Longitudinal analyses suggested that parental emotional feeding in toddlerhood shaped the development of child EOE in middle childhood. Finally, adding to the complex aetiology of EOE and EUE, both behaviours were found to fit into the diathesis-stress framework, whereby the genetic susceptibility for both increased in response to a stressful home environment.

The findings in this thesis provide evidence that the aetiology of child EOE and EUE is very different to the aetiologies of all other eating behaviours in childhood, which are often already under strong genetic influence by the first few months of life. Parental behaviours, especially emotional feeding and maternal emotional eating, are promising intervention targets for the prevention of emotional over- and under-eating in toddlerhood when they first start to emerge.

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# Appendices Chapter 1

## Appendix 1.1 Questionnaire items measuring emotional overeating in adulthood

The Dutch Eating Behaviour Questionnaire (DEBQ), the Three Factor Eating Questionnaire (TEFQ), the Emotional Eating Scale (EES), The Adult Eating Behaviour Questionnaire (AEBQ) and the Emotional Appetite Questionnaire (EAQ)

| DEBQ* (12 items) | TFEQ (6 items) | EES (25 items) | AEBQ (5 items) | EAQ (21 items) |
|---|---|---|---|---|
| Desire to eat when irritated… | I start to eat when I feel anxious. | Do you feel a desire to eat when you are resentful | I eat more when I'm upset | As compared to usual do you eat much less/the same/much more when you are sad |
| Desire to eat when bored… | When I feel sad, I often eat too much. | Do you feel a desire to eat when you are discouraged | I eat more when I'm worried | As compared to usual do you eat much less/the same/much more when you are bored |
| Eating when you feel lonely… | When I feel tense or "wound up", I often feel I need to eat. | Do you feel a desire to eat when you are shaky | I eat more when I'm anxious | As compared to usual do you eat much less/the same/much more when you are confident |
| Desire to eat when somebody lets you down… | When I feel lonely, I console myself by eating. | Do you feel a desire to eat when you are worn out | I eat more when I'm annoyed | As compared to usual do you eat much less/the same/much more when you are angry |
| Desire to eat when angry… | If I feel nervous, I try to calm down by eating | Do you feel a desire to eat when you are inadequate | I eat more when I'm angry | As compared to usual do you eat much less/the |

| | | | |
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| | | | same/much more when you are anxious |
| Desire to eat when feeling unpleasant… | When I feel depressed, I want to eat. | Do you feel a desire to eat when you are excited | As compared to usual do you eat much less/the same/much more when you are happy |
| Desire to eat when anxious… | | Do you feel a desire to eat when you are rebellious | As compared to usual do you eat much less/the same/much more when you are frustrated |
| Desire to eat when things go against you… | | Do you feel a desire to eat when you are blue | As compared to usual do you eat much less/the same/much more when you are tired |
| Desire to eat when frightened… | | Do you feel a desire to eat when you are jittery | As compared to usual do you eat much less/the same/much more when you are depressed |
| Desire to eat when disappointed… | | Do you feel a desire to eat when you are sad | As compared to usual do you eat much less/the same/much more when you are frightened |
| Desire to eat when upset… | | Do you feel a desire to eat when you are uneasy | As compared to usual do you eat much less/the same/much more when you are relaxed |
| | | Do you feel a desire to eat when you are irritated | As compared to usual do you eat much less/the |

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| | same/much more when you are playful |
| Do you feel a desire to eat when you are jealous | As compared to usual do you eat much less/the same/much more when you are lonely |
| Do you feel a desire to eat when you are worried | As compared to usual do you eat much less/the same/much more when you are enthusiastic |
| Do you feel a desire to eat when you are frustrated | As compared to usual do you eat much less/the same/much more when under pressure |
| Do you feel a desire to eat when you are lonely | As compared to usual do you eat much less/the same/much more after a heated argument |
| Do you feel a desire to eat when you are furious | As compared to usual do you eat much less/the same/much more after a tragedy of someone close to you |
| Do you feel a desire to eat when you are on edge | As compared to usual do you eat much less/the same/much more after ending a relationship |

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| Do you feel a desire to eat when you are confused | As compared to usual do you eat much less/the same/much more when engaged in an enjoyable hobby |
| Do you feel a desire to eat when you are nervous | As compared to usual do you eat much less/the same/much more after losing money or property |
| Do you feel a desire to eat when you are angry | As compared to usual do you eat much less/the same/much more after receiving good news |
| Do you feel a desire to eat when you are guilty | |
| Do you feel a desire to eat when you are bored | |
| Do you feel a desire to eat when you are helpless | |
| Do you feel a desire to eat when you are upset | |

*Exact item wording cannot be shown because of copyright restrictions.

## Appendix 1.2 Questionnaire items measuring emotional overeating in childhood

The Dutch Eating Behaviour Questionnaire – Parent (DEBQ - P), Dutch Eating Behaviour Questionnaire – Children (DEBQ - C), Emotional Eating Scale – Children (EES - C), Eating Pattern Inventory – Children and Child Eating Behaviour Questionnaire (CEBQ)

| DEBQ – P (13 items) | DEBQ - C (7 items) | EES – C (26 items) | EPI – C (4 items) | CEBQ (4 items) |
|---|---|---|---|---|
| When your child is irritated, does he/she then have the desire to eat? | I have the desire to eat when depressed | I feel a desire to eat when I am resentful | Eating helps me when I am disappointed. | My child eats more when worried |
| When your child has nothing to do, does he/she then have the desire to eat? | I have the desire to eat when worried | I feel a desire to eat when I am discouraged | When I am lonely, I comfort myself with food. | My child eats more when annoyed |
| When your child is depressed or discouraged, does he/she then have the desire to eat? | I have the desire to eat when feeling lonely | I feel a desire to eat when I am shaky | When I am afraid or worried I eat something. | My child eats more when anxious |
| When your child is feeling lonely, does he/she then have the desire to eat? | I have the desire to eat when feeling restless | I feel a desire to eat when I am worn out | I eat when I am unhappy. | My child eats more when s/he has nothing else to do |
| When your child feels let down, does he/she then have the desire to eat? | I have the desire to eat when afraid | I feel a desire to eat when I am not doing enough | | |
| Has your child a desire to eat when he/she is cross? | I have the desire to eat when I feed sorry | I feel a desire to eat when I am excited | | |
| When your child is expecting something unpleasant to happen does he/she then have the desire to eat? | I have the desire to eat when things go wrong | I feel a desire to eat when I am disobedient | | |
| Does your child have the desire to eat when he/she is anxious, worried or tense? | | I feel a desire to eat when I am down | | |
| When things are going against your child or when things have gone wrong, does | | I feel a desire to eat when I am stressed out | | |

he/she then have the desire to eat?

Does your child have the desire to eat, when he/she is emotionally upset'?

Does your child have the desire to eat when he/she is bored or restless?

When your child is frightened, does he/she then have the desire to eat?

When your child is disappointed, does he/she then have the desire to eat?

I feel a desire to eat when I am sad

I feel a desire to eat when I am uneasy

I feel a desire to eat when I am irritated

I feel a desire to eat when I am jealous

I feel a desire to eat when I am worried

I feel a desire to eat when I am frustrated

I feel a desire to eat when I am lonely

I feel a desire to eat when I am furious

I feel a desire to eat when I am on edge

I feel a desire to eat when I am confused

I feel a desire to eat when I am nervous

I feel a desire to eat when I am angry

I feel a desire to eat when I am guilty

I feel a desire to eat when I am bored

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| I feel a desire to eat when I am helpless |
| I feel a desire to eat when I am upset |
| I feel a desire to eat when I am happy |

## Appendix 1.3 Studies reporting mean for EOE and EUE (CEBQ measured) and their correlations with other CEBQ subscales

| **Wardle et al 2001** | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | N = 400 | Age = 4.2 years | 46% Female | | | | | |
| | **FR** | **EF** | **EOE** | **DD** | **SR** | **SE** | **EUE** | **FF** |
| **EOE** (Mean = 1.7-1.9) | 0.49* | 0.16* | 1 | 0.14* | -0.12 | -0.1 | 0.21* | -0.05 |
| **EUE** (Mean = 2.9 - 3.3) | -0.05 | -0.26 | 0.21* | 0.09 | 0.37* | 0.28* | 1 | .13* |

| **Viana et al 2008** | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | N = 240 | Age = 7.9 years | 49% Female | | | | | |
| | **FR** | **EF** | **EOE** | **DD** | **SR** | **SE** | **EUE** | **FF** |
| **EOE** (Mean = 2.24) | 0.27* | 0.55* | 1 | 0.109 | -0.34* | -0.36* | 0.22 | -0.14* |
| **EUE** (Mean = 2.82) | 0.021 | -0.089 | 0.22 | 0.033 | 0.25* | 0.161 | 1 | 0.086 |

| **Svensson et al 2011** | | | | | | | |
|---|---|---|---|---|---|---|---|
| | N = 174 | Age = 3.8 years | 50% Female | | | | |
| | **EOE/FR** | **EF** | **DD** | **SR** | **SE** | **EUE** | **FF** |
| **EOE/FR** (Mean = 1.5 - 1.6) | 1 | 0.26* | 0.39* | -0.16* | -0.23* | 0.07 | -0.02 |
| **EUE** (Mean = 3.2 - 3.3) | 0.07 | -0.01 | 0.17* | 0.33* | -0.03 | 1 | 0.12 |

| **Cao et al 2012** | | | | | | | |
|---|---|---|---|---|---|---|---|
| | N = 219 | Age = 12-18 months | 48% Females | | | | |
| | **FR (1)** | **FR (2)** | **EOE** | **DD** | **SE** | **EUE** | **FF** |
| **EOE** (Mean = 1.75 - 1.82) | 0.16* | 0.16* | 1 | 0.21* | -0.05 | 0.21* | 0.05 |
| **EUE** (Mean = 2.95 - 3.09) | -0.05 | -0.24* | 0.21 | 0.18 | 0.04 | 1 | 0.05 |

FR1
Even if my child is full up s/he finds room to eat his/her favourite food

FR2
My child is always asking for food
Given the choice my child would eat most of the time

If given the chance, my child would always have food in his/her mouth

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Mallan et al 2013** | | | | | | | | |
| Sample 1 | N = 244 | Age = 24 months | 52% Female | | | | | |
| | **FR** | **EF** | **EOE** | **DD** | **SR** | **SE** | **EUE** | **FF** |
| **EOE** (Mean = 1.60) | 0.47* | -0.1 | 1 | 0.22 | -0.03 | -0.07 | 0.28* | 0.12 |
| **EUE** (Mean = 2.99) | -0.06 | -0.35* | 0.28* | 0.16 | 0.42* | 0.30* | 1 | 0.36* |
| Indian Immigrants | N = 203 | Age = 34 months | 51% Female | | | | | |
| | **FR** | **EF** | **EOE** | **DD** | **SR** | **SE** | **EUE** | **FF** |
| **EOE** (Mean = 1.61) | 0.86* | 0.08 | 1 | 0.27 | 0.03 | -0.04 | 0.11 | -0.04 |
| **EUE** (Mean = 3.05) | -0.07 | 0.02 | 0.11 | 0.27 | 0.64* | 0.36* | 1 | 0.02 |
| Chinese Immigrants | N = 216 | Age = 36 months | 48% Female | | | | | |
| | **FR** | **EF** | **EOE** | **DD** | **SR** | **SE** | **EUE** | **FF** |
| **EOE** (Mean = 1.86) | 0.57* | 0.07 | 1 | 0.49* | -0.1 | -0.22 | 0.04 | -0.00 |
| **EUE** (Mean = 3.25) | 0.15 | -0.18 | 0.04 | 0.20 | 0.40* | 0.43* | 1 | 0.29 |
| **Domoff et al 2015** | | | | | | | | |
| | N = 1002 | Age = 4.05 years | 51% Female | | | | | |
| | **FR** | **EF** | **EOE** | **DD** | **SR** | **SE** | **EUE** | **FF** |
| **EOE** (Mean = 1.80) | 0.48* | 0.17* | 1 | 0.23* | 0.01 | -0.01 | 0.3* | -0.1 |
| **EUE** (Mean = 2.91) | 0.15* | -0.06 | 0.3** | .13* | 0.26* | 0.28* | 1 | 0.17* |
| **Russel et al (2016)** | | | | | | | | |
| | EOE; N = 228 EUE; N = 248 | Age = 3.7 years | | | | | | |
| | **FR** | **EF** | **EOE** | **DD** | **SR** | **SE** | **EUE** | **FF** |
| **EOE** (Mean = 1.92) | 0.63* | 0.31* | 1 | 0.06 | -0.15* | -0.09 | 0.16* | -0.04 |
| **EUE** (Mean = 3.15) | -0.01 | -0.03 | 0.16* | 0.25* | 0.40* | 0.22* | 1 | 0.13* |

| Steinsbekk et al (2016) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Sample 1 | N= 797 | Age = 6.7 years | 50.2% Female | | | | | |
| | **FR** | **EF** | **EOE** | **SR** | **SE** | | | |
| **EOE** (Mean = 1.42) | 0.59* | 0.10* | 1 | -0.01 | 0.02 | | | |
| Sample 2 | N = 689 | Age = 8.8 years | | | | | | |
| | **FR** | **EF** | **EOE** | **SR** | **SE** | | | |
| **EOE** (Mean = 1.41) | 0.52* | 0.04 | 1 | 0.04 | 0.05 | | | |

| Ek et al (2016) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | N = 478 | Age = 5 years | 53% Female | | | | | |
| | **FR** | **EF** | **EOE** | **DD** | **SR** | **SE** | **EUE** | **FF** |
| **EOE** (Mean = 1.40) | 0.68* | 0.25* | 1 | 0.35* | -0.32* | -0.23* | 0.26* | -0.7 |
| **EUE** (Mean = 2.85) | -0.03 | -0.23* | 0.26* | 0.08 | 0.35* | 0.26* | 1 | 0.27* |

Abbreviations: EOE = Emotional overeating; EUE = Emotional under-eating; FR = Food responsiveness; EF = Enjoyment of food; DD = Desire to drink; SR = Satiety responsiveness, FF = Food Fussiness

# Appendices Chapter 3

## Appendix 2.1 Questions relating to zygosity in the baseline questionnaire, adapted from Price et al (2000)

| | | | | | |
|---|---|---|---|---|---|
| The next few questions are all about whether your twins are identical or non-identical. This section needs to be completed only if you have same sex twins (please note: non-identical twins are often called fraternal twins) | | | | | |
| **A1.** Have you ever been told by a health professional (e.g. doctor, nurse, consultant) that your twins are identical or non-identical? | | | | | |
| Yes, identical | ☐ | Yes, non-identical | ☐ | No | ☐ |
| **If YES, why did they think this?**_____ | | | | | |
| _____ | | | | | |
| **A2.** Do you think your twins are identical or non- identical? | | | | | |
| Identical | ☐ | Non-identical | ☐ | | |
| **Why do you think this is?** _____ _____ | | | | | |
| **A3.** As your twins have grown older, has the likeness between them: | | | | | |
| Become less | ☐ | Remained the same | ☐ | Become more | ☐ |
| **A4.** When looking at the twins: | | | | | |

| | None | | Only slight difference | Clear difference | |
|---|---|---|---|---|---|
| Are there differences in the shade of your twins' hair? | | ☐ | ☐ | | ☐ |
| Are there differences in the texture of your twins' hair (fine or coarse, straight or curly etc)? | | ☐ | ☐ | | ☐ |
| Are there differences in the colour of your twins' eyes? | | ☐ | ☐ | | ☐ |

| | | | | | | |
|---|---|---|---|---|---|---|
| Are there differences in the shape of your twins' ear lobes? | | ☐ | ☐ | | | ☐ |
| **A5.** Have either of your twins' teeth begun to come through? | Yes | ☐ | | No | ☐ | |
| If yes, was it at about the same time? | | | | | | |
| Yes, the twins had matching teeth on the same side come through within a few days of each other | | | | | ☐ | |
| Yes, the twins had matching teeth on opposite sides come through within a few days of each other | | | | | ☐ | |
| Yes, the twins had different teeth come through within a few days of each other | | | | | ☐ | |
| No, the twins' first teeth did not come through within a few days of each other | | | | | ☐ | |

**A6.** Do you know your twins' ABO blood group and Rhesus (Rh) factors?

| | | |
|---|---|---|
| Yes ☐ | No ☐ | |

**If YES, what are they?** (please tick a blood group and rhesus factor for each twin)

| | Blood group: | | | | Rhesus factor: | |
|---|---|---|---|---|---|---|
| | A | B | AB | O | Rh+ | Rh- |
| 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | | | | | |

**A7.** When looking at a new photograph of your twins, can you tell them apart (without looking at their clothes or using any other clues)?

| Yes, easily | Yes, but it is hard sometimes | No, I often confuse them in photographs |
|---|---|---|
| ☐ | ☐ | ☐ |

**A8.** Do any of the following people ever mistake your twins for each other?

| | Yes, often | Yes, sometimes | Rarely or never | Not applicable |
|---|---|---|---|---|
| Your partner / husband | ☐ | ☐ | ☐ | ☐ |
| Older brothers or sisters | ☐ | ☐ | ☐ | ☐ |
| Other relatives | ☐ | ☐ | ☐ | ☐ |
| Babysitter or day carer | ☐ | ☐ | ☐ | ☐ |
| Close friends | ☐ | ☐ | ☐ | ☐ |
| Casual friends | ☐ | ☐ | ☐ | ☐ |
| People meeting the twins for the first time | ☐ | ☐ | ☐ | ☐ |

**A9.** If the twins are ever mistaken for one another, does this ever happen when they are together?

| Yes, often | Yes, sometimes | No, almost never | They are not mistaken for one another |
|---|---|---|---|
| ☐ | ☐ | ☐ | ☐ |

**A10.** Would you say that your twins:

| | |
|---|---|
| Are as physically alike as “two peas in a pod” (virtually the same) | ☐ |
| Are as physically alike as brothers and sisters are | ☐ |
| Do not look very much alike at all | ☐ |
| | |

## Appendix 2.2 The Child Eating Behaviour Questionnaire

**Child Eating Behaviour Questionnaire (CEBQ)**

**Please read the following statements and tick the boxes most appropriate to your child's eating behaviour.**

| | Never | Rarely | Some-times | Often | Always | |
|---|---|---|---|---|---|---|
| **My child loves food** | □ | □ | □ | □ | □ | EF |
| **My child eats more when worried** | □ | □ | □ | □ | □ | EOE |
| **My child has a big appetite** | □ | □ | □ | □ | □ | SR* |
| **My child finishes his/her meal quickly** | □ | □ | □ | □ | □ | SE* |
| **My child is interested in food** | □ | □ | □ | □ | □ | EF |
| **My child is always asking for a drink** | □ | □ | □ | □ | □ | DD |
| **My child refuses new foods at first** | □ | □ | □ | □ | □ | FF |
| **My child eats slowly** | □ | □ | □ | □ | □ | SE |
| **My child eats less when angry** | □ | □ | □ | □ | □ | EUE |
| **My child enjoys tasting new foods** | □ | □ | □ | □ | □ | FF* |
| **My child eats less when s/he is tired** | □ | □ | □ | □ | □ | EUE |
| **My child is always asking for food** | □ | □ | □ | □ | □ | FR |
| **My child eats more when annoyed** | □ | □ | □ | □ | □ | EOE |
| **If allowed to, my child would eat too much** | □ | □ | □ | □ | □ | FR |
| **My child eats more when anxious** | □ | □ | □ | □ | □ | EOE |

| | | | | | | |
|---|---|---|---|---|---|---|
| **My child enjoys a wide variety of foods** | □ | □ | □ | □ | □ | FF* |
| **My child leaves food on his/her plate at the end of a meal** | □ | □ | □ | □ | □ | SR |
| **My child takes more than 30 minutes to finish a meal** | □ | □ | □ | □ | □ | SE |
| **Given the choice, my child would eat most of the time** | □ | □ | □ | □ | □ | FR |
| **My child looks forward to mealtimes** | □ | □ | □ | □ | □ | EF |
| **My child gets full before his/her meal is finished** | □ | □ | □ | □ | □ | SR |
| **My child enjoys eating** | □ | □ | □ | □ | □ | EF |
| **My child eats more when she is happy** | □ | □ | □ | □ | □ | EUE |
| **My child is difficult to please with meals** | □ | □ | □ | □ | □ | FF |
| **My child eats less when upset** | □ | □ | □ | □ | □ | EUE |
| **My child gets full up easily** | □ | □ | □ | □ | □ | SR |
| **My child eats more when s/he has nothing else to do** | □ | □ | □ | □ | □ | EOE |
| **Even if my child is full up s/he finds room to eat his/her favourite food** | □ | □ | □ | □ | □ | FR |
| **If given the chance, my child would drink continuously throughout the day** | □ | □ | □ | □ | □ | DD |
| **My child cannot eat a meal if s/he has had a snack just before** | □ | □ | □ | □ | □ | SR |
| **If given the chance, my child would always be having a drink** | □ | □ | □ | □ | □ | DD |
| **My child is interested in tasting food s/he hasn't tasted before** | □ | □ | □ | □ | □ | FF* |

| | | | | | | |
|---|---|---|---|---|---|---|
| **My child decides that s/he doesn't like a food, even without tasting it** | □ | □ | □ | □ | □ | FF |
| **If given the chance, my child would always have food in his/her mouth** | □ | □ | □ | □ | □ | FR |
| **My child eats more and more slowly during the course of a meal** | □ | □ | □ | □ | □ | SE |

Scoring of the CEBQ

(Never=1, Rarely=2, Sometimes=3, Often=4, Always=5)

Food responsiveness = item mean FR

Emotional over-eating = item mean EOE

Enjoyment of food = item mean EF

Desire to drink = item mean DD

Satiety responsiveness = item mean SR

Slowness in eating = item mean SE

Emotional under-eating = item mean EUE

Food fussiness = item mean FF

*Reversed items

## Appendix 2.3 Scripts for phone calls used in pilot study developing the Child Eating Behaviour Questionnaire – Toddler Version

PILOT 15 months questionnaire Gemini

Telephone interview

1. contact details of mother/father:
2. date of interview
3. interviewer's name: Clare, Rebecca, Ellen, other?
4. twins or singletons, other brother and sisters
5. DOB child/twins: and current age:
6. explain reason for interview

1) Would you prefer to call a 15 months year old: a baby or a child?

2) Explain what height chart is. If we would send you a height chart, would you use it and put it up on the wall and measure your child's height every month? What can we do to make this easier for you? What could make it easier for other people?

3) CEBQ – Emotional over / under-eating. More extensive pilot for D21-28

Could you describe situations where emotions of your baby influence their eating?

Which emotions:

- irritable / worried
- grumpy/annoyed
- anxious
- feeling bored / has nothing else to do

- sleepy / tired
- happy
- upset.

What are appropriate emotions, common in 15 months old babies?

| How would you describe your twins' eating styles at a typical day? |
|---|

| | | | Never | Rarely | Sometimes | Often | Always |
|---|---|---|---|---|---|---|---|
| **D21.** | **My baby eats more when irritable / worried (EOE) – PILOT** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ |
| **D22.** | **My baby eats more when grumpy / annoyed (EOE) – PILOT** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ |
| **D23.** | **My baby eats more when anxious (EOE) – PILOT** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ |
| **D24.** | **My baby eats more when he/she "has nothing else to do" Alternative wording? (EOE) – PILOT** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ |
| **D25.** | **My baby eats less** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | **when grumpy / angry (EUE) –PILOT** | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ |
| **D26.** | **My baby eats less when he/she is sleepy / tired (EUE) – PILOT** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ |
| **D27.** | **My baby eats more when he/she is happy (EUE) – PILOT** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ |
| **D28.** | **My baby eats less when upset (EUE) – PILOT** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ |

4) Food cue responsiveness / External eating: More extensive pilot for D37-40.

These questions explore situations in which children ask for food, without being hungry, i.e. outside the normal meal situation… Are these questions phrased appropriately for 15 month old babies?

How would you describe your twins' eating styles at a typical day?

| | | | **Never** | **Rarely** | **Sometimes** | **Often** | **Always** |
|---|---|---|---|---|---|---|---|
| **D37.** | **My baby eats more than usual if he/she really enjoys the taste of a food (EXT) - PILOT** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ |
| **D38.** | **My baby wants to eat (e.g. reaches out or cries for it,** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | **when he/she sees others eating (EXT) - PILOT** | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ |
| **D39.** | **My baby wants to eat (e.g. reaches out or cries for it, when I am in a supermarket or other food shop with him/her and he/she <u>smells</u> certain foods (EXT) -PILOT** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ |
| **D40.** | **My baby wants to eat (e.g. reaches out or cries for it, when I am in a supermarket or other food shop with him/her and he/she <u>sees</u> certain foods (EXT) -PILOT** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ |
| | | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ |

5) More extensive pilot for E6-14

We would like to get some idea about activity levels in 15 months old babies.

In which situation would you encourage your child to walk? For how long can they walk?

If you have to go somewhere close (15 minute walk for your baby) how would you go there?

a) Let baby walk the whole way
b) Let baby walk partly, and put in push chair for the other part
c) In push chair all the way
d) Use the care

How would you do this with two children? How is this affected by having more than one child?

Pilot following questions:

E1 / E2: When your babies were 15 months old, how different were the number of hours he / she watched TV/DVD on a day?

Is it important to divide questions in week day and weekend days or could they be combined in one question.

E3/ E4 / E5: When your babies were 15 months old, how different were sleeping patterns during a week?

Is it OK to combine this in one question or should there be separate questions for week-days and weekend days. How different are sleeping patterns during a week?

| | | | |
|---|---|---|---|
| The following questions are about how many hours your baby watch TV or DVD and how many hours they sleep. Please give estimates for the current situation, and add any comments on the back of the questionnaire if you want to tell us more about it. | | | |
| **E1.** | How many hours would you estimate your baby watches TV or DVD during the following times on a typical weekday (Monday through Friday) at this time of year? (PILOT if routine differs on days of the week or times of the day, what is easier to estimate) | | |
| | Morning (6 am to 12 noon) | Afternoon (12 noon to 6 pm) | Evening (6 pm to midnight) |
| 1st born | ___hrs ___ min per day | ___hrs ___ min per day | ___hrs ___ min per day |
| 2nd born | ___hrs ___ min per day | ___hrs ___ min per day | ___hrs ___ min per day |
| **E2.** | How many hours would you estimate your baby watches TV or DVD during the following times on a weekend day (Saturday or Sunday) at this time of year? (PILOT) | | |
| | Morning (6 am to 12 noon) | Afternoon (12 noon to 6 pm) | Evening (6 pm to midnight) |
| 1st born | ___hrs ___ min per day | ___hrs ___ min per day | ___hrs ___ min per day |
| 2nd born | ___hrs ___ min per day | ___hrs ___ min per day | ___hrs ___ min per day |
| **E3.** | When does your baby usually go to bed in the evening? | | |
| 1st born | ___ . ___ (please write hour.minutes: e.g. 6.15 pm or 18.15) | | |
| 2nd born | ___ . ___ | | |
| **E4.** | When does your baby usually wake up in the morning? | | |

| | | |
|---|---|---|
| | 1st born | ___ . ___ (please write hour.minutes: e.g. 6.15 pm or 18.15) |
| | 2nd born | ___ . ___ |
| **E5.** | How long does you baby usually sleep during daytime? | |
| | 1st born | ___ hours . ___ minutes per day |
| | 2nd born | ___ hours . ___ minutes per day |
| **E6.** | How often does your baby wake up at night and for how long? Write 0 if your baby usually never wakes up at night | |
| | 1st born | ___ times per night for: ___ hours . ___ minutes per night |
| | 2nd born | ___ times per night for: ___ hours . ___ minutes per night |

5) Food diary: we would like to ask mothers to fill in diaries of everything their child eat. Would you be able to do that, for 3 days. How could we make people enthusiastic? Would you be more likely to fill it out if you would tailored feedback on the nutrient content of the diet

6) Food diary: reasons for feeding

(see below)

To be send on paper

7) CEBQ (section D). Quick pilot for questions D1-20 and D29-36 does wording makes sense for 15 months old.
8) Section E: activity: quick pilot of E1-5: wording OK?

We're interested in finding out more about your child's mealtimes.

The diet dairy we use may potentially include extra columns for the mother to enter the time, location and people the twins are eating with along side the description of the actual food they are eating.

Having this information from a diary may mean we don't need the questions I have highlighted in bold in the table below.

We may also want to pilot adding a column to the diet diary that allows the mother to describe why the child ate or was fed at that time, what possible reasons might there be for feeding a child:

- My child was hungry
- My child asked/reached out/signalled for some food
- It was time to eat

What prompts you to give your baby food? In which situations other than that is time to eat (baby is hungry) would you give your baby something to eat or a snack?

When eating a meal, how often is you baby…… (time, when, with whom)

| | | 3 or more times a day | Twice a day | Once a day | 4-6 times a week | 2-3 times a week | Once a week | Never / Rarely | Not applicable |
|---|---|---|---|---|---|---|---|---|---|
| **…..eating with you or your partner?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..eating with another adult (e.g. relative, child minder)?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..eating with their older brothers or sisters?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..eating with other children?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..eating the same food as you?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..having the same drink as you?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..sat in a high chair?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..sat at a table?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| **…..sat in front of the TV?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..interacting with the family?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..watching TV?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..playing?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..wandering about?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..at nursery/childminders?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..in a café/restaurant?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| **…..outside of the home?** | 1st born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | 2nd born | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

## Appendix 2.4 The Child Eating Behaviour Questionnaire – Toddler Version

**CHILDREN'S EATING BEHAVIOUR QUESTIONNAIRE FOR TODDLERS (CEBQ-T)**

**How would you describe your child's eating styles on a typical day?**

| | Never | Rarely | Sometimes | Often | Always | |
|---|---|---|---|---|---|---|
| **1. My child loves food** | ☐ | ☐ | ☐ | ☐ | ☐ | EF |
| **2. My child eats more when irritable** | ☐ | ☐ | ☐ | ☐ | ☐ | EOE |
| **3. My child has a big appetite*** | ☐ | ☐ | ☐ | ☐ | ☐ | SR |
| **4. My child finishes his/her meal quickly*** | ☐ | ☐ | ☐ | ☐ | ☐ | SE |
| **5. My child is interested in food** | ☐ | ☐ | ☐ | ☐ | ☐ | EF |
| **6. My child cannot eat a meal if he/she has had a snack just before** | ☐ | ☐ | ☐ | ☐ | ☐ | SR |
| **7. My child refuses new foods at first** | ☐ | ☐ | ☐ | ☐ | ☐ | FF |
| **8. My child eats slowly** | ☐ | ☐ | ☐ | ☐ | ☐ | SE |
| **9. My child looks forward to mealtimes** | ☐ | ☐ | ☐ | ☐ | ☐ | EF |
| **10. My child is always asking for food** | ☐ | ☐ | ☐ | ☐ | ☐ | FR |
| **11. My child eats more when grumpy** | ☐ | ☐ | ☐ | ☐ | ☐ | EOE |
| **12. If allowed to, my child would eat too much** | ☐ | ☐ | ☐ | ☐ | ☐ | FR |
| **13. My child eats more when upset** | ☐ | ☐ | ☐ | ☐ | ☐ | EOE |
| **14. My child enjoys a wide variety of foods*** | ☐ | ☐ | ☐ | ☐ | ☐ | FF |
| **15. My child leaves food on his/her plate or in the jar at the end of a meal** | ☐ | ☐ | ☐ | ☐ | ☐ | SR |
| **16. My child takes more than 30 minutes to finish a meal** | ☐ | ☐ | ☐ | ☐ | ☐ | SE |

| | | | | | | |
|---|---|---|---|---|---|---|
| **17. Given the choice, my child would eat most of the time** | ☐ | ☐ | ☐ | ☐ | ☐ | FR |
| **18. My child enjoys tasting new foods*** | ☐ | ☐ | ☐ | ☐ | ☐ | FF |
| **19. My child gets full before his/her meal is finished** | ☐ | ☐ | ☐ | ☐ | ☐ | SR |
| **20. My child enjoys eating** | ☐ | ☐ | ☐ | ☐ | ☐ | EF |
| **21. My child is difficult to please with meals** | ☐ | ☐ | ☐ | ☐ | ☐ | FF |
| **22. My child decides that he/she does not like a food, even without tasting it** | ☐ | ☐ | ☐ | ☐ | ☐ | FF |
| **23. My child eats more and more slowly during the course of a meal** | ☐ | ☐ | ☐ | ☐ | ☐ | SE |
| **24. Even when my child has just eaten well, he/she is happy to eat again if offered** | ☐ | ☐ | ☐ | ☐ | ☐ | FR |
| **25. My child gets full up easily** | ☐ | ☐ | ☐ | ☐ | ☐ | SR |
| **26. My child is interested in tasting food he/she has not tasted before*** | ☐ | ☐ | ☐ | ☐ | ☐ | FF |

Scoring of the CEBQ-T

(Never=1, Rarely=2, Sometimes=3, Often=4, Always=5)

Food responsiveness = item mean FR

Emotional over-eating = item mean EOE

Enjoyment of food = item mean EF

Satiety responsiveness = item mean SR

Slowness in eating = item mean SE

Food fussiness = item mean FF

*Reversed items

## Appendix 2.5 The Baby Eating Behaviour Questionnaire

**BABY EATING BEHAVIOUR QUESTIONNAIRE (BEBQ)**

These questions are about your baby's appetite over his/her first few months of life. We are specifically interested in the period during which your baby is fed milk only, i.e. no solid foods or pre-prepared baby food yet.

**How would you describe your baby's feeding style at a <u>typical daytime feed</u>?**

| | Never | Rarely | Sometimes | Often | Always | |
|---|---|---|---|---|---|---|
| **1. My baby seems contented while feeding** | ☐ | ☐ | ☐ | ☐ | ☐ | EF |
| **2. My baby frequently wants more milk than I provide** | ☐ | ☐ | ☐ | ☐ | ☐ | FR |
| **3. My baby loves milk** | ☐ | ☐ | ☐ | ☐ | ☐ | EF |
| **4. My baby has a big appetite** | ☐ | ☐ | ☐ | ☐ | ☐ | GA |
| **5. My baby finishes feeding quickly*** | ☐ | ☐ | ☐ | ☐ | ☐ | SE |
| **6. My baby becomes distressed while feeding*** | ☐ | ☐ | ☐ | ☐ | ☐ | EF |
| **7. My baby gets full up easily** | ☐ | ☐ | ☐ | ☐ | ☐ | SR |
| **8. If allowed to, my baby would take too much milk** | ☐ | ☐ | ☐ | ☐ | ☐ | FR |
| **9. My baby takes more than 30 minutes to finish feeding** | ☐ | ☐ | ☐ | ☐ | ☐ | SE |
| **10. My baby gets full before taking all the milk I think he/she should have** | ☐ | ☐ | ☐ | ☐ | ☐ | SR |
| **11. My baby feeds slowly** | ☐ | ☐ | ☐ | ☐ | ☐ | SE |
| **12. Even when my baby has just eaten well he/she is happy to feed again if offered** | ☐ | ☐ | ☐ | ☐ | ☐ | FR |
| **13. My baby finds it difficult to manage a complete feed** | ☐ | ☐ | ☐ | ☐ | ☐ | SR |
| **14. My baby is always demanding a feed** | ☐ | ☐ | ☐ | ☐ | ☐ | FR |
| **15. My baby sucks more and more** | ☐ | ☐ | ☐ | ☐ | ☐ | SE |

| | | | | | | |
|---|---|---|---|---|---|---|
| **slowly during the course of a feed** | | | | | | |
| **16. If given the chance, my baby would always be feeding** | ☐ | ☐ | ☐ | ☐ | ☐ | FR |
| **17. My baby enjoys feeding time** | ☐ | ☐ | ☐ | ☐ | ☐ | EF |
| **18. My baby can easily take a feed within 30 minutes of the last one** | ☐ | ☐ | ☐ | ☐ | ☐ | FR |

SCORING OF THE BEBQ

(Never=1, Rarely=2, Sometimes=3, Often=4, Always=5)

Food responsiveness (FR) = item mean FR

Enjoyment of food (EF) = item mean EF

Satiety responsiveness (SR) = item mean SR

Slowness in eating (SE) = item mean SE

General appetite (GA) = single item that measures overall/ general appetite

*Reversed items (5 & 6)

# Appendix Chapter 4

## Appendix 3.1a Sex limitation model for EOE measured at 16 months, ACE estimates for males and females

| Model | Male | | | Female | | | | |
|---|---|---|---|---|---|---|---|---|
| | $A_m$ | $C_m$ | $E_m$ | $A_f$ | $C_f$ | $E_f$ | $r_A$ | $r_C$ |
| **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 ($r_A$=0.5, $r_C$=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** | | $r_A$ | $r_C$ |
| **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 |

Abbreviations: A = 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

## Appendix 3.1b Sex Limitation Model for EOE measured at five years, ACE estimates for males and females

| Model | Male | | | Female | | | | |
|---|---|---|---|---|---|---|---|---|
| | $A_m$ | $C_m$ | $E_m$ | $A_f$ | $C_f$ | $E_f$ | $r_A$ | $r_C$ |
| **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) | | 0.99 (0.97-1.00) | |
| | **A** | | **C** | | **E** | | $r_A$ | $r_C$ |
| **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 |

Abbreviations: A = 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

# Appendix Chapter 6

## Appendix 4.1 Skew and kurtosis of variables included in the analysis in Chapter 7 Study 3

| Variable | N | mean | SD | skew | kurtosis |
|---|---|---|---|---|---|
| **Age (years)** | 1168 | 5.15 | 0.13 | 1.64 | 4.05 |
| **Gestational age (weeks)** | 1168 | 36.21 | 2.49 | -1.38 | 2.32 |
| **5years BMI-SDS** | 1168 | -.246 | 1.03 | -0.84 | 4.72 |
| **Child emotional overeating** | 1168 | 2.65 | 0.84 | 0.05 | -0.58 |
| **Child emotional under-eating** | 1168 | 1.55 | 0.51 | 0.85 | 0.26 |
| **Parental emotional eating** | 1168 | 2.14 | 0.96 | 0.78 | 0.21 |
| **Parental restraint** | 1168 | 2.70 | 0.94 | 0.02 | -0.56 |
| **Parental external eating** | 1168 | 3.07 | 0.65 | 0.21 | 0.37 |
| **CHAOS** | 1168 | 0.39 | 0.33 | 0.56 | -0.91 |
| **Instrumental Feeding** | 1168 | 2.32 | 0.62 | 0.14 | -0.28 |
| **Emotional Feeding** | 1168 | 1.69 | 0.55 | 0.64 | -0.16 |
| **Pressure to Eat** | 1168 | 2.72 | 0.648 | -0.01 | -0.04 |
| **Control** | 1168 | 4.16 | 0.43 | -.031 | -0.41 |
| **Mealtime Structure** | 1168 | 4.06 | 0.57 | -0.28 | -0.42 |

Abbreviation: BMI-SDS = Body Mass Index Standard Deviation Score; CHAOS = The Confusion, Hubub and Order Scale;

# Appendix Published Papers

## Appendix 5.1 List of the papers that I have worked on during my PhD, and the conferences that I have presented at and attended

Published Papers:

**Herle M**, Fildes A, Rijsdijk F, Steinsbekk S and Llewellyn, CH (2017): Emotional over- and under-eating in early childhood are learned not inherited. *Scientific Reports*. doi:10.1038/s41598-017-09519-0

**Herle M**, Fildes A, Rijsdijk F, Steinsbekk S and Llewellyn, CH (2017): The home environment shapes emotional eating in childhood. *Child Development*. doi: 10.1111/cdev.12799

Smith AD, **Herle M**, Fildes A, Cooke L, Steinsbekk S, Llewellyn CH (2016). Food fussiness and food neophobia share a common etiology in early childhood. *Journal of Child Psychology and Psychiatry*. doi:10.1111/jcpp.12647

Smith AD, Fildes A, Cooke L, **Herle M**, Shakeshaft N, Plomin R, Llewellyn CH (2016). Genetic and environmental influences on food preferences in adolescence. *American Journal of Clinical Nutrition*. 104, 2016, 446-453.

**Herle, M**., A. Fildes, C. van Jaarsveld, F. Rijsdijk and C. H. Llewellyn (2016). Parental Reports of Infant and Child Eating Behaviors are not Affected by Their Beliefs About Their Twins' Zygosity. *Behavior Genetics*. doi: 10.1007/s10519-016-9798-y.

Papers in preparation:

**Herle M***, Kan C*, Jayaweera K, Adikari A, Siribaddana S, Zavos HMS, Llewellyn CH, Sumathipala A, Ismail K, Hotopf M, Treasure J and Rijsdijk F (2017). Emotional overeating and depression in the Sri Lankan Twin Register.* Joint first authors

Fildes A, **Herle M**, Cooke L, Van Jaarsveld CHM, Plomin R and Llewellyn CH (2017). It's all in the delivery: Genetic and environmental influences on responses to a behavioural dietary intervention in children.

**Herle M**, Fildes A and Llewellyn CH (2017). Low heritability of emotional over- and under-eating in childhood: A replication study in the Twins Early Development Study.

**Herle M**, Fildes A, Steinsbekk S and Llewellyn (2017). Emotional over- and under-eating are characterised by distinct parental feeding practices in early childhood.

Conference Presentations

**International Society of Twin Studies, Madrid, Spain (November 2017)** Household stress increases the heritability of emotional eating in childhood: A gene-environment interaction study.

**Association of the Study of Obesity, UK Congress, Pontypridd, Wales (September 2017)** Emotional over- and under-eating are characterised by distinct parental feeding practices in early childhood. *Oral presentation*

**47th Annual Meeting of the Behavior Genetics Association, Oslo, Norway (June 2017)** Emotional overeating and depression in the Sri Lankan Twin Register. *Oral presentation*

**Association of the Study of Obesity, UK Congress (September 2016)** Emotional over and under-eating are learned not inherited. *Oral presentation (winner of the best abstract award)*

**46th Annual Meeting of the Behavior Genetics Association, Brisbane, Australia (June 2016)** Parental reports of infant and child eating behaviors are not affected by their beliefs about their twins' zygosity. *Oral presentation*

**International Society for Twin Studies, Brisbane, Australia (June 2016)** Parental reports of infant and child eating behaviors are not affected by their beliefs about their twins' zygosity. *Oral presentation*

**Obesity Week 2015, Los Angeles, USA, (November 2015)** The environment shapes emotional eating in childhood. *Poster*

**Psychology postgraduate affairs group (Psypag) conference, Cardiff, UK (2014).** Therapygenetics: The Serotonin Transporter Promoter polymorphism and response to Exposure-based Cognitive Behaviour Therapy in Adult Anxiety. *Oral presentation*

## Appendix 5.2 Published Paper relating to Chapter 4

Child Development, xxxx 2017, Volume 00, Number 0, Pages 1–12

# The Home Environment Shapes Emotional Eating

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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 2,402 British twins born in 2007. EOE was measured using the "emotional overeating" scale of the Child Eating Behavior Questionnaire (CEBQ) 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.

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 ($r$ = .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, 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, appetites of individuals with obesity are abnormal in not being affected by stress. The theory still predicts that individuals with obesity 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–*

We thank the Gemini families who are participating in the study and the Office for National Statistics for their help in recruiting them. The authors would like to acknowledge the substantial intellectual contribution by Professor Jane Wardle who sadly passed away prior to publication.

Correspondence concerning this article should be addressed to Clare Llewellyn, Department of Behavioural Science and Health, University College London, Gower Street, London WC1E 6BT, United Kingdom. Electronic mail may be sent to clare.llewellyn@ucl.ac.uk.

 0009-3920/2017/xxxx-xxxx
DOI: 10.1111/cdev.12799

## Appendix 5.3 Published Paper relating to Chapter 5

www.nature.com/scientificreports

OPEN

# Emotional over- and under-eating in early childhood are learned not inherited

Received: 10 April 2017
Accepted: 27 July 2017
Published online: 23 August 2017

Moritz Herle[1], Alison Fildes[1,2], Silje Steinsbekk[3], Fruhling Rijsdijk[4] & Clare H. Llewellyn[1]

**Emotional overeating (EOE) has been associated with increased obesity risk, while emotional undereating (EUE) may be protective. Interestingly, EOE and EUE tend to correlate positively, but it is unclear whether they reflect different aspects of the same underlying trait, or are distinct behaviours with different aetiologies. Data were from 2054 five-year-old children from the Gemini twin birth cohort, including parental ratings of child EOE and EUE using the Child Eating Behaviour Questionnaire. Genetic and environmental influences on variation and covariation in EUE and EOE were established using a bivariate Twin Model. Variation in both behaviours was largely explained by aspects of the environment completely shared by twin pairs (EOE: C = 90%, 95% CI: 89%-92%; EUE: C = 91%, 95% CI: 90%-92%). Genetic influence was low (EOE: A = 7%, 95% CI: 6%-9%; EUE: A = 7%, 95% CI: 6%-9%). EOE and EUE correlated positively ($r = 0.43$, $p < 0.001$), and this association was explained by common shared environmental influences (BivC = 45%, 95% CI: 40%-50%). Many of the shared environmental influences underlying EUE and EOE were the same ($r_c = 0.50$, 95% CI: 0.44, 0.55). Childhood EOE and EUE are etiologically distinct. The tendency to eat more or less in response to emotion is learned rather than inherited.**

Emotional eating is the tendency to change one's eating behaviour in response to negative emotions[1]. Research with adults has shown that some people tend to consume *more* in stressful situations (so-called emotional *over-eating*, [EOE]), whereas others experience a decrease in appetite when distressed and eat *less* (so-called emotional *under-eating*, [EUE])[1,2]. The tendency to either over- or under-eat in response to negative emotion appears to emerge in the preschool years[3].

Understanding the aetiologies of these behaviours in early life is important because EOE has been hypothesized to play a causal role in overweight, and EUE in under-weight[4]. There has been some support for these hypotheses insofar as EOE has been associated with higher weight cross-sectionally[5–10], and with weight gain longitudinally from 5–6 years to 6–8 years[11] and from 4 to 8 years[12]. On the other hand, EUE has been negatively associated with weight[9,13,14]. However, null findings have sometimes been reported in cross-sectional studies for both EOE[15,16] and EUE[9,15].

Despite their differing associations with weight, EOE and EUE tend to be positively correlated[9,17,18], indicating that some children have an underlying tendency to *both* under- *and* overeat in response to negative emotions. This raises the question as to whether these two behaviours reflect different aspects of the same underlying trait (i.e. a tendency to *both* over- *and* under-eat in response to negative emotion) with a common aetiology, or are distinct traits with different aetiologies. Twin studies offer a powerful method for establishing the extent to which behaviour is shaped by genes and environments; and can also elucidate shared aetiology by quantifying the extent to which different behaviours share common or distinct genetic and environmental influences. We have conducted the only paediatric twin study of EOE, finding this behaviour to be influenced largely by aspects of the environment completely shared by twin pairs, in both toddlerhood and early childhood; with genetic factors playing a minimal role[19]. To date there have been no twin studies of EUE in adults or children. The objectives of this study were to use a twin design to: (i) establish for the first time the relative genetic and environmental contributions to EUE in early childhood, and (ii) establish the extent to which EOE and EUE share a common genetic and environmental aetiology.

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## Appendix 5.4 Published Paper relating to Chapter 10

Behav Genet (2016) 46:763–771
DOI 10.1007/s10519-016-9798-y

ORIGINAL RESEARCH

# Parental Reports of Infant and Child Eating Behaviors are not Affected by Their Beliefs About Their Twins' Zygosity

Moritz Herle[1] · Alison Fildes[1] · Cornelia van Jaarsveld[2] · Fruhling Rijsdijk[3] · Clare H. Llewellyn[1]

Received: 29 January 2016 / Accepted: 15 June 2016 / Published online: 12 July 2016


**Abstract** Parental perception of zygosity might bias heritability estimates derived from parent rated twin data. This is the first study to examine if similarities in parental reports of their young twins' behavior were biased by beliefs about their zygosity. Data were from Gemini, a British birth cohort of 2402 twins born in 2007. Zygosity was assessed twice, using both DNA and a validated parent report questionnaire at 8 (SD = 2.1) and 29 months (SD = 3.3). 220/731 (8 months) and 119/453 (29 months) monozygotic (MZ) pairs were misclassified as dizygotic (DZ) by parents; whereas only 6/797 (8 months) and 2/445 (29 months) DZ pairs were misclassified as MZ. Intraclass correlations for parent reported eating behaviors (four measured at 8 months; five at 16 months) were of the same magnitude for correctly classified and misclassified MZ pairs, suggesting that parental zygosity perception does not influence reporting on eating behaviors of their young twins.

**Keywords** Parental bias · Twin research · Child development · Misclassified zygosity · Eating behaviors · Heritability

Edited by Yoon-Mi Hur

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## Introduction

Over the past century the Twin Method has been used to investigate genetic and environmental contributions to variation in complex human traits. Researchers have been using this methodology to examine a wide spectrum of aspects of human life accumulating in a total of 17,804 investigated traits, spanning disease, to behavior to opinion. Twin research is conducted worldwide and 14,558,903 twins are currently included in a multitude of studies (Polderman, et al. 2015).

The classic Twin Method is based on comparing the correlations or concordance rates of traits between monozygotic (MZ) and dizygotic (DZ) twin pairs. MZs are genetic clones of one another, sharing 100 % of their genes, whereas DZs share on average only 50 % of their segregating genes. Importantly, both types of twins share their environments to a similar extent. For example, both types of twins are gestated together in the same uterus, and are raised together in one family. Any difference in resemblance between MZ and DZ pairs is therefore assumed to reflect genetic differences only. The univariate method can also be extended to understand if multiple traits share a common etiology, and to establish genetic and environmental contributions to trait stability and change over time (Rijsdijk and Sham 2002; van Dongen et al. 2012).

One of the criticisms of parent reported measures of young twin behavior is that parents are biased by their belief about their twins' zygosity. For example, it is