



Epidemiological studies of  
eating problems in the family

Lisanne M. de Barse

# “I Don’t Want to Eat that”

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# “I Don’t Want to Eat that”

Epidemiological studies of  
eating problems in the family

# “Ik wil dat niet eten”

Epidemiologische onderzoeken  
naar eetproblemen in het gezin

## **Proefschrift**

ter verkrijging van de graad van doctor aan de  
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## **Promotiecommissie**

Promotoren:

Prof.dr. H. Tiemeier

Prof.dr. O.H. Franco

Overige leden:

Prof.dr. F.C. Verhulst

Prof.dr. H. Raat

Dr. N. Micali

Copromotor:

Dr. P.W. Jansen

**Paranimfen:**

Charlotte L. Ars

Danique A. de Barse

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# Manuscripts that form the basis of this thesis

## Chapter 2

Nguyen AN, **De Barse LM**, Tiemeier H, Jaddoe VWV, Franco OH, Jansen PW, Voortman T. Maternal history of eating disorders: Diet quality during pregnancy and infant feeding.

*Submitted for publication*

**De Barse LM**, Tharner A, Micali N, Jaddoe VWV, Hofman A, Verhulst FC, Franco OH, Tiemeier H, Jansen PW. Does maternal history of eating disorders predict mothers' feeding practices and preschoolers' emotional eating?

*Appetite, 2014, 84, 1-7*

**De Barse LM**, Tiemeier H, Micali N, Jaddoe VWV, Verhulst FC, Steenweg-de Graaff J, Franco OH, Jansen PW. Children of mothers with a history of eating disorders: Are they at risk of emotional and behavior problems?

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## Chapter 3

**De Barse LM**, Jansen PW, Edelson-Fries LR, Jaddoe VWV, Franco OH, Tiemeier H, Steenweg-de Graaff J. Infant feeding and child fussy eating: The Generation R Study.

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**De Barse LM**, Cardona Cano S, Jansen PW, Jaddoe VWV, Verhulst FC, Franco OH, Tiemeier H, Tharner A. Are parents' anxiety and depression related to child fussy eating?

*Archives of Disease in Childhood, 2015, 101, 533-538*

Cardona Cano S, Hoek HW, Van Hoeken D, **De Barse LM**, Jaddoe VWV, Verhulst FC, Tiemeier H. Behavioral outcomes of picky eating in childhood: A prospective study in the general population.

*Journal of Child Psychology and Psychiatry, 2016 (in press)*

**De Barse LM**, Tiemeier H, Leermakers ETM, Voortman T, Jaddoe VWV, Edelson LR, Franco OH, Jansen PW. Longitudinal association between preschool fussy eating and body composition at 6 years: The Generation R Study.

*International Journal of Behavioral Nutrition and Physical Activity, 2015, 12, 153*









## Background

Eating disorders are severe mental illnesses with multiple health consequences,<sup>1,2</sup> including death.<sup>3,4</sup> Patients with eating disorders – often women – have disturbed eating behaviors, an undue preoccupation with body weight and shape, extreme concerns about their body, and this problematic body image strongly determines how they evaluate themselves.<sup>1</sup> Eating disorders often have a chronic nature<sup>4,5</sup> and do not only impact on women themselves, but possibly also their offspring. Children of mothers with past eating disorders are at risk of developing an eating disorder themselves during adolescence.<sup>6</sup> For the prevention of eating disorders, it is key to identify whether possible precursors of eating disorders become already apparent early in life.

In childhood, the foundations for life-long eating habits are laid and sometimes persisting problems arise. Picky eating is a common eating problem in young children,<sup>7</sup> but a relatively unexplored study area. Picky eating – also called ‘fussy’, ‘choosy’, or ‘selective’ eating – can be a burden for family dinners and is worrisome for parents,<sup>8</sup> who often seek medical advice for their child’s eating behavior. Picky eaters are characterized by a restricted dietary variety due to their consistent rejection of foods, often including vegetables.<sup>7</sup> Limited intake of vegetables can interfere with a healthy diet and related health benefits. In addition, picky eaters may be at risk of weight problems. In this thesis, we studied children’s picky eating as well as children of mothers with a history of eating disorders.

## The intergenerational transmission of eating psychopathology

It has been hypothesized that children of mothers with eating disorders are at risk of developing eating disorders themselves during adolescence.<sup>9</sup> Recently, this has been further supported by showing a temporal association between parental eating disorders and offspring’s incidence of eating disorders in a large Swedish cohort.<sup>6</sup> It could be expected that the foundation of this intergenerational transmission of eating psychopathology is laid early in life. Children of mothers with a history of eating disorders may therefore already from a young age be at risk of disturbances in their development.

Although clinical case reports – often not including a control group – have reported increased eating problems and emotional problems in children of mothers with an active eating disorder,<sup>10</sup> only limited population-based research has been

performed on the development of children whose mothers suffered from an eating disorder in the past. These studies reported higher emotional problems in children of mothers with a history of eating disorders,<sup>11-14</sup> but conflicting results regarding behavior problems in these children.<sup>11,13</sup> Moreover, even less is known about emotional eating in children of mothers with an eating disorder history. Multiple mechanisms could underlie an intergenerational transmission of distorted eating behaviors including genetic factors<sup>15</sup> and modifiable factors<sup>6,9</sup> such as maternal modelling.<sup>16</sup> Enduring eating psychopathology may be present in mothers with a history of eating disorders, given that eating disorders are difficult to treat.<sup>4,5</sup> If mothers engage in disordered eating behaviors in the presence of their children, their children might imitate these behaviors. Critical comments of mothers may even be more important than modelling<sup>17</sup> in the transmission of eating psychopathology. For instance, children might learn that how they look like is extremely important for how they should evaluate themselves if their mothers express their concerns about children's weight and shape. These different mechanisms could account for higher cognitive vulnerabilities for eating disorders in these children, but also for emotional disorders given the high co-occurrence of eating disorders and anxiety or depression.<sup>18,19</sup> These cognitive vulnerabilities for eating and emotional disorders might be expressed by altered body image, dieting behavior, weight issues, and emotional problems. Therefore, we aimed to study the association between maternal history of eating disorders and their offspring's emotional eating tendencies, body mass index (BMI), and emotional and behavior problems. In this thesis, we focused on young children, as it is important to study whether these problems emerge in early childhood to develop targeted prevention programs.

Children of mothers with a history of eating disorders might not only be at risk of disturbances in their development, but may also be more exposed to controlling feeding practices, which may have adverse effects.<sup>20</sup> Controlling feeding practices, such as restricting children's food intake or pressure to eat, might be more prevalent in mothers with eating disorders given their urge to control their own food intake.<sup>17</sup> Also, a mothers' desire for control may impact breastfeeding practices.<sup>21</sup> Difficulties with breastfeeding among women with a history of eating disorders could also occur because of enduring shape concerns and body awareness causing shame or embarrassment of breastfeeding.<sup>22</sup> Alternatively, the common belief that breastfeeding promotes weight loss may increase breastfeeding initiation and duration in women with an eating disorder history.<sup>23</sup> The evidence for different feeding practices in mothers with a history of eating disorders, however, is still inconclusive and many studies relied on small sample sizes.<sup>24-26</sup> Therefore, within a large population-based study in the Netherlands – the Generation R Study – we aimed to study the association between maternal history of eating disorders and feeding practices.

## Picky eating

In this thesis, picky eating refers to the consistent rejection of certain familiar (and non-familiar) foods resulting in an inadequate dietary variety, as this is included in most definitions.<sup>7</sup> Some definitions extend this to an inadequate *amount* of food intake, but being selective in what food to eat does not necessarily imply an overall low energy intake and picky eaters may even compensate their limited intake of disliked foods by eating more palatable, energy-dense foods.<sup>27</sup> Food refusals are often based on the sensory profiles of food, as children avoid foods because of their smell and taste, but also because of their novelty (food neophobia).<sup>7</sup> Importantly, food neophobia and picky eating are theoretically not the same concepts, although highly related in practice. Food neophobia refers to the rejection of new food items and this definition does not include rejection of familiar food items. Picky eaters may theoretically refuse familiar food items only, and accept new foods, but in practice, picky eaters often refuse new food items as well. Researchers therefore often include food neophobia in their measurement of picky eating.

Different measurements and definitions of picky eating may have contributed to the wide range of reported prevalence estimates from 14% to even 50%,<sup>28-30</sup> although this can partly be attributed to age differences. The prevalence of picky eating is considered to peak during the preschool-aged years, and usually declines afterwards, although still high in school-aged children. The high prevalence could also indicate that picky eating is a phase of normal development.<sup>30,31</sup> Indeed, it has been observed that most picky eaters within the Generation R Study – a cohort study on which this thesis is also based – remitted within three years.<sup>30</sup> However, some children (4%) persisted from age 1.5 to 6 years in their picky eating behavior, measured by two items ('does not eat well' and 'refuses to eat') of the Child Behavior Checklist (CBCL).<sup>32,33</sup> These children are chronic picky eaters and may therefore be at risk of adverse health outcomes, even as severe picky eaters. Using multiple scales of the Children's Eating Behaviour Questionnaire (CEBQ),<sup>34</sup> a picky eating profile has been described in Generation R, with only 5% of the children assigned as picky eaters.<sup>27</sup> This distinct group may indicate severe picky eating. Notably, this picky eating profile was not only characterized by high scores on the food fussiness scale – a commonly used approach to measure differences in picky eating – but also by high scores on other avoidant eating behaviors such as eating slowly and being strongly responsive to satiety cues (e.g. getting full easily), and by low food approach behaviors such as low enjoyment of food. When picky eating is not a phase of normal development, it may be explained as rigid behavior, which is characteristic for children with autism spectrum disorders (ASD).<sup>1</sup> Thus, severe picky eating could be a marker for other psychopathology. Severe picky eaters may also be at risk of nutri-

tional deficiencies and a low energy intake with consequent underweight, or potentially overweight if picky eaters eat more tasty energy-dense food to compensate for their food rejections.

Although it would be important to study risk factors for severe picky eating, at first it is needed to study risk factors for picky eating behavior in general, as the etiology of picky eating is not well understood. Genetic factors have been identified, but from a preventive medicine perspective, it is important to study modifiable factors such as timing of complementary feeding and breastfeeding duration. For instance, through flavor exposure of breastmilk,<sup>35</sup> a longer duration of breastfeeding may contribute to lower levels of picky eating. Another risk factor for picky eating could be parents' symptoms of anxiety and depression. It is well known that maternal emotional symptoms have been related to problematic child development,<sup>36</sup> including difficulties in eating behavior,<sup>37,38</sup> and possibly also picky eating. However, previous studies focused on maternal symptoms during the child's life and may have been prone to reverse causation, as the child's picky eating could have impacted on the mother's stress and wellbeing. Therefore, we also aimed to study the association between parents' emotional problems during pregnancy and children's picky eating.

## This thesis

### Main objectives

The first objective of this thesis was to identify early signs of the intergenerational transmission of eating disorders. Specifically, we aimed to study whether young children of mothers with a history of eating disorders were at risk of emotional eating, weight problems, and emotional problems. In addition, we aimed to study feeding practices in mothers with a history of eating disorders.

The second objective of this thesis was to obtain insight in modifiable risk factors for picky eating behavior, such as breastfeeding duration, timing of complementary feeding, and parental anxiety or depressive symptoms. The third aim was to study possible health consequences of more severe or chronic picky eating.

### The Generation R Study

This thesis was embedded within the Generation R Study, a population-based prospective cohort from fetal life onwards in Rotterdam, the Netherlands.<sup>39,40</sup> Generation R was designed to identify early determinants of children's growth, develop-

ment, and health. Pregnant women living in Rotterdam, the second largest city of the Netherlands, with expected delivery data between April 2002 and January 2006 were invited to participate (baseline response rate: 61%). The Generation R Study has been conducted in accordance with Declaration of Helsinki and has been approved by the Medical Ethical Committee at the Erasmus MC, University Medical Center, Rotterdam.

During pregnancy, assessments included physical examinations of the women (e.g. measuring weight and height). Self-administered questionnaires were used to obtain information about the sociodemographic characteristics and health of the participants,<sup>39,40</sup> for instance about parents' mental health during pregnancy.<sup>41</sup> Ascertainment of history of eating disorders and other psychiatric disorders was based on self-report (see chapter 2 for a detailed description) as clinical diagnoses were not feasible within this large cohort.

During the first years, most information on child health and development was obtained by questionnaires.<sup>39,40</sup> We used the food fussiness scale of the CEBQ for etiologic research questions.<sup>34</sup> To identify whether chronic or severe picky eating was related to health outcomes, we used the CBCL picky eating trajectories (never picky eating, remitting picky eating, late-onset picky eating, and persistent picky eating),<sup>30</sup> and the CEBQ-derived picky eating profile.<sup>27</sup>

When the children reached the age of 5 years, we invited them to our dedicated research center in the Sophia's Children Hospital.<sup>39,40</sup> Multiple psychological and physical examinations were performed, including a body scan to measure body fat and fat-free mass. At this age, we also mailed the mothers and teachers a questionnaire about the children's emotional and behavior problems.<sup>32,33</sup>

## Thesis outline

In **chapter 2**, mothers with a history of eating disorders and their children were studied. The associations of maternal history of eating disorders with diet quality of pregnant women and their infants, and with breastfeeding practices is described in *chapter 2.1*. Moreover, we studied whether the children of mothers with a history of eating disorders were at risk of emotional eating behavior in toddlerhood, weight problems, (*chapter 2.2*) and emotional or behavior problems in early childhood (*chapter 2.3*).

In **chapter 3**, potential modifiable risk factors of picky eating were studied, such as infant feeding practices (i.e. breastfeeding and timing of complementary feeding, *chapter 3.1*) and parental anxiety or depression (*chapter 3.2*). The association of picky eating with mental health problems is included in *chapter 3.3*. *Chapter 3.4* describes the longitudinal relation between picky eating and body composition.



**Chapter 4** gives an overview of the main findings and conclusions from all studies described in this thesis. It also includes a discussion about the major strengths and limitations of this thesis, clinical implications, and recommendations for future studies.

Finally, a summary of this thesis is provided in **chapter 5**. Appendixes include a PhD portfolio, information about the author, and acknowledgments.

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CHAPTER 2.1

## Maternal history of eating disorders:

Diet quality during pregnancy and infant feeding

### **Manuscript based on this chapter:**

Anh N. Nguyen, Lisanne M. de Barse, Henning Tiemeier, Vincent W.V. Jaddoe, Oscar H. Franco, Pauline W. Jansen, Trudy Voortman. Maternal history of eating disorders: diet quality during pregnancy and infant feeding. *Submitted for publication*





## CHAPTER 2.2

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# Maternal history of eating disorders:

Feeding practices and preschoolers' emotional eating

### **Manuscript based on this chapter:**

Lisanne M. de Barse, Anne Tharner, Nadia Micali, Vincent W.V. Jaddoe, Albert Hofman, Frank C. Verhulst, Oscar H. Franco, Henning Tiemeier, Pauline W. Jansen. Does maternal history of eating disorders predict mothers' feeding practices and preschoolers' emotional eating? *Appetite*, 2014, 84, 1-7

## Abstract

**Background:** We aimed to examine whether a maternal history of eating disorders predicted mothers' feeding practices and preschoolers' emotional eating patterns.

**Methods:** Data were available from 4,851 mothers and their children, who participated in a Dutch population-based cohort study (the Generation R Study). Maternal history of lifetime eating disorders was assessed during pregnancy using a self-report questionnaire. Mothers filled out the Child Feeding Questionnaire and the Child Eating Behaviour Questionnaire when children were 4 years old. Linear regression analyses were performed, adjusting for potential confounders.

**Results:** Of all mothers, 8.6% had a history of an eating disorder (2.5% anorexia nervosa (AN); 3.9% bulimia nervosa (BN); 2.2% both AN and BN). Compared to mothers without a history of eating disorders, mothers with a history of eating disorders, in particular AN, used less pressuring feeding strategies (standardized  $B = -0.30$ ; 95% CI:  $-0.49, -0.11$ ). Children of mothers with a history of AN had relatively high levels of emotional overeating (standardized  $B = 0.19$ ; 95% CI:  $0.00, 0.39$ ). Maternal history of BN was not related to mothers' feeding practices or children's emotional eating.

**Conclusion:** Overall, the levels of emotional overeating amongst children of mothers with a history of eating disorders are noteworthy, particularly considering the young age (4 years) of participating children. This finding may reflect an effect of maternal eating disorders on the development of disordered eating patterns, but could also be subject to mothers' perception.

## Introduction

Eating disorders are chronic mental illnesses, with a lifetime prevalence amongst women of about 2% for anorexia nervosa (AN) and about 3% for bulimia nervosa (BN)<sup>1</sup> and evidence for increasing incidence.<sup>2</sup> AN and BN are associated with an increased risk of psychopathology, medical problems, and premature mortality.<sup>1,3,4</sup> Furthermore, there is evidence for an intergenerational transmission of eating disorders, such that offspring of women with eating disorders have a higher risk of developing eating disorders themselves.<sup>5</sup>

Women with eating disorders suffer from disturbances in eating patterns. AN is characterized by pathological fears of becoming fat, while in fact the body weight is abnormally low.<sup>6,7</sup> A distorted body image (i.e. perception of being overweight), denial of having a dangerously low weight and an enduring desire to lose weight are core to AN. A distinction is made between the binge eating/purging and restrictive type of AN, differentiating those who engage in subjective binge eating and compensating purging behavior (e.g. self-induced vomiting and misuse of diuretics, laxatives, or enemas) and those who accomplish weight loss mainly by restricting their diet. Similar to the binge eating/purging type of AN, BN is also characterized by recurrent episodes of binge eating followed by compensating behaviors to prevent weight gain. However, as opposed to the subjective binges occurring in AN which in fact contain very few calories, binge eating in individuals with BN refers to eating a disproportional large amount of food, accompanied with experiencing loss of control over eating. Compensating behaviors include purging behaviors (purging type), or fasting and exercising (non-purging type). Like BN, binge eating disorder (BED) is also characterized by the recurrence of binge eating episodes accompanied by feelings of guilt, embarrassment and disgust.<sup>7</sup> However, in BED, these binges are not followed by compensating behaviors.

Women with eating disorders often have difficulties in emotional processing<sup>4</sup> and may experience comorbid mood disorders as well,<sup>8</sup> partially explained by common familial factors which may cause both eating disorders and mood disorders.<sup>9</sup> It has been suggested that emotional eating patterns index vulnerability for eating disorder psychopathology<sup>10</sup> and can be present in both AN and BN.<sup>11,12</sup> Emotional overeating, usually referred to as emotional eating, is the tendency to eat in response to negative emotions such as anxiety, anger, frustration, or depression.<sup>10,13</sup> Emotional undereating is the tendency to lose appetite and eat less when upset.<sup>13</sup> In women with BN, this is mostly reflected by the occurrence of binges in response to emotional distress.<sup>11</sup> Women with AN are likely to eat less in response to emotions,<sup>12</sup> although there is also evidence for elevated levels of emotional overeating.<sup>11</sup>



Considering the evidence for an intergenerational transmission of eating disorders,<sup>5</sup> it is important to enhance knowledge about the underlying mechanisms. One of these mechanisms could involve the transmission of pathological eating behaviors, such as emotional eating. Indeed, in children aged between 8 and 10 years, dietary restraint and concerns about being too fat were more common among those who had mothers with eating disorders.<sup>14,15</sup> Furthermore, preschool aged children of mothers with a history of eating disorders experienced more eating problems such as extreme drinking or eating pace (very fast or extremely slow), frequent vomiting, and altered energy intake and weight development.<sup>16-19</sup> Evidence for patterns of emotional eating behavior among children of mothers with an eating disorder is scarce, but in a sample of healthy Dutch families with adolescent children (aged 13-16) a positive association was found between emotional eating patterns of mothers and their offspring.<sup>20</sup> Furthermore, a mother's use of food to soothe her child – a feeding pattern relatively often seen in mothers with eating disorders<sup>21</sup> – predicted relatively high levels of overeating in response to emotional distress in another population-based study.<sup>22</sup> Together, these studies<sup>14,20,22</sup> suggest that children of mothers with a history of eating disorders may also respond to stress by altering their food intake, just like their mothers.

Regarding mothers' feeding practices, mothers with eating disorders are likely to use food as reward or comfort and have more conflicts with their offspring during mealtimes than women without eating disorders.<sup>21</sup> Recent population-based studies suggest that mothers with a history of eating disorders or with high scores on questionnaires measuring current eating disorder symptoms (e.g. Eating Disorder Inventory-2 (EDI-2)) may also have different feeding strategies,<sup>18,23,24</sup> although the evidence is still inconclusive.<sup>25</sup> Some studies reported that women with a history of BN and with relatively high scores on the EDI-2 subscale bulimia more often used controlling feeding strategies, like restricting children's food intake or pressuring to eat.<sup>18,23,24</sup> This observation, however, is not supported by all studies.<sup>18,23,24,26</sup> In general, a history of AN and EDI-2 subscales drive for thinness and body dissatisfaction were not related to these feeding strategies,<sup>18,23,24</sup> only one study reported a positive correlation with restriction and pressure to eat.<sup>23</sup> However, despite their population-based character, most of these studies were limited by their small sample size ( $n < 100$ ).<sup>23,25,26</sup> Further population-based research with sufficiently large sample sizes is needed to enhance understanding of the relation between mothers' eating disorders and their feeding strategies, as controlling feeding strategies may have adverse effects. Specifically, controlling feeding strategies have been implicated in children's energy intake and risk of overweight.<sup>27</sup>

The current population-based study aimed to enhance understanding of the consequences of maternal history of eating disorders for maternal feeding practices and children's emotional eating in early childhood. We hypothesized that particularly women with a history of BN are more likely to use controlling feeding strategies than mothers without a history of eating disorders. Second, we expected that the foundation of the intergenerational transmission of distorted eating patterns is laid early in life. Therefore, we hypothesized that already at 4 years of age, children of mothers with a history of BN have a tendency to overeat in response to emotional cues and as a consequence, have a relatively high BMI, while children of mothers with a history of AN have high levels of both emotional over- and undereating. Third, we aimed to explore if maternal history of eating disorders is specifically related to children's emotional eating or to other eating behaviors as well.

## Methods

### Study design

This study was embedded in the Generation R Study, a population-based cohort study from fetal life onwards.<sup>28,29</sup> All pregnant women living in Rotterdam, the Netherlands, with an expected delivery date between April 2002 and January 2006, were invited to participate. The participation rate was estimated at 61%. Assessments included physical examinations and parental questionnaires (approximately 86% filled out by mothers). Written informed consent was obtained from all participants. The local Medical Ethical Committee has approved this study. Further information is available elsewhere.<sup>28,29</sup>

### Participants

In total, 5,212 mothers provided information on their history of eating disorders and gave full consent for the postnatal phase of the Generation R Study. Those with missing data on all outcome variables were excluded ( $n=361$ ). However, the different outcome data were not complete for all participants, thus the population for analyses varied per outcome ( $n$  between 3,742 and 4,051). In total, 4,851 (93.1%) mother-child dyads had one or more outcomes assessed at follow-up. To check whether different samples for analysis influenced our findings, we also conducted the analyses in those 3,115 (59.8%) participants with complete outcome data.

## Measures

### ***Maternal history of eating disorders***

Mothers' history of eating disorders was assessed by a self-report questionnaire during pregnancy. A vignette was provided to clarify what was meant by anorexia nervosa (AN) and bulimia nervosa (BN). Diagnostic criteria were the starting point in the design of the vignette, but were slightly changed to create an understandable and clear description of both disorders. AN was described by characteristics as abnormal fears of becoming overweight while being very thin, body dissatisfaction, and trying to lose weight. The description of BN included weekly bouts of compulsive eating and compensating behaviors such as vomiting or using laxatives. The vignette was followed by questions about having suffered from either AN or BN (ever and in the previous year): "Have you *ever* tried to lose weight to the extent that you may have suffered from anorexia?", "Have you suffered from anorexia *in the past year*?", "Have you *ever* had bouts of compulsive eating as described for bulimia?", "Have you suffered from bulimia *in the past year*?". Due to low prevalence of eating disorders in the year before pregnancy, women were grouped according to their lifetime history of eating disorders (a history of eating disorders versus no history of eating disorders). Women with a history of eating disorders were further grouped into the non-overlapping groups: 1. history of lifetime AN (AN), 2. history of lifetime BN (BN), 3. history of lifetime AN *and* BN (cross-over AN and BN). The latter group refers to women who have had an episode of AN at some point in their life and also experienced an episode of BN. These women were considered as a separate category as they may have had a higher degree of eating disorder severity.<sup>30</sup> BED was not included in our study, as this was not a psychiatric diagnosis at the time we assessed ED.<sup>6</sup>

Obtaining a clinical diagnosis was not feasible given our large sample size. However, in a sub-sample (n=928) of the overall Generation R study, self-reported eating disorders were compared with clinical diagnosis (n=8 of AN, n=17 of BN).<sup>31</sup> Micali et al. reported excellent sensitivity (100%) and specificity (96%) for self-reported lifetime AN, and very good sensitivity (94%) and specificity (81%) for self-reported BN.<sup>31</sup>

### ***Maternal feeding practices***

When children were 4 years old, maternal feeding practices were assessed with three scales of the Child Feeding Questionnaire (CFQ): monitoring, restriction, and pressure to eat.<sup>32</sup> The monitoring scale (3 items) indicates the extent to which parents carefully watch their children's eating behaviors (e.g. "How much do you keep track of the high-fat foods that your child eats?"). The restriction scale (8 items) as-

esses whether parents limit or control their children's access to foods (e.g. "I have to be sure that my child does not eat too many sweets (candy, ice-cream, cake, or pastries)"). The pressure to eat scale (4 items) refers to the extent to which parents push their children to eat or to eat more (e.g. "My child should always eat all the food on her/his plate"). Each item was answered on a Likert scale with possible scores from 1 (never/disagree) to 5 (always/agree). Scale scores were calculated by summing the items. Mean scale scores were 13.3 (standard deviation [SD]=2.4, range: 3-15) for monitoring, 23.8 (SD=6.2, range: 8-40) for restriction, and 12.4 (SD=3.9, range: 4-20) for pressure to eat. The Cronbach's alphas for the CFQ scales previously reported within the Generation R Study indicated moderate to high internal consistencies ( $\alpha$  ranging from .66 for pressure to eat to .92 for monitoring).<sup>33</sup>

### ***Children's eating behavior***

At 4 years of age, children's eating behavior was assessed using the Children's Eating Behaviour Questionnaire (CEBQ).<sup>33</sup> The CEBQ is a validated, multi-dimensional parent-report questionnaire designed to measure differences in eating behaviors.<sup>33</sup> The CEBQ consists of eight scales, each containing 3 to 6 items (5-point Likert scale). The present study mainly focused on the subscales emotional undereating (4 items) and emotional overeating (4 items). The emotional undereating scale reflects a tendency to eat less in an upset, angry or tired state, and more in response to happiness. The emotional overeating scale measures the tendency to eat more when anxious, irritated, worried, or bored. To examine whether effects of maternal history of eating disorders on child eating behavior were specific to the emotional eating domain or more general, we also used the scales food responsiveness (e.g. "my child is always asking for food"), enjoyment of food (e.g. "my child loves food"), satiety responsiveness (e.g. "my child gets full up easily"), and food fussiness (e.g. "my child decides that s/he doesn't like food, even without tasting it"). Sum scores for all scales were calculated as described for the CFQ scales. Mean scale scores for children's emotional eating behaviors were 11.0 (SD=3.3, range: 4-20) for emotional undereating and 5.8 (SD=2.4, range: 4-20) for emotional overeating. It has previously been shown that the internal consistencies for the CEBQ scales in the Generation R Study were reasonable to high ( $\alpha$  ranging from .78 to .89).<sup>33</sup> Moreover, the use of the CEBQ in a Dutch population has been previously validated, indicating good psychometric properties.<sup>34</sup>

### ***Children's body mass index***

Children's height and weight were measured by trained staff when children visited our research center at the age of 6 years.<sup>28</sup> These measures were used to calculate child body mass index (BMI, kg/m<sup>2</sup>). Mean BMI was 16.1 (SD=1.7). Age- and

sex-adjusted standard deviation scores (child BMI-SDS) were calculated using Dutch reference curves.<sup>35</sup>

### **Covariates**

Sociodemographic and psychosocial covariates that may confound the studied associations were considered based on previous research.<sup>45</sup> Variables were only included in the models if they changed the effect estimates for maternal eating disorders by 10% or more. Potential confounders included net monthly household income, single parenthood, and maternal age, ethnic background (based on country of birth of the mother and her parents), educational level, BMI, and psychiatric problems. Maternal psychiatric problems were assessed with the Brief Symptom Inventory (BSI), a validated and reliable self-report questionnaire containing 53 items scored on a 5-point Likert scale.<sup>36</sup> The items reflect a spectrum of psychiatric symptoms including anxiety, depressive symptoms, hostility, and psychotic symptoms. We used the overall score which was dichotomized at the 80<sup>th</sup> percentile (cut-off: >0.46) to indicate high levels of psychiatric symptoms. These covariates were assessed by questionnaires during pregnancy. Weight and height were measured at enrollment in the study and used to calculate maternal BMI (kg/m<sup>2</sup>).

Finally, parenting stress was included as a covariate in this study, as it may be a confounder but also a mediating factor in the relation of maternal history of eating disorders with maternal feeding practices or children's eating behavior. The Nijmeegse Ouderlijke Stress Index – Kort (NOSIK),<sup>37</sup> the Dutch version of the Parenting Stress Index – Short Form<sup>38</sup> was used to measure stress related to the parent-child relationship when the children were 18 months of age. In this study, we used the parent domain (11 items, 4-point Likert scale), which reflects stress related to parenthood and child rearing (e.g. "Parenthood of this child is harder than I thought"). The total parenting stress score was dichotomized with the 20% highest scores indicating high parenting stress.

### **Statistical analyses**

We examined sample characteristics using  $\chi^2$ -tests, ANOVAs, and Mann-Whitney tests where appropriate. Linear regression analyses were used to examine the association between maternal history of eating disorders and outcome variables (i.e. maternal feeding practices, children's eating behavior, and BMI), while adjusting for potential confounding factors. To prevent multiple testing, we first compared mothers with and without a history of eating disorders. Only if this comparison indicated statistically significant differences, we then examined whether this difference was due to any specific eating disorder by separately studying mothers with AN, moth-

ers with BN, and mothers with both AN and BN. In all analyses, the CFQ and CEBQ scales were Z-standardized to enhance comparability of the regression coefficients. In sensitivity analyses, we repeated above described analyses in those participants with complete data on all outcomes (CFQ, CEBQ emotional under- and overeating, child BMI;  $n=3,135$ ) to check whether different samples for analysis influenced our findings.

We calculated Cook's distance values to check for cases with an undue influence on the regression models and found that all values were below one. We also checked the assumption of normal distributed residuals, assessed with histograms and P-P-plots, and found no major violations. Moreover, given our large sample size, regression analyses were considered appropriate.<sup>39</sup> Missing values on confounders (ranging from 0% missing values for maternal age to 12% for household income) were estimated using multiple imputation techniques<sup>40</sup> to prevent bias. The effect estimates are the pooled results of 20 imputed datasets. All statistical analyses were performed with SPSS 20.1.

### ***Non-response analyses***

Of the 5,212 mothers who provided information on eating disorder history, mothers with missing data on all outcomes ( $n=361$ ) were compared to mothers and children with at least one outcome measure available ( $n=4,851$ ). Data were more often missing in mothers who were younger ( $t[409]= -12.8, p<0.001$ ), lower educated ( $\chi^2[2]= 101.8, p<0.001$ ), of non-Dutch national origin ( $\chi^2[2]= 141.0, p<0.001$ ), with a lower household income ( $\chi^2[1]= 118.4, p<0.001$ ), or who had higher levels of psychiatric problems ( $\chi^2[1]= 51.3, p<0.001$ ). No difference was found in maternal BMI between those with and without missing data ( $t[398.7]= 1.4, p>0.15$ ).

## **Results**

### **Population characteristics**

Characteristics of the total study population and stratified by maternal history of eating disorders are presented in Table 2.2.1. Of all participants, 8.6% ( $n=415$ ) reported to have ever experienced an eating disorder. More specifically, 2.5% ( $n=121$ ) had a history of AN (but not BN), 3.9% ( $n=189$ ) had a history of BN (but not AN), and 2.2% ( $n=105$ ) had experienced cross-over between AN and BN. These women were relatively likely to have high levels of psychiatric problems (29.7% for women with any eating disorder versus 14.7% for women without eating disorders,  $\chi^2[1]=61.9, p<0.001$ ). All types of eating disorders were associated with relatively high percentages of

psychiatric problems (24.8% for women with AN,  $\chi^2[1]= 9.4, p=0.002$ ; 29.2% for BN,  $\chi^2[1]= 28.7, p<0.001$ ; 36.3% for both AN and BN,  $\chi^2[1]= 35.9, p<0.001$ ). Women with a history of any eating disorder were also more likely to have high levels of parenting stress (21.7% versus 15.9% for women without eating disorders,  $\chi^2[1]= 7.6, p=0.006$ ), especially women with AN (24.0%,  $\chi^2[1]= 4.7, p=0.030$ ) and women with cross-over between AN and BN (28.1%,  $\chi^2[1]= 9.5, p=0.002$ ).

**TABLE. 2.2.1 General characteristics of the study population**

MATERNAL CHARACTERISTICS		N	TOTAL (N=4,851)	MATERNAL HISTORY OF EATING DISORDERS#	
				NO EATING DISORDER (N=4,436)	ANY EATING DISORDER (N=415)
Age at enrollment	mean years, SD	4,851	30.8 (4.8)	30.8 (4.8)	30.8 (5.0)
Net household income	% low (< €2000/ month )	1,248	29.3	29.2	29.9
Ethnic background:	% Dutch	2,867	60.9	60.7	63.5
	% other European	390	8.3	8.0	11.1
	% Non-Western	1,450	30.8	31.3	25.4
Education level:	% lower vocational education or less	2,187	46.7	46.6	47.5
	% higher vocational education	1,098	23.4	23.4	23.9
	% higher academic education	1,403	29.9	30.1	28.6
Single parent	% yes	480	10.3	10.3	10.5
Psychiatric problems	% high	757	16.0	14.7	29.7
BMI at enrollment	median, IQR	4,822	23.6 (4.7)	23.6 (4.7)	23.3 (5.3)
Parenting stress	% high	665	16.4	15.9	21.7
<b>CHILD CHARACTERISTICS</b>					
Sex	% boys	2,397	49.4	49.2	51.8

# Values are percentages for categorical variables, mean (standard deviation) for continuous variables, and median (interquartile range) for non-normally distributed continuous variables (maternal BMI).

## Maternal feeding practices

The associations between maternal history of eating disorders and different feeding practices are presented in Table 2.2.2 (adjusted for potential confounders). Table 2.2.2 shows that mothers with a history of eating disorders used less pressure to eat (adjusted B= -0.20, 95% confidence interval [CI]: -0.32; -0.09) than mothers without a history of eating disorders. Further analyses in the subgroups of eating disorders indicated that particularly mothers with a history of AN (adjusted B= -0.30, 95% CI:

-0.49; -0.11) were likely to use low levels of pressure to eat (see Table 2.2.3). The same tendency was seen in mothers with a history of BN or both AN and BN, but effect estimates were less strong and did not reach statistical significance.

**TABLE 2.2.2 Associations of maternal history of eating disorders with maternal feeding practices (Child Feeding Questionnaire), children's emotional eating (Children's Eating Behaviour Questionnaire), and child body mass index standard deviation scores (BMI-SDS)**

MATERNAL HISTORY OF EATING DISORDERS	B (95% CI) <sup>#</sup> § FOR MATERNAL OUTCOMES: FEEDING PRACTICES		
	MONITORING (N=3,753)	RESTRICTION (N=3,752)	PRESSURE TO EAT (N=3,760)
No eating disorder	0 [reference]	0 [reference]	0 [reference]
Any eating disorder	0.09 (-0.01; 0.20)	0.04 (-0.07; 0.15)	-0.20 (-0.32; -0.09)***
MATERNAL HISTORY OF EATING DISORDERS	B (95% CI) <sup>#</sup> § FOR CHILD OUTCOMES: EMOTIONAL EATING AND BODY MASS INDEX		
	EMOTIONAL UNDEREATING (N=3,763)	EMOTIONAL OVEREATING (N=3,742)	BMI-SDS (N=4,042)
No eating disorder	0 [reference]	0 [reference]	0 [reference]
Any eating disorder	0.04 (-0.08; 0.15)	0.12 (0.01; 0.23)*	0.04 (-0.06; 0.13)

\* p < 0.05, \*\*\*p < 0.001.  
# Values are regression coefficients and reflect differences in z-scores between the eating disorder group and the reference group.  
§ Adjusted for household income, single parenthood and maternal characteristics: age, ethnic background, educational level, psychiatric problems and BMI.

## Children's eating behavior

The lower part of Table 2.2.2 presents the relations of maternal history of eating disorders with children's emotional eating behavior and BMI. Children of mothers with a history of any eating disorder had higher levels of emotional overeating than the reference group (adjusted B=0.12, 95% CI: 0.01; 0.23). Table 2.2.3 shows that this effect was the strongest among mothers with a history of AN (B=0.19; 95% CI: 0.00, 0.39). All analyses were adjusted for possible confounding factors; additional adjustment for parenting stress barely affected the effect estimates (data not shown). To test the specificity of the association between maternal history of eating disorders and offspring's emotional eating, we additionally examined children's food responsiveness, enjoyment of food, satiety responsiveness and food fussiness as outcomes (as measured with the CEBQ). Maternal history of eating disorders was not related to any of these eating behavior dimensions or children's BMI.



**TABLE 2.2.3 Associations of maternal history of specific eating disorders with maternal pressure to eat and child emotional overeating.**

MATERNAL HISTORY OF EATING DISORDERS	B (95% CI) <sup>#§</sup> FOR PRESSURE TO EAT (N=3,760)
No eating disorder	0 [reference]
Anorexia Nervosa only	-0.30 (-0.49; -0.11)**
Bulimia Nervosa only	-0.15 (-0.31; 0.01)
Cross-over Anorexia Nervosa and Bulimia Nervosa	-0.17 (-0.39; 0.05)
MATERNAL HISTORY OF EATING DISORDERS	B (95% CI) <sup>#§</sup> FOR EMOTIONAL OVEREATING (N=3,742)
No eating disorder	0 [reference]
Anorexia Nervosa only	0.19 ( 0.00; 0.39)*
Bulimia Nervosa only	0.04 (-0.12; 0.20)
Cross-over Anorexia Nervosa and Bulimia Nervosa	0.15 (-0.07; 0.37)

\*  $p < 0.05$ , \*\* $p < 0.01$ .  
# Values are regression coefficients and reflect differences in z-scores between a certain eating disorder category and the reference group.  
§ Adjusted for household income, single parenthood and maternal characteristics: age, ethnic background, educational level, psychiatric problems and BMI.

## Sensitivity analyses

Sensitivity analyses among those with complete data on all core outcomes ( $n=3,135$ ) yielded very similar findings. For instance, in this subsample, we also found lower levels of pressure to eat in mothers with a history of eating disorders (adjusted  $B = -0.16$ , 95% CI:  $-0.28$ ;  $-0.03$ ), especially amongst mothers with a history of AN (adjusted  $B = -0.29$ , 95% CI:  $-0.51$ ;  $-0.08$ ). Only the association between maternal history of AN and children's emotional overeating was – though in the same direction – no longer statistically significant ( $B = 0.19$ , 95% CI:  $-0.03$ ;  $0.41$ ), probably due to reduced power.

## Discussion

In this longitudinal population-based study, we found support for our hypothesis that a mother's history of eating disorders is associated with differences in mothers' feeding practices and preschoolers' emotional eating patterns. Our results suggest that mothers with a history of lifetime AN use less pressuring feeding strategies and that their children have a tendency toward overeating in response to emotional

cues. As hypothesized, the associations between maternal history of eating disorders and children's eating behaviors were specific to emotional eating and hardly reflected differences in other eating behaviors.

## Maternal feeding practices

The finding of low levels of pressure to eat amongst mothers with a history of eating disorders, particularly AN, is not congruent with prior studies reporting no association<sup>18</sup> or reporting higher levels of pressuring feeding strategies<sup>15,23</sup> in these women. This inconsistency in results could be related to the assessment of pressure to eat. Stein et al.,<sup>15</sup> and Blisset and Haycraft<sup>23</sup> used observations of pressure to eat during mealtimes, which is probably a more reliable measurement than maternal self-reports of feeding. However, the use of a single meal-time observation and the absence of siblings during the observation could have led to non-representative findings.<sup>23</sup> Moreover, small sample sizes ( $n=23$  and  $n=56$ ) and lack of control for potential confounders have limited conclusions of these studies. Results of Reba-Harrelson et al.<sup>18</sup> are more congruent with our findings, possibly due to similar ascertainment (i.e. the use of maternal self-report). Reba-Harrelson et al. also reported lower levels of pressure to eat in women with a history of AN, although this association did not reach statistical significance as a result of the small number of women with a history of AN ( $n=17$ ).<sup>18</sup>

Several explanations for our finding that mothers with a history of eating disorders, particularly mothers with AN, reported little pressuring feeding must be discussed. First, due to on-going fears of gaining weight, mothers with a history of AN may well be more concerned about their children's weight<sup>41</sup> and therefore less likely to pressure their children to finish all the food on their plate. Another possible explanation is that women with a history of AN experienced much tension and pressure in food-related situations at the time they had an eating disorder, making them hesitant to interfere with their children's eating habits. Moreover, a mother with such a history might actively try not to pressure her child to eat because of concerns that her child will develop an eating disorder. Either way, low levels of pressure to eat can be considered as positive, since pressure to eat is associated with children's negative affective reactions to food.<sup>42</sup> Nevertheless, there may also be an optimal level of pressuring feeding. A lack of encouragement or prompts to eat particular foods or to eat more may also have negative consequences for children's health. Lack of encouragement may contribute to an unhealthy low intake of certain foods (e.g. vegetables) or a limited taste development.

## Children's emotional eating

Regarding child outcomes, we found that a history of eating disorders, again particularly a history of AN, predicted higher levels of emotional overeating in children. In a smaller subsample with complete outcome data, we found a similar, though non-significant association, probably due to reduced statistical power. However, since effect estimates were in the same direction, our findings support our hypothesis that maternal history of eating disorders specifically affects emotional eating rather than eating behavior in general and could be interpreted in two ways. First, this may reflect a true finding, suggesting that the first signs of an intergenerational transmission of eating psychopathology are already apparent early in life. Evidence supporting this comes from a study observing that women with AN retrospectively reported a pattern of emotional eating earlier in life.<sup>43</sup> An alternative explanation is that the reports on children's emotional overeating may have been biased in mothers with a history of AN. A study showed that women with AN reported emotional overeating, while in fact eating only a small amount of food.<sup>41</sup> Possibly, women with a history of AN still have a distorted perception which may be reflected in their judgment and report on children's eating behavior. Over-reporting of problematic eating behavior may also be due to concerns about child weight, as a small clinical study indicated that women with a history of AN were over-concerned about their daughters being overweight.<sup>44</sup> Importantly, the validity of our assessment of child eating behavior is strengthened by prior research indicating that maternal reports were strongly related to observations, even among women with eating disorders.<sup>19</sup> Furthermore, if mothers with a history of eating disorders are biased in reporting their children's eating behavior, we would expect to find differences in a range of eating behaviors, instead of only in emotional eating behavior.

Regardless of the validity of maternal reports, the way mothers perceive their children's emotional eating can possibly affect children's actual eating behavior. Mothers' perceptions may influence their own feeding strategies on the long term, which, consequently, could lead to changes in children's emotional eating behavior. Evidence for such a self-fulfilling prophecy of eating behavior comes from obesity research indicating that parents' perceptions of children's overweight altered parents' feeding strategies which in turn predicted children's eating and weight.<sup>27</sup> Although we did not find an association between maternal history of eating disorders and BMI of 6-year-old children, it is possible that the effect on BMI only becomes evident after a longer period of time. We also did not find an association between maternal history of BN and children's emotional eating. Again, it could be that emotional overeating starts emerging when children are slightly older, but our non-finding might also be explained by possible misclassification of maternal history of BN.

Our assessment of women's history of BN only focused on binge eating and did not incorporate compensating behaviors. However, a description of BN including compensation behaviors was described in the vignette preceding this question and the self-report of BN has been validated in our cohort.<sup>31</sup> Alternatively, a mother's history of AN might be more important for children's emotional eating tendencies than a mother's history of BN, because of the ego-syntonic nature of AN and the ego-dystonic nature of BN.<sup>44</sup> Mothers with a history of BN might be more keen on preventing the transmission of their ego-dystonic behaviors, i.e. behaviors in conflict with one's self-image, to their offspring. Considering the ego-syntonic nature of AN, denial of being ill makes AN not only extremely difficult to treat, but possibly also more prone to passing on eating disturbances to a next generation.

## General well-being of mothers with a history of eating disorders

Our results indicated that the impact of a maternal history of eating disorders extends beyond food related parenting behaviors to general well-being of mothers. In congruence with earlier studies,<sup>4,16</sup> the women in our sample with a history of eating disorders had more psychiatric problems than women without such a history. Moreover, these women also experienced relatively high levels of parenting stress. The analyses were adjusted for these indicators of general well-being of mothers, as psychopathology and parenting stress may precede or confound the association between history of eating disorders and offspring eating behavior. However, controlling for maternal psychopathology may also represent over-adjustment. Aggregation of eating disorders and internalizing psychopathology within families suggests common familial factors, perhaps genetic influences, causing both disorders.<sup>9</sup> Given the high co-occurrence of eating disorders with anxiety and depression,<sup>8</sup> it could be argued that internalizing psychiatric problems are inherent to eating disorders. Given that psychiatric problems and parenting stress are risk factors for adverse child development,<sup>45,46</sup> our findings suggest that stress and psychopathology may underlie the previously reported association between maternal history of eating disorders and children's emotional problems.<sup>18</sup>

## Strengths and limitations

The strengths of this study are its population-based, longitudinal design and a large number of participants which allowed us to distinguish between different types of eating disorders. Despite the strengths of our study, several limitations should be considered. First, ascertainment of eating disorder history was obtained by self-report, which may have resulted in reporter bias. For instance, the subgroup

with lifetime AN and BN may include both women with cross-over AN and BN as well as some women with purging AN as our lifetime assessment did not include very accurate measures of symptom occurrence. However, we previously showed substantial overlap between self-reports and clinical diagnosis of lifetime eating disorders in our sample.<sup>31</sup> Although this validation was performed in a subsample with only very few women receiving a lifetime diagnosis of AN or BN, the overlap between the different ascertainment methods provides some support for the use of self-reports. Second, the reliability of the CFQ scale pressure to eat was only moderate in the Generation R Study<sup>33</sup> and the factor structure of the Dutch translation of the CFQ has not been validated yet. Third, data on children's eating behavior were based on maternal report, because obtaining information from other informants or observations was not feasible given the sample size and the young age of children. As discussed, concerns about eating among mothers with a history of eating disorders may result in differential reporting of problematic eating behavior. However, Whelan and Cooper showed that maternal reports correlate with actual observations of children's eating behavior.<sup>19</sup> Another limitation of our design is the lack of information on current eating disorders amongst mothers, whereby we cannot estimate the impact of possible ongoing eating disorders. Furthermore, loss to follow up in the Generation R Study is higher amongst parents with a low socioeconomic status and non-Western origin.<sup>28</sup> Thus, some caution is needed when generalizing results towards the general population.

## Conclusions

Maternal history of eating disorders was associated with less pressuring feeding in mothers, and higher levels of emotional overeating amongst 4-year-old children compared to unexposed children. Particularly the high levels of emotional eating among children of mothers with a history of eating disorders may have implications if this reflects actual eating behavior rather than maternal perception. These children may be at risk of obesity,<sup>13</sup> but also of developing eating disorders as emotional eating has been related to eating psychopathology.<sup>10,11,43</sup> Our results suggest that assessing lifetime eating disorders can be used for screening of pregnant women to identify children at risk, also considering the high levels of parenting stress and psychiatric problems amongst mothers with a history of eating disorders. Future research is needed to replicate our findings, preferably using independent child observations or multiple informants (e.g. both mothers and fathers) to control for potential reporter bias, and to follow these children up to confirm if emotional eating patterns at 4 years of age predict eating psychopathology later in life.

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CHAPTER 2.3

## Maternal history of eating disorders:

Children's emotional and behavior problems

### **Manuscript based on this chapter:**

Lisanne M. de Barse, Henning Tiemeier, Nadia Micali, Vincent W.V. Jaddoe, Frank C. Verhulst, Jolien Steenweg-de Graaff, Oscar H. Franco, Pauline W. Jansen. Children of mothers with a history of eating disorders: Are they at risk of emotional and behavior problems? *To be submitted for publication*





## Infant feeding & Child picky eating

### **Manuscript based on this chapter:**

Lisanne M. de Barse, Pauline W. Jansen, Lisa R. Edelson-Fries, Vincent W.V. Jaddoe, Oscar H. Franco, Henning Tiemeier, Jolien Steenweg-de Graaff. Infant feeding and child fussy eating: The Generation R Study. *Submitted for publication*





## CHAPTER 3.2

# Parents' anxiety and depressive symptoms & Child picky eating

### **Manuscript based on this chapter:**

Lisanne M. de Barse, Sebastian Cardona Cano, Pauline W. Jansen, Vincent W.V. Jaddoe, Frank C. Verhulst, Oscar H. Franco, Henning Tiemeier, Anne Tharner. Are parents' anxiety and depression related to child fussy eating? *Archives of Disease in Childhood*, 2015, 101, 533-538

## Abstract

**Background:** Our aim was to examine the association between parental anxiety and depression with child picky eating – that is, consistent rejection of particular food items.

**Methods:** This study included 4,746 parent-child dyads, participating in Generation R, a prospective population-based cohort from fetal life onwards in the Netherlands. Parental emotional problems (i.e. symptoms of anxiety and depression) were assessed with the Brief Symptoms Inventory during pregnancy and the preschool period (child age 3years). The food fussiness scale of the Children's Eating Behaviour Questionnaire was administered at age 4 years.

**Results:** Maternal anxiety during pregnancy and during the child's preschool period was related to higher food fussiness sum-scores in children. For instance, per point on the anxiety scale in pregnancy, children had on average a 1.02 higher sum-score (95% CI: 0.59; 1.46) on the food fussiness scale, after adjustment for confounders. Likewise, mothers' depressive symptoms at both time points were associated with picky eating behavior in their children (e.g. in the antenatal period: per point on the depression scale, children had a 0.91 point higher on the food fussiness scale, 95% CI: 0.49; 1.33). We found largely similar associations between fathers' emotional problems and children's picky eating. However, fathers' anxiety during the antenatal period was not related to child picky eating.

**Conclusions:** Maternal and paternal emotional problems were prospectively associated with picky eating in preschoolers. Healthcare practitioners should be aware that non-clinical symptoms of anxiety and depression in parents are risk factors for child picky eating.

## Introduction

Picky eating is characterized by consistent rejection of particular foods, which results in a restricted diet variety,<sup>1</sup> causing major concerns among parents.<sup>2</sup> Child picky eating has been associated with functional constipation,<sup>3</sup> weight problems,<sup>4</sup> and behavior problems.<sup>5</sup> Previous research suggested parental controlling feeding,<sup>4</sup> and parental physical and mental health problems<sup>6</sup> as potential risk factors for picky eating (also called 'fussy' or 'selective' eating). However, the etiology of picky eating is not well understood.<sup>6</sup>

It is well known that emotional problems of parents (i.e. anxiety and depression) are related to problematic child development,<sup>7</sup> including disturbed eating behaviors.<sup>8,9</sup> A complex interplay of multiple factors such as genetics, disturbed parent-child interaction, and modeling of parent behavior account for the increased risk of problems in children,<sup>7</sup> and may also affect children's picky eating. Maternal emotional problems have been related to picky eating in preschool-aged children in population-based studies.<sup>6,10</sup> Maternal emotional problems during the child's preschool period have been found to be predictive for persistent picky eating at a later age.<sup>11</sup> Farrow and Blissett, however, have reported that antenatal and postnatal maternal psychiatric symptoms did not predict picky eating in six-month-old children.<sup>12</sup>

Most studies focused on maternal symptoms during the child's preschool period. This is a sensitive period in development, but the Barker hypothesis<sup>13</sup> highlights the need to also study antenatal anxiety and depression. Another advantage of studying emotional problems in the antenatal period is that the association with children's picky eating is less prone to reverse causation – that is, children's picky eating is not likely to affect their mothers' problems during pregnancy. In addition, most previous studies were limited in their reliance on maternal reports of both exposure (emotional problems) and outcome (child picky eating).<sup>6,10,11,14</sup> Consequently, reported associations may be overestimated due to reporter bias as the depression-distortion hypothesis states that mothers with psychiatric problems might have a biased perception of their child's behavior.<sup>15</sup> Last, most studies focused on mothers' anxiety and depression, without studying the effects of fathers' symptoms.

The current study's objective was to examine whether maternal and paternal emotional problems are prospectively associated with children's picky eating, using multiple informants of child eating behavior. More specifically, we aimed to evaluate the role of anxiety and depressive symptoms in the antenatal and preschool period.

## Methods

### Study design and procedure

This study was embedded in Generation R, a population-based prospective cohort from fetal life onwards.<sup>16,17</sup> Pregnant women living in Rotterdam, the Netherlands, with a delivery date between April 2002 and January 2006 were invited to participate (response rate: 61%). The local Medical Ethical Committee has approved the study. Sociodemographic information was collected by postal questionnaires during pregnancy and from medical birth records completed by gynecologists and midwives. Parental emotional problems were assessed by postal questionnaire during mid-pregnancy, and again when the child was 3 years old. At 3 and 4 years of age, parents filled in postal questionnaires including an assessment of their children's eating behavior. More detailed information about the design and procedure is available elsewhere.<sup>16</sup>

### Participants

Parents of 7,295 children gave full consent for the preschool phase of Generation R. Those with missing data on the food fussiness scale of the Children's Eating Behaviour Questionnaire (CEBQ) were excluded ( $n=2,355$ ). Of the remaining parent-child dyads, 194 participants had missing values of maternal anxiety or depression during pregnancy and 3 years later, yielding a sample size of 4,746 children and mothers. The population for analysis with fathers' anxiety or depression was smaller ( $n=4,144$ ), as 602 participants had missing values for fathers' anxiety or depression on both time points.

### Measures

#### *Parental anxiety and depressive symptoms*

Anxiety and depressive symptoms of both mothers and fathers were assessed with the Brief Symptom Inventory (BSI) at two time points: during mid-pregnancy and 3 years later. The BSI is a validated 53-item self-report questionnaire assessing a spectrum of psychiatric problems in the preceding seven days.<sup>18,19</sup> We used the anxiety scale (e.g. 'feeling fearful') and the depression scale (e.g. 'feeling lonely'). Each scale consists of six items rated on a 5-point Likert-scale from 0 (not at all) to 4 (extremely). For each scale, mean-scores were calculated, with higher scores indicating more problems.



### ***Child picky eating behavior***

At age 4 years, picky eating was assessed with the CEBQ, a validated parent report questionnaire.<sup>20</sup> The CEBQ consists of eight subscales, containing 35-items on which parents rate the frequency of their children's eating behaviors. We used the subscale food fussiness, which consists of six items covering children who are difficult to please with meals, who display food neophobia (e.g. 'My child refuses new foods at first'), and who have a limited diet variety (e.g. 'My child enjoys a wide variety of foods', reverse coded). Each item was answered on a Likert-type scale from 1 (never) to 5 (always). Scale sum-scores were calculated, with higher scores indicating more food fussiness (range: 6-30).

As most CEBQs were filled out by mothers (~88%), we also used the Child Behavior Checklist for toddlers (CBCL/1½-5)<sup>21</sup> for which we had multiple informants. Two items were used as a proxy for picky eating: (1) 'does not eat well' and (2) 'refuses to eat' in the past two months. These questions were answered by both mothers and fathers when the children were 3 years old. Items were rated on a 3-point Likert scale from 1 (not true) to 3 (often true). Sum-scores were calculated (range 2-6) and children with a score of  $\geq 4$  were classified as 'picky eaters'.<sup>22</sup>

### ***Covariates***

During pregnancy, questionnaires were used to assess sociodemographic characteristics: parental age, net monthly household income, parental ethnic background (based on country of birth of parents and grandparents), parental educational level, single parenthood, and parity (defined as number of live births mothers delivered before birth of the participating child). Mode of delivery, sex of child, and birth characteristics (birth weight and gestational age) were obtained from medical records.

### **Statistical analyses**

We used separate linear regression analyses to test whether higher scores of mothers' anxiety and depression on the BSI at each time point (during pregnancy, and at 3 years postnatal) were related to higher sum-scores on the CEBQ's food fussiness scale. We also tested the independent effects of maternal emotional problems on child picky eating by analyzing the two time points in the same model. In addition, we explored whether fathers' anxiety and depression scores at each time point were related to picky eating, using separate linear regression analyses. All antenatal models were adjusted for sociodemographic characteristics. All models with postnatal emotional problems were additionally adjusted for mode of delivery, sex of child, and birth characteristics.

We performed several sensitivity analyses. As the CEBO's food fussiness scale was mainly reported by mothers, the associations of maternal anxiety and depression with this outcome measure may be prone to reporter bias. Therefore, we additionally examined the associations of mothers' anxiety and depression scales (continuously) with the CBCL data on picky eating, as obtained by multiple informants. Separate logistic regression analyses were conducted for mother reports and father reports on the CBCL. Second, using linear regression analyses, we compared the food fussiness scores of the following groups of children: (1) children of mothers who had average or below average anxiety or depression scores (reference group); (2) children of mothers who had above average anxiety scores (0.50 and higher but below clinical cut-off) or above average depression scores (0.33 and higher but below clinical cut-off); (3) children of mothers who had clinically significant anxiety scores (0.71 and higher) or clinically significant depression scores (0.80 and higher). The Dutch cut-offs for the BSI were used to categorize the mothers.<sup>23</sup> All sensitivity analyses were adjusted for the same potential confounders as described above.

Multiple imputation techniques were used to impute missing values on confounders and exposure.<sup>24</sup> The reported B-values are pooled from 20 imputed datasets. In addition, we repeated our main analyses in complete cases. All statistical analyses were performed with SPSS V.21.0.

## Results

### Population characteristics

Sample characteristics are presented in Table 3.2.1. The mean food fussiness sum-score at age 4 years was 17.7 (standard deviation [SD]=4.9). Using the CBCL as proxy for picky eating, ~30% of all children were classified as picky eaters at age 3 years. In total, agreement of mothers and fathers about their child being a picky or non-picky eater was 76.7%. We calculated Yule's  $Y^{25}$  to be 0.47, indicating moderate agreement between mothers and fathers.

**TABLE 3.2.1 Sample characteristics of 4,746 parent-child dyads in the Generation R Study**

FAMILY CHARACTERISTICS		N <sup>§</sup>	PERCENTAGE OR MEAN (SD) <sup>#</sup>	
Maternal age at enrollment	mean years	4,746	31.6	(4.5)
Paternal age at enrollment	mean years	4,746	34.0	(5.4)
Household income	% low (<€2000 / month)	1,262	26.6	
Mothers' ethnic background	% Dutch	3,113	65.6	
	% Moroccan	139	2.9	
	% Surinamese & Dutch Antillean	335	7.1	
	% Turkish	276	5.8	
	% other Western (mainly European)	414	8.7	
	% other non-Western	469	9.9	
Fathers' ethnic background	% Dutch	3,148	66.3	
	% Moroccan	174	3.7	
	% Surinamese & Dutch Antillean	354	7.5	
	% Turkish	258	5.4	
	% other Western (mainly European)	320	6.7	
	% other non-Western	492	10.4	
Mothers' educational level <sup>∞</sup>	% low	678	14.3	
	% medium	1,324	27.9	
	% high	2,744	57.8	
Fathers' educational level <sup>∞</sup>	% low	850	17.9	
	% medium	1,241	26.2	
	% high	2,655	55.9	
Single parent	% yes	373	7.9	
Parity	% multipara	1,964	41.4	
Mode of delivery	% cesarean section	650	13.7	
Parents' anxiety (BSI)	mean scale scores			
	Mothers' anxiety during pregnancy	4,746	0.23	(0.38)
	Mothers' anxiety at 3 years postnatal	4,746	0.18	(0.32)
	Fathers' anxiety during pregnancy	4,144	0.16	(0.29)
	Fathers' anxiety at 3 years postnatal	4,144	0.15	(0.25)
Parents' depression (BSI)	mean scale scores			
	Mothers' depression during pregnancy	4,746	0.17	(0.38)

**TABLE 3.2.1 (CONTINUED) Sample characteristics of 4,746 parent-child dyads in the Generation R Study**

Mothers' depression at 3 years postnatal		4,746	0.14	(0.33)
Fathers' depression during pregnancy		4,144	0.10	(0.26)
Fathers' depression at 3 years postnatal		4,144	0.10	(0.27)
<b>CHILD CHARACTERISTICS</b>				
Sex	% boy	2,363	49.8	
Birth weight	means grams	4,746	3442	(568)
Gestational age	means weeks	4,746	40	(2)
Picky eating				
Food fussiness at 4 years (CEBQ)	mean sum-score	4,746	17.7	(4.9)
Picky eating proxy at 3 years (CBCL)	% picky eaters reported by mothers	944	27.7	
Picky eating proxy at 3 years (CBCL)	% picky eaters reported by fathers	1,047	30.7	

§ N=4,746 as this table represents imputed data for covariates. N=4,144 for fathers' anxiety and depression. Reports of picky eating on the CBCL by both mother and father were available for 3,409 parent-child dyads.

# Values are percentages for categorical variables and means (standard deviations) for continuous variables.

∞ Low: ranging from no education to high school level; medium: lower vocational training; high: higher vocational training and higher academic training.

Abbr. BSI: Brief Symptom Inventory, CBCL: Child Behavior Checklist, CEBQ: Children's Eating Behaviour Questionnaire.

## Parental anxiety symptoms and children's picky eating behavior

Maternal anxiety symptoms during pregnancy and during the preschool period were related to picky eating in their 4-year-old children (Table 3.2.2). For instance, per point on the anxiety scale in pregnancy, children had a 1.02 higher food fussiness sum-score (95% confidence interval [CI]: 0.59; 1.46). By additionally analyzing maternal anxiety at both time points in the same model, we found that mothers' anxiety during pregnancy ( $B=0.81$ , 95% CI: 0.33; 1.29) and during the preschool period ( $B=0.54$ , 95% CI: 0.05; 1.03) were both independently related to child picky eating (not shown in tables). Fathers' anxiety in the preschool period, but not during the antenatal period, was related to picky eating in their child (Table 3.2.2).

Sensitivity analyses showed that not only children of mothers with clinically significant anxiety had elevated food fussiness scores (e.g. antenatal model:  $B=1.06$ , 95% CI: 0.46; 1.67), but children of mothers with anxiety scores above average also had higher food fussiness scores than children of mothers with average or below average anxiety scores (e.g. antenatal model:  $B=0.72$ , 95% CI: 0.21; 1.22) (see Supplementary Table 3.2.1).

**TABLE 3.2.2 Parental anxiety symptoms and picky eating in 4-year-old children (CEBQ)**

PARENTAL ANXIETY SYMPTOMS (BSI)	N	FOOD FUSSINESS SUM-SCORE <sup>§</sup>	
		B (95% CI) <sup>#∞</sup>	P-VALUE
Maternal anxiety scale scores	4,746		
Anxiety during pregnancy		1.02 ( 0.59; 1.46)	<0.001
Anxiety at 3 years postnatal		0.88 ( 0.43; 1.33)	<0.001
Paternal anxiety scale scores	4,144		
Anxiety during pregnancy		0.10 (-0.49; 0.69)	0.74
Anxiety at 3 years postnatal		0.88 ( 0.26; 1.49)	0.01

§ The food fussiness sum-scores range from 6-30.

# All reported regression coefficients are unstandardized B-values (95% confidence intervals) and quantify the difference in food fussiness score per 1 point higher parental anxiety score.

∞ Adjusted for child age when the CEBQ was completed and sociodemographic characteristics (parental age, household income, parental ethnic background, parental educational level, single parenthood, and parity) in the antenatal models and additionally adjusted for mode of delivery, child sex, and child's birth characteristics (birth weight-SDS and gestational age) in the postnatal models.

Abbr. BSI: Brief Symptom Inventory, CEBQ: Children's Eating Behaviour Questionnaire.

## Parental depressive symptoms and children's picky eating behavior

Table 3.2.3 shows that higher maternal depressive symptoms in the antenatal period as well as at 3 years postnatal were related to more picky eating behavior in their 4-year-old children (e.g. per point antenatal depression score, children had a 0.91 higher food fussiness sum-score, 95% CI: 0.49; 1.33). Likewise, the associations between fathers' depressive symptoms at both time points and children's picky eating were in the same direction (Table 3.2.3).

Similar to the independent effects of mothers' anxiety at both time points, we also found that mothers' depressive symptoms during pregnancy and 3 years later were independently related to child picky eating (data not shown). Supplementary Table 3.2.2 shows that mothers' depression scores above average already predicted picky eating, particularly during pregnancy (B=0.87, 95% CI: 0.41; 1.33 for above average scores and B=0.87, 95% CI: 0.22; 1.51 for clinically significant depression).

**TABLE 3.2.3 Parental depressive symptoms and picky eating in 4-year-old children (CEBQ)**

PARENTAL DEPRESSIVE SYMPTOMS (BSI)	N	FOOD FUSSINESS SUM-SCORE <sup>§</sup>	
		B (95% CI) <sup>#∞</sup>	P-VALUE
Maternal depression scale scores	4,746		
Depression during pregnancy		0.91 (0.49; 1.33)	<0.001
Depression at 3 years postnatal		0.81 (0.35; 1.26)	<0.001
Paternal depression scale scores	4,144		
Depression during pregnancy		0.72 (0.07; 1.36)	0.03
Depression at 3 years postnatal		0.68 (0.08; 1.28)	0.03

§ The food fussiness sum-scores range from 6-30.

# All reported regression coefficients are unstandardized B-values (95% confidence intervals) and quantify the difference in food fussiness score per 1 point higher parental depression score.

∞ Adjusted for child age when the CEBQ was completed and sociodemographic characteristics (parental age, household income, parental ethnic background, parental educational level, single parenthood, and parity) in the antenatal models and additionally adjusted for mode of delivery, child sex, and child's birth characteristics (birth weight-SDS and gestational age) in the postnatal models.

Abbr. BSI: Brief Symptom Inventory, CEBQ: Children's Eating Behaviour Questionnaire.

## Mothers' emotional problems and children's picky eating across informants

Table 3.2.4 shows that maternal emotional problems were also associated with both mother and father reports of children's picky eating on the CBCL. Effect estimates were very similar regardless of whether mothers or fathers reported their 3-year-olds' picky eating behavior (e.g. for antenatal anxiety odds ratio [OR]=1.50 (95% CI: 1.18; 1.89) as reported by mothers and OR=1.44 (95% CI: 1.13; 1.83) as reported by fathers).

**TABLE 3.2.4 Maternal emotional problems and a proxy for picky eating at age 3 years as independently reported by both parents on the CBCL**

MATERNAL EMOTIONAL PROBLEMS (BSI)	N	PICKY EATER <sup>§</sup> REPORTED BY MOTHER		PICKY EATER <sup>#</sup> REPORTED BY FATHER	
		OR (95% CI) <sup>∞</sup>	P-VALUE	OR (95% CI) <sup>∞</sup>	P-VALUE
Maternal anxiety scale scores	3,409				
Anxiety during pregnancy		1.50 (1.18; 1.89)	0.001	1.44 (1.13; 1.83)	0.003
Anxiety at 3 years postnatal		1.65 (1.27; 2.13)	<0.001	1.59 (1.23; 2.05)	<0.001
Maternal depressive scale scores	3,409				
Depression during pregnancy		1.24 (0.96; 1.61)	0.10	1.28 (1.00; 1.64)	0.05
Depression at 3 years postnatal		1.38 (1.05; 1.79)	0.02	1.29 (0.99; 1.67)	0.06

§ Values are odds ratios (95% confidence intervals) for picky eaters (n=944) compared with non-picky eaters (n=2,465), reported by mothers.

# Values are odds ratios (95% confidence intervals) for picky eaters (n=1,047) compared with non-picky eaters (n=2,362), reported by fathers.

∞ Adjusted for child age when the CBCL was completed and sociodemographic characteristics (maternal age, household income, maternal ethnic background, maternal educational level, single parenthood, and parity) in the antenatal models and additionally adjusted for mode of delivery, child sex, and child's birth characteristics (birth weight-SDS and gestational age) in the postnatal models.

Abbr. BSI: Brief Symptom Inventory, CBCL: Child Behavior Checklist.

## Additional sensitivity analyses

Results of our full case analyses (data not shown) were very similar to our main findings (Table 3.2.2 and 3.2.3). For instance, mothers' emotional problems were also related to child picky eating at age 4 years (e.g. for antenatal anxiety  $B=1.15$  (95% CI: 0.69; 1.61)). Only the associations of fathers' emotional problems in the preschool period with picky eating were no longer statistically significant, probably because of reduced power (n=2,198), although the magnitude of the associations was also slightly reduced (e.g. for anxiety  $B=0.69$  (95% CI: -0.14; 1.52)).

## Discussion

Higher maternal emotional problems during pregnancy and at 3 years postnatal were prospectively and both independently related to child picky eating in a large population-based cohort. We also found indications that fathers' emotional problems are related to child picky eating.

The finding that maternal emotional problems predicted more picky eating in children is largely consistent with previous research,<sup>6,9-11,26-28</sup> although conflicting studies exist.<sup>12,29</sup> Importantly, we found that mothers' antenatal symptoms of anxiety and depression predicted 4-year-olds' picky eating independent of mothers' symptoms at 3 years postnatal. This strongly suggests that the direction of the associations with mothers' antenatal symptoms is from mother to child. Coulthard and Harris<sup>30</sup> found that infants' persistent food refusal was related to mothers' concurrent state anxiety, but not to their trait anxiety, which is more general and stable. Consequently, they concluded that maternal anxiety is probably a consequence rather than a cause of child food refusal. However, in the present study, child picky eating at age 3 and 4 years cannot be an antecedent of mothers' symptoms during pregnancy. Moreover, our results suggest that not only clinically significant anxiety has an effect on child picky eating, but also slightly elevated anxiety symptoms.

The inclusion of both mothers' and fathers' anxiety and depression as contrasting exposures allows us to speculate about underlying mechanisms. Mothers' anxiety during both pregnancy and during the child's preschool period predicted picky eating in the child. In contrast, fathers' anxiety during pregnancy was not associated with children's picky eating. Thus, a genetic explanation is unlikely, whereas these results provide some support for fetal programming.<sup>13</sup> The association between fathers' anxiety during the preschool period and child picky eating can be explained by parenting factors. Possibly, fathers' anxiety affects children's picky eating via controlling feeding practices such as pressure to eat.<sup>31,32</sup> Such feeding practices could have counterproductive effects by contributing to negative affective reactions to food,<sup>33</sup> thereby increasing the risk of food rejection by the child. Parental anxiety may also influence children's picky eating by affecting difficulties in parent-child interactions.

Like mothers' depressive symptoms, fathers' depressive symptoms during pregnancy were related to children's picky eating. Thus for these associations, fetal programming seems unlikely. Shared heritability of depression and picky eating could underlie this association pattern, especially bearing in mind genetic influences on picky eating.<sup>34</sup> Possibly, lifestyle or socioeconomic factors impact both parental and child behaviors, although we carefully adjusted for education and income. Parenting factors may mediate the associations of both mothers' and fathers'



depressive symptoms at 3 years postnatal with child picky eating. Also, maternal depression has been related to difficulties in the mother-child interaction<sup>35</sup> and, in turn, these problematic interactions could mediate the associations with children's picky eating.<sup>36</sup>

Strengths of our study were its large population-based sample, prospective design, and multiple informant ratings. It is noteworthy that our results were similar for mother and father reports of picky eating at age 3 years, suggesting that mothers with emotional problems do not overrate their children's eating behavior. This also supports the validity of previous findings that relied on mothers' reports of child eating behavior.<sup>6,10,11,14</sup> However, we did not know whether maternal and paternal reports of picky eating were completely independent of each other, although two separate questionnaires were mailed. The BSI was used to assess psychiatric symptoms. Although a well-validated instrument,<sup>19</sup> its brief character may limit the extent to which it captures all aspects of emotional problems. As with all cohort studies, some selective loss to follow-up among families from low socio-economic status and non-Western origin occurred in Generation R.<sup>16</sup>

In conclusion, we observed that maternal and paternal emotional problems were prospectively associated with picky eating in preschoolers. For effective prevention and management of children's picky eating, the role of parents' emotional problems should be considered. Clinicians should be aware that not only severe anxiety and depression, but also milder forms of emotional problems can affect child eating behavior.

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## Supplement

**SUPPLEMENTARY TABLE 3.2.1. Above average and clinically significant anxiety in mothers and picky eating in 4-year-old children (CEBQ).**

MATERNAL ANXIETY SYMPTOMS (BSI)	N	FOOD FUSSINESS SUM-SCORE <sup>§</sup>	
		B (95% CI) <sup># ∞</sup>	P-VALUE
Anxiety during pregnancy	4,746		
Average or below (score <0.50)	3,893	0 [reference]	
Above average (score ≥ 0.50)	475	0.72 ( 0.21; 1.22)	.006
Clinical cut off (score ≥ 0.71)	378	1.06 ( 0.46; 1.67)	.001
Anxiety at 3 years postnatal	4,746		
Average or below (score <0.50)	4,164	0 [reference]	
Above average (score ≥ 0.50)	359	0.61 ( 0.05; 1.17)	.03
Clinical cut off (score ≥ 0.71)	223	0.87 ( 0.19; 1.55)	.01

§ The food fussiness sum-scores range from 6-30.

# All reported regression coefficients are unstandardized B-values (95% confidence intervals) and quantify how the food fussiness score for children of mothers with either 'above average' or 'clinically significant' anxiety scores differs from children of mothers with 'average or lower' anxiety scores.

∞ Adjusted for age child when CEBQ was filled out and sociodemographic characteristics (maternal age, household income, maternal ethnic background, maternal educational level, single parenthood, and parity) in the antenatal models and additionally adjusted for mode of delivery, child sex, and child's birth characteristics (birth weight-SDS and gestational age) in the postnatal models.

Abbr. BSI: Brief Symptom Inventory, CEBQ: Children's Eating Behaviour Questionnaire.

**SUPPLEMENTARY TABLE 3.2.2 Above average and clinically significant depression in mothers and picky eating in 4-year-old children (CEBQ)**

MATERNAL DEPRESSIVE SYMPTOMS (BSI)	N	FOOD FUSSINESS SUM-SCORE <sup>§</sup>	
		B (95% CI) <sup>#∞</sup>	P-VALUE
Depression during pregnancy	4,746		
Average or below (score <0.33)	3,833	0 [reference]	
Above average (score ≥ 0.33)	604	0.87 (0.41; 1.33)	<.001
Clinical cut off (score ≥ 0.80)	309	0.87 (0.22; 1.51)	.01
Depression at 3 years postnatal	4,746		
Average or below (score <0.33)	3,949	0 [reference]	
Above average (score ≥ 0.33)	582	0.48 (0.03; 0.93)	.04
Clinical cut off (score ≥ 0.80)	215	1.11 (0.38; 1.84)	.003

§ The food fussiness sum-scores range from 6-30.

# All reported regression coefficients are unstandardized B-values (95% confidence intervals) and quantify how the food fussiness score for children of mothers with either 'above average' or 'clinically significant' depression scores differs from children of mothers with 'average or lower' depression scores.

∞ Adjusted for age child when CEBQ was filled out and sociodemographic characteristics (maternal age, household income, maternal ethnic background, maternal educational level, single parenthood, and parity) in the antenatal models and additionally adjusted for mode of delivery, child sex, and child's birth characteristics (birth weight-SDS and gestational age) in the postnatal models.

Abbr. BSI: Brief Symptom Inventory, CEBQ: Children's Eating Behaviour Questionnaire.



## Child picky eating & mental health problems

### **Manuscript based on this chapter:**

Sebastian Cardona Cano, Hans W. Hoek, Daphne van Hoeken, Lisanne M. de Barse, Vincent W.V. Jaddoe, Frank C. Verhulst, Henning Tiemeier. Behavioral outcomes of picky eating in childhood: a prospective study in the general population. *Journal of Child Psychology and Psychiatry*, 2016 (in press)





## Child picky eating and body composition

### **Manuscript based on this chapter:**

Lisanne M. de Barse, Henning Tiemeier, Elisabeth T.M. Leermakers, Trudy Voortman, Vincent W.V. Jaddoe, Lisa R. Edelson, Oscar H. Franco, Pauline W. Jansen. Longitudinal association between preschool fussy eating and body composition at 6 years: The Generation R Study. *International Journal of Behavioral Nutrition and Physical Activity*, 2015, 12, 153)



## Abstract

**Background:** Children's picky eating behavior has been related to both underweight and overweight in cross-sectional studies, but the direction of these associations and the relation with more detailed measures of body composition remains unclear. We aimed to examine whether picky eating at age 4 years is longitudinally related to body mass index (BMI), fat mass index (FMI) and fat-free mass index (FFMI) at 6 years of age.

**Methods:** This study was embedded in Generation R, a population-based, prospective cohort. Data were available for 4,191 children. The Children's Eating Behaviour Questionnaire (CEBQ), administered at age 4 years, was used to derive a picky eating profile. This profile is characterized by high scores on food avoidant scales and low scores on food approach scales. At age 6 years, height and weight were measured at our research center. Body fat and fat-free mass were measured using Dual-energy-X-ray absorptiometry. We used age- and sex-specific standard deviation scores (SDS) for all outcomes.

**Results:** After adjustment for confounders, the picky eating profile was related to lower BMI-SDS ( $B = -0.37$ , 95% CI:  $-0.47$ ;  $-0.26$ ), lower FMI-SDS ( $B = -0.22$ , 95% CI:  $-0.33$ ;  $-0.12$ ) and lower FFMI-SDS ( $B = -0.41$ , 95% CI:  $-0.54$ ;  $-0.29$ ). When adjusting for baseline BMI at 4 years, the picky eating profile predicted a 0.11 lower BMI-SDS at age 6 (95% CI:  $-0.19$ ;  $-0.04$ ). This change in BMI was mainly due to a decrease in FFMI ( $B = -0.19$ , 95% CI:  $-0.29$ ;  $-0.09$ ). Picky eaters also had a higher risk of becoming underweight than non-picky eaters (OR=2.28, 95% CI: 1.34; 3.87).

**Conclusions:** Our findings suggest that young picky eaters are at risk of having a lower fat free mass and of becoming underweight over a 2-year period. This implies that picky eaters may benefit from careful monitoring to prevent an adverse growth development.

## Introduction

Although obesogenic eating behaviors and weight development in childhood have been widely studied,<sup>1</sup> picky eating is, despite its high prevalence, a surprisingly unexplored area. Picky eating – also called ‘fussy’, ‘selective’, or ‘choosy’ eating – is a common phenomenon in preschool-aged children, with prevalence estimates ranging from 14 % to 50%.<sup>2,3</sup> Picky eaters often reject new food items (food neophobia), but they are particularly characterized by their consistent rejection of specific familiar foods,<sup>4</sup> especially vegetables.<sup>5,6</sup> This consistent refusal of specific food items may result in a restricted dietary variety,<sup>4</sup> which could have consequences for a child’s health, growth, and development. It is possible that picky eaters have an insufficient energy intake,<sup>5</sup> resulting in underweight. However, picky eaters may compensate for their limited intake of vegetables and other disliked foods by eating more palatable, energy-dense foods, giving them a relatively high energy intake. Indeed, in a study of 8- to 12-year-old children, picky eaters had a higher preference for fast food than did non-picky eaters,<sup>7</sup> suggesting that they may be at risk of overweight. Both overweight and underweight in childhood have been related to several adverse health outcomes.<sup>8–11</sup> Obese children are more likely to face emotional and social problems<sup>8,9</sup>, and they are at risk of cardiovascular health problems across the life course.<sup>8</sup> Although less pronounced, child underweight is also related to increased psychosocial problems, a poorer quality of life<sup>10</sup> and physical health problems like a relatively low bone mass<sup>11</sup> which may put these children at greater risk of fractures.<sup>12</sup>

Several studies have reported that picky eating in children is associated with a lower body mass index (BMI),<sup>6,13–15</sup> lower body fat percentage,<sup>6</sup> and underweight.<sup>3,13,16,17</sup> However, other studies did not find any association between children’s picky eating and weight status,<sup>18–23</sup> while Finistrella et al. reported that overweight/obese children were more likely to be picky, neophobic eaters than normal weight children.<sup>24</sup> Except for a few studies,<sup>6,23</sup> research has focused on BMI only, and not on other measures of body composition. Distinguishing fat mass from lean mass, however, provides a better insight into children’s overall body composition. For example, picky eaters may have a normal or even low overall body weight, potentially masking relatively high levels of body fat due to a high intake of energy-dense food.<sup>7</sup> Another limitation of most previous studies is their cross-sectional design, in which it is difficult to make causal inferences and therefore, longitudinal research is needed.

In the current study, we aimed to examine the longitudinal association between children’s picky eating at the age of 4 years and body mass at 6 years of age. Further, we aimed to explore whether picky eaters differ from non-picky eaters in height and with respect to their fat mass and lean mass at 6 years. This knowledge

about picky eating in childhood and its impact on weight development and body composition will inform whether preventative intervention strategies are needed for picky eaters.

## Methods

### Study design

This study was embedded in the Generation R Study, a population-based cohort from fetal life onwards.<sup>25</sup> The Generation R Study was designed to identify early biological, environmental, and social determinants of growth, development, and health. Pregnant women living in Rotterdam, the Netherlands, with an expected delivery date between April 2002 and January 2006 were invited to participate. Assessments included physical examinations and parental questionnaires. Written informed consent was obtained from all participating parents and the local Medical Ethical Committee has approved this study. Further information about the study is available elsewhere.<sup>25</sup>

### Participants

Full consent for the postnatal phase of the Generation R Study was obtained for 7,295 children and their parents. Of these, 4,914 children (67%) had available information on their eating behavior. In 4,191 children (85.3%), information on height and weight was available at follow up. Of those, a Dual-energy-X-ray absorptiometry scan was missing in 126 children. Therefore, the population for analysis was 4,191 for analyses with weight-related outcomes and 4,065 for analyses with fat and fat-free mass as outcomes.

### Measures

#### *Children's picky eating*

Eating behavior was assessed with the Children's Eating Behaviour Questionnaire (CEBQ) at 4 years of age.<sup>26</sup> The CEBQ is a validated, multi-dimensional parent-report questionnaire designed to measure differences in children's eating behaviors. The CEBQ consists of 8 scales, each containing 3 to 6 items (in total 35 items). Parents rated the frequency of their children's eating behavior on a Likert scale from 1 (never) to 5 (always). Scale scores were calculated by summing the items if at least 75% were completed. Scale scores were corrected for the number of completed items. Research

has shown that the CEBQ has good psychometric properties in terms of internal reliability, test-retest reliability, and factor structure.<sup>19,26</sup>

In this study, we used a picky eating profile, based on a previously performed latent profile analysis on 5 CEBQ subscales.<sup>27</sup> Children assigned to the picky eating profile were characterized by low scores on food responsiveness and enjoyment of food (food approach behaviors) and high scores on satiety responsiveness, food fussiness, and slowness in eating (food avoidance behaviors).<sup>27</sup> In our analyses, picky eaters were compared with non-picky eaters, i.e. all children who were assigned to another eating profile (avoidant, moderate, responsive, joyful, or approaching eating profile).

For sensitivity analyses, we also included picky eating trajectories based on the Child Behavior Checklist<sup>28</sup> (CBCL) assessed at 1.5 years, 3 years, and 6 years of age, as previously described in detail.<sup>29</sup> The previously created picky eating trajectories<sup>29</sup> were used: 1) never picky eaters, 2) 'remitting' picky eaters: picky eater at 1.5 year and/or at 3 years, but not at 6 years; 3) late-onset picky eaters: only picky eater at 6 years; 4) persistent picky eaters: picky eater at all assessment waves (1.5, 3, and 6 years). More information about the trajectories can be found elsewhere<sup>29</sup> (see also chapter 3.3).

### ***Children's body composition***

Children visited our research center at age 6 years, where trained staff performed several measurements of body composition. Height was measured in standing position using a Harpenden stadiometer and weight was measured without heavy clothing using a mechanical personal scale. Height and weight were used to calculate body mass index (BMI, kg/m<sup>2</sup>). Age- and sex-adjusted standard deviation (SD) scores for height and BMI were calculated using Dutch reference growth curves.<sup>30</sup> Children were classified into underweight, normal weight, overweight, or obese, using international age- and sex-specific cut-offs.<sup>31,32</sup> Body fat mass, bone mass, and lean mass were measured by Dual-energy-X-ray absorptiometry (DXA) scans (iDXA, GE-Lunar, 2008, Madison, WI, USA), using enCORE software v.13.6. Fat mass index (FMI) was calculated as total fat mass (kg) divided by squared height (m<sup>2</sup>). Likewise, children's fat-free mass index (FFMI) was calculated ((sum of bone and lean mass in kg) / height in m<sup>2</sup>). Age- and sex-adjusted standard deviation scores (SDS) for FMI and FFMI were calculated with the residual method in all participating Generation R children who had available data on FMI or FFMI (n=6,491).

### ***Covariates***

Several maternal and child characteristics that may confound the association between children's picky eating and body composition were considered. During pregnancy, a questionnaire was used to assess maternal age. In the same questionnaire, maternal psychiatric symptoms were assessed with the Brief Symptom Inventory

(overall mean score, range: 0-4), a 53-item, validated self-report questionnaire reflecting a diverse spectrum of psychiatric problems.<sup>33,34</sup> Birth weight and child sex were obtained from medical records completed by midwives and gynecologists. In postnatal questionnaires, breastfeeding duration, age of introducing fruit and vegetables, history of any food allergy at age 1 year, maternal educational level, net monthly household income, child ethnic background (based on country of birth of both parents), and children's functional constipation in the year before the 4<sup>th</sup> birthday (based on the Rome II criteria)<sup>35</sup> were assessed. Maternal height and weight were measured by trained staff at the research center (when children were 6 years of age) and were used to calculate maternal BMI (kg/m<sup>2</sup>).

To enhance insight into the directionality of the associations, we accounted for BMI at baseline (age 4 years). Children visited the municipal Child Health Centers around their 4<sup>th</sup> birthday. Height and weight were measured by trained staff as part of a routine health care program. Similar to anthropometrics at age 6 years, BMI SD-scores for age and sex were calculated using Dutch reference scores.<sup>30</sup>

## Statistical analyses

To determine whether picky eaters differed in their body composition from non-picky eaters, we performed separate linear regression analyses with the picky eating profile (picky eaters vs. non-picky eaters) as the exposure and BMI-SDS, FMI-SDS, and FFMI-SDS as outcomes. Next, we performed a multinomial logistic regression analysis to assess whether picky eaters had a higher risk of being underweight, overweight, or obese (reference group: normal weight) than non-eaters. All analyses were adjusted for potential confounders that changed the effect estimates by 5% or more<sup>36</sup> (only food allergies did not reach this criterion and was thus left out of the analyses). In separate models, we adjusted the analyses for children's BMI-SDS at baseline (age 4 years) to assess whether picky eating behavior at 4 years predicted change in body composition measures at age 6 years.

Several sensitivity analyses were performed. First, the associations of the different picky eating trajectories from ages 1,5 to 6 years with BMI-SDS, FMI-SDS, and FFMI-SDS were examined with linear regression analyses, controlling for potential confounders and baseline BMI. Second, we checked the association between children's picky eating profile and height (age- and sex-specific SD-scores), adjusting for potential confounders. In a separate model, we adjusted this analysis for children's height at baseline.

Missing values on covariates were estimated using multiple imputation techniques.<sup>37</sup> All statistical analyses were performed with SPSS 20.1.

# Results

## Population characteristics

Of all included children, 5.7% were assigned to the picky eating profile (Table 3.4.1). Most children had a Dutch ethnic background (66.0%) and had mothers with a relatively high education (higher vocational training or academic degree, 63.7%).

**TABLE 3.4.1 General characteristics of 4,191 children included in the study**

POPULATION CHARACTERISTICS		N	PERCENTAGE, MEAN (SD), OR MEDIAN (IQR) <sup>#</sup>
Child sex	% boy	2,085	49.7
Birth weight	mean grams	4,189	3,441 (565)
Child ethnic background	% Dutch	2,768	66.0
	% Moroccan	136	3.2
	% Surinamese & Dutch Antillean	305	7.3
	% Turkish	243	5.8
	% other, Western (mainly European)	394	9.4
	% other, non-Western	345	8.2
Breastfeeding duration	median months	4,191	3.5 (7.0)
Introduction of fruits and vegetables	% <3 months	265	6.3
	% 3-6 months	3,477	83.0
	% >6 months	449	10.7
Functional constipation at age 4 years	% yes <sup>§</sup>	616	14.7
Picky eating profile	% picky eater	240	5.7
Child BMI age 4 years	mean SDS	4,191	0.1 (0.9)
Child BMI age 6 years	mean SDS	4,191	0.2 (0.9)
Maternal age	mean years	4,191	31.6 (4.6)
Maternal educational level	% low <sup>£</sup>	374	8.9
	% medium <sup>£</sup>	1,148	27.4
	% high <sup>£</sup>	2,669	63.7
Household income per month	median in €	4,191	3,600 (2,600)
Maternal BMI	median body mass index	4,191	24.1 (5.6)
Maternal psychiatric symptoms	median score	4,191	0.1 (0.2)

<sup>#</sup> Values are percentages for categorical variables, means (standard deviations) for continuous normally distributed variables and medians (interquartile ranges) for continuous non-normally distributed variables, derived from the imputed dataset.

<sup>§</sup> Children who had had less than 2 bowel movements per week or predominantly hard feces for at least 2 successive weeks were classified as functional constipated.

<sup>£</sup> Low education: ranging from no education up to high school, medium: lower vocational training, high: higher vocational education and higher academic education.

## Picky eating and body composition

The associations between the picky eating profile and body composition measures are presented in Table 3.4.2). Children classified as picky eaters had a 0.37 lower BMI SD-score at age 6 years than other children (95% CI: -0.47; -0.26). Picky eaters also had a 0.22 lower FMI-SDS (95% CI: -0.33; -0.12) and a 0.41 lower FFMI-SDS (95% CI: -0.54; -0.29). After adjustment for BMI at age 4 years, the effect estimates attenuated, but picky eating remained significantly associated with a lower BMI SD-score ( $B = -0.11$ , 95% CI: -0.19; -0.04) and with a lower FFMI-SD-score at age 6 years ( $B = -0.19$ , 95% CI: -0.29; -0.09). Similarly, sensitivity analyses with the picky trajectories showed that persistent picky eaters – but not remittent or late onset picky eaters – had a lower BMI and FFMI after correcting for baseline BMI (Supplementary Table 3.4.1).

Additional sensitivity analyses revealed that picky eaters were also shorter than non-picky eaters (adjusted  $B = -0.26$ , 95% CI: -0.39; -0.14), but this effect estimate attenuated toward null when we adjusted for height at age 4 years (data not shown), implying that picky eating did not predict less height growth over this 2-year period.

**TABLE 3.4.2 Child picky eater profile at 4 years of age and body composition at 4 years and 6 years of age**

PICKY EATING PROFILE AT 4 YEARS	BODY COMPOSITION AT 4 YEARS B (95% CI) <sup>#</sup>		BODY COMPOSITION AT 6 YEARS B (95% CI) <sup>#</sup>	
	BODY MASS INDEX-SDS N=4,191	BODY MASS INDEX-SDS N=4,191	FAT MASS INDEX-SDS N=4,065	FAT FREE MASS INDEX-SDS N=4,065

**Model 1: adjusted for potential confounders<sup>†</sup>**

Picky eater profile vs non-picky eater profile	-0.38 (-0.50; -0.25)***	-0.37 (-0.47; -0.26)***	-0.22 (-0.33; -0.12)***	-0.41 (-0.54; -0.29)***
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**Model 2: additionally adjusted for BMI at age 4<sup>††</sup>**

Picky eater profile vs non-picky eater profile	–	-0.11 (-0.19; -0.04)**	-0.02 (-0.11; 0.07)	-0.19 (-0.29; -0.09)***
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# Values are regression coefficients (95% confidence intervals).

\*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . All body composition outcomes are age- and sex- adjusted standard deviation scores.

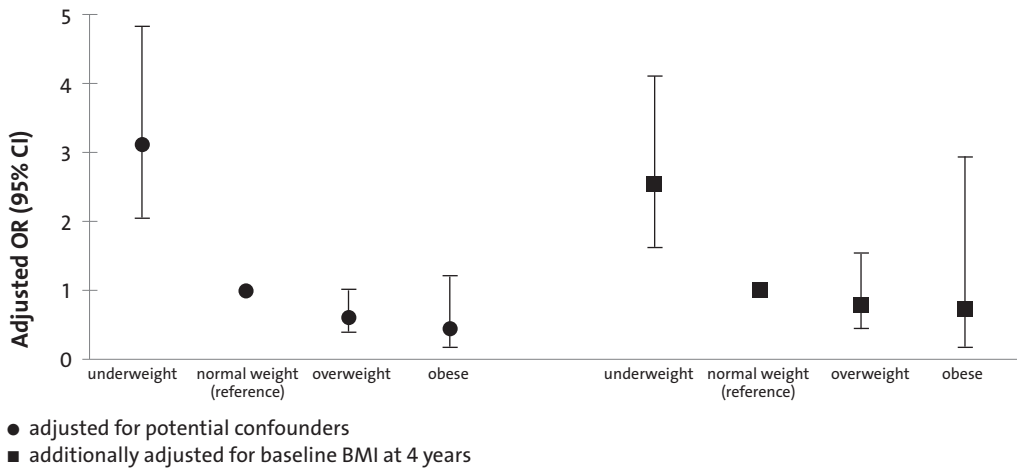
† Model 1: adjusted for potential confounders: maternal age, educational level, BMI, and psychiatric symptoms during pregnancy; household income; child ethnic background, sex, age when CEBQ was filled out, birth weight-SDS, and functional constipation at age 4 years; breastfeeding duration, and age of introducing fruit and vegetables.

†† Model 2: model 1 + additionally adjusted for children's BMI at age 4 years.

## Picky eating and the risk of underweight, overweight, or obesity

Picky eaters had a greater risk of being underweight at age 6 years than non-picky eaters, even after adjustment for baseline BMI at 4 years (OR=2.28, 95% CI: 1.34; 3.87) (Figure 3.4.1 and Supplementary Table 3.4.2). Picky eaters were not at risk of being overweight or obese.

**FIGURE 3.4.1** Child picky eater profile at age 4 years and risk of being underweight, overweight, or obese at 6 years



## Discussion

Using a longitudinal design within a large population-based cohort, picky eating at 4 years of age predicted a lower BMI at age 6. In addition, preschoolers with a picky eating pattern were at risk of developing underweight over 2 years' time. The analyses disentangling the different components of body composition indicated that the lower BMI of picky eaters is mainly explained by a lower fat free mass.

The finding that picky eating was associated with underweight in children is in line with previous studies that also found a lower body fat percentage<sup>6</sup> and lower weight status among picky eaters.<sup>3,6,13-16</sup> However, several other studies did not find any association with weight status<sup>18-23</sup> and some even reported that picky eating was related to overweight.<sup>24,38</sup> This discrepancy in findings might indicate that picky eaters are a heterogeneous group of children who differ, for instance, in the severity of their picky eating behavior. Some children may exhibit "severe" picky eating behavior, either in terms of chronicity or the nature of their behavior. The latter might



be applicable for our group of picky eaters, as they not only scored high on the CE-BQ's food fussiness scale, but also on other avoidant eating behaviors: they got full more easily, were slower eaters, and did not enjoy eating as much as other children.<sup>27</sup> Possibly, the combination of these behaviors reflects a more severe picky eating pattern that may result in underweight. The low prevalence of this picky eating profile in our sample (5.7%) underlines that these children form a distinct group of picky eaters. Further support for this reasoning comes from our analyses with the picky eating trajectories and from another longitudinal study<sup>3</sup> both showing that only *persistent* picky eaters, which could be perceived as severe picky eaters in terms of chronicity, were at risk of a lower BMI and underweight. In contrast, children who were depicted as picky eaters at only 1 or 2 time points had a rather normal weight development. This fits a broader developmental perspective that a period of picky eating during toddlerhood can be a normal developmental phase, not necessarily warranting clinical attention. However, clinicians and parents should pay attention to severe forms of picky eating behavior as it may have consequences for weight development, or it could be an indicator of further developmental problems, as picky eating and sensory sensitivity are also often prevalent in children with pervasive developmental disorders.<sup>39</sup>

In addition to the existing literature that comprises mostly cross-sectional research, our study adds to the discussion about the direction of the association between picky eating and body composition. Due to the cross-sectional design of most previous studies,<sup>3,6,13,14,19,20</sup> it was debated whether children's picky eating behaviors influence weight status or the reverse. In response to this discussion, Jaarsveld, Llewelyn, Johnson and Wardle<sup>40</sup> were among the first to test bidirectional associations between eating behaviors that are captured in our picky eating profile (e.g. slowness in eating, satiety responsiveness) and weight in infancy. They concluded that the pathway of eating behaviors influencing weight was the strongest, though the reverse pathway from weight to eating behaviors was not completely absent. Although we could not test bidirectional associations, the availability of repeated measurements of child BMI enabled us to adjust the analyses for BMI at baseline. Consistent with the study of Jaarsveld et al.,<sup>40</sup> our results also suggest that picky eating at age 4 predicts a significant decrease in children's BMI and – more specifically – fat-free mass over the next 2 years. However, from the current study, we cannot make inferences about the directionality of the association in the first 4 years of life. Picky eaters had a lower BMI at baseline (age 4 years), so it is possible that children's weight in infancy or toddlerhood may already have influenced their eating behavior, for instance through effects on parental feeding practices. Jansen et al. previously showed that a relatively low BMI in children elicited pressuring feeding behaviors in parents.<sup>41</sup> While parents may intend to increase their children's food

intake, pressure to eat could have the opposite effect. Pressuring feeding strategies may induce negative reactions in children toward foods,<sup>42</sup> thereby exacerbating or contributing to the development of picky eating. Future studies should explore the pathway from body composition to picky eating, using repeated measurements and taking parents' feeding strategies into account as a possible mediator.

Picky eating was related to different aspects of body composition. Although we carefully adjusted the analyses for numerous potential confounding factors, there is still a possibility that the association is flawed by residual confounding, for instance due to potential measurement error or misclassification of our confounders.<sup>43</sup> However, assuming that our findings represent at least partly a true association, picky eating seems to have an overall effect of on growth. The strong association of picky eating with a lower fat-free mass suggests that picky eating is associated with a lower muscle mass which is worrisome given that a higher muscle mass and muscle strength are considered to have a beneficial effect on metabolic and cardiovascular health.<sup>44-46</sup> The potential adverse effects of picky eating on health are also underlined by our finding that picky eaters were at risk of developing underweight over time. We hypothesize that this overall effect on growth development is due to an insufficient energy intake or a relatively poor diet quality among picky eaters. Unfortunately, we were not able to test this hypothesis as we lacked data on food intake between 4 and 6 years of age. However, a poor overall diet quality is likely<sup>47</sup> given the restricted diet variety that picky eaters have.<sup>4</sup>

The current study was strengthened by its population-based, prospective design with multiple assessments of BMI, which allowed us to examine the longitudinal associations between picky eating and body composition. Another strength was the inclusion of detailed body composition measures. While most previous studies focused on body mass only, we distinguished fat mass from fat-free mass. One of the studies that also included fat and fat-free mass used skinfold thickness<sup>23</sup> which is a less reliable method than the DXA measurements that we used.<sup>48</sup> Ideally, multiple DXA measurements would have been able to account for baseline fat and fat free mass. We also lacked information on concurrent food intake of children which would be interesting to assess in future studies as a potential mediating factor in the association between picky eating and body composition. Another possible limitation is that picky eating was measured by parent report, and parents of smaller children might be more likely to perceive their children to be picky eaters. However, we addressed this by controlling for baseline BMI at 4 years. Finally, as with all cohort studies, selective follow up is a potential limitation. In the Generation R Study, loss to follow up is higher in those from low socioeconomic status and non-Western origin,<sup>25</sup> which to some extent, limits generalizability of the results to the general population.

## Conclusions

Although picky eating could be considered as a normal phase of development,<sup>29,39</sup> our findings highlight the possible adverse effect of more severe picky eating on healthy growth. Young picky eaters are at risk of developing underweight and a relatively low fat-free mass. Health care practitioners should carefully monitor picky eaters and their dietary quality, particularly children who not only reject certain types of foods, but also show a pattern of slow, joyless eating of foods. In their advice to parents, it is important that health care practitioners show understanding for parents' concerns, frustrations, and possible adverse – but imaginable – reactions to their children's picky eating behavior. Although coercive and pressuring feeding strategies are understandable, the possible counterproductive effects should be explained (i.e. more food refusal, negative atmosphere).<sup>49,50</sup> There is no golden standard for overcoming picky eating yet, but research suggests that repeated exposure to a diversity of food items without coercion of eating is key for food acceptance.<sup>49</sup> Moreover, it is advised to encourage parents to cook<sup>51</sup> and eat<sup>49</sup> together with their children, so that parents act as a role model and children can imitate their parents' eating behaviors.<sup>49</sup> Although these strategies may improve food intake<sup>51</sup> and food enjoyment<sup>50</sup> of children, more research is needed to study the effectiveness for such interventions in picky eaters. Future studies should also follow picky eaters over a longer period of time to examine whether picky eating also has long-term adverse consequences for body composition development and related health outcomes.

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## Supplement

**SUPPLEMENTARY TABLE 3.4.1 Child picky eating trajectories and body composition at 6 years of age**

PICKY EATING TRAJECTORIES	BODY COMPOSITION AT 6 YEARS B (95% CI) <sup>#</sup>		
	BODY MASS INDEX –SDS N=3,094	FAT MASS INDEX-SDS N=3,007	FAT FREE MASS INDEX-SDS N=3,007
	<b>Model 1: adjusted for potential confounders<sup>†</sup> and for baseline BMI<sup>††</sup></b>		
Never picky eating	0 [reference]	0 [reference]	0 [reference]
Remitting picky eating (onset at 1.5 or 3 years, but not persisting)	-0.03 (-0.08; 0.01)	-0.04 (-0.09; 0.01)	-0.04 (-0.10; 0.02)
Late onset picky eating (at 6 years)	-0.04 (-0.15; 0.07)	0.00 (-0.12; 0.12)	-0.11 (-0.26; 0.04)
Persistent picky eating (from 1.5 years till 6 years)	-0.39 (-0.51; -0.26) <sup>***</sup>	-0.29 (-0.42; -0.16) <sup>***</sup>	-0.32 (-0.47; -0.17) <sup>***</sup>

\*\*\* p<0.001.  
<sup>#</sup> Values are regression coefficients (95% confidence intervals). All body composition outcomes are age- and sex-adjusted standard deviation scores.  
<sup>†</sup> Adjusted for potential confounders: maternal age, educational level, BMI, and psychiatric symptoms during pregnancy; household income; child ethnic background, sex, age when CEBQ was filled out, birth weight-SDS, and functional constipation at age 4 years; breastfeeding duration, and age of introducing fruit and vegetables.  
<sup>††</sup> Additionally adjusted for baseline BMI. For remitting picky eating, we adjusted for baseline BMI at 1.3years and at 3 years. For late onset picky eating, we adjusted for baseline BMI at 4 years. For persistent picky eating, we adjusted for baseline BMI at 1.3years.

**SUPPLEMENTARY TABLE 3.4.2 Child picky eater profile and risk of being underweight, overweight, or obese**

PICKY EATING PROFILE AT 4 YEARS	OR (95% CI) FOR RISK OF BEING UNDERWEIGHT, OVERWEIGHT, OR OBESE <sup>#</sup>			
	UNDERWEIGHT N=212 (5.1%)	NORMAL WEIGHT N=3,401 (81.3%)	OVERWEIGHT N=454 (10.9%)	OBESE N=116 (2.8%)
<b>Model 0: unadjusted</b>				
Picky eater profile vs non-picky eater profile	2.98 (1.98; 4.48) <sup>***</sup>	1 [reference]	0.76 (0.47; 1.23)	0.62 (0.23; 1.70)
<b>Model 1: adjusted for potential confounders<sup>†</sup></b>				
Picky eater profile vs non-picky eater profile	3.16 (2.05; 4.88) <sup>***</sup>	1 [reference]	0.62 (0.37; 1.03)	0.43 (0.15; 1.23)
<b>Model 2: additionally adjusted for BMI at age 4<sup>††</sup></b>				
Picky eater profile vs non-picky eater profile	2.28 (1.34; 3.87) <sup>**</sup>	1 [reference]	0.82 (0.44; 1.55)	0.73 (0.18; 2.96)

\*\* p<0.01, \*\*\*p<0.001.  
<sup>#</sup> Values are odds ratios (95% confidence intervals).  
<sup>†</sup> Model 1: adjusted for potential confounders: maternal age, educational level, BMI, and psychiatric symptoms during pregnancy; household income; child ethnic background, sex, age when CEBQ was filled out, birth weight-SDS, and functional constipation at age 4 years; breastfeeding duration, and age of introducing fruit and vegetables.  
<sup>††</sup> Model 2: model 1 + additionally adjusted for children's BMI at age 4 years.



CHAPTER 4

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## General discussion





## Picky eating

### Epidemiological studies of risk factors and consequences of child picky eating

In chapters 3.1 and 3.2, we examined risk factors – such as infant feeding and parental anxiety or depression – of picky eating in childhood. Higher maternal emotional problems during pregnancy and 3 years later were prospectively and independently related to child picky eating (chapter 3.2). Our findings are largely consistent with previous research.<sup>1,7</sup> For instance, in a clinical sample, children with a feeding disorder more often had mothers with high levels of emotional problems than children without eating problems.<sup>2</sup> However, it was not clear whether the child's eating problems caused stress and psychiatric symptoms in their mothers or whether mothers' symptoms predicted child eating problems. Importantly, we showed that mothers' antenatal symptoms of anxiety and depression predicted 4-year-olds' picky eating independent of mothers' symptoms at 3 years postnatal. This suggests that the direction of the associations with mothers' antenatal symptoms is from mother to child. Moreover, with our population-based approach, we also showed that not only clinically significant anxiety of mothers has an effect on child picky eating, but also slightly elevated anxiety symptoms. In addition, we found indications that fathers' emotional problems were related to child picky eating, while most studies focused on mothers' anxiety and depression. It has been suggested that picky eating is an expression of emotional problems in the child,<sup>8</sup> which could explain the relation between parents' emotional problems and child picky eating. However, in chapter 3.3 we did not observe an association between children's picky eating and their emotional problems. Thus, other factors such as parenting or feeding practices are more likely to explain the relation between parents' emotional problems and children's picky eating.

Another factor that could be important for picky eating is infant feeding. A longer breastfeeding duration repeatedly exposes the child to a variety of flavors – reflecting mothers' diet – which can enhance initial acceptance of solid foods.<sup>9,10</sup> Therefore, we hypothesized that a longer breastfeeding duration is also related to acceptance of food later in childhood and thus to less picky eating (chapter 3.1). Given the proposed mechanism of repeated flavor exposure, we expected the strongest contrast between children who were never breastfed and those with the longest, recommended breastfeeding duration ( $\geq 6$  months). In contrast to our hypothesis, however, never breastfed children did not differ in picky eating from those breastfed for 6 months or longer. Therefore, our observed association between a short breast-

feeding duration (between 1 day and 2 months) and higher picky eating scores is not likely to be explained by repeated exposure to various flavors. We proposed that this finding is explained by reverse causation. That is, infants who become picky eaters in toddlerhood may already have been difficult eaters in infancy,<sup>11</sup> potentially inducing their mothers to stop breastfeeding early on. Together, these results suggests that breastfeeding duration does not predict picky eating in 4-year-old children. This may seem to contrast earlier findings reporting an association between a longer breastfeeding duration and higher *initial* food acceptance<sup>9</sup> – i.e. at the start of complementary feeding – but this could indicate that flavor exposure through breastmilk particularly facilitates these initial food acceptances, but is less important when children have already been exposed to solids for a longer time<sup>12,13</sup> as was the case for our 4-year-olds. In addition, breastfeeding may be less important for picky eating than the timing of complementary feeding. Indeed, we particularly observed that children to whom vegetables were introduced early on had lower levels of picky eating behavior than children introduced to vegetables from 6 months on. Introducing vegetables between 4 and 5 months may be protective against picky eating.

Not only did we study risk factors for picky eating, we also studied possible health consequences (chapter 3.4). As picky eating could be a normal phase of development,<sup>14,15</sup> we used a picky eating profile that may reflect more severe picky eating. This profile was not only characterized by high food fussiness scores, but also by slow eating, getting full easily, and low enjoyment of food.<sup>16</sup> We examined the longitudinal associations between the picky eating profile and body composition. Picky eaters at age 4 years had a lower BMI at age 6 years, irrespective of the child's BMI at age 4. This change in BMI was mainly explained by a lower fat free mass. In addition, these picky eaters were also at risk of developing underweight over a two-year period. The low prevalence of this picky eating profile in our sample (5.7%) could indicate that these children form a distinct group of picky eaters. Further support for this reasoning comes from our analyses with the picky eating trajectories and from another longitudinal study<sup>17</sup> both showing that only *persistent* picky eaters were at risk of a lower BMI and underweight. In contrast, children who were depicted as picky eaters at only one or two time points had a rather normal weight development.

Similar to our findings with weight status, persistent picky eaters – but not transient picky eaters – had more pervasive developmental problems at the age of 6 years (as reported by teachers, chapter 3.3). Nevertheless, persistent picky eating was not related to teacher-reported emotional problems. This is in contrast with other studies,<sup>18-20</sup> but those studies may have suffered from shared method variance bias as mothers reported on both picky eating and emotional problems, while we

used teacher reports of emotional problems. Indeed, we found that picky eating was related to mother reported emotional problems, but not to teacher reported problems, suggesting bias in mothers' reports. Alternatively, differences in findings could also fit with the heterogeneous profile of picky eating – with some picky eaters being at risk of emotional problems, others of weight problems, and others just developing normally.

## The chicken or the egg dilemma: What came first?

To determine causality, it is a necessary condition to establish temporal relations.<sup>21</sup> Prospective cohort studies like the Generation R Study are well-designed to establish temporal associations, and in the studies described in this thesis, we aimed to identify temporality to further elucidate a potential causal association.

A difficulty in establishing temporality is that an association between a certain exposure and a certain outcome measured after the exposure does not necessarily imply a temporal association. For instance, we found that picky eating measured at age 4 years was related to a lower BMI at age 6 years (chapter 3.4). Even though the exposure is measured 2 years before the outcome, I would argue that this reflects a cross-sectional association, as we cannot infer whether picky eating induces change in BMI. To answer this question, we controlled for BMI at 4 years, observing an association between picky eating at 4 years and a lower BMI and FFMI at 6 years, thereby showing that 4-year-olds' picky eating was related to change in body composition in a 2-year-period. The same approach – i.e. controlling for baseline problems – was applied in chapter 3.1, as the association between a short breastfeeding duration (between 1 day and 2 months) and higher picky eating scores may be explained by reverse causation. However, it is not well known which initial feeding problems – that could have led to a shorter duration of breastfeeding – develop into picky eating in childhood. Moreover, baseline feeding problems were assessed with a few single items (refusal of breastfeeding, drinking slow or small quantities, and general feeding problems) instead of using validated questionnaires such as the infant version of the CEBO,<sup>22</sup> limiting the extent to which we could address reverse causation.

Controlling for baseline problems was not our only approach in attempting to establish temporal associations. In the chapters concerning maternal psychopathology, we deliberately studied psychiatric symptoms before the child was born. For instance, in chapter 3.2, we evaluated whether maternal anxiety or depression could be a risk factor for picky eating. However, picky eating may also induce stress in parents. Studying maternal symptoms during pregnancy or earlier could be helpful in the establishment of temporality, as picky eating in early childhood cannot be an antecedent of maternal symptoms during pregnancy.

Controlling for baseline problems could provide insight in the temporality of associations, but it does not answer the chicken or egg dilemma: what came first? For the relation between maternal emotional symptoms during pregnancy and picky eating at 4 years, it is clear what came first. However, this does not rule out bi-directional associations between maternal symptoms during the child's life and child picky eating. Likewise, our research design in chapter 3.4 did not answer the question about the directionality of the association between picky eating and BMI. Picky eaters had a lower BMI at baseline (age 4 years), so it is possible that children's weight in infancy or toddlerhood may already have influenced their eating behavior. I do not expect that a lower BMI has a direct effect on children's picky eating, but rather by parental pressure. Jansen et al. have already shown that the pathway from a lower BMI in children to parental pressure was stronger than the reverse.<sup>23</sup> Future studies should therefore specifically assess the bi-directionality between parental pressuring feeding and child picky eating.

To attempt causality in observational studies, it is not only important to establish temporality and bi-directionality, but also to address potential confounding. Although we carefully adjusted all analyses described in this thesis for numerous potential confounders, there is still a possibility that the associations were flawed by residual confounding, for instance due to potential measurement error, categorization of our confounders or misclassification of our confounders.<sup>24</sup> At the baseline assessment of household income, the multiple-choice answering categories ranged from 450 euro to more than 2200 euro per month, creating a ceiling effect (i.e. many participants fell into the highest category) and this may have impaired how well we could control for socioeconomic differences. Moreover, in Generation R, income is expressed as net household income, without correcting for number of persons in the household and related economies of scale.<sup>25</sup> We followed this approach, as the main scope of this thesis was not socioeconomic differences. However, future researchers studying socioeconomic differences in health and behavior in the Generation R Study should consider equivalence scales that take into account the number of adult and child family members and their age, as this could change the ranking in income.<sup>26</sup> Moreover, educational level and household income may not have captured all socioeconomic differences. For instance, stocks, savings, and housing could also account for such differences. In addition to socioeconomic differences, differences in social capital<sup>27</sup> may also have confounded our associations. For instance, our finding that a short breastfeeding duration (between 1 day and 2 months) was related to higher levels of picky eating (chapter 3.1) may be attributed to social capital. To illustrate this, mothers who are embedded in social networks in which breastfeeding is not encouraged may also be less likely to count on others' support when encountering issues in child rais-

ing such as child food refusals. Including measures of social capital could improve future epidemiologic research of eating behavior.

Residual confounding could also have occurred if we missed important confounders. Particularly considering that picky eating is a relatively unexplored research area, it is possible that knowledge is lacking about factors related to picky eating for which we otherwise would have adjusted. For instance, it is not well known whether maternal controlling feeding practices might lead to picky eating or are more likely to be a result of children's picky eating. Also, we lacked information about controlling feeding practices during the first year of life. Information about parental picky eating was not assessed either. It may well be that picky parents have confounded the association with early vegetable introduction and low levels of child picky eating. Parents who are picky eaters themselves may be less likely to introduce vegetables and other disliked food to their child early on.

Thus, future research should continue to study predictors of picky eating, unravel the direction of the association between picky eating and parental controlling feeding, and consider parental picky eating as potential confounder in the association between timing of complementary feeding and child picky eating. Future epidemiologic studies of eating problems could also include measures of social capital. In addition, the use of validated questionnaires to measure initial feeding problems is highly recommended to study whether and which feeding problems may lead to picky eating, which could be used to control for baseline eating problems to establish temporality. A similar cohort as Generation R is currently designed in Rotterdam and I recommend to assess the Baby Eating Behaviour Questionnaire<sup>22</sup> in the first months of life.

## A health-care perspective on picky eating

The high prevalence of picky eating during childhood and the high rate of remission indicates that picky eating might be part of normal development.<sup>14</sup> This is also supported by our finding that picky eaters – even persistent picky eaters – were not at risk of emotional or behavior problems. However, our findings highlight the possible adverse effect of more severe picky eating on body composition. Differences in severity of picky eating might explain why some studies found an association between picky eating and weight status, and others not.<sup>28</sup> As severe picky eaters in our study were at risk of developing underweight and lower fat-free mass, this behavior could also indicate the development of an avoidant/restrictive feeding disorder (ARFID). Children who avoid food intake because of extreme sensitivity to certain senses of food such as smell, texture, and taste – as occurs in picky eating – and who therefore not achieve their energy and nutritional needs resulting in for instance underweight, fulfill important criteria for a diagnosis of ARFID.<sup>29</sup>

Health care practitioners could more carefully monitor picky eaters and their dietary quality, particularly children who not only reject certain types of foods, but also show a pattern of slow, joyless eating. Although picky eating could be an indicator of further developmental problems – as picky eating and sensory sensitivity are also often prevalent in children with autism spectrum disorders (ASD),<sup>15,30</sup> – it is questionable whether picky eating could play a role in the early detection of autism. In the general population, we showed that children who were picky eaters from 1.5 years to 6 years on had higher pervasive developmental problems at 6 years, even when adjusted for baseline problems at 1.5 years. This may indicate that mothers do not recognize pervasive developmental problems at 1.5 years well – supported by Maenner et al. reporting that the median age of ASD diagnose was around 5 years<sup>31</sup> – while picky eating is easily recognized by parents in young childhood. However, given the high prevalence of picky eating at young ages, and the low absolute risk of autism, it remains unclear whether picky eating may aid health care practitioners in the early detection of ASD. Even more caution is warranted, as the persistent picky eating trajectory we described, overlaps with the timing of the outcome under study (6 years). Therefore, it is needed to study whether severe picky eating at a younger age predicts an early ASD diagnosis, which may benefit an early start of intensive behavior intervention. A meta-analysis has shown that early intensive behavior intervention based on applied behavior analyses may improve cognitive functioning and adaptive behaviors.<sup>32</sup>

When parents visit health care practitioners because of their children's picky eating, it is important that health care practitioners address parents' reactions to their children's picky behavior. Parents may engage in coercive and pressuring feeding practices, which are understandable but may have counterproductive effects (e.g. more food refusal)<sup>33-35</sup> of which parents need to be informed. However, researchers also need to address better whether pressure to eat indeed elicits picky eating, or whether coercive feeding strategies are more likely to be a reaction to the child's picky eating.<sup>35</sup> There are no guidelines for overcoming picky eating yet, but studies suggest that parents should cook and eat healthy foods together with their children,<sup>33,36,37</sup> so that parents act as a role model and children can imitate their parents' eating behaviors.<sup>33,37,38</sup> Moreover, repeated exposure to a diversity of food items without coercion of eating is key for food acceptance.<sup>33</sup> Our findings also highlight that early vegetable exposure may have beneficial effects on the prevention of picky eating. Vegetables could be introduced from 4 months on as included in the Dutch recommendations, although the World Health Organization recommends to wait for 6 months before introducing solids to the child diets. Before making any further feeding recommendations, future studies should investigate the potential impact of introducing vegetables between 4 and 5 months of age on dietary quality and other

health outcomes. In addition, our findings need to be replicated, accounting for reverse causation as well as potential confounding bias (for instance due to parents' own picky eating).

## Picky eating & eating disorders

It has been hypothesized that child picky eating is a risk factor for later eating disorders. Marchi and Cohen reported that picky eating during childhood predicted anorexic symptoms in adolescence.<sup>39</sup> This is supported by Kotler et al. who found that particularly struggles around food, unpleasant meals, and eating conflicts in childhood were related to anorexia nervosa (AN).<sup>40</sup> Other studies, as reviewed by Micali et al., showing an association between early feeding or eating problems and eating disorders mostly had a retrospective design, and may have been prone to recall bias.<sup>41</sup> Recall bias occurs when the accuracy of recalling earlier exposures is different for cases and controls.<sup>21</sup> For instance, eating disorder patients or their mothers might be more likely to search their memory thoroughly for factors that could have elicited the eating disorder and therefore be more likely to recall early feeding or eating problems. Thus, cases might over-report early feeding and eating problems, or controls might underreport these problems, both leading to an inflated association between early eating problems and later eating disorders.

Given the likelihood of recall bias in these retrospective studies, it cannot be concluded that picky eating is a risk factor for eating disorders. In a cross-sectional study of 8- to 12-year old children, picky eating was not related to eating psychopathology such as dieting, binge-eating, and feeling too fat,<sup>20</sup> maybe because the food refusal of picky eaters are based on the sensory profiles of food and do not reflect efforts to lose weight. Moreover, in a prospective study, picky eating in early childhood was even related to be less preoccupied to be thin or to experience less social pressure to be thin.<sup>42</sup> Prospective studies should elucidate whether picky eaters are at risk of eating disorders or not.



## Maternal history of eating disorders

### Epidemiological studies on the intergenerational transmission of eating disorder psychopathology

In this thesis, we found that the children of mothers with a history of eating disorders are at risk of disturbances in their emotional development from a young age onwards. Already at 4 years of age, the children of mothers with a history of eating disorders children had a tendency towards overeating in response to emotional cues (chapter 2.2). In addition, children whose mother suffered from eating disorders had higher levels of emotional problems at 6 years of age (chapter 2.3). This confirmed findings from clinical studies showing increased emotional problems among children of mothers with eating disorders.<sup>43</sup> Our findings could be indicative of a general vulnerability for psychopathology or a specific 'marker' of eating disorder risk. Symptoms of anxiety and depression in the child may underlie the higher levels of emotional eating<sup>44</sup> and could also precede eating psychopathology later in life.<sup>45,46</sup>

The specific effect of maternal eating disorders on child emotional eating and emotional problems rather than on behavior problems and other eating behaviors (chapter 2.2 & chapter 2.3) might be explained by genetic factors,<sup>43,47</sup> but could also indicate modeling of mother's behaviors. For instance, mothers with a history of both eating and emotional problems might have a low self-esteem and have a ruminating coping style which they may pass on to their children, as children often copy their mothers' reaction to negative life events.<sup>48</sup> Likewise, if mothers with a history of eating disorders continue to be emotional eaters, their children may learn to cope with negative emotions by eating. Support for modelling of emotional eating comes from a community sample in which mothers with higher levels of eating psychopathology reported that their children imitated eating behaviors, which were not intentionally modelled.<sup>49</sup> Emotional overeating may also be enhanced by mothers' feeding practices when mothers with a history of eating disorders use food to soothe.<sup>43</sup> Indeed, emotional eating occurs more often when mothers use emotional feeding practices.<sup>50,51</sup> It is also important to note that we did not find a tendency towards emotional undereating in the children of mothers with a history of eating disorders (chapter 2.2). Although we hypothesized that women with an eating disorder eat less when distressed, they may eat less in general, and therefore, their children might be at risk of dieting behavior, but not of undereating in response to emotions.

Also, differences in feeding practices were observed between mothers with and without a history of eating disorders. Mothers with a history of eating disorders were slightly less likely to initiate breastfeeding (chapter 2.1) and to pressure

their pre-school-aged children to eat (chapter 2.2) than mothers without such a history, although we found no differences regarding restrictive feeding or monitoring (chapter 2.2). Perhaps, monitoring what and how much a child eats is a very normal parenting behavior when children are young.<sup>52</sup> This could explain why we did not find any differences and why monitoring did not predict child weight either in our and other cohorts.<sup>23,37,53</sup> It is also possible that our assessment of monitoring<sup>54</sup> is prone to reporter bias, as many mothers of young children may not be willing to answer that they hardly keep track of their children's intake of sweets, snacks, and high-fat foods. If monitoring unhealthy food intake of young children is either very normal parenting behavior or highly over-reported by mothers, this could have impaired the variation that is needed to observe associations.

The lower tendency of breastfeeding – although not statistically significant – among mothers with a history of eating disorders in our cohort is in line with some studies,<sup>43,55-58</sup> but contradicts the results from the ALSPAC Study showing a higher initiation and duration of breastfeeding in mothers with a history of eating disorders.<sup>59</sup> These conflicting results may be explained by the same underlying eating disorder psychopathology, which results in a longer breastfeeding duration in some women, but in a lower initiation and shorter duration of breastfeeding in others. Mothers with a history of eating disorders who are particularly embarrassed about their body may be hesitant to start breastfeeding or give up breastfeeding early on,<sup>43</sup> while mothers who are more focused on losing weight and who are aware of the belief that breastfeeding promotes weight loss may continue breastfeeding.<sup>60</sup> Similarly, due to fears of gaining weight, mothers with a history of eating disorders may be concerned about their children's weight as well<sup>60,61</sup> and therefore less likely to pressure their children to finish all the food on their plate. An alternative explanation is that women with a history of eating disorders experienced much tension and pressure in food-related situations at the time they had an eating disorder, making them hesitant to interfere with their children's eating habits. The lower tendency of pressuring feeding is in line with the findings of another population-based study.<sup>62</sup> However, in smaller samples, mothers' eating disorder psychopathology was related to more pressuring feeding.<sup>63,64</sup> Future studies should unravel women's intentions and concerns about feeding, weight loss, and body image to obtain more insight in the underlying mechanisms.

The lower levels of pressuring feeding (chapter 2.2) that we found in mothers with a history of eating disorders, and their higher diet quality during pregnancy as well as the higher diet quality of their infants (chapter 2.1) might indicate that a mothers' history of eating disorders may even have some positive health outcomes. A higher diet quality reflects a higher adherence to the dietary guidelines.<sup>65</sup> If a higher diet quality infancy tracks throughout the life course,<sup>66</sup> this could be ben-

eficial for cardiometabolic health.<sup>67</sup> In addition, low levels of pressure to eat can be considered as positive, as more pressure to eat has been related to children's negative affective reactions to food.<sup>35</sup> However, there may be an optimal level of pressing feeding. A lack of encouragements or prompts to eat particular foods or to eat more may also have negative consequences for children's health, although this needs to be addressed in future research.

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**FIGURE 4.1. Vignette and main questions eating disorders**

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**Having an eating disorders is when someone is severely concerned with their weight**

Some people are abnormally afraid of becoming overweight. However, in comparison with other people of the same age and height, they are very thin. They are nevertheless very dissatisfied about their own body and believe they are overweight, whereas other people find them too thin. They do everything possible to lose more weight. We call these eating disorders **anorexia**.

Some people suffer from recurrent bouts of compulsive eating. This is when at least twice a week a person feels like they cannot stop eating and eats huge amounts of food within a couple of hours. They then try to make sure that they don't put on weight due to the compulsive eating, by vomiting, using laxatives, or other means. The image that they have of themselves is greatly influenced by what they look like and how much they weight. We call these eating disorders **bulimia**.

- 
- 1 Have you *ever* tried to lose weight to the extent that you may have suffered from anorexia?  
Have you suffered from anorexia in the past year?
  - 2 Have you ever had bouts of compulsive eating as described for bulimia?  
Have you ever suffered from bulimia in the past year?
- 

**Assessment of eating disorders in population-based studies**

Eating disorders are often assessed by clinical interviews or validated questionnaires, which are often very time-consuming and were not considered to be feasible in this large cohort study. Therefore, a very short self-report questionnaire was used in which AN and BN were briefly described, followed by questions whether women

had ever experienced these symptoms. (see Figure I). Previous research has indicated the validity of self-report questions for assessing eating disorders.<sup>68</sup> Also, in an attempt to validate our assessment, self-reported eating disorders were compared with diagnoses according to the Composite International Diagnostic Interview (CIDI), which was administered in a subsample (n=928) of the overall Generation R sample.<sup>69</sup> Substantial overlap between self-report eating disorders and CIDI-diagnoses<sup>69</sup> provided some support for the use of self-report. However, only 25 women received a lifetime CIDI-diagnosis of AN or BN, which limits the generalizability of this validation. Our assessment method may have been prone to multiple problems, which I will discuss in the following paragraphs.

First, misclassification of the eating disorders subtypes could have been induced by the specific phrasing of our questions (see Figure 4.1). Although AN was correctly described as having abnormal fears of becoming overweight while being very thin, body dissatisfaction, and trying to lose weight, our question focused on dieting only. Likewise, our question regarding women's history of BN focused on binge eating only. Therefore, women with a history of AN-binge eating/purging type may have been inclined to answer 'yes' to both our AN and BN question, leading to false-positives of BN. Although not a separate diagnosis at the time the data collection of Generation R was ongoing, ascertainment of BN may have also included women who had suffered from binge eating disorder (BED) symptoms, because compensating behaviors were only mentioned in the description of BN and not in the question. Thus, separating AN from BN in our study does not necessarily imply differences between AN and BN, but could also include differences between the AN-types or between AN and BED, which impairs interpretation. Although we once tried to distinguish between AN and BN (chapter 2.2), I now think that a more detailed assessment would have been needed to better distinguish the subtypes. Consequently, we grouped AN and BN together in our subsequent research efforts (described in chapter 2.1 and chapter 2.3). Future cohort studies should for instance consider the Eating Disorder Diagnostic Scale.<sup>70</sup> This is a relatively short, but well-validated questionnaire to obtain information on eating disorders and includes specific questions regarding binge-eating and compensating behaviors.

Another disadvantage of our eating disorder assessment was the retrospective self-report, which may be prone to reporter bias. Asking women whether they have ever tried to lose weight to the extent that they may have suffered from anorexia could also have induced positive answers of women with severe dieting behavior without the pathological fears of gaining weight or extreme body image disturbances that are key to eating disorders. Capturing subthreshold AN or BN does not have to be problematic for our analyses: I would expect the same direction of associations, which is underlined by the findings in chapter 3.2 indicating that not only

clinical levels of mothers' psychopathology are relevant for child eating behavior, but also subclinical levels. Although this chapter focused on maternal anxiety and depression, it clearly showed that subclinical psychiatric symptoms were already related to child eating behavior. Subclinical depressive symptoms during pregnancy were even as strongly as clinical depressive symptoms related to child picky eating. Overall, however, we observed that subclinical anxiety or depressive symptoms were less strongly related to child eating behavior than clinically significant psychopathology. Thus, the moderate associations between maternal history of eating disorders and child outcomes (e.g. emotional overeating, chapter 2.2) could also be explained by the severity of mothers' eating disorder, if our assessment indeed captured cases with a history of subthreshold AN or BN. Moreover, our sample of women with a history of eating disorders was able to conceive, which could indicate a lower severity of eating disorder or some degree of recovery, as severe eating disorders can be life-threatening<sup>71</sup> and increase the risk of fertility and pregnancy complications.<sup>72</sup>

It is also possible that we found only weak associations because it has been a long time ago since the mothers in our study suffered from an eating disorder, given that the incidence of eating disorders peaks in adolescence<sup>73</sup> and almost no women suffered from an eating disorder in the year before pregnancy.<sup>69</sup> We did not have information of mothers' ongoing eating disorder symptoms, while children of mothers with an active eating disorder are more likely to be exposed to, and thus maybe suffer more from their mothers' psychopathology than children of mothers with past eating disorders.

Next to the possible problems arising from the use of self-reports to assess eating disorders in women, the same women also often reported about their children's behavior in our studies. This could have resulted in shared method variance bias inducing inflated associations.<sup>74</sup> This is considered to be a problem in general, but may be even more problematic when mothers have a psychiatric background. The depression-distortion theory states that mothers with psychiatric problems have biased perceptions of their children's behavior,<sup>75</sup> which is underlined by recent findings in our cohort showing that maternal self-reported depression was related to child problems stronger when reported by the mother than by fathers.<sup>76</sup> In chapter 2.1 and chapter 2.2, we only had maternal reports of child diet and eating behavior, which may have implications for our findings. For instance, women with a history of eating disorders may have underreported energy-dense, unhealthy food items, because of embarrassment. If so, the association of maternal history of eating disorders with a higher diet quality may reflect an overestimation. Alternatively, mothers with a history of eating disorders may have over-reported unhealthy food items, because of a higher awareness of eating calorie rich food than women

without a history of ED, resulting in an underestimation of our findings. Also our findings of more emotional overeating in the children of mothers with a history of eating disorders could be attributed to bias. However, Whelan and Cooper indicated that maternal reports of child eating behavior were strongly related to observations, even amongst women with eating disorders.<sup>77</sup> Moreover, in our own cohort, we observed that the associations of maternal history of eating disorders with child mental health problems were largely consistent across mothers and teachers on child problems (chapter 2.3).

## Subtyping eating disorders

This thesis focused on maternal history of *any* eating disorder – i.e. without studying anorexia nervosa (AN) and bulimia nervosa (BN) separately – given the large cross-over and the similar core psychopathology between AN and BN.<sup>29,78</sup> Anorexia is characterized by excessive dieting that leads to a low weight and a pathological fear of becoming fat. Body image problems in anorexia include body distortion, i.e. perceiving themselves as bigger than they are, and body dissatisfaction. These body image problems are also observed in patients with bulimia nervosa, who are characterized by frequent episodes of binge eating followed by compensating behaviors to prevent weight gain. During a binge episode, women lose control over eating and consume significantly more food than most people would eat in the same period of time. Afterwards, they try to compensate the consumed calories to avoid weight gain by engaging in excessive exercising or dieting, or purging behavior (self-induced vomiting or misuse of laxatives or diuretics). Some women with AN also have purging behavior or binge eating episodes (the binge eating/purging type). Although their binges may be subjective of nature (i.e. not consuming an objectively large amount of food), they do experience loss of control over eating. Given all these similarities, it is not surprisingly that anorexic patients often develop bulimia over time (cross-over). Cross-over is also observed from the restrictive anorexic type – characterized by dieting and exercising behaviors – to the binge-eating/purging type. Considering the large cross-over and the shared core psychopathology of over-evaluating body weight and shape in both AN and BN, a trans-diagnostic approach has even been proposed, in which eating disorders are considered as a single diagnostic category.<sup>78</sup>

Besides the similarities between AN and BN, differences between the disorders may be involved in differential transmission of maternal psychopathology to child outcomes. For instance, given that bulimic patients often engage in behaviors that are in conflict with their ideal self-image,<sup>79</sup> mothers with a history of BN may be more keen to prevent these ego-dystonic behaviors in their offspring. It has also

been proposed that particularly mothers with a history of BN extend breastfeeding to postpone cooking for their children, as they might fear to lose control when they prepare their children's meals and engage in binge-eating.<sup>43,59</sup> Mothers with a history of BN have indeed been reported to feed their infants more often with jarred baby-food instead of home-made food.<sup>80</sup> Although home-made food is generally regarded as healthier in adults,<sup>81,82</sup> commercial ready-to-eat-food does not necessarily imply a lower diet quality in infancy.<sup>83</sup> However, when mothers with a history of BN continue to avoid cooking, their offspring might be at risk of an unhealthier diet later in childhood, giving that cooking at home is associated with healthier diets.<sup>81,82</sup> Avoiding to cook out of fear of binge-eating may not only apply to mothers with a history of BN, but also to the binge-eating/purging type of AN (and not to the restricting type of AN). Thus, for some associations, it might be interesting to look at differences between the different eating disorders subtypes. However, our assessment did not separate between the anorexic-restrictive type and the anorexic-binge eating/purging type. Moreover, as discussed in the previous section, our assessment might be more suitable for measuring *any* eating disorder history than for distinguishing AN from BN.

## **Children of mothers with eating disorders: implications and future research**

How mothers perceive their children's emotional eating and diet can affect their children's actual eating behavior. Mothers' perceptions may influence their own feeding strategies on the long term, which, consequently, could lead to changes in children's emotional eating behavior. Evidence for such a self-fulfilling prophecy of eating behavior comes from obesity research indicating that parents' perceptions of children's overweight altered parents' feeding strategies which in turn predicted children's eating and weight.<sup>84</sup> Moreover, our finding that children of mothers with eating disorders are at risk of emotional problems irrespective of who reported on the children's problems also indicates that these children should be carefully monitored. Health care practitioners could simply ask mothers a few questions about her history of eating disorders. Mothers with a history of eating disorders should be screened for active eating disorder psychopathology and anxiety or mood disorders, and treated accordingly. Their children could be selected for intervention programs to prevent eating disorders. It has been reviewed that successful programs (i.e. interventions that decreased eating psychopathology or risk factors such as body dissatisfaction, dieting, and negative affect) focus on body acceptance and are performed by professionals rather than teachers.<sup>85</sup>

From a clinical perspective, it is important to unravel modifiable mechanisms underlying the associations between maternal history of eating disorders and young children's eating or emotional problems. In particular, future research should focus on modifiable mechanisms such as feeding practices or parenting, as these could be targeted in future intervention programs. As children of mothers with eating disorders are at risk of developing eating disorders themselves,<sup>86</sup> it is important to study early signs of this intergenerational transmission, in follow-up to this thesis. Future studies should indicate whether dieting and body image problems already emerge before adolescence in children of mothers with a history of eating disorders.



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CHAPTER 5

## Summary





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CHAPTER 5.1

## Short summary





## Short summary

**Chapter 1** provides a short background of the research presented in this thesis. Briefly, eating disorders are severe mental illnesses and may not only impact on women themselves, but also on their offspring. Children of mothers with eating disorders have an increased risk of developing eating disorders, which typically manifest during adolescence. Earlier in childhood, however, these children may already be at risk of disturbances in their development. Therefore, we aimed to study young children of mothers with a history of eating disorders.

A common eating problem during childhood is picky eating behavior. Picky eating – also called ‘fussy’, ‘selective’ or ‘choosy’ eating – is characterized by the consistent rejection of certain familiar and non-familiar food items, resulting in a low dietary variety. Picky eating is, despite its high prevalence, its burden for family dinners, and its possible health consequences, a relatively unexplored study area. Therefore, our objective was to identify modifiable risk factors for picky eating, which could help parents and health care practitioners understand and prevent picky eating. In addition, picky eaters may be at risk of weight problems, which we also aimed to study. The research described in this thesis was embedded in the Generation R Study, a prospective population-based cohort in the Netherlands.

In **chapter 2**, mothers with a history of eating disorders and their children were studied. The associations between maternal history of eating disorders and infant feeding were assessed in **chapter 2.1**. We found that mothers with a history of eating disorders had a higher dietary quality during pregnancy, as had their children at the age of one year. Mothers with a history of eating disorders were slightly less likely to initiate breastfeeding, although not statistically significant. Although our finding of a lower tendency to initiate breastfeeding among mothers with a history of eating disorders needs further evaluation, our results suggest that women with a history of eating disorders and their infants have a relatively good diet quality. In **chapter 2.2**, we evaluated the relation of maternal history of eating disorders with mothers’ feeding practices and offspring’s emotional eating behavior. The results showed that mothers with a history of eating disorders were less likely to pressure their children to eat, but that they do not differ from mothers without a history of eating disorders in the level of monitoring unhealthy food intake of their children. Preschool-aged children of mothers with a history of eating disorders had higher levels of emotional eating than children of mothers without such a history. We did not find an association between maternal history of eating disorders and children’s BMI at age 6 years. Overall, the levels of emotional overeating amongst children of mothers with a history of eating disorders are noteworthy, particularly considering

the young age (4 years) of participating children. This finding may reflect an effect of maternal eating disorder on the development of disordered eating patterns, but could also be subject to mothers' perception, since maternal reports on child eating behaviors were used. The associations of maternal history of eating disorders with emotional and behavior problems at age 6 years were studied in **chapter 2.3**. Results suggest that children of mothers with a history of eating disorders are at risk of developing emotional problems, but not of behavior problems in the early school years. The associations were consistent among mothers and teachers as informants. These findings could be indicative of a general vulnerability for psychopathology or of an intergenerational transmission of eating disorders, as emotional problems might precede the onset of eating psychopathology.

**Chapter 3** comprises this thesis' research on child picky eating behavior. Risk factors for picky eating behavior were studied in chapters 3.1 and 3.2. In **chapter 3.1**, we hypothesized that a longer duration of breastfeeding exposes the child to a greater variety of flavors and therefore reduces the risk of picky eating. Given this proposed mechanism of repeated flavor exposure, we expected the strongest contrast between children who were never breastfed and those with the longest, recommended breastfeeding duration ( $\geq 6$  months). In contrast, we observed that never breastfed children did not differ in picky eating from those breastfed for 6 months or longer. Therefore, our finding that a short breastfeeding duration (between 1 day and 2 months) was related to higher levels of picky eating is not likely to be explained by repeated exposure to various flavors, but more likely to be explained by reverse causation (i.e. early feeding difficulties that induce breastfeeding cessation). Our results also indicated that children to whom vegetables were introduced early on (between 4 and 5 months of age) showed less picky eating behavior than children introduced to vegetables from 6 months on. Thus, the timing of complementary feeding may be more relevant for picky eating than breastfeeding duration. **Chapter 3.2** describes the associations between parental emotional problems and child picky eating. Maternal postnatal anxiety and depressive symptoms have been previously related to child picky eating, but it was unclear whether maternal symptoms are risk factors for picky eating of the child or rather a result of child picky eating behavior. Our results showed that maternal symptoms of anxiety and depression during pregnancy predicted child picky eating at age 4 years, independent of mothers' symptoms at 3 years postnatal, suggesting that the direction of the associations with mothers' antenatal emotional problems is from mother to child. We also found indications that fathers' emotional problems are related to children's picky eating. Clinicians should be aware that not only severe anxiety and depression of parents, but also milder forms of emotional problems can affect child eating behavior.

In **chapter 3.3**, we focused on picky eating and child mental health. In line with clinical studies reporting that picky eating is often prevalent in children with autism spectrum disorders, we demonstrated that persistent picky eating behavior from 1.5 years to 6 years was also related to higher pervasive developmental problems at 6 years in the general population. In previous studies, picky eating has been related to higher levels of behavior and emotional problems in childhood, but most of these studies were limited in their reliance on maternal reports of both exposure and outcome. This could have led to shared method variance bias, which is strengthened by our results showing that picky eating was indeed related to mother reported emotional and behavior problems of the children, but not to teacher reported problems. Finally, in **chapter 3.4**, we examined the longitudinal associations between ‘severe’ picky eating – i.e. also characterized by low enjoyment of food, and high food avoidant behaviors such as slow eating – and body composition. Our findings suggest that young picky eaters are at risk of having a lower fat-free mass and of becoming underweight in a 2-year period.

In conclusion, the results of the research described in this thesis suggest that both children of mothers with a history of eating disorders and children with severe picky eating need careful monitoring of their eating behavior, mental health and growth to prevent adversities in their development. In **chapter 4**, the main findings of this thesis are summarized and critically discussed considering methodological limitations within the field of psychiatric epidemiology.





CHAPTER 5.2

## Uitgebreide Nederlandse samenvatting



## Uitgebreide Nederlandse samenvatting

In dit proefschrift wordt het onderzoek naar eetgedrag van kinderen van wie de moeder een eetstoornis heeft gehad, beschreven. Uit eerder onderzoek was al bekend dat deze kinderen een groter risico hebben om in de adolescentie zelf een eetstoornis te ontwikkelen, maar het was niet duidelijk of zij op jongere leeftijd al andere eetgewoonten hebben, en of zij meer emotionele of gedragsproblemen hebben.

Niet alleen kinderen van moeders met een eetstoornisverleden kunnen eetproblemen krijgen. Veel kinderen maken een fase door waarin zij lastige eters zijn; met name rond de leeftijd van twee jaar vertonen kinderen vaak 'selectief eetgedrag'. Selectieve eters (ook wel 'kieskeurige eters' genoemd) vermijden bepaald voedsel, waardoor hun voeding weinig gevarieerd is. Kieskeurige kinderen weigeren vaak bepaalde groentes of ander voedsel te eten, vanwege bijvoorbeeld de smaak, geur, textuur, of kleur ervan. "Ik wil dat niet eten" is een veelgehoorde kreet aan de eettafel en dit levert lastige situaties op voor ouders. Zij ervaren het selectieve eetgedrag van hun peuter of kleuter vaak als stressvol en voor veel ouders is het dus belangrijk te weten hoe dit moeilijke eetgedrag voorkomen kan worden. Helaas is er maar weinig bekend over de oorzaken van selectief eetgedrag. Tevens is er weinig bekend over de mogelijke gevolgen voor de lichamelijke en mentale gezondheid van selectieve eters.

In dit proefschrift heb ik zowel selectieve eters (**hoofdstuk 3**) als kinderen van moeders die een eetstoornis hebben gehad, bestudeerd (**hoofdstuk 2**). Voordat de uitkomsten hiervan worden samengevat, wordt de opzet van dit proefschrift-onderzoek (**hoofdstuk 1**) kort uiteengezet.

### Generation R: een bevolkingsonderzoek naar opgroeiende kinderen in Rotterdam

Het onderzoek dat in dit proefschrift wordt beschreven, maakt deel uit van een grootschalig bevolkingsonderzoek, genaamd "Generation R" (**hoofdstuk 1**). De "R" staat voor Rotterdam: alle zwangere Rotterdamse vrouwen die tussen 2002 en 2004 zouden bevallen van een kind, werden uitgenodigd om deel te nemen aan dit bevolkingsonderzoek. Tijdens hun zwangerschap kregen zij onder andere extra echo's om de groei van hun kind te meten, en zij ontvingen vragenlijsten waarin bijvoorbeeld werd gevraagd wat zij tijdens hun zwangerschap aten. In de vragenlijst werd ook geïnformeerd of de vrouwen ooit zo intensief aan de lijn hadden gedaan dat er sprake zou kunnen zijn geweest van de eetstoornis anorexia nervosa, en of zij ooit perioden van onbedwingbare eetbuien hadden gehad zoals bij de eetstoornis



boulimia nervosa. Bij deze vragen werd toegelicht wat eetstoornissen zijn (hoofdstuk 4, figuur 4.1). In de toelichting stond aangegeven dat vrouwen met anorexia extreem bang zijn voor gewichtstoename, ondanks het feit dat zij heel slank zijn. Zij proberen er dan ook alles aan te doen om af te vallen, maar blijven zichzelf desondanks als te dik zien en zijn dan ook erg ontevreden over hun lichaam. In de toelichting over boulimia nervosa werd aangegeven dat vrouwen met deze eetstoornis herhaaldelijke eetbuien hebben. Wanneer deze vrouwen een eetbui hebben, hebben ze het gevoel niet meer te kunnen stoppen met eten en zij eten dan in korte tijd een grote hoeveelheid voedsel. Om gewichtstoename te voorkomen, proberen vrouwen met boulimia nervosa na een eetbui de binnengekregen calorieën kwijt te raken door laxeermiddelen te gebruiken, braken op te wekken, of overmatig te sporten. Op basis van deze toelichtingen hebben de vrouwen, die deelnamen aan "Generation R" de vragen over eetstoornissen beantwoord; ongeveer 400 vrouwen (ca. 8.5%) rapporteerden dat zij anorexia nervosa en/of boulimia nervosa hadden gehad, en ongeveer 4400 vrouwen meldden geen eetstoornis te hebben gehad.

Nadat de vrouwen waren bevallen, volgden we in dit onderzoek de ontwikkeling van hun kinderen door de moeders regelmatig vragenlijsten toe te sturen. Toen de kinderen vier jaar werden, vulden de moeders een vragenlijst in over het eetgedrag van hun kinderen. In deze vragenlijst werden onder andere vragen gesteld over selectief eten (het herhaaldelijk vermijden van bepaald voedsel), en over emotioneel eten (de neiging om meer te eten wanneer je verdrietig, boos, of angstig bent). Tevens werd gevraagd naar voedingsstrategieën die moeders toepasten: in hoeverre letten moeders erop hoeveel hun kind snoept en ongezonde snacks eet, en in welke mate oefenden zij druk uit op hun kind om meer te eten, bijvoorbeeld door er bij hun kind op aan te dringen zijn bord leeg te eten.

Toen de kinderen ongeveer zes jaar waren, kwamen zij samen met hun moeder naar het onderzoekscentrum in het Sophia Kinderziekenhuis, waar verschillende metingen werden uitgevoerd. Het gewicht en de lengte van de kinderen werd opgemeten om het BMI te berekenen. Als iemand een hoog BMI heeft, betekent dat echter niet per definitie dat iemand ook een hoge vetmassa heeft. Daarom is er ook een lichaamsscan van de kinderen gemaakt om hun vetmassa en vetvrije massa te berekenen.

Op negenjarige leeftijd werden alle kinderen opnieuw uitgenodigd voor metingen in het onderzoekscentrum, maar de uitkomsten hiervan waren nog niet beschikbaar ten tijde van het schrijven van dit proefschrift. In 2016 zijn de eerste kinderen van het Generation R onderzoek twaalf jaar geworden en zij worden dit jaar opnieuw uitgenodigd voor nader onderzoek naar hun ontwikkeling tijdens de pubertijd.

## De kinderen van moeders met een eetstoornisverleden

**Hoofdstuk 2** beschrijft het onderzoek naar de voedingsgewoontes en psychische problemen van kinderen van moeders met een eetstoornisverleden. In **hoofdstuk 2.1** wordt uiteengezet dat de kwaliteit van het eetpatroon van zwangere vrouwen met een eetstoornisverleden beter was dan van moeders zonder eetstoornisverleden. Ook aten de éénjarige kinderen van moeders met een eetstoornisverleden meer volgens de richtlijnen voor gezonde voeding dan kinderen van moeders zonder zo'n verleden. Een verklaring hiervoor zou kunnen zijn dat vrouwen die een eetstoornis hebben gehad, meer kennis over voeding hebben en dat zij daarom gezondere voedingskeuzes maken, zowel voor zichzelf tijdens de zwangerschap als voor hun éénjarige kind. Het kan echter ook zijn dat vrouwen met een eetstoornisverleden nog steeds bang zijn om aan te komen en ook niet willen dat hun kind te dik wordt, en daarom dikmakende producten vermijden. Als vrouwen die een eetstoornis hebben gehad inderdaad nog steeds last hebben van sommige symptomen van een eetprobleem, zou het ook kunnen zijn dat ze zich schamen voor hun lichaam en daarom liever geen borstvoeding geven. Dit zou wellicht onze bevinding kunnen verklaren dat vrouwen met een eetstoornisverleden net iets minder vaak borstvoeding gaven dan vrouwen zonder eetstoornisverleden.

Uit het onderzoek beschreven in **hoofdstuk 2.2** bleek dat er ook kleine verschillen zijn in de voedingsstrategieën van moeders met en zonder eetstoornisverleden. Moeders die vroeger een eetstoornis hebben gehad, hadden de neiging om hun kind minder te dwingen om te eten, wellicht uit angst dat hun kind te veel eet en dik wordt – ondanks dat hun kinderen geen hoger BMI hadden – of omdat zij zelf veel druk rondom eten hadden ervaren toen ze aan een eetstoornis leden. Minder druk uitoefenen om te eten kan gunstig zijn voor het kind, omdat dwang om te eten negatieve gevolgen kan hebben voor de sfeer tijdens de maaltijd en wellicht selectief eetgedrag kan bevorderen; dit moet echter verder worden onderzocht. In het onderzoek zijn geen verschillen gevonden tussen moeders met en zonder eetstoornisverleden wat betreft de mate waarin zij erop letten hoeveel hun kind snoept en andere ongezonde snacks at. Dit komt wellicht doordat alle ouders dit bij deze jonge kinderen nog goed in de gaten houden.

Naarmate de kinderen ouder werden, konden ook andere verschillen tussen kinderen van moeders met en die van moeders zonder een eetstoornisverleden worden waargenomen. De kinderen van moeders met een eetstoornisverleden waren al op vierjarige leeftijd vaker emotionele eters (**hoofdstuk 2.2**) en hadden meer emotionele problemen op de leeftijd van zes jaar. De kinderen van moeders met een eetstoornisverleden hadden echter niet meer gedragsproblemen (**hoofdstuk 2.3**). Dit betekent dat deze kinderen met name meer gevoelens van angst of somberheid

hadden. Het is zorgwekkend dat de kinderen van moeders die een eetstoornis hebben gehad al op jonge leeftijd problemen ondervinden in hun emotionele ontwikkeling en het is daarom nodig om deze kinderen goed te blijven volgen.

## Selectief eetgedrag van jonge kinderen

**Hoofdstuk 3** beschrijft het onderzoek naar selectief eetgedrag van jonge kinderen. Uit eerder onderzoek was bekend dat de smaak van borstmelk wordt beïnvloed door het voedingspatroon van de moeder. Kinderen die langdurig borstvoeding krijgen, worden dus voor lange tijd blootgesteld aan de verschillende smaken die afkomstig zijn uit het voedingspatroon van de moeder. Dit verklaart waarschijnlijk waarom uit eerder onderzoek bleek dat deze kinderen in het eerste levensjaar makkelijker vast voedsel accepteren dan kinderen die geen of kort borstvoeding hebben gehad. Daarom was de verwachting dat een kind met een lange borstvoedingsduur minder kans had om een selectieve eter te worden. **Hoofdstuk 3.1** laat zien dat er geen verschil bleek te zijn in de mate van selectief eetgedrag tussen kinderen die nooit borstvoeding hadden gehad en kinderen die minstens zes maanden borstvoeding hadden gekregen. Kinderen met een zeer korte borstvoedingsduur (tussen 1 dag en 2 maanden) vertoonden echter wel meer selectief eetgedrag dan kinderen met de langste borstvoedingsduur. Het is mogelijk dat kinderen die op vierjarige leeftijd selectieve eters zijn, al tijdens het krijgen van de borst lastiger zijn, waardoor hun moeders stoppen met het geven van borstvoeding, maar vervolgonderzoek zou dit moeten uitwijzen. Kinderen die vanaf vier maanden groentehapjes hadden kregen, hadden een kleinere kans op selectief eetgedrag op vierjarige leeftijd dan kinderen die pas later groentehapjes hadden gekregen. Het zou kunnen dat het vroegtijdig introduceren van groentehapjes bijdraagt aan het voorkomen van selectief eetgedrag. Een alternatieve verklaring is dat ouders die zelf selectieve eters zijn, pas later hun kind met groente in aanraking brengen, omdat zij dit zelf niet lekker vinden, en dat kinderen selectieve eters worden vanwege genetische factoren of omdat zij het gedrag van hun ouders kopiëren, maar niet zo zeer vanwege de periode waarin zij voor het eerst groentehapjes hebben gekregen. Dit kon echter helaas niet worden onderzocht, omdat er geen informatie over het selectieve eetgedrag van de ouders beschikbaar was.

In **hoofdstuk 3.2** van dit proefschrift wordt het onderzoek beschreven naar de vraag of kinderen van angstige en depressieve ouders een grotere kans hebben op selectief eetgedrag. Het is namelijk bekend dat angst en depressie van ouders ongunstige effecten kunnen hebben op de ontwikkeling van hun kind. Het was ook al bekend dat er een verband is tussen emotionele problemen van moeders en selectief eetgedrag van hun kind. Het was echter niet duidelijk of angst en depressieve

problemen van moeders van invloed zijn op het ontstaan van selectief eetgedrag bij het kind, of dat selectief eten van het kind juist stress oproept bij de ouders, waardoor emotionele problemen bij de ouders kunnen ontstaan. In dit onderzoek is er daarom voor gekozen om na te gaan of ouders angstig en depressief waren in een periode waarin het eetgedrag van het kind nog geen invloed kan hebben op de gevoelens van zijn/haar ouders, namelijk al voor de geboorte van het kind. Vervolgens hebben we een verband aangetoond tussen depressieve en angstklachten van ouders tijdens de zwangerschap en verhoogd selectief eetgedrag bij hun kinderen. Dit wijst erop dat emotionele problemen van ouders van invloed zijn op het ontstaan van selectief eetgedrag van het kind. Overigens is het niet zo dat selectief eetgedrag alleen voorkwam bij kinderen van moeders met een angststoornis of een depressie, maar ook kinderen van moeders met mildere emotionele problemen hadden een groter risico op selectief eetgedrag.

Het is mogelijk dat het verband tussen emotionele problemen van de ouders en selectief eetgedrag van hun kinderen wordt verklaard door emotionele problemen van het kind. Er zijn onderzoekers die suggereren dat selectief eetgedrag een uiting is van emotionele en gedragsproblemen van het kind. Uit eerder onderzoek is namelijk gebleken dat selectieve eters vaker emotionele en gedragsproblemen hebben. Een beperking van dit eerdere onderzoek is echter dat dit gebaseerd is op de rapportage van moeders over zowel het selectieve eetgedrag als de mentale gezondheid van hun kind. Het is mogelijk dat moeders een bepaalde manier hebben waarop zij een vragenlijst over hun kind invullen. Sommige moeders zijn wellicht geneigd om hun kind hoge scores toe te kennen aan allerlei problemen, waardoor onderzoekers een – wellicht onjuist – verband hebben gevonden tussen selectief eetgedrag en gedrags- of emotionele problemen. In het onderzoek dat dat beschreven staat in **hoofdstuk 3.3**, hebben we er dan ook voor gekozen om leerkrachten te vragen vragenlijsten in te vullen over emotionele en gedragsproblemen van de kinderen in het Generation R onderzoek. Hieruit bleek dat de moeders van selectieve eters inderdaad meer emotionele en gedragsproblemen bij hun kind rapporteerden dan de leerkrachten. Er kon géén verband tussen selectief eetgedrag en gedrags- of emotionele problemen worden vastgesteld wanneer de leerkrachten over emotionele en gedragsproblemen rapporteerden. Dit wijst erop dat de moeders van selectieve eters overschatten in hoeverre hun kind ook andere psychische problemen heeft, en dat selectieve eters in werkelijkheid géén verhoogde kans op gedrags- of emotionele problemen hebben. Wel kwam uit ons onderzoek naar voren dat kinderen met autistische trekken (waarover zowel door de leerkracht als door moeder werd gerapporteerd) vaker langdurig selectieve eters zijn.

In dit proefschrift zijn ook gezondheidsrisico's van selectief eetgedrag onderzocht. Selectief eetgedrag komt erg vaak voor (bij ca. 14% tot 50% van de kinderen). Het is

dan ook mogelijk dat selectief eten - hoeveel stress dat ook oplevert bij ouders - onderdeel is van de normale ontwikkeling van een kind, waarvan het geen schadelijke gevolgen ondervindt op lange termijn. Deze hypothese wordt ondersteund door de onderzoeksresultaten uit hoofdstuk 3.3, die laten zien dat selectieve eters geen hogere kans hadden op emotionele en gedragsproblemen. Het is echter mogelijk dat een bepaalde groep selectieve eters wel gezondheidsproblemen ontwikkelt. Van de Rotterdamse kinderen bestond een kleine groep (6%) uit selectieve eters die behalve kieskeurig met voedsel, ook weinig genoten van eten, snel vol raakten, en zeer langzaam aten. Mogelijk is dit een groep 'ernstig' selectieve eters, die meer risico's lopen op een verstoorde lichaamssamenstelling. In **hoofdstuk 3.4** staat beschreven dat deze kinderen in een periode van twee jaar tijd inderdaad vaker ondergewicht ontwikkelden dan kinderen die geen ernstig selectieve eters waren. Wanneer we de lichaamssamenstelling van deze groep selectieve eters nauwkeurig bestudeerden, bleek verder dat deze kinderen niet een lager BMI hadden door een lagere vetmassa, maar vooral door een lagere vetvrije massa, die grotendeels bestaat uit spieren.

## Tot slot

In **hoofdstuk 4** worden de resultaten van dit proefschrift bediscussieerd. Hierin worden een aantal belangrijke methodologische aspecten van het onderzoek besproken, evenals de implicaties van de resultaten voor de wetenschap en de samenleving. Samenvattend kan worden gesteld dat de resultaten van dit onderzoek aangeven dat zowel kinderen met sterk selectief eetgedrag als kinderen van moeders met een eetstoornisverleden nauwlettend in de gaten moeten worden gehouden.



CHAPTER 6

## Appendices





CHAPTER 6.1

## Abbreviations





# Abbreviations

<b>Abbr.</b>	abbreviations
<b>ALSPAC</b>	Avon Longitudinal Study of Parents and Children
<b>ANOVA</b>	analysis of covariance
<b>AN</b>	anorexia nervosa
<b>ANT</b>	Antillean
<b>ARFID</b>	Avoidant/Restrictive Food Intake Disorder
<b>ASD</b>	autism spectrum disorders
<b>BED</b>	binge eating disorder
<b>BMI</b>	body mass index
<b>BN</b>	bulimia nervosa
<b>BSI</b>	Brief Symptom Inventory
<b>CAPE</b>	Cape Verdian
<b>CBCL</b>	Child Behavior Checklist
<b>CEBQ</b>	Children's Eating Behaviour Questionnaire
<b>CFQ</b>	Child Feeding Questionnaire
<b>CI</b>	confidence interval
<b>CIDI</b>	Composite International Diagnostic Interview
<b>DSM</b>	Diagnostic and Statistical Manual
<b>DXA</b>	Dual-energy-X-ray absorptiometry
<b>EDI</b>	Eating Disorder Inventory
<b>FAD</b>	Family Assessment Device
<b>FFQ</b>	food frequency questionnaire
<b>FFMI</b>	fat-free mass index
<b>FMI</b>	fat mass index
<b>IBQ-R</b>	Infant Behavior Questionnaire-Revised
<b>IQR</b>	interquartile range
<b>kg</b>	kilogram
<b>m</b>	meter
<b>MC</b>	medical center
<b>NOSIK</b>	Nijmeegse Ouderlijke Stress Index Kort
<b>OR</b>	odds ratio
<b>SD</b>	standard deviation
<b>SDS</b>	standard deviation scores
<b>SUR</b>	Surinamese
<b>TRF</b>	Teacher Report Form





CHAPTER 6.2

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## Author affiliations



## Author affiliations

*Behavioural and Brain Sciences Unit, Institute of Child Health, University College London, London, United Kingdom*

Nadia Micali

*Department of Child and Adolescent Psychiatry / Psychology, Erasmus MC-University Medical Center, Rotterdam, the Netherlands*

Pauline W. Jansen, Henning Tiemeier, Frank C. Verhulst

*Department of Epidemiology, Erasmus MC-University Medical Center, Rotterdam, the Netherlands*

Lisanne M. de Barse, Oscar H. Franco, Albert Hofman, Vincent W.V. Jaddoe, Elisabeth T.M. Leermakers, Anh N. Nguyen, Jolien Steenweg-de Graaff, Anne Tharner, Henning Tiemeier, Trudy Voortman

*Department of Epidemiology, Harvard T.H. Chan School of Public Health, Boston, United States*

Albert Hofman

*Department of Epidemiology, Mailman School of Public Health, Columbia University, New York, United States*

Hans W. Hoek

*Department of Pediatrics, Erasmus MC-University Medical Center, Rotterdam, the Netherlands*

Vincent W.V. Jaddoe

*Department of Psychiatry, Erasmus MC-University Medical Center, Rotterdam, the Netherlands*

Henning Tiemeier

*Department of Psychiatry, University Medical Center Groningen, University of Groningen, Groningen, the Netherlands*

Hans W. Hoek

*Department of Psychiatry, Icahn School of Medicine at Mount Sinai, New York, United States*

Nadia Micali

*Department of Psychology, University of Copenhagen, København K, Denmark*

Anne Tharner

*Institute of Psychology, Erasmus University Rotterdam, Rotterdam, the Netherlands*

Pauline W. Jansen

*Mindich Child Health and Development Institute, Icahn School of Medicine at Mount Sinai, New York, United States*

Nadia Micali

*Parnassia Psychiatric Institute, The Hague, the Netherlands*

Sebastian Cardona Cano, Hans W. Hoek, Daphne van Hoeken

*Taste and Behavioral Sciences, Nestlé Research Center, Lausanne, Switzerland*

Lisa R. Edelson-Fries

*The Generation R Study Group, Erasmus MC-University Medical Center, Rotterdam, the Netherlands*

Lisanne M. de Barse, Vincent W.V. Jaddoe, Elisabeth T.M. Leermakers, Anh N. Nguyen, Trudy Voortman



CHAPTER 6.3

## About the author





## About the author

Lisanne de Barse was born on the 23th of March, 1989, in Rotterdam, the Netherlands. She was the firstborn child of Peter de Barse and Annemiek de Barse-Gras. Together with her two brothers, Stephan and Lennard, and her sister Danique, she grew up in Ridderkerk and Strijen, two villages near Rotterdam.

After completing her pre-university education at the Marnix Gymnasium in Rotterdam, she went to Paramaribo in Surinam, where she lived for 3 months in the summer of 2007. Later that year, she began her studies in psychology at the Erasmus University Rotterdam. For her specialization in child and adolescent psychology, she pursued her education at Leiden University, at which she obtained her Master's degree in 2012. During her studies, she gave behavior therapy to children with autism and learning disabilities. Inspired and determined to learn the principles of Applied Behavior Analyses (ABA), she followed numerous workshops at the ABA Institute for four years, and was supervised by Caroline Peters.

Lisanne's interest in research was awakened when she wrote her Bachelor thesis on coping strategies of adolescents with chronic diseases. Consequently, she decided to combine her clinical internship at the department of Clinical Psychology of the Ikazia Hospital in Rotterdam with a scientific internship at the department of Child and Adolescent Psychiatry and Psychology of the Erasmus Medical Center in Rotterdam. In 2012, Lisanne began her PhD program at the Department of Epidemiology of the Erasmus Medical Center. She conducted her research within the behavior group of the Generation R Study, and within ErasmusAGE, a research group focusing on the role of lifestyle and nutrition in health. The results of these projects are presented in this thesis. During her PhD, she was actively involved in supervising students, and she completed a postgraduate Master of Health Sciences in Epidemiology at the Netherlands Institute for Health Sciences.

Lisanne lives in Gouda together with her partner Arend van Haaften, with whom she shares her passion for the outdoors. From October 2016 onwards, she will hike the entire length of New Zealand on the Te Araroa Trail, a 3000 kilometers long route from Cape Reinga in the North of New Zealand to Bluff in the South.





CHAPTER 6.4

## Portfolio

# Portfolio

## Summary of PhD training and teaching

Name PhD student:	Lisanne de Barse
Erasmus MC Department:	Epidemiology
PhD period:	August 2012 – October 2016
Promotors:	Prof. dr. H. Tiemeier and Prof. dr. O.H. Franco
Co-promotor:	Dr. P.W. Jansen

TRAINING	YEAR	ECTS
<b>Master of Science in Health Sciences, Epidemiology (NIHES)</b>		
<i>Core program</i>		
Public Health Research Methods	2013	5.7
Study Design	2012	4.3
Biostatistical Methods I: Basic Principles	2012	5.7
Methodologic Topics in Epidemiologic Research	2012	1.4
Biostatistical Methods II: Classical Regression Models	2012	4.3
<i>Short courses</i>		
Psychiatric Epidemiology	2013	1.1
Women's Health	2013	0.9
Repeated measures in clinical studies	2013	1.4
Principles of Epidemiological Data-analysis	2013	0.7
Maternal and Child Health	2013	0.9
Courses for the Qualitative Researcher	2012	1.4
<i>Erasmus Summer Program</i>		
Cohort Studies	2013	0.7
Causal Inference	2013	0.7
Health Economics	2013	0.7
Logistic Regression	2013	0.7
The Practice of Epidemiologic Analysis	2013	0.7
Principles of Research in Medicine	2012	0.7
Methods of Public Health Research	2012	0.7
Introduction to Global Public Health	2012	0.7
Primary and Secondary Prevention Research	2012	0.7
Markers and Prognostic Research	2012	0.7
Social Epidemiology	2012	0.7
<b>General academic skill courses</b>		
Biomedical English Writing and Communication, Erasmus MC	2015	2.0
Workshop Medical Writing by Philip Greenland, Erasmus Summer Program	2014	0.7
Integrity in scientific research, Erasmus MC	2014	2.0
Endnote, Medical Library, Erasmus MC	2013	0.3
Systematic Literature Search, Erasmus MC	2013	0.3

### Attended seminars and workshops

Erasmus MC meetings (seminars and research meetings of general epidemiology, nutritional epidemiology, and psychiatric epidemiology)	2012-2016	5.0
Workshop Media Training, ErasmusAGE	2016	0.1
Workshop Nutritional Epidemiology, ErasmusAGE	2015	0.1
Seminar "The psychology of obesity", Anita Jansen	2014	0.1
Workshop "De plakkende vloer", Vena, Erasmus MC	2013	0.3

### Conference presentations

#### Oral presentations

"Infant feeding and child picky eating" – Dutch Epidemiology Conference	2016	1.0
"Are parents' anxiety and depression related to child picky eating?" – International Society of Behavioral Nutrition and Physical Activity	2015	1.0
"Maternal history of eating disorders, feeding practices, and child emotional eating" – Sophia Research Day, Erasmus MC	2015	0.5

#### Poster presentations

"Picky eating and body composition" – European Congress of Epidemiology	2015	0.5
"Maternal history of eating disorders, feeding practices, and child emotional eating" – International Society of Behavioral Nutrition and Physical Activity	2015	1.0

TEACHING	YEAR	ECTS
<b>Lectures &amp; supervision of practical and exercises</b>		
Lecture Eating disorders, first year psychology students, EUR	2016	1.0
Training and supervision of behavioral research assistants Generation R	2014-2015	2.0
Teaching assistant Study Design, NIHES	2013	0.5
<b>Supervision of students' thesis work</b>		
Anh Nhi Nguyen, MSc thesis Nutrition and Health, VU University, "Maternal history of eating disorders and child diet quality"	2015-2016	2.0
Eftychia Kotronia, MSc thesis Nutrition and Health, Wageningen University "Children's body image and emotional or behavior problems"	2015-2016	2.0
Monica Walhout, MSc thesis Family Pedagogy, EUR "Maternal sensitivity and food fussiness in early childhood"	2015	2.0
Denise Schneider, MSc thesis Psychology, EUR "Maternal history of eating disorders and child restraint eating behavior and body dissatisfaction"	2015	2.0
Tinke Quaak, MSc thesis Clinical Psychology, EUR "Maternal history of eating disorders and children's emotional or behavior problems"	2014-2015	2.0





CHAPTER 6.5

# Dankwood





## Dankwoord

Het is de dag dat dit proefschrift naar de drukker moet. Een mooi moment om eens stil te staan bij de afgelopen jaren en met name bij diegenen die hebben bijgedragen aan de totstandkoming van dit boekje.

### De ingrediënten

Een proefschrift schrijven doe je niet alleen. Net als bij het klaarmaken van een ingewikkeld recept heb je de juiste ingrediënten nodig om tot het beoogde eindresultaat te komen. “Wat zijn de ingrediënten voor het schrijven van een proefschrift?” vraag u zich wellicht af. Zoals gebruikelijk in de wetenschap, is daar geen universeel antwoord op. Wel zal ik u vertellen wat een aantal belangrijke ingrediënten waren voor dit specifieke proefschrift:

### Een beetje geluk

Zoals wel vaker in het leven, zijn er soms net de juiste omstandigheden nodig die je op de juiste plek bij de juiste persoon doen belanden. Als jonge studente kwam ik bij dr. Pauline Jansen terecht, bij wie ik mijn masterscriptie schreef. Zij stelde me voor aan Prof. Dr. Henning Tiemeier en Prof. Dr. Oscar Franco – Pauline wat ben ik jou hier dankbaar voor – en u raadt het misschien wel, zo ging het balletje rollen en kwam ik op de juiste plek: als promovenda bij de afdelingen epidemiologie en Generation R.

### Een heleboel gegevens van de deelnemende kinderen en ouders van Generation R

Een proefschrift moet natuurlijk wel ergens over gaan en voor gedragsonderzoek heb je gegevens van mensen nodig. Hoe bijzonder is het niet dat duizenden moeders jaren geleden besloten mee te doen met een bevolkingsonderzoek naar opgroeiende kinderen in Rotterdam? Tot op de dag van vandaag vullen zij bijna jaarlijks ellenlange vragenlijsten in en komen zij eens per drie jaar samen met hun kind naar ons onderzoekcentrum. Beste deelnemers van Generation R, ik kan jullie niet genoeg bedanken voor jullie tomeloze inzet en hoop dat jullie nog vele jaren meedoen!

Het verzamelen van gegevens van duizenden kinderen en hun ouders is een intensieve klus. Dagelijks komen er zo'n 12 kinderen met hun ouders naar het onderzoekcentrum, waar de onderzoeksmedewerkers allerlei testen afnemen: van oogmetingen, lichaamsscans, tot gedragsonderzoeken. Beste focusdames, jullie leg-

gen de basis voor alle proefschriften! Bedankt! In het bijzonder wil ik Anneke, Ineke, Rukiye, en Sabah ('de dames van het gedragsblok') bedanken voor de fijne samenwerking!

Bij de organisatie van al die gegevens komt natuurlijk heel wat kijken. Ook achter de schermen staat er een groot team klaar voor alle logistiek en datamanagement. Ik wil jullie allemaal bedanken, maar in het bijzonder wil ik graag Erica, Mirjam, en Ingrid (de rechterhanden van Henning & Oscar), en Claudia, Marjolein, en Patricia noemen (ofwel 'de harde kern van Generation R'). Erica, Mirjam, en Ingrid, jullie stonden altijd klaar om me te helpen en wisten bergen te verzetten! De afdeling kan niet zonder jullie! Claudia en Marjolein, ik geloof niet dat ik écht kan voorstellen wat er allemaal bij datamanagement bij komt kijken, maar ik weet wel dat het een onwijs grote taak is. Ik heb bewondering voor jullie harde werk en wil jullie bedanken voor alle behulpzame antwoorden op mijn vele e-mails over de data. Patricia, ik weet niet hoe je het doet: bij jou kan iedereen terecht en je weet elke vraag in mum van tijd op te lossen en dan ben je ook nog zo heerlijk recht door zee. Wat heb ik gelachen toen we samen de balie draaiden op het focus!

## Een geweldig team van begeleiders

De gegevens waren verzameld, maar hoe ging ik dat nu analyseren en opschrijven? Ik ging van de psychologie naar de epidemiologie, maar ik geloof niet dat ik eigenlijk wel helemaal wist wat de epidemiologie inhield toen ik aan dit promotietraject begon. Gelukkig is gebleken dat dat helemaal niet erg is wanneer er een sterk team van begeleiders naast je staat die vanaf het begin in je geloven.

Pauline, jij bent één van de belangrijkste ingrediënten van dit proefschrift! Een begeleider als jij kan ik aan iedere andere promovendus aanraden; ik kan me in ieder geval geen betere begeleider voorstellen! Je bracht me niet alleen de grondbeginselen van de epidemiologie bij, maar je gaf me ook de vrijheid en het vertrouwen om mijn eigen pad in het onderzoek te ontdekken en gaf me mogelijkheden om studenten te onderwijzen. Je bleef me ook altijd met veel geduld helpen als ik weer eens velen vragen op je afvuurde of alle genomen beslissingen wilde heroverwegen. Want tja, knopen doorhakken, dat was nooit mijn sterkste punt. Tegenwoordig probeer ik soms te denken wat jij zou zeggen of doen in een situatie om tot een praktische oplossing te komen. Misschien heb ik nog wel het meest geleerd van jouw pragmatische instelling! Daarnaast heb ik grote bewondering voor jouw nuchtere kijk op het leven en doorzettingsvermogen en ga ik absoluut onze fijne gesprekken missen!

Henning, dit zou ik natuurlijk niet op moeten schrijven, maar ik moet bekennen: in het begin durfde ik niet zo goed wat tegen je te zeggen. Je zee van epidemiologi-

sche en psychiatrische kennis in combinatie met je sterke persoonlijkheid wekte ontzag op. Naarmate de tijd vorderde, durfde ik gelukkig wel bij je binnen te lopen, en kwam ik er achter dat je meestal om een uur of zes 's avonds nog verbazingwekkend vaak de tijd nam voor lessen in de epidemiologie of om me de kneepjes van het schrijversvak bij te brengen. Ik moet bekennen dat ik ook wel eens langs liep met een vraag, omdat ik er eigenlijk een beetje doorheen zat met mijn proefschrift. Dat zei ik dan natuurlijk niet, maar ik wist dat je vast iets zou zeggen waardoor ik mijn inspiratie voor onderzoek doen weer terugkreeg. Bedankt voor al je tijd, wijze raad, en de fijne samenwerking!

Oscar, you offered me a place in your research group, which I'm incredibly grateful for. I admire you for starting such a wonderful research group from scratch. Your helicopter view is well reflected in your life-course perspective of ErasmusAGE, and was also very clear in your supervision style: you always kept track of the general progression of my PhD! I'm also very grateful that you gave me media training: you know exactly what to say and prepared me very well for my interview with the press. Importantly, you were always keen on making a great working environment in ErasmusAGE. I think it was your great idea to start the social activity committee and I appreciate that you always made time to join for drinks and dinners. Thanks a lot for giving me a lot of opportunities and for always believing in me.

Anne en Jolien, aan het begin en het eind hebben ook jullie een belangrijke rol gespeeld in de begeleiding. Anne, ik weet niet meer hoe vaak we ons paper wel niet opnieuw hebben geanalyseerd en opnieuw hebben herschreven, maar uiteindelijk ben ik heel blij met het eindresultaat. Bedankt voor je geduld en je begeleiding! Jolien, jij kwam er op het allerlaatst nog bij en ik ben blij dat we een paper samen hebben kunnen schrijven, want ook in die korte tijd heb ik nog veel van je opgestoken: wat weet jij veel en ben jij scherp!

## Betrokken co-auteurs & commissieleden

Dit proefschrift is grotendeels een verzameling van wetenschappelijke artikelen. Die schreef ik niet alleen samen met mijn begeleiders, maar ook met een aantal andere co-auteurs, die ik graag op deze plek wil bedanken. Prof. Dr. Jaddoe, beste Vincent, alle papers van Generation R belanden op jouw bureau, en ik sta er van versteld dat je bij elk paper vaak de vinger op de zere plek weet te leggen en het paper razendsnel van raak commentaar voorziet. Prof. Dr. Hofman, ik wil u vooral bedanken voor de geweldige colleges in de epidemiologie en het opzetten van zo'n prachtig bevolkingsonderzoek. Dr. Edelson-Fries, dear Lisa, it was a pleasure working together with you. I really enjoyed our endless mail conversations, dinners and castle visit in Lausanne and Edinburgh, and our close collaboration on the infant

feeding paper in chapter 3.1. Dr. Voortman en Dr. Leermakers, jullie komen nog wel verder op aan bod in het dankwoord, maar ik wil jullie alvast bedanken voor jullie kennis over voeding en body composition. Samen papers schrijven was een feestje! Anh Nhi Nguyen, het is bijzonder leuk om te zien hoe snel en goed jij nu al een paper schrijft, dat gaat helemaal goed komen met jouw PhD! Tevens wil ik de collega's van Parnassia, Prof dr. Hoek, Dr. Van Hoeken, en met name Sebastian Cardona Cano, bedanken voor de samenwerking die geleid heeft tot het paper opgenomen in hoofdstuk 3.3. Sebastian, de epidemiologie en de psychiatrie zijn twee werelden op zich, en ik vond het verfrissend om kennis te maken met jouw klinische blik. Professor Hoek, u wil ik daarnaast bedanken voor de bereidheid om in mijn grote commissie plaats te nemen.

Prof dr. Verhulst, beste Frank, ondanks dat ik niet één van uw promovendi was, hadden we wel projectoverleg en was ik er elke keer weer aangenaam van verrast door uw oprechte interesse in mijn onderzoek en voortgang van mijn promotie. Ook wil ik u bedanken voor uw scherpe commentaren en de bereidheid om plaats te nemen in mijn kleine commissie en de taak als secretaris op u te nemen. Dr. Micali, dear Nadia, I'm so happy that I got the opportunity and pleasure of working with you. Your expertise on eating disorders helped a lot for writing my thesis and for preparing the next line of assessment on eating behaviors in Generation R. I'm honored that you're part of my reading committee. Prof. Raat, beste Hein, ik wil u graag bedanken voor de fijne lessen in public health tijdens mijn NIHES opleiding en voor uw bereidheid om plaats te nemen in mijn kleine commissie. Professor Wake, dear Melissa, in 2013 we met during one of your visits at the Erasmus MC and I was delighted to hear that you will visit the Erasmus MC again for my defense, thanks a lot! Dr. Jager, hartelijk bedankt dat u plaatsneemt in mijn grote commissie, ik zie er naar uit om met u kennis te maken.

## **Een arsenaal aan kamergenoten**

U zult misschien wel denken: wat doen kamergenoten er eigenlijk toe bij het schrijven van een proefschrift? Nou, vanuit mijn ruime ervaring – ik ben inmiddels zo'n 6 kamers met minstens 20 *roomies* verder – kan ik u zeggen: kamergenootjes zijn van uiterst belang! Ik heb per slot van rekening een groot deel van mijn leven de afgelopen 4 jaar tussen de muren van het Erasmus MC doorgebracht. Zonder al die leuke collega's was het schrijven van een proefschrift een stuk moeilijker geworden. Het zijn er helaas te veel om allemaal bij naam te noemen. Daarom wil in het bijzonder diegenen noemen met wie werkdiscussies naadloos over gingen in het delen van lief en leed.

Charlotte, wat was het gek om jou ineens weer op het Erasmus MC tegen te komen, nadat we jaren geleden samen in een toneelgroep hadden gezeten. Het kan gek lopen, vanaf dag één zaten we onafscheidelijk samen in de collegebanken, vertelden we elkaar werkelijk alles over werk en daarbuiten, en het is dan ook vanzelfsprekend dat jij mijn paranimf bent. In een wereld waar het soms alleen maar over het aantal publicaties lijkt te gaan, ben ik blij dat we elkaar hadden om elkaar eraan te herinneren hoe veel meer er buiten het leven binnen de muren van het Erasmus MC is. Lieve Lottie, ik ben gek op jou en hoop dat we volgend jaar weer een stedentripje (hopelijk dan wel zonder brandalarm in hostels) gaan maken (of samen gaan kamperen), want wát was het leuk samen in Edinburgh!! Ik ben ook heel blij dat we nu veel dichterbij wonen – ook al is het in een andere stad – en hopelijk zorgt dit ervoor dat we weer vaker jouw befaamde pasta pesto eten! Jouw geheime recept heb ik nog niet weten te evenaren en daarnaast was je ook voor dit dankwoord het belangrijke snufje zout dat ik nodig had ;-)

Lisan en Trudy, er kwamen geloof ik tranen aan te pas toen ik bij jullie op de kamer kwam (maar ook zeker toen jullie weggingen) en het zijn niet de eerste tranen die daar gelaten waren. Van jullie leerde ik dat je een Nestlé rapport (of algemene discussie, dankwoord, noem maar op) ook best in één dag kan schrijven; waar ik maakte ik me toch altijd zo druk om? ;-)

Lisan, jij bent denk ik toch wel koningin relativieren. Zo heerlijk verfrissend! Ik moet altijd heel hard lachen om je sterke meningen en kan onze discussies – of het nu over confounders gaat, over ethische medische vraagstukken, of over het huwelijk – erg waarderen. Ik hoop dat je ook in Gouda weer snel een keer op de bank komt slapen en hoop dat we van etentjes met z'n drieën een levenslange traditie maken. Trudy, ik ken niemand die net zo lang als ik een zin kan blijven herschrijven en dat ook met veel plezier doet. Wat was het heerlijk om soms samen achter de computer te kruipen om een uur lang aan een paar zinnen te schaven. Ik ben blij dat ons plan om samen een paper te schrijven zonder te analyseren zo goed is uitgekapt. Je bent ook heel vaak mijn vraagbaak geweest voor allerlei kleine dingen, zeker in de afronding van mijn promotie, dankjewel daarvoor! Helaas is het plan om op tien minuten van elkaar te gaan wonen niet helemaal goed gekomen, maar ik hoop dat we de spontane biertjes, cocktails, en koffies erin weten te houden!

## **Heel veel koffie – en nog veel belangrijker: heel veel collega's om koffie mee te drinken**

“The enjoyment of writing a thesis increases exponentially by each cup of coffee but effect-modification by coffee quality exists” schreef één van mijn collega's, Myrte Tielemans, als stelling voor haar proefschrift. En zo is het maar net. Koffie was een

noodzakelijk ingrediënt voor dit proefschrift, evenals koffiemaatjes. Myrte, Loes, en Ester, wat was het fijn om koffiemaatjes te zijn! Loes, ik heb bewondering voor jouw toewijding, maar vergeet niet af en toe te spelen en te verzamelen, weet je nog? Ik zal proberen zand mee te nemen uit Nieuw Zeeland! Myrt, tot twee keer toe waren we kamergenootjes, maar ik geloof nooit officieel. Met jou kon ik knallen, kletsen, en koffie drinken en ik ben blij dat jij je plek hebt gevonden op de microbiologie! Ester, het is maar goed dat wij nooit samen een kantoor hebben gedeeld, want dan was er van onze proefschriften niet veel terecht gekomen. Het lijkt wel alsof we nooit uitgepraat raken!

Ivonne, jij was mijn koffiemaatje in de gedragsgroep! Sinds jij in de gedragsgroep bent gekomen, voelde ik me daar ook veel meer thuis. Het is jammer dat we nog geen paper samen hebben kunnen schrijven – misschien in de toekomst?– want ik merkte al snel dat onze samenwerking van een leien dakje verliep. Maak je niet te druk over je papers en luister goed naar de wijze woorden van Pauline, want die papers komen bij jou vanzelf wel, dat weet ik zeker!

Ook alle andere collega's in Generation R en ErasmusAGE wil ik bedanken voor de fijne samenwerking en alle gezelligheid! Jullie zijn helaas te veel om allemaal persoonlijk te noemen, ook al zou ik dat graag willen, want met bijna ieder van jullie heb ik wel een kamer gedeeld, een kopje koffie gedronken, of geborreld. Het is fijn om zulke goede collega's te hebben! Last, but not least, some special words for Anna. I really enjoyed our time with the girls together, and I'm so glad for you that you found the love of your life and became mother of two beautiful sons (they're adorable!).

## Vrienden & familie

Voor het schrijven van mijn proefschrift was het soms het allerbeste om even afstand te nemen en te ontspannen. En hoe kan dat beter dan met een stel leuke vrienden en vriendinnen. Bedankt dat jullie er altijd zijn en sorry dat we elkaar de afgelopen jaren soms minder zagen dan ik zou willen. Hopelijk komt daar na mijn reis verandering in!

Opgroeien in een warm gezin met stimulerende ouders en lieve broertjes en zusje is het fundament geweest om te kunnen studeren en uiteindelijk dit proefschrift te kunnen schrijven. Lieve pa en ma, ik kan jullie dan ook niet genoeg bedanken. Pap, je hebt me altijd aangemoedigd om door te gaan met studeren en promoveren en je hebt gelukkig altijd vertrouwen in me gehad dat ik mijn proefschrift tot een goed einde kon brengen, zelfs als ik dat niet had. Ik ben er daarnaast van overtuigd dat jij voor een groot deel hebt bijgedragen aan mijn drang naar avontuur, want jij nam ons als kind altijd mee naar buiten de natuur in. Mam, zonder jou was

dit proefschrift er niet geweest, dat mag duidelijk zijn. Hoe vaak ik jou wel niet in lichte (of zware) paniek heb opgebeld.... Jij bent er altijd en dat vind ik heel bijzonder, evenals de band die we hebben. Stephan en Lennard, opgroeien met twee broertjes betekende uren met treinen spelen en tenten maken op zolder, en tegenwoordig betekent het vele discussies voeren. We zijn allemaal heel verschillend, maar dat maakt het juist zo interessant!! Danique, ik vind het heel leuk dat je achter me wil staan als paranimf. Het is grappig om de verschillen tussen ons te zien en ik zou willen dat ik soms wat meer van jouw besluitvaardigheid en planningsvermogen had (dan was dit dankwoord ook vast al eerder af geweest).

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### **Een onmisbaar ingrediënt en absolute smaakmaker: een fabel-fantastische vriend**

Lieve Arend, in jouw eigen woorden: je bent fabel-fantastisch. Ik kan het niet voor mogelijk houden hoe mijn leven is veranderd sinds ik jou – bijna 5 jaar geleden – ontmoette. Het is eigenlijk onmogelijk om onder woorden te brengen wat ik voor jou voel, daarnaast wordt het daar veel te zoetsappig van. Wel wil ik zeggen dat met jou aan mijn zijde alles beter wordt. Je maakt me elke dag weer opnieuw een heel gelukkig mens. Het is vooral jouw liefde, geduld, en nuchterheid geweest die me ertoe brachten dit proefschrift af te maken.





