"I Don’t Want to Eat that"

Epidemiological studies of eating problems in the family

Lisanne M. de Barse
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Acknowledgements

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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 Erasmus Universiteit Rotterdam
op gezag van de rector magnificus Prof.dr. H.A.P. Pols

en volgens besluit van het College voor Promoties.

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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
## Contents

### Chapter 1. General introduction


### Chapter 2. Mothers with a history of eating disorders and their children

2.1. Maternal history of eating disorders: diet quality during pregnancy and infant feeding  
2.2. Maternal history of eating disorders: feeding practices and preschoolers’ emotional eating  
2.3. Maternal history of eating disorders: children’s emotional and behavior problems

### Chapter 3. Picky eating in early childhood

3.1. Infant feeding and picky eating  
3.2. Parents’ anxiety and depressive symptoms and child picky eating  
3.3. Child picky eating and mental health problems  
3.4. Child picky eating and body composition

### Chapter 4 General discussion


### Chapter 5 Summary

5.1. Short summary  
5.2. Uitgebreide Nederlandse samenvatting

### Chapter 6 Appendices

6.1. Abbreviations  
6.2. Authors’ affiliations  
6.3. About the author  
6.4. Portfolio  
6.5. Dankwoord
Manuscripts that form the basis of this thesis

Chapter 2
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International Journal of Behavioral Nutrition and Physical Activity, 2015, 12, 153
CHAPTER 1

General introduction
Background

Eating disorders are severe mental illnesses with multiple health consequences, including death. 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. Eating disorders often have a chronic nature 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. 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, 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, 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. 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. 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. 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, 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, but conflicting results regarding behavior problems in these children. 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 and modifiable factors such as maternal modelling. Enduring eating psychopathology may be present in mothers with a history of eating disorders, given that eating disorders are difficult to treat. 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 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. 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. 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. Also, a mothers’ desire for control may impact breastfeeding practices. 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. Alternatively, the common belief that breastfeeding promotes weight loss may increase breastfeeding initiation and duration in women with an eating disorder history. 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. 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.
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. 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. 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). 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%, 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. 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. 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). 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), a picky eating profile has been described in Generation R, with only 5% of the children assigned as picky eaters. 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). Thus, severe picky eating could be a marker for other psychopathology. Severe picky eaters may also be at risk of nutri-
ational 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, 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, including difficulties in eating behavior, 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. 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 wom-
en (e.g. measuring weight and height). Self-administered questionnaires were used
to obtain information about the sociodemographic characteristics and health of
the participants,\textsuperscript{39,40} for instance about parents’ mental health during pregnancy.\textsuperscript{41}
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.\textsuperscript{39,40} We used the food fussiness scale of the CEBQ for
etiologic research questions.\textsuperscript{34} 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),\textsuperscript{30} and the CEBQ-derived picky eating profile.\textsuperscript{27}

When the children reached the age of 5 years, we invited them to our dedicat-
ed research center in the Sophia’s Children Hospital.\textsuperscript{39,40} 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 question-
naire about the children’s emotional and behavior problems.\textsuperscript{32,33}

\textbf{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 his-
tory of eating disorders were at risk of emotional eating behavior in toddlerhood,
weight problems, (chapter 2.2) and emotional or behavior problems in early child-
hood (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.
References


Maternal history of eating disorders:
Diet quality during pregnancy and infant feeding

Manuscript based on this chapter:
Abstract

Background: This study aimed to examine the associations of maternal history of eating disorders with diet quality of pregnant women and their infants, and breastfeeding practices.

Methods: We included 6,196 mother-child pairs from Generation R, a population-based cohort in the Netherlands. Maternal history of lifetime eating disorders was assessed during pregnancy with a questionnaire. Dietary intake during pregnancy and in infancy was assessed with food-frequency questionnaires and diet quality scores were calculated, reflecting adherence to dietary guidelines. Breastfeeding practices were assessed with questionnaires at 2, 6, and 12 months.

Results: After adjustment for socioeconomic and lifestyle factors, women with a history of eating disorders had a higher diet quality than women without a history of disorders (B= 0.24SD, 95% CI: 0.15; 0.33). Mothers with a history of eating disorders were less likely to breastfeed (unadjusted OR= 0.68, 95% CI: 0.51; 0.93), although no longer statistically significant after adjustment (OR= 0.75, 95% CI: 0.55; 1.03). These findings suggest that mothers with a history of eating disorders seem slightly less likely to initiate breastfeeding, however, this warrants further investigation. At the age of 1 year, infants of mothers with a history of eating disorders had a higher diet quality (B= 0.15SD, 95% CI: 0.02; 0.27).

Conclusion: Mothers with a history of eating disorders and their infants seem to have a relative good diet quality, although follow-up studies are needed to assess the long-term associations with diet in later childhood and adolescence.
Introduction

Eating disorders are mental illnesses characterized by disordered eating and distorted body images.\(^1,2\) Anorexia nervosa (AN) is characterized by an extreme restriction of energy intake, a low body weight, an intense fear of gaining weight, and a distorted body image.\(^1,2\) Bulimia nervosa (BN) is characterized by recurrent periods of uncontrolled binge-eating, followed by compensatory behaviors to prevent weight gain.\(^1,2\) Women who suffered from an eating disorder in the past may be more aware of what they eat during pregnancy\(^3\) and which foods they provide to their infants.\(^3\) As nutrition during pregnancy and in early childhood may have long-term consequences for growth, development, and health,\(^4-6\) it is important to study diet quality during these periods.

Women with a history of eating disorders may have more nutritional knowledge and therefore provide themselves and their children with healthier diets.\(^7,8\) Indeed, pregnant women with a history of eating disorders seem more likely to adhere to a dietary pattern characterized by a high intake of meat substitutes, pulses, nuts and herbal teas,\(^9\) and to have lower intakes of high-fat meats.\(^10\) Among school-aged children of mothers with a history of eating disorders, a higher adherence to a data-driven 'health conscious/vegetarian' dietary pattern has also been reported.\(^11\) Likewise, at ages 1-4 years, these children ate less junk food than children of mothers without a history of eating disorders.\(^12\) Less is known about diet quality in infancy. Moreover, adherence to these data-driven dietary patterns or a low consumption of high-fat foods, does not necessarily imply that the overall diet is actually healthier.\(^13,14\) Therefore, further research examining the overall diet quality beyond specific patterns in women with a history of eating disorders and their infants is needed.

Although mothers with a history of eating disorders may provide themselves and their infants with healthier diets, they may face difficulties with breastfeeding.\(^12\) Enduring shape concerns and body awareness in women with a history of eating disorders could evoke feelings of embarrassment of breastfeeding.\(^12,15\) Alternatively, the common belief that breastfeeding promotes weight loss may increase breastfeeding initiation and duration in women with a history of eating disorders,\(^16\) who may still have the desire to be thin. Contrasting results with regard to breastfeeding have been reported, with a study showing that mothers with a history of eating disorders were more likely to start breastfeeding and to continue for a longer period,\(^17\) whereas other studies found shorter,\(^18,19\) or similar durations.\(^20-22\) Thus, associations between maternal eating disorders and breastfeeding practices remain unclear.
Therefore, we aimed to explore the associations between maternal history of eating disorders and overall diet quality of the mothers during pregnancy, as well as their breastfeeding practices and their infants’ diet quality during the first year of life.

**Methods**

**Study design**

This study was embedded in the Generation R Study, a population-based prospective cohort from fetal life onwards, conducted in Rotterdam, the Netherlands. Pregnant women living in Rotterdam, with an expected delivery date between April 2002 and January 2006 were invited to participate (baseline response rate: 61%). All participating parents gave written informed consent and medical ethical approval was obtained from the local Medical Ethical Committee. Further information is available elsewhere.

**Participants**

A total of 6,608 women were enrolled during pregnancy, provided information on their history of eating disorders and gave full consent for the prenatal and postnatal phase of the study. Those with missing data on all dietary outcome variables were excluded (n=412), resulting in a total of 6,196 mother-child pairs with eligible data. Because data on diet quality and breastfeeding were not complete for all participants, the population for analysis varied per specific analysis (n between 2,933 and 5,035).

**Measures**

**Maternal history of eating disorders**

Mothers’ history of lifetime eating disorders was assessed with a self-report questionnaire during pregnancy. A vignette was provided to clarify what was meant by anorexia nervosa (AN) and bulimia nervosa (BN). This vignette was based on diagnostic criteria, but was slightly changed to create a clear and understandable description of both AN and BN. The vignette was followed by questions whether the participant had suffered from either AN or BN (ever and in the previous year). Additionally, the questionnaire included items about treatment, medication, and the inability to work as a result of the disorder. Women who answered ‘yes’ on at least one of these questions, were categorized as having a history of eating disorders. Due
to a low prevalence of eating disorders in the year before pregnancy, women were grouped according to their lifetime history of any eating disorder (yes / no).

Given the large sample size, it was not feasible to obtain a clinical diagnosis. However, in a sub-sample (n=928) of the Generation R Study, self-reported eating disorders were compared with clinical diagnoses. Excellent sensitivity (100%) and specificity (96%) were found for self-reported AN, and very good sensitivity (94%) and specificity (81%) were found for self-reported BN.

Diet quality during pregnancy

Women’s dietary intake in early pregnancy was assessed using a food frequency questionnaire (FFQ) at enrolment (median 14.6 weeks of gestation, interquartile range [IQR] 12.4-16.1). The FFQ included foods that were frequently consumed in the Dutch population and was modified for use during pregnancy. Energy and nutrient intakes were calculated using the Dutch food composition table from 2006. The FFQ was validated against three 24-hour recalls among 71 pregnant women living in Rotterdam. Intra-class correlation coefficients for macronutrient intakes ranged from 0.5 to 0.7.

National dietary guidelines were used to develop a predefined diet quality score for pregnant women. The following 15 components and cut-offs were included in the diet score: vegetables (≥200 grams/day), fruit (≥200 grams/day), whole grains (≥90 grams/day), legumes (≥135 grams/week), nuts (≥15 grams/day), dairy (≥300 grams/day), fish (≥100 grams/week), tea (≥450 grams/day), grain quality (ratio whole grains of total grains), soft fats and oils (ratio of total fat), red meat (≤375 grams/week), sugar-containing beverages (≤150 grams/day), alcohol (yes/no), salt (≤6 grams/day), and folic acid supplements in early pregnancy (periconceptional/first ten weeks/not). For each component, except for alcohol and folic acid supplements, the ratio of the reported intake and the recommended intake was calculated. For example: a woman with a vegetable intake of 120 grams per day received a score of 0.6 (120 grams/day divided by 200 grams/day) for the vegetable component. The maximum score for each component was 1; if a woman exceeded the recommended intake, her score remained 1. For sugar-containing beverages, red meat, and salt, the scores were reversely coded, meaning that higher scores on these food groups reflect lower intakes. Alcohol intake was dichotomously coded, with no intake scored as 1 and any alcohol intake scored as 0. Intake of folic acid supplements was also categorized, with intake periconceptionally scored as 1; in the first ten weeks of gestation scored as 0.5; and no intake in these periods scored as 0. The scores for the individual components were summed, resulting in an overall score ranging from 0 to 15, with a higher score representing a healthier diet.
Breastfeeding
Information on breastfeeding initiation and duration was obtained from delivery reports and postnatal questionnaires at the child’s ages of 2 months, 6 months, and 12 months. Mothers were asked whether they had ever breastfed their child (yes/no) and if yes, at what age they stopped breastfeeding their children.

Infant’s diet quality
Dietary intake of the child was assessed using a semi-quantitative 211-item FFQ, which was filled out by the mothers when the children were at a median age of 12.9 months (IQR: 12.7-14.0). This FFQ included foods that are frequently consumed by children aged 9-18 months, according to a Dutch national food consumption survey in 2002. Questions covered the frequency of consumption, serving sizes, type of food items, and food preparation over the last month. This FFQ was validated against three 24-hour recalls in a sample of 32 Dutch children aged 14 months living in Rotterdam. This validation showed reasonable to good intra-class correlation coefficients for nutrient intake of 0.4 to 0.7.

The 10 following components were included in the infant diet quality score: vegetables (≥100grams/day), fruit (≥150grams/day), bread and cereals (≥70grams/day), rice, pasta, potatoes and legumes (≥70grams/day), dairy (≥350grams/day), meat, poultry, eggs and meat substitutes (≥35grams/day), fish (≥15grams/day), oils and fats (≥25grams/day), candy and snacks (≤20grams/day), and sugar-sweetened beverages (≤100grams/day). Similar to the maternal diet quality score, ratios of the reported intake and recommended intake were calculated for each component, with reverse coding for the candy and snacks and sugar-sweetened beverage components. Subsequently, these scores were summed, resulting in an overall score ranging from 0 to 10, with higher scores representing a healthier diet. More details on this score are described elsewhere.

Covariates
Based on previous knowledge and studies, several covariates that might influence the associations were considered. Potential covariates included maternal age, ethnic background (based on country of birth of the mother and her parents), educational level, body mass index (BMI), net monthly household income, and psychiatric symptoms. All variables, except for maternal BMI, were assessed using questionnaires during pregnancy. Maternal psychiatric symptoms were measured with the Brief Symptom Inventory (BSI), a validated 53-item self-report questionnaire. The overall score ranged from 0 to 4, with higher scores representing higher levels of psychiatric symptoms. Maternal height and weight were measured at enrollment in the study to calculate BMI (kg/m²). Child sex was examined as a potential effect
modifier, because maternal influence on dietary intake might be different for sons and daughters. Information on child sex was obtained from birth records.

**Statistical analyses**

Linear and logistic regression analyses were used where appropriate to assess whether maternal history of any eating disorder was associated with the different dietary measures. In all analyses, maternal history of eating disorders was coded as ‘history of any eating disorder’ or ‘no history of eating disorders’, with the latter category as the reference. The diet quality scores for pregnant women and infants were standardised for energy intake using the residual method. All associations were analysed in three models: 1) a crude unadjusted model, 2) a model adjusted for maternal age, ethnic background, educational level, BMI, and household income, and 3) a model additionally adjusted for maternal psychiatric symptoms. We adjusted separately for psychiatric symptoms, because controlling for these symptoms may represent over-adjusting due to the high co-occurrence of eating disorders with these symptoms. Effect modification by child sex was assessed in the analyses with child diet quality, by including an interaction term in all models.

Sensitivity analyses were performed in participants with a Dutch ethnic background only (n between 1,975 and 2,971) to reduce the risk of residual confounding, because the FFQs were developed and validated for a Dutch population. Also, analyses with the diet quality score as outcome were repeated, using the original diet quality scores without standardization for energy intake.

To reduce potential bias due to missing values on some of the covariates (ranging from 0% for maternal age to 11.6% for household income), these variables were multiple imputed. The results presented are the pooled regression coefficients or odds ratios of 10 imputed datasets. All statistical analyses were carried out using the statistical software program IBM SPSS statistics, version 21.

**Non-response analyses**

Of the 6,608 mothers who provided information on their history of eating disorders, mothers with missing data on all dietary outcome measures (n=412) were compared to mothers with at least one dietary outcome measure available (n=6,196). Mothers with missing data on all dietary outcomes were younger (t[453.5]= -9.7, p<0.001), more often of non-Dutch origin (χ²[1]= 132.8, p<0.001), lower educated (χ²[1]= 99.7, p<0.001), and had a higher BMI (t[450.6]= 2.4, p<0.05), a lower household income (χ²[1]= 101.6, p<0.001), and higher levels of psychiatric symptoms (t[414.9]= 6.9, p<0.001).
Results

Population characteristics

Characteristics of the study population are presented in Table 2.1.1. In total, 9.5% (n=591) of the mothers reported to have experienced an eating disorder at any point in their life. Mothers with a history of any eating disorders reported more psychiatric symptoms (median BSI score: 0.29 versus 0.15 for women without a history of eating disorders, p <0.001). Women with and without a history of eating disorders did not differ significantly on any other characteristic. The mean diet quality score of children at age 1 years was 4.3 (standard deviation [SD]= 1.4) on a theoretical range from 0 to 10.

| Table 2.1.1 General characteristics of the study population |
|-------------------------------------------------|---------------|-----------------|
| MATERNAL CHARACTERISTICS                        | N             | PERCENTAGE, MEAN (SD), MEDIAN (IQR)* |
| Age at enrollment                               | mean years, SD | 6,196           | 30.3 (5.0) |
| Ethnic background                               | % Dutch       | 3,068           | 55.8       |
| Educational level                               | % high§       | 2,914           | 47.0       |
| Body mass index at enrollment (kg/m2)           | mean, SD      | 6,196           | 24.6 (4.4) |
| Household income                                | % low (< €2000/ month) | 3,824 | 61.7       |
| Psychiatric symptoms                            | median score, IQR | 6,196 | 0.2 (0.1-0.3) |
| History of lifetime eating disorders            | % yes         | 591             | 9.5        |
| Diet quality score in pregnancy∞               | mean score, SD | 4,824           | 7.6 (1.6)  |
| Breastfeeding initiation                        | % yes         | 4,616           | 91.7       |
| Breastfeeding duration                          | mean months, SD | 3,673           | 4.8 (3.8)  |

CHILD CHARACTERISTICS

| Sex % girls | 3,126 | 50.5 |
| Diet quality score at age 1 year∞ | mean, SD | 2,933 | 4.3 (1.4) |

# Values are percentages for categorical variables, means (standard deviation) for continuous normally disturbed variables, and medians (interquartile range) for continuous non-normally distributed variables, derived from the imputed dataset.

§ high: higher vocational training and higher academic education.

∞ Diet quality scores before standardization.
Maternal diet quality score

The associations between maternal history of eating disorders and maternal diet quality score are presented in Table 2.1.2. In all models, including model 3, in which associations were independent of maternal psychiatric symptoms, pregnant women with a history of any eating disorders had a higher diet quality score (B=0.24 SD, 95% confidence interval [CI]: 0.15; 0.33) than pregnant women without such a history.

Breastfeeding

In the unadjusted model (model 1), mothers with a history of eating disorders were less likely to initiate breastfeeding (OR=0.68, 95% CI: 0.51; 0.93, Table 2.1.2). We observed a similar association after adjustment for covariates, however, the effect estimate slightly attenuated and was no longer statistically significant (model 3: OR=0.75, 95% CI: 0.55; 1.03). Among mothers who breastfed their infants, we did not find any significant differences in the duration of breastfeeding between mothers with and without a history of eating disorders (B=0.15 months, 95% CI: -0.27; 0.57).

<table>
<thead>
<tr>
<th>TABLE 2.1.2</th>
<th>Associations of maternal history of eating disorders with maternal and infant diet quality and breastfeeding initiation and duration</th>
</tr>
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<tbody>
<tr>
<td>MATERNAL HISTORY OF EATING DISORDERS</td>
<td>MATERNAL DIET QUALITY SD-SCORE (N=4,824)</td>
</tr>
<tr>
<td>No eating disorder</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>Any eating disorder</td>
<td>0.22 (0.12; 0.31)***</td>
</tr>
<tr>
<td>Model 1</td>
<td>0.23 (0.14; 0.32)***</td>
</tr>
<tr>
<td>Model 2</td>
<td>0.24 (0.15; 0.33)***</td>
</tr>
</tbody>
</table>

* p<0.05, **p<0.01.
# Values are regression coefficients or odds ratios (95% confidence intervals) and can be interpreted as the difference between the eating disorder group and the reference group.
§ Number of cases with a history of eating disorders; n=453 for maternal diet quality analyses, n=478 for breastfeeding initiation analyses, n=330 for breastfeeding duration analyses, and n=266 for infant diet quality analyses.
∞ Model 1: unadjusted model, model 2: adjusted for maternal: age, ethnic background, educational level, and BMI, and household income; model 3: additionally adjusted for maternal psychiatric symptoms.
Infant diet quality score

Table 2.1.2 also shows that infants of mothers with a history of eating disorders had a higher diet quality score (model 3: B=0.15 SD, 95% CI: 0.02; 0.27) than infants of mothers without such a history. Results did not significantly differ between boys and girls (p for interaction >0.05).

Sensitivity analyses

Analyses restricted to the subsample of participants with a Dutch ethnic background only (n between 1,975 and 2,971) showed similar associations between maternal history of eating disorders and the dietary outcomes (Supplementary Table 2.1.1). Only the association between maternal history of eating disorders and infants diet quality was – although in the same direction – no longer statistically significant (B=0.12, 95% CI: -0.03; 0.28), probably due to reduced power. Also the analyses with and without standardization for energy intake showed similar results.

Discussion

In this population-based study, we found that a maternal history of eating disorders was associated with a higher diet quality in both pregnant women and their infants. We did not find a statistically significant association between maternal history of eating disorders and breastfeeding initiation or duration.

Interpretations and comparison with other studies

Our findings of a higher diet quality during pregnancy are in line with previous studies, reporting that women with a history of eating disorders consumed less meat, butter, and full-fat milk and more pulses, margarine, vegetable oils, and skimmed milk than women without a history of eating disorders.9 Congruently, Siega-Riz et al.10 reported lower intakes of high-fat meats and sweetened beverages among pregnant women with past or current BN. Although these studies focused on individual food groups, the choices in individual food groups that these women made, are in line with current dietary guidelines. Thus, these results may imply a desire to make healthy food choices, which is confirmed by our findings of a higher overall diet quality among pregnant women with a history of eating disorders.

Our findings from unadjusted models suggest that mothers with a history of eating disorders were slightly less likely to initiate breastfeeding. However, after
adjustment for covariates, the effect estimate slightly attenuated and was no longer statistically significant. Several explanations might account for this non-significant finding. Although confounding by socioeconomic factors may partly account for this, the attenuation in effect size was only minimal, suggesting that limited power after adjustment may explain why statistically significant differences were no longer detected. In a much larger Norwegian cohort \( n=39,355 \), Torgersen et al. reported differences in breastfeeding practices between mothers with and without an eating disorder,\(^{19}\) whereas studies with small sample sizes (eating disorder cases between \( n=10 \) and \( n=25 \)) found no differences.\(^{20-22}\) In contrast to our findings, Torgersen et al. did not observe differences in breastfeeding initiation, but reported shorter durations of breastfeeding among mothers with eating disorders.\(^{19}\) The same underlying mechanism (i.e. embarrassment and body dissatisfaction\(^ {12,15} \)) could underlie an association of maternal eating disorder history with both breastfeeding initiation and duration. Therefore, contrasting findings could be attributed to eating disorder ascertainment. Torgersen et al. defined the presence of an eating disorder in the 6 months prior to pregnancy and during pregnancy,\(^ {19}\) which may imply that these women had more active and severe psychopathology than the women with a lifetime history of eating disorders in our study. Indeed, they found stronger associations for women with AN than in women with eating disorder not otherwise specified,\(^ {19}\) who may have less severe eating psychopathology.\(^ {42}\) Alternatively, since Torgersen et al. did not observe an association between maternal history of BN and breastfeeding, the associations may be eating disorder-specific. In our study, we did not distinguish between AN and BN because the proposed mechanisms, which were body shame, dissatisfaction, embarrassment, and high body awareness, are characteristics of both AN and BN,\(^ {1,2}\) and because our sample size was too small to distinguish subtypes of eating disorders.

Although infants of mothers with a history of eating disorders seemed to be breastfed somewhat less than infants of mothers without a history of eating disorders, they had a higher diet quality around the age of 1 year. This seems contrary to previous findings, reporting that children who were breastfed, had a higher diet quality.\(^ {31}\) However, this may be different for children of mothers with a history of eating disorders. These mothers often have a negative self-image,\(^ {42}\) and may therefore not feel comfortable about breastfeeding.\(^ {12}\) Previous studies reported that well-functioning breastfeeding requires confidence and belief in the capacity of one’s body.\(^ {43,44}\) Moreover, in the general population, women with higher body image concerns were less likely to initiate breastfeeding and more likely to breastfeed for a shorter period.\(^ {44}\) However, because of a higher awareness of food,\(^ {3}\) mothers with a history of eating disorders may have the desire to provide their children with healthy food and limit unhealthy or high-fat foods, which is reflected by the higher overall diet quality score.
Several explanations might account for our findings of the higher diet qualities in pregnant women with a history of eating disorders and their infants. Some studies suggested that women with eating disorders may have more nutritional knowledge, particularly with regard to the caloric content of food, or that women who recovered from an eating disorder had a higher preference for foods with health benefits. Thus, mothers with a history of eating disorders may be more keen and knowledgeable on providing themselves and their children a healthy diet. Alternatively, our findings may be attributed to ongoing or recurrent eating disorder symptoms. Previous studies have reported that eating disorder symptoms tend to improve during pregnancy, but worsen postpartum. However, other studies reported that eating disorder symptoms may still be present during pregnancy and that women with an eating disorder tend to worry more about weight gain during pregnancy. To prevent further weight gain, they may therefore choose healthier food products with low calories, thereby scoring higher on our quality score. Since reoccurrence of eating disorder symptoms may be present in the postpartum period, concerns about body weight and shape may not only apply to women themselves, but also to their infants. Indeed, several studies have indicated that mothers with a history of eating disorders tend to worry about their children’s diet, and weight— even though we found no differences in actual BMI at the age of 6 years between children of mothers with and without a history of eating disorders (described in chapter 2.2) — which may explain the higher infant diet quality.

Strengths and limitations

This study is one of the first studies that has examined the associations between a mother’s history of eating disorders and diet quality during pregnancy as well as offspring’s diet quality in early childhood. The strengths of this study are its population-based, longitudinal design, the repeated measurements of breastfeeding practices, and availability of several covariates, including maternal psychiatric symptoms allowing us to distinguish between eating disorders and psychiatric problems in general. Another strength is the use of a predefined approach to measure overall diet. An advantage of a predefined approach over a data-driven approach is that predefined diet scores are based on dietary guidelines, which may therefore better reflect a healthy diet.

Despite these strengths, several limitations should be considered. First, maternal history of eating disorder was self-reported, which might have resulted in reporter bias. However, substantial overlap between self-reported lifetime eating disorders and clinical diagnoses in a small subsample of the Generation R Study has been shown previously. Second, a general limitation of FFQs to measure dietary in-
take is that they rely on memory and reported intakes are subject to measurement errors. Moreover, both exposure and outcomes were reported by the same informant (i.e. the mother), which could have led to common method variance bias. Given the possible preoccupation with diet, mothers 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, resulting in an underestimation of our findings. Even though Whelan and Cooper provided evidence that women with eating disorders are capable of reporting reliably on their children’s eating behavior, future research should use additional informants of both mothers’ and children’s food intake. Another limitation is that our FFQs were developed to measure a Dutch diet and were only validated in a Dutch population, whereas women and children with different ethnic backgrounds were included in our study population. Thus, some caution is needed with regard to the diet quality scores of participants with another ethnic background. However, in our sensitivity analyses restricted to participants with a Dutch ethnic background only, similar results were found.

**Conclusions**

Mothers with a history of eating disorders and their infants had a higher diet quality, independent of psychiatric symptoms, suggesting that our findings are specific for eating disorders. 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. However, further research is needed to examine whether these children remain to eat healthier when they start making their own food choices. Further research in which children are followed as they grow older is needed in order to assess long-term associations, preferably with more objectively measured data on eating disorders as well as on dietary intake.
CHAPTER 2  MOTHERS WITH A HISTORY OF EATING DISORDERS AND THEIR CHILDREN

References


**Supplement**

**SUPPLEMENTARY TABLE 2.1.1**  Associations of maternal history of eating disorders with maternal and infant diet quality and breastfeeding initiation and duration among those with a Dutch ethnic background

<table>
<thead>
<tr>
<th>MATERNAL HISTORY OF EATING DISORDERS</th>
<th>MATERNAL DIET QUALITY SD-SCORE (N=2,823)</th>
<th>BREASTFEEDING INITIATION (YES/NO) (N=2,971)</th>
<th>BREASTFEEDING DURATION (MONTHS) (N=2,241)</th>
<th>INFANT DIET QUALITY SD-SCORE (N=1,975)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No eating disorder</td>
<td>0 [reference]</td>
<td>1 [reference]</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>Any eating disorder §</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1</td>
<td>0.28 (0.16; 0.40)**</td>
<td>0.63 (0.44; 0.89)*</td>
<td>-0.00 (-0.54; 0.54)</td>
<td>0.10 (-0.05; 0.25)</td>
</tr>
<tr>
<td>Model 2</td>
<td>0.32 (0.21; 0.43)**</td>
<td>0.73 (0.50; 1.05)</td>
<td>0.20 (-0.32; 0.72)</td>
<td>0.10 (-0.06; 0.25)</td>
</tr>
<tr>
<td>Model 3</td>
<td>0.35 (0.24; 0.46)**</td>
<td>0.73 (0.50; 1.07)</td>
<td>0.22 (-0.30; 0.74)</td>
<td>0.12 (-0.03; 0.28)</td>
</tr>
</tbody>
</table>

* p<0.05, **p<0.01.
# Values are regression coefficients or odds ratios (95% confidence intervals) and can be interpreted as the difference between the eating disorder group and the reference group.
§ Number of cases with a history of eating disorders; n=278 for maternal diet quality analyses, n=291 for breastfeeding initiation analyses, n=203 for breastfeeding duration analyses, and n=178 for infant diet quality analyses.
∞ Model 1: unadjusted model, model 2: adjusted for maternal: age, educational level, and BMI, and household income; model 3: additionally adjusted for maternal psychiatric symptoms.
Maternal history of eating disorders: 
Feeding practices and preschoolers’ emotional eating

Manuscript based on this chapter:
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)\(^1\) and evidence for increasing incidence.\(^2\) AN and BN are associated with an increased risk of psychopathology, medical problems, and premature mortality.\(^1,3,4\) 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.\(^5\)

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.\(^6,7\) 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.\(^7\) However, in BED, these binges are not followed by compensating behaviors.

Women with eating disorders often have difficulties in emotional processing\(^4\) and may experience comorbid mood disorders as well,\(^8\) partially explained by common familial factors which may cause both eating disorders and mood disorders.\(^9\) It has been suggested that emotional eating patterns index vulnerability for eating disorder psychopathology\(^10\) and can be present in both AN and BN.\(^11,12\) 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.\(^10,13\) Emotional undereating is the tendency to lose appetite and eat less when upset.\(^13\) In women with BN, this is mostly reflected by the occurrence of binges in response to emotional distress.\(^11\) Women with AN are likely to eat less in response to emotions,\(^12\) although there is also evidence for elevated levels of emotional overeating.\(^11\)
Considering the evidence for an intergenerational transmission of eating disorders,\(^5\) 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.\(^{14,15}\) 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.\(^{16-19}\) 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.\(^{20}\) Furthermore, a mother’s use of food to soothe her child – a feeding pattern relatively often seen in mothers with eating disorders\(^{21}\) – predicted relatively high levels of overeating in response to emotional distress in another population-based study.\(^{22}\) Together, these studies\(^{14-20,22}\) 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.\(^{21}\) 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,\(^{18,23,24}\) although the evidence is still inconclusive.\(^{25}\) 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.\(^{18,23,24}\) This observation, however, is not supported by all studies.\(^{18,23,24,26}\) In general, a history of AN and EDI-2 subscales drive for thinness and body dissatisfaction were not related to these feeding strategies,\(^{18,23,24}\) only one study reported a positive correlation with restriction and pressure to eat.\(^{23}\) However, despite their population-based character, most of these studies were limited by their small sample size (n<100).\(^{23,25,26}\) 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.\(^{27}\)
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. 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.

**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. BED was not included in our study, as this was not a psychiatric diagnosis at the time we assessed ED.

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). 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.

**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. 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-
sesses 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).

**Children’s eating behavior**

At 4 years of age, children’s eating behavior was assessed using the Children’s Eating Behaviour Questionnaire (CEBQ).\(^{13}\) The CEBQ is a validated, multi-dimensional parent-report questionnaire designed to measure differences in eating behaviors.\(^ {13}\) 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).\(^ {33}\) Moreover, the use of the CEBQ in a Dutch population has been previously validated, indicating good psychometric properties.\(^ {34}\)

**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.\(^ {28}\) These measures were used to calculate child body mass index (BMI, kg/m\(^2\)). Mean BMI was 16.1 (SD=1.7). Age- and
sex-adjusted standard deviation scores (child BMI-SDS) were calculated using Dutch reference curves.35

**Covariates**

Sociodemographic and psychosocial covariates that may confound the studied associations were considered based on previous research.4,5 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.36 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 80th 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/m²).

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),37 the Dutch version of the Parenting Stress Index – Short Form38 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 χ²-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. Missing values on confounders (ranging from 0% missing values for maternal age to 12% for household income) were estimated using multiple imputation techniques 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 (χ²[2]= 101.8, p<0.001), of non-Dutch national origin (χ²[2]= 141.0, p<0.001), with a lower household income (χ²[1]= 118.4, p<0.001), or who had higher levels of psychiatric problems (χ²[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, χ²[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

<table>
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<tr>
<th>MATERNAL CHARACTERISTICS</th>
<th>N TOTAL (N=4,851)</th>
<th>NO EATING DISORDER (N=4,436)</th>
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</tr>
<tr>
<td>Ethnic background:</td>
<td>% Dutch</td>
<td>2,867</td>
<td>60.9</td>
</tr>
<tr>
<td></td>
<td>% other European</td>
<td>390</td>
<td>8.3</td>
</tr>
<tr>
<td></td>
<td>% Non-Western</td>
<td>1,450</td>
<td>30.8</td>
</tr>
<tr>
<td>Education level:</td>
<td>% lower vocational education or less</td>
<td>2,187</td>
<td>46.7</td>
</tr>
<tr>
<td></td>
<td>% higher vocational education</td>
<td>1,098</td>
<td>23.4</td>
</tr>
<tr>
<td></td>
<td>% higher academic education</td>
<td>1,403</td>
<td>29.9</td>
</tr>
<tr>
<td>Single parent</td>
<td>% yes</td>
<td>480</td>
<td>10.3</td>
</tr>
<tr>
<td>Psychiatric problems</td>
<td>% high</td>
<td>757</td>
<td>16.0</td>
</tr>
<tr>
<td>BMI at enrollment</td>
<td>median, IQR</td>
<td>4,822</td>
<td>23.6 (4.7)</td>
</tr>
<tr>
<td>Parenting stress</td>
<td>% high</td>
<td>665</td>
<td>16.4</td>
</tr>
</tbody>
</table>

### CHILD CHARACTERISTICS

<table>
<thead>
<tr>
<th>Sex</th>
<th>% boys</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2,397</td>
</tr>
</tbody>
</table>

# 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 feed-
ing practices (Child Feeding Questionnaire), children’s emotional eating
(Children’s Eating Behaviour Questionnaire), and child body mass index
standard deviation scores (BMI-SDS)

<table>
<thead>
<tr>
<th>MATERNAL HISTORY OF EATING DISORDERS</th>
<th>B (95% CI)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FOR MATERNAL OUTCOMES: FEEDING PRACTICES</td>
</tr>
<tr>
<td></td>
<td>MONITORING</td>
</tr>
<tr>
<td></td>
<td>(N =3,753)</td>
</tr>
<tr>
<td></td>
<td>(N=3,752)</td>
</tr>
<tr>
<td></td>
<td>(N=3,760)</td>
</tr>
<tr>
<td>No eating disorder</td>
<td>0 [reference]</td>
</tr>
</tbody>
</table>
| Any eating disorder                 | 0.09 (-0.01; 0.20)  | 0.04 (-0.07; 0.15) | -0.20 (-0.32; -0.09)***

<table>
<thead>
<tr>
<th>MATERNAL HISTORY OF EATING DISORDERS</th>
<th>B (95% CI)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FOR CHILD OUTCOMES: EMOTIONAL UNDEREATING and BODY MASS INDEX</td>
</tr>
<tr>
<td></td>
<td>EMOTIONAL OVEREATING</td>
</tr>
<tr>
<td></td>
<td>(N=3,763)</td>
</tr>
<tr>
<td>No eating disorder</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>Any eating disorder</td>
<td>0.04 (-0.08; 0.15)</td>
</tr>
</tbody>
</table>

* 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, educa-
tional 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 dis-
orders 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.

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

* 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 or reporting higher levels of pressuring feeding strategies in these women. This inconsistency in results could be related to the assessment of pressure to eat. Stein et al., and Blisset and Haycraft 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. 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. 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).

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 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. 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, 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. 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. 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. 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. 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. 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.31 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.44 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,4,16 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.9 Given the high co-occurrence of eating disorders with anxiety and depression,8 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,45,46 our findings suggest that stress and psychopathology may underlie the previously reported association between maternal history of eating disorders and children’s emotional problems.18

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.35 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 Study33 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.19 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.28 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,19 but also of developing eating disorders as emotional eating has been related to eating psychopathology.10,11,43 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.
References


CHAPTER 2.3

Maternal history of eating disorders:
Children’s emotional and behavior problems

Manuscript based on this chapter:
Abstract

**Background:** Children of mothers with a history of eating disorders are at risk of developing disordered eating behaviors, and other psychopathology. The current study examines the longitudinal association between maternal history of eating disorders and children’s emotional and behavior problems.

**Methods:** Within the Generation R Study, a large Dutch population-based cohort, maternal lifetime history of eating and emotional disorders was assessed during pregnancy. When the children were 6 years old, mothers (n=4,739) and teachers (n=3,230) filled out questionnaires about children’s emotional and behavior problems. Linear regression analyses were performed, adjusting for sociodemographic confounders.

**Results:** Children of mothers with a history of eating disorders score 0.21SD higher on emotional problems, according to teachers (95% CI: 0.08; 0.33) and mothers (B=0.21SD, 95% CI: 0.12; 0.31). After additional adjustment for maternal history of anxiety or depression, maternal history of eating disorders remained associated with child emotional problems (e.g. teacher report: B=0.19, 95% CI: 0.06; 0.31). Maternal history of eating disorders was not related to child behavior problems.

**Conclusion:** Our results confirm previous literature showing that children of mothers with a history of eating disorders are at risk of developing emotional problems in the early school years. This could be indicative of a general vulnerability for psychopathology or a specific ‘marker’ of eating disorder risk, as emotional problems might precede the onset of eating psychopathology. Health care practitioners should ask mothers about their history of eating disorders and monitor their children carefully.
Introduction

It is well established that the offspring of mothers with psychiatric disorders are vulnerable to develop psychopathology themselves. Research studying children of mothers with mood, anxiety, and behavior disorders has indicated that these children are not only at risk of the same disorders as their parents suffered from, but also of discordant disorders. Children of mothers with eating disorders are less well studied, although two large studies have recently indicated that maternal eating disorders were prospectively related to both offspring’s eating disorders and other psychiatric disorders, particularly emotional disorders. Before the onset of these disorders, children may already experience emotional problems. Identifying whether the offspring of mothers with eating disorders indeed develop problems early in life may help preventing the onset of eating and emotional disorders, which typically manifest from late adolescence onwards.

The initial evidence for increased mental health problems among young children of mothers with eating disorders came from clinical case reports, often not including a control group. More recently, a few population-based studies supported these findings by showing higher levels of emotional problems in children of mothers with eating disorders. Some studies also reported behavior problems in children of mothers with a current or past eating disorder, but this finding was not confirmed in other studies. In addition, maternal history of eating disorders may affect girls differently than boys. In adolescence, the intergenerational transmission of eating disorders and depression is mostly observed from mothers to daughters. In childhood, some sex differences have been suggested by two European cohorts, but these differences were not statically tested.

An important limitation of some studies on the effects of maternal eating disorders is the use of maternal reports of child psychopathology only. This might affect results given the likelihood for a biased perception of child problems among mothers with psychiatric disorders. Cimino et al. aggregated mother and father reports of child problems, which may diminish informant bias, but gives no insight in different associations across informants. Micali et al. have provided some evidence for consistent associations between maternal history of eating disorders and offspring problems across informants, but more studies with multiple informants are needed.

Hence, we aimed to assess the association of maternal lifetime history of eating disorders with emotional problems in school-aged children using multiple informants. In addition, we explored the association of maternal eating disorders with child behavior problems, and possible interactions with child sex. Given the
high concurrence between eating and emotional disorders\textsuperscript{7,24} and the known effects of maternal anxiety and depression on child psychopathology,\textsuperscript{4,25} we also accounted for maternal history of emotional disorders.

**Methods**

**Study design**

This study was embedded in the Generation R Study, a population-based cohort of children who are followed from fetal life onwards. The design and procedure has been extensively described elsewhere.\textsuperscript{26,27} Briefly, this multi-ethnic cohort was designed to identify early determinants of children’s growth, development, and health, including emotional and behavior development. Pregnant women living in Rotterdam, the Netherlands, with a delivery date between April 2002 and January 2006 were invited to participate (baseline response rate: 61\%). The study has been conducted in accordance with the Helsinki guidelines and has been approved by the local medical ethical committee.

**Participants**

Participants with prenatal inclusion and full consent for follow-up were eligible for this study (n=7,510). Data on lifetime psychiatric disorders were available in 5,729 mother-child dyads. Of these, information on child emotional and behavior problems was provided for 5,177 children, by either teacher or mother: n=3,122 for teacher report and n=4,583 for mother report, of whom n=2,552 had information from both informants.

**Measures**

**Maternal history of eating disorders**

History of lifetime eating disorders was assessed by a self-report questionnaire during pregnancy, as previously explained in detail\textsuperscript{28} (see also chapter 2.1 and 2.2). Briefly, a short description of anorexia nervosa (AN) and bulimia nervosa (BN) was provided, followed by questions whether mothers ever had experienced these symptoms. Subsequent questions about past year prevalence, treatment history, and interference with daily life were used as additive information to define mothers’ lifetime history of eating disorders. Given the large cross-over and similar core-psychopathology between AN and BN,\textsuperscript{29,30} we categorized mothers into whether they had a
history of any eating disorder (yes / no). Self-reported eating disorders have been previously compared with diagnoses according to the Composite International Diagnostic Interview in a small sub-sample (n=928), indicating good validity of this self-report measure.28

**Child emotional and behavior problems**
Teacher reports on child problems were assessed with the internalizing (emotional problems) and externalizing (behavior problems) scales of the Teacher's Report Form (TRF, 6-18 years) when the children were approximately 6 years old.31 Likewise, mothers scored children's problems, using the Child Behavior Checklist (CBCL/1.5-5) when children were around 6 years of age.32 The TRF is the teacher version of the CBCL.31,32 The internalizing scale consists of respectively 33 (TRF) and 36 (CBCL) items covering a range of children's emotional problems (e.g. “worries”; “too shy or timid”; “whining”). The externalizing scale consists of respectively 32 (TRF) and 24 (CBCL) items covering a range of children's behavior problems (e.g. “can't sit still, restless or hyperactive”; “disobedient”). Items were rated on a scale from 0 (not true) to 2 (very true or often true). The items were summed to create scale scores. As in previous analyses,33 a maximum of 25% missing items was allowed for each scale. The scale scores were corrected for the number of completed items. Next, the scale scores were log transformed (TRF) and square root transformed (CBCL) to approach normality, and then z-standardized to enhance interpretation. Higher standard deviation (SD)-scores indicate more problems. The psychometric properties of the original and Dutch translations of the CBCL and TRF are well-established.31,32

**Covariates**
Maternal age, educational level and ethnic background, as well as household income were assessed by questionnaire during pregnancy26,27 and considered as possible confounders based on previous research.34-36 Child sex was obtained from medical records and considered as a potential effect modifier.5,20 We also adjusted for child age to control for variation in ages when the TRF and CBCL were filled out. Maternal lifetime history of emotional disorders was considered both as possible confounder and as potential effect modifier. Like the assessment of eating disorders, in a self-report questionnaire during pregnancy descriptions of both anxiety and depression were provided, and women were asked whether they had ever experienced these symptoms.23 Women were categorized into whether they had a history of emotional disorders (i.e. anxiety and/or depression) (yes / no).
Statistical analyses

To test whether a maternal history of eating disorders was related to child emotional and behavior problems, linear regression analyses were conducted. Four separate linear regression analyses were performed for teacher and mother reports of emotional and behavior problems. All analyses were performed unadjusted and adjusted for possible confounding by background characteristics. In a separate model, we additionally adjusted for maternal history of emotional disorders to test the specificity of the association between maternal history of eating disorders and child problems. To test for additive interaction, a product term between maternal history of eating disorders and emotional disorders was added to the model. Separately, another interaction term was included to test effect modification by child sex.

Several sensitivity analyses were performed. First, we explored the possible impact of the slightly skewed TRF distribution that remained after transformation. Therefore, logistic regression analyses were performed with child problems dichotomized at the 80th percentile of the entire Generation R sample to indicate elevated emotional or behavior problems, as done in previous analyses. Because our main analyses were performed in separate, only partially overlapping subsamples, we repeated the linear regression analyses in participants with complete data on both TRF and CBCL (n=2,552) to allow direct comparison.

Multiple imputation techniques were used to impute missing values of covariates (percentages missing ranged from 0% for maternal age to 13.6% for household income). The reported estimates reflect the pooled estimates from 20 imputed datasets. All statistical analyses were performed using SPSS version 21.0.

Results

Population characteristics

Table 2.3.1 shows that of the women in our study, approximately 8.7% had a history of eating disorders (4.1% had a history of eating disorders only, and 4.6% had a history of both eating disorders and emotional disorders).
<table>
<thead>
<tr>
<th>MATERNAL CHARACTERISTICS</th>
<th>SUBSAMPLE WITH DATA ON TEACHER REPORTED CHILD PROBLEMS (N=3,122)$^a$</th>
<th>SUBSAMPLE WITH DATA ON MOTHER REPORTED CHILD PROBLEMS (N=4,583)$^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N$^b$</td>
<td>MEDIAN/%</td>
</tr>
<tr>
<td>Age at enrollment</td>
<td>median years, interquartile range</td>
<td>3,122</td>
</tr>
<tr>
<td>Educational level</td>
<td>% high school or lower</td>
<td>680</td>
</tr>
<tr>
<td></td>
<td>% lower vocational training</td>
<td>931</td>
</tr>
<tr>
<td></td>
<td>% higher vocational or academic training</td>
<td>1,386</td>
</tr>
<tr>
<td>Ethnic background</td>
<td>% Dutch</td>
<td>1,711</td>
</tr>
<tr>
<td></td>
<td>% other, Western (mainly European)</td>
<td>258</td>
</tr>
<tr>
<td></td>
<td>% other, non-Western</td>
<td>1,118</td>
</tr>
<tr>
<td>Household income</td>
<td>% low (&lt; €2000 per month)</td>
<td>934</td>
</tr>
<tr>
<td>History of eating disorders</td>
<td>no eating disorder</td>
<td>2,860</td>
</tr>
<tr>
<td></td>
<td>any eating disorder</td>
<td>262</td>
</tr>
<tr>
<td></td>
<td>Values are percentages for categorical variables and medians (interquartile ranges) for continuous variables and represent original, non-imputed data.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>§∞ Data were missing on maternal: education (n=125$^§$ / n=116$^∞$), ethnic background (n=35$^§$), and income (n=424$^§$ / n=427$^∞$); and child age at TRF assessment (n=4$^§$).</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CHILD CHARACTERISTICS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SUBSAMPLE WITH DATA ON TEACHER REPORTED CHILD PROBLEMS (N=3,122)$^a$</td>
</tr>
<tr>
<td></td>
<td>N$^b$</td>
</tr>
<tr>
<td></td>
<td>median years, interquartile range</td>
</tr>
<tr>
<td>Sex</td>
<td>% boy</td>
</tr>
</tbody>
</table>
Maternal history of eating disorders and child emotional problems across informants

Children of mothers with a history of eating disorders had more emotional problems at age 6 years than children of mothers without a history of eating disorders, according to teachers (Table 2.3.2, B=0.21, 95% confidence interval [CI]: 0.08; 0.33) and mothers (B=0.21, 95% CI: 0.12; 0.31). After additional adjustment for maternal history of anxiety or depression, maternal history of eating disorders remained associated with child emotional problems (teacher report: B=0.19, 95% CI: 0.06; 0.31, mother report: B=0.13, 95% CI: 0.03; 0.23).

Children of mothers with a history of both eating and emotional disorders had the highest level of emotional problems (data not shown in a table; teacher report: B=0.34, 95% CI: 0.17; 0.15, mother report: 0.46, 95% CI: 0.34; 0.59), but there was no evidence for an interaction on the additive scale (p for interaction >0.05). We did not observe significant interactions with child sex either (p>0.05).

### TABLE 2.3.2 Maternal history of eating disorders and child emotional problems

<table>
<thead>
<tr>
<th>Maternal History of Eating Disorders</th>
<th>N</th>
<th>Teacher Reported Problems</th>
<th>Adjusted Model 1§</th>
<th>Adjusted Model 2∞</th>
</tr>
</thead>
<tbody>
<tr>
<td>No eating disorder</td>
<td>2,860</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>Any eating disorder</td>
<td>262</td>
<td>0.23 (0.10; 0.36)***</td>
<td>0.21 (0.08; 0.33)**</td>
<td>0.19 (0.06; 0.31)**</td>
</tr>
</tbody>
</table>

Mother Reported Problems

<table>
<thead>
<tr>
<th>Maternal History of Eating Disorders</th>
<th>N</th>
<th>Teacher Reported Problems</th>
<th>Adjusted Model 1§</th>
<th>Adjusted Model 2∞</th>
</tr>
</thead>
<tbody>
<tr>
<td>No eating disorder</td>
<td>4,170</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>Any eating disorder</td>
<td>413</td>
<td>0.21 (0.11; 0.31)***</td>
<td>0.21 (0.12; 0.31)***</td>
<td>0.13 (0.03; 0.23)***</td>
</tr>
</tbody>
</table>

** p<0.01, ***p<0.001.

§ Values reflect regression coefficients (95% confidence intervals).

∞ Adjusted for the child’s age when the questionnaire was filled out, household income, and maternal education, ethnic background, and age.

∞∞ Additionally adjusted for maternal history of emotional disorders.
Maternal history of eating disorders and child behavior problems across informants

Table 2.3.3 (adjusted model 1) shows that maternal history of eating disorders was not related to teacher or mother reported child behavior problems (e.g. teacher report: B=0.03, 95% CI: -0.09; 0.15). No significant interactions of maternal history of eating disorders by emotional disorders or of maternal history of eating disorders by child sex were found (p>0.05).

<table>
<thead>
<tr>
<th>Maternal History of Eating Disorders</th>
<th>N</th>
<th>Teacher Reported Problems</th>
</tr>
</thead>
<tbody>
<tr>
<td>No eating disorder</td>
<td>2,860</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>Any eating disorder</td>
<td>262</td>
<td>0.04 (-0.08; 0.17)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Maternal History of Eating Disorders</th>
<th>N</th>
<th>Teacher Reported Problems</th>
</tr>
</thead>
<tbody>
<tr>
<td>No eating disorder</td>
<td>4,170</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>Any eating disorder</td>
<td>413</td>
<td>0.07 (-0.03; 0.17)</td>
</tr>
</tbody>
</table>

# Values reflect regression coefficients (95% confidence intervals).  
§ Adjusted for the child’s age when the questionnaire was filled out, household income, and maternal education, ethnic background, and age.  
∞ Additionally adjusted for maternal history of emotional disorders.

Sensitivity analyses

Logistic regression analyses with elevated emotional problems (Supplementary Table 2.3.1) yielded very similar results as our main analyses. However, mothers with a history of eating disorder reported more behavior problems in their children (OR=1.28, 95% CI: 1.01; 1.62), while we found no association between maternal history of eating disorder and teacher reported behavior problems of children (OR=0.99, 95% CI: 0.70; 1.40). Similar results were observed in the subsample with complete data on both teacher and mother reports (n=2,552, Supplementary Table 2.3.2).
Discussion

Results from this population-based study suggest that children of mothers with a history of eating disorders are at risk of developing emotional problems. This effect was consistent across mothers and teachers as informants and was independent of maternal history of emotional disorders. Our results provide no evidence for an association between maternal history of eating disorders and child behavior problems.

Interpretations

The finding that mothers’ history of eating disorders predicts child emotional problems at age 6 years, is in line with several studies showing an association between mothers’ eating disorders and child emotional problems. Although children of mothers with a history of both eating and emotional disorders were particularly at risk of emotional problems, this reflected the sum of two risk factors, because we found no evidence for an additive interaction between maternal history of eating disorders and emotional disorders. No departure from additivity was also reported by the ALSPAC Study, a large population-based cohort in Britain, who were the first to test this, and our findings support this.

Maternal history of eating disorders specifically affected emotional problems in 6-year-old children. This might be partly attributed to shared heritability between eating disorders and emotional disorders, but could also indicate parenting practices that specifically affect the development of emotional problems or modeling of mother’s behaviors and thoughts. For instance, mothers with a history of eating disorders might still suffer from perfectionism and negative self-schemas, may tend to attribute negative events to internal, global and stable causes, and possibly cope with negative emotions by ruminating. Consequently, their children might copy these specific vulnerabilities for emotional problems by modelling their mothers’ reaction and coping strategies when facing negative life events. Mothers could also pass on their attributional style by communicating how they interpret the causes and consequences of negative events in the child’s life. Although the reported association between maternal history of eating disorders and child emotional problems was largely independent of maternal history of depression and anxiety, the magnitude of the association slightly attenuated after adjustment for maternal emotional disorders. It is thus not unlikely that mothers share common vulnerabilities, which they may pass on to their children. Recent studies have, for instance, indicated that shame and fear of negative evaluation could be vulnerabilities for both social anxiety and eating disorder symptoms.
The specific effect of maternal eating disorders on child emotional problems rather than on behavior problems is consistent with some studies, but contrasts others. The association of maternal history of eating disorders with child emotional problems is likely to reflect a main effect of any eating disorder history on all children, as this is mostly found for both daughters and sons of mothers with different types of eating disorders. However, the association with behavior problems was found in specific subgroups; for instance, conduct problems were particularly reported in children of mothers with a history of BN. In one study, hyperactivity problems were only found in children of mothers with a history of BN, although another study also reported hyperactivity problems children of mothers with a history of AN, but only in daughters. These findings could reflect true differences between specific subgroups of eating disorders, but may also be attributed to multiple testing. Findings of more behavior problems in children of mothers with a history of eating disorders may also be attributed to biased perception of these mothers, as some studies relied solely on mother reports. This is strengthened by our sensitivity analyses showing that mothers with a history of eating disorders reported higher behavior problems in their child, while this was not observed by teachers. Another explanation for differences between our finding and some studies observing higher behavior problems in children of mothers with eating disorders could be eating disorder status. It is possible that active eating psychopathology – as was the case in the mothers of Cimino’s study while we studied history of eating disorders – is related to disturbances in parenting that also affect behavior problems. Future studies are needed to elucidate the different findings between maternal history of eating disorders and children’s behavior problems.

**Strengths and limitations**

The main strength of this population-based study was the inclusion of multiple informants. We have previously shown that mothers with a history of anxiety or depression tend to over-report their children’s emotional problems. Most likely, mothers with anxiety or depression have a distorted view of their children’s emotional problems. Alternatively, teachers may be less able of picking up child emotional problems. If so, one would expect that the association between maternal history of eating disorders and child emotional problems would be less strong when teachers reported the children’s problems as compared to mother reports. However, this was not the case, as associations between maternal history of eating disorders and child emotional problems were consistent across informants. This strengthens the validity of our findings and suggests that mothers with a history of eating disorders are perhaps less biased in rating their children’s emotions than mothers.
who have a history of anxiety and depression. However, results from our sensitivity analyses suggests that mothers with a history of eating disorders tend to over-report behavior problems of their children, which stresses the importance of using a multiple informant approach.

The results of this study should not only be interpreted within its strengths, but also within its limitations. The classification of eating disorder history was based on self-report, which may be prone to bias. For instance, asking women whether they have ever tried to lose weight to the extent that they may have suffered from anorexia nervosa could 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. Such potential misclassification could have contributed to an underestimation of the associations. However, substantial overlap between self-report and clinical diagnoses of eating disorders has been observed in a subsample of our cohort.\textsuperscript{28} This supports the use of self-reports, although our findings should preferably be replicated in a larger sample with more past and current cases of clinical eating disorders. Further, although our findings were largely consistent between informants suggesting a ‘true’ effect of maternal history of eating disorders on child emotional problems, one should keep in mind that mothers and teachers are not completely independent informants. While not likely to fully explain our results, a mother with a distorted perception of her child’s wellbeing might influence the teacher’s perception by expressing her concerns. In addition, because we used the preschool form of the CBCL and school form of the TRF, the small differences between mother and teacher reports cannot only be attributed to the reporters and the situations they report on but could also be due to subtle differences in the questionnaires.

Conclusions

Children of mothers with a history of eating disorders are at risk of developing emotional problems in the early school years. This could be indicative of the first signs of a developing eating disorder, since emotional problems might precede eating disorder psychopathology.\textsuperscript{7,8} These children may also face mood disorders later in life.\textsuperscript{9} From a clinical perspective, it is important to unravel specific mechanisms other than genetics that could be targeted in intervention programs, for instance parenting or cognitive vulnerabilities. Our observation that both boys and girls were affected by their mothers’ history of eating and emotional disorders needs to be confirmed, but suggests that sons of mothers with such history should not be overlooked when screening for children at risk. Health care practitioners could consider asking mothers whether they ever have experienced an eating disorder in order to monitor their children carefully for disturbances in the emotional development.
References


Supplement

SUPPLEMENTARY TABLE 2.3.1  Effect of maternal eating disorders on child risk of elevated emotional and behavior problems

<table>
<thead>
<tr>
<th>MATERNAL HISTORY OF EATING DISORDERS</th>
<th>CHILD EMOTIONAL PROBLEMS– OR (95% CI)(^#)(^$)</th>
<th>(N^\infty)</th>
<th>TEACHER REPORTED</th>
<th>(N^\infty)</th>
<th>MOTHER REPORTED</th>
</tr>
</thead>
<tbody>
<tr>
<td>No eating disorder</td>
<td></td>
<td>2,860</td>
<td>1 [reference]</td>
<td>4,170</td>
<td>1 [reference]</td>
</tr>
<tr>
<td>Any eating disorder</td>
<td></td>
<td>262</td>
<td>1.49 (1.11; 2.01)**</td>
<td>413</td>
<td>1.44 (1.14; 1.84)**</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CHILD BEHAVIOR PROBLEMS – OR (95% CI)(^#)(^$)</th>
<th>(N^\infty)</th>
<th>TEACHER REPORTED</th>
<th>(N^\infty)</th>
<th>MOTHER REPORTED</th>
</tr>
</thead>
<tbody>
<tr>
<td>No eating disorder</td>
<td>2,860</td>
<td>1 [reference]</td>
<td>4,170</td>
<td>1 [reference]</td>
</tr>
<tr>
<td>Any eating disorder</td>
<td>262</td>
<td>0.99 (0.70; 1.40)</td>
<td>413</td>
<td>1.28 (1.01; 1.62)*</td>
</tr>
</tbody>
</table>

\* \(p<0.05\), ** \(p<0.01\).

\# Values reflect odds ratios (95% confidence intervals) for elevated emotional or behavior problems (cut-off at 80th percentile of entire Generation R population).

\$ Adjusted for the child’s age when the questionnaire was filled out, household income, and maternal education, ethnic background, and age.

\(\infty\) Cases emotional problems: \(n=608\ (19.5\%, \text{score}>5)\) by teacher report, and \(n=879\ (19.2\%, \text{score}\geq10)\) by mother report.

\(\Omega\) Cases reported behavior problems: \(n=520\ (16.7\%, \text{score}>5)\) by teacher report, and \(n=947\ (20.7\%, \text{score}\geq12)\) by mother report.

SUPPLEMENTARY TABLE 2.3.2  Sensitivity analyses in subsample with complete data on both teacher and mother reported emotional problems

<table>
<thead>
<tr>
<th>MATERNAL HISTORY OF EATING DISORDERS</th>
<th>CHILD EMOTIONAL PROBLEMS – B (95% CI) PER SD(^#)(^$)</th>
<th>(N)</th>
<th>TEACHER REPORTED</th>
<th>MOTHER REPORTED</th>
</tr>
</thead>
<tbody>
<tr>
<td>No eating disorder</td>
<td></td>
<td>2,347</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>Any eating disorder</td>
<td></td>
<td>205</td>
<td>0.23 (0.10; 0.37)**</td>
<td>0.26 (0.12; 0.39)**</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CHILD BEHAVIOR PROBLEMS – B (95% CI) PER SD(^#)(^$)</th>
<th>(N)</th>
<th>TEACHER REPORTED</th>
<th>MOTHER REPORTED</th>
</tr>
</thead>
<tbody>
<tr>
<td>No eating disorder</td>
<td>2,347</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>Any eating disorder</td>
<td>205</td>
<td>0.06 (-0.07; -0.19)</td>
<td>0.15 (0.01; 0.28)*</td>
</tr>
</tbody>
</table>

\* \(p<0.05\), ** \(p<0.01\), ***\(p<0.001\).

\# Values reflect regression coefficients (95% confidence intervals).

\$ Adjusted for the child’s age when the questionnaire was filled out, household income, and maternal education, ethnic background, and age.
Infant feeding &
Child picky eating

Manuscript based on this chapter:
Abstract

**Background:** Picky/fussy eating – i.e. consistently avoiding certain foods – is common in childhood and can be worrisome for parents. Repeated exposure to various flavors as occurs in breastmilk and early exposure to complementary feeding may increase food acceptance and thereby decrease picky eating. This study examines the associations of breastfeeding duration and timing of complementary feeding with child picky eating in 4,779 participants of Generation R, a Dutch population-based cohort.

**Methods:** Mothers received postal questionnaires at 2, 6, and 12 months assessing breastfeeding initiation and continuation, and the age at which mothers introduced any solid food, vegetables, and fruit to their children. The food fussiness scale of the Children’s Eating Behaviour Questionnaire was administered at 4 years. Linear regression analyses were performed, adjusted for potential confounders.

**Results:** Children who were never breastfed did not differ in picky eating frequency from children breastfed for 6 months or longer. However, children who were breastfed for less than 2 months had a 0.70 points higher food fussiness sum-score (95% CI: 0.27; 1.12) than children breastfed for 6 months or longer. An earlier introduction of vegetables was associated with less picky eating behavior (p-for-trend: 0.005). In particular, children who were introduced to vegetables between 4-5 months had a 0.60 point lower food fussiness score (95% CI: -1.06; -0.15) than children introduced to vegetables after 6 months. We observed the same trend for an early introduction to fruits or any solids, although less strong and not significant.

**Conclusions:** Results suggest that breastfeeding does not predict picky eating. However, introducing vegetables into a child’s diet before 5 months might be protective against picky eating, although future research should account for parents’ own picky eating.
Introduction

Picky eating behavior is common in toddlerhood and is characterized by the frequent rejection of familiar and unfamiliar food items, consequently leading to a limited dietary variety.\(^1\) Parents are often concerned about their children’s picky eating,\(^2\) but etiological research that could help parents and health care practitioners understand and potentially prevent picky eating is scarce.\(^3\)

Repeated exposure to a variety of flavors – as occurs in breastmilk, but not in formula milk\(^4\) – is associated with greater acceptance of food items\(^4,5\) and might prevent picky eating.\(^6-8\) A longer breastfeeding duration exposes infants to a variety of flavors and can therefore enhance initial acceptance of solid food items,\(^4,9\) and may facilitate later dietary variety\(^7,9\) and reduce food rejections.\(^6,10\) However, some of these studies did not control for potential confounding\(^8,10\) or were performed in small samples.\(^6,8,10\) In a larger population-based study,\(^11\) breastfeeding duration was not related to toddler’s picky eating.

The timing of exposure to complementary feeding could also be important for food acceptance.\(^9,12,13\) From an evolutionary perspective, it would be most adaptive to have a short sensitive period very early in life in which children learn what is edible and what is not.\(^12\) This ‘food learning’ period is likely to happen when infants are fed by parents who select safe foods for them to consume. Once children become mobile, there is a greater risk of consuming dangerous substances if children continue to eat everything within their reach. Thus, after the sensitive period of food learning, more aversive reactions to unknown tastes could be expected.\(^1,12\) Hence, a late introduction to solids could interfere with this food learning period, creating feeding problems.\(^12\) In the domain of texture, this hypothesis is supported by findings from the ALSPAC study, where a late introduction to lumpy solids (after 10 months) was associated with an increased risk of picky eating at 15 months\(^14\) and feeding problems at 7 years\(^15\). Others did not observe any association between timing of complementary feeding and picky eating.\(^16\) Moreover, early complementary feeding (before 6 months) has also been related to more picky eating.\(^6\) It is possible that the type of food introduced is important for picky eating. Young children may particularly need to learn that vegetables are edible, given the evolutionary tendency to avoid vegetable consumption, because of toxic compounds in plants.\(^7\) Having tried a greater number of vegetables early in life has been related to less picky eating behavior.\(^18\)

We hypothesized that a longer duration of any breastfeeding as well as early complementary feeding, particularly with vegetables, would be related to less picky eating at 4 years, and studied these associations within a large, prospective cohort.
Methods

Study design

This study was embedded in the Generation R Study, an ongoing prospective cohort investigating growth, development, and health from fetal life onwards. Pregnant women living in Rotterdam, the Netherlands, with an expected delivery date between April 2002 and January 2006 were invited to participate (baseline response rate 61%). Assessments for the current study mainly included parent-report questionnaires. The local Medical Ethical Committee has approved the study.

Participants

Parents of 7,295 children gave full consent for the preschool phase of the Generation R Study. Children with missing data on infant feeding and children’s picky eating were excluded for the current analyses, yielding a sample size of 4,779 children. The different exposure data were not complete for all participants, thus the population for analyses varied per exposure (sample per exposure are mentioned in the relevant tables).

Measures

Breastfeeding duration

Information on breastfeeding initiation and continuation was obtained from delivery reports and postnatal questionnaires at the ages of 2 months, 6 months, and 12 months, as previously described. Mothers were asked whether they ever breastfed their child (yes/no). In breastfed children, duration of breastfeeding was assessed by asking mothers at what age they stopped breastfeeding and categorized as (1) >0-2 months; (2) 2-4 months; (3) 4-6 months; and (4) ≥6 months.

Timing of complementary feeding

Information on the introduction of any solid food was obtained from two short questionnaires at 6 months and 12 months in which mothers were asked at what age they introduced certain food items or food groups to their child. Timing of introduction of solid foods was defined as the earliest age at which any solid food was given for the first time, and was categorized into the following 4 groups: (1) <4 months; (2) 4-5 months; (3) 5-6 months; and (4) ≥6 months.

Information on the introduction of vegetables and fruits was obtained by questionnaire at 6 months. Mothers were asked at what age they introduced vege-
table puree and fruit puree to their child. Timing of introduction of vegetables and fruits was defined (two separate variables) and categorized similar to the timing of introduction of any solid food.22

**Picky eating behavior in children**

The food fussiness scale of the Children’s Eating Behaviour Questionnaire (CEBQ) was used to assess picky eating.23 When the children were 4 years old, parents filled out the CEBQ, a validated, multi-dimensional questionnaire to identify differences in children’s eating behaviors. The CEBQ consists of eight scales, each containing 3 to 6 items (in total 35 items) on which parents rated the occurrence of their child’s specific eating behaviors on a Likert scale from 1 (never) to 5 (always). The food fussiness scale consists of 6 items assessing whether children are difficult to please with meals, display food neophobia (e.g. “My child refuses new foods at first”), or have a limited diet variety (e.g. “My child enjoys a wide variety of foods”, reverse coded). The items were summed resulting in a scale sum-score from 6 to 30 with higher scores indicating more picky eating behavior. Previous research has reported that the CEBQ has good psychometric properties.23

**Covariates**

Covariates were selected based on previous research.3-6,7,11,24-28 Parental characteristics that were considered included maternal age, net household income, maternal educational level, maternal ethnic background, single parenthood, family history of asthma/house dust mite allergy/hay fever, parity, and maternal psychiatric symptoms (overall score of the Brief Symptom Inventory, BSI)29 and family malfunctioning during pregnancy (overall score on the General Functioning scale of the McMaster Family Assessment Device [FAD], covering for instance experiencing low spouse support).30,31

Child characteristics comprised age at CEBQ assessment, sex, gestational age, birth weight-SDS, hospitalization in the first week of life, hours spent in non-parental child-care at 6 months,32 infants’ overall problem score for crying, sleeping problems and bowel pain (sum-score of 3 items, assessed at 2 months postnatal), infant temperamental difficulties in the sadness (general low mood) and fear (distress and inhibited response to novelty) dimensions of the Infant Behavior Questionnaire-Revised33 that was adapted for our study.34 As a proxy for emotional and behavior problems in infancy, the overall problem score of the Child Behavioral Checklist 1½-5 at 18 months was used.35

Initial feeding difficulties that may later develop into picky eating11,28 may also induce mothers to stop breastfeeding or postpone introducing solids. Thus, to control for potential reversed causality, we additionally included child refusal of breast-
feeding (yes/no), drinking slow or small quantities (sum-score of 2 items), and general feeding problems (“how often does your child have problems with feeding?”), all assessed by questionnaire at 2 months of age.

**Statistical analyses**

We first examined the associations between breastfeeding duration and children’s picky eating. The difference in food fussiness score between children who were never breastfed and those with recommended breastfeeding duration (i.e. 6 months or longer)\(^{36}\) was tested with linear regression analyses. Then we analyzed the association between breastfeeding duration in months and children’s food fussiness sum-score with linear regression analyses, first in all children and then only in the children who received breastfeeding (n=3527). Similar analyses were conducted to examine whether picky eating differed across the following categories of breastfeeding duration: (1) >0-2 months; (3) 2-4 months; (3) 4-6 months; and (4) ≥6 months (reference group).

Second, linear regression analyses were conducted to test whether food fussiness scores differed across children who were introduced to any type of complementary feeding at various ages: (1) <4 months; (2) 4–5 months; (3) 5-6 months; and (4) ≥6 months (reference). Subsequently, we studied the associations between timing of introduction of specific types of food (fruit and vegetables) and later picky eating. Trend tests were performed, using the timing categories as a continuous variable.

All analyses were conducted unadjusted and adjusted for potential confounders that changed the univariate effect estimates by 10% or more.\(^{37}\) Following this criterion, parity, hospitalization, single parenthood, difficult temperament (fear scale), and family history of asthma/house dust mite allergy/hay fever were excluded from the analyses. Potential reversed causality due to initial feeding problems was tested in separate models, additionally adjusting for early feeding problems.

Finally, we performed several sensitivity analyses. First, the analyses on breastfeeding duration were additionally adjusted for timing of complementary feeding. Likewise, the analyses on complementary feeding were additionally adjusted for breastfeeding duration. Second, we checked for a potential interaction effect between breastfeeding duration and timing of complementary feeding (any solid food; vegetables; and fruit). Last, we repeated our main analyses in Dutch children only, to address potential residual confounding by ethnic background.

Missing values on covariates were imputed using multiple imputation (predictive mean matching method).\(^{38,39}\) Percentage of missing values ranged from 0% to 30.9%. The reported effect estimates are pooled estimates of 20 imputed datasets. All statistical analyses were performed with SPSS 21.0 (IBM Corp., Armonk, NY, USA).
Results

Population characteristics

TABLE 3.1.1 General characteristics of 4,779 Generation R participants included in this study

<table>
<thead>
<tr>
<th>CHILD CHARACTERISTICS</th>
<th>MEAN (SD), MEDIAN (IQR), OR PERCENTAGE#</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at CEBQ assessment median months</td>
<td>4,799 48.6 (0.9)</td>
</tr>
<tr>
<td>Sex % girls</td>
<td>2,403 50.3</td>
</tr>
<tr>
<td>Birth weight mean grams</td>
<td>4,776 3448</td>
</tr>
<tr>
<td>Gestational age median weeks</td>
<td>4,733 40.1</td>
</tr>
<tr>
<td>Hours in non-parental day care at 6 months % ≥ 8 hours a week</td>
<td>3,334 69.8</td>
</tr>
<tr>
<td>Crying, sleeping, or bowel pain at 2 months mean problem score</td>
<td>4,779 5.6 (1.7)</td>
</tr>
<tr>
<td>Sadness dimension of temperament mean IBQ-R score</td>
<td>4,779 0.66 (0.29)</td>
</tr>
<tr>
<td>Emotional &amp; behavior problems at 18 months median CBCL score</td>
<td>4,779 20 (19)</td>
</tr>
<tr>
<td>Refusal of breastfeeding at 2 months % yes</td>
<td>101 2.1</td>
</tr>
<tr>
<td>Drinking slow or small quantities at 2 months % yes</td>
<td>897 18.8</td>
</tr>
<tr>
<td>General feeding problems at 2 months median score</td>
<td>4,779 1 (1)</td>
</tr>
<tr>
<td>Food fussiness at 48 months mean CEBQ sum-score</td>
<td>4,779 17.7 (4.9)</td>
</tr>
</tbody>
</table>

MATERNAL CHARACTERISTICS

<table>
<thead>
<tr>
<th>MATERNAL CHARACTERISTICS</th>
<th>MEAN (SD), MEDIAN (IQR), OR PERCENTAGE#</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at enrollment mean years</td>
<td>4,779 31.6 (4.5)</td>
</tr>
<tr>
<td>Educational level % low**</td>
<td>697 14.6</td>
</tr>
<tr>
<td>% medium**</td>
<td>1,331 27.9</td>
</tr>
<tr>
<td>% high**</td>
<td>2,751 57.5</td>
</tr>
<tr>
<td>Ethnic background % Dutch</td>
<td>3,100 64.9</td>
</tr>
<tr>
<td>% Moroccan</td>
<td>144 3.0</td>
</tr>
<tr>
<td>% Surinamese &amp; Dutch Antillean</td>
<td>350 7.3</td>
</tr>
<tr>
<td>% Turkish</td>
<td>279 5.8</td>
</tr>
<tr>
<td>% other, Western (mainly European)</td>
<td>420 8.8</td>
</tr>
<tr>
<td>% other, non-Western</td>
<td>486 10.2</td>
</tr>
<tr>
<td>Psychiatric symptoms median overall BSI score</td>
<td>4,779 0.13 (0.23)</td>
</tr>
<tr>
<td>Net household income % low (&lt; €2000 per month)</td>
<td>1,298 27.2</td>
</tr>
<tr>
<td>General family malfunctioning FAD score</td>
<td>4,779 1.42 (0.67)</td>
</tr>
</tbody>
</table>

\# Values are percentages for categorical variables, means (standard deviations) for continuous normally distributed variables and medians (interquartile ranges) for continuous non-normally distributed variables.

\§ N=4,779 as this table represents imputed data for covariates.

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

General characteristics of the study population are presented in Table 3.1.1. Of all mothers, 90.7% breastfed their child. Of these, the mean duration of breastfeeding was 5.1 months (standard deviation [SD]=3.8 months). The median age at which children received any solid food or fruits for the first time was between 4 and 5 months. Vegetables were introduced, on average, somewhat later, between 5 and 6 months.

**Breastfeeding duration and child picky eating**

We observed a dose-response relation between breastfeeding duration and picky eating (B= -0.06, 95% CI: -0.10; -0.02, Table 3.1.2). However, never breastfed children did not differ in their food fussiness score from children with a recommended breastfeeding duration (≥6 months). Also, children who were breastfed between 2 and 4 months or between 4 and 5 months did not differ from children who were breastfed for at least 6 months either. Only children who were breastfed for a short period of time (between 1 day and 2 months) had 0.70 points higher food fussiness score (95% CI: 0.27; 1.12) than children with a breastfeeding duration of 6 months or longer. Results were very similar (B=0.66; 95% CI: 0.23; 1.09) after adjustment for initial feeding problems (data not shown in tables).

Sensitivity analyses showed that additionally adjusting the analyses on breastfeeding duration for complementary feeding did not meaningfully change the results (see supplementary Tables 3.1.1). We did not observe an interaction effect between breastfeeding duration and timing of complementary feeding (all p-values for interactions>0.05). Last, when restricting the analyses to Dutch children, the associations between breastfeeding and picky eating were very similar to the main findings (supplementary Table 3.1.2).
TABLE 3.1.2  Breastfeeding and picky eating in 4-year-old children

<table>
<thead>
<tr>
<th>IN ALL CHILDREN</th>
<th>FOOD FUSSINESS SUM-SCORE§</th>
<th>N</th>
<th>MODEL 1: B (95% CI) #</th>
<th>MODEL 2: B (95% CI) **</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never breastfeeding</td>
<td>362</td>
<td>-0.13 (-0.70; 0.45)</td>
<td>0.19 (-0.43; 0.81)</td>
<td></td>
</tr>
<tr>
<td>Versus recommended (≥ 6 months)</td>
<td>1,301</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
<td></td>
</tr>
<tr>
<td>Breastfeeding duration continuously (in months)</td>
<td>3,889</td>
<td>-0.04 (-0.08; -0.00) *</td>
<td>-0.06 (-0.10; -0.02) **</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>IN BREASTFED CHILDREN ONLY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breastfeeding duration continuously (in months)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>BREASTFEEDING DURATION IN CATEGORIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;0 – 1.9 months</td>
</tr>
<tr>
<td>2 – 3.9 months</td>
</tr>
<tr>
<td>4 – 5.9 months</td>
</tr>
<tr>
<td>≥ 6 months</td>
</tr>
</tbody>
</table>

* p<0.05, **p<0.01.
§ Food fussiness sum-score: range 6-30.
# Unstandardized regression coefficients (95% confidence interval).
** Model 1 is unadjusted. Model 2 is adjusted for child: age, sex, birth weight-SDS, gestational age, hours spent in non-parental day care, problems with crying, sleeping or bowel pain, difficult temperament, and emotional & behavior problems; for maternal: age, education, ethnic background, and psychiatric symptoms; and for family: income, and malfunctioning.

Timing of complementary feeding and child picky eating

Earlier introduction of vegetables was related to less picky eating behavior (Table 3.1.3, p-for-trend: 0.005). Particularly children who were already introduced to vegetables between 4 and 5 months had a lower food fussiness score (B= -0.60, 95%CI: -1.06; -0.15) than children who were introduced to vegetables at 6 months or later. An early introduction to fruits or to any solid food was also related to less picky eating, but the effect estimates were smaller and did not reach statistical significance (Table 3.1.3). Adjustment for initial feeding problems (data not shown) or breastfeeding duration (including the never breastfed children, see Supplementary Table 3.1.3) yielded similar results. When restricting the analyses to Dutch children, we obtained similar results (Supplementary Table 3.1.2).
### TABLE 3.1.3  
Timing of complementary feeding and picky eating in 4-year-old children

<table>
<thead>
<tr>
<th>TIMING OF INTRODUCING ANY SOLIDS</th>
<th>N</th>
<th>MODEL 1: B (95% CI)</th>
<th>MODEL 2: B (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 4 months</td>
<td>350</td>
<td>0.55 (-1.20; 0.11)</td>
<td>0.46 (-1.12; 0.19)</td>
</tr>
<tr>
<td>4 – 4.9 months</td>
<td>2,110</td>
<td>-0.26 (-0.71; 0.20)</td>
<td>-0.21 (-0.66; 0.24)</td>
</tr>
<tr>
<td>5 – 5.9 months</td>
<td>772</td>
<td>-0.12 (-0.65; 0.41)</td>
<td>-0.13 (-0.66; 0.40)</td>
</tr>
<tr>
<td>≥ 6 months (reference)</td>
<td>560</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>p-for-trend</td>
<td></td>
<td>0.09</td>
<td>0.17</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TIMING OF INTRODUCING FRUIT</th>
<th>N</th>
<th>MODEL 1: B (95% CI)</th>
<th>MODEL 2: B (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 4 months</td>
<td>205</td>
<td>-0.49 (-1.38; 0.40)</td>
<td>-0.45 (-1.35; 0.45)</td>
</tr>
<tr>
<td>4 – 4.9 months</td>
<td>1,980</td>
<td>-0.32 (-0.96; 0.31)</td>
<td>-0.36 (-0.98; 0.28)</td>
</tr>
<tr>
<td>5 – 5.9 months</td>
<td>878</td>
<td>-0.02 (-0.70; 0.66)</td>
<td>-0.14 (-0.81; 0.53)</td>
</tr>
<tr>
<td>≥ 6 months (reference)</td>
<td>253</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>p-for-trend</td>
<td></td>
<td>0.08</td>
<td>0.14</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TIMING OF INTRODUCING VEGETABLES</th>
<th>N</th>
<th>MODEL 1: B (95% CI)</th>
<th>MODEL 2: B (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 4 months</td>
<td>101</td>
<td>-0.77 (-1.79; 0.26)</td>
<td>-0.66 (-1.68; 0.35)</td>
</tr>
<tr>
<td>4 – 4.9 months</td>
<td>1,449</td>
<td>-0.64 (-1.10; -0.18)</td>
<td>** -0.60 (-1.06; -0.15)</td>
</tr>
<tr>
<td>5 – 5.9 months</td>
<td>1,184</td>
<td>-0.21 (-0.68; 0.27)</td>
<td>-0.24 (-0.71; 0.23)</td>
</tr>
<tr>
<td>≥ 6 months (reference)</td>
<td>612</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>p-for-trend</td>
<td></td>
<td>0.002</td>
<td>0.005</td>
</tr>
</tbody>
</table>

* p < 0.05, **p < 0.01.
§ Food fussiness sum-score: range 6–30.
# Unstandardized regression coefficients (95% confidence interval).
∞ Model 1 is unadjusted. Model 2 is adjusted for child: age, sex, birth weight-SDS, gestational age, hours spent in non-parental day care, problems with crying, sleeping or bowel pain, difficult temperament, and emotional & behavior problems; for maternal: age, education, ethnic background, and psychiatric symptoms; and for family: income, and malfunctioning.
Discussion

Given the proposed mechanism of repeated exposure to different flavors in breastmilk, we expected to particularly find differences in picky eating between children who were never breastfed and who were breastfed for prolonged periods of time. In contradiction to this hypothesis, however, never breastfed children in our study did not appear to differ in picky eating from children breastfed for at least 6 months. Therefore, our finding that a short breastfeeding duration (between 1 day and 2 months) was related to more picky eating is not likely to be explained by repeated flavor exposure, but rather by reverse causation (i.e. early feeding difficulties that induced breastfeeding cessation). Our analyses with the timing of complementary feeding suggests that particularly an early introduction to vegetables may protect against picky eating.

Breastfeeding duration and child picky eating

In our population-based study, there was no difference between never-breastfed children and children breastfed for at least 6 months in food fussiness scores at age 4 years. This is in contrast with some reports indicating that children breastfed for longer are more likely to have optimal dietary variety and are less likely to be picky eaters. The results of another large population-based study, however, are in line with our observation: McDermott et al. also found no differences in picky eating at the age of 2 to 4 years between never-breastfed children and children who had been breastfed for 4 months or longer. Researchers have suggested that repeated flavor exposure through breastmilk might particularly facilitate food acceptance at the start of complementary feeding, but may be less important when children have already been exposed to solid foods for a longer time. This could explain why breastfeeding duration did not seem to affect picky eating in the 4-year-olds in our study.

In the absence of a difference in picky eating between never-breastfed children and those with the longest, recommended breastfeeding duration – between whom we expected the strongest contrast – our finding that a short breastfeeding duration (between 1 day and 2 months) was associated with more picky eating behavior is likely to be explained by reverse causation. That is, infants who become picky eaters in toddlerhood may already have been difficult eaters in infancy, for instance sucking less frequently during feeding, or rejecting breastfeeding more often. Such aberrant eating behaviors may induce mothers to stop breastfeeding at an early stage. Nevertheless, when we adjusted for initial feeding problems at 2 months, a short breastfeeding duration remained related to picky eating. Informa-
tion on initial feeding problems was, however, assessed using very few questionnaire items, limiting the extent to which reverse causation can be addressed.

A possible alternative explanation for the absence of a difference in picky eating between never-breastfed children and children breastfed for 6 months is the presence of residual confounding bias in the never-breastfed children group. This might be a distinct group, as women who have never initiated breastfeeding tend to have a lower level of education than women who do start giving breastmilk, as demonstrated previously in this same cohort. Although we carefully adjusted for maternal education and household income, this correction may not have captured all socioeconomic differences, because of possible errors in measurement or categorization, or due to missing information. If we did not observe an association between never-breastfeeding and child picky eating because of residual confounding, the finding that children with a short breastfeeding duration are more likely to have an increased food fussiness score might indicate a protective effect of prolonged breastfeeding against picky eating, with as proposed mechanism the resultant exposure to a variety of flavors.

Another explanation for our finding that children who were breastfed for a short time had higher levels of picky eating could be residual confounding in the shortly-breastfed group by unmeasured or inadequately measured factors, such as controlling parenting or maternal psychiatric symptoms. For instance, a systematic review has shown that maternal depression during pregnancy does not affect breastfeeding initiation, but rather predicts a short breastfeeding duration. Indeed, we have shown that maternal anxiety and depressive symptoms are also related to child picky eating (chapter 3.2). Although here we adjusted for maternal psychopathology using a validated instrument, our assessment may not have captured all variation in psychiatric symptoms.

**Timing of complementary feeding and picky eating**

Children to whom vegetables were introduced early on had lower levels of picky eating behavior than children introduced to vegetables from 6 months on. Our results suggest that introducing vegetables to a child’s diet early on is important. This is supported by findings from Lange et al. who showed that an early introduction to vegetables predicts children’s acceptance of new vegetables. A recent intervention study has also found that 4-to-6-month-old infants who received vegetables as the first solid food followed by a period of exclusive exposure to vegetables had a higher vegetable intake after 18 days than infants who were exclusively exposed to fruit, although a long-term effects on vegetable intake at 2 years follow-up was not observed.
The associations that we found between the timing of introducing fruits or any solid food and picky eating were in the same direction as those found for timing of introducing vegetables and picky eating, but were less strong and not statistically significant. It should be noted that the associations reported by Shim et al. are in the opposite direction, with the introduction of complementary feeding before 6 months predicting more picky eating. However, their small sample (n=129) and retrospective design makes their findings debatable. Moreover, our results are in line with previous reports of an association between a late introduction of solids or lumpy solids (later than 6 or 10 months) and more picky eating. Another large prospective cohort in the Netherlands has also found that introducing complementary feeding after 6 months is related to less enjoyment of food, suggesting an effect in the same direction.

The beneficial effect of an early introduction to vegetables on the risk of picky eating is consistent with Cashdan’s hypothesis regarding a sensitive period for ‘food learning’. Children who learn early on in life that vegetables are edible might be less likely to refuse vegetables later on in childhood. An alternative explanation is that our results are confounded by parents’ picky eating. In other words, the parents of children who are picky eaters might also be picky eaters and may therefore be less likely to introduce vegetables and other disliked foods to their child early on.

**Strengths and limitations**

The strengths of our study are its large population-based sample, its prospective design, the repeated measurements of breastfeeding practices, and the information on multiple types of solids, the latter allowing us to study the effects of the timing of introducing different food groups separately. However, as mentioned above, our observational study may have had limited possibilities to examine reverse causality and may have been hindered by residual confounding. While only a randomized controlled trial might better cope with these phenomena, it is unethical to randomly assign participants to a non-breastfeeding group because of the strong epidemiological evidence that a prolonged period of breastfeeding has other health benefits for both the child and the mother. Future studies should control for parent’s picky eating when studying the association between introduction of solid food types and child picky eating. To better address reversed causation, future research should examine which initial feeding problems – that could have led to a shorter duration of breastfeeding – contribute to the development or are early signs of picky eating. In addition, the use of validated questionnaires to measure initial feeding problems (e.g. the infant version of the CEBQ) – instead of using a few items – is highly recommended. Another limitation is that the timing of introducing
vegetables and fruits was retrospectively assessed at 6 months, possibly leading to misclassification, although we assumed that mothers are well capable of recalling when they introduced these food items in a 6-months-period.

Conclusions

Our results suggest that breastfeeding duration does not predict picky eating in children. An earlier introduction to vegetables was related to less picky eating behavior. It is possible that the timing of complementary feeding is more relevant for picky eating than is breastfeeding duration. Children introduced to vegetables before the age of 5 months were particularly less likely to be picky eaters, which adds to the current discussion regarding guidelines for the timing of complementary feeding in developed countries (from 4 months on, as included in the Dutch recommendations, versus after 6 months, as recommended by the World Health Organization). Before providing policy makers, health practitioners, and parents with further feeding recommendations, however, future studies should replicate our findings, accounting for potential confounding bias (for instance due to parents’ own picky eating) and reverse causation, as well as investigating the potential impact of introducing vegetables between 4 and 5 months of age on other aspects of child development, such as children’s dietary quality, obesity risk, allergies and other health outcomes.
References


## Supplement

### SUPPLEMENTARY TABLE 3.1.1  Breastfeeding and picky eating, additionally adjusted for timing of introducing any solid food

<table>
<thead>
<tr>
<th>BREASTFEEDING DURATION IN CATEGORIES</th>
<th>FOOD FUSSINESS SUM-SCORE§</th>
<th>N</th>
<th>MODEL 1: B (95% CI)* **</th>
<th>MODEL 2: B (95% CI)* **</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never breastfeeding</td>
<td></td>
<td>283</td>
<td>-0.04 (-0.74; 0.66)</td>
<td>-0.05 (-0.77; 0.67)</td>
</tr>
<tr>
<td>Versus recommended (≥ 6months)</td>
<td></td>
<td>1,122</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>Breastfeeding duration continuously (per month)</td>
<td></td>
<td>3,391</td>
<td>-0.05 (-0.09; -0.00)</td>
<td>* -0.06 (-0.10; -0.01)</td>
</tr>
<tr>
<td>BREASTFEEDING DURATION IN CATEGORIES</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;0 – 1.9 months</td>
<td></td>
<td>791</td>
<td>0.66 (0.20; 1.12)</td>
<td>0.76 (0.29; 1.23)</td>
</tr>
<tr>
<td>2 – 3.9 months</td>
<td></td>
<td>738</td>
<td>0.16 (-0.30; 0.62)</td>
<td>0.28 (-0.20; 0.75)</td>
</tr>
<tr>
<td>4 – 5.9 months</td>
<td></td>
<td>457</td>
<td>0.13 (-0.40; 0.66)</td>
<td>0.23 (-0.31; 0.77)</td>
</tr>
<tr>
<td>≥ 6 months</td>
<td></td>
<td>1,122</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
</tr>
</tbody>
</table>

* p<0.05; ** p<0.01.
§ Food fussiness sum-score: range 6-30.
# Unstandardized regression coefficients (95% confidence interval).
∞ Model 1 is adjusted for the same confounders as in our main analyses. Model 2: model 1 + adjustment for timing of introducing any solids.
### SUPPLEMENTARY TABLE 3.1.2  Sensitivity analyses in Dutch children only: Infant feeding and picky eating

<table>
<thead>
<tr>
<th>FOOD FUSSINESS SUM-SCORE§</th>
<th>BREASTFEEDING IN ALL CHILDREN</th>
<th>N</th>
<th>MODEL 1: B (95% CI)*∞</th>
<th>MODEL 2: B (95% CI)*∞</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Never breastfeeding</strong></td>
<td>274</td>
<td>0.00 (-0.66; 0.66)</td>
<td>0.24 (-0.49; 0.98)</td>
<td></td>
</tr>
<tr>
<td>Versus recommended (≥ 6 months)</td>
<td>859</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
<td></td>
</tr>
<tr>
<td><strong>Breastfeeding duration continuously (per month)</strong></td>
<td>2584</td>
<td>-0.04 (-0.09; 0.01)</td>
<td>-0.06 (-0.11; -0.01) *</td>
<td></td>
</tr>
</tbody>
</table>

**IN BREASTFED CHILDREN ONLY**

<table>
<thead>
<tr>
<th>BREASTFEEDING DURATION IN CATEGORIES</th>
<th>N</th>
<th>MODEL 1: B (95% CI)*∞</th>
<th>MODEL 2: B (95% CI)*∞</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;0 – 1.9 months</td>
<td>583</td>
<td>-0.71 (0.19; 1.23)</td>
<td>** -0.79 (0.25; 1.32) **</td>
</tr>
<tr>
<td>2 – 3.9 months</td>
<td>525</td>
<td>0.14 (-0.39; 0.67)</td>
<td>0.16 (-0.38; 0.70)</td>
</tr>
<tr>
<td>4 – 5.9 months</td>
<td>344</td>
<td>0.32 (-0.29; 0.92)</td>
<td>0.32 (-0.29; 0.93)</td>
</tr>
<tr>
<td>≥ 6 months</td>
<td>859</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
</tr>
</tbody>
</table>

**TIMING OF COMPLEMENTARY FEEDING – ALL CHILDREN**

**Timing of introducing any solids**

<table>
<thead>
<tr>
<th>N</th>
<th>MODEL 1: B (95% CI)*∞</th>
<th>MODEL 2: B (95% CI)*∞</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 4 months</td>
<td>213</td>
<td>-0.69 (-1.50; 1.11)</td>
</tr>
<tr>
<td>4 – 4.9 months</td>
<td>1345</td>
<td>-0.34 (-0.88; 0.20)</td>
</tr>
<tr>
<td>5 – 5.9 months</td>
<td>571</td>
<td>-0.09 (-0.70; 0.53)</td>
</tr>
<tr>
<td>≥ 6 months (reference)</td>
<td>403</td>
<td>0 [reference]</td>
</tr>
</tbody>
</table>

**Timing of introducing fruit**

<table>
<thead>
<tr>
<th>N</th>
<th>MODEL 1: B (95% CI)*∞</th>
<th>MODEL 2: B (95% CI)*∞</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 4 months</td>
<td>131</td>
<td>-0.63 (-1.72; 0.47)</td>
</tr>
<tr>
<td>4 – 4.9 months</td>
<td>1260</td>
<td>-0.46 (-1.23; 0.31)</td>
</tr>
<tr>
<td>5 – 5.9 months</td>
<td>642</td>
<td>-0.15 (-0.96; 0.67)</td>
</tr>
<tr>
<td>≥ 6 months (reference)</td>
<td>172</td>
<td>0 [reference]</td>
</tr>
</tbody>
</table>

**Timing of introducing vegetables**

<table>
<thead>
<tr>
<th>N</th>
<th>MODEL 1: B (95% CI)*∞</th>
<th>MODEL 2: B (95% CI)*∞</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 4 months</td>
<td>62</td>
<td>-0.83 (-2.12; 0.45)</td>
</tr>
<tr>
<td>4 – 4.9 months</td>
<td>913</td>
<td>-0.76 (-1.31; -0.21)  **</td>
</tr>
<tr>
<td>5 – 5.9 months</td>
<td>812</td>
<td>-0.27 (-0.83; 0.29)</td>
</tr>
<tr>
<td>≥ 6 months (reference)</td>
<td>434</td>
<td>0 [reference]</td>
</tr>
</tbody>
</table>

* p<0.05, ** p<0.01.
§ Food fussiness sum-score: range 6-30.
# Unstandardized regression coefficients (95% confidence interval).
∞ Model 1 is unadjusted. Model 2 is adjusted for the same confounders as in our main analyses (except for maternal ethnic background).
### SUPPLEMENTARY TABLE 3.1.3

Timing of complementary feeding and picky eating, additionally adjusted for breastfeeding duration

<table>
<thead>
<tr>
<th>FOOD FUSSINESS SUM-SCORE§</th>
<th>TIMING OF INTRODUCING ANY SOLIDS</th>
<th>N</th>
<th>MODEL 1: B (95% CI)#</th>
<th>MODEL 2: B (95% CI)∞</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt; 4 months</td>
<td>334</td>
<td>-0.48 (-1.19; 0.24)</td>
<td>-0.62 (-1.35; 0.11)</td>
</tr>
<tr>
<td></td>
<td>4 – 4.9 months</td>
<td>1975</td>
<td>-0.21 (-0.74; 0.32)</td>
<td>-0.33 (-0.87; 0.21)</td>
</tr>
<tr>
<td></td>
<td>5 – 5.9 months</td>
<td>701</td>
<td>-0.32 (-0.92; 0.29)</td>
<td>-0.32 (-0.92; 0.29)</td>
</tr>
<tr>
<td></td>
<td>≥ 6 months (reference)</td>
<td>381</td>
<td>reference</td>
<td>reference</td>
</tr>
<tr>
<td>Timing of introducing fruit</td>
<td>&lt; 4 months</td>
<td>196</td>
<td>-0.55 (-1.49; 0.38)</td>
<td>-0.66 (-1.60; 0.28)</td>
</tr>
<tr>
<td></td>
<td>4 – 4.9 months</td>
<td>1864</td>
<td>-0.46 (-1.12; 0.21)</td>
<td>-0.54 (-1.21; 0.13)</td>
</tr>
<tr>
<td></td>
<td>5 – 5.9 months</td>
<td>795</td>
<td>-0.31 (-0.12; 0.21)</td>
<td>-0.31 (-1.01; 0.40)</td>
</tr>
<tr>
<td></td>
<td>≥ 6 months (reference)</td>
<td>227</td>
<td>reference</td>
<td>reference</td>
</tr>
<tr>
<td>Timing of introducing vegetables</td>
<td>Model 1: B (95% CI)*</td>
<td>Model 2: B (95% CI)*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 4 months</td>
<td>4 – 4.9 months</td>
<td>1373</td>
<td>-0.57 (-1.06; -0.09) *</td>
<td>-0.66 (-1.15; -0.18) **</td>
</tr>
<tr>
<td>5 – 5.9 months</td>
<td>≥ 6 months (reference)</td>
<td>1095</td>
<td>-0.33 (-0.83; 0.16)</td>
<td>-0.34 (-0.84; 0.15)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>543</td>
<td>reference</td>
<td>reference</td>
</tr>
</tbody>
</table>

*p<0.05, **p<0.01.

§ Food fussiness sum-score: range 6-30.

# Unstandardized regression coefficients (95% confidence interval).

∞ Model 1 is adjusted for the same confounders as in our main analyses. Model 2: model 1 + duration of breastfeeding.
CHAPTER 3.2

Parents’ anxiety and depressive symptoms & Child picky eating

Manuscript based on this chapter:
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 3 years). 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, causing major concerns among parents. Child picky eating has been associated with functional constipation, weight problems, and behavior problems. Previous research suggested parental controlling feeding, and parental physical and mental health problems as potential risk factors for picky eating (also called ‘fussy’ or ‘selective’ eating). However, the etiology of picky eating is not well understood.

It is well known that emotional problems of parents (i.e. anxiety and depression) are related to problematic child development, including disturbed eating behaviors. 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, 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. Maternal emotional problems during the child’s preschool period have been found to be predictive for persistent picky eating at a later age. Farrow and Blissett, however, have reported that antenatal and postnatal maternal psychiatric symptoms did not predict picky eating in six-month-old children.

Most studies focused on maternal symptoms during the child’s preschool period. This is a sensitive period in development, but the Barker hypothesis 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). 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. 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. 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.

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. 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. 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) 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 ≥ 4 were classified as ‘picky eaters’.22

**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 CEBQ’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.23 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.24 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 Y25 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

<table>
<thead>
<tr>
<th>FAMILY CHARACTERISTICS</th>
<th>N</th>
<th>PERCENTAGE OR MEAN (SD)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal age at enrollment mean years</td>
<td>4,746</td>
<td>31.6 (4.5)</td>
</tr>
<tr>
<td>Paternal age at enrollment mean years</td>
<td>4,746</td>
<td>34.0 (5.4)</td>
</tr>
<tr>
<td>Household income % low (&lt;€2000 / month)</td>
<td>1,262</td>
<td>26.6</td>
</tr>
<tr>
<td>Mothers’ ethnic background % Dutch</td>
<td>3,113</td>
<td>65.6</td>
</tr>
<tr>
<td>% Moroccan</td>
<td>139</td>
<td>2.9</td>
</tr>
<tr>
<td>% Surinamese &amp; Dutch Antillean</td>
<td>335</td>
<td>7.1</td>
</tr>
<tr>
<td>% Turkish</td>
<td>276</td>
<td>5.8</td>
</tr>
<tr>
<td>% other Western (mainly European)</td>
<td>414</td>
<td>8.7</td>
</tr>
<tr>
<td>% other non-Western</td>
<td>469</td>
<td>9.9</td>
</tr>
<tr>
<td>Fathers’ ethnic background % Dutch</td>
<td>3,148</td>
<td>66.3</td>
</tr>
<tr>
<td>% Moroccan</td>
<td>174</td>
<td>3.7</td>
</tr>
<tr>
<td>% Surinamese &amp; Dutch Antillean</td>
<td>354</td>
<td>7.5</td>
</tr>
<tr>
<td>% Turkish</td>
<td>258</td>
<td>5.4</td>
</tr>
<tr>
<td>% other Western (mainly European)</td>
<td>320</td>
<td>6.7</td>
</tr>
<tr>
<td>% other non-Western</td>
<td>492</td>
<td>10.4</td>
</tr>
<tr>
<td>Mothers’ educational level % low</td>
<td>678</td>
<td>14.3</td>
</tr>
<tr>
<td>% medium</td>
<td>1,324</td>
<td>27.9</td>
</tr>
<tr>
<td>% high</td>
<td>2,744</td>
<td>57.8</td>
</tr>
<tr>
<td>Fathers’ educational level % low</td>
<td>850</td>
<td>17.9</td>
</tr>
<tr>
<td>% medium</td>
<td>1,241</td>
<td>26.2</td>
</tr>
<tr>
<td>% high</td>
<td>2,655</td>
<td>55.9</td>
</tr>
<tr>
<td>Single parent % yes</td>
<td>373</td>
<td>7.9</td>
</tr>
<tr>
<td>Parity % multipara</td>
<td>1,964</td>
<td>41.4</td>
</tr>
<tr>
<td>Mode of delivery % cesarean section</td>
<td>650</td>
<td>13.7</td>
</tr>
<tr>
<td>Parents’ anxiety (BSI) mean scale scores</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mothers’ anxiety during pregnancy</td>
<td>4,746</td>
<td>0.23 (0.38)</td>
</tr>
<tr>
<td>Mothers’ anxiety at 3 years postnatal</td>
<td>4,746</td>
<td>0.18 (0.32)</td>
</tr>
<tr>
<td>Fathers’ anxiety during pregnancy</td>
<td>4,144</td>
<td>0.16 (0.29)</td>
</tr>
<tr>
<td>Fathers’ anxiety at 3 years postnatal</td>
<td>4,144</td>
<td>0.15 (0.25)</td>
</tr>
<tr>
<td>Parents’ depression (BSI) mean scale scores</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mothers’ depression during pregnancy</td>
<td>4,746</td>
<td>0.17 (0.38)</td>
</tr>
</tbody>
</table>
TABLE 3.2.1 (CONTINUED)  Sample characteristics of 4,746 parent-child dyads in the Generation R Study

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mothers’ depression at 3 years postnatal</td>
<td>4,746</td>
<td>0.14</td>
<td>(0.33)</td>
</tr>
<tr>
<td>Fathers’ depression during pregnancy</td>
<td>4,144</td>
<td>0.10</td>
<td>(0.26)</td>
</tr>
<tr>
<td>Fathers’ depression at 3 years postnatal</td>
<td>4,144</td>
<td>0.10</td>
<td>(0.27)</td>
</tr>
</tbody>
</table>

CHILD CHARACTERISTICS

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td>% boy</td>
<td>2,363</td>
</tr>
<tr>
<td>Birth weight</td>
<td></td>
<td>means grams</td>
<td>4,746</td>
</tr>
<tr>
<td>Gestational age</td>
<td></td>
<td>means weeks</td>
<td>4,746</td>
</tr>
<tr>
<td>Picky eating</td>
<td></td>
<td>Food fussiness at 4 years (CEBQ)</td>
<td>mean sum-score</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Picky eating proxy at 3 years (CBCL)</td>
<td>% picky eaters reported by mothers</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Picky eating proxy at 3 years (CBCL)</td>
<td>% picky eaters reported by fathers</td>
</tr>
</tbody>
</table>

§ 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 mean s (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.


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).
3.2 PARENTS’ ANXIETY AND DEPRESSIVE SYMPTOMS AND CHILD PICKY EATING

Parental anxiety and depressive symptoms and child picky eating

Table 3.2.2 shows that higher maternal anxiety 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 anxiety score, children had a 0.91 higher food fussiness sum-score, 95% CI: 0.49; 1.33). Likewise, the associations between fathers’ anxiety 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)

<table>
<thead>
<tr>
<th>PARENTAL DEPRESSIVE SYMPTOMS (BSI)</th>
<th>N</th>
<th>B</th>
<th>(95% CI)</th>
<th>P-VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal depression scale scores</td>
<td>4,746</td>
<td>0.91</td>
<td>(0.49; 1.33)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Depression during pregnancy</td>
<td></td>
<td>0.81</td>
<td>(0.35; 1.26)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Depression at 3 years postnatal</td>
<td></td>
<td>0.72</td>
<td>(0.07; 1.36)</td>
<td>0.03</td>
</tr>
<tr>
<td>Depression at 3 years postnatal</td>
<td></td>
<td>0.68</td>
<td>(0.08; 1.28)</td>
<td>0.03</td>
</tr>
<tr>
<td>Paternal depression scale scores</td>
<td>4.144</td>
<td>0.72</td>
<td>(0.07; 1.36)</td>
<td>0.03</td>
</tr>
<tr>
<td>Depression during pregnancy</td>
<td></td>
<td>0.68</td>
<td>(0.08; 1.28)</td>
<td>0.03</td>
</tr>
</tbody>
</table>

§ 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.
Abbreviations: 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

<table>
<thead>
<tr>
<th>MATERNAL EMOTIONAL PROBLEMS (BSI)</th>
<th>N</th>
<th>PICKY EATER§ REPORTED BY MOTHER</th>
<th>PICKY EATER# REPORTED BY FATHER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal anxiety scale scores</td>
<td>3,409</td>
<td>OR (95% CI)</td>
<td>P-VALUE</td>
</tr>
<tr>
<td>Anxiety during pregnancy</td>
<td></td>
<td>1.50 (1.18; 1.89)</td>
<td>0.001</td>
</tr>
<tr>
<td>Anxiety at 3 years postnatal</td>
<td></td>
<td>1.65 (1.27; 2.13)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Maternal depressive scale scores</td>
<td>3,409</td>
<td>OR (95% CI)</td>
<td>P-VALUE</td>
</tr>
<tr>
<td>Depression during pregnancy</td>
<td></td>
<td>1.24 (0.96; 1.61)</td>
<td>0.10</td>
</tr>
<tr>
<td>Depression at 3 years postnatal</td>
<td></td>
<td>1.38 (1.05; 1.79)</td>
<td>0.02</td>
</tr>
</tbody>
</table>

§ 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, although conflicting studies exist. 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 suggest that the direction of the associations with mothers’ antenatal symptoms is from mother to child. Coulthard and Harris 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. 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. Such feeding practices could have counterproductive effects by contributing to negative affective reactions to food, 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. 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 and, in turn, these problematic interactions could mediate the associations with children’s picky eating.

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. 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, 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.

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.
References

3.2 PARENTS’ ANXIETY AND DEPRESSIVE SYMPTOMS AND CHILD PICKY EATING

### Supplement

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

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

$\dagger$ 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)

<table>
<thead>
<tr>
<th>Maternal Depressive Symptoms (BSI)</th>
<th>N</th>
<th>B (95% CI)</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression during pregnancy</td>
<td>4,746</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average or below (score &lt;0.33)</td>
<td>3,833</td>
<td>0 [reference]</td>
<td></td>
</tr>
<tr>
<td>Above average (score ≥ 0.33)</td>
<td>604</td>
<td>0.87 (0.41; 1.33)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Clinical cut off (score ≥ 0.80)</td>
<td>309</td>
<td>0.87 (0.22; 1.51)</td>
<td>.01</td>
</tr>
<tr>
<td>Depression at 3 years postnatal</td>
<td>4,746</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average or below (score &lt;0.33)</td>
<td>3,949</td>
<td>0 [reference]</td>
<td></td>
</tr>
<tr>
<td>Above average (score ≥ 0.33)</td>
<td>582</td>
<td>0.48 (0.03; 0.93)</td>
<td>.04</td>
</tr>
<tr>
<td>Clinical cut off (score ≥ 0.80)</td>
<td>215</td>
<td>1.11 (0.38; 1.84)</td>
<td>.003</td>
</tr>
</tbody>
</table>

§ 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.
CHAPTER 3.3

Child picky eating & mental health problems

Manuscript based on this chapter:
Abstract

**Background:** Picky eaters in the general population form a heterogeneous group. It is important to differentiate between children with transient picky eating and persistent picky eating behavior when adverse outcomes are studied. We analyzed 4 picky eating trajectories to determine the associations with child mental health prospectively.

**Methods:** From a population-based cohort, 3,748 participants were assessed for picky eating at 1.5, 3 and 6 years of age using maternal reports. Four trajectories were defined; 1) persistent; picky eating at all ages, 2) remitting; picky eating before 6 years only, 3) late-onset; picky eating at 6 years only, and 4) never; no picky eating at any assessment. Child’s problem behaviors were assessed with the Teacher’s Report Form at 7 years of age. We examined associations between picky eating trajectories and emotional problems, behavior problems and pervasive developmental problems using logistic regressions. Analyses were adjusted for child, parental, and socioeconomic confounders. We also adjusted for maternal-reported baseline problem behavior at age 1.5 years; the never picky eating group was used as reference.

**Results:** Persisting picky eating predicted pervasive developmental problems at age 7 years (OR=2.00, 95% CI: 1.10; 3.63). The association remained when adjusted for baseline pervasive developmental problems at 1.5 years (OR=1.96, 95% CI: 1.10; 3.51). Persistent picky eating was not associated with behavior (OR=0.92, 95% CI: 0.53; 1.60) or emotional problems (OR=1.24, 95% CI: 0.74; 2.07). Other picky eating trajectories were not related to child behavior or emotional problems.

**Conclusions:** Persistent picky eating may be a symptom or sign of pervasive developmental problems, but is not predictive of other behavior problems. Remitting picky eating was not associated with adverse mental health outcomes, which further indicates that it may be part of normal development.
**Introduction**

Picky eating is a frequent eating problem in early childhood, characterized by food refusal, eating a limited variety of food, an unwillingness to try new food (food neophobia). The prevalence of picky eating peaks (14%-50%) in preschool children, and declines (7%-27%) in later childhood. Incidence also declines after preschool age. The high prevalence and incidence are an indication that picky eating in the preschool age is often part of a normal development. Indeed, many health professionals tend to regard picky eating as a normal phase which eventually passes. However, this is in contrast to how many parents experience picky eating, that is, as a major cause of concern. Parents often seek medical help for their child's picky eating, and express frustration with physicians for dismissing their concerns. Our previous report within the Generation R Study on picky eating trajectories, confirmed that the majority of children's picky eating problems in the preschool age remitted before the age of 6 years. However, we also found a small group of children with persisting picky eating problems who had a lower birth weight, and from a non-Dutch and low socioeconomic background, compared with non-picky eaters.

In previous studies, picky eating was associated with higher levels of behavior, emotional and pervasive developmental problems in childhood and was suggested to be a precursor of anorexia nervosa. The most recent studies concluded that picky eating in children of school age should be considered as a risk factor or marker for general psychopathology, rather than a precursor of eating disorders. However, picky eating problems are also specifically associated with pervasive developmental disorders. The prevalence of picky eating in children with autism was found to be as high as 90% and often present from early age onwards. In addition, feeding problems and eating disorders are associated with anxiety problems, and distorted child-parent interactions are suggested to play an important role in feeding problems.

However, most picky eating studies have some important limitations. First, most studies were limited by their cross-sectional design. Second, they did not differentiate between different trajectories, clustering remitting and persistent picky eaters. Third, a lack of correction for baseline differences impairs making temporal inferences. Also, most studies did not adjust or only poorly adjusted for possible confounders, while for instance socioeconomic characteristics, child birth weight, and emotional temperament, and maternal emotional problems have been related to children's picky eating. Lastly, the majority of studies in the field of eating problems rely on one informant to report both the determinant and outcome. However, this practice of using a single informant can lead to spurious associations, i.e. infor...
mation bias (shared method variance). Typically, mothers’ reports are used to assess picky eating as well as emotional and behavior problems, possibly introducing this type of bias.

It is important to study the course and outcome of picky eating in the general population to determine which children are at high risk for adverse mental health outcomes. Furthermore, this should be evaluated in the context of the child’s age. First, we hypothesized that remitting picky eating problems in the preschool age (0-4 years) are part of normal development and are not associated with an increased risk of any adverse mental health problems. Second, we hypothesized that children with persisting picky eating problems have a higher risk of adverse mental health outcomes. In particular, we expect that persistent picky eating is associated with more pervasive developmental problems and anxiety problems. Third, we will test whether late-onset picky eating is associated with emotional or behavior problems; however, there are insufficient studies to date to formulate a specific hypothesis for this association.

**Methods**

**Study design and participants**

This study was embedded within the Generation R Study, a prospective population-based cohort in Rotterdam, the Netherlands, aiming to identify determinants of growth, development and health from fetal life onwards. Pregnant women residing in Rotterdam with an expected delivery date between April 2002 and January 2006 were invited to participate. Written informed consent was obtained from all participants. The Medical Ethical Committee of the Erasmus Medical Center, Rotterdam, has approved this study. Information about child and family characteristics was obtained by postal questionnaires filled out by parents, and from medical records.

Picky eating was assessed by parental report questionnaires when children were 1.5, 3, and 6 years old. Children who were not assessed for picky eating at any of these time points, or with an inconsistent picky eating pattern, were excluded from the study. Mental health outcomes were determined using the Teacher Report Form (TRF) when the child was 7 years old (mean=6.7 years, standard deviation[SD]=1.3 years. A total of 3,506 children were included in the present study.
Measures

Trajectories of picky eating

Picky eating was assessed with two questions of the Child Behavioral Checklist (CBCL)\textsuperscript{23,24} at age 1.5, 3, and 6 years, as described in detail previously.\textsuperscript{11} Briefly, at each assessment wave, mothers indicated whether their child “did not eat well” and “refused to eat” on a 3-point Likert scale. Based on the sum score of these two items (sum range 2-6) children with a score of sometimes and/or often (score of ≥ 4) were identified as a picky eater. Picky eating defined by the CBCL was related to reduced caloric intake, lower variety of foods, higher food fussiness score on the Children’s Eating Behaviour Questionnaire (CEBQ),\textsuperscript{25} as well as higher scores on other food avoidant scales of the CEBQ, suggesting that this method can be used as proxy for picky eating.\textsuperscript{11}

Four main picky eating trajectory groups were created: 1) never picky eaters: those who were never identified as picky eaters, 2) remitting picky eaters: those who were picky eaters at 1.5 and/or 3 years, but not at 6 years of age, 3) late-onset picky eaters: those who were picky eaters at 6 years of age only, 4) persistent picky eaters: those who were picky eaters at all assessment waves (1.5, 3, and 6 years).\textsuperscript{11}

The remaining 242 children with an inconsistent pattern (i.e. children identified as picky eaters at 1.5 years and 6 years, but not at 3 years, and children who were picky eaters at 3 and 6 years, but not at 1.5 years) were excluded from further analyses for two reasons. First, the accurate categorization would depend strongly on future follow-up of picky eating status, with the possibility that these children would then be categorized into remitting, late-onset or persistent picky eaters. Second, analyses of this group revealed a different pattern compared to the other trajectories and did not differ from the never picky eaters (results not shown).

Children’s mental health problems

To determine children’s problem behavior the Dutch translation of the TRF was used.\textsuperscript{26} The TRF is the teacher version of the CBCL, comprising 120 problem items that can be scored on a 3-point Likert scale (i.e. not true, sometimes true, or often true). The TRF has the following six DSM-Oriented Scales: affective problems, anxiety problems, pervasive developmental problems, attention deficit/hyperactivity problems, oppositional defiant problems, and conduct problems. The DSM-Oriented Scale problems were defined using the established borderline clinical cut-offs (84\textsuperscript{th} percentile).\textsuperscript{24} Three main groups of problems were formed in line with Micali et al.:\textsuperscript{5} 1) ‘emotional problems’ consisting of the summed anxiety and affective problems, 2) ‘behavior problems’ consisting of attention/hyperactivity and oppositional defiant problems, and 3) pervasive developmental problems. Conduct problems were ex-
cluded from the behavior problem group, because, at this young age, the diagnosis of conduct disorder has a low prevalence.27

Baseline problem level was reported by the mother, using the CBCL/1.5-5 when the child was 1.5 years of age.23,28 The CBCL is a 99-item parent report questionnaire that assesses child emotional and behavior problems. The same 3 main problem groups as described above for the TRF were formed. However, in contrast to the TRF, the CBCL scale scores were used continuously with a higher score indicating more problems. The CBCL emotional problems scale contained one item that was also used to define picky eating. To avoid bias, this item was excluded from the emotional problems scale score.

For sensitivity analyses, we also included maternal reports of children’s mental health problems at age 6 years. Again, the CBCL/1.5-5 was assessed,23 and continuous scores on the emotional problems (excluding 1 picky eating item), behavior problems, and pervasive developmental problems groups were used. Good reliability and validity have been reported for the Dutch translations of the CBCL and TRF.25,28

**Covariates**
Based on previous studies,11,20,29,30 we defined several child, family, and socioeconomic characteristics as potential confounders. Information about child sex, birth weight, and gestational age at birth was obtained from midwife and hospital registries. Maternal ethnic background, educational level, household income, and birth order were assessed by postal questionnaire. Maternal psychiatric symptoms during pregnancy were assessed with the Brief Symptom Inventory (BSI), a 53-item, validated self-report questionnaire.31,32 It assesses a spectrum of psychiatric problems such as anxiety, depression, somatization, and hostility. The overall mean score (range: 0-4) of all subscales was used, as indicator of general psychopathology, with higher scores indicated more psychiatric problems.

**Statistical analyses**
To examine the relation between picky eating and behavior problems, separate logistic regressions were performed with the picky eating trajectories as exposure (never picky eating as reference group) and the emotional problems, behavior problems, and pervasive developmental problems as outcomes. First, unadjusted logistic regression analyses were conducted. Second, all analyses were adjusted for potential confounders. In a separate model, we additionally adjusted for baseline child behavior problems using the CBCL at age 1.5 years to address the temporal sequence of the relation.
Several sensitivity analyses were performed. First, to avoid findings based on choice of cut-off, we performed linear regression analyses with continuously modeled outcomes. Second, to enable comparison with other studies and highlight possible informant bias, we performed linear regression analyses with maternal reported emotional, behavior and pervasive developmental outcomes using the CBCL at 6 years of age. These additional analyses are contrasted with analyses using the TRF and are conducted in participants with data on both TRF and CBCL (n=2,942) to facilitate comparison.

Except for the dependent variables, missing values were estimated using multiple imputation techniques. As the CBCL data included some missing values (<30% per assessment wave), proportions of trajectories of picky eating were based on multiple imputation if one or more scores were obtained. The pervasive developmental problems group was the only dependent variable (outcome) with missing data (n=3493, missing n=13). The presented results are based on pooled estimates of 5 imputed datasets. Analyses were performed using STATA/SE 12.0.

Results

Population characteristics

General child and family characteristics of the study population are presented in Table 3.3.1. The majority of the mothers were of Dutch ethnicity (57.9%), and from a higher socioeconomic status (56.5% higher vocational or academic education). The majority of the children never had picky eating problems (54.9%; n=1,926). Approximately 5.9% (n=206) were persistent picky eaters, while 34.1% (n=1,197) were remitting picky eaters. These numbers are best estimates (variation <4% of sample) as they are based on imputed data.
### TABLE 3.3.1 Population characteristics of 3,506 child-parent dyads within Generation R study*

<table>
<thead>
<tr>
<th><strong>Child characteristics</strong></th>
<th><strong>MEAN(SD) / %</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Child sex</td>
<td>% boy 50.6</td>
</tr>
<tr>
<td>Birth weight</td>
<td>% normal weight 88.6</td>
</tr>
<tr>
<td></td>
<td>% underweight 4.3</td>
</tr>
<tr>
<td></td>
<td>% overweight 7.2</td>
</tr>
<tr>
<td>Gestational age at birth</td>
<td>% aterm 87.5</td>
</tr>
<tr>
<td></td>
<td>% preterm 4.9</td>
</tr>
<tr>
<td></td>
<td>% postterm 7.6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Parental characteristic</strong></th>
<th><strong>MEAN(SD) / %</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age mother at enrollment</td>
<td>% mean years (sd) 30.9 (5.0)</td>
</tr>
<tr>
<td>Age father at enrollment</td>
<td>% mean years (sd) 33.7 (5.7)</td>
</tr>
<tr>
<td>Maternal ethnic background</td>
<td>% Dutch 57.9</td>
</tr>
<tr>
<td></td>
<td>% Moroccan 5.5</td>
</tr>
<tr>
<td></td>
<td>% Turkish 8.5</td>
</tr>
<tr>
<td></td>
<td>% sur/ant/cape 12.3</td>
</tr>
<tr>
<td></td>
<td>% other Western 8.1</td>
</tr>
<tr>
<td></td>
<td>% other non-Western 7.7</td>
</tr>
<tr>
<td>Maternal educational level</td>
<td>% higher vocational or academic training 56.5</td>
</tr>
<tr>
<td></td>
<td>% high school / lower vocational training 38.8</td>
</tr>
<tr>
<td></td>
<td>% no education / primary school 4.7</td>
</tr>
<tr>
<td>Household income</td>
<td>% high or middle 59.6</td>
</tr>
<tr>
<td></td>
<td>% low 15.3</td>
</tr>
<tr>
<td></td>
<td>% very low 25.1</td>
</tr>
<tr>
<td>Birth order</td>
<td>% firstborn 55.2</td>
</tr>
<tr>
<td>Smoking during pregnancy</td>
<td>% no 75.7</td>
</tr>
<tr>
<td></td>
<td>% stopped at pregnancy 8.8</td>
</tr>
<tr>
<td></td>
<td>% yes 15.5</td>
</tr>
<tr>
<td>Picky eating trajectories</td>
<td>% never 54.9</td>
</tr>
<tr>
<td></td>
<td>% remitting 34.1</td>
</tr>
<tr>
<td></td>
<td>% late-onset 5.1</td>
</tr>
<tr>
<td></td>
<td>% persistent 5.9</td>
</tr>
</tbody>
</table>

* Values are percentages for categorical variables and means (standard deviations) for continuous variables. N=3,748 as this table represents imputed data for covariates. Abbr. Sur: Surinamese, Ant: Antillean, Cape: Cape Verdian.
Picky eating trajectories and behavior problems

No associations were found between remitting, late-onset, persistent picky eaters and never picky eaters, and teacher reported behavior problems (Table 3.3.2).

<table>
<thead>
<tr>
<th>Picky Eating Trajectories</th>
<th>N</th>
<th>Model 1 OR (95% CI)</th>
<th>Model 2 OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never</td>
<td>1,926</td>
<td>1 [reference]</td>
<td>1 [reference]</td>
</tr>
<tr>
<td>Remitting</td>
<td>1,197</td>
<td>1.07 (0.74 – 1.55)</td>
<td>1.03 (0.66 – 1.61)</td>
</tr>
<tr>
<td>Late-onset</td>
<td>177</td>
<td>1.42 (0.64 – 3.13)</td>
<td>1.05 (0.42 – 2.61)</td>
</tr>
<tr>
<td>Persistent</td>
<td>206</td>
<td>0.92 (0.53 – 1.60)</td>
<td>0.66 (0.38 – 1.15)</td>
</tr>
</tbody>
</table>

# Values are odds ratios (95% confidence intervals) and reflect the risk of elevated behavior problems (attention/hyperactivity and oppositional defiant problems) for remitting, late-onset, and persistent picky eaters compared to never picky eaters.

∞ Model 1 is unadjusted, model 2 is adjusted for child sex, birth weight, gestational age, birth order; and maternal ethnic background, education, and psychiatric symptoms.

§ N is based on imputed trajectory groups. The inconsistent group (n=242) was excluded.

Picky eating trajectories and emotional problems

In the unadjusted analyses, remitting (odds ratio [OR]=1.54, 95% confidence interval [CI]: 1.01, 2.36) and persistent picky eaters (OR=1.71, 95% CI: 1.01; 2.36) showed more emotional problems than never picky eaters (Table 3.3.3), although this attenuated and was no longer significant after adjustment for child, family, and sociodemographic characteristics (OR=1.47, 95% CI: 0.92; 2.33 for remitting picky eating; OR=1.24, 95% CI: 0.74; 2.07 for persistent picky eating). No difference in emotional problems was found between late-onset picky eaters and never picky eaters.
TABLE 3.3.3 The associations between picky eating trajectories and borderline emotional problems

<table>
<thead>
<tr>
<th>PICKY EATING TRAJECTORIES</th>
<th>MODEL 1 OR (95% CI)*</th>
<th>MODEL 2 OR (95% CI)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never</td>
<td>1 [reference]</td>
<td>1 [reference]</td>
</tr>
<tr>
<td>Remitting</td>
<td>1.54 (1.01 – 2.36)*</td>
<td>1.47 (0.92 – 2.33)</td>
</tr>
<tr>
<td>Late-onset</td>
<td>1.53 (0.67 – 3.51)</td>
<td>1.21 (0.56 – 2.64)</td>
</tr>
<tr>
<td>Persistent</td>
<td>1.71 (1.01 – 2.91)*</td>
<td>1.24 (0.74 – 2.07)</td>
</tr>
</tbody>
</table>

* p<0.05.

Values are odds ratios (95% confidence intervals) and reflect the risk of elevated behavior problems (anxiety and affective problems) for remitting, late-onset, and persistent picky eaters compared to never picky eaters.

∞ Model 1 is unadjusted, model 2 is adjusted for child sex, birth weight, gestational age, birth order, and maternal ethnic background, education, and psychiatric problems.

§ N is based on imputed trajectory groups. The inconsistent group (n=242) was excluded.

Picky eating trajectories and pervasive developmental problems

Persistent picky eating was associated with more pervasive developmental problems, unadjusted (OR = 2.41, 95% CI: 1.37; 4.22), and after adjusting for confounders (OR = 2.00, 95% CI: 1.10; 3.63, Table 3.3.4). After additionally adjusting for baseline pervasive developmental problems at 1.5 years, persistent picky eating remained associated with a higher risk of pervasive developmental problems (OR=1.96, 95% CI: 1.10; 3.51; data not shown in table). None of the other picky eating trajectories were associated with pervasive developmental problems.
### TABLE 3.3.4  The associations between picky eating trajectories and borderline pervasive developmental problems

<table>
<thead>
<tr>
<th>PICKY EATING TRAJECTORIES</th>
<th>MODEL 1 OR (95% CI)*#</th>
<th>MODEL 2 OR (95% CI)*#</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never</td>
<td>1 [reference]</td>
<td>1 [reference]</td>
</tr>
<tr>
<td>Remitting</td>
<td>1.02 (0.64 – 1.61)</td>
<td>0.97 (0.59 – 1.59)</td>
</tr>
<tr>
<td>Late-onset</td>
<td>0.70 (0.21 – 2.24)</td>
<td>0.57 (0.16 – 2.05)</td>
</tr>
<tr>
<td>Persistent</td>
<td>2.41 (1.37 – 4.22)**</td>
<td>2.00 (1.10 – 3.63)*</td>
</tr>
</tbody>
</table>

*  p < 0.05, **  p<0.01.
#  Values are odds ratios (95% confidence intervals) and reflect the risk of elevated pervasive developmental problems for remitting, late-onset, and persistent picky eaters compared to never picky eaters.
∞  Model 1 is unadjusted, model 2 is adjusted for child sex, birth weight, gestational age, birth order; and maternal ethnic background, education, and psychiatric symptoms.
§  N is based on imputed trajectory groups. The inconsistent group (n=242) was excluded.

### Sensitivity analyses

Similar results were obtained from linear regression analyses with continuously modeled outcomes (see Supplementary Table 3.3.1), demonstrating that our findings do not depend on choice of cut-off. Additional sensitivity analyses with maternal reported child problems indicated that mothers of all picky eaters (remitting, late-onset or persistent) reported more emotional problems in their children (Supplementary Table 3.3.2) Likewise, mothers of late-onset and persistent picky eaters also reported more behavior problems in their children (e.g. for persistent picky eating B=0.36, 95% CI: 0.16; 0.55 as reported by mothers, and B= -0.12, 95% CI: -0.31; 0.07 as reported by teachers).
Discussion

In this population-based study, we found that persistent picky eating was longitudinally associated with pervasive developmental problems at age 7 years as reported by teachers, even after adjustment of baseline pervasive developmental problems at 1.5 years. Remitting and late-onset picky eating were not associated with adverse mental health outcomes.

Remitting picky eating and mental health problems

In line with our first hypothesis, the present study demonstrates that remitting picky eating was not prospectively associated with adverse mental health outcomes. This suggests that remitting picky eating in preschool children can be seen as part of normal development in the general population; a behavior that might be considered as age-appropriate and will eventually remit without behavior or emotional consequences. This is further strengthened by the fact that our findings are based on a longitudinal design. However, in the present study we did not include somatic health measures and other adverse outcomes cannot be ruled out.

Persistent picky eating and pervasive developmental problems

We hypothesized that persistent picky eating would be prospectively associated with pervasive developmental problems. In line with clinical studies reporting more picky eating problems among children with autism spectrum disorders (ASD), our study suggests that persistent picky eating is also more common in children from the general population with elevated pervasive developmental problems. Importantly, when we corrected for baseline pervasive developmental problems, persistent picky eating remained related to pervasive developmental problems at age 7 years. Thus, the finding cannot be explained by developmental problems early in life. This assessment was based on maternal reports, as parents usually recognize signs of autism in an early stage. Potentially, picky eating can help to detect pervasive developmental problems even earlier, as picky eating in young children is easily noticed by parents. In the study of Emond et al, parents reported that difficulty in eating is often present in children with autism from infancy (6 months) onwards and persists throughout early childhood; our finding that persistent picky eating can be an early symptom or sign for pervasive developmental problems extends this observation and suggests that in the general population picky eating can precede other pervasive developmental problems symptoms.
However, the median age of the first ASD diagnosis remains older than age 4 years. Persistent picky eating trajectories in our study are based on assessments from 1.5 to 6 years of age, thus a majority of children with ASD would already be diagnosed before persistent picky eating can be defined. However, as the age at which ASD is diagnosed is inversely associated with the number of symptoms observed, persisting picky eating can be used to detect ASD only in a minority of children in those with less severe or clear symptoms. Future studies are needed to evaluate if a persistent picky eating trajectory can be delineated earlier, that is at age 4-5 years. Since parents often seek medical help for their child’s eating behavior, clinicians should pay attention to children who persist in having picky eating behavior, as these children are at higher risk of pervasive developmental disorders. However, autism spectrum disorders are usually diagnosed around 4 years of age, and thus some caution is warranted as the CBCL assesses pervasive developmental problems and is not a diagnostic instrument; however, several studies have demonstrated that the CBCL pervasive developmental problem scale can be used to screen for ASD, and has a particularly high specificity in the assessment of pervasive developmental disorders.

Persistent picky eating and emotional problems

We did not confirm our hypothesis that persistent picky eating was also prospectively associated with anxiety problems. Rather, persistent picky eating was not associated with problems other than pervasive developmental problems. This is in contrast with an earlier report of the ALSPAC study that found strong associations of picky eating with behavior and emotional problems. In our study, the existing association between persistent picky eating and emotional problems disappeared when confounders were controlled for. Also, the present study found lower odds ratios compared with Micali et al. in the UK. The differences between the two studies might be explained by the design of our study (cross-sectional vs. longitudinal and repeated measures design) and, most importantly by a different informant (a teacher report vs. a mother report) as a measure for outcome. An earlier study showed that, when maternal reports are used for both the determinant and the outcome measure, the associations were strongly inflated. Thus, when mothers report both picky eating and problem behavior of the child, any observed association of picky eating with behavior and emotional problems is prone to reporter bias. Furthermore, mothers who are over-concerned about their child’s wellbeing might rate their child’s behavior as problematic in general. Our results suggest that the associations of picky eating with emotional and behavior problems may be inflated when mothers’ reports of emotional and behavior problems are used. However, others
may argue that teachers underreport (which would probably reduce precision) or more often incorrectly report, which would reduce the estimated effect of the associations.

**Interpretations**

Some caution is required when interpreting these results. First, because the concept of picky eating has not been fully operationalized and the boundaries between picky eating, food neophobia and eating disorders are not yet well defined. Also, in the present study, we defined trajectories of picky eating to differentiate subgroups across time that might have distinct outcomes. Although we found no association between picky eating and emotional problems, picky eating persisting from early to late childhood might predict eating disorders in adolescence or might be a risk factor for other severe psychopathology. However, this was beyond the scope of the present study and more research is required on this topic.

Our results emphasize the importance of differentiating between trajectories of picky eaters, as picky eating comprises distinct groups with different symptom clusters ranging from mild symptoms to clinical disorders such as Avoidant/Restrictive Food Intake Disorder (ARFID). ARFID can be considered an extreme form of picky eating and is associated with more pervasive developmental disorders compared with other eating disorders in a clinical setting. Therefore, we cautiously speculate that persistent picky eaters are at a higher risk for the development of ARFID.

**Late onset picky eating and mental health problems**

Finally, we tested whether late-onset picky eating was associated with emotional or behavior problems. Although late-onset picky eating was not longitudinally associated with any adverse mental health outcome, Micali et al. found more emotional, behavior and pervasive problems, as described above. It is possible that our study was underpowered to detect minor differences, given the relatively small group of late-onset picky eating to find differences when comparing them to children without picky eating problems. In the present study, late-onset picky eaters tended to have more emotional and behavior problems, but only in the unadjusted models; after correcting for confounders the odds ratios were strongly attenuated. This implies that the observed effect of picky eating behavior in early childhood is partially explained by socioeconomic differences between groups.
**Strengths and limitations**

This study had several strengths including the large sample size, its population-based longitudinal design and inclusion of a large amount of confounders. Additional strengths are the use of the teacher report (as an independent measurement for child psychopathology) and correction for baseline problems.

Some limitations should also be discussed. First, the TRF reports on the DSM-Oriented Scales are not equivalent to a DSM diagnosis. Thus some caution is necessary interpreting these results, more so as the borderline clinical cut-off was used. Second, we had no CBCL measurements at 4 and 5 years of age in order to better determine picky eaters with a persistent pattern or late-onset. Also, parents might have adjusted eating regimes to compensate for their child’s pickiness, resulting in a misclassification of the remitting group. However, maternal reports for the assessment of picky eating have been validated. We used only two items (“my child refuses to eat” and “my child doesn’t eat well”) to assess picky eating status. However, previous analyses in the same cohort as this study have indicated that this method correlates well with other measures of picky eating, including a lower variety of food, lower caloric intake, more food fussiness, slowness in eating and lower enjoyment of food. This suggests that our definition is a valid approximation of the concept. Lastly, a small group of picky eaters was excluded from further analysis due to having an inconsistent picky eating pattern. Follow-up of this group is needed to determine whether children in this group should be classified as remitting or persistent picky eaters, and whether picky eating is associated with adverse mental health outcomes.

**Conclusions**

Persistent picky eating was found to be an early symptom for pervasive developmental problems, whereas remitting picky eating was not associated with adverse mental health outcomes. We cautiously propose to regard remitting picky eating as part of normal development and, in line with consensus-based professional health guidelines, suggest a watchful waiting approach to picky eating problems in preschool age. However, health professionals should be aware of the possible mental health implications of persisting picky eating and, if necessary, perform additional testing.
References

3.3 CHILD PICKY EATING AND MENTAL HEALTH PROBLEMS


Supplement

SUPPLEMENTARY TABLE 3.3.1 The associations between picky eating trajectories and continuously modeled child’s mental health problems

<table>
<thead>
<tr>
<th>PICKY EATING TRAJECTORIES</th>
<th>EMOTIONAL PROBLEMS</th>
<th>BEHAVIOR PROBLEMS</th>
<th>PERVERSIVE PROBLEMS&lt;sup&gt;Ω&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>TOTAL N=3,506&lt;sup&gt;§&lt;/sup&gt;</td>
<td>B (95% CI)*</td>
<td>B (95% CI)*&lt;sup&gt;#&lt;/sup&gt;</td>
</tr>
<tr>
<td>Never</td>
<td>1,926</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>Remitting</td>
<td>1,197</td>
<td>0.18 (-0.16; 0.52)</td>
<td>0.06 (-0.49; 0.61)</td>
</tr>
<tr>
<td>Late-onset</td>
<td>177</td>
<td>0.06 (-0.36; 0.48)</td>
<td>-0.24 (-1.41; 0.93)</td>
</tr>
<tr>
<td>Persistent</td>
<td>206</td>
<td>0.08 (-0.55; 0.72)</td>
<td>-0.70 (-1.88; 0.47)</td>
</tr>
</tbody>
</table>

* p < 0.05.

# Values are B-values (95% confidence intervals) and reflect difference in scores on emotional problems (anxiety and affective problems), behavior problems (attention/hyperactivity and oppositional defiant problems) and pervasive developmental problems between picky eaters (remitting / late onset / persistent) and never picky eaters.

∞ Adjusted for child sex, birth weight, gestational age, birth order; and maternal ethnic background, education, and psychiatric symptoms.

§ N is based on imputed trajectory groups.

Ω Pervasive problems: N= 3,493 due to missing values.
## SUPPLEMENTARY TABLE 3.3.2 Possible informant bias in the association between trajectories of picky eating and mental health problems: Using teacher vs. mother reported outcome

<table>
<thead>
<tr>
<th>PICKY EATING TRAJECTORIES</th>
<th>TOTAL N= 2,942§</th>
<th>TEACHER REPORT</th>
<th>MATERNAL REPORT</th>
<th>TEACHER REPORT</th>
<th>MATERNAL REPORT</th>
<th>TEACHER REPORT</th>
<th>MATERNAL REPORT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>B (95% CI)⁺⁺⁺⁺</td>
<td>B (95% CI)⁺⁺⁺⁺</td>
<td>B (95% CI)⁺⁺⁺⁺</td>
<td>B (95% CI)⁺⁺⁺⁺</td>
<td>B (95% CI)⁺⁺⁺⁺</td>
<td>B (95% CI)⁺⁺⁺⁺</td>
</tr>
<tr>
<td>Never</td>
<td>51.8</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>Remitting</td>
<td>32.4</td>
<td>0.07 (-0.06; 0.20)</td>
<td>0.11* (0.00; 0.22)</td>
<td>0.00 (-0.10; 0.10)</td>
<td>0.11 (-0.01; 0.22)</td>
<td>0.02 (-0.13; 0.16)</td>
<td>0.16 (-0.14; 0.45)</td>
</tr>
<tr>
<td>Late-onset</td>
<td>4.4</td>
<td>0.05 (-0.15; 0.24)</td>
<td>0.41 (0.18; 0.65) ***</td>
<td>-0.03 (-0.23; 0.18)</td>
<td>0.28 (0.08; 0.49) ***</td>
<td>-0.05 (-0.33; 0.23)</td>
<td>0.65 (-0.19; 1.11) ***</td>
</tr>
<tr>
<td>Persistent</td>
<td>5.3</td>
<td>0.01 (-0.28; 0.31)</td>
<td>0.56 (0.33; 0.79) ***</td>
<td>-0.12 (-0.31; 0.07)</td>
<td>0.36 (0.16; 0.55) ***</td>
<td>0.26 (0.05; 0.48) *</td>
<td>0.94 (0.54; 1.35) ***</td>
</tr>
</tbody>
</table>

* p < 0.05, ** p < 0.01, *** p < 0.001.

# Values are B-values (95% confidence intervals) and reflect difference in standard deviation scores on emotional problems (anxiety and affective problems), behavior problems (attention/hyperactivity and oppositional defiant problems) and pervasive developmental problems between picky eaters (remitting / late onset / persistent) and never picky eaters.

∞ Adjusted for child sex, birth weight, gestational age, birth order; and maternal ethnic background, education, and psychiatric symptoms.

§ N is based on imputed trajectory groups. An overlapping sample (n=2,942) with available teacher and mother reports was used to facilitate comparison. The mean age of assessment by teacher report was 6.8 years, the mean age of maternal reports was 6.0 years.

Ω Pervasive problems: N= 2,929 due to missings
CHAPTER 3.4

Child picky eating and body composition

Manuscript based on this chapter:
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, 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%. Picky eaters often reject new food items (food neophobia), but they are particularly characterized by their consistent rejection of specific familiar foods, especially vegetables. This consistent refusal of specific food items may result in a restricted dietary variety, which could have consequences for a child’s health, growth, and development. It is possible that picky eaters have an insufficient energy intake, 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, suggesting that they may be at risk of overweight. Both overweight and underweight in childhood have been related to several adverse health outcomes. Obese children are more likely to face emotional and social problems, and they are at risk of cardiovascular health problems across the life course. Although less pronounced, child underweight is also related to increased psychosocial problems, a poorer quality of life and physical health problems like a relatively low bone mass, which may put these children at greater risk of fractures.

Several studies have reported that picky eating in children is associated with a lower body mass index (BMI), lower body fat percentage, and underweight. However, other studies did not find any association between children’s picky eating and weight status, while Finistrella et al. reported that overweight/obese children were more likely to be picky, neophobic eaters than normal weight children. Except for a few studies, 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. 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. 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.

**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. 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.\textsuperscript{19,26} 

In this study, we used a picky eating profile, based on a previously performed latent profile analysis on 5 CEBQ subscales.\textsuperscript{27} 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).\textsuperscript{27} 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\textsuperscript{28} (CBCL) assessed at 1.5 years, 3 years, and 6 years of age, as previously described in detail.\textsuperscript{29} The previously created picky eating trajectories\textsuperscript{29} 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\textsuperscript{29} (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\textsuperscript{2}). Age- and sex-adjusted standard deviation (SD) scores for height and BMI were calculated using Dutch reference growth curves.\textsuperscript{30} Children were classified into underweight, normal weight, overweight, or obese, using international age- and sex-specific cut-offs.\textsuperscript{31,32} 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\textsuperscript{2}). Likewise, children’s fat-	extit{free} mass index (FFMI) was calculated (\textit{sum of bone and lean mass in kg} / \textit{height in m\textsuperscript{2}}). 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. 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 4th birthday (based on the Rome II criteria) 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/m²).

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 4th 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.

**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 (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. 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>
<thead>
<tr>
<th>POPULATION CHARACTERISTICS</th>
<th>N</th>
<th>PERCENTAGE, MEAN (SD), OR MEDIAN (IQR)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child sex</td>
<td></td>
<td>% boy</td>
</tr>
<tr>
<td>Birth weight</td>
<td></td>
<td>mean grams</td>
</tr>
<tr>
<td>Child ethnic background</td>
<td></td>
<td>% Dutch</td>
</tr>
<tr>
<td></td>
<td></td>
<td>% Moroccan</td>
</tr>
<tr>
<td></td>
<td></td>
<td>% Surinamese &amp; Dutch Antillean</td>
</tr>
<tr>
<td></td>
<td></td>
<td>% Turkish</td>
</tr>
<tr>
<td></td>
<td></td>
<td>% other, Western (mainly European)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>% other, non-Western</td>
</tr>
<tr>
<td>Breastfeeding duration</td>
<td></td>
<td>median months</td>
</tr>
<tr>
<td>Introduction of fruits and vegetables</td>
<td></td>
<td>% &lt;3 months</td>
</tr>
<tr>
<td></td>
<td></td>
<td>% 3-6 months</td>
</tr>
<tr>
<td></td>
<td></td>
<td>% &gt;6 months</td>
</tr>
<tr>
<td>Functional constipation at age 4 years</td>
<td></td>
<td>% yes§</td>
</tr>
<tr>
<td>Picky eating profile</td>
<td></td>
<td>% picky eater</td>
</tr>
<tr>
<td>Child BMI age 4 years</td>
<td></td>
<td>mean SDS</td>
</tr>
<tr>
<td>Child BMI age 6 years</td>
<td></td>
<td>mean SDS</td>
</tr>
<tr>
<td>Maternal age</td>
<td></td>
<td>mean years</td>
</tr>
<tr>
<td>Maternal educational level</td>
<td></td>
<td>% low£</td>
</tr>
<tr>
<td></td>
<td></td>
<td>% medium£</td>
</tr>
<tr>
<td></td>
<td></td>
<td>% high£</td>
</tr>
<tr>
<td>Household income per month</td>
<td></td>
<td>median in €</td>
</tr>
<tr>
<td>Maternal BMI</td>
<td></td>
<td>median body mass index</td>
</tr>
<tr>
<td>Maternal psychiatric symptoms</td>
<td></td>
<td>median score</td>
</tr>
</tbody>
</table>

* 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.
§ 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.
£ 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>
<thead>
<tr>
<th>TABLE 3.4.2</th>
<th>Child picky eater profile at 4 years of age and body composition at 4 years and 6 years of age</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BODY COMPOSITION</strong></td>
<td><strong>AT 4 YEARS</strong></td>
</tr>
<tr>
<td>PICKY EATING PROFILE</td>
<td>BODY MASS</td>
</tr>
<tr>
<td>AT 4 YEARS</td>
<td>N=4,191</td>
</tr>
<tr>
<td>Picky eater profile vs non-picky eater profile</td>
<td>-0.38 (-0.50; -0.25)***</td>
</tr>
<tr>
<td>Model 1: adjusted for potential confounders¶</td>
<td></td>
</tr>
<tr>
<td>Model 2: additionally adjusted for BMI at 4¶¶</td>
<td></td>
</tr>
<tr>
<td>Picky eater profile vs non-picky eater profile</td>
<td>-0.11 (-0.19; -0.04)**</td>
</tr>
</tbody>
</table>

# 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

![Diagram showing adjusted OR (95% CI) for underweight, normal weight (reference), overweight, and obese at 6 years, adjusted for potential confounders and additionally adjusted for baseline BMI at 4 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 and lower weight status among picky eaters. However, several other studies did not find any association with weight status and some even reported that picky eating was related to overweight. 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. 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 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.

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, 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 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., 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. 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, 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. 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. 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 given the restricted diet variety that picky eaters have.

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 which is a less reliable method than the DXA measurements that we used. 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, 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,29,39 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).49,50 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.49 Moreover, it is advised to encourage parents to cook 51 and eat 49 together with their children, so that parents act as a role model and children can imitate their parents’ eating behaviors.49 Although these strategies may improve food intake 51 and food enjoyment 50 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.
References


3.4 CHILD PICKY EATING AND BODY COMPOSITION

## Supplement

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

<table>
<thead>
<tr>
<th>Picky Eating Trajectories</th>
<th>Body Composition at 6 Years</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Body Mass Index–SDS</td>
</tr>
<tr>
<td></td>
<td>N=3,094</td>
</tr>
<tr>
<td>Never picky eating</td>
<td>0 [reference]</td>
</tr>
<tr>
<td>Remitting picky eating (onset at 1.5 or 3 years, but not persisting)</td>
<td>-0.03 (-0.08; 0.01)</td>
</tr>
<tr>
<td>Late onset picky eating (at 6 years)</td>
<td>-0.04 (-0.15; 0.07)</td>
</tr>
<tr>
<td>Persistent picky eating (from 1.5 years till 6 years)</td>
<td>-0.39 (-0.51; -0.26)**</td>
</tr>
</tbody>
</table>

** p<0.001.

# Values are regression coefficients (95% confidence intervals). All body composition outcomes are age- and sex-adjusted standard deviation scores.

¶ 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.

¶¶ Additionally adjusted for baseline BMI. For remitting picky eating, we adjusted for baseline BMI at 1.3 years 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 1.3 years.

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

<table>
<thead>
<tr>
<th>Picky Eating Profile at 4 Years</th>
<th>Underweight N=212 (5.1%)</th>
<th>Normal Weight N=3,401 (81.3%)</th>
<th>Overweight N=454 (10.9%)</th>
<th>Obese N=116 (2.8%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 0: unadjusted</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Picky eater profile vs non-picky eater profile</td>
<td>2.98 (1.98; 4.48)**</td>
<td>1 [reference]</td>
<td>0.76 (0.47; 1.23)</td>
<td>0.62 (0.23; 1.70)</td>
</tr>
<tr>
<td>Model 1: adjusted for potential confounders¶</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Picky eater profile vs non-picky eater profile</td>
<td>3.16 (2.05; 4.88)**</td>
<td>1 [reference]</td>
<td>0.62 (0.37; 1.03)</td>
<td>0.43 (0.15; 1.23)</td>
</tr>
<tr>
<td>Model 2: additionally adjusted for BMI at age 4¶¶</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Picky eater profile vs non-picky eater profile</td>
<td>2.28 (1.34; 3.87)**</td>
<td>1 [reference]</td>
<td>0.82 (0.44; 1.55)</td>
<td>0.73 (0.18; 2.96)</td>
</tr>
</tbody>
</table>

** p<0.01, *** p<0.001.

# Values are odds ratios (95% confidence intervals).

¶ 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.
CHAPTER 4

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. 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. 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, 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. 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 (≥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,1,11 potentially inducing their mothers to stop breastfeeding early on. Together, these results suggest 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 – 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 time12,13 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,14,15 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.16 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 study17 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,18-20 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. 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 CEBQ, 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.23 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.24 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.25 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.26 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 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 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. 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. 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.
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),\textsuperscript{15,30} – 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\textsuperscript{31} – 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-analyses has shown that early intensive behavior intervention based on applied behavior analyses may improve cognitive functioning and adaptive behaviors.\textsuperscript{32}

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)\textsuperscript{33-35} 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.\textsuperscript{35} There are no guidelines for overcoming picky eating yet, but studies suggests that parents should cook and eat healthy foods together with their children,\textsuperscript{33,36,37} so that parents act as a role model and children can imitate their parents’ eating behaviors.\textsuperscript{33,37,38} Moreover, repeated exposure to a diversity of food items without coercion of eating is key for food acceptance.\textsuperscript{33} 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.\(^{39}\) 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).\(^{40}\) 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.\(^{41}\) Recall bias occurs when the accuracy of recalling earlier exposures is different for cases and controls.\(^{21}\) 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,\(^{20}\) 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.\(^{42}\) 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. 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 and could also precede eating psychopathology later in life.

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, 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. 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. Emotional overeating may also be enhanced by mothers’ feeding practices when mothers with a history of eating disorders use food to soothe. Indeed, emotional eating occurs more often when mothers use emotional feeding practices. 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.\textsuperscript{52} This could explain why we did not find any differences and why monitoring did not predict child weight either in our and other cohorts.\textsuperscript{33,37,53} It is also possible that our assessment of monitoring\textsuperscript{54} 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,\textsuperscript{43,55-58} but contradicts the results from the ALSPAC Study showing a higher initiation and duration of breastfeeding in mothers with a history of eating disorders.\textsuperscript{59} 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,\textsuperscript{43} while mothers who are more focused on losing weight and who are aware of the belief that breastfeeding promotes weight loss may continue breastfeeding.\textsuperscript{60} Similarly, due to fears of gaining weight, mothers with a history of eating disorders may be concerned about their children’s weight as well\textsuperscript{60,61} 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.\textsuperscript{52} However, in smaller samples, mothers’ eating disorder psychopathology was related to more pressuring feeding.\textsuperscript{63,64} 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.\textsuperscript{65} If a higher diet quality infancy tracks throughout the life course,\textsuperscript{66} this could be ben-
eficial for cardiometabolic health. 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. 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.

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

**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 weigh. 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 1). Previous research has indicated the validity of self-report questions for assessing eating disorders.\(^6\)\(^8\) 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.\(^6\)\(^9\) Substantial overlap between self-report eating disorders and CIDI-diagnoses\(^6\)\(^9\) 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.\(^7\)\(^0\) 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 and increase the risk of fertility and pregnancy complications.

It is also possible that we found only weak associations because it has been a long time age since the mothers in our study suffered from an eating disorder, given that the incidence of eating disorders peaks in adolescence and almost no women suffered from an eating disorder in the year before pregnancy. 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. 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, 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. 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. 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. 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.

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, 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.43,59 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.80 Although home-made food is generally regarded as healthier in adults,81,82 commercial ready-to-eat-food does not necessarily imply a lower diet quality in infancy.83 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.81,82 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.84 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.85
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, 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.
References


CHAPTER 5

Summary
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 (≥ 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 kinderenigeren 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 proefschriftonderzoek (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 calorieëns kwijt te raken door laxeermiddelen te gebruiken, braken op te wekken, of overmatig te sporten. Op basis van deze toelichtingen hebben de vrouwen, die deelden 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). Tenslotte 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 snoepte 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 heb- ben gehad al op jonge leeftijd problemen ondervinden in hun emotionele ontwik- keling 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 eetge- drag. 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 on- gunstige 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 we beschreven staat in hoofdstuk 3.3, hebben we er dan ook voor gekozen om leerkrachten 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

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<th>Abbr.</th>
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<td>Avon Longitudinal Study of Parents and Children</td>
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<td>ANOVA</td>
<td>analysis of covariance</td>
</tr>
<tr>
<td>AN</td>
<td>anorexia nervosa</td>
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<tr>
<td>ANT</td>
<td>Antillean</td>
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<tr>
<td>ARFID</td>
<td>Avoidant/Restrictive Food Intake Disorder</td>
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<td>ASD</td>
<td>autism spectrum disorders</td>
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<td>binge eating disorder</td>
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<td>BMI</td>
<td>body mass index</td>
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<td>BSI</td>
<td>Brief Symptom Inventory</td>
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<td>Cape Verdian</td>
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<td>Child Behavior Checklist</td>
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<td>CEBQ</td>
<td>Children’s Eating Behaviour Questionnaire</td>
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<td>CFQ</td>
<td>Child Feeding Questionaire</td>
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<td>CI</td>
<td>confidence interval</td>
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<tr>
<td>CIDI</td>
<td>Composite International Diagnostic Interview</td>
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<tr>
<td>DSM</td>
<td>Diagnostic and Statistical Manual</td>
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<tr>
<td>DXA</td>
<td>Dual-energy-X-ray absorptiometry</td>
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<td>Eating Disorder Inventory</td>
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<td>FAD</td>
<td>Family Assessment Device</td>
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<tr>
<td>FFQ</td>
<td>food frequency questionnaire</td>
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<td>FFMI</td>
<td>fat-free mass index</td>
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<td>Infant Behavior Questionnaire-Revised</td>
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<td>interquartile range</td>
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<td>kg</td>
<td>kilogram</td>
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<tr>
<td>m</td>
<td>meter</td>
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<td>MC</td>
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<td>NOSIK</td>
<td>Nijmeegse Ouderlijke Stress Index Kort</td>
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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
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

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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

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Dankwoord
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 onderzoekscentrum, 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 gespreken 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 verheugd 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 voor al 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 wat 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 relativeren. 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 kruijen 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 uitgepakt. Je bent ook heel vaak mijn vraagbaak geweest voor allerlei kleine dingen, zeker in de afronding van mijn promotie, dankzij jouw enthousiasme voor het ‘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

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

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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.
Epidemiological studies of eating problems in the family

Lisanne M. de Barse

“I Don’t Want to Eat that”