Predictors of Eating Behavior in Middle Childhood: A Hybrid Fixed Effects Model

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Abstract

Children's eating behavior influences energy intake and thus weight through choices of type and amount of food. One type of eating behavior, food responsiveness, defined as eating in response to external cues such as the sight and smell of food, is particularly related to increased caloric intake and weight. Because little is known about the potential determinants of such behavior, we focus herein on child- and parent predictors of food responsiveness in a large community sample of Norwegian 6-year olds, followed up at ages 8 and 10. To measure children's food responsiveness, parents completed the Children's Eating Behaviour Questionnaire. Potential predictors were children's inhibition and symptoms of ADHD and depression, and parents' instrumental and controlling feeding practices as well as parental restrained eating. After accounting for children's initial levels of food responsiveness within a hybrid fixed effects method that takes into consideration all unmeasured time-invariant confounders, more child ADHD symptoms and greater restrained eating by parents predicted more food responsiveness at both ages 8 and 10. These results may provide important insights for efforts to prevent overeating.

Keywords: eating behavior, food responsiveness, obesity, feeding practices, ADHD, restrained eating

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Overweight and obesity, associated with numerous health risks in children (Han, Lawlor, & Kimm, 2010; Pulgaron, 2013), can be at least partially accounted for by children's eating behavior (Carnell & Wardle, 2008a, 2008b). Thus, identifying and targeting the determinants of overeating at an early age might be a promising avenue to prevent obesity (Carnell & Wardle, 2008b) as well as other eating-related problems (Gahagan, 2012). Although many aspects of eating behavior have been studied (French, Epstein, Jeffery, Blundell, & Wardle, 2012), one is particularly important with respect to children's weight: *Food responsiveness* (i.e. eating more in response to external cues such as sight and smell of food) (Webber, Hill, Saxton, Van Jaarsveld, & Wardle, 2009). Greater food responsiveness predicts greater weight gain in both infants (van Jaarsveld, Boniface, Llewellyn, & Wardle, 2014; van Jaarsveld, Llewellyn, Johnson, & Wardle, 2011) and school-aged children (Steinsbekk & Wichstrøm, 2015). Although research has revealed a great deal about what influences eating behavior (Gahagan, 2012), much still remains unclear about its etiology. To illustrate, it is often assumed that parents influence children's eating behavior – yet most of the existing literature is cross-sectional (Llewellyn, Carnell, & Wardle, 2011). Longitudinal studies provide one means of investigating potential determinants of eating behavior. The aim of the current study was therefore to examine child- and parent predictors of food responsiveness from six to ten years of age in a large community sample of Norwegian children.

An Intrapersonal and Interpersonal Model of Eating Behavior

The development of eating behavior is a complex process involving an interplay between biological tendencies and environmental influences (Ventura & Worobey, 2013), therefore ecological models have been embraced in an effort to delineate numerous and interrelated influences on the development of eating behvior (Story, Neumark-Sztainer, &

French, 2002) and subsequent overweight and obesity (Davison & Birch, 2001; Harrison et al., 2011; Harrist et al., 2012). Although the societal- and community contexts are important to consider when thinking about etiological forces (Story et al., 2002), ecological frameworks typically assume that such distal factors exert their effects though more proximal processes, especially intrapersonal and interpersonal ones (Harrist et al., 2012), that could serve as targets for intervention. It is for this reason that we focus on these proximate factors in the current inquiry.

Ecological models highlighting the interplay of parent- and child characteristics in influencing child development (e.g. Belsky, 1984), including eating behavior (Story et al., 2002), tend to be rather encompassing in order to incorporate the likely multifactorial processes taking place, and simultaneously avoid mis-specifying relations due to omitted variables. To illustrate, children's eating behavior is fairly heritable (Carnell, Haworth, Plomin, & Wardle, 2008; Llewellyn, van Jaarsveld, Johnson, Carnell, & Wardle, 2010), as is parental eating behavior (Rankinen & Bouchard, 2006; Schur, Noonan, Polivy, Goldberg, & Buchwald, 2009). This means that it is entirely possible that observed associations linking children's and parents' eating are genetically mediated. Moreover, alleged child determinants of eating, such as cognitive functioning and symptoms of depression (Harrist et al., 2012), could also exert their presumed effects via underlying genetic influences (Friedman et al., 2008; Rice, Harold, & Thapar, 2002). These observations highlight the need to discount potential genetic effects in studies of the determinants of eating behavior to reduce the risk of them leading to spurious causal conclusions.

Prospective studies of (distal or proximal) determinants of children's eating behavior typically rely on a covariate approach when adjusting for confounders, including controls for preceding (baseline) measurements of the outcome to be explained. Such efforts, however, cannot rule out all potential confounders (Foster, 2010). Fortunately, fixed effects models

have the advantage of succeeding where covariate designs cannot – in adjusting for all unmeasured time-invariant confounders (Allison, 2009; Firebaugh, Warner, & Massoglia, 2013). The implementation of this approach in the work reported herein enables us to illuminate child- and parent predictors of eating behavior net of any influence of factors that are more or less time-invariant – including genetics, family socioeconomic status (SES), regular food availability, stable neighborhood and community characteristics, and parental reporting bias – to name some obvious confounders. Below, we detail the theoretical and empirical reasons for focusing on the intrapersonal and interpersonal factors that we do as potential determinants of children's eating behavior.

The Intrapersonal Level: Child Characteristics

Prior work leads us to focus on three child characteristics; inhibition, ADHD symptoms and depression symptoms. Inhibition is the ability to inhibit a behavior, or stop one's behavior at the appropriate time (Roth, Isquith, & Gioia, 2014), and plays an important role in self-regulation (Nigg, 2017). Children with limited inhibition would be expected to be more responsive to food because they are likely to have greater problems with resisting the temptations of palatable food cues in an obesogenic environment (Stice & Yokum, 2016). To illustrate, impaired inhibitory control may lead to overeating by contributing to sensitization (i.e. repeated exposure to food stimuli resulting in amplification of responsiveness to food) and thus elevated responsivity of brain circuits involved in reward processes (Stice & Yokum, 2016), which in turn prompts craving and overeating when food cues are encountered (Berridge, Ho, Richard, & DiFeliceantonio, 2010). Some support for these claims comes from cross-sectional evidence showing that children with higher inhibitory control are better at self-regulating their food intake (Pieper & Laugero, 2013; Tan & Holub, 2011) and have lower intake of sugar and carbohydrates (Levitan et al., 2015). More notably, prospective research

indicates that lower cool executive functioning (which also includes inhibition) predicts increased food responsiveness in school-aged children (Groppe & Elsner, 2015).

In view of the fact that attention deficit/hyperactivity disorder (ADHD) involves problems with inhibition (Shallice et al., 2002), it is unsurprising that symptoms of ADHD also are associated with loss of control over eating (Reinblatt et al., 2015) and predict binge eating (Sonneville et al., 2015) in school-aged children. In fact, preschoolers with ADHD symptoms were found to have higher levels of food responsiveness in a population-based study (Leventakou et al., 2016) – even after controlling for different aspects of cognitive functioning. In the current investigation, we therefore evaluate whether ADHD symptoms predict food responsiveness in middle childhood, net of co-varying problems with inhibition.

Anhedonia and lack of approach-related behavior seen in depression may also influence children's eating. Indeed, disturbances in appetite and weight are among the symptoms of depression (American Psychiatric Association, 2013), thus providing a basis for expecting depression to affect food responsiveness. Empirical evidence consistent with this hypothesis is scarce. To our knowledge only one study has prospectively investigated potential effects of depression on children's eating behavior, finding that depressive symptoms predict later binge eating (Pearson, Zapolski, & Smith, 2015). Consistent with such a result is additional work showing that depression in adolescents is positively correlated with food responsiveness (Hou et al., 2013; Tanofsky-Kraff et al., 2008). Given the developmental differences between children and adolescents, and the substantially greater rates of depression among teenagers (Avenevoli, Knight, Kessler, & Merikangas, 2008), it remains to be determined whether similar links between depression and food responsiveness are evident in middle childhood. In view of the fact that loss of appetite appears to be a more significant marker than increased appetite when assessing depression in children (D. A. Cole et al., 2012), grounds exist for depression effects to operate differently in younger children. To be

considered, as well, is that gut activity decreases in the presence of emotional arousal, which suppresses hunger (Heatherton, Herman, & Polivy, 1991; van Strien & Ouwens, 2007), further raising the possibility that a natural response to emotional distress in childhood could be to eat less. Clearly, it remains unclear whether greater depressive symptomatology in the elementary-school years should forecast increased or decreased food responsiveness. To the best of our knowledge, the current study is the first to address this issue.

The Interpersonal Level: Parent Characteristics

Children use social information to guide their eating (Shutts, Kinzler, & DeJesus, 2013) and parents are a particularly important source of social cues (Savage, Fisher, & Birch, 2007; Vaughn et al., 2016). Although more general parenting and family factors (e.g. parental structuring, sensitivity, conflict) could prove important (Harrist et al., 2012), we focus on parent characteristics specifically related to eating, namely parents' feeding practices and their own eating behavior.

Feeding practices are specific, goal-directed behaviors or strategies parents use to influence a child's eating (Ventura & Birch, 2008; Wardle & Carnell, 2007). We focus on two feeding practices previously found to adversely affect children's eating behavior (Stang & Loth, 2011); control over eating (i.e. restriction of food, for example when children are prohibited from eating certain foods) and instrumental feeding, involving the use of food as a reward (Wardle, Sanderson, Guthrie, Rapoport, & Plomin, 2002). Associative learning may be an important mechanism accounting for the influence of these feeding practices, as they both place contingencies on eating or not eating a particular food item (Dovey, 2010). In consequence, they both can teach children to rely on external cues to regulate satiety and hunger (e.g. a clean plate) (Stang & Loth, 2011).

Cross-sectional studies provide support for these claims, as both higher levels of parental control over eating (Carnell, Benson, Driggin, & Kolbe, 2014; Webber, Cooke, Hill,

& Wardle, 2010) and instrumental feeding (Ainuki & Akamatsu, 2011; Carnell et al., 2014) have been found to be associated with enhanced food responsiveness. Notably, however, longitudinal evidence confirming such findings is scarce and by no means compelling (Gregory, Paxton, & Brozovic, 2010; Rodgers, Paxton, Massey, et al., 2013; Steinsbekk, Belsky, & Wichstrøm, 2016). Rodgers and colleagues (2013) found that parental control over eating forecast the tendency to overeat in toddlers, an eating behavior similar to food responsiveness (Rodgers, Paxton, Massey, et al., 2013), but this prediction proved insignificant once adjustments were made for potentially confounding factors, a finding that accords with those of another study of preschoolers (Gregory et al., 2010). Steinsbekk, Belsky and Wichstrøm (2016) also failed to detect a relation between control over eating and food responsiveness in 6-year olds, observing instead that instrumental feeding predicted increased food responsiveness two years later. Although heterogeneous, these findings concur with those from other prospective studies examining parenting effects on children's eating and weight (Sokol, Qin, & Poti, 2017), ones in which omission of potentially important confounders (e.g. parents' weight status, family structure) could explain some of the observed hetereogenity in results (Sokol et al., 2017).

Parents may also affect children's eating through intentionally and/or unintentionally modeling certain eating behavior (Palfreyman, Haycraft, & Meyer, 2013). Research investigating parental modeling of disturbed eating behavior – such as dietary restraint and dieting (Wertheim, Martin, Prior, Sanson, & Smart, 2002) – has found it to predict eating disturbances in children (Stice, 2002). In fact, higher levels of maternal dietary restraint precedes the emergence of overeating in children during the first five years of life (Stice, Agras, & Hammer, 1999) and greater increases in preschoolers' weight (Hood et al., 2000; Rodgers, Paxton, McLean, et al., 2013).

Associations such as these do not necessarily provide evidence that parental modeling is the influential mechanism, as others could account for the findings under consideration, most notably, a shared genetic tendency to overeat and gain weight (Carnell, Kim, & Pryor, 2012). More direct evidence of the influence of parental eating comes from experimental studies, as these demonstrate that modeling does indeed affect children's eating (for a review, see Cruwys, Beyelander, & Hermans, 2015). However, such brief manipulations are typically designed to document only short-term effects on eating and may not necessarily reflect processes operative in the home environment on an everyday basis. In addition, it remains an open question whether common genes or other confounding variables in fact explain the associations seen in observational studies.

The Current Study

In seeking to extend the work reviewed linking child- and parent characteristics with child eating behavior, we test an intra- and interpersonal model of children's eating behavior that includes children's inhibition, symptoms of ADHD, and depression, in addition to parents' feeding practices and dietary restraint as potential determinants. Toward these ends, we examine a large, representative sample of Norwegian children followed from 6 to 10 years of age, while implementing a statistical method that controls for all unmeasured time-invariant factors, including genetics. In addition, children and parents' weight status at each time of measurement and food responsiveness at the earliest age of measurement will be accounted for. Consistent with past research, we hypothesize that child inhibition problems, ADHD symptoms and depression symptoms as well as parents' instrumental feeding, control over eating and restrained eating will predict greater food responsiveness in children from 6 to 10 years of age.

Methods

Participants and Procedure

We utilize data from the "Trondheim Early Secure Study" (TESS). All children born in 2003 and 2004 and their parents living in Trondheim, Norway, were invited to participate (N=3,456) by an invitation letter including the Strengths and Difficulties Questionnaire (SDQ) version 4-16 (Goodman, Ford, Simmons, Gatward, & Meltzer, 2000) sent to their homes. SDQ was used because the main purpose of TESS is to examine children's mental health. The parents completed the SDQ and brought it with them when attending the health-checkup for their 4-year olds at the local well-child clinic. Because almost all children of the two birth cohorts appeared at the health-checkup (97,2%, n=3,358), the sample is in effect a representative community sample. A health nurse informed the parents about the study and asked them to participate (n=3,016); 2,475 gave informed consent. The health nurse missed asking 166 of the parents and 176 were not eligible due to not being proficient in Norwegian.

Children with higher scores on SDQ were oversampled by dividing SDQ total difficulty scores into four strata (cut-offs: 0-4, 5-8, 9-11, and 12-40), and the probability for selection increased with increasing SDQ scores (0.37, 0.48, 0.70, and 0.89 in the four strata, respectively), thereby increasing sample variability and thus statistical power. From those consenting, 1,250 families were drawn to participate according to the procedures described above. The Regional Committee for Medical and Health Research Ethics, Mid-Norway, approved the study (approval number: 2009/994; title of the study: The Trondheim Early Secure Study). Approximately two weeks after the health check-up, the child and one of the parents visited the university clinic for testing and observation. Retesting took place two, four and six years later when the children were 6, 8 and 10 years old, respectively. Because children's eating behavior was assessed from age 6 and onwards, the current study is based on data collected at age 6 (n=797, M_{age} = 6.72 years, SD =.17), age 8 (n=699, M_{age} = 8.80 years, SD = .24), and age 10 (n=702, M_{age} = 10.51 years, SD = .17). At baseline (age 6), 50.2% of the children were female, the majority of parents were ethnic Norwegians (93% of both

mothers and fathers) and the parent informant was typically the mother (81.1%). The sample was representative of the Norwegian population in terms of parents' level of education (Statistics Norway, 2012) and children's BMI (Júlíusson et al., 2013).

Attrition at age 8 was higher among children with lower inhibition (OR=1.05, (95%CI, 1.01, 1.10), *p*=.028) and depressive symptoms at age 6 (OR=1.22, (95%CI, 1.01, 1.48), *p*=.037); however, the combined effect was small (Nagelkerke proxy R^2=.021, Cox & Snell=.013). Attrition when children were 10 years old was higher among those with lower inhibition (OR=1.06, (95%CI, 1.02, 1.11), *p*=.007), symptoms of depression (OR=1.29, (95%CI, 1.06, 1.58), *p*=.013) and ADHD symptoms at age 6 (OR=1.09, (95%CI, 1.02, 1.16), *p*=.01). Again, the combined effect of the study variables predicting attrition was small (Nagelkerke proxy R^2=.029, Cox & Snell=.016). Missing data were handled with a Full Information Maximum Likelihood (FIML) procedure, in which analyses were run on all available data given that cases had values for the dependent variables (n= 802). FIML was used under the assumption that data were missing at random, as attrition was not selective according to food responsiveness at ages 6 and 8, but rather that it was entirely selective according to measured predictors.

Measures

Child eating behavior was measured by parent reports on the 5-item food responsiveness scale (α=.65 to .71 for ages 6, 8 and 10 in the present sample) of the Children's Eating Behaviour Questionnaire (CEBQ) (Wardle, Guthrie, Sanderson, & Rapoport, 2001). Illustrative items include "Given the choice, my child would eat most of the time" and "Even if my child is full up, she/he finds room to eat her/his favorite food". Responses are measured on a 5-point scale ranging from "never" to "always". CEBQ has previously shown good test-retest reliability (Wardle et al., 2001) and has been validated against behavioral measures of eating (Carnell & Wardle, 2007).

Child inhibition was assessed using the inhibition subscale of the teacher-reported version of the Behaviour Rating Inventory of Executive Functions (BRIEF) (Isquith, Gioia, & Espy, 2004), measuring the ability to stop one's behavior at the appropriate time. Inhibition is a continuous scale consisting of 10 items (e.g. "Has trouble putting the brakes on his or her actions even after being asked"; α=.95 at age 6 and α=.93 at age 8 in the present sample). Each behavior described is rated as "never", "sometimes" or "often" present; a higher score indicates *more problems* with inhibition. BRIEF has shown good test-retest reliability and validity (i.e. converges with similar rating scale instruments) (Roth et al., 2014).

Child symptoms of ADHD and depression were assessed at age 6 using the Preschool Age Psychiatric Assessment (PAPA) (Egger et al., 2006), a semi-structured psychiatric interview of parents. PAPA was conducted to record symptoms of ADHD and depression according to the Diagnostic and Statistical manual of Mental Disorders, Fourth Edition (American Psychiatric Association, 1994). When the children were 8 years old, the Child and Adolescent Psychiatric Assessment (CAPA) (Angold & Costello, 2000) was used to interview both children and parents about symptoms of depression (also according to DSM-IV criteria), and a symptom was considered present if reported by either parent or child. Only parents were questioned about ADHD. The PAPA and CAPA use a structured protocol including both required and optional follow-up questions and were administrated by trained personnel with at least a bachelor's degree in a relevant field. Variables for ADHD and depression were created using symptom counts (i.e. continuous variables). At 6 years of age, 9% of the interview audio recordings were recoded by blinded raters, and the inter-rater reliability (ICC) was .90 for depression and .97 for ADHD. For the CAPA, 15% of the interviews were recoded by blinded raters and the resulting ICC was .87 for depression and .90 for ADHD.

Parental feeding practices were measured by means of the Parent Feeding Style Questionnaire (PFSQ) (Wardle et al., 2002). The parent-reported PFSQ assesses four

dimensions of feeding, two of which are the focus of this report: Control over eating (10 items, e.g. “I decide how many snacks my child should have”) and instrumental feeding (4 items, e.g. “I reward my child with something to eat when she/he is well-behaved”). All items are rated on a 5-point scale from 1 (never) to 5 (always). The PFSQ scales have previously demonstrated good test-retest reliability (Wardle, et al., 2002), and has been validated in different countries (Tam, Keung, Lee, Lo, & Cheung, 2014; Wardle et al., 2002). In the present study, the internal consistency was α=.66-.67 for both instrumental feeding and control over eating at ages 6 and 8 – which is comparable to the internal consistency reported by the developers of the PFSQ (Wardle et al., 2002).

Parental restrained eating was measured by the restraint subscale of the Eating Disorder Examination Questionnaire (EDE-Q) (Fairburn & Beglin, 1994), capturing restriction of food and avoidance of certain types of food. It consists of 5 items measured on a 7-point scale (e.g. “Have you been deliberately trying to limit the amount of food you eat to influence your shape or weight, whether or not you have succeeded?”; α=.74 at age 6 and α=.73 at age 8 in the present sample). EDE-Q, including the Restraint subscale, is based on the original EDE interview (Fairburn & Cooper, 1993), with which it also correlates highly (Fairburn & Beglin, 1994). Reliability and validity of EDE-Q are well-documented (for a review, see Berg, Peterson, Frazier, & Crow, 2012).

Covariates. When the children were 6 and 8 years old, *child and parent BMI* were determined based on digital assessments of weight (Tanita BC420MA) and height (Heightronic digital stadiometer: QuickMedical, Model 235 A). A correction of 0.5 kg was applied because of light indoor clothing. For the children, standardized BMI according to age and gender (SDS) was calculated (T. J. Cole, Freeman, & Preece, 1998; Pan & Cole, 2012).

Results

We first outline the statistical procedures used before presenting descriptive statistics and results of model fitting and main analyses.

Analysis Plan

All analyses were performed in Mplus version 7.4 (Muthèn & Muthèn, 1998-2015), using a robust maximum likelihood estimator which provides robust standard errors and is robust to moderate deviations from multivariate normality. Furthermore, all analyses were performed using probabilty weights due to the screen-stratified sample in this study. The probability weights corresponded to the number of children in the population in a specific stratum divided by the number of participating children in that stratum to produce accurate population estimates. Preliminary analyses were run to detect multicollinearity issues and thus potential problems with empirical underidentification. Tolerance and variance inflation factors (VIF) (Allison, 1999) were calculated for each of the predictors at both time points; no issues with multicollinearity emerged (tolerance=.70-.98; VIF=1.02-1.43).

To test child- and parent predictors of food responsiveness, a fixed effects regression model was constructed using structural equation modelling (SEM) (Allison, 2009). Fixed effects models hold the promise of illuminating longitudinal relationships while adjusting for all *time-invariant* confounders, even though those factors are not measured (Firebaugh et al., 2013). An advantage of using SEM for fixed effects regression, is that it allows specifying the relations between model parameters to arrive at a best-fitting model, while effectively handling missing data by using the Full Information Maximum Likelihood (FIML) procedure (Allison, 2009).

Children's food responsiveness at ages 8 and 10 were regressed on child- and parent predictors measured two years earlier (i.e., at ages 6 and 8, respectively). To include potential autoregressive effects in children's eating behavior (i.e. stability), food responsiveness at ages 8 and 10 were regressed on food responsiveness at ages 6 and 8, respectively. Fixed effects

were added to the model by consructing a latent variable loading on food responsiveness at age 8 and 10; this latent time-invariant variable was allowed to correlate with the initial levels of food responsiveness at age 6 as well as the predictors, and all predictors were set to correlate.

We had to impose some constraints on the model due to foreseen negative degrees of freedom. Applying a Wald test, we examined whether the autoregressive effect of food responsiveness at age 6 was similar to that at age 8 – which turned out to be the case (Wald=.27, df=1, p=.60). Accordingly, we constrained the stability of food responsiveness to be similar at both time points, enabling us to test a (saturated) fixed effects model with zero degrees of freedom. We also evaluated whether correlations between predictors at age 6 were similar to correlations between the same predictors at age 8 by conducting Wald tests. These indicated that none of these correlations differed by age; we thus set them to be similar at both ages, thereby increasing the degrees of freedom and thus the statistical power of the model.

Fixed effects models have limited statistical power because they rely on within-person variance exclusively. Random effects models, on the other hand, take advantage of both within- and between-subject information and are both more parimoneous and powerful than fixed effects models (Firebaugh et al., 2013). However, random effects models presuppose that predictors are uncorrelated with the fixed effects latent factor (which represents all unmeasured time-invariant confounders), an assumption that may not necessarily be correct. To determine whether a fixed- or random effects model fit the data best, we used the Satorra-Bentler's scaled chi-square test (Satorra & Bentler, 2001), which is a functional equivalent to the Hausman test (1978). If a random effects model is ruled out due to poor fit to the data, hybrid models (i.e. models where insignificant correlations between predictors and the fixed latent variable are set to zero) retain the fixed effects advantage while preserving statistical

power (Allison, 2009; Firebaugh et al., 2013); we thus examine how a hybrid model fits the data compared to pure fixed or random effects models.

Descriptive Statistics

Estimated means, standard deviations and correlations between all study variables are presented in Tables 1 and 2. As can be seen, most parents reported relatively higher levels of control over eating compared to instrumental feeding. Further, the level of food responsiveness was comparable to earlier findings (Leventakou et al., 2016; Rodgers, Paxton, Massey, et al., 2013) and strong continuities in children's food responsiveness were observed (see Table 2).

Model Fitting Results

Inspection of Table 3 indicates that a fixed effects model in which the stability in food responsiveness and the correlations between predictors were similar at both ages (M3) fitted the data better than a fixed effects model where only the stability in food responsiveness was similar at both ages (M2). Thus, we proceeded to compare this better-fitting fixed effects model (M3) with a a random effects model where the stability in food responsiveness and the correlations between predictors were similar at both ages (M4); again, the fixed effects model (M3) fitted the data better (see Table 3).

The results from this fixed effects model (M3) revealed that six of the sixteen predictors and covariates were significantly correlated with the time-invariant factor, which represents all time-invariant unmeasured confounders. Significantly associated with the time-invariant factor, then, were child depression at age 6 (r=.11, p=.038) and 8 (r=.20, p=.005); child ADHD symptoms at age 6 (r=.10, p=.047); instrumental feeding at age 6 (r=.35, p<.001) and 8 (r=.30, p<.001); and control over eating at age 6 (r=-.11, p=.038). In the hybrid model (M5), all of these predictors were allowed to correlate freely with the time-invariant factor, whereas all of the other correlations between the time-invariant factor and the

predictors were set to zero. This hybrid model (M5) did not evidence a worse fit to the data than the fixed effects model (M3) (see Table 3). Because a hybrid model is both more parsimoneous and has greater statistical power than a fixed effects model (Firebaugh et al., 2013), M5 was preferred.

Predictors of Children's Food Responsiveness

As displayed in Table 4, more child ADHD symptoms and greater parental dietary restraint predicted increased food responsiveness at both ages 8 and 10. Further, higher child BMI at age 8 forecasted increased food responsiveness only at age 10, but a Wald test indicated that the effect of BMI at age 8 was not significantly different from the effect of BMI at age 6 (Wald= .44, *df*= 1, *p*= .51).

Discussion

The main objective of the current study was to explore child- and parent predictors – and thus potential determinants – of children's food responsiveness, based on observations from a large and representative sample of Norwegian children followed from age 6 to 10. After accounting for all unmeasured time-invariant confounding variables, including genetics, results indicated that more ADHD symptoms in children and greater parental dietary restraint predicted increased food responsiveness in children at both ages 8 and 10. The other predictors considered – child depression, inhibition problems, and parental feeding practices – did not forecast eating behavior. We elaborate on these findings below.

The Intrapersonal Level: Child Characteristics

As hypothesized, symptoms of ADHD in children predicted more food responsiveness at 8 and 10 years of age. These findings are in line with previous research showing that symptoms of ADHD are associated with loss of control over eating (Reinblatt et al., 2015) and predict binge eating (Sonneville et al., 2015) in school-aged children. Importantly, our findings also correspond to the observations of Leventakou et al. (2016), who found ADHD

symptoms, but not measured cognitive deficits, to be (positively) related to food responsiveness in a community-based study of preschoolers. We thus extend this study by discovering that ADHD symptoms predict such eating behavior in older children as well. Leventakou et al. (2016) speculate that the fat and obesity-associated transcript gene (FTO) allele at rs9939609 plays a role in both eating behavior and ADHD; however, genetic factors, which qualify as time-invariant ones, were accounted for in the present study. Thus, our results cannot be due to genetic confoundment or mediation.

In contrast to our results, prior investigations have documented links between inhibitory control and eating (Levitan et al., 2015; Pieper & Laugero, 2013) and that lower cool executive functioning prospectively predicts more food responsiveness (Groppe & Elsner, 2015). Importantly, Groppe and Elsner (2015) treated cool executive functions as a latent variable loading on inhibition in addition to shifting and working memory updating; thus, other aspects of children's cognitive problems could prove more important rather than limited inhibition specifically. The conflicting results might also be due to methodological factors, as differences between test measures, which was used by Groppe and Elsner (2015), and the Behavioural Rating Inventory of Executive Functions (BRIEF) used in the current inquiry have been reported (McAuley, Chen, Goos, Schachar, & Crosbie, 2010).

Our findings indicate that there might be aspects of ADHD other than inhibition problems that affect children's food responsiveness. Inhibition, or more precisely disinhibition, is considered one component of the construct of impulsivity (Nigg, 2017). However, impulsivity involves other processes in addition to disinhibition, which also characterize individuals with ADHD (Nigg, 2017). Indeed, children who are more impulsive are also more likely to have higher levels of food responsiveness (Farrow, 2012). Further, the hyperactivity seen in ADHD often relates to fine or gross motor activity as well as talkativeness; thus it is viable that hyperactivity could also extend to eating, such as eating

more and eating faster – behaviors shown to correlate with food responsiveness (Carnell & Wardle, 2007). Therefore, our findings could imply that children with symptoms of ADHD are more food-responsive specifically due to more hyperactive and/or impulsive behavior, and not specifically due to problems with inhibition. Only future research can resolve this issue.

Turning to depression, our study is the first to examine the impact of depression symptoms on food responsiveness in middle childhood. Whereas prior research found depression to be positively associated with food responsiveness in adolescents (Hou et al., 2013; Tanofsky-Kraff et al., 2008), we failed to detect a similar effect in middle childhood. Although methodological differences may partly explain the conflicting findings (e.g., use of different measures of depressive symptoms and eating behavior), developmental differences may also be at play. Depression is much more common in adolescents compared with school-aged children (Avenevoli et al., 2008), therefore the potential effect of depression on eating behavior may not be evident until later in the life span.

The Interpersonal Level: Parent Characteristics

Recall that when it came to which, if any, characteristics of parents predicted children's eating behavior, our results indicated that higher levels of parental dietary restraint predicted more food responsiveness in children at both 8 and 10 years of age. Not only has earlier research revealed positive links between parents' and children's disturbed eating patterns (Stice, 2002), but dietary restraint specifically has also been associated with the emergence of overeating from infancy to the preschool period (Stice et al., 1999). In addition, it has been found to predict heavier weight in preschoolers (Hood et al., 2000; Rodgers, Paxton, McLean, et al., 2013).

The present work extends such prior research in showing that parental restrained eating forecasts eating behavior in older children as well. To be appreciated, of course, is that our work overcame a limit of past research, by controlling for genetic effects and those of all

other time-invariant factors, given our reliance on a hybrid fixed effects model. Engaging in dietary restraint might be a response to parents themselves being food-responsive and thus having increased risk of gaining weight; however, this explanation cannot account for our findings. In light of this critical strength of our study, social modeling (Cruwys et al., 2015) may be a more likely mechanism than genes shared by parents and children when it comes to explaining the discerned relation between parental restrained eating and food responsiveness, although this assumption was not tested in the current inquiry. Importantly, modeling typically implies that children observe a model and directly copy the behavior of the model (Dovey, 2010), which might, at first glance, indicate that children of parents who display dietary restraint would be expected to be *less*, rather than *more* food approaching (i.e. show lower vs. higher levels of food responsiveness). If so, how can the current findings be explained?

When parents restrict their own food intake (e.g. avoiding certain types of food), as when dieting, children may think that the food which is being avoided is particularly tempting or desirable – and thus something their parents may particularly enjoy. Therefore, it is possible that children could perceive this food as more attractive due to its status as "forbidden". Some support for this possibility comes from experimental evidence showing that prohibition of food results in greater desire for and consumption of the target food by children (Jansen, Mulkens, & Jansen, 2007). Similarly, Fisher and Birch (1999) found that children focused significantly more on a particular type of food (i.e. the food elicited more positive comments, more requests for it, and more attempts to obtain it) when restricted access was imposed compared to a similar type of palatable control food which was freely available – even though they initially had the same level of preference for both types of food (Fisher & Birch, 1999). In addition to this "forbidden fruit" pathway, it is reasonable to assume that parents high in dietary restraint also display food responsiveness (Groppe &

Elsner, 2014) – which may be observed and potentially modeled by their children. Thus, parents may act as unintentional role models (Palfreyman et al., 2013), communicating their eating behavior indirectly, and children thus adopt this eating behavior. If our findings are replicated, future studies should address the mechanisms responsible for this link. Further, in addition to dietary restraint, future research should also aim to examine other parental eating behaviors as potential determinants of children's eating, such as disinhibited eating (Cutting, Fisher, Grimm-Thomas, & Birch, 1999).

Contrary to what we expected, instrumental feeding and control over eating did not predict food responsiveness. These results are surprising given that previous research has reported positive cross-sectional associations between these feeding practices and food responsiveness cross-sectionally (Ainuki & Akamatsu, 2011; Carnell et al., 2014; Webber et al., 2010) and prospectively between instrumental feeding and food responsiveness (Steinsbekk et al., 2016). Our failure to detect that parental control over eating uniquely predicted eating behavior is, however, consistent with some past research (Gregory et al., 2010; Rodgers, Paxton, Massey, et al., 2013).

Once again, where findings have proven inconsistent across studies, it could be due to reliance on different measures of feeding practices. In addition, the modest internal consistency of our measures of feeding practices could have undermined our ability to detect effects. Differences between findings reported herein and elsewhere could also be the result of the fact that we controlled for potentially important confounders that are stable over time.

Strengths and Limitations

This study has many strengths, including a large representative sample of children in addition to a longitudinal design. As noted already, perhaps the most important strength of this inquiry was the hybrid fixed effects analytic method that took into account time-invariant confounding factors.

Our exclusive reliance on parental reports of children's eating behavior is the first limitation we must acknowledge. Laboratory-based assessments of eating behavior are considered the gold standard, but are too time- and cost-consuming to be implemented in a large, epidemiological study like TESS. Although eating behavior as measured by the Children's Eating Behaviour Questionnaire (CEBQ) reflects eating behavior from a parent's perspective and may thus be biased, it is also noteworthy that eating behavior measured by CEBQ has proven to reflect eating behavior observed in behavioral tests (Carnell & Wardle, 2007).

The use of the Behaviour Rating Inventory of Executive Functions (BRIEF) is another limitation we must acknowledge, as low correspondence between test measures and BRIEF has been documented (McAuley et al., 2010). Therefore, we cannot exclude the possibility that the use of test measures might have yielded different results; again, such measures are unfortunately too time- and cost-consuming in a large study such as this one.

To be appreciated, is that the risk of inflated associations between predictors and outcomes due to common method variance can be ruled out by the hybrid fixed effects approach, as this approach accounts for any effect of measures being completed by the same persons. However, what this method could not rule out, representing another limitation of our study, is the possibility of third-variable effects involving *time-varying* factors. Thus, for example, state effects – like those involving specific thoughts, emotions or behaviors in a given situation (e.g., when parents were completing our questionnaires) that differ from one's usual way of thinking, feeling or behaving – could have contributed to our findings (Gartstein, Bridgett, Dishion, & Kaufman, 2009). Although time-invariant reporting bias was accounted for, this was not the case for time-varying reporting bias such as state effects.

Conclusions

The present research aimed to examine child- and parent predictors of food responsiveness in middle childhood, using a longitudinal design and applying a hybrid fixed effects model that adjusts for all unmeasured time-invariant confounders. Symptoms of ADHD in children and restrained eating in parents predicted more food responsiveness at both ages 8 and 10. The results of our investigation extend the limited literature on the development of eating behavior in middle childhood and may inform efforts to prevent obesity and eating problems.

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Table 1

Descriptive statistics for all study variables

	Age 6				Age 8				Age 10			
	Mean	S.D.	Min	Max	Mean	S.D.	Min	Max	Mean	S.D.	Min	Max
Food responsiveness	1.90	.47	1.00	4.20	1.87	.49	1.00	4.60	1.89	.53	1.00	4.20
Child inhibition	11.88	3.60	10.00	30.00	11.51	3.07	10.00	28.00	-	-	-	-
Child depression symptoms	.52	.86	.00	5.00	.47	.79	.00	5.00	-	-	-	-
Child ADHD symptoms	1.29	2.24	.00	16.00	1.23	2.41	.00	17.00	-	-	-	-
Child BMI SDS	-.07	.92	-2.86	2.63	.14	.92	-2.37	2.91	-	-	-	-
Parental instrumental feeding	1.64	.48	1.00	3.00	1.48	.45	1.00	4.00	-	-	-	-
Parental control over eating	4.06	.36	2.38	4.90	3.98	.37	2.70	5.00	-	-	-	-
Parental restrained eating	1.94	1.14	1.00	7.00	1.90	1.10	1.00	5.80	-	-	-	-
Parent BMI	25.51	4.54	17.94	49.45	25.62	4.43	16.88	51.63				

Table 2

Bivariate correlations between all study variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. Food responsiveness 6 years	-																		
2. Food responsiveness 8 years	.65***	-																	
3. Food responsiveness 10 years	.61***	.67***	-																
4. Child inhibition 6 years	.04	.04	.04	-															
5. Child inhibition 8 years	.01	.01	-.03	.70***	-														
6. Child depression symptoms 6 years	.08*	.09	.06	.12**	.12**	-													
7. Child depression symptoms 8 years	.11*	.15*	.08	.16**	.13**	.27***	-												
8. Child ADHD symptoms 6 years	.11**	.15***	.09*	.32***	.34***	.35***	.20***	-											
9. Child ADHD symptoms 8 years	.06	.07*	.06	.36***	.46***	.24***	.29***	.58***	-										
10. Child BMI SDS 6 years	.07	.05	.11*	.03	.06	-.09	-.16**	.002	-.03	-									
11. Child BMI SDS 8 years	.09	.10*	.17***	.02	.01	-.09	-.14**	-.01	-.05	.84***	-								
12. Parental instrumental feeding 6 years	.32***	.28***	.25***	.01	.02	.11**	.08	.05	.06	-.11	-.09	-							
13. Parental instrumental feeding 8 years	.21***	.25***	.17***	-.02	.04	.08	.09	-.01	.04	-.06	-.02	.56***	-						
14. Parental control over eating 6 years	-.11**	-.05	-.06	-.001	.07	.00	-.01	.03	.01	.04	-.05	-.18***	-.08	-					
15. Parental control over eating 8 years	-.08	-.01	.03	.07	.06	.03	.05	.08	.04	.001	-.03	-.10*	-.16***	.56***	-				
16. Parental restrained eating 6 years	.07	.12**	.07	.003	.03	.05	-.00	.05	.01	-.07	-.06	.03	.01	.02	.01	-			
17. Parental restrained eating 8 years	.13**	.14***	.19***	.02	.03	.03	.06	-.02	-.01	-.001	-.002	.08	.13**	-.02	-.03	.54***	-		
18. Parent BMI 6 years	-.04	.02	.02	-.01	.05	.02	.01	.03	.04	-.02	.05	-.01	.03	.01	-.03	.29***	.32***	-	
19. Parent BMI 8 years	-.01	.05	.06	-.002	.06	.08	.05	.01	.02	.01	.06	-.01	.002	.03	-.01	.27***	.32***	.92***	-

Note. *p<.05; **p<.01; ***p<.001

Table 3

Results of model fitting procedure

	χ^2	df	p-value	$\Delta\chi^2$	df	p-value	RMSEA[b] (90% CI)	SRMR[c]	CFI[d]	TLI[e]
M1: Baseline model[a]	765.178	35	<.001							
M2: Fixed effects, just-identified	0.00	0	<.001				.00 (.00, .00)	.00	1.0	1.0
M3: Fixed effects, correlations between predictors similar at both ages	22.099	28	.78	22.099	28	.78	<.001 (<.001, .02)	.02	1.00	1.01
M4: Random effects, correlations between predictors similar at both ages	80.761	45	<.001	58.494	17	<.001	.03 (.02, .04)	.03	.951	.962
M5: Hybrid model of fixed effects and random effects, correlations between predictors similar at both ages	**38.452**	**38**	**.45**	**17.297**	**10**	**.068**	**.004 (<.001, .02)**	**.02**	**.999**	**.999**

Note. All models are nested and compared with the next model (exception: M5 is compared with M3 rather than M4 because M3 was preferred over M4); $\Delta\chi^2$ is corrected according to Satorra-Bentler's procedure; preferred model in bold; stability in food responsiveness similar at both ages 6 and 8 in all models tested; in accordance with Wald tests, correlations between predictors at age 6 are fixed to be similar to correlations between the same predictors at age 8 in the last three models (M3-M5).

[a] The baseline model is an unstructured model (null model/null hypothesis) assuming zero covariation between the observed variables; [b] Root mean square error of approximation; [c] Standardized root mean square residual; [d] Comparative fit index; [e] Tucker Lewis index

Table 4

Predictors of children's food responsiveness at ages 8 and 10

	Food responsiveness			
	B	95% CI	*β*	p-value
Predictors at age 6		Age 8		
Child inhibition	-.004	-.01; .01	-.03	.45
Child depression symptoms	-.02	-.06; .03	-.03	.42
Child ADHD symptoms	.03	.01; .04	.12	.004
Parental instrumental feeding	-.02	-.12; .07	-.02	.66
Parental control over eating	.03	-.07; .12	.02	.54
Parental restrained eating	.05	.02; .08	.12	<.001
Covariates at age 6				
Child BMI SDS	.05	-.002; .11	.10	.058
Parent BMI	-.002	-.01; .01	-.02	.63
Predictors at age 8		Age 10		
Child inhibition	-.01	-.03; .002	-.07	.081
Child depression symptoms	-.04	-.10; .01	-.06	.15
Child ADHD symptoms	.02	.002; .04	.09	.027
Parental instrumental feeding	-.09	-.20; .01	-.08	.083
Parental control over eating	.08	-.02; .18	.06	.12
Parental restrained eating	.08	.05; .11	.16	<.001
Covariates at age 8				
Child BMI SDS	.07	.02; .13	.12	.010
Parent BMI	<.001	-.01; .01	.003	.95

Note. *B*=unstandardized parameter estimates; *β*=standardized parameter estimates.

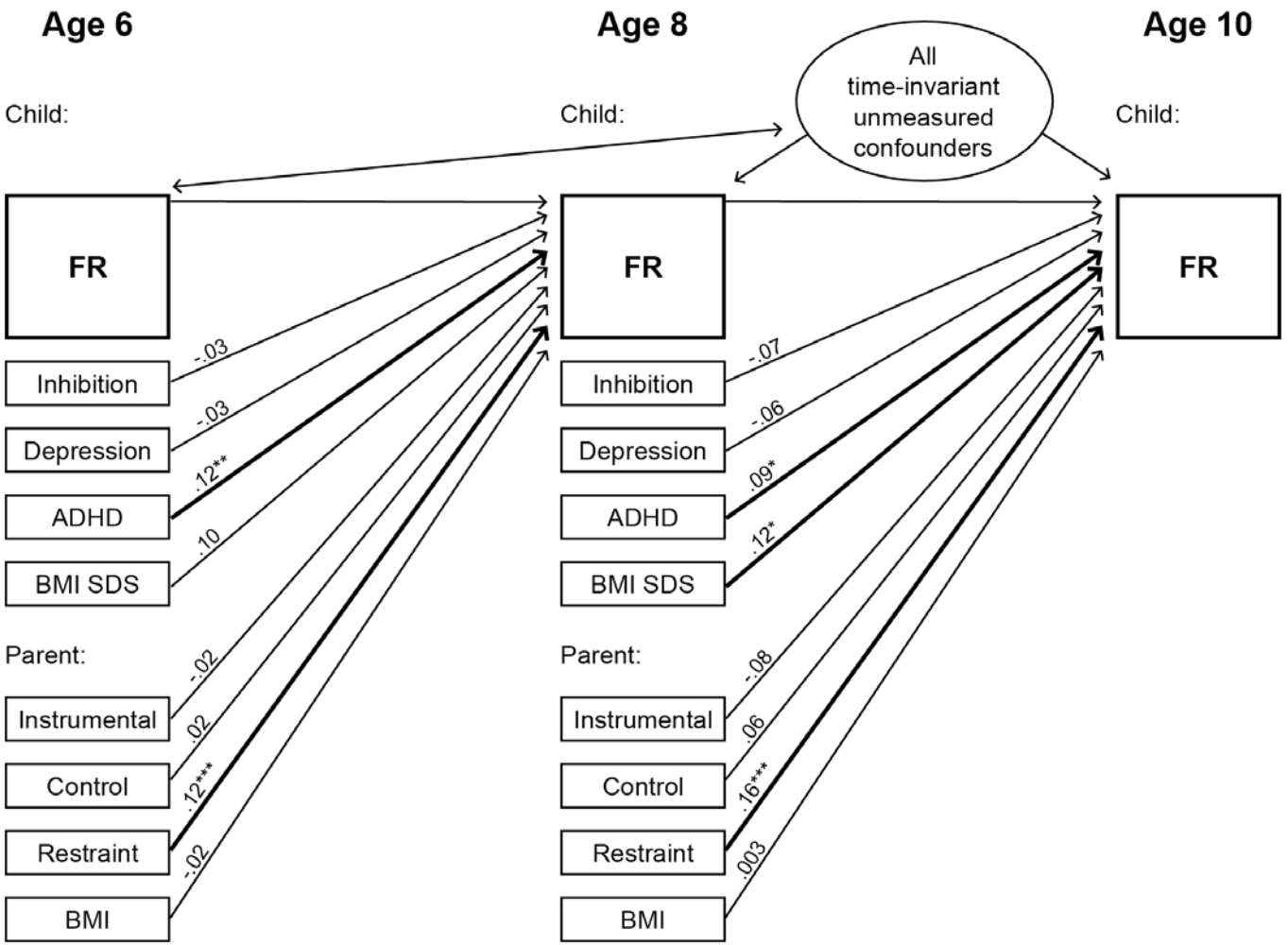

Figure 1. Path diagram of the hybrid fixed effects model of children's food responsiveness. All regression paths are displayed with standardized estimates, and significant paths are highlighted in bold (*p<.05; **p<.01; ***p<.001). Within-time correlations between all measures, correlations between predictors and correlations between predictors and the time-invariant latent factor are not shown. FR=food responsiveness. Child and parent BMI included as covariates at both time points.