Predictors of Change in BMI From the Age of 4 to 8
Silje Steinsbekk,1 PhD and Lars Wichstrøm,1,2 PhD

1Department of Psychology, Norwegian University of Science and Technology (NTNU) and 2NTNU Social Science

All correspondence concerning this article should be addressed to Silje Steinsbekk, PhD, Department of Psychology, Norwegian University of Science and Technology, 7491 Trondheim, Norway. E-mail: Silje.Steinsbekk@svt.ntnu.no

Received August 18, 2014; revisions received April 27, 2015; accepted May 15, 2015

Abstract

Objectives To examine appetite traits, level of physical activity, and television (TV) time as predictors of change in Body Mass Index Standard Deviation Score (BMI SDS) from age 6 to 8 and to explore the effect of BMI SDS (from age 4) on appetite traits. Methods In all, 995 Norwegian children participated at age 4; 760 and 687 of these children took part in the assessment at ages 6 and 8, respectively. Appetite traits were assessed using the Children’s Eating Behavior Questionnaire, activity was measured using accelerometers, and TV time was based on parental reports. Results High food responsiveness predicted a steeper increase in BMI SDS. A reversed effect was also observed: High BMI SDS predicted increased food responsiveness and decreased satiety responsiveness. Physical activity and TV time were unrelated to BMI SDS. Conclusion Children whose eating is especially triggered by the sight and smell of food show prospective increased weight gain. Excess weight and weight gain also predict increased food-approaching behavior.

Key words: children; eating and feeding disorders; health behavior; longitudinal research; obesity.

According to a recent review, more than every third American child is overweight or obese (Ogden, Carroll, Kit, & Flegal, 2014). Childhood obesity is associated with multiple negative health outcomes (Reilly et al., 2003) including metabolic syndrome (Friend, Craig, & Turner, 2013), type 2 diabetes (Mayer-Davis, 2008), and hypertension (Rosner, Cook, Daniels, & Falkner, 2013), as well as mental health problems (Kalarchian & Marcus, 2012), reduced self-esteem, and impaired quality of life (Griffiths, Parsons, & Hill, 2010). Overweight and obesity tend to persist from childhood into adulthood, and the risk of adult overweight increases the longer a child has been overweight (Singh, Mulder, Twisk, van Mechelen, & Chinapaw, 2008). It is therefore vital to identify factors contributing to the development and continuity of an unhealthy weight starting at an early age.

The behavioral susceptibility theory of obesity states that individual differences in appetite traits can explain why some individuals become overweight while others do not (Carnell & Wardle, 2008a, b). Appetite traits are biologically influenced dispositions toward food, such as reduced satiety responsiveness or heightened food cue responsiveness (Carnell, Kim, & Pryor, 2012), characteristics that have been shown to be fairly stable during childhood (Ashcroft, Semmler, Carnell, van Jaarsveld, & Wardle, 2008). Food responsiveness refers to the tendency to eat in response to food cues such as the sight and smell of food (Faith, Carnell, & Kral, 2013), whereas enjoyment of food involves a more general interest in food and desire to eat (Wardle, Guthrie, Sanderson, & Rapoport, 2001). Food responsiveness, enjoyment of food, and emotional overeating are positively associated with overweight (Carnell & Wardle, 2008a; Croker, Cooke, & Wardle, 2011; Webber, Hill, Saxton, Van Jaarsveld, & Wardle, 2009) and have been named “food-approach” traits. Satiety responsiveness is the capacity...
Predictors of Change in BMI

1057

to adjust eating in response to internal feelings of satiety or fullness, and slowness in eating is a measure of eating rate. Both are negatively associated with weight (Carnell & Wardle, 2008a; Webber et al., 2009) and therefore termed “food-avoidant” traits.

Carnell and Wardle (2008b) argue that identifying weight-related appetite traits at an early age allows interventions to be implemented before significant weight gain has occurred. Importantly, such an assumption presupposes a causal relationship between appetite traits and weight development. However, the majority of studies on the relationship between appetitive traits and weight are cross-sectional, and none of the studies included in a recent review (French, Epstein, Jeffery, Blundell, & Wardle, 2012) examined food responsiveness, satiety responsiveness, or enjoyment of food, which are the appetite traits most consistently shown to be associated with weight in pediatric studies (Carnell & Wardle, 2008a; Croker et al., 2011; Webber et al., 2009). Therefore, longitudinal studies are needed to gain knowledge on how appetitive traits affect the development of obesity in children.

Notably, though, cross-sectional associations between appetite traits and weight might also result from the opposite direction of influence, that is, that eating behavior is an effect of weight, not merely affects weight. Cross-sectional studies have repeatedly shown that children who eat in the absence of hunger (i.e., extensive snacking after consuming a meal to the point of satiety) tend to have higher body mass indices (BMIs) than children who do not (French et al., 2012). However, when adjusting for their initially higher BMI, children who eat in the absence of hunger do not seem to have a higher weight increase than children who abstain from eating when they are full (Butte et al., 2007). Moreover, the findings of one study actually suggested that a high BMI may drive such eating in the absence of hunger: Although being overweight at age 5 (i.e., >85% percentile) was not concurrently associated with eating in the absence of hunger, it did predict an increase in the inclination to do so at ages 7 and 9 (Shunk & Birch, 2004). Given the paucity of studies and the possibility that eating behavior may be an effect of weight gain, we therefore present the first attempt to examine the impact of a range of appetite traits on changes in BMI Standard Deviation Scores (BMI SDS) and vice versa during the early school years.

Most simply stated, overweight is the result of an energy imbalance, with energy intake on one side of the energy balance equation and physical activity (PA) on the other. In a recent review of factors associated with the development of excessive fatness in children and adolescents, seven prospective cohort studies using objectively measured PA were identified, six of them reporting that PA reduces fat mass over time (Pate et al., 2013). Notably, however, some of these studies found no effect of PA on fat mass when adjusting for the initial level of fat mass (Janz et al., 2009), and other studies reported no association between PA and weight-related outcomes (Hjorth et al., 2014; Metcalf et al., 2011). This inconsistency is likely related to measurement issues, such as the use of different activity measures (e.g., accelerometers vs. questionnaires), heterogeneity in participant characteristics, diversity of follow-up periods, and different measures of adiposity (e.g., BMI, waist circumference, skin-fold thickness) (Pate et al., 2013). The authors of the review noted that research examining longer follow-ups, especially covering critical periods for changes in BMI such as the adiposity rebound period (ages 4–7) (Taylor, Grant, Goulding, & Williams, 2005) is needed. Our inquiry therefore extends earlier findings by examining the effect of PA on changes in BMI SDS during this critical period of development.

Independent of PA, sedentary behavior may also be important for children’s adiposity. Higher levels of sedentary behavior are associated with weight in both cross-sectional (Tremblay et al., 2011) and longitudinal (Rey-Lopez, Vicente-Rodriguez, Biosca, & Moreno, 2008) studies. Notably, prospective studies of objectively assessed (accelerometer) sedentary behavior as a predictor of adiposity show inconsistent findings (Pate et al., 2013). Television (TV) time, however, is more consistently related to adiposity (Boone, Gordon-Larsen, Adair, & Popkin, 2007; Hjorth et al., 2014), predicting a steeper increase in BMI (Henderson, 2007). TV time, rather than the broader concept of sedentary behavior, was therefore used as a predictor of BMI SDS change in our study.

In summary, the majority of studies on the relationship between appetite and weight have been cross-sectional, research on the effect of PA on weight development show inconsistent findings, and the relative importance of appetite, PA, and TV time in the development of overweight is unknown. Longitudinal designs simultaneously examining these factors, especially in the critical period surrounding adiposity rebound, are needed to identify potential predictors of weight gain that can be targeted in preventive efforts. The current study therefore examined appetite traits, level of PA, and TV time as predictors of change in BMI SDS from age 4 to 8 years in a large, population-representative sample of Norwegian children. Because BMI in children is related to genetics and socioeconomic status (SES) (Gillero & Jago, 2010; Drenowatz et al., 2010; Stamatakis, Wardle, & Cole, 2010; van Stralen et al., 2012), parental BMI and SES were included as covariates in this research. We hypothesized that higher levels of food responsiveness, enjoyment of food, and emotional overeating would be associated with a greater increase in BMI, whereas satiety responsiveness and slowness in eating would be
associated with a slower increase in BMI. We also hypothesized that higher PA and lower TV viewing would be associated with a slower increase in BMI, although the inconsistencies in earlier findings of PA weaken this prediction. The important aim of this study was to examine activity traits alone and in association with appetite. Finally, given the emerging indications that BMI might influence appetite traits in children, we examined whether higher BMI SDS and increases in BMI SDS would prospectively predict increased food-approaching and reduced food-avoidant behavior.

Methods
Participants and Procedure
All children born in 2003 or 2004 in Trondheim, Norway, were invited to participate in the study (N = 3,456). A letter of invitation together with the Strengths and Difficulties Questionnaire (SDQ) 4–16 version (Crone, Vogels, Hoekstra, Treffers, & Reijneveld, 2008), a screening assessment for emotional and behavioral problems, was sent to the children’s homes. Parents brought the completed SDQ when they attended the well-child clinic for the routine health check at age 4 years. Because almost all children in the two cohorts appeared at the checkup (97.2%), the sample is effectively a community sample. The nurse at the clinic informed the parent about the present study using procedures approved by the Regional Committee for Medical and Health Research Ethics and obtained written consent to participate. Of those who were asked to participate (n = 3,016), 82.2% consented. To increase variability, children were placed in four strata according to their SDQ scores. A subsample (n = 1,250) was drawn to participate in the present study. Children with high SDQ scores were oversampled by means of a random number generator. We succeeded in obtaining usable data from 997 participants. At follow-up, 2 years later, data were collected from 797 children, and at age 8 years, 689 children participated. All assessments took place at the University clinic, where parents also completed questionnaires concerning their child and themselves, including demographic information. The mean age of the children was 4.7 years (SD = .30) at Time 1 (T1), 6.7 years (SD = .17) at Time 2 (T2), and 8.8 years (SD = .24) at Time 3 (T3). The majority of informants were mothers (84.8%), 88.9% of the parents were married or cohabitating and they were primarily of Norwegian origin (mothers = 93.0%; fathers = 91.0%). The position of the parent with the highest ranking based on the International Standard Classification of Occupation (ISCO-88; International Labour Office, 1990) was as follows: leaders (5.7%), higher level professionals (25.7%), lower level professionals (39.0%), formally skilled workers (26.0%), and unskilled workers or farmers/fishermen (3.6%). The sample is comparable with the Norwegian population with regard to the parents’ level of education (Statistics Norway, 2012) and children’s BMI (Juliusson et al., 2013). High parental SES predicted attrition from T1 to T3, B = .15, p = .04, indicating that the attrition was to some degrees missing at random.

Setting
Trondheim is the third largest city in Norway with approximately 200,000 inhabitants. The obesity rates in Norway are similar to those reported in other European countries, ranging from 1.4% to 5% for obesity and 13.2% to 19% for overweight (Haug et al., 2009; Juliusson et al., 2010; Kokkvoll, Jeppesen, Juliusson, Flægstad, & Njolstad, 2012; Kolle, Steene-Johannessen, Holme, Andersen, & Anderssen, 2009). Although the same threefold increase in obesity was observed in Norway as well as in other European countries in the past few decades (Juliusson et al., 2007; Lobstein & Frelut, 2003), the prevalence of pediatric obesity is still lower in Norway than in the United States. However, the number of Norwegian children who are physically active for at least 60 min per day is not different from that of American children (Haug et al., 2009).

Measures
Body Mass Index
Weight and height were measured by the health care nurse at the ordinary community health checkup for 4-year-olds (T1) using stadiometers and analogue scales. At T2 and T3, digital scales were used to measure weight (Tanita BC420MA) and height (Heightronic digital stadiometer: QuickMedical, Model 235 A) in children and parents. Correction for light indoor clothing (0.5 kg for children and 1.0 kg for adults) was applied. BMI and BMI SDS were estimated (Cole, Freeman, & Preece, 1995, 1998; Pan & Cole, 2012).

Appetite Traits
The Children’s Eating Behavior Questionnaire (CEBQ) was used to assess appetite traits at age 6. The CEBQ is a parent-report measure with 35 items covering eight appetite traits, of which five were included in the current study because of their association with adiposity: Enjoyment of food (α = .81; e.g., “My child looks forward to mealtimes”), Food responsiveness (α = .65; e.g., “If allowed to, my child would eat too much”), Emotional overeating (α = .75; e.g., “My child eats more when worried”), Slowness in eating (α = .71; e.g., “My child takes more than 30 min to finish a meal”), and Satiety responsiveness (α = .70;
e.g., “My child gets full easily”) (Wardle et al., 2001). Response options are measured on a 5-item Likert scale ranging from “never” to “always.” The CEBQ has shown good validity (Carnell & Wardle, 2007) and test–retest reliability (Wardle et al., 2001).

Physical Activity
PA at age 6 was measured using an ActiGraph GT3X accelerometer (Manufacturing Technology Incorporated, Fort Walton Beach, FL) worn around the waist. In a recent review, ActiGraph was identified as the most validated accelerometer of those examined (Plasqui, Bonomi, & Westerterp, 2013). Children were instructed to wear the monitor for seven consecutive days, except when they were swimming, showering, or bathing. Activity data were processed using accelerometer analysis software (ActiGraph LLC, Pensacola, FL, United States). Analyses were performed using general PA reported in mean counts min\(^{-1}\), which indicates that the counts are summarized based on a user-defined time interval (60 s epoch) and averaged over the total wearing time for each individual (in minutes) to indicate the overall PA (mean counts minute). This accelerometer outcome has proven to be strongly related to free-living PA levels in children (Ekelund et al., 2001). It appears to be a robust parameter that is not affected by epoch length, and can be compared across studies (Dencker & Andersen, 2008). Only daytime activity, defined by a fixed time interval (06:00–24:00), was included in the analysis. Sequences of consecutive zero counts lasting \(\geq 20\) min were interpreted as representing nonwear time and were excluded from each individual recording. Participants who did not manage to record data for at least 3 days, with a minimum of 8 hr of activity per day were excluded from the analysis (9.4%), including lack of data owing to equipment failure. These criteria are comparable with those used in other studies (Riddoch et al., 2004).

TV Time
At T2, the parents were interviewed concerning how long their child watched TV each week and weekend, and a daily mean value in hours was calculated.

Socioeconomic Status
Classification of parents’ occupation was based on the International Classification of Occupations (International Labour Office, 1990), generating six categories ranging from unskilled worker to leader, which were applied as a measure of SES.

Statistical Analyses
All analyses were performed in Mplus 7.0 (Muthén & Muthén, 1998–2010). Change in BMI SDS was estimated using growth modeling (Barker, Rancourt, & Jelalian, 2014; Berlin, Parra, & Williams, 2014), with yearly change in BMI SDS as a metric. A robust maximum likelihood estimator was applied. The analyses were performed on cases with information available on the dependent variable, that is, BMI SDS, and missing data on the independent variables were handled according to a full information maximum likelihood procedure. Because predictors of BMI SDS were measured at T2, only data from T2 and T3 were used in the predictive analyses. The growth model yielded two parameters: the intercept (set at T2), which is the starting level of the growth, and the slope, which represents the linear change in BMI SDS from age 6 to age 8. To model growth with two time points, the residuals of the BMI SDS at each time point were set to zero. The growth parameters were regressed on appetite traits, PA and TV time measured at T2, and adjustments were made for parental BMI and SES. In the first step, bivariate analyses were performed examining one predictor (e.g., food responsiveness) at a time. Next, a multivariate model was tested in which all of the predictors were included. The slope was regressed on the intercept. To model the reverse effect, we conducted analyses in which the intercept (set at age 6) and growth in appetite traits (e.g., satiety responsiveness) from age 6 to age 8 were regressed on the intercept (age 4) and growth in BMI SDS from age 4 to 6. Moreover, the slopes were regressed on their respective intercepts, and parental BMI and SES were included as covariates. Because appetite traits were only measured at two time point, the residuals of CEBQ were set to zero.

Results
Preliminary Analyses
Table I presents the mean BMI and BMI SDS at ages 4 \(\bar{\mu} = 995\), 6 \(\bar{\mu} = 658\), and 8 \(\bar{\mu} = 675\) years for boys and girls. According to the International Obesity Task Force cutoffs (International Obesity Task Force, 2011), 9.3 % of the children were overweight and 0.4% were obese at baseline. At T2, 3.8% were overweight and 0.2% of the participants were obese, whereas the corresponding numbers for T3 were 6.0% and 0.4%, respectively. Mean parental BMI was 25.62 \(SD = 5.46\). The children were reported to watch 1.13 hr of TV a day \(SD = .49\), and their mean PA was 586 \(SD = 209.66\) counts\(\cdot\)min\(^{-1}\). The mean CEBQ scores were: Enjoyment of food 3.44 \(SD = .56\), emotional overeating 1.43 \(SD = .44\), food responsiveness 1.89 \(SD = .47\), satiety responsiveness 2.93 \(SD = .50\), and slowness in eating 2.55 \(SD = .63\), numbers similar to earlier findings (Ashcroft et al., 2008; Mallan, Nambiar, Magarey, & Daniels, 2014). All appetite traits were significantly correlated with BMI SDS at age 6 (Enjoyment of food:
Table I. BMI and BMI SDS at Ages 4, 6, and 8 by Gender

<table>
<thead>
<tr>
<th></th>
<th>Age 4 (N = 995)</th>
<th>Age 6 (N = 658)</th>
<th>Age 8 (N = 675)</th>
<th>Difference in mean from age 4 to age 6</th>
<th>Difference in mean from age 6 to age 8</th>
<th>Difference in mean from age 4 to age 8</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BMI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Girls</td>
<td>15.94 [15.83, 16.05]</td>
<td>15.71 [15.50, 15.86]</td>
<td>16.87 [16.64, 17.10]</td>
<td>14.54 &lt; .001 255.02 1 &lt; .001 80.97 1 &lt; .001</td>
<td>14.63 &lt; .001 230.73 1 &lt; .001 52.31 1 &lt; .001</td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>15.98 [15.88, 16.08]</td>
<td>15.73 [15.57, 15.89]</td>
<td>16.72 [16.50, 16.94]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>BMI SDS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Girls</td>
<td>.12 [ .03, .22]</td>
<td>-.11 [-.20, -.02]</td>
<td>.07 [-.03, .17]</td>
<td>26.38 1 &lt; .001 33.69 1 &lt; .001 1.15 1 .28</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>.20 [.11, .28]</td>
<td>-.00 [-.10, .10]</td>
<td>.25 [.15, .35]</td>
<td>21.19 1 &lt; .001 72.02 1 &lt; .001 .95 1 .33</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. BMI SDS = Body Mass Index Standard Deviation Score.

Table II. Appetite Traits, Levels of Physical Activity, and TV Time Regressed on Intercept (Age 6) and Slope (6–8 Years of Age) of BMI SDS

<table>
<thead>
<tr>
<th></th>
<th>Intercept</th>
<th>Variances (p-value)</th>
<th>Slope</th>
<th>Variances (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>95% CI</td>
<td>P</td>
<td>B</td>
</tr>
<tr>
<td>Predictors (age 6):</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical activity</td>
<td>Bivariate</td>
<td>.00 [-.00, .00]</td>
<td>.03 .35</td>
<td>.00 [.00, .00]</td>
</tr>
<tr>
<td></td>
<td>Multivariate</td>
<td>.00 [.00, .00]</td>
<td>.03 .49</td>
<td>.00 [.00, .00]</td>
</tr>
<tr>
<td>TV time</td>
<td>Bivariate</td>
<td>.13 [-.03, .28]</td>
<td>.07 .11</td>
<td>-.01 [-.06, .03]</td>
</tr>
<tr>
<td></td>
<td>Multivariate</td>
<td>.07 [-.09, .24]</td>
<td>.04 .38</td>
<td>-.04 [-.08, .01]</td>
</tr>
<tr>
<td>Enjoyment of food</td>
<td>Bivariate</td>
<td>.31 [.18, .44]</td>
<td>.19 &lt; .001</td>
<td>.02 [-.02, .05]</td>
</tr>
<tr>
<td></td>
<td>Multivariate</td>
<td>.30 [.16, .16]</td>
<td>.00 .99</td>
<td>.03 [-.03, .08]</td>
</tr>
<tr>
<td>Emotional overeating</td>
<td>Bivariate</td>
<td>.38 [.21, .56]</td>
<td>.19 &lt; .001</td>
<td>-.01 [-.08, .05]</td>
</tr>
<tr>
<td></td>
<td>Multivariate</td>
<td>.14 [-.08, .36]</td>
<td>.07 .22</td>
<td>-.05 [-.13, .03]</td>
</tr>
<tr>
<td>Food responsiveness</td>
<td>Bivariate</td>
<td>.54 [.37, .70]</td>
<td>.28 &lt; .001</td>
<td>.02 [-.03, .08]</td>
</tr>
<tr>
<td></td>
<td>Multivariate</td>
<td>.38 [.14, .62]</td>
<td>.19 &lt; .01</td>
<td>.07 [.00, .13]</td>
</tr>
<tr>
<td>Satiety responsiveness</td>
<td>Bivariate</td>
<td>-.60 [-.73, -.46]</td>
<td>-.33 &lt; .001</td>
<td>.01 [-.04, .05]</td>
</tr>
<tr>
<td></td>
<td>Multivariate</td>
<td>-.50 [-.67, -.33]</td>
<td>-.28 &lt; .001</td>
<td>.04 [-.02, .10]</td>
</tr>
<tr>
<td>Slowness in eating</td>
<td>Bivariate</td>
<td>-.20 [-.32, -.09]</td>
<td>-.14 &lt; .001</td>
<td>-.01 [-.04, .03]</td>
</tr>
<tr>
<td></td>
<td>Multivariate</td>
<td>-.12 [-.23, -.01]</td>
<td>-.08 .08</td>
<td>-.02 [-.06, .02]</td>
</tr>
<tr>
<td>Covariates</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SES</td>
<td>Bivariate</td>
<td>.02 [-.07, .10]</td>
<td>.02 .73</td>
<td>-.02 [-.04, .01]</td>
</tr>
<tr>
<td></td>
<td>Multivariate</td>
<td>.01 [-.08, .10]</td>
<td>.01 .82</td>
<td>-.02 [-.04, .01]</td>
</tr>
<tr>
<td>Parental BMI</td>
<td>Bivariate</td>
<td>.03 [.01, .04]</td>
<td>.16 &lt; .001</td>
<td>.01 [.00, .01]</td>
</tr>
<tr>
<td></td>
<td>Multivariate</td>
<td>.03 [.02, .05]</td>
<td>.18 &lt; .001</td>
<td>.01 [.00, .01]</td>
</tr>
</tbody>
</table>

Note. CI = confidence interval. 95% CI and p are presented for the unstandardized regression coefficient (B). Bivariate = bivariate associations. Multivariate = multivariate model, i.e., adjusted for all other predictors. All appetite traits were allowed to correlate in the multivariate model. Parental BMI and SES were adjusted for in all analyses.

The root mean square error of approximation (RMSEA) = 0.05. As presented in Table II, in the multivariate model, children with high food responsiveness had higher initial BMI SDS (intercept; age 6) and steeper increase in BMI SDS from age 6 to age 8 (slope). Furthermore, high parental BMI predicted higher intercept and comparatively increased BMI SDS from age 6 to age 8. Apart from food responsiveness and parental BMI, none of the other factors examined predicted BMI SDS changes from age 6 to age 8.

BMI SDS as a Predictor of Appetitive Traits

Analyses examining the reverse relationship between BMI SDS and appetite traits, that is, whether BMI

\[ r = .19, \ p < .001; \ \text{emotional overeating: } r = .18, \ p < .001; \ \text{food responsiveness: } r = .27, \ p < .001; \ \text{satiety responsiveness: } r = -.33, \ p < .001; \ \text{slowness in eating: } r = -.13, \ p < .01, \ \text{and age 8 (Enjoyment of food: } r = .22, \ p < .01; \ \text{emotional overeating: } r = .18, \ p < .001; \ \text{food responsiveness: } r = .35, \ p < .001; \ \text{satiety responsiveness: } r = -.37, \ p < .001; \ \text{slowness in eating: } r = -.11, \ p < .01, \) whereas neither PA nor TV time was associated with BMI SDS.

Predictors of Change in BMI SDS

A multivariate growth model including all predictors of change in BMI SDS showed a good fit to the data (\( \chi^2 = 42.82; df = 20; \ p = .01 \); The comparative fit index (CFI) = 0.95; The Tucker-Lewis index (TLI) = 0.95;
SDS affected appetite traits, revealed that the intercept of BMI SDS (age 4) predicted a comparatively higher intercept (age 6) \( (B = .15, 95\% \text{ CI } [.08, .21], \beta = .26, p \leq .001) \) and growth (age 6-8) in food responsiveness \( (B = .04, 95\% \text{ CI } [.02, .06], \beta = .17, p \leq .001) \). The growth of BMI SDS from age 4 to age 6 predicted both higher levels (age 6) \( (B = .31, 95\% \text{ CI } [.17, .44], \beta = .23, p \leq .001) \) and growth (age 6 to 8) of food responsiveness \( (B = .07, 95\% \text{ CI } [.01, .12], \beta = .12, p = .02) \).

Furthermore, comparatively higher BMI SDS at age 4 (intercept) predicted lower initial levels (age 6) \( (B = -.19, 95\% \text{ CI } [-.25, -.14], \beta = -.32, p \leq .001) \) and an additional decrease in satiety responsiveness from age 6 to age 8 \( (B = -.05, 95\% \text{ CI } [-.08, -.03], \beta = -.20, p \leq .001) \). Higher growth in BMI SDS from age 4 to age 6 also predicted lower levels of satiety responsiveness at age 6 (intercept) \( (B = -.35, 95\% \text{ CI } [-.47, -.22], \beta = -.24, p \leq .001) \) as well as decreased satiety responsiveness from age 6 to age 8 (slope) \( (B = -.12, 95\% \text{ CI } [-.17, -.06], \beta = -.18, p \leq .001) \).

The growth of the remaining appetite traits examined in this study was not predicted by BMI SDS, although BMI SDS predicted the level of these traits at age 6: The intercept of enjoyment of food was positively predicted by the level of BMI SDS (age 4) \( (B = .16, 95\% \text{ CI } [.09, .22], \beta = .23, p \leq .001) \), whereas the effect of growth of BMI SDS (age 4-6) on enjoyment of food (age 6) bordered on significance \( (B = .14, 95\% \text{ CI } [.00, .28], \beta = .09, p = .051) \); comparatively higher levels of emotional overeating were predicted by the intercept \( (B = .10, 95\% \text{ CI } [.05, .15], \beta = .19, p \leq .001) \) and growth of BMI SDS \( (B = .18, 95\% \text{ CI } [.04, -.31], \beta = .14, p = .01) \); in contrast, BMI SDS at age 4 predicted lower levels of slowness of eating at age 6 \( (B = -.10, 95\% \text{ CI } [-.17, -.02], \beta = -.12, p = .01) \). Notably, slowness in eating at age 6 was not predicted by the growth of BMI SDS from age 4 to age 6 \( (B = -.14, 95\% \text{ CI } [-.32, .03], \beta = -.08, p = .11) \).

In summary, both BMI SDS at age 4 and the additional growth in BMI SDS from age 4 to age 6 prospectively predicted the initial levels (age 6) and change (from age 6 to 8) in food responsiveness and satiety responsiveness. BMI SDS did not predict growth in the remaining appetite traits examined in this study.

**Discussion**

The current study examined appetite traits, level of PA, and TV time as predictors of change in BMI SDS from age 6 to age 8 in a large and representative sample of Norwegian children. Growth analyses revealed that children with high food responsiveness at age 6 had a steeper increase in BMI SDS from age 6 to age 8. Evidence for the reverse effect was also found: BMI SDS at age 4 and the increase in BMI SDS from age 4 to age 6 predicted decreased satiety responsiveness and increased food responsiveness from age 6 to age 8. PA and TV time were not found to be related to BMI SDS in this study.

Earlier studies show that appetite traits predict increased weight gain in infants (van Jaarsveld, Boniface, Llewellyn, & Wardle, 2014; van Jaarsveld, Llewellyn, Johnson, & Wardle, 2011), but this has not been examined in older children. Our findings on school-aged children partly concur with those of infants by showing high food responsiveness to predict more rapid weight gain. Not too surprisingly, a child who’s eating is especially triggered by the smell and sight of food will eat more in an obesogenic environment where food is easily available and thus gain more weight. However, contrary to the abovementioned studies, we did not find satiety responsiveness and slowness in eating to affect change in BMI SDS, thus suggesting that previously important appetite traits may play a lesser or no role in obesity development as the child ages. Because infants’ innate ability to self-regulate food intake (Birch & Deysker, 1986) decrease with age (Johnson & Taylor-Holloway, 2006), their eating may to a greater extent be shaped by external factors. This shift in the regulation of eating may explain why food responsiveness (eating triggered by external cues), but not satiety responsiveness (the capacity to adjust eating in response to internal feelings of satiety or fullness), predicted change in BMI SDS in the current study. It should be noted though, that the effect of food responsiveness on the increase in BMI SDS was quite small.

Importantly, we also found evidence for the reverse effect: Higher BMI SDS at age 4 and increase in BMI SDS from age 4 to age 6 predicted greater food responsiveness and a weaker increase in satiety responsiveness from age 6 to age 8. Hence, our findings indicate that the previously identified concurrent relationship between appetitive traits and BMI results not only from the effect of BMI on appetite traits but also possibly from the effect of appetite traits on BMI. This accords with the study of Shunk and Birch (2004), showing overweight to predict an increased inclination to eat in the absence of hunger. However, future studies examining the effect of BMI on appetite traits in different cultures and at different ages are needed before reasonable interpretations of the present finding can be presented. Further, before definite conclusions can be drawn, we need to know whether the potential effect of BMI on appetite traits is related to body composition (muscles vs. fat), and whether it holds for both overweight and normal weight individuals (normal phenomenon or pathology?). In the event of such replications, possible biological mechanisms explaining the effect of BMI on appetite traits should be addressed as well.
PA and TV time were unrelated to BMI in our study. Overall, our results join the long line of inconsistent research findings on the relationship between adiposity and PA, and concur with the view that children with higher PA will compensate their energy loss by increased energy intake (Thivel, Aucouturier, Doucet, Saunders, & Chaput, 2013). The findings have also been mixed with regard to the association between TV time and BMI, although some studies have found TV time to predict weight (Danner, 2008; Henderson, 2007). One explanation for our negative result could be that average TV viewing was only 1.1 hr per day, considerably lower than the 2 hr a day that has previously been associated with unfavorable body composition in school-aged children (Tremblay et al., 2011).

As expected, and in accordance with previous research (Silventoinen, Rokholm, Kaprio, & Sorensen, 2010), children’s BMI SDS was associated with parents’ BMI, and unlike some other studies (Fuemmeler, Lovelady, Zucker, & Ostbye, 2013), predicted BMI SDS change even when appetite traits were adjusted for; quite likely there are other aspects of trans-generational transmission of obesity, which are important beyond the appetite traits measured here. SES did not predict BMI SDS, which might be owing to low diversity of SES in Norway, although stronger socioeconomic health inequalities have been observed in the Nordic countries than in many other Western countries despite lower absolute SES differences (Mackenbach et al., 1997).

Although the present study has several strengths, including a large, population-representative sample, objectively measured PA, and the examination of several different predictors of BMI change, some limitations must be noted. First, although the CEBQ has shown good validity, the measure has not been validated in a Norwegian sample. Second, parental evaluations of appetite traits may reflect an element of social desirability, although the advantage of using the CEBQ has shown good validity, the measure has not been validated in a Norwegian sample. Second, parental evaluations of appetite traits may reflect an element of social desirability, although the advantage of using self-report measures has been shown to provide reliable estimates of screen time (Lubans et al., 2011). Furthermore, weight and height were measured differently at age 4 than they were at age 6 and age 8. It should also be noted that the increased risk of obesity is related to an early adiposity rebound, occurring before 5–5.5 years (Taylor et al., 2005). Ideally, BMI measures should have been undertaken at several time points around the age of 5. Because we have only measured BMI every 2 years (ages 4, 6, and 8), we are not in the best position to address predictors of change in BMI related specifically to the adiposity rebound. Future research should therefore aim to examine predictors of change in BMI during the adiposity rebound period measuring BMI at several and more frequent time points.

**Funding**

This research was supported by the Research Council of Norway (grant number 190622/V50, 185760/V50, 213793/H10).

**Conflicts of interest**: None declared.

**References**


