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The longitudinal relationship between sleep duration and body mass index in children: A growth mixture modeling approach

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The longitudinal relationship between sleep duration and body mass index in children: A growth mixture modeling approach

Abstract

OBJECTIVE: A growing number of studies indicate that shorter sleep durations could contribute to obesity in children. The objective of this article was to further examine the longitudinal relationship between sleep duration and body mass index (BMI) in children by using a growth mixture modeling approach. **METHOD:** This article used prospective data from the Longitudinal Study of Australian Children. Participants included 1079 children aged 4 to 5 years (2004) followed up until age 10 to 11 years (2010). Growth mixture modeling was performed to examine the longitudinal association between sleep duration and body mass index within distinct body mass index trajectories. **RESULTS:** The results indicated 3 distinct body mass index trajectories: healthy weight, early onset obesity, and later onset obesity. Longitudinal inverse associations were evident between sleep duration and body mass index in the Early Onset Trajectory. There were some associations between sleep duration and body mass index in the other trajectories. **CONCLUSIONS:** This article provides further insight into the longitudinal relationship between sleep duration and body mass index in children. In particular, the results indicate that shorter sleep durations are primarily associated with body mass index in children with early onset obesity.

Keywords

index, modeling, growth, approach, mass, body, children, duration, mixture, sleep, between, relationship, longitudinal

Disciplines

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Article Title: Identification of Distinct Body Mass Index Trajectories in Australian Children

Running Title: BMI trajectories in children

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What is already known about this subject

- Recent studies have identified distinct trajectories of obesity development in children, but more research is required to further explore these trajectories.
- Several socio-demographic variables such as parental education and obesity are associated with these trajectories.

What this study adds

- This study further demonstrates that there are distinct trajectories of body mass index in children.
- The use of raw body mass index values is more sensitive to changes in body composition compared to body mass index categories (e.g., lean versus overweight). Hence the present results provide a more detailed insight into development patterns of obesity.
- The socio-demographic predictors of the trajectories offer potential avenues for future obesity interventions.

ABSTRACT

Background: A limited number of studies have demonstrated that there may be distinct developmental trajectories of obesity during childhood.

Objectives: To identify distinct trajectories of body mass index (BMI) in a large sample of Australian children.

Methods: Participants included 4601 children aged 4 – 5 years at baseline, who were followed up at ages 6 – 7 years, 8 – 9 years, and 10 – 11 years. Height and weight were measured at each of these time-points, and used to calculate BMI. Growth Mixture Modelling was used to identify the presence of distinct BMI trajectories.

Results: Four distinct trajectories were identified: (1) High Risk Overweight; (2) Early Onset Overweight; (3) Later Onset Overweight; and, (4) Healthy Weight. Further analyses indicated that factors such as parental overweight, parent education, parent smoking, and child birth weight were significant predictors of these trajectories.

Conclusions: These findings indicate that different patterns of BMI development exist in children, which may require tailored interventions.

INTRODUCTION

Childhood obesity is a major public health issue, contributing to an elevated risk of health conditions such as diabetes and hyperlipidemia during childhood and in later life (1). Considerable cross-sectional and longitudinal research has examined population trends in child obesity and underlying risk factors. These findings have been used to inform the development of strategies and interventions to address the obesity epidemic at a population level. Although this research is important, very few studies have examined patterns of obesity at an individual level. This is needed because there may be different trajectories of obesity development in children that have unique risk factors and require tailored interventions.

Some recent studies have examined individual trajectories of obesity in children and adolescents using a variety of approaches. For instance, four studies have investigated trajectories of body mass index (BMI) categories (i.e., overweight versus healthy weight). Mustillo et al. (2) tracked 990 children from age 9 years to age 16 years, and identified four distinct trajectories of obesity: never obese; obese at all ages; initially obese, but healthy during adolescence; and, initially healthy but becoming obese. Li et al. (3) identified three distinct trajectories in 1739 children aged 2 years at baseline and followed for 10 years. These trajectories were labelled as early onset overweight (i.e., children were consistently overweight), late onset overweight (initially healthy, but overweight after age 8 years), and never overweight. Nonnemaker et al. (4) identified four patterns of obesity status in adolescents aged 12 – 17 years over a 6-year period: high risk of obesity; moderate-high risk of obesity; low-moderate risk of obesity; and, low risk of obesity. Using latent transition analysis, O'Brien et al. (5) found four main patterns of changing weight status between the

ages of 2 and 12 years: never overweight, overweight onset at preschool age; overweight onset in elementary school; overweight at preschool age but then normal weight.

Ventura et al. (6) and Pryor et al. (7) examined trajectories based on raw BMI values, which are more sensitive to changes in body composition relative to BMI categories. Ventura et al. (6) found four distinct BMI trajectories in girls aged 5 years over a 10 year period. When compared with standard growth curves, these trajectories were indicative of: consistent overweight; overweight at younger ages but becoming healthy; slight overweight; and healthy weight. Pryor et al. (7) noted three distinct trajectories, which were labelled as low stable BMI, moderate BMI, and high rising BMI. Huang et al. (8) and Smith et al. (9) also examined trajectories based on BMI z scores (these provide an indication of rank order change in BMI over time) and reported a similar pattern of results.

The aim of the present study was to further examine the presence of distinct developmental trajectories of raw BMI values in children aged 4 – 5 years at baseline and followed until age 10 – 11 years. We examined raw BMI values rather than BMI categories since the former are more sensitive to individual changes over time. BMI z scores were not examined because, although these are very useful for measuring obesity status cross-sectionally, there are some issues associated with their use in longitudinal studies (10, 11). In particular, the variability of BMI z scores depends on baseline adiposity status, such that variability in values is smaller for those children who are more overweight (11). Since this may influence the nature of the trajectories identified, we used raw BMI values which are considered to provide a better indication of longitudinal changes in children (10, 11).

Few studies have examined whether there are different trajectories in boys and girls, instead investigating trajectories in females only (6), or in boys and girls combined (2, 3, 5, 8, 9). The exception has been Pryor et al. (7) who investigated trajectories in boys and girls

separately but found no major gender differences in the trajectory patterns. It is important to further explore the possibility of distinct trajectories in boys and girls given the sex differences in body composition change during middle childhood (12, 13). Therefore, we investigated the possibility of different BMI trajectories in boys and girls. Finally, we explored whether socio-demographic factors assessed at baseline were significantly associated with these trajectories. Previous studies have found that factors such as parent education, obesity, and smoking, along with ethnicity are associated with obesity/BMI trajectories in children and adolescents (4, 6). Further understanding the socio-demographic profiles of the trajectories could provide an important insight into the children who are most at risk of obesity, and thus need to be targeted through obesity interventions.

METHODS

Participants

The Longitudinal Study of Australian Children (LSAC) is a national study examining the development of infants and children in Australia. This study commenced in 2004 and includes an infant cohort (aged 0 – 1 years at baseline), and a child cohort (aged 4 – 5 years at baseline). The LSAC was designed to be broadly representative of Australian infants and children in these age ranges. The sampling strategy involved randomly selecting participants from the Medicare Australia database, using geographic stratification. Medicare Australia is Australia's publicly funded healthcare system in Australia, and the database is the most comprehensive of the Australian population. The present research focused specifically on the child cohort, with data available when the children were aged 4 – 5 years (N = 4983), 6 – 7 years (N = 4464), 8 – 9 years (N = 4331), and 10 – 11 years old (N = 4169). This research

received ethical approval from the University of Wollongong's Human Research Ethics Committee.

Measures

Anthropometric Measures

At each time point, children had height and weight measured, and this information used to calculate BMI. Child height was measured by an Invicta stadiometer (Modern Teaching Aids, Australia) at age 4 - 5 years, 6 - 7 years, and 8 - 9 years, and by a laser stadiometer at age 10 - 11 years. In all data collection waves, two measurements were taken and a third was taken if there was more than 0.5cm difference between the first two measurements. Height was determined by the average of the two closest values. At ages 4 - 5 years, 6 - 7 years, and 8 - 9 years, body weight was measured using digital BMI bathroom scales (HoMedics, Australia), with Body Fat scales (Tanita, USA) used to measure body weight at age 10 - 11 years.

Demographic variables

Parents completed a questionnaire at baseline (i.e., when the children were aged 4 - 5 years old) assessing a number of socio-demographic factors. Place of residence was determined from the Accessibility/Remoteness Index of Australia (ARIA) (14), which includes five classes of remoteness: highly accessible (ARIA: ≥ 0 and < 0.2), accessible (ARIA: ≥ 0.2 and < 2.4), moderately accessible (ARIA: ≥ 2.4 and < 5.95), remote (ARIA: \geq

5.95 and < 10.5), and very remote (ARIA: ≥ 10.5). We combined the remote and very remote categories given their low cell sizes. Parents indicated their family type (dual parent versus single parent families), household income (low: <AUD \$1000/week; medium: AUD \$1000 - AUD \$1999/week; and higher income: \geq AUD \$2000/week), and whether their child was born in Australia or another country (country of birth).

Each parent completed two questions regarding the highest year of high school completed and the highest qualification attained. This information was used to categorise maternal and paternal education as < year 12, completed year 12, or completed a tertiary qualification such as a diploma, certificate, or university degree. Maternal and paternal obesity were derived from self-reported height and weight, which was used to categorise underweight (BMI < 18.5 kg/m²), lean (BMI 18.5 – 24.9 kg/m²), or overweight/obesity (BMI ≥ 25 kg/m²). Mothers and fathers were also asked to self-report whether they currently smoked, and whether they had consumed 5 or more standard drinks on more than two occasions in the past one month (a proxy of binge drinking). Mothers were also asked to indicate whether the child had ever been breastfed (including expressed breast milk), and if so the age at which the child stopped being breastfed completely. Responses were coded as never breastfed, breastfed for less than 6 months, and breastfed for 6 months or longer. Finally, the self-report questionnaire asked parents to indicate their child's birth weight, which was recoded into the following categories: low birth weight (< 2500 grams); 'normal' birth weight (2500 – 4000 grams); and high birth weight (> 4000 grams) (15).

Statistical Analysis

Growth mixture modelling (GMM) performed using *Mplus* version 6.11 (16) was used to identify BMI trajectories between ages 4 and 10 years, and associated socio-

demographic factors. GMM is a flexible modelling approach that identifies unobserved subpopulations with similar trajectories over time (17). In contrast to other approaches such as latent class growth analysis, GMM allows for variation in growth factors in each class (18, 19). Modelling within-class variation can pose some potential issues (e.g., errors in model specification) (20). However, we used this approach because it may provide a more realistic representation of complex data compared with other approaches that do not allow within-class variation (18).

Consistent with existing recommendations (17-19, 21) our analyses involved three main steps: (1) preliminary GMM (without covariates) to determine the number of trajectories; (2) identification of significant socio-demographic covariates; (3) the final GMM with significant covariates included. These steps were initially performed separately in boys and girls to explore the possibility of different trajectories by gender.

The first step in the analysis was to decide on the number of trajectories (i.e., latent classes). This was achieved by running a series of models specifying different numbers of latent classes (i.e., distinct trajectories) without the inclusion of covariates. For example, a GMM with one latent class was first tested followed by a model with two latent classes and so on. The optimal number of latent classes was determined on the basis of several sources of information. For instance, we used several criteria to assess model fit, such as Akaike's Information Criterion (AIC), Bayesian Information Criterion (BIC), and sample-size adjusted BIC. For these criteria, lower values for a given model (e.g., 2 classes) indicate an improved model fit relative to another model (e.g., 1 class). There are some problems with relying solely on these criteria, as they can overestimate the number of classes. Therefore, we also utilised bootstrap likelihood ratio tests (BLRT) to compare model fit between two subsequent models (19, 22). A significant BLRT result indicates that the model with k classes (e.g., 2 latent classes) provides an improvement in model fit compared to a model with $k - 1$ classes

(e.g., one latent class). Using BLRT, the number of classes is determined when the specification of one additional class does not produce a significant BLRT value. Finally, we also considered classification accuracy (e.g., appropriate accuracy determine by entropy levels greater than .80), and the size of the latent classes since small (e.g., < 1% of the sample) classes may not be meaningful. Therefore, the number of latent classes was informed by the information criteria, BLRT, entropy, and latent class size.

Once the optimal number of classes was identified, the second stage of the analysis involved examining the socio-demographic variables significantly associated with the BMI trajectories. This involved assigning participants to classes based on maximum posterior probability and conducting multinomial logistic regression to examine whether each of the following variables were significant covariates: family type, place of residence, household income, mother and father education, mother and father obesity, breastfeeding, birth weight, mother and father binge drinking, mother and father smoking, and child country of birth.

In the final step of the analyses, the full GMM model was tested. This involved specifying the number of classes from step 1 and including the significant covariates from step 2 as predictors of trajectory membership and the growth factors. This approach is consistent with the recommendations of Muthén (18, 19).

As is the case in most longitudinal studies, there were some missing data for BMI across the four time points. These data were assumed to be missing at random and were addressed using full information maximum likelihood (23). However, we excluded participants ($n = 357$, 7.2%) if they had more than two time points of missing data for BMI as this may have influenced the identification and pattern of the trajectories. For the logistic regression modelling, missing data on socio-demographic variables were dealt with by creating missing data categories where appropriate.

RESULTS

Descriptive Statistics

The initial sample included 4983 children (2520 boys and 2508 girls) aged 4 – 5 years at baseline. As noted earlier 357 children has missing data from two or more time points, and a further 25 had extreme BMI values (i.e., $> 40 \text{ kg/m}^2$). These individuals were excluded from the analysis, thus leaving a sample size of 4601. The characteristics of the sample are shown in Table 1, and are similar with the age matched Australian population data. For example, the proportion of boys to girls in this sample (51.3% versus 48.7%) is the same as the Australian population (24). Furthermore, 15.5% of children in the LSAC sample were from sole parent families, which is slightly lower than the age matched Australian population data of 17.9% (24). The prevalence of overweight/obesity in this sample is slightly lower but still comparable with population data for 5 – 9 year olds (19.7% versus 21.3%) (25). Finally, the proportion of children with a low birth weight in our sample was also similar with national data (i.e., 6.5% versus 6.2%) (25). These similarities suggests that the present sample is comparable on these variables with national data for children of this age range.

Determining the number of Latent Classes

Preliminary analyses indicated three distinct trajectories in boys and three distinct trajectories in girls, which were very similar in nature. This suggests that, consistent with Pryor et al. (7), BMI trajectories are similar in boys and girls. Therefore, we performed the analyses on boys and girls combined (with gender as a covariate) and report these results in the remainder of this paper.

The model fit statistics for the initial model for boys and girls combined (without covariates) are shown in Table 2. These indicate that the AIC, BIC, and adjusted BIC values were lower as the number of classes increased, indicating improved model fit. The BLRT results indicated that each sequential model had a significantly improved model fit relative to the preceding model. However, the specification of five latent classes led to the identification of a very small class ($n = 27$; $< 1\%$) which may not be meaningful. As a consequence, we concluded that the four-model solution provided the optimal model fit, with an appropriate level of entropy (0.88).

The multinomial logistic regression results indicated that mother and father overweight/obesity, mother and father education, mother and father smoking, and birth weight were significantly associated with the BMI trajectories. Hence, in the final model we included these variables assessed at baseline (i.e., age 4 – 5 years) to examine whether they predicted trajectory membership and the growth factors.

The four BMI trajectories based on the final model (including the covariates listed above) are shown in Figure 1. In order to facilitate interpretation of these results, International Obesity Task Force (26) cut-offs for overweight and obesity at the corresponding ages are included in Figure 1. Also shown in Figure 1 are the 95% confidence intervals for each trajectory, however these are small and not clearly visible in this Figure for the larger two latent classes.

The first trajectory ($n = 104$, 2.3%) was characterised by BMI values just below the obesity cut-off at age 4 – 5 years, indicating that the majority of children were overweight (26). This trajectory showed linear ($\beta = 4.16$, $p < .001$) increase in BMI from age 4 to age 10 years, over and above the obesity cut-off at subsequent ages. In addition, there was a significant quadratic trend indicating a slight reduction in the rate of growth over time ($\beta = -$

.38, $p = .003$). Despite this quadratic effect, the general trend indicates that children in this trajectory were at a high risk of obesity during childhood. Based on these characteristics, we labelled this trajectory High Risk.

Trajectory 2 ($n = 533$; 11.6%) had BMI values below the overweight cut off at age 4 – 5 years, but there was an increase in BMI values over time such that values were slightly higher than the overweight cut-off at age 6 – 7 years and approached the obesity cut-off at ages 8 – 9 years and 10 – 11 years ($\beta = .48$, $p = .020$). The significant quadratic effect ($\beta = .54$, $p < .001$) indicates that BMI increased at an increasing rate. Based on this pattern, we labelled this trajectory Later Onset.

Trajectory 3 ($n = 173$; 4.0%) had BMI levels indicative of obesity at age 4 – 5 years, and BMI levels increased linearly by age to be slightly below the obesity cut-off at age 10 – 11 years ($\beta = 1.81$, $p < .001$). Based on this pattern, we labelled this trajectory Early Onset.

Finally, trajectory 4 accounted for 82.4% ($n = 3791$) of the sample and was characterised by healthy BMI values at all ages. Both the linear ($\beta = -.61$, $p < .001$) and quadratic functions were significant ($\beta = .33$, $p < .001$), but the changes in BMI were substantially less pronounced compared to the other profiles. Given that the BMI values were in the healthy range (26), we labelled this trajectory Healthy.

The associations between each of the covariates and the trajectories from this final model are shown in Table 3. This indicates that mother overweight ($OR = 3.47$, $p < .001$) and mother smoking ($OR = 2.23$, $p < .001$) were significantly associated with the High Risk trajectory compared with the Healthy trajectory. In addition, high birth weight of children was significantly associated with the High Risk trajectory ($OR = 2.65$, $p < .001$). Mother overweight ($OR = 2.06$, $p < .001$) and mother smoking ($OR = 1.49$, $p < .001$) were also significantly associated with the Later Onset trajectory. In addition, lower education level

(i.e., < year 12) for mothers (OR = 1.53, $p < .001$) and fathers (OR = 1.71, $p < .001$) were significantly associated with the Later Onset trajectory. Finally, mother (OR = 2.87, $p < .001$) and father (OR = 2.87, $p < .001$) overweight were significantly associated with the Early Onset trajectory.

DISCUSSION

The present findings indicate distinct developmental trajectories of raw BMI values in a sample of Australian children, which is consistent with some recent studies. Our results indicated four distinct BMI trajectories: High Risk, Early Onset Overweight, Later Onset Overweight, and Healthy BMI. The patterns of these trajectories are very similar with the trajectories identified in previous research, suggesting there may be some commonalities in BMI/obesity trajectories in different samples of children. We also found that socio-demographic factors such as parent overweight/obesity, education, and smoking, and child birth weight were significantly related with these trajectories. This also supports previous studies in this area and provides some insight into these trajectories (6).

For example, the High Risk trajectory accounted for only a small portion of the present sample (2.3%), but is potentially very important given that it was characterised by BMI values indicative of obesity. It is possible that early life factors or genetic factors underlie this trajectory. For example, we found that these children were more likely to have a higher birth weight, which predicts future weight gain and obesity during later childhood (15). Mother overweight and smoking were also associated with this trajectory, and are established risk factors for child weight gain and obesity. Mother overweight/obesity could be linked with an increased risk of child obesity because of a shared genetic propensity for obesity (27, 28) or common environmental and behavioural factors (28). For example, some

overweight mothers may engage in unhealthy behaviours relating to poor diet and sedentary behaviour, which reinforces the development of similar behaviours in children (28). There is also evidence that overweight mothers are less likely to initiate and maintain breastfeeding (28), which may contribute to weight gain and obesity in children (29). Furthermore, maternal smoking, particularly prior to and during pregnancy, is associated with childhood obesity (30-32). The mechanisms by which maternal smoking is associated with childhood obesity are not clear. One possibility is that maternal smoking increases child exposure to chemicals such as nicotine, which may influence appetite (31, 32). Although we did not measure maternal smoking prior to or during pregnancy, it is likely that many mothers who were current smokers when the child was aged 4 – 5 years, had been previous smokers. It is also possible that maternal and paternal smoking reflect less healthy lifestyles and co-occur with reduced physical activity levels and poor diet. A combination of these and other factors may contribute to the obesity risk pattern observed in this trajectory.

Similar to the High Risk trajectory, the Early Onset trajectory had BMI levels that were indicative of obesity at age 4 – 5 years. This suggests that a combination of genetic and early life factors could contribute to obesity in these children. Our results suggest that children in this trajectory were significantly more likely to have an overweight mother or father, which as noted above could contribute to obesity in early childhood because of genetic or shared environmental/behavioural factors. However, the increase in BMI slowed in later childhood suggesting that these children were less likely to be obese at age 10 – 11 years, although they were still overweight. Future research is required to investigate whether this pattern of overweight persists into later childhood and adolescence, or whether some of these children become healthy.

The Later Onset trajectory had relatively healthy BMI at age 4 – 5 years, but had an upward trend in BMI from age 6 – 7 years above the overweight cut-off. Mother overweight

was significantly associated with this trajectory, consistent with the previous two trajectories. Furthermore, lower education levels (i.e., less than year 12) were also significantly associated with the Later Onset trajectory. This is important since lower education levels are linked with lower socio-economic levels of the family, which is also a risk factor for child obesity (33, 34). However, a variety of other mechanisms could underlie this trajectory. One possibility is that the increase in BMI from age 6 – 7 years reflects an increase in fat mass accumulation perhaps reflecting lifestyle changes associated with the start of school. Starting elementary school can be associated with changes in lifestyle and socialisation which contribute to weight gain and obesity (35). For example, some children may experience changes in diet (e.g., school canteen food, soft drinks) and reductions in physical activity which could contribute to weight gain (36, 37).

Finally, the Healthy trajectory was characterised by relatively stable height-to-weight during the six-year period at levels below the overweight cut-off. This trajectory accounted for the vast majority of the present sample (82.4%) indicating that most children had a low risk of obesity during the observed developmental period. It is possible that some of these children become overweight/obese at a later age during adolescence or adulthood, but we were unable to determine this in the present paper.

The present study therefore provides an indication of distinct trajectories of raw BMI values over a six-year developmental period and socio-demographic correlates of these trajectories. It is important to note that raw BMI values provide a useful indication of body composition, correlating well with measures of body fat. However, the relationship between BMI and fat mass can vary on an individual basis, and be influenced by factors such as age, gender, and ethnicity. This has important implications for interpreting the present results because the meaning of the changes in BMI could vary between the trajectories. For example, it is not clear whether the observed changes in BMI in the High Risk, Early Onset,

and Later Onset trajectories reflect changes in visceral or subcutaneous body fat or lean mass. Thus, the clinical implications of the BMI changes between trajectories could be different. Unfortunately, we were unable to determine this in our study, and suggest that future research examines trajectories using measures such as bioelectrical impedance that are able to differentiate lean mass from fat mass. Individual differences in puberty onset could also potentially explain these trajectories, but relevant variables were not available in the LSAC. Again, future research could benefit from inclusion of proxy measures of puberty.

The present study was also limited by the self-report nature of parental factors such as BMI, smoking, binge drinking, and household income. Inaccuracies and biases associated with the self-report nature of these data might have confounded the results. There are also other factors such as diet, physical activity levels, screen time, and sleep duration which may underlie the trajectories identified in this paper. Investigating these behaviours could provide an insight into the factors that influence the patterns of BMI change in these trajectories. For example, as noted above, factors such as physical activity and diet could potentially explain the increase in BMI at age 6 – 7 years in the Later Onset trajectory. Future research needs to investigate this further by modelling lifestyle behaviours as time varying covariates; that is, these variables need to be measured at multiple time points to examine their concomitant and lagged effects on BMI.

Despite these limitations, there are several strengths of this study. First, this study utilised a large representative sample of Australian children and thus provides relevant and important insights into developmental trajectories of BMI. The analytic approach is also a key strength of this study given that GMM is ideal for identifying developmental trajectories. The present findings therefore clearly demonstrate individual differences in BMI change in boys and girls during middle childhood. These findings have important implications, suggesting that interventions could be developed tailored specifically towards these at risk

trajectories. For example, targeting maternal factors such as weight gain and smoking during pregnancy and father obesity may be important in addressing early onset obesity. The Later Onset and High Risk trajectories may also require early interventions that target socio-demographic and parental factors, but may also need to target health behaviours and lifestyle. Future research examining other socio-demographic and behavioural factors associated with these trajectories is needed and may facilitate the treatment and prevention of obesity.

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REFERENCES

1. Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. *Pediatrics* 1998;**101**:518 - 525.
2. Mustillo S, Worthman C, Erkanli A, Keeler G, Angold A, Costello EJ. Obesity and Psychiatric Disorder: Developmental Trajectories. *Pediatrics* 2003;**111**:851 - 859.
3. Li C, Goran MI, Kaur H, Nollen N, Ahluwalia JS. Developmental Trajectories of Overweight During Childhood: Role of Early Life Factors. *Obesity* 2007;**15**:760 - 771.
4. Nonnemaker JM, Morgan-Lopez AA, Pais JM, Finkelstein EA. Youth BMI Trajectories: Evidence from the NLSY97. *Obesity* 2009;**17**:1274 - 1280.
5. O'Brien M, Nader PR, Houts RM, et al. The Ecology of Childhood Overweight: A 12-Year Longitudinal Analysis. *Int J Obesity* 2007;**31**:1469 - 1478.
6. Ventura AK, Loken E, Birch LL. Developmental trajectories of girls' BMI across childhood and adolescence. *Obesity* 2009;**17**:2067 - 2074.
7. Pryor LE, Tremblay R, Boivin M, et al.. Developmental Trajectories of Body Mass Index in Early Childhood and Their Risk Factors. *Arch Pediatr Adolesc Med* 2011;**165**:906 - 912.
8. Huang R-C, de Klerk NH, Smith A, et al. Lifecourse Childhood Adiposity Trajectories Associated With Adolescent Insulin Resistance. *Diabetes Care* 2011;**34**:1019 - 1025.
9. Smith AJ, O'Sullivan PB, Beales DJ, de Klerk N, Straker LM. Trajectories of childhood body mass index are associated with adolescent sagittal standing posture. *Int J Pediatr Obes*,2011;**6**:e97 - e106.

10. Berkley CS, Colditz GA. Adiposity in adolescents: change in actual BMI works better than change in BMI z score for longitudinal studies. *Ann Epidemiol* 2007;**17**:44 - 50.
11. Cole TJ, Faith MS, Pietrobelli A, Heo M. What is the best measure of adiposity change in growing children: BMI, BMI %, BMI z-score or BMI centile? *Eur J Clin Nutr* 2005;**59**:419 - 425.
12. Garnett SP, Högler W, Blades B, et al. Relation between hormones and body composition, including bone, in prepubertal children. *Am J Clin Nutr* 2004;**80**:966 - 972.
13. Veldhuis JD, Roemmich JN, Richmond EJ, et al. Endocrine control of body composition in infancy, childhood, and puberty. *Endocrine Reviews* 2005;**26**:114 - 146.
14. Australian Bureau of Statistics. *Information paper ABS Views on Remoteness 2001*. Canberra: Australian Bureau of Statistics; 2000.
15. Yu ZB, Han SP, Zhu GZ, et al. Birth weight and subsequent risk of obesity: a systematic review and meta-analysis. *Obesity Reviews* 2011;**12**:525- 542.
16. Muthén LK, Muthén BO. *Mplus Users Guide. Sixth Edition*. Los Angeles: CA: Muthén & Muthén; 1998 - 2010.
17. Wang M, Bodner TE. Growth Mixture Modeling: Identifying and Predicting Unobserved Subpopulations With Longitudinal Data. *Organ Res Methods* 2007;**10**:635 - 656.
18. Muthén B. The potential of growth mixture modelling. *Infant Child Dev* 2006;**15**:623 - 625.
19. Muthén BO. Latent variable analysis: Growth mixture modeling and related techniques for longitudinal data. In: Kaplan D (ed). *Handbook of quantitative methodology for the social sciences*. Newbury Park CA: Sage Publications; 2004. p. 345 - 68.

20. Bauer DJ, Curran PJ. The integration of continuous and discrete latent variable models: potential problems and promising opportunities. *Psychol Methods* 2004;**9**:3 - 29.
21. Jung T, Wickrama KAS. An Introduction to Latent Class Growth Analysis and Growth Mixture Modeling. *Social and Personality Psychology Compass* 2008;**2**:302 - 317.
22. Nylund KL, Asparouhov T, Muthén BO. Deciding on the number of classes in Latent Class Analysis and Growth Mixture Modeling: A Monte Carlo Simulation Study. *Struct Equ Modeling* 2007;**14**:535-569.
23. Enders CK, Bandalos DL. The relative performance of full information maximum likelihood estimation for missing data in structural equation models. *Struct Equ Modeling* 2001;**8**:430 - 457.
24. Australian Institute of Family Studies. *The Longitudinal Study of Australian Children 2004 Annual Report*. Melbourne: Australian Institute of Family Studies; 2005.
25. Australian Institute of Health and Welfare. Children's Headline Indicators. 2011; Accessed 23 July 2012 from: <http://www.aihw.gov.au/chi/index.cfm>
26. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *Br Med J* 2000;**320**:1240 - 1243.
27. Freeman E, Fletcher R, Collins CE, Morgan PJ, Burrows T, Callister R. Preventing and treating childhood obesity: time to target fathers. *Int J Obesity* 2012;**36**:12 - 15.
28. Oken E. Maternal and child obesity: the causal link. *Obstet Gynecol Clin North Am* 2009;**36**:361 - 377.
29. Buyken AE, Karaolis-Danckert N, Remer T, Bolzenius K, Landsberg B, Kroke A. Effects of Breastfeeding on Trajectories of Body Fat and BMI throughout Childhood. *Obesity* 2008;**16**:389 - 395.

30. Oken E, Levitan EB, Gillman M. Maternal smoking during pregnancy and child overweight: systematic review and meta-analysis. *Int J Obesity* 2008;**32**:201 - 210.
31. Reilly J, Armstrong J, Dorosty A, et al. Early life risk factors for obesity in childhood: Cohort study. *Br Med J* 2005;**330**:1357-1364.
32. Von Kries R, Toschke AM, LKoletzko B, Slikker W. Maternal Smoking during Pregnancy and Childhood Obesity. *Am J Epidemiol* 2002;**156**:954 - 961.
33. Monasta L, Batty G, Cattaneo A, Lutje V, Ronfani L, van Lenthe F, Brug J. Early-life determinants of overweight and obesity: A review of systematic reviews. *Obesity* 2010;**11**:695-708.
34. Greves Grow HM, Cook AJ, Arterburn DE, Saelens BE, Drewnowski A, Lozano P. Child obesity associated with social disadvantage of children's neighborhoods. *Soc Sci Med* 2010;**73**:584 - 591.
35. Yoshinaga M, Shimago A, Koriyama C, et al. Rapid increase in the prevalence of obesity in elementary school children. *Int J Obesity* 2004;**28**:494 - 499.
36. Blum JW, Jacobsen DJ, Donnelly JE. Beverage Consumption Patterns in Elementary School Aged Children across a Two-Year Period. *J Am Coll Nutr* 2005;**24**:93 - 98.
37. Fox MK, Dodd AH, Wilson A, Gleason PM. Association between School Food Environment and Practices and Body Mass Index of US Public School Children. *J Am Diet Assoc* 2009;**109**:S108 – S117.

Table 1. Baseline characteristics of the sample (i.e., at age 4 – 5 years). Data are presented as counts and percentages unless indicated otherwise.

	n	%
Body weight status		
Normal weight	3671	80.3
Overweight/Obese	900	19.7
Body Mass Index		
Raw value, mean (SD)	16.29 (1.60)	
Z score, mean (SD)	0.54 (0.98)	
Birth Weight		
Low birth weight	294	6.5
Healthy birth weight	3674	81.0
High birth weight	566	12.5
Area of Residence		
Highly Accessible	2469	54.2
Accessible	1098	24.1
Moderately Accessible	787	17.3
Rural/Remote	201	4.4
Mother weight status		
Underweight	380	10.4
Lean	935	25.5
Overweight/Obese	2351	64.1
Father weight status		
Underweight	70	2.4
Lean	906	30.6

Overweight/Obese	1989	69.4
Mother Education		
< year 12	925	20.2
Year 12	691	15.2
Other qualification	2943	64.6
Father Education		
< year 12	573	14.3
Year 12	372	9.3
Tertiary qualification	3028	75.5
Mother smoking status		
Non-smoker	3045	66.2
Current smoker	872	19.0
Father smoking status		
Non-smoker	2330	76.2
Current smoker	727	23.8

Table 2. Fit indices used to identify the optimal number of classes.

Number of latent classes	AIC	BIC	Adjusted BIC	BLRT	Entropy
1	63177.13	63241.47	63209.69	-	-
2	60934.16	61024.24	20979.75	-31578.57	.94
3	60484.84	60600.65	60543.45	-30453.08	.87
4	60221.46	60363.01	60293.00	-30224.42	.88
5 ^a	59960.45	60127.74	60045.12	-30088.73	.90

^a the 5-class solution led to the identification of a small latent class (N = 27, 0.6%).

AIC, Akaike's Information Criterion; BIC, Bayesian Information Criterion (BIC); BLRT, bootstrap likelihood ratio tests (BLRT)

Table 3. Covariates significantly associated with the BMI trajectories. Results are presented as odds ratios (with 95% CI), with the healthy trajectory as the reference category.

	High Risk		Later Onset		Early Onset	
	OR	95% CI	OR	95% CI	OR	95% CI
Sex						
Male	Ref		Ref		Ref	
Female	0.71	(0.36-1.40)	1.04	(0.76-1.43)	1.99	(0.96-4.14)
Mother BMI						
Underweight	1.50	(0.45-5.03)	1.06	(0.60-1.87)	0.40	(0.05-2.86)
Normal Weight	Ref		Ref		Ref	
Overweight	3.47*	(1.59-7.56)	2.06*	(1.47-2.90)	2.45*	(1.36-4.40)
Father BMI						
Underweight	1.62	(0.06-42.13)	1.56	(0.64-3.81)	0.69	(0.02-30.66)
Normal Weight	Ref		Ref		Ref	
Overweight	2.90	(0.86-9.80)	1.41	(0.94-2.10)	2.87*	(1.40-5.89)
Mother smoking status						
Non-smoker	Ref		Ref		Ref	
Current smoker	2.23*	(1.14-4.37)	1.49*	(1.07-2.07)	1.47	(0.83-2.62)
Father smoking status						
Non-smoker	Ref		Ref		Ref	
Current smoker	1.44	(0.65-3.23)	0.96	(0.63-1.45)	1.42	(0.63-3.21)
Mother Education						
< year 12	1.43	(0.77-2.67)	1.53*	(1.12-2.08)	1.36	(0.85-2.16)
Year 12	1.29	(0.61-2.70)	0.97	(0.61-1.36)	0.79	(0.41-1.55)
Tertiary qualification	Ref		Ref		Ref	
Father Education						
< year 12	1.04	(0.48-2.27)	1.71*	(1.17-2.50)	1.26	(0.72-2.22)
Year 12	0.84	(0.29-2.44)	1.26	(0.81-1.95)	1.03	(0.46-2.34)
Tertiary qualification	Ref		Ref		Ref	
Birth weight						
Low birth weight	0.51	(0.10-2.58)	0.70	(0.34-1.43)	1.50	(0.72-3.10)
Healthy birth weight	Ref		Ref		Ref	
High birth weight	2.65*	(1.42-4.95)	1.42	(0.98-2.07)	1.48	(0.78-2.80)

* $p < .05$; Ref, referent category

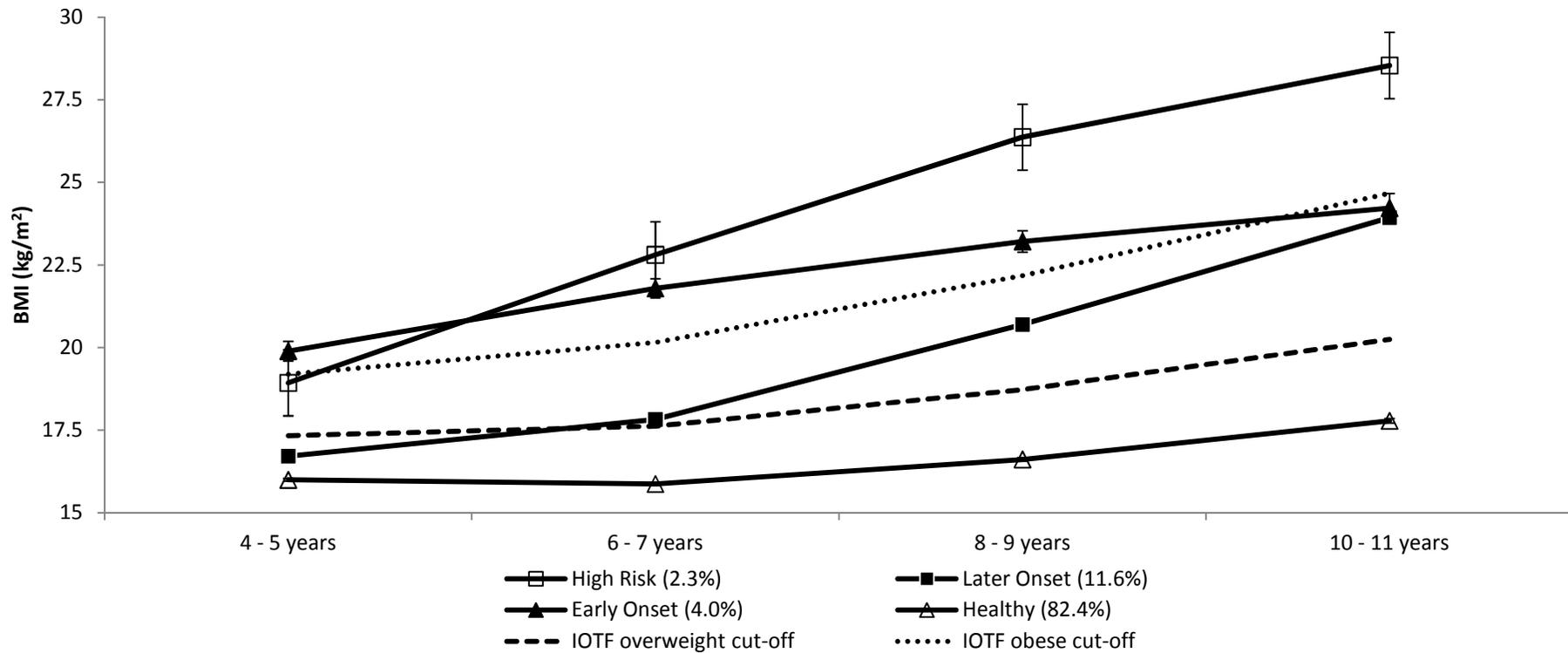


Figure 1. The raw BMI trajectories (with 95% confidence intervals) in children from age 4 – 5 years to 10 – 11 years, relative to the overweight and obese IOTF cut-offs. The 95% confidence intervals were small for the Later Onset and Healthy Trajectories, and hence are not clearly visible in this figure.