BMI Trajectories Associated With Resolution of Elevated Youth BMI and Incident Adult Obesity

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abstract

BACKGROUND AND OBJECTIVES: Youth with high BMI who become nonobese adults have the same cardiovascular risk factor burden as those who were never obese. However, the early-life BMI trajectories for overweight or obese youth who avoid becoming obese adults have not been described. We aimed to determine and compare the young-childhood BMI trajectories of participants according to their BMI status in youth and adulthood.

METHODS: Bayesian hierarchical piecewise regression modeling was used to analyze the BMI trajectories of 2717 young adults who had up to 8 measures of BMI from childhood (ages 3–18 years) to adulthood (ages 34–49 years).

RESULTS: Compared with those with persistently high BMI, those who resolved their high youth BMI by adulthood had lower average BMI at age 6 years and slower rates of BMI change from young childhood. In addition, their BMI levels started to plateau at 16 years old for females and 21 years old for males, whereas the BMI of those whose high BMI persisted did not stabilize until 25 years old for male subjects and 27 years for female subjects. Compared with those youth who were not overweight or obese and who remained nonobese in adulthood, those who developed obesity had a higher BMI rate of change from 6 years old, and their BMI continued to increase linearly until age 30 years.

CONCLUSIONS: Efforts to alter BMI trajectories for adult obesity should ideally commence before age 6 years. The natural resolution of high BMI starts in adolescence for males and early adulthood for females, suggesting a critical window for secondary prevention.

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Ms Buscot conceptualized and designed the study, analyzed the data, drafted the initial manuscript as well as all subsequent drafts, and reviewed and revised the manuscript; Drs Magnussen and Thomson designed the study, drafted the manuscript, and critically reviewed several drafts of the manuscript; Drs Viikari, Lehtimäki, Hutri-Kähönen, Juonala, Jokinen, Tossavainen, and Laitinen collected the clinical data and revised several drafts of the manuscript

WHAT'S KNOWN ON THIS SUBJECT: Because obesity is difficult to reverse, many studies highlight the importance of the early years in the development of lifelong BMI trajectories. However, the dynamics of the development, tracking, and potential resolution of childhood adiposity into adulthood are not completely understood.

WHAT THIS STUDY ADDS: In this cohort study, we investigated BMI trajectories from childhood to mid-adulthood. Results suggest that efforts to alter trajectories predicting adult obesity should commence before age 6 years, although a critical window for secondary prevention was identified in later childhood.

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Overweight and obesity in childhood and adolescence (herein termed youth) is associated with adverse cardiometabolic profiles that tend to persist into adulthood.[1–6] In particular, childhood BMI predicts adulthood obesity[7,8] and other long-term health outcomes, including cardiovascular disease (CVD) and type 2 diabetes mellitus.[6,9–16] Because obesity is difficult to reverse once established,[9,17–19] many studies highlight the importance of the formative years in the development of lifelong BMI trajectories.[20–23] Yet, the dynamic processes that drive the development of childhood overweight and obesity and the tracking or potential resolution of high BMI from childhood into adulthood are not completely understood.[9] Although some recent longitudinal studies have investigated BMI trajectories in infancy, early childhood,[9,12,24–26] and from childhood to adolescence or young adulthood,[23,27–30] few long-term observational studies have available data to examine BMI trajectories from early childhood to midadulthood. Because excess weight in adolescence is known to have a strong, adverse impact on multiple cardiovascular risk factors, the importance of primary prevention early in life has been advocated.[31,32] However, although observational and intervention studies have highlighted the potential benefits of improving BMI levels in obese children and adolescents,[33,34] little is known about the BMI pathways of those who manage to overcome obesity between youth and adulthood. Such data may identify critical windows for intervention.

A recent multicohort study highlighted the potential health impact of improving weight status across the life course by reporting that overweight or obese youth who were not obese in adulthood had similar risks for type 2 diabetes mellitus, dyslipidemia, preclinical atherosclerosis, and hypertension compared with those who were never obese.[13] However, a lack of complete serial data in some of these cohorts precluded the accurate description and analysis of the early-life BMI trajectories of participants who resolved their overweight or obesity by adulthood and of those who developed incident obesity from a normal BMI. Moreover, data from only 2 time points did not provide information as to when in the period between youth and midadulthood that the change in BMI status occurred.[13]

Therefore, we analyzed BMI data across the life course in the Cardiovascular Risk in Young Finns Study (YFS)[35] using Bayesian hierarchical piecewise regression.[36] Our primary aim was to model individual trajectories of BMI from youth to adulthood and investigate differences in trajectories across 4 a priori–defined groups based on BMI status (assessed once in youth and once in adulthood).[13,37]

METHODS

Study Sample and Design

The YFS is a population-based prospective cohort that commenced in Finland in 1980 to investigate cardiovascular risk factors and their determinants from youth to adulthood.[35] The YFS participants are all of white, Northern European ancestry. The current analyses focus on serial BMI measurements taken on up to 8 occasions over 31 years (in 1980, 1983, 1986, 1989, 1992, 2001, 2007, and 2011). We included 2717 YFS participants (1252 male, 1465 female) from 6 different birth cohorts (1962, 1965, 1968, 1971, 1974, and 1977) who had a youth BMI measure at baseline (in 1980, age 3–18 years) and at least 1 adult BMI measure taken 21, 27, or 31 years later (in 2001, 2007, or 2011) (Supplemental Table 5). For the calculations of BMI, standing height was measured to the nearest 0.5 cm by using a wall-mounted seca anthropometer with the participant in bare feet. Weight was measured with participants in light clothes without shoes by using a digital seca weighing scale that recorded to the nearest 0.1 kg. On average, there were 5.25 nonmissing BMI measures per participant (ranging from 3 to 8 individual measures). Participants or their parents provided informed consent, and the study was approved by the local ethics committees in accordance with the Declaration of Helsinki.

Definition of BMI Status Groups

In keeping with previous studies,[13] youth overweight and obesity were defined by using international age- and sex-specific BMI cutoffs for youth aged 6 to 18 years[38]; and adult weight status was defined as overweight for a BMI ≥25 and as obese for a BMI ≥30 for participants aged 21 years and older. On the basis of their movement from 1 weight status category to another between the baseline examination (in 1980) and their most recent follow-up in adulthood, each participant was assigned to 1 of 4 possible BMI status groups (Table 1): Group I had normal BMI in youth and were nonobese in adulthood (reference group), Group II were overweight or obese as youth but nonobese as adults (high-BMI resolving group), Group III were overweight or obese as youth and obese in adulthood (high-BMI persisting group), and Group IV had normal BMI in youth but became obese as adults (incident obese group).

Statistical Analyses

Individual BMI trajectories between ages 6 and 49 years were modeled by using a hierarchical piecewise growth modeling approach with a linear-linear functional form and random change points (CPs). One of the advantages of this approach is that the piecewise specification of the

within-person (Level 1) model maps onto known, key aspects of the BMI development between 6 and 49 years old. Indeed, it can be modeled as a 2-phased process by using 2 distinct linear segments joining at a CP that represents the age at which individuals transition to a slower BMI growth rate (which usually happens at the end of puberty for each sex).[39,40] The Bayesian implementation of this model allows the estimation of between-person heterogeneity (ie, random effects) in all 4 parameters describing individual BMI change across the life course: the baseline BMI level, the yearly rate of change in BMI in youth (youth slope [S_1]), the age at the CP, and the yearly rate of change in BMI in adulthood (adult slope [S_2]) (see Supplemental Methods 1).

This allows for descriptions of group-specific prototypical growth curves showing how the BMI developed on average in each group of interest. Technical aspects of this modeling approach, including detailed model parameterization and Bayesian implementation, are described extensively.[35] Specifically, in the current application, parameters describing sex- and obesity-specific, average BMI trajectories are retrieved by sampling the posterior distribution of the 4 growth parameters, and the between-group differences in these parameters were investigated by using the posterior distributions for the credible differences between the curve parameters for the different groups.[41] Because the YFS participants were born up to 15 years apart, we simultaneously adjusted for any potential intercohort variability in BMI development between ages 6 and 49 years by including the initial age (ie, a participant's age in 1980) as a continuous predictor of each growth parameter.[42] The Bayesian hierarchical piecewise growth curve model analyses were estimated via Markov chain Monte Carlo sampling by using the Rjags and R2jags statistical packages in R.[43] The Bayesian model code used for model fitting in this article, including likelihood formulation and previous specification, is provided in Supplemental Methods 1.

TABLE 1 Summary of the 4 BMI Status Groups Considered Throughout This Study

Childhood BMI Status[a]	Adulthood BMI Status[b]	Group	BMI Status Group
Normal wt	Nonobese (normal or overweight)	1	Reference group
Normal wt	Obese	4	Incident obese
Overweight or obese	Nonobese (normal or overweight)	2	High-BMI resolving
Overweight or obese	Obese	3	High-BMI persisting

[a] Childhood BMI status was assessed at baseline in 1980 for each included participant. The age range at baseline is 6–18 years.
[b] Adulthood BMI status was assessed at the latest follow-up for each participant up to 31 years later. The age range at adult BMI assessment is 34–49 years.

TABLE 2 Number of Participants (%) in Each Obesity Status Group Stratified by Sex

Sex	Group I (Reference), *n* (%)	Group II (High-BMI Resolving), *n* (%)	Group III (High-BMI Persisting), *n* (%)	Group IV (Incident Obese), *n* (%)	Total, *n*
Male	961 (76.7)	35 (2.8)	73 (5.8)	183 (14.7)	1252
Female	1124 (76.7)	58 (3.9)	62 (4.2)	221 (15.1)	1465
Total	2085 (76.7)	93 (3.4)	135 (4.9)	404 (14.9)	2717

RESULTS

Study Cohort

BMI records (N = 15 748) from 2717 participants (n = 1252; 46% male) from up to 8 time points were used in the analyses. Compared with the YFS subjects included in the multicenter study,[13] there were an additional 77 YFS participants (n = 37; 48% male) in the current analysis, with data from follow-up in 2011. The number and relative proportion of participants in each of the 4 adiposity status groups are reported in Table 2. The proportion of youth overweight or obesity was 8.4% at baseline and higher among male subjects on average (9.1% vs 6.7%; Supplemental Table 9). Of participants, 19.8% were obese as adults at their latest assessment.

Participants were on average 24.5 years old (there were no sex differences; independent Welsh corrected t test, t = −1.5; df = 13 835; P = .12; Supplemental Table 10). However, differences in age between the 4 obesity groups were significant (all P < .001), with participants who resolved their high BMI in youth (Group II) being slightly younger than participants who became obese (Group IV) and those who remained nonobese (Group I; Supplemental Table 10). These differences were small, however, suggesting adequate representation of all birth cohorts in all adiposity subgroups.

BMI Trajectories in the 4 BMI Groups

Data exploration suggested that there was significant variability by sex in the BMI trajectories among the 4 groups of interest (see Supplemental Methods 1 for more details). Therefore, models were adjusted to give sex-specific BMI trajectory parameters within each of the 4 groups. The first 2 columns of Tables 3 and 4 show the estimated mean growth parameters for each sex and obesity group and describe the average prototypical BMI trajectories between 6 and 49 years of age represented in Figs 1 and 2. For each sex, the differences in trajectory parameters between the reference and incident obese participants (Group I versus Group IV, Table 3) and between resolving and persistent obese participants (Group II versus Group III; Table 4)

are all significant (see Supplemental Tables 8 and 10 for 95% credible intervals [CIs]), suggesting these groups are different in all aspects of their BMI development (initial levels at age 6 years, childhood growth rate, age at transition, and adult growth rate). The significance and magnitude of sex differences in the means of the 4 growth parameters for each BMI group are shown in the Supplemental Information (see also Supplemental Table 8).

Compared with those who remained nonobese as adults (Group I), participants who developed obesity in adulthood (Group IV) had a 0.8-higher average BMI at age 6 years (SD = 0.3) for male subjects and for female subjects (SD = 0.4) (Fig 1). Similarly, among overweight or obese youth and compared with those who remained obese in adulthood, those who were not obese in adulthood (Group II) had average initial BMI levels that were 2.0 lower for male subjects (SD = 0.6) and 3.4 lower for female subjects (SD = 0.8). Those who had high BMI in youth but avoided becoming obese as adults (Group II) transitioned to an almost-null BMI change rate in adulthood earlier than those who had persistent obesity (Group III; 3.6 and 10.1 years sooner for male and female subjects, respectively; Fig 2 and Table 4), leading to a leveling off in their BMI trajectory. It appears that participants who improved their high youth BMI (Group II) reached this transition in their BMI trajectories sooner than those who were of normal weight in youth and were nonobese in adulthood. That is, they reached their CP at 16.1 (SD = 1.4) years old for female subjects and 21.4 (SD = 1.7) years old for male subjects versus 17.2 years old (SD = 0.3) and 24.1 years old (SD = 0.4) in Group I (Tables 3 and 4). However, although they became nonobese, they remained of average overweight or were borderline overweight in adulthood (ie, average BMI levels in Group II were close to or >25 from ~35 years of age, especially for male participants [Fig 2]). In contrast, male and female subjects in the incident obese group (Group IV) and the persisting obese group (Group III) reached their adult BMI rate at the same age (~30 and ~25 years, respectively; Tables 3 and 4).

Compared with the reference group (Group I), participants who were of normal weight in youth but became obese as adults (Group IV) had significantly greater BMI rates in youth and in adulthood past their CPs (ie, steeper slopes [S_1 and S_2]; Table 3). Compared with the reference group (Group I), the average youth BMI rate of incident obese participants increased by 17%

TABLE 3 Estimated Sex-Specific Differences in BMI Trajectory Parameters (Marginal Posterior Means and PSDs) Between Participants Who Remained Nonobese From Youth to Adulthood (Group I) and Participants Who Become Obese by Adulthood (Group IV)

BMI Trajectory Parameter	Unit	Group I (Reference)	Group IV (Incident Obesity)	Group Differences (I and IV)
Male subjects				
Intercept	kg/m^2	23.9 (0.09)	28.6 (0.24)	−0.83 (0.25)
S_1	kg/m^2 per y	0.46 (0.008)	0.63 (0.01)	−0.17 (0.03)
S_2	kg/m^2 per y	−0.40 (0.007)	−0.55 (0.007)	0.15 (0.05)
Age at transition CP	y	24.1 (0.41)	30.1 (0.8)	−5.89 (1.03)
Female subjects				
Intercept	kg/m^2	24.21 (0.11)	27.62 (0.33)	−0.84 (0.36)
S_1	kg/m^2 per y	0.48 (0.008)	0.62 (0.01)	−0.14 (0.04)
S_2	kg/m^2 per y	−0.39 (0.009)	−0.47 (0.009)	0.08 (0.04)
Age at transition CP	y	17.2 (0.34)	29.7 (0.41)	−12.06 (2.1)

Shown in the table are the following: (1) The 4 BMI trajectory parameters (eg, intercept, which corresponds to the expected BMI levels at 25 years [the age variable is centered around 25 years in the model] provided they are in the first phase of growth BMI [predicted BMI at age 6 years can thus be calculated as $BMI_{age6} = Intercept + S1 * (25 - 6)$]); and S_2 corresponds to the deviation between the S_1 and S_2 so that the BMI rate in adulthood can be calculated as $S_1 + S_2$ and the age at CP for each sex in each obesity group. (2) The differences in BMI trajectory parameters between persistent nonobese and incident obese participants (Group I versus Group IV) for male subjects and female subjects; and all reported estimates are significant (for simplicity, the table does not report the density distribution [95% CI: 25th–95th percentiles] of the parameters and the difference in parameters. All 95% CIs can be found in Supplemental Tables 8 and 9 and in the Supplemental Information.

TABLE 4 Estimated Sex-Specific Differences in BMI Trajectory Parameters (Marginal Posterior Means and PSDs) Between High-BMI Resolving Participants (Group II) and High-BMI Persisting Participants (Group III)

BMI Trajectory Parameter	Unit	Group II (High-BMI Resolving)	Group III (High-BMI Persisting)	Group Differences (II and III)
Male subjects				
Intercept	kg/m^2	28.64 (0.6)	30.51 (1.12)	−2.01 (0.63)
S_1	kg/m^2 per y	0.48 (0.04)	0.55 (0.06)	−0.06 (0.02)
S_2	kg/m^2 per y	−0.44 (0.05)	−0.33 (0.01)	−0.11 (0.04)
Age at transition CP	y	21.4 (1.74)	24.7 (3.36)	−3.6 (0.72)
Female subjects				
Intercept	kg/m^2	28.02 (0.75)	31.4 (1.2)	−3.4 (0.78)
S_1	kg/m^2 per y	0.46 (0.04)	0.56 (0.06)	−0.09 (0.03)
S_2	kg/m^2 per y	−0.39 (0.03)	−0.25 (0.01)	−0.14 (0.03)
Age at transition CP	y	16.1 (1.4)	27.1 (2.58)	−10.1 (0.7)

Shown in the table are the following: (1) The 4 BMI trajectory parameters (eg, intercept, which corresponds to the expected BMI levels at 25 years [the age variable is centered around 25 years in the model] provided they are in the first phase of growth BMI [predicted BMI at age 6 years can thus be calculated as $BMI_{age6} = Intercept + S1 * (25 - 6)$]); and S_2 corresponds to the deviation between the S_1 and S_2 so that the BMI rate in adulthood can be calculated as $S_1 + S_2$ and the age at CP for each sex in each obesity group. (2) The differences in BMI trajectory parameters between those who resolved high-BMI status and those who persisted with high-BMI status (Group 2 versus Group 3) for male subjects and female subjects; and all reported estimates are significant (for simplicity, the table does not report the density distribution [95% CI: 25th–95th percentiles] of the parameters and difference in parameters. All 95% CIs can be found in Supplemental Tables 8 and 9 and in the Supplemental Information.

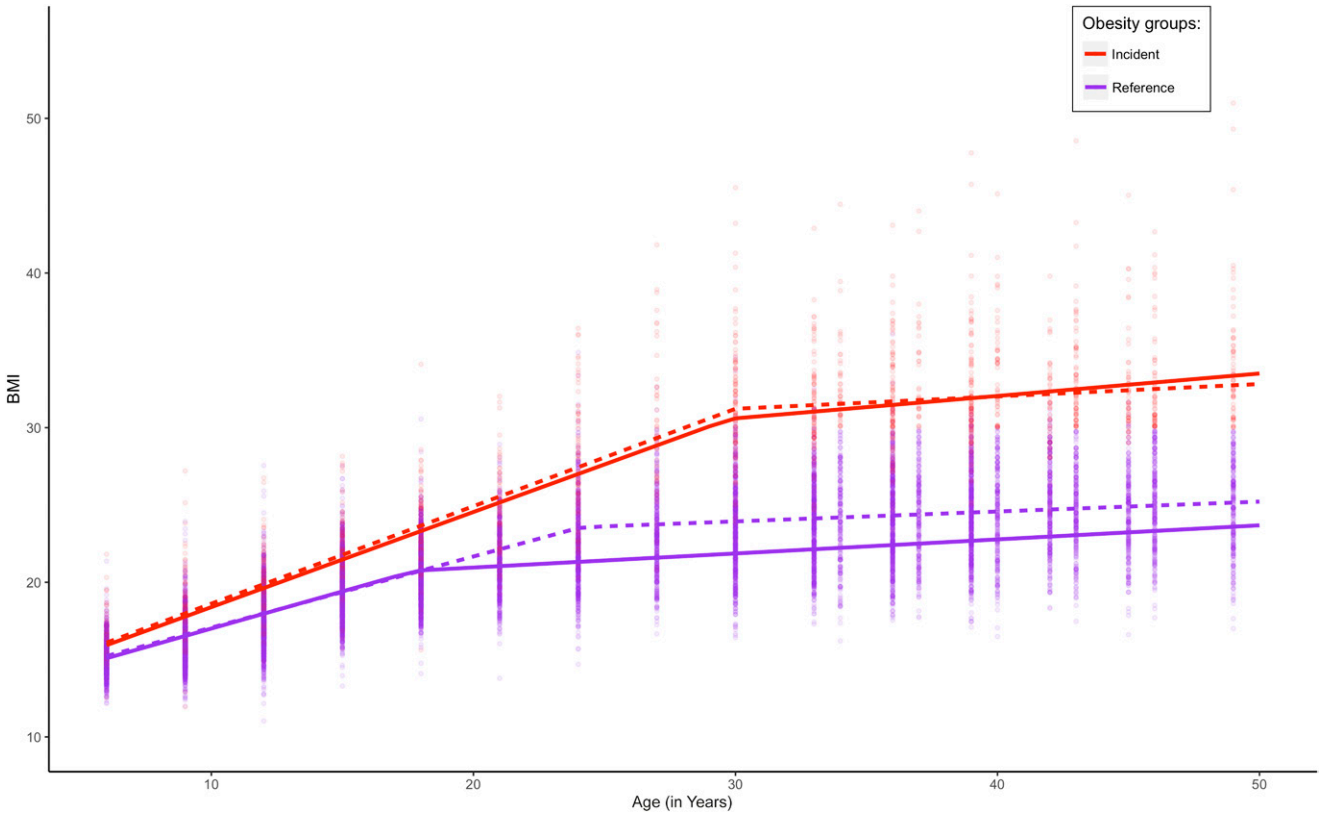

FIGURE 1

Average age-related BMI trajectories in the reference group (Group I; purple lines) and the group that became incident obese in adulthood (Group IV; red lines), estimated from the sex- and group-specific Bayesian hierarchical piecewise regression growth model. Solid lines indicate the average prototypical trajectories for female subjects, and dashed lines indicate the estimated trajectories for male subjects.

in male subjects and 14% in female subjects, and they remained on this rapidly increasing, linear trajectory until ~30 years old. In this group, the female subjects who became obese had higher BMI levels than male subjects by midadulthood because they had significantly steeper adult BMI slopes (S_2) compared with male subjects (Fig 1 and Table 3).

The between-subject variability in the trajectory parameters (especially the age at transition between the youth and adult BMI rate) not accounted for by belonging to 1 of the 8 a priori–defined, sex-by-BMI groups is relatively large ($\sigma^2_{CP} = 25$, Supplemental Table 13). This suggests underlying variability in the sample in different aspects of the BMI change over time within each considered group, particularly in the age at which participants transition to their adult BMI growth rates.

When adjusting for birth cohort in the trajectory model, we did not detect any systematic variation in the BMI trajectory parameters as a function of birth year (Supplemental Table 14), suggesting that the 6 birth cohorts in the YFS sample are relatively homogenous in their developmental trajectories of BMI.

DISCUSSION

Building on findings from a 4-cohort study in which researchers showed that normalizing high BMI by adulthood was associated with a reduction in cardiovascular risk, we took advantage of the 31 years of follow-up in the YFS cohort to characterize (for the first time) the change in BMI from age 6 to 49 years in different BMI groups and examine the timing of normalization or the incidence of elevated BMI in the YFS cohort. To better understand the BMI development in those who normalized their CVD risk by overcoming elevated BMI levels in youth and in those who increased their CVD risk by becoming obese adults, we used Bayesian hierarchical piecewise regression, an advanced multilevel growth curve modeling approach that allows us to estimate biologically meaningful and interpretable growth coefficients as well as between-person variability in key aspects of the BMI development across the life course.[36]

Compared with the reference group, which included those who were not obese in youth or adulthood (Group I), the BMI of normal weight youth who developed obesity in adulthood was higher from age 6 years and increased significantly faster from youth until young adulthood (~30 years), confirming that the roots

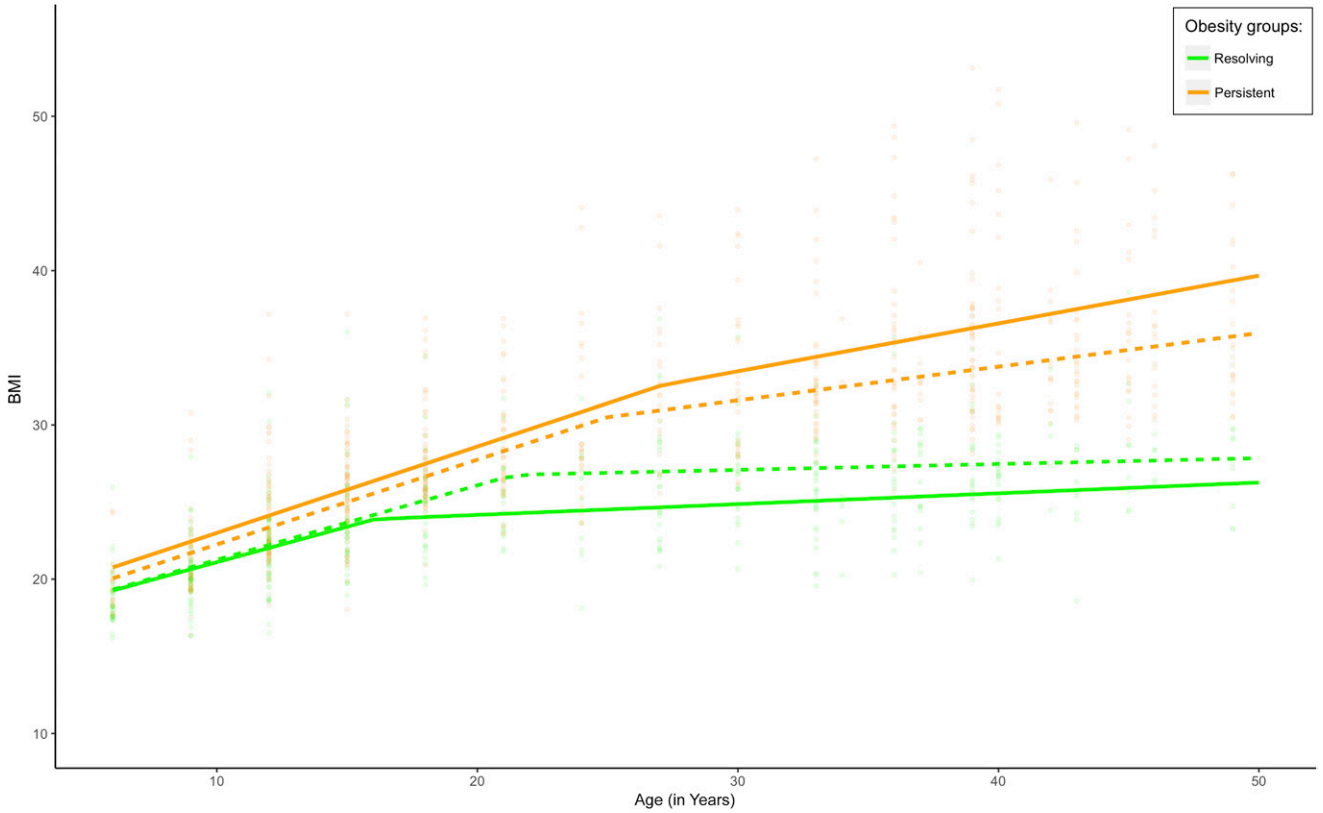

FIGURE 2

Average age-related BMI trajectories in the high-BMI resolving group (Group II; green lines) and in the high-BMI persisting group (Group III; yellow lines), estimated from the sex- and group-specific Bayesian hierarchical piecewise regression growth model. Solid lines indicate the average prototypical trajectories for female subjects, and dashed lines indicate the estimated trajectories for male subjects.

of incident adult obesity lie in BMI trajectories from early youth (beginning before age 6 years) and that maintaining normal BMI rates in youth is crucial to preventing later overweight and obesity.[32] These findings are consistent with previous studies that reported that the majority of excess body weight in children who develop obesity was often gained before age 5 years,[9,44–47] and these individuals had an increased weight-for-height velocity after age 3 years that resulted in a crossing of higher BMI percentiles.[45,47–49]

The group of participants found to have reversed their adult CVD risk in the previous study,[13] which included those who overcame high youth BMI by adulthood (Group II), had lower average BMI levels from age 6 years and slower yearly increases in BMI in youth compared with participants who remained obese as adults (Group III). These findings are in line with those of other studies, which advise that efforts to reverse adverse BMI trajectories should ideally begin in early life, which is in line with current international pediatric guidelines and recommendations for the evaluation, prevention, and management of obesity.[50–54] However, the transitioning to an almost-null BMI growth rate for these participants at ∼16 years old for female subjects and 21 years for male subjects suggests they corrected their adverse weight status by plateauing in their BMI trajectories before the end of adolescence, the period in which the transition to a slower increasing adult BMI rate naturally occurs in normal weight participants. In contrast, the BMI of those who remained obese into adulthood (Group III) continued to increase linearly at the same rate until they were ∼25 years old. This is the first study to describe the natural resolution of high youth BMI, and our findings suggest that puberty is a sensitive window for the secondary prevention of obesity. Secondary interventions targeting the control of obesity progression in overweight older youth (ie, slowing down the increase in BMI yearly rates to stabilize weight[50]) may contribute to reducing the incidence of adult obesity.

Additionally, we found that participants with persistently high BMI (Group III) reached higher asymptotic levels of BMI compared with incident obese participants (Group IV). This is consistent with the established tracking of adverse BMI from youth to adulthood; unresolved obesity generally gets worse with age.[1,2,4,55]

The posterior standard deviations (PSDs) reported along with the marginal posterior means of each growth parameter in Tables 3 and 4 quantify the uncertainty around the point estimate for each parameter (similar to an SD around a regression coefficient in the classic statistical framework). Model validation plots suggest that the estimated trajectory parameters provided an adequate representation of the BMI age curve for all considered groups from age 6 to 49 years despite a slightly larger PSD in the growth parameters estimated for Groups II and III compared with those obtained for the reference group (Group I) and the incident obese group (Group IV). Because all 95% CIs remained significant (Tables 3 and 4 and Supplemental Table 11), we are confident that the relatively small sample size in some of the sex-by-BMI status groups did not overly affect the precision of growth parameter estimates.

Enhanced knowledge of the dynamics of BMI change across the life course, especially the development of pathologic versus normal age-related BMI trajectories, may help guide clinical and public health practice by suggesting critical and sensitive windows for intervention targeted at reducing the incidence of adult obesity with the aim of halting the progression of existing obesity.[26,56,57]

This is the first study in which researchers describe BMI patterns from early childhood to midadulthood in 4 groups of clinical interest. A better understanding of the trajectories of those who develop obesity or those who had high BMI in childhood but did not become obese in adulthood is particularly important from a public health perspective. This study has some limitations. Despite a high prevalence of adult overweight in our sample, similar to the previous multicenter study, we collapsed overweight and normal weight adults into a single nonobese adult status to optimize the number of participants per category.[13] This classification of study participants into 4 groups prevented the discrimination of CVD risk between incident overweight and truly normative BMI trajectories. As a result, the average age-BMI curve we estimated for the reference group (Group I) in this study reached borderline overweight BMI levels (BMI $\geq$25) in midadulthood along with relatively large estimates of the within-group residual error. It has been noted that analyses of different combinations of BMI groups (for example, obese versus nonobese) characterized over the life course can result in a loss of information because of discrete categorization,[58,59] and using dichotomous measures of obesity and overweight can create misclassification bias, especially at young ages.[9] To overcome these potential issues, future researchers should thus consider alternative approaches to identify clusters of participants who share similar BMI profiles across all existing measures rather than at 2 individual assessments. These may provide additional insight into the association between life course adiposity trajectories and later CVD risk factors.[40,60]

CONCLUSIONS

Our study takes advantage of 31 years of follow-up data in the YFS and provides insights into the timing of potential BMI differences in young childhood between those who do or do not maintain high BMI from youth to midadulthood. Our findings suggest that efforts to influence BMI trajectories that lead to adult obesity should begin early in life, ideally before age 6 years. In addition, the natural resolution of high youth BMI levels begins in adolescence for male subjects and early adulthood for female subjects in this cohort, suggesting a critical window for secondary prevention before these stages of the life course.

ABBREVIATIONS

CI: credible interval
CP: change point
CVD: cardiovascular disease
PSD: posterior standard deviation
S_1: youth slope
S_2: adult slope
YFS: Young Finns Study

for critical intellectual content; Dr Raitakari participated in designing the study, collected the clinical data, and revised several drafts of the manuscript for critical intellectual content; Drs Sabin and Burgner revised several drafts of the manuscript for critical intellectual content; and all authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

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REFERENCES

1. Evensen E, Wilsgaard T, Furberg A-S, Skeie G. Tracking of overweight and obesity from early childhood to adolescence in a population-based cohort - the Tromsø Study, Fit Futures. *BMC Pediatr*. 2016;16(1):64
2. Clarke WR, Lauer RM. Does childhood obesity track into adulthood? *Crit Rev Food Sci Nutr*. 1993;33(4–5):423–430
3. Herman KM, Craig CL, Gauvin L, Katzmarzyk PT. Tracking of obesity and physical activity from childhood to adulthood: the Physical Activity Longitudinal Study. *Int J Pediatr Obes*. 2009;4(4):281–288
4. Deshmukh-Taskar P, Nicklas TA, Morales M, Yang SJ, Zakeri I, Berenson GS. Tracking of overweight status from childhood to young adulthood: the Bogalusa Heart Study. *Eur J Clin Nutr*. 2006;60(1):48–57
5. Lakshman R, Elks CE, Ong KK. Childhood obesity. *Circulation*. 2012;126(14):1770–1779
6. Freedman DS, Mei Z, Srinivasan SR, Berenson GS, Dietz WH. Cardiovascular risk factors and excess adiposity among overweight children and adolescents: the Bogalusa Heart Study. *J Pediatr*. 2007;150(1):12.e2–17.e2
7. Kvaavik E, Tell GS, Klepp KI. Predictors and tracking of body mass index from adolescence into adulthood: follow-up of 18 to 20 years in the Oslo Youth Study. *Arch Pediatr Adolesc Med*. 2003;157(12):1212–1218
8. Guo SS, Wu W, Chumlea WC, Roche AF. Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence. *Am J Clin Nutr*. 2002;76(3):653–658
9. Stuart B, Panico L. Early-childhood BMI trajectories: evidence from a prospective, nationally representative British cohort study. *Nutr Diabetes*. 2016;6(6):e198
10. Engeland A, Bjørge T, Søgaard AJ, Tverdal A. Body mass index in adolescence in relation to total mortality: 32-year follow-up of 227, 000 Norwegian boys and girls. *Am J Epidemiol*. 2003;157(6):517–523
11. Freedman DS, Khan LK, Dietz WH, Srinivasan SR, Berenson GS. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa Heart Study. *Pediatrics*. 2001;108(3):712–718
12. Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. The relation of childhood BMI to adult adiposity: the Bogalusa Heart Study. *Pediatrics*. 2005;115(1):22–27
13. Juonala M, Magnussen CG, Berenson GS, et al. Childhood adiposity, adult adiposity, and cardiovascular risk factors. *N Engl J Med*. 2011;365(20):1876–1885
14. Ludwig DS. Childhood obesity–the shape of things to come. *N Engl J Med*. 2007;357(23):2325–2327
15. Mattsson N, Rönnemaa T, Juonala M, Viikari JS, Raitakari OT. Childhood predictors of the metabolic syndrome in adulthood. The cardiovascular risk in Young Finns Study. *Ann Med*. 2008;40(7):542–552
16. Barton M. Childhood obesity: a life-long health risk. *Acta Pharmacol Sin*. 2012;33(2):189–193
17. Bumaschny VF, Yamashita M, Casas-Cordero R, et al. Obesity-programmed mice are rescued by early genetic intervention. *J Clin Invest*. 2012;122(11):4203–4212
18. Wen LM, Rissel C, He G. The effect of early life factors and early interventions on childhood overweight and obesity. *J Obes*. 2015;2015:964540
19. Spruijt-Metz D. Etiology, treatment and prevention of obesity in childhood and adolescence: a decade in review. *J Res Adolesc*. 2011;21(1):129–152
20. Giles LC, Whitrow MJ, Davies MJ, Davies CE, Rumbold AR, Moore VM. Growth trajectories in early childhood, their relationship with antenatal and postnatal factors, and development of obesity by age 9 years: results from an Australian birth cohort study. *Int J Obes (Lond)*. 2015;39(7):1049–1056
21. Monteiro POA, Victora CG. Rapid growth in infancy and childhood and obesity in later life–a systematic review. *Obes Rev*. 2005;6(2):143–154
22. Botton J, Heude B, Maccario J, Ducimetière P, Charles MA; FLVS Study Group. Postnatal weight and height growth velocities at different ages between birth and 5 y and body composition in adolescent boys and girls. *Am J Clin Nutr*. 2008;87(6):1760–1768
23. Tu AW, Mâsse LC, Lear SA, Gotay CC, Richardson CG. Body mass index trajectories from ages 1 to 20: results from two nationally representative Canadian longitudinal cohorts. *Obesity (Silver Spring)*. 2015;23(8):1703–1711
24. Pryor LE, Tremblay RE, Boivin M, et al. Developmental trajectories of body mass index in early childhood and their risk factors: an 8-year longitudinal study. *Arch Pediatr Adolesc Med*. 2011;165(10):906–912
25. Li C, Goran MI, Kaur H, Nollen N, Ahluwalia JS. Developmental trajectories of overweight during childhood: role of early life factors. *Obesity (Silver Spring)*. 2007;15(3):760–771

26. Hejazi S, Dahinten VS, Marshall SK, Ratner PA. Developmental pathways leading to obesity in childhood. *Health Rep*. 2009;20(3):63–69

27. Guo SS, Huang C, Maynard LM, et al. Body mass index during childhood, adolescence and young adulthood in relation to adult overweight and adiposity: the Fels Longitudinal Study. *Int J Obes Relat Metab Disord*. 2000;24(12):1628–1635

28. Huang DY, Lanza HI, Wright-Volel K, Anglin MD. Developmental trajectories of childhood obesity and risk behaviors in adolescence. *J Adolesc*. 2013;36(1):139–148

29. Ventura AK, Loken E, Birch LL. Developmental trajectories of girls' BMI across childhood and adolescence. *Obesity (Silver Spring)*. 2009;17(11):2067–2074

30. Ziyab AH, Karmaus W, Kurukulaaratchy RJ, Zhang H, Arshad SH. Developmental trajectories of body mass index from infancy to 18 years of age: prenatal determinants and health consequences. *J Epidemiol Community Health*. 2014;68(10):934–941

31. Berenson GS, Srinivasan SR, Bao W, Newman WP III, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. *N Engl J Med*. 1998;338(23):1650–1656

32. Srinivasan SR, Bao W, Wattigney WA, Berenson GS. Adolescent overweight is associated with adult overweight and related multiple cardiovascular risk factors: the Bogalusa Heart Study. *Metabolism*. 1996;45(2):235–240

33. Goldschmidt I, Di Nanni A, Streckenbach C, Schnell K, Danne T, Baumann U. Improvement of BMI after lifestyle intervention is associated with normalisation of elevated ELF score and liver stiffness in obese children. *BioMed Res Int*. 2015:2015;457473

34. Kirk S, Zeller M, Claytor R, Santangelo M, Khoury PR, Daniels SR. The relationship of health outcomes to improvement in BMI in children and adolescents. *Obes Res*. 2005;13(5):876–882

35. Raitakari OT, Juonala M, Rönnemaa T, et al. Cohort profile: the cardiovascular risk in Young Finns Study. *Int J Epidemiol*. 2008;37(6):1220–1226

36. Buscot M-J, Wotherspoon SS, Magnussen CG, et al. Bayesian hierarchical piecewise regression models: a tool to detect trajectory divergence between groups in long-term observational studies. *BMC Med Res Methodol*. 2017;17(1):86

37. Wen X, Kleinman K, Gillman MW, Rifas-Shiman SL, Taveras EM. Childhood body mass index trajectories: modeling, characterizing, pairwise correlations and socio-demographic predictors of trajectory characteristics. *BMC Med Res Methodol*. 2012;12:38

38. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ*. 2000;320(7244):1240–1243

39. Li L, Hardy R, Kuh D, Lo Conte R, Power C. Child-to-adult body mass index and height trajectories: a comparison of 2 British birth cohorts. *Am J Epidemiol*. 2008;168(9):1008–1015

40. Li L, Hardy R, Kuh D, Power C. Life-course body mass index trajectories and blood pressure in mid life in two British birth cohorts: stronger associations in the later-born generation. *Int J Epidemiol*. 2015;44(3):1018–1026

41. Kruschke J. *Doing Bayesian Data Analysis: A Tutorial With R, JAGS, and Stan*. 2nd ed. Cambridge, MA: Academic Press; 2014

42. Fitzmaurice G, Davidian M, Verbeke G, Molenberghs G, eds. *Longitudinal Data Analysis Chapman and Hall/CRC*. Boca Raton, FL: CRC Press; 2008

43. *R: A Language and Environment for Statistical Computing* [computer program]. Vienna, Austria: R Foundation for Statistical Computing; 2013

44. Dinsdale H, Hancock C, Rutter H. *National Child Measurement Programme: Changes in Children's Body Mass Index Between 2006/07 and 2012/13*. London, United Kingdom: Public Health England; 2014

45. Taveras EM, Rifas-Shiman SL, Sherry B, et al. Crossing growth percentiles in infancy and risk of obesity in childhood. *Arch Pediatr Adolesc Med*. 2011;165(11):993–998

46. Nader PR, O'Brien M, Houts R, et al; National Institute of Child Health and Human Development Early Child Care Research Network. Identifying risk for obesity in early childhood. *Pediatrics*. 2006;118(3). Available at: www.pediatrics.org/cgi/content/full/118/3/e594

47. Lee JM, Appugliese D, Kaciroti N, Corwyn RF, Bradley RH, Lumeng JC. Weight status in young girls and the onset of puberty [published correction appears in *Pediatrics*. 2007;120(1):251]. *Pediatrics*. 2007;119(3). Available at: www.pediatrics.org/cgi/content/full/119/3/e624

48. Klish WJ. *Clinical Evaluation of the Obese Child and Adolescent*. Alphen aan den Rijn, Netherlands: Wolters Kluwer; 2016

49. Mei Z, Grummer-Strawn LM, Thompson D, Dietz WH. Shifts in percentiles of growth during early childhood: analysis of longitudinal data from the California Child Health and Development Study. *Pediatrics*. 2004;113(6). Available at: www.pediatrics.org/cgi/content/full/113/6/e617

50. Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents; National Heart; Lung, and Blood Institute. Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. *Pediatrics*. 2011;128(suppl 5):S213–S256

51. Speiser PW, Rudolf MC, Anhalt H, et al; Obesity Consensus Working Group. Childhood obesity. *J Clin Endocrinol Metab*. 2005;90(3):1871–1887

52. Barlow SE; Expert Committee. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. *Pediatrics*. 2007;120(suppl 4):S164–S192

53. Krebs NF, Himes JH, Jacobson D, Nicklas TA, Guilday P, Styne D. Assessment of child and adolescent

overweight and obesity. *Pediatrics*. 2007;120(suppl 4):S193–S228

54. Huang JS, Barlow SE, Quiros-Tejeira RE, Scheimann A, Skelton J, Suskind D, et al. Childhood obesity for pediatric gastroenterologists. *J Pediatr Gastroenterol Nutr*. 2013;56(1): 99–109
55. Singh AS, Mulder C, Twisk JW, van Mechelen W, Chinapaw MJ. Tracking of childhood overweight into adulthood: a systematic review of the literature. *Obes Rev*. 2008;9(5): 474–488
56. Gungor NK. Overweight and obesity in children and adolescents. *J Clin Res Pediatr Endocrinol*. 2014;6(3): 129–143
57. Ekberg J, Angbratt M, Valter L, Nordvall M, Timpka T. History matters: childhood weight trajectories as a basis for planning community-based obesity prevention to adolescents. *Int J Obes*. 2012;36(4):524–528
58. Song M, Hu FB, Wu K, Must A, Chan AT, Willett WC, et al. Trajectory of body shape in early and middle life and all cause and cause specific mortality: results from two prospective US cohort studies. *BMJ*. 2016;353:i2195
59. Hirko KA, Kantor ED, Cohen SS, Blot WJ, Stampfer MJ, Signorello LB. Body mass index in young adulthood, obesity trajectory, and premature mortality. *Am J Epidemiol*. 2015;182(5):441–450
60. Hoekstra T, Barbosa-Leiker C, Koppes LLJ, Twisk JWR. Developmental trajectories of body mass index throughout the life course: an application of latent class growth (mixture) modelling. *Longit Life Course Stud*. 2011;2(3):12