ARTICLE

Predictors and Tracking of Body Mass Index From Adolescence Into Adulthood

Follow-up of 18 to 20 Years in the Oslo Youth Study

Elisabeth Kvaavik, MSc; Grethe S. Tell, MPH, PhD; Knut-Inge Klepp, MPH, PhD

Objectives: To examine tracking of body mass index (BMI) (weight in kilograms divided by the square of height in meters) from age 15 to 33 years, to examine the effect of adolescent and adult health-related behavior and parents' BMI and education on adult BMI; and to examine changes in lifestyle factors as predictors of adult overweight and obesity.

Methods: A longitudinal study with 18 to 20 years of follow-up in a cohort from Oslo, Norway (N=485); mean age was 15 years at baseline. Weight, height, physical fitness, leisure time physical activity (LTPA), smoking, and education were assessed at baseline and follow-up. Parents' height, weight, and education were assessed at baseline.

Results: Tracking of BMI from age 15 to 33 years was high (r=0.54). Adolescent BMI, father's BMI, the subject's own LTPA, adult smoking, and sex explained 44.1% of the variation in adult BMI. The odds ratio (95% confidence interval) of having a BMI of 25 or more as an adult was 0.07 (0.03-0.14) for lowest vs highest quartile of adolescent BMI. The corresponding odds ratio of having a BMI of 30 or more was 0.02 (0.002-0.14). Those who increased their LTPA level between adolescence and adulthood had a lower risk of adult overweight than those with a stable low LTPA level.

Conclusions: Tracking of BMI from adolescence into adulthood was substantial. Changes in LTPA between adolescence and adulthood predicted the risk of adult overweight, suggesting that the foundation for adult body weight is laid during adolescence. Implications of this would be to emphasize physical activity among youths.

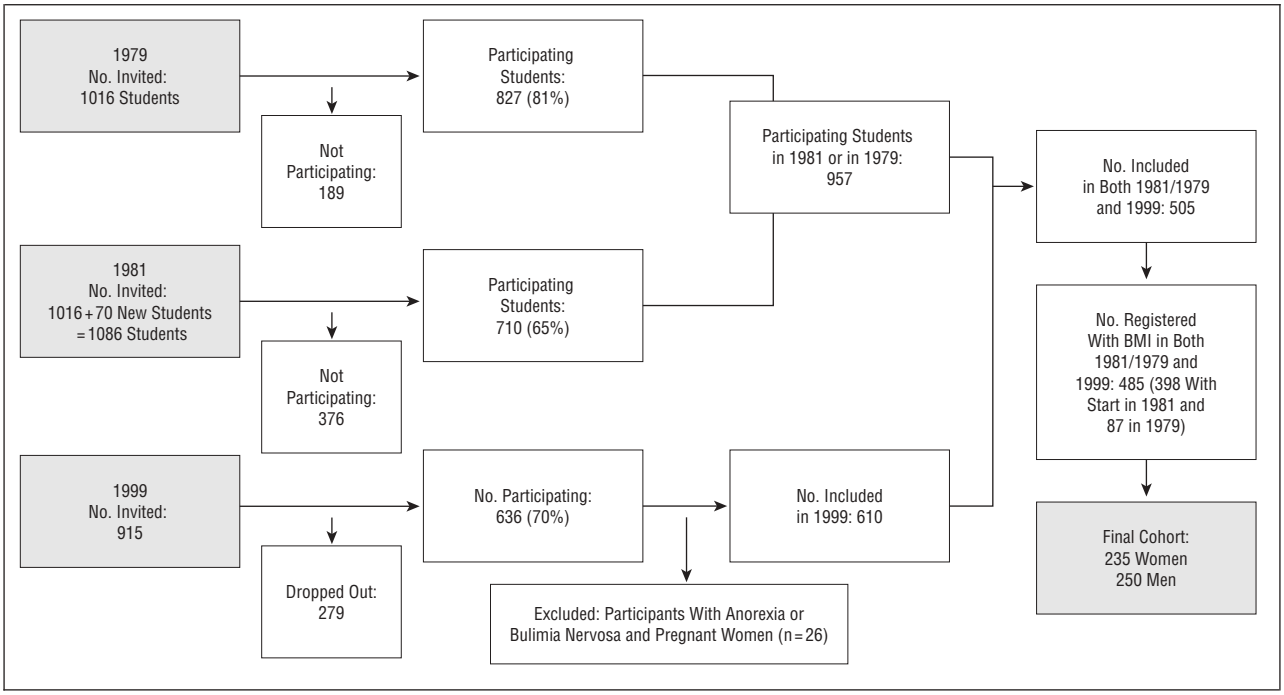
Arch Pediatr Adolesc Med. 2003;157:1212-1218

From the Institute for Nutrition Research, University of Oslo, Oslo, Norway (Ms Kvaavik and Dr Klepp); and Department of Public Health and Primary Health Care, University of Bergen, Bergen, Norway (Dr Tell).

DURING RECENT decades, mean body mass index (BMI) (weight in kilograms divided by the square of height in meters) and the proportion of overweight and obese persons have increased worldwide.[1,2] Several studies have found substantial tracking (defined as subjects maintaining their relative position within their age-sex group over time[3]) of body weight and obesity.[4-9] The follow-up periods vary considerably between these studies, ranging from 6 to 28 years. However, few studies have addressed the age span from adolescence into adulthood.[4,6,10] While parents' BMI is associated with their children's BMI,[10,11] only a few studies have addressed the effect of parents' body weight on change in their children's body weight from childhood or adolescence into adulthood.[10,11]

Studies show that physical activity is inversely related to BMI, and reduced physical activity plays an important role in the increasing body weight reported around the world.[12-14] However, the effect on adult BMI of physical activity and fitness during adolescence and changes in physical activity from adolescent age into adulthood is not known. In addition to physical activity, level of education has been reported to be inversely related to BMI.[15,16] These studies were, however, cross-sectional, and we are not aware of studies on educational attainment from adolescence to adulthood as a predictor of adult BMI. Smoking is reported to be inversely related to BMI in men and women,[17,18] and smoking cessation typically leads to an increase in BMI at least to the level of persons who have never smoked.[19] The effect of changing smoking status between adolescence and adulthood on adult overweight and obesity has not been reported.

The aim of the present study was to examine the degree of tracking of BMI from age 15 to 33 years, and to investigate whether adolescent BMI, physical fitness, leisure time physical activity (LTPA), and smoking, as well as parents' BMI and educational level, predict adult BMI. Another aim was to explore whether parents' BMI, change in LTPA, change in



Flowchart showing participation and participation rates in the Oslo Youth Study. BMI indicates body mass index.

smoking status, and the subject's own and parents' education between 15 and 33 years predict overweight and obesity at age 33 years.

METHODS

STUDY DESIGN

The Oslo Youth Study baseline survey was conducted during 1979, when students in grades 5 to 7 (mean age, 13 years; range, 11-16 years) attending 6 schools in Oslo, Norway, were invited to participate. Participants completed a questionnaire at school and underwent a brief health examination including measurements of height and weight. A nearly identical follow-up study was repeated in 1981. At both times, parents were asked to fill out a questionnaire. In 1999, participants completed a mailed questionnaire. In this study, 1981 was chosen as baseline because the majority of participants at that time were beyond puberty, which is important with regard to body composition and BMI.[20] To increase the sample size, 1981 data were supplemented with data from 1979 for participants who did not participate in 1981. The findings did not change from 1981-only data when these supplemental 1979 data were used.

As part of the study, an intervention was implemented and evaluated. Both the children who received the intervention and those who did not are included in this study. The intervention is thoroughly described elsewhere.[21] The study was approved by the Norwegian Data Inspectorate as well as the City of Oslo's health authorities.

SUBJECTS

This article includes information on 485 participants (**Figure**). The number invited in 1981 was higher than in 1979 because additional students then attended the participating schools. The number invited in 1999 was lower than in 1981 because of deaths (n=6), unknown addresses (n=115), refusals to participate in an intermediate survey in 1989 (n=21), and emigration (n=29). Participants who reported being pregnant (n=22) or having anorexia nervosa (n=1) or bulimia nervosa (n=3) in 1999 were excluded from the analyses. The cohort comprised those who participated both at baseline and in 1999 and included 505 subjects, of whom 485 had information available on height and weight both at baseline and in 1999. The overall participation rate among those eligible in 1981 was 45% (485/1086 minus 6 who had died), while the overall participation rate among those eligible in 1999 was 55% (485/915 minus 26 excluded).

BASELINE DATA COLLECTION

Leisure time physical activity was defined as at least 30 minutes of physical activity of an intensity to produce sweating and being short of breath. Frequencies ranged from "more seldom than twice a month" to "daily." Cigarette smoking frequency ranged from "never" to "daily." The respondent was defined as a regular smoker if he or she smoked cigarettes at least weekly.

The health examination included measurements of height and weight (to the nearest millimeter and 100 g, respectively) and assessment of sexual maturation by the method modified by Tanner for the development of pubic hair.[20,22] Physical fitness was assessed by pulse counting while students performed on ergometric bicycles, as an indirect assessment of maximal oxygen uptake.[23]

A total of 879 mothers and 713 fathers completed the baseline questionnaire. The 1981 to 1999 cohort comprised 452 mothers and 385 fathers. Data from 3 mothers for whom the differences between mother's and child's age were more than 50 years were excluded, leaving 482 students with parental participation. The parental questionnaire included information on educational level, height, and weight. Educational attainment ranged from "elementary school" (7 years) to "college/university." In the analyses, "high school" (12 years) and "college/university" were combined to represent the highest level of education.

DATA COLLECTION IN 1999

Questions included frequency of LTPA, smoking habits, educational level, weight, and height. Questions regarding LTPA and

Table 1. Participant Characteristics at Baseline and Follow-up and Characteristics of Parents at Baseline: The Oslo Youth Study

	Females	Males	All
Adolescence (baseline)	n = 235	n = 250	N = 485
Age, mean (SD), y*	14.6 (1.1)	14.7 (1.1)	14.7 (1.1)
BMI, mean (SD)*	19.7 (2.4)	19.4 (2.6)	19.6 (2.5)
LTPA at least twice a week, %†	53.4	66.3‡	60.0
LTPA once a month or less, %†	20.7	15.7	18.1
Fitness, mL O_2/(kg×min), mean (SD)*§	48.3 (9.2)	53.6 (10.9)‖	51.1 (10.5)
Smokers, smoking weekly, %†	13.7	10.4	12.0
Parental values (baseline)	n = 233	n = 249	N = 482
Mother's age, mean (SD), y*¶	42.1 (6.1)	42.5 (6.5)	42.3 (6.3)
Father's age, mean (SD), y*	45.4 (7.0)	46.2 (7.3)	45.8 (7.2)
Mother's BMI, mean (SD)*	22.4 (3.1)	22.4 (3.0)	22.4 (3.1)
Father's BMI, mean (SD)*	23.9 (2.4)	24.2 (2.5)	24.1 (2.4)
Mother's BMI ≥25, %†	13.3	17.7	15.7
Father's BMI ≥25, %†	31.1	35.8	33.6
Mother's BMI ≥30, %†	3.3	2.6	2.9
Father's BMI ≥30, %†	0	1.5	0.8
Mother's education, high school graduate, %†	32.9	33.1	33.0
Father's education, high school graduate, %†	49.2	42.4	45.5
Adulthood (1999)	n = 235	n = 250	N = 485
Age, mean (SD), y*	32.3 (1.0)	32.5 (1.1)	32.4 (1.0)
BMI, mean (SD)*	23.3 (4.0)	25.7 (3.9)‖	24.5 (4.1)
BMI ≥25, %†	26.4	52.8‖	40.0
BMI ≥30, %†	7.2	10.8	9.1
LTPA at least twice a week, %†	37.3	36.5	36.9
LTPA once a month or less, %†	22.1	24.6	23.4
Smokers, smoking daily, %†	34.3	28.4	31.5
Education, at least 1 y of college, %†	55.4	44.8#	49.9

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters); LTPA, leisure time physical activity; O_2, oxygen.
*Unpaired *t* test to compare sexes.
†χ^2 Test to compare sexes.
‡$P<.01$.
§In 185 females and 200 males.
‖$P<.001$.
¶Subjects in whom the difference between the mother's age and the responder's age was more than 50 years were excluded from analyses including data about the mother.
#$P<.05$.

smoking were the same as at baseline; however, the definition of a regular smoker was daily smoking according to convention.[24] Educational attainment ranged from "9 years of elementary/secondary school" to "more than 4 years of college/university." In the analyses, "1 to 4 years" and "more than 4 years of college/university" were combined and represent the proportion of participants with a high level of education, reflecting that the level of education increased considerably from the 1960s to the 1990s.[25]

CHANGES IN BMI RANK

To assess a subject's movement from one BMI quartile during adolescence to another at follow-up, participants were divided into 3 groups: (1) 1 to 3 BMI quartiles lower, (2) no change, and (3) 1 to 3 BMI quartiles higher.

STATISTICS

Unpaired *t* tests were used to compare men and women with respect to continuous variables, and in the attrition analyses to compare responders and dropouts with respect to age and parents' BMI at baseline. The χ^2 tests were used to compare men and women and responders with dropouts with respect to categorical variables.

Analysis of variance with Bonferroni correction for multiple comparisons was used in the tracking analyses to examine change in BMI within quartiles of BMI at baseline. Analysis of variance was also used to assess adult BMI within categories of parents' overweight and differences in baseline and follow-up variables by change in BMI rank. The models were tested for interaction by sex, and because no interaction effects were seen, the sexes were combined in the analyses. In addition, analysis of variance was used to assess whether there were differences in adolescent BMI and physical fitness between responders and dropouts in the attrition analyses.

Unadjusted and adjusted linear regression analysis was used to predict the variability in BMI in 1999. When 2 independent variables were internally correlated with $r \geq 0.5$ in bivariate analyses, the variable with the lowest correlation with the dependent variable was excluded from the multivariate analyses (applies to parental education).

Logistic regression was used to compute odds ratios for BMI of 25 or more and 30 or more in 1999 by BMI quartile at baseline, parents' BMI, change in LTPA level, and smoking status from baseline to 1999, as well as by subjects' own and parents' education.

The program package SPSS 11.0 for Windows was used in all analyses (SPSS Inc, Chicago, Ill).

ATTRITION ANALYSES

For the attrition analyses, responders were defined as subjects who participated both at baseline and in 1999, including those with information about height and weight at baseline or in 1999 (n=505). Responders also included those excluded in 1999 (n=26). Dropouts were those participating at baseline only (n=426). Responders and dropouts were compared with respect to baseline sex distribution, age, BMI, fitness, LTPA, smoking, parents' education, and parents' BMI. Compared with dropouts, responders were older (14.7 vs 14.3 years; $P<.001$) and the proportions of men and of smokers were lower (49.8% vs 56.2%; $P=.05$; and 12.7% vs 19.0%; $P=.009$, respectively). There were no significant differences between responders and dropouts with regard to mean BMI, LTPA, fitness, parents' education, or parents' BMI.

RESULTS

Table 1 presents data from participants followed up from baseline to 1999 (N=485). Except for baseline LTPA and physical fitness, follow-up BMI, and education, no sex differences were seen. Among men, the proportion who were not physically active during leisure time increased from 15.7% in adolescence to 24.6% in adulthood. Mean BMI and the proportions overweight and obese were greater for the adult participants (mean age, 32.4 years) than for their parents 18 years earlier, when their mean age was 44.0 years.

Tracking of BMI from 15 to 33 years by quartiles of baseline BMI for men and women combined is presented in **Table 2**. The correlation between BMI in adolescence and in adulthood was significant, $r=0.54$ ($P<.001$) for men and women combined. The proportion of subjects who remained in the same quartile in 1999 compared with baseline was 38.6%, while 29.9% de-

Table 2. Adolescent (Mean Age, 14.7 Years) and Adult (Mean Age, 32.4 Years) BMI Within Adolescent BMI Quartiles: The Oslo Youth Study

Adolescent BMI Quartile*	Women			Men		
	No.	Adolescent BMI, Mean (95% CI)	Adult BMI, Mean (95% CI)	No.	Adolescent BMI, Mean (95% CI)	Adult BMI, Mean (95% CI)
1	58	16.9 (16.7-17.1)	21.0 (20.1-22.0)	63	16.7 (16.5-16.9)	23.4 (22.6-24.2)
2	60	18.8 (18.6-18.9)	22.5 (21.6-23.4)	61	18.4 (18.3-18.5)	24.4 (23.6-25.2)
3	59	20.4 (20.3-20.5)	23.6 (22.6-24.5)	64	19.8 (19.7-19.9)	25.4 (24.6-26.2)
4	58	22.9 (22.5-23.3)	26.1 (25.1-27.0)	62	22.9 (22.3-23.5)	29.5 (28.7-30.3)

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters); CI, confidence interval.
*The BMI limits for the adolescent BMI quartiles (Qs) for women are: Q1, 14.9 to 18.0; Q2, 18.1 to 19.6; Q3, 19.7 to 21.2; and Q4, 21.3 to 27.4; and for men: Q1, 15.0 to 17.6; Q2, 17.7 to 19.1; Q3, 19.2 to 20.5; and Q4, 20.6 to 30.0.

Table 3. Baseline Variables (Mean and 95% Confidence Interval) According to Subsequent Changes in Relative BMI: The Oslo Youth Study

	Reduced BMI 1 to 3 Quartiles (n = 144)	Same BMI Quartile (n = 186)	Increased BMI 1 to 3 Quartiles (n = 152)
Baseline variables			
Sex, % female	76.4	46.5	24.7
Age, y*	14.5 (14.3-14.7)	14.3 (14.2-14.5)	14.5 (14.3-14.6)
Puberty development, Tanner stage†	3.9 (3.7-4.0)	3.6 (3.5-3.8)	2.9 (2.8-3.1)
BMI*	20.3 (19.9-20.7)	19.7 (19.4-20.0)	18.1 (17.7-18.4)
BMI ≥25, %*	3.1 (0-7.0)	9.5 (6.3-12.8)	0
Fitness, mL O_2/(kg×min)*	49.4 (47.2-51.5)	49.3 (47.5-51.1)	54.2 (52.3-56.2)
LTPA at least twice a week, %*	63.4 (53.9-72.9)	56.3 (48.6-64.1)	60.4 (52.2-68.7)
LTPA once a month or less, %*	15.6 (8.8-23.1)	20.1 (14.0-26.3)	20.5 (14.0-27.1)
Smokers, smoking weekly, %*	11.9 (5.6-18.3)	10.6 (5.4-15.8)	10.0 (4.5-15.6)
Mother's BMI	22.1 (21.6-22.7)	22.8 (22.3-23.3)	22.0 (21.5-22.5)
Father's BMI	24.0 (23.5-24.4)	24.2 (23.8-24.6)	24.0 (23.6-24.4)
Mother's education, high school graduates, %	32.1 (24.1-40.1)	32.0 (25.0-39.0)	35.0 (27.2-42.7)
Father's education, high school graduates, %	45.0 (35.7-54.4)	46.9 (38.8-55.0)	44.6 (35.7-53.6)
Follow-up variables (1999)			
BMI†	22.4 (21.8-23.1)	25.3 (24.8-25.8)	25.6 (24.9-26.2)
BMI ≥25, %†	14.8 (7.1-22.5)	46.5 (40.0-52.9)	55.4 (48.0-62.8)
LTPA at least twice a week, %†	37.0 (28.7-45.5)	42.7 (35.7-49.6)	29.9 (21.9-37.9)
Smokers, smoking daily, %†	38.2 (30.3-46.2)	35.5 (28.9-42.1)	18.9 (11.3-26.5)

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters); LTPA, leisure time physical activity; O_2, oxygen.
*Adjusted for Tanner stage and sex.
†Adjusted for sex.

creased 1 to 3 quartiles and 31.5% increased 1 to 3 quartiles (sex-specific analyses showed similar results).

As shown in **Table 3**, the group whose BMI quartile increased had been less sexually developed and had a lower BMI at baseline than the other 2 groups, while BMI at follow-up in this group and in the stable group was higher than in the group whose BMI quartile was reduced. The group whose BMI quartile was increased had the lowest prevalence of smokers at follow-up.

When baseline variables were examined in multivariate analyses as predictors of adult BMI, sex, adolescent BMI, father's BMI, and smoking remained significant (**Table 4**). When follow-up variables were included, smoking at baseline was no longer significant, and the final model included LTPA and smoking at follow-up in addition to sex, adolescent BMI, and father's BMI (Table 4).

Those in the highest BMI quartile at baseline had the highest risk of having a BMI of 30 or more in 1999. Correspondingly, those in the 2 lowest BMI quartiles at baseline had the lowest risk of having a BMI of 25 or more at follow-up. Participants whose father and mother both had a BMI less than 25 at baseline had reduced risk of having a BMI of 25 or more as adults (adjustments for adolescent BMI, Tanner stage, and sex did not alter the results) (**Table 5**). The respondents' BMIs in 1999 were higher if at least one of the parents had been overweight at baseline compared with both parents having been in the normal weight range; mean BMI and 95% confidence interval were 25.1 (24.5-25.6) vs 23.9 (23.4-24.3) (adjusted for adolescent BMI, Tanner stage, and parents' age at baseline).

Increased LTPA from adolescence into adulthood reduced the risk of being overweight as adults compared with a stable low level of LTPA, while quitting smoking increased the risk of being overweight in adulthood compared with those who smoked at both time points. The

Table 4. Prediction of BMI at Age 33 Years by Means of Multivariate Analyses Including Baseline Variables in Step 1, and Baseline and 1999 Variables in Step 2: The Oslo Youth Study

	Step 1		Step 2	
Variables	β*	*P* Value	β*	*P* Value
Baseline				
Father's education	−0.04	.41	−0.01	.86
LTPA	0.04	.37	0.06	.16
Fitness	0.02	.63	0.02	.68
Smoking	−0.10	.02	−0.07	.17
Baseline BMI	0.53	<.001	0.54	<.001
Mother's BMI	0.08	.10	0.07	.12
Father's BMI	0.14	.003	0.12	.007
Sex	−0.30	<.001	−0.31	<.001
	R^2 (R^2 adjusted) 45.6% (44.1%)			
1999				
Education			−0.02	.77
LTPA			−0.10	.03
Smoking			−0.11	.03
			R^2 (R^2 adjusted) 46.7% (44.7%)	

Abbreviations: BMI, body mass index; LTPA, leisure time physical activity.
*Standardized regression coefficient.

risk of being overweight was reduced if the educational level was high in both parents and participants compared with both having a low educational level (Table 5).

The results did not change when the intervention status was included as a covariate in the analyses of variance or in the linear and logistic regression analyses.

COMMENT

In this study, we saw a substantial degree of tracking of BMI from adolescence into adulthood. Several studies have investigated BMI tracking between or within childhood and adolescence[7,8] or within adulthood,[9] and one study has mainly dealt with BMI rebound (age at BMI minimum, ie, the point in childhood before BMI starts to increase).[5] One investigated how BMI of 20 or more at age 14 years affected the prevalence of having BMI of 27 or more at age 34 years and found a significant increased risk of overweight in adulthood among those who had a BMI of 20 or more in adolescence.[4] The Bogalusa Heart Study group[6] reported a strong correlation between ponderal index (weight in kilograms divided by the cube of height in meters) in childhood (ages 5 to 14 years) and 15 years later, while Whitaker and colleagues[10] found a significant increase in odds ratio of being obese in young adulthood with obesity (BMI ≥85th percentile) at ages 15 to 17 years. While the methods used to investigate tracking of BMI vary, the results consistently show a high degree of tracking from adolescence into adulthood, a finding supported by our study.

Unlike some previous studies, our study used relative BMI as an indicator of tracking, and we investigated the characteristics of the different tracking groups. Women were more likely than men to move down in BMI rank, whereas men tended to move up. This is in accordance with studies that have shown that weight among men has increased more than among women during recent decades.[26,27] Tracking was less pronounced or lacking among those who were not yet fully sexually mature at baseline, indicating that tracking is more pronounced after puberty when the growth spurt is over. This is in accordance with others who have reported that BMI patterns during postadolescence were more important than BMI rebound (ages 4-10 years) for total and percentage body fat in adulthood (ages 35-45 years).[5]

The subjects' own BMI during adolescence and fathers' BMI were the strongest independent predictors of adult BMI. In bivariate analyses, however, the correlations between fathers', as well as mothers', BMI and subjects' own BMI in adulthood were positive and of the same magnitude. This is consistent with findings from other studies showing that both fathers' and mothers' degree of overweight and obesity are predictive of the children's risk of becoming overweight.[10,11]

In this study, LTPA, rather than physical activity during work, was assessed. If those whose work requires physical labor are less physically active during leisure time, there might be an even stronger effect of physical activity on body weight than found here. This would be consistent with findings from studies on adults showing a considerable protective effect of LTPA on overweight.[13,28]

Smoking is inversely associated with BMI in both men and women,[17,18,29] and smoking status at follow-up was inversely associated with BMI in this study. Although the group was small (n=17), there was an increased risk of being overweight as an adult in the group who stopped smoking between adolescence and adulthood. Those who started smoking after adolescence and those who had been a smoker the entire period did not, however, have a reduced risk of being overweight or obese compared with never-smokers.

Educational level has been shown to be inversely associated with body weight.[15,16] In our study, however, only a high educational level among both parents and participants reduced the risk of adult overweight compared with both having a low educational level. A change in educational level from adolescence to adulthood did not affect the prevalence of either overweight or obesity. It is reasonable to believe that the effect of stable high educational level works through other factors, like physical activity and diet, by increasing the consciousness about these factors.[30,31]

The overall participation rate in this study (55%) is higher than what was observed in Myers and colleagues' study,[6] where 40% were followed up, but lower than in Barnekow-Bergkvist and colleagues' study[4] and Whitaker and colleagues' study,[10] where about 65% were followed up. These studies had a follow-up time comparable to that in our study. In the examination of those followed up vs the dropouts in our study, there were fewer smokers among those followed up. While fewer smokers might have reduced the possibility of studying the effect of smoking on body weight, it is not likely that this attrition impaired the validity of the conclusions with respect to tracking and prediction in our study.

Table 5. Odds Ratio of Having BMI of 25 or More or 30 or More in 1999 Given the BMI Quartile at Baseline, Change in LTPA Level, Change in Smoking Status, and Parents' and Own Education Baseline to 1999: The Oslo Youth Study

	BMI≥25				BMI≥30			
Variable (n)	No. (%)	OR	Adjusted OR	95% CI	No. (%)	OR	Adjusted OR	95% CI
Adolescent BMI								
Quartile 4 (120)	78 (65.0)	1.0	1.0*		33 (27.5)	1.0	1.0*	
Quartile 3 (123)	53 (44.2)	0.43	0.35	0.20-0.61	6 (5.0)	0.14	0.13	0.05-0.33
Quartile 2 (121)	37 (30.6)	0.24	0.14	0.08-0.26	4 (3.3)	0.09	0.08	0.03-0.23
Quartile 1 (121)	24 (19.8)	0.13	0.07	0.03-0.14	1 (0.8)	0.02	0.02	0.002-0.14
Parents' BMI								
One or both parents' BMI ≥25 (146)	78 (53.4)	1.0	1.0†		22 (15.1)	1.0	1.0†	
Both parents' BMI <25 (218)	62 (28.4)	0.35	0.54	0.33-0.89	9 (4.1)	0.24	0.61	0.24-1.60
LTPA level, baseline-1999								
Low-low (138)	56 (40.6)	1.0	1.0†		16 (11.6)	1.0	1.0†	
Low-high (53)	13 (24.5)	0.48	0.38	0.16-0.88	3 (5.7)	0.46	0.25	0.05-1.37
High-low (165)	79 (47.9)	1.35	1.04	0.61-1.75	16 (9.7)	0.82	0.63	0.26-1.54
High-high (124)	44 (35.5)	0.81	0.71	0.40-1.26	9 (7.3)	0.60	0.67	0.25-1.80
Smoking status, baseline-1999								
No-no (312)	124 (39.7)	1.0	1.0†		27 (8.7)	1.0	1.0†	
No-yes (111)	42 (37.8)	0.92	0.74	0.44-1.26	13 (11.7)	1.40	0.84	0.35-2.02
Yes-no (17)	11 (64.7)	2.78	3.21	1.04-9.90	1 (5.9)	0.66	0.26	0.02-4.25
Yes-yes (40)	16 (40.0)	1.01	0.78	0.36-1.69	3 (7.5)	0.86	0.46	0.11-1.98
Educational level, parents-own								
Low-low (170)	81 (47.6)	1.0	1.0†		21 (12.4)	1.0	1.0†	
Low-high (82)	35 (42.7)	0.82	1.27	0.69-2.36	6 (7.3)	0.56	0.75	0.25-2.23
High-low (64)	26 (40.6)	0.75	0.77	0.40-1.51	6 (9.4)	0.73	0.52	0.16-1.74
High-high (151)	42 (27.8)	0.42	0.55	0.33-0.92	8 (5.3)	0.40	0.71	0.27-1.87

Abbreviations: BMI, body mass index (calculated as weight in kilograms divded by the square of height in meters); CI, confidence interval; LTPA, leisure time physical activity; OR, odds ratio.
*Adjusted for Tanner stage and sex.
†Adjusted for adolescent BMI, Tanner stage, and sex.

What This Study Adds

Longitudinal studies may help us understand the effect of behavioral and other factors in adolescence on body weight later in life. This study shows that there is stability of relative body weight through life, and that both parents' BMI and subjects' own LTPA are important factors influencing adult body weight. This suggests that the foundation for adult body weight is laid already during adolescence, and implications of this would be to emphasize physical activity at a young age.

The adult BMI and proportions obese observed in this study are comparable to results from a previous Norwegian study[32] that found mean BMI among men and women 30 to 34 years old in 1994 to 1995 to be 25.0 and 23.4 and prevalence of obesity to be 8% and 6%, respectively. That the proportions of obese subjects were somewhat lower than in our study might be due to a period effect, as the cohorts included were born between 1925 and 1964.

In 1999, the responders' body weight and height were self-reported, as it was for parents' at baseline. Subjects reporting their own weight and height have a tendency to overestimate height and underestimate weight, resulting in an underestimation of BMI.[33] In a previous report from the Oslo Youth Study, the correlation between measured height in 1991 (age 25 years) and self-reported height in 1999 was high (r=0.96), and about 80% of both women and men reported the same height ±1 cm in 1999 as measured in 1991.[34] The heights reported in 1999 were 0.4 and 0.3 cm higher for women and men, respectively, than the heights measured in 1991. Thus, the overestimation of height in this study is not considerable. Also, an overestimation of height in 1999 will not affect the degree of tracking if the overestimation of height is of the same magnitude for all heights and weights. The latter is, however, questionable, as the smaller and heavier subjects tend to overestimate height more.[35,36] This might have attenuated the degree of tracking seen in this study, and the real tracking might be even stronger.

CONCLUSIONS

The main findings of this study were that BMI tracks significantly from adolescence into adulthood and that the subject's own BMI during adolescence, father's BMI, and LTPA and smoking in adulthood were strong predictors

of adult BMI. Smoking cessation between adolescence and adulthood increased the risk of being overweight as adults, while an increase in LTPA and a high educational level among parents and participants reduced the risk of being overweight as adults. The results from this study provide a strong rationale for obesity prevention at a young age. Such efforts should include the parents, and promotion of physical activity appears to be a critical component of such prevention efforts.

Accepted for publication June 9, 2003.

The Oslo Youth Study was supported by the Norwegian Cancer Society, Oslo. This work was supported by a grant from the EXTRA funds from the Norwegian Foundation for Health and Rehabilitation, Oslo (Ms Kvaavik).

We thank David R. Jacobs, Jr, PhD, for valuable comments on an earlier version of this article.

Corresponding author: Elisabeth Kvaavik, MSc, Institute for Nutrition Research, PO Box 1046 Blindern, N-0316 Oslo, Norway.

REFERENCES

1. Livingstone MB. Childhood obesity in Europe: a growing concern. *Public Health Nutr*. 2001;4:109-116.
2. Seidell JC. Obesity, insulin resistance and diabetes—a worldwide epidemic. *Br J Nutr*. 2000;83(suppl 1):S5-S8.
3. Mahoney LT, Lauer RM, Lee J, Clarke WR. Factors affecting tracking of coronary heart disease risk factors in children: the Muscatine Study. *Ann N Y Acad Sci*. 1991;623:120-132.
4. Barnekow-Bergkvist M, Hedberg G, Janlert U, Jansson E. Adolescent determinants of cardiovascular risk factors in adult men and women. *Scand J Public Health*. 2001;29:208-217.
5. 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;24:1628-1635.
6. Myers L, Coughlin SS, Webber LS, Srinivasan SR, Berenson GS. Prediction of adult cardiovascular multifactorial risk status from childhood risk factor levels: the Bogalusa Heart Study. *Am J Epidemiol*. 1995;142:918-924.
7. Wang Y, Ge K, Popkin BM. Tracking of body mass index from childhood to adolescence: a 6-y follow-up study in China. *Am J Clin Nutr*. 2000;72:1018-1024.
8. Williams S, Davie G, Lam F. Predicting BMI in young adults from childhood data using two approaches to modelling adiposity rebound. *Int J Obes Relat Metab Disord*. 1999;23:348-354.
9. Wilsgaard T, Jacobsen BK, Schirmer H, et al. Tracking of cardiovascular risk factors: the Tromso study, 1979-1995. *Am J Epidemiol*. 2001;154:418-426.
10. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med*. 1997;337:869-873.
11. Lake JK, Power C, Cole TJ. Child to adult body mass index in the 1958 British birth cohort: associations with parental obesity. *Arch Dis Child*. 1997;77:376-381.
12. Weinsier RL, Hunter GR, Heini AF, Goran MI, Sell SM. The etiology of obesity: relative contribution of metabolic factors, diet, and physical activity. *Am J Med*. 1998;105:145-150.
13. Haapanen N, Miilunpalo S, Pasanen M, Oja P, Vuori I. Association between leisure time physical activity and 10-year body mass change among working-aged men and women. *Int J Obes Relat Metab Disord*. 1997;21:288-296.
14. Martinez-Gonzalez MA, Martinez JA, Hu FB, Gibney MJ, Kearney J. Physical inactivity, sedentary lifestyle and obesity in the European Union. *Int J Obes Relat Metab Disord*. 1999;23:1192-1201.
15. Choiniere R, Lafontaine P, Edwards AC. Distribution of cardiovascular disease risk factors by socioeconomic status among Canadian adults. *CMAJ*. 2000;162(9, suppl):S13-S24.
16. Iribarren C, Luepker RV, McGovern PG, Arnett DK, Blackburn H. Twelve-year trends in cardiovascular disease risk factors in the Minnesota Heart Survey: are socioeconomic differences widening? *Arch Intern Med*. 1997;157:873-881.
17. Molarius A, Seidell JC, Kuulasmaa K, Dobson AJ, Sans S. Smoking and relative body weight: an international perspective from the WHO MONICA Project. *J Epidemiol Community Health*. 1997;51:252-260.
18. Rasky E, Stronegger WJ, Freidl W. The relationship between body weight and patterns of smoking in women and men. *Int J Epidemiol*. 1996;25:1208-1212.
19. Dennis BH, Pajak A, Pardo B, Davis CE, Williams OD, Piotrowski W. Weight gain and its correlates in Poland between 1983 and 1993. *Int J Obes Relat Metab Disord*. 2000;24:1507-1513.
20. Tell GS. Cardiovascular disease risk factors related to sexual maturation: the Oslo Youth Study. *J Chronic Dis*. 1985;38:633-642.
21. Klepp K-I, Øygard L, Tell GS, Vellar OD. Twelve year follow-up of a school-based health education programme: the Oslo Youth Study. *Eur J Public Health*. 1994;4:195-200.
22. Tanner JM. *Growth at Adolescence*. 2nd ed. Oxford, England: Blackwell Scientific Publications; 1962.
23. Hermansen L, Oseid S. Direct and indirect estimation of maximal oxygen uptake in pre-pubertal boys. *Acta Paediatr Scand Suppl*. 1971;217:18-23.
24. Kelder SH, Perry CL, Klepp KI, Lytle LL. Longitudinal tracking of adolescent smoking, physical activity, and food choice behaviors. *Am J Public Health*. 1994;84:1121-1126.
25. *Development in Norwegian Education 1980-1996*. Oslo: Statistics Norway; 1998. Report No. 3/98.
26. Tverdal A. Height, weight and body mass index of men and women aged 40-42 years [in Norwegian]. *Tidsskr Nor Laegeforen*. 1996;116:2152-2156.
27. Tverdal A. Prevalence of obesity among persons aged 40-42 years in two periods [in Norwegian]. *Tidsskr Nor Laegeforen*. 2001;121:667-672.
28. Rissanen AM, Heliovaara M, Knekt P, Reunanen A, Aromaa A. Determinants of weight gain and overweight in adult Finns. *Eur J Clin Nutr*. 1991;45:419-430.
29. Owen-Smith V, Hannaford PC. Stopping smoking and body weight in women living in the United Kingdom. *Br J Gen Pract*. 1999;49:989-990.
30. Kvaavik E, Meyer HE, Selmer RM, Egeland G, Tverdal A. Food habits in Hedmark related to gender, education and marital status [in Norwegian]. *Tidsskr Nor Laegeforen*. 1999;119:3406-3409.
31. Prattala R, Berg MA, Puska P. Diminishing or increasing contrasts? social class variation in Finnish food consumption patterns, 1979-1990. *Eur J Clin Nutr*. 1992;46:279-287.
32. Jacobsen BK, Njolstad I, Thune I, Wilsgaard T, Lochen ML, Schirmer H. Increase in weight in all birth cohorts in a general population: the Tromso Study, 1974-1994. *Arch Intern Med*. 2001;161:466-472.
33. Niedhammer I, Bugel I, Bonenfant S, Goldberg M, Leclerc A. Validity of self-reported weight and height in the French GAZEL cohort. *Int J Obes Relat Metab Disord*. 2000;24:1111-1118.
34. Kvaavik E, Tell GS, Klepp KI. Stability of body mass index from adolescence to adulthood [in Norwegian]. *Tidsskr Nor Laegeforen*. 2002;122:894-900.
35. Roberts RJ. Can self-reported data accurately describe the prevalence of overweight? *Public Health*. 1995;109:275-284.
36. Rowland ML. Self-reported weight and height. *Am J Clin Nutr*. 1990;52:1125-1133.