Original Investigation

Trajectories of cigarette smoking from adolescence to young adulthood as predictors of obesity in the mid-30s

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Abstract

Introduction: The purpose of this longitudinal study was to examine the relationship between two major health problems, smoking and obesity, and to determine to what extent trajectories of cigarette smoking from early adolescence to young adulthood are related to obesity in the mid-30s.

Methods: Participants ($N = 806$) were interviewed using a structured questionnaire at 6 points in time over a period of 23 years. Semiparametric group-based modeling and logistic regression analyses were used to analyze the data. The main outcome measure was obesity, assessed by body mass index in the mid-30s.

Results: Five distinct trajectories of tobacco use were identified ($N = 806$): heavy/continuous smokers, late starters, quitters/decreasers, occasional smokers, and nonsmokers. Compared with nonsmokers, heavy/continuous smokers or late starters had a significantly lower likelihood of obesity. Also, compared with nonsmokers or occasional smokers, heavy/continuous smokers or late starters had a significantly lower likelihood of being overweight or obese.

Discussion: Smoking cessation programs should focus on weight control methods, such as physical exercise and learning healthy habits. In addition, weight control programs should incorporate smoking cessation efforts as integral components.

Introduction

Obesity and smoking, two major public health concerns, are important causes of poor physical health in the United States. There has been increasing evidence of the negative relationship between obesity and physical health, such as type 2 diabetes, coronary artery disease, and certain malignancies (Bellanger & Bray, 2005; Bray, 2004). As regards smoking, the long-term adverse consequences of smoking include such serious diseases as lung cancer, coronary artery disease, chronic lung disease, stroke, and other malignant diagnoses (Centers For Disease Control and Prevention [CDC], 2005).

Both smoking and obesity are related to risk factors, such as poor diet, low physical activity, background factors (e.g., gender, age, low parental education, and low family income), and depression. Therefore, it is important to control for these factors when examining the association between the trajectories of smoking and subsequent obesity.

There is evidence that smoking aids in maintaining low body weight. Nicotine use can contribute to reducing body weight by adjusting the body weight set point (Cabanac & Frankham, 2002). Smoking is also inversely related to body mass index (BMI) in both men and women (Chatkin & Chatkin, 2007). Furthermore, once people stop smoking, they tend to gain weight (Munafo, Tilling, & Ben-Shlomo, 2009; Pisinger & Jorgensen, 2007), which is perceived as a main barrier against quitting smoking (Pisinger & Jorgensen; Pomerleau, Zucker, Brouwer, Pomerleau, & Stewart, 2001).

The present research examines the interface of these two major health problems by identifying the trajectories of smoking behavior between early adolescence and young adulthood and then examining the relationship of each smoking trajectory to obesity in adulthood (mid-30s). The studies in the literature have not examined the development of smoking behavior during early adolescence and young adulthood as it relates to obesity in adulthood. An approach using trajectory analyses (Nagin, 1999; Roeder, Lynch, & Nagin, 1999) enables one to examine the frequency, length of time, and initiation of smoking simultaneously and their associations with obesity in adulthood. This approach therefore has an advantage over an analysis that examines how early smoking predicts later obesity. Since cigarette smoking commonly begins in early adolescence, it is of interest to study the long-term outcomes associated with a history of smoking beginning in early adolescence. Several investigators have found distinct trajectories of smoking from early adolescence into adulthood. Chassin et al. (2008) identified nine trajectory groups. Costello, Dierker, Jones, and Rose (2008) identified six trajectory groups, and Riggs, Chou, Li, and Pentz (2007) identified four trajectory groups. In our earlier trajectory analyses (D. W. Brook et al., 2008), we found five trajectory groups that we labeled nonsmokers, occasional smokers, quit-

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ters, late starters, and heavy/continuous smokers. In this earlier study, we identified several adolescent predictors of the trajectories of smoking. The predictors included risk factors, such as low ego integration, greater externalizing behavior, and lower educational expectations and aspirations. The present study extends the research cited above by examining the association between the trajectories of cigarette smoking and obesity. The present study has an advantage in that it examines whether sociodemographic and behavioral factors confound or affect that association. The following factors have been found to be inversely related to both smoking and obesity: healthy eating habits and physical activity (Kvaavik, Meyer, & Tverdal, 2004) and socio-demographic background factors, including higher parental education, higher family income, and higher educational attainment (Orlando, Tucker, Ellickson, & Klein, 2004; Rasmussen, Tynelius, & Kark, 2003).

In sum, the present study is the first study to use prospective longitudinal data and growth mixture modeling (GMM) to examine the relationship between the various trajectories of smoking extending from adolescence to young adulthood and obesity in adulthood. We propose three hypotheses: (a) We hypothesize that the participants who belong to different smoking trajectory groups (i.e., heavy/continuous smokers, late starters, and quitters/decreasers) would be less likely to be obese than nonsmokers, with control on the background factors noted above. (b) We hypothesize that heavy/continuous smokers and late starters would be less likely to be obese than occasional smokers. (c) We hypothesize that quitters/decreasers would be more likely to be obese than continuous smokers.

Method

Participants and procedure

Data on the participants in this study came from a community-based random sample residing in one of two upstate New York counties first assessed in 1983. The participants' mothers were interviewed in 1975. The original maternal/youth study assessed problem behavior among youngsters. The sampled families were generally representative of the population of families in Albany and Saratoga, two upstate New York counties, with respect to gender, family intactness, family income, and education. There was a close match of the participants on family income, maternal education, and family structure with the 1980 survey conducted by the U.S. Bureau of Census. Interviews of the youths were conducted in 1983 (T2, $n = 756$), 1985–1986 (T3, $n = 739$), 1992 (T4, $n = 750$), 1997 (T5, $n = 749$), 2002 (T6, $n = 673$), and 2005–2006 (T7, $n = 607$). The mean ages (SD s) of participants at the follow-up interviews were 14.1 (2.8) at T2, 16.3 (2.8) at T3, 22.3 (2.8) at T4, 27.0 (2.8) at T5, 31.9 (2.8) at T6, and 36.6 (2.8) at T7, respectively. There were 806 participants for whom we have data at two or more waves between T2 and T6. These participants were included in the smoking trajectory analyses.

Those participants ($n = 222$) with missing data for BMI at T7 or who were pregnant at T7 were excluded from the analyses of the association between the earlier trajectories of tobacco use and adult obesity ($N = 584$). The excluded participants, compared with the included participants, were similar with respect to age ($t = -1.3$, p value = .19), age- and gender-adjusted T2 BMI ($t = 1.2$, p value = .21), and frequency of smoking at T2

($t = 1.3$, p value = .19). The excluded participants were significantly lower on parental educational level ($t = 2.7$, p value = 0.006) and family income ($t = 2.1$, p value = 0.04) and were less likely to be female, $\chi^2(1) = 19.2$, p value < .001).

Extensively trained and supervised lay interviewers administered interviews in private. Written informed consent was obtained from participants and their mothers in 1983, 1985–1986, and 1992 and from participants only in 1997, 2002, and 2005–2006. The Institutional Review Board of New York University School of Medicine authorized the use of human subjects in this research study at T7. Earlier waves of the study were approved by the Institutional Review Boards of Mount Sinai School of Medicine and New York Medical College. Additional information regarding the study methodology is available in prior publications (e.g., J. S. Brook, Whiteman, Gordon, & Cohen, 1986).

Measures

Cigarette smoking

The data were obtained from interviewer-administered questionnaires. At each follow-up wave (T2–T6), questions about tobacco use (adapted from the Monitoring the Future study; Johnston, O'Malley, Bachman, & Schulenberg, 2006) were included. In order to measure the lifetime quantity and frequency of using tobacco from childhood to the early 30s, at each time wave the questions asked about the frequency of using tobacco during the period from the last time wave through the current time wave. Specifically, the questions used were the lifetime frequency and quantity of tobacco use in childhood and early adolescence for T2 (1983, prior to T2), frequency and quantity of tobacco use during the past 2 years in adolescence for T3 (1985–1986, T2–T3), frequency and quantity of tobacco use during the past 5 years in the early 20s for T4 (1992, T3–T4), frequency and quantity of tobacco use during the past 5 years in the late 20s for T5 (1997, T4–T5), and frequency and quantity of tobacco use during the past 5 years in the late 20s and early 30s for T6 (2002, T5–T6). The tobacco measure at each point in time had a scale coded as *none* (0), *less than daily* (1), *1–5 cigarettes/day* (2), *about half a pack a day* (3), *about a pack a day* (4), and *about 1.5 packs a day or more* (5). The mean (SD) tobacco use scores at each point in time were 0.63 (1.12), 0.80 (1.29), 1.41 (1.63), 1.37 (1.61), and 1.25 (1.65), for T2–T6, respectively. The tobacco measure has been found to predict young adult psychiatric disorders (D. W. Brook, Brook, Zhang, Cohen, & Whiteman, 2002) and health problems (J. S. Brook, Brook, Zhang, & Cohen, 2004).

Obesity and overweight at T7

BMI is a measure of weight that also takes height into consideration. Height (in inches) and weight (in pounds) were assessed by self-report measures obtained at T7. BMI was calculated using weight in pounds and height in inches with the following equation (CDC, 2006):

$$\text{BMI} \left(\frac{\text{Weight}}{(\text{Height}) \times (\text{Height})} \right) \times 703.$$

In the equation, 703 was a constant used to account for the conversion between metric and English measures. For example, a person who weighs 220 pounds and is 6 feet 3 inches tall

(75 inches) has a BMI of 27.5. The CDC classify adults more than 20 years of age into one of the following four categories: (a) underweight, $\text{BMI} \leq 18.5$; (b) normal, $18.5 < \text{BMI} \leq 24.9$; (c) overweight, $24.9 < \text{BMI} \leq 29.9$; and (d) obese, $\text{BMI} > 29.9$.

We calculated the participant's BMI at T2. Due to the age span at T2 (9–19 years) and weight and height differences between males and females, BMI was then adjusted by partialling out the effects of age and gender. That is, in regression analyses, T2 BMI was the dependent variable and age and gender were the independent variables. For each participant, the fitted T2 BMI score was subtracted from the observed T2 BMI score. This difference was labeled the age- and gender-adjusted BMI at T2, which was used as a covariate.

Other covariates

At T6, the participant's personality and behavioral attributes were assessed. The following were included as covariates: (a) healthy habits scale (six items; Cronbach's $\alpha = .72$), which assessed habits such as eating (e.g., eating vegetables and fruits and avoiding fatty food), sleeping (e.g., getting at least 7 hours of sleep per night), and physical exercise (e.g., jogging and swimming). Each item was scored as: 1 = *never*, 2 = *seldom*, 3 = *sometimes*, 4 = *most days*, 5 = *nearly every day*, and 6 = *every day*; (b) physical health condition scale (single item). The item was scored as: 1 = *poor*, 2 = *fair*, 3 = *good*, and 4 = *excellent*; and (c) depression scale (nine items; Cronbach's $\alpha = .86$), which assessed depressive symptoms (e.g., feeling low in energy or slowed down and trouble sleeping). The depression measure has predictive validity (Lipman, Covi, & Shapiro, 1979). Each item was scored as: 1 = *not at all*, 2 = *a little*, 3 = *somewhat*, 4 = *quite a bit*, and 5 = *extremely*. Demographic characteristics were also included as covariates. These factors are gender (0 = *female*

and 1 = *male*), age at T7, mean family income (T2 to T4), the highest level of parental education (T2–T4), and the participant's educational level at T7.

Analysis

Mplus software (L. K. Muthén & Muthén, 2007) was used to identify the developmental trajectories of cigarette use ($N = 806$). For missing data (primarily due to individuals' nonparticipation in waves of data collection), we applied the full information maximum likelihood approach (Schaefer & Graham, 2002). The dependent variable (smoking at each point in time) was treated as a censored normal variable. The independent variables predicting trajectory group membership were gender and age at T2. Each trajectory polynomial was set as cubic. The GMM analyses used a multinomial logistic regression model for unordered polytomous responses (B. Muthén & Shedden, 1999). We used the minimum Bayesian information criterion (BIC) to determine the number of trajectory groups (G). To assure finding the maximum of the likelihood function, we used 50 random sets of starting values. Each participant was assigned to a trajectory group with the largest Bayesian posterior probability (BPP). For each of the trajectory groups, an indicator variable was created, which had a value of 1 if the participant had the largest BPP for that group and 0 otherwise. The observed trajectory for a group was the average of tobacco use at each timepoint for participants assigned to the group (see Figure 1).

We reported the mean (SD) BMI for each of the trajectory groups, with and without statistically adjusting for the covariates cited above (i.e., gender, age at T7, family income at T2–T4, parental education at T2–T4, participant's education at T7, age- and gender-adjusted BMI at T2, healthy habits at T6, physical health condition at T6, and depression at T6). SAS was then

Figure 1. Developmental trajectories of cigarette smoking extending from adolescence to age 32 ($N = 806$). Note. The smoking score categories are 5.00 = 1.5 packs a day or more, 4.00 = one pack per day, 3.00 = 1/2 pack per day, 2.00 = 1–5 cigarettes/day, 1.00 = less than daily smoking, and 0.00 = none.

used to perform logistic regression analyses to investigate the associations between the trajectory group membership and obesity ($n = 584$). The dependent variable was the indicator variable of obesity ($\text{BMI} > 29.9$) at T7. The independent variables were the covariates cited above and the G-1 indicator variables of trajectory group membership. The largest trajectory group was set as the reference group. For each independent variable, we reported the adjusted odds ratio (AOR) and its 95% CI. To facilitate interpretation of the regression coefficients and odds ratios, the continuous covariates (i.e., age at T7, family income at T2–T4, parental education at T2–T4, participant's education at T7, age- and gender-adjusted BMI at T2, healthy habits scale at T6, physical health condition scale at T6, and depression scale at T6) were converted to standardized scores. The SAS likelihood ratio test option was used to test whether there were differences in the likelihood of obesity ($\text{BMI} > 29.9$) between nonusers (the reference group) and other smoking trajectory groups and all other pairwise comparisons. We also ran a logistic regression analysis that added the interaction terms between gender and group memberships to the independent variables to test for differential gender associations. We conducted parallel analyses to examine the associations between the smoking trajectory memberships and being overweight or obese (i.e., $\text{BMI} > 24.9$).

Results

Trajectories of cigarette use

Solutions were calculated for the three-trajectory group model (likelihood = $-4,387$, BIC = $8,927$), four-trajectory group model (likelihood = $-4,290$, BIC = $8,781$), and five-trajectory group model (likelihood = $-4,213$, BIC = $8,673$). The six-trajectory group model did not converge. The five-trajectory group model had the best BIC score and thus was used. Figure 1 presents the observed trajectories and percentage for each of five trajectory groups. For each group, the mean BPP of the participants who were assigned to that group ranged from 89% to 97%.

The trajectory smoking groups were named heavy/continuous smokers (19.2%), late starters (12.7%), occasional smokers (17.6%), quitters/decreasers (8.1%), and nonsmokers (42.4%). As shown in Figure 1, the heavy/continuous smokers started smoking early, achieved the maximum level (i.e., about one pack a day or more) in their late 20s, and then tapered off slightly. In contrast, the late starters started smoking in late adolescence but achieved the same amount of smoking (i.e., one pack a day) as the heavy/continuous smokers in the late 20s. The participants then tapered off from that level. The occasional smokers had increasing smoking from adolescence to the early 20s and then stayed at a level of less than daily smoking during adulthood.

The quitters/decreasers started smoking as early as the heavy/continuous smokers and achieved the maximum level of smoking (i.e., daily smoking) in late adolescence. The participants then tapered off gradually from that level to less than daily smoking during adulthood.

There were no significant gender differences in the trajectory group memberships. As compared with nonsmokers, older participants at T2 were more likely to be heavy/continuous smokers ($t = 5.2$) and quitters/decreasers ($t = 2.2$). Younger participants at T2 were more likely to be late starters ($t = -6.2$) and occasional smokers ($t = -2.3$) than nonsmokers.

Smoking group memberships as predictors of obesity

The mean and SD ($n = 584$) of T7 BMI were 27.4 and 5.8, respectively. The average BMI for the males ($n = 270$, $M = 28.1$, $SD = 4.9$) was significantly greater, $t(574) = 2.8$, $p = .006$, than that for the females ($n = 314$, $M = 26.8$, $SD = 6.4$). Table 1 presents the frequencies and percentages of each T7 BMI category for both males and females. As shown in Table 1, 27.1% (25.8% for females and 28.5% for males) of the participants were obese at T7. As noted in Table 1, even though the frequencies over the four categories were significantly different, $\chi^2(3) = 56.7$, $p < .001$, between the men and the women, there were no significant gender differences in the obese category, $\chi^2(1) = 0.55$, $p = .46$.

Without adjusting for the control variables listed in the Analysis section, the mean (SD) BMI by the smoking trajectory groups were as follows: 28.0 (6.5), 27.8 (5.7), 27.4 (6.2), 26.8 (5.4), and 25.4 (4.2) for heavy/continuous smokers, nonsmokers, occasional smokers, later starters, and quitters/decreasers, respectively. The findings regarding BMI by the smoking trajectory groups differed somewhat with control on the demographic variables, age- and gender-adjusted BMI at T2, healthy habits at T6, physical health condition at T6, and depression at T6. The association of the BMI (mean [SD]) and the smoking trajectory groups after statistically adjusting for the variables cited above were as follows: 26.1 (0.5), 27.7 (0.3), 27.7 (0.5), 25.7 (0.6), and 26.3 (0.7) for heavy/continuous smokers, nonsmokers, occasional smokers, late starters, and quitters/decreasers, respectively. Nonsmokers and occasional smokers had the highest adjusted mean BMI, and the late starters had the lowest adjusted mean BMI.

We then ran the multivariate logistical regressions including the control variables noted in the Analysis section. Table 2 presents the results of the multivariate logistic regression analyses. Compared with nonsmokers, heavy/continuous smokers and late starters had significantly lower likelihood (AOR = 0.45 and 0.23, respectively) of obesity. In addition, greater participant's T7

Table 1. Frequencies of four weight categories based on T7 BMI ($n = 584$)

BMI category	Combined ($n = 584$), n (%)	Female ($n = 314$), n (%)	Male ($n = 270$), n (%)
Underweight (<18.5)	8 (1.4)	8 (2.6)	0 (0.0)
Normal (18.5–24.9)	201 (34.4)	144 (45.9)	57 (21.1)
Overweight (24.9–29.9)	217 (37.2)	81 (25.8)	136 (50.4)
Obese (>29.9)	158 (27.1)	81 (25.8)	77 (28.5)

Note. BMI = body mass index.

Table 2. Logistic regressions: trajectories of cigarette smoking with nonsmokers as the reference group on T7 obesity and overweight (*n* = 584)

Independent variables	Obesity (BMI > 29.9)	Overweight or obesity (BMI > 24.9)
	AOR (95% CI)	AOR (95% CI)
Heavy/continuous smokers	0.45 (0.23–0.90)*	0.39 (0.20–0.77)**
Late starters	0.23 (0.10–0.53)***	0.47 (0.23–0.95)*
Occasional smokers	0.89 (0.46–1.72)	1.52 (0.81–2.84)
Quitters/decreasers	0.45 (0.17–1.21)	0.60 (0.29–1.25)
Gender	0.97 (0.61–1.56)	4.73 (3.01–7.43)***
Age at T7	0.94 (0.73–1.21)	1.06 (0.83–1.34)
Parental educational level	0.92 (0.69–1.22)	0.87 (0.67–1.14)
Parental income T2–T4	0.90 (0.69–1.17)	0.83 (0.64–1.07)
Participant's education at T7	0.74 (0.55–0.99)*	0.90 (0.68–1.18)
Depression at T6	0.99 (0.78–1.26)	0.99 (0.80–1.23)
Better health condition at T6	0.63 (0.49–0.82)***	0.70 (0.56–0.89)*
Healthy habits at T6	0.64 (0.49–0.84)**	0.80 (0.63–1.02)
Age- and gender-adjusted BMI at T2	3.60 (2.68–4.84)***	4.51 (3.20–6.36)***

Note. AOR = adjusted odds ratio; BMI = body mass index.

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

educational level (AOR = 0.74), better health condition (AOR = 0.63), and better healthy habits (AOR = 0.64) were associated with lower likelihood of obesity. Greater T2 BMI adjusted for age and gender (AOR = 3.60) was associated with higher likelihood of T7 obesity.

The likelihood ratio tests (full results not shown) indicated that late starters also had significantly lower likelihood of obesity than occasional smokers, $\chi^2(1) = 7.8, p = .0052$. Heavy/continuous smokers were as likely to be obese as quitters/decreasers, $\chi^2(1) = 0.0, p = .9962$, occasional smokers, $\chi^2(1) = 2.4, p = .1196$, and late starters, $\chi^2(1) = 2.0, p = .1608$. Quitters/decreasers were as likely to be obese as late starters, $\chi^2(1) = 1.2, p = .2772$, and occasional smokers, $\chi^2(1) = 1.4, p = .2301$. The interaction analyses (full results not presented) showed that there were no significant gender differences in the associations between smoking group memberships and obesity (data available upon request).

As shown in Table 2, when the dependent variable was an indicator variable of being overweight or obese (BMI > 24.9), compared with nonsmokers, heavy continuous smokers and late starters had significantly lower likelihood (AOR = 0.39 and 0.47, respectively). The likelihood ratio tests also showed that heavy continuous smokers and late starters had significantly lower likelihood of being overweight or obese than occasional smokers, $\chi^2(1) = 10.7, p = .0011$ and $\chi^2(1) = 7.9, p = .0050$, respectively.

Discussion

We investigated the association between trajectories of tobacco use from adolescence to young adulthood and adult obesity in the mid-30s. The findings provided partial support for our hypotheses. Compared with nonsmokers, heavy/continuous

smokers or late starters had a significantly lower likelihood of obesity. Also, compared with nonsmokers or occasional smokers, heavy/continuous smokers or late starters had a significantly lower likelihood of being overweight or obese.

Our findings that the heavy/continuous smokers or late starters are less likely to be obese than nonsmokers or occasional smokers are partially consistent with the findings of a number of investigators who have conducted cross-sectional or longitudinal studies. For example, several investigators have noted that smokers in comparison with nonsmokers are less obese (Molarius, Seidell, Kuulasmaa, Dobson, & Sans, 1997). Even though smokers are less likely to be obese than nonsmokers, they do have higher morbidity and mortality compared with nonsmokers (CDC, 2005; Ezzati & Lopez, 2003).

Of great interest are the possible mechanisms that intervene between smoking and less obesity. First, smokers, particularly the late starters, may use smoking as a strategy for weight control. This may occur more frequently among adolescents and young adults. Second, nicotine does suppress one's appetite, and a decrease in appetite ultimately leads to lower caloric intake and lower body weight (Williamson, 1993). Third, tobacco use has an effect on the expenditure of energy (Perkins, 1992; Kvaavik et al., 2004). Smoking appears to lower the efficiency of caloric storage and to increase the metabolic rate, each of which may lead to lower body weight. For example, Perkins reported that up to 30 min after smoking, energy expenditure is approximately 10% higher than the normal energy expenditure. As noted by Kvaavik et al., this can result in a substantial increase in the expenditure of energy over a period of time, especially among heavy smokers.

It is important to note that the quitters/decreasers did not differ from the heavy/continuous smokers or late starters in obesity or being overweight. However, Munafò et al. (2009), in their study of males, reported that ex-smokers differ in BMI from current smokers. These findings warrant further research. Although we controlled for many factors that may underlie the relationship between the trajectories of smoking and obesity, it is plausible that there are other factors that affect the relationship between smoking and obesity. Additionally, it is possible that following the participants over a longer period of time may reveal evidence of further differences among the smoking trajectory groups.

The percentage of obese participants who were observed in this investigation is similar to findings obtained from the CDC (2008). According to the CDC Behavioral Risk Factor Surveillance System Survey data in New York State, the proportion of obese adults (18 years and older) is 25.1% (24.8% for females and 25.4% for males). The proportion of obesity is 26.2% for adults aged 35–44 years. These proportions are not appreciably different from our findings, indicating that 27.1% (25.8% of the females and 28.5% of the males) were in the obese category.

Our results have identified a number of nonsmoking behaviors that, if widely adopted, may help reverse the recently noted widespread increase in obesity in adults. Of all the variables assessed, general physical health condition and the establishment of healthy habits had the greatest effect on obesity. As regards physical activity, our findings are in accord with those of Rissanen, Heliovaara, Knekt, Reunanen, and Aromaa (1991),

who found that physical activity was inversely related to being overweight in adult Finns. Moreover, our study found that healthy eating habits (e.g., eating vegetables and fruits and avoiding fat) and getting sufficient sleep were related to less obesity. Our results regarding the significance of less fat consumption and increased fruit and vegetable consumption as protective factors against obesity are in accord with previous findings (Kahn et al., 1997). From a public health perspective, changes in eating behavior such as increased consumption of vegetables, regular exercise, and appropriate sleep are major ways of preventing excess adult weight gain.

One limitation of the research is the fact that the present study is based on self-reported data on height and weight. Most epidemiological studies rely on self-reported height and weight. Gorber, Tremblay, Moher, and Gorber (2007) reported that self-report of height may be overestimated and self-report of weight and BMI may be underestimated. Although self-reported height and weight have some systematic biases, validation studies suggest that the magnitude of any bias is too small to affect conclusions about associations in large-scale epidemiological studies (Craig & Adams, 2009; Gorber & Tremblay, 2009). Nevertheless, future research should not only include self-reported measures of height and weight, but also more objective measures. The present study is also limited by its lack of representation of ethnic minorities. We can only generalize our findings to our population of primarily White adolescents and adults. Nevertheless, our findings with regard to rates of obesity are similar to those reported recently by the CDC, which are highly representative of the current U.S. population. Additionally, our measures of tobacco use at each point in time covered a relatively large span of time during which there may have been some variability in the participants' smoking. Thus, we may have missed trajectory patterns with short periods of cessation. Related to this, we are unable to distinguish various trajectories involving light or infrequent tobacco use. Furthermore, we do not have biochemical measures of smoking status. It is possible that the quitters in particular have underreported their smoking level. In the present study, we identified five smoking trajectory groups, which is in the range (four to nine groups) reported by other investigators (e.g., four groups by Riggs et al., 2007 and nine groups by Chassin et al., 2008). Nevertheless, caution must be exercised in the interpretation of the results, and researchers must be aware of the sensitivity of the results to sample selection and model specifications. Finally, it is possible that factors related to both obesity and smoking status may reflect the existence of unknown additional factors, such as child conscientiousness (Martin & Friedman, 2000). Future research might present developmental graphs embodying both smoking and obesity over the same points in time in order to describe the concurrent evolution of both these behaviors.

Conclusions

The present findings 1) indicate that heavy smoking from early adolescence to young adulthood is associated with less obesity in adulthood, and 2) are consistent with other research documenting an inverse association between smoking and BMI. Any positive health effects associated with a lower rate of obesity among smokers in comparison with nonsmokers, however, are overshadowed by the many and severe health risks associated with smoking.

This study, which addresses important public health issues, contributes to an understanding of the developmental

links between (a) heavy continuous and late starting smoking during the period extending from early adolescence to young adulthood and (b) obesity in adulthood. Despite the fact that the analyses do not permit us to draw causal conclusions, the longitudinal nature of our data and the inclusion of control variables such as diet and physical activity in our analyses provide us with considerable evidence for a linkage between patterns of smoking in adolescence/adulthood and adult obesity. Future research should focus on the mechanisms that connect late starting smoking and a lower risk for obesity. A better understanding of the mechanism underlying the relationship between smoking and obesity will advance our knowledge about the metabolic and behavioral factors that determine body weight in general. For example, there is the danger that some adolescents may use lowered weight as a stimulus or rationalization for continued smoking. Thus, smoking prevention or cessation programs should take this possibility into account. Future research should also examine the relationship between trajectories of smoking and other health-related behaviors.

This research also provides evidence for associations between parental and participants' educational levels, as well as healthy habits including diet and physical exercise and lowered obesity in adulthood. Consequently, prevention programs for smoking should include these areas and should be initiated at a young age. In addition to physical exercise, smoking programs should include knowledge about the consequences of smoking (e.g., physical and mental health problems). Smoking intervention programs should specifically target those individuals who use smoking as a means to control their weight. Related to this, smoking cessation programs should emphasize the importance of appropriate eating, balanced nutrition, and physical exercise as a means of weight control instead of smoking. In addition, weight control programs should have smoking cessation efforts as integral components and should begin at an early age for those with high BMI. From an intervention perspective, an integrated program including both smoking cessation or prevention and encouragement of a healthy lifestyle should be most effective.

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Declaration of Interests

None declared.

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