Original Investigation

Pubertal timing and smoking initiation in adolescent females: Differences by race

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Abstract

Introduction: The purpose of this study was to examine whether (a) early pubertal timing effects on smoking onset existed for both White and Black girls and (b) whether the association between pubertal timing and smoking onset was moderated by race.

Methods: Participants included 264 girls (14.9 ± 2.2 years, 164 White, and 100 Black) at the baseline report of a longitudinal study of whom 153 reported smoking and age at first cigarette.

Results: Kaplan–Meier analysis stratified by racial group showed a significant difference between the pubertal timing groups for Black girls only. After accounting for covariates using Cox regression, there was no significant interaction between pubertal timing and racial group. There was a main effect of pubertal timing indicating that late maturers were at significantly lower risk for smoking initiation compared with the early and on-time groups, but the early and on-time groups were not significantly different from each other.

Discussion: Results point to equal risk of early smoking onset for early and on-time maturers of both racial groups, indicating the need for smoking prevention in early adolescence for both White and Black females.

Introduction

There is substantial evidence that early pubertal timing is associated with higher rates and earlier initiation of substance use (Dick, Rose, Viken, & Kaprio, 2000; Ge, Jin, et al., 2006), particularly in girls. While early timing is related to earlier onset of cigarette use (Wilson et al., 1994), there is limited research that examines whether this association differs by race. Black girls enter puberty earlier than White girls (Biro et al., 2006; Chumlea et al., 2003), which may put them at higher risk for substance use. However, racial disparities are noted in tobacco use with White adolescents initiating at an earlier age and having a

higher prevalence than Blacks (Geronimus, Neidert, & Bound, 1993; Johnston, O'Malley, Bachman, & Schulenberg, 2009). Thus, it is not clear whether the associations between pubertal timing and smoking are the same for White and Black females.

Cigarette smoking continues to be a significant public health problem and is the most preventable cause of death in the United States. The earlier smoking is initiated, the greater the risk of developing smoking-related cancer. Women face increased health risks from smoking compared with men. Approximately 178,000 women died from smoking-related diseases each year (1995–1999), dying an average of 14.5 years earlier than non-smokers (Centers for Disease Control, 2002). Adolescents are also at risk for smoking-related health issues, such as respiratory problems and early atherosclerotic lesions (a risk for cardiovascular disease; Elders, 1997; Prokhorov, Emmons, Pallonen, & Tsoh, 1996). Furthermore, a significant number of adolescents smoke. In 2007, 20% of high school students in a nationwide survey reported smoking in the past 30 days (Eaton et al., 2008).

Adolescence is a crucial period for considering the implications of smoking initiation. Of adult smokers, 80%–90% began smoking in adolescence (Kessler et al., 1996). Early smoking is not only associated with continued cigarette use but also associated with increased risk of future drug use and other substance use disorders (Lewinsohn, Rohde, & Brown, 1999). Typically, cigarette use does not peak until later adolescence (Jackson, Sher, Cooper, & Wood, 2002); however, early involvement, especially use by age 11–13 years, has been found to predict substance use and abuse in later adolescence and adulthood (Sung, Erkanli, Angold, & Costello, 2004). Thus, identifying factors associated with early substance use is important for understanding how initiation begins and may be prevented (Foshee et al., 2007).

Pubertal timing is one factor found to affect the initiation and prolonged use of substances. In particular, there is a strong association between early pubertal timing and early use of cigarettes for men and women (Bratberg, Nilsen, Holmen, & Vatten, 2007; Dick et al., 2000; Wilson et al., 1994). Adolescents with

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early pubertal timing were twice as likely to try cigarettes compared with those who were not early maturers (Westling, Andrews, Hampson, & Peterson, 2008). Similarly, early maturing boys and girls had significantly greater likelihood and higher prevalence of smoking compared with average and late-developing peers (Simon, Wardle, Jarvis, Steggle, & Cartwright, 2003; van Jaarsveld, Fidler, Simon, & Wardle, 2007). Within women, early developers were 2.5 times more likely to have tried cigarettes (Lanza & Collins, 2002) or smoked their first cigarette about 7 months earlier than girls with later puberty (Wilson et al.). Similarly, Tschann et al. (1994) found that 34% of early maturers initiated smoking at younger than 12 years of age. Thus, because girls are beginning to smoke at younger ages and more go on to be regular smokers (Galanti, Rosendahl, Post, & Gilljam, 2001; U.S. Department of Health and Human Services, 2001), particular attention should be paid to the factors associated with early initiation of smoking in adolescent females.

Race is also associated with variation in cigarette use. Blacks have lower cigarette initiation rates than Whites (Johnson & Hoffmann, 2000), and the age of smoking onset is significantly later in Blacks than Whites (Geronimus et al., 1993; Harrell, Bangdiwala, Deng, Webb, & Bradley, 1998). Additionally, risk factors for smoking (e.g., family and peer cigarette use, perceived availability of cigarettes) were less prevalent among Black than among White youth, while regular smoking was more prevalent among White than among Black adolescents (Robinson & Klesges, 1997). Although Blacks are at lower risk for cigarette use in adolescence, by adulthood, the smoking prevalence rates are comparable with Whites, yet Blacks have higher lung cancer rates (U.S. Department of Health and Human Services, 1998).

Although there is extensive evidence that early pubertal timing is related to early cigarette use and higher rates of smoking, no studies have examined this association for Black adolescents. Based on the literature, it was expected that early pubertal timing would be associated with earlier age at first cigarette for White adolescents; however, there is little empirical guidance regarding whether this association would be the same for Black girls because there are no studies that have compared pubertal timing effects on smoking onset by race. Therefore, the purpose of this study was to determine (a) whether early pubertal timing effects on age at smoking onset existed for both White and Black girls and (b) whether the association between pubertal timing and age at smoking onset was moderated by race.

Methods

Participants

The participants were girls enrolled in a longitudinal study on smoking, mood, and its impact on bone and reproductive health in adolescent girls ($N = 264$). Participants were primarily White (62.1%) or Black (32.6%). The remaining 5.3% of the girls were biracial or other race/ethnicities and were combined with the Black group. Participants were recruited from an urban teen health center and the surrounding community. Data for the present study were from the first assessment, which took place from December 2003 to October 2007. Girls were enrolled in the study by age cohorts (11, 13, 15, and 17 years) based on the

cross-sequential design (Miyazaki & Raudenbush, 2000), and an eligibility questionnaire based on five previously defined levels of smoking experience ranging from “not even a puff” to daily smoking. Smoking categories were based on modifications to the methods of Mayhew, Flay, and Mott (2000) and consisted of (a) “Never-smoker,” defined as zero puffs of a cigarette across the lifetime; (b) one puff to two cigarettes in lifetime; (c) smoked 3–99 cigarettes or more than 100 but none in the last 30 days; (d) smoked more than 100 cigarettes in lifetime and 1–19 days of the last 30 days; and (e) smoked more than 100 cigarettes and 20–30 days of the last 30 days. Exclusion criteria were (a) pregnancy or breast feeding within the past 6 months, (b) primary amenorrhea (>16 years) or secondary amenorrhea (<6 cycles/year), (c) body mass index less than the first percentile or body weight greater than 300 pounds, (d) medication/medical disorder influencing bone health, and (e) psychological disabilities impairing comprehension or compliance. This study received approval from the hospital’s institutional review board.

Procedures

Participants came to an urban children’s hospital for study visits. After consent and assent were obtained, the parent was directed to a separate room to complete questionnaires. The adolescent then had a physical examination, during which time, a standardized interview was conducted focusing on menstrual history. Other procedures followed but were not the focus of this paper. The protocol also included the Diagnostic Interview Schedule for Children, which assessed cigarette use.

Measures

Pubertal timing

Age at menarche (in years and months) was obtained through a clinician interview with the adolescent. Early, on-time, and late timing groups were created based on the sample distribution of age at menarche within White and Black racial groups in this study. Plus and minus 1 *SD* away from the mean is typically used as cutoffs for timing groups (Ge, Brody, Conger, Simons, & McBride-Murray, 2006). Thus, girls who were 1 *SD* or more below the mean (within their racial group) were coded as early timing, and those who were 1 *SD* or more above the mean were coded as late timing. Remaining girls were coded as on-time.

At Time 1, 210 (80%) of the sample were menarcheal. For the remaining 54 premenarcheal girls, age at menarche was obtained from a subsequent data collection timepoint. Six premenarcheal girls withdrew before the Year 2 visit and thus could not be assigned to a timing group. At Year 4, there were three girls who had not reached menarche. However, based on our criterion and their chronological age, they were placed in the late timing group.

Age at first cigarette

Age at first cigarette was defined as “how old were you the first time you smoked a cigarette?” that was entered in years. This was obtained from the Diagnostic Interview Schedule for Children version VI (DISC; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). The DISC is a computerized structured diagnostic interview to assess mental health symptoms and diagnoses.

Covariates

The covariates (socioeconomic status [SES], age, friend smoking, and parental smoking) were chosen based on evidence that these variables are associated with the dependent variable (age at smoking initiation). The Hollingshead scale was used as an index of SES (Hollingshead, 1976) with higher scores indicating higher SES. Parent smoking was assessed by the question “are there any parents or step-parents who live in the home that smoke” (0 = *no* and 1 = *yes*). Friends’ smoking was assessed by the girls’ report of whether they had one or more close friends who were regular smokers (0 = *no* and 1 = *yes*).

Data analysis

To examine group differences in age at smoking onset separately by pubertal timing and racial group, analysis of covariance (ANCOVA) was conducted. Age at first cigarette was entered as the dependent variable, and racial group was entered as an independent variable. Covariates included age, SES, parental smoking, and friend smoking. Next, pubertal timing group was entered as the independent variable with the same dependent variable and covariates as the previous analysis with the inclusion of race.

The primary aims were conducted using survival analysis, which estimates the time to a terminal event (“smoking onset”) and the proportion “surviving” within each event period. This analytic technique is used when a large proportion of the participants have not yet had the “event” occur during the data collection period. Thus, using traditional methods such as regression would bias the results by excluding all those who have not initiated the event in question (Singer & Willett, 1991). Because of the nature of the dependent variable (age at smoking initiation), there is inherently a large number of individuals who do not have information on this variable (referred to as censored observations). However, survival analysis is designed to deal with censored observations, which would be problematic in most other statistical approaches. To examine pubertal timing effects on smoking onset for White and Black girls, Kaplan–Meier analysis was used with pubertal timing group as the between groups factor and stratified by race (White vs. Black). This analysis produces median ages for age at first cigarette by timing group and race. The log-rank test was used to examine differences in the survival curves for each pubertal tim-

ing group stratified by race. To examine racial differences in survival curves within pubertal timing groups, Kaplan–Meier analysis was used with racial group as the between-groups factor, stratified by pubertal timing group.

Cox regression was used to examine whether the association between pubertal timing and smoking onset was moderated by race. The covariates were entered in the first block, pubertal timing and race entered in the second block, and the interaction term entered in the third block. A significant chi-square change indicated whether the interaction term accounted for significant variance in the model above that of the constituent variables.

Results

Descriptive statistics

Descriptive statistics appear in Table 1. There were 153 girls who reported smoking and thus also reported age at first cigarette. Of those who reported an age at first cigarette, the mean age was 12.24 years ($SD = 2.56$ years) and the median age was 13 years. Key variables were examined within pubertal timing and race groups adjusting for stated covariates. ANCOVA revealed no significant difference between White (adjusted $M = 12.20$) and Black (adjusted $M = 12.67$) groups on the mean age of smoking initiation, $F(1, 138) = 1.16, p = .28$. For timing group, post-hoc pairwise comparisons indicated a mean difference between the early (adjusted $M = 11.38$) and late (adjusted $M = 13.83$) timing groups ($p = .00$) and the on-time (adjusted $M = 12.22$) and late groups ($p = .00$) but no difference between the early and on-time groups ($p = .13$).

Survival curves by racial group and pubertal timing group

For the racial groups, 42.4% of the White group and 41.4% of the Black group were censored in their age of onset of smoking (i.e., those who had not started smoking). The median survival time was 14 years for both racial groups (95% $CI = 13.49$ – 14.51 years for White group and 13.32 – 14.68 years for Black group). For the pubertal timing groups, 35.9% of the early, 43.4% of the on-time, and 37.8% of the late groups were censored in their age of onset of smoking. The median survival times were 14 years (95% $CI = 12.67$ – 15.33) for the early group,

Table 1. Descriptive statistics by pubertal timing and racial group in 264 adolescent girls

	Pubertal timing group			Racial group	
	Early	On-time	Late	White	Black
<i>n</i>	39	175	45	164	100
Mean age in years (<i>SD</i>)	15.27 (2.06)	14.79 (2.16)	15.59 (2.09)	14.82 (2.23)	15.12 (2.10)
Mean SES (<i>SD</i>)	37.5 (13.0)	36.71 (14.11)	40.02 (11.34)	40.71 (13.59)	31.86 (11.86)
Mean age at menarche in years (<i>SD</i>)	10.56 (0.93)	12.39 (0.70)	14.17 (0.53)	12.64 (1.11)	11.99 (1.34)
Percent White	64.1	60.6	68.9		
Percent that have never smoked	35.9	43.4	37.8	42.4	41.4
Percent that had friends who smoke	35.9	34.3	44.4	37	34.3
Percent that had parents who smoke	43.6	45.7	44.4	40.9	53

Note. SES = socioeconomic status. For the White group, the cutoffs for the early and late groups were 11.52 and 13.74 years, respectively, whereas for the Black group, the cutoffs were 10.67 and 13.34 years, respectively.

14 years (95% CI = 13.46–14.54) for the on-time group, and 15 years (95% CI = 13.89–16.11) for the late group (see Figure 1). The log-rank test was used to examine differences in the survival curves by pubertal timing group stratified by race. Within the White group, the median age at smoking onset was 14 years (95% CI = 12.88–15.12) for the early maturers, 13 years (95% CI = 12.37–13.63) for the on-time, and 14 years (95% CI = 12.44–15.57) for the late timing groups. Pairwise comparisons showed no significant differences between pubertal timing groups. For the Black group, the median age at smoking onset was 12 years (95% CI = 10.59–13.41) for the early maturers, 14 years (95% CI = 13.39–14.61) for the on-time, and 15 years (95% CI = 13.98–16.02) for the late timing group. Pairwise comparisons within the Black group showed that early maturers had the youngest age at smoking onset compared with on-time ($\chi^2 = 4.50, p = .03$) and late maturers ($\chi^2 = 6.42, p = .01$). Kaplan–Meier survival estimates were also computed between racial groups stratified by pubertal timing group, but there were no significant differences, indicating that within each timing group (early, on-time, and late), the White and Black girls were not significantly different in the age at smoking onset. Most importantly, within the early pubertal timing group, the White (median age = 14, 95% CI = 12.88–15.12) and Black (median age = 12, 95% CI = 10.59–13.41) groups were not significantly different in the age at smoking onset ($p = .13$).

Interaction effect between racial group and pubertal timing on age at first cigarette

Using on-time puberty and Black group as the reference variables, there were no significant interactions (controlling for age, SES, friend smoking, and parental smoking). Therefore, the interaction terms were dropped, and the main effect of pubertal timing was examined. Racial group, age, SES, parental smoking, and friend smoking were entered in the first block, followed by pubertal timing group in the second block. The block in which pubertal timing was added improved the overall fit of the model ($\Delta\chi^2(2) = 6.67, p = .04$) above the fit of the model with only the first block of variables ($\chi^2(7) = 40.42, p = .00$). Several of the covariates were significant: higher SES reduced the risk of smoking onset (hazard ratio [HR] = 0.99, 95% CI = .97–1.00), whereas parent and friend smoking increased the risk by 1.63 (95% CI = 1.11–2.39) and two times (95% CI = 1.33–3.01), respectively. However, racial group did not significantly increase the risk of smoking initiation (HR = 1.37, 95% CI = 0.94–1.96). Of primary interest was the significant effect of pubertal timing group. Being early or on-time increased the hazard of smoking initiation in the next year by 1.94 (95% CI = 1.10–3.43) and 1.62 (95% CI = 1.04–2.57) times, respectively, over those in the late pubertal timing group. However, the risk for smoking initiation was not significantly different for early and on-time groups

Figure 1. Kaplan–Meier estimates of the number of participants who will experience the terminal event (smoking initiation) by the next event period. Analyses completed by pubertal timing group for (a) White and (b) Black groups showed significant differences between pubertal timing groups only for the Black group. Within Black girls, early maturers were significantly younger at first cigarette use than on-time or late maturers, as shown by the distinct lines for each group with very little overlap. Censored data refer to those who have not experienced the event (smoking).

(HR = 1.19, 95% CI = 0.75–1.89). The results showed that after adjusting for the covariates, there was no significant interaction effect between racial group and pubertal timing on smoking initiation, although there appeared to be racial group differences in the Kaplan–Meier analysis. There was a main effect of pubertal timing for the total sample showing that late timing was associated with the lowest risk for smoking initiation (Figure 2).

Discussion

The present study examined the association between pubertal timing and age at first cigarette in a large sample of White and Black adolescent females. When Black and White groups were examined separately, early pubertal timing was associated with earlier age of smoking initiation in Black girls only. This was counter to expectations as most of the literature focuses on White samples and the findings between early timing and substance use are well supported (Dick et al., 2000; Lanza & Collins, 2002; Westling et al., 2008). However, when race was tested as a moderator, there was not a significant interaction between racial group and pubertal timing, but there was a main effect of pubertal timing. Most studies have found that only early timing is associated with increased substance use. For example, girls with early puberty reported a younger age of first cigarette than those with later puberty (Wilson et al., 1994) and were 1.5 times more likely to smoke (Bratberg, Nilsen, Holmen, & Vatten, 2005). However, we found that early and on-time groups were at increased risk for early smoking initiation compared with the late timing group.

Although previous research has demonstrated that White adolescents initiate smoking earlier, smoke more, and progress to becoming regular smokers more rapidly than Black adolescents (Harrell et al., 1998; Kandel, Kiros, Schaffran, & Hu, 2004), our study shows that a racial disparity may not exist when assessing the effect of pubertal timing on smoking behavior. Late timing for both Whites and Blacks seems to be protective against early smoking initiation. Smoking behavior in adolescents, and especially early age at initiation, is particularly

important when examining long-term smoking and health consequences. Youth who begin smoking in lower grades are more likely to be adult smokers (Chassin, Presson, Sherman, & Edwards, 1990). Additionally, age of smoking initiation is a significant factor for failure in cessation attempts (Khuder, Dayal, & Mutgi, 1999). These findings point to targeting early and on-time maturers for smoking prevention programs in adolescence. Furthermore, because pubertal timing is occurring earlier for contemporary adolescents than in the past, targeting early and on-time maturers at younger ages may be important.

There are several possible explanations for the differences between our results and other studies. First, variability in how pubertal timing was determined across the studies may yield differences in timing classifications. We used age at menarche, which is an event later in puberty, whereas other studies used ratings of secondary sexual characteristics (e.g., breast and pubic hair development). Although menarche and secondary sexual characteristics are associated, the data suggest only a moderate correlation between the two, thus introducing a potential source of error (Biro et al., 2006). Second, the majority of the participants were in mid- to late adolescence when it becomes more likely that they will have tried smoking. Several studies have found evidence of a “catch-up” effect when assessing older adolescents. Specifically, late maturing females between 16 and 18.5 years caught up to their early and on-time maturing peers in their alcohol use (Dick et al., 2000). Thus, in early adolescence, early timing may be important in predicting substance use, while later in adolescence, girls with on-time development may be using substances as much as the early maturers. It is also possible that in late adolescence, pubertal timing effects may be negligible (Stattin & Magnusson, 1990) with the host of other influences that may be more salient for substance use. However, the age at onset of cigarette use is still important as it influences prolonged substance use into adulthood (Faulkner, Escobedo, Zhu, Chrismon, & Merritt, 1996; Grant & Dawson, 1997).

One limitation of this study may be that our dependent variable was age of first cigarette, which may not necessarily

Figure 2. Survival functions for age at first cigarette by pubertal timing group via Cox regression. Analysis accounted for the effects of age, race, SES, parent smoking, and friend smoking. The early and on-time groups are not significantly different from each other, but both are significantly different from the late group. This is indicated in the graph by the overlapping survival curves for the early and on-time group but the distinct curve for the late group.

reflect continued smoking. An individual may try a cigarette but then not smoke ever again. However, in the present sample, 68% of those who reported an age at first cigarette have continued to smoke. Thus, like other studies (Lewinsohn et al., 1999; Sung et al., 2004), we find that age at first cigarette is an important consideration when assessing future smoking, and intervening early is essential for prevention of long-term health problems.

A second limitation is that self-report was used for age at menarche and age at first cigarette. Although it is impossible to check the veracity of the reported ages, it is probable that for these events, the adolescent is the most accurate reporter. When our study is completed, we will be able to assess the reliability of reports across time for the age of smoking initiation and age at menarche. The number of girls who had not yet smoked might be viewed as a limitation; however, survival analysis accommodates large amounts of censored data, utilizing the maximum amount of available information from censored data for analysis (Singer & Willett, 1991).

An additional limitation may be the sample size for this study. Due to the nature of the pubertal timing variable, there will inherently be more participants in the on-time group than in the early or late groups. However, many studies have used this variable with similar distributions and sample sizes to the current study, thus our confidence is increased. Additionally, because this was a secondary data analysis, we may have limited statistical power, particularly in examining interaction terms. The nonsignificant interaction effect may be due to difficulties with unequal cell sizes and creating categorical variables from continuous ones as well as lack of power (Aguinis, Boik, & Pierce, 2001; Frazier, Tix, & Barron, 2004). However, we feel confident that the cell sizes are adequate for the analyses, and the findings are valid and show some novel associations between pubertal timing, smoking onset, and race.

Other factors that were not measured in this study can influence smoking behavior, such as early dating and parental monitoring (Fidler, West, Jarvis, & Wardle, 2006; Westling et al., 2008). However, the inclusion of parent and friends smoking did account for two large influences on smoking. Other studies found that friend smoking was related to smoking initiation among Whites but not Blacks (Headen, Bauman, Deane, & Koch, 1991) and parent smoking contributed to the onset of daily smoking in their teenagers (Hill, Hawkins, Catalano, Abbott, & Guo, 2005). In the present study, parent smoking was related to age at first cigarette with having a parent who smokes increasing the risk by 1.6 times. Thus, to reduce the risk for daily smoking in adolescents, it may be important to encourage parents to stop or reduce smoking.

Cross-sectional studies have inherent limitations in being unable to unravel causal pathways. Additionally, because we have not observed all the participants through the period of highest risk for smoking initiation, we lose some information about those defined as censored. Some of the girls who enrolled as nonsmokers at the time of the assessment may eventually initiate smoking, but we have no way of knowing when that critical age may be for every individual. Several studies have examined smoking initiation data using survival analysis, and they provide useful information about the risks (Andreeva, Krasovsky, & Semenova, 2007; Chen, Unger, & Johnson, 1999). One study found that after age 14, the risk of new smoking initiation

decreases (Unger & Chen, 1999). The majority of the sample is older than 14 years, and thus, we may be more certain that we are capturing the accurate risk model for smoking initiation. In the future, we can examine the smoking onset age for all girls up to age 17 and assess the reliability of age at smoking onset from the subsequent yearly visits.

Overall, our results indicate that early and on-time pubertal development increases the risk for early smoking onset in adolescent girls. Contrary to expectations, we did not find racial differences in this association, indicating that late timing may be protective for both White and Black girls. Our study shows that a racial disparity may not exist when assessing the effect of pubertal timing on smoking behavior for White and Black adolescents. However, possible differences should be examined for other racial minority groups. Additionally, this result provides evidence for the importance of assessing puberty in studies of substance use. While there are clearly multiple factors that contribute to the etiology and maintenance of substance use, we show that omitting puberty is overlooking a substantial influence on early substance use. Future models should aim to incorporate these numerous influences, including a reliable measure of puberty so that we can better understand these complex relationships and work toward the prevention of prolonged substance use.

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Declaration of Interests

None declared.

References

- Aguinis, H., Boik, R. J., & Pierce, C. A. (2001). A generalized solution for approximating the power to detect effects of categorical moderator variables using multiple regression. *Organizational Research Methods*, 4, 291–323.
- Andreeva, T. I., Krasovsky, K. S., & Semenova, D. S. (2007). Correlates of smoking initiation among young adults in Ukraine: A cross-sectional study. *BMC Public Health*, 7, 106.
- Biro, F. M., Huang, B., Crawford, P. B., Lucky, A. W., Striegel-Moore, R., Barton, B. A., et al. (2006). Pubertal correlates in black and white girls. *Journal of Pediatrics*, 148, 234–240.
- Bratberg, G. H., Nilsen, T. I., Holmen, T. L., & Vatten, L. J. (2005). Sexual maturation in early adolescence and alcohol drinking and cigarette smoking in late adolescence: A prospective study of 2,129 Norwegian girls and boys. *European Journal of Pediatrics*, 164, 621–625.
- Bratberg, G. H., Nilsen, T. I., Holmen, T. L., & Vatten, L. J. (2007). Perceived pubertal timing, pubertal status and the

prevalence of alcohol drinking and cigarette smoking in early and late adolescence: A population based study of 8950 Norwegian boys and girls. *Acta Paediatrica*, 96, 292–295.

Centers for Disease Control. (2002). Annual smoking-attributable mortality, years of potential life lost, and economic costs—United States, 1995–1999. *MMWR. Morbidity and Mortality Weekly Report*, 51, 300–303.

Chassin, L., Presson, C. C., Sherman, S. J., & Edwards, D. A. (1990). The natural history of cigarette smoking: Predicting young-adult smoking outcomes from adolescent smoking patterns. *Health Psychology*, 9, 701–716.

Chen, X., Unger, J. B., & Johnson, C. A. (1999). Is acculturation a risk factor for early smoking initiation among Chinese American minors? A comparative perspective. *Tobacco Control*, 8, 402–410.

Chumlea, W. C., Schubert, C. M., Roche, A. F., Kulin, H. E., Lee, P. A., Himes, J. H., et al. (2003). Age at menarche and racial comparisons in U.S. girls. *Pediatrics*, 111, 110–113.

Dick, D. M., Rose, R. J., Viken, R. J., & Kaprio, J. (2000). Pubertal timing and substance use: Associations between and within families across late adolescence. *Developmental Psychology*, 36, 180–189.

Eaton, D. K., Kann, L., Kinchen, S., Shanklin, S., Ross, J., Hawkins, J., et al. (2008). Youth risk behavior surveillance—United States, 2007. *MMWR. Morbidity and Mortality Weekly Report Surveillance Summaries*, 57, 1–131.

Elders, J. (1997). *Preventing tobacco use among young people: A report of the Surgeon General*. Darby, PA: Diane Publishing.

Faulkner, D. L., Escobedo, L. G., Zhu, B. P., Chrismon, J. H., & Merritt, R. K. (1996). Race and the incidence of cigarette smoking among adolescents in the United States. *Journal of the National Cancer Institute*, 88, 1158–1160.

Fidler, J. A., West, R., Jarvis, M. J., & Wardle, J. (2006). Early dating predicts smoking during adolescence: A prospective study. *Addiction*, 101, 1805–1813.

Foshee, V. A., Ennett, S. T., Bauman, K. E., Granger, D. A., Benefield, T., Suchindran, C., et al. (2007). A test of biosocial models of adolescent cigarette and alcohol involvement. *Journal of Early Adolescence*, 27, 4–39.

Frazier, P. A., Tix, A. P., & Barron, K. E. (2004). Testing moderator and mediator effects in counseling psychology. *Journal of Counseling Psychology*, 51, 115–134.

Galanti, M. R., Rosendahl, I., Post, A., & Gilljam, H. (2001). Early gender differences in adolescent tobacco use—The experience of a Swedish cohort. *Scandinavian Journal of Public Health*, 29, 314–317.

Ge, X., Brody, G. H., Conger, R. D., Simons, R. L., & McBride-Murray, V. (2006). Pubertal maturation and African American children's internalizing and externalizing symptoms. *Journal of Youth and Adolescence*, 35, 531–540.

Ge, X., Jin, R., Natsuaki, M. N., Gibbons, F. X., Brody, G. H., Cutrona, C. E., et al. (2006). Pubertal maturation and early substance use risks among African American children. *Psychology of Addictive Behaviors*, 20, 404–414.

Geronimus, A. T., Neidert, L. J., & Bound, J. (1993). Age patterns of smoking in US black and white women of childbearing age. *American Journal of Public Health*, 83, 1258–1264.

Grant, B. F., & Dawson, D. A. (1997). Age at onset of alcohol use and its association with DSM-IV alcohol abuse and dependence: Results from the national longitudinal alcohol epidemiologic survey. *Journal of Substance Abuse*, 9, 103–110.

Harrell, J. S., Bangdiwala, S. I., Deng, S., Webb, J. P., & Bradley, C. (1998). Smoking initiation in youth: The roles of gender, race, socioeconomic, and developmental status. *Journal of Adolescent Health*, 23, 271–279.

Headen, S. W., Bauman, K. E., Deane, G. D., & Koch, G. G. (1991). Are the correlates of cigarette smoking initiation different for Black and White adolescents? *American Journal of Public Health*, 81, 854–858.

Hill, K. G., Hawkins, J. D., Catalano, R. F., Abbott, R. D., & Guo, J. (2005). Family influences on the risk of daily smoking initiation. *Journal of Adolescent Health*, 37, 202–210.

Hollingshead, A. F. (1976). *Four factor index of social status: Manual*. New Haven, CT: Department of Sociology, Yale University.

Jackson, K. M., Sher, K. J., Cooper, M. L., & Wood, P. K. (2002). Adolescent alcohol and tobacco use: Onset, persistence and trajectories of use across two samples. *Addiction*, 97, 517–531.

Johnson, R. A., & Hoffmann, J. P. (2000). Adolescent cigarette smoking in U.S. racial/ethnic subgroups: Findings from the national education longitudinal study. *Journal of Health and Social Behavior*, 41, 392–407.

Johnston, L. D., O'Malley, P. M., Bachman, J. G., & Schulenberg, J. E. (2009). *Monitoring the future national survey results on drug use, 1975–2008. Volume 1: Secondary school students*. Bethesda, MD: National Institute on Drug Abuse.

Kandel, D. B., Kiros, G. E., Schaffran, C., & Hu, M. C. (2004). Racial/ethnic differences in cigarette smoking initiation and progression to daily smoking: A multilevel analysis. *American Journal of Public Health*, 94, 128–135.

Kessler, D. A., Witt, A. M., Barnett, P. S., Zeller, M. R., Natanblut, S. L., Wilkenfeld, J. P., et al. (1996). The Food and Drug Administration's regulation of tobacco products. *New England Journal of Medicine*, 335, 988–994.

Khuder, S. A., Dayal, H. H., & Mutgi, A. B. (1999). Age at smoking onset and its effect on smoking cessation. *Addictive Behaviors*, 24, 673–677.

Lanza, S. T., & Collins, L. M. (2002). Pubertal timing and the onset of substance use in females during early adolescence. *Prevention Science*, 3, 69–82.

- Lewinsohn, P. M., Rohde, P., & Brown, R. A. (1999). Level of current and past adolescent cigarette smoking as predictors of future substance use disorders in young adulthood. *Addiction*, 94, 913–921.
- Mayhew, K. P., Flay, B. R., & Mott, J. A. (2000). Stages in the development of adolescent smoking. *Drug and Alcohol Dependence*, 59, S61–S81.
- Miyazaki, Y., & Raudenbush, S. W. (2000). Tests for linkage of multiple cohorts in an accelerated longitudinal design. *Psychological Methods*, 5, 44–63.
- Prokhorov, A. V., Emmons, K. M., Pallonen, U. E., & Tsoh, J. Y. (1996). Respiratory response to cigarette smoking among adolescent smokers: A pilot study. *Preventive Medicine*, 25, 633–640.
- Robinson, L. A., & Klesges, R. C. (1997). Ethnic and gender differences in risk factors for smoking onset. *Health Psychology*, 16, 499–505.
- Shaffer, D., Fisher, P., Lucas, C. P., Dulcan, M. K., & Schwab-Stone, M. E. (2000). NIMH Diagnostic Interview Schedule for Children, version IV (NIMH DISC-IV): Description, differences from previous versions, and reliability of some common diagnoses. *Journal of the American Academy of Child & Adolescent Psychiatry*, 39, 28–38.
- Simon, A. E., Wardle, J., Jarvis, M. J., Steggles, N., & Cartwright, M. (2003). Examining the relationship between pubertal stage, adolescent health behaviours and stress. *Psychological Medicine*, 33, 1369–1379.
- Singer, J. D., & Willett, J. B. (1991). Modeling the days of our lives: Using survival analysis when designing and analyzing longitudinal studies of duration and the timing of events. *Psychological Bulletin*, 110, 268–290.
- Stattin, H., & Magnusson, D. (1990). *Paths through life-Vol. 2: Pubertal maturation in female development*. Hillsdale, NJ: Lawrence Erlbaum Associates.
- Sung, M., Erkanli, A., Angold, A., & Costello, E. J. (2004). Effects of age at first substance use and psychiatric comorbidity on the development of substance use disorders. *Drug and Alcohol Dependence*, 75, 287–299.
- Tschann, J. M., Adler, N. E., Irwin, C. E., Millstein, S. G., Turner, R. A., & Kegeles, S. M. (1994). Initiation of substance use in early adolescence: The roles of pubertal timing and emotional distress. *Health Psychology*, 13, 326–333.
- U.S. Department of Health and Human Services. (1998). *Tobacco use among U.S. racial/ethnic minority groups: A report of the Surgeon General*. Washington, DC: Centers for Disease Prevention and Control.
- U.S. Department of Health and Human Services. (2001). *Women and smoking: A report of the Surgeon General*. Atlanta, GA: Centers for Disease Control.
- Unger, J. B., & Chen, X. (1999). The role of social networks and media receptivity in predicting age of smoking initiation: A proportional hazards model of risk and protective factors. *Addictive Behaviors*, 24, 371–381.
- van Jaarsveld, C. H. M., Fidler, J. A., Simon, A. E., & Wardle, J. (2007). Persistent impact of pubertal timing on trends in smoking, food choice, activity, and stress in adolescence. *Psychosomatic Medicine*, 69, 798–806.
- Westling, E., Andrews, J. A., Hampson, S. E., & Peterson, M. (2008). Pubertal timing and substance use: The effects of gender, parental monitoring and deviant peers. *Journal of Adolescent Health*, 42, 555–563.
- Wilson, D. M., Killen, J. D., Hayward, C., Robinson, T. N., Hammer, L. D., Kraemer, H. C., et al. (1994). Timing and rate of sexual maturation and the onset of cigarette and alcohol use among teenage girls. *Archives of Pediatrics and Adolescent Medicine*, 148, 789–795.