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Trajectories of Cigarette Smoking Beginning in Adolescence Predict Insomnia in the Mid Thirties

Jung Yeon Lee, Ph.D.[1], **Judith S. Brook, Ed.D.**[1], **Stephen J. Finch, Ph.D.**[2], and **David W. Brook, M.D.**[1]

[1]New York University School of Medicine, Department of Psychiatry

[2]Stony Brook University, Department of Applied Mathematics and Statistics

Abstract

Background—Insomnia is increasingly recognized as a public health concern in modern society. Insomnia diagnoses appear to be increasing and are associated with poor health outcomes. They may cost $100 billion annually in health services.

Objective—Given the adverse consequences of insomnia such as cardiovascular disease, diabetes, and depression, the present study was designed to examine the relationship of the trajectories of earlier cigarette smoking and later insomnia. The ultimate goal is to reduce the prevalence of insomnia.

Methods—674 participants (53% African Americans, 47% Puerto Ricans, 60% females) were surveyed at 6 points in time. We employed the growth mixture model to obtain the trajectories of cigarette smoking from age 14 to 32. We used logistic regression analyses to examine the associations between the trajectories of smoking and insomnia.

Results—Males were less likely to have insomnia than females (Adjusted odds ratio: AOR=0.34, $p<.05$). A higher Bayesian posterior probability (BPP) for the chronic smoking trajectory group (AOR=2.69, $p<.05$) and for the moderate smoking trajectory group (AOR=5.33, $p<.01$) was associated with an increased likelihood of having insomnia at age 36 compared with the BPP of the no or low smoking trajectory group.

Conclusions—Prevention and treatment programs for individuals who suffer from insomnia should be implemented in parallel with programs for smoking cessation. From a public health perspective, our longitudinal study that examined the association between earlier smoking trajectories and later insomnia suggests that treatments designed to reduce or cease smoking may lessen the occurrence of symptoms of insomnia.

Keywords

Longitudinal study; insomnia; trajectory analysis; cigarette use; ethnic minority youth

Corresponding author: Dr. Judith S. Brook, Professor, Department of Psychiatry, New York University School of Medicine, 215 Lexington Avenue, 15th Floor, New York, NY 10016, U.S.A. Tel: 212-263-4663; Fax: 212-263-4660; judith.brook@nyumc.org.

DECLARATION OF INTERESTS

None declared.

INTRODUCTION

Insomnia is characterized by a spectrum of complaints reflecting dissatisfaction with the quality, duration, or continuity of sleep. The predominant nocturnal symptoms include difficulties falling asleep at bedtime, waking up in the middle of the night and having difficulty going back to sleep, or waking up too early in the morning with an inability to return to sleep. In addition to the above criteria, a diagnosis of insomnia, based on the Diagnostic and Statistical Manual of Mental Disorders-5 (DSM-5), requires the presence of symptoms that do not result from another disorder (American Psychiatric Association, 2013). Insomnia is increasingly recognized as a public health concern in modern society. In the United States, insomnia diagnoses appear to be increasing and are associated with poor health outcomes (Bains, 2006; Namen et al., 2002; Taylor et al., 2007). A recent study conducted in the United Kingdom reported modest increases in the prevalence of insomnia symptoms and diagnoses over their survey periods from 35.0% and 3.1% in 1993 to 38.6% and 5.8% in 2007, respectively (Calem et al., 2012).

Insomnia is related to a wide range of adverse outcomes such as cardiovascular disease, increased body mass index (BMI), diabetes, anxiety, depression, poor interpersonal relationships, car accidents, frequent absenteeism and disability at work (Brenes et al., 2009; Chien et al., 2010; Lee et al., 2009; Leger, Massuel, Metlaine, & Group, 2006; Léger et al., 2010; Ohayon & Roth, 2003; Overland et al., 2008; Vgontzas, Liao, Bixler, Chrousos, & Vela-Bueno, 2009). In addition, a longitudinal study from the National Longitudinal Study of Adolescent Health found that sleep difficulties at earlier waves significantly predicted the use of substances as well as substance-related problems at later waves (Wong, Robertson, & Dyson, 2015). Another longitudinal study found bidirectional relationships between sleep problems and substance use in young adolescents (Pieters et al., 2015). Given the adverse consequences of insomnia, the present study was designed to examine the predictors of insomnia to increase understanding of its genesis. Our ultimate goal is to contribute to a reduction in the prevalence of insomnia.

A number of studies based on self-report data suggest that cigarette smoking plays a role in arousal, sleep disturbance or difficulty, and insomnia (D. W. Brook, Rubenstone, Zhang, & Brook, 2012; Soldatos, Kales, Scharf, Bixler, & Kales, 1980; Wetter & Young, 1994). Most studies (Sahlin, Franklin, Stenlund, & Lindberg, 2009; Soldatos et al., 1980; Wetter & Young, 1994) have used a cross-sectional design. However, Brook and colleagues (D. W. Brook et al., 2012) using data from a 20 year longitudinal study of women in mid-life, found that women with a longitudinal pattern of chronic heavy smoking as compared to non-smokers had an increased likelihood of insomnia.

In addition, physiological research has supported the evidence for the effects of cigarette smoking on sleep by explaining the mechanisms involved. Research has shown that nicotine affects the neurotransmitter systems which may lead to arousal as well as to inhibition of sleep-promoting neurons (Htoo, Talwar, Feinsilver, & Greenberg, 2004; Saint-Mleux et al., 2004). Brain waves also appeared to be different in smokers and non-smokers during sleep. Specifically, increases in alpha frequency waves were found among smokers as compared to non-smokers, suggesting greater arousal (Domino et al., 2009; Zhang, Samet, Caffo,

Bankman, & Punjabi, 2008). In the Sleep Heart Health Study consisting of 6,400 participants, sleep architecture was characterized. Compared with never smokers, current smokers had a longer initial sleep latency, less total sleep time, and more stage 1 sleep (Zhang, Samet, Caffo, & Punjabi, 2006).

Gender, ethnicity, depressive symptoms, age, educational level, partner status, and BMI are also related to insomnia. Women report more frequent symptoms of insomnia than men (Jaussent et al., 2011; Yoshioka et al., 2012). As regards ethnicity, Blacks as compared to Hispanics reported higher odds of insomnia using the diagnostic criteria from the International Classification of Diseases (Roth et al., 2011). In clinical samples, about three quarters of all depressed patients complain of difficulty either in initiation or in maintaining sleep (Tsuno, Besset, & Ritchie, 2005; Yates et al., 2007). Age also has some effects on sleep. For example, linear regression analysis revealed that increasing age is associated with shorter sleep latencies and greater insomnia (Xian et al., 2014). Lower education (Kim et al., 2013; Talala, Martelin, Haukkala, Härkänen, & Prättälä, 2012) and obesity (Roth et al., 2011) were related to insomnia. Studies of impaired sleep show differences in partner status. Married individuals are less likely to report frequent insufficient sleep than never married individuals (Chapman et al., 2012). Therefore, we use these variables as controls in the current study.

Although the adverse effects of cigarette smoking on sleep are clearly documented, there is little research about long-term patterns of smoking and insomnia. The current longitudinal study is uniquely positioned to examine the effects of smoking trajectories beginning in adolescence into the mid thirties on symptoms of insomnia; we identify the trajectories of cigarette smoking as predictors of insomnia over several developmental stages spanning a 22 year period. We also assess the predictors of insomnia among relatively understudied ethnic groups of African Americans and Puerto Ricans living in an urban area. A better understanding of the effects of long-term patterns of smoking on insomnia may help to guide the treatment of smoking cessation programs and treatment programs focused on insomnia.

We hypothesize that: 1) Trajectory groups with higher levels of smoking (e.g., the chronic smoking trajectory group) as compared to the no or low smoking trajectory group will be associated with an increased likelihood of insomnia; and 2) The associations between the patterns of cigarette smoking and insomnia will be maintained after controlling for gender, ethnicity, depressive symptoms, age, educational level, partner status, and BMI.

METHODS

Participants

Sample of the current research consisted of ethnic minority groups of African Americans and Puerto Ricans living in an urban area. Questionnaires were completed by 674 participants (53% African Americans, 47% Puerto Ricans) for the sixth wave (time 6; T6) of this study. Sixty percent were females (n=405). Data on the participants were first collected in 1990 (time 1; T1) when the participants were students attending schools in the East Harlem area of New York City. At T1, the questionnaires were administered in classrooms under the supervision of the study research staff with no teachers present. At time 2 (T2), the

National Opinion Research Center located and interviewed the participants in person or by phone. At time 3 (T3), the Survey Research Center of the University of Michigan collected the data. At Time 4 (T4), Time 5 (T5), and T6, the data were collected by our research group. Table 1 shows time points of data collection, the years, the number of participants, and ages of the participants at each time point. More information regarding the sample description can be found in previous research (Blinded for review).

The Institutional Review Board (IRB) of the New York University School of Medicine approved the study for T4, T5, and T6. The IRBs of both the Mount Sinai School of Medicine and New York Medical College approved the study's procedures for data collections in the earlier waves. A Certificate of Confidentiality was obtained from the National Institute on Drug Abuse for T1-T4 and from the National Cancer Institute at T5 and at T6. At T1 and T2, passive consent was obtained from the parents of minors. At each time wave, we obtained informed assent or consent from each participant.

At T6, we attempted to follow-up all those who participated at T1. We compared the demographic variables for the 674 adults who participated at both T1 and T6 with the 658 who participated at T1 but not at T6. There were no significant differences between the T6 non-participants and the T6 participants in the proportion of African Americans and Puerto Ricans ($\chi^2_1 = 3.48$, p=0.06) and mean level of depressive symptoms at T1 (t_1=0.81, p=0.42). However, the percentage of males at T1 among T6 non-participants (53%) was higher than the percentage of males who participated at T6 (40%) ($\chi^2_1 = 26.06$, p <.001). The mean age at T1 among T6 non-participants (14.5) was higher than the mean age at T1 among T6 participants (14.0) (t_1=7.14, p<.001). The frequency of cigarette use at T1 among T6 non-participants (0.36) was higher than the frequency of cigarette use at T1 among T6 participants (0.20) (t_1=4.26, p<.001).

Measures

Control variables

a. Gender (1=female, 2=male)

b. Ethnicity (1=African American, 2=Puerto Rican)

c. Depressive symptoms (Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974) at T1 were assessed with a 2 item scale, i.e. "Do you sometimes feel unhappy, sad, or depressed?" and "Do you sometimes feel hopeless about the future?" using a 4-point Likert scale that ranged from "not at all" to "extremely." The inter-correlation between the 2 items was 0.47 (p<.001).

d. Age at T6

e. Educational level at T6 (0=11th grade or below, 1=12th grade or GED, 2=Business or technical school, 3=College freshman, 4=College sophomore or Associate's Degree, 5=College junior, 6=College senior or Bachelor's Degree, 7=Postgraduate business, law, medical, masters, or doctoral program)

f. Partner status at T6 (0=Single, separated, divorced, or widowed, 1=Cohabiting or married)

g. Body mass index at T6 [BMI= (Weight in pounds/Height in inches^2)×703] (A. Brook, 2006)

Cigarette use (T1-T5)—The participants reported on their cigarette smoking at each wave between T1 and T6. Participants were asked "How many cigarettes do you smoke?" at T1 and "How many cigarettes did you smoke in the past 5 years?" at T2 through T5. The answer options were 0=none, 1=a few cigarettes or less a week, 2=1–5 cigarettes a day, 3=about half a pack a day, 4=about 1 pack a day, 5=about 1 and half packs a day, and 6=more than 1 and half packs a day.

Insomnia at T6—Insomnia was assessed at T6 by using an adaptation of the Insomnia Severity Index measure (Bastien, Vallières, & Morin, 2001; Morin, 1993). Insomnia in this study included five of the seven Insomnia Severity Index criteria: 1) difficulty falling asleep, 2) difficulty staying asleep, 3) waking up too early in the morning, 4) distress caused by sleep difficulties, and 5) interference of sleep difficulties with daytime functioning. A 5-point Likert scale was used for each item (0=none, 1=mild, 2=moderate, 3=severe, 4=very severe), yielding a total score ranging from 0 to 20. Based on the guidelines for scoring (Morin, 1993), the total score with the percentages is categorized as follows: 0–5=no clinically significant insomnia (62.2%); 6–10=subthreshold insomnia (20.9%); 11–15=moderate insomnia (10.8%); and 16–20=severe insomnia (6.1%). The prevalence of severe insomnia, 6.1%, is consistent with the prevalence reported in the DSM-5 discussion of insomnia, which ranged from 6–10% (American Psychiatric Association, 2013). The internal consistency of the measure as estimated by Cronbach's alpha was 0.93. In this study, a score of 1 was assigned to a participant with insomnia (i.e., a total score of 16 or higher indicating severe insomnia). Otherwise a participant was scored 0.

Analytic Procedure

We employed the growth mixture model method using Mplus software (Muthén & Muthén, 2010) to obtain the trajectories of cigarette smoking from T1 to T6. Cigarette smoking at each point in time was treated as a censored normal variable. We applied the full information maximum likelihood approach for missing data (Muthén & Muthén, 2010). We used the optimal Bayesian Information Criterion (BIC; the smallest absolute value indicates the most parsimonious model) (Raftery, 1985; Schwarz, 1978), the entropy measure (values closer to 1 indicate better fit) (McLachlan & Peel, 2000), and Lo-Mendell-Rubin adjusted likelihood ratio test (LMR-LRT) (Lo, Mendell, & Rubin, 2001) to estimate the number of trajectory groups provided that each trajectory group had a prevalence of 5% or more. The LMR-LRT has a null hypothesis of the model with one less class. We also considered the accuracy of group classification indicated by the average Bayesian posterior probability (BPP). Average BPP of 0.70 or higher are considered sufficient to avoid classification error (Nagin, 2005). The observed trajectory for a group consisted of the average frequency of cigarette smoking at each point in time when participants were assigned to the group with the largest BPP (see Figure 1).

To examine the associations of membership in a trajectory group, we used logistic regression analyses (Cody & Smith, 2005) designating the insomnia indicator variable (i.e.,

insomnia=1; otherwise=0) as the dependent variable and the BPPs of membership in the trajectory groups as the independent variables. The BPPs of the no or low cigarette smoking trajectory group were used as the reference probability. Gender, ethnicity, depressive symptoms at T1, age at T6, educational level at T6, partner status at T6, and BMI at T6 were used as control variables.

RESULTS

Among the 674 participants, the mean and standard deviation (SD) of cigarette smoking at each point in time were 0.2 (0.6), 0.8 (1.3), 1.2 (1.6), 1.1 (1.6), and 1.1 (1.5) for T1-T5, respectively. We computed solutions for 2 through 4 trajectory groups. The BICs, the entropy measures, and p values for LMR-LRT were 6493, 0.95, <0.0001 for the 2 group model, 6411, 0.87, <0.0001 for the 3 group model, and 6405, 0.77, 0.1679 for the 4 group model. We chose the 3 trajectory group model because the 4 trajectory group model had a small difference in BIC as compared to the 3 trajectory group model, the lower entropy measure than the 3 trajectory group model, and non-significant LMR-LRT result. The mean BPP of the participants who were assigned to the groups ranged from 88% to 98%, which indicated an adequate classification.

As shown in Figure 1, we labeled the three cigarette smoking trajectory groups as follows. The no or low cigarette smoking trajectory group had an estimated prevalence of 54% and included participants who reported no or low use of cigarette at each wave. The moderate cigarette smoking trajectory group (17%) included participants who reported no use of cigarettes at age 14, use of a few cigarettes or less a day (i.e., on average use of 0.7 cigarettes) at age 19, about a few cigarettes a day at ages 24, 29, and 32 (i.e., on average use of 1.0–1.2 cigarettes). The chronic cigarette smoking trajectory group (30%) included participants who reported use of less than a few cigarettes a day at age 14, less than 1–5 cigarettes a day at age 19, and about half a pack a day at age 24, and more than half a pack a day at ages 29 and 32. Table 2 contains the means with SDs or percentages in each trajectory group for the variables in the study.

Table 3 presents: a) the odds ratios (OR) without the control variables and b) the adjusted odds ratios (AOR) of each variable after adjustment for the other variables in the model. With regard to the control variables, males were less likely to have insomnia than females (AOR=0.34, $p<.05$). Greater depressive symptoms at T1 (OR=1.69, $p<.01$) and lower educational level at T6 (OR=0.82, $p<.05$) were associated with a greater likelihood of insomnia. Compared to the BPP of the no or low smoking trajectory group, a higher BPP for the chronic smoking trajectory group (OR=3.01, $p<.01$; AOR=2.69, $p<.05$) and for the moderate smoking trajectory group (OR=4.58, $p<.01$; AOR=5.33, $p<.01$) were associated with an increased likelihood of having insomnia at T6 after adjustment for the control variables.

DISCUSSION

As hypothesized, the findings indicated that 1) the higher level of smoking trajectory groups (i.e., the chronic and the moderate smoking trajectory groups) compared to the no or low

smoking trajectory group were associated with an increased likelihood of insomnia; and 2) the associations between the trajectories of cigarette smoking and insomnia were maintained after controlling for gender, ethnicity, depressive symptoms, age, educational level, partner status, and BMI.

Our findings are consistent with the results from other investigators who found, for instance, that current smokers had a longer initial sleep latency and less total sleep time than never smokers (Sahlin et al., 2009; Zhang et al., 2006). Our findings are also consistent with the findings that smokers had more difficulty initiating sleep (Wetter & Young, 1994).

The linkage between smoking and insomnia can be explained by psychosocial and physiological mechanisms. Smoking has been found to be related to depressive symptoms (Almeida & Pfaff, 2005; Holma, Holma, Melartin, Ketokivi, & Isometsä, 2013; Pratt & Brody, 2010). Depressive symptoms often co-occur with insomnia, separated, divorced, or widowed marital status, and higher BMI (Foley, Ancoli-Israel, Britz, & Walsh, 2004; Ohayon & Roth, 2003; Roth, 2007; Stewart et al., 2006). However, our analysis controlled for depressive symptoms, partner status, and BMI as well as demographic variables such as gender, ethnicity, age, and educational level, and our findings were maintained.

Biological effects can also explain the association of smoking with insomnia. Increased alpha frequencies and the release of neurotransmitters are associated with arousal involved in sleep (Saint-Mleux et al., 2004; Zhang et al., 2008). Heavy smoking during the day may cause nicotine withdrawal at night time, leading to sleep disturbances and awakening (Wetter & Young, 1994). Indeed, about 20% of heavy smokers awoke from sleep due to nighttime nicotine withdrawal (Rieder, Kunze, Groman, Kiefer, & Schoberberger, 2001). One possibility is that there are changes in both reward and sleep-related systems that occur during adolescence, and smoking (particularly chronic levels) may impair normal development of these resulting in increased rates of insomnia.

Limitations and Strengths

We did not examine several other factors (e.g., physical or mental health conditions, social support) that may contribute to insomnia among cigarette smokers. Indeed, heart disease, bodily pain, diabetes, lung disease, stroke, sleep apnea, and osteoporosis have been found to be associated with sleep-related problems such as breathing pauses, snoring, daytime sleepiness, restless leg syndrome, or insufficient sleep (Foley et al., 2004). According to Gosling and colleagues, having an insecure job and job skills that are not marketable, rarely taking time to relax, having less support from friends or family, and poor physical or mental health increased the odds of sleep disturbances (Gosling, Batterham, Glozier, & Christensen, 2014). Future studies should include these factors as well as symptoms of insomnia at earlier time points in order to obtain a clearer association between smoking and insomnia. Also, future study should include the shorter intervals between smoking measurements in order to deal with bias and/or more complex trajectories of smoking.

The sample consisted of African American and Puerto Rican inner city adolescents studied until the mid 30’s. Consequently, the association between the trajectories of cigarette smoking and insomnia may not apply to the general population. Further studies should

include other ethnic groups. The individuals in our current sample smoked less at T1 than the individuals who dropped out. This may have some impacts on our results. Our data are also based on self-reports. However, studies have shown that the use of this type of self-report data yields reliable results (Ledgerwood, Goldberger, Risk, Lewis, & Price, 2008).

Despite these limitations, the study supports and adds to the literature on this topic in a number of ways. First, unlike most research studies in this area that focus on one point in time, we assess cigarette smoking at five points in time over a span of up to 18 years. The prospective nature of the data allowed us to go beyond a cross-sectional analysis and to consider the temporal sequencing of variables. Second, a major contribution of the study is a unique set of findings associated with different trajectories of cigarette smoking beginning in adolescence as related to adult insomnia in a sample of African American and Puerto Rican living in an urban area of New York City.

Conclusions and Clinical Implications

The clinical implications of our findings shed light on the importance of quitting smoking in order to reduce insomnia among African American and Puerto Rican adults living in an urban area. As a result, we conclude that treatment programs for insomnia should be implemented in parallel with treatment programs focused on smoking. This could form a component or module of existing smoking treatment programs and also may lead to a reduction in insomnia. Indeed, Luo reported that never quitters who continue to smoke as compared to ever quitters who made successful or unsuccessful attempts to quit were more likely to have insomnia (Luo, 2013).

From a public health perspective, our longitudinal study to examine the association between earlier smoking trajectories and later insomnia suggests that treatments designed to reduce or cease smoking may lessen the occurrence of symptoms of insomnia. In sum, a reduction in smoking may be accompanied by a decrease in insomnia.

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GLOSSARY

Trajectory analysis	An approach using trajectory analysis enables us to examine the magnitude, length of time, and the starting point of the use of substance

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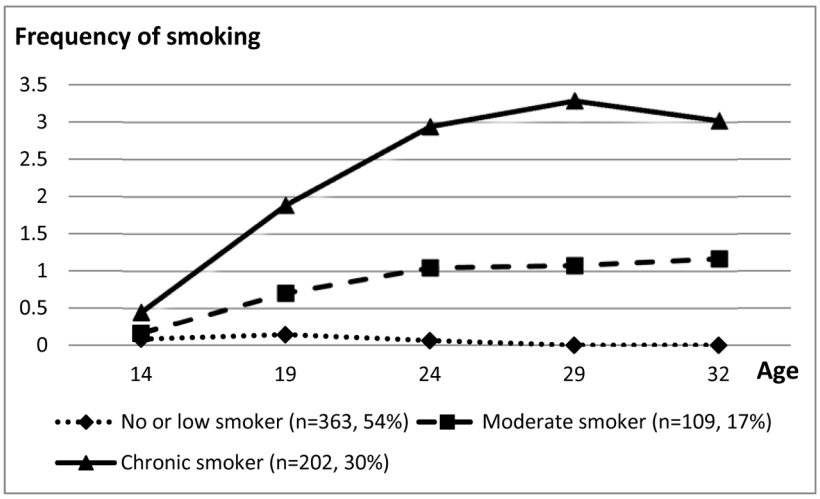

FIGURE 1.

Smoking trajectories from the mid adolescence to the early thirties

Note. The answer options for smoking were 0=none, 1=a few cigarettes or less a week, 2=1–5 cigarettes a day, 3=about half a pack a day, 4=about 1 pack a day, 5=about 1 and half packs a day, and 6=more than 1 and half packs a day.

TABLE 1

Description of the original sample

Wave	Year	Number of participants	Mean age (Standard deviation)	Inter-quartile range of age
Time 1	1990	1,332	14.1 (1.3)	13–15
Time 2	1994–1996	1,190	19.2 (1.5)	18–20
Time 3	2000–2001	662	24.4 (1.3)	23–25
Time 4	2004–2006	838	29.2 (1.4)	28–30
Time 5	2007–2010	816	32.3 (1.3)	31–34
Time 6	2010–2013	674	35.9 (1.4)	35–37

Note: The reduction in funding at T3 is the reason for the somewhat smaller sample at T3.

TABLE 2

Means with standard deviations or percentages of sleep problems and other variables by the smoking trajectory groups

	No or low smoker (54%, n=363)	**Moderate smoker (17%, n=109)**	**Chronic smoker (30%, n=202)**	**Whole sample (N=674)**
Sleep measures				
Insomnia (T6)	3.0%	11.9%	8.4%	6.1%
Difficulty falling asleep (T6)	0.9 (1.1)	1.2 (1.2)	1.1 (1.2)	1.0 (1.2)
Difficulty staying asleep (T6)	0.9 (1.0)	1.2 (1.3)	1.0 (1.2)	1.0 (1.1)
Problems waking up too early (T6)	1.0 (1.1)	1.4 (1.3)	1.2 (1.2)	1.1 (1.2)
Distress caused by sleep difficulties (T6)	1.0 (1.2)	1.3 (1.4)	1.2 (1.3)	1.0 (1.3)
Interference of sleep difficulties with daytime functioning (T6)	0.9 (1.2)	1.2 (1.4)	1.2 (1.4)	1.1 (1.3)
Demographics				
Gender-Female	68.3%	56.9%	47.0%	60.1%
Ethnicity-African American	58.9%	39.5%	49.0%	52.8%
Depressive symptoms (T1)	2.5 (0.9)	2.6 (0.8)	2.7 (0.9)	2.6 (0.9)
Age (T6)	36.0 (1.5)	35.8 (1.3)	35.8 (1.4)	35.9 (1.4)
Educational level (T6)	3.6 (2.3)	3.0 (2.3)	2.1 (2.0)	3.1 (2.3)
Partner status-Cohabiting or married (T6)	52.9%	46.8%	48.5%	50.1%
BMI (T6)	30.2 (6.7)	28.6 (7.5)	29.8 (8.7)	29.8 (7.5)

Notes: T1=Time1 (**Mean age 14)**, T6=Time 6 (**Mean age 36)**

Answer options for sleep problems at T6; 0=none, 1=mild, 2=moderate, 3=severe, 4=very severe

Answer options for depressive symptoms at T1; 0=not at all, 1= a little, 2=somewhat, 3=extremely

Answer options for educational level at T6; 0=11th grade or below, 1=12th grade or GED, 2=Business or technical school, 3=College freshman, 4=College sophomore or Associate's Degree, 5=College junior, 6=College senior or Bachelor's Degree, 7=Postgraduate business, law, medical, masters, or doctoral program

TABLE 3

Odds ratios and adjusted odds ratios with 95% confidence intervals of insomnia at age 36 as predicted by BPPs of smoking trajectories and control variables.

	Odds ratio (95% confidence interval)	Adjusted odds ratio (95% confidence interval)
Gender	0.53 (0.26, 1.08)	0.34 (0.15, 0.81) *
Ethnicity	1.81 (0.95, 3.46)	1.42 (0.68, 2.99)
Depressive symptoms at T1	1.69 (1.16, 2.48) **	1.36 (0.88, 2.12)
Age at T6	0.90 (0.71, 1.14)	0.94 (0.71, 1.24)
Educational level at T6	0.82 (0.70, 0.96) *	0.84 (0.71, 1.01)
Partner status at T6	0.68 (0.36, 1.28)	0.86 (0.42, 1.76)
BMI at T6	1.03 (0.99, 1.07)	1.03 (0.99, 1.07)
Chronic smokers vs. No or low smokers	3.01 (1.34, 6.78) **	2.69 (1.06, 6.82) *
Moderate smokers vs. No or low smokers	4.58 (1.85, 11.34) **	5.33 (1.94, 14.64) **

Notes.

*$p<.05$,

**$p<.01$;

T1=Time1 (Mean age 14), T6=Time 6 (Mean age 36);

Male gender coded with a higher score; Puerto Rican ethnicity coded with a higher score.