Published in final edited form as:

J Addict Med. 2015 ; 9(1): 40–45. doi:10.1097/ADM.0000000000000083.

Insomnia in Adults: The Impact of Earlier Cigarette Smoking from Adolescence to Adulthood

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Abstract

Objectives: This study examined the role of cigarette smoking beginning in adolescence and extending to the fifth decade of life on insomnia **at average age 43** in the Children and Adults in the Community Study.

Methods: Participants were originally assessed in 1983 and came from a community-based random sample of individuals living in two upstate New York counties. Participants were assessed over eight waves of data collection that spanned approximately 29 years, from mean ages 14.1 (T1) to 42.9 (T8). We classified the longitudinal trajectories of cigarette use. Five cigarette use trajectory groups were identified: heavy/continuous smokers, late starters, occasional smokers, quitters/decreasers, and nonsmokers.

Results: The results of the logistic regression analysis **of adult insomnia** for the Bayesian posterior probability (BPP) of the heavy/continuous smokers when compared to the BPP of nonsmokers **was** statistically significant - adjusted odds ratio (AOR) of **3.35** (95% CI=[**1.06, 10.56**], $p < 0.05$) – after adjustment for control variables.

Conclusions: The findings highlight the importance of heavy chronic smoking as contributing to insomnia. Clinicians should focus their efforts **of** smoking prevention and treatment on younger individuals, as well as promoting cessation among older adult smokers in order to decrease the likelihood of insomnia.

Keywords

smoking trajectories; insomnia; longitudinal study; public health

In recent years, insomnia, as both a disorder (Roth, 2007) and a symptom of other psychiatric disorders (Buysse, 2008), has been identified as a major public health concern (Neubauer, 2005). Epidemiologic studies have found that the prevalence rates of one or more symptoms of insomnia in the general adult population range from 30% to 35% (Roth, 2007), while the prevalence of moderate to severe symptoms, or chronic insomnia, ranges between 10% to 15% (Ohayon, 2002; Roth & Roehrs, 2003). The high prevalence of

insomnia throughout the adult population makes understanding the risk factors of insomnia a public health priority. The goal of the present study was to examine the trajectories of cigarette smoking over a significant part of the life course and to identify their consequences on insomnia in the fifth decade of life.

Prior research has established that insomnia may be responsible for a number of mental, medical, and psychosocial conditions. The symptoms of insomnia are associated with decreased psychomotor performance (Bonnet & Arand, 2003), cognitive dysfunction (Fulda & Schulz, 2001), depression (Perlis et al., 2006), and a reduced quality of life (Ishak et al., 2012).

Cigarette smoking is associated with a number of harmful health effects, including cardiovascular disease, respiratory disease, and cancer (USDHHS, 2010). In recent years, there has been an increased focus and a growing body of literature that examines the deleterious effects of cigarette smoking on sleep problems (Liu et al., 2013) and insomnia (Brook et al., 2012; Jaehne et al., 2012). Research carried out by Sabanayagam & Shankar (2011) found that current cigarette smokers were nearly twice as likely to report insufficient sleep and rest **as compared to non-smokers**. Several investigators (e.g., Jaehne et al., 2012; Zhang et al., 2008; Zhang et al., 2006) have reported that current smokers were more likely to **have** symptoms of insomnia, including shorter sleep time, longer sleep latency, higher rapid eye movement, sleep density, more episodes of sleep apnea, and more leg movements during sleep. Zhang et al. (2006, 2008) also suggested that the effects of cigarette smoking on sleep can be reversed by cessation of cigarette smoking. Finally, in a large case-control study, Cohrs and colleagues (2012) reported impaired quality of sleep among smokers as compared with non-smokers.

Previous research has indicated that a number of adults who were depressed had a diagnosis of insomnia (Spiegelhalder et al., 2013). Depression has also been found to be associated with increased risk of cigarette smoking and nicotine dependence (Trosclair & Dube, 2010). For example, Patten et al. (2000) found that depressive symptoms and cigarette smoking predict the development and persistence of sleep problems. Greater body mass index (BMI) and obesity are also linked with a diagnosis of insomnia (Brook, 2005). A number of studies have found **an** elevated BMI to be associated with decreased sleep duration (Patel et al., 2008). Females have been found to be more likely to have insomnia (Neubauer, 2005). In a meta-analysis of twenty-nine research studies on gender and insomnia, conducted by Zhang & Wing (2006), females were found to have a risk ratio of 1.41 compared to males.

Socioeconomic status and marital status have been documented by researchers (Chen & Miller, 2013) to be associated with both cigarette smoking and insomnia. In addition, anxiety (Uhde and Cortese, 2008) and attention deficit hyper-activity disorder (ADHD, Cortese, et al., 2006) have also been found to be associated with insomnia. Therefore, in the present study, we controlled for the above factors (e.g., depression) when examining the relationship of trajectories of cigarette smoking and insomnia.

Both cigarette smoking and insomnia are associated with a number of physical and psychiatric disorders. Therefore, understanding the relationship of the patterns of **heavy and continuous** cigarette smoking and insomnia may reduce the public health burden resulting

from both cigarette smoking and sleep disturbance. The present study adds to the literature on cigarette smoking and insomnia by examining the association between trajectories of cigarette smoking from adolescence into adulthood and insomnia in the early 40s. Previous research (Brook, et al., 2011) with this sample found five longitudinal trajectory groups. One trajectory group was characterized by chronic, heavy cigarette smoking. Another trajectory group consisted of nonsmokers. Additionally, there were three intermediate trajectory groups that consisted of occasional smokers, late starters, and quitters/decreasers. We **hypothesized** that the chronic/heavy smokers **would** be more likely to have insomnia in adulthood than the non-smokers. The quitters/decreasers **would** not differ significantly from the non-smokers with regard to insomnia.

METHODS

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 about their children. The original maternal/youth study assessed problem behaviors 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 U.S. Census (Cohen and Cohen, 1996). For example, 75% of the children lived with married parents, and 19% lived with a mother who was not currently married; the census figures were 79% and 17%, respectively. 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), 2007 (T7, N=607), and 2012-2013 (T8, N=528). The mean ages (SDs) 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, 36.6 (2.8) at T7, and 43.0 (2.8) at T8, respectively. We have data on 806 participants at two or more waves of data collections between T2 and T7. These participants were included in the trajectory analyses of cigarette smoking.

We examined the association between earlier trajectories of cigarette smoking (T2–T7) and adult insomnia at T8, using logistic regression analysis ($N = 528$). The participants with missing data regarding insomnia at T8 ($N = 278$) were excluded from analyses of the association between earlier trajectories of cigarette smoking and adult insomnia. There were no differences between those included in the analysis of adult insomnia ($N = 528$) as compared with those who were excluded ($N = 278$) from it with respect to age ($t = 0.52, p\text{-value} = 0.60$), T2 cigarette smoking ($t = 0.31, p\text{-value} = 0.76$), T2 depressive mood ($t = -0.13, p\text{-value} = 0.89$), T2 anxiety ($t = -1.73, p\text{-value} = 0.08$), and T2/T3 ADHD (8.7% vs. 6.2%, $\chi^2(1) = 3.2, p\text{-value} = 0.07$). There was a significantly higher percentage of females in the analysis of adult insomnia as compared with those who were excluded (54.9% vs. 45.1%, $\chi^2(1) = 21.4, p\text{-value} < 0.001$).

Extensively trained and supervised lay interviewers administered interviews in private (Cohen and Cohen, 1996). Written informed consent was obtained from participants and

their mothers in 1983, 1985-1986, and 1992, and from participants only in 1997, 2002, 2005-2006, and 2012-2013. The Institutional Review Board of the New York University School of Medicine authorized the use of human subjects in this research study. Earlier waves of the study were approved by the Institutional Review Boards of the Mount Sinai School of Medicine and New York Medical College. Additional information regarding the study methodology is available in prior publications (Brook et al., 1986).

Measures

Trajectories of Cigarette Smoking (T2-T7)—The cigarette smoking data were obtained from interviewer-administered questionnaires. At each follow-up wave (T2-T7), questions were asked about the frequency of using tobacco during the period from the last time wave through the current time wave (selected from the Monitoring the Future study; Johnston et al., 2006). The tobacco measure at each point in time had a scale coded as none (0), less than daily (1), 1-5 cigarettes a 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 0.59 (1.10), 0.78 (1.31), 1.37 (1.63), 1.36 (1.62), 1.24 (1.65), and 1.22 (1.73) for T2-T7 respectively. The tobacco measure has been found to predict young adult psychiatric disorders (Brook et al., 2002) and health problems (Brook et al., 2004). In our prior research (Brook et al., 2011), using Mplus software (Muthén & Muthén, 2010), we identified five trajectories of cigarette smoking. Bayesian posterior probability (BPP) variables were obtained for each participant. As shown in Figure 1, the trajectory smoking groups were named: heavy/continuous smokers (19.5%), late starters (10.7%), occasional smokers (18.1%), quitters/decreasers (8.2%), and nonsmokers (43.6%). As shown in the figure, the heavy/continuous smokers started smoking early, reached their maximum level (i.e., about one pack a day or more) in their late twenties, and then maintained that level into their thirties. The late starters started smoking in late adolescence but reached the same level of smoking (i.e., about one pack a day) as the heavy/continuous smokers in the late twenties. The late starters then maintained that level. The occasional smokers increased the amount of smoking from adolescence to the early twenties 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 (i.e., mean age 14) and reached their maximum level of smoking (i.e., daily smoking) in late adolescence. The participants then tapered off gradually from that level to minimal smoking at mean age 32.

Insomnia (T8)—Bergen Insomnia Scale has acceptable two-week test-retest reliability (.77) and good discriminative validity in relation to other self-report measures of insomnia (Pallesen et al., 2008). This insomnia measure was found to be significantly associated with depression (Dørheim, Bjorvatn, Eberhard-Gran, 2012) and the use of a computer or a mobile phone in bed (Fossum, et al., 2013). At T8, the Bergen Insomnia Scale was modified with some elements taken from the DSM-IV-TR diagnostic criteria to assess insomnia. The participants responded to seven questions about their sleeping problems. First, participants reported the severity of their insomnia symptoms over the past month; namely, (1) difficulty falling asleep; (2) difficulty staying asleep; and (3) waking up too early. Second, the participants were asked about the consequences of their sleep problems (if any): (1) to what extent such problem(s) interfered with daily functioning (e.g., daytime fatigue, ability to

function at work, memory, concentration, etc.); (2) how distressed they were about the problem(s); (3) how dissatisfied they were with their sleep **patterns**, and (4) how noticeable their sleep problem(s) were to others. Participants who answered “moderate,” “severe,” or “very severe” to any of the three sleep symptoms and who responded with “much” or “very much” to at least **two** of the **four** consequences of sleep problems, were characterized as having insomnia and assessed as having insomnia (i.e., insomnia indicator set to 1); **42 (8.0%)** participants had insomnia at T8.

Control variables—The control variables were: gender [female (0) – male (1)], T8 age (years), T8 marital status [not married (0) – married (1)], T8 educational level [less than a high school diploma (1) – doctor degree or equivalent (6)], T8 obesity [body mass index (BMI) 29.9 (0) – BMI>29.9 (1)], T8 cigarette smoking in the past year [never (0) – every day (7), Johnston et al., 2006], T7 depressive mood [5 items scored on a four-point scale: false (1) to true (4); alpha=0.75; e.g., “Over the last few years, how much were you bothered by feeling low in energy or slowed down?”(Derogatis et al., 1974)], T7 anxiety [3 items scored on a five-point scale: not at all (1) to extremely (5); alpha=0.81; e.g., “Over the last few years, how much were you bothered by feeling nervous or shaky inside?”(Derogatis et al., 1974)], and T2/T3 ADHD [No (0) - Yes (1), Brook, et al., 2008].

Analysis

SAS was used to perform logistic regression analyses to investigate the associations between the trajectory group membership and insomnia (N=503). The dependent variable was the indicator variable of insomnia at T8. Since specifying which group an individual belongs to is prone to error, we used the BPPs of belonging to each trajectory group as the independent variables (Datta & Satten, 2000). Since one group was chosen as the reference, the number of independent variables was G-1, where G was the number of trajectory groups. First, bivariate analyses of the trajectories of tobacco use as related to insomnia were conducted. Second, multivariate analyses were conducted between the BPPs of the trajectories and insomnia, controlling for gender, T8 age, marital status, educational level, and obesity, cigarette smoking in the past year, T7 depressive mood, T7 anxiety, and T2/T3 ADHD.

RESULTS

There were significant associations between the trajectories of smoking and adult insomnia at T8 (16.0% for heavy/continuous smokers, 11.7% for late starters, occasional smokers, 5.9% for quitters/decreasers, and 5.4% nonsmokers; $\chi^2(4) = 12.9$, p -value=0.01). Table 1 presents the results of the multivariate logistic regression analyses. After controls, compared with nonsmokers, heavy/continuous smokers had a significantly higher likelihood (**A.O.R. = 3.35**, $p<.05$) of insomnia. Among the covariates, T7 depressive mood (A.O.R. = 1.13, $p<.05$) was associated with a greater likelihood of insomnia, while T8 age (A.O.R. = 0.83, $p<.05$) and male gender (A.O.R. = 0.35, $p<.01$) were associated with a lower likelihood of insomnia.

DISCUSSION

The Children and Adults in the Community Study offered a rare opportunity to study different longitudinal relationships between trajectories of cigarette smoking and insomnia. There were five trajectory groups of cigarette smoking within the sample: the heavy/continuous smokers, the late starters, the occasional smokers, the quitters/decreasers, and the nonsmokers. Among these trajectory groups, we found a significant increase in the odds of insomnia in the heavy/continuous smokers when compared to the nonsmokers, but no significant increase in the odds ratio for the late starters, occasional smokers, and the quitters/decreasers, when controlling for age, gender, depression, anxiety, smoking in the past year, obesity, education, marital status, and ADHD in adolescence. These findings indicate an increased risk in insomnia for those who began smoking cigarettes in their mid adolescent years and continued into adulthood. This finding also suggests that there might be some long-term and cumulative pathophysiological effects from cigarette and nicotine use in insomnia over a long period of time, especially beginning in adolescence. The present study is the first, to our knowledge, that examined the association between the trajectories of cigarette smoking from adolescence to the fifth decade of life and insomnia in adulthood.

The findings of the present study are consistent with the limited number of longitudinal investigations of community samples carried out by other researchers on the effects of cigarette smoking on insomnia. Jaehne and colleagues (2012) found that smokers, who averaged a pack a day for a median of 13.1 years, were more likely to experience insomnia-like symptoms, including shorter sleep period time and longer sleep latency, than were non-smokers. A study carried out by Zhang and colleagues (2008) found that subjective reports of the quality of disturbed sleep among cigarette smokers as compared to nonsmokers matched differences in objective polysomnographic readings. Furthermore, in a previous longitudinal study carried out by the present research group (Brook et al., 2012) using a different sample, women in late mid-life who were chronic heavy smokers had a significantly greater likelihood than the non-smokers of having moderate to severe insomnia.

Several psychosocial and physiological factors may account for the association of the trajectories of cigarette smoking and insomnia. In the bio-psychosocial domain, as noted in the Introduction Section, underlying factors might include depression and obesity (Adam, 2005; Spiegelhalter et al., 2013). As regards demographic variables, age, gender, marital status, and low socioeconomic status are reported to be associated with both cigarette smoking and symptoms of insomnia (Bastien et al., 2004; Chen & Miller, 2013). In accord with the findings in the literature, the results of our analyses indicated that these psychosocial and demographic factors were associated with insomnia when the participants were in their early 40s. Among them, depression, in particular, played a pivotal role in insomnia even after controlling for other factors. However, the relation between chronic cigarette smoking and insomnia remained in the multivariate analysis after controlling for these psychosocial factors.

Cigarette use has also been linked to adverse life events and work stress (Bastien et al., 2004), including financial stress (Siahpush et al., 2007), which are also associated with

symptoms of insomnia (Ohayon, 2002). Bastien and colleagues (2004) found that insomnia was often precipitated by occupational and life stressors, including work conflicts, financial strain, and interpersonal relationship difficulties. An important step for future research would be to determine whether occupational and life stressors serve to mediate the association of trajectories of cigarette smoking and insomnia.

The processes that underlie the biochemical relationship between cigarette smoking and insomnia have not been fully explored. Prior research has suggested that nicotine, the primary component of cigarettes, has adverse pathophysiological effects on the brain and the central nervous system (Zhang et al., 2006). There are a myriad of mechanisms by which nicotine may lead to sleep disturbances that characterize insomnia. As a stimulant, nicotine interferes with sleep by elevating blood levels of nicotine towards the afternoon and evening, which may affect the amount of time it takes for smokers to fall asleep (Wetter et al., 1994). While asleep, smokers may experience a short-term withdrawal of nicotine, in which blood levels of nicotine are decreased, leading to awakening mid-sleep (Wetter et al., 1994). In addition, nicotine stimulates neurotransmitters that are responsible for the release of dopamine and serotonin. This in turn may affect the normal patterns of sleep, in particular non-REM sleep (Zhang et al., 2006). Nicotine has also been found to be responsible for inhibiting sleep-promoting neurotransmitters (Saint-Mleux et al., 2004). Furthermore, cigarettes contain other toxic components besides nicotine that may adversely interact with the brain's normal regulation of sleep (Liu et al., 2013). Future research is needed to investigate the biochemical relationship between cigarette smoking and insomnia.

We found that females had an elevated likelihood of having insomnia after adjustment for a number of control variables (see Table 1). This result is in accord with prior research which suggests that females are more likely to have insomnia (Zhang & Wing, 2006). Certain symptoms may be more readily recognized or endorsed by women than by men. It is also possible that variations in estrogen contribute to differences in the expression of insomnia (Polo-Kantola, et al., 1998). Our multivariate analysis showed that depressive mood was associated with a higher likelihood of insomnia. Depression is frequently comorbid with insomnia (Stewart et al., 2006). Studies have suggested a bidirectional relationship between insomnia and depression (e.g., Sivertsen, et al., 2012). Studies have also suggested that depression may share genetic vulnerabilities (e.g., affecting neurotransmitter systems) with insomnia (Saus, et al., 2010).

The findings of the present study should be understood in the context of the limitations of the research design. First, our sample was composed of mostly white individuals living in upstate New York. Consequently, the results cannot be generalized to minority group members. Second, the present study relied on self-reported data from a community sample rather than polysomnographic diagnostic tools in a clinical setting. However, there is research to support the reliability and validity of self-reported measures of insomnia (Vartiainen et al., 2002). As noted previously, Zhang et al. (2006) found that subjective, self-reports on sleep quality were consistent with the findings of objective polysomnographic measures. In addition, because the data are correlational, we cannot ascertain causal relations. Third, the results of the present study cannot rule out the possibility that there may exist a vicious cycle between trajectories of cigarette smoking and

insomnia. For example, insomnia may lead to an increase in cigarette smoking through an increase in depressive symptoms and anxiety as well as having more time to smoke.

The present study has multiple strengths. In contrast to a cross-sectional research design, the present study used longitudinal data to trace the trajectories of cigarette smoking over a time period spanning 23 years from adolescence to adulthood. The group-based trajectory approach used in this study presents a more complete description of patterns of cigarette smoking over a significant part of the life course. The present study also controls for a number of important variables, including age, gender, depressive mood, educational level, marital status, and obesity, which limits confounding, extraneous variables from affecting the analyses.

The findings of the present study add to previous research on the connection between cigarette smoking and insomnia. Our analysis expands the existing literature by using a longitudinal trajectory approach that differentiates between groups of cigarette smokers in regards to levels of usage and duration of use, and estimates their likelihood of experiencing insomnia. Our finding that the heavy/continuous smoker group, but not the late starter group, has a significantly increased risk as compared with the nonsmoker group suggests that adult smokers who begin smoking in adolescence have an increased risk of insomnia. However, on a positive note, our findings also indicate that smoking cessation among previous adolescent smokers can reduce the symptoms of insomnia in adulthood. The major findings suggest that further theorizing and future research are needed on: (1) the mediating variables between chronic cigarette smoking and insomnia and (2) the potential significance of increased efforts in prevention, early interventions, screening, and treatment of chronic cigarette smoking in order to decrease later insomnia. Efforts to incorporate genetic mechanisms affecting the association of cigarette smoking and insomnia may improve and further tailor interventions in order to have a positive effect on individuals who smoke cigarettes and suffer from insomnia.

Response to Reviewer Comments

We thank the reviewers and the editor for their comprehensive review of our manuscript. The contributions of the reviewers have greatly enhanced the paper. We address specific remarks in detail below.

Reviewer #1

Issue 1:

Abstract:

Methods: P2 line 16: “?and came from a community?” Delete “of”.

Results: The five cigarette use trajectory groups were identified in an earlier publication and should thus be moved to the Methods section since they predated this paper.

Re: “The results of the logistic?” “Results of what???? I presume they mean insomnia but this needs to be overtly stated”.

Response: We agree with the reviewer and have made the necessary changes as suggested (see Page 2).

Issue 2:

P3

Line 50: “twice as likely to report insufficient sleep and rest.” As compared to what, non-smokers?

Response: We agree with the reviewer and have made the necessary changes as suggested (see Page 3).

Issue 3:

P3

Line 58: “higher rapid eye movement, sleep density, more episodes of sleep apnea, and more leg movements?” These do not sound like self-report symptoms, but more likely PSG determined. Please clarify.

Response: We agree with the reviewer. These symptoms were PSG determined. We have clarified the issue.

Issue 4:

P4

Line 30: “?factor associated with insomnia.” Which direction?

Line 53: “?smoking and insomnia may HELP TO reduce the public?”

Response: We agree with the reviewer and have made the necessary changes as suggested (see Page 4).

Issue 5:

P5

Lines 12-17: Improper tense. This is retrospective info. “We hypothesized that the chronic/heavy smokers WOULD?” Etc.

Response: We agree with the reviewer and have made the necessary changes as suggested (see Page 5).

Issue 6:

P5

Line 43: Please briefly describe what the values of these demographic features are or provide reference/source to obtain them.

Response: We have briefly described the values of the demographic features and provided a reference (see Page 5).

Issue 7:

P6

Line 22: “?.higher percentage of females.” Included in the sample or excluded from it?

Line 25: Could “extensively” be quantified or described a bit?

Line 47: “?.interviewer-administered questionnaires.” What exactly was asked? A bit of a description, sample, or reference is needed.

Response: We agree with the reviewer have made the necessary changes as suggested (see Page 6). We have also added a reference to the Sample Section.

Issue 8:

P10

Comment should be made about male vs. female gender significance and especially, about the significance of depression and its effects on both insomnia and smoking.

Response: We have included a discussion of the gender differences and the association between depression and insomnia (see 13).

Issue 9:

P10

Lines 52-54: Causation in the opposite direction cannot be ruled out, or at least synergistic effects.

Response: We agree with the reviewer and have added this point in the Limitation Section (see Page 14).

Reviewer #2

Issue 1: Authors need to provide more information on the psychometrics of the Bergen insomnia scale.

Response: We have added more information on the psychometrics of the Bergen Insomnia Scale to the text (see Page 8).

Issue 2: Only major depression was controlled for among psychiatric disorders. Did authors have information on other disorders that are related to insomnia (e.g., anxiety, ADHD, psychosis and bipolar disorder)? Is so, why did they not include those variables.

Response: As suggested by the reviewer, we have included T7 anxiety, T2/T3 ADHD as controls. Data on bipolar disorder and psychosis are not available.

Reviewer #3

Issue 1: The trajectory analysis appears to be a restating of previously published analyses and should be taken out of the results section and added to methods with reference to the 2011 paper. The figure should not be included in the manuscript unless permission is obtained from the publisher of the 2011 paper.

Response: We have taken out the trajectory analysis from the Analysis and Results Sections. We have added the description of the trajectories obtained from the 2011 paper to the Measures Section. We have also asked the publisher of the 2011 paper to allow us to include the figure in the present paper.

Issue 2: The analysis relating trajectory class to insomnia is overly simplistic. It relates trajectories of smoking from T2-T7 to insomnia but ignores smoking status at T8, the time at which insomnia is assessed. There needs to be an attempt to distinguish the effects of prior smoking history from current smoking.

Response: We have added T8 cigarette smoking in the past year to the equation. As shown in the Result Section, the heavy/continuous smokers showed an elevated likelihood of insomnia as compared with non-smokers despite controlling for current smoking. .

Issue 3: Raw data should be reported presenting the % of participants in each trajectory group who have insomnia.

Response: We have reported the % of participants in each trajectory group who have insomnia (see Page 9).

Issue 4: The Method section refers to a continuous measure of sleep problems, but no data are provided on it. Those analyses should be presented.

Response: In the present study, we used the dichotomous measure of insomnia throughout the paper. We have clarified this point in the manuscript.

ACKNOWLEDGEMENTS

This research was supported by the following grants from the NIH, all awarded to Dr. Judith S. Brook: research grant DA003188 and Research Career Award DA000244, both from the National Institute on Drug Abuse, and research grant CA094845, from the National Cancer Institute.

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Figure 1. Developmental Trajectories of Cigarette Smoking Extending from Adolescence to Age 37 (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 a day; 1.00=less than daily smoking; 0.00=none.

TABLE 1

Logistic Regression Analyses: Trajectories of Cigarette Smoking With Nonsmokers as the Reference Group on T8 Insomnia (N=528).

Independent Variables	Insomnia (8.0%) A.O.R. (95% C.I.) (with controls)
Heavy/Continuous Smokers	3.35 (1.06 – 10.56) *
Late Starters	1.16 (0.30 – 4.43)
Occasional Smokers	2.09 (0.44 – 9.97)
Quitters/decreasers	1.03 (0.28 – 3.79)
Gender (Male=1; Female=0)	0.35 (0.16 – 0.78) **
T8 age	0.83 (0.72 – 0.96) *
T8 educational Level	0.76 (0.55 – 1.05)
T8 married	0.69 (0.34 – 1.42)
T8 Obesity	1.47 (0.72 – 3.04)
T8 Cigarette Smoking	1.03 (0.81 – 1.32)
T7 Depressive Mood	1.13 (1.01 – 1.27) *
T7 Anxiety	1.06 (0.88 – 1.28)
T2/T3 ADHD	0.37 (0.12 – 1.15)

Note:

1. O.R. = Odds Ratio; A.O.R. = Adjusted Odds Ratio; C.I. = confidence interval;

2.

*** p<0.001 (one-tailed test);

3. Each of the independent variables were included in the multivariate logistic regression analyses.

* p<0.05;

** p<0.01;