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Trajectories of Cigarette Smoking in Adulthood Predict Insomnia Among Women in Late Mid-life

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Abstract

Objective—To examine the relationship between trajectories of cigarette smoking among a community sample of women (N=498) and insomnia in late mid-life.

Methods—Participants were administered structured interviews at four time waves in adulthood, spanning approximately 25 years (mean ages=40, 43, 48, and 65 years). At each wave, data were collected on participants' cigarette smoking. At the most recent time wave, in late-mid-life, participants reported on their insomnia (difficulty falling asleep, staying asleep, early morning wakening, and daytime consequences of these sleep problems).

Results—Growth mixture modeling extracted four trajectory groups of cigarette smoking (from mean ages 40–65 years): chronic heavy smokers, moderate smokers, late quitters, and non-smokers. Multivariate logistic regression analysis then examined the relationship between participants' probabilities of trajectory group membership and insomnia in late mid-life, with controls for age, educational level, marital status, depressive symptoms, body mass index, and the number of health conditions. Compared with the non-smokers group, members of the chronic heavy smoking trajectory group were more likely to report insomnia at mean age 65 (Adjusted Odds Ratio=2.76; 95% confidence interval = 1.10–6.92; $p<0.05$).

Conclusions—Smoking cessation programs and clinicians treating female patients in mid-life should be aware that chronic, heavy smoking in adulthood is a significant risk-factor for insomnia.

Keywords

Smoking and insomnia; Insomnia; Smoking trajectories; Mid-life women; Women's health

INTRODUCTION

Insomnia is increasingly recognized as a significant public health problem. Approximately 10–30% of adults report experiencing at least one symptom of insomnia several times per week [1,2], and an additional 10–15% suffer from chronic insomnia [3], depending on the criteria used [4,5]. Insomnia is generally defined as difficulty falling asleep, remaining asleep, or early morning wakening [2,6], and may also include the qualitative experience of non-restorative sleep as well as the consequences of poor sleep on daytime functioning [2]. In addition to the above criteria, a clinical diagnosis of insomnia, based on the Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition, Text Revision; DSM-IV-TR) [7], requires the presence of symptoms, which are not the result of another disorder, for

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one month [2,3,4]. The population prevalence of an insomnia diagnosis is estimated to be about 6% [2].

Most sleep research has found higher rates of insomnia among women (e.g., 4,8–12), and among older adults [2,4,9], with an age-related increase in the ratio of females:males with insomnia [12]. Furthermore, some studies (e.g., 13,14) showed that women have lower rates of remission of insomnia as compared with men, thus suggesting that the higher prevalence of insomnia among women may reflect, in part, fewer remitted cases in addition to a higher incidence [6,13]. According to the Centers for Disease Control and Prevention, approximately 35% of women aged 60 to 70 report trouble sleeping [15].

The Consequences of Insomnia

Pertinent to the present study of women in late mid-life, the sequelae of insomnia include both numerous physical morbidities (e.g., cardiovascular disease [16,17], increased body mass index [18], diabetes mellitus [18], and heightened pain sensitivity [19]), as well as psychiatric conditions, such as anxiety and depression [5,20–22]. Depression has been found to be both a precursor (e.g., 14) and, more often, a consequence [23,24] of insomnia (further discussed below). Some investigations (e.g., 25,26) have also reported an association between insomnia and an increased risk of mortality, although not all studies have supported this finding (e.g., 8). Insomnia has also been linked with greater health care utilization [5] and higher medical costs [27,28], as well as social and functional impairment [18,29,30], such as weakened interpersonal relationships [31], motor vehicle accidents [32], falls among older adults [33,34], and greater work absenteeism and disability [32,35]. In addition, individuals with insomnia may use over-the-counter, prescription, or illicit drugs, or drink alcohol in excess, in an attempt to induce sleep [21,23,36,37].

The Effects of Smoking on Sleep

Although smoking cessation medications (e.g., bupropion, the nicotine patch) are known to affect short-term increases in insomnia [38–40], there is relatively little research on the association of cigarette smoking and insomnia, and no studies, to our knowledge, have examined the effects of smoking trajectories on insomnia. Given the high rates of insomnia among women in late mid-life, approximately 14% of whom are lifetime smokers [41], a better understanding of the impact on insomnia of long-term smoking patterns among women will help inform cessation programs and clinicians treating women in mid-life. The present study, therefore, is the first to examine the longitudinal association between adult trajectories of cigarette smoking and insomnia among women in mid-life.

Both physiologic measures (e.g., polysomnography) and self-report research generally support the role of cigarette smoking in arousal, sleep inhibition, and insomnia [11,42,43]. However, most of the epidemiological or psychosocial studies of smoking and insomnia employ a cross-sectional design, and current or lifetime smoking was measured as either present or absent (e.g., 44,45). One exception is a study by Janson et al. [46], which assessed smoking twice over a 10-year interval, using a dichotomized measure, among adult Swedish men. These researchers showed that continuous smoking was related to insomnia at follow-up. Studies of adolescents (e.g., 47,48) have also shown that students who smoked cigarettes reported higher rates of insomnia than their non-smoking peers.

Physiological research also supports the effects of smoking (and nicotine) on sleep, although the mechanisms involved are not completely understood (and the topic is beyond the scope of this paper). Studies have shown that nicotine acts on neurotransmitter systems associated with both arousal [39] and with the inhibition of sleep promotion [49]. Electroencephalographic measures have also demonstrated differences in the brain waves of

smokers versus non-smokers during sleep [50], such as increased alpha-power waves in smokers, suggesting greater arousal [50,51]. Furthermore, smoking may adversely impact sleep through its association with respiratory and other medical conditions, such as chronic obstructive pulmonary disease (COPD) [39,52], and obstructive sleep apnea (OSA) [53,54].

Smoking Among Older Adults

There are few studies on smoking prevalence and its correlates among older individuals, and only two investigations, to our knowledge, have examined trajectories of cigarette smoking into mid-life. Frosch and colleagues [55] assessed smoking across the adult lifespan, and discerned five trajectory groups, including lifetime smokers (8%) and late quitters (16.7%), the majority of whom (in the latter group) smoked until the late sixties. Chassin et al. [56] followed a cohort (of mostly white Americans) from ages 10–42, and reported six trajectory groups. By age 42, half of the six smoking trajectory groups no longer smoked, and half were persistent smokers. Two (of the three) persistent smoking trajectory groups (17.1% of the total sample) continued to smoke heavily into the early forties.

Depression, Smoking, and Insomnia

Depression (and depressive symptoms) have been linked separately with cigarette smoking [57,58], and with insomnia [23,59]. Ford & Kamerow [21], for example, examined the relationship of depression and insomnia among males and females, aged 18 to 65, who participated in the Epidemiologic Catchment Area study. These researchers showed that participants who had insomnia (but not major depressive disorder) at baseline, and whose insomnia hadn't remitted at 1-year follow-up, were almost forty times more likely to have major depression at follow-up than participants who reported no insomnia at either interview. Although studies have suggested that depression may share genetic vulnerabilities (e.g., affecting neurotransmitter systems) with insomnia and with smoking [9,60–64], the specific common mechanisms are not yet known [61].

In sum, a number of studies have demonstrated that smoking and insomnia are related, but no research has examined the association of smoking trajectories and insomnia in adulthood. Drawing on the statistical approaches of Nagin [65] and Roeder, Lynch, & Nagin [66], the present study focuses on the longitudinal relationship of trajectories of cigarette smoking and insomnia in a population at-risk for sleep problems; namely, women in late mid-life. According to Nagin & Odgers [67], group-based trajectory modeling has increasingly been applied in clinical research, and has two major advantages. First, it enables the simultaneous examination of the frequency and length-of-time of smoking. Thus, a trajectory analytic approach, which assesses longitudinal patterns of smoking, may more accurately capture an individual's smoking behavior and its relationship to insomnia than the assessment of smoking at one or two time points. (This issue may be especially germane to women in late mid-life, due to the naturally occurring reduction in smoking among this age group.) Therefore, this approach has an advantage over an analysis that only examines earlier smoking as a predictor of later insomnia. Second, since the trajectories emerge from the analysis of a formal statistical model, they are more objectively defined than if they had been extracted from subjective classification rules alone [67].

Due to the paucity of longitudinal research on smoking patterns that extend into older ages, our hypothesized smoking trajectory groups were largely based on epidemiological data (e.g., the National Survey on Drug Use and Health) [68], and on the work of Frosch et al. [55] and Chassin et al. [56], noted above. Furthermore, prior investigations have found that stable high-level smokers (i.e., individuals who continually smoke at relatively elevated rates throughout adulthood) tend to have greater psychosocial risk factors [56] as well as to report more life stressors [69]. In addition, both duration and intensity of smoking are

related to greater morbidity [70,71,72], which preliminary evidence suggests may include sleep problems [73]. Taking these prior studies together, we postulated that there would be 4–5 smoking trajectory groups, consisting of a) heavy continuous smokers, b) moderate continuous smokers, c) quitters, d) non-smokers, and possibly, e) light or intermittent smokers. We also hypothesized that membership in the heavy continuous smoking trajectory group, as compared with the non-smoking group, would be associated with insomnia in late mid-life. Our analyses controlled for depression which, as noted above, may be a precursor [8] or a consequence [24] of insomnia, as the focus of the present study is the relationship of trajectories of smoking and insomnia, independent of the effects of depression. Similarly, we controlled for body mass index (BMI) and for medical conditions, which have been found to be related to sleep problems, e.g., through their association with sleep-disordered breathing [39].

METHOD

Participants and Procedure

Data on the participants in the present study came from the Longitudinal Study of Women in Midlife, a community-based random sample of families residing two upstate New York counties (Albany and Saratoga) in 1975. A complete description of the study methodology and sampling procedure is available in prior publications (see 74). At Time 1 (1975), 92% of the participant women were white, and their mean age as 32 years. There was a close match of the participants at Time 1 (T1) on family income, maternal education, and family structure with the 1980 census for upstate New York by the U.S. Census Bureau [74].

Since T1 (N=793), the participants in the present study were interviewed four times: in 1983 [Time 2 (T2), N=772; mean age=40], 1985–1986 [Time 3 (T3), N=717; mean age=43], 1992 [Time 4 (T4), N=719; mean age=48], and 2009 [Time 5 (T5), N=498; mean age=65]. The 1983 sample was used as the base for the present analysis. Of the 274 women who participated in 1983 but not in 2009, 104 women had died, 27 refused to participate, and 143 women were lost to follow-up. Eliminating those who were deceased, the participation rate in 2009 was 78% of those participating in 1983 (N=498 of 772–104). We conducted t-test analyses to compare the 498 participants and the 274 non-participants at T5. The results indicated that, compared with the non-participants, the 498 participants had a higher educational level at T2 ($t=6.30$, $p<0.001$), a greater family income at T2 ($t=5.4$, $p<0.001$), lower depressed mood at T2 ($t=2.60$, $p=.009$), and a lower frequency of smoking at T2 ($t=2.95$, $p=0.003$). The frequencies of drinking beer or wine, drinking hard liquor, and using marijuana at T2 did not differ significantly between the 498 participants and the 274 non-participants at T5 ($p>0.05$). Thus, the sample in this study differs somewhat from the sample at T2.

Eighty-seven percent of the participants at T5 (N= 435) took part in all waves of the longitudinal study. Among these participants, 36.1%, 33.5%, 27.4%, and 14.0% smoked cigarettes at T2, T3, T4, and T5, respectively. The mean (SD) family annual income at T5 was \$84,800 (SD=\$66,000). The large standard deviation of income indicates that there was a wide range of income among the women in our sample. Sixty-one percent of the participants had an educational level of high school or lower.

Extensively trained and supervised lay interviewers administered interviews in private at T2, T3, and T4. At T5, the participants were given self-administered questionnaires. Written informed consent was obtained from the participants at each wave. Participants also provided HIPAA (Health Insurance Portability and Accountability Act) authorization as of April, 2002 (the implementation date of this regulation). The Institutional Review Boards of the Mount Sinai School of Medicine and New York Medical College (our former

affiliations), and of the New York University School of Medicine (our current affiliation) approved the procedures used in this research study.

Measures

Cigarette Smoking (T2–T5)—Cigarette smoking from T2 through T5 was assessed by use of a summative index [75]. At each wave of data collection (T2–T5), the participants were asked to report on the amount of their cigarette smoking. The response range was: none (0), less than half a pack a day (1), half a pack to one pack a day (2), and more than one pack a day (3).

Insomnia (T5)—At T5, participants responded to five questions about their sleeping problems, which we had adapted from the DSM-IV-TR [7] diagnostic criteria for insomnia. First, participants reported whether they had experienced one or more of the following sleep problems for a continuous period 2 weeks in the past 12 months: (1) difficulty falling asleep; (2) difficulty staying asleep; and (3) waking up much earlier than necessary. Second, if the participant had endorsed any of the above-cited sleep problems for 2 weeks in the past 12 months, she was then asked about their consequences: if (1) such problem(s) interfered with daily functioning (e.g., daytime fatigue, ability to function at work, memory, concentration, etc.); and (2) how distressed she was about the problem. Participants who answered “moderately,” “severely,” or “extremely severely” to any of the first group of sleep symptoms, and who, in addition, responded “much” or “very much” to at least one of the two consequences of sleep problems, were characterized as having insomnia and assigned a score of 1. Although we did not assess non-restorative sleep, prior research suggests that it is highly related to other criteria included in our insomnia measure [76, 77]. In addition, non-restorative sleep is often symptomatic of medical conditions [78], which were controlled for in the analysis. We also constructed a continuous measure of sleep problems using the 5 items cited above. The internal reliability of the continuous scale was satisfactory (Cronbach’s $\alpha=0.85$).

Depressed Mood (T2–T4)—At T2, T3, and T4, we assessed the participants’ depressed mood [79] (5-item scale; $\alpha=0.80$; e.g., Within the past few years, how much were you bothered by the following: feeling low in energy or slowed down?). Each of the 5 items was scored on a 5 point scale: not at all (1), a little (2), somewhat (3), quite a bit (4), and extremely (5). The mean score of the T2–T4 depressed mood measures was then created and used as a control variable in the analyses. When data were missing, we used the available items to compute the mean score.

Body Mass Index (BMI) (T5)—Our analysis controlled for BMI. BMI is a measure of weight that also takes height into consideration. Height (in inches) and weight (in pounds) were self-reported by participants at T5. BMI was calculated using the following equation [80]:

$$BMI = \frac{Weight}{Height \times Height} \times 703$$

In the equation, 703 was a constant used to account for the conversion between metric and English measures.

Health Conditions (T5)—A control variable of the number of health conditions was used in the analysis. This measure consisted of the participants’ self-reports at T5 of their medical

problems from 1994 to 2009, including diabetes, hypertension, heart disease or any other vascular problems, heart attack, stroke, and asthma.

Analysis

Growth mixture modeling (GMM) analyses were conducted using the Mplus software [81] to identify the developmental trajectories of tobacco use. The dependent variable (frequency and quantity of tobacco use at each time wave) was treated as a censored normal variable¹. The full information maximum likelihood (FIML) approach was applied for missing data in the analysis [82]. Each of the trajectory polynomials was set to be quadratic. The minimum Bayesian Information Criterion (BIC) was used to determine the number of trajectory groups (G). We did not consider groups consisting of less than 5% of the sample because of concern about over-extraction of latent classes. For descriptive analyses, an indicator variable was created for each of the trajectory groups, which had a value of 1 if the participant had the largest Bayesian posterior probability (BPP) for that group, and 0 otherwise. The observed trajectory for a group was the average of tobacco use at each time point for participants assigned to that group. Additionally, we computed the means, standard deviations, and percentages of the other study variables for each smoking trajectory group. We also conducted F-tests or χ^2 tests to test whether the patterns were the same for each trajectory group.

Logistic regression analyses were then conducted using SAS [83] to investigate the associations between the trajectories of tobacco use and insomnia. Since specifying which group an individual belongs to is prone to error, we used the BPP of belonging to each trajectory group as the independent variables (See 84). 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 with insomnia were conducted. Second, multivariate analyses were conducted between the BPPs of the trajectories and insomnia, controlling for T5 age, T5 marital status, T5 BMI, number of health conditions, T2–T4 depressed mood, and T2 educational level. Third, multivariate analyses were conducted between the BPPs of the trajectories and insomnia, controlling for the variables listed above, but not the number of health conditions. In addition, we also examined the interactive effect between the BPPs of the trajectories and T2–T4 depressed mood on insomnia.

RESULTS

Trajectories of Tobacco Use

The solutions for the three-group trajectory (BIC = 2703.44; entropy=0.949), the four-group trajectory (BIC = 2665.94; entropy=0.961), and the five-group trajectory (BIC = 2641.20; entropy=0.965) were calculated. Although the BIC for the 5-group trajectory was lower than that for the 4-group trajectory, we did not consider the 5-group solution because there was one trajectory group that contained only 16 participants (3.2% of the sample). Participants were then assigned to the tobacco use trajectory group that best depicted their smoking over time. The average classification probabilities for group membership ranged from 0.940 to 0.996, which indicates a satisfactory classification.

Figure 1 presents the four observed tobacco use trajectory groups, which were named: a) non-smokers (N=305; 61.2%; mean BPP=99.6%, min. BPP=90.2%, max. BPP=99.8%); b) late quitters (N=85; 17.1%; mean BPP=96.1%, min. BPP=53.9%, max. BPP=100%); c) moderate smokers (N=48; 9.6%; mean BPP=94.0%, min. BPP=65.5%, max. BPP=100%);

¹Alternatively, the dependent variable was treated as an ordinal variable. The results were not substantially different (data not shown).

and d) chronic heavy smokers (N=60; 12.1%; mean BPP=97.5%, min. BPP=65.9%, max. BPP=100%). As noted in Figure 1, the chronic heavy smokers smoked between half and one pack of cigarettes a day from mean age 40 to their mid-sixties. Moderate users smoked less than a half pack a day from mean age 40 to their mid-sixties. The late quitters smoked about a pack of cigarettes a day until mean age 43, reduced their smoking by mean age 48, and then quit completely by mean age 65 (see Figure 1).

Group Membership as a Predictor of Insomnia

Table 2 presents the means and standard deviations (or percentages) of the variables used in the present study by the four smoking trajectory groups. For the whole sample, 9.0% of the participants reported having insomnia. As shown in Table 2, among the smoking trajectory groups, the prevalence of insomnia was: non-smokers (5.9%), late quitters (10.6%), moderate smokers (12.5%), and chronic heavy smokers (20.0%). These fractions differed significantly ($\chi^2(3)=13.4$; $p=0.004$).

The results of the logistic regression analyses indicated that, without controls, as compared with the probability of belonging to the non-smokers group, the probability of belonging to the group of chronic heavy smokers was significantly associated with having insomnia at mean age of 65 years [Odds Ratio (O.R.)=4.07; $p<0.001$]. Multivariate logistic regression analyses were then conducted with controls for T5 age, T5 marital status, T5 BMI, health conditions, T2–T4 depressed mood, and T2 educational level. As shown in Table 3, compared with the probability of belonging to the non-smokers group, the probability of belonging to the chronic heavy smokers group was significantly associated with having insomnia when participants were mean age 65 years [Adjusted Odds Ratio (A.O.R.)=2.76; $p=0.03$]. (See Table 3) The probabilities of belonging to the late quitters group (A.O.R.=1.53; $p=0.36$) or the moderate smokers group (A.O.R.=2.24; $p=0.16$), as compared with the probability of belonging to the non-smokers group, were not significantly associated with having insomnia. Of the controls, T2–T4 depressed mood was significantly ($p<.001$) related to women having insomnia after adjusting for other factors (other control variables and smoking trajectories) in the analyses. In addition, when the number of health conditions was not controlled, the probability of belonging to the chronic heavy smokers group was still significantly associated with having insomnia when participants were mean age 65 years (A.O.R.=2.77; $p=0.03$). We also examined the interactive effects between T2–T4 depressed mood and the trajectory group BPPs. The results indicated that none of the interaction effects were statistically significant ($p>0.05$) with respect to insomnia at T5. These results thus suggest that the effect of smoking trajectories on insomnia was independent of any effect mediated or moderated by depressed mood.

We also conducted linear regression analyses using a continuous measure of sleep problems (i.e., the combined scores of the measures of the three insomnia symptoms and the two consequences of insomnia) as the dependent variable. The findings were essentially the same as with the two-criteria measure described above.

DISCUSSION

This is the first study of the association of smoking trajectories (spanning approximately 25 years) and insomnia among women in late mid-life. Overall, our findings suggest that longitudinal patterns of heavy smoking among women (from mean age 40 to mean age 65 years) are associated with an increased likelihood of insomnia in late mid-life. Women in the continuous heavy smoking group reported more symptoms of insomnia, on average, than members of any other trajectory group. Of note is that there was no significant difference with respect to insomnia at mean age 65 between the late quitters, who reduced their

smoking between mean ages 43–48, and had stopped smoking by late mid-life, and the non-smoking group.

Our results are consistent with the fairly scant literature on smoking and sleep. Zhang and colleagues [85], for instance, showed a longer time to sleep onset (comparable to one of our measures of insomnia) among late mid-life smokers compared to never-smoking matched controls. Further consistent with our findings, these researchers reported no differences in sleep latency between former and never smokers. Sahlin et al. [45] also reported greater sleep latency among smokers in a large sample of adult women. Our results are also supported by Wetter and Young [11], who used the Diagnostic and Statistical Manual of Mental Disorders (Third Edition, Revised; DSM-III-R) [86] criteria to assess insomnia among adults. Findings showed that smokers had more difficulty initiating sleep, and that female smokers had excessive daytime sleepiness.

Although the exact mechanisms which link smoking with insomnia are not yet fully understood, there are several psychosocial and physiological factors that may play a role. Prior research suggests that smokers tend to have higher rates of depression (or depressive symptoms), and lower socioeconomic status (SES), and are less likely to be married than non-smokers [87,88]. Depression is frequently comorbid with insomnia [5,8,10,89], and greater BMI, lower SES (educational attainment and/or income), and divorced, separated, widowed, or cohabitating marital status, are all risk factors for insomnia [2,10,89,90]. Similarly, medical problems may also play a role in the relationship between smoking and insomnia. Smokers are at greater risk than non- or former smokers for respiratory and other medical conditions (e.g., COPD, OSA, pain) [45,53,54,91–93], many of which are associated with sleep problems [89,92,94]. However, our analysis was one of few studies on smoking and sleep that controlled for health conditions, depression, and BMI, in addition to educational level (a proxy for SES), and marital status. Thus, our finding of an association between continuous heavy smoking and insomnia appears to be substantial, since it was maintained with control on these important variables.

One factor that may underlie the link between heavy smoking and insomnia is the role of negative life events. Research has demonstrated that life stressors, e.g., marital or work problems, are important predictors of both smoking and insomnia [36,95,96]. It is possible, therefore, that chronic smokers with sleep problems have come to rely on the behavioral and biochemical effects of cigarette smoking in order to cope, rather than employ more adaptive strategies. Smokers, thus, may be more susceptible to the adverse effect of stressors on sleep. In this vein, Morin and colleagues [97] found that individuals with insomnia reported more stressors and had worse coping techniques.

Smoking (and nicotine) also appear to have both short- and long-term biological effects which could impact sleep. Short-term effects include increased alpha waves (as noted in electroencephalographic studies) and the release of neurotransmitters associated with arousal [49,50] as well as the inhibition of neurotransmitters involved in sleep [49]. In addition, smoking (especially at higher levels) may result in acute nighttime nicotine withdrawal, which could cause sleep disturbance and awakenings [11]. Rieder et al. [98] found that 20% of heavy smokers awoke from sleep due to nighttime nicotine withdrawal. The authors termed this effect “nocturnal sleep-disturbing nicotine craving.” Persistent smoking could also lead to neuroadaptation, such as that seen in nicotine tolerance, which may be unfavorable to sleep. For instance, preliminary evidence has shown lower concentrations of gamma-aminobutyric acid (GABA) among female chronic smokers versus non-smokers [99], as well as among individuals with insomnia [100]. It is also possible that smoking and insomnia share a common genetic diathesis.

Limitations

Although our study is the first to demonstrate a link between smoking trajectories spanning several decades, and insomnia, there are some limitations to our findings. First, our sample was comprised of mostly white women in late mid-life, and thus, the findings may not be generalizable to other populations, such as older men, younger adults, or members of racial/ethnic minority groups. Future research might attempt to test and replicate our results with other age, gender, and racial/ethnic populations. Second, our measures were based on self-report and did not include physiological assessments of either cotinine (the primary nicotine metabolite) or of sleep (e.g., via polysomnography). However, prior research has shown that self-reports of cigarette smoking are valid and reliable [101], and there is some evidence in support of the concordance of self-report and objective assessments of insomnia [102]. Third, we did not collect baseline data on the participants' insomnia and, therefore, were unable to determine whether reports of insomnia at T5, used in the present analysis, represented incident cases or the prevalence of insomnia. However, we used T2–T4 depressed mood as the best available surrogate variable for T2 insomnia. Future research would benefit greatly from the inclusion of baseline data on insomnia. Fourth, we lost some participants due to attrition. Had we been able to include these non-participants in our analyses, we might have had greater variability, which would have strengthened our results. Fifth, in any observational study, there are a number of models that could explain the data equally or nearly equally as well. For example, a variable-centered approach using continuous measures of a history of smoking may also provide significant findings.

Strengths and Conclusions—Despite these limitations, the study has a number of strengths. The group-based trajectory approach has many applications in clinical and therapeutic areas [67]. It provides a powerful statistical tool for summarizing large amount of longitudinal data in a manner that is relatively easy to understand. Moreover, as noted by Nagin & Odgers [67], "...clinical researchers have begun to embrace this new set of tools to evaluate treatment effects and explore individual variation in response to clinical interventions." Furthermore, "this approach enables researchers and clinicians" to test and revise postulations based on "their taxonomic and developmental theories."

In conclusion, our results present evidence that long-term patterns of heavy smoking predict insomnia among women in late mid-life. The clinical implications of our findings highlight the importance of assessing smoking among women with sleep problems, and of referring current smokers with insomnia to smoking cessation or other appropriate treatment programs. From a public health perspective, our longitudinal approach suggests that smoking cessation treatment during the forties could capitalize on and reinforce the naturally occurring decline in smoking among some women during this developmental period. Heavy smokers, who are unable to quit, should also be advised that even a significant reduction of smoking may alleviate the risk of insomnia and its physical, psychosocial, and economic consequences in late mid-life.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.
 Women's Cigarette Smoking Trajectories from Mean Ages 40 to 65 Years (N=498).

Table 1

Demographic Characteristics of the Sample (N=498).

Demographic Characteristics	%
Race/Ethnicity	
White	91.2
Non-White	8.8
Age at T5	
50–60	19.5
61–70	60.2
>70	20.3
Marital Status at T5	
Married	66.4
Single	0.4
Divorced	15.7
Widowed	17.5
Employment Status at T5	
Employed (Full Time and/or Part Time)	40.6
Retired	42.4
Other (e.g., Homemaker, Unemployed)	17.8
Educational Level by T5	
Less than high school	8.1
High school	52.8
Some college or higher	39.1
Annual Personal Income at T5	
\$0–10,000	19.2
\$10,001–35,000	47.9
\$35,001–75,000	28.4
>\$75,000	4.5

Table 2

Mean (Standard Deviation) or Percentage of Sleep Problems and Other Study Variables by the Trajectory Groups of Cigarette Smoking (N=498).

Variables	Non- smokers N=305	Late Quitters N=85	Moderate Smokers N=48	Chronic Heavy Smokers N=60	F test or χ^2 test (p-value)
Insomnia (T5) ^a	5.9%	10.6%	12.5%	20.0%	$\chi^2(3)=13.4$ (p=0.003)
Difficulty Falling Asleep (T5) ^b	0.7 (0.90)	0.8 (1.00)	0.7 (0.94)	1.2 (1.29)	F (3, 494)=4.62 (p=0.003)
Difficulty Staying Asleep (T5) ^b	1.0 (1.02)	1.2 (1.08)	1.1 (1.16)	1.3 (1.14)	F (3, 494)=2.19 (p=0.09)
Waking Up Much Earlier than Necessary (T5) ^b	0.8 (0.96)	1.2 (1.15)	0.9 (1.04)	1.3 (1.20)	F (3, 494)=5.46 (p<0.001)
Interference with Daily Functioning due to Insomnia (T5) ^c	0.7 (0.98)	0.9 (1.12)	0.6 (0.93)	1.3 (1.41)	F (3, 494)=6.66 (p=0.003)
Distress Due to Insomnia (T5) ^c	0.6 (0.94)	0.7 (0.93)	0.6 (1.04)	0.9 (1.27)	F (3, 494)=1.82 (p=0.14)
Depressed Mood (T2–T4) ^d	2.4 (0.69)	2.6 (0.66)	2.5 (0.60)	2.7 (0.70)	F (3, 494)=4.6 (p=0.003)
Health Conditions (T5) ^e	1.0 (0.97)	1.2 (1.06)	1.0 (1.02)	1.1 (1.07)	F (3, 494)=0.74 (p=0.53)
Body Mass Index (T5) ^f	28.5 (6.22)	30.1 (6.86)	30.1 (6.91)	26.5 (5.76)	F (3, 494)=4.57 (p=0.004)
Age (Years) (T5) ^g	66.3 (6.42)	64.5 (5.27)	64.8 (5.70)	61.8 (5.23)	F (3, 494)=10.27 (p<0.001)
Percentage Married (T5) ^g	69.8%	62.4%	68.8%	51.7%	$\chi^2(3)=8.17$ (p=0.04)
Educational Attainment (Years) (T2) ^h	13.3 (2.10)	12.5 (1.77)	12.9 (2.02)	12.2 (2.07)	F (3, 494)=7.25 (p<0.001)

Notes:

T2=Time 2; T3=Time 3; T4=Time 4; T5=Time 5.

Response ranges:

^aNo (0) - Yes (1);

^bNot at all (0); Mildly (1); Moderately (2); Severely (3); Extremely Severely (4);

^cNot at all (0); A little (1); Somewhat (2); Much (3); Very much (4);

^dNot at all (1); A little (2); Somewhat (3); Quite a bit (4); Extremely (5);

^e0–6;

^f16–58;

^g52–84;

*h*₄₋₂₀.

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Table 3

Logistic Regression: Trajectories of Cigarette Smoking with BPP of Belonging to Non-smokers as the Reference Group on Insomnia at T5 (N=498).

Independent Variables	Moderate to Severe Insomnia A.O.R. (95% C.I.)
BPP of Belonging to Chronic Heavy Smokers Group compared to the BPP of belonging to the non-smokers reference group	2.76 (1.10–6.92) *
BPP of Belonging to Late Quitters Group compared to the BPP of belonging to the non-smokers reference group	1.53 (0.61–3.81)
BPP of Belonging to Moderate Smokers Group compared to the BPP of belonging to the non-smokers reference group	2.24 (0.74–6.77)

Notes:

*
p<0.05.

Four smoking trajectories groups were identified: chronic heavy smokers, late quitters, moderate smokers, and non-smokers.

BPP=Bayesian posterior probability; A.O.R.=adjusted odds ratio; C.I.=confidence interval.

T5 age, T5 marital status, T5 body mass index (BMI), health conditions, T2–T4 depressed mood, and T2 educational level were controlled for.