**The Interaction between Anxiety Sensitivity and Cigarette Smoking Level in Relation to
Sleep Onset Latency among Adolescent Cigarette Smokers**

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Abstract

Cigarette smoking during adolescence is linked to a number of sleep disturbances and has been consistently linked to sleep onset latency among adults. However, little research has examined factors that may influence the relation between cigarette smoking level and sleep onset latency among adolescents. One factor that may be particularly important in this regard is anxiety sensitivity (AS). The current study examined whether cigarette smoking level interacted with AS in its association with sleep onset latency among 94 adolescent ($M_{age} = 15.72$) cigarette smokers. As hypothesized, AS interacted with smoking level to relate to sleep onset latency, even after controlling for age and gender. This relation was specific to sleep onset latency as opposed to other types of sleep disturbances, and that adolescents who smoked at higher levels tended to go to sleep later and wake up later than adolescents who smoked at relatively lower levels.

Keywords: Adolescents, Cigarettes, Anxiety Sensitivity, Sleep Onset Latency

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Evidence suggests that up to 36% of adolescents report sleep disturbances, with between 20 and 26% of youth reporting difficulty initiating sleep (Gradisar, Gardner, & Dohnt, 2011). Although various studies suggest adolescent cigarette smoking is associated with sleep difficulties (e.g., reduced total sleep; Pasch, Latimer, Cance, Moe, & Lytle, 2012; Townsend, Wilkes, Haines, & Jarvis, 1991; Wong, Brower, & Zucker, 2009), relatively little adolescent-focused research has examined links between smoking and sleep onset latency specifically. Research with adults suggests cigarette smoking prospectively relates to sleep onset latency, but not with other sleep disturbances such as wake after sleep onset or early morning awakenings (Wetter & Young, 1994). Additional evidence suggests that, compared to healthy controls, adult smokers show significantly prolonged sleep onset, and that sleep onset latency increases for every cigarette smoked per day (Cohrs et al., 2014). Further, using objective measures of sleep onset latency (i.e., in-home polysomnograms), cigarette smokers show longer sleep onset latency than non-smokers (Zhang, Samet, Caffo, & Punjabi, 2006). These findings persist when controlling for alcohol and substance use, psychiatric problems or endocrine diseases, and treatment with any kind of psychotropic medication (Jaehne et al., 2012).

In one of the few studies to examine the association between smoking and subjective sleep onset latency among adolescents, cigarette smoking at baseline predicted difficulties falling or staying asleep one year later (Patten, Choi, Gillin, & Pierce, 2000). However, a recent meta-analysis found that although there is substantial evidence linking cigarette smoking and sleep onset latency among adults, evidence for this relation among youth is less clear, with a handful of studies finding support for, and others against, a sleep latency-cigarette smoking relation

(Bartel, Gradisar, & Williamson, 2015). This inconsistency may be due to the relative dearth of research studies in the area, which reflects a critical gap in the literature given the unique biopsychosocial aspects of adolescence. Adolescents are more sensitive to drugs (Cicchetti & Rogosch, 2002; Monti et al., 2005; Winters & McLellan, 2004), including nicotine (Strong, Schonbrun, Schaffran, Griesler, & Kandel, 2012). Additionally, adolescents tend to be more temporally proximal to smoking initiation than adults (Johnston, O'Malley, & Terry-McElrath, 2004). Finally, adolescence is a developmental period during which adult sleep patterns and behaviors begin to emerge. Most youth experience a sleep-phase shift in which, for a variety of reasons, they begin going to sleep later than in childhood and early adolescence (McGlinchey, 2015). Adolescents who smoke cigarettes may be particularly likely to experience prolonged sleep onset given nicotine is a stimulant that may increase arousal and difficulty initiating sleep (e.g., Kishimoto & Domino, 1998; Myrsten, Elgerot, & Edgren, 1977; Parrott, 1998). Indeed, evidence suggests that adolescents (Urbán, Magyaródi, & Rigó, 2011) and adults (Broms et al., 2011) who report smoking are more likely to report being evening types (i.e., going to sleep later and waking up later) than individuals who have not tried smoking, suggesting smoking may be linked to delayed sleep onset.

The inconsistent findings regarding relations between cigarette smoking and sleep onset latency among adolescents (Bartel et al., 2015) may be due to third factors that influence the relation between smoking and sleep onset. In particular, anxiety has been implicated in the link between cigarette smoking and sleep disturbances among adolescents, with evidence suggesting that the cigarette smoking - sleep disturbance association may be stronger in the context of anxiety symptoms (e.g., Johnson & Breslau, 2001). This suggests that examining anxiety-related

vulnerabilities may be critical for advancing our understanding of the links between cigarette smoking and sleep onset latency among adolescents.

One factor that has been linked to both cigarette smoking (e.g., Leventhal & Zvolensky, 2014) and sleep disturbances (e.g., Gregory & Eley, 2005) is anxiety sensitivity (AS). AS is defined as fear of the negative consequences of anxiety-related sensations (Reiss & McNally, 1985). As an illustrative example, an adolescent high in AS may be afraid that shortness of breath means s/he is not going to be able to breathe, while an adolescent low in AS may view the same sensation as non-threatening. Factor analyses suggest the global AS factor is comprised of three lower-order factors reflecting domain-specific concerns about the consequences of anxiety-related sensations (i.e., physical, mental, and social concerns; Brown et al., 2012; Walsh, Stewart, McLaughlin, & Comeau, 2004; Wright et al., 2010).

There is reason to believe that elevated AS may be associated with sleep onset latency. Individuals high in AS may be attending to physical sensations (e.g., symptoms of arousal) at night while trying to fall asleep, which could lead to extended sleep onset latency. Evidence suggests that negatively valenced cognitive activity adversely impacts sleep. For example, catastrophizing cognitions (e.g., worrying about how delayed sleep onset could affect school the next day) are linked to prolonged sleep onset among adolescents (Hiller, Lovato, Gradisar, Oliver, & Slater, 2014). This suggests that cognitive activity characterized by perseverating negatively on somatic arousal, as may be the case with AS, is associated with prolonged sleep onset latency. For example, the physical component of sleep anticipatory anxiety (e.g., “When I go to sleep, my heart beats fast”) was more strongly related to sleep onset latency in individuals who were higher in the physical concerns facet of AS than in individuals who reported low levels of AS - physical concerns (Babson, Trainor, Bunaciu, & Feldner, 2008). Additionally, among

adults with panic disorder, elevated AS evidences a unique relation with prolonged sleep onset (Hoge et al., 2011).

A small, but emerging, literature suggests AS may also be associated with sleep disturbances among adolescents. For example, evidence suggests that after controlling for anxiety symptoms, AS (in particular, AS mental concerns) is uniquely associated with sleep disturbances among youth between 8 and 11 years old (Gregory & Eley, 2005). Additionally, among 101 6 to 17-year-old youth with anxiety disorders, elevated global AS was uniquely associated with prolonged sleep onset latencies even after controlling for age, severity of anxiety, and depression (Weiner, Meredith Elkins, Pincus, & Comer, 2015).

Despite evidence suggesting that both AS and cigarette smoking are individually associated with sleep onset latency, AS has not been examined in terms of the relation between cigarette smoking level and sleep onset latency among adolescents. This is a notable gap in the literature, as there is reason to believe that AS may influence this association. An adolescent cigarette smoker who is high in AS will likely experience greater anxiety when experiencing smoking-related arousal than an adolescent smoker lower in AS. Indeed, while cigarette smoking may have short term anxiolytic effects for individuals who are elevated in AS, smoking induced sensations may *increase* fear of symptoms over time (Leventhal & Zvolensky, 2014). Consequently, the high-AS adolescent cigarette smoker will likely report longer sleep onset latency than the low AS smoker. Importantly, no work has examined the interplay between AS and cigarette smoking level in relation to sleep onset latency among adolescent smokers.

The current study is designed to address this gap in the literature by examining how AS and cigarette smoking level interact to relate to sleep onset latency among adolescent cigarette smokers. Examining this relation may offer one potential explanation for observed associations

between adolescent cigarette smoking and being an evening type. Two main hypotheses were tested. First, it was predicted that smoking level and AS would relate positively to sleep onset latency. Second, it was hypothesized that AS would interact with smoking level and affect the association with sleep onset latency among adolescent cigarette smokers such that as levels of AS increased, smoking level would be more strongly, positively related to extended sleep onset latency. To obtain a better sense of sleep disturbances among adolescent smokers, we also explored how AS interacted with cigarette smoking level in relation to other sleep disturbances and differences in adolescent sleep-wake times. Finally, given evidence that gender (e.g., Branstetter, Blosnich, Dino, Nolan, & Horn, 2012; Zundert, Roedel, Engels, & Scholte, 2015) and age (e.g., Alfano, Ginsburg, & Kingery, 2007; Reidpath, Davey, Kadirvelu, Soyiri, & Allotey, 2014) may play an important role in adolescent sleep and smoking behavior, both variables were entered as covariates in order to provide a conservative test of the proposed hypotheses.

Method

Participants

Ninety-four adolescents ($n = 50$ males) between the ages of 12 and 17 years ($M_{\text{age}} = 15.72$; $SD = 1.38$) were recruited from the local community to participate in a larger study examining the link between cigarette smoking and adolescent psychological functioning. Adolescents were recruited in equal numbers across four stages of smoking uptake (e.g., Mayhew, Flay, & Mott, 2000); triers ($n = 26$; $M_{\text{cig/day}} = 0$, $SD = 0$), experimenters ($n = 23$; $M_{\text{cig/day}} = 0.3$, $SD = 0.64$), regular smokers ($n = 15$; $M_{\text{cig/day}} = 2.63$, $SD = 1.88$), and daily smokers ($n = 30$; $M_{\text{cig/day}} = 10.53$, $SD = 7.44$). As the larger study included a voluntary hyperventilation procedure, exclusionary criteria were as follows: a) respiratory problems (e.g., asthma), b) heart

problems (e.g., hypertension), c) pregnancy, d) the inability to provide written informed consent/parental assent, and e) lifetime history of panic disorder. Participants for the current study were selected from the larger sample ($n = 180$) based on a) reporting they had at least tried cigarette smoking ($n = 130$), and b) completion of all measures for the current study. Chi-square tests were conducted to examine differences between individuals who completed all measures and those who did not. No significant differences were observed between completers and non-completers in terms of gender $\chi^2(1) = .039, p = .882$ or ethnicity $\chi^2(1) = 1.64, p = .227$, although independent samples t-tests indicated that completers ($M_{age} = 15.72, SD = 1.38$) were older than non-completers ($M_{age} = 14.65, SD = 1.61$), $t(178) = -4.82, p < .001$. Racial and ethnic composition of the current sample reflected the local community: approximately 88.2% Caucasian, 5.4% African American, 3.2% Native American/Alaska Native, and 3.2% “other.”

Measures

The *Childhood Anxiety Sensitivity Index* (CASI; Silverman, Fleisig, Rabian, & Peterson, 1991) was used to measure anxiety sensitivity. The CASI is an 18-item measure that utilizes a 3-point scale (1 = *None*, 2 = *Some*, 3 = *A lot*) to answer questions such as, “unusual feelings in my body scare me.” It has a hierarchical structure, with one global factor and three subfactors (i.e., physical concerns, social concerns, and cognitive concerns; Silverman et al., 1991; Wright et al., 2010). The current study was interested in the global factor, which represents a generalized fear of the consequences of anxiety related sensations, all of which may pose problems for adolescents trying to fall asleep. The CASI is a psychometrically sound instrument that is frequently used in the child and adolescent psychopathology literature. Internal consistency for the CASI in the current study was similar to previously published work ($\alpha = 0.83$; Knapp, Frala, Blumenthal, Badour, & Leen-Feldner, 2013; Graham & Weems, 2015).

The *Pittsburgh Sleep Quality Index* (PSQI; Buysee, Reynolds, Monk, Berman, & Kupfer, 1989) is a 19-item self report measure that was used to index self-reported sleep quality over the preceding month. The PSQI measures sleep quality using two different formats: 1) open-ended questions in which answers are coded using established categories (e.g., if a participant indicates that he or she sleeps 7 hours per night, his or her answer will be coded as “0”), and 2) a 14-item scale on which participants answer questions such as “During the past month, how often have you had trouble sleeping because you wake up in the middle of the night or early morning” using a 4-point Likert-type scale (0 = *Not during the past month*, 1 = *Less than once a week*, 2 = *Once or twice a week*, 3 = *Three or more times a week*). The PSQI consists of seven component scores: subjective sleep quality, sleep duration, sleep onset latency, sleep disturbances, habitual sleep efficiency, sleep medication, and daytime dysfunction. The seven component scores are summed to yield a global score of sleep quality. The PSQI has good psychometric qualities (Buysee et al., 1989) and has successfully been used with adolescents (Babson, Feldner, Connolly, Trainor, & Leen-Feldner, 2010; Noone et al., 2014). The sleep onset latency subscale is composed of two questions, “During the past month, how often have you had trouble sleeping because you could not get to sleep within 30 minutes” using a four point Likert-type scale response, and an open ended question, ““During the past month, how often have you had trouble sleeping because you could not get to sleep within 30 minutes.”

Given all participants included in the current study endorsed at least trying cigarettes, smoking level was measured using a single continuous question from the *Smoking History Questionnaire* (SHQ), a smoking questionnaire developed by the National Cancer Institute consensus panel. Participants were asked to indicate the number of cigarettes smoked per day. The SHQ has been successfully used with adolescents in past research (e.g., Dino et al., 2001;

Horn et al., 1999), and it indexes a number of issues related to cigarette smoking including smoking frequency and patterns of use.

Procedures

Participants who contacted the laboratory in response to community-based advertisements were provided with a description of study procedures and administered a brief telephone screener as an initial evaluation of the study exclusion criteria listed above. Eligible participants and their parent or guardian were scheduled for a laboratory visit. Parental consent for adolescent participation and adolescent assent were obtained upon arrival to the laboratory. Adolescents completed self-report measures in a quiet, private room, with a trained researcher on hand to answer questions. Thereafter, they took part in a series of laboratory-based assessments (e.g., voluntary hyperventilation) and, upon completion, were thanked, debriefed, and compensated \$45.

Analytic Approach

Following the computation of descriptive statistics and correlations, separate hierarchical multiple regression analyses were conducted using the PROCESS custom dialog in SPSS 19 (Hayes, 2013) to examine study hypotheses. The model was divided into three levels: (1) age and gender were entered at level 1; (2) main effects of smoking level and CASI scores were entered at level 2; and (3) the interaction term was entered at level 3. Main effect variables were mean centered prior to computing product terms (Aiken & West, 1991). Post-hoc probing of significant interactions was conducted using PROCESS to examine the predictor criterion at high (i.e., 1 standard deviation about the mean), mean, and low (i.e., 1 standard deviation below the mean) levels of the interaction term. Significant interactions were graphically depicted. For the purposes of this investigation, sr^2 was used as an index of effect size with small, medium, and

large effects being represented by sr^2 values of 0.01, 0.09, and 0.25 respectively (Cohen, Cohen, West, & Aiken, 2003).

Inspection of the data revealed some variables (i.e., CASI, smoking level) were positively skewed, which is relatively common when studying psychopathology in nonclinical samples recruited from the general population (Wright, London, & Field, 2011). Following the recommendations of Wright and colleagues, analyses were performed outside of PROCESS using bootstrapping, a powerful technique that is recommended for use with smaller samples and serves as an effective remedy for non-normality (Hayes, 2013). Results when using bootstrapping were not different from results without bootstrapping, therefore, non-bootstrapped results from the PROCESS custom dialog are presented.

Results

Descriptive Analyses and Zero Order Correlations

See Table 1 for zero order correlations and descriptive statistics. Means and standard deviations for the CASI and PSQI were similar to previously published findings with adolescent samples (Knapp, Blumenthal, Mischel, Badour, & Leen-Feldner, 2015; Zhou et al., 2012). As expected, CASI scores correlated positively with all of the subscales of the PSQI. Number of cigarettes smoked per day was not associated with CASI total score, and daytime dysfunction was the only subscale of the PSQI with which smoking level was positively correlated. There were no significant gender differences in sleep onset latency scores, $t(92) = -.489, p = .626$, or smoking level, $t(92) = .120, p = .205$, but girls reported elevated CASI scores compared to boys, $t(92) = -2.91, p = .004$. Age was not significantly related to any predictor or criterion variables except smoking level, where older adolescents reported higher levels of smoking.

Primary Hypothesis Tests

Table 2 includes results of regression analyses. As hypothesized, a significant omnibus regression model emerged, $F(5,88) = 9.60, p < .001$, explaining 18.24% of the variance in sleep onset latency scores. Age and gender did not account for significant variance in sleep onset latency scores in step 1. In step 2, CASI, $\beta = 0.06, p < .001, sr^2 = 0.09$, and number of cigarettes smoked per day, $\beta = 0.03, p = .052, sr^2 = 0.01$, were significantly related to prolonged sleep onset latency. As hypothesized, at step 3, there was a significant interaction between number of cigarettes smoked per day and CASI scores, $\beta = 0.01, p < .001, sr^2 = 0.09$. Post hoc probing analyses (as depicted in Figure 1) suggested number of cigarettes smoked per day was associated with prolonged sleep onset latency, but only for adolescents who were at high or mean levels of AS. Number of cigarettes smoked per day was not related to sleep onset latency among low-AS adolescent smokers¹.

Exploratory Analyses

Exploratory analyses examined whether the interaction between AS and smoking level related to other sleep disturbances in adolescents. Results are presented in Table 2. AS and smoking level did not interact to relate to any other sleep difficulties. Independent samples *t*-tests were used to examine differences in sleep – wake patterns between adolescents who reported smoking at least daily ($n = 50; M_{cig/day} = 7.91, SD = 7.10$) as compared to those who did not ($n = 44; M_{cig/day} = 0, SD = 0$) in order to better understand this pattern of results. Results suggested that daily smokers went to bed later [$t(92) = -3.04, p = .003$] and woke up later than non-daily smokers, $t(81.53) = -2.53, p = .013$. To ease interpretation, Table 3 presents sleep-wake times grouped as categories, and indicates the frequency with which daily and non-daily smokers endorsed going to bed during these time periods.

Discussion

Improving our understanding of factors that relate to sleep onset latency among adolescents is critical for reducing consequences associated with sleep disturbances during adolescence. Cigarette smoking is associated with sleep onset latency among adults (e.g., Cohrs et al., 2014), although evidence for this association among adolescents is less clear (Bartel et al., 2015), suggesting there is a need to refine our understanding of these relations among adolescents. Although evidence suggests AS is associated with sleep disturbances in youth (e.g., Gregory & Eley, 2005) and may moderate the relation between sleep anticipatory anxiety and sleep onset latency among adults (e.g., Babson et al., 2008), no work has examined links among AS, cigarette smoking, and sleep onset latency among adolescents. Therefore, to begin to address this gap in the literature, the current study tested the hypothesis that AS would interact with cigarette smoking level in relation to sleep onset latency among adolescent cigarette smokers.

A number of notable findings emerged. First, as hypothesized, and consistent with research linking AS to sleep onset latency in adolescents (Weiner et al., 2015), AS related positively to sleep onset latency; this effect was of a medium magnitude ($sr^2 = 0.09$). Also consistent with hypotheses, the association between smoking level and sleep onset latency was significant, this effect was of a small magnitude ($sr^2 = 0.01$). Further, even after accounting for variance attributable to each main effect, age, and gender, AS interacted with smoking level in its association with sleep onset latency among adolescent cigarette smokers; this effect was of a medium magnitude ($sr^2 = 0.09$). Specifically, as levels of AS increased, smoking level was more strongly, positively related to prolonged sleep onset latency among adolescents. In contrast, among adolescents with relatively lower levels of AS, smoking level was less strongly associated with sleep onset latency. Exploratory analyses indicated that AS did not appear to interact with

smoking level to affect the relation between smoking level and any other sleep disturbances. In an effort to understand why AS and smoking level interacted to relate to sleep onset latency and not other sleep disturbances, additional analyses were run to examine differences in sleep-wake times as a function of smoking level. Results indicated that adolescents who smoked at higher levels (i.e., daily smokers) went to bed later and woke up later than adolescents who smoked at lower levels (i.e., non-daily smokers).

These findings provide preliminary support for the idea that smoking is associated with prolonged sleep onset latency among adolescent smokers who report elevated levels of AS. These results also suggest that daily adolescent smokers may be staying up later and waking up later than non-daily smokers. Later sleep times could have a particularly deleterious effect on adolescents who have to get up early to go to school (Owens, Belon, & Moss, 2010). Indeed, insufficient sleep related to normative shifts in adolescents' circadian rhythms that conflict with early school start times have been implicated in a wide array of functional problems among adolescents (Carskadon & Acebo, 2002; Carskadon, Mindell, & Drake, 2006). Additionally, evidence suggests that cigarette smoking is associated with later bed times and wake times in adolescents and adults (Broms et al., 2011; Urban et al., 2011). The current findings suggest one pathway whereby this relationship may emerge. It may be that youth who smoke cigarettes have difficulty falling asleep, even when they go to bed earlier. Taken together, it is possible that cigarette smoking may contribute to poor academic (Curcio et al., 2006) and mental health outcomes (Alfano et al., 2009) among youth high in AS because of its effects on sleep, although additional research is needed to directly test such hypotheses. It also is possible that other factors serve as diatheses for each of these problems. For example, impulsivity and sensation seeking have been linked to both being an evening type and to smoking (Adan, Natale, Caci, & Prat,

2010; Tonetti et al., 2010). More work is needed to further disentangle these relations, but the current results may suggest one path whereby cigarette smoking contributes to poor academic (Curcio et al., 2006) and mental health outcomes (Alfano et al., 2009) among youth.

These findings are particularly interesting when considered in the broader context of sleep difficulties among adolescents and adults. A large body of evidence suggests that individuals with insomnia tend to overestimate their sleep onset latency and underestimate their total sleep time relative to objective measures of sleep (e.g., polysomnograms; Harvey & Tang, 2012). In a large review of the existing literature examining the discrepancy between objective and subjective measures of sleep onset latency, Harvey and Tang (2012) proposed a number of possible explanations for this disconnect, including the notion that elevated physiological arousal may increase mental activity when trying to fall asleep (Bonnet & Arand, 1992). Increased pre-sleep mental activity could cloud the division between sleep and wakefulness, making it more difficult to detect the point of sleep onset. In addition, selective attention and monitoring of bodily sensations (e.g., bodily sensations consistent or inconsistent with falling asleep) are key characteristics of insomnia (Harvey & Tang, 2012). Nonetheless, among children and adolescents, self-reported difficulties with sleep onset latency (cf., objective measures) appear linked to psychopathology (e.g., anxiety disorders; Alfano, Patriquin, & De Los Reyes, 2015). Accordingly, continued work aiming to better understand how anxiety-related vulnerabilities and smoking may influence both perceived and objectively measured sleep onset is needed.

These processes may be critical for understanding why relatively heavier smoking adolescents who reported higher levels of AS reported greater sleep onset latencies than heavier smoking adolescents who are lower in AS. Nicotine is a stimulant that increases arousal (Kishimoto & Domino, 1998; Myrsten et al., 1977; Parrott, 1998), which may, in and of itself,

prolong sleep onset latency. Further, adolescent cigarette smokers who are elevated in AS may be particularly prone to selectively attend to and monitor symptoms of arousal stemming from cigarette use and nicotine withdrawal while trying to fall asleep. This mental activity could make it more difficult for these adolescents to accurately notice the point at which they transition from wake to sleep. However, additional research is needed to further understand the factors contributing to this pattern of results. For example, it will be important to examine how AS and smoking level interact to predict objective measures (i.e., polysomnography, actigraphy) of sleep onset latency.

These findings represent a novel contribution to the literature examining smoking and sleep disturbances among adolescents. A number of study limitations nonetheless merit consideration. First, this study was correlational and cross-sectional. While useful as preliminary evidence (Kraemer, Yesavage, Taylor, & Kupfer, 2000), the current study permits neither causal inferences nor inferences regarding how the variables examined relate to each other across time. Future work would benefit from examining these processes both prospectively and experimentally to examine how AS and cigarette smoking may interact over time to influence sleep onset latency. Second, as alluded to above, these findings were based on self-report measures, suggesting these results may be due, in part, to shared method variance. Further work is needed to better disentangle these processes. For example, these results may be specific to self-reported sleep onset latency and may not generalize to objective measures of sleep onset latency. An additional limitation of the current study is the lack of sleep diary use. Future work would benefit from using sleep diaries to measure sleep onset latency. Third, the sample consisted primarily of Caucasian adolescents who self-selected to participate in research for monetary compensation, and had parents willing and able to accompany them to the laboratory.

Future work would benefit from improving generalizability by examining these processes in a more diverse sample. Next, the current study did not covary for current drug and alcohol use. Given associations among drug, alcohol, and cigarette use in adolescents (e.g., Ehrenreich, Nahapetyan, Orpinas, & Song, 2015), future work would benefit from including these important covariates. An additional limitation is that these data were collected both during the summer and school year, which could affect the times when adolescents in the study were going to bed and waking up. Future research would benefit from employing a more tightly controlled sleep schedule to account for variability in adolescent sleep schedules across the year. Finally, the current study did not examine how psychopathology may have affected these processes. Given evidence that internalizing symptoms are linked to both cigarette use and sleep disturbances among adolescents (Johnson & Breslau, 2001) future work would benefit from examining how these processes may be affected by the presence of anxiety and mood disorders.

Taken together, these findings suggest that adolescent daily smokers who endorse relatively elevated AS may be particularly likely to experience prolonged sleep onset latency, perhaps due to an increased focus on arousal stemming from cigarette use while trying to fall asleep. This is an important extension to the extant literature, which currently includes little data regarding these processes among adolescents. These findings also suggest that daily adolescent cigarette smokers go to bed later and wake up later than non-daily adolescent smokers. These results help clarify mixed results regarding the relation between adolescent cigarette smoking and sleep onset latency (Bartel et al., 2015), and demonstrate the importance of extending beyond main effects to examine how multiple factors interact to affect sleep onset latency. The results of this study lay the groundwork for future research examining specific mechanisms that contribute to sleep onset latency among adolescent smokers. More research is now needed to

better understand the complex relations among anxiety sensitivity, cigarette use, and sleep disturbance among adolescents.

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Footnotes

¹ Hypotheses were also evaluated using the single continuous sleep latency item (“how many minutes does it take you to fall asleep at night?”) from the PSQI as a dependent variable. Results of these analyses indicated that there was a main effect of AS, and that AS and smoking level interact in their relation to sleep onset latency. This represented a small effect.

Table 1.

Descriptive Statistics and Zero Order Correlations among Predictor, Indirect Effect, and Outcome Variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Smoking level	1	-.094	.077	-.007	-.067	-.078	.042	-.010	.211*	0.028	.332**
2. CASI: Global		1	.290**	.289**	.290**	.212*	.509***	.246*	.386***	.477***	.000
3. Sleep Onset Latency			1	.313**	.532***	.246*	.523***	.131	.560***	.743***	.055
4. Subjective Sleep Quality				1	.299**	.260*	.298**	-.035	.268**	.567***	-.089
5. Sleep Duration					1	.500***	.434***	.238*	.447***	.778***	.098
6. Habitual Sleep Efficiency						1	.297***	-.025	.138	.574***	-.001
7. Sleep Disturbances							1	.337**	.485***	.713***	.100
8. Sleep Medication								1	.423***	.412***	.106
9. Daytime Dysfunction									1	.707***	.139
10. PSQI Global Score										1	0.078
11. Age											1
<i>M</i>	3.86	28.00	1.47	0.60	0.74	0.65	1.39	0.46	1.20	6.51	15.72
(<i>SD</i>)	(6.31)	(5.75)	(1.01)	(1.00)	(0.90)	(1.01)	(0.64)	(0.80)	(0.74)	(3.92)	(1.38)

Notes: $n = 94$. CASI: Childhood Anxiety Sensitivity Index; PSQI: Pittsburgh Sleep Quality Index.* $p < .05$ ** $p < .01$ *** $p < .001$

Table 2:
CASI and Smoking Status as Predictors of Sleep Disturbances

	ΔR^2	β	t
<i>Dependent Variable: Sleep Onset Latency</i>			
Step 1	0.01		
Age		-0.00	-0.02
Gender		-0.00	-0.00
Step 2	0.09*		
CASI- total score		0.06***	3.55
Smoking Status		0.03*	1.97
Step 3	.09***	.09	.01
CASI-total x Smoking Status		0.01***	3.36
<i>Dependent Variable: Subjective Sleep Quality</i>			
Step 1	0.03		
Age		-0.08	-0.86
Gender		0.21	0.87
Step 2	0.07*		
CASI- total score		0.05*	2.54
Smoking Status		0.02	0.92
Step 3	.03	.09	.01
CASI-total x Smoking Status		0.01	1.40
<i>Dependent Variable: Sleep Duration</i>			
Step 1	0.03		
Age		0.08	.95
Gender		0.17	.21
Step 2	0.07*		

		CASI- total score	0.04*	2.44
		Smoking Status	-0.01	-0.35
Step 3	.01		.09	.01
		CASI-total x Smoking Status	0.00	0.99
<i>Dependent Variable: Sleep Efficiency</i>		.00		
Step 1				
		Age	0.01	0.10
		Gender	-.15	-.69
Step 2	.05			
		CASI- total score	0.04	1.79
		Smoking Status	-0.01	-0.37
Step 3	.00		.09	.01
		CASI-total x Smoking Status	0.00	0.33
<i>Dependent Variable: Sleep Disturbances</i>		.06		
Step 1				
		Age	0.04	0.83
		Gender	0.12	0.93
Step 2	.22***			
		CASI- total score	0.06***	3.43
		Smoking Status	0.09	0.72
Step 3	.00			
		CASI-total x Smoking Status	0.00	0.64
<i>Dependent Variable: Sleep Medication</i>		.01		
Step 1				
		Age	0.06	0.85
		Gender	0.00	0.01

Step 2	.06		
CASI- total score		0.04**	2.82
Smoking Status		0.00	0.14
Step 3	.02		
CASI-total x Smoking Status		0.00	1.44
<i>Dependent Variable: Daytime Dysfunction</i>	.02		
Step 1			
Age		0.03	0.60
Gender		-0.06	-0.39
Step 2	.19***		
CASI- total score		0.06***	4.22
Smoking Status		0.03*	2.34
Step 3	.02	.09	.01
CASI-total x Smoking Status		0.00	1.47

Note: $n = 94$. CASI: Childhood Anxiety Sensitivity Index. Gender was dummy coded (0 = male, 1 = female).

* $p < .05$

** $p < .01$

*** $p < .001$

Table 3.

Sleep Time by Smoking Level

	Non-Daily Smokers	Daily Smokers
	<i>n</i> (%)	<i>n</i> (%)
<i>Time to Bed</i>		
Before 10:00 pm	11 (25%)	3 (6%)
Between 10:00 pm and 12:00 am	27 (61%)	34 (68%)
After Midnight	6 (14%)	13 (26%)
<i>Time to Wake</i>		
Before 6:00 am	3 (7%)	4 (8%)
Between 6:00 am and 7:59am	28 (64%)	25 (50%)
Between 8:00 am -10:00 am	10 (23%)	10 (20%)
After 10:00 am	4 (9%)	10 (20%)

Figure 1. Interaction of AS Total Score x Smoking Level in predicting sleep onset latency.