Published in final edited form as:

Am J Addict. 2015 March ; 24(2): 166–172. doi:10.1111/ajad.12158.

Smoking-Related Correlates of Psychomotor Restlessness and Agitation in a Community Sample of Daily Cigarette Smokers

Jordan A. Wong, MPH¹ and Adam M. Leventhal, PhD^{1,2}

¹Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, California

²Department of Psychology, University of Southern California, Los Angeles, California

Abstract

Background and Objectives—Psychomotor restlessness and agitation (PMA) is a putatively important, yet understudied, psychopathologic correlate of smoking. The scant smoking research on PMA previously conducted has been narrow in scope and conducted among psychiatric patients. To examine the generalizability and relevance of PMA to smoking, this cross-sectional study investigated associations between PMA and a variety of smoking processes in a community sample.

Methods—Participants in this study were non-treatment-seeking smokers ($N = 254$, 10 cig/day, M age = 44 years) from the community without an active mood disorder. At baseline, they completed a PMA symptom checklist, a composite depressive symptom index, and a battery of smoking questionnaires.

Results—Linear regression models adjusting for depressive symptoms and demographics indicated that PMA level was positively associated with severity of nicotine withdrawal symptoms during prior quit attempts ($\beta = .18, p < .05$), anticipated likelihood of withdrawal in a future quit attempt ($\beta = .19, p < .05$), motivation to smoke for negative reinforcement ($\beta = .14, p < .05$), and smoking expectancies for negative reinforcement ($\beta = .17, p < .05$), negative consequences ($\beta = .22, p < .01$), and positive reinforcement ($\beta = .14, p < .05$). PMA was not significantly associated with smoking chronicity, frequency, or dependence severity.

Conclusion and Scientific Significance—Smokers with elevated PMA appear to experience greater smoking-induced affect modulation and nicotine withdrawal than the average smoker, regardless of other depressive symptoms. Given that PMA differentiates a qualitatively unique profile of smoking characteristics, PMA warrants consideration in tobacco addiction research and practice.

BACKGROUND AND OBJECTIVES

The majority of research on the role of psychopathology in addiction has focused on diagnostic syndromes. This syndrome-based approach overlooks the phenotypic heterogeneity of many psychiatric syndromes and the possibility that certain phenotypic expressions of psychiatric syndromes may be more relevant to substance use than others. Psychomotor restlessness and agitation (PMA) is a phenotypic expression of psychopathology that presents in several psychiatric disorders, including schizophrenia, delirium, mania, and depression.^{1,2} PMA refers to unintentional motor activity stemming from mental tension, manifested by physical signs, such as fidgeting, pacing, moving, stirring, shaking, and restlessness. PMA is commonly considered a symptom of depression and is a criterion for a *DSM-V* major depression diagnosis.^{1,3}

PMA may also reflect a unique depressive phenotype that distinguishes different subtypes or dimensions of depression. Depression with (vs. without) PMA tends to re-occur across multiple depressive episodes and exhibits a unique pattern of correlations with demographic characteristics, other depressive symptoms, and several non-depressive psychiatric conditions.^{4,5} In samples of depressed individuals, the prevalence depression with (vs. without) PMA ranges from 31.3% (vs. 68.4%) to 52.1% (vs. 47.9%).⁵⁻⁷ Hence, investigating PMA as a stand-alone phenotype may aid in isolating the source of psychopathological risk for addictions that is not uncommon in the population.

Emerging data also indicates that depression with PMA (vs. without) is associated with increased prevalence of several substance use disorders, including alcohol, cocaine, and opioid dependence, and more recently nicotine dependence.^{5,6,8,9} For instance, in a study of psychiatric outpatients with an array of depressive symptoms, those with current nicotine dependence (vs. past/no history of nicotine dependence) demonstrated an increased severity of PMA, suggesting the prevalence of depression with PMA in nicotine dependence.¹⁰ Another study indicated that PMA was associated with higher rates of nicotine dependence incrementally to bipolar disorder.⁹

Despite growing evidence-linking PMA to nicotine dependence, several important points remain unclear. First, prior work on PMA and smoking has been conducted in samples of psychiatric patients. Hence, it is unclear whether PMA associates with smoking behaviors in general community samples with relatively low acute psychiatric distress and a wide range of variation across the lower end of the continuum of mental health functioning. Examining PMA-smoking relations in individuals within the general community may yield findings that explain variation in smoking behavior to the larger population of smokers. Second, prior research has utilized binary PMA indicators that categorize only supraclinical PMA levels, which leaves the variation across the entire continuum of PMA undefined. Yet, subclinical variation across the continuum of depressive symptoms among individuals who do not meet clinical criteria for current major depression is associated with more severe nicotine dependence and greater smoking relapse risk in community samples of smokers.¹¹ Thus, exploring variation across the continuum of PMA and its relation to smoking is warranted among individuals without a currently active suprathreshold mood disorder. Finally, little is also known about the profiles of smoking characteristics, behaviors, and motivations related

to PMA; rather, all prior studies of PMA have examined nicotine dependence diagnoses only.¹⁰ Examining various clinically-relevant smoking processes could elucidate mechanisms linking PMA to smoking and identify potential clinical targets for smoking cessation in smokers with elevated PMA.

With a community sample of non-treatment-seeking smokers who did not meet current criteria for major depression, we examined cross-sectional associations of PMA to various smoking characteristics relevant to the etiology and treatment of nicotine dependence. To this end, we investigated nicotine dependence severity, history of smoking, quitting history, smoking motives, and beliefs regarding the effects of smoking and quitting. To illustrate the incremental utility of the PMA phenotype for smoking, we also examined the extent to which PMA associated with smoking characteristics after controlling for overall depressive symptoms.

METHODS

Participants and Study Design

This report examines cross-sectional associations of baseline data from non-treatment-seeking regular smokers participating in a laboratory study on the relation of psychopathology and smoking.¹² Participants were recruited from the Los Angeles area through different forms of advertisements and referral to take part in a laboratory study of individual differences in subthreshold depressive psychopathologic traits and smoking. The current report solely focuses on baseline data at which PMA and smoking characteristics were assessed.

Inclusion criteria required participants to be 18 years of age or older, a regular smoker for at least the past 2 years (≥ 10 cigs/day), and fluent in English. Exclusion criteria included (1) current *DSM-IV* substance dependence (other than nicotine) and a negative breath alcohol to limit effects of drug and alcohol acute effects and withdrawal on mood; (2) current *DSM-IV* mood disorder or psychotic symptoms; (3) breath carbon monoxide (CO) levels <10 ppm at intake to prevent admission of individuals over-reporting their smoking in order to participate; (4) use of non-cigarette forms of tobacco or nicotine products; (5) current use of psychiatric or psychoactive medications; (6) currently pregnant; and (7) planning to quit or substantially cut down their smoking in the next 30 days.

Of the 505 smokers recruited, 155 were ineligible, 7 declined to participate, and 14 had missing data. Additionally, because the questionnaire measuring PMA was introduced midstream into recruitment, PMA data were available for only 254 participants. The University of Southern California Institutional Review Board approved the protocol.

Procedure

Following a preliminary telephone screening, eligible participants were instructed to smoke normally prior to attending the baseline session; however, they were not allowed to smoke once the session began. The 90–120 minutes baseline session consisted of an informed consent and eligibility assessments, including: breath alcohol and CO assessment as well as relevant modules of the Structured Clinical Interview for *DSM-IV*-Axis I Disorders,

Research Version, Non-Patient Edition to assess mood disorders, psychosis, and substance use disorder for eligibility purposes.¹³ Eligible participants continued with the remainder of the baseline session, including completing the measures described. Participants were compensated \$15 at the end of the visit.

Measures

PMA and Depressive Symptoms—Restlessness and agitation questionnaire (RAQ).

The RAQ includes 28 items, which each reflect different symptoms and behaviors indicative of PMA (eg, “feeling fidgety,” “having impulses to move around,” “feeling wound up”). For each item, participants indicate the usual frequency on a 5-point scale 0 (“never”) to 4 (“always”). A composite mean score is computed across the 28 items. Although the psychometric properties of the RAQ have not been previously published, the RAQ indicated strong internal consistency (Cronbach’s $\alpha = .97$) and a moderate correlation with the Center for Epidemiologic Studies Depression Scale total score ($r = .36, p < .0001$), suggesting that the RAQ taps a construct distinct from depression yet associated with overall depressive symptomatology. A principal component analysis of the factor structure of the RAQ illustrated that one major factor accounted for 54% of the variance across the 28 items (the next largest factor accounted for only 5% of the variance), suggesting that the RAQ is largely a one-dimensional factor structure.

Center for epidemiologic studies depression scale (CESD).¹⁴ The CESD (Cronbach’s $\alpha = .87$) is a well-validated 20-item self-report measure of current depressive symptom severity, which yields a total (sum) score. The CESD served as a covariate in this report to determine whether smoking relations involving RAQ were incremental to covariance with overall depressive symptoms. The CESD has displayed strong reliability and validity in prior work.¹⁴

Smoking-Related Measures—Smoking history questionnaire (SHQ).¹⁵ The SHQ included individual items regarding smoking history, including: assessing age at which one started smoking regularly, number of prior unsuccessful quit attempts, and usual number of cigarettes smoked per day. For those who reported a history of at least one serious and deliberate quit attempt ($n = 184$), the SHQ also included follow up items assessing severity of nicotine withdrawal symptoms experienced in prior quit attempts, for which a composite mean score across seven symptoms (ie, craving, irritability, nervousness, difficulty concentrating, physical symptoms, difficulty sleeping, loss of interest or pleasure; Cronbach’s $\alpha = .89$) rated on 5 point scale (1 = not at all to 5 = very severe) was computed. The SHQ has displayed good convergent validity in prior work.¹⁵

Fagerström test of nicotine dependence (FTND).¹⁶ The FTND is a widely used six-item self-report measure of nicotine dependence severity that examines heavy and compulsive smoking behavior (eg, “How soon after you wake up do you smoke your first cigarette?” or “How many cigarettes per day do you smoke?”). Scores range from 0 to 10 with higher scores indicating higher levels of nicotine dependence. Prior research has shown that FTND demonstrates good reliability and predictive/convergent validity to other measures of

nicotine dependence and dependence-relevant processes (ie, nicotine withdrawal, craving, biomarkers indicative of tobacco exposure, and abstinence following cessation).^{17–22}

Michigan nicotine reinforcement questionnaire (MNRQ).²³ The MNRQ assesses subjective motivation to smoke for reinforcement purposes by asking participants to rate self-statements on four-point scales (0–3). The MNRQ contains discrete positive reinforcement (five items; eg, “I crave a cigarette to provide pleasure”) and negative reinforcement (eight items; eg, “I crave a cigarette to provide relief from withdrawal”) subscales that are computed by taking the mean score across the items within each scale, and reflect smoking motivation for positive affect enhancement and negative affect relief, respectively. Analyses of the MNRQ support the factorial validity of distinct positive reinforcement and negative reinforcement dimensions and suggest adequate convergent validity and internal consistency of these scales.²³

Smoking abstinence questionnaire (SAQ).²⁴ The SAQ assesses the expected likelihood that certain consequences would occur upon smoking abstinence by instructing participants to respond to self-statement on a scale from 0 (“not likely at all”) to 6 (“extremely likely”). This study included only the following scales of the SAQ: withdrawal symptoms (seven items; eg, “I would feel short tempered or cranky”), optimistic outcomes (six items; eg, “It would be no problem to find an alternative to smoking that helps reduce stress”), and weight gain (three items; eg, “I would gain weight”). Participants rated the likelihood of these statements, and an average score per item was computed for each respective subscale. The SAQ has displayed good internal consistency and convergent validity in prior work.²⁴

Smoking consequences questionnaire (SCQ).^{25,26} The SCQ assesses smoking outcome expectancies by instructing participants to rate the relevance of self-statements reflecting anticipated effects of smoking on 7-point scales (1 “Not true of me at all” to 7 “Very true of me”). The SCQ yields 4 subscales indicative of different domains of expectancies by computing the mean score per item: negative consequences (18 items; eg, “The more I smoke, the more I risk my health”), negative reinforcement (12 items; eg, “Cigarettes help me concentrate,” “cigarettes help me deal with anxiety”), positive reinforcement (15 items; eg, “cigarettes taste good”), and weight control (5 items; “Smoking controls my appetite”). The SCQ has displayed high internal consistency, convergent validity, discriminant validity, and factorial validity in past studies.^{25,26}

To describe the level of substance use problems in the sample, we also administered two tests: (i) alcohol use disorders identification test (AUDIT), a well-validated 10-item screening survey designed to measure degree of alcohol problems, and (ii) drug abuse screening test, a well-validated 20-item screening survey designed to measure degree of drug problems.^{27,28}

Data Analysis

Preliminary analyses involved computing descriptive statistics and intercorrelation of study variables. Primary analyses involved computing separate individual linear regression models in which the RAQ score served as the predictor and a single smoking variable served as the outcome. For each outcome variable, we ran two analyses: (i) an unadjusted model in which

RAQ score was the sole predictor, and (ii) an adjusted model that included RAQ as a predictor but also adjusting for age, gender, ethnicity, race, and CESD score. Results are reported as standardized regression weights (β) and statistical significance was set to .05 (two-tailed). Because this is the first study to explore relations between PMA and smoking characteristics, we did not correct for type-I error for multiple hypothesis testing to avoid overlooking any potential findings that could be followed up in future work.

RESULTS

Preliminary Analyses

The descriptive statistics of the sample's demographic and smoking characteristics can be found in Tables 1 and 2, respectively. Table 1 indicates the sample was on average, middle aged, predominately male, racially diverse, reported low levels of drug and alcohol use problems, had medium levels of nicotine dependence, made several prior quit attempts and had been smoking regularly since late adolescence. On average, the sample reported low levels of depressive symptoms with a fair degree of variance in CESD scores, with 22.1% surpassing the screening cutoff for possible mild-to-moderate depression on the CESD (16).¹⁴ The mean RAQ score was .91, suggesting the average participant experienced PMA relatively infrequently. However, there was meaningful variability in RAQ scores across the PMA continuum, as 25% of the sample scored .36 or below (lower quartile) and 25% scored 1.32 or higher (upper quartile); the range was 0–3.86. RAQ scores were not associated with gender or ethnicity; however, younger participants, those not of the African American race, and individuals with higher alcohol problems (AUDIT) reported significantly higher scores on the RAQ.

Primary Analyses

As illustrated in Table 2, unadjusted analyses illustrated that higher RAQ scores were significantly associated with the following measures: greater withdrawal symptom severity during prior quit attempts on the SHQ, greater expected likelihood of withdrawal symptoms during smoking abstinence on the SAQ, higher negative reinforcement smoking motivation on the MNRQ, and higher expectancies for smoking-induced effects related to negative reinforcement, negative consequences, and positive reinforcement on the SCQ. After adjusting for depressive symptoms, age, gender, and race, each of the significant relations between RAQ and smoking characteristics were reduced in magnitude but remained statistically significant. RAQ was not significantly associated with nicotine dependence, cig/day, age of onset, number of quit attempts, and several non-affect-related smoking motives.

DISCUSSION AND CONCLUSION

This report documents relations between PMA and a unique profile of smoking characteristics among community cigarette smokers without current major depression. Higher PMA was significantly associated with processes relevant to smoking-related affect modulation and nicotine withdrawal. Specifically, smokers with elevated PMA reported greater salience of: (i) tobacco withdrawal symptoms, both during prior quit attempts and in anticipation of future quit attempts; (ii) motivations and beliefs regarding smoking for

negative reinforcement purposes; and (iii) anticipating positive reinforcement effects of smoking. As these relationships remained after adjusting for depressive symptoms, our results indicate that PMA may explain variance in smoking-related processes that are not accounted for by depressive symptoms. Furthermore, our findings extend previous literature on PMA and smoking in general psychiatric patient samples—which focuses primarily on nicotine dependence status—by providing a comprehensive examination of PMA’s relation with individual differences across a wide range of smoking-related processes within daily smokers.^{9,10}

Despite exhibiting associations with the abovementioned factors that putatively increase risk of smoking behavior, PMA was also associated with the tendency to anticipate greater negative consequences of smoking (eg, health and social problems), which putatively protects against smoking. This pattern of associations with both positive (pro-smoking) and negative (anti-smoking) expectancies of smoking has been previously demonstrated with other constructs (eg, negative affectivity, distress tolerance) and raises the possibility that heightened motivation towards smoke affect-regulation may override any fear of smoking-related negative consequences in smokers with elevated PMA.²⁹

We further found that those with elevated-PMA may not necessarily differ from those without elevated PMA in regards to smoking level, chronicity, and dependence severity. However, because the study inclusion criteria required all participants to smoke at least 10 cig/day, it is possible that this range restriction may have reduced power to detect any relations. Indeed, past research documenting that PMA is associated with nicotine dependence status has contrasted nicotine-dependent smokers to the heterogeneous group of non-smokers and lighter non-dependent smokers.^{10,30} Thus, further research is warranted to explore whether PMA differentiates smoking level among lighter smokers and smoking vs. non-smoking status.

Additionally, although our results indicate that elevated PMA was significantly associated with more severe withdrawal symptoms during prior quit attempts, it was not associated with frequency of prior quit attempts. This finding has clinical implications: (i) smokers with elevated PMA may be equally as likely to make quit attempts as low-PMA smokers despite high-PMA smokers having more severe withdrawal symptoms; and (ii) phenotypic expressions of PMA could reflect a proneness to withdrawal symptoms during smoking abstinence.

Certain elements of PMA overlap with certain symptoms of nicotine withdrawal (eg, impatience, restlessness). Hence, current nicotine withdrawal due to temporary tobacco deprivation experienced prior to completing the questionnaires might have altered responses on the RAQ, such that deprivation-provoked manifestations of agitation (outside of trait PMA) were picked up on the RAQ. While this is an important limitation that we cannot rule out, the RAQ assesses PMA features at the trait level (ie, “how often do you usually have that experience?”). Furthermore, several RAQ items rely on others’ observations of the respondent’s behavior (ie, “Do other people tell you that you look restless?”). These items are less likely to be susceptible to subjective reporting biases that may inflate perceptions of PMA. Finally, in adjusted analyses we controlled for a measure of depression, which

contains a number of symptoms that overlap with the nicotine withdrawal syndrome. Hence, any reporting bias whereby current withdrawal biases reports on prior trait experience is likely to also co-vary with self-report depression; thus, by co-varying for depression, it is likely that variance in reporting biases could be partialled out in adjusted analyses.

We speculate that PMA is a unique construct within larger network maladaptive traits implicated in both psychopathology and tobacco use. PMA shares commonalities with impulsivity and hyperactivity in that each of these constructs involves the expression of maladaptive automatic behaviors. However, whereas PMA behavioral activity reflects a manifestation of mental tension and distress, impulsivity and hyperactivity constructs reflect poor control of appetite, stimulation seeking, or other pre-potent behaviors that may not necessarily be caused by emotional distress.^{31,32} PMA also conceptually overlaps with negative affect traits, such as neuroticism and hostility, in that each may share tendencies towards tense and irritable affect. However, PMA's defining feature is the automatic manifestation of purposeless behaviors as a result of distress, whereas neuroticism and related traits do not necessarily involve the expression of purposeless behaviors.³³

Based on the situation $\times$ trait adaptive response model of smoking that hypothesizes that motivation to smoke is determined by the interaction of trait dispositions and state-specific circumstances, we suspect that trait PMA may amplify one's sensitivity to the acute effects of tobacco administration and abstinence in a trait-consistent manner.³⁴ As has been shown with other psychopathological traits implicated in smoking (ie, neuroticism, hostility, and impulsivity),³⁴⁻³⁷ we suspect that smokers with high (vs. low) PMA may experience more mood enhancement and dysregulation upon tobacco administration and withdrawal, respectively. The current study's cross-sectional relations with mood-relevant smoking expectancies, motivation, and retrospective nicotine withdrawal are consistent with this notion, though future research is required to test these specific hypotheses.

There were certain limitations to this study. First, because this report conducts cross-sectional analysis of baseline data, temporal ambiguity does not allow us to infer the directionality of the relationship documented here. Second, our measurement of PMA included only one recently developed questionnaire that was developed for the purpose of examining the experience of PMA across the entire continuum of functioning. Although the RAQ exhibited strong internal consistency and a largely one-dimensional factor structure in our sample, it would have been preferable to include multiple measures of PMA that span self-report and objective measures.³⁸ Third, our data analysis plan did not correct for type-I error associated with multiple testing in order to avoid overlooking potential key relationships for future research. Thus, it is possible that a chance finding might have been detected; however, the consistent pattern across multiple measures of similar constructs (eg, withdrawal, negative reinforcement) helps to offset concern for type-I error to some extent. Fourth, because of the study entry criteria, there are certain restrictions to the generalizability of these data, including whether similar relations would be present among smokers with an active mood disorder or psychosis, with drug/alcohol dependence, and those interested in making an immediate quit attempt.

In conclusion, these findings suggest that PMA may be worthy of consideration in tobacco addiction etiology research and smoking cessation programs, regardless of depressive symptom or syndrome status. High-PMA smokers may perhaps benefit from increased clinical attention to tobacco withdrawal symptoms and interventions that promote coping skills to prevent smoking in response to affective disturbance. Furthermore, psychiatric assessment protocols to identify subgroups of smokers with unique clinical needs may benefit from incorporation of measures of PMA, as PMA may help to explain variation in smoking-related processes that is not addressed in standard depressive symptom composite approach.

Acknowledgments

This research was supported by National Institute on Drug Abuse Grant (Bethesda, Maryland) R01-DA026831 awarded to Dr. Adam Leventhal and the University of Southern California.

The authors acknowledge staff members at the University of Southern California's Health, Emotion, and Addiction Laboratory, who were instrumental in collecting the data presented here.

The authors alone are responsible for the content and writing of this paper.

REFERENCES

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-5. 5th ed.. Washington, DC: American Psychiatric Association; 2013. American Psychiatric Association. DSM-5 Task Force.
2. Sobin C, Sackeim HA. Psychomotor symptoms of depression. *Am J Psychiatry*. 1997; 154:4–17. [PubMed: 8988952]
3. Hasler G, Drevets WC, Manji HK, et al. Discovering endophenotypes for major depression. *Neuropsychopharmacology*. 2004; 29:1765–1781. [PubMed: 15213704]
4. Leventhal AM, Pettit JW, Lewinsohn PM. Characterizing major depression phenotypes by presence and type of psychomotor disturbance in adolescents and young adults. *Depress Anxiety*. 2008; 25:575–592. [PubMed: 17385727]
5. Leventhal AM, Gelernter J, Oslin D, et al. Agitated depression in substance dependence. *Drug Alcohol Depend*. 2011; 116:163–169. [PubMed: 21277711]
6. Zimmerman M, McGlinchey JB, Young D, et al. Diagnosing major depressive disorder I: a psychometric evaluation of the DSM-IV symptom criteria. *J Nerv Ment Dis*. 2006; 194:158–163. [PubMed: 16534432]
7. Marmorstein NR. Associations between subtypes of major depressive episodes and substance use disorders. *Psychiatry Res*. 2011; 186:248–253. [PubMed: 21035193]
8. Leventhal AM, Zimmerman M. The relative roles of bipolar disorder and psychomotor agitation in substance dependence. *Psychol Addict Behav*. 2010; 24:360–365. [PubMed: 20565163]
9. Leventhal AM, Kahler CW, Ray LA, et al. Refining the depression-nicotine dependence link: patterns of depressive symptoms in psychiatric outpatients with current, past, and no history of nicotine dependence. *Addict Behav*. 2009; 34:297–303. [PubMed: 19062199]
10. Leventhal AM, Ramsey SE, Brown RA, et al. Dimensions of depressive symptoms and smoking cessation. *Nicotine Tob Res*. 2008; 10:507–517. [PubMed: 18324570]
11. Trujillo M, Ameringer K, Tidey JW, et al. Anhedonia and the relative reward value of drug and non-drug reinforcers in cigarette smokers. *J Abnorm Psychol*. 2014; 123:375–386. [PubMed: 24886011]
12. First, MB.; Spitzer, RL.; Gibbon, M., et al. Biometrics Research NYPI. ed.. New York: 2002. Structured clinical interview for DSM-IV-TR Axis I disorders, research version, non-patient edition (SCIDI/NP).

14. Radloff LS. The CES-D Scale A self-report depression scale for research in the general population. *Appl Psychol Meas.* 1977; 1:385–401.
15. Brown RA, Lejuez CW, Kahler CW, et al. Distress tolerance and duration of past smoking cessation attempts. *J Abnorm Psychol.* 2002; 111:180–185. [PubMed: 11866171]
16. Heatherton TF, Kozlowski LT, Frecker RC, et al. The Fagerstrom test for nicotine dependence: a revision of the Fagerstrom tolerance questionnaire. *Br J Addict.* 1991; 86:1119–1127. [PubMed: 1932883]
17. Piper ME, McCarthy DE, Bolt DM, et al. Assessing dimensions of nicotine dependence: an evaluation of the Nicotine Dependence Syndrome Scale (NDSS) and the Wisconsin Inventory of Smoking Dependence Motives (WISDM). *Nicotine Tob Res.* 2008; 10:1009–1020. [PubMed: 18584464]
18. Rios-Bedoya CF, Snedecor SM, Pomerleau CS, et al. Association of withdrawal features with nicotine dependence as measured by the Fagerstrom test for nicotine dependence (FTND). *Addict Behav.* 2008; 33:1086–1089. [PubMed: 18502052]
19. Etter JF. A comparison of the content-, construct- and predictive validity of the cigarette dependence scale and the Fagerstrom test for nicotine dependence. *Drug Alcohol Depend.* 2005; 77:259–268. [PubMed: 15734226]
20. Payne TJ, Smith PO, McCracken LM, et al. Assessing nicotine dependence: a comparison of the Fagerstrom tolerance questionnaire (FTQ) with the Fagerstrom test for nicotine dependence (FTND) in a clinical sample. *Addict Behav.* 1994; 19:307–317. [PubMed: 7942248]
21. Sweitzer MM, Denlinger RL, Donny EC. Dependence and withdrawal-induced craving predict abstinence in an incentive-based model of smoking relapse. *Nicotine Tob Res.* 2013; 15:36–43. [PubMed: 22513801]
22. Courvoisier DS, Etter JF. Comparing the predictive validity of five cigarette dependence questionnaires. *Drug Alcohol Depend.* 2010; 107:128–133. [PubMed: 19926407]
23. Pomerleau OF, Fagerstrom KO, Marks JL, et al. Development and validation of a self-rating scale for positive- and negative-reinforcement smoking: the Michigan nicotine reinforcement questionnaire. *Nicotine Tob Res.* 2003; 5:711–718. [PubMed: 14577987]
24. Hendricks PS, Wood SB, Baker MR, et al. The smoking abstinence questionnaire: measurement of smokers' abstinence-related expectancies. *Addiction.* 2011; 106:716–728. [PubMed: 21205053]
25. Wetter DW, Smith SS, Kenford SL, et al. Smoking outcome expectancies: factor structure, predictive validity, and discriminant validity. *J Abnorm Psychol.* 1994; 103:801–811. [PubMed: 7822583]
26. Brandon TH, Baker TB. The smoking consequences questionnaire: the subjective expected utility of smoking in college students. *Psychol Assess.* 1991; 3:481–491.
27. Saunders JB, Aasland OG, Babor TF, et al. Development of the alcohol use disorders identification test (AUDIT): WHO Collaborative Project on Early Detection of Persons with Harmful Alcohol Consumption-II. *Addiction.* 1993; 88:791–804. [PubMed: 8329970]
28. Gavin DR, Ross HE, Skinner HA. Diagnostic validity of the drug abuse screening test in the assessment of DSM-III drug disorders. *Br J Addict.* 1989; 84:301–307. [PubMed: 2650770]
29. Leyro TM, Bernstein A, Vujanovic AA, et al. Distress tolerance scale: a confirmatory factor analysis among daily cigarette smokers. *J Psychopathol Behav Assess.* 2011; 33:47–57. [PubMed: 23935238]
30. Kassel JD, Yates M, Brown RA. Baseline reaction time predicts 12-month smoking cessation outcome in formerly depressed smokers. *Psychol Addict Behav.* 2007; 21:415–419. [PubMed: 17874893]
31. Antrop I, Roeyers H, Van Oost P, et al. Stimulation seeking and hyperactivity in children with ADHD. Attention deficit hyperactivity disorder. *J Child Psychol Psychiatry.* 2000; 41:225–231. [PubMed: 10750548]
32. Moeller FG, Barratt ES, Dougherty DM, et al. Psychiatric aspects of impulsivity. *Am J Psychiatry.* 2001; 158:1783–1793. [PubMed: 11691682]
33. Costa PT Jr, McCrae RR. Domains and facets: Hierarchical personality assessment using the revised NEO personality inventory. *J Pers Assess.* 1995; 64:21–50. [PubMed: 16367732]

34. Gilbert DG, Gilbert BO. Personality, psychopathology, and nicotine response as mediators of the genetics of smoking. *Behav Genet.* 1995; 25:133–147. [PubMed: 7733855]
35. Quinn A, Sekimura S, Pang R, et al. Hostility as a predictor of affective changes during acute tobacco withdrawal. *Nicotine Tob Res.* 2014; 16:335–342. [PubMed: 24113928]
36. Kahler CW, Leventhal AM, Colby SM, et al. Hostility, cigarette smoking, and responses to a lab-based social stressor. *Exp Clin Psychopharmacol.* 2009; 17:413–424. [PubMed: 19968406]
37. Harrison EL, Coppola S, McKee SA. Nicotine deprivation and trait impulsivity affect smokers' performance on cognitive tasks of inhibition and attention. *Exp Clin Psychopharmacol.* 2009; 17:91–98. [PubMed: 19331485]
38. Parker G, Hadzi-Pavlovic D, Wilhelm K, et al. Defining the personality disorders: description of an Australian database. *Aust N Z J Psychiatry.* 1996; 30:824–833. [PubMed: 9034473]

TABLE 1

Descriptive statistics and intercorrelations among sociodemographic characteristics, psychomotor agitation, and depressive symptoms ($N = 254$)

Sample characteristics		Correlations (<i>rs</i>)	
		RAQ	CESD
Age, <i>M</i> (SD)	43.7 (10.8)	-.14*	.04
Male (%)	67.6%	-.04	-.001
Black (%)	51.2%	-.17**	.09
DAST, <i>M</i> (SD)	3.46 (4.83)	.06	.18*
AUDIT, <i>M</i> (SD)	2.03 (3.65)	.19**	.13
Not Hispanic/Latino (%)	85.1%	-.06	-.05
RAQ, <i>M</i> (SD)	.91 (.70)	–	–
CESD, <i>M</i> (SD)	10.9 (8.7)	–	–

Note: RAQ = restlessness and agitation questionnaire mean of 28 questions involving agitation and restlessness (possible range: 0 “never”–4 “always”), CESD = center for epidemiologic studies depression scale; DAST = drug abuse screening test (possible range: 0–20); AUDIT = alcohol use disorder identification test (possible range: 0–40).

Male coded as 1, female coded as 0. Black coded as 1, non-black coded as 0. Not Hispanic/Latino coded as 1, Hispanic coded as 0. Spearman's correlation coefficient reported.

*
 $p < .05$;

**
 $p < .01$.

TABLE 2

Associations between psychomotor agitation and smoking characteristics ($N = 254$)

	<i>M</i>	<i>SD</i>	RAQ	
			β -Unadj ^A	β -Adj ^B
FTND	5.29	1.87	-.07	-.07
SHQ				
Age started smoking regularly	19.49	5.92	.01	.05
Number of cigarettes/day	16.63	6.63	-.09	-.11
Severity of past withdrawal symptoms during prior quit attempts	2.52	.91	.23***	.18*
Total # of quit attempts	3.64	7.57	.07	.08
MNRQ				
Positive reinforcement	1.58	.63	.03	.08
Negative reinforcement	1.21	.58	.20**	.14*
SAQ				
Withdrawal symptoms	3.77	1.40	.26***	.19*
Optimistic outcomes	2.87	1.33	-.04	-.03
Weight gain	3.21	1.84	.07	.03
SCQ				
Negative consequence	4.87	1.18	.19**	.22**
Positive reinforcement	4.55	1.20	.15*	.14*
Negative reinforcement	4.33	1.73	.27***	.17*
Appetite-weight control	3.00	2.05	.11	.06

Note:^A Standardized coefficient reported from simple linear regression models in which RAQ is the sole predictor of a smoking characteristic.^B Standardized coefficient from multivariate linear regression models reported in which RAQ is the predictor of smoking characteristics after adjusting for age, gender, ethnicity, race, and CESD (center for epidemiologic studies depression scale).

RAQ = restlessness and agitation questionnaire (scale: 0–4), FTND = Fagerstrom test for nicotine dependence (scores: 0–10), SHQ = smoking history questionnaire (scale: 1–5), MNRQ = Michigan nicotine reinforcement questionnaire (scale: 0–3), SAQ = smoking abstinence questionnaire (scale: 0–6), SCQ = smoking consequences questionnaire (scale: 1–7).

* $p < .05$;** $p < .01$;*** $p < .001$.