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Cigarette Smoking and Impulsivity in Bipolar Disorder

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Abstract

Objectives—There is a high prevalence of smoking among individuals with bipolar disorder (BD), yet there have been few efforts to identify potential contributing factors as a means of improving prevention and treatment approaches. The goal of this study was to examine the association between impulsivity and the initiation or maintenance of smoking in BD.

Methods—Participants were 97 adolescents and adults, ages 16–50, with bipolar I disorder who were experiencing a mixed or manic episode at the time of study enrollment. Participants completed the Barratt Impulsiveness Scale-11 (BIS-11) as a self-report indicator of trait impulsivity and the Logan Stop-Signal Task (SST), Delayed Reward Task (DRT), and Degraded Stimulus Continuous Performance Task (DSCPT) as behavioral measures of impulsivity.

Results—Current smokers (34%) and former smokers (23%) generally reported higher trait impulsivity on the BIS-11 than never smokers (43%), with minimal evidence for differences among the two ever-smoking groups. No differences in impulsivity by smoking status emerged on the behavioral measures.

Conclusions—Trait impulsivity is associated with the initiation, but not necessarily the maintenance, of cigarette smoking in adolescents and adults with bipolar disorder. Our findings provide no evidence that smoking is associated with impulsive responding on cognitive tasks during a symptomatic period during which impulsivity is elevated.

Keywords

nicotine; tobacco; smoke; mania; depression

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Introduction

The prevalence of cigarette smoking among individuals with bipolar disorder (BD) is two to three times higher than the rate in the general population (1,2), considerably increasing the risks of morbidity and mortality (3–5). Although it is likely that the etiology of co-occurring BD and cigarette smoking involves a complex mix of genetic, biological, and psychosocial factors (see review by Heffner et al. [61]), the mechanisms underlying this co-occurrence have not received much empirical attention. An understanding of these mechanisms can guide development of more effective approaches to prevention and treatment.

Impulsivity is one potential contributor to the relationship between smoking and BD. Preclinical evidence indicates that impulsivity predicts faster acquisition of nicotine self-administration, lessens ability to inhibit nicotine self-administration during extinction trials, and facilitates resumption of self-administration in a “relapse” paradigm (7). In human studies, impulsivity has also been found to predict onset of smoking (8), greater craving to smoke during nicotine abstinence (9), and relapse to regular tobacco use after a cessation attempt (10). In sum, impulsivity contributes to all phases of tobacco use, from initiation of regular smoking to relapse.

State-dependent elevations in impulsivity occur during manic episodes, but perhaps more importantly, our previous work demonstrated that trait impulsivity is higher in BD than in the general population, even during euthymic periods (11). This suggests that impulsivity may be one reason why individuals with BD are more vulnerable to initiating and maintaining smoking (1). However, we did not previously determine whether trait impulsivity was elevated among smokers with BD, an extension of this hypothesis. We are aware of only one prior study that examined this relationship, which found that current smokers did not differ from current nonsmokers on Barratt Impulsiveness Scale—11 scores (12). However, this study considered only current smoking status, which does not allow for a determination of how trait impulsivity may be differently related to distinct phases of smoking acquisition (i.e., initiation of smoking vs. maintenance of smoking). Thus, one aim of the present study was to compare levels of trait impulsivity in current, ex-, and never-smokers with BD.

Prior studies in individuals without psychiatric disorders suggest that smokers also tend to be more impulsive than nonsmokers on behavioral indicators of impulsivity, with the majority of this research focusing on response inhibition and ability to delay reward (13). The relationship between cigarette smoking and performance on behavioral measures of impulsivity has not been examined in individuals with BD, however. Additionally, because this relationship may be most observable during “hot cognitive states,” such as affective episodes (14), examining smoking-related differences in impulsive responding during a symptomatic period, rather than a euthymic period, may provide greater insight into the relationship between smoking and impulsivity in BD.

In order to begin elucidating some of the mechanisms underlying the high prevalence of smoking in BD, we examined relationships between smoking status and both subjective and objective indicators of impulsivity among individuals with BD, all of whom were experiencing a manic or mixed episode at the time of assessment. Based on prior work, we hypothesized that current smokers would have the highest levels of impulsivity, followed by former smokers and never smokers.

Materials and Methods

Participants

Participants were 97 adolescents and adults with a DSM-IV diagnosis of bipolar I disorder, ranging in age from 16–50 years, who were receiving either inpatient or outpatient treatment for a manic or mixed episode at the time of study enrollment. Participants included in these analyses are a subsample of participants (i.e., those who provided information about cigarette smoking status) as part of a larger study of impulsivity in bipolar disorder, which was reviewed and approved by the University of Cincinnati Institutional Review Board. Informed consent was obtained from all participants. The methods for this study have been described previously (11,15) and will only be repeated briefly here for clarity. Study eligibility required a Young Mania Rating Scale (YMRS; 16) score of ≥ 20 ; absence of a medical or neurological condition that could affect neurocognitive test results; an IQ score > 85 , as assessed using the American Modification of the Adult National Reading Test (ANART; 17); and no evidence of substance intoxication or withdrawal. Co-occurring substance use disorders and other psychiatric disorders were not exclusionary. Table 1 shows the demographic and clinical characteristics of the sample.

Procedures

The Structured Clinical Interview for DSM-IV Axis I disorders—Patient version (SCID-I/P; 18) was administered to establish DSM-IV diagnosis of bipolar I disorder as well as lifetime ADHD and substance use disorders (with the exception of nicotine dependence). Manic symptoms were assessed using the YMRS, as noted previously. The Addiction Severity Index (ASI; 19) was administered to assess the extent and consequences of substance use, with the addition of a set of items addressing cigarette smoking. Specifically, participants were asked to report smoking during the prior 30 days, age-at-onset of regular smoking, and years of regular smoking, with regular smoking being defined as smoking at least 3 times per week for one month or more. Individuals who smoked within the prior 30 days were classified as current smokers. Those who reported prior regular smoking, but no smoking within the past 30 days, were classified as former smokers. The rest of the participants were categorized as never smokers.

Participants completed the 30-item Barratt Impulsiveness Scale-11 (BIS-11; 20) as a self-report measure of trait impulsivity. The BIS-11 has three factors—1) attentional impulsiveness, or a propensity to shift attention quickly, resulting in impulsive decision-making; 2) motor impulsiveness, or a tendency to act without thinking; and 3) non-planning impulsiveness, or a tendency to make decisions based upon present conditions with less consideration for more distal consequences (21). We used both the subscale scores and the total BIS-11 score in comparisons of trait impulsivity among current, former, and never smokers.

The Logan Stop-Signal Task (SST; 22) was conducted as a measure of inhibitory control, or the ability to stop an automatic response in order to formulate a better one. Briefly, the test involved presentation of an X or O stimulus in 1-second intervals, and participants were required to press a button corresponding with the presented letter. In 25% of the trials, an auditory “stop” signal was presented to prompt participants not to respond during that trial. The stop signal latency started at 250 msec and was either increased or decreased by 50 msec during the next “stop” trial depending on whether the participant responded correctly on the previous trial. Participants were given 32 practice trials to learn the procedures, after which they completed a total of 256 trials (in 4 blocks of 64 trials). Consistent with our prior work, we used the stop signal reaction time (SSRT) as our primary outcome. SSRT was

defined as the average response time for the “go” trials minus the average stop signal latency for the “stop” trials.

The Delayed Reward Task (23) was administered to assess inability to delay gratification, another behavioral indicator of impulsivity. This task involved presentation of two stimuli—the letters A and B—and required participants to choose one of the two letters presented simultaneously on a computer screen in order to receive a monetary reward. Choice of the letter A, the impulsive response, caused the letter B to disappear and the letter A to begin flashing following a 5-second delay. While the letter A was flashing, the participant could click the right mouse button to receive a 5-cent reward. Choice of the letter B, on the other hand, resulted in the letter beginning to flash following a 15-second delay, with length of delay increasing by 2 seconds following every trial in which B was selected and decreasing by 2 seconds for every trial that A was selected (up to a maximum of 7 sec decrease). When the letter B was flashing, the participant could click on it to earn a 15-cent reward. Since there were a fixed number of trials, choosing the letter B always resulted in greater total reward value. Participants completed 10 training trials, followed by 25 scored trials. Both response time and proportion of impulsive responses (i.e., choice of the letter A) were calculated as measures of impulsivity on the DRT.

The Degraded Stimulus Version of the Continuous Performance Test (DSCPT; based on a task developed by Neuchterlein [24]) was administered as a measure of attentional impulsivity. This task involved presentation of numeric stimuli—a random series of numbers ranging from 0 to 9—and required participants to press a button when they observed the target stimulus, which was the number 0. To make the task more difficult, the numbers were degraded (i.e., blurred). Numbers appeared for 35 msec and were presented at a rate of 1 per second. Participants were given 80 practice trials, after which they completed 480 trials (in 6 blocks of 80 trials). The target stimulus was presented in 25% of the trials. We calculated three outcome measures for this task—sensitivity, response bias, and reaction time. Sensitivity reflects the ability to separate signal from noise and was calculated as follows: $\text{Sensitivity} = 0.5 + (y - x)(1 + y - x)/4y(1 - x)$, where y is the probability of a false positive and x is the probability of a true positive. Response bias reflects factors other than sensitivity that may affect performance, such as fatigue, and was calculated as follows: $\text{Response bias} = y(1 - y) - x(1 - x)/y(1 - y) + x(1 - x)$, where smaller values equal greater bias. Response time was calculated as the mean time to press the button in response to the target stimulus.

Data Analysis

Comparisons among current, former, and never smokers were made using ANOVAs or ANCOVAs with Tukey HSD post hoc tests. Partial eta-squared values were calculated as a measure of effect size, and results were interpreted using Cohen's (25) guidelines for determining small (.01), medium (.06), and large (.14) effects. Variables chosen as potential covariates were demographic and clinical variables that differed by smoking status and were related to scores on the BIS-11, the SST, the DRT, or the DSCPT. Because there were a considerable number of potential covariates identified for the analyses involving the BIS-11, for the purpose of parsimony, we used regression-based selection methods to identify the set of covariates to be included in the final models. Specifically, we sequentially tested backward, forward, and stepwise methods of entry to evaluate predictors and selected those that demonstrated a consistent association with impulsivity. An alpha level of $p < .05$ was used for all comparisons. Analyses were conducted using PASW Statistics 17.0 (SPSS, Inc., Chicago, IL).

Results

Comparison of the demographic and clinical characteristics of current smokers (CS; $n=33$; 34%), former smokers (FS; $n=22$; 23%), and never smokers (NS; $n=42$; 43%) are shown in Table 1. As shown in the table, the CS and FS groups included a greater number of White patients than the NS group ($p=.02$). Additionally, the NS group was significantly less likely to present in a mixed (vs. manic) episode ($p=.03$). As expected, smoking status was associated with other substance use disorders as well as other psychiatric disorders. Both the CS and the FS groups were more likely than the NS group to have lifetime diagnoses of non-nicotine substance use disorders ($p=.001$) as well as attention-deficit/hyperactivity disorder (ADHD) ($p=.01$). These were the only co-occurring disorders that occurred in a high enough frequency in the sample to be considered as covariates in subsequent analyses. In terms of differences in the smoking histories of the CS and FS groups, there was no difference in age at onset of regular smoking between CS ($M=16$, $SD=7$) and FS ($M=18$, $SD=7$; $p=.412$), but CS reported smoking for more years ($M=11$, $SD=9$) than FS ($M=6$, $SD=4$; $p=.021$).

None of the demographic and clinical variables were associated with performance on the SST or the DSCPT. However, there were sex differences on the DRT response time ($p<.05$). BIS-11 total and subscale scores were related to years of education, baseline YMRS scores, polarity (manic vs. mixed episode), and lifetime diagnoses of ADHD and non-nicotine substance use disorders (all p -values $< .05$). Because race, polarity, and diagnoses of ADHD and non-nicotine substance use disorders were also associated with smoking status, these variables were considered for inclusion in the ANCOVAs examining differences in trait impulsivity among the three smoking status groups. On the basis of additional preliminary tests, however, only ADHD diagnosis and polarity were selected as covariates due to their consistent association with BIS-11 total and subscale scores using backward, forward, and stepwise selection procedures.

In univariate comparisons, BIS-11 scores were uniformly higher among both CS and FS compared with NS (see Table 2). The only exception was in the case of Motor Impulsiveness, in which post hoc tests indicated that CS ($p=.001$), but not FS ($p=.40$), scored significantly higher than NS. In the ANCOVA analyses, after controlling for ADHD diagnosis and polarity, the BIS-11 Total ($p=.004$), and Nonplanning ($p=.015$) subscales were elevated in CS and FS compared to NS. Similar to the results of the univariate comparisons, Motor Impulsiveness differed by smoking status ($p=.004$), with post hoc tests suggesting that only the CS group had elevated Motor Impulsiveness compared to NS ($p=.001$). However, as shown in Table 2, mean scores on the Motor Impulsiveness scale were higher in the FS compared to the NS group, even though this difference was not significant in the post hoc test. Partial eta squared estimates suggested medium effect sizes for all of the BIS-11 scales, ranging from .06 to .12.

As an exploratory follow-up, we repeated the ANCOVA analyses without the participants who had co-occurring ADHD to determine whether this would decrease the strength of the relationship between trait impulsivity and smoking status. Contrary to expectation, effect size estimates increased in most of these analyses, with partial eta squared estimates of .041 for Attentional Impulsiveness (decreased from .055 in the full-sample ANCOVA), .135 for Motor Impulsiveness (increased from .123), .128 for Non-planning Impulsiveness (increased from .097), and .146 for Total Impulsiveness (increased from .124) on the BIS-11. It is important to note that we did not test the significance of the differences in effect sizes due to the reduced sample size and insufficient power for such tests; however, these preliminary results point toward a pattern of similar, or perhaps even increased effect sizes, as opposed to reduced effect sizes (with the exception of the Attentional Impulsiveness scale, which did decrease slightly).

There were no significant differences between the CS, FS, and NS groups on SST, DRT, and DSCPT performance (see Table 2), and effect size estimates were in the small range across all of these measures, with partial eta squared values ranging from $< .01$ to $.04$. Given this discrepancy between the findings from self-report versus behavioral measures of impulsivity, we examined correlations between the BIS-11 scores (including total and subscale scores) and the SST, DRT, and DSCPT scores. Only one of these 24 correlations (i.e., BIS-11 Nonplanning scale and DRT Reaction Time, $r = -.22$, $p = .04$) was significantly different from zero at the $p < .05$ level (see Table 3), highlighting the divergent results generated by these two methods of assessing impulsivity.

Discussion

Trait impulsivity may play a role in the high prevalence of smoking in BD, with the results of our multivariate analyses suggesting that the relationship is moderate in strength and primarily related to the initiation rather than the maintenance of smoking. That is, the current and former smokers both reported higher total trait impulsivity on the BIS-11 than never smokers, and the differences between the two ever-smoking groups were not significant. Thus, these findings provide only partial support for our hypotheses that impulsivity would be most elevated in current smokers due to its relationship with both the initiation and maintenance of smoking. Interestingly, our exploratory analyses suggested that the strength of the relationship between smoking status and trait impulsivity was not notably reduced, as we had expected, when we excluded the subgroup of participants with co-occurring ADHD, a common comorbidity of bipolar disorder that is also associated with greater risk of initiating and maintaining smoking (26). Thus, these findings suggest that the relationship between trait impulsivity and smoking in bipolar disorder is not primarily driven by the high prevalence of ADHD among people with the disorder.

Our failure to detect a relationship between smoking and behavioral measures of impulsivity is in contrast to the findings from previous studies in non-psychiatric populations indicating that smokers demonstrate lower ability to delay reward, and perhaps to inhibit responses, although evidence for the latter is less robust (13). There may be a number of reasons why we did not detect a relationship between smoking and impulsivity on the behavioral measures used in this study. Nicotine, the primary psychoactive ingredient in tobacco smoke, can improve attention and working memory (27–29), both of which could improve current smokers' performance on these measures, thereby confounding the assessment of smoking-related differences in impulsivity. Importantly, the cognitive enhancing effects of nicotine in individuals with psychiatric disorders may be more pronounced than in those without the disorders (30), perhaps explaining the discrepancy between the findings of this study and those of prior studies in non-psychiatric populations that demonstrated smoking-related differences in behavioral task performance. Another possible reason for the discrepant findings is that, during a manic or mixed episode, cognitive state markers of impulsivity may be uniformly elevated such that it becomes difficult to detect differences based on smoking status. However, our data suggest that this is not likely, as there was a significant amount of individual variability in the scores on the SSRT, DRT, and DSCPT. Finally, given the modest size for the three smoking status groups, our sample may not have been large enough to detect small differences in task performance on these measures.

This study has several other limitations that should be noted when interpreting the results. First, we relied on limited assessments of past and current smoking collected as part of a larger study (11), the primary focus of which was not on cigarette smoking. As such, we were unable to determine how recently former smokers had quit (i.e., tobacco abstinence could have ranged from one month to several decades) or to biochemically verify smoking status. At the same time, our definitions of smoking status are generally consistent with

epidemiologic definitions that consider; 1) whether individuals, during their lifetime, surpassed a threshold of use (i.e., met a minimum frequency or quantity of cigarettes smoked), and 2) whether they smoked recently (e.g., past month), to sort individuals into never smokers, former smokers, and current smokers (e.g., 31). Second, assessment of trait impulsivity during a period of heightened impulsivity (i.e., mania) may result in higher overall ratings or change its concordance with the behavioral measures. However, our prior work (11) showed that the stability of BIS-11 scores in BD participants across mood states (i.e., following transition from a manic or mixed state to depression or euthymia) was similar to their stability in healthy control participants with repeated administration, and with only a few exceptions, the discrepancies between the BIS-11 scores and performance on the behavioral tasks were consistently observed across mood states. Third, we did not incorporate measures of the severity of smoking, other substance use disorders, or psychiatric comorbidity; or consider the number and dosage of psychotropic medications taken in our analyses, which may have affected our ability to detect a relationship between smoking and impulsivity. Finally, although there are potentially detrimental effects of smoking abstinence (i.e., withdrawal effects) on cognitive task performance (e.g., sustained attention), the study visits were generally kept to less than 2 hours, decreasing the likelihood of withdrawal-related decrements in performance (32).

Despite these limitations, this study provides the first test of the relationship between cigarette smoking and both self-report and behavioral measures of impulsivity in BD. Similarly, our comparisons of trait impulsivity in current, former, and never smokers expand upon prior work by parsing potential relationships between impulsivity and both the initiation and maintenance of smoking. These results provide a foundation for future efforts to identify factors contributing to the high rates of smoking in adolescents and adults with BD, a critical step toward development of improved prevention and interventions for tobacco smoking. Additionally, as others (12) have suggested previously, past or current smoking may serve as an easily assessed, proxy indicator of impulsivity and its correlates in people with bipolar disorder, including suicidality (albeit not a substitute for a complete risk assessment).

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References

1. Lasser K, Boyd JW, Woolhandler S, Himmelstein DU, McCormick D, Bor DH. Smoking and mental illness: a population-based prevalence study. *JAMA*. 2000; 284:2606–2610. [PubMed: 11086367]
2. Waxmonsky JA, Thomas MR, Miklowitz DJ, et al. Prevalence and correlates of tobacco use in bipolar disorder: data from the first 2000 participants in the Systematic Treatment Enhancement Program. *Gen Hosp Psychiatry*. 2005; 27:321–328. [PubMed: 16168792]
3. Colton, CW.; Manderscheid, RW. Congruencies in increased mortality rates, years of potential life lost, and causes of death among public mental health clients in eight states. *Prev Chronic Dis* [serial online]. 2006 Apr. Available from: URL: http://www.cdc.gov/pcd/issues/2006/apr/05_0180.htm
4. Goldstein BI, Fagioli A, Houck P, Kupfer DJ. Cardiovascular disease and hypertension among adults with bipolar I disorder in the United States. *Bipolar Disord*. 2009; 11:657–662. [PubMed: 19689508]
5. Carney CP, Jones LE. Medical comorbidity in women and men with bipolar disorders: a population-based controlled study. *Psychosom Med*. 2006; 68:684–691. [PubMed: 17012521]

6. Heffner JL, Strawn JR, DelBello MP, Strakowski SM, Anthenelli RM. The co-occurrence of cigarette smoking and bipolar disorder: phenomenology and treatment considerations. *Bipolar Disord.* 2011; 13:439–453. [PubMed: 22017214]
7. Diergaarde L, Pattij T, Poortvliet I, et al. Impulsive choice and impulsive action predict vulnerability to distinct stages of nicotine seeking in rats. *Biol Psychiatry.* 2008; 63:301–308. [PubMed: 17884016]
8. Audrain-McGovern J, Rodriguez D, Epstein LH, Cuevas J, Rodgers K, Wileyto EP. Does delay discounting play an etiological role in smoking or is it a consequence of smoking? *Drug Alcohol Depend.* 2009; 103:99–106. [PubMed: 19443136]
9. Litvin EB, Brandon TH. Testing the influence of external and internal cues on smoking motivation using a community sample. *Exp Clin Psychopharmacol.* 2010; 18:61–70. [PubMed: 20158295]
10. Doran N, Spring B, McChargue D, Pergadia M, Richmond M. Impulsivity and smoking relapse. *Nicotine Tobacco Res.* 2004; 6:641–7.
11. Strakowski SM, Fleck DE, DelBello MP, et al. Impulsivity across the course of bipolar disorder. *Bipolar Disord.* 2010; 12:285–297. [PubMed: 20565435]
12. Ostacher MJ, Lebeau RT, Perlis RH, et al. Cigarette smoking is associated with suicidality in bipolar disorder. *Bipolar Disord.* 2009; 11:766–771. [PubMed: 19840000]
13. Mitchell SH. Measuring impulsivity and modeling its association with cigarette smoking. *Behav Cogn Neurosci Rev.* 2004; 3:261–275. [PubMed: 15812110]
14. deBry SC, Tiffany ST. Tobacco-induced neurotoxicity of adolescent cognitive development (TINACD): a proposed model for the development of impulsivity in nicotine dependence. *Nicotine Tobacco Res.* 2008; 10:11–25.
15. Strakowski SM, Fleck DE, DelBello MP, et al. Characterizing impulsivity in mania. *Bipolar Disord.* 2009; 11:41–51. [PubMed: 19133965]
16. Young RC, Biggs JT, Ziegler VE, Meyer DA. A rating scale for mania: reliability, validity, and sensitivity. *Br J Psychiatry.* 1978; 133:429–435. [PubMed: 728692]
17. Grober E, Sliwinski M. Development and validation of a model for estimating premorbid intelligence in the elderly. *J Clin Exp Neuropsychol.* 2001; 13:933–949. [PubMed: 1779032]
18. First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JBW. Structured Clinical Interview for DSM-IV Axis I Disorders, Patient Edition (SCID-I/P). New York: New York State Psychiatric Institute, Biometrics Research Department; 1995.
19. McLellan AT, Kushner H, Metzger D, et al. The Fifth Edition of the Addiction Severity Index. *J Subst Abuse Treat.* 1992; 9:199–213. [PubMed: 1334156]
20. Barratt, ES. Impulsivity: integrating cognitive, behavioral, biological, and environmental data. In: McCown, WG.; Johnson, JL.; Shure, MB., editors. *The impulsive client: theory, research, and treatment.* American Psychological Association; Washington, DC: 1993. p. 39-56.
21. Patton JH, Stanford MS, Barratt ES. Factor structure of the Barratt Impulsiveness Scale. *J Clin Psychol.* 1995; 51:768–774. [PubMed: 8778124]
22. Logan GD, Cowan WB, Davis KA. On the ability to inhibit simple and choice reaction time responses: a model and a method. *J Exp Psychol Hum Percept Perform.* 1984; 10:276–291. [PubMed: 6232345]
23. Cherek DR, Lane SD. Laboratory and psychometric measurements of impulsivity among violent and nonviolent female parolees. *Biol Psychiatry.* 1999; 46:273–280. [PubMed: 10418703]
24. Neuchterlein KH. Signal detection in vigilance tasks and behavioral attributes among offspring of schizophrenic mothers and among hyperactive children. *J Abnorm Psychol.* 1983; 92:4–28. [PubMed: 6833631]
25. Cohen, J. *Statistical power analysis for the behavioral sciences.* 2. Lawrence Erlbaum Associates; Hillsdale, NJ: 1988.
26. Wilens TE, Martelon M, Joshi G, et al. Does ADHD predict substance-use disorders? A 10-year follow-up study of young adults with ADHD. *J Am Acad Child Adolesc Psychiatry.* 2011; 50:543–553. [PubMed: 21621138]
27. Heishman SJ, Kleykamp BA, Singleton EG. Meta-analysis of the acute effects of nicotine and smoking on human performance. *Psychopharmacol.* 2010; 210:453–469.

28. Mooney ME, Odlaug BL, Kim SW, Grant JE. Cigarette smoking status in pathological gamblers: association with impulsivity and cognitive flexibility. *Drug Alcohol Depend.* 2011 (in press).
29. Wignall ND, de Wit H. Effects of nicotine on attention and inhibitory control in healthy nonsmokers. *Exp Clin Psychopharmacol.* 2011; 19:183–191. [PubMed: 21480731]
30. Barr RS, Culhane MA, Jubelt LE. Effects of transdermal nicotine on cognition in nonsmokers with schizophrenia and nonpsychiatric controls. *Neuropsychopharmacol.* 2008; 33:480–490.
31. Centers for Disease Control and Prevention. Cigarette smoking among adults and trends in smoking cessation—United States, 2008. *MMWR Morb Mortal Wkly Rep.* 2009; 58:1227–1232. [PubMed: 19910909]
32. Parrott AC, Garnham NJ, Wesnes K, Pincock C. Cigarette smoking and abstinence: comparative effects upon cognitive task performance and mood state over 24 hours. *Hum Psychopharmacol.* 1996; 11:391–400.

Table 1

Demographic and clinical characteristics of the sample and differences in these characteristics by smoking status.

Variable	Full sample (n=97)	Never smoker (NS) (n=42)	Former smoker (FS) (n=22)	Current smoker (CS) (n=33)
<i>Demographics</i>				
Age, M (SD)	29.0 (9.6)	28.5 (10.4)	28.6 (7.8)	30.0 (9.7)
Sex (male), n (%)	46 (47.4)	20 (47.6)	8 (36.4)	18 (54.5)
Race (White), n (%) [*]	69 (71.1)	24 (57.1)	19 (86.4)	26 (78.8)
Years of education, M (SD)	13.1 (2.5)	13.6 (2.7)	13.0 (2.1)	12.6 (2.4)
IQ, M (SD)	107.9 (19.2)	108.4 (21.0)	110.3 (26.3)	105.6 (8.9)
<i>Symptom Presentation and Comorbidity</i>				
YMRS (baseline), M (SD)	26.3 (5.5)	26.8 (5.7)	24.5 (5.6)	26.8 (5.1)
Mixed episode (vs. manic, at baseline), n (%) [*]	37 (38.1)	10 (23.8)	12 (54.5)	15 (45.5)
Non-nicotine substance use disorder, n (%) [*]	57 (58.8)	16 (38.1)	16 (72.7)	25 (75.8)
ADHD, n (%) [*]	39 (40.2)	9 (21.4)	13 (59.1)	17 (61.5)
<i>Medications</i>				
Lithium, n (%)	13 (13.4)	5 (11.9)	2 (9.1)	6 (18.2)
Anticonvulsants, n (%)	23 (23.7)	10 (23.8)	4 (18.2)	9 (27.3)
Antipsychotics, n (%)	62 (63.9)	28 (66.7)	13 (59.1)	21 (63.6)
Antidepressants, n (%)	4 (4.1)	2 (4.8)	0 (0.0)	2 (6.1)

Note: IQ score based on American Modification of the Adult National Reading Test (ANART); YMRS=Young Mania Rating Scale; MADRS=Montgomery-Asberg Depression Rating Scale; ADHD=attention-deficit/hyperactivity disorder.

¹ Includes alcohol abuse/dependence (n=45; 46%), cannabis abuse/dependence (n=38; 39%) and cocaine abuse/dependence (n=15; 16%).

^{*} Denotes significant differences among smoking status groups at the p<.05 level.

Table 2

Results of univariate and multivariate comparisons of never, former and current smokers on measures of impulsivity.

Measure	Never smoker (NS)	Former smoker (FS)	Current smoker (CS)	Uni-variate p	Multi-variate ^I p	Partial eta squared
<i>BIS-11 (n=87)</i>						
Total	65.1 (12.4)	76.1 (13.2)	79.1 (16.4)	<.001	.004	.124
Attentional	17.0 (4.3)	20.3 (4.4)	20.2 (5.2)	.007	.097	.055
Motor	24.7 (5.6)	26.6 (5.0)	30.0 (5.3)	.001	.004	.123
Non-planning	23.4 (6.2)	29.2 (6.4)	29.2 (7.8)	.001	.015	.097
<i>SSRT (n=74)</i>						
Reaction time (ms)	202 (84)	175 (57)	198 (66)	.417		.024
<i>DRT (n=95)</i>						
Impulsive choice (%)	50.1 (33.5)	35.6 (21.7)	47.2 (20.8)	.130		.043
Reaction time (ms)	1343 (1258)	1168 (652)	1602 (1465)	.429	.543	.013
<i>DSCPT (n=93)</i>						
Sensitivity	0.83 (0.15)	0.85 (0.11)	0.82 (0.13)	.762		.006
Response bias	0.49 (0.44)	0.64 (0.30)	0.55 (0.36)	.339		.024
Response time (ms)	544 (75)	558 (70)	546 (65)	.742		.007

Note: Values shown in table are unadjusted means (SD). BIS-11=Barratt Impulsiveness Scale-11; SSRT=Stop Signal Reaction Time;

DRT=Delayed Reward Task; DSCPT=Degraded Stimulus Continuous Performance Task.

^I Analyses for BIS-11 total and subscale scores controlled for polarity of the current episode (i.e., mixed or manic) and ADHD diagnosis. Analysis for DRT Reaction time controlled for sex. Because none of these variables were associated with performance on the SST or DSCPT, they were not included as covariates in those analyses.

Table 3

Correlations between self-report and behavioral measures of impulsivity.

	BIS-11 Inatt.	BIS-11 Motor	BIS-11 Nonplan.	BIS-11 Total	SSRT	DRT Impuls. Choice	DRT React. Time	DSCPT Sensitivity	DSCPT Resp. Bias	DSCPT Resp. Time
BIS-11 Inatt.	1									
BIS-11 Motor	.59***	1								
BIS-11 Nonplan.	.63***	.54***	1							
BIS-11 Total	.84***	.83***	.88***	1						
SSRT	-.06	.08	-.13	-.05	1					
DRT Impuls. choice	-.09	-.01	-.11	-.08	.06	1				
DRT React. time	-.08	-.19	-.22*	-.20	.01	-.25*	1			
DSCPT Sensitivity	-.11	-.07	-.04	-.08	-.33**	-.02	-.18	1		
DSCPT Resp. bias	.01	-.05	.06	.01	-.15	.07	-.02	.51	1	
DSCPT Resp. time	-.08	-.02	-.09	-.08	-.01	-.12	.04	.05	.38***	1

Note: BIS-11=Barratt Impulsiveness Scale-11; SSRT=Stop Signal Reaction Time; DRT=Delayed Reward Task; DSCPT=Degraded Stimulus Continuous Performance Task.

* p<.05,
** p<.01,
*** p<.001.