

Effects of smoking abstinence on impulsive behavior among smokers high and low in ADHD-like symptoms

Rebecca L. Ashare · Larry W. Hawk Jr.

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Abstract

Rationale Impulsivity, a multifaceted construct that includes inhibitory control and heightened preference for immediate reward, is central to models of drug use and abuse. Within a self-medication framework, abstinence from smoking may lead to an increase in impulsive behavior and the likelihood of relapse, particularly among persons with disorders (e.g., attention-deficit/hyperactivity disorder, ADHD) and personality traits (e.g., impulsivity) linked to impulsive behavior.

Objectives This study aimed to examine the effects of smoking abstinence on multiple measures of impulsivity among a non-clinical sample of adult smokers selected for high and low levels of ADHD symptoms.

Methods In a within-subjects design, participants selected for high or low levels of self-reported ADHD symptoms ($N=56$) completed sessions following overnight abstinence and when smoking as usual (order counterbalanced). Measures of impulsive behavior included response inhibition (i.e., stop signal task), interference control (i.e., attentional modification of prepulse inhibition (PPI) of startle), and impulsive choice (i.e., hypothetical delay discounting).

Results As hypothesized, abstinence decreased response inhibition and PPI. Although ADHD symptoms moderated abstinence effects on impulsive choice and response

inhibition, the pattern was opposite to our predictions: the low-ADHD group responded more impulsively when abstinent, whereas the high-ADHD group was relatively unaffected by abstinence.

Conclusions These findings highlight the importance of utilizing multiple laboratory measures to examine a multifactorial construct such as impulsive behavior and raise questions about how best to assess symptoms of ADHD and impulsivity among non-abstinent smokers.

Keywords Smoking · Abstinence · Impulsivity · Attention-deficit/hyperactivity disorder · Individual differences · Stop task · Delay discounting · Prepulse inhibition · Inhibitory control

Introduction

Smoking abstinence often results in cognitive and behavioral dysfunction, which may lead to subsequent relapse. Changes in impulsive behavior during abstinence have become a primary focus for identifying predictors of relapse (Perry and Carroll 2008). Impulsivity is a multidimensional construct, reflected in an inability to prevent behaviors with negative consequences, a preference for immediate rewards, and a tendency to engage in risky behaviors (de Wit 2009; Perry and Carroll 2008). In the context of smoking behavior, continued abstinence requires effort to inhibit the prepotent tendency to smoke (de Wit and Richards 2004). From a self-medication perspective, persons with high pre-existing levels of trait impulsivity, including those with attention-deficit/hyperactivity disorder (ADHD), may smoke in part to reduce their impulsive behavior. Consequently, abstinence effects on

R. L. Ashare (✉) · L. W. Hawk Jr.
Department of Psychology, University at Buffalo, SUNY,
206 Park Hall,
Buffalo, NY 14260, USA
e-mail: rlashare@buffalo.edu

L. W. Hawk Jr.
Center for Children and Families, University at Buffalo, SUNY,
Buffalo, NY, USA

impulsive behavior should be even greater among these individuals.

Impulsive behavior: a multidimensional construct

Given the multidimensional nature of impulsivity, the current study includes three facets of impulsive behavior including inhibitory control, which is comprised of response inhibition (Logan et al. 1997), interference control (Barkley 1997; Nigg 2000), and delay discounting, or impulsive choice (Reynolds et al. 2006).

Response inhibition is the ability to inhibit a prepotent response. As in the current study, response inhibition is often measured with the stop signal paradigm (e.g., Logan 1994; Logan et al. 1997), which involves two tasks: the “go task” and the “stop task”. Once the go task, typically a two-choice visual discrimination task, is well practiced, or made prepotent, a stop signal (e.g., a brief tone) is presented after the onset of some go stimuli, indicating that participants should inhibit their response on that trial (Logan et al. 1997). The timing of the stop signal adjusts dynamically based on performance on earlier stop trials to yield approximately 50% inhibition. Stimulants generally improve inhibitory control in humans, although this effect appears to be strongest among those with low baseline levels of response inhibition (de Wit et al. 2000; Fillmore et al. 2003). Smoking abstinence generally impairs response inhibition (Bekker et al. 2005; Dawkins et al. 2007; Harrison et al. 2009).

Interference control refers to the suppression of distracting stimuli in order to protect the processing of relevant stimuli (Barkley et al. 2007; Hawk et al. 2003; Nigg 2000). The present work employed attentional modification of prepulse inhibition (PPI) of startle as a measure of interference control. Prepulse inhibition refers to a decrease in the magnitude of the startle eyeblink response that occurs when a weak prestimulus (prepulse) is presented 60–500 ms prior to the onset of a startle-eliciting stimulus (for review see Filion et al. 1998). Increased allocation of resources to target stimuli results in increased PPI relative to non-targets (Ashare et al. 2007; Filion et al. 1993). The processing of the prepulse is protected from the interference caused by the disrupting startle probe through the engagement of effortful cognitive processing (i.e., interference control). Nicotine abstinence disrupts attentional modification of PPI (i.e., interference control) among smokers (Risling et al. 2007) and nicotine enhances interference among non-smokers (Baschnagel and Hawk 2008).

Delay discounting refers to the finding that rewards become devalued the longer one has to wait for them (Barratt and Patton 1983; Evenden 1999). Typical delay discounting tasks assess the degree to which smaller, sooner rewards are chosen over larger, later rewards, which is reflected in the “steepness” of the discounting curve

(Ainslie 1975). Impulsive choice is most commonly assessed via hypothetical delay discounting tasks, in which participants make choices between outcomes that will never be realized. In general, substance use disorders, including smoking, are associated with greater discounting of delayed rewards (for reviews, see Perry and Carroll 2008; Reynolds 2006). Abstinence effects on hypothetical delay discounting are inconsistent (Field et al. 2006; Giordano et al. 2002).

Role of individual differences: ADHD symptoms and trait impulsivity

Researchers have examined potential moderators, which may represent risk factors for smoking relapse, including disorders (e.g., ADHD) and personality traits (e.g., impulsivity) related to impulse control. ADHD is behaviorally identified as developmentally inappropriate levels of impulsive actions (e.g., trouble waiting turns and interrupting others), hyperactivity (e.g., fidgety), and inattention (e.g., difficulty focusing, distractibility, and forgetfulness) (DSM-IVTR; APA, 2000). Empirically, ADHD is associated with impaired response inhibition (Hervey et al. 2004; Lijffijt et al. 2005), interference control (Conzelmann et al. 2010; Hawk et al. 2003), and greater delay discounting (Barkley et al. 2001). The strong association between smoking and ADHD has led some to suggest that individuals with ADHD smoke to self-medicate symptoms of inattention and impulsivity. Indeed, individuals with ADHD report higher smoking rates and lower quit rates than the general population (McClernon and Kollins 2008; Pomerleau et al. 1993). Emerging evidence also suggests that nicotine reduces symptoms of ADHD and enhances cognitive and behavioral inhibition among adults (Conners et al. 1996; Gehricke et al. 2006; Levin et al. 2001) and adolescents (Potter and Newhouse 2004, 2008) with ADHD.

Impulsivity, a personality trait often linked to ADHD (Nigg et al. 2002), is also associated with stages of smoking behavior including initiation, abstinence, and relapse (Perry and Carroll 2008) and may represent a common link between ADHD and smoking (McClernon and Kollins 2008). In the current study, participants were selected based on self-reported symptoms of ADHD, and trait impulsivity was also assessed, in order to examine whether these individual difference factors moderate abstinence effects on laboratory measures of impulsive behavior.

Hypotheses

The present study investigated the relationship between abstinence-induced changes in several measures of impulsive behavior among adults selected for high and low levels of ADHD symptomatology. We predicted that overnight abstinence would decrease response inhibition (i.e., in-

crease stop signal reaction time (SSRT) during the stop task) and interference control (i.e., decrease differential PPI to targets compared to non-targets) and increase impulsive choice (i.e., less area under the curve (AUC) during the delay discounting task). Based on the self-medication hypothesis, we predicted that abstinence effects on laboratory measures of impulsive behavior would be stronger among participants selected for high levels of ADHD symptoms relative to those reporting few or no symptoms. We predicted a similar pattern for self-reported trait impulsivity (i.e., BIS-11; Patton, Stanford, and Barratt 1995), such that the relationship between abstinence and laboratory measures of impulsive behavior would be stronger among those reporting higher levels of trait impulsivity. When both ADHD symptoms and trait impulsivity moderate abstinence effects, we examined whether these constructs represent shared variance, or whether each uniquely predicted abstinence effects.

Methods

Participants

Adult regular smokers (≥ 10 cigarettes per day for the past year; not actively trying to cut down or quit smoking; 18–65 years old) were primarily recruited from advertisements posted on Craigslist (85%) and flyers posted on the university campus. Eligibility status was determined during an initial phone screen. Exclusion criteria were: self-reported use of psychotropic medication (including prescribed stimulants), vision or hearing problems, history of schizophrenia or bipolar disorder, current diagnosis of major depression, and current treatment for a substance use disorder. During the phone screen, participants were eligible only if they fell within the upper or lower quartile (within sex and age group) on the 18-item total ADHD symptom subscale from the Conners' Adult ADHD Rating Scale–Screening version (CAARS-S: SV; Conners et al. 1999). The final sample consisted of 25 (13 females) low-ADHD and 31 (16 females) high-ADHD participants who completed both sessions and had a CO reduction of at least 40% in the abstinent session compared to the non-abstinent session. Rather than a fixed CO criterion on the abstinence day, we strove for a 50% reduction relative to the non-abstinent visit (Harrison et al. 2009; VanderVeen et al. 2008). Eight participants fell short of this criterion, but were retained because all exhibited a CO reduction of 40% or more. This criterion balances the strength of the abstinence manipulation against the practical demands of retaining participants in an abstinence study with relatively modest remuneration. See Table 1 for demographics, ADHD symptoms, trait impulsivity, and smoking characteristics.

Table 1 Demographics and smoking characteristics ($N=56$)

Characteristic	ADHD group	
	Low ADHD ($n=25$)	High ADHD ($n=31$)
Sex (% female)	52%	52%
Age (years) ^{a, **}	44 (3)	37 (3)
Race (% minority)	20%	10%
Cigarettes/day	20 (2)	17 (2)
FTND	5.2 (0.4)	5.3 (0.4)
No. quit attempts	5 (0.6)	4 (0.6)
CO (ppm)		
Non-abstinent	37 (3)	32 (3)
Abstinent ^{**}	12 (2)	9 (1)
CO reduction (%)	65 (2)	71 (2)
Time since last cig		
Non-abstinent (h)	0.4 (0.3)	0.8 (0.3)
Abstinent (h)	15 (1)	14 (1)
ADHD T-score ^{a, *}	41 (2)	59 (2)
ADHD inattention*	40 (1.2)	60 (2)
ADHD hyperactive/impulsive*	42 (1.2)	56 (1.2)
BIS total ^{b, *, **}	55 (6)	71 (12)
Craving ^{****}		
Non-abstinent	4.0 (0.2)	4.1 (0.2)
Abstinent	4.9 (0.2)	5.3 (0.2)
Withdrawal ^{**} , ****		
Non-abstinent	1.4 (0.11)	1.7 (0.1)
Abstinent	1.7 (0.11)	2.2 (0.1)

Values are mean (SE) except where noted

* $p < 0.05$, ADHD group differences; ** $p < 0.05$, sex differences; *** $p < 0.08$, sex differences; **** $p < 0.01$, main effect of abstinence;

^a ADHD T-score represents the average total ADHD score from the 30-item CAARS/S-SV

^b BIS total reflects total BIS score averaged across abstinent and non-abstinent sessions

Procedure

Abstinence order (first or second session) was randomized and counterbalanced across participants. Informed consent was obtained during the first laboratory session. Participants reported cigarette, medication, and alcohol use in the last 24 h and caffeine use in the last 2 h. Participants provided a baseline breath CO sample, either smoked one of their usual brand cigarettes (non-abstinent session) or had a 5-min break (abstinent session), and provided a second CO sample 5 min later. Participants next completed demographic questionnaires and self-report measures of ADHD symptoms, impulsivity, craving, withdrawal, and mood. Participants completed each of the three lab tasks (order counterbalanced across

participants). Debriefing and remuneration (US \$70) were completed at the end of the second session.

Assessment instruments: laboratory measures

Tasks: stop signal task The stop signal task was presented on a monitor attached to a desktop running E-Prime software (Psychology Software Tools, Pittsburgh, PA). All trials consisted of a 500-ms warning stimulus followed by a 1,000-ms go signal (left- and right-facing arrows) and 1,000-ms blank screen intertrial interval. Participants were instructed to press labeled keyboard keys as quickly and as accurately as possible to indicate the direction the arrow faced (“z” for left; “/” for right). Following a 32-trial practice, stop signals (an 800-Hz, 100-ms, 70-dB tone) were presented on 25% of trials for a 32-trial practice and three task blocks of 64 trials each. The initial stop delay in each block was 250 ms and adjusted by 50 ms increments depending on whether the participant is able to successfully inhibit a response (Logan et al. 1997). The adjusting stop delay allows the determination of the delay at which inhibition occurs on approximately 50% of trials.

Tasks: CPT-startle task The PPI task was modeled after prior work (Rissling et al. 2007). Mini-electrodes (TDE23; Ag/AgCl) for EMG eyeblink were attached bilaterally beneath each eye, and a 30-s rest period followed. Two examples of the startle probes (50-ms 100-dB white noise; <1 ms onset) were presented through headphones. Participants were informed that they could ignore the occasional loud noise from the headphones.

Next, participants watched a series of letters presented pseudorandomly (290 ms exposure time with 1.65 s interstimulus interval) on a monitor placed 1 m in front of the subject. They were instructed to press the left mouse button following an A–X sequence. The startle-CPT task was administered via E-Prime software. Each of the two blocks of the startle-CPT task consisted of 20 single As, 20 target sequences A–X (40 stimuli), and 120 non-targets (e.g., B, T), resulting in target probability of 0.11. During each block, six startle-eliciting probes were presented at 240 ms lead intervals following six targets (As, not Xs, to avoid potential interference from movement associated with the button press) and six non-targets (letters other than A or X). Time between startle probes varied from 13 to 36 s, with a mean interval of 24 s.

Startle eyeblink amplitude unmodulated by visual stimuli was measured, as in previous work (Hazlett et al. 2001; Rissling et al. 2007), with 3-min long baseline periods before the first CPT block, between blocks, and after the second CPT block. Each contained standard CPT letters intermixed with six intervals of either 3 or 24 s, during which no stimuli were present. Startle response magnitude during these

intervals was averaged and used as the baseline for computing percent PPI. VPM software (Cook 2002) presented startle probes and sampled the amplified eyeblink EMG at 1,000 Hz. EMG was filtered at 30–500 Hz.

Tasks: hypothetical delay task Hypothetical discounting was measured using a computerized procedure (Mitchell 1999) in which subjects made 91 choices between a standard (\$100) available after one of five delays (0, 7, 30, 90, or 180 days) and one of 23 alternative amounts available immediately (e.g., “Which would you prefer: \$100 in 180 days or \$30 now?”). Question order was random.

Assessment instruments: individual differences

Adult ADHD symptoms The 30-item CAARS-S—Screening Version was administered during each experimental session (CAARS-S:SV; Appendix D; Conners et al. 1999). Participants rated their own behavior/problems on a four-point scale (0=not at all, never to 3=very much, very frequently) over the last 1-month period. Normative data based on age and sex were used to convert the raw scores to standardized T-scores for analyses.¹ Test–retest reliabilities were high for all three subscales (DSM-IV inattentive symptoms, DSM-IV hyperactive/impulsive symptoms, and DSM-IV ADHD symptoms total), ranging from $r=0.76$ to 0.87 , $ps<0.01$ and the inattentive and hyperactive/impulsive symptom scales were highly correlated, $r=0.75$, $p<0.01$. Two participants (both in the high-ADHD group) endorsed childhood diagnosis and treatment for ADHD.

Trait impulsivity The Barratt Impulsiveness Scale (BIS-11; Appendix E; Patton, Stanford and Barratt 1995) is a 30-item measure of trait impulsivity, which contains three factors: attentional impulsiveness, motor impulsiveness, and non-planning impulsiveness in addition to a total BIS score. The total BIS score, which was normally distributed across the sample, was used in all analyses given the high correlations among the three factors ($rs>0.65$).

Assessment instruments: smoking and self-report measures

In addition to basic smoking characteristics (e.g., cigarettes smoked per day, years smoked, etc.), nicotine dependence was assessed with the Fagerstrom Test for Nicotine

¹ Raw ADHD symptom scores were examined in preliminary analyses and the patterns were consistent with T-scores. Since T-scores take into account overall differences in symptom levels between men and women, T-scores were included in subsequent analyses.

Dependence (FTND; Heatherton et al. 1991). The 14-item Minnesota Withdrawal Scale (Hughes and Hatsukami 1986) assessed the degree to which smokers experienced withdrawal symptoms and was rated on a 0–4 scale (none, slight, mild, moderate, and severe). The 10-item Questionnaire of Smoking Urges—Brief (Cox et al. 2001) was used to assess subjective craving and rated on a 7-point Likert-type scale ranging from “strongly disagree” to “strongly agree”.

Data reduction

Stop task Mean RT for each block is calculated based on valid responses (i.e., RT > 200 ms), and only blocks with 20–80% inhibition and at least 80% accuracy were included in analyses (15% of blocks excluded). Stop signal reaction time was the primary dependent variable and is calculated by subtracting the mean stop delay from the mean RT on go-trials (Band et al. 2003; Logan et al. 1997).

CPT-startle task Eyeblink EMG responses were digitally integrated and scored offline (rectified, low-pass filtered with a 50-ms time constant and high-pass filtered with 30 Hz cutoff; van Boxtel et al. 1998) and scored as in our prior work (Ashare et al. 2007; Hawk et al. 2003). Approximately 5.0% of trials on both eyes were excluded for excessive baseline variability. Percent PPI was computed as: [(startle during lead stimulus–baseline startle/baseline startle) × 100%].

Hypothetical delay task Indifference points were derived using the algorithm described in (Mitchell 1999) and were used to calculate area under the curve using the following equation: $(x_2 - x_1)[(y_1 + y_2)/2]$, where x_2 and x_1 represent successive delays to receiving the standard and y_1 and y_2 the indifference-point values associated with these delays. AUC was chosen as the primary DV because this method reduces some problems associated with other measures (e.g., non-normal distribution of k values) (Myerson et al. 2001). Smaller values indicated greater discounting and impulsive choice.

Data analysis

Separate mixed models using SPSS MIXED were conducted for response inhibition (i.e., SSRT), interference control (i.e., PPI during targets and non-targets), and impulsive choice (i.e., AUC). For PPI, a linear effect of trial was included to account for habituation to startle probes across time. For all models, abstinence was a within-subject variable. In primary models, standardized T-scores for the ADHD total subscale of the CAARS:S-SV were averaged across sessions, mean-centered and entered as a

continuous between-subjects predictor. Significant interactions were followed up using simple linear regression to test simple slopes. Parallel analyses tested whether trait impulsivity (i.e., average, mean-centered total BIS score) moderated abstinence effects in place of ADHD symptoms. Sex and all sex × abstinence interactions were included. None of the three-way interactions between sex and ADHD/BIS were significant and were removed from final analyses. Age and abstinence order were included in supplementary models as covariates, but removed in the final analyses because neither substantially altered the results.

Results

Preliminary analyses

Table 1 provides descriptives for demographic and self-report measures. Participants completed the CAARS:SV and BIS-11 during both experimental sessions; test–retest reliability was high for both measures ($r_s=0.87$ and 0.85 , $p_s<0.01$, respectively), so they are averaged across sessions. ADHD symptoms were highly correlated with the BIS total scale, $r=0.74$, $p<0.01$.

As expected, abstinence increased craving, withdrawal, and negative affect, $F_s(1,53)=35$, 14.2 , and 8.1 , respectively, all $p_s<0.01$. However, these effects were not significantly moderated by either ADHD symptoms or trait impulsivity, all $p_s>0.15$. In general, the high-ADHD group reported greater levels of withdrawal and negative affect and less positive affect, $F_s(1,53)=13.9$, 11.1 , and 12.5 , respectively, all $p_s<0.01$.

Response inhibition

As predicted, abstinence increased SSRT, $F(1, 252)=6.5$, $p=0.01$. Although the interaction was not significant, ADHD symptoms × abstinence $F(1, 252)=2.7$, $p=0.10$, unprotected follow-up tests suggested that in contrast to our predictions, abstinence significantly increased SSRT in the low-ADHD group, $F(1, 252)=14.8$, $p<0.01$, but not the high-ADHD group, $F<1$ (see Fig. 1).² A similar pattern for response inhibition was found when trait impulsivity replaced ADHD symptoms as a continuous predictor.

² Mean reaction time was longer during abstinence compared to the non-abstinent session, means (SEs)=530 (16) and 510 (16), respectively, GoRT $F(1, 251)=9.6$, $p=0.002$. Reaction time variability (i.e., SDRT) was higher and percent inhibition tended to be higher during abstinence, $F(1, 250)=24.2$, $p<0.01$ and $F(1, 250)=3.6$, $p=0.06$, respectively. There were no main effects of ADHD symptoms, nor did ADHD moderate abstinence effects on any of these stop task variables, $p_s>0.17$. There were no significant effects for mean stop delay, $F_s<1$

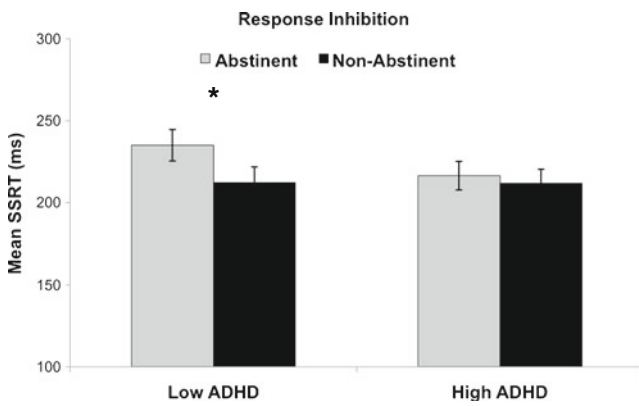


Fig. 1 Response inhibition. Mean (SE) stop signal reaction time (SSRT) for all ADHD group \times abstinence conditions

Specifically, main effects of abstinence and BIS, $ps < 0.05$, were qualified by a BIS \times abstinence interaction, $F(1, 252) = 4.7$, $p = 0.03$. When both ADHD and BIS were included in the model, neither interaction with abstinence was reliable, $ps > 0.15$, suggesting shared variance drove the univariate effects. Although the main effect of sex was not significant, $F < 1$, abstinence significantly increased SSRT in males, mean difference = 25 ms, $F(1, 252) = 12$, $p < 0.01$, but not females, mean difference = 0.7 ms, $F < 1$, sex \times abstinence interaction, $F(1, 252) = 5.9$, $p = 0.02$.

Interference control

Baseline startle magnitude was greater during abstinence, $F(1, 737) = 11.8$, $p = 0.001$ (mean difference = 1.7 μ v, SD = 1.5). Though ADHD symptoms tended to be associated with greater startle magnitude, $\beta = 0.22$, $F(1, 50) = 3.8$, $p = 0.06$, ADHD symptoms did not moderate the effect of abstinence, $p > 0.25$. There were no main effects or interactions with sex for baseline startle magnitude, $ps > 0.3$.

Figure 2 depicts mean percent PPI for all ADHD group \times attend \times abstinence conditions. Percent PPI was significantly reduced during abstinence, $F(1, 1,846) = 8.5$, $p = 0.004$ and PPI was enhanced during targets compared to non-targets, $F(1, 1,841) = 26$, $p < 0.001$. The critical three-way interaction with ADHD symptoms was marginally significant, $F(1, 1,846) = 2.8$, $p = 0.09$. Surprisingly, the enhancement of PPI during targets compared to non-targets was significant during the non-abstinent session in the high-ADHD group, mean difference = 11.3, $p < 0.01$ but not the low-ADHD group, mean difference = 5.2, $p = 0.11$. Abstinence reduced PPI to targets in the high-ADHD group, mean difference = 7.2, $p < 0.01$, but had no reliable effect on PPI in any of the other ADHD \times attend cells, $ps > 0.11$. There were no effects of BIS on baseline startle magnitude, $Fs < 1$ or PPI, $ps > 0.22$. Similar to findings for SSRT, there was a significant sex \times abstinence interaction for PPI, $F(1, 1,847) = 5.2$, $p = 0.023$,

sex $F = 1$. Abstinence reduced PPI among females, mean difference = 8.2, $F(1, 1,847) = 14.3$, $p < 0.01$, but not males, mean difference = 0.92, $F < 1$. The three-way interaction with trial type was not significant, sex \times abstinence \times attend $F < 1$.

Impulsive choice

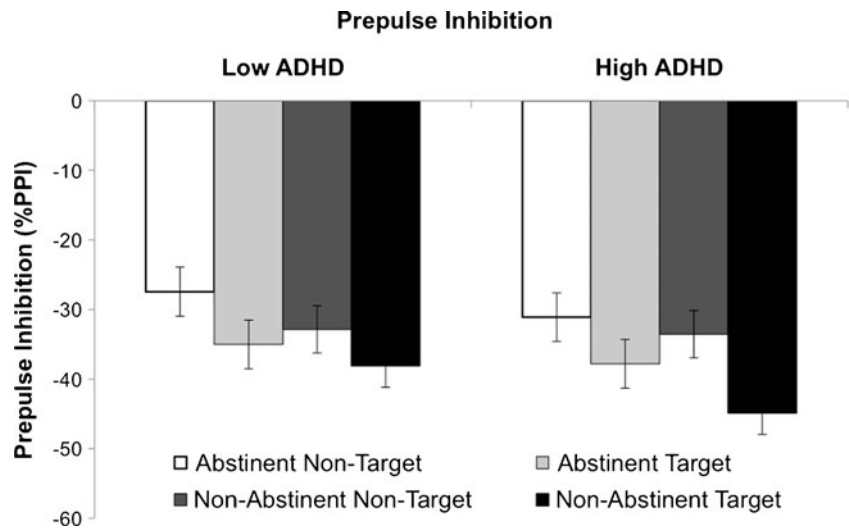
There was a significant ADHD symptoms \times abstinence interaction for AUC on the delay discounting task, $F(1, 53) = 6.2$, $p = 0.02$ (see Fig. 3). Similar to the pattern for SSRT, and contrary to our hypothesis, the low-ADHD group exhibited less AUC when abstinent compared to non-abstinent, $p = 0.03$, whereas AUC did not vary as a function of abstinence among the high-ADHD group, $F < 1$. Males tended to have less AUC than females, mean (SE) = 0.25 (0.02) and 0.30 (0.02), respectively, $F(1, 53) = 2.9$, $p = 0.09$; sex \times abstinence, $F < 1$.

Using BIS as a continuous predictor revealed a similar pattern to the finding for ADHD symptoms, with BIS moderating abstinence effects on AUC, $F(1, 53) = 7.4$, $p = 0.01$, such that abstinence resulted in less AUC among low BIS individuals, $p = 0.01$, but not among high BIS individuals, $p = 0.16$. When both BIS and ADHD symptoms were included in the model, neither variable uniquely moderated the effect of abstinence, $ps > 0.25$.

Post hoc analyses with a lab-based measure as the between-subjects predictor

The finding that abstinence effects on response inhibition and delay discounting were smaller—not greater, as predicted—among participants selected for high compared to low levels of ADHD symptoms was surprising. Because smoking could influence these symptoms of inattention and impulsivity (see Gray et al. 2010), supplementary analyses were conducted in which ADHD symptoms was replaced with a laboratory measure that incorporates attention and impulsivity. Discriminability, or d' , is a signal detection measure calculated from hit rate (i.e., correct target detections, reflecting attention) and false alarm rate (i.e., errors of commission, reflecting impulsivity) on the CPT. Given the problems of adults and children with ADHD in both domains on the CPT (see Hervey et al. 2004; Losier et al. 1996; Seidman 2006), we used response data on the CPT administered for PPI to calculate d' . Higher d' indicates better attention and/or less impulsivity. There were no abstinence effects on d' , $F < 1$ and test–retest reliability was relatively high, $r = 0.56$, $p < 0.01$. Values for d' were averaged across sessions and entered as a continuous predictor. Follow-up tests were conducted using a median-split. The d' groups were comparable on demographic characteristics including sex, age, ethnicity, and smoking characteristics, all $ps > 0.1$.

Fig. 2 Interference control. Mean (SE) percent prepulse inhibition for all ADHD group × abstinence × stimulus type conditions



For response inhibition, main effects of abstinence and d' on SSRT, $ps < 0.06$, were qualified by a significant $d' \times$ abstinence interaction, $F(1, 51) = 4.7, p < 0.05$ (see Fig. 4a). As expected, the low d' (inattentive/impulsive) group showed greater SSRT when abstinent compare to non-abstinent, mean difference = 26.5 ms, $p < 0.01$; the high d' group did not, mean difference = 1.4 ms, $F < 1$. For delay discounting, the lower d' was associated with less AUC than higher d' , $F(1, 53) = 5.2, p < 0.05$ (Fig. 4b), but this did not interact with abstinence condition, $F < 1$. There were no significant effects of d' on interference control, all $ps > 0.2$.

Conclusions

To fill gaps in the literature regarding the facets of impulsivity that may be affected by smoking abstinence and whether these effects are moderated by symptoms of ADHD, the present study examined acute effects of overnight abstinence on multiple aspects of impulsive

behavior among a sample of smokers pre-selected for high- and low-ADHD symptomatology. Although the current design did not directly measure relapse, increases in impulsive behavior during abstinence may represent an increased risk for relapse following cessation among individuals with ADHD (Groman et al. 2009). Based on theoretical models of impulsive behavior and substance use (de Wit 2009) and emerging research with adults with ADHD (Gehricke et al. 2007; McClernon et al. 2008), we predicted that abstinence would increase impulsive behavior, particularly among participants selected for high levels of ADHD symptomatology. Replicating and extending prior work, abstinence reduced response inhibition and overall prepulse inhibition. However, in direct contradiction of models of smoking and ADHD, abstinence had its greatest effects on response inhibition and impulsive choice in the low-ADHD group, whereas the high-ADHD group exhibited good performance that was not disrupted by abstinence. In light of these unexpected findings, post hoc analyses examined whether a lab-based measure of inattention and impulsivity, d' would yield results more in line with our predictions and models than did self-report. In general, this was the case, with low d' participants exhibiting a greater abstinence effect on response inhibition and greater discounting of delayed rewards across abstinence conditions. Each of these findings and the implications for future research are discussed.

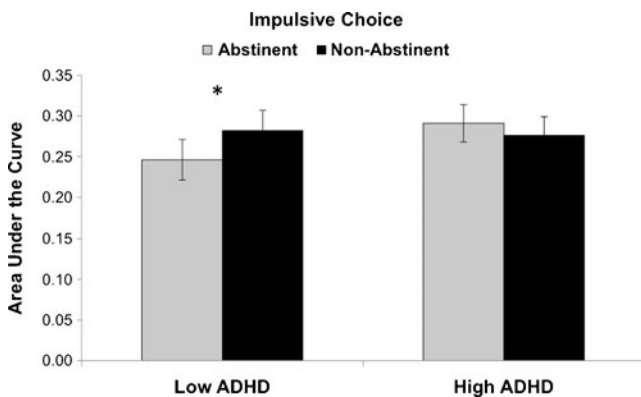
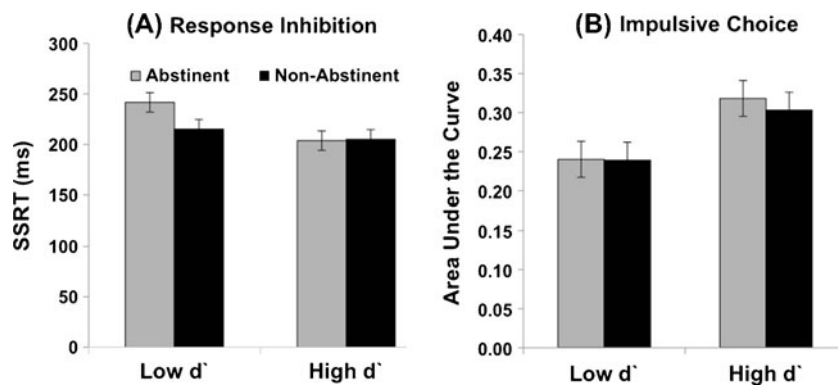


Fig. 3 Impulsive choice. Mean (SE) area under the curve (AUC) for all ADHD group × abstinence conditions on the hypothetical delay discounting task

Acute abstinence from smoking disrupted two of the three facets of impulsive behavior we examined. Consistent with recent research (Dawkins et al. 2007), the current study showed that response inhibition (i.e., SSRT during the stop task) was reduced during abstinence. This decrease in the ability to inhibit an ongoing response may be important during a quit attempt. Specifically, it may reflect a smoker’s ability to inhibit the prepotent tendency to smoke. Furthermore, PPI was generally reduced during

Fig. 4 Post hoc analyses for response inhibition and delay discounting. Means (SEs) for all d' \times abstinence conditions for response inhibition (SSRT; **a**) and impulsive choice (AUC; **b**)



abstinence, which is consistent with previous passive PPI paradigms reporting decreases in PPI following overnight abstinence among smokers (Della Casa et al. 1998; Duncan et al. 2001). Reduced PPI may reflect an abstinence-induced deficit in sensorimotor gating, which may reflect an increase in distractibility. Increased distractibility may hinder an individual's ability to inhibit urges to smoke in the context of smoking-related triggers (de Wit and Richards 2004).

Though the main effects of abstinence are important, the interactions with ADHD symptoms were of even greater interest. We found that ADHD symptoms and trait impulsivity moderated abstinence effects on response inhibition (i.e., SSRT) and impulsive choice (i.e., AUC) and this effect was marginal for interference control (i.e., PPI to targets compared to non-targets). Only the pattern for interference control was in line with our prediction that abstinence effects would be greater among the high-ADHD participants. However, this finding must be interpreted in light of the strong PPI to targets exhibited by the high-ADHD group when smoking as usual. Indeed, in no condition did the high-ADHD participants exhibit reduced attentional modification of PPI relative to the low-ADHD group, in direct contrast to work that has focused on ADHD (Ashare et al. 2010; Conzelmann et al. 2010; Hawk et al. 2003).

The data for response inhibition and impulsive choice were even more surprising. Participants selected for low levels of ADHD symptoms responded more impulsively (i.e., longer SSRT and less AUC) during abstinence compared to when they were smoking as usual. In contrast, the high-ADHD group did not demonstrate abstinence effects on either measure, nor did they exhibit a more general pattern of impulsive responding as would be predicted based on current models of ADHD and existing evidence in adults and children with ADHD (Barkley et al. 2007; Nigg et al. 2005; Shiels et al. 2009; Sonuga-Barke 2003). Since this pattern was consistent across both self-report ADHD symptoms and trait impulsivity, we investigated whether either of these measures accounted for unique variance. For both response inhibition and impulsive choice, neither univariate effect

remained significant, suggesting that moderation by ADHD symptoms and trait impulsivity was due to shared variance. Thus, while ADHD and impulsivity are clearly not interchangeable constructs, they do appear very similar in their relationships to abstinence effects on lab measures of impulsive responding.

These findings are difficult to reconcile with theoretical models regarding the links between ADHD, impulsive behavior, and smoking. The fact that the unexpected pattern occurred across two dependent variables, one based in RT and the other in decision-making, suggests that the pattern did not occur by chance. Moreover, other recent findings are similar. For example, Harrison and colleagues (2009) observed that more impulsive individuals exhibited less change in performance on response inhibition during abstinence, whereas the high impulsive individuals demonstrated few abstinence effects; a pattern that parallels the findings in the current study and ran counter to their hypotheses.

We speculate that the measurement of ADHD symptoms (and perhaps trait impulsivity) in smokers may play a role in the conflicting findings. Smokers in the current study were pre-selected based on self-reported ADHD symptoms when they were smoking as usual. Self-medication of ADHD symptoms with smoking is likely more effective for some individuals than others. The current low-ADHD group might include such "nicotine responders" (Pomerleau et al. 2003), leading them to report few symptoms while smoking. Such participants would be expected to exhibit particularly strong abstinence effects when the ameliorative effects of smoking are removed. Although this hypothesis is tentative, and it cannot account for the small effects of abstinence in the high-ADHD group, they do raise interesting questions about how best to assess impulsivity and ADHD symptoms among active smokers. In contrast to the assessment of childhood ADHD, which includes parent and teacher symptom ratings, the assessment of adult ADHD often relies on self-reported symptoms (McGough and Barkley 2004). Perhaps a more comprehensive assessment, including clinician and informant ratings, may aid our

understanding of the role of ADHD symptomatology in smoking behavior and abstinence.

Because of concerns about our self-report ADHD symptom measure, post hoc analyses tested whether individual differences in CPT discriminability, a measure that reflects two key domains in ADHD, attention and impulsivity, moderated abstinence effects. As expected, individuals with poor discriminability on the CPT exhibited the greatest abstinence effects on response inhibition and more impulsive choice than did individuals with good discriminability. Although these data should be interpreted tentatively, given the post hoc nature of the analysis, they raise the possibility that when selecting for subclinical ADHD or impulsivity domains, it may be useful to incorporate objective measures into the selection criteria. This may be less of an issue when focusing on adults who are presenting for ADHD treatment or who are recruited based on a history of childhood ADHD. Indeed, the issue of how individuals who seek treatment for and are diagnosed with ADHD differ from subclinical populations may be an important one and should be the focus of future work.

It is also plausible that participants' reporting of symptoms resulted in some reactivity during the subsequent laboratory assessments. Specifically, endorsement of symptoms of inattention, impulsivity, and hyperactivity may have led high-ADHD participants to engage more effort to perform well, even during abstinence. Indeed, motivational processes may be critical in understanding both ADHD-related cognitive impairment and abstinence-related impulsive behavior (e.g., Kalivas and Volkow 2005; Luman et al. 2005). Moreover, there is some evidence that providing performance-based incentives can improve and even normalize cognitive performance among children with ADHD, and contingency-management techniques clearly increase abstinence rates among smokers (e.g., Luman et al. 2005; Shiels et al. 2008; Volpp et al. 2009). This literature suggests that future studies of the effects of abstinence on impulsivity in ADHD may benefit from explicitly measuring, and perhaps even manipulating, motivation.

In summary, the current study replicates and extends prior work on abstinence and impulsivity. Abstinence disrupted response inhibition and an aspect of early interference control. These processes may be important predictors of relapse and future work should assess the relationship between abstinence-induced changes in impulsive behavior and treatment outcome (e.g., Mueller et al. 2009). The present data on high- and low-ADHD participants point to potential problems in the assessment of ADHD symptoms among smokers (see also Gray et al. 2010) and suggest a role for supplementing self-report with behavioral assessment. More broadly, the current study suggests that thoughtful multi-method assessment may be

critical to fully elucidating the complex linkages among ADHD, impulsive behavior, and smoking abstinence, and relapse.

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