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Breakdowns of Eye Movement Control toward Smoking Cues in Young Adult Light Smokers

Gregory J. DiGirolamo^{1,2}, Ellen J. Sophis¹, Jennifer L. Daffron¹, Gerardo Gonzalez², Mauricio Romero-Gonzalez^{2,3}, and Sean A. Gillespie¹

¹Department of Psychology, College of The Holy Cross ²Division of Addiction, University of Massachusetts-Medical School ³MAYU of New England

Abstract

Background—Many studies suggest that dependent smokers have a preference or attentional bias toward smoking cues. The purpose of this study was to test the ability of infrequent non-dependent light smokers to *control* their eye movements by look away from smoking cues. Poor control in the lightest of smokers would suggest nicotine cue-elicited behavior occurring even prior to nicotine dependency as measured by daily smoking.

Methods—17 infrequent non-dependent light smokers and 17 lifetime non-smokers performed an antisaccade task (look away from suddenly appearing cue) on smoking, alcohol, neutral, and dot cues.

Results—The light smokers, who were confirmed light smokers and non-dependent ($M_{\text{Faegerström Dependency Score}} = 0.35$), were significantly worse at controlling their eye movements to smoking cues relative to *both* neutral cues ($p < .04$) and alcohol cues ($p < .02$). Light smokers made significantly more errors to smoking cues than non-smokers ($p < .004$).

Conclusions—These data suggest that prior to developing clinical symptoms of severe dependence or progressing to heavier smoking (e.g., daily smoking), the lightest of smokers are showing a specific deficit in control of nicotine cue-elicited behavior.

Keywords

Nicotine; Smoking; Antisaccade; Control; Light Smokers; Young Adults

Corresponding Author: Dr. Gregory J. DiGirolamo, Department of Psychology, College of the Holy Cross, Worcester, MA 01610, gdirol@holycross.edu, Phone: (508) 793-3595, Fax: (508) 793-3709.

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Contributors:

GJD designed the experiment, GJD, GG, and MGG created the protocol, ES, JD, and SG created the stimuli and tested all the participants, GJD and GG prepared the materials and analyzed the data; GJD wrote the first draft, ES, JD, GG, and MR-G made significant contributions to the manuscript. All authors approve the final manuscript.

Conflicts of Interest:

All authors declare that they have no conflicts of interest.

1. Introduction

Despite the well-known and highly adverse health effects of tobacco use, most notably, heart disease, cancers of the lung, throat and mouth, and chronic obstructive pulmonary disease (Surgeon General's Report, 2004), people continue to smoke. These rates are significantly elevated among 18-25 year olds, with young adults having the highest rate of current tobacco use in the last month (37%) amongst all ages (Substance Abuse and Mental Health Services Administration, 2013). Within this age range, only half (43.1%) of these smokers were daily smokers (Substance Abuse and Mental Health Services Administration, 2013). Low-rate and non-daily smoking is, thus, common among young adults. However, intermittent smoking is an unstable pattern that typically progresses to either regular smoking or abstinence (Zhu, Sun, Hawkins, Pierce, & Cunningham, 2003). This progression to dependency is usually indexed by regular daily smoking. However, other more subtle cognitive and behavioral measures may be a more sensitive marker to *nicotine cue-elicited behavior* prior to developing severe nicotine dependency.

One method of assessing sensitivity to smoking cues in *dependent* smokers is to measure their attentional bias toward smoking cues relative to neutral cues. A plethora of studies (Bradley, Field, Mogg, & De Houwer, 2004; Bradley, Mogg, Wright, & Field, 2003; Ehrman, et al., 2002; Field, Mogg, & Bradley, 2004; Hogarth, Mogg, Bradley, Duka, & Dickinson, 2003; Kwak, Na, Kim, Kim, & Lee, 2007; Mogg & Bradley, 2002; Mogg, Bradley, Field, & De Houwer, 2003; Mogg, Field, & Bradley, 2005; Powell, Pickering, Dawkins, West, & Powell, 2004; Waters, Shiffman, Bradley, & Mogg, 2003; Waters, Shiffman, Sayette, et al., 2003) have demonstrated attentional biases to smoking cues in dependent smokers. These studies used a probe-detection task in which cues are presented briefly and a dot occurs at the location of one of the cues. Probes presented at the location where smoking cues were presented are responded to faster than probes at the location where neutral cues were presented, demonstrating an attentional bias toward smoking cues. This attentional bias has been seen in both light (defined as at least 1 cigarette per day) and heavy (≥ 20 cigarettes per day) dependent smokers (Hogarth, Dickinson, & Duka, 2009; Mogg, et al., 2005). This attentional bias to smoking cues continues in former smokers, with ex-smokers showing attentional avoidance to smoking cues (Peucker & Bizarro, 2014).

Likewise, the location and duration of where subjects look when presented with smoking cues are measured to demonstrate dependent smokers' preferences and/or attentional bias. These eye movement studies (Field, et al., 2004; Field, Mogg, & Bradley, 2005; Hogarth, et al., 2009; Kwak, et al., 2007; Mogg, et al., 2003; Mogg, et al., 2005) have demonstrated more frequent and longer fixations on smoking cues than neutral cues in dependent smokers. For example, Mogg and colleagues (2003) demonstrated that smokers, but not control subjects, looked longer at smoking cues than neutral cues. Furthermore, this bias to look longer at smoking cues was associated with an urge to smoke. Even in natural environments with cigarettes, ashtrays and other smoking related objects present, smokers look longer at smoking objects than neutral objects (Baschnagel, 2013). In short, a large body of research suggests that dependent smokers prefer smoking cues to neutral cues and that their attention is drawn more toward smoking cues than neutral cues.

The relevance of this type of attentional bias toward smoking cues has come under question (Hogarth, Dickinson, Janowski, Nikitina, & Duka, 2008). Some research has suggested a direct relationship between attentional bias and nicotine cessation treatments (Waters, Shiffman, Sayette, et al., 2003). In this study (Waters, Shiffman, Sayette, et al., 2003), smokers who were treated with either nicotine replacement therapy or placebo were asked to name the color of ink of words which were either smoking or neutral words. Smokers who showed more attentional bias (more errors or longer color naming times) toward smoking cues were significantly more likely to relapse than smokers who showed less bias. Furthermore, the smokers receiving active nicotine replacement therapy had less attentional bias toward smoking cues than those smokers on placebo. However, an additional study by Waters and colleagues (Waters, Shiffman, Bradley, et al., 2003) found that the amount of attentional bias, as measured by the dot probe task, did not predict subsequent nicotine cessation treatment outcome. A recent review paper (Christiansen, Schoenmakers, & Field, 2015) has suggested no direct relationship between attentional bias and outcomes in addiction treatment. Hence, the attentional bias paradigm might be less directly related to clinical relevance than previously thought.

While the aforementioned studies have investigated dependent smokers' preferences or attentional biases toward smoking cues (and the clinical relevance of attentional bias has been questioned), these studies have not asked smokers to *control* their behavior toward smoking cues. In the above studies, smokers are not specifically instructed to avoid looking at these smoking cues; particularly, to control behavior and inhibit a motoric response to smoking cues. A hallmark of smoking cessation, relapse prevention, and likely prevention of progression to severe dependence is the ability to control one's behavior toward cigarettes. Here we use a new paradigm (drug antisaccade task) to test the ability of young adult light smokers to control their eye movements toward smoking cues. Rather than measuring where light smokers looked when presented with a smoking cue, we specifically asked these them to control their behavior and look away from a suddenly presented cue and make an eye movement in the opposite direction (an antisaccade task, see Blaukopf & DiGirolamo, 2005, 2006; Hallett, 1978). In this task, subjects are presented with a suddenly appearing visual stimulus in the periphery (left or right) and have to suppress an eye movement towards it, in order to look to the opposite empty hemifield (an antisaccade). Subjects fail frequently to suppress this involuntary response; therefore, a good number of both successful control (antisaccades) and breakdowns of control (prosaccadic errors) are made. Within this empirical paradigm, we can test in light smokers control over eye movements to smoking cues, alcohol cues and neutral cues, and determine if smoking cues elicit more deficits in control than other cue types. Note, that we use the term breakdown to index a failure to follow goal-directed behavior in this task. The goal-directed behavior is to look away from the suddenly appearing stimulus regardless of its content, and failure to do so indexes a breakdown in cognitive control (Posner & DiGirolamo, 1998).

Unlike the attentional bias paradigms used previously, this drug antisaccade paradigm tests directly the ability of patients to control their actions (eye movements) toward drug cues. We have used this adapted drug antisaccade task to look at deficits in cognitive control toward cocaine cues in patients with cocaine use disorder (DiGirolamo, Smelson, &

Guevremont, 2015). In this novel drug antisaccade task, using cocaine and neutral cues and patients with cocaine use disorder, we found significantly higher error rates in cocaine dependent patients for cocaine cues than neutral cues (DiGirolamo, et al., 2015) demonstrating a cue-specific breakdown in cognitive control for cues related to the substance of addiction. Here, we look at cognitive control deficits, as indexed by eye movement errors in the drug antisaccade task, in people who are very light smokers and *not addicted* as indexed by daily or even frequent intermittent smoking toward smoking cues.

In the current study, we asked if *any* breakdowns in control over behavior toward smoking cues relative to alcohol or neutral cues, measured as percentage of antisaccade errors, would be found in even the lightest of young adult smokers who had not progressed yet to severe dependency (see, Mogg, et al., 2005 for attentional biases in low but dependent smokers). Also, we asked if alcohol, a drug which young adults have a significant familiarity and use of, would elicit also this type of breakdown of control over behavior, or if any deficits in control that we saw would be specific for smoking cues in these light, infrequent smokers.

2. METHODS

2.1 Participants

17 young adult light smokers ($M=20.12$ yrs. old; $SEM=0.256$), and 17 lifetime non-smoking young adults ($M=19.76$; $SEM=0.398$) were recruited from an undergraduate institution, gave informed consent, had normal or corrected-to-normal vision, and were either paid or received credit for participation. The non-smoking participants (NS) were recruited intentionally based on *never* smoking or using any nicotine products at any point in their lifetime. The smoking participants were recruited intentionally to be light and non-dependent smokers (LS). These LS were non-daily smokers and frequently went several days between smoking episodes. One index of this infrequent smoking in the LS was the last cigarette smoked prior to participating in the experiment was, on average, more than 2 days prior to the experiment ($M=56.64$ hrs., $SEM=14.69$ hrs.).

2.2 Assessments

Participants were administered the Fagerström Test for Nicotine Dependence (FTND, Heatherton, Kozlowski, Frecker, & Fagerström, 1991); a 9-item questionnaire for nicotine dependence derived from the DSM-V (American Psychiatric Association, 2013); and, the very liberal “Hooked on Nicotine Checklist” (HONC, DiFranza, et al., 2002), where any “yes” response qualifies the participant for nicotine dependence (see Hughes & Shiffman, 2008 for a critique of this measure). Alcohol risk was assessed via the “Alcohol Use Disorder Identification Test” (AUDIT, Babor, de la Puente, Saunders, & Grant, 1992), and, participants also completed the Barratt Impulsiveness Scale-Version II (BIS, Patton, Stanford, & Barratt, 1995).

2.3 Stimuli & Procedure

Stimuli were colored images of smoking cues, alcohol cues, neutral cues, and a simple blue dot. All cues were standardized to 275×275 pixels, and were presented on a 21-inch color monitor (1024×768 pixels) placed 70 cm from the participant. Eye movements were

recorded with an EyeLink 1000™ eye tracker with 1000Hz sampling and $\sim 0.1^\circ$ of resolution. Prior to the start of the experiment, after each rest, and after every 8 trials, eye position was calibrated to ensure accuracy of eye tracking.

Participants completed two blocks of 192 trials with a break between each block. Each block contained 48 trials each of smoking, alcohol, neutral, and dot cues. All cues were randomly presented throughout each block.

Each trial began with a black screen showing a central white fixation cross ($0.4^\circ \times 0.4^\circ$) flanked by two white outline boxes ($2.5^\circ \times 2.5^\circ$), 7.2° to the left and right of fixation. After fixation for either 500 or 700 ms, the fixation offset, and 500, 700, 900, or 1100 ms later a cue appeared on the left or right (fixation duration, image delay, and side of screen were presented equally often in each condition, and across each block, and randomized within the block). After an eye movement or 800 ms later, the next trial began.

Participants were instructed to move their eyes to the center of the box opposite the suddenly appearing cue. This instruction to control eye movements and make a saccade away from the cue was stressed throughout the experiment and repeated during each rest and calibration. A saccade in the direction of the cue was labeled an error (a breakdown of control); and one away from the cue was a correct movement (successful control). Saccadic reaction times (sRTs) were calculated as the interval between the onset of the image and the start of an eye movement.

Independent t-tests were computed on assessment scores, and repeated measures analyses of variance were computed as percentage of antisaccade errors (breakdowns of control) and sRTs for both control errors and successful control with within-subject factors of cue type (Smoking, Alcohol, Neutral or Dot) and between-group factor of smoking group (LS or NS).

3. RESULTS

3.1 Assessment Scores

For all tests of nicotine dependency, the lifetime NS had not a single positive answer for any question ($M = 0$; $SEM = 0$ for all nicotine assessments). The LS had a very low average score ($M = 0.35$; $SEM = 0.191$) on the FTND, demonstrating their status as light smokers and non-dependent (Fagerstrom, Heatherton, & Kozlowski, 1990). This very low FTND score, however, still had a trend for a significant difference from the NS ($t(32) = 1.852$, $p = .073$), though the NS' null scores drive this difference. Likewise, for the DSM-IV adapted questionnaire, the LS' scores ($M = 3.47$; $SEM = 0.574$) were not even close to dependency (a score of 9 is required for dependence). This score was, still, significantly higher than the NS' scores ($t(32) = 8.81$, $p < .001$), again indicative of the NS's null scores. Finally, the LS had higher HONC scores ($M = 3.88$; $SEM = 0.574$) than the NS, with $t(32) = 3.758$, $p < .001$. These nicotine assessment tests indicate that the LS are not dependent on nicotine, but are significantly different from a lifetime NS control group.

The scores between the LS and the NS did, however, significantly differ on both the alcohol risk and impulsivity assessment. The LS had significantly higher AUDIT scores ($M = 12.88$;

SEM= 0.887) than the NS (M=3.82, SEM= 0.773), with $t(32)=7.702$, $p<.001$. Likewise, the LS (M= 71.17; SEM= 3.05) had significantly higher impulsivity scores on BIS-II than NS (M=54.47, SEM=1.32), with $t(29)=5.35$, $p<.001$.

3.2 Eye-movement Analyses

Error rates (percentage of prosaccadic errors toward the suddenly appearing cue) were significantly higher overall for LS (M= 0.09, SEM= 0.01) than NS (M= 0.04, SEM= 0.01), with $F(1, 32)= 8.07$, $p< .009$. The cue type had a significant effect on the number of errors participants made to the cues, $F(2, 96)= 25.65$, $p< .0001$, with highest errors for smoking cues than either alcohol, neutral or dot cues ($p< .02$ in all comparisons). Both of these effects were, however, qualified by a significant interaction between cue type and smoker status, with $F(3, 96)= 7.04$, $p< .0001$. As can be seen in Figure 1, LS made significantly more errors overall to the picture cue types than the NS. Importantly, as Figure 1 also demonstrates, LS had a breakdown in control that was specific to smoking cues and present only in the LS.

In LS, there was a significant effect of cue type ($F(3, 48)= 19.79$, $p< .0002$). LS made significantly more errors toward the picture cue types than the dot cue ($p< .03$ in all cases). Importantly, and in sharp contrast to the NS, the LS had significantly more errors toward smoking cues than neutral cues ($p< .04$). This breakdown in control was not driven by any drug image as LS showed equivalent errors for alcohol cues relative to neutral cues ($t< 1$). However, LS showed significantly more errors toward smoking cues than alcohol cues ($p< .02$) demonstrating a nicotine-specific cue-elicited breakdown of control. Finally, LS made significantly more errors toward smoking cues than NS ($p< .004$).

Within NS, there was also a significant effect of cue type ($F(3, 48)= 6.06$, $p< .002$), and like the LS, the effect was driven by significantly more errors the picture cue types than the simple dot ($p< .009$ in all cases). There was no significant difference between the numbers of errors NS made for smoking cues compared to neutral cues, with $t< 1$. And, like the LS, NS showed no more errors for alcohol cues than neutral images ($t< 1$).

Neither the speed of the antisaccade errors or speed of successful antisaccades (sRTs) were significantly different ($p> .3$ in both cases) between the LS and the NS, and there was no interaction between cue type and smoking group in either the speed of breakdowns in control ($F< 1.5$) or successful control ($F< 1$).

4. Discussion

Standard nicotine assessments demonstrated that these young adult LS were not dependent on nicotine. Nevertheless, LS showed a distinct deficit with significantly greater eye-movement error rates for smoking cues over all other cues types including other drug cues. In short, even the lightest of smokers without severe nicotine dependence have a specific nicotine cue-elicited breakdown in control. The NS made the same number of errors to nicotine cues as neutral cues and as alcohol cues. NS had no cue-elicited breakdown in control to any substance. In contrast, the LS made significantly more errors to nicotine cues than neutral cues. In addition to having differences in nicotine usage from the NS, the LS

also had significantly higher AUDIT scores suggesting a risk for alcohol abuse. Notably, the higher alcohol risk in LS did not translate to an alcohol-specific breakdown in control as LS made equal numbers of errors to alcohol cues as neutral cues. These data suggest that while being problem drinkers, this substance is not creating a cue-elicited breakdown in control for alcohol cues. In sharp contrast, even with very low scores on all tests of nicotine dependency, the LS demonstrated a cue-elicited breakdown in control over behavior that was specific to nicotine. The LS made significantly more errors to nicotine cues than neutral cues, and significantly more errors to nicotine cues than alcohol cues. These data suggest that the nicotine may have a differential effect on cognitive control earlier in the addiction process than alcohol. It is possible that nicotine is a more powerful driver of the reward network than alcohol and may create earlier markers of dependence prior to daily use (Kalman, Kim, DiGirolamo, Smelson, & Zedonis, 2010; Kalman & Smith, 2005)

The LS also had significantly higher trait impulsivity as measured by the BIS than NS. Higher impulsivity in addiction populations is commonly found, including in smoking (Bloom, Matsko, & Cimino, 2013). Whether higher impulsivity leads to addiction, or more frequent smoking increases impulsivity remains unknown, but higher impulsivity is directly linked to the transition from infrequent to daily smoking (Hu, Davies, & Kandel, 2006). In our infrequent and non-addicted LS, we find higher impulsivity than in the NS. This higher impulsivity likely translated to an overall greater numbers of errors toward picture cues for LS vs. NS. Even with this general impairment in control over eye-movements, LS still made significantly greater errors toward nicotine cues than neutral or alcohol cues. In our previous work in patients with cocaine use disorder (DiGirolamo et al., 2015), we found that impulsivity was not directly related to breakdowns in control to drug cues. The current data suggest that even at the earliest stages of nicotine use, LS have a baseline difference in impulsivity than lifetime NS, and above that impulsivity is a specific cue-elicited deficit in control toward nicotine cues that accompanies the early stages of nicotine usage.

These data demonstrate a cue-elicited deficit in control even prior to confirmed addiction as indexed by clinic assessment or daily usage. In the current study, our infrequent light smokers were still demonstrating a specific deficit in cognitive control toward nicotine cues. These data are consistent with Mogg, Field & Bradley (2005) in which low-dependent smokers looked longer than moderately-dependent smokers at smoking cues than neutral cues (see also, Bradley, et al., 2003; Hogarth, et al., 2003; Waters, Shiffman, Sayette, et al., 2003). This attentional dwell on smoking cues was interpreted as a bias to hold attention on salient stimuli; and that smoking cues have higher saliency in low-dependency than moderate- or high-dependency. The present results, however, suggest that even in the lightest of smokers, smoking cues are capable of creating some deficiencies in cognitive control toward these cues. In short, rather than just a preference or attentional bias, the current data suggests that even prior to dependency, the lightest of smokers show more deficits in controlling successfully their behavior toward smoking cues than non-smokers. Moreover, this nicotine cue-elicited effect is specific for nicotine and is not found for alcohol stimuli even though the LS were at significantly higher risk for alcohol abuse. Note, that this data does not suggest an inability of control toward nicotine cues on par with addicted smokers who compulsively smoke. We are not suggesting a complete breakdown of control; rather, we are suggesting that on a trial-by-trial basis, LS show more deficits in

controlling their behavior toward smoking cues than NS. In short, these lightest of smokers have some deficits in cognitive control of their behavior altered by the presence of a smoking cue. In our study, LS were tasked with not looking at the smoking cues, and were successful in achieving their goal and exerting control most of the time. However, they were more likely to show deficits in this control toward smoking cues than NS. The source of this deficit in control cannot be determined from the present data. Several factors could lead to a deficit in control over their eye movement behavior, including attentional bias or preference for the smoking cue overriding, on any given trial, the desire to follow the goal-directed task of looking away from the stimulus. The present data does suggest that for the majority of trials, LS were in control of their eye movement behavior, but relative to NS, more deficits in control were seen for smoking cues than any other cue type including other drug cues (i.e., alcohol). Whether this deficit is present in the natural world remains to be determined. But the potential presence of this deficit in LS is consistent with previous data (Shiffman & Paty, 2006). Shiffman & Paty (2006) demonstrated that chippers (light smokers who do not smoke daily and are nondependent) are more stimulus-driven in their smoking behavior than heavy smokers. These authors suggested that the disappearance of stimulus control is more prevalent in light smokers and may be a critical hallmark of transition to dependence.

4.1 Conclusion

The present data suggest that these deficits in control toward smoking cues are found in even the lightest of smokers. Deficits in control toward nicotine may be present in the lightest of smokers even prior to standard measures of dependency including daily smoking. The antisaccade task may be a useful biomarker for indexing the beginnings of nicotine dependence. Subsequent on-going studies may determine the predictive value of the antisaccade task on progression to severe forms of nicotine dependency. Future research will have to determine how often light smokers smoke due to desire versus nicotine cue-elicited behavior that arise from deficits in cognitive control toward smoking cues.

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Highlights

- LS make more antisaccade errors to smoking cues than neutral cues
- LS make more errors toward smoking cues than alcohol cues
- LS make more errors toward smoking cues than NS
- Cognitive control toward smoking cues is disrupted in LS even prior to daily smoking or other indices of addiction

Figure 1.

Percentage of breakdowns in eye movement control (with SEM error bars) based on type of stimuli in light smoking young adults and lifetime non-smoking young adults. Light smokers (LS), and not non-smokers, made significantly more errors toward smoking cues than neutral cues. This breakdown in control was specific to smoking cues and not alcohol cues, and found in only light smokers and not lifetime non-smokers. Asterisks indicate significant difference at the $p < .05$ level in the direct comparison of numbers of errors toward smoking cues compared to either alcohol or neutral cues in LS.