Short communication

No untoward effects of smoking cues in anti-smoking public service announcements

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ABSTRACT

Background: Anti-smoking public service announcements (PSAs) often include smoking-related cues; however, visual drug cues can trigger acute cravings that may impede cognitive processing of the anti-smoking message. This experiment evaluated effects of smoking cues in PSAs on smoking urges, immediate smoking behavior, and persuasion measures in daily smokers.

Methods: Three-hundred and eighteen non-treatment-seeking smokers completed a single laboratory session during which they viewed sets of PSAs differentiated by presence of smoking cues (central to the PSA’s argument, peripheral, or no cues) and argument strength (high versus low). After viewing the PSAs, participants completed self-report measures of smoking urges, attitudes toward quitting, self-efficacy, and intentions to quit smoking. Smoking behavior was recorded during a 1-h ad libitum smoking period immediately following PSA viewing and assessment.

Results: There was a significant positive effect of argument strength on attitudes toward quitting smoking (p = 0.012). There were no main effects of smoking cues or smoking cue by argument strength interactions on any of the outcome measures.

Conclusions: Visual smoking cues in PSAs do not increase urges to smoke, nor is there evidence that the inclusion of such cues impedes the recall or persuasive effects of anti-smoking arguments.

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1. Introduction

Mass media campaigns employing anti-smoking public service announcements (PSAs) have shown promise in reducing smoking prevalence (Emery et al., 2012; Hu et al., 1995), although not all campaigns are successful (Durkin et al., 2012). Anti-smoking PSAs often include smoking-related cues in order to illustrate the negative consequences of smoking. However, visual drug cues can trigger cravings to smoke (Carter and Tiffany, 1999) and may play a role in relapse (Shiffman et al., 2002). Indeed, preliminary work suggests that smoking cues in anti-tobacco PSAs increase smoking urges if the central argument is weak (Kang et al., 2009). Furthermore, smokers display attentional biases to smoking cues (Bradley et al., 2004; Waters et al., 2003) that may affect cognitive processing of the PSA. By distracting smokers from the central message and providing a clear motivator to continue smoking (i.e., increased urge to smoke), the presence of smoking cues in anti-smoking PSAs could be counter-productive to the goal of reducing smoking prevalence.

We examined effects of smoking cues in PSAs on smoking urges, cognitive measures (e.g., attitudes, self-efficacy, intentions, and recall), and smoking behavior in a sample of 318 daily smokers. PSAs were coded by independent raters for the presence of smoking cues, including whether cues were central or peripheral to the PSA’s central argument. Based on prior research (Kang et al., 2009), we included argument strength (AS, low versus high) as a factor, resulting in six PSA conditions (all between-subject). We hypothesized that (1) PSAs containing smoking cues, particularly peripheral cues, would increase smoking urges (primary outcome), have a negative influence on cognitions about quitting smoking, recall of PSA arguments, and increase post-viewing smoking behavior (secondary outcomes); and (2) the negative effects of smoking cues on these measures would be more pronounced for PSAs with weaker arguments (cues by argument strength interaction). An exploratory analysis utilized eye-tracking to examine whether time spent viewing cues predicted primary or secondary outcomes.
2. Methods

2.1. PSA selection

A selection of 99 PSAs coded for argument strength (AS; Strasser et al., 2009; Zhao et al., 2011) were evaluated by both well-trained and naive raters for the presence of smoking cues. Argument strength was assessed for each PSA in this study following procedures detailed in Zhao et al. (2011). Argument strength is an aggregate rating averaged across independent samples of smokers. The ratings were obtained as a part of coding work on a large collection of anti-smoking PSAs (for example, see Zhao et al., 2011; Strasser et al., 2009; Lee et al., 2013). Argument strength raw scores were normalized by conversion into z-scores for each sample of raters and arguments tested. PSAs were selected for the current study based on high and low z score values, as reported in Table S1. PSAs were classified as having “central cues” if cues were directly part of the message, “peripheral cues” if cues were present but not directly related to the message, and “no cues” if no cues were seen in the PSA. The correlations between pairs of expert raters averaged 0.71 and the internal reliabilities for sets of naive raters averaged 0.86. Sets of four PSAs were chosen to represent each of the following AS × cue conditions: (1) low AS, no cue; (2) low AS, peripheral cue; (3) low AS, central cue; (4) high AS, no cue; (5) high AS, peripheral cue; and (6) high AS, central cue (Table S1).

2.2. Participants

Participants were screened for eligibility via telephone: eligible participants were 21 to 65 years old, reported smoking at least 5 cigarettes per day (CPD) for at least the past 6 months, and were not currently seeking smoking cessation treatment. Exclusion criteria included current use of nicotine replacement therapy or other smoking cessation treatment; self-reported history of substance use disorders (not including nicotine); physical or visual impairment that would prevent the participant from viewing the computer monitor, responding on a keyboard, or prevent successful eye-tracking (i.e., glasses); and current or planned pregnancy.

2.3. Study design and procedures

The study utilized a 3 (no cue, peripheral cue, central cue) × 2 (high versus low argument strength) factorial design. During a 2.5-h session, participants provided written informed consent followed by a breath alcohol reading (>0.01 exclusionary) and breath carbon monoxide (CO) reading (<5 ppm exclusionary). In order to standardize time since last cigarette, they smoked one of their own cigarettes and provided a second CO breath sample before completing measures on demographics and smoking history. Participants were seated in a comfortable chair approximately one meter away from the computer monitor. Eye-tracking was calibrated for each participant as described elsewhere (Strasser et al., 2012).

As described above, participants were stratified by nicotine dependence (FTND < 4 versus FTND ≥ 4) and then randomly assigned to view 4 PSAs within one of the 6 conditions. After viewing, participants completed a series of assessments (see Section 2.4) and moved to a ventilated smoking research room equipped with a sofa and television monitor, where they were asked to sit for a 1-h period as the experimenter reviewed their data. Participants were informed that smoking was permitted in this room. Using well-validated procedures for ad libitum smoking assessment (Distefano et al., 1999), a research technician observed the participant and recorded time (in min/s) to first cigarette puff and the total number of puffs taken during this period. At the end of the session, participants completed an additional measure of smoking urges and provided cigarette ratings.

2.4. Measures

2.4.1. Covariates. Participants completed standardized questionnaires on demographics and smoking history. The Fagerström Test for Nicotine Dependence (FTND; Heatherton et al., 1991) assessed nicotine dependence, and the Questionnaire of Smoking Urges – brief version (QSU-Brief; Cox et al., 2001) assessed baseline smoking urges.

2.4.2. Outcome measures. The primary outcome was smoking urges assessed using the QSU-Brief (Cox et al., 2001). Secondary outcomes assessed persuasive effects of the PSAs: Intention to quit smoking in the next 3 months was assessed using two items “how likely to attempt...” and “how likely to succeed...” rated on a 4-point Likert scale (1 = “I definitely will not”, 4 = “I definitely will”); (Cronbach’s alpha = 0.89; (Norman et al., 1999). Attitudes toward quitting were assessed using 7-point differential scales asking participants to rate “quitting smoking completely and permanently in the next 3 months” as: bad/good, unenjoyable/enjoyable, unpleasant/pleasant, foolish/wise, and harmful/beneficial (Cronbach’s alpha = 0.73; Strasser et al., 2009; Yzer et al., 2003). Perceived self-efficacy was assessed using 10 items asking participants to rate on a scale of 1 (“not at all sure”) to 4 (“completely sure”) their ability to “Avoid smoking again after an initial quit attempt,” etc. (Cronbach’s alpha = 0.92; Strasser et al., 2009; Yzer et al., 2003). Finally, participants answered four true/false statements assessing recall for each PSA. Correct answers counted as one point; incorrect answers or an answer of “I don’t know” counted for zero points. An overall recall index was created by summing scores across all four PSAs (maximum possible score = 16).

2.5. Statistical analysis

Descriptive statistics were obtained for all variables. The primary behavioral outcome (pre- to post-PSA change in smoking urges) was examined using a 3 by 2 ANOVA (including CPD as a covariate). Secondary outcomes (cognitive and smoking behavior measures) were examined using similar models. An exploratory analysis utilized linear regression models to estimate the influence of time spent looking at smoking cues on smoking urges (eye-tracking measure), intent to quit smoking, attitudes about quitting, perceived self-efficacy, and recall (AS, cue condition, and CPD were included as covariates). Our sample size of 318 participants provided >80% power to detect a moderate effect size with r = 0.05.

3. Results

3.1. Descriptive statistics

Three hundred eighteen participants completed the study. Of these, 160 (50.3%) were female; the majority were African American (63.8%) and reported education beyond high school (69.8%). The mean age was 32.5 years (SD 9.9, range 20–61) and mean CPD was 13.9 (SD 5.8, range 5–30). There were no significant differences in age, sex, or CPD among PSA conditions.

3.2. Primary outcome: smoking urges

Across all conditions, there was a significant increase in reported smoking urges from baseline to the post-PSA assessment; the mean QSU-B score increased from 2.01 to 2.69 (standard error (SE) 0.07

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1 Supplementary material can be found by accessing the online version of this paper. See Appendix A for more details.
Smoking cues presented in other settings have been shown to increase smoking urges. For example, smokers presented within in vivo smoking cues or visual cues exhibit increases in smoking urges compared to control conditions in various laboratory settings (Carter and Tiffany, 1999). Also, the presence of smoking cues in movies can increase smoking urge in viewers who smoke (Lochbuehler et al., 2009; Sargent et al., 2009). In contrast, the anti-smoking arguments within anti-tobacco PSAs may suppress cue-induced smoking urges, or suppress the reports of such urges; however, the lack of evidence for cue effects on any of the cognitive or smoking behavior measures suggests that these findings do not reflect under-reporting of urges. On the other hand, suppression of cue-induced urges by anti-smoking arguments may suggest a possible mechanism for positive associations between anti-smoking PSA exposure and reduced relapse among recent quitters (Wakefield et al., 2013). Future studies employing a similar design and incorporating a “no-argument” condition may offer more insight into the influence of anti-smoking arguments on smoking urges.

It should be noted that a previous study did report a trend toward greater increases in smoking urge following viewing of anti-tobacco PSAs with smoking cues present, when the PSAs were low in AS (Kang et al., 2009). Another study suggested that smoking cues may undermine PSAs persuasive effects in former smokers, although there was no effect on smoking urges (Lee et al., 2013). However, there are differences in study design between the current study and the prior studies that may account for differing results. For example, Kang et al. (2009) employed a within-subject design wherein the cue condition was always presented after the no-cue condition. Thus, cue presentation and time since last cigarette were confounded; however, it should be noted that increases in urges were observed only in the low argument strength condition. The current study employed a between-subject design for cue condition to minimize this confound, and utilized a larger sample size to provide sufficient power to detect moderate effects on smoking urges and behavioral outcomes; we detected no such effects. It is possible that smoking cues in PSAs produce a smaller effect on smoking urges that we could not detect; however, the clinical relevance of such an effect is uncertain.

This study is not without limitations. We report results based on a single exposure to four PSAs; repeated exposure to anti-smoking messages over time may be necessary to prompt changes in attitudes or behavior (Emery et al., 2012; Wakefield et al., 2008), regardless of smoking cues. Furthermore, the PSAs in this study were drawn from existing campaigns. Since no campaign would deliberately employ weak arguments as interventions, differences in AS across conditions (although validated; Zhao et al., 2011) were limited. Finally, although the lack of cue effects on smoking behavior was consistent with reported smoking urges and persuasion measures, our measurement of smoking behavior was conducted in a laboratory setting which may not fully reflect effects of cues in a more naturalistic setting.

Anti-smoking PSAs can be a powerful tool for tobacco control programs (Siegel, 1998; Wakefield et al., 2010), and identification of factors which impact the persuasiveness of a PSA will aid development of more effective programs. Our results indicate that, despite general associations between smoking cues and urge to smoke, the presence of smoking cues in PSAs does not increase smoking urges, prompt changes in immediate smoking behavior, or have untoward effects on cognitions about quitting smoking.

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Contributors

M. Falcone was responsible for data analysis and manuscript writing; C. Lerman and J.N. Capella were responsible for study design, interpretation and manuscript writing; C. Jepson analyzed data and assisted in manuscript preparation; P.M. Sanborn assisted in data collection, scoring and manuscript preparation; and A.A. Strasser was responsible for study design, data analysis and manuscript writing. All authors have approved the final version of the manuscript.

Conflict of interest

Dr. Lerman has served as a consultant and has received research funding from Pfizer that is unrelated to this project. The other authors declare no conflicts of interest.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.drugalcdep.2013.05.006.

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