RESEARCH REPORT

Cue exposure treatment for smoking relapse prevention: a controlled clinical trial

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Abstract

Aims. In an additive design, test the efficacy of cue exposure treatment for smoking relapse prevention as an adjunct to current standard cognitive behavioral and pharmacological treatments. Design. Randomized, controlled clinical trial. Setting. Outpatient behavioral medicine clinic. Participants. One hundred and twenty-nine cigarette smokers recruited through newspaper advertisements. Intervention. After receiving an initial counseling session for cessation and setting a quit day, 129 smokers were randomly assigned to one of four relapse prevention treatment conditions: (1) brief cognitive behavioral; (2) cognitive behavioral and nicotine gum; (3) cognitive behavioral and cue exposure; and (4) cognitive behavioral and cue exposure with nicotine gum. All smokers met individually with their counselor for six RP sessions. Measures. Seven-day, point-prevalence abstinence rates (CO verified) taken at 1, 3, 6 and 12-months post-treatment and time to first slip. Findings. All manipulation checks and process measures suggested that the treatments were delivered as intended. There were no significant differences between conditions in point-prevalence abstinence rates or in time to first slip. Conclusions. These results call into question the utility of cue exposure treatment for smoking relapse prevention.

Introduction

One-year abstinence rates reported in studies of clinic-based behavioral smoking cessation programs have hovered between 20 and 30% during the last two decades (Schwartz, 1987; Lichtenstein & Glasgow, 1992; Shiffman, 1993; Law & Teng, 1995). Some investigators have argued that cessation rates have remained static because an upper limit has been reached in theory and research developing innovative treatments for smoking cessation and relapse prevention (Abrams et al., 1991; Shiffman, 1993). Exposure-based treatments have shown promise in treating some addictive disorders (Monti et al., 1993; Dawe et al., 1993; see also Drummond et al., 1995), and may be useful for smoking treatment (Abrams et al., 1988; Niaura et al., 1988; Brandon et al., 1995)

Smokers who are exposed in the laboratory to cues associated with smoking show cardiovascular reactivity and increases in smoking urges compared to when they are presented with neutral cues (Abrams et al., 1988; Tiffany & Drobes, 1990; Payne et al., 1991; Niaura et al., 1992; Maude-Griffin & Tiffany, 1996) and increased cue reactivity predicts decreased likelihood of...
successful cessation (Abrams et al., 1987; Abrams et al., 1988; Niaura et al., 1989a, 1989b). Despite some debate as to the precise mechanisms contributing to cue reactivity (Laberg, 1990), there is emerging consensus that such mediating mechanisms involve multiple cognitive, behavioral and physiological response systems (see Niaura et al., 1988; Marlatt, 1990; Tiffany & Drobes, 1990; also see Rapee, 1991; Hammersley, 1992). Thus, the incremental use of cue-exposure treatment along with established pharmacological and cognitive–behavioral skills training (i.e. coping) treatments may provide a stronger relapse prevention strategy for smoking than currently available treatments. To date, however, no randomized, adequately controlled trials which test the efficacy of cue exposure treatment for smoking relapse prevention have been conducted.

Two studies compared cue exposure treatment to a rapid smoking procedure and found nearly equivalent abstinence rates both at a 6- and 12-month follow-up (Raw & Russell, 1980; Corty & McFall, 1984). Gotestam & Melin (1983) reported no differences in abstinence rates at a 1-month follow-up between smokers assigned to receive imaginal cue exposure treatment, relaxation training or a wait-list/control. In an additive design, Brandon, Zelman & Baker (1987) compared the effects of cue exposure treatment alone to cue exposure plus rapid smoking, but found that both of these treatments were as effective as a no-treatment control condition in promoting abstinence at a 1-year follow-up. Although some of these studies were randomized trials, each of them suffered from limitations—for example, a lack of appropriate comparison groups, inadequate theoretically derived treatment rationale and lack of internal validity checks to assure that the interventions were delivered and received as intended.

Shiffman (1993) concluded that research has become stagnant in the clinical approach to smoking cessation and that innovative new approaches are required. Such approaches need to outperform the current “gold standards” of cognitive behavioral and pharmacological treatments. Moreover, he makes several other recommendations to reinvigorate the field: (a) evaluation of treatments that combine theoretically meaningful components; (b) more careful translation of theory into practice by going beyond cognitive instructional methods; (c) measuring treatment process and linking specific (active) components of the treatment amalgam to outcomes; and (d) a reemphasis to basic science on smoking.

The present study tested the efficacy of cue exposure treatment for smoking relapse prevention over and above current “gold standard” cognitive–behavioral and pharmacological treatments, following Shiffman’s (1993) recommendations. A sample of cessation motivated smokers received a standard self-help session based on the American Lung Association guidelines and were randomized on their target quit day to one of four treatment conditions for relapse prevention: (1) brief cognitive–behavioral; (2) cognitive–behavioral treatment with nicotine polacrilex gum; (3) cognitive–behavioral treatment and cue exposure and; (4) cognitive–behavioral treatment and cue exposure and nicotine polacrilex gum. The main outcomes of interest were biochemically verified point prevalence abstinence rates at 1, 3, 6 and 12 months and time to first slip. In addition, we examined the effect of process measures (self-efficacy; urges) on treatment outcome.

Method

Subjects
One hundred and twenty-nine cigarette smokers (50% female) were recruited from the local community through newspaper advertisements. Exclusion criteria were: taking medication for a chronic medical condition; suffering from a major illness within the last 6 months; receiving treatment for any psychiatric disorder in the past year; using recreational drugs; and using other forms of tobacco. There were no significant differences in baseline demographics or in smoking history variables between treatments. Participants had a mean age of 43.5 (SD = 11.1) years and completed an average of 14.5 (SD = 2.3) years of school. They smoked an average of 26.9 (SD = 10.4) years and had a mean daily smoking rate of 27.8 (SD = 12.0) cigarettes, and had a mean Fagerstrom Tolerance score (Fagerstrom, 1978) of 6.4 (SD = 2.0).

Procedure

Overview. All subjects were seen by PhD-level therapists on an individual basis. The main intent of the protocol was to provide all subjects in all conditions with the same program to help
them to quit smoking (brief cessation treatment), then to test whether different relapse prevention programs had differential efficacy in preventing relapse (relapse prevention condition). Thus, all subjects received the same program to help them quit, but received different programs to help with preventing relapse.

Subjects scheduled their target quit day (TQD) 1 week after the brief cessation treatment session. The TQD marked the start of subjects’ assigned relapse prevention condition treatment. Regardless of the relapse prevention condition to which subjects were assigned, all sessions were scheduled twice during for the same week as the quit day, and three more times during the following week. Sessions could extend into a fourth week if scheduling difficulties precluded completion of treatment within the first three weeks. Thus, the relapse prevention sessions were equated on number of sessions in order to control for the effects of contact time on treatment outcome. Follow-up telephone interviews were conducted 1, 3, 6 and 12 months following the end of treatment.

Brief cessation treatment session. All subjects signed informed consent and completed a baseline assessment battery, which included the Smoking Triggers Interview (STI). The STI is a personalized hierarchy of high-risk-for-relapse smoking situations that is generated by asking subjects to identify smoking triggers: situations or events which were accompanied by intense urges to smoke and diminished efficacy to resist smoking. Subjects were asked to recall and rate at least six situations on 11-point scales (1 = not at all; 11 = extremely) assessing levels of smoking urge and self-efficacy to resist smoking urges. The situations were then rank ordered according to most intense urge and least efficacy at resisting smoking urges. Subjects were encouraged to provide details about visual, environmental and olfactory cues, as well as thoughts and feelings accompanying each situation.

All subjects were given the American Lung Association (ALA) self-help manual, Freedom from Smoking for You and Your Family. A CO reading was obtained (Ecolyzer, National Draeger, Inc.), and feedback was provided to the subject. The therapist reviewed the subject’s smoking and quitting history, reasons for smoking and wanting to quit and reinforced his or her commitment to quit. Smoking was presented as both a learned habit and a physical addiction. In addition, the subject was instructed to quit the evening before the second session and to use the ALA manual guidelines. Subjects then signed a quit smoking contract and were informed that in subsequent sessions they would be randomized to different treatments for relapse prevention. Counselors were kept blind to the relapse prevention condition to which subjects were assigned.

Relapse prevention conditions
Brief cognitive–behavioral. The brief treatment was designed to provide an orientation and overview of relapse prevention (i.e. coping skills) training, but to encourage the subjects to read about and learn coping skills through the ALA manual (i.e. self-help). Treatment sessions were designed to be brief (15 minutes); thus, less time was spent in discussion, training and practice of the cognitive–behavioral treatment components. Time was generally spent reviewing subjects’ progress and reinforcing their use of the ALA manual. The minimal treatment was designed to serve as a benchmark (controlling for contact frequency) against which to gauge the efficacy of the more intensive treatments. It also represents the kind of brief, effective intervention that is possible in public health or medical office settings and can be delivered less expensively (Fiore et al., 1996; Goldstein & Niaura, 1998).

Cognitive–behavioral treatment with nicotine gum. This treatment was designed to be an intensive cognitive–behavioral relapse prevention intervention: teaching and rehearsing coping skills, identifying high-risk situations, discussing the abstinence violation effect, coping with slips and relapses and discussing life-style management (see Marlatt & Gordon, 1985). In addition, they were administered nicotine chewing gum (2 mg) and instructed to use it ad libitum according to the manufacturer’s instructions. The gum was presented as an aid to smoking cessation in terms of its ability to attenuate some symptoms of nicotine withdrawal. Treatment was structured around the information and exercises presented in the ALA manual. Treatment components included: self-monitoring to identify and manage smoking triggers via smoking diary; management of nicotine withdrawal symptoms through pharmacological (nicotine gum) and
non-pharmacological means; reviewing reasons for quitting smoking; developing behavioral and coping strategies to deal with high-risk situations; relapse prevention training with a focus on managing the abstinence violation effect; time and stress management; increasing social support for not smoking; and controlling weight gain. This cognitive–behavioral protocol is a multi-component strategy and reflects approaches, based on accepted theory, that have been tested extensively and are currently in use in most smoking cessation programs in this country (Schwartz, 1987; Brown & Emmons, 1991; Abrams et al., 1991; Fiore et al., 1996). This condition was designed to serve as a state-of-the-art comparison for the cue exposure treatments (see Fiore et al., 1996). Treatment sessions were designed to last 60 minutes.

**Cognitive–behavioral treatment with cue exposure.**

Cue exposure was presented as a method for breaking the bond between smoking triggers and urges. Subjects were informed that cue exposure treatment would begin at the next session by using the ratings and detailed descriptions of the high-risk situations in the cue exposure role-play situations. This condition also included the elements of the cognitive–behavioral treatment described above (except for the nicotine gum). Thus, this condition represented the addition of cue exposure to the “gold standard” cognitive behavioral treatment described above (without nicotine gum).

At the next session, the rationale for cue exposure was briefly reviewed, and the highest risk situation from the hierarchy was selected for the first cue exposure exercise. Subjects were instructed to imagine themselves in this situation. They were instructed to imagine the situation as vividly as possible, focusing on the full range of accompanying cues, thoughts and feelings in order to allow the urge to smoke to build to the highest degree possible. To facilitate this process, the therapist described the situation based upon the detailed notes taken during the previous session. Subjects were also encouraged to describe aloud their urge to smoke and cues which rendered the situation salient. In addition, subjects were instructed to observe, manipulate, smell and sham-smoke their favorite brand of cigarette if this would further enhance the urge to smoke. Subjects were prompted to monitor the strength of the urge until it began to diminish and then to inform the therapist when the urge was half as strong as it was at maximum intensity. Subjects were then asked to verbally rate the maximum intensity of the urge just experienced, as well as self-efficacy to not smoke, using the 11-point scales described above. Subsequently, the therapist asked subjects to describe what they noticed when they were imagining this situation, whether they felt comfortable engaging in this process and ways that they might cope with this situation by using cognitive coping strategies such as distraction, delay, thinking about reasons to quit smoking, negative aspects of smoking and positive aspects of quitting. Spontaneously occurring coping strategies were reinforced. The cue exposure exercise was then repeated until the end of the session had been reached.

This same sequence of exercises was then repeated for each of three situations that were next in sequence in the high-risk hierarchy. During subsequent sessions, the cue exposure exercises took the same form, moving down the high-risk hierarchy as the subject habituated within session to each situation. Based on our pilot experience, we anticipated that the situations identified and ranked in the STI would (1) change rank order between sessions; (2) no longer provoke sufficient urge to smoke; or alternatively that (3) new situations would be identified or become more salient. Therefore, at the outset of each subsequent session, subjects were asked to again rank order situations in the hierarchy, and they were allowed to introduce two new situations based upon their immediate experience quitting smoking. Thus, at least the two most powerful situations from the previous session were retained, and a maximum of two new situations could be incorporated into the session. Subjects were encouraged to practice the cue exposure exercises at least once daily between sessions, and to begin to utilize coping skills that were discovered to be useful during the cue exposure sessions. If they felt that they could not resist smoking in some situations outside of the session, subjects were told to try to avoid exposure until they practiced and felt more confident. Each session lasted about 1.25 hours.

**Cognitive–behavioral treatment and cue exposure with nicotine gum.**

Cue exposure treatment proceeded as described above with the addition of nicotine gum. Subjects were provided nicotine
gum free of charge (2 mg Nicorette), which they were instructed to use ad libitum according to the manufacturer’s instructions. The gum was presented as an aid to smoking cessation in terms of its ability to attenuate some symptoms of nicotine withdrawal. It was also presented as potentially complementing the effects of cue exposure. Subjects were able to request nicotine gum for up to 2 months after the end of treatment. Cue exposure with nicotine gum sessions lasted 90 minutes.

The addition of nicotine gum to cue exposure treatment was prompted by theoretical and practical questions. Conceivably, nicotine replacement could boost the effect of cue exposure because it facilitates non-reinforcement of smoking urges. However, studies suggest that gum does not attenuate urges as much as other symptoms of nicotine withdrawal (Fiore et al., 1996). Thus, cue exposure, by specifically diminishing smoking urges, may complement the effects of nicotine gum on withdrawal symptoms.

**Measures**

**Self-efficacy, smoking urge and motivation.** All subjects completed a 19-item self-efficacy questionnaire and a 19-item urge questionnaire during the first visit and at the end of treatment. They rated their self-efficacy to not smoke (1 = not at all confident; 11 = very confident) and urges to smoke (1 = no urges at all; 11 = very strong urge) in 19 high-risk for relapse situations and their responses were summed to yield an aggregate self-efficacy score and an aggregate urge score. Motivation to quit was assessed for all subjects at the first visit using an 11-point scale (1 = not at all motivated; 11 = very motivated). Subjects assigned to the cue exposure conditions rated their self-efficacy to not smoke and their smoking urge at the outset and end of each treatment session on 11-point scales (1 = no urge at all, 11 = very strong urge; 1 = not at all confident, 11 = very confident).

**Consumer satisfaction survey.** At the end of treatment, all subjects completed a questionnaire which asked about their perceptions of and satisfaction with the treatment that they received. Specific questions tapped the following domains regarding treatment fidelity and integrity: subjects’ experience quitting smoking with the program; knowledge of the material they learned in the program; the structure of their program; and their perceptions of their therapist (1 = not at all, 5 = very). In addition, therapists completed an 11-point scale asking them about their confidence in each subject’s ability to quit and maintain abstinence over the course of the next year.

**Smoking outcomes.** Point prevalence smoking status was assessed during treatment and 1, 3, 6 and 12 months after treatment. Initial abstinence during treatment was defined as a self-reported 24-hour abstinence period during the week of the second visit, verified objectively though an expired air carbon monoxide value of <8 p.p.m. Abstinence at the follow-ups was defined as self-reported not smoking for the previous 7 days, and was verified using expired air CO. All subjects who reported abstinence met this verification criterion. In addition, subjects who were lost to follow-up for any reason were coded as smoking in analyses using these status points. Time to first slip began on subjects’ quit day and was defined as number of days to smoking their first cigarette.

**Results**

**Attrition**

Subject attrition at each follow-up was minimal (between 2% and 20% at any one point), and there were no significant differences in attrition at any point between any of the treatment conditions. A total of 126 subjects completed treatment (98% of the original sample; n = 98 completed >50% of treatment sessions) and 80% (n = 103) completed the 12-month follow-up. Subjects who dropped out of the follow-up period were significantly younger than subjects who completed the study (Ms = 41.3 vs. 46.2, t = -2.1, df = 126, p < 0.05). There were no other significant differences between subjects who completed the follow-up and those who dropped out on any of the baseline demographic and smoking history variables, the motivation and self-efficacy measures used in subsequent analyses or in the number of treatment sessions subjects attended.

Furthermore, there were no differences between conditions in the proportion of subjects who completed at least 50% of their assigned treatment sessions. However, subjects who completed at least 50% of their treatment sessions
were, on average, older than subjects who completed < 50% of their treatment (Ms = 46.4 vs. 41.5, t (126) = −2.12, p < 0.05); there were no other differences between groups on any of the demographic or smoking history variables.

**Treatment fidelity and manipulation checks**

Regardless of condition, subjects rated the number of sessions, the length of the treatment sessions and the amount of material covered in each session as “about right”. Subject ratings of their satisfaction with treatment (Ms range from 4.1 to 4.5) and their perceptions of their therapists’ preparation and organization (Ms range from 4.6 to 4.9) did not differ significantly between conditions. Even though the therapists could not be kept blind to which condition each subject was assigned therapists did not appear to be biased in favor of a particular treatment, as they were equally confident across conditions at the end-of-treatment of subjects’ chances of maintaining abstinence during the follow-up (Ms range from 2.9 to 3.5) and there were no therapist effects on treatment outcome at any follow-up point.

Smoking urge and self-efficacy to not smoke changed uniformly between the treatment conditions during treatment. Smoking urges decreased from the beginning of treatment (Ms range from 110.0 to 129.1) to the end-of-treatment (Ms range from 64.7 to 92.0, F = 93.2, df = 1, 88, p < 0.001), and self-efficacy scores increased significantly from the beginning (Ms range from 101.4 to 121.9) to the end of treatment (Ms range from 166.7 to 182.1, F = 174.7 df = 1, 89, p < 0.001).

A number of condition-specific analyses were conducted, suggesting that each treatment was delivered as intended. Subjects in the minimal treatment condition reported spending fewer minutes in each session (M = 17.2) than subjects in the other three conditions (M = 38.0, t = 4.74, df = 75, p < 0.001). In addition, subjects in the minimal treatment condition did not rate their therapists as effective in helping them quit as did subjects in the other conditions (Ms = 3.9 vs. 4.6; 1 = not at all, 5 = extremely, t = −3.45, df = 90, p < 0.001). Subjects who used Nicorette gum reported using about 5.5 (SD = 4.0) pieces of gum per day of treatment. Number of pieces used did not differ between the cognitive behavioral and cue exposure treatments (Ms = 5.4 vs. 5.6, respectively).

Under the theoretical prediction that cue exposure would extinguish smoking urges (Abrams et al., 1988; Niaura et al., 1988), we examined changes in smoking urges during each of the cue exposure sessions. Smoking urge was assessed at the beginning and at the end of each treatment session. Table 1 presents the mean urge ratings over the sessions for both cue exposure conditions. There were no differences between the cue exposure and the exposure with Nicorette gum condition in changes in their urges between pre- and post-treatment. Both conditions showed the greatest decreases in urges during session 1 (F = 3.8, df = 1, 31, p = 0.06) and session 2 (F = 8.8, df = 1, 27, p < 0.001), and no significant changes in urges during the remaining three sessions (Ms range from 2.1 to 3.3. For pre-treatment, to 1.8 to 2.6 for post-treatment). This pattern of results suggests that cue exposure tended to decrease urges early in treatment.

**Treatment outcome analyses**

**Point prevalence abstinence.** Table 2 presents point-prevalence abstinence rates by condition over the 1, 3, 6, and 12 month follow-ups. The table also presents the multiple point-prevalence abstinence rates between conditions, defined as meeting criteria for abstinence at all follow-up points. Pearson chi-squared tests (four 4 × 2 matrices) failed to reveal any significant differences in point-prevalence abstinence rates across any of the follow-ups. There were no significant effects found for multiple point-prevalence abstinence rates between conditions.

<table>
<thead>
<tr>
<th>Session</th>
<th>Enter</th>
<th>Exit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Session 1</td>
<td>4.0 (3.0)</td>
<td>3.3 (2.3)*</td>
</tr>
<tr>
<td>Session 2</td>
<td>4.1 (3.3)</td>
<td>2.3 (2.1)**</td>
</tr>
<tr>
<td>Session 3</td>
<td>2.7 (2.5)</td>
<td>2.3 (1.7)</td>
</tr>
<tr>
<td>Session 4</td>
<td>2.3 (2.3)</td>
<td>1.7 (1.4)</td>
</tr>
<tr>
<td>Session 5</td>
<td>3.4 (3.1)</td>
<td>2.6 (1.9)</td>
</tr>
</tbody>
</table>

*p < 0.10; ***p < 0.001.
Table 2. Percentage abstinent by condition at 1-, 3-, 6- and 12-month follow-ups and multiple point prevalence abstinence rates

<table>
<thead>
<tr>
<th>Treatment condition</th>
<th>Brief cognitive–behavioral</th>
<th>Cognitive–behavioral and nicotine gum</th>
<th>Cognitive–behavioral and cue exposure</th>
<th>Cognitive–behavioral and cue exposure with nicotine gum</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 month</td>
<td>34.4% (11/32)</td>
<td>48.6% (17/35)</td>
<td>32.3% (10/31)</td>
<td>38.7% (12/31)</td>
<td>38.8% (50/129)</td>
</tr>
<tr>
<td>3 months</td>
<td>34.4% (11/32)</td>
<td>34.3% (12/35)</td>
<td>19.4% (6/31)</td>
<td>25.8% (8/31)</td>
<td>28.7% (37/129)</td>
</tr>
<tr>
<td>6 months</td>
<td>25.0% (8/32)</td>
<td>20.0% (7/35)</td>
<td>16.1% (5/31)</td>
<td>12.9% (4/31)</td>
<td>18.6% (24/129)</td>
</tr>
<tr>
<td>12 months</td>
<td>12.5% (4/32)</td>
<td>14.3% (5/35)</td>
<td>12.9% (4/31)</td>
<td>3.2% (1/31)</td>
<td>10.9% (14/129)</td>
</tr>
<tr>
<td>MPPa</td>
<td>17.4% (4/23)</td>
<td>21.1% (4/19)</td>
<td>9.1% (2/22)</td>
<td>5.6% (1/18)</td>
<td>13.4% (11/82)</td>
</tr>
</tbody>
</table>

*MPP denotes multiple point prevalence abstinence rate.

Logistic regression analysis was used to predict point-prevalence smoking status at each follow-up from treatment condition. However, treatment did not predict smoking status at any outcome point.

Survival time. Cox proportional hazards regression was used to predict time to first slip during the 12-month follow-up period as a function of treatment condition. There was no effect of condition on hazard rates or survival time over the 12-month follow-up. The median survival times for each of the treatment groups were: brief cognitive behavioral = 8.5 days (range = 1–275); cognitive–behavioral with nicotine gum = 12 (range = 1–225); cognitive–behavioral and cue exposure = 14 (0–232); and cognitive–behavioral and cue exposure with nicotine gum = 14 (range = 0–237). The majority of subjects slipped at or before the 6-month follow-up: 77.5% of subjects in the brief cognitive–behavioral treatment, 84% in the cognitive–behavioral and nicotine gum condition and 88.1% in both of the conditions which incorporated cue exposure.

Treatment outcome efficacy analysis. We recomputed the logistic regression and Cox proportional hazards regression using only subjects who completed ≥ 50% of their treatment sessions. This efficacy analysis had no effect on outcome; completing better than half of an assigned treatment did not differentially affect treatment outcome.

Process analysis

Point-prevalence abstinence. We next examined the effects of a number of process measures, in addition to treatment condition, on 6- and 12-month abstinence rates. We tested a hierarchical logistic regression model which included treatment condition, gender, baseline smoking rate, self-efficacy, motivation and number of sessions attended as predictors. None of these variables proved significant. We also tested whether changes in self-efficacy and smoking urges over the course of treatment predicted 6- and 12-month point-prevalence abstinence. Decreases in smoking urge (B = 0.01, \( \chi^2 (1) = 4.23, p < 0.05 \)) and increases in self-efficacy (B = −0.01, \( \chi^2 (1) = 5.04, p < 0.05 \)) predicted greater likelihood of abstinence at 6-months. However, changes in smoking urges and changes in self-efficacy did not predict smoking status at 12 months.

Survival time. Using Cox proportional hazards regression, we examined the effects of the same baseline process measures, in addition to treatment condition, on hazard rates and survival time during the 12-month follow-up period. This final model was significant (\( \chi^2 = 24.85, df = 6, p < 0.001 \)). Attendance (coefficient = −0.28, Z = −3.88, p < 0.01) and baseline smoking rate (coefficient = 0.023, Z = 2.75, p < 0.01) were significant predictors of survival time. The greater the number of sessions attended, the greater the probability of remaining abstinent through the follow-up; in contrast, a greater smoking rate at the beginning of treat-
ment predicted a lesser chance of remaining abstinent through the follow-up. Treatment condition, gender, baseline self-efficacy and motivation scores were not significant predictors in the final model.

We next examined whether changes in self-efficacy and smoking urge over the course of treatment were associated with hazard rates and survival time during the 12 months. Change in self-efficacy was the only variable which predicted survival time in the final model \((B = 0.01, \chi^2 (1) = 5.52, p < 0.05)\); greater increases in self-efficacy over treatment predicted an increase in survival time over the 12 months. Changes in smoking urge did not predict survival time.

None of the interactions of these predictors with treatment condition was significant.

**Cue exposure effects.** We focused on whether the within treatment effects of cue exposure on smoking urges predicted time to first slip and point prevalence outcomes at the 6- and 12-month follow-ups. We used Cox proportional hazards regression to examine whether the number of sessions over which subjects reported changes in their smoking urges from the beginning of the session to the end of the session predicted time to first slip and point prevalence abstinence, respectively. The average number of sessions subjects reported urge decreases was 0.7 (SD = 1.0), and the mean number of sessions over which subjects reported either urge increases or no change was 1.4 (SD = 1.4). We entered each of these variables into the regression equations individually to determine their effect on outcome. The only variable which predicted time to relapse was total number of sessions over which urges increased or remained the same. The number of sessions over which urges increased or remained the same positively predicted a longer time to slipping over the 12 months \((B = 0.27, \chi^2 (1) = 6.12, p < 0.01)\) and greater likelihood of being abstinent at both the 6 \((B = -0.73, \chi^2 (1) = 4.65, p < 0.05)\) and 12 \((B = -0.54, \chi^2 (1) = 4.40, p < 0.05)\)-month follow-ups. Urge decreases did not predict survival time or point prevalence status.

**Discussion**

This study tested the efficacy of incrementally adding cue-exposure and coping skills training to existing standard cognitive–behavioral and cognitive behavioral plus nicotine gum treatments. Neither of the cue exposure group conditions outperformed the two comparison groups in the follow-ups at 1, 3, 6 and 12 months. It is unclear whether increasing the dose of the exposure treatment, varying the type of contextual stimuli or extending the length of treatment would have resulted in a more effective or higher dosage of cue exposure active ingredients. It is possible, however, that a different cue exposure paradigm (e.g. with more direct *in vivo* rather than imaginal exposure-based contextual stimuli; cf. Dadds *et al.*, 1997) would have been more effective.

Cue exposure treatment has shown some promise in opiate and cocaine drug abuse treatment (O’Brien *et al.*, 1990) and may also be useful in alcoholism treatment, (Monti *et al.*, 1993; Drummond & Glaudier, 1994). Thus, cue exposure may work better with some addictive behaviors than others (e.g. alcohol versus tobacco), which suggests that research should not assume that all addictive behaviors may be treated in the same fashion (e.g. Abrams, 1995; Niaura & Shiffman, 1995; Monti *et al.*, 1995). In any case, the results of this study clearly suggest that cue exposure treatment may not be useful and would not appear to be cost-effective to add to the currently available cognitive–behavioral and cognitive–behavioral plus nicotine gum treatments for smoking relapse prevention (see also Raw & Russell, 1980; Gotestam & Melin, 1983; Corty & McFall, 1984; Brandon *et al.*, 1987).

Although the primary hypotheses concerning beneficial effects of cue exposure treatment were not confirmed, some additional findings merit comment. First, we found no apparent benefit of adding nicotine replacement, specifically nicotine gum, to standard smoking treatments. This finding contrasts with a large literature which has found that the addition of nicotine gum to behavioral smoking treatments improves smoking cessation outcomes (see Fiore *et al.*, 1996). The reasons for this finding are unclear, although it is possible that subjects in our sample were not using enough pieces of the gum to gain an incremental clinical effect. Secondly, changes in self-efficacy were related positively to outcomes. This generally confirms results of other studies (reviewed in Baer & Lichtenstein, 1988), and reinforces the notion that dynamic changes in self-efficacy during treatment are clinically informative (Baer & Lichtenstein, 1988).
Thirdly, at least in the 6-month point-prevalence analysis, changes in urge ratings from pre- to post-treatment were inversely related to outcomes. This finding is consistent with recent studies which have pointed to the importance of smoking urges in predicting smoking outcomes in treatment-motivated samples (Killen et al., 1991; Doherty et al., 1995; Swan, Ward & Jack, 1996). Importantly, these process analyses provide key information to help explain the negative results. Urges to smoke and self-efficacy ratings in the control conditions (no cue exposure) were both no longer different, compared with the end exposure conditions, at the end of treatment.

More interesting, perhaps, are the results of the analyses of smoking urge changes within sessions for subjects who received the cue exposure treatment. Averaged across sessions, if urges stayed the same within a session or increased, the likelihood of a positive 6-month outcome improved. By contrast, urge decreases were not informative with regard to outcome. This finding is counter-intuitive to our original hypothesis that decreased urges after cue exposure, possibly reflecting increased coping efforts and/or habituation within each session, should predict a better outcome. However, this finding is consistent with results of studies on exposure treatments for anxiety disorders which suggest that increased cognitive processing of the feared stimulus bodes well in terms of improving symptoms (reviewed in Zinbarg, 1993). Thus, smokers who, as a result of cue exposure procedures, continued to ruminate about their experience and who, as a result, experienced no diminishment or even an increase in urges at the end of a cue exposure session, may have been somehow rehearsing or otherwise coping with their imagined encounter with the high-temptation situation. This, of course, is speculation, but these preliminary results warrant replication and further exploration to test this and competing hypotheses to explain the link between increased urges during cue exposure and positive smoking cessation outcome.

The study has limitations. First, our initial a priori estimates of power (Cohen, 1988) suggested that cell sizes of 30–35 subjects would be adequate to test the incremental efficacy of “gold standard” cessation treatments above and beyond a minimal cognitive–behavioral treatment (e.g. a tripling of quit rates; see Fiore et al., 1996) at 6 and 12 months. However, the study produced counter-intuitive results and we were lacking the power (e.g. $1 - \beta = 0.15$) to detect differences that were not initially expected between treatment conditions. Secondly, although the intensity of the cue exposure treatment seemed to diminish after the second of the five massed practice trials (sessions 3–7), it is unclear if this finding is specific to the cue exposure conditions because we did not assess within session changes in smoking urges in the non-cue exposure conditions.

Thirdly, we examined cue exposure as an adjunctive relapse prevention strategy, but it is not known whether cue exposure treatment prior to the quit day would have delayed relapse. Fourthly, we did not evaluate a strict cognitive–behavioral treatment against a strict cue exposure treatment (without coping skills training); thus, we cannot definitively state that cue exposure as a stand-alone relapse prevention strategy was equally as effective as a stand-alone cognitive–behavioral treatment. Fifthly, a number of pharmacological alternatives are currently available to help smokers quit (Fiore et al., 1996) and cue exposure treatment in combination with one of these novel pharmacotherapies may have proved more efficacious than combined treatment with Nicorette gum.

Despite these limitations, this study followed several recent recommendations provided to smoking treatment researchers (see Abrams et al., 1991, 1996; Lichtenstein & Glasgow, 1992; Shiffman, 1993). First, a theory-driven intervention was created and both assessment and treatment components were cumulatively developed and refined over several years of laboratory and treatment-related research on reliability, discriminant and predictive validity (Abrams et al., 1987, 1988; Niaura et al., 1988, 1989b). Secondly, it filled a gap in previous cognitive–behavioral relapse prevention models (Marlatt & Gordon, 1985), which have focused typically on managing negative emotional states and social situations as the basis for relapse prevention skills training (e.g. Monti et al., 1989). Thirdly, it assessed process mechanisms to ensure that the active ingredients in treatment were delivered and received as intended. Fourthly, an incremental design was used with credible and active comparison conditions and with a sufficient sample size to detect clinically meaningful effects. Fifthly, standard outcome measures verified 7 day quit rates at 1, 3, 6 and 12-month follow-ups.
The results of this study also point to the complexity and the challenge of future research in the smoking clinical outcome literature. We and others have argued that overall population prevalence of smoking could be better reduced by proactively disseminating reasonably efficacious self-help and tailored brief interventions (e.g., Lichtenstein & Glasgow, 1992; Orleans Glynn, Manley & Slade, 1993; Abrams et al., 1996) and by combining interventions with public health, mass communications and policy/advocacy (Abrams et al., 1991, 1996, 1999). In parallel, however, we agree with Shiffman (1993) that a return to studying basic biobehavioral mechanisms in addiction and relapse is an equally important priority. Basic science is the wellspring of the next generation of translational applications and is often confronted with negative results. The field can benefit from negative results, if those results derive from theory-driven research that examines process and mediating variables.

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References
Cue exposure treatment and smoking


