PERSONALIZED FEEDBACK FOR SMOKERS WITH ELEVATED ANXIETY SENSITIVITY

A Dissertation

Presented to

The Faculty of the Department

of Psychology

University of Houston

In Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

By

Lorra Garey

August 2019

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ELEVATED ANXIETY SENSITIVITY

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ABSTRACT

Objective: Cigarette smoking is the leading cause of preventable death and disability. The majority of smokers report a desire to quit and most make a serious quit attempt each year. Unfortunately, more than 95% of cessation attempters relapse within 6 months. Clinical and laboratory studies have identified negative affect as a potent precipitant of relapse and more severe smoking behavior. Yet, limited brief, accessible treatments exist to address the range of negative affective symptoms. One promising, integrative approach to address this need is to focus on underlying transdiagnostic processes that capture negative mood states and are related to smoking. Anxiety sensitivity (AS), the tendency to fear anxiety-related sensations, is a core transdiagnostic vulnerability factor for the etiology and maintenance of anxiety disorders and other emotional disorders, and is also related to smoking maintenance and relapse. Progress has been made in developing intensive, integrated treatments that address AS in the context of smoking treatment. However, limited efforts have focused on developing brief (single session) interventions for AS and smoking. The current study was conducted to develop, refine, and test a brief, integrated personalized feedback intervention (PFI) for smoking and AS. **Method:** Participants (N=95; 63.2% male; M_{age} = 46.20 years, SD = 10.90) included general smokers in the early stage of quitting who received either a single session, computer-delivered PFI or smoking treatment as usual. The primary aims focused on examining the effects of PFI on (1) quit attempts, (2) cigarette reduction, and (3) trajectories of affective vulnerability assessed at 2- and 4-week follow-ups. **Results:** Results indicated 48.3% of participants at 2-week follow-up and 53.4% at 4-week follow-up engaged in a self-defined quit attempt. Substantial smoking reduction was observed in 21.8% and 28.4% of participants at 1-week post-baseline and 1-week pre 4-week follow-up. Treatment condition

did not significantly predict quit attempt or smoking reduction. PFI had a significant effect on symptoms of anxiety arousal over time (β = -.32, p = .04). **Conclusions:** Current data provide preliminary evidence for the utility of a PFI to encourage behavior change related to smoking and address physical manifestations of anxiety. The effects, however, were limited in magnitude. Nevertheless, the initial 'signal' observed in this small trial provides a strong rationale for continued work within this domain.

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INTRODUCTION

Smoking Prevalence and Models of Nicotine Dependence

Cigarette smoking is the leading preventable cause of death and disability globally[1] and contributes to over 480,000 premature deaths annually in the United States (US).[2] The majority of US smokers (68%) report a desire to quit[3] and most (55.4%) make a serious quit attempt each year, primarily on their own (i.e., self-guided quit) and to a lesser extent, with assistance from formal treatment.[3,4] Unfortunately, more than 95% of cessation attempters relapse within 6 months.[5] Low cessation success and the need for repeated quit attempts (on average 6-7 with some smokers requiring 30 or more attempts[6,7]) to achieve abstinence has informed the universal recognition that nicotine dependence is a chronic condition that requires specialized treatment and repeated efforts to achieve and maintain abstinence.[8]

Several phase-based models have been proposed to capture the chronicity of nicotine dependence.[9-11] These models highlight specific phases, or stages, of nicotine dependence across a continuum ranging from pre-contemplation/motivation (the period prior to a smoker being ready to make a quit attempt) to maintenance (beyond the two week post-quit period that focuses on the maintenance of abstinence), and offer a synthesis of motivational and behavioral characteristics associated with each phase. Within this conceptual framework, relapse is identified as a critical behavior that perpetuates the chronicity of nicotine dependence by reinserting users into established cycles.[9,10] Importantly, across various models of nicotine dependence, researchers consistently document the preparation or precessation phase (i.e., phase characterized by increased motivation and commitment to change one's smoking behavior and occurs prior to engaging in a long-term quit attempt[10]) as a particularly clinically-important early phase of smoking cessation. The precessation phase

represents an opportunity for smokers to build self-efficacy for quitting and make behavioral modifications in preparation of long-term cessation, including reducing cigarettes per day and practicing initial quit attempts (defined as intentionally quitting for at least 24-hours[10,12]). The degree to which a smoker is able to practice, learn from, and re-engage in preparatory behaviors for cessation during this period is paramount to long-term abstinence.[13,14]

Numerous factors impede smokers' abilities to practice and engage in precessation behavior modifications (e.g., reduce smoking rate and practice cessation attempts). One such factor is negative affect. Negative affect reflects the experience of emotional states of distress or negative emotions, including anger, depression, anxiety, and irritability.[15] Clinical and laboratory studies have identified negative affect as a potent precipitant of relapse and more severe smoking behavior.[16-20] Indeed, negative affect is one of the most robust predictors of smoking across stages of use.[20] Conceptually, when smokers deprive themselves of nicotine, such as when they self-administer less nicotine via reduced cigarettes per day or cessation attempts, they experience acute withdrawal.[21] One of the central components of withdrawal is increased negative affect.[22] In the absence of proper skills to manage acute withdrawal symptoms, a smoker may resume their typical smoking rate or forgo a 24-hour quit attempt. This conceptualization is consistent with the negative reinforcement model of addiction, which proposes that escape and avoidance of negative affect is a prepotent motive for addictive drug use.[21] Importantly, this framework is not constrained to negative affect resulting from withdrawal, but is broad-based and applicable to situations that produce unwanted, aversive emotional states. With proper training and skills, however, smokers may learn to tolerate unwanted, aversive states and maintain reduced smoking or brief smoking abstinence, thereby increasing self-efficacy and chances of smoking cessation success.[10]

Anxiety Sensitivity: Transdiagnostic Target in Smoking Treatment

Targeting 'negative affect' in the context of smoking treatment is challenging because of difficulties clarifying treatment targets given its broad, encompassing definition. To combat these challenges, researchers have developed specialized treatments focused on individual-difference factors related to, yet also unique from negative affect, that have clear treatment targets, are responsive to intervention, and can be reliably measured.[23,24] One such individual-difference is Anxiety Sensitivity (AS). AS is a transdiagnostic, relatively stable individual difference factor that predisposes individuals to the development of anxiety/depressive problems[25] by amplifying negative mood states (e.g., anxiety[26,27]). AS has been documented in smoking maintenance and relapse processes,[24,28] including smoking motives,[24] expectancies,[29-34] and perceived barriers to cessation.[35,36] AS is also related to the tendency to smoke when confronted with smoking-relevant thoughts, feelings, and sensations (e.g., bodily tension[37]), as well as the subjective experience of more severe side effects for smoking cessation pharmacological aids.[38] Higher AS smokers tend to experience more intense nicotine withdrawal and craving during early phases of quitting,[39,40] and higher levels of AS are related to greater odds of early lapse[41] and relapse.[42]

Importantly, AS is malleable in response to psychosocial interventions,[43] making it a prime mechanistic agent to target in prevention/intervention programs. AS reduction treatment can be administered in as few as 1-4 treatment sessions both in-person or via computer, with equal success.[44-47] From a broad-based clinical perspective, reductions in AS improve clinical outcomes among clinical and nonclinical populations, highlighting the relevance of this construct within the general population.[43,48] Furthermore, integrated treatment programs that address AS reduction in the context of a smoking cessation have

demonstrated promising results regarding efficacy to reduce cigarettes per day, increase smoking abstinence, and reduce AS and anxiety/depressive symptoms compared to control conditions.[46,47,49,50] This work has primarily focused on multi-session, intensive treatments that rely on in-person, researcher administered psychosocial protocols to deliver psychoeducation, introduce and model interoceptive exposure, and teach cognitive re-structuring.[47,49,51,52] One study, however, was able to successfully distill smoking and AS information to a single, several hour group treatment.[45] Results from this study suggested that a single-session integrated treatment can increase motivation to change smoking. The impact of this work to translate to actual behavior change, however, remains unknown. These treatments, although promising, may not be sustainable due to time and financial constraints. This work sits in the larger context of a paradigm shift toward digital health for behavioral interventions.[53] Yet, no brief, digital intervention for smoking-AS exists to encourage smoking-specific behavior change and AS management/reduction for smokers in the early stages of smoking cessation.

Computer-Delivered, Personalized Behavioral Health Interventions

Computer-delivered interventions represent an unexplored intervention-delivery approach that has the potential to increase the reach and impact of integrated smoking-AS treatments via reduced patient and provider burden, time-limited (single-session) commitment, and broader dissemination through adoption in point-of-care settings.[54] Computer-delivered interventions targeting smoking[55,56] and emotional vulnerabilities[57-59] have yielded positive results. Notably, participant engagement can largely impact the success of a computer-delivered intervention. Personalizing or tailoring intervention components for the participant increases engagement and is associated with improved health behavior

outcomes.[60,61] Indeed, computer-delivered Personalized Feedback Interventions (PFIs) have been among the most successful computer-delivered treatments for behavior change.[60] These interventions provide users with personalized information on use/severity profile, risk factors, negative consequences, and normative comparisons.[62,63] The intention is to increase the salience of normative discrepancies by correcting normative misperceptions, and to reframe a behavior in terms of associated consequences and risks.

PFIs have been shown to increase motivation to quit smoking[64] and enhance treatment outcomes.[65-67] Smoking-specific personalized feedback significantly impacts smoking behavior, particularly for individuals in the early phases of quitting.[68] Moreover, personalized feedback also decreases anxiety and depressive states and vulnerabilities (e.g., negative or catastrophic thinking styles) and associated symptoms, and increases proactive (adaptive) coping strategies.[69] Personalized feedback has also been linked to reductions in AS.[70] Thus, a computer-delivered PFI for smoking and AS may be an effective, viable solution to target smoking and AS in a single, unified protocol. This approach not only combats current treatment challenges, but also would provide an accessible platform for smokers in the early stages of quitting to easily receive information to support a quit attempt (e.g., psychoeducation on methods to quit and coping with negative mood) and elements to increase motivation to quit (e.g., identification of reasons to quit).

Present Study Aims and Hypotheses

To expand the reach and impact of integrated smoking-AS treatments, the current study aimed to develop and test a novel PFI for smoking and AS among current, daily smokers in the early phase of cessation. Specifically, a pilot study was conducted to evaluate and refine a novel, integrated PFI that incorporates psychoeducation and personalized

feedback related to smoking and AS. Subsequently, the efficacy of the one-session, computer-delivered PFI intervention was compared with a smoking information control intervention in a randomized controlled trial. It is hypothesized that smokers in PFI would report (a) substantial smoking reduction, (b) greater reduction in cigarettes per day, and (c) more quit attempts lasting at least 24 hours at the 2- and 4-week follow-ups relative to the control. Additionally, it is hypothesized that smokers in PFI would report (a) greater reductions in AS, (b) greater reductions in anxiety/depressive symptoms, and (c) increased willingness to use adaptive coping strategies at the 2- and 4-week follow-ups relative to the control. Baseline AS was explored as a potential moderator of treatment effects across smoking outcomes.

PILOT STUDY

METHOD

Participants

The sample consisted of nine (55.6% male; M_{age} = 41.67 years, SD = 11.70) adult daily cigarette smokers recruited from the community. Eligibility included: (1) not being pregnant (self-reported); (2) being between 18-65 years of age; (3) report daily smoking (average ≥5 cigarettes per day for at least 1 year); (4) not presently engaged in a quit attempt or mental health treatment; (5) not currently using non-cigarette tobacco products or illicit substances regularly (defined as 3 or more times per week); (6) computer literacy; and (7) cognitive capacity to provide written, informed consent. Exclusion criteria included: (1) current treatment for an alcohol/drug problem including smoking cessation; (2) current psychiatric treatment; (3) active suicidality (i.e., suicidal ideation, intent, or plan); (4) psychosis; (5) not being fluent in English and (6) inability to use a computer independently.

The sample was primarily Black or African American (88.9%) with 11.1% Caucasian. Regarding level of education, 33.3% had a high school diploma or the equivalent, 44.4% completed some college, and 22.2% had a graduate degree. Participants smoked an average of 15.67 (*SD* = 12.42) cigarettes per day for an average of 22.67 (*SD* = 15.05) years and evinced a high level of cigarette dependence (*M* = 8.89, *SD* = 1.96[71]).

Procedures

Participants were recruited via community postings, newspaper advertising, and online media sites that targeted general smokers. Interested individuals called the laboratory and completed an initial phone-screener. Callers eligible at the phone-screener were scheduled for a single in-person baseline appointment in which full eligibility criteria were evaluated. The baseline appointment included a 30-minute interview assessment, 90-minute pre-intervention online survey, intervention, 15-minute post-intervention online assessment, and 20-minute exit interview. During the initial interview, research staff assessed for eligibility and collected an expired carbon monoxide sample to biochemically verify smoking. Self-report measures were administered at the pre- and post-intervention assessments.

Upon arrival at the baseline appointment, a trained researcher obtained informed consent and administered the interview assessment. Participants then completed the online pre-intervention online survey. After, a trained research assistant evaluated eligibility criteria. Participants who did not meet eligibility criteria were provided the National Cancer Institute's Clearing the Air (CTA) pamphlet, compensated $10, and dismissed. Participants who met eligibility criteria were provided a preliminary version of the PFI and completed a brief, 20-minute interview in which participants provided feedback on how to improve the

PFI. Research staff followed an outline of open-ended questions to conduct the semi-structured interview. Motivational interviewing[72] techniques were employed to facilitate feedback from participants. Interviewers were research assistants trained in motivational interviewing with experience administering semi-structured interviews. Notes were written during the interview and the session was also audio-recorded. Following the interview, participants received $20 and bus fare for their time.

Personalized Feedback Intervention

The PFI was modeled from those that have focused on substance use and targeted negative mood symptoms.[69,73,74] Content for the smoking portion of the PFI was informed by National Cancer Institute's Clearing the Air pamphlet. The PFI was reviewed by leading experts in PFI development, smoking treatment, and mood management, and modifications were made to materials based on their feedback prior to administration. The PFI administered to participants included a digital avatar that was matched to participant race and sex. The purpose of the digital avatar was to increase engagement and relatability. The PFI was interactive and included videos and game-like activities throughout. Both personalized feedback and generic psychoeducation were presented in the PFI. Core tenants of motivational interviewing[72] were used to guide how information was presented. Personalized feedback was based on data collected during the pre-intervention online survey. Specifically, expired CO reading, data from the SHQ, select items about motivation to quit, items regarding perceived smoking norms, and data from the ASI-3 was piped into the PFI. Intervention materials were accompanied by audio-recordings of the presented information.

The PFI included smoking-specific personalized feedback on motivation to change smoking behavior, money spent on cigarettes, carbon monoxide rating, and tactics the

participant used previously to change their smoking as well as general psychoeducation on the negative impact of smoking on health, chemical composition of cigarettes, benefits of quitting, and effective strategies to change smoking behavior. The PFI also included affect-specific personalized feedback on the participant's AS score and normative comparative information, and the relation between the participant's AS score and smoking. Psychoeducation was presented to define AS, its bidirectional relation with smoking, and methods to manage AS. In particular, the PFI included videos that described and presented two interoceptive exercises (i.e., straw breathing and hyperventilation). Participants practiced these exercises as part of the PFI.

Interview Outline

Semi-structured interviews were conducted using an outline to solicit information from participants. Three main topic areas were covered: 1) the general presentation of the PFI; 2) thoughts on smoking information; and 3) thoughts on stress information. The semi-structured interview outline was developed to engage participants and collect qualitative data to guide refinement of the PFI. The outline was informed by motivational interviewing, relied on open-ended questions, and provided opportunities for interviewers to flexibly engage with participants while collecting data. Sample items included: "What were some things that we didn't include in the feedback that you feel would motivate you to reassess your smoking habits" and "What are some things we can do to make the stress portion clearer or easier to understand?". Interviewers were instructed to use the outline as a rubric for the interview, but were not required to follow the guide verbatim. This approach is consistent with the fundamentals of conducting qualitative interviews.[75]

Measures

Demographics Questionnaire. Demographic information collected included sex, age, race, and educational level. Items were used to describe the sample.

Smoking History Questionnaire (SHQ). The SHQ was used to assess smoking rate, years of daily smoking, and other characteristics.[76] Smoking rate was obtained from the question, "Since you started regular daily smoking, what is the average number of cigarettes you smoked per day?" Furthermore, years as a daily smoker was assessed by the question, "For how many years, altogether, have you been a regular daily smoker?".

Fagerström Test for Cigarette Dependence (FTCD). The FTCD is a 6-item scale that assesses gradations in tobacco dependence.[71,77] Scores range from 0-10, with higher scores reflecting high levels of physiological dependence on cigarettes. The FTCD has adequate internal consistency, positive relations with key smoking variables (e.g., saliva cotinine), and high test-retest reliability.[71,78] In the current study, the FTCD total score was used to characterize tobacco dependence across the sample.

Satisfaction Ratings. The Session Evaluation asked participants to rate their overall impression of the PFI (1 = very negative; 5 = very positive), whether they would recommend it (1 = no, definitely not; 5 = yes, definitely), how informative it was (1 = not informative at all; 5 = very informative), how interesting it was (1 = not interesting at all; 5 = very interesting), and how helpful it was (1 = not helpful at all; 5 = very helpful). Items were assessed post-intervention and evaluated separately.

Data Analytic Strategy

A qualitative and quantitative approach was employed to illicit feedback for how to improve the PFI. Qualitative data analysis were guided by the Systematic and Reflexive Interviewing and Reporting (SRIR) method.[79] The interviewer and lead author listened to each audio-recording individually and outlined important topic areas discussed during the interview. The interviewer and lead author then engaged in a reflexive dialogue session to discuss their impressions of the interview and emerging topic areas. Audio-recordings were

played during the reflexive dialogue session and notes written during the interview were reviewed. Themes that emerged across several interviewers were discussed and documented. Audio-transcriptions were not used as part of the qualitative data analysis because of the widely recognized potential for loss of meaning and interpretation bias inherent to transcribed audio-files.[80] Next, PFI satisfaction ratings were evaluated. Finally, the PFI was refined based on data collected during the pilot study and expert opinion.

RESULTS

Interview Analysis. Of the nine participants eligible for the present study, audio-recordings were obtained for seven, as a technical problem precluded audio-recording of two interviews. Thus, seven were discussed following review by at least two research staff and two were discussed primarily from the perspective of the interviewer and relied on interviewer notes.

Regarding the general presentation, participants reported that the PFI length and graphics were appropriate and the information was easy to follow. Participants stated that the audio-recordings were monotone and could benefit from more inflections to improve attention and engagement. Specific to the smoking component, participants consistently indicated that psychoeducation on the chemical composition of cigarettes was new information. Participants suggested incorporating additional information on the chemicals in cigarettes and the negative effects of smoking on health. Most participants reported no previous knowledge of the connection between smoking and stress. Because the information on AS and its relation to smoking was novel to most participants, little feedback was provided for how to improve this portion of the PFI. Participants stated that the information

was clearly presented and that the exercises were modeled clearly, although some participants reported that they were unlikely to use the exercises.

Satisfaction Ratings. Five participants completed the post-intervention online survey. All (100%) participants rated the overall impression of the PFI as 'very positive'; reported 'yes' or 'yes, definitely' that they would recommend the single-intervention session to a friend; indicated that the information was 'informative' or 'very informative'; found the PFI 'somewhat interesting' or 'very interesting'; and reported that the PFI was 'pretty helpful' or 'very helpful.'

PFI Refinement. Guided by collected qualitative and quantitative data, the PFI was refined. Specifically, additional information was integrated into the PFI that focused on the toxins in cigarettes and personalized feedback on the effect of smoking on life expectancy. Many of the audio-recordings that accompanied the PFI were also re-scripted and rerecorded. Expert scientists in PFI development, and smoking and mood treatment then reviewed the refined PFI. Based on their feedback, wording was simplified to an 8th grade reading level and normative comparisons on smoking attitudes were integrated. The refined PFI was then strategically tested in the randomized controlled trial.

RANDOMIZED CONTROLLED TRIAL

METHOD

Participants

The sample consisted of 95 (63.2% male; M_{age} = 46.20 years, SD = 10.90) adult daily cigarette smokers recruited from the community to participate in a randomized controlled trial comparing the efficacy of two computerized-delivered smoking treatments. Eligibility included: (1) not being pregnant (self-reported); (2) being between 18-65 years of age; (3)

report daily smoking (average ≥5 cigarettes per day for at least 1 year) that was biochemically confirmed via Carbon Monoxide [CO] (analysis of at least 5 parts per million[81]); (4) not presently engaged in a quit attempt or mental health treatment; (5) not currently using non-cigarette tobacco products or illicit substances regularly (defined as 3 or more times per week); (6) cognitive capacity to provide written, informed consent; and (7) computer literate. Exclusion criteria included: (1) current treatment for an alcohol/drug problem including smoking cessation; (2) current psychiatric treatment; (3) active suicidality (i.e., suicidal ideation, intent, or plan[82]); (4) psychosis; and (5) not being fluent in English. See Figure 1 for consort.

The sample was primarily African American (76.8%) with 15.2% Caucasian, 4.6% Other (e.g. biracial), 2.0% Native American/Alaskan Native, 0.7% Asian, and 0.7% Native Hawaiian or other Pacific Islander. Almost one-tenth (8.6%) of the sample was Hispanic or Latino. Regarding level of education, 0.7% completed less than 7 years of school, 4.0% completed junior high school, 9.9% completed some high school, 31.8% graduated high school, 35.8% completed some college, 9.9% graduated college, and 7.9% completed graduate school. Participants smoked an average of 15.90 (*SD* = 18.96) cigarettes per day for an average of 22.61 years (*SD* = 11.63) and evinced a moderate level of cigarette dependence (*M* = 4.99, *SD* = 2.06[71]).

Study Design and Procedures

This randomized controlled trial employed a longitudinal experimental design and involved four appointments: (a) phone-screener (pre-screener); (b) baseline appointment consisting of a pre-intervention assessment (eligibility), one-session computer-delivered intervention (PFI versus smoking information control), and a post-intervention assessment;

(c) 2-week post-baseline follow-up; and (d) 4-week post-baseline follow-up. Participants were recruited via community postings, newspaper advertising, and online media sites that targeted general smokers. Interested individuals called the laboratory and completed the initial phone-screener that lasted for approximately 10 minutes. Callers eligible at the phone-screener and willing to participate were scheduled for an in-person baseline appointment, wherein full eligibility was assessed.

Upon arrival at the baseline appointment, a trained researcher obtained informed consent from each participant, administered a 30-minute interview assessment wherein research staff assessed for eligibility and collected an expired CO sample to biochemically verify smoking. Each participant then completed a 90-minute pre-intervention online survey in a private room. Self-report measures were administered at the pre-intervention assessment. Next, the trained research assistant evaluated eligibility criteria. Ineligible participants were provided the National Cancer Institute's Clearing the Air (CTA) pamphlet and a list of things to do instead of smoking handout, compensated $10, and dismissed. Participants who met eligibility criteria were randomly assigned to either the (a) PFI or (b) smoking information control. Participants were instructed to wear headphones and follow the computer prompts. After completing the assigned intervention, participants immediately completed a 15-minute post-intervention assessment and received a printed copy of the intervention materials and a list of things to do instead of smoking handout. Participants were then scheduled for their in-person, 2-week and 4-week follow-up appointments. The follow-up appointments consisted of a researcher-administered interview assessment and an online survey. These appointments were approximately 30 minutes each. Participants were compensated $30 for completing the baseline appointment, $30 for completing the 2-week follow-up, and $40 for completing the

4-week follow-up. Participants were also provided with bus fare, if needed, for each appointment they attended.

Interventions

Personalized Feedback Intervention. The refined PFI described in the Pilot Study above was tested in the randomized controlled trial. Briefly, the refined PFI included personalized feedback on motivation to change smoking behavior, money spent on cigarettes, carbon monoxide rating, tactics used previously to change smoking, normative comparisons of smoking attitudes, and the effect of smoking on life expectancy as well as general psychoeducation on the negative impact of smoking on health, chemical composition of cigarettes, benefits of quitting, and effective strategies to change smoking behavior. The PFI also provided personalized feedback on each participant's AS score and normative comparative information, and the relation between the participant's AS score and smoking, in addition to psychoeducation to define AS, its bidirectional relation with smoking, and methods to manage AS. Similar to the pilot study, the PFI included a race and sex matched digital avatar, was interactive, and audio-recorded. Expired CO reading, data from the SHQ, select items about motivation to quit, items regarding perceived smoking norms, and data from the ASI-3 was piped into the PFI. The PFI required 60 minutes to complete.

Smoking Information Control. Participants in the smoking information control group received the same computer-delivered smoking information content as the PFI condition, including general smoking cessation strategies presented in the National Cancer Institute's Clearing the Air pamphlet. The smoking information control also contained additional information on second hand smoke, additional strategies to quit, and information on smoking and others. No personalized feedback or AS information was presented as part of

the control. Similar to the PFI, the control was interactive and audio-recorded. We have successfully used a similar control condition in intervention trials.[74] The use of this control permits evaluation of personalized feedback for smoking/mood compared to non-personalized smoking intervention. The control required 20 minutes to complete.

Measures

Demographics Questionnaire. Demographic information collected included sex, age, race, and educational level. Items were used to describe the sample.

Smoking History Questionnaire (SHQ). The SHQ was used to assess smoking rate, years of daily smoking, and other characteristics.[76] Smoking rate was obtained from the question, "Since you started regular daily smoking, what is the average number of cigarettes you smoked per day?" Furthermore, years as a daily smoker was assessed by the question, "For how many years, altogether, have you been a regular daily smoker?".

Fagerström Test for Cigarette Dependence (FTCD). The FTCD is a 6-item scale that assesses gradations in tobacco dependence.[71,77] Scores range from 0-10, with higher scores reflecting high levels of physiological dependence on cigarettes. The FTCD has adequate internal consistency, positive relations with key smoking variables (e.g., saliva cotinine), and high test-retest reliability.[71,78] In the current study, the FTCD total score was used to characterize tobacco dependence across the sample.

Anxiety Sensitivity Index-3 (ASI-3). The ASI-3 is an 18-item self-report measure of sensitivity to and fear of the potential negative consequences of anxiety-related symptoms and sensations.[83] Respondents are asked to indicate, on a 5-point Likert-type scale (0 = very little to 4 = very much), the degree to which they are concerned about these possible negative consequences (possible range 0–72). The ASI-3, derived in part from the original ASI,[84] has sound psychometric properties, including excellent internal consistency, predictive validity, and reliability among treatment-seeking smokers.[85] The ASI-3 was administered at the pre- and post-intervention assessment, and at each follow-up. The ASI-3 total score demonstrated

excellent internal consistency at each administration (pre-intervention: α = .95; post-intervention: α = .95; 2-week follow-up: α = .95 4-week follow-up: α = .95).

Mood and Anxiety Symptom Questionnaire–30 (MASQ-D30). The MASQ-D30[86] is a 30-item self-report measure of emotional symptoms based upon the tripartite model of anxiety and depression. The MASQ-D30, derived from the original MASQ,[87] assesses general distress, anxiety and depression experienced over the past week. This scale is rated on a 5-point Likert scale ranging from 1 (not at all) to 5 (extremely) and includes the following subscales: general distress (e.g., "I felt confused"), anxious arousal (e.g., "I was startled easily"), and anhedonic depression (e.g., "I felt really happy" [all items reverse coded]). All subscales were used for the present study and administered at the pre- and post-intervention assessment and at each follow-up. MASQ-D30 subscales demonstrated good to excellent internal constancy at each assessment general distress: α range = .89-.91; anxious arousal: α range = .87-.93; anhedonic depression: α range = .86-.91).

Self-Help Scale (SHS). The SHS assesses willingness to engage in activities to cope with negative mood states.[69,88] Participants indicate the degree to which they would be willing to try each of 10 strategies to cope with feeling of depression or anxiety (0 = definitely not to 4 = extremely willing).[88] Sample items include "do something that might be pleasurable or satisfying" or "try new ways of relating to others." A mean SHS score serves as an indicator of willingness to use coping skills. The SHS was administered at the pre- and post-intervention assessment, and at each follow-up. The SHS demonstrated excellent internal consistency at each administration (pre-intervention: α = .92; post-intervention: α = .95; 2-week follow-up: α = .94; 4-week follow-up: α = .96).

Quit Attempts and Smoking Reduction. Primary outcomes of interest included quit attempts ("Since the last time you were here, have you made a quit attempt?" [0 = no; 1 = yes]; "How many times did you quit for at least 24 hours since your last appointment here?") assessed at 2- and 4-week follow-ups, and substantial (defined as ≥50% reduction; coded: 0 = no; 1 = yes) and quantitative change in cigarettes per day from the week prior to baseline

compared to (a) the week following baseline and (b) the week prior to 4-week follow-up.[89,90] Self-reported cigarettes per day was assessed in-person at baseline, and 2- and 4-week follow-ups using the Timeline Follow-Back (TLFB[91,92]) procedure. The assessment has demonstrated good reliability and validity with biochemical indices of smoking.[93]

Data Analytic Strategy

Baseline demographics, smoking, affective, and willingness to use coping strategies were compared across groups. Variables that differed across PFI and control participants were included as covariates in analyses. Logistic regression models were conducted to evaluate the effect of treatment (0 = control, 1 = PFI) on reported quit attempt (0 = no; 1 = yes) at 2- and 4-week follow-ups, adjusting for sex. Multiple regression analysis was conducted to examine differences in 24-hour quit attempts across condition at each follow-up, controlling for sex. Logistic models were employed to evaluate condition on for substantial change (≥50% reduction) in cigarettes per day for the week post-baseline and the week pre 4-week follow-up (0 = no; 1 = yes), controlling for sex. To evaluate quantitative change, total cigarettes consumed for the week post-baseline and week pre 4-week follow-up were subtracted from total cigarettes consumed for the week pre-baseline. The quantitative change was then regressed on treatment and sex in a multiple linear regression analysis. Subsequently, AS was evaluated as a moderator of smoking outcomes. Analyses were conducted separately for post-baseline and pre 4-week follow-up. Missing data (0.4% at post-baseline and 4.2% at pre 4-week follow-up) were handled using last observation carried forward.[94] Indices of normality were within the range of normal for the difference in cigarette consumption from week pre-baseline to week post-baseline (skewness = .64; kurtosis = 1.79). Difference in week pre-baseline to week pre 4-week cigarette consumption score was outside the range of normal (skewness = -.13; kurtosis = 3.80); visual inspection of data

identified one outlier. Identified outlier was replaced with next highest value, after which the variable was within the range of normal (skewness = .93; kurtosis = .87).

Latent growth curve (LGC) analysis was used to examine the impact of treatment (PFI vs control) on mood (AS and MASQ-D30 subscales) and willingness to use adaptive coping strategies (SHS) subscales growth from post-intervention assessment to 4-week follow-up (with analyses centered on post-intervention), controlling for baseline levels of the respective measure, sex, and baseline cigarette dependence[95]; continuous covariates were grand mean centered. Treatment condition (0 = control; 1 = PNF) was included as a predictor of the intercept and the slope parameters in the conditional model. All analyses were conducted in Mplus version 8[96] using robust maximum likelihood with the Yuan-Bentler (Y-B) scaled χ^2 index to correct for data nonnormality and missing data; one participant was missing data at all three time-points and was therefore excluded from unconditional LGC analyses. Overall model fit was assessed using the Y-B χ^2 value as well as additional χ^2-based fit indices, including the comparative fit index (CFI), root mean square error of approximation (RMSEA) with accompanying 90% confidence intervals (CIs), and the standardized root mean square residual (SRMR). A nonsignificant χ^2 value indicates good fit. CFI values greater than .90, RMSEA values below .08, and SRMR values below .08 suggest good fit.[97] RMSEA lower bound CIs below .05 suggest that good fit cannot be ruled out and upper bound CIs above .10 suggest that poor fit cannot be ruled out.

RESULTS

Screening, Randomization, and Attrition. Seven hundred and eighty-two callers completed the phone-screener. Of those callers, 259 were found eligible and scheduled for a baseline appointment. One hundred and fifty-four individuals attended the baseline

appointment and 151 completed the pre-intervention online assessment. Based on the interview and pre-intervention online assessment, 95 smokers were found eligible for the current study. See Figure 1 for consort. Participants eligible and ineligible for the larger study did not differ in race ($\chi[5] = 2.61$, $p = .76$), age ($t[149] = -1.13$, $p = 0.26$), education achievement ($t[149] = .05$, $p = 1.00$), or annual household income ($t[149] = .73$, $p = 0.323$). A significant association between sex and eligibility status emerged ($\chi[1] = 4{,}92$, $p = .03$), suggesting that fewer women screened were ineligible.

The final sample included 95 daily smokers, 49 (52%) were randomized to the PFI and 46 (48%) were randomized to the control. Participants randomized to each study condition did not differ in race ($\chi[4] = 5.00$, $p = 0.29$), age ($t[93] = -.42$, $p = 0.68$), education achievement ($t[93] = .62$, $p = 0.54$), or annual household income ($t[93] = .92$, $p = 0.36$) at baseline. A significant association between sex and treatment condition emerged ($\chi[1] = 9.01$, $p = .003$), suggesting that fewer women were randomized to the PFI condition. Regarding smoking variables, baseline cigarettes per day, years being a daily smoker, expired baseline CO, and cigarette dependence were equivalent across groups. Further, no differences emerged in affective variables or willingness to use coping skills across those assigned PFI versus control. Mean differences presented in Table 1.

Ninety-four participants provided post-intervention data (control: $n = 46$ [100%]; PFI: $n = 48$ [98.0%]), 87 (92%) provided 2-week follow-up data (control: $n = 39$ [84.8%]; PFI: $n = 48$ [98.0%]), and 88 (93%) provided 4-week follow-up data (control: $n = 43$ [93.5%]; PFI: $n = 45$ [91.8%]). Participants who missed either follow-up did not significantly differ from those who completed both appointments in terms of condition ($\chi[1] = 1.83$, $p = 0.18$), sex ($\chi[1] = 1.02$, $p = 0.31$), age ($t[93] = -1.52$, $p = 0.13$), race ($\chi[4] = .69$, $p = 0.95$), education

achievement (t[93] = .31, p = 0.76), or annual household income (t[93] = .65, p = 0.52). Baseline cigarettes per day, years being a daily smoker, baseline expired baseline CO, cigarette dependence were equivalent across groups (all p's > .05). Additionally, all affective vulnerability variables and willingness to use coping strategies were comparable across groups (all p's > .05).

Bivariate Correlations. Table 2 presents baseline bivariate correlations for study variables. Sex was significantly associated with anxious arousal (r = .22, p = .03). Cigarette dependence was significantly correlated with anxious arousal (r = .21, p = .04). ASI-3 was positively correlated with general distress (r = .60 p < .001) and anxious arousal (r = .54, p < .001). General distress and anxious arousal were positively correlated (r = .78, p < .001). Anhedonia and SHS negative correlated (r = -.45, p < .001).

Quit attempts at 2- and 4-Week Follow-Ups. Forty-two participants (48.3%) reported a quit attempt between baseline and 2-week follow-up. Logistic regression analysis revealed that condition was unrelated to reported engagement in a quit attempt at 2-week follow-up (OR = 1.05, 95% CI = .436–2.542, p = 0.91), with 41.3% of control participants and 46.9% of PFI participants reporting a quit attempt. At 4-week follow-up, 47 (53.4%) participants reported a quit attempt since their previous laboratory session. Logistic regression analysis revealed that condition was marginally related to quit attempt at 4-week follow-up (OR = 2.14, 95% CI = .878–5.19, p = 0.09), with 41.3% of control participants and 57.1% of PFI participants reporting a quit attempt. The joint consideration of quit attempt of 2- and 4-week follow-up revealed that significantly more participants in the PFI, relative to control, who did not report a quit attempt at the 2-week follow-up reported a quit attempt at the 4-week follow-up (PFI: n = 13 [28.9%]; control: n = 4 [10.5%]; p < .05). In separate

models, AS did not significantly moderate treatment on reported quit attempt at the 2-week (*OR* = 1.02, 95% CI = .968–1.069, *p* = 0.50) or 4-week (*OR* = 1.00, 95% CI = .972–1.022, *p* = 0.81) follow-up.

Eleven participants (11.6%) reported successfully quitting for at least 24 hours since their previous study appointment at the 2-week follow-up (control: 17.4%; PFI: 10.2%). The average number of quit attempts was 3.09 (*SD* = 3.30), and ranged from 1-12. Twenty-two participants (23.2%) reported successfully quitting for at least 24 hours since their previous study appointment at the 4-week follow-up (control: 19.6%; PFI: 26.5%). The average number of quit attempts was 3.77 (SD = 3.78), and ranged from 1-12. The low number of participants who reported successfully quitting for 24 hours was statistically underpowered to test the effect of condition on number of quit attempts; therefore, multiple regression analyses were not conducted.

Cigarette Reduction at 1-Week Post-Baseline. Nineteen participants (21.8%; control: 17.4%, PFI: 22.4%) evinced substantial reduction in cigarette consumption from 1-week pre- and post-baseline. In adjusted analysis of substantial reduction, controlling for sex, condition was not associated with likelihood of reduction (*OR* = 1.02, 95% CI = .352–2.98, *p* = 0.97). Regarding quantitative cigarette reduction, participants reduced an average of 24.7 (*SD* = 36.47) cigarettes from 1-week pre-baseline to 1-week post-baseline (control: *M* = 28.90, *SD* = 39.72; PFI: *M* = 23.77, *SD* = 34.01). Condition and sex did not explain significant variance in weekly cigarette consumption change from pre- to post-baseline (R^2 =.004, $F(2,84) = .18$, $p = .84$).

The main effect of AS and its interaction with condition were included as predictors of outcomes in separate post-hoc test of moderation. Sex remained a covariate in all models.

In a logistic model for substantial reduction, the interaction between AS and condition was non-significant (*OR* = 1.04, 95% CI = .971–1.103, $p = 0.29$). The final model for cigarette reduction was also non-significant ($R^2 = .02$, $F(4,82) = .34$, $p = .85$).

Cigarette Reduction at 1-Week Pre 4-Week. Twenty-five participants (28.4%; control: 28.3%, PFI: 24.5%) evinced substantial reduction in cigarette consumption from 1-week pre-baseline and 1-week pre 4-week follow-up. In sex-adjusted analysis of substantial reduction, condition was not associated with likelihood of reduction (*OR* = 0.65, 95% CI = .241–1.733, $p = 0.39$). Regarding quantitative cigarette reduction, participants reduced an average of 27.3 (*SD* = 36.88) cigarettes from 1-week pre-baseline to 1-week pre 4-week follow-up (control: *M* = 29.19, *SD* = 40.16; PFI: *M* = 25.58, *SD* = 33.82). Condition and sex did not explain significant variance in weekly cigarette consumption change from pre- to post-baseline ($R^2 = .013$, $F(2,85) = .55$, $p = .58$).

The main effect of AS and its interaction with condition were included as predictors of outcomes in separate post-hoc tests of moderation. Sex remained a covariate in all models. In a logistic model for substantial reduction, the interaction between AS and condition approached statistical significance (*OR* = 1.06, 95% CI = .994–1.129, $p = 0.08$). The final model for cigarette reduction was marginally significant ($R^2 = .09$, $F(4,83) = 2.05$, $p = .10$). The interaction between baseline AS and condition emerged as a significant predictor ($\beta = .55$, $p = .01$). Specifically, higher baseline AS predicted less reduction for participants in the control condition ($b = -.69$, $SE = .34$, $p = .04$); AS was unrelated to cigarette reduction among PFI participants ($p = .11$).

Latent Growth Curve Model for ASI-3. On average, participants reported an ASI-3 score of 21.01 (*SD* = 17.93) at post-intervention, 23.93 (*SD* = 18.18) at 2-week follow-up,

and 21.24 (*SD* = 16.73) at 4-week follow-up; see Table 3 for average scores by condition across time. The unconditional LGC model for ASI-3 from post-intervention to 4-week follow-up provided adequate fit to the data (Y-B χ^2 = 7.22, *df* = 2, *p* = .03, RMSEA = .17, 90% CI [.05, .31], CFI = .96, SRMR = .03). The intercept parameter was significant (β = 1.37, *p* < .001); the slope parameter was non-significant (β = .80, *p* = .72). The conditional model including treatment condition, sex, cigarette dependence, and baseline ASI-3 predicting the intercept and slope of ASI-3 scores from post-intervention to 4-week follow-up provided adequate fit to the data (Y-B χ^2 = 8.93, *df* = 9, *p* = .44, RMSEA = .00, 90% CI [.00, .12], CFI = 1.00, SRMR = .03). The intercept effect was significant (β = 1.25, *p* < .001) as were the effects of cigarette dependence and baseline ASI-3 on the intercept (β = .11, *p* = .04 and β = .91, *p* < .001). The effect of baseline ASI-3 on the slope was also significant (β = -.26, *p* = .05). Thus, treatment did not influence AS over time. No other parameter estimates were significant. Model parameters are provided in Table 4.

Latent Growth Curve Model for MASQ-D30: General Distress. On average, participants reported a general distress score of 18.18 (*SD* = 7.84) at post-intervention, 19.98 (*SD* = 8.47) at 2-week follow-up, and 19.47 (*SD* = 7.78) at 4-week follow-up; see Table 3 for average scores by condition across time. The unconditional LGC model for general distress from post-intervention to 4-week follow-up provided poor fit to the data (Y-B χ^2 = 11.15, *df* = 2, *p* = .004, RMSEA = .22, 90% CI [.11, .35], CFI = .91, SRMR = .08). The intercept parameter was significant (β = 2.85, *p* < .001); the slope parameter was non-significant (β = .21, *p* = .26). The conditional model including treatment condition, sex, cigarette dependence, and baseline general distress predicting the intercept and slope of general distress scores from post-intervention to 4-week follow-up provided adequate fit to the data

(Y-B χ^2 = 16.73, *df* = 9, *p* = .05, RMSEA = .10, 90% CI [.00, .17], CFI = .97, SRMR = .06). The intercept effect was significant (β = 2.65, *p* < .001) as were the effects of baseline general distress on the intercept (β = .94, *p* < .001) and slope (β = -.31, *p* = .02); no other parameter estimates were significant. Thus, treatment did not influence general distress over time. Model parameters are provided in Table 5.

Latent Growth Curve Model for MASQ-D30: Anxious Arousal. On average, participants reported an anxious arousal score of 16.48 (*SD* = 7.99) at post-intervention, 16.66 (*SD* = 6.83) at 2-week follow-up, and 16.55 (*SD* = 7.47) at 4-week follow-up; see Table 3 for average scores by condition across time. The unconditional LGC model for anxious arousal from post-intervention to 4-week follow-up provided good fit to the data (Y-B χ^2 = .24, *df* = 2, *p* = .89, RMSEA = .00, 90% CI [.00, .10], CFI = 1.00, SRMR = .03). The intercept parameter was significant (β = 2.80, *p* < .001); the slope parameter was non-significant (β = .21, *p* = .68). The conditional model including treatment condition, sex, cigarette dependence, and baseline anxious arousal predicting the intercept and slope of anxious arousal scores from post-intervention to 4-week follow-up provided adequate fit to the data (Y-B χ^2 = 12.82, *df* = 9, *p* = .17, RMSEA = .07, 90% CI [.00, .14], CFI = .97, SRMR = .07). The intercept effect was significant (β = 2.67, *p* < .001) as were the treatment effect on the slope (β = -.32, *p* = .04) and the effect of baseline anxious arousal on the intercept (β = .93, *p* < .001). Thus, treatment had an effect on anxious arousal over time. No other parameter estimates were significant. Model parameters are provided in Table 6.

Latent Growth Curve Model for MASQ-D30: Anhedonic Depression. On average, participants reported an anhedonic score of 33.22 (*SD* = 8.46) at post-intervention, 31.21 (*SD* = 8.81) at 2-week follow-up, and 32.20 (*SD* = 9.13) at 4-week follow-up; see

Table 3 for average scores by condition across time. The unconditional LGC model for anhedonic depression from post-intervention to 4-week follow-up provided poor fit to the data (Y-B $\chi^2 = 9.49$, $df = 2$, $p = .009$, RMSEA = .20, 90% CI [.09, .34], CFI = .93, SRMR = .12. The intercept parameter was significant ($\beta = 4.63$, $p < .001$); the slope parameter was non-significant ($\beta = -.29$, $p = .13$). The condition model including treatment condition, sex, cigarette dependence, and baseline anhedonic depression predicting the intercept and slope of anhedonic depression scores from post-intervention to 4-week follow-up provided adequate fit to the data (Y-B $\chi^2 = 9.66$, $df = 9$, $p = .38$, RMSEA = .03, 90% CI [.00, .12], CFI = 1.00, SRMR = .04). The intercept effect was statistically significant ($\beta = 4.40$, $p < .001$) as was the effect of baseline anhedonic depression on the intercept ($\beta = .84$, $p < .001$); the effects of sex on the intercept and baseline anhedonic depression on the slope effect were marginally significant ($\beta = .14$, $p = .08$ and $\beta = -.18$, $p = .07$, respectively). No other parameter estimates were significant. Thus, treatment did not have effect on anhedonia over time. Model parameters are provided in Table 7.

Latent Growth Curve Model for SHS. On average, participants reported a SHS score of 2.57 (*SD* = 1.01) at post-intervention, 2.52 (*SD* = 0.93) at 2-week follow-up, and 2.57 (*SD* = 1.07) at 4-week follow-up; see Table 3 for average scores by condition across time. The unconditional LGC model for SHS from post-intervention to 4-week follow-up provided good fit to the data (Y-B $\chi^2 = 2.67$, $df = 2$, $p = .26$, RMSEA = .06, 90% CI [.00, .22], CFI = .98, SRMR = .07). The intercept parameter was significant ($\beta = 2.55$, $p < .001$); the slope parameter was non-significant ($\beta = -.003$, $p = .95$). The condition model including treatment condition, sex, cigarette dependence, and baseline SHS predicting the intercept and slope of SHS scores from post-intervention to 4-week follow-up provided good fit to the data

(Y-B $\chi^2 = 6.73$, $df = 9$, $p = .66$, RMSEA = .00, 90% CI [.00, .09], CFI = 1.00, SRMR = .05). The intercept effect was statistically significant ($\beta = 3.21$, $p < .001$) as were the effects of cigarette dependence and baseline SHS on intercept (FTCD: $\beta = .18$, $p = .03$; SHS: $\beta = .73$, $p < .001$); no other parameter estimates were significant. Thus, treatment did not have effect on SHS scores over time. Model parameters are provided in Table 8.

DISCUSSION

The current study developed and evaluated the efficacy of a novel, brief, computer-delivered personalized feedback intervention (PFI) for AS and smoking relative to computer-delivered standard smoking treatment (control). Evaluative outcomes of efficacy included quit attempts and smoking reduction assessed at 2- and 4-week follow-up assessments, and trajectories of affective vulnerability from post-intervention to 4-week follow-up. Statistical interpretation of findings supported equivalence of PFI and control. However, nuanced patterns of quit attempts and smoking reduction over time suggested that each treatment may uniquely promote increased engagement of smoking-specific behavior change such that PFI may promote quit attempts and control may promote reduction. Thus, despite non-significant findings, descriptive patterns suggested that the effect of PFI and control on smoking outcomes manifests differently over time, with PFI promoting quit attempt engagement and control promoting smoking reduction. Additionally, results indicated that AS moderates the effect of treatment on change in cigarettes smoked per week from the week pre-baseline to the week pre 4-week follow-up.

Extensive literature highlights the importance of continued, repeated quit attempts for smokers to achieve abstinence, with many smokers requiring 30 or more quit attempts to successfully quit.[7] Based on the present data, brief, computer-delivered, interactive smoking

treatment can promote self-defined quit attempts. Specifically, nearly half (48.3%) of all participants at the 2-week follow-up and more than half (53.4%) of all participants at the 4-week follow-up self-reported engaging in a quit attempt. Treatment condition did not statistically predict quit attempts reported during the 2-week follow-up or the 4-week follow-up. The relation between treatment and quit attempts at the 4-week follow-up, however, approached significance, with more PFI participants reporting a quit attempt. Interestingly, the percentage of participants who engaged in a quit attempt from 2-week to 4-week follow-up increased by more than 10% among PFI participants, but did not change among control participants. As similar pattern emerged when quit attempt was defined in terms of achieving at least 24-hour abstinence.

Cigarette reduction is a preparatory behavior that can help smokers adjust to and prepare for withdrawal symptoms they may experience when engaging in a quit attempt.[10,98] As such, cigarette reduction is a potentially useful tool to develop skills and build self-efficacy to successfully quit, particularly if a smoker is in the early stages of quitting.[99] Within the current sample, a clinically significant number of participants reported reducing their cigarette consumption by at least 50% at the 1-week post-baseline (21.8%) and 1-week pre 4-week follow-up (28.4%) with reduction, on average, of more than a pack of cigarettes a week. Treatment did not significantly predict reduction in cigarette consumption. Thus, standard smoking treatment and enhanced, integrated treatment are equally effective at encouraging smoking reduction over a 4-week follow-up period.

Notably, in contrast to quit attempts, the percentage of control participants who reduced their smoking by at least half increased by more than 10% from post-baseline to pre 4-week follow-up. Single-session, standard, interactive smoking cessation treatment may,

therefore, encourage smoking reduction whereas integrated PFI smoking treatment encourages more practiced quit attempts. As one explanation for this observation, smokers who receive PFI may feel more equipped and prepared to manage withdrawal symptoms associated with nicotine deprivation because of the psychoeducation and interoceptive exercises related to AS. Conversely, smokers who receive only standard smoking information may be less confident in their ability to practice a quit attempt and therefore seek to reduce their use. Although both practiced quit attempts and smoking reduction can be helpful tools for smokers working toward cessation,[90,100] smoking reduction may lead to reduced smoking for prolonged periods. Considering that even light and intermediate smoking is associated with substantial health risks,[101] it may be more clinically appropriate to administer PFI with the intent of promoting quit attempts.

A key observation with both outcomes is that that prevalence of quit attempts and substantial cigarette reduction increased over time. Post-hoc inspection of change patterns suggested that, among participants who did not attempt to quit prior to the 2-week follow-up, PFI participants were more likely to engage in a quit attempt at 4-week follow-up relative to control. This finding is consistent with work that proposes a 'grace period' for smoking cessation.[102] For instance, individual participants may need additional time to assimilate treatment information before deciding to engage in behavior change. This may be particularly relevant for smokers in the early stages of quitting who are collecting information and trying new skills to assist with cessation.[10] Additionally, although the proportion of control participants who substantially reduced their smoking increased over time, the proportional increase did not differ from that observed in PFI (15.8% vs 8.9%, respectively). The relative proportion of participants who engaged in a quit attempt at any

point post-baseline (68.7%) compared to those who substantially reduced their cigarette consumption (34.9%) and the overall small sample size may have contributed to these results. Future work is warranted to examine these associations in a larger sample of general smokers.

Limited support emerged for AS as a moderator of treatment effects on smoking outcomes. Specifically, AS significantly moderated treatment effects on cigarette reduction from pre-baseline to pre 4-week follow-up; no other tests of moderation were significant. Post-hoc probing of the interaction form indicated that higher baseline AS predicated less reduction in cigarette consumption for participants in the control condition. AS did not relate to change in cigarette consumption for PFI participants. Control participants with higher AS may not have had the necessary skills to manage symptoms indicative of high AS in the context of quitting. Conversely, PFI participants may have internalized information provided during the single-session intervention and learned that these symptoms are not harmful. They may have been more prepared to cope with the negative internal sensations and therefore AS does not impact smoking reduction for these participants. This finding, however, should be interpreted with caution considering that the overall model was only marginally significant. Replication work is needed to confirm this result.

Regarding affective vulnerability factors, treatment exerted a statistically significant effect on the slope of anxious arousal. The effect suggested that PFI exhibited a decrease in self-reported physiological symptoms of anxiety over time relative to control. No other treatment effects emerged. The decrease in anxious arousal symptoms among PFI participant relative to control may have been a function of the interoceptive exercises presented during the treatment. Specifically, participants practiced hyperventilation and straw breathing. Both

exercises elicit uncomfortable physiological sensations, including lightheadedness, increased heart rate, and dizziness. The objective is to cue participants that these symptoms are not harmful. In this context, the exposure to the symptoms and sensations PFI participants experienced during their intervention may have alerted them to the benign nature of these sensations. Subsequently, these participants may not have noticed or been as attentive to these symptoms when they experienced them following the intervention. Considering that the other affective factors focus more on mood, thoughts, and behavior and the PFI focused on physical responses to stress/AS, material presented in the PFI may not have been as centrally related to the other psychological constructs.

Current data provide preliminary evidence for the utility of a PFI to encourage behavior change related to smoking and address physical manifestations of anxiety. The effects, however, were limited in magnitude. Nevertheless, the initial 'signal' observed in this small trial provides a strong rationale for continued work within this domain. Theoretically, within a well-powered randomized controlled trial, PFI may lead to smoking cessation through repeated quit attempts coupled with a scheduled quit date. Additionally, the heightened awareness of one's bodily sensations and psychoeducation on their function may be a key component that an integrated PFI can provide help to smokers to successfully quit. Indeed, this may be particularly important considering the impact of negative states on relapse.[16-20]

Clinically, findings support that a brief, single session treatment for smoking can facilitate quit attempts and smoking reduction. The present data suggest that PFI may promote quit attempts over time whereas standard smoking treatment may encourage smoking reduction. Given that smoking cessation is the optimal outcome for all smokers,[103]

PFI may be a more clinically useful tool to help reach this goal as it focuses on cessation engagement. Indeed, from the cessation induction perspective,[10,102] the current integrated treatment offers a unique method to tailor and deliver information to promote practiced quit attempts. Additionally, the current PFI has the potential to improve mental health outcomes. Ultimately, the current PFI offers a cost and time effective solution to current challenges with offering smoking cessation treatment. An important 'next step' in the clinical examination of this treatment is to evaluate its efficacy when implemented in a point-of-care setting, such as a primary care office.

Several limitations of the current investigation warrant comment. First, the PFI and control were both interactive and encouraged engagement. The degree to which this may influence findings cannot be parsed out based on the present data. Additional work is needed to elucidate the unique impact of interactive and engagement elements on the efficacy of the current treatment. Similarly, the current study compared an integrated, personalized PFI to a generic, smoking-only comparison group. Therefore, the PFI differed from the control in terms of personalization and overall content. Similar to interactive elements of the current treatments, current data do not permit disentangling the effect of personalization relative to AS information. A dismantling study would be particularly useful to identify key elements that promote greatest change. Second, the current study relied on retrospective recall to capture quit attempts. Although this approach offers a 'first glance' into quit behavior following a brief intervention, more sophisticated assessment methods, including ecological momentary assessment, may provide more accurate data regarding quit attempts. Third, few participants were able to successfully quit for 24-hours during the study period. The low rate of successful quitting precluded analyses of quit attempt frequency. Future work may want to

emphasize the importance of 24-hour quit attempt to encourage more participation. Interestingly, significantly more participants reported a self-defined ‘quit attempt.’ How participants defined a ‘quit attempt’ was not assessed. Continued research is needed to understand how smokers self-define a quit attempt and factors related to various gradients of defined attempts. Fourth, the sample was relatively small and followed over a short time period. Therefore, select tests may have been unpowered to detect effects. Additionally, long-term impact of the current treatment on smoking behavior remains unknown. Replication studies that enroll a larger sample of smokers who are followed over longer periods of time are warranted. Fifth, time to complete varied across conditions. Future work should examine the observed patterns across treatments matched on time to control for the potential confound of time and effort engagement.

Overall, the present investigation provides descriptive evidence, though not statistical, for a single-session, computer-delivered PFI to effect smoking behavior related to early stages of quitting, including quit attempts. Moreover, data indicate that the rate of engaging in quit attempts may increase over a 4-week post-intervention trial. Furthermore, PFI exerts a significant effect on physical symptoms of anxiety post-treatment, which may serve a central function to assist with quitting smoking. Future work may benefit from exploring possible mechanisms that underlie observed pattern and should explore the long-term effects of the tested treatments.

REFERENCES

1. Organization WH, Control RfIT. *WHO report on the global tobacco epidemic, 2008: the MPOWER package.* World Health Organization; 2008.
2. Health UDo, Services H. The health consequences of smoking—50 years of progress: a report of the Surgeon General. *Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.* 2014;17.
3. Babb S. Quitting smoking among adults—United States, 2000–2015. *MMWR Morbidity and mortality weekly report.* 2017;65.
4. Jamal A, Phillips E, Gentzke AS, et al. Current cigarette smoking among adults—United States, 2016. *Morbidity and Mortality Weekly Report.* 2018;67(2):53.
5. Hughes JR, Keely J, Naud S. Shape of the relapse curve and long-term abstinence among untreated smokers. *Addiction.* 2004;99(1):29-38.
6. Jones J. Smoking habits stable; most would like to quit. *Gallup News Service.* 2006.
7. Chaiton M, Diemert L, Cohen JE, et al. Estimating the number of quit attempts it takes to quit smoking successfully in a longitudinal cohort of smokers. *BMJ open.* 2016;6(6):e011045.
8. Prevention CfDCa. Quitting smoking. 2014; http://www.cdc.gov/tobacco/data_statistics/fact_sheets/cessation/quitting/index.htm?utm_source=feedburner&utm_medium=feed&utm_campaign=Feed%3A+cdc%2FGEla+(CDC+-+Smoking+and+Tobacco+Use+-+Main+Feed). Accessed October 17, 2014.

9. Prochaska JO, DiClemente CC. Stages and processes of self-change of smoking: toward an integrative model of change. *Journal of consulting and clinical psychology.* 1983;51(3):390.

10. Baker TB, Mermelstein R, Collins LM, et al. New methods for tobacco dependence treatment research. *Annals of Behavioral Medicine.* 2011;41(2):192-207.

11. DiClemente CC, Prochaska JO, Fairhurst SK, Velicer WF, Velasquez MM, Rossi JS. The process of smoking cessation: an analysis of precontemplation, contemplation, and preparation stages of change. *Journal of consulting and clinical psychology.* 1991;59(2):295.

12. Bailey JA, Hill KG, Meacham MC, Young SE, Hawkins JD. Strategies for characterizing complex phenotypes and environments: General and specific family environmental predictors of young adult tobacco dependence, alcohol use disorder, and co-occurring problems. *Drug and alcohol dependence.* 2011;118(2-3):444-451.

13. Marlatt GA, Curry S, Gordon J. A longitudinal analysis of unaided smoking cessation. *Journal of consulting and clinical psychology.* 1988;56(5):715.

14. Burns DM, Anderson C, Johnson M, et al. Cessation and cessation measures among adult daily smokers: National and state-specific data. *Population-Based Smoking Cessation: What Works Smoking and Tobacco Control Monograph No.* 2000;12:25-97.

15. Watson D, Clark LA, Tellegen A. Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of personality and social psychology.* 1988;54(6):1063.

16. Shiffman S, Waters AJ. Negative affect and smoking lapses: a prospective analysis. *Journal of consulting and clinical psychology.* 2004;72(2):192.

17. Conklin CA, Perkins KA. Subjective and reinforcing effects of smoking during negative mood induction. *Journal of abnormal psychology.* 2005;114(1):153.

18. Perkins KA, Ciccocioppo M, Conklin CA, Milanak ME, Grottenthaler A, Sayette MA. Mood influences on acute smoking responses are independent of nicotine intake and dose expectancy. *Journal of abnormal psychology.* 2008;117(1):79.

19. Buczkowski K, Marcinowicz L, Czachowski S, Piszczek E. Motivations toward smoking cessation, reasons for relapse, and modes of quitting: results from a qualitative study among former and current smokers. *Patient preference and adherence.* 2014;8:1353.

20. Kassel JD, Stroud LR, Paronis CA. Smoking, stress, and negative affect: correlation, causation, and context across stages of smoking. *Psychological bulletin.* 2003;129(2):270.

21. Baker TB, Piper ME, McCarthy DE, Majeskie MR, Fiore MC. Addiction motivation reformulated: an affective processing model of negative reinforcement. *Psychological review.* 2004;111(1):33.

22. Piasecki TM, Kenford SL, Smith SS, Fiore MC, Baker TB. Listening to nicotine: Negative affect and the smoking withdrawal conundrum. *Psychological Science.* 1997;8(3):184-189.

23. Perkins KA, Karelitz JL, Giedgowd GE, Conklin CA, Sayette MA. Differences in negative mood-induced smoking reinforcement due to distress tolerance, anxiety sensitivity, and depression history. *Psychopharmacology.* 2010;210(1):25-34.

24. Leventhal AM, Zvolensky MJ. Anxiety, depression, and cigarette smoking: A transdiagnostic vulnerability framework to understanding emotion–smoking comorbidity. *Psychological bulletin.* 2015;141(1):176.

25. Taylor S. *Anxiety sensitivity: Theory, research, and treatment of the fear of anxiety.* Mahwah, NJ, US: Lawrence Erlbaum Associates Publishers; 1999.

26. Zinbarg RE, Barlow DH, Brown TA. Hierarchical structure and general factor saturation of the Anxiety Sensitivity Index: Evidence and implications. *Psychological assessment.* 1997;9(3):277.

27. Reiss S. Expectancy model of fear, anxiety, and panic. *Clinical psychology review.* 1991;11(2):141-153.

28. Zvolensky MJ, Yartz AR, Gregor K, Gonzalez A, Bernstein A. Interoceptive exposure-based cessation intervention for smokers high in anxiety sensitivity: A case series. *Journal of Cognitive Psychotherapy.* 2008;22(4):346-365.

29. Gregor KL, Zvolensky MJ, McLeish AC, Bernstein A, Morissette S. Anxiety sensitivity and perceived control over anxiety-related events: Associations with smoking outcome expectancies and perceived cessation barriers among daily smokers. *Nicotine & Tobacco Research.* 2008;10(4):627-635.

30. Johnson KA, Farris SG, Schmidt NB, Zvolensky MJ. Anxiety sensitivity and cognitive-based smoking processes: Testing the mediating role of emotion dysregulation among treatment-seeking daily smokers. *Journal of Addictive Diseases.* 2012;31(2):143-157.

31. Leyro TM, Zvolensky MJ, Vujanovic AA, Bernstein A. Anxiety sensitivity and smoking motives and outcome expectancies among adult daily smokers: Replication and extension. *Nicotine & Tobacco Research.* 2008;10(6):985-994.

32. Gonzalez A, Zvolensky MJ, Vujanovic AA, Leyro TM, Marshall EC. An evaluation of anxiety sensitivity, emotional dysregulation, and negative affectivity among daily cigarette smokers: Relation to smoking motives and barriers to quitting. *Journal of Psychiatric Research.* 2008;43(2):138-147.

33. Farris SG, Leventhal AM, Schmidt NB, Zvolensky MJ. Anxiety sensitivity and pre-cessation smoking processes: testing the independent and combined mediating effects of negative affect-reduction expectancies and motives. *Journal of studies on alcohol and drugs.* 2015;76(2):317-325.

34. Battista SR, Stewart SH, Fulton HG, Steeves D, Darredeau C, Gavric D. A further investigation of the relations of anxiety sensitivity to smoking motives. *Addictive behaviors.* 2008;33(11):1402-1408.

35. Zvolensky MJ, Vujanovic AA, Miller MOB, et al. Incremental validity of anxiety sensitivity in terms of motivation to quit, reasons for quitting, and barriers to quitting among community-recruited daily smokers. *Nicotine & Tobacco Research.* 2007;9(9):965-975.

36. Langdon KJ, Farris SG, Hogan JB, Grover KW, Zvolensky MJ. Anxiety sensitivity in relation to quit day dropout among adult daily smokers recruited to participate in a self-guided cessation attempt. *Addictive behaviors.* 2016;58:12-15.

37. Zvolensky MJ, Farris SG, Schmidt NB, Smits JA. The role of smoking inflexibility/avoidance in the relation between anxiety sensitivity and tobacco use and

beliefs among treatment-seeking smokers. *Experimental and clinical psychopharmacology.* 2014;22(3):229-237.

38. Zvolensky MJ, Paulus DJ, Garey L, et al. Anxiety Sensitivity and Nicotine Replacement Therapy Side Effects: Examining the Role of Emotion Dysregulation Among Treatment-Seeking Smokers. *Journal of studies on alcohol and drugs.* 2017;78(6):877-883.

39. Johnson KA, Stewart S, Rosenfield D, Steeves D, Zvolensky MJ. Prospective evaluation of the effects of anxiety sensitivity and state anxiety in predicting acute nicotine withdrawal symptoms during smoking cessation. *Psychol Addict Behav.* 2012;26(2):289-297.

40. Langdon KJ, Leventhal AM, Stewart S, Rosenfield D, Steeves D, Zvolensky MJ. Anhedonia and anxiety sensitivity: Prospective relationships to nicotine withdrawal symptoms during smoking cessation. *J Stud Alcohol Drugs.* 2013;74(3):469-478.

41. Brown RA, Kahler CW, Zvolensky MJ, Lejuez C, Ramsey SE. Anxiety sensitivity: Relationship to negative affect smoking and smoking cessation in smokers with past major depressive disorder. *Addict Behav.* 2001;26(6):887-899.

42. Assayag Y, Bernstein A, Zvolensky MJ, Steeves D, Stewart SS. The nature and role of change in anxiety sensitivity during NRT-aided cognitive-behavioral smoking cessation treatment. *Cogn Behav Ther.* 2012;41(1):51-62.

43. Otto MW, Reilly-Harrington NA. The impact of treatment on anxiety sensitivity. In: Taylor S, ed. *Anxiety sensitivity: Theory, research, and treatment of the fear of anxiety*. Mahwah, NJ, US: Lawrence Erlbaum Associates Publishers; 1999:321-336.

44. Keough ME, Schmidt NB. Refinement of a brief anxiety sensitivity reduction intervention. *J Consult Clin Psychol.* 2012;80(5):766-772.

45. Feldner MT, Zvolensky MJ, Babson K, Leen-Feldner EW, Schmidt NB. An integrated approach to panic prevention targeting the empirically supported risk factors of smoking and anxiety sensitivity: Theoretical basis and evidence from a pilot project evaluating feasibility and short-term efficacy. *J Anxiety Disord.* 2008;22(7):1227-1243.

46. Zvolensky MJ, Yartz AR, Gregor K, Gonzalez A, Bernstein A. Interoceptive exposure-based cessation intervention for smokers high in anxiety sensitivity: A case series. *J Cogn Psychother.* 2008;22(4):346-365.

47. Zvolensky MJ, Bogiaizian D, Salazar PL, Farris SG, Bakhshaie J. An anxiety sensitivity reduction smoking-cessation program for Spanish-speaking smokers. *Cogn Behav Pract.* 2014;21(3):350-363.

48. Smits PB, Verbeer JH, Nauta MC, Ten Cate TJ, Metz JC, Van Dijk FJ. Factors predictive of successful learning in postgraduate medical education. *Med Educ.* 2004;38(7):758-766.

49. Zvolensky MJ, Lejuez C, Kahler CW, Brown RA. Integrating an interoceptive exposure-based smoking cessation program into the cognitive-behavioral treatment of panic disorder: Theoretical relevance and case demonstration. *Cognitive and Behavioral Practice.* 2003;10(4):347-357.

50. Zvolensky MJ, Schmidt NB, Farris SG, Allen NP. Anxiety sensitivity reduction for smoking cessation: A randomized clinical trial. Society for Research on Nicotine and Tobacco; 2015; Philadelphia, PA.

51. Schmidt NB, Raines AM, Allan NP, Zvolensky MJ. Anxiety sensitivity risk reduction in smokers: A randomized control trial examining effects on panic. *Behaviour research and therapy.* 2016;77:138-146.

52. Smits JA, Zvolensky MJ, Davis ML, et al. The efficacy of vigorous-intensity exercise as an aid to smoking cessation in adults with high anxiety sensitivity: a randomized controlled trial. *Psychosomatic medicine.* 2016;78(3):354-364.

53. Arigo D, Jake-Schoffman DE, Wolin K, Beckjord E, Hekler EB, Pagoto SL. The history and future of digital health in the field of behavioral medicine. *Journal of behavioral medicine.* 2019;42(1):67-83.

54. Carroll K, Ball S, Martino S, et al. Computer-assisted delivery of cognitive-behavioral therapy for addiction: A randomized trial of CBT4CBT. *Am J Psychiatry.* 2008;165(7):881-888.

55. Fiore MC, Jaen CR, Baker TB, et al. *Treating Tobacco Use and Dependence: 2008 Update.* Clinical Practice Guideline. Rockville, MD: U.S. Department of Health and Human Services. Public Health Service. 2008.2008.

56. Walters ST, Wright JA, Shegog R. A review of computer and Internet-based interventions for smoking behavior. *Addict Behav.* 2006;31(2):264-277.

57. Proudfoot J, Ryden C, Everitt B, et al. Clinical efficacy of computerised cognitive–behavioural therapy for anxiety and depression in primary care: Randomised controlled trial. *Br J Psychiatry.* 2004;185(1):46-54.

58. Schmidt NB, Eggleston AM, Woolaway-Bickel K, Fitzpatrick KK, Vasey MW, Richey JA. Anxiety Sensitivity Amelioration Training (ASAT): A longitudinal

primary prevention program targeting cognitive vulnerability. *J Anxiety Disord.* 2007;21(3):302-319.

59. Richards D, Richardson T. Computer-based psychological treatments for depression: a systematic review and meta-analysis. *Clinical psychology review.* 2012;32(4):329-342.

60. Krebs P, Prochaska JO, Rossi JS. A meta-analysis of computer-tailored interventions for health behavior change. *Preventive medicine.* 2010;51(3-4):214-221.

61. Ryan P, Lauver DR. The efficacy of tailored interventions. *Journal of Nursing Scholarship.* 2002;34(4):331-337.

62. Miller WR, Rollnick S. *Motivational interviewing: Preparing people to change.* 2nd ed. New York: Guilford; 2002.

63. Bandura A. *Self-efficacy: The exercise of control.* 1st ed. New York: Freeman; 1997.

64. McClure JB. Are biomarkers a useful aid in smoking cessation? A review and analysis of the literature. *Behav Med.* 2001;27(1):37-47.

65. Curry SJ, McBride C, Grothaus LC, Louie D, Wagner EH. A randomized trial of self-help materials, personalized feedback, and telephone counseling with nonvolunteer smokers. *J Consult Clin Psychol.* 1995;63(6):1005-1014.

66. Curry SJ, Wagner EH, Grothaus LC. Evaluation of intrinsic and extrinsic motivation interventions with a self-help smoking cessation program. *J Consult Clin Psychol.* 1991;59(2):318-324.

67. Becona E, Vazquez FL. Effectiveness of personalized written feedback through a mail intervention for smoking cessation: A randomized-controlled trial in Spanish smokers. *J Consult Clin Psychol.* 2001;69(1):33-40.

68. Strecher VJ. Computer-tailored smoking cessation materials: A review and discussion. *Patient Educ Couns.* 1999;36(2):107-117.

69. Geisner IM, Neighbors C, Larimer ME. A randomized clinical trial of a brief, mailed intervention for symptoms of depression. *J Consult Clin Psychol.* 2006;74(2):393-399.

70. Korte KJ, Schmidt NB. Motivational enhancement therapy reduces anxiety sensitivity. *Cognit Ther Res.* 2013;37(6):1140-1150.

71. Heatherton TF, Kozlowski LT, Frecker RC, FAGERSTROM KO. The Fagerström test for nicotine dependence: a revision of the Fagerstrom Tolerance Questionnaire. *British journal of addiction.* 1991;86(9):1119-1127.

72. Miller W, Rollnick S. Applications of motivational interviewing. Motivational interviewing: Helping people change (3rd edition). New York, NY …; 2013.

73. Neighbors C, Larimer ME, Lewis MA. Targeting misperceptions of descriptive drinking norms: Efficacy of a computer-delivered personalized normative feedback intervention. *J Consult Clin Psychol.* 2004;72(3):434-447.

74. Neighbors C, Lewis MA, Atkins DC, et al. Efficacy of web-based personalized normative feedback: A two-year randomized controlled trial. *J Consult Clin Psychol.* 2010;78(6):898-911.

75. McGrath C, Palmgren PJ, Liljedahl M. Twelve tips for conducting qualitative research interviews. *Medical teacher.* 2018:1-5.

76. Brown RA, Lejuez C, Kahler CW, Strong DR. Distress tolerance and duration of past smoking cessation attempts. *Journal of abnormal psychology.* 2002;111(1):180.

77. Fagerström K. Determinants of tobacco use and renaming the FTND to the Fagerström Test for Cigarette Dependence. *Nicotine & Tobacco Research.* 2012;14(1):75-78.

78. Pomerleau CS, Carton SM, Lutzke ML, Flessland KA, Pomerleau OF. Reliability of the Fagerstrom tolerance questionnaire and the Fagerstrom test for nicotine dependence. *Addictive behaviors.* 1994;19(1):33-39.

79. Loubere N. Questioning transcription: the case for the systematic and reflexive interviewing and reporting (SRIR) method. Paper presented at: Forum Qualitative Sozialforschung/Forum: Qualitative Social Research2017.

80. Markle DT, West RE, Rich PJ. Beyond transcription: Technology, change, and refinement of method. Paper presented at: Forum Qualitative Sozialforschung/Forum: Qualitative Social Research2011.

81. Perkins KA, Karelitz, J. L., & Jao, N. C. Optimal carbon monoxide criteria to confirm 24-hr smoking abstinence. *Nicotine and Tobacco Research.* 2013;15(5):978-982.

82. Osman A, Bagge CL, Gutierrez PM, Konick LC, Kopper BA, Barrios FX. The Suicidal Behaviors Questionnaire-Revised (SBQ-R): Validation with clinical and nonclinical samples. *Assessment.* 2001;8(4):443-454.

83. Taylor S, Zvolensky MJ, Cox BJ, et al. Robust dimensions of anxiety sensitivity: development and initial validation of the Anxiety Sensitivity Index-3. *Psychological assessment.* 2007;19(2):176.

84. Reiss S, McNally R. *Expectancy model of fear.* San Diego, CA: Academic Press; 1985.

85. Farris SG, DiBello AM, Allan NP, Hogan J, Schmidt NB, Zvolensky MJ. Evaluation of the Anxiety Sensitivity Index-3 among treatment-seeking smokers. *Psychological Assessment.* 2015;27(3):1123-1128.

86. Wardenaar KJ, van Veen T, Giltay EJ, de Beurs E, Penninx BW, Zitman FG. Development and validation of a 30-item short adaptation of the Mood and Anxiety Symptoms Questionnaire (MASQ). *Psychiatry research.* 2010;179(1):101-106.

87. Watson D, Clark LA, Weber K, Assenheimer JS, Strauss ME, McCormick RA. Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of abnormal Psychology.* 1995;104(1):15.

88. Burns DD. The Self-Help Scale. Stanford, CA: Stanford University; 2003.

89. Brose LS, Hitchman SC, Brown J, West R, McNeill A. Is the use of electronic cigarettes while smoking associated with smoking cessation attempts, cessation and reduced cigarette consumption? A survey with a 1-year follow-up. *Addiction.* 2015;110(7):1160-1168.

90. Hughes JR, Carpenter MJ. The feasibility of smoking reduction: an update. *Addiction.* 2005;100(8):1074-1089.

91. Brown RA, Burgess ES, Sales SD, Whiteley JA, Evans DM, Miller IW. Reliability and validity of a smoking timeline follow-back interview. *Psychology of Addictive Behaviors.* 1998;12(2):101.

92. Sobell LC, Sobell MB. Timeline follow-back. *Measuring alcohol consumption*: Springer; 1992:41-72.

93. Sobell MB, Sobell LC. *Problem drinkers: Guided self-change treatment.* Guilford Press; 1996.

94. Hamer RM, Simpson PM. Last observation carried forward versus mixed models in the analysis of psychiatric clinical trials. Am Psychiatric Assoc; 2009.

95. Chou C-P, Chi F, Weisner C, Pentz M, Hser Y-I. Initial status in growth curve modeling for randomized trials. *Journal of drug issues.* 2010;40(1):155-172.

96. Muthén L, Muthén B. Statistical analysis with latent variables using Mplus. *Los Angeles, CA: Muthén & Muthén.* 2007.

97. Kline RB. *Principles and practice of structural equation modeling.* Guilford publications; 2015.

98. McNeill A. Harm reduction. *Bmj.* 2004;328(7444):885-887.

99. Polosa R, Caponnetto P, Morjaria JB, Papale G, Campagna D, Russo C. Effect of an electronic nicotine delivery device (e-Cigarette) on smoking reduction and cessation: a prospective 6-month pilot study. *BMC public health.* 2011;11(1):786.

100. Carpenter MJ, Hughes JR, Gray KM, Wahlquist AE, Saladin ME, Alberg AJ. Nicotine therapy sampling to induce quit attempts among smokers unmotivated to quit: a randomized clinical trial. *Archives of internal medicine.* 2011;171(21):1901-1907.

101. Schane RE, Ling PM, Glantz SA. Health effects of light and intermittent smoking: a review. *Circulation.* 2010;121(13):1518-1522.

102. Hughes JR, Keely JP, Niaura RS, Ossip-Klein DJ, Richmond RL, Swan GE. Measures of abstinence in clinical trials: issues and recommendations. *Nicotine & Tobacco Research.* 2003;5(1):13-25.

103. Westmaas JL, Brandon TH. Reducing risk in smokers. *Current opinion in pulmonary medicine.* 2004;10(4):284-288.

Table 1.
Comparison of Baseline Demographic Variables, ASI-3 Total Score, MASQ-D30 Subscales, Self Help Scale, FTND Score, and Smoking History across PFI and Control Conditions

	Personalized Feedback		Control			Overall	
Baseline Variables	Mean	*SD*	Mean	*SD*	*t* (93)	Mean	*SD*
Age (in years)	46.65	9.94	45.72	11.93	-0.42	45.40	11.32
Highest education level	3.33	1.30	3.48	1.07	0.62	3.40	1.20
Income	2.94	1.81	3.03	2.06	0.18	2.99	1.93
ASI-3 Total	22.63	18.09	22.17	16.91	-0.13	20.29	16.75
MASQ-D30 AD	31.86	6.70	31.93	8.54	0.05	30.91	7.96
MASQ-D30 AA	15.80	7.57	17.37	7.08	1.05	15.93	7.28
MASQ-D30 GD	20.04	8.76	19.59	8.26	-0.26	19.25	8.60
SHS mean	2.71	0.92	2.67	0.81	-0.20	2.68	0.88
CO ppm	16.76	9.62	16.98	11.94	0.10	14.94	10.29
Cigarettes per Day	15.86	11.28	20.61	29.31	1.06	15.90	18.96
FTCD	5.55	1.72	5.27	2.00	-0.74[1]	4.99	2.06
Years Daily Smoker	24.04	10.33	23.78	11.84	-0.11	22.61	11.63
	%	*N*	%	*N*	χ^2 (1)	%	*N*
Sex (% Female)	22.45	11	52.17	24	9.01*	36.84	46

Note. * $p < .05$. ASI-3: Anxiety Sensitivity Index-3; MASQ-D30 AD: Mood and Anxiety Symptoms Questionnaire-Anhedonic Depression; MASQ-D30 AA: Mood and Anxiety Symptoms Questionnaire-Anxious Arousal; MASQ-D30 GD: Mood and Anxiety Symptoms Questionnaire-General Distress; SHS: Self-Help Scale. CO ppm: carbon monoxide parts per million; FTCD: Fagerström Test for Cigarette Dependence. Highest Education Level coded as 1 = graduate school to 7 = less than 7 years of school; Income coded as 1 = $0 to $4,999 to 8 ≥ $75,000).
[1] *t* (92)

Table 2.
Baseline Bivariate Correlation among Study Variables.

Variable	1	2	3	4	5	6
1. Sex	--					
2. FTCD	.02	--				
3. ASI-3	.18	.03	--			
4. MASQ-D30 GD	.09	.20	.60**	--		
5. MASQ-D30 AA	.22*	.21*	.54**	.78**	--	
6. MASQ-D30 AD	.02	-.001	.16	.17	.02	--
7. SHS mean	-.06	-.19	-.07	-.04	-.03	-.45**

Note. ** $p < .001$, * $p < .05$. $N = 95$. Sex: 0 = male, 1 = female. FTCD: Fagerström Test for Cigarette Dependence; ASI-3: Anxiety Sensitivity Index-3; MASQ-D30 GD: Mood and Anxiety Symptoms Questionnaire-General Distress; MASQ-D30 AA: Mood and Anxiety Symptoms Questionnaire-Anxious Arousal; MASQ-D30 AD: Mood and Anxiety Symptoms Questionnaire-Anhedonic Depression; SHS: Self-Help Scale.

Table 3.
ASI-3 Total Score, MASQ-D30 Subscales, and Self Help Scale across Conditions at Follow-ups

	Post-Intervention		2-Week Follow-up		4-Week Follow-up	
Control	Mean	*SD*	Mean	*SD*	Mean	*SD*
ASI-3 Total	20.43	17.41	24.26	16.35	21.84	16.90
MASQ-D30 GD	18.38	7.57	20.08	9.15	20.19	8.94
MASQ-D30 AA	17.11	8.16	17.74	6.71	18.71	9.05
MASQ-D30 AD	33.24	9.40	31.31	9.18	32.76	10.37
SHS mean	2.42	1.01	2.44	0.99	2.53	1.04
PFI						
ASI-3 Total	21.54	18.57	23.67	19.72	20.66	16.74
MASQ-D30 GD	17.98	8.18	19.90	7.98	18.78	6.50
MASQ-D30 AA	15.87	7.87	15.77	6.86	14.58	5.00
MASQ-D30 AD	33.20	7.54	31.13	8.59	31.67	7.89
SHS mean	2.72	0.99	2.59	0.90	2.61	1.10

Note. PFI: Personalized Feedback Intervention. ASI-3: Anxiety Sensitivity Index-3; MASQ-D30 GD: Mood and Anxiety Symptoms Questionnaire-General Distress; MASQ-D30 AA: Mood and Anxiety Symptoms Questionnaire-Anxious Arousal; MASQ-D30 AD: Mood and Anxiety Symptoms Questionnaire-Anhedonic Depression; SHS: Self-Help Scale.

Table 4.

Latent Growth Curve Parameters for ASI-3 Scores from Post-Intervention to 4-Week Follow-Up Predicted by Treatment Condition, Baseline ASI-3, Sex, and Cigarette Dependence.

ASI-3 Model Parameters	*β*	*p*
Intercept	1.250	<.001
Intercept Variance	0.153	0.002
Slope	-0.079	0.761
Slope Variance	0.924	<.001
Covariate Effects (Intercept)	*β*	*p*
Condition	0.019	0.715
BL ASI-3	0.907	<.001
Sex	0.036	0.481
FTCD	0.107	0.041
Covariate Effects (Slope)	*β*	*p*
Condition	0.004	0.975
BL ASI-3	-0.259	0.050
Sex	0.146	0.303
FTCD	0.030	0.829

Note. ASI-3 = Anxiety Sensitivity Index-3. BL = Baseline. Condition coded as 0 = Control, 1 = Personalized Feedback Intervention. Sex coded as 0 = male, 1 = female. FTCD = Fagerström Test for Cigarette Dependence.

Table 5.

Latent Growth Curve Parameters for General Distress Scores from Post-Intervention to 4-Week Follow-Up Predicted by Treatment Condition, Baseline General Distress, Sex, and Cigarette Dependence.

General Distress Model Parameters	*β*	*p*
Intercept	2.649	<.001
Intercept Variance	0.108	0.019
Slope	0.106	0.562
Slope Variance	0.862	<.001
Covariate Effects (Intercept)	*β*	*p*
Condition	-0.006	0.915
BL General Distress	0.942	<.001
Sex	-0.014	0.806
FTCD	0.078	0.201
Covariate Effects (Slope)	*β*	*p*
Condition	-0.083	0.445
BL General Distress	-0.314	0.017
Sex	0.158	0.222
FTCD	-0.080	0.547

Note. BL = Baseline. Condition coded as 0 = Control, 1 = Personalized Feedback Intervention. Sex coded as 0 = male, 1 = female. FTCD = Fagerström Test for Cigarette Dependence.

Table 6.

Latent Growth Curve Parameters for Anxious Arousal Scores from Post-Intervention to 4-Week Follow-Up Predicted by Treatment Condition, Baseline Anxious Arousal, Sex, and Cigarette Dependence.

Anxious Arousal Model Parameters	*β*	*p*
Intercept	2.668	<0.001
Intercept Variance	0.123	0.235
Slope	0.228	0.354
Slope Variance	0.742	0.001
Covariate Effects (Intercept)	*β*	*p*
Condition	0.043	0.531
BL Anxious Arousal	0.931	<0.001
Sex	0.017	0.806
FTCD	0.096	0.253
Covariate Effects (Slope)	*β*	*p*
Condition	-0.324	0.044
BL Anxious Arousal	-0.347	0.184
Sex	0.209	0.344
FTCD	-0.063	0.753

Note. BL = Baseline. Condition coded as 0 = Control, 1 = Personalized Feedback Intervention. Sex coded as 0 = male, 1 = female. FTCD = Fagerström Test for Cigarette Dependence.

Table 7.

Latent Growth Curve Parameters for Anhedonic Depression Scores from Post-Intervention to 4-Week Follow-Up Predicted by Treatment Condition, Baseline Anhedonic Depression, Sex, and Cigarette Dependence.

Anhedonic Depression Model Parameters	*β*	*p*
Intercept	4.402	<0.001
Intercept Variance	0.275	0.005
Slope	-0.096	0.706
Slope Variance	0.918	<0.001
Covariate Effects (Intercept)	*β*	*p*
Condition	0.042	0.604
BL Anhedonic Depression	0.839	< 0.001
Sex	0.136	0.078
FTCD	0.016	0.849
Covariate Effects (Slope)	*β*	*p*
Condition	-0.091	0.540
BL Anhedonic Depression	-0.180	0.069
Sex	-0.054	0.699
FTCD	0.205	0.166

Note. BL = Baseline. Condition coded as 0 = Control, 1 = Personalized Feedback Intervention. Sex coded as 0 = male, 1 = female. FTCD = Fagerström Test for Cigarette Dependence.

Table 8.

Latent Growth Curve Parameters for SHS Scores from Post-Intervention to 4-Week Follow-Up Predicted by Treatment Condition, Baseline SHS, Sex, and Cigarette Dependence.

SHS Model Parameters	*β*	*p*
Intercept	3.213	<0.001
Intercept Variance	0.374	0.002
Slope	-0.146	0.617
Slope Variance	0.759	0.012
Covariate Effects (Intercept)	*β*	*p*
Condition	0.102	0.321
BL SHS	0.734	< 0.001
Sex	-0.140	0.172
FTCD	0.181	0.030
Covariate Effects (Slope)	*β*	*p*
Condition	-0.133	0.585
BL SHS	-0.232	0.265
Sex	0.356	0.263
FTCD	0.035	0.865

Note. SHS = Self Help Scale BL = Baseline. Condition coded as 0 = Control, 1 = Personalized Feedback Intervention. Sex coded as 0 = male, 1 = female. FTCD = Fagerström Test for Cigarette Dependence.

Figure 1. Study Consort