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Treatment Non-response: Associations with Smoking Expectancies among Treatment-Seeking Smokers

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Abstract

Despite the high rate of smoking cessation treatment non-response, relatively little empirical work has examined predictors of treatment non-response. The present study sought to explore the effect of smoking outcome expectancies on treatment response in a sample of treatment-seeking adult daily smokers (N = 182; 53.3% female; M_{age} = 40.67; SD = 13.63). Results indicated that expectancies for smoking to reduce negative affect were related to an increased likelihood of treatment non-response (OR = 0.73, CI: 0.54, 0.98). These findings remained significant after controlling for sex, presence of Axis I disorder, tobacco-related health problems, tobacco dependence, anxiety sensitivity, and condition assignment as well as other smoking expectancy dimensions. Post hoc analyses revealed that this relation was stronger for smokers in the integrated care condition vs. the standard care condition (Interaction: OR = 1.69, CI: 1.05, 2.73). Additionally, expectancies for smoking to enhance positive affect and provide sensory satisfaction were associated with an increased likelihood of treatment response in the standard care condition. The current findings suggest expectancies that smoking will alleviate negative affect may be a risk factor of smoking cessation treatment non-response. Additionally, findings provide evidence that the relation between smoking expectancies and treatment non-response may differ by smoking cessation treatment.

Keywords

smoking cessation; treatment non-response; smoking expectancy; smoking treatment; treatment outcomes

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Rates of smoking cessation treatment non-response, or failure to achieve initial abstinence after completing treatment, are as high as 50% for leading smoking cessation treatments (Burns et al., 2016). Despite the high rate of non-response to these smoking cessation treatments, relatively little empirical work has examined predictors of non-response. Indeed, the concept of treatment non-response has rarely been investigated in the smoking literature (Hughes, Grass, & Pillitteri, 2000) and the vast majority of research focused on smoking cessation treatment non-response has examined the efficacy of novel interventions to treat non-responders (Jiménez-Ruiz et al., 2002). One study found that smokers who held more positive beliefs about smoking were more likely to not respond to treatment (Wynd, 2007). Beyond this single study, to our knowledge, no other work has examined predictors of smoking cessation treatment non-response. This line of inquiry may be underdeveloped, in part, because of unclear definitions of treatment non-response (Hajek, Stead, West, Jarvis, & Lancaster, 2009; Hughes et al., 2003). For example, it is common practice to classify smoking cessation relapsers, drop-outs, and non-responders as ‘relapsers’ (Hughes et al., 2003). Yet, drawing more broadly from non-smoking clinical research (Frank et al., 1991), treatment response followed by relapse, dropping-out of treatment, and treatment non-response represent distinct, yet related, stages of recovery (Fava, 2003; Schottenbauer, Glass, Arnkoff, Tendick, & Gray, 2008; Taylor, Abramowitz, & McKay, 2012). Thus, work devoted to understanding predictors of smoking cessation response is warranted to better understand the nature of ‘non-responders.’

Despite limited understanding of smoking cessation non-response, efforts have been made to address low cessation rates and high rates of non-response. For example, specialized algorithms have been developed to assist clinicians with triaging hard-to-treat smokers (Bittoun, 2006, 2007; Hughes, 2008). Although these programs can be helpful to identify potentially recalcitrant smokers, they do not identify or provide clinical guidance for smokers likely to relapse versus those likely to not respond to treatment (Bittoun, 2006; Hughes, 2008). Indeed, considering the dearth of research focused on smoking cessation non-response, current capabilities to develop an algorithm for treatment that considers non-responders are limited. From a public health perspective, it may be advantageous to examine potential predictors of treatment non-response that could be considered in a clinical algorithm, which may subsequently lead to improved outcomes (Hughes, 2008) and decrease the use of valuable and costly cessation resources (Abrams et al., 1996). As such, empirically investigating determinants of treatment non-response may have the potential to inform universal and targeted cessation efforts.

Smoking outcome expectancies are one promising smoking construct potentially related to smoking cessation treatment non-response. Smoking outcome expectancies reflect beliefs about the expected effects and consequences from smoking, including negative affect relief, positive affect enhancement, weight management, and negative health consequences (Gwaltney, Shiffman, Balabanis, & Paty, 2005). Numerous empirical studies document the importance of outcome expectancies as a proximal mediator of smoking motivation (Brandon, Juliano, & Copeland, 1999; Goldman, Brown, & Christiansen, 1987; Marlatt & Donovan, 2005; Niaura et al., 1988). Smoking expectancies are related to smoking initiation (Doran et al., 2013; Goldman et al., 1987), cigarette dependence (Copeland, Brandon, &

Quinn, 1995; Myers, MacPherson, McCarthy, & Brown, 2003), tobacco withdrawal (Langdon & Leventhal, 2014; Wetter et al., 1994), and cessation outcome (Kenford et al., 2002; Wetter et al., 1999; Wetter et al., 1994). However, past work has not yet examined how smoking outcome expectancies may influence smoking cessation treatment response.

The current study evaluated the relation between smoking expectancies and treatment response. We examined the main effects of smoking expectancy domains (Negative Reinforcement/Negative Affect Reduction, Negative Consequences, Positive Reinforcement/Sensory Satisfaction, and Appetite-Weight Control) on treatment response. We hypothesized that greater positive expectancies and fewer negative smoking expectancies would relate to an increased likelihood of treatment non-response.

Methods

Participants

Participants were 182 (53.3% female; M_{age} = 40.67, *SD* = 13.63; $Median_{age}$ = 44, *Range* = 18-64) treatment-seeking adult daily smokers who reported smoking at least 8 cigarettes per day, elevated anxiety sensitivity based on the Anxiety Sensitivity Index-3 (ASI-3; Taylor et al., 2007) during a baseline assessment, and provided quit-day and 1-week post quit-day data. Exclusion criteria included current suicidality warranting immediate intervention, psychosis, or unmedicated bipolar disorder. The racial and ethnic distribution of this sample was as follows: 89.6% identified as white/Caucasian; 3.9% as black/Non-Hispanic; 0.6% as black/Hispanic; 3.3% as Hispanic; 0.6% as Asian; and 2.2% as 'Other.' At least one current (past year) Axis I diagnosis was endorsed by 35.7% of the sample, most commonly social anxiety disorder (8.79%), generalized anxiety disorder (3.85%), posttraumatic stress disorder (3.85%), current major depressive episode (3.30%), and alcohol dependence (3.30%). On average, participants reported smoking 17.7 cigarettes per day (*SD* = 7.66; *Median* = 18; *Range* = 8-60) and had been a daily smoker for 21.9 years (*SD* = 13.41; *Median* = 22; *Range* = 1-50). A moderate level of cigarette dependence was observed within the sample based on the FTCD (M=5.42, SD=2.05; Heatherton, Kozlowski, Frecker, & Fagerström, 1991).[1]

Procedure

Data for the present study were collected during a large, multi-site randomized controlled clinical trial examining the efficacy of two smoking cessation interventions described in detail elsewhere (Schmidt, Raines, Allan, & Zvolensky, 2015). Baseline data for the larger trial were collected from 579 treatment-seeking adult daily smokers. Participants were recruited at two sites (Vermont, Florida). Interested persons responding to community-based advertisements (e.g., flyers, newspaper ads, radio announcements) contacted the research team and were provided with a detailed description of the study via phone. Participants were then screened for initial eligibility, and if eligible, scheduled for an appointment. After providing written informed consent, participants were interviewed using the Structured

[1]Scores on the FTCD are interpreted as follows: 1-2 = low dependence, 3-4 = low to moderate dependence, 5-7 = moderate dependence, and 8+ = high dependence.

Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV) Diagnosis of Axis I Disorders Non-Patient Version (SCID-I/NP; First, Spitzer, Gibbon, & Williams, 2007) and completed a computerized self-report assessment battery as well as biochemical verification of smoking status.

Regarding the interventions, eligible participants were randomly assigned to one of two smoking cessation treatment programs and scheduled for treatment initiation approximately 1–2 weeks after the baseline assessment. Smoking cessation treatment consisted of either (a) Standard Care for Smoking Cessation or (b) Integrated Care Smoking Cessation. The Integrated Care Smoking Cessation (active) treatment integrated (1) interoceptive exposure, cognitive restructuring, and psychoeducation exercises developed for panic prevention and (2) standard of care treatment for standard smoking cessation. The Standard Care for Smoking Cessation treatment (control) included the smoking-related component of the Integrated Care Smoking Cessation Program as well as a review of general health information not specific to anxiety or smoking (to maintain equal contact time across the two conditions). It is important to note that although anxiety symptoms were not directly targeted in the Standard Care intervention, it had a stress management component, and across both conditions, a significant average decrease in anxiety related symptoms was observed during the treatment period (Schmidt et al., 2015). In addition to counseling, both treatment groups received nicotine replacement therapy via the transdermal nicotine patch, which was initiated at treatment Session 4 (quit-day). Treatment consisted of four 60-minute weekly sessions conducted by trained doctoral-level graduate students. All treatment was supervised by principal investigators (MJZ and NBS) and checked for treatment fidelity by independent reviewers. Data collection began in 2007 and concluded in 2014.

The current study is based on secondary analyses of baseline, quit-day, and 1-week follow-up data for a subset of the sample. Specifically, only those who met inclusion criteria and provided compete data for variables of interest were included. Considering the focus on treatment non-responders, participants who dropped out of treatment prior to the 1-week follow-up were excluded from the current study. The study protocol was approved by the Institutional Review Boards at the University of Vermont and Florida State University (clinicaltrials.gov # NCT01753141).

Measures

Demographics Questionnaire—Demographic information collected included sex, age, and race. Items from this measure were used to describe the sample, and sex was included as a covariate.

Diagnostic Interview—The SCID-NP was used to describe the presence of current psychological disorders of the sample (First et al., 2007). All interviews were administered by advanced doctoral level therapists and supervised by a licensed clinical psychologist. All interviews were audio-taped and reliability of a random selection of 12.5% of interviews were checked for accuracy; no cases of disagreement were noted. The presence of a psychiatric disorder was included as a covariate.

Medical History Form—A medical history checklist was used to assess current and lifetime medical problems. As in past work (Buckner et al., 2015; Farris, Zvolensky, Blalock, & Schmidt, 2014; Leventhal, Zvolensky, & Schmidt, 2011), a composite variable was computed as an index of tobacco-related medical problems (labeled 'Health'). Specifically, items in which participants indicated having ever been diagnosed (heart problems, hypertension, respiratory disease, or asthma; all coded 0 [*no*] or 1 [*yes*]) were summed and a total score was created, with greater scores reflecting the occurrence of multiple markers of tobacco-related disease.

Fagerström Test for Cigarette Dependence (FTCD)—The FTCD is a 6-item scale that assesses gradations in tobacco dependence (e.g, how soon after you wake up do you smoke your first cigarette; Fagerström, 2012; Heatherton et al., 1991). 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 (Heatherton et al., 1991; Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994). In the current study, the FTCD total score was used to characterize cigarette dependence (α = .62).

Anxiety Sensitivity Index-3 (ASI-3)—The ASI-3 is an 18-item self-report measure of the sensitivity to and fear of the potential negative consequences of anxiety-related symptoms and sensations. 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 (e.g., "It is important to me not to appear nervous.") The ASI-3, derived in part from the original ASI (Reiss & McNally, 1985), has sound psychometric properties, including excellent internal consistency, predictive validity, and reliability among smokers (Farris et al., 2015). The baseline ASI-3 total score and treatment session 4 ASI-3 total score were used to evaluate treatment response; the baseline ASI-3 score was also included as a covariate in analyses. In the present study, the baseline ASI-3 total score exhibited excellent internal consistency (baseline: α = .92).

Smoking Consequences Questionnaire (SCQ)—The SCQ (Brandon & Baker, 1991) is a 50-item self-report measure that assesses tobacco use outcome expectancies believed to underlie smoking motivation on a Likert-type scale, ranging from 0 (*completely unlikely*) to 9 (*completely likely*). The measure consists of four key factors: Negative Reinforcement/ Negative Affect Reduction (NR; 12 items; e.g., "Smoking helps me deal with depression"; α = .93), Negative Consequences (NC; 18 items; e.g., "My throat burns after smoking" α = .86), Positive Reinforcement/Sensory Satisfaction (PR; 15 items; e.g., "I will enjoy the flavor of a cigarette"; α = .87), and Appetite-Weight Control (AW; 5 items; e.g., "Smoking helps me control my weight"; α = .92).

Treatment Response—Treatment response was determined in terms of response to (a) smoking abstinence and (b) anxiety sensitivity reduction across both conditions. Consistent with extant work (Burns et al., 2016; MacPherson, Stipelman, Duplinsky, Brown, & Lejuez, 2008), participants who reported any smoking in the first 7 days post-treatment were classified as non-responders and those who reported no smoking were classified as

responders. A modified smoking version of the Timeline Followback Interview (Brown et al., 1998; Sobell & Sobell, 1992) was used to collect daily smoking behavior post-treatment. Additionally, reduction in AS was also considered when evaluating treatment response. Specifically, participants who reported at least a 25% reduction in ASI-3 symptoms from baseline to end of treatment (treatment session 4) were classified as treatment responders whereas participant who evinced a less than 25% reduction in ASI-3 symptoms were classified as non-responders. Extensive work on psychological distress suggests that at least 25% reduction in symptoms is indicative of at least partial response to treatment (Nierenberg & DeCecco, 2001; Pallanti et al., 2002). These data are consistent with observed changes in clinical trials that target anxiety sensitivity (Schmidt, Capron, Raines, & Allan, 2014; Schmidt, Norr, Allan, Raines, & Capron, In Press). Anxiety sensitivity was evaluated in the current conceptualization of treatment response across both conditions because of the observed significant decrease in anxiety related symptoms across both conditions (Schmidt et al., 2015), and scientific evidence for change in anxiety sensitivity in the context of non-specialized treatments (such as the intervention provided for the control condition in the current study (Ablon, Levy, & Katzenstein, 2006; Barlow, 2000; Geckler, McCormack, & Goodman, 1984; McHugh et al., 2014). Participants who responded to at least one aspect of treatment (i.e., smoking abstinence or AS symptom reduction) were classified as a treatment responder. Treatment response was coded as follows: 0 = non-responders; 1 = responders.

Analytic Strategy

Descriptive analyses and zero-order correlations were examined among study variables. To test primary hypotheses, a hierarchical logistic regression model of treatment response (non-responders: $n = 46$; responders: $n = 136$) was estimated. Step 1 included covariates (e.g., sex, presence of Axis I disorder, tobacco-related health problems, FTCD, baseline ASI-3, and condition). Step 2 included the Step 1 covariates and the four SCQ subscales. Post-hoc analyses were conducted to examine the interactive effect of smoking expectancy dimension (i.e., SCQ-NR, SCQ-NC, SCQ-PR, SCQ-AW) by condition on treatment response; an independent model was run for each smoking expectancy dimension.

Results

Descriptive Statistics and Bivariate Correlations

Table 1 presents frequency, means, and standard deviations for all study variables. Bivariate correlations revealed that the presence of psychopathology was significantly correlated with treatment non-response status ($r = -.15$, $p = .05$). SCQ-NR significantly correlated with SCQ-NC ($r = .41$, $p < .001$), SCQ-PR ($r = .50$, $p < .001$), and SCQ-AP ($r = .36$, $p < .001$). SCQ-NC significantly correlated with SCQ-PR ($r = .20$, $p < .001$), and SCQ-AP ($r = .29$, $p < .001$). Lastly, SCQ-PR was significantly related to SCQ-AP ($r = .16$, $p = .03$).

Logistic Regression Model

Step 1 of the logistic regression model was non-significant ($X^2 = 8.39$, $df = 6$, $p = .21$) with a Pseudo R^2 of .04. Presence of psychiatric disorder emerged as the only significant predictor. Step 2 explained more of treatment non-response, but was non-significant ($X^2 =$

15.38, $df = 10$, $p = .12$) with a Pseudo R^2 of .07. Presence of psychiatric disorder and higher SCQ-NR subscale emerged as significant predictors of treatment non-response; see Table 2.

Post-Hoc Analyses

Considering differences in treatment conditions, exploratory analyses were conducted to examine differences in the effect of SCQ subscales on treatment response across condition. Specifically, post-hoc analyses for the interactive effect of treatment condition and each SCQ subscale on treatment response were examined independently. Specifically, four independent models were conducted as a Step 3 to the hierarchical logistic regression wherein all covariates and the four SCQ subscales were included in addition to the interaction term between condition and the specified SCQ subscale.

Step 3a for SCQ-NR revealed a significant model ($X^2 = 20.30$, $df = 11$, $p = .04$) with a Pseudo R^2 of .10. The interaction term emerged as a significant predictor of treatment response; see Table 2. Specifically, increased baseline expectancies for smoking to relieve negative affect were associated with an increased likelihood of treatment non-response in the integrated care condition (b = -10, SE = .03, p = .002) and was unrelated to treatment response in the standard care condition (b = -.02, SE = .03, p = .52). See Figure 1.

Step 3b for SCQ-NC revealed a non-significant model ($X^2 = 16.07$, $df = 11$, $p = .14$) with a Pseudo R^2 of .08. The interaction term was not a significant predictor of treatment response; see Table 2.

Step 3c for SCQ-PR revealed a significant model ($X^2 = 21.71$, $df = 11$, $p = .03$) with a Pseudo R^2 of .11. The interaction term emerged as a significant predictor of treatment response; see Table 2. Increased baseline expectancies for smoking to enhance positive affect and provide sensory satisfaction were associated with an increased likelihood of treatment response in the standard care condition (b = .08, SE = .03, p = .02), but not in the integrated care condition (b = -.04, SE = .03, p = .23). See Figure 2.

Step 3d for SCQ-AP revealed a non-significant model ($X^2 = 16.65$, $df = 11$, $p = .12$) with a Pseudo R^2 of .08. The interaction term was not a significant predictor of treatment response; see Table 2.

Discussion

The current study sought to examine the effect of smoking outcome expectancies on standard care and integrated care smoking cessation treatment response. Specifically, we tested the hypothesis that more positive smoking expectancies would relate to treatment non-response and more negative smoking expectancies would relate to treatment response. Findings revealed that expectancies for smoking to reduce negative affect were related to an increased likelihood of treatment non-response. Importantly, this effect was observed after controlling for factors that may influence responsiveness to treatment, including sex, presence of Axis I disorder, tobacco-related health problems, tobacco dependence, anxiety sensitivity, and condition assignment as well as other smoking expectancy dimensions. These findings are in line with past work indicating a unique relation between positive

beliefs about smoking and difficulty responding to treatment (Wynd, 2007), but suggest there exists unique associations between specific domains of positive expectancies and treatment response.

Theoretically, greater negative beliefs about smoking have been posited to be relevant to treatment response (Fishbein & Ajzen, 1977). However, the current study did not find empirical support for this model. The lack of an association for negative outcome expectancies may have resulted, in part, from the small sample size. Thus, it would be informative to evaluate the proposed models among a larger sample. Alternatively, treatment-seeking smokers who experience elevated negative affect states, such as those recruited for the present trial, are at greater risk for experiencing health problems related to smoking (Bandiera, Anteneh, Le, Delucchi, & Guydish, 2015; CAMH, 2011; Zvolensky et al., 2004). As a result of their own history with smoking-related health consequences, these smokers may report greater beliefs about the negative consequences of smoking. Ultimately, this may lead to an inflated mean with limited variability, which is consistent with the present findings for SCQ-NC across both treatment responders and non-responders. Thus, findings support that smokers with elevated negative affect, overall, expect a high likelihood of experiencing negative consequences of smoking and that this expectancy does not differentiate who will and will not respond to treatment.

Post-hoc analyses revealed two novel additional findings. First, results provided evidence that the effect of expectancies for negative affect reduction on treatment non-response were stronger for smokers in the integrated care condition. Second, expectancies for smoking to enhance positive affect and provide sensory satisfaction were associated with an increased likelihood of treatment response in the standard care condition. Together, these findings suggest integrated care was relatively less effective when smoking was more strongly associated with affect regulation for reducing negative affect, and standard care may be more effective when smokers expect smoking to increase positive affect. These findings may have emerged, in part, because of the novel approach to evaluate treatment response in the current study. Specifically, the current study evaluated response in terms of (a) smoking and (b) anxiety sensitivity. To our knowledge, this is the first study to examine treatment response across multiple of domains of treatment. Conceptualizing treatment response in this way may have the potential to decrease the false discovery rate and enable clinical scientists and practitioners to focus on treatments that demonstrate efficacy across a least one domain of impairment. Thus, the present conceptualization broadens current methods for evaluating treatment response in the context of integrated, as well as standard, smoking cessation treatments. In subsequent research, it may be fruitful to consider tailoring content for individuals who have strong associations between smoking and reducing negative affect and/or increasing positive affect. Indeed, considering the high rate of psychiatric illness among smokers (Lasser et al., 2000; Zvolensky et al., in press), it could be important for future work to employ integrated care options for treatment-seeking smokers and evaluate such interventions in terms of treatment response.

The current findings provide initial empirical evidence for the importance of smoking expectancies when attempting to identify smokers at the highest risk for treatment non-response. As such, it may be clinically informative for clinicians to assess smoking

expectancies prior to triaging treatment-seeking smokers. Indeed, understanding a patient's likelihood to respond to treatment is particularly important for their treatment plan. Unfortunately, measures that may help identify smokers least likely to respond to treatment, such as the SCQ, are not commonly administered to treatment-seeking smokers upon treatment initiation. In light of the present findings, however, administering such measures may offer clinicians a more thorough understanding of the patient's likelihood of treatment response that can then be used to inform a personalized treatment plan.

There are several interpretive caveats to the present study. First, the sample consisted of community-recruited, treatment-seeking daily cigarette smokers with moderate levels of cigarette dependence. Thus, it will be necessary to replicate the study among a sample of both high and low cigarette dependent smokers to improve generalizability. Second, most of the sample were white smokers. The current study should be conducted among a more ethnically/racially diverse sample of smokers in the future. Third, the study examined treatment response at one-week post-treatment. Future work would benefit from examining these relations across a larger post-treatment time-frame (e.g., 3-month, 6-month,) to determine long-term treatment responders. Fourth, the current study relied on self-report measures. Future research could benefit by utilizing multi-method approaches, including biochemical verification of smoking status. Finally, the present study focused exclusively on participants who completed a smoking cessation treatment and omitted those who dropped out. Although research suggest that factors related to treatment non-response and dropout differ (Schottenbauer et al., 2008; Taylor et al., 2012), the exclusion of dropouts may, to some degree, have influenced findings. As a follow-up to the present study, future work should investigate constructs that discriminate treatment responders from non-responders and dropouts. Such work would have important clinical implications for understanding the treatment process.

Overall, the present study serves as an initial investigation on the role of expectancies for smoking to reduce negative affect in treatment response. The results suggest expectancies for smoking to reduce negative affect are related to an increased likelihood of treatment non-response. Future work could build upon this study by examining other smoking processes (e.g., smoking motives, barriers to cessation) that may be associated with treatment response.

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Highlights

- Expectancy for smoking to reduce negative affect predicted treatment non-response.
- Association between smoking expectancies and treatment non-response differed by treatment.
- Clinical utility in addressing smoking expectancies during early stages of quitting.

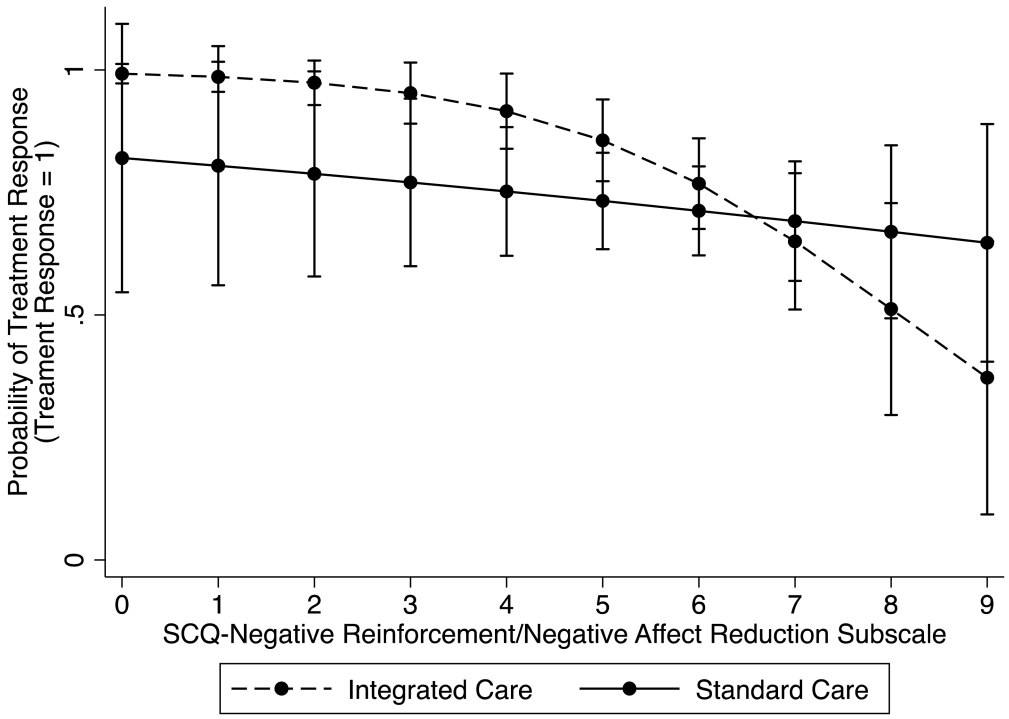

Figure 1. Effect of SCQ-NR on treatment response status by treatment condition

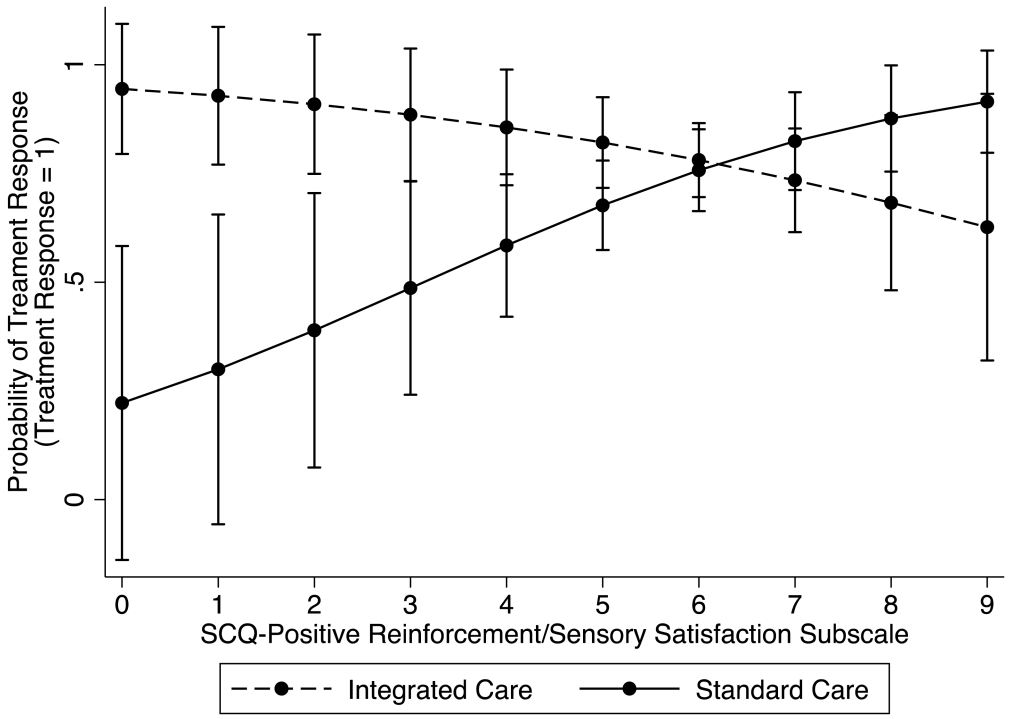

Figure 2. Effect of SCQ-PR on treatment response status by treatment condition

Table 1

Descriptive statistics for the overall sample, treatment responders, and treatment non-responders

Categorical Variables	Overall *(N = 182) N (%)*	Responder *(N = 136) N (%)*	Non-Responder *(N = 46) N (%)*
Condition			
Standard Care	85 (46.70)	59 (43.38)	26 (56.52)
Integrated Care	97 (53.30)	77 (56.62)	20 (43.48)
Gender			
Male	85 (46.70)	60 (44.12)	25 (54.35)
Female	97 (53.30)	76 (55.88)	21 (45.65)
Psychopathology			
Absent	117 (65.29)	93 (68.38)	24 (52.17)
Present	65 (35.71)	43 (31.62)	22 (47.83)
Continuous Variables	Mean (SD)	Mean (SD)	Mean (SD)
Tobacco Health Probs	0.34 (0.60)	0.32 (0.55)	0.41 (0.72)
Baseline FTCD	5.42 (2.05)	5.38 (1.99)	5.54 (2.26)
Baseline ASI-3	13.25 (11.03)	13.38 (11.17)	12.87 (10.75)
SCQ-NR	5.76 (1.74)	5.65 (1.76)	6.11 (1.66)
SCQ-NC	6.67 (1.12)	6.64 (1.04)	6.78 (1.33)
SCQ-PR	5.68 (1.35)	5.67 (1.36)	5.72 (1.35)
SCQ-AW	4.22 (2.26)	4.31 (2.19)	3.96 (2.48)

Note. Tobacco Health Probs=Larger scores reflected occurrence of multiple markers of tobacco-related disease; FTCD=Fagerstrom Test for Cigarette Dependence (Fagerström, 2012; Heatherton et al., 1991); ASI-3=Anxiety Sensitivity Index-3 (Taylor et al., 2007); SCQ-NR=Smoking Consequences Questionnaire-Negative Reinforcement/Negative Affect Reduction (Brandon & Baker, 1991); SCQ-NC=Smoking Consequences Questionnaire-Negative Consequences (Brandon & Baker, 1991); SCQ-PR=Smoking Consequences Questionnaire-Positive Reinforcement/Sensory Satisfaction (Brandon & Baker, 1991); SCQ-AW=Smoking Consequences Questionnaire-Appetite-Weight Control (Brandon & Baker, 1991).

Table 2

Hierarchical logistic regression model of smoking expectancies on treatment responsive status (1 = treatment responder)

Step	Predictor	Odds ratio (95% CI)	p-value
Step 1	Gender	1.58 (95% CI 0.79, 3.15)	0.20
	Tobacco Health Probs	0.86 (95% CI 0.50, 1.49)	0.60
	Psychopathology	0.47 (95% CI 0.22, 1.00)	0.05
	Baseline FTCD	0.98 (95% CI 0.83, 1.16)	0.81
	Baseline ASI-3	1.01 (95% CI 0.98, 1.05)	0.45
	Condition	0.66 (95% CI 0.33, 1.33)	0.25
Step 2	Gender	1.79 (95% CI 0.81, 3.96)	0.15
	Tobacco Health Probs	0.85 (95% CI 0.48, 1.48)	0.56
	Psychopathology	0.545 (95% CI 0.21, 0.96)	0.04
	Baseline FTCD	0.97 (95% CI 0.81, 1.16)	0.73
	Baseline ASI-3	1.03 (95% CI 0.99, 1.07)	0.13
	Condition	0.72 (95% CI 0.35, 1.49)	0.38
	SCQ-NR	0.73 (95% CI 0.54, 0.98)	0.04
	SCQ-NC	0.89 (95% CI 0.61, 1.30)	0.55
	SCQ-PR	1.12 (95% CI 0.81, 1.53)	0.50
	SCQ-AW	1.13 (95% CI 0.94, 1.34)	0.19
Step 3a	SCQ-NR × Condition	1.69 (95% CI 1.05, 2.73)	0.03
Step 3b	SCQ-NC × Condition	1.33 (95% CI 0.67, 2.62)	0.41
Step 3c	SCQ-PR × Condition	2.04 (95% CI 1.15, 3.62)	0.02
Step 3d	SCQ-AW × Condition	1.20 (95% CI 0.87, 1.64)	0.27

Note. N = 182; Gender coded as Male=0, Female=1; Condition coded as 0= Integrated Care, 1=Standard Care; Psychopathology coded as 0=Absent, 1=Present; Response status coded as 0=non-response, 1-response; Tobacco Health Probs =Larger scores reflected occurrence of multiple markers of tobacco-related disease FTCD=Fagerstrom Test for Cigarette Dependence (Fagerström, 2012; Heatherton et al., 1991); ASI-3=Anxiety Sensitivity Index-3 (Taylor et al., 2007); SCQ-NR=Smoking Consequences Questionnaire-Negative Reinforcement/Negative Affect Reduction (Brandon & Baker, 1991); SCQ-NC=Smoking Consequences Questionnaire-Negative Consequences(Brandon & Baker, 1991); SCQ-PR=Smoking Consequences Questionnaire-Positive Reinforcement/Sensory Satisfaction (Brandon & Baker, 1991); SCQ-AW=Smoking Consequences Questionnaire-Appetite-Weight Control (Brandon & Baker, 1991).

*
p<.05,

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p<.01,

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p<.01