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Randomized Controlled Trial of Behavioral Activation Smoking Cessation Treatment for Smokers with Elevated Depressive Symptoms

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Abstract

Objective—Depressive symptoms are associated with poor smoking cessation outcomes and there remains continued interest in behavioral interventions that simultaneously target smoking

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and depressive symptomatology. This pilot study examined whether a behavioral activation treatment for smoking can enhance cessation outcomes.

Method—A sample of 68 adult smokers with mildly elevated depressive symptoms (*M* = 43.8 years old, 48.5% female, 72.7% African-American) seeking smoking cessation treatment were randomized to receive either behavioral activation treatment for smoking (BATS) paired with standard smoking cessation strategies including nicotine replacement therapy (*n* = 35) or standard smoking cessation strategies (ST) alone including nicotine replacement therapy (*n* = 33). BATS and ST were matched for contact time and included 8 sessions of group-based treatment. Quit date was assigned to occur at session 4 for each treatment condition. Participants completed a baseline assessment, and measures of smoking cessation outcomes (7-day verified point prevalence abstinence), depressive symptoms (BDI-II), and enjoyment from daily activities (EROS) were obtained at 1, 4, 16, and 26 weeks post assigned quit date.

Results—Across the follow-ups over 26 weeks, participants in BATS reported greater smoking abstinence (adjusted odds ratio = 3.59; 95% confidence interval = 1.22, 10.53; *p* =.02) than did those in ST. Participants in BATS also reported a greater reduction in depressive symptoms (*B* = -1.99, *SE* = .86, *p* = .02) than did those in ST.

Conclusions—Results suggest BATS is a promising intervention that may promote smoking cessation and improve depressive symptoms among underserved smokers of diverse backgrounds.

Keywords

smoking cessation; behavioral activation; depressive symptoms; low income and minority smokers

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Moderately elevated levels of pre-treatment, *current* depressive symptoms are associated with poor smoking cessation outcomes (e.g., Cinciripini et al., 2003; Niaura et al., 2001). Anti-depressant medications and/or mood specific cognitive behavioral treatments largely have not impacted depressive symptoms during quit attempts (e.g., Kahler et al., 2002) and treatment effects appear unrelated to depressive symptom change (e.g., Piper et al., 2008). Beyond the putative role of depressive symptoms in cessation failure, emerging research indicates a critical role of low positive affect in poor cessation outcomes (e.g., Leventhal et al., 2008; McCarthy et al., 2008) and in deprivation-induced withdrawal and craving (e.g., Cook et al., 2004). Although extant research typically has focused on the role of negative affectivity/mood on cessation failure (c.f., Spring et al., 2008), it remains crucial to consider low positive affect/anhedonia (i.e., reduced positive emotions and a diminished capacity to experience pleasure; Pizzagelli et al., 2005) as these dimensions have also predicted smoking cessation-related changes in withdrawal symptoms and relapse beyond depression history (Leventhal et al., 2008).

Behavioral activation (BA; Jacobson et al., 1996; Lejuez et al., 2001) strategies may be a promising adjunct to standard cessation strategies for smokers with elevated depressive symptoms, as this is a brief approach that targets greater contact with more valued environments through systematic efforts to increase rewarding experiences/enjoyment of daily activities, which may simultaneously reduce negative affect and improve positive affect through overt behavior change (Hopko, Lejuez et al., 2003). We conducted a small scale randomized clinical trial of BA strategies with standard cognitive-behavioral smoking cessation strategies including transdermal nicotine patch (BATS). The comparison condition received standard smoking cessation treatment including transdermal nicotine patch (ST), matched for overall contact time. We hypothesized participants in BATS would evidence higher point prevalence abstinence rates at 1, 4, 16, and 26 weeks post assigned quit date, as

well as decreased depressive symptoms and enjoyment from daily activities at those time periods.

Method

Participants and Procedures

The initial phone screen required participants to be 18 to 65 years of age, a current regular smoker (≥ 1 year), currently smoking ≥ 10 cigarettes per day, a BDI-II ≥ 10, not evidence a current DSM-IV disorder including MDD as assessed by the screening items from the Structured Clinical Interview for DSM-IV, non-patient version (SCID-NP; First, Spitzer, Gibbon, & Williams, 1995), and not meet any of the exclusion criteria b) through e) listed below. At the baseline assessment (scheduled within 2 weeks of the phone screen), participants were excluded if their BDI-II scores dropped lower than 7 to allow some inevitable drift while still providing a meaningful level of depressive symptoms (e.g., Thorndike et al., 2008), or if they evidenced a) a current Axis I disorder including MDD as assessed by the SCID-NP, b) current use of psychotropic medication, c) current participation in psychotherapy, d) physical concerns contraindicating use of the nicotine patch, e) current use of smoking cessation pharmacotherapy, or f) current use of smokeless tobacco products. Recruitment occurred from April 2006 to January 2008 using radio, web-based, and newspaper advertisements for a smoking cessation intervention consisting of group therapy plus nicotine patch. Advertisements did not mention depressive symptoms and participants were blinded to study goals and hypotheses. Of the 1123 potential participants initially screened by phone for eligibility, 68 participants were randomized by cohort (ranging in size of between three and eight participants) to one of the two treatment conditions (see Figure 1 for participant flow). Of these 68, 26 dropped out prior to attending any treatment sessions. Specifically, 17 participants dropped out prior to receiving any treatment in the ST group and 9 participants dropped out prior to receiving any treatment in the BATS group. Dropout cannot be attributed to treatment assignment because it was not indicated to participants prior to Session 1. These 26 participants who dropped did not differ from the 42 who attended one session on demographics or any other baseline characteristics (e.g., smoking history).

Primary analyses include the 68 randomized individuals; 33 participants in ST and 35 in BATS. Groups did not differ on any demographic variable or baseline characteristics (see Table 1). Quit date was assigned to occur at session 4 of both treatment conditions. Assessments were conducted at each treatment session and follow-ups were conducted at 16 and 26 weeks post assigned quit date (i.e., 12 and 22 weeks post end of the 8 week treatment protocol). Assessments were conducted by research assistants blinded to treatment condition.

Standard Treatment (ST)—ST included eight, one-hour weekly group sessions. Participants began transdermal nicotine patch (TNP) on the scheduled quit date with an initial dose of 21 mg for 4 weeks, followed by 2 weeks of 14 mg, and 2 weeks of 7 mg. Participants who smoked on average 10-12 cigarettes per day started with the 14-mg patch for the first 6 weeks, per manufacturer's recommendations. Content was based on the most recent USDHHS clinical practice guidelines (Fiore et al., 2000) and included self-monitoring, identifying effective and ineffective cessation strategies from prior quit attempts, relaxation, coping with triggers, identifying social support for cessation, making lifestyle changes such as increasing physical activity and reducing stress, relapse prevention, and homework.

Behavioral Activation Treatment for Smoking (BATS)—BATS also included TNP and was comprised of 30 minutes of BA (adapted from Lejuez et al., 2001) and 30 minutes of core ST components and content described in the previous paragraph, excluding relaxation strategies. Relaxation strategies were not included in BATS because relaxation has been documented to be neither effective nor iatrogenic for smoking cessation (Fiore et al., 2008). Thus, removal from BATS but retention in ST was useful for equating contact time without unduly harming either condition. Specific to the BA content, Session 1 began with the therapist providing the treatment rationale focused on structuring a variety of reinforcing activities to promote a more rewarding non-smoking lifestyle. The therapist also introduced activity monitoring which involved recording of all daily activities as well as associated mood and smoking at these times. Completion of daily activity monitoring was assigned to occur each day of the following week. At the start of Session 2, the therapist led the group in a review of daily activity monitoring completed for each day of the previous week. Next, participants identified their values and life goals which were used to identify important and/or enjoyable activities. Several activities were then planned for the coming week using the behavioral checkout form, which allowed participants to track their activities and progress towards achieving weekly goals. For homework, participants were instructed to record engagement in each planned activity (Lejuez et al., 2001). Using BA strategies for engagement in activities as part of a nonsmoking lifestyle was encouraged. Sessions 3-8 focused on the behavioral checkout form starting with monitoring of planned activities from the previous week and then planning of activities for the coming week. Session 3 included a focus on activities related to quit preparation. Session 4 focused on quit-related activities, and Sessions 5-8 focused on activities consistent with remaining abstinent and addressing lapses in the larger context of their specific values and life goals. Participants were encouraged to use the monitoring and planning in BA to incorporate activities consistent with the standard smoking cessation strategies, including non-smoking lifestyle and coping with triggers.

Therapists—Therapists were four females and one male; two had clinical psychology doctoral degrees and three were clinical psychology doctoral students. Training for both conditions included a 4 hour workshop, followed by scheduled practice and observation of a full group. Weekly supervision was conducted for all therapists, which included review of therapy audiotapes. Each therapist provided treatment in both conditions. All therapists conducted at least one group in both conditions and no therapist conducted more than 3 groups in any condition. Sessions were audiotaped and a random 20% were rated by independent raters to assess therapist adherence to the protocol, using separate rating checklists and scales developed for the BATS and ST protocols. Adherence rates were over 95% for both treatment conditions.

Measures

At the baseline interview, participants provided demographic and other background information including age, gender, ethnicity, marital status, income level, employment status, and education level. Duration of previous quit attempts, age of onset of regular smoking and recent smoking behavior were assessed. *Motivation to quit* was assessed via a single item “On a 10 point scale where 1 is the lowest and 10 is the highest, please rate your current motivation to quit.” Nicotine dependence was assessed with the 6-item *Fagerström Test for Nicotine Dependence* (FTND; Heatherton, Kozlowski, Frecker, & Fagerström, 1991) with higher scores indicating greater nicotine dependence. The *Beck Depression Inventory-II* (BDI-II; Beck, Steer, & Brown, 1996) was used to assess current elevations in depressed mood. BDI-II scores ranging from 0-13 are indicative of minimal depression and from 14-19 are indicative of mild depression (Beck et al., 1996). Finally, the *Environmental Reward Observation Scale* (EROS; Armento & Hopko, 2007) measured the extent to which

individuals experienced pleasure from daily activities, and was used as the primary measure of behavioral activation. It includes 10 items (e.g., "A lot of activities in my life are pleasurable") rated on a 4 point likert scale, with higher scores indicating greater environmental reward. Internal consistency of the EROS ($\alpha = .86$) was established among adults ($M = 29.6$; $SD = 4.9$; Armento & Hopko, 2007), as well as its sensitivity to BA-based interventions among depressed substance users (Daughters et al., 2008).

Smoking outcome—was point prevalence abstinence defined as self-reported abstinence of ≥ 7 days prior to an assessment point. Smoking was assessed at 1 week-, 4 weeks- (end of behavioral treatment), 16 weeks-, and 26 weeks- post assigned quit date. Self-reported abstinence was verified via expired carbon monoxide. In addition, saliva samples for cotinine analysis were collected at the 16- and 26- week follow-up points where verification of abstinence required a combination of CO ≤ 10 ppm and cotinine ≤ 15 ng/ml (SRNT Subcommittee on Biochemical Verification, 2002). *Continuous abstinence*, defined as the length of time from quit day until the end of follow-up period in which the participant reported no smoking. Of those who attended at least one session of treatment ($n = 42$), biochemically-verified smoking data was obtained for 78.6%, 83.3%, 61.9%, and 64.3% of participants at 1-, 4-, 16- and 26- weeks post assigned quit date. Although rates are lower than typically reported in smoking cessation trials, they are consistent with rates in largely low income and minority samples (cf. El-Khorazaty et al., 2007). The proportion of participants who completed each assessment point was unrelated to treatment condition (*p*s >.25). Only those individuals whose smoking status was biochemically verified were considered abstinent at each time point, while those with missing data were considered as having smoked (Hughes et al., 2003). No subsequent follow-up data was obtained for the 26 participants who dropped prior to treatment and thus all data were coded as having smoked. One participant in BATS died between the 16 and 26-weeks; this death was unrelated to study participation. Unlike other missing data, this 26-week follow-up was retained as missing.

Data Analysis

Repeated measures analyses were conducted with generalized estimating equations (GEEs) to test group differences in the odds of being abstinent at 1-, 4-, 16- and 26- weeks post assigned quit date for both the full randomized sample ($n = 68$) and the sub-sample attending at least one treatment session ($n = 42$). Included covariates were gender, nicotine dependence, BDI-II symptoms, and current income, all of which are commonly linked to poor cessation outcomes (c.f., Cinciripini et al., 2003). We also included a linear effect of time. Hierarchical linear modeling (HLM) analyses were conducted for the sub-sample ($n = 42$) to examine treatment group differences in depressive symptoms and EROS scores, from baseline across the same time periods as the abstinence outcomes. In these analyses, we covaried gender, treatment condition, smoking status at each time point as a time-varying covariate, and the linear effect of time.

Results

The 68 randomized participants and the subsample who completed at least one session ($n = 42$) did not differ by gender, age, ethnicity, employment, income, or education (all *p*s >.15). Additionally, there were no differences on baseline and smoking characteristics between treatment conditions for either sample. See Table 1 for descriptives of the 68 participants.

In GEE analyses for seven-day point prevalence abstinence for the randomized sample ($n = 68$), BATS had significantly greater odds of abstinence across the follow-up period compared to ST (Table 3). A significant linear effect of time indicated abstinence rates

generally decreased from 1 week- through 26 weeks- post assigned quit date. Female gender and higher baseline BDI score were associated with lower odds of abstinence. The interaction between treatment condition and the linear effect of time was nonsignificant, indicating no differential effects of treatment across time. Continuous abstinence rates did not differ across treatment conditions (5.7% in BATS vs. 0% in ST; $p = .11$). There were no group differences in use of TNP or in bio-verified smoking data during treatment or the follow-up period ($ps > .20$). Results of the GEE model examining the main treatment effect on seven-day point prevalence for the sub-sample (n = 42) were similar, although gender and mood did not significantly predict smoking status.

In the HLM model predicting depressive symptoms, the linear effect of time on depressive symptoms was significant ($B = -1.53$, $SE = .68$, $p = .03$), indicating a reduction in depressive symptoms from baseline through the 26 weeks post assigned quit date (see Table 4). However, an interaction between treatment condition and the linear effect of time ($B = -1.99$, $SE = .86$, $p = .02$) revealed that the reduction in depressive symptoms over time was greater for BATS than ST participants (Figure 2). All EROS analyses were nonsignificant.

Discussion

BATS added to standard smoking cessation resulted in greater odds of point prevalence abstinence than ST across the 6-month follow-up period among smokers with elevated depressive symptoms. Additionally, depressive symptoms were lower throughout the follow-up period for BATS participants while slightly increasing over time for ST participants. Sample size prevented testing depressive symptoms as a mediator of the time by treatment effect, but results suggest potential viability of such analyses in future work. The lack of a treatment effect on EROS score was unexpected. Future efforts should aim to determine if the short assessment time frame here is sufficient to capture changes in pleasure that may be slower to occur than overt behavior change, as well as to include the additional assessment of actual activity engagement which is likely to change more quickly than pleasure derived from activities.

There are several study limitations. First, our control condition did not include another psychosocial depression treatment or an antidepressant medication. Second, recruitment issues raise some concerns regarding internal validity. Specifically, the refusal rate was high, although consistent with those seen in other randomized controlled trials of combined behavioral therapy plus pharmacotherapy for smoking cessation in samples with either history or current depressive syndromes (Evins et al., 2008; Spring et al., 2007). Additionally, there was differential pre-treatment attrition across the two treatment conditions. Participants were unaware of treatment assignment prior to the first session, and project staff were blinded to treatment condition during both the telephone screening and the baseline assessments. Thus, this differential attrition likely reflects random chance. The effects of treatment were significant and of similar magnitude even when examining only those participants who attended at least one session. Balancing these limitations, a strength of this work is the ethnic and socioeconomic diversity of the sample (75% ethnic minority and 37% of low income), given the historic under-representation of low income and ethnic minority participants in clinical trials for smoking cessation, but it is notable that generalizability to the larger population of smokers remains unclear.

Results indicate that BATS is a promising intervention for smoking cessation and the reduction of depression among smokers with elevated depressive symptoms. Given its brief nature, it may be a viable option for smoking cessation efforts across multiple settings and populations. This sets the stage for future work to replicate and extend these findings to quantify change in behavioral activation, as well as more comprehensive efforts to control

for key variables such as psychotropic medication utilization, as well as BA treatment acceptability, homework completion, and group cohesion. Future work would benefit from efforts to identify moderators of treatment effects, including biological variables including stress response and reward sensitivity.

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CONSORT Statement 2001 - Checklist

***PAPER SECTION* And topic**	**Item**	**Descriptor**	**Reported on Page #**
TITLE & ABSTRACT	1	How participants were allocated to interventions (*e.g.*, “random allocation”, “randomized”, or “randomly assigned”).	1-2
INTRODUCTION Background	2	Scientific background and explanation of rationale.	3
METHODS Participants	3	Eligibility criteria for participants and the settings and locations where the data were collected.	4
Interventions	4	Precise details of the interventions intended for each group and how and when they were actually administered.	5-6
Objectives	5	Specific objectives and hypotheses.	3-4
Outcomes	6	Clearly defined primary and secondary outcome measures and, when applicable, any methods used to enhance the quality of measurements (*e.g.*, multiple observations, training of assessors).	7-8
Sample size	7	How sample size was determined and, when applicable, explanation of any interim analyses and stopping rules.	4-5
Randomization -- Sequence generation	8	Method used to generate the random allocation sequence, including details of any restrictions (*e.g.*, blocking, stratification)	4
Randomization -- Allocation concealment	9	Method used to implement the random allocation sequence (*e.g.*, numbered containers or central telephone), clarifying whether the sequence was concealed until interventions were assigned.	4
Randomization -- Implementation	10	Who generated the allocation sequence, who enrolled participants, and who assigned participants to their groups.	--
Blinding (masking)	11	Whether or not participants, those administering the interventions, and those assessing the outcomes were blinded to group assignment. If done, how the success of blinding was evaluated.	5
Statistical methods	12	Statistical methods used to compare groups for primary outcome(s); Methods for additional analyses, such as subgroup analyses and adjusted analyses.	9
RESULTS Participant flow	13	Flow of participants through each stage (a diagram is strongly recommended). Specifically, for each group report the numbers of participants randomly assigned, receiving intended treatment, completing the study protocol, and analyzed for the primary	4-5; CONSORT diagram p.20

PAPER SECTION And topic	Item	Descriptor	Reported on Page #
		outcome. Describe protocol deviations from study as planned, together with reasons.	
Recruitment	14	Dates defining the periods of recruitment and follow-up.	4
Baseline data	15	Baseline demographic and clinical characteristics of each group.	Table 1, p. 16
Numbers analyzed	16	Number of participants (denominator) in each group included in each analysis and whether the analysis was by "intention-to-treat". State the results in absolute numbers when feasible (*e.g.*, 10/20, not 50%).	4-5; CONSORT diagram p.20
Outcomes and estimation	17	For each primary and secondary outcome, a summary of results for each group, and the estimated effect size and its precision (*e.g.*, 95% confidence interval).	9-10; Tables 2, 3, 4, p.17-19
Ancillary analyses	18	Address multiplicity by reporting any other analyses performed, including subgroup analyses and adjusted analyses, indicating those pre-specified and those exploratory.	9-10
Adverse events	19	All important adverse events or side effects in each intervention group.	5
DISCUSSION Interpretation	20	Interpretation of the results, taking into account study hypotheses, sources of potential bias or imprecision and the dangers associated with multiplicity of analyses and outcomes.	10-11
Generalizability	21	Generalizability (external validity) of the trial findings.	11
Overall evidence	22	General interpretation of the results in the context of current evidence.	10-11

References

Armento MEA, Hopko DR. The Environmental Reward Observation Scale (EROS): Development, Validity, and Reliability. Behavior Therapy. 2007; 38:107–119. [PubMed: 17499078]

Beck, AT.; Steer, RA.; Brown, G. Beck Depression Inventory II manual. San Antonio, TX: The Psychological Corporation; 1996.

Cinciripini PM, Wetter DW, Fouladi RT, Blalock JA, Carter BL, Cinciripini LG, et al. The effects of depressed mood on smoking cessation: Mediation by postcessation self-efficacy. Journal of Consulting and Clinical Psychology. 2003; 71:292–301. [PubMed: 12699023]

Cook JW, Spring B, McChargue D, Hedeker D. Hedonic capacity, cigarette craving, and diminished positive mood. Nicotine & Tobacco Research. 2004; 6(1):39–47. [PubMed: 14982686]

El-Khorazaty MN, Johnson AA, Kiely M, El-Mohandes AAE, Subramanian S, Laryea HA, et al. Recruitment and retention of low-income minority women in a behavioral intervention to reduce smoking, depression, and intimate partner violence during pregnancy. BMC Public Health. 2007; 7:233–250. [PubMed: 17822526]

Evins AE, Culhane MA, Alpert JE, Pava J, Liese BS, Farabaugh A, et al. A controlled trial of bupropion added to nicotine patch and behavioral therapy for smoking cessation in adults with unipolar depressive disorders. Journal of Clinical Psychopharmacology. 2008; 28:660–666. [PubMed: 19011435]

Fiore, MC.; Bailey, WC.; Cohen, SJ., et al. Treating Tobacco Use and Dependence: Clinical Practice Guideline. Rockville, MD: U. S.: Department of Health and Human Services. Public Health Service; 2000.

First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JBW. Structured Clinical Interview for DSM-IV Axis I Disorders. New York: New York State Psychiatric Institute; 1995.

Heatherton TF, Kozlowski LT, Frecker RC, Fagerstrom KO. The Fagerstrom test for nicotine dependence: A revision of the Fagerstrom Tolerance Questionnaire. British Journal of Addiction. 1991; 86:1119–1127. [PubMed: 1932883]

Hughes JR, Keely JP, Niaura RS, Ossip-Klein DJ, Richmond RJ, Swan GE. Measures of abstinence in clinical trials: Issues and recommendations. Nicotine and Tobacco Research. 2003; 5:13–26. [PubMed: 12745503]

Jacobson NS, Dobson KS, Truax PA, Addis ME. A component analysis of cognitive-behavioral treatment for depression. Journal of Consulting and Clinical Psychology. 1996; 64:295–304. [PubMed: 8871414]

Kahler CW, Brown RA, Ramsey SE, Niaura R, Abrams DB, Goldstein MG, et al. Negative mood, depressive symptoms, and major depression after smoking cessation treatment in smokers with a history of major depressive disorder. Journal of Abnormal Psychology. 2002; 4:670–675. [PubMed: 12428781]

Lejuez CW, Hopko DR, Hopko SD. A brief behavioral activation treatment for depression: Treatment Manual. Behavior Modification. 2001; 25:255–286. [PubMed: 11317637]

Leventhal AM, Ramsey SE, Brown RA, LaChance HR, Kahler CW. Dimensions of depressive symptoms and smoking cessation. Nicotine & Tobacco Research. 2008; 10:507–517. [PubMed: 18324570]

McCarthy DE, Piasecki TM, Lawrence DL, Jorenby DE, Shiffman S, Baker TB. Psychological mediators of bupropion sustained-release treatment for smoking cessation. Addiction. 2008; 103:1521–1533. [PubMed: 18783504]

Niaura R, Britt DM, Shadel WG, Goldstein M, Abrams D, Brown R. Symptoms of depression and survival experience among three samples of smokers trying to quit. Psychology of Addictive Behaviors. 2001; 15:13–17. [PubMed: 11255933]

Piper ME, Federman EB, McCarthy DE, Bolt DM, Smith SS, Fiore MC, et al. Using mediational models to explore the nature of tobacco motivation and tobacco treatment effects. Journal of Abnormal Psychology. 2008; 97:94–105. [PubMed: 18266488]

Pizzagalli DA, Jahn AL, O'Shea JP. Toward an Objective Characterization of an Anhedonic Phenotype: A Signal-Detection Approach. Biological Psychiatry. 2005; 57:319. [PubMed: 15705346]

Spring B, Cook JW, Appelhans B, Maloney A, Richmond M, Vaughn J, et al. Nicotine effects on affective response in depression-prone smokers. Psychopharmacology (Berl). 2008; 196:461–471. [PubMed: 17960366]

Spring B, Doran M, Pagoto S, McChargue D, Cook JW, Bailey K, et al. Fluoxetine, smoking and history of major depression: A randomized controlled trial. Journal of Consulting and Clinical Psychology. 2007; 75:85–94. [PubMed: 17295567]

SRNT Subcommittee on Biochemical Verification. Biochemical verification of tobacco use and cessation. Nicotine and Tobacco Research. 2002; 4:149–159. [PubMed: 12028847]

Thorndike AN, Regan S, McKool K, Pasternak RC, Swartz S, Torres-Finnerty N, et al. Depressive symptoms and smoking cessation after hospitalization for cardiovascular disease. Archives of Internal Medicine. 2008; 168:186–191. [PubMed: 18227366]

Figure 1.
CONSORT flowchart of study participants, randomization, treatment, follow-ups, and inclusion analyses. ST = standard smoking cessation treatment including nicotine replacement therapy (NRT); BATS = ST and NRT that integrates behavioral activation strategies.

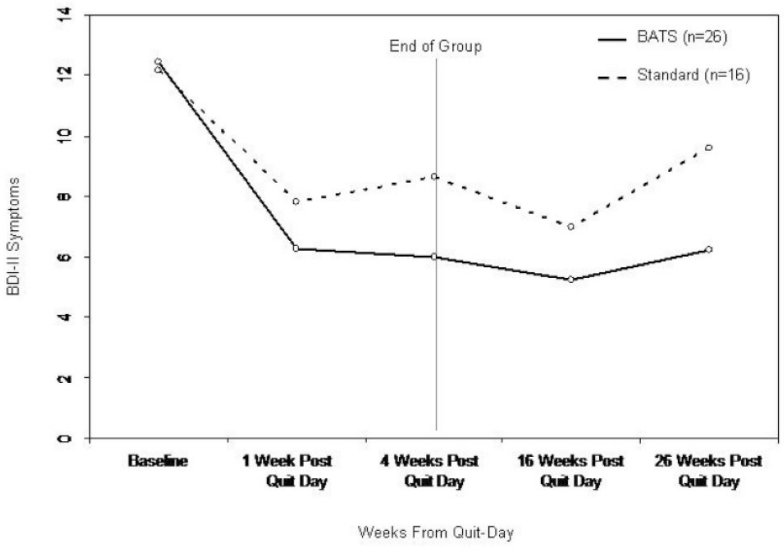

Figure 2.
Changes in BDI-II symptoms for BATS and ST Conditions in the Sub-sample (n = 42).
Figure 2 Note: 1 week post quit day = week 4 of behavioral treatment; 4 weeks post quit day = week 8 and end of behavioral treatment; 16 weeks post quit day = 12 weeks from end of behavioral treatment; 26 weeks post quit day = 22 weeks from end of behavior treatment.

Table 1

Comparisons on baseline demographic, smoking history, and affective variables across treatment condition.

Characteristics	BATS (*n* = 35)	ST (*n* = 33)	*P*
Demographic Variables			
Age, mean (SD)	45.0 (12.2)	42.6 (11.5)	.40
Gender, % female	48.6	48.5	.99
Ethnicity, % African-American	69.7	75.8	.34
Employment Status, (% employed)	54.8	58.6	.96
Education (%)			
HS Graduate/GED or lower	21.2	24.4	.24
Some College/Tech School/College	78.8	65.6	
Household Income (%)			
$0-$49,9999	63.3	61.5	.99
$50,000-$99,999	30.0	30.8	
$100,000+	6.7	7.7	
Smoking History Variables, (Mean (SD))			
Smoking history in years	24.1 (12.7)	23.2 (14.0)	.79
Nicotine dependence (FTND)	5.8 (1.8)	6.1 (2.1)	.49
Average Cigarettes Per Day	18.8 (7.1)	17.3 (8.1)	.44
Number of prior quit attempts	3.6 (2.4)	4.3 (4.1)	.39
Motivation to quit	8.4 (1.7)	8.8 (1.3)	.28
Affective Variables			
BDI score, mean (SD)	10.8 (5.2)	10.4 (7.5)	.80
EROS score, mean (SD)	26.6 (3.6)	26.5 (4.8)	.89

Table 2

Abstinence rates across treatment conditions for randomized sample and for sub-sample who completed one treatment session

	Randomized Sample (*n* = 68)			Sub-Sample (*n* = 42)		
	ST (*n* = 33)	**BATS (*n* = 35)**	**OR (95% CI)**	**ST (*n* = 16)**	**BATS (*n* = 26)**	**OR (95% CI)**
Point Prevalence Abstinence (%)						
1-week post quit	9.1	28.6	4.00	18.8	38.5	2.71
4-week post quit	9.1	17.1	2.06	18.8	23.1	1.30
16-week post quit	3.0	11.4	2.71	6.3	15.4	2.71
26-week post quit	0.0	14.3	*	0.0	19.2	*

Note.

* Odds ratios were not computed for the 26-week post quit date due to the 0% abstinence in ST.

Table 3

Generalized Estimation Equations Analyses Predicting 7-Day Point Prevalence Smoking Abstinence at 1, 4, 16, and 26 Weeks Post Quit Date for Randomized Sample (n = 68)

Variable	*OR*	95% CI	*p*
Main effects			
Time	0.42	0.21, 0.87	.02
Female Gender	0.27	0.08, .95	.04
FTND	0.79	0.54, 1.14	.21
BDI-II score	1.11	1.02, 1.21	.01
Household Income	1.23	0.65, 2.33	.53
BATS compared with ST	3.59	1.22, 10.53	.02
BATS X Time	1.64	0.72, 3.73	.24

Note. *N* = 68; ORs < 1 indicate reduced odds of abstinence; ORs > 1 indicate increased odds of abstinence. All continuous variables were centered. OR = odds ratio; CI = confidence interval.

Table 4

Hierarchical Linear Modeling (HLM) Analyses Predicting Depressive Symptoms (BDI-II scores) at Baseline and 1, 4, 16, and 26 Weeks Post Quit Date for Sub-Sample (n = 42)

Variable	*B*	*SE*	*t*	*p*
Main effects				
Time	-1.53	0.68	-2.23	.03
Female Gender	0.87	1.53	0.57	.58
Smoking Status	1.89	1.01	1.88	.07
BATS compared with ST	-2.54	1.83	-1.44	.17
BATS X Time	-1.99	0.86	-2.31	.02

Note. *n* = 42; All continuous variables were centered.