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Behavioral activation for smoking cessation and the prevention of smoking cessation-related weight gain: A randomized trial☆

Janet Audrain-McGovern [a,*], E. Paul Wileyto [c], Rebecca Ashare [b], Benjamin Albelda [a], Divya Manikandan [a], Kenneth A. Perkins [d]

[a] Department of Psychiatry, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA, USA
[b] Department of Psychology, University at Buffalo, State University of New York, Buffalo, NY, USA
[c] Department of Biostatistics and Epidemiology, University of Pennsylvania, Philadelphia, PA, USA
[d] University of Pittsburgh Medical Center, University of Pittsburgh, Pittsburgh, PA, USA

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ABSTRACT

Background: Post-cessation weight gain (PCWG) is an obstacle to smoking cessation. This trial evaluated a behavioral intervention targeting alternative rewards to smoking and high calorie snacking to promote smoking cessation while mitigating PCWG.

Methods: Adult smokers (n = 288; 119 females, 169 males) received eight weeks of transdermal nicotine and were randomized to eight sessions of behavioral activation for smoking cessation and the mitigation of PCWG (BAS+) or standard smoking cessation counseling (SC). Primary outcomes were 7-day point prevalence abstinence and PCWG 26 weeks after the target quit date. Change in caloric intake from pre-treatment through the 26-week follow-up was a secondary outcome. Data were collected from September 2016 to February 2021, and analyses were completed in July 2022.

Results: BAS+ and SC did not differ in smoking abstinence rates at the 26-week follow-up (OR=0.80, 95%CI 0.50–1.27, p = 0.34; 18% versus 23%). There were no significant differences in PCWG between BAS+ and SC who were 7-day point prevalence abstinent (β = −0.29, 95%CI −2.13 to 1.65, p = 0.77; 2.60 versus 2.20 pounds, respectively) or among those continuously abstinent (5.78 versus 5.34 pounds, respectively). There were no significant differences in caloric intake between BAS+ and SC from baseline to the 26-week follow-up (β = 110.65, 95%CI −96.72 to 318.02, p = 0.30; −19.1 versus −116.9 kcals/day, respectively).

Conclusions: The results do not support the efficacy of BAS+ for smoking cessation and the prevention of PCWG. These findings join a growing body of research highlighting the challenge of minimizing PCWG and promoting smoking abstinence.

1. Introduction

An estimated 31 million adults in the US smoke cigarettes, a significant contributor to premature morbidity and mortality (Cornelius et al., 2022). Weight gain as a consequence of smoking cessation, referred to as post-cessation weight gain (PCWG), can deter a quit attempt, promote smoking relapse, and contribute to health issues related to excess body weight (Audrain-McGovern and Benowitz, 2011; Hartmann-Boyce et al., 2021). PCWG has been attributed primarily to increased caloric intake due to between-meal snacking on foods high in fat and sugar (Perkins, 1993). The majority of weight gain, about 8–11 pounds on average, occurs within 3–6 months of quitting smoking (Aubin et al., 2012; Klesges et al., 1997), with many former smokers continuing to gain weight in the years following cessation (Lycett et al., 2011; Veldheer et al., 2015). Although the mortality rates for overweight ex-smokers may be less than those for normal-weight smokers (Siahpush et al.,

Abbreviations: PCWG, Post-cessation weight gain; BAS+,, behavioral activation for smoking cessation and prevention of PCWG; SC, standard smoking cessation counseling; TN, transdermal nicotine; BMI, body mass index; CO, carbon monoxide; TQD, target quit day; GEE, general estimating equations; OR, odds ratio; CI, confidence interval.

☆ Trial Registration: The trial is registered as NCT02906787 at www.clinicaltrials.gov.

* Correspondence to: Department of Psychiatry, Perelman School of Medicine, University of Pennsylvania, 3535 Market St., Suite 4100, Philadelphia, PA 19104, United States of America.

E-mail address: audrain@pennmedicine.upenn.edu (J. Audrain-McGovern).

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2014), most smokers find the average weight gain associated with quitting smoking intolerable and a reason to resume smoking (Levine, 2013; Pisinger and Jorgensen, 2007).

Over two decades of treatment research to prevent PCWG has yielded modest success at best (Hartmann-Boyce et al., 2021). Interventions targeting diet and exercise have been ineffective at preventing PCWG, while having modest long-term effects on smoking cessation (Spring et al., 2009). Smoking and eating share common reward mechanisms, thus reducing one of these behaviors can lead to compensatory increases in the other (Audrain-McGovern and Benowitz, 2011; Blum et al., 2011; Blumenthal and Gold, 2010; Kenny, 2011; Olsen, 2011). Novel theory-driven behavioral interventions that consider the mechanisms that underlie increased food intake following smoking cessation may reduce weight gain and smoking relapse. Behavioral Economic Theory suggests that PCWG may stem, in part, from the reward deficit produced by smoking cessation. Upon quitting, smokers lose a significant reinforcer (Caggiula et al., 2009; Chaudhri et al., 2006), have fewer alternative reinforcers from which to choose (Audrain-McGovern and Benowitz, 2011; Audrain-McGovern et al., 2009), and may experience less pleasure from the available reinforcers after quitting smoking (Audrain-McGovern et al., 2014; Dawkins et al., 2007).

Compensatory increases in between-meal snacking on foods high in fat and sugar (Perkins, 1992, 1993; Perkins et al., 1990) may offset the reward deficit due to quitting smoking. Highly palatable snack food is a readily available reinforcer that shares common reward mechanisms with nicotine (Audrain-McGovern and Benowitz, 2011; Blum et al., 2011; Volkow et al., 2008). Behavioral Economic Theory indicates that the reinforcing value of snacking can be enhanced, or reduced, based on the availability of alternative reinforcers (Bickel, 2014; Madden, 2000). Fewer alternative reinforcers and a reduction in pleasure derived from available reinforcers forge an over-reliance on palatable snack foods to substitute for the reinforcement previously derived from cigarettes. Indeed, research has shown that smoking cessation increases the reinforcing value of snack foods, which predicts subsequent food intake and weight gain (Epstein et al., 2004; Lerman et al., 2004).

To avoid smoking cessation-induced increases in food intake and weight gain that precipitate smoking relapse, the present study evaluated a novel application of a behavioral activation intervention to smoking cessation and PCWG (BAS+), which focused on increasing opportunities for reinforcement (Lejuez et al., 2011; MacPherson et al., 2010) and enhancing the pleasure obtained from typical reinforcers (Duckworth et al., 2005; Seligman et al., 2006) versus standard smoking cessation counseling (SC). The primary outcomes were biochemically verified 7-day point prevalence abstinence and PCWG 26 weeks after the target quit date. Change in food intake was a secondary outcome. We hypothesized that participants randomized to BAS+ (versus SC) would have higher smoking cessation rates, less post-cessation food intake, and gain less weight at week 26.

2. Methods

2.1. Study sample

Participants were 288 treatment-seeking smokers 18–65 years old who reported smoking > 5 cigarettes per day for the past 6 months (verified by carbon monoxide (CO) > 8 ppm). Exclusion criteria were: current use of other nicotine products or smoking cessation medications, an unstable medical condition (e.g., recent cardiovascular event, cancer diagnosis), pregnancy or breastfeeding, a diagnosis that contraindicated nicotine patch use (e.g., latex allergy), use of a contra-indicated medication (e.g., prescription stimulants, opiate medications), a current self-reported psychiatric condition (e.g., psychoses, illicit substance use, substance use treatment in past 12 months, alcohol consumption > 25 standard drinks a week) or low intellectual functioning (estimated IQ < 85 Shipley Institute of Living Scale) (Zachary, 2000) that could hamper informed consent or participation in the behavioral counseling. An initial telephone screen assessed most of these exclusion criteria with an in-person screen to verify final eligibility (see 2.2. below). Participants were recruited from the Philadelphia area through print and media advertisements. The study was located at the University of Pennsylvania by the Institutional Review Board. Recruitment and enrollment were initiated on September 13, 2016 and follow-up was completed on February 28, 2021. The trial profile is summarized in Fig. 1.

2.2. Procedures

Participants who were eligible based on telephone screening completed an in-person eligibility screening to document a negative urine drug screen, a breath alcohol test < .000, an expired breath CO reading (> 8 ppm) to confirm smoking status, a negative urine pregnancy test, blood pressure measurement (< 160 systolic and < 100 diastolic), and a Shipley screen of intellectual functioning (>85). Those eligible returned for the baseline assessment of demographics, smoking, dietary intake, weight concerns, and alternative reinforcers two weeks later. These participants were randomized at the baseline visit. Randomization (1:1) of participants to smoking cessation counseling treatment was stratified by sex, body mass index (BMI >30), and nicotine dependence. Each stratum had a randomization stream made up of small, permuted blocks.

Participants were randomized to receive 8 individual BAS+ or SC sessions plus transdermal nicotine (TN) over 10 weeks (description below). Both treatments were manualized to ensure standardization of treatment delivery for each session. Treatment was delivered by trained counselors with a baccalaureate in psychology. Participants had two pre-quit smoking cessation counseling sessions, were provided with TN, and instructed to initiate TN use on the morning of the target quit day (TQD). Participants then had a counseling session on their TQD and at 1, 2, 4, 6, and 8 weeks post-TQD. All sessions were audio recorded to ensure counselor adherence to the treatment protocols. Substitute alternative reinforcers were assessed again at end of treatment. Food intake was measured by three 24-hour food recalls at baseline and weeks 4, 8, 12, and 26. Smoking status was assessed via self-report and biochemically verified (CO < 5 ppm) (Perkins et al., 2013) at each clinic visit, EOT (week 8), and follow-up (weeks 12 and 26). Weight was measured at these same time points. Staff (except counselors) were blind to treatment assignment.

2.2.1. Transdermal nicotine (TN)

All participants initiated a standard 8-week regimen of transdermal nicotine therapy (NicoDerm CQ; 21 mg x 4 weeks, 14 mg x 2 weeks, 7 mg x 2 weeks) on the morning of the target quit date, three weeks after the start of behavioral counseling, and continued through week 10. TN use was monitored throughout the treatment period using a timeline follow-back at each visit. Adherence was defined as using at least 6 patches, on average, per week (unused patches collected).

2.2.2. Standard smoking cessation counseling (SC)

SC content followed the best practices for smoking cessation counseling. The initial session began with a review of smoking and quitting history, reasons for smoking and quitting, triggers for smoking, and obtaining social support for quitting (Perkins, 2008). The second session focused on the management of smoking triggers, slip recovery, recurrence prevention, and TN use. The third session (TQD) focused on the quit day experiences given that participants were instructed to quit the morning of this session. Sessions 4 through 8 focused on reinforcing progress with cessation, problem-solving challenges, and recurrence prevention. Overeating and weight gain are common concerns reported during smoking cessation treatment. Per convention (e.g., NCI's Clearing the Air), SC addressed these concerns through standard recommendations to consume low-calorie snack foods, drink water, eat nutritious meals, and exercise.

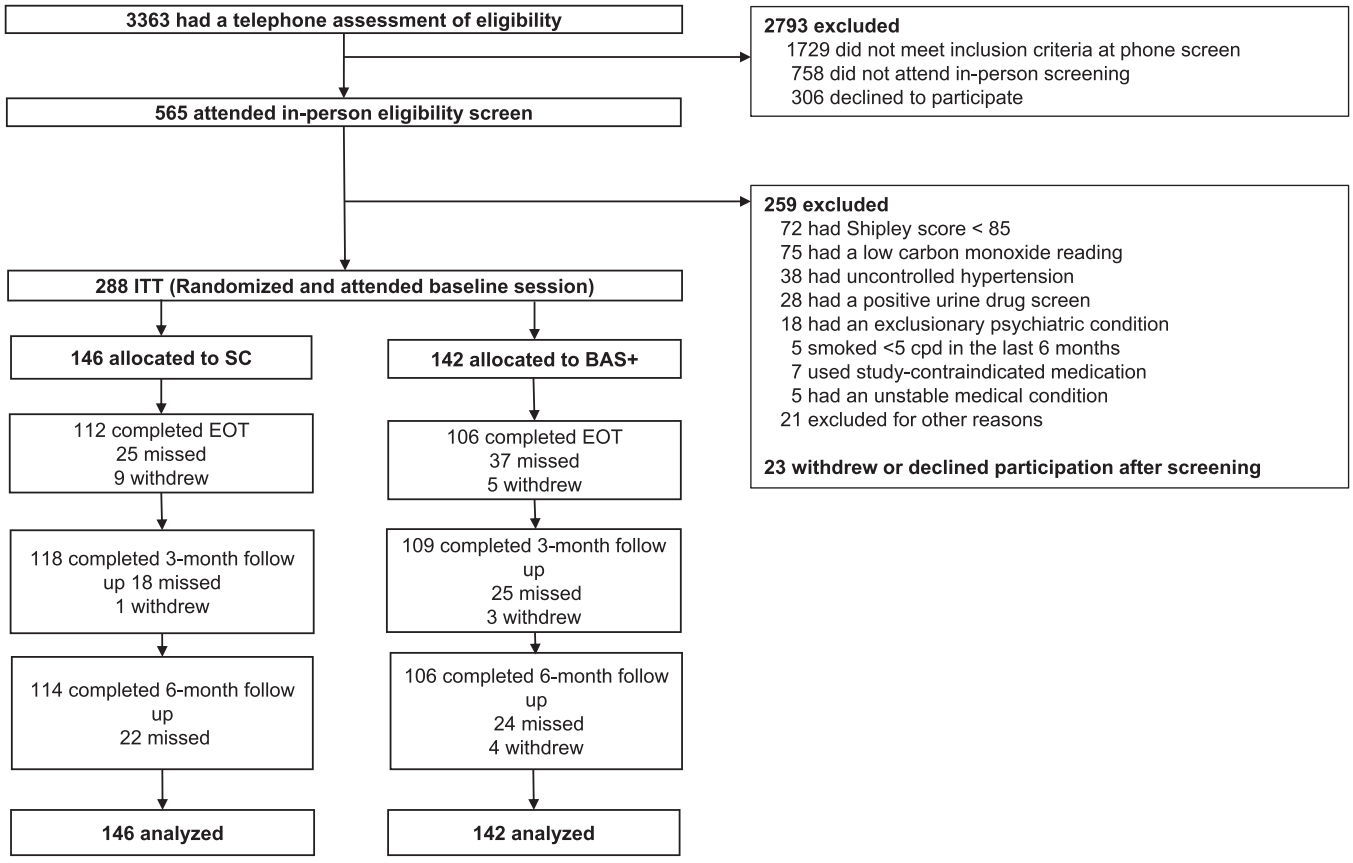

Fig. 1. Flow diagram of study participants, randomization, treatment, follow-ups and inclusion in analysis. ITT = intent-to-treat, SC = standard smoking cessation counseling, BAS+ = behavioral activation for smoking cessation and the minimization of post-cessation weight gain.

2.2.3. Behavioral activation (BAS+)

SC content was incorporated into BAS+ in an additive design to cover best practice guidelines for smoking cessation. The goal of BAS+ was to maintain a level of overall reward after cessation to ensure that not smoking is as reinforcing as smoking (Smith et al., 2001) and prevent an over-reliance on food as a substitute reinforcer for smoking so that PCWG does not precipitate smoking recurrence. The unique treatment components of BAS+ included facilitating the identification of and engagement in a variety of rewarding activities (other than eating and not associated with smoking) and maximizing the enjoyment derived from these rewarding activities.

The first session began with a discussion of why snacking substitutes for cigarettes after smoking cessation, and the consequences for weight gain and continued abstinence. The counselor provided the treatment rationale focused on structuring a variety of reinforcing activities to promote a more rewarding nonsmoking lifestyle (Correia, 2005; Green et al., 2000; Miller and Miller, 2009); one that has many rewarding options besides food and cigarettes (MacPherson et al., 2010; Pagoto et al., 2013). The counselor introduced daily activity monitoring to promote awareness of time spent in important and/or enjoyable activities and their association with smoking (MacPherson et al., 2010).

In the second session, the goals and experiences with daily activity monitoring were reviewed, noting the time spent in important and/or enjoyable activities. The counselor introduced the identification of values and life goals in several domains (e.g., health, recreation, personal growth, relationships) and assisted the participant in identifying rewarding activities within these life domains (e.g., physical activity, spending time with family, volunteering, hobbies) through a life activities checklist. Participants set daily goals for selecting and engaging in these activities (Lejuez et al., 2001) to begin to increase alternative reinforcers before quitting smoking and kept track of their daily progress with the daily activity monitoring form.

Given that nicotine can increase the pleasure derived from available reinforcers (Audrain-McGovern et al., 2014; Perkins and Karelitz, 2013), BAS+ included a component to build skills to enhance the pleasure derived from smoke-free and snack-free reinforcers. These types of "savoring" skills are effective at promoting long-term increases in positive emotions and life enjoyment (Duckworth et al., 2005; Seligman et al., 2005). At the end of each day, while rating the enjoyment level of planned activities or experiences, participants mentally re-visited three aspects that made these activities enjoyable.

Sessions 3 through 8 focused on daily activity monitoring, engagement, and planning consistent with quitting smoking and remaining abstinent. Based on successful engagement in selected activities, new and progressively challenging activities were added. Reward enhancement through savoring was reviewed. The sessions focused on reviewing successes and problem-solving difficulties, such as whether slips/smoking recurrence or over-snacking coincided with no alternative reinforcers or reinforcers not consistent with remaining abstinent. Smoking recurrence was considered within the context of available alternative reinforcers and the participant's specific values and life goals. Participants who began smoking again were directed to set another quit date, to re-evaluate their selection of alternative reinforcers for value and links to smoking and encouraged to revisit overall values and goals. The rationale for these core behavioral activation practices were reiterated from the standpoint of remaining abstinent, lessening PCWG, and achieving value-driven life goals.

2.2.4. Fidelity monitoring

The smoking cessation counselors were trained by the principal investigator to deliver SC and BAS+ using standardized procedures (e.g., audio recordings of smoking cessation counseling, completion of a

four-hour didactic educational sessions on the underpinnings of the behavioral economic-informed BAS+ counseling, mock sessions with research staff and PI, and shadowing and supervision of smoking cessation counseling delivery). Counselors were monitored and provided regular feedback on intervention delivery based on audio recordings. All sessions were audiotaped, and a random sample (25%) of the sessions was evaluated for adherence to the treatment protocol. Fidelity checklists were used to assess adherence to elements of the counseling protocol, intervention drift and contamination, session duration, and to provide counselor feedback. The standardized fidelity checklists for each intervention yielded a protocol adherence score (1 = not at all to 7 = extensively).

2.3. Measures

2.3.1. Outcome variables

Smoking abstinence (primary outcome) was assessed and biochemically verified at end-of-treatment (EOT, week 8), and 12 and 26 weeks after the target quit date (Hughes et al., 2003). A reliable and valid timeline follow-back method (Brown et al., 1998) was used to assess daily smoking (presence and rate). The primary smoking outcome variable was 7-day point prevalence abstinence (no smoking, not even a puff, for at least 7 days prior to the assessment) biochemically verified by CO < 5 ppm at EOT (week 8), and at the 12- and 26-week follow-ups (Hughes et al., 2003).

Weight gain (primary outcome) was measured by a digital scale (pounds, ounces) wearing light clothing without shoes prior to each session and at follow-up. Height was measured at baseline using a mounted stadiometer and BMI was calculated as weight (kg)/height $(m)^2$. Pre-cessation weight was computed as the average of weights at the intake and baseline sessions prior to any change in smoking behavior. Weight change from baseline to the 26-week follow-up served as the primary weight outcome variable.

Dietary intake (secondary outcome) was assessed via three telephone-administered, 24-hour dietary recalls at baseline, mid-treatment (week 4), EOT (week 8), and the 12- and 26-week follow-ups. The interviewer administered ASA24® is a multi-pass method with an interactive computerized software program developed by the National Cancer Institute, to determine total kcal/day. Three recalls are considered optimal for assessing dietary intake, especially when weekend and weekdays are assessed (Tran et al., 2000) as was done in this study.

2.3.2. Covariates and predictor variables

Demographics including race, sex, age, and income were self-reported at baseline. Nicotine dependence was measured by the Fagerstrom Test for Nicotine Dependence (FTND). The FTND is a 6-item measure with good internal consistency (α = .64) and high test-retest reliability (r = .88) (Heatherton et al., 1991). Weight concerns associated with quitting smoking were measured with a reliable (α = .87) and valid 6–item scale (Borrelli and Mermelstein, 1998; Sepinwall and Borrelli, 2004). These items were averaged for a total score (1 =not at all to 10 = very much). Substitute alternative reinforcers were measured at baseline and at the end of treatment (week 8) with the adapted Pleasant Events Schedule (Audrain-McGovern et al., 2014; Schnoll et al., 2016), designed to assess reinforcers that occur in the natural environment. The 78 items are rated once in terms of frequency (0 = none to 2 = often) and once in terms of enjoyability (0 = none to 2 = very) over a specified number of days, yielding a *frequency* score, an *enjoyability score*, and the cross product is the reinforcement from the activity. This measure is sensitive to changes in reinforcers across smoking cessation treatment (Goelz et al., 2014; Schnoll et al., 2016).

2.4. Statistical analyses

Statistical analyses were conducted in July, 2022 using Stata v17 software (Stata Corporation, College Station, Texas). The smoking abstinence outcome of 7-day point prevalence at the 26-week follow-up was binary. PCWG (pounds) and caloric intake (total kilocalories per day) were continuous. We assessed the randomization by testing for association of treatment assignment with demographic, smoking, and weight-related variables using t-tests or χ^2 as appropriate (See Table 1).

All of our outcome variables were repeated measures and were analyzed with longitudinal regression methods using generalized estimating equations (GEE). GEE regression provides flexibility in modeling various outcome families (Gaussian for weight related change scores, binomial for the binary smoking abstinence outcome) while at the same time accounting for various types of correlation structures within subject (in our case, exchangeable random effects). In order to test the hypothesis of treatment (BAS+ versus SC) effects on 6-month outcomes, our models included categorical effects of time (EOT, 12 weeks, and 26 weeks), and treatment (BAS+ versus SC). We also included terms for the time by treatment interaction, which were omitted in favor of main effects only if the overall interaction was not significant (tested by Wald χ^2). Models included covariates (e.g., sex, race, income) and smoking and weight-related predictors (e.g., nicotine dependence, weight concerns, substitute alternative reinforcers) to assess their effects on the outcome variables irrespective of treatment. Change in smoking rate was included as a covariate in the models of PCWG and caloric intake given smoking's effect on dietary intake and body weight (Audrain-McGovern and Benowitz, 2011).

For each model, adjusted and unadjusted results are reported. Hypotheses were tested at an overall 5% type-1 error, which was Bonferroni corrected for the two primary hypotheses (α = 0.025) involving smoking abstinence and PCWG. The sample of 288 provided 80% power to detect a 14.5% difference (13% versus 27.5%) in 7-day point prevalence abstinence at 26 weeks, and a 7.28-pound difference in post-cessation weight gain. With respect to missing data, an all-available data approach was followed and missing = smoking was assumed for

Table 1
Sample Characteristics at Baseline (N = 288).

Variable	All (N = 288) N (%)	SC (N = 146) N (%)	BAS (N = 142) N (%)	p value
Sex				0.69
Male	169 (58.7%)	84 (57.5%)	85 (59.9%)	
Female	119 (41.3%)	62 (42.5%)	57 (40.1%)	
Race				0.62
African American	171 (59.4%)	86 (58.9%)	85 (59.9%)	
White	101 (35.1%)	50 (34.2%)	51 (35.9%)	
Not reported	16 (5.6%)	10 (6.8%)	6 (4.2%)	
Education				0.95
HS Graduate or less	105 (36.5%)	54 (37.0%)	51 (35.9%)	
Some College	109 (37.8%)	54 (37.0%)	55 (38.7%)	
College Graduate	74 (25.7%)	38 (26.0%)	36 (25.4%)	
Income				0.26
< $20,000	99 (34.5%)	45 (31.0%)	54 (38.0%)	
$20,000 - $50,000	129 (44.9%)	72 (49.7%)	57 (40.1%)	
> $50,000	59 (20.6%)	28 (19.3%)	31 (21.8%)	
	Mean (SD)	**Mean (SD)**	**Mean (SD)**	
Age	45.99 (12.13)	45.97 (12.01)	46.01 (12.30)	0.98
Cigarettes per day	14.25 (7.01)	14.33 (7.61)	14.18 (6.36)	0.86
Weight (lbs)	191.57 (49.26)	191.43 (47.97)	191.71 (50.73)	0.96
Total kcals	2311.97 (907.43)	2319.30 (781.62)	2304.47 (1022.95)	0.89
Body mass index	29.44 (7.13)	29.35 (6.92)	29.53 (7.38)	0.83
Nicotine dependence	4.86 (1.88)	4.97 (1.79)	4.75 (1.97)	0.31
Substitute reinforcers	40.86 (30.14)	40.43 (32.80)	41.31 (27.25)	0.81
Weight concerns	4.85 (2.18)	4.98 (2.13)	4.72 (2.24)	0.31

the analyses of smoking abstinence. Attrition was nearly identical for BAS+ and SC across time. Covariate values were not significantly different between participants lost and those retained.

3. Results

3.1. Sample characteristics

We recruited 288 subjects to the trial, with 146 assigned to SC and 142 assigned to BAS+ . Forty-one percent of participants were female, 59% identified as African American and 35% as White, and 45% of the sample reported an annual income between 20 and 50 thousand dollars. On average participants were 46 years of age, moderately nicotine dependent (FTND = 4.86), smoked 14 cigarettes per day, weighed 192 pounds, BMI of 29.44, and consumed 2312 kcals per day. As noted in Table 1, there were no significant differences between the treatment groups for any of the variables. There were no significant treatment group differences in the number of counseling sessions attended (80.6% [SD = 2.3] for BAS+ and 81.4% [SD = 2.3] for SC, $p > 0.80$) or adherence to recommended transdermal nicotine patch use (74% BAS, 71% SC, $p = 0.53$). Retention through the 6-month follow-up was similar between treatment groups (75% BAS, 78% SC, $p = 0.50$).

3.2. Smoking abstinence

The overall model of smoking abstinence revealed a significant effect of time but no significant effects of treatment. The time by treatment interaction was not significant ($\chi^2(2) = 0.61$, $p = 0.74$), and thus we report main effects only. As noted in Table 2, the overall effect of treatment on smoking abstinence (uniform across end-of-treatment, 12-week and 26-week follow-up) was not significant, with a trend toward BAS+ participants abstaining less than SC participants (OR=0.80, 95% CI 0.50–1.27, $p = 0.34$). The quit rates for BAS+ at EOT, 12-weeks and 26 weeks were 30%, 26%, and 18%, respectively. The quit rates for SC at EOT, 12-weeks and 26 weeks were 36%, 29%, and 23%, respectively.

The significant effect of time-point indicated that quit rates declined over time, as expected (OR at 26 weeks = 0.55, 95%CI 0.42–0.70, $p < .0001$). White participants (OR = 1.96, 95%CI 1.22–3.17, $p = 0.006$) and those with a higher annual income (OR = 3.34, 95%CI 1.79–6.22, $p = .0002$) had increased odds of being abstinent at 26 weeks, while more nicotine dependent participants had lower odds (OR = 0.83, 95%CI 0.73–0.94, $p = .004$). Participants who increased their substitute alternative reinforcers by one standard deviation from baseline had increased odds of being smoking abstinent at the 26-week follow-up (OR = 1.25, 95%CI 1.06–1.47, $p < .007$).

3.3. Post-cessation weight gain

Over the course of the trial, both groups gained weight. BAS participants who reported a 7-day point prevalence abstinence at the 26-week follow-up gained 2.6 pounds, while SC participants gained 2.2 pounds. BAS+ participants who were continuously abstinent since quit day gained 5.78 pounds and SC participants gained 5.34 pounds. The linear mixed models revealed that the time by treatment interaction was not significant ($\chi^2(2) = 1.69$, $p = 0.43$), and the main effects model revealed no significant effects of time ($p = 0.85$) or treatment. As noted in Table 3, the remaining overall effect of treatment on PCWG was not significant ($\beta = -0.29$, 95%CI: −2.23 to 1.65, $p = 0.77$). Participants with weight concerns gained 0.61 pounds additional for each 1-point increase of the weight concerns scale ($\beta = 0.61$, 95%CI: 0.16–1.07, $p = 0.008$). Among participants who were still smoking, for every 1 cigarette per day increase above their baseline smoking rate, they lost 3.26 pounds ($\beta = -3.26$, 95%CI: −5.53 to −0.99, $p = 0.005$). Among participants smoking at the 26-week follow-up, 11 (4.6%) increased their smoking rate from baseline, while 94% reduced their smoking rate from baseline.

3.4. Caloric intake

At baseline, participants were consuming approximately 2300 Kcal per day (2304 for BAS+ and 2319 for SC), and overall, changed little across the trial (−19.1 for BAS+ and −116.9 for SC). The overall model of calorie change was not significant ($p = 0.47$). There was no evidence of a time by treatment interaction ($\chi^2(2) = 2.08$, $p = 0.35$). As noted in Table 4, the effect of treatment was not significant, and the effect of time was not significant in the adjusted model.

4. Discussion

This randomized clinical trial of 288 treatment-seeking adult smokers found that behavioral activation counseling plus adjunctive transdermal nicotine did not increase the likelihood of successful quitting or alter post-cessation weight gain above standard smoking cessation counseling plus adjunctive transdermal nicotine. Quit rates at six months for both groups were similar to the average quit rates among those who quit with nicotine replacement therapy (Patnode et al., 2015); (Cahill et al., 2014). Post cessation weight gain among those smoke-free since quitting was about 5.5 pounds, slightly less than the

Table 2
The Effects of BAS+ versus SC on Seven-Day Point Prevalence Abstinence (N = 288).

	Adjusted				Unadjusted			
Predictor Variable	OR	SE	CI 95%	P-value	OR	SE	CI 95%	P-value
12 Weeks post TQD	0.80	0.11	0.61–1.04	0.0982	0.83	0.10	0.66–1.05	0.1204
26 Weeks post TQD	**0.50**	0.07	0.38–0.66	0.0000	**0.55**	0.07	0.42–0.70	0.0000
Treatment Group	0.71	0.18	0.44–1.16	0.1668	0.80	0.19	0.50–1.27	0.3433
Sex (ref male)								
Female	0.91	0.25	0.54–1.54	0.7234	0.90	0.22	0.57–1.45	0.6725
Race (ref African American)								
White	**1.85**	0.52	1.08–3.19	0.0265	**1.96**	0.48	1.22–3.17	0.0057
Other	0.98	0.57	0.31–3.08	0.9657	1.12	0.61	0.38–3.27	0.8347
Income (ref < 20k annually)								
20–50 K annually	1.20	0.35	0.67–2.13	0.5433	1.21	0.34	0.70–2.10	0.4923
50 K+ annually	**2.88**	1.06	1.41–5.91	0.0038	**3.34**	1.06	1.79–6.22	0.0002
Nicotine Dependence	**0.80**	0.06	0.70–0.92	0.0012	**0.83**	0.05	0.73–0.94	0.0035
Weight Concerns	1.02	0.07	0.90–1.15	0.7987	1.07	0.06	0.96–1.19	0.2504
Substitute Reinforcers	**1.41**	0.13	1.18–1.68	0.0001	**1.25**	0.10	1.06–1.47	0.0074
Reference	0.95	0.48	0.36–2.54	0.9200	**0.62**	0.12	0.44–0.86	0.0047

Note: Model includes main effects only. Time by treatment interaction was not significant ($\chi^2(2) = 0.61$, $p = 0.74$). TQD = target quit day; Treatment (SC = 0, BAS+ = 1), Sex (Male = 0, Female = 1), Race (African American = 0, White =1, Other = 2), and Income (< 20k annually = 0, 20–50 K annually = 1, 50k+ = 2). Substitute Reinforcers is the change in substitute reinforcers from baseline. Boldface indicates statistical significance.

Table 3
The Effects of BAS+ versus SC on Weight Gain from Baseline Across Follow Up (N = 198).

	Adjusted				Unadjusted			
Predictor Variable	β	SE	CI 95%	P-value	β	SE	CI 95%	P-value
12 Weeks post TQD	0.17	0.58	-0.97–1.31	0.7682	0.06	0.58	-1.08–1.19	0.9238
26 Weeks post TQD	0.37	0.64	-0.89–1.63	0.5646	-0.20	0.60	-1.38–0.97	0.7348
Treatment Group	-0.31	0.95	-2.17–1.56	0.7462	-0.29	0.99	-2.23–1.65	0.7675
Sex (ref male)								
Female	-0.20	1.09	-2.34–1.95	0.8582	0.43	1.01	-1.55–2.41	0.6680
Race (ref African American)								
White	1.78	1.13	-0.43–4.00	0.1150	**2.32**	1.03	0.30–4.34	0.0247
Other	-0.76	2.23	-5.12–3.60	0.7322	-0.87	2.20	-5.19–3.45	0.6937
Income (ref < 20k annually)								
20–50 K annually	-0.79	1.09	-2.93–1.35	0.4690	-0.76	1.11	-2.93–1.41	0.4906
50 K+ annually	1.02	1.46	-1.84–3.88	0.4833	**2.75**	1.34	0.13–5.36	0.0395
Nicotine Dependence	0.05	0.26	-0.46–0.56	0.8585	0.01	0.26	-0.50–0.53	0.9669
Weight Concerns	**0.52**	0.26	0.01–1.03	0.0439	**0.61**	0.23	0.16–1.07	0.0077
Smoking	**-3.17**	1.26	-5.63–0.70	0.0118	**-3.26**	1.16	-5.53 to − 0.99	0.0049
Substitute Reinforcers	-0.29	0.35	-0.98–0.39	0.4034	-0.17	0.35	-0.86–0.51	0.6175
Baseline Weight	0.00	0.01	-0.02–0.02	0.8214	0.00	0.01	-0.02–0.02	0.6393
Reference	0.44	2.78	-5.88–5.00	0.8745	**2.76**	0.78	1.24–4.29	0.0004

Note: Weight gain measured in pounds. Model includes main effects only. Time by treatment interaction was not significant ($\chi^2(2) = 1.69$, p = 0.43). TQD = target quit day; Treatment (SC = 0, BAS+ = 1), Sex (Male = 0, Female = 1), Race (African American = 0, White =1, Other = 2), and Income (< 20k annually = 0, 20–50 K annually = 1, 50k+ = 2). Smoking is the change in average self-reported cigarette per day from baseline (normalized). Substitute Reinforcers is the change in substitute reinforcers from baseline. Boldface indicates statistical significance.

Table 4
The Effects of BAS+ versus SC on Total Caloric Intake (kcals per day) from Baseline Across Follow Up (N = 213).

	Adjusted				Unadjusted			
Predictor Variable	β	SE	CI 95%	P-value	β	SE	CI 95%	P-value
12 Weeks post TQD	37.00	50.71	-62.39–136.39	0.4657	33.13	51.17	-67.16–133.41	0.5173
26 Weeks post TQD	-68.94	55.23	-177.19–39.30	0.2119	**-100.86**	51.86	-202.51–0.80	0.0518
Treatment Group	102.11	88.81	-71.95–276.17	0.2502	110.65	105.80	-96.72–318.02	0.2956
Sex (ref male)								
Female	**298.2**	100.71	-495.59 to − 100.81	0.0031	17.64	108.28	-194.58–229.86	0.8706
Race (ref African American)								
White	-164.86	102.94	-366.62–36.90	0.1093	9.18	113.60	-213.47–231.82	0.9356
Other	-126.83	199.93	-518.68–265.02	0.5258	-150.42	234.98	-610.97–310.12	0.5221
Income (ref < 20k annually)								
20–50 K annually	93.62	100.55	-103.46–290.71	0.3518	113.71	119.09	-119.70–347.12	0.3396
50 K+ annually	100.56	135.46	-1164.93–366.04	0.4579	165.71	147.41	-123.21–454.62	0.2610
Nicotine Dependence	33.94	24.28	-13.65–81.52	0.1621	-2.31	28.20	-57.58–52.96	0.9347
Weight Concerns	-21.72	22.75	-65.79–23.41	0.3517	1.99	24.53	-46.09–50.07	0.9354
Smoking	-136.19	124.02	-444.19–41.97	0.1049	**-237.74**	118.73	-470.46 to − 5.02	0.0453
Substitute Reinforcers	39.89	32.79	-24.38–104.16	0.2238	0.13	1.24	-2.31–2.56	0.9187
Baseline Kcals	**-0.55**	0.05	-0.66 to − 0.45	0.0000	**-0.46**	0.05	-0.56 to − 0.36	0.0000
Reference	1232.86	228.37	-615.33–235.01	0.0000	-106.46	79.78	-262.82–49.91	0.1821

Note: Model includes main effects only. Time by treatment interaction was not significant ($\chi^2(3) = 2.08$, p = 0.35). TQD = target quit day; Treatment (SC = 0, BAS+ = 1), Sex (Male = 0, Female = 1), Race (African American = 0, White =1, Other = 2), and Income (< 20k annually = 0, 20–50 K annually = 1, 50k+ = 2). Smoking is the change in average self-reported cigarette per day from baseline (normalized). Substitute reinforcers is the change in substitute reinforcers from baseline. Boldface indicates statistical significance.

8–11 pound average reported in the literature (Lycett et al., 2011; Veldheer et al., 2015).

The observed smoking cessation rates for behavioral activation are comparable to those reported in a small study in the U.S. (MacPherson et al., 2010). Also, the lack of significant differences between behavioral activation and standard smoking cessation counseling are consistent with the findings of a recent clinical trial conducted in Spain (Martínez-Vispo et al., 2019). While the present study is the first to evaluate a behavioral activation intervention for smoking cessation and the mitigation of post-cessation weight gain, it appears that the standard smoking cessation counseling is as efficacious for both outcomes.

The unique elements of behavioral activation focused on increasing the frequency and pleasure derived from self-selected alternative reinforcers to cigarettes and high calorie snack foods. Irrespective of treatment group, smokers who increased their alternative reinforcers across the 10 weeks of treatment had a 41% increase in their odds of being abstinent at the 26-week follow-up. These findings, coupled with previous research suggest that smokers who increase alternative reinforcers as they quit smoking even without treatment supporting such efforts, remain abstinent (Audrain-McGovern et al., 2009; Schnoll et al., 2016). Those who have a sustained increase in substitute reinforcers are likely those who are more responsive to nonpharmacological reinforcers (Schnoll et al., 2016). Chronic substance use is associated with decreased responsivity to natural, nonpharmacological rewards based on self-report and neural indicators (Garfield et al., 2014; Huhn et al., 2016). As such, a dampened response to alternative reinforcers relative to cigarettes may result in smoking recurrence, especially among those more dependent on nicotine. Offering combination versus singular nicotine replacement therapy may have provided the opportunity to combine short-acting forms of nicotine replacement therapy (e.g., lozenge or gum) while engaging in alternative reinforcers to smoking, potentially increasing their reinforcing value.

Post cessation weight gain among those continuously abstinent since quit day was over five pounds for both treatment groups, with

participants reporting modest changes in caloric intake across treatment and follow-up. The findings emphasize the difficulty preventing weight gain as a consequence of quitting smoking, although weight gain in the current study was less than typically observed (e.g., 8–11 pounds) (Lycett et al., 2011; Veldheer et al., 2015). Smokers who were more concerned about weight gain prior to treatment tended to gain more weight. Research among weight concerned women smokers documented that a cognitive-behavioral intervention to reduce weight concerns, rather than weight gain produced greater smoking abstinence rates and less PCWG at a one year follow-up compared to standard smoking cessation counseling and a weight control adjunct treatment (Perkins et al., 2001). However, weight concerns did not predict smoking abstinence in the current study, only weight gain. Consistent with the literature, participants who were abstinent for a shorter period of time (i.e., 7-day point prevalence versus continuous abstinence), gained less weight (Klesges et al., 1997) and smokers who reported an increase in their pre-treatment smoking rate at the 26-week follow-up tended to lose weight.

The findings may also suggest that alternative reinforcers could not compete with the immediate reinforcement associated with consuming highly palatable snack foods. Laboratory studies have documented that the temporal window over which reinforcers occur may drive the choice among alternative reinforcers (Bickel et al., 2021). Extending this temporal window through Episodic Futuristic Thinking has shown promise in altering choice behavior (Bickel et al., 2020).

Efforts to avoid palatable foods as a substitute for cigarettes may have placed greater difficulty on smoking cessation efforts among BAS+ participants at a time when the drive for palatable foods is biologically heightened. Emerging research on the gut-brain axis suggests that quitting smoking leads to perturbations in the gut microbiota (Biedermann et al., 2013), which have the potential to alter neuroactive metabolites involved in appetite regulation and food hedonics (Cani et al., 2013; Fetissov, 2017; Zhang and Davies, 2016). Such shifts, happening at the level of the gut with direct communication to the brain, may provide a biological basis for the drive to consume highly palatable foods upon smoking abstinence (Fluhr et al., 2021) and abstinence from other substances (Hodgkins et al., 2003; Jackson and Grilo, 2002).

The study has several methodological strengths that confer confidence in our findings, albeit not as hypothesized. The two groups were quite similar at baseline, differing on none of the covariates, predictors, or outcomes. Adherence to transdermal nicotine and attendance at the 8 counseling sessions was high for both groups. Treatment fidelity was monitored via audio-recordings and a fidelity checklist. The retention rate through the follow-up was almost 80% and did not vary by treatment group. The sample size was sufficient to detect clinically meaningful effects for BAS+ . Thus, the lack of significant differences in cessation and weight change between groups are valid indications that behavioral activation does not add treatment efficacy to that due to standard counseling. Similar findings for smoking cessation were noted in a study comparing cognitive training to the same standard smoking cessation counseling (Loughead et al., 2016).

Despite the strengths, several study limitations should be acknowledged. First, we did not measure elements of treatment engagement, such as the level of homework completed, or satisfaction with participants' assigned intervention. It is possible that these features affected intervention efficacy. Although treatment fidelity was monitored via audio-recordings and a fidelity checklist by the principal investigator, inter-rater reliability was not computed. Second, we excluded smokers who were also using other nicotine products, which limits the generalizability of the findings to dual or poly-tobacco users. Finally, the sample was not specifically recruited for elevated PCWG concerns. BAS+ may have only resonated with smokers who were concerned about gaining weight.

5. Conclusion

While behavioral activation has been shown to be an efficacious inpatient treatment for the use of other substances (Daughters et al., 2018), the present findings do not support its efficacy over standard counseling for outpatient treatment of smoking cessation and the prevention of weight gain. These findings join a growing body of research highlighting the challenge of promoting smoking cessation while preventing PCWG.

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The funding agency had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

CRediT authorship contribution statement

JAM led the conceptualization and design of the study, wrote significant portions of the manuscript text, and provided input on the analyses and the interpretation of the data. EPW conducted the analyses, drafted the interpretation of the analysis, and provided feedback on drafts of the manuscript. RA provided input on the measurement of dietary intake and provided comments and edited drafts of the manuscript. KAP provided feedback on the manuscript and drafted portions of discussion. BA oversaw data collection and study management. DM conducted the literature search, prepared tables, and edited manuscript drafts.

Access to data and data analysis

The corresponding author (JAM) had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Conflict of interest

The authors report no potential conflicts of interest.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.drugalcdep.2023.109792.

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