The Journal of Positive Psychology
Vol. 5, No. 2, March 2010, 122–138

Routledge
Taylor & Francis Group

The energization of health-behavior change: Examining the associations among autonomous self-regulation, subjective vitality, depressive symptoms, and tobacco abstinence

Christopher P. Niemiec[a]*, Richard M. Ryan[a], Heather Patrick[b], Edward L. Deci[a] and Geoffrey C. Williams[a]

[a]*Department of Clinical and Social Sciences in Psychology, University of Rochester, New York, USA;* [b]*Division of Cancer Control and Population Sciences, Behavioral Research Program, Health Promotion Research Branch, National Cancer Institute*

(*Received 23 July 2009; final version received 6 December 2009*)

Most research on the psychological correlates of smoking behavior has focused on negative indices of wellness, but findings are mixed, contradictory, controversial, and, thus, inconclusive. This study, guided by self-determination theory, examined both positive (viz., vitality) and negative (viz., depressive symptoms) indices of psychological health as predictors of long-term tobacco abstinence in the context of a randomized clinical trial. It also examined autonomous self-regulation and cigarette use as predictors of psychological health. Results supported the proposed conditional indirect effect model in which change in cigarette use mediated the relation of change in autonomous self-regulation for smoking cessation to change in vitality, and this indirect effect was moderated by treatment condition. Further, change in vitality predicted long-term tobacco abstinence. Results for depressive symptoms were largely null. Discussion focuses on the importance of considering positive indices of psychological health for understanding the psychological correlates of smoking behavior.

Keywords: depressive symptoms; self-determination theory; tobacco abstinence; vitality

Trial Registration: ClinicalTrials.gov number NCT00178685

Introduction

Three decades ago the Surgeon General (1979) concluded that tobacco use is the most important preventable behavioral factor directly associated with disease and death in the US. Today, tobacco use is the leading cause of mortality in the US (Mokdad, Marks, Stroup, & Gerberding, 2004), accounting for nearly 20% of all deaths (Centers for Disease Control, 1999). A plethora of behavioral and pharmacological interventions have been developed for smoking cessation and their efficacy has been examined. Many treatments have been found to promote smoking cessation, although the percentage of smokers who quit is modest and a high probability of relapse threatens maintained abstinence (Killen, Fortmann, Kraemer, Varady, Davis, & Newman, 1996), as most who quit relapse within one year after treatment (Fiore et al., 2000). Thus, tobacco use is a major health risk that requires attention to multiple factors related to cessation and maintained abstinence. Recently, there has been increased interest in psychological factors associated with tobacco use (Fiore et al., 2008).

Depressive symptoms and smoking

Most research on the psychological correlates of smoking behavior has focused on negative indices of wellness. Initial research (Waal-Manning & de Hamel, 1978) suggested that, relative to non-smokers, current smokers reported elevated indices of negative mood, including depressive symptoms. This association between depressive symptoms and smoking has been found among adolescents (Malkin & Allen, 1980) and adults (Anda, Williamson, Escobedo, Mast, Giovino, & Remington, 1990). Some studies (Frerichs, Aneshensel, Clark, & Yokopenic, 1981), however, found no support for this relation after controlling for relevant confounding variables (age, social class). Thus, there is some (possibly spurious) association between depressive symptoms and smoking.

Longitudinal findings

Researchers have also examined the directionality among these constructs, although considerable debate exists and competing hypotheses have received support. Some have proposed that depressive symptoms

*Corresponding author. Email: niemiec@psych.rochester.edu

ISSN 1743–9760 print/ISSN 1743–9779 online

DOI: 10.1080/17439760903569162
http://www.informaworld.com

are a precursor to smoking later in life. Consistent with this self-medication hypothesis, Prinstein and La Greca (2009) found that childhood depressive symptoms predicted adolescent cigarette use, and Kandel and Davies (1986) showed that adolescent depressive symptoms predicted adult cigarette use. Others have proposed a neuropharmacologic hypothesis, whereby nicotine exposure heightens vulnerability to depressive symptoms because both involve similar neurochemical mechanisms (Dilsaver, Pariser, Churchill, & Larson, 1990). Consistent with this hypothesis, Wu and Anthony (1999) found that childhood cigarette use predicted adolescent depressive symptoms (childhood depressive symptoms did not predict adolescent cigarette use), and Steuber and Danner (2006) showed that, relative to never smokers, adolescents who had used tobacco had increased risk for developing depressive symptoms. Interestingly, smoking cessation and maintained abstinence have been shown to predict a reduction in depressive symptoms over time (Kahler et al., 2002). Still others have suggested a bidirectional causal model. In a longitudinal study of adolescents, Munafò, Hitsman, Rende, Metcalfe, and Niaura (2007) found that, among never smokers, depressive symptoms predicted progression to smoking initiation (consistent with a self-medication hypothesis), whereas progression to smoking initiation predicted increased depressive symptoms (consistent with a neuropharmacologic hypothesis).

Smoking cessation

Evidence suggests that depressive symptoms impair smoking cessation. Berlin and Covey (2006) showed that depressive symptoms predicted smoking cessation failure, while Anda et al. (1990) found that quit ratios decreased as depressive symptoms increased. Other published reports, however, have provided evidence to the contrary. Although Hayford et al. (1999) found that increased depressive symptoms predicted smoking outcomes at the end of treatment they did not predict tobacco abstinence at a one-year follow-up. Recently, Kodl et al. (2008) reported that depressive symptoms did not predict future tobacco abstinence after controlling for relevant covariates and time of assessment.

Brief summary

This evidence suggests potential comorbidity between depressive symptoms and smoking, although the direction of this association is unclear. Further, whereas some studies have indicated that depressive symptoms impair smoking cessation, others have not. Thus, findings on the relation of depressive symptoms to smoking are mixed, contradictory, and controversial, making it difficult to draw sound, definitive conclusions on the association between negative indices of psychological health and smoking behavior.

Toward a consideration of positive indices of psychological health

Clinical literatures often define psychological health as the absence of psychopathology (Cicchetti, 1991). As a result, research on the importance of positive indices of psychological health in predicting smoking behavior is lacking (Doran et al., 2006). This dearth of research is hardly surprising given the general medical model's primary focus on disease, physical illness, and alleviation of suffering. The lack of consideration given to positive indices of psychological health may also reflect an assumption that positive and negative affect exist along a single mood dimension (Russell & Carroll, 1999), in which positive mood suggests the absence of negative mood, and vice versa. As Cook, Spring, McChargue, Borrelli, et al. (2004) and Doran et al. (2006) summarized, however, positive and negative affect are distinct, as they are located in different brain regions (Davidson, 1992) and relate to different psychological constructs (Watson, Clark, & Tellegen, 1988). Thus, a consideration of positive indices of psychological health may inform practitioners on effective ways to facilitate health-behavior change.

Several studies have examined how positive indices of psychological health affect smoking behavior. This growing literature suggests that low positive mood predicts smoking and cessation failure. Presson, Chassin, and Sherman (2002) reported that adults with low positive mood were more likely than those with high positive mood to be smokers, and chronically low positive mood has been associated with increased craving (Cook, Spring, McChargue, & Hedeker, 2004) and temptation to smoke (Rabois & Haaga, 2003). Importantly, low positive mood has been shown to predict relapse following smoking cessation (al'Absi, Hatsukami, Davis, & Wittmers, 2004; Doran et al., 2006), even incrementally to negative affect and somatic features, and while controlling for nicotine dependence, smoking frequency, and history of major depression (Leventhal, Ramsey, Brown, LaChance, & Kahler, 2008). These results underscore the importance of considering how positive indices of psychological health affect smoking behavior, although such evidence is minimal (Cook, Spring, McChargue, Borrelli, et al., 2004).

An important positive indicator of psychological health that has received considerable attention in recent years, but has not yet been examined vis-à-vis long-term tobacco abstinence, is vitality. Ryan and Frederick (1997) defined vitality as 'a positive feeling of aliveness and energy' (p. 529), which is the psychological energy available to an individual that reflects

well-being and promotes behaviors that support a healthy lifestyle. As expected, vitality has been found to relate negatively to depressive symptoms (e.g., Niemiec, Lynch, Vansteenkiste, Bernstein, Deci, & Ryan, 2006).

The concept of vitality was developed within the framework of self-determination theory (SDT; Deci & Ryan, 2000; Niemiec, Ryan, & Deci, 2010; Ryan & Deci, 2000). Within SDT, autonomous self-regulation (ASR) involves behaving with the experiences of volition and self-endorsement, and is considered the basis for optimal functioning. Conversely, controlled regulation involves the experiences of pressure or coercion to think, feel, or behave in particular ways. With ASR for health-behavior change, people are expected to report higher psychological wellness and lower psychological distress, and to engage in behaviors that support a healthy lifestyle. Considerable research has linked ASR to psychological wellness, including vitality (Ryan & Deci, 2008). In contrast, controlled regulation has been shown to deplete the energy people have available for self-regulation (Moller, Deci, & Ryan, 2006).

In the health-care domain, Ryan and Frederick (1997) found that vitality was lower among patients entering a pain clinic who reported more controlled regulation for treatment (Study 4), whereas vitality was higher among morbidly obese patients who reported more ASR for following program guidelines (Study 5). Importantly, maintained weight loss was associated with higher vitality among the patients in that study. Thus, people tend to experience higher psychological energy when they feel volitional to undertake health-behavior change. Evidence also suggests that vitality is related to physical health. Ryan and Frederick reported that vitality related negatively to somatic distress (Study 1); physical symptoms (Study 2); physical pain, particularly among those who experienced their pain as debilitating (Study 4); and daily variations in physical symptoms (Study 6). Conversely, vitality related positively to body functioning self-esteem, perceived physical ability, and physical self-presentation confidence (Study 1). With its relations to ASR and physical health, vitality represents a central indicator of organismic wellness that appears to be important for health-relevant processes (Ryan & Deci, 2008). This review suggests that a growing body of evidence supports a link between vitality and ASR for health-behavior change, as well as actual behavior change. Thus, an examination of whether vitality facilitates long-term tobacco abstinence seems warranted.

The present research

The present research examined the relations of ASR and cigarette use to both positive (viz., vitality) and negative (viz., depressive symptoms) indices of psychological health, and the relations of those indices to long-term tobacco abstinence. This study was conducted in the context of a randomized clinical trial of an SDT-based intervention for smoking cessation and prolonged abstinence from tobacco, and had two primary aims.

Research aim 1

The first aim was to test whether change in cigarette use would mediate the relation of change in ASR for smoking cessation to change in psychological health, and whether an intensive tobacco dependence intervention designed to promote smokers' autonomy would moderate this indirect effect. Thus, using SDT we proposed a conditional indirect effect model to account for subsequent change in psychological health (see Figure 1). The first component (labeled A) was that increased ASR from baseline to 6 months would predict increased vitality, and decreased depressive symptoms, from 6 to 18 months.[1] This was expected based on previous research showing that ASR and vitality were positively correlated, ASR and depressive symptoms were negatively correlated,[2] and vitality and depressive symptoms were negatively correlated (Niemiec et al., 2006; Ryan & Frederick, 1997). Importantly, this

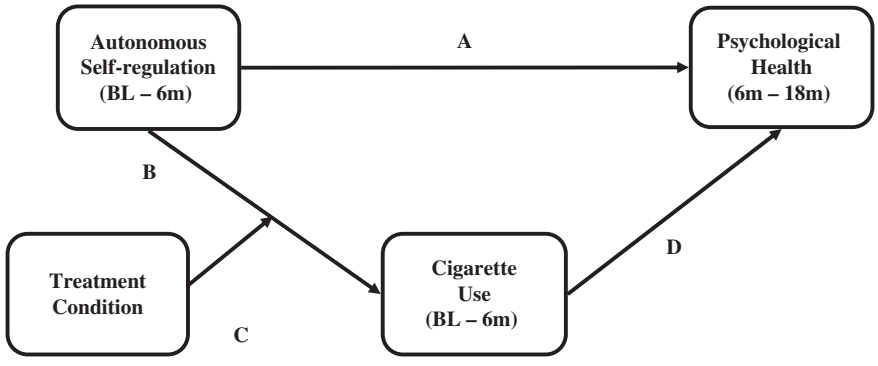

Figure 1. The hypothesized conditional indirect effect model.

provided a test of directionality between the predictor and the outcomes, as change in ASR temporally preceded change in psychological health.

The second component (labeled B) was that increased ASR from baseline to 6 months would predict decreased cigarette use from baseline to 6 months. This was expected because smokers who experience higher ASR are more likely to initiate and maintain health-behavior change (Williams, McGregor, Sharp, Levesque, et al., 2006). We examined change in these variables from baseline to 6 months because that was the duration of the clinical trial, which was designed to promote autonomy for smoking cessation. So, although we could not examine directionality, we wanted to capture the association between ASR and cigarette use *during* the SDT-based intervention and then test whether treatment condition would moderate this relation.

The third component (labeled C) was that the association between change in ASR and change in cigarette use would be moderated by treatment condition, such that participants randomized to the intensive intervention, relative to those in community care, would show a stronger negative association between ASR and cigarette use. This interaction was expected because behavior change is more likely to occur when people feel volitional and are in autonomy-supportive contexts (Williams, Deci, & Ryan, 1998). Thus, we anticipated that those higher in ASR and who received the autonomy-supportive intervention would report the most health-behavior change (decreased cigarette use) during the clinical trial.

The fourth component (labeled D) was that decreased cigarette use from baseline to 6 months would predict increased vitality, and decreased depressive symptoms, from 6 to 18 months. Because smoking is likely to undermine wellness, we predicted a negative relation of change in cigarette use to change in vitality. Because of the comorbidity between smoking and depressive symptoms (Anda et al., 1990), we predicted a positive relation of change in cigarette use to change in depressive symptoms. Importantly, as with Component A, this provided a test of directionality between the mediator (change in cigarette use) and the outcomes.

In sum, we expected that change in ASR for smoking cessation would predict subsequent change in psychological health, and that change in cigarette use would account for some of this direct relation. We hypothesized mediation because we expected that smokers would be more likely to reduce their cigarette use when they feel volitional to do so, and that such health-behavior change would predict subsequent changes in vitality and depressive symptoms. Further, we expected that this mediation would be evident only among those who received an intensive tobacco dependence intervention designed to promote smokers' autonomy.

We used a bootstrap approach to test our hypotheses concerning both simple mediation and moderated mediation. Bootstrapping is a resampling strategy widely advocated for assessing indirect effects, as this technique makes no assumptions about the normality of the sampling distribution and can be used to generate *bias-correction* and *bias-correction and acceleration* confidence intervals (see Preacher, Rucker, & Hayes, 2007). We followed the guidelines and analytic methods discussed in Preacher and Hayes (2004) to test for simple mediation, and those of Preacher et al. (2007) to test for moderated mediation.

Research aim 2

The second aim was to examine whether changes in vitality and depressive symptoms, both during (baseline to 6 months) and after (6 to 18 months) the clinical trial, would predict long-term (24 months post-intervention) tobacco abstinence. We expected change in vitality to predict the likelihood of attaining tobacco abstinence. However, we did not hypothesize about change in depressive symptoms because of the contradictory findings on this association. We conducted binary logistic regression analyses to examine these relations, which provided a test of directionality between the predictors and the outcomes.

Method

Participants and procedure

Adult smokers were recruited using signs in physicians' offices and by newspaper advertisements to participate in the *Smoker's Health Study*. Between January 2000 and July 2002, 2681 smokers were screened for eligibility, of whom 2037 met eligibility criteria and provided phone consent to have two fasting lipid profiles 7 days apart prior to their baseline appointment, which provided a risk-assessment of heart disease. Those who were eligible had smoked more than 100 cigarettes in their lifetime and had smoked 5 or more cigarettes per day during the week prior to enrollment, were 18 years of age or older, read and spoke English, had no history of psychotic illness (anxiety and depression were allowed), had a minimal life expectancy of 18 months, and planned to live in the area for at least 18 months. Smokers could participate in the study regardless of whether they intended to quit. Of those who were eligible, 1006 (360 male, 643 female, 3 who did not indicate gender) came to an initial appointment, provided informed consent, completed baseline questionnaires, and were randomized to treatment condition.

Randomization was stratified by whether participants had normal versus elevated LDL-C values based on the results of their fasting lipid profiles and defined by the National Cholesterol Education Program's (1997) guidelines. Previous analysis of these data indicated that the dietary intervention had no effect on tobacco outcomes (Williams et al., 2006), so we collapsed across dietary and tobacco conditions and focused only on the tobacco intervention and outcomes. Seventy percent of participants ($n = 714$) were randomized to a 6-month SDT-based intervention designed to promote smokers' autonomy, whereas the rest ($n = 292$) received community care. This ratio for random assignment was used to minimize harm to the community care group because the intervention was expected to have a more pronounced effect on smoking cessation (Fiore et al., 2000). All community care participants were offered intensive treatment after the 24-month post-intervention follow-up. This study was approved by the University of Rochester Human Subjects Review Board. Figure 2 depicts participant flow through the 30-month study period.

A detailed description of the study design, recruitment procedures, and treatment approach have been presented elsewhere (Williams et al., 2002), as have baseline demographics and the primary outcome results at 6 months (Williams et al., 2006), 18 months (Williams et al., 2006), and 30 months (Williams, Niemiec, Patrick, Ryan, & Deci, 2009) post-randomization. Following randomization, participants in the SDT-based intervention met with a counselor and were asked about their smoking history and attitudes toward smoking, and were informed about the potential benefits of tobacco abstinence. They were also encouraged to discuss their life aspirations and the ways in which they believed smoking helped and/or hindered their attaining those goals (Niemiec, Ryan, Deci, & Williams, 2009). Finally, participants were asked whether they wanted to quit smoking. If yes, counselors provided competence support; if no, counselors asked participants to return again in 2 months to discuss their smoking. Throughout the intervention, counselors used an autonomy-supportive approach, which means they reflected participants' perspectives, responded to their questions, emphasized choice about smoking versus cessation, and provided relevant information in a non-pressuring way (Williams et al., 1998). Participants in community care were encouraged to meet with their physician and were given contact information for all local smoking cessation resources, including the New York State Quit Line. All participants were paid US$75, and those who provided data at 24 months post-intervention received an additional US$5 honorarium.

Measures

Autonomous self-regulation for smoking cessation

The Treatment Self-Regulation Questionnaire (Williams, Grow, Freedman, Ryan, & Deci, 1996) presented participants with the stem, 'The reason I would stop smoking permanently or continue not smoking is...' Participants rated pre-selected responses assessing identified (4 items; e.g., because I personally believe it is the best thing for my health) and integrated (2 items; e.g., because stopping smoking is consistent with my life goals) reasons for behavior change. Responses were made on a 7-point scale from 1 (*not at all true*) to 7 (*very true*). The identified and integrated items were averaged to form an ASR composite. The observed range was 1.17 to 7.00 at baseline and 1.00 to 7.00 at 6 months.

Cigarette use

Participants responded to the question, 'During a typical 7 day period, how many cigarettes did you smoke per day?' The observed range was 2 to 60 cigarettes smoked per day at baseline and 0 to 80 cigarettes smoked per day at 6 months.

Vitality

The Subjective Vitality Scale (Ryan & Frederick, 1997) assessed vitality (7 items; e.g., In general, I feel alive and vital). Responses were made on a 7-point scale from 1 (*strongly disagree*) to 7 (*strongly agree*). The observed range was 1.00 to 7.00 at baseline, 6, and 18 months.

Depressive symptoms

The Center for Epidemiological Studies–Depression scale (Radloff, 1977) assessed depressive symptoms during the past week (20 items; e.g., I was bothered by things that usually don't bother me). Responses were made on a 4-point scale from 0 (*rarely or none of the time—less than 1 day*) to 3 (*most or all of the time—5 to 7 days*). The observed range was 0.00 to 2.40 at baseline, 0.00 to 2.75 at 6 months, and 0.00 to 2.95 at 18 months.

Smoking status

At 24 months post-intervention, participants responded either 'yes' or 'no' to having smoked a cigarette, even a puff, in the past 7 days and to having currently used a pipe, cigars, snuff, or chewing tobacco. To be classified as having attained 7-day point prevalence (7 dPP) tobacco abstinence, participants must have responded 'no' to having used each form of tobacco listed above. To be classified as having

attained 24-month prolonged abstinence (24 mPA; Hughes, Keely, Niaura, Ossip-Klein, Richmond, & Swan, 2003) from tobacco, participants must have quit smoking by the end of the 6-month intervention (with a 2 week 'grace period'), assessed with a biochemically validated 7 dPP measure, and must not have used any form of tobacco listed above between that time and 24-months post-intervention.

Analytic overview

Two types of analyses were conducted to assess our hypotheses. First, as in previous published reports from this trial, we conducted an intention-to-treat analysis, which included all 1006 participants. Missing data regarding ASR for smoking cessation, cigarette use, vitality, and depressive symptoms were replaced by participants' last known reports or, if necessary, by

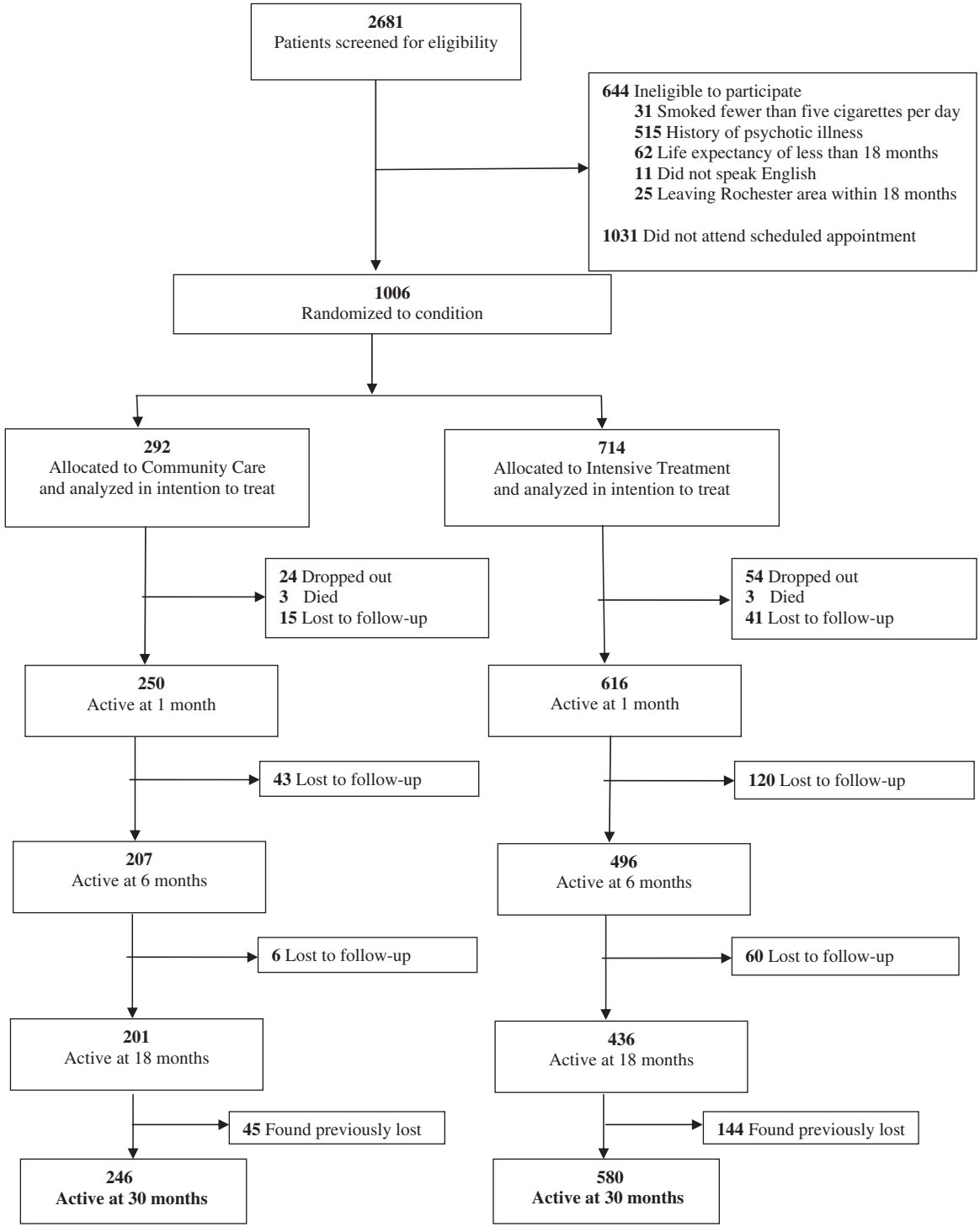

Figure 2. CONSORT recruitment and retention of participants.

mean replacement. If smoking status was unavailable, the participant was considered smoking. This analytic technique is considered the standard for biomedical and epidemiological research. Second, we conducted an as-treated analysis, which used all available data without replacing missing values.

Results

Those randomized to the intervention had an average of 4.42 visits with a counselor (51% in person, 49% via phone). Of those participants, 323 chose to see a study doctor and had an average of 1.3 visits with the doctor (33% in person, 67% via phone). At 6 months, those in the intervention had an average of 155.04 minutes of contact time.

Independent samples *t*-tests with Bonferroni protection revealed one significant male-female difference. Males [M (SD) = 4.72 (1.05)] reported higher vitality at 18 months than females [M (SD) = 4.52 (1.11)], t (1001) = 2.85, $p < 0.005$. Table 1 presents descriptive statistics and group differences for the study measures. Randomization was effective, as groups did not differ on the baseline variables. Although not presented in Table 1, analysis of covariance (ANCOVA) revealed significant differences between groups on 6-month ASR and cigarette use (controlling for baseline). Smokers in the intervention reported more ASR from baseline to 6 months [$M(SE) = 6.22$ (0.03)] than those in community care [$M(SE) = 6.12$ (0.04)], $F(1, 1003) = 4.08$, $p < 0.05$. Smokers in the intervention reported less cigarette use from baseline to 6 months [$M(SE) = 13.27$ (0.34)] than those in community care [$M(SE) = 15.43$ (0.54)], $F(1, 1003) = 11.46$, $p < 0.001$. No significant differences on psychological health were found between groups. Table 2 presents descriptive statistics and intercorrelations for the baseline, 6-month, and 18-month measures.[3]

Table 1. Descriptive statistics and group differences for the measures at baseline, 6, and 18 months.

	Community Care M (SD)	Intensive Intervention M (SD)	t (1004)
Baseline Measures			
ASR	5.99 (1.19)	6.11 (1.04)	1.58
Cigarette Use	20.86 (9.97)	20.32 (9.83)	0.80
Vitality	4.60 (1.07)	4.66 (1.06)	0.84
Depressive Symptoms	0.77 (0.50)	0.73 (0.46)	1.17
6-month Measures			
ASR	6.06 (1.18)	6.25 (1.00)	2.54*
Cigarette Use	15.65 (10.10)	13.18 (11.11)	3.29***
Vitality	4.70 (1.14)	4.71 (1.10)	0.18
Depressive Symptoms	0.77 (0.53)	0.76 (0.53)	0.27
18-month Measures			
Vitality	4.55 (1.11)	4.61 (1.09)	0.79
Depressive Symptoms	0.77 (0.60)	0.75 (0.56)	0.59

Note: ASR = Autonomous self-regulation for smoking cessation.
*$p < 0.05$; ***$p < 0.001$.

Table 2. Descriptive statistics, intercorrelations, and scale reliabilities (α) for measures at baseline, 6, and 18 months.

Measures	1	2	3	4	5	6	7	8	9	10
1. ASR—BL	0.85									
2. Cig—BL	−0.09**	–								
3. Vit—BL	0.20***	−0.09**	0.67							
4. Dep—BL	−0.06^{+}	0.11***	−0.51***	0.76						
5. ASR—6m	0.72***	−0.08**	0.17***	−0.05	0.89					
6. Cig—6m	−0.15***	0.53***	−0.12***	0.07*	−0.19***	–				
7. Vit—6m	0.15***	−0.12***	0.68***	−0.43***	0.18***	−0.16***	0.73			
8. Dep—6m	−0.02	0.07*	−0.34***	0.60***	−0.05	0.09**	−0.60***	0.76		
9. Vit—18m	0.18***	−0.18***	0.61***	−0.39***	0.21***	−0.22***	0.79***	−0.44***	0.66	
10. Dep—18m	0.02	0.10***	−0.32***	0.56***	−0.03	0.10**	−0.46***	0.68***	−0.51***	0.82
M	6.08	20.47	4.64	0.75	6.19	13.90	4.71	0.76	4.59	0.75
SD	1.09	9.87	1.06	0.47	1.06	10.88	1.11	0.53	1.10	0.57

Notes: ASR = Autonomous self-regulation for smoking cessation, Cig = Cigarette use, Vit = Vitality, Dep = Depressive symptoms, BL = Baseline, 6m = 6 months, 18m = 18 months. Scale reliabilities (Cronbach's α) are shown on the diagonal.
$^{+}p < 0.10$; *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

Testing research aim 1

The first aim was to test whether change in cigarette use would mediate the relation of change in ASR for smoking cessation to change in psychological health, and whether an intensive tobacco dependence intervention designed to promote smokers' autonomy would moderate this indirect effect. Change scores were computed as unstandardized residuals. For each outcome (viz., vitality and depressive symptoms), we conducted an intention-to-treat analysis followed by an as-treated analysis.

Intention-to-treat analysis: vitality

We used the analytic methods discussed in Preacher and Hayes (2004) to test for simple mediation. Results are presented in Table 3. The unconditional indirect effect was significant (95% CI: $\{0.0017, 0.0205\}$ with 5000 resamples; Sobel $z = 2.05$, $p < 0.05$). Change in ASR from baseline to 6 months predicted change in cigarette use from baseline to 6 months ($b = -0.13$, $p < 0.001$), which in turn predicted change in vitality from 6 to 18 months ($b = -0.08$, $p < 0.05$). Controlling for the mediator, the relation of change in ASR to change in vitality was reduced from $b = 0.07$ ($p < 0.05$) to $b = 0.06$ ($p < 0.05$).

We then used the analytic methods discussed in Preacher et al. (2007) to test a moderated mediation model in which the indirect effect was presumed to be moderated by treatment condition. This analysis generated two multiple regression models. The *mediator variable model* examined change in cigarette use as the dependent variable, and the *dependent variable model* examined change in vitality as the dependent variable. Results are presented in Table 4. In the mediator variable model the interaction of change in ASR with treatment condition predicted change in cigarette use ($b = -0.13$, $p < 0.05$). In the dependent variable model change in cigarette use predicted change in vitality ($b = -0.07$, $p < 0.05$). Table 4 also presents the *conditional indirect effect* within treatment conditions. In community care there was no indirect effect of change in ASR to change in vitality, whereas this indirect effect was significant in the intervention.

We then calculated bootstrap confidence intervals for these indirect effects. With 5000 resamples, the indirect effect in community care yielded a bootstrap 95% *bias correction and acceleration* confidence interval (BCa CI) of $\{-0.0035, 0.0144\}$. Because this interval contained 0, the conditional indirect effect in community care was not significantly different from 0 at $\alpha = 0.05$. Repeating this procedure for those in the intervention yielded a 95% BCa CI of $\{0.0026, 0.0276\}$. Because this interval did not contain 0, the conditional indirect effect in the intervention was significantly different from 0 at $\alpha = 0.05$.

As-treated analysis: vitality

Results testing for simple mediation are presented in Table 5. The unconditional indirect effect was significant (95% CI: $\{0.0081, 0.0500\}$ with 5000 resamples; Sobel $z = 2.51$, $p < 0.05$). Change in ASR from baseline to 6 months predicted change in cigarette use from baseline to 6 months ($b = -2.12$, $p < 0.001$), which in turn predicted change in vitality from 6 to 18 months ($b = -0.01$, $p < 0.01$). Controlling for the mediator, the relation of change in ASR to change in vitality was reduced from $b = 0.09$ ($p < 0.05$) to $b = 0.07$ (*ns*).

Table 3. Unconditional indirect effect of change in ASR to subsequent change in vitality through change in cigarette use: intention-to-treat analysis.

Sample Size = 1006 Number of Bootstrap Resamples = 5000	*b*	*SE*	*t*	
Direct and Total Effects				
b (YX)	0.0730	0.0315	2.32*	
b (MX)	−0.1285	0.0313	−4.11***	
b (YM.X)	−0.0770	0.0317	−2.43*	
b (YX.M)	0.0631	0.0317	1.99*	
	Value	*SE*	95% CI	*z*
Indirect Effect and Significance Using Normal Distribution	0.0099	0.0048	{0.0004, 0.0194}	2.05*
	Mean	*SE*	95% CI	
Bootstrap Results for Indirect Effect	0.0099	0.0049	{0.0017, 0.0205}	

Notes: *b* (YX) = the total effect of the independent variable (change in ASR) on the dependent variable (change in vitality); *b* (MX) = the effect of the independent variable on the proposed mediator (change in cigarette use). *b* (YM.X) = the effect of the mediator on the dependent variable, controlling for the independent variable. *b* (YX.M) = the effect of the independent variable on the dependent variable, controlling for the mediator.
*$p < 0.05$; ***$p < 0.001$.

Results testing for moderated mediation are presented in Table 6. In the mediator variable model the interaction of change in ASR with treatment condition did not predict change in cigarette use ($b = -1.44$, *ns*). In the dependent variable model change in cigarette use predicted change in vitality ($b = -0.01$, $p < 0.01$). Table 6 also presents the *conditional indirect effect* within treatment conditions.[4] In community care there

Table 4. Conditional indirect effect of change in ASR to subsequent change in vitality through change in cigarette use: intention-to-treat analysis.

Sample Size = 1006
Number of Bootstrap Resamples = 5000

Predictor	Mediator Variable Model (DV = Change in Cigarette Use) *b*	*SE*	*t*
Constant	0.0038	0.0311	0.12
Change in ASR	−0.1348	0.0318	−4.25***
Treatment Condition	−0.2227	0.0687	−3.24**
Interaction	−0.1323	0.0640	−2.07*
Predictor	**Dependent Variable Model (DV = Change in Vitality) *b***	***SE***	***t***
Constant	−0.0007	0.0314	−0.02
Change in Cigarette Use	−0.0740	0.0319	−2.32*
Change in ASR	0.0644	0.0324	1.99*
Treatment Condition	0.0490	0.0697	0.70
Interaction	0.0245	0.0648	0.38
Treatment Condition	**Conditional Indirect Effect within Treatment Conditions $(a_1 + a_3W)b_1$**	***SE***	***z***
Community Care	0.0031	0.0043	0.71
Intensive Intervention	0.0128	0.0063	2.04*

Notes: The conditional indirect effect is calculated by $(a_1 + a_3W)b_1$, where a_1 is the path from change in ASR to change in cigarette use (from the mediator variable model), a_3 is the path from the interaction of change in ASR with treatment condition to change in cigarette use (from the mediator variable model), W is treatment condition, and b_1 is the path from change in cigarette use to change in vitality (from the dependent variable model).
*$p < 0.05$; ***$p < 0.001$.

Table 5. Unconditional indirect effect of change in ASR to subsequent change in vitality through change in cigarette use: as-treated analysis.

Sample Size = 515
Number of Bootstrap Resamples = 5000

	b	*SE*	*t*	
Direct and Total Effects				
b (YX)	0.0936	0.0430	2.18*	
b (MX)	−2.1158	0.4674	−4.53***	
b (YM.X)	−0.0124	0.0040	−3.09**	
b (YX.M)	0.0673	0.0435	1.55	
	Value	***SE***	**95% CI**	***z***
Indirect Effect and Significance Using Normal Distribution	0.0263	0.0105	{0.0058, 0.0469}	2.51*
	Mean	***SE***	**95% CI**	
Bootstrap Results for Indirect Effect	0.0263	0.0107	{0.0081, 0.0500}	

Notes: *b* (YX) = the total effect of the independent variable (change in ASR) on the dependent variable (change in vitality); *b* (MX) = the effect of the independent variable on the proposed mediator (change in cigarette use). *b* (YM.X) = the effect of the mediator on the dependent variable, controlling for the independent variable. *b* (YX.M) = the effect of the independent variable on the dependent variable, controlling for the mediator.
*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

Table 6. Conditional indirect effect of change in ASR to subsequent change in vitality through change in cigarette use: as-treated analysis.

Sample Size = 515
Number of Bootstrap Resamples = 5000

Predictor	Mediator Variable Model (DV = Change in Cigarette Use) b	SE	t
Constant	0.0682	0.3864	0.18
Change in ASR	−2.1640	0.4727	−4.58***
Treatment Condition	−2.3241	0.8193	−2.84**
Interaction	−1.4400	0.9575	−1.50
Predictor	**Dependent Variable Model (DV = Change in Vitality) b**	**SE**	**t**
Constant	−0.0004	0.0356	−0.01
Change in Cigarette Use	−0.0123	0.0041	−3.02**
Change in ASR	0.0700	0.0445	1.58
Treatment Condition	0.0100	0.0761	0.13
Interaction	0.0301	0.0884	0.34
Treatment Condition	**Conditional Indirect Effect within Treatment Conditions $(a_1 + a_3W)b_1$**	**SE**	**z**
Community Care	0.0143	0.0117	1.22
Intensive Intervention	0.0314	0.0131	2.40*

Notes: The conditional indirect effect is calculated by $(a_1 + a_3W)b_1$, where a_1 is the path from change in ASR to change in cigarette use (from the mediator variable model), a_3 is the path from the interaction of change in ASR with treatment condition to change in cigarette use (from the mediator variable model), W is treatment condition, and b_1 is the path from change in cigarette use to change in vitality (from the dependent variable model).
*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

was no indirect effect of change in ASR to change in vitality, whereas this indirect effect was significant in the intervention.

We then calculated bootstrap confidence intervals for these indirect effects. With 5000 resamples, the indirect effect in community care yielded a bootstrap 95% BCa CI of {−0.0032, 0.0453}. Because this interval contained 0, the conditional indirect effect in community care was not significantly different from 0 at $\alpha = 0.05$. Repeating this procedure for those in the intervention yielded a 95% BCa CI of {0.0106, 0.0629}. Because this interval did not contain 0, the conditional indirect effect in the intervention was significantly different from 0 at $\alpha = 0.05$. Overall, then, using both intention-to-treat and as-treated analyses, the signs of the path coefficients and the conditional indirect effects were consistent with the interpretation that increased ASR predicted decreased cigarette use, which in turn predicted increased vitality, but this indirect effect was only significant for those in the intensive intervention.

Intention-to-treat analysis: depressive symptoms

Results testing for simple mediation are presented in Table 7. The unconditional effect was nonsignificant (95% CI: {−0.0040, 0.0035} with 5000 resamples, Sobel $z = -0.12$, *ns*). Change in ASR from baseline to 6 months predicted change in cigarette use from baseline to 6 months ($b = -0.13$, $p < 0.001$), but change in cigarette use was unrelated to change in depressive symptoms from 6 to 18 months ($b = 0.00$, *ns*). Results testing for moderated mediation are presented in Table 8.[5] In the mediator variable model the interaction of change in ASR with treatment condition predicted change in cigarette use ($b = -0.13$, $p < 0.05$). In the dependent variable model, change in cigarette use was unrelated to change in depressive symptoms ($b = 0.00$, *ns*). The indirect effect was nonsignificant in both community care and the intervention.

As-treated analysis: depressive symptoms

Results testing for simple mediation are presented in Table 9. The unconditional effect was nonsignificant (95% CI: {−0.0154, 0.0098} with 5000 resamples, Sobel $z = -0.45$, *ns*). Change in ASR from baseline to 6 months predicted change in cigarette use from baseline to 6 months ($b = -2.10$, $p < 0.001$), but change in cigarette use was unrelated to change in depressive symptoms from 6 to 18 months ($b = 0.00$, *ns*). Results testing for moderated mediation are presented

Table 7. Unconditional indirect effect of change in ASR to subsequent change in depressive symptoms through change in cigarette use: intention-to-treat analysis.

Sample Size = 1006
Number of Bootstrap Resamples = 5000

	b	*SE*	*t*	
Direct and Total Effects				
b (YX)	−0.0219	0.0132	−1.66^{+}	
b (MX)	−0.1285	0.0313	−4.11***	
b (YM.X)	0.0017	0.0133	0.13	
b (YX.M)	−0.0217	0.0133	−1.63	
	Value	*SE*	95% CI	*z*
Indirect Effect and Significance Using Normal Distribution	−0.0002	0.0018	{−0.0037, 0.0032}	−0.12
	Mean	*SE*	95% CI	
Bootstrap Results for Indirect Effect	−0.0002	0.0019	{−0.0040, 0.0035}	

Notes: *b* (YX) = the total effect of the independent variable (change in ASR) on the dependent variable (change in depressive symptoms); *b* (MX) = the effect of the independent variable on the proposed mediator (change in cigarette use). *b* (YM.X) = the effect of the mediator on the dependent variable, controlling for the independent variable. *b* (YX.M) = the effect of the independent variable on the dependent variable, controlling for the mediator.
$^{+}p < 0.10$; ***$p < 0.001$.

Table 8. Conditional indirect effect of change in ASR to subsequent change in depressive symptoms through change in cigarette use: intention-to-treat analysis.

Sample Size = 1006
Number of Bootstrap Resamples = 5000

	Mediator Variable Model (DV = Change in Cigarette Use)		
Predictor	*b*	*SE*	*t*
Constant	0.0038	0.0311	0.12
Change in ASR	−0.1348	0.0318	−4.25***
Treatment Condition	−0.2227	0.0687	−3.24**
Interaction	−0.1323	0.0640	−2.07*
	Dependent Variable Model (DV = Change in Depressive Symptoms)		
Predictor	*b*	*SE*	*t*
Constant	0.0001	0.0132	0.01
Change in Cigarette Use	0.0010	0.0134	0.08
Change in ASR	−0.0217	0.0136	−1.60
Treatment Condition	−0.0129	0.0293	−0.44
Interaction	−0.0032	0.0272	−0.12
	Conditional Indirect Effect within Treatment Conditions		
Treatment Condition	$(a_1 + a_3W)b_1$	*SE*	*z*
Community Care	−0.0001	0.0009	−0.12
Intensive Intervention	−0.0002	0.0025	−0.09

Notes: The conditional indirect effect is calculated by $(a_1 + a_3W)b_1$, where a_1 is the path from change in ASR to change in cigarette use (from the mediator variable model), a_3 is the path from the interaction of change in ASR with treatment condition to change in cigarette use (from the mediator variable model), W is treatment condition, and b_1 is the path from change in cigarette use to change in depressive symptoms (from the dependent variable model).
*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

Table 9. Unconditional indirect effect of change in ASR to subsequent change in depressive symptoms through change in cigarette use: as-treated analysis.

Sample Size = 518
Number of Bootstrap Resamples = 5000

	b	*SE*	*t*	
Direct and Total Effects				
b (YX)	−0.0455	0.0276	−1.65^{+}	
b (MX)	−2.1024	0.4677	−4.50***	
b (YM.X)	0.0012	0.0026	0.47	
b (YX.M)	−0.0430	0.0282	−1.53	
	Value	*SE*	95% CI	*z*
Indirect Effect and Significance Using Normal Distribution	−0.0025	0.0056	{−0.0136, 0.0085}	−0.45
	Mean	*SE*	95% CI	
Bootstrap Results for Indirect Effect	−0.0024	0.0063	{−0.0154, 0.0098}	

Notes: *b* (YX) = the total effect of the independent variable (change in ASR) on the dependent variable (change in depressive symptoms); *b* (MX) = the effect of the independent variable on the proposed mediator (change in cigarette use). *b* (YM.X) = the effect of the mediator on the dependent variable, controlling for the independent variable. *b* (YX.M) = the effect of the independent variable on the dependent variable, controlling for the mediator. $^{+}p < 0.10$; ***$p < 0.001$.

Table 10. Conditional indirect effect of change in ASR to subsequent change in depressive symptoms through change in cigarette use: as-treated analysis.

Sample Size = 518
Number of Bootstrap Resamples = 5000

	Mediator Variable Model (DV = Change in Cigarette Use)		
Predictor	*b*	*SE*	*t*
Constant	0.1243	0.3857	0.32
Change in ASR	−2.1489	0.4731	−4.54***
Treatment Condition	−2.3159	0.8177	−2.83**
Interaction	−1.4291	0.9583	−1.49
	Dependent Variable Model (DV = Change in Depressive Symptoms)		
Predictor	*b*	*SE*	*t*
Constant	−0.0022	0.0229	−0.10
Change in Cigarette Use	0.0008	0.0026	0.29
Change in ASR	−0.0405	0.0287	−1.41
Treatment Condition	−0.0746	0.0490	−1.52
Interaction	0.0078	0.0571	0.14
	Conditional Indirect Effect within Treatment Conditions		
Treatment Condition	$(a_1 + a_3W)b_1$	*SE*	*z*
Community Care	−0.0011	0.0042	−0.26
Intensive Intervention	−0.0021	0.0078	−0.27

Notes: The conditional indirect effect is calculated by $(a_1 + a_3W)b_1$, where a_1 is the path from change in ASR to change in cigarette use (from the mediator variable model), a_3 is the path from the interaction of change in ASR with treatment condition to change in cigarette use (from the mediator variable model), W is treatment condition, and b_1 is the path from change in cigarette use to change in depressive symptoms (from the dependent variable model). *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

in Table 10. In the mediator variable model the interaction of change in ASR with treatment condition did not predict change in cigarette use ($b = -1.43$, *ns*). In the dependent variable model, change in cigarette use was unrelated to change in depressive symptoms ($b = 0.00$, *ns*). The indirect effect was nonsignificant in both community care and the intervention. Overall, then, using both intention-to-treat and as-treated

Table 11. Logistic regression analyses of change in psychological health (vitality and depressive symptoms) to long-term tobacco abstinence: intention-to-treat analysis.

	7 dPP				24 mPA			
	b	Wald	OR	95% CI	*b*	Wald	OR	95% CI
Change in Vitality								
Vitality at Baseline	−0.10	0.52	0.91	{0.70, 1.18}	−0.20	0.82	0.82	{0.54, 1.26}
Vitality at 6 Months	0.36	6.98**	1.44	{1.10, 1.88}	0.50	4.63*	1.64	{1.05, 2.58}
Change in Vitality								
Vitality at 6 Months	−0.14	0.76	0.87	{0.63, 1.19}	−0.25	0.89	0.78	{0.46, 1.31}
Vitality at 18 Months	0.58	11.49***	1.78	{1.28, 2.49}	0.83	8.14**	2.30	{1.30, 4.07}
Change in Depressive Symptoms								
Depressive Symptoms at Baseline	0.45	2.65	1.56	{0.91, 2.67}	0.69	2.48	1.99	{0.85, 4.69}
Depressive Symptoms at 6 Months	−0.70	6.66**	0.50	{0.29, 0.85}	−0.74	2.74^{+}	0.48	{0.20, 1.15}
Change in Depressive Symptoms								
Depressive Symptoms at 6 Months	−0.21	0.51	0.82	{0.47, 1.43}	−0.31	0.42	0.74	{0.29, 1.86}
Depressive Symptoms at 18 Months	−0.32	1.42	0.72	{0.43, 1.23}	−0.04	0.01	0.97	{0.42, 2.24}

Notes: 7 dPP = 7-day point prevalence tobacco abstinence, 24 mPA = 24-month prolonged abstinence from tobacco, OR = Odds ratio, 95% CI = 95% Confidence interval.
$^{+}p < 0.10$; $*p < 0.05$; $**p < 0.01$; $***p < 0.001$.

Table 12. Logistic regression analyses of change in psychological health (vitality and depressive symptoms) to long-term tobacco abstinence: as-treated analysis.

	7 dPP				24 mPA			
	b	Wald	OR	95% CI	*b*	Wald	OR	95% CI
Change in Vitality								
Vitality at Baseline	−0.22	1.98	0.80	{0.59, 1.09}	−0.28	1.53	0.76	{0.48, 1.18}
Vitality at 6 Months	0.55	11.29***	1.73	{1.26, 2.38}	0.65	6.72**	1.91	{1.17, 3.12}
Change in Vitality								
Vitality at 6 Months	−0.09	0.24	0.92	{0.65, 1.30}	−0.17	0.45	0.85	{0.52, 1.38}
Vitality at 18 Months	1.02	23.44***	2.76	{1.83, 4.17}	1.10	12.97***	2.99	{1.65, 5.44}
Change in Depressive Symptoms								
Depressive Symptoms at Baseline	0.53	3.55^{+}	1.71	{0.98, 2.97}	0.60	1.96	1.82	{0.79, 4.20}
Depressive Symptoms at 6 Months	−0.69	5.84*	0.50	{0.29, 0.88}	−0.74	2.83^{+}	0.48	{0.20, 1.13}
Change in Depressive Symptoms								
Depressive Symptoms at 6 Months	−0.35	1.43	0.70	{0.39, 1.25}	−0.35	0.63	0.71	{0.30, 1.66}
Depressive Symptoms at 18 Months	−0.13	0.25	0.88	{0.52, 1.48}	−0.08	0.05	0.92	{0.43, 1.97}

Notes: 7 dPP = 7-day point prevalence tobacco abstinence, 24 mPA = 24-month prolonged abstinence from tobacco, OR = Odds ratio, 95% CI = 95% Confidence interval.
$^{+}p < 0.10$; $*p < 0.05$; $**p < 0.01$; $***p < 0.001$.

analyses, there was no evidence of either unconditional or conditional indirect effects of change in ASR to change in depressive symptoms through change in cigarette use.

Testing research aim 2

The second aim was to examine the relation of changes in vitality and depressive symptoms to 7 dPP tobacco abstinence and 24 mPA from tobacco. Results using intention-to-treat and as-treated analyses are presented in Tables 11 and 12, respectively. In both analyses, change in vitality from baseline to 6 months and from 6 to 18 months predicted 7 dPP tobacco abstinence and 24 mPA from tobacco. Change in depressive symptoms from baseline to 6 months predicted 7 dPP tobacco abstinence and marginally predicted 24 mPA from tobacco. However, change in depressive symptoms from 6 to 18 months did not predict 7 dPP tobacco abstinence or 24 mPA from tobacco. Thus, change in vitality predicted the likelihood of attaining long-term tobacco abstinence, whereas the results for change in depressive symptoms were largely nonsignificant.

Discussion

We sought to contribute to the growing body of research on the psychological correlates of smoking behavior by examining the relations of ASR and

cigarette use to both positive (viz., vitality) and negative (viz., depressive symptoms) indices of psychological health, and the relations of those indices to long-term tobacco abstinence. Participants received either an intensive intervention designed to promote smokers' autonomy or community care, and data were collected at baseline, and at 6, 18, and 30 months post-randomization. In general, results supported our hypotheses, the implications of which we consider in detail below.

The first aim of this research was to test whether change in cigarette use would mediate the relation of change in ASR for smoking cessation to change in psychological health, and whether this indirect effect would be moderated by treatment condition. We found support for the proposed conditional indirect effect model predicting subsequent change in vitality (but not depressive symptoms). Increased ASR predicted decreased cigarette use from baseline to 6 months, and decreased cigarette use predicted increased vitality from 6 to 18 months. Change in ASR still significantly predicted change in vitality after controlling for change in cigarette use, suggesting that autonomy has important well-being consequences beyond those resulting from health-behavior change. Moreover, the relation of change in ASR to change in cigarette use was moderated by treatment condition, such that there was a stronger negative association between those variables among those in the intensive intervention. Thus, autonomy-supportive contexts seem to mobilize smokers' ability to utilize autonomy to initiate health-behavior change. These findings suggest that change in vitality (compared to depressive symptoms) is better predicted by changes in ASR and cigarette use and, therefore, vitality may help in constructing a theoretical model of the psychological correlates of smoking behavior, as called for by Hall (2004).

The second aim of this research was to examine whether change in psychological health would predict long-term tobacco abstinence. Change in vitality both during (baseline to 6 months) and after (6 to 18 months) the clinical trial predicted 7 dPP tobacco abstinence and 24 mPA from tobacco. In contrast, change in depressive symptoms was largely unrelated to long-term tobacco abstinence. These findings also underscore the importance of considering positive indices of psychological health to promote maintained health-behavior change.

This investigation was the first to examine vitality vis-à-vis tobacco abstinence. Using SDT, we proposed that vitality represents energy available to help smokers struggle effectively through the difficulties of quitting. Empirically, we demonstrated that changes in both ASR and cigarette use related to change in vitality, which in turn predicted maintained tobacco abstinence at 2 years post-intervention. Results using depressive symptoms were largely null. We do not suggest, however, that research ignore depressive symptoms and other indices of psychological distress (e.g., anxiety). Indeed, although findings are mixed, a large literature points to an association between depressive symptoms and smoking behavior. Rather, we recommend that future research attend to both positive and negative indices of psychological health to develop a more complete theory of the psychological correlates of smoking behavior.

Clinical implications

Together, these findings elucidate some of the antecedents and consequences of smokers' psychological health in the context of a clinical trial. According to SDT, the amount of energy available to the self is affected by the experiences of ASR and optimal physical health (Ryan & Frederick, 1997). In the current study, smokers who experienced increased ASR for smoking cessation reported increased vitality, and this relation was partially explained by smokers' reducing their cigarette use. Thus, our findings support the prediction that, when practitioners focus on ASR and smoking factors, an important consequence may be an increase in patients' psychological energy, which can help promote long-term tobacco abstinence. Indeed, change in vitality related positively to long-term tobacco abstinence. As such, these results underscore the importance of practitioners' considering positive indices of psychological health in developing treatments for tobacco use. Interventions that support autonomy, competence, and relatedness have been found to enhance smokers' ASR (e.g., Williams et al., 2009), which predicted enhanced vitality in the current study. Practitioners can support their patients' psychological needs by providing choice and relevant information, acknowledging perspectives, and supporting self-initiations (autonomy); establishing a plan for health-behavior change and reframing failures as short successes (competence); and relating to patients in a warm, empathic manner (relatedness).

Limitations

Several limitations deserve mention. First, both vitality and depressive symptoms were based on self-report data. Future research could use clinical assessments to understand more fully the relations of those variables to smoking cessation and the treatment conditions under which such relations occur. Second, the findings reported herein were part of a secondary analysis and were primarily correlational, although the primary analyses had examined differences in participants randomized either to an autonomy-supportive intensive intervention or community care. Thus, the results of the current report preclude a conclusion of causality.

Nonetheless, an important strength of our findings is that many of the variables were assessed at different points in time, which allowed us to determine directionality among the constructs. One notable exception to this was that changes in ASR and cigarette use were assessed concurrently (baseline to 6 months). Thus, it is possible that decreased cigarette use may predict increased ASR and increased vitality, although this is inconsistent with our theoretical model. Future research that uses additional points of assessment is needed. Third, the associations among the study variables were small in magnitude, but it is notable that our hypotheses were supported using an as-treated analysis, which reduced the sample size roughly by half. It is important for future research to replicate these results.

Conclusions

Changes in ASR and cigarette use predicted change in vitality, which in turn predicted long-term tobacco abstinence. Change in depressive symptoms was largely unrelated to ASR and tobacco abstinence. The importance of considering positive indices of psychological health in the development of clinical interventions for health-behavior change is apparent.

Acknowledgements

This research was supported in part by grants from the National Institute of Mental Health (R01-MH59594) and the National Cancer Institute (R01-CA106668) to Dr. Williams.

Notes

1. Separate conditional indirect effect models were used to examine changes in vitality and depressive symptoms.
2. Although the association between ASR and depressive symptoms has been found to be negative, it typically is small in magnitude and does not always reach conventional levels of significance.
3. The means and standard deviations presented in Tables 1 and 2 appear different because the descriptive statistics in Table 2 were computed using the entire sample of participants, whereas the descriptive statistics in Table 1 were computed separately within community care and the intensive intervention.
4. Although the interaction did not predict the mediator, we deemed it appropriate to examine the conditional indirect effects, as they were the focus of our hypothesis (K. Preacher, personal communication, November 13, 2009).
5. A significant unconditional indirect effect is not a prerequisite for testing conditional indirect effects (Preacher et al., 2007).

References

al'Absi, M., Hatsukami, D., Davis, G.L., & Wittmers, L.E. (2004). Prospective examination of effects of smoking abstinence on cortisol and withdrawal symptoms as predictors of early smoking relapse. *Drug & Alcohol Dependence, 73*, 267–278.

Anda, R.F., Williamson, D.F., Escobedo, L.G., Mast, E.E., Giovino, G.A., & Remington, P.L. (1990). Depression and the dynamics of smoking: A national perspective. *The Journal of the American Medical Association, 264*, 1541–1545.

Berlin, I., & Covey, L.S. (2006). Pre-cessation depressive mood predicts failure to quit smoking: The role of coping and personality traits. *Addiction, 101*, 1814–1821.

Centers for Disease Control (1999). Achievements in public health, 1900–1999: Tobacco use—United States, 1900–1999. *Morbidity & Mortality Weekly Report, 48*, 986–999.

Cicchetti, D. (1991). Fractures in the crystal: Developmental psychopathology and the emergence of self. *Developmental Review, 11*, 271–287.

Cook, J.W., Spring, B., McChargue, D.E., Borrelli, B., Hitsman, B., Niaura, R., et al. (2004). Influence of fluoxetine on positive and negative affect in a clinic-based smoking cessation trial. *Psychopharmacology, 173*, 153–159.

Cook, J.W., Spring, B., McChargue, D., & Hedeker, D. (2004). Hedonic capacity, cigarette craving, and diminished positive mood. *Nicotine & Tobacco Research, 6*, 39–47.

Davidson, R.J. (1992). Anterior asymmetry and the nature of emotion. *Brain and Cognition, 20*, 125–151.

Deci, E.L., & Ryan, R.M. (2000). The 'what' and 'why' of goal pursuits: Human needs and the self-determination of behavior. *Psychological Inquiry, 11*, 227–268.

Dilsaver, S.C., Pariser, S.F., Churchill, C.M., & Larson, C.N. (1990). Is there a relationship between failing efforts to stop smoking and depression? *Journal of Clinical Psychopharmacology, 10*, 153–154.

Doran, N., Spring, B., Borrelli, B., McChargue, D., Hitsman, B., Niaura, R., et al. (2006). Elevated positive mood: A mixed blessing for abstinence. *Psychology of Addictive Behaviors, 20*, 36–43.

Fiore, M.C., Bailey, W.C., Cohen, S.J., Dorfman, S.F., Goldstein, M.G., Gritz, E.R., et al. (2000). *Treating tobacco use and dependence*. Rockville, MD: US Department of Health and Human Services.

Fiore, M.C., Jaén, C.R., Baker, T.B., Bailey, W.C., Benowitz, N.L., Curry, S.J., et al. (2008). *Treating tobacco use and dependence: 2008 update*. Rockville, MD: US Department of Health and Human Services.

Frerichs, R.R., Aneshensel, C.S., Clark, V.A., & Yokopenic, P. (1981). Smoking and depression: A community survey. *American Journal of Public Health, 71*, 637–640.

Hall, S.M. (2004). The Covey-Hitsman exchange. *Nicotine & Tobacco Research, 6*, 751–752.

Hayford, K.E., Patten, C.A., Rummans, T.A., Schroeder, D.R., Offord, K.P., Croghan, I.T., et al. (1999). Efficacy of bupropion for smoking cessation in smokers with a former history of major depression or alcoholism. *British Journal of Psychiatry, 174*, 173–178.

Hughes, J.R., Keely, J.P., Niaura, R.S., Ossip-Klein, D.J., Richmond, R.L., & Swan, G.E. (2003). Measures of abstinence in clinical trials: Issues and recommendations. *Nicotine & Tobacco Research, 5*, 13–25.

Kahler, C.W., Brown, R.A., Ramsey, S.E., Niaura, R., Abrams, D.B., Goldstein, M.G., et al. (2002). Negative mood, depressive symptoms, and major depression after smoking cessation treatment in smokers with a history of major depressive disorder. *Journal of Abnormal Psychology, 111*, 670–675.

Kandel, D.B., & Davies, M. (1986). Adult sequelae of adolescent depressive symptoms. *Archives of General Psychiatry, 43*, 255–262.

Killen, J.D., Fortmann, S.P., Kraemer, H.C., Varady, A.N., Davis, L., & Newman, B. (1996). Interactive effects of depression symptoms, nicotine dependence, and weight change on late smoking relapse. *Journal of Consulting and Clinical Psychology, 64*, 1060–1067.

Kodl, M.M., Fu, S.S., Willenbring, M.L., Gravely, A., Nelson, D.B., & Joseph, A.M. (2008). The impact of depressive symptoms on alcohol and cigarette consumption following treatment for alcohol and nicotine dependence. *Alcoholism: Clinical and Experimental Research, 32*, 92–99.

Leventhal, A.M., Ramsey, S.E., Brown, R.A., LaChance, H.R., & Kahler, C.W. (2008). Dimensions of depressive symptoms and smoking cessation. *Nicotine & Tobacco Research, 10*, 507–517.

Malkin, S.A., & Allen, D.L. (1980). Differential characteristics of adolescent smokers and non-smokers. *Journal of Family Practice, 10*, 437–440.

Mokdad, A.H., Marks, J.S., Stroup, D.F., & Gerberding, J.L. (2004). Actual causes of death in the United States, 2000. *The Journal of the American Medical Association, 291*, 1238–1245.

Moller, A.C., Deci, E.L., & Ryan, R.M. (2006). Choice and ego-depletion: The moderating role of autonomy. *Personality and Social Psychology Bulletin, 32*, 1024–1036.

Munafò, M.R., Hitsman, B., Rende, R., Metcalfe, C., & Niaura, R. (2007). Effects of progression to cigarette smoking on depressed mood in adolescents: Evidence from the National Longitudinal Study of Adolescent Health. *Addiction, 103*, 162–171.

National Cholesterol Education Program (1997). *Cholesterol lowering in the patient with coronary heart disease: physician monograph* (NIH Publication No. 97-3794). Bethesda, MD: Institutes of Health, National Heart, Lung, and Blood Institute.

Niemiec, C.P., Lynch, M.F., Vansteenkiste, M., Bernstein, J., Deci, E.L., & Ryan, R.M. (2006). The antecedents and consequences of autonomous self-regulation for college: A self-determination theory perspective on socialization. *Journal of Adolescence, 29*, 761–775.

Niemiec, C.P., Ryan, R.M., & Deci, E.L. (2010). Self-determination theory and the relation of autonomy to self-regulatory processes and personality development. In R.H. Hoyle (Ed.), *Handbook of personality and self-regulation* (pp. 169–191). Malden, MA: Blackwell Publishing.

Niemiec, C.P., Ryan, R.M., Deci, E.L., & Williams, G.C. (2009). Aspiring to physical health: The role of aspirations for physical health in facilitating long-term tobacco abstinence. *Patient Education and Counseling, 74*, 250–257.

Preacher, K.J., & Hayes, A.F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments, & Computers, 36*, 717–731.

Preacher, K.J., Rucker, D.D., & Hayes, A.F. (2007). Addressing moderated mediation hypotheses: Theory, methods, and prescriptions. *Multivariate Behavioral Research, 42*, 185–227.

Presson, C.C., Chassin, L., & Sherman, S.J. (2002). Psychosocial antecedents of tobacco chipping. *Health Psychology, 21*, 384–392.

Prinstein, M.J., & La Greca, A.M. (2009). Childhood depressive symptoms and adolescent cigarette use: A six-year longitudinal study controlling for peer relations correlates. *Health Psychology, 28*, 283–291.

Rabois, D., & Haaga, D.A.F. (2003). The influence of cognitive coping and mood on smokers' self-efficacy and temptation. *Addictive Behaviors, 28*, 561–573.

Radloff, L.S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385–401.

Russell, J.A., & Carroll, J.M. (1999). On the bipolarity of positive and negative affect. *Psychological Bulletin, 125*, 3–30.

Ryan, R.M., & Deci, E.L. (2000). Self-determination theory and the facilitation of intrinsic motivation, social development, and well-being. *American Psychologist, 55*, 68–78.

Ryan, R.M., & Deci, E.L. (2008). From ego depletion to vitality: Theory and findings concerning the facilitation of energy available to the self. *Social and Personality Psychology Compass, 2*, 702–717.

Ryan, R.M., & Frederick, C. (1997). On energy, personality, and health: Subjective vitality as a dynamic reflection of well-being. *Journal of Personality, 65*, 529–565.

Steuber, T.L., & Danner, F. (2006). Adolescent smoking and depression: Which comes first? *Addictive Behaviors, 31*, 133–136.

Surgeon General (1979). *Smoking and health: A report of the Surgeon General* (DHEW Publication No. PHS 79-50066). Washington, DC: US Government Printing Office.

Waal-Manning, H.J., & de Hamel, F.A. (1978). Smoking habit and psychometric scores: A community study. *New Zealand Medical Journal, 88*, 188–191.

Watson, D., Clark, L.A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063–1070.

Williams, G.C., Deci, E.L., & Ryan, R.M. (1998). Building health-care partnerships by supporting autonomy: Promoting maintained behavior change and positive health outcomes. In A.L. Suchman, P. Hinton-Walker, & R. Botelho (Eds.), *Partnerships in healthcare: Transforming relational process* (pp. 67–87). Rochester, NY: University of Rochester Press.

Williams, G.C., Grow, V.M., Freedman, Z.R., Ryan, R.M., & Deci, E.L. (1996). Motivational predictors of weight loss and weight-loss maintenance. *Journal of Personality and Social Psychology, 70*, 115–126.

Williams, G.C., McGregor, H., Sharp, D., Kouides, R.W., Lévesque, C., Ryan, R.M., et al. (2006). A self-

determination multiple risk intervention trial to improve smokers' health. *Journal of General Internal Medicine, 21*, 1288–1294.

Williams, G.C., McGregor, H.A., Sharp, D., Levesque, C., Kouides, R.W., Ryan, R.M., et al. (2006). Testing a self-determination theory intervention for motivating tobacco cessation: Supporting autonomy and competence in a clinical trial. *Health Psychology, 25*, 91–101.

Williams, G.C., Minicucci, D.S., Kouides, R.W., Levesque, C.S., Chirkov, V.I., Ryan, R.M., et al. (2002). Self-determination, smoking, diet and health. *Health Education Research, 17*, 512–521.

Williams, G.C., Niemiec, C.P., Patrick, H., Ryan, R.M., & Deci, E.L. (2009). The importance of supporting autonomy and perceived competence in facilitating long-term tobacco abstinence. *Annals of Behavioral Medicine, 37*, 315–324.

Wu, L.-T., & Anthony, J.C. (1999). Tobacco smoking and depressed mood in late childhood and early adolescence. *American Journal of Public Health, 89*, 1837–1840.