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Impact of a brief motivational smoking cessation intervention: The Get PHIT trial

Jennifer B. McClure, PhD, Evette J. Ludman, PhD, Louis Grothaus, MA, Chester Pabiniak, and Julie Richards, MPH

All authors are at the Group Health Center for Health Studies in Seattle, Washington.

Abstract

BACKGROUND—Few studies have rigorously evaluated whether providing biologically-based health risk feedback increases smokers' motivation to quit and long-term abstinence above standard interventions.

DESIGN—Randomized controlled trial conducted from 2005-2008. Data were analyzed in 2008.

SETTING/PARTICIPANTS—Smokers (n = 536) were recruited from the community, regardless of their interest in quitting smoking.

INTERVENTION—Smokers received brief (~20 minutes), personally-tailored counseling based on their lung functioning, carbon monoxide (CO) exposure, and smoking-related health conditions versus generic smoking risk information and personalized counseling about their diet, BMI, and physical activity. All were advised to quit smoking and offered access to a free phone-counseling program.

MAIN OUTCOME MEASURES—Treatment utilization and abstinence at 6 and 12 months post-intervention.

RESULTS—Experimental participants demonstrated no greater motivation to quit, use of treatment services, or abstinence compared to controls at either follow-up. In fact, controls reported greater motivation to quit at 12 months (mean 3.42 vs. 3.20, $P = .03$), use of pharmacotherapy at 6 months (37.8% vs. 28.0%, $P = .02$), and 30 day PPA at 6 months after controlling for relevant covariates (10.8% vs. 6.4%, adjusted $P = .04$).

CONCLUSIONS—The present study found no support for adding a personalized health risk assessment emphasizing lung health and CO exposure to generic cessation advice and counseling for community-based smokers not otherwise seeking treatment.

Keywords

smoking cessation; spirometry; motivation; tobacco; health risk assessment; lung age; carbon monoxide

Introduction

Nearly 70% of smokers want to quit smoking someday,¹ but only 20% or less are ready to take action in the next month when asked²⁻⁵. Of those who do quit, only 20-30% use proven effective behavioral or pharmacological treatment⁶. Most smokers prefer to try quitting on

their own, without benefit of formal intervention. To achieve the Center for Disease Control's goal of reducing smoking prevalence to $\leq 12\%$ of adults⁷, ever assertive efforts are needed to proactively reach out to smokers, build and strengthen their motivation for quitting, promote utilization of empirically-validated cessation programs, and thereby, increase tobacco abstinence.

Motivation is a complex construct and motivating behavior change can be difficult. Many of the leading health behavior change theories (e.g., the Health Belief Model, Health Decision Model, and Protection Motivation Theory)⁸⁻¹⁰ suggest that health behavior is influenced, at least in part, by one's perceived disease susceptibility. Increasing one's awareness of personal risk or harm caused by unhealthy habits could, theoretically, increase motivation for behavior change. When paired with accessible, effective treatment providing feedback about biological indices of smoking-related harm this could also promote treatment utilization and thereby help smokers quit.

Prior research in this area has evaluated the use of ultrasound images of atherosclerotic plaque^{11, 12}, lung age and respiratory symptom feedback¹³, pulmonary functioning feedback¹⁴⁻¹⁸, genetic marker feedback¹⁹, carbon monoxide exposure^{16, 17, 19, 20}, and spiral CT lung scans²¹ to promote smoking cessation; however, the empirical support for using personalized health risk feedback to motivate behavior change is mixed. Several recent literature reviews concluded that there have been too few studies of acceptable methodological quality to draw any firm conclusions about the utility of this approach, both with respect to health behavior change in general²² and smoking cessation in particular²³⁻²⁷. Additional research was called for in the latest Clinical Practice Guideline for Treating Tobacco Use and Dependence²⁵. Furthermore, it is an open question whether spirometry screening is an effective cessation aid. The National Lung Health Education Program concluded that "spirometry testing probably enhances smoking cessation rates"²⁸ and routine use of office-based spirometry has been called for with smokers²⁹, but a recent empirical review concluded that "available evidence is insufficient to determine whether obtaining spirometric values and providing that information to patients improves smoking cessation"²⁷. Thus, further evaluation of this intervention strategy is needed.

The goal of Get PHIT (Proactive Health Intervention for Tobacco-Users) was to provide feedback on participants' carbon monoxide (CO) exposure (expired CO and estimated carboxyhemoglobin [COHb] levels), pulmonary functioning assessed via portable office-based spirometry, and self-reported smoking-related symptoms in order to build and strengthen their motivation for receiving treatment and quitting smoking. The intent was to design a brief, one time intervention applicable to all smokers, regardless of their readiness to quit; create a "teachable moment," regardless of the risk assessment findings, which could be offered outside a traditional medical encounter; and include standardized feedback so the intervention could be replicated. Portable spirometry and CO assessment were used as the basis for the feedback because they were compatible with these goals. Expired CO is elevated in recent smokers and, therefore, a relevant marker of harm. It is also inexpensive and easy to assess. Similarly, spirometry assessment allows a quick and inexpensive evaluation of smoking-related risk which can be used to justify advice to quit smoking whether or not there is demonstrable evidence of lung impairment. Persons with abnormal lung functioning can be advised to quit based on the evidence of harm. Persons with no lung impairment can be advised to quit on the basis that continued smoking will likely result in impairment based on epidemiological data³⁰.

It was hypothesized that smokers who received the personalized risk feedback would have increased motivation for quitting in the short-term, mediated in part by increased perceived disease susceptibility. Increased motivation would, in turn, lead to increased uptake of a free

empirically-validated phone counseling program and higher long-term cessation rates in the long term, compared to persons who received generic smoking risk information and personalized advice regarding other health behaviors. The immediate and short-term impact of this intervention on motivation and perceived disease susceptibility has been previously reported³¹. Longer-term effects on treatment utilization and cessation are reported here.

Methods

Setting and Participants

Study enrollment began in March, 2005 and ended September, 2007. Data collection ended in September, 2008 and analyses were conducted September to November 2008. All activities were reviewed and approved by the Group Health Institutional Review Board. This trial is registered with ClinicalTrials.gov (NCT00169260).

Smokers do not typically seek cessation treatment unless they are ready to quit. In order to recruit smokers at all stages of readiness to change, likely smokers were proactively identified via health plan records, data from the Washington State Quitline (limited to local persons who had received a single counseling call more than a year ago), and a purchased mailing list of smokers. Each person was mailed an introductory study invitation letter and then called to be screened for interest and eligibility. Some participants (n = 48) were also recruited through ads placed in local media, public clinics, and other local venues. Additional recruitment details are available elsewhere^{31, 32}. The study was presented as a health risk screening intervention study, as opposed to a smoking cessation trial, and people were informed that they did not have to want to quitting smoking to participate.

Potentially eligible smokers were scheduled for an in-person appointment. Attendees were eligible if they were: 18 or older; could read and write in English; were not currently receiving cessation treatment; had no significant physical or mental impairments that prevented use of a computer or phone, or impaired their comprehension ability; and reported no medical contraindications for spirometry assessment. Participation was also limited to smokers with elevated expired CO levels consistent with current smoking (≥ 10 ppm)³³ and who either smoked an average of 15 cigarettes per day for the past year or smoked at least 10 cigarettes per day, but had smoked for 10 years or more. The latter criteria were used to screen out new and light smokers who would be less likely to have evidence of lung impairment. Eligible smokers were randomized to treatment using an automated randomization algorithm, completed a baseline survey and relevant health risk screening, and received a single brief counseling session.

Intervention

The intervention was similar in contact time and structure for both treatment groups. Each group received a personalized health risk report written at an 8th grade reading level and brief (~20 minutes) counseling based on their risk assessment results, using the principles of motivational interviewing (MI)^{34, 35}. The health assessment and interventions were conducted by a health educator who was trained in spirometry and brief MI. All participants were advised to quit smoking, given self-help smoking cessation materials (Clearing the Air)³⁶, and given access to an empirically-validated, free phone-counseling program which they could enroll in anytime in the next 12 months free of charge if they decided to quit smoking. The phone counseling was provided by Free & Clear, Inc. While the contact and structure were similar, the motivational content and health behavior feedback varied by treatment group as described below.

Experimental Intervention—Participants in the experimental condition participated in a health-risk assessment focused on CO-exposure and lung functioning. CO level was assessed using a Bedfont MicroIII monitor and lung functioning was assessed using a Bedfont MicroIII monitor. Participants also completed a self-report survey of their medical history. Each participant then received a personally-tailored report which detailed their self-reported smoking related symptoms (e.g., persistent cough) and diagnosed smoking-related medical conditions; included their CO level (expired and estimated COHb level) and normative CO values for non-smokers; explained the spirometry test and results; and included a standardized graph depicting the average decline in lung functioning over time for a never smoker, a smoker who quits at age 45, one who quits at age 65, and one who never quits, based on epidemiological data (adapted from Fletcher & Peto ³⁰). The reports also contained standardized text highlighting the association between smoking and various smoking-related conditions (e.g., heart disease, cancer, cataracts), the impact of smoking on lung functioning, the association between smoking and CO exposure, and the health effects of chronic and acute CO exposure. Spirometry feedback focused on three measures: forced vital capacity (FVC), or the maximum volume of air that can be forcefully exhaled after maximal inhalation; forced expiratory volume (FEV₁), or the volume of air that can be forcefully exhaled in the first second of the FVC exercise; and FEF₂₅₋₇₅, a measure of air flow during the middle portion of the test and indicator of small airway obstruction. Performance was measured in terms of the percent predicted of normal as calculated by the spirometer. Participants' test values were presented in a table, along with a brief description of each outcome measure (FEV₁, FVC, and FEF₂₅₋₇₅), and a qualitative interpretation of the test results based on standard cut-off values reflecting normal functioning, mild impairment, moderate impairment, or severe impairment for FVC and FEV₁, and normal vs. reduced airflow for FEF₂₅₋₇₅. The goal was not to make a clinical diagnosis, but to provide standardized feedback based on objective test measures. Cutoff values were established by a consulting pulmonologist. If FEV₁ percent predicted was less than 80%, suggesting possible impairment, lung age was calculated ³⁷ and presented.

Control Intervention—The control group report contained generic feedback about the risks of smoking, advice to quit, and instructions for accessing the free phone-counseling program. Additionally, control participants received personalized written and verbal feedback highlighting relevant changes they should make based on their self-reported diet, physical activity level, and body mass index.

Assessment

Participants were surveyed at baseline, 6, and 12 months post-enrollment. Primary long-term outcomes were use of the free counseling program based on Free & Clear treatment records and self-reported 7 day point prevalent abstinence (PPA). Motivation to quit was examined as a secondary outcome and included self-reported motivation for quitting measured on a 5 point Likert scale (from 'not at all' to 'extremely' motivated) and presence of an intentional 24-hour quit attempt. Self-reported use of other smoking cessation treatments (e.g., pharmacotherapy or other interventions) and 30 day PPA were also examined.

Additional assessment measures included participant demographics, smoking history and exposure to smokers in the home, the Fagerstrom Test of Nicotine Dependence ³⁸, stage of change for smoking cessation ^{39, 40}, insurance coverage for cessation treatment, likelihood of quitting in the next month, and self-efficacy for quitting smoking. Likelihood of quitting and self-efficacy were assessed on a five-point Likert scale ranging from 'not at all' to 'extremely'. At follow-up, participants were also asked whether they had spoken with their physician about the feedback received.

Statistical Analyses

Descriptive statistics were used to characterize participants by treatment group. Groups were compared using t-tests for means and chi-square tests for percentages. Primary outcomes (7-day PPA and use of the provided phone counseling) and secondary outcomes were identified *a priori* and assessed at 6 and 12 months. By convention, PPA was calculated two ways - using an intent-to-treat (ITT) analysis in which missing respondents were counted as smokers and using a respondent-only analysis. Adjusted analyses were also run using logistic and least-squares regression to control for five baseline predictors, also chosen *a priori*, based on their known association with cessation (FTND score, self-efficacy for quitting, number of quit attempts in prior year, likelihood of quitting in next month, and whether other smokers lived in the household). The goal of this adjustment was to improve power by reducing the standard error of the treatment effect (no variables differed significantly at baseline). The study had 80% power to detect an intervention effect equal to 9.4% or an odds ratio of 1.87 for the primary outcome, 7 day PPA. Power calculations assumed a 2-sided significance test at the .05 level and an abstinence rate of 14.1% in the control group. Power was calculated using the normal approximation to the chi-square test without continuity correction.

Results

Participants

Five hundred forty-two participants were randomized to treatment, but six were removed from the sample post-randomization and pre-analysis because their CO level did not meet cut-off and they should not have been randomized (see Figure 1).

Baseline characteristics are presented in Table 1. Groups did not significantly differ pre-intervention. Among experimental participants, the average expired CO level was 26 ppm and 37% of this group had lung functioning indicative of at least mild impairment based on their FEV₁, FVC, or FEF₂₅₋₇₅ scores. Among those with evidence of lung impairment, the average chronological age was 50.9 years and the average estimated lung age was 79.8 years.

Treatment Impact by Intervention Group

Treatment utilization—The proportion of people who enrolled in the provided phone-counseling program or self-reported use of other cessation treatments by each follow-up is presented in Table 2. Control participants were more likely to report use of pharmacotherapy for smoking cessation at six month follow-up (38% vs. 28%, $P = .02$). No other significant group differences were observed.

Abstinence—Seven day PPA rates were similar between groups, both for the ITT and respondent-only analyses, but controls reported greater 30 day PPA at 6 months after adjusting for covariates (see Table 2).

Motivation to quit and medical follow-up—Additional indices of treatment impact are presented in Table 3. There was no difference in whether groups made a quit attempt or talked with their physician as a result of the intervention. Continuing smokers were equally motivated to quit at six months, but controls reported slightly higher motivation to quit at one year ($P = .03$).

Missing Data—There was no evidence of a response bias based on comparisons of response rates for each outcome presented in Tables 2 and 3 at each follow-up. Seven day PPA was missing for 9% of controls and 8% of experimentals ($P = .74$) at six months and 13% of each group at one year ($P = .77$). Response rates for the other outcomes in Tables 2 and 3 were similar with all P values greater than 0.50.

Discussion

The current study examined the effectiveness of a brief, motivational intervention using biologically-based feedback to alter behavioral smoking outcomes. The intent was to build and strengthen motivation to quit through a personalized health risk assessment and brief motivational intervention, reduce common barriers to empirically validated treatment by offering free access to phone counseling which could be utilized anytime in the next year, and thereby increase treatment uptake and abstinence rates. The intervention was also purposefully designed to be applicable to all smokers, regardless of their readiness to quit. The experimental group did not exhibit higher levels of motivation to quit, greater use of the provided cessation treatment, or higher abstinence rates at either follow-up compared to the control group. In fact, controls reported a higher level of motivation to quit at one year, were more likely to have used pharmacotherapy at six months and had a higher 30 day PPA rate at 6 months.

An important question when the intervention does not have a statistically significant benefit is whether the intervention is ineffective or whether the trial lacked statistical power to detect the intervention effect. To answer this question, confidence intervals were examined for PPA and treatment utilization. Confidence intervals (CI) indicate what range of effects, both positive and negative, are consistent with the trial data. Based on the 95% CI of -7.8% to +3.6% for the difference (intervention minus control) in 7 day ITT PPA at 6 months, our results are consistent with the true underlying effect of the intervention lying somewhere between a 3.6% decrease and a 7.8% increase in abstinence rates. In other words, one can rule out that the intervention increased abstinence rates by more than 3.6%. In contrast, one cannot rule out that the intervention may have decreased abstinence rates by up to about 8%. Virtually identical results were seen at one year: the 95% CI was -7.7% to +4.1% for ITT 7 day PPA. The 95% CI for the proportion enrolling in the provided cessation counseling program at one year was -13.0% to 2.2%, indicating that the intervention benefit was at most 2.2%, but the data are consistent with up to a 13% reduction in the utilization rate. In sum, we can rule out all but small benefits due to the intervention.

Several explanations for the lack of an experimental intervention effect should be considered. These may be considered caveats or study limitations. First, counseling smokers about their increased risk of lung disease and CO exposure simply may not be an effective way to promote cessation, or if so, it may require more than a one-time, brief intervention to present and discuss these risks. It is well-documented that abstinence is correlated with intervention dose.²⁵ The current study provided access to intensive counseling, but more proactive outreach may be required to motivate smokers to take advantage of this resource. Next, the control group received an active intervention, designed to match the experimental treatment in terms of contact time, focus on smokers' health, and access to self-help materials and subsequent counseling. By holding these features constant, the additional contribution of the personalized smoking-risk assessment could be better evaluated. However, it is possible that the control intervention had an important effect on motivation, treatment utilization, and abstinence, which would explain the apparent trend toward better outcomes in the control group. Based on a meta-analysis of the literature, the estimated abstinence rates when no advice to quit or intervention are provided is 7.9%,²⁵ compared to the 14% - 15% 7 day PPA ITT rates seen among controls in this trial. Thus, if a no-treatment intervention had been given to controls, a more robust intervention effect may have been seen. The lack of an experimental treatment effect could also have resulted from the population studied. If the population were restricted to smokers ready to quit, rather than all smokers, participants might have been more receptive to change, but this would defeat the important goal of identifying interventions which are effective among *all* smokers, not simply the minority ready to take action. It is also possible that the experimental intervention undermined motivation and attempts to quit smoking, rather than promoting them. Telling some smokers they had no demonstrable evidence of lung impairment may have

inadvertently reinforced continued smoking, dampening abstinence rates in the experimental group. If this occurred, the impact was not substantial enough to have caused a marked treatment group difference in cessation outcomes, but the possibility of an unintended impact among some participants must be considered. Finally, abstinence rates may have been inflated by self-report, since biochemical verification was not obtained, but there is no reason to expect a response bias favoring the control intervention, given the nature of the minimal intervention. Biochemical verification is not recommended in this type of minimal contact intervention and may, in fact, introduce a selection bias unrelated to smoking status.³³ Moreover, prior research has shown that the rate of under-reporting of smoking, particularly for brief intervention studies, is minimal.⁴¹⁻⁴³

This study has a number of strengths, including its focus on action-oriented behavioral outcomes (treatment utilization and quitting smoking), inclusion of all smokers and not just those ready to quit, the use of treatment records to verify enrollment in the provided phone program, and a well-controlled intervention design. Additional detail on the intervention and measures used to ensure treatment fidelity are discussed elsewhere.³¹ The use of an active control may have obscured an intervention effect, but it was a reasonable comparison group since our goal was to identify the unique contribution of the personalized, biologically-based, smoking-risk feedback on outcomes. From this perspective, the intervention did not have a significant effect.

In sum, the present study found no support for adding a personalized health risk assessment emphasizing lung health and CO exposure to generic cessation advice and counseling for community-based smokers not otherwise seeking treatment. Despite this, it would be premature to conclude that personalized risk assessments and motivational counseling cannot be an effective part of an intervention for smoking cessation. Recent evidence shows that smokers newly diagnosed with stroke, cancer, lung disease, heart disease, or diabetes are 3.2 times more likely to quit smoking compared to smokers not receiving a recent health diagnosis.⁴⁴ Similar results have been reported by others.⁴⁵⁻⁴⁷ Thus, there is something about a change in one's health status that can be motivating for smoking cessation. However, it may be that increased disease risk alone is not sufficient to lead to sustained behavior change for most smokers and the absence of demonstrable health effects may undermine efforts to quit. As such, targeting smokers with new or worsening medical conditions may be more a useful strategy for using health-related feedback to promote cessation.

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Figure 1.
Overview of screening, enrollment, randomization, and follow-up data collection

Table 1

Baseline demographic characteristics of study sample

	Overall (n = 542)	Control (n= 269)	Experimental (n=267)	P
	%	%	%	
Female	53.2	52.8	53.6	0.86
White	82.4	80.9	83.8	0.37
Married/living with partner	55.6	58.4	53.2	0.48
Live with other smokers	37.7	38.6	37.1	0.72
Education				
Some college or greater	75.5	78.1	72.9	0.17
Insurance coverage for tobacco treatment	42.0	42.4	41.5	0.70
Stage of change				0.99
Precontemplation	24.8	25.1	24.5	--
Contemplation	50.8	50.6	50.9	--
Preparation	24.4	24.3	24.5	--
Lung Impairment ^a	--	--	37.1	--
Impaired FVC	--	--	26.6	--
Impaired FEV ₁	--	--	33.3	--
Impaired FEF ₂₅₋₇₅	--	--	13.1	--

	Mean (SD) ^c	Mean (SD) ^c	Mean (SD) ^c	P
Age	50.8 (9.8)	50.6 (9.5)	50.9 (10.1)	0.74
Cigarettes/day	20.7 (8.2)	20.6 (8.0)	20.8 (8.4)	0.79
FTND	5.12 (1.96)	5.22 (1.93)	5.03 (2.0)	0.27
Prior quit attempts – ever	10.1 (18.0)	9.7 (16.8)	10.5 (19.1)	0.58
Prior quit attempts – last year	1.56 (6.3)	1.59 (7.0)	1.53 (5.7)	0.92
Expired CO	26.5 (10.8)	26.7 (11.1)	26.3 (10.6)	0.63
Self-efficacy for quitting ^b	2.96 (1.02)	2.89 (1.04)	3.04 (0.99)	0.08
Motivation for quitting ^b	3.49 (0.97)	3.50 (0.98)	3.48 (0.95)	0.80
Likelihood of quitting in next month ^b	2.60 (1.09)	2.58 (1.11)	2.62 (1.07)	0.64

^a Defined as spirometry performance indicative of impairment on FEV₁, FVC, or FEF₂₅₋₇₅. Experimental participants only.

^b Likert scale ranging from 1 to 5 (from 'not at all' to 'extremely').

^c SD = standard deviation.

Table 2

Treatment utilization and abstinence among intervention groups

Treatment utilization	Control (n= 269)		Experimental (n= 267)		Unadjusted		Adjusted ^a	
	%		%		OR ^b	P	OR ^b	P
Treatment utilization								
Enrolled in provided phone counseling ^c								
6 months	22.3		20.2		0.88	0.56	0.87	0.51
12 months	30.9		25.5		0.77	0.17	0.75	0.15
Used self-help materials ^d								
6 months	30.4		25.1		0.77	0.19	0.75	0.16
12 months	37.7		33.7		0.84	0.36	0.84	0.36
Used other counseling ^d								
6 months	4.5		4.1		0.91	0.86	0.98	0.96
12 months	7.1		8.1		1.15	0.67	1.20	0.59
Used pharmacotherapy ^d								
6 months	37.8		28.0		0.64	0.02	0.65	0.03
12 months	45.6		38.3		0.74	0.10	0.76	0.14
Abstinence								
7 Day PPA – ITT ^e								
6 months	14.1		12.0		0.83	0.46	0.77	0.33
12 months	14.9		13.1		0.86	0.56	0.80	0.38
7 Day PPA – Respondents ^f								
6 months	15.4		13.2		0.84	0.48	0.76	0.31
12 months	17.0		15.2		0.88	0.58	0.79	0.37
30 Day PPA – ITT ^e								
6 months	10.8		6.4		0.56	0.07	0.51	0.04
12 months	13.0		10.9		0.82	0.44	0.77	0.34
30 Day PPA – Respondents ^f								
6 months	11.7		7.0		0.57	0.07	0.50	0.04
12 months	14.9		12.6		0.82	0.46	0.76	0.33

^a Analyses adjusted for baseline FTND score, self-efficacy, number of quit attempts in prior year, likelihood of quitting in next month, and whether other smokers lived in the household.

^b OR = Odds Ratio

^c Proportion who enrolled in the provided free phone-counseling program by each assessment, based on automated treatment records.

^d Cumulative proportion who self-reported use of other smoking cessation treatments by each follow-up. Counseling included programs other than the provided phone-counseling program. Pharmacotherapy included use of any medications to quit smoking, including: nicotine replacement products, bupropion, and varenicline.

^e ITT PPA = intent to treat point prevalent abstinence.

^f PPA for only those participants who provided smoking status data at follow-up.

Table 3

Motivation to quit indices and self-reported medical follow-up

	Control (n=269)		Experimental (n=267)		Unadjusted		Adjusted ^a	
	Mean		Mean		Difference ^b	P	Difference ^b	P
Motivation to quit ^c								
6 months	3.40		3.26		-0.14	0.16	-0.14	0.12
12 months	3.42		3.20		-0.22	0.03	-0.21	0.03
Quit attempt								
6 months	47.4	50.6	1.14	0.47	1.10	0.63		
12 months	62.4	61.5	0.96	0.84	0.91	0.66		
Talk to doctor								
6 months	30.6	32.9	1.11	0.59	1.10	0.62		
12 months	46.2	54.4	1.39	0.08	1.44	0.06		

^aAnalyses adjusted for baseline FIND score, self-efficacy, number of quit attempts in prior year, likelihood of quitting in next month, and whether other smokers lived in the household.^bDifference between mean scores.^cLikert scale ranging from 1 to 5 (from 'not at all' to 'extremely'). Motivation to quit assessed among continuing smokers only.^dOR = Odds ratio.