Table 1
Participant Characteristics

	All smokers (N = 160)	Intervention (N = 81)[a]	Control (N = 79)[a]
Sociodemographic characteristics			
Age, *M* (*SD*)	31.7 (10.7)	30.9 (10.7)	32.6 (10.8)
Male, % (*n*)	56.3 (90)	55.6 (45)	57.0 (45)
IMD, *M* (*SD*)[b]	32.0 (13.1)	32.2 (13.3)	31.7 (12.9)
BMI, *M* (*SD*)	23.9 (4.0)	23.6 (4.1)	24.1 (3.8)
Smoking characteristics			
Cigarettes per day, *M* (*SD*)	13.8 (5.9)	13.4 (5.8)	14.3 (6.0)
Length of time of smoking in years, *M* (*SD*)	14.3 (11.1)	13.7 (11.0)	15.0 (11.3)
HSI, *M* (*SD*)[c]	2.4 (1.5)	2.3 (1.6)	2.5 (1.5)
Quit attempt in last 5 years, % (*n*)	56.3 (90)	54.3 (44)	58.2 (46)
Want to quit next month, % (*n*)	11.3 (18)	11.1 (9)	11.4 (9)
Baseline cotinine levels in ng/ml, *M* (*SD*)	224 (141)	211 (139)	237 (144)
Post-cigarette CO level in ppm, *M* (*SD*)[c]	18.5 (7.7)	18.0 (7.5)	19.1 (7.9)

Note. IMD = Index of Multiple Deprivation; BMI = body mass index; HSI = Heaviness of Smoking Index (scale = 0–6); ppm = parts per million.
[a] There were no baseline differences between groups. [b] 25 cases missing. [c] 2 cases missing.

Measures

Biomarkers (expired air CO and cotinine). A standard monitor (Smokerlyzer, Bedfont Scientific Ltd, Kent, United Kingdom) was used to obtain expired air CO levels. A reading was taken before and after having smoked a cigarette following a minimum of an half-hour interval of not smoking. CO monitors provide a valid and reliable measure of expired air CO levels (Jarvis, Belcher, Vesey, & Hutchison, 1986), which have been related to a number of lung diseases including chronic obstructive pulmonary disease (COPD), cystic fibrosis, and asthma (Kharitonov & Barnes, 2002), as well as lung cancer (Law, Morris, Watt, & Wald, 1997), and CO is one of the cigarette constituents believed to be involved in cardiovascular disease (Ludvig, Miner, & Eisenberg, 2005).

Saliva samples were collected using a dental roll, which participants were asked to keep in the mouth until saturated. Samples were assayed for cotinine, a major metabolite of nicotine that provides a very sensitive and specific quantitative measurement of tobacco intake, using a tandem mass spectrometric method (Feyerabend & Russell, 1990).

Sociodemographic characteristics (T1). Participants were asked about general demographic characteristics (age, gender) in the baseline questionnaire. In addition, deprivation level was determined using the Index of Multiple Deprivation (IMD), a measure of relative poverty based on post codes (Jordan, Roderick, & Martin, 2004). Body mass index (BMI) was calculated from participants' self-reported height and weight (kg/m^2).

Smoking characteristics (T1). The baseline questionnaire T1 also asked for information on participants' smoking history, quit attempts ("Have you attempted to stop smoking at all in the last 5 years?"), as well as nicotine dependence using the Heaviness of Smoking Index, a short version of the Fagerström test for nicotine dependence (Heatherton, Kozlowski, Frecker, Rickert, & Robinson, 1989).

Cognitive (primary) outcomes (T1, T2). Cognitive outcomes were assessed at the baseline (T1) and outcome (T2) questionnaire. Fear about smoking was assessed using two questions on 7-point scales (Dijkstra & Brosschot, 2003) with content-specific anchors regarding airway disease ("*not at all afraid*" and "*very afraid*"; "*not at all worried*" and "*extremely worried*") and the mean items score was used (Cronbach's α = 0.79–0.80). Perceived severity and susceptibility were measured with two single 7-point response scales each with content-specific anchors regarding airway disease, which have been successfully used in similar form before (Hall, Weinman, & Marteau, 2004). For perceived susceptibility, participants were asked to rate their likelihood as well as risk (in comparison with nonsmokers) of developing airway disease (from "*very unlikely*" to "*very likely*" and "*much higher*" to "*much lower*," respectively; Cronbach's α = 0.61–0.66). For perceived severity, participants were asked whether they believed that airway disease was (a) a serious disease and (b) a severe illness (both from "*strongly agree*" to "*strongly disagree*"; Cronbach's α = 0.82–0.84). Perceived response efficacy and self-efficacy were assessed by two 7-point rating scales each, and the respective mean item score was used. Both measures have been shown to display good reliability (Hall, Bishop, & Marteau, 2003). Response efficacy was determined by asking smokers whether they believed that stopping smoking can reduce (a) their risk and (b) their likelihood of getting airway diseases (both from "*strongly agree*" to "*strongly disagree*"; Cronbach's α = 0.66–0.70). Self-efficacy (the belief that one can do something, e.g., change a given behavior) was assessed by asking participants how confident they are to be able to stop smoking (from "*very confident*" to "*not at all confident*") and how easy it would be for them to stop smoking (from "*very easy*" to "*not at all easy*"; Cronbach's α = 0.70–0.78). In addition, participants were asked about their intention to stop smoking in the next month measured using two 7-point Likert response scales ranging from "*very unlikely*" to "*very likely*," and "*definitely will*" to "*definitely will not*" (Cronbach's α = 0.73–0.86). While intention to stop smoking does not form part of either the EPPM or EP, we have included it here as an immediate measure of potential changes in subsequent behavior. For each of the measures, the mean value was used in analysis.

Behavioral (secondary) outcomes (T3). At 6 months' follow-up, participants were asked to indicate their current smoking status ("Have you smoked in the last seven days?" Yes/No), whether they had attempted to stop smoking, as well as their intention to stop smoking in the next month (see above).

Analysis

This study was powered for the laboratory study, which provided 80% power at a standard Type I error rate ($\alpha = .05$) to detect a medium-to-large effect size (Cohen's d $\sim$ 0.5–0.7) for group differences in primary (cognitive) and secondary (behavioral) outcomes in a two-tailed comparison of means or proportions. This effect size for cognitive outcomes is largely comparable to those found in previous studies with similar measures and intervention design (Hall et al., 2003; Hall et al., 2004; Shahab, Hall, & Marteau, 2007). Change scores were calculated from responses to baseline and outcome questionnaire for cognitive outcomes. Although interactions between group and outcome variables could be assessed with repeated measures analysis of variance (ANOVA), change scores were calculated and group differences assessed as this yields equivalent results and has the advantage that nonparametric tests can be used to determine interactions. Group differences were tested with *t* tests, changes within groups with paired *t* tests and where appropriate Mann–Whitney U and Wilcoxon's tests were used to validate results. Multivariate logistic regression was used to predict behavioral outcomes using treatment allocation, sociodemographic and smoking characteristics and where appropriate cognitive measures as predictors. Where it was impossible to use regression owing to the distribution of data, log-linear models were fitted to be able to estimate moderation effects. Mediation (using the Sobel method) and moderated mediation were analyzed with bootstrapping in SPSS (Preacher, Rucker, & Hayes, 2007). All analyses of behavioral outcomes used an intention-to-treat approach.

Results

The study sample was relatively young with a mean age of 31, and slightly more men than women (Table 1). Participants had been smoking for an average of 14 years and smoked nearly 14 cigarettes per day. The majority had attempted to quit in the last 5 years, but only one-tenth agreed or very strongly agreed with the statement that they were intending to stop smoking in the next month. There were no differences on baseline demographic or smoking characteristics between the intervention and control group.

Primary Outcomes

The level of perceived response efficacy as well as perceived susceptibility increased significantly from Visit 1 to Visit 2 in both the intervention and control group and self-efficacy only in the intervention group (Table 2). While perceived severity was the only cognitive measure that did not increase across visits in either group, possibly because of a ceiling effect as baseline levels were already very high (average of 5.7 and 5.3 on 7-point scale in control and treatment group, respectively), as hypothesized threat appraisal in the form of perceived susceptibility increased significantly more in the intervention than control group, $t(158) = 2.33$, $p = .021$.

Although self-reported fear levels were significantly increased across visits in the intervention group only, $t(80) = 3.2$, $p = .002$, this increase was not significant relative to the control group, $t(158) = 1.4$, $p = .180$. As predicted, participants in the intervention group displayed a greater rise in their reported intention to stop smoking in the next month than those in the control group, $t(151) = 2.9$, $p = .004$. Yet, this increase was short-lived. As shown in Figure 2, when only looking at participants with complete data and excluding those who had stopped at the time of follow-up ($N = 51$ and $N = 48$ for control and intervention group, respectively), intention to stop smoking at 6 months' follow-up had dropped again for both groups and was not significantly different from baseline intention to stop smoking in either the control or treatment group, $t(50) = 0.34$, $p = .731$ and $t(47) = 0.97$, $p = .335$, respectively.

Secondary Outcomes

There were no differences in terms of quit attempts; the same proportion had tried to stop in both the intervention (17.3%, $N = 14$) and control group (15.2%, $N = 12$). Logistic regression was conducted to predict quit attempts and included group allocation, sociodemographic and smoking characteristics. The only predictors to emerge were past quit attempts (odds ratio [OR] = 6.37; 95% confidence interval [CI] = 1.81–22.47) and cotinine level (OR = 0.99; 95% CI = 0.99–1.00) suggesting that those who had attempted to stop in the previous 5 years and those who had lower

Table 2
Change in Cognitive Outcomes Pre- to Postintervention

Self-reported measures	Mean change score (95% CI); *p* (within group)		
	Intervention ($N = 81$)	Control ($N = 79$)	*p* (between group)
Perceived efficacy			
Self-efficacy	0.346 (0.101–0.590); .006	0.260 (−0.011–0.530); .059	.638
Response efficacy	0.253 (0.015–0.491); .037	0.348 (0.049–0.647); .023	.620
Perceived threat			
Susceptibility	0.654 (0.401–0.908); <.001	0.272 (0.069–0.476); .009	.021
Severity	0.099 (−0.312–0.510); .634	0.013 (−0.136–0.162); .866	.699
Fear	0.401 (0.152–0.650); .002	0.184 (−0.020–0.387); .076	.180
Intention to stop	1.108 (0.793–1.420); <.001	0.519 (0.270–0.768); <.001	.004