The effect of stopping smoking on perceived stress levels

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ABSTRACT

Aims Many smokers believe that smoking helps them to cope with stress, and that stopping smoking would deprive them of an effective stress management tool. Changes in stress levels following long-term smoking cessation are not well mapped. This longitudinal project was designed to provide more robust data on post-cessation changes in perceived stress levels by following a cohort of smokers admitted to hospital after myocardial infarction (MI) or for coronary artery bypass (CAB) surgery, as such patients typically achieve higher continuous abstinence rates than other comparable samples.

Design A total of 469 smokers hospitalized after MI or CAB surgery and wanting to stop smoking were seen in the hospital and completed 1-year follow-ups. Ratings of helpfulness of smoking in managing stress at baseline, smoking status (validated by salivary cotinine concentration) and ratings of perceived stress at baseline and at 1-year follow-up were collected.

Findings Of the patients, 41% (n = 194) maintained abstinence for 1 year. Future abstainers and future smokers did not differ in baseline stress levels or in their perception of coping properties of smoking. However, abstainers recorded a significantly larger decrease in perceived stress than continuing smokers, and the result held when possible confounding factors were controlled for (P < 0.001).

Conclusions In highly dependent smokers who report that smoking helps them cope with stress, smoking cessation is associated with lowering of stress. Whatever immediate effects smoking may have on perceived stress, overall it may generate or aggravate negative emotional states. The results provide reassurance to smokers worried that stopping smoking may deprive them of a valuable coping resource.

Keywords Nicotine withdrawal, smoking cessation, stress.

INTRODUCTION

Dependent smokers cite stress relief as one of the major reasons for smoking [1]. At the same time, the incidence of high stress levels and low mood is higher in smokers than in non-smokers and ex-smokers [2–8]. The common explanation for this apparent paradox is that smoking can provide a valuable coping and affect regulation tool which makes it particularly attractive for individuals prone to stress [9]. The high stress levels among smokers are thus due to self-selection. An alternative hypothesis suggests that far from helping with stress, smoking can in fact act as a stressor, e.g. via periods of withdrawal discomfort arising during each inter-cigarette interval, or due to some other neurotoxic effects of tobacco use [10]. According to this view, the perception of a calming effect of smoking is a misattributed withdrawal relief [11–13]. Smoking may be providing stress relief acutely, while generating stress across a longer time-period.

Laboratory studies of acute effects of smoking after short abstinence cannot easily differentiate ‘genuine’ stress and anxiety relief from alleviation of withdrawal discomfort [14]. There exists some support for both genuine anxiolytic effects of smoking as well as for the withdrawal relief hypothesis. For example, some animal studies and human studies on non-smokers suggest that nicotine can alleviate stressful states acutely [15], but smokers most sensitive to sedative effects of smoking report above-average stress just before lighting up a
cigarette rather than below-average stress after smoking [16], which supports the withdrawal relief interpretation of the effect in this population.

Regarding chronic tobacco use and long-term habitual stress levels there is no doubt that smokers have much poorer mental health than non-smokers, but the temporal sequence remains unclear. In most studies which examined the issue smoking precedes mental health problems [5–8,17–19], although some studies found the relationship to go both ways. For example, adolescent smokers are more likely than non-smokers to develop depression later on, but depressed adolescents are also more likely than those who are not depressed to become smokers [20].

It would appear that a simple and elegant way to clarify the effects of smoking on long-term stress levels would be to look at longitudinal changes in stress following smoking cessation in heavy smokers. If smoking aids coping, stress levels should increase following smoking cessation; if it has no real effect on stress the overall levels should remain the same; and if smoking is a stressor, stopping smoking should lead to a decrease in stress levels. Such studies would need to include a follow-up period longer than just a few weeks, as short-term results could be influenced by effects of acute withdrawal discomfort [21] and by the participants’ satisfaction with the success of their quit attempt or disappointment with failure [22].

This interesting issue has not been well researched. In a pioneering longitudinal study by Cohen & Lichtenstein (1990), a sample of unaided quitters was followed-up for 6 months [23]. The perceived stress levels decreased in those who achieved validated continuous abstinence compared to those who failed, but only 5% managed to quit. The study does not provide data on participants’ perception of effects of smoking on stress or on their dependence and thus it is possible, for instance, that successful quitters may have been exceptionally light smokers who did not derive any stress management benefits from smoking while the remaining 95% of the sample did. A similar, more recent report [24], facing identical problems with interpretation, found a significant decrease in stress levels in 88 spontaneous quitters identified in a large community sample.

Several studies examined health-related subjective outcomes of long-term smoking cessation. In another community sample, Sarna et al. (2008) did not find improvements in health-related quality of life [28]. The studies of smokers seeking treatment used more homogeneous samples compared to the community surveys, and so the impact of participant self-selection is likely to have been smaller. However, they did not focus specifically on changes in stress levels and are also open to some of the same caveats as the earlier reports.

Smokers hospitalized after myocardial infarction or coronary artery bypass surgery are a group well suited to study post-cessation changes in stress. They are typically highly dependent smokers who up to now were unable to quit despite suffering from a serious smoking-related condition, and some 40% can be expected to remain abstinent after the event [29]. We followed-up a large sample of such patients to see whether and in what direction smoking cessation affects their stress levels, and whether stress at baseline and the strength of the belief that smoking is a coping resource contribute to any such relationship and to relapse to smoking.

METHODS

Participants

The participants were smokers who were either recovering from a myocardial infarction or were admitted for coronary artery bypass surgery across 17 hospitals in England. None of the participants had smoked since being admitted to hospital (usually within the previous 48 hours). They were all motivated to remain abstinent from smoking permanently and had agreed to take part in a randomized controlled trial of a brief bedside intervention by cardiac rehabilitation nurses to prevent relapse to smoking [30]. A total of 540 patients were recruited to the study, of whom 469 provided baseline and 1-year ratings of stress. The remaining 71 patients had moved to an unknown address, died, discontinued the study, or did not provide stress ratings. The characteristics of the study sample are shown in Table 1. The patients smoked on average 21 cigarettes a day [standard deviation (SD) = 13], and 75% smoked within 30 minutes of waking up.

Procedures and measures

At baseline the participants completed a questionnaire with items concerning demography (age, sex, marital status, employment status, educational qualifications), smoking behaviour (time since last cigarette, number smoked per day, time to first cigarette of the day) and the following ratings: ‘Did smoking help you to cope with stress?’ (1 = very much, 2 = somewhat, 3 = a little, 4 = not really); and ‘leaving aside your heart condition, how much stress is there in your life at the moment?’
Table 1 Baseline characteristics of participants who provided stress ratings at baseline and 1-year follow-up.

<table>
<thead>
<tr>
<th>n (%)</th>
<th>Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>56.0 (9.9)</td>
</tr>
<tr>
<td>Male</td>
<td>363 (77.4)</td>
</tr>
<tr>
<td>Admitted for:</td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>436 (93.0)</td>
</tr>
<tr>
<td>Coronary artery bypass surgery</td>
<td>33 (7.0)</td>
</tr>
<tr>
<td>Cigarette consumption (cigarettes/day)</td>
<td>20.7 (12.6)</td>
</tr>
<tr>
<td>Smoke within the first 30 minutes of waking</td>
<td>351 (75.3)</td>
</tr>
<tr>
<td>'Did smoking help you to cope with stress?'</td>
<td></td>
</tr>
<tr>
<td>Not really</td>
<td>74 (15.8)</td>
</tr>
<tr>
<td>A little</td>
<td>59 (12.6)</td>
</tr>
<tr>
<td>Somewhat</td>
<td>104 (22.2)</td>
</tr>
<tr>
<td>Very much</td>
<td>231 (49.4)</td>
</tr>
<tr>
<td>Time since last cigarette in days</td>
<td>7.2 (11.8)</td>
</tr>
<tr>
<td>Baseline stress ratings (1 = none at all; 10 = extreme amount)</td>
<td>5.3 (2.8)</td>
</tr>
<tr>
<td>One-year follow-up stress ratings</td>
<td>4.9 (2.6)</td>
</tr>
<tr>
<td>Continuously abstinent at 1 year</td>
<td>194 (41.4)</td>
</tr>
</tbody>
</table>

*Sample size varies due to missing data. SD: standard deviation.

(measured on a scale from 1 to 10 where 1 = no stress at all and 10 = extreme amount of stress).

At 1 year after the recruitment, smoking status was established and the ratings were repeated. Continuous abstinence at 1 year was defined as a self-report of having not smoked a single puff in the past week, having a salivary cotinine level of less than 20 ng/ml, an expired-air carbon monoxide (CO) reading of less than 10 parts per million (p.p.m.) and having smoked no more than five cigarettes (or rollups, cigars or pipes) since recruitment. Where the cotinine reading was absent (e.g. because the saliva sample was insufficient, or because the patient was using nicotine replacement treatment) CO reading alone was used to validate the self-report. The study was approved by the appropriate local Research Ethics Committees.

Statistical analysis

Simple descriptive statistics were used to analyse the sample characteristics. Analyses of variance were used to examine the relationship between smoking status at follow-up and baseline stress levels, perception of effects of smoking on stress and stress levels at follow-up. Baseline stress ratings were included as a covariate in the latter. Finally, we examined the predictors of stress at follow-up using regression analysis. Independent variables included smoking status at follow-up and the following baseline variables: stress, age, sex, marital status, employment status, educational qualifications, number smoked per day, time to first cigarette of the day, diagnosis and time of stopping smoking relative to hospital admission. The brief bedside intervention had no effect on abstinence rates [20]. It had no effect on mean stress ratings either [4.9, SD = 2.5 versus 4.9, SD = 2.8] in the intervention and control groups, respectively. We therefore analysed the data from both groups together. Analyses were undertaken using SPSS version 16.0 and all statistical tests were two-tailed.

RESULTS

Saliva samples were insufficient or contaminated in four cases. In all four, CO reading indicated non-smoker status and the patients were classified as non-smokers. Two further participants were taking nicotine replacement therapy at the time their saliva sample was collected and their cotinine reading was discounted. Carbon monoxide readings allowed both patients to be classified as non-smokers.

Of 469 participants who provided complete stress ratings at both time-points, 194 (41%) were abstinent continuously since their hospital admission 1 year earlier (see Table 1).

There was no difference between future abstainers and continuing smokers in mean baseline stress levels (5.3, SD = 2.8; 95% CI: 4.9–5.7 and 5.4, SD = 2.8; 95% CI: 5.1–5.7, respectively; F = 0.5, P = 0.5). There was also no difference in the perception that smoking helps to cope with stress (2.0, SD = 1.2; 95% CI: 1.9–2.2 and 1.9, SD = 1.1; 95% CI: 1.8–2.1, respectively; F = 0.6, P = 0.4).

At 1-year follow-up stress scores were significantly lower in those who remained abstinent (4.4; 95% CI: 4.1–4.8) than for those who relapsed to smoking (5.2; 95% CI: 4.9–5.6; F = 11.4, P = 0.001) (see Fig. 1). This difference remained significant when baseline stress was included as a covariate (F = 9.1; P = 0.003).

With all baseline variables entered into the model, only the baseline stress level, smoking status at follow-up and age were associated significantly with stress level at 1 year (see Table 2).

As patients hospitalized after myocardial infarction (MI) may differ from those admitted for bypass surgery we repeated the analyses with MI patients only. This made no difference to the results [stress ratings at baseline were 5.26 and 5.46 in future quitters and future smokers, respectively; NS; stress ratings at 1 year were 4.42 and 5.20 for the two groups, F = 9.68, P = 0.002; and smoking status at follow-up remained a significant predictor of stress level at 1 year in multiple regression analysis, b = –0.72, standard error (SE) b = 0.26, Beta = –0.14; P < 0.01].
DISCUSSION

Long-term abstainers experienced a reduction in their stress levels compared to baseline while stress levels in continuing smokers did not change. The difference between the two groups was significant and remained so even after potential confounding variables had been controlled for.

The stress reduction effect of smoking cessation in this sample cannot be explained by quitters being less stressed or less sensitive to presumed coping effects of smoking than those who did not quit. The two groups did not differ in their baseline stress levels, or in their perception of coping properties of smoking. In this sample of people who continued to smoke despite having a serious smoking-related illness, most participants reported that smoking helps them cope with stress (close to half of the sample gave the maximum rating). It could be argued that the difference in stress change between the two groups was caused by continuing smokers being stressed by their inability to stop smoking, or by the increasing social disapproval of smoking. They may also have experienced higher levels of stress for other reasons, which may have precipitated their relapse to smoking. Although this cannot be ruled out altogether it seems unlikely, because the stress levels in this group did not increase.

The difference was due to a decrease in stress in subjects who stopped smoking.

The study had several limitations. It included predominantly highly dependent smokers self-selected to continue to smoke despite suffering from a smoking-related illness, and the results may not generalize to less dependent, younger and healthier smokers. Conversely, it is primarily the highly dependent smokers who are supposed to derive coping benefits from their smoking. As not all participants quit smoking, it remains possible that for some of those who did not quit, stopping smoking would generate stress long-term. This was a much stronger possibility in studies where only small fractions of heterogeneous samples succeeded in quitting. The fact that future quitters considered smoking to be as valuable for coping with stress as future smokers is also reassuring. However, only an experimental design can eliminate any self-selection bias altogether. Perceived stress levels were assessed by a single-item measure. It should be noted that any real or assumedcrudeness of the measure we used would be expected to mask rather than amplify any background relationships. However, the assumption that longer questionnaires automatically ensure increased accuracy is questionable. In a previous study looking at measuring post-cessation changes in anxiety, a similar single-item rating was shown to be at least as sensitive as an extensive multi-item measure (State–Trait Anxiety Inventory-State Form X) [31]. Future studies, however, may consider more inclusive measures of stress which could also provide information on specific types of stress. The absence of normative data for the stress rating instrument we used makes it difficult to assess the clinical significance of the 20% decrease in stress levels, but this does not affect the key result that after smoking cessation, stress levels went down rather than up. We did not collect data on life events, health status, social support, etc. during the follow-up period, and a possibility needs

![Figure 1](https://example.com/figure1.png)  
**Figure 1** Perceived stress at baseline and at 1 year in abstainers (n = 194) and continuing smokers (n = 275). The error bars represent 95% confidence intervals around the mean.

### Table 2 Predictors of stress at 1 year.

<table>
<thead>
<tr>
<th>Predictor</th>
<th>b</th>
<th>SE</th>
<th>Beta</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Constant)</td>
<td>6.21</td>
<td>1.13</td>
<td></td>
</tr>
<tr>
<td>Continuous abstinence at 1 year</td>
<td>-0.78</td>
<td>0.25</td>
<td>-0.146**</td>
</tr>
<tr>
<td>Stress at baseline</td>
<td>0.21</td>
<td>0.04</td>
<td>0.22***</td>
</tr>
<tr>
<td>Age</td>
<td>-0.03</td>
<td>0.02</td>
<td>-0.12*</td>
</tr>
</tbody>
</table>

b: unstandardized regression coefficients; SE b: standard errors of b; Beta: standardized regression coefficients. $R^2$: 0.13 ($F = 4.91$, $P < 0.001$).

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$. 

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to be considered that the life trajectories of the two groups of patients differed in some relevant aspect, e.g. successful quitters may have received treatments or experience life events which contributed to their abstinence and also decreased their stress levels. The one-off follow-up did not allow us to determine at what timepoint the effect of smoking cessation on stress levels appears. The patients in this sample were acutely ill at baseline and this may have led to higher initial stress ratings despite the way the question was phrased, but this would be unlikely to affect the difference between the two groups.

Despite these limitations, the results add weight to the hypothesis that smoking does not help with general stress levels. They are also compatible with the notion that despite the perception that smoking alleviates stress acutely, it may in fact worsen negative emotional states overall. The data complement the findings that, compared to non-smokers, smokers are more likely to suffer from high stress levels with indications of a dose–response relationship; and that smoking seems to precede more often than follow the onset of mental health problems [3]. A causal link has been suggested to explain these findings proposing that smoking may, at least in vulnerable individuals, contribute to psychiatric morbidity [6,7,17–19]. The study results may allow other interpretations, and the notion that smoking may harm not just physical but also mental health is controversial at present, but the hypothesis deserves further empirical scrutiny

Apart from contributing to one of the key theoretical discussions on subjective effects of smoking, the present study has also practical and clinical implications. Dependent smokers often express high regard for stress management properties of smoking, and are afraid that stopping smoking will leave them more vulnerable to stress. The findings of this study can be used to provide reassurance to smokers who are worried that stopping smoking would result in the loss of a valuable coping tool.

Declarations of interest

Drs Hajek and McRobbie have undertaken research and consultancy for, and received honoraria for speaking at meetings for, the manufacturers of smoking cessation medications. Tamara Taylor has no competing interests to declare.

Acknowledgements

P.H. and T.T. had the original idea for the study and were responsible for design, collecting data, analysis and interpretation of data and writing the paper. H.M. contributed towards analysis and interpretation of data, and writing the paper. P.H. is guarantor for the study. This study was supported by a grant from the NHS R&D Programme on Cardiovascular Disease and Stroke.

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