Impact of biomarker feedback on smoking

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The motivational impact of showing smokers with vascular disease images of their arteries: a pilot study.

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Abstract

Objective: To examine the potential impact of visual personalised biomarker feedback on intention to stop smoking and to evaluate possible underlying causal pathways.

Design: This study is a pilot for a randomised controlled trial. Outcome measures were assessed immediately after the intervention and at four weeks follow-up.

Method: Twenty-three smokers attending a cardiovascular outpatient clinic in London were randomly allocated to one of two groups: to either receive a print-out of an ultrasound image of their carotid artery showing atherosclerotic plaque alongside an image of a disease-free artery, or to receive routine verbal feedback.

Results: The intervention significantly increased perceptions of susceptibility to smoking-related diseases ($\chi^2=5.24, p<.05$) and led to an increase in intentions to stop smoking. The latter was moderated by self-efficacy: the intervention increased intention to stop smoking only in people with higher levels of self-efficacy in stopping smoking ($t(10)=2.33, p<.05$).

Conclusions: This study provides preliminary support for the potential effectiveness of personalised biomarker feedback to increase intentions to stop smoking. It also highlights the need to target and increase self-efficacy in smoking cessation interventions.

Key words: harm biomarker feedback, smoking cessation intervention, atherosclerotic plaque, cardiovascular patients, extended parallel processing model, self-efficacy
1. Introduction

Smoking remains one of the most enduring public health problems of modern society, killing over 4.9 million people per year worldwide (World Health Organization (WHO), 2002). Stopping smoking can prevent the development of tobacco-related diseases within a few years of cessation (Peto et al., 2000; Rich-Edwards, Manson, Hennekens, & Buring, 1995). The fact that 70-80% of smokers would like to quit, but only half of smokers ever achieve abstinence (World Health Organization (WHO), 1998), underlines the need for the development of more effective interventions, which both motivate smokers to quit and help sustain long-term cessation.

One promising approach for smoking cessation interventions is the use of personalised information of smoking-induced health damage provided by biomarker feedback (Lerman, Orleans, & Engstrom, 1993). Its inclusion in cessation programmes has been proposed on the basis of theoretical considerations: to simply tell people they are at risk of developing a disease is rarely sufficient to change behaviour (Leventhal et al., 1997). Personalised information can counteract perceptions of invulnerability to the health consequences of tobacco-use, which are common among smokers (Strecher, Kreuter, & Kobrin, 1995), thus raising threat perceptions and fear, which motivate behaviour change to reduce this threat (Cameron, 2003).

According to the Extended Parallel Process Model (EPPM, Witte, 1998) people engage in protective behaviours when they perceive themselves to be at risk of a threat (threat appraisal) and feel that they can reduce this threat (efficacy appraisal). People first appraise the threat by evaluating the severity of the threat (e.g. trivial or serious) and their susceptibility to it. This is followed by an efficacy appraisal. This involves an
assessment of their ability to perform a behaviour (self-efficacy) that is effective in averting this threat (response efficacy).

When both threat and efficacy appraisals are high, danger control processes ensue, which lead to the acceptance of a threat message such as “stop smoking”. In this context, fear (resulting from high threat appraisal) may guide people towards a behavioural solution, i.e. cessation. However, when threat appraisal is high and efficacy appraisal is low, fear could lead people towards a cognitive solution (i.e. avoidance) and fear control processes take over, which can result in the rejection of the threat message (Witte & Allen, 2000).

Biomarker feedback may increase smokers’ threat perceptions, and thus instigate smoking cessation, for a variety of reasons. Visual personalised biomarker feedback showing harm is postulated to maximally impact on threat perceptions as imagery allows for the spanning of the conscious-unconscious continuum more readily than language (Horowitz, 1970) and is therefore less likely to be filtered through the conscious critical apparatus. Consequently, visual personalised biomarker feedback would increase the likelihood of danger control processes occurring since it avoids derogation of the threat message by “disengagement beliefs”, which distort the threatening meaning of potentially motivating information (Bandura, Barbaranelli, Caprara, & Pastorelli, 1996). Imagery with reference to the self also leads to greater problem elaboration and more stable changes in attitude (Burnkrant & Unnava, 1995).

However, as visual personalised biomarker feedback showing harm does not increase perceptions of self-efficacy, its impact on cognitive and behavioural responses may be particularly pronounced in smokers with higher levels of self-efficacy. For instance, in the
absence of sufficient self-efficacy, a threat message does not increase motivation to stop smoking (Bishop, Marteau, Hall, Kitchener, & Hajek, 2005). In contrast, perceptions of response efficacy may be increased either by the provision of information (e.g. outlining a behaviour-disease link) or by a biomarker evidencing the positive impact of a behaviour (e.g. smoking cessation) on an objective outcome (e.g. lung function improvement).

An earlier unreplicated study, which was not theoretically based, demonstrated that providing healthy smokers with ultrasound photographs of their own atherosclerotic plaque increased quit rates (Bovet, Perret, Cornuz, Quilindo, & Paccaud, 2002). The current study was set in a cardiovascular disease (CVD) outpatient clinic. Non-hospitalized smokers would particularly benefit from smoking cessation since the development of smoking-induced diseases like atherosclerosis can be halted, if not reversed, after smoking cessation (Wiggers, Smets, de Haes, Peters, & Legemate, 2003). The objective of this study was to assess the motivational impact of personalised biomarker feedback and to explore possible causal pathways as postulated by the EPPM, as well as the acceptability and feasibility of such an approach in a clinical setting. Our hypotheses were as follows:

a.) Showing CVD outpatients who smoke images of their damaged arteries increases threat perceptions, i.e. perceptions of susceptibility and severity.

b.) Showing CVD outpatients who smoke images of their damaged arteries and providing them with information about the link between smoking and CVD increases response efficacy but not self-efficacy perceptions.

1 In atherosclerosis smooth muscle cells proliferate and fatty substances, in particular cholesterol and triglycerides, accumulate in the walls of arteries to create plaque (Tortora & Grabowski, 2002)
c.) Showing CVD outpatients who smoke images of their damaged arteries increases intention to stop smoking, particularly in smokers with higher levels of self-efficacy.
2. Methods

Participants

This study was approved by Guy’s Hospital Research Ethics Committee (Ref 2004/02/02). Between April and July 2004 cardiovascular outpatients at a London hospital were sent written information about this study one week prior to their clinic appointments. On the day of their appointments, patients who were smokers and literate in English were invited to participate in the study. Demographic and baseline characteristics of the sample are shown in Table 1. These indicate that the majority of patients were likely to be older and had no formal education. They also displayed high nicotine dependence as more than half of patients reported smoking within thirty minutes of waking and consuming more than eleven cigarettes per day.

Procedure

By means of a computer generated random numbers table consenting patients were allocated to either the intervention or the control group (routine care) and asked to fill in a baseline questionnaire. Both groups were then seen by a trained clinician who imaged their carotid arteries. This is a routine clinical procedure, which lasts about five minutes and uses a high-frequency ultrasound transducer (Philips HDI 5000, Letchworth, UK). Following the scan, patients in both groups were seen by a cardiovascular consultant who provided them with verbal feedback of the scan result. In addition to this routine care, the consultant showed people in the intervention group their scans and gave them photographs contrasting a healthy artery with their own arteries, together with a leaflet describing the link between CVD and smoking as well as general health benefits of smoking cessation. After their appointments, all participants were given a follow-up questionnaire to complete. Patients in the intervention group were also briefly interviewed about their feelings regarding the scan, and asked to provide any thoughts.
about or problems with this procedure. Four weeks after the clinic date, patients were telephoned to obtain follow-up information.

**Measures**

The baseline questionnaire assessed demographic details (age, gender, educational attainment), nicotine dependence (Fagerstrom, 1978) and readiness to stop smoking (Prochaska & DiClemente, 1983). The first follow-up questionnaire assessed intention to stop smoking in the next month, perceived susceptibility to and perceived severity of smoking-related illnesses, perceived response efficacy of stopping smoking and perceived self-efficacy to do so. These were each measured with two 7-point scales, e.g. ‘Do you intend to stop smoking in the next month?’ from ‘definitely do not’ to ‘definitely do’ and ‘How likely is it that you will stop smoking in the next month?’ from ‘very unlikely’ to ‘very likely’. The combined scales were reliable (Cronbach's alphas 0.71-0.87) and their means were used in the analysis (for more details of measures see Hall, Weinman, & Marteau, 2004).

At the four week telephone follow-up, various smoking cessation behaviours (use of cessation services and products, quit attempts, talking to a GP or a nurse at a primary care centre about quitting, calling a stop-smoking helpline) were assessed. Engagement in these behaviours predicts the transition from smoking to non-smoking (France, Glasgow, & Marcus, 2001).

**Analyses**

Parametric assumptions were tested and group comparisons were conducted using either t-tests or, if results indicated a non-parametric distribution, $\chi^2$ and Mann-Whitney U tests. Interaction effects were explored with univariate ANOVA. As the current study
was powered only to detect large effects, Cohen’s $d$ and $h$ (Cohen, 1988) were also
calculated to estimate effect sizes. Qualitative data on open-ended questions were
analysed using content analysis (Boyatzis, 1998). As is recommended procedure (Willig,
2001), reliability and validity of reported findings were assessed by an independent audit
involving an outside researcher.
3. Results

The two groups did not differ on any demographic variables or baseline readiness to stop smoking (Table 1). All but two patients in the treatment group had abnormal arteries and there were no differences in scan results between the two groups.

Table 1 about here

a.) Perceptions of the severity of CVD were similarly high in both groups (Table 2). Since susceptibility scores showed a markedly different shape of distribution between groups, this measure was dichotomised. Compared to the control group, the intervention group reported higher perceptions of susceptibility to smoking-related diseases (Cohen's $h=0.99$).

Table 2 about here

b.) Perceptions of response efficacy did not differ significantly between groups. As hypothesised, there were no significant differences between groups in perceived self-efficacy.

c.) Immediately after the scan, the two groups did not differ significantly in reported intention to stop smoking. However, the mean difference was in the expected direction evidencing a medium-sized effect. At follow-up, those in the intervention group were also more likely to report engaging in smoking cessation behaviours (Cohen's $h=0.79$, Table 2).
As this study was powered to detect only large effect sizes, and because of the observed ceiling effect in some threat and efficacy measures, interactions between these measures and group allocation on intention to stop smoking could not be investigated. The analysis was therefore limited to self-efficacy. As postulated, self-efficacy interacted with group on intention to stop smoking ($F(1,19)=5.73$, $p=.03$; Figure 1). Compared to the control group, the intervention increased intentions to stop smoking only in patients with high levels of self-efficacy ($t(10)=2.33$, $p<.05$) and not in those with low levels of self-efficacy ($t(9)=-1.031$, n.s.).

Figure 1 about here

In order to assess the acceptability of the intervention, patients in the intervention group were briefly interviewed after their scan to ascertain their views about the procedure. They unanimously reported that the scan was unproblematic and did not make them feel uncomfortable or scared. At follow-up, only patients in the intervention group mentioned that their clinic visit had made them think more seriously about giving up smoking.
4. Discussion

Despite the relatively small sample size and short follow-up, the results of this pilot study are encouraging. As hypothesised, patients who received personalised biomarker feedback visualising harm reported higher susceptibility to heart disease, indicating that this feedback increased their awareness of this smoking-related risk. Confirming previous research involving biomarker feedback (McClure, 2001), there was some evidence that the intervention altered both motivational and behavioural outcomes. Patients in the intervention group had a higher mean intention to stop smoking and reported engaging in more cessation behaviours including attempting to quit and contacting quit-smoking services. Although these group differences were not statistically significant, the effect sizes are suggestive of the potential effectiveness of the intervention.

Contrary to expectation, the study groups did not differ in their perceptions of response efficacy despite the provision of information about the effectiveness of smoking cessation in the intervention group. This probably reflects characteristics of the study population. Patients suffering from CVD have direct experience of the consequences of the disease and, through previous consultations, are also likely to have been exposed to relevant information regarding the benefits of cessation resulting in both high levels of perceived severity and response efficacy.

In contrast to response efficacy, the intervention did not attempt to alter self-efficacy perceptions and group differences on this measure were therefore not anticipated. However, among smokers with higher self-efficacy to stop smoking the intervention had a significant impact on intention to quit. This moderation is consistent with previous
research (Bishop et al., 2005) and in agreement with the predictions of EPPM. Showing patients with CVD images of their own arteries increased threat perceptions but only smokers with high self-efficacy subsequently engaged in danger control processes and accepted the health warning as shown by an increase in intention to stop smoking. In contrast, patients with lower self-efficacy engaged in fear control in response to the intervention and rejected the message thus displaying somewhat lower intentions to stop smoking. In terms of research practice, this finding highlights the need for smoking interventions to include procedures that increase smokers’ self-efficacy levels in order to obtain optimal results.

This intervention was easy to implement without interrupting the clinic timetable. As most cardiovascular disease clinics use ultrasound transducers, the intervention is likely to be cost- and time-effective, requiring approximately five minutes of clinicians’ time. Interviews with patients showed the intervention to be acceptable to them, and at follow-up, some commented that they felt it had helped them become more determined to deal with their smoking habit.

This pilot study provides preliminary evidence of the feasibility, acceptability and potential effectiveness of a novel smoking intervention to increase intentions to stop smoking. Providing patients with an image of their own arteries could create an opportune moment during which smokers are more amenable to quit-advice or referrals to specialist smoking clinics. This minimal intervention could supply the crucial initial motivation to stop smoking for patients who are already suffering from a smoking-related condition. However, this effect may be restricted to smokers with a higher level of self-efficacy and a clinical trial is now needed to assess its effectiveness on sustained smoking cessation.
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Reference List


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### Table 1 Demographic and baseline characteristics

<table>
<thead>
<tr>
<th>Categorical Variables</th>
<th>Total Sample (N=23)</th>
<th>Intervention Group (N=11)</th>
<th>Control Group (N=12)</th>
<th>Test of Differences</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Proportions (N)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>Female 47.8 (11)</td>
<td>36.4 (4)</td>
<td>58.3 (7)</td>
<td>(\chi^2=1.11,) n.s.</td>
</tr>
<tr>
<td></td>
<td>Male 52.2 (12)</td>
<td>63.6 (7)</td>
<td>41.7 (5)</td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td>No Qualification 73.9 (17)</td>
<td>81.8 (9)</td>
<td>66.7 (8)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>GCSE 4.3 (1)</td>
<td>9.1 (1)</td>
<td>-</td>
<td>(U = 53.0,) n.s.</td>
</tr>
<tr>
<td></td>
<td>A Level 13.0 (3)</td>
<td>9.1 (1)</td>
<td>16.7 (2)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Higher Education 8.7 (2)</td>
<td>-</td>
<td>16.7 (2)</td>
<td></td>
</tr>
<tr>
<td>Daily cigarette</td>
<td>10 or less 39.1 (9)</td>
<td>36.4 (4)</td>
<td>41.7 (5)</td>
<td>(U = 62.5,) n.s.</td>
</tr>
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<td>consumption (in</td>
<td>11-20 52.2 (12)</td>
<td>54.5 (6)</td>
<td>50.0 (6)</td>
<td></td>
</tr>
<tr>
<td>cigarettes)</td>
<td>21 or more 8.7 (2)</td>
<td>9.1 (1)</td>
<td>8.3 (1)</td>
<td></td>
</tr>
<tr>
<td>Daily smoking start</td>
<td>After 60 13.0 (3)</td>
<td>18.2 (2)</td>
<td>8.3 (1)</td>
<td>(U = 59.5,) n.s.</td>
</tr>
<tr>
<td>(in minutes after</td>
<td>Within 31-60 34.8 (8)</td>
<td>45.5 (5)</td>
<td>25.0 (3)</td>
<td></td>
</tr>
<tr>
<td>waking)</td>
<td>Within 6-30 30.4 (7)</td>
<td>-</td>
<td>58.3 (7)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Within 5 21.7 (5)</td>
<td>36.4 (4)</td>
<td>8.3 (1)</td>
<td></td>
</tr>
<tr>
<td>Readiness to stop</td>
<td>Next month 13.0 (3)</td>
<td>18.2 (2)</td>
<td>8.3 (1)</td>
<td>(U = 56.5,) n.s.</td>
</tr>
<tr>
<td>smoking in:</td>
<td>Next 6 months 65.2 (15)</td>
<td>63.6 (7)</td>
<td>66.7 (8)</td>
<td></td>
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<tr>
<td></td>
<td>Next 5 years 8.7 (2)</td>
<td>9.1 (1)</td>
<td>8.3 (1)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Not next 5 years 13.0 (3)</td>
<td>9.1 (1)</td>
<td>16.7 (2)</td>
<td></td>
</tr>
<tr>
<td>Continuous Variable</td>
<td>Mean (SD)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (in years)</td>
<td>62.8 (11.6)</td>
<td>61.91 (9.2)</td>
<td>63.67 (13.8)</td>
<td>(t(21)=-0.36,) n.s.</td>
</tr>
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</table>
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Table 2 Outcome variables by group

<table>
<thead>
<tr>
<th></th>
<th>Intervention Group (N=11)</th>
<th>Control Group (N=12)</th>
<th>Effect size (Cohen’s $d$)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Continuous Variables</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Intention to stop smoking in the next month</td>
<td>4.0 (1.8)</td>
<td>3.3 (1.5)</td>
<td>0.44, t(21)=1.04, ns</td>
</tr>
<tr>
<td>Perceived self-efficacy</td>
<td>3.1 (1.6)</td>
<td>3.0 (2.0)</td>
<td>0.06, t(21)=0.18, ns</td>
</tr>
<tr>
<td>Perceived response efficacy</td>
<td>5.1 (1.3)</td>
<td>5.0 (1.9)</td>
<td>0.06, t(21)=0.26, ns</td>
</tr>
<tr>
<td>Perceived severity</td>
<td>5.8 (1.8)</td>
<td>6.0 (0.9)</td>
<td>0.15, t(21)=-0.39, ns</td>
</tr>
<tr>
<td><strong>Categorical Variables</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perceived Susceptibility</td>
<td>Low 27.3 (3)</td>
<td>75 (9)</td>
<td>0.99, $\chi^2=5.24$, $p=.02$</td>
</tr>
<tr>
<td></td>
<td>High 72.7 (8)</td>
<td>25 (3)</td>
<td></td>
</tr>
<tr>
<td>Smoking Cessation Behaviours at 4 week follow-up</td>
<td>None 9.1 (1)</td>
<td>41.7 (5)</td>
<td>0.79, $\chi^2=3.16$, $p=.08$</td>
</tr>
<tr>
<td></td>
<td>At least one 90.9 (10)</td>
<td>58.3 (7)</td>
<td></td>
</tr>
</tbody>
</table>

With the exception of smoking cessation behaviours at 4 weeks, all outcomes were assessed immediately after the intervention.
Figure Legend

Figure 1: Impact of intervention on intention to stop smoking for smokers with higher and lower levels of self-efficacy; ^Median split scale (lower self-efficacy: 1-2.5, higher self-efficacy: 3-7); *p<.05
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Figure 1

![Graph showing the impact of biomarker feedback on smoking intention.

- Intention to stop smoking
- Lower Self-Efficacy
- Higher Self-Efficacy
- Treatment
- Control

Bars indicate the intention to stop smoking with standard error bars. The graph shows a significant difference in higher self-efficacy with the treatment group compared to the control group.

N=5, N=6