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Effects of a brief cognitive intervention aimed at communicating the negative reinforcement explanation for smoking on relevant cognitions and urges to smoke

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Keywords
aimed, effects, communicating, brief, negative, reinforcement, explanation, smoking, relevant, cognitions, urges, smoke, cognitive, intervention

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Effects of a Brief Cognitive Intervention Aimed at Communicating the Negative Reinforcement Explanation for Smoking on Relevant Cognitions and Urges to Smoke

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Introduction: The aim of the current study was to assess the impact of an intervention aimed at communicating the negative reinforcement explanation for smoking, a set of ideas derived from popular self-help books, upon participants’ cognitions and urges to smoke. Methods: Smokers (n = 205) undergoing standard stop-smoking treatment were randomised to receive either the experimental intervention, a brief intervention aimed at communicating the explanation or a control intervention, a video on the health risks of smoking. Outcomes were participants’ acceptance of the negative reinforcement explanation for smoking, positive outcome expectations for smoking, self-efficacy and urges to smoke reported at one week post-cessation. Results: Post-cessation urges to smoke were similar in the two groups (Adjusted expt. group mean = 2.50, Control group mean = 2.75, F(1,60) = 0.98, p = .33). Other cognitive measures were also unchanged. Conclusions: The brief cognitive intervention offered as an adjunct to standard care failed to reduce urges to smoke or alter smokers’ cognitions. Changing smokers’ cognitions may be as challenging as changing their behaviours. Suggestions are provided for further research.

Keywords: negative reinforcement, cognitive intervention, smoking cessation

Despite several effective treatments being available to smokers, successful cessation of smoking remains beyond the reach of many. In order to maximise abstinence rates from smoking cessation interventions, a combination of both pharmacological and psychological treatments appears to be the most effective strategy (US Department of Health and Human Services, 2008; West, McNeill, & Raw, 2000). Yet even with combined interventions, cessation rates remain modest. NHS Stop Smoking Services (SSSs) in England have 12-month quit rates of approximately 15% (Ferguson, Bauld, Chesterman, & Judge, 2005) and the proportion of adults smoking in the population has remained unchanged at 22% in recent years following a period of gradual decline since the 1970s (UK National Statistics, 2009). Treatment programs are therefore likely to benefit from new developments in either of the two approaches.

Withdrawal-oriented therapy is the current model for group behavioural support offered at NHS SSSs. The therapy is based on the notion that smokers seeking treatment are addicted to nicotine, and their addiction to nicotine and accompanying withdrawal symptoms are leading causes of relapse. In this context, pharmacological treatment is used to ease withdrawal discomfort and
abstinence-induced urges, and group processes are used to assist people in remaining abstinent throughout the difficult initial period (Hajek, 1989). There is, however, no specific attention paid to smokers’ smoking-related cognitions as part of the treatment. Therefore, once treatment has ended and smokers return to their normal lives, their ability to maintain abstinence may be jeopardised should their underlying motivations to smoke remain unchanged.

In 1985, independently of any empirical or theoretical developments, a book for smokers, *The Easy Way to Stop Smoking*, by Allen Carr was published and became a bestseller in the United Kingdom and subsequently in several other countries (Carr, 1985). The book represents a cognitive approach to smoking cessation based on communicating a set of ideas aimed at changing the way smokers perceive the benefits of smoking and the post-quit withdrawal discomfort. The approach focuses on helping smokers to perceive smoking as a vicious cycle of relieving discomfort induced by nicotine dependence, rather than as a positive and pleasurable activity.

The hypothesis that smoking behaviour is driven primarily by negative reinforcement has been around since the 1970s (Schachter, 1978; Parrott, 1999), but until Carr’s book there were no systematic attempts to incorporate this notion into treatment. Carr managed to communicate its key propositions clearly and forcefully, using easily understandable and memorable metaphors. A number of smokers, including several celebrities, claimed that the book had a profound impact on their smoking. The book has its downside in that it discourages the readers from using effective pharmacotherapies (Hajek, 1988) and it is unlikely that such a cognitive intervention would be sufficient to counter the strong motivational effects of acute nicotine withdrawal in the absence of accompanying pharmacological treatment. Two more recent popular books tried to avoid this limitation and make the Carr ideas compatible with pharmacotherapies, but they lacked Carr’s gift for communication, and their sales were limited (Casey, 2002; Ivings, 2006).

The efficacy of the Carr method has not been evaluated in randomised trials so far. Two cohort studies from Austria reported high, long-term abstinence rates (Moshammer & Neuberger, 2007; Hutter, Moshammer, & Neuberger, 2006); while a UK study detected only modest effects (Foulds, 1996). The existing literature on the method has been summarised in 2006, with the conclusion that the method is awaiting evaluation (McRobbie, Hajek, Bullen, & Feigin, 2006).

The proposition that changing the way a smoker views the rewards of smoking and the reasons that they smoke can impact upon their motivation to smoke, and behaviour, has a strong intuitive validity. Leading smokers to view smoking as an artificial need, which does not really provide any objective benefits and which can be overcome by simply waiting for the acute effects of nicotine deprivation to subside can be expected to result, for example, in reduced positive outcome expectations of smoking (Bandura, 1977) and increased self-efficacy (Rhodes & Blanchard, 2007; Maddux, 1999; Corcoran, 1995). There is some evidence that cognitive interventions can change smokers’ cognitions (Chen & Yeh, 2006; Johnson, Budz, Mackay, & Miller, 1999; Dijkstra, DeVries, & Roijackers, 1998) and that this may produce behaviour change (Dijkstra & DeVries, 2001).

Up to now though, no attempt has been made to test the key Carr hypothesis that smokers who accept the negative reinforcement explanation for smoking, that is, that smoking is driven by relieving withdrawal discomfort, which creates an illusion of positive effects; and that their withdrawal discomfort is self-limiting and can be seen as a signal of recovery rather than as a sign of self-deprivation, are better able to cope with withdrawal discomfort.

Prior to planning a larger study, with abstinence from smoking as an end-point, a ‘proof of principle’ pilot study was conducted to test the impact of a brief intervention designed to communicate the negative reinforcement explanation for smoking upon participants’ cognitions and post-cessation urges to smoke.

**Methods**

**Participants**

Participants were recruited from smokers attending for treatment at the NHS SSS at The Royal London Hospital in East London. All clinic attendees aged 16 or over who were able to fill in the study forms in English were eligible for the trial.

**Design**

The study was designed as a two-group, cluster-randomised controlled trial in which groups of smokers attending the specialist smoking cessation clinic received either an additional brief intervention aimed at communicating the negative reinforcement explanation for smoking, or an additional control intervention matched on contact time with patients. The study was approved by the Hounslow & Hillingdon Research Ethics Committee (Ref: 08/H0709/31).

**Randomisation**

As smokers were treated in groups, and mixing the two interventions in the same group could have led to contamination, groups of smokers were allocated to a treatment arm rather than individual smokers. Smoking cessation groups to be run over the 8-month recruitment period were assigned consecutive numbers by the clinic administrator, and each number was randomised to the intervention or control condition by the lead researcher using a random numbers table. Smokers either self-referred or were referred by a medical practi-
Kozlowski, Frecker, Rickert, & Robinson, 1991). The Mood & Physical Symptoms Scale (MPSS, West, & Hajek, 2004) was administered to obtain baseline ratings of mood and other items known to be affected by cigarette withdrawal. The cognitive measures were repeated at T2, and MPSS was repeated at both T2 and T3.

The strength and intensity of participants' urges to smoke were assessed 1 week following the target quit date (T3). As smoking during the first week of abstinence could also have affected post-cessation urges to smoke, only data from those who remained abstinent throughout the week were used. Participants were considered to be abstinent if they reported not smoking a single puff during the previous week and had a CO reading of ≤9 parts-per-million. For the evaluation of abstinence rates, participants lost to follow-up were considered to be continuing smokers.

**Data Analyses**

The two trial arms were first compared in terms of baseline characteristics using t tests or where the data indicated a non-parametric distribution, chi-square or Mann-Whitney U-tests to allow assessment of whether the randomisation procedure succeeded in producing comparable arms. Any differences in baseline variables were controlled for in subsequent analyses.

In view of the hierarchical structure of the data, due to cluster-randomisation, resulting in dependencies between measurements, a likelihood ratio test was conducted using MLwiN (Version 2.02) to test for an effect of this structure on study outcomes. There was no evidence of a group effect on any outcome variables.

All analyses were conducted per protocol, that is, the trial population comprised all those who consented to participate in the trial, received both parts of the intervention and provided valid data on cognitions or urges to smoke. Returning for the second session was pre-specified as a condition for inclusion as participants who did not attend the quit date session did not receive the interventions, make a quit attempt, or provided data on urges or cognitive change.

As it was hypothesised that any intervention effect on participants’ urges to smoke would be mediated by changes in certain cognitions, the first step in the analysis was to test for an effect of condition on the cognitive variables at T2 using Analysis of Covariance (ANCOVA), controlling for baseline levels and any baseline differences between groups. Following the manipulation of the cognitive variables, the second step was to test for an intervention effect on urges to smoke, controlling for baseline levels and any baseline differences between groups.

<p>| Table 1 |</p>
<table>
<thead>
<tr>
<th>Participant Characteristics</th>
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<tbody>
<tr>
<td><strong>Demographic characteristics for trial sample (n %)</strong></td>
</tr>
<tr>
<td>Gender</td>
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<tr>
<td>Age</td>
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<td></td>
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<td>Ethnicity</td>
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<tr>
<td>In paid employment?</td>
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<tr>
<td>Qualifications</td>
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<td></td>
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<tr>
<td><strong>Baseline smoking characteristics</strong></td>
</tr>
<tr>
<td>Nicotine dependence (FTND)</td>
</tr>
<tr>
<td>No. cigarettes smoked per day</td>
</tr>
<tr>
<td>Type of medication prescribed</td>
</tr>
<tr>
<td>NRT</td>
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<tr>
<td></td>
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<tr>
<td>Varenicline</td>
</tr>
<tr>
<td><strong>Baseline cognitive variables: mean (SD)</strong></td>
</tr>
<tr>
<td>Negative Reinforcement Explanation for Smoking Scale (NRESS)</td>
</tr>
<tr>
<td>General factor (NRESS-GE)</td>
</tr>
<tr>
<td>Expectation of rapid recovery from dependence (NRESS-RE)</td>
</tr>
<tr>
<td>Evaluation of the ease of reinstatement of dependence (NRESS-EA)</td>
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<tr>
<td>Other cognitive variables</td>
</tr>
<tr>
<td>Positive outcome expectations for smoking</td>
</tr>
<tr>
<td>Self-efficacy</td>
</tr>
</tbody>
</table>

Note: *NS vary due to missing data; ** p < .01.
tioner to the specialist smoking cessation clinic throughout the recruitment period and were then allocated to groups by the clinic administrator.

Concealment
It is not possible to conduct research of this type as a double blind trial. The clinic administrator who assigned smokers to treatment groups was unaware of the group allocation and the lead researcher himself did not open the allocation envelope until recruitment for the group was complete. Participants were informed that the study was comparing two interventions to see if they differed in their potential to aid successful cessation.

Interventions
Both experimental and control interventions were offered as an adjunct to the standard 7-week group smoking cessation treatment, which combines behavioural and pharmacological elements as used within NHS SSSs (withdrawal-oriented therapy; Hajek, 1989). Treatment comprises seven weekly sessions lasting approximately 1 hour each. Pharmacotherapy is also provided on prescription at the clinic. Choice of medication is by individual preference, although varenicline supply is restricted by contraindications and cautions for use. Participants are expected to stop smoking at session 3.

The first part of both the experimental and control interventions was delivered during the second weekly session to allow participants to consider the negative reinforcement explanation for smoking while still actively smoking, and to have the opportunity in the second session to give feedback on their observations. The second session for both conditions was delivered 1 week later on the participants' target quit date (session 3), again during the standard treatment session. Both conditions were delivered by the same trained stop-smoking advisors.

Experimental Intervention
The first part of the intervention comprised three parts (a) a 10-minute presentation delivered in the normal group setting, detailing the main points of the negative reinforcement explanation for smoking, accompanied by group discussion; (b) a leaflet summarising the presentation; and (c) a self-monitoring task to be completed between the sessions. The second part of the intervention consisted of 10 minutes of revision and group discussion 1 week later.

Both the presentation and leaflet were designed in accordance with the Elaboration Likelihood Model (ELM; Petty & Cacioppo, 1986) a model of persuasion used to inform messages that has been applied successfully to changing outcome expectations (Vogt, Hall, Hankins, & Marteau, 2009). The explanation was described in a logical order, detailing how dependence on nicotine and a withdrawal-relief cycle for smoking behaviour develops, how it sustains continued smoking and underlies the subjectively experienced effects of smoking, and how it relates to the quitting process. A number of visual images were used to aid comprehension of the explanation. The slide set is available on request from the lead author.

The self-monitoring task involved participants monitoring their urge to smoke over a three-hour period of abstinence and completing a small ‘task card’ to record the increase in urges to smoke relative to the increasing period of abstinence, and the abrupt alleviation of the resulting tension after smoking. They were asked to reflect on these observations in the light of the negative reinforcement explanation for smoking. The task was intended as an observational behavioural experiment (Bennett-Levy, Westbrook, Fennell, Cooper, Rouf, & Hackman, 2004), suited to challenging and reformulating drug-related beliefs (Beck, Wright, Newman & Liese, 1993).

Control Intervention
The control intervention was delivered in the same two-session format as the experimental intervention. In place of the presentation, participants viewed a DVD on the health risks of smoking entitled Smoking and Human Physiology (AIMS Multimedia, 1993). The running time of the DVD was reduced from 19 minutes to 10 in order to match the length of the presentation in the experimental intervention. The edited DVD details the adverse health consequences of smoking. The information provided by the video was expected to be seen as relevant, but unlikely to provide much additional assistance to quit attempts by people who were already highly motivated to quit. Preliminary piloting work suggested that smokers found the video quite engaging and informative. One week later, participants returned for a revision and discussion session, as in the intervention group.

Assessments
Participants were assessed at baseline prior to receiving the interventions (T1). Outcomes were then measured immediately following the second intervention session (T2, participants’ target quit date) and 1 week later when the participants were expected to be abstinent for the past 7 days (T3).

At T1, demographic data, smoking history and scores on cognitive variables of interest were assessed. Participants’ level of acceptance of the negative reinforcement explanation for smoking was assessed using an eight-item scale developed for the current project. Positive outcome expectations for the benefits of smoking was assessed using three items taken from the Pros of Smoking Scale (Dijkstra, Conjon & DeVries, 2006) and self-efficacy (which was assessed using a single item: ‘How confident are you of succeeding in giving up smoking this time?’) were obtained (see Table 1). Nicotine dependence was assessed using Fagerström Test of Nicotine Dependence (FTND, Heatherton,
check, an effect of condition on participants’ post-
cessation urges to smoke was tested using ANCOVA.

Results

Participant Flow and Study Dropout Rates

Two hundred and five smokers attended group informa-
tion sessions between October 2008 and March 2009 and consented to take part in the study. The total of 145 (81 in the experimental group, 64 in the control group, imbalance due to cluster randomisation) attended both intervention sessions, that is, made a quit attempt, and provided cognitive data. Sixty-five (33 in the experimental group, 32 in the control group) provided valid data on urges to smoke (see above). There was no difference in rates of attrition between experimental and control groups, nor were there any significant differences between participants who valid data on urges and those who did not on any baseline variable.

Effectiveness of the Randomisation Procedure

The effectiveness of randomisation was evaluated by examining mean differences between the intervention and control groups at baseline (see Table 1). Significant baseline differences between groups were found in self-
efficacy and in the medications chosen by participants and were controlled for in the analyses.

Testing for Between Group Differences in Cognitions

Parametric assumptions for ANCOVA were tested and variables were square-root transformed where necessary to achieve a normal distribution. Separate ANCOVA were conducted to test for differences between the conditions in cognitions. Separate analyses were conducted for each of the hypothesised cognitive mediators. The intervention failed to impact on any of the relevant cognitive variables (see Table 2).

Testing for Between Group Differences in Urges to Smoke

Despite a trend in favour of the experimental condition observed also in cognitive variables, there was no significant difference between the groups in urges to

| Table 2 |
| Study Outcomes (Unadjusted) |

<table>
<thead>
<tr>
<th>Cognitive outcomes (T2); mean (SD)</th>
<th>Experimental (n = 81)</th>
<th>Control (n = 64)</th>
<th>Adjusted (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Negative Reinforcement Explanation for Smoking Scale (NRESS)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>General factor (NRESS-GE)</td>
<td>13.50 (2.71)</td>
<td>12.98 (2.82)</td>
<td>.25</td>
</tr>
<tr>
<td>Expectation of rapid recovery from dependence (NRESS-RE)</td>
<td>6.08 (1.97)</td>
<td>6.04 (2.10)</td>
<td>.77</td>
</tr>
<tr>
<td>Evaluation of the ease of reinstatement of dependence (NRESS-EA)</td>
<td>7.70 (1.93)</td>
<td>6.98 (1.97)</td>
<td>.06</td>
</tr>
<tr>
<td>Positive outcome expectations for smoking</td>
<td>9.16 (2.99)</td>
<td>9.59 (3.14)</td>
<td>.52</td>
</tr>
<tr>
<td>Self-efficacy</td>
<td>8.00 (1.79)</td>
<td>7.42 (2.07)</td>
<td>.99</td>
</tr>
<tr>
<td><strong>Smoking outcomes (T3)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Experimental (n = 33–81)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urges to smoke; mean (SD)</td>
<td>2.72 (0.92)</td>
<td>3.07 (1.06)</td>
<td>.33</td>
</tr>
<tr>
<td>1-week abstinence: n(%)</td>
<td>33 (40.7)</td>
<td>33 (51.6)</td>
<td>.19</td>
</tr>
</tbody>
</table>

A chi-square test indicated that there was also no difference between the groups in rates of continuous, CO-verified 1-week abstinence from smoking, \( \chi^2(1) = 1.69, p = .19 \), although it must be noted that the trial was not powered to detect such a difference.

Discussion

Prior to testing the hypothesis that acceptance of the negative reinforcement explanation for smoking facilitates smoking cessation, an intervention is first needed that can successfully communicate these ideas. The current intervention failed to achieve this.

The most likely explanation of the negative result is that the 10-minute presentation provided in the context of a complex multimodal treatment had not been communicated successfully, or was rapidly forgotten. The intervention was delivered within a comprehensive package where other input concerning medications and behavioural advice competed for patients’ attention. Future attempts to evaluate such interventions may need to give it more time and prominence within the treatment program.

A question arises whether such interventions should be evaluated on their own, to avoid interference from the other input. We believe that this may provide data of theoretical interest, but the practical value of such studies would be limited. Cognitive interventions can be expected at best to provide a small addition to more powerful supportive and drug treatments, and are unlikely to be used on their own. Even if they did achieve a small effect in isolation, it would be necessary to test them in combination with standard treatment to make sure that any such impact is not lost among more powerful effects.

Other factors may have contributed to the negative finding. There was relatively limited room for the intended cognitive shift, as many participants subscribed to the negative reinforcement explanation of smoking already at baseline. Allen Carr’s ideas have penetrated the popular consensus in the United Kingdom, and many
smokers attending for treatment were exposed to them even if they had not read his books. There are also smokers for whom the explanation does not apply, that is, smokers who do not smoke regularly react more to external cues rather than to internal discomfort, and do not experience an increase in tension between their cigarettes.

Finally, some trials of cognitive interventions (e.g., Wiggers, Oort, Dijkstra, deHaes, Legemate, & Smets, 2005) concluded that only participants with higher levels of education can benefit from them. Due to the small sample size, we could not explore such mediators, but over half of our participants had no education beyond age 15. Future studies may consider focusing on the type of participants likely to respond to cognitive interventions.

Included in all of the above considerations is a possibility that we did not communicate the core ideas optimally. Although we piloted the explanation extensively and believe that it was understandable and clear, anecdotal evidence from clinicians running the groups was that compliance with the self-monitoring task was not high and this may have contributed to the absence of a significant intervention effect.

As we did not manage to change participants’ cognitions, we were unable to answer the question as to whether a relevant cognition change affects craving and abstinence. The lesson from the trial is that it may be as difficult to change smokers’ cognitions as it is to change their behaviour. Psychological treatments often assume that advice — for example, to use a coping strategy, whether cognitive or behavioural — means that the strategy is now available and will be used. Research on the effects of cognitive–behavioural interventions for smokers rarely verifies whether any new cognitions or behaviours were acquired and any advice followed (e.g., Zernig, Wallner, Grohs, Kriechbaum, Kemmler, & Saria, 2008). Our finding further supports the need to do so.

In summary, our study results demonstrate a lack of a large effect of a brief cognitive intervention. Subtle effects cannot be ruled out, although the potential of this type of intervention to improve on current specialist multimodal treatment with the full range of clients accessing such services is likely to be limited. Nevertheless, given the popularity of the Easy Way to Stop Smoking and the intuitive appeal of its approach, further work to evaluate its effects may be warranted. Future ‘proof of principle’ studies may consider targeting smokers with higher levels of education, using large study samples or excluding comprehensive treatment programmes competing for participants’ attention, and devoting more time and effort to ensure that the intervention ideas are successfully communicated.

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Declaration of Interests

Prof Hajek has undertaken research and consultancy for, and received honoraria for speaking at meetings for, the manufacturers of smoking cessation medications.

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