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A randomised controlled trial of adding expired carbon-monoxide
feedback to brief stop smoking advice: evaluation of cognitive and
behavioural effects

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ABSTRACT

Objective: To determine the effect of adding biomarker feedback (expired air carbon-monoxide) to standard quit advice on cognitive antecedents of behaviour change and smoking cessation and to identify potential effect moderators and mediators.

Design: Smokers (N=160) were randomised to a control (quit advice plus leaflet) or an intervention condition (as control group plus carbon-monoxide level feedback). Cognitive measures were assessed immediately after the intervention and behavioural measures at six months follow-up.

Main Outcome Measures: Primary outcome measures were threat and efficacy appraisal, fear arousal and intention to stop smoking. Secondary outcome measures were quit attempts within the last six months and 7-day point prevalence abstinence.

Results: Threat appraisal was significantly enhanced in the intervention compared with the control group ($t(158)=2.29$, $p=0.023$) as was intention to stop smoking in the next month ($t(151)=2.9$, $p=0.004$). However, this effect on intention to stop smoking was short-lived. Groups did not differ in terms of quit attempts or abstinence at follow-up, but the intervention increased the likelihood of cessation in smokers with higher self-efficacy ($\chi^2(1)=5.82$, $p=0.016$).

Conclusions: Carbon-monoxide level feedback enhances the effect of brief quit advice on cognitive antecedents of behaviour change and smoking cessation rates but further research is required to confirm the longevity of this effect and its applicability to smokers with low self-efficacy.

Key words: *smoking cessation interventions, fear appeal, biomarker feedback, expired air carbon-monoxide, self-efficacy*

INTRODUCTION

Tobacco use remains the leading cause of premature, preventable deaths worldwide killing more people than HIV, illicit drug use and alcohol combined (Ezzati & Lopez, 2003). Smoking rates are still on the rise in many low-and middle income countries and prevalence reductions have stalled in most developed countries (Mackay, Eriksen, & Shafey, 2006). Consequently, there is an unmet need for the development of new smoking cessation interventions to reduce smoking prevalence and prevent future deaths from smoking. One approach to smoking cessation interventions is to provide smokers with biomarker feedback evidencing smoking-related risk or harm. Health is often cited as one of the main reasons for people attempting to stop smoking (Vangeli & West, 2008). However, simply telling people they are at risk of developing a disease in the future is seldom enough to change behaviour (Leventhal, Benyamini, Brownlee, Diefenbach, Leventhal et al., 1997). For this reason, making threatening information more salient, as in the case of fear appeals using biomarkers, has been proposed as a more effective approach (Witte & Allen, 2000).

A number of models have been used to explain the likely mode of action of fear appeals; in particular social cognition models such as the Drive Reduction Model, Protection Motivation Theory or the Parallel Response Model have been ubiquitously used (Ruiter, Abraham, & Kok, 2001). The Extended Parallel Process Model (EPPM, Witte, 1992) attempts to unify and improve on earlier models, postulating that fear appeals trigger coping responses, such as fear control and danger control. People engage in protective behaviour and danger control only when they perceive themselves to be susceptible to severe threats (threat appraisal) and feel that they are able to perform a behaviour (i.e. display self-efficacy) that is effective (i.e. has response efficacy) in averting this risk (efficacy/coping appraisal). If no threat is perceived, there is no response to the fear

appeal. In contrast, when people perceive a threat and positively appraise their efficacy to avert this threat, they are motivated to protect themselves and thus accept the fear appeal message and engage in a behavioural solution (e.g. smoking cessation). Evidence from a meta-analysis suggests that the stronger the fear reaction, the higher the likelihood of a desired effect (Witte & Allen, 2000). However, threat in the absence of a sufficient level of self-efficacy is unlikely to increase motivation to stop smoking (Bishop, Marteau, Hall, Kitchener, & Hajek, 2005). Indeed, in the context of smoking, self-efficacy has been argued to be instrumental to the way in which fear impacts on subsequent behaviour (Dijkstra & Brosschot, 2003) as it could result in the opposite effect because low efficacy appraisal in the presence of a threat leads to an increase in fear levels according to the EPPM. Consequently, a person will become defensive and be more concerned with managing their fear rather than the causes of their fear. People may distort or deny the meaning of threatening information (disengagement beliefs, Bandura, 1986) and engage in cognitive (i.e. avoidance) rather than behavioural (i.e. quitting) solutions.

At the neurophysiological level, it has been proposed that these cognitions are underpinned by fear structures in the limbic system and associated areas that process emotional stimuli to compute appropriate responses (Keightley, Winocur, Graham, Mayberg, Hevenor et al., 2003). This fear network is activated through the presentation of fear-inducing material and the provision of fear congruent or incongruent material is then thought to either strengthen or weaken the respective fear structure according to emotional processing (EP) theory (Foa & Kozak, 1986). The general outline of this theory is supported by the success of exposure therapy in treating various phobias (Barlow, 1988) and fear appeals in changing behaviour (Witte & Allen, 2000), both of which are aimed at reducing or increasing fear levels, respectively.

Based on the reviewed fear appeal and emotional processing literature, it is suggested that fear appeals function by their ability to access neurologically based cognitive fear networks by increasing physiological arousal through the provision of fear-inducing material, thereby initiating emotional processing as expressed by increased fear levels. Biomarkers, as one form of fear appeal, are thought to achieve this due to the nature of the stimulus provided; by personalising information, they counteract perceptions of invulnerability to the health consequences of tobacco-use, which are common among smokers (Strecher, Kreuter, & Kobrin, 1995), thus raising arousal levels and threat perceptions. Socio-cognitive models of persuasion like the elaboration likelihood model (Petty & Cacioppo, 1986) would predict that only messages that are considered relevant for oneself are thoroughly analysed and thus lead to stronger changes in attitudes, which would favour personalised feedback. Further, while biomarkers may provide confirmation of exposure and thus possible harm caused by smoking, they are also helpful in evidencing positive changes in the body after smoking cessation, which would increase a smoker's perception of the response efficacy of cessation. Lastly, biomarkers provide a clear and coherent message about the inherent harm associated with exposure to smoking, which should reduce the likelihood of the threat message being derogated.

Notwithstanding the rationale for using biomarkers, a recent Cochrane review suggests that there is currently insufficient evidence to make a definitive statement about the utility of biomedical risk assessment as an aid for smoking cessation (Bize, Burnand, Mueller, Rege, & Cornuz, 2009). This study aimed to add to this literature by investigating the impact of providing feedback of expired-air carbon-monoxide levels to smokers on cognitive antecedents of behaviour change. There exists a multitude of biomarkers that evidence exposure to smoking (e.g. cotinine), risk (genetic markers) or actual harm caused by smoking (e.g. atherosclerotic plaque). However, in contrast to these

biomarkers, determining carbon-monoxide levels is quick, relatively inexpensive and not invasive, making it an ideal addendum to existing interventions. Feedback of carbon-monoxide levels on its own has had varying success in changing smokers' behaviour but tends to increase smoking cessation rates when compared with minimal control conditions (Jamrozik, Vessey, Fowler, Wald, Parker et al., 1984; Sanders, Fowler, Mant, Fuller, Jones et al., 1989) and when incorporated with motivational interviewing increases abstinence compared with standard treatment (Borrelli, McQuaid, Novak, Hammond, & Becker, 2010). Moreover, carbon-monoxide levels are routinely assessed in UK smoking cessation services (McNeill, Raw, Whybrow, & Bailey, 2005) mainly as a means of validating abstinence. Providing tailored advice on what high expired air carbon-monoxide levels mean for toxin intake and health may offer an opportunity for further increasing motivation to stop smoking and due to the simplicity of this intervention, it may also be a useful addition to brief advice by physicians.

This randomised trial therefore aimed to assess the short- and long-term efficacy of adding tailored carbon-monoxide feedback to brief standard quit advice. In particular, we tested the hypotheses that combining biomarker feedback with generic quit advice will lead to:

- 1.) Appropriate changes in cognitive antecedents of behaviour change (increase in threat appraisal, self-reported fear levels and intention to stop smoking) compared with generic quit advice alone (primary outcomes).
- 2.) Appropriate changes in behaviour (increase in quit attempts and smoking cessation rates) at 6 months follow up compared with generic quit advice alone (secondary outcomes). On the basis of EPPM and EP, it was further postulated that impact of the intervention on smoking cessation would be moderated by self-efficacy and mediated by fear levels.

METHODOLOGY

Procedure

This randomised control trial was carried out in 2006/7 at University College London as part of a laboratory study that assessed the differential exposure of hand-rolled and manufactured cigarette smokers to carcinogens (see Shahab, West, & McNeill, 2009). Participants were recruited from the general population through advertisements in local newspapers, flyers, emails, or posters on public bulletin boards at and around University College London. The study was presented as a laboratory-based study, not as an intervention study, and smokers were not required to intend to stop smoking. Smokers who responded to the advertisements were screened for eligibility through a telephone interview and provided with information about the study. Participants were included if they were between 18 and 60 years of age and had smoked more than five cigarettes daily for the past year. Smokers were ineligible if they had a history of lung or heart disease or if they were pregnant.

Participants visited the laboratory on two occasions, 24 hours apart. At the first visit, the main purpose of the study, as pertaining to the measurement of exposure to carcinogens, was explained and participants were asked to sign a consent form. At this stage participants also completed the baseline questionnaire (T1). Following the questionnaire, smokers provided a breath sample both before and after having smoked a cigarette by blowing into a monitor which analyses expired carbon-monoxide (CO) content. Since alveolar CO levels change relatively rapidly with exposure to cigarette smoke, participants were asked to refrain from smoking half an hour before each laboratory sessions to obtain a standardised reading. Urine and saliva samples were also collected. At the beginning of the second visit, 24 hours later, the researcher randomly assigned participants to the control or treatment condition by means of

opening a sealed envelope containing the random number generated group allocation (restricted to equal numbers per group). Participants were blinded to the allocation but this was not possible for the researcher providing the intervention. Participants again provided a breath sample before and after smoking a cigarette before urine and saliva samples were collected. Both the control and experimental group were provided with a generic leaflet about lung disease. The control group received standardised brief advice to quit smoking; the treatment group also received brief targeted feedback about their CO levels in relation to the development of cardiovascular and respiratory diseases. At the end of the session, all participants completed the outcome questionnaire (T2); received a debriefing letter (in the treatment group with the personalised CO reading); agreed to being contacted for a follow-up phone call (all participants consented) and received £50 for their time. Six months after the second laboratory visit, participants were contacted by a researcher blinded to group allocation to complete the follow-up questionnaire (T3). If participants did not answer their phone on more than three occasions, they were contacted by email and if this failed by, post. The study received ethical approval from the UCL Ethics Committee.

Participants

A total of 160 participants were included in this study, of whom, 51 (31.9%) were lost to follow-up (see Figure 1); there were no significant differences between groups in terms of attrition. Participants lost to follow-up did not differ on any of the assessed demographic or cognitive variables other than age; those lost to follow-up were younger than those who remained in the study ($t(119)=2.2$, $p=0.027$). The characteristics of included participants are presented in Table 1.

Figure 1 about here

Intervention

Everybody received a generic leaflet about the dangers of smoking and respiratory disease. In addition to the leaflet, smokers in the control group were given standardised brief quit advice (“I would urge you to stop smoking; quitting is the single-most important thing you can do to feel better & improve your health”) while smokers in the intervention group also received individualised brief quit advice that related their carbon-monoxide reading to cardiovascular, malignant and non-malignant lung diseases. A post-doctoral researcher described how carbon monoxide (CO) from smoking causes damage and illustrated how their CO reading relates to disease risk on a chart plotting smoking intensity against risk of developing heart and airways disease (“Your CO reading suggests that you smoke cigarettes very intensely and this means you are at greater risk of suffering heart problems and lung cancer as shown on this graph’). Participants in this group were also given a print-out that included their personal CO level in order to make the result more salient. This intervention lasted about three minutes and was aimed to increase smokers’ perception of their own susceptibility to smoking-related diseases and thus raise fear levels regarding smoking in order to increase quit intentions and motivate cessation. Participants in the control group were not shown their CO reading and told that the measurement of expired air was standard procedure for the laboratory study.

Leaflet

A leaflet was developed and piloted on smokers attending a smoking cessation clinic in North London between January and February 2006. This leaflet contained information about the link between smoking and respiratory illnesses and was designed to increase smokers’ awareness of both the seriousness of these diseases and their risk of developing diseases thus manipulating perceived severity and susceptibility. In addition,

the leaflet also contained information about the effectiveness of smoking cessation for preventing smoking-related diseases (thus attempting to raise perceived response efficacy) as well as practical information about how to get support for quitting smoking (attempting to raise perceived self-efficacy).

Measures

Biomarkers (expired air carbon-monoxide and cotinine)

A standard monitor (Smokerlyzer[®], Bedfont Scientific Ltd, Kent, UK) was used to obtain expired air carbon monoxide levels. A reading was taken before and after having smoked a cigarette following a minimum of a half-hour interval of not smoking. Carbon-monoxide 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 COPD, cystic fibrosis and asthma (Kharitonov & Barnes, 2002) as well as lung cancer (Law, Morris, Watt, & Wald, 1997) and carbon-monoxide 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 non-smokers) 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 a.) reduce 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 behaviour) 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 behaviour. For each of the measures, the mean value was used in analysis.

Behavioural (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 ($\alpha=0.05$) to detect a medium-to-large effect size (Cohen's $d \sim 0.5-0.7$) for group differences in primary (cognitive) and secondary (behavioural) 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 ANOVA, change scores were calculated and group differences assessed as this yields equivalent results and

has the advantage that non-parametric 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 tests were used to validate results. Multivariate logistic regression was used to predict behavioural outcomes using treatment allocation, socio-demographic 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 in order to be able to estimate moderation effects. Mediation (using the Sobel method) and moderated mediation were analysed with bootstrapping in SPSS (Preacher, Rucker, & Hayes, 2007). All analyses of behavioural 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 five years but only a 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.

Table 1 about here

1.) 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 due to 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 hypothesised threat appraisal in the form of perceived susceptibility increased significantly more in the intervention than control group ($t(158)=2.33$, $p=0.021$).

Table 2 about here

Although self-reported fear levels were significantly increased across visits in the intervention group only ($t(80)=3.2$, $p=0.002$), this increase was not significant relative to the control group ($t(158)=1.4$, $p=0.180$). 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 ($t(151)= 2.9$, $p=0.004$). 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 ($t(50)=0.34$, $p=0.731$ and $t(47)=0.97$, $p=0.335$, respectively).

Figure 2 about here

2.) 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, socio-demographic and smoking characteristics. The only predictors to emerge were past quit

attempts (OR 6.37; 95%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 five years and those who had lower cotinine values were more likely to have attempted to quit in the following 6 months.

Overall, 5% (95%CI 2.2-9.6) of participants were abstinent at 6 months. While more smokers in the intervention (6.2%, N=5) than control group (3.8%, N=3) had stopped at follow up, this difference was not significant (Fisher's exact test, $p=0.374$). Logistic regression including group allocation, socio-demographic and smoking characteristics yielded no significant baseline predictor of abstinence, although those who were less nicotine dependent were marginally more likely to have quit smoking (OR 0.45; 95%CI 0.20-1.05; $p=0.065$).

As the distribution of data yielded unstable results in logistic regression, log-linear models were fitted to evaluate the possibility of baseline self-efficacy moderating the effect of the intervention on smoking cessation. Self-efficacy moderated the impact of the intervention on smoking cessation as shown by a significant three-way effect of smoking rate at follow-up, baseline self-efficacy level and group allocation in the log-linear model (Likelihood ratio $\chi^2(1)=5.82$, $p=0.016$, see Figure 3). As expected, only in the intervention but not the control group were high self-efficacy levels associated with a greater quit rate (Fisher's exact test, $p=0.022$). Indeed, in the intervention group no participants with low-self-efficacy had quit. Amongst those with high self-efficacy levels, there was a borderline effect of the intervention on smoking cessation compared with the control group (Fisher's exact Test, $p=0.089$). Interestingly, baseline self-efficacy did not moderate the impact of the intervention on quit attempts.

Figure 3 about here

Sobel mediation analysis showed that post-intervention fear levels did not mediate the impact of the intervention on smoking cessation. Given the moderating effects of self-efficacy, moderated mediation was also analysed. Self-efficacy did not moderate mediation either in the path from treatment allocation to fear levels or in the path from fear levels to smoking cessation. However, the analysis confirmed that self-efficacy levels moderated the impact of the intervention on abstinence in the mediation model (group by self-efficacy interaction term coefficient 0.16, $p=0.02$).

DISCUSSION

The purpose of this study was twofold. The primary aim was to assess changes in cognitive antecedents of behaviour change following an intervention that provided smokers with personalised biomarker feedback of their expired air CO levels. The secondary aim was to evaluate the efficacy of this approach to motivate smoking cessation and to delineate possible moderators and mediators of behaviour change as postulated by the extended parallel processing model (EPPM) and the emotional processing model (EP).

A general increase in perceived threat and efficacy levels was observed in both the control and treatment group. This may reflect the impact of the provided leaflet, designed to raise threat and efficacy appraisal, as well as the provision of brief quit advice. However, as hypothesised, personalised quit advice incorporating CO level feedback led to a greater change in perceived susceptibility in treatment group participants compared with the generic quit advice that was provided to control group participants. Given that our perceived susceptibility measure asked smokers to rate their disease risk compared

with non-smokers, this suggests that biomarker feedback may reduce unrealistic optimism regarding the acquisition of smoking-related diseases. Moreover, as anticipated, showing smokers evidence of exposure to, and thus potential harm from, cigarette smoke increased their fear levels across visits but not significantly more than in the control group. These results are consistent with EP theory in that the presentation of CO levels (fear-inducing material) would have enabled access to the fear network (leading to increased fear levels), which could then, by presentation of incongruent material (quit advice and leaflet) be modulated to include new information resulting in changed threat perceptions (increased susceptibility). Consequently, there was also an increase in intention to stop smoking in the treatment group compared with the control group. However, this change was transient and intention levels had fallen back to baseline values at the six months follow-up suggesting that the stimulus used in this study may have been too weak to induce lasting changes in cognitions. This is consistent with similar transient effects on cognitive antecedents of behaviour change in smoking cessation trials using different biomarkers (e.g. Lerman, Gold, Audrain, Lin, Boyd et al., 1997; McClure, Ludman, Grothaus, Pabiniak, Richards et al., 2009). The findings, nonetheless, underline not only the importance of the emotive and preconscious level over and above a purely semantic and intellectual understanding of the threat of smoking but also the motivating power of emotions in order to alleviate the unpleasant state of fear (Easterling & Leventhal, 1989).

In terms of quit attempts no group differences were observed. However, consistent with earlier research (Sutton, 1994; Nides, Rakos, Gonzales, Murray, Tashkin et al., 1995) past behaviour, i.e. attempting to quit in the last 5 years, and the amount smoked, i.e. cotinine levels, were predictive of future quit attempts. Seven-day point prevalence abstinence was five percent at the six months follow-up, which is comparable to rates

observed in some (Page, Walters, Schlegel, & Best, 1986; Wilson, Wakefield, Steven, Rohrsheim, Esterman et al., 1990) but not most other studies that have looked at the effect of brief smoking cessation advice (Lancaster & Stead, 2004). Although more people in the treatment than control group had stopped smoking, in contrast to a previous studies using CO feedback (Risser & Belcher, 1990; e.g. Jamrozik et al., 1984; Sanders et al., 1989), this difference did not reach a significant level overall.

As hypothesised, self-efficacy levels moderated the impact of the intervention. Whereas in the treatment group there was a significant difference in smoking rates between those with low and high self-efficacy, there were no such differences in the control group. Indeed, no-one with low self-efficacy in the treatment group had stopped smoking. This finding would support the view that high self-efficacy may instigate behavioural (quit) and low self-efficacy cognitive (disengagement) solutions, therefore leading to improved outcome in only one of the sub-groups in the intervention. In contrast, in the control group, self-efficacy would not be expected to significantly moderate smoking cessation in the absence of sufficient threat appraisal (Witte, 1992); that is, since the intervention in the control condition was less intensive, perceived threat was only partly increased and therefore would be less likely to lead to further efficacy appraisal. However, contrary to expectation, fear levels did not appear to mediate the impact of the intervention on any behavioural outcome. This may reflect both limited power to detect mediation in the absence of a direct overall effect of the intervention on behavioural outcomes as well as the limitations of self-report in assessing fear levels.

There are a number of reasons why biomarker feedback lead to short-term changes in cognitive antecedents of behaviour change but not to behaviour change itself. This study was primarily powered to detect differences in primary not secondary outcomes and

owing to a greater attrition than expected, power to detect more subtle differences in cognitive outcomes at follow-up was small. Moreover, the sample was recruited to participate in a laboratory study about smoking (not cessation) and were therefore not explicitly seeking to quit; participants were also comparatively young, both of which could have biased results and may explain why cessation rates in the treatment and control groups were at the low end compared with similar interventions using biomarker feedback (see Bize et al., 2009). In part, the result may also present an artefact of the study design. CO feedback has been shown to impact on smoking cessation when treatment and control conditions are not equally matched, that is when an intensive treatment is compared with a minimal control (Jamrozik et al., 1984; Sanders et al., 1989). However, in the current study both treatment and control conditions were of similar duration to clarify the effect of expired air carbon-monoxide feedback

This study has a number of limitations. The sample was not randomly selected, which may have introduced systematic differences in comparison with the general population. Yet, demographic data (with the possible exception of age) compared favourably to large-scale epidemiological studies. While it is assumed that the low-demand nature of this study minimised self-report bias on measures, this cannot be ruled out. Although biochemical outcome validation would have been desirable, it was not practically possible in this intervention. Another problem relates to the quasi-experimental design. It is feasible that partaking in the study itself may have led to an increase in the cognitions assessed as there was no control arm in which no quit advice or leaflet was provided. However, this is unlikely since the laboratory study did not attempt to change perceptions and behaviour but rather the opposite: smokers were asked to continue smoking as normal over the 24 hours of the study period. Lastly, in order to gauge better the influence of EP, it would have been preferable to include more direct measures of

emotional changes, such as blood pressure. As Foa and Kozak (1986) point out, people have an imperfect knowledge of the information contained in their fear networks, therefore physiological arousal would be particularly compelling evidence that their fear network had been activated.

In conclusion, this study provides novel insights into the process of fear appeals in a non-clinical population that was explicitly unmotivated to quit smoking and suggests that the effects of offering a leaflet and brief quit advice on cognitive antecedents of behaviour change can be enhanced by the provision of a simple intervention designed to increase emotional processing. Presenting feedback of expired-air carbon-monoxide levels to smokers was shown to increase perceptions of susceptibility to smoking-related illnesses and was associated with a greater intention to stop smoking. However, findings suggest that these effects are short-lived and do not necessarily translate into action but rather are modified by perceptions of self-efficacy. This study could only provide partial confirmation for the causal interaction of the constructs of the EPPM and the predictions made by the EP theory.

Future research should recruit a larger sample to overcome power issues, have an additional intervention arm (minimal – no leaflet or feedback) to avoid confounding and use more impactful biomarkers to build on the findings from this study. CO feedback may not be enough to instigate such behaviour change in most smokers and other more visually powerful biomarkers, e.g. those that display actual harm caused by smoking such as atherosclerotic plaques (e.g. Bovet, Perret, Cornuz, Quilindo, & Paccaud, 2002), may prove generally more effective as an addendum to fear appeals and it would be interesting to test this in future studies. Nonetheless, this study provides some

preliminary evidence for the utility of a simple addendum to quit smoking advice. In terms of clinical practice, our results have three implications. First, while fear appeals will raise the issue of smoking cessation among all smokers, they may be a more effective tool for highly motivated and self-confident smokers and, arguably, be restricted to these given their potential for discouraging other smokers from quitting. Second, given that CO levels vary depending on the amount of cigarettes smoked, this kind of feedback may be particularly useful for cut-down to stop interventions as reductions in CO levels could over time increase efficacy perceptions of smokers and further bolster motivation to stop completely. Third, the effect of self-efficacy on smoking cessation and intention to stop highlights the need for successful smoking interventions to include techniques that can increase self-efficacy per se, such as proposed by Bandura (1986), in order to translate the momentum gained from any type of health interventions into concrete behavioural outcomes.

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COMPETING INTERESTS

Lion Shahab has received an honorarium for a talk and travel expenses from Pfizer. Robert West undertakes research and consultancy for the following developers and manufacturers of smoking cessation treatments; Pfizer, J7J, McNeil, GSK, Nabi, Novartis and Sanofi-Aventis. Robert West also has a share in the patent of a novel nicotine delivery device.

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TABLES AND FIGURES

Table 1: Participant characteristics[#]

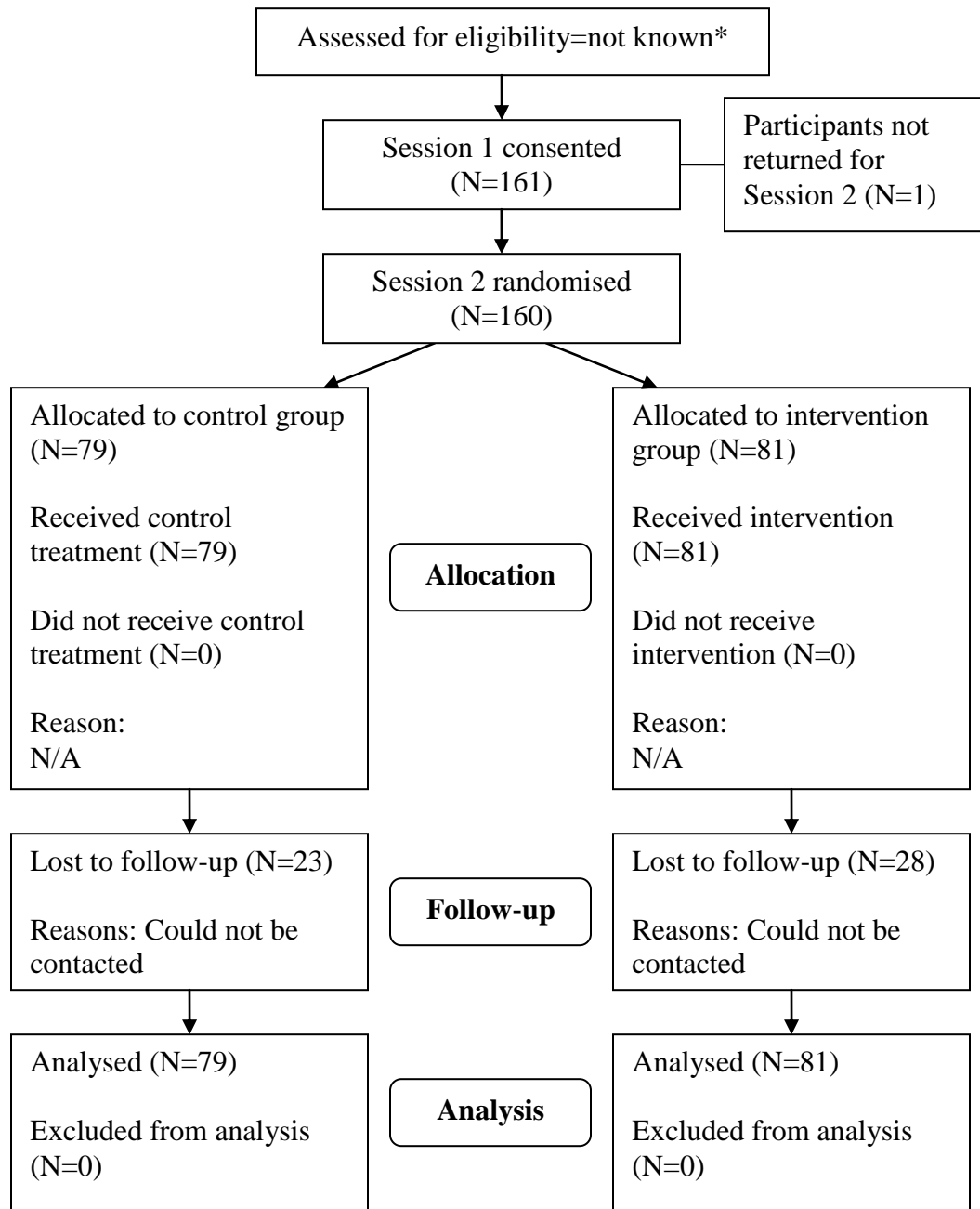
| | All smokers (N=160) | Intervention (N=81) [#] | Control (N=79) [#] |
|--|------------------------|-------------------------------------|--------------------------------|
| <i>Sociodemographic characteristics</i> | | | |
| Mean (SD) age | 31.7 (10.7) | 30.9 (10.7) | 32.6 (10.8) |
| Percent (N) male | 56.3 (90) | 55.6 (45) | 57.0 (45) |
| Mean (SD) IMD ^{†‡} | 32.0 (13.1) | 32.2 (13.3) | 31.7 (12.9) |
| Mean (SD) BMI [†] | 23.9 (4.0) | 23.6 (4.1) | 24.1 (3.8) |
| <i>Smoking characteristics</i> | | | |
| Mean (SD) cigarettes per day | 13.8 (5.9) | 13.4 (5.8) | 14.3 (6.0) |
| Mean (SD) length of time of smoking in years | 14.3 (11.1) | 13.7 (11.0) | 15.0 (11.3) |
| Mean (SD) HSI ^{†‡} | 2.4 (1.5) | 2.3 (1.6) | 2.5 (1.5) |
| Percent (N) quit attempt in last 5 year | 56.3 (90) | 54.3 (44) | 58.2 (46) |
| Percent (N) Want to quit next month | 11.3 (18) | 11.1 (9) | 11.4 (9) |
| Mean (SD) Baseline cotinine levels in ng/ml | 224 (141) | 211 (139) | 237 (144) |
| Mean (SD) Post-cigarette CO level in ppm ^{†‡} | 18.5 (7.7) | 18.0 (7.5) | 19.1 (7.9) |

[†]IMD: Index of multiple deprivation, BMI: Body mass index, HSI: Heaviness of smoking index (Scale 0-6), ppm: parts per million; [‡]25 cases missing; [‡]2 cases missing [#]There were no baseline differences between groups

Table 2: Change in cognitive outcomes pre- to post-intervention

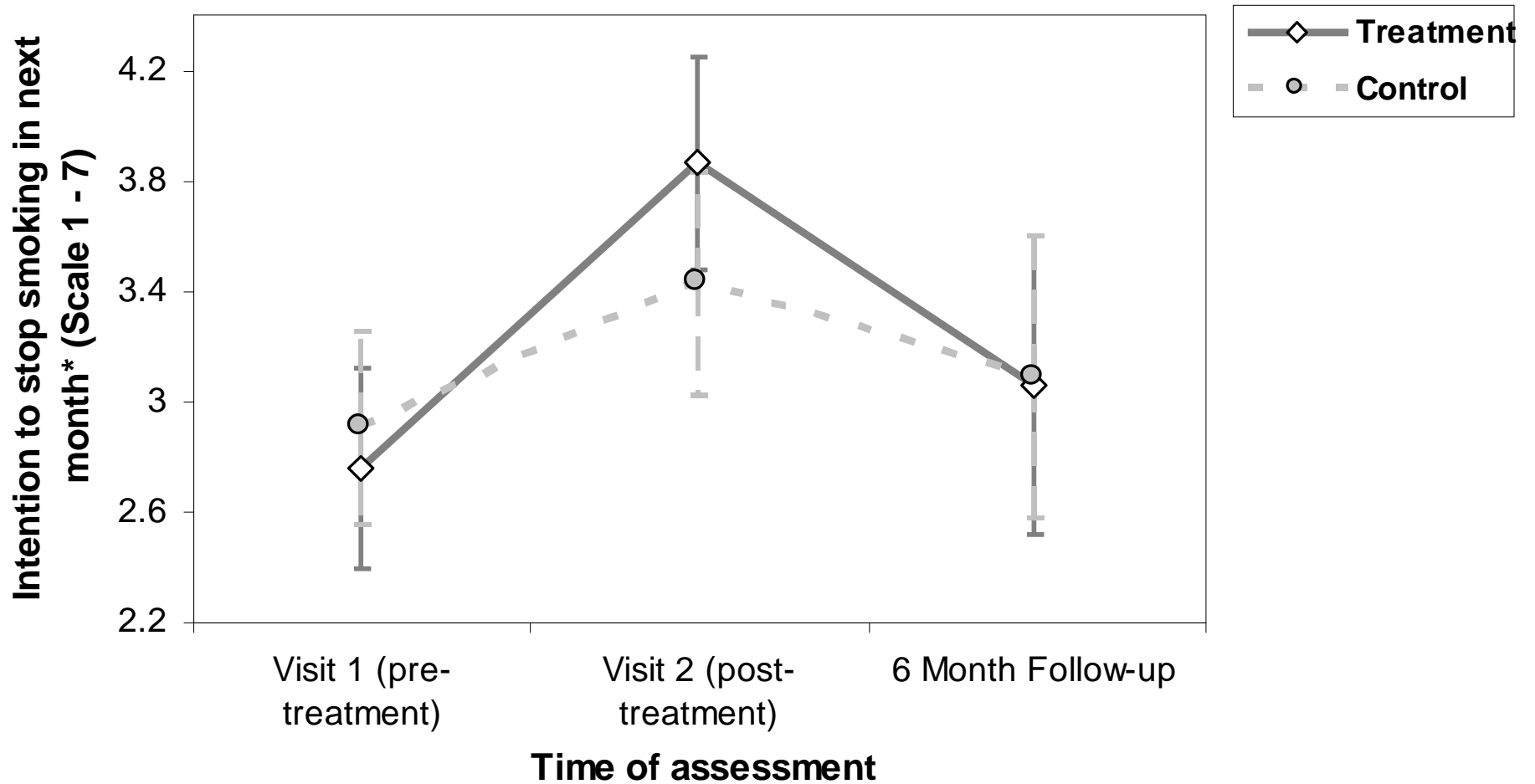
| Self-reported measures | | Intervention (N=81) | Control (N=79) | <i>p</i> (between group) |
|------------------------|--------------------------|--|-----------------------------|-----------------------------|
| | | Mean Change Score (95%CI); <i>p</i> (within group) | | |
| Perceived efficacy | <i>Self-efficacy</i> | 0.346 (0.101–0.590); 0.006 | 0.260 (-0.011–0.530); 0.059 | 0.638 |
| | <i>Response efficacy</i> | 0.253 (0.015–0.491); 0.037 | 0.348 (0.049–0.647); 0.023 | 0.620 |
| Perceived threat | <i>Susceptibility</i> | 0.654 (0.401–0.908); <0.001 | 0.272 (0.069–0.476); 0.009 | 0.021 |
| | <i>Severity</i> | 0.099 (-0.312–0.510); 0.634 | 0.013 (-0.136–0.162); 0.866 | 0.699 |
| Fear | | 0.401 (0.152-0.650); 0.002 | 0.184 (-0.020-0.387); 0.076 | 0.180 |
| Intention to stop | | 1.108 (0.793-1.420); <0.001 | 0.519 (0.270-0.768); <0.001 | 0.004 |

Figure 1: Participant flow chart



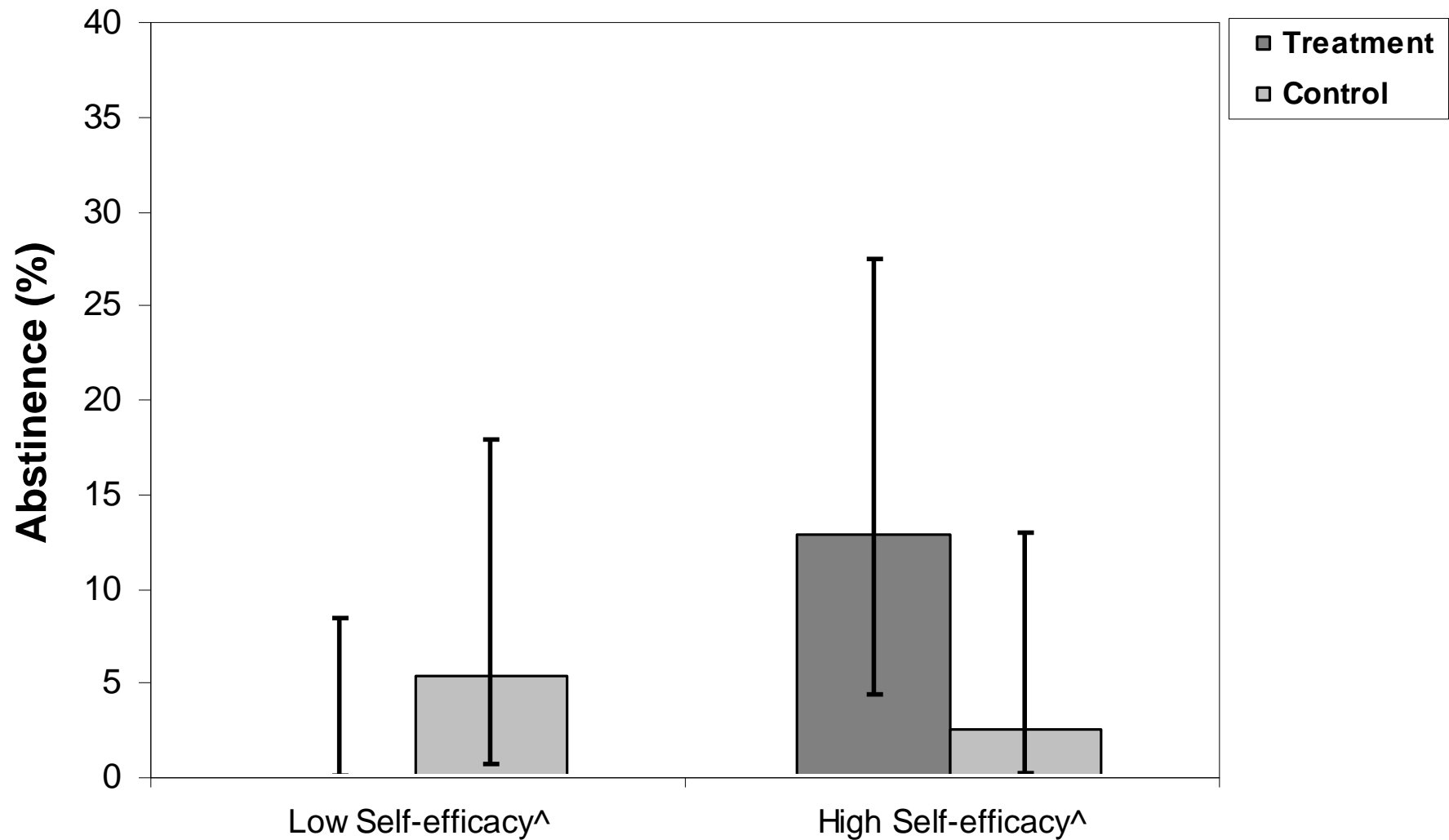
*Pre-consent data not recorded

Figure 2: Intention to stop smoking in the next month by group over time



*Excluding abstainers and drop-outs

Figure 3: Point Prevalence Abstinence at 6 months follow up by group and self-efficacy level[#]



[#]Pre-intervention self-efficacy (T1); [^]Median split (low: 1 < 3.5; high: 3.5–7)