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The effect of a minimal contact smoking cessation programme in out-patients with chronic obstructive pulmonary disease: a pre–post-test study☆

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Abstract

This study assessed the efficacy of an individual, minimal contact, smoking cessation programme in chronic obstructive pulmonary disease (COPD) patients, using a pre–post-test design. The study was part of a large ongoing investigation into the efficacy of self-management in patients with COPD (the COPE-study). In total, the participants received three 15–30 min home-based counselling sessions. Additionally, patients were provided with a written self-help manual. On the patient's request, the chest physician prescribed bupropion or nicotine replacement therapy (NRT). Cessation rates after nine months were based on self-report, and afterwards confirmed by salivary cotinine analysis. Patients were biochemically classified as smoker if their cotinine levels exceeded 20 ng/ml. At baseline, one third of the 269 patients in the COPE-study were active smokers (according to self-report). Almost 70% ($n = 64$) of these patients were willing to participate in the smoking cessation program. After nine months follow-up, 23 (36.5%) patients self-reported abstinence. However, the cotinine validated abstinence rate was much lower: 12.7% ($n = 8$), implying that the actual abstinence rate is severely overestimated by self-report in this study. The results suggest that the (validated) effectiveness of this intervention is probably in line with that of comparable programmes for "healthy" persons. However, considering the urgent need for quitting in COPD patients, a more intensive programme resulting in higher quit rates, seems to be required for this high-risk population.

Keywords: COPD; Smoking; Smoking cessation; Cotinine; Validation

1. Introduction

Chronic obstructive pulmonary disease (COPD) is the result of many years of accelerated decline in pulmonary function due to heavy cigarette smoking [1,2]. Smoking cessation is the most important intervention in the management of COPD because it reduces the decline in pulmonary function and improves the prognosis for the COPD patient [3,4]. Furthermore, quality of life improves when smokers become non-smokers [5]. As COPD patients generally have a long smoking history, they are considered strongly addicted to smoking, both physically and psychologically. A study of Jimenez et al. [6] found that smokers with COPD have higher tobacco consumption and higher dependence on nicotine. This different smoking pattern for COPD patients is likely to require a specific smoking cessation programme.

In many countries, physicians indicate that they are reluctant to discuss smoking cessation with their patients. This might be explained by lack of training in the management of nicotine addiction in clinical practice and lack of routinely available smoking cessation programmes [7]. Currently, various smoking cessation programmes that include pharmacological or behavioural elements have been developed, but these are often unstructured and, more important, not especially tailored to COPD patients. In The Netherlands, the minimal contact smoking cessation programme (MIS) for "healthy" smokers has been disseminated in 1995 among general practitioners (GP) [8]. In a trial involving subjects with GP contacts largely unrelated to smoking, a 13.4%

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self-reported abstinence rate among patients receiving the MIS was observed, compared to 7.3% in a usual care control group [9]. A recent survey indicated that 28% of the general practitioners in The Netherlands are using the MIS [10]. This implies that at present only a minority of Dutch COPD patients will be exposed to this intervention.

We modified the MIS into a slightly more intensive intervention for COPD patients, administered by pharmacy assistants. The modified MIS was tested in the smoking population of a large randomised controlled trial of self-management education in COPD (the COPE-study). We assessed the efficacy of the modified MIS on the basis of a one group pre–post-test design, using both self-reported and biochemically validated cessation rates.

2. The intervention

2.1. Theory

The MIS is based on a frequently applied behavioural change theory: the ASE-model [11–13]. This model describes the determinants of both behaviour and behavioural change. The determinants of behavioural change in the ASE-model are Attitude, Social support and self-Efficacy. Attitude represents the outcome expectations of a person with regard to the intended behaviour (for example, smoking cessation) based on a balance of pros and cons. Social support represents the positive or negative incentives from the person's social environment. Attitude and social support together determine the person's motivation and intention to stop smoking. Self-efficacy expectations refer to the belief in one's capabilities to execute the intended behaviour successfully. The person needs to increase general and smoking-specific self-efficacy expectations and outcome expectations, and the person's environment needs to provide adequate social support, in order to strengthen the person's determination to quit smoking. For example, an individual may be convinced that quitting smoking is beneficial with regard to pulmonary complaints and exercise capacity (high outcome expectations), but at the same time this person may not consider himself capable of abstaining from cigarettes in stressful situations (low self-efficacy). Most important in the ASE-model are the self-efficacy expectations because these can directly change behaviour. The internal relations of the determinants of the ASE-model are shown in Fig. 1.

Smoking cessation is not a single event but a comprehensive process. Prochaska's Transtheoretical model describes this process in six stages of motivational change [14]. The counselling strategy in this study is tailored to the smoker's motivational stage in order to obtain points of departure for efficient and effective counselling. Further, the MIS is intended to increase both the outcome expectations and self-efficacy expectations of the patient. Counselling in the MIS is personalised and consisted for a large part of problem solving strategies for expected difficult moments in order to increase self-efficacy expectations.

2.2. Practice

In preparation for the execution of the modified MIS, 14 pharmacy assistants from local pharmacies and a respiratory nurse received 4 h of skills training, which consisted of education about the theoretical background of smoking cessation, information about COPD, COPD-specific problems in smoking cessation, and training of counselling skills. In addition, the counsellors were supplied with a written manual that described the procedure of the MIS in detail.

The smoking cessation programme was offered to the current smokers included in the COPE-study. When motivated to quit (i.e. smoking cessation contemplators according to the stages-of change model), the pharmacy assistant contacted the patient to arrange a first session. If the patient's pharmacy was not involved in the study, our respiratory nurse performed the counselling. The intervention took place at the patient's home to increase the involvement of family members in the smoking cessation process (increase of social support). Additionally, home counselling gives counsellors more insight in the social barriers for smoking cessation, allowing them to anticipate problems better.

In total, the participants received three 15–30 min home-based counselling sessions. The goal of the first session was

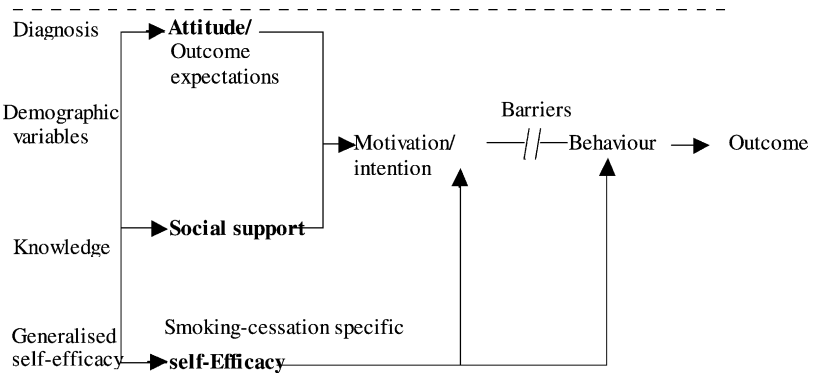

Fig. 1. Attitude, Social support, self-Efficacy model.

to analyse the determinants of the ASE-model specific for smoking cessation. Problems and barriers were discussed, and the patients were provided with a written self-help manual. During this first session, the counsellor arranged a quitting date with the patient. On the patient's request, the chest physician prescribed bupropion sustained release (SR) (Zyban®) or nicotine replacement therapy (NRT). The first follow-up session was scheduled two weeks after the quitting date and the second session after six–eight weeks. The goal of the second and third session was to keep the patient motivated and to support the quitting process.

3. Methods

The smoking cessation programme was one group pre–post-test comparison nested in the COPE-study. The hospital's ethical committee approved the protocol for the COPE-study, and written informed consent was obtained from all participants.

The study population consisted of current smokers included in the COPE-study. These were outpatients with a clinical diagnosis of stable COPD of the department of pulmonary medicine of Medisch Spectrum Twente in Enschede, The Netherlands. All patients were aged between 40 and 75 years. We confirmed the diagnosis of COPD by spirometry (ratio of FEV1 to FVC $\leq$60%) and absence of a bronchodilator response (ΔFEV1% predicted following inhalation of 80 mcg of ipratropium bromide $\leq$12%) [15]. Patients were excluded if they had asthma as primary diagnosis, medical conditions with a low survival rate, or other diseases that might influence compliance, bronchial symptoms or lung function (e.g. alcoholism, cardiac insufficiency, and sarcoidosis).

At baseline, we obtained information about demographic variables, nicotine dependency, smoking history and behavioural determinants. The patients completed a modified version of the Fagerström questionnaire [16] to study the degree of physical nicotine dependency. This questionnaire consists of eight items summing up to a maximum score of eight points.

At baseline and after nine months follow-up, smoking habits were assessed by an interview by a lung function technician. Two questions were asked: "Do you smoke cigarettes? Yes/No", and, if yes, "What is the average daily cigarette consumption?" Additionally, a salivary sample was collected for cotinine assessment. The samples were frozen until assayed. Cotinine is the major metabolite of nicotine with a half-life in the body of about 20 h. It takes about four days of abstinence from smoking for the cotinine level to decrease to that of a non-smoker [17]. The laboratory of our hospital assayed cotinine from the salivary samples using a gas chromatography technique (GC–MS) [18]. The accuracy and precision of the method was checked by means of reference samples.

Table 1
Smoking status according to cotinine levels at baseline[a]

	Smoker (cotinine >20 ng/ml)	Non-smoker (cotinine $\leq$20 ng/ml)	Total
Self-reported smoker	68	2	70
Self-reported non-smoker	16	102	118
Total	84	104	188

[a] Based on the patients with valid saliva samples.

Prevalence rates of self-reported and biochemically validated smoking cessation at baseline and nine months follow-up were estimated with corresponding 95% confidence intervals (CI). Patients were biochemical classified as smokers if their salivary cotinine level exceeded 20 ng/ml [19]. Saliva samples were considered to be invalid if less than 500 μl saliva was available, and the cotinine concentrations were between 20 and 100 ng/ml. The proportion of patients who misstated their smoking habits, in the literature referred to as "smoking deceivers", was assessed [20,21].

Statistical analyses were performed using SPSS version 10 [22].

4. Results

Two hundred and sixty-nine (269) COPD patients were enrolled in the COPE-study of whom 90 (33%) self-reported to be current smokers. At baseline, we obtained saliva samples of 197 (73%) patients: 126 patients reported to be non-smokers and 71 to be smokers. Cotinine concentrations of nine saliva samples were invalid, leaving 188 valid cotinine samples. Table 1 illustrates the calculation of the proportion of expected smokers at baseline using the patients with valid cotinine samples. Of the 118 patients with self-reported non-smoking, 16 (13.6%) were classified as smokers based on their cotinine levels. Assuming that the subgroup with valid cotinine values is representative for the study population, we calculate that about 45% (84/188) is a smoker at baseline.

Of the patients who admitted smoking, 64 (64/90; 71%) were willing to participate in the smoking cessation programme. The baseline characteristics of this population are shown in Table 2.

About half of the patients ($n = 29$) used nicotine replacement therapy ($n = 23$) and/or bupropion SR (Zyban®) ($n = 9$).

Of the 64 patients who were enrolled in the smoking cessation programme, 63 could be followed for nine months. At that time, 23 patients reported abstinence (36.5%). However, cotinine levels exceeding 20 ng/ml were detected in 12 out of these reported quitters, and in another 3 no saliva could be obtained. So, the actual abstinence rate is between 12.7%

Table 2
Baseline characteristics of COPD patients who entered the smoking cessation program

Male (%)	55 (85.9)
Age (mean ± S.D.) (years)	62.6 ± 7.1
Body mass index (mean ± S.D.) (kg/m^2)	26.4 ± 4.7
Education level (%)	
Low	41 (64.1)
Intermediate	15 (23.4)
High	7 (10.9)
FEV1% predicted (mean ± S.D.) (l)	49.4 ± 14.9
Pack (mean ± S.D.) (years)	44.3 ± 16.8
Daily cigarette consumption (mean ± S.D.)	16.5 ± 12.4
Fagerström score (mean ± S.D.) ($n = 55$)	3.1 ± 2.1
Quit attempts (%) ($n = 48$)	
0	14 (28)
1–2	16 (32)
2–4	9 (18)
>5	9 (18)

(8/63) and 13.3% (8/60) depending on the interpretation of the missing values.

An attempt to discriminate between successful ($n = 8$) and unsuccessful patients ($n = 55$) on the basis of the baseline characteristics described in Table 2 did not show any predictors.

5. Discussion and conclusion

This study has two main findings. First, disappointingly few COPD patients actually quit smoking after nine months follow-up. Second, biochemical validation is essential in smoking cessation programmes. In this study, 12 (52%) of the self-reported non-smokers were not truthful about their smoking status. Both findings will be discussed below.

5.1. Effectiveness of smoking cessation programme

Our success rate may appear to be low when compared with two other behavioural smoking cessation studies in patients with COPD published after 1990: 33 and 35% success in the intervention groups, respectively [23,24]. Several reasons might explain this. The smoking interventions in both studies were more intensive and, in fact, it is stated that the intensity of cessation counselling has a strong dose-response relation with effectiveness [25]. Another reason can be the timing of the intervention. In the study of Pederson and co-workers, counselling occurred while the patients were in hospital. Hospitalisation can be an opportune time to intervene with smoking, because patients can be more motivated to quit by the perceived vulnerability related to hospitalisation for their COPD. However, only a minority of the self-reports in this study were confirmed by COHb analysis in blood. In the Lung Health Study, participants had mild (symptom free) COPD and this population can be more sensitive to smoking interventions than those with symptomatic disease as in our study, as they probably constitute a 'negative' selection of patients.

In "healthy" smokers the MIS is considered as an effective and easy applicable smoking cessation intervention. The proportion of adults who self-report abstinence at 12 months follow-up is twice that of the reference rate without intervention (13% versus 6%) [9]. With this result the MIS can compete internationally with other smoking intervention abstinence proportions [26].

Although in our population of COPD patients the efficacy of the modified MIS was studied without randomised comparison, the biochemically validated abstinence rate is at least in line with that of comparable programmes. It should be noted that COPD patients who still smoke form a highly selected group because they probably have resisted many urgent advises to quit smoking when they contacted their physician because of their COPD-symptoms [27]. Also, patients in this study can be considered highly motivated because they participate in a time-intensive self-management trial for three years and can be considered to be in the preparation phase of the Stages of Change Model [14].

As the urgency for quitting is much greater for COPD patients than for "healthy" smokers, and there is no indication yet, for which COPD patient a smoking cessation intervention is especially effective, these minimal interventions should be supplemented or intensified in order to increase the success rates.

5.2. Biochemical validation

Self-reports of smoking abstinence in special subgroups with smoking related diseases are considered to be less reliable than in healthy smokers [20,28,29]. As symptomatic smokers feel more pressure to quit smoking, whilst at the same time they are unable to achieve this goal. Additionally, reporting bias will occur when a health worker in a clinical setting inquires after smoking habits [30]. Literature suggests proportions of smoking deceivers varying from 4 to 24% in patients with coronary heart disease, not participating in smoking cessation trials [20,21,31–34]. Little is known about deception in patients with COPD. One major study reported 6.5% smoking deceivers using saliva cotinine in patients with early stage COPD [35]. In another large smoking cessation trial in patients with smoking related disease (80% COPD), about 26% dishonest self-reports were found. We found a much higher percentage of deception in our COPD patients (52%), indicating a much higher degree of social desirable response. COPD patients realise that smoking cessation is the most indisputable option to improve their health status.

5.3. Practice implication

We learned from this experience that in smoking cessation studies in general, but especially when COPD patients

are involved, biochemical assessment of abstinence rates is essential.

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