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What mediates the effect of confrontational counselling on smoking cessation in smokers with COPD?

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ABSTRACT

Objective: Within the framework of a randomized, active treatment controlled trial, we used a mediation analysis to understand the mechanisms by which an intervention that uses confrontation with spirometry for smoking cessation achieves its effects.
Methods: Participants were 228 smokers from the general population with previously undetected chronic obstructive pulmonary disease (COPD), who were detected with airflow limitation by means of spirometry. They received two equally intensive behavioural treatments by a respiratory nurse combined with nortriptyline for smoking cessation: confrontational counselling with spirometry versus conventional health education and promotion (excluding confrontation with spirometry and COPD).
Results: Cotinine validated abstinence rates from smoking at 5 weeks after the target quit date were 43.1% in the confrontational counselling group versus 31.3% in the control group (OR = 1.67, 95%CI = 0.97–2.87). The effect of confrontational counselling on abstinence was independently mediated by the expectation of getting a serious smoking related disease in the future (OR = 1.76, 95%CI = 1.03–3.00), self-exempting beliefs (OR = 0.42, 95%CI = 0.21–0.84), and self-efficacy (OR = 1.38, 95%CI = 1.11–1.73).
Conclusion: We conclude that confrontational counselling increases risk perceptions and self-efficacy, and decreases self-exempting beliefs (risk denial) in smokers with previously undetected COPD. These changes in mediators are associated with a higher likelihood of smoking cessation.
Practice implications: Apart from the intensity, the content of smoking cessation counselling may be an important factor of success. A confrontational counselling approach as we applied may have the potential to alter smoking-related cognitions in such a way that smokers are more successful in quitting. Nurses can be trained to deliver this treatment.

1. Introduction

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease which is characterized by airflow limitation that is not fully reversible [1]. Spirometry is the gold standard for the diagnosis and assessment of the disease [1]. COPD is currently the fifth leading cause of death worldwide [2], and projections for 2020 indicate further increase in global mortality, placing COPD on the third position of lethal diseases [3]. Cigarette smoking is by far the most important risk factor for COPD, and smoking cessation is the single most effective way to reduce the risk of developing COPD and to affect the outcome in patients at all stages of the disease [4,5].

Discussing abnormal test results with smokers has been suggested as a "teachable moment" that may increase motivation to quit smoking, but there is only weak evidence to support such an approach [6]. Various studies have been performed on the efficacy of spirometry as a motivational tool for smoking cessation but their results are inconclusive [7–9]. Findings are often of limited validity because of one or more important biases such as unstandardized counselling intensity, incomparable or uncontrolled use of pharmacological aids for smoking cessation between experimental and control group, or different (or unclear) baseline levels of lung function and motivation to quit smoking [10]. A recent randomized

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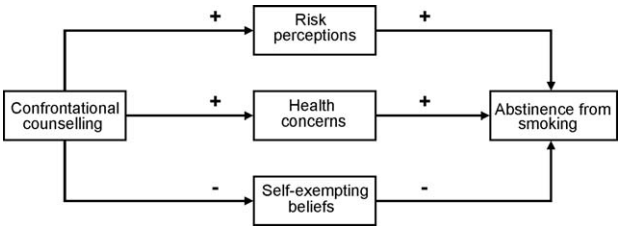

Fig. 1. Hypothetical model of the mediating effects of confrontational counselling.

trial clearly showed a positive effect; telling smokers their "lung age" (based on spirometry) increased the 12-month abstinence rate by 7.2%, but the mechanisms by which the intervention achieved its effect were unclear [11].

We conducted a randomized controlled trial on the efficacy of what we have termed "confrontational counselling" [12]. This is a patient-centred approach which involves confronting smokers with the consequences of their addiction (previously undiagnosed COPD) and which uses specific communication skills to identify and challenge irrational beliefs about smoking. In this article, we will use a mediation analysis to understand how exactly confrontational counselling may effect short-term abstinence from smoking (5 weeks after the target quit date). We hypothesised that confrontational counselling – through labelling with a potentially life-threatening illness status – increases risk perception and health concerns and decreases self-exempting beliefs in smokers, factors which in turn account for abstinence from smoking (see Fig. 1). Mediation analysis allows us to open up the "black box" that conceals the mechanisms of change in our intervention.

2. Methods

We used data from a randomized controlled trial comparing two active smoking cessation treatments in smokers with previously undiagnosed COPD: medium intensity confrontational counselling delivered by a respiratory nurse combined with nortriptyline for smoking cessation (experimental group) with medium intensity health education and promotion delivered by a respiratory nurse combined with nortriptyline for smoking cessation (control group). The third trial arm, "care as usual by the GP", was not included in the current analysis because the goal was to assess mediation of the specific effect of the nurses' counselling. The trial has been approved by the medical ethics committee of Maastricht University and Maastricht University Hospital and registered at the Netherlands Trial Register (ISRCTN 64481813). A detailed description of the trial protocol has been published elsewhere [13].

2.1. Recruitment, eligibility, informed consent, and randomization of participants

Current smokers aged 35–70 years who were interested in quitting smoking were recruited from the general population and from primary care practices in Dutch- and Belgian-Limburg (the region surrounding Maastricht). They were invited to take part in a study on individual counselling and medication for smoking cessation. Neither information about the target condition we were looking for (i.e. COPD) nor the difference in individual counselling between the groups was given to participants before randomization.

Eligibility was assessed during an initial telephone interview. Inclusion criteria were: smoking history of 10 or more pack years (=number of cigarettes smoked per day $\times$ number of years smoking/20); being competent to read and speak Dutch; and reporting at least one of the respiratory symptoms cough, sputum production, or shortness of breath. Exclusion criteria were: evidence of a prior respiratory diagnosis, defined by an affirmative answer to the question "Do you have COPD, chronic bronchitis, asthma or asthmatic bronchitis?" Subjects were also not allowed to have undergone a lung function test (spirometry) during the preceding 12 months. One or more contraindications for using the smoking cessation medication (nortriptyline) were also criteria for exclusion, among others the current use of anti-depressants.

Subjects filled out a baseline questionnaire at home and handed it in during the spirometry visit. Spirometry was performed according to American Thoracic Society (ATS)/European Respiratory Society (ERS) criteria [14,15] using a Vitalograph® 2120 (Vitalograph Ltd., Buckingham, England). Subjects were eligible if they had airflow limitation defined as post-bronchodilator forced expiratory volume in 1 s (FEV_1)/forced vital capacity (FVC) $<70\%$ in combination with post-bronchodilator $FEV_1 \geq 50\%$ of predicted value; i.e. mild or moderate airflow limitation, according to the international GOLD guideline [1]. The results of spirometry were not discussed at that moment. Eligible subjects were contacted by telephone shortly after spirometry and randomized using a database which precluded that any person involved in the study could predict or influence which treatment group the next participant would be allocated to.

2.2. Interventions

Participants from both the experimental group and the control group received medium intensity counselling delivered by a respiratory nurse combined with nortriptyline for smoking cessation. The common basis for the counselling in both groups was the so-called "L-MIS" protocol for the treatment of nicotine and tobacco addiction which had been implemented among all respiratory nurses in the Netherlands in previous years (Table 1, top) [16]. Specific elements of "confrontational counselling" [12] were added to the L-MIS in the experimental group which discriminated the treatment from that in the control group (Table 1, bottom). Confrontational counselling is a supportive, non-judgmental and non-directive approach which involves communication skills and elements of cognitive therapy [12,13]. The confrontational part includes discussing the abnormal results from spirometry and aims at identifying certain cognitions about smoking such as health concerns, risk perception, and self-exempting beliefs. The beliefs were challenged by the respiratory nurse during the counselling sessions. Notes from a smoking cessation diary that smokers from the experimental group used served as input for the counselling.

Participants from both groups received an equal dosage of nortriptyline (Nortrilen®) for smoking cessation. Nortriptyline is a tricyclic anti-depressant which has been shown to be a cheap and effective alternative for the anti-depressant bupropion (Zyban™) [17,18]. The nurse monitored the correct use of the medication and the occurrence of side-effects. In case of unpleasant or severe side-effects, the dosage was reduced or the use of the medication was stopped.

2.3. Measurements

Participants completed a questionnaire at baseline and at follow-up (day 50; approximately 5 weeks after the quit date). Health perceptions included self-designed questions measuring the domains health concerns (3 single items), risk perception (2 single items), and self-exempting beliefs on a 5-point Likert scale (for an overview of these questions see Appendix A). Self-exempting beliefs are statements of risk denial that smokers

Table 1
Components of counselling in experimental group and control group.

General components in both experimental group and control group
FC1 (day 1): 40 min counselling by RN
Assess and discuss smoking characteristics
Assess and increase motivation to quit
Discuss cons of smoking and pros of quitting
Start use of nortriptyline
FC2 (day 8): 40 min counselling by RN
Evaluate use nortriptyline
Assess and increase self-efficacy to quit
Prepare of the TQD
Anticipate on barriers of quitting and withdrawal
TQD: TC (day 14): 5 min counselling by RN
Evaluate the quit attempt
Give advice about quitting and abstaining
FC3 (day 15): 40 min counselling by RN
Evaluate quit attempt
Evaluate use nortriptyline
Give advice about relapse prevention
FC4 (day 22): 40 min counselling by RN
Evaluate quit attempt
Evaluate use nortriptyline
Give advice about relapse prevention
End counselling
Additional components of confrontational counselling in the experimental group only
Incorporated in FC1 + 2
Discuss the results from spirometry
Confront with the consequences of smoking: the diagnosis COPD
Discuss the severity and prognosis of COPD and the benefits of quitting smoking by using the "Fletcher curve" and images of normal and smoker's lungs [51]
Incorporated in FC3 + 4
Reflect on the smoker's thoughts, feelings, and beliefs about COPD
Challenge irrational beliefs about smoking by raising the smoker's consciousness about these beliefs, testing their reality, and by exploring the relationship between beliefs and behaviour
Use of a smoking cessation diary to monitor smoking behaviour and beliefs about smoking

FC = face-to-face counselling session; TC = telephone counselling session; TQD = target quit date; RN = respiratory nurse.

may use to relieve fear and anxiety that may be associated with the detrimental consequences of smoking on health. The 9 items measuring these beliefs were combined into one scale which showed a good reliability (Cronbach's $\alpha = 0.84$). Quality of life was measured with the Short-form 36-item questionnaire (SF-36) [19,20], the Chronic Respiratory Questionnaire self-reported (CRQ) [21,22], and the EuroQol (EQ-5D) [23,24]. Mental health (depression and anxiety) was measured with the Beck Depression Inventory (BDI) [25] and the Hospital Anxiety and Depression Scale (HADS) [26–28].

Motivation to quit was measured by the nurse during FC1 by asking: "How important do you find it to stop smoking on a scale from 0 (not important at all) through 10 (very important)?" Self-efficacy towards smoking cessation was measured by the nurse during FC2, thus before the target quit date, by asking: "How high do you consider your chance to succeed in stopping smoking on a scale from 0 (very low) through 10 (very high)?"

To validate non-smoking, urine was collected from every self-reported non-smoker at the follow-up visit and analysed at the laboratory of the Department of Health Risk Analysis and Toxicology (GRAT) of Maastricht University. The concentration of cotinine in urine was measured by a highly specific radioimmunoassay using monoclonal antibodies [29].

The outcome measure for the current analysis was abstinence from smoking at 5 weeks after the target quit date. A participant was defined as abstinent from smoking if both of the following two conditions were met: urine cotinine level of <50 ng/mL [30] in combination with self-reported non-smoking (i.e. not a single cigarette since the target quit date). All randomized subjects were included in an intention-to-treat analysis and subjects not showing up at the follow-up visit or with a missing value on one of the two criteria for abstinence were regarded as smokers.

2.4. Statistical analyses

Baseline demographic and smoking characteristics as well as mean scores of health perceptions, quality of life, and mental health were compared to assess potential differences between the groups before treatment. Within-subject difference scores were calculated by subtracting the score at baseline from the score at follow-up (except for self-efficacy which was measured at one point of time). Statistical differences in mean difference scores between the groups were tested with *t*-tests. Candidate variables ($p < 0.05$) were entered into the mediation analysis.

We used an approach for the mediation analysis which was based on a method described by Baron and Kenny [31]. They define a mediator as a variable that "accounts for the relation between the predictor and the criterion". In our case, the mediator would account for the relation between intervention and abstinence from smoking. The mediation analysis comprised four steps for each candidate mediator using regression analysis. In step 1, we regressed abstinence (1 = abstinent from smoking, 0 = not abstinent) on intervention (1 = experimental group, 0 = control group) in a simple logistic regression. In step 2, we regressed the candidate mediator on intervention in a simple linear regression. In step 3, we regressed abstinence on the candidate mediator, again in a simple logistic regression. In step 4, we regressed abstinence on intervention and the candidate mediator in a multiple logistic regression model. We defined a mediator as a variable that was significantly (at $p < 0.1$) associated with the intervention in step 2 and with the outcome in steps 3 and 4.

Finally, we built multiple logistic regression models with all mediators verified by the previous mediation analysis. In model 1, we regressed abstinence on intervention. In model 2, we added all verified mediators. We then adjusted model 2 for all important baseline characteristics.

We did not impute missing values. The percentage missing values on the measures of health perceptions, quality of life, and mental health was on average 14% in the experimental group and 15% in the control group. In ancillary analyses, we ran all logistic regression models with missing values imputed by the series mean.

3. Results

A total of 116 smokers with previously undetected COPD were randomly allocated to the experimental group, 112 to the control group. The baseline characteristics are shown in Table 2. Mean baseline scores on all measures of health perceptions, quality of life, and mental health were comparable between the groups (not shown in the table), except for the subscale "physical role limitations" on the SF-36; the mean score in the experimental group was substantially higher ($M = 84.1$, S.D. = 30.0) than in the control group ($M = 73.4$, 39.1). The abstinence from smoking 5 weeks after the target quit date were 43.1% ($N = 50$ of 116) in the experimental group and 31.3% ($N = 35$ of 112) in the control group (OR = 1.67, 95%CI = 0.97–2.87, $p = 0.065$, two-tailed). This association remained unchanged when adjusting for the variable "physical role limitations" on the SF-36.

Table 2
Demographic and smoking characteristics and lung function at baseline.

	Experimental group ($N = 116$)	Control group ($N = 112$)
Years of age, mean (S.D.)	53.8 (7.0)	54.9 (8.0)
Male sex, *N* (%)	71 (61.2)	74 (66.1)
Educational background, median (range) [range from 1 (lowest category) through 7 (highest)]	4 (1–7)	4 (1–7)
Pack years[a], mean (S.D.)	44.1 (18.3)	44.2 (19.1)
FTND[b], mean (S.D.) [range from 0 (lowest level of nicotine dependence) through 10 (highest)]	4.6 (1.5)	4.5 (1.5)
Motivation to quit: "How important do you find it to stop smoking?" mean (S.D.) [range from 0 (not important at all) through 10 (very important)]	9.0 (1.2)	8.8 (1.2)
Number of previous quit attempts, median (range)	3 (1–25)	3 (1–50)
FEV1 post-bd. %pred.[c], mean (S.D.)	80.5 (14.7)	83.7 (16.8)
FVC post-bd. %pred.[c], mean (S.D.)	103.9 (14.9)	107.6 (17.8)
FEV1/FVC post-bd., mean (S.D.)	62.5 (5.9)	63.0 (6.1)

[a] 1 pack year = number of cigarettes smoked per day × number of years smoking/20.
[b] Fagerström Test for Nicotine Dependence.
[c] Post-bronchodilator Forced Expiratory Volume in 1 s and post-bronchodilator Forced Vital Capacity, as percentage of predicted value.

Table 3
Mean within-subject differences from baseline to follow-up between experimental and control group for measures of health perceptions, quality of life, and mental health.

Variables	Experimental group: mean difference between baseline and follow-up	Control group: mean difference between baseline and follow-up	Differences in means between experimental and control group[a]	95%CI around mean difference, lower–upper bound	Level of statistical significance (p)
Health concerns 1: "Do you think you have a disease or disorder at this moment which is caused by smoking?" [range from 1 (certainly not) through 5 (certainly yes)]	0.61	−0.20	0.81	0.45 to 1.16	<0.001
Health concerns 2: "How worried are you to get a disease or disorder which is caused by smoking (such as a serious heart or lung disease)?" [range from 1 (not worried at all) through 5 (very worried)]	0.16	−0.05	0.21	−0.10 to 0.53	n.s.
Health concerns 3: "How important is it to you to reduce your risk of getting a disease or disorder which is caused by smoking?" [range from 1 (very unimportant) through 5 (very important)]	0.13	−0.21	0.35	0.04 to 0.66	0.029
Risk perception 1: "How high do you estimate your risk of getting a serious disease at a later age (when you are elderly) when you do not stop smoking?" [range from 1 (very low) through 5 (very high)]	0.13	−0.20	0.32	0.09 to 0.56	0.008
Risk perception 2: "How high do you estimate your risk of getting a serious disease within the next 10 years when you do not stop smoking?" [range from 1 (very low) through 5 (very high)]	0.19	−0.14	0.34	0.11 to 0.55	0.003
Self-exempting beliefs [range from 1 (very low self-exempting beliefs) through 5 (very high)]	−0.22	0.09	−0.31	−0.48 to −0.14	<0.001
SF-36[b] subscales [range from 0 (lowest quality of life) to 100 (highest)]					
Physical functioning	2.98	4.49	−1.52	−6.08 to 3.05	n.s.
Social functioning	−0.85	−0.90	0.05	−6.43 to 6.53	n.s.
Physical role limitations	2.32	6.18	−3.86	−13.19 to 5.47	n.s.
Emotional role limitations	−2.38	−6.25	3.87	−6.35 to 14.09	n.s.
Mental health	0.44	1.90	−1.46	−6.20 to 3.27	n.s.
Vitality	3.54	5.67	−2.13	−7.00 to 2.74	n.s.
Bodily pain	7.54	6.54	1.00	−5.46 to 7.46	n.s.
General health perceptions	7.40	10.99	−3.59	−8.07 to 0.89	n.s.
CRQ[c] subscales [range from 1 (lowest quality of life) through 7 (highest)]					
Fatigue	0.32	0.47	−0.15	−0.49 to 0.18	n.s.
Emotional function	0.51	0.45	0.06	−0.21 to 0.33	n.s.
Mastery	0.22	0.27	−0.05	−0.30 to 0.19	n.s.
EuroQol-5D health status [range from 0 (lowest health status) through 1 (highest)]	0.05	0.05	0.00	−0.04 to 0.05	n.s.
BDI[d] [range from 0 (lowest degree of depression) through 63 (highest)]	−2.11	−1.72	−0.39	−2.54 to 1.76	n.s.
HADS[e] subscales [range from 0 (lowest degree of anxiety/depression) through 21 (highest)]					
Anxiety	−1.26	−1.05	−0.20	−1.12 to 0.71	n.s.
Depression	−1.25	−0.98	−0.27	−1.17 to 0.64	n.s.

[a] A positive mean difference indicates an increase of the measure from baseline to follow-up in the experimental group relative to the control group, and a negative mean difference a relative decrease.
[b] Short-form 36-item questionnaire.
[c] Chronic Respiratory Disease Questionnaire self-reported.
[d] Beck Depression Inventory.
[e] Hospital Anxiety and Depression Scale mean; n.s. = not statistical significant at $p = 0.1$.

Table 4
Multiple logistic regression modelling the odds of abstinence from smoking for adjusted mediators.

Independent variables included (β)	Exp(β)	95% confidence interval around Exp(β); lower–upper	Level of statistical significance (p)
Model 1			
Intervention (control group = reference)	1.67	0.97–2.87	0.065
Model 2			
Intervention (control group = reference)	0.87	0.43–1.73	0.687
Risk perceptions 2[a]	1.43	0.94–2.19	0.098
Self-exempting beliefs[b]	0.46	0.25–0.85	0.013
Self-efficacy[c]	1.35	1.12–1.63	0.002
Model 2 adjusted for baseline characteristics[d]			
Intervention (control group = reference)	0.92	0.42–2.00	0.836
Risk perceptions 2[a]	1.76	1.03–3.00	0.037
Self-exempting beliefs[b]	0.42	0.21–0.84	0.014
Self-efficacy[c]	1.38	1.11–1.73	0.004

[a] "How high do you estimate your risk of getting a serious disease within the next 10 years when you do not stop smoking?"
[b] Range from 1 (very low self-exempting beliefs) through 5 (very high).
[c] "How high do you consider your chance to succeed in stopping smoking?" [range from 0 (very low chance) through 10 (very high)].
[d] Adjusted for age, sex, educational background, nicotine dependence (FTND), motivation to quit, number of previous quit attempts, airflow limitation (FEV1 post-bd. %pred.), and the subscale "physical role limitations" of the SF-36.

3.1. Effect of the interventions on health perceptions, quality of life, mental health, and self-efficacy

Table 3 shows the mean difference scores between baseline and follow-up for the experimental group and the control group, and the difference in means between the groups (experimental group minus control group). A positive mean difference indicates an increase of the measure from baseline to follow-up in the experimental group relative to the control group, and a negative mean difference a relative decrease in the experimental group. Four measures of health perceptions were significantly increased in the experimental group: health concerns 1 (mean difference = 0.81), health concerns 3 (0.35), risk perception 1 (0.32), and risk perception 2 (0.34). A fifth measure, self-exempting beliefs, was significantly decreased in the experimental group (−0.31). There were no differences between the groups with regard to measures of quality of life and mental health. Self-efficacy towards smoking cessation, which was measured at one point in time (at FC2, before the quit date), and is therefore not shown in Table 3, was significantly higher in the experimental group (M = 8.4, S.D. = 2.0) than in the control group (M = 7.7, S.D. = 1.7, p = 0.004).

3.2. Mediators of the association between intervention and abstinence

The five measures of health perceptions and the variable self-efficacy were entered into the mediation/moderation analysis. In step 1, the intervention was associated with abstinence from smoking: the odds of abstinence was 67% higher in the experimental group than in the control group (OR = 1.67, 95%CI = 0.97–2.87, p = 0.065). Three measures were verified as mediators of the association between intervention and abstinence in steps 2–4: risk perception 2, self-exempting beliefs, and self-efficacy. In step 4, the odds for being abstinent from smoking adjusted for the effects of variable intervention per one point increase on the scale of the mediator were: OR = 1.40 (95%CI = 0.96–2.04, p = 0.081) for risk perception 2, OR = 0.58 (95%CI = 0.34–1.01, p = 0.052) for self-exempting beliefs, and OR = 1.23 (95%CI = 1.03–1.46, p = 0.020) for self-efficacy.

Running all models with missing values replaced by the series mean resulted in similar results except for the variable risk perception 2 that failed to be a mediator.

We also performed a moderation analysis [31] but did not find a variable that affected the direction and/or strength of the relation between intervention and abstinence.

3.3. Adjusted effects of mediators of the association between intervention and abstinence

The three verified mediators were entered into a multiple logistic regression, modelling abstinence from smoking (Table 4). When the mediators were entered into model 2, the effect of the intervention was eliminated (OR = 0.87, p = 0.687). This does not mean that the intervention was ineffective, but rather that "perfect mediation" [31] occurred: risk perception, self-exempting beliefs, and self-efficacy accounted for the effect of confrontational counselling on abstinence from smoking. The mediators had an independent effect on abstinence after adjustment for potentially confounding baseline characteristics age, sex, educational background, nicotine dependence, motivation to quit, number of previous quit attempts, airflow limitation, the subscale "physical role limitations" of the SF-36, and the other mediators. One point increase on the scale for risk perceptions 2 increased the odds of smoking abstinence by 76%, and one point increase on the scale for self-efficacy increased the odds by 38%. One point increase on the scale for self-exempting beliefs decreased the odds of smoking abstinence by 58%.

When excluding the variable risk perception 2 from the adjusted model, the odds of smoking abstinence increased by 38% for one point increase on the scale for self-efficacy and decreased by 63% for one point increase on the scale for self-exempting beliefs.

4. Discussion and conclusion

4.1. Discussion

We found three factors that mediated the effect of confrontational counselling on smoking cessation: self-efficacy towards successful smoking cessation, the expectation of getting a serious smoking-related disease within the next 10 years, and self-exempting beliefs towards smoking. All three mediators were independently associated with abstinence and accounted for the effect of the intervention.

The current evidence on the efficacy of spirometry as a motivational tool for smoking cessation is inconclusive [7,8]. In one primary care study, for example, current smokers motivated to quit were randomized to undergo spirometry followed by brief advice for smoking cessation or to brief advice alone [32]. The proportion of non-smokers was about 5% higher in the spirometry group at 6, 12, and 24 months follow-up (not statistically

significant at $p = 0.05$). The study had several limitations including lack of information of actual use of smoking cessation medication in participants as well as lack of a valid measure of abstinence that meets international guidelines [30,33]. A different and more valid primary care study clearly showed that telling smokers their "lung age" (based on spirometry) increased the 12-month abstinence rate by 7.2%. However, the mechanisms by which the intervention achieved its effect were unclear [11]. The present study may explain some of the underlying mechanisms.

In contrast to previous studies, our study is the first to attempt a mediation analysis of the treatment effects of confronting smokers with the results from spirometry and the diagnosis COPD. The design of our study accounts for potential biases by standardizing counselling intensity, using pharmacological aids for smoking cessation, and by randomizing smokers into two groups with comparable baseline levels of lung function and motivation to quit smoking. This design enables us to isolate the effects of labelling with disease and to unseal the "black box" of mechanisms of change which may account for the effects of the intervention.

There is sufficient evidence from the field of health education and promotion that fear arousal on its own is not a good motivator but that the a combination of factors is more likely to result in change of health behaviour: high levels of perceived severity, susceptibility, outcome expectancy, and self-efficacy [34–36]. Our experimental intervention, confrontational counselling, aimed to target theses factors. The severity of the disease COPD was stressed by discussing the prognosis in case of continued smoking (deterioration of lung function resulting in decreased quality of life and finally premature death). The smoker's susceptibility was demonstrated by confronting the smoker with the results from spirometry and his/her respiratory symptoms. Our results show that confrontational counselling increased risk perception, which subsequently led to smoking cessation. Outcome expectancy and self-efficacy were triggered by making the smoker understand that there is an effective and feasible therapy for the disease: smoking cessation, aided by behavioural counselling and smoking cessation medication. Our results show that confrontational counselling increased self-efficacy, which again led to smoking cessation.

Self-exempting beliefs are common among smokers and these beliefs may interfere with smoking cessation [37–42]. Smokers have these beliefs in order to reduce cognitive dissonance and alleviate anxiety [43]. We challenged these beliefs by confronting smokers with objective evidence of the health consequences of their own smoking behaviour (COPD measured by spirometry) and by using specific communication skills deriving from cognitive therapy. The results from the mediation analysis show that confrontational counselling decreased self-exempting beliefs and that this led to smoking cessation.

The p-value resulting from statistical testing of the difference in observed effect between the intervention groups was $p = 0.064$. A p-value is often misinterpreted as an indicator of clinical significance and misused when treated in a dichotomous manner as either "significant" ($p < 0.05$) or "not significant" ($p > 0.05$) [44–46]. The p-value does not provide information about the magnitude of the effect and the precision of the estimate, whereas confidence intervals do [44,47,48]. The estimate of the effect size (OR = 1.67) and the 95% confidence interval around this estimate (0.97–2.87) show that the true population estimate is likely to be higher than 1. Thus, the p-value would probably have reached a level below 0.05 when the sample size (and therefore the power) would have been bigger. It should also be noted that the observed effect size is large for a trial comparing two active treatments for smoking cessation which differ only in type of counselling and of clinical importance (67% increase of the odds of abstinence from smoking in the experimental group compared with the control group). It should also be noted that the question if confrontational counselling is effective will be determined in a different analysis, as the efficacy of smoking cessation interventions should be based on the 12 month prolonged abstinence rates [33]. This mediation analysis concerns a different topic; understanding *how* confrontational counselling works.

The ancillary analyses with missing values replaced by the series mean resulted in similar results. However, the variable risk perception 2 failed to be a significant mediator. This may be an indication that this factor is a less strong mediator compared with the mediators self-exempting beliefs and self-efficacy. Removing the variable risk perception 2 from the final model only slightly changed the estimated odds ratios of the other two mediators, indicating the robustness of the model.

Respiratory nurses from the experimental group received a group training and regular supervision in confrontational counselling. The effects of the intervention may be partly due to increased non-specific counselling skills of these nurses compared to nurses in the control group. One might therefore ascribe the combination of specific skills in confrontational counselling with training effects to the efficacy of the intervention.

The informed consent design we used raises some ethical issues as participants were not fully informed about the real purpose of the study, which is to detect and confront smokers with COPD. Participants randomized to the control group were not informed about their results of spirometry during the intervention but only after completing the follow-up period. This procedure was approved by a medical ethics committee and was regarded as ethical because smokers participating in this trial would probably not have been diagnosed outside the trial setting early due to the problem of underdiagnosis of COPD in primary care. The second reason is that all smokers from this trial received the most effective therapy for mild to moderate COPD, which is smoking cessation treatment.

Another important issue in the discussion about early detection of airflow limitation in smokers is the suggested counterproductive effect of communicating negative tests results (i.e. normal lung function) to smokers [49]. Smokers with normal lung function might use their result from spirometry as a message that they are not susceptible to the effects of smoking and as an argument to continue smoking. If such an effect occurs, the abstinence rates of smokers diagnosed with normal lung function should be very low. In a recent large-scaled prospective study on the association between airway obstruction and smoking cessation in Poland, 12-month abstinence rates from smoking were only slightly lower in subjects with normal lung function (12%) than in subjects with abnormal lung function [50]. To test whether a counterproductive effect occurred in our sample, we randomized another 59 smokers with normal lung function (formally defined as GOLD 0 COPD) to the three intervention groups. Among these, 22 subjects (37%) were abstinent from smoking at follow-up. This is about the same success rate as within the subgroup with GOLD 1 COPD (36%; $N = 46/128$) and within the subgroup with GOLD 2 COPD (39%; $N = 61/100$). These findings do not provide evidence of counterproductive effects of reporting normal lung function in our sample. This might be due to the high initial motivation of all trial participants to stop smoking. This might also be due to the case-finding approach we chose. Therefore, our results may not be transferable to a large-scaled screening approach.

4.2. Conclusion

We conclude that confrontational counselling increases risk perceptions and self-efficacy, and decreases self-exempting beliefs in smokers with previously undetected COPD. These changes in mediators are associated with a higher likelihood of smoking cessation.

4.3. Practice implications

The best treatment for smoking cessation is a combination of evidence-based pharmacotherapy in combination with counselling. But with the currently available treatments, the majority of smokers fails to quit or relapses into smoking. It is therefore important to optimize available treatments and to develop new ones. The dose–response relationship between counselling intensity and treatment success has been well established. In order to further increase the efficacy of counselling we must understand the psycho-social mechanisms that underlie changes in smoking behaviour and how these can be targeted. The results from our study show that risk perceptions, self-exempting beliefs, and self-efficacy are three important cognitions that may be associated with treatment success. Our confrontation counselling approach, delivered by trained respiratory nurses, altered these cognitions in such a way that smokers were more successful in quitting smoking in the short term.

Competing interest

The authors have no competing interests.

Disclosure statement

I (Daniel Kotz) confirm all patient/personal identifiers have been removed or disguised so the patient/person(s) described are not identifiable and cannot be identified through the details of the story.

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Appendix A

Questions measuring "health concerns".

Health concerns 1: "Do you think you have a disease or disorder at this moment which is caused by smoking?"

certainly not	probably not	**indecisive/don't know**	probably yes	certainly yes
☐	☐	☐	☐	☐

Health concerns 2: "How worried are you to get a disease or disorder which is caused by smoking (such as a serious heart or lung disease)?

not worried at all	not worried	**indecisive/don't know**	worried	very worried
☐	☐	☐	☐	☐

Health concerns 3: "How important is it to you to reduce your risk of getting a disease or disorder which is caused by smoking?

very unimportant	unimportant	**indecisive/don't know**	important	very important
☐	☐	☐	☐	☐
Scoring:				
1	**2**	**3**	**4**	**5**

Questions measuring "risk perceptions".

Risk perception 1: "How high do you estimate your risk of getting a serious disease at a later age (when you are elderly) when you do not stop smoking?

very low	low	**indecisive/don't know**	high	very high
☐	☐	☐	☐	☐

Risk perception 2: "How high do you estimate your risk of getting a serious disease within the next 10 years when you do not stop smoking?

very low	low	**indecisive/don't know**	high	very high
☐	☐	☐	☐	☐
Scoring:				
1	**2**	**3**	**4**	**5**

Questions measuring "self-exempting beliefs".

Appendix A (*Continued*)

In how far do you agree or disagree with the following statements?

	totally disagree	disagree	indecisive/ don't know	agree	totally agree
1. The scientific evidence that smoking is harmful is not convincing.	☐	☐	☐	☐	☐
2. Smoking is possibly not very harmful because many smokers live long.	☐	☐	☐	☐	☐
3. I would need to smoke far more to jeopardize my health.	☐	☐	☐	☐	☐
4. The harmful effects can be cleared away, e.g. by eating healthy or exercising regularly.	☐	☐	☐	☐	☐
5. My body is by nature not susceptible to the harmful effects of smoking.	☐	☐	☐	☐	☐
6. Smoking is not more harmful than many other things people do.	☐	☐	☐	☐	☐
7. Everything causes cancer nowadays.	☐	☐	☐	☐	☐
8. Smoking light cigarettes is not harmful.	☐	☐	☐	☐	☐
9. Only heavy smoking (20 cigarettes a day or more) is harmful.	☐	☐	☐	☐	☐
Scoring:	**1**	**2**	**3**	**4**	**5**

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