ORIGINAL ARTICLE

Risk of hospital admission for COPD following smoking cessation and reduction: a Danish population study

N S Godtfredsen, J Vestbo, M Osler, E Prescott

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See end of article for authors' affiliations

Correspondence to: Dr N S Godtfredsen, Høyrups Allé 28a, DK-2900 Hellerup, Denmark; ng@ipm.hosp.dk

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Background: Little is known about the effects of changes in smoking habits on the subsequent risk of chronic obstructive pulmonary disease (COPD). The aim of this study was to investigate the relationship between smoking cessation and reduction and admission to hospital for COPD in a general population sample.

Methods: A total of 19 709 participants from three prospective population studies in Copenhagen were followed with record linkage for date of first hospital admission for COPD until 1998 (mean follow up 14 years). Heavy smokers (≥15 cigarettes/day) who reduced their tobacco consumption by at least 50% between the two initial examinations without quitting and smokers who stopped smoking during this time were compared with continuous heavy smokers using a Cox proportional hazards model.

Results: During the follow up period 1260 subjects (741 men and 519 women) were admitted to hospital for COPD. After multivariate adjustment, quitting smoking was associated with a significant reduction in the risk of hospital admission. The relative hazard (HR) was 0.57 (95% confidence interval (CI) 0.33 to 0.99). Those who reduced smoking did not show a significantly lower risk of hospitalisation than continuing heavy smokers (HR 0.93 (95% CI 0.73 to1.18)). Exclusion of events during the first 5 study years, detailed adjustment for lung function, or restriction of analyses to participants with impaired pulmonary function did not reverse the observed trend.

Conclusions: Self-reported smoking cessation is associated with a reduction in the risk of COPD morbidity of approximately 40%; the benefit of smoking reduction is questionable.

Chronic obstructive pulmonary disease (COPD) is the fourth leading cause of death and chronic morbidity in the world.[1] It is therefore a major public health problem and the burden on society from this disease is predicted to increase in the forthcoming decades.[2] One of the most important risk factors for COPD is tobacco smoke, and abstaining from this substance is clearly related to lower prevalence of tobacco related diseases such as COPD.[3] Stagnation in the decline in smoking cessation rates and a corresponding increase in the uptake of smoking among adolescents seen in many parts of the world[4] has led tobacco researchers to seek alternative strategies to total tobacco abstinence for reducing the harmful effects of smoking. One component of the "harm reduction" approach is a reduction in the daily number of cigarettes in smokers who are unable or unmotivated to quit. However, this is a highly controversial strategy because preliminary results are conflicting[5 6] and it has recently been concluded that there is still a lack of evidence for potential health benefits of harm reduction.[7] In particular, the long term consequences on the pulmonary, cardiovascular, and immunological systems are virtually unknown.

In contrast, the beneficial effect of smoking cessation on the course of the decline in forced expiratory volume in 1 second (FEV_1) and prognosis in COPD is well established,[8] although hospital data are limited. Lung function, respiratory symptoms, and sex have all been shown to be associated with hospital admissions for COPD,[9–11] but little is known about the possibilities of preventing COPD admissions through, for example, smoking intervention. To our knowledge no other population based studies have addressed the issue of smoking reduction or cessation directly with respect to hospital admission. The aim of this study was to examine the effects of changes in smoking on the risk of first hospital admission for COPD using data from a general population sample and a nationwide hospital register.

METHODS

Population, hospitalisation and follow up

Pooled data were used from three prospective population studies conducted in and around Copenhagen: the Copenhagen City Heart Study (CCHS), the Glostrup Population Studies (GPS) and the Copenhagen Male Study (CMS). The CCHS comprised 18 039 individuals, the CMS 5241, and the GPS, from which three birth cohorts (1897, 1914, 1936) and the MONICA I were used, consisted of 7582 individuals. All studies have been described in detail previously.[12–14] Initial examinations took place between 1964 and 1993 with the majority of participants being recruited in the 1970s and re-examinations occurring at intervals of 5–10 years. All examinations included a self-administered questionnaire containing health and lifestyle related items, as well as a detailed physical examination. The mean response rate was 77% (range 69–88%). The present study comprises 19 709 subjects (11 148 men and 8561 women)—10 984 from the CCHS, 4003 from the CMS, and 4722 from the GPS—who participated in and provided adequate information on smoking habits at two consecutive investigations approximately 5 years apart. This implied for nearly all participants the use of baseline information from the first investigation in the mid 1970s and assessment of risk factor changes at the second investigation in the early 1980s. The study population is outlined in table 1.

Information on time of hospital admission and diagnoses on discharge in the period from the second survey to 31 December 1997 was obtained from the National Patient Register administered by the National Board of Health. Diagnoses were classified according to the International Classification of Diseases (ICD), 8th revision until 31 December 1993 and 10th revision for the remaining period. Only the main diagnosis on discharge was used. For hospital admissions caused by COPD this corresponded to the ICD-8 and ICD-10

Table 1 Overview of the study population (n=19 709)

Cohort of origin	Year of examinations	No of men	No of women	Age at examinations	No of hospital admissions†
CCHS	1976/83	4775	6212	20–93	850
GPS, 1897 cohort	1967/77	84	112	70–80	246
GPS, 1914 cohort	1964/74	331	284	50–60	12
GPS, 1936 cohort	1976/81	456	500	40–45	63
MONICA I*	1981/88	1499	1453	30–65	22
CMS	1970/76	4003	–	39–65	67
Total	1964–88	11148	8561	20–93	1260

*The MONICA project is an international study conducted by the World Health Organisation to monitor trends in, and determinants of, mortality from cardiovascular disease. †Participants with a register diagnosis of COPD before enrolment (n=149) were excluded.

diagnosis codes 490–492 and J40-J44, respectively. Participants with known hospital admission for COPD before the beginning of the follow up period (n=149) were excluded. The only possible loss to follow up was through emigration (<0.4%). The mean duration of follow up was 14.4 years.

Assessment of smoking, changes in smoking habits, and covariates

Smoking status and changes in smoking habits in this study are based on self-reports. At each examination the participants were asked whether or not they smoked and, if affirmative, the amount, duration, inhalation, and preferred type of tobacco (cigarettes, cheroots, cigars, pipe, and/or mixed). Ex-smokers were asked about the duration of smoking and years since quitting. Tobacco consumption was calculated by equating a cigarette to 1 g tobacco, a cheroot to 3 g, and a cigar to 5 g. In order to measure a substantial reduction in tobacco consumption, only those who were heavy smokers (≥15 g/day) at first examination were considered. Smoking reduction was defined as self-reported smoking of 15 g tobacco or more at first examination and a reported decrease of at least 50% without quitting at the second examination. This definition of smoking reduction is similar to that used in clinical studies of smoking reduction.[5 15] The study population was then divided into the following smoking categories: reducers, new ex-smokers (quitters), sustained never smokers, sustained ex-smokers, sustained light smokers, and sustained heavy smokers (reference group). Furthermore, the quitters were stratified according to whether their tobacco consumption at first examination was 1–14 g/day or 15+ g/day. The few individuals who did not meet the criteria for the above mentioned changes in smoking habits—for example, reduced by less than 50% or ex-smokers resuming smoking—were placed in the category to which they belonged at the second examination.

For baseline comparison of the smoking groups of primary interest, cumulative tobacco exposure calculated as pack-years (years of smoking × daily number of cigarettes/20), inhalation habits, preferred type of tobacco among those currently smoking at first examination, and presence of chronic mucus hypersecretion was analysed. Chronic mucus hypersecretion was defined as reporting cough and phlegm for at least three consecutive months for 2 years or more. Comparisons of baseline lung function measured as percentage predicted forced expiratory volume in 1 second (FEV_1) in 7823 individuals from the CCHS were also made.[11] There were no differences between the reducers and the continuous heavy smokers with respect to such baseline covariates as body mass index, educational level, marital status, and self-reported asthma.

Biochemical verification of smoking status

Carbon monoxide (CO) in expired air was measured in CCHS III (1991–3) in approximately 10 000 individuals and blood cotinine levels were measured in 3300 men in the CMS in 1985. The cross sectional values of CO were used for validation of smoking cessation and smoking reduction.

Statistical analysis

For comparison of dichotomous and continuous variables the χ^2 statistic with two tailed p values and two sample *t* tests were applied. Cox's proportional hazards regression model[16] was used to calculate the hazard ratios for the different categories of smoking associated with COPD hospitalisation with sustained heavy smokers as the reference group. Age was chosen as the underlying time scale and the model allowed for delayed entry—that is, participants entered analysis at their age at the second examination. All analyses were stratified by sex, thus assuming equal effects of coefficients for the variables of interest but allowing baseline hazards to differ with sex. In the final multivariable model the following potential confounders were included: age, sex, cohort of origin, type of tobacco (cigarettes; cigars, cheroots or pipe; mixed), inhalation habits (yes/no), and duration of smoking (years). Duration of smoking was chosen instead of pack-years because the primary variable of interest—the smoking categories—was constructed using the amount smoked, which was also a component in the calculation of pack-years. Spirometric tests were not performed on all participants, but additional analyses on the subset of the population (the CCHS) that included these values were carried out.

Possible effect modification by any of the covariates and our primary independent variable of interest (the six smoking categories described above) was examined by adding an interaction term between them using a likelihood ratio test. The proportional hazards assumption was tested in two different ways: the standard graphical check based on the log of the cumulative hazard and through a formal test of proportionality based on Schoenfeld residuals. Both methods revealed that there were proportional hazards between the smoking groups, and the model was considered to be appropriate. Results of the survival analyses are presented as hazard ratios (HR) and 95% confidence intervals (CI). The analyses were made using Stata statistical software, release 7.0.[17]

RESULTS

The analyses were based on 11 148 men and 8561 women (table 1). During follow up 741 men (6.6%) and 519 women (6.0%) were admitted to hospital for COPD. Between the first and second surveys 12.1% (n=1454) of the baseline current smokers stopped smoking and 10.7% (n=832) of the initial heavy smokers reduced their tobacco intake by at least 50% without quitting smoking (table 2). The amount reduced was from a mean of 22.5 g tobacco per day at first investigation to a mean of 8.7 g/day at the second investigation. Comparison of reducers and continuous heavy smokers showed that reducers had higher mean daily tobacco consumption at baseline than the heavy smokers. However, the heavy smokers had significantly more pack-years of smoking at baseline. Furthermore, the heavy smokers were more likely to inhale the smoke, to smoke cigarettes only (as opposed to a mixed tobacco intake), and to report suffering from chronic mucus

Table 2 Characteristics at first examination according to smoking status at second examination for the pooled study population

	Never smokers (n=4006)	Ex-smokers (n=2880)	Quitters (n=1454)	Light smokers (n=3189)	Reducers (n=832)	Heavy smokers (n=7348)	p value*
Sex (% men)	33.7	65.9	61.2	47.5	72.1	66.6	0.001
Age (years)	49.5 (11.9)	51.5 (10.4)	50.5 (11.0)	50.1 (10.7)	51.3 (10.3)	48.0 (9.4)	<0.001†
Tobacco consumption (g/day)	–	–	14.5 (12.2)	9.0 (4.2)	22.4 (12.2)	19.9 (8.6)	<0.001†
Inhalers (%)	–	–	60.4	74.0	70.6	78.0	<0.001
Type of tobacco smoked							
Cigarettes (%)	–	–	51.1	73.9	33.2	57.9	<0.001
Cigars, cheroots, pipe, mixed (%)	–	–	48.9	26.1	66.8	42.1	<0.001
Chronic mucus (%)	3.9	5.9	10.4	9.1	10.4	14.1	0.004
Pack years of smoking	–	–	19.4 (18.1)	14.0 (8.4)	27.0 (20.9)	30.9 (19.3)	<0.001†
FEV_1 (% predicted)‡	89.6 (16.3)	87.9 (18.9)	81.8 (19.9)	87.4 (16.5)	80.9 (16.3)	82.0 (16.7)	0.31
Expired CO (ppm)§	2.2 (1.3)	2.4 (2.2)	4.0 (5.9)	8.4 (7.7)	8.7 (8.1)	13.2 (10.0)	<0.001†
Admission for COPD, n (%)	71 (1.8)	106 (3.7)	83 (5.7)	228 (7.1)	82 (9.8)	690 (9.4)	

Results are presented as absolute values, percentages, or mean (SD) values.
*p values represent differences between the reducers and the continuous heavy smokers.
†Two sample *t* tests. All other are χ^2 tests with two tailed p values.
‡Results only from the CCHS, 1st survey 1976–8, n=7823.
§Results only from the CCHS, 3rd survey 1991–3, n=7016.

hypersecretion. Results from the CCHS showed that there was no difference in baseline FEV_1 % predicted between quitters, reducers, and continuous heavy smokers. Levels of exhaled CO were significantly greater in individuals who continued to smoke heavily than in those who reduced.

Multivariate Cox regression analysis revealed that there was a dose-response relationship between smoking behaviour and hospital admissions for COPD (table 3). The adjusted HR for never smokers was 0.14 (95% CI 0.08 to 0.25) compared with continuous heavy smokers. Participants who were ex-smokers at both examinations reduced the risk of being admitted to hospital by approximately 70% compared with the reference group (HR 0.30, 95% CI 0.18 to 0.50). Subjects who stopped smoking between the first and second examination (quitters) had an HR of 0.57 (95% CI 0.33 to 0.99) for the group as a whole, with a greater reduction in risk for those who were light smokers at first examination and a significantly reduced risk in heavy smokers who ceased (table 3). Continuous light smokers had an HR of 0.59 (95% CI 0.51 to 0.70), whereas there was no significant decrease in the risk of hospitalisation for COPD among participants who reduced their smoking (HR 0.93, 95% CI 0.73 to 1.18). Independent of smoking habits, inhaling the smoke and smoking cigarettes (compared with smoking other types of tobacco) were associated with an increased risk of hospital admission for COPD. To avoid biased risk estimates caused by changes in smoking habits due to preclinical or existing disease—that is, reversed causality—a similar regression analysis omitting the first 5 years after the second examination was performed. This lowered the estimates for quitters and reducers, as shown in table 3, indicating a likelihood of an induction period or "ill quitter" effect. However, the association between reduced smoking and hospital admission was still not significant. There were no signs of an interaction between smoking behaviour and inhalation habits or preferred type of tobacco on the risk of hospital admissions for COPD.

In the CCHS we performed Cox's regression analysis with adjustment for lung function expressed as tertiles of FEV_1 %

Table 3 Risk of admission to hospital for COPD by changes in smoking habits, pattern of inhalation, and type of tobacco smoked

		Total study population (n=19709)		Omitting first 5 years
Smoking habits	No of events	Crude hazard ratio (95% CI)*	Adjusted hazard ratio (95% CI)†	Adjusted hazard ratios (95% CI)‡
Smoking category				
Heavy smokers	690	1	1	1
Never smokers	71	0.13 (0.10 to 0.17)	0.14 (0.08 to 0.25)	0.10 (0.08 to 0.14)
Sustained ex-smokers	106	0.25 (0.20 to 0.31)	0.30 (0.18 to 0.50)	0.20 (0.15 to 0.26)
Quitters (<15 g/day)§	42	0.36 (0.26 to 0.49)	0.40 (29 to 0.55)	0.28 (0.18 to 0.44)
Quitters (≥15 g/day)§	41	0.58 (0.43 to 0.80)	0.66 (0.47 to 0.93)	0.52 (0.33 to 0.80)
Light smokers	228	0.58 (0.49 to 0.67)	0.59 (0.51 to 0.70)	0.55 (0.46 to 0.66)
Reducers	82	0.84 (0.66 to 1.05)	0.93 (0.73 to 1.18)	0.80 (0.60 to 1.06)
Inhalation habits				
Non-inhalers	154	1	1	1
Inhalers	846	3.98 (3.55 to 4.47)	3.23 (2.69 to 3.88)	3.56 (2.88 to 4.40)
Tobacco type				
Cigarettes	666	1	1	1
Cigars, cheroots or pipe	185	0.41 (0.35 to 0.48)	0.65 (0.54 to 0.79)	0.66 (0.53 to 0.81)
Mixed tobacco types	143	0.54 (0.45 to 0.66)	0.65 (0.52 to 0.80)	0.71 (0.56 to 0.90)

Results from Cox's proportional hazards regression analysis.
*Crude model adjusted for age, sex, and cohort of origin.
†Multivariate hazard ratios adjusted for age, sex, cohort of origin, inhalation habits, type of tobacco smoked, and years of smoking (continuous covariate).
‡Multivariate model and with exclusion of hospital admissions until 5 years after second examination (number of events=947).
§Stratified according to light or heavy smoking at the first examination.

Table 4 Risk of admission to hospital for COPD by changes in smoking habits and lung function measurements in the Copenhagen City Heart Study

	CCHS (n=3640)			Omitting first 5 years	
	No of events	Crude hazard ratio (95% CI)*	Adjusted hazard ratio (95% CI)†	No of events	Adjusted hazard ratio (95% CI)‡
Smoking category					
Heavy smokers	324	1	1	241	1
Quitters	27	0.58 (0.39 to 0.86)	0.65 (0.44 to 0.96)	18	0.41 (0.23 to 0.72)
Light smokers	14	0.36 (0.21 to 0.61)	0.84 (0.47 to 1.51)	13	1.07 (0.56 to 2.01)
Reducers	32	1.11 (0.83 to 1.24)	1.19 (0.82 to 1.71)	13	0.95 (0.58 to 1.54)
Tertile of FEV_1 (% predicted)§					
High	49	1	1	42	1
Middle	62	1.16 (0.80 to 1.69)	1.01 (0.70 to 1.48)	51	0.99 (0.66 to 1.50)
Low	286	4.70 (3.47 to 6.37)	3.98 (2.93 to 5.41)	192	3.39 (2.42 to 4.75)

Results from Cox's proportional hazards regression analysis.
*Adjusted for age and sex (total number of events = 397).
†Multivariate analysis adjusted for age, sex, inhalation habits, FEV_1 % predicted, and years of smoking (continuous).
‡Multivariate analysis and with exclusions of hospital admissions until 5 years after second examination. Total number of events = 285.
§Mean FEV_1 percent predicted: 103.7%, 85.4%, and 64.6%, respectively.

Table 5 Risk of admission to hospital for COPD in subjects with lowest baseline tertile of FEV_1 (% predicted) in the Copenhagen City Heart Study

	CCHS (n=1354)			Omitting first 5 years	
Smoking category	No of events	Crude hazard ratio (95% CI)*	Adjusted hazard ratio (95% CI)†	No of events	Adjusted hazard ratio (95% CI)‡
Heavy smokers	232	1	1	165	1
Quitters	21	0.61 (0.39 to 0.96)	0.69 (0.44 to 1.08)	11	0.47 (0.25 to 0.87)
Light smokers	6	0.35 (0.16 to 0.79)	0.73 (0.31 to 1.71)	5	0.96 (0.37 to 2.50)
Reducers	27	1.33 (0.89 to 1.99)	1.49 (1.00 to 2.23)	13	1.15 (0.65 to 2.04)

Results from Cox's proportional hazards regression analysis.
*Adjusted for age and sex (total number of events = 286).
†Multivariate analysis adjusted for age, sex, inhalation habits, and years of smoking (continuous).
‡Multivariate analysis and with exclusions of hospital admissions until 5 years after second examination (total number of events = 194).

predicted (table 4). Due to interaction effects between lung function and smoking history, this analysis was restricted to ever-smokers in the present study. FEV_1 in the lowest tertile was associated with an increased risk of hospital admission (HR 3.98, 95% CI 2.93 to 5.41). This adjustment for lung function did not alter the risk estimates significantly for quitters compared with the pooled analyses, whereas the association between light smoking and hospital admission was attenuated. In those who reduced smoking there was a non-significant increased risk (HR 1.19, 95% CI 0.82 to 1.71) which was reversed when the initial 5 years of analysis were omitted.

Confining analyses to participants with deteriorated baseline lung function (lowest tertile of FEV_1, mean 64.6% predicted) revealed that, after an "induction period" of 5 years, smoking cessation was associated with a decreased risk of admission to hospital for COPD compared with continued smoking (HR 0.47, 95% CI 0.25 to 0.87); this was not the case for light smokers or those who reduced smoking (table 5). This analysis was performed to study the possible effects of changes in smoking habits in participants presenting with a clinical diagnosis of COPD as determined by FEV_1.

DISCUSSION

Smoking cessation

Quitting smoking early in the study period was associated with a reduction in the risk of hospital admission for COPD of approximately 40%. This result remained unchanged after taking the "ill quitter" effect into account by omitting the first 5 years of analysis and by detailed adjustment for lung function in the largest cohort under study, the CCHS. Furthermore, participants who were ex-smokers at entry to the study had a risk of the outcome under study that was much lower than any of the other ever smoking groups. Smoking cessation has repeatedly been shown to be associated with improvement in pulmonary symptoms and slower deterioration in lung function compared with continuous smoking,[18–21] features that ultimately have been linked to improved survival. Furthermore, evidence is emerging that intermittent quitting also has a positive effect on the rate of decline in FEV_1.[22 23] Hospital admission for COPD reflects exacerbation and disease severity but most studies of this end point are small and the study populations highly selected. In this respect it is important to emphasise the role of smoking cessation compared with any other intervention on the risk of hospitalisation.

Smoking reduction

The results regarding smoking reduction were not so clearcut. In our primary multivariate model we found no difference in the risk of hospitalisation for COPD between heavy smokers who reduced to approximately 9 cigarettes/day and continuous heavy smokers. In comparison, individuals who were sustained light smokers throughout the study had a considerably lower risk of hospital admission than reducers and continuous heavy smokers. However, after excluding hospital admissions possibly related to this change in smoking behaviour, the results suggested a slight but not significant trend in favour of reducing. This trend persisted when adjusting for lung function. Smoking reduction was only associated with an increased risk of hospital admission in individuals with impaired baseline ventilatory function, a result which could reflect disease severity in these patients but which could also be a chance finding in a subgroup. The individual reasons for reducing or quitting smoking are many, but can be the result of physicians' advice or occur in response to adverse symptoms such as increasing dyspnoea. Certainly, a larger proportion of

those who reduced or quit smoking (85% and 78%, respectively versus 71% of the continuous heavy smokers) had an FEV_1 % predicted in the lowest tertile, suggesting that this could indeed be the case. Nevertheless, among this subgroup smoking cessation was associated with a halving of the risk of admission to hospital (table 5). Thus, some other health related factors that were not measured in this study could perhaps partially explain why there was no favourable effect of reducing tobacco consumption.

Some researchers have proposed implementation of smoking reduction as a means of reducing smoking related health threats in individuals who are unable or unwilling to quit the habit, but no long term evidence exists to support such a recommendation.[7 24] Studies of "cigarette fading" and subsequent smoking cessation, with or without the aid of nicotine replacement therapy (NRT), have indicated that it is possible to achieve and maintain a lower amount of tobacco intake, although these "reducers" seem to differ in some ways from subjects who succeed in cessation or continue smoking unchanged.[25–27] From the CCHS it has previously been shown that smoking reduction in subjects younger than 55 years of age was associated with a less pronounced decline in FEV_1 compared with persistent heavy smokers, whereas there was no effect in older subjects.[28] This finding is in part supported by a clinical trial showing signs of diminished lower respiratory tract inflammation in heavy smokers who underwent bronchoscopy before and after a short term smoking reduction.[29] Results from studies of smoking reduction in relation to tobacco related toxins and biomarkers of smoking are conflicting, but certainly mechanisms of compensatory smoking must be considered as an explanation for the discrepancy between the absolute magnitude of reduction and the measured smoke derivatives.[30] It is possible that our results reflect a causal relationship in that the pack-years of smoking accumulated up to the point of reducing, together with the fact that the airways are still exposed to smoke unlike those who quit, is not sufficient to reverse progressive airways disorder.

This study has the advantage of being prospective with a long follow up period and is large enough to include a sufficient number of events. In our analyses we have tried to account for the obvious sources of bias such as background differences in smoking experience and respiratory symptoms between reducers and continuing heavy smokers that would affect the outcome under study. However, it is still possible in an observational study to miss some potentially important factors. The reported changes in smoking habits took place chiefly in the late 1970s when there was not so much focus on smoking and health in general, and we have no information as to whether smoking cessation or reduction was intended and/or occurred with any kind of aid. Random misclassification of both exposure (smoking habits) and outcome (register diagnosis of COPD) could also be important and, despite our large sample size and frequent events, we might in general have underestimated the associations between the different smoking categories and COPD hospitalisations, hence failing to detect an effect of smoking reduction. Another limitation of the present study is the lack of repeated measures of biomarkers of smoking for validation. However, for a proportion of the quitters and reducers we found that their information regarding smoking habits at the second examination correlated well with levels of CO and cotinine at the third examination; expired CO was significantly lower in those who had reduced smoking than in sustained heavy smokers (table 2). Finally, it is likely that some reducers later quit smoking completely, in which case we will be overestimating the effect of reduction. Nevertheless, compensatory smoking alone and/or reuptake of moderate/heavy smoking could perhaps partially explain the less than expected benefit of reduced smoking.

In summary, this longitudinal population study confirms that smoking cessation is associated with a considerably decreased risk of COPD morbidity, even among predominantly middle aged individuals as well as those with impaired lung function. The risk of hospital admission for COPD in heavy smokers who reduce their tobacco consumption by at least 50% decreases by 7–20%, which is not statistically significant, compared with heavy smokers who do not change their habit. More research is needed, especially on the effects of smoking reduction, but the current results suggest that this is not a viable alternative or supplement to the existing strategies to reduce the harmful effects of tobacco.

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Authors' affiliations

N S Godtfredsen, E Prescott, The Copenhagen Centre for Prospective Population Studies, Danish Epidemiology Science Centre at the Institute of Preventive Medicine, Copenhagen University Hospital, Denmark
J Vestbo, Department of Respiratory Medicine, Hvidovre University Hospital, Denmark
M Osler, Department of Social Medicine and Psychosocial Health, Institute of Public Health, University of Copenhagen, Denmark

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