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Determinants of Chronic Mucus Hypersecretion in a General Population with Special Reference to the Type of Tobacco Smoked

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Data from a prospective study of 3884 smokers and 3676 non-smokers followed for five years were analysed to investigate the determinants of chronic mucus hypersecretion (CMH). During the follow-up 414 (10.7%) smokers and 140 (3.8%) non-smokers developed CMH. The influence of the type of tobacco smoked (plain cigarettes, filter cigarettes, pipe and cheroots/cigars), lifetime tobacco consumption, age, alcohol consumption, and socioeconomic status on the development of CMH was assessed in men and women separately using multiple logistic regression. In smokers of both sexes, the risk of developing CMH increased significantly with lifetime tobacco consumption and almost significantly with age. In male smokers, the risk of developing CMH increased with alcohol consumption but was not significantly related to the type of tobacco smoked. In female smokers, the risk of CMH increased significantly with short school education and was, after adjustment for the amount of tobacco smoked, approximately twice as high in cigarette smokers as in cheroot smokers. However, as female cheroot smokers on the average consumed much more tobacco than female cigarette smokers the incidence of CMH was almost the same in the two groups. Among current non-smokers, the risk of developing CMH increased with age and previous tobacco consumption. It is concluded that although a number of factors are associated with the development of CMH, tobacco smoking, regardless of the type of tobacco, is a major determinant of CMH.

Chronic mucus hypersecretion (CMH) is a common respiratory disorder.[1] Although CMH contrary to chronic airflow limitation does not seem to be related to mortality from chronic obstructive pulmonary disease (COPD),[2] it is associated with increased respiratory morbidity and may be related to the development of pulmonary neoplasms.[1-4] Tobacco smoking has for many years been identified as a major risk factor for CMH in western countries, but relatively little is known about possible differences in the effects of different types of smoking tobacco on CMH.[4-16] A number of earlier, mainly cross-sectional, studies have found that the risk of CMH increases with increasing cigarette tar yields and with the absence of filters, but two more recent studies have suggested that the benefit of tar reduction may have been overemphasized.[13,14] Studies comprising subjects smoking types of tobacco other than cigarettes have indicated that pipe and cigar smokers have a lower risk of CMH than cigarette smokers, but the observations were based on small number of subjects.[4,5,15,16]

The aim of this prospective study was to analyse the effects of different types of smoking tobacco (plain cigarettes, filter cigarettes, cheroots/cigars, pipe) and of other potential risk factors like poor socioeconomic status[17,18] and alcohol consumption[19,20] on the development of CMH in a general population.

METHODS

Population

All subjects included in the present analysis have participated in the Copenhagen City Heart Study, a pros-

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pective epidemiological survey of more than 14 000 subjects aged 20 years or more randomly selected from the general population of the city of Copenhagen. The subjects have been examined twice, the first time from 1976 to 1978 and secondly five years later from 1981 to 1983. Details on the selection procedure of the study sample, the questionnaire and the complete examination programme have been given previously.[21,22] From the total sample of 11 135 subjects who attended both examinations, the following subjects were excluded from the analysis (a) 344 people with a history of bronchial asthma; (b) 1866 people who changed the type of tobacco smoked during the follow-up or smoked more than one type of tobacco; (c) 626 subjects who stopped, started or restarted smoking during the observation period; (d) four female pipe smokers; (e) 170 subjects with incomplete data required for the analysis; (f) and 565 subjects who reported CMH at the first examination. The final study sample comprised 3884 smokers and 3676 non-smokers.

The subjects were grouped according to self-reported smoking habits during the follow-up:

NS: Non-smokers: subjects who at both examinations reported not smoking tobacco.
CIGP: subjects smoking plain, ie non-filter, cigarettes only.
CIGF: subjects smoking filter cigarettes only.
CER: women smoking cheroots, or cigarillos only and men smoking cigars, cheroots, or cigarillos only.
PIPE: men smoking pipe only.

In the assessment of tobacco consumption the following weight-equivalents for the tobacco content were used: plain cigarette or filter cigarette 1 g; cigar 5 g; cheroot and cigarillo 3 g; 1 g of pipe tobacco 1 g. For current smokers an estimate of life-time tobacco exposure was calculated as pack-years (current tobacco consumption (g/day) * duration of smoking (years)/20).

At both examinations the subjects reported their daily consumption of beer, wine, and spirits. For each participant mean daily alcohol consumption during the follow-up was estimated as the number of drinks a day (one drink = 10 g of alcohol).

As an indicator of socioeconomic status we used the duration of school education as reported at first examination. The subjects were registered as having CMH if they reported to have chronic phlegm ie cough and sputum production daily for at least three months a year for at least two years.

Statistical Methods

Data from smokers and non-smokers were analysed using multiple logistic regression. The dependent variable was development of CMH between the two examinations. In the regression model of smokers, the independent variables were binary variables representing the smoking groups (for men: X_{CIGP}, X_{CIGF}, X_{CER}, X_{PIPE} and for women: X_{CIGP}, X_{CIGF}, X_{CER}), age (with three levels: 20-39 years, 40-59 years, 60 years or more), lifetime tobacco consumption (with three levels: 1-14 pack-years, 15-29 pack-years, 30 pack-years or more), school education (with three levels: 1-7 years, 8-10 years, 11 years or more), and daily alcohol consumption (with two levels: 0-2 drinks/day, three drinks/day or more).

The regression model for non-smokers included age, school education, and alcohol consumption using similar levels as for smokers. Instead of life-time tobacco consumption, the duration of smoking was included using three levels: 0-14 years, 15-29 years, and 30 years or more.

Separate analyses were made for men and women. However, for non-smokers the results are shown for the two sexes together as the regression coefficients for men and women were very similar. The results are given in terms of odds ratios (OR) with 95% confidence intervals (CI).

First order interaction terms were investigated to see if they significantly improved the fit of the data. The level of significance was set to 5%.

RESULTS

General characteristics of the participants are given in Table 1. Cheroot/cigar smokers were on average older and consumed more than twice as much tobacco a day than smokers in the other smoking groups. Non-smokers consumed less alcohol a day than smokers. With regard to the length of school education the subjects in different smoking groups were very similar.

During the follow-up, 414 (10.7%) smokers and 140 (3.8%) non-smokers developed CMH. The differences in the incidence of CMH between subjects smoking different kinds of tobacco were small (Table 2).

The results of multiple logistic regression analysis on current smokers are presented in Table 3. In women, the risk of developing CMH increased significantly with pack-years and short school education and almost significantly with age. Women smoking cheroots had a significantly lower risk of developing CMH than women smoking plain cigarettes. The risk of CMH in women smoking filter cigarettes did not differ significantly from the risk among women smoking plain cigarettes.

In men, the risk of developing CMH increased significantly with number of pack-years, alcohol con-

TABLE 1 *General characteristics of the participants according to sex and type of tobacco smoked. Arithmetic means*

	Women				Men				
	NS	CIGP	CIGF	CER	NS	CIGP	CIGF	CER	PIPE
n =	2381	627	1331	373	1295	653	418	269	213
Age, years	53.5	50.3	48.0	59.2	52.1	50.8	46.3	56.6	53.7
Smoking duration, years	4.6	28.4	24.4	28 4	14.5	33.0	27.9	37.2	34.3
Tobacco consumption, g/day	0	13.4	12.5	30.7	0	17.1	16.6	35.8	15.6
Alcohol consumption, drinks/day	0.2	0.3	0.3	0.3	1.1	2.2	1.7	1.4	1.2
Education, years	8.5	8.2	8.5	8.2	8.9	8.4	8.7	8.7	8.8

Abbreviations:
NS: Non-smokers.
CIGP: Plain cigarette smokers.
CIGF: Filter cigarette smokers.
CER: Cheroot/cigar smokers.
PIPE: Pipe smokers.

sumption and almost significantly with age, but was not significantly related to the length of school education and the type of tobacco smoked (Table 3).

Among current non-smokers, the development of CMH was significantly associated with age and almost significantly associated with previous tobacco consumption (Table 4). Neither sex nor alcohol consumption nor education were significantly associated with the development of CMH.

No significant interactions could be demonstrated between pairs of the independent variables ie the OR for the independent variables may be considered as multiplicative.

DISCUSSION

The results of this population study show that smokers have a much higher incidence of CMH than non-smokers. After adjustment for the amount of tobacco smoked, women smoking cheroots have a significantly lower risk of developing CMH than women smoking cigarettes, but the differences between the effects of the remaining types of tobacco on the development of CMH are small. The amount of tobacco consumed is very closely related to the development of CMH. In addition to smoking, advanced age, and among smokers alcohol consumption and low socioeconomic status are associated with development of CMH.

In this study, we have chosen to look at the incidence of CMH rather than at the prevalence. This was done, as the choice of the tobacco smoked might be affected by pre-existing respiratory symptoms, making cross-sectional findings less interpretable. As the development of CMH is likely to be affected by the amount of tobacco smoked and by the duration of smoking, we have adjusted for the estimated life-time tobacco consumption in current smokers and for duration of previous smoking in non-smokers.

The main objective of the present analysis was to explore whether different types of tobacco have different effects on the development of CMH. In a number of earlier studies a lower prevalence of CMH in smokers of lower tar cigarettes compared with smokers of cigarettes with higher tar yields has been reported.[5,7,9-12] However, two recent studies have ques-

TABLE 2 *Number of subjects who developed chronic mucus hypersecretion (CMH) during the five year follow-up according to sex and smoking group*

	Women				Men				
	NS	CIGP	CIGF	CER	NS	CIGP	CIGF	CER	PIPE
New cases of CMH	84	64	109	34	56	93	52	35	27
Incidence %	3.5	10.2	8.2	9.1	4.3	14.2	12.4	13.0	12.7

Abbreviations:
NS: Non-smokers.
CIGP: Plain cigarette smokers.
CIGF: Filter cigarette smokers.
CER: Cheroot/cigar smokers.
PIPE: Pipe smokers.

TABLE 3 *Risk of developing chronic mucus hypersecretion in female and male smokers according to age, smoking group, pack-years, daily consumption of alcohol, and duration of school education*

	Women			Men		
	OR*	CI†	p‡	OR	CI	p
Age, years			0.10			0.08
20–39	1.0			1.0		
40–59	1.0	0.6–1.6		1.1	0.6–1.7	
60+	1.5	0.9–2.5		1.5	0.9–2.6	
Smoking group			0.02			0.46
Plain cigarettes	1.0			1.0		
Filter cigarettes	0.9	0.7–1.3		1.1	0.7–1.5	
Cheroots/cigars	0.5	0.3–0.8		0.7	0.5–1 1	
Pipe	—			1.0	0.6–1.5	
Pack—years			<0.001			<0.001
1–14	1.0			1 0		
15–29	1.5	1.1–2 2		2.0	1.2–3.4	
30+	3.2	2.1–4.9		2.8	1.6–4.9	
Alcohol, drinks/day			0.34			0.02
0–2	1.0			1.0		
3+	1.4	0.7–2.9		1.5	1.1–2 0	
Education, years			<0.01			0.42
0–7	1 0			1.0		
8–11	0.8	0.6–1.1		0.8	0.6–1.1	
12+	0.4	0.2–0.8		1.0	0.6–1.6	

Abbreviations:
* OR: odds ratio.
† CI: 95% confidence interval.
‡ Significance levels for testing coefficient(s) = 0.

tioned this association.[13,14] In the UK Heart Disease Prevention Project, the men who changed to lower tar cigarettes were significantly more likely to stop producing phlegm than the men who continued to smoke cigarettes with higher tar yields.[14] Yet, there was no dose-response relationship between the reduction in the tar yields of the cigarette smoked and the cessation of CMH. This was explained by compensation ie a more intense smoking among smokers who changed to cigarettes with very low tar yields. In addition, smokers who initially did not have CMH and changed to cigarettes with lower tar yields experienced a higher incidence of CMH during the follow-up than those who continued to smoke cigarettes with higher tar yields.[14] In a recent study from Finland there was no significant difference in the prevalence of CMH between smokers of low tar and medium tar cigarettes.[13] In the present study no significant difference between the risk of developing CMH between smokers of filter cigarettes and smokers of plain cigarettes has been observed. This might be due to the relatively small difference in the tar yields of the cigarettes compared. According to the data from The Scandinavian Tobacco Company the difference in tar yields was approximately 12 mg tar per cigarette, as plain cigarettes contained approximately 35 mg per cigarette and filter cigarettes approximately 23 mg. In view of the conflicting findings of previous studies and in view of the fact that manufactured cigarettes are constantly changing, further prospective studies should be conducted to investigate the possible differences in effect of the type of cigarettes on CMH.

After adjustment for the amount of tobacco consumed, the risk of developing CMH in men was only slightly and non-significantly lower in cheroot/cigar smokers than in cigarette smokers, whereas women smoking cheroots had a significantly lower risk than cigarette smokers. However, this difference had no practical implications as the consumption of tobacco in g/day was more than twice as high in cheroot smokers as in cigarette smokers resulting in almost the same incidence of CMH (Table 2). These observations diverge from the results of previous studies where the prevalence of CMH among pipe and cigar smokers has been considerably lower than among cigarette smokers.[1,15,16] This discrepancy might be due to very small numbers of cigar and pipe smokers included in previous studies and to the lack of adjustment for tobacco consumption. In studies from the US and UK,

TABLE 4 *Risk of developing chronic mucus hypersecretion in non-smokers according to sex, age, duration of smoking, daily consumption of alcohol, and duration of school education*

	OR*	CI†	p‡
Sex			0.84
Women	1.0		
Men	1.0	0.7–1.6	
Age, years			<0.01
20–39	1.0		
40–59	1.3	0.7–2.5	
60+	2.2	1.1–4.2	
Duration of smoking, years			0.09
0–14	1.0		
15–29	1.3	0.8–2.1	
30+	1.7	1.1–2.9	
Alcohol, drinks/day			0.55
0–2	1.0		
3+	1.2	0.6–2.3	
Education, years			0.39
0–7	1.0		
8–11	0.9	0.6–1.2	
12+	0.7	0.3–1.2	

Abbreviations:
* OR: Odds rato.
† CI: 95% confidence interval.
‡ Significance levels for testing coefficient(s) = 0.

the frequency of inhalers among pipe and cigar smokers is substantially lower than among cigarette smokers.[23,24] In our study, as in other studies from Scandinavia,[25] inhalation habits of cigarette smokers and of cigar/cheroot smokers and pipe smokers differ less. A number of European studies have shown that smoking of pipe, cigars and cheroots may be associated with as high a risk of lung cancer[26,27] and myocardial infarction[28] as cigarette smoking. Our results provide further evidence for the opinion that cheroot/cigar and pipe smoking should be considered as dangerous as cigarette smoking.

The observation of increased prevalence of CMH with age has been reported in many previous studies. However, it is not known whether this reflects the effect of cumulated exposure to environmental or occupational agents or the effect of ageing *per se*.[29] The weak association of short school education and alcohol consumption with increased risk of development of CMH in smokers is in accordance with our cross-sectional findings[30] and with findings from other studies.[17-20,31] However, our study cannot resolve whether these associations express causality or merely an association with other potential risk factors for CMH like poor living conditions, intercurrent respiratory infections, occupational stresses, or passive smoking, as these variables were not registered in our study. It is therefore possible that inclusion of such variables in the regression model would have changed the association between CMH and alcohol consumption and education. It is, however, less likely that this would have affected the strong relation of pack-years to CMH.[29] Finally, it is also possible that the association between high socioeconomic status and a lower risk of CMH is due to underreporting of CMH by women with higher socioeconomic status.

We conclude that smoking is a major determinant of CMH in the general population and that the differences in the risk of developing CMH between smokers of different types of tobacco are negligible.

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REFERENCES

[1] *Health or Smoking. Follow-up report of the Royal College of Physicians.* London: Pitman, 1983.
[2] Peto R, Speizer F E, Cochrane A L. *et al.* The relevance in adults of airflow obstruction, but not of mucus hypersecretion, to mortality from chronic lung disease *Am Rev Respir Dis* 1983; **128:** 491–500.
[3] Krzyzanowski M, Wysocki M. The relation of 13-year mortality to ventilatory impairment and other respiratory symptoms: The Cracow study. *Int J Epidemiol* 1986; **15:** 56–64.
[4] Fletcher C M, Peto R, Tinker C, Speizer F E. *The natural history of chronic bronchitis and emphysema.* Oxford: Oxford University Press, 1976.
[5] Comstock G, Brownlow W J, Stone R W, Sartwell P E. Cigarette smoking and changes in respiratory findings *Arch Environ Health* 1970; **21:** 50–7.
[6] Freedman S, Fletcher C M, Field G B. Effects of smoking modified cigarettes on respiratory symptoms and ventilatory capacity. *J Natl Cancer Inst* 1972; **48:** 1805–10.
[7] Rimington J. Phlegm and filters. *Br Med J* 1972; **2:** 262–4.
[8] Freedman S, Fletcher C M. Changes of smoking habits and cough in men smoking cigarettes with 30% tobacco substitute. *Br Med J* 1976; **1:** 1427–30.
[9] Dean G, Lee P N, Todd G F, Wicken A J, Sparks D N. Factors related to respiratory and cardiovascular symptoms in the United Kingdom. *J Epidemiol Comm Health* 1978; **32:** 86–96.
[10] Hawthorne V M, Fry J S. Smoking and health: The association between smoking behaviour, total mortality and cardiorespiratory disease in west central Scotland. *J Epidemiol Comm Health* 1978; **32:** 260–6.
[11] Higinbottam T, Clark T J H, Shipley M J, Rose G. Lung function and symptoms of cigarette smokers related to tar yield and number of cigarettes smoked. *Lancet* 1980; **1:** 409–12.
[12] Schenker M B, Samet J M, Speizer F E. Effect of cigarette tar content and smoking habits on respiratory symptoms in women. *Am Rev Respir Dis* 1982; **125:** 684–90.
[13] Rimpela A H, Rimpela M K. Increased risk of respiratory symptoms in young smokers of low tar cigarettes. *Br Med J* 1985; **1:** 1461–3.
[14] Peach H, Hayward D M, Ellard D R, Morris R W, Shah D. Phlegm production and lung function among cigarette smokers changing tar groups during the 1970. *J Epidemiol Comm Health* 1986; **40:** 110–6.

[15] Boudik F, Goldsmith J R, Teichman V, Kaufman P C. Epidemiology of chronic bronchitis in Prague. *Bull W H O* 1970; **42:** 711–22.

[16] Mueller R E, Keble D L, Plummer J, Walker S H. The prevalence of chronic bronchitis, chronic airway obstruction and respiratory symptoms in a Colorado City. *Am Rev Respir Dis* 1971; **103:** 203–28.

[17] Lebowitz M D. The relationship of socioeconomic factors to the prevalence of obstructive lung problems and other chronic conditions. *J Chron Dis* 1977; **30:** 599–611.

[18] Higgins M W, Keller J B, Metzner H L. Smoking, socioeconomic status, and chronic respiratory disease. *Am Rev Respir Dis* 1977; **116:** 403–10.

[19] Lebowitz M D. Respiratory symptoms and disease related to alcohol consumption. *Am Rev Respir Dis* 1981; **123:** 16–9.

[20] Holma B, Kjær G. Alcohol, housing and smoking in relation to respiratory symptoms. *Environ Res* 1980; **21:** 126–42.

[21] Jensen G. Epidemiology of chest pain and angina pectoris (thesis). *Acta Med Scand* 1984; **Suppl.** 682.

[22] Appleyard M. (ed.) The Copenhagen City Heart Study. *Scand J Soc Med* 1989; **Suppl. 41.**

[23] Doll R, Peto R. Mortality in relation to smoking: 20 years observations on male British doctors. *Br Med J* 1976; **2:** 1525–36.

[24] Hammond E C. *Smoking in relation to death rates of one million men and women. Natl Cancer Inst Monograph no.19.* Bethesda, Maryland: NIH 1966: 127–204.

[25] Carstensen J M, Pershagen G, Eklund G. Mortality in relation to cigarette and pipe smoking: 16 years observation of 25 000 Swedish men. *J Epidemiol Comm Health* 1987; **41:** 166–72.

[26] Abelin T, Gsell O R. Relative risk of pulmonary cancer in cigar and pipe smokers. *Cancer* 1967; **20:** 1288–96.

[27] Damber L, Larsson L G. Smoking and lung cancer with special regard to type of smoking and type of cancer. A case control study in North Sweden. *Br J Cancer* 1986; **53:** 673–81.

[28] Gyntelberg F, Lauridsen L, Pedersen P B, Shubell K. Smoking and risk of myocardial infarction in Copenhagen men aged 40-59 with special reference to cheroot smoking. *Lancet* 1981; **1:** 987–89.

[29] Burrows B. An overview of obstructive lung diseases. *Medical Clinics of North America* 1981; **65:** 455–71.

[30] Lange P, Groth S, Nyboe J. *et al.* Chronic obstructive lung disease in Copenhagen: cross-sectional epidemiological aspects. *J Int Med* 1989; 25–32.

[31] Saric M, Lucic-Palaic S, Horton R J M. Chronic non-specific lung disease and alcohol consumption. *Environ Res* 1977; **14:** 14–21.

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