Smoking habits and carboxyhaemoglobin

A cross-sectional study of an urban population of middle-aged men

LARS JANZON, SVEN-ERIK LINDELL, ERIK TRELL, AND PER LARME

From the Departments of Preventive Medicine, Internal Medicine, Surgery, and Clinical Physiology, University of Lund, Sweden

SUMMARY In this cross-sectional population study we report on the distribution of carboxyhaemoglobin concentrations in the morning, before smoking, in an urban population of 1037 men born in 1931. The median concentration was the same in non-smokers as in ex-smokers: 0.5%. It increased with increasing daily tobacco consumption. But when carboxyhaemoglobin concentrations are measured in reasonably well-standardised circumstances there are large variations between individuals, even in those who smoke equal amounts of tobacco a day. This makes it difficult to predict the concentration in the individual smoker when only his daily tobacco consumption is known. Measurements of carboxyhaemoglobin concentration should be a valuable complement to smoking history to identify the smoker at high risk of cardiovascular disease, to provide an extra argument to make the patient give up the habit, and to reinforce the efforts of those who try to do so.

Smoking is a well-established independent risk factor for cardiovascular disease.¹ According to results from animal experiments^{2,3} and cross-sectional population studies,⁴ the damaging effect of tobacco on the vessel wall seems to be related to the amount of carbon monoxide absorbed. The blood concentration of carbon monoxide is dependent on the amount of tobacco smoked a day, the brand of tobacco, inhalation habits, daily exercise habits, and exposure to other environmental sources of carbon monoxide.⁵ Therefore in the individual smoker the blood concentration cannot be predicted with certainty by knowing the number of cigarettes smoked a day. Hence, to identify which smokers are at high risk of cardiovascular diseases, direct measurements of their blood concentrations of carboxyhaemoglobin (COHb) should be a valuable complement to questions about their daily tobacco consumption. To control external sources of variation in COHb, measurements should be made in standardised conditions in terms of physical activity before blood sampling and time since the last cigarette was smoked.

The aim of the present cross-sectional population study was to measure COHb in middle-aged men in the morning, after a night's sleep and before smoking, and to relate the findings to their smoking habits.

Material and methods

The sample represented all men attending a screening programme in Malmö who were born in 1931. Smoking habits were assessed by questionnaire. The categories were defined as:

<i>Non-smoker</i>	Had not smoked 1 g of tobacco daily for one year.
<i>Ex-smoker</i>	Had not smoked for at least one month.
<i>Smoker</i>	Had smoked at least 1 g of tobacco daily for at least one year.
<i>Pure cigarette smoker</i>	(a) <10 a day (b) 10–20 a day (c) > 20 a day.
<i>Pure cigar smoker</i>	Not grouped.
<i>Pure pipe smoker</i>	(a) 1 pack a week (b) > 1 pack a week.
<i>Mix. smoker</i>	Smoked combinations of pipe, cigarettes, and/or cigars.

All the men were asked not to smoke in the morning before the examination. Compliance was checked by questioning. The carbon monoxide in the

Table Smoking category and carboxyhaemoglobin levels in middle-aged men in Malmö

Carboxyhaemoglobin (%)	SMOKING CATEGORY								All
	Non-smokers	Ex-smokers	Under 10	10-20	Over 20	Pipe smokers	Cigar smokers	Mixed smokers	
0-0.99	226	275	14	9	3	7	11	29	574
1-1.99	4	4	16	77	42	24	4	48	219
2-2.99	—	—	3	50	38	9	3	37	140
3-3.99	—	—	—	3	14	1	3	10	31
4-4.99	—	—	—	1	4	—	—	2	7
5-5.99	—	—	—	0	1	—	—	—	1
6-6.99	—	—	—	1	1	—	—	—	2
All	230	279	33	141	103	41	21	126	974
Per cent	23.6	28.6	3.4	14.5	10.6	4.2	2.2	12.9	100
Median COHb (%)	0.5	0.5	1.2	1.8	2.2	1.5	0.9	1.7	

blood was determined as the COHb concentration according to the method described by Collinson.⁶

The 1037 men in the study comprised 78% of the total available population in Malmö of men born in 1931. Sixty-three had to be excluded because they had smoked in the morning before the sample was taken. Of the participants, 230 (23.6%) were non-smokers, 279 (28.6%) ex-smokers, 277 (28.5%) cigarette smokers, 41 (4.2%) pipe smokers, 21 (2.2%) cigar smokers, and 126 (12.9%) mixed smokers. The distribution of the COHb concentrations in the different categories is shown in the Table. The median concentration was the same in non-smokers as in ex-smokers: 0.5%. Neither of these two groups had a concentration higher than 1.6%. With increasing cigarette consumption, the median concentration increased from 1.2% in those who smoked fewer than 10 cigarettes a day to 1.8% in those smoking 10 to 20 a day and 2.2% in those smoking more than 20 a day. The median value in pipe smokers, 1.5%, was about three times that in ex-smokers and non-smokers. The men classified as pure cigar smokers had concentrations about twice as high as those of ex-smokers and non-smokers.

Among 574 subjects with a COHb concentration of under 1%, 501 (87%) were non-smokers or ex-smokers. Only three of the 103 subjects who smoked more than 20 cigarettes a day had a concentration of under 1%. All those with a concentration of more than 3% smoked at least 10 g of tobacco daily. More than half of those who smoked more than 20 g of tobacco daily had a COHb value higher than 2% in the morning and 6% of them had a morning value higher than 4%.

Discussion

According to results from animal experiments,^{2,3} as well as from cross-sectional population studies,⁴ it seems that the damaging effect of tobacco on the vessel wall is related to the blood concentration of

carbon monoxide. Hence it would be rational to assess an individual's COHb concentration to identify those at high risk of cardiovascular disease. There is a large variation in COHb concentrations between people who smoke equal daily amounts of tobacco, which makes it difficult to predict that their concentrations in the morning will be twice as high as those of non-smokers. Almost 50% of those who smoked more than 10 cigarettes a day had concentrations four times as high as those of the average non-smoker. Twenty per cent of heavy smokers had COHb values six times as high as those of non-smokers. Whether a single measurement of the COHb value really reflects a person's average smoking pattern is, of course, open to question. The reproducibility of the test would depend on how well different external sources of variation were controlled. Measurements taken in the morning, before smoking and after a night's sleep, show smaller variations: in an earlier study of heavy smokers, the mean difference in COHb values between two samples (with an interval of eight to nine weeks between observations) was less than 1% (Janzon L, personal communication). Whether the morning value is a better predictor of future arteriosclerotic disease than the value later in the day is a question that has still to be determined in a prospective study. The difference in afternoon and evening values was found by Wald *et al*⁷ to be between 3% and 4%.

Whether direct measurement of COHb is a more valid and sensitive test to identify the smoker at high risk of cardiovascular diseases than assessment of daily tobacco consumption by questionnaire is another question that has still to be evaluated in a prospective study. However, in the case-control study by Wald *et al*,⁴ the concentration of COHb was found to be better correlated with the prevalence of angina pectoris, intermittent claudication, and previous myocardial infarction than the smoking history. There are now quick, cheap methods of measuring

Smoking habits and carboxyhaemoglobin

carbon monoxide in the expired air and the correlation between carbon monoxide in the expired air and in the blood has been found to be more than 0.9.⁸ Direct measurements of individual exposure to different harmful components of tobacco smoke should also be of value in programmes designed to persuade people to stop smoking and in providing information to help those who have already done so. Knowing the magnitude of different tobacco-induced disease¹ and the benefits of stopping smoking,⁹ we think that direct measurements of expired carbon monoxide should be used as a screening technique and also in most outpatient clinics.

This study was supported by the Swedish Tobacco Company.

Reprints from Dr. Lars Janzon, Department of Preventive Medicine, Malmö General Hospital, S-214 01 Malmö, Sweden.

References

- ¹US Department of Health, Education and Welfare. *Smoking and health (4). Cardiovascular diseases*. Washington DC: USDHEW, 1979: 7-67.
- ²Astrup P, Kjeldsen K, Wanstrup J. Enhancing influence of carbon monoxide on the development of atheromatosis in cholesterol-fed rabbits. *J Atheroscler Res* 1967; 7: 343-54.
- ³Thomsen HK. Carbon monoxide-induced atherosclerosis in primates. *Atherosclerosis* 1974; 20: 233-40.
- ⁴Wald N, Howard S, Smith PG, Kjeldsen K. Association between atherosclerotic diseases and carboxyhaemoglobin levels in tobacco smokers. *Br Med J* 1973; i: 761-5.
- ⁵Wald N, Howard S, Smith PG, Bailey A. Use of carboxyhaemoglobin levels to predict the development of diseases associated with cigarette smoking. *Thorax* 1975; 30: 133-40.
- ⁶Collison HA, Rodkey FL, O'Neal JD. Determination of carbon monoxide in blood by gas chromatography. *Clin Chem* 1968; 14: 162.
- ⁷Wald N, Howard S. Variations of carboxyhaemoglobin levels in smokers. *Br Med J* 1975; i: 393.
- ⁸Cohen SI, Perkins NM, Ury HK, Goldsmith JR. Carbon monoxide uptake in cigarette smoking. *Arch Environ Health* 1971; 22: 55-60.
- ⁹Schuman LM. The benefits of cessation of smoking. *Chest* 1971; 59: 421-7.