CLINICAL RESEARCH

Smoking, lung function, and body weight

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Abstract

In a cross-sectional study of steelworkers aged 45-55 years, smokers (n=105; mean weight 76·1 kg) were found to weigh significantly less than non-smokers (n=54; 81·6 kg) and ex-smokers (n=51; 82·6 kg). The lower weight of smokers was attributable to a group with airflow obstruction (n=37; forced expiratory volume in one second/vital capacity (FEV_1/VC) <66%), who weighed less (4·8 kg; $p<0·05$) than smokers with normal FEV_1/VC (n=68). In smokers, but not in ex-smokers or non-smokers, body mass index and FEV_1/VC ratio were closely related ($r=0·34$; $p<0·001$). This association was apparently not due to an effect of body weight on lung function.

Weight loss in smokers may be the consequence of impaired lung function or reflect the effect of cigarette smoking on both the respiratory tract and metabolism in susceptible subjects.

Introduction

Differences in lung function between smokers and non-smokers are due to the presence among smokers of an appreciable minority of "susceptible" subjects. In these susceptible smokers the decline in lung function with age is more rapid than in non-smokers and in most other smokers.[1] On the other hand, non-smoking men are on average slightly heavier than smokers, although this seems to be the case only in manual workers.[2] There is evidence, mainly from the study of subjects who gained weight after stopping smoking, that these differences in body weight are due to an effect on metabolism rather than to suppression of appetite by smoking.[2 3] Whether the effects of cigarette smoking on body weight and lung function are interrelated, however, remains to be determined.

We report on the observation that in a homogeneous population of working men smokers with airflow obstruction weighed less than non-smokers, ex-smokers, and smokers without airflow obstruction.

Subjects and methods

We studied manual steelworkers aged 45-55 with at least 10 years of service in a steelworks near Brussels. Details of the subjects and lung function values have been reported.[4 5] In brief, the subjects comprised 54 lifelong non-smokers free of respiratory symptoms, 105 current smokers, and 51 former smokers out of 272 subjects studied. Of the 62 subjects whom we excluded, 38 had not completed all lung function tests, 12 were symptomatic non-smokers, seven had bronchial asthma or other lung disease, and five were irregular smokers or had recently stopped smoking. Among the various indices of lung function measured we selected for the present analysis the ratio of forced expiratory volume in one second (FEV_1) to vital capacity (VC), expressed as a percentage. This index is considered to be a reliable predictor of airflow obstruction and to be virtually unaffected by body size.[6]

Standing height without shoes was measured to the nearest cm, and weight was measured to the nearest kg, the subjects wearing their usual working clothes. These measurements were made by the same person and with the same gauge and balance throughout. Body mass index (weight (kg) divided by height (m) squared) was used, since this is best related to body fatness.[7] Standard statistical methods (Student's t test and orthogonal linear regression) were used[8] and the level of significance fixed at $p<0·05$.

Results

Table I gives the anthropometric characteristics and FEV_1/VC ratios of the subjects according to their smoking habits. Age and height were comparable in the three groups. Smokers weighed significantly less than both non-smokers and ex-smokers ($p<0·001$). By the classification of Bray,[9] most of the subjects were overweight (body mass index 25-30) in all three categories. Of the 105 smokers, however, only 11 were obese (body mass index $\geqslant 30$[9]), as opposed to 32 of the 105 non-smokers and ex-smokers ($p<0·001$). Conversely, 32 of the smokers were not overweight (body mass index <25[9]), as opposed to only seven subjects in each of the other two groups ($p<0·01$). The FEV_1/VC ratio was less in smokers than in non-smokers and ex-smokers ($p<0·01$).

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The smokers were further classified (table II) according to whether or not they had an FEV_1/VC ratio less than 66·6%—that is, lower than the one-sided 95% limit derived from the data on the asymptomatic non-smokers.[4] The lower body mass index of the smokers was apparently fully accounted for by the lower body mass index ($p < 0·005$) of the subjects with airflow obstruction ($FEV_1/VC < 66·6\%$). This may be expressed another way (figure): in smokers the linear correlation of FEV_1/VC and body mass index was highly significant ($r = 0·34$; $p < 0·001$). In neither non-smokers nor ex-smokers was this correlation significant. No substantial changes in any of these correlations occurred when age, pack-years smoked, or time since giving up smoking were taken into account by computing partial correlations. Body mass index and FEV_1/VC ratio were comparable in asymptomatic non-smokers and smokers without airflow obstruction ($p > 0·05$).

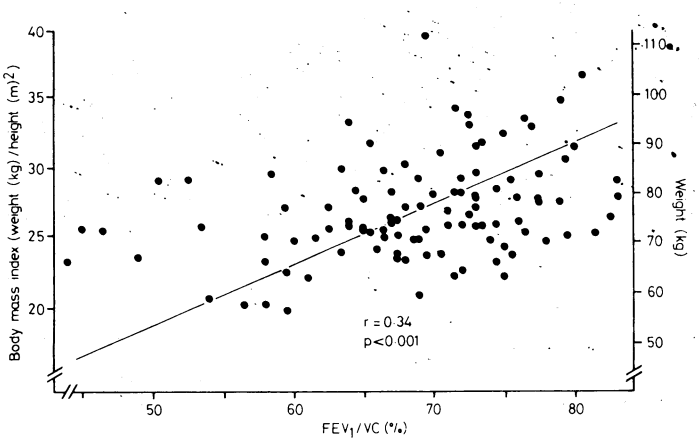
TABLE I—*Characteristics of non-smokers, ex-smokers, and smokers. Values are means ± SD*

	Asymptomatic non-smokers (n = 54)	Ex-smokers (n = 51)	Smokers (n = 105)	
Age (years)	49·3 ± 3·2	49·2 ± 2·9	49·6 ± 3·0	
Height (m)	1·70 ± 0·06	1·69 ± 0·07	1·69 ± 0·06	
Weight (kg)	81·6 ± 9·3	82·6 ± 13·0	76·1 ± 10·3	($p < 0·001$*)
Body mass index (weight (kg)/height (m)²)	28·26 ± 3·81	28·86 ± 3·81	26·84 ± 3·55	($p < 0·005$*)
FEV_1/VC (%)	73·9 ± 4·5	72·0 ± 5·3	68·6 ± 8·3	($p < 0·01$*)

*Significance of difference between smokers and non-smokers plus ex-smokers.

TABLE II—*Characteristics of smokers with and without airflow obstruction. Values are means ± SD*

	Smokers without airflow obstruction ($FEV_1/VC > 66·6\%$) (n = 68)	Smokers with airflow obstruction ($FEV_1/VC < 66·6\%$) (n = 37)	
Age (years)	49·5 ± 3·2	49·6 ± 2·7	
Height (m)	1·68 ± 0·06	1·69 ± 0·06	
Weight (kg)	77·8 ± 11·0	73·0 ± 8·2	($p < 0·025$)
Body mass index (weight (kg)/height (m)²)	27·56 ± 3·59	25·51 ± 3·08	($p < 0·005$)
Smoking (pack-years)	32 ± 14	33 ± 15	
Current smoking (No of cigarettes daily)	19 ± 10	20 ± 10	
FEV_1/VC (%)	73·4 ± 4·3	59·8 ± 6·4	



Relation of body mass index (weight/height²) *v* FEV_1/VC ratio in 105 current smokers (slope of regression line given by orthogonal regression coefficient). Weight for standardised height of 169 cm presented on right side of ordinate.

Discussion

We found that among middle-aged steelworkers smokers weighed less than lifelong non-smokers and former smokers (table I). Our results agreed closely with those of Khosla and Lowe,[10] who also studied steelworkers: most of their men were above the limits of desirable weights, smokers being on average lighter than non-smokers and ex-smokers. Khosla and Lowe, however, did not discuss the reason for these differences, nor did they mention any association with lung function findings. The novel observation in our study was that the lower weight and body mass index of smokers as a group was attributable to a subset of subjects with airflow obstruction, as evidenced by a low FEV_1/VC ratio (table II). Indeed, body mass index was comparable in non-smokers and smokers without airflow obstruction. Body mass index and FEV_1/VC were highly significantly ($p < 0·001$) correlated in smokers (figure) but not in non-smokers. It could be argued that the correlation coefficient was small and that therefore only a small part of the variances of these indices could be explained by their correlation. Nevertheless, this was not surprising in view of the many different determinants of body weight and lung function.

There are three possible explanations for the association: body weight may influence lung function, lung function may influence body weight, or both body weight and lung function may be influenced by one or more common factors. The first possibility—that body weight affects FEV_1/VC—may be readily excluded, since there was no significant correlation between body mass index and FEV_1/VC in non-smokers. This agrees with other data: the influence of body weight alone on lung function may be considered negligible within the range of the usual weights, since two opposite effects seem to operate. Increasing weight is accompanied to some extent by an improvement in lung function ("muscularity effect"), but with further increases in weight there is a decrease in lung function ("obesity effect").[11] This results in a virtual plateau of the relation between FEV_1/VC and body weight over the usual weights.[11 12] This was not so in our smokers, who showed a significant relation between body mass index and FEV_1/VC ratio.

The second possibility may therefore be considered—that is, that impaired lung function caused the affected smokers to lose weight. Progressive weight loss occurs in patients with overt chronic obstructive lung disease and has been attributed to loss of appetite and reduced food intake,[13] to increased energy requirements,[14] or to reduced production of anabolic steroid hormones.[15] Our population of active smokers, however, was clearly different from the hospital populations in those studies. Indeed, only four of our smokers had an FEV_1/VC ratio below 50% (see figure), and only three subjects reported shortness of breath when walking on a level floor. If lung function did influence body weight in these men (and there was little evidence to support this) our findings would mean that weight loss, which is known to be an unfavourable prognostic factor in overt chronic obstructive lung disease,[13 16] may well start quite early in the course of the disease.

The third possibility is that body weight and loss of pulmonary function are influenced by some common factor or factors. For instance, subjects who are genetically susceptible to chronic obstructive lung disease may also be leaner than non-susceptible subjects. Leanness has been identified as one of the risk factors predicting chronic obstructive airways disease in men.[17] Alternatively, cigarette smoking may affect both lung function[1] and energy metabolism.[3] Decreases in basal oxygen consumption, protein-bound iodine concentration, and 30-minute postprandial blood glucose values have been observed shortly after giving up smoking.[3] More recently it has been shown that smoking women have a smaller weight increase during pregnancy than non-smoking women, food intake being apparently similar in both groups.[18] We may hypothesise that susceptible smokers differ from resistant ones by their cellular response (from the respiratory tract and body fat regulation system) to cigarette smoke. Recently, polymorphs from patients with irreversible obstructive airways disease were shown to be more sensitive to the cytotoxicity of cigarette smoke than polymorphs from subjects with no respiratory disability.[19] It is not clear whether this increase in sensitivity is restricted to polymorphs (which are probably implicated in the development of emphysema[20]) or whether it reflects a more general process.

Whatever the reasons for the association between body weight and lung function in smokers, the implications of our findings

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are threefold. Firstly, an understanding of the mechanisms by which body weight and lung function are possibly linked could help in understanding, at the cellular level, the pathogenesis of smoking-induced chronic obstructive lung disease. Secondly, the relation between lower weight and impaired lung function, as found in this study, may partly explain the conflicting results relating overweight and general mortality in epidemiological studies.[21] Finally, from a practical point of view, loss of excess weight in a middle-aged smoker should not always be regarded as favourable, since it could point to deteriorating lung function and susceptibility to chronic obstructive lung disease.

C Pahulycz and C Veriter gave invaluable technical help in this study. We also thank Drs M Mawet, A Kivits, and J M Cordier; Mr J Pinese (Centre Belge de Médecine du Travail); and the management, unions, and participating workers from the Forges de Clabecq SA for their collaboration; Professor D V Parke (University of Surrey) and Dr D Rodenstein for their comments; Mr R Van Leemputten for drawing the figure; and Mrs J Bleeckx for typing the manuscript.

The study was supported by grant No 7246-22-2-002 from the European Coal and Steel Community.

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(*Accepted 22 October 1982*)

Acute acalculous cholecystitis complicating systemic lupus erythematosus: case report and review

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Abstract

A case of acalculous cholecystitis presented as an acute abdominal emergency in a 22 year old woman with severe systemic lupus erythematosus. At the time of presentation the patient was receiving high doses of prednisone and cyclophosphamide to control her underlying disease. Histological examination of the biopsy specimen from the gall bladder showed lupus vasculitis.

This complication of systemic lupus erythematosus has not been reported before. Laboratory studies and changes in lupus activity may fail to predict the onset of cholecystitis.

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Introduction

Mesenteric ischaemia, with or without bowel perforation, is reportedly the commonest acute abdominal complication of systemic lupus erythematosus,[1 2] and acute pancreatitis is also well documented.[3] Vasculitis of the gall bladder presenting as an acute abdomen occurred in a patient with giant-cell arteritis,[4] and gall-bladder disease may also occur in polyarteritis nodosa and allergic granulomatosis.[5]

We report a case of acute acalculous cholecystitis due to lupus vasculitis in a patient with severe systemic lupus erythematosus. This complication has not been reported before.

Case report

A 22 year old woman presented with diffuse vasculitis of the skin. Serological tests suggested systemic lupus erythematosus, and LE cells were identified. Creatinine clearance was low (normal urinary sediment), and renal biopsy showed type 4 (WHO classification) lupus nephritis. Steroids and cyclophosphamide were begun. Repeat renal biopsy six months later showed a noticeable improvement, and the steroids were subsequently reduced and cyclophosphamide stopped.

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