Tar yield of cigarettes and risk of acute myocardial infarction

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Abstract

***Objective*—To analyse the relation between tar and nicotine yield of cigarettes smoked in the recent past and the risk of myocardial infarction.**

***Design*—Multicentre case-control study conducted between September 1988 and June 1989.**

***Setting*—Over 80 coronary care units in various Italian regions.**

***Subjects*—916 patients with acute myocardial infarction without history of ischaemic heart disease and 1106 controls admitted to hospital for acute conditions not related to known or suspected risk factors for ischaemic heart disease.**

***Main outcome measures*—Relative risk of myocardial infarction according to type of cigarette smoked adjusted for identified potential confounding factors. Brands of cigarettes classified according to yield of tar and nicotine.**

***Results*—Patients with acute myocardial infarction were more often smokers and among smokers they tended to smoke more cigarettes. Compared with non-smokers their estimated relative risks were 3·8, 4·3, 3·2, and 3·7 in the four categories of tar yield (<10, 10-15, >15-20, and >20 mg, respectively). No trend in risk across yields was evident when analysis was restricted to smokers and allowance was made for number of cigarettes. Compared with risks in subjects in the lowest category of tar yield the relative risks were 1·2, 0·8, and 1·0 for the subsequent yields. Compared with risks in non-smokers the relative risks ranged from 9·3 to 12·6 below the age of 50 but no trend was observed with increasing yield.**

***Conclusions*—Changing to cigarettes with a lower tar yield is not an effective means of reducing tobacco related morbidity from myocardial infarction.**

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Introduction

There is definite evidence that smoking cigarettes increases the risk of acute myocardial infarction and other cardiovascular diseases.[1] In several developed countries the concern about the health hazards of cigarette smoking led many smokers to stop.[2] Encouraged by the tobacco industry, many others have changed to low tar and low nicotine cigarettes with the hope that this would considerably reduce the health risks associated with smoking.[2] There is in fact consistent evidence that the risks of lung and other tobacco related cancers are directly related to the tar yield of cigarettes.[3,4]

Most deaths caused by smoking are, however, due to cardiovascular disease,[5] and thus the relation between risk of cardiovascular disease and cigarette yield is a crucial issue in public health, but the evidence is scanty and controversial.[6-11] We analysed the relation between cigarette yield and risk of acute myocardial infarction by using data from a large Italian case-control study.

Subjects and methods

Our study was conducted between September 1988 and June 1989 in over 80 hospitals in various parts of Italy that were participating in the second study of the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto (GISSI-2), a randomised clinical trial of alteplase *v* streptokinase and heparin *v* no heparin for treating acute myocardial infarction in 12 490 subjects.[12] The design of this investigation has already been described.[13]

In the present case-control investigation there were 916 subjects (801 men and 115 women, referred to as cases) randomised to this second study who were admitted to hospital for a confirmed episode of acute myocardial infarction and with no history of ischaemic heart disease. The median age was 57 (range 24-74) years.

The controls were subjects admitted to the same hospitals for acute conditions not related to known or suspected risk factors of acute myocardial infarction. Subjects with a history of coronary heart disease or admitted for neoplastic, cardiovascular, or cerebrovascular disease or for any chronic condition were specifically excluded from the comparison group. A total of 1106 control subjects (976 men and 130 women) were interviewed. Of these, 487 were admitted for traumatic conditions, 121 for non-traumatic orthopaedic disorders, 277 for surgical conditions, and 221 for other miscellaneous illnesses such as ear, nose, throat, or dental disorders. Controls were frequency matched to cases for age, sex, and hospital. Their median age was 57 (range 23-74) years. Fewer than 3% of all subjects approached (cases and controls) refused to be interviewed.

A structured questionnaire including questions on sociodemographic factors and lifestyle habits, diet, coffee and alcohol consumption, physical activity, history of selected conditions, and family history of cardiovascular and cerebrovascular events was administered to cases and controls by trained interviewers. For 614 cases and 792 controls a measure of serum cholesterol was also obtained.

Questions on smoking included smoking habit (never, former, current), and for current and former smokers the number of cigarettes or cigars or pipes smoked a day, duration of smoking, history of the brands of cigarettes used in the past and specifically in the previous six months, and for former smokers time since stopping. Subjects who had stopped smoking less than two years before were considered as current smokers.

Tar and nicotine yields of cigarettes sold in Italy are published annually by the Ministry of Finances.[14] The brands were classified according to their tar yield into four categories: <10 (low yield), 10-15, >15-20, and over 20 mg (high yield). The lowest category included most newer, vented filter cigarettes; the highest category the oldest unfiltered ones; and the two

intermediate categories other filtered cigarettes. Though tar was used for the classification, the brands were also classified according to their nicotine yield with only few exceptions. The nicotine yield of the four categories was, respectively, <0·6, 0·5-0·8, 0·8-1·3, and 1·1-1·6 mg. No measure of carbon monoxide was available.

STATISTICAL ANALYSIS

Relative risk of myocardial infarction and the corresponding 95% confidence intervals for various categories of smokers compared with those who had never smoked and within smokers were estimated by multiple logistic regression models.[15] The variables included in the models were sex, age (in five year groups), education (three levels) serum cholesterol concentration (tertiles plus one dummy variable for missing values), history of diabetes and hypertension, family history of acute myocardial infarction, body mass index (three levels), and coffee consumption (four levels). The significance of the linear trends in risk was assessed by comparing the difference of the deviances of the models with and without the relevant term to the χ^2 distribution with one degree of freedom.

Results

The two groups were comparable in terms of sex and age. Patients with myocardial infarction tended to be more educated, had higher serum cholesterol concentrations, more commonly had diabetes and hypertension, more often had relatives with ischaemic heart diseases, drank more coffee, and had higher body mass indices.[13]

Table I shows the distribution of subjects and controls according to smoking habit and for current smokers the number of cigarettes smoked daily, the duration of smoking, and the corresponding relative risks. Patients with acute myocardial infarction were more often smokers and among smokers they tended to smoke more cigarettes. Compared with non-smokers, former smokers had a relative risk of 1·3, and the risk for current smokers was 2·1 for smokers of fewer than 15 cigarettes a day, 3·1 for smokers of 15-24 cigarettes a day, and 4·3 for smokers of 25 or more cigarettes a day. No trend in risk was evident among categories of duration, the risk being around 3·0 for all categories.

There was a significant interaction between smoking and age (χ^2_3=20·08, p<0·01), the risk estimates being substantially higher at younger ages. No significant interaction was found with the other covariates included in the models.

Table II presents the distribution of current smokers and the corresponding estimated relative risks according to cigarette yield. Most smokers were in the third category of yield. Compared with those in non-smokers the estimated risks were 3·8 in the lowest category and 4·3, 3·2, and 3·7 in the subsequent categories.

In table III the risks associated with various cigarette yields are presented in current smokers only. In both models allowance was made also for number of cigarettes. When compared with those in subjects in the lowest yield category the risk estimates were 1·2, 0·8, and 1·0 for the subsequent yields. Thus no trend in risk with yield was observed.

Table IV presents the estimated relative risks of current smokers of different yields of cigarettes relative to non-smokers stratified for age. Below the age of 50 the relative risks ranged from 9·3 to 12·6, but no trend with increasing yield was observed. No trend was evident even above the age of 50, although the risk estimates were considerably lower, ranging from 2·6 to 3·7.

Discussion

This case-control study on acute myocardial infarction conducted within the framework of the GISSI-2 clinical trial[12] further confirms the importance of smoking as a cause of the disease, independently from tar and nicotine yield of the last brand smoked. The absence of major advantage by smoking low yield cigarettes on the risk of acute myocardial infarction is now unequivocally established.

The choice of hospital controls could be criticised as smokers tend to be admitted to hospital more often than non-smokers and tend to stay for longer periods.[16] This could lead to an over-representation of smokers in the control group compared with the general population and hence to an underestimation of the relative risk of smoking. A major underestimation would be surprising, however, as the risk estimates of this study were among the highest reported to date. Furthermore, we excluded from the comparison group all patients with diagnoses known to be or potentially related to smoking, and the smoking related risks were comparable across major diagnostic categories of the controls. Patients with acute myocardial infarction lived long enough to be admitted to hospital and interviewed; therefore, they do not represent all patients with acute myocardial infarction. In this study we could classify the most recent brand smoked according to tar and nicotine but not yield of carbon monoxide. Although the exact component of tobacco associated with acute myocardial infarction has not been defined[1] and carbon monoxide is not necessarily correlated with tar and nicotine yields,[17-19] the classification produced is detailed enough to allow distinction

TABLE I—*Distribution of 916 cases of acute myocardial infarction and 1106 controls, estimated relative risks, and 95% confidence intervals according to number of cigarettes smoked and duration of smoking*

Details of smoking habit	No (%) of cases	No (%) of controls	Relative risk (95% confidence interval) Model 1*	Model 2†
Never smoked	150 (16·4)	320 (28·9)	1‡	1‡
Former smokers	139 (15·2)	243 (22·0)	1·4 (1·0 to 2·0)	1·3 (0·9 to 1·8)
Pipe or cigar smokers	3 (0·3)	9 (0·8)	0·8 (0·2 to 3·2)	0·8 (0·2 to 3·0)
No of cigarettes/day smoked by current smokers:				
<15	111 (12·1)	147 (13·3)	1·9 (1·4 to 2·7)	2·1 (1·5 to 3·0)
15-24	262 (28·6)	256 (23·1)	2·9 (2·2 to 3·9)	3·1 (2·2 to 4·2)
≥25	251 (27·4)	131 (11·8)	5·6 (4·1 to 7·8)	4·3 (3·0 to 6·2)
χ^2 trend§			121·43‖	73·78‖
Duration of smoking (years)¶:				
<30	189 (20·6)	166 (15·0)	3·4 (2·3 to 4·9)	3·1 (2·0 to 4·6)
30-39	192 (21·0)	170 (15·4)	3·3 (2·4 to 4·6)	3·2 (2·2 to 4·6)
≥40	243 (26·5)	197 (17·8)	3·0 (2·2 to 4·1)	2·9 (2·1 to 4·2)

*Estimates from multiple logistic regression models including terms for sex, age, and education.
†Estimates from multiple logistic regression models including terms for sex, age, education, cholesterol concentration, history of diabetes and hypertension, family history of acute myocardial infarction, body mass index, and coffee consumption.
‡Reference category.
§Former smokers and pipe/cigar smokers excluded.
‖p<0·01.
¶Information missing for one control subject.

TABLE II—*Distribution of cases of acute myocardial infarction and controls,* estimated relative risks, and 95% confidence intervals according to the tar and nicotine yield of brand smoked during past six months*

Details of smoking habit	Cases	Controls	Relative risk (95% confidence interval) Model 1†	Model 2‡
Never smoked	150	320	1§	1§
Tar and nicotine yield for current smokers:				
1 (< 10 mg tar, < 0·6 mg nicotine)	72	51	3·8 (2·4 to 5·8)	3·8 (2·3 to 6·1)
2 (< 10-15 mg tar, 0·5-0·8 mg nicotine)	103	57	4·6 (3·1 to 7·0)	4·3 (2·8 to 6·8)
3 (> 15-20 mg tar, 0·8 to 1·3 mg nicotine)	364	307	3·4 (2·5 to 4·5)	3·2 (2·3 to 4·3)
4 (> 20 mg tar, 1·1-1·6 mg nicotine)	54	51	3·3 (2·1 to 5·2)	3·7 (2·3 to 6·2)
Unknown	31	68		

*Former smokers and pipe or cigar smokers excluded.
†Estimates from multiple logistic regression models including terms for sex, age, and education.
‡Estimates from multiple logistic regression models including terms for sex, age, education, cholesterol concentration, history of diabetes and hypertension, family history of acute myocardial infarction, body mass index, and coffee consumption.
§Reference category.

TABLE III—*Estimated relative risks of acute myocardial infarction and 95% confidence intervals among current cigarette smokers according to tar and nicotine yield of brand smoked during past six months*

Tar and nicotine yield	Relative risk (95% confidence interval) Model 1*	Model 2†
1 (< 10 mg tar, < 0·6 mg nicotine)	1‡	1‡
2 (10-15 mg tar, 0·5-0·8 mg nicotine)	1·3 (0·8 to 2·1)	1·2 (0·7 to 2·1)
3 (> 15-20 mg tar, 0·8-1·3 mg nicotine)	0·9 (0·6 to 1·4)	0·8 (0·5 to 1·3)
4 (> 20 mg tar, 1·1-1·6 mg nicotine)	0·9 (0·5 to 1·6)	1·0 (0·5 to 1·8)

*Estimates from multiple logistic regression models including terms for sex, age, education, and number of cigarettes.
†Estimates from multiple logistic regression models including terms for sex, age, education, number of cigarettes, cholesterol concentration, history of diabetes and hypertension, family history of acute myocardial infarction, body mass index and coffee consumption.
‡Reference category.

TABLE IV—*Estimated relative risks* of acute myocardial infarction and 95% confidence intervals for current cigarette smokers stratified for age according to tar and nicotine yield of brand smoked in past six months*

Details of smoking habit	Age (years) < 50	> 50
Never smoked	1†	1†
Tar and nicotine yield for current smokers:		
1 (< 10 mg tar, < 0·6 mg nicotine)	9·3 (3·7 to 23·2)	3·1 (1·8 to 5·1)
2 (10-15 mg tar, 0·5-0·8 mg nicotine)	12·6 (5·3 to 29·8)	3·7 (2·3 to 6·0)
3 (> 15-20 mg tar, 0·8-1·3 mg nicotine)	9·6 (4·7 to 19·5)	2·6 (1·9 to 3·6)
4 (> 20 mg tar, 1·1-1·6 mg nicotine)	9·7 (3·4 to 27·6)	2·6 (1·5 to 4·3)

*Estimates from multiple logistic regression models adjusted for sex, age, and education.
†Reference category.

between newer low yield cigarettes and higher yield ones in terms of public health.

The risk of acute myocardial infarction was not associated with duration of smoking, and former smokers had a substantially lower risk than current smokers.[1] Thus, the risk of acute myocardial infarction was influenced principally, if not completely, by smoking habit in the recent past, and therefore we used for all analyses the yield of the brand smoked in the previous six months. This might even explain the higher risk estimates of this study compared with several cohort studies in which assessment of smoking habits was often made several years before the disease developed and thus a greater misclassification among categories of smoking is conceivable. We also ensured the accurate definition of diagnosis for cases, a comparable setting of interview for cases and controls, and the adequate allowance for quantity smoked and other tobacco related variables. The response rate was particularly high.

Our results are consistent with those from two large case-control studies from America based on 502 cases of myocardial infarction in men aged 30-54[8] and 910 in women under 65.[11] In both sexes the risk did not vary according to the nicotine or carbon monoxide yield of the cigarette. The results from some cohort studies are also similar,[7,9] but evidence from cohort studies is not totally consistent. These studies were, however, based on small numbers of cases[10] and could not provide adequate information on covariates or on smoking in the recent past before the episode of infarction.[6]

In conclusion, therefore, shifting to lower yield cigarettes is not an effective means of reducing tobacco related morbidity from myocardial infarction.

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Prospective study of incidence of juvenile diabetes mellitus over 10 years in Dar es Salaam, Tanzania

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Abstract

***Objective*—To ascertain the annual incidence of diabetes requiring treatment with insulin in children and adolescents aged 0-19 years in Dar es Salaam, Tanzania, during a 10 year period from 1 January 1982 to 31 December 1991.**

***Design*—Prospective registration at a major urban hospital of all patients with newly diagnosed diabetes who were resident in Dar es Salaam.**

***Setting*—Muhimbili Medical Centre, Dar es Salaam, Tanzania.**

***Patients*—86 patients: 45 male, 41 female.**

***Results*—The annual incidence of juvenile diabetes for both sexes was 1·5 per 100 000 population aged 0-19 years (95% confidence interval 1·3 to 1·7). Incidence per 100 000 population per year increased with age: 0·6 (0·0 to 0·13) in the age group 0-4 years, 0·5 (0·3 to 0·7) at 5-9 years, 2·2 (1·8 to 2·6) at 10-14 years, and 3·4 (2·9 to 3·9) at 15-19 years.**

***Conclusion*—Juvenile diabetes mellitus is fairly rare in sub-Saharan Africa. If environmental factors such as infection and material deprivation were important determinants of insulin dependent diabetes in Africans, as they may be in Europeans, much higher rates would have been expected unless genetic factors possibly exert a protective role. The eightfold greater incidence in African Americans than in Tanzanians may be related to greater genetic admixture in African Americans with people from countries in Europe with a high incidence.**

Introduction

The incidence of juvenile diabetes varies widely throughout the world, with higher rates in white populations than in most other ethnic groups.[1,2] Highest rates have been reported in Finland and lowest rates in China, Japan, and Korea. Incidences are also considered to decrease progressively from northern to equatorial latitudes.[2] Contrary to this trend a surprisingly high prevalence of 0·95 per 1000 children aged 7-14 years was found in Khartoum, Sudan.[3] Incidences, however, were not reported, and to our knowledge there are no published studies of incidence from sub-Saharan Africa. We describe the incidence and prevalence of known juvenile diabetes mellitus in Dar es Salaam, Tanzania, based on registry data collected prospectively over 10 years.

Patients and methods

Dar es Salaam is Tanzania's largest city with a population of 1·5 million. Most citizens are of African origin, but there are sizable minorities of Indian Asians, Arabs, and people of mixed race. In June 1981 a registry of all patients of indigenous African origin with newly diagnosed diabetes was begun in the diabetic clinic of Muhimbili Medical Centre, the city's largest government hospital. Most African patients with diagnosed diabetes are seen in this hospital. This study includes all such patients aged 19 years and under and resident in Dar es Salaam who were registered between 1 January 1982 and 31 December 1991.[4] All required insulin from the time of diagnosis.

To ensure completeness of ascertainment, doctors in the city's two major non-governmental hospitals were asked if they knew of any African patients with juvenile diabetes who were not known to us. They did not. We consider therefore that ascertainment of diagnosed juvenile diabetes was as complete as possible.

In the calculation of incidence data from the 1978 and 1988 national censuses were used. The number of children in age groups 0-4, 5-9, 10-14, and 15-19 years were noted and the difference in population numbers in each age group between 1978 and 1988 calculated. From this the annual rate of increase in the population was estimated and assumed to be the same each year. We thus derived population figures for 1982 and 1991 and used the mean of these two figures as denominator.

The estimated mean numbers of subjects in the age groups 0-4, 5-9, 10-14 and 15-19 years during the years 1982 to 1991 were 158 272, 142 939, 129 719, and 146 656, respectively (total 576 586). All 95% confidence intervals for the incidences are based on the Poisson distribution.

Results

During the 10 years of 1982-91 inclusive 86 children and teenagers (45 males, 41 females) between the ages of 0 and 19 years and resident in Dar es Salaam were seen. Table I shows the number of patients seen in each age group each year. Of the 86 patients, 63 were seen in the first five years and 23 in the second five years. Only one patient below the age of 5 years was seen during the 10 years. The average crude annual incidence of diagnosed diabetes over the 10 year period for both sexes aged 0 to 19 years was 1·5 per 100 000 population (95% confidence interval 1·3 to 1·7). Incidence per

TABLE I—*Numbers of patients with newly diagnosed juvenile diabetes seen between 1 January 1982 and 31 December 1991 in Dar es Salaam, Tanzania*

	Age (years)				
Year	0·4	5·9	10·14	15·19	0·19
1982		1	3	7	11
1983			2	9	11
1984	1	1	2	6	10
1985			4	5	9
1986		3	7	12	22
1987			2		2
1988			1	4	5
1989			1	3	4
1990		1	2	2	5
1991		1	4	2	7
Total	1	7	28	50	86

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