Cigarette smoking and blood pressure in a worker population: a cross-sectional study

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Background Cigarette smoking has been reported to cause an acute increase in blood pressure (BP). Nevertheless, many epidemiological studies have found lower average BP values in smokers than in non-smokers. The aim of this study was to evaluate the possible existence of a systematic difference in BP values between smokers and non-smokers in a worker population.

Methods We studied 7109 employees of a metallurgical factory, all men, aged 18–60 years, 3237 non-smokers and 3872 smokers; of the latter, 816 smoked less than 10 cigarettes per day (light smokers), the others smoked 10 or more cigarettes per day. Clinical examination included measures of resting BP (by mercury sphygmomanometer), heart rate (HR) (by pulse palpation), body weight and height. Data were adjusted for age and body mass index (BMI). Four age groups (18–30, >30, >40 and >50 years) and 3 BMI groups (<25, 25–30, >30) were considered.

Results In smokers, the adjusted values of systolic BP (SBP) and HR (127.72 mmHg and 75.16 beats/min, respectively) were slightly but significantly higher than in nonsmokers (127.1 mmHg, P < 0.05 and 72.64 beats/min, P < 0.001), whereas diastolic BP (DBP) was significantly lower (83.37 versus 84.31 mmHg, P < 0.001). Considering the amount of cigarettes smoked, the mean BP values of light smokers were not significantly different from those of subjects smoking 10 or more cigarettes per day, whereas HR mean values were significantly higher in the latter. The prevalence of hypertension (WHO criteria) was similar in smokers and non-smokers in each age group.

Conclusions Our data showed slightly but statistically higher SBP and HR, and lower DBP mean values in smokers than in non-smokers; however, the differences in BP, although significant from the statistical point of view, were not of actual clinical significance.

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Introduction

Cigarette smoking has been reported to produce an acute increase in blood pressure (BP) with a peak elevation ranging from 3 to 12 mmHg systolic and from 5 to 10 mmHg diastolic, and a 20-30 min duration of effects [1-6]. Such acute BP elevation has been attributed to a nicotine-mediated activation of the sympathetic nervous system with local and systemic release of catecholamines and, possibly, release of vasopressin [7-10].

Paradoxically, many epidemiological studies have found lower average systolic and diastolic BP values in smokers than in non-smokers [11-25]. A dose-response relationship of lower BP with a greater number of cigarettes smoked per day has been observed by some authors [5,16,21] but not by others [19,23]. Also, the results of epidemiological studies on the effects of cessation of smoking on BP are equivocal, with some studies showing a rise in BP [14] and others showing no change [26-29].

The mechanisms of the observed negative relationship between cigarette smoking and BP remain essentially unknown. Possible explanations include: a) a 'withdrawal phenomenon': BP rises during the act of smoking and might drop below baseline during abstinence; since in epidemiological studies, BP is generally measured after the subjects have refrained from smoking for some time, the values obtained might not represent the subjects' usual BP [23,30,31]; b) a 'training effect', related to the heart rate (HR) acceleration seen with regular cigarette smoking and myocardial dysfunction with impaired contractility and reduced cardiac output [23]; recent evidence implicating cigarette smoking as a factor in cardiomyopathy and ventricular wall abnormalities may partly explain its dampening effect on BP [32]; c) the influence of confounding variables (age, relative weight, family history of hypertension, etc.) and possible sampling and measurements bias. Some authors have suggested that smokers with hypertension die younger than non-smokers, leaving a population of smokers with lower than average BP [16]. Recently it was shown that concentrations of serum cotinine (a metabolite of nicotine) are inversely related to BP, tending to confirm a negative association between smoking and BP [33]. Cotinine may contribute to a reduction of BP in smokers through a relaxation of vascular smooth muscle and dilation of peripheral blood vessels. Whatever the mechanisms, some authors have suggested that the difference in BP between smokers and non-smokers is small and of no real physiological or clinical significance [16,19]. However, it is of considerable importance to define whether cigarette smoking can be considered a risk factor for hypertension and whether or not it has a negative significant effect on BP. The aim of this cross-sectional study was to evaluate the possible existence of systematic difference in BP values and hypertension prevalence between smokers and non-smokers in a worker population.

Methods

We studied 7109 subjects, all men, aged 18-60 years, all belonging to the same working community (Agusta SpA), in which there was an established work-site hypertension detection and treatment programme. They represented 94% of the entire number of employees, who agreed to be screened. Clinical examination included measures of resting BP and HR, body weight and height, as well as a detailed medical and personal habits history. On the visit morning, between 8.00 and 9.00 a.m., the subjects arrived, having fasted from the preceding evening and having refrained from cigarette smoking and from drinking caffeine-containing beverages during this period. BP was measured by trained physicians using a standard mercury sphygmomanometer (Korotkoff phases I and V) with a cuff size appropriate to individual body habitus. Measurements were taken on the right arm in the seated position, after the subject had rested for 5 min. Three readings were taken and the mean value was reported. Subjects were classified as hypertensive if they were receiving antihypertensive treatment and/or had a DBP of 95 mmHg or greater and/or a SBP of 160 mmHg or greater. HR was determined by pulse palpation for 30 s. Body weight and height were measured with the subjects in light clothes only and barefoot according to the WHO manual [34].

Smokers were defined as those who had smoked cigarettes within the past year. Cigarette consumption was reported as less than 10 cigarettes per day or 10 or more cigarettes per day. Since body mass has been found to be strongly related to BP and smoking habit, the analyses were adjusted for this variable. Body mass index [BMI = weight in kg/(height in m)²] was used as the measure of body mass. According to BMI, three groups were identified: normal weight (BMI < 25), overweight (BMI 25-30) and obese (BMI > 30). To examine any interaction with age, the data were analysed in four age groups: 18-30, 31-40, 41-50 and 61-60 years.

Continuous variables were compared using an analysis of variance model extracting smoking effects: this effect was adjusted by subjects' age and BMI. The adjustment was also calculated in each stratum (for instance, in the age group 30-40 years, SBP was adjusted by the actual age of the subjects and their BMI). For the calculations we used CLM SAS 6.06 procedures with LSMEANS options; the values have been expressed as means \pm SEM. For discrete variables (e.g. number of subjects with hypertension) we used the χ^2 -test or Fisher's exact test, if the number of observations was large. Observed differences in data were considered significant if P < 0.05.

Results

Of the 7109 participants, 3237 (45.5%) were non-smokers and 3872 (55.5%) were smokers. The two groups were

Table 1 Distribution of smokers and non-smokers by age and body mass index

	Non-smokers (n = 3237)	Smokers (n = 3872)	All subjects (n = 7109)
Age			
18–30	647	562	1209
31–40	1319	1714	3033
41–50	972	1255	2227
5160	299	341	640
Body mass index			
Normal weight	1851	1967	3818
18–30	510	389	899
31-40	796	905	1701
4150	443	550	993
51–60	102	123	225
Overweight	1162	1593	2755
18–30	124	151	275
31-40	438	685	1123
41–50	448	582	1030
51-60	152	175	327
Obese	224	312	536
18–30	13	22	35
31–40	85	124	209
41-50	81	123	204
51-60	45	43	88

comparable with respect to their distribution by age and BMI (Table 1) as well as alcohol consumption, social status and exercise level. Among smokers, 816 reported smoking less than 10 cigarettes/day, whereas 3056 smoked 10 or more cigarettes/day. Table 2 shows the mean values of SBP, DBP and HR in smokers and non-smokers before and after correction for age and BMI. In smokers, uncorrected SBP was significantly higher than in non-smokers (128.05 versus 126.72 mmHg, P < 0.005) as well as HR (75.17 versus 72.659.47 beats/min, P < 0.001), whereas DBP was significantly lower (83.6 versus 84.04 mmHg, P < 0.001). After adjustment, such differences in SBP, DBP and HR persisted, SBP and HR being significantly higher and DBP lower in smokers, although the difference in SBP was rather small in absolute values (127.72 mmHg in smokers versus 127.01 mmHg in non-smokers, P = 0.0385).

Considering the adjusted data in the four age groups (Table 3), no significant difference in SBP was observed between smokers and non-smokers in any group, DBP was significantly lower in smokers aged 18-30, 31-40 and 51-60 years and HR was always higher in smokers of each age group.

Table 2 Systolic blood pressure, diastolic blood pressure and heart rate in smokers and non-smokers before and after correction for age and body mass index

	Non-smokers (n = 3237)	Smokers (n = 3872)	Ρ
Systolic blood pressure	126.62 ± 14.84	128.05 ± 15.46	0.039
After correction	127.01 ± 0.25	127.72 ± 0.22	0.0385
Diastolic blood pressure	84.04 ± 9.36	83.60 ± 9.52	0.001
After correction	84.31 ± 0.15	83.37 ± 0.14	0.0001
Heart rate	72.65 ± 9.47	75.17 ± 9.52	0.0001
After correction	72.64 ± 0.16	75.16 ± 0.15	0.0001

Values are expressed as means \pm SD before and means \pm SEM after correction.

Table 3 Values of systolic blood pressure, diastolic blood pressure and heart rate (means \pm SEM), adjusted for age and body mass index, in smokers and non-smokers stratified by age

Age	Systolic blood pressure		Diastolic blood pressure		Heart rate	
	Non-smokers	Smokers	Non-smokers	Smokers	Non-smokers	Smokers
18–30	122.03 ± 0.48	122.00 ± 0.52	80.06 ± 0.31	78.21 ± 0.34***	72.64 ± 0.38	74.18 ± 0.40**
31-40	125.14 ± 0.36	125.89 ± 0.31	83.77 ± 0.23	82.83 ± 0.20**	72.93 ± 0.26	75.33 ± 0.23***
41–50	129.09 ± 0.48	130.33 ± 0.42	85.84 ± 0.28	85.58 ± 0.25	72.38 ± 0.29	75.34 ± 0.26***
5160	137.41 + 1.07	138.57 + 1.01	89.74 + 0.61	87.53 + 0.53**	72.12 + 0.54	75.37 + 0.50***

***P* < 0.01, *** *P* < 0.001.

Table 4 Adjusted values of systolic blood pressure, diastolic blood pressure and heart rate (means \pm SEM) in normal weight, overweight and obese smokers and non-smokers stratified by age

Age	Norma	Normal weight		Overweight		Obese	
	Non-smokers	Smokers	Non-smokers	Smokers	Non-smokers	Smokers	
Systolic blood	pressure						
18–30	120.85 ± 0.52	119.93 ± 0.60	125.62 ± 1.15	126.46 ± 1.04	121.59 ± 4.50	135,19 ± 3,45*	
31–40	123.10 ± 0.46	123.94 ± 0.43	126.92 ± 0.62	127.44 ± 0.49	131.69 ± 1.59	133.89 ± 1,32	
4150	126.76 ± 0.70	127.45 ± 0.63	129.68 ± 0.71	131.91 ± 0.62*	137.46 ± 1.87	136.38 ± 1.51	
51–60	132.80 ± 1.63	136.02 ± 1.49	137.92 ± 1.47	139.31 ± 1.38	146.51 ± 3.28	142.36 ± 3.36	
Diastolic bloo	d pressure						
18–30	79.15 ± 0.35	76.99 ± 0.40***	82.31 ± 0.74	80.95 ± 0.67	83.83 ± 2.25	87.28 ± 1.72	
31–40	82.20 ± 0.29	80.88 ± 0.27 ⁺	85.04 ± 0.40	84.43 ± 0.32	89.21 ± 1.05	90.11 ± 0.87	
41–50	84.06 ± 0.42	83.60 ± 0.37 [‡]	86.41 ± 0.41	86.67 ± 0.36	92.02 ± 1.15	89.52 ± 0.93	
5160	86.41 ± 0.94	85.62 ± 0.86	91.13 ± 0.79	87.53 ± 0.73	92.97 ± 1.71	92.46 ± 1.75	
Heart rate							
1830	73.02 ± 0.41	74.04 ± 0.47	70.82 ± 0.91	74.12 ± 0.82**	75.18 ± 3.86	77.07 ± 2.96	
31–40	72.78 ± 0.34	75.43 ± 0.32***	72.91 ± 0.44	75.23 ± 0.35***	74.27 ± 1.10	75.24 ± 0.91	
41-50	72.23 ± 0.43	75.30 ± 0.39***	71.96 ± 0.43	75.44 ± 0.38***	75.81 ± 1.10	74.88 ± 0.89	
5160	70.64 + 0.92	75.71 ± 0.85***	72.83 + 0.73	75.05 + 0.68**	73.30 + 1.59	75.46 + 1.62	

P* < 0.05, *P* < 0.01, *** *P* < 0.001.

Table 4 shows the adjusted values of BP and HR in normal weight, overweight and obese smokers and non-smokers stratified by age. Among normal weight subjects, no significant difference in SBP values was observed between smokers and non-smokers in any age group, DBP was significantly lower in smokers aged 18–30, 31–40 and 41–50 years, whereas no difference was observed in the older age group and HR was higher in smokers from age 30 years onwards. Among overweight subjects, no significant difference in SBP and DBP values was observed between smokers and non-smokers in any age group, with the exception of the 41–50 year old group, which showed higher SBP in smokers;

 $_{\text{Table 5}}$ Adjusted values of systolic blood pressure, diastolic blood pressure and heart (means \pm SEM) in subjects smoking less, than 10 cigarettes daily and in those smoking 10 or more cigarettes per day

	Cigarettes smoked < 10 per day	Cigarettes smoked > 10 per day	Ρ
Systolic blood pressure	127.39 ± 0.41	128.35 ± 0.28	0.0574
Diastolic blood pressure Heart rate	83.95 ± 0.25 73.91 ± 0.27	83.42 ± 0.17 75.75 ± 0.18	0.0850 0.0001

Table 6 Prevalence of hypertension (percentage) among smokers and non-smokers; in parentheses are reported the percentages of treated hypertensives.

Age (years)	Non-smokers	Smokers	P
18–30	3.86% (1.0%)	2.85% (0.8%)	NS
31-40	8.19% (3.4%)	7.93% (3.0%)	NS
41–50	16.87% (6.7%)	14.58% (5.8%)	NS
51–60	30.10% (12.0%)	26.10% (11.0%)	NS

HR was always higher in smokers. Among obese subjects, no difference in DBP or HR was observed between smokers and non-smokers in any age group, whereas SBP was significantly higher in smokers aged 18–30 years.

Considering the amount of cigarettes smoked, the mean SBP and DBP values of subjects smoking less than 10 cigarettes per day were not significantly different from those of subjects smoking 10 or more cigarettes per day (Table 5), although a rather small tendency towards higher SBP values was seen in the latter (128.35 versus 127.39 mmHg, P = 0.574). On the other hand, HR mean values were significantly higher in subjects smoking 10 or more cigarettes per day. The prevalence of hypertension was similar in smokers and nonsmokers in each age group (Table 6).

Discussion

The results of this study showed slightly higher SBP and lower DBP (i.e. higher pulse pressure), in smokers than in non-smokers. Such differences remained substantially unchanged after adjusting for age and body weight. These findings, which seem to indicate that in smokers the vascular tree becomes less compliant, are in agreement with most epidemiological trials with respect to DBP, but not SBP data [11-25]. The latter resemble some observations obtained from ambulatory BP monitoring, showing higher SBP values in smokers than in non-smokers, at least during daytime [35]. The large number of subjects studied and the use of appropriate statistical methods for epidemiological investigations, with proper controls for relevant confounding factors, such as age and BMI, might offer an explanation for the discrepancy between our findings and those of most epidemiological trials. However, it must be emphasized that the differences in both SBP and DBP values between smokers and non-smokers, although statistically significant (probably due to the numerosity of the case-record), are small (+0.71 mmHg and -0.94 mmHg) and not of real clinical significance. Furthermore, in age-specific groups, the differences in SBP between smokers and non-smokers are not statistically significant.

It is of interest that the subject being overweight and, even more so, obese, appears to nullify the differences in both SBP and DBP values between smokers and non-smokers, which seems to indicate a major influence of body weight on BP.

HR mean values were significantly faster in smokers than in non-smokers, which confirms previous reports [11-25]. It has been demonstrated that cigarette smoking causes longterm reduction in vagal cardiac control and blunts postural responses in autonomic cardiac regulation, which may explain, respectively, the higher HR in smokers and their lower average BP, found mainly when it is measured in the sitting position [36]. The reduction in vagal control adds one more mechanism to the well-known association between smoking and cardiac death, as many studies have shown that patients with decreased vagal cardiac control have increased susceptibility to sudden coronary death [37] and increased mortality after acute myocardial infarction [38].

Regarding the number of cigarettes smoked, our findings did not confirm the significant dose-response relationship reported by other authors, which observed lower BP at increasing levels of cigarette consumption [5,16,21]. In our study, the mean BP values of subjects smoking less than 10 cigarettes per day were not significantly different from those of subjects smoking 10 cigarettes per day or more. On the other hand, HR mean values were significantly higher in heavy than in light smokers, which indicates a dose-response relationship between smoking and HR.

The prevalence of hypertension was similar in smokers and non-smokers in each age group, which is in agreement with other reports [23,26]. It has been suggested that smokers with hypertension previously stopped smoking on medical advice, which would tend to reduce the prevalence of hypertension among smokers relative to non-smokers, but it does not seem that this bias plays an important role [23].

Although the complex relationship between cigarette smoking and BP deserves further study, particularly longitudinal studies, according to our data there is no difference of actual clinical significance in BP values or in hypertension prevalence between smokers and non-smokers.

The failure of our and other cross-sectional epidemiological studies to demonstrate an association between cigarette

smoking and BP should not detract from the well-known synergistic effect of smoking and hypertension on the atherosclerotic process. Besides, it should be stressed that, although cessation of cigarette smoking does not seem to reduce BP values, it has repeatedly been found to result in a reduced mortality rate for CHD. Therefore, the attempts to encourage patients to eliminate this major risk factor remain of central importance, although they must not delay the initiation of drug therapy or other measures for the treatment of hypertension.

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