

Effects of Smoking Cessation on Changes in Blood Pressure and Incidence of Hypertension

A 4-Year Follow-Up Study

Duk-Hee Lee, Myung-Hwa Ha, Jang-Rak Kim, David R. Jacobs, Jr

Abstract—We performed the present study to investigate the effects of smoking cessation on changes in blood pressure and incidence of hypertension. We evaluated 8170 healthy male employees at a steel manufacturing company who had received occupational health examinations at the company's health care center in 1994 and were reexamined in 1998. Adjustment covariates were the baseline age, body mass index, cigarette smoking, alcohol consumption, exercise, family history of hypertension, systolic or diastolic blood pressure, and changes in body mass index and alcohol consumption during the follow-up period. The adjusted relative risks of hypertension in those who had quit smoking for <1, 1 to 3, and ≥ 3 years were 0.6 (95% CI 0.2 to 1.9), 1.5 (95% CI 0.8 to 2.8), and 3.5 (95% CI 1.7 to 7.4), respectively, compared with current smokers. The trends for increased risk of hypertension for longer periods of smoking cessation were observed in subgroups of those who maintained weight as well as those who gained weight after smoking cessation. The adjusted increments in both systolic and diastolic blood pressure were higher in those who had quit for ≥ 1 year than in current smokers. These trends among weight losers, as well as gainers and maintainers, were similar. We observed progressive increases in blood pressure with the prolongation of cessation in men, although at this time the mechanism remains unknown and must be clarified. This study implies that the cessation of smoking may result in increases in blood pressure, hypertension, or both. (*Hypertension*. 2001;37:194-198.)

Key Words: smoking ■ blood pressure ■ hypertension ■ body weight

Some epidemiological studies have reported lower blood pressure (BP) in smokers than in nonsmokers.¹ This finding is regarded as a paradox, because nicotine has potent sympathomimetic effects, which affects BP levels and heart rate.² Furthermore, ex-smokers tend to have BPs similar to those of people who never smoked.^{3,4} The lower average BP found in smokers has occasionally been attributed to differences in relative weight. However, BP differences among smokers and nonsmokers tend to persist even after body weight is controlled.⁴ The results of follow-up studies on the effects of smoking^{5,6} or the cessation of smoking⁷⁻⁹ on the changes of BP are equivocal.

The aim of the present study was to investigate the effect of smoking cessation on the changes of BP and incidence of hypertension in male workers at a steel manufacturing company in the Republic of Korea. Since 1994, this company has campaigned actively for smoking cessation. In addition, we explored BP change according to the duration of smoking cessation and investigated whether the relationship was influenced by changes in weight.

Methods

Study Population

All workers at this steel manufacturing company were required to receive an annual health check-up, which consisted of clinical and laboratory measurements. Throughout 1994, a health check-up was performed between 9 AM and 12 noon, after an overnight fast, in a healthcare center located in the factory. Male workers between 25 and 50 years old without definite hypertension (systolic BP [SBP] ≥ 160 mm Hg, diastolic BP [DBP] ≥ 95 mm Hg, or on antihypertensive medication) were eligible for follow-up in this study. Of the 12 617 men who met these criteria, 9302 (73.7% follow-up rate) were reexamined in 1998. To avoid the inclusion of cases of mild hypertension, 851 subjects with baseline levels of SBP between 140 and <160 mm Hg or of DBP between 90 and <95 mm Hg were excluded from the study. In addition, 192 men with hypercholesterolemia, diabetes mellitus, other known cardiovascular diseases, and other diseases that required continuous medication were excluded. We also excluded 89 employees who provided incomplete or inconsistent information. After all of these exclusions, 8170 men were included in the analysis.

Measurements

Information on lifestyle factors, including cigarette smoking, alcohol consumption, and exercise; medical history; and family history of hypertension were obtained primarily through self-report question-

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TABLE 1. Baseline Characteristics of the Cohort With Respect to the Smoking Habit During the Follow-Up Periods in 8170 Male Workers

Characteristic	Current Smoker (n=5372)	Current Nonsmoker (n=2090)	Quitter (n=708)	P
SBP, mm Hg	114.7±10.1	115.5±9.9	114.9±10.1	0.012†
DBP, mm Hg	72.4±6.7	73.3±6.6	72.8±6.6	<0.000†
Age, y	34.3±5.8	35.3±6.1	35.8±5.7	<0.000†‡
BMI, kg/m ²	22.5±2.3	22.6±2.3	22.5±2.2	0.131
Alcohol consumption, g/wk	123.7±123.0	77.4±94.6	112.1±114.6	<0.000†‡§
Exercise, times/wk	0.76±0.63	0.80±0.61	0.76±0.61	0.066
Smoking amount, cigarettes/d	22.7±12.2	...	21.1±11.9	0.001
Duration of smoking, y	15.4±6.4	...	15.9±6.5	0.048
Family history of hypertension, n (%)				
Yes	195 (3.6)	78 (3.7)	24 (3.4)	0.915
No	5177 (96.4)	2012 (96.3)	684 (96.6)	...

*P values were based on 1-way ANOVA, unpaired t test, or χ^2 test. Statistically significant ($P<0.05$) by Tukey's multiple comparison: †nonsmokers vs smokers, ‡nonsmokers vs quitters, and §smokers vs quitters. Values are mean±SD.

naires. Each year, all workers were asked to fill out the same, or slightly modified, questionnaire. For each questionnaire, changes spotted by a computerized data system (which contained information from previous years) were confirmed by a nurse in a direct interview. Data on smoking included the number of cigarettes smoked per day. Nonsmokers were asked whether they had previously smoked, how many cigarettes per day they had smoked, and the approximate date of quitting. SBP and DBP were recorded with an automatic device (TM-2650A; A&D Company) in the sitting position after the subjects rested on a chair for ≥5 minutes. For employees with SBP of ≥160 mm Hg or DBP of ≥95 mm Hg, BP was measured again with an ordinary sphygmomanometer by an experienced nurse after an additional 5 minutes of rest. SBP and DBP were determined with the first and fifth Korotkoff sounds. Individuals with hypertension were those who had an SBP of ≥160 mm Hg or a DBP of ≥95 mm Hg in both measurements. Those on antihypertensive medication were also considered hypertensive. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m²).

Statistical Analysis

The relationship between smoking cessation and change in BP and the incidence of hypertension were studied by ANCOVA and

multiple logistic model with the Statistical Analysis System (SAS Institute), version 6.12. Tukey's method was used for comparisons among groups. Subjects were placed into one of the following 3 classes with respect to smoking habits. The numbers of subjects in each class are shown in parentheses: (1) current smokers (5372), who were subjects with a history of cigarette smoking at entry and during follow-up (5356), including attempted quitters and subjects without prior history of smoking who began to smoke during the follow-up period (16); (2) current nonsmokers (2090) were subjects with no history of smoking at baseline and during the follow-up periods (1563) or with a history of cigarette smoking who did not smoke at the time of entry and during the follow-up period (527); and (3) quitters (708) were subjects who were smoking at the beginning of the study but stopped smoking during the follow-up period and maintained smoking cessation until the reexamination in 1998. Quitters were further classified according to their particular length of smoking cessation: those who stopped smoking for <1 year, those who stopped smoking for >1 year but <3 years, and those who stopped smoking for ≥3 years.

Covariates included in the multivariate analysis were the baseline age, BMI, cigarette smoking (pack-years), alcohol consumption (grams per week), exercise (times per week), family history of

TABLE 2. Changes From Baseline in SBP and DBP With Respect to Smoking Habit During the Follow-Up Periods Among 8170 Male Workers

	Smoking Habit (sample size)					P*	Adjusted R ²
	Current Smoker (n=5372)	Current Nonsmoker (n=2090)	Quitter (n=708)				
			<1 y (n=210)	1-3 y (n=374)	≥3 y (n=124)		
Crude							
SBP	3.9 (3.6-4.2)	4.4 (3.9-4.8)	4.4 (2.8-5.9)	6.2† (5.1-7.4)	6.1 (4.1-8.2)	<0.000	...
DBP	3.1 (2.9-3.3)	3.3 (3.0-3.6)	3.7 (2.7-4.7)	4.8† (3.9-5.7)	4.1 (2.6-5.6)	<0.000	...
Adjusted‡							
SBP	3.8 (3.5-4.0)	4.9† (4.4-5.3)	3.6 (2.3-4.8)	5.2† (4.3-6.2)	5.6 (3.9-7.2)	<0.000	28.9%
DBP	2.9 (2.7-3.1)	3.8† (3.5-4.2)	3.1 (2.2-4.1)	4.1† (3.4-4.8)	4.2 (2.9-5.4)	<0.000	22.7%

*P values were based on 1-way ANOVA or ANCOVA.

†Statistically significant ($P<0.05$) compared with the current smokers by Tukey's multiple comparison.

‡Adjusted for the baseline age, BMI, smoking (pack-y), alcohol consumption (g/wk), exercise (times/wk), family history of hypertension, SBP or DBP (baseline for the dependent variable), and changes in BMI and alcohol consumption during the follow-up period.

Values are mean (95% CI).

TABLE 3. Relative Risks for the Incidence of Hypertension With Respect to Smoking Habit During the Follow-Up Periods in 8170 Male Workers

	Smoking Habit (cases/population)				
	Current Smoker (96/5372)	Current Nonsmoker (48/2090)	Quitter		
			<1 y (3/210)	1–3 y (12/374)	≥3 y (10/124)
Crude	1.0	1.3 (0.9–1.8)	0.8 (0.3–2.5)	1.8 (1.0–3.4)	4.8 (2.4–9.5)
Adjusted*	1.0	0.9 (0.6–1.5)	0.6 (0.2–1.9)	1.5 (0.8–2.8)	3.5 (1.7–7.4)

*Adjusted for the baseline age, BMI, smoking (pack-y), alcohol consumption (g/wk), exercise (times/wk), family history of hypertension, SBP or DBP (baseline for the dependent variable), and changes in BMI and alcohol consumption during the follow-up period.

Values are relative risks (95% CIs).

hypertension, SBP or DBP (baseline for the dependent variable), and changes in BMI and alcohol consumption during the follow-up period. To determine whether weight change modified the association between smoking cessation and BP, we performed stratified analyses based on weight changes during 4 years. These individuals were classified as (1) weight loser (weight decrease of ≥ 1 kg), (2) weight maintainer (weight change of < 1 kg), or (3) weight gainer (weight increase of ≥ 1 kg). All probability values quoted are 2-sided, and $P < 0.05$ is regarded as statistically significant.

Results

Baseline Characteristics

At the beginning of the study, the SBP ($P = 0.012$) and DBP ($P < 0.000$) were significantly different between current smokers and current nonsmokers (Table 1). The variables that were significantly different between smokers and quitters were age ($P < 0.000$), alcohol consumption ($P < 0.000$), smoking amount ($P = 0.001$), and duration of smoking ($P = 0.048$). The quitters were significantly older and drank less than the smokers. Although the quitters smoked fewer cigarettes per day than smokers, they had smoked for a longer duration.

Changes From Baseline in BP

In adjusted as well as crude analyses, the increments in both SBP and DBP were significantly different among groups (Table 2). When the quitters were divided into 3 groups according to the duration of smoking cessation, linear trends of increments in both SBP and DBP, in relation to years of smoking cessation, were observed. Compared with current

smokers, the current nonsmokers and quitters for 1 to 3 years showed statistically larger increases in BP, and the quitters for ≥ 3 years showed the largest increase in BP in adjusted analyses. The adjusted changes in BP in the quitters of < 1 year were similar to or even smaller than those of the current smokers. The coefficients of determination for our final models were 28.9% for the dependent variable SBP and 22.7% for the dependent variable DBP.

Incidence of Hypertension

During the 4-year period, 169 of the 8170 workers (2.1%) became hypertensive: 48 of the current nonsmokers (2.3%), 96 of the current smokers (1.8%), and 25 of the quitters (3.5%) (Table 3). Compared with the current smokers, the crude relative risks were 1.3 (95% CI 0.9 to 1.8) in the current nonsmokers, 0.8 (95% CI 0.3 to 2.5) in the quitters for < 1 year, 1.8 (95% CI 1.0 to 3.4) in the quitters for 1 to 3 years, and 4.8 (95% CI 2.4 to 9.5) in the quitters for ≥ 3 years. The adjusted relative risks of the quitters for < 1 , 1 to 3, and ≥ 3 years also showed a dose-response relationship: 0.6 (95% CI 0.2 to 1.9), 1.5 (95% CI 0.8 to 2.8), and 3.5 (95% CI 1.7 to 7.4), respectively.

Stratified Analysis by Weight Change

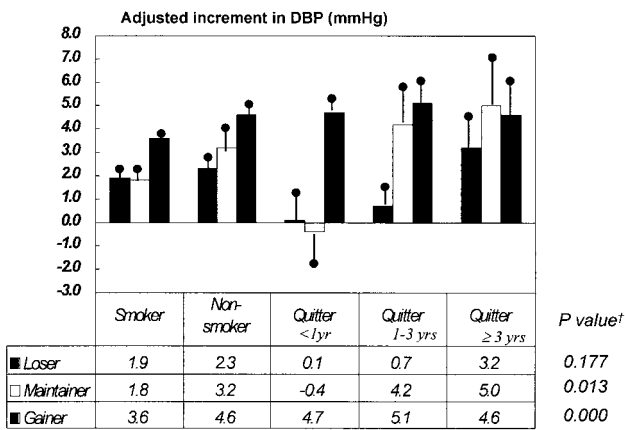
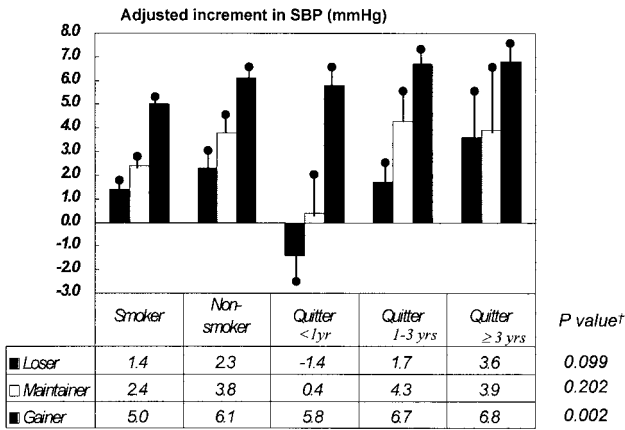
The mean weight change was -2.3 kg in weight losers, 0 kg in weight maintainers, and $+3.2$ kg in weight gainers (Figure, Table 4). Among those who quit smoking, 72.7% gained

TABLE 4. Adjusted Relative Risks for the Incidence of Hypertension After Stratification by the Changes of Weight During Follow-Up Periods in 8170 Male Workers

Category of Weight Change	Current Smoker	Current Nonsmoker	Quitter		
			<1 y	1–3 y	≥3 y
Gainer	1.0	1.2 (0.7–2.2)	0.7 (0.2–2.4)	1.6 (0.8–3.4)	3.1 (1.3–7.5)
	(62/3302)	(32/1275)	(3/144)	(11/279)	(7/93)
Maintainer	1.0	1.3 (0.3–6.0)	...	2.7 (0.3–26.7)	40.4 (4.2–385.9)
	(8/699)	(5/275)	(0/26)	(1/33)	(2/12)
Loser	1.0	0.3 (0.1–0.9)	1.4 (0.1–14.8)
	(26/1371)	(11/540)	(0/40)	(0/62)	(1/19)

Adjusted for the baseline age, BMI, smoking (pack-y), alcohol consumption (g/wk), exercise (times/wk), family history of hypertension, SBP or DBP (baseline for the dependent variable), and changes in BMI and alcohol consumption during the follow-up period.

Values are relative risks (95% CIs) with numbers of cases per population given in parentheses.



Adjusted* increments and SEMs in SBP and DBP after stratification according to changes in weight during the follow-up periods in 8170 male workers. *Adjustments were made for the baseline age, BMI, smoking (pack-y), alcohol consumption (g/wk), exercise (times/wk), family history of hypertension, SBP or DBP (baseline for the dependent variable), and changes in BMI and alcohol consumption during the follow-up period. †P values were based on ANCOVA.

weight and 32.9% gained >4 kg. Among those who continued smoking, 61.6% gained weight and 20.8% gained >4 kg, similar to the current nonsmokers.

In all 3 weight change groups, increments of both SBP and DBP generally occurred with the increase in the duration of smoking cessation. The quitters for ≥3 years and current nonsmokers showed larger increases in BP than the current smokers in all groups. However, the quitters for <1 year among weight losers or maintainers showed a smaller increase than the current smokers.

Compared with the current smokers, the adjusted relative risks of incident hypertension in the quitters for ≥3 years were 3.1 (95% CI 1.3 to 7.5) in the weight gainers, 40.4 (95% CI 4.2 to 385.9) in the weight maintainers, and 1.4 (95% CI 0.1 to 14.8) in the weight losers. The incidence of hypertension in the quitters for 1 to 3 years were also higher than in the current smokers, as seen in the relative risks of 1.6 (95%

CI 0.8 to 3.4) for the weight gainers and 2.7 (95% CI 0.3 to 26.7) for the weight maintainers. The current nonsmokers had a significantly lower incidence than the current smokers only in the weight loser group; their relative risk was 0.3 (95% CI 0.1 to 0.9).

Discussion

This 4-year prospective study suggests that the increases in BP among the quitters and current nonsmokers, especially the quitters, were generally larger than those of the current smokers. More interestingly, the increments of BP in the quitters for <1 year were very similar to those of the current smokers. Quitters for ≥1 year, however, showed larger increases in BP than did the current smokers. This trend was observed consistently in all groups classified by weight changes, despite small numbers in these subgroups. Furthermore, the incidence of hypertension was also higher in the group of subjects who had stopped smoking for ≥1 year, whereas the incidence was lower in the quitters for <1 year. Relative risks increased in direct relationship with the increasing periods of smoking cessation. These relationships were consistently seen as trends in both weight maintainers and gainers, again despite small numbers at risk developing hypertension.

Although cigarette smoking has been reported to produce an acute rise in BP under laboratory conditions, 1 review¹ mentions a fairly consistent negative association between smoking and BP, and a dose-response relationship has been reported in several studies,^{4,10} with a lower BP observed at increasing levels of cigarette consumption. In addition, there are studies reporting an inverse relationship between cotinine, the major nicotine metabolite, and BP in smokers.^{11,12} However, follow-up studies have failed to come to a consensus regarding the role of cigarette smoking in the occurrence of hypertension in generally healthy persons.^{5,6}

Previous cross-sectional epidemiological findings have led to the prediction of an increase in BP as a consequence of smoking cessation. However, longitudinal studies^{3,7-9} did not show consistent results. The Normative Aging Study³ reported that greater increases in BP were observed in quitters than in those who continued to smoke. This also occurred in subgroups classified according to weight change during follow-up periods, thus being consistent with our results. However, other longitudinal studies, such as the Framingham study,⁷ the Evans County study,⁸ and the Israeli Cordis Study,⁹ did not show significant increases in BP in subjects after smoking cessation compared with subjects who continued to smoke.

In interpretation of the change in BP after smoking cessation, the influence of smoking cessation on other factors that can affect BP must be considered. These factors include an increase in total food intake and a subsequent increase in weight, and a reduction in alcohol intake.¹³ A similar trend was observed in this study; however, adjustment of these changes did not alter the relationship. Nevertheless, it is possible for the effect of obesity on BP to not be completely removed through statistical adjustment.⁶ In the present study, the quitters showed consistently larger increases in BP for all 3 weight change groups and higher incidences of hyperten-

sion in both weight maintainers and gainers. This suggests that our result did not come from an insufficient adjustment of body weight changes. The other possible change is that after giving up smoking, the subjects feel a relative increase in stress; many smokers report themselves to be calmer and more relaxed while smoking,¹⁴ and some studies have suggested that psychophysiological stress responsiveness was inhibited in habitual smokers compared with nonsmokers.^{11,15} Therefore, if smoking modifies the pressor response to other stressful environmental stimuli, the long-term outcome of stopping smoking might be an increase in BP, consistent with observed BP differences between smokers and nonsmokers in epidemiological studies.

It has been suggested that the lower BP found in smokers might be only a transient decline in BP. This resulted from the short time off smoking before the measurement of BP. To clarify this question, several studies were performed that measured ambulatory BP. Some have found that smokers have a higher BP,^{11,16} whereas others have reported similar¹⁷ or even lower^{18,19} BP in smokers. Among these studies, there are many discrepancies in the selection and characteristics of subjects. The withdrawal phenomenon could explain the larger BP increase in the quitters compared with the current smokers in the present study, but it does not seem to be an appropriate explanation for the result of a linear association between the duration of quitting and the increase in BP.

It is possible that our results were influenced by changes in other health-related behavior or that dietary habits we did not measure might modify the effects of smoking cessation. For example, we did not include coffee consumption as a possible confounder. We guess that the quitters may be more health conscious and tend to limit their coffee drinking. Coffee consumption may be modestly associated with higher BP.²⁰ In this scenario, the observed association between smoking cessation and BP change would likely be larger if we could adjust for coffee consumption. Hence, this is not a possible explanation of this study.

We excluded from the analyses mildly hypertensive persons at baseline. Because more mildly hypertensive persons tend to quit smoking and because they more easily become definite hypertensives, they could have distorted the association in our study. However, inclusion of the mildly hypertensives in the analyses caused little change in our results (data not shown). It should also be stressed that the present study was conducted among healthy male workers only and should be replicated among women before any generalizations can be made. Several studies reported that cigarette smoking might differently affect the hemodynamic responses of men and women.²¹ In addition, the use of a single reading of BP in our study may have served as a drawback. A single reading is generally considered inadequate to determine an individual's usual BP level because of large random fluctuations in casual readings. However, although random errors due to single determinations weaken the association, they should not cause a spurious association. Moreover, our diagnosis of hypertension was based on 2 measurements of BP.

In summary, the findings of the present study imply that smoking cessation itself may result in increasing BP, even hypertension, in men through an unknown mechanism that needs to be clarified. However, this finding should not distract from the well-known harmful effects of cigarette smoking, nor should it encourage smoking.

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