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Changes in blood pressure and body weight following smoking cessation in women

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Objective. Few have studied the long-term effects of smoking and smoking cessation on weight gain and blood pressure increase and compared with the age-related increases experienced by most adults. This study compared the development of weight and blood pressure in female never smokers, continuing smokers and smokers who quit smoking.

Design. Weight, systolic (SBP) and diastolic (DBP) blood pressure and smoking habits were assessed at baseline and re-assessed after a mean follow-up of 9.0 ± 5.8 years.


Subjects. A total of 2381 female never smokers and 1550 female smokers. At the re-examination, 388 of the smokers had quit smoking.

Results. Mean weight gain was 7.6 ± 6.1, 3.2 ± 5.8 and 3.7 ± 5.2 kg, respectively, in quitters, continuing smokers and never smokers (P < 0.001). In women without blood pressure treatment, mean SBP increase was 20.9 ± 16.8, 19.1 ± 15.8 and 16.1 ± 16.3 mmHg, respectively, in these groups (P < 0.001). Mean DBP increase was 6.2 ± 8.7, 5.7 ± 9.3 and 3.1 ± 8.0 mmHg, respectively (P < 0.001). After adjustments for potential confounders, the increased weight gain in quitters remained highly significant. The differences in SBP and DBP increase were attenuated after adjustments, but remained significant. Incidence of hypertension (≥ 160/95 mmHg or treatment) was significantly higher in quitters [adjusted odds ratio (OR): 1.8; CI: 1.4–2.5] when compared with continuing smokers (OR: 1.3; CI: 1.07–1.6) and never smokers (reference).

Conclusion. Over a long follow-up, weight gain was approximately 3–4 kg higher in quitters when compared with continuing smokers or never smokers. Although the differences in blood pressure increase were moderate, smoking cessation was associated with an increased incidence of hypertension.

Keywords: age, blood pressure, longitudinal, smoking cessation, weight gain, women.

Introduction

Although smoking is associated with an increased blood pressure during the acute phase [1, 2], many cross-sectional studies have reported lower blood pressure in smokers when compared with former smokers [1]. However, to what extent the increased blood pressure could be explained by the weight gain experienced by many former smokers is unclear [1, 3]. Furthermore, the follow-up period of most studies have been relatively short, and few have studied how blood pressure increases in smokers, quitters and life-long never smokers over several years of follow-up.

In Sweden, the prevalence of smoking is now higher in women than in men [4]. Fear of weight gain is a major obstacle for women who want to quit smoking. Although many women who stop smoking report that they gain weight, it is difficult to distinguish the effects of smoking cessation from the age-related weight increase experienced by a majority of the middle-aged population.
The present study explored how weight and blood pressure increased over a mean follow-up of 9 years in life-long never smokers, continuing smokers and smokers who quit smoking.

**Methods**

**Baseline cohort**

Between 1977 and 1991, 10,902 women (participation rate 71%) were examined as a part of a screening programme for early detection and treatment of cardiovascular risk individuals at the Department of Preventive Medicine in Malmö, Sweden [5]. A total of 3830 were smokers and 4937 were never smokers. Women who had stopped smoking before the baseline examination \( (n = 2082) \) or with missing data on smoking \( (n = 53) \) were excluded for the purpose of this study. Mean age at the baseline examination was 50.3 ± 6.8 (range 28.3–57.1).

Between 1991 and 1996, all women between 45 and 73 years of age living in the city of Malmö were invited to participate in the Malmö Diet and Cancer study [6]. Of the women from the baseline examination, 3268 smokers and 4417 never smokers belonged to the eligible population. In all, 2431 (55%) of the never smokers and 1594 (49%) of the smokers participated. When comparing data from the first examination, those who participated at the re-examination were somewhat older (mean age 50.4 years vs. 49.8 years), had lower SBP (124.0 mmHg vs. 126.1 mmHg), and weight (65.1 kg vs. 66.2 kg) and had a lower prevalence of smoking (29.8% vs. 40.8%) than the ‘dropouts’. After exclusion of subjects with a follow-up <1 year (26 smokers and 40 never smokers), women with a history of myocardial infarction (18 smokers, and 10 never smokers), 2381 never smokers and 1550 smokers remained. Of the smokers, 388 had quit smoking at the time of re-examination and had been smoke-free for a mean period of 5.5 years. Mean age at follow-up was 59.3 ± 4.9 (range 46.0–70.0).

The Ethics Committee of the Lund University approved the study and all participants gave signed informed consent.

**Examinations**

Smoking was assessed in a questionnaire at both examinations. The never smokers \( (n = 2381) \) consisted of women who at the follow-up examination reported that they never had been smoking. The ‘continuing smokers’ \( (n = 1162) \) consisted of women who reported daily smoking at the baseline examination and who reported regular or occasional smoking at the follow-up. The ‘quitters’ consisted of women who reported daily smoking at the baseline examination and who were ex-smokers at the follow-up \( (n = 388) \). This group was divided into those who had been smoke-free for <1, 1–2, 2–5, 5–10 and >10 years. Information about smoke-free years was missing for eight women.

Women who had quit smoking before the baseline examination were not included in the present study as no information on their precessation weight and blood pressure was available.

**Weight and blood pressure**

At baseline, blood pressure was measured twice after 10 min rest with a sphygmomanometer and a rubber cuff, with the subjects in supine position. At the follow-up examination, blood pressure was measured once after a 5-min rest, with the same equipment. Subjects who had systolic blood pressure (SBP) ≥ 160 mmHg, diastolic blood pressure (DBP) ≥ 95 mmHg or treatment of hypertension were classified as hypertensive [7]. Because the most recent definition requires several repeated measurements [8], the previous definition was used in order to reduce the number of false-positive cases. Height (cm) and weight (kg) were measured whilst the subject wore light indoor clothing and was without shoes.

At the follow-up examination a trained nurse measured the waist circumference (cm) at the umbilicus [9] and the hip circumference (cm) was measured at the broadest hip point. The waist to hip ratio (WHR) was calculated.

Body mass index (BMI) at baseline was calculated as weight/height\(^2\) (kg m\(^{-2}\)).

Weight increase (\(Δ\text{weight}\)) increase in SBP (\(Δ\text{SBP}\)) and DBP (\(Δ\text{DBP}\)) was calculated as the baseline value subtracted from the follow-up value.

**Physical activity**

At the follow-up examination physical activity was assessed by a comprehensive questionnaire. Seventeen activities together with open alternatives were
used to describe leisure-time physical activity [10]. For each type of physical activity, the participants were asked how many minutes per week they spent during each season. The average time was multiplied with an intensity factor to create a physical activity index. The quartiles of the physical activity score were used as an ordinal variable in the analyses.

**Diabetes**

Diabetes at the baseline examination was defined as fasting venous whole blood glucose ≥ 6.1 mmol L⁻¹ or self-reported diabetes according to questionnaire.

**Alcohol consumption**

Diet registration in the Malmö Diet and Cancer Study included alcohol consumption and has previously been described elsewhere. The quartiles of alcohol consumption were used in the analysis [6].

**Marital and socio-economic status**

Information about occupation and marital status was assessed by questionnaire at the follow-up examination. High- and medium-level nonmanual workers and self-employed women were categorized as ‘high’ occupational groups [socio-economic index (SEI); SEI group 46–79] (n = 892) [11, 12]. Low-level nonmanual workers, manual workers and unspecified occupational groups (early retired women, housewives, students, unemployed) were categorized as ‘low occupational groups’ (SEI groups 11–36 and 91–99) (n = 3023) [11, 12]. Information about socio-economic status was missing on 16 women. The women were divided into those who were married or cohabiting (n = 2727) and those who were divorced, widows or unmarried (‘single’; n = 1201). Information about marital status was missing for three women.

**Statistics**

One-way analysis of variance was used to compare differences between groups. A general linear model was used to adjust the mean values for confounders. Tukey HSD was used for posthoc comparisons. A linear multiple regression tested the relationship between Δweight and blood pressure increase. Logistic regression was used to compare incidence of hypertension with adjustments for confounders.

**Results**

At baseline there was no significant difference in weight, SBP or DBP between women who continued to smoke and women who later stopped smoking (Table 1). Age, SBP, DBP and weight at baseline were higher amongst never smokers. The difference in blood pressure remained significant after adjustments for age, baseline physical activity, BMI, blood pressure treatment and diabetes (Table 1). The
relationship was similar after excluding women with treatment for hypertension (data not shown).

Weight increase between baseline and follow-up

Weight increase was highest in quitters and lowest in continuing smokers (Table 2). The differences were significant after adjustments for potential confounders. Similar differences were observed in women whose baseline BMI was below and above 28 kg m\(^{-2}\) (data not shown). The results were virtually identical after exclusion of women with diabetes at baseline (not shown).

The quitters were divided into those who had been smoke-free <1 year (\(n = 88\)), 1–2 years (\(n = 59\)), 2–5 years (\(n = 94\)), 5–10 years (\(n = 65\)) and >10 years (\(n = 74\)). Adjusted for age, follow-up time and baseline BMI, the mean (standard error, SE) weight gain in these groups was 7.3 (0.63), 8.8 (0.77), 7.9 (0.61), 7.7 (0.73) and 6.2 (0.72), respectively (not significant).

Blood pressure increase

\(\Delta\)SBP and \(\Delta\)DBP was studied in women (\(n = 3228\)) who were without blood pressure medication both at baseline and at follow-up (Table 3). \(\Delta\)SBP and \(\Delta\)DBP was highest in quitters and lowest in never smokers. The differences in \(\Delta\)SBP and \(\Delta\)DBP between the groups remained significant after adjustment for follow-up time and age at baseline and also after adjustments for other risk factors (Table 3).

Adjusted for age, follow-up time and SBP at baseline, \(\Delta\)SBP (mean, SE) was 20.1 (1.8), 18.7 (2.1), 19.3 (1.6), 20.5 (2.2) and 22.2 (2.0), respectively, for quitters who had been smoke-free <1, 1–2, 2–5, 5–10 and >10 years (trend: not significant).

Relationship between weight gain and blood pressure increase

The correlation coefficient between \(\Delta\)weight and \(\Delta\)SBP was \(r = 0.16\) (\(P < 0.001\)) for all subjects. The corresponding correlation in never smokers was \(r = 0.15\) (\(P < 0.001\)), in smokers it was \(r = 0.13\) (\(P < 0.001\)) and in quitters \(r = 0.17\) (\(P < 0.001\)). In a multiple linear regression model \(\Delta\)weight remained significantly associated with \(\Delta\)SBP (\(r = 0.13\); \(P < 0.001\)) after adjustments for other risk factors (age, SBP, BMI and diabetes at baseline, follow-up time, physical activity, waist–hip ratio, alcohol consumption, civil status and socio-economic factors at follow-up). \(\Delta\)DBP showed essentially the same relationship with weight gain (\(r = 0.13\), \(P < 0.001\) for all subjects).

Incidence of hypertension

Incidence of hypertension was studied in 3317 women who had normal blood pressure at baseline (<160/95 mmHg and no blood pressure treatment). Incidence of hypertension was highest in quitters (Table 4).

Discussion

Although smokers in most cross-sectional studies have been found to have lower BP than former

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**Table 2** Weight increase between baseline and follow-up in never smokers, continuing smokers and smokers who stopped smoking during follow-up (\(n = 3931\))

<table>
<thead>
<tr>
<th></th>
<th>Stopped smoking during follow-up ((n = 388))</th>
<th>Continued to smoke ((n = 1162))</th>
<th>Never smokers ((n = 2381))</th>
<th>Posthoc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Follow-up time (years)</td>
<td>11.0 ± 4.4</td>
<td>9.2 ± 4.5</td>
<td>8.7 ± 4.2</td>
<td>**abc</td>
</tr>
<tr>
<td>Weight increase (kg)</td>
<td>7.6 ± 6.1</td>
<td>3.2 ± 5.8</td>
<td>3.7 ± 5.2</td>
<td>**ac</td>
</tr>
<tr>
<td>Adjusted mean</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1</td>
<td>6.7 (0.3)</td>
<td>3.1 (0.2)</td>
<td>3.9 (0.1)</td>
<td>**abc</td>
</tr>
<tr>
<td>Model 2</td>
<td>6.6 (0.3)</td>
<td>3.1 (0.1)</td>
<td>3.8 (0.1)</td>
<td>**abc</td>
</tr>
<tr>
<td>Model 3</td>
<td>6.6 (0.3)</td>
<td>3.0 (0.1)</td>
<td>3.9 (0.1)</td>
<td></td>
</tr>
<tr>
<td>Weight at follow-up (kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age-adjusted mean</td>
<td>70.6 (0.61)</td>
<td>66.3 (0.35)</td>
<td>69.5 (0.24)</td>
<td>**bc</td>
</tr>
</tbody>
</table>

Values are mean ± SD or adjusted mean (SE). Model 1: Adjusted for age at baseline and follow-up time. Model 2: Model 1+BMI, diabetes mellitus at baseline and physical activity, waist–hip ratio, alcohol at follow-up. Model 3: Model 2+socio-economic level and marital status. *\(P < 0.05\), **\(P < 0.001\) significant for a quitters versus never smokers, b continuing smokers versus never smokers, c quitters versus continuing smokers.
smokers, the long-term effects of smoking and smoking cessation on blood pressure are unclear. In the present study, mean weight increase was about 3 kg higher amongst quitters than it was amongst continuing smokers. Although the differences in $\Delta_{SBP}$ and $\Delta_{DBP}$ were small, incidence of hypertension was higher in quitters.

Women who stop smoking may do so for many reasons. In some cases smoking cessation may be a result of increased awareness of the potential health consequences. For many of these women quitting may be accompanied by improved health behaviour, increased physical activity and a more healthy diet, which hypothetically could reduce blood pressure. Other women may stop smoking because of poor health. Whether ex-smokers who at the follow-up were treated for hypertension had stopped before or after the diagnosis of hypertension is not known and it is unclear whether this diagnosis influenced their decision to quit smoking. In contrast to incidence of hypertension, $\Delta_{SBP}$ and $\Delta_{DBP}$ showed rather small differences between quitters and smokers. $\Delta_{SBP}$ and $\Delta_{DBP}$ was however studied only amongst those without blood pressure treatment. Inverse causation as well as the heterogeneity amongst the quitters may explain why the relationship between smoking

### Table 3 Blood pressure increase over a mean follow-up of 9 years in 3228 women without blood pressure treatment at baseline and follow-up

<table>
<thead>
<tr>
<th></th>
<th>Stopped smoking during follow-up ($n = 317$)</th>
<th>Continued to smoke ($n = 979$)</th>
<th>Never smoked ($n = 1932$)</th>
<th>Posthoc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure increase</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\Delta_{SBP}$</td>
<td>$20.9 \pm 16.8$</td>
<td>$19.1 \pm 15.8$</td>
<td>$16.1 \pm 16.3$</td>
<td>$^{**abc}$</td>
</tr>
<tr>
<td>$\Delta_{DBP}$</td>
<td>$6.2 \pm 8.7$</td>
<td>$5.7 \pm 9.3$</td>
<td>$3.1 \pm 8.0$</td>
<td>$^{**abc}$</td>
</tr>
<tr>
<td>Model 1</td>
<td>$20.5 (0.9)$</td>
<td>$19.3 (0.5)$</td>
<td>$16.0 (0.3)$</td>
<td>$^{**abc}$</td>
</tr>
<tr>
<td>$\Delta_{SBP}$</td>
<td>$6.2 (0.5)$</td>
<td>$5.6 (0.3)$</td>
<td>$3.2 (0.2)$</td>
<td>$^{**abc}$</td>
</tr>
<tr>
<td>$\Delta_{DBP}$</td>
<td>$5.4 (0.4)$</td>
<td>$4.6 (0.3)$</td>
<td>$3.8 (0.2)$</td>
<td>$^{abc}$</td>
</tr>
<tr>
<td>Model 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\Delta_{SBP}$</td>
<td>$19.7 (0.7)$</td>
<td>$18.3 (0.5)$</td>
<td>$16.7 (0.3)$</td>
<td>$^{ac}$</td>
</tr>
<tr>
<td>$\Delta_{DBP}$</td>
<td>$5.3 (0.4)$</td>
<td>$4.6 (0.2)$</td>
<td>$3.8 (0.2)$</td>
<td>$^{abc}$</td>
</tr>
<tr>
<td>Blood pressure at follow-up (age-adjusted mean)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>$139.0 (0.99)$</td>
<td>$137.2 (0.56)$</td>
<td>$139.4 (0.34)$</td>
<td>$^{bc}$</td>
</tr>
<tr>
<td>DBP</td>
<td>$84.2 (0.31)$</td>
<td>$83.0 (0.29)$</td>
<td>$84.4 (0.21)$</td>
<td>$^{bc}$</td>
</tr>
</tbody>
</table>

Values are mean ± SD, adjusted mean (SE) or number (%) for SBP, systolic blood pressure; DBP, diastolic blood pressure. Model 1: Adjustments for age at baseline and follow-up time. Model 2: Model 1 + BMI, blood pressure (SBP or DBP), diabetes at baseline and waist-hip ratio, physical activity and alcohol consumption at follow-up, Model 3: Model 2 + socio-economic level and marital status. *$P < 0.05$; **$P < 0.001$ significant for $^a$quitters versus never smokers, $^b$continuing smokers versus never smokers, $^c$quitters versus continuing smokers.

### Table 4 Incidence of hypertension over a mean follow-up of 9 years in 3117 initially normotensive never smokers, continuing smokers and smokers who stopped smoking during follow-up

<table>
<thead>
<tr>
<th></th>
<th>Stopped smoking during follow-up ($n = 350$)</th>
<th>Continued to smoke ($n = 1033$)</th>
<th>Never smoked ($n = 1934$)</th>
<th>Posthoc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence of hypertension n (%)</td>
<td>$110 (31.4)$</td>
<td>$242 (23.4)$</td>
<td>$478 (24.7)$</td>
<td>$^{**ac}$</td>
</tr>
<tr>
<td>Blood pressure treatment n (%)</td>
<td>$46 (13.1)$</td>
<td>$91 (8.8)$</td>
<td>$131 (6.8)$</td>
<td>$^{**abc}$</td>
</tr>
<tr>
<td>Incidence of hypertension OR (CI)</td>
<td>$1.4 (1.05–1.8)$</td>
<td>$0.95 (0.8–1.1)$</td>
<td>$1.0$</td>
<td>$^a$</td>
</tr>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 2</td>
<td>$1.9 (1.4–2.5)$</td>
<td>$1.3 (1.0–2.5)$</td>
<td>$1.0$</td>
<td>$^{**abc}$</td>
</tr>
<tr>
<td>Model 3</td>
<td>$1.8 (1.4–2.5)$</td>
<td>$1.3 (1.0–2.5)$</td>
<td>$1.0$</td>
<td>$^{**abc}$</td>
</tr>
</tbody>
</table>

Values are mean ± SD or adjusted mean (SE). Model 1: Adjusted for age at baseline and follow-up time. Model 2: Model 1 + diabetes mellitus, systolic blood pressure and body mass index at baseline, physical activity, waist–hip ratio and alcohol at follow-up, Model 3: Model 2 + socio-economic level and marital status. *$P < 0.05$; **$P < 0.001$ significant for $^a$quitters versus never smokers, $^b$continuing smokers versus never smokers, $^c$quitters versus continuing smokers.
cessation and incidence of hypertension was stronger than between smoking cessation and ΔSBP and ΔDBP.

The many positive effects of tobacco cessation (e.g. on health) and the economical benefits are well known. Weight increase after tobacco cessation is a well-known phenomenon. Fear of weight gain is therefore a major obstacle for women who want to quit smoking [13, 14] and smoking women often use tobacco as weight control [15]. Several explanations for the relationship between smoking and a lower body weight have been proposed. Nicotine seems to affect the monoaminergic regulation of food intake in the hypothalamus [16, 17]. Smokers seem to have a higher metabolic rate [17–19], which might contribute to weight increase after tobacco cessation. Studies on the effects on tobacco cessation usually have a short follow-up period of 6 months or 1 year [14, 19, 20]. More seldom studies discuss the effects of smoking cessation in relation to the normal age-related weight gain [21]. Similar to the results of Williamson et al. [22], the present study shows that quitters gained 3.5 kg more than continuing smokers and 2.8 kg more than never smokers after adjustments for biological risk factors. At baseline, the adjusted mean weight of quitters was 2.0 kg lower than in never smokers. This suggests that the weight gain experienced by quitters mostly reflects a ‘normalization’ to the corresponding weight in those who never smoked.

ΔSBP and ΔDBP was significantly higher in quitters when compared with never smokers. As ΔSBP and ΔDBP in women without blood pressure treatment was similar at follow-up, this suggests that the blood pressure increase in quitters also reflects a normalization, similar to weight increase. However, as the incidence of hypertension and blood pressure treatment was higher in quitters, we can conclude that new cases with hypertension are more common in women who quit smoking.

The long-term effects of smoking on blood pressure are complex and findings are sometimes contradictory [1, 2]. Most cross-sectional studies report lower blood pressure in smokers [23–25]. Others have studied the effect of smoking cessation on blood pressure. Green and Harari reported unchanged blood pressure in men after a mean follow-up of 2.5 years, despite increased weight [26]. In this study, we found that ΔSBP was high already after <1 year of tobacco abstinence and was similar for those who had been smoke-free for longer periods. Lee et al. reported similar results in men, although their follow-up time was shorter [27].

Although smoking is known to be a strong health hazard, quitting smoking is not without ‘side-effects’. This could hypothetically reduce the positive effects of quitting smoking. However, many studies have shown that ex-smokers have lower cardiovascular risk than those who continue to smoke. Unpublished data from the present cohort suggest that the combination of smoking and hypertension synergistically increased the risk of myocardial infarction. We also found that hypertensive women who were ex-smokers had a substantially lower risk than hypertensive smokers and also lower risk than smokers with normal blood pressure. Hence, there is no reason to believe that the increased risk of hypertension counterbalances the positive effect of tobacco abstinence.

Misclassification of exposure is a potential cause of bias in epidemiological studies. Blood pressure is characterized by large intra-individual variations. Several measurements should ideally be performed to reduce misclassification [28]. In this follow-up study blood pressure was measured twice at baseline after 10 min rest. At follow-up, blood pressure was measured only once after 5 min rest. This could have reduced the relationship between BP and smoking habits. Another limitation is that some risk factors were assessed only at one of the examinations, e.g. alcohol consumption and socio-economic circumstances. We do not know whether these factors changed during follow-up. Selective mortality of high-risk individuals during follow-up and nonparticipants of the follow-up examination is also a potential cause of bias. We do not know whether nonparticipation at follow-up was related to smoking cessation or weight and blood pressure increase. We cannot claim that the study cohort is truly representative and results are hence based on the internal validity of the study population.

Over a long follow-up, women who stop smoking gained weight approximately 3 kg more than never smokers. Although the effects on ΔSBP and ΔDBP were moderate, the incidence of hypertension was significantly higher in women who stopped smoking.

**Conflict of interest statement**

No conflict of interest was declared.
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