Smoking Initiation and Cessation in Relation to Body Fat Distribution Based on Data from a Study of Swedish Women

ABSTRACT

In a representative sample of Swedish women, smokers were significantly less obese than nonsmokers. However, a smoker was likely to have significantly more upper-body fat than a nonsmoker of similar body mass index. Women who quit smoking experienced less upper-body fat deposition than would be expected by their accompanying weight gain, suggesting that weight gained as a consequence of smoking cessation is not preferentially deposited in the region associated with increased cardiovascular risk. (*Am J Public Health.* 1992;82:273–275)

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Introduction

A recent survey showed that 39% of female cigarette smokers viewed smoking as a tool for weight control.[1] Weight gain and increased appetite were cited as important reasons for relapse following attempts to quit smoking. In agreement with this popular notion, epidemiological studies have demonstrated clearly that smokers weigh less than nonsmokers[2,3] and that smoking cessation leads to weight gain among both men[3–5] and women.[4–6] In two studies involving male subjects, associations have been observed between cigarette smoking and centralized body fat distribution.[7,8] Furthermore, in one of these latter studies, smoking cessation was associated with a smaller degree of upper-body fat deposition than would be anticipated from the accompanying weight gain.[7] Less is known, however, about the effects of smoking initiation and cessation on body fat distribution in women.

Thus, the aim of this paper is twofold: to describe cross-sectional associations between smoking and body fat distribution in a representative sample of Swedish women; and to examine changes in fat distribution that occur as a consequence of smoking initiation and cessation. If the findings in women are similar to those in men, this would confirm that gains in body weight following smoking cessation do not follow the androgenic fat distribution pattern associated with increased cardiovascular risk in women[9–12] and in men.[11–13]

Subjects and Methods

This analysis is based on data from the Prospective Population Study of Women in Gothenburg, Sweden. This is a representative sample of female Gothenburg residents born in 1908, 1914, 1918, 1922, and 1930. The analysis presented here uses data from the first two examinations, which took place in 1968/69 and in 1974/75. Details of the sampling, participation rates, and examination protocol have been given elsewhere.[6,9,14,15]

Anthropometric measurements were taken at the same time of day and year at both examinations. A subject's body mass index (BMI) ($weight_{kg}/height_m^2$) was used as an indicator of overall adiposity, while waist-hip ratio (waist circumference/hip circumference) reflected the distribution of her body fat, with high values indicating upper-body obesity. Changes in BMI and waist-hip ratio were calculated as 1974/75 values minus 1968/69 values. Smoking history was ascertained by means of standardized questions at each examination. Subjects who had never smoked or who had not smoked for at least a year were classified as nonsmokers. Women who reported quitting at any time during the previous 12 months were excluded.

In the cross-sectional portion of this investigation, analysis of variance was used to compare BMI and waist-hip ratio in current smokers with those in nonsmokers, using 1974/75 data. All cross-sectional findings were replicated using the 1968/69 measurements not shown. Similarly, the longitudinal analysis used data from both examinations to compare continuing smokers with women who quit smoking, and nonsmokers with women who started smoking. After controlling for age and (when appropriate) BMI, adjusted least square means were computed and reported here with their standard errors.

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TABLE 1—Comparisons of Smokers and Nonsmokers, 1974/75: Age-adjusted (top); Adjusted for Age and Body Mass Index (bottom).

	Smokers		Nonsmokers		Smokers vs Nonsmokers
	(n = 486)	SE	(n = 805)	SE	*P*
BMI[a]	23.8	(0.18)	25.1	(0.14)	.0001
waist-hip ratio	0.793	(0.003)	0.782	(0.002)	.003
BMI[a]-adjusted waist-hip ratio	0.798	(0.003)	0.779	(0.002)	.0001

Note. Least square means are shown with standard errors.
[a]BMI = body mass index.

TABLE 2—Effects of Smoking Initiation and Cessation between Examinations 1 and 2 on Body Mass Index and Waist-Hip Ratio: Age-adjusted (top); Adjusted for Age and Change in Body Mass Index (bottom).

	Initiation			Cessation		
	Never Smoked	Started		Continue Smoking	Quit	
Change in	(n = 701)	(n = 26)	*P* diff	(n = 435)	(n = 63)	*P* diff
BMI[a]	0.62 (0.067) ****	−0.37 (0.35)	**	0.50 (0.089) ****	1.4 (0.24) ****	***
Waist-hip ratio	0.048 (0.0016) ****	0.046 (0.0084) ****	n.s.	0.050 (0.003) ****	0.040 (0.0079) ****	n.s.
BMI[a]-adjusted waist-hip ratio	0.048 (0.0015) ****	0.054 (0.0079) ****	n.s.	0.051 (0.0029) ****	0.033 (0.0077) ****	*

Note. For each group, least square means are shown with standard errors and significance for H_0:change = 0. Right columns show significance levels for between-group differences.
[a]BMI = body mass index.
*P <.05. **P <.01. ***P <.001.****P < .0001. N.S. = not significant.

Results

Cross-sectional Analysis

The rate of cigarette smoking was 41.0% in 1968/69 and 37.6% in 1974/75. Smokers had lower BMIs (P = .0001) and higher waist-hip ratios (P = .003) than nonsmokers, after controlling for age, in agreement with previous findings among this cohort[6,9] (see Table 1). To determine whether smoking was associated with fat patterning independent of generalized obesity, further statistical adjustment was made for BMI. The differences between waist-hip ratios of smokers and nonsmokers were accentuated after controlling for BMI (P = .0001), indicating that a smoker is likely to have a significantly higher proportion of upper- to lower-body fat than a nonsmoker of similar body mass.

Longitudinal Analysis

Between the two examinations, 3.5% of nonsmokers started smoking and 12.7% of smokers quit. The purpose of the second analysis was to evaluate specific effects of smoking initiation and cessation on BMI and waist-hip ratio. To test for an initiation effect, comparisons were made between subjects who did not smoke at either exam and those who began smoking between the two exams. As expected, the women who started smoking tended to lose weight while the nonsmokers gained weight, and the difference between these groups was significant (P = .005). Conversely, a comparison of women who smoked at both exams with those who quit indicated that, although both groups gained weight, the quitters gained significantly more (P = .0006) (see Table 2).

To evaluate effects of smoking initiation and cessation on fat distribution, changes in waist-hip ratio were examined, with and without adjustment for changes in BMI (see Table 2). The initial analysis showed no significant effects of smoking initiation or cessation on waist-hip ratio; all four groups displayed significant increases in this ratio over time (P = .0001). However, after controlling for possible confounding by BMI changes, significant differences in the waist-hip ratio emerged in smokers versus quitters (P = .03). Waist-hip ratio increased by slightly more in smokers (+0.05) than in quitters (+0.03), which is contrary to what one would predict from their respective BMI gains of +0.5 and +1.4. It is concluded that women who stop smoking do not experience the degree of upper-body fat deposition that generally accompanies weight gain.

Discussion

International comparisons of cigarette smoking trends suggest that women are quitting less often and/or starting more often than men worldwide.[16,17] At current rates, it has been projected that the majority of smokers in the United States could be women after the year 1995.[18] Therefore, the observation that smoking cessation resulted in less upper-body fat deposition than would be expected from the concomitant weight gain may be of interest to many female smokers. Upper-body fat distribution in women is related to increased rates of coronary heart disease,[9,11] stroke,[9] diabetes,[12] and unfavorable lipid profiles,[10,12] and it may be a more potent cardiovascular risk factor than generalized obesity.[9–12] In this context, our results suggest that the weight gain that often accompanies smoking cessation need not be perceived as an adverse health effect because weight gained as a consequence of quitting smoking is not preferentially deposited in the region associated with increased cardiovascular risk. Such information, together with the similar findings in males,[7] could provide an incentive to a health-conscious public to modify smoking practices in line with a more advantageous body fat distribution, even if some overall weight gain is involved. □

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ABSTRACT

Between January 1986 and June 1989, 1916 New Jersey workers were identified through a surveillance system for occupational lead exposure. The average annual proportion of workers with a blood lead level above 2.42 μmol/L was 12%. Industries with the highest proportion of workers with blood lead levels above 2.42 μmol/L were special trade construction (35%) and industries dealing with scrap and waste materials (27%). (*Am J Public Health.* 1992;82:275–277)

Surveillance of Occupational Lead Exposure in New Jersey: 1986 to 1989

Allison Tepper, PhD

Introduction

Public health agencies in several states have described surveillance programs for occupational lead exposure using biological monitoring data.[1–3] The experience in these states provides useful lessons about program design and operation for others beginning to implement lead surveillance activities. Ongoing reporting of findings helps promote the use of surveillance data for targeting and evaluating lead poisoning prevention efforts. This paper examines the results obtained by the New Jersey Department of Health (NJDOH) occupational lead exposure surveillance project between January 1986 and June 1989.

Methods

A passive surveillance system for occupational lead exposure was begun by the NJDOH in October 1985. In-state clinical laboratories are required to report test results and identifying information for adults (aged 16 and above) with blood lead levels above 1.21 μmol/L* (New Jersey Administrative Code 8:44–2.11).

The NJDOH provides educational materials to physicians and reported individuals by mail and conducts telephone interviews to ascertain the source of exposure. When a work-related exposure is

*1 μmol/L = 20.7 μg/dL.

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