Smoking in the age of obesity: an investigation of secular trends in body fat and cigarette smoking

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Abstract

Objectives: In recent decades, obesity reached epidemic proportions in western countries, while cigarette smoking decreased. Traditionally, smoking is associated with lower relative weight (body mass index, BMI) but high abdominal obesity (waist-hip ratio, WHR). However, several recent studies suggest that BMI is higher among today’s smokers compared with non-smokers. Therefore, the present study examined whether the relationship between smoking and each of the body measures, BMI and WHR, has changed over time.

Material and Methods: Data were collected from 5907 male and female residents of Gothenburg, Sweden, aged 25-65 years, and drawn randomly in four sequential cross-sections (1985, 1990, 1995, 2002) from the city’s population register. The study used general linear models to test trends over time, and adjusted all differences for age.

Results: The data reported here showed higher WHR in both male and female smokers compared with non-smokers. BMI was lower in female smokers compared with female non-smokers, but did not differ significantly between male smokers and male non-smokers. Among female participants, differences in WHR between smokers and non-smokers increased significantly throughout the study. Although male participants showed a similar tendency, the differences were not significant. Adjustment for educational level did not affect the results.

Conclusions: WHR was higher among smokers compared with non-smokers; this difference increased over time. Thus, concern for obesity does not provide a valid reason to continue smoking; on the contrary, it may be a reason to quit.

Key words
body mass index, waist-hip ratio, smoking
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INTRODUCTION

Investigation of the relationship between smoking and obesity

Smoking and obesity are two of the most significant yet preventable risk factors underlying today’s most serious health conditions in high-income countries, for example, heart disease and cancer. Smoking and obesity also contribute increasingly to death and disease in low and middle-income countries (1). Many of these countries, in fact, face the double-burden of undernutrition and obesity (2), as well as experiencing an increase in smoking among both males and females (3, 4). Thus, there is a need to better understand risk factors for prevalent and emerging diseases worldwide. However, predicting trends in these diseases has become more complicated in recent decades, since the prevalence of smoking has decreased while the prevalence of obesity has increased.

To better understand the combined health effects of smoking and body fatness, it is thus of particular interest to investigate whether the relationship between them also has changed. In fact, several Scandinavian studies suggest that the well-known relationship between smoking and thinness has weakened in recent years (5-7). However, no studies have examined changes in the relationship between smoking and body fatness in a representative adult sample over time, which will be the focus of this study. It examines data from 1985-2003, which is when the studies analyzed below, MONICA and INTERGENE, were conducted. This period is characterized by an increase in obesity and a decrease in smoking in Northern and Western Europe (2, 3).

Obesity

Determining body fatness

Excess body fatness is known as ‘obesity’ in its extreme form. ‘Overweight’ is a term that can both express a milder form of excess body fatness and serve as a comprehensive term for all degrees excess body fatness; the latter definition will be applied in this paper.

There are many methods for measuring body fatness. The most accurate measurements are obtained from methods such as underwater weighing and imaging (8, 9), but costs, time requirements and potential health consequences prevent them from being implemented in large-scale. Large-scale population studies tend to approximate true body fat using various anthropometric measurements (10). These measurements are taken most often by trained professionals, since self-reporting is associated with bias, for example, overweight persons differentially underreport their weight, compared to persons who are not overweight (11).

In the literature on large population-based studies, body fatness is seen as a global measure of ‘general’ fatness and as a specific regional measure of e.g. ‘central’ fatness.
The most common anthropometric measures of general and regional fatness include ‘body mass index’ (BMI), ‘waist circumference’ (WC), and ‘waist-hip ratio’ (WHR).

General fatness is often measured in terms of BMI (kg/m$^2$) and is interpreted as weight adjusted for height. BMI is strongly correlated with total fat mass, especially among middle-aged adults (8), and is also predictive of morbidity and mortality (12). There is also a practical advantage to using BMI, since weight and height are commonly collected in health surveys. However, BMI is affected by build: people with a large bone and/or muscular structure will have a larger BMI, independent their body composition (2).

The most studied type of regional adiposity, central obesity, is often operationalized in terms of body circumferences. One commonly employed measure of central obesity, WHR, is calculated by dividing waist by hip circumference (HC) and is a good predictor of mortality and morbidity. Central obesity can also be measured by considering WC alone. This measure gives a measure of general and central fatness (13) and is a particularly good predictor of risk for cardiovascular disease (CVD) (14). HC alone reflects various aspects of body size, including muscle mass and subcutaneous fat mass (13). WC and HC have the advantage of each requiring only one measurement of central obesity and having readily interpretable units, as compared to WHR.

**Combining several measures of body fatness**

To describe overall body fatness, one option is to employ both a measure of general fatness and a measure of central fatness. However, these two measures risk can provide redundant information, as persons with high BMI tend to be more centrally obese (10, 15). This criticism may be more applicable to the combination of BMI and WC than to the combination of BMI and WHR, as the latter pair are less correlated, particularly in women (13), and thus should contribute more complementary information. In fact, BMI explains about three quarters of the variation of WC but only a third of the variation in WHR (15). Jee et al. demonstrated furthermore that BMI and WHR provide different enough information to motivate including both in the investigation of obesity among Koreans (16).

Another argument for measures of general and central fatness being redundant is that central obesity has been shown to have superior predicting power for mortality and morbidity. Yet, many such studies fail to adjust for general fatness and thus do not acknowledge that measures of central fatness only have marginally better predicting power than measures of general fatness (10, 13). Moreover, general and central fatness may correlate better with different health outcomes, e.g. central obesity may better predict diabetes and stroke, while general obesity may better predict CVD and total mortality (13). Thus, the combination of BMI and WHR appears to provide the most complete and non-redundant picture of obesity using anthropometric measures.

WHR and BMI have several drawbacks, which are tied to the properties of ratios. In all ratios, the individual contributions of the numerator and denominator are masked. For
instance, high BMI can reflect general fatness, short stature and/or muscul arity (8), and high WHR can reflect high central fatness, low gluteal muscle mass and/or low gluteal fat mass. Even WHR and BMI in normal ranges can both mask obesity if the numerator and denominator are proportionately extreme and underestimate change if the numerator and denominator decrease proportionately.

Another consideration is that BMI and WHR can vary systematically according to body build, ethnic group, age and gender, since these groups inherently have different amounts of subcutaneous fat, visceral fat and muscle mass (2, 17). Anthropometric ratios are further criticized for not having units with a clear biological interpretation and thus being less useful in clinical settings.

To deal with the above considerations, some advocate single measures, like substituting WC for BMI and WHR (14), while others argue for replacing anthropometric ratios with their respective components, e.g. studying WC and HC individually, rather than WHR (17). Yet, BMI and WHR continue to be used extensively and thus provide a basis for comparison over time for general and central adiposity, respectively.

**Calculation of prevalence levels and trends from anthropometry**

When expressing anthropometric measures, such as BMI and WHR, one can choose between a continuous scale, e.g. BMI of 23.5 or WHR of 0.89, or a categorical label, e.g. *generally obese* or *centrally obese*. The latter corresponds to the range between two predetermined cutoff point, such as those advocated for BMI and WHR by the World Health Organization (WHO) (2). A BMI of at least 25 was suggested to be defined as *overweight*, which is further categorized into *pre-obese* (BMI 25 to 30) and *obese* (BMI 30+), and central obesity was suggested to be defined as a WHR of at least 1.0 for men and 0.85 for women (2, 13). It also should be noted that universal cut-off points are controversial, and it has been suggested that they be set differently for different populations, since optimal anthropometric values are likely to be population-specific (9). In sum, cutoff points are used widely and provide an intuitive standard of comparison, but they arbitrarily dichotomize continuous scales (8), resulting in a loss of information, as compared to using continuous values.

Within the WHO’s European region, general obesity has tripled during the last 20 years, and 10-20% of adult males and 10-25% of adult females were obese in 2000 (2, 18). Similarly, average BMI has increased in all Nordic countries in the past decades (19-23). The prevalence of obesity also has increased in nationally representative samples (19, 24), as well as in various regions and birth cohorts (21, 25-27). Obesity rates more than doubled in the past several decades, and pre-obesity rates have also increased (21, 24, 28). Previous research in Gothenburg, Sweden based on the current study’s sample found that both overweight and obesity have increased (Figure 1).
Central fatness as measured in WHR also increased among both males and females in the current study. This result is supported by WHR having been observed to increase over time in other single populations in the Nordic region (30, 31). Unfortunately, no studies that compare international trends over time in central obesity have been published.

**Smoking behavior**

**Measures of tobacco intake**

Cigarette smoking is usually assessed by self-report questionnaires that define participants as belonging to smoking categories, such as *current smokers, ex-smokers* or *never-smokers*. Such questions are often worded to ask both whether the participant currently smokes cigarettes regularly and whether the participant has ever smoked cigarettes regularly.

**Potential pitfalls of categorizing smoking status**

When smoking status is established qualitatively without information on smoking intensity and duration, one cannot determine whether smokers or ex-smokers have had a greater lifetime exposure to smoking. That is, the category *smokers* can include long-
time smokers, new smokers and re-starters, and the category ex-smokers can include a range of persons, from those who were essentially never-smokers and quit many years ago, to those who are essentially smokers and quit very recently. Furthermore, participants who smoke occasionally may classify themselves as either smokers or never-smokers.

To avoid such potential confounding, one can focus exclusively on the difference between never-smokers and smokers and between never-smokers and ex-smokers, since never-smokers represent an absolute baseline to which smokers and ex-smokers can be compared. The present study will focus primarily on the comparison between smokers and never-smokers.

**Trends in smoking**

Lopez et al. suggest that national smoking prevalence and smoking-related mortality change in a predictable fashion based on gender and the country’s income (3, 4). That is, smoking rates first rise and then fall with increasing country income. Males in a given population pass through the model stages before their female counterparts. In accordance with model predictions for high-income countries, smoking among high-income countries is decreasing, particularly among men (16, 32). This pattern also can be observed in the Nordic countries (Figure 2). For comparison, smoking rates in the WHO Region Europe were at 38% among men and 23% among women by the late 1990s (33).

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**Figure 2.** Secular trends in smoking prevalence from 1980 to 2005 within four Nordic countries according to Statistics Sweden, Statistics Norway, Statistics Finland and Statistics Iceland (34-37)
The relationship between smoking and obesity

The relationship between smoking and obesity has been examined by comparing anthropometric measures for never-smokers, smokers and ex-smokers. In such studies, BMI has been consistently lower for smokers than for never-smokers (Table 1). Two of these are international meta-studies of BMI: the Diverse Populations study (DP) (38) and the WHO MONICA project (the World Health Organization’s MONItoring of trends and determinants in CArdiovascular disease) (39). In DP, the outcomes of 21 independent studies are compared statistically, while MONICA is a multi-center study consisting of data obtained using a standard protocol in 32 collaborating centers worldwide (40). In these two meta-studies, it has been estimated that smokers were approximately one BMI unit leaner than never-smokers, a difference of 2.89 kg for 170 cm tall adults (38, 39).
<table>
<thead>
<tr>
<th>Country (study reference)</th>
<th>Study dates</th>
<th>Age in years old</th>
<th>BMI difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Males</td>
</tr>
<tr>
<td>USA (40)</td>
<td>1960-86</td>
<td>19-102</td>
<td>-0.3</td>
</tr>
<tr>
<td>China, Iceland, Israel,</td>
<td>1956-90</td>
<td>25-92</td>
<td>-0.97*</td>
</tr>
<tr>
<td>Norway, Puerto Rico, USA, Scotland and Yugoslavia (38)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sweden (42)</td>
<td>1974-75</td>
<td>45, 53, 57, 61, 67</td>
<td>-1.3</td>
</tr>
<tr>
<td>USA (43)</td>
<td>1976-80</td>
<td>19-74</td>
<td>-0.3</td>
</tr>
<tr>
<td>Sweden (7)</td>
<td>1963</td>
<td>50</td>
<td>-0.9</td>
</tr>
<tr>
<td></td>
<td>1973</td>
<td></td>
<td>-0.3</td>
</tr>
<tr>
<td></td>
<td>1983</td>
<td></td>
<td>-0.5</td>
</tr>
<tr>
<td></td>
<td>1993</td>
<td></td>
<td>0.2</td>
</tr>
<tr>
<td>China, eastern and western European countries, New Zealand, USA and Australia (39)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1979-89</td>
<td>35-64</td>
<td>-1.1*</td>
</tr>
<tr>
<td>Finland (6)</td>
<td>1982</td>
<td>25-44</td>
<td>-0.83</td>
</tr>
<tr>
<td></td>
<td>1987</td>
<td></td>
<td>-0.52</td>
</tr>
<tr>
<td>USA (44)</td>
<td>1984-87</td>
<td>50-79</td>
<td>-1.2*</td>
</tr>
<tr>
<td>Finland (45)</td>
<td>1978-95</td>
<td>15-64</td>
<td>0.13</td>
</tr>
<tr>
<td>Israel (46)</td>
<td>1985-87</td>
<td>&quot;middle-aged&quot;</td>
<td>-0.3</td>
</tr>
<tr>
<td></td>
<td>1988-90</td>
<td></td>
<td>-0.13</td>
</tr>
<tr>
<td>Scotland (47)</td>
<td>1998</td>
<td>16-24</td>
<td>0.9</td>
</tr>
<tr>
<td></td>
<td>25-34</td>
<td></td>
<td>-1.3**</td>
</tr>
<tr>
<td></td>
<td>35-44</td>
<td></td>
<td>-1.8**</td>
</tr>
<tr>
<td></td>
<td>45-54</td>
<td></td>
<td>-2.4**</td>
</tr>
<tr>
<td></td>
<td>55-64</td>
<td></td>
<td>-1.3**</td>
</tr>
<tr>
<td></td>
<td>65-74</td>
<td></td>
<td>-2.2**</td>
</tr>
</tbody>
</table>

Although no meta-studies have compared WHR among smokers and never-smokers, several studies on single populations have estimated WHR to be approximately 0.01 units greater among smokers (Table 2), that is a difference of one centimeter in waist circumference for a hip circumference of 100 cm. This combination of relatively low BMI and high WHR indicates that smokers on average have less general but more central fatness than never-smokers.
Table 2. Comparison of age-adjusted WHR for smokers minus never-smokers

<table>
<thead>
<tr>
<th>Country (study reference)</th>
<th>Study dates</th>
<th>Age in years old</th>
<th>WHR difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Males</td>
</tr>
<tr>
<td>USA (41)</td>
<td>1960-86</td>
<td>19-102</td>
<td>0.011**</td>
</tr>
<tr>
<td>Sweden (30)</td>
<td>1968-69</td>
<td>38</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50</td>
<td>0.02</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1980-81</td>
<td>38</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50</td>
<td>0.02</td>
</tr>
<tr>
<td>Sweden (42)</td>
<td>1974-75</td>
<td>44, 52, 56, 61, 67</td>
<td>0.011</td>
</tr>
<tr>
<td>USA (43)</td>
<td>1984-87</td>
<td>50-79</td>
<td>0.016**</td>
</tr>
<tr>
<td>Finland (31)</td>
<td>1987-97</td>
<td>25-64</td>
<td>0.0102**</td>
</tr>
<tr>
<td>United Kingdom (48)</td>
<td>&lt;10 pack-yrs for smokers</td>
<td>1993-97</td>
<td>45-79</td>
</tr>
<tr>
<td></td>
<td>≥10 pack-yrs for smokers</td>
<td>1993-97</td>
<td>45-79</td>
</tr>
</tbody>
</table>

Key: ** Difference significant at 0.01

Changes over time in the relationship between smoking and BMI have only been examined in a few studies. Approximately 20 years ago, Marti et al. found that BMI increased more among Finnish smokers than never-smokers (6). Similarly, studies from Gothenburg, Sweden showed that BMI increased significantly among 50-year-old male smokers but not among their never-smoking counterparts (7) and among 70-year-old males and females (5) between the 1960s/1970s and 1990s. Examination of changes in the relationship between smoking and WHR is even rarer, and one study found no change for 38 or 50 year old females (30). In summary, current evidence suggests that the difference in BMI between smokers and non-smokers may be narrowing, whereas the difference in WHR between smokers and never-smokers has been less extensively studied. Thus, the present study investigates whether the relationships of smoking to BMI and to WHR have changed over time for participants in a broader age range.

AIM

To determine whether the relationship between smoking and obesity has changed between 1985, 1990, 1995 and 2001-3 among Swedish adults.
METHOD

Selection of participants

Study participants were selected from the population census register in Gothenburg, Sweden in four independent samples. A basic design difference between MONICA and INTERGENE was that sampling for MONICA was done in ten-year age strata, while the INTERGENE sample was randomly identified across the whole age range, as described below.

The WHO MONICA study (the World Health Organization’s MONItoring of trends and determinants in Cardiovascular disease) is a large, international study consisting of data on smoking, fatness and other health-related variables. In Gothenburg, approximately 300 Gothenburg residents of each gender were selected randomly within four 10-year age intervals: 25-34, 35-44, 45-54 and 55-64. Those selected were sent a postal questionnaire and information about the study. Once the questionnaire was returned, each individual was invited to a clinical examination (49).

The INTERGENE project examines the INTERplay between GENetic susceptibility and environmental factors, including lifestyle and psychosocial background, for the risk of cardiovascular diseases in western Sweden (50, 51). Potential participants were chosen by simple random sampling among individuals living in Gothenburg on April 1, 2001 who were 25-74 years old. Those selected were sent a postal questionnaire, along with information about the study and later were contacted about attending a clinical examination. The data used for this study were, however, collected between 2001 and 2003, rather than between 2001 and 2002, as in the studies referenced above. Only data from participants in the age range 25-64 years old was used in this study.

Ethical considerations

Both the MONICA and INTERGENE studies were approved by the regional ethics committee at University of Gothenburg. In return for participation, all participants received results from their blood tests, which included a recommendation for those with unhealthy values to consult a physician.

Clinical Examinations

All study participants attended a clinical examination that included anthropometric measurements (height, weight, waist and hip), ECG recordings, blood samples and questionnaires (49-51). Participants were requested to have fasted for four hours before examination. Pregnant women were included.

Anthropometric measurements were taken with participants in light clothing without shoes and were estimated to the nearest cm or 0.1 kg. Waist was defined as midway
between the lower rib margin and iliac crest. Hip was defined as the largest circumference around the buttocks.

**Questionnaire Data**

Responses to the questionnaires were used to determine smoking status, educational level and physical activity. The questionnaires were created before the Author joined the study, but the Author participated in the later stages of data collection for the INTERGENE questionnaire.

**Smoking categories**

Among MONICA participants, the assessment of smoking status was based on the question: *Do you currently smoke cigarettes?* In INTERGENE, the corresponding question was: *Have you ever smoking cigarettes regularly?*

Thus, never-smoking in MONICA was determined if participants first responded that they did not currently smoke cigarettes regularly and then that they never have smoked cigarettes regularly. In INTERGENE, never-smoking was determined if participants’ answer to the first question was that they never had smoked cigarettes regularly.

Current smoking was determined if participants smoked at least one cigarette per day on average. In MONICA those categorized as current smokers answered first that they smoked cigarettes regularly, then that they still smoke and finally that they smoke at least one cigarette per day. In INTERGENE, two questions were employed, first whether the participants currently smoke regularly and then how many cigarettes they smoke per day.

Former smoking in MONICA was determined when participants answered that they did not currently smoke but that they had smoked previously. In INTERGENE, participants who answered that they had once smoked cigarettes regularly but then had quit smoking were counted as former smokers.

**Education categories**

Education categories reflect the Swedish educational system: *elementary school, junior secondary school, vocational school/girls’ school, high school* and *college/university*. INTERGENE, an additional category was present, *other education*. Participants were to mark the highest category of education that they had attended.
Physical activity categories

There were four levels of physical activity during one’s leisure time: sedentary (TV, computer, reading etc.), moderate exercise (light exercise at least four hours per week), regular exercise and training (running, swimming etc. at least 2-3 hours per week) and hard training or competitive sports (regularly or several times per week).

Analyzed Variables

Questionnaire items were coded for the analysis as follows: smoking status was divided into the categories never-smokers, ex-smokers and current smokers; education was divided into the categories pre-university and university education; and physical activity was divided into the categories sedentary (level 1: sedentary) and active (levels 2-4: moderate, regular and hard exercise). The variables obtained from the clinical examinations, BMI and WHR, were treated as continuous, numeric data. Additional variables in the analysis include survey, which corresponded to the examination year, that is, survey 1: 1985, survey 2: 1990, survey 3: 1995 or survey 4: 2001-2003 and age, which was calculated from each person’s national identity number, which includes birth date.

Statistical Methods

To examine the study’s participation rates, participants first were grouped according to sex, 10-year age group and survey before the rates presented as proportions and counts (Table 3). Smoking prevalence was then investigated by calculating proportions and counts grouped by sex and survey (Table 4)

Linear and logistic regression models were defined for each gender. First, trends in smoking prevalence (binary) were modeled by the continuous variables age (in years) and survey (1-4) using logistic regression.

Next, trends in BMI and WHR (both continuous) over the four surveys were investigated using linear regression, which included the variables age, survey and two dummy variables for smoking, current smoking and ex-smoking, with never-smoking as the implicit reference variable. The same model allowed for comparison of BMI and WHR values between smoking statuses.

Then, interaction effects of survey and smoking status on BMI and WHR were examined using linear regression, which included the variables: age, survey, current smoking, ex-smoking and two interaction dummy variables, survey*current smoking and survey*ex-smoking.

Finally, age (continuous), as well as university education and physical activity (both binary) were described using logistic and linear regression, respectively. Like the
previous model, this one also employed the independent variables age, survey, current smoking, ex-smoking and two interaction dummy variables, survey*current smoking and survey*ex-smoking. However, age was not included as a dependent variable when it was modeled as an independent variable.

All of the above analyses that included education and WHR were limited to the final three study periods, since data from the first were not available. The values of the dependent variables that were estimated using the above models were based on an age of 45 years old, since this was the approximate average age in the study and since a high participation in this age range suggests good estimates. A significance level of $\leq 0.05$ was considered statistically significant.

RESULTS

Participation rates

In total, the study included 5907 participants, of which 48% were males and 52% were females. Participation rates, however, varied by gender and 10-year age group (Table 3).

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td><strong>Males, % of eligible (n)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25–34 years old</td>
<td>65% (162)</td>
<td>56% (158)</td>
<td>52% (150)</td>
<td>25% (108)</td>
</tr>
<tr>
<td>35–44 years old</td>
<td>60% (149)</td>
<td>67% (198)</td>
<td>63% (184)</td>
<td>50% (180)</td>
</tr>
<tr>
<td>45–54 years old</td>
<td>77% (192)</td>
<td>70% (205)</td>
<td>65% (189)</td>
<td>44% (150)</td>
</tr>
<tr>
<td>55–64 years old</td>
<td>73% (175)</td>
<td>70% (210)</td>
<td>69% (210)</td>
<td>83% (198)</td>
</tr>
<tr>
<td>All ages</td>
<td>69% (678)</td>
<td>66% (771)</td>
<td>63% (733)</td>
<td>47% (636)</td>
</tr>
</tbody>
</table>

| **Females, % of eligible (n)** |         |         |         |         |
| 25–34 years old        | 72% (171)| 56% (149)| 52% (178)| 34% (148)|
| 35–44 years old        | 72% (178)| 71% (206)| 64% (222)| 56% (185)|
| 45–54 years old        | 84% (206)| 74% (216)| 66% (232)| 61% (186)|
| 55–64 years old        | 68% (170)| 73% (214)| 65% (227)| 91% (201)|
| All ages               | 73% (725)| 69% (785)| 62% (859)| 57% (720)|

Smoking rates

The percentage of participants in each smoking category was then calculated for each of the four surveys: 1985, 1990, 1995 and 2001-2003 (Table 4). When adjusted for age and survey date, the prevalence of smoking was found to decrease by an average of 21% per survey among males ($\text{Exp(}_\beta\text{)} = 0.786; P < 0.001$) and 18% among females ($\text{Exp(}_\beta\text{)} = 0.821; P < 0.001$).
Comparisons of smokers’ and never-smokers’ WHR and BMI

To examine the association between smoking status and the two anthropometric measures, the same model, which takes all four study periods into account, was again employed. When the effect of survey was thus controlled for, WHR was found to be an average of 0.015 units greater among smokers than among never-smokers for males ($P < 0.001$) and 0.014 units for females ($P < 0.001$). BMI among female smokers was 0.7 units less than among never-smokers ($P < 0.001$), while the difference between BMI among male smokers and never-smokers was not significant ($\beta = 0.1; P = 0.654$). When BMI and WHR among ex-smokers were compared with never-smokers, both body measures were consistently and significantly greater among ex-smokers: BMI among males ($\beta = 0.5; P < 0.001$) and females ($\beta = 0.4; P = 0.048$); WHR among males ($\beta = 0.012; P < 0.001$) and females ($\beta = 0.010; P = 0.002$).

Secular trends in WHR and BMI

Age-adjusted secular trends were then investigated for the two anthropometric measures, BMI and WHR, in multivariate models that also took smoking status and the four study periods into account. For each successive survey, BMI increased by an average of 0.5 BMI units among males ($P < 0.001$) and by 0.3 units among females ($P < 0.001$). WHR also increased both among males by 0.004 units ($P = 0.002$) and among females by 0.019 WHR units ($P < 0.001$) per survey between the surveys done in 1990 and 2001-2003. It should be reiterated that these changes are simultaneously adjusted for changes in smoking that are also occurring at this time, indicating that the increases in obesity prevalence cannot be explained by less smoking and quitting smoking, as often speculated.
**Interactions between smoking and survey for the anthropometric measures**

Another statistical model then was used to explore whether the relationship between smoking and obesity had changed during the study. Figure 3 shows that anthropometric values increase more for smokers than never-smokers in the case of WHR among females and males, as well as BMI among females. However, a significant interaction between smoking and survey occurred only for WHR among females (P = 0.013) and not for WHR among males (P = 0.143), BMI among females (P = 0.356) or BMI among males (P = 0.669). Among ex-smokers, there were no significant interactions for WHR among females (P = 0.812), WHR among males (P = 0.917), BMI among females (P = 0.320) or BMI among males (P = 0.283). All interactions were also adjusted for educational level, but none was appreciably affected (data not shown).

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**Figure 3.** Age-adjusted BMI and WHR among males and females from 1985 to 2001-2003

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*Key:*  
- current smokers
- never-smokers
- ex-smokers
Interactions between smoking and survey for the background variables

Age

Changes in the background factors *age*, *physical activity* and *university education* were investigated. Unadjusted values are presented graphically (Figure 4). For the independent variable *age*, there was a significant interaction between survey and current smoking among males (P = 0.005) and a non-significant tendency among females (P = 0.069). Figure 4 shows that average age among smokers increased relative to average age among never-smokers of both genders. Among male smokers and never-smokers, average age diverged in the figure; the average age of current smokers increased, while the average age of never-smokers remained constant (Figure 4).

Education

When the percentage of participants with *university education* was investigated as an independent variable, the interaction between survey and current smoking was significant among males (P = 0.007) but not among females (P = 0.114). This significant interaction indicates that the association between smoking and education is changing over time for males. Among both males and females, the figure illustrates that the percentage with university education was highest among never-smokers, followed by ex-smokers and finally current smokers. University education decreased among male smokers but increased in other groups, relative to never-smokers. Approximately 30% of never-smokers compared to 20% of current smokers had university education in 1985, and this difference approximately doubles from 10% to approximately 20% in 2001-2003 (Figure 4).

Physical activity

The statistical analyses revealed no significant interaction between survey and current smoking for males (P = 0.776) or females (P = 0.231) with physical activity as an independent variable. This suggests that there was no change in the relationship between physical activity and smoking during the study. The figure shows that physical activity was generally between 75 and 90%.

In summary, both average age and percentage with university education changed significantly among male smokers as compared to never-smokers during the study. It further can be observed graphically that these differences increased. One can observe that average age tended to increase in smokers while remaining stable in never-smokers, and that the percent with university education was stable or decreased among smokers and increased among never-smokers (Figure 4).
Figure 4. Unadjusted values for mean age, for percentage of participants with university education and for percentage who engage in regular physical activity according to smoking status and sex from 1985 to 2001-2003.
DISCUSSION

Result summary

This study investigated the relationship between smoking and the two anthropometric measures, BMI and WHR. The results show that the prevalence of smoking decreased, while both BMI and WHR increased from 1985 to 2001-2003. These results are in line with numerous Nordic and international studies on smoking and obesity, as described above in the Introduction.

Additionally, smoking was associated with high WHR among both males and females, as indicated by previous research (Table 2). Smoking was furthermore associated with low BMI among females, a result echoed in many studies (Table 1). No relationship between smoking and BMI was observed among males. This findings was at odds with most of the literature (Table 1) but was found in several Nordic studies (5-7). Ex-smoking was associated with high BMI and WHR among both genders.

When ascertaining changes in the association between smoking and the anthropometric variables over time, a significant result was obtained for WHR among females. Although neither WHR for males nor BMI for females reached significance, both revealed a similar pattern to WHR among females in that they increased more among current smokers than never-smokers during the study (Figure 3). Differences in BMI between male smokers and never-smokers were exceedingly small but showed a similar tendency. Turning to ex-smokers, no significant interactions or consistent patterns were found. The interactions both among smokers and ex-smokers were additionally adjusted for educational level, but the results were not appreciably affected (data not shown).

An investigation of effect modification by background variables showed that smoking was associated with increasingly higher average age and lower prevalence of university education among males. There were also similar but non-significant tendencies among females during the study. That is, average age among female smokers showed a tendency to increase as compared to never-smokers, and rate of university education among female never-smokers showed a tendency to increase with respect to female smokers. No effect modification was evident for physical activity.

Comparison between the results and the background literature

WHR

Previous studies that have compared WHR among smokers and never-smokers have found WHR to be greater for smokers than never-smokers. Although only one explored changes over time within a population, the studies’ results appeared to be consistent over several decades for various populations (Table 2). In accordance with the literature, the current study indicates that WHR is greater for smokers than never-smokers by approximately 0.015 units among both males and females.
Interestingly, the difference in WHR between smokers and never-smokers was not constant throughout the study; smokers had an increasingly high WHR as compared to never-smokers among females. A similar but non-significant pattern could be observed among males (Figure 3). In fact, both the average difference in WHR between smokers and never-smokers (0.014 for females and 0.015 for males) and the average change in this difference per survey (0.019 for females and 0.004 units for males) was small in terms of absolute value. Yet, it is noteworthy that WHR is a fraction, which thus can hide changes of several centimeters in waist and/or hip diameter. The ramifications of an increasing WHR among smokers include an increasing risk for morbidity and mortality associated with high WHR, including diabetes, stroke and heart disease, as compared to never-smokers (13, 52).

**BMI**

In the literature, smokers tend to have lower BMI than never-smokers (Table 1). This was confirmed cross-sectionally with an inverse dose-response effect of number of years of smoking duration on BMI (6, 43) and longitudinally, as smokers lost more/gained less in terms of BMI or weight than never-smokers between the early 1980s and 1990s (53-55). Starting smoking furthermore has been associated with a decrease in weight and that quitting smoking with an increase in weight (41).

However, more recent findings suggest that smokers do not necessarily have lower BMI than never-smokers. For example, in the above, two meta-studies, average BMI among smokers was not significantly lower than among never-smokers for over 25% of the each study’s populations (38, 39). In fact, studies from several Western countries have shown that BMI among smokers increased compared to never-smokers, suggesting that the relationship between smoking and fatness has changed over time (5-7). Furthermore, BMI among smokers is lower relative to never-smokers in older age cohorts and higher in younger age cohorts (6, 47). In summary, the negative cross-sectional relationship that was evident between smoking and obesity appears to have weakened in the past several decades.

Results from the present study were in accordance with these recent findings, in that smokers did not have a consistently higher BMI than never-smokers. Only among females was there an inverse relationship between smoking and BMI, and this difference appeared to decrease throughout the study (Figure 3). Among males, there was no significant difference between smokers’ and never-smokers’ BMI. This was a noteworthy finding, since several Nordic studies have found that BMI increased more quickly among male smokers than among male never-smokers (6, 7).

**Clustering of risk factors**

As the changing relationship between obesity and smoking is becoming increasingly well-documented, the next challenge is to explain this change. It is possible that overall
health awareness has increased to the degree that smoking is no longer accepted by the mainstream in certain countries. That is, in high-income countries, smoking was more commonplace several decades ago, and the lifestyles of smokers may not have differed much from those of never-smokers. More recently, the risks of smoking have been highly publicized, and smoking has become both less common and more marginalized in many societies e.g. smoking prohibitions in bars and restaurants (56). Thus, those who do smoke in such a country may be more likely to have additional risk factors and be appreciably less healthy more than their smoking counterparts. This is suggested in studies that have found risk factors such as high smoking intensity, obesity, low education, poor diet, high alcohol intake and physical inactivity to cluster together (57, 58). Similarly, male smokers in the current study became progressively older and less educated, and there was also a non-significant tendency among females suggesting that they have become progressively older.

Yet, it is also important to consider that no single risk factor can explain the changing relationship between smoking and obesity. For instance, one study found that reduction in smoking prevalence alone could not explain the simultaneous increase in overall obesity prevalence (59). In the present study, the gap between never-smokers and smokers increased among neither males nor females for physical activity, a risk factor that directly affects BMI and WHR (Figure 4). Thus, a complete explanation of the changing relationship between smoking and fatness appears to require consideration of the conjoint effects of multiple risk factors.

The effects of individual risk factors on this relationship also require further investigation. For example, when the effects of socioeconomic status (SES) on the relationship between smoking and fatness have been investigated, education has been inversely associated with BMI (45, 60, 61). That is, smokers weighed less than never-smokers among the least educated and the opposite was true for the most educated. Yet, when the MONICA study was adjusted for years of education, there was no change in the results (39). Similarly, the anthropometric results of the present study were not affected by adjusting for university education.

To further investigate how the relationship between smoking and obesity is affected by multiple risk factors, it would be interesting to collect comparable longitudinal data in which a variety of such risk factors were followed over time for individual participants. In the current sequential cross-sectional design, values were only reported at a group level, and one cannot disentangle whether the dependent variable varies due to 1) changes in the characteristics of a group’s membership over time and/or 2) individual change among the group members. Thus, if the increase in the difference between smokers’ and never-smokers’ BMI and WHR indeed is due to changes in group membership, these differences would not be evident in a longitudinal study (39). An examination of multiple risk factors could offer potential explanations for the findings in the present study’s anthropometric measures.
Validity and reliability of study

This study had participation rates that ranged from 63-84% in 1985, 55-74% in 1990, 52-69% in 1995, and 26-82% in 2001-2003 depending on age and gender (Table 3). Low participation suggests that the study results may not be representative and thus not generalizable to the source population. Decreasing participation over the four study years means furthermore that there is a risk that certain subgroups have become increasingly self-selected. If this is true, the results for each subsequent study date would be increasingly non-representative and external validity thus would decrease.

Internal validity, however, did not appear to be further compromised by non-response for individual items, as the rate generally did not exceed 5% missing data. Exceptions occurred in the final survey for WHR among females, which had 7% missing data, and for the education item, which had approximately 20% missing data (data not shown) and must be interpreted more cautiously.

Despite decreasing participation rates and missing data, this study’s prevalence estimates for smoking, BMI, WHR and the background variables were not necessarily biased. While some European studies on nonattendance have shown that participants differ from non-participants in smoking and obesity, in addition to age, gender, intelligence and SES (measured as educational level, income and/or social class) others have found that low participation rates did not affect prevalence estimates of either smoking or obesity (62-64). It is also important to note that relationships between risk factors have been shown to be resilient to low participation rates, even in such cases where estimates of the actual risk factor levels have been compromised (63-65). Thus, the decreasing participation rate in the present study may have affected prevalence estimates of smoking and fatness, but the association between these variables is likely to be less vulnerable to participation biases.

Another factor that potentially could affect the study’s validity was sampling differences between the surveys. The first three MONICA surveys employed age-stratified samples, whereas the final INTERGENE survey employed a simple random sample of the population. The risk was that the stratified MONICA samples misrepresented the population’s actual age structure, as compared to the INTERGENE sample. However, plotting the participation rate for each age group and survey period revealed no evident differences between MONICA and INTERGENE sampling distributions for neither anthropometric measures nor background variables (data not shown).

An additional concern related to validity is related to the differences in how several questions were worded between MONICA and INTERGENE could compromise the study’s validity. In MONICA, smoking was addressed by first asking about current smoking and then asking about previous smoking, whereas the opposite was true in INTERGENE. Yet, this did not necessarily affect the results, since the wording of these questions was nearly identical e.g. both studies used the word “regularly” to define smoking frequency. Another difference in the MONICA and INTERGENE questionnaires was that the latter included an additional, “other” category for education.
level. This difference could affect the results if someone with post-graduate education marked that category, but comments from participants indicated that those who marked this answer choice had not studied within the university context.

Measurement error is also likely to have been low. All anthropometric measures were taken by trained health personnel using a detailed, standard protocol and professional equipment. The self-report questions for smoking and education level were uncomplicated and expected to have high validity and reliability, particularly because other research has demonstrated these variables to have high repeatability (66).

Public health implications

The World Health Organization estimates that 86% of all deaths and 77% of all illness in Europe are due to non-communicable diseases, like cancer, mental health problems, heart disease, stroke and type 2 diabetes, the highest proportion in all WHO regions. They also estimate that at least 80% of premature heart disease, stroke and type 2 diabetes and 40% of cancer could be prevented through lifestyle factors, including healthy diet, regular physical activity and avoidance of tobacco products (67, 68). In accordance with other studies in developed countries, the current study indicates that the prevalence of smoking has decreased and the prevalence of obesity has increased in the source population. It furthermore suggests that smoking is increasingly associated with other risk factors e.g. central obesity, age and low level of education.

Both smoking and obesity have documented effects on mortality and morbidity. In 2005, tobacco was responsible for approximately 5.5 million deaths worldwide. In the next 20 years, deaths due to tobacco are expected to double in low and middle-income countries and to drop by 9% in high-income countries (69). However, smoking is still one of the leading causes of mortality, for example, accounting for 25-29% of all deaths in the WHO European region (33).

The prevalence of obesity and thus obesity-attributable morbidity and mortality are expected to increase in countries of all income levels. The burden is double in low and middle countries, which also must continue to deal with undernutrition. In the WHO European region, obesity accounts for 10-13% of all deaths (2).

Healthy life-years are estimated to be reduced three to eight years by smoking and one to two years by obesity (70-72). The risks associated with smoking accumulate over time and are thus most profound for long-term smokers (2), and the difference in morbidity and mortality between obese and non-obese adults is greater among young adults than among older adults (71). Even moderate overweight has been associated with higher all-cause mortality (2) and CVD (73, 74).

Furthermore, prevalence changes in smoking and obesity are directly related to subsequent health outcomes at the population level. For example, a 9% increase in smoking prevalence among females predicted an 11% increase in heart disease, while a
16% reduction in smoking prevalence among males predicted a 10% decline in death due to ischemic heart disease in a Finnish population (75).

In fact, both smoking and body fatness have been shown to be independently associated with numerous mortality and morbidity outcomes, including all-cause mortality and CVD, especially coronary events; cancer; diabetes; and hypertension (2, 73, 74, 76). Smoking and body fatness are additionally associated with respiratory, gastrointestinal, chronic musculoskeletal, skin and reproductive conditions. Moreover, it has been found that smoking and body fatness precede their associated mortality and morbidity outcomes in prospective studies, indicating that these two risk factors are chiefly causes rather than effects (2, 33). In the coming decades, smoking and obesity likely will continue to be primary risk factors behind morbidity and mortality, as pulmonary disorders, including cancers, and CVD are predicted to continue to be the leading causes of death and disability though the year 2020 (77).

Although smoking and obesity independently affect morbidity and mortality, the combined effect of these two risk factors is more controversial. Several studies show that the combined risk of smoking and obesity is the sum of these two risks for all-cause mortality (78) and mortality due to coronary heart disease (79). Other studies find an interaction, as the combined risk of smoking and obesity on coronary events is greater than would be expected by simply adding these two risks (78). Although it is unclear whether the combined effects of smoking and obesity on mortality and morbidity are synergistic, both risk factors clearly contribute.

Interestingly, smoking is associated both with slimness and with morbidity and mortality. In fact, the relationship between smoking and slimness is also well-documented on the population-level, as smoking is associated with lower BMI than non-smoking (38, 39), and on the physiological level, as smoking results in decreased appetite and higher energy expenditure (80). However, the health effects of smoking are so extensive and so varied that it should not be considered a healthy means of losing weight tool (56).

Smoking is furthermore associated with central obesity, which is related at least as strongly as general obesity to multiple CVD risk factors and mortality (10). Particularly because the relationship between smoking and central obesity appears to have become stronger during the past years, smoking cannot be viewed as an effective means of controlling central body fatness and its related consequences.

To understand whether the relationship between smoking and fatness is changing and, if so, in which way, one must investigate which additional factors may have disturbed the balance. Since it is difficult to identify genetic developments in humans that would affect their biological reactions to smoking and/or obesity in the last several decades, the answer must depend on environmental and/or societal changes. The first step to recognizing such a pattern is to determine the association between smoking and overweight in terms of strength, direction and variability over time in different populations and with regard to background variables. In the present study, female smokers and never-smokers differed increasingly with respect to central obesity. Male
smokers and never-smokers, on the other hand, differed increasingly by education and age over time. It was noteworthy that the relationship between smoking and physical activity did not change over time, which may suggest that improved measures of physical activity are needed in epidemiologic research. It would be interesting if future research focuses on additional aspects of lifestyle, including diet quality, which might change in parallel with changes in smoking habits. While these results suggest an increasing risk profile among smokers of both sexes, further studies are necessary to understand this phenomenon.
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