Body fat distribution in the Finnish population: environmental determinants and predictive power for cardiovascular risk factor levels

Bernard Marti, Jaakko Tuomilehto, Veikko Salomaa, Leena Kartovaara, Heikki J Korhonen, Pirjo Pietinen

Abstract

**Study objective**—The aim was to examine (1) whether health habits are associated with body fat distribution, as measured by the waist/hip girth ratio, and (2) to what extent environmental factors, including anthropometric characteristics, explain the variability in levels of cardiovascular risk factors.

**Design**—The study was a population based cross sectional survey, conducted in the spring of 1987 as part of an international research project on cardiovascular epidemiology.

**Setting**—The survey was conducted in three geographical areas of eastern and south western Finland.

**Subjects**—2526 men and 2756 women aged 25–64 years took part in the study, corresponding to a survey participation rate of 82%.

**Measurements and main results**—In men, waist/hip ratio showed stronger associations with exercise (Pearson’s \( r = -0.24 \)), resting heart rate (\( r = 0.10 \)), alcohol consumption (\( r = 0.07 \)), smoking (\( r = 0.05 \)), and education (\( r = -0.23 \)) than did body mass index. Jointly, exercise, resting heart rate, alcohol consumption, education, and age explained 18% of variance in male waist/hip ratio, but only 9% of variance in male body mass index. In women, environmental factors were more predictive for body mass index than for waist/hip ratio, with age and education being the strongest determinants. Waist/hip ratio and body mass index were approximately equally strong predictors of cardiovascular risk factor levels. The additional predictive power of waist/hip ratio over and above body mass index was tested in a hierarchical, stepwise regression. In this conservative type of analysis the increase in explained variance uniquely attributable to waist/hip ratio was 2–3% for female and 1–2% for male lipoprotein levels, and less than 0.5% for female and 0–2% for male blood pressure values.

**Conclusions**—The distribution of abdominal obesity in Finland is significantly influenced by health habits and sociodemographic factors in both men and women. This in turn is obviously one reason for the relatively small “independent” effect of body fat distribution on cardiovascular risk factor levels.

Recent epidemiological studies suggest that an excess deposition of fat in the abdominal region may be more predictive for the risk of myocardial infarction, stroke, and diabetes than is body mass.1–4 These observations have renewed the interest in the study of body fat distribution as a cardiovascular risk factor, a concept that was proposed by Vague more than 30 years ago.5 Recently, several studies have reported that excess abdominal fat, expressed as an increased ratio of waist to hip circumference, is independently associated with higher levels of blood pressure and serum total cholesterol, and lower levels of high density lipoprotein (HDL) cholesterol.6–13

Given the obvious importance of fat patterning as a determinant of health and the risk of disease, it is important to identify environmental factors that might influence this pattern.14 Based on the lack of substantial behavioural effects on the waist to hip girth ratio in the San Antonio Heart Study, Stern and Haffner15–16 argued that body fat distribution is primarily under genetic control. However, Bouchard et al17 and other genetic epidemiologists18 recently suggested that biological inheritance accounts for only a small part of the variance of fat distribution, and that non-genetic influences seem to contribute significantly to the amount and distribution of body fat in the population.

Population based data from Finland were thus analysed, addressing the following two questions: (1) to what extent are waist/hip ratio and body mass index dependent on individual health habits and sociodemographic factors, and (2) to what extent do environmental factors such as lifestyle and anthropometry explain the variability in levels of serum lipoproteins and blood pressure in the population? The present analysis used population based data recently collected in Finland as a part of the World Health Organization’s multifactorial project of monitoring trends and determinants in cardiovascular diseases (MONICA19–21).

Methods

In the Spring of 1987 the second cross sectional survey of risk factors of the Finnish part of the MONICA project was carried out in three areas in Finland.20 Independent random samples were drawn from these three populations covering the age range 25–64 years. Participation rate in the survey was 82%. The present analyses are based on data from 2526 men and 2756 women, for whom full information on all environmental factors and cardiovascular risk factors studied was available. The survey included a self administered...
questionnaire, checked by an interviewer, and physical measurements.

Weight, height, and girth of waist and hips were measured in light clothing by trained personnel. The body mass index (weight (kg) divided by squared height (m²)) was used as a measure of relative body weight. Horizontal circumferences were measured on subjects in the standing position, waist girth at a level midway between lower rib margin and the iliac crest, and hip girth as the widest circumference over the greater trochanter. Abdominal obesity was estimated by calculating the ratio of waist girth to hip girth.

Individual values of serum non-HDL cholesterol were calculated by subtracting HDL cholesterol from total serum cholesterol. Given the relevance of the concentration of non-HDL cholesterol for the risk of death from coronary heart disease as well as for the degree of atherosclerosis, non-HDL cholesterol rather than total serum cholesterol was included into all exploratory analyses. Laboratory and quality control methods of serum lipid analyses are explained elsewhere.

Based on recent evidence for the value of resting heart rate as a "surrogate measure" of physical fitness, resting heart rate was also included in the set of environmental factors in this study.

Information on smoking was obtained by seven standardised questions in the questionnaire and served for compilation of a seven point scale as an index of smoking, from 1 = never smoked to 7 = current smoker of ≥ 25 cigarettes per day. Information on alcohol drinking was obtained by nine questions, referring to the frequency of drinking beer, wine, mild alcoholic beverages, and strong alcoholic beverages, and the usual amount drunk. Average alcohol consumption in g/week (estimated on a one year basis) was then calculated by applying the average alcohol content and sizes of bottles or portions in Finland.

Dietary fat composition was assessed with seven questions, referring to the type of fat usually used on bread and used for baking and cooking, the type of milk usually drunk, and usual use of cream or milk in coffee. Based on this information a nine point scale of the type of dietary fat was computed so that higher index values corresponded to an increasing polyunsaturated/saturated fatty acid (P/S) ratio. In a validation study of a subsample of the Finnish MONICA population this qualitative index of dietary fat composition was correlated (r = 0.6 to 0.7) with the dietary P/S ratio as estimated from a 3 d food record.

Habitual physical activity during leisure and exercise were assessed by six questions, some of which referred to the quantity and some to the intensity of exercise. To combine these two dimensions of physical activity a five point scale was computed as an exercise index, with 1 = no, 2 = little low intensity exercise, and 5 = training vigorously at least 3 h/week and jogging (cross country skiing during winter time) more than 25 km/week. Educational level was expressed as the number of years of full time education. Responders also indicated in the questionnaire whether they were taking antihypertensive drugs, or any other medication because of heart disease. All subjects taking drugs for cardiovascular reasons were excluded from the multivariate analysis of the determinants of risk factor levels.

Statistical procedures included calculation of Pearson product-moment correlations to examine unadjusted associations, and stepwise multiple regression to evaluate the independent impact of single predictor variables on the dependent variables of interest, anthropometric characteristics, and cardiovascular risk factors. Although some of the predictor variables had a somewhat skewed distribution, no transformations were done in the final analyses as none of these transformations would have substantially improved linearity or strength of associations. A standard statistical software package (SPSSX, Statistical package for the social sciences, Chicago, IL, USA) was used for all analyses. Two sided p values < 0.05 were accepted as statistically significant.

Results

Table I shows a summary of descriptive statistics of the variables used in the following analyses. Table II shows behavioural and sociodemographic correlates of the two anthropometric characteristics under study, waist/hip ratio and body mass index. In both genders, age was the strongest determinant of the two variables. Education was inversely related to both variables, as was exercise, which was a consistent, inverse predictor in men as well as in women. In males, the type of dietary fat was unrelated to either variable, while

### Table I Descriptive statistics for health habits, anthropometric characteristics and cardiovascular risk factors in the study population, by sex.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men (n = 2526)</th>
<th>Women (n = 2756)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking (index)</td>
<td>3.00 ± 2.20</td>
<td>1.84 ± 1.59</td>
</tr>
<tr>
<td>Current regular smokers (%)</td>
<td>36.6</td>
<td>18.3</td>
</tr>
<tr>
<td>Exercise (index)</td>
<td>1.84 ± 1.19</td>
<td>1.61 ± 0.98</td>
</tr>
<tr>
<td>Alcohol consumption (g/week)</td>
<td>73.4 ± 141.9</td>
<td>14.4 ± 32.5</td>
</tr>
<tr>
<td>No alcohol at all (%)</td>
<td>11.5 ± 24.6</td>
<td>246 ± -</td>
</tr>
<tr>
<td>Type of dietary fat (index)</td>
<td>4.3 ± 2.57</td>
<td>4.9 ± 2.45</td>
</tr>
<tr>
<td>Education (No. of years)</td>
<td>9.8 ± 3.7</td>
<td>10.3 ± 3.7</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>26.68 ± 3.66</td>
<td>25.99 ± 4.80</td>
</tr>
<tr>
<td>Waist/hip girth ratio</td>
<td>0.905 ± 0.065</td>
<td>0.779 ± 0.061</td>
</tr>
<tr>
<td>Resting heart rate</td>
<td>71.8 ± 12.7</td>
<td>73.4 ± 12.0</td>
</tr>
<tr>
<td>Total serum cholesterol (mmol/litre)</td>
<td>6.08 ± 1.21</td>
<td>5.90 ± 1.26</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/litre)</td>
<td>1.29 ± 0.32</td>
<td>1.59 ± 0.34</td>
</tr>
<tr>
<td>Non-HDL cholesterol (mmol/litre)</td>
<td>4.79 ± 1.27</td>
<td>4.31 ± 1.26</td>
</tr>
<tr>
<td>HDL/non-HDL cholesterol ratio</td>
<td>0.298 ± 0.138</td>
<td>0.404 ± 0.155</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>141.5 ± 17.3</td>
<td>136.0 ± 20.4</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>86.5 ± 11.2</td>
<td>81.7 ± 11.3</td>
</tr>
<tr>
<td>Age (years)</td>
<td>44.5 ± 11.2</td>
<td>44.1 ± 11.3</td>
</tr>
</tbody>
</table>

**Notes:**
- Seven point scale, ranging from 1 = never smoked to 7 = current smoker of ≥ 25 cigarettes a day.
- Five point scale, ranging from 1 = no, or little low intensity exercise to 5 = training vigorously at least 3 h/week, and jogging (cross country skiing) more than 25 km/week.
- Average alcohol consumption in g/week, estimated from the frequency of drinking beer, wine, mild alcoholic beverages, and strong alcoholic beverages.
- Nine point scale based on the type of fat usually used on bread, for baking and cooking, the type of milk drunk, and use of cream or milk in coffee; higher index values indicate a decreasing proportion of saturated fat intake, i.e. an increase in the dietary P/S ratio.

### Table II Unadjusted correlations of health habits and sociodemographic factors with anthropometric characteristics.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Waist/hip girth ratio</td>
<td>0.05 ± 0.01</td>
<td>0.02 ± 0.01</td>
</tr>
<tr>
<td>Body mass index</td>
<td>0.02 ± 0.04</td>
<td>0.04 ± 0.04</td>
</tr>
</tbody>
</table>

Significance of correlation coefficients: p < 0.05 if (r) ≥ 0.04, and p < 0.001 if (r) ≥ 0.07.

a Seven point scale, ranging from 1 = never smoked to 7 = current smoker of ≥ 25 cigarettes a day.

b Average alcohol consumption in g/week, estimated from the frequency of drinking beer, wine, mild alcoholic beverages, and strong alcoholic beverages.

c Nine point scale based on the type of fat usually used on bread, for baking and cooking, the type of milk drunk, and use of cream or milk in coffee; higher index values indicate a decreasing proportion of saturated fat intake, i.e. an increase in the dietary P/S ratio.

Significance of correlation coefficients: p < 0.05 if (r) ≥ 0.04, and p < 0.001 if (r) ≥ 0.07.

a See Table I for explanation.
in women a lower proportion of saturated fat intake tended to correlate inversely with both waist/hip ratio and body mass index. Notably, in men aged 25–44 the correlation coefficient with waist/hip ratio was $r=0.12$ for alcohol consumption, 0.11 for smoking, and 0.14 for resting heart rate (all $p<0.001$), while in men aged 45–64 the corresponding correlation coefficients were 0.06 ($p<0.05$), 0.04 (NS), and 0.09 ($p<0.01$) respectively.

In the multiple linear regression analysis age was confirmed as the most important determinant of waist/hip ratio and body mass index in both men and women (table II). In women, the second most important predictor was education, while in men it was exercise. Alcohol consumption was a fairly strong predictor of waist/hip ratio in men. The five strongest behavioural and sociodemographic factors explained nearly twice as much variance of waist/hip ratio than of body mass index in men, whereas in women more of the variance in body mass index than waist/hip ratio was explained. When the same regression analysis was run separately in two age strata, 25–44 and 45–64 years, the models were clearly more predictive for waist/hip ratio and body mass index in younger than in older men, and slightly more predictive in older than in younger women. Given the significant proportion of variance in anthropometric characteristics explained by behavioural variables, waist/hip ratio and body mass index may also be regarded as important mediating factors between health habits and biological risk factors of cardiovascular disease. Both measures were approximately equally strong determinants of levels of serum lipoproteins and blood pressure (table IV), with little difference between genders. In women age was by far the strongest predictor of risk factor levels, while in men waist/hip ratio and body mass index were the strongest predictors.

Multiple linear regression analysis was used to estimate the overall impact of environmental factors on biological cardiovascular risk factor levels as well as the independent effect of single predictor variables (tables V and VI). The full model of nine environmental variables explained somewhat more of the variance in risk factor levels in women than in men. For example, smoking was a stronger inverse determinant of HDL cholesterol concentration in women than in men, and the type of dietary fat was also more related to the level of non-HDL cholesterol in women than in men. The proportion of saturated fat intake was significantly and directly related to systolic, but not diastolic blood pressure in both genders.

The independent predictive power of waist/hip ratio over and above body mass index was tested by entering waist/hip ratio into the regression equation explaining cardiovascular risk factor levels after the inclusion of health habits, education, and body mass index in the model (tables V and VI). In this conservative method of analysis the increase in variance in lipoprotein levels explained uniquely attributable to waist/hip ratio was 2.3% in women and 1.2% in men.

**Discussion**

This study addressed the question to what extent the two anthropometric characteristics waist/hip ratio and body mass index are dependent on health habits and sociodemographic variables. In men, waist/hip ratio was clearly more dependent on these factors than body mass index, while in women the opposite was true. The interrelationship between behavioural variables and waist/hip ratio may also explain why in men this measurement reached only comparatively modest though significant “independent” predictive power on risk factor levels in our study. Given the associations with exercise, smoking,
alcohol consumption, and resting heart rate, waist/hip ratio seems to be an important "marker of lifestyle" in Finnish men despite this modest statistical predictive power on risk factors. Especially in younger middle aged men an increased waist/hip ratio may be a relevant indicator of overall unfavourable health habits. A recent analysis of time trends has shown that the cluster of negative health habits associated with waist/hip ratio—including smoking, high alcohol and saturated fat intakes, and little exercise—even tends to become more visible in Finland.28

Age turned out to be the strongest determinant of both waist/hip ratio and body mass index in the sex specific regressions on behavioural and sociodemographic factors. Its statistical importance could, at first sight, be explained by an "intrinsic" effect of aging, which tends to increase both variables. However, we believe that in analyses like the present one age may also act as a "proxy variable" for several age related changes in exercise, which shows as a decrease in exercise, which themselves tend to increase waist/hip ratio and body mass index and which are insufficiently assessed with the few questionnaire variables used in this study. Therefore, our cross sectional analysis probably exaggerated the size of the true, biologically inevitable deteriorating effect of age on body fat and its distribution. Research into the aging processes has indeed shown that the magnitude of the intrinsic effects of aging is surprisingly modest compared with the effects of age related changes in behaviour.29 30

Our observation that, at least in men, waist/hip ratio depended significantly—and more than body mass index—on health habits, education, and age is not in accord with findings from the San Antonio Heart Study in Mexican Americans14 15 and with evidence from Canadian genetic epidemiology studies.17 The former study was unable to show a substantial effect of environmental factors on waist/hip ratio, and the latter suggested that for waist/hip ratio genetic factors may be more important, and the non-transmissible variance may be less, than for body mass index. Our data support this latter hypothesis only for women, but not for men.

Table V  Multiple linear regression analysis* (standardised regression coefficient = SRC; (p value); cumulated variance explained = R²) of cardiovascular risk factors on the environmental factors: Men.b

<table>
<thead>
<tr>
<th>Predictor variable</th>
<th>Non-HDL cholesterol SRC (p)</th>
<th>R²</th>
<th>HDL cholesterol SRC (p)</th>
<th>R²</th>
<th>HDL/non-HDL cholesterol SRC (p)</th>
<th>R²</th>
<th>Systolic blood pressure SRC (p)</th>
<th>R²</th>
<th>Diastolic blood pressure SRC (p)</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking (index)b</td>
<td>0.05 (0.014)</td>
<td></td>
<td>0.06 (0.002)</td>
<td></td>
<td>0.06 (0.003)</td>
<td></td>
<td>0.01 (0.070)</td>
<td></td>
<td>-0.07 (0.001)</td>
<td></td>
</tr>
<tr>
<td>Exercise (index)b</td>
<td>-0.06 (0.002)</td>
<td></td>
<td>0.11 (0.001)</td>
<td></td>
<td>0.09 (0.001)</td>
<td></td>
<td>0.09 (0.001)</td>
<td></td>
<td>0.06 (0.037)</td>
<td></td>
</tr>
<tr>
<td>Alcohol consumptionb</td>
<td>-0.06 (0.002)</td>
<td>6.00</td>
<td>0.20 (0.001)</td>
<td>5.40</td>
<td>0.17 (0.001)</td>
<td>6.80</td>
<td>0.13 (0.001)</td>
<td></td>
<td>3.70</td>
<td></td>
</tr>
<tr>
<td>Type of dietary fatb</td>
<td>-0.05 (0.015)</td>
<td></td>
<td>0.02 (0.001)</td>
<td></td>
<td>0.03 (0.001)</td>
<td></td>
<td>0.06 (0.001)</td>
<td></td>
<td>-0.06 (0.01)</td>
<td></td>
</tr>
<tr>
<td>Education (No years)</td>
<td>-0.07 (0.012)</td>
<td></td>
<td>0.07 (0.001)</td>
<td></td>
<td>0.02 (0.001)</td>
<td></td>
<td>0.05 (0.001)</td>
<td></td>
<td>0.01 (0.088)</td>
<td></td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>0.14 (0.001)</td>
<td>11.0</td>
<td>0.20 (0.001)</td>
<td>14.3</td>
<td>0.20 (0.001)</td>
<td>16.8</td>
<td>0.07 (0.001)</td>
<td></td>
<td>3.20</td>
<td></td>
</tr>
<tr>
<td>Waist/hip ratio</td>
<td>0.09 (0.001)</td>
<td>12.0</td>
<td>0.17 (0.001)</td>
<td>15.7</td>
<td>0.17 (0.001)</td>
<td>18.5</td>
<td>0.02 (0.001)</td>
<td></td>
<td>1.60</td>
<td></td>
</tr>
<tr>
<td>Resting heart rate</td>
<td>0.01 (0.540)</td>
<td>14.0</td>
<td>0.15 (0.002)</td>
<td>15.9</td>
<td>0.03 (0.001)</td>
<td>19.7</td>
<td>0.14 (0.001)</td>
<td></td>
<td>1.10</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>0.00 (0.001)</td>
<td>14.0</td>
<td>0.12 (0.001)</td>
<td>15.9</td>
<td>0.12 (0.001)</td>
<td>19.7</td>
<td>0.14 (0.001)</td>
<td></td>
<td>1.10</td>
<td></td>
</tr>
</tbody>
</table>

* Forward stepping procedure with forced entry of the following predictor variables in this order: five health habits (including education), BMI, waist/hip ratio, resting heart rate, age.

a 393 men taking antihypertensive or other cardiovascular drugs excluded from analysis.

b See table 1 for explanation.

Table VI  Multiple linear regression analysis* (standardised regression coefficient = SRC; (p value); cumulated variance explained = R²) of cardiovascular risk factors on the environmental factors: Women.b

<table>
<thead>
<tr>
<th>Predictor variable</th>
<th>Non-HDL cholesterol SRC (p)</th>
<th>R²</th>
<th>HDL cholesterol SRC (p)</th>
<th>R²</th>
<th>HDL/non-HDL cholesterol SRC (p)</th>
<th>R²</th>
<th>Systolic blood pressure SRC (p)</th>
<th>R²</th>
<th>Diastolic blood pressure SRC (p)</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking (index)b</td>
<td>0.05 (0.007)</td>
<td></td>
<td>0.11 (0.001)</td>
<td></td>
<td>0.09 (0.001)</td>
<td></td>
<td>0.05 (0.004)</td>
<td></td>
<td>-0.07 (0.001)</td>
<td></td>
</tr>
<tr>
<td>Exercise (index)b</td>
<td>-0.00 (0.11)</td>
<td></td>
<td>0.03 (0.001)</td>
<td></td>
<td>0.01 (0.001)</td>
<td></td>
<td>0.01 (0.021)</td>
<td></td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>Alcohol consumptionb</td>
<td>-0.07 (0.001)</td>
<td>14.4</td>
<td>0.14 (0.001)</td>
<td>4.20</td>
<td>0.13 (0.001)</td>
<td>12.3</td>
<td>0.01 (0.035)</td>
<td></td>
<td>10.9</td>
<td></td>
</tr>
<tr>
<td>Type of dietary fatb</td>
<td>-0.09 (0.001)</td>
<td></td>
<td>0.03 (0.001)</td>
<td></td>
<td>0.03 (0.001)</td>
<td></td>
<td>0.01 (0.003)</td>
<td></td>
<td>0.04</td>
<td></td>
</tr>
<tr>
<td>Education (No years)</td>
<td>-0.09 (0.006)</td>
<td></td>
<td>0.03 (0.002)</td>
<td></td>
<td>0.06 (0.002)</td>
<td></td>
<td>0.01 (0.003)</td>
<td></td>
<td>0.00</td>
<td></td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>0.03 (0.005)</td>
<td>17.1</td>
<td>0.17 (0.001)</td>
<td>9.90</td>
<td>0.15 (0.001)</td>
<td>20.1</td>
<td>0.12 (0.001)</td>
<td></td>
<td>1.91</td>
<td></td>
</tr>
<tr>
<td>Waist/hip ratio</td>
<td>0.16 (0.001)</td>
<td>20.3</td>
<td>0.19 (0.001)</td>
<td>12.1</td>
<td>0.19 (0.001)</td>
<td>23.0</td>
<td>0.04 (0.001)</td>
<td></td>
<td>1.72</td>
<td></td>
</tr>
<tr>
<td>Resting heart rate</td>
<td>0.04 (0.007)</td>
<td>20.4</td>
<td>0.13 (0.001)</td>
<td>12.3</td>
<td>0.01 (0.001)</td>
<td>23.0</td>
<td>0.16 (0.001)</td>
<td></td>
<td>1.72</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>0.04 (0.001)</td>
<td>30.9</td>
<td>0.24 (0.001)</td>
<td>12.9</td>
<td>0.24 (0.001)</td>
<td>26.5</td>
<td>0.36 (0.001)</td>
<td></td>
<td>27.3</td>
<td></td>
</tr>
</tbody>
</table>

* Forward stepping procedure with forced entry of the following predictor variables in this order: five health habits (including education), BMI, waist/hip ratio, resting heart rate, age.

a 383 women taking antihypertensive or other cardiovascular drugs excluded from analysis.

b See table 1 for explanation.
Plainly, sex itself is an overwhelmingly important determinant of waist/hip ratio. Population wide, this measurement followed a bimodal distribution with relatively little overlap between sexes, since the female mean value plus one standard deviation (0-84) corresponded to the male mean value minus one standard deviation (0-84). However, we did not evaluate sex statistically as a predictor variable of waist/hip ratio and performed only sex specific analyses.

The group of Stern, Haffner et al. was among the first to describe a significant positive association between smoking and waist/hip ratio—a finding confirmed in a large group of upper middle class North American men with an average in the ratio of independent predictors, Haffner et al were able to explain 12% of male, and 22% of female variance in the HDL/total cholesterol ratio in their sample of Mexican Americans. In our data, similar regression models explained a slightly larger variation in HDL/total cholesterol, 20% in men and 26% in women. Other workers have reported comparable or lower proportions of variance in risk factors that can be explained by environmental factors.

Our hierarchical stepwise regression analysis of risk factor levels in which waist/hip ratio was always entered after the behavioural variables and after body mass index into the model was a very "hard" test of the truly independent predictive power of waist/hip ratio. Like American workers who had adopted a similar statistical approach to test the effect of fat distribution on risk factor levels, we found that waist/hip ratio significantly and consistently contributed to the variation in risk factor levels. This was more pronounced for serum lipoprotein concentrations than for blood pressure, and it tended to be stronger in women than in men. For example, in the multiple linear regression of the HDL/total cholesterol ratio in women the standardised regression coefficient of waist/hip ratio approached that of age.

It is interesting to ask to what extent the observed male/female difference in waist/hip ratio may explain observed "typical" differences in serum cholesterol concentrations between men and women. Remarkably, both the simple correlation coefficients and the multivariable standardised regression coefficients for waist/hip ratio in the regression of HDL cholesterol were very similar in men and women. Thus waist/hip ratio predicted the HDL cholesterol level independently of sex. In the multiple linear regression analysis of HDL level on environmental factors, the parameter estimate of waist/hip ratio also adopted similar values in men and women (not shown). From the magnitude of these regression coefficients, it was estimated that one third to one half of the sex differential in HDL cholesterol concentration (ie, 0-1 to 0-15 of 0-3 mmol/litre) could be directly and independently attributed to the actual difference in mean waist/hip ratio observed between men and women. Correspondingly, 50 to 80% of the sex differential in non-HDL cholesterol concentration (ie, 0-25 to 0-4 of 0-5 mmol/litre) were estimated to be explained directly and independently by the difference in waist/hip ratio between sexes. Similar findings have recently been reported from North American adults in the seventh decade of life, even though the predictive power of waist/hip ratio for HDL levels was not tested statistically in presence of body mass index or any other measure of relative body weight in that study.

Björntorp has suggested that, compared with body mass index, waist/hip ratio is a stronger predictor of serum lipoprotein levels, and an equally strong predictor of blood pressure levels. We could not completely confirm this. In multivariate analysis, waist/hip ratio was indeed a better predictor of lipoprotein concentrations than body mass index in women but not in men; and in both genders body mass index was more strongly related to blood pressure than waist/hip ratio. Swedish studies did not take alcohol consumption and physical activity into account, which could lead to an overestimation of the effect of waist/hip ratio.

Our multivariate regression analysis of risk factor levels in a population based sample on health habits confirmed several cross sectional associations that have been described earlier. Habitual exercise related to a favourable serum lipoprotein profile, in men more than in women. A high dietary intake of saturated fat was consistently related to higher non-HDL cholesterol level and higher systolic blood pressure, but unrelated to HDL cholesterol. Interestingly, the regression coefficients of the type of dietary fat, corresponding to estimated P/S ratio, tended to be higher in women than in men, which could be due to a better accuracy in the self report of dietary habits in women. Alcohol consumption was positively related to the HDL cholesterol level in men as well as in women but only weakly related to diastolic and not at all related to systolic blood pressure. Given the irregular alcohol drinking pattern of the Finnish population, it may not be a surprise that we found a more long term effect of alcohol consumption on the HDL cholesterol level but no comparable short term effect on blood pressure in our study. Finally, we could confirm the recent observation that education is a strong and consistent determinant of anthropometric characteristics and cardiovascular risk factor levels, especially in women.

Several limitations of this study should be noted. Firstly, its cross sectional design does not permit any definitive interpretation of the direction of the observed associations. Our study could not, for example, address the question of time sequence of abdominal obesity in the natural history of coronary heart disease and increased risk factor levels. Therefore, a high waist/hip ratio could be either a cause or an effect of, for example, low exercise, or both. Our regression models explained between 14 and 21% of male and
between 13 and 31% of female variance in risk factor levels, more than in most similar studies. Clearly some of the interindividual differences in risk factor levels depend on health habits and related anthropometric characteristics. In epidemiological studies, categorising subjects by response to a single or few questions may be sufficient to detect apparent associations between a behavioural factor like exercise and a biological risk factor. However, misclassification, which is common in such a crude assessment, increases random error and thereby decreases the likelihood that a true relationship will be detected and makes it difficult to estimate the magnitude of such a relationship.

We argue that antihypertensive and other cardiovascular drugs may significantly lower blood pressure levels and may also modify serum lipid concentrations. These individuals have also been given specific advice to change their health habits. Therefore, it was decided to exclude them from multivariate analyses. This reduced the variation in blood pressure values and, to some extent, in blood lipids, which weakened the associations between these and other variables, e.g., waist/hip ratio.

Ultimately, the most substantial amount of variance in cardiovascular risk factor levels in the population might be explained by specific gene-environment interactions. For example, the associations between exercise and HDL cholesterol may be mediated by and thus depend on the largely genetically determined factor of muscle fibre composition.47 48

Another limitation of the present study is that the set of dependent variables was confined to risk factors of cardiovascular diseases. Obesity is considered as a risk factor for several chronic diseases, and it has been proposed that abdominal, or android, obesity in particular represents a separate clinical entity46 that is associated with increased risk for coronary heart disease, stroke, non-insulin-dependent diabetes mellitus, and female carcinomas. It has been hypothesised that endocrine differences observed between females with android obesity (increased waist/hip ratio) and females with gynoid obesity (low waist/hip ratio), for example irregular ovulation, longer menstrual cycles, lower levels of sex hormone binding globulin, and higher levels of free testosterone in women with android obesity, are related to the risk of endometrial cancer and breast cancer.46 However, preliminary evidence from population based studies for an excess risk of female cancer associated with android obesity is not consistent. In a cohort study of middle aged women from Gothenburg, Sweden, centrally localised adipose tissue (increased waist/hip ratio) was related to the risk of endometrial carcinoma but not to the risk of breast cancer.49 On the other hand, a recent case-control study of female breast cancer in the United States found a significant, direct and graded relation between waist/hip ratio and the risk of developing breast cancer.50 Thus, metabolic and hormonal abnormalities associated with increased abdominal fat accumulation as well as the potential health hazards related to android obesity merit further study in both women and men. Android, or abdominal, obesity can readily be identified by measuring waist and hip circumference and determining the waist/hip circumference ratio. Measurements of abdominal distribution of adipose tissue appear to be a valuable addition to the predictors of several chronic, non-communicable diseases.51

In summary, this study showed that variation in body fat distribution observed in the Finnish population is significantly influenced by health habits and sociodemographic factors in both men and women. This relation was, in turn, one important reason for the apparently small "independent" effect of abdominal obesity on cardiovascular risk factor levels. The joint presence of behavioural and anthropometric factors to explain variance, in levels of risk factors was between 15 and 30%.

We thank Ms Raali Vanninen for typing the manuscript.

Bernard Marti's work was supported by National Swiss Science Foundation (grant No 31–9255.87).


