# Trends in cardiovascular disease biomarkers and their socioeconomic patterning among adults in the Scottish population 1995 to 2009: cross-sectional surveys 

Joel William Hotchkiss, ${ }^{1}$ Carolyn Anne Davies, ${ }^{1}$ Linsay Gray, ${ }^{1}$ Catherine Bromley, ${ }^{2}$ Simon Capewell, ${ }^{3}$ Alastair Leyland ${ }^{1}$

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#### Abstract

Objectives: To examine secular and socioeconomic changes in biological cardiovascular disease risk factor and biomarker prevalences in the Scottish population. This could contribute to an understanding of why the decline in coronary heart disease mortality in Scotland has recently stalled along with persistence of associated socioeconomic inequalities. Design: Cross-sectional surveys. Setting: Scotland. Participants: Scottish Health Surveys: 1995, 1998, 2003, 2008 and 2009 (6190, 6656, 5497, 4202 and 4964 respondents, respectively, aged 25-64 years). Primary outcome measures: Gender-stratified, agestandardised prevalences of obesity, hypertension, hypercholesterolaemia and low high-density lipoprotein cholesterol blood concentration as well as elevated fibrinogen and C reactive protein concentrations according to education and social class groupings. Inequalities were assessed using the slope index of inequality, and time trends were assessed using linear regression. Results: The prevalence of obesity, including central obesity, increased between 1995 and 2009 among men and women, irrespective of socioeconomic position. In 2009, the prevalence of obesity (defined by body mass index) was $29.8 \%$ ( $95 \% \mathrm{Cl} 27.9 \%$ to $31.7 \%$ ) for men and $28.2 \%$ ( $26.3 \%$ to $30.2 \%$ ) for women. The proportion of individuals with hypertension remained relatively unchanged between 1995 and 2008/2009, while the prevalence of hypercholesterolaemia declined in men from 79.6\% ( $78.1 \%$ to $81.1 \%$ ) to $63.8 \% ~(59.9 \%$ to $67.8 \%$ ) and in women from $74.1 \%$ ( $72.6 \%$ to $75.7 \%$ ) to $66.3 \%$ ( $62.6 \%$ to $70.0 \%$ ). Socioeconomic inequalities persisted over time among men and women for most of the biomarkers and were particularly striking for the anthropometric measures when stratified by education. Conclusions: If there are to be further declines in coronary heart disease mortality and reduction in associated inequalities, then there needs to be a favourable step change in the prevalence of


## ARTICLE SUMMARY

## Article focus

- In Scotland, as in other developed countries, coronary heart disease mortality has substantially declined over time.
- This decline may have slowed among younger ages and there are still large socioeconomic inequalities in mortality.
- Examination of the secular and socioeconomic changes in biological cardiovascular disease risk factor and biomarker prevalences in the Scottish population.


## Key messages

- In Scotland, over a 14-year period since 1995, there has been a substantial increase in the prevalence of obesity with a persistence of large inequalities.
- At the same time, the prevalence of hypertension has changed little, while that of hypercholesterolaemia has declined, albeit from a very high level. Inequalities were generally smaller and, in the case of cholesterol in men, ill defined.
- Such trends can only serve to curb any further declines in coronary heart disease mortality and maintain associated inequalities.

Strengths and limitations of this study

- This study utilised data from nationally representative surveys conducted over a 14-year period.
- Bias may have been introduced by declining survey response levels. Differential non-response by the socioeconomically disadvantaged may lead to an underestimation of the magnitude of inequalities.
cardiovascular disease risk factors. This may require radical population-wide interventions.


## INTRODUCTION

The rapid absolute decline in Scottish coronary heart disease (CHD) mortality since the $1970 \mathrm{~s}^{1}$ has slowed from 1994 onwards for individuals $<55$ years of age. ${ }^{2}$ Such plateaus in CHD mortality rates have also been identified in the USA, Australia and England. ${ }^{3-5}$ Risk factor reduction is believed to account for at least half of the decline that has occurred, the remainder being mainly ascribed to advancements in treatment. ${ }^{6-8}$

There is a persistent socioeconomic gradient across CHD mortality in many countries, and in some places, inequalities may be widening. ${ }^{2}{ }^{9}$ Much of the excess CHD associated with socioeconomic disadvantage can be explained by the corresponding patterning in cardiovascular disease (CVD) risk factors. ${ }^{10-12}$

In Scotland, over a 13-year period leading up to 2008, there had been minimal decline in behavioural CVD risk factors and no substantial change in socioeconomic inequalities. ${ }^{13}$ Of further concern is the increase in selfreported diabetes and hypertension during this time. This study investigates the socioeconomic patterning of biological CVD risk factors and markers that are intermediate between behavioural risk factors and CHD, in Scotland between 1995 and 2009. Our hypothesis was that there would be stagnation in prevalence trends in these biomarkers for CVD and that this might provide some explanation for the worrying trends in CHD mortality.

## METHODS

## Survey method and sample

The 1995, 1998, 2003, 2008 and 2009 Scottish Health Surveys (SHeSs) are cross-sectional nationally representative surveys reporting the health and health-related behaviours (with emphasis on CVD) of people living in private households in Scotland; details are described elsewhere. ${ }^{14-18}$ Briefly, samples were selected using a multistage, stratified, clustered probability sampling design. Data were collected during two household visits: first by an interviewer and then by a nurse. During the face-to-face interviews, information such as age, gender, educational status and occupational social class was ascertained. Bodyweight was measured to the nearest 100 g using electronic scales, an estimate was requested from respondents that exceeded the scales' upper limit of 130 kg . Height was measured to the nearest millimetre using a stadiometer.

During the nurse visit, blood pressure measurements, details of prescription drug use and non-fasting venous blood samples, as well as waist and hip circumferences, were obtained. The latter were measured using a tape with an insertion buckle and recorded to the nearest millimetre with at least two measurements combined to provide a mean. The waist was defined as the midpoint between the lower rib and the upper margin of the iliac crest. Hips were measured at the widest circumference around the buttocks below the iliac crest.

Blood pressure readings were taken using an automated device with the informant in a seated position,
after a 5 min rest. Data used in this study are based on the mean of the second and third readings of participants that had not eaten, smoked, drunk alcohol or taken vigorous exercise in the 30 min preceding measurement. The first two surveys employed Dinamap 8100 monitors (Critikon, Tampa, Florida, USA), while from 2003, Omron HEM 907 devices (Omron Healthcare, Kyoto, Japan) were used. Regression equations generated from a calibration study were used to derive predicted Omron readings from Dinamap readings. ${ }^{15}$

The same laboratory conducted blood sample analyses except for the 1995 survey. Total cholesterol serum concentration was determined by a cholesterol oxidase assay. In 1995 and 1998, high-density lipoprotein (HDL) cholesterol serum concentration was estimated using the cholesterol oxidase assay following PTA precipitation. From 2003, a direct method was used for analysis. Serum C reactive protein was estimated from 1998 onwards using the N Latex CRP mono Immunoassay. Plasma fibrinogen concentration was estimated using a modification of the Clauss thrombin clotting method, again from 1998 with different analysing equipment used from 2008.

The 2008 and 2009 surveys were part of a continuous survey running until 2011; sample sizes were smaller than in previous surveys and the nurse interview was a subsample (tables 1 and 2). The main interview response proportion declined from $81 \%$ in 1995 to $56 \%$ in 2009. The surveys were age limited in 1995 (16-64 years) and in 1998 ( $2-74$ years). Analyses were restricted to individuals aged between 25 and 64 years at the time of interview for all surveys. The lower limit was adopted because educational achievement is believed to be relatively stable by this age, while the 1995 survey dictated the upper limit. To ensure nationally representative data, the nurse examination subsamples from $2008(\mathrm{n}=720)$ and $2009(\mathrm{n}=762)$ were merged. Pregnant women were excluded from all measurements and the blood sample collection.

## Risk factors and risk markers

Biomarkers were dichotomised, using thresholds that indicated increased risk of CVD, as follows: body mass index (BMI; weight (kg) divided by height squared $\left(\mathrm{m}^{2}\right)$ ) with obesity defined as $\geq 30 \mathrm{~kg} / \mathrm{m}^{2}{ }^{19}$; genderspecific definitions of excessive waist circumference (WC) were $\geq 102 \mathrm{~cm}$ for men, $\geq 88 \mathrm{~cm}$ for women ${ }^{19}$; waist-hip ratio (WHR; WC divided by hip circumference) excess was defined as $\geq 1$ and $\geq 0.85$ for men and women, respectively ${ }^{19}$; hypertension (uncontrolled) was reported for those individuals with systolic pressure $\geq 140 \mathrm{~mm} \mathrm{Hg}$ or diastolic pressure $\geq 90 \mathrm{~mm} \mathrm{Hg}$, irrespective of treatment ${ }^{20}$; hypercholesterolaemia was defined as a serum total cholesterol $\geq 5.0 \mathrm{mmol} / \mathrm{l}$, medication was ignored ${ }^{21}$; high-risk HDL cholesterol serum concentration was defined by gender-specific thresholds: $<1.0 \mathrm{mmol} / \mathrm{l}$ for men and $<1.2 \mathrm{mmol} / \mathrm{l}$ for women ${ }^{21}$; total cholesterol: HDL cholesterol ratio cut-off

Table 1 Characteristics of the 1995, 1998, 2003, 2008 and 2009 Scottish Health Surveys: interview samples restricted to 25-64-year old respondents (unweighted data)

|  | Scottish Health Survey |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  | 1995 | 1998 | 2003 | 2008 | 2009 |
| Sample size, n | 6910 | 6656 | 5497 | 4202 | 4964 |
| Mean age (SD) | 43.3 (11.6) | 43.8 (11.3) | 45.3 (10.9) | 46.0 (11.0) | 45.6 (11.0) |
| Gender, n (\%) |  |  |  |  |  |
| Male | 3049 (44.1) | 2969 (44.6) | 2437 (44.3) | 1839 (43.8) | 2136 (43.0) |
| Female | 3861 (55.9) | 3687 (55.4) | 3060 (55.7) | 2363 (56.2) | 2828 (57.0) |
| Ethnicity, n (\%) |  |  |  |  |  |
| White | 6852 (99.2) | 6571 (98.7) | 5357 (97.5) | 4066 (96.8) | 4819 (97.1) |
| Other | 55 (0.8) | 70 (1.1) | 103 (1.9) | 117 (2.8) | 112 (2.3) |
| Missing | 3 (0.0) | 15 (0.2) | 37 (0.7) | 19 (0.5) | 33 (0.7) |
| BMI, n (\%) |  |  |  |  |  |
| $<30 \mathrm{~kg} / \mathrm{m}^{2}$ | 5158 (74.6) | 4691 (70.5) | 3447 (62.7) | 2519 (59.9) | 2949 (59.4) |
| $\geq 30 \mathrm{~kg} / \mathrm{m}^{2}$, obese | 1218 (17.6) | 1359 (20.4) | 1207 (22.0) | 1090 (25.9) | 1270 (25.6) |
| Missing* | 534 (7.7) | 606 (9.1) | 843 (15.3) | 593 (14.1) | 745 (15.0) |
| Social class, n (\%) |  |  |  |  |  |
| I, professional | 297 (4.3) | 280 (4.2) | 316 (5.7) | 225 (5.4) | 277 (5.6) |
| II, managerial | 1644 (23.8) | 1696 (25.5) | 1646 (29.9) | 1232 (29.3) | 1670 (33.6) |
| IIINM, skilled non-manual | 1534 (22.2) | 1404 (21.1) | 1125 (20.5) | 889 (21.2) | 1011 (20.4) |
| IIIM, skilled manual | 1424 (20.6) | 1348 (20.3) | 984 (17.9) | 773 (18.4) | 839 (16.9) |
| IV, semi-skilled manual | 1111 (16.1) | 1177 (17.7) | 939 (17.1) | 703 (16.7) | 794 (16.0) |
| $V$, unskilled manual | 575 (8.3) | 468 (7.0) | 320 (5.8) | 253 (6.0) | 252 (5.1) |
| Other (armed forces, unknown) | 325 (4.7) | 283 (4.3) | 167 (3.0) | 127 (3.0) | 121 (2.4) |
| Education, n (\%) |  |  |  |  |  |
| Level, 3 degree level or above | 1793 (25.9) | 1727 (25.9) | 1482 (27.0) | 1177 (28.0) | 1458 (29.4) |
| Level, 2 intermediate | 1043 (15.1) | 1209 (18.2) | 1070 (19.5) | 1046 (24.9) | 1265 (25.5) |
| Level 1, end of compulsory schooling | 1839 (26.6) | 1723 (25.9) | 1641 (29.9) | 1156 (27.5) | 1337 (26.9) |
| No qualifications | 2226 (32.2) | 1978 (29.7) | 1274 (23.2) | 802 (19.1) | 888 (17.9) |
| Other qualifications or missing | 9 (0.1) | 19 (0.3) | 30 (0.5) | 21 (0.5) | 16 (0.3) |
| Nurse sample size selected, n | 6910 | 6656 | 5497 | 1193 | 1255 |
| Declined nurse | 306 (4.4) | 387 (5.8) | 867 (15.8) | 278 (23.3) | 275 (21.9) |
| Agreed to nurse visit but not completed | 525 (7.6) | 752 (11.3) | 913 (16.6) | 195 (16.3) | 218 (17.4) |
| Agreed to nurse visit and completed | 6079 (88.0) | 5517 (82.9) | 3717 (67.6) | 720 (60.4) | 762 (60.7) |

for increased risk was 4.5, irrespective of medication; for both fibrinogen and C reactive protein values in the highest quintile determined for each survey sample as a whole, stratified by gender, were defined increased risk. Total cholesterol and hypertension were investigated irrespective of drug treatment as this study was interested in risk marker changes, whatever the cause.

## Socioeconomic measures

Individual respondents' Registrar General's occupational social class was divided into seven categories I, II, III non-manual, III manual, IV, V and others (including armed forces/unknown). From 2003, the Registrar General's social class was not directly recorded, this necessitated its derivation from the National Statistics Socio-Economic Classification (NS-SEC). ${ }^{22} \quad 23$ The highest educational qualification that respondents achieved was divided into four levels: no qualifications, level 1: age at end of compulsory schooling $=16$ years from 1971, 15 years from 1947 and 14 years prior to 1947 (O grade, standard grade, GCSE or equivalent), level 2 (higher grade, A level, GSVQ advanced or equivalent
and HNC, HND, SVQ level 4 or 5 or equivalent) and level 3 (first degree, higher degree or professional qualifications). Between each survey, there was slight variation in the questions used to determine educational attainment to reflect changes in the education system.

## Statistical analyses

The prevalence of each risk factor was determined for the period 1995-2008/2009 according to social class or education; participants in the 'other' categories were excluded (tables 1 and 2). Individuals with missing data were excluded from affected analyses. Survey weights were used to adjust for disproportionate sampling, differing selection probabilities and differential nonresponse. Nurse sample and blood sample weights were available for the 2003 and 2008/2009 data sets. Genderstratified prevalences were age standardised (direct method) to the European standard population using 5year age groups. ${ }^{24}$ For each risk factor and survey year, the slope index of inequality (SII), an absolute measure of inequality, was estimated using the weighted least squares method. The SII is the linear regression

Table 2 Characteristics of the 1995, 1998, 2003, 2008 and 2009 Scottish Health Surveys: nurse samples restricted to 25-64-year old respondents (unweighted data)

|  | Scottish Health Survey |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  | 1995 | 1998 | 2003 | 2008 | 2009 |
| Nurse sample size, n | 6079 | 5517 | 3717 | 720 | 762 |
| Mean age (SD) | 43.4 (11.5) | 44.1 (11.2) | 45.9 (10.8) | 46.5 (10.9) | 46.3 (11.1) |
| Gender, n (\%) |  |  |  |  |  |
| Male | 2703 (44.5) | 2479 (44.9) | 1614 (43.4) | 325 (45.1) | 315 (41.3) |
| Female | 3376 (55.5) | 3038 (55.1) | 2103 (51.5) | 395 (54.9) | 447 (58.7) |
| Waist circumference, n (\%) |  |  |  |  |  |
| $\mathrm{M}<102$; W <88 cm | 4697 (77.3) | 4035 (73.1) | 2337 (62.9) | 410 (56.9) | 428 (56.2) |
| $M \geq 102$; $W \geq 88 \mathrm{~cm}$ | 1215 (20.0) | 1374 (24.9) | 1237 (33.3) | 296 (41.1) | 319 (41.9) |
| Missing | 167 (2.7) | 108 (2.0) | 143 (3.8) | 14 (1.9) | 15 (2.0) |
| Waist-hip ratio, n (\%) |  |  |  |  |  |
| $\mathrm{M}<1.0$; $\mathrm{W}<0.85$ | 5052 (83.1) | 4569 (82.8) | 2697 (72.6) | 507 (70.4) | 523 (68.6) |
| $\mathrm{M} \geq 1.0$; $\mathrm{W} \geq 0.85$ | 831 (13.7) | 835 (15.1) | 868 (23.4) | 199 (27.6) | 224 (29.4) |
| Missing | 196 (3.2) | 113 (2.0) | 152 (4.1) | 14 (1.9) | 15 (2.0) |
| Hypertension* (uncontrolled), n (\%) |  |  |  |  |  |
| SBP <140, DBP < 90 mm Hg | 4145 (68.2) | 3646 (66.1) | 2326 (62.6) | 484 (67.2) | 507 (66.5) |
| SBP $\geq 140, \mathrm{DBP} \geq 90 \mathrm{~mm} \mathrm{Hg}$ | 845 (13.9) | 744 (13.5) | 681 (18.3) | 130 (18.1) | 144 (18.9) |
| Missing | 1089 (17.9) | 1127 (20.4) | 710 (19.1) | 106 (14.7) | 111 (14.6) |
| Blood sample, n (\%) |  |  |  |  |  |
| Yes | 5498 (90.4) | 4660 (84.5) | 2984 (80.3) | 607 (84.3) | 618 (81.1) |
| No-refused | 315 (5.2) | 394 (7.1) | 379 (10.2) | 53 (7.4) | 71 (9.3) |
| Missing $\dagger$ | 266 (1.7) | 463 (8.4) | 355 (9.5) | 60 (8.3) | 73 (9.6) |
| Total cholesterol, n (\%) $\ddagger$ |  |  |  |  |  |
| $<5.0 \mathrm{mmol}$ | 1267 (20.8) | 1458 (26.4) | 755 (20.3) | 182 (25.3) | 175 (23.0) |
| $\geq 5.0 \mathrm{mmol} / \mathrm{l}$ | 4184 (68.8) | 3124 (56.6) | 2115 (56.9) | 400 (55.6) | 404 (53.0) |
| Missing | 628 (10.3) | 935 (16.9) | 847 (22.8) | 138 (19.2) | 183 (24.0) |
| HDL cholesterol, n (\%)§ |  |  |  |  |  |
| $\mathrm{M} \geq 1.0 ; \mathrm{W} \geq 1.2 \mathrm{mmol} / \mathrm{l}$ | 4574 (75.2) | 3755 (68.1) | 2515 (67.7) | 484 (67.2) | 486 (63.8) |
| $\mathrm{M}<1.0 ; \mathrm{W}<1.2 \mathrm{mmol} / \mathrm{l}$ | 789 (13.0) | 716 (13.0) | 200 (5.4) | 41 (5.7) | 37 (4.9) |
| Missing | 716 (11.8) | 1046 (19.0) | 1002 (27.0) | 195 (27.1) | 239 (31.4) |
| Total cholesterol: HDL cholesterol ratio, n (\%) $\ddagger$ § |  |  |  |  |  |
| <4.5 | 3272 (53.8) | 2957 (53.6) | 1910 (51.4) | 389 (54.0) | 385 (50.5) |
| $\geq 4.5$ | 2091 (34.4) | 1513 (27.4) | 804 (21.6) | 136 (18.9) | 138 (18.1) |
| Missing | 716 (11.8) | 1047 (19.0) | 1003 (27.0) | 195 (27.1) | 239 (31.4) |
| Fibrinogen, $\mathrm{n}(\%) \mathbb{T}$ |  |  |  |  |  |
| Four quintiles | - | 3399 (61.6) | 2028 (54.6) | 388 (53.9) | 405 (53.1) |
| Highest quintile | - | 800 (14.5) | 473 (12.7) | 102 (14.2) | 82 (10.8) |
| Missing |  | 1318 (23.9) | 1216 (32.7) | 230 (31.9) | 275 (36.1) |
| C reactive protein, n (\%)** |  |  |  |  |  |
| Four quintiles | - | 3573 (64.8) | 2288 (61.6) | 462 (64.2) | 475 (62.3) |
| Highest quintile | - | 937 (17.0) | 570 (15.3) | 120 (16.7) | 104 (13.6) |
| Missing |  | 1007 (18.3) | 859 (23.1) | 138 (19.2) | 183 (24.0) |
| Social class, n (\%) |  |  |  |  |  |
| I, professional | 265 (4.4) | 228 (4.1) | 221 (5.9) | 38 (5.3) | 46 (6.0) |
| II, managerial | 1481 (24.4) | 1427 (25.9) | 1138 (30.6) | 238 (33.1) | 291 (38.2) |
| IIINM, skilled non-manual | 1356 (22.3) | 1187 (21.5) | 776 (20.9) | 149 (20.7) | 155 (20.3) |
| IIIM, skilled manual | 1261 (20.7) | 1120 (20.3) | 643 (17.3) | 132 (18.3) | 112 (14.7) |
| IV, semi-skilled manual | 965 (15.9) | 971 (17.6) | 640 (17.2) | 111 (15.4) | 110 (14.4) |
| $\checkmark$, unskilled manual | 488 (8.0) | 381 (6.9) | 207 (5.6) | 36 (5.0) | 37 (4.9) |
| Other (armed forces, unknown) | 263 (4.3) | 203 (3.7) | 92 (2.5) | 16 (2.2) | 11 (1.4) |
|  |  |  |  |  | Continued |

Table 2 Continued

|  | Scottish Health Survey |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  | 1995 | 1998 | 2003 | 2008 | 2009 |
| Education, n (\%) |  |  |  |  |  |
| Level 3, degree level or above | 1610 (26.5) | 1441 (26.1) | 1072 (28.8) | 223 (31.0) | 248 (32.5) |
| Level 2, intermediate | 925 (15.2) | 1023 (18.5) | 750 (20.2) | 183 (25.4) | 203 (26.6) |
| Level 1, end of compulsory schooling | 1644 (27.0) | 1432 (26.0) | 1084 (29.2) | 185 (25.7) | 185 (24.3) |
| No qualifications | 1894 (31.2) | 1614 (29.3) | 808 (21.7) | 129 (17.9) | 125 (16.4) |
| Other qualifications or missing | 6 (0.1) | 7 (0.1) | 3 (0.1) | - | 1 (0.1) |

*1995 and 1998 blood pressure readings were taken using Dinamap monitors and then converted to Omron monitor readings.
$\dagger$ Reasons for a missing blood sample included failed collection, pregnancy or a clotting disorder.
$\ddagger \ln 1995$, a different laboratory completed analyses. Different analysers were used but methods and reference standards were the same.
§Determined in 1995 and 1998 using a different method, not comparable to 2003 and 2008/2009.
|Thresholds for highest quintile of plasma fibrinogen concentration 1998: $\mathrm{M}>3.1 \mathrm{~g} / \mathrm{l}, \mathrm{W}>3.3 \mathrm{~g} / \mathrm{l}$; 2003: $\mathrm{M}>3.3 \mathrm{~g} / \mathrm{l}, \mathrm{W}>3.6 \mathrm{~g} / \mathrm{l}$; 2008/2009:
$\mathrm{M}>3.4 \mathrm{~g} / \mathrm{l}$, $\mathrm{W}>3.6 \mathrm{~g} / \mathrm{l}$.
${ }^{* *}$ Thresholds for highest quintile of serum C reactive protein concentration 1998 : $\mathrm{M}>3.4 \mathrm{mg} / \mathrm{l}, \mathrm{W}>4.3 \mathrm{mg} / \mathrm{l} ; 2003$ : $\mathrm{M}>3.5 \mathrm{mg} / \mathrm{l}, \mathrm{W}>4.8 \mathrm{mg} / /$; 2008/2009: $\mathrm{M}>3.71 \mathrm{mg} / \mathrm{l}, \mathrm{W}>4.80 \mathrm{mg} / \mathrm{l}$. Pregnant women were excluded from all measurements and the blood sample.
DBP, diastolic blood pressure; HDL, high-density lipoprotein; M, manual; M, men; NM, non-manual; SBP, systolic blood pressure; W, women.
coefficient for the relationship between the prevalence of each risk factor in each socioeconomic category and the hierarchical ranking of each socioeconomic group category on the social scale. ${ }^{25}$ It can be interpreted as the absolute change in health level when an individual goes from the highest status to the lowest status in the social hierarchy. In this context, a positive SII indicates that there is higher prevalence of an adverse CVD risk factor in the most disadvantaged compared with the least disadvantaged in the socioeconomic hierarchy. A positive SII is therefore bad from a public health perspective. Time trends for prevalence and the SIIs were assessed using generalised linear regression. For the aggregated 2008/2009 data sets, the year was taken as 2008.5. All analyses were performed using SAS V.9.2 (SAS Institute Inc.), and statistical significance was taken as $\mathrm{p}<0.05$.

## Sensitivity analyses

Individuals taking drugs affecting blood pressure or lipid-lowering drugs were, respectively, excluded from analyses involving hypertension and hypercholesterolaemia. Also a definition of hypertension that included respondents on drugs prescribed for high blood pressure, irrespective of measurements, was investigated (1998 onwards). Similarly, the effect of including individuals taking lipid-lowering drugs in the high-risk total cholesterol group was explored. The proportion of individuals taking either drugs prescribed for hypertension or lipid-lowering drugs was also investigated. Limited within-survey response analyses were undertaken by comparing individuals that did not complete the nurse interview or give a blood sample with those that did comply with these requests; details of analyses and results are provided in section 3 of the supplementary information.

## RESULTS

The participants' mean age increased across the five surveys, from 43.3 years in 1995 to 45.6 years in 2009
(table 1). The proportion of women was greater than that of men in all the surveys (tables 1 and 2). Over time, the population distribution shifted according to both socioeconomic measures. In general, the proportion of individuals in the 'upper' strata (eg, managerial class or level 3 qualifications) increased compared with those in the 'lower' strata (eg, unskilled class or no qualifications). Those that did not complete a nurse interview or a blood sample collection were more likely to be younger and to be smokers in almost all the surveys (supplementary tables 11-20). In the majority of the surveys, those from a lower socioeconomic background were less likely to have participated in later stages of the survey, particularly for the blood sample.

Overall, for individuals with data on education, the prevalence of obesity according to BMI increased by $0.7 \%$ per year $(\mathrm{p}<0.001)$ for men and $0.6 \%$ per year ( $\mathrm{p}<0.001$ ) for women (table 3). The fastest rate of increase was among unqualified men ( $0.9 \%$ per year, $\mathrm{p}=0.02$ ) reaching a prevalence of $34.6 \%$ ( $95 \%$ CI $28.1 \%$ to $41.1 \%$ ) in 2009. An educational differential was maintained in all the surveys; in 2009, the SII was 12.4 ( $\mathrm{p}=0.08$ ). The findings were similar for women; in 2009, those with no qualifications had the highest obesity prevalence ( $34.9 \%, 27.5 \%$ to $42.3 \%$ ) and the SII was 17.4 $(\mathrm{p}=0.004)$. There were social class inequalities in obesity among women, although there was an overall decline in the SII from $1995(-0.4, \mathrm{p}=0.11)$ (table 4 ), while they were relatively small for men.

As for BMI, there were significant, or borderline significant, increases in the prevalence of excess adiposity in terms of both WC and WHR (tables 3 and 4). The proportion of men with excessive WHR was much lower than the proportions with extreme BMI or WC. In 1995, the three anthropometric measures yielded similar proportions of women with excess adiposity; however, by 2008/2009, a greater proportion had extreme values according to WC and WHR than BMI. Large educational inequalities existed for men and women for both WC
Table 3 Gender-specific, age-standardised prevalences for three anthropometric measures of excess adiposity in five Scottish Health Surveys 1995, 1998, 2003, 2008 and 2009 stratified by highest educational qualification achieved

Table 3 Continued

and WHR. Over half of unqualified women had an excessive WC (52.6\%, 43.5\% to 61.6\%) and WHR ( $56.4 \%, 47.2 \%$ to $65.7 \%$ ) in $2008 / 2009$. For women, there was a significant increase in the WHR SII across education of 1.1 per year ( $p<0.001$ ). There were also social class inequalities in WC and WHR (table 4).

Overall prevalence of hypertension (uncontrolled) in both sexes did not change significantly over time, although the trend was upwards (table 5). Prevalences generally increased between 1998 and 2003, coinciding with the change from predicted to actual Omron readings. Socioeconomic inequalities in hypertension were relatively small with the exception of those for social class among men in 2008/2009 (SII=13.9, $\mathrm{p}=0.05$ ) (table 5). The larger inequalities partly reflect an increase in prevalence of $1.1 \%$ per year $(p=0.01)$ among unskilled men from 1995. Sensitivity analyses, excluding respondents taking drugs affecting blood pressure or including those taking drugs prescribed for hypertension, identified similar socioeconomic patterns (supplementary tables 3 and 4). The proportion of respondents taking drugs prescribed for hypertension increased across the surveys with inequalities widening among men, those from lower in the hierarchy having a greater increase in uptake (supplementary tables 9 and 10).

Hypercholesterolaemia declined across the surveys: from 1995 to 2008/2009, among those with educational information, the prevalence decreased from $79.6 \%$ ( $78.1 \%$ to $81.1 \%$ ) to $63.8 \% ~(59.9 \%$ to $67.8 \%$ ) for men and from $74.1 \%$ ( $72.6 \%$ to $75.7 \%$ ) to $66.3 \%$ ( $62.6 \%$ to $70.0 \%$ ) for women (table 6). For both education and social class, inequalities were minimal for men except by education in 2008/2009. According to education, in the first three surveys, positive SIIs existed for women but in $2008 / 2009$, the SII was $-16.9(\mathrm{p}=0.23)$, while for social class, SIIs were generally small and positive (table 7). Sensitivity analyses, excluding those on lipid-lowering drugs or including them in the high-risk hypercholesterolaemia group, had minimal impact on estimates until the 2008/2009 survey where prevalences estimates were elevated, particularly among men (supplementary tables 5 and 6). The prevalence of individuals taking lipid-lowering drugs increased across the surveys while inequalities in uptake existed, with educational inequalities widening (supplementary tables 9 and 10).

There was minimal change in the prevalence of low HDL cholesterol between comparable surveys: namely 1995-1998 (supplementary tables 1 and 2) and 2003 to 2008/2009 (tables 6 and 7). There were significant educational inequalities for women in all surveys, in 2008/2009 the SII=11.9 ( $\mathrm{p}=0.01$ ) (table 6). Absolute inequalities for men were generally smaller than in women and in the case of social class were not consistent. The proportion with high total cholesterol: HDL cholesterol ratio decreased between comparable surveys (supplementary tables 7 and 8). Strong inequalities existed for women, while for men they were minimal.
Table 4 Gender-specific, age-standardised prevalences for three anthropometric measures of excess adiposity in five Scottish Health Surveys 1995, 1998, 2003, 2008 and 2009 stratified by occupational social class

Table 4 Continued

Table 5 Gender-specific, age-standardised prevalences for hypertension (uncontrolled) in five Scottish Health Surveys 1995, 1998, 2003 and 2008/2009 stratified by highest educational qualification achieved and occupational social class


[^1]Table 6 Gender-specific, age-standardised prevalences for hypercholesterolaemia and low-serum HDL cholesterol concentration in five Scottish Health Surveys 1995, 1998, 2003 and 2008/2009 stratified by highest educational qualification achieved

|  | Men |  |  |  |  | Women |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Proportion (\%) (95\% CI) |  |  |  | Trend (p value) | Proportion (\%) (95\% CI) |  |  |  | Trend (p value) |
|  | 1995 | 1998 | 2003 | 2008/2009* |  | 1995 | 1998 | 2003 | 2008/2009* |  |
| Hypercholesterolaemia $\dagger$ |  |  |  |  |  |  |  |  |  |  |
| All | $\begin{aligned} & 79.6 \\ & (78.1 \text { to } 81.1) \end{aligned}$ | $\begin{aligned} & 69.5 \\ & (67.7 \text { to } 71.3) \end{aligned}$ | $\begin{aligned} & 71.8 \\ & \text { (69.4 to } 74.2 \text { ) } \end{aligned}$ | $\begin{aligned} & 63.8 \\ & (59.9 \text { to } 67.8) \end{aligned}$ | -1.0 (0.14) | $\begin{aligned} & 74.1 \\ & \text { (72.6 to } 75.7 \text { ) } \end{aligned}$ | $\begin{aligned} & 65.8 \\ & (64.0 \text { to } 67.7) \end{aligned}$ | $\begin{aligned} & 69.3 \\ & (67.0 \text { to } 71.5) \end{aligned}$ | $\begin{aligned} & 66.3 \\ & (62.6 \text { to } 70.0) \end{aligned}$ | -0.4 (0.40) |
| Level 3 | $\begin{aligned} & 80.7 \\ & (77.3 \text { to } 84.2) \end{aligned}$ | $\begin{aligned} & 69.1 \\ & (65.1 \text { to } 73.2 \text { ) } \end{aligned}$ | $\begin{aligned} & 72.2 \\ & (68.0 \text { to } 76.5) \end{aligned}$ | $\begin{aligned} & 60.6 \\ & (53.7 \text { to } 67.5) \end{aligned}$ | -1.2 (0.13) | $\begin{aligned} & 72.3 \\ & (69.5 \text { to } 75.1) \end{aligned}$ | $\begin{aligned} & 66.7 \\ & (63.5 \text { to } 70.0) \end{aligned}$ | $\begin{aligned} & 67.4 \\ & (63.3 \text { to } 71.5) \end{aligned}$ | $\begin{aligned} & 74.2 \\ & (68.5 \text { to } 80.0) \end{aligned}$ | 0.2 (0.66) |
| Level 2 | $\begin{aligned} & 79.3 \\ & (76.1 \text { to } 82.5) \end{aligned}$ | $\begin{aligned} & 69.9 \\ & (66.4 \text { to } 73.5) \end{aligned}$ | $\begin{aligned} & 71.5 \\ & (66.4 \text { to } 76.6) \end{aligned}$ | $\begin{aligned} & 65.0 \\ & (57.9 \text { to } 72.1) \end{aligned}$ | -0.9 (0.14) | $\begin{aligned} & 75.4 \\ & \text { (71.2 to } 79.7 \text { ) } \end{aligned}$ | $\begin{aligned} & 66.9 \\ & (62.4 \text { to } 71.3) \end{aligned}$ | $\begin{aligned} & 63.9 \\ & (58.8 \text { to } 69.1) \end{aligned}$ | $\begin{aligned} & 65.9 \\ & (59.0 \text { to } 72.8) \end{aligned}$ | -0.6 (0.28) |
| Level 1 | $\begin{aligned} & 79.3 \\ & (76.7 \text { to } 81.8) \end{aligned}$ | $\begin{aligned} & 69.5 \\ & (66.2 \text { to } 72.8) \end{aligned}$ | $\begin{aligned} & 72.7 \\ & (68.5 \text { to } 76.9) \end{aligned}$ | $\begin{aligned} & 68.9 \\ & (58.5 \text { to } 79.3) \end{aligned}$ | -0.6 (0.31) | $\begin{aligned} & 73.2 \\ & (70.0 \text { to } 76.4) \end{aligned}$ | $\begin{aligned} & 63.4 \\ & (59.5 \text { to } 67.3) \end{aligned}$ | $\begin{aligned} & 72.0 \\ & (67.6 \text { to } 76.4) \end{aligned}$ | $\begin{aligned} & 57.9 \\ & (49.9 \text { to } 66.0) \end{aligned}$ | -0.8(0.35) |
| No qualifications | $\begin{aligned} & 80.3 \\ & (77.2 \text { to } 83.4) \end{aligned}$ | $\begin{aligned} & 69.5 \\ & (65.3 \text { to } 73.8) \end{aligned}$ | $\begin{aligned} & 67.7 \\ & (56.1 \text { to } 79.4) \end{aligned}$ | $\begin{aligned} & 70.2 \\ & (58.8 \text { to } 81.5) \end{aligned}$ | -0.6 (0.36) | $\begin{aligned} & 78.1 \\ & (75.1 \text { to } 81.1) \end{aligned}$ | $\begin{aligned} & 70.0 \\ & (66.1 \text { to } 73.9) \end{aligned}$ | $\begin{aligned} & 75.1 \\ & (69.6 \text { to } 80.6) \end{aligned}$ | $\begin{aligned} & 65.6 \\ & (55.3 \text { to } 75.9) \end{aligned}$ | $-0.7(0.26)$ |
| SII (p value) Low HDL cholestero (direct method) $\ddagger$ | -0.1 (0.94) | 0.2 (0.79) | -3.8 (0.41) | 13.3 (0.006) | 0.9 (0.31) | 8.0 (0.12) | 3.8 (0.56) | 11.6 (0.20) | -16.9 (0.23) | $-1.6(0.28)$ |
| All | - | - | $\begin{aligned} & 6.6 \\ & (5.2 \text { to } 8.0) \end{aligned}$ | $\begin{aligned} & 7.1 \\ & (4.9 \text { to } 9.3) \end{aligned}$ | 0.1 | - | - | $\begin{aligned} & 9.3 \\ & (7.7 \text { to } 10.8) \end{aligned}$ | $\begin{aligned} & 8.4 \\ & (6.0 \text { to 10.7) } \end{aligned}$ | -0.2 |
| Level 3 | - | - | $\begin{aligned} & 3.0 \\ & (1.2 \text { to } 4.7) \end{aligned}$ | $\begin{aligned} & 5.4 \\ & (2.2 \text { to } 8.5) \end{aligned}$ | 0.4 | - | - | $\begin{aligned} & 6.9 \\ & (4.4 \text { to } 9.3) \end{aligned}$ | $\begin{aligned} & 4.3 \\ & (1.3 \text { to } 7.3) \end{aligned}$ | -0.5 |
| Level 2 | - | - | $\begin{aligned} & 7.4 \\ & (4.2 \text { to } 10.6) \end{aligned}$ | $\begin{aligned} & 7.6 \\ & (3.1 \text { to 12.0) } \end{aligned}$ | 0.0 | - | - | $\begin{aligned} & 7.9 \\ & (4.8 \text { to } 11.0) \end{aligned}$ | $\begin{aligned} & 8.9 \\ & (4.3 \text { to 13.5) } \end{aligned}$ | 0.2 |
| Level 1 | - | - | $\begin{aligned} & 8.3 \\ & (5.6 \text { to 11.1) } \end{aligned}$ | $\begin{aligned} & 6.1 \\ & (1.5 \text { to } 10.6) \end{aligned}$ | -0.4 | - | - | $\begin{aligned} & 10.8 \\ & (7.6 \text { to 14.0) } \end{aligned}$ | $\begin{aligned} & 11.6 \\ & \text { (5.6 to 17.6) } \end{aligned}$ | 0.1 |
| No qualifications | - | - | $\begin{aligned} & 8.3 \\ & (3.6 \text { to 12.9) } \end{aligned}$ | $\begin{aligned} & 14.6 \\ & (5.4 \text { to 23.8) } \end{aligned}$ | 1.2 | - | - | $\begin{aligned} & 10.8 \\ & (7.1 \text { to 14.6) } \end{aligned}$ | $\begin{aligned} & 12.8 \\ & (6.0 \text { to 19.6) } \end{aligned}$ | 0.4 |
| SII <br> (p value) |  |  | $\begin{aligned} & 7.5 \\ & (0.11) \\ & \hline \end{aligned}$ | $\begin{aligned} & 7.0 \\ & (0.32) \\ & \hline \end{aligned}$ | -0.1 |  |  | $\begin{aligned} & 6.1 \\ & (0.05) \\ & \hline \end{aligned}$ | $\begin{aligned} & 11.9 \\ & (0.01) \end{aligned}$ | 1.1 |

Educational qualification categories = level 3-degree level or above; level 2-intermediate; level 1-statutory school leaving; no qualifications.
$\dagger$ Total cholesterol serum concentration $\geq 5.0 \mathrm{mmol} / \mathrm{I}$. A different laboratory completed analyses in 1995 than in later surveys. Different analysers were used but methods and reference standards were the same. Health Surveys using a cholesterol oxidase method and analyses relating to these surveys are reproduced in supplementary table 1.

[^2]Table 7 Gender-specific, age-standardised prevalences for hypercholesterolaemia and low-serum HDL cholesterol concentration in five Scottish Health Surveys 1995, 1998, 2003 and 2008/2009 stratified by occupational social class

|  | Men |  |  |  |  | Women |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Proportion (\%) (95\% CI)* |  |  |  | Trend (p value) | Proportion (\%) (95\% CI)* |  |  |  | Trend (p value) |
|  | 1995 | 1998 | 2003 | 2008/2009 $\dagger$ |  | 1995 | 1998 | 2003 | 2008/2009 $\dagger$ |  |
| Hypercholesterolaemia $\ddagger$ |  |  |  |  |  |  |  |  |  |  |
| All | $\begin{aligned} & 79.4 \\ & (77.9 \text { to } 80.9) \end{aligned}$ | $\begin{aligned} & 69.5 \\ & (67.6 \text { to } 71.3) \end{aligned}$ | $\begin{aligned} & 71.8 \\ & \text { (69.4 to } 74.1 \text { ) } \end{aligned}$ | $\begin{aligned} & 64.6 \\ & (60.6 \text { to } 68.5) \end{aligned}$ | $-0.9(0.16)$ | $\begin{aligned} & 74.2 \\ & (72.6 \text { to } 75.8) \end{aligned}$ | $\begin{aligned} & 65.6 \\ & (63.8 \text { to } 67.5) \end{aligned}$ | $\begin{aligned} & 69.0 \\ & (66.7 \text { to } 71.3) \end{aligned}$ | $\begin{aligned} & 66.9 \\ & (63.0 \text { to } 70.7) \end{aligned}$ | $-0.4(0.45)$ |
| I, professional | $\begin{aligned} & 76.1 \\ & (70.3 \text { to } 82.0) \end{aligned}$ | $\begin{aligned} & 72.5 \\ & (66.1 \text { to } 78.8) \end{aligned}$ | $\begin{aligned} & 70.1 \\ & (62.3 \text { to } 77.8) \end{aligned}$ | $\begin{aligned} & 66.4 \\ & (53.1 \text { to } 79.7) \end{aligned}$ | $-0.7(0.01)$ | $\begin{aligned} & 81.6 \\ & (74.1 \text { to } 89.0) \end{aligned}$ | $\begin{aligned} & 70.0 \\ & (61.2 \text { to } 78.9) \end{aligned}$ | $\begin{aligned} & 70.6 \\ & (60.5 \text { to } 80.7) \end{aligned}$ | $\begin{aligned} & 65.9 \\ & (54.8 \text { to } 76.9) \end{aligned}$ | $-1.0(0.16)$ |
| II, managerial | $\begin{aligned} & 82.1 \\ & (79.3 \text { to } 84.9) \end{aligned}$ | $\begin{aligned} & 68.0 \\ & (64.3 \text { to } 71.7) \end{aligned}$ | $\begin{aligned} & 72.7 \\ & (68.5 \text { to } 76.9) \end{aligned}$ | $\begin{aligned} & 65.8 \\ & (58.7 \text { to } 73.0) \end{aligned}$ | -0.9 (0.27) | $\begin{aligned} & 70.5 \\ & (67.2 \text { to } 73.9) \end{aligned}$ | $\begin{aligned} & 65.5 \\ & (62.1 \text { to } 68.9) \end{aligned}$ | $\begin{aligned} & 64.0 \\ & (59.9 \text { to } 68.1) \end{aligned}$ | $\begin{aligned} & 68.3 \\ & (62.6 \text { to } 73.9) \end{aligned}$ | $-0.1(0.75)$ |
| III, skilled NM | 80.3 <br> (76.1 to 84.6) | $\begin{aligned} & 67.1 \\ & \text { (61.3 to } 72.8 \text { ) } \end{aligned}$ | $\begin{aligned} & 65.0 \\ & (56.9 \text { to } 73.1) \end{aligned}$ | 59.5 <br> (48.8 to 70.2) | $-1.3(0.10)$ | $\begin{aligned} & 75.7 \\ & \text { (73.2 to } 78.2 \text { ) } \end{aligned}$ | $\begin{aligned} & 62.9 \\ & (59.7 \text { to } 66.2) \end{aligned}$ | 68.4 <br> (64.3 to 72.5) | $\begin{aligned} & 66.0 \\ & (58.7 \text { to } 73.4) \end{aligned}$ | $-0.4(0.53)$ |
| III, skilled M | $\begin{aligned} & 77.9 \\ & (75.4 \text { to } 80.5) \end{aligned}$ | $\begin{aligned} & 70.3 \\ & (67.2 \text { to } 73.4) \end{aligned}$ | $\begin{aligned} & 74.6 \\ & (70.3 \text { to } 78.9) \end{aligned}$ | $\begin{aligned} & 69.9 \\ & (62.8 \text { to } 77.0) \end{aligned}$ | -0.4 (0.39) | 77.0 <br> (71.3 to 82.7) | $\begin{aligned} & 65.9 \\ & (59.6 \text { to } 72.2) \end{aligned}$ | $\begin{aligned} & 83.5 \\ & (75.8 \text { to } 91.2) \end{aligned}$ | $\begin{aligned} & 73.3 \\ & (63.5 \text { to } 83.1) \end{aligned}$ | $0.2(0.85)$ |
| IV, partly skilled | 79.8 <br> (75.7 to 84.0) | $\begin{aligned} & 72.2 \\ & (67.8 \text { to } 76.6) \end{aligned}$ | $\begin{aligned} & 68.6 \\ & (62.1 \text { to } 75.1) \end{aligned}$ | 54.4 <br> (45.3 to 63.6) | -1.8(0.02) | $\begin{aligned} & 75.6 \\ & (71.9 \text { to } 79.4) \end{aligned}$ | $\begin{aligned} & 69.0 \\ & (64.7 \text { to } 73.3) \end{aligned}$ | $\begin{aligned} & 75.1 \\ & \text { (69.9 to } 80.2 \text { ) } \end{aligned}$ | $\begin{aligned} & 66.7 \\ & (55.4 \text { to } 77.9) \end{aligned}$ | $-0.4(0.41)$ |
| V, unskilled | $\begin{aligned} & 80.4 \\ & (73.8 \text { to } 87.1) \end{aligned}$ | $\begin{aligned} & 68.9 \\ & (59.5 \text { to } 78.3) \end{aligned}$ | $\begin{aligned} & 69.8 \\ & (59.8 \text { to } 79.8) \end{aligned}$ | $\begin{aligned} & 56.6 \\ & (43.2 \text { to } 70.0) \end{aligned}$ | -1.5 (0.08) | $\begin{aligned} & 76.2 \\ & (70.6 \text { to } 81.9) \end{aligned}$ | $\begin{aligned} & 66.7 \\ & (59.2 \text { to } 74.1) \end{aligned}$ | 64.1 <br> (55.7 to 72.5) | $\begin{aligned} & 75.0 \\ & (60.9 \text { to } 89.2) \end{aligned}$ | 0.0 (0.98) |
| SII (p value) | -1.7 (0.63) | 3.1 (0.33) | -0.8 (0.89) | -8.0 (0.42) | -0.6 (0.27) | 5.6 (0.21) | 2.9 (0.48) | 11.1 (0.26) | 1.3 (0.77) | $-0.1(0.83)$ |
| Low HDL cholesterol (direct method)§ |  |  |  |  |  |  |  |  |  |  |
| All | - | - | $\begin{aligned} & 6.6 \\ & (5.2 \text { to } 8.0) \end{aligned}$ | $\begin{aligned} & 7.1 \\ & (4.8 \text { to } 9.3) \end{aligned}$ | 0.1 | - | - | 9.2 <br> (7.6 to 10.7) | $\begin{aligned} & 7.4 \\ & (5.2 \text { to } 9.7) \end{aligned}$ | $-0.3$ |
| I, professional | - | - | $\begin{aligned} & 7.3 \\ & (2.8 \text { to } 11.7) \end{aligned}$ | $\begin{aligned} & 8.8 \\ & (2.3 \text { to } 15.3) \end{aligned}$ | 0.3 | - | - | $\begin{aligned} & 3.6 \\ & (0 \text { to } 8.0) \end{aligned}$ | $\begin{aligned} & 5.5 \\ & (0 \text { to 11.5) } \end{aligned}$ | 0.3 |
| II, managerial | - | - | $\begin{aligned} & 3.1 \\ & (1.3 \text { to } 4.9) \end{aligned}$ | $\begin{aligned} & 5.7 \\ & (2.0 \text { to } 9.4) \end{aligned}$ | 0.5 | - | - | $\begin{aligned} & 6.9 \\ & (4.5 \text { to } 9.3) \end{aligned}$ | $\begin{aligned} & 4.3 \\ & (1.6 \text { to } 7.0) \end{aligned}$ | -0.5 |
| III, skilled NM | - | - | $\begin{aligned} & 4.4 \\ & (0.4 \text { to } 8.4) \end{aligned}$ | $\begin{aligned} & 3.1 \\ & (0 \text { to } 7.0) \end{aligned}$ | -0.2 | - | - | $\begin{aligned} & 11.2 \\ & (8.1 \text { to } 14.3) \end{aligned}$ | $\begin{aligned} & 9.8 \\ & (4.9 \text { to } 14.7) \end{aligned}$ | -0.3 |
| III, skilled M | - | - | $\begin{aligned} & 9.7 \\ & (6.6 \text { to } 12.7) \end{aligned}$ | $\begin{aligned} & 10.9 \\ & (5.6 \text { to } 16.3) \end{aligned}$ | 0.2 | - | - | $\begin{aligned} & 6.9 \\ & (2.0 \text { to } 11.9) \end{aligned}$ | $\begin{aligned} & 6.0 \\ & (0 \text { to 12.8) } \end{aligned}$ | -0.2 |
| IV, partly skilled | - | - | $\begin{aligned} & 8.5 \\ & (4.4 \text { to } 12.6) \end{aligned}$ | $\begin{aligned} & 3.9 \\ & (0.1 \text { to } 7.8) \end{aligned}$ | -0.8 | - | - | $\begin{aligned} & 11.2 \\ & (7.3 \text { to } 15.1) \end{aligned}$ | $\begin{aligned} & 11.3 \\ & (4.4 \text { to 18.3) } \end{aligned}$ | 0.0 |
| V, unskilled | - | - | $\begin{aligned} & 12.7 \\ & (4.5 \text { to 20.9) } \end{aligned}$ | $\begin{aligned} & 2.4 \\ & (0 \text { to } 7.3 \text { ) } \end{aligned}$ | -1.9 | - | - | $\begin{aligned} & 11.5 \\ & (4.7 \text { to } 18.3) \end{aligned}$ | $\begin{aligned} & 5.0 \\ & (0 \text { to 12.4) } \end{aligned}$ | -1.2 |
| SII (p value) |  |  | 8.0 (0.09) | -0.5 (0.93) | -1.6 |  |  | 6.7 (0.07) | 7.9 (0.09) | 0.2 |

 relating to these surveys are reproduced in supplementary table 2. *Negative lower limit has been set to zero.
 were the same.
HDL, high-density lipoprotein; M, manual; NM, non-manual; SII, slope index of inequality.

The threshold fibrinogen plasma concentration for the upper quintile increased from $3.1 \mathrm{~g} / 1$ in 1998 to $3.4 \mathrm{~g} / \mathrm{l}$ in $2008 / 2009$ for men and from 3.3 to $3.6 \mathrm{~g} / 1$ for women (tables 8 and 9). In 2008/2009, the threshold C reactive protein serum concentration for the highest quintile had increased to $3.71 \mathrm{mg} / 1$ for men and $4.80 \mathrm{mg} / \mathrm{l}$ for women (tables 8 and 9). For both inflammatory risk markers, socioeconomic inequalities existed that appeared greater in magnitude for men than for women.

## DISCUSSION

In Scotland, between 1995 and 2009, the changes in the prevalence of three major risk factors for CVD present a mixed story. Overall, prevalences have increased for obesity (including central obesity), stagnated for hypertension and decreased for hypercholesterolaemia. Many of the biomarkers considered in this study had socioeconomic gradients that persisted throughout the surveys. Considering this intransigence (with the exception of total cholesterol), it is perhaps not surprising that behavioural risk factors have also changed little over the same period in Scotland. ${ }^{13}$

Consistent with the rest of the developed world the Scottish population is becoming more obese. ${ }^{26}$ The larger increase in the prevalence of women with excessive WC and WHR could suggest that the rise in obesity among women has been particularly manifested as an increase in central adiposity. For all three measures of adiposity, absolute inequalities existed for both genders. However, there were some inconsistencies by social class, particularly for men. Greater inequalities for women have been observed elsewhere. ${ }^{27-30}$ The inequalities remained unchanged over time, even widening for women by WHR when stratified by education, similar to findings in other European countries. ${ }^{27}{ }^{28}$ It should be noted that for all the biomarkers considered in this study, precise comparison with other studies is difficult because of different definitions, sample demographics and study design.

An overall declining trend in mean systolic blood pressure in the developed world, and in particular Western Europe, has been identified. ${ }^{31}$ In contrast, although not directly comparable, our study identified that the prevalence of hypertension remained relatively static, with the hint of an increase, in Scotland. Levelling of declines, or even an increase in the case of England, have been identified in other countries over similar time periods. ${ }^{32-34}$ Inequalities have been reported for blood pressure in other countries ${ }^{32}{ }^{35}$ and in the past for Scotland among just women. ${ }^{36}$ The static trend in Scotland may be in part a consequence of improved hypertension management and medication, as in England, ${ }^{34}$ along with reduced behavioural risk factors such as increased physical activity and reduced discretional salt use ${ }^{13}$ being counteracted by increased obesity trends and consumption of high salt content processed foods. ${ }^{37}$

The good news of a modest decline in hypercholesterolaemia in this study concurs with findings for the developed world. ${ }^{38}$ This is tempered by the stark fact that in 2008/2009, the majority of respondents still had excessive serum concentrations of total cholesterol. Inequalities existed for women but these were less apparent by $2008 / 2009$, while for men, there were limited social gradients. A lack of socioeconomic inequalities for total cholesterol is a common finding. ${ }^{32}{ }^{33} \mathrm{~A}$ social class differential has been reported for women for hypercholesterolaemia in Scotland in the past ${ }^{36}$ and elsewhere in the UK. ${ }^{39}$ Whereas historically for men in Scotland, high cholesterol has been associated with nonmanual social class. ${ }^{36}$ The decline in hypercholesterolaemia prevalence is probably due to changes in health behaviours, for example, a decrease in saturated fats consumption, along with the increased use of lipidlowering drugs over the surveys, their use being more prevalent in more disadvantaged groups, particularly in the 2008/2009 surveys.

The proportion with low HDL cholesterol concentration changed little between 2003 and 2008/2009. Within each of the survey years, stronger social gradients existed for women than for men with the disadvantaged having a higher prevalence of low HDL cholesterol. Socioeconomic gradients in this direction have been identified for men and women, ${ }^{40}$ just women ${ }^{41} 42$ and appeared to be absent for both sexes. ${ }^{30}$ Elevated levels of C reactive protein and fibrinogen are associated with an increased risk of CHD but are considered to be markers as they are unlikely to be causal. ${ }^{4344}$ The absence of defined clinical thresholds indicating higher risk of CHD, plus skewed distributions, prompted the use of upper quintiles; some have considered this to represent 'high risk'. ${ }^{45}$ The threshold demarking the upper quintile was always higher for women, as seen in other studies, ${ }^{46}$ and the increase for both genders across the surveys would imply a shift to the right for at least some of the population. Much of the social gradient in these inflammatory markers, as seen in this study, can be explained by risk factors such as smoking, adiposity, cholesterol and physical activity. ${ }^{46-48}$ These risk factors are socially patterned in the SHeSs , although the latter two principally for women. ${ }^{13}$

One of this study's strengths is the use of data from nationally representative surveys. We also recognise a number of limitations. Only individuals living in private households were surveyed, and the declining response levels in these surveys are of concern, possibly introducing bias. This problem is common across surveys in general. The clustered design of the sampling in this study will lead to larger standard errors than those from a simple random sample of the same size because members of a cluster are more likely to resemble one another. Nevertheless, the effect of clustering was reduced as a high percentage of all possible postcode sectors were included in the SHeSs. A switch to sampling multiple adults within households from 2003 probably
Table 8 Gender-specific, age-standardised prevalences for elevated plasma fibrinogen and serum C reactive protein concentrations in five Scottish Health Surveys 1995 , 1998, 2003 and 2008/2009 stratified by highest educational qualification achieved

Educational qualification categories = level 3-degree level or above; level 2-intermediate; level 1-statutory school leaving; no qualifications.
$\dagger$ A different analyser was used for fibrinogen in 2008/2009.
Table 9 Gender-specific, age-standardised prevalences for elevated plasma fibrinogen and serum C reactive protein concentrations in five Scottish Health Surveys 1995, 1998, 2003 and 2008/2009 stratified by occupational social class
 *Negative lower limits have been set to zero.
$\ddagger$ A different analyser was used for fibrinogen in 2008/2009.
contributed to the decline as interviewers, after enrolling one individual, may have been disincentivised to pursue additional householders. Responders to health examination surveys tend to be older, women, of higher socioeconomic class and live healthier lifestyles. ${ }^{49}$ The 2003 SHeS response was modestly skewed towards those in less deprived areas. ${ }^{50}$ A differential response could lead to underestimates for most risk factors in the lower socioeconomic groups, which suggests that true inequalities are even greater than shown here, particularly so for the most recent surveys.

Measurement error will have contributed to bias in these analyses, as will the need to use predicted Omron readings in the first two surveys, the change in laboratories between 1995 and 1998 and the change in fibrinogen analyser between 2003 and 2008/2009. However, the laboratories did use the same methods and reference standards ${ }^{16}$ and a high correlation has been demonstrated between the different analysers. ${ }^{51}$ These limitations mean caution should be exercised when interpreting prevalence trends but should not greatly distort comparison of socioeconomic gradients. The cross-sectional nature of these surveys does not capture the lifelong burden of these risk markers within individuals, something that might have a considerable effect on subsequent CVD. The increase over time in the proportion of individuals with intermediate educational qualification may in part be a consequence of the subtle changes in how educational achievement was determined across the surveys. These changes would have had no impact on those with no qualifications or those with the highest qualifications. A substantial component of this increase is likely to be due to the rise in the school staying on rates beyond compulsory education seen since the 1970s in Scotland, where intermediate qualifications would be achieved. ${ }^{52}$

Inconsistency between the socioeconomic measures highlights the benefits of using both education and social class and why they should not be used interchangeably. ${ }^{53} 54$ There were sometimes apparent differences in inequalities depending on the measure used. This may reflect the imperfection of these proxies as measures of deprivation or it may demonstrate the underlying heterogeneity of inequalities. Further research would be valuable. Education reflects the longterm influence of early life circumstance and not only strongly determines an individual's occupation, implying some interdependency with social class, but also provides the knowledge and life skills required to adopt healthier lifestyles and maximise access to health resources. ${ }^{55}$ Alternative indices such as area of deprivation, income and NS-SEC were not consistently available across these surveys. The use of NS-SEC-derived social class in the last three surveys may have led to the misclassification of individuals. This, combined with the shift over time in the composition of social classes and education, may have led to some of the observed alterations in prevalence patterning. However, the SII accounts for the size
of the total population and changes in the distribution of groupings making comparison over time possible. One disadvantage of the SII is that it assumes a linear relationship across the socioeconomic groups, something that may not necessarily be true. The low numbers in the highest and lowest categories may have led to potentially volatile individual estimates, especially in the last two surveys. This was particularly a problem in the 2008/2009 surveys among men with no qualifications in the younger age groups and this may have led to imprecise age-standardised estimates for the lowest educational category and care should be taken in their interpretation.

Small changes in risk factor prevalence at the population level could elicit substantial reductions in CHD mortality. ${ }^{56} 57$ Interventions to achieve such reductions can centre on targeting high-risk individuals, ${ }^{21}$ such as the Keep Well programme ${ }^{58}$ that is aimed at increasing the rate of health improvement among high-risk individuals in the most deprived communities in Scotland. It is too early to assess the impact of this programme on biomarkers for CVD risk but its proposed expansion may increase its effectiveness. Altering the health behaviours of the whole population may have greater impact. ${ }^{59}{ }^{60}$ In Scotland, there are a number of population-based initiatives aimed at improving health behaviours and consequently the biomarkers for CVD. These include a tobacco control action plan, ${ }^{61}$ a national physical strategy, ${ }^{62}$ healthy eating advice ${ }^{63}$ and an action plan relating to obesity. ${ }^{64}$ If best practice interventions to reduce classic risk factors were successful, most of the absolute socioeconomic differences in CHD mortality could be eliminated. ${ }^{12}$ Elimination of relative inequalities may require interventions to be targeted at the socioeconomically disadvantaged (eg, the Keep Well programme) or possibly by improving the circumstances that create social inequality. ${ }^{6566}$

The increase in obesity prevalence is probably the driver behind the rise in prevalence of diabetes reported in Scotland over the same period. ${ }^{13}$ In contrast, there has not been a comparable increase in hypertension and cholesterol prevalence, as might be expected. This disassociation between obesity and other risk factors could be due to interventions, such as medication, ameliorating the adverse effects of obesity. It has been shown that obese people in the current century, although having higher risk factor levels than the nonobese, have a lower prevalence of high cholesterol, high blood pressure and smoking than obese individuals 40 years ago. ${ }^{67} 68$ All the above could contribute to explanations as to why overall CHD death rates have continued to fall, despite increasing rates of obesity. If obesity had not increased, there might have been a decline in hypertension prevalence and a greater reduction in hypercholesterolaemia. It is sobering to consider that any gains in CHD mortality by reduction in some risk factors may be offset by additional deaths from obesity and diabetes. These trends could certainly
contribute to the observed levelling or slowing of the decline among younger groups. The lack of progress in reducing risk factor socioeconomic inequalities can only ensure such differentials in CVD mortality will remain in the Scottish population. More effective population-wide policies are now urgently required.

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## Competing interests None.

Ethical approval No ethical approval was required for this study, which was based on publicly available, anonymised Scottish Health Survey data sets.

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[^0]:    ${ }^{1}$ Measuring Health, MRC/CSO Social and Public Health Sciences Unit, Glasgow, UK
    ${ }^{2}$ Scottish Centre for Social Research, Edinburgh, UK
    ${ }^{3}$ Division of Public Health, University of Liverpool, Liverpool, UK

    ## Correspondence to

    Dr Joel William Hotchkiss; joel.hotchkiss@sphsu.mrc. ac.uk

[^1]:    Hypertension (uncontrolled) defined as systolic blood pressure $\geq 140 \mathrm{~mm} \mathrm{Hg}$ or diastolic blood pressure $\geq 90 \mathrm{~mm} \mathrm{Hg}$, irrespective of treatment.
    tIn 1995 and 1998, blood pressure readings were taken using Dinamap monitors and then converted to Omron monitor readings.
    §Educational qualification categories = level 3-degree level or above; level 2-intermediate; level 1-statutory school leaving; no qualifications.
    NM, non-manual; M, manual; SII, slope index of inequality.

[^2]:    HDL, high-density lipoprotein; SII, slope index of inequality.

