Social class differences in coronary heart disease in middle-aged British men: implications for prevention

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Background

Though social class differences in coronary heart disease (CHD) are well recognized, few studies have assessed the effect of imprecision in social class assessment on the relationship or the overall contribution of social class to attributable CHD risk.

Methods

Prospective observational study of the relationship between occupational social class (assessed at baseline and after 20 years), major CHD (coronary death and non-fatal myocardial infarction) and all-cause mortality rates over 20 years among 5628 middle-aged British men with no previous evidence of CHD.

Results

The age-adjusted hazard of major CHD for manual men relative to non-manual men was 1.41 (95% CI: 1.21, 1.64) before correction and 1.50 (95% CI: 1.25, 1.79) after correction for imprecision of social class measurement. The imprecision-corrected estimate was attenuated to 1.28 (95% CI: 1.06, 1.54) after adjustment for the adult coronary risk factors (blood cholesterol, blood pressure, body mass index, cigarette smoking, alcohol, physical activity, and lung function) and to 1.20 (95% CI: 0.99, 1.45) following further adjustment for height. The population attributable risk fraction of major CHD for social class (manual versus non-manual) was 22% after correction for imprecision in social class, which was reduced to 14% after adjustment for the adult coronary risk factors, and 10% after further adjustment for height. Similar results were obtained for all-cause mortality.

Conclusions

Even taking account of measurement imprecision, the contribution of social class to overall CHD risk is modest. Population-wide strategies to reduce major CHD risk factors are likely to have greater potential benefits for CHD prevention than strategies designed specifically to reduce social inequalities in CHD.

Keywords Coronary heart disease, social class, risk factor assessment, attributable risk

Social inequalities in the incidence of coronary heart disease (CHD) in the UK are well documented, but while absolute CHD rates have fallen during the last 20 years, the fall has been concentrated among the highest social class groups so that the relative differences between those at the top and those at the bottom of the social scale have widened. In epidemiological studies that relate social conditions to subsequent disease risk, social class (as determined by job occupation) is often used as a convenient and available indicator of the underlying socioeconomic factors. However, despite considerable emphasis being placed in recent public health policies on reducing social class inequalities in CHD, several important aspects of social class differences remain unresolved.

First, even as an approximate index of socioeconomic status, adult social class is not always precisely estimated and may change over time. Though these factors will lead to underestimation of the true extent of social class differences in CHD, the extent of such underestimation has not been established. Furthermore, the relative contribution of adult and early life factors to social inequalities in CHD, as well as the likely value of identifying further factors influencing social inequalities, remains uncertain.

In this study, using a cohort of 5628 middle-aged British men with no previous evidence of CHD followed for 20 years between

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1978 and 2000, we examine how wide social class differences in CHD are, before and after correction for imprecision in the measurement of social class. We estimate the amount that can be explained by the established coronary risk factors, and present estimates of population attributable risk for social class before and after adjustment for the established coronary risk factors. Parallel analyses for all-cause mortality are shown.

Methods

Subjects

The British Regional Heart Study (BRHS) is a prospective study of cardiovascular disease in men in one General Practice in each of 24 British towns, representing all major British regions. Participants were enrolled in 1978–1980 aged 40–59 years and have been followed up for all-cause mortality using the National Health Service (NHS) Central Registers and for cardiovascular morbidity through regular biennial reviews of General Practice records. Ethical approval was obtained from all relevant local Research Ethics Committees.

Baseline risk factors

Height without shoes and weight in trousers and socks were measured, to the nearest millimetre and 0.1 kg respectively. Blood pressure was measured twice in succession in the right arm using the London School of Hygiene and Tropical Medicine sphygmomanometer, with the subject seated and the arm supported. Adjustment for observer variation within each town was performed and the mean of the two blood pressure measurements was used in analyses. Serum total cholesterol was measured twice in succession in the right arm using a Vitalograph spirometer. Values were height standardized to 1.73 m, the average height of the men in the study.

Social class

At baseline, each man was asked about his longest held occupation in terms of type, designation, and status. The social class distribution observed in the study participants was almost exactly the same as that obtained from national census data recorded around the same time. After 20 years, all surviving men were invited for a re-screening where they were asked to record their current or most recent occupation and the duration of that occupation. Social class was determined using the Registrar General’s six-category classification on both occasions and categorized as non-manual (I, II and IIIIM) or manual (IIIM, IV and V). Men in the armed forces at baseline were excluded from this analysis.

Pre-existing coronary disease

At baseline, men were identified as having a possible history of CHD if: (1) they had ever been told by a doctor that they had angina or a heart attack; (2) their answers to the Rose angina questionnaire indicated that they had had definite or possible angina; (3) they had electrocardiographic evidence of definite or possible myocardial infarction or ischaemia; or (4) they had ever had a history of severe chest pain lasting half an hour or more that caused them to consult a doctor.

Major CHD events during follow-up

Subjects were followed up for mortality and cardiovascular morbidity, with 99.5% of subjects successfully traced over a 20-year follow-up period. Information on all deaths was collected through the established ‘flagging’ procedures provided by the National Health Service registers in Southport (England and Wales) and Edinburgh (Scotland). Fatal CHD events were defined as deaths with ischaemic heart disease (International Classification of Diseases, Ninth Revision [ICD-9] 410–414) as the underlying cause including sudden death of presumed cardiac origin. Evidence regarding non-fatal heart attacks was obtained by reports from general practitioners and by biennial reviews of the patients’ notes, through to the end of the study period. Diagnosis was based on WHO criteria, (any report of myocardial infarction accompanied by at least two of: history of severe chest pain, electrocardiographic evidence of myocardial infarction, and cardiac enzyme changes associated with myocardial infarction). Major CHD was defined as non-fatal myocardial infarction or death from CHD.

Statistical methods

Time to major CHD events by social class status

For men with no baseline evidence of CHD, Kaplan Meier curves stratified by social class (manual versus non-manual) were used to display the differences in major CHD and all-cause mortality rates by social class over 20 years (Figure 1). The 20-year event rates were calculated per 1000 person years of exposure and directly standardized to the age distribution of the entire cohort. Relative hazards were estimated using Cox proportional hazards regression. The relative difference between social classes explained by the coronary risk factors was estimated by the equation \( \hat{\beta}_0 \) the age-adjusted log hazard ratio for social class, and \( \beta_1 \) the fully adjusted coefficient. Approximate 95% CI were calculated using bias-corrected bootstrap re-sampling of size 1000 to estimate the upper and lower limits.

Adjustment for imprecision in social class status

We considered two distinct sources of social class imprecision: (1) misclassification of social class at baseline; and (2) true changes in social class within the first 10 years of follow-up. For the remainder of this paper we refer to these two influences as ‘social class imprecision’. The magnitude of social class imprecision was assessed by comparing the social class measurements taken at baseline with those recorded at the 20-year follow-up (survivors only); the analysis was restricted to subjects who reported at follow-up that they had been in the same occupation for at least 10 years, thus capturing the second component of imprecision mentioned above. The proportion of individuals measured imprecisely \( \hat{\tau} \) was then estimated through the equation \( \hat{\tau} = 0.5 - 0.5 \sqrt{(N-2\hat{n})/N} \), where \( N \) individuals are measured twice and there are \( n \) disagreements between the baseline and follow-up measurements. The
adjusted log hazard ratio $\beta^*$ can then be obtained from the observed estimate $\hat{\beta}$ through the equation $\beta^* = \hat{\beta} (1 - 2\hat{\tau})$, with approximate CI calculated from formulae that take into account the variability in both $\beta$ and $\hat{\tau}$. The effect of imprecision in other CHD risk factors (particularly total cholesterol and blood pressure) on the estimated hazard for social class was assessed using the methods of Rosner.19

**Population attributable risk fraction (AR)**

The population attributable risk fraction (AR) for manual social class is the proportion of all disease events in the population that can be attributed to the excess risks experienced by manual men over those of non-manual men, and is calculated through the equation $p(RR - 1)/(1 + p(RR - 1))$, where $p$ is the proportion of manual men in the population and RR is the relative risk of major CHD for manual men relative to non-manual men (approximated in this paper by the relative hazard from the Cox regression20). The 95% CI for the AR were calculated by assuming that $p$ was fixed and calculating the AR values corresponding to the lower and upper confidence limits of the RR. This method of estimation yielded virtually exactly the same estimates and CI as methods based on logistic regression prediction.

**Results**

**Subjects and baseline risk factors**

In all, 7735 men attended for the baseline examination between 1978 and 1980, of whom 5791 (74.9%) had no evidence of CHD. Social class was recorded for 5779 of these men (99.8%), 5628 of whom were not in the army. Of these men, 2397 (42.6%) were classed as non-manual and 3231 (57.4%) as manual. The baseline characteristics of these two groups are displayed in Table 1. Manual men were, on average, slightly older than non-manual men, had higher BMI and blood pressure, were considerably more likely to be current cigarette smokers and heavy drinkers and less likely to be physically

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*Figure 1* (a) 20-year major coronary heart disease (CHD) event and (b) all-cause death rates (with 95% CI) by social class (above) and respective Kaplan Meier survival curves (manual versus non-manual; below) among 5628 men with no baseline evidence of CHD. Figures are not adjusted for imprecision in social class.
Table 1 Baseline characteristics of the 5628 men with no evidence of coronary heart disease by social class

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>I (n = 468)</th>
<th>II (n = 1364)</th>
<th>III NM (n = 565)</th>
<th>III M (n = 2430)</th>
<th>IV (n = 586)</th>
<th>V (n = 215)</th>
<th>P Non-manual</th>
<th>Manual</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (years)</td>
<td>48.9</td>
<td>49.6</td>
<td>49.9</td>
<td>50.0</td>
<td>50.0</td>
<td>49.8</td>
<td>0.001</td>
<td>49.5</td>
<td>50.0</td>
</tr>
<tr>
<td>Mean total cholesterol (mmol/l)</td>
<td>6.35</td>
<td>6.36</td>
<td>6.35</td>
<td>6.20</td>
<td>6.18</td>
<td>6.15</td>
<td>&lt;0.001</td>
<td>6.36</td>
<td>6.20</td>
</tr>
<tr>
<td>Mean body mass index (kg/m²)</td>
<td>24.8</td>
<td>25.2</td>
<td>25.3</td>
<td>25.6</td>
<td>25.2</td>
<td>25.2</td>
<td>&lt;0.001</td>
<td>25.1</td>
<td>25.5</td>
</tr>
<tr>
<td>Mean systolic blood pressure (mmHg)</td>
<td>140.0</td>
<td>141.5</td>
<td>144.6</td>
<td>146.0</td>
<td>144.5</td>
<td>146.8</td>
<td>&lt;0.001</td>
<td>141.9</td>
<td>145.8</td>
</tr>
<tr>
<td>Mean height (cm)</td>
<td>176.4</td>
<td>175.2</td>
<td>174.3</td>
<td>172.6</td>
<td>171.2</td>
<td>171.0</td>
<td>&lt;0.001</td>
<td>175.2</td>
<td>172.2</td>
</tr>
<tr>
<td>Mean FEV₁³ (ml/sec)</td>
<td>360</td>
<td>348</td>
<td>339</td>
<td>332</td>
<td>319</td>
<td>324</td>
<td>&lt;0.001</td>
<td>348</td>
<td>329</td>
</tr>
<tr>
<td>Proportion moderately active (%)</td>
<td>55.7</td>
<td>47.7</td>
<td>41.1</td>
<td>35.4</td>
<td>29.1</td>
<td>25.5</td>
<td>&lt;0.001</td>
<td>47.7</td>
<td>33.6</td>
</tr>
<tr>
<td>Proportion heavy drinkers (%)</td>
<td>4.7</td>
<td>7.3</td>
<td>7.1</td>
<td>12.8</td>
<td>14.2</td>
<td>15.8</td>
<td>&lt;0.001</td>
<td>6.8</td>
<td>13.2</td>
</tr>
</tbody>
</table>

³ Forced expiratory volume in one second, height standardized.

Table 2 Repeat determination of social class status over a 20-year period for men without evidence of coronary heart disease at baseline who, at the 20-year screening, had worked in the same job for at least 10 years (N = 2810)

<table>
<thead>
<tr>
<th>20-year follow-up</th>
<th>Baseline social class</th>
<th>Manual</th>
<th>Non-manual</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manual</td>
<td>1181</td>
<td>222</td>
<td>1403</td>
<td></td>
</tr>
<tr>
<td>Non-manual</td>
<td>171</td>
<td>1236</td>
<td>1407</td>
<td></td>
</tr>
</tbody>
</table>

Imprecision in social class status

Table 2 shows how the social class status obtained at baseline compares with that ascertained at 20 years for men without CHD at baseline who, at the 20-year screening, claimed to have worked in the same job for at least 10 years (N = 2810). From these data, the estimated probability of being measured imprecisely was 0.076 (Methods). There were no differences in age, blood lipids, blood pressure, cigarette smoking, physical activity, or geographical region between those men that were classified the same at baseline and follow-up and those men that were classified differently (data not shown).

Social class variation in major CHD and total mortality rates over 20 years

After 20 years of follow-up, 479 manual men (14.8%) and 261 non-manual men (10.9%) had had a major CHD event, and 847 manual men (26.2%) and 403 non-manual men (16.8%) had died. Figure 1 displays event rates by baseline social class (six levels) and Kaplan Meier curves for manual men compared with non-manual men. Hazard ratio estimates for manual men relative to non-manual men before and after adjusting for the established coronary risk factors and before and after correction for imprecision of social class are shown in Table 3.

For major CHD events, the observed age-adjusted hazard ratio for manual men relative to non-manual men before correction for social class imprecision was 1.41, which attenuated to 1.23 after adjustment for the adult coronary risk factors (total cholesterol, systolic blood pressure, BMI, cigarette smoking, alcohol intake, physical activity, and FEV₁). Correcting for imprecision of social class increased these estimates to 1.50 (95% CI: 1.25, 1.79) and 1.28 (95% CI: 1.06, 1.54) respectively; indicating a 39% (95% CI: 18%, 79%) reduction in magnitude (on the log scale). Once height was also taken into account, the observed differences between manual and non-manual social classes became non-significant, and 56% (95% CI: 30%, 107%) of the original difference between social classes was accounted for. Of the individual risk factors, cigarette smoking accounted for the greatest proportion of the CHD differences between social classes, explaining 28% of the social class divide. Systolic blood pressure explained 15% and physical activity 17%, whilst alcohol intake, FEV₁, and BMI each explained less than 10% of the observed difference. Adjustment for total cholesterol increased the corrected hazard ratio for social class to 1.61. This is because total cholesterol levels were on average lower amongst the manual men (Table 1). Thus the proportion of the social class divide explained by total cholesterol was ~18% (indicating that the age-adjusted log hazard ratio would have been 18% greater had total cholesterol levels been the same in the two groups). Height made a notable additional contribution to the social class gradient, explaining 21% of the difference. The inclusion of high density lipoprotein cholesterol as one of the coronary risk factors had no additional effect on the size of the adjusted social class differences observed.

For total mortality, the age-adjusted hazard ratio for social class was 1.75 (95% CI: 1.52, 2.01) after correction for imprecision in social class. Taking into account the adult coronary risk factors (as before), this attenuated to 1.34 (95% CI: 1.16, 1.55), a 48% reduction in magnitude. Cigarette smoking explained the greatest amount of the social class divide (24%), whilst FEV₁ explained 16%. The remaining coronary risk factors explained little or none of the difference in mortality rates between manual and non-manual men. Height made no additional contribution to explaining the differences in all-cause mortality.
The multivariate effects that regression dilution of total cholesterol, systolic blood pressure, and BMI over 20 years may have on the estimated relative hazard for social class were assessed. The simultaneous influence on the hazard ratio was found to be negligible, due mainly to the fact that BMI was fairly insensitive to the effects of regression dilution and the effects for blood pressure and blood cholesterol were acting in opposite directions (since manual men had higher blood pressure but lower total cholesterol levels than non-manual men). Furthermore, taking changes in cigarette smoking, physical activity, and alcohol intake over the study period into account (by fitting time updated covariates in the Cox model) had virtually no effect on the adjusted social class effect.

Population attributable risk fraction (AR) estimates
For major CHD and total mortality, Table 4 shows the AR for social class (manual versus non-manual) before and after correction for imprecision in social class status and before and after adjustment for the adult coronary risk factors. For major CHD events, the age-adjusted AR for social class was 22% (95% CI: 13%, 31%) after correction for social class imprecision. Assuming manual men had the same average levels of total cholesterol, systolic blood pressure, BMI, physical activity, FEV₁, alcohol intake, and smoking rates as non-manual men, the age- and imprecision-adjusted AR for social class would have been 14% (95% CI: 3%, 24%). Further adjustment for height at baseline reduced this estimate to 10% (95% CI: 2%, 21%). For all-cause mortality, the age-adjusted AR for social class was 30% (95% CI: 23%, 37%) after correction for social class imprecision, which decreased to 16% (95% CI: 8%, 23%) after adjustment for the adult coronary risk factors. Further adjustment for height had no additional effect. Including men with baseline evidence of CHD in analyses had little effect on the estimated AR for all-cause mortality.

Further analyses: effect of redefining the low-risk group
When the 'low-risk' group was re-defined to include only social class I (rather than all non-manual social classes), the hazard ratios for the high-risk group (social classes II–V) relative to the low-risk group (social class I) and the corresponding AR estimates increased. The uncorrected age-adjusted hazard ratio was 1.62 (95% CI: 1.17, 2.24), which attenuated to 1.31 (95% CI: 0.94, 1.82) after adjustment for the adult risk factors and 1.23 (95% CI: 1.26, 1.30) after further adjustment for height.

Table 3 Relative hazard of major coronary heart disease (CHD) and all-cause mortality (manual versus non-manual) before and after correction for imprecision of social class. All models are adjusted for age (N = 5628)

<table>
<thead>
<tr>
<th>Variables adjusted for</th>
<th>Major CHD</th>
<th>All-cause mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before correction</td>
<td>After correction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>95% CI</td>
</tr>
<tr>
<td>None</td>
<td>—</td>
<td>1.41 (1.21, 1.64)</td>
</tr>
<tr>
<td>Smoking</td>
<td>28%</td>
<td>1.28 (1.10, 1.49)</td>
</tr>
<tr>
<td>Systolic blood pressure (SBP)</td>
<td>15%</td>
<td>1.34 (1.15, 1.56)</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>−18%</td>
<td>1.50 (1.29, 1.74)</td>
</tr>
<tr>
<td>Body mass index (BMI)</td>
<td>5%</td>
<td>1.38 (1.19, 1.61)</td>
</tr>
<tr>
<td>Physical activity</td>
<td>17%</td>
<td>1.33 (1.14, 1.55)</td>
</tr>
<tr>
<td>Alcohol</td>
<td>8%</td>
<td>1.37 (1.18, 1.60)</td>
</tr>
<tr>
<td>FEV₁ (height standardized)</td>
<td>7%</td>
<td>1.38 (1.18, 1.60)</td>
</tr>
<tr>
<td>Height</td>
<td>21%</td>
<td>1.31 (1.12, 1.53)</td>
</tr>
<tr>
<td>Smoking, SBP, and total cholesterol</td>
<td>25%</td>
<td>1.29 (1.11, 1.51)</td>
</tr>
<tr>
<td>Adult coronary risk factors</td>
<td>39%</td>
<td>1.23 (1.05, 1.44)</td>
</tr>
<tr>
<td>Adult coronary risk factors plus height</td>
<td>56%</td>
<td>1.16 (0.99, 1.37)</td>
</tr>
</tbody>
</table>

a Calculated on the log scale (Methods). The combined contribution of multiple risk factors is less than the sum of the individual contributions because of the multifactorial nature of CHD.

b Significant predictor (P < 0.01) of both CHD and total mortality.
c Significant predictor (P < 0.01) of CHD only.
d U-shaped relationship with CHD and total mortality.
e Forced expiratory volume in one second.

Effects of imprecision and changes in established coronary risk factors
The multivariate effects that regression dilution of total cholesterol, systolic blood pressure, and BMI over 20 years may have on the estimated relative hazard for social class were assessed. The simultaneous influence on the hazard ratio was found to be negligible, due mainly to the fact that BMI was fairly insensitive to the effects of regression dilution and the effects for blood pressure and blood cholesterol were acting in opposite directions (since manual men had higher blood pressure but lower total cholesterol levels than non-manual men). Furthermore, taking changes in cigarette smoking, physical activity, and alcohol intake over the study period into account (by fitting time updated covariates in the Cox model) had virtually no effect on the adjusted social class effect.

Population attributable risk fraction (AR) estimates
For major CHD and total mortality, Table 4 shows the AR for social class (manual versus non-manual) before and after correction for imprecision in social class status and before and after adjustment for the adult coronary risk factors. For major CHD events, the age-adjusted AR for social class was 22% (95% CI: 13%, 31%) after correction for social class imprecision. Assuming manual men had the same average levels of total cholesterol, systolic blood pressure, BMI, physical activity, FEV₁, alcohol intake, and smoking rates as non-manual men, the age- and imprecision-adjusted AR for social class would have been 14% (95% CI: 3%, 24%). Further adjustment for height at baseline reduced this estimate to 10% (95% CI: −2%, 21%). For all-cause mortality, the age-adjusted AR for social class was 30% (95% CI: 23%, 37%) after correction for social class imprecision, which decreased to 16% (95% CI: 8%, 23%) after adjustment for the adult coronary risk factors. Further adjustment for height had no additional effect. Including men with baseline evidence of CHD in analyses had little effect on the estimated AR for all-cause mortality.

Further analyses: effect of redefining the low-risk group
When the ‘low-risk’ group was re-defined to include only social class I (rather than all non-manual social classes), the hazard ratios for the high-risk group (social classes II–V) relative to the low-risk group (social class I) and the corresponding AR estimates increased. The uncorrected age-adjusted hazard ratio was 1.62 (95% CI: 1.17, 2.24), which attenuated to 1.31 (95% CI: 0.94, 1.82) after adjustment for the adult risk factors and 1.23 (95% CI: 1.26, 1.30) after further adjustment for height.

Table 3 Relative hazard of major coronary heart disease (CHD) and all-cause mortality (manual versus non-manual) before and after correction for imprecision of social class. All models are adjusted for age (N = 5628)
Table 4 Population attributable risk fraction (AR) for manual social class for men with no baseline evidence of CHD (N = 5628). Estimates are presented before and after correction for measurement imprecision of social class status. All estimates are adjusted for age and risk factors.

<table>
<thead>
<tr>
<th>Risk factors adjusted for</th>
<th>Major CHD Before correction for social class imprecision</th>
<th>Major CHD After correction for social class imprecision</th>
<th>All-cause mortality Before correction for social class imprecision</th>
<th>All-cause mortality After correction for social class imprecision</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>AR 19% (95% CI 11%, 27%)</td>
<td>AR 22% (13%, 31%)</td>
<td>AR 26% (20%, 32%)</td>
<td>AR 30% (23%, 37%)</td>
</tr>
<tr>
<td>Adult coronary risk factors</td>
<td>AR 12% (3%, 20%)</td>
<td>AR 14% (3%, 24%)</td>
<td>AR 14% (7%, 21%)</td>
<td>AR 16% (8%, 23%)</td>
</tr>
<tr>
<td>Adult coronary risk factors plus height</td>
<td>AR 8% (~1%, 18%)</td>
<td>AR 10% (~2%, 21%)</td>
<td>AR 14% (7%, 21%)</td>
<td>AR 16% (8%, 24%)</td>
</tr>
</tbody>
</table>

4 Cigarette smoking (current, ex, never), systolic blood pressure (SBP), total cholesterol, body mass index (BMI), physical activity (none, occasional, light, moderate or more), alcohol intake (none, occasional, light, moderate, heavy), and forced expiratory volume in one second (FEV 1) (height standardized).

Discussion

In a population of middle-aged British men with no evidence of CHD at entry followed up over 20 years, the age-adjusted relative hazard of major CHD for manual men relative to non-manual men was 1.50 (95% CI: 1.25, 1.79), after correction for social class imprecision. This was reduced to 1.28 (95% CI: 1.06, 1.54) once differences in the adult coronary risk factors were taken into account. For all-cause mortality these figures were 1.75 (95% CI: 1.52, 2.01) and 1.34 (95% CI: 1.16, 1.55) respectively. In our cohort, if manual men had experienced the same baseline levels of risk as non-manual men, then 22% of all first major CHD events and 30% of all deaths over the follow-up period would have been prevented. After taking account of adult coronary risk factors, the AR for manual social class was 14% for major CHD cases and 16% for all-cause mortality, indicating that if social inequalities in adult coronary risk factors could be eliminated, the remaining risk differences between manual and non-manual men would account for 14% of major CHD cases during middle age and 16% of all deaths. The figure for CHD was reduced to 10% after further adjustment for height. When risk estimates were recalculated using only social class I as the reference group, estimates of relative and attributable risk increased, demonstrating that reducing the risks to those of social class I would have substantial further benefits.

Strengths and weaknesses of analyses

Using individuals who, at the 20-year screening, claimed to have been working in the same job for at least 10 years, we were able to correct for both random misclassification of social class status at baseline and true changes in social class during the first 10 years, thus enabling estimation of associations between ‘usual’ social class held throughout the study period and disease risk over the study period. Although it is possible that the onset of CHD during the study may have led to a change in social class for some individuals, separate examination of social class changes in subjects developing CHD during the follow-up period suggested that any such effect would be small. Our results are particularly relevant since the social class distribution of our men at study entry was close to that of all British men aged 35–64 at that time,14 and our estimates of social class differences were very similar to those observed in national CHD mortality statistics over a similar period (1981–1992).21 In contrast, the Whitehall study of British civil servants working in London22 contained a more polarized social class distribution, which may have contributed to the greater CHD gradients observed in this study.23 However, this may also have been due to the different measures of social deprivation used in the two studies. We compared the strength of association between social class and CHD risk with those of two other potential measures (car ownership and housing tenure; available from a 5-year follow-up questionnaire). Though owning a car and being an owner-occupier were related to lower subsequent CHD risk (between 5 and 20 years), neither was more strongly related than adult social class (over the same period). However, it is recognized that a combined baseline measure of the three socioeconomic factors may have been able to predict CHD outcome better than any single measure in isolation. Social class, though the most widely used, is only one potential measure of socioeconomic status and a combination of different socioeconomic measures may encapsulate different ‘social class dimensions’ better than any single measure in isolation.24,25 Furthermore, area-based measures of social deprivation may contribute additional socioeconomic information over and above that obtained from ‘individual-level’ factors.

Estimating the social inequalities remaining after adjustment for baseline risk factors depends on the assumption that imprecision of assessment and changes in risk factors over time does not differ by social class. This would not be true for cigarette smoking, where differential rates of quitting exist.26 In our study 33% of non-manual smokers had given up by 5 years compared with 24% of manual smokers. The effect of this on the corrected relative hazard for social class after adjustment for the adult risk factors was small (decreasing it marginally). Similarly, other differential changes in risk factors would tend to lead to overestimation, rather than underestimation, of the real social class differences in CHD, as they are more likely to result in improvements in the risk profile of non-manual men over manual men.
Explanations for the social gradient in CHD risk

The extent to which social class differences can be explained by established coronary risk factors has been assessed in a number of studies. In the current analysis we found that cigarette smoking was the most important single risk factor accounting for 28% of the CHD gradient. Height explained 21% of the variation, whilst the remaining risk factors each explained 15% or less. As was found in the Scottish Heart Health Study, average total cholesterol levels for non-manual men were greater than for manual men. The observed relative hazard for social class was therefore smaller than it would have been had total cholesterol levels been the same for all men. Therefore, had analyses been performed excluding total cholesterol as a risk factor, the remaining adult risk factors would have explained more of the social class divide than was estimated in this paper. When considered together, the adult coronary risk factors explained 39% of the CHD difference between social classes, less than the sum of the individual contributions due to the multifactorial nature of CHD. Our estimate is the same as was found in the Scottish Heart Health Study, using a wider range of risk factors (although the significant inverse relation with total cholesterol was not accounted for in their analysis). A recent 25-year follow-up of men in the Whitehall study found that cigarette smoking, blood pressure, total cholesterol, and glucose together accounted for 56% of the CHD risk difference between low-risk men in the lowest and highest grades of employment. Our observation that height is the second most important determinant of the social class gradient in CHD suggests that early life factors may be important in the development of social differences in CHD—an observation consistent both with the relationship between height and CHD in individuals and with recent evidence that childhood socioeconomic environment may be directly related to the risk of CHD. The addition of height to the adult CHD risk factors resulted in 56% of the CHD difference being explained. Some of the remaining association may result from imprecision in risk factor assessment, or additional contributions made by homocysteine level, by job stress, or by other unmeasured early life factors.

Implications for CHD prevention

Though reducing the CHD risk of all subjects in manual occupations to the level of non-manual subjects would have an appreciable effect (approximately one-fifth of CHD events during middle-age would be prevented), it would be modest compared with the expected reductions following population-wide reductions in mean total cholesterol and blood pressure, where it is estimated that 57% of major CHD cases during middle age would be prevented if long term mean population levels of total cholesterol and blood pressure could be reduced by 15% (JR Emberson—unpublished data). Indeed, it has been suggested that the CHD epidemic could effectively be ended if the proportion of the population at lifetime low exposure levels of total cholesterol, blood pressure, and cigarette smoking could be substantially increased. In our analysis, the AR for manual social class after adjustment for the adult risk factors and correction for social class imprecision was 14%, but increased to over 22% (corrected estimate not available) when social class I was used as the sole low risk group (rather than all non manual occupations). These results suggest that the potential impact of any unidentified factors predisposing manual workers to higher risks of CHD are likely to be relatively modest compared with the impact of targeting known key risk factors across all social classes.

Although these conclusions only apply directly to the period 1980–2000 on which the data are based, they are still likely to be relevant to CHD prevention in the early 21st century. Current patterns of blood cholesterol and blood pressure (as measured in the Health Survey for England) show less evidence of a social class gradient than was observed in the British Regional Heart Study at baseline in 1978–1980, so that the case for population-wide prevention by reducing total cholesterol and blood pressure levels remains compelling. However, to secure both effectiveness and equity in CHD prevention (and in the reduction of all-cause mortality), these population-wide measures would logically include measures to encourage smoking cessation among socially disadvantaged groups.

Conclusions

Attempts to reduce social inequalities in CHD will have an appreciable, but comparatively modest effect on reducing CHD compared with the potential effects from strategies aimed at reducing the levels of major established CHD risk factors in the whole population.

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KEY MESSAGES

- After correction for measurement imprecision approximately one-fifth of major coronary heart disease (CHD) events and nearly one-third of all deaths occurring during middle age can be attributed to the excess risks experienced by manual men over non-manual men.
- Established coronary risk factors explain up to one-half of these differences, leaving only approximately 10% of major CHD and 16% of premature deaths attributable to other socioeconomic factors.
- Population-wide strategies to reduce major CHD risk factors are likely to have greater potential benefits for CHD prevention than strategies designed specifically to reduce social inequalities in CHD.