Inequalities in heart failure in older men: prospective associations between socioeconomic measures and heart failure incidence in a 10-year follow-up study

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Aims
Socioeconomic position has been linked to incident heart failure (HF), but the underlying mechanisms are unclear. We examined the association of socioeconomic measures with incident HF in older adults and examined possible underlying pathways.

Methods and results
A socially representative cohort of men aged 60–79 years in 1998–2000 from 24 British towns was followed-up for 10 years for incident HF. Adult socioeconomic position was based on a cumulative score, including occupation, education, housing tenure, pension, and amenities. Childhood socioeconomic measures included father’s occupational social class and household amenities. Prevalent myocardial infarction and HF cases were excluded. Among 3836 men, 229 incident cases of HF occurred over 10 years. Heart failure risk increased with an increasing score of adverse adult socioeconomic measures (P for trend = <0.0001). Compared with men with a score of 0, the hazard ratio for men with a score of ≥4 was 2.19 (95% confidence interval, CI, 1.34–3.55), which was attenuated to 1.87 (95% CI 1.12–3.11) after adjusting for systolic blood pressure, body mass index, smoking, HDL-cholesterol, diabetes, and lung function. Adjustment for left ventricular hypertrophy, atrial fibrillation, heart rate, and renal function made little difference. Further adjustment for C-reactive protein, von Willebrand Factor, N-terminal pro-brain natriuretic peptide, and plasma vitamin C also made little difference to the hazard ratio [1.89 (95% CI 1.10–3.24)]. Heart failure risk did not vary by childhood socioeconomic measures.

Conclusion
Heart failure risk in older men was greater in the most deprived socioeconomic groups, which was only partly explained by established risk factors for HF. Novel risk factors contribute little to the associated risk.

Keywords
Heart failure • Inequalities • Socioeconomic factors • Pathways

Introduction
Heart failure (HF) is an important contributor to morbidity and mortality in the elderly and thus, a significant public health challenge in countries with growing older populations.¹,² Studies indicate that inequalities exist in HF, such that lower compared with higher socioeconomic groups have a greater risk of HF.³,⁴ However, compared with studies on coronary heart disease (CHD), there are fewer community-based studies that report longitudinal associations between socioeconomic factors and HF among older people. Moreover, there is little evidence on the influence of early life and adult socioeconomic measures on HF in older people. Furthermore, possible pathways underlying these associations remain to be explored. These issues were also identified in a recent review on socioeconomic factors and HF.⁴ Therefore, this paper investigates longitudinal associations between socioeconomic measures (in adulthood and early life) and incident HF over 10 years in a population-based cohort of older men from across Britain. To

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investigate possible mechanisms underlying the associations, we assessed the influence of established risk factors for HF, such as body mass index (BMI), blood pressure, diabetes, and smoking, which are also known to be related to socioeconomic factors. Additionally, we examined the role of clinical markers of HF (left ventricular function, atrial fibrillation, and renal function), and novel risk factors [including inflammatory markers such as C-reactive protein, N-terminal pro-brain natriuretic peptide (NT-proBNP), and plasma vitamin C]—the role of these markers underlying socioeconomic differences have not been previously assessed in longitudinal studies. We also used a wide range of socioeconomic measures in both early and adult life, such as occupational social class and access to amenities, to assess the cumulative impact of adverse socioeconomic measures.

Methods

The British Regional Heart Study is a longitudinal study comprising a socially and geographically representative sample of 7735 men recruited from general practices in 24 British towns. The cohort has been followed-up since 1978–80 when aged 40–59 years. All men provided written informed consent to the investigations, which were carried out in accordance with the Declaration of Helsinki. An ethical approval was provided by relevant local research ethics committees throughout. The men, aged 60–79 years, were invited for a follow-up examination between 1998 and 2000. This re-assessment included completion of a questionnaire on their lifestyle and medical history; physical examination; and blood samples after a minimum 6-h fast were collected using the Sarstedt Monovette system. Four thousand two hundred and fifty-two men (77% of surviving subjects; 80% in non-manual social classes; and 70% in manual groups) attended the examination; 4094 had at least one measurement of biological factors. The men were asked whether a doctor had ever told them that they had angina or had experienced a myocardial infarction (MI; heart attack and coronary thrombosis), HF, or stroke.

Cardiovascular risk factors

Details of risk factors [smoking, physical activity, BMI, waist circumference, alcohol intake, blood pressure, blood lipids, glucose, lung function, renal function to estimate glomerular filtration rate (eGFR), inflammatory markers and plasma vitamin C] in this cohort have been described. Prevalent diabetes included men with doctor-diagnosed diabetes or fasting glucose ≥7 mmol/L. Electrocardiographic left ventricular hypertrophy (LVH) was defined according to Minnesota codes (codes 3.1 or 3.3).

Measures of socioeconomic position

The longest-held occupation of subjects at study entry (aged 40–59 years) was used to define social class using the Registrar Generals’ Social Class Classification—I (professionals, e.g., physicians, engineers), II (managerial, e.g., teachers, sales managers), III non-manual (semi-skilled non-manual, e.g., clerks, shop assistants), III manual (semi-skilled manual, e.g., bricklayers), IV (partly skilled, e.g., postmen), and V (unskilled, e.g., porters, general labourers). Men in the armed forces were excluded from analyses (n = 112). Apart from occupational social class, other measures available in the cohort are education (age at leaving full-time education), car and house ownership, pension (state only or state plus private pension), and availability of central heating at home; these were collected as part of questionnaires completed at age 60–79 years. A composite score combining adverse socioeconomic measures was used to examine the cumulative impact of low socioeconomic position, and to consider a range of measures that may have a greater influence than occupation alone. One point was assigned for each of—manual social class (social Class III manual, IV or V), not a car or house owner, age at leaving full-time education <14 years, no central heating, and state pension only (similar to cumulative scores used previously).

Childhood socioeconomic measures were collected through postal questionnaires in 1992. Subjects completed a questionnaire asking about the kind of job their fathers did for the longest period of their lives. Subjects were categorized into manual (3752 (71%)) and non-manual (1436 (27%)) childhood social class groups using the Office of Population Censuses and Surveys Classification of Occupations (1980) social class coding index manual. Men (n = 115) whose fathers’ longest-held occupation was the armed forces were excluded. Information was also collected on childhood household amenities to enable a better assessment of early life socioeconomic position. Subjects were asked if their home had a bathroom, hot water supply, family car ownership, and whether they shared a bedroom with siblings.

Follow-up

The cohort has been followed-up through regular 2-yearly reviews of general practitioner records for morbidity and through the National Health Service Central Register for mortality. Incident HF from 1998–2000 until 2010 was based on a doctor-confirmed diagnosis of HF from medical records (including hospital and clinic correspondence). All cases were verified by a review of available clinical information from primary and secondary care records (symptoms, signs, investigations, and treatment response) to ensure they are consistent with current recommendations on HF diagnosis. CHD incidence included non-fatal and fatal MI. Non-fatal MI was defined by the presence of at least two of—severe prolonged chest pain, electrocardiograph (ECG) evidence of MI, and cardiac enzymes changes consistent with MI—ascertained by regular 2-yearly reviews of general practitioner records. Fatal MI was identified as deaths with International Classification of Diseases, 9th revision (ICD-9) codes of 410–414 (equivalent to ICD-10 codes I20–I25). Four hundred and thirteen men with doctor-diagnosed prevalent HF or MI were excluded from this analysis.

Statistical analysis

Cox proportional hazards models were used to examine the risk of incident HF across socioeconomic groups. Hazard ratios (HRs) and 95% confidence intervals (95% CIs) were obtained for adult social class groups (social class I as the reference group), and for the combined score of adverse socioeconomic conditions (score 0 was the reference group). Similarly, HRs were also obtained for childhood social class and household amenities (score indicating the number of childhood household amenities available). Cox models were adjusted for age followed by further adjustments sequentially for systolic blood pressure, diabetes, BMI, HDL-C, full expiratory volume in 1 s (FEV1), smoking, and alcohol consumption; LVH, atrial fibrillation, heart rate, and eGFR; CRP, von Willebrand factor (vWF), and NT-proBNP; and finally, plasma vitamin C. For the adjustments, age, systolic blood pressure (BP), high density lipoprotein cholesterol (HDL-C), BMI, heart rate, eGFR, C-reactive protein (CRP), vWF, NT-proBNP, and vitamin C were fitted as continuous variables. Distributions of CRP, NT-proBNP, and plasma vitamin C were skewed and logarithm transformations were used. Adult social class (six levels); combined adverse socioeconomic conditions (six levels); childhood social class (two levels), smoking (eight levels), physical activity (five levels), and alcohol intake (five levels) were fitted as categorical variables in adjusted models. All analyses were carried out using SAS version 9.3.
Results

Among 3836 individuals free of HF in 1998–2000, 229 incident cases of HF occurred over the follow-up period of 10 years. Table 1 summarizes the distribution of age, established and novel risk factors, and medication use by occupational social class. The proportions of current smokers, overweight, and physically inactive men were higher in lower social class groups, particularly III manual. Levels of CRP, vWF, and NT-proBNP were greater in lower social classes, while plasma vitamin C was lower.

Hazard ratio (95% CI) according to the social class and the score of adverse socioeconomic factors are given in Table 2. The HR for HF was highest in social classes IV and V compared with social class I, although not statistically significant; HRs did not vary appreciably in other social class groups. The HR for social classes IV and V compared with the rest of the groups was 1.53 (95% CI 1.06–2.19); this weakened on adjustment for systolic blood pressure, HDL-C, BMI, diabetes, smoking, and alcohol consumption (1.45; 95% CI 0.99–2.12).

Hazard ratio for HF increased with an increasing score of adverse socioeconomic factors (P for trend = <0.0001). Hazard ratio was greatest in those with a score of 3 (1.87; 95% CI 1.24–2.82) or ≥4 (2.19; 95% CI 1.34–3.55) adverse socioeconomic factors compared with those with 0. These estimates weakened slightly on adjustment for systolic blood pressure, HDL-C, BMI, smoking, alcohol consumption, and lung function (FEV1) [1.53 (95% CI 0.98–2.38) for score 3 and 1.87 (95% CI 1.12–3.11) for score ≥4]. Further adjustment for LVH, atrial fibrillation, and renal function made little difference to the results. Additional adjustment for CRP, vWF, NT-proBNP, and plasma vitamin C also did not materially change these estimates, although the increased risk in those with the highest score of ≥4 still remained statistically significant (HR = 1.89; 95% CI 1.10–3.24). Sensitivity analysis excluding men with incident CHD that occurred before a HF event (n = 24) was carried out and showed similar results (results not shown).

Table 3 presents age-adjusted HR (95% CI) for HF according to childhood social class and a score of increasing number of household amenities. No difference in HR was observed across these groups. No association was also observed with combined childhood and adult socioeconomic positions (Table 4).

Discussion

This study reports marked socioeconomic differences in HF risk in older British men aged 60–79 years followed-up for 10 years.

### Table 1 Baseline characteristics among 3836 men aged 60–79 years in 1998–2000 with no previous history of heart failure or myocardial infarction according to the occupational social class

<table>
<thead>
<tr>
<th>Social class</th>
<th>I (n = 376)</th>
<th>II (n = 1021)</th>
<th>III non-manual (n = 399)</th>
<th>III manual (n = 1499)</th>
<th>IV and V (n = 436)</th>
<th>P-value for trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>68 (5)</td>
<td>69 (6)</td>
<td>69 (6)</td>
<td>69 (5)</td>
<td>69 (6)</td>
<td>0.28</td>
</tr>
<tr>
<td>Childhood manual social class</td>
<td>144 (42)</td>
<td>550 (58)</td>
<td>243 (66)</td>
<td>1096 (83)</td>
<td>340 (88)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>≥ 3 Adverse socioeconomic factorsa</td>
<td>2 (0.53)</td>
<td>22 (2)</td>
<td>24 (6)</td>
<td>466 (31)</td>
<td>190 (44)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Current smokers</td>
<td>22 (6)</td>
<td>89 (9)</td>
<td>43 (11)</td>
<td>238 (16)</td>
<td>82 (19)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Heavy drinkersb</td>
<td>85 (23)</td>
<td>193 (19)</td>
<td>69 (17)</td>
<td>268 (18)</td>
<td>72 (17)</td>
<td>0.04</td>
</tr>
<tr>
<td>Physically inactive</td>
<td>93 (25)</td>
<td>283 (28)</td>
<td>146 (37)</td>
<td>503 (34)</td>
<td>174 (40)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Obese (BMI ≥ 30 kg/m²)</td>
<td>46 (12)</td>
<td>136 (13)</td>
<td>62 (16)</td>
<td>292 (19)</td>
<td>81 (19)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Systolic blood pressure, mmHg</td>
<td>149 (25)</td>
<td>150 (25)</td>
<td>151 (23)</td>
<td>150 (23)</td>
<td>150 (25)</td>
<td>0.87</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td>1.39 (0.4)</td>
<td>1.35 (0.33)</td>
<td>1.32 (0.33)</td>
<td>1.30 (0.34)</td>
<td>1.33 (0.35)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>FEV1, L</td>
<td>2.8 (0.6)</td>
<td>2.7 (0.6)</td>
<td>2.6 (0.7)</td>
<td>2.6 (0.7)</td>
<td>2.4 (0.7)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Diabetics</td>
<td>39 (10)</td>
<td>109 (11)</td>
<td>40 (10)</td>
<td>182 (12)</td>
<td>37 (9)</td>
<td>0.86</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td>26 (7)</td>
<td>76 (7)</td>
<td>28 (7)</td>
<td>120 (8)</td>
<td>42 (10)</td>
<td>0.15</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>12 (3)</td>
<td>48 (5)</td>
<td>8 (2)</td>
<td>42 (3)</td>
<td>14 (3)</td>
<td>0.10</td>
</tr>
<tr>
<td>Heart rate</td>
<td>64 (14)</td>
<td>65 (12)</td>
<td>66 (11)</td>
<td>67 (13)</td>
<td>67 (13)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>eGFR, mL/min (renal function)</td>
<td>74 (13)</td>
<td>72 (12)</td>
<td>73 (13)</td>
<td>73 (13)</td>
<td>73 (13)</td>
<td>0.80</td>
</tr>
<tr>
<td>CRP, mg/L</td>
<td>1.21 (0.60–2.14)</td>
<td>1.48 (0.70–2.98)</td>
<td>1.84 (0.87–3.90)</td>
<td>1.90 (0.90–3.94)</td>
<td>2.08 (1.00–4.38)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>vWF, IU/dL</td>
<td>131.28 (45.14)</td>
<td>134.09 (44.32)</td>
<td>138.88 (44.94)</td>
<td>139.76 (45.97)</td>
<td>150.60 (48.53)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>NT-proBNP, pg/mL</td>
<td>84.8 (38–165)</td>
<td>86.5 (42–160)</td>
<td>93.7 (46–166)</td>
<td>91.8 (45–179)</td>
<td>101.5 (48–201)</td>
<td>0.01</td>
</tr>
<tr>
<td>Plasma vitamin C, μmol/L</td>
<td>27.1 (19–42)</td>
<td>25 (17–43)</td>
<td>24 (16–42)</td>
<td>19.9 (12–37)</td>
<td>18.9 (10–37)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Use of ACE inhibitors, angiotensin II inhibitors, or aldosterone antagonists</td>
<td>7 (2)</td>
<td>40 (4)</td>
<td>7 (2)</td>
<td>47 (3)</td>
<td>9 (2)</td>
<td>0.29</td>
</tr>
<tr>
<td>Use of beta-blockers</td>
<td>45 (12)</td>
<td>115 (11)</td>
<td>52 (13)</td>
<td>160 (11)</td>
<td>63 (15)</td>
<td>0.31</td>
</tr>
</tbody>
</table>

Values are mean (SD), number (%), or geometric mean (inter-quartile range).

aScore includes: no car, not house owner, state pension only, fuel poverty—not central heating, manual SC, education ≤ 14 years.
b≥6 units (1 UK unit = 10 g) of alcohol daily/on most days in the week.
These inequalities were particularly apparent with a low socioeconomic position based on a combined measure of adverse socioeconomic factors in adult life. Childhood socioeconomic factors did not appear to be related to HF risk in older age. The increased risk of HF in those with greater adverse socioeconomic factors was partly attenuated by established risk factors including blood pressure, BMI, smoking, physical activity, and lung function; the residual increased risk in lower socioeconomic groups remained unexplained by established and novel risk factors.

To our knowledge, this is the first report of longitudinal associations between socioeconomic measures over the lifecourse and HF in an older British population, which also investigates the role of childhood measures.
of novel risk factors, including inflammatory markers and plasma vitamin C. Our results confirm those of other studies demonstrating socioeconomic differences in incident HF. These studies, including a recent English study, reported differences in HF risk according to neighbourhood-level socioeconomic deprivation, whereas others have reported inequalities based on education and occupational social class. Similar to the results of our study, the only other longitudinal studies (to our knowledge), which examine the role of cardiovascular risk factors, found that the increased HF risk in lower socioeconomic groups was partly weakened by established cardiovascular factors, including blood pressure, cholesterol, diabetes, smoking, and physical activity. Two of these longitudinal studies found stronger associations between occupational social class and HF risk (adjusted increased risk of 55 and 72% in low vs. high occupational groups) than reported in our study. However, the age of subjects was lower (<55 years) in these studies compared with our cohort of older men (60–79 years). Unlike the previously reported strong association in our cohort of occupational social class with CHD in older age, the relationship with HF was weak, with a modest increased risk observed only in the lowest occupational social classes.

This report also extends previous findings by the use of a combination of different socioeconomic factors, including occupation, education, car and house ownership, pension status, and central heating. The association of this composite score of socioeconomic position with HF was stronger than that of occupational social class. The influence of more adverse socioeconomic factors (including material deprivation) on HF is present in older age and remained after adjustment for established and novel risk factors as possible pathways underlying socioeconomic differences. Clinical risk factors such as LVH, atrial fibrillation, and renal function made little contribution, suggesting a minimal role underlying these associations. Novel inflammatory markers also made little further difference to the associations, and a residual increased relative risk of 1.89 remained in the most deprived socioeconomic group.

It is notable that these associations observed were also not due to the greater risk of MI in lower socioeconomic groups (those with previous MI were excluded and exclusion of those with incident MI did not alter the results). The socioeconomic differences in HF did not appear to be due to differences in medication use; use of beta-blockers and other anti-hypertensive drugs was similar across socioeconomic groups. However, limited access to healthcare, particularly diagnostic facilities, among lower socioeconomic groups, may contribute to an underestimation of socioeconomic differences in HF; such underlying inequalities along the diagnostic pathway need further exploration. The role of other factors such as diet, and treatment for HF, also needs to be explored. There is also growing evidence for environmental factors such as air pollution related to the incident HF; it is possible that these environmental factors contribute to inequalities in HF. The role of environmental factors along with neighbourhood-level socioeconomic measures in older age may shed more light on pathways underlying inequalities in HF.

Our study observed no difference in HF risk according to the childhood socioeconomic position or its combined effect with adult socioeconomic position. The Atherosclerosis Risk in Communities (ARIC) study was the only other study we found to report the influence of early life socioeconomic factors on HF. That study, with a younger cohort (45–64 years) than our study, reported a modestly increased risk in participants of low childhood socioeconomic position, which was abolished on adjustment for adult socioeconomic position. Although the influence of childhood socioeconomic position on adult socioeconomic and cardiovascular risk factors has been well reported, its independent influence on HF was not observed.

**Strength and limitations**

Our cohort is a socially and geographically representative sample of older British men. However, the study comprised mostly Caucasian men and, therefore, generalizability to other ethnic groups may be limited. Generalizability to women may also be limited due to possible gender differences in associations with socioeconomic measures. Nevertheless, studies report socioeconomic differences in different ethnic groups among men and women. Since the purpose of this study was to investigate inequalities in HF in older age, the cohort, inevitably, was without those who would have died at an earlier age with more severe forms of HF. Nevertheless, contact has been maintained with >98% of the cohort since initial recruitment in 1978–80. A particular strength of the study is the high response rate (77%) at the re-examination when subjects were aged 60–79 years, which minimizes a response bias. Although non-response was slightly greater among manual compared with non-manual groups, levels of BMI and cholesterol were similar in responders and non-responders, although smoking levels were greater in non-responders. If anything, the difference in responders and non-responders may have led to a slight underestimation of the extent of inequalities observed in HF.

Moreover, a range of socioeconomic measures were used including those that are likely to be stable across most of adult life (occupational social class and education), as well as socioeconomic measures more relevant in older age (including pension arrangements and availability of central heating). Socioeconomic position based on the longest-held occupation of the subjects assessed at middle age was available for almost all participants; this changed only for a small proportion of subjects (<10%) after a 20-year follow-up.

**Conclusion**

This report shows that the impact of adverse socioeconomic factors on HF risk is present in older age. Given the high risk of HF with increasing age, the results highlight the importance of addressing inequalities in older populations. Potentially modifiable risk factors such as BMI, physical activity, smoking, and lung function made some, albeit modest contribution to these, inequalities. Control of these established cardiovascular risk factors in older populations will make some contributions to the inequalities in HF. Although individual-level measures were explored, the role of other possible factors including environmental and area-level socioeconomic factors merits further research. This study emphasizes the need to better understand pathways linking socioeconomic factors and HF risk.

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