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CARDIOVASCULAR DISEASE

Income and recurrent events after a coronary event in women

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Abstract Strong evidence supports the existence of a social gradient in poor prognosis in patients with coronary heart disease (CHD). However, knowledge regarding what factors may explain this relationship is limited. We aimed to analyze in women CHD patients the association between personal income and recurrent events and to determine whether lifestyle, biological and psychosocial factors contribute to the explanation of this relationship. Altogether 188 women hospitalized for a cardiac event were assessed for personal income, demographic factors, lipids, inflammatory markers, cortisol, creatinine, lifestyle and psychosocial factors, i.e. alcohol consumption, smoking habits, body-mass index, depressive symptoms, anxiety, vital exhaustion, availability of social interaction, hostility and anger-related characteristics and were followed for cardiovascular death and recurrent acute myocardial infarction (AMI). During the 6-year follow-up 18 patients deceased and 31 experienced cardiovascular death or non-fatal AMI. After adjustment for confounders, patients with medium and high income had lower risk for recurrent events relative to those with low income (HR (95% CI): 0.38 (0.15–0.97) and 0.39 (0.17–0.93), respectively). Controlling for smoking reduced by 12.8% the risk for recurrent events associated with high versus low income, while adjusting for depression decreased the risk for middle versus low income by 13.5%. Anger symptoms explained 16.7% of the risk for recurrent events associated with middle versus low income and 10.2% of the risk for high versus low income. We suggest that in women with CHD low income is associated with recurrent events and that smoking, depressive symptomatology and anger symptoms may contribute to the explanation of this relationship.

Keywords Coronary heart disease · Income · Recurrent events · Socioeconomic status · Women

Abbreviations

ACE inhibitor	Angiotensin-converting enzyme inhibitor
ANOVA	Analysis of variance
AMI	Acute myocardial infarction
ApoA1	Apolipoprotein A1
ApoB	Apolipoprotein B
BDI	Beck Depression Inventory
BMI	Body mass-index
CABG	Coronary artery bypass grafting
CHD	Coronary heart disease
CI	Confidence interval
HDL	High density lipoprotein
HR	Hazard ratio
hsCRP	High-sensitivity C-reactive protein
IL-6	Interleukin 6
LP (a)	Lipoprotein (a)
LDL	Low density lipoprotein
PCI	Percutaneous coronary intervention
SD	Standard deviation
SEK	Swedish crown
SES	Socioeconomic status

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Introduction

Socioeconomic status (SES), defined most often by means of income, educational attainment, occupational class, or a combination of these factors, has been repeatedly found in Western societies to be inversely associated with coronary heart disease (CHD) incidence [1–5], prevalence [6, 7] and mortality [6, 8, 9]. There is evidence for a similar social gradient in morbidity and mortality among patients with an already developed CHD. Patients lower in the socioeconomic hierarchy have worse prognosis and are at higher risk for mortality compared to those in a better socioeconomic position [4, 10–12].

Although the mechanisms that may explain the social gradient in CHD are not entirely understood, it has been suggested that several biological, behavioural and psychosocial risk factors may mediate the association between SES and CHD [13, 14]. Compelling evidence suggests that, compared to those with a better position, individuals from lower socioeconomic groups are more likely to be obese [3, 7, 9, 15–17], smokers [3, 7, 9, 15–18] and heavy drinkers [3, 16], to do less physical exercise [7, 9, 16] and to consume more atherogenic food [15].

Biological risk factors for CHD, such as lipids [3, 15–18], inflammatory markers [19, 20], haemostatic factors [21, 22], blood pressure [23], glucose levels [16, 17], heart rate [3], history of diabetes [17, 18] and lower cortisol response to stress [24] have also been shown to be related to socioeconomic measures.

At the same time, those in lower socioeconomic position seem to score higher on psychological questionnaires measuring depression [5, 25], anxiety [5], vital exhaustion [24], stress [26], work-related stressors [16], hostility [27], anger [16], while they report lower levels of social support [16, 24, 26].

Due to their relation to socioeconomic measures, on the one hand, and to CHD on the other, the above factors may be regarded as potential mediators of the relationship between socioeconomic position and disease. However, despite this theoretical background, only a limited number of studies have investigated whether these risk factors really contribute to the explanation of the socioeconomic differences in cardiovascular morbidity and mortality in initially healthy samples [1, 6–8, 28–30] or in CHD patients.

Except for the SESAMI Study [10] and the Beta Blocker Heart Attack Trial [31] we know of no other studies that have examined biological, lifestyle-related or psychosocial factors as potential explanatory factors of the socioeconomic differential in prognosis in CHD. These two studies were, however, conducted on either mixed or male samples, therefore paid less or no attention to women patients. Women's socioeconomic position [32], cardiovascular risk factors [33], the pattern of the development and prognosis of CHD [33, 34] are known to differ from that of men; consequently, explanatory factors of the socioeconomic differential in prognosis in CHD might, as well, be different for the two genders.

Therefore, our purpose was two-fold. The first objective was to analyze the association between personal income, a measure of socioeconomic position and recurrent events in women with CHD. The second aim was to determine whether clinical, behavioural and psychosocial factors can explain the social gradient in recurrent events in women with established CHD.

Methods

Study population

The original study population consisted of 247 women that had either acute myocardial infarction (AMI), or undergone a revascularization procedure either percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG) and were hospitalized between 1996 and 2000 at Karolinska University Hospital or St Göran's Hospital in Stockholm, Sweden. The diagnosis of AMI was based on World Health Organization's criteria of typical enzyme patterns and chest pain and/or diagnostic electrocardiographic changes [35]. Consecutively, all eligible women below 75 years were approached and offered to participate in a cardiac rehabilitation program specifically designed for women [36]. Subsequently, all those who agreed to participate were randomly assigned to either the control (128 patients) or to the intervention group (119 patients). Finally, out of the originally randomized 247 patients, 12 (6 from the intervention group, 6 from the control group) did not participate in the study, resulting in 235 eligible patients. Due to missing data on personal income, 188 women were included in the present analyses. Women with complete data did not differ significantly from those with missing data in terms of most of the demographic, lifestyle, psychosocial or clinical characteristics. However, those with missing data were more likely to be from the control group of our intervention program, to have CABG as inclusion diagnose and to have higher levels of cortisol.

The Ethics Committee of Karolinska Institute at Karolinska University Hospital approved the study.

Measures

All variables were obtained in the stable phase, approximately 6–8 weeks after hospitalization.

Income assessment

Patients were asked to disclose their yearly personal income from the previous year. Six answer possibilities were provided: (1) <119,999, (2) 120,000–159,999, (3) 160,000–199,999, (4) 200,000–229,999, (5) 230,000–259,999 and (6) ≥260,000 Swedish crowns (SEK)/year, respectively. In order to optimize the statistical power for the analyses these answer alternatives were categorized into tertiles based on their distribution. Those with income below 119,999 SEK formed the low income group, the medium income group consisted of those in the 120,000–159,999 SEK interval, while those with yearly income above 160,000 SEK were assigned to the high income group.

Ascertainment of biological factors

Blood samples from the patients were drawn at 10 ± 1 h AM. Blood lipids, such as total cholesterol, high- and low-density lipoproteins, triglycerides, apolipoprotein A1, apolipoprotein B, lipoprotein (a) were assessed. Cortisol and creatinine levels were measured, as well.

Levels of high-sensitivity C-reactive protein (hsCRP) were measured by nephelometry using N-dilutent for Nephelometry, Behring OUMT 61 (Dade Behring GmbH, Marburg, Germany). Interleukin-6 (IL-6) concentrations were determined by enzyme-linked immunoassay (R and D Systems, Abingdon, UK). For IL-6, high sensitivity kits were used in order to accurately determine low levels of the cytokine [37].

Assessment of lifestyle-related factors

Smoking status was categorized as never, current or former smoker. Average daily alcohol intake was calculated in grams. Height and weight were assessed, and body-mass index (BMI) was calculated by dividing the weight with the square of the height value (kg/m^2).

Measurement of psychosocial variables

Psychosocial factors were determined using standardized psychological questionnaires. The 21 items Beck Depression Inventory (BDI) [38] was used to assess depressive symptomatology. Vital exhaustion was measured by means of the Maastricht Questionnaire [39], while the State-Trait Anxiety Inventory [40] was used to determine trait anxiety level. In measuring the availability of social interaction, the shortened version of the Interview Schedule for Social Interaction [41] was used. To determine anger-related characteristics of the participants, the anger symptoms, the anger-in, the anger-out and the anger-discuss subscales of the Framingham Anger Scale [42] were administered. Hostility scores were extracted from the Jenkins Activity Survey [43].

Other covariates

Patients were asked to indicate their household's income for the previous year; answer possibilities were identical with those provided to the item concerning personal income. The number of persons relying on the family income was also assessed. Educational attainment was classified into two levels: mandatory schooling only and completion of high school, college or university. Marital status was classified as with or without a partnership. Data on retirement, on drug therapy (beta-blockers, Ca-channel blockers, statins, aspirin and ACE inhibitors) and on whether the patient has been hospitalized due to heart disease in the last few years were collected.

Follow-up

Patients were followed for all-cause and cardiovascular mortality, and non-fatal AMI over a period of 6 years. The centralized health care system in Sweden provides virtually complete follow-up information for all patients by matching their unique 10 digit person identification numbers to the death and hospital discharge registers. The Swedish hospital discharge registers of AMI were validated using hospital discharge data and mortality data and were found to have adequate sensitivity and specificity [44].

Statistical analyses

Variables that showed skewed distribution were logarithmically transformed for all analyses to approximate normal distribution. However, in Table 1 we present the mean and standard deviation of these data without logarithmic transformation to allow comparison with other studies. One-way ANOVA was used to determine the statistical significance of differences between continuous variables for three groups. Categorical data were compared by chi-square tests.

Un- and multiadjusted Cox proportional hazard models were performed to examine the association between personal income and all-cause death, cardiovascular mortality and the combination of cardiovascular mortality and non-fatal AMI. Due to limited statistical power only age and confounders that were found to modify the regression coefficient associated with low income at least by 10% [45], i.e. marital status, education, and the interaction term between marital status and age were included in the base model. We also performed several alternative base models when we adjusted—in addition to age, marital status, education, and the interaction term between marital status

Table 1 Distribution of the study variables according to the level of personal income

Variables	*N*	Total sample ($n = 188$)	Low personal income ($n = 53$)	Medium personal income ($n = 53$)	High personal income ($n = 82$)	*P* value[a]
Age (years)	188	62.0 (8.9)	64.6 (8.8)	64.8 (6.4)	58.4 (9.0)	<0.001
Mandatory education only (%)	187	60.4	71.2	69.8	47.6	0.006
Married or cohabiting (%)	186	57.5	67.9	48.1	56.8	0.11
Retired (%)	188	66.5	81.1	86.8	43.9	<0.001
Inclusion diagnose[b] (%)	188					
AMI		56.9	60.4	56.6	54.9	0.81
CABG		30.9	30.2	22.6	36.6	0.78
PCI		28.2	30.2	30.2	25.6	0.22
Previously hospitalized due to heart disease (%)	185	96.8	98.1	96.2	96.3	0.82
Drug therapy (%)						
ACE inhibitors	188	21.3	18.9	18.9	24.4	0.65
Statins	188	56.4	52.8	60.4	56.1	0.73
Aspirin	188	88.3	92.5	83.0	89.0	0.30
Calcium channel blockers	188	20.2	28.3	18.9	15.9	0.20
Beta blockers	188	80.9	75.5	81.1	84.1	0.45
Participated in our subsequent rehabilitation (%)	188	52.1	45.3	60.4	51.2	0.29
Participated in other rehabilitation programs (%)	188	22.9	15.1	22.6	28.0	0.22
Lifestyle factors						
Smoking	188					0.96
Never		36.2	35.8	37.7	35.4	
Former		53.7	52.8	54.7	53.7	
Current		10.1	11.3	7.5	11.0	
Alcohol consumption (g/days)[c]	166	2.4 (3.5)	2.1 (3.1)	1.8 (2.4)	2.9 (4.2)	0.31
BMI (kg/m^2)	188	26.1 (4.5)	26.2 (4.7)	26.6 (5.2)	25.7 (3.9)	0.73
Biological factors						
Total cholesterol (mmol/l)	179	5.0 (1.1)	5.0 (0.9)	5.2 (1.1)	4.8 (1.1)	0.08
HDL (mmol/l)[c]	179	1.0 (0.3)	1.0 (0.3)	1.1 (0.4)	1.0 (0.4)	0.96
LDL (mmol/l)	179	3.1 (1.0)	3.1 (0.9)	3.3 (1.1)	2.9 (0.9)	0.90
ApoA1 (g/l)	180	1.5 (0.3)	1.6 (0.3)	1.5 (0.3)	1.4 (0.2)	0.05
ApoB (g/l)	180	0.9 (0.2)	0.9 (0.2)	1.0 (0.2)	0.9 (0.2)	0.23
Triglycerides (mmol/l)[c]	179	1.8 (1.0)	1.8 (0.9)	1.8 (1.2)	1.8 (0.9)	0.95
LP (a) (g/l)[c]	180	410.3 (426.2)	383.0 (407.7)	517.2 (443.8)	393.7 (442.0)	0.04
IL-6 (mg/l)[c]	179	4.5 (5.1)	5.9 (6.7)	4.2 (3.9)	3.7 (3.2)	0.08
hsCRP (mg/l)[c]	181	5.4 (9.2)	7.3 (11.4)	4.7 (7.2)	4.1 (6.9)	0.27
Cortisol (nmol/l)[c]	181	284.9 (120.1)	283.6 (130.2)	284.2 (112.4)	268.6 (114.9)	0.11
Creatinine (μmol/l)[c]	179	71.4 (27.8)	72.2 (16.0)	75.3 (40.5)	69.7 (27.7)	0.53
Psychosocial factors						
Depressive symptomatology	154	10.9 (6.6)	12.8 (6.7)	9.7 (6.0)	10.15 (6.6)	0.05
Trait anxiety	175	44.6 (4.9)	45.2 (5.2)	44.5 (5.2)	44.1 (4.4)	0.44
Vital exhaustion	159	21.1 (10.1)	20.9 (9.2)	22.0 (9.1)	19.8 (11.4)	0.54
Availability of social interaction	162	20.7 (4.8)	20.6 (4.6)	19.0 (4.9)	22.2 (4.7)	0.002
Hostility	184	7.1 (0.9)	7.0 (0.9)	7.1 (0.9)	7.2 (0.9)	0.52

Table 1 continued

Variables	*N*	Total sample ($n = 188$)	Low personal income ($n = 53$)	Medium personal income ($n = 53$)	High personal income ($n = 82$)	*P* value[a]
Anger symptoms	181	5.1 (3.2)	5.3 (3.4)	4.8 (3.1)	5.1 (3.1)	0.79
Anger-in	182	2.6 (1.8)	2.9 (1.7)	2.7 (1.9)	2.2 (1.8)	0.13
Anger-out	184	0.6 (0.9)	0.5 (0.8)	0.5 (0.8)	0.6 (0.9)	0.7
Anger-discuss	182	3.0 (1.7)	2.6 (1.7)	2.8 (1.4)	3.4 (1.8)	0.02
AMI and cardiovascular death (%)	188	16.5	26.4	13.2	12.2	0.07
Cardiovascular mortality (%)	188	5.3	9.4	7.5	1.2	0.08
Total mortality (%)	188	9.6	20.8	7.5	3.7	0.004

Data are presented as means and standard deviations for continuous variables and as percent for categorical variables. AMI, acute myocardial infarction; CABG, coronary artery bypass grafting; PCI, percutaneous coronary intervention; HDL, high density lipoprotein; LDL, low density lipoprotein; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; LP (a), lipoprotein (a); hsCRP, high sensitivity C-reactive protein; IL-6, interleukin-6; BMI, body-mass index

[a] P is the probability value calculated according to one-way ANOVA for continuous data and according to chi-square test for categorical data

[b] The categories are not mutually exclusive

[c] The variable was logarithmically transformed because of skewed distribution. Means and standard deviations are presented as values before the logarithmical transformation

and age—for (1) retirement, (2) previous hospitalization in the last years due to CHD, (3) inclusion diagnosis, (4) drug therapy, (5) participation in our subsequent rehabilitation program and (6) participation in other rehabilitation programs. Stratified analyses and formal tests for interactions were conducted, as well, to assess possible effect modification.

In order to examine potential mediators of the association between income and the combination of cardiovascular death and recurrent AMI several lifestyle-related, biological and psychosocial CHD risk factors were added one by one to the base model. We used the change-in-point-estimate strategy [45] to determine to what extent each risk factor contributes to the explanation of the association of interest. The percentage of the contribution of individual risk factors was computed according to the formula:

$$\Delta = \frac{\ln \mathrm{HR}_{\text{base model}} - \ln \mathrm{HR}_{\text{base model} + \text{explanatory factor}}}{\ln \mathrm{HR}_{\text{base model}}} \times 100$$

SAS 9.1 and SPSS 11.5 for Windows were used for statistical analyses.

Results

Baseline characteristics

Table 1 presents the distribution of demographic, lifestyle, clinical and psychosocial factors according to the three levels of the personal income. Women with high personal income were younger than those who earned less. The mean age in the high, medium and low-income groups was 58.4 (SD = 9.0), 64.8 (6.4) and 64.6 (8.8) years, respectively. Women with higher income tended to be more educated. The percentage of women who had attended only mandatory school was 71.2%, 69.8% and 47.6% in the low-, medium- and high-income groups, respectively. Women with low and medium income were more likely to have been retired (81.1% and 86.8%) compared to women with high income (43.9%). Women with low income were somewhat more likely to live in a partnership (67.9%) when compared to women with medium (48.1%) or high income (56.8%). Inclusion diagnoses, previous hospitalization due to CHD, drug therapy, participation in our rehabilitation program and lifestyle factors were largely comparable across the income groups. Participation in other rehabilitation programs tended to be more frequent as income increased. There was no clear trend concerning the relationship between the different lipids, cortisol and creatinine and income categories. Serum levels of both IL-6 and hsCRP decreased with increasing income.

Women with low personal income had higher BDI scores than women with medium or high income, 12.8 (6.7) versus 9.7 (6.0) and 10.15 (6.6), respectively. The availability of social interaction was the lowest among women with a medium income. Scores on the anger-discuss scale tended to increase with increasing income, while for the anger-in scores an opposite tendency was observed.

Personal income and recurrent events

During the follow-up period there were 18 deaths from any cause (9.6%), 10 cardiovascular deaths (5.3%), while 31 patients had either cardiovascular death or non-fatal AMI (16.5%). Income showed an inverse relationship with adverse outcome. Table 2 presents the hazard ratios when

Table 2 Associations between personal income and prognosis after AMI

Outcome	Income tertile	*N*	Number of events	HR and 95% CI	
				Unadjusted	Base model[a]
Cardiovascular mortality and non-fatal AMI	Low	53	14	1	1
	Medium	53	7	0.47 (0.19–1.16)	0.38 (0.15–0.97)
	High	82	10	0.46 (0.20–1.04)	0.39 (0.17–0.93)
Cardiovascular mortality	Low	53	5	1	1
	Medium	53	4	0.77 (0.20–2.86)	0.57 (0.13–2.39)
	High	82	1	0.12 (0.02–1.09)	0.12 (0.01–1.18)
All-cause mortality	Low	53	11	1	1
	Medium	53	4	0.34 (0.11–1.08)	0.33 (0.10–1.09)
	High	82	3	0.17 (0.04–0.63)	0.19 (0.05–0.75)

AMI, acute myocardial infarction; HR, hazard ratio; CI, confidence interval

[a] Base model includes confounders, such as age, marital status, education and the interaction between marital status and age

medium- and high-income groups were compared to the low-income group. When we adjusted for confounders, i.e. age, marital status, education and the interaction between marital status and age, both the medium and high income groups had lower risk for recurrent events than those with low income. Patients in the middle-income group had significantly lower risk for the combination of cardiovascular death and non-fatal AMI than those in the low-income group, the hazard ratio (HR) and the 95% confidence interval (CI) being 0.38 (0.15–0.97). When the groups with high and low income were compared, the multiadjusted models showed significantly higher total mortality and higher risk for the combination of cardiovascular mortality and non-fatal AMI for the latter group. The corresponding HR (95% CI) were 0.19 (0.05–0.75) and 0.39 (0.17–0.93), respectively. When alternatively we categorized income as quartiles we obtained similar results in essence though with less power.

We have also performed alternative base models when we adjusted—in addition to the factors already included to the base model—for (1) retirement, (2) previous hospitalization in the last years due to CHD, (3) inclusion diagnosis, (4) drug therapy (beta blocker, calcium channel blocker, statin, aspirin and ACE inhibitor), (5) participation in our and (6) in other rehabilitation programs. We obtained essentially similar results in these alternative models, i.e. there was no evidence for confounding from these variables.

We have also examined possible effect modifications. We performed stratified analyses according to age (median split), marital status, education, retirement, previous hospitalizations due to CHD, participation in our rehabilitation program, hospital catchment area and inclusion diagnoses. We found roughly similar associations between income and recurrent events in these selected subgroups.

Mediators between income and recurrent events

We have investigated if lifestyle and psychosocial factors, lipids, inflammatory markers, cortisol or creatinine contribute to the explanation of the association between income and recurrent events (Table 3). We found slight decrease in risk associated with the lower income category when adjusting for smoking, depression and anger symptoms. Adjustment for smoking resulted in a decrease of 12.8% of the risk for the high versus low income group. With depression, the corresponding decrease was 13.5% when middle and low income groups were compared and 9.3% when high and low income groups were compared. When adding the anger symptoms scale to the base model the risk of the middle versus low income group was reduced by 16.7%, whereas that corresponding to the high versus low income groups dropped by 10.2%. After controlling for alcohol consumption, anger-in and anger discussion the association between income and the combined endpoint of cardiovascular death and non-fatal AMI became even stronger. The regression coefficient for the high versus low income decreased by 19.4% after adjustment for alcohol intake and by 14.6% after controlling for anger discuss. Adjustment for anger-in resulted in a 14.6% decrease of the regression coefficient for low versus middle income groups. The effect of the additional adjustment for the rest of the potential mediators was negligible.

Additional analyses

In secondary analyses, we investigated the association between two other measures of SES—educational attainment and household income—and recurrent events. After adjustment for potential confounders, i.e. age, education, marital status and the number of persons relying on the

Table 3 Hazard ratios and 95% confidence intervals for the association between income and recurrent events before and after adjustment for potentially mediating factors

	N	Corresponding base model[a] HR (95% CI)	HR (95% CI)
Lifestyle factors			
Base model + smoking	187	0.38 (0.15–0.96)	0.40 (0.16–1.03)
		0.39 (0.16–0.93)	0.44 (0.18–1.07)
Base model + alcohol consumption	166	0.46 (0.16–1.33)	0.46 (0.16–1.34)
		0.55 (0.20–1.52)	0.49 (0.17–1.38)
Base model + BMI	187	0.38 (0.15–0.96)	0.37 (0.14–0.93)
		0.39 (0.16–0.93)	0.42 (0.18–1.01)
Biological factors			
Base model + total cholesterol	179	0.46 (0.17–1.19)	0.47 (0.18–1.24)
		0.47 (0.19–1.16)	0.48 (0.19–1.19)
Base model + HDL cholesterol	179	0.46 (0.17–1.19)	0.43 (0.16–1.14)
		0.47 (0.19–1.16)	0.47 (0.19–1.16)
Base model + LDL cholesterol	179	0.46 (0.17–1.19)	0.48 (0.18–1.25)
		0.47 (0.19–1.16)	0.48 (0.19–1.20)
Base model + ApoA1	180	0.45 (0.17–1.17)	0.45 (0.17–1.17)
		0.47 (0.19–1.16)	0.47 (0.19–1.16)
Base model + ApoB	180	0.45 (0.17–1.17)	0.45 (0.17–1.17)
		0.47 (0.19–1.16)	0.46 (0.19–1.16)
Base model + triglycerides	179	0.46 (0.17–1.19)	0.45 (0.17–1.17)
		0.47 (0.19–1.16)	0.48 (0.19–1.19)
Base model + LP (a)	180	0.45 (0.17–1.17)	0.45 (0.17–1.18)
		0.47 (0.19–1.16)	0.46 (0.18–1.15)
Base model + IL-6	179	0.48 (0.18–1.27)	0.46 (0.17–1.22)
		0.50 (0.20–1.26)	0.49 (0.20–1.24)
Base model + hsCRP	181	0.46 (0.17–1.19)	0.45 (0.17–1.17)
		0.48 (0.19–1.18)	0.47 (0.19–1.16)
Base model + cortisol	181	0.46 (0.17–1.19)	0.46 (0.18–1.19)
		0.48 (0.19–1.18)	0.48 (0.19–1.18)
Base model + creatinine	179	0.46 (0.17–1.19)	0.45 (0.17–1.18)
		0.47 (0.19–1.16)	0.48 (0.19–1.17)
Psychosocial factors			
Base model + depressive symptomatology	154	0.37 (0.11–1.25)	0.42 (0.12–1.45)
		0.43 (0.15–1.18)	0.47 (0.16–1.31)
Base model + trait anxiety	174	0.45 (0.17–1.17)	0.45 (0.17–1.17)
		0.44 (0.17–1.10)	0.43 (0.17–1.10)
Base model + vital exhaustion	158	0.41 (0.14–1.13)	0.41 (0.14–1.13)
		0.41 (0.16–1.04)	0.41 (0.16–1.04)
Base model + availability of social interaction	161	0.36 (0.13–0.99)	0.36 (0.13–1.01)
		0.45 (0.18–1.13)	0.42 (0.16–1.08)
Base model + hostility	183	0.37 (0.15–0.96)	0.37 (0.14–0.95)
		0.38 (0.16–0.91)	0.38 (0.16–0.91)
Base model + anger symptoms	181	0.48 (0.18–1.23)	0.56 (0.21–1.48)
		0.49 (0.20–1.22)	0.54 (0.21–1.35)
Base model + anger-in	182	0.41 (0.16–1.04)	0.36 (0.14–0.95)
		0.42 (0.17–1.02)	0.45 (0.18–1.09)
Base model + anger-out	184	0.40 (0.15–1.02)	0.40 (0.16–1.03)
		0.42 (0.17–1.01)	0.39 (0.16–0.96)

Table 3 continued

	N	Corresponding base model[a] HR (95% CI)	HR (95% CI)
Base model + anger discussion	181	0.40 (0.16–1.03)	0.37 (0.14–0.96)
		0.42 (0.17–1.02)	0.37 (0.15–0.92)

[a] The base model when patients with missing values for the given potential mediator were excluded. Base model includes age, marital status, education and the interaction between marital status and age. For each variable, the first row represents the hazard ratio for the middle versus the low income group, while the second row represents the risk of the high compared to the low income group

AMI, acute myocardial infarction; CABG, coronary artery bypass grafting; PCI, percutaneous coronary intervention; HDL, high-density lipoprotein; LDL, low-density lipoprotein; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; LP (a), lipoprotein (a); hsCRP, high sensitivity C-reactive protein; IL-6, interleukin-6; BMI, body-mass index

family income, household income was not significantly related to the combined endpoint of cardiovascular mortality and new AMI, the HR (95% CI) being 0.78 (0.32–1.91) for the middle versus the low household income tertile and 0.41 (0.12–1.39) when comparing groups with high and low household income. Education was not significantly associated with the combined endpoint of cardiovascular mortality and new AMI, the HR (95% CI) being 0.92 (0.41–2.06) when those having at least high school were compared to those with less than high school education.

Discussion

This study investigated whether personal income predicts recurrent events in women patients with CHD. In line with previous research [4, 10, 12, 46] we found that low income was associated with higher risk of total and cardiovascular mortality, as well as with an increased risk for the combination of all cause mortality and recurrent AMI.

In explaining socioeconomic inequalities in health two major types of explanations have been suggested [47, 48]. According to the "health selection" or the "reverse causation" hypothesis health determines social position [47, 48]. This health selection can be direct, when unhealthy individuals reduce their social position as a consequence of their inferior health status or indirect, when it operates on the basis of characteristics or background factors that are related to both health and SES [47, 48]. The second set of explanations, known as the "social causation" hypothesis [47] posits that SES affects health and the risk of dying [48].

Health selection as potential explanation of our findings

Although direct health selection, i.e. the outcome measure determining income at baseline was not possible in our study, we can not exclude that previous health condition influenced both income and recurrent events. To address the possibility that those experiencing earlier a cardiac event would be more likely not to be able to work and thereby have a lower income [48], we included previous hospitalizations due to CHD in our multivariate analyses and found no evidence for confounding from this factor.

Similarly, it may be argued that psychosocial factors such as a long history of depression, anxiety, ineffective ways of coping with anger and hostility could eventually cause lower income. However, Lynch and Kaplan [49] and Kristenson and colleagues [50] argue that by differential exposure to environmental challenges, e.g. financial strain, insecure employment, low control over life, stressful life events, low self-esteem [51] and by differences in protective resources, socioeconomic factors are more likely to structure the development and maintenance of social and psychological characteristics than vice versa. For example, in the Whitehall II study the social variation in depression and psychological well-being was largely mediated by factors related to environmental challenges and protective resources, i.e. individual behaviours, psychosocial characteristics at work and social circumstances outside work [47]. Moreover, during the period when our study was conducted the amount of sick allowance in Sweden represented 90% of the previous salary; therefore a sick leave period due to previous CHD or depression was not likely to cause considerable income reduction.

CHD risk factors as explanatory factors for the social gradient in recurrent events

Besides upstream determinants of the social gradient in recurrent events, we also investigated whether lifestyle-related, biological and psychosocial factors contribute to the explanation of the relationship between income and recurrent events in women cardiac patients. By adding these risk factors one by one to the base model we analyzed to what extent each of the 23 factors contributed to the explanation of the social gradient in CHD outcome.

Concerning the traditional cardiovascular risk factors, adjustment for smoking reduced by 12.8% the excess risk of recurrent events of the low versus the high income group. This is in agreement with findings from several studies

showing smoking to contribute to the explanation of the social gradient in CHD morbidity and mortality [1, 7, 9, 30]. Results from studies regarding socioeconomic differences in smoking have to be interpreted with caution given that smoking is more socially accepted in low socioeconomic strata and therefore individuals from these groups might report their smoking more honestly. Differences in smoking among the income groups may therefore be even smaller than we actually found, thus the mediatory effect of smoking could eventually be overestimated. Adjustment for alcohol consumption resulted in a stronger association between income and recurrent events. The traditional biological risk factors included in our study and BMI contributed only modestly to the differences in recurrent events across the income groups.

Besides the well established CHD risk factors, other, so called non-traditional risk factors—inflammatory markers and psychosocial factors among others—have been suggested to be pathways through which unfavourable social circumstances may lead to CHD [8, 28, 29, 52] or to poor outcome in established disease [10, 20, 31]. Although others have found evidence for an inverse relationship between socioeconomic status and inflammatory markers [19, 20, 53] and inflammatory markers and CHD outcome [54], our data did not support a contribution of IL-6 or hsCRP to the explanation of the differences in recurrent events among the income groups.

Adjustment for anger symptoms reduced the excess risk for recurrent events associated with being in the low income group, whereas adjustment for anger-in and anger discussion resulted in stronger income-recurrent events relationship. Anger has been shown to differ among SES groups [16] and to predict prognosis in CHD [55]. We believe our study is the first to examine it as a potential intermediate factor for the social differences in CHD.

Depressive symptomatology also contributed to the explanation of the association between income and recurrent events. The social gradient in depressive symptoms is well documented [56], whereas depression has been consistently shown to predict CHD or poor outcome in already established disease [57]. So far, depression as a link between socioeconomic status and recurrent events in CHD women patients has not yet been investigated. Studies conducted on this topic on male AMI survivors [31] or on initially healthy samples did not show a mediatory effect of depression for the association between SES and CHD-related outcome [5, 58].

Similarly to other studies investigating social support as a link between poor socioeconomic circumstances and recurrent events in CHD [10, 31], we did not find evidence for a mediating effect for this factor. Neither anxiety, nor vital exhaustion, hostility or the three other anger- related behaviours contributed to the explanation of the investigated association.

Differences in treatment as potential explanations for the social gradient in recurrent events

Differences in access to medical care among the income groups in our study are not likely to have contributed to differences in survival as the healthcare system in Sweden is universal. However, studies conducted in both countries with and without universal health care indicate that relative to their needs, cardiac patients with low socioeconomic position are less frequently offered revascularization procedures, adequate drug therapy and rehabilitation programs compared to their better situated counterparts [46, 59, 60]. Nevertheless, we found no differences in inclusion diagnose, medication or participation in cardiac rehabilitation among women with different SES, nor was there evidence that these factors contributed to the explanation of the relationship between income and recurrent events. These results are in agreement with those of a recent Swedish study which found no socioeconomic differences in cardiac revascularization procedures in women patients with CHD [61].

Limitations

Our study has several limitations which need to be considered when interpreting the results.

First, including only women from the larger Stockholm area who survived at least 6–8 weeks after hospitalization for a cardiac event limits the generalizibility of our findings to only urban dwelling women who are in a stable phase after a cardiac event.

Second, since only women were included in our study, no conclusions regarding male survivors of CHD can be drawn. However, since women have been underrepresented in cardiovascular research, studies conducted among women cardiac patients have a good potential to add to this area of research.

Third, recruitment in the study could have also resulted in selection bias as patients who are healthier and otherwise more advantaged are more likely to be willing to participate in rehabilitation programs than their worse situated counterparts [62, 63].

Fourth, due to the small number of recurrent events occurring during the follow-up the number of confounders we could adjust for in the base model was limited. However, we performed several alternative base models and found no indication for residual confounding. Similarly, the changes in point estimates after adding the potential mediators to the base model should be regarded as indicative. Comparing estimates before and after adjustment for the potential mediators is the most common method to evaluate intermediary effects. However, it has limitations. The actual percentage change does not quantify the actual mediation, rather just indicates it [64]. To decide whether

the changes in the point estimates after adjustment for potential mediators reflect causal relations and are not due to chance, our analyses need to be replicated in other samples of women with CHD.

Finally, using income as an indicator of socioeconomic position has the disadvantage of being subject to reverse causation, i.e. health status may affect levels of income. However, as already presented, we found no evidence for confounding from previous hospitalizations due to CHD. Similarly, as personal income and psychological factors were measured at the same point in time it is not possible to determine the causal relationship between these factors. However, Lynch and Kaplan [49] and Kristenson and colleagues [50] argue that by differences in exposure to environmental challenges and in protective resources, socioeconomic factors are more likely to structure the development and maintenance of social and psychological characteristics than the other way round. Despite its drawbacks, income is a useful measure of SES because it relates directly to the material conditions that may influence health [49]; it provides means in purchasing health care, better nutrition, housing, schooling and recreation [65]. It was suggested to be a better indicator of SES in adulthood and old age than education or occupational class because education is more reflective of adolescence and young adulthood SES, while occupational class can be applied only for working individuals [49]. Similarly, it may be argued that the socioeconomic position of the partner or household income may be a better indicator for women's SES than their personal income. However, we believe that in a country like Sweden, where the majority of women and almost the same proportion as men (80% of women and 86% of men) are gainfully employed [66], personal income is a good measure for women's social position. These advantages of the personal income as an indicator of SES may explain eventually why personal and not household income or education were predictive of recurrent events in this sample of women CHD patients.

Conclusions

In conclusion, our results indicate that low personal income is a risk factor for long term cardiovascular mortality or new AMI in women patients after a cardiac event and that smoking habits, depressive symptomatology and anger symptoms may contribute to the explanation of this relationship.

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