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Social relations in late adolescence and incident coronary heart disease: a 38-year follow-up of the Swedish 1969–1970 Conscription Cohort

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ABSTRACT

Objectives Increasing evidence suggests that low social support is associated with an elevated risk of coronary heart disease (CHD). Earlier studies in this field were conducted in predominantly middle-aged or older samples; thus, the associations reported previously may have been confounded by subclinical manifestations of the disease. We investigated whether social relationships in late adolescence, that is, well before symptoms of subclinical disease manifest, are associated with CHD during a 38-year follow-up.

Setting Sweden.

Participants Men born 1949–1951 and conscripted for military service in Sweden during 1969–1970 (n=49 321). At conscription, participants completed questionnaires about social relationships, lifestyle and health-related factors and underwent a medical examination.

Primary and secondary outcome measures CHD, acute myocardial infarction (AMI).

Results We found no relationship between having no confidant and frequency of confidential discussions with friends and the risk of CHD or AMI in the first 30 years of follow-up. However, after 30 years, men with no confidant at baseline had increased CHD and AMI risks relative to those having a confidant; the childhood socioeconomic status-adjusted HR and 95% CIs (CI) were 1.25 (1.10 to 1.41) and 1.27 (1.08 to 1.49), respectively. The frequency of confidential discussions with friends had an inverse U-shaped relationship with the outcomes after 30 years; the HR (95% CI) for 'sometimes' versus 'quite often' was 1.16 (1.04 to 1.29) for CHD and 1.16 (1.01 to 1.33) for AMI. These associations persisted after adjusting for mental ill-health, lifestyle factors and systolic blood pressure. A low number of friends in late adolescence was not related to an increased CHD or AMI risk.

Conclusions Not having a confidant in late adolescence was associated positively, while the frequency of confidential discussions with friends had an inverse U-shaped relationship with CHD and AMI after 30 years of follow-up, suggesting that these associations are not due to subclinical disease manifestations.

Strengths and limitations of this study

- ► Increasing evidence suggests that low social support is associated with an elevated risk of coronary heart disease (CHD); earlier studies in this field were conducted in predominantly middle-aged or older samples; thus, the associations reported previously may have been confounded by subclinical manifestations of the disease.
- ► This was the first study to analyse whether social relationships assessed in late adolescence, that is, well before symptoms of subclinical disease can manifest, are associated with CHD during a 38-year follow-up in a large sample of Swedish men and thus to eliminate confounding by subclinical disease.
- ► Since the sample included only men, it is not clear whether our findings are generalisable to women.
- ► Some misclassification of the exposure is possible as social relationships were assessed only at baseline and with single items instead of validated questionnaires.

BACKGROUND

Compelling evidence suggests that low social support is associated with an increased risk of cardiovascular mortality.[1–4] A large number of studies have consistently documented an association between low social support and poor prognosis in patients with coronary heart disease (CHD); knowledge regarding the link between poor social support and incident CHD is more limited and less consistent.[1 3 4] Two recent meta-analyses reported that low social support, defined in terms of structural support (ie, being socially isolated or having few social contacts), and functional support (ie, being lonely or appraising one's social support as inadequate) are associated with a 29%–50% increased risk of incident CHD.[5 6] The main hypothesised underlying mechanisms involve adverse changes in lifestyle and in stress-related endocrine, immune, metabolic and haemostatic activity that increase the risk of CHD.[1 4 7] In addition, social relationships may provide resources in terms of emotional, appraisal, instrumental

and/or material support which may buffer the adverse effects of acute and chronic stress on CHD.[1 2 4 7]

An alternative explanation for the link between social support and incident CHD is reverse causation or residual confounding from subclinical manifestations of the disease.[4 6] Atherosclerosis, the underlying pathophysiological mechanism involved in CHD, develops over several decades and may influence social functioning before clinical manifestations of the disease present.[8] Most studies in this area were conducted among predominantly middle-aged or elderly individuals and the longest follow-up was 21 years; thus, though participants were free of overt CHD at baseline, many were not free of subclinical CHD.[8]

To reduce the possibility of reverse causation, we investigated the association between social relations assessed in late adolescence—that is, when subclinical manifestations of CHD are unlikely to impact social functioning—and the risk of incident CHD during a 38-year follow-up.

METHODS

Study population and design

We studied men born in 1949–1951 who participated during 1969–1970 in the mandatory military conscription in Sweden (n=49 321). At conscription, men completed questionnaires about social, familial, lifestyle and health-related factors and participated in clinical examinations by a team of medical professionals and psychologists. The information obtained during these examinations was linked to several socioeconomic and health-related nationwide registers using the unique personal identification number assigned to all Swedish residents.[8 9]

Measures

Exposures

The questionnaire included three measures of social support. The first question inquired about the presence of a confidant: 'With whom do you talk most often about your personal problems? (a) mother, father or both, (b) sibling, (c) teacher/boss, (d) friend, (e) someone else, (f) no one'. We categorised study participants as having (categories a–e) versus not having a confidant (category f). A second question assessed the quality of social relationship with friends, that is, 'Do you have confidential discussions with your friends? (a) quite often, (b) sometimes and (c) never.' The third question inquired about the quantity of friends that is, 'How many do you consider to be your personal friends?'; we categorised the variable as having (a) more than five friends, (b) 3–5 friends and (c) 0–2 friends.

Outcomes

Participants were followed for CHD and acute myocardial infarction (AMI) with the Swedish Patient Register and Cause of Death Register. The Patient Register contains information on inpatient care in Sweden since 1964; its coverage increased gradually and became nationwide in 1987.[10] The Cause of Death Register was established in 1952 and contains information on date and cause of death for all Swedish residents.[11] Diseases and causes of death have been coded during the follow-up according to the 8th, 9th and 10th revisions of the International Classification of Diseases (ICD). We defined CHD using the ICD-8/9 codes 410–414 and the ICD-10 codes I20–I25 and AMI using the ICD-8/9 codes 410 and the ICD-10 codes I21 and I22. Follow-up ended on the date of the first event, death, emigration or 31 December 2008, whichever came first.

Covariates

Information on childhood socioeconomic status (SES) was defined based on the occupation of the head of the family (usually the father) and was retrieved from the National Population and Housing Census from 1960; the variable was classified as unskilled worker, skilled worker, farmer, low-level non-manual employee, medium-level non-manual employee or high-level non-manual employee.

During conscription, the men attended a structured clinical interview with a trained psychologist; men who reported any psychiatric symptoms were referred to a psychiatrist for further evaluation. Psychiatric diagnoses were recorded according to ICD-8; we extracted information on depression using codes 296 and 300.4 and on anxiety using codes 300.0. Trained nurses measured blood pressure on the first day of the clinical examination. In case the systolic blood pressure was >145 mm Hg or if the diastolic blood pressure was <50 mm Hg or >85 mm Hg, an additional measurement was performed the next day. Height and weight were measured and body mass index (BMI) was calculated. Cardiorespiratory fitness was assessed based on a bicycle ergometer submaximal exercise test performed after obtaining a normal resting ECG[12]; detailed descriptions of the assessment procedure, including its validity, have been published elsewhere.[13 14] Cardiorespiratory fitness was calculated by dividing the maximal work capacity during the exercise test with weight; the obtained value was transformed in stanine scores.[13]

Information on smoking and alcohol use and on perceived home environment during childhood was obtained from the questionnaire. Smoking was assessed with the item 'How much do you smoke per day?' with the answer possibilities (a) more than 20 cigarettes/day, (b) 11–20 cigarettes/day, (c) 6–10 cigarettes/day, (d) 1–5 cigarettes/day and (e) do not smoke. Study participants reported the frequency and quantity of their beer, wine and spirits consumption. The average weekly alcohol intake (in grams) was calculated by Andreasson *et al* based on information from the Swedish alcohol retail monopoly on the estimated alcohol content of the beverages available in Sweden during 1969–1970[15]. Four alcohol consumption categories were created: (a) abstainers or very low alcohol consumers (<1 g alcohol/week), (b) light (1–100 g alcohol/week), (c) moderate (101–250 g alcohol/week) and (d) heavy alcohol consumers (>250

g alcohol/week)[15]; this categorisation was based on health risks associated with similar alcohol consumption categories, as estimated by Hollstedt and Rydberg.[16] The perceived quality of the family environment during upbringing was assessed with the item 'Taken all together, how did you feel at home?' with the answer possibilities (a) excellent, (b) very well, (c) quite well and (d) did not feel well at all; we regarded the first three categories indicative of a positive, and the last category indicative of a negative home environment.

Statistical analyses

We analysed the prospective association between the three indicators of social relationships and CHD and AMI risk using Cox regression. The proportional hazards assumption was investigated using formal tests of interaction with each of the three exposures and (1) time and (2) the log of time. Since we observed some variation over time in the associations in case of the variables 'having a confidant' and 'frequency of confidential discussions with friends', that is, we found evidence for non-proportionality of hazards or a trend in this direction, in case of these variables we also performed analyses with the follow-up split at 30 years. This categorisation was an a priori decision to reflect (1) the age-related impairment in the allostatic load,[17] that is, that the cardiovascular system is likely to be more resilient to adverse psychosocial factors in the approximately first three decades of follow-up than after men enter middle age, and (2) the fact that the incidence of CHDs in men increases exponentially in the studied life period.[18] We present estimates for these variables both with the total follow-up and with the follow-up split at 30 years. In case of each of the three social support measures, we performed several models. Models 1 were unadjusted. Models 2 were adjusted for childhood SES, a potential confounder of the investigated associations. Models 3 were adjusted—in addition to childhood SES—for depression, anxiety, smoking, alcohol consumption, cardiorespiratory fitness, BMI and systolic blood pressure measured at conscription, factors that may be regarded both confounders (as they may influence social support and the risk of CHD) and mediators of the studied associations (ie, they may be on the causal pathway between social support and CHD).[1 4 7] We applied listwise deletion in case of missing information on covariates. Given the inverse association between the quality of the family environment and the risk of CHD[19 20] and that Sweden has several family-related welfare policies, we explored the hypothesis that a good family environment during upbringing may moderate the relationship between low peer support and the risk of CHD/AMI; we repeated our analyses concerning the association of (1) frequency of confidential discussions with friends and (2) the number of friends with the risk of CHD/AMI after stratifying for family environment during upbringing and conducted formal tests of interaction between our exposures and family environment. To screen for further possible effect modifications, we also conducted stratified analyses according to childhood SES (non-manual employee vs other), depression, anxiety, BMI (≤25 vs >25 kg/m^2), smoking (none vs any smoking), alcohol consumption (≤100 vs >100 g/week), cardiorespiratory fitness (stanine score <6 vs ≥6) and systolic blood pressure (<140 vs ≥140 mm Hg) and conducted formal tests of interaction between these variables and our exposures (as a routine procedure).

Analyses were conducted using SAS V.9.4.

Patient and public involvement

We have not involved study participants or the public in decisions concerning the research question, the design of the study, the study outcome, the method of recruitment, the conduct of the study or the writing of the manuscript. There are no specific plans to involve the public in the choice of the methods for the dissemination of the study results.

RESULTS

Men who developed CHD during the follow-up had a higher systolic blood pressure, higher BMI, a lower childhood SES, smoked more cigarettes, drank more alcohol, were more likely to have anxiety, no confidant and confidential discussions with friends 'sometimes' compared with those who did not experience CHD (table 1).

Not having a confidant was not associated with an increased risk of CHD or AMI in the first 30 years of follow-up. However, participants without a confidant had an increased CHD and AMI risk after 30 years of follow-up relative to those reporting not having a confidant; the childhood SES-adjusted HR and 95% CI were 1.25 (1.10 to 1.41) and 1.34 (1.15 to 1.57), respectively (table 2). The association between having no confidant and CHD and AMI after 30 years of follow-up was slightly attenuated but still present after adjusting for depression, anxiety, smoking, alcohol consumption, cardiorespiratory fitness, BMI and systolic blood pressure, factors that may potentially be on the causal pathway between social support and CHD/AMI (table 2).

There was no association between the frequency of confidential discussions with friends and CHD and AMI in the first 30 years of follow-up. The frequency of confidential discussions with friends had an inverse U-shaped relationship with CHD and AMI after 30 years of follow-up; the childhood-SES adjusted HR (95% CI) comparing the 'sometimes' to the 'quite often' category was 1.16 (1.04 to 1.29) in case of CHD and 1.26 (1.01 to 1.33) in case of AMI. The association between confidential discussions with friends and the risk of CHD or AMI after 30 years did not change after adjusting for factors in model 3 (table 3).

Having 3–5 friends tended to be associated with a modestly decreased risk of CHD during the 38 years of follow-up; the childhood SES-adjusted HR (95% CI) was 0.91 (0.84–1.00) relative to having >5 friends. The risk of CHD did not differ between those having 0–2 and >5

Table 1 Characteristics of the Swedish 1969–1970 Conscription Cohort according to coronary heart disease during the follow-up

		Coronary heart disease			
		Yes (n=2546)		No (n=46 775)	
Variable	Total N	Mean (SD)		Mean (SD)	
Continuous variables					
Systolic blood pressure (mm Hg)	48 900	128 (12)		126 (12)	
Body mass index (kg/m^2)	48 904	21.6 (3.0)		21.0 (3.2)	
		N	%	N	%
Categorical variables					
Childhood socioeconomic status*					
Non-manual (high or medium level)	10 890	432	17.0	10 458	22.4
Non-manual (low level)	4997	217	8.5	4780	10.2
Skilled worker	10 547	590	23.2	9957	21.3
Unskilled worker	16 351	984	38.6	15 367	32.8
Farmer	5419	252	9.9	5167	11.1
Others	1117	71	2.8	1046	2.2
Number of cigarettes per day					
0	20 103	695	27.8	19 408	42.2
1–5	5432	232	9.3	5200	11.3
6–10	10 057	596	23.9	9461	20.6
11–20	11 190	814	32.6	10 376	22.5
>20	1745	161	6.4	1584	3.4
Alcohol consumption, grams/week					
Abstainer or below 1 g	2781	123	5.0	2658	5.9
1–100 g	33 526	1656	67.7	31 870	70.6
101–250 g	9547	554	22.7	8993	19.9
>250 g	1724	113	4.6	1611	3.6
Cardiorespiratory fitness, stanine score					
1	34	0	0	34	0.1
2	212	15	0.6	197	0.4
3	2480	149	5.9	2331	5.0
4	7079	403	16.0	6676	14.4
5	11 532	638	25.3	10 894	23.4
6	9302	485	19.3	8817	19.0
7	5755	287	11.4	5468	11.8
8	4647	212	8.4	4435	9.5
9	7926	330	13.1	7596	16.4
Depression					
No	48 564	2507	98.5	46 057	98.5
Yes	757	39	1.5	718	1.5
Anxiety					
No	49 163	2531	99.4	46 632	99.7
Yes	158	15	0.6	143	0.3
Perceived quality of the family environment					

Continued

Table 1 Continued

		N	%	N	%
Excellent, very good or quite good	48342	2489	98.7	45853	98.9
Not good	541	34	1.3	507	1.1
Has a confidant					
Yes	40198	2034	81.8	38164	83.5
No	7983	452	18.2	7531	16.5
Frequency of confidential discussions with friends					
Quite often	17272	822	33.0	16450	35.9
Sometimes	28988	1572	63.2	27416	59.8
Never	2051	94	3.8	1957	4.3
Number of friends					
>5	29337	1559	62.7	27778	60.5
3–5	15374	743	29.9	14631	31.8
0–2	3715	185	7.4	3530	7.7

*Defined based on the father's occupation or the occupation of the other head of household if it was not the father.

friends. We observed no association between the number of friends and AMI (table 4).

The point estimates corresponding to the link between (1) the frequency of confidential discussions with friends and (2) the number of close friends and CHD/AMI were generally comparable or lower among those who reported to having had a positive home environment during their upbringing (n=48342) than among those who did not (n=541); however, due to the relatively small number of study participants in the latter group, our power to detect statistical differences in these associations was generally limited (data not shown). We found no evidence of effect

Table 2 HRs for coronary heart disease and acute myocardial infarction according to having a confidant

Outcome by follow-up period	Has a confidant	Events/person years	HR (95% CI) Model 1* (n=49321)	Model 2† (n=49321)	Model 3‡ (n=47061)
Coronary heart disease					
All follow-up	Yes	2034/1 452 401	1.00	1.00	1.00
	No	452/286 879	1.13 (1.02 to 1.25)	1.12 (1.01 to 1.24)	1.09 (0.98 to 1.21)
First 30 years of follow-up	Yes	802/1 166 915	1.00	1.00	1.00
	No	147/230 744	0.93 (0.78 to 1.11)	0.92 (0.77 to 1.09)	0.91 (0.76 to 1.09)
Last 8 years of follow-up	Yes	1232/285 385	1.00	1.00	1.00
	No	305/56 116	1.26 (1.11 to 1.43)	1.25 (1.10 to 1.41)	1.21 (1.06 to 1.37)
Acute myocardial infarction					
All follow-up	Yes	1252/1 457 339	1.00	1.00	1.00
	No	288/287 956	1.17 (1.03 to 1.33)	1.15 (1.02 to 1.31)	1.11 (0.97 to 1.27)
First 30 years of follow-up	Yes	494/1 168 165	1.00	1.00	1.00
	No	86/231 020	0.88 (0.70 to 1.11)	0.87 (0.69 to 1.09)	0.86 (0.68 to 1.09)
Last 8 years of follow-up	Yes	758/289 073	1.00	1.00	1.00
	No	202/56 916	1.36 (1.16 to 1.58)	1.34 (1.15 to 1.57)	1.27 (1.08 to 1.49)

*Model 1 is unadjusted.
†Model 2 is adjusted for childhood socioeconomic status.
‡Model 3 is adjusted for childhood socioeconomic status, depression, anxiety, smoking, alcohol consumption, cardiorespiratory fitness, body mass index and systolic blood pressure.

Table 3 HRs for coronary heart disease and acute myocardial infarction according to frequency of confidential discussions with friends

Outcome by follow-up period	Confidential discussions with friends	Events/person years	HR (95% CI) Model 1* (n=49 321)	Model 2† (n=49 321)	Model 3‡ (n=47 061)
Coronary heart disease					
All follow-up	Quite often	822/622 359	1.00	1.00	1.00
	Sometimes	1572/1 047 993	1.14 (1.04 to 1.24)	1.12 (1.03 to 1.22)	1.12 (1.03 to 1.22)
	Never	94/73 664	0.97 (0.78 to 1.20)	0.96 (0.77 to 1.19)	0.94 (0.75 to 1. 17)
First 30 years of follow-up	Quite often	324/500 161	1.00	1.00	1.00
	Sometimes	591/842 168	1.08 (0.94 to 1.24)	1.06 (0.93 to 1.22)	1.06 (0.92 to 1.22)
	Never	34/59 180	0.89 (0.62 to 1.26)	0.88 (0.62 to 1.25)	0.90 (0.63 to 1.29)
Last 8 years of follow-up	Quite often	498/122 155	1.00	1.00	1.00
	Sometimes	981/205 753	1.17 (1.05 to 1.30)	1.16 (1.04 to 1.29)	1.16 (1.04 to 1.30)
	Never	60/14 479	1.02 (0.78 to 1.33)	1.01 (0.77 to 1.32)	0.96 (0.72 to 1.27)
Acute myocardial infarction					
All follow-up	Quite often	519/624 279	1.00	1.00	1.00
	Sometimes	966/1 051 836	1.10 (0.99 to 1.23)	1.09 (0.98 to 1.21)	1.09 (0.98 to 1.22)
	Never	57/73 901	0.93 (0.71 to 1.22)	0.92 (0.70 to 1.21)	0.90 (0.67 to 1.20)
First 30 years of follow-up	Quite often	210/500 638	1.00	1.00	1.00
	Sometimes	354/843 138	1.00 (0.84 to 1.19)	0.99 (0.83 to 1.17)	0.99 (0.83 to 1.18)
	Never	17/59 255	0.69 (0.42 to 1.12)	0.68 (0.41 to 1.11)	0.69 (0.42 to 1.15)
Last 8 years of follow-up	Quite often	309/123 598	1.00	1.00	1.00
	Sometimes	612/208 625	1.17 (1.02 to 1.36)	1.16 (1.01 to 1.33)	1.16 (1.01 to 1.33)
	Never	40/14 641	1.09 (0.79 to 1.52)	1.09 (0.78 to 1.51)	1.03 (0.73 to 1.46)

*Model 1 is unadjusted.
†Model 2 is adjusted for childhood socioeconomic status.
‡Model 3 is adjusted for childhood socioeconomic status, depression, anxiety, smoking, alcohol consumption, cardiorespiratory fitness, body mass index and systolic blood pressure.

Table 4 HRs for coronary heart disease and acute myocardial infarction according to the number of friends

Number of friends	Events/person years	HR (95% CI) Model 1* (n=49 321)	Model 2† (n=49 321)	Model 3‡ (n=47 061)
Coronary heart disease, all follow-up				
>5	1559/1 059 477	1.00	1.00	1.00
3–5	743/555 932	0.90 (0.83 to 0.99)	0.91 (0.84 to 1.00)	0.92 (0.84 to 1.00)
0–2	185/132 908	0.95 (0.81 to 1.10)	0.94 (0.81 to 1.10)	0.96 (0.82 to 1.12)
Acute myocardial infarction, all follow-up				
>5	961/1 063 297	1.00	1.00	1.00
3–5	476/557 662	0.94 (0.84 to 1.05)	0.95 (0.85 to 1.06)	0.96 (0.85 to 1.07)
0–2	107/133 349	0.89 (0.73 to 1.09)	0.88 (0.72 to 1.08)	0.93 (0.76 to 1.14)

*Model 1 is unadjusted.
†Model 2 is adjusted for childhood socioeconomic status.
‡Model 3 is adjusted for childhood socioeconomic status, depression, anxiety, smoking, alcohol consumption, cardiorespiratory fitness, body mass index and systolic blood pressure.

modification by childhood SES, depression, anxiety, BMI, smoking, alcohol consumption, cardiorespiratory fitness and systolic blood pressure on the association between our three measures of social support and CHD or AMI (data not shown).

DISCUSSION

Not having a confidant in late adolescence was associated positively, while the frequency of confidential discussions with friends had an inverse U-shaped relationship with the risk of CHD and AMI after 30 years of follow-up. A low number of friends in late adolescence was not related to an increased CHD or AMI risk.

Most of the previous studies regarding the link between social relationships and incident CHD focused on structural measures of social support, often defined in terms of the number and the frequency of social contacts. These earlier studies have yielded mixed findings; several of them reported an increased risk of CHD in individuals with poor social networks,[21–26] while several others did not find such an association.[27–31] Considerably fewer studies focused on functional measures of social support—that is, the emotional, instrumental, appraisal and/or material support that one's network may provide[3]—in relation to CHD risk. Nevertheless, these studies were rather consistent in showing that low functional social support, often defined in terms of not having a confidant, is associated with an increased CHD risk.[21 22 24 31 32] The finding that our two measures of low functional support were associated with an increased risk of incident CHD and AMI (after 30 years of follow-up), while having a low number of friends was not, is thus in line with the earlier evidence suggesting that the support provided by one's environment may be more important for health than the size of the network.[31] The earlier investigations regarding the link between social support and CHD involved predominantly middle aged or older samples and follow-ups shorter than 21 years; thus, though the study participants were free of clinical CHD at baseline, many of them were probably not free from atherosclerosis.[8] Since subclinical manifestations of the disease may influence both social functioning and the risk of incident CHD, the previously reported associations were prone to residual confounding.[4 5 31] To our knowledge, our study is the first to investigate the association between social support assessed in young adulthood—that is, decades before the first subclinical manifestations of the disease present—and CHD risk and thus to virtually eliminate confounding by subclinical disease.[31 33]

The main potential explanations for the link between our measures of low functional support and the increased CHD risk after 30 years of follow-up involve negative changes in self-esteem, mental health, lifestyle and in stress-related physiological measures that increase the risk of CHD.[1 4–7] In addition, according to the buffering hypothesis, social support may reduce CHD risk by mitigating the adverse effects of stress.[1 4–7] We found that adjustment for several potential mediators of this association, for example, depression, anxiety, smoking, alcohol consumption, cardiorespiratory fitness, BMI and systolic blood pressure attenuated the association between our two measures of functional social support and the risk of CHD and AMI observed after 30 years of follow-up at most modestly. A potential explanation for these findings is that the suggested explanatory factors were measured only once, at a young age and at the same time as social support; exposure to low functional social support and to the investigated potential mediators needs to be chronic to induce physiological changes that increase the risk of CHD. The finding that the association between our functional support measures and CHD was not observed in the first 30 years, but only in the fourth decade of the follow-up is supportive of this hypothesis. It may also suggest that in the first three decades of the follow-up, the cardiovascular system is resilient to the adverse effects of low functional support, but as the chronic allostatic load increases and atherosclerosis becomes more advanced, poor social support may induce cardiac events in middle age.[17]

The lack of association between having a low number of friends and an increased risk of CHD could have several explanations. First, the fact that the great majority, that is, more than 98%, of the cohort members considered that they had felt well at home during their upbringing suggests that the low number of friends may have been compensated by family support; studies suggest that family experiences may play a more important role in health and well-being than peer experiences, both in adolescence[34 35] and in adulthood.[36] Furthermore, as this cohort lived during a period when the Swedish welfare state had increasingly provided several forms of instrumental and material support that in other settings are provided by family and friends (eg, related to childcare, elderly care, healthcare, support in case of several life crises, etc.) and when autonomy and self-realisation became increasingly important compared with social interdependence, we speculate that having a low number of close friends in late adolescence did not necessarily result in a disadvantage with regard to practical support that was important enough to increase the risk of CHD later in life.[31 37 38] In contrast, though a wide network of friends may increase the chances of receiving emotional support, the findings that study participants having 3–5 friends tended to have a slightly lower CHD risk than those with more than five friends might suggest that for some a large network of friends might also entail more conflict, negative social interactions and stress.[31]

Our study has several limitations. First, given that the sample only included men, it is not clear to what extent the associations between our three measures of support assessed in young adulthood and the risk of CHD up to the age of 58 observed in this study would be different in women. Men and women may differ with respect to the structural and functional aspects of social support, their perceived importance over the lifecourse and

the underlying mechanisms linking social support and health.[39] Overall, women have more intimate relationships, whereas men living during a period similar to that in which our cohort members lived may have had larger networks than women.[39] CHD tends to develop approximately 10 years later in women than in men.[40] The meta-analysis of Valtorta *et al*[6] investigated the association of loneliness and social isolation and CHD primarily in middle-aged and older samples and did not find gender differences in these associations. Second, as social relationships were assessed with single items instead of validated questionnaires, some exposure misclassification is possible; this is likely to be non-differential and, if anything, to result in an underestimation of the strength of the investigated association. Nevertheless, as research on the association between social support and health started only in the 1970s, at the time of our exposure ascertainment there were no validated questionnaires for social support.[41 42] Third, some additional misclassification of exposure might have arisen due to our lack of subsequent social support measurements. Certain trait-like characteristics related to social support, for example, social skills and need for social interactions, are likely to show some degree of stability over time, while others, for example, time available for and physical ability to participate in social events and foster social relationships, may be different in different phases of life. Fourth, it is possible that some of the CHDs experienced before 1987 by our cohort members were not included in our follow-up due to the incomplete coverage of the Patient Register prior to 1987.[10] However, given the low incidence of CHD in the cohort before 1987, it is unlikely that this potential misclassification substantially affected our results. Furthermore, we did not have data on the outcome after 2008; thus, the cohort was still young at the end of the follow-up and the generalisability of our findings to older populations is limited. Fifth, though our sample was large and our follow-up was very long, in some of the subcategories of our exposures the number of individuals was low and thus our statistical power may have been limited to detect modest effects.

In conclusion, we found that not having a confidant at the age of 18–20 was associated positively, while the frequency of confidential discussions with friends had an inverse U-shaped relationship with the risk of CHD in the last 8 years of our follow-up. Our findings may suggest that these associations are not due to confounding by subclinical symptoms of the disease and that improving functional social support in young age may have implications for the primary prevention of CHD. In contrast, having a low number of friends was not associated with an increased CHD risk.

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