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ORIGINAL ARTICLE

The emotional heart: prospective associations of anger, depression, and anxiety as risk factors for myocardial infarction in a 22-year follow-up of a working cohort of middle-aged men

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Abstract

Aim The study aim was to further clarify the relationship between psychological factors and myocardial infarction (MI) by simultaneously examining anger, depression, and anxiety as risk factors for incident MI in a healthy working sample.

Subject and method Baseline measurements of psychological variables were assessed through a self-reported questionnaire in a healthy cohort of 968 middle-aged men working at the Volvo Corporation. Single-item questions assessed depression and anxiety. Anger was assessed by the Trait Anger subscale of the Spielberger State-Trait Anger Expression Inventory. The endpoint was incident MI verified by national registers or medical records with follow up after 22 years. The main outcome was computed through logistic regression, reported as odds ratios. Additional correlation analyses were performed between psychological variables and coronary risk factors.

Results None of the psychological variables was significantly associated with the outcome; thus, the results failed to show an association between anger, depression, or anxiety and incident MI in this sample. There were some significant, but weak, correlations between psychological factors and negative health behaviors. Other components of traditional risk scoring instruments did not correlate with the psychological factors.

Conclusion A cohort restricted to middle-age healthy men limits applicability. However, our failure to replicate earlier results of population samples suggests a need for further research on associations between psychological factors and MI in healthy samples.

Keywords Myocardial infarction · Anger · Depression · Anxiety · Cohort study

Introduction

Major known risk factors for coronary heart disease (CHD) include high blood pressure, smoking, dyslipidemia, diabetes, and obesity (Blaha et al. 2021; Hubert et al. 1983; Pencina et al. 2019). However, there is a gap between risk estimation and those affected by disease (Brindle et al. 2003, 2006; Hense et al. 2003) and, as a result, other possible predictors for disease are investigated. In this context, the field of behavioral cardiology has been exploring connections between social environment, mood, and heart disease, pointing out several psychosocial stressors—for example, negative emotional states, social strain, and work stress—that are now considered probable additional risk factors for developing CHD (Das and O'Keefe 2006; Everson-Rose and Lewis 2005; Fishta and Backé 2015; Kivimäki et al. 2006; Rosengren et al. 2004; Rozanski et al. 2005). The proposed mechanisms behind them are an upregulation of the hypothalamic–pituitary–adrenal axis, a dysregulation of the autonomic nervous system, as well as altered behaviors coupled with psychosocial factors (Rozanski et al. 2005). The main goal of this study is to further examine three suggested psychological risk factors for CHD: depression, anxiety, and anger–hostility by studying them in a healthy working sample of middle aged men.

A growing body of research has focused on these three affective dispositions that seem to predict CHD (Everson-Rose and Lewis 2005). The most convincing evidence derives

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from prospective epidemiological cohort studies where depression, anxiety, and anger–hostility separately have been found to significantly associate with an increased risk for developing CHD in initially healthy populations and to similarly significantly associate with poorer prognosis in populations with pre-existing CHD (Celano et al. 2015, Chida and Steptoe 2009; Nicholson et al. 2006; Roest et al. 2010; Rozanski et al. 1999; Wu and Kling 2016). However, a number of individual studies have not shown any effect (Chida and Steptoe 2009; Janszky et al. 2010; Mykletun et al. 2007; Roest et al. 2010), and several meta-analyses have reached disparate conclusions regarding generalizability (Celano et al. 2015; Chida and Steptoe 2009, Nicholson et al. 2006; Roest et al. 2010; Wu and Kling 2016). However, other studies have linked depression, anxiety, and anger–hostility to several surrogate markers of CHD, among these coronary artery calcification (Santos et al. 2016) and carotid intima-media thickness (Matthews et al. 1998; Pollitt et al. 2005; Räikkönen et al. 2004; Santos et al. 2015) as signs of subclinical atherosclerosis, and decreased heart rate variability (Chalmers et al. 2014; Kemp et al. 2012; Sloan et al. 1994; Suls 2013), implying autonomic dysregulation of the heart. Notably, anger and hostility represent different psychological constructs but are largely intertwined and have often been interchangeably used due to a lack of standard definition and the degree of overlap in-between them (Chida and Steptoe 2009; Everson-Rose and Lewis 2005; Martin et al. 2000; Schulman and Stromberg 2007; Suls 2013). For simplicity, the term "anger" will be applied here onward.

Current evidence singles out depression as the best studied and most robust association (Celano et al. 2015; Nicholson et al. 2006; Rozanski et al. 1999), with a 2016 meta-analysis reporting a CHD risk augmentation of 22% in originally healthy populations (Wu and Kling 2016). For comparison, a traditional risk factor such as smoking entails an excess risk starting at 50% (Law and Wald 2003, U.S. Department of Health and Human Services 2010). Nevertheless, considering the substantial prevalence of depression, it thus appears as a contributory disease-driving element in a sizeable amount of CHD (Wu and Kling 2016). Some studies even report a dose-response relationship, where higher levels of depressive symptoms seem to couple with a higher risk gradient for CHD outcomes, both in healthy persons (Barefoot and Schroll 1996; Brunner et al. 2014), and in patients post-MI (Lesperance et al. 2002). Other studies have suggested anxiety to be an affective component in the development of CHD (Chida and Steptoe 2009; Shen et al. 2008), amounting to an increased risk of 26% for those initially healthy in the most recent meta-analysis (Roest et al. 2010). Finally, for anger, a meta-analysis demonstrated an associated 19% increase in risk of developing CHD in healthy populations. However, these findings were markedly mixed and the association for anger did not persist when controlling for covariates (Chida and Steptoe 2009).

Regrettably, literature provides little insight into whether these factors give rise to disease or if they simply act as risk markers for CHD, as common methodological problems in epidemiology limit assessment of causation. First, the temporal relationship between exposure and disease must be determined to confirm which condition preceded the other (Hill 1965). Considering that the pathogenesis of atherosclerosis is a decades-long process (Insull 2009), studies should ideally start with a relatively young and healthy sample and prospectively follow such a cohort for an extensive amount of time. The majority of studies to date have either had a cross-sectional design or been prospective cohort studies with follow-ups of less than a decade. Many cohort studies have focused on elderly people (Chida and Steptoe 2009; Roest et al. 2010; Wu and Kling 2016) in whom, even if healthy in relation to manifest CHD, silent atherosclerosis is likely to be widespread at study entry (Insull 2009). Second, one needs to consider possible confounding factors (von Elm et al. 2008), a practice that has often been deficient (Chida and Steptoe 2009; Wu and Kling 2016), even though potential confounders are manifold in community samples (Chida and Steptoe 2009; Roest et al. 2010; Wu and Kling 2016). In this respect, it would be advantageous to begin with a more homogeneous healthy population. For example, only a small number of studies have examined any of these affects in a healthy working sample of a narrow age-interval (Albert et al. 2005; Brunner et al. 2014; Nicholson et al. 2005; Whang et al. 2009), and these have shown few significant associations. One exception is the longitudinal cohort Nurse's Health Study, in which depression was linked to CHD outcomes with an excess risk of 37% (Whang et al. 2009). However, this relationship was strongest for fatal MI and sudden cardiac deaths, why authors speculated whether depression could have more of a pro-arrhythmic rather than an atherosclerotic effect.

Psychological constructs pose a problem when looking independently at these factors in relation to CHD. There is well-known diagnostic overlap between anxiety and depression (Krueger, 1999; Mineka et al. 1998; Suls and Bunde 2005), which can cause misclassification. Furthermore, anger, anxiety, and depression have the tendency to cluster in the same individual (Suls and Bunde 2005). Despite this, the majority of studies to date focus on either one or two affects and suggest independent effects on CHD when it is largely unknown how much of the reported risks should be attributed to either depression, anger, or anxiety in isolation (Suls and Bunde 2005). In actuality, when this has been specifically explored, the considerable overlap between these affects was such that their joint aspects emerged as a more important risk factor than any of their unique dimensions (Boyle et al. 2006; Kubzansky et al. 2006), presenting with effect sizes varying between 23% (Boyle et al. 2006) and 108% (Kubzansky et al. 2006). Consistent with this, a common disease-driving element, or at least a general emotional cardio-toxic pathway,

has been advanced as a more plausible model than one related to each single affect (Suls and Bunde 2005). Meanwhile, the evidence on the inter-relationship between these traits as risk factors for CHD, pointed out by Suls and Bunde (2005) as "a serious gap in literature," remains today particularly scarce.

This study emanates from the Renault-Volvo (R-V) Coeur Study, a prospective longitudinal study initiated in 1992 in collaboration between the two automotive companies. In brief, its initial main objective was to understand why France had significantly lower rates of CHD than many other Western countries when the French lifestyle itself contained some risk-aspects, what has been called a "French Paradox" (Simon et al. 1997; Tunstall-Pedoe 1988; Tunstall-Pedoe et al. 1994). This current study is a 22-year follow-up limited to the Swedish Volvo cohort from the R-V Coeur Study, using a prospective longitudinal approach. The general aim is to further investigate anger, depression, or anxiety as risk factors for developing coronary heart disease by attempting to overcome some methodological limitations of the past, with the specific aim to study these factors simultaneously in a homogeneous healthy sample.

Methods

This study emanates from the R-V Coeur Study, the original study design of which has already been elaborately reported in previous publications (Denarié et al. 2001; Dimberg et al. 2019; Kumlin et al. 1997, 2001; Rose et al. 1998, 2006; Simon et al. 1997).

Ethical considerations

The Research Ethics Committee of Gothenburg University approved the original study protocol and the consent to participate form on 11 February 1993 (Dnr. 23–93). This study was performed in line with the principles of the Declaration of Helsinki. For the present study, a cover letter reminded participants of their participation in the R-V Coeur Study, stated the aims of the follow up, and emphasized that continued participation was voluntary and confidential. A form of written consent asked participants to approve validation of reported health information by use of medical records. The study reporting was executed in line with STROBE guidelines for observational studies.

Participants

This study reports only on the Swedish cohort from the original R-V Coeur Study. In 1993, a source population of all male workers born between 1943 and 1948 at participating plants of the Volvo Corporation were randomly screened for participants (see Fig. 1). Using date of birth, men born in a segment of the month (15–31) were selected to render a list from which identified participants were successively approached and requested to enroll. The sample size was set to 1000 participants for each country, chosen to within 90% probability detect a 20% difference in CHD endpoints between the Swedish and the French cohorts after 10 years. Given that men in this age group have twice the risk of developing CHD compared to women, the sample was limited to men to ensure a sufficient amount of endpoint events at follow up. Of the potential Swedish participants, 144 declined participation for a variety of reasons reported elsewhere (Simon et al. 1997), and a total of 1000 men in the age segment 45–50 years were finally recruited. Among these, 23 participants were missing baseline data and 9 reported CHD at baseline and were therefore excluded from the present study, leaving a total of 968 for analysis.

Procedure and settings

At study baseline in 1993 (T1), all enrolled participants were invited to an appointment set during working hours. Participants were first administered a self-reported questionnaire on medical history, lifestyle factors, working conditions, and psychological characteristics. Next, they underwent an extensive nurse-administered health examination covering traditional CHD-related risk factors using blood pressure, resting electrocardiogram (ECG), anthropometrics, and a fasting venous blood sample. For the 2015 follow up (T2), a study form including five questions covering current or past heart disease or stroke was sent to participants' home addresses. History of MI was identified through the statement "I have suffered a heart attack" (yes/no). To verify self-reported events, an additional free-text statement read "I have been cared for in the following hospital or health care facility for the above-mentioned condition." As an incentive for participation, 20 scratch lottery tickets were randomly distributed among the respondents. A survey reminder was sent out in week 4. Data collection ended after 5 weeks. Self-reported events of MI were subsequently verified through medical records. Both self-reported events and events among non-responders were verified against SWEDEHEART (2017), a national registry for MIs collecting data from all Swedish hospitals caring for patients with acute CHD as far back as 1991. For non-responders, MIs were additionally mapped using the Swedish National Board on Health and Welfare's National Cause of Death Registry.

Variables

T1 exposure to psychological factors

All psychological factors were assessed by self-reports at T1. Due to cultural reasons, depression and anxiety were each evaluated through single-item questions, modified from

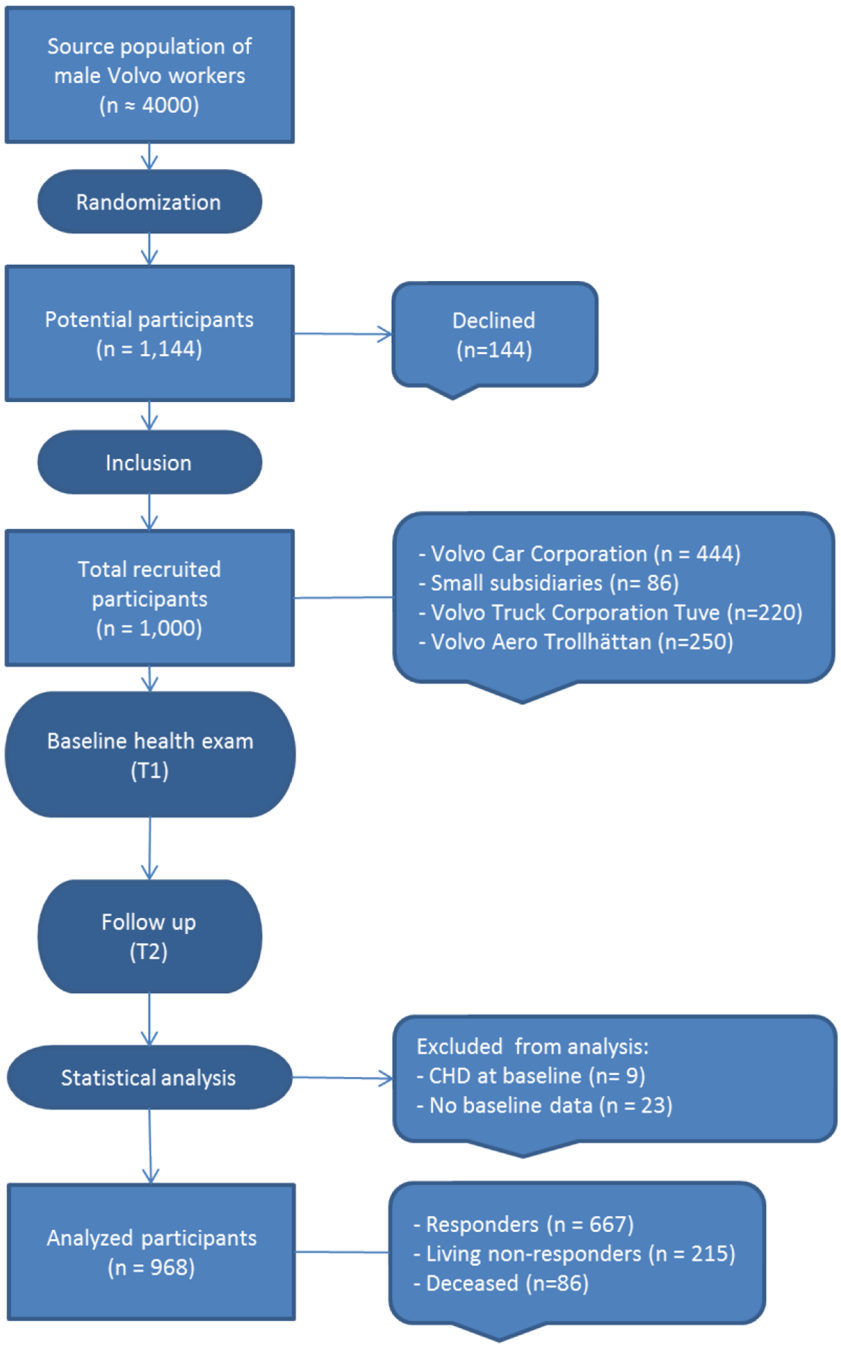

Fig. 1 Study flowchart presenting participant randomization, recruitment, and inclusion as well as loss of participants at each level of the study

validated and widely used psychological instruments previously applied in CHD research, the Beck Depression Inventory (Beck 1987; Beck et al., 1988; Nicholson et al. 2006) and the Spielberger State-Trait Anxiety Inventory (STAI) (Elwood et al. 2012; Gafarov et al. 2007; Spielberger et al. 1983). Depression was assessed with "How often do you feel unhappy, depressed, or sad?", and anxiety evaluated by "How often do you feel fidgety, nervous, or tense?", both answered on a 9-point Likert type scale ranging from "never" to "always." Separately, anger was assessed with the Trait Anger subscale of the State-Trait Anger Expression Inventory (STAXI) (Spielberger, 1988), which has previously linked anger to both CHD (Mendes de Leon et al. 1996; Williams et al. 2000) and its subclinical markers (Bleil et al. 2004; Matthews et al. 1998). Trait Anger is comprises 10 items evaluating disposition to experience anger and how this feeling manifests (Forgays et al. 1997) with items answered on a 4-point Likert scale ranging from "almost never" to "almost always" (Spielberger, 1988). For analysis, a sum anger score was generated, range 10–40.

T2 endpoint variable: myocardial infarction

The outcome was MI, defined as a verified incident event of MI that occurred between T1 and T2. The diagnostic definition of myocardial infarction was International Classification of Diseases ninth version (ICD-9) code 410, or International Classification of Diseases tenth version (ICD-10) code I21-I23 (World Health Organization 2017). For living responding participants, survey-reports of MI were verified through medical records or SWEDEHEART, or if not verifiable trough these sources, by a confirmative telephone call to the affected participant ($n = 5$). For non-responders, MIs were assessed directly through SWEDEHEART and the Swedish National Cause of Death Registry.

Covariates

The following variables were proposed as plausible covariates for found associations. As referenced, the details of these measures have already been carefully reported elsewhere.

Demographics Age at study entry, marital status, level of education, white or blue collar work (Dimberg et al. 2019).

Biological factors The Framingham risk index (FRI), calculated according to Anderson et al. (1991), obesity assessed through the body mass index (BMI) (kg/m^2) (Simon et al. 1997).

Health behaviors Smoking in pack–years (Kumlin et al. 2001; Rose et al. 1998; Simon et al. 1997), alcohol intake in gram/day (Simon et al. 1997), social support as a global social support network index (Kumlin et al. 2001), physical exercise as hours per week (Rose et al. 1998).

Statistical methods

Data were coded and compiled into an SPSS file providing the basis for all statistical analyses. Simple statistics using means for continuous variables and percentages for dichotomous variables were used to report baseline participant characteristics for all participants as well as for responders, living non-responders, and deceased participants at follow up. Binary logistic regression models were applied to explore the associations between the suspected psychological risk factors at baseline as independent continuous variables and incident MI as the outcome dependent dichotomous variable. The main output was reported as odds ratios with confidence intervals and p values within 95% probability limits. Significant associations were set to be controlled for possible confounding factors presented above using multivariate regression models. Correlation analyses were performed between the psychological variables and CHD covariates, reported as Pearson correlation coefficients (r) with their p values within 95% or greater probability limits.

Results

T1: baseline characteristics

Out of the 1000 men enrolled, 9 participants reported CHD at T1 (angina $n = 7$, heart disease $n = 3$) and an additional 23 participants lacked baseline information, which excluded them from the data set, thereby leaving a total of 968 men for subsequent analyses. Among these, the majority (61%) performed clerical (white-collar) work at Volvo and were either married or cohabitating (76%) at study start. A smaller proportion had a university level education (24%). Mean body mass index (BMI) was 25.6, 28% were active smokers, 9.4% had hypertension and the group had an average 9.1% 10-year risk of CHD as assessed by the Framingham risk index. The mean (± standard deviation) score for each of the psychological variables reported at T1 were 3.2 ± 1.4 for depression, 3.9 ± 1.6 for anxiety, and 16.4 ± 4.0 for anger. Full baseline characteristics are reported in Table 1.

Reports of incident MI

Over 22 years, there were 84 cases of incident MI, affecting 8.7% of the entire cohort. In this subgroup, further introduced in Table 1, a larger proportion performed manual work at Volvo (49%) at T1, and a lesser proportion had a university education (14%) compared to those who were free of MI at follow up. Furthermore, their mean BMI was 26.7, 44% were smokers, 16% had hypertension, and the average Framingham risk index 10-year risk was 13.6%. Additionally, among those who later had an MI, mean alcohol consumption per week was higher, and somewhat fewer men performed regular physical exercise compared to those who did not have an MI. Figure 2 illustrates the estimated cumulative incidence of incident myocardial infarction by age in the entire original cohort of 980 men with baseline data (i.e., including those with CHD at T1) observed during the 22-year follow-up.

Responding status at T2

Baseline characteristics were further subdivided according to responding status at T2, as seen in Table 2. Notably, in the group with deceased participants, prevalence of diabetes and hypertension was higher than for the other two, and 49% reported active smoking at T1. More men in this subgroup performed manual work at Volvo, fewer had a university education, and a lesser proportion was married or cohabitant at

Table 1 Baseline characteristics for entire cohort and participants with incident MI and free of incident MI at follow up

	All analyzed participants at T1 (n =968)	Incident MI at T2 (n =84)	No MI at T2 (n =885)
Age at T1, mean±SD, years	47.7±1.5	47.9±1.4	47.7±1.5
White-collared work, n (%)	591 (61)	42 (50)	549 (62)
Blue-collared work, n (%)	375 (39)	41 (49)	335 (38)
University education, n (%)	236 (24)	12 (14)	224 (25)
Married or cohabitant, n (%)	739 (76)	62 (74)	677 (77)
Social support index, mean±SD	11.3±6.3	10.3±4.6	11.4±6.5
Hypertension, n (%)	91 (9.4)	13 (16)	78 (8.8)
Body mass index, mean±SD, kg/m^2	25.6±3.3	26.7±3.2	25.5±3.3
Diabetes, n (%)	13 (1.3)	6 (7.1)	7 (0.8)
Framingham risk index score,mean±SD, 10-year risk percentage	9.1±5.8	13.6±7.7	8.6±5.4
Active smoker, n (%)	270 (28)	44 (52)	226 (26)
Smoking in pack years, mean±SD	11.4±13	19.4±15	10.7±12
Alcohol consumption, mean±SD, g/day	7.42±8.3	8.79±13.4	7.29±7.7
Exercise> 1 h per week, n (%)	664 (69)	54 (64)	610 (69)
Anxiety score, mean±SD, 1–9	3.9±1.6	4.0±1.7	3.9±1.6
Depression score, mean±SD, 1–9	3.2±1.4	3.2±1.5	3.2±1.4
Trait anger score, mean±SD, 10–40	16.4±4.0	16.8±4.6	16.4±3.9

SD standard deviation

Baseline characteristics for the entire cohort free of CHD at baseline (T1; n = 968) and with distribution of two subcategories at follow up, T2: participants with incident MI (n = 84) and participants free of incident MI (n = 885). Continuous variables are presented with means and standard deviations. Dichotomous variables are presented with counts and percentages

T1. Rates of incident MI was markedly higher (24%) in this subgroup at T2 than for the other two.

Main outcome at T2: psychological variables

For psychological variables, T1-reported anxiety, depression, and anger were similar between those men who later had an incident MI and those who remained free of MI throughout the study (see Table 1). Principally, as Table 3 shows, none of the psychological variables was significantly associated with the outcome of incident MI. Covariate effects were further explored through multivariate logistic regression analyses, but were not found to have any relevant influence on outcome. However, some correlations between the psychological variables and other CHD risk factors were observed and are reported in Table 4. Between the psychological variables there were significant but weak correlations between anger and anxiety ($r = 0.29$, $p < 0.001$) and anger and depression ($r = 0.34$, $p < 0.001$) and a moderate significant correlation between depression and anxiety ($r = 0.51$, $p < 0.001$). Blue collar workers ($r = -0.11$, $p < 0.001$) and those with a university level education ($r = -0.11$, $p = 0.001$) were slightly less prone to anxiety. Anxiety was furthermore slightly negatively associated with BMI ($r = -0.08$, $p = 0.02$). Those men scoring high on depression were marginally less likely to be married or cohabitating ($r = -0.08$, $p = 0.02$) and scored somewhat lower on social support ($r = -0.09$, $p < 0.01$). Both anger and depression had significant but weak correlations with smoking ($r = 0.10$, $p < 0.01$ and $r = 0.06$, $p < 0.05$, respectively). In turn, anger and anxiety were associated with having marginally higher alcohol consumption ($r = 0.09$, $p < 0.01$ and $r = 0.07$, $p = 0.03$, respectively). Other traditional CHD risk factors did not significantly correlate with any of the psychological variables.

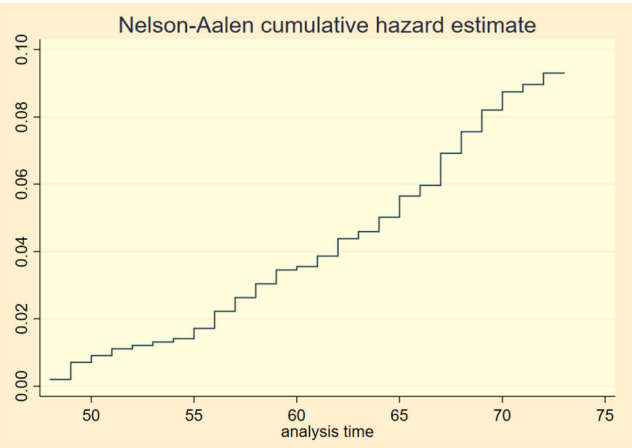

Fig. 2 Estimated cumulative incidence of myocardial infarction by age in the entire original cohort of 980 men during the observed time period (MIs = 89), calculated by a Nelson–Aalen cumulative hazard estimate

Table 2 Baseline (T1) characteristics for entire cohort as well as for subgroups according to responding status at follow up (T2)

	All analyzed participants at T1 (n=968)	Responders at T2 (*n* =667)	Living non-responders at T2 (*n* =215)	Deceased participants at T2 (*n* =86)
Age at T1, mean±SD, years	47.7±1.5	47.7±1.5	47.6±1.5	47.9±1.5
White-collared work, n (%)	591 (61)	440 (66)	116 (54)	35 (41)
Blue-collared work, n (%)	375 (39)	224 (34)	101 (47)	51 (59)
University education, n (%)	236 (24)	169 (25)	55 (26)	12 (14)
Married or cohabitant, n (%)	739 (76)	537 (81)	151 (70)	51 (59)
Social support index, mean±SD	11.3±6.3	11.7±7.0	10.2±4.6	11.4±4.8
Hypertension, n (%)	91 (9.4)	53 (7.9)	22 (10.2)	16 (19)
Body mass index, mean±SD, kg/m^2	25.6±3.3	25.3±3.2	26±3.3	26.8±4.3
Diabetes, n (%)	13 (1.3)	6 (0.9)	3 (1.4)	4 (4.7)
Framingham risk index score,mean±SD, 10-year risk percentage	9.1±5.8	8.4±5.1	9.8±6.5	12.8±8.1
Active smoker, n (%)	270 (28)	159 (24)	69 (32)	42 (49)
Alcohol consumption, mean±SD, g/day	7.42±8.3	7.49±8.2	7.56±8.4	6.53±8.8
Exercise> 1 h per week, n (%)	664 (69)	469 (70)	141 (66)	54 (63)
Anxiety score, mean±SD, 1–9	3.9±1.6	3.9±1.6	4.0±1.6	3.7±1.7
Depression score, mean±SD, 1–9	3.2±1.4	3.1±1.4	3.3±1.3	3.1±1.5
Trait anger score, mean±SD, 10–40	16.4±4.0	16.2±3.9	17±4.2	16.5±3.9
Incident myocardial infarction, n (%)	84 (8.7)	44 (6.6)	19 (8.8)	21 (24)

SD standard deviation

Discussion

Overall, our results could not support an association between depression, anxiety, or anger and an increased risk of MI for this initially healthy working sample of middle-age men. There were weak to moderate significant correlations between the psychological variables, and minor correlations between these factors and some of the behavioral covariates of CHD. No association was found between the psychological variables and the Framingham risk index score.

The scarce number of earlier studies on healthy homogenous cohorts exploring psychological factors in relation to CHD have shown disparate and inconclusive effects. In a large-scale cohort study conducted on Swedish men 18–20 years of age examined for military service with a follow up of 37 years, Janszky et al. (2010) found anxiety, but not depression, to independently predict MI. On the contrary, Ford et al. (1998) found clinical depression to be an independent risk factor for CHD in male medical students with a mean follow up of 37 years. The Whitehall II study, examining civil servants aged 35–55 years, did not find any significant association between one time-assessment of depressive symptoms (Brunner et al. 2014; Nicholson et al. 2005), or anxiety (Nicholson et al. 2005) and CHD death or non-fatal MI, but did however report a dose-relation when looking at six repeated measures of depressive symptoms during 24 years (Brunner et al. 2014), examining the cumulative effect of increasing levels of depressive symptomatology.

We speculate that a main reason for failure to replicate earlier findings for these psychological factors is that our cohort was unexpectedly healthy, both physically and psychologically, at study entry. Low scores of initial psychological distress and what appears as a low number of MIs simply could have limited the possibility for predicted associations to appear. In this setting our sample size may have been too small. Indeed, occupational samples are known for providing a "healthy worker effect" (Li and Sung 1999). In keeping with this, total mortality in the cohort was lower than expected (Dimberg et al. 2019), amounting to 4.8 per 1000 person years, whereas the official death rate for Swedish males aged 50 was 6.4 in 2015 (Statistics Sweden 2016). Furthermore, the

Table 3 Main outcome for each of the psychological variables

	OR	*95% CI*	*p* value
Trait anger, subscale	1.03	0.97–1.08	0.37
Depression, single item	1.01	0.86–1.19	0.91
Anxiety, single item	1.02	0.89–1.17	0.79

OR odds ratio, *CI* confidence interval

Each of the predicted psychological factors as a continuous variable was tested against the binary outcome of incident myocardial infarction using logistic regression models. The result is presented as odds ratios with their 95% confidence intervals and ***p*** values

Table 4 Correlations between psychological variables and covariates of coronary heart disease

	Anger		Depression		Anxiety	
	r	$p \leq$	*r*	$p \leq$	*r*	$p \leq$
Anger	1.00					
Depression	0.34	0.001	1.00			
Anxiety	0.29	0.001	0.51	0.001	1.00	
Blue-collar work	0.04	ns	0.01	ns	−0.11	0.001
University education	−0.02	ns	−0.04	ns	−0.11	0.001
Married or cohabitant	−0.01	ns	−0.08	0.05	0.02	ns
Social support index	0.01	ns	−0.09	0.01	−0.04	ns
Smoking	0.10	0.01	0.06	0.05	−0.02	ns
Total alcohol consumption, g/day	0.09	0.01	0.05	ns	0.07	0.05
Body mass index (BMI)	0.04	ns	−0.03	ns	−0.08	0.05
Less than 1 h ofexercise per week	−0.06	ns	0.01	ns	0.04	ns
Systolic blood pressure	−0.05	ns	−0.05	ns	−0.03	ns
Probable left ventricularhypertrophy according to Sokolow	0.00	ns	0.02	ns	−0.04	ns
Total cholesterol	0.04	ns	−0.02	ns	−0.03	ns
Heart rate	0.01	ns	0.01	ns	−0.01	ns
Fasting blood glucose	0.00	ns	0.05	ns	0.05	ns
Framingham risk index score	−0.01	ns	0.00	ns	−0.05	ns

r Pearson correlation coefficient, *ns* non-significant

Correlation analyses were performed between each of the predicted psychological variables and covariates of CHD, and are presented below with Pearson correlation coefficients (r) and their *p* values. Significant associations with p-values at the 0.05, 0.01 and 0.001 level are presented

average calculated 10-year risk for MI according to the FRI was 9.1% at T1, which amounts to a predicted number of 88 MIs after 10 years, suggesting that the number of incident events of MIs in this cohort over 22 years was considerably lower than expected. Additionally, the Volvo Corporation is well-known for having implemented a more flattened workplace organization in the early 1990s (Dimberg 1996). A more decentralized hierarchy increased distribution of responsibility and control among workers, which might have further increased the well-being in the cohort.

Moving to psychological measurements, the average scores of anxiety and depression appear to have been in the lower range. We used singular questions to define depression and anxiety in our baseline survey. While this is a significant limitation, the questions did correlate significantly with the psychological well-being index (PGWB) used in the 5-year follow-up presented in the study by Rose et al. (2006) where a higher score of anxiety and depression at baseline was significantly associated with poorer psychological well-being at follow-up. For depression, this study found correlations for total well-being ($r = 0.415$; $p < 0.0001$) as well as with the index' sub-scale for depression ($r = 0.310$; $p < 0.0001$). When it comes to anger, the Trait Anger subscale (mean cohort score 16.4 ± SD 4.0, range 10–40), has been previously applied in CHD research, e.g., in a study by Williams et al. (2000) where the average group Trait Anger score was 16.0 (± SD 3.7) with significant found effects in regard to CHD outcome, suggesting that the level of anger in itself was not the principal reason for the absence of predicted effect in our study.

An earlier study (Dimberg et al. 2019) conducted on the 22-year follow-up data for this current Volvo cohort similarly found traditional CHD risk factors (age, gender, tobacco smoking, blood pressure, cholesterol lipoproteins, body mass index, diabetes mellitus, left ventricular hypertrophy) to have a rather weak role in explaining incidence of MI in this cohort, with the Framingham risk score as the strongest predictor of MI, explaining 5.3% of the risk variation. Other significant risk factors were low education and manual labor. The authors proposed that one possible reason for the weak relationships might be that participants had changed their health behaviors over the follow up period, which also could be an applicable explanation here. Findings presented in another longitudinal Swedish study of 50-year-old men followed for 50 years showed distinctly decreased rates of cigarette smokers over time, also likely to apply to our cohort (Zhong et al. 2017).

In the light of a relevant debate on the failure to replicate studies in social psychology (Dominus 2017), another possible explanation could be that in actuality no associations between suggested psychological factors and MI exists, in which case earlier findings pointing to such effects could be confounded by covariate effects or poor study design. However, this seems unlikely considering convincing results from recent

meta-analyses on heterogeneous samples (Roest et al. 2010; Wu and Kling 2016). However, it may still be true that for healthier populations, such as the present cohort, there could for example be significant protective effects at play.

Correlation analyses provided some clues to elucidate the relationships between anger, depression, and anxiety in CHD research. As expected, there were significant correlations between the psychological variables indicating comorbidity and/or construct overlap and, although these were weak to moderate in size, this finding implies that research on these factors as risk factors for CHD indeed should, as suggested (Suls and Bunde 2005), simultaneously focus on all three factors in order to clarify to what extent they represent independent risk factors for CHD. Concerning associations with adverse health behaviors, these were inadequate in size to explain possible associations between psychological variables and CHD.

Limitations

Several study limitations exist. First, the cohort was limited to an occupational sample of Caucasian middle-aged men, limiting generalization of the findings to women, or other ethnic groups or ages, where results might be different.

The study was not initially dimensioned to evaluate psychological variables but rather to distinguish the predictive effects of traditional risk factors between the Swedish and French Coeur cohorts, seriously limiting the possibility to draw extensive conclusions from a null finding.

Future studies of this sort should ideally use validated and matching scales to measure all three psychological variables. If possible, it would additionally be meaningful for cohort studies to re-examine health behaviors and CHD risk factor prevalence at follow up with the purpose to explore to what extent changes in these variables influence between-study differences in outcome.

Conclusions

In this cohort study of a homogeneous healthy sample of 968 working middle-aged men, results failed to show that anxiety, anger, or depression were risk factors for myocardial infarction after 22-year follow-up. Sample restrictions limit generalizability to other types of cohorts; however, a plausible reason for failure to replicate earlier findings from community and patient samples could be that the study was not primarily designed to explore these connections and the cohort was in fact too healthy for relationships to appear. Consistent with the present study, the relatively few studies conducted on homogeneous healthy employed samples show inconclusive effects, suggesting a need for further research on these associations in these healthier samples. As expected there was some overlap between the psychological variables suggesting that anger, anxiety, and depression should indeed in the future be studied simultaneously as risk factors in order to elucidate their putative independent effects on CHD.

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Authors' contributions Dr. Lennart Dimberg, who at the time worked as an occupational physician at the Volvo Corporation, was involved in the design of and oversaw the initial Renault-Volvo Coeur Study. Dr. Dimberg also oversaw the collection of follow-up data at 22 years. Rebecca Vella executed the analysis and compiled the results of the current study as a master thesis student under the supervision of Dr. Dimberg and guidance of Professor Richard P Sloan. Professor Bo Eriksson oversaw and contributed to the statistical method and analysis. All authors contributed to the interpretation of data. The first draft of this manuscript was written by Rebecca Vella and was subsequently read and revised by all authors. Unfortunately, professor Bo Eriksson recently died and has therefore not read the final manuscript.

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Availability of data and material Data would be available upon request.

Code availability All codes are anonymized and real IDs will not be made available.

Declarations

Conflicts of interest/competing interests The authors have no conflicts of interest to declare that are relevant to the content of this article.

Ethics approval The Research Ethics Committee of Gothenburg University approved the original study protocol and the consent to participate form on 11 February 1993 (Dnr. 23–93). This study was performed in line with the principles of the Declaration of Helsinki. For the present study, a cover letter reminded participants of their participation in the R-V Coeur Study, stated the aims of the follow up, and emphasized that continued participation was voluntary and confidential. A form of written consent asked participants to approve validation of reported health information by use of medical records. The study reporting was executed in line with STROBE guidelines for observational studies.

Consent to participate All participants of the baseline study in 1993 were informed that only aggregated data would be published and agreed upon participation.

Consent for publication For the present study, the need to consent was waivered by the ethics committee since only register information was collected and all data are anonymized.

Informed consent Informed consent was obtained from all included study participants.

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