Chapter 6

Risk of coronary heart disease in men with poor emotion regulation: a prospective study

Marieke R. Potijk, Imre Janszky, Sijmen A. Reijneveld, and Daniel Falkstedt

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ABSTRACT

Objective Many psychosocial factors have been associated with coronary heart disease (CHD), such as hostility, anger, and depression. We tested the hypothesis that these factors may have fundamentals in emotion regulation capacities. Therefore, our aim was to determine whether poor emotion regulation directly predicted long-term risk of CHD.

Methods This nationwide study comprises a population of 46,393 men who were conscripted for compulsory military service in Sweden, in 1969/1970. Psychologists assessed emotion regulation at conscription (age 18-20 years), in a 20-30 minutes-long semi-structured interview about controlling emotions during childhood. CHD was defined as a first fatal or nonfatal event. We calculated hazard ratios (HRs) and 95% confidence intervals (CIs) for poor and adequate versus good emotion regulation.

Results After 38 years of follow-up (1971 until 2009), 2,456 incident cases of CHD had occurred. Poor emotion regulation increased the risk of CHD (HR 1.31, 95% CI 1.18-1.45), adjusting for childhood socioeconomic position, anxiety, depression, and parental history of CHD. Full adjustment for lifestyle-associated factors, e.g. smoking and BMI, attenuated the HR to 1.08 (95% CI 0.97 to 1.21). In stratified analyses, the fully adjusted HR for poor emotion regulation remained increased among men with a parental history of CHD (HR 1.49, 95% CI 1.11 to 2.01).

Conclusions In the overall study population, poor emotion regulation had no direct effect on CHD beyond lifestyle-associated factors. Specifically in men with a parental history of CHD, poor emotion regulation in adolescence directly predicted long-term CHD risk.
INTRODUCTION

Many psychosocial factors have been associated with coronary heart disease (CHD), but disease pathways remain uncertain. Research on the relation between psychosocial factors and CHD gained attention after Type A personality had been associated with a higher occurrence of CHD in 1959. Type A personality was characterized as ‘manifesting an intense, sustained drive for achievement and as being continually involved in competition and deadlines, both at work and in avocations’. Subsequently, also other psychosocial factors have been linked with CHD, such as hostility and anger, anxiety, depression, social isolation, and chronic psychological stress.

In order to explain associations between psychosocial factors and CHD, several biological and behavioral mechanisms have been proposed, from inflammatory processes to lack of exercise and other lifestyle-associated factors. Recently, some researchers have investigated whether poor emotion regulation could be associated with CHD. Emotion regulation is an important predictor of depressive symptoms and of distress over the life course. It is defined as the capacity to effectively use and control emotions in relationships and across a range of emotional events and has its foundations in early childhood, a period characterized by rapid cognitive and social-emotional development.

Emotion regulation may be a fundamental factor contributing to the pathophysiology of CHD, in that it may influence the response to emotional events and psychological stress, covering adverse features of mental disorders and personality characteristics. The relation between anger outbursts and CHD, for example, may have its fundaments in emotion regulation. To date, however, very few studies have addressed whether poor emotion regulation has a direct and prospective association with CHD, and the level of evidence is limited. Therefore, the aim of this study was to determine whether poor emotion regulation in late adolescence directly predicted subsequent long-term risk of CHD, independent of lifestyle-associated factors.
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METHODS

Study population
This study is based on data from the nationwide survey of Swedish males who were conscripted for compulsory military service in 1969/1970, at age 18 to 20 years. The procedures and variables of the nationwide survey have been described previously.\textsuperscript{19,20} Of all men conscripted, 5.5\% were born in 1949, 17.8\% in 1950, and 76.6\% in 1951. Of the Swedish male population only 2-3\% was exempted from conscription, in most cases due to severe handicaps or congenital disorders. All men underwent a two-day screening procedure, including extensive health examination at one of seven regional conscription centers in Sweden. Furthermore, the conscripts had to complete two extensive questionnaires: one about social and behavioral factors, and another about substance use. Ethical approval was granted by the Research Ethics Committee of the Karolinska Institutet, Stockholm. Due to the character of the database, it was impossible to trace persons and ask for informed consent. In an early stage, therefore, this was specifically mentioned in several applications to the Karolinska Institutet Ethical Review Board. The Board decided to waive the normal requirement for informed consent, since the database exists solely of anonymized record linkage data.

Assessment of emotion regulation
Trained psychologists were instructed to assess specific dimensions of a broader concept called 'mental functioning', including the emotional capacities 'social maturity', 'mental energy', and 'emotional control'.\textsuperscript{21} They did this by means of a semi-structured interview which lasted 20 to 30 minutes per conscript. Below we describe how emotional control was assessed. For detailed information on other dimensions of mental functioning we refer to a recent article on conscript data.\textsuperscript{21}

Emotional control was measured as the situational-dependent regulation of emotions, which is a fundamental aspect of the concept emotion regulation.\textsuperscript{22} The psychologists were instructed to ask the conscripts how they emotionally responded to important situational-dependent events (e.g. in the family, at school or at work) which they had experienced in childhood and adolescence. Based on the answers of the conscripts, the psychologists rated emotional control on a five-point scale. Ratings 1 (very poor) and 2 (poor) were given to conscripts who seemed to lack the ability to regulate emotions effectively, having difficulties in controlling nervousness and aggression. Rating 3 (average) was given to conscripts who had
adequate emotion regulation capacity, not having particularly negative or positive deviations. Ratings 4 (good) and 5 (very good) were given to individuals who appeared to respond calmly and purposefully, with good control of nervousness and aggression. The inter-rater reliability among interviewing psychologists was tested a few years after conscription, on the basis of 30 tape-recorded interviews scored by 30 psychologists, and was rated as 'very high' (r=0.86) for the overall assessment of mental functioning. Prevalence rates for ratings 1 to 5 were 6.1%, 23.9%, 40.3%, 24.1%, and 5.6%, respectively. For the analyses, we merged the five ratings into three categories of emotion regulation, a) poor, i.e. rating 1 to 2; b) adequate, i.e. rating 3; and c) good, i.e. rating 4 to 5.

Assessment of CHD
Participants were followed-up for a first event of fatal or nonfatal CHD from 1971 until 2009. At the end of follow-up participants were 58 to 60 years of age. CHD was defined as acute myocardial infarction, acute and sub-acute forms of ischemic heart disease, chronic ischemic heart disease, old myocardial infarction, angina pectoris, or asymptomatic ischemic heart disease (ICD-8 and ICD-9, 410-414 and ICD-10, I20-I25). Data was obtained by record linkage with the National Cause of Death Register and the National Hospital Discharge Register for 1971 to 2009. Both registers are administered by the Centre for Epidemiology at the National Board of Health and Welfare in Sweden. In Sweden, the registration of hospital diagnoses started in 1964, but did not cover the whole country until January 1, 1987. A few cases of CHD could be missed, but it would have little effect because CHD events at those young ages are very rare.

Covariates
The conscription examination provided information on cardiorespiratory fitness, resting systolic and diastolic blood pressures, body weight and height, and smoking habits. Cardiorespiratory fitness was rated as a score from 1 to 9 on a bicycle ergonometric test. Blood pressure measurements were made after 5-10 minutes of rest on the first day of the conscript examination. We used body weight and height to calculate the body mass index (BMI, kg/m²). In the questionnaire on substance use, participants were asked about their smoking habits (no smoking, 1-5 cigarettes, 6-10 cigarettes, 11-20 cigarettes, or > 20 cigarettes per day). Further, men who reported or presented any psychiatric symptoms in the psychologists’ interview at conscription were seen by a psychiatrist. Diagnoses of anxiety and
depression at age 18 were recorded according to the ICD-8. Information on years of education was obtained through linkage with the Longitudinal Database of Education, Income and Occupation, held by Statistics Sweden, for the year of 1990.

Data on parental history of CHD and childhood socioeconomic position was obtained via linking with parental records. The conscripts and parents were linked to each other through personal identification numbers by Statistics Sweden. Information on parental history of CHD mortality was obtained from the National Cause of Death Register for the years 1952-2003, administered by the Centre for Epidemiology at the National Board of Health and Welfare in Sweden. Parental history of CHD was defined as death known from CHD at age 65 years or younger of the father or mother. Information on childhood socioeconomic position, i.e. parental occupation at age 9 to 11 of participants, was obtained from the National Population and Housing Census of 1960 (response rate 99%). Childhood socioeconomic position was classified based on the occupation of the father or other head of household and has previously been linked to CHD. Because of missing values on covariates, the analyses were limited to 46,393 out of 49,321 participants.

Statistical analysis
First, we examined differences in characteristics of the conscripts with poor, adequate, and good emotion regulation. Chi-square tests and analysis of variance were used to compare proportions (categorical variables) and means (continuous variables), respectively, between the three groups. Next, we used Cox proportional-hazards regression models to examine the association between emotion regulation and incident CHD during follow-up. In the regression models BMI and blood pressure were entered as continuous variables. All other variables were entered as categorical variables. We computed hazard ratios (HRs) and 95% confidence intervals (CIs) for poor and adequate emotion regulation, with good emotion regulation serving as the reference category. We tested the proportionality of hazard using log-log curves, and there was no evidence against the proportionality assumption. In the first regression model, we adjusted for parental history of CHD, childhood socioeconomic position, anxiety, and depression. These factors were considered as confounders, as they may have caused emotion regulation deficits through genetic or other (common) pathophysiological mechanisms. We also conducted stratified analyses to assess whether the association of emotion
regulation and CHD was modified by the potential confounders of Model 1. In the second model, we further adjusted for lifestyle-associated factors, i.e. education, cardiorespiratory fitness, BMI, systolic blood pressure, diastolic blood pressure, and smoking, which were rather considered as mediators of the association between emotion regulation and CHD. We tested mediation effects of the lifestyle-associated factors by calculating the percent change in the regression coefficient relative to Model 1. Empiric 95% confidence intervals of the change in the coefficients were derived using 1000 bootstrapped datasets. Lastly, we restricted to hospital-verified diagnoses of acute myocardial infarctions as a sensitivity analysis to check if findings would be similar using an outcome with less chance of misclassification. Statistical analyses were performed in SPSS for Windows v. 22.0 (Chicago, Illinois).

RESULTS

Baseline characteristics by emotion regulation are shown in Table 1. Some characteristics of men with poor emotion regulation (30.0% of the men) were: smoking more than ten cigarettes a day, having a diagnosis of anxiety or depression, having lower cardiorespiratory fitness, and having fewer years of education. Parental history of CHD mortality was also more common in men with poor emotion regulation.

Of the 46 393 participants, 2456 (5.3%) experienced a first CHD event during follow-up. Table 2 shows the HRs and 95% CIs for CHD during follow-up according to emotion regulation in the total population. Men with poor and adequate emotion regulation, respectively, had 1.39 and 1.12 times greater hazard than men with good emotion regulation. The point estimates were fairly robust in multivariable models including the covariates anxiety, depression, childhood socioeconomic position, and parental history of CHD (Model 1). After further adjustment for education, cardiorespiratory fitness, BMI, smoking, systolic blood pressure, and diastolic blood pressure (Model 2), the HR for poor emotion regulation was 1.08 (95% CI 0.97 to 1.21). Additional tests for mediation showed that smoking was the factor that most attenuated the association between poor emotion regulation and CHD, followed by education and cardiorespiratory fitness (Supplementary Table).
Table 1 Baseline characteristics of study participants by emotion regulation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Poor (n=13915)</th>
<th>Adequate (n=18711)</th>
<th>Good (n=13767)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total % (n)</td>
<td>30.0</td>
<td>40.3</td>
<td>29.7</td>
<td></td>
</tr>
<tr>
<td><strong>Variables, % (n)</strong></td>
<td></td>
<td></td>
<td></td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Childhood socioeconomic position</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High non-manual</td>
<td>36.2 (5035)</td>
<td>33.6 (6296)</td>
<td>29.2 (4016)</td>
<td></td>
</tr>
<tr>
<td>Intermediate non-manual</td>
<td>22.0 (3066)</td>
<td>22.4 (4198)</td>
<td>19.7 (2715)</td>
<td></td>
</tr>
<tr>
<td>Low non-manual</td>
<td>9.5 (1325)</td>
<td>9.8 (1836)</td>
<td>11.6 (1598)</td>
<td></td>
</tr>
<tr>
<td>Skilled manual</td>
<td>14.9 (2073)</td>
<td>16.0 (2995)</td>
<td>20.3 (2793)</td>
<td></td>
</tr>
<tr>
<td>Self-employed</td>
<td>4.5 (623)</td>
<td>4.5 (837)</td>
<td>6.9 (950)</td>
<td></td>
</tr>
<tr>
<td>Unskilled manual</td>
<td>10.3 (1438)</td>
<td>11.7 (2180)</td>
<td>11.1 (1531)</td>
<td></td>
</tr>
<tr>
<td>Reporting no occupation</td>
<td>2.6 (355)</td>
<td>2.0 (369)</td>
<td>1.2 (164)</td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
<td>&lt;.001</td>
</tr>
<tr>
<td>9 years or less</td>
<td>31.3 (4361)</td>
<td>26.6 (4971)</td>
<td>17.4 (2393)</td>
<td></td>
</tr>
<tr>
<td>10-11 years</td>
<td>29.7 (4139)</td>
<td>29.6 (5537)</td>
<td>24.7 (3402)</td>
<td></td>
</tr>
<tr>
<td>12-13 years</td>
<td>13.4 (1860)</td>
<td>16.7 (3128)</td>
<td>18.8 (2589)</td>
<td></td>
</tr>
<tr>
<td>14 years</td>
<td>9.5 (1319)</td>
<td>10.9 (2047)</td>
<td>15.0 (2065)</td>
<td></td>
</tr>
<tr>
<td>15 years or more</td>
<td>11.9 (1657)</td>
<td>13.3 (2484)</td>
<td>21.1 (2899)</td>
<td></td>
</tr>
<tr>
<td>Smoking, cigarettes per day</td>
<td></td>
<td></td>
<td></td>
<td>&lt;.001</td>
</tr>
<tr>
<td>No smoking</td>
<td>34.4 (4793)</td>
<td>41.7 (7806)</td>
<td>48.7 (6705)</td>
<td></td>
</tr>
<tr>
<td>1-5</td>
<td>9.6 (1341)</td>
<td>11.4 (2138)</td>
<td>12.6 (1732)</td>
<td></td>
</tr>
<tr>
<td>6-10</td>
<td>19.5 (2711)</td>
<td>21.9 (4101)</td>
<td>20.6 (2833)</td>
<td></td>
</tr>
<tr>
<td>11-20</td>
<td>29.2 (4065)</td>
<td>22.6 (4230)</td>
<td>17.0 (2341)</td>
<td></td>
</tr>
<tr>
<td>&gt; 20</td>
<td>7.2 (1005)</td>
<td>2.3 (436)</td>
<td>1.1 (156)</td>
<td></td>
</tr>
<tr>
<td>Anxiety disorder</td>
<td>1.0 (145)</td>
<td>0.0 (5)</td>
<td>0.0 (1)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Depressive disorder</td>
<td>4.1 (567)</td>
<td>0.1 (15)</td>
<td>0.0 (2)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Parental history of CHD*</td>
<td>7.8 (1086)</td>
<td>7.0 (1309)</td>
<td>6.5 (890)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Variables, mean (SD)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiorespiratory fitness score</td>
<td>5.6 (1.8)</td>
<td>6.1 (1.8)</td>
<td>6.6 (1.8)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>20.9 (3.5)</td>
<td>20.9 (2.8)</td>
<td>21.2 (3.0)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Systolic blood pressure, mmHg</td>
<td>125.9 (12.0)</td>
<td>126.3 (11.8)</td>
<td>126.0 (11.7)</td>
<td>.002</td>
</tr>
<tr>
<td>Diastolic blood pressure, mmHg</td>
<td>73.1 (9.4)</td>
<td>72.9 (9.3)</td>
<td>72.5 (9.7)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Parental history of fatal CHD before 65 years of age

Table 2 Hazard ratios (95% confidence intervals) for CHD during follow-up according to emotion regulation

<table>
<thead>
<tr>
<th>Emotion regulation</th>
<th>Cases N (%)</th>
<th>Incidence rate</th>
<th>Unadjusted</th>
<th>Model 1</th>
<th>Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poor</td>
<td>859 (6.2)</td>
<td>186</td>
<td>1.39 (1.25 - 1.54)</td>
<td>1.31 (1.18 - 1.45)</td>
<td>1.08 (0.97 - 1.21)</td>
</tr>
<tr>
<td>Adequate</td>
<td>959 (5.1)</td>
<td>138</td>
<td>1.12 (1.01 - 1.24)</td>
<td>1.08 (0.98 - 1.20)</td>
<td>0.98 (0.89 - 1.09)</td>
</tr>
<tr>
<td>Good</td>
<td>638 (4.6)</td>
<td>124</td>
<td>Reference</td>
<td>Reference</td>
<td>Reference</td>
</tr>
</tbody>
</table>

*Incidence rate expressed as number of events per 100,000 person-years.

Model 1: adjustment for anxiety, depression, childhood socioeconomic position, and parental history of CHD.

Model 2: further adjustment for education, cardiorespiratory fitness, BMI, smoking, systolic blood pressure, and diastolic blood pressure.
Stratified analyses for explanatory variables indicated effect modification by parental history of CHD: $P$ values for interaction between poor emotion regulation and parental history of CHD were 0.043 (Model 1) and 0.037 (Model 2). Stratification by other variables did not indicate effect modification. Even after full adjustment for cardiovascular risk factors, the association between poor emotion regulation and CHD was more pronounced in participants with a parental history of CHD (HR 1.49, 95% CI 1.11-2.01) than in those without (HR 1.02, 95% CI 0.90-1.14), as shown in Model 2 of Table 3. This difference is illustrated in Figure 1, showing the cumulative rates of CHD stratified by parental history of CHD. The interaction between poor emotion regulation and parental history of CHD (i.e. CHD mortality before age 65) was not explained by the loss of a parent at a young age. After we had stratified by ‘all-cause parental death apart from CHD’ instead of by ‘early parental death due to CHD’, HRs were 1.33 (95% CI 1.00-1.76) and 1.04 (95% CI 0.78-1.39) for Model 1 and 2, respectively.

Sensitivity analyses, i.e. restriction to hospital-registered diagnoses of acute myocardial infarction, showed similar or higher HRs as in the main analyses. E.g. for Model 1, the HRs for poor compared to good emotion regulation were 1.79 (95% CI 1.23-2.59) and 1.26 (95% CI 1.08-1.47) in men with and without parental history of CHD, respectively.

Table 3 Hazard ratios (95% confidence intervals) for CHD during follow-up according to emotion regulation, stratified by parental history of CHD

<table>
<thead>
<tr>
<th>Emotion regulation</th>
<th>Positive parental history ($N=3285$)</th>
<th>Negative parental history ($N=43108$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 1$^a$</td>
<td>Model 2$^b$</td>
</tr>
<tr>
<td>Poor</td>
<td>1.68 (1.27-2.22)$^c$</td>
<td>1.49 (1.11-2.01)$^c$</td>
</tr>
<tr>
<td>Adequate</td>
<td>1.23 (0.93-1.63)</td>
<td>1.17 (0.88-1.56)</td>
</tr>
<tr>
<td>Good</td>
<td>Reference</td>
<td>Reference</td>
</tr>
</tbody>
</table>

$^a$ Model 1: adjustment for anxiety, depression, and childhood socioeconomic position.
$^b$ Model 2: further adjustment for education, cardiorespiratory fitness, BMI, smoking, systolic blood pressure, and diastolic blood pressure.
$^c$ $P$ values for interaction between poor emotion regulation and parental history of CHD were 0.043 (Model 1) and 0.037 (Model 2).
Figure 1 Cumulative incidence of CHD by level of emotion regulation capacity, in men with negative and positive parental history of CHD before age 65. The error bars depict 95% confidence intervals.

DISCUSSION

We found that poor emotion regulation in late adolescence was associated with an increased risk of CHD. This was more pronounced in participants with a parental history of CHD than in those without. In the total study population, adjustment for lifestyle-associated risk factors such as smoking, BMI, education, and blood pressure, largely attenuated the association between poor emotion regulation and CHD.

Our finding that poor emotion regulation was associated with an increased risk of CHD supports the evidence from some small prior studies, although these studies used alternative measures for emotion regulation. Two prospective cohort studies provided evidence for an inverse association between emotion regulation
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and CHD, and cardiovascular risk factors of CHD. In the somewhat larger study, including 1122 men, higher levels of self-reported emotion regulation were associated with a decreased risk of CHD. Results of the other study, among 415 adults, showed that higher levels of childhood attention regulation – measured as a specific form of self-regulation – increased the probability of having a favorable cardiovascular risk. Separate later analyses on data of this study showed that emotion regulation deficits were not associated with higher cardiovascular disease risk. Our study adds to the current evidence, as it provides greater statistical power, a more objective assessment of emotion regulation by psychologists, and little probability of reverse causation.

Poor emotion regulation may lead to CHD via two main pathways. First, having difficulties in regulating emotions may lead to health-compromising behaviors. Our findings support this explanation because lifestyle-associated factors seemed to explain much of the link between poor emotion regulation and CHD in the majority of the men. In the causal pathway, we consider lifestyle-associated factors as mediators rather than confounders because they are more likely to be consequences of poor emotion regulation than causes of it, e.g. in the case of smoking. Also in the association between depression and CHD, lifestyle-associated factors seem to have a mediating role. Treating lifestyle-associated risk factors as confounders may even lead to underestimation of the potential effect of psychosocial factors.

Second, chronic psychological stress may explain the association between poor emotion regulation and CHD, as emotion regulation may (partly) reflect one’s ability to respond to psychological stress. Low ability to control stressful situations may result in chronically high stress levels, which leads to activation of the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system, ultimately resulting in higher blood pressure and acceleration of the atherosclerotic process. Psychological stress due to poor emotion regulation can also be considered as a dysfunctional feature of certain personality traits and mental disorders. Several personality traits, such as neuroticism, adjustment disorders, and hostility and anger, have been associated with unhealthy responses to daily psychological stressors and higher cardiovascular risk. Chronic psychological stress itself often goes along with depression and anxiety, which have independently been associated with cardiovascular diseases. Genetic factors may be involved here: CHD patients who are carrier of the s allele of 5-HTTLPR are more vulnerable to depression and perceived stress. Our findings may be interpreted
as supporting the psychological stress-pathway, because emotion regulation was
inversely associated with CHD, independent of lifestyle-associated risk factors, in
men with a parental history of CHD.

Our finding of a stronger association between poor emotion regulation and
CHD in men with a parental history of CHD implies that familial factors might
be involved. However, this was an unexpected finding, and we can only speculate
about its background. One possibility is that poor emotion regulation increases
the impact of psychological stress in families with high susceptibility to stress.
Indeed, evidence from prior studies indicates that the combination of specific
genes with psychological stress increases the risk of CHD.\textsuperscript{1,38,39} Furthermore, twin
studies have shown a significant genetic contribution to individual differences in
cardiocvascular reactivity to psychological stress.\textsuperscript{40} Further research is needed to
identify and specify the link of familial CHD risk with poor emotion regulation. In
this context, it is also interesting which mediation pathways are important in the
association between poor emotion regulation and CHD. In this study, the pathway
between emotion regulation and development of CHD appeared to be mediated
by smoking, education, and cardiorespiratory fitness but not by BMI and blood
pressure. Therefore, future studies should focus on disentangling the specific
pathways that link poor emotion regulation with a higher occurrence of CHD.

We believe that this study has major strengths. First, the large cohort provided
an ample statistical power to investigate the research question. Second, there was
little risk of selection bias since very few men were exempted from conscription
examinations, and there was no self-selection because military service was
mandatory in Sweden around 1969/1970. In addition, CHD events were followed
prospectively via registers, providing high quality of follow-up and no loss to
follow-up, except for the rare case of emigrating from Sweden.\textsuperscript{41} Conscript data
was linked with data from National registers on hospitalization and cause of death.
Lastly, a unique characteristic of this study is that psychologists assessed emotion
regulation capacity at age 18 to 20, which greatly reduced the probability of
reverse causation. The latter was a weakness of previous studies in which emotion
regulation had been measured at middle age or even older ages.

Our study also has limitations. Study participants were exclusively Swedish
men, which limits generalizability. The findings also do not necessarily apply
to CHD events at older ages because participants were about age 60 at the end
of follow-up. Furthermore, the effect of emotion regulation as measured at
conscription might have been diluted over the long follow-up time, although
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it is unknown yet how emotion regulation relates to (healthy) ageing.22 Some lifestyle-associated factors, i.e. BMI, cardiorespiratory fitness, smoking, and blood pressure, were also likely to change during follow-up. However, changes in these risk factors later on are consequences rather than causes of our exposure, which may have led to an underestimation of the real effects. We also consider it a limitation that the assessment of specific dimensions of the conscripts' mental functioning took place in 1969/1970, which limited us in interpreting what exactly the psychologists measured in relation to the current theory on emotion regulation. In this study, the term emotion regulation was used as a measure for the conscripts’ emotional responses on important situational-dependent events in childhood and adolescence. This description fits with core features of emotion regulation, but it may also fit to related constructs, such as coping ability and mood regulation. However, it is yet uncertain how these constructs are related to each other.22 Finally, we only have information on reliability tests for the overall assessment of 'mental functioning' – which was found to be 'very high' – and not for the specific dimension of emotional control.

Summarizing, in the overall study population poor emotion regulation had no direct effect on CHD beyond lifestyle-associated factors. However, the same thing cannot be concluded for subjects with a parental history of early CHD, as poor emotion regulation had a moderately strong association with CHD in this high-risk group, also after adjustments. If other studies would confirm these findings, they may provide new possibilities for primary and secondary prevention of CHD in high-risk groups. Many individuals are exposed to difficulties in emotion regulation (30% in this study), and a variety of psychological interventions, including emotion regulation interventions and coping with stress exposure, have shown to improve health in clinical populations.42-45 However, intervention studies should also include non-clinical, high-risk populations because very little is known about the effectiveness of psychological interventions in healthy, but at risk populations.

Acknowledgments
We would like to thank Professor Tomas Hemmingsson at the Centre for Social Research on Alcohol and Drugs, Stockholm University, Sweden, for making the database available to us.
Chapter 6

References


Supplementary Table
Mediation effects of lifestyle-associated factors on the association between emotion regulation and CHD

<table>
<thead>
<tr>
<th>Lifestyle factor</th>
<th>Overall ((N=46393))</th>
<th>Positive parental history ((N=3285))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(\beta)</td>
<td>% (\beta) change (95% CI)(^b)</td>
</tr>
<tr>
<td>Reference(^a)</td>
<td>0.27</td>
<td>Ref</td>
</tr>
<tr>
<td>+Education</td>
<td>0.19</td>
<td>-29.6 (-22.9, -53.3)</td>
</tr>
<tr>
<td>+Cardiorespiratory fitness</td>
<td>0.23</td>
<td>-14.8 (-12.6, -27.5)</td>
</tr>
<tr>
<td>+Body mass index</td>
<td>0.28</td>
<td>+3.7 (+1.1, +7.8)</td>
</tr>
<tr>
<td>+Smoking</td>
<td>0.14</td>
<td>-48.1 (-35.0, -80.2)</td>
</tr>
<tr>
<td>+Systolic blood pressure</td>
<td>0.27</td>
<td>=</td>
</tr>
<tr>
<td>+Diastolic blood pressure</td>
<td>0.27</td>
<td>=</td>
</tr>
</tbody>
</table>

\(^a\) Model 1: adjustment for anxiety, depression, childhood socioeconomic position (and parental history of CHD in overall study population).

\(^b\) Regression coefficients (\(\beta\)) were derived from Cox regression analyses. Empiric 95% confidence intervals of the change in the coefficients were derived using 1000 bootstrapped datasets.