Published in final edited form as:
Psychosom Med. 2010 February ; 72(2): 148–155. doi:10.1097/PSY.0b013e3181c925cb.

Quality of Parental Emotional Care and Calculated Risk for Coronary Heart Disease

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Abstract

Objective—Little is understood about the role of parental emotional care in contributing to the risk for coronary heart disease (CHD). We evaluated associations between perceived quality of parental emotional care and calculated 10-year risk for CHD.

Methods—The study sample was composed of 267 participants from the New England Family Study. Quality of parental emotional care was measured using a validated short version of the Parental Bonding Instrument (PBI) as the average care scores for both parents (range: 0-12), with higher scores indicating greater care. Ten-year CHD risk was calculated using the validated Framingham Risk Algorithm that incorporates the following prevalent CHD risk factors: age, sex, diabetes, smoking, total cholesterol, HDL cholesterol, and blood pressure. Multiple linear regression assessed associations of PBI with calculated CHD risk after adjusting for childhood socioeconomic status, depressive symptomatology, educational attainment and body mass index.

Results—Among females, a one-unit increase in the parental emotional care score resulted in a 4.6% (p = .004) decrease in the 10-year CHD risk score, after adjusting for covariates. There was no association between parental emotional care score and calculated CHD risk score in males (p = .22).

Conclusion—Quality of parental emotional care was inversely associated with calculated 10-year CHD risk in females, and not males. While the gender differences need further investigation, and these findings require replication, these results suggest that the early childhood psychosocial environment may confer risk for CHD in adulthood.

Keywords

coronary heart disease; early childhood environment; parental bonding instrument

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Disclosures: None of the authors has any conflict of interest concerning the topic or contents of this article.

Introduction

Coronary heart disease (CHD) remains a leading cause of death world-wide (1). While a considerable amount of research has concentrated on adult risk factors for CHD, newer studies are increasingly focusing on the developmental origins of CHD. With help from Barker's fetal origins hypothesis which linked birth weight to risk of future chronic disease (2), it is increasingly realized that precursors for coronary heart disease may manifest early in life. For example, fibrous plaque formations were detected in coronary arteries and aorta of adolescents and young adults (3). Blood pressure, cholesterol, and obesity measured in adolescence are positively associated with levels measured in later life (4).

One underexplored early-life factor with respect to its potential influence on CHD is the quality of parental emotional care. Quality of parental care (often assessed using the Parental Bonding Instrument (PBI) which measures amounts of parental affection and authoritarianism towards offspring) has been consistently inversely associated with depression in offspring (5,6). Depression itself is a risk factor for CHD (7). Furthermore, quality of parental care may influence other risk factors for CHD, including educational attainment (8), smoking (9) and obesity (10). There is evidence to suggest that low intelligence (11), socioeconomic position (12), and exposure to childhood maltreatment (13) are risk factors for chronic diseases including CHD, and it is plausible, although minimally tested, that early childhood enrichment through high quality parental care may influence risk for chronic diseases such as CHD. Consequently, the objectives of this study were to investigate the associations of parental emotional care with 10-year calculated risk for coronary heart disease using the Framingham Risk Algorithm (which includes age, sex, smoking, blood pressure, HDL cholesterol, and diabetes prevalence). We hypothesized that the quality of parental emotional care is inversely associated with 10-year calculated risk for CHD. Figure 1 elucidates the anticipated mechanisms by which quality of parental emotional care may influence risk for CHD.

Methods

Study population

Please refer to Figure 2 for a flowchart of the study sample. Study participants were drawn from the New England Family Study, which comprised 17,921 offspring of pregnant women who had participated in the National Collaborative Perinatal Project at the Providence, Rhode Island and Boston, Massachusetts sites between 1959 and 1966. The National Collaborative Perinatal Project, conducted in 12 United States cities between 1959 and 1974, studied the children of 60,000 women from *in utero* to age 8 years (14). Our study participants were also participants in two later studies derived from the New England Family Study: the Brown-Harvard Transdisciplinary Tobacco Use Research Center (TTURC) study during the years 2001-2004, described in detail elsewhere (15); and the EdHealth study comprising 617 participants of the TTURC study (assessed during the years 2005-2007), who were selected with preference for those who were of racial/ethnic minority, and of low or high educational attainment. Inclusion criteria were that participants have data for maternal and paternal components of the PBI (obtained from the TTURC study), and 10-year CHD risk variables (obtained from the EdHealth study). Consequently, this is a cross-sectional study embedded within a longitudinal cohort. Of 617 participants interviewed, all outcome variables (including blood-based biomarkers) were available for 430 participants. Four hundred sixteen of the 430 participants had data on all CHD risk variables, and of these, 270 had information on primary male and female caretaker care variables. The low number of participants completing the PBI was primarily due to its being administered as a mailed-in supplementary questionnaire of which approximately 70% were completed. Three participants who reported having either angina or a myocardial infarction were excluded, resulting in a final sample of 267 participants

(99 males and 168 females). Included participants (n=267) were significantly younger ($p < .001$), had lower BMI ($p = .01$), higher childhood SES ($p = .03$), higher HDL ($p = .01$), lower CHD risk (mean: 3.8 versus 5.0%; $p = .002$) and were less likely to be of Black race/ethnicity ($p < .001$) compared to EdHealth study participants excluded from our sample (n=350). Included and excluded participants were similar for systolic and diastolic blood pressure, total cholesterol, depressive symptomatology, adulthood SEP, smoking, and diabetes ($p>0.05$). All calculations for included vs. excluded subjects used participants who were included in the TTURC study. Study participants signed informed consent and the studies from which data were obtained were reviewed by Brown University or Harvard School of Public Health Institutional Review Board.

Parental Bonding Instrument (PBI)

The original PBI is a set of 25 questions designed to measure two principal components of the parent-child relationship: care (12 questions) and overprotection (13 questions), based on participant's recollection of relationships with her/his parents up to age 16 years (16). The PBI has been shown to have good concordance between monozygotic and dizygotic twins (mean intraclass correlation of 0.70 and 0.71, respectively) (17), and is fairly stable over a 20-year period independent of depression, gender and major life events (stability co-efficient for maternal care: 0.73) (18). Our study used a shortened validated version of the PBI, consisting of 8 questions for each parent. The care and overprotection components were each measured by 4 separate items chosen based on factor loadings in the original PBI analysis. The quality of care questions were: (a) was affectionate to me, (b) understood my problems and worries, (c) did not understand what I needed or wanted, (d) was emotionally cold to me. Responses were scored on a Likert scale of 0-3 and, after reverse-scoring questions c and d, were summed to obtain the care score. Cronbach's α for the maternal care and paternal care scales in our study sample were 0.86 and 0.88, respectively. Of the 267 participants in our sample, 261 (97.8%) reported their biological mother as the primary female caretaker, while 228 (85.4%) had a biological father as their primary male caretaker. For the purposes of our study, a parent was considered to be the primary caretaker reported by the participant. Quality of reported parental emotional care was calculated as the mean of the maternal and paternal care scores (Spearman's correlation coefficient between maternal and paternal care score = 0.51, $p < .001$; range of mean score: 0 to 12). Secondary analyses were performed using maternal and paternal care scores separately. We focused on the care sub-scale, as recent evidence suggests that it is most consistently related to health outcomes (5,6,19,20). Parental care, but not parental control, has been associated with psychological distress (20), and links between low care and depression have been demonstrated repeatedly (5,6,19). Our shortened PBI scale was validated in an external population of 192 McGill University students (aged 18 to 31 years). All 4 subscales of the shortened PBI (care and overprotection for mother and father) were strongly correlated with those of the full PBI (Spearman's correlation coefficient ranged from 0.88 to 0.94), and demonstrated good internal consistency (Cronbach's $\alpha = 0.77$ to 0.82). The shortened scale also compared well with the full scale in terms of its predictive validity for depression and anxiety, with the maternal care scale demonstrating the strongest correlation with depression ($r = -0.46$; $p < .001$).

10-Year Calculated Coronary Heart Disease (CHD) Risk

The 10-year risk of coronary heart disease was calculated as a percentage, separately for men and women, using the validated Framingham Risk Algorithm. This algorithm uses sex-specific Cox regression models that incorporate age, diabetes, smoking, total and HDL cholesterol, and systolic and diastolic blood pressure and is described in detail elsewhere (21). The Framingham algorithm has good predictive validity for CHD events in the Framingham Heart Study (c-statistic = 0.74 and 0.77 for men and women, respectively) (21). Current smoking was measured as self-report (yes/no). Lipids were measured in non-fasting plasma samples at CERLab

(Harvard Medical School, Boston, MA) using a Hitachi 911 analyzer. CERLab participates in the Centers for Disease Control and Prevention/National Heart, Lung, and Blood Institute Lipid external quality control standardization program. Total cholesterol was measured enzymatically as described elsewhere (CV = 1.7%) (22). HDL cholesterol was determined using a direct enzymatic colorimetric assay shown to meet requirements established by the Lipid Standardization Program (CV = 3.3%) (23). Presence of diabetes was assessed by self-report to the question "Have you ever been told by a doctor or health professional that you have diabetes? (yes/no)". Systolic and diastolic blood pressure were measured in seated, resting participants, using automated blood pressure monitors (VSMedTech BpTru, Coquitlam, BC, Canada) with good validity and reliability compared with auscultation (24). Five blood pressure readings were obtained; systolic and diastolic values were calculated as the mean of the lowest three systolic or diastolic blood pressure readings, excluding the first recorded blood pressure.

Covariates

Childhood socioeconomic index was constructed as a composite index using parental education, occupation and income (range: 0-9.3) described elsewhere (25). Participants' own education was assessed through self-report of the number of years of education completed (range: 0-21 years). Depressive symptomatology scores were computed as the mean of 10 questions from the short-form Center for Epidemiologic Studies Depression Scale (CES-D) (range: 1-4). Body mass index (BMI) was calculated as the ratio of weight in kilograms to the square of height in meters (kg/m^2).

Statistical Approach

Sex-specific descriptive statistics were generated for covariates and individual components of the Framingham Risk Algorithm, according to average parental emotional care score (dichotomized as low and high care). Furthermore, sex-specific mean 10-year CHD risks were calculated according to quartiles of average parental emotional care score. Difference in parental emotional care score between males and females was assessed by the student's t-test. All analyses were sex-specific. Inclusion of an interaction term for parental emotional care and sex (care × sex) in multiple regression models of the association between parental care and 10-year CHD risk revealed a significant interaction ($p = .005$).

Multiple linear regression assessed sex-specific associations of parental emotional care with 10-year CHD risk. The distribution of the 10-year CHD risk variable was strongly skewed, and was hence log (natural) transformed. Our final model was not adjusted for age as the CHD risk score incorporates age, and further adjustment would induce excessive collinearity. Further analyses adjusted for childhood socioeconomic index, as the socioeconomic position of parents has been demonstrated to potentially influence quality of parental care (26). Parental mental illness was not included due to the small number of participants with a parent documenting mental illness ($N = 9$). Further models adjusted for potential mechanisms by which parental emotional care may influence CHD risk, including depressive symptomatology (5), adulthood socioeconomic position (27), and BMI (28) (Figure 1). As there were few participants of racial-ethnic minority (Black = 23, Hispanic = 3 and Other races = 17), primary analyses were not adjusted for race/ethnicity. However, sensitivity analyses additionally adjusting for race/ethnicity were performed. Multivariable-adjusted linear and logistic regression analyses evaluated associations between parental emotional care and individual components of the Framingham Risk Algorithm. Analyses were conducted using SAS version 9.1.3 (SAS Institute, Cary, NC).

Results

Descriptive characteristics of the study sample, categorized by high and low parental emotional care are shown in Table 1. Females who reported high parental emotional care were younger (p = .04), reported lower depressive symptomatology (p = .02) and smoking prevalence (p = .009), and higher educational levels (p = .04), compared to females with low parental emotional care. Males reporting high parental emotional care had a non-significantly higher likelihood of smoking (p = .10) compared to those reporting lower parental emotional care. Parental emotional care scores did not differ between men and women (score=8.5 for males and 8.3 for females; t-test p=0.58).

When the study population was classified by quartiles of the care scores, mean 10-year CHD risk among female offspring decreased with increasing parental emotional care score (p for trend = .02). Among male offspring, parental emotional care was not conclusively associated with elevated 10-year CHD risk (p = .12) (Table 2).

In unadjusted linear regression analyses among female offspring, average parental care score was inversely associated with CHD risk. In order to obtain results on the non log-transformed scale, regression beta coefficients were exponentiated and interpreted in terms of percent change of the non-transformed variable (29). Thus, a one unit increase in average parental emotional care score in females resulted in a 5.7% decrease [1-exp (-0.059)] in CHD risk (p = .002). Further adjustment for childhood SEP, which is expected to influence the quality of parental emotional care, reduced the magnitude of the regression coefficient to 0.047, i.e. a decrease in CHD risk of 4.6% (p = .009). Additional adjustment for adulthood SEP, depressive symptomatology, and BMI did not further change the results (p = .004). Therefore, if the average 10-year CHD risk among females in our study sample is 2.5%, then our models suggest that a one-unit increase in parental emotional care would decrease this risk by 4.6% after adjusting for covariates, resulting in a new 10-year CHD risk of 2.4%. A larger decrease of 5 points on the care scale would result in a 10-year CHD risk of 3.2%. In males, a one unit increase in reported parental emotional care score resulted in a statistically non-significant increase of 2.5% in average CHD risk (p = .22), and this association was not markedly influenced by further adjustment for covariates (Table 3).

In regression analyses for the association between the care score and individual components of the Framingham risk algorithm, we found that among female offspring, a one unit increase in reported parental emotional care decreased the odds of smoking in unadjusted analyses (odds ratio = 0.84, p = .004), and the association remained significant after adjusting for childhood SEP, but was no longer significant in the fully-adjusted model (Table 4). Furthermore, there was a decrease in depressive symptomatology (β = -0.036, p = .01) per unit increase in care score, but this was no longer significant in the fully adjusted model (Table 4). Among males, only depressive symptomatology was significantly associated with the care score (β = -0.071, p = .001) (Table 4).

The variance (R^2) in 10-year CHD risk score explained by the independent variable and each covariate in linear regression analyses is shown in Table 5. For female offspring, BMI explains almost 25% of the variance in CHD risk while all other variables explain between 5.0 and 9.8%. Parental emotional care explained 5.8% of the variance in 10-year CHD risk score. This showed that it explained fairly comparable amounts of variance to other proposed social and psychological determinants of CHD (depression, childhood SEP and educational attainment) but substantially less than that for obesity. Among males, all of the covariates independently explained only between 0.7 and 4.8% of the variance in CHD risk; the care score explained only 1.5% of the variance.

In secondary analyses that assessed maternal and paternal care components separately, instead of as a mean score for both parents, associations between emotional care and CHD risk among female offspring in the full model were similar to that for the average care score for both parents: a one unit increase in maternal care score resulted in a 3.5% decrease in CHD risk (β = -0.035, p = .01), and a one unit increase in paternal care score caused a 3.6% decrease in risk (β = -0.036, p = .02). Among male offspring, while no significant association was found between maternal care and risk (β = 0.012, p = .58), a weak positive association was observed between paternal care and risk, in the fully-adjusted model (β = 0.033, p = .05).

Sensitivity analyses demonstrated that additional adjustment for race/ethnicity did not markedly change associations of mean parental emotional care score with calculated 10-year CHD risk (females: β = -0.047, p = .01; males: β = 0.009, p = .67 in fully adjusted models). Further sensitivity analyses evaluated whether the association between parental emotional care and calculated 10-year CHD risk among females was primarily due to the association between parental care and smoking (as seen in Table 4). Multivariable regression analyses were performed on associations between parental emotional care and the calculated 10-year CHD risk with the smoking component removed from the Framingham algorithm. Findings remained strong for associations in females (see Table, Supplemental Digital Content 1, http://links.lww.com/PSYMED/A6, for details of the analysis). Table A).

Discussion

This study examined associations between parental emotional care and calculated 10-year risk of CHD, and found that associations differed by gender. The mean quality of emotional care for both parents was inversely associated with 10-year CHD risk in females, but not males, after adjusting for childhood SEP, adult SEP, depressive symptomatology and BMI. Both mother's and father's emotional care were inversely associated with 10-year CHD risk score in females. In males, mother's care score was not associated with CHD risk score, while father's emotional care score was weakly directly associated with CHD risk.

Prior Literature

Little is known about associations between parental emotional care and adult risk of CHD. Our study findings largely support the few findings to date, although sex-specific analyses have been rare. In a study by Dong *et al.* of 8529 members of a large health management organization, participants reporting familial emotional neglect in the first 18 years of life were 1.3 times more likely to report prevalent ischemic heart disease (95% CI, 1.1-1.6) compared to those reporting no emotional neglect, after adjusting for age, sex, race and education (13). In a study on general health as an outcome, the prospective Harvard Mastery of Stress study showed in 116 male participants that those who rated their parents poorly on measures of parental caring at baseline (aged approximately 20 years) were significantly more likely to have prevalent physician-diagnosed coronary artery disease, hypertension, and other diseases 35 years later (30).

Childhood abuse and maltreatment are extreme measures of parental neglect, which our study did not explicitly evaluate. However, other study findings provide some insight on extremely low levels of quality of parental care and its association with CHD. For example, the aforementioned study by Dong et al., demonstrated that measures of adverse childhood experiences, including emotional, physical and sexual abuse, domestic violence, parental mental illness, parental substance abuse, crime and physical neglect were associated with increased risk for ischemic heart disease (13). In the US National Comorbidity Survey, it was found that women (N = 2696) with a history of childhood maltreatment had an odds ratio of having a self-reported cardiovascular disorder of 8.8 (p < .001) compared to those not exposed to childhood maltreatment; associations were not found in males (N = 2697, OR = 0.90, p > .

05) (31). These findings were consistent with our study which found inverse associations between parental emotional care and CHD risk in females and not males. A study that used the prospective Dunedin cohort of 1030 participants, aged 32 years, found mixed results for associations between childhood maltreatment and cardiovascular risk clusters: participants defined as experiencing definite maltreatment had no association with cardiovascular risk clusters, while those experiencing probable maltreatment had a significant association [ORs of 1.33 (95% CI, 0.80-2.20) and 1.50 (95% CI, 1.09-2.08) respectively] (32).

Mechanisms

Adverse economic conditions are associated with poorer quality of parenting (33,34), and recent systematic reviews have demonstrated inverse gradients between childhood socioeconomic disadvantage and cardiovascular disease in most studies (35,36). Thus poor parental emotional care could merely be a proxy for low childhood SEP, and if this were the case, we would expect the association between parental emotional care and CHD risk to disappear after adjusting for childhood SEP. However, in our results among females, parental emotional care remained a significant risk factor for CHD, over and above childhood SEP.

We hypothesized that the association between parental emotional care and CHD risk could also be mediated by depression, adult SEP and BMI (Figure 1). Studies have consistently demonstrated inverse associations between parental care and depression (5,6). As depression is a known risk factor for CHD (7), it could be one mechanism through which poor parental care could lead to higher risk of CHD. Similarly, poor parenting could influence adult offspring's SEP and BMI by affecting childhood educational attainment and influencing eating and physical activity, respectively (10,37). Despite these three factors being potential mediators of the parental care-CHD risk relationship, and hence on the causal pathway, we adjusted for them and did not observe any further reduction in association strength. This indicates that while it is plausible that parental care could be mediated by these variables, it may influence CHD risk through other unmeasured factors.

Reasons for the gender differences observed in our study are not clear. In our sample, mean parental emotional care was inversely associated with calculated 10-year CHD risk in females but not males. One hypothesis is that male and female offspring interact differently with each parent, with females deriving a more beneficial outcome from higher interpersonal and caring relationships than males (38). For example, a recent longitudinal study that followed 806 adolescents for 5 years found that females reporting at least 5 family meals per week at baseline were significantly less likely to report regular use of cigarettes, alcohol and marijuana at follow-up compared to those reporting fewer family meals, after adjusting for family connectedness, race, SEP and baseline substance use. No association was seen for males (39). In addition, due to the divergent effects seen among males and females for associations of parental emotional care with smoking and BMI in our study, and that BMI was a much greater predictor of CHD risk in females than in males, it is possible that parental care may have a more influential effect on BMI and prevention of risky behavior in females. These findings are in keeping with those of another study that found that females reporting higher perceptions of parental care in adolescence were less likely to be overweight or obese as young adults, while the opposite was true for males (28). To test the plausible scenario that the significant inverse relationship between parental emotional care and CHD risk in females was mainly due to the association between parental care and smoking, we re-ran analyses after removing the smoking component in the Framingham risk algorithm. If parental care were merely predicting the smoking component of the CHD risk algorithm, we would expect the point estimates to be greatly attenuated. However, the results remained similar, even after adjusting for smoking. This suggests that the relationship between poor emotional care and heightened CHD risk in females is not accounted for solely by smoking, but likely by a more complex combination of risk

factors. In our study for male offspring, father's emotional care was weakly directly associated with calculated 10-year risk for CHD. During the era the study participants were youths (1960's) smoking rates were substantially higher in male adults (71.7% current or former smokers) than females (41.9% current or former smokers) (40). If sons who had positive relationships with their fathers modeled the smoking behavior, it may be that this contributed to the higher smoking rates seen in males who reported higher quality emotional care (26.4%) compared with lower quality care (13.0%, Table 1), thereby contributing to positive gradients between father's emotional care and calculated 10-year CHD risk. Other plausible reasons for gender differences merit further study.

Strengths and Limitations

Strengths of the study include accurate measurement of biological measures (including cholesterol, blood pressure, and BMI) using substantial internal and external quality control protocols. The exposure variable (PBI) is a validated measure of quality of parental emotional care. Furthermore, this study had measures of many potential confounders and mediators, enabling analyses to account for these covariates.

Limitations of the study include the small sample size, particularly for men, which limited statistical power. The low sample size among men may have introduced selection bias into the study, making these men less representative of the initially recruited sample. Although men included in our sample were similar on all demographic variables compared to excluded EdHealth participants, we were unable to test for differences on blood biomarkers as most of the excluded participants were missing this information. Thus, it is plausible that the null results among males are due to these differences, and larger studies are necessary to verify our results. In addition, the sampling method limited its generalizability to other populations. While the retrospective and self-reported aspect of the PBI could introduce misclassification bias into our study, it has been shown to have good validity(17,41) and reliability in numerous settings (18). Finally, this was a cross-sectional study as both the exposure and outcome variables were measured at single time-points. Consequently, causal inferences on associations between parental emotional care and risk for CHD are limited compared with prospective or intervention studies.

Conclusion

Our results demonstrate that, for females, perceptions of poor parental emotional care are associated with a significantly higher calculated 10-year risk for CHD, after adjusting for childhood and adult SEP, depression and BMI. Thus, these findings suggest that, if replicated using other study populations and study designs, that quality of parental emotional care may be a risk factor for CHD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding Sources: This work was supported in part by a Canadian Institutes of Health New Investigator Award (EBL), and was funded by a grant from NIH/NAI (grant # 1R01AG023397).

References

1. The World Health Organization. Cardiovascular diseases. [April 2, 2009]. 2008 Available from http://www.who.int/cardiovascular_diseases/en/

2. Barker DJ. The fetal and infant origins of adult disease. BMJ 1990;301:1111. [PubMed: 2252919]
3. Berenson GS, Srinivasan SR, Bao W, Newman WP 3rd, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. N Engl J Med 1998;338:1650–6. [PubMed: 9614255]
4. Lynch J, Smith GD. A life course approach to chronic disease epidemiology. Annu Rev Public Health 2005;26:1–35. [PubMed: 15760279]
5. Parker G, Hadzi-Pavlovic D, Greenwald S, Weissman M. Low parental care as a risk factor to lifetime depression in a community sample. J Affect Disord 1995;33:173–80. [PubMed: 7790669]
6. Sato T, Uehara T, Narita T, Sakado K, Fujii Y. Parental bonding and personality in relation to a lifetime history of depression. Psychiatry Clin Neurosci 2000;54:121–30. [PubMed: 10803804]
7. Frasure-Smith N, Lesperance F. Recent evidence linking coronary heart disease and depression. Can J Psychiatry 2006;51:730–7. [PubMed: 17168247]
8. Maccoby, E.; Martin, J. Socialization in the context of the family: Parent-child interaction. In: Mussen, P., editor. Handbook of Child Psychology. New York: Wiley; 1983. p. 1-101.
9. Hill KG, Hawkins JD, Catalano RF, Abbott RD, Guo J. Family influences on the risk of daily smoking initiation. J Adolescent Health 2005;37:202–10.
10. Lissau I, Sorensen TI. Parental neglect during childhood and increased risk of obesity in young adulthood. Lancet 1994;343:324–7. [PubMed: 7905145]
11. Batty, GDab; Wennerstad, KMc; Smith, GDd; Gunnell, Dd; Deary, IJb; Tynelius, Pc; Rasmussen, Fc. IQ in Early Adulthood and Mortality By Middle Age: Cohort Study of 1 Million Swedish Men. Epidemiology 2009;20:100–9. [PubMed: 19234402]
12. Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. Circulation 1993;88:1973–98. [PubMed: 8403348]
13. Dong M, Giles WH, Felitti VJ, Dube SR, Williams JE, Chapman DP, Anda RF. Insights into causal pathways for ischemic heart disease: adverse childhood experiences study. Circulation 2004;110:1761–6. [PubMed: 15381652]
14. Niswander, K.; Gordon, M. The Women and their Pregnancies: The Collaborative Perinatal Study of the National Institute of Neurological Diseases and Stroke. Washington D.C.: National Institutes of Health; 1972.
15. Gilman SE, Martin LT, Abrams DB, Kawachi I, Kubzansky L, Loucks EB, Rende R, Rudd R, Buka SL. Educational attainment and cigarette smoking: a causal association? Int J Epidemiol 2008;37:615–24. [PubMed: 18180240]
16. Parker G, Tupling H, Brown LB. Parental Bonding Instrument. Brit J Med Psychol 1979;52:1–10.
17. Parker G. Validating an experiential measure of parental style: the use of a twin sample. Acta Psychiatr Scand 1986;73:22–7. [PubMed: 3962700]
18. Wilhelm K, Niven H, Parker G, Hadzi-Pavlovic D. The stability of the Parental Bonding Instrument over a 20-year period. Psychol Med 2005;35:387–93. [PubMed: 15841874]
19. Rey JM. Perceptions of poor maternal care are associated with adolescent depression. J Affect Disord 1995;34:95–100. [PubMed: 7665811]
20. Chambers JA, Power KG, Loucks N, Swanson V. Psychometric properties of the Parental Bonding Instrument and its association with psychological distress in a group of incarcerated young offenders in Scotland. Soc Psych Psych Epid 2000;35:318–25.
21. Wilson PW, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. Prediction of coronary heart disease using risk factor categories. Circulation 1998;97:1837–47. [PubMed: 9603539]
22. Allain CC, Poon LS, Chan CS, Richmond W, Fu PC. Enzymatic determination of total serum cholesterol. Clin Chem 1974;20:470–5. [PubMed: 4818200]
23. Rifai N, Cole TG, Iannotti E, Law T, Macke M, Miller R, Dowd D, Wiebe DA. Assessment of interlaboratory performance in external proficiency testing programs with a direct HDL-cholesterol assay. Clin Chem 1998;44:1452–8. [PubMed: 9665423]
24. Mattu GS, Heran BS, Wright JM. Overall accuracy of the BpTRU--an automated electronic blood pressure device. Blood Press Monit 2004;9:47–52. [PubMed: 15021078]

25. Myrianthopoulos NC, French KS. An application of the U.S. Bureau of the Census socioeconomic index to a large, diversified patient population. Soc Sci Med 1968;2:283–99. [PubMed: 5760819]
26. Pulkki L, Keltikangas-Jarvinen L, Ravaja N, Viikari J. Child-Rearing Attitudes and Cardiovascular Risk among Children: Moderating Influence of Parental Socioeconomic Status. Prev Med 2003;36:55–63. [PubMed: 12473425]
27. Power C. Social and economic background and class inequalities in health among young adults. Soc Sci Med 1991;32:411–7. [PubMed: 2024156]
28. Crossman A, Anne Sullivan D, Benin M. The family environment and American adolescents' risk of obesity as young adults. Soc Sci Med 2006;63:2255–67. [PubMed: 16828216]
29. UCLA. Academic Technology Services SCG. Introduction to SAS. [April 2, 2009]. Available from http://www.ats.ucla.edu/stat/mult_pkg/faq/general/log_transformed_regression.htm
30. Russek LG, Schwartz GE. Perceptions of parental caring predict health status in midlife: a 35-year follow-up of the Harvard Mastery of Stress Study. Psychosom Med 1997;59:144–9. [PubMed: 9088050]
31. Batten SV, Aslan M, Maciejewski PK, Mazure CM. Childhood maltreatment as a risk factor for adult cardiovascular disease and depression. J Clin Psychiatry 2004;65:249–54. [PubMed: 15003081]
32. Melchior M, Moffitt TE, Milne BJ, Poulton R, Caspi A. Why do children from socioeconomically disadvantaged families suffer from poor health when they reach adulthood? A life-course study Am J Epidemiol 2007;166:966–74.
33. Dodge KA, Pettit GS, Bates JE. Socialization mediators of the relation between socioeconomic status and child conduct problems. Child Dev 1994;65:649–65. [PubMed: 8013245]
34. McLoyd VC. Socioeconomic disadvantage and child development. Am Psychol 1998;53:185–204. [PubMed: 9491747]
35. Galobardes B, Lynch JW, Davey Smith G. Is the association between childhood socioeconomic circumstances and cause-specific mortality established? Update of a systematic review. J Epidemiol Commun H 2008;62:387–90.
36. Galobardes B, Lynch JW, Davey Smith G. Childhood socioeconomic circumstances and cause-specific mortality in adulthood: systematic review and interpretation. Epidemiol Rev 2004;26:7–21. [PubMed: 15234944]
37. Johnson J, Cohen P, Kasen S, Judith S. Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. Am J Psychiat 2002;159:394–400. [PubMed: 11870002]
38. Davies PT, Linsay LL. Interparental Conflict and Adolescent Adjustment: Why Does Gender Moderate Early Adolescent Vulnerability? J Fam Psychol 2004;18:160–70. [PubMed: 14992618]
39. Eisenberg ME, Neumark-Sztainer D, Fulkerson JA, Story M. Family Meals and Substance Use: Is There a Long-Term Protective Association? J Adolescent Health 2008;43:151–6.
40. Centers for Disease Control and Prevention. Atlanta: 2007. Percentage of adults who were current, former, or never smokers, overall and by sex, race, Hispanic origin, age, education, and poverty status. National Health Interview Surveys, Selected Years - United States, 1965–2006. http://www.cdc.gov/tobacco/data_statistics/tables/adult/table_2.htm;
41. Parker G. The Parental Bonding Instrument. A decade of research. Soc Psych Psych Epid 1990;25:281–2.

Abbreviations

CHD	coronary heart disease
PBI	parental bonding instrument
BMI	body mass index
SEP	socioeconomic position
HDL	high density lipoprotein
CES-D	Center for Epidemiologic Studies Depression Scale

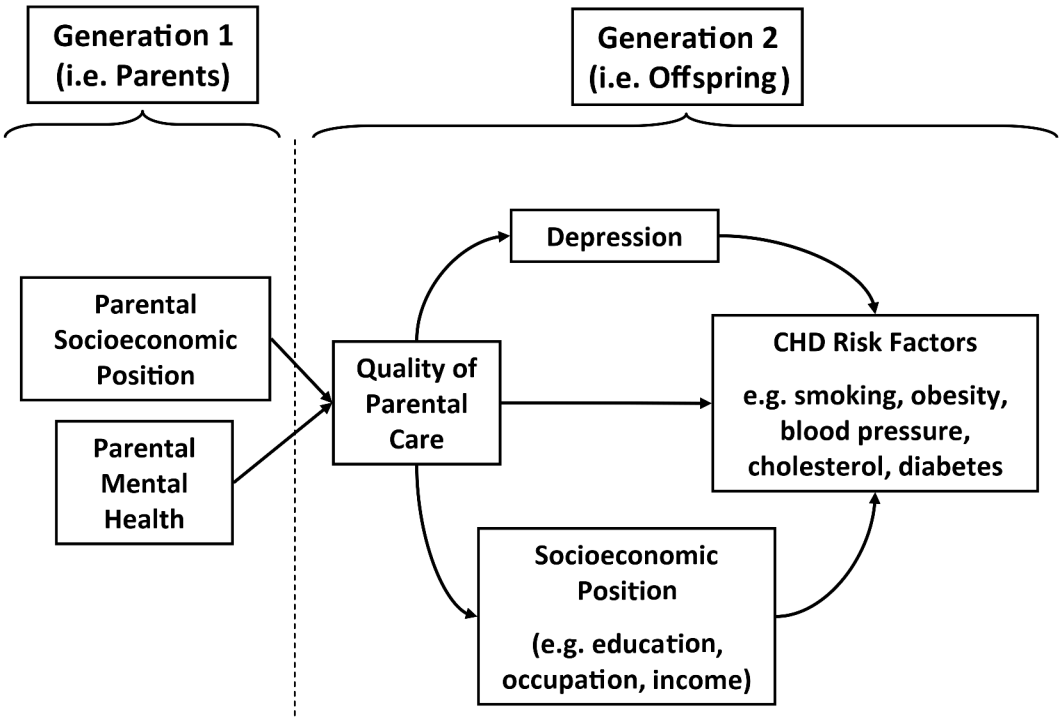

Figure 1.
Conceptual diagram demonstrating the potential mechanisms by which quality of parental emotional care may influence coronary heart disease (CHD) risk factors.

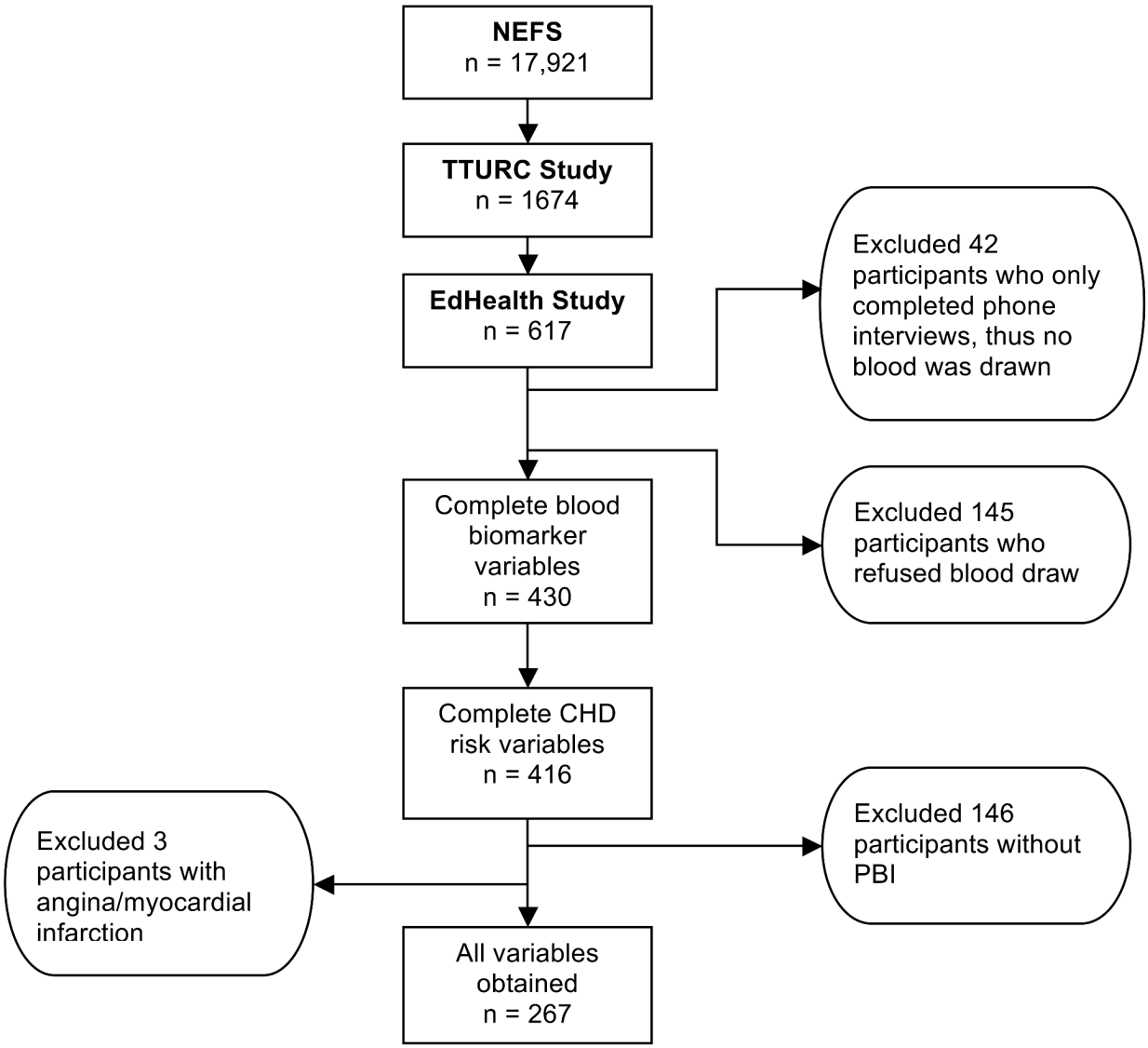

Figure 2.
Flowchart of included and excluded study participants. PBI, Parental Bonding Instrument; CHD, Coronary Heart Disease; NEFS, New England Family Study; TTURC, Transdisciplinary Tobacco Use Research Center study.

Table 1

Characteristics of study participants, according to quality of parental emotional care.

	Parental care					
	Females (*N* = 168)			Males (*N* = 99)		
	Low	High	*p**	Low	High	*p**
Age, y	42.3 (1.8)	41.8 (1.6)	**.04**	42.3 (1.7)	42.5 (1.8)	.60
Race, %						
Non-Hispanic white	84.5	90.5		78.3	77.4	
Black	7.1	8.3	.88	8.7	11.3	.75
Hispanic	1.2	0		0	3.8	
Other	7.2	1.2		13.0	7.6	
Childhood socioeconomic index	5.6 (1.9)	5.9 (1.9)	.47	5.4 (1.9)	5.9 (2.1)	.24
Parental mental illness, %	5.0	3.7	.72	2.4	2.0	1.00
Smokers, %	35.7	17.9	**.01**	13.0	26.4	.10
BMI, kg/m^2	28.9 (7.7)	26.8 (7.7)	.08	30.0 (7.3)	30.9 (7.4)	.52
Systolic blood pressure, mm Hg	110 (15)	110 (17)	1.00	116 (15)	117 (11)	.77
Diastolic blood pressure, mm Hg	72 (11)	73 (11)	.60	76 (11)	77 (9)	.67
Total cholesterol, mg/dl	192.4 (41.3)	193.8 (37.7)	.82	201.0 (41.8)	197.7 (39.8)	.69
HDL cholesterol, mg/dl	53.8 (19.6)	57.5 (15.6)	.18	43.9 (12.0)	42.0 (12.6)	.44
Diabetes, %	4.8	1.2	.37	2.2	5.7	.62
Adulthood socioeconomic position (years of education)	13.5 (2.4)	14.3 (2.5)	**.04**	13.3 (2.0)	13.6 (3.4)	.59
Depressive symptoms (CES-D)	1.7 (0.6)	1.5 (0.5)	**.02**	1.6 (0.6)	1.4 (0.5)	.07

For continuous variables, values are mean (SD).

BMI, body mass index; HDL, high density lipoprotein; CES-D, Center for Epidemiologic Studies Depression Scale.

* *p* values generated from t-tests (for continuous variables) and χ^2 (for categorical variables).

Psychosom Med. Author manuscript; available in PMC 2011 February 1.

Table 2

Calculated 10-year coronary heart disease (CHD) risk score (% risk for coronary heart disease in the next 10 years) according to quartile of parental emotional care.

Quartile of Average Parental emotional care Score	10-yr CHD risk (%)					
	Females			Males		
	N	Mean (95% CI)	*p*†	*N*	Mean (95% CI)	*p*†
Low (0-6)*	40	3.3 (2.4,4.2)	.02	18	5.7 (4.1,7.4)	.12
Medium-Low (6.5-8.5)*	44	2.5 (2.0,3.0)		28	5.7 (4.6,6.9)	
Medium-High (9-10.5)*	42	2.1 (1.6,2.6)		33	5.8 (4.7,6.9)	
High (11-12)*	42	2.2 (1.5,2.9)		20	7.5 (5.6,9.5)	

*Numbers in parentheses indicate range of mean parental care score for each category

†*p* values generated using tests for trend.

Table 3

Multivariable-adjusted linear regression analyses for the association between parental emotional care score and log-transformed 10-year coronary heart disease risk

	Model Adjustment								
Sex	**Unadjusted**			**Childhood SEP**			**Childhood SEP, Adulthood SEP, Depressive Symptomatology, BMI**		
	β*	**95% CI**	***p***	**β**	**95% CI**	***p***	**β**	**95% CI**	***p***
Females	**-0.059**	**-0.096, -0.023**	**.002**	**-0.047**	**-0.083,-0.012**	**.009**	**-0.047**	**-0.079,-0.015**	**.004**
Males	0.025	-0.015,0.066	.22	0.029	-0.012,0.069	.17	0.025	-0.017,0.068	.24

SEP, socioeconomic position; BMI, body mass index

*Regression co-efficient (β) represents change in logarithmic 10-year CHD risk for a 1-unit change in average parental emotional care score.

Table 4

Linear and logistic regression analyses for associations between parental emotional care score and CHD risk factors.

	Model Adjustment								
	Unadjusted			Childhood SEP			Childhood SEP, Adulthood SEP, Depressive Symptomatology, BMI		
	β*	95% CI	*p*	β	95% CI	*p*	β	95% CI	*p*
Systolic BP									
Females	0.079	-0.765,0.922	.85	0.167	-0.690,1.024	.70	0.196	-0.602,0.994	.63
Males	0.407	-0.589,1.402	.42	0.477	-0.535,1.488	.35	0.472	-0.582,1.524	.38
Diastolic BP									
Females	0.120	-0.454,0.694	.68	0.159	-0.427,0.744	.59	0.132	-0.426,0.690	.64
Males	0.163	-0.583,0.910	.67	0.141	-0.602,0.883	.71	0.325	-0.454,1.104	.41
Total cholesterol									
Females	-1.682	-3.778,0.414	.12	-1.705	-3.888,0.478	.13	-2.196	-4.456, 0.064	.06
Males	0.754	-2.268,3.777	.62	0.533	-2.574,3.639	.73	0.820	-2.543,4.183	.63
BMI									
Females	-0.194	-0.612,0.225	.36	-0.056	-0.483,0.373	.80	-0.036‡	-0.392,0.464	.87
Males	0.304	-0.238,0.846	.27	0.435	-0.021,0.890	.06	0.532‡	0.065,0.999	**.03**
CES-D									
Females	-0.036	-0.065,-0.007	**.01**	-0.031	-0.060,-0.001	**.04**	-0.025§	-0.054,0.003	.08
Males	-0.065	-0.105,-0.024	**.002**	-0.063	-0.105,-0.021	**.004**	-0.071§	-0.114,-0.029	**.001**
Education									
Females	0.128	-0.004,0.260	.06	0.092	-0.038,0.222	.16	0.072¶	-0.059,0.203	.28
Males	-0.013	-0.223,0.197	.90	-0.062	-0.265,0.140	.54	-0.051¶	-0.269,0.166	.64
	OR†	95% CI	*p*	OR	95% CI	*p*	OR	95% CI	*p*
Smoking									
Females	0.84	0.75,0.95	**.004**	0.87	0.77 0.99	**.03**	0.90	0.78,1.03	.12

	Model Adjustment								
	Unadjusted			Childhood SEP			Childhood SEP, Adulthood SEP, Depressive Symptomatology, BMI		
	β*	95% CI	*p*	β	95% CI	*p*	β	95% CI	*p*
Males	1.12	0.91,1.37	.30	1.17	0.94, 1.46	.15	1.12	0.88,1.42	.35

SEP, socioeconomic position; BMI, body mass index; CES-D, Center for Epidemiology Studies Depression scale.

*Regression coefficients (β) represent change in logarithmic 10-year CHD risk per1-unit change in average parental emotional care score.

†Odds ratios (OR) represent the odds of smoking per one-unit increase in the parental emotional care score.

‡Analyses not adjusted for BMI.

§Analyses not adjusted for CES-D.

¶Analyses not adjusted for education.

Table 5

Univariate (unadjusted) linear regression associations between the independent variables and logarithmic 10-year CHD risk, in female and male offspring

	Females				Males			
	β*	**95% CI**	***p***	**$R^{2\dagger}$**	**β**	**95% CI**	***p***	**R^2**
Average care score	**-0.059**	**-0.096,-0.023**	**.002**	5.8%	0.025	-0.015,0.066	.22	1.5%
Childhood SEI	**-0.112**	**-0.165,-0.059**	**<.001**	9.8%	**-0.059**	**-0.115,-0.002**	**.04**	4.4%
CES-D	**0.348**	**0.159,0.538**	**<.001**	7.3%	0.082	-0.111,0.276	.40	0.7%
Body mass index	**0.045**	**0.033,0.057**	**<.001**	24.7%	0.012	-0.003,0.027	.10	2.7%
Years of schooling	**-0.065**	**-0.107,-0.022**	**.003**	5.2%	**-0.043**	**-0.081,-0.004**	**.03**	4.8%

SEI, socioeconomic index; CES-D, Center for Epidemiology Studies Depression scale.

*Regression coefficients (β) represent change in logarithmic 10-year CHD risk for a 1-unit change in the independent variable

†R^2 indicates the variance in 10-year CHD risk explained by each of the independent variables