ORIGINAL ARTICLE

Emotional Vitality and Incident Coronary Heart Disease

Benefits of Healthy Psychological Functioning

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Context: The potentially toxic effects of psychopathology and poorly regulated emotion on physical health have long been considered, but less work has addressed whether healthy psychological functioning may also benefit physical health. Emotional vitality—characterized by a sense of energy, positive well-being, and effective emotion regulation—has been hypothesized to reduce risk of heart disease, but no studies have examined this relationship.

Objectives: To examine whether emotional vitality is associated with reduced risk of coronary heart disease (CHD). Secondary aims are to consider whether effects are independent of negative emotion and how they may occur.

Design: A prospective population-based cohort study.

Setting: National Health and Nutrition Examination Survey I and follow-up studies (a probability sample of US adults).

Participants: Six thousand twenty-five men and women aged 25 to 74 years without CHD at baseline, followed up for a mean 15 years after the baseline interview.

Main Outcome Measures: Measures of incident CHD were obtained from hospital records and death certificates. During the follow-up period, 1141 cases of incident CHD occurred.

Results: At the baseline interview (1971-1975), participants completed the General Well-being Schedule from which we derived a measure of emotional vitality. Compared with individuals with low levels, those reporting high levels of emotional vitality had multivariate-adjusted relative risks of 0.81 (95% confidence interval, 0.69-0.94) for CHD. A dose-response relationship was evident ($P<.001$). Significant associations were also found for each individual emotional vitality component with CHD, but findings with the overall emotional vitality measure were more reliable. Further analyses suggested that one way in which emotional vitality may influence coronary health is via health behaviors. However, the effect remained significant after controlling for health behaviors and other potential confounders, including depressive symptoms or other psychological problems.

Conclusion: Emotional vitality may protect against risk of CHD in men and women.

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THE POTENTIALLY TOXIC EFfects of psychopathology and poorly regulated emotion on physical health have long been considered, but less work has addressed whether healthy psychological functioning may also benefit physical health.[1] Folk wisdom and popular press have long suggested that positive psychological states promote health. While empirical tests of this idea are limited, existing evidence is provocative. Emerging work has demonstrated some protective benefit of positive thoughts or attitudes (ie, optimistic beliefs about the future) in relation to various health outcomes, including premature mortality and adverse cardiovascular outcomes.[2-6] In a recent review of findings that have linked both psychopathology and positive attitudes to risk of coronary heart disease (CHD), Rozanski and colleagues[7] identified emotional vitality as one critical positive psychological factor (related to but separate from optimism) that may promote cardiovascular health and reduce disease risk. Emotional vitality is characterized by a sense of energy and positive well-being in addition to being able to regulate emotions effectively. No studies to date have tested this hypothesis, though one study found

a protective association between emotional vitality and progression of disability and mortality in older women with disabilities.[8]

Identification of pathways through which emotional vitality may reduce CHD risk would provide further evidence that positive emotional functioning protects cardiovascular health. Emotional vitality may influence risk of CHD in several ways. It may alter disease susceptibility by acting directly on biological systems (eg, increasing vagal tone), motivating health-promoting behavior (eg, physical activity), and/or indirectly by buffering against potentially harmful negative emotional responses to stressful life experiences.[1] Because emotional vitality is conceptualized as an enduring characteristic of an individual, effects on both physiology and health behaviors are expected to result from cumulative lifetime exposure. In the present investigation, we prospectively examined the association between emotional vitality and CHD incidence using data from the National Health and Nutrition Examination Survey I (NHANES I), a nationally representative study of the US population, and follow-up studies. We hypothesized that, compared with low levels, high levels of emotional vitality will be associated with reduced risk of incident CHD. We further consider specific behavioral and biologic pathways through which emotional vitality may influence CHD risk.[9] Finally, given the many studies that have found an association between negative emotions and CHD,[9] we also evaluate the likelihood that observed effects are merely caused by the absence of negative emotion, which may be indicated by the presence of positive feelings. Of the various forms of psychological distress, depression has most consistently been linked with CHD[10]; hence, we specifically consider depression in the present analysis.

METHODS

SAMPLE AND STUDY DESIGN

Study participants were respondents to NHANES I, a multistage national probability survey conducted between 1971 and 1975 on the US civilian, noninstitutionalized population aged 1 to 74 years. The study oversampled women of childbearing age, persons living in poverty areas, and elderly persons. The baseline assessment, including a medical examination, blood draw, and in-person structured interview, was conducted on the full cohort. A detailed medical examination and selected psychological measures were obtained on a representative subsample of 6913 adults aged 25 to 74 years.[11] In those initially contacted for participation, the interview nonresponse rate was 1.4%, and the examination nonresponse rate was 30.5%. Interview nonresponders did not differ from participants on any demographic characteristics. However, older age, lower education, and residence in large urban centers were associated with examination nonresponse. Details of study design and sampling procedures are published elsewhere.[12]

Follow-up studies (NHANES Epidemiologic Follow-up Study [NHEFS]) were conducted in 1982, 1987, and 1992 on the entire surviving NHANES I cohort aged 25 to 74 years at baseline.[13-15] The NHEFS of 1986 was conducted only on those members aged 55 to 74 years at baseline.[16] Assessments included either in-person interviews (NHEFS 1982) or automated telephone interviews (NHEFS 1986, 1987, 1992) with respondent or proxy (for decedents), blood pressure and weight measurements (NHEFS 1982), tracking of members via the National Death Index and obtainment of death certificates, as well as records of reported overnight hospital and nursing home stays (at all follow-ups).

Our study included members of the detailed subsample. All members were traced at one or more of the follow-ups. Of these 6913 participants, 453 had baseline evidence of present or past cardiovascular disease by self-report or physical examination and were excluded from the analysis. An additional 195 had missing values for one or more covariates. The final sample available for analyses included 6265 participants (2853 men; 3412 women). The mean age of the study population at baseline was 47.5 years (SD, 14.03).

EMOTIONAL VITALITY AND OTHER PSYCHOLOGICAL MEASURES

Earlier work has identified a sense of vitality—a positive state associated with feelings of enthusiasm, energy, and interest—as a relevant barometer of emotional health.[7,17-19] Building on prior theory and empirical research, we define emotional vitality as a sense of positive energy, the ability to effectively regulate emotion and behavior, and positive well-being, which includes feeling engaged and interested in life. Emotional vitality was measured at baseline only, using items from the General Well-being Schedule, a validated measure with known psychometric properties.[20] Participants were assessed during NHANES I by trained interviewers. The General Well-being Schedule is commonly used in health research and contains 6 subscales that deal with depressed mood, anxiety, general health, vitality, emotional self-control, and a sense of positive well-being.[21,22] Items refer to how respondents have been feeling during the last month; however, these items have been demonstrated to be highly stable over time, likely reflecting enduring characteristics of an individual.[23] To be consistent with previous use of these items, we used the response scales as originally developed. Responses for all but one item range from 0 (none or not at all) to 5 (a lot or all of the time). For one item, responses ranged from 0 to 10 (additional analyses demonstrated that this item was not unduly influencing the overall measure). We used the vitality (eg, how much energy, pep, or vitality the participant had), sense of positive well-being (eg, how happy, satisfied, or pleased the participant has been in his/her personal life), and emotional self-control (eg, feeling emotionally stable and sure of oneself) subscales to derive the emotional vitality measure, but modified them by excluding items (1-2 for each subscale) that might be confounded with either negative affect or physical health (actual items used: "Have you been waking up fresh and rested?" "How much energy, pep, vitality have you felt?" "How happy, satisfied, or pleased have you been with your personal life?" "Has your daily life been full of things that were interesting to you?" "Have you been in firm control of your behavior, thoughts, emotions or feelings?" and "Have you been feeling emotionally stable and sure of yourself?").

Internal consistency reliability coefficients for the subscales were modest, ranging from 0.55 to 0.66. The 3 subscales were moderately intercorrelated, with correlation coefficients ranging from 0.44 to 0.52 ($P<.001$). Item scores from these subscales were combined additively to create an emotional vitality score (range, 0-35); this scale had high internal consistency reliability ($\alpha=0.79$). Low values indicate less of the attribute. To capture effects of meaningful differences in emotional vitality scores, we categorized scores into high, medium, and low levels based on tertiles of the score distribution in this sample. We then tested for both threshold and dose-response effects. We also considered effects of a single-unit change in emotional vitality on CHD using the continuous measure of emotional vitality.

Psychological distress was assessed in 2 ways. Depressive symptoms were assessed using the validated depressed mood subscale of the General Well-being Schedule.[21] No items on the General Well-being Schedule depressed mood scale were included in the emotional vitality scale. In addition, at the baseline interview, participants were asked to report on their history of psychological problems by indicating whether they had taken medication for psychological problems in the past 6 months or had ever had a physician tell them they had a nervous breakdown. The correlation between emotional vitality and depressive symptoms was somewhat higher than expected, perhaps because both measures were derived from the same parent scale ($r=-0.75$, $P<.001$). Individuals who reported use of medications for psychological problems had significantly lower levels of emotional vitality compared with those who did not report use (mean, 22.10 [SD, 6.38] vs 25.38 [SD, 5.36], respectively; $t_{6263}=17.99$; $P<.001$). Findings were similar when comparing individuals who did or did not report a nervous breakdown.

INCIDENT CHD

Coronary heart disease events were identified by hospital or nursing home discharge reports and death certificates. At each follow-up, participants reported all hospital or nursing home stays since last study contact. Hospitals or nursing homes were then contacted with participant permission and discharge reports were obtained for all visits in the study period. Participants were also tracked via the National Death Index; death certificates were obtained for decedents. A CHD event was recorded if *International Classification of Diseases, Ninth Revision* (*ICD-9*), codes 410 through 414 were listed on the hospital or nursing home discharge report or as the cause of death on the death certificate. The date of nonfatal CHD events was coded as the discharge date; if no discharge date was available, the event date was that of admission. The date of a fatal CHD event was date of death on the death certificate. For participants with more than one event (eg, myocardial infarction followed by CHD death), the earliest event was used.

COVARIATES

Most covariate measures were obtained from the NHANES I interview. Gender, marital status, smoking status (current vs never/former), leisure time physical activity (none to little, moderate, or high), and alcohol use (none, up to 2 servings per day, or >2 servings per day) were assessed. Current smokers were defined as individuals who smoke 1 or more cigarettes per day. Physical activity was measured by one item asking about level of recreational exercise, which was found to predict cardiovascular outcomes in this sample.[24] Educational attainment was used to indicate socioeconomic status, because it is attained relatively early in life and is stable over time. Education was divided into 4 categories: less than high school, high school graduate, some college, and college graduate. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Age and race/ethnicity (white vs nonwhite) obtained at the NHANES I interview were updated/corrected in NHEF 1982 to resolve discrepancies between interviews[13] (corrected values were used in the present analyses). Diabetes and hypertension statuses were based on self-reported (past or present), physician-diagnosed health conditions, and/or past or present medication use for the condition. Systolic blood pressure and diastolic blood pressure values were derived from one seated measurement taken during the NHANES I physical examination. Serum total cholesterol levels were determined from the blood drawn at this examination. Individuals excluded from the analyses owing to missing data on 1 or more of the covariates differed significantly from those in the analytic sample in that they were more likely to be nonwhite, unmarried, sedentary, have higher BMIs, and be more likely to drink alcohol, but they did not differ with regard to other covariates.

STATISTICAL ANALYSIS

Follow-up time was calculated as date of the baseline interview to the date of a CHD event, a non-CHD death, or the date last known alive. Relative risks (RRs) of incident CHD and 95% confidence intervals (CIs) associated with emotional vitality were estimated in multivariate Cox proportional hazards regression (PROC PHREG; SAS Institute Inc, Cary, North Carolina) to account for unequal follow-up time. A basic model was estimated adjusting for age, race/ethnicity, and gender, and survival plots were created by marking estimated Cox survival functions. A second model was subsequently estimated adjusting for known coronary risk factors, including educational attainment, marital status, smoking status, physical activity, alcohol use, systolic blood pressure, diastolic blood pressure, BMI, cholesterol, hypertension, diabetes, use of psychotropic medication, and reported psychological condition. Because many of the covariates may in fact be on the causal pathway between emotional vitality and CHD, we generally present findings with the basic models, but briefly report findings from the multivariate models when appropriate. Because psychological problems are often underdiagnosed, to ensure with more confidence that effects of emotional vitality were not confounded by negative affective symptoms, secondary analyses also (1) controlled for General Well-being Schedule depressed mood subscale score and (2) excluded highly depressed individuals identified using a cutoff point on the General Well-being Schedule depressed mood subscale validated by previous investigators against Center for Epidemiologic Studies Depression scale clinical cutoff points.[25]

The impact of covariates on the relationship between emotional vitality and CHD risk was estimated by adjusting for blocks of covariates: (1) demographics (age, race/ethnicity, gender, marital status, and educational attainment); (2) health behaviors (physical activity, smoking, and alcohol use); (3) cardiovascular factors (hypertension and systolic and diastolic blood pressure); and (4) metabolic factors (diabetes, cholesterol, and BMI). The effect of each block of covariates on the relationship between emotional vitality and CHD risk relative to the model including demographics only, was estimated as $1-\log(\text{hazard ratio}_{\text{adjusted}})/\log(\text{hazard ratio}_{\text{unadjusted}})$.[26] The degree of reduction of this hazard ratio was considered evidence of the degree to which each block of variables may serve as mechanisms that link emotional vitality to CHD.

Analyses were conducted using SAS, version 8.2 (SAS Institute). Possible effect modification by gender was examined in all models. We also examined effects excluding *ICD-9* codes 412 (old myocardial infarction) and 413 (angina) from the quantification of CHD events. Because results were largely unchanged, we have presented results for all CHD events (*ICD-9* codes 410 through 414) together. We also considered whether effects differ for nonfatal vs fatal CHD. Finally, we examined the association of emotional vitality with all-cause mortality. Models were subsequently estimated to account for the complex survey design, incorporating sample weights, clustering, and stratification within SAS-callable SUDAAN (Research Triangle Institute, Research Triangle Park, North Carolina). Findings were somewhat attenuated, but the conclusions were unchanged. Because sampling weights have been identified as problematic,[27] for ease of interpretability, we presented results unadjusted for the complex survey design.

Table 1. Distribution of Coronary Risk Factors According to Level of Emotional Vitality[a]

Risk Factor	Emotional Vitality Score		
	Low, 0-22 (n=1950)	Medium, 23-27 (n=2080)	High, 28-35 (n=2235)
Mean age (SD), y	47.5 (13.9)	47.4 (13.9)	47.7 (14.3)
Male sex[b]	706 (36.2)	925 (44.5)	1222 (54.7)
Nonwhite race/ethnicity[b]	348 (17.8)	232 (11.1)	259 (11.6)
Unmarried status[b]	333 (19.4)	300 (15.4)	285 (13.5)
Mean systolic blood pressure (SD), mm Hg	132.5 (23.5)	132.9 (22.1)	133.3 (21.9)
Mean diastolic blood pressure (SD), mm Hg	84.8 (13.8)	84.3 (12.4)	84.5 (11.6)
Hypertension	149 (7.6)	125 (6.0)	136 (6.1)
Diabetes[b]	104 (5.3)	75 (3.6)	56 (2.5)
Mean total cholesterol (SD), mg/dL	221.4 (36.6)	222.4 (46.9)	219.8 (45.6)
Mean body mass index (SD)[c]	26.0 (5.5)	25.5 (4.9)	25.3 (4.5)
Current cigarette smoking[b]	815 (41.8)	794 (38.2)	777 (34.8)
Alcohol use[b]			
None	525 (26.9)	515 (24.8)	504 (22.5)
1-2 servings per day	1299 (66.6)	1444 (69.4)	1574 (70.4)
> 2 servings per day	126 (6.5)	121 (5.8)	157 (7.0)
Physical activity[b]			
None to little	1010 (51.8)	803 (38.6)	638 (28.5)
Moderate	719 (36.9)	907 (43.6)	978 (43.8)
High	221 (11.3)	370 (17.8)	619 (27.7)
Educational attainment[b]			
< High school diploma	908 (46.6)	764 (36.7)	789 (35.3)
High school diploma	638 (32.7)	772 (37.1)	763 (34.1)
Some college	229 (11.7)	266 (12.8)	314 (14.0)
≥ College diploma	175 (9.0)	278 (13.3)	369 (16.5)

SI conversion factor: To convert total cholesterol to millimoles per liter, multiply by 0.0259.
[a] Values are number of participants (percentage) unless otherwise indicated.
[b] Differences across categories significant at $P<.05$.
[c] Calculated as weight in kilograms divided by height in meters squared.

RESULTS

The mean emotional vitality score among 6265 subjects was 24.79 (SD, 5.69; possible score range, 0-35). We examined the distribution of coronary risk factors by level of emotional vitality (**Table 1**) and tested for significant differences using χ^2 and analysis of variance tests as appropriate. All but 3 risk factors significantly varied by level of emotional vitality ($P<.01$). Individuals who were female; widowed, divorced, or separated; nonwhite; or had less education reported lower emotional vitality levels. Sedentary behavior and smoking were associated with lower emotional vitality levels, while moderate use of alcohol was associated with higher levels. Both diabetes and higher BMIs were associated with lower emotional vitality levels. We adjusted for all variables presented in Table 1 in further models.

EMOTIONAL VITALITY AND CHD INCIDENCE

Of the 6265 individuals, 1141 developed CHD during the mean 15-year follow-up period (mean, 15.1 [SD, 5.9]). After controlling for age, gender, and race/ethnicity, those with the highest levels of emotional vitality had an RR of 0.68 for CHD (95% CI, 0.58-0.78) compared with individuals who had the lowest levels of emotional vitality. The **Figure** illustrates the survival functions according to low, medium, and high levels of emotional vitality. Significant RR was still apparent (0.81; 95% CI, 0.69-0.94) after controlling for standard cardiovascular risk factors and history of psychological problems (**Table 2**). However, since many of the covariates may serve as mechanisms by which emotional vitality is related to CHD, estimates from the multivariate model may underestimate the association. Findings also indicate a dose-response relationship ($P<.001$). We also found a significant effect of emotional vitality on CHD incidence when emotional vitality was used as a continuous measure (RR, 0.97; 95% CI, 0.96-0.98; multivariate-adjusted RR, 0.98; 95% CI, 0.97-0.99), suggesting a 2% to 3% decrease in risk of CHD for each unit increase on the emotional vitality measure.

A separate analysis indicated that a history of psychological problems was associated with greater risk of CHD (eg, nervous breakdown, RR, 1.42; 95% CI, 1.03-1.96). However, as indicated in the multivariate analyses reported previously, effects of emotional vitality were maintained after controlling for measures, indicating a history of psychological problems. Follow-up analyses were also conducted using the General Well-being Schedule depression subscale as a covariate instead. In analyses adjusting for age, gender, race/ethnicity, and depressive symptoms, effects of emotional vitality on CHD were also maintained (high vs low levels of emotional vitality, RR, 0.82; 95% CI, 0.68-0.99). In analyses that instead excluded individuals reporting high levels of depressive symptoms, a protective effect of emotional vitality on CHD

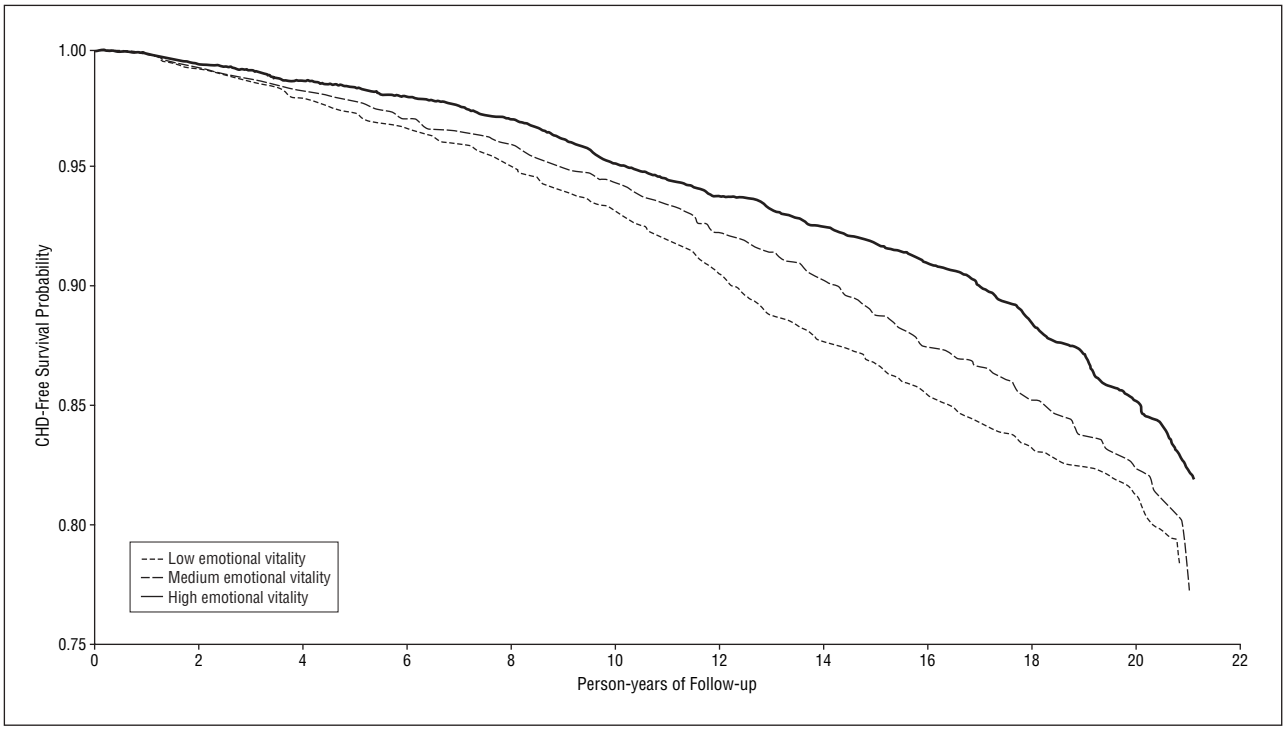

Figure. Relative risk of incident coronary heart disease (CHD) associated with level of emotional vitality, adjusted for age, gender, and race/ethnicity.

Table 2. Relative Risks (RRs) of Incident CHD and Mortality According to Level of Emotional Vitality[a]

End Point	Emotional Vitality Score			*P* Trend
	Low (0-22)	Medium (23-27)	High (28-35)	
Incident CHD				
Cases, No. (%)	380 (19.5)	395 (19.0)	366 (16.4)	
Basic RR[b]	1 [Reference]	0.86 (0.75-0.99)	0.68 (0.58-0.78)	< .001
Multivariate-adjusted RR[c]	1 [Reference]	0.93 (0.81-1.08)	0.81 (0.69-0.94)	.006
Mortality				
Cases, No. (%)	651 (30.2)	585 (26.4)	640 (27.4)	
Total mortality (with CHD death)[b]	1 [Reference]	0.81 (0.72-0.91)	0.76 (0.68-0.85)	< .001
Cases, No. (%)	468 (21.7)	407 (18.4)	474 (20.3)	
Total mortality (excluding CHD death)[c]	1 [Reference]	0.79 (0.70-0.91)	0.80 (0.70-0.91)	.001

Abbreviation: CHD, coronary heart disease.
[a] Data are presented as RR (95% confidence interval) unless otherwise indicated.
[b] Adjusted for age, gender, and race/ethnicity.
[c] Adjusted for age, gender, marital status (married or unmarried), race/ethnicity (white or nonwhite), educational attainment (less than high school, high school, some college, or college or more) systolic blood pressure, diastolic blood pressure, cholesterol, body mass index, smoking status (current or never/former), alcohol use (none, up to 2 servings/d, or more than 2 servings/d), physical activity (sedentary to light, moderate, or regular exercise), diabetes status (yes or no), hypertension status (yes or no), psychological condition (yes or no), and use of psychotropic medication (yes or no).

was still evident (high vs low levels of emotional vitality, RR, 0.74; 95% CI, 0.63-0.87).

POTENTIAL PATHWAYS BETWEEN EMOTIONAL VITALITY AND CHD

Given the sizeable reduction in the association between emotional vitality and CHD in the fully adjusted models, we explored what specific covariates might account for this reduction. To do so, in models controlling for all demographic covariates (ie, age, gender, race/ethnicity, marital status, and educational attainment), we considered 3 blocks of covariates separately—health behaviors (smoking status, physical activity, and alcohol use), metabolic factors (diabetes, cholesterol, and BMI), and blood pressure variables (systolic and diastolic blood pressure and hypertension)—and calculated the reduction in the hazard ratio corresponding to emotional vitality. Health behaviors were associated with the largest attenuation in the association between emotional vitality and CHD (reducing effect of emotional vitality by 27%), with metabolic factors accounting for some portion of the relationship (reducing effect of emotional vitality by 16%) (**Table 3**). However, it is

Table 3. Factors Accounting for the Relationship Between Emotional Vitality and Incident CHD in 6265 Participants

Factor	Relative Risk of Incident CHD			
	Model 1[a]	Model 2[b]	Model 3[c]	Model 4[d]
Emotional vitality				
Low	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]
Medium	0.88 (0.77-1.01)	0.92 (0.80-1.07)	0.89 (0.77-1.02)	0.89 (0.78-1.03)
High	0.70 (0.61-0.81)	0.77 (0.66-0.89)	0.71 (0.61-0.82)	0.74 (0.64-0.85)
Age, y	1.08 (1.07-1.08)	1.08 (1.07-1.09)	1.07 (1.06-1.07)	1.07 (1.07-1.08)
Female sex	0.55 (0.49-0.62)	0.54 (0.47-0.61)	0.54 (0.47-0.61)	0.50 (0.44-0.57)
Nonwhite race/ethnicity	0.89 (0.74-1.06)	0.83 (0.69-1.00)	0.79 (0.66-0.95)	0.80 (0.67-0.96)
Unmarried status	1.08 (0.94-1.25)	1.05 (0.91-1.21)	1.06 (0.91-1.22)	1.07 (0.93-1.24)
Education				
< High school	1.71 (1.36-2.15)	1.48 (1.17-1.86)	1.69 (1.34-2.12)	1.58 (1.25-1.98)
High school	1.34 (1.06-1.71)	1.25 (0.98-1.59)	1.34 (1.05-1.70)	1.26 (0.99-1.60)
Some college	1.35 (1.02-1.78)	1.30 (0.98-1.72)	1.37 (1.03-1.81)	1.31 (0.99-1.74)
≥ College	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]
Physical activity				
None to little		1.33 (1.12-1.58)		
Moderate		0.99 (0.83-1.18)		
High		1 [Reference]		
Alcohol consumption				
None		1.26 (0.97-1.63)		
Moderate		0.96 (0.97-1.63)		
High		1 [Reference]		
Smoking		1.58 (1.39-1.80)		
Hypertension			1.31 (1.07-1.59)	
Systolic blood pressure			1.01 (1.01-1.01)	
Diastolic blood pressure			1.00 (0.99-1.01)	
Cholesterol				1.00 (1.00-1.01)
Diabetes				1.98 (1.60-2.46)
Body mass index				1.05 (1.03-1.06)
Change in emotional vitality (high vs low), %[e]		– 27	– 4	– 16

Abbreviation: CHD, coronary heart disease.
[a] Adjusted for age, race/ethnicity, gender, marital status, and educational attainment.
[b] Adjusted for age, race/ethnicity, gender, marital status, educational attainment, exercise, alcohol use, smoking, and physical activity.
[c] Adjusted for age, race/ethnicity, gender, marital status, educational attainment, systolic blood pressure, diastolic blood pressure, and hypertension.
[d] Adjusted for age, race/ethnicity, gender, marital status, educational attainment, cholesterol, diabetes, and body mass index.
[e] Using formula 1 – log(hazard ratio$_{adjusted}$) / log(hazard ratio$_{unadjusted}$).

worth noting that the relationship between emotional vitality and incident CHD remained significant even after controlling for all covariates.

ADDITIONAL ANALYSES

Given gender differences in levels of emotional vitality, we considered whether the association between emotional vitality and CHD varied across men and women. Stratified analyses indicated virtually identical parameter estimates across men and women. When we formally tested the interaction term between gender and emotional vitality in the models, it was not significant ($P = .81$). We examined whether effects of emotional vitality might differ for nonfatal (n = 722) and fatal (n = 419) CHD outcomes. No difference was apparent. For example, significant RR was apparent for both nonfatal (high vs low levels of emotional vitality, RR, 0.66; 95% CI, 0.55-0.79) and fatal CHD (high vs low levels of emotional vitality, RR, 0.72; 95% CI, 0.57-0.92) in basic models. Results were similar in the multivariate-adjusted models for nonfatal CHD (high vs low levels of emotional vitality, RR, 0.77; 95% CI, 0.63-0.93) but were no longer significant for fatal CHD (high vs low levels of emotional vitality, RR, 0.91; 95% CI, 0.71-1.16). To address the possibility that those with lower emotional vitality had undetected disease at baseline, we examined the association between emotional vitality and CHD after excluding any CHD cases that developed within 3 years of baseline. Findings were unchanged from those reported previously. Finally, we examined whether emotional vitality was associated with all-cause mortality after controlling for age, gender, and race/ethnicity. Emotional vitality was associated with both all-cause mortality and mortality excluding deaths from CHD (Table 2).

To consider whether any single component of emotional vitality could be considered the primary or active factor of its effect on CHD, we conducted analyses with the 3 contributing subscales separately. Findings suggested that each subscale considered separately was associated with incident CHD in both basic and multivariate-adjusted models. For example, using the continuous measures, vitality (RR, 0.94; 95% CI, 0.93-0.96), positive well-being (RR, 0.95; 95% CI, 0.91-0.98), and emotional self-control (RR, 0.94; 95% CI, 0.91-0.98) were each associated with CHD in basic models. Effects were similar but somewhat attenuated in multivariate-adjusted models. However, CIs around the effect estimates for the

individual subscales were wider than for the overall measure of emotional vitality, suggesting greater reliability of the overall measure than its component parts.

COMMENT

This is the first study, using prospective data from a nationally representative sample of US men and women, to evaluate the hypothesis that emotional vitality may reduce risk of incident CHD. Findings suggest that individuals with higher levels of emotional vitality had reduced risk of developing CHD during a 15-year follow-up period. Further analyses indicated that one mechanism underlying this relationship may be health behaviors. Greater emotional vitality was significantly associated with less smoking, higher alcohol consumption, and more physical activity; after including these behaviors in the models, the relationship between emotional vitality and incident CHD was attenuated. However, the association remained significant after controlling for these behaviors as well as a history of psychological problems, use of psychotropic medications, current depressive symptoms, and other covariates. Such results suggest we need to consider additional mechanisms through which emotional vitality might influence CHD risk.

Positive emotional functioning and feelings have long been cited as an important component of psychological health. Findings from our study are consistent with and extend prior work showing relationships between healthy psychological functioning and cardiovascular health, in both magnitude and direction of the effects. For example, prospective studies have found positive beliefs (eg, optimism) to be associated with reduced risk of various adverse cardiovascular outcomes.[2-6,28-30] However, whereas this prior work is aimed at positive attitudes or thoughts, there is limited work looking more explicitly at effects of positive emotional functioning on coronary health. In addition, much of the existing work has been conducted in specialized samples of older men and women and often includes only one sex. To our knowledge, our study is the first to consider the relationship between a measure of emotional vitality and CHD incidence in a representative sample of US men and women.

Because negative emotion has frequently been identified as a risk factor for CHD,[9,31] a common concern with studies of positive emotion and health is that these observed associations simply reflect the absence of negative affect.[1] In fact, the high correlation between emotional vitality and depressive symptoms suggests that the measure of emotional vitality likely encompasses the absence of depressive symptoms. However, it is important to note that the association between emotional vitality and CHD was maintained after taking into account history of psychological difficulties and current depressive symptoms or after excluding highly distressed individuals from the analysis. The consistency of the association between emotional vitality and CHD after accounting for negative affect in various ways suggests important and separate relationships between positive emotional factors and CHD. These findings are broadly consistent with the understanding that optimal functioning transcends the simple absence of distress.

This study makes use of existing data to explore whether one instantiation of positive psychological functioning—emotional vitality—may protect health. A measure of emotional vitality was constructed by combining theoretically derived affective, cognitive, and behavioral components into a larger measure.[7] We note that items were chosen based on their theoretical relevance to emotional vitality and their distinction from depression. To increase the distinction between emotional vitality and depression, we excluded items if they appeared to be assessing depressed mood (ie, "How depressed or cheerful have you been?"). However, we also considered the emotional vitality components separately to determine whether any single component primarily may be driving observed associations with CHD. Each component was positively and significantly associated with CHD. However, the SEs of the estimates for the subscales considered separately tended to be larger than for the total emotional vitality scale, and the internal consistency correlation coefficient was robust for the total scale unlike the somewhat modest reliability estimates for each subscale. Taken together, this suggests that it may be more parsimonious to consider emotional vitality overall rather than its components in relation to CHD.

Mechanisms by which emotional vitality or other positive psychological factors may promote coronary health remain undetermined. Our study suggested some evidence that emotional vitality may motivate health-promoting behavior. However, since these behaviors could not fully account for the relationship between emotional vitality and CHD, other pathways must be considered. Positive emotions have been hypothesized to be restorative and regenerative, providing a sense of positive energy as well as enhancing a variety of capacities, like the ability to concentrate and problem solve or to mobilize social or other resources.[7,32] Other work has speculated that positive emotions may allay the atherogenic effects of conditions associated with chronic hyperarousal by reducing activation of neuroendocrine, cardiovascular, and inflammatory processes.[7] For example, recent work has demonstrated associations of positive affect with lower heart rate, lower levels of cortisol, and attenuated fibrinogen stress responses as well as with reduced ambulatory systolic blood pressure assessed 3 years later.[33,34]

Our study must be considered in light of several potential limitations. It is possible that there is residual confounding or that some unmeasured third variable (eg, a genetic factor) accounts for both greater emotional vitality and reduced risk of CHD. While we controlled for standard cardiovascular risk factors, it is possible that other factors are also relevant, eg, dietary intake. It is also notable that measures of health behaviors were assessed in the 1970s and that the findings may differ depending on the period in which the data were collected. In addition, we have only a single assessment of emotional vitality; however, other research has suggested that related measures of positive psychological attributes are quite stable over time

regardless of changes in life circumstances.[23,35,36] The measure of emotional vitality was derived for our study and we have no independent measures of its reliability or validity. However, it correlated with conceptually related measures and predicted outcomes in the expected ways, suggesting that it is capturing a real and important aspect of psychological functioning. The magnitude of the effects found are modest, though they are in line with other work on psychological risk or resilience factors in relation to CHD.[1] The study's strengths include its prospective design with CHD events quantified via hospital records and death certificates. It also includes its long follow-up during a 20-year period in a nationally representative US sample, which increases the generalizability of results.

This study suggests that healthy psychological functioning may have far-reaching benefits, beyond those we traditionally identify. It adds to the growing evidence that suggests an important role of healthy psychological functioning in cardiovascular health that transcends the simple absence of negative psychological factors. Emotional vitality and other aspects of healthy psychological functioning may allow individuals to respond effectively to environmental challenges as well as provide reserve capacity for coping with the myriad experiences and situations with which individuals are confronted daily.[7] Although these positive feelings and other aspects of healthy psychological functioning are due in part to early learning[37] and current social environmental conditions,[38] emerging work suggests interventions that may enhance these positive emotional processes.[39] For example, positive psychotherapeutic techniques that more strongly emphasize enhancing positive cognitions and affect, personal strengths, and meaningful life engagement have recently been proposed, rather than focusing solely on psychopathology or on reducing negative cognitions or affect.[40] Aspects of these techniques include learning skills to amplify the intensity and duration of positive emotion, transforming the structure of daily life toward being more engaged, and identifying and acting on characteristics that represent an individual's strengths.[41] Newer work has begun more rigorous testing of interventions derived from these techniques in relation to mental health outcomes as well as identifying components related to sustained change.[41,42] Thus, one implication of these findings may be for prevention and intervention. For example, it may be helpful for clinicians to consider not only reducing psychological distress but also to focus on promoting positive emotions, skills, and engagement with life.[9] Additional work will be needed to determine whether a focus on building or enhancing individuals' existing emotional resources may ultimately improve physical health.

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Correction

Error in Table. In the article titled "Two-Year Randomized Controlled Trial and Follow-up of Dialectical Behavior Therapy vs Therapy by Experts for Suicidal Behaviors and Borderline Personality Disorder," by Linehan et al, published in the July 2006 issue of the *Archives* (2006;63[7]:757-766), there was an error in Table 2. On page 761, Table 2, "Diagnostic Data" subsection, last row, the entire entry for "Psychiatric disorder not otherwise specified" should have been omitted.