ORIGINAL ARTICLE

Healthy Psychological Functioning and Incident Coronary Heart Disease

The Importance of Self-regulation

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Context: Studies have documented effects of positive and negative emotion on the risk of developing coronary heart disease (CHD), leading investigators to speculate about the importance of effective self-regulation for good health. Little work has directly assessed the role of self-regulation in risk of incident CHD.

Objective: To examine whether self-regulation is associated with reduced risk of coronary heart disease (CHD). Secondary aims were to consider whether the effects are independent of other measures of psychological functioning and how they may occur.

Design: A prospective population-based cohort study.

Setting: The Normative Aging Study, an ongoing cohort study of community-dwelling men in the Boston area.

Participants: One thousand one hundred twenty-two men aged 40 to 90 years without CHD or diabetes mellitus at baseline, followed up for an average of 12.7 years.

Main Outcome Measures: Measures of incident CHD obtained from hospital records, medical history, physical examination, and death certificates. During follow-up, 168 cases of incident CHD occurred, including 56 cases of incident nonfatal myocardial infarction (MI), 44 cases of fatal CHD, and 68 cases of angina pectoris.

Results: In 1986, 1122 men completed the revised Minnesota Multiphasic Personality Inventory, from which we derived a measure of self-regulation. Compared with men with lower levels, those reporting higher levels of self-regulation had an age-adjusted hazard ratio of 0.38 (95% confidence interval, 0.22-0.64) for combined nonfatal MI and CHD death. Moreover, a dose-response relation was evident, as each 1-SD increase in self-regulation level was associated with a 20% decreased risk of combined angina, nonfatal MI, and CHD death. Significant associations were also found after adjusting for anxiety, anger, or depression and after controlling for positive affect. The association could not be explained by known demographic factors, health behaviors, or biological factors.

Conclusion: Findings suggest that self-regulation may protect against risk of CHD in older men.

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NUMEROUS STUDIES HAVE documented the increased risk of developing coronary heart disease (CHD) associated with negative emotions, such as anxiety, depression, and anger.[1-3] Other work has demonstrated the protective effects of positive emotions and positive thoughts or attitudes on risk of CHD or cardiovascular death.[4-9] In recent work on positive psychological functioning and CHD risk, investigators have suggested that optimal functioning entails more than the simple absence of distress.[9,10] Moreover, what may be most protective for health is the ability to respond successfully to environmental challenges.[11,12] Thus, investigators have speculated about the importance of effective self-regulation in relation to health, with the capacity to regulate emotions central to this formulation.[11,13,14] Dysregulated emotion is a component of many forms of psychopathology. Although chronic high levels of distress may be indicative of poorly regulated emotion, effective self-regulation (or lack thereof) may be captured imperfectly by frequent reporting of negative or positive emotions. More direct assessment of the ability to regulate effectively may yield greater insight into how and why emotions influence cardiac health.

Research on the role of emotion regulation (and self-regulation more generally) in physical health is limited, with much of the work focused on health-related behaviors or on specific aspects of

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regulation, such as suppression or disclosure.[15-17] Regulating emotion is not simply suppressing emotions but a broader skill that involves effectively using and controlling emotions (positive and negative) in relationships and across a range of settings.[18] Inability to regulate effectively may be evident in childhood and persist into adulthood,[19,20] suggesting that regulatory capacity contributes to biological and behavioral alterations that accumulate over time and influence risk of adverse health in adulthood. Moreover, if we accept that chronically high levels of negative emotions (and personality traits associated with them) are markers of poor regulation,[14] the epidemiologic literature on emotion and disease development suggests that self-regulation matters for health.[21,22]

Healthy physiological functioning is characterized by flexibility and resilience. In contrast, aging and disease are characterized by loss of complexity in physiological and anatomic systems, resulting in decreased ability to compensate for changes in the internal or external environment.[11] Similarly, flexibility and capacity for adaptation are key components of healthy psychological functioning. For example, the ability to modulate emotions is associated with less psychological distress[23] and may also be linked with healthier physiological functioning. Effective self-regulation may influence risk of CHD by maintaining emotional flexibility and preventing chronic negative states.[11]

In the present investigation, we prospectively examined the association between self-regulation and CHD incidence using data from the Veterans Affairs Normative Aging Study (NAS), a longitudinal study of 2280 community-dwelling men. We hypothesized that, relative to less-effective self-regulation, highly effective self-regulation will be associated with reduced risk of incident CHD. We further considered specific behavioral and biological pathways by which self-regulation may influence CHD risk.[24]

METHODS

SAMPLE AND STUDY DESIGN

The NAS is a longitudinal study of aging established by the Veterans Administration in 1961.[25] The study cohort consisted of 2280 community-dwelling men from the greater Boston area aged 21 to 80 years at the time of entry. Volunteers underwent screening at entry according to health criteria and were free of any known chronic medical conditions at the start of follow-up.[25] Because men with known chronic conditions were excluded at baseline, the cohort originally consisted of healthy individuals. Study participants provided written informed consent, and the study protocol was approved by the institutional review boards of all participating institutions, including the Department of Veterans Affairs Boston Healthcare System.

For the present study, men without complete data on the measure of self-regulation (n=808) or who had preexisting CHD (angina pectoris or history of myocardial infarction [MI]) or diabetes mellitus were excluded (n=350), resulting in a study population of 1122 men. Although no studywide systematic analysis of loss to follow-up has been conducted, analyses of nonrespondents suggest that they do not differ from the overall cohort on key clinical and demographic variables.[26] **Figure 1** summarizes NAS participation and criteria for inclusion in the study sample. The mean (SD) age of the study population was 60.3 (8.3) years (range, 40-90 years).

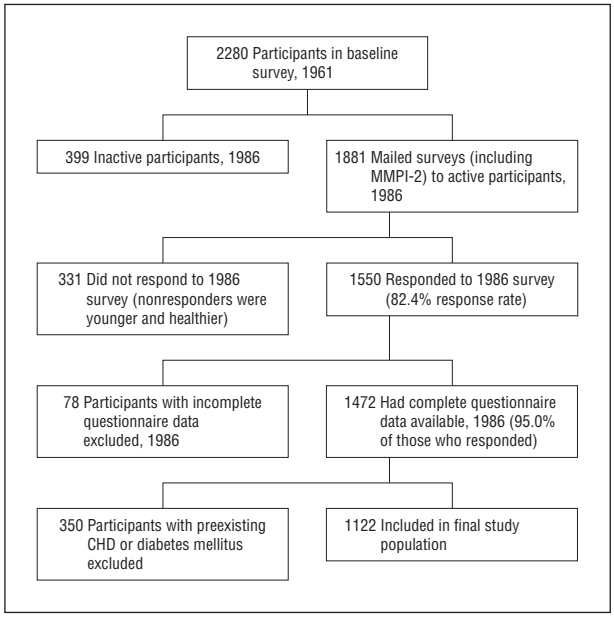

Figure 1. Flow diagram describing the Normative Aging Study participation and criteria for inclusion in the sample. CHD indicates coronary heart disease; MMPI-2, revised Minnesota Multiphasic Personality Inventory.

ASSESSMENT OF SELF-REGULATION

Conceptual Definition

Self-regulation has been defined in many ways, but most formulations include similar basic components.[27] Emotion regulation is of central importance, whereas other key components include planning or impulse control, beliefs about control, and actual control.[27,28] From a developmental perspective, the capacity to regulate is fundamental to adaptation, and a major task of early childhood is developing this ability.[18] Emotion regulation is particularly relevant and may powerfully contribute to or undermine a range of skills and competencies. Being able to control frustration, delay gratification, and self-soothe have important implications for learning in all domains, for navigating the social world, and for other aspects of self-regulation. Thus, in a recent formulation, self-regulation has been identified as a psychological asset that enables individuals to regulate what they feel and do and can be measured as an individual difference in adulthood.[29,30]

Validated measures of self-regulation as formulated in the preceding paragraph or more specific measures of emotion regulation have recently been developed,[29,31] but at the time the NAS was initiated few well-developed measures were available. However, extensive information on psychological functioning was obtained from which we were able to derive a measure of self-regulation. Building on prior theory and empirical research, we define *self-regulation* as a psychological asset that enables individuals to manage feelings, thoughts, impulses, and behavior, with the capacity to regulate emotions serving as a central component. Following recent theory, various aspects of regulation are assessed via the use (or not) of strategies such as selecting and modifying situations, directing attention to the self or other aspects of the situation, exerting mental control or reappraisal, and modulating responses.[15,32-34] Building on other work that has developed approaches for scoring positive traits from archival material,[35] self-regulation was assessed using 37 items selected on a priori theoretical grounds from the revised Minnesota Multiphasic Personality Inventory (MMPI-2 [available from

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the authors on request]). Comparison of the newly derived measure of self-regulation with other MMPI-2–based scales (eg, social responsibility, hostility control, and self-criticism) via item analysis, correlates, and conceptual meaning suggest that the new measure is conceptually distinct.[36-38]

Assessment

The MMPI-2 was administered by mail to all active cohort members (n=1881) in 1986.[39] Of these, 1550 men responded (82.4% response rate), in whom complete and valid questionnaire data were available in 95.0% (n=1472). Comparison of nonresponders with those who completed the MMPI-2 suggested that the nonresponders were somewhat younger and healthier (lower levels of blood pressure, cholesterol, and body mass index [BMI]). Previous research has indicated the validity of MMPI-2 items within populations ranging in age from 18 to 84 years and more specifically within the NAS population.[26]

Items for the MMPI-2–derived measure are similar to items specifically developed for and contained within previously validated trait measures of self-regulation, such as the Values in Action Inventory of Strengths or the Self-control Scale.[40-42] Exemplary items from these measures include "I control my emotions" or "Without exception, I do my tasks at work or school or home by the time they are due" (Values in Action Inventory of Strengths) and "I say inappropriate things" or "I do certain things that are bad for me, if they are fun" (Self-control Scale). Respondents rated each MMPI-2 item as true or false. Items indicating poor regulation were reverse scored, and the items were summed to obtain an overall scale score with high score indicating more effective self-regulation. Scale scores in the present sample ranged from 13 to 36, and the mean (SD) score was 29.03 (4.23).

Psychometrics

The internal consistency reliability of the scale was assessed as $\alpha=0.75$. Given that theory and empirical evidence suggest that emotions and their management play a central role in self-regulatory adaptive processes,[28] a measure of self-regulation should be correlated with negative and positive affect. As a check on construct validity, we examined the correlation of self-regulation with a range of measures obtained from 1986 to 1991, all designed to capture stable traits. Anxiety, hostility/anger, and depression (measured with the Symptom Checklist-90-R)[43] were each inversely correlated with self-regulation ($r=-0.45$ to $r=-0.51$ [$P<.001$]). Positive affect (measured with the Positive and Negative Affect Scale)[44] and happiness (measured with the Happiness Ladder)[45] were positively correlated with self-regulation ($r=0.11$ [$P=.004$] and $r=0.28$ [$P<.001$], respectively). Theory suggests that self-regulation may also be related to optimism. Previous work in this cohort has been published reporting a protective effect of optimistic explanatory style on CHD.[4] Models of optimism include self-regulatory components but focus primarily on individuals' expectations and sense that goals can be achieved. Thus, self-regulation is a broader attribute influencing more than a sense of agency. In the present study the correlation between self-regulation and optimism as measured by the Life Orientation Test[46] was $r=0.31$ ($P<.001$).

MEASUREMENT OF OTHER CARDIOVASCULAR RISK FACTORS

Every 3 to 5 years, participants in the NAS are followed up by physical examination, updating of medical history, and measurement of a variety of biochemical values, including serum cholesterol levels. Cigarette smoking status (current, former, and never) and alcohol drinking (usually take ≥2 drinks [yes/no]) are ascertained by a trained interviewer. Current smokers are defined as men who smoke at least 1 cigarette per day. Weight and height are measured with participants wearing only socks and underpants, from which BMI (calculated as weight in kilograms divided by height in meters squared) is calculated. Blood pressure is measured by an examining physician using a standard mercury sphygmomanometer with a 14-cm cuff. With the participant seated, systolic blood pressure and fifth-phase diastolic blood pressure are measured in each arm to the nearest 2 mm Hg. The average of systolic and diastolic blood pressures in each arm was used in analyses. Participants also reported whether they had a family history of heart disease (yes/no), and whether they completed education beyond high school (yes/no).

ASSESSMENT OF MORBIDITY AND MORTALITY

This study included all confirmed CHD end points that occurred from the return of the 1986 survey to May 2001, with a mean (SD) of 12.7 (4.1) years of follow-up. Individuals were censored at the time of developing a coronary event (or death) or up to the time of their most recent follow-up visit.

A medical history was obtained from each participant at his regular follow-up visit. Hospital records were obtained for every report of a possible CHD event and reviewed by a board-certified cardiologist (P.V.). Diagnostic categories of CHD include angina pectoris, nonfatal MI, and total CHD (nonfatal MI plus fatal CHD). Criteria for MI and angina pectoris were those used in the Framingham Heart Study.[47] Myocardial infarction was diagnosed only when documented by unequivocal electrocardiographic changes (ie, pathologic Q waves), by a diagnostic elevation of serum enzyme (aspartate aminotransferase and lactic dehydrogenase) levels together with chest discomfort consistent with MI, or by autopsy findings. Angina pectoris was diagnosed by a board-certified cardiologist (P.V.) on the basis of medical history and physical examination findings, using Framingham Heart Study criteria.[47] Angina was diagnosed when a study participant reported recurrent chest discomfort lasting up to 15 minutes distinctly related to exertion or excitement and was relieved by rest or by nitroglycerin adminstration.[47]

Death from CHD was confirmed when a death certificate (coded according to the *International Classification of Diseases, Ninth Revision, Clinical Modification*)[48] indicated an underlying cause of death coded 410 to 414. Medical records for each CHD death were reviewed by a board-certified cardiologist (P.V.) to ensure accurate coding. Most deaths occurring in this cohort are reported through next of kin or postal authorities. Birthday cards and supplemental questionnaires mailed to participants provided additional opportunities to ascertain vital status, and the records of the Department of Veterans Affairs and the Social Security Administration Death Master File were searched for possible unreported deaths.

DATA ANALYSIS

To capture effects of meaningful differences in self-regulation scores, we categorized scores into high, medium, and low levels based on tertiles of the score distribution in this sample. Differences in covariate distribution according to self-regulation level were assessed using analyses of variance for continuous covariates and χ^2 tests for categorical covariates. We ran Cox proportional hazards models using commercially available software (SAS)[49] to estimate the hazard ratios (HRs) of CHD according to self-regulation level, controlling for age, BMI, smok-

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Table 1. Distribution of Coronary Risk Factors According to Level of Self-regulation[a]

	Self-regulation Level		
	Low (13.00-27.75)	Medium (28.00-31.00)	High (31.56-36.00)
No. of participants	335	426	361
Age, y[b]	59.3 (8.7)	59.9 (8.3)	61.6 (7.8)
Education beyond high school, No. (%)[b]	176 (52.5)	267 (62.7)	225 (62.3)
Family history of CHD, No. (%) (n=1121)	83 (24.8)	90 (21.1)	65 (18.1)
Current smoker, No. (%) (n=1092)	94 (28.8)	98 (23.6)	50 (14.3)
Former smoker, No. (%) (n=1092)	166 (50.8)	192 (46.3)	162 (46.3)
Consumes ≥2 drinks of alcohol per day, No. (%)[b]	101 (30.1)	105 (24.6)	75 (20.8)
BMI[b]	26.9 (3.2)	26.5 (3.0)	25.9 (2.9)
Systolic blood pressure, mm Hg	125.9 (15.4)	126.9 (15.3)	128.43 (15.3)
Diastolic blood pressure, mm Hg	78.3 (8.6)	78.4 (8.9)	77.9 (7.9)
Serum cholesterol level, mg/dL[b]	252.5 (50.5)	244.0 (45.6)	244.7 (43.3)
Negative affect[c]			
Depression[b]	0.55 (0.40)	0.26 (0.35)	0.14 (0.19)
Hostility/anger[b]	0.46 (0.53)	0.19 (0.26)	0.09 (0.16)
Anxiety[b]	0.36 (0.46)	0.15 (0.24)	0.06 (0.11)
Positive affect[d]	3.28 (0.74)	3.40 (0.69)	3.38 (0.71)

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); CHD, coronary heart disease.
SI conversion factor: To convert cholesterol levels to millimoles per liter, multiply by 0.0259.
[a] Unless otherwise indicated, data are expressed as mean (SD). Percentages refer to percentage of individuals within the self-regulation category with this characteristic.
[b] Differences across categories were significant ($P<.05$).
[c] Measured by the Symptom Checklist-90.
[d] Measured by the Positive and Negative Affect Scale.

ing status, systolic and diastolic blood pressure, serum cholesterol level, family history of CHD, participant consumption of 2 or more drinks of alcohol per day, and participant completion of education beyond high school. Initial analyses included a quadratic function for age to assess the possibility of nonlinear relations between age and CHD. No evidence of nonlinearity was found; hence, models presented herein include only the standard age term. Age- and multivariate-adjusted analyses are presented to illustrate the role of potential confounders. To consider threshold and dose-response effects, we examined effects of a 1-SD change in self-regulation on CHD, as well as effects according to self-regulation tertiles.

Prior theory and empirical work suggest that effective self-regulation reduces chronic distress and enhances positive emotional experience. Empirical tests of these effects and their interrelationships in the context of developing CHD are not feasible in the current study owing to data availability. However, to ensure that effects of self-regulation capture something above and beyond the known association between negative affect and CHD, we used 2 strategies. First, we reran the models using a reduced measure of self-regulation by removing any items that were also included in emotion-related MMPI-2 content scales (ie, anger, anxiety, and depression). After removal of overlapping emotion content items, 26 items remained in the reduced self-regulation measure, with a significantly weaker internal consistency reliability ($\alpha=0.57$). Second, we reran all models controlling for anxiety, anger/hostility, or depression (measured with the Symptom Checklist-90-R), and separately for positive affect.

The impact of covariates on the relation between self-regulation and CHD risk was estimated by adjusting for the following blocks of covariates: (1) demographics (age, educational attainment, and family history of heart disease); (2) health behaviors (smoking and alcohol use); and (3) biological factors (systolic and diastolic blood pressure, cholesterol level, and BMI). The impact of each block of covariates (models 2 and 3, adjusted) on the relation between self-regulation and CHD risk relative to the model including demographics only (model 1, unadjusted) was estimated as $1-[\log(HR_{adjusted})/\log(HR_{unadjusted})]$.[50] The degree of reduction of this HR was considered as evidence of the degree to which each block of variables may serve as mechanisms linking self-regulation to CHD.

RESULTS

The mean (SD) self-regulation score among 1122 subjects was 29.03 (4.23) (range, 13-36). Tertiles were created according to the distribution of scores in this sample, with cut points at 13.00 to 27.75, 28.00 to 31.00, and 31.56 to 36.00. We examined the distribution of coronary risk factors by self-regulation level (**Table 1**). Some differences emerged in accordance with prior theory suggesting that self-regulation may be relevant for behavior and biological outcomes. Thus, although individuals with higher levels of self-regulation are somewhat older, they also have higher levels of education, are less likely to engage in unhealthy behaviors (ie, smoking and drinking), and have lower BMI and cholesterol levels. Although some of these factors may in fact be on the causal pathway linking self-regulation with CHD onset, to be conservative we adjusted for these variables in the standard multivariate proportional hazards analyses.

SELF-REGULATION AND CHD INCIDENCE

Of the 1122 men, 168 developed CHD during the average 12.7-year follow-up period. There were 56 cases of incident nonfatal MI, 44 cases of fatal CHD, and 68 cases of angina pectoris. Age-only analyses were highly simi-

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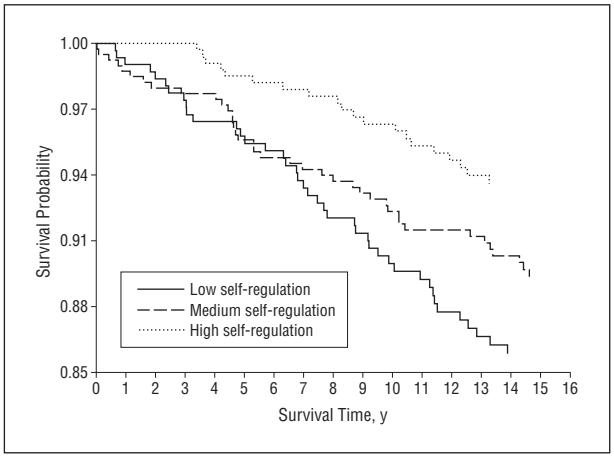

Figure 2. Kaplan-Meier survival curve for self-regulation and incident total coronary heart disease. Self-regulation levels include 355 participants with low, 426 with medium, and 361 with high self-regulation.

lar to multivariate-adjusted analyses. In analyses using self-regulation as a continuous variable, men with higher levels of self-regulation had multivariate-adjusted relative risks (HRs; per 1-SD increase in self-regulation) of 0.77 (95% confidence interval [CI], 0.60-0.99) for nonfatal MI, 0.79 (0.57-1.09) for fatal CHD, 0.81 for angina pectoris (0.64-1.02), and 0.78 (0.64-0.95) for total CHD. When all end points were combined (nonfatal MI, fatal CHD, and angina pectoris), each 1-SD increase in level of self-regulation was associated with a 20% decreased risk of incident CHD (HR, 0.80; 95% CI, 0.69-0.93). These analyses also provide evidence of a dose-response relationship for each outcome.

We also categorized self-regulation into tertiles. **Figure 2** illustrates the Kaplan-Meier survival probability according to low, medium, and high levels of self-regulation. This crude analysis indicates significantly reduced risk of developing total CHD associated with higher levels of self-regulation (log-rank test, $P = .006$). Significant relative risk was even more apparent after controlling for standard cardiovascular risk factors (**Table 2**). For example, relative to men with the poorest self-regulation, the men with the highest levels of self-regulation had a multivariate-adjusted HR of 0.46 (95% CI, 0.26-0.82) for combined nonfatal MI and CHD death.

ADDITIONAL ANALYSES

We examined whether self-regulation captures something above and beyond the known association between negative affect and CHD given that various negative emotions have been shown to increase CHD risk.[3] When items that are also included in emotion-related MMPI-2 content scales were removed from the measure of self-regulation, findings on the association between self-regulation and incident CHD were virtually identical. For example, with this measure, each 1-SD increase in self-regulation was associated with a multivariate-adjusted HR of 0.82 (95% CI, 0.67-1.00) for combined nonfatal MI and fatal CHD, and 0.82 (0.71-0.96) for combined nonfatal MI, fatal CHD, and angina pectoris. Similarly, effects of self-regulation on CHD outcomes were markedly unchanged when estimated by models that adjusted for all covariates, as well as anxiety, hostility/anger, or depression or for positive affect.

Because many of the covariates may serve as mechanisms by which self-regulation is related to CHD, we considered whether estimates from the multivariate model could be underestimating the association. To do so, in models controlling for all demographic covariates, we considered 2 blocks of covariates available in these data separately (health behaviors; metabolic and blood pressure variables) and calculated the reduction in the HR corresponding to self-regulation (**Table 3**). Health behaviors were associated with a modest attenuation in the association between self-regulation and CHD (reducing the effect of self-regulation by 5%), with biological factors accounting for a slightly greater portion of the relationship (reducing the effect of self-regulation by 9%). However, the association between self-regulation and incident CHD remained significant even after controlling for all covariates, suggesting that the effects of these covariates as possible confounders or mediators in this population is relatively modest.

Finally, there was no evidence of an association between self-regulation and all-cause mortality (multivariate-adjusted HR for a 1-SD increase in self-regulation, 0.96; 95% CI, 0.83-1.10).

COMMENT

A great deal of work has suggested the likely importance of self-regulation in cardiovascular health. This is, to our knowledge, the first study to evaluate directly the hypothesis that self-regulation may reduce the risk of incident CHD, using prospective data in a sample of community-dwelling men. Findings suggest that individuals with higher levels of self-regulation had reduced risk of developing CHD during a mean follow-up of 12.7 years. Similar to other studies of healthy psychological functioning and CHD, findings were strongest with the objectively measured CHD outcomes and moderate or nonsignificant with angina, the outcome most likely to be subject to self-report bias. Moreover, the association was unchanged after removing items with emotion-related content or controlling for measures of distress or positive affect. Further analyses were unable to identify a clear mechanism underlying this relationship as the association remained significant after controlling for available behavioral and biological risk factors for CHD. However, better self-regulation was significantly associated with a healthier cardiovascular profile at baseline, including less smoking, less excess alcohol consumption, lower BMI, and lower cholesterol levels.

The ability to self-regulate may be considered a critical psychological asset relevant for promoting mental and physical health. Failure to self-regulate has long been recognized as a component of numerous mental health disorders and clinical manifestations of distress that have been linked with a greater risk of developing CHD.[1,24,51] Conversely, effective self-regulation is associated with a number of constructs that have been linked with better health, including a sense of control, conscientiousness, optimism, and better cognitive performance.[4,7,52-55] In a

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Table 2. Hazards of Incident CHD According to Level of Self-regulation

End Point[a]	Level of Self-regulation, HR (95% CI)		
	Low	Moderate	High
Angina pectoris			
Age adjusted	1 [Reference]	0.88 (0.51-1.52)	0.54 (0.29-1.03)
Multivariate	1 [Reference]	1.12 (0.63-1.99)	0.75 (0.38-1.48)
Nonfatal MI			
Age adjusted	1 [Reference]	0.75 (0.42-1.34)	0.34 (0.16-0.73)[b]
Multivariate	1 [Reference]	0.81 (0.45-1.48)	0.42 (0.19-0.91)[c]
Fatal CHD			
Age adjusted	1 [Reference]	0.67 (0.34-1.31)	0.39 (0.18-0.85)[c]
Multivariate	1 [Reference]	0.75 (0.36-1.55)	0.50 (0.22-1.18)
Total CHD[d]			
Age adjusted	1 [Reference]	0.73 (0.47-1.13)	0.38 (0.22-0.64)[b]
Multivariate	1 [Reference]	0.80 (0.50-1.27)	0.46 (0.26-0.82)[b]
Combined angina and total CHD[e]			
Age adjusted	1 [Reference]	0.80 (0.57-1.12)	0.45 (0.30-0.67)[b]
Multivariate	1 [Reference]	0.93 (0.65-1.33)	0.57 (0.37-0.88)[b]

Abbreviations: CHD, coronary heart disease; CI, confidence interval; HR, hazard ratio; MI, myocardial infarction.

[a] A small number of cases (1-5, depending on the outcome) were dropped in multivariate analyses because of missing information on covariates. Multivariate analyses adjust for age, family history of CHD, educational attainment, smoking status, alcohol intake, systolic and diastolic blood pressure, serum total cholesterol level, and body mass index.

[b] $P < .01$.

[c] $P < .05$.

[d] Includes nonfatal MI and fatal CHD.

[e] Includes angina combined with nonfatal MI and fatal CHD.

Table 3. Hazards of Incident CHD According to Level of Self-regulation and Factors Accounting for the Relationship[a]

	Incident Total CHD, HR (95% CI)		
	Model 1	Model 2	Model 3
Self-regulation			
Low	1 [Reference]	1 [Reference]	1 [Reference]
Medium	0.76 (0.49-1.18)	0.78 (0.49-1.22)	0.79 (0.50-1.24)
High	0.41 (0.24-0.70)[b]	0.43 (0.24-0.75)[b]	0.44 (0.25-0.77)[b]
Age	1.06 (1.03-1.08)[b]	1.06 (1.04-1.09)[b]	1.05 (1.03-1.08)[b]
Education beyond high school	0.68 (0.46-1.02)	0.66 (0.44-0.98)[c]	0.67 (0.45-1.01)
Family history of CHD	1.80 (1.18-2.75)[b]	1.83 (1.19-2.82)[b]	1.75 (1.14-2.68)[b]
Alcohol consumption		0.88 (0.55-1.41)	
Current smoker		1.48 (0.78-2.79)	
Former smoker		1.53 (0.93-2.52)	
Systolic blood pressure			1.01 (0.99-1.02)
Diastolic blood pressure			1.01 (0.97-1.04)
Cholesterol level			1.00 (1.00-1.01)[c]
BMI			1.04 (0.97-1.11)

Abbreviations: BMI, body mass index; CHD, coronary heart disease; CI, confidence interval; HR, hazard ratio.

[a] Model 1 includes age, educational attainment, and family history of heart disease; model 2, model 1 covariates plus alcohol use and smoking; and model 3, model 1 covariates plus systolic and diastolic blood pressure, cholesterol level, and BMI. Incident CHD includes nonfatal myocardial infarction and fatal CHD. The effect of covariates on high vs low levels was a reduction of the effect of self-regulation of 5% for model 2 HRs and of 9% for model 3 HRs using the formula $1 - [\log(HR_{adjusted})/\log(HR_{unadjusted})]$.

[b] $P < .01$.

[c] $P < .05$.

prospective study of another aspect of healthy psychological functioning, emotional vitality was strongly associated with reduced risk of CHD.[9] One component of emotional vitality was emotional self-regulation. In fact, the extent to which psychological assets (or deficits) are related to regulatory capacity may provide a unifying framework for understanding which psychological attributes are relevant for health and why. Thus, self-regulation directly measured may be viewed not as yet another risk/resilience factor in competition with those already identified but as a higher-order aspect of functioning that may give insight into why or how healthy psychological functioning matters for cardiovascular health.

Self-regulation is a key component of healthy psychological functioning. Several other concepts that have been identified as health relevant are related to self-regulation. For example, mastery refers to the extent to which one regards changes in one's life as being under one's own control rather than fatalistically ruled,[56] and self-efficacy is situ-

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ation specific and refers to having the confidence to perform the behavior necessary to reach a desired goal,[57] whereas self-management refers to choosing to engage in behaviors to maintain wellness in the context of having a chronic disease.[58] Although each of these concepts is related to important components of self-regulation, they capture other aspects of adaptive functioning. A distinct concept—capacity to regulate—has been identified as an important component of maintaining healthy systems throughout the life course. Systems theories of health suggest that living systems need significant biological adaptability to maintain a harmonious equilibrium.[59,60] Moreover, no part of the system functions independently of the whole, and interactions between the multiple system components are regulated. Thus, appropriate regulation may lead to accruing resilience, whereby each protective asset gained leads to an increasing array of protective resources and greater adaptability.[60]

To our knowledge, our study is the first to consider the relation between a direct measure of self-regulation and CHD incidence. Findings from the present study are consistent with and extend previous work showing relations between healthy psychological functioning and cardiovascular health, in both magnitude and direction of the effects. Whether psychological capacity to regulate simply mirrors biological capacity or can actually alter biological functioning cannot be fully determined with the present study.[61] However, a number of plausible specific mechanisms can be identified by which self-regulation might influence CHD risk. A likely set of mechanisms involves enhancing capacity to engage in health-promoting behaviors. For example, individuals with poorer self-regulation are more likely to smoke or abuse other substances, whereas those with more capacity for self-regulation are less likely to overeat, to consume excess alcohol, and to smoke.[62] However, similar to other studies of healthy psychological functioning and CHD,[9] because health behaviors did not account for a significant portion of the association between self-regulation and incident CHD, we must also consider other potential pathways.

Individuals able to regulate effectively may have more opportunities for rest and restoration[11] and for regeneration, thereby maintaining a sense of positive energy. Positive physiological responses, including higher heart rate variability, more rapid return to basal heart rate, and recovery of cortisol response to stress, have been linked to adaptive psychological processes and positive traits.[63] Effective regulation may also facilitate one's ability to concentrate and solve problems or to mobilize social or other resources.[64] Given that emotion regulation is an important component of self-regulation, it is likely that effective regulation reduces the intensity or frequency of negative emotions often associated with chronic states of arousal. As a result, effective regulation may mitigate atherogenic processes by reducing activation of neuroendocrine, cardiovascular, and inflammatory systems.[11] Other work has found increased capacity to mitigate adverse biological reactions (eg, oxidative stress and cellular damage) with the relaxation response, a technique applied to enhance stress regulation.[65] Recent research has also focused on potential biological benefits of positive emotions.[66] Increasingly, investigators have argued that optimal functioning transcends the absence of distress and that what matters for health is the balance or capacity to regulate across the spectrum of emotion.[14,22,67] A greater focus on self-regulation as a higher-order attribute may help to understand why positive and negative affect matter in relation to cardiovascular health.

The present findings are limited in that they pertain to white men and thus cannot be generalized to women or to nonwhite populations. Moreover, the measure of self-regulation, although theoretically based, was derived for the present study, and we have no independent measures of its validity. However, reliability was strong, and it correlated with conceptually related measures in the expected ways. In fact, it is possible that there is residual confounding or that some unmeasured third variable (eg, a genetic factor) accounts for effective self-regulation and reduced risk of CHD. We controlled for known cardiovascular risk factors but did not have information on all relevant risk factors, such as dietary intake or physical activity. However, study strengths include a prospective design with objectively assessed CHD events, a long follow-up, and a range of coronary risk factors assessed. The magnitude of the effects found are substantial, with a 43% reduced risk of incident CHD (all outcomes combined) associated with the highest levels of self-regulation.

This study adds to the growing evidence base that healthy psychological functioning may have far-reaching benefits beyond those we traditionally identify. It highlights the importance of potentially shifting the focus from effects of negative psychological factors (or their absence) and considering positive assets in relation to cardiovascular health. Moreover, considering self-regulation in relation to health may help to organize the sometimes disparate literature linking aspects of both positive and negative psychological functioning to heart health. As a higher-order capacity integrally linked with healthy psychological functioning, self-regulation may provide individuals with the ability to respond effectively to an ever-changing environment.[11] The ability to self-regulate is due in part to early learning[35] and to prior and current social environmental conditions.[68] Although interventions to mitigate self-regulatory deficits have been developed,[69] whether interventions that broadly enhance self-regulation in adulthood can be developed and applied is yet to be determined. Some work has identified strategies for enhancing self-regulation in childhood. For example, psychological distancing, strategic distraction, and flexible deployment of attention have been found to increase ability to regulate in short-term experimental settings.[70-72] Other work has successfully developed techniques for teaching psychological distancing to adults to facilitate regulation of negative emotions.[73]

Findings from the current study may suggest a renewed consideration of self-regulation across the life course, with efforts directed at understanding its determinants, developmental trajectory, plasticity, and long-range consequences. A primary implication of these findings relates to prevention and intervention. For example, it may be helpful for clinicians to consider not only reducing psychological distress but also focusing on pro-

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moting positive functioning and capacity to regulate more broadly.[24] Additional work is needed to determine whether a focus on building or enhancing individuals' existing psychological resources may ultimately improve cardiovascular health.

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