



# Is the Glass Half Empty or Half Full? A Prospective Study of Optimism and Coronary Heart Disease in the Normative Aging Study

## Citation

Kubzansky, Laura D., David Sparrow, Pantel Vokonas, and Ichiro Kawachi. 2001. "Is the Glass Half Empty or Half Full? A Prospective Study of Optimism and Coronary Heart Disease in the Normative Aging Study." *Psychosomatic Medicine* 63 (6): 910–16. <https://doi.org/10.1097/00006842-200111000-00009>.

## Permanent link

<http://nrs.harvard.edu/urn-3:HUL.InstRepos:41288264>

## Terms of Use

This article was downloaded from Harvard University's DASH repository, WARNING: This file should NOT have been available for downloading from Harvard University's DASH repository.

## Share Your Story

The Harvard community has made this article openly available.  
Please share how this access benefits you. [Submit a story](#).

[Accessibility](#)

# Is the Glass Half Empty or Half Full? A Prospective Study of Optimism and Coronary Heart Disease in the Normative Aging Study

LAURA D. KUBZANSKY, PhD, DAVID SPARROW, DSc, PANTEL VOKONAS, MD, AND ICHIRO KAWACHI, MD

**Objective:** A sense of optimism, which derives from the ways individuals explain causes of daily events, has been shown to protect health, whereas pessimism has been linked to poor physical health. We examined prospectively the relationship of an optimistic or pessimistic explanatory style with coronary heart disease incidence in the Veterans Affairs Normative Aging Study, an ongoing cohort of older men. **Methods and Results:** In 1986, 1306 men completed the revised Minnesota Multiphasic Personality Inventory, from which we derived the bipolar revised Optimism-Pessimism Scale. During an average of 10 years of follow-up, 162 cases of incident coronary heart disease occurred: 71 cases of incident nonfatal myocardial infarction, 31 cases of fatal coronary heart disease, and 60 cases of angina pectoris. Compared with men with high levels of pessimism, those reporting high levels of optimism had multivariate-adjusted relative risks of 0.44 (95% confidence interval = 0.26–0.74) for combined nonfatal myocardial infarction and coronary heart disease death and 0.45 (95% confidence interval = 0.29–0.68) for combined angina pectoris, nonfatal myocardial infarction, and coronary heart disease death. A dose-response relation was found between levels of optimism and each outcome ( $p$  value for trend, .002 and .0004, respectively). **Conclusions:** These results suggest that an optimistic explanatory style may protect against risk of coronary heart disease in older men. **Key words:** coronary disease, optimism, pessimism, explanatory style, stress.

BMI = body mass index; CABG = coronary artery bypass graft; CAVE = content analysis of verbatim explanations; CHD = coronary heart disease; CI = confidence interval; LOT = Life Orientation Test; MI = myocardial infarction; MMPI-2 = revised Minnesota Multiphasic Personality Inventory; NAS = Normative Aging Study; PSM-R = revised Optimism-Pessimism Scale; RR = relative risk; SCL-90 = Symptom Checklist 90; SD = standard deviation.

## INTRODUCTION

Folk wisdom has extolled the health benefits of “thinking positively.” Although pessimism has been identified as a risk factor for poor psychological and physical health (1, 2), fewer studies have explored whether a sense of optimism, which derives from the ways individuals explain causes of both good and bad life events, may protect health. An optimistic explanatory style is characterized by the belief that the future will be pleasant because one can control important outcomes (3). A pessimistic explanatory style has been linked to a sense of hopelessness and is marked by the

view that problems are permanent and reflect one’s shortcomings (4, 5).

Previous research has suggested links between pessimism, hopelessness, and risk of heart disease (6–8). Other empirical work has also begun to suggest that generalized positive expectancies (also associated with optimism) are associated with better coronary health. For example, in one study of 54 patients recovering from coronary artery bypass graft (CABG) surgery, patients reporting higher levels of positive expectancies preoperatively demonstrated a faster rate of recovery after surgery (9). In a similar study of 309 patients undergoing CABG surgery, individuals with more positive expectancies were half as likely to be rehospitalized 6 months later for problems including postsurgical sternal wound infection, angina, myocardial infarction (MI), need for another bypass surgery, or angioplasty (10). Effects of positive expectancies were independent of sociodemographic and clinical variables as well as the diagnosis of depression. However, as yet no evidence of an association between an optimistic explanatory style and coronary heart disease (CHD) incidence has been presented.

In the present investigation we prospectively examined the effects of an optimistic versus a pessimistic explanatory style on CHD incidence in the Veterans Affairs Normative Aging Study (NAS), a longitudinal study of 2280 community-dwelling men. We hypothesized that relative to a pessimistic explanatory style, an optimistic one would lower CHD risk. Previous research has suggested that pessimism is associated with higher levels of negative emotions such as anger, anxiety, and depression (2, 11, 12). Because negative emotions may themselves be risk factors for CHD (13–16), we also examined whether a relationship between explanatory style and CHD might be largely explained

---

From the Department of Health and Social Behavior, Harvard School of Public Health (L.K., I.K.), and Channing Laboratory, Harvard Medical School (I.K., D.S.), Boston; Normative Aging Study, Department of Veterans Affairs Outpatient Clinic, and Department of Medicine, Boston University School of Medicine (D.S., P.V.), Boston, MA.

Address reprint requests to: Dr. L. Kubzansky, Department of Health and Social Behavior, Harvard School of Public Health, 677 Huntington Ave., Boston, MA 02115. Email: Lkubzans@hsph.harvard.edu

Received for publication August 15, 2000; revision received April 9, 2001.

## OPTIMISM AND CORONARY HEART DISEASE

by the association between pessimism and negative emotions.

### METHODS

The NAS is a longitudinal study of aging established by the Veterans Administration in 1961 (17). The study cohort consists of 2280 community-dwelling men from the Greater Boston area aged 21 to 80 years at the time of entry. Volunteers were screened at entry according to health criteria and were free of any known chronic medical conditions at the start of follow-up (17). Because men with known chronic conditions (including diabetes mellitus) were excluded at baseline, the cohort was originally composed of healthy individuals.

### Assessment of Optimistic Versus Pessimistic Explanatory Style

Optimism and pessimism are related to the ways in which people routinely explain events in their lives (5). Explanatory style theory suggests that the manner in which people explain causes of life events may vary in terms of internality, stability, and globality (18, 19). For example, individuals with a pessimistic explanatory style generally explain bad events as being caused by themselves (internal), due to chronic factors (stable), and generalizable to other situations (global), whereas good events are interpreted as external, transient, and due to specific causes. In contrast, individuals with an optimistic explanatory style are likely to explain bad events as due to external, transient, and specific causes, making opposite attributions for good events. Because pessimistic individuals expect bad events to occur consistently, they feel hopeless about changing the future (20).

We assessed optimism and pessimism in the NAS using the revised Optimism-Pessimism Scale (PSM-R) developed and validated by Malinchoc et al. (21). This bipolar scale measures explanatory style on a continuum from optimistic to pessimistic by using 263 items selected from the revised MMPI (MMPI-2). Based on explanatory style theory (22), the scale was developed using the content analysis of verbatim explanations (CAVE) technique, one of the two primary ways in which explanatory style may be measured (23). The CAVE technique may be used to evaluate the content of spontaneous written or verbal explanations to determine explanatory style, and it has been demonstrated to be valid and reliable (24, 25). Based on an assumption that MMPI statements endorsed as true are equivalent to spontaneous discourse, expert raters first classified which MMPI items were appropriate for evaluating causal explanation (21). Subsequently this pool of items was submitted to three independent raters, who evaluated each item for scores on internality, stability, and globality. Finally, a composite weight of the three scores was derived for each item, and the items (and their associated weights) were combined to derive optimism and pessimism scores, which were then normed. Further details about this scale have been described elsewhere (21). Scale scores in the present sample ranged from 15 to 76, and the internal consistency reliability of the scale was 0.86. Prior research suggests that this scale has a high test-retest reliability of 0.90 (19). A high score on this scale indicates a pessimistic explanatory style, whereas a low score indicates an optimistic one.

The MMPI-2 was administered by mail to all active cohort members ( $N = 1881$ ) in 1986 (26). A total of 1550 men responded (82.4% response rate), and complete and valid questionnaire data were available for 95% ( $N = 1472$ ). One hundred sixty-six men with preexisting CHD (angina pectoris or history of MI) were excluded,

resulting in a study population of 1306 men. Comparison of nonresponders with those who completed the MMPI-2 suggested that the nonresponders were somewhat younger and healthier (lower blood pressure, cholesterol, and body mass index [BMI]). The mean age of the study population was 60.8 years ( $SD = 8.3$  years, range = 40–90 years).

Both theory and empirical evidence from prior research suggest that the PSM should be correlated with negative emotions (27). As a check on construct validity, we found that anxiety, hostility/anger, and depression (measured with the Symptom Checklist-90 [SCL-90]) (28) were each positively correlated with the PSM-R ( $r$  values ranging from 0.41 to 0.53, all  $p$  values  $< .01$ ), whereas positive affect (measured with the Positive and Negative Affect Scale) (29) and happiness (measured with the happiness ladder) (30) were negatively correlated ( $r = -0.30$ ,  $p < .01$  and  $r = -0.42$ ,  $p < .01$ , respectively). As an additional check we correlated the PSM-R with the Life Orientation Test (LOT), another measure of optimism ( $r = -0.49$ ,  $p < .01$ ) (31).<sup>1</sup>

PSM-R scores were analyzed as a continuous variable. We also categorized PSM-R scores into terciles based on the distribution of scores in this cohort to assess the possibility of a threshold effect on CHD risk.

### 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. Cigarette smoking status (current, former, or never) is ascertained by a trained interviewer. Current smokers are defined as men who smoke  $\geq 1$  cigarette per day. Weight and height are measured with participants wearing only socks and underpants, from which BMI (weight/height<sup>2</sup>) is calculated. Blood pressure is measured by an examining physician using a standard mercury sphygmomanometer with a 14-cm cuff. With the subject seated, systolic blood pressure and fifth-phase diastolic blood pressures 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.

### Assessment of Morbidity and Mortality

This study included all confirmed CHD end points that occurred between the return of the 1986 survey and July 1998 with an average of 10 years of follow-up ( $SD = 2.7$  years). Individuals were censored either at the time of developing a coronary event (or death) or up to the time of their most recent follow-up visit. Of the total sample, approximately 82% were followed for a full 10 years.

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 (32). MI was diagnosed only when documented by unequivocal electrocardiographic changes (ie, pathologic Q waves), by a diagnostic elevation of serum enzymes (serum glutamic-oxalacetic transaminase and lactic dehydrogenase) together with chest

<sup>1</sup> We were unable to examine whether optimism as measured by the LOT was associated with CHD in this cohort because the measure was administered many years after the MMPI and too little follow-up time was available to conduct these analyses.

discomfort consistent with MI, or by autopsy. The diagnosis of angina pectoris was made by a board-certified cardiologist (P.V.) on the basis of medical history and physical examination using Framingham Heart Study criteria (32). Angina was diagnosed when a subject reported recurrent chest discomfort lasting up to 15 minutes distinctly related to exertion or excitement that was relieved by rest or nitroglycerin (32).

Death from CHD was designated when a death certificate, coded according to the eighth revision of the International Classification of Diseases (33), indicated an underlying cause of death coded to rubric 410 to 414. Medical records for each CHD death were reviewed by a board-certified cardiologist (P.V.) to ensure accurate coding. We routinely search state vital records as well as records of the Department of Veterans Affairs to pick up deaths that may have gone unreported by next of kin or postal authorities.

## Data Analysis

We ran Cox proportional hazards models using the Statistical Analysis System (34) to estimate the relative risks of CHD according to level of optimism or pessimism, controlling for age (years), BMI ( $\text{kg}/\text{m}^2$ ), smoking status (never, former, or current), systolic and diastolic blood pressure (mm Hg), serum cholesterol (mg/dl), family history of CHD (yes or no), whether participants drank two or more drinks of alcohol per day (yes or no), and whether participants had completed education beyond high school (yes or no). Age- and multivariate-adjusted analyses are presented to illustrate the role of potential confounders. Only 12 participants had diabetes in 1986. Results of analyses excluding men with diabetes were unchanged from those with the full sample; thus, analyses with the full sample are presented.

To test whether effects of optimism-pessimism simply reflect the presence of negative emotions like anxiety, depression, and anger, we ran models both excluding individuals who scored in the top 10% of one or any of these emotions, measured with the appropriate MMPI-2 content scale (26), and controlling for anxiety, anger/hostility, and depression as measured by the SCL-90. (The PSM-R and MMPI-2 content scales have overlapping items. As a result, MMPI-2 content scales were not used in this analysis because we were concerned about multicollinearity.)

## RESULTS

The mean optimism-pessimism score among 1306 subjects was 46.22 (SD = 10.52, range = 14.99–79.75), which was close to the mean value (mean = 50.0, SD = 10) in normative reference samples (21). Figure 1 shows the distribution of responses to the PSM-R scale in the entire cohort. Terciles were created on the basis of the distribution of scores in this sample, with cutoff points of 15 to 41, 41.1 to 49.9, and 50 to 79.7. We examined the distribution of coronary risk factors by level of optimism-pessimism (Table 1). Few differences emerged, although it is noteworthy that more pessimistic individuals were more likely to drink more than two drinks of alcohol a day and to have lower levels of educational attainment. We adjusted for all variables shown in Table 1 in the proportional hazards analyses.

Of the 1306 men, 162 developed CHD over the average 10-year follow-up period. There were 71 cases of

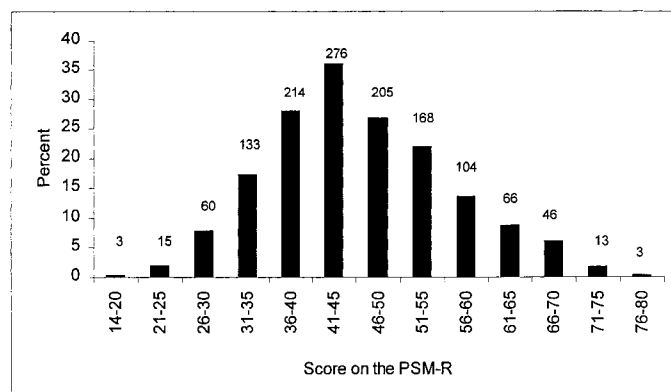


Fig. 1. Distribution of responses to PSM-R scale.

TABLE 1. Distribution of Coronary Risk Factors According to Level of Optimism-Pessimism<sup>a</sup>

| Risk Factor                                   | Level of Optimism-Pessimism          |   |                                     |
|---|--------------------------------------|---|-------------------------------------|
|   | Optimistic<br>15.0–41.0 <sup>b</sup> | Neutral<br>(Balanced)<br>41.1–49.9 <sup>b</sup> | Pessimistic<br>50–79.7 <sup>b</sup> |
| Number of subjects                            | 431                                  | 431   | 444                                 |
| Age, y  | 60.3                                 | 60.7  | 61.4                                |
| Current smokers, %                            | 28.5                                 | 33.8  | 37.7                                |
| Former smokers, %                             | 33.0                                 | 32.7  | 34.3                                |
| Systolic blood pressure, mm Hg                | 128.4 (15.7)                         | 128.5 (16.3)                                    | 128.2 (15.5)                        |
| Diastolic blood pressure, mm Hg               | 78.6 (8.0)                           | 78.4 (9.0)                                      | 78.4 (8.6)                          |
| Serum cholesterol level, mg/dl                | 246.1 (46.8)                         | 246.2 (44.4)                                    | 247.0 (47.0)                        |
| BMI, $\text{kg}/\text{m}^2$                   | 26.3 (2.8)                           | 26.6 (3.6)                                      | 26.8 (3.4)                          |
| Family history of CHD, %                      | 33.1                                 | 30.6  | 36.3                                |
| Education beyond high school, %               | 36.7                                 | 33.7  | 29.6                                |
| Consume $\geq 2$ drinks of alcohol per day, % | 34.1                                 | 26.4  | 39.5                                |

<sup>a</sup> Values in parentheses are SDs.

<sup>b</sup> Range of PSM-R score.

incident nonfatal MI, 31 cases of fatal CHD, and 60 cases of angina pectoris. In this cohort, the incidence rate of CHD was 0.10 over 10 years of follow-up. General incidence rates for total CHD were unavailable, although the incidence rate for MI found among white men for an equivalent length of time in other studies has been reported as 0.10 (eg, estimates from the Atherosclerosis Risk in Communities Study, 1987–1994) (35). In analyses using the PSM-R as a continuous variable, men with a more optimistic explanatory style had multivariate-adjusted relative risks (per 1-SD decrease in PSM-R) of 0.72 (95% CI = 0.57–0.90) for nonfatal MI, 0.70 for angina pectoris (95% CI = 0.55–0.90), and 0.75 (95% CI = 0.62–0.91) for total CHD.



## OPTIMISM AND CORONARY HEART DISEASE

When all end points were combined (nonfatal MI, fatal CHD, and angina pectoris), each SD increase in level of optimism was associated with an approximately 25% decreased risk of combined angina and total CHD (Table 2).

We also categorized the PSM-R into tertiles (Table 3). Relative to the most pessimistic men, the most optimistic men had multivariate-adjusted relative risks of 0.44 (95% CI = 0.26–0.74) for combined nonfatal MI and CHD death and 0.45 (95% CI = 0.29–0.68) for combined CHD death/nonfatal MI plus angina pectoris. Moreover, we found evidence of a dose-response relationship for each of these outcomes (multivariate *p*, trend = .002 and .0004, respectively). There was no evidence of an association between optimism and all-cause mortality (multivariate-adjusted RR for 1-SD increase in optimism = 0.94, 95% CI = 0.81–1.10).

We examined whether our findings might reflect the well-known relationship between pessimism and negative emotions given that various negative emotions (anxiety, depression, and anger) have been shown to increase CHD risk (13, 14, 36). When individuals scoring in the top decile of anxiety, anger, or depression were excluded from analyses, a 1-SD increase in opti-

**TABLE 2. Age- and Multivariate-Adjusted Relative Risks of a 1-SD Increase in Optimism<sup>a</sup>**

|  | Age-Adjusted RR  | Multivariate-Adjusted RR <sup>b</sup> |
|--|------------------|---------------------------------------|
| Angina pectoris                            |                  |                                       |
| Cases <sup>c</sup>                         | 60               | 58                                    |
| RR   | 0.71 (0.56–0.90) | 0.70 (0.55–0.90)                      |
| Nonfatal MI                                |                  |                                       |
| Cases <sup>c</sup>                         | 70               | 68                                    |
| RR   | 0.71 (0.57–0.89) | 0.72 (0.57–0.90)                      |
| Fatal CHD                                  |                  |                                       |
| Cases <sup>c</sup>                         | 31               | 30                                    |
| RR   | 0.71 (0.55–1.11) | 0.85 (0.59–1.23)                      |
| Total CHD <sup>d</sup>                     |                  |                                       |
| Cases <sup>c</sup>                         | 101              | 98                                    |
| RR   | 0.74 (0.61–0.89) | 0.75 (0.62–0.91)                      |
| Combined angina and total CHD <sup>e</sup> |                  |                                       |
| Cases <sup>c</sup>                         | 161              | 156                                   |
| RR   | 0.73 (0.63–0.85) | 0.74 (0.64–0.86)                      |

<sup>a</sup> Values in parentheses are 95% CIs.

<sup>b</sup> Adjusted for age, smoking status (never, former, or current), systolic and diastolic blood pressure (in mm Hg), serum total cholesterol level (mg/dl), BMI (kg/m<sup>2</sup>), family history of CHD (yes or no), educational attainment, and alcohol intake (≥2 drinks per day).

<sup>c</sup> A small number of cases (from 1 to 5 depending on outcome) and 33 noncases were dropped in multivariate analyses because of missing information on covariates.

<sup>d</sup> Nonfatal MI and fatal CHD.

<sup>e</sup> Angina combined with nonfatal MI and fatal CHD.

**TABLE 3. Age- and Multivariate-Adjusted Relative Risks of CHD According to Level of Optimism-Pessimism<sup>a</sup>**

| End Point                                  | Level of Optimism-Pessimism         |   |                                      |
|--|-------------------------------------|---|--------------------------------------|
|  | Pessimistic<br>50–79.7 <sup>b</sup> | Neutral<br>(Balanced)<br>41.1–49.9 <sup>b</sup> | Optimistic<br>15.0–41.0 <sup>b</sup> |
| Angina pectoris                            |                                     |   |                                      |
| Cases <sup>c</sup>                         | 28                                  | 20  | 12                                   |
| Age-adjusted RR                            | 1.00                                | 0.68 (0.38–1.20)                                | 0.40 (0.20–0.78)                     |
| Multivariate-adjusted RR <sup>d</sup>      | 1.00                                | 0.72 (0.39–1.30)                                | 0.42 (0.21–0.83)                     |
| Multivariate <i>p</i> , trend              | .003                                |   |                                      |
| Nonfatal MI                                |                                     |   |                                      |
| Cases <sup>c</sup>                         | 34                                  | 21  | 16                                   |
| Age-adjusted RR                            | 1.00                                | 0.58 (0.33–0.98)                                | 0.44 (0.24–0.79)                     |
| Multivariate-adjusted RR <sup>d</sup>      | 1.00                                | 0.59 (0.34–1.03)                                | 0.40 (0.21–0.75)                     |
| Multivariate <i>p</i> , trend              | .002                                |   |                                      |
| Fatal CHD                                  |                                     |   |                                      |
| Cases <sup>c</sup>                         | 15                                  | 10  | 6                                    |
| Age-adjusted RR                            | 1.00                                | 0.69 (0.31–1.53)                                | 0.43 (0.16–1.11)                     |
| Multivariate-adjusted RR <sup>d</sup>      | 1.00                                | 0.72 (0.31–1.70)                                | 0.54 (0.24–1.43)                     |
| Multivariate <i>p</i> , trend              | .19                                 |   |                                      |
| Total CHD <sup>e</sup>                     |                                     |   |                                      |
| Cases <sup>c</sup>                         | 49                                  | 31  | 22                                   |
| Age-adjusted RR                            | 1.00                                | 0.61 (0.39–0.96)                                | 0.44 (0.27–0.73)                     |
| Multivariate-adjusted RR <sup>d</sup>      | 1.00                                | 0.62 (0.39–0.98)                                | 0.44 (0.26–0.74)                     |
| Multivariate <i>p</i> , trend              | .002                                |   |                                      |
| Combined angina and total CHD <sup>f</sup> |                                     |   |                                      |
| Cases <sup>c</sup>                         | 77                                  | 51  | 34                                   |
| Age-adjusted RR                            | 1.00                                | 0.64 (0.45–0.92)                                | 0.44 (0.29–0.65)                     |
| Multivariate-adjusted RR <sup>d</sup>      | 1.00                                | 0.66 (0.46–0.95)                                | 0.45 (0.29–0.68)                     |
| Multivariate <i>p</i> , trend              | .0004                               |   |                                      |

<sup>a</sup> Values in parentheses are 95% CIs.

<sup>b</sup> Range of PSM-R score.

<sup>c</sup> A small number of cases (from 1 to 5 depending on outcome) and 33 noncases were dropped in multivariate analyses because of missing information on covariates.

<sup>d</sup> Adjusted for age, smoking status (never, former, or current), systolic and diastolic blood pressure (in mm Hg), serum total cholesterol level (mg/dl), BMI (kg/m<sup>2</sup>), family history of CHD (yes or no), educational attainment, and alcohol intake (≥2 drinks per day).

<sup>e</sup> Nonfatal MI and fatal CHD.

<sup>f</sup> Angina combined with nonfatal MI and fatal CHD.

mism was still associated with multivariate-adjusted relative risks of 0.67 (95% CI = 0.47–0.82) for combined nonfatal MI and fatal CHD and 0.61 (95% CI = 0.49–0.70) for combined nonfatal MI, fatal CHD, and angina pectoris. Similarly, results were largely unchanged when we simultaneously controlled for these emotions. The one slight difference was in the effects on angina; when emotions were controlled in the analysis, optimism-pessimism had a somewhat attenuated effect on angina (see Table 4).

## DISCUSSION

These prospective data are among the first to demonstrate that a more optimistic explanatory style, or

TABLE 4. Multivariate Relative Risks of a 1-SD Increase in Optimism Controlling for Negative Emotion<sup>a</sup>

|  | Multivariate-Adjusted RR <sup>b</sup> | RR Controlling for Anxiety <sup>b</sup> | RR Controlling for Anger/Hostility <sup>b</sup> | RR Controlling for Depression <sup>b</sup> |
|--|---------------------------------------|---|---|--|
| Angina pectoris                            |                                       |   |   |  |
| Cases                                      | 58                                    | 55                                      | 54  | 56   |
| RR   | 0.70 (0.55–0.90)                      | 0.78 (0.58–1.07)                        | 0.75 (0.56–1.01)                                | 0.78 (0.57–1.06)                           |
| Nonfatal MI                                |                                       |   |   |  |
| Cases                                      | 68                                    | 68                                      | 67  | 68   |
| RR   | 0.72 (0.57–0.90)                      | 0.62 (0.47–0.82)                        | 0.69 (0.53–0.90)                                | 0.61 (0.46–0.80)                           |
| Fatal CHD                                  |                                       |   |   |  |
| Cases                                      | 30                                    | 28                                      | 28  | 26   |
| RR   | 0.85 (0.59–1.23)                      | 0.65 (0.41–1.02)                        | 0.79 (0.52–1.20)                                | 0.70 (0.44–1.22)                           |
| Total CHD <sup>c</sup>                     |                                       |   |   |  |
| Cases                                      | 98                                    | 96                                      | 95  | 94   |
| RR   | 0.75 (0.62–0.91)                      | 0.63 (0.50–0.80)                        | 0.71 (0.57–0.89)                                | 0.63 (0.49–0.80)                           |
| Combined angina and total CHD <sup>d</sup> |                                       |   |   |  |
| Cases                                      | 156                                   | 151                                     | 149   | 150  |
| RR   | 0.74 (0.64–0.86)                      | 0.70 (0.58–0.84)                        | 0.73 (0.61–0.87)                                | 0.69 (0.57–0.84)                           |

<sup>a</sup> Values in parentheses are 95% CIs.

<sup>b</sup> Adjusted for age, smoking status (never, former, or current), systolic and diastolic blood pressure (in mm Hg), serum total cholesterol level (mg/dl), BMI (kg/m<sup>2</sup>), family history of CHD (yes or no), educational attainment, and alcohol intake ( $\geq 2$  drinks per day).

<sup>c</sup> Nonfatal MI, and fatal CHD.

<sup>d</sup> Angina combined with nonfatal MI and fatal CHD.

viewing the glass as half full, lowers the risk of CHD in older men. The protective effect seems independent of health behaviors like cigarette smoking or alcohol consumption. Given the bipolar nature of the PSM-R scale and its dose-response relationship with CHD risk, our findings could apply equally to increased CHD risk with greater pessimism. Although two other studies have found an association between an optimistic explanatory style and mortality (3, 37), this study did not. This discrepancy may be due to the fact that all the men in this population had access to health care; in addition, there was no indication of earlier health screening, as is the case with our NAS subjects. High access to medical care in a healthy population might be expected to attenuate the association between a risk factor and mortality while having less impact on the pattern of incident, nonfatal disease.

Psychologists suggest that optimism may allow individuals to mobilize highly effective coping resources (psychological, social, and behavioral) when confronted with adversity (1, 10, 38). Thus, explanatory style affects an individual's ability to adapt to a myriad of environmental demands and seems to be stable across time and situations (1, 2, 39). Some investigators have suggested that optimism and pessimism are indistinguishable from neuroticism and negative emotions more generally and that optimism may predict health simply because it reflects a broad disposition to experience positive as opposed to negative emotions and cognitions (40). A review of the literature on negative emotion and CHD *incidence* suggests that both

anxiety and anger are linked to increased risk, whereas the association with depression remains inconsistent (although the role of depression in the *prognosis* after MI is well established) (41). Our finding that the relationship between explanatory style and CHD risk persisted after excluding individuals reporting high levels of anxiety, anger, or depression (or after controlling for these emotions) provides support for the notion that optimism and pessimism are independent of negative emotions.

The biologic mechanisms by which an optimistic or pessimistic explanatory style may influence CHD are unclear, although several pathways are possible. Explanatory style may influence the degree of stress to which individuals are exposed. In turn, repeated episodes of stress may mobilize hemodynamic responses that cause or exacerbate endothelial damage and promote the acceleration of atherosclerosis (42–46). Because optimistic individuals actively engage in planning and problem solving, they may experience fewer stressors, or they may have more resources with which to deal with stress. Research has suggested that individuals with an optimistic explanatory style report less stress as measured by daily hassles than do their pessimistic counterparts during stressful tasks (6). In related research, individuals with more positive expectancies exhibit lower levels of cardiovascular reactivity (47, 48).

Explanatory style may also influence health through psychosocial pathways by promoting a sense of control, positive social interactions, and other health-en-

## OPTIMISM AND CORONARY HEART DISEASE

hancing behaviors (49, 50). For example, optimists tend to be less socially isolated (51). In addition, optimists are more likely to experience positive emotions (38). Fredrickson (52) has proposed a "broaden and build" model that posits that positive emotions build individuals' resources in multiple domains, including physical, cognitive, and social. These resources may in turn promote health. Positive emotions may also serve as an antidote to lingering effects of negative emotions (53). One study found that experimentally induced cardiovascular arousal dissipated more quickly when followed by positive versus negative feelings (53). Optimism has been found in other studies to be associated with increased likelihood of engaging in health-promoting behaviors such as avoiding smoking, exercising, and not drinking to excess (54). Because data on exercise frequency and diet were not available for most participants in 1986, we were unable to include these variables in the analyses.

The present study findings are limited in that they pertain to white men and thus cannot be generalized to women or to nonwhite populations. Moreover, the focus of this study is on optimism and pessimism as defined by explanatory style and measured by the PSM-R. This measure is relatively recently developed; as a result, extensive evidence on its relation to other measures of explanatory style is not yet available. Further work is needed to examine the construct validity of this measure. We also note that there is a separate literature that has looked at the relationship between health and dispositional optimism as measured by generalized positive expectancies. This literature has been largely separate from the explanatory style literature, but findings in both have been generally similar (50). However, Peterson (55) suggests that the two measures may tap related but distinct constructs, and a systematic examination of how they differ might yield important insight into resilience and health processes.

Explanatory style may arise partly as a consequence of being in a hostile or benign environment. It is of note that in this study, individuals with less education were significantly more likely to be pessimistic; factors like poverty, unemployment, or work stress may increase the likelihood of pessimism and thereby the risk of CHD. Further work is needed to determine whether optimism is one mechanism by which sociocultural factors affect cardiovascular health (though we adjusted for socioeconomic status in our study). An additional question is whether optimism may buffer the effects of poor social environments. Research has indicated that explanatory style is at least partially learned (19), and training programs that reliably increase optimism have been developed (56, 57). Should further research corroborate findings from the present study, a program of intervention may be warranted (20, 38, 57).

*This study was supported by grants from the National Heart, Lung, and Blood Institute HL45089 and from the National Institute on Aging, AG02287. The Normative Aging Study is supported by the Cooperative Studies Program/Epidemiologic Research and Information Centers (ERIC), Department of Veterans Affairs, and is a component of the Massachusetts Veterans Epidemiology Research and Information Center. Dr. Sparrow is a Research Career Scientist of the Medical Research Service of the Department of Veterans Affairs. Dr. Kawachi is supported by a Career Development Award from the National Heart, Lung, and Blood Institute and by the MacArthur Foundation Network on Socioeconomic Status and Health.*

## REFERENCES

1. Peterson C, Seligman MEP, Vaillant GE. Pessimistic explanatory style is a risk factor for physical illness: a thirty-five year longitudinal study. *J Pers Soc Psychol* 1988;55:23-7.
2. Peterson C, Seligman MEP. Causal explanations as a risk factor for depression: theory and evidence. *Psychol Rev* 1984;91:347-74.
3. Peterson C, Seligman MEP, Yurko KH, Martin LR, Friedman HS. Catastrophizing and untimely death. *Psychol Sci* 1998;9:127-30.
4. Jenkins CD. While there's hope, there's life: an editorial comment. *Psychosom Med* 1996;58:122-4.
5. Seligman MEP. *Learned optimism*. New York: Knopf; 1991.
6. Dykema J, Bergbower K, Peterson C. Pessimistic explanatory style, stress, and illness. *J Soc Clin Psychol* 1995;14:357-71.
7. Everson SA, Goldberg DE, Kaplan GA, Cohen RD, Pukkala E, Tuomilehto J, Salonen JT. Hopelessness and risk of mortality and incidence of myocardial infarction and cancer. *Psychosom Med* 1996;58:113-21.
8. Everson SA, Kaplan GA, Goldberg DE, Salonen R, Salonen JT. Hopelessness and 4-year progression of carotid atherosclerosis: the Kuopio Ischemic Heart Disease Risk Factor Study. *Arterioscler Thromb Vasc Biol* 1997;17:1490-5.
9. Scheier MF, Matthews KA, Owens JF, Magovern GJ Sr, Lefebvre RC, Abbott RA, Carver CS. Dispositional optimism and recovery from coronary artery surgery: the beneficial effects on physical and psychological well-being. *J Pers Soc Psychol* 1989;57:1024-40.
10. Scheier MF, Matthews KA, Owens JF, Schulz R, Bridges MW, Magovern GJ, Carver CS. Optimism and rehospitalization after coronary artery bypass graft surgery. *Arch Intern Med* 1999;159:829-35.
11. Marshall GN, Wortman CB, Kusulas JW, Hervig LK, Vickers RR Jr. Distinguishing optimism from pessimism: relations to fundamental dimensions of mood and personality. *J Pers Soc Psychol* 1992;62:1067-74.
12. Scheier MF, Carver CS, Bridges MW. Distinguishing optimism from neuroticism (and trait anxiety, self-mastery, and self-esteem): a reevaluation of the Life Orientation Test. *J Pers Soc Psychol* 1994;67:1063-78.
13. Kawachi I, Sparrow D, Spiro A, Vokonas P, Weiss ST. A prospective study of anger and coronary heart disease: the Normative Aging Study. *Circulation* 1996;94:2090-5.
14. Sesso HD, Kawachi I, Vokonas PS, Sparrow D. Depression and the risk of coronary heart disease in the Normative Aging Study. *Am J Cardiol* 1998;82:851-6.
15. Kubzansky LD, Kawachi I, Weiss S, Sparrow D. Anxiety and cor-

- onary heart disease: a synthesis of epidemiological, psychological, and experimental evidence. *Ann Behav Med* 1998;20:47–58.
16. Ford DE, Mead LA, Chang PP, Cooper-Patrick L, Wang N-Y, Klag MJ. Depression is a risk factor for coronary artery disease in men. *Arch Intern Med* 1998;158:1422–6.
  17. Bell B, Rose CI, Damon A. The Normative Aging Study: an interdisciplinary and longitudinal study of health and aging. *Int J Aging Hum Dev* 1972;3:5–17.
  18. Abramson LY, Seligman MEP, Teasdale JD. Learned helplessness in humans: critique and reformulation. *J Abnorm Psychol* 1978;87:49–74.
  19. Colligan RC, Offord KP, Malinchoc M, Schulman P, Seligman MEP. CAVEing the MMPI for an optimism-pessimism scale: Seligman's attributional model and the assessment of explanatory style. *J Clin Psychol* 1994;50:71–95.
  20. Kamen LP, Seligman MEP. Explanatory style and health. *Curr Psychol Res Rev* 1987;6:207–18.
  21. Malinchoc M, Offord KP, Colligan RC. PSM-R: revised optimism-pessimism scale for the MMPI-2 and MMPI. *J Clin Psychol* 1995;51:205–14.
  22. Seligman MEP. Helplessness: on depression, development, and death. San Francisco: Freeman; 1975.
  23. Schulman P, Castellon C, Seligman MEP. Assessing explanatory style: the content analysis of verbatim explanations and the Attributional Style Questionnaire. *Behav Res Ther* 1989;27:505–12.
  24. Peterson C, Luborsky L, Seligman MEP. Attributions and depressive mood shifts: a case study using the symptom-context method. *J Abnorm Psychol* 1983;92:96–103.
  25. Peterson C, Bettes BA, Seligman MEP. Depressive symptoms and unprompted causal attributions: content analysis. *Behav Res Ther* 1985;23:379–82.
  26. Butcher JN, Dahlstrom WG, Graham JR, Tellegen A, Kaemmer B. MMPI-2: Minnesota Multiphasic Personality Inventory-2. Manual for administration and scoring. Minneapolis: University of Minnesota Press; 1989.
  27. Buchanan GM, Seligman MEP. Explanatory style. Hillsdale (NJ): Erlbaum; 1995.
  28. Derogatis LR, Lipman RS, Rickels K, Uhlenhuth EH, Covi L. The Hopkins Symptom Checklist (HSCL): a measure of primary symptom dimensions. In: Pichot P, editor. Psychological measurement: modern problems in pharmacotherapy. Basel: S Karger; 1973.
  29. Watson D, Clark LA, Tellegen A. Development and validation of brief measures of positive and negative affect: the PANAS scales. *J Pers Soc Psychol* 1988;54:1063–70.
  30. Andrews FM, Robinson JP. Measures of subjective well-being. In: Robinson JP, Shaver PR, Wrightsman LW, editors. Measures of personality and social psychological attitudes. New York: Academic Press; 1991. p. 61–114.
  31. Scheier MF, Carver CS. Dispositional optimism and physical well-being: the influence of generalized outcome expectancies on health. *J Pers* 1987;55:169–210.
  32. Shurtleff D. Some characteristics related to the incidence of cardiovascular disease and death: Framingham Study, 18-year follow-up. Bethesda (MD): US Department of Health, Education, and Welfare; 1974.
  33. US Department of Health, Education, and Welfare. International classification of diseases adapted for use in the United States, eighth revision. Washington DC: US Department of Health, Education, and Welfare; 1963.
  34. SAS Institute. SAS/STAT user's guide. Cary (NC): SAS Institute Inc; 1990.
  35. American Heart Association. 2000 heart and stroke statistical update. Dallas: American Heart Association; 1999.
  36. Kawachi I, Sparrow D, Vokonas PS, Weiss ST. Symptoms of anxiety and risk of coronary heart disease: the Normative Aging Study. *Circulation* 1994;90:2225–9.
  37. Maruta T, Colligan RC, Malinchoc M, Offord KP. Optimists vs. pessimists: survival rate among medical patients over a 30-year period. *Mayo Clin Proc* 2000;75:140–3.
  38. Peterson C, Bossio LM. Health and optimism. New York: Free Press; 1991.
  39. Burns MO, Seligman MEP. Explanatory style across the life span: evidence for stability over 52 years. *J Pers Soc Psychol* 1989;56:471–7.
  40. Smith TW, Pope MK, Rhodewalt F, Poulton JL. Optimism, neuroticism, coping, and symptom reports: an alternative interpretation of the Life Orientation Test. *J Pers Soc Psychol* 1989;56:640–8.
  41. Kubzansky LD, Kawachi I. Going to the heart of the matter: do negative emotions cause coronary heart disease? *J Psychosom Res* 2000;48:323–37.
  42. Manuck SB, Krantz DS. Psychophysiologic reactivity in coronary heart disease and essential hypertension. In: Matthews KA, Weiss SB, Detre T, Dembroski TM, Falkner B, Manuck SB, editors. Handbook of stress, reactivity, and cardiovascular disease. New York: John Wiley & Sons; 1986. p. 11–34.
  43. Glagov S, Zarins C, Giddens DP, Ku DN. Hemodynamics and atherosclerosis. *Arch Pathol Lab Med* 1988;112:1018–31.
  44. Manuck SB, Kaplan JR, Adams MR, Clarkson TB. Studies of psychosocial influences on coronary artery atherosclerosis in cynomolgus monkeys. *Health Psychol* 1988;7:113–24.
  45. Cox DA, Vita JA, Treasure CB, Fish RD, Alexander RW, Ganz P, Selwyn AP. Atherosclerosis impairs flow-mediated dilation of coronary arteries in humans. *Circulation* 1989;80:458–65.
  46. Vita JA, Treasure CB, Ganz P, Cox DA, Fish RD, Selwyn AP. Control of shear stress in the epicardial coronary arteries of humans: impairment by atherosclerosis. *J Am Coll Cardiol* 1989; 14:1193–9.
  47. Williams RD, Riels AG, Roper KA. Optimism and distractibility in cardiovascular reactivity. *Psychol Rec* 1990;40:451–7.
  48. Krantz DS, Manuck SB. Acute psychophysiologic reactivity and risk of cardiovascular disease: a review and methodologic critique. *Psychol Bull* 1984;96:435–64.
  49. Robbins AS, Spence JT, Clark H. Psychological determinants of health and performance: the tangled web of desirable and undesirable characteristics. *J Pers Soc Psychol* 1991;61:755–65.
  50. Gillham JE, Shatte AJ, Reivich KJ, Seligman ME. Optimism, pessimism, and explanatory style. In: Chang EC, editor. Optimism and pessimism: implications for theory, research, and practice. Washington DC: American Psychological Association; 2000.
  51. Anderson CA, Arnoult LH. Attributional style and everyday problems in living: depression, loneliness, and shyness. *Soc Cogn* 1985;3:16–35.
  52. Fredrickson BL. What good are positive emotions? *Rev Gen Psychol* 1998;2:300–19.
  53. Fredrickson BL, Levenson RW. Positive emotions speed recovery from the cardiovascular sequelae of negative emotions. *Cogn Emotion* 1998;12:191–220.
  54. Peterson C. Explanatory style as a risk factor for illness. *Cogn Ther Res* 1988;12:117–30.
  55. Peterson C. The future of optimism. *Am Psychol* 2000;55:44–55.
  56. Hollon SD, Shelton RC, Loosen PT. Cognitive therapy and pharmacotherapy for depression. *J Consult Clin Psychol* 1991;59: 88–99.
  57. Beck AT, Rush AJ, Shaw BF, Emery G. Cognitive therapy of depression. New York: Guilford Press; 1979.