Personality, Disease Severity, and the Risk of Long-term Cardiac Events in Patients With a Decreased Ejection Fraction After Myocardial Infarction

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**Background**—Patients with myocardial infarction (MI) with a decreased left ventricular ejection fraction (LVEF) have a poor prognosis, but the role of emotional stress in prognosis is not known. We hypothesized that emotional stress in these patients (1) is unrelated to the severity of cardiac disorder, (2) predicts cardiac events, and (3) is a function of basic personality traits.

**Methods and Results**—Eighty-seven patients with MI (age, 41 to 69 years) with an LVEF of ≤50% underwent psychological assessment at baseline. Patients and their families were contacted after 6 to 10 years (mean, 7.9 years); cardiac events were defined as cardiac death or nonfatal MI. Emotional distress was unrelated to the severity of cardiac disorder. At follow-up, 21 patients had experienced a cardiac event (13 fatal events). These events were related to LVEF of ≤30%, poor exercise tolerance, previous MI, anxiety, anger, and depression (all *P* ≤.02). Patients with a distressed personality (type D; ie, the tendency to suppress negative emotions) were more likely to experience an event over time compared with non–type D patients (*P* = .00005). Cox proportional hazards analysis yielded LVEF of ≤30% (relative risk, 3.0; 95% confidence interval, 1.2 to 7.7; *P* = .02) and type D (relative risk, 4.7; 95% confidence interval, 1.9 to 11.8; *P* = .001) as independent predictors. Anxiety, anger, and depression did not add to the predictive power of type D; these negative emotions were highly correlated and reflected the personality domain of negative affectivity.

**Conclusions**—Personality influences the clinical course of patients with a decreased LVEF. Emotional distress in these patients is unrelated to disease severity but reflects individual differences in personality. Clinical trials should take a broad view of the target of intervention; assessment of LVEF and personality may identify patients at risk.

Key Words: myocardial infarction ▪ mortality ▪ risk factors ▪ prognosis

In recent years, several reports have indicated that anxiety,1,2 anger,3,4 depression,5,6 worry,7 and mental stress8 are associated with CHD, cardiac death, and myocardial ischemia. The similarity of results of these reports suggests that negative emotions in general are related to CHD, but there remains a tendency to focus on only one of these emotions at a time in this context. Accordingly, one generally assumes that depression is associated with a poor prognosis in post-MI patients.5 Hence, it is important to examine a broader scope of psychosocial factors than has previously been considered in relation to CHD.7 Personality, for example, may act as a third variable that promotes both emotional stress and CHD risk.19 Emotional stress as a CHD risk factor reflects a psychological characteristic6 and entails a whole spectrum of negative emotions1–16; “negative affectivity” is a basic personality trait that refers to this tendency to experience negative emotions.20 CHD patients who simultaneously tend to experience negative emotions and tend to inhibit self-expression (ie, “social inhibition,” another basic trait) are at risk for emotional distress21; patients with this “distressed” personality type (type D) have a high mortality risk compared with non–type D patients.22

Little is known of psychosocial factors in MI patients with a reduced LVEF. These patients have a poor prognosis,23,24 but more research is required to reliably predict which patients in particular are at risk for adverse cardiac events. The inverse of type A behavior has been related to mortality in these patients25; however, research on type A behavior has produced inconsistent findings.26
The present study examined the roles of emotions, behavior, and personality in the prognosis of patients with an LVEF of $\leq 50\%$ after MI; $50\%$ has been used by others as a cutoff to identify CHD patients with a poor prognosis.\textsuperscript{24} There is much debate regarding the extent to which emotional distress is caused by the severity of cardiac disorder.\textsuperscript{27} Hence, the purpose of this study was to examine the hypothesis that emotional distress in patients with an LVEF of $\leq 50\%$ after MI (1) is independent of the severity of cardiac disorder, (2) is an independent predictor of adverse cardiac events, and (3) is a function of basic traits, implying that type D accounts for any associations between negative emotions and cardiac events.

**Methods**

**Subjects**
A subset of 91 post-MI patients were selected from an original cohort.\textsuperscript{22} Patients were eligible for the present study if they had experienced a MI within the 2 months before entry into the study and had a global LVEF of $\leq 50\%$ as calculated from left ventricular angiography after MI; patients with another serious medical condition at baseline (eg, renal failure, cancer) were excluded. Four patients with an LVEF of $\leq 50\%$ died from noncardiac causes during follow-up (two of them had a type D personality) and were also excluded in the present study. Hence, the final sample in this study consisted of 87 post-MI patients (81 men and 6 women; age, 41 to 69 years; mean age, 55.1 years). All patients participated in the Antwerp Cardiac Rehabilitation Program between 1985 and 1988; enrollment in this outpatient program did not reflect the severity of cardiac disorder but rather the attitude of referring physicians toward rehabilitation. Standard medical care during the follow-up interval was similar for all patients and basically consisted of a routine cardiologic check-up every 6 months.

**Biomedical Factors**
Global left ventricular dysfunction and great extent of coronary obstructive disease are well-established risk factors for mortality after MI.\textsuperscript{24} Global left ventricular dysfunction is usually defined as LVEF of $\leq 20\%$ to $40\%$; we used LVEF of $\leq 30\%$ as a definition. A great extent of CHD was defined as three vessels with $\geq 70\%$ reduction in internal diameter. Biomedical risk assessment also included poor exercise tolerance, history of previous MI, anterior location of MI, no thrombolytic therapy after MI, no therapy with aspirin/ACE inhibitor/β-blocker at discharge from the rehabilitation program, poor compliance with the exercise regimen, and failure to quit smoking. A poor exercise tolerance was defined by a median split for peak workload (ie, $\leq 140$ W) on a bicycle exercise stress test 6 weeks after MI.

**Emotions and Behavior**
A cutoff at the 75th percentile on the State Anxiety Scale\textsuperscript{25} and Trait Anger Scale\textsuperscript{26} was used to classify patients as being high in anxiety (ie, $\geq 48$) and/or anger (ie, $\geq 22$). These scales have previously been associated with triggering of MI onset\textsuperscript{1} and progression of CHD,\textsuperscript{16} respectively. Patients were classified as being prone to depression if they scored above the median of both the “pessimism” (ie, $\geq 10$) and “despair” (ie, $\geq 12$) scales of the Millon Behavioral Health Inventory; these scales correlate .60 and .53 with the Beck Depression Inventory, respectively.\textsuperscript{27} Type B behavior (ie, the inverse of type A behavior, as characterized by relatively low levels of energy and arousal) was measured with a standardized interview.\textsuperscript{28} Evidence suggests that the emotional status of post-MI patients reflects basic personality traits;\textsuperscript{21,22} these traits were also assessed in the present study.

**Personality**
Although type A behavior is often mistaken for a personality type, this construct was designed to avoid association with global personality traits; in fact, type A reflects a “heterogeneous hodgepodge” of behavioral symptoms and signs\textsuperscript{29} without a conceptual basis in psychological theory.\textsuperscript{30} A more accurate approach would involve the delineation of more homogeneous subgroups on the basis of personality traits that contribute to affective styles and degree of stress reactivity.\textsuperscript{31} Accordingly, type D refers to a homogeneous subgroup that is defined by the interaction of two personality traits; ie, negative affectivity and social inhibition (as indicated by test scores above the medians for both these traits).

Negative affect can be observed either as a transient emotional state or as a persistent difference in general affective level. Negative affectivity is a trait that reflects the tendency to experience negative emotions across time and situations.\textsuperscript{32} This trait overlaps with neuroticism and trait anxiety\textsuperscript{33}; includes subjective feelings of tension, worry, anxiety, anger, and sadness\textsuperscript{30}, and has a major impact on the emotional status of CHD patients.\textsuperscript{34} Social inhibition reflects the tendency to inhibit the expression of emotions and behaviors in social interaction.\textsuperscript{35} Inhibited individuals feel insecure among other people, often lack assertiveness, may adopt self-enhancing strategies such as withdrawal, and are less talkative. Hence, the type D construct is embedded in psychological theory and is relevant to behavior in a large number of situations that deal with the nonexpression of negative emotions.

Negative affectivity was measured by the “trait” scale of the State-Trait Anxiety Inventory.\textsuperscript{28} This scale was originally developed to measure trait anxiety, but later evidence indicated that it actually taps negative affectivity\textsuperscript{36} and that it correlates .82 with neuroticism\textsuperscript{37} and .72 with depression.\textsuperscript{38} Most depression scales, including the Beck and Zung scales, have high correlations with negative affectivity.\textsuperscript{39} Social inhibition was measured by the “social inhibition” scale of the Heart Patients Psychological Questionnaire.\textsuperscript{40} As described previously,\textsuperscript{41} a median split on both of these scales was used to classify 27 patients as type D (ie, $\geq 43$ on the “trait” and $\geq 12$ on the “inhibition” scales) and 60 patients as not type D.

**Procedure and Analyses**
The design of this study can be summarized as follows. Basically, all patients filled out psychological questionnaires at entry in the Antwerp Cardiac Rehabilitation Program.\textsuperscript{22} After 6 to 10 years (mean, 7.9 years), patients and their families were contacted to determine the end point in this study; adverse cardiac events were defined as cardiac death or nonfatal MI. Cardiac death was used as a secondary end point in some additional analyses. Mortality data were derived from hospital records and discussed with the patient’s attending physician.

Unpaired t test was used to examine the association between psychosocial factors and severity of cardiac disorder. By analogy with previous research,\textsuperscript{1} baseline measures were dichotomized at points suggested in the literature, and the odds ratio for cardiac events for each pair of groups was assessed using logistic regression analysis and the $\chi^2$ statistic. Cumulative hazard functions and the Wilcoxon statistic were calculated to estimate the rate of cardiac events per year for type D patients and non-type D patients. Cox proportional hazards model was used to determine the best independent predictors of adverse cardiac events. Relative risk and 95% CI values were calculated for these predictors. The $\chi^2$ statistic was used to examine the relationship between type D and emotional stress. Pearson correlations were calculated among measures of personality and negative emotions; principal components analysis was used to examine the structural validity of these measures.

**Results**
No patients were lost to follow-up. After 6 to 10 years, 21 patients had experienced an adverse cardiac event (13 cardiac deaths). Patients in the present study had three times the risk of
cardiac events (21 of 87, or 24%) compared with patients from the Antwerp Cardiac Rehabilitation Program with an LVEF of 50% (15 of 195, or 8%) \( (P<.0001) \). Mean LVEF levels did not differ significantly as a function of anxiety, anger, type B behavior, or type D personality (Table 1); the association between depression and LVEF was marginally significant. Hence, self-reported levels of emotional distress at baseline were largely unrelated to the severity of cardiac disorder as measured by LVEF after MI.

### Emotional Distress and Disease Severity

To examine the extent to which emotional distress was caused by the severity of cardiac disorder, we analyzed the association between distress and LVEF after MI. Patients in this study had a significantly reduced LVEF (mean, 40±8%) compared with patients from the Antwerp Cardiac Rehabilitation Program with a preserved pump function (mean, 66±10%) \( (P<.0001) \). Mean LVEF levels did not differ significantly as a function of anxiety, anger, type B behavior, or type D personality (Table 1); the association between depression and LVEF was marginally significant. Hence, self-reported levels of emotional distress at baseline were largely unrelated to the severity of cardiac disorder as measured by LVEF after MI.

### Emotions, Personality, and Cardiac Events

Patients who experienced a cardiac event differed from event-free survivors in several biomedical and psychosocial characteristics at baseline that have previously been related to adverse health outcomes in coronary patients (Table 2). Adverse cardiac events were significantly associated with LVEF of <30% after MI, poor exercise tolerance, and history of previous MI and marginally significant with three-vessel disease and failure to quit smoking. Apart from these standard risk factors, cardiac events were also significantly associated with negative emotions in general, including symptoms of anxiety, anger, and depression.

Type D personality was associated with cardiac events in univariate analysis (Table 2); the rate of cardiac events was 52%
(14 of 27) for type D patients versus 12% (7 of 60) for non–type D patients. Examination of cumulative hazard functions confirmed that type D patients were more likely to experience a cardiac event over time than were non–type D patients (Fig 1). A Cox proportional hazards model was used to estimate the relative risk of cardiac events according to personality type, controlling for a range of potential confounding variables, including the standard risk factors of LVEF of ≤30%, three-vessel disease, poor exercise tolerance, history of previous MI, smoking after MI and negative emotions (anxiety, anger, depression). This model included LVEF of ≤30% (relative risk, 3.0; 95% CI, 1.2 to 7.7; P = .02) and type D personality (relative risk, 4.7; 95% CI, 1.9 to 11.8; P = .001) but not symptoms of anxiety, anger, or depression. Hence, (1) type D was a predictor of cardiac events after adjustment for the severity of cardiac disorder at baseline, and (2) specific emotions did not add to the predictive power of type D. However, given the small number of patients and end points, this analysis may have lacked statistical power to evaluate the independent contributions of specific negative emotions.

Secondary analyses indicated that patients who died from cardiac causes also differed from event-free survivors in several baseline variables. Cardiac death was associated with cardiac (LVEF of ≤30%, three-vessel disease, poor exercise tolerance, previous MI), emotional (anxiety, depression), and personality (type D) variables but also with type B behavior (Table 3). By analogy with total cardiac events as an end point, LVEF of ≤30% (P = .006) and type D personality (P = .0003) were retained as independent predictors of cardiac death; however, due to the relatively small number of fatal events, this finding should be interpreted with caution.

### Personality and Emotional Distress

These analyses indicated that type D personality was an independent predictor of adverse cardiac events in addition to standard risk factors. Symptoms of depression, anger, or anxiety, however, did not add significantly to the predictive power of type D. Accordingly, type D patients were likely to experience symptoms of anxiety, depression, and anger (Fig 2).

### TABLE 3. Significant Associations Between Baseline Characteristics and Long-term Cardiac Death

<table>
<thead>
<tr>
<th>Baseline Characteristic</th>
<th>Vital Status at 6 to 10 y</th>
<th>Odds Ratio (95% CI)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Event Free (n=66)</td>
<td>Cardiac Death (n=13)</td>
<td></td>
</tr>
<tr>
<td>Biomedical factor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEF ≤30%</td>
<td>11% (7)</td>
<td>46% (6)</td>
<td>7.2 (1.9 to 27.7)</td>
</tr>
<tr>
<td>Three-vessel disease</td>
<td>36% (24)</td>
<td>69% (9)</td>
<td>3.9 (1.1 to 14.2)</td>
</tr>
<tr>
<td>Poor exercise tolerance</td>
<td>42% (28)</td>
<td>92% (12)</td>
<td>16.3 (2.0 to 132.6)</td>
</tr>
<tr>
<td>Previous MI</td>
<td>20% (13)</td>
<td>54% (7)</td>
<td>4.8 (1.4 to 16.6)</td>
</tr>
<tr>
<td>Psychosocial factor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>24% (16)</td>
<td>54% (7)</td>
<td>3.7 (1.1 to 12.4)</td>
</tr>
<tr>
<td>Depression</td>
<td>42% (28)</td>
<td>85% (11)</td>
<td>7.5 (1.5 to 36.4)</td>
</tr>
<tr>
<td>Type B behavior</td>
<td>36% (24)</td>
<td>69% (9)</td>
<td>3.9 (1.1 to 14.2)</td>
</tr>
<tr>
<td>Type D personality</td>
<td>20% (13)</td>
<td>77% (10)</td>
<td>13.6 (3.3 to 56.5)</td>
</tr>
</tbody>
</table>

Number of subjects given in parentheses.

*Univariate analysis.
trait may add new prognostic information that is not being measured by standard distress scales. In keeping with this proposition, principal components analysis with varimax rotation yielded two personality factors (Table 4, right). Factor I (eigenvalue, 3.95; 66% variance) loaded on the negative affectivity, anxiety, depression, and anger scales and thus clearly represented the personality domain of negative affectivity. Factor II (eigenvalue, 1.07; 18% variance) had a high loading on the social inhibition scale but not on the negative affectivity scales.

Accordingly, post hoc analyses showed that social inhibition had a moderating effect on the relation between negative affectivity and prognosis; that is, the rate of cardiac events for patients who were high in negative affectivity but low in social inhibition was smaller than that for type D patients who were high in negative affectivity and social inhibition (1 of 13 [8%] versus 14 of 27 [52%], respectively; \( P = .01 \)), whereas these subgroups did not differ in negative affectivity (52.0 ± 7.8 versus 53.3 ± 7.7, respectively; \( P = .62 \)). Rate of cardiac events for high negative affectivity/low social inhibition patients did not differ significantly from that for low negative affectivity patients (6 of 47 [13%]), \( P = .52 \). Hence, negative affectivity was associated with adverse cardiac events only in patients who were high in social inhibition.

**Discussion**

The present findings indicated that global left ventricular dysfunction and type D personality were independent predictors of long-term cardiac events in patients with a reduced ejection fraction after MI. Other biomedical factors, such as poor exercise tolerance, history of previous MI, and, albeit to a lesser degree, three-vessel disease and smoking, were also associated with outcome in univariate analyses. Hence, these findings were consistent with those of previous studies reporting on biomedical risk factors in patients with CHD.22,24

The findings of this study are the first to suggest that emotions, behavior, and personality may matter in patients with a moderate to severe degree of cardiac disorder after MI. Self-reports of negative emotions, the interview rating of type B behavior, and the diagnosis of type D personality were unrelated to the extent of global left ventricular dysfunction. These findings add to a growing body of evidence suggesting that emotional distress in CHD patients is not related to the severity of cardiac disorder.37–39 It therefore is unlikely that individual differences in emotional distress are a proxy for the severity of cardiac disorder.

The findings from univariate analyses were consistent with those of prior studies showing that symptoms of anxiety,1,2,12 anger,1,4,16 and depression5,6,9 were associated with adverse cardiac events (type B behavior25 was related to only cardiac death in this study). Hence, negative emotions in general were predictive of cardiac events. As a matter of fact, a diversity of self-report measures have been used to document an association between emotional stress and CHD, including measures of depressive symptoms,5,9 phobic anxiety,1 state anxiety,3,12 state anger,1 trait anger,6,16 chronic distress,6 worry,7 general psychosocial stress,11,14 feelings of malaise,15,16 and hopelessness.80,41 Again, this observation suggests that emotional stress in general is related to CHD. The clinical picture of type D patients that emerged from the present analyses indicated that these patients are prone to anxiety, depression, and anger, suggesting that they are at risk for emotional stress in general. The estimated risk of cardiac events was 4.7 times greater for type D patients than for non–type D patients, with adjustment for severity of cardiac disorder.

Personality traits accounted for any associations between emotional stress and cardiac events in the present study; negative emotions did not add to the predictive power of type D. Personality refers to structures and processes that underlie regularities in emotions and behaviors. Accordingly, self-report measures of anxiety, depression, and anger correlated highly with each other and loaded on one personality factor reflecting negative affectivity. These findings confirm those of prior research in normal52 and CHD12,16 populations. Evidence also suggested that social inhibition was largely independent of negative emotions and had a moderating effect on the association between negative affectivity and prognosis. However, the inclusion of individual difference variables in life stress research is not currently a common practice.34 This is unfortunate because basic personality traits are more powerful than environmental factors in predicting emotional stress.52 In contrast, research on emotional stress and CHD has largely ignored the role of basic personality traits.

This paradox can perhaps best be understood in the light of previous type A research. The contradictory findings of this

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**TABLE 4. Correlations Between Measures of Personality and Negative Emotions**

<table>
<thead>
<tr>
<th>Correlation Matrix</th>
<th>Component Analysis</th>
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<tbody>
<tr>
<td></td>
<td>Factor I</td>
</tr>
<tr>
<td>1 Negative affectivity</td>
<td>...</td>
</tr>
<tr>
<td>1a Anxiety</td>
<td>.83*</td>
</tr>
<tr>
<td>1b Pessimism</td>
<td>.80*</td>
</tr>
<tr>
<td>1c Despair</td>
<td>.78*</td>
</tr>
<tr>
<td>1d Anger</td>
<td>.64*</td>
</tr>
<tr>
<td>2 Social inhibition</td>
<td>.22</td>
</tr>
</tbody>
</table>

Negative affectivity was measured with the "trait" scale of the State-Trait Anxiety Inventory; anxiety was measured with the "state" scale of the State-Trait Anxiety Inventory; pessimism and despair were measured with the Millon Behavioral Health Inventory; anger was measured with the "trait" scale of the State-Trait Anger Scale; and social inhibition was measured with the Heart Patients Psychological Questionnaire. 

*P < .01; †P < .05.

n = 87.
research caused prejudice against the role of personality in CHD (despite the fact that type A behavior is not a personality construct) and stimulated the search for specific emotional factors that may be related to CHD. However, a wide variety of negative emotions have been associated with CHD, implying that the situation is more complex than is conveyed by the notion that anger/hostility is a risk factor for the development and depression for the progression of CHD. Recent reports have suggested the importance of examining more stable and broader psychological constructs than have previously been considered in relation to CHD risk.

The stable and broad tendency to experience negative emotions (ie, negative affectivity) is one of the diagnostic criteria for type D personality; hence, the construct of type D summarizes research on emotion-related CHD. The present findings suggest that the interaction between (1) the tendency to experience negative emotions and (2) the tendency to inhibit self-expression (ie, social inhibition) creates a form of chronic psychosocial stress that may be detrimental to health in patients with a moderate to severe degree of CHD.

As noted earlier, the relatively small number of patients and events in the present study implies that the statistical power may have been too low to retain more than one psychosocial predictor variable in the Cox regression model. Undoubtedly, measures of specific emotions may yield significant prognostic information in CHD. The fact that the Trait Anxiety scale (ie, the measure of negative affectivity in the present study) was highly correlated with self-report scales of other negative emotions does not necessarily imply that these self-report scales are measuring the same construct; ie, negative emotions may be highly correlated but still may require different treatment approaches. Accordingly, CHD patients may be confronted with specific emotional disorders that deserve appropriate treatment. With reference to this issue, evidence suggests that patients who are not depressed in the hospital may develop major depression in the year after an MI and (2) that the clinical picture of major depression is dynamic and pleomorphic in nature. However, the present findings highlight the fact that in addition to assessing specific emotional factors, it is important to assess the broad and stable dimensions of normal personality in CHD. In other words, clinical diagnoses of affective disorder, self-report measures of negative emotions, and personality test scores may be independent predictors of adverse cardiac events, but this line of research has been too much neglected in the past. Thus, the most powerful prediction scheme is likely to be one that incorporates both biomedical and psychosocial factors, including specific emotions and global personality traits.

The mechanisms linking psychosocial stress to increased risk for cardiac events are not fully understood. Possible mechanisms include the induction of coronary spasm, increased platelet activity, or decreased heart rate variability. Hence, given their elevated level of chronic psychosocial stress, coronary patients with a type D personality may be prone to myocardial ischemia, ventricular arrhythmias, and cardiac events. Another possible mechanism entails that social inhibition, a basic characteristic of type D, may lead to less support seeking, which in turn may have an adverse effect on prognosis in post-MI patients. Future research must examine whether these or other mechanisms account for the link between personality and cardiac death in coronary patients.

The present findings should be interpreted with caution, but they are consistent with the underlying theoretical perspective on personality and CHD. This perspective has implications for clinical research and practice. First, clinical trials should take a broad view of the problem areas of stress and inhibition that may be relevant to high-risk patients with CHD. Antidepressant medication has been proposed as a treatment for these patients, but it is too soon to limit clinical trials to the narrow scope of depression; the diagnosis of major depression did not add to the predictive power of standard risk factors in post-MI patients. There is, however, evidence that comprehensive treatment programs may reduce mortality and emotional distress in CHD. Second, patients with a decreased LVEF after MI have a poor prognosis; inclusion of type D may help to predict which patients in particular are at risk. The combined assessment of LVEF and personality yielded high- and low-risk groups of post-MI patients, but these findings need to be replicated in confirmatory research.

The present findings are the first to suggest that psychological factors may influence the clinical course of coronary patients with a moderate to severe degree of cardiac disorder. These findings are provocative because they are based on a broad scope of psychological factors (including emotions, behavior, and personality) and a long-term follow-up interval. In conclusion, this research not only suggests that emotional distress in patients with an LVEF of ≤50% after MI (1) is independent of the severity of cardiac disorder and (2) may add to the predictive power of standard risk factors in terms of cardiac events but also that (3) personality factors may account for this relation between emotional distress and prognosis.

References


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