Type A Behavior, Personality Hardiness, and Cardiovascular Responses to Stress

Richard J. Contrada
Rutgers—The State University of New Jersey

Type A behavior and hardiness were examined as predictors of cardiovascular responses to stress in 68 male undergraduates. Systolic and diastolic blood pressure (SBP and DBP) and heart rate were monitored while subjects performed a difficult mirror-tracing task. Type A assessments based on the Structured Interview, but not those based on the Jenkins Activity Survey, were associated with significantly enhanced SBP and DBP elevations. Hardiness was associated with significantly reduced DBP responsiveness. In addition, a significant interaction indicated that the Type B–high hardiness group showed the least DBP reactivity. A near-significant interaction (p = .06) suggested that Type B–high hardiness subjects also reported the least anger. Further exploration of the data indicated that the challenge component of hardiness accounted for its relationship to DBP reactivity. These results have implications both for the psychophysiological study of Type A behavior and for understanding the health-promoting effects of hardiness.

Interest in the effects of personality on health and illness has grown considerably in recent years. One factor that has drawn attention to this topic is the extensive literature examining Type A behavior as a contributor to coronary heart disease (CHD; Houston & Snyder, 1988). Type A behavior consists of competitive achievement striving, hostility, impatience, and vigorous speech and motor mannerisms. Type B refers to the relative absence of these characteristics and a more relaxed style of coping. Initially, epidemiological work provided rather consistent evidence that individuals exhibiting Type A behavior show a greater risk of developing CHD than their Type B counterparts (Cooper et al., 1981). However, more recent studies have failed to confirm earlier findings (Shekelle, Gale, & Norusis, 1985; Shekelle, Hulley, et al., 1985).

Two recent meta-analyses have helped to clarify the status of Type A behavior as a coronary risk factor. The first (Booth-Kewley & Friedman, 1987) included both cross-sectional and prospective studies and suggested that Type A behavior is reliably associated with CHD. The second meta-analysis (Matthews, 1988) differed in that it focused exclusively on prospective studies, had access to more recent findings, and used different decision rules in aggregating data across studies. Results indicated that Type A behavior predicts CHD (a) when Type A is assessed using a Structured Interview (SI; Dembroski, 1978; Rosenman, 1978), but not when measured using the Jenkins Activity Survey (JAS; Jenkins, 1978; a self-administered questionnaire) and (b) in population-based studies that examined initial CHD in subjects who were healthy at intake, but not in studies of high-risk individuals.

As noted by Friedman and Booth-Kewley (1988), the two meta-analyses agree in supporting the hypothesis that Type A behavior is related to CHD but raise several issues regarding the nature of that relationship. Most germane to the present study are questions about the magnitude of the Type A–CHD relationship. Even in population-based studies in which Type A was assessed by Structured Interview, results indicate only a modest association with CHD. The Type A variable identifies as coronary prone many individuals who do not develop CHD and as noncoronary prone many individuals who do.

Efforts to increase predictive validity have primarily taken the approach of evaluating separately the component behaviors reflected in global Type A assessments based on the SI. This has led to the discovery that hostility and other anger-related variables may constitute the “toxic” elements of Type A behavior (Dembroski & Costa, 1987). Less attention has been given to the study of Type A behavior in conjunction with other personality factors that may contribute to coronary risk. The Matthews (1988) and Booth-Kewley and Friedman (1987) meta-analyses identified several personality constructs that appear promising in this regard, given their relationships to CHD in studies in which Type A was not measured.

The epidemiological literature on Type A behavior has stimulated a considerable amount of research examining mechanisms that may explain the relationship between Type A and CHD. This work is based on the hypothesis that coronary risk is enhanced by cardiovascular and other physiological responses associated with psychological stress and that these responses are more pronounced in Type A individuals. Emerging from this research are three trends that parallel epidemiological findings: (a) Psychophysiological research has more often yielded positive results where Type A is assessed by SI rather than by questionnaire (Contrada, Wright, & Glass, 1985); (b) reported associations account for what is, at best, only a small proportion of
Further investigation of hostility and other components of Type A may determine more precisely which aspects of the behavior pattern are most strongly associated with physiological responses that may promote CHD. However, as in the prediction of CHD, it also might be useful to examine Type A behavior in the context of other personality characteristics, particularly those that themselves show a relationship to health. It is reasonable to hypothesize that personality characteristics that are conceptually distinct from Type A behavior and its components, but that also bear a relationship to physical health or illness, may account for some of the variance in disease and in disease-promoting physiological responses that is left unexplained by Type A.

One variable worth examining in this regard is hardiness, a personality dimension that is believed to confer resistance against the effects of psychological stress (Kobasa, 1979). Hardiness is a composite consisting of internal locus of control (vs. powerlessness), commitment (vs. alienation), and challenge (vs. threat). In a series of retrospective (Kobasa, 1979, 1982; Kobasa, Maddi, & Courington, 1981) and prospective (Kobasa, Maddi, & Kahn, 1982) studies, results were obtained that suggest that hardiness may exert a protective effect against reported illness.

Recent studies have raised both conceptual and methodological criticisms regarding hardiness research (e.g., Funk & Houston, 1987; Hull, Van Treuren, & Vrinielli, 1987). One concern is whether hardiness embodies a unitary construct, as originally proposed, or a set of three independent dimensions. Another is that hardiness appears to promote health independently of stress, rather than operating as a buffer by reducing illness primarily among individuals experiencing high stress levels (Cothen & Edwards, 1989; Funk & Houston, 1987; Hull et al., 1987). These issues typically have been investigated in research oriented toward the prediction of adaptive outcomes, such as psychological distress or reports of physical illness.

Relatively little effort has been devoted to identifying mechanisms that may mediate the effects of hardiness on health outcomes. There is evidence to suggest that hardy individuals perceive stressful events as more positive and controllable than individuals low in hardiness (Allred & Smith, 1989; Rhodeart & Augustsdottir, 1984; Rhodeart & Zocn, 1989). Thus it is possible that hardiness short-circuits the stress response by influencing the processes whereby psychological threat is appraised. However, it has yet to be demonstrated that hardy individuals show reduced physiological responsiveness when confronted by laboratory stressors.

It is the question of whether hardiness is related to physiological responses to stress that forms a basis for the present study. To the degree that hardiness protects against the development of physical disease through mechanisms associated with psychological stress, it may be predicted that individuals high in hardiness will show less pronounced physiological responses to a stressor than those low in hardiness. In the absence of such a finding, pathways to disease that do not necessarily involve stress, such as cigarette smoking, poor diet, and failure to detect and/or act on physical symptoms, might be regarded as more plausible explanations for data suggesting that hardiness is health promoting. Since Type A behavior appears to be associated with enhanced physiological reactivity, it would be useful to determine whether hardiness contributes to the prediction of reactivity independently of any association between hardiness and Type A. To the extent that this is the case, it would suggest that research on the psychophysiology of Type A behavior might benefit from a consideration of hardiness and, perhaps, other personality characteristics that influence the stress response.

Accordingly, a psychophysiological experiment was conducted to assess the independent and conjoint effects of Type A behavior and hardiness on subjective and cardiovascular responses to a frustrating psychomotor task. Previous work does not provide a specific basis for predicting an interaction between Type A behavior and hardiness. Therefore, it was expected that Type A behavior would be positively associated with physiological reactivity to the task, and that hardiness would be inversely associated with reactivity, such that physiological responses would be greatest among Type A subjects low in hardiness, and lowest among Type B subjects high in hardiness. It also was predicted that Type A effects based on SI assessments would be stronger than those based on the JAS.

**Method**

**Subjects**

Subjects were 68 male introductory psychology students who participated in partial fulfillment of course requirements. Subjects ranged in age from 18 to 22 years (M = 21.1).

**Measurement of Type A Behavior and Hardiness**

The principal measure of Type A behavior was the student version of the SI, administered by the author, who was trained in the technique by Ray H. Rosenman. The interviews were tape-recorded and reviewed independently by an auditor who had been trained by Theodore M. Dembroski. Behavior pattern assessments were made using a 4-point scale (A1 = 4, A2 = 3, X = 2, and B = 1). Degree of agreement between the two raters' discrete classifications was 75%. Intercorrelation of the 4-point ratings yielded a value of .87. The 4-point ratings were averaged to construct a continuous Type A score for use in correlational and regression analyses. For descriptive purposes, a dichotomous measure of Type A behavior was derived by combining discrepant ratings, with the use of a procedure recommended by MacDougall, Dembroski, and Mustane (1979): A1/A2 and A/X = A; B/X = B (there were no A–B disagreements, nor did any subject receive a Type X classification from both raters). This yielded 35 Type As and 33 Type Bs. Subjects also completed Form T of the JAS (Krantz, Glass, & Snyder, 1974).

Hardiness was assessed through the use of five questionnaires. The Alienation From Work and Alienation From Self scales (Maddi, Kobasa, & Hoover, 1979) were used to measure commitment. The External Locus of Control Scale (Rutter, Seeman, & Liverant, 1962) and Powerlessness Scale (Maddi et al., 1979) were used to measure control. The Security scale of the California Life Goals Evaluation Schedule (Hahn, 1966) was used to measure challenge. For each of these measures, high scores reflect a relative lack of hardiness. Following procedures described by Kobasa et al. (1982), the appropriate scale scores were standardized and then summed to create measures of commitment and control. Because challenge was measured by only one scale (Security), its scores were doubled before adding together the five standardized scores.
Affective Measures

Affective responses to task performance were measured using the State-Trait Personality Inventory (Spielberger, Jacobs, & Russell, 1983), which contains three 10-item subscales: Anger, Anxiety, and Curiosity. Instructions requested that the subject respond with reference to his feelings while working on the mirror-tracing task. The Anger and Anxiety scales were used to assess subjective distress. The Curiosity scale was included to detect positive appraisals of the task situation, such as might be expected from Type Bs or subjects high in hardiness. It contains items reflecting interest, desire to explore, and inquisitiveness.

Procedure

Subjects participated individually in two sessions. At the first session, I greeted the subject and escorted him to a small office where the purpose of the study was described as an attempt to determine the effects of personality on subjective and physiological correlates of psychomotor activity. Informed consent was obtained at this point. The experimenter then administered the SI. Following the interview, the JAS and hardiness questionnaires were completed in an order individually randomized for each subject. An appointment for the second session then was scheduled, and the subject was thanked and dismissed.

Session 2 was conducted by a second male experimenter who was unaware of the hypotheses under study as well as the subject's Type A assessment and hardiness scores. The subject was escorted to a sound-attenuated experimental room, where the experimenter reiterated the purposes of the study. A baseline period ensued, during which time the subject was asked to sit quietly and relax. Measurements of SBP, DBP, and HR were taken at 2-min intervals, until SBP values remained relatively stable (+5 mmHg) across two successive readings, after a minimum of 8 min had elapsed. Means for the final two readings on each cardiovascular measure were taken as baseline values.

Following the baseline period, the experimenter reentered the experimental room, placed the mirror-tracing task on the table, and gave the subject a single sheet of paper containing task instructions. The experimenter then left the room, after explaining that any further communication would take place over an intercom system. Instructions described the mirror-tracing task as a measure of spatial ability and hand-eye coordination, and indicated that the subject was to attempt to complete at least one tracing, without activating the buzzer, within a 5-min period. It was stated that "about 50%" of the participants participating in the project had been able to do this, and that the main concern of the investigators was that the subject give his best effort. These instructions were developed on the basis of research indicating that Type As are most likely to show enhanced physiological activity in response to tasks characterized by moderate levels of difficulty (Contrada et al., 1985).

The instructions went on to request that the subject "think aloud" while working on the task. The subject was encouraged to verbalize whatever thoughts and feelings he might experience, whether or not they were related to the immediate situation. It was explained that these verbalizations would be used to determine the information processing style used by the subject in working on the task.1

When the subject was ready to proceed, the experimenter activated a tape recorder (connected to the hidden microphone serving the intercom system from the experimental room) and then instructed the subject to begin working. Blood pressure and HR measurements were initiated 5 s later, and every 60 s thereafter, for the next 5 min. After recording the final measurements, the experimenter instructed the subject to stop working, reentered the experimental room, and administered the postexperimental questionnaire. Following completion of the questionnaire, the first experimenter conducted a postexperimental interview and debriefing.

Data Reduction and Analysis

The principal mode of statistical analysis was multiple regression analysis, since both predictors (Type A and hardiness) and dependent measures (cardiovascular activity and affect) reflect continuous dimensions. However, in order to maintain comparability with most previous research on Type A, which has taken an analysis of variance approach,1...
significant regression effects were described by reporting means for groups formed on the basis of the dichotomous Type A and hardiness measures described earlier. Crossing the dichotomous Type A and hardiness classifications yielded the following numbers of subjects in each category: Type A–high hardiness, 21; Type A–low hardiness, 14; Type B–high hardiness, 13; Type B–low hardiness, 20.

As in previous research, physiological reactivity was measured as the difference between baseline and task values for each cardiovascular measure. Preliminary analysis indicated that reactivity on each of the cardiovascular measures showed no reliable changes over the five task readings, and that this pattern held, regardless of SI, JAS, and hardiness scores (ps > .20). Therefore, a single index of reactivity was computed for each variable by averaging across the five change scores. Baseline values from which change scores were derived were included in the regression model in order to remove variance attributable to initial values (Wilder, 1968).

Results

Intercorrelations Among Type A and Hardiness Measures

Intercorrelations among the Type A and hardiness measures are presented in Table 1. The SI and JAS showed a moderate degree of association (r = .38, p < .01). Relationships among the hardiness components were similarly modest in magnitude (rs ranging from .28 to .49, ps < .05). Intercorrelations between the SI and each of the hardiness components, as well as the composite hardiness index, were low and nonsignificant (rs ranging from -.18 to -.06). The JAS showed a similar dissociation with hardiness (rs from -.18 to -.02), except for a significant relationship with control (r = -.28, p < .05), indicating that subjects scoring in the Type A direction tended to report feeling capable of influencing the events in their lives.

Table 1 also presents the internal consistencies of the Type A and hardiness measures. Reliability coefficients for all measures are acceptable, except for Challenge (Cronbach’s α = .56).

Resting Cardiovascular Activity

A series of regression analyses was conducted to determine the relationships between Type A and hardiness and cardiovascular baselines. The only significant effect was a Type A × Hardiness interaction for resting SBP values, F(1, 64) = 4.54, p < .05. Among Type Bs, subjects high in hardiness had a lower resting SBP than those low in hardiness (Ms = 98.4 mmHg and 105.2 mmHg, respectively); by contrast, among Type As, those high in hardiness had a higher resting SBP than those low in hardiness (Ms = 109.8 mmHg and 101.7 mmHg, respectively). No other effect approached significance for any of the cardiovascular measures (ps > .18).

Cardiovascular Reactivity

As expected, a regression analysis for SBP change scores yielded a significant main effect for Type A, F(1, 63) = 4.97, p < .03. Type As showed significantly greater reactivity than Type Bs (Ms = 20.1 mmHg and 15.2 mmHg). Whereas subjects low in hardiness tended to have higher SBP elevations than those high in hardiness (Ms = 19.1 mmHg and 16.3 mmHg), neither the hardiness main effect nor the Type A × Hardiness interaction was significant (p = .40).²

Also consistent with predictions was a Type A main effect for DBP change scores, F(1, 63) = 6.54, p < .02. As in the case of SBP, Type As showed significantly greater DBP responses than their Type B counterparts (Ms = 14.5 mmHg and 10.9 mmHg, respectively). There was also a hardiness main effect, F(1, 63) = 7.54, p < .01. As expected, DBP elevations were significantly higher among subjects with low hardiness scores compared with those high in hardiness (Ms = 14.0 mmHg and 11.1 mmHg, respectively). The two main effects were qualified by a Type A × Hardiness interaction, F(1, 63) = 4.22, p < .05. The relevant data are depicted in Figure 1. It can be seen that the interaction reflected particularly low DBP elevations among Type B subjects with high hardiness scores, the group expected to be least reactive.

An analysis of HR change scores yielded only a nonsignificant Type A main effect.

Table 1 Internal Consistency and Intercorrelations of the Hardiness and Type A Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Challenge</td>
<td>(.56)</td>
<td>.28*</td>
<td>.34**</td>
<td>.74**</td>
<td>-.02</td>
<td>-.06</td>
</tr>
<tr>
<td>Commitment</td>
<td>(.75)</td>
<td>.49**</td>
<td>.76**</td>
<td>-.14</td>
<td>-.18</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>(.83)</td>
<td>.78**</td>
<td>-.28*</td>
<td>-.11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hardiness Composite</td>
<td>(.86)</td>
<td>-.18</td>
<td>-.15</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jenkins Activity Survey</td>
<td>(.73)</td>
<td>.38**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Structured Interview</td>
<td>(.87)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. Cronbach alpha coefficients are given in parentheses, except for the Structured Interview, where the interrater correlation is presented. * p < .05. ** p < .01.
Three Regression Models Predicting DBP Reactivity From Type A and Hardiness

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Model 1: Hardness composite&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Model 2: Three hardness components&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Model 3: Five hardness scales&lt;sup&gt;c&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type A</td>
<td>.38*</td>
<td>.31*</td>
<td>.31**</td>
</tr>
<tr>
<td>Challenge</td>
<td>.82**</td>
<td>.76*</td>
<td>.88**</td>
</tr>
<tr>
<td>Type A × Hardness</td>
<td>-.62*</td>
<td>Type A × Challenge</td>
<td>-.70*</td>
</tr>
</tbody>
</table>

Note: The regression models are described in the text. Only significant terms are presented in the table. Not shown are regression results for DBP baseline values, which were significant in each model.  
<sup>a</sup>F(4, 63) = 6.09, p < .0005, corrected R^2 = .23.  
<sup>b</sup>F(8, 59) = 3.71, p < .002, corrected R^2 = .24.  
<sup>c</sup>F(12, 55) = 2.82, p < .005, corrected R^2 = .25.

Effects of Type A and Hardiness on Affective Measures

Regression analysis of State Anger scores yielded a reliable hardness main effect, F(1, 63) = 4.13, p < .05, and a near-significant Type A × Hardiness interaction, F(1, 64) = 3.68, p = .06. As reflected by the marginal interaction, the hardness main effect was carried primarily by the Anger scores of the Type B—high hardness group (M = 14.0), which were lower than those of Type B—low hardness subjects (M = 16.0) and the two Type A groups (both M = 16.9). No other effects approached significance for any of the state affect scales (ps > .10).

Correlations between Anger ratings and cardiovascular change scores only approached significance in the case of DBP (r = .19, p = .11). This suggests that the effects of Type A behavior and hardness on anger were largely independent of the reactivity effects.

Hardiness Components and Cardiovascular Reactivity

In order to explore the relative contributions of the individual components of hardness in predicting DBP reactivity, two additional multiple regression analyses were carried out. One included the following predictors: DBP baseline values, SI Type A assessments, individual scores on the five scales that make up the hardness composite, and product terms reflecting interactions between Type A ratings and each of the hardness scales.

The second analysis differed in that the External Locus of Control and Powerlessness scales were combined into a single measure of control, and the Alienation from Self and Alienation from Work scales were combined into a single measure of commitment. The Security scale was included as a measure of challenge and, as in the first series of regressions, product terms were constructed to reflect the interaction between Type A and each hardness component. Thus, the two regression analyses allow a comparison of two levels of decomposition of the overall hardness variable: One resolves hardness into measures of its three constituent constructs, the other into five individual scale scores.

Significant effects are presented in Table 2, which also summarizes the initial regression analysis that used the overall hardness index. Because the number of predictors in the models varies, eachcapitalizes on chance to a different degree. Therefore, the R^2 coefficients were adjusted using the shrinkage correction formula described by Olkin and Pratt (1958). Inspection of these data suggests a number of conclusions. First, after correction for shrinkage, the three models are shown to account for a similar, statistically significant portion of the variability in DBP responses to the task. Second, each model contains a term reflecting a hardness effect that adds significantly to the prediction of DBP responses above and beyond the contribution of Type A behavior. Third, the relationship between hardness and DBP responses largely reflects a single hardness component, challenge. Whether the remaining four scales are considered individually (Table 2, Model 1), combined into control and commitment scores (Table 2, Model 2), or summed together with challenge scores to generate a single composite index of hardness (Table 2, Model 1), they add little to the ability of the challenge component to predict individual differences in DBP responses to the task. The only exception appeared in Model 2 in the form of a nonsignificant trend toward a main effect for the control component (p = .08).

Table 3 presents simple correlations between control, challenge, and overall hardness and DBP change scores. It can be seen that the pattern of results for challenge resembles that of the composite hardness index. High scores (indicating low challenge) were associated with heightened DBP reactivity to the task, and this relationship is evident among Type Bs but not among Type As. Similarly, DBP reactivity tended to be greater

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Type A</th>
<th>Type B</th>
<th>Combined</th>
</tr>
</thead>
<tbody>
<tr>
<td>Challenge</td>
<td>-.06</td>
<td>.45***</td>
<td>.19</td>
</tr>
<tr>
<td>Control</td>
<td>.13</td>
<td>.38**</td>
<td>.21*</td>
</tr>
<tr>
<td>Hardiness composite</td>
<td>.11</td>
<td>.44**</td>
<td>.22*</td>
</tr>
</tbody>
</table>

Note. N = 68 (33 Type B and 35 Type A).

* p < .10. ** p < .05. *** p < .01.
among subjects who reported a lack of control, and this relationship was also somewhat stronger among Type Bs than Type As.

Discussion

The results of this study suggest the following observations regarding the role of Type A behavior and personality hardiness in accounting for physiological responses to stress. First, the data are consistent with previous studies demonstrating that Type A assessments based on the SI show a modest but reliable association with cardiovascular reactivity. Second, individual differences in reactivity that are left unexplained by SI Type A assessments are significantly related to other aspects of personality encompassed by the term hardiness. Third, an exploration of the relationship between hardness and cardiovascular responses to stress supports the utility of examining separately the individual characteristics that make up the hardness composite. In particular, the stress-dampening effects of hardiness in the present study were largely attributable to the challenge component.

Type A Behavior and Cardiovascular Reactivity

As expected, Type A assessments based on the SI were associated with enhanced SBP and DBP responses to task performance. The HR data yielded only a nonsignificant tendency toward greater reactivity among Type As ($p = .10$). By contrast, the JAS was unrelated to reactivity. These results are consistent with the preponderance of previous findings demonstrating that the SI is more strongly associated with physiological reactivity than the JAS (Contrada et al., 1985). Moreover, as noted earlier, these reactivity data are congruent with epidemiological findings that support the validity of SI assessments in the prediction of CHD in population-based studies, but draw into question the status of the JAS as a measure of coronary-prone behavior (Matthews, 1988). In view of these observations, it would seem incumbent on the investigator who relies on the JAS as a measure of Type A behavior to justify its use on some basis other than its relevance to CHD.

That Type A effects were obtained for SBP also accords with earlier studies in that positive results have been observed more consistently for SBP than for any other cardiovascular parameter. However, the fact that a significant Type A effect was obtained for DBP but not for HR is somewhat surprising, because HR effects have been obtained more frequently than DBP effects in previous research. Both the absence of a significant Type A-B effect for HR and the overall low level of HR reactivity ($M = 1.0$ bpm) may be consequences of a cognitive requirement of mirror-tracing (close attention to sensory inputs), which has been associated with reduced cardiac rate (Williams, 1986). This same factor may account for the positive DBP effects, because sensory intake also appears to produce vasoconstriction in skeletal muscles, which would tend to increase peripheral resistance, and could raise DBP (Williams, 1986). Whatever the explanation, mirror-tracing does seem to elicit pronounced DBP elevations, as seen in the present study as well as in a recent investigation by Kasprowicz, Manuck, and Krantz (1988). This suggests that inconsistencies in findings for DBP in Type A research might reflect qualitative variations in the tasks used to induce reactivity in those studies.

Hardiness and Cardiovascular Reactivity

Analyses using the composite hardiness index indicated that DBP responsiveness was significantly lower among subjects with high hardiness scores than among those low in hardiness. The main effects of Type A and hardiness on DBP responses were qualified by a significant interaction indicating that the combination of a Type B behavior classification and high hardiness scores was associated with lowest DBP reactivity. Although an interaction was not specifically predicted, it was expected that Type B subjects high in hardiness would show the lowest levels of cardiovascular response.

These data provide initial support for the notion, implicit in the hardiness construct, that the hardy individual possesses some attribute that dampens the body's response to psychological stress. This conclusion should be viewed guardedly, because two recent studies unexpectedly found hardiness to be associated with enhanced cardiovascular responsiveness to psychological stress (Allred & Smith, 1989; Van Treuren & Hull, 1987). Note, however, that these unanticipated findings involved SBP and HR, not DBP. Moreover, Allred and Smith reported a marginally significant trend toward reduced finger pulse volume in hardy subjects during anticipation of the experimental task. Reductions in finger pulse volume, like the DBP elevations reported here, reflect the constriction of blood vessels resulting from increased sympathetic nervous system activity. Thus, it may tentatively be concluded that hardiness is associated with lower levels of a physiologic response pattern that was elicited by some feature of the mirror-tracing task used in the present study and, perhaps, during the anticipation phase of the Allred and Smith study.

The fact that hardiness exerted a greater modulating effect on the stress response among Type B as compared with Type A individuals deserves comment. Although it would be of theoretical interest if Type As were less likely than Type Bs to benefit from the health-promoting effects of hardiness, clearly it would be premature to conclude this on the basis of the present investigation. Indeed, in another study relevant to this issue, Kobasa, Maddi, and Zola (1983) reported data suggesting that hardiness may exert a greater health-protective effect among Type As, rather than among Type Bs. However, the interaction term reflecting that effect fell short of statistical significance. In addition, Kobasa et al. (1983) measured Type A behavior using the JAS, whereas the DBP results reported here were based on SI assessments. It is generally recognized that the SI and the JAS measure substantially different aspects of the Type A behavior pattern. Behavioral classifications derived from the SI are based mainly on manifest hostility and the use of vigorous speech and motor mannerisms, whereas the JAS is largely a self-report measure of a pressured drive to succeed (Matthews, Krantz, Dembroski, & MacDougall, 1982). It remains for future research to determine how the specific Type A behaviors reflected in SI and JAS assessments may interact with hardiness in determining health outcomes.
**Hardiness Components and Cardiovascular Reactivity**

Multiple regression analyses provided interesting information concerning the contribution of the individual hardiness components to the prediction of physiological responses to stress. Under the conditions of this study, it would appear that challenge, as measured by the Security scale, accounted for the overall effect of the hardiness composite in dampening DBP reactivity among Type Bs. Challenge contributed significantly and to an equivalent degree to the prediction of DBP change scores in the context of regression equations in which the remaining hardiness indicators showed no significant relationship with DBP, regardless of whether those indicators were treated independently or combined into measures of control and commitment. In addition, summing all five scales together to form a single hardiness index conferred no predictive power beyond that attributable to the challenge component alone.

These results must be interpreted with caution. Even modest associations (i.e., multicollinearity) between predictors in a multiple regression equation introduce a degree of instability in the results. In the present study, the challenge and control variables were correlated both with each other and with DBP change scores (see Tables 1 and 3). Therefore, firm conclusions about the results for Model 2 (see Table 2), indicating an independent association between challenge and DBP, must await cross-validation in a larger sample.

With this qualification in mind, it is worth considering the implications of the positive results reported here for the challenge variable. At a general level, these data support the utility of treating the hardiness components as independent constructs in order to determine the degree to which they are differentially associated with adaptive outcomes. This view was not reflected in early hardiness research, in which commitment, control, and challenge were treated as indicators of a single underlying dimension (e.g., Kobasa et al., 1981). However, the trend in more recent work has been to consider the components separately, and this has generated evidence suggesting that the health-promoting effects of hardiness are obtained more reliably for some components than for others (Hull et al., 1987). In this sense, the results of the present study are consistent with other developments in hardiness research. It would appear that future work in this area would benefit from efforts to generate hypotheses concerning the conditions under which the individual hardiness components may differentially influence health-related outcomes.

That subjects high in challenge showed lower physiological reactivity to a frustrating task is consistent with Kobasa's (1979) conceptualization of challenge as a tendency to construe life changes and obstacles in a positive way. Evidently, this orientation is measured somewhat indirectly by the Security scale, which contains items dealing with the desire for protection against physical and economic threat (e.g., "The more able person has a greater responsibility for the welfare of the less able" and "There are no conditions which justify endangering the health, food, and shelter of one's family or of oneself"). Previous research does not provide a basis for expecting that challenge would fare better than commitment and control in accounting for physiological responses to stress. Indeed, the Security scale and its variants have been criticized both for their low levels of internal consistency and for their failure to contribute to the prediction of health outcomes. These findings have led to the recommendation that researchers interested in hardiness focus on the commitment and control components and, presumably, discard the challenge concept (Hull et al., 1987).

Results of the present study favor a somewhat different perspective. To be sure, the internal consistency of proposed measures of the challenge component is inadequate. However, the data reported here suggest that efforts to improve those measures might well prove rewarding: To the limited degree that the Security scale items reflect a single underlying dimension, that dimension is associated with physiological responses to stress. This relationship is consistent with the construct meaning of challenge, and provides evidence for its nomological validity (Cronbach & Meehl, 1955). Therefore, the present study should serve as a stimulus for reconsidering the construct definition of challenge and for developing additional items that better fit that definition in order to revise the scale and improve item homogeneity (Jackson, 1971).

Results for the challenge component also raise questions concerning the practice of using illness reports as the sole basis for assessing the validity of the hardiness concept and for evaluating the relative importance of the hardiness components. Illness reports are a convenient starting point for demonstrating that a personality characteristic may have some relevance to health. However, given the ambiguities of self-report indexes of physical well-being, they should not be relied on too heavily as a criterion for either establishing or disconfirming linkages to disease.

Even quite serious medical disorders (e.g., cancer and hypertension) may exist asymptomatically and, therefore, go undetected by subjective measures of health. Researchers have tended to ignore this possibility, however, focusing instead on the hypothesis that illness reports often may have no basis in organic dysfunction, reflecting what has been referred to as negative affectivity (Watson & Clark, 1984) or a neurotic tendency to report high levels of unfounded somatic complaints (Costa & McRae, 1987). Following this line of reasoning, two recent studies reported that significant associations between hardiness and self-reported health were eliminated when individual differences in psychological adjustment were controlled statistically (Funk & Houston, 1987; Rhodewalt & Zone, 1989). Although these findings might be taken to indicate that the health-enhancing effects of hardiness observed in earlier studies have been inflated, such a conclusion would apply only to self-reported illness.

It is interesting to note, moreover, that neither Rhodewalt and Zone (1989) nor Funk and Houston (1987) examined the relationship between hardiness and health separately for each of the hardiness components. Furthermore, Funk and Houston reported that challenge was unrelated to the two measures of psychological maladjustment that were used to evaluate the neuroticism confound. It is possible, therefore, that challenge was associated with illness in both studies. In any case, it clearly remains for future research, which would, preferably, involve objective measures of disease, to confirm or refute the validity of the hardiness components as personality characteristics that provide resistance to stress-related disorders.

It is worth reiterating that an important adjunct to work that examines disease end points is research concerned with mecha-
nisms that may culminate in illness, such as physiological responses to stress. Note that stress-response dampening represents only one process whereby hardness may promote health. For example, there is evidence that hardy individuals engage in health-promoting practices, such as proper diet and personal hygiene (Wiebe & McCa llum, 1986), and may be less likely to report physical discomfort (Van Treuren & Hull, 1987). Further examination of factors such as physiological reactivity, health-promoting behaviors, and reactions to symptoms may lead to a better understanding of the relationship between hardness and physical well-being.

Conclusion

This study demonstrates that weak associations between Type A behavior and physiological reactivity may, in part, reflect a failure to assess other sources of variation in reactivity. Specifically, although individual differences in reactivity are likely to reflect constitutional and environmental influences (Krantz & Manuck, 1984), the present data support the hypothesis that personality hardness contributes to this variation as well. This finding has implications for understanding the health-promoting effects of hardness, because the sympathetic nervous system activity underlying heightened DBP responses might reflect processes involved in the development of physical disorders. Results of the present study also support the utility of a more multivariate approach in research concerned with the relationship between personality and health.

References


Received November 30, 1988
Revision received March 17, 1989
Accepted May 9, 1989

Members of Underrepresented Groups:
Reviewers for Journal Manuscripts Wanted

If you are interested in reviewing manuscripts for APA journals, the APA Publications and Communications Board would like to invite your participation. Manuscript reviewers are vital to the publication process. As a reviewer, you would gain valuable experience in publishing. The P&C Board is particularly interested in encouraging members of underrepresented groups to participate more in this process.

If you are interested in reviewing manuscripts, please write to Leslie Cameron at the address below. Please note the following important points:

- To be selected as a reviewer, you must have published articles in peer-reviewed journals. The experience of publication provides a reviewer with the basis for preparing a thorough, objective evaluative review.

- To select the appropriate reviewers for each manuscript, the editor needs detailed information. Please include with your letter your vita. In your letter, please identify which APA journal you are interested in and describe your area of expertise. Be as specific as possible. For example, "social psychology" is not sufficient—you would need to specify "social cognition" or "attitude change" as well.

- Reviewing a manuscript takes time. If you are selected to review a manuscript, be prepared to invest the necessary time to evaluate the manuscript thoroughly.

Write to Leslie Cameron, Journals Office, APA, 1400 N. Uhle Street, Arlington, Virginia 22201.