Journal of Personality and Social Psychology
1989, Vol. 56, No. 2, 257-266

0022-3514/89/$00.75

The Hardy Personality: Cognitive and Physiological Responses to Evaluative Threat

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Hardy persons are hypothesized to be resistant to stress-induced illness, because of their adaptive cognitive style and a subsequently reduced level of physiological arousal. We assessed the cognitive and physiological responses of high and low hardy male undergraduates to a challenging task under high and low evaluative threat. As predicted, hardy subjects endorsed more positive self-statements than did low hardy subjects in the high threat condition. High hardy subjects also reported fewer negative self-statements overall, but this was attributable to the overlap of measures of hardiness and neuroticism. Hardy subjects displayed marginally lower arousal while waiting for the task to begin, but this finding did not approach significance when neuroticism was controlled. Hardy subjects also had higher levels of systolic blood pressure, perhaps because of their active coping efforts. Results support the hypothesized hardy cognitive style but raise questions about the type and timing of organismic strain linking hardiness and health.

Considerable research indicates that stressful life events contribute to the development of physical illness (Dohrenwend & Dohrenwend, 1974; Gentry & Kobasa, 1984; Rabkin & Struening, 1976). Although consistent, this relation is small. One approach to understanding this small but reliable association has been the examination of moderator variables: characteristics of persons or their environments that make them more or less vulnerable to the negative effects of stressful events.

Hardiness (Kobasa, 1982) is one such moderator variable and has been a major aspect of the recent resurgence of interest in the role of personality factors in physical health (Suls & Rittenhouse, 1987). Hardy persons are hypothesized to display *commitment* or involvement in daily activities, perceived *control* over life events, and a tendency to view unexpected change or potential threat as a positive *challenge* rather than as an aversive event. Nonhardy persons, in contrast, are hypothesized to display alienation (i.e., a lack of commitment), an external locus of control, and a tendency to view change as undesirable. Gentry and Kobasa (1984) argued that the collection of personality characteristics composing hardiness "mitigates the potential unhealthy effects of stress and prevents the organismic strain that often leads to illness" (p. 99).

Evidence for the positive effects of hardiness has come from a series of retrospective and prospective studies comparing hardy and nonhardy persons' reports of illness in response to high levels of life stress. As predicted by Kobasa's (1982) model, the results have generally demonstrated that hardy persons report less illness than do nonhardy subjects under conditions of high life stress (Kobasa, 1979; Kobasa, Maddi, & Courington, 1981; Kobasa, Maddi, & Kahn, 1982; Kobasa, Maddi, & Puccetti, 1982; Kobasa, Maddi, & Zola, 1983; Kobasa & Puccetti, 1983). Some recent studies, however, have failed to replicate the predicted relation between hardiness and illness reports (Funk & Houston, 1987; Schmied & Lawler, 1986).

Limitations in Hardiness Research

Evidence of the Hardy Cognitive Style

In addition to occasional failures to replicate the hardiness–illness report relation, this area is characterized by several potential problems. To begin, the stress-buffering effect of hardiness is hypothesized to result from an adaptive cognitive appraisal process (Lazarus & Folkman, 1984). It is presumed that hardy persons respond to potential stressors with positive cognitions or appraisals concerning both the level of threat present and their ability to cope effectively (i.e., primary and secondary appraisal, respectively; Lazarus & Folkman, 1984). Nonhardy persons, on the other hand, are hypothesized to respond to the same event with fewer positive cognitions and more negative thoughts. It is clear that cognitive differences between high and low hardy persons are the central component of this model of stress buffering.

Some evidence of the hardy cognitive style was found in a study by Rhodewalt and Agustsdottir (1984). Hardy subjects were more likely than nonhardy subjects to perceive reported life events as positive and under their complete control, despite both groups appearing to experience relatively similar events. It is important to note, however, that the potential stressors were reported rather than manipulated. As a result, it is uncertain whether hardy and nonhardy subjects—as would be predicted by the model—displayed these different cognitions in response to the same objective stressors. To address this issue, we examined cognitive processes in high and low hardy subjects as they

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confronted a controlled laboratory stressor (i.e., a potential evaluative threat).

Evidence of "Organismic Strain"

The hardiness model assumes that the adaptive cognitions of hardy persons result in a lower level of organismic strain (Gentry & Kobasa, 1984) in response to potential stressors. Presumably, this would be reflected in lower levels of physiological arousal when hardy persons, relative to nonhardy persons, confront a potential stressor. This corollary hypothesis is consistent with a large number of studies indicating that positive appraisals produce decreases in physiological arousal while subjects anticipate and confront potential stressors (e.g., Bennett & Holmes, 1975; Holmes & Houston, 1974; Houston & Holmes, 1974; Lazarus, Opton, Nomikos, & Rankin, 1965).

A similar model has been proposed concerning the relation between Type A behavior and coronary heart disease. A large number of initial tests of the model have generally indicated that Type A behavior is associated with exaggerated physiological responses to controlled laboratory stressors (for reviews, see Contrada, Wright, & Glass, 1985; Houston, 1988). Only one study to date, however, has examined the hypothesized psychophysiological correlates of hardiness. Contrary to the general hardiness model, however, Van Treuren and Hull (1987) found that high hardy subjects had higher levels of skin conductance overall and, under some task conditions (i.e., contingent success feedback), higher levels of heart rate (HR) and systolic blood pressure (SBP) relative to low hardy subjects.

Although the hardiness model would seem to predict dampened physiological responses to stressors among hardy persons, other studies of personality and physiological reactivity, as well as studies of the physiology of coping, suggest a more complex situation. This more complex pattern would also be more consistent with the findings of the one previous study of physiological responses and hardiness. An internal, rather than external, locus of control (Rotter, 1966) has been found to be associated with increased HR and SBP responses to laboratory stressors (Houston, 1972; Manuck, Harvey, Lechleiter, & Neal, 1978). Presumably, this is because internally oriented persons attempt to cope actively with the stressors (i.e., influence or control stressful events). Active coping efforts or attempts to exert control over events in turn produce increases in HR and SBP (Light & Obrist, 1980; Smith, Houston, & Stucky, 1985; Solomon, Holmes, & McCaul, 1980). An internal locus of control is a central aspect of the conceptual and operational definition of hardiness. Therefore, it may be expected that high hardy subjects would display increased levels of those physiological parameters that reflect active coping efforts (i.e., HR, SBP).

Given that hardy persons are less likely to appraise an event as threatening, however, they would also be expected to display lower levels of the physiological parameters that are sensitive to the effects of threat but not influenced by coping efforts. Recent research suggests that vasoconstriction in the skin (i.e., finger pulse volume [FPV]) is sensitive to the effects of threat (e.g., Bloom, Houston, & Burish, 1976; Smith, Houston, & Zurawski, 1984a). That is, the threat of shock or a threat to self-esteem produces decreased FPV. However, unlike HR and SBP, FPV is apparently not influenced by attempts to exert control over or actively cope with threatening events. For example, two studies (Smith et al., 1985; Solomon et al., 1980) showed that the threat of electric shock produced highly significant decreases in FPV but that the opportunity to avoid shock by successful task performance (i.e., active coping or control over shock) did not influence FPV levels. In this study we examined the effect of a potential stressor on these different physiological responses (i.e., HR and SBP vs. FPV) in high and low hardiness groups. Although these psychophysiological effects of active coping and threat may not be completely distinct, examination of the pattern of these responses may provide a test of the various possible physiological correlates of hardiness.

The Potential Neuroticism Confound

Recent reviews of the hardiness literature have concluded that the relation between hardiness and health reports is most frequently found to be a main effect rather than the Hardiness × Life Stress interaction implied by the notion of a moderator variable (Cohen & Edwards, in press; Funk & Houston, 1987; Hull, Van Treuren, & Virnelli, 1987). Although some statistically significant interactions have been reported (Ganellen & Blaney, 1984; Kobasa, Maddi, & Kahn, 1982; Rhodewalt & Zone, 1989), these are exceptions to the typical pattern of hardiness main effects. This fact is not only inconsistent with the hardiness model; it also presents the possibility that previous hardiness effects may actually reflect the operation of a different, more fundamental individual difference dimension: neuroticism. Costa and McCrae (1985, 1987), as well as Watson and Pennebaker (in press), have demonstrated that chronically dysphoric, worried (i.e., high neuroticism) persons, relative to more emotionally stable persons, consistently report high levels of somatic complaints, even though they are no more likely to display actual physical illness. That is, there is a consistent correlation between neuroticism and illness reports. Further, several authors have demonstrated that measures of hardiness are correlated with measures of neuroticism or maladjustment (Funk & Houston, 1987; Hull et al., 1987). Hull et al. found that hardiness measures were as closely correlated with measures of emotional distress as they were with a measure of optimism (Scheier & Carver, 1985), a construct that in theory is more similar to hardiness than is simple emotional distress. Finally, Funk and Houston (1987) found that the otherwise significant correlations between hardiness and health reports were eliminated when emotional maladjustment was statistically controlled, a finding recently replicated by Rhodewalt and Zone (1989).

Taken together, these findings indicate that (a) the measures of hardiness are contaminated with neuroticism and (b) the relation between hardiness and health reports or other processes may reflect neuroticism rather than the construct proposed in Kobasa's (1982) model. That is, the hardiness–health relation may actually reflect a relation between neuroticism and somatic complaints (but not actual illness). Although Kobasa et al. (1981) attempted to validate their measure of health reports by comparing them with physicians' ratings, the overwhelming majority of the research on hardiness has assessed health com-

plaints but not actual health. Furthermore, the confounding or contamination of hardiness with neuroticism creates the possibility that the relation between hardiness and processes such as appraisal may actually involve neuroticism instead of the hardiness construct. To address this issue while evaluating the organismic strain and cognitive appraisal aspects of the hardiness model, we examined the relation between hardiness and cognitive processes and physiological responsiveness with and without controlling for neuroticism. In this way we could explore the potential confound with neuroticism as an alternative explanation of any obtained effects.

Measurement of Hardiness

Previous authors have criticized measures of hardiness on the basis that the construct is measured negatively (Funk & Houston, 1987; Hull et al., 1987). That is, hardiness is operationally defined as the relative absence of alienation, powerlessness, need for security, and external locus of control. This problem, noted previously by Gentry and Kobasa (1984), may be the source of the potential confounding of hardiness and neuroticism. It is also true that it may be best to examine hardiness at the level of its components rather than as a composite pattern (Hull et al., 1987). Finally, others have noted considerable variability across studies in the specific scales used to measure hardiness (Hull et al., 1987; Scheier & Carver, 1985), raising questions about the similarity of findings. Thus, measurement issues compound many of the difficulties just outlined.

In this study we examined hardiness as it is typically measured. Thus, the problems with negative measurement and with measurement as a composite rather than as components remained. Although not providing a solution to these problems, this study thereby remained applicable to the previous literature. That is, in this study we assessed the cognitive and physiological correlates of hardiness as it was operationally defined in the studies of hardiness as a moderator of the stress–illness relation. Regarding the changing scales issue, however, we attempted to deal with this by selecting subjects for the laboratory experiment on the basis of their meeting criteria on two separate measures of hardiness. By using two converging measures, subjects were more likely to reflect those conceptually defined by Kobasa (1982) as high hardy and low hardy than if a single method had been used.

Overview of the Present Study

As described earlier, we examined the cognitive appraisal and organismic strain aspects of the hardiness model by assessing cognitive and physiological responses of high and low hardiness groups to an experimental task that was either high or low in potential threat. We predicted that, relative to low hardy subjects, high hardy subjects would respond to high threat with a high level of positive thoughts and a low level of negative thoughts. We also predicted that high hardy subjects would display relatively lower levels of physiological arousal indicative of threat (i.e., FPV) but that they may show increased levels of arousal associated with active coping efforts (i.e., HR, SBP). To evaluate the potential neuroticism confound, we examined these effects with and without controlling for neuroticism.

Method

Subjects

Eighty-four male undergraduate psychology students participated in the experiment in exchange for course extra credit. Because the majority of studies addressing hardiness have been with a male population, we used only male subjects to ensure relevance to previous literature.

Measures of Hardiness

As noted earlier, hardiness has been assessed with a variety of methods in previous studies. Unfortunately, it is not yet clear which method is the most reliable and valid assessment of hardiness. In this study we assessed hardiness at two separate times. First, we assessed hardiness in a preliminary mass testing of about 300 men using the 20-item Abridged Hardiness Scale developed by Kobasa and Maddi (S. C. Kobasa, personal communication, July 1982). Those researchers selected the items of this scale from the unabridged scale (the Hardiness Scale) because of their high correlation with total scale scores. In addition, Kobasa (personal communication, July 1982) reported a correlation of .89 between the abridged and unabridged forms and stated that all hardiness findings in previous studies using the unabridged scale were replicated when Abridged Hardiness Scale scores were substituted. Furthermore, two independent studies using the Abridged Scale have replicated expected hardiness effects (Rhodewalt & Agustsdottir, 1984; Rhodewalt & Zone, 1989). Following the mass testing, we telephoned individuals scoring in the extreme quartiles of the Abridged Hardiness Scale distribution and randomly assigned them to experimental conditions.

At the end of the experimental session, subjects completed the 36-item Revised Hardiness Scale (S. C. Kobasa, personal communication, November 1982). The 36-item Revised Hardiness Scale, like the Abridged Hardiness Scale, also consists of a subset of the original scale items. Kobasa (personal communication, November 1982) stated that this scale also correlated with the full scale at .89 and that all major findings were replicated when this scale was substituted for the full scale in her earlier samples. Hull et al. (1987) reported a correlation of .76 between the original long form and the 36-item Revised Hardiness Scale, and Schlosser and Sheeley (1985) independently replicated the usual hardiness–health relation with the Revised Scale. It should be noted that 9 items in the Abridged Hardiness Scale also appear on the Revised Hardiness Scale.

We performed a median split on the Revised Hardiness Scale scores and included only individuals who scored in the corresponding upper or lower end of the distribution for both the Revised and Abridged scales in the analyses. Of the original 84 subjects, 58 met criteria on both measures and were included in the analyses. This two-criteria classification system resulted in the following cell sizes: high hardiness/high stress (n = 17); high hardiness/low stress (n = 12); low hardiness/high stress (n = 13); and low hardiness/low stress (n = 16). The inclusion of only those subjects classified similarly by two operational definitions of hardiness should provide a more reliable and valid classification of hardiness (cf. Sacco, 1981).[1]

The Revised Hardiness Scale was originally composed of six subscales (S. C. Kobasa, personal communication, November 1982). In a check of internal consistency, it was found that Subscale 5 (cognitive

[1] The correlation between the two forms of the Hardiness Scale was .50 (df = 84), $p < .001$, in spite of the fact that the scales shared only nine items and were administered an average of 6 weeks apart.

structure) had either no correlation or was negatively correlated with the other subscales; Cronbach's alpha (using subscale scores as items) increased from .59 to .73 when Subscale 5 was deleted. This is consistent with more recent hardiness studies in which the Cognitive Structure subscale was not included in formulating an overall hardiness score (e.g., Kobasa, Maddi, & Puccetti, 1982; Kobasa et al., 1983; Kobasa, Maddi, Puccetti, & Zola, 1985; Kobasa & Puccetti, 1983; Wiebe & McCallum, 1986). Consequently, we did not include this subscale in the computation of an overall Revised Hardiness Scale score. Although this change did not produce any change in statistically significant hardiness effects, it appears to be a more reliable means of assessing hardiness in this sample.

Measurement of Neuroticism

After completing the 36-item Revised Hardiness Scale, subjects completed the Trait scale of the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970). The Trait scale of the STAI is a widely used 20-item scale with considerable evidence of validity (Spielberger et al., 1970). Several recent studies have demonstrated that the Trait scale, in normal populations, is highly correlated with a variety of other measures of anxiety, depression, and maladjustment (Gotlib, 1984; Tanaka-Matsumi & Kameoka, 1986; Watson & Clark, 1984). The conclusion of these researchers is that in the normal (i.e., nonclinical) population, measures of anxiety, depression, and many other types of maladjustment are virtually indistinguishable and are best viewed as measuring a single, more general dimension rather than the specific affective disturbances implied by the individual scale names. This general dimension is variously labeled *neuroticism* (Costa & McCrae, 1985, 1987), *general dysphoria* (Gotlib, 1984), or *negative affectivity* (Watson & Clark, 1984). We use the term *neuroticism* in this discussion, because this is the term used in discussions of the relation between this construct and somatic complaints. As mentioned earlier, the Hardiness Scale, the Abridged Hardiness Scale, and the Revised Hardiness Scale have been found to be significantly correlated with various measures of neuroticism (e.g., Funk & Houston, 1987; Hull et al., 1987; Rhodewalt & Zone, 1989).

Procedure

On arriving for the experiment, the experimenter gave subjects a written informed consent statement. Transducers to measure HR, SBP, diastolic blood pressure (DBP), and finger pulse volume (FPV) were attached and monitored using a Beckman Type-R Dynograph. Subjects were seated at a table facing the wall and were separated from the polygraph and the experimenter by a partition. Following a 6-min baseline period, subjects completed a State scale of the State-Trait Personality Inventory (STPI; Spielberger, 1984) to provide a baseline measure of state affect. This 30-item inventory provides scores for anxiety, anger, and curiosity.[2]

Stress manipulation. After the baseline measures were completed, subjects in the high stress condition were told via an audio recording that the experiment was concerned with the physiological correlates of intelligence. They were told they would soon be given an abridged version of the bogus Western Verbal-Spatial Abilities Test, previously found to predict success in a variety of academic and vocational activities. Subjects in the low stress condition were told via audio recording that the experiment was concerned with the physiological correlates of cognitive processes. They were told they would soon be given some questions that were created for the experiment and that the accuracy of their responses was not important. This type of high versus low threat manipulation has successfully influenced physiological and reported affective measures of stress in previous research (Smith et al., 1984a). All subjects were then asked to sit quietly for a few minutes while the test was prepared.

Experimental task. After a 2-min anticipation period, slides were presented with questions in two categories: mental rotations and analogies. First, subjects were shown analogies taken from an Educational Testing Service preparation manual for the Graduate Record Examination. Next, subjects completed a mental rotation task (Shepard & Metzler, 1971), in which they indicated if two configurations of 10 adjoining cubes were different or the same but rotated to different angles. For both types of items, approximately half were relatively easy and the others were very difficult. The task period lasted approximately 6 min, and a record was kept of the number of correct responses. On completion of the slides, subjects completed a self-statement inventory (cf. Kendall & Hollon, 1981) and a second form of the STPI, which inquired about affect during the task. Following this, attachments for physiological recordings were removed. Subjects then completed the previously described 36-item Revised Hardiness Scale and the STAI.

Self-Statement Inventory

Positive and negative thoughts occurring during the task period were assessed by means of a self-statement inventory developed for this study (cf. Kendall & Hollon, 1981). Positive thoughts referred to positive aspects of the self or performance (e.g., "I will probably know the answer to the next question," "I think I am performing well"). Negative thoughts similarly referred to negative aspects of the self or performance (e.g., "I am thinking lower of my ability," "I think the experimenter thinks negatively of me"). Given this item content, the scale is likely to reflect elements of both primary and secondary cognitive appraisal processes (cf. Lazarus & Folkman, 1984) rather than one or the other specific type of cognition. For both of these positive and negative categories, subjects rated four items on 5-point scales in terms of the extent to which they experienced the particular thought during the task period. Previous research has indicated that the self-statement inventory procedure in general has been reliable and valid (for a review, see Kendall & Hollon, 1981).

Recording and Reduction of Physiological Measures

SBP and DBP were recorded with a standard, manually inflated occluding cuff and a pulse sounds microphone attached to the dominant arm. Cuff inflation and deflation were controlled, remotely, from behind the partition separating the seated subject from the experimenter and polygraph. HR was recorded with a three-lead EKG procedure, and FPV was recorded with a photoelectric plethysmograph attached to the middle finger of the nondominant hand. Blood pressure recordings were made at 1-min intervals (i.e., 30 s between inflations) in the last 3 min of the baseline period, once during the last minute of the anticipation period, and at 1-min intervals throughout the task period. FPV and HR were recorded throughout the course of the experiment. Values for HR and FPV subjected to statistical analyses were mean scores derived from a 30-s period prior to each inflation of the blood pressure cuff. For FPV, each mean was computed from seven evenly spaced samples within each 30-s period (cf. Bloom et al., 1976). HR was determined by counting the number of beats occurring within each 30-s period. For each physiological measure, an average of the last 2 baseline period readings served as the baseline value, and four trials

[2] As only the Anxiety subscale of the State-Trait Personality Inventory was relevant to predictions from the hardiness model, we do not discuss results involving the Anger and Curiosity scales. Note that the Curiosity and Anger scales produced no evidence of differential responses to threat in high versus low hardy subjects.

corresponded to the one anticipation period measurement and three task period measurements.

Results

Correlation Between Hardiness and Neuroticism

In this sample the Revised Hardiness Scale and the Abridged Hardiness Scale were both significantly correlated with STAI Trait scores, $rs(84) = .53$ and .48, respectively, $ps < .001$. Furthermore, the correlation (point biserial) between the previously described twofold classification of hardiness (i.e., high vs. low) and STAI Trait scores was .50 ($df = 59$), $p < .001$. Thus, as in previous studies (Funk & Houston, 1987; Hull et al., 1987), hardiness and neuroticism were clearly confounded in this sample.

Validity of the Cognitive Assessment

To evaluate the validity of the Self-Statement Inventory, we computed correlations among the cognitive measures and the State Anxiety scores. Because our interest was in anxiety aroused in response to the task rather than initial anxiety, we performed partial correlations between the task period anxiety scores and the cognitive measures, controlling for baseline anxiety scores. This was equivalent to correlating task-related increases in anxiety with the self-statement measures. Given that the Anxiety scale of the STPI has shown adequate reliability and validity, and given that high levels of negative thoughts and low levels of positive thoughts are associated with the arousal of anxiety (Cacioppo, Glass, & Merluzzi, 1979; Kendall et al., 1979; Schwartz & Gottman, 1976), statistically significant relations between the cognitive measures and the anxiety scores should provide some evidence as to the validity of the cognitive measures. The positive and negative self-statement scores had significant, expected relations with task period anxiety ($rs = -.24$ and .33, respectively, $ps < .05$). Positive and negative self-statement scores were also negatively related to each other ($r = -.52$, $p < .01$). Thus, although they were not large, the observed relations appear to provide evidence for the validity of the Self-Statement Inventory.

Effectiveness of the Experimental Manipulation

We evaluated the stress manipulation by comparing physiological measures, cognitive measures, and self-report affect measures in the high stress and low stress conditions. An examination of baseline values showed no statistically significant differences at baseline between the two conditions on any of the physiological or affect measures. For anticipation and task period analyses, we used baseline values in repeated measures (i.e., one anticipation period and three task period trials or measurements) analyses of covariance (ANCOVAs) in order to provide a base-free measure of change (Kinsman & Staudenmayer, 1978).

On the physiological measures, subjects in the high stress condition had lower FPV (i.e., higher arousal) than did subjects in the low stress condition, $F(1, 52) = 5.16$, $p < .03$, and this difference was evident when subjects were anticipating the task, $t(52) = 2.96$, $p < .01$, and participating in the task, $t(52) = 3.45$, $p < .01$. High stress subjects also displayed higher SBP, $F(1, 43) = 5.73$, $p < .025$, and marginally higher DBP, $F(1, 43) = 3.40$, $p < .075$. The stress main effect for SBP was qualified by a Trial × Condition interaction, $F(3, 135) = 4.40$, $p < .01$, as was the DBP main effect, $F(3, 132) = 3.26$, $p < .025$. Mean comparisons (Bernhardson, 1975) within the SBP interaction showed that high stress subjects displayed higher SBP than did low stress subjects during each task period (all $ps < .05$), but not during the anticipation period. A similar pattern was found for the DBP interaction, with DBP higher in the high stress condition during all three task periods (all $ps < .05$), but again not during the anticipation period. Means for the physiological measures are presented in Table 1.

Table 1
Anticipation and Task Period Means for Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), and Finger Pulse Volume (FPV) in the High and Low Threat Conditions

Measure	Anticipation trial	Period 1	Period 2	Period 3
SBP				
High threat	112.5	118.6	116.4	117.8
Low threat	113.2	113.9	113.2	113.9
DBP				
High threat	77.8	82.6	82.8	82.2
Low threat	76.7	79.2	78.1	79.2
FPV				
High threat	1.18	1.09	1.12	1.19
Low threat	1.94	1.54	1.74	1.67

No statistically significant conditions effects were found for state anxiety or either cognitive measure, although the cell means for each of these measures were in the expected direction (i.e., high threat subjects reported nonsignificantly higher state anxiety and negative thoughts). Furthermore, the state anxiety results revealed the presence of a marginally significant Condition × Period interaction, $F(1, 54) = 2.37$, $p = .13$. Because we expected the overall stress effect and because different effects across periods could explain the lack of an overall stress conditions effect, we compared the specific period means. These comparisons indicated that although the high and low stress conditions did not differ during the anticipation period ($Ms = 18.77$ and 18.37, respectively), $t(53) < 1.0$, during the task period high stress subjects reported higher levels of state anxiety than did low stress subjects ($Ms = 20.87$ and 18.20, respectively), $t(53) = 2.02$, $p < .05$. Thus, although low stress subjects reported nonsignificantly less anxiety during the task than during the anticipation period, high stress subjects reported significantly more anxiety during the task than during the anticipation period, $t(54) = 2.04$, $p < .05$. Thus, state anxiety reports suggested that at least during the task period the manipulation was effective. Nonetheless, evidence for the effectiveness of the stress manipulation was stronger among the physiological measures than among the self-report measures. Low levels of convergence among stress measures is not uncommon (Baum, Grunberg, & Singer, 1982; Nietzel & Bernstein, 1981). To some extent, however, it obscures the precise meaning of the variable that

was manipulated. If evaluative threat had been manipulated, the cognitive and affective measures might not have been as sensitive as were the physiological measures. It is possible, however, that something in addition to or other than threat to self-esteem was manipulated, possibly task involvement (Singer, 1974) or attempt to exert control (Houston, 1972; Manuck et al., 1978), and that the physiological measures were sensitive to changes in these variables. It should be recalled, however, that FPV has been found to be sensitive to threat (Bloom et al., 1976; Smith et al., 1985), but not the effects of active coping or attempts to exert control (Smith et al., 1985; Solomon et al., 1980). Thus, it is most likely that threat, rather than one of these other dimensions, was manipulated as intended.[3] Finally, an analysis of subjects' task performance scores showed no effect for experimental condition.

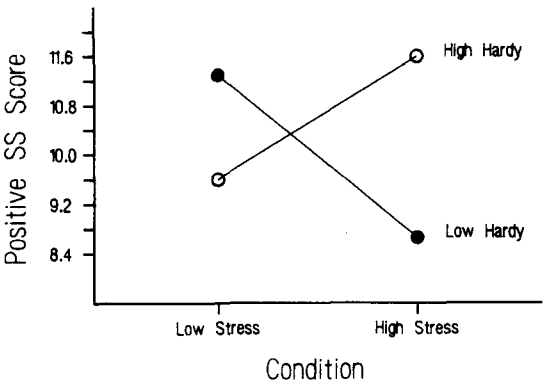

Figure 1. Effects of stress and hardiness on positive self-statements (SS).

Hardiness Effects on Affect, Cognitive, and Physiological Measures

A 2 (hardiness) × 2 (condition) analysis of variance (ANOVA) of physiological and affective baseline scores revealed a marginally significant effect of hardiness on state anxiety scores, $F(1, 54) = 3.38, p < .075$. High hardy subjects reported experiencing less anxiety at baseline than did low hardy subjects. When neuroticism was controlled by including STAI Trait scores as a covariate in this analysis, the marginal effect for baseline state anxiety was eliminated. No other baseline differences were found (i.e., no effects of hardiness on state anger, HR, FPV, DBP, or SBP). It should also be noted that an analysis of the high and low hardy subjects' performance scores showed no significant differences.

Cognitive effects. The 2 (hardiness) × 2 (condition) ANOVA of the Self-Statement Inventory revealed two effects involving hardiness. First, subjects high in hardiness reported fewer negative thoughts overall (i.e., regardless of condition) than subjects low in hardiness, $F(1, 54) = 4.21, p < .05$. When neuroticism was controlled by including STAI Trait scores as a covariate, however, the hardiness main effect was eliminated, $F(1, 53) = 0.19$. Consistent with previous research (Cacioppo et al., 1979; Smith, Houston, & Zurawski, 1984b), trait anxiety was closely related to negative self-statements, covariate $F(1, 53) = 9.20$, $p < .005$.

Second, the 2 × 2 ANOVA of positive self-statements revealed a significant Hardiness × Condition interaction, $F(1, 54) = 8.44, p < .006$ (see Figure 1). Comparisons among the means (Bernhardson, 1975) indicated that low hardy subjects in the high stress condition endorsed fewer positive self-statements than did low hardy subjects in the low stress condition ($p < .01$). In contrast, high hardy subjects in the high stress condition endorsed more positive self-statements than did high hardy subjects in the low stress condition ($p < .06$). Within the high stress condition, high hardy subjects endorsed more positive self-statements than did low hardy subjects ($p < .01$). Within the low stress condition, low hardy subjects tended to report more positive self-statements than did high hardy subjects, but this difference did not approach significance. Consistent with predictions, then, high hardy individuals responded to the high stress condition with more positive self-statements than did low hardy individuals. Furthermore, high hardy individuals tended to be more positive in the high stress condition than in the low stress condition, whereas low hardy individuals were more positive in the low stress condition than in the high stress condition. It is important to note that the interaction remained significant, $F(1, 53) = 7.86, p < .01$, when neuroticism was controlled through an ANCOVA. Trait anxiety was not related to positive self-statements, covariate $F(1, 53) = 1.50$. Furthermore, the pattern and specific differences remained as described in the ANOVA results.

Physiological effects. In 2 (condition) × 2 (hardiness) × 4 (one anticipation and three task period trials) mixed ANCOVAs (i.e., with baseline values as a covariate) of the physiological measures, a main effect for hardiness was found for SBP, $F(1, 44) = 5.42, p < .025$. Subjects high in hardiness showed an overall higher SBP than did subjects low in hardiness (covariance adjusted task period means were 116.2 mmHg and 113.6 mmHg, respectively). For descriptive purposes (i.e., change scores were not analyzed), this represented a task-related increase (i.e., average raw task period value minus raw baseline value) of 6.6 mmHg for high hardy subjects, compared with 2.8 mmHg for their low hardiness counterparts. This hardiness main effect remained when neuroticism was controlled, although it was only marginally significant, $F(1, 44) = 3.49, p < .07$. There was no relation between neuroticism and SBP, covariate $F(1, 43) = 0.04$.

A Trial × Hardiness interaction was found for FPV, $F(3, 159) = 4.54, p < .005$ (see Figure 2). Mean comparisons revealed a marginally significant difference at the anticipation period ($p < .08$), in which high hardy subjects showed greater FPV than did low hardy subjects. This difference suggests that

[3] This assertion is consistent with the results of additional post hoc analyses of the state anxiety measure. Newman-Keuls comparisons indicated that subjects in the high threat condition reported significantly more state anxiety during the task than they did at baseline ($p < .01$), whereas subjects in the low threat condition reported nonsignificantly less state anxiety during the task than they did during baseline ($p > .20$). Thus, these post hoc analyses suggest that the high threat condition, unlike the low threat condition, tended to produce increases in reported state anxiety over levels observed at baseline.

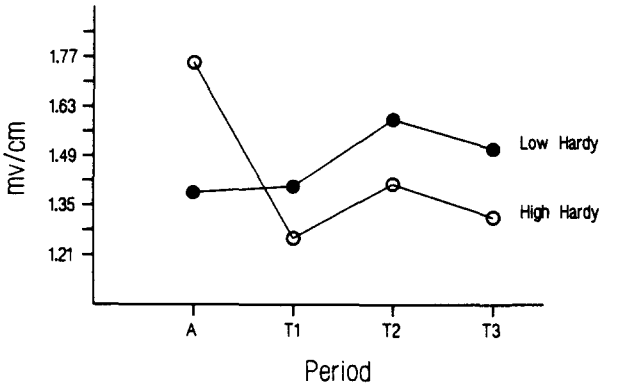

Figure 2. Finger pulse volume during anticipation and task periods in high and low hardiness groups.

high hardy individuals tended to be less aroused than low hardy individuals while waiting for the task to begin. The *t* tests performed on the high and low hardy FPV differences during the three task periods were all nonsignificant (all *ps* > .20), indicating that high hardy subjects became as aroused as low hardy subjects once the task began. Accordingly, high hardy subjects showed a significant decrease in FPV (i.e., increase in arousal) from the anticipation period to the initial task period, $t(159) = 3.95$, $p < .001$. To repeat, then, high hardy individuals tended to be less aroused, as measured by FPV, than low hardy individuals at the anticipation period and were equally aroused during the task period.

This interaction remained significant and the pattern was similar when neuroticism was controlled in an ANCOVA, $F(3, 159) = 4.54$, $p < .005$. Furthermore, there was no significant relation between neuroticism and FPV, covariate $F(1, 51) = 0.88$. However, the size of the difference between high and low hardiness groups was attenuated, so that hardiness differences in FPV did not approach significance during any period. The only significant comparison within this interaction was the decrease in FPV (i.e., increase in arousal) among hardy subjects from the anticipation period to the initial task period trial, $t(158) = 3.01$, $p < .01$. Again, however, we emphasize that when neuroticism was controlled, no differences in FPV between hardy and nonhardy groups approached significance.

Finally, note that there were no hardiness effects on measures of affect during the task or on the HR and DBP measures.[4]

Discussion

Cognitive and Physiological Correlates of Hardiness

This study provides some support for the hypothesized cognitive style of the hardy person. In the high stress condition, high hardy individuals endorsed more positive self-statements than did low hardy persons. Furthermore, high hardy subjects reported more positive self-statements in the high stress condition than did high hardy subjects in the low stress condition. In contrast, low hardy subjects reported fewer positive thoughts in the high stress condition than in the low stress condition. This pattern is consistent with the model proposed by Kobasa (1982) and with previous studies of the relation between hardiness and descriptions of stressful life events (Rhodewalt & Agustsdottir, 1984). Although hardy subjects also demonstrated the predicted lower level of endorsement of negative self-statements relative to nonhardy subjects, this difference was attributable to the confounding of hardiness and neuroticism. The positive self-statement results, however, cannot be attributed to neuroticism and instead appear to reflect the predicted cognitive correlates of hardiness in response to stress. These results are at least consistent with the view that hardiness may moderate the effects of stress by way of cognitive processes.

Results for the physiological questions, however, provided little evidence of the lowered organismic strain, hypothesized to follow such adaptive cognitions. There was some marginal evidence of lower physiological arousal (i.e., higher FPV) while high hardy subjects anticipated the task. This difference was not maintained during the task, however, and the marginal anticipation period finding did not approach significance when neuroticism was controlled. The only significant physiological difference, and one that was not attributable to neuroticism, indicated that hardy subjects had larger SBP responses to the task than did nonhardy subjects. As noted previously, attempts to cope actively rather than passively with stressors elicit such increases in SBP (Light & Obrist, 1980; Manuck et al., 1978; Smith et al., 1985). In addition, an internal locus of control is associated with increased physiological indicators of active coping during confrontations with stressful tasks (Houston, 1972; Manuck et al., 1978). Thus, increased arousal often reflects potentially adaptive, effortful coping rather than distress, and such coping efforts would be entirely consistent with the hardiness model.

Nonetheless, these physiological findings raise questions regarding the nature of the link between hardiness and health. In this experiment high hardy individuals displayed a physiological response (i.e., increased reactivity) that has been suggested as a link between stress and increased risk of illness (Cohen, 1979; Matthews et al., 1986) rather than as a mitigator of illness.

[4] Although the different administration times (i.e., weeks before vs. immediately after) render direct comparisons somewhat ambiguous, we recalculated the hardiness effects just described using the Revised and Abridged Hardiness scales to classify subjects for two parallel separate sets of analyses. The two ways of classifying hardiness (i.e., pretest Abridged Scale scores vs. posttest Revised Scale scores) produced similar but not identical results. Analyses of finger pulse volume and baseline state anxiety produced virtually identical results, reflecting the same pattern as that described in the main analyses. The Revised Scale ($p < .02$), but not the Abridged Scale ($p > .2$), reproduced the systolic blood pressure reactivity main effect. The main effect on negative self-statements was significant for the Revised Scale ($p < .05$) but only approached significance for the Abridged Scale ($p < .12$). The previously described Hardiness × Condition interaction on positive self-statements was significant using the Abridged Scale ($p < .01$), but not when using the Revised Scale to classify subjects ($p = .14$). However, the pattern among the means in the positive self-statements interaction was similar across all three methods of classification; even in the nonsignificant result with the Revised Scale the conceptually crucial high versus low hardiness difference in the high stress condition was significant ($p < .05$).

There are a variety of explanations that could possibly resolve this apparent contradiction. First, some researchers (e.g., Gal & Lazarus, 1975) have suggested that it is not the level of physiological reactivity to an event but the recovery time necessary to return to baseline levels that is important to the development of illness. We did not assess physiological recovery following the task period. Second, it may be that other physiological parameters (e.g., catecholamine or cortisol levels or both) would have provided evidence of organismic strain. Third, it may be that the stressor used in this study did not engage hardy subjects sufficiently to produce differences in reactivity. This suggestion is at least somewhat inconsistent, however, with the predicted hardiness effects on positive cognitions.

Fourth, a recent study by Wiebe and McCallum (1986) suggests that hardiness may influence the stress–illness relation via health practices. That is, individuals high in hardiness appear to maintain better health practices under conditions of high life stress than individuals low in hardiness. Thus, physiological reactivity may not even be relevant to the disease-mitigating effects of hardiness.

The final explanation for the absence of a relation between hardiness and physiological responses concerns shared variance with neuroticism. The studies that have provided support for hardiness as a buffer in the stress–illness relation have used self-report measures of illness. As described earlier, research has shown that the personality dimension of neuroticism is associated with illness reports, but not with actual illness (Costa & McCrae, 1985, 1987; Watson & Pennebaker, in press). For the neurotic individual, illness reports may reflect heightened somatic sensitivity or exaggerated bodily concern rather than organic disease. Hardiness, to the extent that it is related to neuroticism, may be associated with lower illness reports or illness behavior but not with actual illness. This hypothesis is supported by recent reports in which the expected relation between hardiness and health reports was eliminated when measures of neuroticism were statistically controlled (Funk & Houston, 1987; Rhodewalt & Zone, 1989).

The suggestion of a relation between hardiness and symptom reporting is consistent with a recent study (Van Treuren & Hull, 1987) on reports of discomfort in response to the cold-pressor task. Although high and low hardy groups kept their hands immersed for equal amounts of time, the low hardy group reported significantly greater discomfort. If hardiness is related to illness behavior but not to actual illness, then high hardy subjects would not necessarily be expected to display dampened physiological reactivity.

Implications for Hardiness Research and Theory

Overall, our results provide some support for some aspects of the hardiness construct, but they also underscore the need for both conceptual and methodological refinements. The primary support lies in the findings concerning adaptive cognitive responses to potential stressors, a central aspect of the hardiness model. One area for conceptual refinement concerns the nature of the link between hardiness and health. The simple suggestions of reduced organismic strain must be expanded and clarified for theory-driven tests to continue. If the hypothesized link remains in the area of stress physiology, physiological activity during contact with stressors may not be the best point to test the model. Arousal during these periods may reflect a complicated mixture of distress (i.e., organismic strain) and the physiological effects of adaptive coping efforts. High hardy persons would be expected to have low levels of the former but high levels of the latter, thereby obscuring hardiness effects. Physiological recovery may be a more likely point of difference. Distinguishing between the physiological arousal attributable to adaptive, ultimately health-enhancing active coping efforts and pathogenic physiological affects is not a dilemma unique to hardiness research. Rather, it is a much broader psychosomatic question, with particular relevance for hardiness theory given the hypothesized coping behaviors (i.e., attempts to exert control) and what is known about the short-term physiological affects of such coping.

A second conceptual refinement concerns alternative pathways between hardiness and illness, such as self-care behaviors. The adaptive cognitive style of the hardy person may not necessarily dampen the physiological effects of stress, but instead may facilitate the maintenance of health behaviors, indirectly lowering risk of actual illness. Finally, as others have noted (Hull et al., 1987; Funk & Houston, 1987; Scheier & Carver, 1985), the operational definitions of hardiness are in need of refinement. The confounding with neuroticism observed in this study and others is a potentially serious problem. Relations between personality measures and health reports may not reflect actual illness; they may reflect a correlation between personality and simple somatic complaints (Cohen, 1979; Costa & McCrae, 1985, 1987; Watson & Pennebaker, in press). As long as measures of hardiness are contaminated with neuroticism, and as long as reported illness or other health behaviors (e.g., physician visits, sick days, etc.) are used as an outcome measure, studies of hardiness and health are open to serious alternative interpretations. Attention to these issues in future research will help outline the nature of the health outcomes influenced by hardiness, as well as the processes underlying such a relation.

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Received June 22, 1987
Revision received January 12, 1988
Accepted June 16, 1988 ■