Multivariate Experimental Clinical Research, Volume 11, No. 3, 1997, 109-120

A LONGITUDINAL, STRUCTURAL EQUATION ANALYSIS OF STRESS, HARDINESS, SOCIAL SUPPORT, DEPRESSION, AND ILLNESS

Jeffrey B. Brookings
Department of Psychology
Wittenberg University

Brian Bolton
Arkansas Research and Training Center
in Vocational Rehabilitation
University of Arkansas

ABSTRACT

College undergraduates (45 males, 88 females) completed measures of life stress, depression, and physical illness on two different occasions, along with measures of hardiness at Time 1 and perceived social support at Time 2. Structural equation modeling analyses provided only modest support for the hypothesis that hardiness functions prospectively to buffer the effects of stress on depression, and gave no support for the assumed mitigating effects of hardiness on the stress/physical illness relationship. Time 2 social support had the largest effect on Time 2 depression, while the best predictors of Time 2 physical illness were Time 1 physical illness (i.e., the stability coefficient) and Time 2 stress, respectively.

INTRODUCTION

Over the past 25 years, numerous studies have documented the deleterious effects of life stress on physical health and psychological well-being (for reviews, see Dohrenwend & Dohrenwend, 1978, 1981; Sarason & Sarason, 1984). Equally well-documented, however, are the relatively modest zero-order correlations between measures of stress and indexes of distress (Rabkin & Struening, 1976); that is, stressful life events typically account for no more than 9% of the variance in the dependent measures. More recently, therefore, researchers have turned their attention to a variety of personality and social characteristics — termed "resistance resources" by Antonovsky (1979) — that might buffer or moderate stress effects.

Kobasa (1979) proposed hardiness as a personality characteristic that helps protect or buffer individuals from the effects of life stress. According to Kobasa, hardy persons believe that they control events relevant to their own experience, feel committed to their activities, and view change as a stimulus or challenge to

personal growth. Studies by Kobasa and her colleagues (e.g., Kobasa, Maddi, & Kahn, 1982; Kobasa, Maddi, Puccetti, & Zola, 1985; Kobasa, Maddi, & Zola, 1983) showed that hardiness functions prospectively to buffer the effects of life stress on physical health, and Wiebe (1991) reported that hardiness moderated the effects of a stress-inducing task on affect and physiological measures. However, other reports indicate that hardiness has direct (but not stress-buffering) effects on physical illness (Wiebe & McCallum, 1986), and that hardiness predicts psychological distress, but not physical illness (Nowack, 1989).

Prompted perhaps by the inconsistent findings, several reviews of the hardiness literature have been published recently, and the reviews have been quite critical. Funk and Houston (1987) were unable to confirm the Hardiness Scale's proposed three-subscale structure, and they argued that the statistical procedures used in many hardiness studies (e.g., analysis of variance) are inappropriate because they do not allow the researcher to assess the contribution of hardiness, while controlling for the influence of other predictors with which it is typically correlated (e.g., social support). Hull, Van Treuren, and Virnelli (1987) found that the challenge component was unrelated to health outcomes, while (lack of) commitment and control had direct, rather than stress-buffering effects, and they identified several psychometric problems with the Hardiness Scale. Finally, Cohen and Edwards (1989) concluded that the evidence for the stress-buffering properties of hardiness is mixed, in part because the correlations among the scales used to measure the three hardiness components are not large enough to justify the calculation of a total score.

Typically, hardiness researchers have used prospective designs, which take prior symptom levels into account when predicting symptoms at a later date. Unfortunately, the statistical techniques used in most of these studies (e.g., analysis of variance, multiple regression, path analysis) do not allow determination of the extent to which non-significant findings are attributable to psychometric deficiencies in the Hardiness Scale (or in whatever scale the researcher uses to operationalize the hardiness construct), the absence of relationships between hardiness and well-being, or both.

A more appropriate data-analytic technique for addressing these issues is structural equation modeling (SEM). Briefly, SEM involves the specification and evaluation of: 1) a measurement model, representing relationships between measured variables (e.g., questionnaire items) and latent variables (i.e., factors); and 2) a structural model, which posits causal relations among the latent variables (i.e., constructs). The principal strength of SEM is its incorporation of latent variables represented by multiple indicators, which removes measurement error from the regression coefficients estimated in the structural model.

The purpose of this study was to investigate the effects of life stress, hardiness, and social support on psychological depression and physical illness, using a prospective design. Structural equation modeling was used to examine the relative contributions of the predictor variables.

METHOD

SUBJECTS

The sample consisted of 133 college undergraduates (45 male, 88 female) at a midwestern liberal arts college, who provided complete data on two different

occasions. Subjects participated at Time 1 as part of an introductory psychology course requirement, and participation at Time 2 was voluntary. Subjects ranged in age from 17 to 23 ($\underline{M}$ = 18.1), and 98% (130) were caucasian.

MEASURES

At Time 1, subjects completed the following self-report measures:

1. The revised Hardiness Scale (Kobasa & Maddi, 1982) consists of 36 items in a four-point, agree-disagree format. Item scores are standardized (z-scores) and summed to generate scores for the composite Hardiness Scale and three subscales: Control (16 items), Commitment (12 items), and Challenge (8 items).

2. The student version of the Life Experiences Survey (LES; Sarason, Johnson, & Siegel, 1978) lists 44 stressful life events. Subjects checked the events that they had experienced within the past 12 months and on a seven-point scale estimated the perceived positive or negative impact of each event upon them. Total stress scores were calculated by summing the impact scores of those experienced events perceived as having negative effects.

3. The Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977) lists 20 depression-related symptoms. Subjects indicated how frequently they had experienced each symptom during the past week, ranging from "Rarely or none of the time" (0) to "Most or all of the time" (3).

4. The Seriousness of Illness Rating Scale (SIRS; Wyler, Masuda, & Holmes, 1968) is a weighted checklist measure of physical illness. Subjects checked those illnesses (from a list of 121) they had had during the past 12 months. Then, total illness scores were computed by summing the weights associated with the checked illnesses.

At Time 2, subjects again completed the LES, CES-D, and SIRS, along with the college version of the Interpersonal Support Evaluation List (ISEL; Cohen & Hoberman, 1983), which consists of 48 true-false items scored on four subscales (Tangible, Belonging, Appraisal, Self-Esteem) and a total support scale (see Brookings & Bolton, 1988). Descriptive statistics for the scales and correlations among them are shown in Tables 1 and 2, respectively.

PROCEDURE

Subjects completed the Time 1 battery in small-group sessions, and the Time 2 tests individually. Followup intervals ranged from 6 to 12 months. There were no significant differences on any of the Time 1 or Time 2 measures as a function of followup interval, and multiple regression analyses indicated that length of followup interval had no effect on the predictor/criterion relationships. Females scored higher than males on Time 1 depression, but there were no differences on Time 1 stress and hardiness, or on Time 2 stress, social support, or depression. Therefore, the structural equation modeling analyses were performed on the correlation matrix for the combined groups, using LISREL VI (Jöreskog & Sörbom, 1984).

TABLE 1
MEANS, STANDARD DEVIATIONS, AND INTERNAL CONSISTENCY RELIABILITIES FOR THE MEASURED VARIABLES

Variable	No. of items	Time 1			Time 2		
		M	SD	r[a]	M	SD	r[a]
1. Hardiness[b]							
Control	16	.40	6.97	.72	-	-	-
Commitment	12	.28	5.34	.65	-	-	-
Challenge	8	-.11	3.69	.49	-	-	-
Total	36	.63	10.71	.71	-	-	-
2. LES	44	8.88	6.31	-	8.01	8.95	-
3. CES-D	20	16.30	9.39	.89	14.71	9.84	.91
4. ISEL							
Tangible	12	-	-	-	10.52	1.91	.69
Belonging	12	-	-	-	9.20	2.07	.61
Appraisal	12	-	-	-	10.69	2.11	.83
Self-Esteem	12	-	-	-	8.90	1.95	.64
Total	48	-	-	-	39.32	6.10	.86
5. SIRS	121	1332.40	806.42	-	1103.85	813.65	-

Note. N = 133. LES — Life Experience Scale; CES-D — Center for Epidemiological Studies Depression Scale; ISEL — Interpersonal Support Evaluation List; SIRS — Seriousness of Illness Rating Scale.

[a] Reliabilities for the Hardiness and CES-D scales are alpha coefficients; reliabilities for the ISEL subscales and ISEL total are KR20s. Because the LES and SIRS are checklist measures, internal consistency reliabilities are not meaningful.

[b] Means and standard deviations for the Hardiness Scale and subscales are based on sums of standardized item scores.

STRUCTURAL EQUATION MODELING (SEM) ANALYSES

The first analysis consisted of an assessment of the measurement model, which specified relationships between the latent variables and their respective measured indicators. Then, for the structural model, an initial model with all paths fixed at zero was modified by adding causal paths in a pre-determined sequence. First, causal paths connected Time 1 stress and depression factors to the corresponding factors at Time 2, to assess the stability of these constructs. Next, all other Time 1/Time 2 causal paths were assessed. Finally, within-time causal paths were hypothesized, based on the time referents of the measures (see Figure 1).

TABLE 2
CORRELATIONS AMONG THE MEASURED VARIABLES AT TIMES ONE (1) AND TWO (2)

Variable	1	2	3	4	5	6	7	8
1. LES (1)	-							
2. Hardiness (1)[a]	-193	-						
3. CES-D (1)	313	-274	-					
4. SIRS (1)	270	-132	284	-				
5. LES (2)	474	-048	302	239	-			
6. ISEL (2)	-264	140	-503	-172	-332	-		
7. CES-D (2)	406	-182	541	263	564	-554	-	
8. SIRS (2)	270	-087	207	615	511	-244	391	-

Note. Decimals are omitted. Correlations > .170 are significant at .05 (two-tailed); correlations > .230 are significant at .01. N = 133.

[a] The Challenge subscale is not included in this composite.

After each analysis, non-significant paths were fixed at zero in subsequent analyses. (For a detailed description of this model-building strategy, see Aneshensel & Frerichs, 1982).

Correlations between the depression and illness measures (CES-D and SIRS) were statistically significant ($p < .05$) but modest in magnitude. Therefore, the depression and illness data were analyzed separately, and the presentation of findings will focus primarily on depression.

PRELIMINARY ANALYSES

Because the Hardiness Challenge subscale was uncorrelated with the Control and Commitment subscales, and research by Hull, et al. (1987) indicated that Challenge is unrelated to health outcomes, this subscale (8 items) was deleted from the Hardiness composite. (Deleting these items increased the internal consistency reliability of the Hardiness composite from .71 to .79.) Then, to ensure a sufficient number of measured indicators for each latent variable, while maintaining an acceptable ratio of subjects to variables: 1) the 28 Control and Commitment items were allocated to nine within-subscale miniscales or "parcels" for the SEM analyses; 2) the 20 CES-D items were allocated to four subscales (Impaired Motivation, Positive Affect, Negative Affect, Impaired Relations), based on the factor structure reported by Harlow, Newcomb, and Bentler (1986); and 3) the social support construct was represented jointly by the four ISEL subscales (inter-subscale r's ranged from .30 to .85). This resulted in a total of 23 measured varia-

bles (listed in Table 3) for the SEM analyses. Finally, the Time 1 and Time 2 stress and illness constructs were each represented by only a single indicator (LES and SIRS total scores, respectively). Therefore, following the recommendations of Herting (1985), the factor loadings and error/uniquenesses of these indicators were fixed at .90 and .19, respectively.

FIT INDEXES

Because no single index is an infallible indicator of model adequacy, the measurement model was evaluated using the following three indexes: 1) the χ^2/df ratio (values of 2 or less indicate adequate fit); 2) the Root Mean Square Residual (RMSR), reflecting the average residual difference between the model-generated and sample correlation matrices (values less than .10 suggest adequate fit); and 3) the Tucker-Lewis Index (TLI), which compares the fit of a model relative to a null model (one which assumes that the measured variables are uncorrelated). TLI values > .90 are generally regarded as indicating good fit. Of these indexes, the χ^2/df ratio is most influenced by sample size (Marsh, Balla, & McDonald, 1988).

For the structural models, fit was assessed using the χ^2/df ratio, RMSR, and a new fit index derived by Mulaik, et al. (1989), called the relative normed fit index (RNFI). The RNFI provides for the assessment of structural models — independent of the fit (or lack of fit)of the measurement model — by comparing the fit of the model relative to a "latent variable null model" that constrains correlations among the latent variables to zero. This makes it possible to evaluate assumed causal relations between hardiness and other constructs (e.g., depression, illness), while taking account of possible psychometric deficiencies in the Hardiness Scale.

RESULTS AND DISCUSSION

MEASUREMENT MODEL

For the depression data analyses, the fit indexes indicated that the overall fit of the six-factor model to the data was adequate (χ^2/df = 1.49, RMSR = .08, TLI = .88), and the loadings of all measured variables on their respective factors (see Table 3) were statistically significant ($p < .05$). The fit of the six-factor measurement model was adequate for the illness data as well (χ^2/df = 1.17, RMSR = .07, TLI = .95).

STURCTURAL MODELS

For the depression data, unstandardized regression coefficients for statistically significant ($p < .05$), direct causal effects among the latent variables are shown in Figure 1 for the best-fitting structural model (χ^2/df = 1.50, RMSR = .09, RNFI = .98). Time 1 stress and hardiness had significant effects on Time 1 depression, and the effect of hardiness on Time 1 stress was significant as well. At Time 2, stress and perceived social support had significant effects on depression, and there was a significant effect of stress on social support. The only significant Time 1/Time 2 direct effects were for Stress-1 on Stress-2 and Depression-1 on Support-2. In contrast to previous longitudinal studies, Time 1 depression had no direct effect on Time 2 depression, although it had a large indirect effect via Time 2 social support.

TABLE 3
ESTIMATED FACTOR LOADINGS AND ERROR/UNIQUENESSES FOR THE MEASUREMENT MODEL

	Factor Loadings						
	Time 1			Time 2			
Variable	Hardy	Stress	Depress	Stress	Support	Depress	Error/ Uniquenesses
1. Control_P1	.50						.75
2. Control_P2	.48						.77
3. Control_P3	.57						.68
4. Control_P4	.44						.81
5. Control_P5	.46						.79
6. Commit_P1	.51						.74
7. Commit_P2	.58						.66
8. Commit_P3	.52						.73
9. Commit_P4	.58						.66
10. LES (1)		.90					.19
11. Motivate (1)			.79				.40
12. Pos. Affect (1)			.71				.45
13. Neg. Affect (1)			.83				.42
14. Relations (1)			.76				.56
15. LES (2)				.90			.19
16. Tangible					.58		.67
17. Belonging					.75		.44
18. Appraisal					.57		.68
19. Esteem					.77		.41
20. Motivate (2)						.79	.36
21. Pos. Affect (2)						.71	.56
22. Neg. Affect (2)						.83	.25
23. Relations (2)						.76	.33

Note. Blanks indicate parameters fixed at zero. Variables 1-9 are the Hardiness Scale parcels, variables 11-14 and 20-23 are the CES-D subscales for Times 1 and 2, respectively, and variables 16-19 are the ISEL subscales. Loadings of the stress measures — LES (1) and LES (2) — on their respective factors were fixed at .90, and their error uniquenesses were fixed at .19 (see Herting, 1985). Loadings of corresponding CES-D subscales on the Time 1 and Time 2 Depression factors were constrained to equality; none of these constraints degraded significantly the fit of the model.

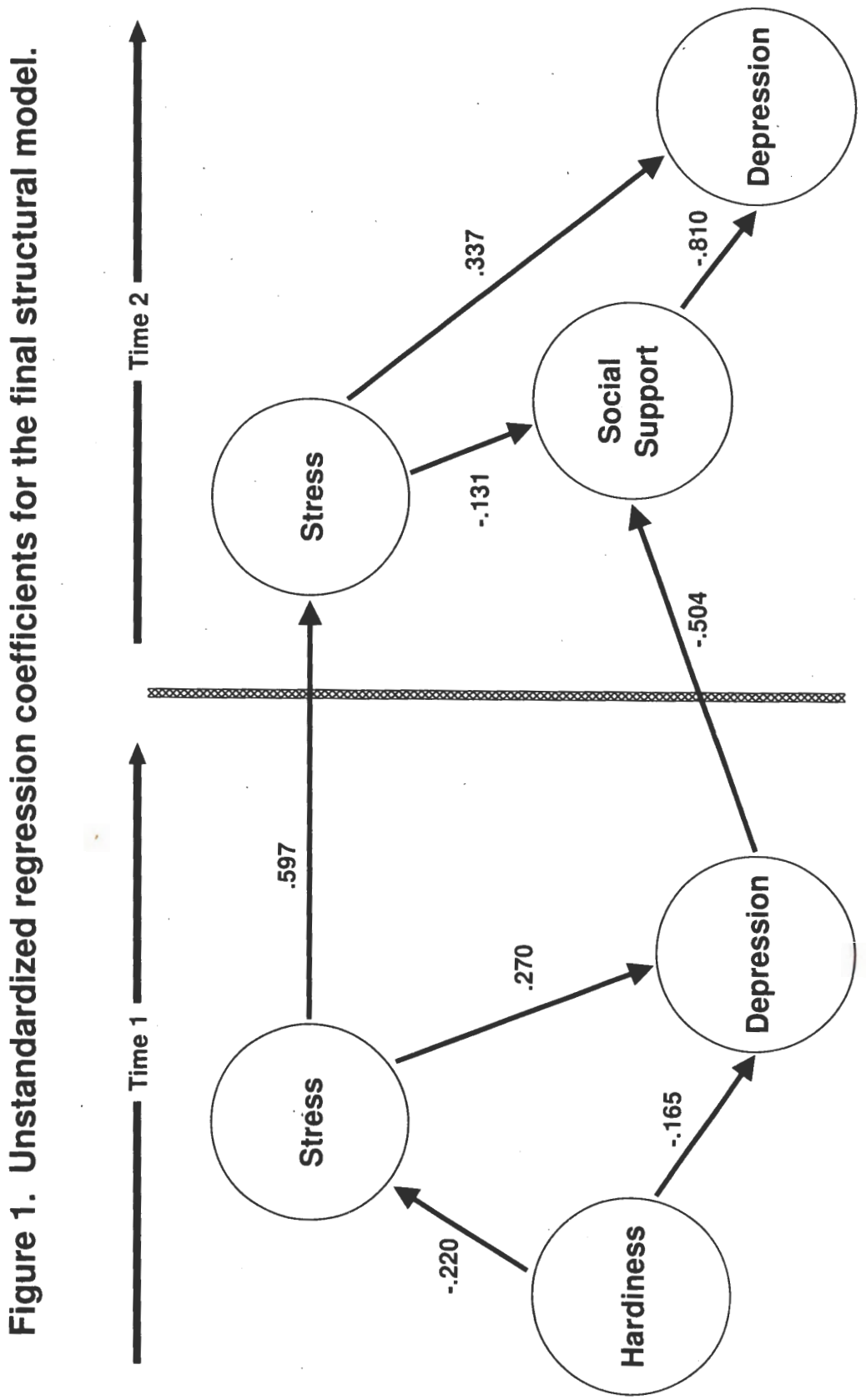

Figure 1. Unstandardized regression coefficients for the final structural model.

For the best-fitting structural model of the illness data ($\chi^2/df = 1.21$, RMSR = .09, RNFI = .97), the best predictors of Time 2 illness were Time 1 illness (.678) and Time 2 stress (.596). There were no statistically significant within- or across-time effects for hardiness, and no effect of Time 2 social support on Time 2 illness.

Direct, indirect, and total effects of the latent predictor variables on depression at Times 1 and 2 are summarized for the best-fitting models in Table 4. Hardiness had relatively modest total effects on depression at Times 1 and 2. Time 1 stress and depression had substantial total effects on Time 2 depression, even though neither had significant direct effects, while social support had — by far — the largest direct effect on Time 2 depression. For physical illness, the picture was simpler; the largest total effect on Time 2 illness was the direct effect of Time 1 illness (i.e., the stability coefficient), while lesser contributions were made by Time 2 and Time 1 stress.

TABLE 4
DIRECT, INDIRECT, AND TOTAL EFFECTS OF THE LATENT PREDICTOR VARIABLES ON DEPRESSION AT TIMES 1 AND 2

	Depression-1			Depression-2		
Latent Predictor Variable	Direct	Indirect	Total	Direct	Indirect	Total
Hardiness-1	-.165	-.060	-.225		-.150	-.150
Stress-1	.270		.270		.375	.375
Depression-1					.409	.409
Stress-2				.337	.106	.443
Support-2				-.810		-.810

Note. Effects were calculated from the unstandardized solution. Blanks indicate non-significant paths, which were fixed at zero in the final model.

CONCLUSIONS

The results of this study provided only modest support for the hypothesis that hardiness functions prospectively to mitigate the effects of stress on depression, and gave no support for the assumed mitigating effects of hardiness on the stress/physical illness relationship. The only significant causal paths involving hardiness were within-time effects on stress and depression at Time 1, and the total effects of hardiness on Time 2 depression (all indirect effects) were quite small. The total effects of stress and Time 1 depression on Time 2 depression

were considerably larger, in part because of indirect links via social support, while the strongest predictors of Time 2 physical illness were Time 1 physical illness and — to a slightly lesser extent — Time 2 stress.

These results do not appear to be attributable to reliability problems with the Hardiness Scale; as noted earlier, the SEM procedure estimates relationships among the latent variables, assuming error-free measurement. Rather, the findings indicate that hardiness — as operationalized by the revised Hardiness Scale — explained little of the variance in depression and illness, once the effects of other constructs (prior depression and physical illness, social support) were taken into account.

These results should be interpreted with caution, for at least three reasons. First of all, the stress (LES) and social support (ISEL) measures used in this study assess the perceived impact of stressful life events and the perceived availability of social support, respectively. Consequently, the substantial total effects of stress and social support on depression may reflect — in part — redundancy in the predictor and criterion measures (see Dohrenwend & Shrout, 1985; Monroe & Steiner, 1986). Secondly, statistical confirmation of a model does not imply rejection of all possible competing models (Cliff, 1983), and correlational data — even data obtained from longitudinal designs — are inadequate for drawing unambiguous causal conclusions (Breckler, 1990). Finally, because repeated statistical analyses on a data set raise a variety of logical and statistical issues and may not, in any event, lead to discovery of the "correct" model (see MacCallum, 1986), cross-validation of our best-fitting models (Cudeck & Browne, 1983) would strengthen the findings.

REFERENCES

Aneshensel, C. S., & Frerichs, R. R. (1982). Stress, support, and depression: A longitudinal causal model. *Journal of Community Psychology, 10*, 363-376.

Antonovsky, A. (1979). *Health, stress, and coping*. San Francisco, CA: Jossey-Bass.

Breckler, S. J. (1990). Applications of covariance structure modeling in psychology: Cause for concern? *Psychological Bulletin, 107*, 260-273.

Brookings, J. B., & Bolton, B. (1988). Confirmatory factor analysis of the Interpersonal Support Evaluation List. *American Journal of Community Psychology, 16*, 137-147.

Cliff, N. (1983). Some cautions concerning the application of causal modeling methods. *Multivariate Behavioral Research, 18*, 115-126.

Cohen, S., & Edwards, J. R. (1989). Personality characteristics as moderators of the relationship between stress and disorder. In R. W. J. Neufeld (Ed.), *Advances in the investigation of psychological stress* (pp. 235-283). New York: Wiley.

Cohen, S., & Hoberman, H. (1983). Positive events and social supports as buffers of life change stress. *Journal of Applied Social Psychology, 13*, 99-125.

Cudeck, R., & Browne, M. W. (1983). Cross-validation of covariance structures. *Multivariate Behavioral Research, 18*, 147-167.

Dohrenwend, B. S., & Dohrenwend, B. P. (1978). Some issues in research on stressful life events. *Journal of Nervous and Mental Diseases, 166*, 7-15.

Dohrenwend, B. S., & Dohrenwend, B. P. (1981). *Stressful life events and their contexts*. New York: Neale Watson.

Dohrenwend, B. P., & Shrout, P. E. (1985). "Hassles" in the conceptualization and measurement of life stress variables. *American Psychologist, 40*, 780-785.

Funk, S. C., & Houston, B. K. (1987). A critical analysis of the Hardiness Scale's validity and utility. *Journal of Personality and Social Psychology, 53*, 572-578.

Harlow, L. L., Newcomb, M. D., & Bentler, P. M. (1986). Depression, self-derogation, substance abuse, and suicide ideation: Lack of purpose in life as a mediational factor. *Journal of Clinical Psychology, 42*, 5-21.

Herting, J. R. (1985). Multiple indicator models using LISREL. In H. M. Blalock, Jr. (Ed.), *Causal models in the social sciences* (2nd ed.) (pp. 263-319). New York: Aldine.

Hull, J. G., Van Treuren, R. R., & Virnelli, S. (1987). Hardiness and health: A critique and alternative approach. *Journal of Personality and Social Psychology, 53*, 518-530.

Jöreskog, K. G., & Sörbom, D. (1984). LISREL VI: *Analysis of linear structural relationships by the method of maximum likelihood*. Mooresville, IN: Scientific Software, Inc.

Kobasa, S. C. (1979). Stressful life events, personality, and health: An inquiry into hardiness. *Journal of Personality and Social Psychology, 37*, 1-11.

Kobasa, S. C., & Maddi, S. R. (1982, November). [*The Revised Hardiness Scale*]. (Available from Suzanne Kobasa, Department of Psychology, CUNY Graduate School & University Center, 33 W. 42nd St., New York, NY 10036)

Kobasa, S. C., Maddi, S. R., & Kahn, S. (1982). Hardiness and health: A prospective study. *Journal of Personality and Social Psychology, 42*, 168-177.

Kobasa, S. C. O., Maddi, S. R., Puccetti, M. C., & Zola, M. A. (1985). Effectiveness of hardiness, exercise and social support as resources against illness. *Journal of Psychosomatic Research, 29*, 525-533.

Kobasa, S. C., Maddi, S. R., & Zola, M. A. (1983). Type A and hardiness. *Journal of Behavioral Medicine, 6*, 41-51.

MacCallum, R. (1986). Specification searches in covariance structure modeling. *Psychological Bulletin, 100*, 107-120.

Marsh, H. W. Balla, J. R., & McDonald, R. P. (1988). Goodness-of-fit indexes in confirmatory factor analysis: The effect of sample size. *Psychological Bulletin, 103*, 391-410.

Monroe, S. M., & Steiner, S. C. (1986). Social support and psychopathology: Interrelations with preexisting disorder, stress, and personality. *Journal of Abnormal Psychology, 95*, 29-39.

Mulaik, S. A., James, L. R., Van Alstine, J., Bennett, N., Lind, S., & Stilwell, C. D. (1989). Evaluation of goodness-of-fit indices for structural equation models. *Psychological Bulletin, 105*, 430-445.

Nowack, K. M. (1989). Coping style, cognitive hardiness, and health status. *Journal of Behavioral Medicine, 12*, 145-158.

Rabkin, J., & Struening, E. (1976). Life events, stress, and illness. *Science, 194*, 1013-1020.

Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385-401.

Sarason, I. G., Johnson, J. H., & Siegel, J. M. (1978). Assessing the impact of life changes: Development of the Life Experiences Survey. *Journal of Consulting and Clinical Psychology, 46*, 932-946.

Sarason, I. G., & Sarason, B. R. (1984). Life changes, moderators of stress, and health. In A. Baum, S. E. Taylor, & J. E. Singer (Eds.), *Handbook of psychology and health: Social psychological aspects of health* (Vol. 4) (pp. 279-299). Hillsdale, NJ: Erlbaum.

Wiebe, D. J. (1991). Hardiness and stress moderation: A test of proposed mechanisms. *Journal of Personality and Social Psychology, 60*, 89-99.

Wiebe, D. J., & McCallum, D. M. (1986). Health practices and hardiness as mediators in the stress-illness relationship. *Health Psychology, 5*, 425-438.

Wyler, A. R., Masuda, M., & Holmes, T. H. (1968). Seriousness of Illness Rating Scale. *Journal of Psychosomatic Research, 11*, 363-375.

Author Notes

This research was supported by a Faculty Research Grant from Wittenberg University to the first author.

An earlier version of this article was presented at the American Psychological Association meeting in Boston, MA, on August 13, 1990.

Address requests for reprints and other correspondence to Jeffrey B. Brookings, Psychology Department, Wittenberg University, P.O. Box 720, Springfield, OH 45501. email: jbrook@wittenberg.edu.