PSYCHOLOGICAL HARDINESS AND BIOCHEMICAL MARKERS OF ACUTE STRESS

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Dissertation Prepared for the Degree of

DOCTOR OF PHILOSOPHY

UNIVERSITY OF NORTH TEXAS
August 2001

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McCoy, Paula K., Psychological Hardiness and Biochemical Markers of Acute Stress. Doctor of Philosophy (Psychology), August 2001, 68 pp. 4 tables, 8 Figures, references.

The establishment of physiological norms for psychologically hardy vs. non-hardy individuals was attempted by examination of levels of salivary cortisol and urinary norepinephrine before and after a mid-term examination stressor. Normative data was collected on the reported frequency of stressors and their severity one week prior to the examination, and self-reported ratings of stress immediately prior to the examination. Performance on the examination as a function of hardiness was explored. Associations between demographic variables and psychological hardiness were also studied. Results from this study were inconclusive in establishing physiological norms for psychologically hardy individuals. Associations were found between: 1) hardiness and frequency of stressors; 2) hardiness and age; and 3) self-reported ratings of stress and anxiety as measured by the State-Trait Anxiety Inventory (STAI).

ACKNOWLEDGEMENTS

With thanks and praise to God, who is the source of all wisdom; to Mike, for his constant love and support, and for helping to make this dream a reality; to Fr. Juan, who epitomizes hardiness, for assistance with graphics and for spiritual guidance during the rough spots; to Fr. Ken, for encouraging me to take this step and for spiritual direction through all the trials in my life; to Ernest Harrell, Mike Richmond, the Welch Foundation, and the Department of Psychology for funding my research; to Aunt Charlotte and Uncle Tony, for their emotional and financial support; to Scott for assistance in data collection and for being the best friend and colleague anyone could hope for; to Paul Lambert and Patti Price for allowing me to collect data in their classes; to Clarice and Shannon for all the laughter and for keeping me grounded; to Kim Kelly, Joe Doster, Rob Hawkins, Joe Wyatt, and Sigrid Glenn, who are the finest psychologists I know, for encouragement and critical comments on this journey; to Suzanne Ouellette (Kobasa), the creator of the hardiness construct for her genius, and without whom my current research would not be possible; and last but not least, to my clients, my first and best teachers about hardiness. This dissertation is dedicated to the memory of Sammy Trump and Carl McComas.

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INTRODUCTION

Since ancient times, physicians and philosophers, as well as patients, have contemplated the existence of a relationship between personality and illness. Hippocrates believed the four bodily humors (blood, black bile, yellow bile, and phlegm) were the basis of personality and Galen conceptualized these same substances as the causes of disease (Allport, 1961). In modern times Freud was a proponent of the personality-disease relationship and claimed the cure of numerous "conversion" disorders such as hysterical paralysis or hysterical blindness (Freud, 1955). Friedman and Booth-Kewley (1974) became so convinced of this connection they posited the concept of a "disease-prone" personality. Other researchers have focused on the relationship between personality factors and specific disorders such as the "Type A personality" and coronary heart disease (Friedman and Rosenman, 1974), the "migraine personality" (Adams, Feuerstein, & Fowler, 1980), the "asthmatic personality" (Creer, 1978) or the "arthritic personality" (Anderson, Bradley, Young, McDaniel & Wise, 1985).

While the development of illness or disease is assuredly not attributable to any single factor given the potential for multiple environmental influences, the interrelatedness of physiological systems in the body, and genetic predispositions, personality dimensions having a beneficial/deleterious effect on health should be explored in an effort to maximize preventive intervention

strategies. Research on personality and health has historically examined direct and indirect mechanisms of action of personality as the cause of illness. Direct influences of personality on illness include those approaches, which believe personality affects some physiological process such as immune competency or vulnerability to stress. Indirect influences encompass the theories which view personality as an influence on the performance of unhealthy behaviors (such as individuals who respond to anxiety by overeating, resulting in obesity, which contributes to the development of diabetes) and biological third variables (Krantz & Durel, 1983; Kahn, Kornfeld, Frank, Heller, & Hoar, 1980). An example of a biological third variable would be the discovery of a hyper-responsive nervous system as an underlying factor in the development of an anxious personality and a hyper-responsive nervous system as an underlying factor in the development of heart disease; chronic anxiety would be a marker for heart disease, but the anxiety itself would not necessarily play a causal role in the development of heart disease.

In the proposed study, the role of personality in vulnerability to stress was explored. One of the more promising personality constructs examining this relationship is that of psychological hardiness (Kobasa, 1979). Hardiness theory suggests individuals who experience significant stress without becoming physically ill have a personality structure different from persons who develop illness in response to stress. Hardy persons are assumed to possess three characteristics: 1) the belief they can control or influence their environment, 2) a

strong commitment to self and 3) the ability to view change as a challenge to personal growth. Individuals low on the hardiness dimension are believed to have greater vulnerability to stress as their experience of stress activates sympathetic and neuroendocrine systems resulting in a greater magnitude of physiological arousal than individuals scoring higher on this dimension. Frequent or prolonged periods of arousal are presumed to cause illness by placing excessive strain on bodily systems (Krantz & Manuck, 1984; Menkes et al., 1989) and impairing immune functioning (O'Leary, 1990; Jemmott & Locke, 1984).

Psychological hardiness operates via cognitive appraisal. Cognitions consist of an individual's ideas, beliefs, thoughts, and images about a particular subject or event. These may be formed from previous experience with the phenomenon in question, from vicarious learning, or may be a reflection of one's values or a general orientation to the world. When one makes a cognitive appraisal of a situation, one takes these cognitions and makes a subjective judgment about the occurrence of an objective event. For purposes of this discussion, two appraisals an individual may make will be considered--threat vs. non-threat. Psychological stress and the concomitant physiological arousal are believed to occur when an event is appraised as threatening and beyond one's abilities to meet the demands it presents (Lazarus and Folkman, 1984). A physiological chain of events occurs when an individual has made a "threat appraisal" of a given situation (Davis, Eshelman, & McKay, 1995). Greater threats or personal demands lead to a more pronounced physiological response; this resulting

physiological response is generally referred to as the “fight or flight response” (Cannon, 1932). The “fight or flight response” is a series of biochemical changes which prepare an organism to deal with threats or danger. When a threat is perceived (real or imagined), the cerebral cortex sends an alarm message to the hypothalamus. The hypothalamus then stimulates the sympathetic nervous system (SNS), causing an increase in heart rate, breathing rate, muscle tension, metabolism, blood pressure, and release of epinephrine and norepinephrine. Almost simultaneously, the adrenal glands start to secrete glucocorticoids. The short term effect of release of glucocorticoids is increased glucose metabolism which prepares the body to deal with the stressor, and, in the case of physical injury, suppresses the inflammation response and eventually assists in returning the body to homeostasis. The long term effects of continued glucocorticoid release include inhibition of digestion, reproduction, growth, tissue repair, and the responses of the immune system (immunosuppression). Illness may occur subsequent to prolonged immunosuppression. Cortisol inhibits many functions of lymphocytes, macrophages and leukocytes and may affect their trafficking patterns (Black, 1994). Cortisol elevations decrease the production of many cytokines and mediators of inflammation and decreases the effects of certain inflammatory molecules on various tissues (Chrousos & Gold 1992; Munck & Guyne, 1986; Munck, Guyne, & Holbrook, 1984). Continued elevation of norepinephrine and epinephrine levels may produce changes in lymphocyte, monocyte and leukocyte functions. Several stress experiments suggest plasma

epinephrine level is inversely related to immune system functions (Dantzer & Kelley, 1989; Kiecolt-Glaser, Cacioppo, Malarkey & Glaser, 1992). Both lymphocytes and macrophages have β2-adrenergic receptors, and norepinephrine, epinephrine and β-adrenergic agonists generally down regulate immune system function, especially late in the immune response, by a decrease in the production of and response to cytokines and general inhibiton of macrophage and lymphocyte function (Black, 1994).

Almost every system in the body can be damaged by stress. Stress-induced changes in the lungs increase the symptoms of asthma, bronchitis, and other respiratory conditions. Loss of insulin during the stress response may contribute to the onset of adult diabetes. Stress suspends tissue repair which in turn results in decalcification of the bones, osteoporosis, and susceptibility to fractures. A prolonged stress response can worsen conditions such as arthritis, chronic pain, and diabetes. The continued release and depletion of norepinephrine during chronic stress may contribute to depression (Davis, Eshelman and McKay, 1995). As previously stated, as long as the mind perceives a threat, physiological arousal continues. If the arousal continues for extended periods of time, the risk of a stress-related illness or disease is increased.

Hardiness is believed to reduce one's vulnerability to stress at several junctures in this process. First, the beliefs and expectations previously described are hypothesized to reduce the likelihood that any given event is appraised as stressful, thereby diminishing its potential to induce pathophysiological arousal

(Kobasa, Maddi, Puccetti and Zola, 1985). Research support for this theory includes studies by Rhodewalt and Zone (1989) and Rhodewalt and Agustsdottir (1984) in which high-hardy individuals reported experiencing the same types of life events as low-hardy individuals but rated these events as more positive and controllable. Several studies revealed through path analyses that stress appraisals mediate the relationship between hardiness and self-reported physical symptoms (Rhodewalt and Zone, 1989; Roth et al., 1989; and Wiebe, 1991) and between hardiness and reported health practices (Wiebe and McCallum, 1986). More specifically, hardiness has been associated with low levels of negatively appraised stress which has been associated with fewer symptom reports and more positive health behaviors.

Secondly, hardiness is believed to influence the types of coping strategies used when an event is perceived as stressful. High-hardy individuals are hypothesized to engage in "transformational coping" in which an objective stressor is modified by cognitive and behavioral actions to become more positive (Gentry and Kobasa, 1984; Maddi and Kobasa, 1981). Low-hardy individuals are believed to engage in maladaptive coping strategies such as avoidance and denial. Additional research provides evidence suggesting high-hardy individuals adopt a variety of strategies which are more active and problem-focused, while low-hardy individuals are more likely to avoid and deny (by pretending a stressful event did not occur or eating and drinking to forget about it) (Carver, Scheier, and Weintraub, 1989; Williams, Wiebe and Smith, 1992). An additional finding was

that adaptive coping processes mediated the hardiness effects on symptom reporting (Williams et al., 1992).

To date, there have been relatively few psychophysiological studies investigating the stress-buffering hardiness model. Contrada (1989) found high-hardy males displayed smaller diastolic blood pressure responses to a mirror-tracing task than did low-hardy males. Wiebe studied control-related appraisals of stressors, heart rate, skin conductance and finger pulse volume. The results of this investigation revealed high-hardy men who perceived the stressors as more controllable responded to the stressors with smaller increases in heart rate and skin conductance and smaller decreases in finger pulse volume than did low-hardy men. Appraisal manipulations had negligible effects on women.

The proposed study will investigate the stress buffering effects of hardiness by examining baseline levels of and elevations in catecholamines and cortisol (both of which may be viewed as end products of stress-activated responses of the SNS and hypothalamic-pituitary-adrenal (HPA) axis, respectively), in response to an academic examination stressor. Central nervous system (CNS) perception and processing of stressor stimulation is immediate (Kusnecov & Rabin, 1994). The two main systems which have been characterized most in relation to stressor-induced activation are the SNS and the HPA axis (Axelrod & Reisine, 1984; Whitnall, 1993). SNS activation occurs within seconds (Lefkowitz, Hoffman & Taylor, 1990), evidenced by the rapid appearance in blood of the two major neurotransmitters of the SNS, the

catecholamines epinephrine and norepinephrine (Kusnecov & Rabin, 1994; Kvetnansky, Fukuhara et al., 1993; Pacak et al., 1993). There is consensus among researchers that the best indicator of the rapidity of the norepinephrine sympathetic response to stress is the increase in heart rate due to stimulation of adrenergic receptors by norepinephrine (Manuck, Cohen, Rabin, Muldoon, & Bachen, 1991). The SNS innervates a variety of vital organ and tissues and plays an active role in the homeostatic function of the autonomic nervous system. Primary and secondary lymphoid organs, particularly the spleen, are richly innervated with noradrenergic sympathetic fibers (Ackerman, Bellinger, Felten, & Felten, 1991; Felten & Felten, 1991). It has been documented that numerous catecholaminergic varicosities terminate in the white pulp of the parenchyma of the spleen, that lymphocytes and monocytes possess β-adrenergic receptors for catecholamines and that incubation of lymphocytes, monocytes, and natural killer (NK) cells with epinephrine and norepinephrine exerts functional alterations (Roszman & Carlson, 1991). Short-term restraint in rats results in up to 50% β-adrenergic receptor redistribution from the peripheral mononuclear cell surface to the cytoplasm without affecting the total number of receptors; however, long-term stress results in receptor downregulation as indicated by a reduction in specific binding sites. Similar changes occur following immunization of mice with sheep red blood cells (SRBC) (Fuchs, Albright, & Albright, 1988). This evidence highlights the readiness of the immune system to interact and respond to neurotransmitters produced by the SNS (Kusnecov & Rabin, 1994).

The response of the HPA to stressor stimulation is relatively slow compared to the SNS. The predominant end product following activation of the HPA axis is the adrenal glucocorticosteroid cortisol. Although cortisol release is the result of a complex series of events, the primary factor is corticotropin-releasing factor (CRF), a neuropeptide that is stored in the paraventricular nucleus of the hypothalamus and released via terminal neuron projections in the median eminence. From there it travels to the anterior region of the pituitary gland where it stimulates release of adrenocorticotropic hormone (ACTH) (Whitnall, 1993). ACTH then stimulates cells in the adrenal cortex to synthesize and secrete cortisol. High levels of cortisol have been demonstrated to be immunosuppressive (Black, 1994). Immunosuppression may result in development of physical symptoms and illness if environmental challenge occurs. For example, the development of cold symptomatology in experimentally infected individuals was correlated with immunosuppression and recent stressful life events (Stone, Bovbjerg, Neale, Napoli, Valdimarsdottir, Cox, Hayden, & Gwaltney, 1992) and the progression of AIDS is believed a function of a dominant TH1 state which is influenced by psychosocial factors (Clerici and Shearer, 1993). Animal studies with BALB/c mice revealed changes in immunity and metastases of syngeneic line 1 tumor cells were related to increases in plasma epinephrine in response to a handling stressor (Moynihan, Brenner, Koota, Dreneman, Cohen, & Ader, 1990).

In summary, psychological hardiness is a construct which attempts to explain the relationships between personality, vulnerability to stress and illness/disease. An individual's personality may be "stress-buffering" if they are "high" on the hardiness dimension or may make them vulnerable to stress if they are "low" on the hardiness dimension. Vulnerability to stress results in more frequent and possibly more intense activation of the SNS and the HPA axis, which may lead to illness or disease. Cortisol levels and catecholamine levels are evidence of activation of these systems. By examining baseline levels of cortisol and catecholamines in high- and low-hardy individuals, as well as the fluctuation of these substances in response to a stressor, biochemical markers may be provided distinguishing these two groups. Such discrimination could provide crucial information needed to target individuals at risk for development of illness and to develop intervention strategies. The following hypotheses are offered:

Hypothesis 1. Hardiness will be negatively associated with baseline levels of cortisol and catecholamines.

Hypothesis 2. Hardiness will be negatively associated with elevations of cortisol and catecholamines in response to a stressor (as evidenced by lower elevations of these substances at pre-test and post-test1).

Hypothesis 3. Hardiness will be positively associated with rate of return to baseline levels of cortisol and catecholamines.

Hypothesis 4. Hardiness will be positively associated with scores on the research design exam.

Hypothesis 5. Hardiness will be negatively associated with reported frequency of daily hassles (as a result of their transformational coping style which results in decreased instances of perceiving events as stressful).

Hypothesis 6. Hardiness will be negatively associated with scores on the STAI (pre-test) and anxiety/arousal as measured by the STAI will be positively associated with anxiety/arousal as measured by a self-rating scale.

METHOD

Participants

Fifty student volunteers from an undergraduate statistics class at the University of North Texas were recruited for this study. Minority participation was encouraged. Participants received course credit for their informed participation.

Measures

Psychological.

Personal Views Survey. The Personal Views Survey (PVS) is a 50-item, third-generation hardiness measure (Hardiness Institute, 1985). A factor analysis has yielded three factors identifiable as commitment, control, and challenge (Bartone, 1989). Estimates of internal consistency for commitment, control, and challenge and total hardiness scores have ranged from 0.68 to 0.89; these components have shown positive intercorrelation with adults and college students (Bartone, 1989; Okun, Zantra and Robinson, 1988; Parkes and Rendall, 1988). The third-generation hardiness test has shown intercorrelation and validity of component and total scores with adults and high school/college adolescents (Bartone, 1991; Maddi and Hess, 1992; and Parkes and Rendall, 1988). This study examined the *composite* hardiness score in relation to variables of interest as previous research on multifaceted personality constructs revealed this approach produces a more reliable and valid assessment of the underlying variable of interest (Hull, Lehn, and Tedlie, 1991).

In addition, Carmines and Zeller (1979) reported that as the length of the scale increases, the reliability of the composite score supercedes the reliability of the individual subcomponents.

Regarding convergent validity, the third generation test has shown the predicted positive association with self-reported health status (Campbell, Amerikaner, Swank and Vincent, 1989; Okun et al., 1988) and with level of immune system T-cells (Okun et al., 1988). Regarding discriminant validity, the third-generation measure appears unrelated to social desirability bias (Parkes and Rendall, 1988).

The Hassles Scale. The Daily Hassles scale is a 50-item self-report survey contained in the Daily Hassles and Uplifts Scale (Lazarus and Folkman, 1989). This instrument is designed to measure the frequency and severity of stressful events in an individual's life. This scale may be used to investigate stress as an independent or dependent variable. As a dependent variable, stress is an individual's reaction to his or her ongoing relationships with the environment which is appraised as harmful, threatening or challenging (Lazarus and Folkman, 1989). As this appraisal component is integral to the hardiness construct, this instrument is well-suited to the present study.

This scale was developed as an alternative to the life events scales popular in the last decade, as a weakness of those scales was their focus on "powerful" life events (death of a spouse, divorce, job loss), which occur infrequently in the lives of most people. Given their infrequency, these major life events are less

than optimal standard measure of life stress for the general population under routine conditions (Lazarus and Folkman, 1989). The “Daily Hassles Scale” is believed a superior measure of psychological stress as it more accurately reflects the daily occurrences of major or minor stresses in an individual’s life which may be a source of harm, loss, threat or challenge.

The theoretical approach to the “Daily Hassles Scale” assumes that how people construe or appraise the personal significance of their encounters with the environment will determine what is psychologically stressful to them. Such appraisals need not be accurate reflections of what has actually occurred. A person’s appraisals reflect environmental circumstances as well as personality characteristics, goal hierarchies and personal beliefs, all of which may result in special sources of vulnerability to stress (Lazarus and Folkman, 1989).

Normative data on the “Daily Hassles Scale” are available on three different samples: 1) white, middle-class adults, aged 45-64 (Kanner et al., 1981) 2) college students (MacPhee, personal communication); and 3) adults aged 20-60 (Young, 1987). the reliability of Hassles frequency scores was .79 and the reliability of severity scores, .48. As the Daily Hassles Scale assesses events appraised by the person as stressful rather than objective stressors, the test items are believed to have a high degree of face and content validity and to offer a representative sampling of psychological stress for a stated time period (Lazarus and Folkman, 1989).

The State-Trait Anxiety Scale (State-Anxiety Scale). The State-Anxiety Scale (STAI Form Y-1) consists of 20 statements which evaluate feelings of apprehension, tension, nervousness and worry in respondents at the time of administration. Scores on the S-Anxiety scale increase in response to physical danger and psychological stress and decrease as a result of relaxation (Spielberger, 1983). The scale has been used extensively to assess S-Anxiety induced by stressful experimental procedures and by unavoidable real-life stressors such as imminent surgery, dental treatment, job interviews or important school tests (Spielberger, 1983).

The concept of state (and trait) anxiety was first introduced by Cattell (1966; Cattell and Scheier, 1961, 1963) and then elaborated by Spielberger (1979). Personality states are “temporal cross-sections in the stream-of-life of a person” (Thorne, 1966) and emotional reactions are expressions of personality states (Spielberger, 1972). An emotional state exists at a given moment in time and at a particular level of intensity. Anxiety states are characterized by subjective feelings of tension, apprehension, nervousness and worry, and by activation or arousal of the autonomic nervous system. The STAI has already proven useful in assessing psychological stress/test anxiety (Culler and Holahan, 1980; Guidry and Randolph, 1974; Smith et al., 1982; Tobias et al., 1974) and is believed a suitable instrument to determine the amount of stress participants will be experiencing prior to the examination stressor.

Given the transitory nature of anxiety states, measures of internal consistency such as the alpha coefficient provide a more meaningful index of the reliability of S-Anxiety scales than test-retest correlations (Spielberger, 1983). Alpha coefficients for the Form Y S-Anxiety were computed by Formula KR-20 as modified by Cronbach (1951). S-Anxiety alphas ranged from 0.90-0.94 for samples of working adults, students, and military recruits, with a median coefficient of 0.93. Individual STAI items were required to meet validity criteria at each stage of the test development process to be retained for further evaluation and validation (Spielberger, 1983). The test construction process is described by Spielberger and Gorsuch (1966) and Spielberger et al., 1970).

Self-Rating Scale. The self-rating scale was a numerical rating scale based on the Likert Scale. The Likert Scale was devised in 1932 to assess attitudes and public opinion. The original scale was a 5-point continuum anchored by the adverbs “strongly agree” at “5” and “strongly disagree” at “1.” Since its development, many researchers have modified the scale to determine subjective ratings of such diverse phenomena as severity of pain and effectiveness of intervention programs.

Physiological. Catecholamines. Urinary catecholamines were assayed using low pressure liquid chromatography (Wingo, Ennis, Lambert, & Kelly, manuscript in preparation) using a weak cation-exchange resin (Bio-Rad BioRex-70, 150-200 mesh) to selectively bind total catecholamines from each urine sample. Catecholamines were eluted by reducing the column pH and then

measured at 210 nanometers on a Hewlett-Packard 8452 A Diode Array Spectrophotometer. A linear regression plot of standard samples was used to determine sample concentrations (μg/mL) from measured light absorbance values. Additional equipment employed in these analyses were the Bio-Rad Model EP-1 Econo Pump, Bio-Rad Model 2110 Fraction Collector, Bio-Rad Econo-Column Flow Adaptor, and a Corning 240 pH meter with a Corning G-P combo with RJ electrode.

Cortisol. The samples were collected, frozen and sent to Germany for analysis. They were measured via radioimmunoassay in a method described elsewhere (Kirschbaum, Strasburger, Jammers, and Hellhammer, 1989).

Procedure

Participants completed the PVS and the Hassles portion of the Combined Hassles and Uplifts Scale in class as a group one week prior to the test date. Scores on the PVS indicated the degree of hardiness manifested by a particular individual. Scores on the Hassles Scale provided a general index of stress the participants were experiencing prior to the presentation of the exam stressor. Upon completion of the above-named instruments, a baseline saliva and urine sample were collected. On the test date, participants presented to the laboratory thirty minutes prior to the exam. After an explanation of the procedures involved, each participant supplied a urine and saliva sample prior to the exam. Urine was obtained in collection cups and saliva via salivettes. They also completed the “state” portion of the State-Trait Anxiety Scale immediately before departure

to take the exam. Participants additionally rated their level of stress regarding the exam by selecting a number from a self-rating scale with 0 = no stress regarding the exam and 10 = the most stress I have ever experienced prior to an exam. The participants then presented to class, took the exam and returned to the laboratory for the remaining procedures. Upon arrival at the lab, participants took the “state” portion of the State-Trait Anxiety Scale and supplied a urine and saliva sample. They were encouraged to sit quietly and were provided with non-controversial reading materials to read until the final urine and saliva samples were collected. The final samples were collected one-half hour following their arrival at the lab. Participants experiencing difficulty in urinating at any stage of the experiment were provided with eight ounces of water.

RESULTS

The data were analyzed using the SPSS statistical software program. Unless otherwise stated, an alpha level of 0.05 was used for all statistical manipulations.

Table 1 shows the nominal characteristics of the participants in this study and Table 2 shows the internal characteristics of the participants. Sixty-nine per cent of the participants in the study were female (n = 40) and thirty-one per cent were male (n = 18). Ninety-three per cent of the participants were Caucasian (n = 54) and seven per cent were Hispanic (n = 4). The ages of individuals participating in this study ranged from 18 to 37 years (M = 23.62). Education varied from 14-20 years of schooling (M = 15.91). Representation of participants by income level was as follows: 57.1% reported a household income of <$15,000, 26.8% reported an income of $15,000-$30,000, 12.5% reported an income between $30,000-$45,000, 1.8% reported yearly earnings between $45,000-$60,000 and 1.8% reported earnings in excess of $60,000. Linear regression analyses were conducted on scores on the PVS and all demographic variables. One significant finding emerged from these analyses. Hardiness was found to be positively correlated with age; the more hardy a person was, the older they were (this was evidenced by the association of low scores on the PVS and higher age values, $r = -0.279$, $p < 0.03$). Please refer to Table 3 for additional detail.

Testing of Hypotheses

The first hypothesis, hardiness is negatively associated with baseline levels of cortisol and catecholamines, was not supported (PVS/BLCORT r = 0.09, n = 60, p < 0.48; PVS/BLNE r = -0.18, n = 37, p< 0.28). PVS scores and baseline cortisol were virtually independent of each other and while low scores on the PVS predicted high baseline levels of norepinephrine (NE); it should be remembered, however, that hardiness is *inversely* related to scores on the PVS and the expected finding was that hardy individuals would exhibit lower baseline levels of norepinephrine (NE).

Hypothesis 2, hardiness is negatively associated with cortisol and catecholamines in response to a stressor (as evidenced by lower elevations of these substances at pre-test and post-test1), was not confirmed (PVS/difcrt1 r = 0.076, df = (1,35), n = 33, p < 0.65; PVS/difcrt2 r = 0.026, df = (1,35), n = 33, p < 0.877) and (PVS/difne1 r = 0.044, df = (1, 30), p < 0.812); PVS/difne2 r = 0.028, df = (1, 30), n = 33, p < 0.874). Please refer to Tables 3 and 4 for mean values of NE and cortisol at each data collection point.

Hypothesis 3 stated hardiness is positively associated with faster return to baseline levels of cortisol and catecholamines. In exploring this relationship an analysis of variance (ANOVA) was run to determine change in the physiological variables NE and cortisol over time. The ANOVA for NE did not reveal statistically significant change over time (F = 2.58, df = (3, 96), n = 33, p < 0.0582). The ANOVA for cortisol did reveal significant change over time (F =

18.12, df = (4,144), n = 33, $p < 0.0001$). To test the rate of return to baseline of cortisol and NE and the relationship of rate of return to hardiness, difference scores were created by subtracting each sample from baseline. A regression analysis was then run with hardiness as the predictor variable and the difference score as the criterion variable. The slope of the regression line illustrates the rate of return to baseline. No evidence was found for the relationship stated in Hypothesis 3. The results for cortisol were as follows: PVS/difcrt 1 slope = 0.047, $p < 0.65$; PVS/difcrt2 slope = -0.0176, $p < 0.88$; PVS/difcrt3 slope = -0.005, $p < 0.955$; PVS/difcrt4 slope = -0.022, $p < 0.80$; and difcrt5 slope = -0.010, $p < 0.09$. The results for NE were: PVS/difne1 slope = 0.0015, $p < 0.81$; PVS/difne2 slope = -0.0011, $p < 0.87$; PVS/difne3 slope = 0.0084, $p < 0.99$; and PVS/difne4 slope = 0.0025, $p < 0.65$.

Regarding Hypothesis 4, hardiness and scores on the academic exam are positively associated, no support was found for this relationship ($r = 0. -246$, $n = 59$, $p < 0.06$). High hardy individuals actually scored lower on the exam (see Figure 2).

Hypothesis 5, hardiness is negatively associated with reported frequency of daily hassles (low scores on the hardiness scale, which indicates a person high on the hardiness dimension, will predict low scores on the hassles scale), was confirmed ($r = 0.36$, $n = 61$, $p < 0.0001$). Low scores on the PVS were indicative of the endorsement of fewer daily hassles on the Combined Hassles and Uplifts Scales (see Figure 3).

The sixth hypothesis suggested a negative relationship between hardiness and scores on the STAI (pre-test) and a positive relationship between anxiety/arousal as measured by the STAI and anxiety/arousal on a self-rating scale. A significant, positive relationship was found between anxiety/arousal as measure by the STAI and anxiety/arousal on the self-rating scale ($r = 0.75$, $n = 53$, $p < 0.0001$). Please refer to Figure 4. The association between hardiness and scores on the STAI (pre-test) was not validated ($r = 0.02$, $n = 51$, $p < 0.867$). PVS scores and scores on the STAI were virtually independent of each other (see Table 6).

For the distribution of scores on the Personal Views Survey, the Hassles Scale, and the State-Trait Anxiety Inventory, Please refer to Figures 5-8.

* It should be noted that hardiness is inversely related to scores on the Personal Views Survey.

DISCUSSION

One of the primary objectives of this study was to provide further evidence for the link between personality and illness by establishing the relationship between hardiness and physiological parameters which lead to strain on bodily systems, and eventually, to illness or disease. Another objective included formal assessment of one of the main tenets of hardiness theory--that one of the mechanisms by which hardiness serves as a buffer against illness is through the appraisal of fewer numbers of events as stressful by individuals scoring high on this personality trait.

Analyses of hardiness and the demographic variables revealed a significant finding of an association between hardiness and age--individuals who are psychologically hardy are typically older than their less hardy counterparts. One explanation for this may be that older individuals having more life experience and having encountered more stressors in the past, through the successful negotiation and resolution of these situations, are less likely to have either extreme psychological or physiological reactions to similar stressors.

Based on the outcome of this study alone, it does not appear that a relationship exists between hardiness and salivary cortisol/urinary norepinephrine. Although preliminary results are not encouraging in the exploration of the relationship between hardiness and these physiological parameters, several research design issues must be explored before abandoning

this line of inquiry. The discovery of a time effect in the expected direction between pretest cortisol/NE and posttest3 cortisol/NE indicates the primary source of concern is with the baseline cortisol/NE levels, most of which were much higher than anticipated.

Future studies examining the relationship between baseline levels of these substances and hardiness may explore the means of baseline collection of cortisol/NE:

1) One confound contributing to the unexpected results in this study may have been the time chosen to collect baseline samples. The samples were collected one week prior to the mid-term research design exam. At this university, professors commonly "stagger" the giving of mid-terms, oftentimes beginning one week prior to the stated date in the university catalog and extending to the week afterward. Given this, many of the participants in this study could have been experiencing considerable stress regarding other examinations, contributing to an elevated baseline level of cortisol/NE. It may also be helpful in assessing the reliability of the baseline sample to collect self-rating scale data on anxiety at baseline as well as immediately prior to the exam.
2) A more accurate estimate of the baseline levels of cortisol/NE for any given participant may be obtained by the collection of multiple samples and averaging the values obtained.

3) Another consideration would be the collection of blood plasma catecholamines instead of urinary catecholamines. The decision to collect urinary catecholamines in this study was related to some of the disadvantages of collecting blood plasma catecholamines--many individuals find having blood drawn aversive (especially multiple instances of blood drawing) and the collection of blood plasma catecholamines requires a phlebotemist or medical technician.

An interesting finding gained from perusal of the database is some participants in this study, instead of returning to baseline levels of cortisol/NE, returned to lower than baseline levels of these substances. The most likely explanation for this observation is that the observed levels of cortisol/NE were not "true" baseline levels, i.e., they were falsely elevated because the person was experiencing a stress reaction at the baseline sample collection time. For individuals who were not experiencing a stress reaction, an alternative explanation may be found in Hans Selye's theory of General Adaptation Syndrome or GAS (1956,1974). This theory describes the physiological reaction our body experiences in response to stress and consists of 3 stages--alarm, resistance, and exhaustion. In the alarm stage, the body's resources are quickly mobilized as the sympathetic division of the autonomic nervous system springs into action. If the stressor persists, the body shows a defensive reaction--the resistance stage--in an attempt to counteract the stressor. If the stressor continues indefinitely, an individual's bodily resources become depleted and

lower than normal levels of neurochemicals exist, constituting the exhaustion phase of the GAS. This same depletion of bodily resources is believed to occur with discrete stressors if the individual does not deal with stress productively, i.e., the stress response continues long after it is warranted. This is believed to occur with low hardy individuals.

A more pronounced elevation of cortisol/NE may have been elicited with the use of a different stressor, e.g., the MCAT, LSAT, GRE, or a semester exam which had more bearing on an individual's future goals, such as one of only two organic chemistry exams for a student attempting admission to medical school. Public speaking may also elicit greater autonomic/HPA arousal.

The failure to establish a relationship between hardy individuals and scores on the exam may lie in a previously discovered psychological phenomenon--the Yerkes-Dodson Law--which indicates that optimum performance is achieved when a moderate level of arousal is present. Quite possibly, the individuals high on the hardiness dimension were less aroused in taking the test, which subsequently affected their performance. We would expect this, as, hardy individuals, by definition, would view the exam stressor as an event over which they exercised cognitive control and be less likely to interpret the exam as stressful, which presumably would result in less physiological arousal during the exam.

The observation of a strong association between anxiety/arousal as measured by the STAI and anxiety/arousal as measured by a self-rating scale

has widespread implications for clinicians and researchers. There is ongoing debate in the psychological and medical communities as to whether or not clients/patients can provide an accurate, subjective description of their current physical/psychological status to care providers. Results of the present study along with results of previous studies such as Pincus, Wolfe, and Callahan (1994) suggest that our consumers are, indeed, capable of providing accurate information which is needed for routine clinical care. The use of self-rating scales for research applications provides a more parsimonious means of data collection, with the promise of increased success at recruiting research participants and additional possibilities for more data collection at any given time.

The finding of a moderate correlation between hardy individuals and the reporting of fewer daily hassles lends credence to the hardiness construct and its theoretical underpinnings. More specifically, this is related to Kobasa's first hardiness hypothesis--"Among persons under stress, those who have a greater sense of control over what occurs in their lives will remain healthier than those who feel powerless in the face of external forces. They have cognitive control, or the ability to interpret, appraise, and incorporate various sorts of stressful events into an ongoing life plan, and, thereby, deactivate their jarring effects . . ." (Kobasa, 1979). For example, a high hardy individual who views a negative event as a natural occurrence en route to a goal would be less likely to endorse it as a daily hassle on the Combined Hassles and Uplifts Scale. It also suggests

cognitive restructuring as a direction which we, as clinicians, may pursue in the training of psychologically hardy individuals.

Appendix A

Personal Views Survey

Personal View Survey

Below are some items that you may agree or disagree with. Please indicate how you feel about each one by circling a number from 0 to 3 in the space provided. A zero indicates that you feel the item is not at all true; circling a three means that you feel the item is completely true.

As you will see, many of the items are worded very strongly. This is to help you decide the extent to which you agree or disagree. Please read all the items carefully. Be sure to answer all on the basis of the way you feel now. Don't spend too much time on any one item.

0 = Not at all true
1 = A little bit true
2 = Quite a bit true
3 = Completely true

1.	I often wake up eager to take up my life where it left off the day before	0	1	2	3
2.	I like a lot of variety in my work	0	1	2	3
3.	Most of the time, my bosses or superiors will listen to what I have to say	0	1	2	3
4.	Planning ahead can help avoid most future problems	0	1	2	3
5.	I usually feel that I can change what might happen tomorrow, by what I do today	0	1	2	3
6.	I feel uncomfortable if I have to make any changes in my everyday schedule	0	1	2	3
7.	No matter how hard I try, my efforts will accomplish nothing	0	1	2	3
8.	I find it difficult to imagine getting excited about working	0	1	2	3
9.	No matter what you do, the "tried and true" are always the best.....	0	1	2	3

0 = Not at all true
1 = A little bit true
2 = Quite a bit true
3 = Completely true

10.	I feel that it's almost impossible to change my spouse's mind about something.....	0	1	2	3
11.	Most people who work for a living are just manipulated by their bosses.....	0	1	2	3
12.	New laws shouldn't be made if they hurt a person's income.....	0	1	2	3
13.	When you marry and have children you have lost your freedom of choice	0	1	2	3
14.	No matter how hard you work, you never really seem to reach your goals	0	1	2	3
15.	A person whose mind seldom changes can usually be depended on to have reliable judgement	0	1	2	3
16.	I believe most of what happens in life is just meant to happen.....	0	1	2	3
17.	It doesn't matter if you work hard at your job, since only the bosses profit by it anyway .	0	1	2	3
18.	I don't like conversations when others are confused about what they mean to say	0	1	2	3
19.	Most of the time it just doesn't pay to try hard, since things never turn out right anyway.....	0	1	2	3
20.	The most exciting thing for me is my own fantasies.....	0	1	2	3
21.	I won't answer a person's questions until I am very clear as to what he is asking	0	1	2	3
22.	When I make plans I'm certain I can make them work	0	1	2	3

0 = Not at all true
1 = A little bit true
2 = Quite a bit true
3 = Completely true

23.	I really look forward to my work	0	1	2	3
24.	It doesn't bother me to step aside for a while from something I'm involved in, if I'm asked to do something else.....	0	1	2	3
25.	When I am at work performing a difficult task I know when I need to ask for help	0	1	2	3
26.	It's exciting for me to learn something about myself.....	0	1	2	3
27.	I enjoy being with people who are predictable	0	1	2	3
28.	I find it's usually very hard to change a friend's mind about something	0	1	2	3
29.	Thinking of yourself as a free person just makes you feel frustrated and unhappy	0	1	2	3
30.	It bothers me when something unexpected interrupts my daily routine.....	0	1	2	3
31.	When I make a mistake, there's very little I can do to make things right again	0	1	2	3
32.	I feel no need to try my best at work, since it makes no difference anyway	0	1	2	3
33.	I respect rules because they guide me	0	1	2	3
34.	One of the best ways to handle most problems is just not to think about them.....	0	1	2	3
35.	I believe that most athletes are just born good at sports.....	0	1	2	3

0 = Not at all true
1 = A little bit true
2 = Quite a bit true
3 = Completely true

36.	I don't like things to be uncertain or unpredictable	0	1	2	3
37.	People who do their best should get full financial support from society	0	1	2	3
38.	Most of my life gets wasted doing things that don't mean anything.....	0	1	2	3
39.	Lots of times I don't really know my own mind	0	1	2	3
40.	I have no use for theories that are not closely tied to facts.....	0	1	2	3
41.	Ordinary work is just too boring to be worth doing	0	1	2	3
42.	When other people get angry at me, its usually for no good reason	0	1	2	3
43.	Changes in routine bother me	0	1	2	3
44.	I find it hard to believe people who tell me that the work they do is of value to society	0	1	2	3
45.	I feel that if someone tries to hurt me, there's usually not much I can do to try and stop him	0	1	2	3
46.	Most days, life just isn't very exciting for me...	0	1	2	3
47.	I think people believe in individuality only to impress others.....	0	1	2	3
48.	When I'm reprimanded at work, it usually seems to be unjustified	0	1	2	3
49.	I want to be sure someone will take care of me when I get old.....	0	1	2	3
50.	Politicians run our lives	0	1	2	3

Appendix B

Catecholamine Processing Protocol

BASIC PROTOCOL FOR PROCESSING CATECHOLAMINES

Operation of pH Meter

1. Calibrate against the yellow buffer solution--pH = 7.0.
2. Everything measured in relation to catecholamines will be in the range of 7.0 ± 0.1.
3. Press “mode” to get the pH to 7.0.
4. Calibrate pink buffer as the other anchor at 4.0 ± 0.1.
5. Wash pH electrode between calibrations and measurements and remove excessive moisture.

Synthesis of Buffer:

0.1 M = $\frac{1.321\text{g}}{100\text{ mL Ammonium Hydrogen Phosphate}}$ + 0.1% EDTA

pH to 7. The buffer can be used for up to one week.

Creation of Column:

1. Measure 2 g bioresin and pour into small flask.
2. Cover with buffer.
3. Mix well.
4. pH to 7.
5. Pour resin into column.
6. Let solution drip through column until it is ½ inch from the top.
7. Connect tube 1 to the column.
8. Attach flow adaptor.

Synthesis of Boric Acid:

1. Measure 4 g boric acid into large flask for every 100 ml H_2O.
2. Stir on electric stirrer with heat.
3. Boric acid must be made each day.

To Process Catecholamines:

1. Turn pump on. Place tube from pump into ammonium phosphate buffer.
2. Connect column tube (#2) to the second pathway right of syringe.
3. Get norepinephrine (NE) High Standard or a catecholamine sample.
4. Set the top timer on 4 minutes. (The bottom timer is set for 6 minutes.)
5. Put tubes in buffer solution.
6. The 4 minute timer is running with the buffer and sample or standard. Turn the knob until the icon is in the 4 o'clock position.
7. Turn the pump on.
8. Inject the sample (5 mL).
9. Stop when the timer gets to zero and turn the pump off. Turn the knob back to its original position.
10. Take the buffer out. Pinch the tube to avoid air bubbles.
11. Dip the tube in double de-ionized H_2O and pinch again to avoid air bubbles.

    Note: Catecholamines are lighter than proteins. H_2O washes off everything that does not stick to the column.
12. Start the pump (button with the man icon) and start the 6 minute timer.

13. Turn the pump off when the timer is at 0.
14. Put tube in boric acid solution.
15. Turn the pump on.
16. Press the syringe button to activate the program. When the program runs, the solution will go into one tube.
17. The boric acid solution competes with the charges on the column. The program elutes the catecholamines into different test tubes.
18. Place the pump tube back into the buffer solution.
19. When you re-use the column again, make sure the pH = 7.0. Make sure the buffer pH which comes off the column equals the buffer pH which went into the column.
20. As all these solutions are light-sensitive, they should be left in the darkened refrigerator until they are ready to be processed.

Use of Spectrophotometer:

1. Turn on the UV lamp.
2. Allow to warm up for 30 min.
3. Go to lambda.
4. Select 210 nanometers.
5. Get 1 mL H_2O in pipette and transfer to cuvette.
6. Put into spectrophotometer.
7. Press calibration (or auto 0). Steps 3-4 are for calibration.
8. Mix sample with vortex or by hand by gently tapping test tube several times.

9. Get 1 mL of sample.
10. Wipe off sides of cuvette with kimwipe.
11. Put sample in spectrophotometer.
12. Wash out cuvette each time.
13. Only touch the frosted part of the cuvette.
14. Calibrate with H_2O periodically (if the values are suspect).
15. Press UV button to “off” position.

Appendix C

Tables

Table 1

Characteristics of Participants

Nominal Characteristics	%
Gender	
Female	69
Male	31
Ethnicity	
Caucasian	93
Hispanic	7
Annual Income	
$0-$15,000	57
$15,000-$30,000	27
$30,000-$45,000	12
$45,000-$60,000	2
Over $60,000	2

Table 2

Means, Standard Deviations and Ranges for Participant Characteristics

Internal Characteristics	Range	Mean	SD
Age	18-37	23.61	4.17
Education (in years)	14-20	15.91	1.11

Table 3

Means, Standard Deviations and Ranges for Urinary Norepinephrine

Sample	Range	Mean	Standard Deviation
BLNE	0.08-1.84	0.6522	0.4171
PRENE	0.13-1.73	0.5765	0.3808
POSTNE1	0.09-1.88	0.5160	0.4115
POSTNE2	0.06-1.83	0.5750	0.4925
POSTNE3	0.00-1.09	0.4171	0.3198

Table 4

Means, Standard Deviations and Ranges for Salivary Cortisol

Sample	Range	Mean	Standard Deviation
BLCORT	3.19-26.98	8.07	4.77
PRECORT	3.63-34.17	9.84	5.41
POSTCORT1	2.58-19.61	7.58	4.06
POSTCORT2	1.91-14.73	5.71	2.90
POSTCORT3	1.95-20.51	5.78	3.48
POSTCORT4	2.13-14.16	5.53	2.72

Appendix D

Figures

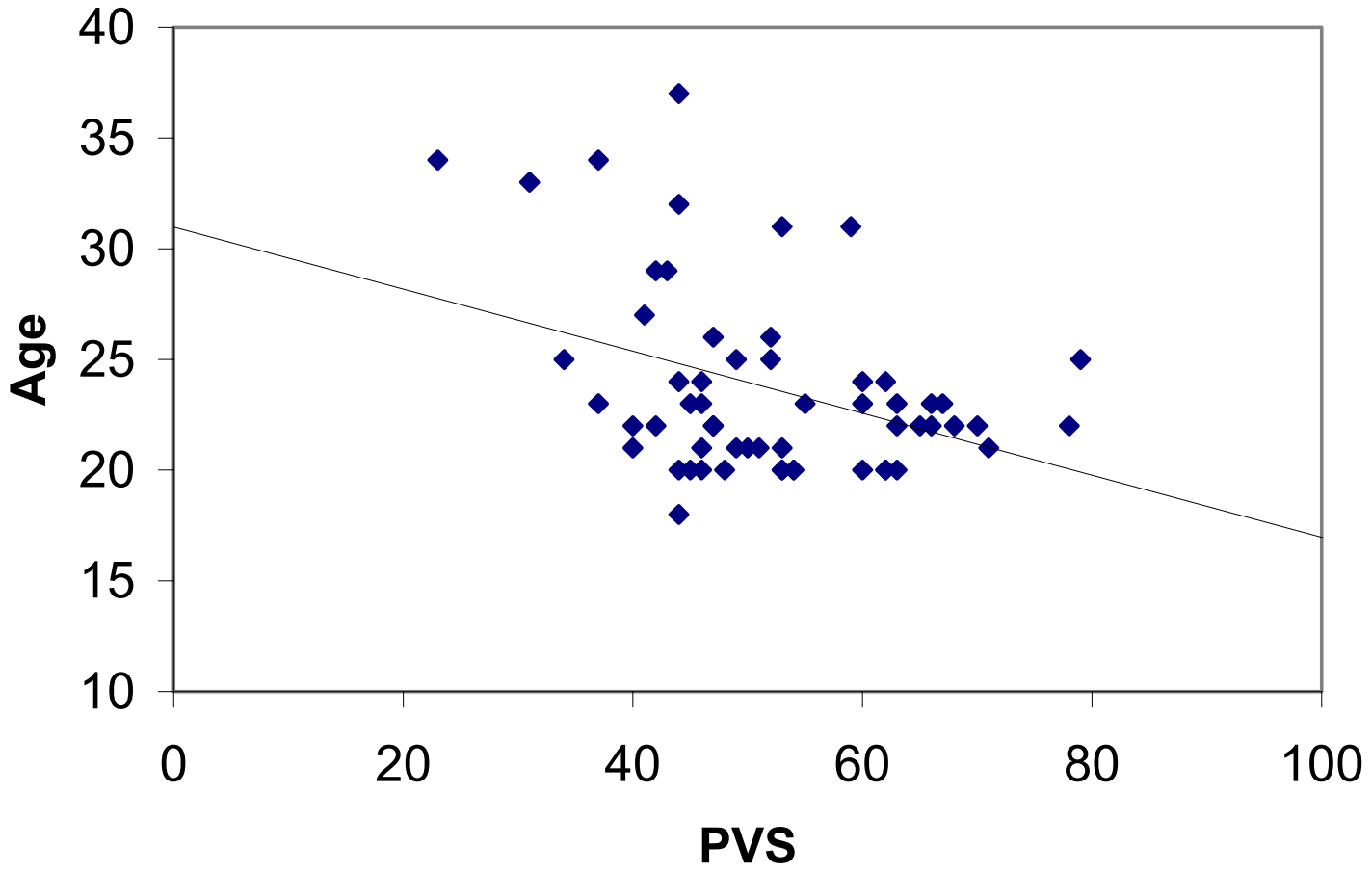

Figure 1

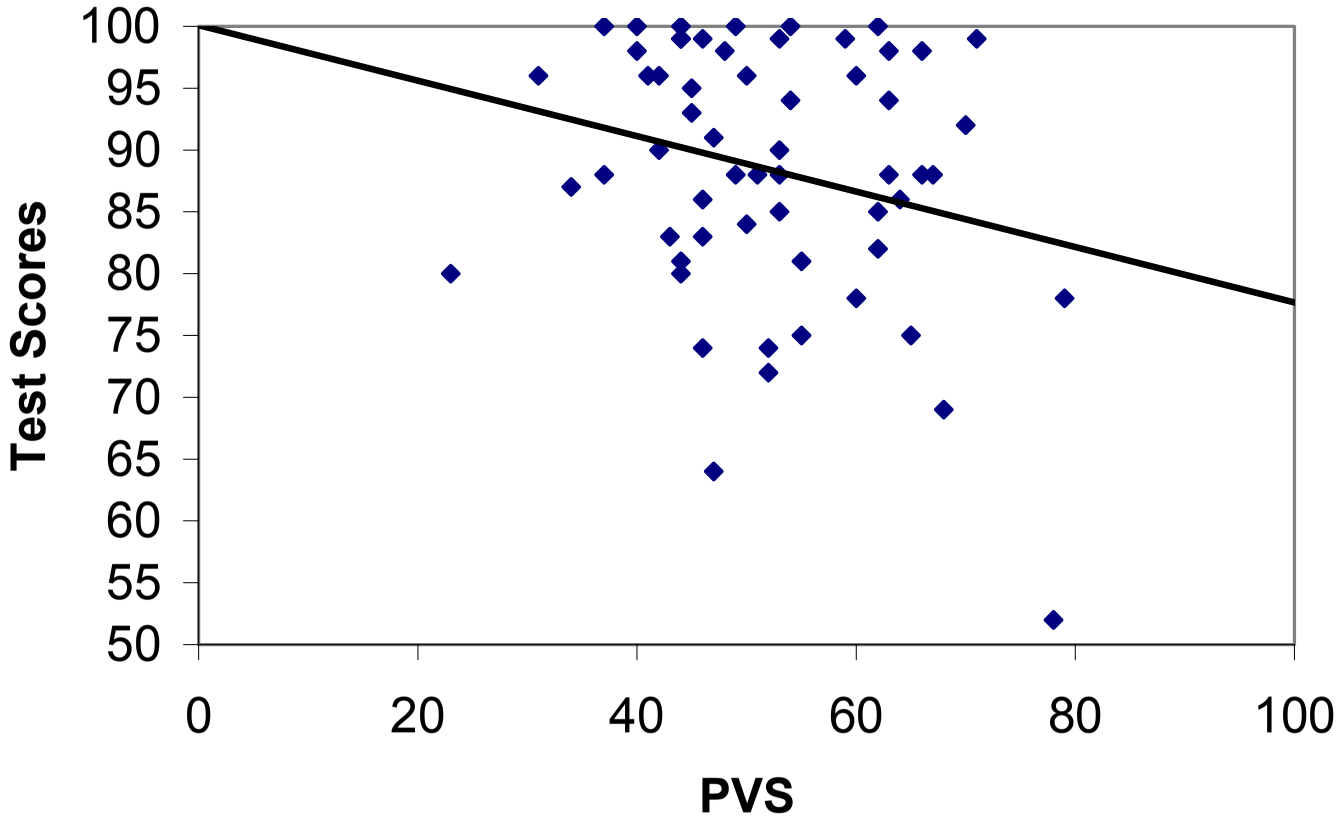

Figure 2

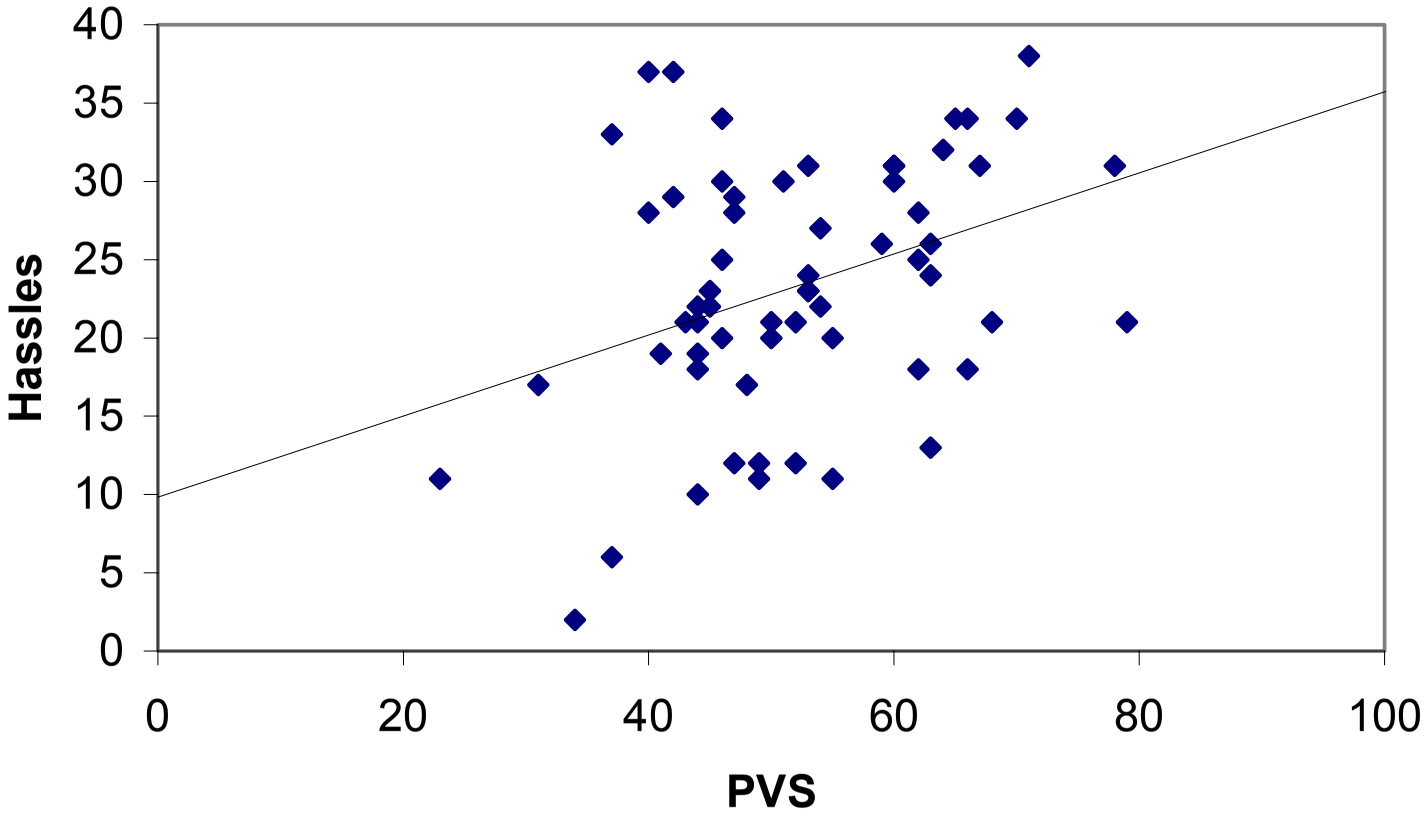

Figure 3

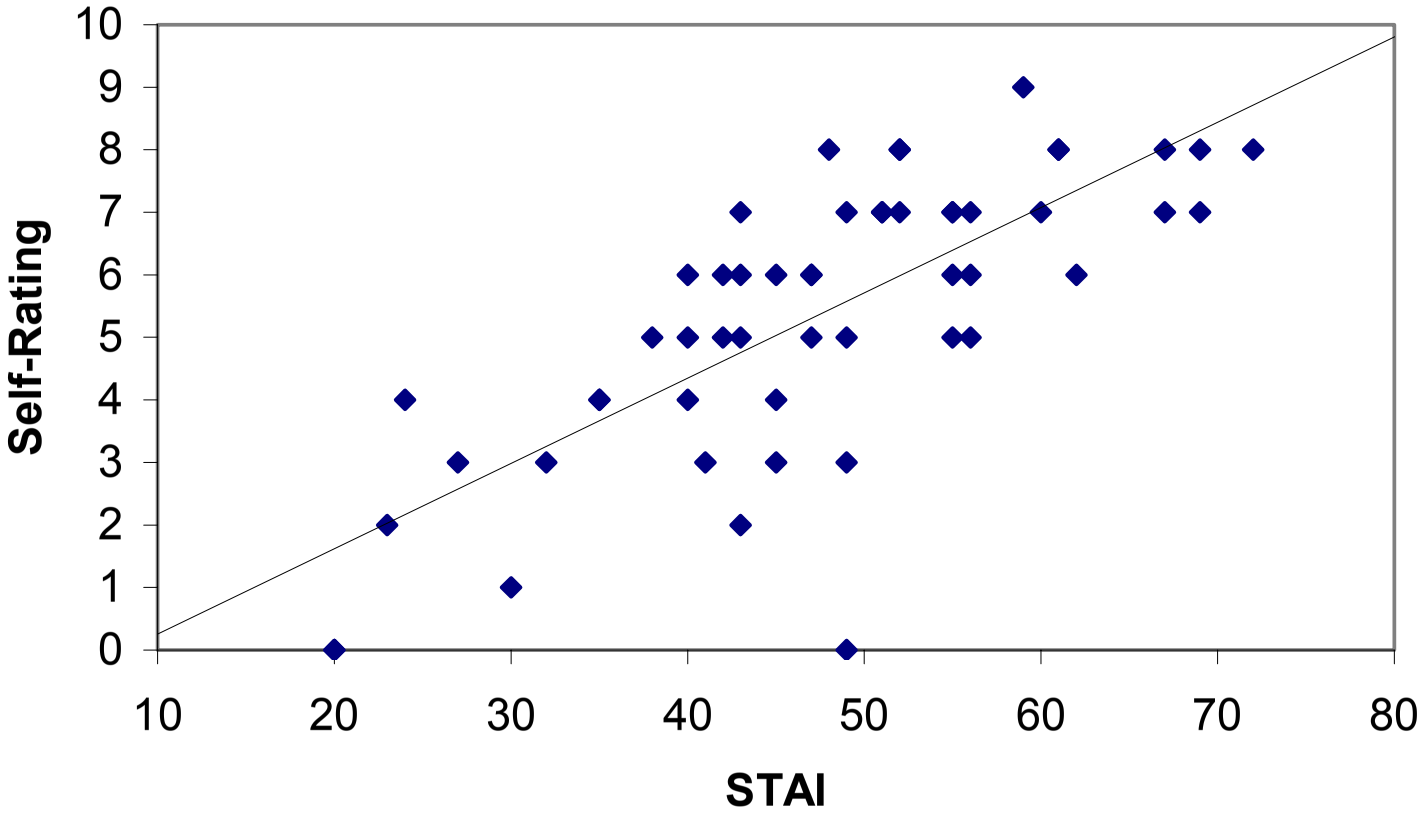

Figure 4

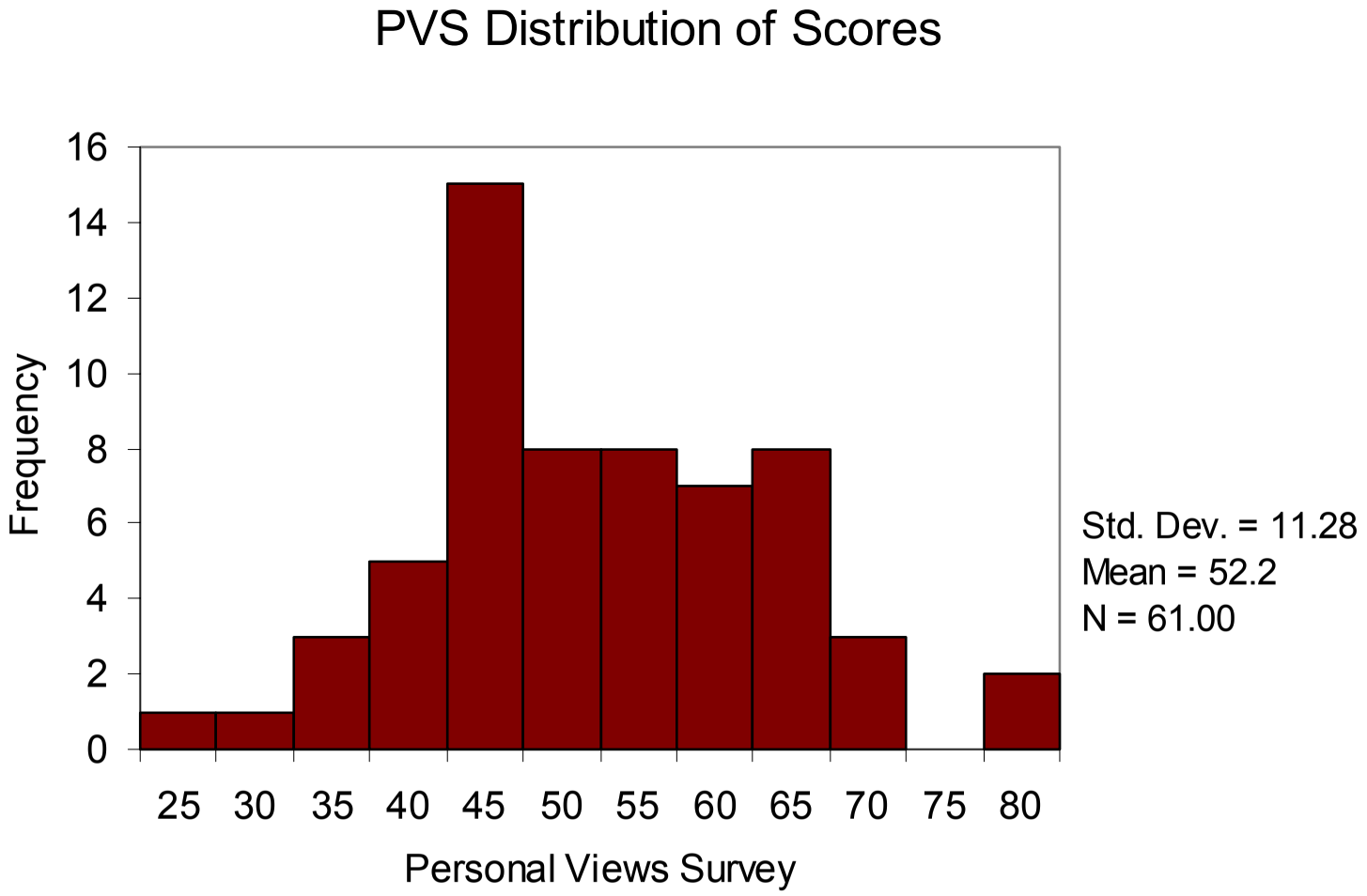
PVS Distribution of Scores
Frequency
16
14
12
10
8
6
4
2
0
25
30
35
40
45
50
55
60
65
70
75
80
Personal Views Survey
Std. Dev. = 11.28
Mean = 52.2
N = 61.00

Figure 5

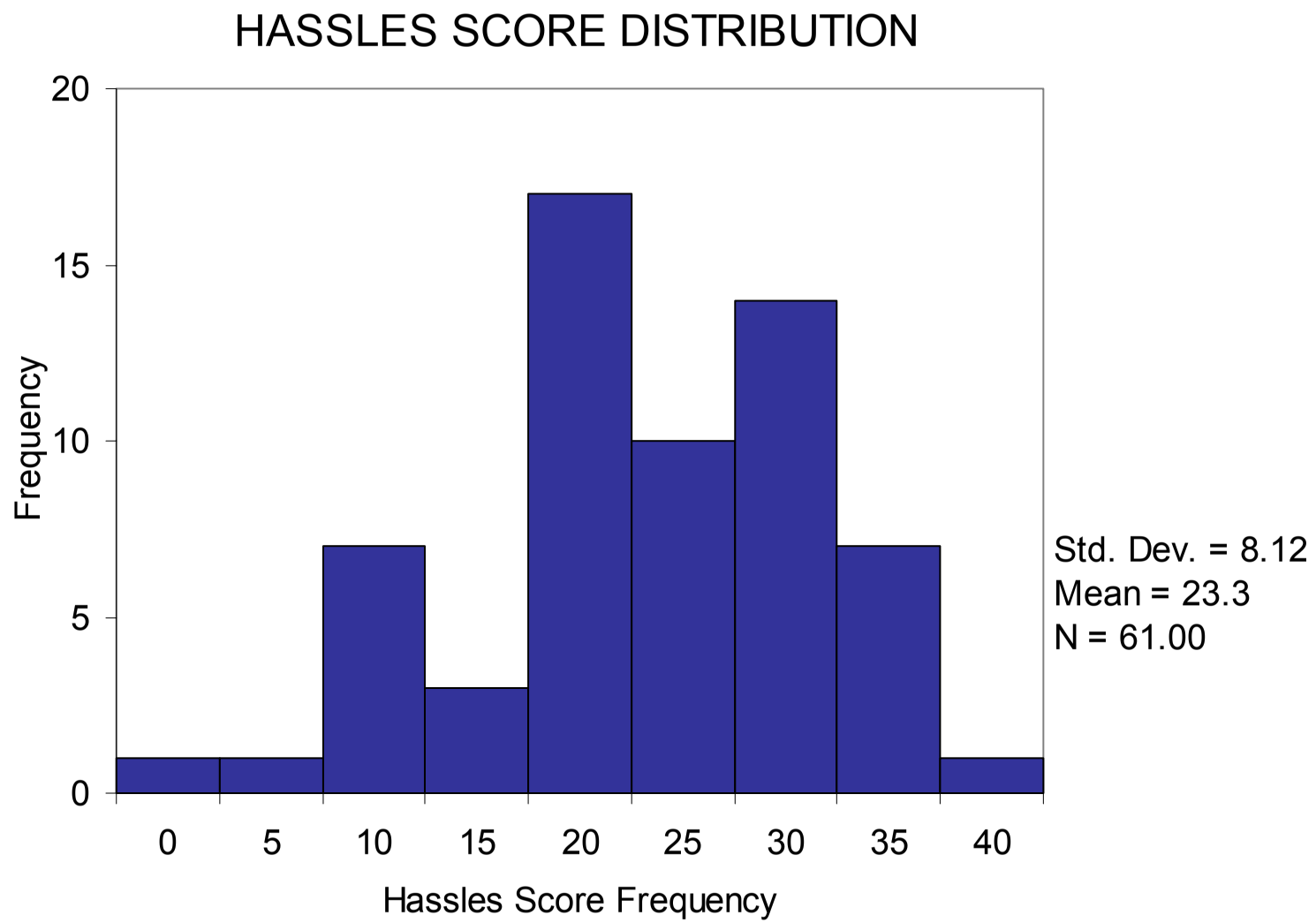
HASSLES SCORE DISTRIBUTION
Frequency
20
15
10
5
0
0
5
10
15
20
25
30
35
40
Hassles Score Frequency
Std. Dev. = 8.12
Mean = 23.3
N = 61.00

Figure 6

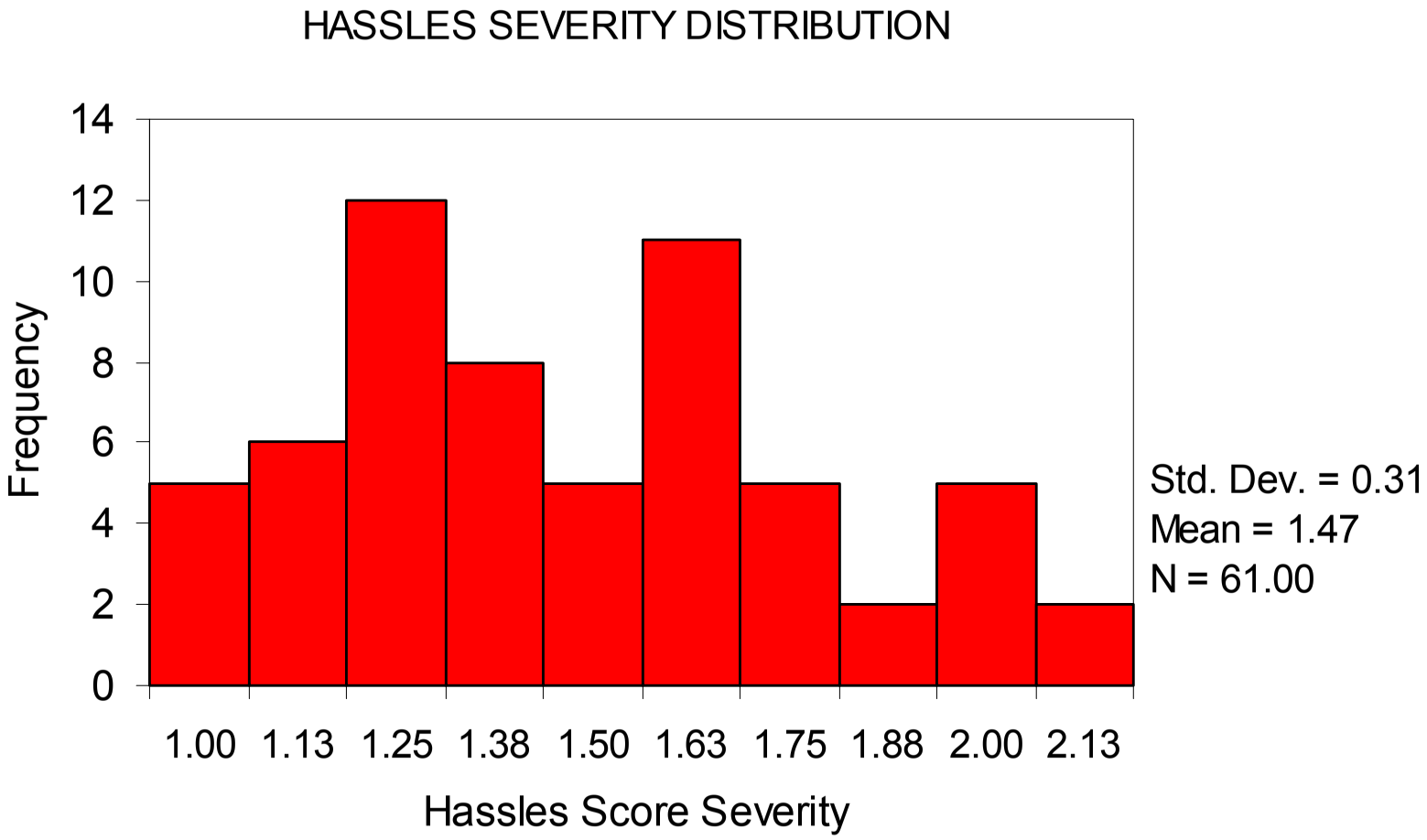
HASSLES SEVERITY DISTRIBUTION
Frequency
14
12
10
8
6
4
2
0
1.00
1.13
1.25
1.38
1.50
1.63
1.75
1.88
2.00
2.13
Hassles Score Severity
Std. Dev. = 0.31
Mean = 1.47
N = 61.00

Figure 7

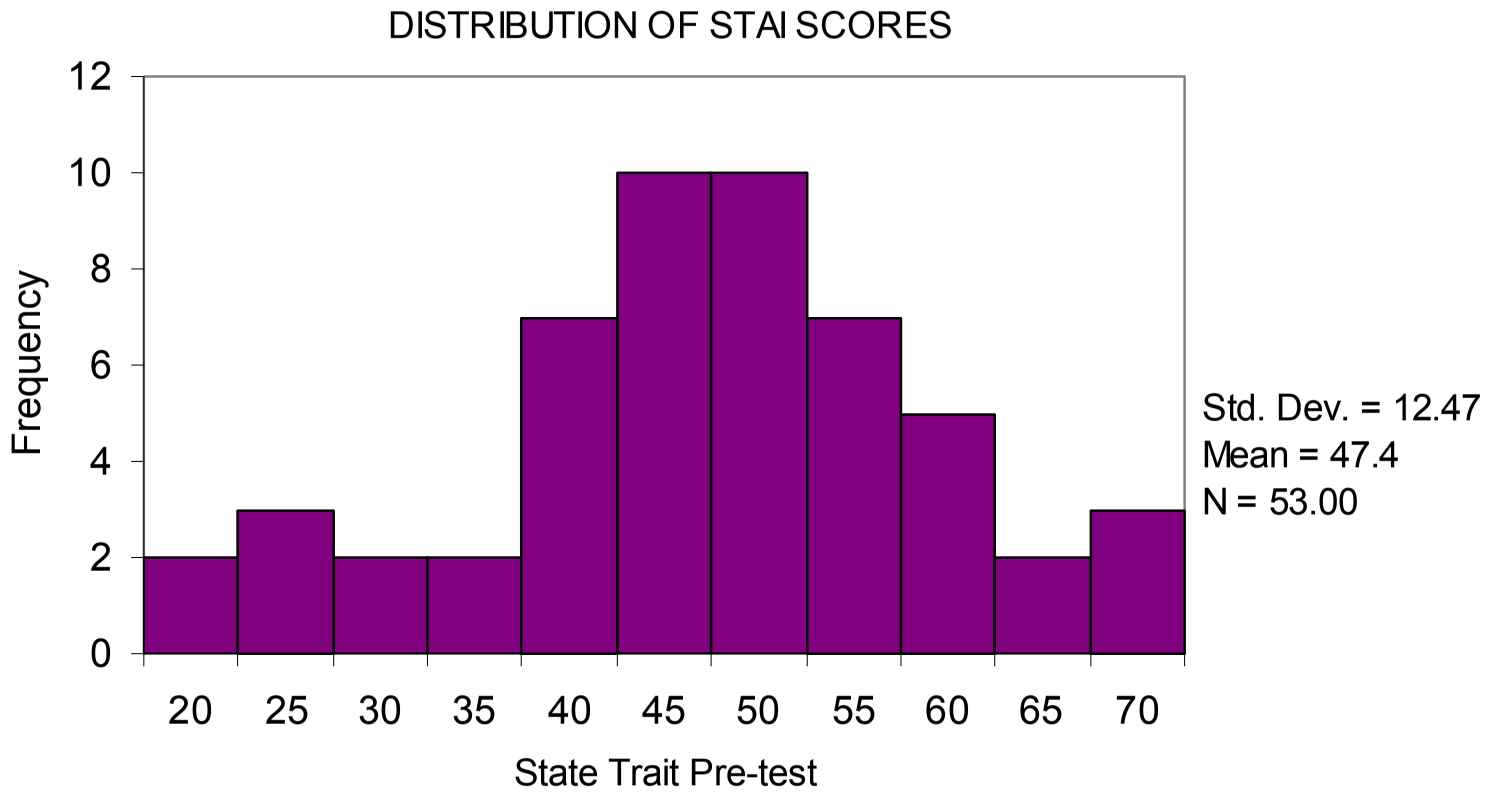
DISTRIBUTION OF STAI SCORES
Frequency
12
10
8
6
4
2
0
20
25
30
35
40
45
50
55
60
65
70
State Trait Pre-test
Std. Dev. = 12.47
Mean = 47.4
N = 53.00

Figure 8

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