REPORT DOCUMENTATION PAGE	Form Approved OMB No. 0704-0188

1. AGENCY USE ONLY *(Leave blank)*	2. REPORT DATE	3. REPORT TYPE AND DATES COVERED
	1 AUG 97	

4. TITLE AND SUBTITLE
THE EFFECTS OF COGNITIVE HARDINESS ON STRESS, HEALTH, PERFORMANCE, AND CARDIOVASCULAR/NEUROENDOCRINE FUNCTION

5. FUNDING NUMBERS

6. AUTHOR(S)
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7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)
KANSAS STATE UNIVERSITY

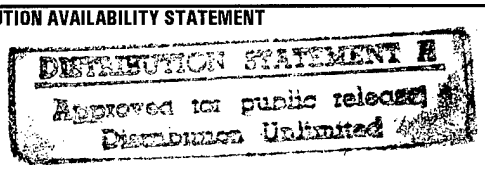
8. PERFORMING ORGANIZATION REPORT NUMBER
97-097

9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)
DEPARTMENT OF THE AIR FORCE
AFIT/CI
2950 P STREET
WRIGHT-PATTERSON AFB OH 45433

10. SPONSORING/MONITORING AGENCY REPORT NUMBER

11. SUPPLEMENTARY NOTES

12a. DISTRIBUTION AVAILABILITY STATEMENT



12b. DISTRIBUTION CODE

13. ABSTRACT *(Maximum 200 words)*

14. SUBJECT TERMS	15. NUMBER OF PAGES
	339
	16. PRICE CODE

17. SECURITY CLASSIFICATION OF REPORT	18. SECURITY CLASSIFICATION OF THIS PAGE	19. SECURITY CLASSIFICATION OF ABSTRACT	20. LIMITATION OF ABSTRACT

THE EFFECTS OF COGNITIVE HARDINESS ON STRESS, HEALTH, PERFORMANCE, AND CARDIOVASCULAR/ NEUROENDOCRINE FUNCTION

by

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B. S., United States Air Force Academy, 1988

A THESIS

submitted in partial fulfillment of the

requirements for the degree of

MASTER OF SCIENCE

Department of Psychology
College of Arts and Sciences

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1997

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Abstract

Cognitive hardiness is a psychological construct of stress resiliency which has been postulated to moderate stress-illness and stress-performance relationships. Hardiness has also been thought to exert main effects on health and performance outcomes. In Study 1, relationships between hardiness, perceived stress, depression, and academic performance were investigated. Hardiness was found to be positively predictive of academic performance; the effect was partially mediated by course load. Hardiness was also revealed to moderate the stress-depression relationship. The negative relationship between stress and academic performance was mediated by depression. A model explaining 30% of the variance in academic performance is presented and discussed. Study 2 was an extensive exploratory effort that investigated the relationships between hardiness, stress, performance, illness/injury, appraisal processes, and physiological reactivity to a realistic stressor in 23 helicopter pilots. Main and moderating effects for hardiness were demonstrated in stress-performance and stress-illness relationships and outcomes. Hardiness was predictive of challenge appraisals, cortisol baselines and reactivity, and performance. Mediated relationships are discussed. Relations between cortisol reactivity and performance suggest profound and disturbing adverse impact on work-related cognitive function. Higher order curvilinear relationships between hardiness, cortisol reactivity, challenge appraisals, and performance were revealed. Implications, future research initiatives, and appropriate research designs are discussed.

Table of Contents

List of Figures

List of Tables

Acknowledgements

I would like to thank my thesis committee, Dr. Clive Fullagar, Dr. Pat Knight, and Dr. Stephen Kiefer, for their time and support in creating this thesis; in particular, I am immensely grateful for the intellectual freedom, opportunities, and encouragement granted by Dr. Fullagar, my advisor. Thanks also to Sharon Sterling for her assistance, smiles, and all the coffee one can drink in two years. I would also like to thank Rick Best for his support and friendship over the last two years and Jeff Bartel for his management of the "sporting life." Thanks also to Phil Elmore, Doug Stickle, the staff of the Immunochemistry Section at Brooke Army Medical Center, Dan Jimenez, and the lab staff at Irwin Army Community Hospital for their support in completing this effort. This thesis would also not have been possible without the aid and aviation expertise of Ron Olsen, Rich Nowland, and Bruce Thomas (rotary-wing beats fixed-wing anyday). My appreciation is also extended to all those individuals who provided expert advice and encouragement, to include Dr. Kaleem Quadry and Dr. Ken Nowack. Most importantly, I would like to express my heartfelt gratitude and love to my wife, Deanna, my daughter, Shaye, and the rest of my family for their undying support. Finally, I would like to thank the two individuals below for their insight and the inspirational wisdom in their words; many a time their words permitted me to see things as they are.

> "O Shariputra, form does not differ from emptiness; emptiness does not differ from form. That which is form is emptiness; that which is emptiness form. The same is true of feelings, perceptions, impulses, consciousness."
>
> -Avalokiteshvara Bodhisattva, from the Heart Sutra

> "...And what you thought you came for
> Is only a shell, a husk of meaning
> From which the purpose breaks only when it is fulfilled
> If at all. Either you had no purpose
> Or the purpose is beyond the end you figured
> And is altered in fulfillment."
>
> - T. S. Eliot, from "Little Gidding"

The Effects of Cognitive Hardiness on Stress, Health, Performance, and Cardiovascular/Neuroendocrine Function

Introduction

Over the past several decades, the term "stress" has become something of a "household word," a phenomenon popularly thought to be pervasive in industrialized society (Cacioppo, 1997). Additionally, stress is widely believed, by both researchers and the general populace, to be generally unhealthy and detrimental to optimal performance in one's personal and professional life (Matteson & Ivancevich, 1987). This, of course, is not to deny the postulation by some that there is both positive/beneficial (eustress or non-pathological stimulation) and negative/adversely impacting (distress) stress (Selye, 1956; Pollock, 1986). Certainly, such a characterization of stress is integral to the inverted-U stress-performance relationship first proffered by Yerkes and Dodson in 1908; this conventional perspective and several others will be later discussed. The body of stress-related literature, advice, and self-help guidance provided to the general populace has exploded in recent years; popular concern over stress has led to provision of such information by entities ranging from consulting organizations (Great Performance Incorporated, 1992) to counseling centers (University of Illinois Counseling Center, personal communication, 1996; State University of New York at Buffalo, 1996). There is a growing body of evidence to warrant such widespread concern.

The adverse impact of stress can be examined in a number of ways and in a number of domains. Stress appears to degrade health via a number of psychoneuroendocrinim-

munological and behavioral pathways—these will be later discussed in depth. Stress also appears to have severe economic and organizational impacts, leading to solemn concern in the corporate world. In general, stress "is implicated in industrial accidents, absenteeism, turnover, increased health care costs, and decrements in the quantity and quality of production" (Matteson & Ivancevich, 1987, p. 5; Pollock, 1986). Such concerns and myriad ways to counter the adverse impacts of stress have been echoed as well in more recent compendiums of applied research (Murphy, Hurrell, Sauter, & Keita (Eds.), 1995; Sauter & Murphy (Eds.), 1995). Matteson and Ivancevich, in their 1987 review of extant literature, note that the economic impact of stress in the United States has been estimated to be anywhere from 17 to over 100 billion dollars annually ($27.5 to over $137 billion when adjusted for inflation dating from the respective years of the estimates). Jones (1984) estimates that: 75 to 85 percent of all industrial accidents may be caused by a less than optimal ability to cope with stress; stress-associated heart disease results in the loss of 135 million work days annually; stress may be implicated in as much as 60 percent of all long-term disability cases; and stress-related headaches are perhaps the prime factor in lost work hours in American industry. More recently, Cartwright and Cooper (1997) report that the "collective cost of stress to U.S. organizations for absenteeism, reduced productivity, compensation claims, health insurance, and direct medical expenses has been estimated at...$150 billion per year" (p. 2); Cartwright and Cooper conclude a similar situation exists in the U.K. It is now believed that the positive correlation between age and blood pressure in industrial societies is the result of the increasing complexity and rate of change (and the accompanying stress) in those societies (Cacioppo, 1997). Rosen (1995)

and Sauter and Murphy (1995) share these concerns over workplace change, complexity, and technological advance. Diminished worker control over his or her environment, the growth of service sector jobs, the prospect of workplace violence (the third leading cause of death from injury among workers), computerization, use of increasingly advanced information processing systems, advanced manufacturing technologies, increased psychological and cognitive demands driven by technological and information processing advances, and electronic performance monitoring have all been postulated to contribute to worker stress (Sauter & Murphy, 1995). Disturbingly, Rosen (1995) implies the situation is worsening; that is, the workplace is becoming more stressful. Concerns over the potential detriment of stress have led some governments to become involved in workplace structuring; the most notable example is the Swedish Work Environment Act passed in 1987 (Sauter & Murphy, 1995). This legislation mandates structuring of the work environment in ways that reduce stress and increase worker autonomy and control. It is important to note here that stress is not something strictly confined to the workplace. A host of researchers have argued that stress should be considered as both a subjective phenomenon and a construct driven by the totality of one's life experiences, interpretations, and appraisals (Turner, Wheaton, & Lloyd, 1995; Lazarus & Folkman, 1984; Cohen, Kamarck, & Mermelstein, 1983). Research by Turner et al (1995) suggests that an encompassing construct of social stress alone accounts for "between 23 and 50 percent of observed differences in mental health by sex, marital status, and occupation" (p. 104). In sum, there appears to be a substantial foundation for concerns over stress and its adverse impact in the workplace, on individual psychological and physiological well-being,

and on an American health care industry that, in 1990, consumed 12 percent of the gross national product (Cacioppo, 1997).

For several pages, I have discussed the "epidemic" of stress, but what, exactly, is stress? This is a very difficult question in that a review of the literature reveals there is little agreement over the definition or operationalization of the construct. This is not to say that the varied definitions are not complementary—they most certainly provide an informative (though eclectic) conceptualization of stress. First, consider popular definitions of stress. Matteson & Ivancevich (1987) relate that when asked to complete the sentence "Stress is _____," common responses in the general population include:

> "Having too much to do and too little time to do it in.
> Fighting the traffic to and from work.
> Not being sure what is expected on the job or at home.
> Not getting promoted (or getting promoted).
> Never seeming to be able to catch up financially.
> Being responsible for other people.
> Wondering if career goals are realistic.
> Trying to balance job demands with family responsibilities.
> Not being kept informed about what one needs to know to do the job.
> Worrying about becoming obsolete" (p. 9).

As a law enforcement officer for seven years and in conducting a stress management workshop for a police department over the last 10 months, I've heard many officers proclaim that the aspect of their jobs which is both most rewarding and most stressful is "dealing with people." Perhaps the most popular lay definition of stress, and one that has been branded onto products ranging from bumper stickers to coffee cups, is "the uncontrollable urge to choke the living hell out of someone."

Before moving on to discuss the conceptualizations which have been offered by researchers in the field, however, it is valuable to note commonalities in the above comments and responses. Qualitatively, subjective variation in comments/responses between and within individuals is quite evident, and while all the above comments refer to an external event or condition, the subjective (and differential) interpretation or appraisal (sometimes of identical or nearly identical events) is inescapable. This is an important point that will be raised numerous times in the pages to come. Matteson and Ivancevich (1987) have, understandably, declared that the "...word stress means so many different things to so many different people that it has been described as the most imprecise in the scientific dictionary" (p. 9). Unfortunately, researchers in the field don't fare much better in agreeing upon working definitions than the general public.

Perhaps the most eloquently simple definition of stress is that provided by Sapolsky (1992a). He asserts that a stressor is "anything that disrupts physiological balance," (p. 288), be it physical or psychological, real or perceived (a perspective also endorsed by Kusnecov & Rabin (1994)). Sapolsky goes on to say the stress response is "the body's adaptations designed to reestablish the balance" (p. 288). Sapolsky unifies the two in saying that the term "stress" refers to the condition of stressors provoking a stress response. Matteson and Ivancevich (1987) view stress similarly, defining the construct as "an adaptive response, *moderated by individual differences* [emphasis added], that is a consequence of any action, situation, or event that places special demands upon a person" (p. 10). By "special," Matteson and Ivancevich mean those demands that are "unusual, out of the ordinary, physically or psychologically threatening, or outside an

individual's usual set of behaviors" (p. 10). Lazarus and his colleagues (1985) declare "...stress lies not in the environmental input but in the person's appraisal of the *relationship* between that input and its demands and the person's agendas (e. g. beliefs, commitments, goals) and capabilities to meet, mitigate, or alter these demands in the interests of well-being" (p.770). The preceding comment is inclusive of Lazarus and Folkman's (1984) conceptualization of an appraisal process that they assert often mediates the relationship between the person-environment interaction and the subjective experience of stress. One can see that in both popular and scientific conceptualizations of stress, the environmental aspect of the stress experience alone appears insufficient to explain the stress response and experience. This is the case, presumably, because of those perceptions, appraisals, and individual differences mentioned in the above definitions.

Accordingly, Epstein and Katz (1992) found that the variance in total stress is almost completely explained by behaviorally or psychologically self-produced (versus externally produced) stress. External (environmental) stress was uncorrelated with any of the scales of the Constructive Thinking Inventory, psychological hardiness, and eight of eleven psychophysiological symptoms (of the three psychophysiological symptoms in which there was a significant correlation, explained variance never exceeded four percent). Epstein and Katz conclude that "...people create much of their own stress, not only by how they construe events and cope with them after they have construed them as stressful...but also because of the part they play in instigating the stressors they experience" (p. 824). Similarly, Ellis and Grieger (1977), in their seminal work on rational-emotive therapy (RET), declare:

> "So says the central theory of RET (as Epictetus observed some 2000 years ago): The things that occur do not upset you—but your view of those things does. Or, in RET terms, A (Activating Event) does not directly cause C (emotional or behavioral Consequence); B (your Beliefs about A) does" (p. 7-8).

Ellison and Genz (1983) note it has been estimated that as much as 80 percent of all visits to physicians may be stress-related or psychosomatic (though empirical support for such an estimate is questionable). That considered, it is surprising that life event measures of stress consistently share a correlation with illness and symptoms measures of only about .30 (Swindle, Heller, & Lakey, 1988; Maddi & Kobasa, 1984). However, when one considers the subjective element that appears to be prominent in the experience of stress, the surprise wanes. Many of the researchers mentioned in the preceding paragraphs comprise part of a growing community of scholars that are increasingly focusing on the person factors (individual differences) that moderate and mediate the relationships between stressors, subjective stress, and outcomes of interest. Since 1979, cognitive hardiness has emerged as one of the most promising individual differences contributing to resiliency against the adverse effects of psychologically induced stress.

Hardiness is a multifaceted personality construct developed by Kobasa (now Ouellette) (1979a, 1979b) and deeply rooted in existential philosophy and its psychological outgrowth, existential personality theory. Kobasa postulated that hardiness functions as a stress resilience resource, moderating the relationship between stress and illness (Kobasa, Maddi, Puccetti, & Zola, 1985; Maddi & Kobasa, 1984; Kobasa & Puccetti, 1983; Kobasa, Maddi, & Zola, 1983; Kobasa, 1982; Kobasa, Maddi, & Kahn, 1982; Kobasa, 1979a, 1979b). In the last eighteen years, a great amount of research has

been conducted investigating the proposed moderating (as well as main) effects of hardiness on not only the stress-illness relationship, but also on relationships between stress and depression/negative affect (Hodgkinson & Shepherd, 1994; Hull, Lehn, & Tedlie, 1991; Maddi, 1987; Manning, Williams, & Wolfe, 1988; Rhodewalt & Zone, 1989), mood disturbance (Goss, 1994), performance (Herlich, 1985; Westman, 1990), absenteeism (Neubauer, 1992; Tang & Hammontree, 1992), coping/adaptation (Florian, Mikulincer, & Taubman, 1995; Nowack, 1989; Pollock, Christian, & Sands, 1990; Solcova & Tomanek, 1994), post traumatic stress disorder (PTSD) diagnosis (Sutker, Davis, Uddo, & Ditta, 1995), job satisfaction (Neubauer, 1992), burnout (Duquette, Kerouac, Sandhu, Ducharme, & Saulnier, 1995; Topf, 1989), physiological arousal (Allred & Smith, 1989; Contrada, 1989; Maddi, 1987; Solcova & Sykora, 1995; Wiebe, 1991), neuroendocrinological/immunological function (Okun, Zautra, & Robinson, 1988; Zorilla, DeRubeis, & Redei, 1995), and withdrawal intentions (Rush, Schoel, & Barnard, 1995). The purpose of the present project was to investigate the moderating and main effects of hardiness on both stress-illness and stress-job performance relationships, and in so doing, address many of the concerns (the unity, structure, and appropriate instrument in measuring the hardiness construct, its ability to function as a buffer against stress, various methodological flaws, inappropriate and inadequate data analysis, and psychoneuro-endocrinological and cardiovascular pathways by which the effects of hardiness are mediated) which have arisen in hardiness research. To the extent hardiness can moderate the adverse effects of stress, and/or directly impact health and performance, given that

stress truly degrades performance and health, hardiness has the potential to positively contribute to important organizational outcomes.

The Hardiness Construct and Existential Underpinnings

As mentioned above, hardiness has its basis in existential philosophy and, more particularly, in the existential personality theory outlined by Kobasa and Maddi (1977; Kobasa, 1979a; Orr & Westman, 1990). Perhaps the most fundamental principle in existential philosophy is that in a meaningless and chaotic world, existence is prior to essence (Grene, 1970). That is, man creates meaning in his life from the chaos of existence; he creates himself, his essence. Frost (1962), in his overview of the existential philosophy of Heidegger, Jaspers, and Sartre, eloquently concluded "...from his existence man is free to make of himself what he chooses" (p. 266). In existential personality theory, the struggle and anxiety engendered in a person's quest to make existence meaningful, to construct him- or herself, are absolutely necessary precursors to the development of the authentic personality (Feshbach & Weiner, 1986; Kobasa & Maddi, 1977). Authenticity is that maturity and full personal development which comes about when one has the courage, willingness, and fortitude to confront anxiety and choose to change meaningfully in an ever-changing world. An existentially authentic person accepts the anxiety associated with a yet-to-be-constructed future and its uncertainty as a "necessary concomitant of vigorous living" (Kobasa & Maddi, 1977, p. 243). Conversely, the inauthentic person has been characterized as one driven by predetermined social roles, biology (beyond the truly unchangeable thrownness or facticity of life), and various external influences (Kobasa & Maddi, 1977). The inauthentic person engages in stereotypical behavior (lacking original assertion and action), is insecure, fears the uncertainty of the future and all the change and instability which it inevitably brings, and

fails to actively live in the moment, being instead in a state of fear or anxiety about what is to come or locked into the guilt and regret of an unrealized past (Kobasa & Maddi, 1977). The authentic and dynamic state of existence has been referred to as being-for-itself (Sartre, 1956), being-in-the-world (Ewen, 1988), or Dasein (Heidegger, as related in Kobasa and Maddi (1977)); it is, in sum, "that mode of existence distinctive to the human being which is never static but is always in the process of revealing new things about itself and its world through decision making as a vehicle for creating meaning" (Kobasa & Maddi, 1977, p. 245).

Existential personality theory is deeply rooted in the work and writings of Binswanger and his conceptualization of a "fundamental meaning structure" (Kobasa & Maddi, 1977). This concept essentially refers to the unique and pervasive, unlearned human capacity to transcend concrete situations through the creative attribution of meaning to that which occurs in the realm of events (Binswanger, 1963). The congruency between this fundamental meaning structure and the postulated role of appraisal and perceptions of events in the experiencing of stress (as mentioned in the introduction of this work) is rather profound. Various psychotherapies which have arisen from existential philosophy, such as Frankl's logotherapy (heavily influenced by his survival of a Nazi concentration camp and the observation that those who survived created a meaningful existence) (Frankl, 1984) and the Daseinanalyse of Binswanger, are grounded in the existence of a unique human capability to give meaning to existence (Kobasa & Maddi, 1977). The following assertions are key to existential personality theory and are outgrowths of the collective existential philosophy of the philosophers mentioned above as

well as many others (Kobasa & Maddi, 1977). The association between these assertions, the construct of hardiness, and behaviors presumed to be related to hardiness will become rather self-evident in the pages ahead. Important and relevant assertions for this effort include:

> "Personality is primarily *constructed* (italics added) through the person's attribution of meaning.
> Persons are characterized by symbolization, imagination, and judgment.
> Persons are characterized by their *participation* (italics added) in society.
> Persons are characterized by their *participation* (italics added) in a physical and biological environment.
> Human life is best understood as a *series of decisions* (italics added).
> Personality is a synthesis of *facticity and possibility* (italics added).
> Development is best understood as the interaction of psychological, social, and biological-physical components of existence.
> The *imposition of limits* (italics added) stimulates positive development.
> *Richness of experience* (italics added) stimulates positive development.
> Personality development ideally becomes increasingly *self-determined* (italics added).
> The experience of failure stimulates *self-determined* (italics added) development.
> In social interaction, the authentic being is *oriented toward intimacy* (italics added), whereas the inauthentic being is oriented toward superficial and contractual relationships.
> In relationship to social institutions, the authentic being is *active and influential* (italics added) whereas the inauthentic being is passive and acquiescent.
> The authentic being shows *continual change* (italics added), whereas the inauthentic being remains the same" (Kobasa & Maddi, 1977, pp. 251-261)

A couple of positive states (attitudes) that come about in the authentic personality are caring and courage (Kobasa & Maddi, 1977; Orr & Westman, 1990). Caring is active and aggressive involvement in confronting one's needs and considering the resources one possesses, as well as those offered by the environment and others, in meeting those needs and so making life meaningful. Courage refers to one's recognition of hard facts, of the ever-changing, chaotic nature of the world, and the conviction one is able to exert control

over life events in this maelstrom. That is, while change is normal and certain, one can act courageously, welcoming the challenge, yet still exerting a great deal of control, a whitewater kayaker challenged and tossed by the rapids of life, but not afraid of them, and controlling his or her progress through them. The states of caring and courage are key to hardiness, giving rise to the three subcomponents of the hardiness construct.

Hardiness is composed of three subcomponents: commitment, control, and challenge (Kobasa et al., 1982; Kobasa, 1979a; Orr & Westman, 1990). Commitment is a direct outgrowth of the authentic state of caring, while control and challenge are derived from courage. Commitment is a tendency to involve oneself in (rather than being alienated from) whatever one does. Committed people have a sense of purpose in their lives, and the events and others in their environment are meaningful. Committed people develop intimate and meaningful relationships with others. A committed person's relationship to him/herself and the environment is characterized by active (versus passive or avoidant) involvement. Control is a tendency to "feel and act as if one is influential (rather than helpless) in the face of varied contingencies of life" (Kobasa et al., 1982, p.169). Someone with an internal (versus external) locus of control is not overwhelmed by life's events, being more likely to act to transform events into something consistent with one's life plans, and so maintain meaningfulness in one's life. Finally, challenge is the conviction that change, not stability, is the normal life condition. Changes are seen as "interesting incentives to growth rather than threats to security" (Kobasa et al., 1982, p.170) by a person with the challenge disposition. For someone high in challenge, events demanding action are more likely to be perceived as stimulating, not threatening, and they will foster

transformation and growth versus avoidance or a clinging to the secure existence of the past. It is evident that many of the key assertions in existential personality theory (participation, self-determination, richness of experience, an active and influential orientation, and embracing change), as outlined in the preceding paragraphs, are mirrored in the three subcomponents of hardiness. Having outlined the subcomponents of hardiness, it is important to now address some of the concerns about hardiness and the unity of the construct.

Structure and Unity of the Hardiness Construct

A number of researchers have expressed concerns about the structure and unity of hardiness and Kobasa's conception that the three subcomponents interact to produce unique effects on stress and health which are different from the individual effects of the subcomponents. Before continuing an investigation of these criticisms, however, it will be necessary to discuss Kobasa's original operationalization of the hardiness construct and the construction of her questionnaire (Kobasa, 1979a, 1979b). The control dimension of hardiness was measured using a locus of control scale, subscales (powerlessness versus personal control) of an instrument measuring the construct of alienation, achievement and dominance subscales of a personality instrument, and a leadership orientation scale. The commitment dimension was measured using, again, a test for the alienation construct, and an instrument measuring social role consistency. Finally, challenge was measured using scales and/or instruments designed to measure experiential preferences, vegetativeness versus vigorousness (another alienation test subscale), security (stability) orientation, need for cognitive structure, a need for endurance instrument (to guage persistence), and an adventurousness versus responsibility scale. Obviously, the collection of instruments was rather eclectic in an initial attempt to effectively measure a theoretical construct. In all, Kobasa used 19 different scales/subscales in her 1979 effort. In this groundbreaking study, Kobasa found a number of scales that discriminated between high stress/high illness and high stress/low illness groups of executives (in addition to these "hardiness" scales, stress perceptions, but not life events measurements of stress, also differentiated the

groups, providing further insight into the role appraisal and individual differences may have in stress-illness relationships). The resulting 71-item long form of her hardiness instrument was composed of six scales: the Powerlessness Scale (Maddi, Kobasa, & Hoover, 1979) and the External Locus of Control Scale (Rotter, Seeman, & Liverant, 1962) to measure control, the Alienation From Self and Alienation From Work scales (Maddi et al., 1979) to measure commitment, and the Security Scale (Hahn, 1966) and Cognitive Structure Scale (Jackson, 1974) to measure challenge (the Cognitive Structure Scale was later eliminated as it did not share common variance with other scales; the Security Scale has often been doubled in calculating the challenge score per the precedent set by Kobasa et al., 1982). This 71-item instrument was abridged into 36- and 20-item instruments in 1982 (Funk, 1992; Orr & Westman, 1990). Since then, two "third-generation" instruments have also been created from the groundwork laid by Kobasa: the 50-item Personal Views Survey (PVS) (marketed by the Hardiness Institute) and the 45-item Dispositional Resilience Scale (DRS) (Bartone, Ursano, Wright, & Ingraham, 1989). Both of these instruments attempted to improve upon some of the psychometric problems which will be discussed in both this and a later section. Additionally, Nowack (1990) independently developed a 30-item scale which has, psychometrically, demonstrated great promise.

The challenge subcomponent of hardiness has come under tremendous scrutiny. In their review, Hull, Van Treuren, and Virnelli (1987), examined five studies on hardiness and found challenge to have predicted effects in only one of the five; they also reported that challenge is inappropriately related to commitment and control with hardiness

accounting for only 41% of variance in burnout scores compared to explaining 57% of variance when the challenge subscale was not included. In a factor analysis of Kobasa's 71-item hardiness questionnaire, still the most frequently used, Hull et al. (1987) found that items from the challenge subscale loaded weakly on the commitment and control factors and not at all on the challenge factor. Orr & Westman (1990) reported findings of only a modest correlation (0.46) between challenge and total hardiness with Kobasa's 36-item scale.

Funk and Houston (1987) also had disconcerting findings when they conducted a principal-components factor analysis on the hardiness subscales of the 71-item questionnaire. Using an orthogonal rotation, they found the powerlessness scale (indexing control) loaded on the commitment factor, and the security scale (indexing challenge) loaded heavily on the same factor as the external locus of control scale (measuring control). Kobasa et al. (1982) found control and commitment subscales loaded onto one factor in her single attempt at factor analysis while the Security Scale (challenge) loaded poorly onto this factor; as mentioned before, the Cognitive Structure Scale did not load onto this "general hardiness" factor and was subsequently dropped from the 71-item instrument. While all this duly generates scepticism about the structure of hardiness and its unity, others have supported the soundness of the construct. In their review, Orr and Westman (1990) report findings that Kobasa's 20-item and the 50-item (PVS) questionnaires loaded satisfactorily and appropriately on three interrelated factors, with challenge "appearing as a distinct and salient concept in these two scales" (p.70). Orr and Westman note the challenge subscale used on the 36- and 71-item questionnaires was

originally constructed to measure a wide variety of security concerns, many of which were inconsistent with challenge as it relates to existential personality theory concepts. Deletion of inappropriate items on the challenge subscale was done, accordingly, for the 20- and 50-item scales. Funk's (1992) review of factor analyses concerning the structure of hardiness instruments indicated that the DRS appeared to load onto three appropriate factors; correlations between the construct's components, however, were significant, and provide a basis for arguing that the representation is that of a unified construct. In 1991, Hull, Lehn, and Tedlie used structural equation modelling to examine hardiness as measured by the 71-item long form. Finding acceptable goodness of fit, they concluded "...hardiness would seem to be adequately modeled as a single latent variable..." (p. 938). However, they qualified their conclusion, noting two of the five measures, the security scale (challenge) and the external locus of control scale (control), shared little variation with each other or the hardiness construct (for both, more than 90% of the variance appears to be unrelated to hardiness). Rush et al (1995), incorporating other scales in measuring hardiness subcomponents, found acceptable goodness of fit in their structural equation modelling analysis, concluding hardiness represents a single latent construct. Nowack's (1990) Cognitive Hardiness Survey robustly demonstrates a unitary factor structure (K. Nowack, personal communication, February 1996) which has yet to be challenged in the literature. Huang (1995), in a critical review of hardiness and stress, concluded that, despite cause for concern over inconsistent correlations between the dimensions of hardiness, the construct should be treated as a single composite measure of its components. Ouellette (1993) notes Bartone has also advocated use of the composite

(versus componential) hardiness (DRS instrument) score. Interestingly, it is the challenge component which appears to most distinguish hardiness from other personality variables (optimism, sense of coherence, general expectancy of control, type A) thought to moderate and/or mediate the stress-health relationship (Ouellette, 1993). Finally, Contrada (1989) notes that challenge was the factor differentiating cardiovascular reactivity (diastolic blood pressure (DBP)) in hardy versus less hardy individuals. Given these conflicting findings, I agree with Orr & Westman's (1990) and Ouellette's (1993) recommendation challenge (and consideration of hardiness as a unified construct) not be eliminated (as Hull et al. suggested in 1987) pending further empirical testing with the available instruments.

The Psychoneuroendocrinology of the Stress Response

Hardiness is postulated to moderate stress via several pathways involving cognitive appraisal (Kobasa, 1985; Maddi & Kobasa, 1984; Orr & Westman, 1990; Westman, 1990). Importantly, appraisal, a necessarily psychological event, may well initiate a psychoneuroendocrine cascade of responses to stressors. Cognitive appraisal is a continual evaluative process by which we categorize events, determine their significance for our well-being, and determine which course of action is appropriate (Lazarus & Folkman, 1984). Appraisal has been demonstrated to have a strong relationship with selected coping strategies (Folkman, Lazarus, Dunkel-Schetter, DeLongis, & Gruen, 1986). Stroebe and Stroebe (1995) similarly denote cognitive appraisal is "an evaluative process which determines why and to what extent a particular situation is perceived as stressful by a given individual" (p. 185). Primary appraisal is, then, the determination as to whether something is threatening or beneficial and in what ways it is so, at present or in the future; secondary appraisal, on the other hand, is the determination of options, skills, abilities, and resources which may be brought to bear on coping with an event (Lazarus & Folkman, 1984). In general, hardy people are thought to perceive and experience less stress because they perceive events and situations as less threatening (primary appraisal), and they believe they have the ability to effectively cope with the stressors (secondary appraisal) (Kobasa et al., 1985; Kobasa et al., 1982; Orr & Westman, 1990; Rush et al., 1995; Wiebe, 1991; Westman, 1990). Primary appraisal appears to most strongly mediate the relationship between stimuli and the stress experience (Dobson & Neufeld, 1979; Florian, Mikulincer, & Taubman, 1995).

Primary appraisals can be either irrelevant, benign-positive, or stressful (Lazarus & Folkman, 1984). Irrelevant appraisals are self-explanatory and can be characterized by feelings of indifference (Larsson, 1987). Benign-positive appraisals occur when the event is construed as maintaining or enhancing well-being. Stressful appraisals are harm/loss, threat, and challenge appraisals. Threat appraisals concern the anticipation of harm or loss when confronted with a certain event; they may be associated with emotion-focused (regressive) coping patterns which do not transform the situation (Lazarus, 1993a). Threat appraisals may be conceptualized to subsume post-event harm/loss appraisals as well (Lazarus & Folkman, 1984). Challenge appraisals of an event focus on the "potential for gain or growth inherent in an encounter" (Lazarus & Folkman, 1984, p. 33). While threat appraisals are associated with negative emotions such as fear, anxiety, and anger, challenge appraisals are characterized more often by emotions such as excitement and exhilaration (Lazarus & Folkman, 1984). Indeed, the appraisals made may only be revealed in the emotions elicited; often, appraisals may be done automatically or below the level of consciousness (Lazarus, 1993a, 1993b). In contrast to threat appraisals, challenge appraisals may be more often associated with problem-focused (transformational) ways of coping with events (Lazarus, 1993a). Relevantly, Maddi and Kobasa (1984) vehemently assert coping (transformational versus regressive) mediates the hardiness-stress relationship. In sum, whether or not an event is stressful, whether or not it elicits positive or negative emotional states, likely depends largely on how it is appraised. Also pertinent is the contention that challenge and threat appraisals may be expressed in physiologically different ways (Lazarus, 1993a). Lazarus and Folkman (1984) state appraisal processes

"provide a common pathway through which person and environment variables modify psychological response, and hence emotions and their biological concomitants" (p. 224).

It is encouraging that literature supportive of the above position exists. In animal studies, those animals that are socially dominant respond to stressful events in a physiologically different (more beneficial) way than those which are socially subordinate (Blanchard, Sakai, Weiss, & Blanchard, 1993; see Drummond, 1996; Fuchs, Uno, & Flugge, 1995; Sapolsky, 1992a, 1992b) in interaction with such such factors as dominance style and social stability (Sapolsky, 1992b). Individual experience with a stressor may alter physiological stress responses (Southwick et al., 1994), perhaps through processes of habituation and sensitization (McCarty, Konarska, & Stewart, 1992). The psychological component, to include situational appraisal, cannot be ignored in examining such physiological response (Wolff, Friedman, Hofer, & Mason, 1964; Sapolsky, 1992b), as it may meaningfully explain variance in the physiological response. As previously mentioned, there is empirical support for the contention that most stress is, in actuality, the product of our perceptions, and, therefore, self-produced (Epstein & Katz, 1992). It is not surprising primary appraisal has been found to be predictive of psychological adaptation (Folkman, Lazarus, Gruen, & DeLongis, 1986) and is explanatory of variance in anxiety (Edwards & Endler, 1989).

All considered, it appears reasonable to consider cognitive appraisal as a mediating factor between events and experienced stress. Again, hardiness may well influence the appraisals that are made and the effectiveness of the coping that flows from such appraisals (Kobasa et al., 1982; Kobasa, 1979a; Maddi & Kobasa, 1984). It is apparently

from the ultimate perception of stress, then, that the neuroendocrine response flows and potentially degrades various bodily functions to hinder health and performance. It is important, therefore, to now examine what happens neuroendocrinologically in the stress response; the cascading events of such a response may well mediate stress-illness and stress-performance relationships.

When a stressor is perceived (and presumably appraised as stressful), a number of neurotransmitters and neuropeptides (including acetylcholine (ACh), serotonin (5-HT), norepinephrine (NE), gamma-aminobutyric acid (GABA), epinephrine, central corticotropin-releasing hormone/factor (CRH/CRF), neuropeptide Y (NPY), and neurotensin, to name a few) collectively act to regulate the hypothalamus (HTH) and, in particular, the paraventricular nucleus (PVN) of the HTH (Black, 1994a; Dunn & Berridge, 1990; Reichlin, 1993; Rowe, Viau, Meaney, & Quirion, 1995; Whitnall, 1993). With the exception of GABA, the above seem to exhibit predominantly stimulatory effects upon the PVN. Within moments, increased CRH (a 41-amino acid peptide) mRNA is detectable in the parvocellular neurons of the PVN (Black, 1994a; Whitnall, 1993). The CRH (along with other HTH generated hormones) is transported along axons to the external zone of the median eminence, where it is released into capillaries of the HTH-pituitary gland portal venous circulation (Black, 1994a; Whitnall, 1993). Upon reaching the anterior pituitary gland, the CRH induces cleavage of proopiomelanocortin (POMC) into adrenocorticotropic hormone (ACTH), β-endorphin, and other compounds in corticotropic cells (Brown, 1994; Black, 1994a; Sheridan, Dobbs, Brown, & Zwilling, 1994).

It is worth noting that vasopressin (VP) has also been implicated in stimulating ACTH release (Sawchenko, 1991; Whitnall, 1993). In rats, about half of the parvocellular CRH-expressing neurons also express and secrete VP; in mice, all neurosecretory CRH cells contain VP, the two appearing to act synergistically to drive synthesis and release of ACTH from the anterior pituitary corticotrope cells. Minton (1994) notes that in some species (sheep, for example), VP is far more potent than CRH in its capacity to stimulate ACTH secretion (although most species appear to respond primarily to CRH). Consistent with this, ACTH exhibits short-loop feedback regulation of VP (in addition to CRF) expression in the parvocellular neurosecretory neurons (Sawchenko & Arias, 1995). It may well be the case in most species that VP potentiates the effect of CRH on ACTH secretion; this has been demonstrated in rat anterior pituitary corticotrope cells in vitro (Whitnall, 1993). Whitnall also reviewed research strongly suggesting that ACTH release in different corticotrope cell subpopulations may be either VP or CRH dependent; perhaps this structuring is partially responsible for the anterior pituitary's capability to maintain appreciable ACTH release in response to repeated sequential stimuli.

It is well known that the neurosecretory magnocellular cells in the PVN, producing primarily VP and oxytocin (OXT), project to the posterior pituitary (Sapolsky, 1992a). Some researchers (Sawchenko, 1991; Whitnall, 1993) postulate these neurohypophyseal projections could, conceivably, posit VP in the median eminence, or in the anterior pituitary (via diffusion), although this is not yet supported and a matter of great debate. Recently, a role for histamine (perhaps of importance given popular consumption of various antihistamine drugs for allergies) has been discovered in such processes. Knigge,

Kjoer, Larsen, Jorgensen, Bach, Moller, et al. (1995) found histamine, acting differentially via two receptor subtypes, profoundly stimulates gene expression and release of POMC in both the murine anterior and intermediate lobes of the pituitary gland. Certainly, the stress-related activities of the PVN and hypothalamic-pituitary-adrenal (HPA) axis are not yet fully defined. Nevertheless, ACTH enters the circulatory system from the anterior pituitary, and, arriving at the adrenal cortex, stimulates the release (from the zona fasciculata of the cortex) of a family of steroid hormones called glucocorticoids (GCs—the most dominant human form is cortisol). Elevated blood levels of GCs are noticeable within a few minutes of exposure to a stressor (Sapolsky, 1992a) due to HPA axis activation although plasma levels do not peak in response to an acute stressor until 30 minutes after initial exposure (Glaser & Kiecolt-Glaser, 1994). To appreciate the pervasive (and immunosuppressive) effect GCs are postulated to have on physiological functioning, it is worth noting GC receptors have been identified in "virtually every nucleated cell type in the body" (Munck, Guyre, & Holbrook, 1984, p. 27), to include cells of the immune system (Shepherd, 1994).

In addition to the actions of the HPA axis in elevating glucocorticoid levels, stressors (or rather, the concomitant appraisals) appear to drive HTH and pituitary regulation of a vast array of secretagogs and hormones (Sapolsky, 1992a). Through neural projections into the posterior pituitary, as mentioned earlier, the PVN releases VP and OXT. HTH release of somatostatin (SS) and growth hormone-releasing hormone (GHRH) causes anterior pituitary inhibition/release, respectively, of growth hormone (GH); in humans, brief stressors seem to stimulate GH release while chronic stressors tend

to inhibit GH release. HTH release of gonadotropin-releasing hormone (GnRH), and accompanying release of reproductive hormones and gonadal steroids, is inhibited by stress. Pancreatic release of insulin is inhibited by stress, while release of glucagon is stimulated. Finally, the HTH stimulates release of prolactin by the anterior pituitary during stress.

Complementing neuroendocrine mediation of the stress response, the autonomic nervous system (ANS), and specifically the sympathetic nervous system (SNS), directs facets of the physiological stress response (Sapolsky, 1992a). Sympathetic activation of the adrenal medulla (and the whole of the sympathetic adrenal medullary (SAM) system) stimulates release of epinephrine into the bloodstream within seconds, and sympathetic NE projections innervate virtually every organ in the body (innervation of lymphoid tissues will be addressed later). Demonstrating the magnitude of sympathetic activation, intracerebroventricular (icv) administration of CRF (central CRF is widely postulated as mediating not only HTH-pituitary activity, but also stress-related sympathetic activation (Black, 1994a; Chrousos & Gold, 1992; Dunn & Berridge, 1990; Irwin, Hauger, Brown , & Britton, 1988)) in rats results in significant increases in plasma levels of epinephrine (238%) and NE (209%) (Kurosawa et al., 1986). In general, sympathetic stimulation in response to stress is characterized by arousal, vigilance, and increased heart rate, blood pressure (also influenced by the anti-diuretic effects of VP), and respiration.

It is important to note that apparent cardiovascular reaction (as measured by heart rate (HR), systolic blood pressure (SBP), and DBP) to stressors in humans is, in reality, regulated both sympathetically and parasympathetically, calling into question their

reliability as markers of SAM activity (Cacioppo, 1997; Cacioppo et al., in press). Heart rate (HR), for example, may rise due to sympathetic input or vagal withdrawal or some combination of the two. It is true, however, that HR and SBP, as observed in several research efforts, may be somewhat acceptable as imperfect markers of of sympathetic reactivity (Cacioppo, 1997, Cacioppo et al, in press; Uchino, Cacioppo, Malarkey, & Glaser, 1995). While ventricular pre-ejection period (PEP) is the preferred indicator of sympathetic activity (via use of impedance cardiography), it correlates with HR, and SBP appears to mediate the relationship between PEP and some parameters of immunofunction (Cacioppo, 1997; Uchino et al., 1995).

It is worthwhile reviewing the key role CRH plays, not only in its aforementioned ability to cleave POMC and release its various compounds (for a more complete treatment of POMC cleavage, the reader is directed to Brown (1994)), but also in its neuromodulatory functions in the stress response. Bioactive CRH and CRH receptors have been identified in various regions of the brain other than the HTH PVN (the PVN displays a high density of CRH receptors); these include cerebral and cerebellar cortex, the olfactory bulb, limbic system, choroid plexus, and regions involved in autonomic nervous system regulation such as the locus ceruleus (LC) (Dunn & Berridge, 1990; Licinio, Bongiorno, Gold, & Wong, 1995). Both acute and chronic stressors have been associated with the increase of cerebral CRH concentrations (Chappell, Smith, Kilts, Bissette, Richie, Anderson, et al., 1986). It is well-known that PVN axons project widely to brain stem autonomic nuclei, especially the LC, and icv CRH infusion induces the physiological stress response as well as arousal behaviors; icv introduction of the CRH antagonist, α -helical

CRF, into mice prior to restraint stress largely abolishes (in a dose-dependent manner) the stress-induced physiological changes and behavior (Black, 1994a; Dunn & Berridge, 1990). Recent discovery of a gene encoding for proopiomelanocortin corticotropin-releasing hormone responsive element binding protein 1 (PCRH-REB-1) (Licinio et al, 1995), hypothesized to be a marker for cellular response to CRH in many of the above mentioned areas of the brain (including the LC), suppports the notion that CRH plays a central role in the stress response aside from its strictly HPA activity. Indeed, similar to the above, CRH antagonists infused into the LC decrease NE secretion and behavioral aspects of the stress response (Valentino, Foote, & Page, 1993). Of great interest, and perhaps indicative of the expansive function of central CRF in the stress response, Liang, Melia, Campeau, Falls, Miserendino, and Davis (1992) found the excitatory effects of icv infused CRF on the murine acoustic startle reflex were blocked by lesioning the central nucleus of the amygdala. Lesioning of the PVN did not block excitation, and CRF infusion of the amygdalar central nucleus could not create the stimulatory phenomenon. It is evident that some aspects of centrally CRF-mediated stress responses are not PVN dependent, and while the amygdala is a key component of the stress response, it is not, apparently, the primary CRF receptor site in the acoustic startle reflex. Liang et al. have implicated the parabrachial nucleus and the dorsal lateral tegmental nucleus as possible primary sites for icv CRF excitation of the acoustic startle reflex.

In controlling the stress response, the limbic system, especially the amygdala, appears to play a key role as the gateway between the cortex and the HTH. The amygdala seems to integrate sensory information, emotional transactions, and other cortical inputs

(Melnechuk, 1988; Uwano, Nishijo, Ono, & Tamura, 1995) in its communications with the HTH via two major efferent descending projections, the stria terminalis (and bed nucleus of the stria terminalis) and the ventral amygdalofugal pathways (Feldman et al, 1990, as cited in Whitnall, 1993; Kupferman, 1991). As might be expected, microinjection of glutamate into the amygdala has been shown to result in a dose-dependent stimulation of CRF release from the median eminence resulting in relatively long-lived serum corticosterone increases in rats (Gabr, Birkle, & Azzaro, 1995). Further implicating amygdalar involvement, projections from the amygdala (and other limbic structures for that matter), are known to terminate rather densely among cell bodies in the perinuclear area around the PVN; this has been postulated as an indirect pathway by which the amygdala regulates PVN activity during the stress response (see Whitnall, 1993). Anterograde tracing of amygdaloid projections to the LC, again, a major brain stem structure in SNS activation (Black, 1994a), suggests the amygdala can also directly control NE cells in the LC projecting to the HTH, presumably in the integration of responses to stressors (Wallace, Magnuson, & Gray, 1992). Bilateral ablation of the afferent ventral noradrenergic bundle from the brainstem to the PVN inhibited CRH gene expression in the HTH, POMC gene expression in the anterior pituitary, and, accordingly, adenohypophyseal ACTH release normally associated with intraperitoneal interleukin-1 β injection (Parsadaniantz, Gaillet, Malaval, Lenoir, Batsche, et al., 1995); not surprisingly, stimulation of these pathways has been shown to demonstrate increases in CRH levels in the infundibular stalk (see Whitnall, 1993). NE projections from the caudal nucleus of the solitary tract and the LC and epinephrine-containing axons have been observed to

terminate on PVN parvocellular CRH neurosecretory cells, perhaps preferentially upon those co-expressing for VP (Whitnall, 1993). Chrousos and Gold (1992) also endorse the likelihood of complex and prominent interactions between neuromodulatory CRH and NE pathways as suggested in the past several pages. An elaborate interrelationship between the HPA axis and SAM systems is further indicated by the recent work of Cacioppo et al (in press).

Cacioppo and his colleagues (Cacioppo, 1997, 1994; Cacioppo et al., in press; Cacioppo, Malarkey, Kiecolt-Glaser, Uchino, Sgouta-Emch, Sheridan, Berntson, & Glaser, 1995; Kiecolt-Glaser & Glaser, 1995; Uchino et al., 1995) have found that those individuals demonstrating greater SAM activation (high reactors) during brief stressors (public speaking and mathematical tasks), as indicated by HR increase or diminished PEP, also exhibit the most profound changes in HPA activity (increased cortisol secretion). Those who demonstrate cardiovascular reactivity that is parasympathetic in character or those who are low reactors do not exhibit the increased cortisol secretion. HPA activation, as marked by sympathetic reactivity, has been associated with immunocompetence compromise, and forms the foundation of the emerging stress reactivity hypothesis (Cacioppo, 1997; Cacioppo et al., in press). All this supports a conceptualization of an interrelated and highly complex neuroendocrine-autonomic response to stress, perhaps modulated by central CRH. To further complicate matters, recent research suggests differential neural pathways for chronic and acute stimulation of the HPA axis and SAM systems (Malarkey, Lipkus, & Cacioppo, 1995).

The hippocampus provides inputs to the HTH (Kupferman, 1991), and, noting the presence of hippocampal GC receptors, appears to play a role in modulating (inhibiting) stress-induced adrenocortical activity (DeKloet, Oitzl, & Schobitz, 1994; Wilson, 1985). To the extent it does play a role in HPA regulation, and given prominent hippocampal function in learning and memory, it is of concern that elevated GC levels suppress hippocampal plasticity (Pavlides, Kimura, Magarinos, & McEwen, 1995) and may cause development of "permanent cognitive deficits" (Yau, Olsson, Morris, Meaney, & Seckl, 1995, p. 579). Indeed, elevated GC levels (as indicated by hyperplastic adrenal cortices) in vervet monkeys have been associated with catastrophic hippocampal neurodegeneration (Uno, Tarara, Else, Suleman, & Sapolsky, 1989). Such ominous findings and their pertinence to job-related cognitive functioning will be later examined. Probable hippocampal influence on HPA control and evidence tumor necrosis factor (TNF) can dramatically downregulate type II (low affinity) glucocorticoid receptors in the rat hippocampus (Betancur, Borrell, & Guaza, 1995) indicate hippocampal involvement in immunoregulation which is not yet fully understood. Limbic system involvement in control of HTH-directed aspects of neuroendocrine and sympathetic stress response appears prominent in its potential to mediate cognitive and other CNS influences on immune function.

There appear to be a whole host of CNS inputs to the PVN, in particular, and the HTH, in general, which are probably involved in regulation of the stress response. Contributing structures include the suprachiasmatic nucleus, septal area, organum vasculosum lamina terminalis (OVLT), arcuate nucleus, and midbrain raphe nuclei. A

wide variety of neurotransmitters and neuropeptides are also likely involved, and investigation of central regulation of the PVN and the stress response is truly in its infancy. The reader is directed to Whitnall's (1993) outstanding review of the literature for a fuller treatment of central structures implicated in regulation of the stress response.

As a whole, the neuroendocrine and SNS facets of the stress response are quite sensible in that they promote the "fight or flight" capability and mobilize energy (Sapolsky, 1992a). For example, plasma glucose levels are increased, VP increases blood volume (particularly important if blood loss occurs), while increased heart rate, peripheral vasoconstriction/internal vasodilation, and respiration get more oxygen and glucose to the muscle mass faster. ANS shutdown of vegetative functions liberates more blood to fuel muscles that need it. GCs suppress inflammatory responses while opiate-mediated analgesia blunts pain. Growth and tissue repair, reproductive functions, and the immune system are likely inhibited because they can reasonably be considered a low priority in responding to the physical stressors our evolutionary ancestors would have faced. While the stress response is effective in dealing with physical threats to survival and well-being, the response appears to become pathological when it is chronic or when it is activated without physiological reason (due to psychological and psychosocial stressors) (Sapolsky, 1992a, 1992b). In essence, activation of the stress response to psychosocial stressors may well be an unadaptive vestige of some evolutionary past , although this is open to debate. Appropriately, Whitnall (1993, p. 604) discusses the need to better answer (in future research) questions such as, "What is the adaptive value of a glucocorticoid response to stress?" Having reviewed pertinent aspects of the psychoneuroendocrine response to

stress, it will be timely to review some of the hardiness research, and, perhaps, begin to contextualize research findings in a multidisciplinary approach to hardiness, in particular, and stress, in general.

Hardiness: An Overview of the Research

As mentioned before, hardiness is thought to moderate stress through pathways involving cognitive appraisal (Kobasa, 1985; Maddi & Kobasa, 1984; Orr & Westman, 1990; Westman, 1990). To review, hardy people are thought to perceive and experience less stress because they view events and situations as less threatening (primary appraisal), and they believe they have the ability to effectively cope with stressors (secondary appraisal) (Kobasa et al., 1985; Kobasa et al., 1982; Orr & Westman, 1990; Rush et al.,1995; Wiebe, 1991; Westman, 1990). The postulation is reasonable, consistent with hardiness and existential theory, and has been supported in a number of studies (Allred & Smith, 1989; Kobasa et al, 1985; Maddi & Kobasa, 1984; Rhodewalt & Zone, 1989; Wiebe, 1991; Westman, 1990). Interestingly, Rush et al. (1995) found that while hardiness was significantly correlated with control (active) coping, hardiness exhibited a far stronger direct impact on perceived stress, its effect not being mediated by coping strategies. This implies the prominence of primary appraisal in determining the relationship between events and subjective stress. Dobson and Neufeld (1979) found the pathway between stimulus and stress was mediated by perceptions of aversiveness and anticipation of degree of stress. Noticeably, the stimulus-stress relationship was not significantly mediated by perceived coping inefficacy. Florian et al. (1995) found that the commitment and control dimensions of hardiness were positively related to psychological well-being via their negative relationship to threat appraisals, again supporting a prominent role for primary appraisal processes. Additionally, such a finding lends credence to aforementioned conceptualizations of appraisal as a mediating mechanism in the hardiness-

stress relationship. Florian et al also found commitment to be negatively correlated with distancing (regressive) methods of coping, and distancing was predictive of psychological distress. Consistent with the idea that appraisal (versus the actual stressor) determines the stressfulness of events and situations, recall Epstein and Katz (1992) reported total stress was correlated with self-produced stress and not correlated with externally produced stress. This is significant in that it may indicate a methodological flaw in much of the first eighteen years of hardiness research, wherein perceived stress was not measured, the favored approach being to tally the occurrence and frequency of postulated stressors (via scales such as the Holmes & Rahe Social Readjustment Scale (1967) and other life events inventories). Some have raised the issue that subjective valuations of stress are confounded with outcomes of interest (Dohrenwend, Dohrenwend, Dodson, & Shrout, 1984). Yet others eloquently argue for the usefulness of a subjective conceptualization (Cohen et al., 1983; Hills, & Norvell, 1991; Lazarus, DeLongis, Folkman, & Gruen, 1985) demonstrating explanations of unique variance in outcomes (beyond variance explained by life events or postulated confounds such as negative affect, depression, or neuroticism) and insisting stress is "best regarded as a complex rubric consisting of many interrelated variables and processes rather than as a simple variable that can be readily measured and correlated with adaptational outcomes" (Lazarus et al, 1985, p. 770). As a "complex rubric," stress is similar to inclusive constructs such as emotion and motivation. Indeed, Lazarus et al. (1985) assert removal of the subjective component from conceptualizations of stress necessarily returns one to behaviorist perspectives in denial of meaningful cognitive processes. Such a perspective, for example, could not account for the findings

of Epstein and Katz (1992) nor the explanatory power and parsimony of Lazarus's holistic cognitive-motivational-relational theory of emotion (which, by the way, views stress as a subset of the emotions) (Lazarus, 1993a). Such a sterile view of stress and its measurement denies an interactive person-environment relationship (Lazarus et al, 1985) and inevitably would result in conclusions of human passivity, powerlessness, and meaninglessness in an existential universe of events (Maddi, 1987; Kobasa, 1979b). To equate the experience of stress (and interest in that experience and its consequences) with a series of unchangeable events that "happen" to us not only imprisons psychological investigation in some inaccessible "black box," it also prevents the advance of an understanding which may well be dependent upon characterization of cognitive processes. It is fairly evident, then, that perceptions of stress (and resultant strain), more appropriately than stressors, and perhaps along with stressors (where appropriate), should be measured in hardiness research (this has been done by Rush et al. (1995) and Westman (1990), for example).

Despite the volume of Kobasa's work in the early 1980s, it is worthwhile to inquire whether or not hardiness has uniformly demonstrated stress moderating properties. Funk (1992) and Funk and Houston (1987), in reviewing a number of studies, infrequently found the theorized buffering effects of hardiness and concluded hardiness does not seem to demonstrate a moderating effect on stress. However, recent literature, as well as the observation that Funk and Houston (1987) based their conclusion on a relatively small number of cases, seems to indicate hardiness is capable of moderating the relationship between stress and certain outcomes. Researchers have found significant hardiness X

stress interaction in stress-illness (Solano, Battisti, Coda, & Stanisci, 1993; Hills & Norvell, 1991; Banks & Gannon, 1988; Kobasa & Pucetti, 1983; Kobasa, 1982; Kobasa et al., 1982), stress-performance (Westman, 1990; Herlich, 1985), and stress-absenteeism (Tang & Hammontree, 1992) relationships. So, it appears that hardiness can moderate stress. It is important, however, to more closely examine selected studies which have demonstrated both the moderating and main effects of hardiness; in the following paragraphs, I will discuss several studies examining the role of hardiness as related to stress and various outcomes of psychological well-being and health. In doing so, I will maintain a roughly chronological review; doing so will allow the reader the opportunity to see the progression (or lack thereof) of hardiness research over the last 18 years.

Hardiness has often demonstrated its ability to moderate the stress-illness relationship. Kobasa et al. (1982) found in a prospective effort (covering 2 years) that hardiness interacting with stressful life events buffered middle and upper level managers against self-reported illness (when controlling for prior illness). Hardiness also exhibited robust main effects on illness in this study. However, Kobasa et al. (1983) later found the hardiness X stress interaction to be only "marginally significant" ($p = .06$) in its relationship to illness. Interestingly, this 1983 effort looked at both hardiness and Type A behavior; in stressful environments, those executives high in Type A traits only suffered greater illness (self-reported) if they were also low in hardiness! High "Type As" who were also high in hardiness did not experience greater incidence of illness. Howard, Cunningham, and Rechnitzer (1986) also published findings that suggest personality

hardiness moderates the relationship between Type A behaviors and the adverse cardiovascular outcomes that are oft associated with them.

Banks and Gannon (1988) examined 88 undergraduates in a prospective study covering a period of nine months. Hardiness was found to be a stable construct which buffered (moderating effect) the students from the adverse impact (self-reported illness) of stressors. Further, hardy individuals experienced fewer stressors and experienced stressors as being less subjectively stressful than did their less hardy counterparts. This finding would seem to support a mediational role for appraisal processes in the hardiness-stress relationship as previously discussed. Wiebe (1991) also found that high hardy subjects made less threatening appraisals and had less negative affect for a frustration tolerance task, while Williams, Wiebe, and Smith (1992) suggest that the main (not moderating) effects of hardiness on illness are mediated by coping processes. Williams et al. (1992) reported that hardiness was positively related to problem-focused coping and support-seeking and negatively related to avoidance coping.

Lawler and Schmied (1992) found that hardiness moderated the stress-illness (again, a self-report measurement) relationship, although the control subcomponent predominated. The study was prospective in design and covered a 12-month period. Future illness frequency was best predicted by the control X stress interaction, high resting systolic blood pressure, and low systolic reactivity (counter to what Cacioppo's stress reactivity hypothesis might predict). Future illness severity was best predicted by external locus of control and high resting systolic blood pressure. The possibility that cardiovascular parameters may mediate the hardiness/control-illness relationship appears

to not have been aggressively pursued by Lawler and Schmied (1992). Solano et al. (1993) also investigated the relationship between hardiness (and a number of other personality factors) and the stress-illness phenomenon in 112 Italian army officer cadets undergoing six months of training. In general, the Solano et al. study constitutes a well-designed prospective study in which illness was assessed rather objectively by personnel staffing the program's infirmary. Like many studies in the area of hardiness research, however, it was disappointing to note their dichotomization of data (so discarding information) and application of ANOVA analyses instead of regression or structural equation modelling. As life change units (stressful life events as measured by the Life Experiences Survey (LES) (Sarason, Johnson, & Siegel, 1978)) increased, those who were high in hardiness manifested decreased incidence of illness episodes while those who were low in hardiness displayed increased episodes of illness. It is worth noting the high and low hardiness groups did not differ when life change units were low. This is an important consideration in that lack of findings in hardiness research may be a product of design.

A retrospective 1989 study by Rhodewalt and Zone found that hardiness interacted with undesirable life changes and their effect on depression and illness. In addition to finding that hardiness buffered subjects against adverse psychological and physical health (self-reported) outcomes, and consistent with previous discussion, non-hardy individuals appraised a greater proportion of life experiences as being undesirable. Non-hardy individuals also felt greater adaptation was needed to cope with subjectively negative events. One concern about the Rhodewalt and Zone effort involves an ambiguous

theoretical grounding of their appraisal measurement; though informative, their appraisal measure is not referenced and has the appearance of being rather arbitrarily constructed. Hills and Norvell (1991) also found hardiness acted to buffer highway patrol officers from the adverse impact (retrospectively self-reported symptoms) of perceived global stress; the hardiness X stress interaction accounted for 27% of the variance in physical symptoms. Hardiness additionally demonstrated a positive predictive relationship with job satisfaction.

Tang and Hammontree (1992) found that hardiness failed to buffer 60 police officers against illness (self-reported) related to police or life stress. Hardiness appeared to moderate the police stress-absenteeism relationship when police stress was low, but not when it was high! These findings would seem counter to expected outcomes and direction of outcomes. Perhaps hardiness does not act as a stress resiliency resource beyond some given level of stressor intensity, severity, or duration. While the hardy trainees in the Solano et al. (1993) study appeared to be more resilient in high stress situations than their low hardy counterparts, it may be important to consider their environment (training versus actual). Tang and Hammontree's finding is not entirely unique. Neubauer (1992) found that critical care nurses with high absenteeism rates in high pressure, low control environments were more hardy than contemporaries with lower rates of absenteeism. Perhaps in ill-defined work environments, especially if penalties are minimal, hardy individuals may use absenteeism to actively control some aspect of their work environment, such as their availability for work. In Neubauer's study, the less hardy nurses who remained in the high pressure, low control environment manifested higher

illness rates. It is worth noting that this positive relationship between hardiness and absenteeism was not found outside of these two studies.

Westman (1990) found, in her prospective study, that hardiness robustly moderated the stress-performance relationship in 326 officer cadets undergoing training in the Israeli Defense Forces (IDF). Hardiness was positively correlated with performance on navigation and obstacle clearance tasks. Hardiness was also associated positively with grades in a follow-up course and performance through the first year of commissioned service in the IDF. Westman's study currently stands as the seminal piece on hardiness and its relationship to performance. A couple of other research efforts have found hardiness to be positively related to performance in police training (Herlich, 1985) and resident assistants in college dormitories (Nowack & Hanson, 1983). The body of research investigating hardiness-performance relationships remains impoverished and will be targeted in the two studies which follow. Certainly, if hardy individuals are more likely to engage in productive coping efforts, as discussed earlier, then it would seem plausible that hardiness might be positively related to performance in a number of domains.

Goss (1994) found that hardiness buffered swimmers (on Canada's Olympic team) engaged in intense overtraining from various mood disturbances, including feelings of tension, depression, anger, fatigue, and confusion. These appear to be important psychological outcomes, although Goss questions their relationship to swimmers' performance in her closing comments. In all, hardiness has demonstrated an ability to moderate the relationships between stress and various outcomes of psychological well-being, health, and job-related importance.

Hardiness, however, has also demonstrated main effects on a number of dependent variables of interest. It is the frequency of such main effects, despite theoretical maintenance of the primacy of moderating effects, that has fueled the aforementioned criticisms by Funk (1992), Funk and Houston (1987), and more recently, Parkes (1994). Kobasa et al. (1985) found, in a prospective study, that hardiness was negatively predictive of illness (self-reported at one and two years from the start of the study). At one year into the study, hardiness had explained 22% of the variance in illness. At the two year mark, hardiness impressively accounted for 33% of the variance in illness. Further, hardiness appeared to work additively with other stress resiliency resources (exercise and social support) to buffer subjects against illness. Despite the implication that a moderating (versus main) effect for hardiness exists, the published article did not support other than a powerful main effect. Wiebe and McCallum (1986) also found that hardiness had a direct negative effect on illness (again via a self-report survey). While hardiness alone exhibited a weak but significant relationship to illness, its effects were mediated by health practices and subjective stress. When illness was regressed onto hardiness, stress, and health practices, 25% of the variance was accounted for. Such a finding again implies the effect hardiness may have on appraisal processes and transformational coping strategies.

The Tang and Hammontree (1992) and Neubauer (1992) studies discussed above suggest that hardiness, a relatively stable trait, may at times be subject to environmental influences. Relatedly, Maddi (1987) has supported the ability to train executives to be more hardy in their world view. In two courses lasting 15 hours each, Maddi was able to train 46 Illinois Bell Telephone managers to be more hardy. Pre- and post-testing (at

program completion and again 6 months later) support a significant training effect (increased hardiness). In addition, the increased hardiness was related to increased job satisfaction, reduced anxiety, reduced depression, reduced incidence of somatic symptoms, and decreases in SBP and DBP. This all suggests not only main effects for hardiness, but the malleability of what has been portrayed as a relatively stable trait. This malleability is a matter of concern in retrospective studies, for it does not permit inference of causal direction; in other words, with a retrospective study, one is left clueless as to whether the experience has influenced hardiness or vice versa.

Funk and Houston (1987) investigated the hardiness-illness and hardiness-psychological well-being relationships through both retrospective and prospective methodology. While measuring illness by self-report, it is notable that they had subjects keep a log of illness over eight weeks. This method may be beneficial in overcoming the fallibility and/or state-dependent dynamics of recall. While Funk and Houston found that the health log was significantly related to a post-period health problems survey, the correlations between the log and the survey dimensions of number of problems and severity of illness were somewhat unimpressive (.45 and .54, respectively). When subject maladjustment was controlled for, hardiness was associated with depression, but not health problems. Manning, Williams, and Wolf (1988) found, in a retrospective design, that hardy individuals in two organizations (insurance and manufacturing industries) experienced a higher quality of life, more positive affect, higher levels of job satisfaction, fewer work tensions, and were less depressed/anxious than their less hardy peers. Unlike

Funk and Houston (1987), they found that hardy individuals reported fewer somatic complaints.

Sharpley, Dua, Reynolds, and Acosta (1995) conducted a marvelous retrospective study in that they sampled from six professions comprising the staff at a major Australian university. Results from their sample may be more generalizable given the sample's cultural and educational diversity; in comparison, *every* study done by Kobasa and colleagues in the first decade of hardiness research appeared to be largely comprised of middle-aged, caucasian males in managerial positions. In measuring health, Sharpley et al. also used a self-report measure; however, inclusion of multiple dimensions addressed not only particular illness episodes, but also health related absenteeism, physician visits, and subjective overall ratings of general health. For the outcomes of anxiety, experiencing daily hassles, and job stress, hardiness accounted for 21, 15.2, and 13.7 percent of the variance, respectively. This exceeded variance explained by Type A behaviors, social support, and coping behaviors. Hardiness was also negatively associated with ill health, although Type A behaviors explained slightly more unique variance (3.4 versus 2.3 percent of the variance). Duquette et al. (1995) found hardiness to be the single largest predictor of variance in burnout for geriatric nurses, uniquely explaining 22% of the variance alongside various stressor measurements, social support in the workplace, and coping strategies (all variables explained 49% of the variance in burnout). Similarly, Topf (1989) reported that the commitment component of hardiness explained as much as 24% of the variance in burnout in nurses from a variety of specialties. Solcova and Tomanek (1994), in their retrospective effort, found also that hardy individuals appear to be more

psychologically healthy. Their results suggest that hardy individuals have greater self-efficacy, perceive fewer daily hassles, and employ problem-focused coping strategies. Rush et al. (1995), in yet another retrospective study, found that coping strategies did not mediate the hardiness-stress relationship in 325 senior-level state government employees enduring a period of pressure for change. Hardiness did, however, have a negative direct effect on perceived stress and a direct positive relationship with job satisfaction.

Nowack (1989, 1991) and Greene and Nowack (1996) conducted a series of investigations examining the relationship between hardiness and health. In the 1989 study, apparently of retrospective design, hardiness was found to be negatively associated with psychological distress, explaining 33% of the variance; hardiness was not, however, associated with physical illness outcomes. Hardiness was also positively related to occurrence of positive intrusive thoughts and problem-focused coping, while being negatively related to occurrence of negative intrusive thoughts. It is worth noting that the sample in this study was gender balanced and racially diverse, enhancing generalizability of results. In the prospective 1991 study, hardiness was significantly predictive of job burnout (one year later) on dimensions of emotional exhaustion and decreased personal accomplishment, accounting for 6 and 17 percent of the variance, respectively. Without controlling for initial psychological well-being, hardiness and stress together predicted over 55% of the variance in frequency of physical illness; that is, individuals with high stress perceptions and low hardiness were physically ill (per self-report survey) more often than their peers over the course of a year. Again, characteristic of Nowack's research, the sample was gender balanced and racially diverse. In the Greene and Nowack (1996)

study, a prospective three year effort with the Los Angeles Police Department, Nowack's Cognitive Hardiness Survey, but not Kobasa's 36-item instrument, predicted hospitalization (self-reported) for illness and injury. Hardiness was not, however, predictive of absenteeism. Finally, for those who become ill, is hardiness important? Pollock et al. (1990) would answer with a resounding "yes." In patients suffering from chronic illness (rheumatoid arthritis, hypertension, and multiple sclerosis), hardiness is associated, in the expected directions, with psychological and physiological adaptation, initiative and involvement in health-promoting activities, and participation in patient education efforts.

Main effects for hardiness have been found in two recent studies, both retrospective, and both involving stressors at the extremes of the human condition: combat and peacetime disasters with large numbers of fatalities. Hodgkinson and Shepherd (1994) assessed the effects of hardiness in 67 social workers providing psychological support (presumably to survivors and family members) following the Piper Alpha North Sea oil platform explosion in July 1988 (167 died and 62 were injured) and the Clapham Rail crash in December 1988 (35 fatalities and 118 injured). Hardiness explained 12 percent of the variance in psychological symptomology; significant effects in the predicted direction were found for somatization, obsessive/ compulsive behaviors, interpersonal sensitivity, and depression. Hardiness further explained 27% of the variance in overall psychological well-being (specifically on the total well-being and positive affect dimensions). Sutker et al. (1995) examined possible correlates of post-traumatic stress disorder (PTSD) in Persian Gulf War veterans. Avoidance coping, the control and

commitment dimensions of hardiness, and family cohesion emerged as the premier variables explaining variance in PTSD diagnosis; that is, greater use of avoidance coping, less family cohesion, and lower hardiness were all positively correlated with PTSD diagnosis. In their final analyses, Sutker et al. note the commitment component of hardiness explained 20% of the variance in PTSD beyond stress severity, ethnicity, education, and rank. Avoidance coping explained an additional 6% of the variance while family cohesion added another 3% of explained variance. Of course, it is an admitted weakness of the study that it is of a retrospective design; it is not certain whether these findings contribute to our understanding of stress-related psychopathology or merely represent symptoms of such pathology. It is not inconsequential to note that PTSD does seem to be associated with apparently permanent biochemical changes (Southwick, Bremner, Krystal, & Charney, 1994). In sum, however, hardiness does seem to have rather profound main effects on both physical and psychological health.

Early studies done by Kobasa indicate the stress buffering effects of hardiness (in the stress-illness relationship) are of greater magnitude than, and independent of, other stress resiliency resources, to include social support (boss support, family support, marital status), exercise, and constitution (Kobasa et al., 1985; Kobasa & Pucetti, 1983; Kobasa, Maddi, & Courington, 1981). Social support, in particular, has been lately postulated to be a primary source of stress resiliency (Cacioppo, 1997; Uchino, Cacioppo, & Kiecolt-Glaser, 1996). Yet, Kobasa and Puccetti (1983) found the effects of social support were differential by source. While boss support appeared constructive, contributing to transformational coping, family support was negatively related to health in low hardy

individuals. For low hardy individuals, family support may well be a refuge which permits avoidance of pressing issues and situations; in effect, the social support and acceptance in such a case may foster regressive coping behaviors (Kobasa & Puccetti, 1983; Maddi & Kobasa, 1984).

In investigating the moderating effects of hardiness on the stress-illness relationship, it must be noted that many studies (as alluded to in the foregoing discussion) have been (perhaps) methodologically flawed in that illness was measured retrospectively by symptoms (physiological and psychological) surveys and not by potentially more reliable and timely methods such as physician diagnosis, physiological change, or real-time recording (physiological monitoring, diary keeping) as illness occurs. Thus, to an extent, the above findings must be considered somewhat suspect. That the health problems survey used by Funk and Houston (1987) in measuring health outcomes over only two months could only account for about 25% of the variance evident in the health log maintained over the same two months is disconcerting. However, Kobasa, Maddi, and Courington (1981) did compare their symptoms surveys with the medical records of 48 executives in their study. They note that agreement between the surveys and medical records averaged 89 percent, though they did not provide information as to whether low and high hardy executives differed on agreement between surveys and records (Maddi & Kobasa, 1984). Nevertheless, it is a concern that without improved procedure and measures providing some methods variance, it may well be the relationship between stress and illness behaviors (not stress and illness) which is examined (Cohen & Williamson, 1991).

In their review, Orr and Westman (1990) uncovered four studies which used objective health/physiological measures. However, one did not sufficiently describe the study and two employed suspect methodologies. The fourth (Okun, Zautra, & Robinson, 1988), and most cited in the hardiness literature, reported a significant correlation between the hardiness control subscale and percentage of circulating T cells in women with rheumatoid arthritis. That this was published is incredible, but the unnoticed confound glaringly illustrates the "disconnect" between personality/social psychologists and our peers in the biological sciences. (It is noteworthy to comment that the varied disciplines are being brought together in the fields of psychoneuroimmunology, psychoneuroendocrinimmunology, and what Cacioppo has dubbed "social neuroscience" (Cacioppo, 1997; K. Quadry, personal communication, May 1996; Norris, 1996)). The confound in the Okun et al. (1988) study is simply this: rheumatoid arthritis is an autoimmune disorder in which connective tissue is acted upon as an antigen by the immune system. Given that T cells execute cell-mediated immunity, it is just as, or perhaps more, likely the observed T cell percentages are influenced by the disorder itself and not locus of control (Munck, Guyre, & Holbrooke, 1984; Paul, 1991). It is not possible to draw meaningful conclusions from the study by Okun et al (1988).

There is a paucity of research that has examined physiological measures, tonic or acute, in relation to hardiness. Wiebe (1991) found high hardy men displayed lower HR elevations and physiological arousal (peripheral vasoconstriction) than low hardy men during experimental performance of an evaluative threat task (a frustration tolerance task involving a proportion of unsolvable puzzles). Such a pattern did not exist for women.

Wiebe's research suggests the composite hardiness measure explained the effect; for Lawler and Schmied (1992), however, only the control dimension was associated with reduced physiological arousal. Wiebe's (1991) results are consistent with those of Contrada (1989), who found hardiness (interacting with Type B personality) to be associated with reduced DBP reactivity while performing a mirror-tracing task; as previously mentioned, it appears that the DBP reactivity was differentiated by the challenge subcomponent of hardiness. Allred and Smith (1989) found that hardy subjects demonstrated lower arousal when waiting for the experimental task to begin (analogies and mental rotation exercises), although this was significantly confounded with neuroticism measures. Apparently contrary to Wiebe's (1991) and Contrada's (1989) results, however, Allred and Smith (1989) found that high hardy individuals had higher cardiovascular reactivity (SBP) during the task, possibly because of their active coping efforts. Such an increase in cardiac output has been associated with active (versus passive) coping in other research (Lovallo, Wilson, Pincomb, Edwards, Tompkins, & Brackett, 1985); noticeably, such increase in cardiac output during active coping is not concomitant with increased peripheral resistance (Lovallo et al., 1985). Thus, while the findings of Allred and Smith (1989) appear to run counter to the work of Wiebe and Contrada, deeper examination reveals the outcomes are potentially compatible. The precise composition of cardiovascular response (sympathetic versus parasympathetic, promoting cardiac output while suppressing peripheral vasoconstriction) is not known for the above findings and presents a challenge to science; it is also worth noting that the little research that has been done may not be sufficient to permit generalization of the character

of cardiovascular response in hardy versus low hardy individuals. In one sense, however, the above efforts indirectly provide some support for postulated relationships between hardiness, stress, and health in regards to psychophysiological mediators.

More recent studies may provide additional insight into the relationship between hardiness and physiological processes. Solcova and Sykora (1995) found that while awaiting dental surgery and presumably experiencing anticipatory stress, hardy individuals demonstrated lower basal HR and negative HR reactivity (less hardy individuals displayed increased HR) during an arithmetic task. Additionally, hardy individuals showed an elevated pain perception threshhold to a heat stimulus applied to their hands. This is interesting when one considers Cacioppo et al's (in press) on-going work which associates sympathetic (HR) reactivity to HPA axis behavior; while HR reactivity is not in the positive direction for hardy individuals in Solcova and Sykora's study, there is increased threshhold for pain. If this threshhold increase is not a purely cognitive phenomenon, it is reasonable to think there may be endogenous opiate mediators. As we have seen, β-endorphin is one product of POMC cleavage, as is ACTH; there appears to be the potential for HPA axis behavior in hardy people which is counter to that which might reasonably be predicted (the expectation is that HPA reactivity in hardy subjects should be less than that in low hardy subjects; the relevance of HPA activity to illness will be elaborated upon later). Surprisingly, unanticipated HPA behavior is what Zorilla, De Rubeis, & Redei (1995) found. Despite the expectation that hardiness (and both self-esteem and affective stability) would be associated with decreased cortisol secretion, the opposite was revealed. That is, hardy people had higher *basal* cortisol secretion. While

some of this may have been caused by methodology (Zorilla et al drew blood, and venipucture can be a stressful experience for some, thereby leading to HPA activation (Kirschbaum & Hellhammer, 1994)), the results are still surprising. Zorilla et al suggest that, perhaps, HPA reactivity (elevation of cortisol secretion) for hardy individuals in the presence of an acute stressor is less than that for less hardy individuals; this postulation has not been empirically investigated—the second study in this effort will attempt to address this issue. Such a finding (lesser cortisol reactivity in hardy individuals) would fit well with Cacioppo's (1997; Cacioppo et al, in press) reactivity hypothesis, lending it an informative multidimensionality. Surely, the little research that has examined the relationship between hardiness and physiological parameters has raised interesting questions and suggests that hardiness may propagate its health-preserving effects via physiological mechanisms which are less than clearly understood at present. Hardiness research is in need of pursuing such investigations into physiological mediation given unheeded appeals to conduct such research since 1979 (Kobasa, 1979a, 1979b; Wood, 1987).

The construct of hardiness has come under criticism for potentially being confounded with various measures of negative affect, such as depression, the construct of negative affectivity, neuroticism, and maladjustment (Allred & Smith, 1989; Funk & Houston, 1987; Hull et al., 1987; Ouellette, 1993; Williams et al., 1992). However, as Ouellette points out, this is not an issue which can simply be resolved empirically. Indeed, most of the criticism is proffered by researchers who have made a causal inference that negative affect precedes and, perhaps, determines one's hardiness. The opposite may well

be the case and is consistent with existential underpinnings, findings in many of the prospective designs discussed above, Lazarus's contention that cognition precedes affect (Lazarus, 1984), and a body of research suggesting stressful/negative life events (or events so appraised and/or moderated by stress resiliency resources such as hardiness) often precede depression (Kessler, 1997). As Chrousos and Gold (1992) state, "...the cardinal manifestations of melancholic depression are the hyperarousal and redirection of energy that are extremes of the classic manifestation of the stress response" (p. 1247). Perkins, Leserman, Gilmore, Petitto, and Evans (1991) also note a role for stress in the causation of depression, suggesting that depression may physiologically be the result of a generalized stress response which has escaped various neurological and biochemical homeostatic restraints.

One psychometric aspect which may well contribute to a response bias in hardiness research is the negative wording which predominates in many of the earlier instruments. In Kobasa's 20-, 36-, and 71-item instruments, all items are negatively keyed; in the 50-item PVS and Bartone's 45-item DRS scale, negatively keyed items comprise 78 and 67 percent of the instruments, respectively (Funk, 1992). Review of Nowack's 30-item instrument reveals that only 15 or 16 items can be considered to be negatively worded/presented, a potentially important consideration in instrument selection. It may well be that subjects in states of negative affect may "acquiesce" in response to negatively worded items.

It is, further, unreasonable to expect that there would be no overlap between various measures of affect and hardiness given the buffering effects hypothetically

attributed to hardiness (Ouellette, 1993). Indeed, measures of negative affect are often synonymous with measures of psychological strain, the very outcome which hardiness is theoretically posited to, at least in part, ameliorate, and Maddi suggests that theoretical imprecision in various theories of negative affect may well contribute to the controversy (Ouellette, 1993). Lazarus (as quoted in Ouellette, 1993) makes an eloquent case for the parsimony engendered when appraisal and coping styles are thought to precede negative affectivity. Certainly, this dispute is one which will not be easily resolved; it is my conviction, consistent with that expressed by Ouellete and Lazarus, that the resolution of this controversy is ultimately a matter of theoretical grounding. Additionally, the realms of psychology and physiology would do well to better integrate their respective (and complementary) bodies of literature.

It is reasonable to think, given discussion to this point, that hardiness would exert its most profound stress resiliency effects in situations of greater stress (or situations with a greater propensity to induce stress). In a relatively low stress environment, it is likely that resilience resources are of little need. Indeed, the research to date supports this contention (Herlich, 1985; Solano et al., 1993; Wiebe, 1991; Westman, 1990), although the Tang and Hammontree (1992), Neubauer (1992), and Maddi (1987) studies discussed above suggest hardiness (or its behavioral expression) may be influenced by the environment. Nonetheless, the preponderance of the research suggests observable effects of hardiness in moderating relationships between perceived stress and outcomes are more likely to be found in situations and environments characterized by more frequent and more

intense stressors. Low stress environments may well be a factor in the lack of findings by Funk and Houston (1987).

Johnston, Anastasiades, and Wood (1990), in a rather impressive publication, compared cardiovascular response to laboratory stressors and real life (field) stressors/events. Despite the popular belief that the two are sufficiently equal for research purposes, the assumption has been little tested. Johnston et al. found that the assumption may be unwarranted as heart rate and pulse transit time in response to laboratory stressors did not relate in a consistent manner to such measures outside laboratory conditions. Especially notable was the lack of relationship between cardiovascular response to passive coping tasks (for example, the cold pressor test) and cardiovascular responses in the field. Peak cardiovascular responses, but not averaged responses, to laboratory stressors which demand active (problem-focused) coping were most related to and identify "...important and generalized features of the cardiovascular response to stress..." (p. 43). Katkin, Dermit, and Wine (1993) also note "tasks that require active coping have been found to be more potent elicitors of metabolically excessive sympathetic influences on the myocardium than tasks that require passive coping" (p. 152). These results may help explain lack of findings in not only some hardiness research, but also in stress research in general. Indeed, the work of Johnston et al. suggests use of field stimuli when possible and laboratory tasks that profoundly require active coping. Cacioppo (personal communication, 1997), for example, has had success using arithmetic and public speaking tasks. Another factor in the realism of the stressor (and its ability to elicit a response approximating field responses) may well involve the centrality (importance) of the task or

event. Based largely on the accumulated work of Lazarus, Peacock and Wong (1990), in designing their Stress Appraisal Measure, have included centrality as a key aspect of primary appraisal. The potential for harm/loss inherent in threat appraisals and the possibility of meaningful gain/growth in making challenge appraisals reasonably suggests a precondition of centrality. In field situations, centrality can reasonably expected to be present. In effective laboratory tasks requiring active coping, the centrality may be related to performance anxiety, or in the case of public speaking, for example, the threat of embarrassment in the presence of peers who will, undoubtedly, make various attributions about the actor's behavior.

Finally, work stress has been negatively correlated with components of the hardiness construct such as internal locus of control (Cooper, Kirkcaldy, & Brown, 1994; Kirkcaldy, Furnham, & Cooper, 1994; Kirkcaldy, Cooper, Furnham, & Brown, 1993) and a preference for competitive recreational activities (challenge) (Kirkcaldy, Shephard, & Cooper, 1993). Macan, Shahani, Dipboye, and Phillips (1990) reported that students who believed thay had control of their time experienced greater work and life satisfaction, better performance, less role ambiguity, less role overload, and less somatic tension. Ashforth (1997) reported control was negatively associated with feelings of helplessness in the work environment. Meanwhile, Felsten and Wilcox (1992) found that mastery beliefs (approximating an internal locus of control) in students were associated with reduced depression and anxiety. This further suggests hardiness may have much to offer in explaining stress-influenced outcomes. Before progressing further, however, it will first be necessary to examine what is known of the relationships between performance/illness and

stress. If stress is not causally related to performance and illness outcomes, then the proposed moderating effects of hardiness are of little importance to this investigation.

Stress and Performance

It has long been thought that stress impacts performance and other outcomes, although there is much debate about the characteristics of that relationship. Since roughly the mid-1960's and the work of such prominent researchers as Scott, Monat, and Lazarus, most researchers have adopted the view that stress is related to performance nonlinearly in a fashion symbolized by the inverted U; that is, some lower levels of stress are thought to enhance and motivate performance while stress beyond some optimal level serves only to degrade and deteriorate performance (Abramis, 1994; Ellison & Genz, 1983). Abramis (1994) also comments that a minority of researchers have adhered to two other possible relationships: the "motivator theory," in which stressors challenge and motivate performance, and "interference theory," in which stressors uniformly disrupt job performance and form a negative, monotonic relationship with performance.

To enhance clarification in further discussing this issue, Abramis notes stressors cause strain, which he defined as "any deviation from healthy psychological or physiological states of a person, as defined by current psychological and medical knowledge" (p. 548). Sapolsky's (1992a) definition of stress, given earlier, easily supplements Abramis's definition of a job stressor as "characteristics of the job environment which make demands on (tax or exceed) the abilities or resources of people for meeting the demand or which may otherwise threaten attainment of people's needs" (p. 348). Note, also, the similarities to Lazarus and Folkman's (1984) primary and secondary appraisal processes. For Sapolsky (1992a, 1992b), the stress response need not take on the negative flavor of Abramis's strain, being merely "the body's adaptation

designed to reestablish the balance" (p. 288); indeed, no one would argue against the value of the energizing stress response against a physical stressor threatening organismic survival.

In Abramis's (1994) review of the literature, he notes that numerous studies positing the inverted-U relationship noticeably have failed to examine both traditional work-role stressors and actual job performance. Indeed, many studies can largely be characterized by introduction of short-term, simplistic stressors in laboratory settings where performance is measured via outcomes on some brief and facile task. Similar to Johnston et al's (1990) concern that laboratory tasks did not engender the physiological response experienced in actual situations, Abramis convincingly argues the same design flaw exists in the examination of work-relevant outcomes such as performance. Studies reviewed by Abramis using work-role stressors and job performance appraisals suggested to him the stress-performance relationship may be better characterized by interference theory. In his own study, Abramis (1994) found both the technical and social job performance of a wide variety of employees was significantly negatively correlated with both stressors (role ambiguity, person-role conflict, sender-role conflict, and job insecurity) and strains (job dissatisfaction, anxiety, depression, and anger). Performance relationships with both stressors and strains were monotonic, and in no instance did a nonlinear modelling explain greater than an additional 4.5% of the variance. His results suggest that when actual performance is the consideration, the optimal levels of these stressors is zero.

In a study examining the consequences of occupational subjective stress for job performance in nurses, Motowidlo, Packard, and Manning (1986) had findings consistent with Abramis's assertions. Subjective stress was positively correlated with degradation of interpersonal and cognitive/motivational aspects of job performance. In particular, stress degraded composure, quality of patient care, interpersonal effectiveness, warmth toward other nurses, tolerance with nurses and doctors, and cognitive effectiveness, with the relationship being mediated by depression. Felsten and Wilcox (1992) found stress to be associated with decreased grade point average (GPA) in college students. Similarly, Lloyd, Alexander, Rice, and Greenfield (1980) determined that life events were negatively related to GPA in the first two years of college. Bhagat, McQuaid, Lindholm, and Segovis (1985) also found strong negative relationships between stress and performance-related organizational outcomes. In a recent study, police trainees were evaluated for eyewitness memory in realistic role playing scenarios (Yuille, Davies, Gibling, Marxsen, & Porter, 1994). Those under stress recalled less information one and twelve weeks later (although their accuracy and informational resistance to decay was greater, probably due to a cognitive phenomenon known as "remarkable memories"). Other studies have also found decrements in performance among police officers (Perrier & Toner, 1984), military parachutists (Sharma, Sridhavan, Selvamurthy, Mukherjee, et al., 1994), and Israeli Defense Force officer cadets (Westman, 1990) in relation to stress.

The relationship between stress and performance, however, may not be so simplistic as the preceding discussion might suggest. The nature of the task may also be vitally important. Easterbrook (1959) suggests that, as arousal increases, and as the

demand upon cognitive resources increases, there is a progressive narrowing of the cues and stimulus details attended to and encoded. In other words, stress may result in the commission of valuable cognitive resources to the task at hand, leaving little available to attend to other issues/stimuli. This implies that the less automatic the task (and the more it requires application of conscious cognitive resources), the more likely it is that the task may be degraded by stress. Perhaps the more realistic tasks and outcomes to which Abramis (1994) directs his attention are subject to degradation matching interference theory patterns because they are more cognitively complex. Previously discussed findings in which stress was negatively related to academic performance may also reflect the cognitive complexity of academic achievement. Indeed, Klein and Barnes (1994) found that life stress and anxiety were negatively related to performance only in the attempted resolution of relatively more complex word problems (analogies). Relatedly, Wickens, Stokes, Barnett, and Hyman (1993) found that stress increasingly decreased performance as spatial and knowledge demands increased in an instrument flight rules (IFR) flight simulation.

In conjunction with the dynamics of cognitive resource allocation, it is likely that various physiological substrates also influence cognitive performance in stressful situations. Cortisol reactivity appears to be dramatically related to hippocampal functioning, and, accordingly, outcomes dependent upon working memory, spatial working memory, and declarative/explicit recall (this will be discussed in some depth in a later section). Central adrenergic/noradrenergic pathways involving the limbic system may enhance recall of survival enhancing details, however; this dual pathway approach to

cognitive function in stressful situations may well explain, for example, the 1994 findings of Yuille et al. mentioned above (for a fuller discussion, the author can provide a 1996 unpublished manuscript dealing in depth with the dual pathway postulation). Counter to the traditional inverted-U hypothesis about stress-performance relationships, there may be some cases in which stress and performance are related in an upright-U pattern. Using biogenic amine urinary excretion as a stress response marker, Hubalik, Krahenbuhl, Harris, and Stearn (1992) examined golfers at the collegiate level in the conditions of play (golfing for pleasure), tournament qualification, and competition. The majority of the golfers demonstrated their best performances in the lowest stress (play) condition, their worst performances in the moderate stress (qualifying) condition, and generally intermediate performances (with rather large variance in scores) in the high stress (competition) condition.

To further complicate matters, the conclusions of Orasanu and Backer (1996) in their examination of stress and military performance note that while given stressors may generally lead to performance decrements (interference theory), stressors differ uniquely in their effects, individuals differ uniquely in their vulnerability to stress (hardiness may well be one of those individual discriminators), and various tasks are differentially affected by various stressors. The relationship between stress and performance is highly complex and does not appear to lend itself to simplistic representations. The task, individual, familiarity with the task (degree of automaticity), and environmental complexity are among the factors which must be considered in examining the stress-performance relationship. In general, it appears stress can adversely impact performance on tasks of greater

complexity while various individual differences (such as hardiness or task-specific experience) may buffer the individual against such negative outcomes. In the interest of promoting other directions in research, it is worth emphasizing the sanctity of the inverted-U relationship between stress and performance, at least concerning such relationships in the workplace, is questionable.

Immune System Overview

To advance the discussion to consideration of the stress-illness relationship, it is necessary to overview some of the key elements of the immune system. The body's defense mechanisms can be divided into innate (nonspecific) and specific immunity components (Maier, Watkins, & Fleshner, 1994). Innate components provide pathogen resistance and are composed of such nonspecific elements as the skin, mucous membranes, and phagocytic microorganisms such as macrophages. Specific immunity, on the other hand, is acquired and involves two major processes, recognition of antigens followed by destruction/removal of antigens (Maier et al., 1994). Specific immunity may be characterized as either humoral or cellular.

Humoral immunity is effected by B lymphocytes (B cells) and their production of antibodies (proteins, also called immunoglobulins, which can recognize specific antigens in extracellular fluids and on cellular surfaces) (Paul, 1991). Immunoglobulins consist of two polypeptide chains (a heterodimer), one designated as the "heavy" chain, the other as the "light" chain. Variable and hypervariable regions of the heterodimer combined with well-defined constant regions allow for a tremendous amount of random variability in light/heavy chain pairing (Paul, 1991; Leder, 1991). It is this variability that makes immmunoglobulins so adaptive in the the immune response. The heavy chain type (there are five) defines immunoglobulin (Ig) class (IgA, IgD, IgE, IgG, IgM) and function; for example, IgG is likely to circulate in the blood while IgA associates with various surface-lining cells such as those in the nasal passageways (Leder, 1991). B lymphocytes initially act as antigen receptors, using a specialized membrane-spanning Ig. When a specific

antigen stimulates the B cell, it becomes an antibody secreting cell; the soluble antibody secreted is identical to the membrane-spanning Ig mentioned above, but without the membrane-spanning characteristics and properties (Paul, 1991). Once the immune response is underway, B lymphocytes also begin an incredible process of hypermutation, apparently in the effort to produce antibodies with even greater antigen binding affinity than the original antibody; of course, a higher affinity antibody, if created, is selected for and comes to dominate the assault on the antigen (Paul, 1991). The antibodies produced by the B cells bind to the antigen, neutralizing it or marking it for destruction by phagocytic cells (i.e. macrophages) or plasma proteins known as complement (Sapolsky, 1992a; Kuby, 1991). When the antigen threat ceases, B cells specific to that antigen remain to act as antigen detectors, composing what is called immunological memory and facilitating a more rapid response to that antigen if encountered in the future. Antibodies, again, are limited in that they combat antigens in extracellular fluid and on cellular surfaces. T lymphocytes (T cells), however, have the ability to attack cell-associated antigens such as viruses, bacterial products, and other intracellular pathogens.

Cellular immunity is effected by T cells. T lymphocytes have specific antigen receptors similar to the B cell immunoglobulins mentioned above. Instead of light and heavy chains, they possess what are known as alpha and beta (and rarely gamma or delta) heterodimeric chains with similar combinational diversity; in contrast to B cells, T cells cannot hypermutate (Paul, 1991). To detect intracellular antigen, T cells need some help from antigen presenting cells (APCs) such as macrophages, B cells, or dendritic cells (Kuby, 1991). A recognizable peptide component of the antigen is bound with

specialized surface proteins of the APC known as class I or II major histocompatibility complex (MHC) molecules. The MHC-peptide pairing is brought to the APC cell surface for T cell recognition and activation (Paul, 1991). Certain T cell subtypes tend to bond with either class I or class II MHC molecules. Once activated, T cells may act in a variety of ways including helping B cells make antibodies specific to the presented antigen, enabling macrophages to destroy the antigen, or killing the whole virus-infected cell (Paul, 1991). In their regulatory capacities, T cells may act as helpers or suppressors (helper and suppressor/cytotoxic T cells) of the immune response; more actively, T cells can act cytotoxically. T helper and T suppressor/cytotoxic cells usually can be differentiated by membrane glycoproteins, these being CD4+ and CD8+, respectively; T helper (CD4+) cells most often recognize antigen associated with MHC II molecules while T cytotoxic (CD8+) cells predominantly recognize antigen with MHC I molecules (Kuby, 1991). When T helper cells are activated, they often release a key cytokine (cytokines are comprised of interferons—IFN, TNFs, interleukins—IL, colony-stimulating factors—CSF, and other factors which collectively regulate immune system functioning and communication), IL-2, which promotes lymphoproliferation of B cells, T cells, and stimulates nonspecific natural killer (NK) cell activity (Smith, 1991; Old, 1991). The NK cell, an especially virulent weapon in the immune system arsenal, is thought to be closely related to the T cell (especially the cytotoxic T cell); it destroys undesirable cells through release of a protein called perforin, which literally forms pores in the target's surface membrane (Young & Cohn, 1991). IL-1 is thought to mediate initial T cell activation as well (Maier et al., 1994). As with B cells, many T cells will remain as immunological

memory to counter future threats by the same antigen (Maier et al., 1994). The complex communication by cytokines, immunological memory, and the sensory/motor functions of the immune system in dealing with antigens have led some to consider the immune system as something of a "mobile" or "parallel" brain, a model with some merit. In fact, IL-1, IL-2, TNF, and IL-6 have been shown to act on the CNS, apparently entering the CNS in the area of the preoptic nucleus of the HTH where the blood-brain barrier is somewhat lacking (Shepherd, 1994; Black, 1994b; Betancur et al, 1995). Besedovsky and Rey (1987) found, for example, that IL-1 can centrally stimulate ACTH/corticosterone release and insulin output in rats.

Both B cells and T cells originate in bone marrow, immature T cells migrating to the thymus (thus their name), where they mature and differentiate. At maturity, both B and T cells migrate throughout the body via circulatory and lymph systems (Maier et al., 1994). Mature B cells, T cells, and other immune cells have also been found to have receptors for a wide variety of hormones, GCs, endogenous opiates, and catecholamines (Shepherd, 1994; Black, 1994a; Sheridan et al., 1994). Norris (1996) asserts receptors for "all known hormones, neuromodulators, neuropeptides, and neurotransmitters" have been found on lymphocytes. As will be seen, these characteristics of immune system function will assist in explaining how the stress response may effect immunosuppression, and, in turn, affect health. Lastly, it is beyond the scope and purpose of this effort to more fully address the highly complex dynamics of immune function. If further investigation is desired by the reader, Kuby (1991) and the volume edited by Paul (1991) are highly recommended.

Relationships Between Stress, the CNS, and Illness

If the psychophysiological effects of the stress response mediated by various CNS structures (and subsequently, neuroendocrinological and SNS pathways) are immunosuppressive, lesioning in these CNS structures should produce changes in immune functioning. While there are some mixed results, perhaps due to the imprecision of some techniques, lesioning of stress-involved CNS structures does appear to impact immune functioning. In a review of 31 experiments, Jankovic (1989) found lesioning, particularly in the HTH, produced pronounced effects on immune functioning. Lesions in the posterior, medial, and anterior HTH were found to suppress antibody production, reduce lymphocyte populations, and induce thymal involution. Rószman, Jackson, Cross, Titus, Markesberry, and Brooks (1985) found bilateral electrolytic lesioning of the anterior HTH in rats decreased lymphocyte populations in the spleen and thymus, splenic mitogen responsiveness, systemic antigen responsiveness, and NK cell activity, while macrophage suppression was increased. Conversely, lesioning of the hippocampus or amygdala increased splenic and thymocyte mitogen responsiveness and thymal lymphocyte populations (hippocampal lesion), while decreasing macrophage suppression activity. The amygdalar response to lesioning is consistent with the work of Liang et al. (1992), and their previously mentioned finding that bilateral lesions of the central nucleus of the amygdala blocked the excitatory effects of icv infusion of CRF on the acoustic startle reflex; normally, again, CRF produces a dose-dependent amplification of reflex behavior. Thus, amygdaloid lesions dampen the stress response while simultaneously enhancing some aspects of immune functioning. It is also interesting to note that effects of HTH

lesioning can be often prevented by hypophysectomy (removal of the pituitary gland), indicating the degree to which immune system functions may be under CNS directed, pituitary-mediated neuroendocrine control (Black, 1994a). In consideration of the above, it appears a link between stress, CNS functioning, and illness does exist.

The extent of CNS control over immune functioning is revealed in a number of studies in which immunologically impacting stress responses and/or immune function have been conditioned. Camphor or a saccharin-lithium chloride mixture paired with polyinosinic : polycytidylic acid, which stimulates NK cell activity, for as little as nine trials, became conditioned stimuli which could thereafter effectively enhance NK cell activity to include NK cell activity directed against active tumors (Ghanta, Hiramoto, Solvason, Tyring, Spector, & Hiramoto, 1987; Ghanta, Hiramoto, Solvason, & Spector, 1985). The implication of such findings in treating cancer, for example, are self-evident. In contrast, conditioned immunosuppression where a conditioned stimulus is paired with a powerful, but highly toxic, immunosuppressant agent like cyclophosphamide, holds promise in helping tissue transplant recipients lessen systemic rejection of the new tissue (Maier et al., 1994). Yokoo, Tanaka, Yoshida, Tsuda, Tanaka, and Mizoguchi (1990) were able to condition fear-elicited NE release in the rat HTH; similarly, Lysle, Luecken, and Maslonek (1992) have conditioned endogenous opioid mediated suppression of mitogen induced proliferation in rat splenocytes.

Relevantly, Murison and Overmier (1993) found unconscious rats subjected to cold water immersion did not develop gastric ulcers while conscious rats did. This strongly suggests the perception of stress, not the impact of the stressor itself, may be key

to stress-induced pathology; the importance of this observation to the present investigation is self-evident. Consistent with this reasoning, in combative encounters between mice, submissive mice manifested reduced T cell proliferation and suppressed IL-2 production whereas the dominant mice from the encounters displayed elevated T cell proliferation and IL-2 production (Hardy, Quay, Livnat, & Ader, 1990). Miczek, Thompson, and Tornatzky (1990) found differences in SNS stress response between dominant and submissive mice, and dominant mice displayed faster return of GCs to baseline levels following stressor exposure than did the submissive mice. In non-human primates, French (1997) notes that low social status in a colony is associated with increased cortisol secretion and decreased gonadal steroids; lack of control in a social environment is suggested in such a display of chronic physiological stress response. Sapolsky (1992b) notes also that baboons and other primates that are socially subordinate manifest elevated basal cortisol levels, an accompanying sluggish HPA secretory response to stressors, and enlarged adrenal glands. The HPA disregulation in social subordinates appears to be hypothalamically driven. Sapolsky (1992b) asserts that this chronic disregulation of HPA function may result from altered feedback sensitivity in the axis as a result of initial exposure to frequent stressors (an habituation response). Importantly, the presence of elevated GC concentrations in subordinate primates is associated with atherosclerotic occlusion, lower HDL cholesterol concentrations, and fewer circulating lymphocytes (Sapolsky, 1992b). Those dominant primates with the lowest basal cortisol levels were further characterized as those males who were "best at differentiating between threatening and neutral interactions with social rivals" (Sapolsky, 1992b, p. 278), those

who intiated interactions, those who seemed to have outlets through which to displace aggression, and those who engaged in the greatest degree of nonsexual interaction with females and their infants. In sum, Sapolsky (1992b) notes that

> "Collectively, low cortisol concentrations were associated with high degrees of social skillfulness and predictability (knowing that a sleeping rival is different from a threatening one, knowing which fights to pick), social control, an ability to differentiate between positive and negative reinforcers, having outlets for frustrations, and having a social support network...what seemed to be an endocrine marker of dominance also reflects personality style" (pp. 279-280).

The parallels to hardiness and other stress resiliency resources in the above passage is quite remarkable. If hardiness moderates the stress-illness relationship via neuroendocrine mechanisms, then one might well expect to see HPA behavior in high hardy subjects similar to the above trends for dominant members of mammalian social groups. Of course, the previously discussed findings of Zorilla et al (1995) are troublesome, and, should they not be due to artifact or type I error, may indicate greater complexity in human psychoneuroendocrine function than previously thought. The link between stress and illness does indeed appear to be mediated by CNS control of stress response mechanisms and, prominently, the cognitive temperance (or exacerbation) of that response.

If stress does indeed engender immunosuppression, we would expect to find suppression of key immune system elements and greater incidence of illness in the presence of actual or perceived stress. This is precisely what is found, although some studies have been appropriately criticized for poor control and methodological flaws (Cohen & Williamson, 1991). Jankovic (1989), in a review of 35 studies involving animals, found eight commonly applied stressors increased infection and tumor growth,

and suppressed NK cell activity, mitogenic responsiveness, and serum Ig levels. Immobilization stress in mice caused severe thymal and splenic atrophy with an accompanying decrease in both mature suppressor T cells and NK cells, particularly in the thymus (Teshima, Sogawa, Kihara, Hagata, Ago, & Nakagawa, 1987). When mice underwent immunization with sheep erythrocytes and treatment with mitogenic catalysts, those that were stressed by crowding produced less than 25% the IL-2 of the uncrowded mice, a major finding given the lymphoproliferative properties of IL-2 (Rabin, Lyte, Epstein, & Caggiula, 1987). As a sidenote, this might well imply the importance in attending to qualitative differences in social support when it's researched as a stress buffer. Brosschot, Benschop, Godaert, and Olff (1994) found that teachers reporting high numbers of daily "hassles" manifested decreased levels of T cells and NK cells in peripheral blood samples than relatively non-stressed teachers. In another study, mice were injected with mycobacterium avium or mycobacterium tuberculosis and then subjected to restraint stress for varying numbers of periods, each period being 18 hours in duration. The more restraint periods a mouse endured, the more the mycobacteria fluorished in splenic and lung tissue; since mycobacterial resistance is considered to be macrophage mediated, it appears stress suppressed macrophage ability to fight the infection (Sheridan et al., 1994). In mice restrained for several periods (16 hours/period) following exposure to influenza A/PR8 virus, IL-2 levels dropped as restraint periods increased. After eight restraint periods, IL-2 levels were approximately 25% (depending upon the tissue examined) of starting levels (Sheridan et al., 1994). At West Point, 1400 cadets were followed for four years; those cadets under greater stress as a result of high

levels of motivation and poor academic performance were more likely to become seropositive for the Epstein-Barr virus, more likely to develop infectious mononucleosis once they became seropositive, and tended to spend more time hospitalized once they manifested the infection (Black, 1994b). Similarly, medical students undergoing academic examinations demonstrated heightened reactivation of latent Epstein-Barr virus as revealed by virus capsid antigen antibody titers (Glaser, Pearl, Kiecolt-Glaser, & Malarkey, 1994). Stone, Reed, and Neale (1987) found undesirable life events increased and desirable events decreased three to four days prior to onset of infectious symptoms in subjects. Suppression of IgA was associated with various infections, and Stone et al suggest IgA suppression during stressful stimuli enhances infectious opportunities, the three to four day lag corresponding with incubation periods for the infection. Cohen, Tyrell, and Smith (1993) conducted a viral (3 types of rhinovirus, respiratory syncytial virus, and a coronavirus) challenge study with 394 human subjects. Those reporting high perceived stress levels had significantly greater rates of successful infection and clinical symptomology. The study by Cohen et al. (1993) is also noteworthy in their extensive efforts to control and account for other factors which may impact stress levels and likelihood of illness, providing a model for future conduct of viral challenges and stress-illness inquiries. Thus, the proposition that psychological stress is associated with immunosuppression and susceptibility to infectious and other disease appears to be supported. From this point forward, I will examine mechanisms by which this demonstrated illness/immunosuppression may occur.

Glucocorticoid Mediated Mechanisms of Immunosuppression, Illness, and Performance-Related Dysfunction

Stress-induced GC secretion is one of the primary mechanisms by which stress can suppress the immune system. In fact, Sapolsky (1992a) postulates GCs are perhaps the most important mediators of stress-induced immunosuppression, showing generally inhibitory systemic effects. GCs and GC analogs have been demonstrated to inhibit lymphocyte and macrophage activity, suppress phagocytosis, decrease immune tissue production of many cytokines (gamma interferon (IFN-γ), granulocyte macrophage-CSF (GM-CSF), and macrophage inflammatory protein-2 (MIP-2), IL-1,2,3, 5, 6, 8, and TNF), inhibit class II MHC expression, downregulate dendritic cell function, suppress serum Ig levels, suppress inflammatory mechanisms of the immune system, disrupt cytokine receptor function, pull lymphocytes from the bloodstream and destroy them via lysis, decrease lymphocyte cytotoxicity, depress NK cell activity, inhibit serotonergic activity (an interesting effect in that serotonin deficiency has been postulated as mediating aggression; notably, it is hostility which may link Type A personality traits with illness, and, as will be shortly elaborated upon, serotonin is a key neurotransmitter in hippocampal regulation of the HPA axix), and suppress production of dopaminergic growth factors by glial cells (Engele &Lehner, 1995; Moser, DeSmedt, Sornasse, Tielemans, Chentoufi, Muraille, et al., 1995; Williams & Coleman, 1995; Black, 1994a; Maier et al., 1994; Savastano, Tommaselli, Valentino, Scarpitta, D'Amore, Luciano, et al., 1994; Reichlin, 1993; Sapolsky, 1992a, 1992b; Tobler, Meier, Seitz, Dewald, Baggiolini, & Fey, 1992; Munck & Guyre, 1991; Lee, Tsou, Chan, Thomas, Petrie, Eugui, & Allison, 1988; Munck

et al., 1984; Kalat, 1984). Sheridan et al. (1994), in reviewing microbial pathogenesis experiments in animals, found laboratory administration of GCs often promoted immunosuppression and infectious advances resulting in death. Conversely, use of RU-486 to blockade GC receptors prevented macrophage suppression and enhanced post-injury macrophage killing of candidas albicans in mice administered open femoral fractures (Cech, Shou, Gallagher, & Daly, 1994). Mice exposed to 30 minutes of footshock stress demonstrated decreased splenic lymphocyte response to mitogens (concanavalin A and phytohemagglutinin), an effect notably not observed in adrenalectomized mice (Dunn, 1989). Solomon, Amkraut, and Rubin (1985) noted thymal involution occurs during acute stress, and GC induced thymal involution under laboratory conditions is identical to that found in stressed subjects (Sapolsky, 1992a). Differentiating T cells in the thymus are quite susceptible to GC damage, and it has long been known that near physiological concentrations of GCs are sufficient to induce thymocyte apoptosis (Wyllie, 1980). GC-mediated thymal involution may be of further concern because it is now known several thymosins are necessary for lymphocyte development in bone marrow, T-cell maturation and differentiation, and immune system regulatory feedback to the CNS (Hall, McGillis, Spangelo, & Goldstein, 1985). As one would expect, adrenalectomy or suppression of cortisol/corticosterone synthesis with an agent such as metapyrone eliminates much, but not all, of the stress-induced immunosuppression, indicating the presence of other mediators (such as sympathetic activation and endogenous opiate secretion) (Black, 1994a; Ader & Cohen, 1993; Besedovsky, Rey, & Sorkin, 1985; Keller, Weiss, Schleifer, Miller, and Stein, 1983).

While GCs serve us well in mobilizing energy and countering inflammation (Maier et al, 1994; Sapolsky, 1992a), they obviously appear to undermine well-being to the extent they are immunosuppressive. Why should this be so? There is extensive support that innoculation of foreign antigens, infection, and inflammation activate the HPA axis in a way that is similar to the stress response; in the course of immune system activation, IL-1, IL-2, IL-6, and TNF-α have all been shown to stimulate HTH (PVN) release of CRF and VP (Black, 1994b; Maier et al, 1994; Reichlin, 1993; Rey, Besedovsky, Sorkin, & Dinarello, 1987). Additionally, we now know many immune cells can also produce and secrete their own ACTH (Shepherd, 1994). At the height of an immune response, increased electrophysiological activity has been observed in neurons in the PVN and LC (Black, 1994b). The HPA activating effects of IL-1 have, accordingly, been abolished by adrenalectomy (Chover-Gonzalez, Harbuz, & Lightman, 1993). While HPA activation by the stress response is almost immediate, activation by the immune system is much slower; this, along with realization that many immune system toxins and activities can damage healthy tissue (as in autoimmune disorders) led Munck et al (1984) to propose GC mediated immunosuppression prevents the body's immune defenses from "overshooting" and becoming dangerously hyperactive. This theory is today widely endorsed by researchers in the fields of psychoneuroimmunology and neuroendo-crinimmunology (Black, 1994b; Maier et al, 1994; Sapolsky, 1992a; Munck & Guyre, 1991). The overshoot prevention theory is sensible in that a truly temporary stress response would not appreciably suppress systemic immune function (unless such acute responses occurred with some frequency; such reactivity (or repeated reactivity) lies at the heart of

Cacioppo's (1997) stress reactivity hypothesis); however, the potentially pathological consequences of chronic psychosocial stress become quite apparent (Savastano et al., 1994). Dysfunctional HPA activity and suppressed GC levels have, logically, been associated with autoimmune disorders such as chronic fatigue syndrome, experimental allergic encephalomyelitis (EAE--a murine model for multiple sclerosis (MS)) and various arthritic conditions (Purba, Roadsheer, Hofman, Ravid, Polman, Kamphorst, & Swaab, 1995; Black, 1994b; Swain & Maric, 1994). In human MS patients, immunocytochemical analyses determined MS patients had 2.4 times the CRH containing cells in the PVN than controls; this is suggestive of the body's effort to respond to MS and has also been correlated with the high prevalence of depression in MS patients (Purba et al., 1995). (It is worth noting that depressed patients are less responsive in dexamethasone suppression tests (Perkins et al., 1991), and recently it has been found that in depressed patients, GC receptor function is markedly decreased (Modell, Yassouridis, Huber, & Holsboer, 1997)). It is possible that MS patients suffer from GC insensitivity or decreased GC receptor function, allowing a hyperactive immune response during infection and lack of inhibitory feedback by endogenous glucocorticoids (Reder, Makowiec, & Lowy, 1994).

While it has long been known that GCs are immunosuppressive, being one of the two most effective chemotherapeutic treatments (the other being cyclosporine) when immunosuppression is desired (Smith, 1991), we are just beginning to understand the way in which they effect immunosuppression. In thymocyte apoptosis, Wyllie (1980) noticed one of the first structural changes to take place was widespread chromatin condensation, and he noted the chain of destructive events that followed seemed to be "programmed."

His observations led him to theorize GCs activated some type of endogenous endonuclease, an enzyme that fragments the cell's DNA. It is now believed GCs activate endonuclease synthesis to destroy lymphocytes, the genetic alteration literally causing them to burst; this lysis, consistent with Willie's (1980) observations, is often called programmed cell death (PCD) (Vito, Lacana, & D'Adamio, 1996; Sapolsky, 1992a). Genetic mechanisms similar to the above have been revealed in GC mediated IL-1 and IL-2 suppression, although IL-2 (along with protein-kinase C inhibitors) has also been reported to inhibit apoptosis of immature T cells (Savastano et al., 1994), perhaps via intermediate affinity IL-2 receptors (Rebollo, Pitton, Garcia, Gomez, & Silva, 1995).

IL-1, as previously noted, is a key mediator of the immune response (produced primarily by macrophages and other monocytes), apparently inducing subsequent IL-2 production and having lymphoproliferative impact on helper and suppressor/cytotoxic T cells; additionally, it has been implicated in hepatic, pancreatic, and other functions during antigenic challenge (Rey et al., 1987). Lee et al. (1988) subjected monocytes to antigenic challenge, and, using in situ hybridization and related analytical techniques, discovered GCs inhibited transcription of the IL-1 β gene in the monocytes. Incredibly, GCs also acted to destabilize IL-1 β mRNA already in existence, inducing a rather rapid decay (IL-1 β mRNA levels dropped more than 50% in three hours)! Furthermore, the IL-1 β mRNA destabilization was highly selective; GCs did not destabilize other types of mRNA with similar nucleotide sequencing. The research of Lew, Oppenheim, and Matsushima (1988) supports this mechanism of GC mediated IL-1 suppression, finding further that

production of IL-1 α appears to be suppressed in an identical fashion. In 1992, Northrop, Crabtree, and Mattila investigated GC suppression of IL-2 production. When GC receptors are activated (occupied), they appear to interact directly with transcription factors, proteins vital to successful transcription of the IL-2 gene. A series of interactions between the receptor complex and a particular transcription factor (an as yet unidentified octamer-associated protein) were demonstrated. It is likely other transcription factors are involved as well. The GC receptor-transcription factor interaction appears to be responsible for IL-2 gene transcription inhibition. Northrop et al. (1992) note other GC mechanisms may well be involved with IL-2 suppression, postulating GCs may destabilize IL-2 mRNA (as with IL-1) and the IL-2 protein itself. These GC effects are consistent with a general model of GC-receptor mediated genomic mechanisms proposed by Munck and Guyre (1991). Changes in the transcription process, of course, result in altered mRNA levels and the proteins (such as IL-1 or IL-2) they encode.

Recent research has revealed expression of a certain protein, Bcl-2, protects murine splenic T cells (Broome, Dargan, Bessent, Krajewski, & Reed, 1995), ileal Peyer's patch B cells (Motyka & Reynolds, 1995), and a T cell hybridoma (Memon, Moreno, Petrak, & Zacharchuk, 1995) from GC induced apoptosis, but not other apoptotic pathways (Fas or activation-induced). Broome et al's (1995) findings are consistent with those of Rebollo et al. (1995) in that Bcl-2 expression in T cells appeared to be IL-2 dependent, perhaps providing a mechanism by which IL-2 receptor-mediated rescue from GC induced apoptosis is effected. Bcl-2's protective effects have, notably, been observed both in vitro and in vivo (Motyka & Reynolds, 1995). Memon et al (1995) report TNF or

Fas induced cell death can be suppressed by cowpox virus gene product CrmA, which is also known to be a potent inhibitor of IL-1 β-converting enzyme (ICE) which, in turn, shares homology with an effector gene product required for apoptosis called CED-3. It is interesting that Bcl-2 is homologous to a similar effector gene product, CED-9. Memon et al (1995) speculate GC induced apoptosis in T cell hybridomas utilizes a "Bcl-2 sensitive CrmA-resistant ICE-like protease path" (p. 4651), although a great deal of research is likely needed to fully comprehend differences in apoptotic pathways.

Finally, to somewhat round out the picture, Vito et al (1996) have identified the ALG-2 gene as being prominent in apoptotic pathways. The ALG-2 gene codes for a Ca^{2+}-binding protein prominent in T cell receptor, Fas, and GC induced cell death. Reducing the ALG-2 protein in a T cell hybridoma was, expectedly, associated with reduced cell death. The findings are informative in that thymocytes undergoing GC induced cell death show sustained intracellular calcium increase prior to apoptosis, and apoptosis can be prevented by blocking calcium increase. Vito et al (1996) suggest ALG-2 may represent the prototype of a gene family mediating calcium regulated cell death. Perhaps the ALG-2 pathway is itself dependent upon ICE-CED-9 or other enzymatic activity. Certainly, there is much yet to learn about the mechanisms by which GCs so robustly induce death in immune cells.

It is not only the cells of immunological function, however, that appear to be endangered by GCs. Returning to a topic mentioned earlier in this paper, GCs have been implicated in hippocampal degeneration, an ominous eventuality given well-known hippocampal involvement (presumably via long-term potentiation (LTP)) in learning and

memory (Carlson, 1994). GC involvement in hippocampal damage is postulated to occur both during stress (Uno et al., 1989) and aging (Savastano et al, 1994). It is known that the hippocampus is dense with both high affinity type I (GC sensitive mineralcorticoid) and low affinity (six to tenfold lower affinity) type II (glucocorticoid) GC receptors; type II receptors are known to be occupied during both stress and peaks in the diurnal GC rhythm (DeKloet et al., 1994), and, it is type II receptors which predominate in lymphoid tissue (thymus, spleen, and lymph nodes) shown to be stress-sensitive (Kusnecov & Rabin, 1994). IL-1 stimulation of the HPA axis has been shown to cause shifts in the type I/type II receptor balance, perhaps driving the long-term HPA activity witnessed during the immune response (DeKloet et al., 1994); such IL-1 induced activity appears to impair limbic control over HPA activity. It is thought, for example, that GCs exert control over serotonergic (inhibitory) and noradrenergic (excitatory) activity in hippocampal neurons; type I receptors are thought to mediate excitation (block 5-HT activity) while type II receptors may mediate inhibition (block NE transmissions) of hippocampal inhibition of PVN activity (Meijer & DeKloet, 1995; DeKloet et al., 1994). Further, it is the occupation of type II receptors during increased HPA activity which has been associated with decreased LTP and synaptic inhibition of pyramidal neurons in the CA1 field of the hippocampus (DeKloet et al., 1994). In the same vein, Pavlides et al. (1995) found RU 28362, a type II receptor agonist, substantially suppressed LTP induction in the hippocampus. It is probably no coincidence Yehuda et al. (1995) found cognitive deficits in combat veterans diagnosed with post traumatic stress disorder (PTSD) that are consistent with deficits observed in patients with known hippocampal damage. It would

be a worthwhile research effort to examine hippocampal tissue in these veterans or others diagnosed with PTSD upon their deaths; neural damage in these veterans, though perhaps less extreme, might be similar to that found by Uno et al. (1988) in vervet monkeys. The relationship between PTSD and GC biochemistry is just emerging and appears to be quite complex. While PTSD diagnosed individuals appear to have diminished basal cortisol secretion (Chrousos & Gold, 1992; Southwick et al., 1994), there is evidence, unlike depression, that they may have increased GC receptor sensitivity at hypothalamic and pituitary levels with simultaneously blunted response to CRF (Southwick et al., 1994).

An emerging body of research over the last 5 years suggests that GC biochemistry exerts very powerful effects, presumably via hippocampal receptors, on such cognitive functions as working memory, memory consolidation, declarative recall, and spatial orientation, learning, and processing. If true, such effects could be expected to drastically affect the performance of those in cognitively demanding occupations, or those in occupations where the application of working memory is vital and spatial demands are high (pilots, for example).

Lupien, Lecours, Lussier, Schwartz, Nair, and Meaney (1994) examined cognitive function in 19 healthy elderly subjects who had shown relatively stable differences/patterns in cortisol secretion over the preceding four years. Among the tests of cognitive function were one testing explicit memory and one testing selective attention. In the explicit memory evaluation, subjects had to recall a word from a paired word list when given one of the pair as a cue. In the selective attention task, subjects had to conduct a visual search on a computer screen for a target specified by a combination of color and shape features.

It is worth noting, for future reference, the similarity between this selective attention task and scanning a cockpit instrument panel for relevant information (considered a spatial task (flexibility of closure) in the Wickens et al. (1993) IFR simulator study). Lupein et al. found that cortisol slope (representing change in basal cortisol secretion over the preceding year) was negatively associated with performance in the explicit memory and selective attention tasks. Deficits in the selective attention task were more pronounced in comparison to controls as the number of items in the visual search increased; the effect was significant when the targets were absent, implying, further, deficits in spatial working memory. In sum, the Lupien et al. (1994) study suggests chronically increasing cortisol elevation is perhaps causally associated with some declining cognitive function. Relatedly, Newcomer, Craft, Hershey, Askins, and Bardgett (1994) administered daily doses of dexamethasone to 10 subjects over the course of 4 days and administered a series of cognitive evaluations on days 0 (baseline), 1, 4, and 11 (7 days post-treatment). Compared to controls, the experimental group showed significant deficits in a paragraph recall (a test of declarative memory performance) evaluation on days 4 and 11. Again, elevated GCs appear to be dramatically associated with cognitive dysfunction.

Diamond, Ingersoll, Fleshner, and Rose (1996) subjected rats to an unfamiliar environment for 4 hours in the midst of a food-foraging task in a 14-arm radial maze. Prior to the delay, the rats had been allowed to locate and eat food placed in 4 of the 7 arms. Post-delay, they were returned to the maze to find and eat the food in the other 3 arms. Placement in the unfamiliar environment (but not a familiar one) during the delay was associated with increased errors in locating the remaining food post-delay. Over the

course of 8 days, the stressor effect disappeared. On day 9, the experimental rats were exposed to a novel stressor (placement in water during the delay), and the increased error rate was again seen post-delay. The rats apparently habituated to this stressor as well over the ensuing 7 days. Thus, it appears that psychological stress can adversely impact working memory, interfering with retrieval of previously stored information. In fact, the demonstrated retrograde amnesia was nearly complete as 98% of the errors consisted of entry into each of the 7 arms in the baited set. The working memory degradation was associated with a significant increase in serum corticosterone.

In a study involving human subjects, Kirschbaum, Wolf, May, Wippich, and Hellhammer (1996) investigated the effect of cortisol elevation on declarative memory performance. In their first study, the exposure of 13 subjects to stressful tasks of mental arithmetic and public speaking was followed by a delayed word recall task (the initial list had 24 nouns, and subjects were asked to recall those beginning with the letters "Mo;" there were 10 such words in the original list). Those showing the greatest increase in cortisol elevation demonstrated the poorest performance (least correctly recalled words). Of the top 3 performers, 2 actually demonstrated small declines in cortisol secretion from baseline. In the second study, 40 males were administered 10mg of hydrocortisone acetate and subjected to a series of memory tasks beginning an hour later. The hydrocortisone treatment was associated negatively with decalarative memory performance (again, a cued recall task), but not procedural memory performance. Also, those receiving cortisol made significantly more errors in two spatial thinking tasks (reporting object location from previously studied narratives describing placement of

various objects in a park and in a barn). Here, again, it is worth noting the similarity between this task and spatial orientation of self and aircraft in an IFR scenario; such position visualization and spatial awareness also characterized evaluation of spatial demand and performance in the Wickens et al. (1993) study. Again, there is a pattern in which GC elevation is associated with decreased cognitive function.

While the body of literature associating GCs and cognitive dysfunction, like the research associating GCs and immunosuppression, suggests that elevated cortisol levels exert adverse effects, disparate evidence finds that hypocortisolism may also be deleterious. This is, perhaps, quite sensible in that the type I and type II receptors imply biphasic GC properties. DeKloet et al (1994), Oitzl and DeKloet (1992), and DeKloet, Oitzl, and Joels (1992) have found that adrenalectomy (removal of circulating steroids) impairs spatial learning in rats. The same effect can be accomplished with central pharmacological blockade of type I and type II receptors (Oitzl & DeKloet, 1992) as measured by rat performance in hidden escape platform water mazes. While type I antagonists affected search escape strategies and escape associated behavior, type II antagonists appeared to cause significant latency in platform finding. Oitzl and DeKloet (1992) suggest, on the basis of selective blockade of central type I and type II GC receptors, that "central MRs (type I receptors) are involved in processes of evaluation of the situation and response selection; central GRs (type II receptors) are involved in the consolidation of (spatial) information" (p. 69). GC receptor balance appears, then, to be of paramount importance in HPA regulation and cognitive function (DeKloet et al, 1994; DeKloet et al, 1992). Smriga, Saito, and Nishiyama (1996) found that adrenalectomy in

rats suppressed LTP and caused cell loss in the hippocampal dentate gyrus area. Corticosterone injection restored LTP. When corticosterone was administered to adrenal intact subjects, LTP was again impaired, although less severely than in the adrenalectomized rats. The Smriga et al. (1996) study robustly implies the biphasic properties of GCs in cognitive functioning. "Falling off" the periphery of an inverted-U relating cognitive performance to GC secretion, versus GC elevation, may well explain the profound GC effects discussed in the last few pages. Similarly, Chrousos and Gold (1992) note a number of psychological disorders appear to be biphasically related to cortisol secretion; both hyper- and hypoactive HPA axis activity/reactivity, and not just hyperreactivity, may be detrimental. It is possible that the predominance of findings for the adverse impacts of hyperactivity may well be a product of research design (for example, Kort and Weijma (1991) suggest that the severe stressors to which animals are subjected do not, for the most part, approximate the stressors which commonly confront humans).

While elevated (or depressed) serum GCs may act to directly damage or endanger hippocampal neurons via type II receptors, another mechanism is possible. Ben-Nathan (1994) showed cold and isolation stress on mice innoculated with viral encephalitis (west Nile virus—WNV) doubled their mortality rates. More shocking, stress-induced serum corticosterone elevation was associated with 55-75% mortality rates from innoculation with WN-25 or Sindbis virus (SVN), two neurovirulent viruses which normally lack neuroinvasive properties; the phenomena could largely be reproduced with dexamethasone injection (60% mortality)! These findings suggest an urgent research direction and the

possibility hippocampal neurodegeneration (as well as other neurodegenerative disease) may well be mediated by normally nonneuroinvasive pathogens entering the CNS during periods (duration?) of elevated serum GC levels, such as those experienced during stress. It may be worth examining, for example, in detail, the stress histories of Alzheimer's patients. Also, these concerns could, conceivably, lead to greater understanding of neurodegeneration in HIV as well (Sapolsky, personal communication, May 1997). Whether or not destructive, though less virulent, pathogens may cause chronic, and less severe, degradation of central function is also a matter for future research.

Stress-induced plasma GC elevation has also been implicated in immuno-suppressive lymphocyte cell-trafficking and distribution alteration. Hermann, Beck, and Sheridan (1995) found mice infected with influenza A/PR8 virus and exposed to restraint stress manifested reduced accumulation of mononuclear lymphocytes in the lungs and draining lymph nodes as compared to infected, unstressed controls. The immunosuppressive effect was abolished with RU486 (a GC antagonist) treatment. Supportive of the postulated role GCs may play in preventing immune system hyperactivity, continued treatment of infected mice with RU486 resulted in both increased mononuclear cell infiltration of infected tissue and high levels of mortality! Hermann et al. (1995) suggest cytotoxic by-products of immune function such as nitric oxide may induce life-threatening tissue damage in an unchecked immune response. Indeed, GCs appear to inhibit induction of nitric oxide synthase, without which nitric oxide cannot be produced (Moncada, 1992, as cited in Hermann et al., 1995). Similarly, circadian rhythms in peripheral lymphocyte concentrations are inversely related to the diurnal rhythm of cortisol

secretion (Milad, Ludwig, Anne, Middleton, & Jusko, 1994), perhaps explaining the increased incidence of fever and aches in the evening (plasma cortisol nadir) during infectious disease. It is important, however, that caution is exercised at this juncture in judging whether GC influence upon lymphocyte cell trafficking is immunosuppressive or not. Dhabhar, Miller, McEwen, and Spencer (1995) note lymphocyte exit from peripheral vasculature may protect them from stress-induced damage, a plausible scenario given observed serum GC, NE, and epinephrine increases during stress (sympathetic mechanisms of immunosuppression will be addressed shortly). Lymphocyte redistribution may also posit them at lymphoid tissue "battle stations" for maximal response to antigenic challenge (Dhabhar et al., 1995).

Indeed, stress has been shown in some cases to be immunoenhancing, exacerbating a wide variety of autoimmune disorders (Steinman, 1994). However, a possible explanation for this involves CRF itself. CRF has been isolated, for example, in the synovial fluid of rheumatoid arthritis patients and appears to be immunoenhancing. There is some thought animals susceptible to experimentally-induced arthritis may well release enough CRF to be immunoenhancing, but perhaps not enough during stress to drive immunosuppressive levels of GC secretion in that animal (Steinman, 1994). Short-term rotation stress (78 revolutions/min for one hour) has been shown by Korneva, Rybakina, Fomicheva, Kozinets, and Shkhinek (1992) to stimulate elevations in plasma IL-1α in mice, a finding correlated with serum corticosterone elevation. Kusnecov and Rabin (1994) suggest stressor characteristics and duration may well provide insight into the mixed findings encountered in stress-related research, an important point to ponder given

Cacioppo et al's (in press) reactivity hypothesis and emphasis on reactivity as a marker for changes in immunocompetence; consideration of such stressor properties is a must in advancing our understanding. GCs have also been shown to stimulate neutrophil egress from bone marrow (see Bateman, Singh, Kral, & Solomon, 1989), enhance IFN-γ activated macrophage activity (Munck & Guyre, 1991), and emerging research suggests some presence of GCs is vital in the survival and differentiation of thymocytes (King, Vaacchio, Dixon, Hunziker, Marguiles, & Ashwell, 1995). Again, it is likely GCs express some biphasic properties. Relatedly, caloric restriction in rats has been shown to increase longevity (K. Quadry, personal communication, April 1996). This longevity increase has been highly correlated with increases in baseline corticosterone secretion, and Nelson (1996) has proposed that these secretion patterns may well represent some optimal level of serum corticosterone. One is immediately reminded of the Zorilla et al. (1995) finding that elevated basal cortisol levels were positively associated with hardiness and positive affect. Perhaps there is some optimal serum cortisol level, and perhaps it marks generally beneficial decreased HPA reactivity (as Zorilla et al. (1995) suggest in their discussion). Blunted cortisol response (versus normal reactivity) to restraint stress in socially subordinate rats has also been associated with adverse biochemical outcomes (Blanchard, Sakai, McEwen, Weiss, & Blanchard, 1993). Coming full circle from the comments on CRF which opened this paragraph, ACTH also may have immunoenhancing properties. It has been shown in vitro to stimulate lymphocyte and NK cell activity, and may well counteract the predominantly immunosuppressive GC influence to prevent an "overshooting" of GC mediated immunosuppression (Gatti, Masera, Pallavicini, Sartori,

Staurenghi, Orlandi, & Angeli, 1993). Undoubtedly, the minority of studies finding stress-induced, GC mediated immunoenhancement provide clues to the dynamics of an intricate biochemical phenomenon which we do not yet fully understand.

Obviously, GCs are vitally important components in stress-induced immunosuppression, but they do not explain the totality of such immunosuppressive effects. In a certain respect, the findings above suggest further research and generate more questions than they answer. *How* do GCs effect all their immunosuppressive and infrequently immunoenhancing activities? What are the contributions to GC mediated immunosuppression and neuroendocrine response of stressor properties such as stressor typology and duration? What role does HPA axis habituation play? How does GC secretion and reactivity vary with measurable buffering psychological factors such as hardiness? What is the importance of findings opposite of conventionally predicted directions (immunoenhancement or GC receptor antagonist mediated disruption of spatial working memory function)? For many years, GC mediated explanations of stress-induced immunosuppression and illness have dominated this field of research; some have cautioned that stress-induced immunosuppression is a vastly more complex process (Sapolsky, 1992a; Mormede, 1990). Accordingly, we'll now turn our attention to sympathetically mediated mechanisms of immunosuppression.

Sympathetic Nervous System Mediated Mechanisms of Immunosuppression

Vigorous activation of the SNS during the immune response was mentioned earlier; like GC secretion, SNS activation appears to have a predominantly suppressive effect on immune function. SNS activation may affect immunological response through modulation of lymphocyte proliferation and trafficking, antibody secretion, and cell lysis (Madden & Livnat, 1991). The PVN projects heavily to autonomic nuclei in the brain stem, particularly the LC, and extensive activity in this neural circuitry is present in both stress and immune responses (Black, 1994a, 1994b). This suggests SNS activation, while beneficial in response to temporary stress situations, also becomes pathological when chronic or repeatedly "triggered" because of its additional function in preventing immune system hyperactivity. This is consistent with Agius's (1987) finding that sympathectomy increases the severity of experimental autoimmune myasthenia gravis. While overwhelmingly inhibitory, it is worth noting some SNS function is apparently immunoenhancing. Chemical sympathectomy of the rat spleen was followed by an 80% reduction in antibody response to certain antigenic challenges (Bellinger, Felten, Collier, & Felten, 1987). Additionally, it is the magnitude of SNS activation which may be key to understanding its influence on the immune system. Hellstrand, Hermodsson, and Strannegard (1985) found lower serum concentrations of epinephrine increased NK cell activity against leukemic cells by 30%. At higher concentrations, however, epinephrine suppressed NK cell activity (as well as that of lymphocytes in general). Both the immunoenhancing and immunostimulatory effects were abolished by the β-adrenergic receptor antagonist propanolol, suggesting epinephrine exerted its effects directly via

lymphocyte receptors; indeed, it is well established lymphocytes and macrophages possess adrenergic receptors believed to participate in observed immune system downregulation by epinephrine, NE, and β-adrenergic agonists (Black, 1994a; Sapolsky, 1992; Stein, Keller, & Schleifer, 1985). That higher serum levels of epinephrine may foster stress-induced immunosuppression is consistent with Stock, Zimmerman, and Teuchert-Noodt's (1993) observation that epinephrine levels rose 1800% and NE levels rose 200% in undergraduates during a two week examination period! These elevated catecholamine levels were associated with desensitization of β-adrenergic receptors on monocytes. In essence, the stress response likely elevates plasma epinephrine to immunosuppressive concentrations.

There is a great deal of experimental support for the generally immunosuppressive effects of SNS activation. Irwin et al. (1988) found central administration of CRF in rats activated autonomic and behavioral activity similar to that observed in stress. As alluded to previously, central CRF is considered to be a primary neuromodulator mediating sympathetic activation (as well as stimulating pituitary ACTH release in its hormonal role) (Black, 1994a; Dunn & Berridge, 1990). This administration of CRF also induced suppression of NK cell activity. Pretreatment with the ganglionic blocking agent chlorisondamine, however, abolished peripheral sympathetic activation and NK cell suppression; ACTH and corticosterone levels (i.e. the HPA axis) were unaffected. SNS mediation of NK cell suppression was clearly supported. NE has also been shown to inhibit class II MHC molecule/antigen expression on cultured astrocytes, apparently via

the β-adrenergic receptor (Frohman, Vayuvegula, Gurta, & Noort, 1988). To the extent NE might inhibit MHC expression in other cells, T cell function can be expected to be impaired. In a study by Manuck, Cohen, Rabin, and Muldoon (1991), only those subjects who displayed prominent SNS activation, as measured by elevated peripheral catecholamines and cardiovascular activity (HR, SBP, and DBP), showed suppressed lymphoproliferation (following administration of a mitogenic agent) of T cells in response to a 20 minute stressor. This is consistent with previously discussed work by Cacioppo et al. (in press). Again, as part of the stress reactivity hypothesis, Cacioppo et al. assert sympathetic reactivity, as measured by PEP, is a marker for elevated cortisol secretion and accompanying suppression of immunocompetence (presumably via GC mechanisms), although that was not supported in Manuck et al's (1991) study (cortisol secretion did not appear to change significantly, although Manuck et al note they did not allow time (30-40 minutes) for plasma cortisol levels to rise and peak). Of course, it is possible, even likely, the compromised immunocompetence is both GC and SNS mediated, given what is known of the interrelated nature of SNS and HPA axis activation. All this suggests the magnitude of SNS activation during the stress response may well determine an individual's immunological response to the given stressor. In mice injected with sheep red blood cells (antigen), sympathetic ablation using 6-hydroxydopamine hydrobromide (6-OHDA), a neurotoxin which acts specifically on NE terminal buttons, enhanced antibody production among splenic lymphocytes; the same immunoenhancing effect against other antigens (with the exception of thymus dependent antigens) was observed (Miles, Chelmicka-Schorr, Atweh, Otten, &Arnason, 1985). After sympathetic ablation, Miles et al.

observed increases in β-adrenergic receptor density on both splenic B and T cells, again suggesting SNS immunosuppression may be effected via lymphocyte receptors, and that immune function is directly under neural control. The observation that antibody response to some antigens (particularly thymus dependent antigens) is suppressed by sympathectomy (contrary to the general pattern) seems troublesome initially. If, however, the SNS serves an immune system "overshoot" preventive function, the thought it may not suppress thymus dependent antigen antibody response may be of little concern given the previously illustrated thymal sensitivity to GCs.

It should come as no suprise lymphocytes and other immune cells may well be under direct neural control. Felten, Felten, Carlson, Olschowka, and Livnat (1985) and Felten and Felten (1991) found extensive sympathetic NE innervation of lymphoid tissue to include the thymus, spleen, bone marrow, lymph nodes, and gut-associated lymphoid tissue (GALT). In many of these tissues, NE terminal buttons are found among lymphocytes and lymphocyte dense tissue; in the GALT, for example, Felten et al. (1985) note NE fibers "ramify profusely among lymphocytes" (p.755s). Consistent with these findings, Bulloch Cullen, Schwartz, and Longo (1987) transplanted embryonic and adult thymic tissue under the kidney capsule of syngeneic nude mice; ANS innervation followed, terminating among thymocytes, and was necessary for onset of thymal immunocompetence, underscoring the extent to which immune function and the SNS are integrated. Felten, Ackerman, Wiegand, and Felten (1987) found NE splenic innervation associating with T cell, macrophage, B cell, and IgM immunoreactive cell populations, and, in the splenic white pulp, NE terminal buttons formed contacts on lymphocytes that

can be characterized as "synaptic-like" (Felten & Olschowka, 1987). The overwhelming experimental data supports the ability of stress-induced SNS activity to suppress immune function, presumably via immune cell β-adrenergic receptors.

However, as with GC function, there may be a biphasic component to all of this, as previously suggested, and one that is time course dependent as well. While the mechanisms are not yet well understood, it appears that some NE promotes immunological activation early in the immunological response to pathogens (Madden & Livnat, 1991). Immunoenhancing effects of NE may be mediated via the α-adrenergic receptors as well as the β-adrenergic receptors. The latter appear to be prominently involved in immunosuppression later in the time course of pathogenic challenge (conceivably as part of the immunological overshoot prevention function), perhaps through altered sensitivity, and affinity for GCs as well as epinephrine and NE (Madden & Livnat, 1991). Overall, SNS modulation of immunological function appears to be quite complex, biphasic, and has been less thoroughly investigated than GC mechanisms of immunosuppression. There is reason to believe, however, that the two systems are highly integrated (as earlier suggested); additionally, there is reason to believe that both are under an unique mechanism of central control involving central opiate pathways (Jodar, Takahashi, & Kaneto, 1994). As such, it is not surprising that Cacioppo (1997) and colleagues have found sympathetically influenced reactivity such as HR and SBP to be markers for HPA reactivity.

In the preceding pages, I have attempted to thread a coherent and suggestive needle of thought and causality through a highly complex fabric of concepts and theory ranging from existential philosophy through psychology and physiology and into the realm of immunological competence and cognitive function which may have important organizational outcomes. The discussion to this point has outlined the structure and properties of cognitive hardiness, a stress resiliency construct firmly grounded, ultimately, in existential conceptualizations of reality, authenticity, and meaningfulness. Consistent with its existential heritage, hardiness appears to exert its effects by facilitating transformational coping strategies in a variety of domains, and in so doing, fosters authentic, healthful living and effective performance; it is also suggested that the effects of hardiness on health may be mediated by physiological reactivity. That is, in the psychoneuroendocrine response to stress, hardiness may contribute to cognitive function and situation appraisal that determines the character of physiological response to the stressor. In particular, the response of the HPA axis and the SAM system appear to be able to potently affect immunofunction and, for the HPA axis, cognitive function as well; should hardiness be able to influence these facets of physiological response to a stressor, positive outcomes are likely. It is hoped that the stitch linking this diverse collection of concepts has been tightened, drawing together what at first may appear as unrelated panels into a well constructed quilt of inquiry, a framework in which to embed the following investigations. In the two studies which follow, the relationships between hardiness, primary appraisal, stress, health, performance, and physiological reactivity will

be explored in two rather diverse samples; perhaps the results of such inquiry will advance understanding of the relationships in question.

Study 1

The first study explored the relationships between cognitive hardiness, stress perceptions, depression, and academic performance in college students. A PsycLit search conducted early in September, 1996, to identify research done on the relationship between hardiness and academic performance produced no results. Thus, it appears this study may constitute an original investigation into the effects of hardiness upon academic performance outside of strictly military and police training environments (Herlich, 1985; Westman, 1990). In keeping with earlier discussion and demonstration that hardiness has, in past research, demonstrated both moderating and main effects, the following hypotheses were established.

Hypothesis 1: Hardiness will moderate the stress-performance relationship.

Hypothesis 2: Hardiness will be positively predictive of performance.

Hypothesis 3: Hardiness will moderate the stress-depression relationship.

Hypothesis 4: Hardiness will be negatively related to depression.

Hypothesis 5: Hardiness will be negatively related to stress.

Given that achievement at the undergraduate level is a cognitively complex endeavor, and considering the previously discussed literature suggesting that stress degrades academic performance and contributes to states of negative affect/depression, the following hypotheses were also tendered.

Hypothesis 6: Stress will be negatively predictive of performance.

Hypothesis 7: Stress will be positively related to depression.

As Motowidlo et al. (1986) had found, it was believed depression would mediate the relationship between stress and performance.

Hypothesis 8: The stress-performance relationship will be mediated by depression.

The postulated relationships expressed in the hypotheses are illustrated in Figure 1.

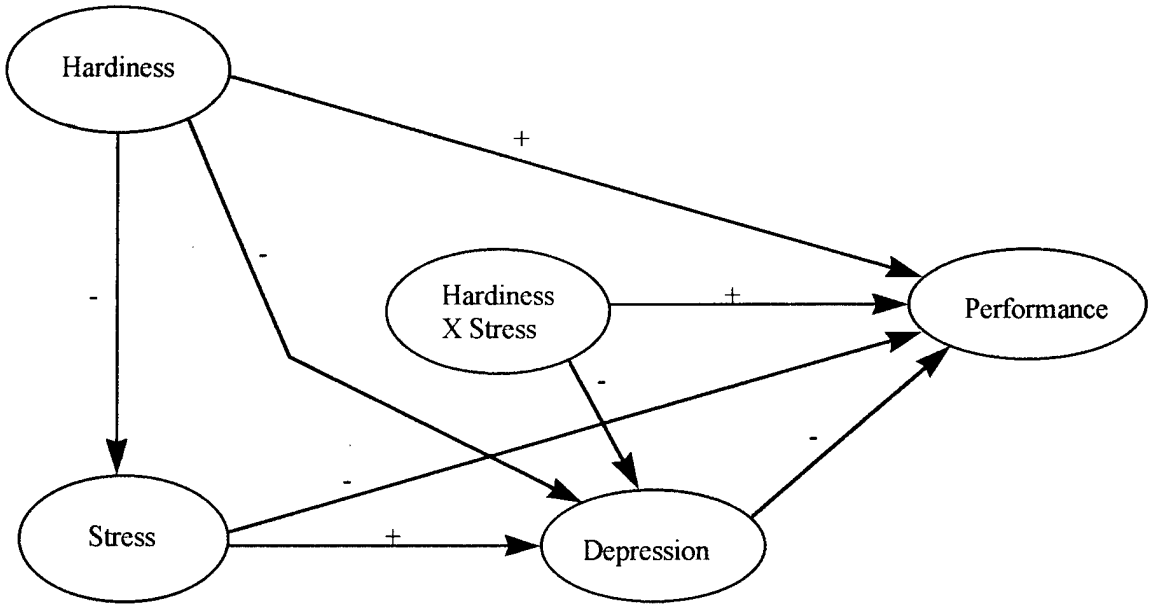

Figure 1. Hypothesized relationships, study 1.

Method

Participants

Initial participants consisted of 404 undergraduate students at a large midwestern university. Females represented 57.9% (234) of the sample; males represented 42.1% (170) of the respondents. Mean age of respondents was 19.3 years. The participants were predominantly in their first year of college (71%), while 20.5% were sophomores, 5.7% were juniors, and 2.7% were seniors (note that totals on class do not equal 100%

due to rounding). Only 134 participants, however, completed the depression measurement as part of a joint effort with another researcher. Of those, 9 submissions were dropped from the study for reasons to be later discussed. Therefore, there were 125 undergraduate students who ultimately participated in those phases of the study involving depression as a variable. Females represented 50.4% (63) of the sample while males accounted for 49.6% (62) of the participants. The mean age of respondents in this reduced sample was 18.6 years. The respondents were largely in their first year of college (74.4%); sophomores accounted for 21.6% of the participants, while 2.4% were in their third year of undergraduate education, and 1.6% were seniors.

Measures

Hardiness. Nowack's (1990) Cognitive Hardiness Scale (CHS) is a 30-item instrument requiring participants to respond to belief statements on a 5-point Likert scale ranging from "Strongly Agree" (1) to "Strongly Disagree" (5) (see Appendix A). Items 1-6, 13-14, 16, 18-19, 22, and 30 are reverse scored. The CHS is scored simply by summing the values of all 30 items (Nowack, personal communication, February 1996). The instrument has routinely demonstrated high internal consistency. Nowack (personal communication, February 1996) reports the CHS has an established Cronbach's alpha of .84, a unidimensional factor structure, and has demonstrated convergent validity with various coping and optimism scales as well as Kobasa's 71-item instrument (with the exception that the CHS shows little correlation ($r=.05$) with Kobasa's troubled challenge subscale). The CHS has also demonstrated criterion-related validity with outcomes of absenteeism and job satisfaction, subjective and objective health outcomes, and indices of

psychological health such as job burnout, anxiety, and mood (Nowack, personal communication, February 1996; Goss, 1994; Greene & Nowack, 1996; Sharpley et al., 1995). Nowack (1996) reports a test-retest reliability of .95 over two weeks and .55 over three years. Goss (1994) found a test-retest reliability of .84 over one year while, in study 2 of this effort, a test-retest reliability of .89 was demonstrated over an average interim of 172 days. As mentioned before, the CHS balances negatively and positively worded items. In two unpublished studies, the CHS was found to assess hardiness as a construct significantly independent of (not confounded with) negative affectivity and psychological distress characteristic of neuroticism (Greene & Nowack, 1996; Schwartz, Schwartz, Nowack, & Eichling, 1992). Finally, Nowack's CHS was constructed and validated using a gender balanced, culturally diverse sample (Nowack, 1990).

Stress. In measuring participant's perceptions of stress, the Perceived Stress Scale (PSS) (Cohen et al., 1983) was selected as a clearly worded, psychometrically sound, 14-item instrument validly measuring global perceptions of stress. The scale required participants to respond to questions about how often they have felt or thought a certain way in the preceding month; response was given via a 5-point Likert scale ranging from "Never" (1) to "Very Often" (5) (see Appendix A). For comparison of this study to other uses of the PSS, the reader should note that Cohen et al. (1983) originally used a Likert scale ranging from "0" to "4." Items 4-7, 9-10, and 13 are reverse scored, and the PSS is scored by summing the scores on all 14 items. The PSS is expressly designed to "measure the degree to which situations in one's life are appraised as stressful" (Cohen et al., 1983, p. 385). The PSS has emerged as being a valid and better predictor of adverse stress-

related outcomes (depression, physical symptomatology, physician visitation, anxiety, and burnout) than various life-events and daily hassles scales despite criticism it is confounded with measures of psychological distress (Cohen et al., 1983; Hills & Norvell, 1991). The PSS has an established Cronbach's alpha ranging from .84 to .86 in multiple studies (Cohen et al., 1983). Cohen et al suggest that the PSS's predictive validity extends over a relatively short period of time ($\leq$2 months); in study 2 of this effort, however, the PSS test-retest reliability over an average interim of 172 days was .60 (n=14), suggesting the construct of perceived stress may fluctuate more slowly in some populations. Finally, it has been suggested (Cohen et al., 1983; Hills & Norvell, 1991) that the PSS may, because it assesses perceptions of stress, be ideal for research in the areas of locus of control, hardiness, and stress research in general.

Depression. Depression was measured using the 21-item Beck Depression Inventory (BDI) (Beck, 1987; Beck, 1978). The BDI is a well-established instrument designed to "assess the severity of depression in adolescents and adults" (Beck, 1987, p.1). Each of the 21 items is rated on a 4-point scale (0-3), and the BDI score is a summation of scores on all 21 items. Scores from 0-9 are considered to be within normal range, 10-18 indicates mild to moderate depression, 19-29 indicates moderate to severe depression, and scores exceeding 30 suggest severe depression (Beck, 1987). In non-clinical populations, scores greater than 15 may reflect depression requiring professional mental health care (Beck, 1987). The BDI has an established Cronbach's alpha of .81 over 15 non-clinical samples and has robustly demonstrated content, discriminant,

construct, and convergent validity (Beck, 1987). The BDI has not been included in Appendix A due to copyright restrictions.

Performance. The selected measure of performance for the undergraduate participants in this study was the Fall 1996 semester GPA. All participants had the option to sign a release granting researcher access to their semester GPA (see Appendix A); participants were informed they could withdraw permission to access GPA at any time.

Demographic Information. Participants were asked to provide gender (female=0, male=1), age, academic class (freshman=1, sophomore=2, junior=3, and senior=4), semester course load, and student identification (social security number) (see Appendix A). Information on race and ethnicity was requested as an optional item.

Procedures

Questionnaires were completed by participants during mass testing sessions on the dates of September 10, September 25, and October 30, 1996 (see Appendix A for questionnaire—note again that the Beck Depression Inventory (BDI) is not included as a result of copyright restrictions). The depression instrument was only completed by those participants attending the September sessions. For those who granted access, semester grades were obtained from the university registrar's office in February, 1997. All respondents were assured of complete confidentiality and were duly informed that only group responses would be used in any reports resulting from this research effort. All participants also completed an informed consent form in duplicate (a copy maintained on file and a copy to be retained by the participant for their records/reference). All participants were thoroughly debriefed following completion of the questionnaires;

participants scoring greater than 9 on the BDI were privately informed of their scores and assisted in interpreting the scores. Participants were treated in strict accordance with the American Psychological Association's (APA) current principles of ethical conduct.

Statistical Analyses

As earlier mentioned, hardiness research has often made use of ANOVA in analyzing the data; in such an approach, hardiness scores, for example, are dichotomized, usually as the result of median splitting. Unfortunately, such an approach discards information, does not treat hardiness as the continuous variable that it is, may lead to invalid inferences, and may make comparisons of studies rather difficult (Hull et al., 1991; Parkes, 1994; Tabachnick & Fidell, 1996). In an effort involving use of multiple independent variables (IVs) to explain variance in, or predict, a continuous dependent variable (DV), regression analyses are an appropriate approach (Ott, 1993; Rencher, 1995; Tabachnick & Fidell, 1996). Further, regression analyses have been more recently cited as an improved and appropriate methodology to use in hardiness research (Hull et al, 1991; Parkes, 1994). While Hull et al. (1991) do express some thoughtful reservations over careless use of regression analyses on the grounds of problems such as the threat of multicollinearity, it is also true that there are various techniques to identify the presence of multicollinearity, and, in many instances, correct or control it (Fox, 1991; Tabachnick & Fidell, 1996). Finally, multiple linear regression, based on the general linear model, demands no distributional assumptions about the IVs, "other than uncorrelation with the errors, (making) the domain of the linear-regression model...broader than it first appears" (Fox, 1991, p. 9). Indeed, multiple linear regression can accordingly accommodate such

non-normally distributed variables as dummy coded variables, interaction terms (used to check moderated relationships in this effort), and polynomial terms (Fox, 1991). Of course, there are assumptions about the normality, linearity, and homoscedasticity of the residuals (Tabachnick & Fidell, 1996), although the general linear model may well be (and this is a hotly debated issue) robust to less severe violations of these assumptions (Tabachnick & Fidell, 1996; Keppel, 1991). As a result of the above, regression analyses have been selected to analyze the data gathered in this study. More particularly, regression analyses were all conducted with unix-based SAS version 5 (SAS Institute, 1989, 1985); uniquely, extensive use was made (in this and the following study) of setwise regression (also sometimes referred to as "all possible regressions" (T. Loughin, personal communication, July 1996)), an option not currently available in SPSS software. Setwise regression allows use of multiple criteria and theoretical consideration in determination of regression models which simultaneously maximize variance explained and efficiency; this procedure permits competing models to be rather easily compared (T. Loughin, personal communication, July 1996; Tabachnick & Fidell, 1996).

Results

Descriptive analyses were conducted to ensure correct data entry and assess initial means, standard deviations, and intercorrelations among the variables previously discussed. Another motivating factor in this effort was to determine the importance of depression in predicting academic performance. The bivariate correlations revealed that depression appeared to be related to performance ($r=-.20$, $N=132$, $p<.05$). The difference in value for N from the previous discussion reflects the unavailability of GPA for 2

participants completing the BDI; per the university registrar's office, this implies they either provided an incorrect student ID or they withdrew from all coursework. Therefore, the decision was made to include only those sets of data with a BDI score in hypotheses testing (such a requirement obviously existed for the testing of hypotheses 3, 4, 7, and 8). This sample size was still sufficient to test the stated hypotheses; that is, it is recommended $N \geq 50+8$(the number of predictors) (Tabachnick & Fidell, 1996). With the available sample determined, it was appropriate to screen the data and test assumptions of residual normality, linearity, and homoscedasticity.

A number of varied methods were applied in the screening of the sample. These included a number of univariate analyses (including mean, standard deviation, variance, skewness, kurtosis, use of the Kolomogorov D statistic (SAS Institute, 1989) to test normality, stem-and-leaf plots, box plots, and normal probability plots), examination of scatter and residual plots, examination of residuals (raw and studentized), studentized residual plots, construction of 95% confidence intervals around predicted values for depression and academic performance, and various influence diagnostics examining predictors and their relation to the two DVs (Cook's D, HAT DIAG, DFBETAS, DFFITS, and partial regression leverage plots of residuals for hypothesized regression modelling) (Tabachnick & Fidell, 1996; Loughin, personal communication, July 1996; Fox, 1991; SAS Institute, 1989, 1985). Of couse, the influence deletion statistics are mathematically dependent upon the "hat values" and residuals (Fox, 1991).

Scatterplots, residual plots, and hypothesis-driven partial regression leverage plots identified a number of influential outliers that can be characterized as high in discrepancy

and influence (Tachnick & Fidell, 1996; Fox, 1991). These graphical representations were corroborated by examining the calculated residuals, whether or not they fell within 95% CIs constructed around predicted values, and by examining the various indicators of influence listed above. As a result, 3 residuals were identified as falling outside of the 95% CI due to the unique combination of all predictors of performance (examination of DFBETAS revealing no particular IV to be especially influential). Five other residuals fell outside of the 95% CI; examination of the DFBETAS values, scatterplots, and partial regression leverage plots indicated that depression values were influential (high discrepancy, high influence) for 2 subjects, perceived stress values were influential for 2 subjects, course load was influential for 2 subjects, and hardiness was influential for one participant. One value for load could be considered a significant outlier (-3.43 s.d.), while one value for depression was a significant outlier (+3.94 s.d.). A ninth observation contained a significant hardiness outlier (-3.98 s.d.). These outlying and/or influential observations/data points are a matter of concern because they may unduly influence the results and precision of regression analyses (Tabachnick & Fidell, 1996; Fox, 1991). Such influential data should, accordingly, be deleted, transformed, or rescored. For clarity, and because of the exploratory nature of this original investigation into the relationship between hardiness and academic performance, I have chosen to delete the 3 observations falling outside of the 95% CI for residuals as a result of unexplained multivariate influence. In the other 6 observations, the unduly influential values were deleted. As a result, the final sample for further analyses contained 123 complete sets of data (125 complete sets of data when only considering depression as the DV of interest).

While regression analyses with larger sample sizes tend to be relatively robust to violations of the normality of residual distribution, maximal efficiency of least-squares estimation is obtained when errors are normally distributed (Fox, 1991); additionally, regression analyses assume measure reliability, a condition which appears to have been reasonably met in the instruments selected. It was desirable to examine the residuals for normality in the above described sample. Plots of residuals for each observation were examined for normality; normally distributed residuals should be symetrically distributed above and below the predicted value (Tabachnick & Fidell, 1996). No substantial violations in residual normality were observed, although there was a slight positive skewing of the residuals for depression. The assumption that the residuals are normally distributed appears to have been met. Additionally, residual plots revealed no substantial evidence of nonlinearity. Finally, residual plots were also examined to validate the assumption of homoscedasticity. While the residuals for academic performance were homoscedastic, the residuals for depression demonstrated moderate heteroscedasticity (variance in residuals increasing with larger predicted values). The concern is that heteroscedasticity may weaken the analysis (Tabachnick & Fidell, 1996); however, in this case, the heteroscedasticity was not deemed severe enough to warrant transformation and possibly generate the interpretive difficulties inherent in DV transformations (Loughin, personal communication, July 1996). Thus, it appeared underlying assumptions for the application of regression analyses were reasonably met.

As previously mentioned, extensive univariate analyses were also conducted. While, as previously addressed, there are no assumptions about IV distributions,

prediction equations are often enhanced by normally distributed IVs (Tabachnick & Fidell, 1996). Using a conservative α of .01 for the Kolomogorov D statistic, both hardiness and stress were determined to be normally distributed. Depression was not normally distributed (Kolomogorov D =.78, p=.0001), due to both positive skew (2.19) and kurtosis (6.12), although such a distribution is within theoretical bounds (Beck, 1987). Seven outliers (all $\geq$ 18) were maintained in the data; although they exerted leverage in relationships with other variables, previously described analyses determined these points to be of low discrepancy and influence. In sum, then, it is suggested that the relationship between IVs and each of the two DVs should serve to enhance the regression equation.

The means, standard deviations, and intercorrelations between the variables in this study are displayed in Table 1. It is worth noting some relationships not previously anticipated. Gender was found to be negatively related to both depression (r=-.22, p<.05) and GPA (r=-.27, p<.005). This pattern suggests the possibility of a gender-depression interaction in the prediction of GPA which must be investigated. Also, course load was found to be robustly related to hardiness (r=.27, p<.005) and GPA (r=.38, p<.001). Hardiness and stress were related to depression in the predicted directions (r=-.57, p<.001 and r=.63, p<.001, respectively), supporting hypotheses 4 and 7. Hardiness was related to stress in the predicted direction as well (r=-.70, p<.001), supporting hypothesis 5.

Hypotheses 1, 2, and 6 were then tested using regression analyses. However, the unexpected relationships between GPA, course load, and gender suggested the need for a modelling of relationships other than that initially suggested and illustrated in Figure 1. Accordingly, the variables of gender, load, hardiness, stress, depression, the hardiness X

Table 1

Means, Standard Deviations, and Intercorrelations for Study 1 Variables

Variables	N	Mean	Std Dev	1	2	3	4	5	6	7
1. Gender	125	.50	.50							
2. Age	125	18.64	1.52	.25**						
3. Class	125	1.31	.60	.26+	.49++					
4. Load	125	14.30	1.65	.00	.10	.24**				
5. Hardiness	125	111.27	12.18	-.05	.08	.09	.27+			
6. Stress	125	38.86	7.61	-.14	-.21*	-.16	-.24**	-.70++		
7. Depression	125	6.17	6.20	-.22*	-.09	-.07	-.21*	-.57++	.63++	
8. GPA	123	2.78	.76	-.27+	-.01	-.06	.38++	.36++	-.20*	-.25+

Note: *p<.05, **p<.01, +p<.005, ++p<.001

stress interaction term (to check for moderating effects (Jaccard, Turrisi, & Wan, 1990; Baron & Kenny, 1986)), and the gender X depression interaction term were submitted to a setwise regression approach in which the best 3 models (per an R^2 criterion) for GPA regressed onto 1-7 IVs were produced. That is, the 3 best 1-variable models, the 3 best 2-variable models, and so on were produced for examination. In addition to the R^2 criterion, the adjusted R^2, Mallows' C_p, mean squared error (MSE) between predicted and actual DV value, and Akaike's information criterion (AIC) were considered in selecting the simultaneously most explanatory and efficient model in conjunction with theoretical considerations and stated hypotheses. In selecting an appropriate model, the desire was to maximize R^2 and its adjusted value (Tabachnick & Fidell, 1996; Rencher, 1995; Achen, 1982), minimize Mallows' C_p such that its value is $\leq$ p, where p is the number of IVs (Rencher, 1995; Ott, 1993; Achen, 1982), minimize MSE (Achen, 1982), and minimize AIC (T. Loughin, personal communication, July 1996).

The optimal model selected in consideration of the above criteria and applicable theory was a model that included gender, load, hardiness, stress, and depression as predictor variables; the model was significant ($R=.54$, $F(5, 117)=9.86$, $p<.0005$). Table 2 displays the unstandardized regression coefficients (B), the standardized regression coefficients (β), the squared semipartial correlations (sr_i^2, representing the unique variance in GPA explained by the particular variable), R^2, and adjusted R^2. Examination of Table 2 reveals that hypothesis 2 was supported; hypotheses 6, however, was only partially supported (GPA regressed on to only stress yielded a significant result, $R=.20$, $F(1,121)=5.059$, $p<.05$) as stress does not significantly predict academic performance in

the presence of other predictors. Inclusion of the hardiness X stress and gender X depression interactions in the above model (and multiple permutations of that model) revealed they were not significantly predictive of academic performance, explaining almost no unique variance ($sr^2_{\text{hardiness X stress}}$=.0054 and $sr^2_{\text{gender X depression}}$=.0003). Hypothesis 1 was not supported.

Table 2

Results of Multiple Regression Analyses with GPA as the Dependent Variable (N=123).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
Intercept	-0.961	0.0	-0.777	.4389	
Gender	-0.417	-0.276	-3.341	.0011	.07
Load	0.138	0.296	3.632	.0004	.08
Hardiness	0.015	0.238	2.059	.0417	.03
Stress	0.012	0.121	1.014	.3126	.01
Depression	-0.023	-0.191	-1.811	.0727	.02
R^2=.30				Unique variability=.21	
Adjusted R^2=.27				Shared variability=.09	

The above model was then examined for singularity and multicollinearity. No evidence of singularity was found. Hardiness and stress did, however, have a bivariate correlation of -.70, and an |r| of .70 or higher is suggested as a possible marker for multicollinearity, although substantial threats to the multivariate solution appear to be associated with an |r| of .90 or higher (Tabachnick & Fidell, 1996; Fox, 1991). Collinearity diagnostics reflected that the sixth principal component for the solution explained 77% of the variance in hardiness and 54% of the variance in stress; this, along

with the observation that addition of stress to the solution maximized the adjusted R^2, minimized MSE, and yielded an acceptable C_p (4.405) suggested that multicollinearity was a negligible threat in this solution.

Hypothesis 3 was next examined; since gender and load were unexpectedly related to depression, they, along with hardiness, stress, and the hardiness X stress interaction term were submitted to a setwise regression analysis using the five model selection criteria described above. The optimal solution included gender, hardiness, stress, and the interaction term as predictors; the model was significant ($R=.72$, $F(4, 120)=32.08$, $p<.0005$). Solution information is displayed in Table 3. Consistent with the squared semi-partial correlation values, a hierarchical approach (entering the interaction term last) does not alter the solution. Examination of Table 3 reveals that hypothesis 3 was robustly supported. Inclusion of load in the model (and various permutations of the model) showed it did not explain unique variance in depression ($sr^2=.0003$).

Table 3

Results of Multiple Regression Analyses with Depression as the Dependent Variable (N=125).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
Intercept	-53.752	0.0	-2.813	.0057	
Gender	-2.087	-0.169	-2.573	.0113	.03
Hardiness	0.451	0.886	2.685	.0083	.03
Stress	1.994	2.446	4.397	.0001	.08
Hardiness X Stress	-0.016	-1.500	-3.736	.0003	.06
$R^2=.52$				Unique variablility=.20	
Adjusted $R^2=.50$				Shared variability=.32	

For a moment it is worth discussing the results reported in Table 3, especially in regards to the seemingly unexpected direction in the hardiness term coefficients. This reversal of coefficient direction, as well as interaction term coefficients themselves, have often been cited as problematic in the interpretation of regression analyses, but it need not be; the reversal of sign is reflective of the conditional relationships between the moderator (hardiness), stress, and depression (Jaccard et al., 1990). A clearer understanding can be arrived at through algebraic manipulation (Jaccard et al., 1990) which may aid the reader's interpretation of this and later regression tables. The regression equation produced by the analyses is:

$$\text{Depression} = -53.75 - 2.09X_{gender} + .45X_{hardiness} + 1.99X_{stress} - .02X_{hardiness}X_{stress}$$

When hardiness is high (143, for example (the highest score in this sample)),

$$\text{Depression} = 10.65 - 2.09X_{gender} - .87X_{stress}$$

When hardiness is low (84, for example (the lowest score in this sample)),

$$\text{Depression} = -15.9 - 2.09X_{gender} + .31X_{stress}$$

That is, when hardiness is high, a one unit increase in stress is associated with a predicted .87 unit decrease in depression; when hardiness is low, a one unit increase in stress is associated with a predicted .31 unit increase in depression. Hardiness, then, robustly buffers participants against stress-related depression.

This model was also examined for the threats of singularity and multicollinearity. Evidence of singularity was not found. Some threat of multicollinearity was suggested by the bivariate correlations between stress and hardiness (-.70) and stress and the interaction term (.81) (Tabachnick & Fidell, 1996), although, again, neither correlation exceeded .90.

Collinearity diagnostics were conducted and revealed the fifth principal component for the solution explained 99% of the variance in hardiness, 99% of the variance in stress, and 95% of the variance in the interaction term; this suggests multicollinearity (SAS Institute, 1985). Yet, all 5 criteria used in model selection robustly supported the selected model as offering a superior explanation of variance in depression. I have, therefore, retained the model as is; however, caution in interpretation is warranted.

Hypothesis 8 was next investigated. Baron and Kenny (1986) suggest that mediation should be investigated via application of three regression equations: (1) regressing the mediator on the IV, (2) regressing the DV on the IV, and (3) regressing the DV on both the IV and the proposed mediator. For mediation to exist, the IV must predict the mediator, the IV must predict the DV, and the mediator must predict the DV in the third equation with a decrement in the effect of the IV on the DV. Perfect mediation exists if the IV in the third regression has no effect on the DV in the presence of the mediator. Thus, to have perfect mediation:

1) IV → Mediator (relationship significant),
2) IV → DV (relationship significant), and
3) IV → Mediator → DV (relationship between IV and DV nonsignificant in the presence of the Mediator).

The first two regressions constitute a univariate analysis; inspection of the correlation matrix indicates the first two conditions are met. Academic performance was then regressed onto depression and stress; when depression is controlled for, stress is not

predictive of performance (for H_0: B=0, t(122)=-.60, n.s.). Hypothesis 8 was robustly supported.

Preceding analyses also suggested that load might mediate the relationship between hardiness and academic performance. When performance was regressed on to hardiness and load (R=.46, F(2, 120)=16.32, p<.001), both predictors remained significant, although, by inspection of standardized path coefficients in regression 3 (.30 (load) versus .27 (hardiness)) and variance explained in regressions 2 and 3 (.13 and .21, respectively), the predictive contribution of hardiness was diminished. Load appears, then, to only partially mediate the relationship between hardiness and performance.

In sum, hypotheses 2, 3, 4, 5, 7, and 8 were supported, hypothesis 6 was conditionally supported, and hypothesis 1 was not supported. The resultant relationships are depicted in Figure 2; standardized path coefficients from the above regressions are indicated.

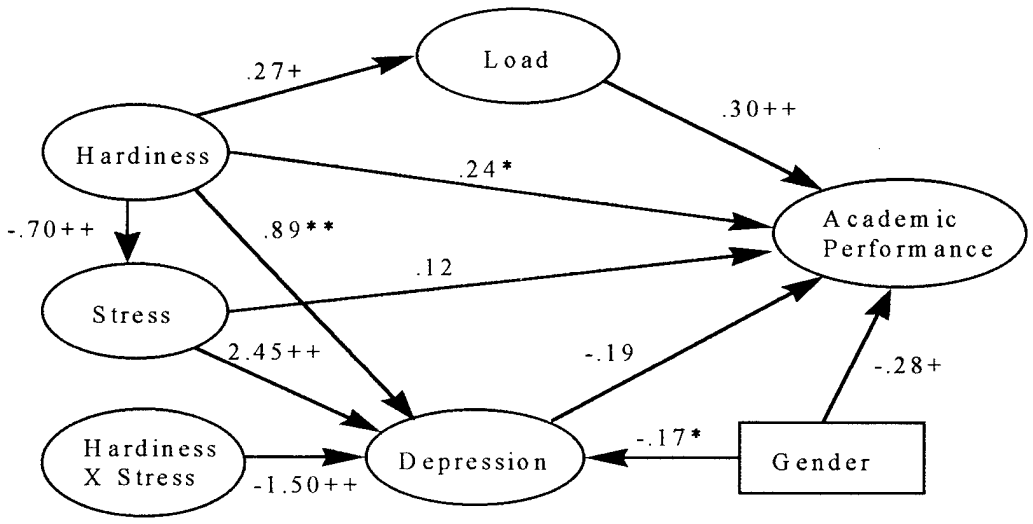

Figure 2. Variable relationships, study 1. Note: *p<.05, **p<.01, +p<.005, ++p<.001

Discussion

Hardiness has been posited in previous research to moderate the relationship between stress and performance (Herlich, 1985; Westman, 1990). It has also been shown to both moderate the relationship between stress and adverse psychological outcomes and have main effects on such outcomes (Goss, 1994; Hills & Norvell, 1991; Manning et al., 1988; Nowack, 1991; Rhodewalt & Zone, 1989; Rush et al., 1995; Sharpley et al., 1995). In the present study, the relationships between hardiness, stress, depression, and academic performance were explored in a sample of 125 undergraduate students. Surprisingly, it does not appear relationships between hardiness and academic performance have been previously explored. While hardiness was not found to moderate the relationship between stress and academic performance, a significant main effect was demonstrated in the predicted direction. Hardiness robustly moderated the stress-depression relationship as well as exerting main effects on depression in the predicted direction. Before continuing a discussion of these findings, however, limitations inherent in this study must be addressed.

One possible confound in this study is the narrowing of the sample to those who had participated in joint data gathering sessions that involved the measurement of depression. It is possible that the sample drawn to a jointly conducted depression-related memory experiment may not be representative of the population of college students. Support for such a confound may be drawn from the significant negative relationship between gender and depression (females being more depressed than males). Such a relationship is not usually found in non-clinical populations (Beck, 1987), although Beck (1987) does not express need for concern if the relationship does not exceed |.30|. That

gender was a significant predictor may also signal a Type I error; of course, demographic characteristics at the university in question may reflect a population different than those sampled from in other studies. At least one researcher has commented that the students at the university in question tend to maintain appreciably more conservative values and beliefs than their counterparts at other universities (C. Cozzarelli, personal communication, October, 1995); perhaps the gender effect is reflective of psychosociological differences between the students at this university and those at other institutions of higher learning.

The sample in this study was also very homogenous with respect to age, state of residence, and the domain of performance measured. Accordingly, one should exercise caution in generalizing these results to domains other than academic performance and to demographic clusters that differ in age and geographic location. The external validity of these findings should not be a matter of careless assumption.

Some may take issue with the regression of depression on to various predictors and the causal relationship suggested, especially given the BDI was completed concurrently with stress and hardiness measures. While correlational analyses themselves do not permit causal inference, the previously cited works by Kessler (1997), Chrousos and Gold (1992), Perkins et al (1991), and Lazarus (1984) provide a psychological and physiological theoretical framework in which such directionality can be presumed likely. While I am not proclaiming a directional relationship, I certainly am suggesting causal direction as grounded in theory.

The emergence of gender and course load as significant predictors of academic performance was surprising. I have already addressed the possibility of both a Type I error and psychosociological influences being responsible for the gender effect in depression; the same may be the case in the relationship between gender and GPA. If anything, increased course load would intuitively have been negatively associated with GPA. While the relationship between load and academic performance at first seems illogical, upon closer inspection one finds rather strong consonance with theory. Course load appears to partially mediate the relationship between hardiness and academic performance. That is, hardy people appear to take heavier course loads and academically perform better than their less hardy counterparts! When one examines the existential grounding of hardiness and its expression through the 3 components of hardiness, acceptance of heavier loads can be considered consistent with a challenge orientation and pursuit of opportunities for the purpose of growth and transformation. Voluntarily taking on heavier course loads may also reflect an internal locus of control; hardy people are likely to believe they can successfully handle the heavy workload. Lastly, an orientation high in commitment to activities and relationships deemed to be meaningful may well explain perseverence and successful completion of activities such as the pursuit of higher education. Upon closer inspection, then, the observed relationship between hardiness, course load, and academic performance is both reasonable and theoretically consistent.

Unexpectedly, stress was not associated with academic performance as Felsten and Wilcox (1992) and Lloyd et al. (1980) had found. Only in the univariate analysis did stress emerge as being predictive of performance. Consistent with Lazarus' (1993a, 1984)

contentions and the work of Lazarus and Folkman (1984), however, appraisal and individual differences (such as hardiness) may prominently moderate the relationships between stressors, stress, and various outcomes. Stress, per se, may not be harmful to the extent various individual resources are stress-buffering or facilitative of active coping (given, in this instance, the premise that active or problem-focused coping characterizes academic achievement). Robbins, Spence, and Clark (1991), for example, found that GPA was not related to stress, but was related to achievement striving and academic expectations. Perhaps the lack of a relationship between stress and performance in the presence of a stress resiliency resource (hardiness) and related course loads, then, is not surprising. However, stress may at times, especially in the absence of resiliency resources, indirectly affect performance through various mediators, such as mood or affect (Goss, 1994; Motowidlo et al., 1986). Motowidlo et al's finding that depression mediates stress-performance relationships was vigorously supported in this study.

While stress was strongly associated with depression, hardiness was impressively capable of moderating that relationship. The algebraic calculations presented earlier suggest that the more hardy the individual, the less depressed they are likely to become as stress increases. Perhaps this occurs because hardy individuals perceive the stressful situation as a challenge, a potential outcome to be influenced, a meaningful endeavor which demands committed effort. Less hardy individuals, on the other hand, become more depressed as stress increases; perhaps the more stressful situation is threatening, disruptive of valued stability, perceived as uncontrollable, and associated with powerlessness.

The model presented in Table 2 explains a respectable 30% of the variance in GPA. This is appreciable when one considers that traditional predictors of academic and work performance often explain equivalent or comparatively less variance. Robbins et al. (1991) conducted a study laudable for its inclusion of a wide range of potential correlates of academic performance; the Scholastic Aptitude Test (SAT), for example, was only modestly correlated with GPA ($r=.35$ for males, $r=.38$ for females). In the workplace, various tests of cognitive ability and "g" appear to be widely accepted as having the greatest validity in predicting job performance; Saal and Knight (1995) reported that some researchers have claimed general cognitive ability alone has a predictive validity of .54 across all jobs. Yet, the model presented herein explains either more or equivalent variance in performance outcomes. Hardiness and load alone explain 21.4% of the variance in GPA. This suggests that although personality traits and various individual differences have traditionally been poorly associated with performance (Saal & Knight, 1995), hardiness may well represent a discriminating individual difference. Further research to assess the predictive validity of hardiness (and, perhaps, other individual differences) across diverse cultures is warranted and, at present, promising.

In sum, hardiness demonstrated a main effect on academic performance and both main and moderating effects on depression; these effects were in the predicted directions. Further, the hardiness-academic performance relationship was mediated, in part, by course load, suggesting that hardy individuals take heavier course loads, and they perform better with those heavier course loads than less hardy peers with lighter loads. The stress-academic performance relationship was mediated by depression, supporting previous

findings by Motowidlo et al. (1986). Given the demonstrated ability of hardiness to be predictive of performance in a manner theoretically consistent with its existential underpinnings, it is desirable to examine the effects of hardiness on more varied outcomes in a vastly different domain; findings in predicted directions would permit greater generalization of results. Accordingly, in the following study both previously demonstrated and novel relationships between hardiness, appraisal processes, stress, physiological reactivity, health, and performance outcomes were extensively investigated in a sample of UH-1 helicopter pilots.

Study 2

The second study explored a broad range of relationships between hardiness, stress, appraisal processes, physiological (cardiovascular and neuroendocrine) function, illness/injury, and performance in United States Army (USA) UH-1 helicopter pilots. This small N study should be viewed as a complex exploratory effort which both makes original contributions to the literature in the psychological and physiological realms and suggests rather novel research directions for future inquiry. This study also is divided into "global" relationships between the variables listed above and "acute" relationships per a demanding, full motion, IFR helicopter simulation. Additionally, "cross-over effects" will be a term applied to relations between variables in the acute and global portions of the study (primarily, these cross-over effects will be concerned with the relationships between physiological reactivity and illness/injury rates).

Hypothesized global relationships will be addressed first. Given that hardiness has demonstrated both moderating and main effects in past research, and given that some physiological parameters (especially cortisol secretion) appear to be related to cognitive function, the following hypotheses were established.

Global hypothesis 1: Hardiness will moderate the stress-performance relationship.

Global hypothesis 2: Hardiness will be positively predictive of performance directly and as mediated by physiological baselines.

Global hypothesis 3: Hardiness will moderate the relationship between stress and illness/injury.

Global hypothesis 4: Hardiness will be negatively predictive of illness/injury rates as mediated by physiological baselines.

Global hypothesis 5: Hardiness will be negatively related to stress.

Global hypothesis 6: Hardiness will be negatively related to physiological baselines.

On the assumption that piloting aircraft and active involvement in an organization responsible for aeromedical evacuation is cognitively demanding, and given that stress has been associated with cardiovascular parameters and tonic cortisol secretion, the following hypotheses are offered.

Global hypothesis 7: Stress will be negatively predictive of performance directly and as mediated by physiological baselines.

Global hypothesis 8: Stress will be positively predictive of illness/injury as mediated by physiological baselines.

The next two hypotheses logically flow from the above.

Global hypothesis 9: Physiological baselines will be positively related to illness/injury rates.

Global hypothesis 10: Physiological baselines will be negatively predictive of performance as mediated by illness/injury rates.

A single "cross-over" hypothesis seeks to investigate the validity of Cacioppo's (in press) reactivity hypothesis.

Cross-over hypothesis 1: Physiological reactivity will be positively related to

illness/injury.

Hypothesized acute relationships will be addressed last. Again, these hypotheses refer to participant involvement in an evaluated helicopter simulator scenario. These hypotheses follow from previously discussed research and theory suggesting both moderating and main effects for hardiness in stress-performance relationships, the premise that hardy individuals appraise events differently than less hardy individuals, and suspected relationships between hardiness, physiological reactivity (especially cortisol secretion/reactivity), and performance. The 10 "acute" hypotheses are as follows.

Acute hypothesis 1: Hardiness will moderate the stress-performance relationship.

Acute hypothesis 2: Hardiness will be positively predictive of performance directly and as mediated by physiological reactivity, threat, and challenge appraisals.

Acute hypothesis 3: Hardiness will be negatively predictive of stress directly and as mediated by threat and challenge appraisals.

Acute hypothesis 4: Stress will be negatively related to performance directly and as mediated by physiological reactivity.

Acute hypothesis 5: Hardiness will moderate the relationship between stress and physiological reactivity.

Acute hypothesis 6: Hardiness will be negatively predictive of physiological reactivity (with the exception of SBP) directly and as mediated by stress.

Acute hypothesis 7: Hardiness will be negatively predictive of threat appraisals.

Acute hypothesis 8: Hardiness will be positively predictive of challenge appraisals.

Acute hypothesis 9: Stress will be positively related to physiological reactivity.

Acute hypothesis 10: Physiological reactivity (with the exception of SBP) will be negatively related to performance.

The hypothesized relationships are depicted in Figures 3 and 4.

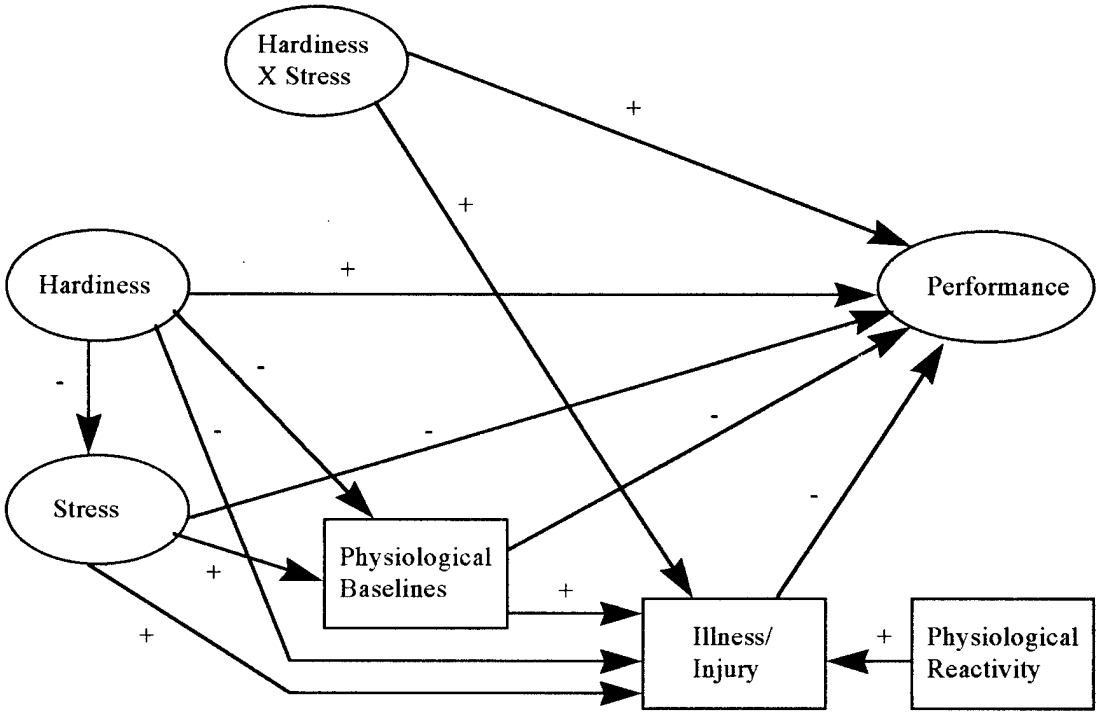

Figure 3. Hypothesized global and cross-over relationships, study 2.

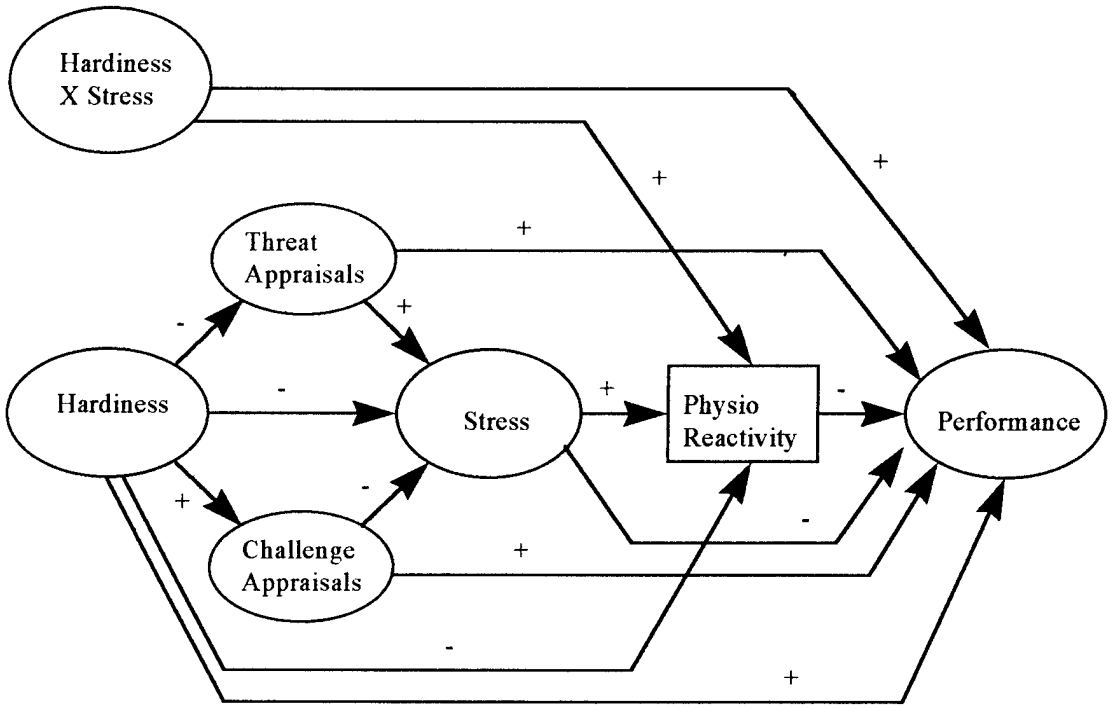

Figure 4. Hypothesized acute relationships, study 2.

Method

Participants

Initial participants, as designated in early July, 1996, included 31 active duty USA UH-1 helicopter pilots from an aeromedical evacuation company. Five of these participants became unavailable to participate in the study prior to the first round of data gathering. Three participants then withdrew from the study shortly after it began on July 31; two provided no reasons for withdrawal, while one stated official responsibilities did not allow sufficient time for participation. The remainder comprised a sample of 23 pilots, of which 22 were male. The pilots ranged in rank from warrant officer 1 (WO1) to captain. The mean age of the participants was 29.7 years (s.d.=2.07), and mean time on

active duty was 97.1 months (s.d.=41.9). Nineteen participants were married. Three regularly used tobacco. Mean education consisted of an associate's degree plus additional coursework towards a bachelor's degree (education ranged from less than two years of college coursework to a master's degree). Participants dwindled and surged throughout the course of the 9-month study (participants entered and departed the study on varied schedules due to their availability (14 participants averaged 50.6 days deployed to other geographic locations over a period of 6 months)); two participants were transferred to other units within 60 days of entering the study, and one separated from active duty approximately 120 days after entry. Finally, availability of simulator facilities and mission priorities limited the number of pilots contributing to data on acute relationships; only 10 pilots participated in the simulator scenario. The n on any given variable, therefore, ranged from 8 to 23.

Measures

Hardiness. As in study 1, Nowack's (1990) CHS was the instrument used to measure hardiness. The CHS has an established Cronbach's alpha of .84 and a unidimensional factor structure (Nowack, personal communication, February 1996). Test-retest reliability in this study was .89 over an average period of 172 days (n=14). Refer to Study 1 for more detailed information on the CHS.

Stress. Multiple measures of stress were used. As in study 1, the PSS (Cohen et al., 1983) was used as a global measure of perceived stress. The PSS has demonstrated a Cronbach's alpha of .84-.86 (Cohen et al., 1983) and has also demonstrated validity as a predictor of adverse stress-related outcomes (Hills & Norvell, 1991; Cohen et al., 1983).

Test-restest reliability for the PSS in this study over an average interim period of 172 days was .60 (n=14). Refer to Study 1 for more detailed information on the PSS.

In assessing global stress, the Life Experiences Survey (LES; Sarason et al., 1978) was also used (retrospectively) in this study (see Appendix E). The LES permits a respondent to not only record the occurrence of 47 various life events (10 items of the orginal 57-item instrument relating solely to academia were not used in this study), but also to rate their impact on a 7-point Likert scale ranging from "Extremely Negative" (-3) to "No Impact" (0) to "Extremely Positive" (+3). Accordingly, it provides not only the number of positive and negative incidents, but also a quantifiable measure of subjective negativity or positivity of life events. In scoring the LES, which requested participant information over the preceding 6 months, negative and positive incidents were totalled for negative and positive incident scores (variables LESNGIN and LESPSIN), respectively. The impact ratings were also totalled for negative and positive incidents to produce negative and positive change scores (variables LESNG and LESPS), respectively. Two modifications were made to the LES. All dollar figures in the original 1978 instrument were adjusted for inflation. Also, item 40 in the original instrument, "Retirement from work" (Sarason et al., 1978, p. 945), was not applicable to this sample. Required duty at other geographical locations (TDYs) was assessed instead by requesting participant response to "TDYs; please provide number of TDYs and total number of days on TDY in the last 6 months. # TDYs_____ # days total _____." The LES has demonstrated a test-retest reliability over a 6-week period of .53 for the positive change score and .88 for the negative change score (Sarason et al., 1978). The LES has also demonstrated

convergent validity with various measures of state and trait anxiety and academic performance; the negative change score has demonstrated the most robust relationships with trait and state anxiety (r=.29 and r=.46, respectively) (Sarason et al., 1978). Finally, the LES has been used successfully in past hardiness research (Banks & Gannon, 1988; Solano et al., 1993; Wiebe & McCallum, 1986).

Stress (variable PSTSTR) in the simulator scenario was assessed post-scenario via the 4-item stress subscale of Peacock and Wong's Stress Appraisal Measure (personal communication, August 1996; 1990) (see Appendix D). Wording was minimally tailored to the sample in this study, and phrased in the form of a statement to which participants responded via a 5-point Likert scale ranging from "Strongly Agree" (1) to "Strongly Disagree" (5). The stressfulness subscale of Peacock and Wong's (1990) instrument has an established Cronbach's alpha ranging from .75 to .81 (Peacock & Wong, 1990). This measure has also demonstrated strong correlations with stress-related outcomes such as psychological symptoms and dysphoric mood (Peacock & Wong, 1990). The stress score was calculated as the mean of the reverse scoring of all 4 items.

Illness/Injury. Similar to Funk and Houson's (1987) use of a health log, illness/injuries were tracked in this study with an illness/injury diary. The diary consisted of prepared sheets which participants completed and turned in every two weeks (see Appendix C); negative responses were required. While this is still a self-report measure (access to medical records was not permitted), it may well avoid some of the previously mentioned pitfalls arising out of the fallibility of memory. Also, the diary sheets required respondents to indicate whether or not the illness/injury was self- or physician-diagnosed.

While such a method is open to error, military aviators are regularly required to self-report illness and medication as well as seek the advice of, and examination by (when necessary), an assigned flight surgeon or specialist in aerospace medicine. Relatively common illnesses can be hazardous in flight duties; for example, congestion which does not permit one to valsalvo can lead to tremendous (and dangerously distracting) inner ear pain with rapid altitude change. That considered, it is believed that the responses provide an accurate depiction of health and illness/injury. Physician- versus self-diagnosis may also provide some insight into whether the report reflects illness behaviors or verified illness/injury. Participants were asked to "document any illness or injury if you believe it prevents you from performing your duty to your fullest potential or in some way interferes with your being able to do those things you wish to do in the way you'd like to do them." The diary sheets included spaces to provide information about the dates of the illness/injury, self- versus physician-diagnosis, diagnosis/description of the illness/injury, date(s) of missed duty, and dates of duty not including flying. The sheets were then examined for the number of days of illness (descriptions categorized as illness reflected viral/bacterial infections and allergies) and injury (descriptions categorized as injury most often reflected sports-related joint and muscle trauma/strain). Days of illness/injury and number of incidents were then divided by continuous days for which inputs were submitted since there was some variation in days for which diary sheets were provided (53-134 days; 16 of 21 respondents provided inputs for 71-89 days) due to transfer out of the unit, failure to follow instructions, and refusal (one participant) to provide illness/injury data. As a result, data was provided for self- and physician-diagnosed illness

rates (variables ILSDDYS, ILPDDYS), self- and physician-diagnosed illness incident rates (ILSINDY, ILPINDY), total illness rate (TOILDYS), total illness incident rate (TILINDY), self- and physician-diagnosed injury rates (INSDDYS, INPDDYS), self- and physician-diagnosed injury incident rates (INSINDY, INPINDY), total injury rate (TOINDYS), total injury incident rate (TININDY), total illness and injury rate (TODYS), and total illness and injury incident rate (TINDY). Such measurement of illness/injury is consistent with various long-established methods of measuring absenteeism in organizations (Mowday, Porter, & Steers, 1982).

Challenge Appraisals. Challenge appraisals were assessed both before and after the simulator scenario. Prior to the scenario, participants completed a 17-item questionnaire (see Appendix D), of which 5 statements (items 1, 4, 8, 13, and 15) assessed challenge appraisals. These statements were scored via a 5-point Likert scale ranging from "Strongly Agree" (1) to "Strongly Disagree" (5); all items are reverse scored. The five statements were prepared and tailored to the scenario according to the 4 key challenge appraisal phrases and their semantic content provided by Peacock and Wong (personal communication, August 1996; 1990) in the construction of their Stress Appraisal Measure (SAM). Two items (1 and 4) were prepared for Peacock and Wong's "eager to tackle" dimension (which had loaded the most heavily of their four challenge items on the controllable-by-self/challenge factor); omission of item 1 from the scoring yielded the most internally consistent instrument with a rather unimpressive Cronbach's alpha of .50. Comparatively, Cronbach alphas reported by Peacock and Wong (1990) for their challenge scale ranged from .66 to .79. While the selected wording in tailoring their

instrument to this sample may have weakened the instrument's internal consistency, group homogeneity may also have contributed to a low Cronbach's alpha value (Crocker & Algina, 1986). Peacock and Wong's challenge scale has been correlated in predicted directions with locus of control and dysphoric mood measures (Peacock & Wong, 1990). The pre-simulator challenge score (PRECHL) represents the mean of items 4, 8, 13, and 15.

Following the scenario, challenge (PSTCHL) was also assessed via Larsson's (1989) retrospective Emotional Stress Reaction Questionnaire (ESRQ). On the ESRQ, respondents rated various adjectives describing how they felt during what they perceived to be the most intense part of the scenario on a 4-point scale ranging from "the word does not describe how I felt" (1) to "the word completely corresponds to how I felt" (4) (see Appendix D). The ESRQ has been successfully used with military samples in past research and has demonstrated a Cronbach's alpha ranging from .66 to .81 (Larsson, 1989). The ESRQ, like the SAM discussed above, is theoretically grounded in the work of Lazarus and Folkman (1984). Items 5-8 of the ESRQ comprise the challenge subscale and are scored by totalling the scores on the four items.

Threat Appraisals. Threat appraisals were also assessed pre- and post-simulator. Five items on the 17-item pre-simulator questionnaire (2, 5, 7, 9, and 12) assessed threat and were based upon the key phrases and semantic content of Peacock and Wong's (personal communication, August 1996; 1990) SAM. As with the challenge items, the threat items were also tailored to the sample in this study, and 2 items (2 and 7) were prepared for the SAM threat dimension ("feel anxious") most heavily and singularly

loading on the threat/centrality factor (Peacock & Wong, 1990). Elimination of item 2 yielded the most internally consistent scale with a Cronbach's alpha of .72. This is comparable to Peacock and Wong's original threat appraisal scale which has demonstrated Cronbach alpha values ranging from .65 to .75. Peacock & Wong's threat scale has been strongly correlated in predicted directions with measures of psychological symptoms and dysphoric mood (Peacock & Wong, 1990). As with the challenge score, the pre-scenario threat appraisal score (PRETHR) represents the mean reverse scoring of 4 items (5, 7, 9, and 12).

The post-simulator threat appraisal (PSTTHR), like PSTCHL, was provided by the threat subscale of the ESRQ (Larsson, 1989). Again, the ESRQ has demonstrated Cronbach alphas ranging from .66 to .81 (Larsson, 1989). The threat appraisal score was calculated by summing the scores on items 9-14.

Centrality. Centrality, while not one of the prime variables mentioned in the hypotheses, is likely a key component in the primary appraisal process (Peacock & Wong, 1990), as earlier discussed in this paper; it has been included in the study to afford examination of its relationship with variables of interest. Centrality (PRECEN), like PRECHL and PRETHR, was measured prior to the simulator scenario via 5 items (3, 6, 10, 11, and 14) on the 17-item pre-simulator questionnaire (see Appendix D). The 5 items were constructed (tailored to this sample) on the basis of Peacock and Wong's (personal communication, August 1996; 1990) key phrases and semantic content for their 4-item centrality subscale of the SAM. Two items (3 and 6) corresponded to the "important consequences" dimension of the scale, which had most heavily loaded on to the threat/

centrality factor in their validation research. Note also that this factor loading may suggest a relationship between threat appraisals and centrality. Removal of item 3 produced the most internally consistent scale for this study with a Cronbach's alpha of .73. This is somewhat lower than the Cronbach alphas (.84-.90) obtained by Peacock and Wong (1990) for their original centrality scale, but again, homogeneity in this study's small sample may be responsible for depressed measures of internal consistency (Crocker & Algina, 1986). Peacock and Wong's centrality scale has also been correlated positively with psychological symptoms and dysphoric mood (Peacock & Wong, 1990). Finally, scoring of the centrality scale is accomplished by calculating the mean reverse scoring of items 6, 10, 11, and 14.

Controllability/Uncontrollability. While primary appraisal is the main focus of this study, it is exploratory in nature. Accordingly, 2 items based upon the semantic content of Peacock and Wong's (personal communication, august 1996; 1990) SAM controllable-by-self and uncontrollable scales were included to assess the secondary appraisal characteristics of controllability (PRECON) and uncontrollability (PRUNCN) of the pending scenario. The controllability item (item 16 in the pre-simulator questionnaire (see Appendix D)) requests response to the statement "I can handle anything and everything thrown at me." The uncontrollability item (item 17 in the pre-simulator questionnaire) elicits response to the statement "I know the limits of my abilities, and I am concerned the scenario might be designed so as to exceed my abilities and skills." Both statements are reverse scored.

Performance. Global performance was assessed in 2 ways. Participants' supervisors (primary raters) were asked to complete portions of Department of the Army Form 67-8, US Army Officer Evaluation Report (GLOPA) (this form is not included in the appendices; interested parties should contact the author), for a period covering 6 months. The 14 professional competence items (PROCOMP) (part IVa) were rated per the scale indicated on the form. The Cronbach's alpha for PROCOMP in this scale was an impressive .92. The 14 items were reverse scored and the mean of these reverse-scored items was recorded as the PROCOMP score. Performance during the rating period (PERFRAT) (part Vb) was scored on a 5-point scale ranging from "Always Exceeded Requirements" (5) to "Usually Failed Requirements" (1). The officer's potential for promotion to the next grade (OFFPOT) (part Vd) was scored on a 4-point scale ranging from "Promote Ahead of Contemporaries" (4) to "Other" (1). Finally, an evaluation of overall potential (POTEVAL) (part VIIa) was also completed by the immediate rater (not a senior rater as indicated on the form). POTEVAL was scored on a descending 9-point scale (from top 1% to bottom 1%) per the schematic on the form. The composite GLOPA was calculated by summing the scores of PROCOMP, PERFRAT, OFFPOT, and POTEVAL. GLOPA demonstrated a decent Cronbach's alpha of .87.

Global performance, as regards piloting skills only (GLPAPI), was evaluated by 4 members of the company's Standardization section. It is the responsibility of this section to "establish aviation standardization procedures, training programs, administrative procedures and evaluations of rated and non-rated aviators" (B. Thomas, personal communication, April 1997) within the company. The 4 evaluators rated the study's

participants over an 8-month period on a 10-point scale ranging from "superior performer; one of the very best I've seen; in a hazardous/combat scenario, I would want this person flying with me" (10) to "unsatisfactory performer; one of the worst I've seen; I am not comfortable flying with this person" (1). See Appendix F for the entire scale and rater instructions. Interrater reliability was a respectable .82.

Acute performance in the simulator scenario was also measured in two ways. Performance on various maneuvers and procedures (SPAPRO) was rated using the Department of the Army's "Maneuver/Procedure Gradeslip for UH-1H/V RCM" form (note: a copy of this form is not included in the appendices; interested parties should consult the author). Applicable procedures were rated by an evaluator from the Standardization section on a 10-point scale ranging from "best possible performance on this procedure" (10) to "average performance on this procedure" (5/6) to "worst possible performance on this procedure" (1). Procedures evaluated included crew mission briefing, IFR flight planning, fuel management procedures, simulated engine failure at altitude, manual throttle operations, emergency procedures, instrument takeoff, radio navigation, holding procedures, unusual attitude recovery, radio communication procedures, nonprecision and precision approaches, and simulated hydraulic malfunction (usually graded as part of emergency procedures). Emergency procedure (EP) was the most important individual maneuver/procedure for purposes of this study; the scoring of this procedure represents how successfully the pilot resolved the presented emergencies while maintaining applicable in-flight protocols. SPAPRO was scored as the mean of all graded procedures/maneuvers. In this study, SPAPRO demonstrated a Cronbach's alpha of .70.

Acute performance was also assessed by rating 13 "aircrew coordination basic qualities" (SPAACQ); the "Maneuver/Procedure Gradeslip for UH-1H/V RCM" was also used to record these evaluations. These qualities were crew climate, premission planning and rehearsal, decision-making techniques (DECTEC), action prioritization and workload management (WRKLD), management of unexpected events (UNEXEV), clarity and completeness of information transfer (INFOX), maintenance of situational awareness (SITAWR), communications and acknowledgement, information sought (INFOSOT), cross-monitoring of crew member actions (XMONITR), information offered (INFOOFR), advocacy and assertion (ADVASS), and conducting after-action reviews. Aircrew coordination is defined as "a set of principles, attitudes, procedures, and techniques that transforms individuals into an effective crew" (Department of the Army, 1995, p. 1-9); such aircrew coordination training is now mandatory in the USA. Aircrew coordination has also been recently (Kanki, 1996) hailed as a factor which may prevent aviation disasters and aid aviators in successfully managing such stressors as excessive workload, weather changes, limited information, inoperative systems, aspects of cockpit displays and warning systems, unexpected events, and various combinations of all of these.

Nine of the 13 aircrew coordination qualities were individually examined as part of this study; these are the qualities for which variable names have been provided above. While in-depth definitions are available (Department of the Army, 1995), a few comments on some of these are warranted. DECTEC involves rendering a solution to a problem and establishing a plan of action to implement that solution. It is inclusive of risk assessment, information availability and seeking, information sharing by crew members, problem

pattern-recognition to solve high stress/time critical problems, and recognition of the most critical factors in the problem. WRKLD involves the degree to which crew efforts are focused upon essential activities, effective distribution of task load among the crew, and appropriate identification and prioritization of competing tasks. UNEXEV reflects crew performance under unusual circumstances and high stress. Effective (active) coping is subsumed under this quality. Effective execution of this quality demands action and information exchange with minimal prompting from the other crew member as well as proactive prioritization of actions and distribution of work and information load. SITAWR involves crew awareness (and promotion of awareness in the other crew member) of aircraft and mission status, spatial orientation of the aircraft, environmental conditions, and active efforts to recognize and manage stress, boredom, fatigue, and anger. ADVASS reflects proactivity in advocating a correct course of action, even when the other crew member disagrees. This demands clear communication of rationale as well as good listening skills.

The SPAACQ score reflects the mean of the 13 crew coordination basic qualities as rated on a 10-point scale ranging from "best possible performance on this crew coordination quality" (10) to "average performance" (5-6) to "worst possible performance on this crew coordination quality" (1). Each participant in the scenario was designated the pilot-in-command (PC) for the simulation as evaluated by one of the Standardization section evaluators; accordingly, the PC is responsible for (and rated upon maintenance of) the state of crew coordination on his/her flight. SPAACQ yielded a Cronbach's alpha of .64 in this research effort.

Cardiovascular Function. HR, SBP, and DBP were measured using a modified SunMark™ 146 oscillometric electronic digital blood pressure and pulse monitor. A readily available device, the the SunMark™ 146 is accurate to within ± 3 mm Hg; it has performed well in trials with other methods and provides a rather inexpensive, yet sufficiently accurate, device for stress research (H. Erickson, personal communication, May 1996). Device modification consisted of a 7-foot extension of the cuff-inflation hose using Fisherbrand 3/16-inch internal diameter, 1/16-inch wall plastic tubing to permit constant wear and measurement in the simulator scenario. Measurement was not affected by the modification. Baseline and scenario measurements were taken on the left arm with participants in the sitting position; the modified device was used for all measurements.

Cortisol/Creatinine. Urinary cortisol has been shown to mirror serum cortisol levels (French, 1997) and has been successfully assayed in past stress research (Cummins & Gevirtz, 1992; Pollard, Ungpakorn, & Harrison, 1992). Urinary sampling and assay for urinary free (unbound/bioactive) cortisol was chosen over salivary sampling due to resource limitations and the desire to assess cortisol secretion over a period of time (simulator scenario). Venipuncture was also avoided over concerns it may confound sampling if perceived as a stressor (Kirschbaum & Hellhammer, 1994). Urine was collected in 4-ounce specimen collection cups, transferred to 4-ounce light-proof plastic storage bottles, and frozen to -20° C until analyzed with the Abbott TDx/TDxFLx cortisol assay, a fluorescence polarization immunoassay (FPIA) (Abbott Laboratories, 1994). FPIA is a competitive binding immunoassay in which fluorescin-labeled cortisol and sample cortisol compete for binding sites on antibody molecules . Bound tracer

(fluorescin-labeled cortisol) emits polarized light when excited by polarized light while unbound tracer does not. Polarized light emissions are inversely proportional, then, to sample cortisol concentration. Polarization values are used in conjunction with a stored calibration curve to calculate the sample concentration of urinary free cortisol. FPIA shows minimal cross-reactivity with endogenous and synthetic steroids (with the exception of a 27% cross-reactivity with prednisolone), and is 95% accurate to $\pm$ .61 μg/dL at concentrations of 4.0 μg/dL and $\pm$ .76 μg/dL at sample concentrations of 15.0 μg/dL (Abbott Laboratories, 1994).

Urine was also assayed for creatinine. This end product of creatine catabolism is a reliable indicator of urine flow rate. Used in ratio with urinary cortisol, it controls for differences in urine production and concentration (P. Elmore, personal communication, June 1996; French, 1997), although several researchers have found it unnecessary to correct for flow rate or urinary concentration (Cummins & Gevirtz, 1992; Pollard et al., 1992). Creatinine was assayed using a rate-blanked kinetic reaction; in alkaline medium, creatinine and picric acid form a yellow-orange complex which can be measured kinetically at a wavelength of 505 nm. The rate of color development is directly proportional to creatinine concentration (Immunochemistry Section, Brooke Army Medical Center, 1997). Neither cortisol nor creatinine have a single specimen "panic value." All biochemical analyses were conducted by the Immunochemistry Section, Department of Pathology and Area Lab Services, Brooke Army Medical Center, Fort Sam Houston, Texas.

Demographic Information. Participants were asked to provide social security numbers and were assigned a participant number to aid tracking of measures and samples. Participants also provided information on rank, gender (GNDR; female=0, male=1), age, time on active duty (TIMEAD), time in the unit (TIMUNIT), hours of flight experience in all aircraft (FLYALL), hours of flight experience in the UH-1 (FLYUH1), hours of experience in all aircraft simulators (SIMALL), and hours of experience in the UH-1 simulator (SIMUH1). These measures of flight and simulator experience were taken twice in the study over an average interim period of 172 days. Participants also provided information on education level (on a scale ranging from high school (1) to doctoral degree (9)), marital status (married=1, single=0), and whether they regularly used tobacco (yes=1, no=0) since tobacco use may affect cortisol measurement in some individuals (Kirschbaum & Hellhammer, 1994). Initially, the attempt was made to track caffeine consumption, but this was abandoned shortly after study initiation due to an inability to accurately assess intake. A large number of participants also refused to cooperate with instructions to abstain from caffeine consumption at certain times in the study. Zorilla et al. (1995), however, did not find caffeine intake to be related to cortisol secretion. Throughout the study, participants were also asked to provide information on drug ingestion (to eliminate confounding) and the presence of stressors preceding certain measures. Again, this was to avoid confounded measures to the extent possible in the research setting. Finally, participants were asked whether they had been diagnosed with hepatitis B (within the preceding 12 months) or PTSD; both disorders would have disqualified participants from the study due to concerns over handling of infectious waste

and disturbed HPA regulation. The demographic information was gathered via the instruments in Appendices B and E.

Procedures.

Before describing the procedures in this study, it is important that I clarify a few important issues. The procedures will be described in terms of what was done at time 1, time 2, and so on. While these designations are, indeed, primarily chronological, this is not always the case. As a result of resource and participant availability, different participants progressed through the study on slightly varied schedules. The most unstable scheduling was for participation in the simulator scenario. Participants engaged in the simulation anywhere from 16-232 days after entry into the study. For this reason, you'll note that some measurements and demographic data were gathered at two times during the study (see Appendices B and E), an average of 172 days apart, to guage any prominent changes. One participant demonstrated profound change and instability on various measures; that participant was dropped from consideration in all performance and illness/injury outcomes as a result. This participant, however, is an important and informative outlier whose case will be later discussed.

Time 1. Participants provided informed consent and received guarantees of confidentiality. General information about the pending study was provided. Participants were provided with and completed the initial questionnaire (see Appendix B), to include provision of demographic information, the CHS, and the PSS. Envelopes were provided for the return of the questionnaires either directly or via the Standardization section (members of this section were instructed not to open any of the envelopes; they only

collected submitted materials for delivery). Illness/injury diary instructions were also provided along with 20 diary sheets (see Appendix C) and 6 envelopes in which the diary sheets were to be turned in bi-weekly. Diary turn-in dates were adjusted for individuals as they entered the study. The Standardization section collected the sealed, unmarked envelopes. Participants were required to make negative responses (as necessary) to both provide information there was no illness or injury, and also to protect confidentiality (the Standardization staff would be unable to determine who had made positive or negative responses).

Times 2-4. Measurements of HR, SBP, DBP, and urine samples were collected between 0800 and 1000 hours for purposes of standardization and to control for the diurnal rhythm of cortisol secretion (Whitnall, 1993). At this time of day, cortisol secretion should be just post-peak and declining, although there is some evidence that the cortisol peak occurs earlier (phase shift) in those engaged in physical training (Wittert, Livesey, Espiner, & Donald, 1996); the participants in this study were required to engage in at least three physical training sessions per week as part of military training. Measurements were taken only on duty mornings in which the participants had engaged in no physical activity (which can increase cortisol secretion (Kirschbaum & Hellhammer, 1994)).

While some researchers have implied baseline measurements for cortisol be taken in the afternoon (reactivity may be greater near secretion nadir, and changes from near nadir when exposed to a stressor may be easier to detect) (Uchino, Cacioppo, & Kiecolt-Glaser, 1996), the decision was made to take morning measures for three reasons. Firstly,

there is too little research to be certain about whether reactivity can be detected when compared against a morning baseline; indeed, morning cortisol elevations (compared to morning baselines) were observed in 18 surgeons the morning of a planned surgical procedure (Jezova, Slezak, Alexandrova, Motovska, Jurankova, Vigas, & Cerny, 1992). Secondly, it was believed that morning measurements might eliminate confounding (for both cardiovascular and cortisol measures) from workplace stressors and reactions to those stressors which may arise and accumulate over the course of the workday. Participants were asked at the time of measurement if they had recently experienced stressful events which might confound the measures. Thirdly, and perhaps most profoundly, using a nadir baseline assumes directionality of acute reaction and may well contribute to the previously discussed preponderance of findings for cortisol elevation in response to stress. Using a more elevated baseline, unlike measurement at nadir, allows for the possibility of detecting cortisol decrease or blunted responses (as Sapolsky (1992b) and Blanchard et al. (1993) have observed in animal models), and is more consistent with what appears to be a biphasic function (Chrousos & Gold, 1992).

The measures taken at times 2 and 3 were averaged to produce baseline values for HR (BLHR), SBP (BLSBP), DBP (BLDBP), cortisol (in μg/dL) (BLCORT), and cortisol in ratio with creatinine (in μg cortisol / mg creatinine X 10^{-2}) (BLCOCR). If these values differed (on any measure) by more than 10% of the smaller value, a third measure (of both cardiovascular baselines and urinary cortisol) was taken (time 4) as

possible. Seven participants were required to provide a third (time 4) measure of HR, SBP, DBP, and a urine sample for the purpose of calculating baseline values.

Time 5. Time 5 constitutes the simulator scenario and related data gathering. For the simulator scenario, participants were chosen as available. Individuals were identified and informed of their selection by the Standardization staff within 24 hours of the scenario. Examination of demographic data and time 1 measures revealed the participants so selected for the simulator evaluation represented a diverse sampling from the 23 pilots. Participants, a non-evaluated crew member, and an evaluating Standardization staff member were joined during the pre-flight planning stage. Following the pre-flight planning, participants were asked to complete the 17-item pre-simulator questionnaire, which included items assessing PRECHL, PRETHR, PRECEN, PRECON, and PRUNCN (see Appendix D). Participants were also asked to void their bladder if they had not done so in the preceding 30 minutes; all participants did elect to void their bladder. Participants were then led into the simulator chamber for the scenario.

Participants and their non-evaluated crew member then took their positions in the simulator cockpit. The simulator used was a Singer Link 2B24 IFR UH-1 Flight Training Simulator. The evaluator remained at the control console with the on-duty technician. From here the evaluator could monitor communications, coordinate inputs with the technician per the pre-planned scenario, and evaluate the PC's performance. Cameras in the simulator also afforded the evaluator visual information, if desired. The experimenter was seated in a third seat behind both crew members; crew members in the UH-1 are seated side-by-side. From this position, the crew members, all displays, aircraft controls,

and communications could be observed; qualitative notes were recorded. Opportunity to freeze the simulation was also afforded from this position (a button located on a central control panel permitted motion to be immediately frozen) .

The crew was permitted to complete all pre-flight checklists/preparations. When done, the blood pressure cuff was placed upon the PC's left arm, and the inflation hose routed up the PC's arm and over the back of the seat to the SunMark^{TM} 146's console/display. The PC was given the following instructions:

> "Momentarily, I will take a blood pressure and heart rate reading. Also, several times during this scenario, I will freeze the simulation and measure your blood pressure and heart rate. You may feel the cuff inflating before I freeze the simulation. When I have frozen the simulation, place your left hand on your left knee until I have told you I am done. Please remain motionless while your heart rate and blood pressure are being measured. You may continue to mentally problem-solve while the simulation is frozen. When I have told you I am done, you may wish to squeeze the cuff to help expel all the air. You will then be told to continue the scenario."

Once the PC acknowledged he had understood the instructions, an initial measurement was taken. The scenario then began with clearance and take-off.

All scenarios were conducted in simulated airspace with which the pilots were familiar (northeast Kansas or airspace in the vicinity of Fort Rucker, Alabama). The scenarios were divided roughly into 4 segments. The first consisted of becoming airborne and initiating the flight plan. This was followed by the first emergency; engine malfunction/failure or tail rotor malfunction were the situations used. The third segment consisted of flying a less than fully operative aircraft (although eventual resolution of the first emergency was permitted in all cases) and enduring many heading changes and "re-directs" to alternate landing sites. The flight ended with a second emergency; a major

hydraulic malfunction/cyclic cardover was the scenario used. Throughout the flight, the PC had to deal with various major and minor irritants; these included such things as icing, air turbulence, "throwing" of various circuit breakers, loss of attitude and glide slope indicators, loss of fuel guages, governor high/low side malfunction, failure of the main generator, failure of the main invertor, and fuel boost pump failure.

Cardiovascular measures were taken approximately 30 seconds after initiation of the first emergency, during a fairly routine task (for example, changing heading) in the third segment, and approximately 30 seconds after initiation of the final emergency scenario. The performance in the scenario was graded by the evaluator, providing SPAPRO, SPAACQ, and various subscale scores. Scenarios lasted an average of 74.9 minutes. Various measures of cardiovascular tone and reactivity were then calculated, to include average HR, SBP, and DBP during the scenario (SHRAV, SSBPAV, SDBPAV, respectively), average HR, SBP, and DBP change from baseline as a raw value and as a percentage (HRCHRW, HRCHPC, SBPCRW, SBPCPC, DBPCRW, DBPCPC), maximum HR, SBP, and DBP during the scenario (SHRMX, SSBPMX, SDBPMX, respectively), and maximum HR, SBP, and DBP change from baseline as a raw value and as a percentage (HRCRX, HRCPX, SBPCRX, SBPCPX, DBPCRX, DBPCPX).

Following the scenario, the PC was asked to complete the 14-item post-simulator questionnaire (ESRQ) which included the PSTCHL and PSTTHR subscales. Also, the participant completed the 4-item stress scale (PSTSTR). When these two questionnaires were completed, the participant provided a urine sample. From this sample, various measures of cortisol secretion and reactivity were calculated, to include adjusted (in ratio

with creatinine) and raw cortisol secretion (SCOCR and SCORT, respectively), raw change in SCOCR and SCORT (SCOCRR, SCORTR), magnitude of raw change in SCOCR and SCORT (COCRRM, CORTRM), percentage of change in SCOCR and SCORT (SCOCRP, SCORTP), and magnitude of percentage change in SCOCR and SCORT (COCRPM, CORTPM).

Time 6. Participants completed a final package of demographic information and questionnaires (see Appendix E) at an average of 172 days after entry into the study. Questionnaires included a retaking of the CHS and PSS. Also, participants completed the LES at this time. All participants were provided envelopes; sealed responses were returned directly or via the Standardization section.

Time 7. In January-March 1997, primary raters were asked to complete Department of the Army Form 67-8 (GLOPA) on the various participants in the study. Self-addressed, stamped envelopes were provided for return of this measure. Also, in April 1997, four staff members of the Standardization section rated participants' overall performance as a pilot (GLPAPI) in the preceding 8 months. These ratings were collected directly from the staff members.

During the course of the study, all participants were debriefed on the portion of the study which they had completed. Participants were treated in strict accordance with the APA's current principles of ethical conduct.

Statistical Analyses

As in study 1, regression analyses were selected as an appropriate approach in the analysis of most of the collected data. Canonical correlation was also used in examining

cross-over effects. I will not here discuss the general considerations in regression analyses that were provided in the Study 1 "method" section. Refer to Study 1 for such information. There were, however, some unique and challenging considerations given the nature of this study and its small sample size. First, and foremost, the ratio of cases to IVs in this effort is less than that recommended by Tabachnick and Fidell (1996). Nonetheless, the effort was made to ferret out the most robust predictors for various outcomes and so maximize the cases to IV ratio. Also, as Achen (1982) states, "...the strength of ordinary regression is its great resilience...if the researcher sets up the problem correctly, regression will tend to the right answer under any reasonable, practical circumstances, even if a great many of the classical postulates are violated" (p. 37).

One of the most powerful tools available when it is not possible to obtain as many cases as desired is setwise regression (Tabachnick & Fidell, 1996). Accordingly, and consistent with an exploratory approach, setwise regression was used to reveal those IVs which are most informative. As a result, I will address only the most robust relationships in testing the hypotheses stated earlier. I cannot overemphasize this point; certainly, there are IVs and DVs that demonstrate substantial bivariate relationships, but many of these will not be among the most powerful predictors given the setwise approach and the need to maximize, as possible, the cases to IV ratio.

Finally, with small sample sizes, it is realized that influential outliers may drastically alter a solution (Fox, 1991). In consideration of this, and also cognizant of the potential contributory value of each and every observation, extensive use was made of various plots to corroborate statistical indications. Also, many data transformations were used in this

study. Because it is difficult to assess trends and non-linearity with so few observations, purposeful transformations were only made in those cases where the need for transformation was grossly apparent (or initially believed to be so).

Results

The results in this study are many and complex; I will freely use the variable names which have previously been stated in the interest of brevity and clarity. A reference listing of variable names, their brief definition, and calculation of some terms (especially in the case of transformed variables) has been provided in Appendix H. Also, given the exploratory nature of this effort and its small sample size, a liberal α value of .1 was employed in examining the results of statistical analyses. Such a liberal α value was selected to better reveal relationships which may warrant consideration and/or future investigative effort.

As in Study 1, descriptive analyses were conducted to ensure correct data entry and examine initial means, standard deviations, and intercorrelations among the variables. Following examination of these statistics, various methods were applied in screening the data and identifying disproportionately influential and/or aberrant observations. A number of methods were used, including extensive univariate analyses (skewness, kurtosis, tests of normality using the Shapiro-Wilk statistic (SAS Institute, 1989), stem-and-leaf plots, box plots, and normal probability plots), extensive examination of scatterplots, residual plots (following identification of promising models via setwise regression), calculated raw and studentized residuals, studentized residual plots, construction of 95% CIs around predicted values for DVs in hypothesized relationships, and multiple influence diagnostics

examining the relationship of predictors and the various DVs (Cook's D, HAT DIAG, DFBETAS, DFFITS, and partial regression leverage plots) (Tabachnick & Fidell, 1996, Loughin, personal communication, July 1996; Fox, 1991; SAS Institute, 1989, 1985).

In the global relationships, some outliers and unexpected observations did emerge. One individual demonstrated an extremely high value for BLCOCR (+4.13 s.d.); additionally, in at least one urine sample, this individual's cortisol concentration exceeded the linear range of the assay and required dilution for further analysis. As a result, this individual's BLCOCR and BLCORT values were dropped from the analysis of global relationships.

Another individual appeared to have experienced a tremendous number of both negative and positive life changes (LESNGIN and LESPSIN). Values for LESNGIN, LESPSIN, LESNG, and LESPS were all appreciable outliers (+6.71 s.d., +8.09 s.d., +6.59 s.d., and +5.81 s.d., respectively). Also, over the course of 176 days, this individual's score on the CHS dropped from 135 (+1.11 s.d.) to 94 (-1.60 s.d.), and the score on the PSS rose from 24 (-1.22 s.d.) to 42 (+1.82 s.d.)! Much of this individual's data was discarded because of the apparent instablility of the predictors and outcomes; it is not possible to determine which IVs were related (and how they were related) to specific outcomes at various times. Data on the initial measures of hardiness and stress (PSS) and baseline physiological measures were maintained in the analysis since they were all taken within a very short time (3 days) upon entry into the study and can be assumed to be relatively stable. As previously mentioned, this individual's case will be discussed later.

Again, two individuals left the study within 60 days of entry as a result of being transferred to another unit. They were not included in the global performance appraisals (GLOPA and GLPAPI) since they were present for so little of the rating period, and there was a concern that any evaluations would be grossly biased. Indeed, some of the raters in the two measures expressed discomfort at the thought of attempting to rate these two, and were unable to recall specific behaviors. The decision was made to include the individual who had separated from active duty at approximately 120 days into the study; this individual was present for greater than half of the respective rating periods. Also, qualitatively, raters did not express difficulty recalling this individual's duty behaviors. Notably, these differences in recalling work behavior do not appear to be associated with differential levels of ratee performance ("halo" effect confounding did not appear to be a concern).

The raw and adjusted values for cortisol (BLCORT and BLCOCR, respectively, were highly correlated ($r=.71$, $n=22$, $p<.0005$). This suggests it was not necessary to control for urine flow rates/concentration. Nonetheless, the correlation is not perfect, suggesting some difference (this was seen also in the acute relationships and will be addressed later). While BLCOCR adjusts for concentration, it is a reality that creatinine production is not consistent across individuals (for example, in males, the expected range is from 1000-2000 mg/24 hours (Immunochemistry Section, Brooke Army Medical Center, 1997)). Thus, the additional assay and range fluctuation may introduce error as it is used to adjust BLCORT values. Caught in such a quandary, both values were maintained in the analyses. Means, standard deviations, and intercorrelations for the

global variables are presented in Table G1. It should be noted that the size of the correlation matrix demanded that not all variables be presented. Only those variables demonstrating strong or theoretically interesting relationships were included; variables not significantly related to any other variables were not included in the correlation matrix.

In the acute relationships, 3 variables emerged as being singularly influential; they were all highly discrepant as revealed by numerous scatterplots and descriptive statistics (influence diagnostics and bivariate correlations). One individual's SITAWR score (also an outlier at -3.43 s.d.), another's DECTEC rating, and a third's PRECHL score were, thus, dropped from the amassed data for each of the respective individuals. All other data was included in the study.

For the acute relationships, the SCOCR and SCORT measurements were also highly correlated ($r=.84$, $n=10$, $p<.005$). This again suggests it is not necessary to control for urine flow rates and concentrations. However, the imperfection of the relationship and examination of various scatterplots pairing measures of reactivity suggested that 2-3 observations likely differed in urinary concentration (hyperhydration was suggested in 2 individuals) from the others. Again, as with the global measures, the decision was made to maintain both measures in the analyses. Later regressions would indeed reveal that unique contributions to explained variance were made by adjusted and raw values.

A potential confound in the acute relationships also demanded examination. Because of resource availability, the simulator scenarios were not all completed at the same time. Collection of post-simulator urine samples, therefore, ranged from 0935 to 1510 hours (9 were collected between 1145 and 1510 hours). Given the diurnal rhythm of

cortisol secretion, then, systematic variation with time per expected curves would necessarily need to be controlled. However, neither SCOCR nor SCORT values were correlated with time ($r=.23$, $n=10$, n.s. and $r=.12$, $n=10$, n.s., respectively). It would appear, then, that SCOCR and SCORT values are primarily a product of the stress-inducing scenario.

Of course, one cannot assume the simulator scenario was stress-inducing. However, examination of maximal changes in HR and SBP did suggest this was the case. T-tests revealed a significant increase in HR ($t(18)=-2.84$, $p<.05$) and SBP ($t(18)=-3.61$, $p<.005$) during the scenario. Significant results were not obtained for cortisol secretion; this occurred as a result of individuals demonstrating both increases and decreases in cortisol secretion as a response to the simulator scenario. Additionally, the scenario was believed stressful because it was evaluated and because the scenario had been designed to be more taxing than ordinary simulations, yet not so intense that successful outcomes were impossible. In other words, the simulation was designed with the help of the Standardization section to be extraordinarily demanding, yet all problems were, if coped with correctly and actively, solvable. Participants also made many comments during the flight which suggested it was stress-inducing. These included the following:

"We're dying."
"What the hell?"
"Shit!"
"We've never had so many EPs!"
"We're screwing this up!"
"I knew this would be a bad flight."
"Everything's gone to hell in a handbasket!"
"This sucks!"

Finally, various other behaviors indicated the scenarios were stressful. Many will be discussed later, but suffice it to say landing approaches were missed, dysfunctional displays were not noticed, authorized flight levels were violated for reasons other than what the emergency should have caused, and several pilots flew in the wrong direction for several minutes (surprisingly, correct headings had been acknowledged; the error was not one of miscommunication with the air traffic controllers). One crew did "crash" the simulator in an attempted landing at the end of the scenario.

The means, standard deviations, and intercorrelations between acute and cross-over variables are shown in Table G2. Again, as with the global variables, not all possible variables are included. As is, the correlations constitute an 86 X 86 matrix. Those variables showing no relationships to other variables are not included. Many data transformations have been included in the matrix to enhance appreciation of the effects of using data transformations to linearize curvilinear relationships. Again, consult Appendix H for brief definitions of the variable names and information on calculation of the various transformed variables.

Setwise regression analyses, again, were used to identify those solutions which were most promising in explaining the variance in the DVs of interest per the hypotheses. As in Study1, optimal models were selected per theoretical considerations in conjunction with examination of 5 criteria (R^2, adjusted R^2, Mallows' C_p, AIC, and MSE). Refer to Study 1 for discussion of these criteria. Potential models were then subjected to scrutiny to determine whether assumptions of residual normality, linearity, and homoscedasticity had been met. All residual plots indicated there were no substantial departures from

normality in residual distributions, although the residuals for GLOPA and GLPAPI were somewhat negatively skewed and those for SDBPMX showed a slight positive skew.

In general, non-linearity and heteroscedasticity (sometimes severe) were observed in the residuals for illness/injury variables. Attempts to transform the data in accordance with the guidance offered by Ott (1993) and Fox (1991) were unsuccessful. While such violations do not invalidate regression analyses, they do, however, weaken them (Tabachnick & Fidell, 1996). Therefore, as one considers those findings in which the DV of interest is illness/injury, one should consider that the demonstrated relationship is the result of analyses in which the assumptions of linearity and homoscedasticity have been, collectively, threatened. One should also consider the strength of the relationships that have been uncovered despite such weakening of the analytical technique. Other than these instances, residual plots revealed no substantial deviations from assumptions of linearity and homoscedasticity.

Univariate analyses revealed that hardiness, PSS, LESNGIN, LESPSIN, LESPS, BLHR, BLSBP, BLDBP, BLCOCR, BLCORT, GLPAPI, SIMALL, SIMUH1, and TIMEAD were normally distributed among the global variables per a conservative α of .01 for the Shapiro-Wilk statistic. Among the acute variables, all but SCORTR, CORTRM, INFOX, and INFOOFR were normally distributed per the same criterion. While regression analyses require no assumptions be made about variable distributions, Tabachnick and Fidell (1996) note that regression equations are often enhanced by normally distributed IVs. To the extent this is true in any given case presented in the

forthcoming pages, the solutions can reasonably be expected to be enhanced by inclusion of these normally distributed variables.

In the regressions that follow, and throughout the remainder of this section, it should be noted that singularity never presented a problem. Also, the threat of multicollinearity was minimal throughout. Accordingly, the issue of multicollinearity will be addressed only as regards those regressions in which it is a pertinent factor and should be considered by the reader in the interpretation of presented analyses.

Global hypothesis 1 was first tested. GLOPA was best predicted by a combination of LESNGIN, CHLNGIN (hardiness X LESNGIN), and CHLNG (hardiness X LESNG) ($R=.56$, $F(3, 10)=1.48$, n.s.). Only CHLNG ($\beta=-1.45$) was significant (for H_0: $B=0$, $t(13)=-2.07$, $p<.1$), however. Algebraic substitution similar to that demonstrated in Study 1 suggests that as subjectively negative change (LESNG) increases, the advantageous moderating effect of hardiness decreases. Hardy individuals, nonetheless, still perform better (higher GLOPA) than less hardy individuals until subjectively negative change (LESNG) becomes quite profound (> 13, a score +2.37 s.d. above the mean in this study). It should be noted that multicollinearity was a minor concern in this problem, although the solution does not appear to be ill-conditioned; of course, there was some correlation between the LESNGIN term and the interaction term to which it contributed. The solution's fourth principal component explained a large proportion of the variance in both LESNGIN and CHLNGIN.

The components of GLOPA were next examined. PROCOMP was best predicted by INPDDYS and CHPSS1 (hardiness X PSS (time 1)) ($R=.69$, $F(2, 16)=7.37$, $p<.01$).

Only INPDDYS (β=-0.57), however, was statistically significant (for H_0: B=0, t(19)=-3.11, p<.01). PERFRAT was best predicted by ILPDDYS and INPDDYS (R=.61, F(2, 16)=4.78, p<.05) (see Table 4). ILPDDYS emerged as being the predictor most strongly related to OFFPOT (see Table G1) (r=-.41, p<.1), and POTEVAL was best predicted by ILPINDY and SIMUH1 (R=.64, F(2, 15)=5.11, p<.05), although neither IV was individually predictive in the presence of the other.

Table 4

Results of Regressing PERFRAT on to ILPDDYS and INPDDYS (N=19).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
Intercept	4.878	0.0	29.130	.0001	
ILPDDYS	-11.296	-0.409	-2.066	.0554	.17
INPDDYS	-3.604	-0.467	-2.362	.0312	.22
R^2=.37				Unique variability=.39*	
Adjusted R^2=.30				Shared variability=.00	

*Note: Unique variability exceeds R^2 due to rounding.

Global performance as a pilot only was predicted by CHLNG (hardiness X LESNG) and education (EDUC) (R=.78, F(2, 10)=7.84, p<.01) (see Table 5). As with the findings for GLOPA, algebraic substitution revealed that as LESNG increases, the buffering effect of hardiness becomes less advantageous. With an LESNG score greater than 4 (just above the LESNG mean of 3.43, s.d.=4.03), the hardy individual no longer performs better than the less hardy individual. Thus, global hypothesis 1 is supported,

though rather weakly. The hypothesis is not supported for the various components of GLOPA, as they, in general, are better predicted by physician-diagnosed illness and injury.

Table 5

Results of Regressing GLPAPI on to CHLNG and EDUC (N=13).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
Intercept	9.304	0.0	10.773	.0001	
CHLNG	-0.002	-0.491	-2.229	.0499	.19
EDUC	-0.480	-0.428	-1.944	.0805	.15
R^2=.61				Unique variability=.34	
Adjusted R^2=.53				Shared variability=.27	

Per the analyses above, hardiness never emerged as a direct predictor of performance in the presence of other IVs; hardiness was significantly correlated with OFFPOT, however (r=.38, N=20, p<.1). That is, hardiness explained about 14% of the variance in OFFPOT. Mediated relationships were not supported. Therefore, global hypothesis 2 is partially supported (hardiness was predictive of OFFPOT), though rather weakly.

Global hypothesis 3 was next investigated. Both ILPDDYS and ILPINDY were predicted by LESNGIN, CHLNGIN (hardiness X LESNGIN), and CHLNG (hardiness X LESNG) (R=.92, F(3, 10)=19.06, p<.0005, and R=.91, F(3, 10)=15.42, p<.0005, respectively) (see Table 6 for the ILPDDYS solution). The effects of CHLNGIN on ILPDDYS were specifically examined via algebraic substitution. As negative life incidents increase, the physician-diagnosed illness rate increases for both high and low hardy

individuals. However, what is revealed is that the increase in illness rate is 3.25 times higher for low hardy individuals than for high hardy individuals per unit increase in negative incidents! The relationship between ILPINDY and CHLNGIN is similar.

Table 6

Results of Regressing ILPDDYS on to LESNGIN, CHLNGIN, and CHLNG (N=14).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
Intercept	0.004	0.0	0.730	.4822	
LESNGIN	0.071	5.665	5.776	.0002	.50
CHLNGIN	-0.000	-6.151	-5.301	.0003	.42
CHLNG	0.000	1.272	3.933	.0028	.23

R^2=.85
Adjusted R^2=.81

Note: Unique and shared variability are not provided in this table, as they total to greater than .85, indicating an extreme circumstance (Tabachnick & Fidell, 1996). High values of t for H_0: B=0 and low df_{res} contribute to such a situation.

As with the regression of GLOPA on to these variables, multicollinearity was a minor concern. In the regression of ILPDDYS, however, each of the variables demonstrated relatively greater orthogonality, and explained substantially unique portions of the variance in ILPDDYS.

Self-diagnosed injury incident rate (INSINDY) was regressed on to PSS, LESPSIN, BLCORT, and CHLPS (hardiness X LESPS) per the results of setwise analyses (R=.87, F(4, 9)=7.091, p<.01). All IVs other than BLCORT were predictive of INSINDY (see Table 7). Interestingly, the interaction term suggests that as LESPS increases, hardier individuals are likely to have a higher self-diagnosed injury incident rate

than low hardy individuals. Hardiness and LESPS alone are both negatively related to INSINDY, however (see Table G1).

Table 7

Results of Regressing INSINDY on to PSS, LESPSIN, BLCORT, and CHLPS (N=14).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
Intercept	-0.011	0.0	-1.634	.1366	
PSS	0.001	0.646	3.412	.0077	.31
LESPSIN	-0.007	-2.030	-3.520	.0065	.33
BLCORT	-0.000	-0.288	-1.456	.1793	.06
CHLPS	0.000	1.620	2.742	.0228	.20

R^2=.76
Adjusted R^2=.65

Note: Unique and shared variability are not provided in this table, as they total to greater than .76, indicating an extreme circumstance (Tabachnick & Fidell, 1996).

Finally, TILINDY was predicted by LESNGIN and CHLNGIN (hardiness X LESNGIN) (R=.78, F(2, 11)=8.74, p<.01) (see Table 8). As negative life events increase, hardy individuals show a slight decrease in total illness incident rate while low hardy individuals demonstrate an increase in TILINDY that is eightfold the magnitude of the decrease in the hardiest individuals!

In general, hypothesis 3 appears to be supported; hardiness boldly moderates the relationship between stress and physician-diagnosed (but not self-diagnosed) illness and illness incident rates. This effect also appears for the total illness incident rate (which is inclusive of the physician-diagnosed illness incident rate). A rather interesting effect was found for the way in which hardiness appears to interact with subjectively positive change

to moderate the relationship between positive change and INSINDY; this finding will be discussed later.

Table 8

Results of Regressing TILINDY on to LESNGIN and CHLNGIN (N=14).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
Intercept	0.003	0.0	0.460	.6546	
LESNGIN	0.035	4.111	3.268	.0075	.38
CHLNGIN	-0.000	-3.577	-2.843	.0160	.28
R^2=.61					
Adjusted R^2=.54					

Note: Unique and shared variability are not provided in this table, as they total to greater than .61, indicating an extreme circumstance (Tabachnick & Fidell, 1996).

Global hypothesis 4 was next tested. Only ILSINDY was predicted by hardiness in the presence of other variables; hardiness and tobacco use were the two predictors that emerged to explain variability in ILSINDY (R=.29, F(2, 18)=3.69, p<.05) (see Table 9). Hardiness and BLSBP were predictive of total illness and injury incident rate (TINDY) (R=.53, F(2, 18)=3.50, p<.1), although only BLSBP was significantly predictive (for H_0: B=0, t(20)=-2.053, p<.1). Mediated relationships were not supported. A review of the correlation matrix (Table G1) reveals that hardiness is negatively related to (predictive of) ILPDDYS (r=-.40, p<.1), ILPINDY (r=-.38, p<.1), and TILINDY (r=-.40, p<.1) when not in the presence of other variables. Hypothesis 4 is not supported, although relatively weak direct relationships between hardiness and illness were discovered.

Table 9

Results of Regressing ILSINDY on to hardiness and tobacco use (N=21).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
Intercept	0.066	0.0	1.984	.0627	
Hardiness	-0.001	-0.382	-1.923	.0704	.15
Tobacco	0.016	0.402	2.022	.0583	.16

R^2=.29
Adjusted R^2=.21

Note: Unique and shared variability are not provided in this table, as they total to greater than .29, indicating an extreme circumstance (Tabachnick & Fidell, 1996).

Examination of the correlation matrix (Table G1) reveals that hardiness is strongly related to perceived stress (r=-.77, p<.001), but not to any of the other measures of stress; hypothesis 5 is supported. Hardiness is non-significantly related to LESNGIN (r=-.41) and LESNG (r=-.26) in the expected directions, however.

Hardiness is also related to only one of the physiological baselines; the correlation matrix shows that hardiness is related to BLCORT (r=.37, p<.1) and BLCOCR (r=.41, p<.1). While this is opposite of the predicted direction, it is precisely consistent with Zorilla et al's (1995) finding. This will be later discussed. Hypothesis 6, as stated, was not supported.

Global hypothesis 7 was next examined. A review of the analyses conducted for global hypothesis 1 indicates that stress exerted no direct effects on performance in the presence of other variables. Individually, however, a review of the correlation matrix (Table G1) demonstrates that both LESNGIN and LESNG were individually related to

GLPAPI (r=-.58, p<.05, and r=-.68, p<.05, respectively). There was no support for any mediated relationships. Thus, global hypothesis 7 is supported, but only when bivariate relationships are considered.

Stress appeared to be positively predictive of illness/injury. A review of the analyses used to test global hypothesis 3 indicates LESNGIN is predictive of ILPDDYS, ILPINDY, and TILINDY (see Tables 6 and 8) (LESNG is also comparably related to ILPDDYS and ILPINDY (see Table G1)) while PSS is predictive of INSINDY (see Table 7). Also, PSS appears to be predictive of total illness rate (TOILDYS) in conjunction with BLSBP (R=.61, F(2, 18)=5.331, p<.05) (see Table 10). Mediated relationships, however, were not supported. Global hypothesis 8 is, therefore, not supported as stated, although stress is positively predictive of illness/injury.

Table 10

Results of Regressing TOILDYS on to PSS1 and BLSBP (N=21).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
Intercept	0.097	0.0	1.029	.3169	
PSS	0.004	0.489	2.601	.0180	.24
BLSBP	-0.002	-0.427	-2.269	.0358	.18

R^2=.37
Adjusted R^2=.30

Note: Unique and shared variability are not provided in this table, as they total to greater than .37, indicating an extreme circumstance (Tabachnick & Fidell, 1996).

Global hypothesis 9 can be examined via correlation matrix (Table G1). BLHR is negatively related to INSDDYS (r=-.46, p<.05), INSINDY (r=-.53, p<.05), and

TININDY (r=-.49, p<.05). BLSBP is negatively related to INPINDY (r=-.38, p<.1), TININDY (r=-.42, p<.1), TODYS (r=-.41, p<.1), and TINDY (r=-.41, p<.1). BLDBP is negatively related to ILPDDYS (r=-.41, p<.1), ILPINDY (r=-.40, p<.1), TOILDYS (r=-.40, p<.1), INPINDY (r=-.40, p<.1), TININDY (r=-.40, p<.1), TODYS (r=-.45, p<.05), and TINDY (r=-.42, p<.1). BLCOCR and BLCORT are both negatively related to INSINDY (r=-.42, p<.1, and r=-.45, p<.05, respectively). Global hypothesis 9 is not supported; all relationships were opposite of predicted direction.

Global hypothesis 10 was not supported. While, as noted earlier, illness/injury was related to some performance measures (GLOPA subscales), physiological baselines were not predictive of performance in any of the analyses conducted. In sum, testing of the global hypotheses yielded support (or partial support) for global hypotheses 1, 2, 3, 5, and 7. With the global hypotheses now tested, the cross-over hypothesis must be considered.

Before testing cross-over hypothesis 1 and the acute hypotheses which follow, it is necessary to enunciate a few points. In the following pages, the reader will note many relationships involving transformed variables. A review of Table G2 displays quite clearly the effect of transforming, and so linearizing, a number of IVs. Most of the transformed variables are physiological variables. The reader may wish to examine Table G2 to better appreciate the effect of the transformations. Again, the reader is reminded that brief explanations of variable terms can be found in Appendix H. In general, a "2" added to the end of a variable name implies the original variable has been squared (a "3" similarly indicates a variable has been cubed). An "IN" prefix indicates the inverse of the original variable (-1/(original variable)). Finally, in the analyses which follow, only transformed

IVs will be included; transformation of DVs was not done as such transformations generally impair interpretation (T. Loughin, personal communication, July 1996). Transformations were conducted in accordance with guidance provided by Ott (1993) and Fox (1991).

Physiological reactivity was highly predictive of all measures of illness. Systolic reactivity (SBPCPC2) and magnitude of cortisol reactivity (CORTPM2) were predictive of both ILPDDYS and ILPINDY ($R=.86$ (Adjusted $R=.81$), $F(2, 6)=8.78$, $p<.05$ for both outcomes). SBPCPC3 and CORTPM3 emerged as being highly predictive of both ILSDDYS and ILSINDY ($R=.91$ (Adjusted $R=.88$), $F(2, 6)=14.09$, $p<.01$, and $R=.91$ (Adjusted $R=.88$), $F(2, 6)=14.45$, $p<.01$, respectively). SBPCPC3 and CORTPM3 were predictive of both TOILDYS ($R=.96$, $F(2, 6)=37.91$, $p<.0005$) and TILINDY ($R=.94$, $F(2, 6)=24.38$, $p<.005$) (see Table 11).

In general, measures of injury displayed a much weaker relationship with physiological reactivity. INPDDYS was the only injury measure meaningfully predicted by physiological reactivity in the simulator scenario. SBPCPX3, SDBPMX, SCORTR, and CORTPM3 were predictive of INPDDYS ($R=.97$, $F(4,4)=14.22$, $p<.05$) (see Table 12), although the relationships were opposite of predicted. There was some multicollinearity in this solution as the variables are intercorrelated (see Table G2). This, along with the poor cases to IV ratio suggests the solution be interpreted with caution. Nonetheless, this model was selected per the criteria previously discussed, and the respective predictors do explain unique variance.

Table 11

Results of Regressing TOILDYS and TILINDY on to SBPCPC3 and CORTPM3 (N=9).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
			TOILDYS		
Intercept	-0.013	0.0	-1.672	.1455	
SBPCPC3	0.000	0.733	5.542	.0015	.38
CORTPM3	0.000	0.340	2.568	.0425	.08
R^2=.93				Unique variability=.46	
Adjusted R^2=.90				Shared variability=.47	
			TILINDY		
Intercept	-0.009	0.0	-1.951	.0990	
SBPCPC3	0.000	0.569	3.516	.0126	.23
CORTPM3	0.000	0.503	3.109	.0209	.18
R^2=.89				Unique variability=.41	
Adjusted R^2=.85				Shared variability=.48	

Table 12

Results of Regressing INPDDYS on to SBPCPX3, SDBPMX, SCORTR, and CORTPM3 (N=9).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
Intercept	0.926	0.0	3.986	.0163	
SBPCPX3	0.000	0.373	1.536	.1993	.04
SDBPMX	-0.010	-0.807	-4.180	.0139	.29
SCORTR	-0.030	-0.851	-4.335	.0123	.31
CORTPM3	-0.000	-0.641	-2.289	.0840	.09
R^2=.93				Unique variability=.73	
Adjusted R^2=.87				Shared variability=.20	

In an additional analysis, all relationships between illness/injury measures and physiological reactivity were carefully screened/examined. Relationships between cardiovascular reactivity and cortisol reactivity were also examined to see if the cardiovascular reactivity might possibly be a marker for cortisol reactivity, as Cacioppo (1997) had found in a series of studies. As a result, HRCPX, SSBPMX, and SBPCPX3 were canonically correlated with CORTPM and COCRPM (bivariately, HRCPX was negatively correlated with CORTPM ($r=-.70$, $p<.05$), SSBPMX was positively correlated with COCRPM ($r=.69$, $p<.05$), and SBPCPX3 was positively correlated with CORTPM ($r=.67$, $p<.05$)). The first canonical correlation was .93 (.91 adjusted), and the second canonical correlation was .66 (.65 adjusted). With both canonical correlations included, Wilks-lambda = 0.0757, $F(6, 10)=4.39$, $p<.05$. The cardiovascular measures explained a cumulative 63% of the standardized variance in magnitude of cortisol reactivity. It appears, then, that several cardiovascular measures can reasonably be examined as markers for cortisol reactivity. The cross-over hypothesis was well-supported, especially with respect to the relationship between illness and physiological reactivity.

Acute hypothesis 1 was next examined. Hardiness did not moderate the relationship between PSTSTR and either SPAPRO or SPAACQ. It did, however, moderate the relationship between stress and DECTEC, UNEXEV, and SITAWR. Hardiness X PSTSTR (CH1STR), SSBPAV, and CORTRM were predictive of DECTEC ($R=.96$ (Adjusted $R=.93$), $F(3, 5)=19.16$, $p<.005$). CORTRM was the most powerful predictor (for H_0: $B=0$, $t(8)=-3.299$, $p<.05$), accounting uniquely for 17% of the variance. CH1STR and SSBPAV were less powerful predictors (for H_0: $B=0$, $t(8)=2.533$, $p<.1$, and

t(8)=2.571, p<.1, respectively). Algebraic substitution revealed that for every unit increase in stress, the hardiest individual's DECTEC rating rose 4.25 units; for the least hardy individuals, DECTEC ratings dropped 2.35 units for every single unit increase in stress!

CH1STR, and several measures of cortisol reactivity (COCRRM, CORTRM, and SCORTP), were predictive of the UNEXEV score (R=.96 (Adjusted R=.92), F(4, 5)=14.17, p<.01). CH1STR was the strongest predictor (for H0: B=0, t(9)=4.283, p<.01), explaining 30% unique variance. Hardiness buffered individuals from the deleterious effects of stress in a manner similar to that discussed for DECTEC. This solution, it must be noted, is somewhat threatened by multicollinearity (COCRRM and CORTRM are highly correlated (r=.90, p<.001); yet, they each explain unique variance (.14 and .21, respectively)); the solution should be interpreted with caution.

Finally, CH1STR, SIMUH1, and CORTRM were all significantly predictive of SITAWR (R=.99, F(3, 4)=69.3, p<.001) (see Table 13). Algebraic substitution to assess the nature of the interaction suggests that the hardiest people score almost twice as well as the least hardy individuals. As stress increases, the effect becomes more profound, (less hardy individuals show some increase in SITAWR as stress increases, but at a rate that is .24 less than hardy individuals). Acute hypothesis 1 is supported for the three aircrew coordination qualities of DECTEC, UNEXEV, and SITAWR.

Acute hypothesis 2 was next investigated. Hardiness was found to be predictive of three performance outcomes, SPAACQ, EP, and WRKLD. SPAACQ was predicted by PRECHL and PRECON (R=.79 (Adjusted R=.71), F(2, 6), p<.1). PRECHL was found to

mediate the hardiness-SPAACQ relationship (r=.68, p<.05); when PRECHL is controlled for, hardiness is not predictive of SPAACQ (for H_0: B=0, t(8)=0.570, n.s.).

Table 13

Results of Regressing SITAWR on to CH1STR, SIMUH1, and CORTRM (N=8).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
Intercept	2.099	0.0	3.696	.0209	
CH1STR	0.010	0.642	8.802	.0009	.37
SIMUH1	0.016	0.403	5.514	.0053	.14
CORTRM	-0.045	-0.303	-4.407	.0155	.08
R^2=.98				Unique variablility=.59	
Adjusted R^2=.97				Shared variability=.39	

EP was predicted by hardiness and 3 measures of cortisol reactivity (COCRRM, SCORTR, and CORTRM) (R=.99 (Adjusted R=.98), F(4, 5)=46.72, p<.0005). Hardiness was the strongest predictor (for H_0: B=0, t(9)=7.105, p<.001), accounting uniquely for 26% of the variance. COCRRM, SCORTR, and CORTRM explained 23 (for H_0: B=0, t(9)=6.675, p<.005), 16 (t(9)=-5.509, p<.005), and 23 (t(9)=-6.614, p<.005) percent of unique variance in EP, respectively. Similar to the case discussed previously involving predictors of UNEXEV, the interrelationships between the measures of cortisol reactivity create some threat of multicollinearity. The solution should, therefore, be carefully interpreted, despite the maximization/minimization of appropriate model selection criteria.

WRKLD evaluation was predicted by hardiness, FLYALL, and CORTPM3 (R=.89 (Adjusted R=.83), F(3,6)=7.571, p<.05), although hardiness was a relatively weak

predictor (for H_0: B=0, t(9)=2.264, p<.1) despite accounting for a unique 18% of the variance in WRKLD. FLYALL was the most robust predictor (t(9)=2.573, p<.05) and uniquely explained 23 percent of the variance. CORTPM3 was a non-significant predictor in the presence of these other 2 IVs. Acute hypothesis 2, then, is supported for SPAACQ and partially supported for EP and WRKLD.

Acute hypothesis 3 was next examined. Hardiness was in no way predictive of, nor significantly related to (r=.36, n.s.), PSTSTR. Acute hypothesis 3 was not supported.

In testing acute hypothesis 4, stress was found to be related to the performance outcomes of DECTEC, INFOSOT, and SITAWR. A reduced regression modelling of DECTEC on to PSTSTR and CORTRM was significant (R=.95, F(2, 6)=29.69, p<.001) (see Table 14). Surprisingly, stress was positively related to performance. There was no support for a mediated relationship.

Table 14

Results of Regressing DECTEC on to PSTSTR and CORTRM (N=9).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
Intercept	1.224	0.0	0.996	.3575	
PSTSTR	1.685	0.622	5.021	.0024	.39
CORTRM	-0.163	-0.694	-5.602	.0014	.48
R^2=.91				Unique variability=.87	
Adjusted R^2=.88				Shared variability=.04	

INFOSOT was predicted by the inverse of SCORTP (INSCORTP) and CORTPM (R=.88 (Adjusted R=.85), F(2, 7)=12.40, p<.01). Both predictors contributed to the

model (for H_0: B=0, t(9)=-2.768, p<.05 (INSCORTP), and t(9)=3.003, p<.05 (CORTPM)). In turn, both SCORTP and CORTPM were predicted by a transformation of PSTSTR (PSTSTR3), and PSTSTR3 was significantly related to INFOSOT (r=-.63, p<.1). Mediated relationships per the guidance of Baron and Kenny (1986) were supported. That is, PSTSTR3, as a predictor of INFOSOT, was non-significant when INSCORTP and CORTPM were controlled for (for H_0: B=0, t(9)=-1.760, n.s., and t(9)=-0.434, n.s., respectively).

Finally, SITAWR was related to PSTSTR (r=.69, p<.05), although the relationship does not emerge in the presence of other variables (CH1STR, for example). Also, the relationship is, unexpectedly, positive. No mediated relationships were supported. Acute hypothesis 4, therefore, was supported only for INFOSOT, although the relationships between stress and both DECTEC and SITAWR are quite intriguing.

Only the relationship between PSTSTR and SDBPMX appeared to be moderated by hardiness. CH1STR and PRUNCN were predictive of SDBPMX (R=.84, (Adjusted R=.79), F(2, 7)=8.57, p<.05), although only CH1STR was significantly related to SDBPMX (for H_0: B=0, t(9)=2.996, p<.05). Examination of the effects of hardiness via algebraic substitution revealed that at lower levels of stress, hardy individuals have a lower SDBPMX. As stress increases, SDBPMX rises for hardy individuals and falls for those who are less hardy. At PSTSTR values > 3, the most hardy individuals have higher SDBPMX readings (and they continue to rise) than the least hardy individuals. Acute hypothesis 5 is supported for SDBPMX, although the nature of the relationship is

somewhat unexpected. Indeed, inspection of the correlation matrix (Table G2) shows that hardiness is positively related to SDBPMX ($r=.57$, $p<.1$).

In the investigation of acute hypothesis 6, some background information is first necessary. The relationship between hardiness and cortisol reactivity appears to be curvilinear, and some data transformation was necessitated. For example, Figure 5 illustrates the relationship between hardiness and the change, as a percentage (SCORTP), of SCORT from BLCORT. The highest hardiness score (136) corresponds with a SCORTP value of -48.8; in fact, the 5 most hardy participants displayed a mean deviation from BLCORT of -41.6%. A similar, though less dramatic, pattern is seen if SCOCRP is plotted against hardiness (the 5 most hardy participants displayed a mean deviation from BLCOCR of -31.5%). What is indicated by these respective plots is that hardiness appears to be related to the magnitude of deviation from baseline values (for example, CORTPM or COCRPM), or more particularly, magnitude of deviation from some sub-baseline value. One transformed variable considerate of this is CRTPDVM, mathematically defined as: (|(SCORTP - (-48.8)|).

COCRPM and CRTPDVM were both predicted by FLYUH1 and PRECHL ($R=.86$ (Adjusted $R=.81$), $F(2, 6)=8.86$, $p<.05$, and $R=.83$ (Adjusted $R=.77$), $F(2, 6)=6.83$, $p<.05$, respectively). As noted previously, PRECHL is predicted by hardiness, and hardiness is robustly related to (predictive of) COCRPM ($r=-.69$, $p<.05$) and CRTPDVM ($r=-.70$, $p<.05$). When PRECHL is controlled for, hardiness is neither predictive of COCRPM (for H_0: $t(8)=-0.670$, n.s.) nor CRTPDVM ($t(8)=-0.429$, n..s.).

That is, hardiness is negatively predictive of COCRPM and CRTPDVM, but the relationship is mediated by challenge appraisals (PRECHL), and not stress (PSTSTR).

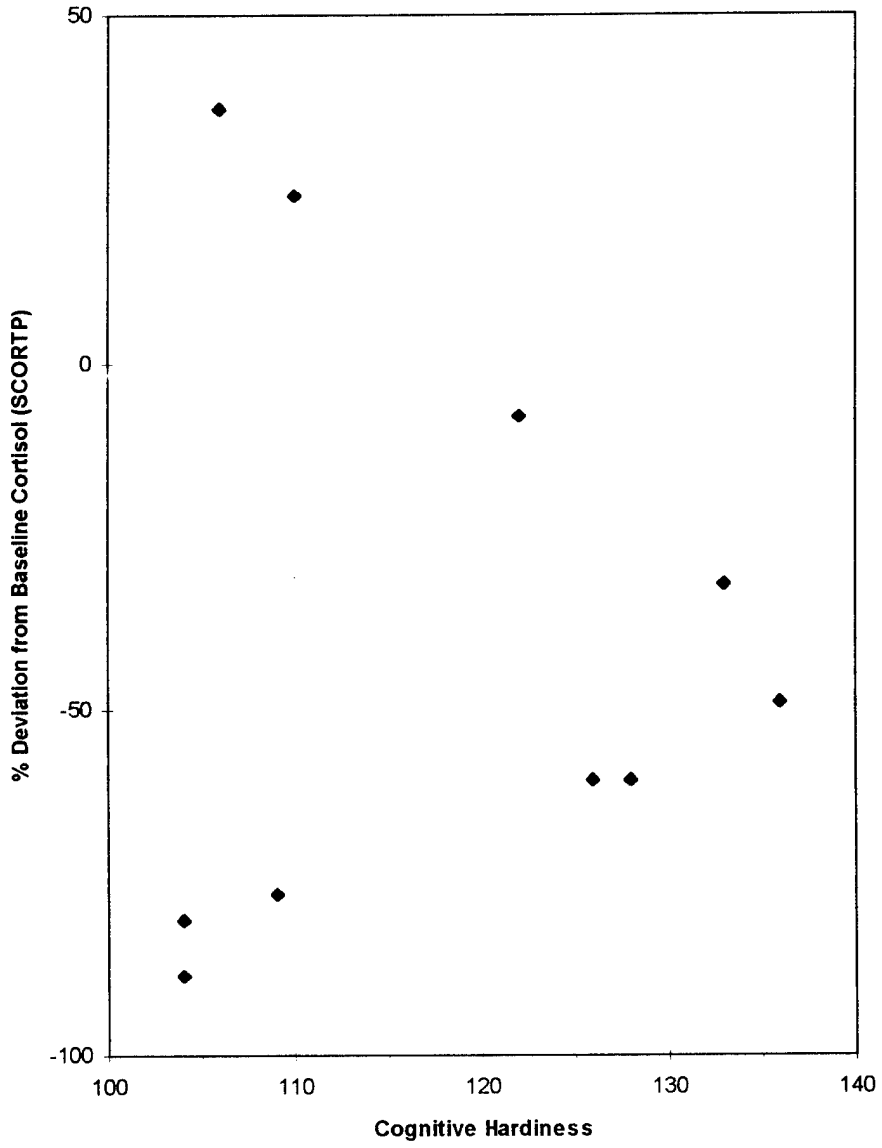

Figure 5. The relationship between hardiness and SCORTP.

Hardiness, as discussed in testing acute hypothesis 5, is also predictive of SDBPMX; however, that relationship is not mediated by PSTSTR either. Further, hardiness demonstrated no relationship with HR or SBP reactivity. Acute hypothesis 6, as stated, is unsupported.

Hardiness was not related to, nor predictive of, threat appraisals. Hardiness is, conversely, positively predictive of both PRECHL ($r=.86$, $p<.005$) and PSTCHL ($r=.57$, $p<.1$). PSTCHL is also negatively predicted by PRUNCN ($r=-.62$, $p<.1$) and together, hardiness and PRUNCN explain 74 percent of the variance in PSTCHL ($R=.86$ (Adjusted $R=.81$), $F(2, 7)=9.79$, $p<.01$). Acute hypothesis 7 is, therefore, unsupported. Acute hypothesis 8 is supported.

Contrary to what is postulated in acute hypothesis 9, stress appears to be related both positively and negatively to various measures of physiological reactivity. PSTSTR is positively related to various outcome measures of HR reactivity, especially maximal values of reactivity; the correlation matrix (Table G2) reveals PSTSTR is correlated with HRCRX ($r=.56$, $p<.1$) and HRCPX ($r=.55$, $p<.1$). At the same time, PSTSTR is negatively related to measures of systolic reactivity such as SBPCPX ($r=-.58$, $p<.1$) while being positively related to SDBPMX ($r=.67$, $p<.05$) (but not significantly related to diastolic reactivity). Finally, PSTSTR is positively related to SCORTP ($r=.57$, $p<.1$), while being negatively related to CORTPM ($r=-.68$, $p<.05$). These results are intriguing and will be discussed later. Acute hypothesis 9 is supported only for the relationships between PSTSTR and HR reactivity/SCORTP.

In examining acute hypothesis 10, a number of relationships between physiological reactivity and performance were observed per inspection of the correlation matrix (Table G2). HRCHRW and HRCHPC were both related to INFOSOT ($r=-.56$, $p<.1$, and $r=-.60$, $p<.1$, respectively) and INFOOFR ($r=.65$, $p<.1$, for both measures of average HR reactivity). While SSBPAV was positively related to performance measures such as DECTEC ($r=.80$, $p<.01$), WRKLD ($r=.57$, $p<.1$), and UNEXEV ($r=.60$, $p<.1$) and aviation experience such as FLYALL ($r=.80$, $p<.01$) and SIMALL ($r=.71$, $p<.05$), such was not the case for averaged systolic reactivity. SBPCRW was negatively related to SPAPRO ($r=-.56$, $p<.1$) and ADVASS ($r=-.61$, $p<.1$). SBPCPC was also negatively related to ADVASS ($r=-.66$, $p<.1$). The relationship between averaged systolic reactivity and ADVASS was, further, curvilinear; accordingly, SBPCRW3 and SBPCPC3 were highly correlated with ADVASS ($r=-.84$, $p<.005$, and $r=-.84$, $p<.01$, respectively (differing levels of significance reflect rounding)). Flight experience (FLYALL, FLYUH1) was positively related to SSBPMX ($r=.61$, $p<.1$, and $r=.64$, $p<.05$, respectively). Maximal systolic reactivity, however, was negatively related to several measures of performance. SBPCRX and SBPCPX were negatively related to SPAACQ ($r=-.64$, $p<.05$, for both measures) and WRKLD ($r=-.56$, $p<.1$, and $r=-.58$, $p<.1$, respectively), while SBPCRX2 and SBPCPX2 were negatively related to DECTEC ($r=-.71$, $p<.05$, and $r=-.70$, $p<.05$, respectively), UNEXEV ($r=-.63$, $p<.05$, and $r=-.61$, $p<.1$, respectively), and SITAWR ($r=-.67$, $p<.05$, for both measures).

SDBPAV was positively associated with multiple measures of performance such as SPAPRO ($r=.64$, $p<.05$), EP ($r=.61$, $p<.1$), SPAACQ ($r=.60$, $p<.1$), DECTEC ($r=.68$,

p<.05), and WRKLD (r=.64, p<.05). Experience (FLYALL and SIMALL) was also positively related to SDBPAV (r=.64, p<.05, and r=.59, p<.1, respectively). DBPCRW and DBPCPC, however, were negatively related to ADVASS (r=-.74, p<.05, and r=-.78, p<.05, respectively). SDBPMX was positively related to EP (r=.62, p<.1), DECTEC (r=.69, p<.05), and WRKLD (r=.64, p<.05), while DBPCRX and DBPCPX were negatively related to ADVASS (r=-.60, p<.1, and r=-.65, p<.1, respectively).

Finally, it was necessary to examine cortisol reactivity and its relationship to performance. SCOCR was, interestingly, positively related to flight experience (FLYUH1 (r=.70, p<.05) and SIMUH1 (r=.60, p<.1)), while SCORT was related only to INFOX (-.72, p<.05). SCOCRR was positively related to DECTEC (r=.67, p<.05) and UNEXEV (r=.70, p<.05) while SCORTR was positively related to DECTEC (r=.73, p<.05), UNEXEV (r=.77, p<.01), and SITAWR (r=.60, p<.1). SCOCRP was unrelated to any performance outcomes, although it was positively associated with FLYUH1 (r=.61, p<.1) and SIMUH1 (r=.73, p<.05). SCORTP, however, was positively associated with DECTEC (r=.60, p<.1), UNEXEV (r=.57, p<.1), and SITAWR (r=.65, p<.1). FLYUH1 and SIMUH1 were also related to SCORTP (r=.56, p<.1, and r=.64, p<.1, respectively). Raw magnitude of reactivity from baseline values was next examined for its relationship to performance. COCRRM was negatively related to EP (r=-.63, p<.1) and UNEXEV (r=-.59, p<.1) while being positively related to ADVASS (r=.60, p<.1). CORTRM, comparatively, was negatively related to EP (r=-.69, p<.05), DECTEC (r=-.72, p<.05), UNEXEV (r=-.76, p<.05), and SITAWR (r=-.61, p<.1). COCRPM was negatively related to INFOX (r=-.66, p<.1), while being positively related to ADVASS (r=.65, p<.1)

and positively associated with FLYUH1 ($r=.71$, $p<.05$). CORTPM, on the other hand, was robustly related to several performance outcomes, the relationship sometimes being non-linear. CORTPM was negatively related to DECTEC ($r=-.74$, $p<.05$) and positively related to INFOSOT ($r=.73$, $p<.05$). CORTPM2 was negatively related to EP ($r=-.79$, $p<.01$) and SITAWR ($r=-.92$, $p<.001$). CORTPM3 was negatively related to WRKLD ($r=-.64$, $p<.05$) and UNEXEV ($r=-.80$, $p<.01$). Acute hypothesis 10 appears to be largely supported, although systolic reactivity (but not elevated systolic tone) was also negatively related to performance. Cortisol reactivity also appeared to be differential in its effects; while elevated reactivity was often positively associated with performance, magnitude of reactivity was predominantly negative in its relationship to performance. Summarily, acute hypotheses 1, 2, 4, 5, 8, 9, and 10 are supported or partially supported.

Having investigated the acute hypotheses, a re-visitation of hypothesis 2 is warranted. Recall that hardiness was found to be predictive of SPAACQ (as mediated by PRECHL), EP, and WRKLD. Also, and not previously discussed, examination of Table G2 reveals that hardiness is also related to UNEXEV ($r=.58$, $p<.1$) and SITAWR ($r=.76$, $p<.05$), although hardiness did not emerge as a prominent predictor in the presence of other variables (including the CH1STR interaction term) per setwise regression analyses. Keeping that in mind, recall the unique curvilinear relationship between hardiness and cortisol reactivity (SCORTP) (see Figure 5) that was uncovered in the investigation of acute hypothesis 6. Given the relationships between cortisol secretion and performance in tasks demanding working memory, information consolidation, and spatial relationships (see pp. 80-86), the prospect of investigating the relationships between working memory-

intensive performance outcomes, hardiness, and cortisol reactivity becomes rather attractive.

Scatterplots reveal that several such performance outcomes (SPAACQ, EP, WRKLD, UNEXEV, and SITAWR) are related to SCORTP in a pattern which is quite similar (though less dramatic) to that displayed in Figure 5. That is, all these performance outcomes are generally maximized at a cortisol reactivity (SCORTP) between -30 and -50 percent from baseline, with performance declining in a generally progressive manner as reactivity falls outside this range. As an example, the relationship between EP and SCORTP is displayed in Figure 6. Given the directionality of the relationship as implied by previous research and theory, EP should, perhaps, have been displayed on the Y-axis; however, the plot has been presented with EP on the X-axis to facilitate comparison with Figure 5. Also, another pattern becomes apparent as well when the various scatterplots are compared. Of the 5 least hardy individuals, note from Figure 5 that 2 demonstrated cortisol elevation while 3 showed dramatic declines in secretion that could be characterized as a "blunted" response of the HPA axis. In Table 15, hardiness scores, SCORTP values, and corresponding scores on EP and several crew coordination qualities are listed. While performance decrements (compared to the 5 hardiest individuals) are associated with both the elevated response and the depressed cortisol response, it is apparent, by inspection, that the blunted response is associated with far more profound performance degradation. It is this pattern, by the way, that yields positive correlations between SCORTP and various performance outcomes while CORTPM is associated negatively with the same crew coordination quality evaluations (see Table G2). These

patterns strongly suggest that cortisol reactivity, despite the lack of findings in testing acute hypothesis 2, may mediate the relationship between hardiness and performance outcomes.

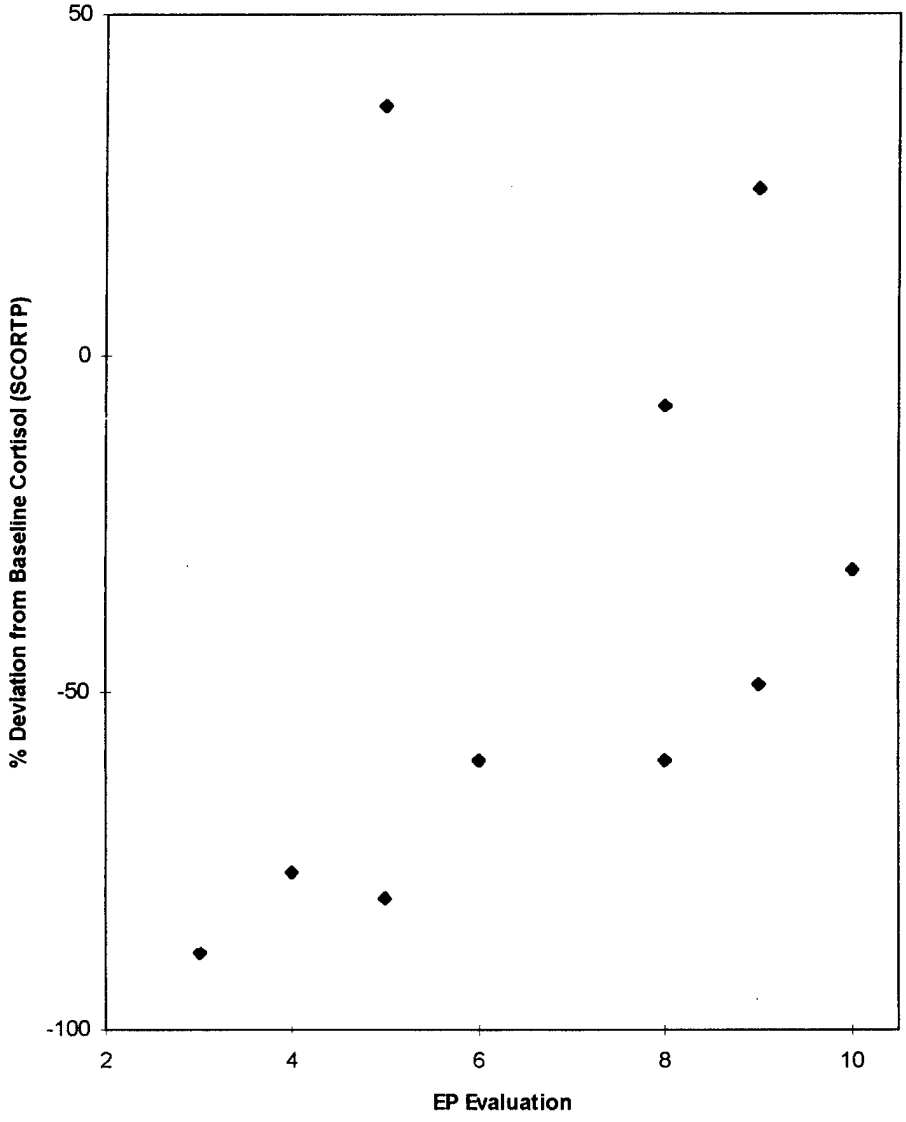

Figure 6. The relationship between EP and SCORTP.

Table 15

Relationships Between SCORTP and Performance Outcomes in the 5 Least Hardy Simulator Scenario Participants.

Hardiness	SCORTP	EP	DECTEC	WRKLD	UNEXEV
104	-88.5	3	3	2	2
109	-76.7	4	3	3	2
104	-80.3	5	6	4	7
106	36.7	5	7	6	9
110	24.4	9	8	8	9

As revealed in testing acute hypothesis 10, several crew coordination qualities and EP were negatively associated with CORTPM and its transformations. Also, many of these same outcomes were robustly related to hardiness. To examine relationships between hardiness and these outcomes as mediated by CORTPM (and transformations to linearize relationships), CORTPM should be predicted by hardiness per Baron and Kenny (1986). Examination of Table G2 reveals that this condition for mediation is not met, but the correlations are reasonably large (-.38 to -.54) and in a predicted direction. It is entirely possible that this represents an extant relationship not significant in this case as a result of small sample size. Accordingly, several of these outcomes were regressed on to hardiness and CORTPM (or transformations thereof). When EP is regressed on to hardiness and CORTPM2 ($R=.90$ (Adjusted $R=.87$), $F(2, 7)=14.66$, $p<.005$), hardiness remains significantly predictive (for H_0: $B=0$, $t(9)=2.630$, $p<.05$), but the predictive contribution of hardiness is diminished; partial mediation is, therefore, suggested. When UNEXEV is regressed on to hardiness and CORTPM3 ($R=.82$ (Adjusted $R=.76$),

F(2, 7)=6.99, p<.05), hardiness is not predictive (for H_0: B=0, t(9)=0.812, n.s.). This is supportive of mediation. Finally, when SITAWR is regressed on to hardiness and CORTPM2 (R=.96, F(2,6)=31.77, p<.001), hardiness is no longer significant at the p<.05 level (see Table 16). This is supportive of mediation. It is also worth noting that CORTPM2 alone accounts for more than 84% of the variance in SITAWR.

Table 16

Results of Regressing SITAWR on to Hardiness and CORTPM2 (N=9).

Variable	B	β	t for H_0: B=0	p	sr^2 (unique)
Intercept	4.822	0.0	2.387	.0542	
Hardiness	0.034	0.329	2.184	.0717	.07
CORTPM2	-0.000	-0.720	-4.781	.0031	.33
R^2=.91				Unique variability=.40	
Adjusted R^2=.89				Shared variability=.51	

Such strong support for mediation and the curvilinear nature of the relationships suggests another possibility; while the mediated relationships may not have revealed themselves due to such small sample size, it is conceivable the relationships remain hidden in more complex higher order explanations. I have, up to this point, avoided these for 2 reasons. Firstly, they may be difficult to interpret, and secondly, it is somewhat speculative to fit complex curvilinear equations to patterns formed by few data points. Nonetheless, this effort is somewhat ground-breaking and exploratory. Use of regression analyses permitted derivation of a polynomial equation to fit the hardiness-SCORTP relationship; hardiness is roughly equivalent to

$$121.325 - .46279(SCORTP) - .001795(SCORTP^2) + .000072566(SCORTP^3).$$

Hardiness is highly related to a term set equal to this mathematical representation ($r=.96$, $p<.0005$).

Various performance outcomes were then regressed on to this polynomial term, including SPAACQ ($R=.58$ (Adjusted $R=.51$), $F(1, 8)=4.136$, $p<.1$), EP ($R=.75$ (Adjusted $R=.71$), $F(1, 8)=10.243$, $p<.05$), WRKLD ($R=.63$ (Adjusted $R=.56$), $F(1, 8)=5.189$, $p<.1$), UNEXEV ($R=.57$ (Adjusted $R=.49$), $F(1, 8)=3.774$, $p<.1$), and SITAWR ($R=.71$, (Adjusted $R=.66$), $F(1, 7)=7.212$, $p<.05$). This, as with previously discussed results, suggests that performance outcomes are associated with cortisol reactivity. It remained to regress these outcomes on to hardiness and this polynomial representation of cortisol reactivity. When this was done, in *every* instance hardiness was non-significantly predictive when the polynomial was controlled for (see Table 17). Cortisol reactivity, then, mediated the relationship between hardiness and working memory-intensive, spatially demanding performance outcomes.

Table 17

Predictive Significance of Hardiness When Controlling for Cortisol Reactivity.

Outcome Variable	$B_{hardiness}$	$\beta_{Hardiness}$	t for H_0: B=0	p	Overall R^2
SPAACQ	0.113	1.453	1.617	.1499	.52
EP	0.093	0.485	0.577	.5817	.58
WRKLD	0.177	0.836	0.871	.4125	.45
UNEXEV	0.109	0.457	0.433	.6782	.34
SITAWR	0.098	0.953	1.087	.3189	.59

Finally, it is worth noting that INFOSOT (positively correlated with CORTPM, $r=.73$, $p<.05$) demonstrates a relationship with SCORTP which is largely the inverse of that between previously mentioned performance outcomes. That is, a SCORTP value of -7.6 is paired with a value of 2 (lowest value) for INFOSOT. For the 5 least hardy individuals, those with a blunted cortisol response scored highest on INFOSOT (8, 6, and 9) while the 2 individuals displaying positive cortisol reactivity scored 7 and 6 on INFOSOT.

These results, to include those in which liberties were admittedly taken with Baron and Kenny's (1986) guidance, collectively suggest that hardiness is predictive of performance as mediated by cortisol reactivity. Testing for hypothesis 6 further suggests that challenge appraisals mediate the relationship between hardiness and cortisol reactivity. While it appears that cortisol elevation and decrease in response to the simulator scenario were associated with performance degradation, blunted response was associated with comparatively more adverse impact. Finally, it appears that INFOSOT is associated with cortisol reactivity in a manner diametrically opposite to that of EP and crew coordination qualities. As I'll elaborate upon in the discussion section, both the statistical and qualitative nature of these relationships may be profoundly important.

Discussion

Hardiness has been proffered as a stress resiliency resource capable of moderating stress-illness and stress-performance relationships (see pp. 34-56). It has also been found to be related to some physiological function and reactivity (Allred & Smith, 1989; Contrada, 1989; Wiebe, 1991), although only Zorilla et al. (1995) have explored its relationship with cortisol secretion, perhaps the most robustly supported mediator of the stress-illness dynamic (Sapolsky, 1992a). In the past, hardiness has also been associated with the way in which individuals appraise possibly stressful situations (Banks & Gannon, 1988; Maddi & Kobasa, 1984). In the present effort, relationships between hardiness, appraisal processes, stress, physiological function, illness/injury, and performance were explored. A number of potentially profound, original, and provocative findings emerged from this prospective venture. Before discussing the findings, their implications, and future research directions, however, I'd like to address both the limitations and strengths of this study.

There are a number of limitations and weaknesses in the present effort which deserve attention. The most obvious is the small sample size. Statistically, almost any applied methodology is best used and more powerful with a larger sample size; the sample size/cases to IVs ratios in this study were, indeed, less than that recommended by Tabachnick and Fidell (1996) for regression analyses. Given a small sample size, there is the possibility that a set of predictors may explain a great deal of variance in the DV, and yet, provide a solution which lacks theoretical meaningfulness (Tabachnick & Fidell, 1996). Also, with small samples, solutions are more susceptible to the influence of

individual observations and individual errors in observation/measurement (Fox, 1991). Contrarily, Achen (1982) notes that regression is an appreciably resilient statistical technique which tends to the correct solution, even in instances characterized by violations of classical postulates and convention. Furthermore, as mentioned, great efforts were taken to ensure results were not biased by individual observations. Additionally, setwise regression has been recommended as a technique which, when sample sizes are small, can be used to identify optimal solutions (tempered, of course, by theoretical considerations) and promising predictors (Tabachnick & Fidell, 1996). Accordingly, extensive use was made of setwise regression in this effort. Finally, it is worth again noting that a surprising number of variables were normally distributed; while not a necessity for regression, such distribution may enhance the integrity of the solution (Tabachnick & Fidell, 1996).

The sample in this study was quite homogenous. All were members of a highly specialized profession and part of an organization with, undoubtedly, a strong set of unique and shared organizational values. As previously addressed, this homogeneity may have suppressed the Cronbach's alpha values for some of the measures. This sample was remarkably hardy as well; mean hardiness (118.17) was .92 s. d. above the mean for the normative data (K. Nowack, personal communication, February 1996). All this suggests caution be taken in generalizing the results of this study to the general population. While generalization of the findings to military aviators is probably acceptable, one must be careful even in this occupational domain due to the small sample size.

Finally, it is possible that the varied schedules forced by the company's deployment schedule, operational priorities, and limited resource availability might have introduced

error into the measurements taken. Many participants, for example, had to be continuously contacted to collect illness/injury inputs, final questionnaires, etc. Also, despite responses indicating that this did not occur for the simulator scenario, it is possible that information was shared; this would not be unexpected in a unit that appears (qualitatively) to have an increasing degree of unit cohesion following the assumption of command by a new commander. Lastly, the simulator scenario, despite apparently providing a realistic stressor (mean for PSTSTR=3.63, s. d.= .73), did vary slightly for each participant as a result of that subject's responses, decisions, and the components of mandatory training/evaluation that the individual evaluators worked into the scenario. Such variation is probably not avoidable in any realistic and complex simulation. The Standardization staff did express their conviction that the scenarios were equally difficult, challenging, and technically demanding of all participants; that is, the scenarios are thought to have produced reliable outcome measures.

I would be remiss to ignore the strengths of this study, however. There was some control in that all participants were subjected to the same work environment—a highly demanding work environment in a unit which, over 8 months, saw significant change (improvements) in operational readiness. All participants were healthy, engaged in regular exercise, and, by Department of the Army requirements, maintained normative body weights (individuals were neither underweight nor clinically overweight/obese). Illness/injury was recorded in a relatively real time manner in subjects accustomed to self-reporting illness/injury; per the findings of Funk and Houston (1987) (see p. 43), it is thought that this methodology provided relatively accurate information on illness/injury

rates. The simulator scenario provided a realistic, intense, workplace stressor in a controlled environment; the scenario demanded active/transformational coping and such effective coping was, inherently, directly proportional to performance outcomes. The scenario was especially demanding of active coping strategies, working memory capacity, and manipulation of spatial relationships per both the Standardization staff and comparison to the study done by Wickens et al. (1993). The social support available in a given scenario was also controlled for, and while crew combinations differed, all simulator participants had the same sources (crew member and air traffic controller) of social support (also, as previously discussed, a stress resiliency resource). Comments by both participants and the Standardization staff also suggested that the emergencies in the scenario were highly stressful and constituted critical incidents in emergency management (coping with hydraulic systems failure was especially thought to be stressful and demanding). As with Study 1, the design was prospective, permitting more valid conclusions about directionality of relationships as well as appropriate use of regression analyses. Also, collection of urine over the simulator period avoided previous design flaws in which cortisol secretion has often been assessed without permitting sufficient time for serum levels to peak and for unbound cortisol to be discovered in serum, saliva, or urine (Glaser & Kiecolt-Glaser, 1994; Manuck et al., 1991).

Prospective design, recording of life experiences, and a test-retest of hardiness and perceived stress over a 176-day interim revealed marked changes in the hardiness and stress perceptions of one individual (see page 150). This individual's hardiness score fell from 135 (+1.11 s. d.) to 94 (-1.60 s. d.) while stress perceptions increased. This

individual did experience a tremendous number of major life events over the 176 days in question. This, plus the work of Maddi (1987), suggests that hardiness is malleable. The findings in this case demonstrate that frequent and intense life changes (environmental influence) may degrade hardiness in *some* individuals (another individual in the study had a child diagnosed with possibly fatal illness—this individual's hardiness did not change (130 @ time 1, 125 @ time 2)). This finding (that intense/frequent stressors/changes can degrade hardiness) has not, to my knowledge, previously appeared in the hardiness literature, and calls into question, at least to some degree, the validity of findings in retrospective designs; this is especially the case in retrospective studies dealing with extreme stressors (Hodgkinson & Shepherd, 1994; Sutker et al, 1995). Observations on this individual constitute informative outliers which call for future hardiness research to be prospective in design. That is, future hardiness research should include assessment of outcomes at some time following measurement of hardiness; test-retest of hardiness is also suggested as a design consideration. Current attempts to track the above individual's hardiness at 6-month intervals over the coming years have not met with success; the individual has not, at present, agreed to participate further.

Hardiness was found to both moderate the stress-performance relationship and exert main effects on performance. Globally, the moderating effect of hardiness was rather weak; further, hardiness was revealed to provide less of a buffer against the adverse impact of stress as negative life experiences and their impact increased. For global performance (GLOPA), it was revealed that hardiness provided a stress resiliency resource until a rather high number of negative life experiences occurred over a 6 month period.

For the global appraisal of piloting performance (GLPAPI), however, the buffering effect of hardiness disappeared with relatively little increase in perceived negative impact above the mean. Interestingly, the components of the global performance appraisal were, in general, best predicted by physician-diagnosed (but not self-diagnosed) illness and injury rates (but not absenteeism); in fact, 37% of the performance rating during the 6 month rating period was explained by these illness/injury rates. What is not known (and perhaps the subject of future research) is whether the physician-diagnosed illness/injury actually degraded performance or merely biased the perceptions of raters. The moderating effect of hardiness was more robust in a few specific domains of acute performance; further, the moderating relationship was classically interactionist. For both decision technique (DECTEC) and management of unexpected events (UNEXEV), it was found that as stress increased, hardy individuals performed increasingly better, while less hardy individuals performed progressively worse. As stress increased, situational awareness (SITAWR) increased for both high and low hardy individuals, but for the hardiest individuals, it increased 34% faster than it did for the least hardy participants. Interestingly, cortisol reactivity was also associated with performance in these domains and will be shortly discussed. Not unexpectedly, experience in the UH-1 simulator was also predictive of situational awareness.

The main effects of hardiness on performance varied. While hardiness was a weak predictor of officer potential for promotion (OFFPOT), it was strongly predictive of many crew coordination qualities and effective employment of procedure/maneuver in an emergency situation (EP). The relationship between hardiness and overall performance

on aircrew coordination qualities (SPAACQ) was found, as hypothesized, to be mediated by challenge appraisals. The relationship between hardiness and employment of emergency procedures (EP), SPAACQ, workload management (WRKLD), management of unexpected events (UNEXEV), and situational awareness (SITAWR) was mediated by cortisol reactivity, which will be shortly discussed. Together, these findings provide support for contentions that hardiness influences appraisal processes and acts as both a moderator and directly in shielding performance from the adverse impact of stress. In this study, the effects of hardiness appeared to be most theoretically consistent in an intense, acutely stressful situation (versus a long-term, chronic situation such as global performance in all assigned duties). Finally, these findings demonstrated a solid relationship between hardiness, cortisol reactivity, and some dimensions of performance; this is an original finding, although a relationship between hardiness and cortisol reactivity had been postulated by Zorilla et al (1995).

Hardiness was found to robustly moderate the stress-illness relationship, while it exerted only weak direct effects on illness. Hardiness was not related to injury rates or incidence. Physician-diagnosed illness and incident rates, as well as total illness incident rate were predicted by hardiness-stress interactions. As negative life incidents increase, it was found that illness increased in both high and low hardy individuals; however, in the least hardy individuals, for example, the physician-diagnosed illness rate increased with greater negative life experiences at a rate 3.25 times that of the rate of increase in the hardiest individuals. It is interesting to note that self-diagnosed illness rates were related to a completely different set of predictors (marital status and time on active duty being

prominent); this suggests that self- and physician-diagnosed illness may be different phenomena. In future research, these two types of illness should, perhaps, be delineated. The above relations suggest, for example, that social support may play a greater resiliency role in the relationship between stress and self-diagnosed illness than in the stress-physician-diagnosed illness relationship. For total illness incident rate, it was found that an increase in negative life experiences was associated with decreasing illness incidence in the most hardy individuals while being associated with total illness incident increase in the least hardy individuals that was 8 times the magnitude of decrease in hardy individuals! Hardiness was shown to be a potent moderator of the stress-illness (physician-diagnosed) relationship.

Unexpectedly, the hardiness-positive life change interaction was associated with self-diagnosed injury incident rate. That is, hardier individuals are likely to have a higher self-diagnosed injury incident rate than low hardy individuals as positive life change increases. This is a bit puzzling and may represent a type I error. It is also possible that hardy individuals, as positive perceptions increase, may, as a result of seeking challenge and exerting control, engage more frequently in activities which could lead to minor injury. Competitive athletics may be such an activity (per a review of illness/injury diary sheets). Hardiness may also be related to risk-taking in certain domains. Given this perspective, it may not be that surprising to find higher rates of minor injury incidence in hardy individuals who, consistent with theory, might be expected to have more active and authentic lifestyles.

For those who participated in the simulator scenario, total illness and incident rates were solidly related to systolic (increased) and cortisol (magnitude of deviation from baseline) reactivity. The relationships between physiological baselines and illness/injury, however, were generally weak and in a direction opposite of predicted. This provides support for Cacioppo's (1997) reactivity hypothesis. That is, illness may be better explained by SAM and HPA reactivity to acute stressors than by tonic states of cardiovascular tone and cortisol secretion patterns. Some of the global relationships should even have been expected. For example, the negative relationship between baseline cortisol levels and self-diagnosed injury incidence is consistent with the anti-inflammatory effects of cortisol.

In the past, Cacioppo (1997, 1994) and Uchino et al (1995) have reported findings which show relationships between HR/SBP reactivity (as imperfect markers of sympathetic reactivity) and both cortisol secretion as well as immunological change believed to be cortisol mediated. In this effort, measures of HR and systolic reactivity were canonically correlated with measures of cortisol reactivity to determine if they, collectively, might serve as markers for cortisol reactivity. While HR reactivity was negatively associated with magnitude of cortisol reactivity, systolic reactivity was positively associated with magnitude of cortisol reactivity. Together, HR and systolic measures of reactivity accounted for 63% of the variance in magnitude of cortisol reactivity. While PEP may indeed be a more precise marker of sympathetic reactivity and associated HPA reactivity (Cacioppo, 1997), these results suggest that HR and SBP reactivity are still valuable as easily measured indices of SAM/HPA reactivity.

Finally, it must be noted that magnitude, not elevation per se, of cortisol reactivity was a predictor of illness. Per the in-depth discussion in the "Glucocorticoid Mediated Mechanisms of Immunosuppression, Illness, and Performance-Related Dysfunction" chapter of this work, such a finding suggests that both cortisol elevation and blunted responses/decreases may have adverse impacts on health. This is notable because it unifies what appear to be disparate research findings. The present finding is consistent with a biphasic function for GCs in the maintenance of health and well-being, a position echoed by Chrousos and Gold (1992) and Nelson (1996) in their respective fields. Further, as Chrousos and Gold (1992) suggest, such a finding is supportive of an inverted-U dose response curve marking the relationship between well-being and/or performance and reactivity to stress, such that well-being/performance are degraded in those who are either hyper- or hyporeactive (i. e. those who "fall off" the edges of the curve). Further, the reader will note the variables of cortisol reactivity which most emerged in these analyses were individualized measures of response (percentage changes from baseline), and not raw changes. This is sensible in that there is a tremendous range of cortisol secretion in healthy individuals over the diurnal rhythm (the 95% CI for normal range of urinary free cortisol in healthy individuals is 9-53 μ g/24 hours (Abbott Laboratories, 1994)). It is reasonable to conclude, then, that in a given population there is a collection of individual cortisol response curves, such that for any individual hyper- or hyporeactivity in cortisol secretion is unique. All this indicates that individualized, and not raw, measures will yield the most informative body of data in future investigation of cortisol reactivity; additionally, it is recommended that, in the future, baseline cortisol measurements be taken at times in

the diurnal rhythm that afford observation of both increased and decreased cortisol secretion in response to stressors. It appears, then, that those who "fall off" the edges of their inverted-U (relating health/performance to cortisol reactivity) are most likely to become ill through the various cellular, molecular, and biochemical processes previously discussed! More evidence that there is an optimal level of secretion and that both hyper- and hyporeactivity are adversely impacting will be addressed in discussion about the cortisol reactivity-performance relationship.

Hardiness was found to be related to global perceptions of stress (PSS) but not to other indices of stress in either a direct manner or as mediated by challenge and threat appraisals. Hardiness was predictive of challenge appraisals, but not threat appraisals. These results suggest that hardiness may exert its effects (recall challenge appraisals mediated the hardiness-SPAACQ relationship and relationships between hardiness and cortisol reactivity) by facilitating challenge appraisals while not acting at all on threat appraisals. This effect is still of great benefit. Lazarus and Folkman (1984) suggest that challenge and threat/harm/loss appraisals may not exist independently; instead, they postulate that both appraisals may be present, to varying degrees, in many situations. If primary appraisal is the result of summation of threat and challenge appraisals, then increasing challenge appraisals still has great influence on the overall primary appraisal. This is consistent with the postulated effects of hardiness, although it is intriguing not to find hardiness-stress relationships. Stress in the simulator scenario was strongly and positively related to threat appraisals, as expected.

In global relationships, stress was found to be related, in the expected directions, to performance and illness/injury. Negative life experiences and their impact were directly and negatively related to global piloting performance (GLPAPI). Negative life experiences and their impact were also positively predictive of physician-diagnosed illness and incident rates as well as total illness incident rates. Perceived stress was positively predictive of self-diagnosed injury incident rate and total illness rate. These global stress-illness relationships, however, were not mediated by physiological baselines; this is not surprising given the preceding discussion on the relationships between tonic physiological states, physiological reactivity to an acute stressor, and illness. Stress in the simulator scenario was related to decision technique (DECTEC), information seeking (INFOSOT) (as mediated by cortisol reactivity), and situational awareness (SITAWR); however, for DECTEC and SITAWR, the relationship was positive. This is puzzling. It may be that tailoring (though minimal) of the stress instrument may have damaged its validity. Another plausible explanation is that this measure of stress (Peacock & Wong, 1990) may be assessing vigilance or eustress. Vigilance, for example, should be related to situational awareness. Similarly disconcerting, this measure of stress is related to cortisol reactivity, systolic reactivity, and illness/injury in directions opposite of that indicated or suggested by the data and relationships in the data. In this sample, at least, the validity of the PSTSTR measure is questionable, and not consistent with the rest of the data. Future research efforts would do well to assess the validity of Peacock and Wong's (1990) stressfulness measure and its relationship to other measures of acute stress.

Hardiness was found to be positively related to baseline cortisol levels (for BLCOCR, r=.41, p,>1; for BLCORT, r=.37, p<.1). This counterintuitive finding is remarkably similar to that of Zorilla et al. (1995); they reported a correlation of .36 between hardiness and serum cortisol levels. Hardiness was not related to other baseline physiological measures. In the simulator scenario, hardiness was related positively to maximum diastolic blood pressure; hardiness was also related curvilinearly to cortisol reactivity in such a way that low hardy individuals showed greater deviation (both increased and decreased cortisol secretion) from baseline values in general, and more precisely, from a SCORTP value of -48.8 (-48.8% of baseline value). Hardiness was not related to various indices of cardiovascular reactivity as had been found by Allred and Smith (1989) and Contrada (1989), for example. These findings support the postulation by Zorilla et al. (1995) that although hardy individuals may have higher baseline cortisol secretion, they may demonstrate less reactivity under stress. This relationship, to the author's knowledge, has not been demonstrated elsewhere. This is prominent in that hardiness may exert its effects on health and performance through its influence on the HPA axis. That influence on the HPA axis, in turn, appears to be mediated by challenge appraisals.

A number of physiological measures were associated with performance, and the pattern is quite interesting. In general, both systolic and diastolic measures were positively related to performance and flight experience. This seems consistent with the previously discussed research which associates elevated cardiovascular tone with active coping efforts . However, systolic and diastolic *reactivity* were negatively related to several

performance outcomes on a realistic stressor. This suggests that high reactors may not only be susceptible to illness as previously discussed per the stress reactivity hypothesis, but also susceptible to performance degradation.

Cortisol reactivity was highly and curvilinearly associated with performance outcomes. Those who demonstrated cortisol secretion between roughly -30 and -50 percent of baseline typically demonstrated the best performance on dimensions which reflect application of working memory, require information consolidation and manipulation of spatial relationships, and necessitate use of declarative recall. Interestingly, if one reviews the cortisol secretion (diurnal) pattern for control subjects in previous research efforts (Kirschbaum & Hellhammer, 1994; Wittert et al., 1996), these values (-30 to -50 % of the morning baseline) approximate a level of cortisol secretion between peak and nadir. That is, the hardiest individuals and those who performed best on a number of performance dimensions in the scenario (and, as might be expected, engaged in less information-seeking (perhaps because they better retained information in working memory)) demonstrated cortisol secretions that least deviated from values mid-point between diurnal peak and nadir. This pattern of outcomes suggests that magnitude of deviation from cortisol secretion levels between diurnal peak and nadir is associated with work-related cognitive dysfunction. Furthermore, those who showed cortisol elevations performed relatively less poorly than those who showed a blunted or absent response. This observation is *absolutely consistent* with what Smriga et al. (1996) observed in their work with rats! Recall they had found that both adrenalectomy and corticosterone elevation suppressed LTP in the hippocampal dentate gyrus area, with

adrenalectomy being associated with the most profound deficit. Also, Blanchard et al. (1993) and Sapolsky (1992b) have observed blunted/sluggish HPA responses (with concomitant adverse consequences) in some rats and non-human primates, respectively, that are socially subordinate (a parallel to low hardiness?). The present findings are consistent with, and unifying of, the work of Diamond et al. (1996), Kirschbaum et al. (1996), Newcomer et al. (1994), DeKloet et al. (1994), Oitzl and DeKloet (1992), and DeKloet et al. (1992) (see pp. 80-86 of this effort for relevant literature review). This study has demonstrated the ability of cortisol reactivity to degrade performance on tasks that can be theoretically presumed to be hippocampal dependent; to the best of the author's knowledge this is an original demonstration of cortisol-related performance degradation in the workplace! It appears very much that the adverse impact of GC increase/decrease on cognitive function demonstrated in laboratory conditions by the researchers above may well occur in the workplace!

This is a disturbing finding. The behavioral correlates of blunted HPA response suggest rather severe deficits as well. The 4 individuals showing the most blunted cortisol responses (SCORTP values of -60, -76.7, -80.3, and -88.5) demonstrated the following:

-2 individuals appeared to avoid flying by passing control of the aircraft to the other crew member for more than one-third of the flight.
-1 individual flew *12 minutes* without noticing the attitude indicator was dysfunctional despite encountering air turbulence; the evaluator later debriefed this pilot about onset of vertigo.
-1 individual exceeded the main rotor RPM limit and exceeded the maximum allowable aircraft velocity.
-Individuals flew 800 ft and 1300 ft above authorized flight levels.
-2 individuals missed landing approaches.

-1 individual assumed an incorrect heading despite acknowledging the correct heading, selected the wrong approach plan for an attempted landing, and became argumentative with his crew member.
-1 individual appreciably exceeded authorized airspeed.

While the scenario was designed to be intense and effortful, all problems were solvable. These findings, despite small sample size, suggest that cortisol reactivity and its influence on cognitive function in tasks demanding of working memory and manipulation of spatial relationships may have dire, and perhaps catastrophic (in some professions), effects on performance.

To summarize, hardiness appears to be predictive of cortisol reactivity in a curvilinear fashion. Less hardy individuals tend to deviate more greatly from morning baseline cortisol levels, in general, and, particularly, from a level roughly equivalent to the midpoint between diurnal peak and nadir. Challenge appraisals appear to mediate the hardiness-cortisol reactivity relationship. Hardiness is also predictive of performance in a number of dimensions, and cortisol reactivity seems to mediate those relationships. Notably, these results (the curvilinear relationships between cortisol reactivity, hardiness, challenge appraisals, and various performance outcomes) cannot be attributed to common method variance; together, these relationships provide convergent and rather convincing evidence for the existence of a hardiness → challenge appraisal → cortisol reactivity → performance (cognitively demanding) cascade. These relationships are diagrammed in Figure 7.

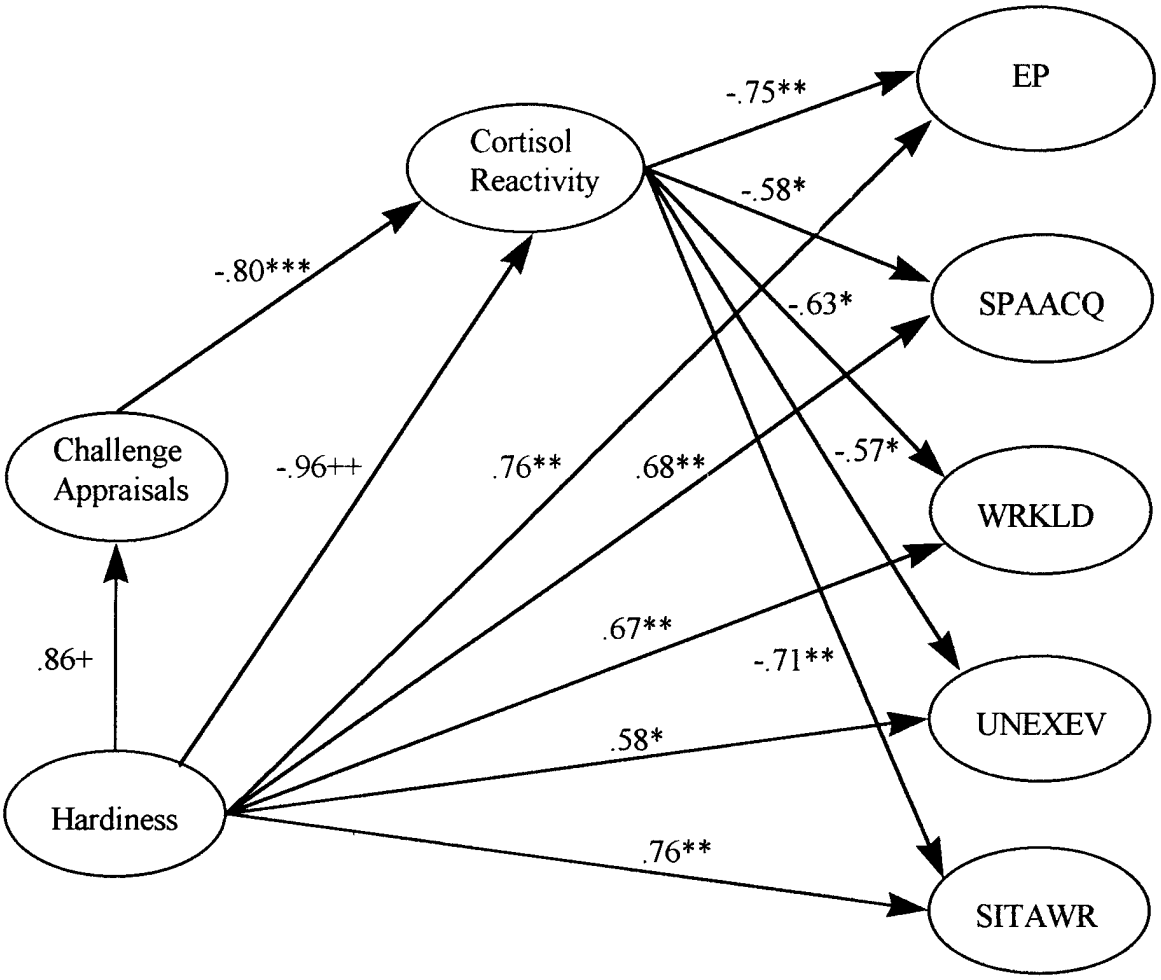

Figure 7. Bivariate relationships between hardiness, challenge appraisals, cortisol reactivity, and performance outcomes in the simulator scenario (*p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001). Cortisol reactivity is defined here by the polynomial representation given on page 184; relationship direction is based upon reactivity equating to magnitude of deviation from a SCORTP value of approximately -48.8 (value of SCORTP at greatest hardiness (136)).

In conclusion, hardiness has been shown to moderate stress-performance and stress-illness relationships as well as being directly related to performance outcomes. Hardiness was also found to be related to cortisol reactivity with less hardy individuals deviating more greatly from baseline values and, more precisely, from values that are mid-

point between the diurnal peak and nadir of cortisol secretion. This hardiness-cortisol reactivity relationship was defined by a higher order equation which captured almost 92% of the variance in the relationship. Such cortisol reactivity was, in turn, associated with illness and performance decrements, while baseline physiological tone was weakly related to illness and unrelated to performance. The relationship between cortisol reactivity and performance, if replicated, has some potentially profound implications. In pilots, it appears that the potential exists for cortisol reactivity to be associated with cognitive dysfunction severe enough to be causally implicated in major, and possibly catastrophic, aviation mishaps. In addition to the original findings associating hardiness with cortisol reactivity and demonstrating the relationship between cortisol reactivity and workplace performance, hardiness was found to change drastically over a 6 month period in an individual experiencing a tremendous amount of life change; this particular case dramatically underscores the need for future research to be of a prospective design. Hardiness was also found to be strongly related to challenge appraisals and these appraisals mediated the relationship between hardiness and performance and hardiness and cortisol reactivity.

Future reseach efforts should attempt to replicate the present findings in a more diverse and larger sample using other realistic workplace stressors to enhance generalizability. Cortisol baselines should be measured at points in the diurnal rhythm which permit detection of increases and decreases in response to stressors; that is, the a priori belief that all stress increases cortisol secretion may grossly bias (and probably already has biased) results. Peacock and Wong's (1990) stressfulness subscale of the

SAM should be employed with other measures of acute stress to assess its validity and the possibility it is measuring vigilance as well as distress. Finally, both psychologists and physiologists, in advancing this vein of research, should be aware that curvilinear relationships may predominate; further, it is the higher order explanations that appear to have the greatest parsimony and account for what have, in the past, been viewed as disparate findings. If GCs function biphasically in response to stressors, in regulation of immunological function, and in feedback to the hippocampus, and there is every reason to believe they so function, then designs constructed towards linear results can be expected to fail us while consideration of complex curvilinear relationships may well advance our level of inquiry.

Concluding Remarks

In this effort, cognitive hardiness has been shown to demonstrate both main and moderating effects upon stress-illness, stress-performance, and stress-depression relationships, academic performance, pilot performance, depression, challenge appraisals, and physiological reactivity. Indeed, hardiness appears to be a potent stress resiliency resource, and its effects appear to be mediated through behavioral response (heavier course loads, challenge appraisals of situations/events) and physiological reactivity. In fact, the relationship between hardiness and cortisol reactivity, if replicated, demonstrates a promising pathway by which hardiness, and presumably other individual and personality differences (per the work of Zorilla et al. (1995)), influence health and performance. Such replication would provide a tremendous impetus to link what are presently and largely uncoordinated efforts in the fields of psychology and psychiatry, endocrinology, neurology, and immunology. In continuing to move towards collaborative efforts across these fields, the barriers that obscure our understanding of mind/body interaction may finally begin to collapse with increased rapidity.

Returning to the construct of hardiness, Nowack's (1990) instrument was used with great success in these two studies, producing the varied and impressive findings mentioned above. Of great import, the CHS appeared in both studies to be robustly related to the theoretical conceptualization of challenge, addressing one of the greatest debates in the hardiness literature. The relationship between hardiness and increased course load in Study 1 and the relationship between hardiness and 2 measures of challenge appraisals in Study 2 suggest that Nowack's scale validly taps the challenge dimension of

hardiness. These results contribute to a rapidly growing body of literature suggesting that Nowack's instrument may be the most theoretically and psychometrically sound hardiness instrument, although, unfortunately, the CHS does not permit determination of the relative effects of control, commitment, and challenge. In future inquiry, researchers might consider inclusion of Nowack's (1990) instrument and inventories in which the components of hardiness can be individually assessed. Such a design consideration might permit better examination of CHS validity while also contributing to resolution of debate over the unity of the hardiness construct.

Finally, these two studies suggest research directions that have been little investigated. The findings in the first study suggest that measures other than intelligence tests and various tests of cognitive ability may explain/predict large proportions of variance in academic and workplace performance. Various individual differences, such as hardiness, appear to have great potential as predictors of performance, and they should be investigated more aggressively. In the second study, hardiness was found to be complexly related to cortisol reactivity, suggesting a highly logical pathway by which hardiness may affect health and performance. Additionally, an emerging body of evidence, including the results herein, suggests that cortisol reactivity may be profoundly related to cognitive function, and, it follows, performance in professions that are cognitively demanding in various ways. One must ask, "Is catastrophic pilot error in an aircraft emergency more likely if the pilot displays certain patterns of cortisol reactivity?" Another question follows, "Can detrimental physiological response patterns be avoided as a result of psychological hardiness and the appraisals made?" The results in Study 2, despite its small

sample size, are unsettling enough to warrant an urgent call for research into relationships between hardiness, appraisal processes, cortisol reactivity, and performance of tasks that are cognitively complex and/or demanding of working memory, declarative recall, and manipulation of spatial relationships.

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Appendix A

Study 1 Questionnaire

GENERAL INFORMATION

Please complete the following by filling in the blanks as appropriate.

GENDER_____ AGE (Years)_____

CLASS (Fr, So, Jr, or Sr)_____

COURSE LOAD (Total credit hours for Fall 1996 semester)_____

RACE/ETHNICITY (Optional)_____

In addition to completing the items which follow, we'd also like you to allow us to access your final semester (Fall 1996) GPA. Your GPA is an important variable in the study. If you grant your permission, your identity and GPA will be maintained in strict confidentiality. Even if you choose to not grant permission to access your semester GPA, please complete the remainder of the questionnaire.

PERMISSION TO ACCESS SEMESTER GPA (Please print clearly):

I, _____ / _____ grant
(Print Name) (Student ID—SSN)
Jon Drummond, Department of Psychology, permission to access my final semester GPA for the Fall 1996 semester. I understand that my GPA will only be accessed for this research effort. I also understand that my identity and GPA will be maintained in strict confidentiality. I understand I may revoke my permission at any time. If I have any questions or concerns, I understand I may contact Jon Drummond, Department of Psychology.

_____ _____
(Signature) (Date)

BELIEFS QUESTIONNAIRE (CHS)

Below is a list of common beliefs people hold. How strongly do you agree or disagree with each statement (1 = Strongly Agree, 2 = Agree, 3 = Neither Agree or Disagree, 4 = Disagree, 5 = Strongly Disagree)? For each item, circle the one response which best describes how strongly **you** agree or disagree with the statement.

	Strongly Agree				Strongly Disagree
1. My involvement in non-work activities and hobbies provides me with a sense of meaning and purpose.	1	2	3	4	5
2. By taking an active part in political and social affairs, people can strongly influence world events and politics.	1	2	3	4	5
3. When all else appears bleak, I can always turn to my family and friends for help and support.	1	2	3	4	5
4. I prefer to do things that are risky, exciting, and adventuresome rather than adhere to the same comfortable routine and lifestyle.	1	2	3	4	5
5. Becoming a success is mostly a matter of working hard; luck plays little or no role.	1	2	3	4	5
6. There are relatively few areas about myself in which I feel insecure, highly self-conscious, or lacking in confidence.	1	2	3	4	5
7. In general, I tend to be a bit critical, pessimistic, and cynical about most things in work, school, and life.	1	2	3	4	5
8. It would take very little change in my present circumstances at work/schoool to cause me to leave (or try to leave) this job/school.	1	2	3	4	5
9. I do not feel satisfied with my current involvement in the day-to-day activities and well-being of my family and friends.	1	2	3	4	5
10. In general, I would prefer to have things well planned out in advance rather than deal with the unknown.	1	2	3	4	5
11. Most of life is wasted in meaningless activity.	1	2	3	4	5
12. I often feel awkward, uncomfortable, or insecure interacting with others socially.	1	2	3	4	5

	Strongly Agree				Strongly Disagree
13. I rarely find myself saying out loud or thinking that I'm not good enough or capable of accomplishing something.	1	2	3	4	5
14. I am committed to my job/school/work activities that I am currently pursuing.	1	2	3	4	5
15. I tend to view most work, school, or life changes, disappointments, and setbacks as threatening, harmful, or stressful rather than challenging.	1	2	3	4	5
16. Just for variety's sake, I often explore new and different routes to places that I travel to regularly (e.g. home, school, work).	1	2	3	4	5
17. Others will act according to their own self-interests no matter what I attempt to say or do to influence them.	1	2	3	4	5
18. If I get a chance to see how others have done something or get the opportunity to be taught what to do, I am confident that I can be successful at most anything.	1	2	3	4	5
19. I expect some things to go wrong now and then, but there is little doubt in my mind that I can effectively cope with just about anything that comes my way.	1	2	3	4	5
20. Overall, most of the things that I am involved in (e.g. work, school, community, social relationships) are **not** very stimulating, enjoyable, & rewarding.	1	2	3	4	5
21. I am likely to get frustrated and upset if my plans do not unfold as I hoped, or if things do not happen the way I really want them to.	1	2	3	4	5
22. There is a direct relationship between how hard I work and the success and respect that I will have.	1	2	3	4	5
23. I don't feel that I have accomplished much lately that is really important or meaningful with respect to my future goals and objectives in life.	1	2	3	4	5
24. I often think that I am inadequate, incompetent, or less important than others with whom I work and that I know.	1	2	3	4	5
25. Many times I feel that I have little or no control and influence over things that happen to me.	1	2	3	4	5

	Strongly Agree				Strongly Disagree
26. If anything else changes or goes wrong in my life right now, I feel that I might not be able to effectively cope with it.	1	2	3	4	5
27. When change occurs at work, home, or school, I often find myself thinking that the worst is going to happen.	1	2	3	4	5
28. At the moment, things at work, home, and school are fairly predictable and any more changes would just be too much to handle.	1	2	3	4	5
29. You can't really trust that many people because most individuals are looking for ways to improve their welfare and happiness at your expense.	1	2	3	4	5
30. Most of the meaning in life comes from internal, rather than external, definitions of success, achievement, and selfsatisfaction.	1	2	3	4	5

FEELINGS AND THOUGHTS QUESTIONNAIRE (PSS)

The questions in this scale ask you about your feelings and thoughts during the last month. In each case, you will be asked to indicate *how often* you felt or thought a certain way. Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to answer each question fairly quickly. That is, don't try to count up the number of times you felt a particular way, but rather indicate (circle) the alternative that seems like a reasonable estimate. For each question, choose from the following alternatives:

1 = Never
2 = Almost Never
3 = Sometimes
4 = Fairly Often
5 = Very Often

	Never				Very Often
1. In the last month, how often have you been upset because of something that happened unexpectedly?	1	2	3	4	5
2. In the last month, how often have you felt that you were unable to control the important things in your life?	1	2	3	4	5
3. In the last month, how often have you felt nervous and "stressed?"	1	2	3	4	5
4. In the last month, how often have you dealt successfully with irritating life hassles?	1	2	3	4	5
5. In the last month, how often have you felt that you were effectively coping with important changes that were occurring in your life?	1	2	3	4	5
6. In the last month, how often have you felt confident about your ability to handle your personal problems?	1	2	3	4	5
7. In the last month, how often have you felt that things were going your way?	1	2	3	4	5
8. In the last month, how often have you found that you could not cope with all the things that you had to do?	1	2	3	4	5
9. In the last month, how often have you been able to control irritations in your life?	1	2	3	4	5
10. In the last month, how often have you felt that you were on top of things?	1	2	3	4	5
11. In the last month, how often have you been angered because of things that happened that were outside of your control?	1	2	3	4	5

	Never				Very Often
12. In the last month, how often have you found yourself thinking about things that you have to accomplish?	1	2	3	4	5
13. In the last month, how often have you been able to control the way you spend your time?	1	2	3	4	5
14. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?	1	2	3	4	5

Appendix B

Study 2 Initial Questionnaire

Participant #_____ Last Four SSN_____

INITIAL QUESTIONNAIRE

Please complete the attached pages. You should have 7 pages (including this one) and 3 parts to the questionnaire. If you do not have a complete questionnaire, please inform Capt Drummond (Duty ph: 532-6850, Home ph: 587-8403). Please answer all questions and print clearly. Seal the completed questionnaire in the envelope provided, and return it to Capt Drummond. Thank you for your participation.

PART I. (Demographic Data)

Please print clearly when providing the following information.

Participant #_____Last Four SSN_____Rank_____Name (last, first)_____

Rater's Rank_____ Rater's Name (last)_____

Gender_____ Age_____ Date of Birth_____

Total time on active duty: Years_____ Months_____

Total Time Assigned to This Unit: Years_____ Months_____

Flying hours (all aircraft)_____ Flying hours (UH-1 only)_____

Simulator hours (all aircraft)_____ Simulator hours (UH-1 only)_____

Education (check one):

High School_____	0-2 Years College_____
Associate's Degree_____	Associate's Degree + additional course work_____
Bachelor's Degree_____	Bachelor's Degree + Some Graduate Work_____
Master's Degree_____	Master's Degree + Some Doctoral Work_____
PhD._____	Other (please specify)_____

Marital Status_____

The following questions address important considerations for lab analysis should you agree to provide urine samples. Again, your responses will remain confidential.

Do you smoke, dip, chew, or otherwise use tobacco on a regular basis?_____

Have you consumed any coffee (or other caffeine source) today?_____ If yes, how much coffee (caffeine) have you consumed today?_____ If the caffeine source was other than coffee, please indicate what it was_____

Have you been diagnosed with hepatitis B in the last 12 months?_____ If yes, when, and what is your current status_____

Have you ever been diagnosed with Post Traumatic Stress Disorder (PTSD)?_____ If yes, when?_____

Are you currently taking any steroid medications? (prednisone, hydrocortisone, dexamethasone, etc.)_____

If you answered "yes" to the preceding question, please indicate the medication being taken_____

Would you be willing to provide urine samples for this research (NOTE: Urine will be analyzed for concentrations of a naturally occuring steroid produced by your adrenal glands and a substance associated with caffeine consumption. No other analysis will be conducted. Your confidentiality will be guaranteed. Lab results will not be placed in your medical records. In the event abnormally high or low levels of the steroid are discovered, you will be notified privately so you can pursue medical attention if you so choose.)?_____

GO ON TO PART II.

PART II. (CHS)

Below is a list of common beliefs people hold. How strongly do **you** agree or disagree with each statement (1 = Strongly Agree, 2 = Agree, 3 = Neither Agree or Disagree, 4 = Disagree, 5 = Strongly Disagree)? For each item, circle the one response which best describes how strongly you agree or disagree with the statement.

	Strongly Agree				Strongly Disagree
1. My involvement in non-work activities and hobbies provides me with a sense of meaning and purpose.	1	2	3	4	5
2. By taking an active part in political and social affairs, people can strongly influence world events and politics.	1	2	3	4	5
3. When all else appears bleak, I can always turn to my family and friends for help and support.	1	2	3	4	5
4. I prefer to do things that are risky, exciting, and adventuresome rather than adhere to the same comfortable routine and lifestyle.	1	2	3	4	5
5. Becoming a success is mostly a matter of working hard; luck plays little or no role.	1	2	3	4	5
6. There are relatively few areas about myself in which I feel insecure, highly self-conscious, or lacking in confidence.	1	2	3	4	5
7. In general, I tend to be a bit critical, pessimistic, and cynical about most things in work and life.	1	2	3	4	5
8. It would take very little change in my present circumstances at work to cause me to leave (or try to leave) either this unit or the U.S. Army.	1	2	3	4	5
9. I do not feel satisfied with my current involvement in the day-to-day activities and well-being of my family and friends.	1	2	3	4	5
10. In general, I would prefer to have things well planned out in advance rather than deal with the unknown.	1	2	3	4	5
11. Most of life is wasted in meaningless activity.	1	2	3	4	5
12. I often feel awkward, uncomfortable, or insecure interacting with others socially.	1	2	3	4	5

	Strongly Agree				Strongly Disagree
13. I rarely find myself saying out loud or thinking that I'm not good enough or capable of accomplishing something.	1	2	3	4	5
14. I am committed to my job and work activities that I am currently pursuing.	1	2	3	4	5
15. I tend to view most work and life changes, disappointments and setbacks as threatening, harmful, or stressful rather than challenging.	1	2	3	4	5
16. Just for variety's sake, I often explore new and different routes to places that I travel to regularly (e.g. home, work).	1	2	3	4	5
17. Others will act according to their own self-interests no matter what I attempt to say or do to influence them.	1	2	3	4	5
18. If I get a chance to see how others have done something or get the opportunity to be taught what to do, I am confident that I can be successful at most anything.	1	2	3	4	5
19. I expect some things to go wrong now and then, but there is little doubt in my mind that I can effectively cope with just about anything that comes my way.	1	2	3	4	5
20. Overall, most of the things that I am involved in (e. g. work, community, social relationships) are **not** very stimulating, enjoyable, & rewarding.	1	2	3	4	5
21. I am likely to get frustrated and upset if my plans do not unfold as I hoped, or if things do not happen the way I really want them to.	1	2	3	4	5
22. There is a direct relationship between how hard I work and the success and respect that I will have.	1	2	3	4	5
23. I don't feel that I have accomplished much lately that is really important or meaningful with respect to my future goals and objectives in life.	1	2	3	4	5
24. I often think that I am inadequate, incompetent, or less important than others with whom I work and that I know.	1	2	3	4	5

	Strongly Agree				Strongly Disagree
25. Many times I feel that I have little or no control and influence over things that happen to me.	1	2	3	4	5
26. If anything else changes or goes wrong in my life right now, I feel that I might not be able to effectively cope with it.	1	2	3	4	5
27. When change occurs at work or home I often find myself thinking that the worst is going to happen.	1	2	3	4	5
28. At the moment, things at work and at home are fairly predictable and any more changes would just be too much to handle.	1	2	3	4	5
29. You can't really trust that many people because most individuals are looking for ways to improve their welfare and happiness at your expense.	1	2	3	4	5
30. Most of the meaning in life comes from internal, rather than external, definitions of success, achievement, and self-satisfaction.	1	2	3	4	5

GO ON TO PART III

PART III. (PSS)

The questions in this scale ask you about **your** feelings and thoughts during the last month. In each case, you will be asked to indicate *how often* you felt or thought a certain way. Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to answer each question fairly quickly. That is, don't try to count up the number of times you felt a particular way, but rather indicate (circle) the alternative that seems like a reasonable estimate. For each question, choose from the following alternatives:

1 = Never
2 = Almost Never
3 = Sometimes
4 = Fairly Often
5 = Very Often

	Never				Very Often
1. In the last month, how often have you been upset because of something that happened unexpectedly?	1	2	3	4	5
2. In the last month, how often have you felt that you were unable to control the important things in your life?	1	2	3	4	5
3. In the last month, how often have you felt nervous and "stressed?"	1	2	3	4	5
4. In the last month, how often have you dealt successfully with irritating life hassles?	1	2	3	4	5
5. In the last month, how often have you felt that you were effectively coping with important changes that were occurring in your life?	1	2	3	4	5
6. In the last month, how often have you felt confident about your ability to handle your personal problems?	1	2	3	4	5
7. In the last month, how often have you felt that thing were going your way?	1	2	3	4	5
8. In the last month, how often have you found that you could not cope with all the things that you had to do?	1	2	3	4	5
9. In the last month, how often have you been able to control irritations in your life?	1	2	3	4	5
10. In the last month, how often have you felt that you were on top of things?	1	2	3	4	5

	Never				Very Often
11. In the last month, how often have you been angered because of things that happened that were outside of your control?	1	2	3	4	5
12. In the last month, how often have you found yourself thinking about things that you have to accomplish?	1	2	3	4	5
13. In the last month, how often have you been able to control the way you spend your time?	1	2	3	4	5
14. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?	1	2	3	4	5

THANK YOU FOR YOUR PARTICIPATION

Appendix C

Illness/Injury Diary Sheet and Instructions

ILLNESS/INJURY DIARY INSTRUCTIONS

You have been provided an initial packet of 20 sheets to track all illnesses and injuries you have during the course of this study. Completed illness/injury diary sheets will be collected every two (2) weeks. At the dates indicated below, please collect all completed sheets, seal them in the appropriate envelope (envelopes are dated), and give the sealed envelopes to XXXXXXX who will, in turn, pass them to Capt Drummond. (Note: XXXXXXX will not open the envelope or examine its contents). If you have had no illness or injury, use one of the sheets to indicate this; whether you have been ill/injured or not, a response is required. If you should need additional sheets, please feel free to either copy blank sheets before they are used or contact Capt Jon Drummond (532-6850, 587-8403). It is vitally important you maintain this diary until it is collected. Please continue to document the diary even if you go TDY.

When completing the diary, please be as accurate as possible in the space provided, especially in the Diagnosis/Description and Date blocks. This is best done by documenting an illness/injury when it occurs. If you should not wish to disclose the nature of a certain illness, please provide some indication as to the type of illness; for example, you might indicate "bacterial infection treated with antibiotics" as a more general way of conveying an illness. Of course, an accurate description is more helpful, and remember, your confidentiality will be maintained.

To clarify, "physician diagnosed" refers to an illness diagnosed and/or treated by a licensed medical practitioner. "Self-Diagnosed" refers to an injury or illness not being treated by a medical practitioner; an example of this might be a cold, headache, or hay fever which you self-diagnose but for which you do not seek medical treatment. Also, "Date(s) of missed duty" should be completed if the illness/injury (or treatment of that illness/injury) causes you to be away from duty 4 or more hours in a given day.

What constitutes an illness/injury? When should you document an illness/injury? As a general rule, please document any illness or injury if **you** believe it prevents you from performing your duty to your fullest potential or in some way interferes with your being able to do those things you wish to do in the way you'd like to do them. This is intended to be subjective; it is expected you might document some things another may not and vice versa, depending on how it uniquely affects you.

Please contact Capt Jon Drummond at any time if you have any questions or concerns about maintaining this diary. Again, a detailed diary is most beneficial to this research. Thank you for your participation.

DATES OF ILLNESS/INJURY COMPLETION/TURN-IN:

9 August
23 August
6 September
20 September
4 October
Final Turn-in (Date TBD)

ILLNESS/INJURY DIARY FOR

PARTICIPANT #_____LAST FOUR SSN_____ RANK_____

NAME_____

Date(s) of Illness/Injury:

Check one: Physician Diagnosed_____ Self-Diagnosed_____

Brief Diagnosis/Description of Illness/Injury:

Date(s) of missed duty:

Duty Not Including Flying (DNIF)?_____ If yes, date(s) DNIF:

Any other comments you'd like to make about this illness/injury:

Date(s) of Illness/Injury:

Check one: Physician Diagnosed_____ Self-Diagnosed_____

Brief Diagnosis/Description of Illness/Injury:

Date(s) of missed duty:

DNIF?_____ If yes, date(s) DNIF:

Any other comments you'd like to make about this illness/injury:

Appendix D

Simulator Scenario Questionnaires

Participant #_____Last Four SSN_____Rank_____Name (last, first)_____

PRE-SIMULATOR QUESTIONNAIRE

You are about to participate in an intense simulator exercise which will severely test your abilities as a pilot. Your blood pressure and pulse will be monitored periodically throughout the 1.5 to 2.0 hour scenario. Your performance in this scenario may affect your future training requirements. It is in your best interest to perform as well as you possibly can.

Please empty your bladder now if you have not done so in the last 30 minutes.

Before the blood pressure cuff is placed on your arm and the simulator scenario begins, you will need to answer some questions. Please indicate how strongly **you** agree or disagree with the following statements; for each statement, circle the one response which best describes how strongly you agree or disagree with the statement (1 = Strongly Agree, 2 = Agree, 3 = Neither Agree or Disagree, 4 = Disagree, 5 = Strongly Disagree).

		Strongly Agree				Strongly Disagree
1.	I am looking forward to the scenario.	1	2	3	4	5
2.	I wish I didn't have to do this scenario now.	1	2	3	4	5
3.	I am worried that I would be adversely impacted by failure or a poor performance in the scenario.	1	2	3	4	5
4.	I am excited this scenario may vigorously challenge my skills and abilities as a pilot.	1	2	3	4	5
5.	I feel threatened by the upcoming scenario and its potential consequences.	1	2	3	4	5
6.	I believe my career and/or future training may be positively impacted by a strong performance in the scenario.	1	2	3	4	5
7.	I feel anxious.	1	2	3	4	5
8.	I believe this experience will positively impact my career and/or professional knowledge and experience.	1	2	3	4	5
9.	I am concerned my performance in this scenario may be poor and negatively impact my career and future training requirements.	1	2	3	4	5
10.	Whatever the outcome, I believe my career and future training requirements will somehow be affected by my performance in this scenario.	1	2	3	4	5

	Strongly Agree				Strongly Disagree
11. The implications of either doing well or poorly on the scenario appear to be somewhat serious.	1	2	3	4	5
12. I am worried about a negative outcome in this scenario.	1	2	3	4	5
13. I am excited to see how I will perform in this scenario.	1	2	3	4	5
14. I believe there may be some long-term consequences of my performance in this scenario.	1	2	3	4	5
15. I expect to learn something valuable in the pending scenario.	1	2	3	4	5
16. I can handle anything and everything thrown at me.	1	2	3	4	5
17. I know the limits of my abilities, and I am concerned the scenario might be designed so as to exceed my abilities and skills.	1	2	3	4	5

Participant #_____Last Four SSN_____Rank_____Name (last, first)_____

POST-SIMULATOR QUESTIONNAIRE (ESRQ)

Below is a list of words describing different emotions. Beside each word are four response choices. Circle the choice which best describes how **you** felt during the most intense part of the scenario you have just completed. Respond with (circle) the choice that first comes to your mind. The choices are as follows:

1 = The word does not describe how I felt.
2 = The word partly corresponds to how I felt.
3 = The word fairly well corresponds to how I felt.
4 = The word completely corresponds to how I felt.

1. Indifferent	1	2	3	4
2. Happy	1	2	3	4
3. Relaxed	1	2	3	4
4. Satisfied	1	2	3	4
5. Bright	1	2	3	4
6. Concentrated	1	2	3	4
7. Sharp	1	2	3	4
8. Vigorous	1	2	3	4
9. Angry	1	2	3	4
10. Disappointed	1	2	3	4
11. Heated	1	2	3	4
12. Mad	1	2	3	4
13. Uncertain	1	2	3	4
14. Worried	1	2	3	4

Please turn in this questionnaire. You will then be given a container and asked to provide a urine sample. Please do not discuss with anyone the scenario you have just completed. Thank you for your participation.

Part. #_____ Last Four SSN_____Rank_____Name (last, first)_____

SCENARIO EVALUATION (Stressfulness)

Below are a few statements evaluating the scenario you have just completed. Please indicate how strongly **you** agree or disagree with the following statements; for each statement, circle the one response which best describes how strongly you agree or disagree with the statement (1 = Strongly Agree, 2 = Agree, 3 = Neither Agree or Disagree, 4 = Disagree, 5 = Strongly Disagree).

		Strongly Agree				Strongly Disagree
1.	This scenario created tension in me.	1	2	3	4	5
2.	This scenario taxed or exceeded my abilities.	1	2	3	4	5
3.	I perceived this situation as stressful.	1	2	3	4	5
4.	This scenario required much effort to succeed.	1	2	3	4	5

Appendix E

Study 2 Final Questionnaire

Participant #_____ Last Four SSN_____ Name_____

Date_____

FINAL QUESTIONNAIRE

Please complete the attached pages. You should have 11 pages (including this one) and 4 parts to the questionnaire. If you do not have a complete questionnaire, please inform Capt Drummond (Duty ph: 532-6850, Home ph: 587-8403). Please answer all questions and print clearly. Seal the completed questionnaire in the envelope provided, and return it to Capt Drummond. Thank you for your participation.

PART I.

Please print clearly when providing the following information.

Participant #_____Last Four SSN_____Rank_____Name (last, first)_____

Rater's Rank_____ Rater's Name (last)_____

Gender_____ Age_____ Date of Birth_____

Total time on active duty: Years_____ Months_____

Total Time Assigned to This Unit: Years_____ Months_____

Flying hours (all aircraft)_____ Flying hours (UH-1 only)_____

Simulator hours (all aircraft)_____ Simulator hours (UH-1 only)_____

Marital Status_____

Do you smoke, dip, chew, or otherwise use tobacco on a regular basis?_____

GO ON TO PART II.

(Note: Parts II (CHS) and III (PSS) are not included in this Appendix.
Refer to Appendix B to review those measures.)

PART IV. (LES)

Listed below are a number of events which sometimes bring about change in the lives of those who experience them and which necessitate social readjustment. *Please check only those events which you have experienced in the last six (6) months.* Be sure to place all check marks on the appropriate line before the item number or letter.

Also, for each item checked below, *please indicate (circle) the extent to which you viewed the event as having either a positive or negative impact on your life* at the time the event occurred. That is, *indicate the type and extent of impact that the event had.* A rating of -3 would indicate an extremely negative impact. A rating of 0 suggests no impact either positive or negative. A rating of +3 would indicate an extremely positive impact.

	Extremely Negative		Somewhat Negative		Slightly Posivtive		Extremely Positive
		Moderately Negative		No Impact		Moderately Positive	
_____1. Marriage	-3	-2	-1	0	+1	+2	+3
_____2. Detention in jail or a comparable institution	-3	-2	-1	0	+1	+2	+3
_____3. Death of spouse	-3	-2	-1	0	+1	+2	+3
_____4. Major change in sleeping habits (either much more or much less sleep)	-3	-2	-1	0	+1	+2	+3
5. Death of close family member:							
_____a. Mother	-3	-2	-1	0	+1	+2	+3
_____b. Father	-3	-2	-1	0	+1	+2	+3
_____c. Brother	-3	-2	-1	0	+1	+2	+3
_____d. Sister	-3	-2	-1	0	+1	+2	+3
_____e. Grandmother	-3	-2	-1	0	+1	+2	+3
_____f. Grandfather	-3	-2	-1	0	+1	+2	+3
_____g. other (specify)	-3	-2	-1	0	+1	+2	+3
_____6. Major change in eating habits (much more or less food intake)	-3	-2	-1	0	+1	+2	+3
_____7. Foreclosure on mortgage or loan	-3	-2	-1	0	+1	+2	+3
_____8. Death of close friend	-3	-2	-1	0	+1	+2	+3
_____9. Outstanding personal achievement	-3	-2	-1	0	+1	+2	+3
_____10. Minor law violations (traffic/parking tickets, disturbing the peace, etc.)	-3	-2	-1	0	+1	+2	+3

	Extremely Negative		Somewhat Negative		Slightly Positive		Extremely Positive
		Moderately Negative		No Impact		Moderately Positive	
_____11. *Male:* Wife/girlfriend's pregnancy	-3	-2	-1	0	+1	+2	+3
_____12. *Female:* Pregnancy	-3	-2	-1	0	+1	+2	+3
_____13. Changed work situation (different work responsibility, major change in working conditions, working hours, etc.)	-3	-2	-1	0	+1	+2	+3
_____14. New job/position	-3	-2	-1	0	+1	+2	+3
15. Serious illness or injury of close family member:							
_____a. Father	-3	-2	-1	0	+1	+2	+3
_____b. Mother	-3	-2	-1	0	+1	+2	+3
_____c. Sister	-3	-2	-1	0	+1	+2	+3
_____d. Brother	-3	-2	-1	0	+1	+2	+3
_____e. Grandfather	-3	-2	-1	0	+1	+2	+3
_____f. Grandmother	-3	-2	-1	0	+1	+2	+3
_____g. Spouse	-3	-2	-1	0	+1	+2	+3
_____h. Other (specify)	-3	-2	-1	0	+1	+2	+3
_____16. Sexual difficulties	-3	-2	-1	0	+1	+2	+3
_____17. Trouble with employer/ boss (in danger of losing job, being suspended, demoted, reprimanded, etc.)	-3	-2	-1	0	+1	+2	+3
_____18. Trouble with in-laws	-3	-2	-1	0	+1	+2	+3
_____19. Major change in financial status (a lot better off or a lot worse off)	-3	-2	-1	0	+1	+2	+3
_____20. Major change in closeness of family members (increased or decreased closeness)	-3	-2	-1	0	+1	+2	+3
_____21. Gaining a new family member (through birth, adoption, family member moving in, etc.)	-3	-2	-1	0	+1	+2	+3
_____22. Change of residence	-3	-2	-1	0	+1	+2	+3
_____23. Marital separation from mate (due to conflict)	-3	-2	-1	0	+1	+2	+3

	Extremely Negative		Somewhat Negative		Slightly Positive		Extremely Positive
		Moderately Negative		No Impact		Moderately Positive	
_____24. Major change in church activities (increased or decreased attendance)	-3	-2	-1	0	+1	+2	+3
_____25. Marital reconciliation with mate	-3	-2	-1	0	+1	+2	+3
_____26. Major change in number of arguments with spouse (a lot more or a lot less arguments)	-3	-2	-1	0	+1	+2	+3
_____27. *Married Male:* Change in wife's work outside the home (beginning work, ceasing work, changing to a new job, etc.)	-3	-2	-1	0	+1	+2	+3
_____28. *Married female:* Change in husband's work (loss of job, beginning new job, retirement, etc.)	-3	-2	-1	0	+1	+2	+3
_____29. Major change in usual type and/or amount of recreation	-3	-2	-1	0	+1	+2	+3
_____30. Borrowing more than $23,000 (buying home, business, etc.)	-3	-2	-1	0	+1	+2	+3
_____31. Borrowing less than $23,000 (buying car, TV/stereo systems, various loan, etc.)	-3	-2	-1	0	+1	+2	+3
_____32. Being fired from job position	-3	-2	-1	0	+1	+2	+3
_____33. *Male:* Wife/girlfriend having abortion	-3	-2	-1	0	+1	+2	+3
_____34. *Female:* Having abortion	-3	-2	-1	0	+1	+2	+3
_____35. Major personal illness or injury	-3	-2	-1	0	+1	+2	+3

	Extremely Negative	Moderately Negative	Somewhat Negative	No Impact	Slightly Positive	Moderately Positive	Extremely Positive
_____36. Major change in social activities, e.g. parties, movies, visiting (increased or decreased participation)	-3	-2	-1	0	+1	+2	+3
_____37. Major change in living conditions of family (building new home, remodeling, deterioration of home, neighborhood, etc.)	-3	-2	-1	0	+1	+2	+3
_____38. Divorce	-3	-2	-1	0	+1	+2	+3
_____39. Serious illness or injury of close friend	-3	-2	-1	0	+1	+2	+3
_____40. TDYs; please provide number of TDYs and total number of days on TDY in the last 6 months.	-3	-2	-1	0	+1	+2	+3
# TDYs_____ # days total_____							
_____41. Son or daughter leaving home (due to marriage, college, etc.)	-3	-2	-1	0	+1	+2	+3
_____42. Ending of formal schooling (civilian, professional military education, etc.)	-3	-2	-1	0	+1	+2	+3
_____43. Separation from spouse (due to work, travel, etc.)	-3	-2	-1	0	+1	+2	+3
_____44. Engagement	-3	-2	-1	0	+1	+2	+3
_____45. Breaking up with boyfriend/ girlfriend	-3	-2	-1	0	+1	+2	+3
_____46. Leaving home for the first time	-3	-2	-1	0	+1	+2	+3
_____47. Reconciliation with boyfriend/ girlfriend	-3	-2	-1	0	+1	+2	+3

	Extremely Negative	Moderately Negative	Somewhat Negative	No Impact	Slightly Positive	Moderately Positive	Extremely Positive
Other recent events which have had an impact on your life. List and rate.							
_____48._____							
_____							
_____	-3	-2	-1	0	+1	+2	+3
_____49._____							
_____	-3	-2	-1	0	+1	+2	+3
_____50._____							
_____	-3	-2	-1	0	+1	+2	+3

THANK YOU FOR YOUR PARTICIPATION

Appendix F

Global Appraisal, Pilot Performance (GLPAPI)

PILOT PERFORMANCE EVALUATION

YOUR NAME AND RANK_____ DATE_____

Listed below are several individuals participating in the study for which you have provided assistance. For each of the below individuals, I'd like you to consider their overall performance as a pilot over the last 8 months (or from August 96 until their PCS/separation). Consider all knowledge, skills, abilities, performance of tasks/procedures, aircrew coordination, professionalism, ability to handle pressure/task saturation/information overload, etc. Based on your observations as a trainer, evaluator, and fellow pilot, rate the individual's performance as a pilot using the following scale. Please put the number of your overall rating in the space next to the ratee's last name. If you feel you have not observed the individual enough to rate him/her, please put an "NR" (No Rating) in the space next to their last name. (NOTE: Your responses will be maintained in confidentiality; Capt Drummond is the only one who will view them.)

10 = superior performer; one of the very best I've seen; in a hazardous/combat scenario, I would want this person flying with me
9 = excellent performer; always exceeds standards
8 = above average performer; consistently exceeds standards
7 = slightly above average performer; often exceeds standards
6 = average performer; meets standards
5 = average performer; meets standards
4 = slightly below average performer; meets standards
3 = below average performer; meets minimal standards
2 = poor performer; often meets minimal standards
1 = unsatisfactory performer; one of the worst I've seen; I am not comfortable flying with this person

Appendix G

Study 2 Correlation Matrices

Table G1
Means, Standard Deviations, and Intercorrelations for Global Variables

Variables	N	Mean	Std Dev	1	2
1. CH1	23	118.17	15.13		
2. PSS1	23	33.13	7.50	-.77++	
3. LESNGIN	14	2.29	2.49	-.41	.67***
4. LESNG	14	3.43	4.03	-.26	.58**
5. LESPSIN	14	2.21	1.58	-.02	.10
6. LESPS	14	5.14	4.11	.03	.00
7. BLHR	23	65.93	7.14	.09	-.06
8. BLSBP	23	124.29	12.06	.02	.07
9. BLDBP	23	78.68	8.62	-.05	.26
10. BLCOCR	22	4.87	2.04	.41*	-.43**
11. BLCORT	22	8.08	3.87	.37*	-.40*
12. ILPDDYS	21	.0105	.0264	-.40*	.41*
13. ILPINDY	21	.0014	.0036	-.38*	.39*
14. ILSDDYS	21	.0114	.0344	-.13	.28
15. ILSINDY	21	.0052	.0160	-.36	.36
16. TOILDYS	21	.0219	.0468	-.32	.44**
17. TILINDY	21	.0067	.0180	-.40*	.40*
18. INPDDYS	21	.0324	.0943	-.18	-.07
19. INPINDY	21	.0024	.0054	-.11	-.06
20. INSDDYS	21	.0190	.0487	.01	.17
21. INSINDY	21	.0029	.0064	-.19	.36
22. TOINDYS	21	.0154	.1085	-.15	.02
23. TININDY	21	.0057	.0108	-.12	.13
24. TODYS	21	.0729	.1165	-.26	.19
25. TINDY	21	.0124	.0253	-.33	.34
26. GLOPA	20	21.60	2.05	.22	.02
27. PROCOMP	20	4.90	.28	-.02	.35
28. PERFRAT	20	4.60	.75	.23	.02
29. OFFPOT	20	3.70	.47	.38*	-.06
30. POTEVAL	20	8.40	.75	.15	-.05
31. GLPAPI	20	6.79	1.63	.07	-.35
32. SIMALL	21	121.13	35.41	.25	.04
33. SIMUH1	22	111.30	32.22	.07	-.10
34. TIMEAD	21	97.14	41.94	-.20	.19
35. MARITAL	23	.83	.39	-.12	-.20
36. EDUC	22	3.82	1.59	.14	.07
37. TOBAC	23	.17	.39	.13	.04

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G1 (cont.)

Variables	3	4	5	6	7
1. CH1					
2. PSS1					
3. LESNGIN					
4. LESNG	.89++				
5. LESPSIN	-.17	.04			
6. LESPS	-.19	.04	.97++		
7. BLHR	-.38	-.16	.44	.45	
8. BLSBP	-.27	-.22	.38	.38	.24
9. BLDBP	-.39	-.30	.16	.24	.34
10. BLCOCR	-.53**	-.38	.64**	.70***	.57***
11. BLCORT	-.21	.04	.44	.49*	.69++
12. ILPDDYS	.66**	.66**	.17	.17	-.12
13. ILPINDY	.66***	.66**	.16	.16	-.11
14. ILSDDYS	.07	-.08	.10	.14	-.03
15. ILSINDY	.49*	.18	-.26	-.22	-.20
16. TOILDYS	.43	.32	.18	.20	-.09
17. TILINDY	.57**	.29	-.20	-.17	-.20
18. INPDDYS	-.20	-.24	-.04	-.03	-.02
19. INPINDY	-.06	-.18	-.23	-.20	-.22
20. INSDDYS	-.10	-.16	-.50*	-.44	-.46**
21. INSINDY	.49*	.19	-.56**	-.50*	-.53**
22. TOINDYS	-.21	-.26	-.13	-.11	-.23
23. TININDY	.24	.00	-.45	-.40	-.49**
24. TODYS	-.01	-.10	-.04	-.02	-.25
25. TINDY	.50*	.21	-.30	-.26	-.35
26. GLOPA	-.03	-.21	-.07	-.08	-.16
27. PROCOMP	.30	.24	.17	.15	-.13
28. PERFRAT	.04	-.13	-.14	-.11	-.18
29. OFFPOT	.01	-.22	-.22	-.22	-.14
30. POTEVAL	-.24	-.41	.00	-.06	-.13
31. GLPAPI	-.58**	-.68***	.24	.19	.10
32. SIMALL	-.59**	-.54*	-.10	-.10	-.08
33. SIMUH1	-.58**	-.61**	-.21	-.24	-.12
34. TIMEAD	-.13	-.06	-.11	-.12	.09
35. MARITAL	.13	-.06	-.48*	-.55**	.00
36. EDUC	.32	.40	-.13	.03	-.14
37. TOBAC	.65**	.46*	-.30	-.38	-.03

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G1 (cont.)

Variables	8	9	10	11	12
1. CH1					
2. PSS1					
3. LESNGIN					
4. LESNG					
5. LESPSIN					
6. LESPS					
7. BLHR					
8. BLSBP					
9. BLDBP	.77++				
10. BLCOCR	.36*	.17			
11. BLCORT	.35	.22	.71++		
12. ILPDDYS	-.19	-.41*	-.24	-.18	
13. ILPINDY	-.20	-.40*	-.23	-.17	1.00++
14. ILSDDYS	-.36	-.25	.21	-.16	.17
15. ILSINDY	-.33	-.31	-.10	-.25	.44**
16. TOILDYS	-.37	-.41*	.02	-.22	.69++
17. TILINDY	-.33	-.35	-.14	-.25	.59+
18. INPDDYS	-.24	-.30	.12	.06	-.03
19. INPINDY	-.38*	-.40*	-.06	-.08	.06
20. INSDDYS	-.17	-.13	-.30	-.31	-.11
21. INSINDY	-.27	-.24	-.42*	-.45**	.23
22. TOINDYS	-.29	-.31	-.03	-.09	-.08
23. TININDY	-.42*	-.40*	-.31	-.29	.15
24. TODYS	-.41*	-.45**	-.02	-.17	.21
25. TINDY	-.41*	-.42*	-.23	-.31	.49**
26. GLOPA	.31	.30	-.14	-.23	-.38
27. PROCOMP	.29	.18	-.25	-.24	.13
28. PERFRAT	.31	.36	-.12	-.19	-.39*
29. OFFPOT	.26	.30	-.12	-.13	-.41*
30. POTEVAL	.25	.19	-.10	-.25	-.43*
31. GLPAPI	.13	-.09	.23	.00	-.30
32. SIMALL	.09	.32	-.08	-.08	-.41*
33. SIMUH1	-.32	.14	-.10	-.24	-.50**
34. TIMEAD	.27	.31	-.35	-.05	-.05
35. MARITAL	-.14	-.20	-.17	.04	-.13
36. EDUC	.10	.37*	-.08	.00	.09
37. TOBAC	-.14	-.21	-.14	-.08	.13

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G1 (cont.)

Variables	13	14	15	16	17
1. CH1					
2. PSS1					
3. LESNGIN					
4. LESNG					
5. LESPSIN					
6. LESPS					
7. BLHR					
8. BLSBP					
9. BLDBP					
10. BLCOCR					
11. BLCORT					
12. ILPDDYS					
13. ILPINDY					
14. ILSDDYS	.18				
15. ILSINDY	.47**	.69++			
16. TOILDYS	.70++	.83++	.75++		
17. TILINDY	.62+	.65+	.98++	.81++	
18. INPDDYS	-.03	.11	.35	.06	.30
19. INPINDY	.07	.17	.49**	.16	.45**
20. INSDDYS	-.11	-.09	-.04	-.13	-.06
21. INSINDY	.25	.21	.53**	.28	.52**
22. TOINDYS	-.07	.05	.28	.00	.24
23. TININDY	.17	.19	.54**	.23	.52**
24. TODYS	.22	.38*	.56***	.40*	.54**
25. TINDY	.51**	.54**	.93++	.67++	.93++
26. GLOPA	-.37	.09	-.07	-.15	-.14
27. PROCOMP	.13	.04	-.08	.10	-.05
28. PERFRAT	-.37	.10	.00	-.15	-.07
29. OFFPOT	-.40*	.15	.07	-.12	-.02
30. POTEVAL	-.44*	.04	-.20	-.21	-.26
31. GLPAPI	-.32	.05	-.21	-.14	-.25
32. SIMALL	-.41*	.14	-.22	-.13	-.28
33. SIMUH1	-.51**	.26	-.14	-.09	-.23
34. TIMEAD	-.07	-.45*	-.30	-.37	-.28
35. MARITAL	-.15	-.34	.01	-.32	-.02
36. EDUC	.12	.09	.08	.10	.09
37. TOBAC	.15	.12	.38*	.17	.37

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G1 (cont.)

Variables	18	19	20	21	22
1. CH1					
2. PSS1					
3. LESNGIN					
4. LESNG					
5. LESPSIN					
6. LESPS					
7. BLHR					
8. BLSBP					
9. BLDBP					
10. BLCOCR					
11. BLCORT					
12. ILPDDYS					
13. ILPINDY					
14. ILSDDYS					
15. ILSINDY					
16. TOILDYS					
17. TILINDY					
18. INPDDYS					
19. INPINDY	.92++				
20. INSDDYS	.06	.22			
21. INSINDY	.12	.37	.79++		
22. TOINDYS	.89++	.90++	.50**	.46**	
23. TININDY	.55***	.79++	.67++	.84++	.78++
24. TODYS	.84++	.89++	.41*	.54**	.92++
25. TINDY	.45**	.65+	.24	.72++	.50**
26. GLOPA	-.48**	-.38	.21	.19	-.34
27. PROCOMP	-.63+	-.51**	.13	.15	-.51**
28. PERFRAT	-.46*	-.34	.17	.21	-.34
29. OFFPOT	-.32	-.22	.20	.25	-.21
30. POTEVAL	-.42*	-.36	.23	.10	-.28
31. GLPAPI	-.05	-.06	-.11	-.23	-.09
32. SIMALL	-.07	-.12	.04	-.15	.00
33. SIMUH1	.07	.01	.18	-.01	.16
34. TIMEAD	.09	.10	.17	.01	.15
35. MARITAL	.17	.22	-.29	-.17	.02
36. EDUC	-.28	-.21	.13	.18	-.19
37. TOBAC	-.07	.01	-.14	.17	-.12

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G1 (cont.)

Variables	23	24	25	26	27
1. CH1					
2. PSS1					
3. LESNGIN					
4. LESNG					
5. LESPSIN					
6. LESPS					
7. BLHR					
8. BLSBP					
9. BLDBP					
10. BLCOCR					
11. BLCORT					
12. ILPDDYS					
13. ILPINDY					
14. ILSDDYS					
15. ILSINDY					
16. TOILDYS					
17. TILINDY					
18. INPDDYS					
19. INPINDY					
20. INSDDYS					
21. INSINDY					
22. TOINDYS					
23. TININDY					
24. TODYS	.81++				
25. TINDY	.79++	.73++			
26. GLOPA	-.08	-.37	-.13		
27. PROCOMP	-.18	-.42*	-.10	.78++	
28. PERFRAT	-.05	-.36	-.07	.94++	.73++
29. OFFPOT	.04	-.23	.00	.91++	.55**
30. POTEVAL	-.13	-.33	-.24	.92++	.67+
31. GLPAPI	-.19	-.14	-.25	.50**	.32
32. SIMALL	-.16	-.08	-.27	.31	.07
33. SIMUH1	.00	.08	-.16	.43*	.01
34. TIMEAD	.07	.00	-.17	.23	.14
35. MARITAL	.03	-.12	.00	-.03	-.15
36. EDUC	.04	-.14	.08	-.05	.07
37. TOBAC	.08	-.04	.30	.23	.18

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G1 (cont.)

Variables	28	29	30	31	32
1. CH1					
2. PSS1					
3. LESNGIN					
4. LESNG					
5. LESPSIN					
6. LESPS					
7. BLHR					
8. BLSBP					
9. BLDBP					
10. BLCOCR					
11. BLCORT					
12. ILPDDYS					
13. ILPINDY					
14. ILSDDYS					
15. ILSINDY					
16. TOILDYS					
17. TILINDY					
18. INPDDYS					
19. INPINDY					
20. INSDDYS					
21. INSINDY					
22. TOINDYS					
23. TININDY					
24. TODYS					
25. TINDY					
26. GLOPA					
27. PROCOMP					
28. PERFRAT					
29. OFFPOT	.83++				
30. POTEVAL	.76++	.80++			
31. GLPAPI	.31	.33	.71++		
32. SIMALL	.32	.39	.22	-.09	
33. SIMUH1	.29	.39*	.58***	.45*	.57***
34. TIMEAD	.09	.35	.30	.21	.10
35. MARITAL	-.10	-.05	.10	.38*	-.49**
36. EDUC	.16	-.02	-.31	-.72++	.29
37. TOBAC	.27	.33	.07	-.17	-.31

Note: *p<.1, **p<.05. ***p<.01, +p<.005, ++p<.001

Table G1 (cont.)

Variables	33	34	35	36	37
1. CH1					
2. PSS1					
3. LESNGIN					
4. LESNG					
5. LESPSIN					
6. LESPS					
7. BLHR					
8. BLSBP					
9. BLDBP					
10. BLCOCR					
11. BLCORT					
12. ILPDDYS					
13. ILPINDY					
14. ILSDDYS					
15. ILSINDY					
16. TOILDYS					
17. TILINDY					
18. INPDDYS					
19. INPINDY					
20. INSDDYS					
21. INSINDY					
22. TOINDYS					
23. TININDY					
24. TODYS					
25. TINDY					
26. GLOPA					
27. PROCOMP					
28. PERFRAT					
29. OFFPOT					
30. POTEVAL					
31. GLPAPI					
32. SIMALL					
33. SIMUH1					
34. TIMEAD	-.02				
35. MARITAL	.03	.12			
36. EDUC	-.28	-.28	-.56		
37. TOBAC	-.25	-.06	.21	-.10	

Note: *p<.1. **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2

Means, Standard Deviations, and Intercorrelations for Acute and Illness/Injury Variables

Variables	N	Mean	Std Dev	1	2
1. CH1	10	117.80	12.51		
2. PRECHL	9	3.86	.38	.86+	
3. PRETHR	10	2.60	.57	-.02	.05
4. INPRETHR	10	-.40	.0845	.09	.12
5. PRECEN	10	2.55	.84	.13	.38
6. PRECON	10	3.10	.99	.27	.08
7. PRUNCN	10	3.70	.95	.03	.28
8. PSTCHL	10	10.00	2.62	.57*	.34
9. PSTTHR	10	11.10	4.07	.20	.00
10. INPSTTHR	10	-.10	.0389	.31	.17
11. PSTSTR	10	3.63	.73	.36	.32
12. PSTSTR2	10	13.62	4.69	.35	.29
13. PSTSTR3	10	52.43	23.80	.33	.27
14. ILPDDYS	9	.0156	.0309	-.20	.00
15. ILPINDY	9	.0022	.0044	-.20	.00
16. ILSDDYS	9	.0111	.0267	-.55	-.22
17. ILSINDY	9	.0100	.0235	-.56	-.23
18. TOILDYS	9	.0267	.0517	-.40	-.11
19. TILINDY	9	.0122	.0264	-.52	-.21
20. INPDDYS	9	.0678	.1385	-.28	-.31
21. INPINDY	9	.0044	.0073	-.27	-.24
22. INSDDYS	9	.0322	.0648	-.12	.02
23. INSINDY	9	.0056	.0088	-.38	-.05
24. TOINDYS	9	.1000	.1452	-.32	-.29
25. TININDY	9	.0100	.0122	-.43	-.18
26. TODYS	9	.1256	.1511	-.43	-.32
27. TINDY	9	.0222	.0356	-.54	-.22
28. SHRAV	10	74.95	16.92	-.03	.27
29. HRCHRW	10	8.69	14.51	.00	.30
30. HRCHPC	10	13.09	22.14	.00	.28
31. SSBPAV	10	130.12	7.39	.07	-.35
32. SBPCRW	10	5.09	7.61	-.34	-.22
33. SBPCRW2	10	78.03	63.29	.09	.17
34. SBPCRW3	10	606.77	1126.00	-.21	.04
35. SBPCPC	10	4.49	6.05	-.32	-.18
36. SBPCPC2	10	53.07	50.29	-.01	.14
37. SBPCPC3	10	423.40	726.31	-.20	.04

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	N	Mean	Std Dev	1	2
38. SDBPAV	10	83.87	7.47	.48	.59*
39. DBPCRW	10	5.84	7.52	.29	.65*
40. DBPCPC	10	8.19	9.90	.30	.64*
41. SHRMX	10	84.60	18.93	.04	.38
42. HRCRX	10	18.34	17.02	.07	.42
43. HRCPX	10	27.94	25.87	.07	.40
44. SSBPMX	10	140.00	5.33	-.38	-.79**
45. SBPCRX	10	14.97	10.92	-.47	-.42
46. SBPCRX2	10	331.41	226.55	-.40	-.29
47. SBPCRX3	10	6970.00	6106.00	-.38	-.25
48. SBPCPX	10	12.68	9.11	-.45	-.36
49. SBPCPX2	10	235.50	175.84	-.39	-.24
50. SBPCPX3	10	4311.00	4026.00	-.38	-.21
51. SDBPMX	10	93.20	11.91	.57*	.59*
52. DBPCRX	10	15.17	12.33	.40	.64*
53. DBPCPX	10	20.41	16.80	.39	.62*
54. SCOCR	10	3.75	2.98	-.18	-.41
55. SCOCRR	10	-1.64	4.86	.07	-.19
56. SCOCRR2	10	23.95	44.16	-.49	-.28
57. COCRRM	10	3.72	3.36	-.57*	-.43
58. SCOCRP	10	-12.89	76.62	-.14	-.37
59. COCRPM	10	64.87	37.14	-.69**	-.77**
60. SCORT	10	4.41	3.61	-.21	-.41
61. SCORTR	10	-4.93	8.55	.27	.04
62. CORTRM	10	5.87	7.87	-.41	-.22
63. SCORTP	10	-39.25	43.90	-.03	-.20
64. INSCORTP	10	.0186	.0456	.37	.43
65. SCRTPDV	10	9.55	43.90	-.03	-.20
66. CRTPDVM	10	33.85	27.48	-.70**	-.71**
67. CORTPM	10	51.47	26.37	-.38	-.27
68. CORTPM2	10	3275.00	2688.00	-.48	-.29
69. CORTPM3	10	230662.00	242807.00	-.54	-.30
70. SPAPRO	10	7.00	.96	.27	.44
71. EP	10	6.70	2.41	.76**	.72**
72. SPAACQ	10	6.90	.97	.68**	.65*
73. DECTEC	9	6.11	1.90	.42	.25
74. WRKLD	10	5.90	2.64	.67**	.50

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	N	Mean	Std Dev	1	2
75. UNEXEV	10	7.20	2.97	.58*	.43
76. INFOX	8	7.00	1.77	.49	.82**
77. SITAWR	9	7.56	1.33	.76**	.57
78. INFOSOT	10	6.30	1.83	-.39	-.23
79. XMONITR	8	6.75	1.67	-.08	-.49
80. INFOOFR	9	6.56	1.42	.09	.03
81. ADVASS	9	7.89	1.27	-.23	-.37
82. FLYALL	10	406.31	204.09	.10	-.21
83. FLYUH1	10	281.31	159.40	-.35	-.47
84. SIMALL	9	122.57	40.69	.49	.05
85. SIMUH1	9	101.68	31.44	.12	-.01
86. TOBAC	10	.30	.48	.10	.44

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	3	4	5	6	7
1. CH1					
2. PRECHL					
3. PRETHR					
4. INPRETHR	.97++				
5. PRECEN	.64**	.57*			
6. PRECON	-.02	-.04	.16		
7. PRUNCN	-.20	-.24	-.05	-.32	
8. PSTCHL	-.02	.06	.34	.47	-.62*
9. PSTTHR	.52	.45	.44	.22	-.05
10. INPSTTHR	.37	.30	.28	.14	.00
11. PSTSTR	.03	-.02	.00	-.25	.38
12. PSTSTR2	.00	-.05	-.03	-.28	.35
13. PSTSTR3	-.04	-.08	-.05	-.31	.33
14. ILPDDYS	.20	.26	-.31	-.06	.42
15. ILPINDY	.20	.26	-.31	-.06	.42
16. ILSDDYS	.20	.23	.14	-.32	.30
17. ILSINDY	.19	.22	.14	-.30	.27
18. TOILDYS	.22	.27	-.11	-.20	.41
19. TILINDY	.20	.24	.07	-.28	.31
20. INPDDYS	-.09	-.03	-.01	.23	-.68**
21. INPINDY	.05	.11	.07	.09	-.55
22. INSDDYS	-.50	-.63*	-.11	-.10	.39
23. INSINDY	-.20	-.30	.03	-.34	.60*
24. TOINDYS	-.31	-.31	-.06	.18	-.48
25. TININDY	-.12	-.15	.06	-.19	.11
26. TODYS	-.21	-.20	-.09	.10	-.31
27. TINDY	.11	.12	.08	-.27	.27
28. SHRAV	.00	-.05	.13	-.31	-.09
29. HRCHRW	-.18	-.22	.06	-.60*	.13
30. HRCHPC	-.24	-.28	-.01	-.63*	.16
31. SSBPAV	-.19	-.23	-.23	.19	.31
32. SBPCRW	-.17	-.14	-.57*	-.52	.18
33. SBPCRW2	.14	.22	-.13	-.23	.62*
34. SBPCRW3	-.10	-.02	-.35	-.40	.42
35. SBPCPC	-.12	-.08	-.53	-.53	.21
36. SBPCPC2	.10	.19	-.16	-.32	.57*
37. SBPCPC3	.00	.09	-.24	-.39	.46

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	3	4	5	6	7
38. SDBPAV	.13	.04	.25	.21	.63*
39. DBPCRW	-.30	-.26	-.34	-.34	.52
40. DBPCPC	-.26	-.20	-.33	-.37	.53
41. SHRMX	-.02	-.08	.17	-.33	.00
42. HRCRX	-.17	-.23	.12	-.56*	.20
43. HRCPX	-.25	-.30	.03	-.62*	.26
44. SSBPMX	-.14	-.22	-.31	.29	-.31
45. SBPCRX	-.06	-.05	-.39	-.35	-.24
46. SBPCRX2	.15	.17	-.15	-.17	-.30
47. SBPCRX3	.13	.16	-.08	-.11	-.33
48. SBPCPX	.00	.01	-.34	-.36	-.22
49. SBPCPX2	.18	.21	-.10	-.21	-.25
50. SBPCPX3	.17	.22	-.02	-.16	-.25
51. SDBPMX	-.15	-.18	-.12	.18	.58*
52. DBPCRX	-.40	-.35	-.47	-.17	.46
53. DBPCPX	-.37	-.30	-.48	-.21	.45
54. SCOCR	-.68**	-.72**	-.51	.11	-.05
55. SCOCRR	-.75**	-.70**	-.61*	-.18	.27
56. SCOCRR2	.62	.46	.43	.03	-.33
57. COCRRM	.48	.30	.35	.19	-.36
58. SCOCRP	-.64**	-.68**	-.53	-.12	.06
59. COCRPM	-.06	-.19	-.07	.10	-.02
60. SCORT	-.37	-.40	-.30	.15	-.13
61. SCORTR	-.73**	-.62*	-.51	-.20	.36
62. CORTRM	.69**	.56*	.44	.20	-.42
63. SCORTP	-.66**	-.70**	-.47	-.24	.21
64. INSCORTP	.18	.31	.04	-.53	-.01
65. SCRTPDV	-.66**	-.70**	-.47	-.24	.21
66. CRTPDVM	-.40	-.51	-.35	-.33	.11
67. CORTPM	.61*	.58*	.36	.35	-.30
68. CORTPM2	.65**	.61*	.38	.18	-.28
69. CORTPM3	.65**	.60*	.40	.08	-.26
70. SPAPRO	.27	.15	.67**	.67**	.18
71. EP	-.40	-.38	.06	.01	.44
72. SPAACQ	-.02	-.01	.41	.57*	.20
73. DECTEC	-.40	-.38	-.24	.06	.68**
74. WRKLD	-.36	-.34	-.06	.51	.34

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	3	4	5	6	7
75. UNEXEV	-.38	-.32	-.08	-.01	.50
76. INFOX	.26	.17	.26	.08	.23
77. SITAWR	-.32	-.28	-.01	-.14	.13
78. INFOSOT	.32	.27	.33	.29	-.01
79. XMONITR	.03	.18	.03	-.08	.12
80. INFOOFR	-.07	.06	-.24	-.61*	-.20
81. ADVASS	.06	-.11	.32	.19	-.23
82. FLYALL	-.25	-.34	-.25	.52	.30
83. FLYUH1	-.55*	-.68**	-.18	.33	.22
84. SIMALL	.07	.07	.10	.39	-.17
85. SIMUH1	-.44	-.45	-.14	-.24	-.32
86. TOBAC	-.12	-.02	.16	-.53	.46

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	8	9	10	11	12
1. CH1					
2. PRECHL					
3. PRETHR					
4. INPRETHR					
5. PRECEN					
6. PRECON					
7. PRUNCN					
8. PSTCHL					
9. PSTTHR	.24				
10. INPSTTHR	.25	.93++			
11. PSTSTR	-.01	.63**	.83+		
12. PSTSTR2	.00	.62*	.81+	.99++	
13. PSTSTR3	.01	.59*	.78***	.98++	.99++
14. ILPDDYS	-.64*	-.37	-.40	-.27	-.31
15. ILPINDY	-.64*	-.37	-.40	-.27	-.31
16. ILSDDYS	-.49	-.54	-.73**	-.61*	-.63*
17. ILSINDY	-.48	-.55	-.74**	-.64*	-.66*
18. TOILDYS	-.64*	-.50	-.61*	-.48	-.51
19. TILINDY	-.54	-.55	-.73**	-.61*	-.64*
20. INPDDYS	.26	-.56	-.71**	-.89+	-.84+
21. INPINDY	.16	-.66*	-.82***	-.94++	-.92++
22. INSDDYS	-.31	-.26	-.13	.12	.09
23. INSINDY	-.59*	-.50	-.50	-.19	-.24
24. TOINDYS	.11	-.65*	-.74**	-.79**	-.76**
25. TININDY	-.33	-.75**	-.85+	-.69**	-.72**
26. TODYS	-.12	-.78**	-.91++	-.91++	-.89+
27. TINDY	-.51	-.67**	-.83***	-.69**	-.72**
28. SHRAV	.13	.16	.35	.47	.51
29. HRCHRW	.03	.00	.21	.51	.56*
30. HRCHPC	-.01	-.04	.18	.52	.57*
31. SSBPAV	-.09	.53	.51	.48	.46
32. SBPCRW	-.61*	-.69**	-.54	-.21	-.24
33. SBPCRW2	-.57*	-.28	-.34	-.13	-.19
34. SBPCRW3	-.61*	-.68**	-.63*	-.32	-.36
35. SBPCPC	-.63*	-.68**	-.55*	-.23	-.26
36. SBPCPC2	-.59*	-.42	-.46	-.22	-.27
37. SBPCPC3	-.60*	-.61*	-.62*	-.35	-.39

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	8	9	10	11	12
38. SDBPAV	-.16	.46	.47	.55	.51
39. DBPCRW	-.38	-.62*	-.40	.06	.04
40. DBPCPC	-.39	-.60*	-.40	.06	.04
41. SHRMX	.14	.14	.37	.53	.55*
42. HRCRX	.04	.01	.24	.56*	.59*
43. HRCPX	-.01	-.06	.18	.55*	.59*
44. SSBPMX	-.05	.17	.07	-.16	-.16
45. SBPCRX	-.39	-.75**	-.69**	-.55	-.55*
46. SBPCRX2	-.30	-.70**	-.77***	-.78***	-.79***
47. SBPCRX3	-.21	-.72**	-.82+	-.85+	-.85+
48. SBPCPX	-.40	-.75**	-.71**	-.58*	-.58*
49. SBPCPX2	-.31	-.70**	-.79***	-.78***	-.79***
50. SBPCPX3	-.25	-.71**	-.82+	-.85+	-.85+
51. SDBPMX	-.13	.31	.47	.67**	.66**
52. DBPCRX	-.25	-.37	-.10	.32	.32
53. DBPCPX	-.27	-.38	-.12	.30	.30
54. SCOCR	.10	-.01	.15	.24	.24
55. SCOCRR	-.02	-.20	-.08	.22	.23
56. SCOCRR2	-.10	.38	.35	.05	.03
57. COCRRM	-.07	.41	.36	.01	-.01
58. SCOCRP	.00	-.05	.12	.31	.30
59. COCRPM	-.23	.33	.25	.08	.07
60. SCORT	.19	.37	.47	.40	.41
61. SCORTR	.06	-.23	-.14	.20	.21
62. CORTRM	-.42	.31	.23	-.15	-.17
63. SCORTP	.21	.11	.30	.57*	.59*
64. INSCORTP	-.01	.00	.06	.26	.32
65. SCRTPDV	.21	.11	.30	.57*	.59*
66. CRTPDVM	.11	.03	.09	.22	.24
67. CORTPM	-.30	-.07	-.30	-.68**	-.73**
68. CORTPM2	-.28	-.09	-.32	-.66**	-.69**
69. CORTPM3	-.26	-.10	-.31	-.61*	-.63**
70. SPAPRO	.22	.39	.31	.06	.00
71. EP	.32	.08	.24	.51	.51
72. SPAACQ	.51	.32	.33	.23	.17
73. DECTEC	-.10	.27	.35	.65*	.65*
74. WRKLD	.30	.19	.29	.35	.31

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	8	9	10	11	12
75. UNEXEV	.20	.18	.30	.54	.50
76. INFOX	-.23	.04	.06	.12	.14
77. SITAWR	.56	.29	.48	.69**	.70**
78. INFOSOT	-.14	-.09	-.23	-.47	-.55*
79. XMONITR	.09	.12	-.04	-.11	-.14
80. INFOOFR	.16	.00	.06	.24	.33
81. ADVASS	.25	.62*	.57	.39	.44
82. FLYALL	-.18	.31	.27	.22	.19
83. FLYUH1	-.11	.06	.01	-.01	.00
84. SIMALL	.41	.58*	.50	.33	.30
85. SIMUH1	.41	-.20	-.01	.23	.22
86. TOBAC	-.09	-.24	-.21	.12	.14

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	13	14	15	16	17
1. CH1					
2. PRECHL					
3. PRETHR					
4. INPRETHR					
5. PRECEN					
6. PRECON					
7. PRUNCN					
8. PSTCHL					
9. PSTTHR					
10. INPSTTHR					
11. PSTSTR					
12. PSTSTR2					
13. PSTSTR3					
14. ILPDDYS	-.35				
15. ILPINDY	-.35	1.00++			
16. ILSDDYS	-.63*	.61*	.61*		
17. ILSINDY	-.65*	.60*	.60*	1.00++	
18. TOILDYS	-.54	.91++	.91++	.88+	.88+
19. TILINDY	-.64*	.71**	.71**	.99++	.99++
20. INPDDYS	-.79**	-.11	-.11	.27	.30
21. INPINDY	-.89+	.04	.04	.49	.51
22. INSDDYS	.06	-.19	-.19	-.12	-.12
23. INSINDY	-.28	.29	.29	.56	.54
24. TOINDYS	-.73**	-.20	-.20	.21	.23
25. TININDY	-.73**	.23	.23	.69**	.70**
26. TODYS	-.87+	.13	.13	.50	.52
27. TINDY	-.72**	.60	.60*	.97++	.97++
28. SHRAV	.55	-.36	-.36	-.39	-.39
29. HRCHRW	.61*	-.41	-.41	-.25	-.25
30. HRCHPC	.62*	-.40	-.40	-.25	-.26
31. SSBPAV	.42	-.16	-.16	-.37	-.39
32. SBPCRW	-.25	.57	.57	.41	.41
33. SBPCRW2	-.23	.82***	.82***	.63*	.61*
34. SBPCRW3	-.39	.83***	.83***	.71**	.70**
35. SBPCPC	-.27	.62*	.62*	.47	.46
36. SBPCPC2	-.31	.85+	.85+	.72**	.70**
37. SBPCPC3	-.42	.85+	.85+	.80***	.79**

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	13	14	15	16	17
38. SDBPAV	.46	.01	.01	-.30	-.32
39. DBPCRW	.03	.50	.50	.19	.17
40. DBPCPC	.03	.53	.54	.22	.21
41. SHRMX	.58*	-.36	-.36	-.39	-.39
42. HRCRX	.62*	-.39	-.39	-.26	-.27
43. HRCPX	.62*	-.38	-.38	-.24	-.25
44. SSBPMX	-.17	-.26	-.26	-.21	-.20
45. SBPCRX	-.54	.38	.38	.43	.45
46. SBPCRX2	-.78***	.41	.41	.59*	.61*
47. SBPCRX3	-.84+	.38	.38	.63*	.64*
48. SBPCPX	-.57*	.42	.42	.49	.50
49. SBPCPX2	-.78***	.46	.46	.66*	.67**
50. SBPCPX3	-.84+	.44	.44	.72**	.73**
51. SDBPMX	.64**	.13	.12	-.49	-.51
52. DBPCRX	.32	.42	.42	-.15	-.16
53. DBPCPX	.31	.45	.45	-.12	-.13
54. SCOCR	.23	-.36	-.36	-.42	-.42
55. SCOCRR	.23	-.25	-.25	-.22	-.23
56. SCOCRR2	.00	-.33	-.33	-.22	-.21
57. COCRRM	-.04	-.27	-.27	-.20	-.18
58. SCOCRP	.29	-.36	-.36	-.36	-.37
59. COCRPM	.04	-.14	-.14	.00	.00
60. SCORT	.40	-.31	-.31	-.43	-.43
61. SCORTR	.23	-.28	-.28	-.15	-.17
62. CORTRM	-.19	.17	.17	.04	.06
63. SCORTP	.61*	-.48	-.48	-.50	-.51
64. INSCORTP	.38	-.06	-.06	-.07	-.07
65. SCRTPDV	.61*	-.48	-.48	-.50	-.51
66. CRTPDVM	.26	-.23	-.23	-.04	-.04
67. CORTPM	-.76**	.53	.53	.62*	.63*
68. CORTPM2	-.71**	.56	.56	.73**	.74**
69. CORTPM3	-.65**	.56	.56	.80**	.81***
70. SPAPRO	-.06	-.08	-.08	-.14	-.14
71. EP	.51	-.43	-.43	-.50	-.52
72. SPAACQ	.10	-.16	-.16	-.28	-.30
73. DECTEC	.66*	.00	.00	-.35	-.38
74. WRKLD	.25	-.08	-.08	-.50	-.51

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	13	14	15	16	17
75. UNEXEV	.46	-.18	-.18	-.33	-.36
76. INFOX	.15	.05	.05	-.20	-.19
77. SITAWR	.70**	-.60	-.60	-.69*	-.71**
78. INFOSOT	-.63*	.43	.43	.59*	.58
79. XMONITR	-.15	.21	.21	.48	.47
80. INFOOFR	.41	-.26	-.26	-.14	-.14
81. ADVASS	.49	-.84***	-.84***	-.53	-.52
82. FLYALL	.16	-.02	-.02	-.38	-.39
83. FLYUH1	-.01	-.28	-.28	-.11	-.11
84. SIMALL	.27	-.53	-.53	-.53	-.53
85. SIMUH1	.21	-.69*	-.69*	-.37	-.37
86. TOBAC	.18	.19	.19	.44	.43

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	18	19	20	21	22
1. CH1					
2. PRECHL					
3. PRETHR					
4. INPRETHR					
5. PRECEN					
6. PRECON					
7. PRUNCN					
8. PSTCHL					
9. PSTTHR					
10. INPSTTHR					
11. PSTSTR					
12. PSTSTR2					
13. PSTSTR3					
14. ILPDDYS					
15. ILPINDY					
16. ILSDDYS					
17. ILSINDY					
18. TOILDYS					
19. TILINDY	.93++				
20. INPDDYS	.07	.25			
21. INPINDY	.28	.46	.94++		
22. INSDDYS	-.18	-.14	-.13	-.08	
23. INSINDY	.46	.53	-.07	.15	.74**
24. TOINDYS	-.01	.18	.90+	.87+	.32
25. TININDY	.49	.66*	.51	.70**	.49
26. TODYS	.33	.49	.87+	.91++	.25
27. TINDY	.86+	.97++	.36	.58*	.06
28. SHRAV	-.41	-.41	-.24	-.38	-.13
29. HRCHRW	-.37	-.29	-.28	-.34	.12
30. HRCHPC	-.37	-.30	-.28	-.34	.16
31. SSBPAV	-.29	-.37	-.59*	-.64*	.23
32. SBPCRW	.56	.46	.05	.22	.29
33. SBPCRW2	.81***	.68**	-.26	.00	.05
34. SBPCRW3	.86+	.76**	-.02	.23	.11
35. SBPCPC	.61*	.51	.04	.24	.26
36. SBPCPC2	.88+	.77**	-.17	.11	.03
37. SBPCPC3	.92++	.84+	-.03	.24	.04

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	18	19	20	21	22
38. SDBPAV	-.15	-.28	-.63*	-.61*	.38
39. DBPCRW	.40	.24	-.19	-.05	.34
40. DBPCPC	.43	.27	-.20	-.05	.29
41. SHRMX	-.42	-.41	-.33	-.43	.00
42. HRCRX	-.37	-.30	-.37	-.39	.22
43. HRCPX	-.35	-.29	-.36	-.37	.30
44. SSBPMX	-.26	-.22	.21	.11	.29
45. SBPCRX	.45	.46	.52	.63*	.20
46. SBPCRX2	.55	.61*	.72**	.85+	.08
47. SBPCRX3	.55	.64*	.78**	.90++	.02
48. SBPCPX	.50	.52	.54	.66*	.17
49. SBPCPX2	.61*	.68**	.69**	.84+	.03
50. SBPCPX3	.63*	.73**	.73**	.88+	-.03
51. SDBPMX	-.18	-.43	-.70**	-.78**	.17
52. DBPCRX	.17	-.08	-.37	-.36	.13
53. DBPCPX	.21	-.04	-.36	-.34	.08
54. SCOCR	-.43	-.43	-.19	-.30	.39
55. SCOCRR	-.26	-.25	-.40	-.37	.51
56. SCOCRR2	-.31	-.24	.09	-.07	-.01
57. COCRRM	-.26	-.21	.12	-.07	.01
58. SCOCRP	-.40	-.39	-.28	-.32	.52
59. COCRPM	-.08	-.02	-.16	-.23	.19
60. SCORT	-.41	-.44	-.34	-.49	-.07
61. SCORTR	-.24	-.20	-.59*	-.51	.40
62. CORTRM	.12	.08	.61*	.44	-.47
63. SCORTP	-.55	-.54	-.49	-.59*	.40
64. INSCORTP	-.07	-.07	-.06	-.07	-.46
65. SCRTPDV	-.55	-.54	-.49	-.59*	.40
66. CRTPDVM	-.16	-.08	-.19	-.27	.33
67. CORTPM	.64*	.65*	.56	.66*	-.28
68. CORTPM2	.71**	.76**	.61*	.72**	-.31
69. CORTPM3	.74**	.81***	.64*	.74**	-.30
70. SPAPRO	-.12	-.14	-.17	-.18	.24
71. EP	-.51	-.54	-.53	-.52	.42
72. SPAACQ	-.24	-.29	-.35	-.29	.13
73. DECTEC	-.18	-.34	-.96++	-.94++	.31
74. WRKLD	-.30	-.47	-.53	-.53	.32

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	18	19	20	21	22
75. UNEXEV	-.28	-.35	-.81***	-.70**	.31
76. INFOX	-.07	-.17	.00	-.06	.25
77. SITAWR	-.72**	-.73**	-.56	-.59	.26
78. INFOSOT	.56	.59*	.14	.33	.11
79. XMONITR	.38	.45	-.17	.00	-.49
80. INFOOFR	-.23	-.17	.03	-.06	-.52
81. ADVASS	-.78**	-.61	-.09	-.29	.23
82. FLYALL	-.21	-.35	-.29	-.41	.42
83. FLYUH1	-.23	-.14	-.05	-.17	.51
84. SIMALL	-.58	-.56	-.11	-.21	.07
85. SIMUH1	-.59	-.44	.29	.18	.52
86. TOBAC	.34	.41	-.22	-.11	-.30

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	23	24	25	26	27
1. CH1					
2. PRECHL					
3. PRETHR					
4. INPRETHR					
5. PRECEN					
6. PRECON					
7. PRUNCN					
8. PSTCHL					
9. PSTTHR					
10. INPSTTHR					
11. PSTSTR					
12. PSTSTR2					
13. PSTSTR3					
14. ILPDDYS					
15. ILPINDY					
16. ILSDDYS					
17. ILSINDY					
18. TOILDYS					
19. TILINDY					
20. INPDDYS					
21. INPINDY					
22. INSDDYS					
23. INSINDY					
24. TOINDYS	.26				
25. TININDY	.81***	.70**			
26. TODYS	.41	.94++	.84+		
27. TINDY	.67**	.37	.83***	.65*	
28. SHRAV	-.36	-.28	-.48	-.41	-.46
29. HRCHRW	-.05	-.22	-.23	-.33	-.30
30. HRCHPC	-.01	-.20	-.21	-.31	-.29
31. SSBPAV	-.01	-.46	-.38	-.53	-.41
32. SBPCRW	.54	.18	.52	.37	.52
33. SBPCRW2	.54	-.23	.39	.07	.64*
34. SBPCRW3	.61*	.03	.57	.33	.76**
35. SBPCPC	.56	.16	.54	.37	.57
36. SBPCPC2	.58*	-.15	.48	.16	.73**
37. SBPCPC3	.61*	-.01	.59*	.31	.83***

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	23	24	25	26	27
38. SDBPAV	.19	-.43	-.22	-.45	-.29
39. DBPCRW	.46	-.03	.30	.11	.28
40. DBPCPC	.44	-.06	.29	.09	.30
41. SHRMX	-.23	-.32	-.42	-.44	-.45
42. HRCRX	.05	-.25	-.19	-.36	-.29
43. HRCPX	.13	-.21	-.12	-.32	-.26
44. SSBPMX	.04	.32	.09	.22	-.13
45. SBPCRX	.40	.59*	.66*	.71**	.57
46. SBPCRX2	.38	.72**	.78**	.87+	.72**
47. SBPCRX3	.35	.75**	.79**	.90+	.74**
48. SBPCPX	.41	.59*	.69**	.73**	.62*
49. SBPCPX2	.40	.67**	.79**	.84+	.77**
50. SBPCPX3	.39	.68**	.80***	.86+	.81***
51. SDBPMX	-.11	-.59*	-.54	-.62*	-.51
52. DBPCRX	.05	-.30	-.18	-.22	-.12
53. DBPCPX	.03	-.30	-.18	-.21	-.09
54. SCOCR	.03	-.01	-.16	-.15	-.37
55. SCOCRR	.32	-.15	.01	-.23	-.18
56. SCOCRR2	-.22	.08	-.20	-.03	-.25
57. COCRRM	-.20	.12	-.19	.02	-.22
58. SCOCRP	.21	-.04	-.04	-.17	-.30
59. COCRPM	.14	-.07	-.04	-.09	-.03
60. SCORT	-.34	-.36	-.54	-.48	-.51
61. SCORTR	.32	-.39	-.07	-.44	-.17
62. CORTRM	-.48	.37	-.08	.39	.03
63. SCORTP	.03	-.29	-.33	-.46	-.51
64. INSCORTP	-.39	-.26	-.32	-.27	-.16
65. SCRTPDV	.03	-.29	-.33	-.46	-.51
66. CRTPDVM	.21	-.03	-.01	-.08	-.06
67. CORTPM	.12	.41	.48	.60*	.65*
68. CORTPM2	.16	.45	.54	.66*	.75**
69. CORTPM3	.21	.47	.59*	.70**	.81***
70. SPAPRO	.12	-.06	-.02	-.09	-.11
71. EP	.10	-.32	-.24	-.47	-.48
72. SPAACQ	.00	-.28	-.18	-.34	-.27
73. DECTEC	.15	-.79**	-.46	-.82**	-.41
74. WRKLD	.02	-.36	-.30	-.44	-.45

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	23	24	25	26	27
75. UNEXEV	.18	-.64*	-.29	-.69**	-.36
76. INFOX	.05	.11	.00	.08	-.12
77. SITAWR	-.34	-.50	-.56	-.70*	-.72**
78. INFOSOT	.50	.18	.56	.37	.63*
79. XMONITR	.00	-.39	.00	-.24	.33
80. INFOOFR	-.53	-.21	-.43	-.29	-.27
81. ADVASS	-.21	.02	-.34	-.26	-.57
82. FLYALL	.08	-.09	-.18	-.16	-.32
83. FLYUH1	.28	.18	.10	.09	-.07
84. SIMALL	-.30	-.01	-.30	-.31	-.51
85. SIMUH1	.19	.55	.21	.15	-.26
86. TOBAC	.09	-.34	.00	-.21	.30

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	28	29	30	31	32
1. CH1					
2. PRECHL					
3. PRETHR					
4. INPRETHR					
5. PRECEN					
6. PRECON					
7. PRUNCN					
8. PSTCHL					
9. PSTTHR					
10. INPSTTHR					
11. PSTSTR					
12. PSTSTR2					
13. PSTSTR3					
14. ILPDDYS					
15. ILPINDY					
16. ILSDDYS					
17. ILSINDY					
18. TOILDYS					
19. TILINDY					
20. INPDDYS					
21. INPINDY					
22. INSDDYS					
23. INSINDY					
24. TOINDYS					
25. TININDY					
26. TODYS					
27. TINDY					
28. SHRAV					
29. HRCHRW	.89++				
30. HRCHPC	.86+	.99++			
31. SSBPAV	-.40	-.32	-.28		
32. SBPCRW	-.10	.03	.08	-.28	
33. SBPCRW2	-.60*	-.44	-.40	.08	.48
34. SBPCRW3	-.29	-.16	-.13	-.28	.86+
35. SBPCPC	-.15	-.01	.03	-.28	1.00++
36. SBPCPC2	-.51	-.35	-.32	-.07	.61*
37. SBPCPC3	-.38	-.24	-.22	-.24	.76**

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	28	29	30	31	32
38. SDBPAV	-.18	-.11	-.10	.50	-.33
39. DBPCRW	.08	.24	.28	-.36	.65**
40. DBPCPC	.05	.21	.26	-.35	.65**
41. SHRMX	.98++	.92++	.88++	-.37	-.06
42. HRCRX	.86+	.98++	.97++	-.28	.05
43. HRCPX	.79***	.97++	.98++	-.24	.12
44. SSBPMX	-.50	-.50	-.47	.62*	-.06
45. SBPCRX	-.04	-.01	.01	-.57*	.86+
46. SBPCRX2	-.29	-.28	-.27	-.62*	.66**
47. SBPCRX3	-.29	-.29	-.28	-.67**	.58*
48. SBPCPX	-.07	-.04	-.02	-.60*	.85+
49. SBPCPX2	-.29	-.26	-.25	-.65**	.66**
50. SBPCPX3	-.31	-.29	-.29	-.67**	.58*
51. SDBPMX	.16	.14	.16	.36	-.19
52. DBPCRX	.30	.34	.37	-.19	.42
53. DBPCPX	.29	.32	.36	-.21	.45
54. SCOCR	.03	.09	.12	.56*	.06
55. SCOCRR	-.27	.01	.08	.61*	.19
56. SCOCRR2	.41	.15	.08	-.22	-.04
57. COCRRM	.34	.05	-.01	-.07	-.15
58. SCOCRP	-.05	.13	.18	.59*	.20
59. COCRPM	-.06	-.13	-.14	.55*	-.09
60. SCORT	.25	.15	.14	.57*	-.22
61. SCORTR	-.28	.03	.10	.49	.09
62. CORTRM	.29	-.03	-.10	-.36	-.08
63. SCORTP	.23	.42	.47	.59*	.00
64. INSCORTP	.48	.54	.55	-.45	-.01
65. SCRTPDV	.23	.42	.47	.59*	.00
66. CRTPDVM	.25	.34	.36	.38	.17
67. CORTPM	-.41	-.63**	-.68**	-.33	.08
68. CORTPM2	-.25	-.47	-.51	-.45	.13
69. CORTPM3	-.13	-.33	-.38	-.50	.16
70. SPAPRO	-.14	-.28	-.33	.15	-.56*
71. EP	.05	.28	.31	.28	-.28
72. SPAACQ	-.28	-.27	-.30	.36	-.50
73. DECTEC	-.31	-.10	-.04	.80***	-.18
74. WRKLD	-.32	-.27	-.25	.57*	-.29

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	28	29	30	31	32
75. UNEXEV	-.21	.03	.05	.60*	-.15
76. INFOX	.18	.13	.14	-.31	-.12
77. SITAWR	.26	.44	.46	.32	-.30
78. INFOSOT	-.47	-.56*	-.60*	-.01	.18
79. XMONITR	-.56	-.41	-.41	.40	-.11
80. INFOOFR	.60*	.65*	.65*	-.28	.00
81. ADVASS	.39	.40	.39	.39	-.61*
82. FLYALL	-.49	-.49	-.45	.80***	-.25
83. FLYUH1	-.25	-.17	-.15	.62*	-.25
84. SIMALL	-.48	-.42	-.39	.71**	-.58
85. SIMUH1	.12	.37	.39	.10	.13
86. TOBAC	.40	.55	.54	-.39	.05

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	33	34	35	36	37
1. CH1					
2. PRECHL					
3. PRETHR					
4. INPRETHR					
5. PRECEN					
6. PRECON					
7. PRUNCN					
8. PSTCHL					
9. PSTTHR					
10. INPSTTHR					
11. PSTSTR					
12. PSTSTR2					
13. PSTSTR3					
14. ILPDDYS					
15. ILPINDY					
16. ILSDDYS					
17. ILSINDY					
18. TOILDYS					
19. TILINDY					
20. INPDDYS					
21. INPINDY					
22. INSDDYS					
23. INSINDY					
24. TOINDYS					
25. TININDY					
26. TODYS					
27. TINDY					
28. SHRAV					
29. HRCHRW					
30. HRCHPC					
31. SSBPAV					
32. SBPCRW					
33. SBPCRW2					
34. SBPCRW3	.79***				
35. SBPCPC	.56*	.89++			
36. SBPCPC2	.98++	.89++	.68**		
37. SBPCPC3	.86+	.98++	.81+	.95++	

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	33	34	35	36	37
38. SDBPAV	.32	-.17	-.30	.15	-.11
39. DBPCRW	.54	.70**	.66**	.60*	.62*
40. DBPCPC	.59*	.72**	.67**	.65**	.65**
41. SHRMX	-.54	-.24	-.11	-.46	-.34
42. HRCRX	-.38	-.12	.01	-.30	-.20
43. HRCPX	-.31	-.06	.08	-.24	-.15
44. SSBPMX	-.21	-.29	-.09	-.29	-.30
45. SBPCRX	.18	.64**	.84+	.33	.55
46. SBPCRX2	.28	.58*	.68**	.41	.55*
47. SBPCRX3	.25	.55*	.60*	.38	.54
48. SBPCPX	.23	.67**	.84+	.38	.58*
49. SBPCPX2	.34	.63*	.68**	.47	.61*
50. SBPCPX3	.33	.61*	.61*	.47	.61*
51. SDBPMX	.18	-.10	-.19	.06	-.13
52. DBPCRX	.30	.44	.41	.32	.33
53. DBPCPX	.33	.47	.44	.36	.37
54. SCOCR	-.39	-.17	-.01	-.38	-.28
55. SCOCRR	.10	.12	.17	.08	.08
56. SCOCRR2	-.39	-.23	-.06	-.35	-.26
57. COCRRM	-.49	-.33	-.18	-.46	-.36
58. SCOCRP	-.22	-.05	.14	-.22	-.15
59. COCRPM	-.28	-.22	-.13	-.29	-.22
60. SCORT	-.50	-.38	-.29	-.51	-.44
61. SCORTR	.21	.15	.08	.18	.14
62. CORTRM	-.32	-.20	-.09	-.28	-.21
63. SCORTP	-.32	-.22	-.06	-.34	-.30
64. INSCORTP	.01	-.02	.01	.04	.00
65. SCRTPDV	-.32	-.22	-.06	-.34	-.30
66. CRTPDVM	-.35	-.09	.11	-.31	-.15
67. CORTPM	.24	.23	.12	.27	.30
68. CORTPM2	.20	.25	.17	.25	.31
69. CORTPM3	.15	.24	.19	.22	.30
70. SPAPRO	-.01	-.32	-.54	-.12	-.25
71. EP	.05	-.19	-.28	-.03	-.19
72. SPAACQ	.11	-.23	-.48	.00	-.17
73. DECTEC	.34	-.02	-.16	.20	.02
74. WRKLD	.17	-.12	-.29	.05	-.12

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	33	34	35	36	37
75. UNEXEV	.26	.00	-.14	.18	.03
76. INFOX	.11	-.11	-.10	.04	-.11
77. SITAWR	-.18	-.34	-.32	-.24	-.36
78. INFOSOT	.33	.37	.21	.37	.43
79. XMONITR	.41	.21	-.06	.39	.33
80. INFOOFR	-.28	-.15	-.02	-.23	-.17
81. ADVASS	-.76**	-.84+	-.66*	-.82***	-.84***
82. FLYALL	.10	-.25	-.26	-.06	-.24
83. FLYUH1	-.23	-.28	-.28	-.30	-.28
84. SIMALL	-.09	-.60*	-.57	-.28	-.52
85. SIMUH1	-.43	-.17	.08	-.37	-.26
86. TOBAC	.24	.32	.08	.32	.36

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	38	39	40	41	42
38. SDBPAV					
39. DBPCRW	.19				
40. DBPCPC	.20	1.00++			
41. SHRMX	-.09	.16	.13		
42. HRCRX	-.02	.31	.28	.92++	
43. HRCPX	.01	.37	.34	.85+	.99++
44. SSBPMX	-.07	-.58*	-.59*	-.54	-.53
45. SBPCRX	-.60*	.41	.40	-.06	-.03
46. SBPCRX2	-.52	.27	.27	-.32	-.31
47. SBPCRX3	-.57*	.23	.24	-.32	-.32
48. SBPCPX	-.58*	.42	.41	-.09	-.06
49. SBPCPX2	-.52	.30	.31	-.31	-.29
50. SBPCPX3	-.54	.25	.26	-.34	-.32
51. SDBPMX	.80***	.41	.41	.21	.19
52. DBPCRX	.24	.87+	.86+	.35	.37
53. DBPCPX	.21	.88++	.87+	.32	.35
54. SCOCR	-.15	-.19	-.24	.06	.12
55. SCOCRR	.01	.11	.10	-.22	.04
56. SCOCRR2	-.15	-.39	-.42	.38	.15
57. COCRRM	-.17	-.54	-.57*	.30	.04
58. SCOCRP	-.09	-.07	-.11	.01	.17
59. COCRPM	-.12	-.62*	-.64**	-.08	-.13
60. SCORT	-.18	-.48	-.50	.23	.14
61. SCORTR	.07	.21	.22	-.23	.05
62. CORTRM	-.16	-.36	-.38	.24	-.05
63. SCORTP	.05	-.05	-.07	.27	.43
64. INSCORTP	-.08	.30	.33	.43	.46
65. SCRTPDV	.05	-.05	-.07	.27	.43
66. CRTPDVM	-.27	-.28	-.31	.24	.30
67. CORTPM	-.21	-.25	-.23	-.45	-.64**
68. CORTPM2	-.27	-.21	-.20	-.31	-.49
69. CORTPM3	-.30	-.19	-.18	-.19	-.37
70. SPAPRO	.64**	-.16	-.18	-.07	-.17
71. EP	.61*	.37	.36	.17	.37
72. SPAACQ	.60*	-.02	-.02	-.16	-.14
73. DECTEC	.68**	.18	.19	-.20	.01
74. WRKLD	.64**	.17	.16	-.21	-.15

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	38	39	40	41	42
75. UNEXEV	.51	.20	.21	-.08	.14
76. INFOX	.57	.43	.42	.18	.14
77. SITAWR	.35	.14	.14	.33	.49
78. INFOSOT	-.10	-.17	-.17	-.41	-.47
79. XMONITR	-.16	-.34	-.28	-.56	-.42
80. INFOOFR	-.41	.02	.05	.51	.52
81. ADVASS	.11	-.74**	-.78**	.35	.32
82. FLYALL	.64**	-.16	-.17	-.47	-.46
83. FLYUH1	.17	-.35	-.39	-.23	-.16
84. SIMALL	.59*	-.46	-.44	-.50	-.43
85. SIMUH1	-.26	.01	-.03	.21	.44
86. TOBAC	-.08	.41	.43	.42	.54

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	43	44	45	46	47
38. SDBPAV					
39. DBPCRW					
40. DBPCPC					
41. SHRMX					
42. HRCRX					
43. HRCPX					
44. SSBPMX	-.48				
45. SBPCRX	.01	.03			
46. SBPCRX2	-.28	.05	.90++		
47. SBPCRX3	-.30	.00	.86+	.99++	
48. SBPCPX	-.02	-.01	1.00++	.93++	.89++
49. SBPCPX2	-.26	-.03	.88++	.99++	.99++
50. SBPCPX3	-.30	-.07	.83+	.97++	.99++
51. SDBPMX	.20	-.29	-.52	-.61*	-.66**
52. DBPCRX	.41	-.57*	.15	-.08	-.11
53. DBPCPX	.39	-.58*	.17	-.04	-.08
54. SCOCR	.15	.56*	-.06	-.34	-.36
55. SCOCRR	.14	.37	-.09	-.29	-.32
56. SCOCRR2	.05	.11	.18	.14	.11
57. COCRRM	-.06	.29	.08	.05	.03
58. SCOCRP	.25	.55	.01	-.29	-.33
59. COCRPM	-.15	.69**	-.10	-.21	-.23
60. SCORT	.11	.42	-.34	-.59*	-.59*
61. SCORTR	.16	.12	-.21	-.34	-.33
62. CORTRM	-.16	.05	.21	.27	.26
63. SCORTP	.49	.32	-.24	-.56*	-.60*
64. INSCORTP	.46	-.66**	-.03	-.04	-.04
65. SCRTPDV	.49	.32	-.24	-.56*	-.60*
66. CRTPDVM	.33	.48	.10	-.18	-.22
67. CORTPM	-.70**	.13	.34	.62*	.64**
68. CORTPM2	-.56*	.04	.42	.66**	.68**
69. CORTPM3	-.43	-.01	.45	.66**	.68**
70. SPAPRO	-.24	-.05	-.51	-.32	-.27
71. EP	.43	-.29	-.53	-.58*	-.57*
72. SPAACQ	-.16	-.11	-.64**	-.52	-.47
73. DECTEC	.09	.08	-.62*	-.71**	-.74**
74. WRKLD	-.12	.06	-.56*	-.57*	-.56*

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	43	44	45	46	47
75. UNEXEV	.19	-.06	-.54	-.63**	-.63**
76. INFOX	.14	-.45	-.06	.09	.04
77. SITAWR	.54	-.25	-.53	-.67**	-.67**
78. INFOSOT	-.50	.21	.23	.36	.40
79. XMONITR	-.40	.16	-.31	-.19	-.13
80. INFOOFR	.51	-.42	.02	-.11	-.11
81. ADVASS	.29	.51	-.45	-.49	-.50
82. FLYALL	-.41	.61*	-.42	-.39	-.43
83. FLYUH1	-.12	.64**	-.28	-.33	-.32
84. SIMALL	-.39	.53	-.63	-.47	-.51
85. SIMUH1	.50	.26	.16	-.05	-.06
86. TOBAC	.53	-.82+	-.10	-.14	-.08

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	48	49	50	51	52
38. SDBPAV					
39. DBPCRW					
40. DBPCPC					
41. SHRMX					
42. HRCRX					
43. HRCPX					
44. SSBPMX					
45. SBPCRX					
46. SBPCRX2					
47. SBPCRX3					
48. SBPCPX					
49. SBPCPX2	.92++				
50. SBPCPX3	.87+	.99++			
51. SDBPMX	-.53	-.61*	-.66**		
52. DBPCRX	.13	-.06	-.12	.68**	
53. DBPCPX	.16	-.03	-.08	.65**	1.00++
54. SCOCR	-.14	-.40	-.43	.00	-.03
55. SCOCRR	-.14	-.32	-.33	.08	.14
56. SCOCRR2	.17	.13	.09	-.27	-.39
57. COCRRM	.06	.02	.00	-.27	-.47
58. SCOCRP	-.07	-.34	-.38	-.02	-.01
59. COCRPM	-.15	-.24	-.25	-.26	-.54
60. SCORT	-.41	-.62*	-.63	.04	-.15
61. SCORTR	-.24	-.34	-.32	.17	.24
62. CORTRM	.22	.26	.24	-.25	-.35
63. SCORTP	-.32	-.60*	-.64**	.22	.14
64. INSCORTP	.01	.01	-.01	.15	.37
65. SCRTPDV	-.32	-.60	-.64**	.22	.14
66. CRTPDVM	.03	-.22	-.25	-.20	-.19
67. CORTPM	.39	.63*	.67**	-.47	-.45
68. CORTPM2	.47	.68**	.71**	-.51	-.42
69. CORTPM3	.49	.68**	.70**	-.52	-.41
70. SPAPRO	-.50	-.31	-.26	.36	-.16
71. EP	-.54	-.57*	-.56*	.62*	.42
72. SPAACQ	-.64**	-.51	-.44	.43	.02
73. DECTEC	-.63*	-.70**	-.70**	.69**	.33
74. WRKLD	-.58*	-.58*	-.56*	.64**	.30

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	48	49	50	51	52
75. UNEXEV	-.56*	-.61*	-.60*	.49	.25
76. INFOX	-.02	.07	.02	.57	.40
77. SITAWR	-.55	-.67**	-.68**	.46	.29
78. INFOSOT	.25	.39	.44	-.42	-.42
79. XMONITR	-.29	-.12	-.03	-.33	-.40
80. INFOOFR	.02	-.09	-.11	-.10	.18
81. ADVASS	-.49	-.54	-.55	-.06	-.60*
82. FLYALL	-.44	-.44	-.47	.52	-.02
83. FLYUH1	-.33	-.38	-.35	.05	-.27
84. SIMALL	-.62*	-.50	-.52	.24	-.40
85. SIMUH1	.10	-.10	-.13	-.31	-.11
86. TOBAC	-.07	-.05	.01	.09	.38

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	53	54	55	56	57
38. SDBPAV					
39. DBPCRW					
40. DBPCPC					
41. SHRMX					
42. HRCRX					
43. HRCPX					
44. SSBPMX					
45. SBPCRX					
46. SBPCRX2					
47. SBPCRX3					
48. SBPCPX					
49. SBPCPX2					
50. SBPCPX3					
51. SDBPMX					
52. DBPCRX					
53. DBPCPX					
54. SCOCR	-.07				
55. SCOCRR	.13	.76**			
56. SCOCRR2	-.40	-.11	-.65**		
57. COCRRM	-.50	.07	-.53	.96++	
58. SCOCRP	-.04	.95++	.86+	-.21	-.08
59. COCRPM	-.57*	.62*	.25	.44	.62*
60. SCORT	-.18	.84+	.50	.11	.30
61. SCORTR	.24	.53	.94++	-.85+	-.77***
62. CORTRM	-.36	-.32	-.82+	.94++	.90++
63. SCORTP	.11	.86+	.82+	-.26	-.15
64. INSCORTP	.42	-.54	-.24	-.19	-.37
65. SCRTPDV	.11	.86+	.82+	-.26	-.15
66. CRTPDVM	-.22	.73**	.47	.24	.38
67. CORTPM	-.43	-.49	-.65**	.47	.46
68. CORTPM2	-.40	-.57*	-.73**	.56*	.52
69. CORTPM3	-.39	-.57*	-.77***	.63*	.57*
70. SPAPRO	-.21	-.17	-.37	.23	.27
71. EP	.39	.15	.44	-.61*	-.63*
72. SPAACQ	-.03	.07	.14	-.29	-.25
73. DECTEC	.31	.39	.67**	-.60*	-.54
74. WRKLD	.25	.35	.47	-.51	-.42

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	53	54	55	56	57
75. UNEXEV	.23	.39	.70**	-.61*	-.59*
76. INFOX	.39	-.60	-.54	.06	-.06
77. SITAWR	.28	.30	.52	-.46	-.50
78. INFOSOT	-.44	-.03	-.18	.36	.39
79. XMONITR	-.36	.10	.40	-.36	-.31
80. INFOOFR	.23	-.12	.05	-.17	-.25
81. ADVASS	-.65*	.39	.00	.46	.60*
82. FLYALL	-.05	.38	.35	-.20	-.03
83. FLYUH1	-.33	.70**	.51	-.10	.14
84. SIMALL	-.40	.10	.26	-.30	-.23
85. SIMUH1	-.13	.60*	.58	-.14	-.14
86. TOBAC	.40	-.33	.00	-.32	-.42

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	58	59	60	61	62
38. SDBPAV					
39. DBPCRW					
40. DBPCPC					
41. SHRMX					
42. HRCRX					
43. HRCPX					
44. SSBPMX					
45. SBPCRX					
46. SBPCRX2					
47. SBPCRX3					
48. SBPCPX					
49. SBPCPX2					
50. SBPCPX3					
51. SDBPMX					
52. DBPCRX					
53. DBPCPX					
54. SCOCR					
55. SCOCRR					
56. SCOCRR2					
57. COCRRM					
58. SCOCRP					
59. COCRPM	.54				
60. SCORT	.71**	.71**			
61. SCORTR	.65**	-.02	.31		
62. CORTRM	-.45	.26	-.08	-.96++	
63. SCORTP	.90++	.43	.74**	.69**	-.53
64. INSCORTP	-.45	-.69**	-.37	-.01	-.14
65. SCRTPDV	.90++	.43	.74**	.69**	-.53
66. CRTPDVM	.71**	.81+	.70**	.22	-.01
67. CORTPM	-.58*	.10	-.40	-.69**	.66**
68. CORTPM2	-.64**	.08	-.45	-.77***	.73**
69. CORTPM3	-.64**	.10	-.44	-.81+	.77***
70. SPAPRO	-.28	.06	-.19	-.34	.28
71. EP	.24	-.41	-.03	.59*	-.69**
72. SPAACQ	.04	-.15	.05	.23	-.28
73. DECTEC	.46	.10	.32	.73**	-.72**
74. WRKLD	.34	-.09	.22	.51	-.53

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	58	59	60	61	62
75. UNEXEV	.51	-.06	.28	.77***	-.76**
76. INFOX	-.59	-.66*	-.72**	-.42	.21
77. SITAWR	.43	-.34	.25	.60*	-.61*
78. INFOSOT	-.06	.39	-.05	-.30	.36
79. XMONITR	.16	.29	.27	.48	-.41
80. INFOOFR	-.07	-.33	.06	.18	-.20
81. ADVASS	.31	.65*	.51	-.16	.30
82. FLYALL	.34	.37	.26	.25	-.20
83. FLYUH1	.61*	.71**	.54	.35	-.21
84. SIMALL	.16	.03	.07	.28	-.28
85. SIMUH1	.73**	.08	.28	.45	-.36
86. TOBAC	-.27	.08	-.19	.25	-.35

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	63	64	65	66	67
38. SDBPAV					
39. DBPCRW					
40. DBPCPC					
41. SHRMX					
42. HRCRX					
43. HRCPX					
44. SSBPMX					
45. SBPCRX					
46. SBPCRX2					
47. SBPCRX3					
48. SBPCPX					
49. SBPCPX2					
50. SBPCPX3					
51. SDBPMX					
52. DBPCRX					
53. DBPCPX					
54. SCOCR					
55. SCOCRR					
56. SCOCRR2					
57. COCRRM					
58. SCOCRP					
59. COCRPM					
60. SCORT					
61. SCORTR					
62. CORTRM					
63. SCORTP					
64. INSCORTP	-.15				
65. SCRTPDV	1.00++	-.15			
66. CRTPDVM	.72**	-.36	.72**		
67. CORTPM	-.83+	-.33	-.83+	-.32	
68. CORTPM2	-.84+	-.19	-.84+	-.26	.97++
69. CORTPM3	-.81+	-.14	-.81+	-.18	.93++
70. SPAPRO	-.30	-.44	-.30	-.34	.29
71. EP	.42	.19	.42	-.23	-.73**
72. SPAACQ	.03	-.30	.03	-.43	-.11
73. DECTEC	.60*	-.30	.60*	.11	-.74**
74. WRKLD	.33	-.34	.33	-.23	-.37

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	63	64	65	66	67
75. UNEXEV	.57*	-.09	.57*	-.02	-.63*
76. INFOX	-.42	.36	-.42	-.59	.04
77. SITAWR	.65*	.42	.65*	-.12	-.88+
78. INFOSOT	-.39	-.70**	-.39	-.02	.73**
79. XMONITR	.08	-.14	.08	-.06	.10
80. INFOOFR	.20	.85+	.20	.08	-.48
81. ADVASS	.44	-.47	.44	.53	-.28
82. FLYALL	.30	-.57*	.30	.14	-.11
83. FLYUH1	.56*	-.74**	.56*	.63*	-.19
84. SIMALL	.17	-.12	.17	-.21	-.18
85. SIMUH1	.64*	-.12	.64*	.33	-.56
86. TOBAC	-.01	.60*	-.01	-.10	-.30

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	68	69	70	71	72
38. SDBPAV					
39. DBPCRW					
40. DBPCPC					
41. SHRMX					
42. HRCRX					
43. HRCPX					
44. SSBPMX					
45. SBPCRX					
46. SBPCRX2					
47. SBPCRX3					
48. SBPCPX					
49. SBPCPX2					
50. SBPCPX3					
51. SDBPMX					
52. DBPCRX					
53. DBPCPX					
54. SCOCR					
55. SCOCRR					
56. SCOCRR2					
57. COCRRM					
58. SCOCRP					
59. COCRPM					
60. SCORT					
61. SCORTR					
62. CORTRM					
63. SCORTP					
64. INSCORTP					
65. SCRTPDV					
66. CRTPDVM					
67. CORTPM					
68. CORTPM2					
69. CORTPM3	.99++				
70. SPAPRO	.20	.16			
71. EP	-.79***	-.80***	.27		
72. SPAACQ	-.28	-.36	.75**	.66**	
73. DECTEC	-.77**	-.79**	.24	.72**	.65*
74. WRKLD	-.55	-.64**	.51	.75**	.87+

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	68	69	70	71	72
75. UNEXEV	-.75**	-.80***	.17	.82+	.70**
76. INFOX	.11	.13	.41	.34	.06
77. SITAWR	-.92++	-.91++	-.04	.89+	.49
78. INFOSOT	.65**	.60*	.41	-.43	.23
79. XMONITR	.00	-.06	-.17	-.08	.31
80. INFOOFR	-.35	-.28	-.82***	.01	-.64*
81. ADVASS	-.21	-.14	.32	.02	.02
82. FLYALL	-.24	-.31	.41	.25	.37
83. FLYUH1	-.26	-.27	.26	.12	.18
84. SIMALL	-.31	-.40	.27	.44	.50
85. SIMUH1	-.58	-.55	-.24	.41	.11
86. TOBAC	-.20	-.13	-.21	.28	-.05

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	73	74	75	76	77
38. SDBPAV					
39. DBPCRW					
40. DBPCPC					
41. SHRMX					
42. HRCRX					
43. HRCPX					
44. SSBPMX					
45. SBPCRX					
46. SBPCRX2					
47. SBPCRX3					
48. SBPCPX					
49. SBPCPX2					
50. SBPCPX3					
51. SDBPMX					
52. DBPCRX					
53. DBPCPX					
54. SCOCR					
55. SCOCRR					
56. SCOCRR2					
57. COCRRM					
58. SCOCRP					
59. COCRPM					
60. SCORT					
61. SCORTR					
62. CORTRM					
63. SCORTP					
64. INSCORTP					
65. SCRTPDV					
66. CRTPDVM					
67. CORTPM					
68. CORTPM2					
69. CORTPM3					
70. SPAPRO					
71. EP					
72. SPAACQ					
73. DECTEC					
74. WRKLD	.86+				

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	73	74	75	76	77
75. UNEXEV	.89+	.82+			
76. INFOX	-.08	.03	-.25		
77. SITAWR	.66*	.58	.79**	-.06	
78. INFOSOT	-.35	.01	-.11	-.35	-.56
79. XMONITR	.35	.15	.47	-.72**	.17
80. INFOOFR	-.45	-.51	-.13	-.05	.45
81. ADVASS	.01	-.07	-.06	-.06	.15
82. FLYALL	.69**	.63*	.35	.15	.02
83. FLYUH1	.46	.35	.24	-.31	-.13
84. SIMALL	.55	.53	.52	.10	.46
85. SIMUH1	.10	.17	.46	-.42	.65*
86. TOBAC	.12	-.15	.19	.00	.25

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	78	79	80	81	82
75. UNEXEV					
76. INFOX					
77. SITAWR					
78. INFOSOT					
79. XMONITR	.35				
80. INFOOFR	-.76**	.01			
81. ADVASS	-.29	-.20	.02		
82. FLYALL	.02	-.04	-.57	.29	
83. FLYUH1	.16	.10	-.46	.58	.68**
84. SIMALL	-.21	.19	-.13	.38	.62*
85. SIMUH1	-.15	-.08	.10	.30	-.12
86. TOBAC	-.24	.28	.58	-.40	-.59*

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Table G2 (cont.)

Variables	83	84	85	86
75. UNEXEV				
76. INFOX				
77. SITAWR				
78. INFOSOT				
79. XMONITR				
80. INFOOFR				
81. ADVASS				
82. FLYALL				
83. FLYUH1				
84. SIMALL	.27			
85. SIMUH1	.20	.17		
86. TOBAC	-.33	-.50	-.15	

Note: *p<.1, **p<.05, ***p<.01, +p<.005, ++p<.001

Appendix H

Study 2 Variables Glossary

ADVASS - Advocacy and assertion; proactively advocating what is believed to be a correct course of action. This is one of the aircrew coordination qualities.

BLCOCR - Baseline cortisol in ratio over creatinine; measured in μ g cortisol/ mg creatinine X 10^{-2}.

BLCORT - Baseline cortisol; measured in μ g/dL.

BLDBP - Baseline diastolic blood pressure.

BLHR - Baseline heart rate.

BLSBP - Baseline systolic blood pressure.

CH1 - Cognitive hardiness as measured at time 1.

COCRPM - The individualized magnitude of deviation from BLCOCR (|SCOCRP|).

COCRRM - The raw magnitude of deviation of from BLCOCR (|SCOCRR|).

CORTPM - The individualized magnitude of deviation from BLCORT (|SCORTP|).

CORTPM2 - $CORTPM^2$.

CORTPM3 - $CORTPM^3$.

CORTRM - The raw magnitude of deviation from BLCORT (|SCORTR|).

CRTPDVM - The magnitude of deviation from a SCORTP value of -48.8 (the peak of the SCORTP X hardiness curve as illustrated in Figure 5); mathematically, |SCRTPDV| or |(SCORTP - (-48.8))|.

DBPCPC - The averaged individualized change in diastolic blood pressure from BLDBP (((SDBPAV - BLDBP)/BLDBP) X 100).

DBPCPX - The maximum individualized change in diastolic blood pressure from BLDBP (((SDBPMX - BLDBP)/BLDBP) X 100).

DBPCRW - The averaged raw change in diastolic blood pressure from BLDBP (SDBPAV - BLDBP).

DBPCRX - The maximum raw change in diastolic blood pressure from BLDBP (SDBPMX - BLDBP)

DECTEC - Decision-making technique; includes risk assessment, information consolidation, problem-pattern recognition, and identification of critical factors to define and implement a solution. This is one of the aircrew coordination qualities.

EDUC - Education level.

EP - Emergency procedure; resolving presented emergency situations while maintaining in-flight protocols (for example, maintaining authorized headings and flight levels). This is a component of SPAPRO.

FLYALL - Flight experience (hours) in all aircraft.

FLYUH1 - Flight experience (hours) in the UH-1 helicopter.

GLOPA - Global performance appraisal by immediate supervisor per Department of the Army Form 67-8, US Army Officer Evaluation Report.

GLPAPI - Global performance appraisal of knowledge, skills, and general abilities as a pilot; completed by members of the company's Standardization section.

HRCHPC - The averaged individualized change in heart rate from BLHR (((SHRAV - BLHR)/BLHR) X 100).

HRCHRW - The averaged raw change in heart rate from BLHR (SHRAV - BLHR).

HRCPX - The maximum individualized change in heart rate from BLHR (((SHRMX - BLHR)/BLHR) X 100).

HRCRX - The maximum raw change in heart rate from BLHR (SHRMX - BLHR).

ILPDDYS - Physician-diagnosed illness rate.

ILPINDY - Physician-diagnosed illness incident rate.

ILSDDYS - Self-diagnosed illness rate.

ILSINDY - Self-diagnosed illness incident rate.

INFOOFR - Information offered; this is one of the aircrew coordination qualities.

INFOSOT - Information sought; this is one of the aircrew coordination qualities.

INFOX - Clarity and completeness of information transfer among crew and with external contacts; this is one of the aircrew coordination qualities.

INPDDYS - Physician-diagnosed injury rate.

INPINDY - Physician-diagnosed injury incident rate.

INPRETHR - (-1/PRETHR).

INPSTTHR - (-1/PSTTHR).

INSCORTP - (-1/SCORTP).

INSDDYS - Self-diagnosed injury rate.

INSINDY - Self-diagnosed injury incident rate.

LESNG - Negative change (impact) score.

LESNGIN - Number of negative life experiences/incidents.

LESPS - Positive change (impact) score.

LESPSIN - Number of positive life experiences/incidents.

MARITAL - Marital status.

OFFPOT - Officer's potential for promotion to the next grade; this is a component of GLOPA.

PERFRAT - Officer's performance during the rating period; this is a component of GLOPA.

POTEVAL - Evaluation of the officer's overall potential; this is a component of GLOPA.

PRECEN - Centrality of the simulator scenario; measured pre-simulator.

PRECHL - Challenge appraisal for the simulator scenario; measured pre-simulator.

PRECON - Appraisal of controllability of the simulator scenario; measured pre-simulator.

PRETHR - Threat appraisal for the simulator scenario; measured pre-simulator.

PROCOMP - Professional competence on 14 dimension; this is a component of GLOPA.

PRUNCN - Appraisal of uncontrollability of the simulator scenario; measured pre-simulator.

PSS1 - Global perceived stress as measured at time 1.

PSTCHL - Challenge appraisal for the simulator scenario; measured post-simulator.

PSTSTR - Stressfulness of the simulator scenario; measured post-simulator.

PSTSTR2 - $PSTSTR^2$.

PSTSTR3 - $PSTSTR^3$.

PSTTHR - Threat appraisal for the simulator scenario; measured post-simulator.

SBPCPC - The average individualized change in systolic blood pressure from BLSBP (((SSBPAV - BLSBP)/BLSBP) X 100).

SBPCPC2 - $SBPCPC^2$.

SBPCPC3 - $SBPCPC^3$.

SBPCPX - The maximum individualized change in systolic blood pressure from BLSBP (((SSBPMX - BLSBP)/BLSBP) X 100).

SBPCPX2 - $SBPCPX^2$.

SBPCPX3 - $SBPCPX^3$.

SBPCRW - The averaged raw change in systolic blood pressure from BLSBP (SSBPAV - BLSBP).

SBPCRW2 - $SBPCRW^2$.

SBPCRW3 - $SBPCRW^3$.

SBPCRX - The maximum raw change in systolic blood pressure from BLSBP (SSBPMX - BLSBP).

SBPCRX2 - $SBPCRX^2$.

SBPCRX3 - $SBPCRX^3$.

SCOCR - Scenario cortisol in ratio over creatinine; measured in μg cortisol/ mg creatinine X 10^{-2}.

SCOCRP - The individualized change in cortisol (in ratio over creatinine) from BLCOCR (((SCOCR - BLCOCR)/BLCOCR) X 100).

SCOCRR - The raw change in cortisol (in ratio over creatinine) from BLCOCR (SCOCR - BLCOCR).

SCOCRR2 - $SCOCRR^2$.

SCORT - Scenario cortisol; measured in μg/dL.

SCORTP - The individualized change in cortisol from BLCORT (((SCORT - BLCORT)/BLCORT) X 100).

SCORTR - The raw change in cortisol from BLCORT (SCORT - BLCORT).

SCRTPDV - (SCORTP - (-48.8)); -48.8 is that value of SCORTP at the peak of the SCORTP X hardiness curve as illustrated in Figure 5.

SDBPAV - The averaged diastolic blood pressure for the simulator scenario.

SDBPMX - The maximum diastolic blood pressure for the simulator scenariol

SHRAV - The averaged heart rate for the simulator scenario.

SHRMX - The maximum heart rate for the simulator scenario.

SIMALL - Experience (hours) in all aircraft simulators.

SIMUH1 - Experience (hours) in all UH-1 helicopter simulators.

SITAWR - Situational awareness; awareness of aircraft and mission status, spatial orientation of the aircraft, environmental conditions, and active efforts to recognize and manage these as well as stress, boredom, fatigue, and anger. This is one of the aircrew coordination qualities.

SPAACQ - Scenario performance appraisal; the mean of aircrew coordination quality scores.

SPAPRO - Scenario performance appraisal; the mean of maneuver/procedure scores.

SSBPAV - The averaged systolic blood pressure for the scenario.

SSBPMX - The maximum systolic blood pressure for the scenario.

TILINDY - Total illness incident rate.

TIMEAD - Time (months) on active military duty.

TINDY - Total illness/injury incident rate.

TININDY - Total injury incident rate.

TOBAC - Tobacco use.

TODYS - Total illness/injury rate.

TOILDYS - Total illness rate.

TOINDYS - Total injury rate.

UNEXEV - Management of unexpected events; this is performance under unusual and high stress circumstances, to include proactive information exchange, prioritization of actions, and optimal distribution of information and workload. This is one of the aircrew coordination qualities.

WRKLD - Workload management and action prioritization; this includes focus upon essential actions, optimal distribution of task load among the crew, and optimal identification and prioritization of competing tasks. This is one of the crew coordination qualities.

XMONITR - Cross-monitoring of crew member actions. This is one of the aircrew coordination qualities.